SCREENING HEALTH RISK ASSESSMENT OF PARTICULATE EMISSIONS FROM ALCOA'S PINJARRA REFINERY RESIDUE DISPOSAL AREA

for

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ENVIRON

21 August 2008

Ecowise Environmental
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Attention: Neil Evans

Dear Neil,

SCREENING HEALTH RISK ASSESSMENT OF PARTICULATE EMISSIONS FROM ALCOA'S PINJARRA REFINERY RESIDUE DISPOSAL AREA

We are pleased to present our report for the Particulate Emissions Screening Health Risk Assessment for the Pinjarra Refinery Residue Disposal Area incorporating comments received from yourself and Alcoa.

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

Yours faithfully,
ENVIRON Australia Pty Ltd

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Manager WA

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EXECUTIVE SUMMARY

A Screening Health Risk Assessment (SHRA) of the particulate emissions from Alcoa's Pinjarra Refinery Residue Disposal Area (RDA) has been undertaken to investigate the potential health risks arising from these emissions. This SHRA was conducted to complement a preceding Health Risk Assessment (Toxikos, 2003) which investigated particulate emissions from Refinery point sources only (e.g. calciners, oxalate kiln and alumina leach dryer), and was undertaken as part of the approval conditions for Alcoa's *Pinjarra Refinery Efficiency Upgrade Environmental Protection Statement* (ENVIRON, 2003). This SHRA considers the potential health risks associated with particulate emissions from the RDA only, examined for both baseline RDA and upgraded RDA scenarios, defined as follows:

- *Baseline scenario* – previous emissions scenario representative of baseline particulate emissions from Pinjarra Refinery's RDA (prior to the efficiency upgrade); and
- *Upgrade scenario* – an upgraded emissions scenario representative of particulate emissions from Pinjarra Refinery's upgraded RDA, including changes in dust management and a new disposal area constructed to accommodate a 17% increase in alumina production.

The SHRA has generally been confined to the inhalation pathway as this is expected to represent the most significant exposure route to the Pinjarra Refinery's RDA emissions. Therefore, it did not empirically examine alternative exposure pathways (e.g. ingestion of water from local rainwater tanks or food, dermal absorption *etc.*), in any detail. However, 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 and these were considered via use of the Californian Hot Spots Analysis and Reporting Program (HARP) software. This analysis found that exposure pathways other than inhalation were potentially significant for (i) arsenic, cadmium and mercury for chronic non-carcinogenic effects; and (ii) arsenic and lead for carcinogenic effects. A subsequent assessment indicated that the potential for non-inhalation exposure pathways for these metal compounds to cause unacceptable health effects represented no cause for concern.

The following quantitative health risk indicators were calculated for key receptors located in the vicinity of the RDA:

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

ENVIRON was provided with ground level concentrations of PM_{10} predicted from air dispersion modelling conducted by Air Assessments (2007a) for both the baseline and upgraded RDA emissions scenarios. Particulate samples were analysed to assess the total and potentially bioavailable metal contents as part of the particulate monitoring program (Air Assessments 2007b) and these results were used in the SHRA by ENVIRON.

The potential health effects arising from the predicted short-term (acute; 1-hour and 24-hour averages) and long-term (chronic; annual averages) exposure to non-carcinogenic compounds, and potential carcinogenic risks were considered in the SHRA 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 (HIs) 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 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.

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 carcinogenic risk.

If the HI or *de minimis* ICR criterion is exceeded at any receptor, it does not imply that there is a heightened or unacceptable level of risk to health; since due to the conservative nature of the exposure and toxicity assumptions made in performing the SHRA, there are many areas where compounding

conservatism may result in exaggeration of the true likelihood of adverse health outcomes. Rather it would imply that the causes and likelihood of the assumptions leading to the assessed level of risk should be examined for more realistic assessment of the most probable applicable risk level. Thus the conservative screening risk levels adopted in this SHRA are intended to be used as a trigger for more detailed assessment if they are breached, and not until this detailed assessment has occurred might one conclude that the assessed risk level may be unacceptable.

The acute and chronic HIs and the ICRs were calculated for 14 discrete receptor locations identified by Alcoa to represent populations or individual residences that could be potentially exposed to the RDA particulate emissions.

Based upon the results of the health risk screening assessment it can be concluded that at all of the residential receptors considered:

- the potential for emissions from the baseline or upgraded RDA to cause acute health effects is primarily driven by PM_{10} exposure rather than the individual metals in the particulates, but represents no cause for concern;

- the potential for emissions from the baseline or upgraded RDA to cause chronic non-carcinogenic health effects represents no cause for concern; and

- the potential for emissions from the baseline or upgraded RDA to contribute to the incidence of cancer is primarily driven by arsenic exposure, but is below the USEPA *de minimis* threshold of one in a million (i.e. 1×10^{-6}).

Acute exposure to PM_{10} at Receptor 4 was assessed as requiring further assessment based on initial screening utilising maximum ground level concentrations. The predicted acute HI value greater than one at this receptor was primarily associated with the maximum predicted 24-hour average PM_{10} concentration. Consideration of the more realistic, yet still conservative 99.9th percentile (i.e. 9th highest) 1-hour and 99.5th percentile (i.e. 2nd highest) 24-hour average ground level concentrations, results in the Receptor 4 acute HI reducing to below 0.71 for both the baseline and upgraded RDA scenarios. Additionally, the NEPC's (1998) *Ambient Air Quality National Environment Protection Measure* guideline allows up to five exceedances of the target value in a calendar year, and it is therefore concluded that acute exposure to PM_{10} at Receptor 4 does not result in any cause for concern.

As with any risk evaluation, there are areas of uncertainty in this SHRA. To ensure that potential risks are not underestimated, uniformly conservative assumptions have been used to characterise exposure and toxicity (as detailed throughout this Report) and this is considered appropriate for a screening level assessment. 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 Alcoa's Pinjarra Refinery RDA.

Finally, while the RDA is likely to be a major anthropogenic source of particulate emissions to the adjacent area, and inhalation is considered the main pathway of exposure, it is nevertheless recommended that Alcoa continue to consider the potential risk of other sources, as well as indirect exposure pathways, in any future health risk assessments of particulate emissions from the Pinjarra Refinery RDA. Following, the completion of air dispersion modelling for Pinjarra Refinery Efficiency Upgrade, Alcoa will incorporate the results of this SHRA into another SHRA that considers the cumulative impacts of both the Pinjarra Refinery and RDA.

SCREENING HEALTH RISK ASSESSMENT OF PARTICULATE EMISSIONS FROM ALCOA'S PINJARRA REFINERY RESIDUE DISPOSAL AREA

for

Ecowise Environmental Pty Ltd

1. INTRODUCTION

Ecowise Environmental Pty Ltd (Ecowise) has commissioned ENVIRON Australia Pty Ltd (ENVIRON) to conduct a Screening Health Risk Assessment (SHRA) of the potential health risks arising from particulate and constituent metal emissions from Alcoa's Pinjarra Refinery Residue Disposal Area (RDA). A preceding Health Risk Assessment was conducted by Toxikos (2003) as a component of the environmental impact assessment of an efficiency upgrade of the Refinery (i.e. Alcoa's *Pinjarra Refinery Efficiency Upgrade Environmental Protection Statement* [ENVIRON, 2003]); however; the assessment only investigated the potential impacts of particulate emissions from Refinery point sources (e.g. calciners, oxalate kiln and alumina leach dryer) and did not include particulate emissions from the RDA. To address this gap, the present SHRA considers the potential health risks associated with particulate emissions from the RDA only, examined for both baseline RDA and upgraded RDA scenarios, defined as follows:

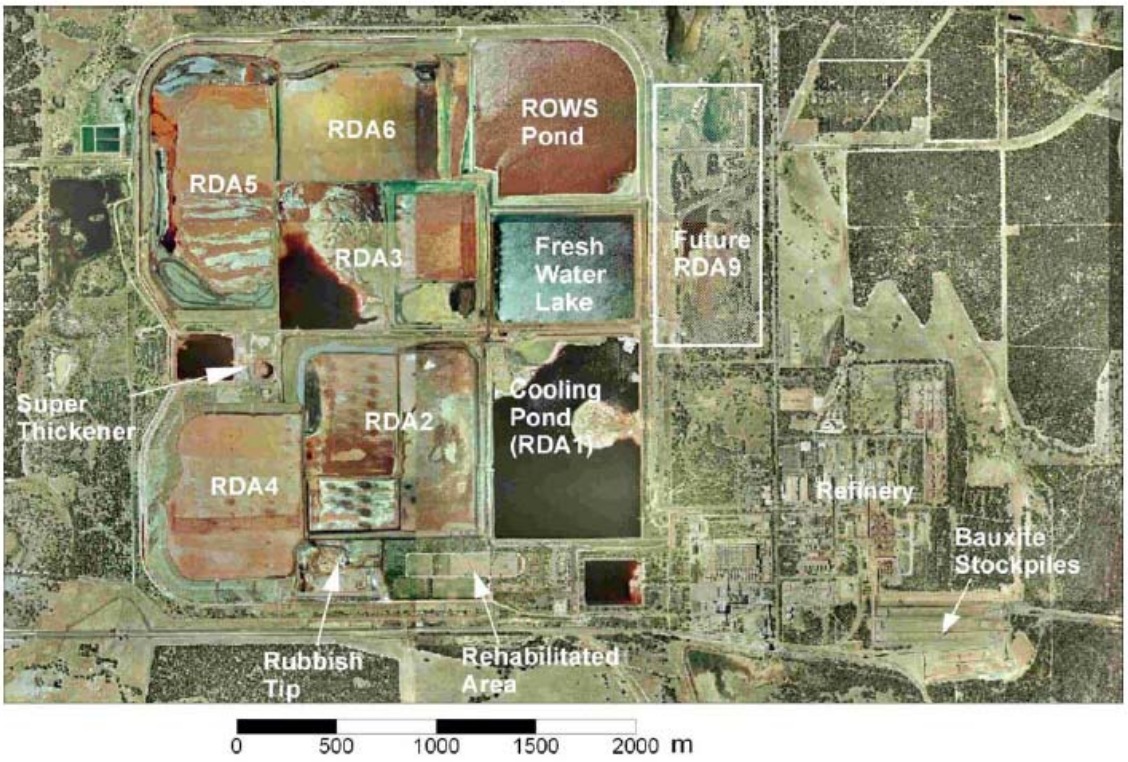
- *Baseline scenario* – previous emissions scenario representative of baseline particulate emissions from Pinjarra Refinery's RDA (prior to the efficiency upgrade); and
- *Upgrade scenario* – an upgraded emissions scenario representative of particulate emissions from Pinjarra Refinery's upgraded RDA, including changes in dust management and a new disposal area (i.e. RDA 9; see Figure 1) constructed to accommodate a 17% increase in alumina production[1].

The air dispersion modelling was completed by Air Assessments (2007a) and the modelling results for 14 nominated receptors were provided to ENVIRON for use in the SHRA. Particulate samples were analysed to assess the total and potentially bioavailable metal contents as part of the particulate monitoring program (Air Assessments 2007b) and these results were incorporated into the SHRA by ENVIRON.

This report outlines the approach used to conduct the SHRA and presents the results of potential acute non-carcinogenic, chronic non-carcinogenic and incremental carcinogenic risks arising from exposure to the RDA particulate emissions and potential metals contained on those emissions at key receptor locations in the vicinity of the Refinery.

[1] For detailed information on the Pinjarra Refinery and RDA upgrade, please refer to ENVIRON (2003) and Air Assessments (2007a).

Figure 1: Pinjarra Refinery RDAs (Air Assessments, 2007a)



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-term (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 SHRA conducted for Pinjarra Refinery's RDA particulate 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 in the particulates emitted from Pinjarra Refinery's RDA are additive. The additive approach is considered to be appropriate for screening assessment purposes, and is generally considered to be conservative (i.e. health protective).

On account of the conservatism of such a screening assessment, the results are considered more likely to over-estimate than under-estimate the potential health risks associated with particulate emissions from the Refinery's RDA. The results of the SHRA are able to be used to assess the relative change to potential health risks associated with the upgraded Pinjarra Refinery RDA, and identify the individual sources and compounds exhibiting the highest contribution to potential health risks in order to help define particulate emissions management strategies.

3. EXPOSURE ASSESSMENT

3.1 Compounds Considered

Alcoa has previously undertaken a review of emission monitoring data available for its Pinjarra, Wagerup and Kwinana refineries and associated RDAs. These studies enabled Alcoa to characterise the atmospheric emissions released from its operations, and to characterise particulate emissions expected to be released from Pinjarra Refinery's upgraded RDA. The previous screening assessment for the Pinjarra Refinery Efficiency upgrade found that 27 individual compounds or compound groups, including particulates and their metal constituents, contributed over 93% of the acute hazard indices (HI), over 86% of the chronic HI, and 100% of the incremental carcinogenic risk (ICR) calculated for the maximally affected receptor (Toxikos, 2003). However that study did not consider the potential impacts associated with particulates from the RDA. This SHRA was therefore

undertaken to quantify the potential risks associated with exposure to the RDA particulate emissions and their associated metal constituent compounds.

The following compounds were selected for the RDA particulate emissions SHRA as they are the only compounds in the list of compounds tested for, that have health risk guidelines defined by reputable sources (i.e. from which acute HIs, chronic HIs or ICRs may be calculated [for further information see Sections 4 and 5]):

- PM_{10};
- Arsenic;
- Selenium;
- Manganese;
- Cadmium;
- Chromium;
- Nickel;
- Mercury;
- Beryllium;
- Lead;
- Molybdenum; and
- Cobalt.

A sensitivity analysis in considering the potential health effects of 'other' metal constituents of particulate dust was also undertaken using the Texas Commission on Environmental Quality's (TCEQ) Effects Screening Levels (ESL) and is presented as Appendix A. The methodological approach of including other metal species has various limitations (discussed in Appendix A) and is thus not included in the main body of this SHRA.

3.2 Potential Receptor Locations

In association with Toxikos (2003), Alcoa identified 14 receptor locations to represent the populations or individual residences that are considered to provide a representative range of potential exposure to atmospheric emissions from the Pinjarra Refinery, as presented in Table 1.

Table 1: Receptor Locations

Receptor No.	Approximate No. of Residences Represented	Description of Use
1	5	Residence, Farmhouse
2	15	Permanent & Short-stay Farm Accommodation
3	500*	Nearest Residence in Carcoola town site
4	2000*	Nearest Residence in Pinjarra town site
5	4	Residence, Farmhouse
6	5	Residence, Farmhouse
7	4	Residence, Farmhouse
8	4	Residence, Farmhouse
9	4	Residence, Farmhouse
10	4	Residence, Farmhouse
11	4	Residence, Farmhouse
12	5	Residence, Farmhouse
13	1-3	Residence, Alcoa Employee & Family
14	4	Residence, Alcoa Farmlands Manager & Family

Note: * - approximate town population.

The locations of the receptors in relation to the Alcoa Refinery site are presented in Figure 2.

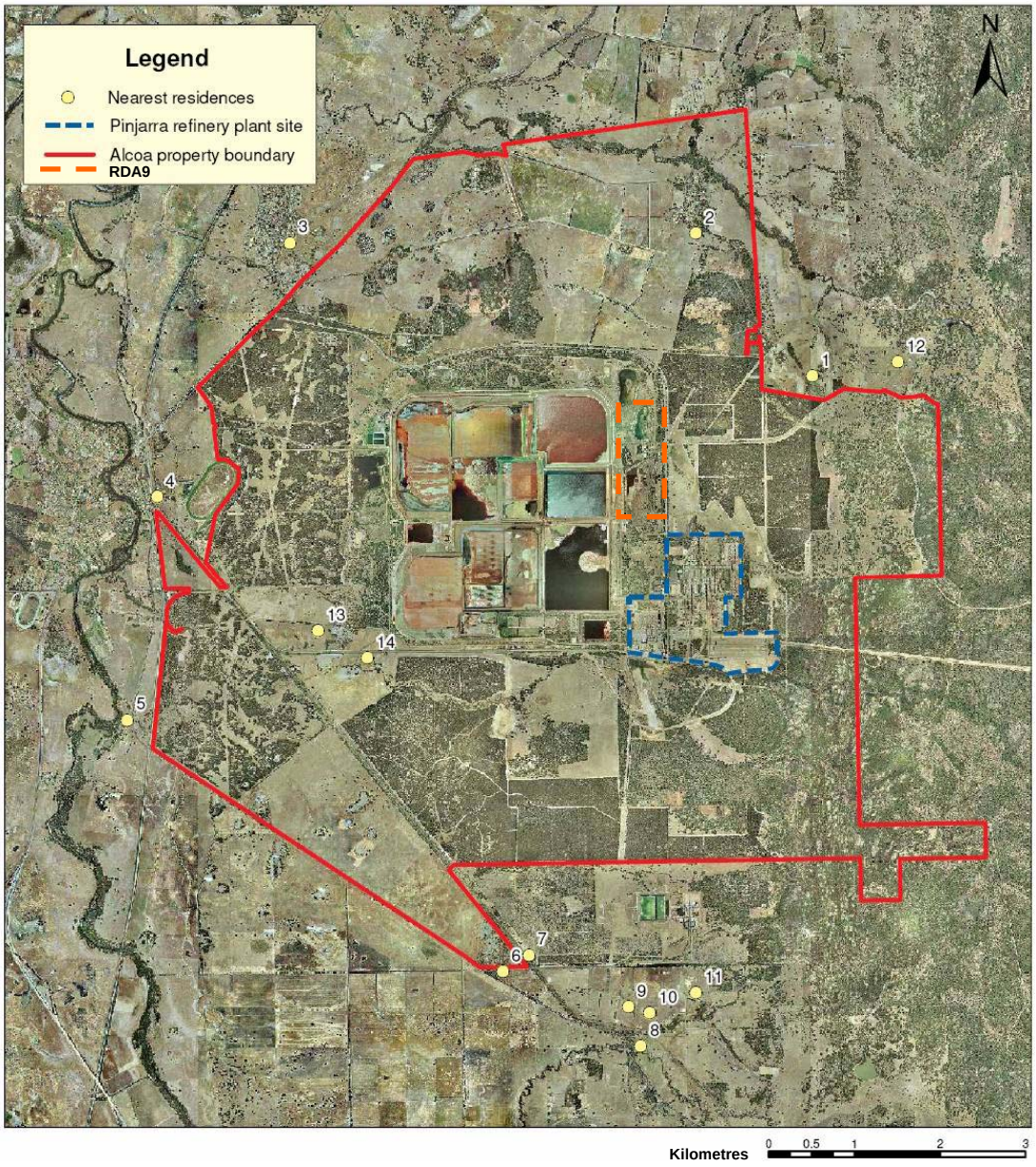
For the purposes of this screening assessment, all receptor sites were assumed to be occupied by residents, including potentially sensitive subpopulations such as children and the elderly. This assumption is inherent in the health protective guidelines selected (refer to Section 4).

3.3 Bioavailability of Particulate Compounds

This SHRA presumes that the concentration of metal compounds present in the RDA particulate emissions is equivalent to that available for human absorption; however, this approach is conservative as not all of the metals are bioavailable. The uptake, distribution and absorption of inhaled metals present in dust particles are primarily a function of particle size, the metal species and solubility. The size of particulate matter is one of the key determinants for identifying the region of the respiratory tract where a particle deposits (United States Environmental Protection Authority [US EPA], 2007). In turn, the site of deposition governs absorption following inhalation exposure. In general, particles 1 µm and smaller reach the alveoli, with larger particles (5 µm and larger) being removed from the nasopharyngeal region by sneezing or blowing the nose, or from the tracheobronchi (1-5 µm) by mucociliary clearance. Once in the lower airways (i.e. bronchiolar and alveolar regions), particles are cleared by phagocytosis, or absorbed into the bloodstream or the lymphatic system (Witschi and Last, 1996). No data indicate that absorption of particulates occurs in the upper airways. From an analysis

of human experimental data, the US EPA (1989) concluded that for inhalation that occurs *via* both the nose and mouth (such as may occur in healthy exercising adults), particles up to approximately 3.5 µm can deposit in alveolar regions, in amounts that can reach approximately 60% of an exposure concentration.

Figure 2: Location of Sensitive Receptors (adapted fromToxikos, 2003)



The US Agency for Toxic Substances and Disease Registry's (ATSDR, 2005a,b) interpreted the US EPA analysis (1989) to be applicable to most respirable particles, including metal particulates, concluding that 30% to 60% of respirable particles are deposited onto the lung surface (i.e. lower airway). Although some portion of the particles may be removed from the lower airway *via* phagocytosis, estimates of the efficiency of this removal mechanism are not available. These data

indicate that in the absence of compound-specific information, it is reasonable to assume that the deposition fraction represents the percentage of particulate ***available*** for absorption. Although availability does not necessarily imply that absorption will occur, or that absorption will be complete, the 30-60% fraction available likely represents a plausible upper bound on the amount that may actually be absorbed from the lower airways into the body. The conservatism of this SHRA due to uncertainty associated with bioavailability of particulate metals is discussed further in Section 5.5.2.

3.4 Potential Exposure Pathways

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 (e.g. such as ingestion *via* food consumptions or drinking water from local rainwater tanks). 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 particulates 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. Metal constituents of particulates emitted from the Pinjarra Refinery RDA that appear in the Air Toxics Hot Spots list of compounds requiring multi-pathway exposure assessment include:

- Arsenic;
- Cadmium;
- Chromium (VI);
- Nickel; and
- Mercury.

A multi-pathway exposure assessment of these metals completed for the initial Pinjarra Refinery Health Risk Assessment found that pathways other than inhalation did not present potentially significant health risks (ENVIRON, 2004). Therefore this SHRA has been confined to the inhalation pathway.

Section 5.5.3 discusses the ENVIRON (2004) assessment and limitations due to uncertainty associated with the potential health risks associated with other pathways of exposure to emissions of particulate compounds from Pinjarra Refinery's RDA.

3.5 Estimated Concentrations in Air

Concentrations or particulates and the associated metals concentrations in the ambient air have been estimated based on the results of air dispersion modelling conducted by Air Assessments (2007a). Air Assessments used the CALMET/CALPUFF dispersion modelling system to predict the ground level concentrations of particulate matter with effective aerodynamic diameter of less than ten microns (PM_{10}) resulting from the RDA emissions. Additional information on modelling methodology, including particulate emission estimates and meteorological inputs, can be found in Air Assessments (2007a).

The metallic composition of PM_{10} has also been reported in Air Assessments (2007b), based on acid digestion of the source dust. In determining the metals composition two types of acid digestion were undertaken:

(i) nitric acid digest – this method provides metal concentrations that may conservatively represent their availability to humans[2].
(ii) 'total' digest – this is an aggressive method utilising four acids to extract 'all' metals from the source particulates. As such, these metal recovery fractions represent total availability to humans (i.e. an unlikely worst case scenario).

Air Assessments (2007a) predicted the ground level concentrations of PM_{10} for each hour over a year and analysed the predicted concentrations to produce the following statistics for PM_{10} for each of the 14 receptors included in the study:

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

The ground level concentrations of each of the nominated metals were then calculated from these predicted PM_{10} concentrations using the maximum metal concentrations (for 1-hr and 24-hr acute exposure) and average metal concentrations (for chronic exposure and ICRs) measured in the

particulate samples via the nitric acid and total digests[3]. These data are provided in Appendix B.

[2] N.B. Conservatism is implied because the nitric digest method utilised may still provide higher metal concentrations than the metal bioavailability to humans (i.e. it over-estimates bioavailability) (Air Assessments, 2007b).

The predicted ground level concentrations of PM_{10} and metals were then used in this SHRA. Since the air dispersion model was "calibrated" against ambient monitoring data, use of the maximum predicted 1-hour and 24-hour concentration statistics was deemed an appropriate first step in screening for potential acute health risks. However, it should be noted that the predicted 99.9th percentile 1-hour average and the 99.5th percentile 24-hour average concentrations have also been considered in this SHRA. These data are often chosen as the key statistics to represent the extremes in the predicted concentrations (CSIRO, 2005), rather than the modelled maximums, due to the tendency of air dispersion models to over predict the maximum concentrations.

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 the 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 defence 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:

[3] Supplementary to the data provided by Air Assessments (2007b), Alcoa provided updated chromium VI concentrations to ENVIRON in May 2008 which have been utilized in this SHRA (*pers. comm.*. P. Coffey, Alcoa 7/05/2008)). The data provided were obtained via total digests performed on a total of 81 samples, of which an average value of 1.6 ppm was obtained. In the absence of nitric digest chromium VI data, the total digest value of 1.6 ppm has been used in the SHRA to conservatively calculate chronic HI and ICR values.

- National Environment Protection (Ambient Air Quality) Measure (NEPC, 1998);
- 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 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 (with preference in that order), 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. 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 SHRA 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 SHRA are presented in Table 2, and are briefly discussed in the following sections.

Table 2: Health Protective Guidelines

Compound Name	Guideline	Units	Averaging Period	Reference
Acute Health Effects				
Particulate matter < 10 µm	50	µg/m^3	24 h	NEPC
Nickel	6	µg/m^3	1 h	OEHHA
Mercury	1.8	µg/m^3	1 h	OEHHA
Copper	100	µg/m^3	1 h	OEHHA
Vanadium	30	µg/m^3	1 h	OEHHA
Chronic Non-Carcinogenic Health Effects				
Arsenic	1	µg/m^3	Annual	RIVM
Selenium	20	µg/m^3	Annual	OEHHA
Manganese	0.15	µg/m^3	Annual	WHO
Cadmium	0.005	µg/m^3	Annual	WHO
Chromium (VI)	0.1	µg/m^3	Annual	IRIS
Nickel	0.09	µg/m^3	Annual	ATSDR
Mercury	1	µg/m^3	Annual	WHO
Copper	1	µg/m^3	Annual	RIVM
Beryllium	0.02	µg/m^3	Annual	IRIS
Lead	0.5	µg/m^3	Annual	NEPC
Molybdenum	12	µg/m^3	Annual	RIVM
Cobalt	0.01	µg/m^3	Annual	ATSDR
Incremental Carcinogenic Risk				
Arsenic	1.50×10^{-3}	per µg/m^3	Annual	WHO
Cadmium	1.80×10^{-3}	per µg/m^3	Annual	IRIS
Chromium (VI)	4.00×10^{-2}	per µg/m^3	Annual	WHO
Nickel	3.80×10^{-4}	per µg/m^3	Annual	WHO
Beryllium	2.40×10^{-3}	per µg/m3	Annual	IRIS
Lead	1.20×10^{-5}	per µg/m3	Annual	OEHHA

Note: 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 health response to a chemical, other than 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 short-term 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 3.

Table 3: 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 Pinjarra Refinery that are classified by the IARC as Group 1, Group 2A or Group 2B are presented in Table 4.

Table 4: IARC Compound Classifications

Compound Name	IARC Classification
Arsenic and compounds	1
Cadmium and compounds	1
Beryllium and compounds	1
Chromium (VI)	1
Nickel compounds	1
Lead and compounds	2A

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.

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 upgraded Pinjarra Refinery RDA 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 5.5.

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 1) and chronic (Equation 2) exposures.

$$HI_{Acute} = \sum^{i} \frac{C_{\leq 24h}}{Gdl_{Acute}} \qquad \textbf{Equation 1}$$

$$HI_{Chronic} = \sum^{i} \frac{C_{Annual}}{Gdl_{Chronic}} \qquad \textbf{Equation 2}$$

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

A 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 3).

$$Risk = \sum_1^i C_{i\ Annual} \times \frac{EF \times ED}{AT} \times UR_i = \sum_1^i C_{i\ Annual} \times UR \qquad \textbf{Equation 3}$$

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.

If the HI or *de minimis* ICR criterion is exceeded at any receptor, it does not imply that there is a heightened or unacceptable level of risk to health; since due to the conservative nature of the exposure and toxicity assumptions made in performing the SHRA, there are many areas where compounding conservatism may result in exaggeration of the true likelihood of adverse health outcomes. Rather it would imply that the causes and likelihood of the assumptions leading to the assessed level of risk

could be examined for more realistic assessment of the most probable applicable risk level. Thus the conservative screening risk levels adopted in this SHRA are intended to be used as a trigger for more detailed assessment if they are breached, and not until this detailed assessment has occurred might one conclude that the assessed risk level may perhaps not be acceptable.

5.2 Acute Non-Carcinogenic Effects

Table 5 presents the calculated acute HIs determined from the nitric acid digest and the very conservative 'total' digest of metals contained in the particulates (see Section 3.5) for each of the receptor locations for the baseline and upgraded Pinjarra Refinery RDA emission scenarios. The percentage contribution that the predicted PM_{10} concentrations make to the overall acute HIs for the existing and upgraded RDA emission scenarios are also presented in Table 5, in addition to the absolute change in HIs associated with the Pinjarra Refinery RDA upgrade scenario compared to the baseline.

Regardless of the digest method (i.e. nitric acid or total) or averaging percentile used to calculate the acute HIs, every receptor, except Receptor 4, is predicted to have an acute HI of less than one (Table 5) for both the baseline and upgrade scenarios.

Firstly based on the maximum 1-hour and maximum 24-hr predicted ground level concentrations as a screening tool, Receptor 4 has an acute HI that is (i) between 3% (nitric digest) and 6% (total digest) above the defined threshold of one for the baseline scenario; and (ii) between 6% and 8% above one for the upgrade scenario (Table 5). It is noted that exposure to PM_{10}, rather than exposure to the constituent metals in the particulates, predominantly contributes (i.e. by between 85.1% and 99.6%) to the acute HI at each receptor (Table 5). Thus, the acute HIs calculated for Receptor 4 are in excess of one primarily as a result of the maximum 24-hour average predicted PM_{10} concentration being in excess of the NEPC's (1998) *Ambient Air Quality National Environment Protection Measure* guideline value of 50 µg/m3; whilst exposure to the particulates constituent metals is only a negligible contributor to the acute HI at Receptor 4 and at all other receptors. It should also be noted that the NEPC's (1998) guideline allows up to five exceedances of the target value in a calendar year.

Further, when the 99.9th percentile (i.e. 9th highest) 1-hour and 99.5th percentile (i.e. 2nd highest) 24-hour average ground level concentrations are considered, Receptor 4 has an acute HI that is below 0.72 for both the baseline and upgraded RDA scenarios for both of the particulate digest methods. The use of these percentiles represent a more realistic, yet still conservative estimate of actual acute exposures (see Section 5.1), and indicates that acute health effects due to particulate exposure at Receptor 4 represent no cause for concern.

Table 5 shows that the Pinjarra Refinery RDA upgrade scenario is predicted to result in both decreases and increases in the acute HIs at receptors depending upon the receptor location, due to nuances in the upgrade configuration of the RDA. Based on the maximum 1-hour and maximum 24-hr predicted ground level concentrations, receptors to the south of the Refinery (Receptors 6 to 11) are predicted to experience slight decreases in the acute HIs; whilst all other receptors are predicted to receive slight increases in acute HIs. Regardless of the direction of change, it should be emphasised that unacceptable acute health effects due to particulate exposure are not expected at any of the receptors for either the baseline or upgraded RDA scenario.

Table 5: Summary of Acute Hazard Indices

Receptor No.	Acute HI derived from 'Nitric' Digest of Particulate Metals					Acute HI derived from 'Total' Digest of Particulate Metals				
	Baseline HI	Upgrade Case HI	Change from Baseline	% Contribution of PM_{10} to HI		Baseline HI	Upgrade Case HI	Change from Baseline	% Contribution of PM_{10} to HI	
				Baseline	Upgrade Case				Baseline	Upgrade Case
Based on the Maximum 1-hour and Maximum 24-hr Predicted Ground Level Concentrations										
1	0.05	0.08	0.028	97.8	98.5	0.05	0.08	0.028	89.3	92.3
2	0.11	0.11	0.008	98.8	98.5	0.11	0.12	0.010	94.0	92.6
3	0.10	0.13	0.026	98.4	98.7	0.11	0.13	0.026	91.8	93.5
4	**1.03**	**1.06**	0.023	99.4	99.4	**1.06**	**1.08**	0.023	96.8	96.9
5	0.22	0.24	0.021	98.9	98.9	0.23	0.25	0.021	94.2	94.2
6	0.09	0.09	-0.001	96.9	96.9	0.10	0.10	-0.002	85.1	85.4
7	0.12	0.12	-0.003	97.3	97.4	0.14	0.13	-0.004	87.1	87.5
8	0.07	0.07	-0.004	97.7	97.7	0.08	0.07	-0.005	88.6	88.9
9	0.08	0.07	-0.006	97.7	97.7	0.09	0.08	-0.006	88.7	88.8
10	0.06	0.06	-0.003	97.7	97.8	0.07	0.07	-0.003	88.5	89.1
11	0.06	0.06	-0.002	98.1	98.2	0.07	0.06	-0.002	90.7	90.9
12	0.03	0.04	0.008	96.9	97.5	0.04	0.04	0.008	85.3	87.8
13	0.87	0.93	0.055	99.0	99.0	0.91	0.97	0.057	94.7	94.8
14	0.44	0.47	0.032	98.5	98.4	0.47	0.51	0.035	92.3	92.1
Based on the 99.9th Percentile 1-Hour and 99.5th Percentile 24-hr Predicted Ground Level Concentrations										
1	0.04	0.07	0.031	99.3	99.4	0.04	0.07	0.032	97.1	97.4
2	0.07	0.10	0.029	99.3	99.4	0.08	0.11	0.030	96.9	97.2
3	0.08	0.10	0.020	99.3	99.4	0.08	0.10	0.020	96.8	97.1
4	0.70	0.68	-0.012	99.6	99.6	0.70	0.69	-0.012	98.2	98.1
5	0.08	0.09	0.005	99.1	99.1	0.08	0.09	0.005	96.1	96.3
6	0.03	0.04	0.012	98.2	98.7	0.03	0.04	0.012	94.2	95.7
7	0.03	0.04	0.014	98.0	98.6	0.03	0.05	0.014	93.8	95.5
8	0.02	0.02	-0.001	98.6	98.6	0.03	0.02	0.000	95.5	95.2
9	0.03	0.03	-0.001	98.6	98.6	0.03	0.03	-0.001	95.5	95.2
10	0.03	0.03	0.001	98.9	98.9	0.03	0.03	0.001	96.3	96.2
11	0.02	0.02	0.002	98.7	98.8	0.02	0.02	0.002	95.4	95.7
12	0.02	0.04	0.014	99.0	99.2	0.02	0.04	0.014	96.4	96.9
13	0.42	0.43	0.002	99.4	99.3	0.43	0.43	0.002	97.3	97.3
14	0.22	0.24	0.013	99.2	99.2	0.23	0.24	0.014	97.1	97.0

Note: Numbers that are in a bold font are greater than 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.

5.3 Chronic Non-Carcinogenic Effects

Table 6 presents the chronic HIs calculated for the baseline and upgraded Pinjarra Refinery RDA emission scenarios using the metals concentrations as determined for both nitric acid and total digests. Data for Chromium VI was only available for the more conservative total digest. As such, this figure was also used within the nitric digest calculations to generate chronic HI values. Utilising the more conservative total digest, a maximum chronic HI of 5.1×10^{-3} is predicted to occur at Receptor 4 based on the Refinery upgrade scenario. Since this maximum is three orders of magnitude less than the threshold of one, it indicates no cause for concern of chronic health risk from exposure to particulates at Receptor 4, or at any other receptor.

Table 6 also indicates that the efficiency upgrade of the Pinjarra Refinery is predicted to result in an increase in the chronic HI at all receptors, but in all cases the absolute change is slight (i.e. three to five orders of magnitude less than the acceptable threshold of one).

As previously mentioned in Section 3.1, a preliminary consideration of the potential for cumulative chronic health effects for other metal constituents of particulates, where a reputable health protective guideline could not be found, is presented as Appendix A.

Table 6: Summary of Chronic Hazard Indices

Receptor No.	Chronic HI derived from 'Nitric' Digest of Particulate Metals			Chronic HI derived from 'Total' Digest of Particulate Metals		
	Baseline HI	Upgrade Case HI	Change from Baseline	Baseline HI	Upgrade Case HI	Change from Baseline
1	5.6×10^{-5}	1.0×10^{-4}	4.4×10^{-5}	3.4×10^{-4}	6.1×10^{-4}	2.6×10^{-4}
2	2.0×10^{-4}	3.2×10^{-4}	1.2×10^{-4}	1.2×10^{-3}	1.9×10^{-3}	7.2×10^{-4}
3	2.8×10^{-4}	3.3×10^{-4}	5.4×10^{-5}	1.7×10^{-3}	2.0×10^{-3}	3.3×10^{-4}
4	8.1×10^{-4}	8.4×10^{-4}	2.4×10^{-5}	4.9×10^{-3}	5.1×10^{-3}	1.5×10^{-4}
5	1.2×10^{-4}	1.3×10^{-4}	9.1×10^{-6}	7.1×10^{-4}	7.7×10^{-4}	5.6×10^{-5}
6	6.6×10^{-5}	9.3×10^{-5}	2.7×10^{-5}	4.0×10^{-4}	5.7×10^{-4}	1.6×10^{-4}
7	6.3×10^{-5}	9.1×10^{-5}	2.7×10^{-5}	3.8×10^{-4}	5.5×10^{-4}	1.7×10^{-4}
8	2.6×10^{-5}	3.1×10^{-5}	5.6×10^{-6}	1.6×10^{-4}	1.9×10^{-4}	3.4×10^{-5}
9	3.0×10^{-5}	3.7×10^{-5}	6.8×10^{-6}	1.8×10^{-4}	2.3×10^{-4}	4.2×10^{-5}
10	2.7×10^{-5}	3.3×10^{-5}	5.8×10^{-6}	1.7×10^{-4}	2.0×10^{-4}	3.5×10^{-5}
11	2.3×10^{-5}	2.7×10^{-5}	4.5×10^{-6}	1.4×10^{-4}	1.7×10^{-4}	2.7×10^{-5}
12	3.7×10^{-5}	5.9×10^{-5}	2.2×10^{-5}	2.2×10^{-4}	3.6×10^{-4}	1.3×10^{-4}
13	5.6×10^{-4}	5.8×10^{-4}	2.2×10^{-5}	3.4×10^{-3}	3.5×10^{-3}	1.3×10^{-4}
14	5.5×10^{-4}	5.7×10^{-4}	2.2×10^{-5}	3.3×10^{-3}	3.5×10^{-3}	1.3×10^{-4}

5.4 Carcinogenic Effects

The incremental carcinogenic risk (ICR) has been calculated for the baseline and upgraded Pinjarra Refinery RDA emission scenarios, as determined for both nitric and 'total' acid digests of particulate metals and the results are presented in Table 7. As noted previously, data for Chromium VI were only available for the more conservative total digest and as such, this has been used within the nitric digest calculations to generate ICR values. Utilising the more conservative 'total' digest as a screening tool, a maximum ICR of 1.8×10^{-7} is predicted to occur at Receptor 4 for the RDA upgrade scenarios. Since this maximum is well below the USEPA's *de minimis* criteria (i.e. 1.0×10^{-6}), it indicates no cause for concern of carcinogenic risk from exposure to particulates at Receptor 4, or at any other receptor.

Utilising the more realistic nitric acid digest (see Section 3.5), a maximum ICR of 3.3×10^{-8} is predicted to occur at Receptor 4, under the Refinery's RDA upgrade scenario. Since this maximum is much less than the USEPA's *de minimis* threshold, it indicates negligible carcinogenic health risk from exposure to particulates at Receptor 4 and at all other receptors.

An increase in the incremental carcinogenic risk compared to the baseline incremental carcinogenic risk is predicted to result from the Pinjarra Refinery RDA upgrade at all receptor locations (Table 7). However, the magnitude of increase at any of the receptors is only slight and the overall incremental carcinogenic risk remains well below the USEPA's *de minimis* level of 1×10^{-6} (Table 7)).

Table 7: Summary of Incremental Carcinogenic Risk

Receptor No.	ICR derived from 'Nitric' Digest of Particulate Metals			ICR derived from 'Total' Digest of Particulate Metals		
	Baseline HI	Upgrade Case HI	Change from Baseline	Baseline HI	Upgrade Case HI	Change from Baseline
1	2.2×10^{-9}	3.9×10^{-9}	1.7×10^{-9}	1.2×10^{-8}	2.1×10^{-8}	9.4×10^{-9}
2	7.7×10^{-9}	1.2×10^{-8}	4.7×10^{-9}	4.2×10^{-8}	6.8×10^{-8}	2.5×10^{-8}
3	1.1×10^{-8}	1.3×10^{-8}	2.1×10^{-9}	5.9×10^{-8}	7.1×10^{-8}	1.2×10^{-8}
4	3.2×10^{-8}	3.3×10^{-8}	9.5×10^{-10}	1.7×10^{-7}	1.8×10^{-7}	5.2×10^{-9}
5	4.6×10^{-9}	5.0×10^{-9}	3.6×10^{-10}	2.5×10^{-8}	2.7×10^{-8}	2.0×10^{-9}
6	2.6×10^{-9}	3.7×10^{-9}	1.1×10^{-9}	1.4×10^{-8}	2.0×10^{-8}	5.8×10^{-9}
7	2.5×10^{-9}	3.6×10^{-9}	1.1×10^{-9}	1.4×10^{-8}	1.9×10^{-8}	5.9×10^{-9}
8	1.0×10^{-9}	1.2×10^{-9}	2.2×10^{-10}	5.6×10^{-9}	6.8×10^{-9}	1.2×10^{-9}
9	1.2×10^{-9}	1.5×10^{-9}	2.7×10^{-10}	6.5×10^{-9}	8.0×10^{-9}	1.5×10^{-9}
10	1.1×10^{-9}	1.3×10^{-9}	2.3×10^{-10}	5.9×10^{-9}	7.1×10^{-9}	1.3×10^{-9}
11	9.0×10^{-10}	1.1×10^{-9}	1.8×10^{-10}	4.9×10^{-9}	5.9×10^{-9}	9.5×10^{-10}
12	1.4×10^{-9}	2.3×10^{-9}	8.6×10^{-10}	7.9×10^{-9}	1.3×10^{-8}	4.7×10^{-9}

Receptor No.	ICR derived from 'Nitric' Digest of Particulate Metals			ICR derived from 'Total' Digest of Particulate Metals		
	Baseline HI	Upgrade Case HI	Change from Baseline	Baseline HI	Upgrade Case HI	Change from Baseline
13	2.2×10^{-8}	2.3×10^{-8}	8.6×10^{-10}	1.2×10^{-7}	1.2×10^{-7}	4.7×10^{-9}
14	2.2×10^{-8}	2.2×10^{-8}	8.7×10^{-10}	1.2×10^{-7}	1.2×10^{-7}	4.7×10^{-9}

Note: Numbers that are in a bold font are greater than 1×10^{-6}.

5.5 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).

For this SHRA, 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.

5.5.1 Emissions Characterisation and Quantification Uncertainty

There is uncertainty associated with the identification and quantification of particulate metal emissions from the Pinjarra Refinery's RDA.

Although not incorporating emissions from the RDA, the previous HRA (Toxikos, 2003) included 27 individual or groups of compounds, including particulates and six metal constituents (i.e. Arsenic, Selenium, Manganese, Cadmium, Nickel and Mercury). Toxikos (2003) estimated that these 27 individual compounds or groups of compounds were found to contribute over 93% of the acute HI, over 86% of the chronic HI, and 100% of the incremental carcinogenic risk calculated at the maximally affected receptor (Receptor 1). Based on these findings, the nine metal constituent compounds considered in this particulates screening assessment are expected to contribute the vast majority of the potential health risks associated with residue dust emissions.

5.5.2 Bioavailability Assumptions Uncertainty

As noted in Section 3.3, the ambient air concentration or inhaled dose of a particulate metal does not necessarily equate to the fraction of absorption that will occur for that particular metal. The uptake, distribution and absorption of inhaled metals present as particles in dust will be a function of particle size, the metal species and solubility. In this brief review of the likely bioavailability of six metal species[4] for which information is readily available, inhaled dose refers to the total particulate concentration in ambient air. The alveolar deposition fraction refers to the percentage of an inhaled dose that is available for absorption.

For arsenic, data from occupational studies have shown that 30% to 60% of an inhaled dose of arsenic particulate is excreted in urine, the principal route of elimination. Since the deposition fraction is also 30% to 60%, this indicates that while virtually all of the deposited arsenic is absorbed, the remaining portion of an inhaled dose is not biologically available. This is consistent with the findings of the US EPA (1989), and indicates that a significant portion of inhaled arsenic particulate may not reach the lower airways.

From a comprehensive review of available data, the ATSDR (2005b) concluded that subsequent to inhalation exposure, approximately 20% to 30% of the retained nickel particulate is absorbed. Because only a fraction of inhaled nickel particulate is deposited to the lower airways, where it is subject to retention, (US EPA, 1989), this statement suggests that when expressed as a percentage of inhaled dose, the amount absorbed is markedly lower than the fraction cited by the ATSDR. However, given uncertainties with respect to the nickel species and solubility, use of the ATSDR data likely represents a health-conservative estimate of the bioavailability of inhaled nickel particulate.

There are no data from human studies that have characterized airway deposition, retention, or net absorption of cadmium following inhalation exposure to cadmium particulate. ATSDR's review of animal data (ATSDR, 1999a) show that retention of cadmium ranges from 5% to 20% following exposures of 15 minutes to 2 hours, and decreases with increasing exposure duration. A physiologically-based pharmacokinetic (PBPK) model of inhaled cadmium (Nordberg *et al.*, 1985 as cited in ATSDR, 1999a) indicates that between 50% and 100% of inhaled cadmium deposited (retained) in the alveoli will be absorbed. Integrating the PBPK analysis with that of the US EPA (1989), suggests that 15% to 60% of inhaled particulate cadmium is available for absorption.

[4] These six metals were also the initial candidates targeted by Alcoa due their potential health effects and known likely constituency in Pinjarra RDA dust. However, subsequent analyses comprehensively determined the particulate constituency of other metal species which were later included in the health risk analysis.

The absorption of selenium following inhalation exposure is the least well documented of the six metals in question. Selenium is a somewhat unique metal in the context of human toxicity, in that it exhibits the lowest margin between human deficiency (it is an essential element) and excess. There are no direct or quantitative human data on the extent or rate of absorption of inhaled selenium particulate. Qualitative human data establish that airborne selenium particulate is absorbed by inhalation, and that the quantity eliminated in urine increases with increasing exposure concentration (ATSDR, 2003). Similarly, there are no quantitative or specific data on the absorption of manganese particulate by humans exposed by inhalation (ATSDR, 2000). Experimental animal data have confirmed that particle size is one of the most significant variables that affect manganese uptake, deposition, and retention, with smaller particles (1.3 μm) resulting in higher lung burdens than large (18 μm) particles (Fetcher *et al*. 2002). In the absence of specific data on selenium and manganese, the general conclusions of the US EPA (1989) can be used to support an estimate that 30% to 60% of inhaled selenium or manganese may be available for absorption.

Mercury represents a unique case, in that elemental (i.e. metallic) mercury volatilizes at standard temperature and pressure. Mercury vapor partitions readily across membranes, and is rapidly and extensively absorbed from the alveoli into the circulatory system (ATSDR, 1999b). Analyses of blood, plasma, and urine in humans exposed by inhalation provide an estimate of absorption that ranges between 69% and 80% (ATSDR, 1999b; Hursch *et al*., 1976; Sandborgh-Englund *et al*., 1998).

The range of realistic inhalation absorption values for arsenic, nickel, cadmium, selenium, manganese and mercury are summarised in Table 8. By assuming that that the ambient air concentration (deposition fraction) of these and other constituent metals are all available for absorption, this SHRA has adopted a conservative approach likely to be considerably overestimating their bioavailability.

Table 8: Absorption of Metals after Inhalation Exposure.

Metal	Absorption (expressed as a percentage of total particulate concentration in ambient air)	Primary Sources
Arsenic	30% to 60 %	ATSDR (2005a); US EPA (1989)
Nickel	25% to 35 %	ATSDR, 2005b; US EPA (1989)
Cadmium	15% to 60 %	ATSDR (199a); Nordberg *et al*. (1985); US EPA(1989)
Selenium	30% to 60 %	ATSDR (2003); US EPA (1989)
Manganese	30% to 60 %	ATSDR (2000); US EPA (1989)
Mercury	69% to 80 %	ATSDR (1999b); Hursch *et al*.(1976); Sandborgh-Englund *et al*. (1998).

5.5.3 Exposure Assumptions Uncertainty

To calculate the incremental carcinogenic risk it has been assumed that residents 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's RDA emissions 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 SHRA 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. Particulate compounds are 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. Furthermore, there is potential for accumulation of particulate metals in water bodies and local rainwater tanks.

Particulate metal compounds considered in this SHRA that are likely to require multi-pathway exposure assessment (refer to Section 3.4) include:

- Arsenic;
- Cadmium;
- Chromium (VI);
- Nickel; and
- Mercury.

To assist with the assessment of multi-pathway exposure assessments, the Hot Spots Analysis and Reporting Program (HARP) software has been developed in consultation with various Californian environmental agencies. The HARP was applied by ENVIRON (2004) for a multi-pathway exposure assessment; however, the analysis was 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) found that exposure pathways other than inhalation were potentially significant for (i) arsenic, cadmium and mercury for chronic non-carcinogenic effects; and (ii) arsenic and lead for carcinogenic effects. For these compounds, alternate pathways of exposure need consideration in calculation of the overall HI or ICR (i.e. including the contribution to health risk from the alternate exposure pathways listed above).

As detailed in Section 5.1, HI and ICR values are calculated based on simultaneous exposure to multiple compounds by summing the health risk posed by individual compounds. For an individual compound, the estimated long-term average concentration in air expressed as: (i) a ratio of the relevant chronic risk health protective guideline is termed the Hazard Quotient (HQ); and (ii) a multiplication of the relevant carcinogenic unit risk factor guideline is termed the Carcinogenic Risk (CR)[5]. For a given compound, if the proportion of total health risk attributable to the inhalation pathway is known (e.g. as defined by HARP analysis), then HQ and CR values for the inhalation pathway may be extrapolated to be representative of the overall health risk (i.e. including both inhalation and non-inhalation exposure pathways). These overall HQs or CRs, for those compounds requiring multi-pathway analysis, may then be summed with the HQs or CRs for compounds where

[5] Technically, the CR for individual compounds may be defined as an incremental carcinogenic risk (i.e. an ICR value), which are summed to calculate the potential Total ICR from exposure to multiple compounds (i.e. the ICR as defined in his HRA); however, for the purposes of this HRA the incremental carcinogenic risk posed by an individual compound has been abbreviated to 'CR'.

only the inhalation pathway is important, to represent an overall HI or ICR value, that is inclusive of alternate exposure pathways.

For compounds where alternate pathways of exposure has been found significant, Table 9 gives the approximate percentage contribution of the estimated potential health risk arising from inhalation and non-inhalation exposure pathways, as defined by ENVIRON (2004). Table 9 further provides the chronic HQ and CR values for the inhalation pathway for the maximally exposed receptor (i.e. Receptor 4, see Table for explanation), and the extrapolation of these values to represent overall chronic HQ and CR values that are inclusive of non-inhalation pathways. Finally, Table 9 provides the overall chronic HI (1.2×10^{-2}) and ICR (8.8×10^{-7}) values at the maximally exposed receptor.

Since both of these values are below the acceptable guideline threshold, it can be concluded that at all of the residential receptors considered, even when including non-inhalation exposure pathways, the potential for emissions from the baseline or upgraded RDA to:

(i) cause chronic non-carcinogenic health effects represents no cause for concern; and
(ii) contribute to the incidence of cancer is below the USEPA *de minimis* threshold.

Table 9: Potential Chronic, Non-Carcinogenic Health Risks [A] and Carcinogenic Health Risks [B] Arising from Multi-Exposure Pathways at the Maximally* Exposed Receptor.

[A]

Metal Compound	% Contribution to Chronic, Non-Carcinogenic Health Risk by Exposure Pathway (ENVIRON, 2004)		Inhalation Pathway Maximum[a] Hazard Quotient (This Study)	Overall Maximum[a] Hazard Quotient (Inhalation plus Non-Inhalation Pathways)	Overall Maximum[a] Hazard Index
	Inhalation	Non-Inhalation			
Arsenic	~50%	~50%	1.1×10^{-4}	2.2×10^{-4}	1.2×10^{-2}
Cadmium	~55%	~45%	5.8×10^{-5}	1.1×10^{-4}	
Mercury	~10%	~90%	1.6×10^{-7}	1.6×10^{-6}	

[B]

Metal Compound	% Contribution to Carcinogenic Health Risk by Exposure Pathway (ENVIRON, 2004)		Inhalation Pathway Maximum[b] Carcinogenic Risk (This Study)	Overall Maximum[b] Carcinogenic Risk (Inhalation plus Non-Inhalation Pathways)	Overall Maximum[b] ICR
	Inhalation	Non-Inhalation			
Arsenic	~20%	~80%	2.2×10^{-8}	1.1×10^{-7}	8.8×10^{-7}
Lead	~15%	~85%	5.8×10^{-10}	3.86×10^{-9}	

* - Maximum exposure to particulate compounds is estimated to occur at Receptor 4 under the Refinery Upgrade Scenario. Results are based on: (a) 'total' digest of particulate metals for chronic health risk indices (see Section 5.3); and (b) nitric digest of particulate metals for carcinogenic health risk indices (see Section 5.4).

5.5.4 Toxicity Assessment Uncertainty

A further uncertainty associated with the SHRA is related to the derivation of the health protective guidelines. Health protective guidelines published by reputable authorities have been applied within this assessment and 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 considerable overestimation of risk.

5.5.5 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 may 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 generally considered to be conservative (i.e. health protective).

6. SUMMARY

ENVIRON has conducted a screening SHRA of the potential health risks associated with particulate emissions from Alcoa's Pinjarra Refinery Residue Disposal Area, considering the potential risks associated with a baseline and upgraded RDA emissions scenarios.

Quantitative health risk indicators were calculated for exposure via the inhalation pathway, to particulate emissions from the RDA, but empirical examination of alternative exposure pathways (e.g. drinking water from local rainwater tanks, ingestion via food, dermal absorption etc.) was not undertaken, nor was consideration given to other sources of emissions of particulate compounds (such as Refinery point source/stack emissions). However, based on preliminary multi-pathway exposure assessment (ENVIRON, 2004), it was found that exposure pathways other than inhalation were potentially significant for: (i) arsenic, cadmium and mercury for chronic non-carcinogenic effects; and (ii) arsenic and lead for carcinogenic effects. A subsequent assessment indicated that the potential for non-inhalation exposure pathways for these metal compounds to cause unacceptable health effects represented no cause for concern.

The following quantitative health risk indicators were calculated for key receptors located in the vicinity of the RDA:

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

Based upon the results of the health screening assessment it can be concluded that at all of the residential receptors considered:

- the potential for emissions from the baseline or upgraded RDA to cause acute health effects is primarily driven by PM_{10} exposure rather than the individual metals in the particulates, but represents no cause for concern;

- the potential for emissions from the baseline or upgraded RDA to cause chronic non-carcinogenic health effects represents no cause for concern; and

- the potential for emissions from the baseline or upgraded RDA to contribute to the incidence of cancer is primarily driven by arsenic exposure, but is below the USEPA *de minimis* threshold of one in a million (i.e. 1×10^{-6}).

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 characterise 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 Alcoa's Pinjarra Refinery RDA.

Finally, while the RDA is likely to be a major anthropogenic source of particulate emissions to the adjacent area, and inhalation is considered the main pathway of exposure, it is nevertheless recommended that Alcoa continue to consider the potential risk of other sources, as well as indirect exposure pathways, in any future health risk assessments of particulate emissions from the Pinjarra Refinery RDA.

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APPENDIX A

Potential Chronic Health Effects Inclusive of Exposure to Other Particulate Metals

A.1 Potential Chronic Non-Carcinogenic Effects Inclusive of Other Metals

For the main body of this SHRA, selection of the metal constituents in particulate dust with the greatest potential known to cause chronic health effects (and contributing to the calculated chronic HIs; i.e. arsenic, selenium, manganese, cadmium, chromium, nickel, mercury, beryllium and lead) was determined by including all metal species with chronic health protective guidelines published by reputable authorities, as outlined in Section 4.

The Texas Commission on Environmental Quality (TCEQ) is one jurisdiction that has derived chronic health protective guidelines for a wide range of compounds including many metals other than those listed above (see Table A.1). However, the TCEQs effect screening levels (ESLs) are extremely conservative and set at levels much lower than levels reported to produce adverse health effects. If the air concentration for any given compound is above the TCEQ ESL, it does not indicate that an adverse effect will occur, but rather that further evaluation is warranted. ESL values published by the TCEQ are typically not included in the calculation of chronic HI values, as they can markedly overestimate the 'true' HI values.

Nevertheless, as a preliminary (albeit highly conservative) exercise in considering the potential cumulative health effects of inclusion of additional metal constituents present in the particulates, this Appendix discusses inclusion and application of the TCEQ chronic ESLs in the calculation of chronic HIs at each receptor. Concentration estimates of these additional metal constituents at each receptor were determined using the same methodology as outlined in Section 3.5 (i.e. nitric and 'total' acid digests of particulate dust).

The chronic HIs have been calculated for the baseline and upgraded Pinjarra Refinery emission scenarios for both the nitric and 'total' acid digests, including these additional metal constituents of particulates. Table A.2 presents the calculated HIs. Utilising the more conservative total digest, a maximum chronic HI of 0.837 is predicted to occur at Receptor 4 based on the Refinery upgrade scenario. Since this maximum is below the acceptable threshold of one, and given the high conservatism of the TCEQ ESLs, it indicates no cause for concern of chronic health risk from exposure to particulates at Receptor 4, nor at any other receptor.

Table A.1: Chronic Non-Carcinogenic Health Protective Guidelines, including those derived by the TCEQ

Compound Name	Guideline	Units	Averaging Period	Reference
Arsenic	0.03	µg/m^3	Annual	OEHHA
Selenium	20	µg/m^3	Annual	OEHHA
Manganese	0.15	µg/m^3	Annual	WHO
Cadmium	0.005	µg/m^3	Annual	WHO
Chromium (VI)	0.1	µg/m^3	Annual	IRIS
Nickel	0.05	µg/m^3	Annual	OEHHA
Mercury	1	µg/m^3	Annual	WHO
Copper	1	µg/m^3	Annual	RIVM
Beryllium	0.02	µg/m^3	Annual	IRIS
Lead	0.5	µg/m^3	Annual	NEPC
Molybdenum	12	µg/m^3	Annual	RIVM
Cobalt	0.01	µg/m^3	Annual	ATSDR
Vanadium	0.05	µg/m^3	Annual	TCEQ
Uranium	0.05	µg/m^3	Annual	TCEQ
Aluminium	5	µg/m^3	Annual	TCEQ
Antimony	0.5	µg/m^3	Annual	TCEQ
Calcium	5	µg/m^3	Annual	TCEQ
Iron	1	µg/m^3	Annual	TCEQ
Lithium	1	µg/m^3	Annual	TCEQ
Magnesium	10	µg/m^3	Annual	TCEQ
Potassium	2	µg/m^3	Annual	TCEQ
Silicon	5	µg/m^3	Annual	TCEQ
Silver	0.01	µg/m^3	Annual	TCEQ
Sodium	2	µg/m^3	Annual	TCEQ
Strontium	2	µg/m^3	Annual	TCEQ
Thallium	0.1	µg/m^3	Annual	TCEQ
Zinc	5	µg/m^3	Annual	TCEQ

Table A.2: Summary of Chronic Hazard Indices Inclusive of 'Other' Metals

Receptor No.	Chronic HI derived from 'Nitric' Digest of Particulate Metals			Chronic HI derived from 'Total' Digest of Particulate Metals		
	Baseline HI	Upgrade Case HI	Change from Baseline (%)	Baseline HI	Upgrade Case HI	Change from Baseline (%)
1	0.019	0.034	77.8	0.056	0.100	77.8
2	0.068	0.108	60.4	0.196	0.315	60.4
3	0.095	0.113	19.6	0.275	0.329	19.6
4	0.280	0.288	3.0	0.813	0.837	3.0
5	0.040	0.043	7.8	0.117	0.126	7.8
6	0.023	0.032	41.2	0.066	0.093	41.2
7	0.022	0.031	43.5	0.063	0.091	43.5
8	0.009	0.011	21.7	0.026	0.031	21.7
9	0.010	0.013	22.5	0.030	0.037	22.5
10	0.009	0.011	21.3	0.027	0.033	21.3
11	0.008	0.009	19.5	0.023	0.027	19.5
12	0.013	0.020	59.7	0.037	0.058	59.7
13	0.193	0.201	3.9	0.560	0.582	3.9
14	0.188	0.196	4.0	0.547	0.569	4.0

APPENDIX B

Modelled PM_{10} Ground Level Concentrations and Metal Composition Raw Data

Table B.1: Modelled PM_{10} Ground Level Concentrations in µg/m^3 (Air Assessments, 2007a)

Av. Period / Receptor	Maximum 1-hr		99.9th Percentile 1-hr		Maximum 24-hr		99.5th Percentile 24-hr		Annual	
	Base	Upgrade	Base	Upgrade	Base	Upgrade	Base	Upgrade	Base	Upgrade
1	54.1	59.8	8.51	15.7	2.34	3.73	1.87	3.43	0.11	0.19
2	64.6	87.2	20.6	26.0	5.25	5.62	3.73	5.18	0.38	0.61
3	85.4	83.6	22.0	25.	4.96	6.25	3.93	4.91	0.53	0.63
4	324	328	114	117	51.3	52.5	34.6	34.0	1.57	1.61
5	130	140	26.6	26.2	10.8	11.9	4.05	4.29	0.23	0.24
6	148	143	11.4	11.9	4.37	4.31	1.51	2.13	0.13	0.18
7	169	159	11.5	12.9	5.89	5.75	1.50	2.21	0.12	0.17
8	87.0	80.2	7.23	8.00	3.51	3.32	1.22	1.19	0.05	0.06
9	96.3	89.0	8.26	8.97	3.92	3.66	1.37	1.33	0.06	0.07
10	78.9	71.2	6.54	7.61	3.15	3.02	1.35	1.39	0.05	0.06
11	60.3	57.2	6.56	6.92	3.06	2.95	0.99	1.07	0.04	0.05
12	52.9	52.9	5.62	9.18	1.58	1.97	1.11	1.81	0.07	0.11
13	464	486	93.8	93.0	43.2	46.0	21.1	21.2	1.08	1.12
14	349	389	50.4	53.2	21.8	23.4	11.2	11.8	1.05	1.10

Table B.2: PM_{10} Metal Composition Data in Parts Per Million (Air Assessments, 2007b)

Metal (Total)	"Total" Particulate Digest		Nitric Acid Particulate Digest	
	Maximum	Average	Maximum	Average
Total Arsenic	113	66	12.9	9
Total Selenium	14	14	5.8	4
Tot. Manganese	629	369	17	14
Total Cadmium	0.23	0.18	0.21	0.21
Total Nickel	17	16	5.6	4
Total Mercury	0.22	0.1	0.07	0.07
Tot.Beryllium	1.3	0.7	0.2	0.2
Total Chromium	477	394	257	181
Total Copper	510	260	256	180
Total Lead	53	41	36	30
Total Vanadium	2860	1,741	469	469
Tot. Gallium	171	150	98	82
Tot. Molybdenum	189	74	31	17
Tot. Uranium	43	26	33	24
Tot.Aluminium	237,000	192,000	83,500	64,000
Total Antimony	1.4	0.9		1.2
Total Calcium	60,000	21,230	20,800	14,600
Total Cobalt	0.51	0.2	0.9	0.63
Total Iron	459,000	368,000	167,000	111,000
Total Lithium	14	14	6	6
Total Magnesium	17,400	4,818	1,926	1,163
Total Potassium	22,600	11,835	195	179
Total Silicon	165,000	103,000	5,700	4,400
Total Silver	0.44	0.44	2	2
Total Sodium	167,000	77,100	144,000	77,000
Total Strontium	296	174	199	121
Total Thallium	0.29	0.18	0.29	0.29
Total Thorium	1280	796	1,027	616
Total Zinc	116	90	125	81