Toxikos
Toxicology Consultants

ABN 30 095 051 791
PO Box 74
Caulfield East
Victoria, 3145
Ph: +61 3 9569 3088
Fax: +61 3 9569 3618
rogerdrew@bigpond.com

Preliminary health risk assessment of air emissions from proposed Biomass Power Station, Manjimup

Prepared by: Roger Drew, PhD, DABT
Toxikos Pty Ltd.,
PO Box 74 East Caulfield,
Vic, 3145

Prepared for: Neil Mackenzie,
Senior Associate,
Connell Wagner Pty Ltd.,
Adelaide

Toxikos Document: TR17208-RF
11th April 2008

……

Roger Drew PhD, DABT
(Diplomate American Board of Toxicology)

Executive Summary

A biomass power station (BSP) is intended to be built at Manjimup in SW Western Australia. The power station will generate electricity by burning plantation waste. The site is immediately surrounded by State Forest and beyond by farmland. Within 2.5 km of the proposed site there are only three dwellings.

Connell Wagner has requested Toxikos Pty Ltd to conduct a screening human health risk assessment (HRA) for predicted emissions from the power station. They supplied Toxikos with ground level concentrations (GLC) for some of the substances that will be in the emissions for two scenarios. The first being emissions from the BSP only and the second a cumulative scenario of BSP plus background. The GLCs were simulated with the air dispersion model TAPM. For the majority of substances maximum GLCs were provided, however since a 'back of envelope' risk assessment indicated the majority of the risks were associated with current background exposures, in the cumulative scenario the 98th percentile GLC for both background and BSP emissions of particulate matter (PM), NO_2 and SO_2 were also supplied. Background particulate data was obtained from CSIRO measurements for Manjimup and is considered to be relevant for the assessment at hand.

The HRA has been conducted according to national and international guidelines and included an assessment of acute and chronic health risks that might arise from direct inhalation exposure to:

- individual components of the emissions.
- the mixture of chemicals in the emissions.

Also incorporated into the HRA is

- a quantitative evaluation of cancer risks, and
- a preliminary evaluation of the potential health risks associated with exposure of emission components through the food chain (i.e. indirect exposure pathways).

The HRA takes cognisance of background exposures when these were provided. The following conclusions are influenced by uncertainty with background exposure information with regards to whether there is concordance of substance exposure of both maximum GLC due to BSP and that from background. In these respects the HRA is conservative. It was concluded direct health risks from exposure to the emissions from the power station is unlikely, however there is

uncertainty associated with the conclusion because the accuracy of the estimates, and/or by how much actual emissions may have been overestimated, is unknown.

In the cumulative assessment of BSP emissions plus other sources of emission components, the acute health risks are dominated by background airborne particulates, approximately 90% of the risk is due to background particulate (PM). Although the calculated hazard indices (an indication of health impact) are greater than unity when the maximum estimated ground level concentrations are used in the calculations, it does not imply heath effects will occur. This is primarily because the circumstances giving rise to these values occur very infrequently and it is presumed unlikely a person will be at the location, at the same time the concordance of maximum values occur. Using 98th percentile GLCs for particulates in BSP emissions and for local background concentrations results in a marked decrease in the risk calculations, such that they are at or about the target for declaring it would be very unlikely health effects would occur.

Calculated cancer risks (~0.3 x 10^{-5}) are within the band of acceptability used by many Australian and overseas jurisdictions, which in conjunction with the small population that may potentially be exposed, indicates it is unlikely a person will develop cancer as a result of exposures to the emissions. The uncertainty associated with this conclusion is again the accuracy of the emission estimations and the extent they may have been over estimated, but also absence of detailed knowledge of the size of the population potentially at risk.

Dioxins, PAH and the metals were assessed for potential health risks associated with exposure of emission components through the food chain using screening techniques developed by Toxikos. These techniques are conservative, i.e. err on the side of safety, and are applied to initially determine if health effects from the secondary food exposure pathways are likely to be of concern, and hence if detailed evaluation of these exposures is necessary.

On the basis that (a) the predicted air concentrations of dioxins are significantly less than in Europe, Japan or what has been measured in the air elsewhere in Australia, and (b) calculated total intake (background, direct inhalation and via food) is much less than the tolerable intake set by health authorities, it was concluded there is negligible health risk associated with dioxin emissions from the proposed power station.

It was similarly concluded that polyaromatic hydrocarbons (PAH) and the metals cadmium and mercury emitted by the proposed power station present negligible health risks via exposure through the food chain.

Contents

Executive Summary 2
Contents 4
Limitations 6

1. Introduction 7
    1.1 General 7
    1.2 What is a health risk assessment? 9
    1.3 Scope of the risk assessment 11

2. Risk assessment methodology 11
    2.1 General overview 11
    2.2 Issue identification 13

3. Exposure assessment 13
    3.1 Exposure pathways and exposure estimations 13
    3.2 Exposure estimations 15

4. Hazard identification/toxicity 19

5. Risk characterisation methodology 21
    5.1 Introduction to hazard quotients and the hazard index 21
    5.2 Interpretation of hazard quotients and indices 23
    5.3 Calculating cancer risk 24
    5.4 Consideration of background exposures 26

6. Risk characterisation 26
    6.1 Direct acute health effects 26
    6.2 Direct chronic health effects: non-cancer 29
    6.3 Chronic health effects: cancer 29
    6.4 Conclusions 31

7. Dioxins 32
    7.1 Introduction 32
    7.2 Overview of assessment methodology 33
    7.3 Comparison with existing air levels 34
    7.4 Calculating dioxin intakes 35
    7.4.1 Background intake of dioxin-like substances 37
    7.4.2 Relative contribution to intake by inhalation 37
    7.4.3 Calculations 38
    7.5 Conclusions 40

8. Secondary exposure pathways 40

9. Uncertainty discussion 43

References 45

Appendix 1: Health effects and guideline values of emission constituents 49
A1.1: Toxicity category summary 50
A1.2: Summary acute health effects guidelines 54
A1.3: Summary chronic health effect guidelines 57
A1.4: Derivation of guideline values for criteria pollutants 59
A1.5: Polyaromatic hydrocarbons (PAHs) 72
A1.6: Dioxins 75
A1.7: References for Appendix 1 80

Appendix 2: Screening for secondary exposure pathways 88
A2.1: Introduction 88
A2.2: Polyaromatic hydrocarbons 88
A2.3: Metals 89
A2.3.1: Development of screening criteria for metals: 90
A2.3.2: Discussion of metal screening criteria 93
A2.3.3: Application of criteria 95
A2.4: Notes on the bioaccumulation of metals. 97
A2.5: References for Appendix 2 100

Appendix 3 102

Limitations

This report was prepared by Toxikos Pty Ltd for Connell Wagner Pty Ltd (the 'Client'). The material in it reflects Toxikos' best judgement in the light of the information available to it at the time of preparation. However, as Toxikos cannot control the conditions under which this report may be used, Toxikos will not be responsible for damages of any nature resulting from use of or reliance upon this report. Toxikos' responsibility for the information herein is subject to the terms of engagement with the Client.

This report is based on the scope of services defined by the Client, budgetary and time constraints imposed by the Client, the information supplied by the Client or its agents, and the method consistent with the preceding. Toxikos has not attempted to verify the accuracy or completeness of the information supplied by the Client, or its agents.

About Toxikos Pty Ltd

Toxikos Pty Ltd is a consulting company formed on December 1st 2000 to provide clients with independent excellence in toxicology and health based risk assessment. Its charter is to assist industry and government make science based decisions regarding potential effects and management of environmental and occupational chemicals. For over twelve years, prior to and since the establishment of Toxikos, staff have provided toxicology and health risk assessment advice to clients in a wide range of industries and government in Australia, New Zealand and South Africa.

About the author: Dr Roger Drew is one of the Directors and Principal consultants of Toxikos Pty Ltd. He has primary degrees in biochemistry and pharmacology and postgraduate degrees in toxicology. Postdoctoral training was undertaken at the National Institutes of Health, National Cancer Institute in the USA and he spent twelve years teaching medical students and conducting toxicological research at Flinders University of South Australia. He was corporate Toxicologist to ICI/Orica Pty Ltd for twelve years before creating Toxikos Pty Ltd. Dr Drew is the only consultant toxicologist in Australia certified by the American Board of Toxicology.

Dr Drew has been a toxicology consultant to Australian Federal and State Authorities; a member of several standing expert committees of the National Health & Medical Research Council of Australia and the National Occupational Health and Safety Commission of Australia. He has been a member of many expert task groups of the World Health Organization for the International Programme on Chemical Safety.

Dr Drew was Adjunct Professor of Biochemical Toxicology at RMIT University and teaches various aspects of toxicology to undergraduate and postgraduate students at RMIT, Monash and Melbourne Universities. He is a member of several professional toxicology societies and is a recognised national and international expert in toxicology and risk assessment. He is currently on the editorial board of the international scientific journal "Regulatory Toxicology and Pharmacology" and a past board member of the Australian National Research Centre for Environmental Toxicology.

1. Introduction

1.1 General

A biomass power station is intended to be built at Manjimup in SW Western Australia. The power station will generate electricity by burning plantation waste (i.e. cuttings, tree limbs, bark). The site is currently cleared, immediately surrounded by State Forest and beyond by farmland. The nearest farmland is approximately 0.7 km from the proposed facility but the majority is more than 2 km away. At distances of 1.4 – 2.3 km there are three residential dwellings; these are to the north (1.4 km), NW (2.3 km), and SSW (1.9 km). The township of Manjimup is 10.4 km distant to the NNE (CW 2008a) and the coast approximately 30 km SW of the site.

Dominant winds in autumn and winter are from the north and south west. Summer winds are mainly from the south and are stronger than in any other season. Throughout the year only a small percentage of winds blow from the NE (CW 2008a).

To facilitate the assessment Connell Wagner supplied Toxikos with an extract of an air dispersion modelling report (CW 2008a). This contained a summary of the modelling undertaken with TAPM and the estimated ground level concentrations (GLCs) for a number of emission constituents considered by the modeller to be import for assessing the impact of emissions. The GLC information in CW (2008a) was refined and supplemented by additional modelling and provided in a series of emails to Toxikos (CW 2008 b,c,d,e,f,g,h,i) and consolidated in a letter report to Toxikos (CW 2008g). Mass emission rates for the emission components were primarily estimated using methods contained in the National Pollution Inventory (NPI) 'Emissions Estimation Technical Manual for Combustion in Boilers".

The overall methodology employed in this preliminary risk assessment is consistent with that of the Australian enHealth Council (enHealth 2004), the US Environmental Protection Agency (US EPA 1989, 2000a) and the US Agency for Toxic Substances and Disease Registry (ATSDR 1992).

However Toxikos has not assessed the veracity of the emissions estimations or dispersion modelling undertaken by Connell Wagner. The GLCs provided to Toxikos by Connell Wagner have been used on face value in the risk assessment. The air dispersion modelling report by Connell Wagner should be consulted in regard to the likely locations of the maximum GLCs for each compound modelled.

There are a number of important considerations that need to be kept in mind when interpreting the information contained in this risk assessment:

1. The predicted ground level concentrations (GLCs) are not real, they are conservative predictions based on general engineering knowledge of the emission profiles of the equipment used to produce the electricity, the efficiency performance of pollution abatement apparatus that will be installed in the plant, and assumptions regarding the meteorological conditions dispersing the emissions. Additional conservatism is embedded in the HRA as described in the various sections of this report.
2. The exact details of the major emission components that will be released to air will not be known until the plant is built and appropriate stack testing has been done. Consequently, although the major emission components have been addressed, i.e. the ones recognised from experience as being potential problems if not abated, not all possible compounds may be included in the HRA. This is not in itself a significant problem because the HRA shows the health risks from the BSP are overshadowed by background airborne substances, also the HRA purposefully errs on the side of safety. In addition, it is Toxikos' understanding that the proponent has committed to producing a detailed emissions inventory as soon as the plant has been built, if in the unlikely situation, evaluation of the impact of any of the emissions gives rise for concern the proponent will appropriately ameliorate the situation.
3. The HRA has primarily been conducted using the *maximum* predicted GLCs that will occur anywhere within the dispersion modelling domain as provided by Connell Wagner. Such maximum concentrations will rarely occur. Where appropriate, in order to provide contextual information a risk assessment has also been performed using estimates for the 98th percentile GLC.
4. In the HRA when assessing potential effects from exposure to the emission mixture it has been assumed maximum or 98th percentile concentrations of different emission constituents might occur at the same time and at the same place, in concert with maximum (or 98th percentile) background concentrations of particulate matter, NO_2 and SO_2. Intuitively these concentrations are unlikely to coincide.
5. The proposed facility will be built in a forested rural area with only a few houses within 2 km.
6. For exposure to occur an individual must be in the same location, at the same time, as the predicted concentrations occur.
7. The HRA is preliminary, and subject to refinement as more detailed and certain information becomes available.

1.2 What is a health risk assessment?

Health is defined by the World Health Organization as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity (WHO 1948)[1]. Well being is broadly described as an individual's self assessment of their state of happiness, healthiness and prosperity. It relates to the quality of life and ones ability to enjoy it[2]. There are many social and economic factors that impinge upon well being.

The following are examples of determinants of health well being (enHealth 2004, NHC 2005):

- social and cultural factors (e.g. social support, participation, access to cultural resources).
- economic factors (e.g. income levels, access to employment, property values).
- environmental factors (e.g. land use, air quality).
- population-based services (e.g. health and disability services, leisure services).
- individual/behavioural factors (e.g. physical activity, smoking).
- biological factors (e.g. age).

According to enHealth (2004) all developments have a potential impact on health. Some will have positive health impacts by providing jobs, attracting health services to an area, and improving overall economic well being of a community etc. Other projects may have negative impacts such as increased risk of disease, social disruption, increased noise etc. Many developments will have both positive and negative aspects.

Air quality is one of the many parameters influencing well being. This HRA seeks to evaluate, in a predictive manner, the likelihood for direct health effects should exposure to emissions from the proposed biomass power station occur.

An assessment of the holistic nature of health as per the WHO definition is usually termed a health impact assessment. They are not readily undertaken without commitment of significant resources and usually limited to major infrastructure projects, they are usually qualitative and draw upon a wide range of information within the environmental impact statement. Health impact assessments are also done once an impact has occurred and there is a need to identify the possible causes for mal-health. On the other hand, a prospective health risk assessment is an analysis that uses information about toxic substances to estimate a theoretical level of risk for people who might be exposed to defined levels of these substances in the future. The

[1] Although it has been reviewed by the WHO since 1948 this definition of health has not been amended.
[2] This description of health is consistent with the enHealth (2004) model of health and the concepts of well being in New Zealand (NHC 2005).

information comes from scientific studies and measurement data of factory emissions and knowledge of the efficiency of emission control strategies that are/will be put in place. This information is interpolated to estimate emissions from the proposed facility. The risk assessment helps regulatory officials, facility managers and the public determine strategies that will ensure overall protection of human health and the environment should the proposed development proceed. In other words, the risk assessment is undertaken to help define the boundaries/conditions under which the proposed development may obtain approval. Approval usually only occurs when the regulatory authorities are satisfied that the appropriate conditions have been put in place to ensure the future safety of the public.

It is important to note a prospective risk assessment does not measure the actual health effects that hazardous substances may have on a community because the development project has not yet taken place. Risk assessments are often conducted by considering possible or theoretical community exposures predicted from air dispersion modelling of 'known' or assumed concentrations of emissions from a specific point of release at the industrial facility.

During the risk assessment analysis, the most vulnerable people (e.g. children, the sick and elderly) are carefully considered. Conservative safety margins are built into a risk assessment analysis to ensure protection of the public. Therefore people will not necessarily become ill even if they are exposed to materials at higher levels than those estimated by the risk assessment. Commensurate with common international practice, a safe exposure level for the substance is taken to be the ambient air guideline established by a competent authority. Air guideline values (i.e. standards) are established with protection of the most vulnerable people in mind and usually contain wide margins of safety (NHMRC 2006).

The risk assessment helps answer common questions for people who might be exposed to hazardous compounds in the environment, in this case components of the emissions from the proposed biomass power station:

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

1.3 Scope of the risk assessment

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

The HRA includes an assessment of acute and chronic health risks that might arise from:

- Direct inhalation exposure to individual components of the emissions.
- Direct inhalation exposure to the mixture of chemicals in the emissions.
- A quantitative evaluation of cancer risks.
- A preliminary evaluation of the potential health risks associated with exposure of emission components through the food chain.
- A qualitative statement regarding the potential of tank water collected from roofs to be contaminated by the emissions.

The HRA therefore evaluates the potential of emissions from the proposed power station to cause direct toxic effects on individuals who may be exposed either on a short term, infrequent basis, or long term; the latter assumes 24 hours per day for each day of the year for 70 years and is thus conservative (i.e. errs on the side of safety), and also through possible exposure through food.

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

2. Risk assessment methodology

2.1 General overview

The overall methodology employed in this risk assessment is consistent with that of the Australian enHealth Council (enHealth 2004) as endorsed by the Western Australian Department of Health (WA DoH 2006), the principles of the NHMRC (2006), the US Environmental Protection Agency (US EPA 1989, 2000a) and the US Agency for Toxic Substances and Disease Registry (ATSDR 1992).

Although this risk assessment is quantitative, there are aspects that are primarily of a screening nature due to the fact that it deals with risks for persons who are hypothetically exposed to the highest atmospheric emission concentration that is reasonably expected to occur anywhere within the air dispersion modelling domain.

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

By necessity, to ensure protection of public health this risk assessment is conservative; that is, it errs on the side of safety by over predicting the likelihood for health risk. However to provide reality and contextual information in the assessment a qualitative analysis has been undertaken for the uncertainty inherent in the assessment. Although detailed aspects of uncertainty are discussed within the section where a particular topic is discussed, the major uncertainties are drawn together in overview at Section 9. Many of the uncertainties have already been flagged in the introduction where the reader is alerted to a number of issues that should be borne in mind when reading the HRA.

International and Australian regulatory agencies consider a ‘safe’ exposure level to be the same as, or less than, the relevant regulatory standard (i.e. an ambient air guideline value, AGV). Hence, by definition an unacceptable health risk potentially occurs when the predicted ground level concentration is greater than the regulatory standard. The process of characterising the health risk by comparing predicted ground level concentration (GLC) to an AGV is common practice in risk assessments for air pollutants (Morello-Frosh et al. 2000, Pratt et al. 2000, Tam and Neumann 2004). It is a pragmatic approach used to identify important chemicals in polluted air or industrial emissions. The ratio of the GLC to AGV is called the ‘hazard quotient’ (HQ). By adding hazard quotients together to yield a hazard index (HI), an appreciation of the likelihood of an adverse health outcome from exposure to the emissions as a mixture can be obtained. The mechanics and interpretation of this method is described in Section 5.

2.2 Issue identification

With respect to direct health impact, the basic issues associated with air emissions from the proposed power station have commonality with almost any new, or proposed expansion of an industrial facility, they revolve around the following:

1. There maybe community concern that there may be new substances, currently not released into the air, which if emitted in sufficient quantity may cause health effects not presently experienced by residents in the area.
2. People may be worried the proposed expansions may increase current emissions and cause, or exacerbate health effects.

3. Exposure assessment

3.1 Exposure pathways and exposure estimations

Exposure to substances in the emissions from the power station may occur through a number of different environmental media (Figure 3.1). However, direct exposure to the emissions via inhalation is usually by far the most important exposure route and is the primary subject of this HRA.

Nevertheless, exposure to components in the emissions may also theoretically occur via deposition directly onto food plants or soil which may be ingested by humans and/or food producing animals. Also some contaminants in soil or deposited onto plants may theoretically be bioaccumulated by the plants, or animals that feed upon them and are used as food sources for humans. Over the last few decades it has been appreciated that only relatively few chemicals have the potential to present significant health risks to humans as a result of accumulation through the food chain. Such chemicals have a common set of chemical and biological properties:

- They are poorly degraded in the environment and hence have long environmental half lives.
- They are only slowly metabolised and excreted by animals and humans and hence have long biological half lives in the body.
- If organic compounds, they are highly soluble in fat which together with the poor metabolism means they can accumulate and be stored in fatty tissue. If this storage in the body is significant, body burdens of the chemical may reach a level where toxicity might occur.

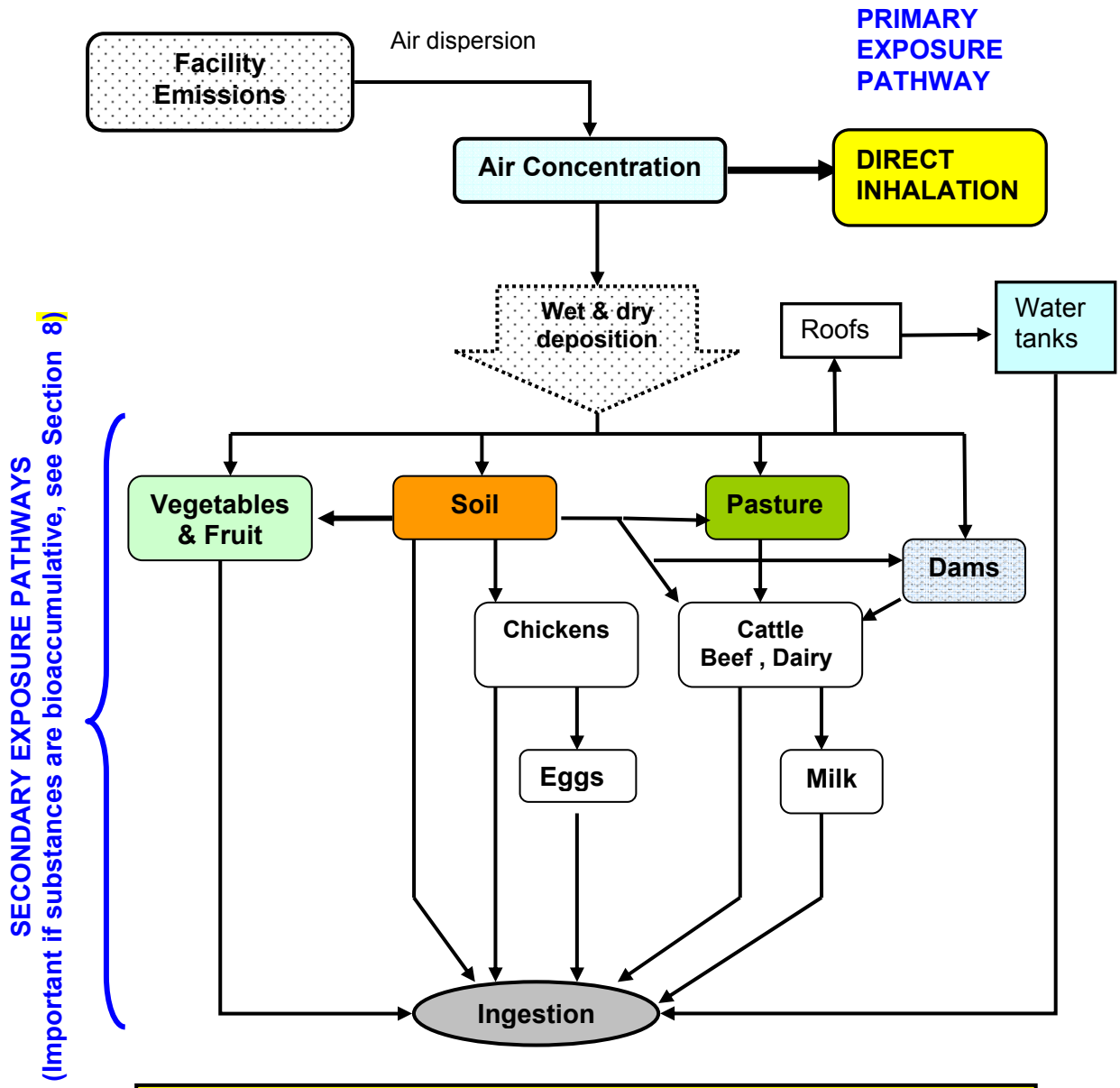

Figure 3.1: Theoretical exposure pathways to air emissions.

Organic chemicals in emissions from the proposed facility that have the above properties are dioxins/furans, but these will be released in very small amounts and do not pose a risk to human health (see Section 7).

Other substances that may be emitted into the environment that have the potential to bioaccumulate under certain conditions are cadmium (Cd) and mercury (Hg). These also are released in very small amounts and also do not pose a risk to health (Section 8).

3.2 Exposure estimations

Connell Wagner provided Toxikos with various documents from which the ground level concentrations of emission components in Tables 3.1, 3.2 and 3.3 were obtained. These represent exposures that are considered to be higher than reality because:

- they are maximum GLC concentrations or 98th percentile values,
- will occur infrequently,
- are unlikely to occur at the same location for all compounds and background,
- are unlikely to occur at the same time, and
- are unlikely to occur when a person is present at the location.

Not withstanding the above, to factor a person's behaviour (i.e. average daily movements) into a risk assessment is quite challenging, and is rarely done. Instead, the assumption is made that throughout their entire life a person is in a situation where they could be exposed to the highest concentrations predicted to occur by the dispersion modelling. This assumption adds conservatism (i.e. safety) into the risk assessment. One way of adding a touch of realism and contextual information to the exposure assessments is to conduct the HRA with different percentile estimates of GLC. Since these are statistical estimates which take into consideration the variability of meteorological conditions they also represent probability estimates for the frequency with which a given GLC is likely to occur at a specified location. For this HRA a range of percentile GLC data were not available hence it is not possible to provide the reader with an appreciation of likelihood of effects other than in qualitative discussion.

Table 3.1 summarises the predicted maximum GLC concentrations due to the power station on its own. It should be noted that data are available for a relatively limited number of compounds. Although these more than likely account for the majority of the mass emissions, there are nonetheless additional substances in the emissions that are not addressed in this risk

assessment. However in Toxikos experience other VOCs, not explicitly estimated for this HRA, do not materially affect the outcome of health risk assessments.

Table 3.2 provides GLCs for the maximum and 98th percentile 24 hour average GLCs for PM_{10} and $PM_{2.5}$ for the power station and background. Background particulate data has been sourced from measurement undertaken by CSIRO at Manjimup for the period 30/11/06 to 14/12/07. For NO_2 and SO_2 the corresponding GLCs are in Table 3.3. The air dispersion modelling report of Connell Wagner should be consulted for a detailed explanation of background data. In the absence of different percentile estimations for other substances (i.e. other than PM, NO_2 and SO_2) their maximum GLC concentrations were used with the 98th percentile GLCs of PM and NO_2 to determine the likelihood of health effects.

Table 3.1: Maximum anywhere ground level concentrations (GLCs) attributed to the proposed power station.

Emission constituents [a]	Maximum Predicted GLC [a] ($\mu g/m^3$)		
	1 hour (alternative averaging period in parenthesis)	**24 hour**	**Annual**
NO_2	60.5 [d, n]	26.1 [d,n]	3.2 [d,n]
SO_2	65.8 [b] (86.8 =15 min ave) [c]	5.7 [b] (7.5 =15 min ave) [c]	1.02 [b]
CO	7.5 [l] (4.9 = 8 hour ave) [c]	2.1 [i]	2.1 [p]
PM_{10} [g]	-	3.23 [n]	-
$PM_{2.5}$ [g]	-	3.2 [L]	-
PAH	0.0064 [j]	-	0.00099 [n]
Dioxin & Furans	6.8×10^{-9} [N/A] [h]	-	0.00000000097 [b]
VOCs [h]			
TVOCs	1.25 [i]	0.3 [i] [N/A]	0.3 [i]
Ethyl benzene	0.006 [n]	-	0.00088 [n]
Phenols [m]	0.01 [n]	-	0.00044 [n]
Styrene	0.37 [n]	-	0.054 [n]
Toluene	0.18 [n] (0.13 = 6 hour ave) [c]	-	0.026 [n]
Xylene	0.005 [n] (0.006 = 30 min ave) [c]	-	0.00071 [n]
Benzene	0.25 [n] (0.17 = 6 hour ave) [c]	-	0.0036 [n]
Aldehydes [h]			
Acetaldehyde	0.16 [n]	0.085 [c]	0.023 [n]
Benzaldehyde	0.00017 [n]	-	0.000024 [n]
Crotonaldehyde	0.002 [n]	-	0.00028 [n]
Formaldehyde	0.87 [n] (1.0 = 30 min ave) [c]	-	0.12 [n]
Iso-butyraldehyde	0.024 [n]	-	0.00034 [n]
Propionaldehyde	0.012 [n]	-	0.0017 [n]
o-Tolualdehyde	0.0014 [n,o]	-	0.0002 [n]
p-Tolualdehyde	0.0022 [n,o]	-	0.00031 [n]
Metals			
Arsenic	0.000012 [n] (9×10^{-6} = 4 hour ave) [c]	-	0.0000017 [n]
Cadmium	0.000022	-	0.0000031 [f, n]
Chromium III	0.000016	-	0.0000017 [n]
Chromium VI	0.000004	-	0.00000058 [n]
Copper	0.000027 [n]	-	0.0000039 [n]
Lead	0.00009	-	0.000013 [j,n]
Manganese	0.001		0.00014 [n]
Mercury	0.000052 [n]	-	0.0000074 [f,n]
Nickel	0.000079 [n]	-	0.0000011 [n]
Selenium	0.000019	-	0.0000027 [n]

Footnotes for Table 3.1

N/A = Not applicable, not used in the HRA. This averaging time does not have an air guideline value (AGV) because toxicological concerns are with chronic exposure, not acute.
NM = Not Modelled.
[a] Emission constituents and ground level concentrations (GLC) were obtained from Connell Wagner (CW 2008a).
[b] From Tables provided with email dated 8th April 2008 (CW 2008h).
[c] Where an AGV is not grounded on a toxicological or health effect, the modelled 1 hr average GLC was adjusted to the relevant time frame of the AGV using the Power Rule [$C_2 = C_1 \times (T_1/T_2)^p$ where p = 0.2].
[d] GLCs were provided in parts per billion (ppb) and were converted to µg/m^3 at STP (25^{o}C and 760 mm Hg). From CW (2008b) the TAPM estimates for NO_2 GLC from BPS are 32.2 ppb, 13.9 ppb & 1.7 ppb for 1 hr, 24 hr & annual average respectively.
[e] No background data provided.
[f] Data provided by CW (2008c).
[g] See also Tables 3.2 & 3.3 for additional percentile GLC and background estimates.
[h] Estimates for these organic compounds are for uncontrolled emissions (CW 2008g).
[i] From Table 1.17 (CW 2008a).
[j] From CW (2008a).
[k] From Table 1.15 (CW 2008a).
[L] From Table 1.16 (CW 2008a).
[m] Taken as phenol.
[n] Data from CW (2008g).
[o] An acute guideline value was not located for tolualdehyde, it was therefore assumed that this substance may have health effects (sensory irritation) equivalent to the most potent of the other aldehydes. As judged from the AGVs in Appendix 1, crotonaldehyde is the most potent as it has the lowest AGV.
[p] Considered to be worst case; since the annual average was not modelled it was assumed to be the same as the 24 hour maximum modelled average (CW 2008a).

Table 3.2: Estimates of particulate GLCs (Maximum and 98th percentile 24 hr averages) – background and cumulative

Scenario	PM_{10} (µg/m^3)		$PM_{2.5}$ (µg/m^3)	
	Maximum	98th %'le	Maximum	98th %'le
BSP	3.23	0.68	3.2	0.68
Background [b]	79.2	29.1	58.9	21.8
Cumulative	82.4	29.78	62.1	22.48

[a] Ground level concentrations (GLC) were obtained from Connell Wagner (CW 2008g, h, i and personal correspondence).
[b] Background particulate data has been sourced from measurement undertaken by CSIRO at Manjimup for the period 30/11/06 to 14/12/07 (CW (2008i).

BPS = Biomass Power Station

Table 3.3: Nitrogen dioxide GLC estimates (µg/m^3) –background and cumulative

Scenario	1 hour ave		Annual ave
	Maximum	98th %'le	
NO_2			
BSP	60.5	15.6	3.2
Background [a]	-	-	-
Cumulative	88.7	43.8	3.02
SO_2			
BSP	65.8 (86.7 = 15 min)[b]	5.7 (7.5 = 15 min)[b]	1.02
Background [a]	-	-	-
Cumulative	72.8 (96 = 15 min)[b]	10.92 (14.4 = 15 min)[b]	1.52

[a] Specific information for existing background GLC of NO_2 and SO_2 was not generated, nevertheless background sources of NO_2 & SO_2 were included in dispersion simulations for the cumulative scenario of BSP and background (CW 2008h, i).

[b] For calculation of hazard indices the 15 min SO_2 average is used as this averaging time matches the time of the health based acute guideline value.

4. Hazard identification/toxicity

Appendix 1 contains very brief summaries, in tabular and text format, of the primary health hazard associated with the emission components. Included is information associated with the sensitive endpoint upon which the air guideline value was set and a sketch overview on how the air guideline value was established. The latter is useful as it provides the level of safety that a regulatory authority has built into the guideline.

The information in Appendix 1 has been confined to identifying the broad toxicological effect categories for each chemical (e.g. carcinogenicity, genotoxicity, reproductive toxin, central nervous system depression (narcosis), respiratory tract effects etc). These may not be the health effects of concern for which the relevant health guideline has been established. Because this is a screening hazard assessment, review documents and electronic databases produced by competent agencies [3] have been used as information resources rather than conducting a

[3] National Environment Protection Council (NEPC), Australia; World Health Organization (WHO)-International Programme for Chemical Safety (IPCS) & International Agency for Research on Cancer (IARC); Agency for Toxic Substances and Disease Registry (ATSDR), US Dept Health & Human Services; Office of Environmental Health Hazard Assessment (OEHHA), California EPA; The Dutch National Institute of Public Health and the Environment (RIVM); and the Integrated Risk Information

thorough toxicological evaluation for each chemical. The information in Appendix 1 does not take into consideration the exposures necessary to cause the toxicity that has led to the categorisation. Consequently, although a competent authority, or review, may consider the substance of being capable to cause the effect noted in the Appendix at some level of exposure, in reality exposures may never be high enough for it to occur.

Because overview documents or electronic databases have been used to determine the hazard category of emission components a formal assessment has not been made regarding dose response aspects for all emission components, or whether the toxicological effects used to categorise the potential hazard have a realistic probability of being realised at the exposure levels in question for the scenarios evaluated herein. That is, there has been no evaluation to determine the exposure circumstances required for different effects to be elicited for a given chemical. General toxicological knowledge shows that for many of the compounds the doses required to cause, say, liver toxicity are much higher than the dose required to cause the most sensitive health effect (which forms the basis of the relevant guideline). For example, the acute or chronic health guideline for a particular chemical may be based on irritancy or perhaps central nervous system depression because this is the most sensitive health end point, but at higher concentrations some other effect may occur that another agency has used to classify the compound as possessing a particular hazard capability if the exposure is high enough. Thus because public health guidelines are based on the most sensitive effect, usually the effect that occurs at the lowest concentration, comparison of modelled ground level concentrations with the guideline will automatically take into account effects that may occur at higher exposures of the chemical.

There are number of substances in the emissions that are known human carcinogens, or are regarded by the International Agency for Research on Cancer (IARC) as being probable or possible human carcinogens by inhalational exposure: arsenic, chromium^{IV}, cadmium, actaldehyde, benzene, and PAHs. For these substances it is commonly regarded, for regulatory purposes, that theoretically there is no absolutely safe level of exposure and that there is some level of cancer risk at any exposure. The level of this risk is calculated for each substance and described in Section 6.3.

System (IRIS), US EPA. Wherever it has been practical to do so, the hierarchal preferred reference list of enHealth (2004) has been used to source guidelines.

5. Risk characterisation methodology

5.1 Introduction to hazard quotients and the hazard index

For assessing the potential non-cancer health impact of individual chemicals, predicted ground level concentrations are compared to individual health based ambient air guidelines generated to protect public health. This comparison is performed by calculating a hazard quotient[4] (HQ) which is the ratio of ground level concentration (GLC) to the ambient air guideline value (AGV)[5].

The hazard quotient[6] is calculated for each contaminant using the simple equation below.

HQ = Estimated ground concentration/Health based air guideline value …….**Equation 1**

For assessing the potential effects of the mixture of chemicals in the emissions, it has been assumed individual components may have additive effects and an overall hazard index (HI) has been calculated (US EPA 2000a). The HI is the sum of all the emission component hazard

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

[5] The hazard quotient is commonly reported to one significant figure (US EPA 1989). For example, a hazard quotient of 0.13 is rounded to 0.1, and a hazard quotient of 1.6 is rounded to 2. In this risk assessment, HQs and HIs in summary tables have also been calculated to one significant number. This is done so as not to imply there is a certain level of precision in the assessment when using HQs and HIs.

[6] For the hazard quotient to be informative both the predicted ground level concentration estimated from the air dispersion modelling and the air guideline value must relate to the same time frame of exposure, i.e. averaging time. In this risk assessment the modelled 1 hr average GLC was adjusted to the relevant time frame of the AGV using the Power Rule [$C_2 = C_1 \times (T_1/T_2)^p$ where $p = 0.2$].

quotients determined from either the acute or chronic air guideline values[7], thus an acute and a chronic hazard index[8] can be generated.

$$HI_j = \sum HQ_{i \ldots j} \quad \textbf{Equation 2}$$

Where HI_j is the sum of HQ's for all pollutants from i to j

This process assumes:

- there is a threshold level of exposure below which no adverse health effects will occur,
- either the toxicological effect of chemicals and/or the dose is additive, and
- multiple subthreshold exposures may result in an adverse health effect.

In strict toxicological terms it is only valid to sum the effects and/or dose of chemicals if they have the same mode of toxicological action and affect the same target tissues. Similarly it is not be expected for substances in a mixture to have interactive health impacts if they were individually present at concentrations significantly below their biological threshold levels (i.e. below their true low observed effect level)[9]. Some investigators therefore prefer only to sum

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

[8] In some instances the reported HI in a table may not add up to the sum of the HQs. The apparent discrepancy is the result of rounding the HQs for tabular reporting versus use of the actual numerical HQ values that were summed to reach the HI.

[9] Because the true LOAEL cannot be readily established empirically, for public health purposes the experimental no observed adverse effect level (NOAEL) is often taken as being the threshold exposure level for eliciting an adverse health effect. Sometimes any meaningful biological effect, whether adverse or not, is taken as the threshold exposure, such an exposure level is called the no observed effect level (NOEL). It should be noted however that the NOEL, the NOAEL and the LOAEL are all influenced by the experimental design of toxicology studies, especially the dose spacing intervals. It should especially be noted that because air guideline values usually have large uncertainty/safety factors incorporated in them, that a HQ less than one signifies the GLC is much less than the biological threshold concentration for causing an effect.

hazard quotients for pollutants that effect common organs, thus yielding effect-specific cumulative HIs (Fox et al. 2004, Morello-Frosch et al. 2000). Others, while recognising that adding HQs with different health end points will not give an accurate idea of the non-cancer HI nonetheless add all HQs together (Pratt et al. 2000). Some investigators limit this latter practice to only those pollutants whose HQ is greater than unity (Tam and Neumann 2004) (i.e. for substances whose concentrations may be exceeding guidelines and perhaps nearing their biological thresholds).

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

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

5.2 Interpretation of hazard quotients and indices

An 'unacceptable' risk, as defined by regulatory standards and requirements, is often determined as the exposure (i.e. GLC) being larger than the air guideline value used to calculate the hazard quotient, i.e. the HQ>1. Because of the safety factors incorporated into the majority of AGV, this definition of unacceptable risk does not equate with imminent adverse health effects or even high risk of adverse health effects. It simply means that the health guideline level has been exceeded.

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

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

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

5.3 Calculating cancer risk

The lifetime risk of developing cancer for exposure to carcinogens whose mode of action is by directly altering genetic material (i.e. they are genotoxic) is calculated by multiplying the average lifetime chemical exposure by an estimate of the carcinogenic potency of the chemical. The

latter is commonly called the unit risk factor, or slope factor. For air borne carcinogens, the "unit" is generally 1 µg/m^3 and depending on the nature of the data used to determine the carcinogenic potency, the numerical value refers to the probability of developing or dying of cancer. Thus a lifetime exposure to 1 µg/m^3 of a substance may carry a risk of 1 chance in 2,000 of developing cancer; this is often interpreted as meaning, if 2000 people were exposed to 1 µg/m^3 for their lifetime then one individual may develop cancer. This probability is expressed as 0.5 in 1000, or 0.5 x 10^{-3} per µg/m^3, written as 0.5 x 10^{-3} (µg/m^3) $^{-1}$. The target acceptable risk band adopted in many countries is 1 x 10^{-6} to 1 x 10^{-5}; i.e. with a lifetime exposure there is a chance developing a tumour between one in a million and one in one hundred thousand[10].

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

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

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

It is common practice to assume cancer risks due to different genotoxic carcinogenic air pollutants are additive. Summing the individual cancer risks is used to estimate a total lifetime risk of developing cancer (Morello-Frosch et al. 2000, Tam and Neumann 2004, Pratt et al. 2000). However unit risk estimates are upper bound 95% confidence estimates and do not reflect the central tendency or average. When several upper bound estimates are added together, a question is raised as to whether the predicted cancer risk is plausible. The greater the number of carcinogens being considered the more unlikely the true risk for each carcinogen will lie near the upper bound estimate. The process of adding upper bound cancer risk estimates together is inherently conservative. Cogliano (1997) has shown that the resulting risk estimate becomes increasingly improbable the greater the number of risk estimates added, but nonetheless is not necessarily misleading. However, to obtain a cancer risk estimate closer to

[10] Risk assessments for cancer or chronic non-cancer health effects assume an individual is exposed to the substances at the defined concentrations, usually those at the high end of possible air concentrations, for their entire life time. Points to note are most people in Australia do not live in one location for their entire lifetimes, and the high concentrations do not occur all the time exposure is assumed to occur.

the true risk Cogliano (1997) considers central estimates of risk yield a more plausible result. This requires central estimates of cancer potency which are generally not readily available.

5.4 Consideration of background exposures

It is usual to include background exposures when assessing health risks to industrial emissions. Although the location of the proposed power station is such that it would not be expected there would be significant contribution to background exposures from other industrial sources there are a few components in the emissions from the power station that are inherently expected to be in the receiving air shed as part background, and hence at least theoretically could contribute to health effects. For example it is likely there are background concentrations of particulate matter (PM_{10} and $PM_{2.5}$) due to domestic fires or burning off, the result of wind-raised dust from paddocks, and wind swept salt spray.

It is noted that background assumptions of particulates significantly affects the risk calculations (HIs) for acute and chronic health effects. The background information used by Connell Wagner in the dispersion modelling came from recent measurements undertaken by the CSIRO at Manijimup.

6. Risk characterisation

6.1 Direct acute health effects

Table 6.1 and Figure 6.1 summarise the acute hazard indices for the predicted ground level concentrations of substances in the emissions. For the power station the calculated HIs are <1, indicating the emissions from the power station on its own is unlikely to cause acute health effects. However in the cumulative scenario where other sources of particulates, NO_2 and SO_2 are incorporated in the dispersion modelling some of the HI's move above the target HI of 1.

Because $PM_{2.5}$ is a subset of PM_{10} the HIs have been calculated using the maximum GLC of any substance with either the maximum or 98^{th} percentile estimated GLC of PM_{10} or $PM_{2.5}$. From Table 6.1 it is apparent that the combination of maximum BSP emissions with maximum background PM_{10} GLC results in a hazard index somewhat above the target of unity. When 98^{th} percentile GLCs are used the HI is <1, indicating little risk of health effects.

For $PM_{2.5}$ either combination of maximum or 98th percentile GLCs results in a HI ≥ 1. This does not necessary mean an unacceptable health risk. Approximately 90% of the overall acute health risk is assicted with background PM.

From Figure 6.1 it is readily seen that background particulate estimations swamp the cumulative hazard index. The detail of the background particulate sources, the time of year, or time of day that they occur is not known to Toxikos. From past experience it is likely that the combination of maximum or 98th percentile GLCs of NO_2 and particulate matter from the power station will not coincide on the same days as the maximum or 98th GLCs due to background sources. However while in Toxikos' opinion this is considered to be the likely situation Toxikos is not in a position to definitively demonstrate the same.

Table 6.1: Summary of acute Hazard Indices [a]

	Maximum	98th percentile
With PM_{10}		
BPS only	0.7	0.1
Background	1.6 [b, c]	0.6 [c]
BPS + Background	2.3	0.7
With $PM_{2.5}$		
BPS only	0.4	0.1
Background	3.1 [b,c]	0.9 [c]
BPS + Background	3.5 [b,c]	1.0 [b,c]

[a] The HI calculations are provided in Appendix 3.
Because $PM_{2.5}$ is a subset of PM_{10} the HI has been calculated with either PM_{10} or $PM_{2.5}$ GLC estimates. Since only the 98th percentile GLC was available for PM and NO_2 the maximum estimated GLCs have been used for other substances when calculating the HI.

[b] Shaded cells indicate the calculated HI is above the nominal target of 1.0

[c] These hazard indices are calculated for particulate matter only as background information on other substances was not provided. Nonetheless dispersion modelling for the cumulative scenario (i.e. BPS and background) was performed using biogenic and anthropogenic emissions for NO_2 and SO_2 from other sources affecting the area of the modelling grid (see Table 3.3).

BPS = Biomass Power Station

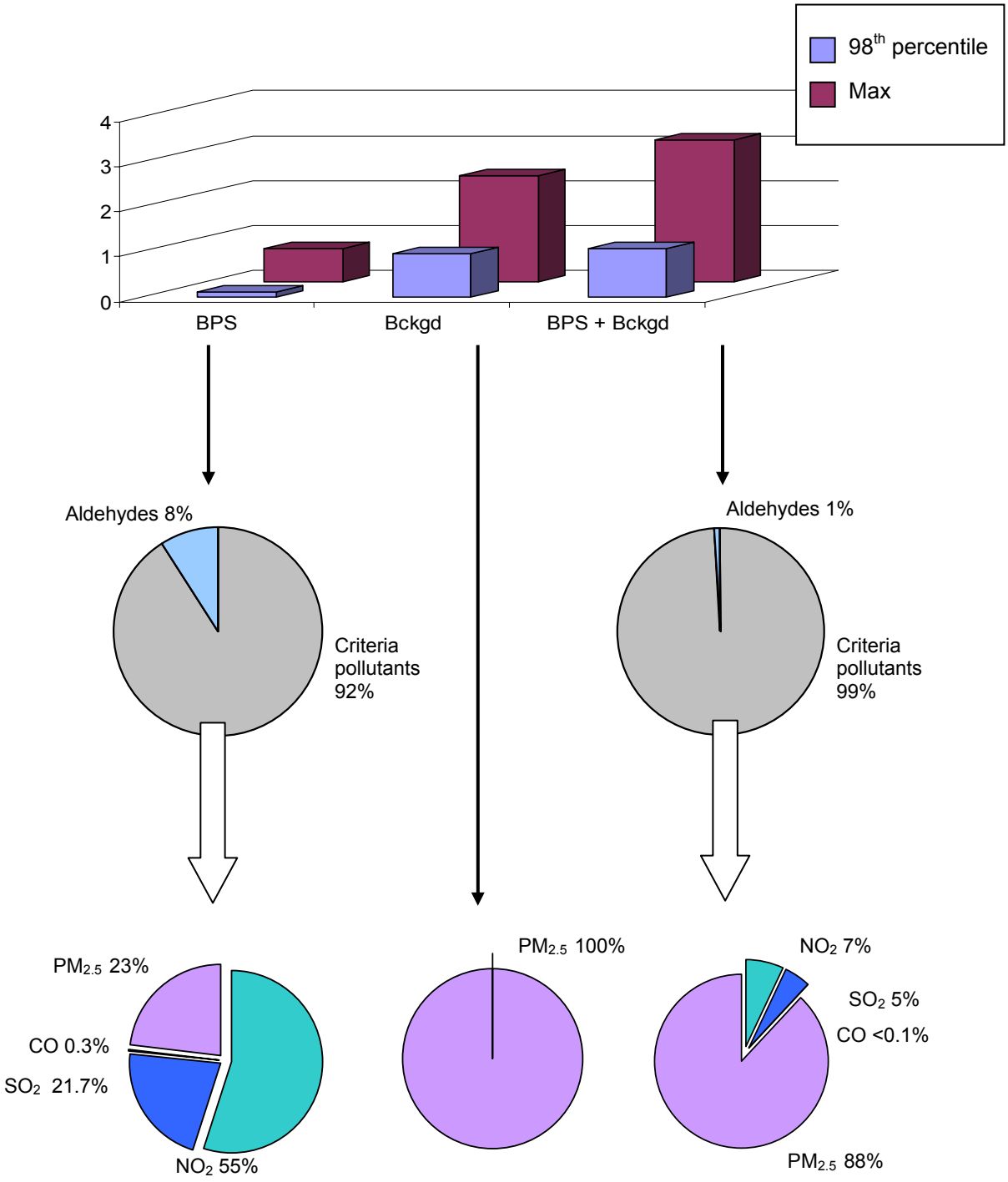

Figure 6.1: Acute Hazard Indices calculated with 98th percentile $PM_{2.5}$
Background sources of PM, NO_2 and SO_2 were included in the simulation modeling of the cumulative scenario (I.e. BSP + bkgd). It is readily seen that background particulates are the major contributor to the health risk of the cumulative scenario of background plus emissions from the BSP.
Metals and VOC other than aldehydes contribute <1% to the overall risk (Appendix 3).

6.2 Direct chronic health effects: non-cancer

Table 6.3 presents the chronic non-cancer hazard indices. The HI's are all less than unity signifying low likelihood of chronic health risks associated with exposure to the emissions should it occur.

Table 6.2: Chronic Hazard Indices

	Hazard Index
BPS only	0.08
Background [a]	0.03
BPS + Background	0.1

[a] The background value relates solely to the nitrogen dioxide. The chronic HI calculations do not include aldehydes as the inhalational toxicity of these compounds relates to respiratory irritation and short-term exposures.

6.3 Chronic health effects: cancer

The methodology for calculating cancer risks is described in Section 5.3. Table 6.3 contains the carcinogenic risks for the individual compounds and also the total cancer risk, assuming additivity between substances.

Overall the emissions from the proposed power station expansion do not pose a significant carcinogenic risk to persons living around the proposed biomass power station.

The calculated total cancer risk[11] is approximately 0.3×10^{-5} (Table 6.3). This is within the commonly accepted risk band of between one in one million and one in one hundred thousand[12]. The higher boundary of this risk range becomes more tolerable as less people are potentially affected.

---

[11] Although all of the substances may produce tumours of the lung they don't all necessarily act on the same cell type or by the same toxicological mechanism. Although benzene may produce pulmonary tumours in animals the signature cancer of benzene is acute myeloid leukaemia.

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

Total PAH equivalents account for 90% of the calculated total cancer risk. Of the total PAH equivalents two individual PAH compounds contribute approximately 83% to the total PAH equivalents (naphthalene 44% and benzo(b,k)flouranthene 39%). The ground level concentrations for each PAH compound were estimated by Connell Wagner (CW 2008g) using emission factors from the relevant Australian NPI emission estimation handbook for combustion in boilers. The emission factors are average values where particulate controls are in place. It is expected by Connell Wagner that these estimates are conservative (CW 2008g).

Table 6.3: Genotoxic Cancer Risk

Emission constituent	Annual GLC [a] µg/m^3	Unit Risk [b] (µg/m^3)$^{-1}$	Cancer Risk [c]
Acetaldehyde	2.34×10^{-2}	9×10^{-7}	2.11×10^{-8}
Arsenic	1.65×10^{-6}	1.5×10^{-3}	2.48×10^{-9}
Cadmium	3.12×10^{-6}	1.8×10^{-3}	5.62×10^{-9}
Chromium VI	5.78×10^{-7}	4×10^{-2}	2.31×10^{-8}
Benzene	3.57×10^{-2}	6×10^{-6}	2.14×10^{-7}
PAH (as benzo(a)pyrene equivalents) [d]	2.8×10^{-5}	8.7×10^{-2}	2.44×10^{-6}
Total Cancer risk			***2.7 x 10^{-6}***

[a] Annual GLCs are from Table 3.1

[b] The following table provides contextual information for each carcinogen including the target organ on which the unit risk factor is based and the reference source, plus the IARC classification (refer to Appendix 1 for a description of the IARC classification scheme.

Compound	Target Organ	IARC	Source
Acetaldehyde	Rat nasal tumours	2B	WHO (2000b)
Arsenic	Lung cancer in workers	1	WHO (2000a)
Chromium VI	Lung cancer in workers	1	WHO (2000b)
Cadmium	Lung cancer	1	WHO (2000a)
Benzene	Leukaemia	1	WHO (2000a)
PAH	Lung cancers	2A	WHO (2000a)

[c] Cancer risk = Annual GLC x Unit Risk

[d] The cancer risk that may be associated with exposure to the PAHs has been calculated using the benzo(a)pyrene equivalency approach in Table 6.4.

Table 6.4: Cancer risk assessment of PAHs

PAH	GLC [a] ($\mu g/m^3$)	B(a)P Potency Equivalence Factor [b,c]	Benzo[a]pyrene equivalence ($\mu g/m^3$)
Acenaphthene	1.50×10^{-5}	0.001	1.50×10^{-8}
Acenaphthylene	1.71×10^{-6}	0.01	1.71×10^{-8}
Anthracene	1.21×10^{-5}	0.01	1.21×10^{-7}
Benzo(a)anthracene	1.14×10^{-5}	0.1	1.14×10^{-6}
Benzo(c)phenanthrene	1.50×10^{-6}	0.023	3.45×10^{-8}
Benzo(a)pyrene	2.43×10^{-7}	1	2.43×10^{-7}
Benzo(b,k)fluoranthene	1.07×10^{-4}	0.1	1.07×10^{-5}
Benzo(ghi)perylene	5.07×10^{-6}	0.01	5.07×10^{-8}
Benzo(k)fluoranthene	2.71×10^{-6}	0.1	2.71×10^{-7}
Benzofluoranthenes	3.86×10^{-6}	0.1	3.86×10^{-7}
Chrysene	1.64×10^{-6}	0.01	1.64×10^{-8}
Fluoranthene	6.57×10^{-5}	0.01	6.57×10^{-7}
Fluorene	2.93×10^{-5}	0.001	2.93×10^{-8}
Indeno(1,2,3-cd)pyrene	1.29×10^{-6}	0.1	1.29×10^{-7}
Methyl anthracene	1.44×10^{-4}	0.01	1.44×10^{-6}
Naphthalene	1.21×10^{-2}	0.001	1.21×10^{-5}
Phenanthrene	1.79×10^{-4}	0.001	1.79×10^{-7}
Pyrene	6.00×10^{-5}	0.001	6.00×10^{-8}
Sum of Benzo[a]pyrene equivalents ($\mu g/m^3$)			**2.8×10^{-5}**
Cancer Risk due to PAH mixture	= Unit Risk Factor x GLC B[a]P equivalence = (2.8×10^{-5}) ($\mu g/m^3$) x (0.087) $(\mu g/m^3)^{-1}$ = **2.4×10^{-6}**		

[a] Ground level concentrations are from CW (2008g).

[b] Potency equivalence factors are from WHO (1998 Table A1.9 pg 660). A potency equivalence factor for Benzo(c)phenanthrene was sourced from Toronto MoE (1997) as it is not available in WHO (1998).

6.4 Conclusions

- It is concluded direct health risks from exposure to the emissions from the power station is unlikely.
- It is not possible with the available data to quantitatively describe the extent to which exposure to components of the emissions may be over or under estimated. Intuition

however suggests the risks may be markedly overestimated. This however cannot be confidently demonstrated.

- The cancer risk assessment indicates the risks are within the band of acceptability used by many Australian and overseas jurisdictions.

7. Dioxins

7.1 Introduction

Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are collectively called ‘dioxins’. Co-planar polychlorinated biphenyls (co-planar PCBs) possess toxicity similar to that of dioxins and are called ‘dioxin-like’ compounds. Dioxin and furan molecules consist of two benzene rings joined together by oxygen atom(s) with various amounts of chlorine or hydrogen atoms attached in vaying combination at any of 8 positions around the molecule. There are potentially 75 kinds of PCDDs, 135 PCDFs and more than 10 co-planar PCBs. The different types of dioxins are called congeners. Humans are invariably exposed to a complex mixture of many dioxins and furans, but the degree of toxicity of different dioxins varies from compound to compound, not all of them are toxic. The tetrachlorinated dibenzo-p-dioxin with chlorine atoms attached in the 2, 3, 7 and 8 positions (2,3,7,8-TCDD usually simplified to TCDD) is known to possess the highest toxic potency and toxic effects of this congener have been the most studied. The toxicity of other congeners is related to TCDD by assigning them a toxicity equivalency factor (TEF). The TEFs in a mixture are them summed after taking into account the relative proportions of each conger present; this summation then gives a toxicity equivalency (TEQ) for the mixture in terms of how much TCDD would produce the same toxicity as the mixture. The TEF scheme was developed by the World Health Organisation and is used by Australian and international jurisdictions to assess the risk associated with exposure to mixtures of dioxins and furans.

In this report the term ‘dioxin’ is used to refer to all dioxins and furans, this term does not include dioxin-like PCBs.

An important fact is that dioxins are produced by anything that is burnt. They are created by domestic fires, automobile engines and volcanoes, as well as by certain industrial chemical manufacturing processes. In Australia the biggest source of dioxins is bushfires.

Thus dioxins are present 'naturally' in the Australian rural and urban environments. Thus all Australians are exposed to dioxins. Dioxins that have been deposited on soil or pasture have the capability of being transferred and concentrated into the fatty tissues of animals. Humans who eat meat and dairy products may therefore also be exposed. More than 95% of the exposure of Australians is via food (OCS 2004).

The World Health Organisation (WHO 1998b) and the Australian Health Authorities have established a safe level of intake of dioxins (NHMRC 2002). In Australia the guideline value for dioxins is called the tolerable monthly intake (TDI) and is set at 70 pg TEQ/kg/m (equivalent to 2.3 pg TEQ/kg bw/d).

Additional contextual information on the toxicity and health effects of dioxins, the relative sensitivity of humans and how the guideline was established can be found in Appendix 1 (Section A1.7).

7.2 Overview of assessment methodology

The assessment of dioxins has been undertaken in two ways:

- Comparison with existing measured air concentrations in various parts of the world and Australia, and
- Calculation of the overall intake of dioxins from combined background and assumed power station emission exposure, and then comparison with the TDI set by Australian authorities.

The first assessment is straight forward, but the second relies on a number of conservative assumptions and data in the scientific literature and is described in Section 7.4 below. Because data from each of the potential secondary exposure pathways depicted in Figure 3.1 cannot be reasonably determined without expenditure of much resource and many years of study, a screening assessment method has been developed by Toxikos to determine whether a detailed examination of those exposure pathways is warranted.

Briefly, the screening assessment assumes environmental equilibrium between the annual dioxin air concentrations estimated by Connell Wagner (CW 2008a). It takes advantage of the relative contribution of inhalational intake to total intake determined by detailed overseas studies, and using the upper bound background intake for Australians calculates an overall intake. The method is conservative such that there is a high level of confidence that if the total

dioxin intake is less than the TDI the risk associated with dioxin emissions from the power station is very low.

The screening assessment via intake calculations requires the following information:

- A health based intake guide. This is the TDI (see Appendix).
- Determination of the relative contribution to total intake by inhalation exposure (Section 7.4.2).
- Estimation of background intake by Australians (Section 7.4.1).

7.3 Comparison with existing air levels

The maximum annual average ground level concentration within the modelling domain (i.e. highest annual average concentration likely) was provided to Toxikos as 0.00000000097 µg TEQ/m^3 or 0.00097 pg TEQ/m^3 (CW 2008h).

As far as Toxikos has been able to ascertain, an ambient air guideline level for dioxins/furans has not been declared in Australia, nor by the European Commission or its member states (EC 1999), the US EPA or Environment Canada. However a Japanese air quality standard of 0.6 pg TEQ/m^3 has been established and several US States (ATSDR 1998) also have set ambient air guideline values for dioxins. As an annual average, these are reported to range between 0.023 to 35 pg/m^3 (ATSDR 1998).

Information on relative concentrations of dioxin like substances in air is provided in Table 7.1. The highest annual average ground level concentration predicted to occur as a result of the power station is 0.00097 pg TEQ/m^3 (CW 2008d). In Europe, a background concentration of 0.1 pg TEQ/m^3 is assumed but certain industrial and urban areas, as well as areas close to major sources, may have up to 20 times higher air concentrations (WHO 2000). In Japan, atmospheric concentrations of 0.55 pg TEQ/m^3 for dioxins/furans plus PCBs have been measured and used for assessing risk (EA/MoHW 1999). Concentrations at Griffith University are approximately 0.009 – 0.017[13] pg TEQ/m^3 (Muller et al 1998).

[13] These are calculated as NATO toxic equivalents, the difference between the NATO and WHO_{98} TEFs used for the power station emission is insignificant in the context of this information.

Table 7.1: Relative concentrations (pg TEQ/m^3) of dioxin like substances in air.

Location	Concentration	Reference
Maximum air concentration predicted for power station	0.00097 [a]	From CW (2008h).
Wattleup [c]	0.016	Gras et al. (2004).
Duncraig [d]	0.057	Gras et al. (2004).
Griffith University, Brisbane	0.009 – 0.017 [b]	Muller et al. (1998).
Urban Brisbane	0.0047 [b]	Muller et al. (1998).
Urban Sydney	0.0016 – 0.062 [b]	Cited in Muller et al. (1998)
Assumed for Europe	0.1	WHO (2000).
Japan	0.55	EA/MoHW (1999).

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

Conclusions:

The information in Table 7.1 shows the estimated air concentrations arising from the power station are at least an order of magnitude less than in Europe, Japan or what has been measured in Australia. Since there are no health effects associated with these concentrations, either from direct or indirect exposures, it follows it will be quite unlikely dioxin emissions from the power station will cause health effects.

7.4 Calculating dioxin intakes

Methodology for assessing risk from dioxins emissions:

The methodology and equations described below assess all the exposure pathways depicted in Figure 3.1 (i.e. direct and indirect secondary pathways) but they do not provide a detailed examination of each of those secondary pathways.

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

- Steady state equilibrium is established between the presumed environmental incremental increase of dioxins resulting from facility emissions and human food, soil and water sources.

- The same proportional relationship between inhalation exposure and total intake is maintained in the new steady state conditions surrounding the power station as exists for background intakes, and exposures to dioxins from incinerator emissions for which there is data.
- Existing background intake of dioxins, from all sources, is conservatively incorporated into the risk assessment by assuming people in the area have current intakes of dioxins equivalent to the upper bound estimate of the general Australian population.

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

The intake of dioxin like substances occurs from a range of sources (food, air, soil) whose contribution to the total intake is not equal; the majority of dioxin intake by humans comes from animal fat (US EPA 2000b, OCS 2004).

The total monthly intake (MI_{TOTAL}) of dioxins is mathematically represented thus:

$$(MI_{TOTAL}) = MBI + MI_{INHAL} + MI_{FOOD} + MI_{SOIL} + MI_{WATER} \quad \textbf{Equation 7.1}$$

Where MI_{TOTAL} = Total Monthly Intake

MBI = Monthly Background Intake from all sources (Section 7.4.1).

MI_{INHAL} = Monthly Intake from direct inhalation (to be calculated Section 7.4.3).

MI_{FOOD} = Monthly Intake from food due to incremental increase in air concentration from emissions (to be calculated Section 7.4.3).

MI_{SOIL} = Monthly Intake from soil due to incremental increase in air concentration from emissions (to be calculated Section 7.4.3).

MI_{WATER} = Monthly Intake from water due to incremental increase in air concentration from emissions (to be calculated Section 7.4.3).

7.4.1 Background intake of dioxin-like substances

A pivotal aspect of the screening risk assessment for exposure pathways for dioxin exposure is the estimation used for background intake of dioxin like substances. The Australian Government has recently published estimates for background intake of dioxin like substances for Australians (OCS 2004). The estimated total background intakes from all sources of exposure for dioxins and furans, polychlorinated biphenyls (PCBs) and total dioxin like substances for Australian adults are presented in Table 7.2. For the purposes of this risk assessment, the upper bound total intake estimates are used.

Table 7.2: Estimated total intakes of Australian adults to dioxin like substances [a]

Total Intake (pg WHO TEQ/kg bw/month)					
Dioxins & Furans		**PCBs**		**Total Dioxin TEQ**	
Lower bound	Upper bound	Lower bound	Upper bound	Lower bound	Upper bound [b]
1.06	10.37	2.83	5.42	3.89	**15.79**

[a] Data taken from OCS 2004 (Table 3-32).
[b] This upper bound intake estimate is used in the risk calculations below.

7.4.2 Relative contribution to intake by inhalation

WHO (2000) has not established an air quality guideline for dioxins because they consider direct inhalation exposures constitute only a small proportion of the total body burden, generally less than 5% of the daily intake is from airborne exposures (more than 95% of human intake of dioxins comes from fatty foods). According to Eduljee and Gair (1996), for exposure conditions typically encountered by the general population, inhalation contributes up to 2% of the total intake of dioxins. Table 7.3 summarises some of the available information regarding the relative contribution of exposure via air to dioxin-like substances. From this information, a relative percentage contribution of 1% by inhalation to the total intake has been used in the screening risk assessment. This is lower than most estimates in Table 7.3 but close to that for Australians. That is, the ratio of intake from inhalation compared to other exposures is 1:99. With respect to the calculations for overall dioxin intake from all pathways in Equation 7.1 (below) a lower proportion assigned to inhalation is more conservative.

Table 7.3: Contribution of exposure via air to total intake of dioxin like substances.

Situation	Contribution by inhalation	Reference
Average daily intake by individuals living near waste incinerator	6.8%	Hattemer-Fry & Travis (1991)
A variety of receptor types living near waste incinerator	1 – 11%	UK (1996)
General UK population	<2%	Eduljee & Gair (1996)
Background intakes in Japan	6.5% 2.1% 2.3%	EA/MoHW (1999). (2001) (2003)
Living near a municipal waste incinerator	9.3% at 50th percentile cumulative risk 6.6% at 75th percentile 3% at 95th percentile	Ma (2002)
Australian adults	0.8% of upper bound 95th percentile estimate.	OCS (2004, Table 3 -34)
American adults	2.5% of middle bound mean intake estimate.	As cited by OCS (2004)

7.4.3 Calculations

The total monthly intake (MI_{TOTAL}) of dioxins is mathematically represented thus:

$$(MI_{TOTAL}) = MBI + MI_{INHAL} + MI_{FOOD} + MI_{SOIL} + MI_{WATER} \quad \textbf{Equation 7.1}$$

Where MI_{TOTAL} = Total Monthly Intake

MBI = Monthly Background Intake from all sources = 15.79 pg/kg bw/d (from Table 7.3).

MI_{INHAL} = Monthly Intake from direct inhalation (Calculate with Equation 7.2).

MI_{FOOD} = Monthly Intake from food due to incremental increase in air concentration from emissions (Calculate with Equation 7.2).

MI_{SOIL} = Monthly Intake from soil due to incremental increase in air concentration from emissions (Calculate with Equation 7.2).

MI_{WATER} = Monthly Intake from water due to incremental increase in air concentration from emissions (Calculate with Equation 7.2).

- The maximum anywhere ground level concentration for dioxin like substances is 0.00097 pg TEQ/m^3, this is the predicted annual average provided by Connell Wagner (2008d). Hence the monthly average intake for an adult via inhalation[14] (MI_{INHAL}) is:

MI_{INHAL} = air dioxin conc x amount air breathed/month……**Equation 7.2**

= [(0.00097 pg TEQ/m^3) x (22m^3/d x 30d)] ÷ 70kg

= 0.0091 pg TEQ/kg /mth (rounded to 0.009)

- Since 1% of dioxin intake by humans is from inhalation and 99% from all other exposures (Section 7.4.2), the incremental increase from pathways other than inhalation is:

$[MI_{FOOD} + MI_{SOIL} + MI_{WATER}] \simeq 99 \times MI_{INHAL}$ ……..…..…**Equation 7.3**

(99 is the proportional ratio of intake from all other media compared to air, see previous text).

Substituting into Equation 7.1

$MI_{TOTAL} = MBI + MI_{INHAL} + [MI_{FOOD} + MI_{SOIL} + MI_{WATER}]$……**Equation 7.1**

$= MBI + MI_{INHAL} + 99[MI_{INHAL}]$

$= MBI + 100[MI_{INHAL}]$ ……..…**Equation 7.4**

= 15.79 pg TEQ/kg /m + 100 [0.009 pg TEQ/kg /m]

= 15.79 pg TEQ/kg /m + 0.9 pg TEQ/kg /m

= 16.69 **pg TEQ/kg /m**

Therefore the total human intake of dioxins, including background, arising from a theoretical long term (30 – 40 years[15]) increase in exposure due to air emissions which gives rise to a supposed incremental increase in environmental burden, is 24% of the Tolerable Monthly Intake[16]. Most of this is associated with the assumed upper bound background intake.

[14] The US EPA (1997) sum the relative proportions of air intake for an adult male while at rest and undertaking a mixture of light, moderate and heavy breathing activities during a day to give a total daily inhalation rate of 21.4 m^3/d. enHealth (2004) of Australia recommend a default inhalation rate for adults of 22 m^3/d, this is the same as the average of males and females suggested by IPCS (1994). Health Canada (1999) recommends 23 m^3/d. For this risk assessment Australian enHealth recommendations are followed and 22 m^3/d used.

[15] 30 – 40 years is the time to reach environmental steady state conditions. Intake will gradually increase during this time but once steady state is reach there will be no further increase in intake nor body burden.

[16] The percentage calculation is (16.69 pgTEQ/kg/m ÷ 70 pgTEQ/kg/m) x 100 = 24%.

It should be noted that because inhalation intake (Equation 7.3) is used as the basis for estimating intake under conditions of steady state, the conservatism in the inhalation exposure estimate is multiplied by a factor of 100 when estimating the steady state intake from food (Equation 7.4).

The increase in dioxin intake by people living around the proposed power station due to direct exposure to air emissions from the power station is very small (less than 0.06% of current upper bound background intakes). The total intake including current upper bound background intakes is 24% of the recommended tolerable intake considered by Australian health authorities to be without adverse health effects. The total intake includes current background intake plus the incremental intake from air emissions (direct and indirect). More than 95% of the estimated total intake is due to current background exposures.

7.5 Conclusions

Estimated maximum annual average air concentrations of dioxins are at least 10 times less than the air concentrations in Europe or Japan, and measured in Australia. These air concentrations are not reported as causing either direct or indirect health effects.

The calculated total intake of dioxins from background exposures plus direct and indirect exposures due to power station emissions is less than the TDI.

It is concluded the risk of adverse health effects from dioxin emissions from the proposed power station is negligible.

8. Secondary exposure pathways

As depicted in Figure 3.1 exposure to emissions from the power station could occur through secondary exposure pathways. The process of evaluating health risks from exposure via secondary pathways is difficult and uncertain because empirical relationships for the movement of most substances from air to other media, which humans may be exposed have not been established. Thus the evaluation of secondary exposure pathways inherently requires many postulates which impart unquantifiable uncertainties to the assessment. Consequently in most risk assessments the secondary exposure pathways are either not considered or unrealistic conjectures are made to establish gross worse case scenarios. In lieu of these approaches a pragmatic alternative is to perform screening assessments to evaluate the need for, and hence

benefit from a detailed multi-pathway risk assessment. Usually the pre-evaluation is qualitative and based on the assessor's experience. In this risk assessment the pre-screens have been structured to provide a more objective and transparent process for deciding whether secondary exposure pathways are required to be evaluated.

The methodology devised by Toxikos for pre-screening the need to conduct a health risk assessment for secondary exposures is explained in Appendix 2. The detailed analysis undertaken for the substances of interest to the power station is also in Appendix 2.

Based on current knowledge substances in the emissions from the power station likely to be of interest with regard to secondary exposure pathways are dioxin like substances (evaluated in Section 7), metals (Cd and Hg) and PAHs.

It is recognised that these substances are likely to be bound to particulates and hence could be deposited directly onto vegetation; the extent to which humans may be exposed via this deposition pathway will be influenced by how much particulate is deposited, the extent to which rain washes particulates off vegetation, the extent of washing fruit and vegetables before consumption, and how much of the fruit and vegetables consumed are sourced from the local area. Assuming the vast majority of vegetables and fruit consumed by individuals will originate from areas outside of the power station emission dispersion zone and most of the time the food will be washed prior to consumption, then exposure via direct deposition will be very small. This premise is supported by comparison of predicted annual ground level air concentrations with background concentrations from remote and rural areas around the world.

Dioxins:

For dioxins, the screening procedure is based upon the proportional relationship between intake via inhalation and other exposure pathways under steady state environment-body burden that has been established by many international studies. In the analysis a conservative estimate of background intake[17] by Australians is factored into the process. The calculated increase over background monthly intake of dioxin like substances from the emissions is very small and the total intake, including background, is less than the tolerable monthly intake recommended by Australian health authorities. It was therefore concluded in Section 7 that the low level of dioxin emissions from the power station do not present a likely human health risk from direct and/or

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

indirect exposures, and that it is not necessary to conduct a detailed analysis of secondary dioxin exposure pathways.

PAHs:

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

Metals:

For metals, the following criteria developed by Toxikos should be met in order that there is a presumption of possible health effects due to secondary food exposure pathways of metals.

1. The available weight of literature evidence must indicate the metal is able to bioaccumulate, or biomagnify, into human food sources, and
2. to trigger detailed evaluation of secondary exposure pathways the predicted incremental increase in annual ground level concentration of the metal must be above those measured in rural and remote areas, or
3. the hazard quotient for direct inhalational exposure of a metal must be more than 0.05.

The first requires a brief review of the readily available scientific information for potential for bioaccumulation/biomagnification in the human food chain. The second is pragmatically grounded in a comparison of predicted receptor ground level concentrations with rural background concentrations that are not associated[18] with significant exposures via secondary pathways. Thirdly the previous two criteria are augmented with a requirement for a significant margin of exposure[19] via inhalation for individual metals such that if exposure was to occur via secondary pathways then there is ample conservatism in the screen to ensure the additional non-inhalation intakes will not result in adverse health effects. This is essentially saying the metal has to be toxic/hazardous to a certain level of potency before there is concern.

[18] This assumption is based on the background concentrations used for the comparison being from rural or remote areas i.e. areas regarded as being unpolluted, where deposition to soil/pasture is not expected or shown to be a problem causing health effects due to exposure by secondary pathways.
[19] The margin of exposure (MoE) is traditionally established from comparison of estimated exposure concentrations with the No Observed Adverse Effect Level (NOAEL) for the compound. However in this risk assessment the MoE is established by comparison with the guideline value (i.e. the hazard quotient). With this latter comparison the safety factors embedded in the guideline are augmented to provide greater safety.

It is concluded in Section A2.3 of Appendix 2 that since the screening criteria for metals were not satisfied, detailed examination of the secondary exposure pathways is not required, and that health effects from secondary exposure pathways for PAH, cadmium and mercury are negligible.

9. Uncertainty discussion

Uncertainties have been discussed within the text of each section as the issue has arisen. The most important areas of uncertainties in this preliminary risk assessment relate to the accuracy, or otherwise of the emission estimations and GLCs provided to Toxikos by Connell Wagner (CW 2008g). Mass emission estimations by Connell Wagner have largely been accomplished using NPI emission factors. Apart from noting that in some instances the average emission factor has been used, which depending on how the engineering of the proposed BPS compares with that of the sources of the emission factors, may either under or over estimate the emissions. Toxikos is not in a position to comment further on the techniques used in calculating the emissions or the dispersion modelling used for determining ground level concentrations.

Another potentially important source of uncertainty is the background data used in calculating the hazard indices. Background air concentrations were provided only for PM, however additional area sources of NO_2 and SO_2 were incorporated into the simulation modelling of the cumulative scenario. The lack of consideration of background sources of other substances is unlikely to impact on the calculations given the significant influence of background PM. The detail of the background particulate sources, the time of year or time of day that they occur is not known to Toxikos. From past experience it is likely that the combination of maximum or 98^{th} percentile GLCs of NO_2 and particulate matter from the power station will coincide on the same days as the maximum or 98^{th} GLCs due to background sources. However while in Toxikos' opinion this is considered to be the likely situation Toxikos is not in a position to definitively demonstrate the same.

It is noted the plant is to be built in a sparsely populated area, hence the probability that a person will be at the same location at the same time the maximum concentration occurs is low. Nevertheless contour plots of the GLCs used in the risk assessment are not available to Toxikos hence it is not known with certainty that the potentially impacted population is very small.

Not withstanding the above comments, the fact that the HRA is returning higher than ideal hazard indexes is not an indication that health effects are necessarily likely to occur if there is exposure to the emissions.

References

Information supplied by Connell Wagner

CW (2008a). Air quality PER extract of EIS

CW (2008b). Letter from Dr Neil Mackenzie, Connell Wagner to Roger Drew regarding "Biomass Power Station, Manjimup, Western Australia", dated 12 February 2008.

CW (2008c). Email from Ashok Kaniyal, Connell Wagner to Roger Drew "Re: Health Risk Assessment" for calculation of cadmium and mercury GLCs, dated 14 February 2008.

CW (2008d). Email from Ashok Kaniyal, Connell Wagner to Roger Drew "Re: Health Risk Assessment" for calculation of dioxin TEQ and PAH GLCs, dated 14 February 2008.

CW (2008e). Email from Ashok Kaniyal, Connell Wagner to Roger Drew "Re: data needed" for calculation of annual PM GLCs from BPS, dated 22 February 2008.

CW (2008f). Email from Ashok Kaniyal, Connell Wagner to Roger Drew "Re: data needed" for calculation of background annual PM GLCs, dated 22 February 2008.

CW (2008g). Letter from Dr Neil Mackenzie, Connell Wagner. Subject "Biomass Power Station – GLC Qualification for Other Pollutants", dated 13 March 2008.

CW (2008h). Email from Ashok Kaniyal, Connell Wagner to Roger Drew "Re: BPS Risk Assessment" updated modelling data for NO_2, SO_2, dioxins, and 1 hr metals, dated 8th April 2008.

CW (2008i). Email from Ashok Kaniyal, Connell Wagner to Roger Drew "Re: BPS Risk Assessment Urgent" Points of clarification in data supplied by email, dated 9th April 2008.

Information cited from the scientific literature

ATSDR (1992). Public health assessment guidance manual. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

ATSDR (1995). Toxicological profile for polycyclic aromatic hydrocarbons (PAHs) (Update). U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. (PB/90/258245/AS).

ATSDR (1998). Toxicological profile for chlorinated dibenzo-p-dioxins (CDDs). U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. (Update) (PB/99/121998).

ATSDR. (2001). Guidance manual for the assessment of joint toxic action of chemical mixtures. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Feb 2001.

Cogliano, V.J. (1997). Plausible upper bounds: Are their sums plausible? Risk Analysis. 17: 77-84.

Denier van der Gon et al. (2006). The contribution of sea salt aerosol to PM_{10} in the Netherlands and a methodology to correct the annual PM_{10} concentration and number of PM_{10} exceedance days for sea salt aerosol. Geophysical Research Abstracts 8: 03978.

EA/MoHW (1999). Report on tolerable daily intake (TDI) of dioxins and related compounds (Japan). Environmental Health Committee of the Central Environment Council, Environment Agency. Living Environment Council and Food Sanitation Investigation Council, Ministry of Health and Welfare, Government of Japan.

EA/MoHW (2001). Dioxins. Ministry of the Environment, Office of Dioxins Control, Administration Division, Environment Management Bureau, Government of Japan.

EA/MoHW (2003). Dioxins. Office of Dioxins Control, Environment Management Bureau, Ministry of the Environment, Government of Japan.

EC (1999). Compilation of EU dioxin exposure and health data. Task 1- Member State Legislation and Programmes. Prepared by Petersen, A. for European Commission DG Environment and UK Department of the Environment, Transport and the Regions. October 1999.

Eduljee, G. H. and Gair, A. J. (1996). Validation of a methodology for modelling PCDD and PCDF intake via the foodchain. Sci. Total Environ. 187: 211-229.

enHealth (2004). Environmental Health Risk Assessment. Guidelines for assessing human health risks from environmental hazards. Department of Health and Ageing, and enHealth Council, Commonwealth of Australia.
http://enhealth.nphp.gov.au/council/pubs/pdf/envhazards.pdf

Fox, M.A., Tran, N.L., Groopman, J.D. and Burke, T.A. (2004). Toxicological resources for cumulative risk: an example with hazardous air pollutants. Reg. Toxicol. Pharmacol. 40: 305-311.

Gras, J., Müller J., Graham, B., Symons, R., Carras, J. and Cook, G. (2004). Dioxins in ambient air in Australia, National Dioxins Program Technical Report No. 4. Department of the Environment and Heritage, Commonwealth of Australia.

Hattemer-Frey, H. and Travis,C.C. (1991). An overview of food chain impacts from municipal waste combustion. In Municipal Waste Incineration Risk Assessment, Editor C.C. Travis. Plenum Press, New York. As cited in UK 1996.

Health Canada (1999). Human health risk assessment for priority substances. Canadian Environmental Protection Act. Cat. No. En40-215/41E.

IPCS (1994). Environmental Health Criteria 170. Assessing human health risks of chemicals: Derivation of guidance values for health-based exposure limits. International Programme on Chemical Safety, World Health Organisation, Geneva.

Ma, H-W. (2002). Using Stochastic Risk Assessment in setting Information Priorities for managing Dioxin Impact from a Municipal Waste Incinerator. Chemosphere. 48: 1035-1040.

Morello-Frosch, R.A., Woodruff, T.J., Axerad, D.A. and Caldwell, J.C. (2000). Air toxics and health risks in California: The public implications of outdoor concentrations. Risk Analysis. 20: 273-291.

Muller, J.F., McLachlan, M.S., Hawker, D.H. and Connell, D.W. (1998). Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans in the atmospheric environment of Brisbane, Australia. Clean Air. 32: 27-31.

NHC (2005). A Guide to Health Impact Assessment: A Policy Tool for New Zealand. Second edition. Public Health Advisory Committee, National Health Council of New Zealand. June 2005. Available at http://www.phac.health.govt.nz/moh.nsf/pagescm/764/$File/guidetohia.pdf

NHMRC (2002). Dioxins: Recommendation for a tolerable monthly intake for Australians. National Health and Medical Research Council, Commonwealth of Australia.

NHMRC (2006). Ambient Air Quality Standards Setting: An approach to health-based hazard assessment. National Health and Medical Reseach Council and Environmental Health Council (enHealth), Australian Government, Canberra. September 2006.

NZ MfE (1997). Chapter 5 Soil Acceptance Criteria, In “Health and Environmental Guidelines for Selected Timber Treatment Chemicals”. New Zealand Ministry for the Environment. Publication Reference: ME240. http://www.mfe.govt.nz/publications/hazardous/timber-guide-jun97/.

NZ MfE (1999). Guideline for Assessing and Managing Petroleum Hydrocarbon Contaminated Sites in New Zealand. User’s Guide. New Zealand Ministry for the Environment. August 1999. ISBN 0478 09065 X.

NZ MoH (2000). Drinking Water Standards for New Zealand. New Zealand Ministry of Health, ISBN 0-478-23904.

OCS (2004). Human health risk assessment of dioxins in Australia, National Dioxins Program Technical Report No. 12. Office of Chemical Safety, Australian Government Department of Health and Ageing, Australian Government Department of the Environment and Heritage, Commonwealth of Australia.

Pratt, G.C., Palmer, K., Wu, C.Y., Oliaei, F., Hollerbach, C. and Fenske, M.J. (2000). An Assessment of Air Toxics in Minnesota. Environ. Health Perspect. 108: 815-825.

Tam, B.N. and Neumann, C.M. (2004). A human health assessment of hazardous air pollutants in Portland, OR. J. Environ. Management 73: 131-145.

Toronto MoE (1997) Scientific Criteria Document for Multimedia Standards Development. Polycyclic Aromatic Hydrocarbons (PAH). Part1: Hazard Identification and Dose-Response Assessment. Ministry of the Environment, Toronto, Ontario cited in ToxProbe (2002). Potential For Occupational and Environmental Exposure to Ten Carcinogens in Toronto. Appendix B. Benzo[a]pyrene and other polycyclic aromatic hydrocarbons. Report prepared for Toronto Public Health.

UK (1996). Risk assessment of dioxin releases from municipal waste incineration processes. Her Majesty’s Inspectorate of Pollution. Department of the Environment, HIMP – Commissioned Research. DOE Reference: HMIP/CPR2/41/1/181.

US EPA (1989). Risk assessment guidance for Superfund. Volume I: Human health evaluation manual (Part A). Washington, DC: U.S. Environmental Protection Agency, Office of Emergency and Remedial Response. EPA/540/1-89/001.

US EPA (1997). Exposure factors handbook. Volume 1: General factors. EPA/600/8-89/043, May 1989. Update to Exposure Factors Handbook. EPA/600/P-95/002Fa, August 1997. Office of Research and Development, National Center for Environmental Assessment, U.S. Environmental Protection Agency.

US EPA (2000a). Supplementary guidance for conducting health risk assessment of chemical mixtures. Risk Assessment Forum, U.S. Environmental Protection Agency. EPA/630/R-00/002.

US EPA (2000b). Exposure and health assessment for 2,3,7,8 – Tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. National Centre for Environmental Assessment Office of Research and Development, U.S. Environmental Protection Agency.

WA DoH (2006). Health Risk Assessment in Western Australia, Department of Health, Western Australia.

WHO (1948). Preamble to the Constitution of the World Health Organization as adopted by the International Health Conference, New York, 19-22 June, 1946; signed on 22 July 1946 by the representatives of 61 States (Official Records of the World Health Organization, no. 2, p. 100) and entered into force on 7 April 1948.

WHO (1998a). Environmental Health Criteria 202. Selected Non-Heterocyclic Polycyclic Aromatic Hydrocarbons. International Programme on Chemical Safety, World Health Organization, Geneva.

WHO (1998b). Assessment of the Health Risks of Dioxins: Re-evaluation of the Tolerable Daily Intake (TDI). Executive summary of the WHO Consultation, May 25 – 29, Geneva, Switzerland.

WHO (2000). Air Quality Guidelines for Europe 2nd Edition. WHO Regional Publications, European Series 91. World Health Organisation. Regional Office for Europe, Copenhagen. http://www.euro.who.int/air/activities/20050223_4

Appendix 1: Health effects and guideline values of emission constituents

The toxicity category table (Section A1.1) identifies whether an emission constituent has been evaluated by a relevant competent authority for genotoxic, carcinogenic or reproductive toxicity potential. It also details the major target organ for which critical effects have been observed. This information can help in deciding whether there may be biological legitimacy for assuming additivity of effects between components. In strict biological terms additivity would only be expected if emission components were affecting the same organ systems.

Sections A1.1, A1.2 and A1.3 provide a brief summary of toxicity guideline values for each emission component detailing the critical effect, the no observed effect concentration or lowest observed effect concentration and the uncertainty factors applied by the authority to create the toxicity guideline value.

There are four substances that are considered genotoxic carcinogens and seven reproductive toxicants. The respiratory system is a common target for many of the substances.

Substances were placed into toxicity categories by consulting the following sources:

1. IARC Monographs and Supplements on the Evaluation of Carcinogenic Risks to Humans.
2. Environmental Health Criteria Monograph Series from the International Programme on Chemical Safety, World Health Organization.
3. Toxicological Profiles for Chemical Substances, Agency for Toxic substances and Disease Registry (ATSDR), US Department of Health and Human Services.
4. Office of Environmental Human Hazard Assessment (OEHHA), Californian EPA.
5. Re-evaluation of Human Toxicological Maximum Permissible Risk Levels, Dutch National Institute of Public Health and the Environment (RIVM 2001).
6. WHO Guidelines for Air Quality (2000a); Air Quality Guidelines for Europe, 2nd Edition (2000b), World Health Organization.
7. EU Directive Dangerous Substances and Preparations, Annex 1 26th Adaption European Commission (2000). Directive 67\548|EEC.
8. WHO (2000c). Safety Evaluation of Certain Food Additives and Contaminants Simple Aliphatic and Aromatic Sulfides and Thiols. WHO Food Additives Series: 44 Prepared by the Fifty-Third Meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA) World Health Organization, Geneva
9. Patty's Industrial hygiene and toxicology / George D. Clayton and Florence E. Clayton, editors. Electronic edition.
10. Dictionary of Substances and their Effects. Editor, M.L. Richardson; Cambridge, England: Royal Society of Chemistry. Electronic edition.

A1.1: Toxicity category summary

Substance	Target Organ of Concern	Toxicity Category		
		Genotoxic	Carcinogenic[a]	Reproductive Toxicant
Criteria Pollutants				
Carbon monoxide (CO)	Blood & cardiovascular system.	N	N	**Y**
PM_{10}	Respiratory system effects, decreased pulmonary function in responding subpopulations. Cardiovascular system.	N	N	N
$PM_{2.5}$	Respiratory system effects, decreased pulmonary function in sensitive subpopulations. Cardiovascular system.	N	N	N
Nitrogen dioxide (NO_2)	Respiratory system, decreased pulmonary function in asthmatics.	N	N	N
Sulphur dioxide (SO_2)	Respiratory system irritation and decreased pulmonary function in responding subpopulations of exercising asthmatics.	N	N	N
Metals				
Arsenic (As)	Respiratory; skin; reproductive/developmental; cardiovascular; nervous system; lung cancer.	**Y**	**Y** (IARC 1, USEPA A)	N
Cadmium (Cd)	Respiratory system and kidney. Lung cancer.	**Y**	**Y** (IARC 1, USEPA B1)	**Y**
Chromium (Cr^{III})	Essential element.	N	N	N
Chromium (Cr^{VI})	Respiratory system. Lung cancer.	**Y**	**Y** (IARC 1, USEPA A)	N
Copper (Cu)	Respiratory system irritation.	N	N (USEPA D)	N
Lead (Pb)	Associated with impaired neurobehavioural functioning and IQ in children.	N	**Y**(IARC 2B, USEPA B2)	**Y**
Manganese (Mn)	Nervous system; lungs; reproductive system.	N	N (USEPA D)	**Y**
Mercury (Hg)	Central nervous system and kidneys.	N	N	**Y**
Nickel (Ni)	Lung and nasal tumours.	**Y**	**Y** (IARC 1, USEPA A)	N

Substance	Target Organ of Concern	Toxicity Category		
		Genotoxic	Carcinogenic[a]	Reproductive Toxicant
Selenium (Se)	Clinical selenosis. Characteristic "garlic odour" thickened brittle nails, hair and nail loss, lowered haemoglobin levels, mottled teeth, skin lesions and possible CNS abnormalities with severe intoxication.	N	N	N
Dioxins and Furans	Liver, reproductive, developmental, endocrine, respiratory, hematopoietic effects.	N	**Y** (IARC 1)	**Y**
Polycyclic aromatic hydrocarbons	Respiratory system cancer.	**Y**	**Y** (IARC 2B, US EPA B2)	N
Total Volatile Organic Compounds TVOC	Sensory responses such as odour detection, eye and nose irritation.	N[b]	N[b]	N[b]
Benzene	Reproductive/developmental; immune system; haematologic system; CNS depression effects.	**Y**	**Y** (IARC 1, USEPA A)	**Y**
Ethylbenzene	Increased liver weight.	N	N (IARC 2B, US EPA D)	N
Phenol	Eye and respiratory irritation; alimentary system; cardiovascular system; kidney; nervous system.	N	N (IARC 3, USEPA D)	N
Styrene	Central nervous system (narcosis).	N	N (IARC 2B)	N
Toluene	CNS depression effects, eyes and respiratory system.	N	N (IARC 3, USEPA D)	N
Xylene	CNS depression effects; eyes and respiratory system.	N	N (IARC 3, USEPA D)	**Y**
Aldehydes				
Acetaldehyde	Irritant to URT.	**Y**	**Y** (IARC 2B, USEPA B2)	N
Benzaldehyde	Irritant to URT.	N	Not evaluated	N
Crontonaldehyde	Irritant to URT.	N	N (IARC 3, USEPA C)	N
Formaldehyde	Irritant to eyes and URT.	**Y**(weak)[c]	**Y** (IARC 2A, USEPA B1)	N
Isobutyraldehyde	Irritant to URT.	N	Not evaluated	N
Propionaldehyde	Irritant to nose and URT.	N	Not evaluated	N

Substance	Target Organ of Concern	Toxicity Category		
		Genotoxic	Carcinogenic[a]	Reproductive Toxicant
Tolualdehyde	Irritant to URT.	N	Not evaluated	N

[a] For the purposes of this report category 3 carcinogens have been designated with a “N” for carcinogenicity. A Category 3 classification does not mean that a substance is not carcinogenic, onlthe information available is insufficient for classification.

[b] Refer also to section A1.6. Individual volatile organic carbon compounds may be associated with chronic health effects for instance benzene is a genotoxic carcinogen.

[c] A weight of evidence assessment indicates that formaldehyde may be weakly genotoxic at the site of contact (i.e. nasal mucosa) (NICNAS 2006).

Carcinogen classifications of IARC and US EPA

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

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

A1.2: Summary acute health effects guidelines

Compound	Health endpoint for guideline value	Critical effect level [mg/m^3] [b]	UF [c]	Air Guideline Value [µg/m^3] [a]	Averaging time for guideline value	Source
Acetaldehyde	Sensory irritation in humans	45 (NOAEL)	20	2,000	24 hours	WHO (2000a)
Arsenic (As)	Developmental toxicity	0.19 (LOAEL)	1,000	0.19	4 hours	OEHHA (1999a)
Benzaldehyde	Respiratory irritation	9 (TWA OEL)	10	900 [f]	1 hour	ACGIH (2006)
Benzene	Reproductive / developmental toxicity	130 (NOAEL)	100	1,300	6 hours	OEHHA (1999b)
Cadmium (Cd)	Effects of concern are associated with long term exposure. No acute health guideline located.					
Carbon monoxide	COHb formation & cardiovascular function	Critical level of COHb < 2.5%	Not applic able	11,000	8 hours	NEPC (1998)
Chromium (Cr)	Effects of concern are associated with long term exposure. No acute health guideline located.					
Copper (Cu)	Based on prevention of metal fume fever	1 (NOAEL)	10	100	1 hour	OEHHA (1999c)
Crotonaldehyde	Respiratory irritation	0.86 (OEL ceiling value)	10	86 [f]	1 hour	ACGIH (2006)
Dioxins	Effects of concern are associated with long term exposure. No acute health guideline located.					
Ethyl benzene	Auditory threshold deterioration	1,302 (HEC converted from a NOAEL)	30	43,400	Acute MRL (<14 days) [d]	ATSDR (2007a)
Formaldehyde	Nose, throat irritation in humans	0.1 (NOAEL)	1	100	30 mins	WHO (2000a)
Isobutyraldehyde	Respiratory irritation	74 (TWA OEL)	10	7,400 [f]	1 hour	ACGIH (2006)
Lead (Pb)	Effects of concern are associated with long term exposure. No acute health guideline located.					
Manganese (Mn)	Effects of concern are associated with long term exposure. No acute health guideline located.					
Mercury (Hg)	Developmental toxicity (behavioural deficits in foetus)	1.8	1,000	1.8	1 hour	OEHHA (1999d)

Compound	Health endpoint for guideline value	Critical effect level [mg/m^3] [b]	UF [c]	Air Guideline Value [µg/m^3] [a]	Averaging time for guideline value	Source
Nickel (Ni)	Slight changes in lung function in asthmatics	0.033 (LOAEL)	6	6	1 hour	OEHHA (1999e)
NO_2	Exacerbation of asthma & increased responsiveness to environmental bronchoconstrictors in asthmatics	LOEL ~376 – 565 (~200 – 300 ppb)[e] For 20' -4h exposures	2	226 (120 ppb)[e]	1 hour	NEPC (1998)
Phenol	Eye, nose and throat irritation in humans	20 (NOAEL)	10	5,800	1 hour	OEHHA (1999f)
PM_{10}	Aggravation of existing respiratory & cardiovascular diseases			50	24 hour	NEPC (1998)
$PM_{2.5}$	Aggravation of existing respiratory & cardiovascular diseases			25	24 hour	NEPC (2003)
Polycyclic aromatic hydrocarbons (PAH)	Effects of concern are associated with long term exposure. No acute health guideline located.					
Propionaldehyde	Nasal irritation	48 (TWA OEL)	10	4,800 [f]	1 hour	ACGIH (2006)
Selenium (Se)	Effects of concern are associated with long term exposure. No acute health guideline located.					
SO_2	Changes in lung function &/or exacerbation of respiratory symptoms in responding subpopulations of exercising asthmatics.	0.524 (NOAEL) (200 ppb)[e]	2	262 (100 ppb)[e] 456 (175 ppb)[e] 524 (200 ppb)[e] 210 (80 ppb)[e]	15 min 10 min 1 hour 24 hour	UK DoE (2000). WHO (2000a) NEPC (1998)
Styrene	Eye and throat irritation in humans	210 (NOAEL)	10	21,000	1 hour	OEHHA (1999g)
o-Tolualdehyde [g]	Respiratory irritation	0.86 (OEL ceiling value)	10	86 [f]	1 hour	ACGIH (2006)
p-Tolualdehyde [g]	Respiratory irritation	0.86 (OEL ceiling value)	10	86 [f]	1 hour	ACGIH (2006)
Toluene	N/A	N/A	N/A	15,070	6 hours	NEPC (2004a)

Compound	Health endpoint for guideline value	Critical effect level [mg/m^3] [b]	UF [c]	Air Guideline Value [µg/m^3] [a]	Averaging time for guideline value	Source
o-Xylene	N/A	N/A	N/A	4,340 [h]	30 minutes	NEPC (2004a)

[a] Because Australia has very few guidelines for acute exposures, guidelines have been sourced from a variety of competent authorities where documentation is available for subsequent verification of the guideline if required.

[b] NOAEL = No Observed Effect Adverse Level, LOAEL = Low Observed Effect Adverse Level

[c] UF = Uncertainty factor applied to the critical effect level (i.e. NOAEL or LOAEL).

[d] Acute MRL = minimal risk level with an exposure duration of less than 14 days. MRLs are intended to serve as a screening tool to help public health professionals decide where to look more closely. When an acute MRL from the ATSDR has been used as the guideline a one hour averaging time has been conservatively assigned to the value for easy comparison against the modelled 1 hour ground level concentrations.

[e] Conversions from ppb into µg/m^3 performed at 25^{0}C and 101.7pa.

[f] A health based ambient air guideline value was not available from any of the following sources: Ambient Air NEPM, Californian OEHHA , US EPA IRIS, WHO Air Quality Guidelines (2000a,b), ATSDR Toxicological Profiles and Dutch RIVM (2001). A value was therefore derived from an occupational exposure limit (OEL); the OEL listed by the ACGIH (2006) is based on prevention of upper respiratory tract irritation. Since irritation is usually a relatively short biological response it is independent of exposure times over about 0.5 to 8 hours and therefore does not follow Haber's Rule. For this risk assessment it has been assumed the OEL protection level against respiratory tract irritation is independent of exposure time, the OEL value has therefore been applied to the 1 hour modelled ground level concentrations for calculation of hazard indices. There has however been an adjustment of 10 times to account for possible increased sensitivity in the general population compared to a workforce.

[g] An acute guideline value was not located for tolualdehyde, it was therefore assumed that this substance may have health effects (sensory irritation) equivalent to the most potent of the other aldehydes. As judged from the AGVs, crotonaldehyde is the most potent as it has the lowest AGV.

[h] The guideline value applies to all isomers of xylene.

A1.3: Summary chronic health effect guidelines

Compound	Health endpoint	Critical effect level [mg/m^3] [a]	UF [b]	AGV [c] [µg/m^3] [d]	Averaging time for guideline value	Source
Arsenic (As)	Developmental toxicity in mice	0.03 (LOAEL of 200 converted to a HEC[f] of 0.03)	1000	0.03	NS[e]	OEHHA (2001a)
Benzene	Haematopoietic, development, nervous system	0.60 (NOAEL of 1.59 for workers (8hr exposure) converted to a HEC[f] of 0.60)	10	60	NS[e]	OEHHA (2000a)
Cadmium (Cd)	Renal effects in a population	N/A	N/A	0.005	1 yr	WHO (2000a)
Carbon monoxide	Effects are related to COHb formation from short term exposure. Chronic guideline not located.					
Chromium Cr^{III}	Respiratory system	0.6 (NOAEC) for metallic chromium	10	60 [g]	NS[e]	RIVM (2001)
Cr^{IV}	Lower respiratory effects	0.034 (BMD adjusted for continuous exposure)	300	0.1 [j]	NS [e]	US EPA IRIS (1998)
Copper (Cu)	Respiratory and immunological effects.	0.6 (NOAEC)	600	1	NS[e]	RIVM (2001)
Dioxins/ Furans	Refer to text (Section 7)					
Ethyl benzene	Nephropathy in female rats	325 (HEC[f] converted from LOAEL)	300	1,300	NS[e]	ATSDR (2007a)
Lead (Pb)	Neurotoxicity	Critical level of Pb in the blood is 100 µg Pb/L	N/A	0.5	1 yr	NEPC (1998) WHO (2000a)
Manganese (Mn)	Neurotoxic effects in workers	0.03 (NOAEL)	200	0.15	1 yr	WHO (2000a)
Mercury - inorganic (Hg)	Renal toxicity in workers	0.02 (LOAEL)	20	1	1 yr	WHO (2000a)
Nickel (Ni)	Respiratory system, haematopoietic	0.0016 (HEC[f] converted from NOAEL)	30	0.05	NS[e]	OEHHA (2000b)
NO_2	Development of recurrent upper and lower respiratory symptoms in children.	LOAEL 75-150 (40 - 80 ppb)[d]	2 on mid LOAEL	58 (30 ppb) [d]	1 yr	NEPC (1998)

Compound	Health endpoint	Critical effect level [mg/m^3] [a]	UF [b]	AGV [c] [µg/m^3] [d]	Averaging time for guideline value	Source
Polyaromatic Hydrocarbons (PAHs)	Refer to text (Section A1.6)					
PM_{10}	Aggravation of existing respiratory diseases (increased use of bronchodilators among asthmatics)	Estimated (based on linear extrapolation of exposure response curves from epidemiologic-al findings).	-	20	1 yr	EC (2004)
$PM_{2.5}$	As for PM_{10}			8	1 yr	NEPC (2003)
Phenol	Neurological effects	20 (NOAEL)	100	200	NS[e]	OEHHA (2000c)
Selenium	Clinical selenosis (liver, blood, skin and CNS)	0.015 mg/kg/day (NOAEL)	3	20 [h]	NS[e]	OEHHA (2001b)
SO_2	Community patterns of respiratory illness measured by prevalence of respiratory symptoms.	100 (LOAEL in combination with particulates).	2	52 (20 ppb)[d]	1 yr	NEPC (1998)
Styrene	Neurological effects	85 (LOAEL) (20 ppm)	100	850	NS[e]	ATSDR (2007b)
Toluene	Neurological effects in humans	30 (Adjusted for continuous exposure from a LOAEL of 130)	100	300	NS[e]	ATSDR (2000)
o-Xylene	Respiratory and neurological effects in workers exposed to 70% xylene	60 (geometric mean LOAEL)	300	220 [i]	NS[e]	ATSDR (2007c)

[a] NOAEL = No Observed Adverse Effect Level, LOAEL = Lowest Observed Adverse Effect Level

[b] UF = Uncertainty factor applied to the critical effect level (i.e. NOAEL or LOAEL).

[c] AGV is the air guideline value in units of µg/m^3

[d] Conversions from ppb into µg/m^3 performed at 25^{0}C and 101.7pa.

[e] The averaging time is not specified (NS) but is a chronic duration GV, hence the averaging time = 1 yr.

[f] HEC = Human Effect Concentration derived by converting the animal effect conc. with physiological scaling data..

[g] The guideline value is for metallic and insoluble Chromium ^{III}.

[h] The inhalation reference exposure level for selenium was calculated by the OEHHA by route to route extrapolation of the oral exposure level to an inhalation concentration.

[i] The guideline value applies to all isomers of xylene.

[j] Guideline value for Cr^{IV} as particulates.

N/A = Not Available.

A1.4: Derivation of guideline values for criteria pollutants

Carbon Monoxide:

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

Nitrogen Dioxide:

Key findings:

- Concentrations of around 2,000 µg/m^3 (~1,000ppb) are needed to affect respiration of healthy people.
- The low effect level for increased bronchial reactivity in sensitive asthmatics is 375-575 µg/m^3 (~200-300 ppb) for exposures from 20 minutes up to 4 hours.
- The no effect level for increased bronchial reactivity is ~200 µg/m^3 (100 ppb).
- Lowest observed adverse effect level for recurrent upper and lower respiratory tract symptoms are reported following chronic exposures between 75-150 µg/m^3 (40-80 ppb)
- The increased bronchial reactivity may remain for up to 10 hours after cessation of NO_2 exposure.

Only very high concentrations of NO_2 (approximately 2,000 µg/m^3 (~1,050 ppb)) affect breathing in healthy people[20]. However small changes in lung function (< 5%) and changes in airway responsiveness have been reported in several studies of sensitive asthmatics or the elderly exposed to concentrations as low as 375-575 µg/m^3 (~200-300 ppb) over 20 minutes to 4h (Bauer et al., 1986; Bylin et al., 1988; Roger et al., 1985; Morrow et al., 1992; Strand et al., 1996, 1997, Streeton 1997). These levels represent a clear low-observed-effect level (LOEL) for NO_2 based on increased responsiveness in mild asthmatics to bronchoconstrictors or in subjects with chronic obstructive pulmonary disease (COPD). The study by Bauer et al (1986) did not find a significant change in pulmonary function when asthmatics were exposed to 560 µg/m^3 NO_2 when resting, with decreases recorded only after the subjects exercised. Similarly, testing asthmatics the day after exposure to 490 µg/m^3 NO_2 did not decrease lung function before allergen challenge (Strand et al., 1997).

The identification of an obvious no effect level is less clear but it seems to be around 200 µg/m^3 (approx 100 ppb). Studies have shown that effects can be detected in mild asthmatics after short-term exposure to 488-500 µg/m^3 (260-240 ppb) NO_2 who are subsequently exposed to an inhalation challenge (Strand et al., 1996, 1997; Kraft et al., 2005). However, in a study where mild asthmatic subjects were exposed for 1h to 200 µg/m^3 (~100 ppb) NO_2 and then immediately exposed to a house dust mite challenge, the late asthmatic response (as tested

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

using forced expiratory volume in one second; FEV_1) was found to be greater than when compared to air (NO_2 -7.76% vs. Air -2.85%), but the results were not found to be significant (Tunnicliffe et al., 1994). The current air guideline for acute exposure to NO_2 in the NEPM is 0.12 ppm (226 µg/m^3) measured as a 1h average.

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

Based upon a review of the literature, Streeton (1997) considered short-term ambient exposures to 200-300 ppb (375-565 µg/m^3) NO_2 and chronic exposures between 40-80 ppb (75-150 µg/m^3) capable of causing recurrent upper and lower respiratory tract symptoms, an increased incidence of respiratory infection and onset of symptoms in mild asthmatics. Streeton considered these effects as a low observed adverse effect levels (LOAEL) and has suggested that an uncertainty factor of 2 need apply to account for susceptible people within the population therefore establishing a short-term guideline in the range 100-150 ppb as a 1h average and a chronic guideline between 20-40 ppb for longer term exposures as an annual average (Streeton, 1997). The Australian ambient air guideline has been established as 58 µg/m^3 (30 ppb) (NEPC 1998).

The WHO (1997, 2000b) took a different approach to reach a similar conclusion. Similar to Streeton, the WHO noted the epidemiological studies suggesting human health effects associated with long-term NO_2 exposures however the WHO (1997) state this is supported by animal toxicological findings showing increased susceptibility to respiratory infections and impairment of host defences as a result of subchronic or chronic exposures to NO_2 concentrations near ambient concentrations (i.e. 20-60 µg/m^3; 11-32 ppb). On the basis of a background level of 15 µg/m^3 (8 ppb) as determined in Finland during the 1980s (Jaakkola et al., 1991) and the fact that significant adverse health effects occur with an additional concentration of 28.2 µg/m^3 (15 ppb) or more, which is an estimate of an increased risk of about 20% for respiratory symptoms and disease (Hasselblad et al., 1992; WHO, 1997), an annual guideline value of 40 µg/m^3 (22 ppb) was derived by the WHO (1997). The WHO considers the guideline value will be protective of most serious effects. The fact that a no-effect level for

subchronic or chronic NO_2 exposure concentrations has not yet been determined was emphasised.

Particulate matter (PM_{10}):

Key findings

- There is general agreement in the scientific literature that there is a concentration-response relationship (with no indication of a threshold) between PM_{10}/$PM_{2.5}$ and various measures of population based health effects.
- The exact form of the relationship is unclear, depending upon the health measure some studies indicate the relationship to be linear while others suggest non-linearity (RIVM 2002).
- It is noted that people with compromised respiratory or cardiopulmonary function (either through disease or old age) are more susceptible to the effects of particulates.
- Ambient air quality standards around the world are based primarily on data obtained from large urban populations where background incidences of the 'effects' are measurably increased during episodes of high ambient PM_{10}

There is compelling evidence that small airborne particulates can cause/exacerbate heath conditions in vulnerable sectors of the population. The detailed toxicological mechanism(s) by which particulate matter causes adverse health effects is not known. Most of the data indicate a role for oxidative stress causing inflammation and immunotoxicity in airways and lungs, or a mechanism involving impairment of respiratory and cardiac neurological functions. According to a publication from the Dutch Institute of Public Health and Environment [21], rather than assigning the observed association between particulate matter and adverse health effects to the toxicity of particulate matter itself, a more plausible explanation is that the association is the result of reduced capacity of an individual to withstand stress and maintain a stable, relatively constant internal environment. It is therefore plausible, and indeed consistent with observations, that the population at risk is largely defined in particular by individuals with failing health, attributable to ageing or illness (RIVM 2002).

Populations shown to be susceptible to the effects of airborne particles are primarily those with compromised health, especially respiratory and/or cardiopulmonary function. 'At risk' groups include the elderly, people with existing respiratory disease such as asthma, chronic obstructive pulmonary disease (COPD) and bronchitis; people with cardiovascular disease; people with

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

pulmonary infections such as pneumonia; and children (Streeton 1997). In relation to the data underpinning establishment of the national ambient air standard (NEPC 1998) most of the 'effects' due to particles are associated with exacerbation of existing disease states. The 'effects' observed with elevated PM_{10} concentrations are increased hospital visits and/or admissions for respiratory conditions, decrements in pulmonary function (especially in adults with obstructive airways disease but also in young children), increases in prevalence of pulmonary symptoms and increased mortality (Streeton 1997, RIVM 2002).

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

From daily time series studies (these look at associations between short term, acute exposures to elevated ambient PM_{10} and various population health measures) there is increasing evidence the health effects associated with particulate matter may occur at quite low particulate levels, and also primarily with the $PM_{2.5}$ respirable fraction, furthermore studies have not identified a clear no effect(s) threshold (EC 1997, RIVM 2002, US EPA 2003a). It should not be assumed that only the most old and most frail, who may be very near death, are at risk of dying from PM_{10} exposure. There is evidence that mortality may also be advanced in other 'at risk' groups (Zeger et al. 1999). In addition the number of those susceptible to less serious health effects such as increased respiratory symptoms, decreased lung function, or other physiologic changes may be broader than those at risk of dying. For most people, these effects are likely to be small, transient, and maybe even unnoticed. For a few, the decline in lung function may be clinically relevant resulting in increased bronchodilator use and/or emergency hospital visits, or the effects may result in short-term absence from work or school (Pope 2000).

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

increased mortality. However there is not sufficient information at this time to confidently dismiss crustal particulates as not having a role in causing adverse respiratory effects.

Sulphur dioxide:

Key findings

- The population of concern for the effects of short-term (approximately 5 – 15 minutes) SO_2 exposure consists of individuals who are mild to moderate asthmatics, are SO_2 responders and are undertaking exercise/activity that raises ventilation rate.
- The no observed effect level for acute exacerbation of asthma symptoms in SO_2 responding exercising asthmatics is about 525 µg/m^3 (200 ppb).
- The low effect level for acute effects is approximately 1570 µg/m^3 (600 ppb)
- Evaluation of acute health effects should occur over an averaging period of 15 minutes, the UK guideline value of 262 µg/m3 (100 ppb) is utilised as the acute guideline value in this HRA.

It is clear from a variety of reviews on SO_2 (US EPA 1994, 1996, Streeton 1997, CalEPA 1999c, EC 2005) that healthy, non asthmatic individuals are essentially unaffected by acute exposures to SO_2 when concentrations are about 1-2 ppm (approximately 2,800 – 5,700 µg/m^3), and that the population of concern for the effects of short-term (approximately 5 – 15 minutes) SO_2 exposure consists of individuals who are mild to moderate asthmatics, are SO_2 responders and are undertaking exercise/activity that raises ventilation rate[22]. However not all asthmatics in these circumstances will experience adverse effects on exposure to SO_2 even at concentrations > 0.6 ppm. The proportion of asthmatic individuals who respond, the magnitude of the response and the occurrence of symptoms increase as SO_2 concentration and ventilation rates increase.

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

a. ***Establish a 1 hour standard and guidance for local jurisdictions for interpreting short term exceedances.*** A discussion of whether to adopt a 10 minute sulphur dioxide standard is ongoing in Australia, however it is likely that a national 10 minute advisory standard for SO_2 will not be adopted, but instead a non-statutory guideline value could

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

be developed to assist in health impact assessments of SO_2 emissions from individual sources [23]. This approach is similar to the approach taken in the United States. In 1994, 1996 and finally in 1998, the US EPA declined to establish a short term (5 or 10 minute) National Ambient Air Quality Standard (NAAQS) for SO_2. They argued that short term exposures to concentrations of SO_2 sufficient to produce adverse effects over 5 minutes were associated with point source emissions, were infrequent and affected only a small portion of the susceptible population (i.e. exercising asthmatics) hence a NAAQS for the whole population was not warranted. However the US EPA have proposed an 'Interim Protection Level Program' to assist states in managing risks associated with short term (5 minute) exposures but allowing due cognisance to be given to specific local factors in deciding whether adverse health risks were likely.

b. **Establish a short term guideline value.** The WHO (2000a, 2006) has established a 10 minute guideline value of 0.175 ppm.

The UK (UK EPAQS 1995) considered that most of the acute clinical studies did not show an effect below 250 ppb and although 'occasional subjects' had responded to lower concentrations with transient changes in measurements of lung function, these transient changes were "*insufficient to be associated with symptoms*". Furthermore it was considered that the transient changes occurred in asthmatic subjects breathing through a mouthpiece while exercising on a bicycle thus the normal protective mechanism of nasal mucosal scrubbing of SO_2 was bypassed. The Panel recommended an Air Quality Standard for SO_2 in the United Kingdom of 100 ppb, measured over a 15 minute averaging period. Dose response analysis suggests an effect would not be anticipated in sensitive individuals until the UK guideline had been exceeded 2 -3 fold. The rationale used by the UK panel for setting such a low standard was that exacerbation of asthmatic symptoms could occur with exposures as short as 1 minute however it is impractical to measure SO_2 over this very short time frame. Fifteen minutes was considered the shortest practical period for measurement. Nevertheless this period of measurement could include very brief times of higher concentrations, which could be as much as double the average, and therefore have an effect on susceptible individuals when the average appears safe. The UK panel took this into consideration, as well as the need for an adequate margin of safety for those individuals more severely affected by asthma when they set a level of 100 ppb measured over a 15 minute averaging period. In essence the UK DoE took a NOAEL of 200 ppb and applied a safety factor of

[23] Based on personal communication with the NEPC Secretariat.

2 to account for SO_2 spikes within the 15 min averaging measurement.

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

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

- According to Ayres (1998) the WHO assessed the available literature on long term exposure to SO_2 in 1990 (WHO, 1992) and concluded that:
    - With chronic exposure to SO_2 concentrations of 250 µg/m^3 (87 ppb) there was a measurable increase in respiratory morbidity amongst susceptible adults suffering COPD, and perhaps also in children, with the effect being more marked at concentrations of around 400 µg/m^3 (140 ppb).
    - At chronic SO_2 levels of approximately 200 µg/m^3 (70 ppb), quite small transient reductions in lung function could be seen in children and adults that could last for as much as two to four weeks.
    - Based on prevalence of respiratory symptoms the lowest observed adverse effect level of SO_2 in epidemiological investigations was judged to be 100 µg/m^3 (35 ppb) as an annual average. From this an air quality guideline of 20 ppb was set.
    - At that time it was not possible for the epidemiological studies to reliably distinguish between the effects of particulates or SO_2. There was uncertainty as to whether or not SO_2 was in fact responsible for the observed adverse health effects, or whether it might be acting as a surrogate for another pollutant, especially particulates.
- Since then further review by the WHO (2000b) concluded:

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

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

Table A1.4: Summary of derivation of guideline values for criteria pollutants.

Guideline µg/m^3 / ppb			Derivation	Reference
Carbon monoxide (CO)				
23,000	26,349	Inhalation reference exposure level (REL) 1 hr average	Prevention of angina in persons with known angina and cardiovascular diseases who are exercising heavily. No observed effect level 1.1 – 1.3% COHb which corresponds to 23,000 µg/m^3 calculated toxicokinetically.	Cal EPA (1999a)
30,000	34,368	Ambient air quality guideline 1 hr average	To protect non-smoking, middle-aged and elderly population groups with documented or latent coronary artery disease from acute ischaemic heart attacks, and to protect the foetuses of non-smoking pregnant women from untoward hypoxic effects, a COHb level of 2.5% should not be exceeded. This is achieved at the guideline level. The guideline takes into account all the known physiological variables affecting carbon monoxide uptake.	WHO (2000a&b)
10,310	9,000	Ambient air quality guideline 8 hr avg	Consistent with the recommendations of the WHO (see description of the WHO guideline above).	NEPC (1998), Streeton (1997)
Nitrogen dioxide (NO_2)				
226[b]	120	Ambient air quality guideline 1 hr average	The Australian National Environmental Protection Council ambient air quality standard. It is based on a low observed adverse effect level (LOAEL) of 0.2 to 0.3 ppm derived from statistical reviews of epidemiological data suggesting an increased incidence of lower respiratory tract symptoms in children and aggravation of asthma. An uncertainty factor of 2 to protect susceptible people (i.e. asthmatic children) was applied to the LOAEL.	NEPC (1998), Streeton (1997)
470	250	Inhalation reference exposure level (REL) 1 hr average	The REL is also the ambient air quality standard of California. It is the no observed adverse effect level in sensitive asthmatics for NO_2 mediated increased responsiveness to other bronchoconstrictors (e.g. exercising in cold air).	Cal EPA (1999b)
200	106	Ambient air quality guideline 1 hr average	Lowest concentration causing small (~5%) changes in lung function in mild asthmatics is 560 µg/m^3. Some but not all studies show increased responsiveness to bronchoconstrictors at NO_2 levels as low as 376–560 µg/m^3. In other studies, higher levels had no such effect. Allergen challenges showed no effects at 190 µg/m^3. According to WHO there have been no studies of 1 hour exposures to NO_2 at 100µg/m^3.	WHO (2000b)
40	21	Ambient air guideline Annual avg	WHO (1997) reviewed the epidemiological studies suggesting human health effects associated with long-term NO_2 exposures. On the basis of a background level of 15 µg/m^3 (8 ppb) and the fact that significant adverse health effects could be expected occur with an additional level of 28.2 µg/m^3 (15 ppb) or more, an annual guideline value of 40 µg/m^3 (0.023 ppm) was derived by the WHO (1997). It is considered that the guideline will be protective of most serious effects. The fact that a no-effect level for subchronic or chronic NO_2 exposure concentrations has not yet been determined should be emphasized.	WHO (2000b), WHO (1997)

Guideline µg/m^3	/ ppb		Derivation	Reference
56	30	Ambient air guideline Annual avg	A low observed adverse effect level (LOAEL) of the order of 40 - 80 ppb (approx 75-150 µg/m^3) during early and middle childhood years can lead to the development of recurrent upper and lower respiratory tract symptoms, such as recurrent 'colds', a productive cough and an increased incidence of respiratory infection with resultant absenteeism from school. An uncertainty factor of 2 was applied to the LOAEL to account for susceptible people within the population resulting in a guideline of 20-40 ppb (38-75 µg/m^3).	NEPC (1998), Streeton (1997)
Particulates (PM_{10})				
50	N/A	Ambient air quality guideline 24 hour avg	The Australian National Environmental Protection Council ambient air quality standard was based on increased hospital visits and/or admissions for respiratory conditions, decrements in pulmonary function (especially in adults with obstructive airways disease but also in young children), increased prevalence of pulmonary symptoms and increased mortality.	NEPC (1998), Streeton (1997).
50[d]	N/A	European Union Limit value 24 hour avg	The European limit value is based on the lowest reasonably practical value. The European review was unable to identify a threshold concentration below which ambient PM has no effect (see WHO description below) therefore the limit value was based on the lowest reasonably practical value.	EU (2004)
150	N/A	National air quality standard Annual average[c]	The national air quality standard of 150 µg/m^3 with no more than one expected exceedance per year was first promulgated in 1979. The basis for the standard is not described in recent US EPA reviews of PM standards (US EPA 1996, US EPA 2005) It is important to note the standard is under review. The US EPA is currently considering whether to revise the primary standard for coarse particulate matter to be specific for urban particulate matter. Non urban sources are not consistently associated with health effects (US EPA 2005).	US EPA (2004)
50	N/A	National air quality standard Annual average[c]	The national air quality standard of 50 µg/m^3 was first promulgated in 1979. The basis for the standard is not described in recent US EPA reviews of PM standards (US EPA 1996, US EPA 2005).	US EPA (2004)
40	N/A	European Union Limit value Annual avg	The European limit value is based on the lowest reasonably practical value. The Europeans reviewed the findings of the WHO and studies published since the WHO review and concluded that some studies suggest that long-term exposure to particulate matter is associated with possible effects below 20 µg/m^3 (as $PM_{2.5}$) or 30 µg/m^3 (as PM_{10}).	EU (2004)

Guideline µg/m^3 / ppb			Derivation	Reference
No recommendation for acute or chronic duration.			WHO concluded that the existing database of studies did not enable the derivation of specific guideline values for either acute or chronic duration. The database of studies did show clear and consistent associations between concentrations of particulate matter and adverse effects on human health at low levels of exposure commonly encountered in developed countries. Associations were found for increased lower respiratory symptoms and reduced lung function in children, chronic obstructive pulmonary disease and reduced lung function in adults as well as mortality. Fewer studies were available investigating associations between PM and chronic health effects however some studies identified a reduction of life expectancy (in the order of 1–2 years), increased prevalence of bronchitis symptoms in children, and reduced lung function in children and adults. These effects were considered to be observed at annual average concentration levels at or below current background levels (i.e. below 20 µg/m^3 (as $PM_{2.5}$) or 30 µg/m^3 (as PM_{10})). For this reason, no guideline value for long-term average concentrations is recommended.	WHO (2000a & b)
Sulphur dioxide (SO_2)				
459	175	Ambient air guideline 10 min avg	The WHO considered that only small changes of non clinical significance were seen at 524 µg/m^3 (0.2 ppm).	WHO (2000b)
262	100[e]	Ambient air guideline 15 min avg	The UK (UK EPAQS 1995) considered that most of the acute clinical studies did not show an effect below 250 ppb. For the NOAEL of 200 ppb a guideline was derived by applying a safety factor of 2 to account for SO_2 spikes within the 15 min averaging measurement resulting in an ambient air guideline of 100 ppb.	UK DoE (2000)
524	200	Ambient air quality guideline 1 hr average	The derivation of this value could not be found from the documentation of the NEPC ambient air guidelines. Streeton (1997) does not discuss a 1 hour guideline value as the short term effects of SO_2 occur over a time frame of between 5 and 15 minutes.	NEPC (1998), Streeton (1997).
655	250	Acute Reference Exposure Level 1 hour	Considered that most of the acute clinical studies did not show an effect below 250 ppb (i.e. NOAEL in exercising asthmatics).	CalEPA (1999c)
210	80	Ambient air quality guideline 24 hr avg	Based on epidemiological studies in which the effects of SO_2, particulates, and other associated pollutants are considered. These studies mainly focus on the exacerbation of symptoms in groups of selected sensitive subjects, with symptoms generally developing when SO_2 levels exceeded 0.087 ppm (250 µg/m^3), usually in the presence of particulates (PM_{10}). Thus the Australian 24 hr guideline appears to be based on a LOAEL without application of a 2-fold safety factor to account for susceptible populations. WHO (2000a&b), UK DoE (2000), and EC (2005) all apply a two-fold safety factor to derive a 24 hr guideline value of 40 or 47 ppb.	NEPC (1998), Streeton (1997).
123	47[e]	Ambient air guideline 24 hr avg	Based on epidemiological studies in which the effects of SO_2, particulates, and other associated pollutants are considered. These studies mainly focus on the exacerbation of symptoms in groups of selected sensitive subjects, with symptoms generally developing when SO_2 levels exceeded 87 ppb (250 µg/m^3), usually in the presence of particulates (PM_{10}). The LOAEL is divided by 2 to account for susceptible people.	UK DoE (2000)

Guideline µg/m^3 / ppb			Derivation	Reference
52	20	Ambient air quality guideline Annual avg	Based on prevalence of respiratory symptoms the LOAEL of SO_2 in epidemiological investigations was judged to be 100 µg/m^3 (35 ppb) annual average. From this an air quality guideline of 20 ppb was set.	NEPC (1998), Streeton (1997).
52	20	Ambient air guideline Annual avg	Based on prevalence of respiratory symptoms the LOAEL of SO_2 in epidemiological investigations was judged to be 100 µg/m^3 (35 ppb) annual average. From this an air quality guideline of 20 ppb was set.	WHO (2000b)
52	20 [e]	Ambient air guideline Annual avg	Based on prevalence of respiratory symptoms the LOAEL of SO_2 in epidemiological investigations was judged to be 100 µg/m^3 (35 ppb) annual average. From this an air quality guideline of 20 ppb was set.	UK DoE (2000)
52	20 [e]	Ambient air guideline Annual avg	Based on prevalence of respiratory symptoms the LOAEL of SO_2 in epidemiological investigations was judged to be 100 µg/m^3 (35 ppb) annual average. From this an air quality guideline of 20 ppb was set.	EC (2005)

A1.5: Polyaromatic hydrocarbons (PAHs)

PAHs are a group of chemicals that are formed during the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances, such as tobacco and meat. Active or passive inhalation of tobacco smoke is a major source of exposure for many people. Cooking meat or other food at high temperatures increases the amount of PAHs in the food. There are more than 100 different PAHs which generally occur as part of complex mixtures, thus environmental exposures are not to single PAH compounds. PAHs occur naturally and enter the environment mostly as releases to air from volcanoes, burning off and bush fires, residential wood burning, and exhaust from automobiles and trucks.

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

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

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

Each PAH varies in its potency to cause cancer. Some are not or are only very weak carcinogens, but others such as benzo(*a*)pyrene and dibenzoanthracene are quite potent.

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

It should also be noted however there is information to indicate that co-exposure to mixtures of PAHs often decreases the carcinogenic potency of the most potent PAH (usually benzo(*a*)pyrene), in the mixture, by competitive inhibition of metabolism to the pro-carcinogen or by competitive binding at the nuclear aryl hydrocarbon receptor (WHO 1998a, ATSDR 1995). We consider therefore that the method is more likely to overestimate carcinogenic risk than under estimate it. This will tend to overestimate the carcinogenic potency of the mixture because many of the congeners are not as potent in causing cancer as benzo(*a*)pyrene. The differences in potency between the various PAH congeners are shown in Table A1.5. Here the highest consensus benzo(*a)*pyrene potency equivalency factor for specific PAHs listed by the World Health Organisation (WHO 1998a) are listed.

Due to different assumptions and experimental approaches, estimation of the carcinogenic potency of benzo(*a*)pyrene using either epidemiological or animal data is complex. There are a range of cancer potency values (i.e. unit risk values) for benzo(*a*)pyrene that could be used to calculate cancer risk (Table A4). For this risk assessment we have deferred to the latest value of 8.7×10^{-2} (μg B(*a*)P /m^3)$^{-1}$ quoted by WHO (2000), this is an inhalation cancer potency value for benzo(*a*)pyrene derived from studies of coke-oven workers.

Table A1.5: Potency equivalence factors for PAH congeners

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

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

Table A4: Range of Unit Risk factors for inhalation exposure benzo(a)pyrene [a]

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

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

A1.6: Dioxins

Dioxin Toxicity:

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

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

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

Human sensitivity:

According to WHO (van Leeuwen et al. 2000), data for *Ah* receptor binding affinity and responses directly dependent on *Ah* receptor activation suggest humans may be less susceptible to dioxin than the ‘responsive’ rodent strains often used in experimental studies.

Conversely, other biochemical or cellular effects suggest comparable susceptibility, however these latter effects are not associated with adverse health and their clinical significance is largely unknown (OCS 2004). Hays et al. (1997) evaluated the relative susceptibility of humans and rats for cancer using several dose metrics applied to the pivotal rat bioassay (Kociba et al. 1978, Goodman and Sauer 1992) and the US National Institute of Occupational Safety and Health (NIOSH) worker cohort (Fingerhut et al. 1991). Both these studies had data available on biological dose (blood lipid or adipose tissue TCDD levels) and cancer response. The authors concluded humans are much less sensitive than rats to the carcinogenic effects of TCDD. Others have also suggested that humans are less or no more susceptible to the toxic effects of TCDD and hence exposure of the general population to environmental levels of dioxins should not be of concern (Kimbrough 1990, Leung et al. 1990). More recent comparisons of cytochrome P450 (CYP1A1) induction by TCDD in fresh hepatocytes from human donors, rats and rhesus monkeys indicates that humans are about 10 – 100 times less sensitive than are rats (Silkworth et al. 2005). Since the TEFs for dioxin congeners are in large part based on the responsiveness of the rat tissues to *Ah* –receptor mediated biochemical responses it suggests the TEF allocation for congeners may be over estimating the risk to humans by at least an order of magnitude.

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

Health Guideline:

To emphasise the relatively long time frames required for exposure to dioxin like substances before human health effects are likely to occur the Australian NHMRC/TGA recommend [24] (NHMRC 2002b) a 'Tolerable Monthly Intake' (TMI) of 70 pg TEQ/kg bw; this is instead of the more common 'Tolerable Daily Intake' recommended for most other substances. The TMI is a monthly intake of dioxins and dioxin like PCBs that can occur over 40 - 50 years, such that the

[24] The TGA recommendation for a tolerable monthly intake of dioxin-like substances for Australians is based on deliberations of the WHO (1998b), EC-SCF(2001) and JECFA (2001) and was endorsed by the NHMRC on 24th October 2002. The guideline was established through the NHMRC process to ensure national acceptability. The report upon which the guideline is based underwent public consultation processes and was subject to external review before finalisation. This health reference value for dioxin like substances is the appropriate value for use in risk assessments for Australia.

body burden associated with adverse health effects is not achieved. The TMI is based on accumulated body burdens in experimental animals associated with subtle adverse effects and a safety factor of about 10 fold is incorporated for humans. That is the TMI is an intake that can pragmatically be considered safe.

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

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

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

mixture have different half lives in the body, prompted the WHO to use an overall composite factor of 10 to account for the uncertainties.

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

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

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

Thresholds and dioxins:

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

Dioxins can cause both non-cancer and cancer effects. It is widely accepted that thresholds exist for the non-cancer effects (EC-SCF 2001, JECFA 2001, FSA 2001, NHMRC 2002b, OCS 2004). However the US EPA (2003b), contrary to other regulatory agencies around the world, has adopted a policy of using a linearised low-dose mathematical model for estimating cancer

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

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

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

A1.7: References for Appendix 1

ACGIH (2006). 2006 guide to occupational exposure values. American Conference of Governmental Industrial Hygienists. Cincinnati, OH.

ATSDR (1995). Toxicological profile for polycyclic aromatic hydrocarbons (PAHs) (Update). U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. (PB/90/258245/AS).

ATSDR (2000). Toxicological profile for toluene. Update (PB/2000/108028). U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. September, 2000. http://www.atsdr.cdc.gov/toxprofiles/tp56.html

ATSDR (2007a). Toxicological profile for ethylbenzene. Draft for public comment. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. http://www.atsdr.cdc.gov/toxprofiles/tp110.html

ATSDR (2007b). Toxicological profile for styrene. Update, draft for public comment. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. http://www.atsdr.cdc.gov/toxprofiles/tp53.html

ATSDR (2007c). Toxicological profile for xylenes. Update (PB2008-100008). U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. August, 2007. http://www.atsdr.cdc.gov/toxprofiles/tp71.html

Ayres, J.G. (1998). Health effects of gaseous air pollutants. In 'Air Pollution and Health'. Ed Hester, R.E., Harrison, R.M. Electronic version from Knovel Corporation.

Bauer, M.A., Utell, M.J., Morrow, P.E., Speers, D.M. and Gibb, F.R. (1986). Inhalation of 0.30 ppm nitrogen dioxide potentiates exercise-induced bronchospasm in asthmatics. American Reviews of Respiratory Disease 134(6): 1203-8. As cited in IEH, 1996.

Bylin, G., Hedenstierna, G., Lindvall, T. and Sundin, B. (1988). Ambient nitrogen dioxide concentrations increase bronchial responsiveness in subjects with mild asthma. European Respiratory Journal 1(7): 606-12.

CalEPA (1999a). Determination of Acute Reference Exposure Levels for Airborne Toxicants Acute Toxicity Summary: Carbon monoxide. Office of Environmental Health Hazard Assessment Californian Environmental Protection Agency.

CalEPA (1999b). Determination of Acute Reference Exposure Levels for Airborne Toxicants Acute Toxicity Summary: Nitrogen dioxide. Office of Environmental Health Hazard Assessment Californian Environmental Protection Agency.

CalEPA (1999c). Determination of Acute Reference Exposure Levels for Airborne Toxicants Acute Toxicity Summary: Sulfur dioxide. Office of Environmental Health Hazard Assessment Californian Environmental Protection Agency.

Connor, K.T. and Aylward, L.L. (2006). Human response to dioxin: Aryl hydrocarbon receptor (AhR) molecular structure, function, and dose-response data for enzyme induction indicate an impaired human AhR. J. Toxicol. Environ. Health, Part B 9: 146 – 171.

Culp, S.J., Gaylor, D.W., Sheldon, W.G., et al. (1998). A comparison of the Tumours induced by Coal Tar and Benzo(*a*)pyrene in a 2-year Bioassay. Carcinogenesis. 19: 117-124.

EC (2004.). Second Position Paper on Particulate Matter. CAFE Working group on Particulate Matter. Europa – Environment. 16 April 2004.

EC (2005). Commission Staff Working Document. Annex to the Report in support of the review of Council Directive 1999/30/EC relating to limit values for Sulphur dioxide, Nitrogen dioxide and Oxides of Nitrogen, Particulate matter and Lead in Ambient Air, with consideration of Council Directive 96/62/EC on Ambient Air Quality Assessment and Management. Commission of the European Communities. Brussel 4/1/2005.

EC-SCF (2001). Opinion of the Scientific Committee on Food (SCF) on the risk assessment of dioxins and dioxin-like PCBs in food. Update based on new scientific information available since the adoption of the SCF Opinion of 22nd November 2000. EC Health and Consumer Protection Directorate- General. Document CS/CNTM/DIOXIN/20 final. Adopted on 30th May 2001.

EU (1997). Ambient air pollution by particulate matter position paper. Technical Working Group on Particles, European Commission. http://ec.europa.eu/environment/air/pdf/pp_pm.pdf

EU (2004). Second position paper on particulate matter. CAFE Working Group on Particulate Matter.

Fingerhut, M.A., Halperin, W.E., Marlow, D.A., Piacitelli, L.A., Honchar, P.A., Sweeney, M.H., Griefe, A.L. Dill, P.A., Steenland, K., Suruda, A.J. (1991). Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. New England Journal of Medicine. 324: 212-218. As cited in Hays et al 1997.

FSA (2001). Statement on the tolerable daily intake for dioxins and dioxin-like polychlorinated biphenyls. Committee on Toxicity of Chemicals in Food, Consumer Products And The Environment. Food Standards Authority UK http://www.food.gov.uk/multimedia/pdfs/cot-diox-full.pdf

Goodman, D.G., and Sauer, R.M. (1992). Hepatotoxicity and carcinogenicity in female sprague-dawley rats treated with 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD): A pathology working group reevaluation. Regulatory Toxicology and Pharmacology. 15: 245-252.

Hasselblad, V., Eddy, D.M. and Kotchmar, D.J. (1992). Synthesis of environmental evidence: nitrogen dioxide epidemiology studies. Journal of Air and Waste Management Association 42: 662-671.

Hays, S.M., Aylward, L.L., Karch, N.J., and Paustenbach, D.J. (1997). The relative susceptibility of animals and humans to the carcinogenic hazard posed by exposure to 2,3,7,8-TCDD: An analysis using standard and internal measures of dose. Chemosphere. 34(5-7): 1507-1522.

IEH (1996). Assessment on Indoor Air Quality in the Home: Nitrogen Dioxide, Formaldehyde, Volatile Organic Compounds, House Dust Mites, Fungi and Bacteria. Assessment A2. Medical Research Council. Institute for Environment and Health, Leicester, United Kingdom. http://www.le.ac.uk/ieh/pdf/ExsumA2.pdf

Jaakkola, J.J.K., Paunio, M., Virtanen, M. and Heinonen, O.P. (1991). Low-level air pollution and upper respiratory infections in children. American Journal of Public Health 81: 1060-1063. As cited in WHO 1997.

JECFA (2001). 57th Meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA), Rome, 5 – 14 June 2001. Summary and Conclusions, Annex 4, pp 24-40. Full report published in Food Additive Series 48 as "Polychlorinated Dibenzodioxins, Polychlorinated Dibenzofurans, and Co-planar Polychlorinated Biphenyls" (2002). pp 451-664.

Kayajanian, G.M. (2002). The j-shaped dioxin dose response curve. Ecotoxicology and Environmental Safety. 51: 1-4.

Kimbrough, R.D. (1990). How toxic is 2,3,7,8-tetrachlorodibenzodioxin to humans? Journal of Toxicology and Environmental Health. 30: 261-271. As cited in Hays et al 1997.

Kociba, R.J., Keyes, D.G., Beyer, J.E., Carreon, R.M., Wade, C.E., Dittenber, D.A., Kalnins, R.P., Frauson, L.E., Park, C.N., Barnard, S.D., Hummel, R.A., Humiston, C.G. (1978). Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rats. Toxicology and Applied Pharmacology. 46: 279-303. As cited in Hays et al 1997.

Kraft, M., Eikmann, T., Kappos, A., Künzli, N., Rapp, R., Schneider, K., Seitz, H., Voss, J-U. and Wichmann, E-H. (2005). The German view: Effects of nitrogen dioxide on human health – derivation of health-related short-term and long-term values. International Journal of Hygiene and Environmental Health 208: 305-318.

Leung, H.W., Poland, A., Paustenbach, D.J., Murray, F.J., Andersen, M.E. (1990). Pharmacokinetics of [125I]-2-iodo-3,7,8-trichlorodibenzo-p-dioxin in mice: analysis with a physiological modeling approach. Toxicology and Applied Pharmacology 103: 411-419.

Laden, F., Neas, L.M., Dockery, D.W. and Schwartz, J. (2000). Association of fine particulate matter from different sources with daily mortality in six US cities. Environ. Health Perspect. 108: 941 – 947.

Morrow, P.E., Utell, M.J., Bauer, M.A., Smeglin, A.M., Frampton, M.W., Cox, C., Speers, D.M. and Gibb, F.R. (1992). Pulmonary performance of elderly normal subjects and subjects with chronic obstructive pulmonary disease exposed to 0.3 ppm nitrogen dioxide. American Reviews of Respiratory Disease 145(2 Pt 1):291-300.

Muller, P., Leece, B. and Raha, D. (1995a). Estimated Risk of Cancer from Exposure to PAH Fractions of Complex Mixtures. In "Fifteenth International Symposium on Polycyclic Aromatic Compounds: Chemistry, Biology and Environmental Impact". Blegirate, Italy. 19-22 September 1995. Ispra, Joint Research Centre European Commission. pp 159-160. As cited in WHO (1998a).

Muller, P., Leece, B. and Raha, D. (1995b). Dose-Response Assessment PAH. Ottawa, Ontario. Ministry of the Environment and Energy, 197 p. As cited in WHO (1998a).

Muller P., Leece, B. and Raha, D. (1996). Scientific Criteria Document for Multimedia Environmental Standards Development: Polycyclic Aromatic Hydrocarbons (PAH). Part 1. Dose Response Assessment. Ottawa, Ontario. Ministry of the Environment and Energy, 203 p. As cited in WHO (1998a).

NEPC (1998). National Environment Protection Measure for Ambient Air Quality. National Environment Protection Council. 26 June 1998.
http://www.ephc.gov.au/nepms/air/air_nepm.html

NEPC (2003). National Environment Protection Measure for Ambient Air Quality, National Environment Protection Council. May 2003.
http://www.ephc.gov.au/nepms/air/air_variation.html.

NEPC (2004a). National Environment Protection (Air Toxics) Measure. National Environment Protection Council. December 2004.
http://www.ephc.gov.au/pdf/Air_Toxics/FinalAirToxicsNEPM.pdf

NEPC (2004b). NEPC Annual Report 2003 – 2004. Jurisdictional Reports on Implementation and Effectiveness of NEPMs. National Environmental Protection Council.
http://www.ephc.gov.au/pdf/annrep_03_04/173_176_AAQ_7_Tas.pdf

NHMRC (2002a) Ambient Air Quality Goals recommended by the National Health and Medical Research Council. Rescinded 19/3/2002.

NHMRC (2002b). Dioxins: Recommendation for a tolerable monthly intake for Australians. National Health and Medical Research Council, Commonwealth of Australia.

NICNAS (2006). Priority Existing Chemical Assessment Report No. 28 – Formaldehyde. National Industrial Chemicals Notification and Assessment Scheme. Commonwealth of Australia. November 2006
http://www.nicnas.gov.au/Publications/CAR/PEC/PEC28/PEC_28_Full_Report_PDF.pdf

OCS (2004). Human health risk assessment of dioxins in Australia, National Dioxins Program Technical Report No. 12. Office of Chemical Safety, Australian Government Department of Health and Ageing, Australian Government Department of the Environment and Heritage, Commonwealth of Australia.

OEHHA (1999a). Arsenic and inorganic arsenic compounds Acute Toxicity Summary; Determination of Acute Reference Exposure Levels for Airborne Toxicants. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency.
http://www.oehha.ca.gov/air/acute_rels/pdf/ArsInArsA.pdf

OEHHA (1999b). Benzene Acute Toxicity Summary; Determination of Acute Reference Exposure Levels for Airborne Toxicants. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. http://oehha.ca.gov/air/acute_rels/pdf/71432A.pdf

OEHHA (1999c). Copper Acute Toxicity Summary; Determination of Acute Reference Exposure Levels for Airborne Toxicants. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. http://oehha.ca.gov/air/acute_rels/pdf/CusA.pdf

OEHHA (1999d). Mercury Acute Toxicity Summary; Determination of Acute Reference Exposure Levels for Airborne Toxicants. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. http://oehha.ca.gov/air/acute_rels/pdf/HgA.pdf

OEHHA (1999e). Nickel and nickel compounds Acute Toxicity Summary; Determination of Acute Reference Exposure Levels for Airborne Toxicants. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency.
http://www.oehha.ca.gov/air/acute_rels/pdf/NiA.pdf

OEHHA (1999f). Phenol Acute Toxicity Summary; Determination of Acute Reference Exposure Levels for Airborne Toxicants. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. http://www.oehha.ca.gov/air/acute_rels/pdf/108952A.pdf

OEHHA (1999g). Styrene Acute Toxicity Summary; Determination of Acute Reference Exposure Levels for Airborne Toxicants. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. http://www.oehha.ca.gov/air/acute_rels/pdf/100425A.pdf

OEHHA (2000a). Benzene. Chronic Toxicity Summary; Determination of Chronic Reference Exposure Level for Airborne Toxicants. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency.
http://www.oehha.ca.gov/air/chronic_rels/22Chrels.html

OEHHA (2000b). Nickel and compounds. Chronic Toxicity Summary; Determination of Chronic Reference Exposure Level for Airborne Toxicants. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. February, 2000.
http://www.oehha.ca.gov/air/chronic_rels/22Chrels.html

OEHHA (2000c). Phenol. Chronic Toxicity Summary; Determination of Chronic Reference Exposure Level for Airborne Toxicants. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. April, 2000.
http://www.oehha.ca.gov/air/chronic_rels/16Chrels.html

OEHHA (2001a). Arsenic and arsenic compounds. Chronic Toxicity Summary; Determination of Chronic Reference Exposure Level for Airborne Toxicants. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. January, 2001.
http://www.oehha.ca.gov/air/chronic_rels/22more.html

OEHHA (2001b). Selenium and selenium compounds. Chronic Toxicity Summary; Determination of Chronic Reference Exposure Level for Airborne Toxicants. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. December, 2001. http://www.oehha.ca.gov/air/chronic_rels/12Dec2001CRELs.html

OEHHA (2002). Technical Support Document for Describing Available Cancer Potency Factors. Benzo (α) pyrene. Office of Environmental Health Hazard Assessment.

Pohl, H.R., Hicks, H.E., Jones, D.E., Hansen, H. and De Rosa, C.T. (2002). Public health perspectives on dioxin risks: Two decades of evaluations. Human and Ecological Risk Assessment. 8(2): 233-250.

Pope, C.A .(2000). Epidemiology of fine particulate air pollution and human health: Biologic mechanisms and Who's at Risk? Environ. Health Persp. 108 Suppl. 4: 713-723.

Popp, J.A., Crouch, R.L. and McConnell, E.E. (2006). A weight-of-evidence analysis of the cancer dose-response characteristics of 2, 3, 7, 8- tetradibenzodioxin (TCDD). Toxicological Sciences. 89(2): 361-369.

RIVM (2001). Re-evaluation of Human-toxicological Maximum Permissible Risk Levels. Report 711701 025.

RIVM (2002). On health risks of ambient PM in the Netherlands, Netherlands Aerosol Programme, National Institute of Public Health and the Environment (RIVM), Report 650010 032, October 2002.

Roger, L.J., Horstman, D.H., McDonnell, W.F., Kehrl, H., Seal, E., Chapman, R.S. and Massaro, E.J. (1985). Pulmonary effects in asthmatics exposed to 0.3 ppm NO_2 during repeated exercise. Toxicologist 5: 70. As cited in IEH, 1996.

SAB (1995). Review of the Office of Research and Development's reassessment of dioxin and dioxin-Like compounds by the dioxin reassessment review committee. An EPA SAB Report: A Second Look at Dioxin. Science Advisory Board of the US EPA, September 29, 1995.

Schwarz, M., and Appel, K.E. (2005). Carcinogenic risks of dioxin: Mechanistic considerations. Regulatory Toxicology and Pharmacology. 43: 19-34.

Silkworth, J.B., Koganti, A., Illouz, K., Possolo, A., Zhao, M., Hamilton, S.B. (2005). Comparison of TCDD and PCB CYP1A induction sensitivities in fresh hepatocytes from human donors, sprague-dawley rats, and rhesus monkeys and HepG2 cells. Toxicological Sciences. 87: 508-519.

Slooff, W., Janus, J.A., Matthijsen, A.J.C.M. et al. (1989). Integrated Criteria Document on PAHS (Report No. 758474011). Bilthoven, National Institute of Public Health and Environmental Protection. P15-36. As cited in WHO (1998a).

Strand, V., Salomonsson, P., Lundahl, J. and Bylin, G. (1996). Immediate and delayed effects of nitrogen dioxide exposure at an ambient level on bronchial responsiveness to histamine in subjects with asthma. European Respiratory Journal 9(4): 733-40.

Strand, V., Rak, S., Svartengren, M. and Bylin, G. (1997). Nitrogen dioxide exposure enhances asthmatic reaction to inhaled allergen in subjects with asthma. American Journal of Respiratory and Critical Care Medicine 155(3): 881-7.

Streeton, J. A. (1997). A Review of Existing Health Data on Six Air Pollutants. Prepared for the National Environment Protection Council. May. NEPC Service Corporation. http://www.ephc.gov.au/

Tunnicliffe, W.S., Burge, P.S. and Ayres, J.G. (1994). Effect of domestic concentrations of nitrogen dioxide on airway responses to inhaled allergen in asthmatic patients. Lancet 344(8939-8940): 1733-1736.

UK DoE (2000). The Air Quality Strategy for England, Scotland, Wales and Northern Ireland. Working Together for Clean Air Published by the Department of the Environment, Transport and the Regions, in partnership with the Scottish Executive, The National Assembly for Wales, and the Department of the Environment for Northern Ireland. Section 9.

UK EPAQS (1995). A Recommendation for a United Kingdom Air Quality Standard for Sulphur Dioxide Expert Panel on Air Quality Standards, Department of the Environment, Transport and the Regions.

US EPA (1994). Review of the National Ambient Air Quality Standards for Sulfur Oxides. Assessment of Scientific and Technical Information. EPA-452/R-94-013.

US EPA (1996). National Ambient Air Quality Standards for Sulfur Oxides. Final Decision. Federal Register. Vol 61, No. 100. Wednesday, May 22, 1996. Notices 25566.

US EPA (2000). Exposure and health assessment for 2,3,7,8 – Tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. National Centre for Environmental Assessment Office of Research and Development, U.S. Environmental Protection Agency.

US EPA (2003a). Fourth External Review Draft of Air Quality Criteria for Particulate Matter, US Environmental Protection Agency National Center for Environmental Assessment EPA/600/P-99/002aD June 2003.

US EPA (2003b). Exposure and human health reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and related compounds. December 2003. NAS Review Draft National Center for Environmental Assessment Research and Development. U.S. Environmental Protection Agency, Washington, DC. http://www.epa.gov/ncea/dioxin

US EPA (2004). Air Quality Criteria for Particulate Matter U.S. Environmental Protection Agency, Washington, DC, EPA 600/P-99/002aF-bF, 2004. http://cfpub2.epa.gov/ncea/cfm/recordisplay.cfm?deid=87903

US EPA (2005). Review of the National Ambient Air Quality Standards for Particulate Matter: Policy assessment of scientific and technical information. OAQPS Staff Paper. United States Environmental Protection Agency. http://www.epa.gov/ttn/naaqs/standards/pm/data/pmstaffpaper_20051221.pdf

US EPA IRS (1998). Integrated Risk Information System, IRIS Summary – ChromiumVI, United States Environmental Protection Agency. http://www.epa.gov/ncca/iris/subst/0144.htm

van den Berg, M., Birnbaum, L., Bosveld, A. T. C., Brunstrom, B., Cook, P., Feeley, M., Giesy, J.P., Hanberg, A., Hasegawa, R., Kennedy, S.W., Kubiak, T., Larsen, J.C., van Leeuewen, F.X.R., Liem, A.K.D., Nolt, C., Peterson, R.E., Poellinger, L., Safe, S., Schrenk, D., Tillitt, D., Tysklind, M., Younes, M., Waern, F. and Zacharewski, T. (1998). Toxic equivalency factors (TEFS) for PCBs, PCDDs, PCDFs for humans and wildlife. Environ. Health Persp. 106: 775-792.

van Leeuwen, F.X.R., Feeley, M., Schrenk, D., Larsen, J.C., Farland, W., and Younes, M. (2000). Dioxins: WHO's tolerable daily intake (TDI) revisited. Chemosphere. 40: 1095-1101.

WHO (1987): Air Quality Guidelines for Europe. WHO Regional Publications, European Series No. 23. World Health Organisation, Geneva.

WHO (1992). European Series No 43, Copenhagen. (As cited by Ayres (1998).

WHO (1997). Environmental Health Criteria 188, Nitrogen Oxides. Second Edition. World Health Organisation, Geneva.

WHO (1998a). Environmental Health Criteria 202. Selected Non-Heterocyclic Polycyclic Aromatic Hydrocarbons. International Programme on Chemical Safety, World Health Organization, Geneva.

WHO (1998b). Assessment of the Health Risks of Dioxins: Re-evaluation of the Tolerable Daily Intake (TDI). Executive summary of the WHO Consultation, May 25 – 29, Geneva, Switzerland.

WHO (2000a). Guidelines for Air Quality. World Health Organisation, Geneva.

WHO (2000b). Air Quality Guidelines for Europe. 2nd Edition. World Health Organization Regional Publications, European Series Number 91. http://www.euro.who.int/document/e71922.pdf

WHO (2000c). Safety Evaluation of Certain Food Additives and Contaminants Simple Aliphatic and Aromatic Sulfides and Thiols. WHO Food Additives Series: 44. Prepared By The Fifty-Third Meeting Of The Joint FAO/WHO Expert Committee On Food Additives (JECFA) World Health Organization, Geneva.

WHO (2006). Air Quality Guidelines Global Update 2005. Particulate matter, ozone, nitrogen dioxide and sulphur dioxide. World Health Organisation. http://www.euro.who.int/Document/E90038.pdf

Zeger, S.L., Dominici, F. and Samet, J. (1999). Harvesting-resistant estimates of air pollution on mortality. Epidemiology 10: 171 – 175.

Appendix 2: Screening for secondary exposure pathways

A2.1: Introduction

Risk assessments involving secondary exposure pathways (Figure 3.1) via the food chain are very complicated, dogged by lack of specific data and therefore necessitate numerous assumptions, and are time and resource intensive. Consequently Toxikos has developed screening methodologies to help determine if health effects by food related secondary exposure pathways is likely and therefore if a detailed risk assessment for them is needed.

For organic compounds the screen is simply whether the substance is recognised as having bioaccumulative properties. Dioxins met this criteria and so have been assessed separately in Section 7. As seen below PAHs do not met the criteria.

Metals present different risk assessment issues than do organic compounds. To address metals a set of three criteria have been developed, these are explained in Section A2.3.

A2.2: Polyaromatic hydrocarbons

PAHs occur naturally and enter the environment mostly as releases to air from volcanoes, burning off and bush fires, residential wood burning, and exhaust from automobiles and trucks. PAHs breakdown in air by reacting with sunlight and other substances over a period of days to weeks, in soil, water and sediment breakdown generally takes weeks to months and is primarily through the actions of micro organisms (ATSDR 1995).

The concentrations of individual PAHs in ambient air around the world varies over several orders of magnitude but are generally in the range <0.1 – 100 ng/m^3. The average levels of individual PAHs in ambient air of rural areas are generally 0.1 – 1 ng/m^3, and in urban areas 1 – 30 ng/m^3 with some locations being greater than 200 ng/m^3 for specific PAHs (WHO 1998). The highest predicted annual ground level concentrations for total PAH from the power station is 0.99 ng/m^3 (Table 3.1). This concentration is at the top end of the rural background range of 0.1 – 1 ng/m^3 reported by WHO (1998).

The concentration of PAH in vegetation is generally considerably lower than that in soil; bioaccumulation factors ranging from 0.0001-0.33 for benzo (α)pyrene and from 0.001-0.18 for 17 other PAHs have been reported (WHO 1998a). In UK cropland soils, given repeated

applications of PAHs in sewerage sludge over a number of years, the concentrations of PAHs in plants did not correlate with soil concentrations, and PAH on above ground plant parts were concluded as probably being the result of atmospheric deposition. In a separate study there was minimal movement of PAHs from the root peel of carrots to the inner core, suggesting simple adsorption onto the roots was the major process whereby PAHs may be found on plants (ATSDR 1995). Thus there is little uptake and translocation of PAHs by plants from soil.

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

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

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

A2.3: Metals

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

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

Specific information on the metal content of particulate size fractions of the emissions from the power station is not available. Indeed application of the screening process herein suggests such information, required for estimating deposition rates of metals onto soil and pasture surfaces is not required because the screen criteria are not met for consideration of secondary exposure pathways.

The following general assumptions are made in relation to the possibility of significant incremental exposure/intake of metals over background via secondary exposure pathways. The assumptions are used as the screening criteria to decide whether secondary exposure pathways should be evaluated.

A2.3.1: Development of screening criteria for metals:

Based on primary toxicological principles and knowledge of situations where metal toxicity via secondary exposure mechanisms in the food chain has been shown to be a significant contributor to adverse health effects the following screening criteria have been developed by Toxikos.

1) The metal should exhibit significant bioaccumulation by plants, especially common vegetables and grasses, and animals. If biomagnification occurs then concern for human

exposure via food increases as does the need for secondary exposure pathway evaluation. Obviously if the metal in question does not accumulate in human food sources then concern regarding potential health effects via secondary exposure pathways is not warranted.

Screening criteria 1:
The available weight of literature evidence must indicate the metal is able to bioaccumulate, or biomagnify, into human food sources.

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

Screening criteria 2:
To trigger detailed evaluation of secondary exposure pathways the predicted incremental increase in annual ground level concentration of the metal must be above those measured in rural and remote areas[25].

3) The intake of metals into the body via inhalation is a relatively small proportion of total intake; most metals enter the body via the diet. For bioaccumulative metals a small increase in intake via inhalation could result in a larger overall intake since inhalation is only a small part of total intake. The requirement therefore is to ensure the incremental increase in inhalation intake is sufficiently small to keep total intakes low. This will ensure an adequate

[25] Background airborne concentrations of metals are rarely known at the locations of interest. An increase equal to or greater than background levels cited in the literature for remote or rural areas implies at least a two fold increase of those background concentrations. Such an increase may or may not lead in the long term to an increase in metal content of human foods; intuitively it is suspected that it would not lead to an increase in food chain concentrations. This criteria is therefore conservative and is unlikely to lead to a 'false negative' situation for the metals.

safety margin is maintained in the screening process such that total intake in the future is kept below any intake level that may be deleterious to health.

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

Within this criteria is an assumption that 5% of the total intake is via inhalation and that the remaining 95%, which is ingested either in diet or by consuming soil, has the same toxicological potency and health effects as inhalation exposure, this is often not the case. Ingested metals are often less toxic[26] because homeostatic mechanisms limit their absorption and the spectrum of effects are different. Therefore the tacit assumption embedded in using a low inhalation hazard quotient as triggering criteria errs on the side of safety.

The rationale underpinning the inhalation exposure 'cut-off' is similar to that used in the dioxin risk assessment. Inhalation exposure of metals generally constitutes only approximately <1 – 20% of background intake (e.g. Langley 1991a,b; Maynard 1991). Arsenic and cadmium are two metals for which concern is often expressed regarding accumulation in the environment, according to the European Commission (EC 2000) the percentage for arsenic and cadmium uptake via ambient air is less than 1 and 3 % respectively.

Screening criteria 3:
To trigger detailed evaluation of secondary exposure pathways the hazard quotient for direct inhalational exposure of a metal must be more than 0.05.

[26] The notable exceptions to this are mercury, cadmium and thallium. With these metals route of intake has relatively little impact on final health outcome and health effects are generally elicited at a lower exposure/intake via inhalation than by ingestion or dermal exposure.

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

*The criteria **for triggering** a detailed multimedia/multipathway health risk assessment for metals in the power station emissions are:*

1. *The metal must be capable of being bioaccumulated by plants and animals relevant for the human food chain, and*
2. *The predicted annual ground level concentration increment must be above those measured in rural and remote areas, or*
3. *The direct inhalation hazard quotient (HQ) for incremental annual average concentration any given metal is greater than 0.05.*

A2.3.2: Discussion of metal screening criteria

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

Boudet et al. (1999) evaluated a modern municipal waste incinerator in France. The authors only evaluated direct inhalation health effects. While acknowledging secondary pathways are potentially relevant to such assessments, they cited previous studies[27] which had shown the burden of metals in the food chain in the vicinity of modern facilities was not increased. It can therefore be rationalised that inclusion of those pathways would not influence the results from assessment of direct inhalation.

The general approach and conservativeness of the criteria applied herein for assessing the need for evaluating secondary exposure pathways for metals is supported by the results of a

[27] At the time of writing these studies were not available to Toxikos.

multimedia, multipathway risk assessment for waste combustors[28] conducted for the US EPA (HWC) in support of establishing technical emission standards for these facilities (RTI 1999a). This very comprehensive health risk assessment addressed direct and indirect (i.e. secondary food chain) exposures for 79 HWCs, it focussed on the population within a radius of 20 km and evaluated 14 metals[29] for each facility. The assessment included likely maximum exposed individuals who, due to their activities, could be at increased risk (e.g. recreational fishermen, and subsistence farmers and home gardeners).

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

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

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

The US Department of Energy (US DOE 2003) conducted a quantitative risk assessment to assess mercury health risks associated with two coal fired power plants. Specifically the study quantified the impact of local mercury deposition from coal-fired power plants on risks from

[28] The hazardous waste combustors were comprised of cement kilns, light-weight kilns and incinerators.
[29] Antimony, chromium VI, chromium III, arsenic, lead, barium, nickel, beryllium, selenium, cadmium, silver, thallium, cobalt, copper, and manganese.

foetal exposure through maternal consumption of fish. The risk assessments performed included three different test cases for each plant and two population groups. The population near one plant consumed 17% locally caught fish, similar to the average value in the northeast USA and the population near the other plant consumed 22% locally caught fish, similar to the average value for the Southeast of the United States. Subsistence fishers were assumed to consume 100% locally caught fish. The authors concluded that the risk to the general population from local deposition of mercury from coal-fired power plants is small. The authors identified that the risk assessment is dependent on the size and distance to the water body where fish are caught and the consumption rate of fish. In contrast to these risk assessments is a recent suggestion that environmentally released mercury may be associated with higher local incidences of autism in children (Palmera et al. 2005). While this report calls for further research on the possible link it is noted that the health point measured has not been included as part of previous risk assessments for environmental exposure to mercury.

A2.3.3: Application of criteria

Criteria 1: Bioaccumulation of metals

Of the metals considered herein, only cadmium appears to have potential for accumulation and bioconcentration by green plants (Fergusson 1990). Notes on the bioaccumulation potential of the metals can be found in Section A2.4.

It is to be noted that the air guideline used for cadmium in this risk assessment has been established by WHO (2000) to prevent further increases of cadmium in agricultural soils that would be likely to increase the dietary intake of future generations[30]. This was established because renal effects were observed in inhabitants of areas contaminated by past emissions of cadmium suggesting the cadmium body burden of the general population in some parts of Europe cannot be increased without endangering renal function. It is unlikely that such a precarious situation exits in Australia.

Criteria 2: Air concentrations relative to background.

Because background air concentrations of metals in the general area are not known at this time, background concentrations for other parts of the world have been sought from authority reviews. These are presented in Table A2.1 together with the predicted highest ground level

[30] Toxikos has been unable to obtain the documentation used by the European Chapter of the WHO to support this statement.

concentration. For all the metals the predicted ground level concentrations are within background concentrations at rural or remote locations elsewhere in the world. Metal exposure via either direct inhalation or secondary pathways has not been flagged as an issue at these background locations.

Table A2.1: Comparison of background ambient air concentrations for metals with the predicted increment in annual ground level concentrations due to the power station .

Metal	Predicted annual GLC (ng/m^3)	Ambient background (ng/m^3)	Comment on location	Reference
Cadmium	0.0031	< 5 1-5 5-50 0.03-1	US general ambient. Rural Locations. Urban/ Industrialised Areas NSW Urban	ATSDR (1999b) WHO (1992) NSW (2003)
Lead	0.013	0.1-8 200-400 100 1000-3000 5000-1000 2.4-99 140-1570 20 44-173	Remote areas. US urban areas. Australia Rural. Australia Urban Australia- Near heavy traffic NSW Urban WA Urban WA CBD Launceston, Tasmania	WHO (1977) ATSDR (1999c) Maynard (1991) NSW (2003) EA (2002) NEPC (2002) EA (2002)
Mercury	0.0074	10 – 20 1 2-4 10	Industrialised areas. Remote Southern Hemisphere Rural areas. Urban areas	ATSDR (1999a) WHO (1989) WHO (2000)

Criteria 3: Air concentration relative to health guideline.

A through evaluation of the contribution of inhalation exposure to total intake of metals has not been undertaken. However Langley (1991a,b) and Maynard (1991) indicate for arsenic, cadmium and lead, inhalation represents approximately 0.2%, 10% and 20% respectively of total intake. According to a European Commission (EC 2000) the percentage for arsenic and cadmium uptake via ambient air is less than 1 and 3 % respectively. An inherent assumption in the screening criteria is that oral and inhalation exposure have the same propensity for causing an adverse health effect. However, for many of the metals there is quite different potency for adverse health effects for oral versus inhalation exposure, the latter being of greater concern[31]. Hence since health based guidelines for inhalation exposure is used as the basis for the

[31] This statement is based on the general knowledge of the writer; a formal analysis to support the statement has not been undertaken.

screening criteria in this section, they are intuitively conservative. Given the above information, for metals in emissions from the power station, it was considered an incremental increase in inhalation exposure of 5% relative to the inhalation health guidelines, i.e. a hazard quotient of 0.05, would leave sufficient conservatism for screening purposes to ensure health effects arising from long term exposure from all exposure pathways was unlikely to result in adverse health effects.

The absolute amount of metals added to the air shed by the power station is very small and the highest chronic metal hazard quotient (Table A3.3) at any receptor is only 0.0006, this being for cadmium (Appendix 3). Hence it is unlikely a demonstrable increase in either air concentration or deposited metal within the air shed will occur as a result of the power station emissions.

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

Table A1.2: Chronic HQ for metals due to emissions.

Metal	Predicted annual ground level concentration (ng/m^3)	Chronic Guideline Value [a] (ng/m^3)	Hazard Quotient
Cd	0.0031	5	0.0006
Hg	0.0074	1,000	0.000007
Pb	0.013	500	0.00003

[a] Guideline values can be found in Appendix 1.

A2.4: Notes on the bioaccumulation of metals.

Cadmium

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

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

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

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

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

Since cadmium accumulates largely in the liver and kidneys of vertebrates and not in the muscle tissue and intestinal absorption of cadmium is low, biomagnification through the food chain may not be significant. Nevertheless, uptake of cadmium from soil by feed crops may result in high levels of cadmium in beef and poultry (especially in the liver and kidneys). This accumulation of cadmium in the food chain has important implications for human exposure to cadmium, whether or not significant biomagnification occurs because these animal parts are often used to manufacture salami and other processed meat products (ATSDR 1999b).

Lead

Plants and animals may bioconcentrate lead but biomagnification has not been detected. In general, the highest lead concentrations are found in aquatic and terrestrial organisms that live near lead mining, smelting, and refining facilities; storage battery recycling plants; areas affected by high automobile and truck traffic; sewage sludge and soil disposal areas; sites where dredging has occurred; areas of heavy hunting (lead source from spent shot); and in

urban and industrialized areas. Lead may be present on plant surfaces as a result of atmospheric deposition; its presence in internal plant tissues indicates biological uptake from the soil and leaf surfaces. Although the bioavailability of lead in soil to plants is limited because of the strong absorption of lead to soil organic matter, the bioavailability increases as the pH and the organic matter content of the soil are reduced. Lead is not biomagnified in aquatic or terrestrial food chains. It may contaminate terrestrial plants as a result of atmospheric deposition and uptake from soil, and animals as a result of inhalation of contaminated ambient air or ingestion of contaminated plants. Older organisms tend to contain the greatest body burdens of lead. In aquatic organisms, lead concentrations are usually highest in benthic organisms and algae, and lowest in upper trophic level predators (e.g., carnivorous fish) (ATSDR 1999c).

Mercury

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

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

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

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

The potential for bioaccumulation in terrestrial food chains is demonstrated by the uptake of mercury by edible mushroom, grown on compost containing mercury at concentrations of up to 0.2 mg/kg (ppm). The bioaccumulation factors reported ranged from 65 to 140, indicating that there are potential risks to human health if these mushrooms are eaten in large quantities. Data from higher plants indicate that virtually no mercury is taken up from the soil into the shoots of plants such as peas, although mercury concentrations in the roots may be significantly elevated and reflect the mercury concentrations of the surrounding soil (ATSDR 1999a).

A2.5: References for Appendix 2

ATSDR (1995). Toxicological profile for polycyclic aromatic hydrocarbons (PAHs) (Update). U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. (PB/90/258245/AS).

ATSDR (1999a). Toxicological Profile for Mercury. Agency for Toxic Substances and Disease Registry, Public Health Service, US Department of Health and Human Services. (Update) (PB/99/142416).

ATSDR (1999b). Toxicological Profile for Cadmium. Agency for Toxic Substances and Disease Registry, Public Health Service, US Department of Health and Human Services. (PB/99/166621).

Boudet, C., Zmirou, D., Laffond, M., Balducci, F. and Benoit-Guyod, J.L. (1999). Health Risk Assessment of a Modern Municipal Waste Incinerator. Risk Analysis. 19: 1215-1222.

EA (2002). Technical Report 3: Review of Data on Heavy Metals in Ambient Air in Australia. Department of the Environment and Heritage. Environment Australia.

EC (2000) Ambient Air Pollution by As, Cd and Ni Compounds. Position Paper Final Version. Working Group On Arsenic, Cadmium And Nickel Compounds. European Commission.

Fergusson, J.E. (1990). Chapter 10. Heavy Metals in Plants. In “The Heavy Elements: Chemistry, Environmental Impact and Health Effects”. Pergamon Press, pp 337 – 405.

Greim, H. (1990). Toxicological Evaluation of Emissions from Modern Municipal Waste Incinerators. Chemosphere. 20: 317-331.

Langley, A.J. (1991a). Response levels for arsenic. In “The health risk Assessment and Management of Contaminated Sites – Proceedings of a National Workshop on the health Risk Assessment and Management of Contaminated Sites” Eds O.El Saadi and A. Langley, South Australian Health Commission, pp 123 – 136.

Langley, A.J. (1991b). Response levels for cadmium. In “The health risk Assessment and Management of Contaminated Sites – Proceedings of a National Workshop on the health Risk Assessment and Management of Contaminated Sites” Eds O.El Saadi and A. Langley, South Australian Health Commission, pp 137 - 152.

Maynard, E.J. (1991). Response levels for lead. In “The health risk Assessment and Management of Contaminated Sites – Proceedings of a National Workshop on the health Risk Assessment and Management of Contaminated Sites”. Eds O.El Saadi and A. Langley, South Australian Health Commission, pp 102 - 122.

NEPC (2002). WA Ambient Air Quality. In “National Environment Protection Council, Annual Report 2001-2002”. National Environment Protection Council.

NSW (2003). Ambient Air Quality Research Project. Working paper 4. Ambient Concentrations of Heavy Metals in NSW. Department of Environment and Conservation (NSW) (2002).

Palmera, R.F., Blanchard, B., Steina, Z., Mandellc, D. and Miller, C. (2006). Environmental mercury release, special education rates, and autism disorder: an ecological study of Texas. Health Place 12(2): 203-209.

RTI (1999a). Human Health and Ecological Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes Background Document Prepared for U.S. Environmental Protection Agency Office of Solid Waste 401 M Street SW (5307W) Washington, DC 20460 EPA Contract Number 68-W6-0053 RTI Project Number 92U-7298-005.

RTI (1999b). Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes Human Health and Ecological Risk Results Volume 1: Sections I, II, IV, V, VI, and VIII Prepared for U.S. Environmental Protection Agency Office of Solid Waste 401 M Street SW (5307W) Washington, DC 20460 EPA Contract Number 68-W6-0053.

RTI (1999c). Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes Human Health and Ecological Risk Results Volume 3: Section IX, Baseline Prepared for U.S. Environmental Protection Agency Office of Solid Waste 401 M Street SW (5307W) Washington, DC 20460 EPA Contract Number 68-W6-0053.

US DoE (2003). Assessing the Mercury Health Risks Associated with Coal-Fired Power Plants: Impacts of Local Depositions, US Department of Energy. http://www.netl.doe.gov/coal/E&WR/air_q/health_effects/reduced_mercury.html

WHO (1977). Environmental Health Criteria 3 Lead. International Program for Chemical Safety. World Health Organisation, Geneva.

WHO (1989). Environmental Health Criteria 86 Mercury. International Program for Chemical Safety. World Health Organisation, Geneva.

WHO (1992). Environmental Health Criteria 134 Cadmium. International Program for Chemical Safety. World Health Organisation, Geneva.

WHO (1998). Environmental Health Criteria 202. Selected Non-Heterocyclic Polycyclic Aromatic Hydrocarbons. International Programme on Chemical Safety, World Health Organization, Geneva.

WHO (2000). Air Quality Guidelines for Europe 2nd Edition. WHO Regional Publications, European Series 91. World Health Organisation. Regional Office for Europe, Copenhagen. http://www.euro.who.int/air/activities/20050223_4

Appendix 3

Table A3.1: Calculation of Acute Hazard Indices using Maximum predicted Ground Level Concentrations

MAXIMUM BPS			
Pollutant	**AGV ug/m3**	**GLC ug/m3**	**HQ**
NO_2	226	60.5	0.3
SO_2 (15 mins)	262	*86.8*	0.3
CO	11000	4.9	0.0004
PM_{10} (24hr)	50	3.23	0.06
$PM_{2.5}$ (24hr)	25	3.2	0.1
PAH	-	-	
Dioxin/Furan	-	-	
TVOCs	-	-	
Ethylbenzene	43400	0.006	0.0000001
Phenol	5800	0.01	0.000002
Styrene	21000	0.37	0.00002
Toluene (6 hrs)	15070	*0.13*	0.000008
Xylene (30 mins)	4340	*0.006*	0.000001
Benzene (6hr)	1300	*0.17*	0.0001
Acetaldehyde (24 hr)	2000	*0.085*	0.00004
Benzaldehyde	900	0.00017	0.0000002
Crotonaldehyde	86	0.002	0.00002
Formaldehyde (30 mins)	100	*1.0*	0.01
Isobutyraldehyde	7400	0.024	0.000003
Propionaldehyde	4800	0.012	0.000003
o-Tolualdehyde	86	0.0014	0.00002
p-Tolualdehyde	86	0.0022	0.00003
Arsenic (4hr)	0.19	*0.000009*	0.00005
Cadmium	-	0.000022	-
Chromium	-	0.000016	-
Copper	100	0.000027	0.0000003
Lead	-	0.00009	-
Manganese	-	0.001	-
Mercury	1.8	0.000052	0.00003
Nickel	6	0.000079	0.00001
Selenium	-	0.000019	-
Hazard Index (with PM_{10} only)			**0.7**
Hazard Index (with $PM_{2.5}$ only)			**0.8**

BACKGROUND ONLY			
Pollutant	**AGV ug/m3**	**GLC ug/m3**	**HQ**
NO_2	226	-	
SO_2	262	-	
CO	11000	-	
PM_{10} (24hr)	50	79.2	1.6
$PM_{2.5}$ (24hr)	25	58.9	2.4
PAH	-	-	
Dioxin/Furan	-	-	
TVOCs	-	-	
Ethylbenzene	43400	-	
Phenol	5800	-	
Styrene	21000	-	
Toluene (6 hrs)	15070	-	
Xylene (30 mins)	4340	-	
Benzene (6hr)	1300	-	
Acetaldehyde (24 hr)	2000	-	
Benzaldehyde	900	-	
Crotonaldehyde	86	-	
Formaldehyde (30 mins)	100	-	
Isobutyraldehyde	7400	-	
Propionaldehyde	4800	-	
o-Tolualdehyde	86	-	
p-Tolualdehyde	86	-	
Arsenic (4hr)	0.19	-	
Cadmium	-	-	
Chromium	-	-	
Copper	100	-	
Lead	-	-	
Manganese	-	-	
Mercury	1.8	-	
Nickel	6	-	
Selenium	-	-	
Hazard Index (with PM_{10} only)			**1.6**
Hazard Index (with $PM_{2.5}$ only)			**2.4**

MAX BPS + BACKGROUND			
Pollutant	**AGV ug/m3**	**GLC ug/m3**	**HQ**
NO_2	226	64.3	0.3
SO_2 (15 mins)	262	*96*	0.4
CO	11000	4.9	0.0004
PM_{10} (24hr)	50	82.43	1.6
$PM_{2.5}$ (24hr)	25	62.1	2.5
PAH	-	-	
Dioxin/Furan	-	-	
TVOCs	-	-	
Ethylbenzene	43400	0.006	0.0000001
Phenol	5800	0.01	0.000002
Styrene	21000	0.37	0.00002
Toluene (6 hrs)	15070	*0.13*	0.000008
Xylene (30 mins)	4340	*0.0057*	0.000001
Benzene (6hr)	1300	*0.17*	0.0001
Acetaldehyde (24 hr)	2000	*0.08*	0.00004
Benzaldehyde	900	0.00017	0.0000002
Crotonaldehyde	86	0.002	0.00002
Formaldehyde (30 mins)	100	*1.0*	0.01
Isobutyraldehyde	7400	0.024	0.000003
Propionaldehyde	4800	0.012	0.000003
o-Tolualdehyde	86	0.0014	0.00002
p-Tolualdehyde	86	0.0022	0.00003
Arsenic (4hr)	0.19	*0.000009*	0.00005
Cadmium	-	0.000022	-
Chromium	-	0.000016	-
Copper	100	0.000027	0.0000003
Lead	-	0.00009	-
Manganese	-	0.001	-
Mercury	1.8	0.000052	0.00003
Nickel	6	0.000079	0.00001
Selenium	-	0.000019	-
			-
Hazard Index (with PM_{10} only)			**2.3**
Hazard Index (with $PM_{2.5}$ only)			**3.1**

N.B: Values in italics were adjusted using the Power Rule to match the averaging time of the corresponding air guideline value.

Table A3.2: Calculation of Acute Hazard Indices using the 98th percentile for PM and NO_2, maximum GLCs for all other substances.

BPS			
Pollutant	**AGV ug/m3**	**GLC ug/m3**	**HQ**
NO_2	226	15.6	0.07
SO_2 (15 mins)	262	*7.5*	0.029
CO	11000	4.9	0.0004
PM_{10} (24hr)	50	0.68	0.01
$PM_{2.5}$ (24hr)	25	0.68	0.03
PAH	-	-	
Dioxin/Furan	-	-	
TVOCs	-	-	
Ethylbenzene	43400	0.006	0.0000001
Phenol	5800	0.01	0.000002
Styrene	21000	0.37	0.00002
Toluene (6 hrs)	15070	*0.13*	0.000008
Xylene (30 mins)	4340	*0.0057*	0.000001
Benzene (6hr)	1300	*0.17*	0.0001
Acetaldehyde (24 hr)	2000	*0.085*	0.00004
Benzaldehyde	900	0.00017	0.0000002
Crotonaldehyde	86	0.002	0.00002
Formaldehyde (30 mins)	100	*1.0*	0.01
Isobutyraldehyde	7400	0.024	0.000003
Propionaldehyde	4800	0.012	0.000003
o-Tolualdehyde	86	0.0014	0.00002
p-Tolualdehyde	86	0.0022	0.00003
Arsenic (4hr)	0.19	*0.000009*	0.00005
Cadmium	-	0.000022	-
Chromium	-	0.000016	-
Copper	100	0.000027	0.0000003
Lead	-	0.00009	-
Manganese	-	0.001	-
Mercury	1.8	0.000052	0.00003
Nickel	6	0.000079	0.00001
Selenium	-	0.000019	-
Hazard Index (with PM_{10} only)			0.1
Hazard Index (with $PM_{2.5}$ only)			0.1

BACKGROUND ONLY			
Pollutant	**AGV ug/m3**	**GLC ug/m3**	**HQ**
NO_2	226	-	
SO_2	262	-	
CO	11000	-	
PM_{10} (24hr)	50	29.1	0.6
$PM_{2.5}$ (24hr)	25	21.8	0.9
PAH	-	-	
Dioxin/Furan	-	-	
TVOCs	-	-	
Ethylbenzene	43400	-	
Phenol	5800		
Styrene	21000		
Toluene (6 hrs)	15070		
Xylene (30 mins)	4340		
Benzene (6hr)	1300		
Acetaldehyde (24 hr)	2000	-	
Benzaldehyde	900	-	
Crotonaldehyde	86	-	
Formaldehyde (30 mins)	100	-	
Isobutyraldehyde	7400	-	
Propionaldehyde	4800	-	
o-Tolualdehyde	86	-	
p-Tolualdehyde	86	-	
Arsenic (4hr)	0.19	-	
Cadmium	-	-	
Chromium	-		
Copper	100		
Lead	-		
Manganese	-		
Mercury	1.8		
Nickel	6		
Selenium	-		
Hazard Index (with PM_{10} only)			0.6
Hazard Index (with $PM_{2.5}$ only)			0.9

BPS + BACKGROUND			
Pollutant	**AGV ug/m3**	**GLC ug/m3**	**HQ**
NO_2	226	15.8	0.1
SO_2 (15 mins)	262	*14.4*	0.055
CO	11000	4.9	0.0004
PM_{10} (24hr)	50	29.8	0.6
$PM_{2.5}$ (24hr)	25	22.48	0.9
PAH	-		
Dioxin/Furan	-		
TVOCs	-		
Ethylbenzene	43400	0.006	0.0000001
Phenol	5800	0.01	0.000002
Styrene	21000	0.37	0.00002
Toluene (6 hrs)	15070	*0.13*	0.000008
Xylene (30 mins)	4340	*0.0057*	0.000001
Benzene (6hr)	1300	*0.17*	0.0001
Acetaldehyde (24 hr)	2000	*0.085*	0.00004
Benzaldehyde	900	0.00017	0.0000002
Crotonaldehyde	86	0.002	0.00002
Formaldehyde (30 mins)	100	*1.0*	0.01
Isobutyraldehyde	7400	0.024	0.000003
Propionaldehyde	4800	0.012	0.000003
o-Tolualdehyde	86	0.0014	0.00002
p-Tolualdehyde	86	0.0022	0.00003
Arsenic (4hr)	0.19	*0.000009*	0.00005
Cadmium	-	0.000022	-
Chromium	-	0.000016	-
Copper	100	0.000027	0.0000003
Lead	-	0.00009	-
Manganese	-	0.001	-
Mercury	1.8	0.000052	0.00003
Nickel	6	0.000079	0.00001
Selenium	-	0.000019	-
Hazard Index (with PM_{10} only)			0.7
Hazard Index (with $PM_{2.5}$ only)			1.0

N.B: Values in italics were adjusted using the Power Rule to match the averaging time of the corresponding air guideline value.

Table A3.3: Calculation of Chronic Hazard Indices for the annual average concentration.

BPS only			
Pollutant	**AGV ug/m3**	**GLC ug/m3**	**HQ**
NO_2	58	3.2	0.06
SO_2	52	0.3	0.006
CO	-	2.1	
PM_{10}	20	-	
$PM_{2.5}$	8	-	
PAH	-	0.00099	
Dioxin/Furan	-	6.1E-09	
TVOCs	-		
Ethylbenzene	1300	0.00088	0.0000007
Phenol	200	0.00044	0.000002
Styrene	850	0.054	0.00006
Toluene	300	0.026	0.00009
Xylene	220	0.00071	0.000003
Benzene	60	0.0036	0.00006
Acetaldehyde	-	0.023	
Benzaldehyde	-	0.000024	
Crotonaldehyde	-	0.00028	
Formaldehyde	-	0.12	
Isobutyraldehyde	-	0.00034	
Propionaldehyde	-	0.0017	
o-Tolualdehyde	-	0.0002	
p-Tolualdehyde	-	0.00031	
Arsenic	0.03	0.0000017	0.00006
Cadmium	0.005	0.0000031	0.0006
Chromium III	60	0.0000017	0.00000003
Chromium VI	0.1	0.00000058	0.000006
Copper	1	0.0000039	0.000004
Lead	0.5	0.000013	0.000026
Manganese	0.15	0.00014	0.0009
Mercury	1	0.0000074	0.000007
Nickel	0.05	0.0000011	0.00002
Selenium	20	0.0000027	0.0000001
		Hazard Index =	**0.06**

BACKGROUND ONLY			
Pollutant	**AGV ug/m3**	**GLC ug/m3**	**HQ**
NO_2	58	1.9	0.03
SO_2	52	-	
CO	-	-	
PM_{10}	20	-	
$PM_{2.5}$	8	-	
PAH	-	-	
Dioxin/Furan	-	-	
TVOCs	-	-	
Ethylbenzene	1300	-	
Phenol	200	-	
Styrene	850	-	
Toluene	300	-	
Xylene	220	-	
Benzene	60	-	
Acetaldehyde	-	-	
Benzaldehyde	-	-	
Crotonaldehyde	-	-	
Formaldehyde	-	-	
Isobutyraldehyde	-	-	
Propionaldehyde	-	-	
o-Tolualdehyde	-	-	
p-Tolualdehyde	-	-	
Arsenic	0.03	-	
Cadmium	0.005	-	
Chromium III	60	-	
Chromium VI	0.1		
Copper	1	-	
Lead	0.5	-	
Manganese	0.15	-	
Mercury	1	-	
Nickel	0.05	-	
Selenium	20	-	
		Hazard Index =	**0.03**

BPS + BACKGROUND			
Pollutant	**AGV ug/m3**	**GLC ug/m3**	**HQ**
NO_2	58	5.1	0.09
SO_2	52	0.3	0.006
CO	-	2.1	
PM_{10}	20	-	
$PM_{2.5}$	8	-	
PAH	-	0.00099	
Dioxin/Furan	-	6.1E-09	
TVOCs	-		
Ethylbenzene	1300	0.00088	0.0000007
Phenol	200	0.00044	0.000002
Styrene	850	0.054	0.00006
Toluene	300	0.026	0.00009
Xylene	220	0.00071	0.000003
Benzene	60	0.0036	0.00006
Acetaldehyde	-	0.023	
Benzaldehyde	-	0.000024	
Crotonaldehyde	-	0.00028	
Formaldehyde	-	0.12	
Isobutyraldehyde	-	0.00034	
Propionaldehyde	-	0.0017	
o-Tolualdehyde	-	0.0002	
p-Tolualdehyde	-	0.00031	
Arsenic	0.03	0.0000017	0.00006
Cadmium	0.005	0.0000031	0.0006
Chromium III	60	0.0000017	0.00000003
Chromium VI	0.1	0.00000058	0.000006
Copper	1	0.0000039	0.000004
Lead	0.5	0.000013	0.00003
Manganese	0.15	0.00014	0.0009
Mercury	1	0.0000074	0.000007
Nickel	0.05	0.0000011	0.00002
Selenium	20	0.0000027	0.0000001
		Hazard Index =	0.1