and

Benchmark Toxicology Services

HEALTH RISK ASSESSMENT OF ATMOSPHERIC EMISSIONS EXPANSION OF WAGERUP REFINERY TO 4.7 MTPA

for

Alcoa World Alumina Australia

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19 April 2005

ENVIRON

19 April 2005

Alcoa World Alumina Australia
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Attention: David Hanham

Dear David,

HEALTH RISK ASSESSMENT OF ATMOSPHERIC EMISSIONS
EXPANSION OF THE WAGERUP REFINERY TO 4.7 MTPA

We are pleased to present our final report for the Atmospheric Emissions Health Risk Assessment for the expansion of the Wagerup refinery to 4.7 Mtpa.

Should you require any additional information, please contact the undersigned directly.

Yours faithfully
ENVIRON Australia Pty Ltd

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EXECUTIVE SUMMARY

A Health Risk Assessment (HRA) of the atmospheric emissions from Alcoa's Wagerup refinery has been undertaken to investigate the potential health risks arising from the emissions. The HRA considered the potential health risks associated with a baseline and an expanded refinery emissions scenario, defined as follows:

- baseline emissions scenario representative of emissions from the existing Wagerup refinery operating at an alumina production rate of 2.41 Mtpa; and
- expanded emissions scenario representative of emissions from an expanded Wagerup refinery operating at an alumina production rate of 4.7 Mtpa.

The HRA has been confined to the inhalation pathway as this is expected to represent the most significant exposure route to the Wagerup refinery's emissions and therefore did not take into account the alternative exposure pathways (e.g. ingestion, dermal absorption), nor other sources of atmospheric emissions of these compounds. Of the pollutants considered in this HRA, only cadmium (chronic non-carcinogenic HI (Hazard Index)) and arsenic (incremental carcinogenic risk) were assessed as requiring further assessment of alternative exposure pathways based on the results of the Hot Spots Analysis and Reporting Program (HARP) developed by Californian environmental agencies. The subsequent assessment indicated that non inhalation exposure pathways for these substances did not result in any unacceptable impacts.

The following quantitative health risk indicators were calculated across the model domain and for key receptors located in the vicinity of the Wagerup refinery:

- acute HI;
- chronic HI; and
- ICR (Incremental Cancer Risk).

ENVIRON was provided with ground level concentrations predicted from air dispersion modelling for a number of compounds present in the atmospheric emissions from the Wagerup refinery for both the baseline and expanded emissions scenarios. The air dispersion modelling included both the refinery and residue drying areas (RDA) emissions and was completed by the CSIRO (Refinery) (CSIRO, 2005a, 2005b) and Air Assessments (RDA) (Air Assessments, 2005) with the modelling results integrated by ENVIRON.

The potential health effects arising from the predicted short-term (acute; 1-hour and 24-hour averages) and long-term (chronic; annual averaged) exposure to non-carcinogenic compounds, and potential carcinogenic risks were considered in the HRA assessment by comparing the exposure concentrations predicted by the modelling with health protective guidelines for ambient air developed by reputable authorities such as the National Environment Protection Council (NEPC), World Health Organisation (WHO) and the U.S Environmental Protection Agency (USEPA).

The acute and chronic Hazard Indices (HI) were calculated to evaluate the potential for non-carcinogenic adverse health effects from simultaneous exposure to multiple compounds by summing the ratio of the predicted concentration in air to the health protective guidelines for individual compounds. A HI of less than one is generally considered to represent no cause for concern with respect to adverse health effects.

To assess the potential health effects associated with exposure to carcinogens, the incremental carcinogenic risk (ICR) was calculated to provide an indication of the incremental probability that an individual will develop cancer over a lifetime as a direct result of exposure to potential carcinogens. The incremental carcinogenic risk that is considered acceptable varies amongst jurisdictions, typically ranging from one in a million ($1x10^{-6}$) to one in ten thousand ($1x10^{-4}$). The most stringent criterion of one in a million represents the USEPA's *de minimis*, or essentially negligible incremental risk level, and has therefore been adopted for this screening assessment as a conservative (i.e. health protective) indicator of acceptable carcinogenic risk.

The acute and chronic HIs and the ICRs were calculated for each model grid point and these data were contoured to provide the calculated health risks across the entire model domain. The HIs and ICRs at 16 discrete receptor locations were then calculated from the contours. The discrete receptor locations (Figure 1) were identified by Alcoa to represent populations or individual residences that could be potentially exposed. The HI and ICR contours can also be used to estimate the potential health risks at other locations, including other discrete receptor locations subsequently identified by Alcoa, if required.

Based upon the results of the health screening assessment it can be concluded that:

- the potential for emissions from the existing or expanded Wagerup refinery to cause acute health effects is low and is primarily driven by the particulate emissions from the RDA and oxides of nitrogen emissions from the refinery;

- the potential for emissions from the existing or expanded Wagerup refinery to cause chronic non-carcinogenic health effects is very low; and

- the potential for emissions from the existing or expanded Wagerup refinery to contribute to the incidence of cancer based on inhalation exposure is below USEPA *de minimis* threshold of one in a million (i.e. 1×10^{-6}) at all of the residential receptors considered;

As with any risk evaluation, there are areas of uncertainty in this assessment. To ensure that potential risks are not underestimated, uniformly conservative assumptions have been used to characterize exposure and toxicity. Due to the resultant compounding of conservatism, the quantitative risk indicators should be considered as over-estimates of potential health risks associated with the atmospheric emissions from the Wagerup refinery.

HEALTH RISK ASSESSMENT OF ATMOSPHERIC EMISSIONS EXPANSION OF THE WAGERUP REFINERY TO 4.7 MTPA

for

Alcoa World Alumina Australia

1. INTRODUCTION

Alcoa World Alumina Australia (Alcoa) has commissioned ENVIRON to conduct a Health Risk Assessment (HRA) of the potential health risks arising from atmospheric emissions from the Wagerup refinery. The HRA has considered the potential health risks associated with a baseline and an expanded refinery emissions scenario, defined as follows:

- baseline emissions scenario representative of emissions from the existing Wagerup refinery operating at an alumina production rate of 2.41 Mtpa; and
- expanded emissions scenario representative of emissions from an expanded Wagerup refinery operating at an alumina production rate of 4.7 Mtpa. Alcoa has identified two expansion scenarios, one including the installation of cogeneration units (Case 6) and the other without (Case 7).

The air dispersion modelling results used in this HRA includes the predicted cumulative impacts from both the refinery and the Residue Drying Areas (RDA) and was completed by the CSIRO (Refinery) and Air Assessments (RDA) with the modelling results integrated by ENVIRON.

This report outlines the approach used to conduct the HRA and presents the results of potential acute, chronic non-carcinogenic, and incremental carcinogenic health risks arising from atmospheric emissions from the Wagerup refinery over the model domain and at key receptor locations in the vicinity of the refinery and RDA.

2. OVERVIEW OF THE SCREENING ASSESSMENT APPROACH

Risk assessment provides a systematic approach for characterising the nature and magnitude of the risks associated with environmental health hazards, and is an important tool for decision-making (enHealth, 2002). The generic steps involved in health risk assessment include:

Exposure Assessment: defines the amount, frequency, duration and routes of exposure to compounds present in environmental media. In this assessment, exposure is estimated as the concentration of a compound that a person may be exposed to over both short- (i.e. acute) and long-term (i.e. chronic) exposure periods;

Toxicity Assessment: identifies the nature and degree of toxicity of chemical compounds, and characterises the relationship between magnitude of exposure and adverse health effects (i.e. the dose-response relationship);

Risk Characterisation: the combining of exposure and toxicity data to estimate the magnitude of potential health risks associated with exposure periods of interest; and

Uncertainty Assessment: identification of potential sources of uncertainty and qualitative discussion of the magnitude of uncertainty and expected effects on risk estimates.

This HRA conducted of the Wagerup refinery's emissions is considered to be a screening-level assessment in that it makes generally conservative default assumptions regarding the potential magnitude of exposure and uses conservative toxicity criteria. The quantitative health risk indicators calculated for potential acute and chronic health effects are based on the assumption that the health effects arising from exposure to each of the individual compounds emitted from the Wagerup refinery are additive. The additive approach is considered to be appropriate for screening assessment purposes, and is considered to be conservative (i.e. health protective) in most circumstances. It should however be noted that it does not account for potential synergistic effects which are discussed in more detail in Appendix A.

On account of the conservatism of such a screening assessment, the results are considered more likely to over- than under-estimate the potential health risks associated with atmospheric emissions from the Wagerup refinery and the RDA. The results of the HRA are able to be used to assess the relative

change to potential health risks associated with an expansion of the Wagerup refinery, and identify the individual sources and compounds exhibiting the highest contribution to potential health risks in order to help define atmospheric emissions management strategies.

3. EXPOSURE ASSESSMENT

3.1 Compounds Considered

Alcoa has undertaken a review of emission monitoring data available for its Wagerup, Pinjarra and Kwinana refineries to characterise atmospheric emissions released from its operations, and to characterise atmospheric emissions expected to be released from an expanded Wagerup refinery. ENVIRON (2005a) provides details on the process that Alcoa has undertaken to identify the 27 individual compounds or groups of compounds that have been considered in this HRA. In selecting the 27 compounds to be included within the HRA, Alcoa initially considered the 141 individual compounds or groups of compounds that were quantified as part of the Pinjarra Refinery Efficiency Upgrade health risk evaluation. A screening assessment of these compounds found that the 27 individual compounds or groups of compounds considered in this assessment contributed over 93% of the acute HI, over 86% of the chronic HI, and 100% of the incremental carcinogenic risk calculated for the Pinjarra Refinery Efficiency Upgrade health risk evaluation at the maximally affected receptor (receptor 1) (Toxikos, 2003). Based on the findings of the Pinjarra Refinery Efficiency Upgrade health risk evaluation (Toxikos, 2003), the compounds considered in the Wagerup refinery screening assessment are expected to contribute the vast majority of the potential health risks. ENVIRON believes that the process used to identify and select the compounds included within the HRA was comprehensive and appropriate given the current state of knowledge of the refinery and RDA emissions

The 27 individual compounds or groups of compounds comprise the following compound classes:

- particulates;
- products of combustion;
- metals;
- organic compounds (e.g. aldehydes, ketones and aromatics [including polycyclic aromatic hydrocarbons (PAHs)]); and
- ammonia.

Emissions for the baseline emissions scenario for the refinery have been derived by Alcoa based on various stack emission monitoring programs conducted primarily at the Wagerup refinery but the monitoring results from the Pinjarra and Kwinana refineries have also been considered. The emissions inventory has been produced by Alcoa and is summarised in ENVIRON (2005b). The

emissions inventory for the RDA used in the modelling has been determined from source and ambient monitoring (ENVIRON, 2005b).

Emissions for the expanded refinery emissions scenario have been derived by Alcoa based on a notional design for the expanded refinery, and using conservative estimates of pollution control efficiencies for those sources to be upgraded with new air pollution control equipment as part of the Wagerup refinery expansion.

For both the baseline and expanded refinery emission scenarios, "peak" and "average" emissions have been estimated and applied to the assessment of acute and chronic exposure respectively. Typically peak emissions have been defined using the maximum measured emission concentration while the average emissions have been defined by calculating the average of all measured emissions concentrations over the defined monitoring period.

Table 1, lists the compounds modelled and included in the HRA and provides information on the peak and average mass emission rates for the baseline and expanded refinery emission scenarios.

Table 1: Compounds Modelled with the Peak and Average Emission Rates from the Refinery

No.	Compound Name	Baseline Wagerup Refinery Emissions (g/s)		Expanded Wagerup Refinery Emissions (g/s)			
				With Cogeneration (Case 6)		Without Cogeneration (Case 7)	
		Average Case	Peak Case	Average Case	Peak Case	Average Case	Peak Case
1	Oxides of Nitrogen[1]	31.9	75.2	62.6	92.9	39.8	71.7
2	Carbon monoxide	28.9	78.0	51.8	102.6	44.9	98.7
3	Sulphur dioxide	2.2	9.3	3.6	10.5	3.4	11.4
4	Particulate matter	1.9	8.4	2.1	5.3	2.1	5.3
5	Arsenic	2.55E-03	3.49E-03	2.45E-03	2.68E-03	6.71E-03	7.29E-03
6	Selenium	1.02E-03	1.15E-03	9.94E-04	1.21E-03	9.94E-04	1.21E-03
7	Manganese	0.010	0.017	0.004	0.005	0.006	0.007
8	Cadmium	2.23E-07	2.23E-07	0.0	4.45E-07	0.0	4.45E-07
9	Chromium (VI)	2.09E-05	2.09E-05	2.26E-05	2.46E-05	3.08E-05	3.35E-05
10	Nickel	5.15E-04	8.23E-04	2.84E-04	4.73E-04	2.84E-04	4.73E-04
11	Mercury	0.007	0.007	0.003	0.003	0.003	0.003
12	Ammonia	0.32	0.50	0.28	0.29	0.28	0.29
13	Polycyclic Aromatic Hydrocarbons	1.96E-05	2.73E-05	1.19E-05	1.28E-05	1.19E-05	1.28E-05
14	Acetone	1.13	2.64	1.18	1.24	1.24	1.30
15	Acetaldehyde	0.38	0.81	0.63	0.67	0.63	0.68
16	Formaldehyde	0.54	1.15	0.73	0.79	0.71	0.76
17	2-Butanone	0.11	0.31	0.13	0.14	0.15	0.16
18	Benzene	0.07	0.17	0.07	0.08	0.08	0.09
19	Toluene	0.10	0.18	0.05	0.05	0.05	0.05
20	Xylenes	0.02	0.02	0.01	0.01	0.01	0.01
21	Acrolein	0.04	0.04	0.07	0.08	0.07	0.08

No.	Compound Name	Baseline Wagerup Refinery Emissions (g/s)		Expanded Wagerup Refinery Emissions (g/s)			
				With Cogeneration (Case 6)		Without Cogeneration (Case 7)	
		Average Case	Peak Case	Average Case	Peak Case	Average Case	Peak Case
22	Ethylbenzene	0.001	0.002	0.002	0.003	0.002	0.003
23	Methylene Chloride	0.16	0.24	0.12	0.13	0.15	0.16
24	Styrene	0.007	0.007	0.005	0.005	0.005	0.005
25	1,2,4 Trimethylbenzene	7.06E-04	1.18E-03	3.61E-04	4.04E-04	3.61E-04	4.04E-04
26	1,3,5 Trimethylbenzene	4.44E-04	6.27E-04	5.62E-04	6.28E-04	5.62E-04	6.28E-04
27	Vinyl chloride	2.38E-04	2.75E-04	4.65E-04	5.20E-04	4.65E-04	5.20E-04

Notes: 1. Oxides of Nitrogen expressed as Nitrogen Dioxide.

Compounds included in the modelling for the RDA were particulate matter, arsenic, selenium, manganese, cadmium, nickel, mercury, polycyclic aromatic hydrocarbons, acetone, acetaldehyde, formaldehyde, 2-butanone, benzene, toluene and xylenes. Air Assessments (2005), provides details of the derivation of the RDA emissions used in the modelling.

3.2 Potential Receptor Locations

Alcoa identified 16 receptor locations to represent the populations or individual residences that are considered to represent the range of potential exposure to atmospheric emissions from the Wagerup refinery, as presented in Table 2.

Table 2: Receptor Locations

Receptor No.	Type
1	Individual residence
2	Individual residence
3	Individual residence
4	Individual residence
5	Individual residence
6	Individual residence
7	Individual residence
8	Individual residence
9	Individual residence
10	Individual residence
11	Individual residence
12	Residential population
13	Residential population
14	Individual residence
15	Individual residence
16	Individual residence

The locations of the receptors in relation to the Alcoa refinery site are presented in Figure 1, overlain on a map of the local area.

For purposes of this screening assessment, all receptors are assumed to be residents, including potentially sensitive subpopulations such as children and the elderly. This assumption is inherent in the health protective guidelines selected (refer to Section 4).

The potential health risks associated with the refinery and RDA atmospheric emissions for locations other than the 16 identified above can be estimated directly from the HI and ICR contours (see Sections 5.1 to 5.3).

3.3 Potentially Complete Exposure Pathways

Inhalation is expected to represent the most significant exposure route for the Wagerup refinery and therefore the exposure assessment has been confined to the inhalation pathway.

The California Air Toxics Hot Spots Program Risk Assessment Guidelines (OEHHA, 2000) provides a list of compounds for which multi-pathway exposure needs to be assessed. The list has been developed based on a theoretical model for the portioning of the exchangeable fraction of an airborne compound between the vapour and particulate phases in the ambient air. The compounds tending towards the particulate phase have been identified as the most likely candidates for multi-pathway exposure as they will tend to deposit on to surfaces (e.g. soil and crops) and be available for ingestion. Compounds emitted from the Wagerup refinery that appear in the Air Toxics Hot Spots list of compounds requiring multi-pathway exposure assessment include:

- arsenic;
- cadmium;
- chromium (VI);
- nickel;
- mercury; and
- PAHs with three rings or greater.

Section 5.6.3 discusses the potential health risks associated with emissions of these compounds from the Wagerup refinery.

3.4 Estimated Concentrations in Air

Concentrations in the ambient air have been estimated based on the results of air dispersion modelling conducted by CSIRO (2005a, 2005b) and Air Assessments (2005). The CSIRO used TAPM to predict the ground level concentrations for each of the 27 compounds emitted from the refinery. Air Assessments used the CALPUFF (v5.714) model to predict the ground level concentrations for the nominated compounds arising from the RDA. The CALPUFF model was run utilising the same wind field as derived and used by the CSIRO. ENVIRON integrated the TAPM and CALPUFF modelling results for each pollutant for each grid point, for each hour of the year modelled. Once the files were integrated, the predicted concentrations were analysed to produce the following:

1. 99.9th and 99.5th percentile 1-hour average concentration;
2. 99.5th and 95th percentile 24-hour average concentration; and
3. annual average concentration,

for each pollutant for each grid point over the model domain.

The predicted 99.9th percentile 1-hour average and the 99.5th percentile 24-hour average concentrations have been used to represent the maximum exposures for use in the HRA based on CSIRO's report that indicated that the 9th highest concentration (99.9th percentile) is often chosen as the key statistic to represent the extremes, rather than the modelled maximum (CSIRO, 2005b, page 76).

3.4.1 Averaging Period Adjustment

Some acute health protective guidelines refer to an averaging period that does not correspond to the 1-hour or 24-hour averages predicted by the air dispersion modelling. To ensure consistency between the averaging period corresponding to acute health protective guidelines and the predicted ground level concentration, the power law of Hanna, Briggs and Hosker (Equation 1) has been applied to the predicted ground level concentrations of those compounds for which the health protective guidelines refer to averaging periods other than 1-hour or 24-hours (i.e. carbon monoxide [8-hour], acrolein [30-minute], and styrene [1 week]).

$$GLC_n = GLC_m \times \left[\frac{m}{n}\right]^{0.2} \qquad \textbf{\textit{Equation 1}}$$

Where:

n = averaging period of health protective guideline (hours)

GLC_n = ground level concentration averaged over n hours ($\mu g/m^3$)

m = averaging period of predicted ground level concentration (i.e. 1-hour or 24-hour) (hours)

GLC_m = ground level concentration averaged over m hours ($\mu g/m^3$)

For carbon monoxide and acrolein the 1-hour average predicted ground level concentration was used in Equation 1 (i.e. m = 1-hour), and for styrene the 24-hour average predicted ground level concentration was used in Equation 1 (i.e. m = 24-hours).

Additional discussion on averaging times is provided in Section 7.1 of Appendix A.

4. TOXICITY ASSESSMENT

The toxicity assessment determines the relationship between the magnitude of exposure to a chemical of interest and the nature and severity of adverse health effects that may result from such exposure. Chemical toxicity is divided into two categories for purposes of risk assessment: carcinogenic and non-carcinogenic. Some chemicals exert both types of effects. Whilst all non-carcinogenic effects are assumed to occur only at exposure levels greater than some threshold at which defense mechanisms are overwhelmed, carcinogens are thought to act via both threshold and non-threshold mechanisms. By convention, exposure to even one molecule of a genotoxic carcinogen is assumed to incur some small but finite risk of causing cancer; hence, the action of such compounds is considered to lack a threshold below which adverse effects are not expected to occur. In contrast, the effects of non-genotoxic carcinogens are thought to be manifested only at exposures in excess of compound-specific thresholds. Potential health risks are calculated differently for threshold and non-threshold effects because their toxicity criteria are based on different mechanistic assumptions and expressed in different units.

A number of national and international regulatory agencies have reviewed the toxicity of environmental chemicals and developed acceptable exposure criteria (herein referred to as "health protective guidelines') in accordance with both carcinogenic and non-carcinogenic endpoints. Health protective guidelines from the following reputable authorities were considered for use in the screening assessment:

- National Environment Protection (Ambient Air Quality) Measure (NEPC, 1998);
- National Environment Protection (Air Toxics) Measure (NEPC, 2004);
- World Health Organisation (WHO) Air Quality Guidelines for Europe Second Edition (WHO, 2000);
- Guidelines for Air Quality (WHO, 2000a)
- U.S. Environment Protection Agency's (USEPA) Integrated Risk Information System (IRIS);
- U.S. Agency for Toxic Substances and Disease Registry's (ATSDR) Minimal Risk Levels (MRLs) for Hazardous Substances;
- Dutch National Institute of Public Health and the Environment (RIVM) human-toxicological Maximum Permissible Risk Levels (RIVM, 2001);
- Health Canada's health-based Tolerable Daily Intakes/Concentrations and Tumorigenic Doses/Concentrations for priority substances (Health Canada, 1996); and

- California Office of Environmental Health Hazard Assessment's (OEHHA) Toxicity Criteria Database.

Health protective guidelines published by the National Environment Protection Council (NEPC), followed by the WHO, have been applied in preference to the other health protective guidelines listed above. This is consistent with the enHealth Guidelines for Assessing Human Health Risks from Environmental Hazards (2002), and consistent with advice received from the Department of Health (Western Australia) for the Pinjarra Refinery Efficiency Upgrade.

For those compounds not covered by the NEPC or WHO, the guidelines most recently determined (on an individual compound basis) by the USEPA (IRIS), ATSDR, RIVM and Health Canada have been applied, on the basis that the most recent guidelines are most likely to have been developed from the most up-to-date toxicological information.

The OEHHA guidelines have been applied for the compounds not covered by the other health protective guidelines. The other published guidelines have been used in preference to the OEHHA as the OEHHA guidelines are not applicable at a national level whilst the other health protective guidelines are. Also the OEHHA guidelines tend to be based upon values published by other reputable authorities rather than being developed from first principles based on results of actual toxicological studies. The OEHHA guidelines are, however, considered useful for the HRA in that they are one of the few sources that publish acute health protective guidelines for a comprehensive list of compounds.

The health protective guidelines applied within the HRA are presented in Table 3, and are briefly discussed in the following sections. A comprehensive discussion of the hazardous characteristics of the chemicals of concern and the derivation and selection of reference values is provided in Appendix A.

Table 3: Health Protective Guidelines

No.	Compound Name	Guideline	Units	Averaging Period	Value (µg/m^3)	Reference
Acute Health Effects						
1	Nitrogen Dioxide	246	µg/m^3	1 h	246	NEPC
2	Carbon monoxide	11250	µg/m^3	8 h	11,250	NEPC
3	Sulphur dioxide	571	µg/m^3	1 h	571	NEPC
4	Particulate matter < 10 µm	50	µg/m^3	24 h	50	NEPC
10	Nickel	6	µg/m^3	1 h	6.0	OEHHA
11	Mercury	1.8	µg/m^3	1 h	1.8	OEHHA

No.	Compound Name	Guideline	Units	Averaging Period	Value (µg/m^3)	Reference
12	Ammonia	3200	µg/m^3	1 h	3,200	OEHHA
14	Acetone	26	ppm	24 h	67,414	ATSDR
15	Acetaldehyde	2000	µg/m^3	24 h	2,000	WHOa
16	Formaldehyde	0.04	ppm	24 h	54	NEPC (AT)
17	2-Butanone	13000	µg/m^3	1 h	13,000	OEHHA
18	Benzene	1300	µg/m^3	6 h	1,300	OEHHA
19	Toluene	1	ppm	24 h	4,113	NEPC (AT)
20	Xylenes	0.25	ppm	24 h	1,183	NEPC (AT)
21	Acrolein	0.2	µg/m^3	1 h	0.2	OEHHA
23	Methylene Chloride	3	mg/m^3	24 h	3,000	WHO
24	Styrene	0.26	mg/m^3	1 week	260	WHO
27	Vinyl chloride	180.00	mg/m^3	1 h	180,000	OEHHA
Chronic Non-Carcinogenic Health Effects						
1	Nitrogen Dioxide	62	µg/m^3	Annual	62	NEPC
3	Sulphur dioxide	57	µg/m^3	Annual	57	NEPC
5	Arsenic	1	µg/m^3	Annual	1	RIVM
6	Selenium	20	µg/m^3	Annual	20	OEHHA
7	Manganese	0.15	µg/m^3	Annual	0.15	WHO
8	Cadmium	0.005	µg/m^3	Annual	0.005	WHO
9	Chromium (VI)	0.1	µg/m^3	Annual	0.1	IRIS
10	Nickel	0.00009	mg/m^3	Annual	0.09	ATSDR
11	Mercury	1	µg/m^3	Annual	1	WHO
12	Ammonia	0.1	mg/m^3	Annual	100	IRIS
13	Polycyclic Aromatic Hydrocarbons	0.0003	µg/m^3	Annual	0.0003	NEPC (AT)
14	Acetone	13	ppm	Annual	33,707	ATSDR
15	Acetaldehyde	50	µg/m^3	Annual	50	WHOa
16	Formaldehyde	0.008	ppm	Annual	11	ATSDR
17	2-Butanone	5	mg/m^3	Annual	5,000	IRIS
18	Benzene	0.017	ppm	Annual	60	OEHHA
19	Toluene	0.1	ppm	Annual	411	NEPC (AT)
20	Xylenes	0.2	ppm	Annual	946	NEPC (AT)
21	Acrolein	0.06	µg/m^3	Annual	0.06	OEHHA
22	Ethylbenzene	22000	µg/m^3	Annual	22,000	WHOa
23	Methylene Chloride	0.30	ppm	Annual	1,137	ATSDR
24	Styrene	900	µg/m^3	Annual	900	RIVM/OEHHA
25	1,2,4 Trimethylbenzene [1]	800	µg/m^3	Annual	800	RIVM
26	1,3,5 Trimethylbenzene [1]	800	µg/m^3	Annual	800	RIVM
27	Vinyl chloride	0.10	mg/m^3	Annual	100	IRIS
Carcinogenic Health Effects						
5	Arsenic	1.50E-03	per µg/m^3	Annual	1.50E-03	WHO
8	Cadmium	1.80E+00	per mg/m^3	Annual	1.80E-03	IRIS

No.	Compound Name	Guideline	Units	Averaging Period	Value (µg/m^3)	Reference
9	Chromium (VI)	4.00E-02	per µg/m^3	Annual	4.00E-02	WHO
10	Nickel	3.80E-04	per µg/m^3	Annual	3.80E-04	WHO
13	Polycyclic Aromatic Hydrocarbons	8.70E-05	per ng/m^3	Annual	8.70E-02	WHO
15	Acetaldehyde	9.00E-07	per µg/m^3	Annual	9.00E-07	WHOa
18	Benzene	6.00E-06	per µg/m^3	Annual	6.00E-06	WHO
23	Methylene Chloride	4.70E-07	per mg/m^3	Annual	4.70E-10	IRIS
27	Vinyl chloride	1.00E-06	per µg/m^3	Annual	1.00E-06	WHO

Notes: 1. Only those compounds with a health protective guideline are listed under each category (i.e. acute, chronic non-carcinogenic and carcinogenic).

4.1 Non-Carcinogenic Effects

A non-carcinogenic effect is defined as any adverse response to a chemical that is not cancer. Any chemical can cause adverse health effects if given at a high enough dose. When the dose is sufficiently low, no adverse effect is observed. Indeed, increasing evidence suggests that low doses of chemicals generally have beneficial effects, a phenomenon known as hormesis (e.g., Calabrese, 2004). Thus, in characterising the non-carcinogenic effects of a chemical, the key parameter is the threshold dose at which an adverse effect first becomes evident. Doses below the threshold are considered to be "safe" (i.e. not associated with adverse effects), while doses above the threshold may cause an adverse effect.

The threshold dose is typically estimated from toxicological or epidemiological data by finding the highest dose level that produces no observable adverse effect (a NOAEL) or the lowest dose level that produces an observable adverse effect (a LOAEL). Where more than one such value is available, preference is given to studies using most sensitive species, strain and sex of experimental animal known, the assumption being that humans are no less sensitive than the most sensitive animal species tested. For the guidelines developed by all the authorities considered, NOAELs or LOAELs are divided by the product of a series of uncertainty factors representing experimental vs. environmental exposure duration, inter- and intra-species variability and the quality and completeness of the toxicological database. This procedure ensures that the resultant health protective guidelines are not higher than (and may be orders of magnitude lower than) the threshold level for adverse effects in the most sensitive potential receptor. Thus, there is a “margin of safety” built into the guideline, and doses equal to or less than that level are nearly certain to be without any adverse effect. The likelihood of an adverse effect at doses higher than the guideline increases, but because of the margin of safety, a greater dose does not mean that such an effect will necessarily occur.

4.1.1 Short-Term (Acute) Exposure

Health protective guidelines for acute non-carcinogenic health effects are expressed as concentrations in air that are not expected to cause any adverse effects as a result of continuous exposure over a defined averaging period (typically 24 hours or less). These guidelines are appropriate for comparison with 1-hour or 24-hour average exposure estimates. Although derived from different sources, the guidelines selected for this assessment are all intended to be protective of continually exposed (i.e. residential) receptors, including potentially sensitive subpopulations.

4.1.2 Long-Term (Chronic) Exposure

Health protective guidelines for chronic non-carcinogenic health effects are expressed as concentrations in air that are not expected to cause any adverse health effects as a result of continuous long-term exposure (a year or more). These guidelines are appropriate for comparison with annual average exposure estimates.

4.2 Carcinogenic Effects

Cancers are generally defined as diseases of mutation affecting cell growth and differentiation. Although many chemicals are known to cause cancer at high doses in studies with experimental animals, relatively few chemicals have been shown to be carcinogenic in humans at doses likely to be encountered in the ambient environment. Cancers are relatively slow to develop, and usually require prolonged exposure to carcinogenic chemicals. As a result, potential carcinogenic risks are only calculated for long-term exposures.

The International Agency for Research on Cancer (IARC) classifies substances according to their potential for human carcinogenicity as indicated in Table 4.

Table 4: IARC Classification Criteria

Group	Description
1	Carcinogenic to humans (sufficient evidence of carcinogenicity to humans)
2A	Probably carcinogenic to humans (sufficient evidence of carcinogenicity in animals, limited evidence of carcinogenicity in humans)
2B	Possibly carcinogenic to humans (less than sufficient evidence of carcinogenicity in animals, limited evidence of carcinogenicity in humans)
3	Not classifiable as to carcinogenicity in humans (inadequate or limited evidence of carcinogenicity in animals, inadequate evidence of carcinogenicity in humans)
4	Probably not carcinogenic to humans (evidence suggesting lack of carcinogenicity in animals and humans)

Those compounds present in the emissions from the Wagerup refinery that are classified by the IARC as Group 1, Group 2A or Group 2B are presented in Table 5.

Table 5: IARC Compound Classifications

Compound Name	IARC Classification
Benzene	1
Arsenic	1
Cadmium	1
Chromium (VI)	1
Vinyl chloride	1
Formaldehyde	2A
Acetaldehyde	2B
Nickel	2B
Ethylbenzene	2B
Methylene Chloride	2B
Styrene	2B
Naphthalene [1]	2B

Notes:
1. Naphthalene is one of five PAH compounds found to be present in emissions from the Wagerup refinery.

Health protective guidelines for genotoxic compounds carcinogens are expressed as unit risk (UR) factors. A UR factor is defined as the theoretical upper bound probability of extra cases of cancer occurring in the exposed population assuming lifetime exposure by inhalation to 1 $\mu g/m^3$ of the

compound (hence units are per $\mu g/m^3$) (WHO 2000). These guidelines are appropriate for comparison with annual average exposure estimates.

As irritation occurs from formaldehyde at concentrations associated with very low cancer risk, irritation is considered the more sensitive and hence more appropriate endpoint for guideline development for formaldehyde. WHO (2000) determined that 100 $\mu g/m^3$, "over one order of magnitude lower than a presumed threshold for cytotoxic damage to the nasal mucosa…, represents an exposure level at which there is a negligible risk of upper respiratory tract cancer in humans." However, because this value is higher than the 24-hour NEPM Investigation Level of 54 $\mu g/m^3$, ENVIRON has used the ATSDR chronic MRL of 10.7 $\mu g/m^3$ for assessment of chronic health risks associated with formaldehyde emissions from the Wagerup refinery. Appendix B provides a detailed review of the toxicological information that supports the treatment of formaldehyde within this screening assessment.

Some individual PAHs are clearly carcinogenic and others appear not to cause cancer, but the majority of this large class of chemicals cannot be classified as to potential carcinogenicity due to lack of sufficient data. The individual PAH compounds detected in emissions from the Wagerup refinery include:

- naphthalene
- 2-methylnaphthalene
- phenanthrene
- acenaphthene
- fluoranthene

Of these PAH compounds, naphthalene, phenanthrene and fluoranthene have been classified by the IARC as to their human carcinogenicity. Naphthalene is classified as Group 2B (possibly carcinogenic to humans). Phenanthrene and fluoranthene are classified as Group 3 (not classifiable as to human carcinogenicity).

The complex and variable composition and behaviour of PAH mixtures in the environment hinder attribution of health consequences to specific compounds. As a result, no one risk assessment approach is universally accepted. Three principal approaches reviewed by WHO (1998) are:

1 toxicity equivalence factors (TEFs);
2 comparative potency; and

3 use of benzo[*a*]pyrene as a surrogate.

WHO used the benzo[*a*]pyrene surrogate approach in its *Air Quality Guidelines for Europe* (2000); however, as benzo[*a*]pyrene has not been detected in testing for PAH emissions from the Wagerup refinery, the TEF approach has been applied for this assessment. The highest potency (relative to benzo[a]pyrene) for individual PAH compounds published in the WHO's *Environmental Health Criteria 202: Selected Non-heterocyclic Polycyclic Aromatic Hydrocarbons* (1998) has been applied in calculating exposure to the mixture of PAHs emitted from the Wagerup refinery. The relative potency applied in this assessment compared to the range of relative potencies published by the WHO is presented in Table 6.

Table 6: Relative Potency of Individual PAH Compounds [(1)]

Individual PAH Compound	Maximum Relative Potency	Range of Relative Potencies [(1)]
Naphthalene	0.001	0.001 [(2) (3)]
2-methylnaphthalene	0.001 [(2)]	
Phenanthrene	0.001	0 [(4)], 0.00064 [(6)], 0.001 [(2) (3)]
Acenaphthene	0.001	0 [(5)], 0.001 [(2) (3) (4)]
Fluoranthene	0.01	0.001 [(2) (3)], 0.01 [(4)]

Notes:
1. As published by the WHO (1998).
2. Nisbelt & LaGoy (1992).
3. Malcolm & Dobson (1994).
4. Kalberlah et al. (1995).
5. US Environmental Protection Agency (1993).
6. McClure & Schoeny (1995).

As indicated by the range of relative potencies presented in Table 6, there is a high degree of uncertainty in the toxicity data available for PAHs. Nevertheless the 1998 WHO publication is considered a credible source of such information, and hence these data have been applied for this screening assessment.

To calculate the carcinogenic risk associated with exposure to PAH emissions from the Wagerup refinery, the WHO's UR factor for benzo[a]pyrene of 8.7×10^{-2} per $\mu g/m^3$ has been applied for this assessment, which is based on studies in coke-oven workers (WHO, 2000).

4.3 Chemicals Lacking Health Protective Guidelines

Health protective guidelines for inhalation exposure for non-carcinogenic (acute or chronic) and/or carcinogenic health effects have been published by the reputable authorities mentioned above (Section 4) for all of the compounds considered in this assessment.

5. RISK CHARACTERISATION

Quantitative health risk indicators have been calculated for potential acute and chronic non-carcinogenic health effects, and carcinogenic health effects for the baseline and expanded Wagerup refinery emission scenarios. The quantitative risk indicators are described in Section 5.1, and the findings of the risk characterisation are presented in Sections 5.2 to 5.6.

5.1 Quantitative Risk Indicators

The Hazard Index (HI) is calculated to evaluate the potential for non-carcinogenic adverse health effects from simultaneous exposure to multiple compounds by summing the ratio of the estimated concentration in air to the health protective guidelines for individual compounds. The HI is calculated for acute (Equation 2) and chronic (Equation 3) exposures.

$$HI_{Acute} = \sum^{i} \frac{C_{\leq 24h}}{Gdl_{Acute}} \qquad \textbf{Equation 2}$$

$$HI_{Chronic} = \sum^{i} \frac{C_{Annual}}{Gdl_{Chronic}} \qquad \textbf{Equation 3}$$

Where:

HI_{Acute} = acute Hazard Index

$C_{\leq 24h}$ = ground level concentration predicted over an averaging period of typically $\leq$ 24-hours, matching the averaging time of the health protective guideline for compound ($\mu g/m^3$)

Gdl_{Acute} = acute health protective guideline for compound ($\mu g/m^3$)

$HI_{Chronic}$ = chronic Hazard Index

C_{Annual} = annual average ground level concentration predicted for compound ($\mu g/m^3$)

$Gdl_{Chronic}$ = chronic health protective guideline for compound ($\mu g/m^3$)

For the HRA the acute air concentration used to calculate the acute HI has been based upon the 99.9th percentile (i.e. 9th highest) 1-hour and 99.5th percentile (i.e. 2nd highest) 24-hour average ground level concentrations predicted by the air dispersion modelling. In addition, acute HIs have also been calculated from the 99.5th percentile (i.e. 44th highest) 1-hour average and 95th percentile (i.e. 18th highest) 24-hour average ground level concentrations predicted from the air dispersion modelling, representing a more realistic, yet still conservative estimate of actual acute exposures.

The general rule of thumb for interpreting the HI (Toxikos, 2003) is that:

- values less than one represent no cause for concern;
- values greater than one but less than 10 generally do not represent cause for concern because of the inherent conservatism embedded in the exposure and toxicity assessments; and
- values greater than ten may present some concern with respect to possible health effects.

The carcinogenic risk provides an indication of the incremental probability that an individual will develop cancer over a lifetime as a direct result of exposure to potential carcinogens, and is expressed as a unitless probability. The ICR for individual compounds is summed to calculate the potential total ICR from exposure to multiple compounds (Equation 4).

$$Risk = \sum_1^i C_{i\,Annual} \times \frac{EF \times ED}{AT} \times UR_i = \sum_1^i C_{i\,Annual} \times UR \qquad \textbf{Equation 4}$$

Where:

$Risk$	= lifetime incremental total cancer risk
C_{Annual}	= annual average ground level concentration for compound (µg/m^3)
EF	= exposure frequency (365 days/year)
ED	= exposure duration (70 years)
AT	= averaging time (365 days/year x 70 years, or 25,550 days)
UR_i	= Unit Risk factor for compound (per µg/m^3)

The incremental carcinogenic risk that is considered acceptable varies amongst jurisdictions, typically ranging from one in a million ($1x10^{-6}$) to one in ten thousand ($1x10^{-4}$). The most stringent criterion of one in a million represents the USEPA's *de minimis*, or essentially negligible incremental risk level, and has therefore been adopted for this screening assessment as a conservative (i.e. health protective) indicator of acceptable carcinogenic risk.

5.2 Acute Non-Carcinogenic Effects

Acute HIs have been calculated for the baseline and expanded Wagerup refinery emission scenarios and are presented as Figures 2a, 2b and 2c for the base case and the two expansion scenarios calculated using the predicted 99.9th percentile 1-hour and 99.5th percentile 24-hour ground level concentrations. Figures 3a, 3b and 3c present the Acute HIs for the base case and the two upgrade scenarios calculated using the predicted 99.5th percentile 1-hour and 95th percentile 24-hour ground level concentrations. Figures 4a and 4b present the percentage contribution that the predicted PM_{10} concentrations make to the overall acute HIs for the existing and upgraded refinery emission scenarios (note that Upgrade Case 6 and Upgrade Case 7 have essentially the same predicted impacts arising from the PM_{10} emissions). These figures show the significance of the particulate emissions from the RDA and bauxite stockpile areas on the overall acute HIs, particularly in close proximity to these sources.

The Acute HIs calculated for each of the receptor locations are presented in Table 7 along with the relative change associated with the Wagerup refinery upgrade scenarios compared to the baseline. Receptors 7 and 16 are predicted to have the highest acute HI with receptors 12 and 13 predicted to have the lowest.

The maximum acute HIs for the baseline and the two upgrade emission scenarios presented in Table 7 are less than one, indicating no cause for concern based on the predicted ground level concentrations, the health protective guidelines used and the compounds considered. The maximum acute HI for either of the expanded Wagerup refinery emission scenarios is predicted to occur at Receptor 7 and is approximately 80% of the acceptable threshold of one. The acute HI at Receptor 7 is strongly influenced by the particulate emissions from the RDA (see Figure 4) and proposed improvements to the dust management measures employed at the RDA are predicted to result in a decrease in the acute HI calculated at this receptor based on the 99.9th percentile 1-hour average concentration and the 99.5th percentile 24-hour average ground level concentrations predicted by the modelling.

Table7: Summary of Acute Hazard Indices

Acute HI					
		Upgrade Case 6		Upgrade Case 7	
Receptor No	Base Case HI	HI	Change from Base Case (%)	HI	Change from Base Case (%)
Based on the 99.9th Percentile 1-Hour and 99.5th Percentile 24-hr Predicted Ground Level Concentrations					
1	0.3248	0.2917	-10.2%	0.3059	-5.8%
2	0.3861	0.3314	-14.2%	0.3433	-11.1%
3	0.3155	0.3167	0.4%	0.3326	5.4%
4	0.4028	0.3974	-1.3%	0.4131	2.6%
5	0.3001	0.2948	-1.8%	0.3133	4.4%
6	0.3375	0.3118	-7.6%	0.3240	-4.0%
7	0.8997	0.8128	-9.7%	0.8049	-10.5%
8	0.2759	0.3121	13.1%	0.3141	13.9%
9	0.3045	0.3038	-0.3%	0.3137	3.0%
10	0.3582	0.3709	3.5%	0.3749	4.6%
11	0.4385	0.4902	11.8%	0.4928	12.4%
12	0.1757	0.2004	14.0%	0.2038	16.0%
13	0.1793	0.2077	15.8%	0.2044	14.0%
14	0.3648	0.4188	14.8%	0.4287	17.5%
15	0.4703	0.4439	-5.6%	0.4653	-1.1%
16	0.5060	0.5959	17.8%	0.6139	21.3%
Based on the 99.5th Percentile 1-Hour and 95th Percentile 24-hr Predicted Ground Level Concentrations					
1	0.1894	0.1938	2.3%	0.1980	4.5%
2	0.2112	0.1938	-8.3%	0.2020	-4.4%
3	0.1615	0.1583	-2.0%	0.1628	0.8%
4	0.2103	0.2122	0.9%	0.2140	1.7%
5	0.1568	0.1527	-2.7%	0.1571	0.2%
6	0.1869	0.1808	-3.3%	0.1901	1.7%
7	0.3264	0.3422	4.8%	0.3413	4.6%
8	0.1413	0.1532	8.4%	0.1523	7.7%
9	0.1831	0.2092	14.2%	0.2082	13.7%
10	0.1806	0.2186	21.1%	0.2146	18.9%
11	0.2675	0.3246	21.3%	0.3235	20.9%
12	0.0831	0.1019	22.6%	0.0998	20.1%
13	0.1050	0.1205	14.8%	0.1206	14.9%
14	0.2264	0.2470	9.1%	0.2517	11.2%
15	0.2789	0.2780	-0.3%	0.2867	2.8%
16	0.3252	0.3981	22.4%	0.4009	23.3%

Note:

1. The 99.9th percentile 1-hour average concentration is derived from the 9th highest 1-hour average predicted ground level concentration. The 99.5th percentile 24-hour average concentration is derived from the 2nd highest 24-hour average predicted ground level concentration.
2. The 99.5th percentile 1-hour average concentration is derived from the 44th highest 1-hour average predicted ground level concentration. The 95th percentile 24-hour average concentration is derived from the 18th highest 24-hour average predicted ground level concentration.
3. The Upgrade Case 6 emission scenario includes cogeneration units while the Upgrade Case 7 emission scenario includes additional boilers.

Table 7 shows that the upgrade scenarios are predicted to result in both decreases and increase in the acute HI's depending upon their location. Receptors to the southwest of the refinery are predicted to experience a decrease in the acute HIs, primarily due to the proposed development of the RDA from the northeast of the existing facility. The expansion of the RDA is also predicted to contribute to the predicted increase in the acute HI at receptor 8, although the maximum acute HI presented in Table 7 for this location is less than one third of the acceptable threshold of one.

The data presented in Table 7 also indicates that Upgrade Case 6 scenario is generally expected to result in the prediction of lower HIs than the Upgrade Case 7 scenario as a result of the enhanced dispersion of the emissions from the cogeneration units (Upgrade Case 6) compared to the boilers (Upgrade Case 7) although neither scenario results in acute HIs above the acceptable threshold of one.

5.3 Chronic Non-Carcinogenic Effects

Chronic HIs have been calculated for the baseline and expanded Wagerup refinery emission scenarios and are presented in Table 8 and as Figures 5a, 5b and 5c respectively. The maximum chronic HI is predicted to occur at Receptor 16 and that this (maximum of 0.047 for the Upgrade Case 6 scenario) is well below the acceptable threshold of one, indicating no cause for concern.

Table 8 also indicates that the expansion of the Wagerup refinery is generally predicted to result in increases in the chronic HI's and although the percentage increases are relatively large (up to 38.7%) the absolute magnitude of these changes is low being less than 0.01, or one hundredth of the acceptable threshold of one.

Table 8: Summary of Chronic Hazard Indices

Receptor No	Chronic HI				
	Base Case HI	Upgrade Case 6		Upgrade Case 7	
		HI	Change from Base Case (%)	HI	Change from Base Case (%)
1	0.01394	0.01422	2.1%	0.01495	7.3%
2	0.01442	0.01366	-5.3%	0.01369	-5.1%
3	0.00986	0.01050	6.4%	0.01050	6.4%
4	0.01408	0.01599	13.6%	0.01599	13.5%
5	0.00902	0.00969	7.4%	0.00969	7.4%
6	0.01268	0.01172	-7.6%	0.01212	-4.4%
7	0.01169	0.01215	3.9%	0.01136	-2.8%
8	0.00876	0.01005	14.7%	0.00996	13.7%
9	0.01513	0.01723	13.9%	0.01703	12.6%
10	0.01300	0.01802	38.7%	0.01702	31.0%
11	0.02315	0.03135	35.4%	0.03034	31.1%
12	0.00611	0.00811	32.8%	0.00797	30.5%
13	0.00699	0.00900	28.7%	0.00846	21.0%
14	0.01735	0.01995	15.0%	0.01946	12.2%
15	0.01879	0.01704	-9.3%	0.01767	-5.9%
16	0.03751	0.04729	26.1%	0.04717	25.7%

5.4 Carcinogenic Effects

The incremental carcinogenic risk (ICR) has been calculated for the baseline and expanded Wagerup refinery emission scenarios and the results are presented in Table 9 and Figures 6a, 6b and 6c respectively. These figures indicate that the highest incremental carcinogenic risks are predicted to occur in the immediate vicinity of the refinery and the RDA. Receptor 16, located near the refinery and the RDA, is predicted to experience the highest ICR with the maximum of 0.632×10^{-6} predicted for the Upgrade Case 7 scenario which is below the USEPA's *de minimis* threshold of one in a million (i.e. 1×10^{-6}). Arsenic emissions from the refinery are predicted to be one of the major contributors to the calculated ICR and the increase in the ICRs predicted for the upgrade scenarios.

The expression of the incremental carcinogenic risk values presented in Table 9 are best explained by way of example, with the incremental carcinogenic risk calculated for Receptor 16 for the baseline emissions scenario of 3.68×10^{-7} (0.000000368) which can also be interpreted as a risk of 1 in 2,717,391.

An increase in the incremental carcinogenic risk compared to the baseline incremental carcinogenic risk is predicted to result form the Wagerup refinery expansion at all receptor locations, with an increase in the incremental carcinogenic risk ranging from approximately 33% (Receptor 2, Upgrade Case 6) to 160% (Receptor 15, Upgrade Case 7). However, while the predicted percentage increases in the ICRs is significant, the absolute maximum increase at any of the receptors is 0.26×10^{-6} at Receptor 16, the closest receptor to the refinery and the RDA.

Table 9: Summary of Incremental Carcinogenic Risk

Receptor No	Incremental Carcinogenic Risk (ICR)				
	Base Case ICR	Upgrade Case 6		Upgrade Case 7	
		ICR	Change from Base Case (%)	ICR	Change from Base Case (%)
1	9.16E-08	1.28E-07	40.2%	1.79E-07	95.9%
2	7.41E-08	9.85E-08	32.9%	1.58E-07	113.2%
3	6.37E-08	8.76E-08	37.5%	1.26E-07	98.2%
4	1.04E-07	1.43E-07	38.1%	1.92E-07	84.7%
5	5.27E-08	7.85E-08	49.0%	1.16E-07	120.2%
6	6.05E-08	8.51E-08	40.6%	1.48E-07	144.3%
7	1.21E-07	1.63E-07	34.4%	1.97E-07	62.7%
8	6.22E-08	9.43E-08	51.5%	1.26E-07	102.0%
9	1.11E-07	1.63E-07	46.8%	2.16E-07	94.8%
10	1.05E-07	1.51E-07	43.8%	2.05E-07	94.5%
11	2.06E-07	3E-07	45.4%	3.81E-07	84.9%
12	4.67E-08	6.74E-08	44.1%	9.15E-08	95.8%
13	4.44E-08	6.27E-08	41.1%	9.33E-08	110.2%
14	9.26E-08	1.38E-07	48.6%	2.21E-07	139.2%
15	7.66E-08	1.08E-07	41.6%	1.99E-07	160.1%
16	3.68E-07	5.29E-07	43.5%	6.32E-07	71.8%

The data presented in Table 9 also indicates that the increases in the predicted ICR for the Upgrade Case 6 scenario are considerably less than those predicted for the Upgrade Case 7 scenario. This is primarily due to the increase in the arsenic emissions estimated to occur from the new boilers (boilers 4 & 5) whereas the proposed cogeneration units are not predicted to have any arsenic emissions.

5.5 Irritancy

For the purposes of this screening assessment irritancy refers to a direct physiological response arising from short-term exposure to a compound that may result in mild, transient adverse health effects that are reversible upon cessation of exposure. The health reference values used in the health risk assessment are derived from information on the most sensitive toxicological endpoint. In a number of cases the end point is irritancy (see Appendix A). In cases where the most sensitive, critical end point is not irritancy, the reference value derived is also protective of irritancy. The HI for acute effects, for both the baseline and expanded Wagerup refinery emission scenarios, are less than one. This indicates that the risk for emissions from the existing or expanded Wagerup refinery to cause irritation is very low.

5.6 Uncertainties Associated with Calculated Risks

The risk assessment process relies on a set of assumptions and estimates with varying degrees of certainty and variability. Major sources of uncertainty in risk assessment include:

- natural variability (*e.g.*, differences in body weight in a population);
- lack of knowledge about basic physical, chemical, and biological properties and processes;
- assumptions in the models used to estimate key inputs (*e.g.*, air dispersion modelling, dose-response models); and
- measurement error (e.g., used to characterise emissions).

Perhaps the greatest single source of uncertainty in risk assessment is the chemicals' dose-response relationships, particularly carcinogenic unit risks.

For this HRA, uniformly conservative assumptions have been used to ensure that potential exposures and associated health risks are over- rather than under-estimated. As a result of the compounding of conservatism, the quantitative risk indicators are considered to be upper-bound estimates, with the actual risk likely to be lower.

5.6.1 Emissions Characterisation and Quantification Uncertainty

There is uncertainty associated with the identification and quantification of atmospheric emissions from the Wagerup refinery and to a greater extent with the RDA emissions. The extent of these uncertainties is discussed in ENVIRON (2005b).

The HRA included 27 individual or groups of compounds which is a relatively small subset of the compounds identified as being emitted from one or all of Alcoa's WA refineries. However, the 27 individual compounds or groups of compounds that were considered in this assessment were found to contribute over 93% of the acute HI, over 86% of the chronic HI, and 100% of the incremental carcinogenic risk calculated for the Pinjarra Refinery Efficiency Upgrade health risk evaluation at the maximally affected receptor (receptor 1) (Toxikos, 2003). Based on the findings of the Pinjarra Refinery Efficiency Upgrade health risk evaluation, the compounds considered in the Wagerup refinery screening assessment are expected to contribute the vast majority of the potential health risks.

5.6.2 Estimation of Exposure Concentration Uncertainty

The air dispersion modelling was completed by CSIRO using TAPM (for the Refinery) and Air Assessments using the CALPUFF model (for the RDA). The separate model outputs were then combined for each pollutant for each hour of the year and for each grid point modelled to provide the predicted concentrations of the 27 compounds in ambient air at each model grid point. The modelling was conducted using a 13 km by 13 km grid with a grid interval of 250 m. Detailed modelling reports have been prepared by the CSIRO (CSIRO, 2005a, 2005b) and Air Assessments (2005) and while these reports have not been reviewed as part of this HRA, they will be independently peer reviewed.

The acute HIs were calculated for two statistical combinations of the predicted 1-hour and 24-hour average concentrations as follows:

1. the predicted 99.9th percentile (i.e. 9th highest) 1-hour and the 99.5th percentile (i.e. 2nd highest) 24-hour average ground level concentrations; and
2. the predicted 99.5th percentile (i.e. 44th highest) 1-hour and the 95th percentile (i.e. 18th highest) 24-hour average ground level concentrations.

In interpreting the air dispersion modelling results, the CSIRO (2005b) noted that the 9th highest concentration (99.9th percentile) is often chosen as the key statistic to represent the extremes, rather than the modelled maximum and on this basis, the 99.9th percentile has been selected as the upper bound for the calculation of the acute HIs across the model domain. The 99.5th percentile 1-hour average concentration represents the predicted concentration that is only predicted to be exceeded for 0.5% of the time. Therefore, for the vast majority of the year, the potential acute health effects are expected to be less significant than the calculated acute HIs presented in this report for the two statistical combinations considered.

Stack testing results for dioxins and furans emissions from the Wagerup refinery have returned results below detection limit, and therefore the emissions for this class of compounds have been set to zero.

5.6.3 Exposure Assumptions Uncertainty

To calculate the incremental carcinogenic risk it has been assumed that residences located at the key receptor locations spend every hour of every day outdoors at that location for 70 years. Clearly, these exposure conditions are unlikely to be realised, with the actual exposure concentration resulting from the refinery and RDA emissions is typically expected to be lower in the indoor environment than that experienced in the outdoor air, and the exposure frequency (i.e. days per year) and exposure duration (years) likely to be considerably lower as people move about.

The HRA has been confined to exposure via the inhalation pathway. There is therefore a potential that total exposure to specific compounds has been underestimated. Exposure to compounds can occur via direct and indirect exposures, defined as follows:

Direct exposure: when exposure to a chemical occurs in the media in which it is released from the source. For an atmospheric emission source direct exposure occurs via inhalation.

Indirect exposure: when exposure to a chemical occurs after it has crossed into a different media. For an atmospheric emission source indirect exposure may occur, for example, as a result of deposition of the chemicals onto soils from which home grown vegetables are consumed

In most circumstances direct exposure (i.e. inhalation) is expected to represent the most significant exposure route for atmospheric emission sources. However exceptions do occur, most notably if the chemicals tend to bioaccumulate, or are particularly persistent and hence do not break-down readily in the environment. The compounds tending towards the particulate phase have been identified as the most likely candidates for multi-pathway exposure as they will tend to deposit on to the surfaces (e.g. soil and crops) and be available for ingestion.

Compounds considered in this HRA that are likely to require multi-pathway exposure assessment (refer to Section 3.3) include:

- arsenic;
- cadmium;
- chromium (VI);
- nickel;

- mercury; and
- PAHs (with three rings or greater).

To assist with the assessment of multi-pathway exposure assessments, a software program, the Hot Spots Analysis and Reporting Program (HARP), has been developed in consultation with various Californian environmental agencies. The HARP has been applied for this assessment; however the analysis has been confined to the following indirect exposure pathways:

- Soil ingestion;
- Dermal;
- Vegetable ingestion; and
- Water ingestion.

The remaining pathways were either not listed as applicable to the relevant trace metals (i.e. breast milk ingestion), or were considered unlikely to be a significant exposure route based on the very low default values for the percent of a person's consumption obtained from home grown produce (i.e. home grown meat, milk and eggs).

ENVIRON (2004) applied the HARP program as part of an assessment of the Pinjarra RDA and found that exposure by other than inhalation pathways was likely to be significant for arsenic and cadmium. Cadmium contributes to the overall chronic HI (maximum hazard quotant of 0.00026 across the model domain) and as the maximum chronic HI is so small, exposures via dermal absorption and ingestion will not make an appreciable difference to the overall chronic HI. However, the results presented in Section 5.4 indicated that arsenic exposure via inhalation is the major contributor to the predicted ICR and as such this requires further evaluation. The HARP program was utilised assuming a particulate deposition velocity of 0.003 m/s (approximately an order of magnitude greater than recommended in the CALPUFF user manual for fine particulate emission) and this indicated that the inhalation exposure pathway was likely to account for approximately 75% of the carcinogenic exposure to arsenic. The remaining 25% of the exposure was predicted to occur as a result of soil ingestion (14%), vegetable ingestion (8%), dermal absorption (2%) and drinking water (1%). It should be noted that there is a great deal of uncertainty associated with this and that the assumptions inherent in the HARP are designed to err on the side of health protection in order to avoid underestimation of risk to the public (OEHHA, 2003). Further, there is a great deal of uncertainty and actual variability in much of the data used for this assessment (e.g. amount of local vegetables produce consumed; particle size distribution of particulate containing arsenic). Therefore, the potential alternative exposure pathways presented above should be considered as broadly

indicative only. At the maximally affected receptor (ie. Receptor 16, Upgrade Case 7), the ICR attributable to arsenic via inhalation exposure was $0.28x10^{-6}$. Assuming that this accounts for 75% of the potential ICR attributable to arsenic, then the potential total ICR associated with arsenic would be approximately $0.37x10^{-6}$ and the total ICR for all compounds would increase from $0.63x10^{-6}$ to $0.72x10^{-6}$ at this location which is less than the reference value of 1×10^{-6}. Therefore, the alternative exposure pathways for arsenic, are not expected to have a significant impact at the maximally affected receptor (i.e. Receptor 16) and will have a lower level of impacts at the less affected receptors..

5.6.4 Toxicity Assessment Uncertainty

The primary uncertainties associated with the toxicity assessment are related to the derivation of the health protective guidelines. Health protective guidelines published by reputable authorities have been applied within this assessment have been derived by applying various conservative (i.e. health protective) assumptions. The extrapolation of animal bioassay results or occupational exposure studies to human risk at much lower levels of exposure involves a number of assumptions regarding effect threshold, interspecies extrapolation, high- to low-dose extrapolation, and route-to-route extrapolation. The scientific validity of these assumptions is uncertain; because each of the individual extrapolations are intended to prevent underestimation of risk, in concert they result in unquantifiable but potentially very significant overestimation of risk.

5.6.5 Risk Characterisation Uncertainty

It should be noted that the summing of the quantitative risk indicators for individual compounds to calculate the overall risk from exposure to multiple compounds does not take into account that different compounds can target different organs and therefore the potential health risk arising from exposure to multiple compounds is not necessarily additive, nor does it account for potential antagonistic or synergistic effects. However, the additive approach is considered to be conservative (i.e. health protective) in most circumstances.

6. SUMMARY

ENVIRON has conducted a HRA of the potential health risks arising from atmospheric emissions emitted from the Wagerup refinery, considering the potential risks associated with a baseline (i.e. representative of emissions from the existing refinery operating at an alumina production rate of 2.41 Mtpa) and expanded (i.e. representative of emissions from an expanded refinery operating at an alumina production rate of 4.7 Mtpa) emissions scenarios.

Quantitative health risk indicators were calculated for exposure via the inhalation pathway to atmospheric emissions from the Wagerup refinery in isolation, and therefore did not take into account the alternative exposure pathways (e.g. ingestion, dermal absorption), nor other sources of atmospheric emissions of these compounds. Of the pollutants considered in this HRA, only cadmium (chronic non-carcinogenic HI) and arsenic (incremental carcinogenic risk) were assessed as requiring further assessment based on the results of the HARP developed by Californian environmental agencies. This subsequent assessment indicated that non inhalation exposure pathways for these substances did not result in any unacceptable impacts.

The following quantitative health risk indicators were calculated across the model domain and for key receptors located in the vicinity of the Wagerup refinery:

- acute HI;
- chronic HI; and
- ICR.

Based upon the results of the health screening assessment it can be concluded that:

- the potential for emissions from the existing or expanded Wagerup refinery to cause acute health effects is low and is primarily driven by the particulate emissions from the RDA and oxides of nitrogen emissions from the refinery;

- the potential for emissions from the existing or expanded Wagerup refinery to cause chronic non-carcinogenic health effects is very low; and

- the potential for emissions from the existing or expanded Wagerup refinery to contribute to the incidence of cancer based on inhalation exposure is below USEPA *de minimis* threshold of one in a million (i.e. 1 x 10^{-6}) at all of the residential receptors considered;

As with any risk evaluation, there are areas of uncertainty in this assessment. To ensure that potential risks are not underestimated, uniformly conservative assumptions have been used to characterize exposure and toxicity. Due to the resultant compounding of conservatism, the quantitative risk indicators should be considered as over-estimates of potential health risks associated with emissions from the Wagerup refinery.

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FIGURES

Figure 1

Residential Receptors Identified by Alcoa for the HRA

Client: Alcoa	ENVIRON	
Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

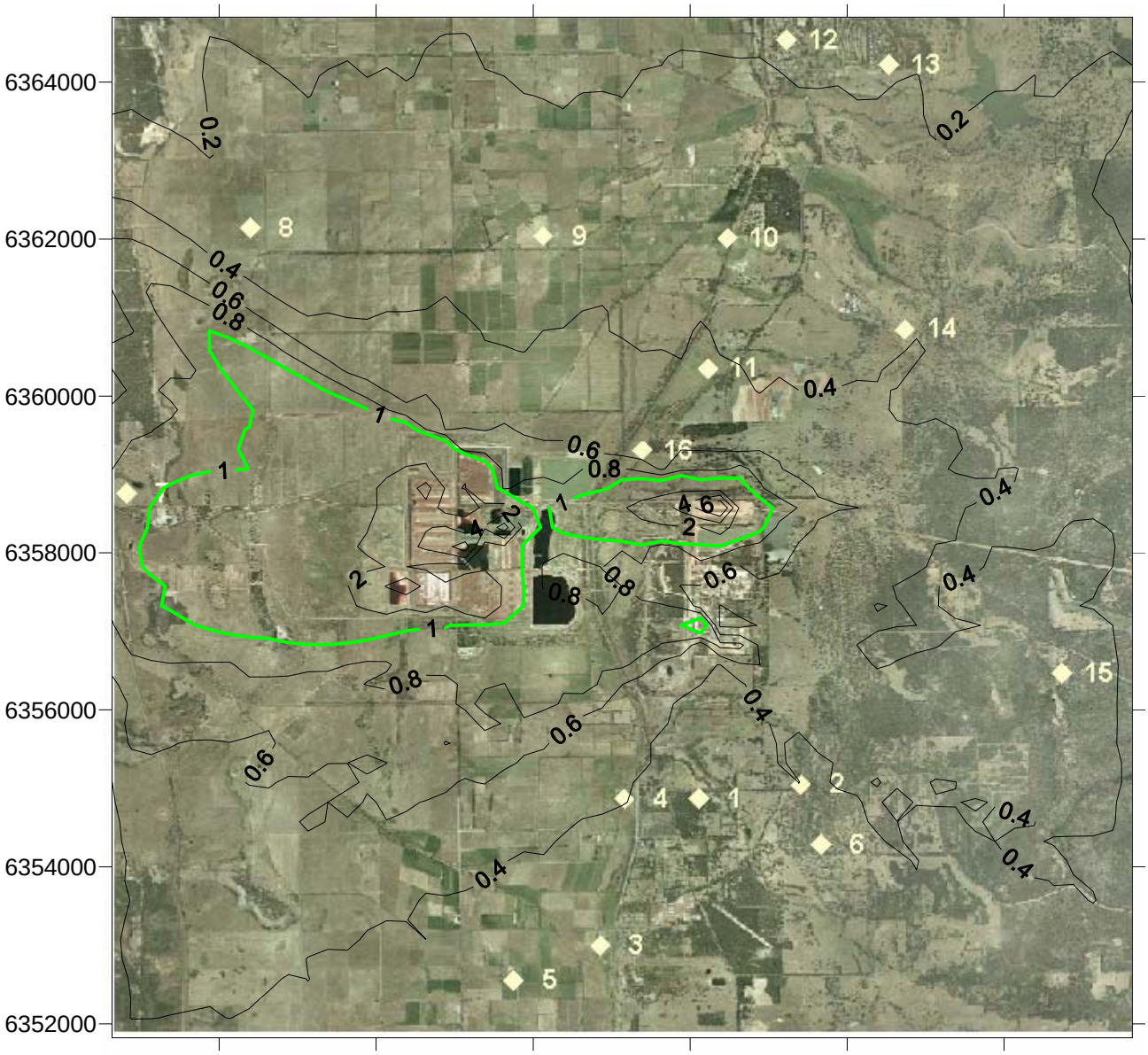

Figure 2a
Acute HI – Base Case
99.9th 1-hour and 99.5th 24-hour percentiles

Client: Alcoa	ENVIRON	
Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

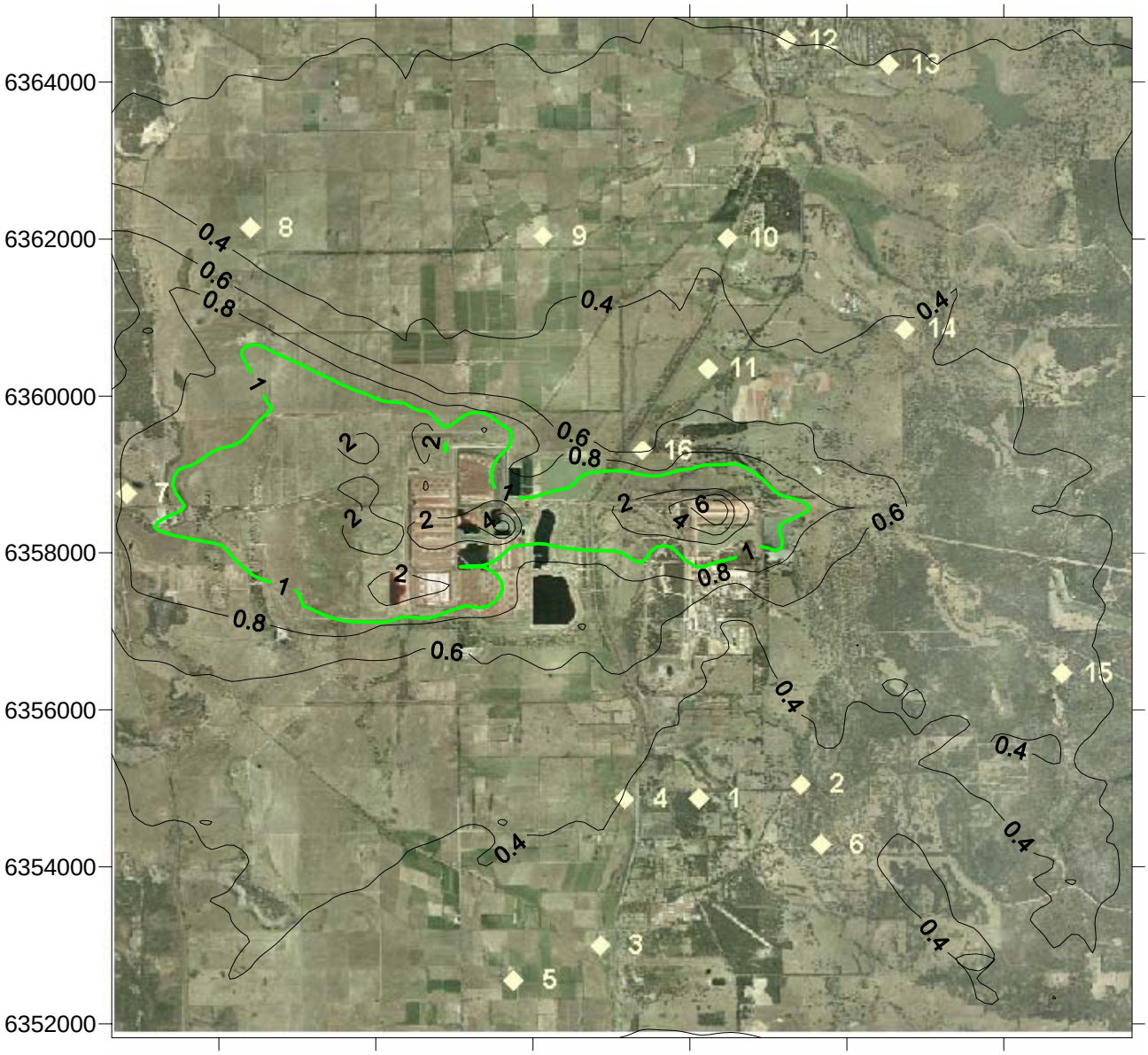

Figure 2b
Acute HI –Upgrade Case 6 (Cogeneration)
99.9th 1-hour and 99.5th 24-hour percentiles

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Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

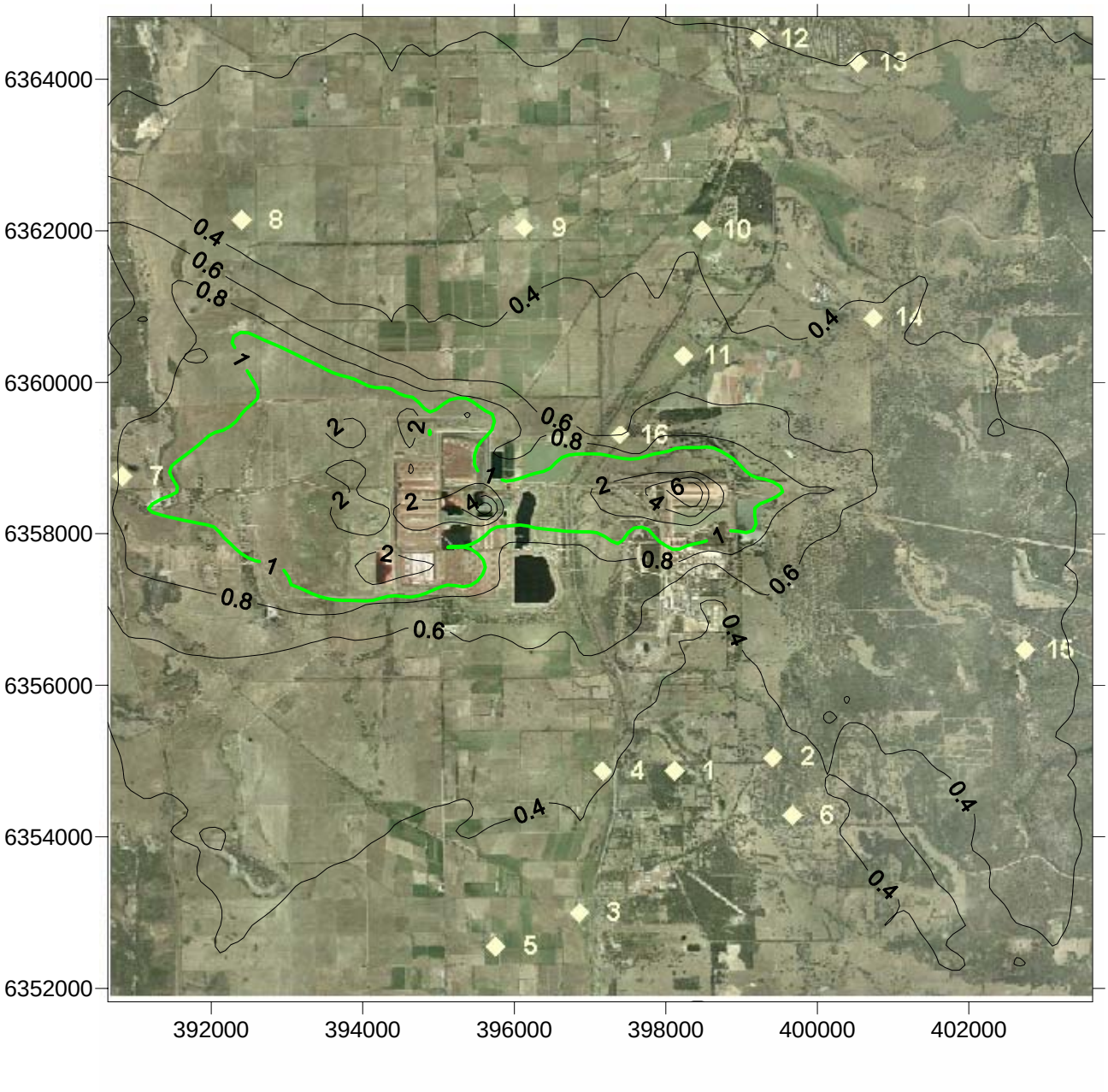

Figure 2c
Acute HI – Upgrade Case 7 (Boilers)
99.9th 1-hour and 99.5th 24-hour percentiles

Client: Alcoa	ENVIRON	
Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

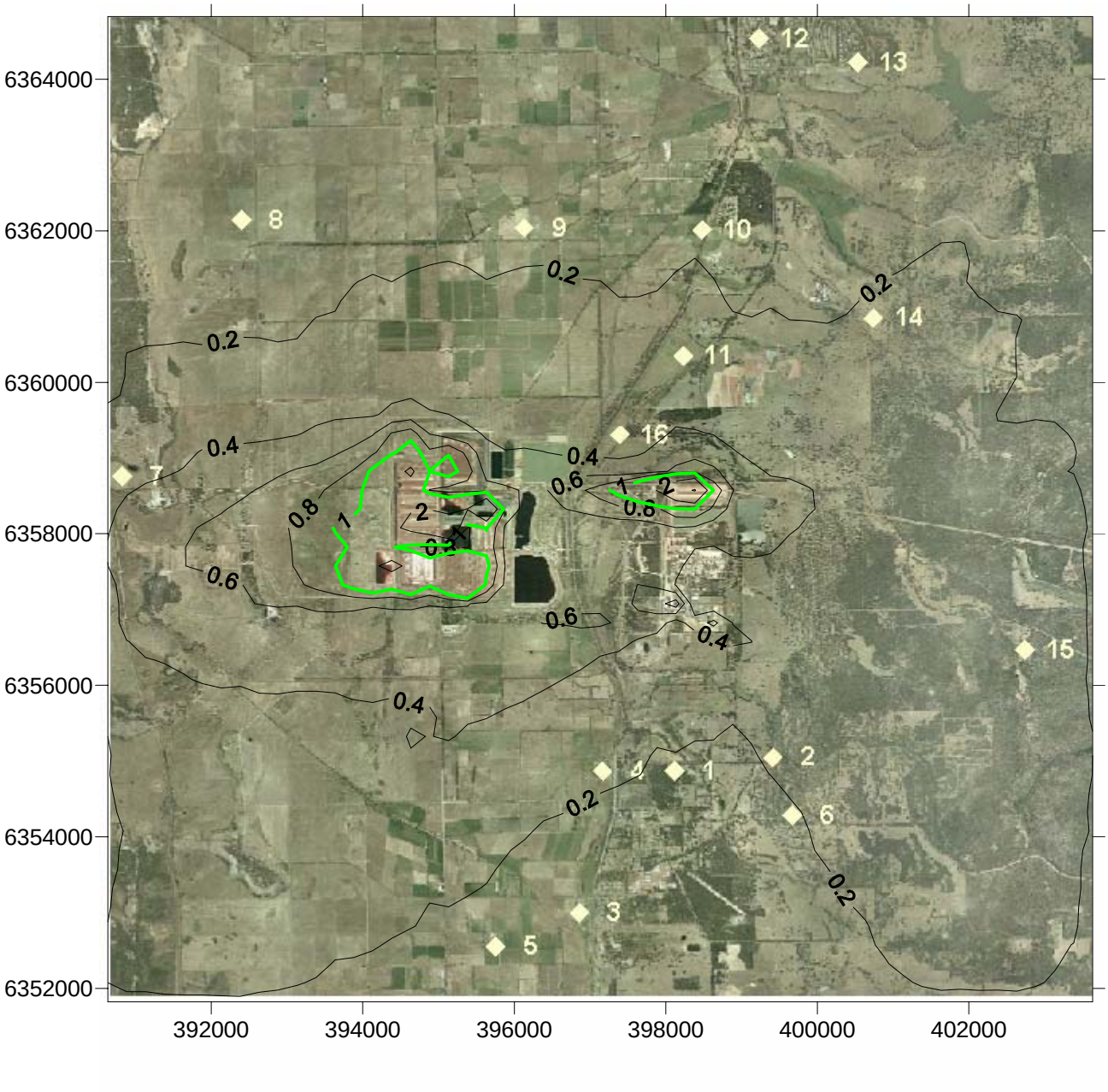

Figure 3a
Acute HI – Base Case
99.5th 1-hour and 95th 24-hour percentiles

Client: Alcoa	ENVIRON	
Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

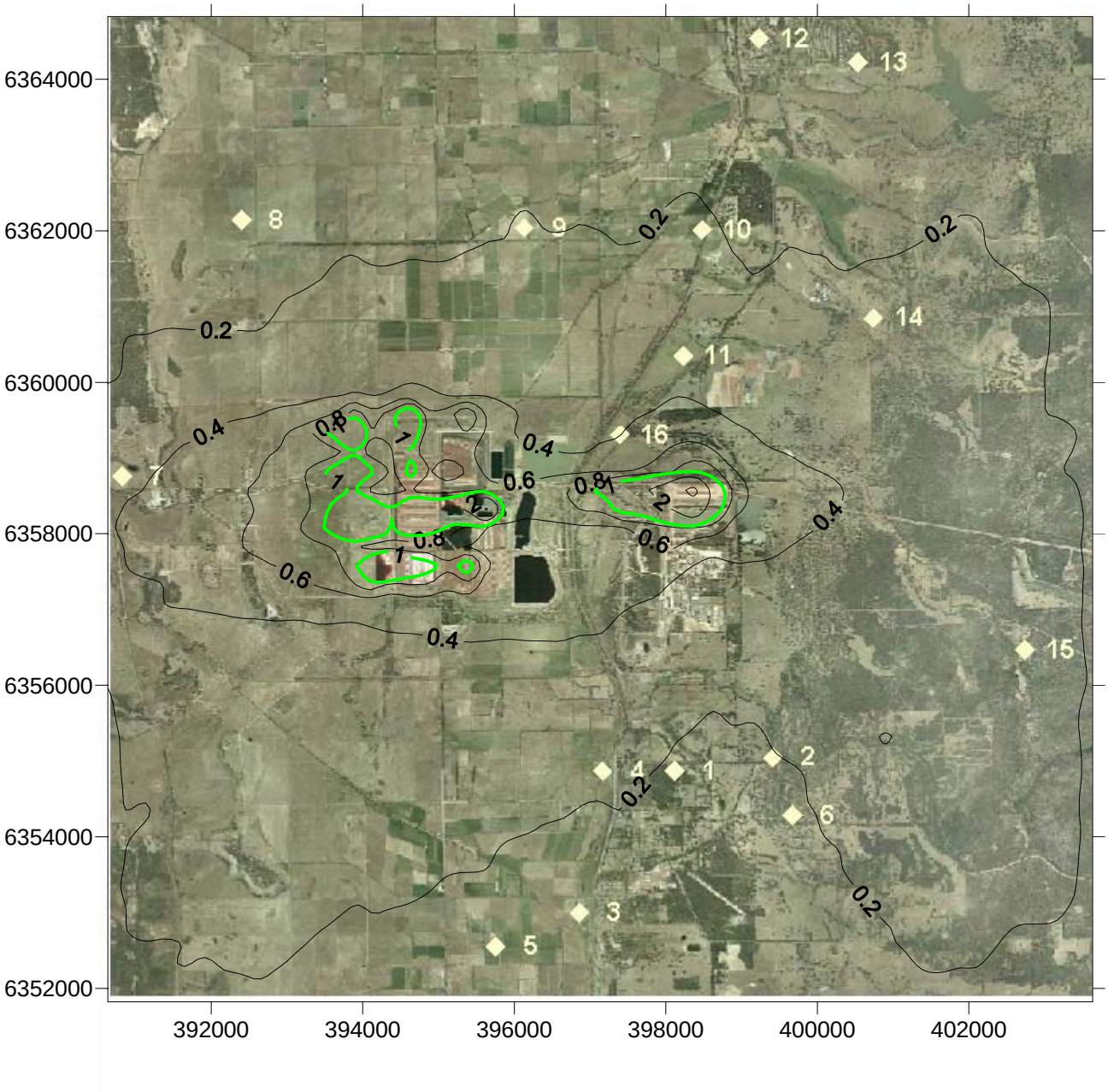

Figure 3b
Acute HI –Upgrade Case 6 (Cogeneration)
99.5th 1-hour and 95th 24-hour percentiles

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Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

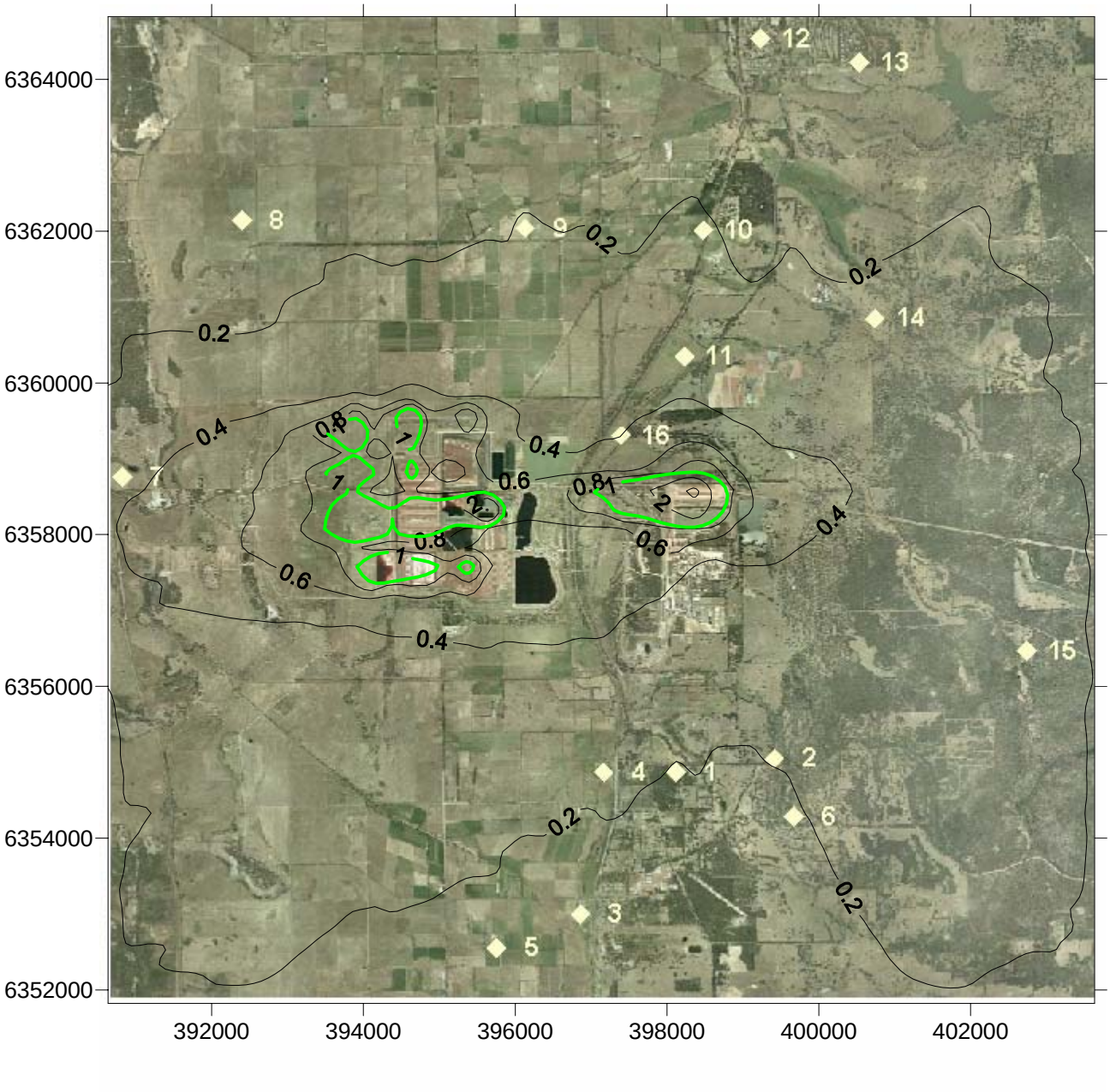

Figure 3c
Acute HI – Upgrade Case 7 (Boilers)
99.5th 1-hour and 95th 24-hour percentiles

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Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

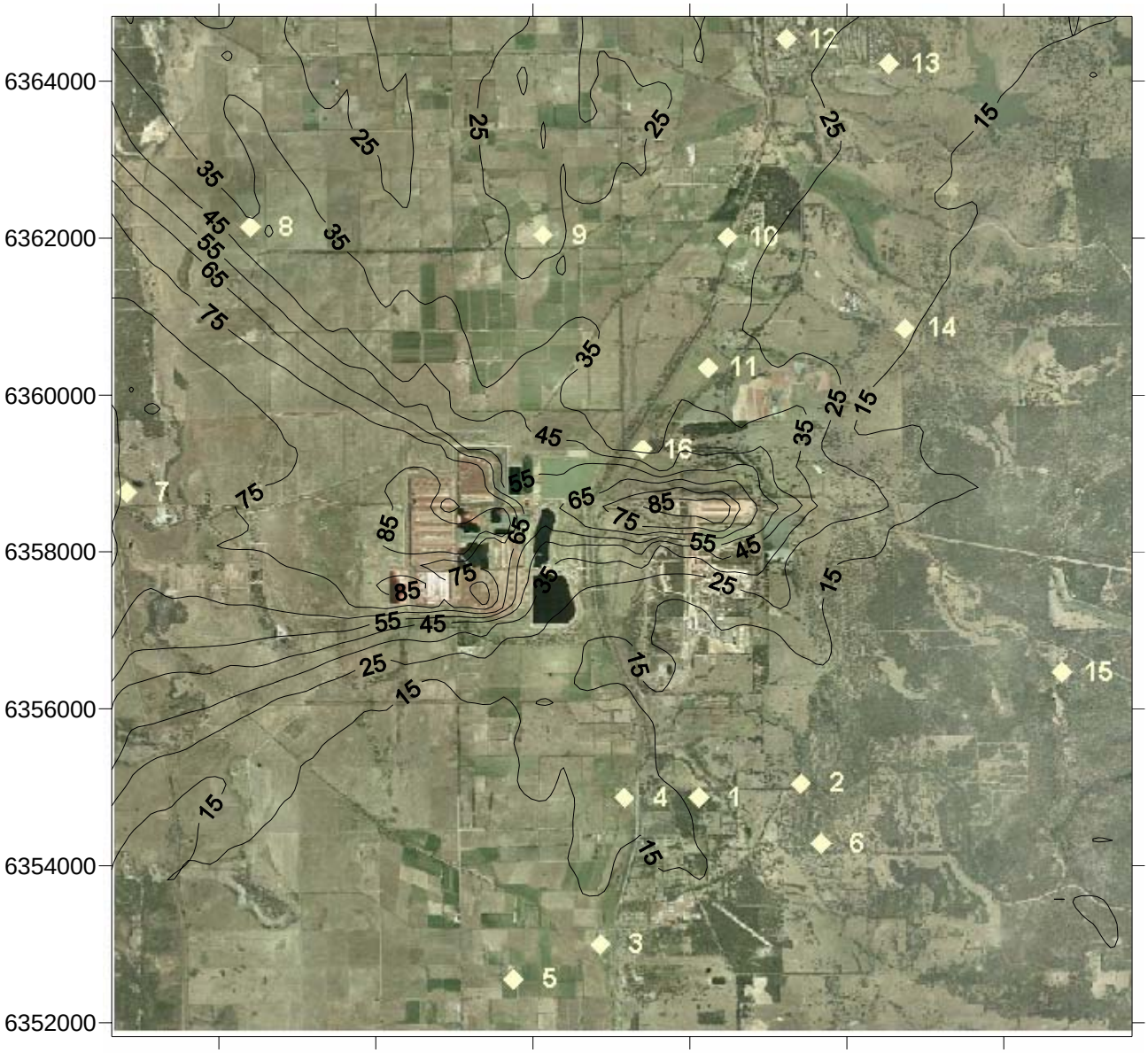

Figure 4a
Percentage Contribution of PM_{10}
Acute HI – Base Case
99.5th 1-hour and 95th 24-hour percentiles

Client: Alcoa	ENVIRON	
Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

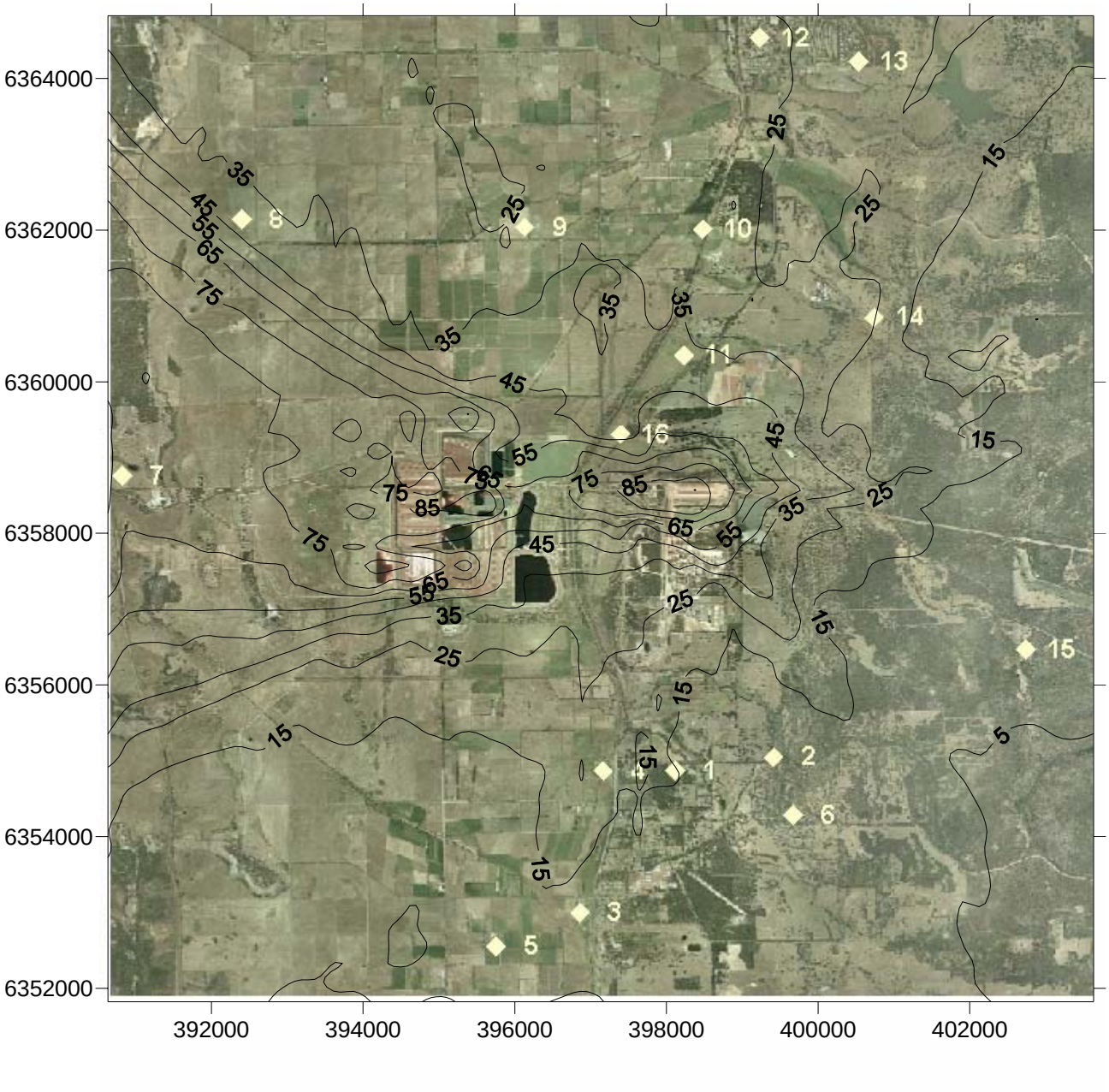

Figure 4b
Percentage Contribution of PM_{10}
Acute HI – Upgrade Cases
99.5th 1-hour and 95th 24-hour percentiles

Client: Alcoa	ENVIRON	
Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

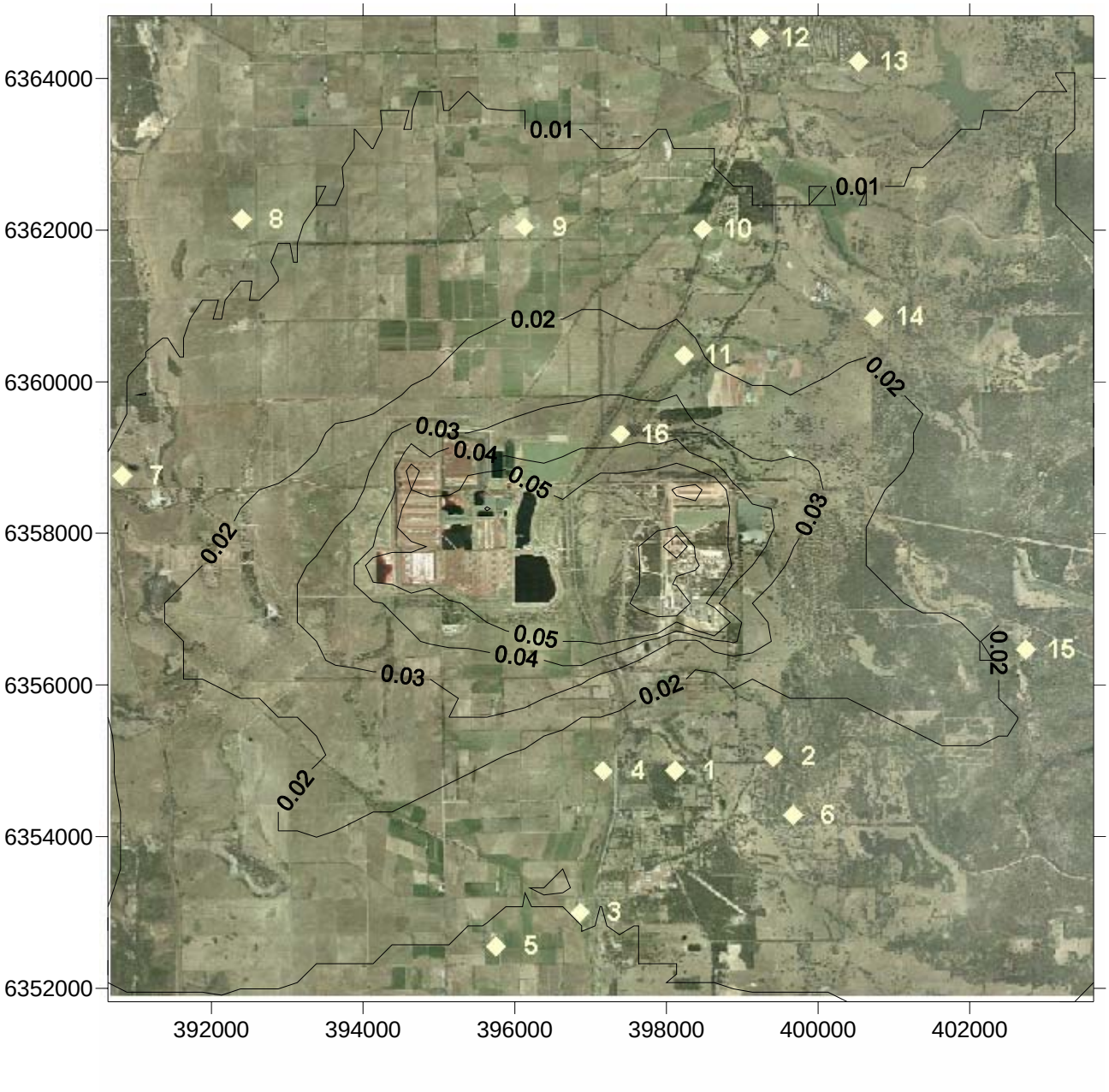

Figure 5a
Chronic HI – Base Case

Client: Alcoa	ENVIRON	
Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

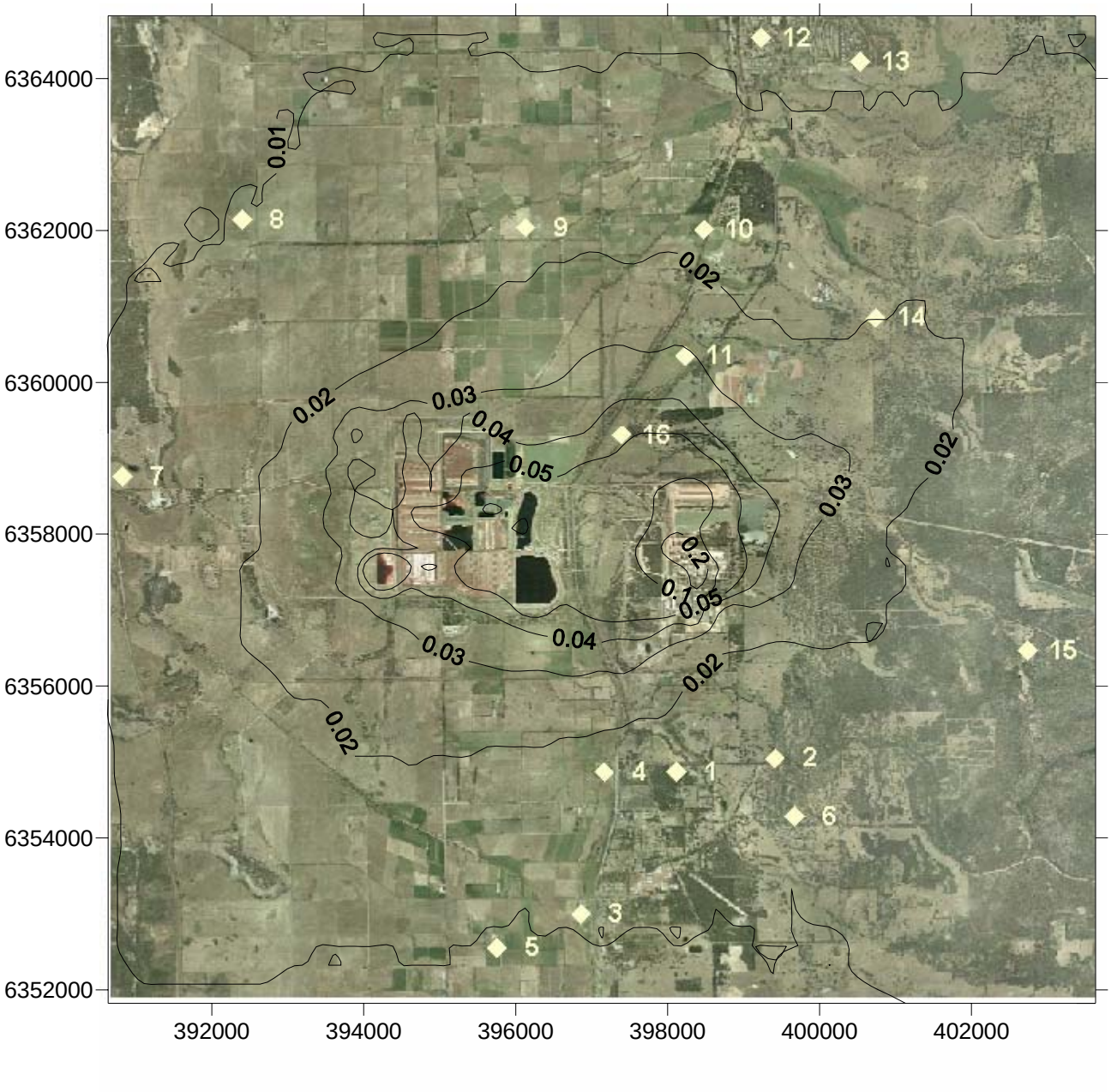

Figure 5b
Chronic HI –Upgrade Case 6 (Cogeneration)

Client: Alcoa	ENVIRON	
Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

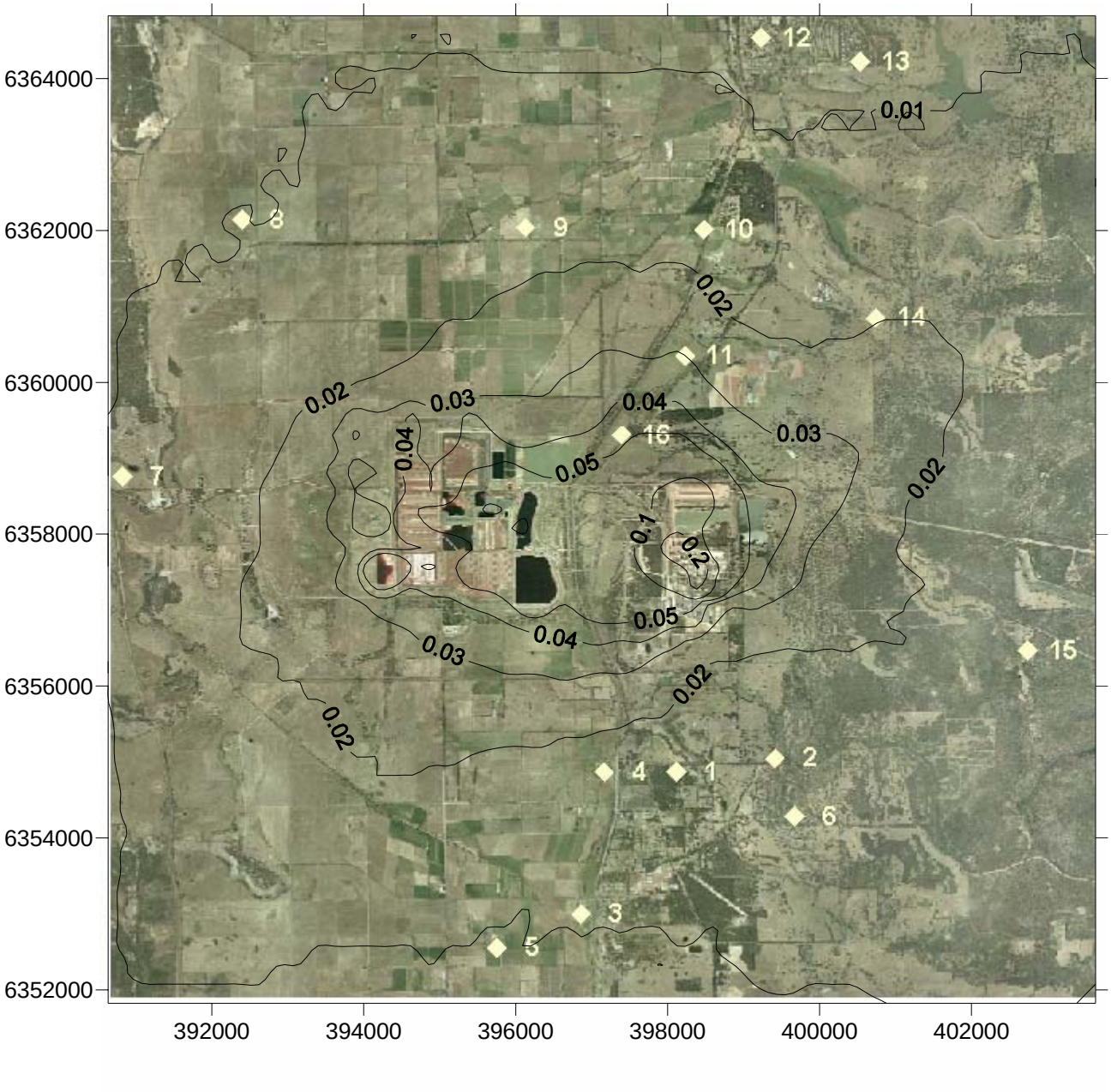

Figure 5c
Chronic HI – Upgrade Case 7 (Boilers)

Client: Alcoa	ENVIRON		
Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05	

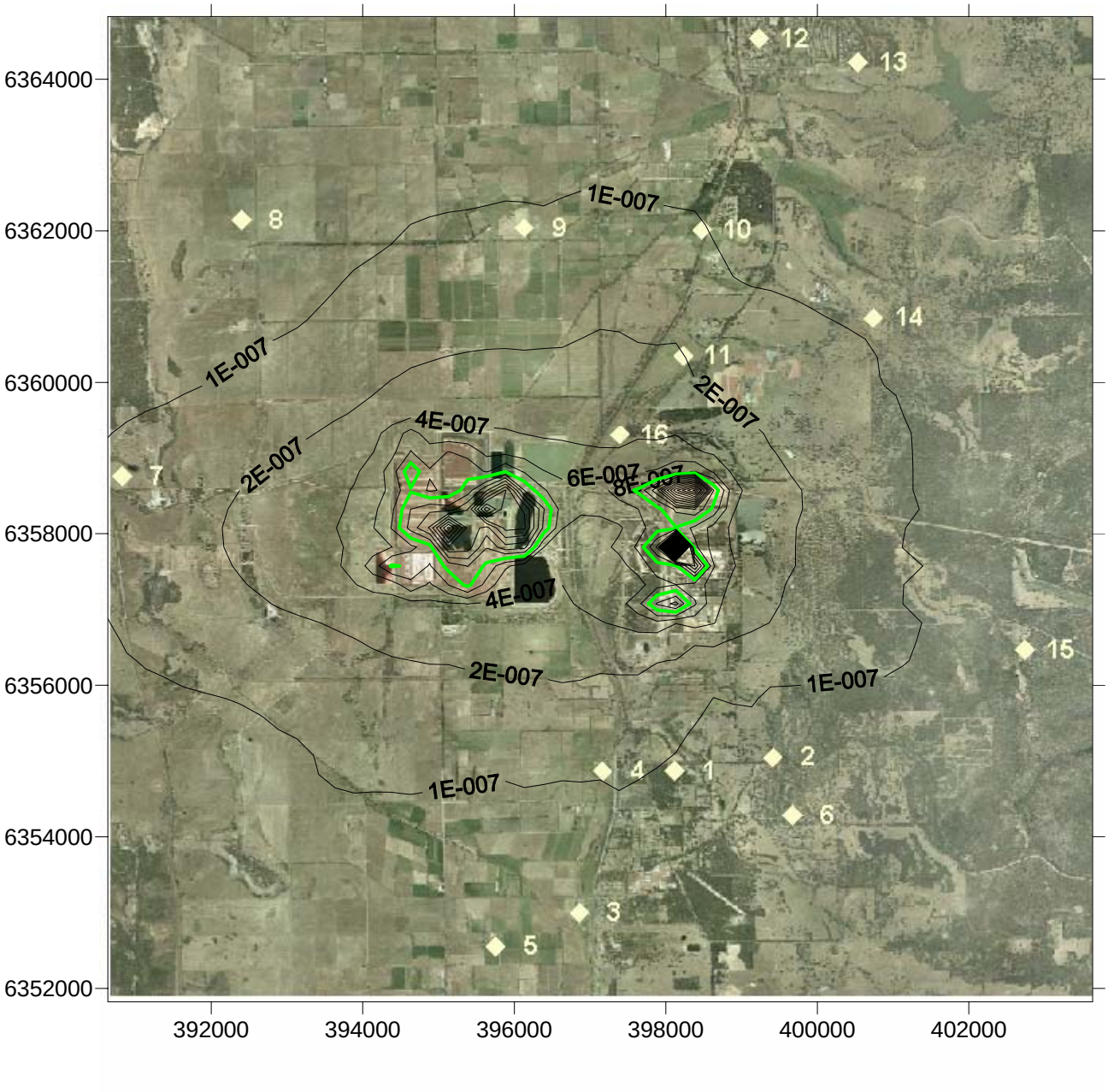

Figure 6a
Incremental Carcinogenic Risk
Base Case

Client: Alcoa	ENVIRON	
Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

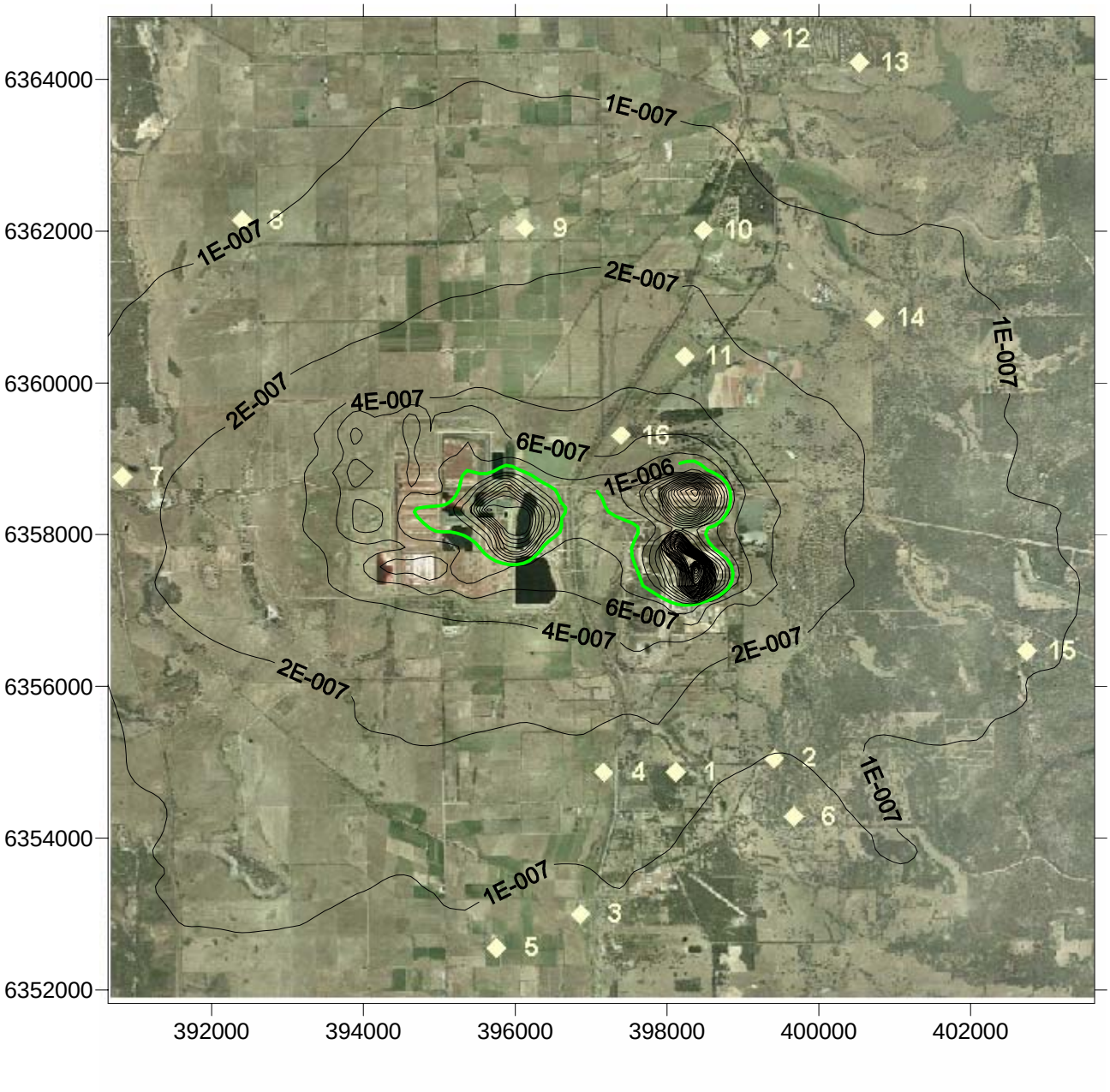

Figure 6b
Incremental Carcinogenic Risk
Upgrade Case 6 (Cogeneration)

Client: Alcoa	ENVIRON	
Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

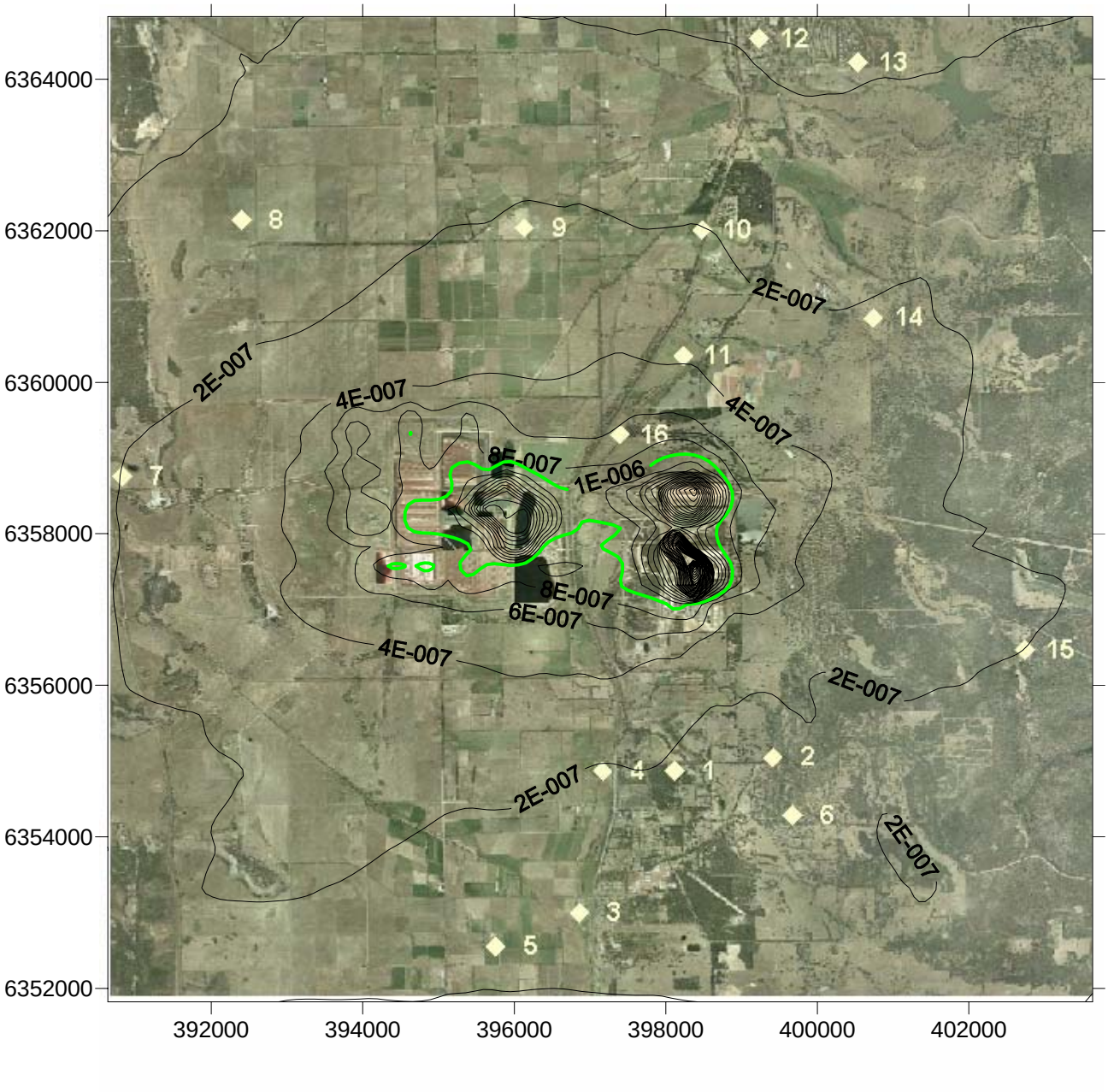

Figure 6c
Incremental Carcinogenic Risk
Upgrade Case 7 (Boilers)

Client: Alcoa	ENVIRON	
Project: Wagerup Expansion HRA	Drawn: BPB	Date: 29 March 05

APPENDIX A

Toxicity Assessment
Prepared by
Benchmark Toxicology Services

BenchMark Toxicology Services

As Trustee for the P & K Family Trust (ABN 72 217 434 679)

Empowering Through Knowledge

Health Risk Assessment of Atmospheric Emissions for the Proposed Emissions on the Wagerup Aluminium Refinery

Appendix A
Toxicity Assessment

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Prepared by:

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BenchMark Toxicology Services Pty Ltd

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BTS File Number: BTS 04/007
Date: 19 April 2005

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Abbreviations

ADI	Acceptance Daily Intake
AT	Air Toxics
ATSDR	Agency for Toxic Substances and Disease Registry
CCA	copper chrome arsenate
cm	centimetre = one hundredth of a metre
cm^2	square centimetre
Cr	chromium
CO	Carbon monoxide
CO_2	Carbon dioxide
COPD	Chronic obstructive pulmonary disease
CSIRO	Commonwealth Scientific and Industrial Research Organisasation
DEH	Department of Environment and Heritage
DMSA	disodium methyl arsenate
EU	European Union
FAO	Food and Agriculture Organization
FEV	Forced Expiratory Volume
FSANZ	Food Standards Australia and New Zealand
g	gram(s)
GLC	ground level concentration
h	hour(s)
Hg	mercury
IAQ	Indoor Air Quality
IARC	International Agency for Research on Cancer
IPCS	International Programme on Chemical Safety
IRIS	Integrated Risk Information System
JECFA	The Joint FAO/WHO Expert Committee on Food Additives
L	litre
LOAEL	Lowest Observable Adverse Effect Level
m	metre
m/s	metre per second
m^2	square metre
m^3	cubic metre
mg	milligram = one thousandth of a gram
mm	Millimetre = one thousandth of a metre
MCS	multiple chemical sensitivity
MRL	minimal risk level
MSMA	monosodium methyl arsenate
MW	molecular weight
ng	nanogram = one thousand millionth of a gram (0.000000001 g)
NEPC	National Environmental Protection Council
NEPM	National Environmental Pollution Measure
NHMRC	National Health and Medical Research Council
NIOSH	National Institute of Occupational Safety & Health (US)
NO_2	Nitrogen Dioxide
NOAEL	No Observable Adverse Effect Level

NOHSC	National Occupational Health and Safety Commission
OEHHA	Office of Environmental Health Hazard Assessment (EPA, California)
OSHA	Occupational Safety and Health Administration
O_3	ozone
^oC	Degrees Centigrade
PAH	Polycyclic aromatic hydrocarbons
PM_n	particulate matter with aerodynamic diameter < *n* µm
ppb	parts per billion
ppm	parts per million
REL	refernce exposure level
RfC	reference concentration
RfD	reference dose
RIVM	National Institute of Public Health and the Environment (The Netherlands)
RSD	Risk specific dose
SO_2	Sulfur dioxide
TC	Tolerable concentration
TCA	Tolerable concentration in air
TDI	Tolerable daily intake
TCEQ	Texas Commission on Environmental Quality
TEF	toxicity equivalency factors
TEQ	toxicity equivalent quotient
TSP	total suspended particulates
UK	United Kingdom
UR	Unit risk
US	United States
US EPA	United States Environmental Protection Agency
VSD	Virtually safe dose
VOC	volatile organic compound
WHO	World Health Organization
µg	microgram(s) = one millionth of a gram
µm	micrometre(s) = one millionth of a metre
≥	equal to or more than
≤	equal to or less than
>	more than
<	lesser than

TABLE OF CONTENTS

TOXICITY ASSESSMENT

1. Introduction

Toxicity assessment is the second step in the environmental health risk assessment process described by the National Environmental Health Council (enHealth, 2002)[1]. It comprises hazard assessment and dose-response assessment. The objective of the toxicity assessment is to identify the hazard(s) associated with a particular chemical (the potential to cause adverse effects) and the conditions under which the hazard may manifest or the dose response relationship). The major outcomes of the toxicity assessment are estimates of acceptable doses (toxicity values) which can be used to regulate chemicals in the various environmental media or to assess risks posed by the chemicals

In this assessment, the term toxicity value is used generically to refer to estimates of Tolerable Intakes (TI) - named variably by different jurisdictions as Tolerable Daily Intake (TDI), Acceptable Daily Intake (ADI), Tolerable Concentration (TC), Reference Dose (RfD), Reference Concentration (RfC), Maximal Risk Levels (MRL) - and probabilistic estimates of risk such as unit risk (UR, the risk associated with exposure to a unit concentration such 1 $\mu g/m^3$), risk specific dose (RSD, risk associated with a particular dose), slope factors (the gradient – slope of the dose-response curve) or Virtually Safe Dose (VSD, the dose at an acceptable or negligible level of risk).

1.1. Derivation of toxicity values

For risk assessment purposes two dose–response models are used in deriving toxicity values: threshold and non-threshold models. The assumptions associated with each of the models and the choice of model reflect science policy, rather than mechanisms of action of the different chemicals.

1.1.1. Threshold model

The underlying assumption in the threshold model is that there is a threshold dose for exch chemical, below which no adverse or toxic effect occurs. Toxicity values are expressed as the dose or concentration that is unlikely to cause any appreciable adverse health effects over a lifetime (70 years). This model is used in deriving toxicity values for substances that do not have carcinogenic properties or for non-cancer adverse effects for substances that do, as well as for some substances that have carcinogenic properties. The outcomes are toxicity values such as the TDI, ADI, TC, RfD, RfC, and MRL.

Toxicity values are derived using the No Observable Adverse Effect Level (NOAEL)[2] or the Lowest Observable Adverse Effect Level (LOAEL)[3] in experimental studies (as well as human studies) and a number of substance specific factors derived to account for inter and intra-species differences (species and human individual sensitivities), study duration and quality of the database (confidence in the experimental or epidemiological results used), severity of the

[1] National Environmental Health Council (enHealth, 2002). Environmental Health Risk Assessment. Guidelines for assessing human health risks from environmental hazards. Commonwealth of Australia, 2002.

[2] The NOAEL is the highest dose or concentration of a chemical that causes no effects in experimental animal studies or human studies.

[3] The LOAEL is the lowest dose or concentration of a chemical that causes a measurable, statistically significant adverse effect in experimental studies in animals or in human studies.

effect, using a LOAEL when a NOAEL is not determined, and a substance specific factor for any other concerns that might arise.

Substance specific factors or uncertainty factors are used to account for the uncertainties associated with extrapolation from the more specific conditions in experimental animals or human studies to the more general conditions under which the general population may be exposed, for example to environmental chemicals. The substance specific factors can vary between 1 and a few thousands, with the most commonly used being 100 to account for differences between experimental animals and humans (a factor of 10) and differences between individual humans (a factor of 10).

1.1.2. Non-threshold model

The underlying assumption in the non-threshold model is that there is a finite probability of adverse effects no matter how low the dose or the concentration. In mathematical terms the dose response relationship is described as having a positive slope at the origin (zero dose), ie, the risk is zero at zero dose.

At very low doses or concentrations, however, the risks are so low that for practical purposes they may be considered to be zero. In setting standards for carcinogens, the US EPA generally considers a risk less than or equal to one in a million to be a *de minimis* or trivial risk, hence considers it to be an acceptable goal (US EPA (1991)[4].

The toxicity values are expressed as risk probabilities (eg, UR[5], slope factor[6]) from which a dose or concentration (RSD[7], VSD[8]) that poses a negligible or acceptable risk (eg, one in one million risk) can be determined. This model is used in deriving toxicity values for substances that have carcinogenic properties, particularly those that also exhibit genotoxic properties (damage to genetic material).

1.1.3. Interpretation

The outcomes of both models are toxicity values, which in essence are estimates of a safe or acceptable dose of the substance based on the knowledge available at the time. Both models are based on sets of assumptions, which are intentionally conservative to ensure overestimation of the likely risks posed by the substance; hence to be protective of human health. The non-threshold model generally gives more conservative estimates of risk than the threshold model because of the assumptions made and the processes used.

Is it safe?

The concept, as well as the perception, of safety or risk, is a subjective one and will vary between individuals and in different circumstances. Safety does not necessarily mean the absence of risk, nor does risk mean the absence of safety. The toxicity value is a measure of the likely effects of a substance expressed in a consistent and verifiable way that reflects the best judgement based on the available information.

In case of probabilistic expressions of risk, the use of the toxicity value requires some judgement on an acceptable level of risk. The reference level of risk used in the screening risk assessment is the incremental *de minimis* risk of one

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[4] U.S. EPA. 1991. Amendments to the water quality standards regulation; compliance with CWA Section 303(c) (2) (B); proposed rule. Fed Reg 56(223):58420-58437.

[5] UR is the risk associated with exposure to a unit concentration such 1 $\mu g/m^3$ or dose.

[6] Slope factor is the gradient or slope of the dose-response curve.

[7] RSD is the risk specific dose – the risk associated with a particular dose.

[8] VSD is the virtually safe dose - the dose at an acceptable or negligible level of risk.

additional case of cancer in one million exposed people over a lifetime of 70 years.

Use of toxicity values

The toxicity values so derived are then used to calculate guideline values or criteria in a particular medium (air, water, soil, food), which are the basis for regulating the levels of contaminants in the medium and the environment.

In the case of air quality, the guideline concentration or standard may be the same as the inhalational toxicity value (TC or RfC).

However, this is not necessarily the case for all chemicals as the derivation of guidelines or standards may take into account factors in addition to the toxicity value (eg, limitations of analytical techniques) or may be derived using endpoints other than traditional toxicity values; or take other factors into consideration, eg, criteria pollutants in the NEPM on ambient air quality (NEPC, 1998)[9], in which cost/benefit is also considered in deriving the criteria.

Notwithstanding, the main objective of the guideline or standard is the protection of public health.

The toxicity values, guideline values or standards are used as reference values with which estimated exposures are compared to assess the likely risks to health for exposed populations.

Generally, for air contaminants, the reference values are derived for different averaging periods, corresponding with potential effects over the short term or over the longer term (eg, 1-h, 24-h or annual averages).

The reference values are expressed as concentrations in air in ppm (parts per million of air) for volatile substances or mg substance/m^3 of air for non-volatile substances or both types of substances.

For pure volatile substances, conversion between the two units requires consideration of the molecular weight (MW) of the substance at a set temperature and pressure – usually at 25 ^oC and 760 mm mercury (Hg) – and the volume of the volatilised substance at the set temperature and pressure.

Thus, Concentration (mg/m^3) = Concentration (ppm) x MW ÷ 22.4126, where 22.4126 L is the volume occupied by an ideal gas at 25 ^oC and 760 mm Hg.

Conversely, Concentration (ppm) = Concentration (mg/m^3) x 22.4126 ÷ MW.

The reference values for the chemicals of concern used in the screening risk assessment are summarised in the following tables.

[9] National Environment Protection Council (1998). National Protection Measure on Ambient Air Quality. http://www.ephc.gov.au/pdf/Air_Quality_NEPM/air_nepm0698.pdf

Reference values for chemical of concern

Acute and chronic non-carcinogenic effects

		Reference Values					
		Acute Health Effects			Chronic Health Effects		
No	Compound	Time (h)	µg/m³	Source		µg/m³	Source
	Criteria Pollutants						
1	Carbon monoxide	8	11,250	NEPC			
2	Nitrogen Dioxide	1	246	NEPC	Annual	62	NEPC
3	PM_{10}	24	50	NEPC			
4	Sulphur dioxide	1	571	NEPC	Annual	57	NEPC
	Metals						
5	Arsenic				Annual	1	RIVM
6	Cadmium				Annual	0.005	WHO
7	$Chromium^{6+}$				Annual	0.1	IRIS
8	Manganese				Annual	0.15	WHO
9	Mercury	1	1.8	OEHHA	Annual	1	WHO
10	Nickel	1	6.0	OEHHA	Annual	0.09	ATSDR
11	Selenium				Annual	20	OEHHA
	Organics						
12	Acetaldehyde	24	2,000	WHO	Annual	50	WHO
13	Acetone	24	67,414	ATSDR	Annual	33,707	ATSDR
14	Acrolein	1	0.2	OEHHA	Annual	0.06	OEHHA
15	Benzene	6	1,300	OEHHA	Annual	60	OEHHA
16	2-Butanone	1	13,000	OEHHA	Annual	5,000	IRIS
17	Ethylbenzene				Annual	22,000	WHO
18	Formaldehyde	24	54	NEPC (AT)	Annual	11	ATSDR
19	Methylene Chloride	24	3,000	WHO	Annual	1,137	ATSDR
20	PAH				Annual	0.0003	NEPC (AT)
21	Styrene	168	260	WHO	Annual	900	RIVM/OEHHA
22	Toluene	24	4,113	NEPC (AT)	Annual	411	NEPC (AT)
23	Vinyl chloride	1	180,000	OEHHA	Annual	100	IRIS
24	Xylenes	24	1,183	NEPC (AT)	Annual	946	NEPC (AT)
25	1,2,4 Trimethylbenzene [1]				Annual	800	RIVM
26	1,3,5 Trimethylbenzene [1]				Annual	800	RIVM
	Other						
27	Ammonia	1	3,200	OEHHA	Annual	100	IRIS

ATSDR: Agency for Toxic Substances and Disease Registry (US Department of Health & Human Services)

IRIS: Integrated Risk Information System (US EPA)

NEPC: National Environmental Protection Council (Australia); AT: Air Toxics

OEHHA: Office of Environmental Health Hazard Assessment (EPA California)

RIVM: National Institute of Public Health & the Environment (The Netherlands)

WHO: World Health Organization

Carcinogenic Health Effects

No	Substance	IARC Group	Unit risk		Source
5	Arsenic	1	1.5×10^{-3}	per $\mu g/m^3$	WHO
6	Cadmium	1	1.8	per mg/m^3	IRIS
7	Chromium (VI)	1	4.0×10^{-2}	per $\mu g/m^3$	WHO
10	Nickel	1	3.8×10^{-4}	per $\mu g/m^3$	WHO
12	Acetaldehyde	2 B	9.0×10^{-7}	per $\mu g/m^3$	WHO
15	Benzene	1	6.0×10^{-6}	per $\mu g/m^3$	WHO
19	Methylene Chloride	2 B	4.7×10^{-7}	per mg/m^3	IRIS
20	Polycyclic Aromatic Hydrocarbons	2 A	8.7×10^{-7}	per ng/m^3	WHO
23	Vinyl chloride	1	1.0×10^{-6}	per $\mu g/m^3$	WHO

The derivation of the reference values, the sources and hazardous properties of the chemicals of concern are summarised in the following Sections.

It should be noted that in describing the adverse effects of the substances or the hazardous characteristics, the narrative refers to both the effects of the substances as such at different concentrations or doses and the effects of the substances in air. In general the effects are described loosely in rank order of severity from the least to the most severe effect as exposure concentrations increase.

1.1.4. Toxicity and the respiratory tract

The human respiratory tract can be divided into three areas: upper, middle and lower respiratory tract, each with different functions and affected differently by toxicant.

The upper respiratory tract comprises the mouth, nose, throat and trachea. These areas are covered by a thin layer of protective mucous, that tends to neutralise small quantities of acidic or alkaline materials, and very fine hairs (cilia) which trap some suspended particles that are breathed in. The cilia beat in unison and move the trapped material to the throat where it is expelled as sputum or ingested. Additionally the nasal passages are covered in fine hairs which act a filter for larger suspended particles in the air that might be breathed in.

At rest, most people normally breathe through the nose. However, with exercise, there is an increase in the amount of air inhaled and increased breathing through the mouth, which increases the air intake, hence the dose, as well as bypassing the filtering action of the hairs in the nose.

The majority of chemical breathed in the air will have a local effect on the upper respiratory tract, eg, irritation. However, systemic effects (effects at sites distant from the point of contact after absorption and distribution through the body by the blood) are also possible from absorption in the lungs or from the gastrointestinal tract from swallowed material. Absorption of gaseous materials is

very low in the upper respiratory tract although absorption from particles trapped ion the mucous membranes might occur over time, but not to any significant extent compared with the lower respiratory system.

The middle respiratory tract comprises the trachea, the bronchi and larger bronchioles. The surfaces are covered with mucous and cilia, which trap particles that have penetrated deeper into the respiratory tract (aerodynamic diameter between 10 and 50 µm – 1 µm –micrometre – equals one one millionth of a metre; 0.000001 m) and move them back toward the mouth, where they may be swallowed.

The lower respiratory tract comprises the smaller bronchioles and the alveoli, the area of the lungs where gaseous exchange (normally mainly oxygen and carbon dioxide) take place. The alveoli have a very large surface area (about 100 m^2; the size of a small house), are 1-2 cells thick and have a rich blood supply; hence a number of substances, particularly gases, are readily absorbed or transferred to the blood and distributed to other parts of the body. Particles of aerodynamic diameter of ≤ 10 µm will reach the lower respiratory tract. Some will be trapped by the cilia on the bronchioles and swept back towards the mouth. Most will reach the alveoli where they can accumulate or be dissolved by the fluids and absorbed into the systemic circulation. The alveoli have a variety of defence mechanisms that protect them against foreign particles.

Toxicants in air can have a local effect (eg, irritation, inflammation) or systemic effects when in contact with the lower respiratory tract. Some will cause local damage to the lungs that eventually leads to the development of cancer.

Dust in air can be classed as inspirable dust and respirable dust, the latter comprising particles with an aerodynamic diameter of ≤ 10 µm which can reach the lower respiratory tract. Of the inspirable dust in the air that we take into the respiratory system with each breath, 25% is expired; about half of the remainder gets deep into the lungs, and the other half is trapped by the nose, the remainder of the upper and the middle respiratory tract and unknowingly swallowed (or expelled in sputum).

Children may be at an increased risk from inhaled toxicants compared with adults because of their higher activity and because they tend to breathe more air per unit body weight than adults, hence they receive a higher dose per unit body weight of chemicals in the air than adults.

1.2. Criteria Pollutants

1.2.1. Carbon monoxide

Carbon monoxide (CO) is an odourless, colourless and tasteless gas that, in high concentrations, is poisonous to humans.

WHO (2000)[10] describe global background levels in the atmosphere as ranging between 0.01 to 0.2 ppm (0.06 – 0.14 mg/m^3), with 8-h average concentrations in European cities < 17 ppm or 20 mg/m^3.

Enclosed areas (car parks, road tunnels) with poor ventilation and in which combustion engines are used would have much higher levels (> 100 ppm).

CO affects human health by reducing the oxygen carrying capacity of the blood. When inhaled, CO binds preferentially to haemoglobin, the blood's oxygen carrying molecule, leading to the formation of carboxyhaemoglobin. The haemoglobin in the form of carboxyhaemoglobin no longer binds oxygen; thus

[10] WHO (2000). Carbon monoxide. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 75 -79.

depending on the extent of carboxyhaemoglobin formation, critical tissues such as the brain and peripheral nervous system, the heart and other specialised tissues that require large amounts of oxygen, may not function properly because of insufficient oxygen (carboxyhaemoglobin concentrations > 2.5%).

The initial symptoms of CO poisoning are associated with effects on the Central Nervous System (CNS) and similar to those of influenza (but without the fever). They include:

- Impaired vigilance
- Headache
- Fatigue
- Shortness of breath
- Nausea
- Dizziness

At sufficiently high concentrations CO can cause loss of consciousness and death when the carboxyhaemoglobin concentrations reach levels > 40%).

Particularly at risk or sensitive groups include, people with ischaemic heart disease, cyanotic heart disease, hypoxemic lung disease, cerebrovascular disease, peripheral vascular disease, anaemia and other haemoglobin abnormalities, children and the developing foetus.

The National Environmental Protection (Ambient Air Quality) Measure (NEPM) (NEPC, 1998)[11] also specifies a standard for carbon monoxide of 9 ppm (10 mg/m^3) (allowable exceedance of 1 day a year) for an 8-h averaging period.

The National Health and Medical Research Council (NHMRC, 1996)[12] recommended an indoor air quality goal for CO of 9 ppm (10 mg/m^3), as an 8-h average, not to be exceeded more than once a year. An 8-h averaging time is used for CO because it takes about 6-8 h for the concentration of carboxyhaemoglobin in the blood to reach a steady state.

WHO (2000)[13] recommended guideline values for a number of averaging periods, aimed at ensuring that blood carboxyhaemoglobin levels do not exceed 2.5% and any time. These values include CO concentrations of 90 ppm (100 mg/m^3) for a 15-min average, 25 ppm (30 mg/m^3, rounded up from 27.8) for a 1-h average, and 9 ppm (10 mg/m^3) for an 8-h average.

The NEPC 8-h average standard is used as the reference value in the health risk assessment.

1.2.2. Nitrogen Dioxide

Nitrogen dioxide (NO_2) is a pungent acidic gas – corrosive and oxidising. It is produced mainly from combustion processes (burning of fossil fuel –coal, gas and oil). In cities, 80% of the ambient NO_2 comes from vehicle emissions. Natural background levels concentrations are in the range 0.4 – 9.4 µg/m^3 (0.2 – 4.6 ppb – parts per billion), 1-h averages in outdoor urban areas can range up to 1 mg/m^3 (~ 0.5 ppm) and in indoor air up to 2 mg/m^3 (1 ppm)(WHO, 2000)[14].

Combustion converts the nitrogen in the atmosphere and in the fuel (if present) into mainly nitrogen oxide (NO), which then slowly oxides into NO_2 in the atmosphere, a reaction that is speeded up by ozone (O_3). The oxides of nitrogen

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[11] National Environment Protection Council (NEPC) (2003). Ambient Air Quality NEPM. http://www.ephc.gov.au/nepms/air/air_nepm.html (Accessed March 2005)

[12] NHMRC (1996). Interim national indoor air quality goals recommended by the National Health and Medical Research Council. www.nhmrc.gov.au/publications/pdf/rec1-2.pdf (Accessed March, 2005)

[13] WHO (2000). Carbon monoxide. *Ibid*.

[14] WHO (2000). Nitrogen dioxide. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 186 – 193.

can react with volatile organic compounds in the presence of sunlight to form photochemical smog. NO_2 will dissolve in water to form nitrates and nitric acid.

NO_2 appears to affect humans both directly and indirectly: directly, by irritation that leads to an inflammatory reaction in the lungs, and indirectly by affecting the immune system.

The short term effects of NO_2 are mainly associated with the respiratory system, generally in combination with other pollutants such as irritant gases and particles. The effects include increase in wheezing, cough, sputum production in asthmatics and people with chronic inflammatory lung disease. At higher concentrations it can contribute to illness (morbidity) and mortality of especially sensitive sub groups, such as children, asthmatics and people with chronic lung diseases such as chronic bronchitis.

It has not been possible to separate the effects of NO_2 from ambient air and indoor air (un-vented gas stoves and heaters are a major contributor to NO_2 in indoor air).

Long term effects, include increased incidence of coughing, wheezing and respiratory infections in young children (from infancy to late childhood; about 5 - 12 years of age), particularly on exposure to NO_2 indoors. These reactions appear to involve effects on the immune defence mechanisms in the pulmonary airways. These effects are not reported in adults. In animal studies, organs other than the lung are also affected (spleen, liver and blood).

There is considerable uncertainty in the health database on NO_2, leading to regulatory agencies taking a more conservative approach to setting exposure guidelines and standards.

WHO (2000)[15] recommended a 1-h average guideline value of 200 $\mu g/m^3$ (0.1 ppm) stating that at 400 $\mu g/m^3$ (0.2 ppm) there is evidence to suggest small effects on the pulmonary function of asthmatics. There is more uncertainty about the long term exposure data, with WHO recommending a guideline value of 40 $\mu g/m^3$ (0.02 ppm)

The Australian standards for NO_2 in ambient air are 0.12 ppm (1-h average; 246 $\mu g/m^3$) and 0.03 ppm (annual average; 62 $\mu g/m^3$) (NEPC, 1998)[16].

The NEPC values are used as the reference values in the health risk assessment.

1.2.3. Particulates (PM_{10})

Particulate matter refers to a variety of minute solid or liquid particles that remain suspended in the air and can be inhaled into the respiratory system. The terms particulate matter, particulates, particles and aerosols are used interchangeably. The terms dust, fumes, smoke, mist, fog, smog, and haze, are used often to describe physical forms of airborne particulate matter.

Particles can be characterised by size, number, their mechanism of formation or origin, chemical composition, physical properties or by what is measured by a particular measuring technique (NEPC, 1998)[17]. Particles can be referred to as total suspended particulates (TSP), as black smoke, or by direct or indirect description of their size, which is mainly related to their capacity to penetrate deep into the lungs. Particles may also be classified as primary particles eg, road dust, or secondary particles, ie those formed in the atmosphere.

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[15] WHO (2000). Nitrogen dioxide. *Ibid*.

[16] National Environment Protection Council (NEPC, 1998). National Protection Measure on Ambient Air Quality. http://www.ephc.gov.au/pdf/Air_Quality_NEPM/air_nepm0698.pdf

[17] NEPC (1998). *Ibid*.

TSP (also referred to as total dust) has been variously reported as comprising particles with aerodynamic diameters ranging from ≤ 30 µm and as high as ≤ 500 µm. In Australia, the TSP fraction is considered to comprise suspended particles with an aerodynamic diameter of ≤ 50 µm (NEPC, 2001)[18]. The NHMRC (1996)[19] recommended an ambient air quality guideline of 90 µg/m^3 for TSP.

Specific particle size fractions include PM_{10} (particles of aerodynamic diameter of 10 µm or less) and $PM_{2.5}$ (particles of aerodynamic diameter of 2.5 µm or less). The PM_{10} fraction is also the respirable fraction; particles that have a small enough aerodynamic diameter to reach deep into the lungs. Hence, they can affect the whole respiratory system.

The effects of particulates can vary depending on the composition and individual components, mainly determined by the source or the presence of other chemicals in air. For example, irritant gases such as sulfur dioxide, nitrogen dioxide and hydrogen chloride or volatile organic compounds in the air may react with particles and alter their toxicological properties. Other substances or particles may react with them, eg, polycyclic aromatic hydrocarbons and render them carcinogenic.

Some particulates are referred to as nuisance dust ie, dust that is generally innocuous and not recognized as the cause of serious pathological conditions. Some mineral dust, eg, from agricultural activities or desert sources, would fall into this category. WHO (2000)[20] reports that limited evidence from studies on dust storms indicates that the PM_{10} fraction from dust storms is much less toxic than the PM_{10} fraction associated with combustion sources. Further, that additional studies suggest that the observed effects of PM_{10} are associated with very fine particles, strong aerosol acidity or sulfates and not with the coarser fraction between PM_{10} and $PM_{2.5}$.

Adverse effects particulates can range from minor, acute and reversible effects such as eye and upper respiratory tract discomfort because of irritation, to more serious effects such as cancer and death.

Acute effects of particulates are generally mucosal irritation of eyes, nose and throat and middle respiratory tract that can result in increased morbidity, hospital admissions and mortality. The respiratory effects include increased use of bronchodilators (asthmatics), cough, increase incidence of respiratory symptoms and reduced lung efficiency.

Long term exposure appears to be associated with increased rates of bronchitis and reduced lung function as well as increased mortality from heart and lung disease, although the impacts of particulates on life expectancy is not known.

Asthmatics, people with compromised respiratory systems (eg, COPD - Chronic obstructive pulmonary disease) and other respiratory disease, and the elderly may be at increased risk.

The NEPC (2003)[21] has established an ambient air quality standard of 50 µg/m^3 for PM_{10}.

The NEPC value is used as the reference value in the health risk assessment.

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[18] NEPC (2001). National Environment Protection (Ambient Air Quality) Measure. Issues paper – The need for a $PM_{2.5}$ standard in Australia. http://www.ephc.gov.au/pdf/Air_Variation_PM25/issues_paper.pdf

[19] NHMRC (1996). *Ibid*.

[20] WHO (2000). Particulate matter. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 186 – 193.

[21] NEPC (2003). *Ibid*.

Particulates and metals

Metal species in the emissions are likely to be in the form of particulates. They are considered, therefore, both as part of the PM_{10} fraction and individually. Whilst this may appear to be double counting of the metals concentration, it is not the case, since the reference values for particulates and those for metals are based on information relating to different toxic effects.

Aluminium oxide

The Wagerup community has expressed concern about the potential impact on human health of aluminium oxide or alumina dust. The principal concern seems to be about the effects of aluminium oxide on the immune system as it is used in medicine as an adjuvant. A comment on aluminium oxides used as adjuvants has been prepared separately.

Aluminium oxide appears to be a relatively non toxic chemical. The major national and international regulatory jurisdictions around the world have not set any ambient air quality guidelines or standards. The only jurisdiction to derive guidelines for aluminium oxide is the Texas Commission on Environmental Quality (TCEQ, 2003)[22] that has published acute and chronic effects screening levels (ESL) of 50 µg/m^3 and 5 µg/m^3, respectively, for aluminium oxide [CAS No 1344-28-1]. However, the derivation of the reference values could not be identified on the TCEQ website[23].

The TCEQ defines ESL as guideline concentrations used to evaluate ambient air concentrations of various substances. They are based on the potential for the constituents to cause adverse health effects, odour nuisances, vegetation effects, or materials damage. Health-based screening levels are set at levels lower than levels reported to produce adverse health effects, and are set to protect the general public, including sensitive subgroups such as children, the elderly, or people with existing respiratory conditions. If the air concentration is below the screening level, adverse effects are not expected. If the air concentration is above the screening level, it is not indicative that an adverse effect will occur, but rather that further evaluation is warranted.

The acute ESL determined by TCEQ is 100 times lower that the occupational short term exposure level in the US (OSHA) for the respirable fraction of aluminium oxide powder (5 mg/m^3). It is thus possible that the TECQ value may have been adopted from the occupational exposure limit for aluminium oxide dust, by adjusting from discontinuous to continuous exposure (24 h per day, 7 days/week, 52 weeks per year, 70 years compared with 8 h per day; 5 days/week, 48 weeks per year and 40 years exposure). Thus the substance specific conversion factor from occupational to environmental exposure = (24÷ 8) (7÷ 5) (52÷ 48) (70÷ 50) = 10.6. Adding a factor of 10 for human heterogeneity, ie, increased sensitivity of the more heterogeneous non occupational population, then the composite conversion factor would be 106.

Aluminium oxide in the Wagerup emissions is considered as part of the PM_{10} fraction. Given that the particulates emissions comply with PM_{10} standards, then the aluminium oxide is unlikely to pose a health risk.

Particulates and Alkaline dust particles

Concerns have also been expressed about alkaline dust.

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[22] Texas Commission on Environmental Quality (ECEQ, 2003). TCEQ Effects Screening Levels (ESLs). http://www.tceq.state.tx.us/comm_exec/tox/Esl2003_136681.pdf

[23] Texas Commission on Environmental Quality. http://www.tceq.state.tx.us/

Alkaline dusts are part of mineral dusts and mining dusts. No environmental guidelines or standards have been identified for alkaline dust. Occupational health standards appear to refer to general dust limits in the workplace.

From a health perspective the important aspect is whether or not the dust is respirable, ie, PM_{10}. Thus alkaline dust is assessed as part of the PM_{10} assessment.

Alkaline dust may be more of an issue with impacts on amenities in the immediate area around the refinery. The larger particles in the dust would settle first because of their size and possibly impact on amenities because of their alkalinity.

Some of the dust is likely to react with acid gases and be neutralised.

Particulates and Silica

Another compound about which the community has expressed concern is silica.

Free crystalline silica or silicon dioxide (SiO_2) is a ubiquitous and plentiful mineral in the earth's crust. It is found in sand, many rocks such as granite, sandstone, flint and slate, and in some coal and metallic ores. The three most common forms are quartz, cristobalite and tridymite.

Cristobalite and tridymite are polymorphs of quartz, ie, they are composed of the same chemical entity, SiO_2, but have different physical structures.

Silicon dioxide exists in two forms, amorphous and crystalline. In crystalline forms, the structures are characterised by tetrahedral configuration of atoms within the crystals, whereas in the amorphous forms, the silicate (SiO_4) subunits show no regular lattice pattern in the structures.

Silica dust is released during operations in which rocks, sand, concrete and some ores are crushed or broken. In a recent review on the properties and health effects of silica dust, De Klerk *et al.* (2002)[24] suggest that exposure to crystalline silica in Australia is predominantly to crystalline quartz.

The available information on the health effects of silica is almost entirely from occupational studies in the workplace. Available adequate studies on environmental exposure to ambient silica dust and toxicological data from animal studies are limited in scope and design. The results of human studies on occupational exposure are more appropriate than animal studies for assessing the health effects of silica in ambient dusts (US EPA, 1996)[25].

Prolonged or repeated exposure to fine airborne crystalline silica dust may cause a number of lung diseases, the most notable being silicosis or severe scarring of the lungs. The formation of scar tissue inhibits the flow of oxygen into the lungs and into the bloodstream eventually leading to other systemic diseases. De Klerk *et al.* (2002)[26] provide a very comprehensive description and analysis of the health effects of silica dust.

Silicosis is an occupational disease associated with relatively high and prolonged exposures to crystalline silica in the workplace. It is not a disease in the general population that is not occupationally exposed to silica. For example, WHO

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[24] De Klerk N, Ambrosini G and Musk AW (2002). A Review of the Australian Occupational Exposure Standard for Crystalline Silica. University of WA (Prepared for NOHSC). http://www.nohsc.gov.au/OHSInformation/Databases/ExposureStandards/Crystalline-silica/ReviewExpStdCrystallineSilica.pdf

[25] US EPA (1996). Ambient Levels and Noncancer Health Effects of Inhaled Crystalline and Amorphous Silica: Health Issue Assessment. National Center for Environmental Assessment Office of Research and Development U.S. Environmental Protection Agency Research Triangle Park, NC 27711.

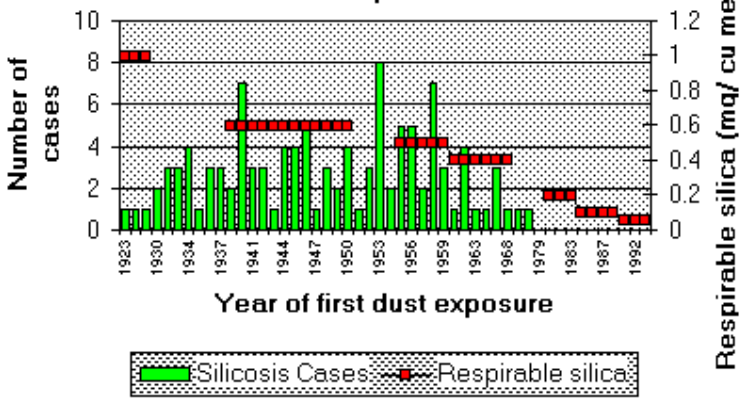
[26] De Klerk N, Ambrosini G and Musk AW (2002). *Ibid*.

(2000a)[27] states that to date (ie, date of its report), there have been no adverse effects associated with non-occupational exposure to quartz dust. Moreover, the US EPA (1996)[28] considered that the risk of exposure to silica in ambient dust at concentrations that comply with ambient air quality (PM_{10} standards) is close to zero.

Silicosis, is one of the oldest occupational diseases, still kills thousands of people every year everywhere in the world, and is incurable (WHO, 2000b)[29]. It is irreversible and progressive, progressing even when exposure stops.

In Western Australia, however, the incidence of silicosis in workers in the last 50 years has decreased considerably since the introduction of stricter occupational exposure standards and better dust control measures as illustrated by the following chart reproduced from Wan and Lee (1998)[30].

Figure (1)
Silicosis Cases 1984-1993 by Year of First Exposure



Some cases of silicosis are still emerging, particularly in some types of mining industries (De Klerk *et al.*, 2002)[31], although this may be a consequence of high exposure to crystalline silica dust in the past (Wan and Lee, 1998)[32].

The risk of developing, and the severity, of silicosis will depend on the physical and chemical properties of the silica and the other particles with which it is mixed, as well as the airborne concentration of respirable size silica dust and duration of exposure.

Generally, silicosis, also known as *chronic silicosis*, develops gradually over ≥ 20 years exposure. Extremely high exposures are associated with much shorter latency and more rapid disease progression (known as *accelerated silicosis*). In some cases of extremely high, short-term exposures, *acute silicosis* can develop within several weeks or up to five years after exposure.

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[27] World Health Organisation (WHO, 2000a). Crystalline Silica, Quartz - Concise International Chemical Assessment Document N° 24, International Programme on Chemical Safety.

[28] US EPA (1996). Ambient Levels and Noncancer Health Effects of Inhaled Crystalline and Amorphous Silica: Health Issue Assessment. *Ibid*.

[29] WHO (2000b). Silicosis. Fact Sheet No 238, May 2000. http://www.who.int/mediacentre/factsheets/fs238/en/

[30] Wan KC & Lee E (1998). Silicosis in W.A. 1984-1993. Safetyline Institute. http://www.safetyline.wa.gov.au/institute/level2/course21/lecture67/index.asp

[31] De Klerk N, Ambrosini G and Musk AW (2002). *Ibid*.

[32] Wan KC & Lee E (1998). *Ibid*.

Early symptoms of the disease are non-specific (eg, cough, mucous production and shortness of breath upon exertion). Importantly, the disease may progress undetected to an advanced stage of development, at which stage it can be detected by X-rays.

In mild silicosis, there is typically no significant respiratory impairment, although there is X-ray evidence of lung injury. In moderate to severe cases, significant and increasingly severe respiratory and cardiac impairment develops. In some cases, pulmonary function will be impaired to the point where the patient will need to be supplied with oxygen. Life expectancy may be reduced, depending on the severity of the case; death appears to be related to complications associated with the disease.

The International Agency for Research on Cancer classified crystalline silica (quartz and cristobalite) as carcinogenic to humans in 1997 (IARC, 1997)[33].

There is some suggestion from occupational studies that the development of bronchogenic cancer and silicosis may be correlated; however, it is not entirely clear if silicosis is a prerequisite change before the development of lung cancer. Notwithstanding, occupational exposure standards derived for protection from silicosis will also protect from lung cancer (De Klerk *et al.* 2002)[34].

The nature of health effects arising from exposure to crystalline silica depend on its source, as the physical properties of different forms of silica induce different health effects. Cocco *et al.* (2003)[35] further point out that in addition to the mineralogical characteristics of silica itself, the biological properties of silica are expected to vary according to its dilution in total respirable dust and the qualitative mineral composition of the dust mix. Importantly, freshly ground quartz particles appear to me more toxic than aged particles (US EPA, 1996)[36]. This is because grinding is thought to break the silicon-oxygen bond generating more toxic reactive oxygen species on the surface of the particles, which decay with time, thus reducing the toxicity of aged particles.

There are no Australian ambient air quality guidelines for silica. In addition, no air quality guidelines for silica were identified from national regulatory jurisdictions overseas, nor from international advisory organisations, such as WHO.

The Texas Commission on Environmental Quality (TCEQ, 2003)[37] has published ESL for crystalline silica, quartz (CAS No 14808-60-7), of 1 µg/m^3 for short-term exposure and 0.1 µg/m^3 for long-term exposure.

The basis for the derivation of the ESL for silica by TCEQ could not be identified, but it is likely to be from the occupational exposure level (TLV) of 50 µg/m^3 set by ACGIH (short-term ESL 1 µg/m^3 = (50 ÷ 42), where 42 if the factor sometimes used to adjust between occupational exposure and environmental exposure and to account to the more heterogeneous general population compared with the more homogeneous worker population.

The Office of Environmental Health Hazard assessment (OEHHA) of California Environment Protection Agency (OEHHA, 2004)[38] has proposed an ambient air

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[33] IARC (1997). Silica. In *Silica, some silicates, coal dust and para-aramid fibrils*. IARC Scientific Publications No. 68 International Agency for Research on Cancer, Lyon, France, pp. 41-242.

[34] De Klerk N, Ambrosini G and Musk AW (2002). *Ibid*.

[35] Cocco P (2003). The long and winding road from silica exposure to silicosis and other health effects Occupational and Environmental Medicine; 60:157-158

[36] US EPA (1996). Ambient Levels and Noncancer Health Effects of Inhaled Crystalline and Amorphous Silica: Health Issue Assessment. *Ibid*.

[37] Texas Commission on Environmental Quality (TCEQ, 2003) http://www.tceq.state.tx.us/comm_exec/tox/ESL2003.html

chronic Reference Exposure Level (REL) of 3 μg/m^3 for respirable silica as defined by NIOSH (currently undergoing public consultation). The REL was derived from occupational health studies, using appropriate adjustment factors for exposure and to account for sensitive sub types in the general population.

On the potential risks of silica exposure, the US EPA (1996)[39] states:

> *Thus, current data suggest that, for healthy individuals not compromised by other respiratory ailments and for ambient environments expected to contain 10% or less crystalline silica fraction in PM_{10}, maintenance of the 50 μg/m^3 annual NAAQS for PM_{10} should be adequate to protect against silicotic effects from ambient crystalline silica exposures.*

Thus it appears that, provided the concentrations of PM_{10} comply with the standards in Australia of 50 μg/m^3, any silica present in the particulate fraction is unlikely to pose a health risk.

1.2.4. Sulphur dioxide

SO_2 is a colourless, irritating and reactive gas with a strong odour. The odour is perceptible at different levels depending on the individual's sensitivity, but is generally perceived between 0.3-1.4 ppm and is easily noticeable at 3 ppm (8.6 mg/m^3).

Emissions of sulfur dioxide come primarily from major industrial activities that burn sulfur containing fuels (eg, coal, oil, petroleum and gas).

SO_2 is highly soluble in water and is quickly absorbed in the moist environment of the upper or lower airways of the respiratory tract, where it exerts its adverse effects. It causes a reduction in the diameter of airways and a reduction in airflow by acting on cells that cause inflammation, constriction and create mucus (NEPC, 2004)[40].

It first acts on the upper and middle airways (nose, throat, trachea and major bronchi) where it is mostly absorbed, resulting in an acidic solution which is irritating. Intake into the lower respiratory tract and the lungs occurs with exposure to higher concentrations and extended duration as well as increased breathing through the mouth.

Health effects from short-term exposures to SO_2 are most pronounced in people with asthma and other respiratory conditions such as COPD, and particularly in exercising asthmatics (exercise increases the amount of air inhaled and it increases the amount of mouth breathing that allows for deeper penetration of the air into the respiratory tract and dries the airways (NEPC, 2004). The elderly are also a susceptible population as they have reduced respiratory reserve as a result of the ageing process, which can be exacerbated by pre-existing cardio-respiratory disease.

The effects of SO_2 are reversible on cessation of exposure, and in asthmatics can be prevented or ameliorated by medication.

The most direct information on the short term effects of SO_2 comes from controlled studies for exposure periods ranging up to 1 h with volunteers in environmental chambers (WHO, 2000)[41]. The response to SO_2 exposure occurs

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[38] OEHHA (2004). Chronic Toxicity Summary for Silica (Crystalline, Respirable). Draft for Comment. http://www.oehha.ca.gov/air/chronic_rels/pdf/SILICAcREL_SRP2.pdf

[39] US EPA (1996). *Ibid*.

[40] National Environment Protection Council (NEPC, 2004). Review of the Practicability of a 10 Minute Sulfur Dioxide Standard Issues Paper March 2004. http://www.ephc.gov.au/nepms/air/air_nepm_so2_review.html.

[41] WHO (2000). Sulfur dioxide. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp194 – 198.

rapidly, within the first few minutes after inhalation, and continuing the exposure further does not seem to increase the severity of the effects.

There is a wide range of sensitivity both in normal subjects and compromised subjects. Results of studies with sensitive at risk groups have been used to establish ambient and indoor air quality guidelines or standards.

The table that follows, in which SO_2 standards and guidelines worldwide are summarised, has been adapted from NEPC (2004)[42].

Country	Averaging time	Maximum permissible concentration		
		ppm	µg/m³	Exceedances
Australia (NEPM standards and goals for 2008)	1 h	0.20	571*	1
	24 h	0.08	228*	1
	annual	0.02	57*	
New Zealand	1 h	0.122	350	
	24 h	0.042	120	
US (1997)	3 h	0.500	1300	
	24 h	0.140	365	
	1 year	0.030	80	
California	1 h	0.25	714	
	24	0.04	115*	
Canada (1989) (National Ambient Air Quality Objectives NOT Canada Wide Standards)	1 h	0.334	954*	maximum acceptable level
	24 h	0.115	328*	maximum acceptable level
	annual	0.023	66*	maximum acceptable level
WHO Guidelines (2000)	10 min	0.175	500	
	24 h	0.04	125	
	1 year	~0.018	50	
UK (2000)	15 min	0.100	266	35/year
	1 h	0.132	350	24/year
	24 h	0.047	125	3/year
EU (1999)	1 h	0.131	350	
	24 h	0.047	125	
	1 year	0.008	20	
NHMRC (1996)[43]	10 min#	0.25	700	

*: Calculated as outlined in Section 1.1.3 ; other values as reported by NEPC (2004)[44]

#: Incorrectly stated by NEPC (2004) to have been rescinded in 2002.

In describing the results of the critical study with normal, mild asthmatic and severe asthmatic volunteer subjects, the results of which have been

[42] NEPC (2004). *Ibid*.

[43] NHMRC (1996). Ambient air quality goals recommended by the National Health and Medical Research Council. www.nhmrc.gov.au/publications/pdf/rec1-2.pdf (Accessed March, 2005)

[44] NEPC (2004). *Ibid*.

predominantly used to establish reference values, WHO (2002)[45] states the following:

> *Only small changes, not regarded as of clinical significance, were seen at 572 µg/m^3 (0.2 ppm); reductions representing about 10% of baseline FEV_1* (an index of respiratory capacity or lung efficiency) *occurred at about 1144 µg/m^3 (0.44 ppm); and reductions of about 15% occurred at about 1716 µg/m^3 (0.6 ppm). The response was not greatly influenced by the severity of the asthma.*

This is the basis for WHO recommending guideline for SO_2 of 500 µg/m^3 (0.175 ppm) for a 10-min average exposure, although it refers to an earlier study with two asthmatic subjects in which small changes in airways resistance were reported at 286 µg/m^3 (0.1 ppm).

In interpreting the same experimental results, NEPC (2004) considered that the lowest concentration used of 0.2 ppm was the Lowest Observed Adverse Effect Level (LOAEL) for adverse effects associated with a 15-min average exposure to SO_2 for exercising asthmatics.

The standards set by NEPC are used as reference values in the health risk assessment.

2. Metals

2.1. Arsenic

Arsenic is metalloid element that is widely distributed in the earth's crust. Elemental arsenic is ordinarily a steel grey metal-like material that sometimes occurs naturally. However, arsenic is usually found in the environment as inorganic arsenic salts; arsenic combined with other elements such as oxygen, chlorine, and sulfur. Another form is organic arsenic; arsenic combined with carbon and hydrogen. The organic forms are usually less harmful than the inorganic forms.

Most arsenic compounds have no smell, and most have no special taste.

Inorganic arsenic occurs naturally in soil and in many kinds of rock, especially in minerals and ores that contain copper or lead. When these ores are heated in smelters, most of the arsenic goes up the stack and enters the air as a fine dust. Volcanic eruptions are another source of arsenic.

Most of the arsenic produced industrially is used as a preservative for wood to make it resistant to rotting and decaying in the form of chrome copper arsenate (CCA) – currently being phased out in Australia. Arsenic, as arsenic trioxide, is also used as a termiticide (against termites or white ants); some organic arsenicals (cacodylic acid, disodium methyl arsenate (DSMA), and monosodium methyl arsenate (MSMA) are also used as pesticides. Small quantities of elemental arsenic are added to other to form alloys with improved properties. The greatest use of arsenic in alloys is in lead-acid batteries. Another important use of arsenic compounds is in semiconductors and light-emitting diodes.

Inorganic arsenic can have acute, sub acute or chronic effects which may be either local (at the site of contact) or systemic (absorbed and transported in the blood stream elsewhere in the body and affect sites other that that of contact or exposure).

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[45] WHO (2000). Sulfur dioxide. *Ibid*.

The toxicity of inorganic arsenic compounds depends on the valence or oxidation state of the arsenic (-3, +3, or +5), as well as on the physical and chemical properties of the compound in which it occurs. Trivalent (As^{+3}) compounds such as arsenic trioxide (As_2O_3), arsenic trisulfide (As_2S_3), and sodium arsenite ($NaAsO_2$), are generally more toxic than pentavalent (As^{+5}) compounds such as arsenic pentoxide (As_2O_5), sodium arsenate (Na_2HAsO_4), and calcium arsenate (Ca_3 $(AsO_3)_2$) (ATSDR, 2000). The relative toxicity of the trivalent and pentavalent forms may also be affected by factors such as the water solubility of the compound.

Arsenic is a general metabolic poison that can affect a number of tissues and organs. Trivalent arsenic binds strongly to sulfur groups on protein molecules in the body and pentavalent arsenic substitutes for phosphate groups, which are important in oxidative phosphorylation (production of energy).

Inorganic arsenic has been recognized as a human poison since ancient times. Large oral doses (above 60 ppm in food or water; mg/kg and mg/L, respectively) can produce death. Lower amounts (0.3 to 30 ppm in food or water), may cause irritation of the stomach and intestines, with symptoms such as stomach ache, nausea, vomiting, and diarrhoea. Other effects include decreased production of red and white blood cells which may cause fatigue, abnormal heart rhythm, blood-vessel damage resulting in bruising, and impaired nerve function in hands and feet.

The most characteristic effect of long-term oral exposure to inorganic arsenic is a pattern of skin changes, called "black foot disease" (a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso). Some of the skin lesions may ultimately develop into skin cancer. Ingested arsenic may increase the risk of cancer in the liver, bladder, kidneys, prostate, and lungs.

Short term inhalation of high amounts of inorganic arsenic may cause a sore throat, irritated lungs and some skin effects seen after ingestion. Longer term exposure at lower concentrations can cause skin effects and circulatory and peripheral nervous disorders. There are some data suggesting that inhalation of inorganic arsenic may also interfere with normal fetal development, although this is not certain (ATSDR, 2000)[46].

Inhaled inorganic arsenic has been shown to increase the risk of lung cancer, mostly in workers exposed to arsenic at smelters, mines, and chemical factories, but also in residents living near smelters and arsenical chemical factories.

The International Agency for Research on Cancer (IARC, 1987)[47] has classified inorganic arsenic as carcinogenic to humans (Group 1). Similarly, in the United States of America (US) the Department of Health and Human Services (DHHS), the EPA and the National Toxicology Program (NTP) have classified inorganic arsenic as a known human carcinogen.

Levels of arsenic in ambient air from industrial emissions are highly unlikely to be sufficiently high to be acutely toxic.

OEHHA (2000[48], 2005[49]) has derived a short term reference exposure level (REL) value of 0.19 µg/m^3 averaged over 4 h based on reproductive and developmental

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[46] ATSDR (2000). Toxicological profile for arsenic. http://www.atsdr.cdc.gov/toxprofiles/tp2.pdf

[47] International Agency for Research on Cancer (IARC) - Summaries & Evaluations. ARSENIC AND ARSENIC COMPOUNDS (Group 1). Supplement 7: (1987) (p. 100). http://www.inchem.org/documents/iarc/suppl7/arsenic.html

[48] OEHHA (2000). Acute Reference Exposure Levels (RELs), Averaging Times, and Toxicologic Endpoints. http://www.oehha.ca.gov/air/acute_rels/allAcRELs.html

[49] OEHHA (2000). Chronic Reference Exposure Levels (RELs). http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html

effects of arsenic in animals and a chronic inhalational REL of 0.03 μg/m^3 based on effects on the cardiovascular and nervous system.

The chronic (annual average) reference concentration used for arsenic is 1 μg/m^3 published by the National Institute of Public Health and the Environment (RIVM, 2001)[50] of the Netherlands. RIVM states (page 27):

> *The most critical effect after chronic inhalation exposure of humans is lung cancer. The LOAEC* (Lowest Observed Adverse Effect Concentration) *for trivalent arsenic for this effect is 10 μg/m^3. For the variation in susceptibility of humans an extrapolation factor of 10 is used to derive a TCA* (Tolerable Concentration in Air) *for chronic inhalation exposure of 1 μg/m^3. It is proposed to use this TCA for both trivalent and pentavalent arsenic.*

The derivation is based on the observation that there is general consensus that the carcinogenic action of inorganic arsenic is based on a non-genotoxic mechanism; consequently health based exposure limits are derived based on the threshold model.

WHO (2000)[51] estimates a unit cancer risk for arsenic of 1.5 x 10^{-3} (μg/m^3)$^{-1}$ based on studies in exposed human populations in Sweden and the United States. Based on this estimate, an incremental lifetime cancer risk of one in one million is estimated to be associated with an air concentration of 0.66 ng/m^3.

The acute REL derived by OEHHA, the chronic TCA derived by RIVM and the unit cancer risk derived by WHO have been used as reference values in the health risk assessment.

2.2. Cadmium

Pure cadmium is a soft, silver-white metal. It occurs naturally in the earth's crust as various salts (mostly as complex oxides, sulfides, and carbonates in zinc, lead, and copper ores). The solubility of cadmium salts is variable, with the chlorides and sulfates the more soluble forms.

Cadmium is produced as a by-product of zinc- and sulfide-ore processing. The primary use of cadmium is in the production of nickel-cadmium batteries. It is also used for metal plating and in pigments and plastics. The major exposure source in the population in general is from food and tobacco.

Food and cigarette smoke are the biggest sources of cadmium exposure for people in the general population. Sources of cadmium in air are from uses, mining and smelting of cadmium as well as some industrial emissions. Occupational exposure to cadmium and cadmium compounds occurs mainly in the form of airborne dust and fume.

Cadmium predominantly affects the kidney causing tubular proteinuria and tubular dysfunction. It accumulates in kidney and liver. Cadmium has been associated with the development of cancer of lung and prostate in occupationally exposed workers. Similar effects have been reported in animal studies.

Based on occupational studies, WHO (2000)[52] estimates that a continuous lifetime exposure to cadmium at air concentrations if 300 μg/m^3 would not result in renal toxicity. However, WHO recommends a guideline value of 0.005 μg/m^3. This level is estimated to prevent further accumulation of cadmium in agricultural soil,

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[50] National Institute of Public Health and the Environment (RIVM, 2001). Reevaluation of human maximum permissible risk levels. RIVM Report No 71170/025.

[51] WHO (2000). Arsenic. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 125 – 127.

[52] WHO (2000). Cadmium. *Ibid*. pp136 -138.

hence prevent increases in dietary intakes of cadmium. By contrast OEHHA (2000)[53] has derived a chronic REL for cadmium of 0.02 μg/m^3 based on effects on the kidney and respiratory system.

An acute reference level for cadmium could be identified.

IARC (1993)[54] has classified cadmium and cadmium compounds as carcinogenic to humans (Group 1) by inhalation based on sufficient evidence of carcinogenicity both in humans and animals studies. However, WHO does not recommend a unit risk for deriving guideline values for cadmium in air.

the US EPA (1992)[55] derived a cancer unit risk of 1.8 x 10^{-3} (μg/m^3)$^{-1}$ for assign the cancer risks of cadmium.

The annual average derived by WHO and the cancer unit risk derived by the US EPA have been used as the reference values in health risk assessment.

2.3. Chromium^{6+}

Metallic chromium (Cr) is a steel-grey solid with a high melting point. Chromium occurs naturally in rocks, animals, plants, soil, and in volcanic dust and gases. It exists in several different valency forms, with the most common and stable forms being metallic chromium (chromium0 – Cr^0), trivalent chromium (or chromium III – Cr^{3+}), and hexavalent chromium (or chromium VI – Cr^{6+}).

Cr^{3+} occurs naturally in the environment and is an essential nutrient in both humans and animals. It is required by the body to promote the action of insulin and is essential for lipid, protein, and fat metabolism in animals and humans (ATSDR, 2000)[56]. Chromium deficiency causes changes in the metabolism of glucose and lipids and may be associated with maturity-onset diabetes, cardiovascular diseases, and nervous system disorders (US EPA, 1998)[57].

Cr^{6+} compounds exist mostly as oxides and are strongly oxidising. They are toxic and carcinogenic, with a wide range of potencies by the various compounds (WHO, 2000)[58].

Cr^{6+} and Cr^0 are generally produced by industrial processes. Metallic Cr is used mainly for making steel and other alloys. The naturally occurring mineral chromite (Cr^{3+}) is used as brick lining for high-temperature industrial furnaces, for making metals and alloys, and chemical compounds.

Chromium compounds, mostly in Cr^{3+} or Cr^{6+} forms, are used for chrome plating, the manufacture of dyes and pigments, leather tanning, and wood preserving. Smaller amounts are used in drilling mud, rust and corrosion inhibitors, textiles, and toner for copying machines.

Chromium is found in the ambient air as dust particles mostly in the Cr^{3+} and Cr^{6+} forms, originating from natural sources, industrial and product uses, and burning of fossil fuels and wood. The most important source of chromium in the air is from ferrochrome production. The Cr^{6+} in the air reacts with dust particles and other chemicals and is eventually reduced to Cr^{3+}.

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[53] OEHHA (2000). Chronic Toxicity Summary. Cadmium and Cadmium Compounds. http://www.oehha.ca.gov/air/chronic_rels/pdf/7440439.pdf

[54] International Agency for Research on Cancer (IARC) - Summaries & Evaluations. Cadmium and cadmium compounds. (Group 1). Vol: 58 (1993) (p. 119). http://www.inchem.org/documents/iarc/vol58/mono58-2.html

[55] US EPA (1992). Cadmium. Integrated Risk Information System. http://www.epa.gov/IRIS/subst/0141.htm

[56] ATSDR (2000). Toxicological profile for chromium. http://www.atsdr.cdc.gov/toxprofiles/tp7.html

[57] US EPA (1998). Toxicological review of trivalent chromium (CAS No. 16065-83-1) In Support of Summary Information on the Integrated Risk Information System (IRIS). http://www.epa.gov/iris/toxreviews/0028-tr.pdf

[58] WHO (2000). Chromium. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 139 - 142.

Waste from electroplating, leather tanning and textile industries as well as those that make dyes and pigments can discharge both Cr^{3+} and chromium Cr^{6+} into water and soil.

Occupational exposure to chromium compounds has been studied in the chromate-production, chrome-plating and chrome pigment, ferrochromium production, gold mining, leather tanning, and chrome alloy production industries (US EPA, 1998a)[59].

The US EPA states that chromium is one of the most common contact sensitizers in males in industrialized countries associated with occupational exposures to numerous materials and processes, including chrome plating baths, chrome colours and dyes, cement, tanning agents, wood preservatives, anticorrosive agents, welding fumes, lubricating oils and greases, cleaning materials, and textiles and furs. Solubility and pH appear to be the primary determinants of the capacity of individual chromium compounds to elicit an allergic response, with Cr^{6+} a more potent allergen than Cr^{3+}.

Chromium causes irritant and allergic contact dermatitis when exposed to the skin, through direct cytotoxic effects and inflammatory response mediated by the immune system, respectively. Chromium allergic dermatitis is characterized by symptoms of erythema, swelling, papules, small vesicles, dryness, scaling, and fissuring.

Chronic inhalational exposure to Cr^{6+} leads to effects on the upper and lower respiratory tract and the kidneys, with the effects on the respiratory tract being the most sensitive.

The US EPA (1998a) has derived a chronic reference dose (RfC) of 0.1 $\mu g/m^3$ based on respiratory effects of chromium dusts in animal studies. The RfC is similar to the tolerable concentration, ie, exposure for a lifetime is unlikely to lead to adverse health effects.

In occupationally exposed workers, Cr^{6+} has been associated with lung cancer. Lung tumours have also been observed in animal studies exposed to Cr^{6+} by inhalation or intratracheal administration. Cr^{6+} also causes damage to the genetic material (ie, is genotoxic).

IRAC (1990)[60] has classified Cr^{6+} as carcinogenic to humans (Group I) by inhalation and metallic chromium and Cr^{3+} as Group 3 (not classifiable as to their carcinogenicity to humans).

Based on the occupational studies, WHO (2000)[61] has derived a cancer unit risk of 4×10^{-2} $(\mu g/m^3)^{-1}$. Based on this unit risk, the concentration of chromium in air associated with an incremental cancer risk of one in one million is 0.025 ng/m^3. The US EPA (1998a) has derived a unit cancer risk of 1.2×10^{-2} $(\mu g/m^3)^{-1}$. Based on this unit risk, the concentration of chromium in air associated with an incremental cancer risk of one in one million is 0.083 ng/m^3.

The RfC derived by the US EPA and cancer unit risk derived by WHO are used as the reference value in the health risk assessment.

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[59] US EPA (1998a). Toxicological review of hexavalent chromium (CAS No. 18540-29-9) In Support of Summary Information on the Integrated Risk Information System (IRIS). http://www.epa.gov/iris/toxreviews/0144-tr.pdf

[60] International Agency for Research on Cancer (IARC) - Summaries & Evaluations: chromium and chromium compounds Chromium[VI] (Group 1) Metallic chromium and chromium[III] compounds (Group 3). Vol.: 49 (1990) (p. 49). http://www.inchem.org/documents/iarc/vol49/chromium.html

[61] WHO (2000). Chromium. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 139 - 142.

2.4. Manganese

Pure manganese is a silver-coloured metal. It occurs naturally in many types of rock as oxides, sulfates and chloride compounds, usually associated with iron ores. Manganese also occurs in soil, sediments and water as a result of environmental contamination.

Metallic manganese is used in the manufacturing of steel, carbon steel, stainless steel, cast iron, and super alloys to increase hardness, stiffness, and strength. Manganese chloride is used in dyeing, disinfecting, batteries, and as a paint drier and dietary supplement. Manganese oxide is used in textile printing, ceramics, paints, coloured glass, fertilizers, and as food additives. Manganese dioxide is used in batteries and may also be generated from the welding of manganese alloys.

Annual averages of manganese in urban or rural air without significant pollution are reported to be in the range of 0.01 – 0.07 μg/m^3, and about 10 times higher near industrial activities that use manganese (WHO, 2000)[62].

Manganese is an essential element required for the normal functions of the body. It is important for bone mineralization, protein and energy metabolism, metabolic regulation, cellular protection from damaging free radical species, and the formation of glycosaminoglycans (ATSDR, 2000)[63]. Manganese acts as both a constituent of enzymes and as an enzyme activator. However, is also toxic at high doses.

The Food Standards Australia and New Zealand (FSANZ, 2004)[64] has recommended an upper limit for oral manganese intake of 11.5 mg/day stating:

> *High doses of manganese can result in neurotoxicity. This is a serious adverse effect, with the elderly especially sensitive. The margin between the recommended daily intake and adverse effects levels are small, therefore an upper limit is necessary. A level of 11.5 mg manganese/day (mean of UK and US) has been established as an upper limit for the purpose of developing a standard for a standard for FSMP.* (pp 92-93).

FASNZ is the Australian food regulatory body with the responsibility of providing nutritional advice, based on a variety of considerations, including the toxicological profile of the substances where appropriate.

A concentration of manganese in air of 575 μg/m^3 would result in a daily inhalational dose equivalent to an oral dose of 11.5 mg manganese per day (based on an inhalation rate of 20 m^3 of air per day for an adult).

Excessive manganese exposure by inhalation is associated with effects on the central nervous system. This is particularly apparent in miners, smelters and workers involved in the manufacture of dry batteries. The disease is often termed manganism and is characterised by various psychiatric and movement disorders. Clinical manifestations may include slowing motor function, increased tremor, reduced response speed, enhanced smelling sensations, possible memory and intellectual loss, and mood changes.

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[62] WHO (2000). Manganese. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 154 - 156

[63] ATSDR (2000). Toxicological Profile for Manganese CAS# 7439-96-5. September 2000. http://www.atsdr.cdc.gov/toxprofiles/tp151.html

[64] Food Standards Australia New Zealand (FSANZ, 2004). Proposal P242 Food for special medical purposes. Pp92-93. http://www.foodstandards.gov.au/_srcfiles/P242_FSMP_PFAR.pdf

The most severe neurological symptoms resemble those seen in Parkinson's disease and Wilson's disease. The disease of manganese toxicity progresses even after exposure to manganese stops

Respiratory effects such as pneumonitis and pneumonia and reduced libido are also associated with occupational manganese toxicity.

WHO (2000)[65] has derived an air quality guideline (annual average) of 0.15 µg/m^3 based on the neurotoxic effects observed in occupationally exposed workers. OEHHA (2005)[66] has established a chronic REL of 0.2 µg/m^3 also based on results of occupational health studies. ATSDR (2000)[67] derived a chronic MRL of 0.04 µg/m^3 for manganese in respirable dust, based on neurotoxic effects in occupational studies (same as used by WHO). However, ATSDR used a different method for estimating a NOAEL and an additional substance specific factor for the quality of the date base and possible reproductive effects in women. Other substance specific factors used in the calculations of the toxicity values were as used by WHO.

The guideline value derived by WHO was used as the chronic reference value in the health risk assessment.

2.5. Mercury

Mercury occurs naturally in the environment and exists in several forms: metallic mercury (also known as elemental mercury), inorganic mercury, and organic mercury.

Metallic mercury, the familiar liquid metal used in thermometers and some electrical switches, is a shiny, silver-white metal that is a liquid at room temperature. At room temperature, some of the metallic mercury will evaporate and form mercury vapours - the higher the temperature, the more vapours will be released from liquid metallic mercury.

Inorganic mercury compounds or mercury salts comprise compounds of mercury combined with other elements such as chlorine, sulfur, or oxygen; most are white powders or crystals, except for mercuric sulfide (also known as cinnabar) which is red and turns black after exposure to light.

Organic mercury compounds or organomercurials comprise compounds of mercury combined with carbon and hydrogen. The most common organic mercury compound in the environment is methylmercury

The most common natural forms of mercury found in the environment are metallic mercury, mercuric sulfide (cinnabar ore, which is mined and refined to metallic mercury), mercuric chloride, and methylmercury. Some microorganisms (bacteria and fungi) and natural processes can change the inorganic mercury in the environment to organic mercury compounds such as methylmercury. Methylmercury can build up in certain fish and marine mammals to high levels which can lead to mercury poisoning in people who eat the fish, eg, Minamata disease.

Metallic mercury is used in producing chlorine gas and caustic soda, and in extracting gold from ore or articles that contain gold. It is also used in thermometers, barometers, batteries, and electrical switches. Dental amalgams typically contain metallic mercury.

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[65] WHO (2000). Manganese. *Ibid*.

[66] OEHHA (2005). *Chronic toxicity summary. M*anganese and compounds. http://www.oehha.ca.gov/air/chronic_rels/pdf/mangnREL.pdf

[67] ATSDR (2000). Toxicological profile for manganese. *Ibid*.

Mercury compounds have a very wide range of chemical uses. Mercuric chloride is used as a wood preservative, a photographic intensifier, a dry battery depolarizer, a tanning agent for leather, and for separating lead from gold. Mercuric nitrate is used in the manufacture of felt, and in the manufacture of bronze.

Some inorganic mercury compounds are or have been used as fungicides. Inorganic salts of mercury, including ammoniated mercuric chloride and mercuric iodide, have been used in skin-lightening creams. Mercuric chloride is a topical antiseptic or disinfectant agent. In the past, mercurous chloride was widely used in medicinal products including laxatives, worming medications, and teething powders. It has since been replaced by safer and more effective agents. Other chemicals containing mercury are still used as antibacterials, eg, mercurochrome. Mercuric sulfide and mercuric oxide may be used to colour paints, and mercuric sulfide is one of the red colouring agents used in tattoo dyes.

Methylmercury and ethylmercury compounds were used to protect seed grains from fungal infections, but following mass poisoning accidents they have not been used since the 1970s.

Mercury is also found in various industrial emissions primarily from fossil fuel combustion, mining, and smelting, and from solid waste incineration. Another contributor to mercury in the environment is municipal solid waste (for example, from waste that contains discarded batteries, electrical switches, or thermometers). Elemental mercury (Hg^0) vapour is the dominant form in the atmosphere, followed by mercuric ionic species (Hg^{2+}) and methylmercury (CH_3Hg).

In poisoning incidents, some people who ate fish contaminated with large amounts of methylmercury or seed grains treated with methylmercury or other organic mercury compounds developed permanent damage to the brain and kidneys. Metallic mercury also affects the central nervous system. However, inorganic mercury is a less potent neurotoxicant since it does not easily pass from the blood into the brain (ATSDR, 1999)[68]. However, it can cause effects on the central nervous system (the expression "mad as a hatter" appears to relate to the neurotoxic effects of mercury in workers making hats from felt treated with mercury).

Symptoms of mercury intoxication include personality changes (irritability, shyness, nervousness), tremors, changes in vision (constriction or narrowing of the visual field), deafness, muscle incoordination, loss of sensation, and difficulties with memory.

The kidneys are also sensitive to the effects of mercury, because mercury accumulates in the kidneys and causes higher exposures to these tissues, and thus more damage. All forms of mercury can cause kidney damage at high enough doses. The effects of mercury on the kidney are reversible if not severe in the first place once the body clears itself of the mercury contamination.

Short-term exposure (h) to high levels of metallic mercury vapour in the air, as can happen in occupational exposure, can damage the lining of the mouth and irritate the lungs and airways, causing tightness of the chest, a burning sensation in the lungs, and coughing. Other symptoms include nausea, vomiting, diarrhoea, increases in blood pressure or heart rate, skin rashes, and eye irritation.

Ingested inorganic mercury can also damage the stomach and intestines, causing nausea, diarrhoea, or severe ulcers if swallowed in large amounts. Effects in

[68] ATSDR (1999). Toxicological Profile for Mercury CAS# 7439-97-6, March 1999. http://www.atsdr.cdc.gov/toxprofiles/tp46.html

children after they accidentally swallowed mercuric chloride included rapid heart rate and increased blood pressure.

OEHHA (1999)[69] has established an acute REL of 1.8 µg/m^3 for mercury based on behavioural deficits after in utero exposure to metallic mercury vapour in animals. OEHHA has also established a chronic REL of 0.09 µg/m^3 for both metallic mercury and mercury salts. The reference value is based on effects on the nervous system such as hand tremor, memory disturbances, neurobehavioral and autonomic dysfunction in humans (OEHHA, 2005)[70].

WHO (2000)[71] has recommended an ambient air quality guideline (annual average) for organic and inorganic mercury of 1 µg/m^3, based on the LOAEL for mercury vapour effects on central nervous system and kidneys.

For consistency in the assessment, the acute and chronic REL derived by OEHHA were used as the reference values in the health risk assessment.

2.6. Nickel

Nickel is a malleable, hard, silvery-white metal; it is the 24th most abundant element. Nickel and its compounds have no characteristic odour or taste.

Nickel combined with other elements, principally oxygen or sulfur as oxides or sulfides, occurs naturally in the earth's crust. It is found in all soil, and is also emitted from volcanoes. Nickel is also found in meteorites and on the ocean floor in lumps of minerals called sea floor nodules. The earth's core is composed of 6% nickel.

Nickel is released into the atmosphere during nickel mining and by industries that make or use nickel, nickel alloys, or nickel compounds. Nickel is also released into the atmosphere by oil-burning power plants, coal-burning power plants, and incinerators.

Nickel has properties that make it very desirable for combining with other metals, such as iron, copper, chromium, and zinc, to form alloys. Nickel alloys are used in making metal coins and jewellery and in industry for making items such as valves and heat exchangers. Most nickel is used to make stainless steel. Nickel compounds are also used for nickel plating, to colour ceramics, to make batteries, and as catalysts (substances that increase the rate of chemical reactions, but do not take part in the reaction).

About 99% of the daily absorbed nickel is estimated to come from food and water in no-smokers, while the estimate is 75% in smokers (WHO, 2000)[72]. Another source of nickel exposure is from consumer products made of nickel alloys or nickel-plated products, eg, jewellery. The most common airborne exposures to nickel compounds are to insoluble nickel compounds such as elemental nickel, nickel sulfide, and the nickel oxides from dusts and fumes. The solubility of nickel salts varies considerably.

Allergic skin reactions are the most common health effects of nickel, affecting about 2% of the male and 11% of the female population (WHO, 2000). Allergic reactions in the respiratory system also occur in occupational exposure to nickel.

Occupational exposure in the nickel refining industries has been shown to be associated with an increased risk of lung and nasal cancers, thought to be a

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[69] OEHHA (1999). Determination of Acute Reference Exposure Levels for Airborne Toxicants - Mercury (Inorganic), acute toxicity summary. http://www.oehha.ca.gov/air/acute_rels/pdf/HgA.pdf

[70] OEHHA (2005). Chronic toxicity summary, mercury, inorganic. http://www.oehha.ca.gov/air/chronic_rels/pdf/7439976.pdf

[71] WHO (2000). Mercury. *Ibid* pp 157 – 161.

[72] WHO (2000). Nickel. *Ibid*, pp 162 – 165.

consequence of inhalation of mixtures of nickel salts such as oxides, sulfides/sulfates and other soluble nickel salts for many years. Nickel oxide and nickel subsulfide have been shown to cause tumours in animal studies. The tumorigenic potency varies with solubility, chemical composition and particle surface properties, with the nickel ion considered to be the active species which causes the cancers in all cases. The IARC (1990)[73] has classified nickel compounds as carcinogenic to humans (Group 1), similarly to a number of other jurisdictions, based on sufficient evidence in human and animal studies.

Exposure to metallic nickel fine particles has not been shown to cause cancer in workers, although there is sufficient evidence of carcinogenicity in animal studies. Consequently, IARC (1990) classified nickel metal and alloys as possibly carcinogenic to humans (Group 2B).

Based on studies in occupationally exposed workers, WHO (2000) has derived a unit risk of 3.8×10^{-4} $(\mu g/m^3)^{-1}$ for nickel compounds, corresponding to an ambient air concentration of 0.0025 $\mu g/m^3$ being associated with an incremental cancer risk of one in one million.

OEHHA (1999)[74] derived an acute ERL of 6 $\mu g/m^3$, (1-h average) This value is based on acute effects on the respiratory system in metal plater workers with occupational asthma, such as small decreases in airway function tests. A similar, but slightly lower, value (1.6 $\mu g/m^3$) is derived if toxicity of the immune system in animal studies is taken as the critical adverse effect. The higher value of 6 $\mu g/m^3$ was used by OEHHA as the acute REL because of the higher confidence in a value derived in human studies compared with animal studies.

Soluble nickel compounds appear to be the greatest concern for acute health effects. They are absorbed as Ni^{2+} which competes with copper for binding to serum albumin and is systemically transported in this way. Exposure to nickel in occupational settings causes dermatitis and asthma in some individuals with repeated exposures. The nickel is thought to bind to proteins in the dermis of the skin and lead to formation of antibodies, hence sensitisation.

Asthmatics or atopic individuals may be especially at risk for developing nickel-induced asthma.

OEHHA (2005)[75] has also derived a chronic ERL of 0.1 $\mu g/m^3$ for nickel oxide (based on lung and lymphatic effects in male and female rats) and 0.05 $\mu g/m^3$ for nickel compounds (except nickel oxide), based on effects on the lung, nasal epithelial and lymphatic system in male and female rats. Similarly, ATSDR (2003)[76] derived a chronic MRL of 0.09 $\mu g/m^3$ based on the results of a number of studies in animals that identified inflammatory changes in the lungs as a critical effect by a number of nickel salts.

The acute REL derived by OEHHA and the chronic MRL were used as reference values in the health risk assessment.

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[73] IARC. International Agency for Research on Cancer (IARC) (1990). Summaries & Evaluations. Nickel and nickel compounds (Group 1) Metallic nickel (Group 2B) Vol.: 49 (1990) (p. 257)

[74] OEHHA (1999). Determination of Acute Reference Exposure Levels for Airborne Toxicants - Nickel and Nickel Compounds. Acute toxicity summary. March 1999 C – 236.
http://www.oehha.ca.gov/air/acute_rels/pdf/NiA.pdf

[75] OEHHA (2005). *Chronic Toxicity Summary* Nickel And Nickel Compounds Nickel Oxide.
http://www.oehha.ca.gov/air/chronic_rels/pdf/NiComp.pdf

[76] ATSDR (2003). Draft toxicological profile for nickel. http://www.atsdr.cdc.gov/toxprofiles/tp15.pdf

2.7. Selenium

Selenium, in its pure form of metallic grey to black crystals, is often referred to as elemental selenium or selenium dust. Elemental selenium is commercially produced, primarily as a by-product of copper refining.

Selenium is widely but unevenly distributed in the earth's crust, commonly found in rocks and soil combined as sulfide compounds or with silver, copper, lead, and nickel minerals as oxides. Some selenium compounds are gases.

Selenium and its compounds are used in some photographic devices, gun bluing (a liquid solution used to clean the metal parts of a gun), plastics, paints, anti-dandruff shampoos, vitamin and mineral supplements, fungicides, and certain types of glass. For example, selenium sulfide is used in anti-dandruff shampoos by the common trade name Selsun Blue. Selenium is also used to prepare drugs and as a nutritional feed supplement for poultry and livestock. They are also used in the glass industry as decolourizing agents and in the rubber industry as vulcanizing agents.

Selenium occurs in four valence states: selenates (Se^{6+}), selenites (Se^{4+}), selenides (Se^{2-}), and elemental selenium (Se^{0}),

Selenium is an essential elements required for the normal function of the body. Selenium is a cofactor in important enzymes in the body. Deficiency can cause heart problems and muscle pain. Babies born early may be more sensitive to selenium deficiency, and this may contribute to lung effects.

The normal intake of selenium by eating food is enough to meet the essential requirement, unless food is grown in a selenium deficient area.

Exposure to high levels of selenium in air in the occupationally exposed workers has resulted in dizziness, fatigue, and irritation of mucous membranes (at concentrations higher than exposure limits). In extreme cases, collection of fluid in the lungs (pulmonary oedema) and severe bronchitis have been reported. Acute occupational exposure to SeO_2 resulted in bronchospasm, irritation of the upper respiratory passages, violent coughing, and gagging with nausea and vomiting.

Intentional or accidental swallowing of a large amount of sodium selenate or sodium selenite (for example, a very large quantity of selenium supplement pills) could be life-threatening without immediate medical treatment. Effects of ingestion of lower, but excessive levels of selenium, over long periods can cause selenosis, characterised by brittle hair, deformed nails discoloration and decay of the teeth and central nervous system disturbances, which in extreme cases includes loss of feeling and control (anaesthesia) of the extremities (arms and legs) (ATSDR, 2003)[77].

Industrial selenium compounds have been reported to cause rashes, redness, heat, swelling, and pain on contact with skin. Selenium dioxide, as dust or fumes in the workplace, have caused burning, irritation, and tearing of the eyes. These effects are unlikely to occur at levels of selenium in ambient air.

OEHHA (2001)[78] has derived a chronic inhalational REL of 20 µg/m^3. The REL is based on the results of epidemiological studies in China where it was shown that excessive intake of selenium caused liver, blood, skin and central nervous system toxic effects.

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[77] ATSDR (2003). Toxicological Profile for Selenium. http://www.atsdr.cdc.gov/toxprofiles/tp92.html

[78] OEHHA (2001). Determination of Noncancer Chronic Reference Exposure Levels. Selenium and Selenium Compounds, December 2001. http://www.oehha.ca.gov/air/chronic_rels/pdf/selenium.pdf

This REL derived by OEHHA is used as the reference value in the health risk assessment.

3. Organic emissions

3.1. 1,2,4 Trimethylbenzene and 1,3,5 Trimethylbenzene

Toxicological information on trimethylbenzene is very limited. The following is extracted from a US EPA (1994)[79] fact sheet.

1,2,4-Trimethylbenzene is a colourless, flammable liquid. It occurs naturally in coal tar and petroleum crude oil. It is a major component (typically 40%) of a petroleum refinery distillation fraction known as the C9 aromatic fraction (or simply the C9 fraction).

Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin. Breathing the vapour is irritating to the respiratory tract causing pneumonitis.

1,2,4-Trimethylbenzene is also the central nervous system depressant. Breathing high concentrations of the chemical vapour causes headache, fatigue, nervousness and drowsiness.

Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis.

The occupational exposure limits (TWA) for all isomers of trimethylbenzene is 25 ppm (~ 120 mg/m^3) based on symptoms of nervousness, tension and anxiety, and asthmatic bronchitis in exposed workers (OSHA, 2002)[80]. RIVM (2001)[81] derived a chronic TCA of 800 µg/m^3, about 150 times lower that the occupational exposure limit. The basis for the derivation of this value could not be identified.

3.2. Acetaldehyde

Acetaldehyde is a colourless, volatile liquid with a pungent suffocating odour; at dilute concentrations it has a fruity, pungent odour. The reported odour threshold is 0.09 mg/m^3 (IPCS, 1995)[82]. Acetaldehyde is a highly flammable and reactive compound that is miscible in water and most common solvents.

Acetaldehyde is a metabolic intermediate in humans and higher plants and a product of alcohol fermentation. It has been identified in food, beverages, and cigarette smoke. It is also present in vehicle exhaust and in wastes from various industries. Degradation of hydrocarbons, sewage, and solid biological wastes produces acetaldehyde, as well as the open burning and incineration of gas, fuel oil, and coal.

Levels of acetaldehyde in ambient air generally average 5 µg/m^3 (IPCS, 1995).

By far, the main source of exposure to acetaldehyde for the majority of the general population is through the metabolism of alcohol. Acetaldehyde has been implicated as toxic metabolite in the induction of alcohol-associated liver damage, facial flushing, and developmental effects. Cigarette smoke is also a significant source of exposure.

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[79] US EPA (1994). 1,2,4-TRIMETHYLBENZENE (CAS NO. 95-63-6). OPPT Chemical Fact Sheet EPA 749-F-94-022. Chemicals in the environment: Office of Pollution Prevention and Toxics, Us Environmental Protection Agency, August 1994. http://www.epa.gov/chemfact/

[80] OSHA (2002). http://www.osha.gov/dts/chemicalsampling/data/CH_273880.html; http://www.cdc.gov/niosh/pel88/25551-13.html

[81] RIVM (2001). *Ibid*.

[82] International Programme on Chemical Safety (IPCS, 1995). Acetaldehyde. Environmental Health Criteria, 167. http://www.inchem.org/documents/ehc/ehc/ehc167.htm

Toxic effects of acetaldehyde are similar to those of formaldehyde and at relatively low concentrations are limited principally to the sites of initial contact.

In limited studies on human volunteers, acetaldehyde was mildly irritating to the eyes and upper respiratory tract following exposure for very short periods to concentrations exceeding approximately 90 and 240 mg/m^3, respectively. Acetaldehyde also caused cutaneous erythema in patch testing on skin.

IPCS (1995)[83] derived an acute (24-h average) tolerable concentration (TC) of 2 mg/m^3 (2,000 µg/m^3) based on irritation in humans.

Increased incidences of tumours (nasal adenocarcinomas and squamous cell carcinomas) have been observed in inhalation studies in rats and hamsters exposed to acetaldehyde. All concentrations of acetaldehyde used in the studies induced chronic tissue damage in the respiratory tract, suggesting that, as for formaldehyde, tissue damage in the upper respiratory tract is a necessary prerequisite for the development of cancer.

IARC (1999)[84] has classified acetaldehyde as a possible carcinogenic to humans (Group 2 B) based on inadequate evidence in human, but sufficient evidence of carcinogenicity in animals.

IPCS (1995)[84], however, considered at the time that the mechanism of induction of tumours by acetaldehyde had not been well studied; hence, used both the threshold and non-threshold approaches to derive a reference value for the carcinogenic effects.

Based on the threshold model, IPCS derived a TC of 0.3 mg/m^3 using the NOAEL of 273 mg/m^3 for irritation of the upper respiratory tract in a 4-week study in rats and a substance specific factor of 1000 to account for the uncertainties in the derivation of the TC.

In its Guidelines for air Quality, WHO (2000a)[85] reported the TC as 0.05 mg/m^3 (50 µg/m^3) averaged over a year, quoting the IPCS document as the source. The reasons for the differences in the two values reported could not be readily discerned from the documentation available. However, it appears that the WHO value has been adjusted for continuous exposure from the experimental exposure of 6 h/day 5 days/week (adjusted value = 273 µg/m^3 x [6/24] x [5/7] = 48.75, rounded up to 50 µg/m^3).

The lower, adjusted value reported by WHO is used as the reference value for chronic effects of acetaldehyde.

Based on the non-threshold model, IPCS (1995)[84] derived a cancer unit risks that ranged between 1.5 x 10^{-7} and 9 x 10^{-7} (µg/m^3)$^{-1}$ from a number of studies. The IPCS estimates that acetaldehyde concentrations in air 1.1-6.5 µg/m^3 are associated with a one in a million incremental lifetime cancer risk.

Given the similarities in the carcinogenic profiles of acetaldehyde and formaldehyde, it would be appropriate to use either the acute or the chronic TC as a reference value to assess the risks of both cancer and non cancer effects of acetaldehyde. The induction of tumours in animals by formaldehyde is preceded by severe irritation and damage to the upper respiratory tract. The studies on acetaldehyde showed similar effects.

Nonetheless, acetaldehyde in included in the assessment of carcinogenic risks. The cancer unit risk of 9 x 10^{-7} (µg/m^3)$^{-1}$ is used in the risk assessment as it is

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[83] IPCS (1995). Acetaldehyde. *Ibid*.

[84] International Agency for Research on Cancer (IARC, 1999) - Summaries & Evaluations. Acetaldehyde (Group 2B) Vol.: 71 (1999) (p. 319). http://www.intox.org/databank/documents/chemical/acetalde/iarc534.htm

[85] WHO (2000a). Guidelines for air Quality, WHO, Geneva, 2000.

the more conservative reference value in the estimated range; hence, it more likely overestimates the risks.

It is likely that the effects of acetaldehyde, formaldehyde and acrolein are additive.

The acute TC derived by IPCS, the chronic TC derived by WHO and the upper value of the cancer unit risk derived by UPCS are used as reference values in the health risk assessment.

3.3. Acetone

Acetone is a highly volatile, highly water-soluble aliphatic ketone. It is a colourless liquid with a distinct smell and taste. The odour threshold of acetone in air at 100 to 140 ppm (240 – 330 mg/m^3), though some people can smell it at much lower levels (ATSDR, 1994)[86]. Acetone evaporates readily into the air and mixes well with water.

Most acetone produced is used to make other chemicals that are then used to make plastics, fibres, and drugs. Acetone is also used as a general solvent either alone or mixed with other solvents.

Acetone occurs naturally in plants, trees, volcanic gases, and forest fires. People and animals breathe out acetone produced from the natural breakdown of body fat. Acetone is also released during its manufacture and use, in exhaust from automobiles, and from tobacco smoke, landfills, incineration of waste materials and in industrial emissions.

Several consumer products, including certain nail polish removers, particle board, some paint removers, many liquid or paste waxes or polishes, and certain detergents or cleansers, contain acetone.

As a solvent, acetone causes irritation of the nose, throat, lung, and eyes. At much higher levels, it has anaesthetic properties and it causes central nervous system toxicity with headache, lack of energy, light headedness, dizziness, unsteadiness, and confusion, some mild behavioural effects and unconsciousness. Acetone is also a skin irritant, causing cellular damage when applied as a liquid directly to the skin at high concentrations.

Accidental or intentional ingestion of acetone can cause erosions in the mouth, coma, and diabetes-like symptoms in humans. Oral administration of large amounts of acetone in animals causes bone marrow hypoplasia (fewer new cells being made), degeneration of kidneys, heavier than normal livers and bigger liver cells, and collapse and listlessness. There is also some evidence from animal studies that acetone can affect reproduction and fetal development. Detection of acetone odour and feelings of irritancy are good warning signs for more serious effects.

One of the most studied effects of acetone is the induction of microsomal enzymes (particularly of cytochrome P-450IIEl). Because of this induction it can potentiate the toxicity of a number of other chemicals by enhancing their metabolism to reactive intermediates.

Men may be more susceptible than women to the haematological, hepatic, and renal effects, and effects on reproductive organs. People with pre-existing

[86] ATSDR (1994).Toxicological profile for acetone U.S. Department of Health and Human Services. Public Health Service Agency for Toxic Substances and Disease Registry May 1994. http://www.atsdr.cdc.gov/toxprofiles/tp21.html

haematological, liver, kidney, or reproductive disorders as well as the very young and the elderly may be more susceptible (ATSDR, 1994) [87].

ATSDR (1994) has established an acute MRL of 26 ppm (67 mg/m^3) based on a LOAEL of 237 ppm for four hours in humans at which neurobehavioral effects were reported. ATSDR also established a chronic MRL of 13 ppm (34 mg/m^3) based on a LOAEL of 1250 ppm over six weeks in humans for neurological effects. A higher substance specific adjustment factor was used in the derivation of the chronic MRL (cf acute MRL) because of the intermediate duration of the study used.

The acute and chronic MRL values derived by ATSDR were used as reference4 values in the health risk assessment.

3.4. Acrolein

Acrolein is a clear or yellow liquid with a disagreeable (pungent, suffocating) odour. Its odour thresh colourless to straw-coloured liquid old is 0.16 ppm (400 µg/m^3). It dissolves in water very easily and is volatile; quickly changing to a vapour with increasing temperature (can reach toxic concentrations in air at room temperature).

It also burns easily to produce toxic gases (peroxides and oxides of carbon). Small amounts of acrolein can be released to air from the combustion of animal and vegetable fats, plants, tobacco, and fossil fuel (petrol, oil, coal) (IARC, 1985). Acrolein produced in property and bush fires may pose acute effects to fire fighters.

Acrolein is used in the synthesis of other chemicals and as a pesticide to control algae, weeds, bacteria, and molluscs.

Acrolein is toxic by all exposure routes. Exposure causes inflammation and irritation of the skin, respiratory tract, and mucous membranes. Systemic effects may occur after exposure by any route. The skin, eyes, and mucous membranes irritation can be severe at sufficiently high quantities of acrolein, at which chemical burns can result.

The mechanism by which acrolein produces toxic symptoms is not known, but the compound is highly reactive. It has also been shown to suppress pulmonary antibacterial defences, to release oxygen radicals, and to react with proteins.

Effects on the respiratory system include irritation increased airway resistance and tidal volume, and decreased respiratory frequency, and delayed pulmonary oedema abd death (≥ 10 ppm; ≥ 25 mg/m^3). While the irritation is immediate, but pulmonary oedema may be delayed and respiratory insufficiency may persist for up to 18 months after exposure. It is also affects the cilia in the respiratory passages, reducing the effectiveness of the clearing function of the cilia.

Inhalation may also cause an asthmatic reaction in sensitized individuals.

Persons with pre-existing eye, skin, respiratory, allergic, asthmatic or heart diseases might be at increased risk from acrolein exposure in air.

OEHHA (1999)[88] has derived an acute REL (1-h average) for acrolein of 0.09 ppb (0.00009 ppm or 0.19 µg/m^3) based on a LOAEL of 0.06 ppm for eye irritation (subjective reporting) in healthy volunteers exposed for 5 min (concentration

87 ATSDR (1994).Toxicological profile for acetone. *Ibid*.

[88] OEHHA (1999). Determination of Acute Reference Exposure Levels for Airborne Toxicants – Acrolein. Acute toxicity summary CAS Registry Number: 107-02-8 March 1999 C – 2. http://www.oehha.ca.gov/air/acute_rels/pdf/107028A.pdf

adjusted for time over 1 h) and appropriate, composite substance specific adjustment factor of 60. OEHHA considered that the acute REL is protective against more serious irritant effects on eyes, skin, nose and lungs that occur at higher concentrations.

There is limited evidence on the health effects of chronic exposure to acrolein in humans (OEHHA, 2000)[89]. In several animal species exposed to acrolein in air for variable periods up to 52 weeks, the observed irritant effects of acrolein on the upper and lower respiratory tract were consistent between studies and species. Some of the histological changes reported were similar to the changes observed with exposure to acetaldehyde and formaldehyde. The effects were in the main localised at the point of contact, although there was some evidence of systemic effects also occurring, including effects on the immune system.

It is likely that the effects of acrolein, acetaldehyde and formaldehyde are additive.

IARC (1995)[90] has described acrolein as not classifiable as to its carcinogenicity to humans (Group 3) based on inadequate animal and human evidence.

OEHHA (2000)[90] states that persons with pre-existing eye, skin, respiratory, allergic, asthmatic or heart diseases might be at increased risk due to acrolein exposure. Individuals with cystic fibrosis or asthma should be excluded from acrolein exposure. Cancer patients treated with cyclophosphamide could be at increased risk because acrolein is a metabolite of cyclophosphamide

OEHHA (2000)[90] derived a chronic REL (annual average) for acrolein of 0.00003 ppm (0.03 ppb or 0.06 µg/m^3) based on a LOAEL of 0.4 ppm for histological lesions of the upper airways in rats exposed for 6 h/day, 5 days/week for 62 days and a composite substance specific adjustment factor of 300.

The acute and chronic REL derived by OEHHA are used as the reference values in the health risk assessment.

3.5. Benzene

Benzene is a colourless liquid with a sweet odour, with an odour threshold of between 1.5 to 4.7 ppm or 5 - 16 mg/m^3 (ATSDR, 1997)[91]. It is commercially derived from petrochemical and petroleum refining industries. Mean ambient air concentrations in rural and urban areas are about 1 µg/m^3 and 5 – 20 µg/m^3, respectively (WHO, 2000)[92].

Benzene is a by-product of various combustion processes, such as forest fires and the burning of wood, garbage, organic wastes, and cigarettes; it is also released to the air from crude oil leakages and volatilizes from plants.

Environmental exposure to benzene can occur during refuelling motor vehicles, from motor vehicle emissions (particularly extended travel in car with elevated benzene levels). WHO (2000)[93] estimates that exposure from cigarette smoke and car travel can contribute about 30% to the daily exposure to benzene of the general urban population.

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[89] OEHHA (2000). Determination of Noncancer Chronic Reference Exposure Levels. *Chronic toxicity summary* - acrolein *(2-propenal, acraldehyde, allyl aldehyde, acryl aldehyde)* CAS Registry Number: 107-02-8. Batch 2A December 2000 A – 1. http://www.oehha.ca.gov/air/chronic_rels/pdf/107028.pdf

[90] International Agency for Research on Cancer (IARC, 1995) - Summaries & Evaluations. Acrolein (Group 3). Vol.: 63 (1995) (p. 337) CAS No.: 107-02-8. http://www.inchem.org/documents/iarc/vol63/acrolein.html

[91] ATSDR (1997). Toxicological Profile for Benzene. CAS# 71-43-2. http://www.atsdr.cdc.gov/toxprofiles/tp3.html

[92] WHO (2000). Benzene. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 62 - 66

[93] WHO (2000). Benzene. *Ibid*.

Benzene has been widely used as a multipurpose organic solvent. This use is now discouraged due to its high toxicity, including carcinogenicity. It has also been used in the manufacture of other chemicals. It is an important component of petrol.

Acute effects of short term exposure to high levels of benzene as such can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, unconsciousness and, at sufficiently high doses, death.

The primary toxicological effects of chronic benzene exposure are on the hematopoietic system (blood forming tissues). Neurological and reproductive/developmental toxic effects (birth effects and other reproductive toxicities in animals) are also of concern at slightly higher concentrations. Impairment of immune function and/or various type of anaemia may result from the toxicity on blood. The haematologic lesions in the bone marrow can lead to decreases in lymphocytes (white blood cells). Severe benzene exposures can also lead to life-threatening aplastic anaemia.

These lesions may lead to the development of leukaemia years after apparent recovery from the damage to blood tissues, of which the most common type is acute myeloblastic leukaemia, a disease characterized by a proliferation of cells morphologically indistinguishable from myeloblasts. Myoblast are immature myeloid cells that include red blood cells, platelets and some white blood cells.

IARC (1987)[94] has classified benzene as carcinogenic to humans (Group 1). Benzene is also genotoxic. WHO (2000)[95] has derived an inhalational cancer unit risk of 6×10^{-6} $(\mu g/m^3)^{-1}$ for benzene in air. Based on this estimate of the cancer unit risk, the concentration of benzene in air associated with an incremental cancer risk of one in one million is estimated to be 0.17 $\mu g/m^3$ (0.05 ppb).

OEHHA (1999)[96] has derived an acute REL (6-h average) of 1,300 $\mu g/m^3$ (0.4 ppm) based on the results of reproductive studies in rats, in which a NOAEL of 40 ppm was determined. The NOAEL was divided by a composite substance specific adjustment factor of 100 to derive the REL. This level is considered to be protective against more serious adverse and life threatening effects.

OEHHA (2005)[97] has derived a chronic (annual average) REL for benzene of 60 $\mu g/m^3$ (0.02 ppm) based on the results of studies in occupationally exposed male refinery workers in which a NOAEL of 0.53 ppm was identified for decreases in WBC counts. An appropriate substance specific adjustment factor was used in the calculation.

The acute and chronic REL derived by OEHHA and the cancer unit risk derived by WHO are used as reference values in the health risk assessment.

3.6. 2-Butanone (Methyl ethyl ketone)

2-Butanone, also known as methyl ethyl ketone (MEK), is a colourless liquid with a sweet, but sharp odour, with an odour threshold of 5 – 8 ppm (16 – 26 $\mu g/m^3$)

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[94] International Agency for Research on Cancer (IARC, 1999) - Benzene. Acetaldehyde (Group 1) Supplement 7: (1987) (p. 120). http://monographs.iarc.fr/htdocs/monographs/suppl7/benzene.html

[95] WHO (2000). Benzene. *Ibid*.

[96] OEHHA (1999). Determination of Acute Reference Exposure Levels for Airborne Toxicants – Benzene. Acute toxicity summary *(benzol; benzole; cyclohexatriene)* CAS Registry No: 71-43-2, March 1999 C – 38. http://www.oehha.ca.gov/air/acute_rels/pdf/71432A.pdf

[97] OEHHA (2005). Benzene. Chronic toxicity summary *(benzol; benzole; cyclohexatriene)* CAS Registry No: 71-43-2. http://www.oehha.ca.gov/air/chronic_rels/pdf/71432.pdf

(ATSDR, 2002)[98]. OEHHA (1999)[99] describe MEK as having a mean odour threshold of 16 ppm (range 2 – 85 ppm).

The primary use of MEK is as a solvent (found in mixtures with acetone, ethyl acetate, *n*hexane, toluene, or alcohols) in processes involving gums, resins, cellulose acetate, and cellulose nitrate. It is also used in the synthetic rubber industry, in the production of paraffin wax, and in household products such as lacquer and varnishes, paint remover, and glues.

Symptoms of acute MEK exposure include irritation of the eyes, nose, and throat (OEHHA, 1999). In human case studies, inhalation of MEK for its euphoric effect has also resulted in slight excitement, followed by drowsiness or unconsciousness at higher concentrations.

Occupationally exposed workers have complained of mild neurologic effects including headaches, dizziness, and nausea. However, these exposures were to multiple solvents. Human volunteers exposed to pure MEK did not report these symptoms

OEHHA (1999) has derived an acute REL (1-h average) of 13,000 µg/m^3. The REL is based on the results of studies with human volunteers exposed for 2 h in an inhalation chamber who reported eye, nose, and throat irritation (reported subjectively); tearing and sneezing at 270 ppm (about 800 mg/m^3). A relatively strong odour (described as unpleasant and irritating) was noted at 90 ppm; the odour threshold varies between 2 and 85 ppm. Irritation of eyes, nose, and throat became more severe as the concentration increased, which eventually led to lacrimation and sneezing sometime during the exposure. No consistent effects were observed in another study with concentrations up to 200 ppm for 4 h. The acute REL is lower than levels found to have more severe adverse effects (eg, reproductive effect).

OEHHA has not derived a chronic REL for methyl ethyl ketone.

The US EPA (2003)[100] states that limited information is available on the chronic effects of MEK in humans. Chronic inhalation studies in animals have reported slight neurological, liver, kidney, and respiratory effects. No information is available on the developmental, reproductive, or carcinogenic effects of MEK in humans. Developmental effects, including decreased fetal weight and fetal malformations, have been reported in mice and rats exposed to MEK by inhalation or ingestion.

The US EPA (2003) has derived a chronic RfC for MEK of 5 µg/m^3 based on the results of studies on the development of fetal mice. The US EPA states that this RfC replaces an earlier RfC of 1 mg/m^3 that was entered on IRIS 7/01/1992. This latter RfC is still available in some documents on the US EPA web site (US EPA, 1992)[101].

The acute REL derived by OEHHA and the RfC derived by the US EPA are used as reference values in the health risk assessment.

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[98] (ATSDR, 2002) Toxicological Profile for 2-Butanone. CAS# 78-93-3 July 1992. http://www.atsdr.cdc.gov/toxprofiles/tp29.html

[99] OEHHA (1999). Determination of Acute Reference Exposure Levels for Airborne Toxicants - Methyl Ethyl Ketone. Acute toxicity summary *(2-butanone, 3-butanone, methyl acetone, ethyl methyl ketone)* March 1999. http://www.oehha.ca.gov/air/acute_rels/pdf/78933A.pdf

[100] US EPA (2003). Toxicological review of methyl ethyl ketone (CAS No. 78-93-3) In Support of Summary Information on the Integrated Risk Information System (IRIS) September 2003. http://www.epa.gov/iris/toxreviews/0071-tr.pdf

[101] US EPA (1992). Methyl Ethyl Ketone (2-Butanone). http://www.epa.gov/ttn/atw/hlthef/methylet.html

3.7. Ethylbenzene

Ethylbenzene is an aromatic hydrocarbon manufactured by alkylation from benzene and ethylene. Ethylbenzene is a colourless organic liquid with a sweet, petrol-like odour; the odour threshold is reported as being 2 ppm (9.5 µg/m^3) (ATSDR, 1990)[102].

The greatest use - over 99 percent - of ethylbenzene is to make styrene, another organic liquid used as a building block for many plastics. It is also used as a solvent for coatings, and in making rubber and plastic wrap. It is also used in technical xylene as a solvent in paints and lacquers and in the rubber and chemical manufacturing industries. It is found in crude oils, refined petroleum products and combustion products (petrol contains about 2% ethylbenzene by weight).

Ethylbenzene levels in air at rural sites are generally less than 2 µg/m^3. Mean levels of ethylbenzene ranging from 0.74 to 100 µg/m^3 have been measured at urban sites.

Ethyl benzene is emitted to air in motor vehicle emissions, industrial emissions, and from building interiors in which materials containing it have been used, tobacco smoke, and fossil fuel combustion (eg, coal fired power stations).

The acute and chronic toxicities of ethylbenzene are low. The toxic effects in humans and animals relate to depression of the central nervous system (CNS) and to irritation of the mucous membranes and eyes. No data concerning carcinogenic or reproductive effects have been reported. Ethylbenzene does not have significant mutagenic properties or teratogenic effects.

Ethylbenzene has low acute and chronic toxicity for both animals and humans. It is toxic to the central nervous system and is an irritant of mucous membranes and the eyes. The threshold for these effects in humans after short single exposures was estimated to be about 430-860 mg/m^3 (100-200 ppm).

Inhalation of ethylbenzene for 13 weeks by rats and mice at concentrations up to 4300 mg/m^3 (1000 ppm) did not lead to histopathological lesions. The no-observed-effect level, based on increased liver weight in rats, was 2150 mg/m^3 (500 ppm).

Ethylbenzene is an inducer of liver microsomal enzymes. It is not mutagenic or teratogenic in rats and rabbits. No information is available on reproductive toxicity or carcinogenicity of ethylbenzene.

IPCS (1998)[103] derived a chronic exposure guideline value of 22 mg/m^3 (5 ppm) based on a NOAEL of 2150 mg/m^3 (500 ppm) for increased liver weights (only effect observed at the LOAEL of 750 ppm) in a 13 week study in rats. Long-term occupational exposure to ethylbenzene concentrations estimated to be of this order of magnitude did not cause adverse health effects in workers.

IARC (2000)[104] has classified ethylbenzene as possibly carcinogenic to humans (Group 2 B).

The chronic guideline derived by IPCS (WHO) is used as reference value in the health risk assessment.

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[102] ATDR (1999). Toxicological Profile for Ethylbenzene CAS# 100-41-4. July 1999. http://www.atsdr.cdc.gov/toxprofiles/tp110.html

[103] International Programme on Chemical Safety (IPCS, 1996). Ethylbenzene. Environmental Health Criteria 186. World Health Organization Geneva, 1996. http://www.inchem.org/documents/ehc/ehc/ehc186.htm

[104] International Agency for Research on Cancer (IARC, 2000). Ethylbenzene (Group 2B). Vol.: 77 (2000) (p. 227) CAS No.: 100-41-4. http://monographs.iarc.fr/htdocs/monographs/vol77/77-05.html

3.8. Formaldehyde

Formaldehyde (also known as methanal, methylene oxide, oxymethylene, methylaldehyde, and oxomethane) is a colourless, flammable gas at room temperature. It has a pungent, distinct door and may cause a burning sensation to the eyes, nose, and lungs at high concentrations (ATSDR, 1999)[105]. An odour threshold between 0.05 and 1.0 ppm (0.06 – 1.3 mg/m^3) has been reported for formaldehyde.

It is produced in very small amounts in our bodies as a part of our normal, everyday metabolism.

Formaldehyde is used in the manufacture of melamine, polyacetal, and phenolic resins. It is also used as a hardening and reducing agent, a corrosion inhibitor, a sterilizing agent, in laboratories for preserving tissues, and in embalming fluids (mixed with methanol and buffers).

Formaldehyde is found in higher concentrations in indoor air than ambient air. There are a number of sources for formaldehyde in the home including cigarette smoke and other tobacco products, gas cookers, open fireplaces and many products used every day around the house (antiseptics, medicines, cosmetics, dish-washing liquids, fabric softeners, shoe-care agents, carpet cleaners, glues and adhesives, lacquers, paper, and plastics). A major source of formaldehyde in the home is from manufactured or building pressed wood products (chipboard, wood veneers) and carpets, particularly in new homes or caravans. It is also used as a preservative in some foods.

Ambient air sources of formaldehyde include; motor vehicle exhaust, manufacturing plants that produce or use formaldehyde or substances that contain formaldehyde (eg. glues), petroleum refineries, coking operations, incineration, wood-burning, tobacco smoke and other indoor sources of formaldehyde. Smog in the lower atmosphere is a major source of formaldehyde.

Formaldehyde primarily affects the mucous membranes of the upper airways and eyes. It is a pungent smelling gas that can cause watery eyes; burning sensations in the eyes, nose and throat; nausea; coughing; chest tightness; wheezing; skin rashes and other irritating effects.

Formaldehyde affects people in various ways. Some people are very sensitive to formaldehyde (allergic contact dermatitis) while others may have no noticeable reaction at the same level of exposure. Sensitive people can experience symptoms at levels below 0.1 ppm (120 µg/m^3; 0.12 mg/m^3).

The OEHHA of the Californian EPA (OEHHA, 1999)[106] describe the acute effects of formaldehyde as follows (references removed).

> *Exposure to moderate levels of formaldehyde (1 - 3 ppm) can result in eye and upper respiratory tract irritation. Most people cannot tolerate exposures to more than 5 ppm formaldehyde in air; above 10-20 ppm symptoms become severe and shortness of breath occurs. High concentrations of formaldehyde may result in nasal obstruction, pulmonary oedema, choking, dyspnoea, and chest tightness.*

Long term exposure to elevated levels of formaldehyde in the occupational setting has been shown to result in upper and lower airway irritation and eye irritation in

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[105] Agency for Toxic Substances and Disease Registry (ATSDR, 1999). Toxicological Profile for Formaldehyde. http://www.atsdr.cdc.gov/toxprofiles/tp111.html

[106] Office of Environmental Health Hazard Assessment (OEHHA, 1999). Determination of Acute Reference Exposure Levels for Airborne Toxicants March 1999 – Formaldehyde, acute toxicity summary. http://www.oehha.ca.gov/air/acute_rels/pdf/50000A.pdf.

humans; and degenerative, inflammatory and hyperplastic changes of the nasal mucosa in humans and animals.

Formaldehyde causes cancer of the upper respiratory tract in experimental animals. There seems to be some evidence for a weak association between nasopharyngeal cancer and formaldehyde exposure in humans. The International Agency for Research on Cancer (IARC, 1995)[107] has classified formaldehyde as probably carcinogenic in humans (Group 2A) based on insufficient evidence in humans, but sufficient evidence in animal studies.

The evidence from animal studies suggests that formaldehyde-induced cancer will occur only at exposure levels that extensively damage epithelium tissue of the nose. Damage to the epithelial issue is a consequence of the irritant effects of formaldehyde on the nose. Thus, concentrations in air that do not cause irritation are highly unlikely to cause nasopharyngeal cancer.

It is likely that the effects of formaldehyde, acetaldehyde and acrolein are additive.

In setting the ambient air quality monitoring investigation levels for formaldehyde, NEPC (2004)[108] state:

> *The end points chosen were the irritation of the eyes and the upper respiratory tract. It was considered that by protecting persons from the irritative effects of formaldehyde, then they would be protected from the more serious nasal cellular changes in humans and animals and potential carcinogenic effects that are seen to arise in animals with long periods of formaldehyde exposure.*

NEPC set a 24-h average for formaldehyde on 0.04 ppm (54 µg/m^3). The WHO (2000)[109] recommends that exposure should not exceed 0.1 mg/m^3 (100 µg/m^3) also concluding that levels in air which do not cause irritation will also be protective against the risk of cancer. NHMRC (1996)[110] established an indoor air quality guideline of 0.1 ppm.

The NEPC has not set an annual average for formaldehyde. ATSDR (1999)[111] has established a chronic (365 days or longer) inhalational minimal risk level (MRL) of 0.008 ppm (11 µg/m^3) for formaldehyde, which will be used as the chronic reference value in the health risk assessment. The chronic inhalation MRL of 0.008 ppm was calculated based on a LOAEL of 0.24 ppm for mild nasal lesions in chemical factory workers and using an composite substance specific adjustment factor of 30 (3 for the use of a LOAEL and 10 for human variability). The ATSDR has also established an acute inhalational MRL of 0.04 ppm, which is the same as the value set by the NEPC.

The acute (24-h) standard by NEPC and the chronic MRL derived by ATSDR are used as reference levels in the health risk assessment.

3.9. Methylene Chloride (dichloromethane)

Methylene chloride, also known as dichloromethane, is a colourless liquid that has a mild sweet odour and evaporates easily. The odour threshold in air has been

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[107] International Agency for Research on Cancer (IARC, 1995) - Summaries & Evaluations Formaldehyde (Group 2A). Volume 62 (1995) (p. 217). http://www.inchem.org/documents/iarc/vol62/formal.html

[108] NEPC (2004). Formaldehyde. National Environment Protection (Air Toxics) Measure. http://www.ephc.gov.au/pdf/Air_Toxics/Form_Health_Review.pdf

[109] WHO (2000). Formaldehyde. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 87-91.

[110] NHMRC (1996). *Ibid*.

[111] Agency for Toxic Substances and Disease Registry (ATSDR, 1999). Toxicological Profile for Formaldehyde. http://www.atsdr.cdc.gov/toxprofiles/tp111.html

reported as 200 ppm or about 80 mg/m^3 (ATSDR, 2000)[112]. Methylene chloride does not appear to occur naturally in the environment.

Methylene chloride is widely used as an industrial solvent and as a paint stripper. It can be found in certain aerosol, pesticide products, some spray paints, automotive cleaners and other household products and is used in the manufacture of photographic film. It is also used in plastics processing and in extraction of fats and oils from food products.

Methylene chloride in air is released predominantly from its industrial and consumer uses.

Methylene chloride affects the central nervous system in humans causing central nervous system depression. Because it is metabolised to carbon monoxide, it also causes an increase in carboxyhemoglobin content of blood. Signs and symptoms of methylene chloride intoxication include changes in hearing and vision, dizziness, nausea, tingling or numbness of the fingers and toes, loss of coordination, and drunkenness. In most cases, the effects are readily reversible after exposure ends. Direct skin contact with methylene chloride liquid causes intense burning and mild redness of the skin. Studies in animals suggest that exposure to higher concentrations (> 8,000 ppm) can lead to unconsciousness and death.

In animals studies, methylene chloride is an eye irritant and causes the liver and kidney toxicity by inhalation, but similar effects have not been observed in humans. Methylene chloride does not appear to cause birth defects or affect reproduction, even at high concentrations.

Methylene chloride has not been shown to cause cancer in humans exposed to vapours in the workplace. However, methylene chloride by inhalation increased the incidence of lung and liver tumours cancer in animal studies. IARC (1999)[113] has classified methylene chloride in Group 2B, possibly carcinogenic to humans based on inadequate evidence in humans, but sufficient evidence in experimental animals.

OEHHA derived an acute (1-h average) REL of 14 mg/m^3 (4 ppm) based on subtle effects on the central nervous system in human volunteers (OEHHA, 1999)[114] and a chronic (annual average) REL of 400 µg/m^3 (0.1 ppm) based on elevated levels of carboxyhaemoglobin in workers (OEHHA, 2005)[115].

WHO (2000a)[116] derived a short term (24-h average) guideline value of 3000 µg/m^3 based on exposure that causes formation of carboxyhaemoglobin in workers.

ATSDR (2000)[117] derived an intermediate and chronic MRL of 0.3 ppm (1.1 mg/m^3) for methylene chloride based on experimental studies in animals.

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[112] Agency for Toxic Substances and Disease Registry (ATSDR, 2000). Toxicological Profile for methylene chloride. http://www.atsdr.cdc.gov/toxprofiles/tp14.html

[113] International Agency for Research on Cancer (IARC, 1999) - Summaries & Evaluations. Dichloromethane (Group 2B) Vol.: 71 (1999) (p. 251). CAS No.: 75-09-2. http://www.inchem.org/documents/iarc/vol71/004-dichloromethane.html

[114] Office of Environmental Health Hazard Assessment (OEHHA, 1999). Determination of Acute Reference Exposure Levels for Airborne Toxicants- Acute toxicity summary. Methylene chloride *(dichloromethane, methylene dichloride)* CAS Registry Number: 75-09-2 March 1999 C – 229. http://www.oehha.ca.gov/air/acute_rels/pdf/75092A.pdf

[115] Office of Environmental Health Hazard Assessment (OEHHA, 2005). Chronic toxicity summary. Methylene chloride *(dichloromethane, methylene dichloride)* CAS Registry Number: 75-09-2. http://www.oehha.ca.gov/air/chronic_rels/pdf/75092.pdf

[116] WHO (2000a). Guidelines for air Quality, WHO, Geneva, 2000

[117] Agency for Toxic Substances and Disease Registry (ATSDR, 2000). Toxicological Profile for methylene chloride. http://www.atsdr.cdc.gov/toxprofiles/tp14.html

The US EPA (1987)[118] derived a cancer unit risk of 4.7 x 10^{-7} $(\mu g/m^3)^{-1}$ based on the incidence of lung and liver tumours in mice. Based on this unit risk, an incremental lifetime cancer risk of one in one million is associated with a concentration of methylene chloride in air of 2 $\mu g/m^3$.

In addition to the acute and chronic risks of methylene chloride being assessed, carcinogenic risk has also been assessed. Generally speaking, substances classified as Group 2B by IARC are not assessed for carcinogenic risk using the non-threshold model. However, the approach taken in the screening risk assessment is consistent with being conservative and cautionary.

The acute (24-h average) guideline derived by WHO, the chronic MRL derived by ATSDR and the cancer unit risk derived by the US EPA are used as reference values in the health risk assessment.

3.10. Styrene

Pure styrene is a colourless liquid that evaporates easily and has a sweet smell, with an odour threshold of 70 $\mu g/m^3$. It often contains other chemicals that give it a sharp, unpleasant smell recognised at concentrations 3 – 4 times higher than the odour threshold (WHO, 2000)[119]. Styrene dissolves in some liquids, but dissolves only slightly in water.

Styrene is used mostly to make rubber and plastics (polystyrene). Products produced from styrene include packaging, insulation (electrical and thermal), fibreglass, pipes, automobile parts, drinking cups and other "food-use" items, and carpet backing. These products may contain some residue of unlinked styrene.

Styrene is released into the air from industries that make and use styrene. It is also released from automobile exhaust, cigarette smoke, building materials, and consumer products that may contain residual amounts of unlinked styrene.

Epidemiological and clinical studies on workers have demonstrated that inhalation exposure to styrene (both short and long term) may cause alterations of central nervous system function. The symptoms are typical of central nervous system depression, and appear to be the most sensitive end point for styrene exposure by inhalation (ATSDR, 1992)[120].

The effects include depression, concentration problems, muscle weakness, tiredness, and nausea. Styrene also causes irritation of the eyes, nose, and throat. The effects of short-term exposure are readily reversible after styrene exposure ends.

Studies in animals indicate that chronic styrene exposure causes liver and kidney effects and may induce cancer. Styrene has been shown to be genotoxic in some experimental studies and to affect chromosome structure in some workers (WHO, 2000)[121].

IARC (1994)[122] describes styrene as possibly carcinogenic to humans (Group 2B), based on inadequate evidence in humans and limited evidence in experimental animals.

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[118] US EPA (1987). Dichloromethane (CASRN 75-09-2). Integrated Risk Information System. http://www.epa.gov/iris/subst/0070.htm

[119] WHO (2000). Styrene. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 106 - 108

[120] Agency for Toxic Substances and Disease Registry (ATSDR, 1992).Toxicological Profile for Styrene. CAS# 100-42-5 September 1992. http://www.atsdr.cdc.gov/toxprofiles/tp53.html

[121] WHO (2000). Styrene. *Ibid*.

[122] International Agency for Research on Cancer (IARC, 1994) - Summaries & Evaluations Styrene (Group 2B) CAS No.: 100-42-5. Vol.: 60 (1994) (p. 233). http://www.inchem.org/documents/iarc/vol60/m60-06.html

WHO (2000) has derived a short term guideline (weekly average) for air of 260 µg/m^3 based on neurotoxic effects in workers. It has also recommended an a 30-min average guideline of 70 µg/m^3 for, based on the odour threshold.

OEHHA (2005)[123] has derived a chronic REL (annual average) of 900 µg/m^3 (0.2 ppm) based on a LOAEL of 15 ppm for effects on the central nervous system in workers as measured by memory and sensory/motor function tests.

The weekly average guideline derived by WHO and the chronic REL derived by OEHHA are used as reference values in the health risk assessment.

3.11. Toluene

Toluene (common name for methylbenzene) is a clear, colourless liquid with a distinctive smell. IPCS (1996)[124] reports that the odour threshold for toluene in human beings is estimated to be 9.4 mg/m^3 (2.5 ppm); ATSDR (2000)[125] reports that the odour threshold is 8 ppm. Toluene is volatile, flammable, and explosive in air.

Toluene is a commercially-important intermediate chemical produced throughout the world in large quantities. Toluene is used in the production of other chemicals; in making paints, paint thinners, fingernail polish, lacquers, adhesives and solvent based cleaning agents; pharmaceutical products and as an additive in cosmetic products; and rubber and in some printing and leather tanning processes.

It is produced in the process of making oetrol and other fuels from crude oil, in making coke from coal, and as a by-product in the manufacture of styrene. Other sources of toluene emissions to air include: motor vehicles, aircraft, petroleum refineries and terminals, service stations, lawn mowers and other petrol-fuelled implements, chemical industry, rubber manufacturers, manufacture and use of paints, varnishes and lacquers, metal degreasing, printing and tobacco smoke (NEPC, 2004)[126].

In urban areas, a toluene level in ambient air of 0.0001 - 0.204 mg/m^3 has been detected (IPCS, 1996). Background levels monitored at sites throughout the world indicate that the general population is exposed to trace levels (0.00075 mg toluene/m^3).

The general population is exposed to toluene mainly through inhalation of vapour in ambient air, cigarette smoking, and, to a minor extent, by ingestion of food or water contaminated with toluene.

Toluene primarily affects the central nervous system (CNS), with effects typical of those of narcotic drugs - initial excitability followed by a depression in response. . It also causes transient eye (tearing at higher doses) and respiratory tract irritation.

The effects of toluene (increasing in severity with increasing concentrations and duration of exposure) include fatigue and drowsiness, mild throat and eye irritation, some impairment of cognitive function, headache, dizziness, and

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[123] Office of Environmental Health Hazard Assessment (OEHHA, 2005). *Chronic toxicity summary.* Styrene *(ethenylbenzene, phenylethylene, vinylbenzene)* CAS Registry Number: 100-42-5. http://www.oehha.ca.gov/air/chronic_rels/pdf/100425.pdf

[124] International Programme on Chemical Safety (IPCS, 1996). Toluene. Environmental Health Criteria 82. World Health Organization Geneva, 1996. http://www.inchem.org/documents/ehc/ehc/ehc186.htm

[125] ATSDR (2000). Toxicological Profile for Toluene CAS# 108-88-3 September 2000 http://www.atsdr.cdc.gov/toxprofiles/tp56.html

[126] National Environmental Protection Council (NEPC, 2004). National Environment Protection (Air Toxics) Measure Toluene, 2004. http://www.ephc.gov.au/nepms/air/air_toxics.html

sensation of intoxication lacrimation, loss of sensation in the skin (paraesthesia), gross signs of incoordination, and mental confusion. Effects appear to be reversible on cessation of exposure. Exposure to very high concentrations (above 15,000 mg/m^3) leads to narcosis and death.

Neurological effects have also been observed after repeated occupational exposures over a period of years. Toluene-containing mixtures have been implicated in the development of peripheral neuropathy but, in most cases, known neurotoxins such as *n*-hexane or methyl ethyl ketone have been present, and the role of toluene is not clear (IPCS, 1996)[127].

Irreversible neurological effects, such as encephalopathy, optic atrophy, and equilibrium disorders have been described in adult chronic toluene abusers who appear to be routinely exposed to toluene concentrations in excess of 3750 mg/m^3. There is also some evidence that toluene may be toxic to the liver and kidneys in abusers.

There is some evidence that toluene causes cleft palate in foetal mice after oral administration on days 6 - 15 of gestation.

Toluene does not appear to be carcinogenic in humans. IARC (1999)[128] describes toluene as not classifiable as to its carcinogenicity to humans (Group 3) because there is inadequate evidence in humans for the carcinogenicity of toluene and there is evidence suggesting lack of carcinogenicity of toluene in experimental animals. Toluene does not appear to be genotoxic.

NEPC (2004)[129] derived acute (24-h average) and chronic (annual average) monitoring investigation levels of 1 ppm (4113 µg/m^3) and 0.1 ppm (411 µg/m^3), respectively.

WHO (2000)[130] determined a weekly average guideline value of 260 µg/m^3 for protection against central nervous system and reproductive effects. WHO also suggest a 30-min average air quality guideline of 1 mg/m^3 which is the same as the 24-h average derived by NEPC.

OEHHA (2005)[131] derived a chronic REL of 300 µg/m^3 (0.7 ppm), which it considered protective against adverse effects on the central nervous system. This REL is comparable to the chronic reference values by WHO and NEPC.

The acute and chronic monitoring investigation levels derived by NEPC are used as reference values in the health risk assessment.

3.12. Vinyl chloride

Vinyl chloride, known also as chloroethene, chloroethylene, ethylene monochloride, or monochloro ethylene, it is a colourless gas at room temperature. It has a mild, sweet odour, with an odour threshold of 3,000 ppm in air.

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[127] IPCS (1996). Toluene. *Ibid*.

[128] International Agency for Research on Cancer (IARC) - Summaries & Evaluations. Toluene (Group 3) CAS No.: 108-88-3. Vol.: 71 (1999) (p. 829) http://www.inchem.org/documents/iarc/vol71/030-toluene.html

[129] NEPC (2004). National Environment Protection (Air Toxics) Measure Explanatory document April 2004. http://www.ephc.gov.au/pdf/Air_Toxics/FinalAirToxicsNEPM.pdf

[130] WHO (2000). Toluene. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 112 - 114.

[131] OEHHA (2005). *Chronic toxicity summary.* Toluene *(Methyl benzene; methyl benzol; phenyl methane; toluol).* CAS Registry Number: 108-88-3. http://www.oehha.ca.gov/air/chronic_rels/pdf/108883.pdf

Vinyl chloride is a manufactured substance that does not occur naturally. It is used to make the polymer polyvinyl chloride (PVC), which consists of long repeating units of vinyl chloride. PVC is used to make a variety of plastic product including pipes, wire and cable coatings, packaging materials, furniture and automobile upholstery, wall coverings, house ware, and automotive parts. Vinyl chloride has also been used as a coolant, as a propellant in spray cans, and in some cosmetics.

Most of the vinyl chloride that enters the environment comes from vinyl chloride manufacturing or processing plants; it is also a component of industrial emissions.

The primary acute toxicological effect of vinyl chloride inhalation is depression of the central nervous system (narcotic effects). Symptoms include dizziness and sedation. On long term exposure to high level in the occupational setting it can affect the liver, the immune system, circulation of blood and damage nerve cells. Effects on male and female reproductive functions have also been reported, but not birth defects.

Very high levels of vinyl chloride in experimental animals can damage the liver, lungs, and kidneys. These levels can also damage the heart and prevent blood clotting. Additional effects in animal studies include damage to sperm and testes, miscarriages early in pregnancy, and decrease fetal weight and development.

Several independent but mutually confirmatory studies have shown that exposure to vinyl chloride results in an increased carcinogenic risk in humans, involving the liver, brain, lung and haemo-lymphopoietic system. The incidence of liver and mammary gland tumours is also increased in animal studies. Vinyl chloride is also genotoxic.

Children up to the age of 10 may be more susceptible to the effects of vinyl chloride.

IARC (1979)[132] classified vinyl chloride as carcinogenic to humans (Group 1).

OEHHA (1999)[133] derived an acute REL (1-h average) of 180 mg/m^3 (72 ppm) based on subjective reports of mild headaches and dryness of eyes and nose in studies with healthy human volunteers. OEHHA has not derived a chronic REL for vinyl chloride.

WHO (2000)[134] derived a cancer unit risk for vinyl chloride of 1 x 10^{-6} ($\mu g/m^3$)$^{-1}$.

The US EPA (2000)[135] derived an inhalation RfC of 100 $\mu g/m^3$ based on effects on the liver in an experimental study in rats in which a NOAEL of 2.5 $\mu g/m^3$ and a LOAEL of 25.3 $\mu g/m^3$ (continuous human exposure concentrations) were established.

The acute REL derived by OEHHA, chronic RfC derived by the US EPA and the cancer unit risk derived by WHO are used as reference values in the health risk assessment.

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[132] International Agency for Research on Cancer (IARC, 1979) - Summaries & Evaluations. Vinyl chloride, polyvinyl chloride and vinyl chloride-vinyl acetate copolymers. Vol.: 19 (1979) (p. 377). http://www.inchem.org/documents/iarc/vol19/vinylchloride&polymers.html

[133] OEHHA (1999). Determination of Acute Reference Exposure Levels for Airborne Toxicants. Acute toxicity summary Vinyl Chloride *(chloroethene; chloroethylene; vinyl chloride monomer; VC; VCM)* CAS Registry Number: 75-01-4 March 1999 C - 345 - Vinyl Chloride. http://www.oehha.ca.gov/air/acute_rels/pdf/75014A.pdf

[134] WHO (2000). Vinyl Chloride. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 118 - 121

[135] US EPA (2000). Vinyl chloride (CASRN 75-01-4). Integrated Risk Information System. http://www.epa.gov/iris/subst/1001.htm

3.13. Xylenes

Xylene, dimethyl benzene, is a colourless, oily, sweet-smelling liquid. The odour threshold in air has been reported to be about 0.08–3.7 ppm.

Xylenes exist in ambient air as a mixture of ortho, meta and para isomers in which the methyl groups vary on the benzene ring: *meta*-xylene, *ortho*-xylene, and *para*-xylene (*m*-, *o*-, and *p*-xylene).

Xylene is primarily a synthetic chemical. Sources of xylenes include petrol, motor vehicles, petroleum refineries and terminals, service stations, lawnmowers and other petrol-fuelled implements, chemical manufacture, polyester manufacture, manufacture and use of paints, dyes, and lacquers, wood burning stoves and fireplaces.

The first signs of adverse effects on humans are irritation of the nose, throat and eyes, followed by irritation of the lower respiratory tract that can lead to difficulty in breathing and problems with the lungs.

Xylenes also affect the central nervous systems (narcotic effect) causing headaches, lack of muscle coordination, dizziness, confusion, delayed reaction time; memory difficulties, and changes in one's sense of balance. It can also cause stomach discomfort and possibly changes in the liver and kidneys. It can cause unconsciousness and death at very high levels.

Xylenes have been shown to cause increased numbers of fetal deaths and delayed growth and development when administered in high concentrations to pregnant experimental animals.

IARC (1999)[136] described xylenes as not classifiable as to their carcinogenicity to humans (Group 3) based on inadequate human and animal evidence of carcinogenicity.

NEPC (2004)[137] chose irritation as the critical end point for xylenes, because it occurs at low levels after short exposures, and derived acute and chronic monitoring investigation levels of 1183 µg/m^3 (0.25 ppm, 24-h average) and 946 µg/m^3 (0.2 ppm, annual average), respectively.

The NEPC monitoring investigational levels are used as reference values in the health risk assessment.

4. Ammonia

Ammonia is a colourless gas with a very sharp odour, which is familiar to most people because it is used in smelling salts, household cleaners, and window cleaning products. It is also responsible for the typical smell of some blue cheeses.

Ammonia is very important to plant, animal, and human life. It is found in water, soil, and air, and is a source of much needed nitrogen for plants and animals. Most of the ammonia in the environment comes from the natural breakdown of manure and dead plants and animals.

Ammonia is widely used in industry as a feed stock for nitrogen based chemicals such as fertilizers, plastics and explosives.

Ammonia is a strongly alkaline, corrosive substance. The main toxic effects are restricted to the sites of direct contact (i.e., skin, eyes, respiratory tract, mouth,

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[136] International Agency for Research on Cancer (IARC, 1999) - Summaries & Evaluations Xylenes (Group 3). Vol.: 71 (1999) (p. 1189). http://www.inchem.org/documents/iarc/vol71/052-xylenes.html

[137] NEPC (2004). National Environment Protection (Air Toxics) Measure Explanatory document April 2004. http://www.ephc.gov.au/pdf/Air_Toxics/FinalAirToxicsNEPM.pdf

and digestive tract). Ammonia vapours cause irritation of the eyes and respiratory tract. Higher concentrations cause conjunctivitis, laryngitis, and pulmonary oedema, possibly accompanied by a feeling of suffocation. Contact with the skin may cause burns and blistering.

Persons with asthma and other respiratory ailments including underlying cardiopulmonary disease and persons with no tolerance, developed from recent exposures to ammonia, may be more susceptible to the irritant effects of ammonia.

OEHHA (1999)[138] derived an acute (1-h) REL of 3,200 µg/m^3 (4.5 ppm) based on eye and respiratory irritation in human volunteers. OEHHA has not derived a chronic REL.

The US EPA (1991)[139] derived a chronic RfC for ammonia of 100 µg/m^3 based on effects on the respiratory system occupational studies.

The acute REL derived by OEHHA and the chronic RfC derived by the US EPA are used as reference values in the health risk assessment

5. PAH

Polycyclic aromatic hydrocarbons (PAH) consist of a number of closely related individual chemicals (congeners) of complex chemical structures.

The main sources of non occupational exposure to airborne PAHs are from combustion processes; these include motor vehicles, petroleum refineries, power plants using fossil fuels, coking plants, bitumen and asphalt production plants, aluminium refineries, iron and steel foundries, crop residue and forest management burning, bushfires, smoke from open fireplaces, environmental tobacco smoke and cooking food. Exposure also occurs through ingestion of PAH containing foods, raw food does not normally contain high levels of PAHs, but they are formed by roasting, baking, frying or processing (NEPC, 2004)[140].

The major health concern with PAH is the development of cancer after long term exposure. Many individual PAH are carcinogenic to animals and may be carcinogenic to humans, and exposure to several PAH-containing mixtures has been shown to increase the incidence of cancer in human populations. There is concern that those PAH found to be carcinogenic in experimental animals are likely to be carcinogenic in humans. PAH produce tumours both at the site of contact and at distant sites (IPCS, 1998)[141].

IARC has classified a number of the individual PAH congeners variably as Group 3 (not classifiable as to carcinogenicity in humans, Group 2B (possibly carcinogenic to humans, and Group 2A (probably carcinogenic to humans) based on results of experimental studies in animals.

Some of the PAH congeners have similar toxicological profiles, but different toxicity potencies. For risk assessment purposes, it is assumed that all PAH congeners act through a common mechanism and their toxicity potencies are expressed as a ratio to the toxicity potency of benzo(a)pyrene, the reference compound, yielding toxicity equivalency factors (TEF) or relative potencies.

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[138] OEHHA (1999). Determination of Acute Reference Exposure Levels for Airborne Toxicants - Ammonia acute toxicity summary *(anhydrous ammonia, aqueous ammonia)* CAS Registry Number: 7664-41-7 March 1999 C – 13. http://www.oehha.ca.gov/air/acute_rels/pdf/7664417A.pdf

[139] US EPA (1991). Ammonia (CASRN 7664-41-7) Integrated Risk Information System (IRIS) January 1991. http://www.epa.gov/iris/subst/0422.htm

[140] NEPC (2004). Polycyclic aromatic hydrocarbons (PAHs). National Environment Protection (Air Toxics) Measure. http://www.ephc.gov.au/pdf/Air_Toxics/Form_Health_Review.pdf

[141] International Programme on Chemical Safety (IPCS, 1998). Selected non-Heterocyclic Polycyclic Aromatic Hydrocarbons. Environmental Health Criteria 202. http://www.inchem.org/documents/ehc/ehc/ehc202.htm

The TEF ratios are used to calculate the relative contribution to the overall dose (TEQ) from the concentration of each congener in the medium. That is, the concentration of the congener in the medium (air) is multiplied by the TEF to give a benzo(a)pyrene equivalent concentration and the TEF for each congener present added to give an overall a toxicity equivalent quotient (TEQ) – a single concentration equivalent to a benzo(a)pyrene concentration. Thus only one concentration (dose), equivalent to a dose of benzo(a)pyrene, is used in the risk assessment and compared to the toxicity profile and toxicity value for benzo(a)pyrene.

The following table listing the relative potencies of a number of PAH congeners and the extrapolated unit cancer risks has been reproduced from WHO (2000a)[142].

Table 3.5. Estimate of unit risks for several polycyclic aromatic hydrocarbons

Compound	Relative potency range compared to BaP	Unit risk $[mg/m^3]^{-1}$
Anthanthrene	0.28 - 0.32	$(2.4 - 2.8) \times 10^{-2}$
Benz[a]anthracene	0.014 - 0.145	$(1.2 - 13) \times 10^{-4}$
Benzo[a]pyrene	1.0	8.7×10^{-2}
Benzo[b]fluoranthene	0.1 - 0.141	$(0.87 - 1.2) \times 10^{-2}$
Benzo[j]fluoranthene	0.045 - 0.1	$(0.4 - 0.87) \times 10^{-2}$
Benzo[k]fluoranthene	0.01 - 0.1	$(8.7 - 87) \times 10^{-4}$
Chrysene	0.001 - 0.1	$(8.7 - 870) \times 10^{-5}$
Cyclopenta[*cd*]pyrene	0.012 - 0.1	$(1 - 8.7) \times 10^{-3}$
Dibenzo[a,e]pyrene	1	8.7×10^{-2}
Dibenz[a,c]anthracene	0.1	8.7×10^{-3}
Dibenz[a,h]anthracene	0.89 – 5.0	$(7.7 - 43.5) \times 10^{-2}$
Dibenzo[a,l]pyrene	100	8.7×10^{-0}
Dibenzo[a,e]fluoranthene	1.0	8.7×10^{-2}
Dibenzo[a,h]pyrene	1 - 1.2	$(8.7 - 10.4) \times 10^{-2}$
Dibenzo[a,i]pyrene	0.1	8.7×10^{-3}
Fluoranthene	0.001 - 0.01	$(8.7 - 87) \times 10^{-5}$
Indeno[1,2,3,-*cd*]pyrene	0.067 - 0.232	$(5.8 - 20.2) \times 10^{-3}$

WHO (2000)[143] has derived a cancer unit risk of 8.7×10^{-7} $(ng/m^3)^{-1}$ for benzo(a)pyrene. The corresponding concentration of benzo(a)pyrene or benzo(a)pyrene TEQ associated with an incremental lifetime risk of one in one million is 0.012 ng/m^3. WHO did not recommend a specific guideline for PAH as

[142] WHO (2000a). Guidelines for air Quality, WHO, Geneva, 2000

[143] WHO (2000). Polycyclic aromatic hydrocarbons. Air Quality Guidelines for Europe. Second Edition. WHO Regional Office Publications, European Series, No 91. pp 92-96

such in air as they occur typically as constituents of complex mixtures with differing concentrations of the individual congeners, depending on the source.

NEPC (2004) has derived a monitoring investigation level for benzo(a)pyrene of 0.3 ng/m^3 as an annual average.

The annual average monitoring investigating level derived by NEPC and the cancer unit risk derived by WHO are used as reference values in the health risk assessment

6. Limitations of the toxicity values

Generally, a conservative approach is taken in risk assessment to compensate for its limitations. The toxicity values tend to be underestimated (lower values) and exposure tends to be overestimated (higher values). This is to ensure, as far as possible, that overall risks to health are overestimated rather than underestimated.

However, there are uncertainties associated with the quality and quantity of the information available, extrapolating from animal studies to human environmental exposure, extrapolating from very high doses in animals to relatively low doses environmental exposure in humans, and human heterogeneity (individual factors that might affect response to chemicals). The health risk assessment process generally addresses these uncertainties by the use of chemical specific adjustment factors.

As far as possible or known, individual or groups who might be at increased risk or are especially sensitive to the effects of a particular chemical are taken into account when deriving the toxicity values. However, the amount of available information on each chemical and scientific understanding may limit the extent to which all sensitive individual can be identified and included in the assessment.

Notwithstanding, there is a reasonable degree of confidence in the outcomes that human health is protected if exposure is less than the reference toxicity value.

The toxicity values are based on toxicological effects, pathological changes and generally measurable (sometimes subjectively) adverse health effects in experimental or epidemiological studies. It does not assess health in the broader context of wellbeing. This is not unique to any one health risk assessment model. It is a limitation that applies generally to current risk assessment practices and the available information on which they are based.

Assessment of health in the broader context requires different methodologies, which have not been fully developed nor used to any great extent in Australia. The development of the proposed Health Impact Assessment in Australia will go some way towards addressing this issue, in particular engendering pro-activity and fostering cooperation between stakeholders and interested parties. Individual who appear not be protected by the outcomes of the health risk assessment can be identified and management options explored to mitigate any potential impact.

Thus, it is important to stress that health risk assessment is only one of the tools that inform decision-making in environmental issues. It is not a solution or an end in itself.

Highly sensitive receptors

Whilst the derivation of the reference values generally takes into account readily identifiable sensitive subgroups, they do not necessarily address the potential impacts on individuals who may be at increased risk because of some specific personal characteristic or disease state, or individuals who may be particularly

sensitised to chemical exposures, such as sufferers of multiple chemical sensitivity (MCS).

The Health and Emissions Reference Group has requested the author to provide a commentary on the syndrome of MCS. The following are private views of the author that reflect his personal understanding of the syndrome.

The syndrome of MCS does not fit any of the classical toxicological models used in risk assessment, although the process of sensitisation appears to follow similar developments as toxicologically definable sensitisation to chemicals in some cases.

Sufferers of MCS appear to be highly sensitised and react to a variety of disparate chemicals found in the environment and in the home. Once sensitised, they are affected by chemicals at concentrations that are much lower than would affect other individuals in the community. Moreover, they appear to react to levels of chemicals that are much lower than guidelines or standards and sometimes that cannot be measured analytically. Consequently, compliance with regulatory standards or toxicity values may not necessarily protect people who suffer from MCS.

A seemingly paradoxical observation is that MCS sufferers appear to react to a number of chemicals which may not have been involved in the sensitisation process and that are not chemically or physically related. However, given that the causative agents cannot be readily identified in most cases, it is unknowable whether or not the agents that trigger a response were involved in the sensitising process.

MCS appears to be a debilitating medical condition, although not necessarily a single illness entity. As such, the symptoms may be the results of the combined effects of more than one illness. An additional complicating factor is that the symptoms often are similar to, or the same as, those for other illnesses; they may also be generic or non specific.

MCS is difficult to diagnose and there do not appear to be any specific quantitative diagnostic investigations that can identify the causative agents, both in causing the sensitivity initially and triggering the subsequent reactions to the chemicals involved. The difficulty is compounded by a general lack of specifically trained medical specialist that could investigate and research the syndrome further, hence lead to a better understanding and management of the illness.

It does appear, however, that MCS requires a variety of carefully considered management strategies on a case-by-case basis. Each sufferer has a unique set of problems and will usually exhibit a number of symptoms when exposed to chemicals in air, food, water or consumer products. Because of the number of organs and systems that seem to be affected and the variety of symptoms suffered, it is possible that different medical specialist and para-medical specialists may be required in the management of the patient.

7. Exposure assessment

Ground level concentrations (GLC) for 27 individual compounds or groups of compounds were estimated using air dispersion modelling based on known or estimated emissions for alumina refinery for current operation and the proposed expansion at Wagerup (Section 3 Main document). The compounds modelled were selected on the basis of their hazardous characteristics and the estimated quantities in the emissions.

GLC concentrations were modelled for different averaging times of 1-h, 24-h and 12 months (annual). In addition, shorter averaging times were calculated for 3-min and 10-min averages (CSIRO, 2005)[144].

7.1. Averaging times

Broadly speaking, chemicals can have two types of effects, acute or chronic. Acute effects generally occur within a short time of coming in contact with relatively high levels of a substance. They can range from simple, mild irritation of mucous membranes, eyes or skin to serious organ damage and death at sufficiently high concentrations. At elevated concentrations that might be found in ambient air, chemicals are likely to have only minor acute effects. With spillage accidents or occupational exposure in the unregulated workplace, acute effects can be more serious. The time of the effect will depend on the chemical properties as well as the dose, but can range from immediately coming in contact with the chemical, for irritants for example, to a several hours of contact with the chemical for a systemic poison. For example, it takes about 6-8 h for the concentration of carboxyhaemoglobin in the blood to reach a steady state on exposure to CO (Section 1.2.1).

Chronic effects tend to occur after continued exposure for some time and at lower doses or concentrations than acute effects.

To address these types of effects reference criteria are expressed in term of averaging periods: ≤ 24-h averaging periods for acute effects; > 24-h averaging period (usually annual averages) for chronic effects. Generally, the lowest averaging time used for reference values is 1 h.

For some substances, such as strong irritants, duration of exposure is not the critical determinant for the effect to occur if the substance is present at concentrations above the threshold for the effect. Thus the effect may occur in the first few seconds or minutes of exposure and shorter averaging times such as 3 min averages and 10 min averages would be more appropriate to assess their potential risk.

Unfortunately guidelines or standards for short term averaging periods in this range are rarely established – a 10-min average concentration for sulfur dioxide is one of the few exceptions (see Section 1.2.4) – mainly because it may not be possible to measure substances over such short periods using current analytical techniques. Similarly, air dispersion models may not be able to estimate short term GLC for periods < 1 h.

The toxicological or epidemiological data may not be available for setting short-term reference values. Studies on irritants with human subjects in environmental chambers may be useful in extrapolating to short term averages, since concentrations can be controlled and maintained constant and the time the effects first occur can be noted by the subject or the experimenter.

In the absence of reference values for short-term averaging times, it is still possible to assess potential impacts over the shorter averaging periods, albeit in a limited way, by comparing with the reference values for 1-h or longer averaging period. If the estimated 3-min or 10-min average concentration are less that the reference value for the longer averaging time, then the substance is unlikely to pose a health risk. If on the other hand the short term average concentration exceeds the longer averaging period reference value, then the likelihood of adverse effects needs to be examined on a case by case basis.

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[144] CSIRO (2005). Meteorological and Dispersion Modelling Using TAPM for Wagerup Phase 3B: HRA (Health Risk Assessment) Concentration Modelling – Expanded Refinery Scenario *Prepared for* Alcoa World Alumina Australia *By* CSIRO Atmospheric Research Private Aspendale, Vic. Report C/0986 11 February 2005.

A comparison of modelled maximum 3-min and 10-min GLC reported in table 9 by CSIRO (2005)[145] with the reference values used in this risk assessment (Section 1.1.3) indicates that the short term averaging GLC are lower than the reference values for 1-h averages or annual averages (where no 1-h average was available). In most cases, the short term estimated GLC were lower that the reference values for annual averages.

These observations indicates that short peaks in the concentration of irritant substances in air are unlikely to be sufficiently high to cause adverse health effects at any of the sixteen receptor locations examined.

8. Risk characterisation

Once the chemicals of concern have been identified, the toxicity values defined and the ground level concentrations determined, hence exposure defined, the potential risks are assessed by comparing the estimated exposure with the reference toxicity values or guideline values.

The risks hazard quotient (HQ), the ratio of predicted exposure divided by the reference value, is calculated for each chemical of concern. Moreover, the HQ for each substance is summed to produce the hazard index (HI), which provides a measure of the cumulative impact of all the emissions assessed.

It is generally agreed that a chemical present at a concentration that results in a HQ less than one does not pose a health risk. Similarly, if the HI for a group of substances is less than one, then the group of substances does not pose a heath risk. The HQ and HI are a measure of the margin of safety, which is reflected in the size of the HI or the HQ - the smaller the HQ or HI, the larger the margin of safety. The HQ and HI are calculated for both cancer and non cancer effects.

If the HQ or HI exceeds one, it does not necessarily mean that the chemical or group of chemicals poses a health risk. In these cases, it is necessary to review the scientific data on which the reference toxicity value is based to assess the likelihood of an adverse effect. For example, the reference toxicity value may have been based on a serious, debilitating and irreversible adverse effect with a steep dose response curve (marked increases in severity or incidence with small increases in dose), in which case only relatively small exceedances may be tolerable. On the other hand the reference toxicity value may have been based on a relatively trivial and reversible effect or the dose response curve is flat (small increases in severity or incidences of adverse health effects with large increases in dose), in which cases higher exceedances may be tolerated.

In the end the decision whether or not exceedances are likely to lead to adverse health effects is one of expert judgement based on the weight and strength of the scientific evidence. Notwithstanding, it is good practice to take appropriate steps to reduce levels that exceed health guideline values or criteria.

8.1. Additive and synergistic effects

The approach of summing the HQ to asses the likely cumulative impact of the group of substances in the emissions is consistent with the default approach usually taken to assess the potential cumulative impacts of groups of chemicals.

The approach may be conservative, hence overestimate risks, because it adds the risks of chemicals with different target organs and different mechanisms of action as well as those of substances that do have common effects. The effects of

[145] CSIRO (2005). Meteorological and Dispersion Modelling Using TAPM for Wagerup Phase 3B: HRA (Health Risk Assessment) Concentration Modelling – Expanded Refinery Scenario *Prepared for* Alcoa World Alumina Australia *By* CSIRO Atmospheric Research Private Aspendale, Vic. Report C/0986 11 February 2005. pp

chemicals that affect different organs in different ways may be mutually exclusive - not interdependent - hence not additive.

On the other hand, the approach does not take account of synergistic and potentiating interactions between the chemicals that can lead to an increased risk nor antagonistic interactions that can lead to a reduction in risk. These interactions are more difficult to define and to quantify, hence to consider in risk assessment.

It is extremely difficult, if not impossible, to study experimentally the effects of mixtures beyond simple mixtures of a few chemicals because of the number of possible permutations and combinations that need to be investigated. It is also extremely difficult to predict exposure to the different combinations and concentrations in air (variations with time and three dimensionally in the medium).

There are limited situations in which the effects of groups of chemicals on health can be investigated, for example, in cases where a confined body of water with relatively constant chemical composition is used as a potable water source for a particular community. Similarly, buildings with characteristic chemical contaminant profiles in the air could be investigated, although it would pose more limitations than in the previous example.

There are some data to suggest that chemicals present at concentrations well below the threshold for effects are unlikely to interact synergistically. In addition, in some cases where synergistic interactions occur or are predicted in the whole organism, the resultant increase in effect is not inordinately large and using the additive approach does not appear to underestimate the risks.

For example, ATSDR (2004)[146] has completed a number of interaction profiles assessments, of which the profiles on arsenic, cadmium, chromium and lead and benzene, toluene ethylbenzene and xylenes (BETEX) are of some relevance the Wagerup assessment. The individual chemicals in the two groups of chemicals have some common toxicological effects and target organs.

ATSDR predicts inhibitory, additive and some "more than additive" interactions. However, overall there does not appear to be any consistent interaction that results in synergistic effects that would lead to increases in HQ for any substance far in excess of the HI for the group.

Hence, the most common approach of adding the effects and risks (HI) of the individual chemicals (HQ) is the best approximation to the assessment of cumulative risks of mixtures in practical terms.

[146] ATSDR (2004). Interaction Profiles for Toxic Substances. http://www.atsdr.cdc.gov/iphome.html

APPENDIX B

Carcinogenesis of Formaldehyde

Classified 2A by IARC, formaldehyde is a highly reactive, water-soluble gas that is rapidly absorbed and metabolised at the site of contact. It is also a common product of intermediary metabolism. At high concentrations, it is a genotoxic irritant, producing tissue damage, regenerative hyperplasia, and DNA–protein cross-links at the site of entry (nose). Formaldehyde causes nasal tumours in rats at high exposure concentrations (≥ 6 ppm), with a clearly non-linear dose-response. The dose-response relationships for cell turnover, hyperproliferation, DNA-protein cross-linking, and neoplastic changes are very similar, suggesting that cytotoxicity followed by regenerative proliferation of respiratory epithelium is an obligatory intermediate step (necessary but not sufficient) in carcinogenesis (WHO 2000b, 2002). WHO (2000b) concluded that, "the inhalation of formaldehyde *under conditions that induce cytotoxicity and sustained regenerative proliferation* is considered to present a carcinogenic hazard to humans" (emphasis added). "Thus, if the respiratory tract tissue is not repeatedly damaged, exposure of humans to low, noncytotoxic concentrations of formaldehyde can be assumed to be associated with a negligible cancer risk. This is consistent with epidemiological findings of excess risks of nasopharyngeal and sinonasal cancers associated with concentrations above about 1 mg/m^3" (WHO 2000b).

The U.S. Chemical Industry Institute for Toxicology, USEPA, and Health Canada have developed a biologically motivated case-specific model that integrates dosimetry calculations from computational fluid dynamics modelling of formaldehyde flux in various regions of the nose and single-path modelling for the lower respiratory tracts of animals and humans with a biologically based two-stage clonal growth model of carcinogenesis. This model is summarised in a Concise International Chemical Assessment Document (CICAD) published by WHO (WHO 2002; available online at http://www.who.int/pcs/cicad/full_text/cicad40.pdf). As noted in 66 FR 11165 (http://www.epa.gov/iris/frn02-22-01.htm), the USEPA has a revised and updated assessment underway for formaldehyde that will also apply the biologically motivated model. As indicated in Table 1, estimated human cancer risks calculated using this model are extremely low.

Table 1: Potential Human Cancer Risk at Formaldehyde Concentration, Assuming Lifetime (80-Year) Continuous Exposure

Formaldehyde Exposure Concentration (µg/m^3)	Non-smoking	Mixed	Smoking
1	2.3×10^{-10}	3.9×10^{-9}	4.9×10^{-9}
20	4.8×10^{-9}	1.0×10^{-7}	1.2×10^{-7}
50	1.0×10^{-8}	2.1×10^{-7}	2.5×10^{-7}
70	1.5×10^{-8}	3.3×10^{-7}	3.8×10^{-7}
100	2.1×10^{-8}	4.5×10^{-7}	5.3×10^{-7}

Formaldehyde Exposure Concentration (µg/m^3)	**Non-smoking**	**Mixed**	**Smoking**
120	2.7×10^{-8}	5.8×10^{-7}	6.7×10^{-7}

Because irritation occurs at formaldehyde levels associated with very low cancer risk, irritation is considered the more sensitive and hence more appropriate endpoint for guideline development. WHO (2000b) determined that 100 µg/m^3, "over one order of magnitude lower than a presumed threshold for cytotoxic damage to the nasal mucosa…, represents an exposure level at which there is a negligible risk of upper respiratory tract cancer in humans." However, because this value is higher than the draft 24-hour NEPM of 16.9 µg/m^3, ENVIRON has used the ATSDR chronic MRL of 10.7 µg/m^3 for assessment of chronic health risks associated with Liquor Burner emissions.