

National Environment Protection (Air Toxics) Measure

# Impact Statement for the National Environment Protection (Air Toxics) Measure

May 2003

# IMPACT STATEMENT FOR AIR TOXICS

GLOSSARY1				
Executive Summary				
1	Introduction	5		
	1.1 Purpose of this National Environment Protection Measure			
	1.2       National Environment Protection Council	6		
	1.3 Purpose of the Impact Statement			
	1.4 Stakeholders			
	1.5 Consultation Strategies	8		
2	Purpose of the Air Toxics NEPM	10		
	2.1 Reasons for Intervention	10		
	2.2 Regulatory Objectives	11		
3	Health Effects of Air Toxics	12		
	3.1 Benzene	12		
	3.2 Formaldehyde	16		
	3.3 Polycyclic Aromatic Hydrocarbons	18		
	3.4 Toluene	21		
	3.5 Xylenes	25		
4	Air Toxics in Australia	27		
	4.1 Benzene	28		
	4.2 Formaldehyde	29		
	4.3 Polycyclic Aromatic Hydrocarbons			
	4.4 Toluene			
	4.5 Xylene	32		
5	Management of Air Torrise in Australia	94		
	Management of Air Toxics in Australia	34		
6	Alternative Methods of Achieving the Desired Environmental Outcome			
		35		
	Alternative Methods of Achieving the Desired Environmental Outcome	<b>35</b> 35		
6	Alternative Methods of Achieving the Desired Environmental Outcome	<b>35</b> 35 <b>38</b> ng		
6	Alternative Methods of Achieving the Desired Environmental Outcome	<b>35</b> 35 <b>38</b> ng 39		
6	Alternative Methods of Achieving the Desired Environmental Outcome	<b>35</b> 35 <b>38</b> ng 39 40		
6	<ul> <li>Alternative Methods of Achieving the Desired Environmental Outcome</li></ul>	<ul> <li><b>35</b></li> <li><b>38</b></li> <li>ng</li> <li>39</li> <li>40</li> <li>41</li> </ul>		
6	<ul> <li>Alternative Methods of Achieving the Desired Environmental Outcome</li></ul>	<b>35</b> 35 38 ng 39 40 41 41		
6	<ul> <li>Alternative Methods of Achieving the Desired Environmental Outcome</li></ul>	<b>35</b> 35 38 ng 39 40 41 41		
6	<ul> <li>Alternative Methods of Achieving the Desired Environmental Outcome</li></ul>	<b>35</b> 35 <b>38</b> ng 39 40 41 41 41 42		
6 7	<ul> <li>Alternative Methods of Achieving the Desired Environmental Outcome</li></ul>	<ul> <li>35</li> <li>35</li> <li>38</li> <li>ng</li> <li>39</li> <li>40</li> <li>41</li> <li>41</li> <li>42</li> <li>42</li> <li>42</li> <li>42</li> </ul>		
6 7	<ul> <li>Alternative Methods of Achieving the Desired Environmental Outcome</li></ul>	<ul> <li>35</li> <li>35</li> <li>38</li> <li>ng</li> <li>39</li> <li>40</li> <li>41</li> <li>41</li> <li>42</li> <li>42</li> <li>42</li> <li>42</li> </ul>		
6 7	<ul> <li>Alternative Methods of Achieving the Desired Environmental Outcome</li></ul>	<ul> <li>35</li> <li>35</li> <li>38</li> <li>39</li> <li>40</li> <li>41</li> <li>41</li> <li>42</li> <li>42</li> <li>42</li> <li>45</li> <li>47</li> </ul>		
6 7	Alternative Methods of Achieving the Desired Environmental Outcome	<ul> <li>35</li> <li>35</li> <li>38</li> <li>39</li> <li>40</li> <li>41</li> <li>41</li> <li>42</li> <li>42</li> <li>42</li> <li>45</li> <li>47</li> <li>47</li> </ul>		
6 7	<ul> <li>Alternative Methods of Achieving the Desired Environmental Outcome</li></ul>	<ul> <li>35</li> <li>35</li> <li>38</li> <li>39</li> <li>40</li> <li>41</li> <li>41</li> <li>42</li> <li>42</li> <li>42</li> <li>45</li> <li>47</li> <li>47</li> </ul>		
6 7	<ul> <li>Alternative Methods of Achieving the Desired Environmental Outcome</li></ul>	<ul> <li>35</li> <li>38</li> <li>39</li> <li>40</li> <li>41</li> <li>42</li> <li>42</li> <li>42</li> <li>45</li> <li>47</li> <li>47</li> <li>53</li> <li>56</li> </ul>		
6 7 8	<ul> <li>Alternative Methods of Achieving the Desired Environmental Outcome</li></ul>	<b>35</b> 38 39 40 41 41 42 42 42 45 47 53 <b>56</b>		
6 7 8	Alternative Methods of Achieving the Desired Environmental Outcome.         6.1       Alternative Approaches         6.1       Alternative Approaches to Evaluating Results of Air Toxics Monitoring	<b>35</b> 35 38 39 40 41 41 42 42 45 47 47 53 <b>56</b> 57		
6 7 8	Alternative Methods of Achieving the Desired Environmental Outcome.         6.1       Alternative Approaches         6.1       Alternative Approaches to Evaluating Results of Air Toxics Monitoring         7.1       Option 1: Standard with Compliance Goal and Specified Monitoring and Reporti Protocol         7.2       Option 2: Advisory Reporting Standard         7.3       Option 3: Investigation Levels         7.4       Preferred Option         7.5       Regional Environmental Differences         Approach to Setting Air Quality Investigation Levels         8.1       Approaches To Setting Air Quality Standards And Guidelines         8.2       Derivation Of Investigation Levels         8.3       Health Endpoints         8.4       Assessment of Overseas Standards         8.5       Proposed Investigation Levels         9.1       Introduction         9.2       Cost Of Desktop Assessment And Monitoring         9.3       Motor Vehicle Impacts	<b>35</b> 35 38 39 40 41 41 42 42 45 47 53 <b>56</b> 57 60		
6 7 8	Alternative Methods of Achieving the Desired Environmental Outcome.         6.1       Alternative Approaches         6.1       Alternative Approaches to Evaluating Results of Air Toxics Monitoring         7.1       Option 1: Standard with Compliance Goal and Specified Monitoring and Reporti Protocol         7.2       Option 2: Advisory Reporting Standard         7.3       Option 3: Investigation Levels         7.4       Preferred Option         7.5       Regional Environmental Differences         Approach to Setting Air Quality Investigation Levels         8.1       Approaches To Setting Air Quality Standards And Guidelines         8.2       Derivation Of Investigation Levels         8.3       Health Endpoints.         8.4       Assessment of Overseas Standards.         8.5       Proposed Investigation Levels.         9.1       Introduction.         9.2       Cost Of Desktop Assessment And Monitoring         9.3       Motor Vehicle Impacts         9.4       Photochemical Smog Impacts	<b>35</b> 35 <b>38</b> 39 40 41 41 42 42 42 45 47 53 <b>56</b> 57 60 62		
6 7 8	Alternative Methods of Achieving the Desired Environmental Outcome.         6.1       Alternative Approaches         6.1       Alternative Approaches to Evaluating Results of Air Toxics Monitoring         7.1       Option 1: Standard with Compliance Goal and Specified Monitoring and Reporti Protocol         7.2       Option 2: Advisory Reporting Standard         7.3       Option 3: Investigation Levels         7.4       Preferred Option         7.5       Regional Environmental Differences         Approach to Setting Air Quality Investigation Levels         8.1       Approaches To Setting Air Quality Standards And Guidelines         8.2       Derivation Of Investigation Levels         8.3       Health Endpoints         8.4       Assessment of Overseas Standards         8.5       Proposed Investigation Levels         9.1       Introduction         9.2       Cost Of Desktop Assessment And Monitoring         9.3       Motor Vehicle Impacts	<b>35</b> 35 <b>38</b> 39 40 41 41 42 42 45 47 53 <b>56</b> 56 57 60 62 63		

9	9.7 Prescribed Burning Impacts	68		
9	9.8 Agricultural Burning Impacts	70		
9	9.9 Cultural Issues			
9	9.10 Waste Burning and Land Development Impacts	71		
9	9.11 Transport and Land Use Planning Impacts			
REFERENCE LIST				
APPEN	NDIX 1 - Air Toxics in Australia	86		
]	Benzene	86		
	Formaldehyde			
]	Polycyclic Aromatic Hydrocarbons(Pahs) Measured As Benzo(A)Pyrene	87		
r	Toluene	89		
2	Xylenes	90		
APPEN	NDIX 2 - Australian Personal Exposure Studies	93		
]	BTEX Personal Exposure Monitoring Study (October 2002)	93		
APPEN	NDIX 3 - Current Jurisdictional Approaches to the Management of Air Toxics			
(	Commonwealth			
]	New South Wales			
	Victoria	99		
(	Queensland	102		
	Western Australia	103		
9	South Australia			
r	Tasmania	105		
1	Australian Capital Territory	106		
]	Northern Territory	107		
APPEN	NDIX 4 –Membership of Air Toxics NEPM Groups	108		

#### 5 GLOSSARY

ADR	Australian Design Rule
AED	Aggregated Emission Data
ANLL	Acute Non-Lymphatic Leukaemia
ANZECC	Australian and New Zealand Environment and Conservation Council
AS	Australian Standard
ATSDR	Agency for Toxic Substances and Disease Registry
BaP	Benzo(a)pyrene
BMD	Bench Mark Dose
CCI	Colour Confusion Index
CEPA	Canadian Environmental Protection Agency
CICAD	Concise International Chemical Assessment Document
CIIT	Chemical Industry Institute of Toxicology
CMA	Chemical Manufacturers Association
CNS	Central Nervous System
EC	European Commission
ECETOC	European Centre for Ecotoxicology and Toxicology of Chemicals
EPA	Environmental Protection Agency
HRA	Health Risk Assessment
IARC	International Agency for Research into Cancer
IPCS	International Program on Chemical Safety
IRIS	Integrated Risk Information Service
JRN	Jurisdictional Reference Network
LOAEL	Lowest Observed Adverse Effects Level
MDS	Myelo Dysplastic Syndrome
MRL	Minimal Risk Level
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
ng/m <sup>3</sup>	Nanogram (1 thousand millionth of 1 gram) per cubic metre
NGO	Non Government Organisations
NHMRC	National Health and Medical Research Council
NICNAS	National Industrial Chemicals Notification and Assessment Scheme
NHL	Non-Hodgkin's Lymphoma
NOAEL	No Observed Adverse Effects Level
NPI	National Pollutant Inventory
NTP	National Toxicology Program
OEHAA	Office of Environmental Health Hazard Assessment
PAH's	Polycyclic Aromatic Hydrocarbons
$PM_{10}$	Particles which have an aerodynamic diameter less than $10\mu m$
ppm / ppb	Parts per million / Parts per billion
PRC	Peer Review Committee
REL	Reference Exposure Level
RfC	Reference Concentration Relative Risk
RR	
SMR TWA	Standardised Mortality Rate
	Time weighted average Micrograms (1 millionth of 1 gram) per cubic metre
µg/m³ UKEPAQS	Micrograms (1 millionth of 1 gram) per cubic metre
US EPA	United Kingdom Expert Panel on Air Quality Standards United States Environment Protection Agency
VOC	Volatile Organic Compound
WA CALM	Western Australia Dept. Conservation and Land Management
WHO	World Health Organization
<b>MIO</b>	

#### 5 EXECUTIVE SUMMARY

In February 2001, NEPC established a Working Group to scope development of a NEPM for Air Toxics. In June 2001 the Working Group reported to Council, recommending 5 priority air toxics to be the subject of the NEPM from a list of 29 identified priority air toxics. In June 2001 NEPC commenced the development of a NEPM for air toxics to address benzene, toluene, formaldehyde, xylenes and polycyclic aromatic hydrocarbons (PAHs).

An Issues Paper was released for public consultation in September 2001. The Issues Paper provided an initial discussion document to scope the issues relating to air toxics and the broad direction that the NEPM might take. It provided discussion around the differences between air toxics and the criteria pollutants addressed through the Ambient Air Quality NEPM. Based on feedback from the Issues Paper a Discussion Paper was released for public consultation in March 2002.

A critical question in the development of the proposed NEPM was "what is the most efficient way to generate improved information about air toxics in Australia?" Air toxics exist at relatively low concentrations in urban airsheds, with significantly elevated levels only occurring near specific sources such as industrial sites, heavily trafficked roads and areas impacted by wood smoke. Therefore it is important that monitoring resources are focussed on such 'peak' sites rather than at generally representative sites and for any standards or 'benchmarks' adopted for the proposed Air Toxics NEPM to apply at these sites.

In the Discussion Paper, various types of standards or 'benchmarks' were reviewed. The options considered for the proposed NEPM were:

- Standard with Compliance Goal and Specified Monitoring and Reporting Protocol;
- 30 Advisory Reporting Standard; and
  - Investigation Levels.

Outcomes from public consultation on the Discussion paper demonstrated:

- support for the development of the NEPM;
- support for a 'peak sites' approach;
  - preference for investigation levels as the most appropriate form of a 'standard';
  - agreement on the adoption of overseas standards, provided they were rigorously assessed for suitability; and
  - support for the identified health end points.
- 40

10

15

Data on levels of the five air toxics dealt with in the NEPM are limited to the extent that it has been judged inappropriate to attempt at this stage to develop any form of national environment protection standards for inclusion in the proposed NEPM. The goal of the proposed NEPM reflects this judgment by focussing on the collection of sufficient data to

- facilitate the development of standards following a review of the Measure within eight years of commencement. In the meantime, it is proposed to incorporate a set of 'investigation levels' in the NEPM. These are in the form of a national environment protection guideline. Under the draft NEPM, each jurisdiction will be required to report annually on progress in meeting the goal of the NEPM. Such reporting will involve: listing of sites identified for
- <sup>50</sup> monitoring; setting out monitoring results; evaluating the nature and significance of any exceedances of the investigation levels; and indicating the nature of any action planned or taken in relation to such exceedances.

- As noted above, the main aim of the Air Toxics NEPM is to facilitate collection of data on the 5 priority air toxics in a nationally consistent manner. The NEPM sets out a 2-stage process for selecting sites for monitoring. This involves firstly a desktop assessment to identify "Stage 1" sites - that is, sites at which significantly elevated levels of one or more of the air toxics are expected to occur. Secondly, a further desktop assessment is undertaken to
- identify "Stage 2" sites that is, those stage 1 sites that are judged to be a priority for monitoring on the basis of the a rapid assessment of the likelihood of significant population exposure to one or more air toxic. Any monitoring undertaken would be done at Stage 2 sites. The desktop identification of sites is to be repeated to identify any additional sites for monitoring no later than four years after commencement of the NEPM.
- 15

The draft NEPM establishes a number of protocols one of which provides guidance on the likely types and sources of data for identifying Stage 1 and Stage 2 sites. In the case of stage 1 sites these including:

- existing monitoring data;
- inventory and modelling data; meteorological and geographical data; and
  - information on seasonal effects.

Information to be taken into account in undertaking a Stage 2 desktop assessment includes:

- estimated levels of the air toxics (both long term and short term);
- size and susceptibility of potentially exposed population; and
  - the nature of the pollutant (eg carcinogen, irritant).

Population data available from the Australia Bureau of Statistics will assist jurisdictions to make a general assessment of population demographics.

30

40

Where a Stage 2 site is an industrial point source subject to jurisdictional control, the jurisdiction may elect not to conduct NEPM monitoring. Such cases will be reported under the reporting requirements of the NEPM.

- <sup>35</sup> The NEPM also establishes a monitoring protocol which defines reference methods for monitoring of the 5 air toxics addressed in the NEPM. Reference methods for monitoring at Stage 2 sites are as follows:
  - For benzene, toluene and xylenes, USEPA method TO14a or TO15 is to be used.
  - For formaldehyde, USEPA method TO11A is to be used (however, TO15 can be used as an alternative for formaldehyde but must be validated).
  - For benzo(a)pyrene (as a marker for PAHs) USEPA methods TO13A must be used.

Where monitoring is to take place at a Stage II site, sampling is to be undertaken once every six days for an entire year, or,

- For benzene and benzo(a)pyrene, 30 samples per season over 2 seasons one in three day sampling.
  - For formaldehyde, toluene and xylenes, 30 samples collected over a sufficient timeframe to include periods of maximum concentration one in three day sampling.
- 50 Finally, the NEPM includes a reporting protocol to be used for annual reporting to NEPC. In the first year only the results of the desktop assessment are required to be reported to NEPC. The results of any monitoring undertaken may also be reported. More specifically, reporting must include:
  - identification of Stage 1 and Stage 2 sites (required in the first year); Reporting of results of monitoring;

- reporting on assessment of any exceedances of investigation levels and any management actions undertaken and planned;
  - identification of additional Stage 1 and Stage 2 sites (required in year 4).

As noted above, the draft NEPM includes investigation levels that have been incorporated into a guideline. These values are intended to assist jurisdictions in the interpretation of monitoring data and to evaluate the nature and extent of any risk to the health of the communities in the areas around Stage 2 sites.

The investigation levels that have been adopted from overseas standards/ guidelines. For
formaldehyde, toluene and xylenes the California EPA, Office for Environmental Health
Hazard, Acute Reference Exposure Levels have been adopted and adjusted to a 24-hour
averaging time. For benzene, the European Commission standards have been adopted and
the New Zealand guideline for BaP (as a marker of PAHs) has been adopted. The
investigation levels for benzene and BaP are both in the form of an annual average, reflecting
the nature of the health concern associated with these compounds ie. risk of cancer.

The proposed NEPM deals only with the monitoring and assessment of levels of air toxics by governments. Consequently, the direct costs which will flow from the adoption of the NEPM (those associated with undertaking desktop assessments and with monitoring) will be incurred only by governments. Jurisdictions will need to assess the most cost-effective means of complying with the monitoring protocol.

While there are no direct impacts other than for governments, there may be a range of indirect impacts where NEPM monitoring prompts additional action by jurisdictions to deal
with particular emission sources. In this context, it should be noted that, jurisdictions already have a variety of programs in place to control industrial emissions and reduce emissions from domestic wood heating. More stringent national fuel quality standards and new Australian design rules for motor vehicles have been introduced to reduce emissions from the motor vehicle fleet and States and Territories conduct in-service motor vehicle programs

- designed to ensure vehicles meet the relevant emissions standards. In the case of emissions from industry and motor vehicles, there is sound evidence that such programs have already achieved some measure of success (for example in reducing emissions of volatile organic compounds such as benzene from some key industrial sources).
- <sup>40</sup> It is expected that monitoring carried out under the proposed NEPM will help jurisdictions assess the effectiveness of existing programs and, where appropriate, adjust programs or develop new ones. Consequently, while there are no direct impacts of this NEPM on industry, such new or changed jurisdictional programs may lead to indirect impacts. However, one would expect that the nature and potential extent of such effects relative to likely benefits would be taken into account by inridictions in deciding on particular new or
- <sup>45</sup> likely benefits would be taken into account by jurisdictions in deciding on particular new or changed programs.

There are many benefits of the proposed NEPM for air toxics. As previously stated there are very few data available in Australia for the air toxics addressed. The proposed NEPM will provide a sound basis for the generation of a significantly improved national dataset that will greatly improve jurisdictional ability to assess the extent and nature of any problems in relation to levels of these air toxics. The proposed NEPM will therefore provide information that will assist governments in setting priorities for various air quality management programs.

55

50

25

Impact Statement for the National Environment Protection (Air Toxics) Measure

- <sup>5</sup> By establishing monitoring and reporting protocols for air toxics and investigation levels against which the results of monitoring can be assessed, the proposed NEPM will provide a much improved information base for use in communicating with the public on important air quality issues and for engaging the community in a more informed discussion of priorities and of the effectiveness of air quality management programs. It will also provide a sound
- 10 database for future studies on the nature and significance of risks posed by these air toxics to the health of the Australian population. Overall, the adoption of the proposed NEPM should, through time, prove to be a significant step towards improved protection of public health.

### 15 **1 INTRODUCTION**

35

#### 1.1 PURPOSE OF THIS NATIONAL ENVIRONMENT PROTECTION MEASURE

The purpose of the National Environment Protection (Air Toxics) Measure is:

- to provide a nationally consistent framework for the monitoring and reporting on air toxics;
- to provide information that will enable NEPC to establish national air quality standards in the future which are protective of human health; and
  - to enable jurisdictions to assess air quality in a consistent manner.

Currently, there are no nationally consistent ambient air quality standards for air toxics in Australia. The guideline in the draft NEPM incorporates investigation levels for each of the following toxic compounds found in the air ("air toxics"): benzene, formaldehyde, benzo(a)pyrene as a marker for polycyclic aromatic hydrocarbons (PAHs), toluene and xylenes. These are designed to provide nationally consistent benchmarks to assess the results of monitoring data generated under the proposed NEPM.

- <sup>30</sup> The desired environmental outcome of this Measure is to facilitate management of air toxics in ambient air that will allow for the equivalent protection of human health and well being, by:-
  - 1) providing for the generation of comparable, reliable information on the levels of toxic air pollutants ("air toxics") at sites where significantly elevated concentrations of one or more of these pollutants are likely to occur ("Stage 1 sites") and where the potential for significant population exposure to air toxics exists ("Stage 2 sites").
    - 2) establishing a consistent approach to the identification of such sites for use by jurisdictions.
- a establishing a consistent frame of reference ("investigation levels") for use by
   jurisdictions in assessing the likely significance of levels of air toxics measured at Stage 2 sites.
  - 4) adopting a nationally consistent approach to monitoring air toxics at a range of locations (eg. major industrial sites, major roads, areas affected by wood smoke).
- <sup>45</sup> The implementation of the Air Toxics NEPM would allow jurisdictions to flexibly implement monitoring, as resources become available. The goal of the proposed NEPM is to improve the information base regarding ambient air toxics within the Australian environment in order to facilitate the development of standards through a health risk assessment following a review of the NEPM within eight years of its making.

### 5 1.2 NATIONAL ENVIRONMENT PROTECTION COUNCIL

The *National Environment Protection Council Acts* of the Commonwealth, States and Territories establish the National Environment Protection Council (NEPC), which comprises Ministers representing each of the participating governments. The NEPC is empowered by the Acts to develop and make National Environment Protection Measures (NEPMs).

10

The Acts provide for the development of NEPMs that relate to a specific set of environmental matters listed in section 14 of the Acts, and require that each NEPM must comprise one or more of a standard, goal, guideline or protocol. The object of the Acts is to ensure that all Australians enjoy the benefits of equivalent protection from air, water, soil and noise pollution, and that business decisions are not distorted nor markets fragmented by variations in major environment protection measures between participating governments.

15

20

40

Once a NEPM has been finalised, it is then formally "made" by NEPC. A decision to make a NEPM requires a two-thirds majority of members of NEPC. NEPMs are implemented by the jurisdictions that participate in the Council within their own jurisdictional legal frameworks.

#### **1.3 PURPOSE OF THE IMPACT STATEMENT**

In making a NEPM, NEPC must have regard to the following matters (section 15 of the NEPC Acts):

- consistency with the Inter-Governmental Agreement on the Environment (IGAE) 1992;
  - environmental, economic, and social impacts;
  - relevant international agreements; and
  - any regional environmental differences.
- <sup>30</sup> Prior to making a NEPM, a draft NEPM and an Impact Statement must be prepared. The Impact Statement must include (section 17 of the NEPC Acts):
  - a) the desired environmental outcomes;
  - b) the reason for the proposed measure and the environmental impact of not making the measure;
- 35 c) a statement of the alternative methods of achieving the desired environmental outcomes and the reasons why those alternatives have not been adopted;
  - d) an identification and assessment of the economic and social impact on the community (including industry) of making the proposed measure;
  - e) a statement of the manner in which any regional environmental differences in Australia have been addressed in the development of the proposed measure;
  - f) the intended date for making the proposed measure; and
  - g) the timetable (if any) in relation to the proposed measure.

The NEPC Acts require that both the draft NEPM and the Impact Statement be made available for public consultation for a period of at least two months. NEPC must have regard to the Impact Statement and submissions received during public consultation in deciding whether to make the NEPM.

An Impact Statement relating to a proposed NEPM should address the impacts of the proposed actions or program, demonstrate that the proposal is justified, and provide a reasonable basis for informed comment by stakeholders and the community.

The proposal under consideration in this Impact Statement is the development of the NEPM 5 for air toxics, which will target five priority air toxics, namely: benzene, formaldehyde, benzo(a)pyrene as a marker for Polycyclic Aromatic Hydrocarbons (PAHs), toluene and xylenes.

#### 1.4 **STAKEHOLDERS** 10

Air pollutants, such as the air toxics under consideration, can cause significant adverse effects on human health if they are present in air at sufficient concentrations and for a sufficient length of time. Monitoring to characterise air quality and population exposure is to be conducted in areas identified in accordance with the selection criteria established under the proposed NEPM.

15

20

40

'Stage 1 sites' are areas in which significantly elevated levels of any of the air toxics under consideration could reasonably be expected (eg areas significantly affected by emissions of these pollutants from a variety of sources such as heavily trafficked roads, industrial and domestic sources).

Stage 2 sites are those Stage 1 sites prioritised on the basis of the likelihood of significant population exposure to one or more air toxic. In this context, assessment of the significance of population exposure needs to take into account factors such as population density and the presence of land uses (such as kindergartens, schools, age care facilities etc.) which are indicative of the presence in the area of sensitive groups. There is no threshold population

- 25 size associated with the expression "significant population exposure" nor is it in any way related to the threshold population value of 25,000 established in the NEPM for Ambient Air Quality in relation to Performance Monitoring Stations.
- A monitoring protocol has been established under the proposed NEPM and provides a 30 framework for monitoring for the priority air toxics, including identification of appropriate sampling and analytical techniques.

The proposed NEPM will introduce investigation levels for air toxics that will act as benchmarks against which the quality of the ambient air at Stage 2 sites can be assessed. 35

In both urban and rural environments, there are a large number of sources contributing to levels of the air toxics under consideration in ambient air. They include: motor vehicles; lawn mowers; domestic and commercial solid fuel burning; service stations; recreational and commercial boating; fires; iron and steel manufacturing; petroleum refining; mineral/metal/chemical manufacturing and wholesaling; aeroplanes; architectural surface coatings and a number of other sources. The management of emissions from these sources will assist in meeting the proposed NEPM investigation levels.

- The stakeholders who may be affected by the proposed NEPM are: 45
  - Communities living in areas in which significantly elevated levels of these pollutants could reasonably be expected;
  - State and Territory government agencies responsible for monitoring and reporting under the NEPM:
- Government and private sector agencies responsible for dealing with the adverse health 50 effects of air pollution;

- State and Territory government agencies responsible for the management and control of point source emissions;
  - Companies/industries which are major sources of emissions of the air toxics under consideration;
  - State and Territory government agencies and local government authorities responsible for transport and land use planning;
  - Government and industry bodies responsible for motor vehicle engine design and fuel quality standards;
  - State and Territory government agencies responsible for fire risk management;
  - Manufacturers of solid fuel heaters; and
- Members of the community as consumers of products emitting air toxics under consideration such as diesel and petrol fuelled motor vehicles, lawn mowers and solid fuel heaters.

# **1.5 CONSULTATION STRATEGIES**

10

#### 20 1.5.1 Process leading to the decision to develop the NEPM

In February 2001 NEPC resolved to establish a working group to scope the development of an Air Toxics NEPM. This working group focused on identifying air toxics for which sufficient information (including ambient air monitoring data) was available, so that a NEPM incorporating ambient standards could be developed. The working group also identified

- <sup>25</sup> other priority air toxics for which further information needs to be obtained to enable their consideration at a later date. This phased approach allowed progress on the most important priority pollutants while also providing a mechanism for addressing other priority air toxics in the future.
- <sup>30</sup> In April 2001 NEPC held a meeting with stakeholders from industry, academic, health, medical and environment groups. Participants provided advice on a range of issues such as the need for a national approach, criteria for identifying priority pollutants and how standards for ambient air toxics could be developed.
- <sup>35</sup> In June 2001 the working group reported its findings to the NEPC, which decided to develop a NEPM addressing five pollutants, namely benzene, formaldehyde, polycyclic aromatic hydrocarbons, toluene and xylenes.
- The NEPC nominated Victoria as the sponsoring jurisdiction for the proposed NEPM. A Project Team (comprising representatives from the Commonwealth, New South Wales, Victoria, Queensland, and South Australia) was subsequently established to develop the proposed NEPM.
- To provide advice to the Project Team and to coordinate jurisdictional input a Jurisdictional Reference Network (JRN) was established with a representative from each State and Territory and the Commonwealth. JRN members have prime carriage for jurisdictional public consultation strategies.
- To facilitate consultation with non-government stakeholders, a Non-Government 50 Organisation (NGO) Advisory Group was established comprising key health, scientific, environment, community and industry representatives.

<sup>5</sup> Throughout the development of the proposed NEPM there has been close consultation with representatives of the health sector in Australia. This process has assisted both in the identification of the appropriate health endpoints for use in the proposed NEPM and in the approach that has been adopted to develop the investigation levels for the air toxics under consideration.

10

# 1.5.2 Issues Paper

The first step in the NEPM development process was the preparation of an Issues Paper by the Project Team. This paper was released for stakeholder comment in September 2001. The purpose of the Issues Paper was to identify, early in the process, issues that would need to be addressed during development of the NEPM. Issues raised in the paper were considered at the meetings of the NGO Advisory Group and the JRN in September 2001. The paper was also placed on the NEPC website and comment on it was sought from a broad range of stakeholders. Fourteen written submissions were received. These submissions were subsequently reviewed and considered during the development of the discussion paper.

20

15

# 1.5.3 Discussion Paper

The next step in the development process was the preparation of a Discussion Paper *"Towards a National Environment Protection (Ambient Air Toxics) Measure"*, which was released in March 2002. The primary purpose of the Discussion Paper was to elaborate on standards considered for the NEPM (and a process for setting them), and to establish the basis for consideration of an option acceptable to the majority of stakeholders. The NGO Advisory Group and the JRN considered the Discussion Paper in April 2002. The Discussion Paper was also placed on the NEPC website and comment was sought from a broad range of stakeholders. 24 written submissions were received.

30

25

The Discussion Paper and a summary of submissions received by NEPC in relation to the Discussion Paper are available on the NEPC website <u>www.ephc.gov.au</u>

# 1.5.4 Draft NEPM and Impact Statement

- <sup>35</sup> Following analysis of the comments received on the Discussion Paper, the Project Team commenced preparation of the draft NEPM and Impact Statement. This phase included consultation with individuals with recognised health and air monitoring expertise. Their inputs were relevant to:
  - identification of the health endpoints to be assessed in the standard setting process;
- approaches to setting standards for the air toxics under consideration;
  - development of appropriate selection criteria for identifying sites; and
  - identification, review and recommendation of appropriate monitoring methods for the air toxics under consideration.
- <sup>45</sup> The NGO Advisory Group, JRN and the health and monitoring experts assisted in the refinement of the approach adopted in the proposed NEPM and Impact Statement.

# 1.5.5 Finalising the draft NEPM

NEPC has agreed to release the proposed NEPM and Impact Statement for the statutory public consultation required by the NEPC Acts. These documents may be downloaded from the NEPC website at *www.ephc.gov.au*.

- <sup>5</sup> The views of stakeholders will be sought through meetings to be held in capital cities during the consultation period, and written submissions are welcome. NGO Advisory Group and JRN views will also be sought. The meeting program will be available from the NEPC website shortly after the commencement of the public review period.
- <sup>10</sup> Following completion of the consultation process, the draft NEPM will be reviewed and, if appropriate, amended taking into account comments received. It is anticipated that the NEPC will consider 'making' the NEPM at its meeting in October 2003.

# 1.5.6 Making a submission

15 The NEPC encourages you to provide comment on the draft NEPM and Impact Statement. Written submissions should be sent to:

Ms Kerry Scott Project Manager – Air Toxics NEPC Service Corporation

20 NEPC Service Corporation Level 5, 81 Flinders Street ADELAIDE SA 5000

Telephone: (08) 8419 1202 25 Facsimile: (08) 8224 0912 Email: kscott@ephc.gov.au

The closing date for submissions is 30 July 2003.

30 All submissions are public documents unless clearly marked "confidential" and may be made available to other interested parties, subject to Freedom of Information Act provisions.

# 1.5.7 Form of Submission

An electronic form for lodging comments is available. The form can be emailed to you by the NEPC Service Corporation or downloaded from the NEPC website (*www.ephc.gov.au*). The completed form can be submitted electronically. Consideration of your submission will be facilitated if it is provided, if possible, in this format.

Should you wish to provide your comments in another format, submissions may be made:

- in hardcopy;
  - on a 3.5 inch floppy disk; or
  - emailed to kscott@ephc.gov.au.

Hardcopy submissions should be unbound to allow ease of photocopying. Electronicsubmissions should preferably be provided in Microsoft Word format.

# 2 PURPOSE OF THE AIR TOXICS NEPM

# 2.1 **REASONS FOR INTERVENTION**

The effects of the air toxics benzene, toluene, xylenes, formaldehyde and PAHs on health are well documented (see Section 3). The results of these studies have shown that these air toxics are associated with a variety of adverse health impacts including cancer, central nervous system (CNS) effects, respiratory irritation and eye irritation.

5 At present, the national database of ambient monitoring data for air toxics is limited. This makes it impractical to compile meaningful estimates of population exposures and health risks associated with current ambient levels of air toxics in Australia. Furthermore, there is no consistently applied methodology for monitoring and reporting on air toxics in ambient air in Australia, and there are no national ambient air quality standards against which the quality of ambient air can be assessed.

It is intended to address these issues through the proposed NEPM by standardising the collection and reporting of ambient air quality data for the nominated air toxics and providing benchmarks against which ambient air quality data can be assessed.

15

# 2.2 **REGULATORY OBJECTIVES**

The objectives of the proposed NEPM are to:

- facilitate collection of monitoring data for ambient air toxics in order to inform future risk assessments and the development of standards;
- establish a set of investigation levels which can be applied nationally to the air toxics benzene, toluene, xylenes, formaldehyde and benzo(a)pyrene as a marker for PAHs, as benchmarks against which the quality of ambient air can be assessed; and,
  - Establish nationally agreed methodologies for determining appropriate locations for monitoring these air toxics, conducting monitoring, and reporting results of monitoring.

25

50

The goal of the proposed NEPM is to "improve the information base regarding ambient air toxics within the Australian environment in order to facilitate the development of standards following a Review of the NEPM within eight years of its making".

- <sup>30</sup> The proposed NEPM establishes processes that include a desktop assessment process to be used by jurisdictions to determine locations where significantly elevated ambient concentrations are more likely to occur (these locations are referred to as "Stage 1 sites"). Jurisdictions may then prioritise their monitoring activities at sites ("Stage 2 sites") based on the results of a further desktop assessment focussing on the likely extent and significance of human exposure, and on available resources
- 35 human exposure, and on available resources.

Jurisdictions monitoring for air toxics under the proposed NEPM will be able to assess the results of monitoring air quality at their sites using the investigation levels as benchmarks. If jurisdictions find that ambient concentrations at Stage 2 sites are above investigation levels, then they will investigate the reasons for and the simulations are supported by the second seco

<sup>40</sup> then they will investigate the reasons for, and the circumstances surrounding, the significantly elevated concentrations. Based on the results of the investigations, jurisdictions may undertake further investigation or implement appropriate management actions.

Under the proposed NEPM, jurisdictions will be required to report to NEPC:

- the results of desktop assessments;
  - locations of Stage 1 and Stage 2 sites
  - the extent of monitoring undertaken or planned;
  - the results of any monitoring;
  - sites where ambient concentrations exceed the investigation levels;
  - the number of exceedances of the investigation levels at Stage 2 sites;
  - the results of investigations into these exceedences; and

• any action taken in response to results of investigations.

By establishing monitoring and reporting protocols, associated desktop procedures and investigation levels for these air toxics, the proposed NEPM will provide a tool for:

- collecting comparable data on concentrations of these air toxics in ambient air in Australia;
  - communicating information to the community on ambient air quality related to these air toxics;
  - assessing the effectiveness of current air quality management programs in managing these air toxics.
- 15

20

25

10

Together these outcomes should in turn facilitate more cost effective programs, better priority setting by governments, improvements in infrastructure development planning, more informed choices by individuals and consequential reduction in the risk of adverse health effects.

# **3 HEALTH EFFECTS OF AIR TOXICS**

#### 3.1 BENZENE

The adverse health effects of benzene exposure have been assessed by numerous agencies NICNAS (2001); WHO (2000); International Program on Chemical Safety, (1993); Commission of European Communities, (1998); United Kingdom Expert Panel on Air Quality Standards, (1994); US EPA (2000); Environment Canada (1993).

The critical human health effects from long term exposure to benzene are bone marrow depression and leukaemia, specifically acute non-lymphocytic leukaemia (also known as acute myeloid leukaemia). Benzene is classified as a known human carcinogen. It is considered to be a genotoxic carcinogen for which no threshold has been established. (NICNAS 2001, US EPA 2000, WHO 2000)

There are four key occupational cohort studies demonstrating an association between benzene and an increase in the incidence of leukaemia for which the exposures have been assessed in detail. These are the Goodyear Pliofilm (Rinsky et al, 1981), the Chemical Manufacturers Association (CMA), (Wong, 1987a, 1987b), Dow Chemical (Bond et al, 1986) and the Chinese Factory Worker (Hayes et al, 1997) cohorts. Most assessments have considered that the analyses of the Goodyear Pliofilm study has given them the most robust database on which to base their assessments of benzene concentrations associated with development of leukemia and the degree of risk.

Most of the human health-exposure data have been obtained from retrospective epidemiological studies relating to occupational settings. It is accepted that there are difficulties in relating these studies usually in fit, healthy adults to the population in general, which consists of all ages and various levels of health and infirmity.

# 3.1.1 The Goodyear Pliofilm cohort

An excess incidence of leukaemia in rubber workers at two Goodyear facilities in Ohio, USA was reported in a preliminary paper by Infante et al. (1977) and in more detail by Rinsky et al. (1981). Depending on its definition, this cohort comprises 1165-1212 male workers employed from 1936-75 in the manufacture of Pliofilm. The manufacturing process used large volumes of benzene as a solvent and there was no exposure to other known

50

carcinogenic substances. Excluding deaths before 1950, Rinsky et al. (1987) identified 15 deaths from lymphatic and haematopoietic cancers versus 6.6 expected (Standardised Mortality Rate {SMR} = 2.27 {1.27- 3.76}) and 9 deaths from leukaemia versus 2.7 expected (SMR = 3.37 {1.54-6.41}).

# 10 3.1.2 The Chemical Manufacturers Association (CMA) cohort study

This is a study of 4602 male chemical workers who were employed for  $\geq 6$  months from 1946-75 at 7 US plants (Wong, 1987a, 1987b). Two comparison groups were used: the general US population and 3074 unexposed male workers employed at the same plants at the same time as the cohort. The vital status of all subjects was followed until the end of 1987 and the findings compared to average and peak exposures as determined from available air monitoring data and employment records obtained from the participating companies. There were 19 deaths from cancer of the blood and lymphatic system in the exposed workers compared to 3 in the unexposed group. In the exposed group, 7 of the observed cases were diagnosed with leukaemia and the remaining 12 with lymphoma. In the unexposed workers, all 3 cases were diagnosed with lymphoma, there were no cases of leukaemia in the unexposed workers. The SMRs for all cancers of the blood and lymphatic system were 0.91, 1.47, and 1.75, and for leukaemia 0.97, 0.78 and 2.76 for cumulative exposures of less than180,

180-719 or  $\geq$ 720 ppm-months respectively, but none of the ratios was significantly different<br/>from unity. The trend for all cancers of the blood and lymphatic system was significant (p =<br/>0.02), and (p = 0.01) for leukaemia for trend with cumulative exposure.

# 3.1.3 The Dow Chemical cohort

15

20

This study comprised 956 male chemical workers employed at a single site in Michigan, USA, between 1940 and 1982. The workers were exposed to benzene in chlorobenzene or
alkylation plants which used benzene as a raw material, or in an ethyl cellulose plant where benzene was used as a solvent (Bond et al, 1986; Ott et al, 1978). Each job entry was assigned an exposure intensity level on the basis of job classification and representative personal air monitoring data. There were 6 deaths from cancer of the blood and lymphatic system against 4.8 expected, including 4 cases of myelogenous leukaemia against 0.9 expected. The excess of myelogenous leukaemia was statistically significant (p = 0.011; SMR and 95% CI not stated).

# 3.1.4 US National Cancer Institute (NCI) and Chinese Academy of Preventive Medicine (CAPM) Chinese factory workers cohort study

- A follow up on a large cohort study commenced in 1982 to assess the risks of specific bone marrow disorders in relationship to occupational benzene exposure (Hayes et al, 1997). The final cohort comprises 74,828 male and female benzene-exposed workers employed from 1972 to 1987 in 672 factories in 12 cities in China and 35,805 unexposed workers. Relative risks (RRs) were determined for incident cancer of the blood and lymphatic system, non-
- <sup>45</sup> Hodgkin's Lymphoma (NHL), leukaemia, Acute non-lymphatic leukaemia (ANLL), a diagnosis of either ANLL or Myelo Dysplastic Syndromes (MDS), and leukaemia other than ANLL, with stratification by age and sex. The exposed workers held permanent jobs in the painting, printing, footwear, rubber and chemical industries. Exposure levels were estimated from available area monitoring data, detailed production and process information,
- 50 and employee records.

There were 58 specified cancers of the blood and lymphatic system and 18 other bone 5 marrow disorders (2 cases of agranulocytosis, 9 of aplastic anaemia and 7 of MDS) in the cohort, compared to 13 and 0 respectively in the control group.

When the cohort was divided into three categories, according to the estimated cumulative benzene exposure level, the RR for all cancer of the blood and lymphatic system was 10 elevated from <40 ppm-years 2.2 (1.1-4.5). The RRs for leukaemia was elevated from 40-99 ppm-years 3.1 (1.2-8.0), and ANLL/MDS from 40-99 ppm-years 6.0 (1.8-20.6).

# 3.1.5 Modes of action

Several reviews of benzene metabolism and the proposed mechanisms of toxicity have been 15 published (Ross, 1996; Snyder, 2000; Snyder et al, 1993; Snyder & Hedli, 1996; Yardley-Jones et al, 1991).

Exposure to benzene can result in haematotoxicity, immunotoxicity and carcinogenicity in humans and animals. Haematotoxicity resulting from chronic benzene exposure can present 20 as anaemia, aplastic anaemia, leukopenia, lymphocytopenia, thrombocytopenia, or pancytopenia (Aksoy, 1989). While the liver is the initial site for the biotransformation of benzene, hepatotoxicity is not a consequence of benzene exposure. Subsequently, these metabolites become localised within the bone marrow (Rickert et al, 1979) where they undergo activation by peroxidase enzymes, which are present in bone marrow. While 25 individual benzene metabolites appear not to induce bone marrow toxicity, the combination

of phenol and hydroquinone have been shown to induce the same effects on bone marrow as benzene (Eastmond et al, 1987). This effect appears to be due to the ability of phenol to act as a co-oxidant in the activation of metabolites.

30

35

Subsequent changes in cellular function result in altered growth factor production with inhibition of bone marrow stem cell proliferation, differentiation and maturation. The formation of reactive oxygen species damage cells and result in DNA adduct formation, DNA base modification, chromosomal aberrations that can lead to cellular damage which may result in leukaemia in humans or solid tumours in animals.

# 3.1.6 Non cancer endpoints

# Effects of long term human exposure

- Tsai et al (1983) examined the mortality from all cancers and leukaemia, in addition to haematological parameters in 454 male workers exposed to benzene for 1-21 years in a 40 refinery from 1952-1978. The median air concentration was 0.53 ppm in the work areas of greatest exposure to benzene. The average length of employment in the cohort was 7.4 years. The analysis of overall mortality in this population revealed no significant excesses. A subset of 303 workers was followed for medical surveillance. Up to four haematological tests per year showed all parameters to be within normal limits in this group.
- 45

Collins et al. (1997) used routine data from Monsanto's medical/industrial hygiene system to study 387 workers with daily 8-hour time-weighted exposures (TWA) averaging 0.55 ppm benzene (range = 0.01 – 87.69 ppm; based on 4213 personal monitoring samples, less than 5% of which exceeded 2 ppm). There was no increase in the prevalence of lymphopenia

50

(decreases in lymphocyte numbers), an early, sensitive indicator of benzene toxicity, or other measures of haematotoxicity.

- <sup>5</sup> Rothman et al (1996) studied a small number (44) of Chinese workers heavily exposed to benzene (31 ppm, 2 - 329 ppm range) and showed decreases in white blood cell counts and absolute lymphocyte counts and other blood parameters when compared to matched unexposed controls. In a much smaller subgroup of 11 of the 44 workers, with a recorded lower median exposure to benzene of 7.6 ppm (1 -20 ppm range), only the absolute
- <sup>10</sup> lymphocyte count was decreased compared to the controls. Their results support the use of the absolute lymphocyte count as the most sensitive indicator of benzene-induced haematotoxicity.

#### 3.1.7 Summary of benzene non-cancer health effects

15 The No Observed Adverse Effect Level (NOAEL) for haematotoxicity in humans was established by Tsai et al (1983) at 0.53 ppm, and by Collins et al (1997) at 0.55 ppm, from long-term worker exposure studies, with daily 8 hours exposures, 5 days per week. NICNAS (2001) also conclude NOAELs to be around the 0.5 ppm level and a LOAEL (lowest observed adverse effect level) at 7.6 ppm in a subgroup of 11 exposed workers (Rothman et al 1996).

Although the study by Tsai et al. (1983) is a freestanding NOAEL of 0.53ppm, the endpoint examined is a known sensitive measure of benzene toxicity in humans. The recent results of Collins et al. (1997) that included a NOAEL of 0.55 ppm and of Rothman et al (1996) that included a LOAEL of 7.6 ppm are consistent with those of Tsai et al. (1983).

#### 25

30

#### 3.1.8 Effects of laboratory animal exposure to benzene

A number of animal studies have demonstrated that benzene exposure can induce bone marrow damage Ward et al (1985), Keller and Snyder (1978), changes in circulating blood cells, Aoyama (1986), developmental and reproductive effects Kuna and Kapp (1981), and cancer at multiple organ sites. With respect to long term exposure toxicity, haematological changes appear to be the most sensitive indicator.

#### 3.1.9 Use of health data in setting air quality guidelines and standards.

- <sup>35</sup> The most widely used study as the basis of overseas standards and guidelines is the Goodyear Pliofilm study. NICNAS have used this as the basis for their review of the Occupational Health and Safety Standards in Australia. The WHO, European Commission, UK Expert Panel on Air Quality Standards and the USEPA all use the Pliofilm study as the key study when assessing carcinogenic risk form exposure to benzene.
- 40

45

For purposes of guideline derivation, the WHO decided to use the 1994 risk calculation of Crump (of the Pliofilm cohort) rather than to derive new estimates. The geometric mean of the range of estimates of the excess lifetime risk of leukaemia at an air concentration of 1  $\mu$ g/m<sup>3</sup> is 6 ×10<sup>-6</sup>. Using this unit risk factor, the concentrations of airborne benzene associated with an excess lifetime risk of 1/10 000, 1/100 000 and 1/1 000 000 are 17, 1.7 and 0.17  $\mu$ g/m<sup>3</sup>, (5.3, 0.53, 0.053 ppm) respectively

The European Commission Working Group on Benzene Position Paper (1998) stated that the Goodyear Pliofilm cohort was the most thoroughly studied group. For purposes of guideline derivation, the Working Group chose to use the 1994 risk calculation of Crump (as did

<sup>50</sup> WHO) rather than to derive new estimates. It was considered to result in the highest plausible estimate of risk – an excess lifetime risk of leukaemia at an air concentration of 1  $\mu$ g/m<sup>3</sup> of 6 × 10<sup>-6</sup>.

5 The USEPA used the Goodyear Pliofilm study as reported by Rinsky et al (1981, 1987) for their quantitative risk estimation. They estimated a range of 2.2 × 10<sup>-6</sup> to 7.8 ×10<sup>-6</sup> as the increase in the lifetime risk of an individual who is exposed for a lifetime to 1 μg/m<sup>3</sup> benzene in air. This is based on a linear model and is dependent on which exposure measurements were used (ie Crump and Allen, 1984 or Paustenbach et al. 1992). This unit risk factor extrapolates to air concentrations of 1.3 to 4.5 μg/m<sup>3</sup> for a risk level of 1 in 100,000).

**3.2 FORMALDEHYDE** 

The health effects of formaldehyde have been extensively reviewed (CICAD, 2002; OEHHA, 1999; ATSDR, 1999; WHO, 2000; IARC, 1995). The critical human health effects related to acute exposures are irritation of the eyes and upper respiratory tract.

There is some evidence in animals and humans that formaldehyde has carcinogenic properties. However, unlike benzene and other genotoxic carcinogens, there appears to be a two-stage mechanism for the induction of neoplastic changes induced by exposure to formaldehyde. Repeated irritation to the pasel musces is believed to be the presure to

<sup>20</sup> formaldehyde. Repeated irritation to the nasal mucosa is believed to be the precursor to cellular changes that may lead to carcinogenic effects. Therefore, protecting against the irritative effects of formaldehyde is thought to protect against the more serious carcinogenic effects. The CICAD (2002) document is considered to be the most recent assessment of the toxicity of formaldehyde.

25

30

35

15

# 3.2.1 Irritant effects

There are numerous reports that exposure to formaldehyde causes direct irritation of the respiratory tract. In a number of clinical studies, generally mild to moderate sensory eye, nose, and throat irritation was experienced by volunteers exposed for short periods to levels of formaldehyde ranging from 0.25 to 3.0 ppm (0.30 to 3.6 mg/m<sup>3</sup>) (Andersen & Molhave, 1983; 1986; Kulle, 1993; Pazdrak et al 1993).

There is evidence of formaldehyde inducing pathological and cytogenetic changes in the nasal mucosa of humans in studies with reported mean exposures ranged from 0.02 ppm to 2 ppm, with peaks between 4.2 ppm and 15 ppm. The LOAEL for short-term exposure is 0.08 ppm (WHO/IPCS, 1989).

The studies by Kulle et al (1993; 1987) are key studies used in the development of air quality guidelines and standards. Kulle et al (1987; 1993) exposed healthy subjects to 0, 1.0, and 2.0 ppm for 3-hour periods and asked them to note symptoms of eye and nose/throat irritation

ppm for 3-hour periods and asked them to note symptoms of eye and nose/throat irritation and to rate severity on a 0-3 scale: 0=none; l=mild (present but not annoying); 2=moderate (annoying); and 3=severe debilitating). Ten of the subjects were also exposed to 0.5 ppm and nine were exposed to 3 ppm for 3-hour periods. The frequencies of subjects reporting eye irritation or nose/throat irritation increased with increasing exposure concentration,
especially at concentrations ≥ 1 ppm. Under non-exposed conditions, 3 of the 19 subjects noted mild nose/throat irritation and l noted mild eye irritation. At 0.5 ppm, l of 10 subjects noted mild nose/throat irritation, but none reported eye irritation. Frequencies for subjects with mild or moderate eye irritation were 4 of 19 at 1 ppm (1 moderate), 10 of 19 at 2 ppm (4 moderate), and 9 of 9 at 3 ppm (4 moderate). The increased frequency for eye irritation

<sup>50</sup> (compared with controls) was statistically significant at 2.0 ppm. Frequencies for mild nose/throat irritation were l of 19 at 1 ppm, 7 of 19 at 2 ppm, and 2 of 9 at 3 ppm. Compared with control frequency for nose/throat irritation, only the response at 2 ppm was significantly elevated.

- <sup>5</sup> Weber-Tschopp et al (1977) exposed a group of 33 healthy subjects for 35 minutes to concentrations of formaldehyde that increased during the period from 0.03 to 3.2 ppm; another group of 48 healthy subjects was exposed to 0.03, 1.2, 2.1, 2.8, and 4.0 ppm for 1.5 minute intervals. Eye and nose irritation were reported on a l-4 scale (l=none to 4=strong) in both experiments, and eye blinking rate was measured in the second experiment. Average
- <sup>10</sup> indices of eye and nose irritation were increased in both experiments to a small, but statistically significant 1.2 ppm compared with indices for non-exposed controlled conditions. The published report of this study graphically showed average severity scores of about 1.3-l.4 for both indices at 1.2 ppm compared with 1.0-l.1 for non exposed conditions. The average severity score was increased to a greater degree at higher concentrations, but
- <sup>15</sup> was less than about 2.5 at the highest exposure concentration, 4 ppm. Average rates of eye blinking were not significantly affected at 1.2 ppm, but were statistically significantly increased at 2.1 ppm (about 35 blinks/minute at 2.1 ppm versus about 22 blinks/minutes under non-exposed conditions).
- A study by Pazdrak et al (1993) which included a group of subjects sensitised to formaldehyde, is a further study that has been used in deriving air quality standards and guidelines. However, there have been some concerns raised about the exposure estimates used in this study (OEHHA, 1999). The Pazdrak study investigated the effects of formaldehyde exposure on the severity of symptoms of nasal and eye irritation and the cellular makeup of nasal discharge in occupationally exposed patients with skin
- hypersensitivity to formaldehyde and unexposed controls. The study was comprised of two study groups, all non-smokers. Group 1 consisted of 7 male and 3 female volunteers, all of whom suffered from skin hypersensitivity to formaldehyde; Group 2 consisted of 11 healthy males with no history of allergic diseases, normal serum IgE levels, and negative skin tests to
- 30 common allergens. Nasal washings were performed in both groups immediately before and after a 2-hour exposure to 0 and 0.4 ppm formaldehyde and at 4 and 18 hours after completion of the exposure periods. Symptoms of were evaluated through the exposure period and through 4- and 18-hour periods after the exposure period (maximum score = 7). In both groups, placebo inhalation periods were without effects on nasal wash cellular
- contents or symptom score. During exposure to 0.4 ppm formaldehyde, both groups showed statistically significantly increased average symptom scores compared with average placebo scores (about 4 versus <0.5). Symptom scores were no longer elevated 18 hours after exposure. The authors concluded that the symptoms observed were the result of a non-specific, non-allergic process in response to low-level formaldehyde vapour exposure.</p>

40

A study by Anderson and Molhave (1983) identified an apparent effect level (0.2 ppm), based on subjective reports of irritation that is lower than the effect levels (0.35-0.4 ppm) in the studies by Pazdrak et al (1993), and Bender et al (1993), which used more objective measures of acute irritation.

45

50

Studies in animals confirm that the upper respiratory tract is a critical target for inhaled formaldehyde and describe exposure-response relationships for upper respiratory tract irritation and epithelial damage in several species. Acute inhalation animal studies show that inhaled formaldehyde, at appropriate exposure concentrations, damages epithelial tissue in specific regions of the upper respiratory tract in rats, mice, and monkeys (Chang et al 1983; Monticello et al 1989, 1991).

#### 5 3.2.2 Carcinogenic effects

effects occurs.

Damage to the nasal mucosa, such as squamous cell metaplasia and mild dysplasia of the respiratory epithelium, have been reported in humans, but these findings may have been confounded by concomitant exposures to other substances (IARC 1995). There is also epidemiological evidence of associations between relatively high occupational exposure to formaldehyde and both nasopharyngeal and sinonasal cancers (Blair et al 1990; Partanen 1993; McLaughlin 1994). There is substantial variation in individual responses to formaldehyde in humans. Significant increases in signs of irritation occur at levels above 0.08 ppm in healthy subjects. At concentrations above 1 ppm, a progression of symptoms and

15

20

10

Differing conclusions have been reached regarding the evidence from the epidemiological studies for the carcinogenic effects of formaldehyde. On one side, IARC (1995) and US EPA (1991) judged that there was limited evidence in humans and sufficient evidence in animals and NTP (1998) judged that formaldehyde was reasonably anticipated to be a human carcinogen. McLaughlin (1994) and ECETOC (1995), on the other side, concluded that a causal relationship was not established by the available data. A more recent review of the data by CIIT (1998) appears to take a middle stand concluding, "it appears that a weak association between nasopharyngeal cancer and formaldehyde exposure cannot be completely ruled out".

25

In contrast to the equivocal, limited, or weak nature of the evidence in humans, replicated inhalation studies in laboratory animals have consistently shown that formaldehyde induces nasal tumours in rats exposed to high concentrations (10–15 ppm) as well as nasal epithelial necrosis and cellular proliferation, but not when exposed to lower concentrations (0.3–2 ppm) that do not markedly damage nasal epithelial tissue (Albert et al. 1982; Kamata et al

<sup>30</sup> ppm) that do not markedly damage nasal epithelial tissue (Albert et al. 1982; Kamata et al 1997; Kerns et al 1983; Monticello et al 1996; Wouterson et al 1989). Exposure-related cancer or non-cancer lesions at sites distant from the portal-of-entry were not found in these studies, consistent with the water solubility and reactivity of formaldehyde and the ubiquity of rapid cellular metabolism of formaldehyde.

35

40

Chronic exposure to formaldehyde concentrations ranging from about 6 ppm to 15 ppm induced increased incidences of nasal tumours (squamous cell carcinomas, squamous cell papillomas, or polyploid adenomas) in three bioassays with rats (Kamata et al 1997; Kerns et al 1983b; Monticello et al 1996; Swenberg et al 1980). Increased incidences of lower respiratory tract tumours or distant site tumours were not found in these studies, and exposure to concentrations of 2 ppm and lower induced no malignant nasal tumours.

# 3.2.3 Use of health data in setting air quality guidelines and standards.

For air quality standards and guidelines based on the irritative effects of formaldehyde the
studies by Kulle (1993; 1987) and Pazdrak (1993) appear to be the most widely used. The
LOAEL observed appears to be between 0.08 and 0.1 ppm.

# 3.3 POLYCYCLIC AROMATIC HYDROCARBONS

The health effects of polycyclic aromatic hydrocarbons (PAHs) have been reviewed extensively (EC, 2001; WHO, 2000; OEHHA, 1999; UK EPAQS, 1999; IPCS, 1998; ATSDR, 1995; Env. Canada, 1994). One of the complexities in evaluating the health effects of PAHs is that they exist as a mixture of compounds not individual compounds. The toxicity of these compounds varies quite markedly, with the most toxic being benzo(a)pyrene (BaP), which is classified as a carcinogen.

- <sup>5</sup> There is little information on human exposure to single, pure PAH. That which is available includes reports of accidental exposure to naphthalene and some data from defined short-term studies of volunteers. All other reports are of exposure to mixtures of PAH, which also contained other (non-PAH) potentially carcinogenic chemicals, in occupational and environmental situations.
- 10

15

Several epidemiological studies have shown increased mortality due to cancer, which has been associated with exposure to PAH-containing mixtures in humans exposed to coke oven emissions, roofing-tar emissions, and cigarette smoke. The cancers occur predominantly in the lungs and skin following inhalation and dermal exposure, respectively but can occur in other tissues away from the major route of exposure. It is thus impossible to evaluate the contribution of any individual PAH to the total carcinogenicity of these mixtures in humans because of the complexity of the mixtures and the presence of other carcinogens, and the potential interactions that could occur with other toxic substances in the mixtures. Despite these limitations, reports of this nature provide qualitative evidence of the potential for mixtures containing PAHs such as benzo(a)pyrene, chrysene, benz(a)anthracene,

20 mixtures containing PAHs such as benzo(a)pyrene, chrysene, ber benzo(b)fluoranthene, and dibenz(a,h)anthracene to cause cancer in humans.

The critical endpoint for health risk evaluation is the well-documented carcinogenicity of several PAHs (IARC 1983). BaP is by far the most extensively studied PAH in experimental
animals. It produces tumours of many different tissues, depending on the species tested and the route of application. BaP is the only PAH that has been tested for carcinogenicity following inhalation, and it produced respiratory tract tumours (not lung tumours) in hamsters, the only species tested (Thyssen et al, 1981). Induction of lung tumours in rats and hamsters has also been documented for BaP and several other PAHs following direct application, such as intratracheal instillation into the pulmonary tissue (Deutsch-Wenzel et al, 1983).

The lung carcinogenicity of BaP can be enhanced by co-exposure to other substances such as cigarette smoke, asbestos and airborne particles. Several studies have shown that the benzene-soluble fraction, containing 4 to 7-ring PAHs of condensates from car exhausts, domestic coal-stove emissions and tobacco smoke, contains nearly all the carcinogenic potential of PAHs from these sources (Pott & Heinrich, 1990).

- Because several PAHs have been shown to be carcinogenic, and many more have been shown to be genotoxic in *in vitro* assays, a suitable indicator for the carcinogenic fraction of the large number of PAHs in ambient air is desirable. The most appropriate indicator for the carcinogenic PAHs in air seems to be BaP concentrations, given present knowledge and the existing database.
- <sup>45</sup> The proportion of different PAHs detected in emissions and in workplaces differs widely from each other and from PAH profiles in ambient air. Nevertheless, the profiles of PAHs in ambient air do not seem to differ very much from one area to another, although large variations may be seen under special conditions. Furthermore, the carcinogenicity of PAH mixtures may be influenced by synergistic and antagonistic effects of other compounds
- 50 emitted together with PAHs during incomplete combustion. It should also be recognised that in ambient air, the carcinogenic 4 to 7-ring PAHs (representing the majority of PAHs) are solids and are preferentially attached to particles and only a minor fraction, depending on the temperature, exist as volatiles. A few studies indicate that the toxicokinetic properties of inhaled BaP attached to particles are different from those of pure BaP alone. Virtually 55 nothing is known about other PAHs in this respect.
  - nothing is known about onler 17 mis in this respect.

WHO presented an excess lifetime cancer risk, expressed in terms of the BaP concentration and based on observations in coke oven workers exposed to mixtures of PAHs. It was emphasised that the composition of PAHs to which coke oven workers are exposed may not be similar to that in ambient air.

10

5

The WHO adopted the lung cancer risk estimate calculated by the US Environmental Protection Agency. The US EPA based its calculations on extensive studies of coke oven workers in Pennsylvania. The US EPA used a linearised multistage model.

The unit risk for BaP is estimated to be  $8.7 \times 10^{-5}$  (ng/m<sup>3</sup>)<sup>-1</sup>. The corresponding concentrations of BaP producing excess lifetime cancer risks of 1/10 000, 1/100 000 and 1/1 000 000 are 1.2, 0.12 and 0.012 ng/m<sup>3</sup> respectively.

# 3.3.1 Key studies

- <sup>20</sup> The epidemiological study by Armstrong et al (1994) of lung cancer deaths in men who had worked in an aluminium smelter in Canada is considered a key study into the health effects of PAHs as it addressed the confounder of smoking. In this investigation exposure to BaP as a marker of PAH exposure (benzene soluble coal tar pitch volatiles) was estimated for workers in each type of job within the plant. The heaviest exposure occurred for workers in
- <sup>25</sup> two parts of the process known as 'the pot room' and 'anode manufacture', where BaP concentrations were 20-40µg/m<sup>3</sup>. After adjustment for confounding by cigarette smoking and age, a clear association was found between increased exposure to BaP and lung cancer deaths RR 2.23 (95% CI 1.46-3.39) at 100-199 µg/m<sup>3</sup>-years of BaP.
- Costantino et al (1995) reported a significantly increased risk for lung cancer (SMR, 1.95 with 95% CI of 1.59-2.33) among a cohort of over 5000 workers who were heavily exposed at coke ovens in coke plants and were followed-up for over 30 years. The authors concluded that 124 deaths from lung cancer occurred among these coke-oven workers that could be attributed to exposure to coal-tar pitch volatiles, (2.3% of the cohort). Although no data were available on smoking habits, the observed effect is not likely to be due to smoking since unexposed steel
- workers in a comparison group were assumed to have similar smoking habits. In addition, a high correlation was seen between the risk for respiratory cancer and the concentration and duration of exposure.

The respiratory health of 667 workers in a rubber factory was investigated (Gupta et al 1993). Respiratory health was evaluated and examined for correlations to length of employment at the factory. In addition, total suspended particulate matter and benzo(a)pyrene concentrations were monitored in various parts of the factory and examined for possible correlation with the respiratory health of the workers in the same area of the factory. Statistically significant decrements in ventilation function occurred following prolonged exposure as assessed by duration of employment. When different sections of the factory were considered, workers in the compounding section were the most affected, which was associated with the highest exposure to particulate matter and benzo(a)pyrene. Workers in the compounding section exhibited radiographic abnormalities including patch opacities,

<sup>50</sup> prominent bronchiovascular markings, and pleural effusions. Other symptoms included bloody vomit, breathing problems, chest pains, chest irritation, throat irritation, and cough. Workers in other areas of the plant exposed to lower levels of particulate matter and benzo(a)pyrene were similarly affected although to a lesser degree and in fewer numbers. 5 No attempt was made to separate the effects of exposure to benzo(a)pyrene and particulate matter, or to identify possible simultaneous exposure to other toxic chemicals.

# 3.3.2 Effects on laboratory animals

The carcinogenic effects of exposure to PAHs by inhalation have been examined in only a few limited identified studies, all of which were restricted to BaP (Thyssen et al 1981; Heinrich et al 1986; Laskin et al 1970). Moreover, in two of the investigations, animals were concomitantly exposed to other compounds (Heinrich et al 1986; Laskin et al 1970). In the study by Heinrich et al. (1986), the incidence of lung tumours was increased in rats exposed to combustion gases of a coal furnace for an average of 16 hours/day, 5 days/week over a maximum of 22 months. The incidence of respiratory tract tumours was also increased in rats that inhaled 10 ppm (103 mg/m<sup>3</sup>) BaP and the atmospheric irritant, sulfur dioxide (Laskin et al., 1970).

Benzo(a)pyrene has been tested in a range of species, including rats, guinea pigs, rabbits, marmosets, and rhesus monkeys. Tumours have been observed in all experiments with small animals, and the failure to induce neoplastic responses in large animals has been attributed to lack of information on the appropriate route or dose and the inability to observe the animals for a sufficient time (Osborne & Crosby, 1987). In studies with other PAHs, BaP was often used as a positive control and therefore administered at only one concentration.

25

BaP has been shown to be carcinogenic when given by a variety of routes, including diet, gavage, inhalation, intratracheal instillation, intraperitoneal, intravenous, subcutaneous, and intrapulmonary injection, dermal application, and transplacental administration. The carcinogenicity of individual PAH and PAH-containing mixtures in experimental animals

has been well studied. Virtually no data exist on the carcinogenicity of individual PAHs in humans, although a limited database on the carcinogenicity of PAH-containing mixtures is available: these have been shown to increase the incidence of cancer in occupationally exposed human populations. The finding that a number of individual PAH are carcinogenic to experimental animals indicates that they are potentially carcinogenic to humans. PAH can produce tumours both at the site of contact and distantly, and the carcinogenic potency of

PAH may vary with the route of exposure.

# 3.3.3 Use of health data in setting air quality guidelines and standards

The study by Armstrong et al (1994) of Canadian aluminium smelter workers is the one most often used by agencies in developing air quality standards and guidelines. The US EPA unit risk factor is the most commonly used and has been adopted by the WHO.

# 3.4 TOLUENE

45

The adverse health effects of toluene exposure have been assessed by numerous agencies including WHO (2000); USEPA, IRIS (2002); OEHHA CEPA (1999); US Department Health and Human Services ATSDR (2000); Health and Environment Canada (1992).

The critical health endpoint following short-term exposure of humans to toluene is respiratory irritation and central nervous system depressant effects.

50

The following human exposure studies have been used as key studies by various agencies to develop air quality guidelines and standards.

#### 5 3.4.1 Short term exposures

Andersen et al (1983) reported the effects of toluene on 16 healthy young male subjects with no previous regular exposure to organic solvents. Groups of subjects were in a chamber for 6 hours a day on 4 consecutive days. After an hour of exposure, subjects went through all physiological, discomfort, and performance measurements for the next 1.5 hours. After a 1

- hour lunch, a similar series of measurements were made during the 5 th and 6 th hours of exposure. The concentration of toluene was 0, 10, 40, or 100 ppm with each group exposed to a different toluene concentration each day. There was a significant change in nasal mucus flow from control values during all of the toluene exposures. During the 100 ppm exposure, statistically significant increased irritation was experienced in the eyes and in the nose, but
- not in the throat or lower airways. There was also a statistically significant increase in the occurrence of headaches, dizziness, and feelings of intoxication during the 100 ppm exposure, but not during the other concentrations. No adverse effects were reported at the 10 and 40 ppm levels.
- Baelum et al (1985) reported a LOAEL of 100 ppm for neurological effects in humans. In this study, occupationally exposed subjects were exposed to either clean air or air containing 100 ppm toluene for 6.5 hours in a climate chamber. A battery of ten tests of visuo-motor coordination, visual performance, and cortical function were administered during the 6.5 hour period. For toluene exposed subjects, there were complaints of air quality, irritation of
   the nasal passages, and increased feelings of fatigue and sleepiness. Subjects also
- the nasal passages, and increased feelings of fatigue and sleepiness. Subjects also complained of headaches and dizziness. Toluene exposure decreased performance on four of the neuro-behavioural tests; three on visual perseverance, one of visuo-motor function.
- Baelum et al. (1990) evaluated effects of toluene at 0 or 100 ppm , or to varying exposures
  with peaks up to 300 ppm (with TWA = 102 ppm), for 7 hours. Volunteers exercised on an ergometer cycle during the exposure. Exposed subjects (with and without peak exposures) reported an significant increase over non-exposed subjects in nose and lower respiratory irritation, feelings of intoxication, dizziness, increased coughing, and headaches. No significant differences were found in the performances between the exposed and control groups in a battery of tests for performance, visual attention, and reaction times.

Echeverria et al. (1991) reported a LOAEL of 75 ppm for neurological effects in humans. In this study, two groups of 42 students were exposed to 0, 75, and 150 ppm toluene for a 7 hour period. A complete battery of 12 tests was administered before and at the end of each exposure. Toluene caused a dose-related impairment of function on digit span pattern recognition, the one hole test, and pattern memory at the 150 ppm level only. Test results for visual perception differed from control values for both exposure levels.

# 3.4.2 Longer term exposures in humans

- <sup>45</sup> Studies of workers repeatedly exposed to toluene in workplace air at concentrations ranging from about 30 to 150 ppm have found evidence for increased incidence of self-reported neurological symptoms, (Orbaek and Nise 1989; Yin et al. 1987), performance deficits in neurobehavioural tests, (Boey et al. 1997; Foo et al. 1990; Orbaek and Nise 1989), hearing loss (Abbate et al. 1993; Morata et al. 1997), changes in visual-evoked brainstem potentials (Vrca
- et al. 1995, 1997a, 1997b), and colour vision impairment (Zavalic et al. 1998a, 1998b, 1998c). ATSDR considered that sufficient data for the inhalation route were available to derive a chronic inhalation guideline based on colour vision impairment in toluene exposed workers (Zavalic et al. 1998a).

<sup>5</sup> In conclusion, the available studies each have a number of limitations. However, when considered jointly, these studies indicate that humans repeatedly exposed to toluene concentrations ranging from 40 to 132 ppm have an increased risk of developing neurological effects.

### 10 Effects on Visual Impairment

15

30

40

45

Zavalic et al.(1998a) examined two groups of Croatian workers occupationally exposed to toluene for effects on colour vision, relative to a group of unexposed controls. The first exposed group (Group E1) consisted of 46 shoe gluing workers, mean exposure durations of 16.21  $\pm$ 6.1 years while the second group (Group E2) consisted of 37 rotogravure printing workers mean exposure durations of 18.34  $\pm$ 6.03 years. Measured toluene (medians) concentrations were 22 ppm - range of 11.2 40.2 ppm for Croup E1 and 122 ppm - range of

- concentrations were 32 ppm ; range of 11.3-49.3 ppm for Group E1 and 132 ppm , range of 66-250 ppm for Group E2. Analysis of colour vision was performed and results reported as the colour confusion index (CCI) or age-and alcohol intake-adjusted colour confusion index (AACCI). In the high-exposure group (Group E2) CCI scores on both Wednesday and
- Monday were significantly higher in Group E2 relative to both controls and to Group E1. CCI scores for those workers who consumed no alcoholic beverages were significantly greater for Group E1 than for non-consumers in the control group. The AACCI scores were significantly higher (p<0.05) Group E2, but not Group E1, compared to controls. This study identified a NOAEL of 32 ppm Group E1, and a LOAEL of 132 ppm Group E2, for alterations in colour vision in toluene-exposed workers based on AACCI scores.</p>

Other studies of human colour vision impairment suggest that vision impairment result from chronic, rather than acute, exposure to toluene (Muttray et al. 1999; Zavalic et al. a, 1998b, c). The mechanism by which toluene exposure influences colour vision is not known. Vrca et al. (1995, 1997a, 1997b) showed that visual evoked potentials are affected in chronically exposed

individuals and show exposure-related changes in amplitude and latency. However, it was not clear whether the impairment of colour vision produced by toluene exposure is due solely to neurological damage or also involves damage to the eyes.

35 Neurological effects

Foo et al. (1990,1993) conducted a cross-sectional study involving exposed female workers employed at an electronic assembly plant where toluene was emitted from glue. The average number of years ( $\pm$  Standard Deviation) worked by the exposed population was 5.7  $\pm$  3.2. Toluene levels reported in the study were from personal sample monitoring and reported as an 8-hour time weighted average (TWA). Co-exposure to other solvents was not addressed in the study. Exposed workers breathed toluene air levels of 88 ppm as a TWA and control workers 13 ppm. A battery of eight neurobehavioural tests was administered to all exposed workers and control workers. The tests were performed midweek, before the workers reported to their stations for the day. Group means revealed statistically significant differences in 6 of 8 tests; all tests showed that the exposed workers performed poorly compared with the control cohort.

Yin et al 1987, reported that in a group of workers exposed to TWA of 41–46 ppm toluene the incidence of health-related complaints among the toluene exposed workers was 2–3 times
that of the controls. Dizziness was reported by about two-thirds of the toluene exposed respondents. These subjects also complained of headaches, sore throat, eye irritation, and difficulty with sleep. When the exposed subjects were divided into 2 groups, one with TWA exposures of less than 40 ppm and the other with exposures greater than or equal to 40 ppm,

<sup>5</sup> the incidence of headache and sore throat, but not dizziness, showed a concentrationresponse pattern.

# 3.4.3 Effects of exposure in laboratory animals

# Laboratory animal studies CNS effects:

- <sup>10</sup> Dose-dependent decreases in behavioural performance activity depression and central nervous system depression were observed in mice and rats exposed by inhalation to toluene at concentrations ranging from 100 to 12,000 ppm (Forkman et al 1991). Younger animals were more susceptible to toluene toxicity and mice were more sensitive than rats of the same age. Changes in the levels of brain neurotransmitters in rodents exposed to toluene have
- been observed. Significant localised changes in dopamine or noradrenaline brain levels were noted in rats exposed to 400 ppm toluene 24 hours/day for 30 days (Ikeda et al 1986) and in newborn male rats 7 weeks after a 10-day exposure to 80 ppm toluene for 6 hours per day (von Euler et al 1989). Neurotransmitter levels in some areas of the brain were increased, in some areas were decreased, and in other areas remained the same. Because of the variability in response these data are difficult to evaluate.
- 20 in response, these data are difficult to evaluate.

Hillefors-Berglund et al (1995) exposed male rats to toluene (0, 40, 80, 160 or 320 ppm, 4 weeks, 6 hours/day, 5 days/week), followed by a post exposure period of 29-40 days. Hillefors-Berglund et al reported that the rats had decreased caudate-putamen (p < 0.05) and subcortical limbic area brain wet weights (p < 0.001) compared to controls at concentrations

<sup>25</sup> subcortical limbic area brain wet weights (p < 0.001) compared to controls at concentrations of 80 ppm and higher (with trend test for dose-response significant at p < 0.01). Toluene exposure did not significantly affect the wet weights of the whole brain. The significance of regional changes in the brains of rats is difficult to relate to potential effects in humans.

# 30 Reproductive and Developmental Toxicity in Laboratory Animals

Results from several inhalation exposure studies of animals indicate that exposure to levels of toluene that begin to produce maternal toxicity can cause deleterious foetal effects, including reduced foetal survival and retardation of growth and skeletal development (Hudak and Ungvary 1978; IRDC 1985; Courtney et al 1986). No-effect levels (NOAEL) in animals for toluene on standard developmental end points range from about 133 ppm for a

- animals for toluene on standard developmental end points range from about 133 ppm for a 24 hour/day exposure protocol (Ungvary and Tatrai 1985) to 133–750 ppm with 3–6 hours/day protocols (Huntingdon Research Centre 1992; Thiel and Chahoud 1997). Courtney et al (1986) reported a NOAEL at toluene concentrations of 200ppm for developmental effects in mice.
- 40

# 3.4.4 Use of health data in setting air quality guidelines and standards

The short term human exposure studies by Andersen et al (1983) and Baelum et al (1985, 1990) have been used by various agencies (OEHHA 1999; Health and Environment Canada 1992; ATSDR 2000) to base their short-term exposure goals/standards.

45

OEHHA (1999) considered that the critical effects of short-term toluene exposure were impaired reaction time and symptoms of headache, dizziness, a feeling of slight intoxication (CNS depressant effects), and eye and nose irritation. The Andersen et al (1983) study has shown a LOAEL at 100ppm, and a NOAEL at 40ppm for both irritant effects and CNS depression of toluene. Similar views were expressed by ATSDR (2000) in their derivation of their Acute MRL.

<sup>5</sup> Health and Welfare Canada, and Environment Canada (1992) judged the study to be an adequate clinical study in human volunteers. The study by Baelum et al (1985) on neurological function was stated to be less reliable owing to limitations of study design.

# 3.5 XYLENES

- <sup>10</sup> The adverse health effects of xylenes exposure have been assessed by various agencies, including OEHHA CEPA (1999), US Dept of Health and Human Services, ATSDR (2000), IPCS (1998). The first signs of adverse effects of xylenes on humans are irritation of the nose, throat and eyes. The irritation has been chosen as the critical end point because it occurs at a low level after short exposures.
- 15

# 3.5.1 Short-term exposures in humans

Hastings et al (1984) exposed 50 healthy individuals to 100, 200, or 400 ppm mixed xylenes for 30 minutes to evaluate eye, nose, and throat irritation. The percent of subjects reporting eye irritation was 56% for controls (clean air), 60% at 100 ppm, 70% at 200 ppm, and 90% at 400 ppm. The authors concluded there was no effect on eye irritation at 100 ppm because the

<sup>20</sup> 400 ppm. The authors concluded there was no effect on eye irritation a incidence of irritation was as low as the control group.

Carpenter et al (1975) evaluated eye irritation in 6 human volunteers exposed for 15 minutes to 460, 1000, 2000, or 3000 mg/m<sup>3</sup>. One volunteer noted mild throat discomfort at 460 mg/m<sup>3</sup>, but not at 2000 mg/m<sup>3</sup>. Four subjects reported eye irritation after exposure to 2000 or 3000 mg/m<sup>3</sup> (460 or 690 ppm) xylene for 15 min while one subject reported eye irritation at 1000 mg/m<sup>3</sup> (230 ppm) and none at 478 mg/m<sup>3</sup> (110 ppm).

Nelson et al (1943) exposed 10 healthy human volunteers for periods of 3 to 5 minutes to
estimated concentrations of 100 or 200 ppm technical grade xylene. The subjects reported
eye, nose, and throat irritation at 200 ppm but not at 100 ppm. A significant area of
uncertainty arising from the study is the use of estimated rather than measured exposure
concentrations.

<sup>35</sup> Dudek et al (1990) studied ten male volunteers who were exposed to 100 ppm xylene (purity not specified) or 100 ppm toluene or a mixture of 50 ppm of each. Exposure time was 4 hours and each person participated in four exposure sessions. Changes in CNS functions were tested by nine psychological tests. Xylene had the most adverse effect on simple reaction time and choice reaction time, while the combined exposure gave weaker effects than xylene alone but stronger than toluene alone. This was considered to be the LOAEL by the investigators.

Gamberale et al (1978) confirmed that the 100 ppm is near the threshold for adverse effects as no effects on reaction times were seen in volunteers exposed to xylenes at 100 and 299 ppm
for 70 minutes. However when subjects exercised (bicycle ergometer) for the first 30 minutes of the 70 minutes exposure to 299 ppm xylenes, reaction times (choice and addition test) were increased and short term memory was impaired significantly.

# 3.5.2 Longer term exposures in Humans

<sup>50</sup> Information on the toxicity of xylenes to humans is almost exclusively limited to case reports of acute exposures and studies of occupational exposures in which persons often inhaled a mixture of hydrocarbon solvents 8 hours per day, 5-6 days per week. These studies often have incomplete information on the airborne concentrations of xylene and other hydrocarbons. One study examining chronic effects in humans from inhalation of

- <sup>5</sup> predominantly mixed xylenes for up to 7 years was identified (Uchida et al 1993) and one 4week controlled exposure study examining the effects of p-xylene exclusively was identified (Hake et al 1981).
- A survey by Uchida et al. (1993), of Chinese workers involved in the production of rubber boots, plastic coated wire and printing processes employing xylene solvents was carried out. The exposures were for an average of 7 years to xylenes predominantly. Results of analysis of the diffusive samplers showed that workers were exposed to a geometric mean of 14.2 ± 2.6 ppm xylene. This was broken down into geometric means of 1.2 ppm o-xylene, 7.3 ppm m-xylene, 3.8 ppm p-xylene, 3.4 ppm ethyl benzene, and 1.2 ppm toluene. Analysis of data from the health examinations found no statistically significant difference between blood
- examinations for xylene-exposed and unexposed populations.

Results of the survey on subjective symptoms found differences in symptoms occurring during work and during a similar analysis over the proceeding three-month period apparently related to effects on the functions of the central nervous system and to local effects on the eyes, nose and throat in the exposed workers. Dose dependency appeared to exist for 3 subjective symptoms noted during work: irritation in the eyes, sore throat and floating sensations.

# 25 **3.5.3 Laboratory Animal Exposures**

Animal data are consistent with human data in documenting respiratory effects from xylene exposure. Acute and subacute exposures in mice, rats, and guinea pigs have been associated with decreased respiratory rate, laboured breathing, irritation of the respiratory tract, pulmonary edema, and pulmonary inflammation by a number of studies (Carpenter et al 1975; Korsak et al 1988, 1993). The study by Tatrai et al (1981) exposed rate for one year. 7

- 30 1975; Korsak et al 1988, 1993). The study by Tatrai et al (1981) exposed rats for one year, 7 days/week, 8 hours per day to 1096 ppm o-xylene. This exposure level was a LOAEL for body weight gain in males and a NOAEL for hepatic effects in male rats.
- Jenkins et al (1970) exposed rats, guinea pigs, squirrel monkeys, and beagle dogs for 90-127 days continuously to 78 ppm of o-xylene. The study examined body weight gain, haematological, serum and liver function. No effect was observed in any of the parameters examined in this study. This study found a NOAEL for all effects examined of 78 ppm oxylene.

# 40 Reproductive and developmental toxicity in laboratory animals

Effects on foetal weights and skeletal development were seen from rats exposed to o-xylene at 350 ppm and for m- and p-xylene at 700 ppm (Ungvary et al 1980). Similarly in mice, foetal skeletal development was decreased at 230 ppm concentrations of mixed xylenes. Ungvary and Tatrai (1985) exposed rats by inhalation to air concentrations of xylene (60 ppm 440 ppm 800 ppm) for 24 hr/day on days 7-15 of gestation. Maternal toxicity was

- <sup>45</sup> ppm, 440 ppm, 800 ppm) for 24 hr/day on days 7-15 of gestation. Maternal toxicity was described as moderate and dose-dependent. They observed weight retarded foetuses at all air concentrations. However, there was no increase in malformations, and an increase in minor anomalies and resorbed foetuses occurred only at the highest concentration.
- <sup>50</sup> Mice were exposed by inhalation continuously to 120 ppm or 230 ppm xylene for 24 hr/day on days 6-15 of gestation. For developmental effects (weight and skeletal development retardation), the LOAEL was 230 ppm and the NOAEL was 120 ppm.

- <sup>5</sup> Hass et al (1995) exposed pregnant rats to 500 ppm xylene 6 hr/day on gestation days 7-20. Xylene exposure caused no signs of maternal toxicity and no difference in the number of live or dead foetuses. The absolute brain weights in exposed litters were lower than for control litters. Exposed offspring showed impaired performance in tests for neuromotor abilities and for learning and memory.
- 10

In a follow-up study under the same conditions, exposed offspring exhibited impaired performances for learning and memory for up to 28 weeks of age (Hass et al 1997). These data indicate that xylene exposure during development may cause long-lasting deficits on learning and memory in offspring. It is not apparent if 500 ppm is a LOAEL concentration for these effects.

15 for these effect

# 3.5.4 Use of health data in setting air quality guidelines and standards.

OEHHA (1999) have concluded that respiratory and eye irritation were the principal effects of xylenes following short term exposures in healthy human volunteers and considered that the study of Hastings et al (1984) was most suitable in setting an acute reference exposure level. They also considered that studies by Carpenter et al (1975) and Nelson et al (1943) were supportive of findings of the Hastings et al (1984) results. And that taken together, are consistent with a human NOAEL for eye irritation of about 100 ppm for at least a 30-minute exposure.

25

ATSDR (2000) have derived an acute-duration air quality guideline based on CNS depressant effects from an acute inhalation exposure study by Dudek et al (1990). They considered that CNS effects noted in studies by Gamberale et al (1978) and Carpenter et al (1975) were supportive of the endpoint chosen.

30

35

45

# 4 AIR TOXICS IN AUSTRALIA

Monitoring for air toxics in Australia has been limited. Most monitoring has been conducted on a campaign basis with a focus on 'hot-spot' issues within jurisdictions. Monitoring has been conducted mainly near major roads and industrial complexes. NSW EPA recently completed a 5-year study of air toxics in Sydney. The focus of this monitoring was at generally representative sites rather than at peak sites. Other studies have included personal exposure monitoring during various activities such as commuting (Torre et al., 2000).

A Commonwealth funded Personal Exposure Monitoring Study, conducted in Sydney,
 Melbourne, Perth and Adelaide, examined exposure to benzene, toluene and xylenes as a result of various activities (see Appendix 2).

The results of monitoring from all jurisdictions have indicated that levels of air toxics in Australia, even at hot spots, are generally quite low. In areas significantly impacted by wood smoke, elevated levels of PAHs have been observed. Some roadside monitoring has found elevated levels of benzene.

The following section provides a summary of the air monitoring data that is available in Australia. Appendix 1 provides a more detailed overview of the available data in Australia.
It should be noted that the data summarised below and in Appendix 1 is not directly comparable in many cases due to the difference in averaging times and locations in which the data was collected. As previously described, air toxics only occur at elevated levels close to sources, such as heavily trafficked roads and industrial complexes. Therefore, the location

<sup>5</sup> of the monitoring site relative to the source will impact on the resulting levels of air toxics recorded.

# 4.1 BENZENE

Of the air toxics under consideration in the proposed NEPM, the most extensive air monitoring data set exists for benzene. This data has been collected on a campaign basis mainly around industrial facilities and next to major roads. Although it is the most extensive data set available there is still a limited amount of data available.

Given that the major source of benzene in Australian cities is motor vehicles, benzene will be widely distributed within the airsheds. However, the highest concentrations will be observed next to major roads. As industry contributes only a small percentage to the total benzene emissions, these impacts are likely to be experienced in the local area close to the source rather than widely distributed across the airshed. Therefore the contribution in that local area could be higher than indicated by the airshed wide estimates.

20

25

In New South Wales campaign roadside monitoring has shown overall mean benzene levels between 0.0011ppm ppb (2 samples, 48 hours averaging period) and 0.0025ppm (21 samples, 24 hours averaging period) with the peak result of 0.0042ppm (24 hours averaging period). Overall means for residential areas are lower, ranging from 0.0003ppm (6 samples, 24 hours averaging periods) to 0.0022ppm (8 samples, 24 hours averaging periods) with the peak result of 0.0061ppm (NSW EPA, 1998).

A recently released report '*Ambient Air Quality Research Project (1996-2001) – Dioxins, Organics, Polycyclic Aromatic Hydrocarbons and Heavy Metals*' (NSW EPA 2002) indicates ambient levels of benzene in high traffic areas between 0.0009ppm and 0.0028ppm (annual average) and in residential areas between 0.0004ppm and 0.0012ppm (annual average). In Victoria air monitoring data for benzene has been collected near major roads and adjacent to major chemical complexes. The studies near or on major roads have shown that mean benzene levels at these sites range between 0.0024ppm to 0.0122 (one hour averaging period)

with peak values up to 0.0172ppm (one hour averaging period).

Personal exposure studies looking at exposure to commuters on various forms of transport have shown that the highest exposure to benzene (and other motor vehicle related pollutants) occurs within the vehicle itself. Other high exposures were reported for cyclists riding along major roads or in traffic, with 0.0132ppm result (1-hour exposure) reflecting exposure while riding a bicycle (Torre and Bardsley, 1998).

Monitoring near a major chemical complex has shown mean benzene levels ranging between 0.0005ppm and 0.008ppm (respective averaging periods 24 hours and 63.5 hours) with peak values up to 0.020ppm (63.5 hours averaging period) (Bardsley, 1991).

In Queensland air monitoring data collected in a high traffic area show the overall mean level of 0.001ppm (mean for the entire period of the study: April 2000-May 2002) with peak value of 0.0035ppm (24 hours averaging period).

50

45

40

A study conducted in an industrial area showed the overall mean level of 0.0011ppm (mean for the entire period of the study: May 2000-May 2002) with the peak result of 0.0024ppm (24 hours averaging period).

<sup>5</sup> Monitoring in a residential area resulted in the overall mean of 0.0009ppm (mean for the entire period of the study: May 2000-May 2002) with the peak result of 0.0021pp (24 hours averaging period) (Queensland EPA 2002).

In South Australia studies in high traffic areas (with some industry present at one location) showed overall mean levels of benzene of between 0.008ppm (mean over the entire monitoring period of 12 days at this location) and 19 ppb (mean over the entire monitoring period of 34 days at this location) with the peak result of approximately 0.078ppm (1 hour averaging period) (Mitchell, Peat and Caruso, 1884).

- <sup>15</sup> An industry oriented study showed the overall mean level for the entire sampling period (two months) of 0.003ppm and the range of results between 0 and 0.008ppm (1 hour averaging period) (Agar *et al*, 2000; Mitchell *et al*, 1994).
- In Western Australia studies in high traffic areas showed overall mean levels of benzene of 0.00168ppm (63 samples, 24 hours averaging periods) and 0.002ppm (65 samples, 24 hours averaging periods) with the peak results of 0.0176ppm and 0.0112ppm respectively.

The results in industrial areas were much lower with overall means of 0.00028ppm (33 samples, 24 hours averaging periods) and 0.00043ppm (10 samples, 24 hours averaging periods) and the peak results of 0.00105ppm and 0.00095ppm respectively.

In residential areas overall means ranged from 0.00012ppm (4 samples, 24 hours averaging periods) to 0.00039ppm (14 samples, 24 hours averaging periods) with the peak result of 0.0009ppm (WA DEP, 2002).

30

25

A study conducted in the Australian Capital Territory in an area likely to be affected by traffic emissions showed range of results between 0 and 0.0001ppm (24 hours averaging period) (Environment ACT, 2001).

# 35 4.2 FORMALDEHYDE

For formaldehyde the major sources are industry and motor vehicles. Of the five air toxics addressed in this NEPM, the database for formaldehyde is the most limited. The only data available comes from studies conducted in Victoria, Queensland and South Australia.

- <sup>40</sup> The data collected in Victoria come from two sites in areas of high traffic one in the CBD Melbourne and one near a freeway in Malvern. The levels observed in both studies were similar with values ranging from 0.0004ppm to 0.0076ppm (averaging periods 19-27 hours) (VicRoads/EPA Victoria, 1994).
- <sup>45</sup> In Queensland, the data collected in a high traffic area (CBD site) show the overall mean level of formaldehyde of 0.0032ppm (mean for the entire period of the study: May 2000-May 2002) with a peak result of 0.0077ppm (24 hours averages).

The data collected in an industrial area show the overall mean level of formaldehyde of 0.0057ppm (mean for the entire period of the study: October 1999-May 2002) with a peak result of 0.0178ppm (24 hours average) (Queensland EPA 2002).

The South Australian data collected in high traffic areas show overall mean levels of formaldehyde between 0.01ppm (mean for the entire monitoring period of 12 days at this

<sup>5</sup> location), and 0.018ppm (mean for the entire monitoring period of 34 days at this location) with the peak value of approximately 0.135ppm (1 hour averaging period) (Mitchell, Peat and Caruso, 1994).

The data collected near an industrial source show the overall mean of 0.02ppm (mean over the entire monitoring period of two months) and the range of results between 0.005ppm and 0.036ppm (30 minute averaging period) (Agar *et al* 2000; Agar *et al* 2001a; Mitchell *et al* 1994).

No data have been identified for New South Wales, Western Australia, Tasmania, Australian Capital Territory and Northern Territory.

15

20

10

#### 4.3 **POLYCYCLIC AROMATIC HYDROCARBONS**

Of all the air toxics under consideration in this NEPM, PAHs are the most complex category, as this group includes a large number of different compounds. The majority of studies have focussed on PAHs associated with particles (one study, Muller *et al* 1998, included also vapour phase PAHs). The data have been collected in areas near major roads or in wood smoke affected areas.

Given that the major source of PAHs in Australia is combustion processes (domestic solid fuel burning, motor vehicles, bushfires, etc.) they will be widely distributed within the airsheds. Although industry contributes a small percentage to the total PAHs emissions, these impacts are likely to be experienced in the local area close to the source rather than widely distributed across the airshed.

A recent study conducted in NSW in a number of areas with smoke emissions associated with domestic solid fuel heating shows overall mean levels of total PAHs between 0.22 ng/m<sup>3</sup> (mean of four 24 hours samples collected during summer) and 23.8 ng/m<sup>3</sup> (mean of fourteen 24 hours samples collected during winter) with the peak value of 52.3 ng/m<sup>3</sup> (24 hours average). Corresponding values for benzo(a) pyrene were 0.22 ng/m<sup>3</sup>, 4.21 ng/m<sup>3</sup> and 8.99 ng/m<sup>3</sup> respectively (NSW EPA 2002).

35

In Victoria studies have been conducted both near roads and in areas impacted by domestic wood smoke. A study conducted near a major road in Flemington over a 12 month period has shown 24-hour values ranging from 0 ng/m<sup>3</sup> to 1.17 ng/m<sup>3</sup> (VicRoads/EPA 1991). The annual average value for this period was 0.17 ng/m<sup>3</sup>.

40

45

50

Gras et al., (1992) monitored a number of PAHs at Alphington and Footscray. Values obtained at the two sites varied by up to a factor of four, with the higher values being recorded at Alphington. In this study samples were only collected on days when fine particle levels ( $PM_{2.5}$ ) were elevated (FPM> 30 µg/m<sup>3</sup>). Peak 24 hour values for BaP were 13.3 ng/m<sup>3</sup> at Alphington and 6.4 ng/m<sup>3</sup> at Footscray. The higher levels at Alphington were thought to be due to a higher contribution from domestic wood smoke at that site.

In Queensland data collected near areas of high traffic show overall mean levels of total PAHs ranging from 20.34 ng/m<sup>3</sup> to 90.19 ng/m<sup>3</sup> with the peak result of 150.91 ng/m<sup>3</sup> (averaging period between 2 and 9 days).

Data collected in areas with no easily identifiable major sources of PAHs show lower mean levels of total PAHs. The results range from 16.1 ng/m<sup>3</sup> to 23.2 ng/m<sup>3</sup> with the peak result of 49.6 ng/m<sup>3</sup> (averaging period between 2 and 9 days) (Muller *et al*, 1998).

- <sup>5</sup> In Western Australia data collected in areas of high wood smoke emissions (one area, Duncraig, is also affected by traffic emissions) show levels of PAHs ranging from 1.07 ng/m<sup>3</sup> to 2004.3 ng/m<sup>3</sup> (12 hours averaging period) (Gras, 1996), with the higher levels being observed during periods impacted by domestic wood smoke.
- <sup>10</sup> In Tasmania data collected in areas with that are significantly impacted by domestic wood smoke show mean levels of benzo(a)pyrene ranging from 0.5 ng/m<sup>3</sup> to 3.79 ng/m<sup>3</sup> with the peak result of 34.28 ng/m<sup>3</sup> (24 hours averaging period) (Expert Working Party, 1996).
- In the Australian Capital Territory data collected near areas of moderate traffic show mean levels of total PAHs ranging from 0.053 ng/m<sup>3</sup> to 1.7 ng/m<sup>3</sup> (24 hours averaging period). Data collected in a residential area show mean levels of total PAHs ranging from 0.053 ng/m<sup>3</sup> to 0.13 ng/m<sup>3</sup> (24 hours averaging period) (Fox, 1999).
- In the Northern Territory data collected in areas with emissions from biomass burning show mean levels of total PAHs ranging from 0.8 ng/m<sup>3</sup> to 3.3 ng/m<sup>3</sup> with the peak result of 13.1 ng/m<sup>3</sup> (24 hours averaging period) (Vanderzalm *et al*, 1998).

No data was identified for South Australia.

# 25 **4.4 TOLUENE**

The data set for toluene is comparable to the one for benzene, however, it is slightly less comprehensive. Air monitoring data for toluene have been collected from the similar locations to those used for benzene monitoring, ie. predominantly near major roads and near industrial facilities.

30

35

Again, given that the major source of toluene in Australian cities is motor vehicles, toluene will be widely distributed within the airsheds. However, the highest concentrations will be observed next to major roads. Although industry contributes only a small percentage to the total toluene emissions, these impacts are likely to be experienced in the local area close to the source rather than widely distributed across the airshed. Therefore the contribution in

that local area could be higher than indicated by the airshed wide estimates.

In New South Wales data collected near major roads show overall mean levels of toluene between 0.002ppm (48 hours averaging period) and 0.0049ppm (24 hours averaging period) with the peak result of 0.0094ppm (24 hours averaging period). Overall means for residential areas were similar at between 0.0008ppm and 0.0058ppm with the peak result higher at 0.020ppm (24 hours averaging period) (NSW EPA, 1998).

- In Victoria data collected near or on major roads show mean levels of toluene between 0.0031ppm and 0.0208ppm (respective averaging periods of 12 hours and 1 hour). In industrial areas mean levels were between 0.0012ppm and 0.0033ppm with the peak result of 0.0089ppm (24 hours averaging period) (Bardsley, 1996; Bardsley, 1997; Torre, Bardsley & Eriksen, 1996; Torre, Bardsley *et al*, 1998).
- <sup>50</sup> In Queensland air monitoring data collected in a high traffic area show the overall mean level of 0.0041ppm (mean for the entire period of the study: April 2000-May 2002) with the peak value of 0.035ppm (24 hours averaging period).

<sup>5</sup> A study conducted in an industrial area showed the overall mean level of 0.0031ppm (mean for the entire period of the study: May 2000-May 2002) with the peak result of 0.0105ppm (24 hours averaging period).

Monitoring in a residential area resulted in the overall mean of 0.0035ppm (mean for the entire period of the study: May 2000-May 2002) with the peak result of 0.0176ppm (24 hours averaging period) (Queensland EPA, 2002).

In South Australia studies in high traffic areas (with some industry present at one location) showed overall mean levels of toluene of between 0.037ppm (mean for the entire monitoring period of 12 days at this location) and 0.084ppm (mean for the entire monitoring period of 34 days at this location) with the peak result of approximately 0.555ppm (1 hour averaging period).

An industry oriented study showed the overall mean level for the entire sampling period (two months) of 0.005ppm and the range of results between 0 and 0.038ppm (30 minutes averaging period) (Agar *et al*, 2000; Mitchell *et al*, 1994).

In Western Australia studies in high traffic areas showed mean levels of toluene of 0.00297ppm (63 samples, 24 hours averaging periods) and 0.00393ppm (65 samples, 24 hours averaging periods) with the peak results of 0.02996ppm and 0.01327ppm respectively.

The results from industrial areas were much lower with overall means of 0.00044ppm (33 samples, 24 hours averaging periods) and 0.00164ppm (10 samples, 24 hours averaging period) and the peak results of 0.00165ppm and 0.00259ppm respectively.

30

25

15

In residential areas overall means ranged from 0.00019ppm (4 samples, 24 hours averaging periods) to 0.00109ppm (8 samples, 24 hours averaging periods) with the peak result of 0.00198ppm (WA DEP, 2002).

<sup>35</sup> No data have been identified for Tasmania, Northern Territory or Australian Capital Territory.

# 4.5 XYLENE

The data set for xylenes is comparable to the one for benzene and toluene, however, as for toluene it is slightly less comprehensive than the one for benzene. Air monitoring data for xylenes has been collected from similar locations to those used for toluene monitoring, ie. predominantly near major roads and industrial facilities and is available for a similar number of jurisdictions.

Again, given that the major source of xylenes in Australian cities is motor vehicles, xylenes will be widely distributed within the airsheds. However, the highest concentrations will be observed next to major roads. Although industry contributes only a small percentage to the total xylenes emissions, these impacts are likely to be experienced in the local area close to the source rather than widely distributed across the airshed. Therefore the contribution in that local area could be higher than indicated by the airshed wide estimates.

In New South Wales data collected near major roads show overall mean levels of xylenes between 0.0005ppm (48 hours averaging period) and 0.0025ppm for m-, p-xylenes (24 hours averaging period) with the peak result of 0.0045ppm (24 hours averaging period) and

<sup>5</sup> 0.0005ppm (48 hours averaging period) and 0.0009ppm (24 hours averaging period) with the peak result of 0.0016ppm (24 hours averaging period).

Overall means in residential areas were similar at between 0.0003ppm and 0.0023ppm for m-, p-xylenes with the peak result of 0.0066ppm and 0.0001ppm and 0.0009ppm for o-xylenes with the peak result of 0.0035ppm (24 hours averaging period) (NSW EPA, 1998).

In Victoria data collected near or on major roads show mean levels of xylenes between 0.0038ppm and 0.0098ppm for m-, p-xylenes and 0.0013ppm and 0.0034ppm for o-xylenes (1 hour averaging periods).

15

10

In industrial areas in Victoria mean levels were between 0.0004ppm and 0.0011ppm for m-, p-xylenes and 0.0002ppmand 0.0003ppm for o-xylene with the peak result of 0.0045ppm for m-,p-xylenes and 0.0008ppm for o-xylene (24 hours averaging period) (Bardsley, 1996; Bardsley, 1997; Torre *et al*, 1996; Torre *et al*, 1998).

20

In Queensland air monitoring data collected in a high traffic area show the overall mean level of p-xylene of 0.0013ppm (mean for the entire period of the study: April 2000-May 2002) with the peak value of 0.0023ppm (24 hours averaging period).

- A study conducted in an industrial area showed the overall mean level p-xylene of 0.0014ppm (mean for the entire period of the study: May 2000-May 2002) with the peak result of 0.003ppm (24 hours averaging period).
- Monitoring in a residential area resulted in the overall mean level of p-xylene of 0.001ppm (mean for the entire period of the study: May 2000-May 2002) with the peak result of 0.0022ppm (24 hours averaging period) (Queensland EPA, 2002).

In South Australia studies in high traffic areas (with some industry present at one location) showed overall mean levels of p-xylene between 0.005ppm (mean for the entire monitoring period of 12 days at this location) and 0.013ppm (mean for the entire monitoring period of 34 days at this location) with the peak result of approximately 0.043ppm (1 hour averaging period) (Agar *et al*, 2000; Mitchell *et al*, 1994).

- In Western Australia studies in high traffic areas showed mean levels of m,p-xylenes and oxylene of 0.00193ppm and 0.00078ppm in one location (63 samples, 24 hours averaging periods) and 0.00238ppm and 0.00093ppm in another location (65 samples, 24 hours averaging periods). The peak results were 0.01672ppm, 0.00586ppm and 0.01327ppm, 0.00476ppm respectively.
- <sup>45</sup> The results from industrial areas were much lower with mean levels of m,p-xylenes and oxylene of 0.00032ppm and 0.00011ppm in one location (33 samples, 24 hours averaging periods) and 0.00104ppm and 0.00036ppm in another location (10 samples, 24 hours averaging period). The peak results were 0.00286ppm, 0.00055ppm and 0.00186ppm, 0.00099ppm respectively.
- 50

In residential areas overall means for m-, p-xylenes and o-xylenes ranged from 0.00025ppm and <0.0001ppm (4 samples, 24 hours averaging periods) to 0.00062ppm and 0.00025ppm (8 samples, 24 hours averaging periods) with the peak results of 0.00145ppm and 0.00065ppm respectively (WA DEP, 2002).

5 No data have been identified for Tasmania, Northern Territory or Australian Capital Territory.

#### 5 MANAGEMENT OF AIR TOXICS IN AUSTRALIA

- Specific emission limits and maximum ground level concentrations for individual sources are used in some jurisdictions to control emissions from industrial sources. These emissions of air toxics in Australia are regulated through State, Territory and local government regulatory mechanisms.
- Emissions of air toxics from new motor vehicles are controlled through Australian Design Rules that set emission standards for a range of pollutants including hydrocarbons. Recently the Commonwealth introduced national fuel quality standards that should also reduce the levels of some air toxics in ambient air.
- Wood heaters can be a significant source of air toxics, especially PAHs, during the winter months. Most States and Territories have developed regulations that ban the sale of wood heaters that do not comply with Australian Standard 4013 which sets emission limits for particles. In addition, Victorian legislation is currently being developed to ensure that wood heaters that do not comply with AS4013 are not manufactured or installed in that State. Reducing particle emissions will also lead to a reduction in emissions of PAHs. Other initiatives to reduce wood smoke emissions include the introduction of legislation to regulate
- the moisture content of fire wood sold by wood merchants (WA and ACT) and the development of legislation to make the operation of excessively smoky heaters an offence (Tasmania).
- At the national level there have been two NEPMs developed that may lead to reductions in ambient levels of air toxics – the Diesel Vehicle Emissions NEPM and the National Pollutant Inventory NEPM. These measures are implemented through State and Territory frameworks and legislation. In recognition of the need to ensure that the in-service emissions performance of diesel motor vehicles is sustained, NEPC made the Diesel Vehicle Emissions NEPM in June 2001. The goal of the NEPM is to reduce exhaust emissions from diesel
- NEPM in June 2001. The goal of the NEPM is to reduce exhaust emissions from diesel vehicles by facilitating compliance with in-service emissions standards. Developed conjointly by the National Road Transport Commission, the Diesel Vehicles Emission NEPM includes guidelines for use by jurisdictions to reduce exhaust emissions. The guidelines relate to:
- 40 test and repair programs;
  - audited fleet maintenance programs;
  - smoky vehicle programs;
  - retrofit programs (fitting exhaust after-treatment devices to diesel vehicles); and
  - engine re-build programs.
- 45

The National Pollutant Inventory (NPI) NEPM provides for comprehensive estimated pollution emissions for Australia. Industry estimates emissions from individual facilities for nearly 2,400 facilities. The NPI also includes Aggregated Emission Data (AED) estimates for all large urban centres and many regional areas from sub-threshold industry and non-industry sources such as domestic solid fuel burning. AED estimates are made by State and Territory government agencies for airsheds and water estelements within their inriedictions.

industry sources such as domestic solid fuel burning. AED estimates are made by State and Territory government agencies for airsheds and water catchments within their jurisdictions. Reporting on the full list of 90 substances (including all of the air toxics under consideration) was required for 2001/02 and information is available on the NPI website: www.npi.gov.au 5 A summary of existing programs for the management of air toxics emissions in each jurisdiction can be found in Appendix 3.

# 6 ALTERNATIVE METHODS OF ACHIEVING THE DESIRED ENVIRONMENTAL OUTCOME

<sup>10</sup> Section 17(b) of the NEPC Act requires that an Impact Statement include: *"a statement of the alternative methods of achieving the desired environmental outcomes and the* 

reasons why those alternatives have not been adopted".

This section discusses alternatives approaches for achieving the desired environmental outcome specified in the proposed NEPM.

#### 6.1 ALTERNATIVE APPROACHES

There are several approaches that may be considered in the light of their ability to deliver the desired environmental outcome. These are:

- Commonwealth legislation;
  - use of overseas guidelines;
  - inter-governmental agreement or memorandum of understanding;
  - maintain the status quo; and
  - National Environment Protection Measure.
- 25

#### 6.1.1 Commonwealth legislation

Legal advice, obtained at the time of making the Ambient Air Quality NEPM in 1998, indicated that it may not be possible for the Commonwealth (given its powers under the Constitution) to introduce legislation that could deliver the desired environmental outcome being pursued through the development of the Air Toxics NEPM. Further, given the constrained approach being taken at present in relation to environmental issues.

35

30

being pursued through the development of the Air Toxics NEPM. Further, given the cooperative national approach being taken at present in relation to environmental issues, particularly through the NEPC and the Environment Protection and Heritage Council and the fact that the States have responsibility for air quality management, the Commonwealth is unlikely to pursue unilateral action to develop a nationally consistent framework for the monitoring and reporting of air toxics.

## 6.1.2 Use of overseas guidelines

NHMRC has previously determined a set of air quality guidelines for some of the major air pollutants based on their human health effects. These guidelines are employed by several jurisdictions and provide guidance in assessing the results of air monitoring programs. These guidelines do not provide a framework for monitoring and reporting air quality and different approaches in the application of the NHMRC guidelines between jurisdictions have significantly reduced the level of certainty envisaged by the Inter-Governmental Agreement on the Environment. The NEPC was established with the ability to develop standards for ambient air quality at the same time as developing nationally consistent monitoring and reporting protocols. The clear intention at the time of making the Ambient Air Quality NEPM was that the NEPM standards and associated monitoring and reporting protocols would replace NHMRC guidelines for air quality. This is also the case for the development of the Air Toxics NEPM.

50

<sup>5</sup> Although there are current NHMRC guidelines for formaldehyde they make no reference to standardising monitoring or reporting requirements between jurisdictions, making cross-jurisdictional comparisons difficult and possibly creating compliance difficulties for companies with operations in more than one jurisdiction.

#### 10 6.1.3 Inter-governmental Agreement or memorandum of understanding

An overarching agreement would provide for a common starting point for the development and implementation of a national framework for monitoring and reporting for the air toxics under consideration in the proposed NEPM.

- <sup>15</sup> The issue of how such a framework should be developed and the impacts of any standards/guidelines that may be adopted would need to be addressed. This could be achieved by agreement, either in line with the NEPC process or by each jurisdiction agreeing to manage this issue within their jurisdiction. In the latter case, several jurisdictions would need to establish mechanisms of the type currently envisaged under the draft NEPM. This
- <sup>20</sup> approach would not necessarily provide a sufficient degree of uniformity nor compatibility in the monitoring and reporting requirements necessary to make the assessment of air quality data meaningful. It is considered that the most effective way to ensure consistency in data collection is the development of the Air Toxics NEPM.
- As with the overseas guidelines option discussed above, the agreed monitoring and reporting requirements would not have any legislative basis under this option, and therefore there would be no requirement for uniformity of application across Australia.

This approach offers no obvious advantage over the proposed Air Toxics NEPM as a similar process would be required, but without the likelihood of achieving uniformity in practice.

### 6.1.4 Maintaining the status quo

30

Arguments to maintain the *status quo* imply that the present approach to monitoring and reporting of air toxics and the development of air quality objectives for air toxics, whereby
individual jurisdictions develop their own guidelines and or standards (or adopt/modify existing guidelines/standards) is the most efficient. It also assumes that any 'natural evolution' of air quality standards/guidelines would address issues such as equivalent protection and variations in jurisdictional approaches. The *status quo* has the potential to create, or may have already created, market distortions, and may not be in keeping with National Competition Policy.

The *status quo* needs to take into account systems as they evolve and does not necessarily mean that a national approach to monitoring and reporting of air toxics or ambient air quality standards for the selected air toxics would not develop at some point. It is recognised that there are current developments in jurisdictions that will result in substantial improvements in ambient levels of air toxics. Some improvements are the result of national strategies, for example, the introduction of vehicle fuel quality standards. Other strategies have been developed by individual jurisdictions to improve particular aspects of air quality.

<sup>50</sup> At present, for pollutants not covered by the Ambient Air Quality NEPM, air quality reporting standards differ widely between jurisdictions, reflecting their often different requirements for use of the data collected. Costs are also incurred by some jurisdictions in developing and revising their respective air quality objectives resulting in duplication of costs and effort. The different procedures and interests of each jurisdiction can also result in <sup>5</sup> additional industry costs and effort in providing data and input into standard setting or revision.

The level of community input to air quality management and policy development varies from jurisdiction to jurisdiction. It is also unclear whether the development of standards for air toxics evolving under these circumstances would provide industry and the general community with the level of access and input into standards development that occurs with the NEPC process.

- It is also likely that any evolution in the monitoring and reporting of air toxics and air quality standards would occur at different rates among jurisdictions, depending on the environmental management experience and supporting systems already in place, making it difficult for industry to plan at the national level. A national picture of air quality relating to the air toxics specified in the proposed NEPM would also be less likely to emerge.
- 20 Community submissions on the Issues and Discussion Papers prepared as part of the development of the draft NEPM show that there is strong support for a national approach for the monitoring and reporting of air toxics data. The '*status quo*' option does not deliver any improved national uniformity.

#### **6.1.5** Adoption of the Air Toxics National Environment Protection Measure

A NEPM for Air Toxics will facilitate a harmonised national framework for monitoring and reporting of these substances. The investigation levels proposed in the National Environment Protection Guideline are intended to achieve the NEPC objective of providing equivalent protection, in this case from the adverse health effects associated with air pollution. The framework and approach to assessing and monitoring air toxics outlined in the proposed NEPM provide a well-defined objective for management of air quality, enhancing national certainty in environmental protection.

As indicated above, other means of approaching the issue would not provide a sufficient degree of uniformity or compatibility in the monitoring and reporting requirements necessary to make the data meaningful. Consideration of the alternatives clearly points to adoption of the Air Toxics NEPM as the preferred option, which is considered to be the most effective way to achieve national consistency in the monitoring and assessment of air quality relating to ambient air toxics.

40

45

50

30

10

#### 6.1.6 Consequences of not making the Air Toxics NEPM

If the Air Toxics NEPM is not made, it is likely that the current levels of monitoring and reporting by jurisdictions will continue in their current form for air toxics. Without national consistency, some jurisdictions may adopt standards, but these may vary between jurisdictions. This could result in differing environmental performance requirements between jurisdictions which would fail to deliver the objectives of the Inter-Governmental Agreement on the Environment, in particular the goal of the National Competition Policy, the "level playing field" and certainty for business decision-making objectives. Voluntary attempts to achieve harmonisation between jurisdictions have had mixed success. The NEPC was specifically established to overcome the problems associated with those voluntary attempts in a manner consistent with the nature of Australian governance.

Not making the Air Toxics NEPM would remove an essential stimulus for a harmonised national air monitoring and reporting system for Air Toxics. The current situation whereby

<sup>5</sup> jurisdictions collect data using different monitoring regimes, and store and report the data in varying formats is likely to continue in the absence of a national approach.

#### 6.1.7 Summary

Consideration of the alternatives clearly points to an Air Toxics NEPM as the preferred option to achieve national consistency in the monitoring and reporting of air toxics in Australia.

#### 7 ALTERNATIVE APPROACHES TO EVALUATING RESULTS OF AIR TOXICS MONITORING

- <sup>15</sup> The Guideline contained in Schedule 2 of the draft NEPM introduces air quality "investigation levels", based on the protection of human health, that can be used in the assessment of air toxics monitoring data. The intent of these levels is to provide a benchmark for use in assessing the results of monitoring of air toxics.
- A standard or level established in a guideline is a benchmark against which ambient air quality data can be compared. No mechanism is currently available to ensure that all jurisdictions adopt the same benchmark. In the past, guidelines such as those issued by NHMRC and ANZECC dealing with a range of issues have not delivered consistency throughout the country.

25

30

In developing the ambient air quality NEPM all jurisdictions recognised that developing air quality standards through NEPC was the only way to ensure national consistency and the desired environmental outcome of equivalent protection for all Australians. The development of a NEPM for air toxics in Australia is consistent with this position. The proposed NEPM investigation levels for benzene, formaldehyde, Polycyclic Aromatic Hydrocarbons (PAHs), toluene and xylenes. These investigation levels comprise a maximum concentration and an averaging period for measurement for each pollutant.

The proposed NEPM also establishes a goal that specifies the aim of the NEPM in collecting sufficient data to facilitate the development of standards following a review of the Measure within eight years of commencement.

Each jurisdiction manages emissions of air toxics and would be required to report annually on progress in meeting the goal of the NEPM.

40

For the air toxics contained in the NEPM, data are limited. As the introduction of the NEPM will require jurisdictional monitoring of these pollutants, it is important to consider the implications for monitoring of ambient levels.

As previously discussed air toxics exist at relatively low concentrations in urban airsheds, with significantly elevated levels only occurring near sources such as industrial sites or heavily trafficked roads. Therefore it is appropriate for the benchmarks adopted for the proposed Air Toxics NEPM to apply at Stage 2 sites rather than at generally representative sites. The cost and resource intensiveness of monitoring air toxics also needed careful consideration in determining the form of the proposed NEPM and was critical in the development of the associated monitoring protocols.

- <sup>5</sup> A critical question in the development of the proposed NEPM was "what is the most efficient way to generate improved information about air toxics in Australia?" This issue has shaped the draft NEPM and, in turn, will help place jurisdictions in a stronger position to understand and manage sources of air toxics.
- 10 The types of benchmarks that were considered for the proposed NEPM were:
  - Standard with Compliance Goal and Specified Monitoring and Reporting Protocol;
  - Advisory Reporting Standard; and
  - Investigation Levels.
- <sup>15</sup> These options were considered in the Discussion Paper and are summarised below.

## 7.1 OPTION 1: STANDARD WITH COMPLIANCE GOAL AND SPECIFIED MONITORING AND REPORTING PROTOCOL

Consideration was given to standards structured in a manner similar to those in the Ambient Air Quality NEPM standards as maximum concentrations and with a goal framed in terms of the achievement of the standard over a specified time frame. The significant difference between the proposed NEPM and the Ambient Air Quality NEPM is that the former would apply at Stage 2 sites rather than generally representative monitoring sites. All jurisdictions would be required to achieve the goal within the specified time frame. The monitoring and

<sup>25</sup> reporting protocols for this option would be similar to those specified for the Ambient Air Quality NEPM, ie requiring fixed site monitoring.

The monitoring protocol for this option would be rigorous and parallel the protocol already in place for the Ambient Air Quality NEPM. This would require extensive monitoring of the five air toxics and the development of a prescriptive air monitoring plan.

The main advantage of this option is that it would provide the most comprehensive collection of data. A further advantage is that a timeframe to reach compliance with the NEPM would be specified.

35

The principal disadvantage of this option is that it is the most resource-intensive of the options. The cost of mandatory monitoring is higher than for any other of these three options and maybe prohibitive. Given the costs associated with this type of standard, a detailed understanding of the potential impact of concentrations of the air toxics under consideration would be required to justify such a standard. At present the necessary data do not exist in

40 would be required to justify such a standard. At present the necessary data do not exist Australia or elsewhere.

Nationally there are insufficient monitoring data resulting in reduced certainty in the assessment of the potential impacts arising from the implementation of the proposed NEPM.

45 Any standard based on such information may either not be stringent enough, or be unnecessarily stringent, and could therefore create unforeseen problems for jurisdictions in implementation.

Overall, the uncertainty associated with this option and the likely cost of implementation make this option unrealistic at this time.

#### 5 7.2 OPTION 2: ADVISORY REPORTING STANDARD

Advisory reporting standards based on the protection of human health would have the same numerical values as those under Option 1, but without an associated goal setting a specific time frame set for achieving compliance with the standards. The monitoring and reporting protocol associated with such advisory reporting standards would establish a reference

- <sup>10</sup> method for monitoring and requirements for assessment, monitoring and reporting. Unlike Option 1 it would give jurisdictions flexibility in relation to the timing of monitoring conducted while still enabling data to be collected and assessed against protective health standards.
- <sup>15</sup> Monitoring methods would be stipulated so that consistent and comparable data would be collected nationally. Jurisdictions would be required to report annually on the monitoring undertaken and whether or not standards were exceeded.

The monitoring protocol for this option would be the same as Option 1, requiring fixed site monitoring at peak sites, but the timeframe for compliance would be flexible. The objective of the monitoring protocol under this option would be to ensure the improvement in the collection of Australia-wide air toxics data over time as resources become available. The setting of a numerical standard would allow jurisdictions to compare their air quality against a health protective value. Jurisdictions can then choose to take action as appropriate

to reduce levels of air toxics in ambient air.

This Option would require fixed site monitoring as in Option 1 but the extent of monitoring would be decided by jurisdictions as resources become available. Jurisdictions would report to NEPC as part of their annual reporting cycle, on the monitoring undertaken or on plans to introduce monitoring as well as the results of any monitoring undertaken.

The major objective of the monitoring protocol under this option would be to ensure an improvement in the collection of Australia-wide air toxics data in a consistent and comparable manner. Under this option, jurisdictions not currently monitoring air toxics may initially elect to only undertake limited monitoring in areas that are significantly impacted, while others might decide to establish comprehensive monitoring systems.

This Option would require fixed site monitoring. Jurisdictions would be expected to implement a fixed-site monitoring network for air toxics as resources become available.

40

30

35

The main advantage of this option is that it allows maximum flexibility for jurisdictions in introducing monitoring for air toxics, as resources become available. It also allows for jurisdictions to focus resources on priority areas while providing a mechanism for the generation of a nationally consistent data set.

45

The main disadvantage is that this option, as with Option 1 requires fixed site monitoring which cannot be justified at this time given the current knowledge of air toxics in Australia. Significant resources could be spent on establishing such networks when monitoring may indicate that this was not necessary in the long-term.

50

As with Option 1, this option is highly resource-intensive. The cost of monitoring may be cost prohibitive for many jurisdictions. Given the costs associated with this type of standard, a detailed understanding of the potential impact of concentrations of the air toxics under consideration would be required to justify such a standard. At present the necessary data do

<sup>5</sup> not exist in Australia or elsewhere. Overall, the uncertainty associated with this option and the likely cost of implementation make this option unrealistic at this time.

#### 7.3 **OPTION 3: INVESTIGATION LEVELS**

This option would set numerical values that are protective of human health and if these values were exceeded then further investigation may be triggered. This investigation may involve further monitoring and assessment of the circumstances that may have led to the levels being exceeded. Measurements would be made at locations where significantly elevated ambient levels of the pollutant might be expected and where significant numbers of people may be exposed.

15

20

30

Where monitoring indicates ambient levels of the pollutant exceed the investigation level, management actions of a localised rather than regional nature may be required. This would be a matter for jurisdictions. The proposed NEPM requires the collection of data that reflect the distribution of air toxics in urban airsheds and facilitates an assessment of the health risks posed by air toxics in the Australian air environment.

The associated monitoring protocol would provide guidance on statistical sampling design, monitoring methods and selection of sites where such a standard may be applied. Sampling would involve campaign monitoring rather than fixed site monitoring (required by Options 1 and 2).

25 1 and

Jurisdictions would report annually on the application of the NEPM and results of the monitoring undertaken as well as details of any actions taken or proposed to reduce levels of air toxics when the investigation levels have been exceeded. This would meet the intent of the NEPC Act to provide equivalent protection to all Australians.

There are significant advantages associated with Option 3. The main advantages are that:

- Resources are focused on locations of potential high exposure as a priority and therefore where problems occur.
- Jurisdictions have the flexibility to allocate resources to priority locations. This is likely to be the lowest cost option for monitoring and address likely high exposure levels as a priority.
  - Equipment would not be locked into long term sites enabling data to be collected from Stage 2 sites thus giving a more cost effective measure of population exposure.
- It is a cost effective option with only limited monitoring equipment required to cover a number of monitoring locations.

#### 7.4 **PREFERRED OPTION**

45

50

Given the limited data and information on sources of air toxics available in Australia the setting of full compliance standards or advisory reporting standards cannot be justified at this time. The only viable option at this time is to set investigation levels.

The aim of the monitoring protocol, associated with campaign monitoring, is to provide statistically valid monitoring data that reflect the nature and distribution of air toxics within an airshed. The protocol would be developed to ensure that any assessment of air quality within a defined area would consider the land use of that area and the distribution of the pollutant in that area (eg highly exposed kindergarten versus non-populated area).

Impact Statement for the National Environment Protection (Air Toxics) Measure

#### 5 7.5 **REGIONAL ENVIRONMENTAL DIFFERENCES**

15

35

40

45

In making any NEPM or variation to an existing NEPM, the National Environment Protection Council must have regard to, *inter alia*, "any regional environmental differences in Australia" (Section 15(g) of the National Environment Protection Council Acts). In addition, Section 17(b)(v) of the Acts requires that the Impact Statement to be prepared with the draft NEPM include "a statement of the manner in which any regional environmental differences.

10 NEPM include "a statement of the manner in which any regional environmental differences in Australia have been addressed in the development of the proposed Measure".

While the Acts do not provide any explicit definition of the term "regional environmental differences", Sections 15 and 17 provide a clear indication that the term is not intended to encompass regional economic and social differences.

The term "regional environmental differences" recognises that fundamental environmental characteristics of different regions may be very different, and that to apply uniform monitoring and reporting protocols and investigation levels would not necessarily further the desired outcome of equivalent protection espoused in the legislation. For example, the

- the desired outcome of equivalent protection espoused in the legislation. For example, the issue of salinity in water bodies would provide a clear need for regional environmental differences to be taken into account in developing NEPM standards and goals for water quality.
- For ambient air quality, there are no clear-cut differences in the natural state of the atmosphere that could be a meaningful reflection in different ambient air quality standards for the protection of human health. While atmospheric conditions can change rapidly and dramatically across Australia, this provides a challenge for air quality management strategies but cannot, in any practical sense, be reflected in standards nor in the monitoring and reporting protocols.

In determining appropriate standards for the protection of human health, available evidence suggests that within any population the variation in physiological response to pollutants is likely to be significantly greater than any potential variation in impact due to meteorological or other differences across Australia.

Air quality objectives have been applied uniformly in several overseas jurisdictions that have far more diversity in climate than does Australia. Primary Air Quality Standards legislated in the United States of America applies in all states of that country. They do not make allowances for regional climatic differences. Also the European Commission does not make such allowances in determining its air quality objectives.

Although it is expected that the distribution of air toxics will differ from that of the criteria pollutants, these differences are expected to be reflected in each region across Australia. Thus there is no need to set different standards for air toxics to account for regional differences or to develop differing monitoring and reporting protocols.

#### 8 APPROACH TO SETTING AIR QUALITY INVESTIGATION LEVELS

#### 8.1 APPROACHES TO SETTING AIR QUALITY STANDARDS AND GUIDELINES

<sup>50</sup> The process of standard setting involves a range of environmental, health, technical, social, economic, political, legislative and cultural considerations. The selection of a standard can be done by various means, including risk assessment (eg health and/or ecological risk assessment), the adoption of overseas standards or the use of expert panels.

- <sup>5</sup> Health risk assessment (HRA) is a systematic approach for characterising the nature and magnitude of risks arising from environmental hazards such as air pollution. HRA can be either qualitative or quantitative and takes into account a range of factors including the exposure to the pollutant and potential health effects arising from such exposure. The HRA process has to take into account groups within the population that may be more sensitive to
- <sup>10</sup> the effects of a pollutant than the general population, eg, children and the elderly. As outlined in the Risk Assessment Taskforce report to NEPC (NEPC 2000), risk assessment usually involves five stages:
  - Issues identification
  - Hazard identification
  - Dose-response assessment
    - Exposure assessment

15

• Risk characterisation

A full quantitative risk assessment would require all stages outlined above to be conducted.
 However, a qualitative approach would not require all stages to be undertaken with the same level of rigour. With the limited data sets available for air toxics in Australia there are insufficient data for a full quantitative risk assessment to be undertaken.

National air quality standards for the air toxics under consideration in the proposed NEPM
 have only been set in a limited number of countries. No one country has set standards for all the pollutants. In setting the proposed investigation levels a range of approaches have been used, all of which incorporate elements of HRA to varying degrees.

In the UK, where an ambient air quality standard for benzene has been adopted, expert panels are used to make recommendations to Government on potential air quality standards. In making such recommendations a qualitative risk assessment was undertaken. A full exposure assessment and quantification of risk was done in subsequent stages of the development of the standard.

In the USA, there are no national air quality standards for air toxics. Nationally ambient levels of air toxics in the USA are controlled by setting emission limits for industry and through control of motor vehicle emissions. Individual states, such as California and Texas, have set ambient air quality objectives aimed at the protection of human health. These values are developed using risk assessment approaches. However, full exposure assessment and risk characterisations are not conducted.

WHO guideline values is derived through a similar process, with no exposure assessment or risk characterisation being conducted. The WHO Guidelines for Air Quality (2000) explicitly state that to convert the guidelines to ambient air quality standards consideration must be

- <sup>45</sup> given to the local (ie national) situation including exposure assessment, risk characterisation, acceptable levels of risk and economic considerations in each country. These vary significantly from country to country and it is therefore not appropriate to adopt these guideline values as ambient air quality standards for Australia without consideration of these other parameters. Therefore, prior to adopting any overseas standards an assessment
- <sup>50</sup> of the process and assumptions used in the derivation of the standards would have to be undertaken to ensure that they are appropriate for Australia.

#### 8.1.1 Qualitative and quantitative risk assessment 5

As discussed above, the risk assessment process involves five stages. In any risk assessment process the first three stages of the process - issues and hazard identification and doseresponse assessment - will be undertaken. However at this point the decision can be made as to whether to proceed with a full quantitative assessment, which involves a quantification

of the risk to the population through the risk characterisation stage, or to proceed with a 10 qualitative assessment. The main difference between a qualitative and a quantitative risk assessment is that a qualitative assessment does not involve rigorous risk characterisation.

A qualitative risk assessment involves the identification of the adverse health effects that may be experienced after exposure to a particular hazard, eg air toxics, (hazard 15 identification) and the concentrations at which these effects are observed (dose-response assessment). For threshold pollutants, (ie those pollutants for which there is a level below which no adverse effects are observed), this may involve the identification of a No Observed Adverse Effects Level (NOAEL) or a Lowest Observed Adverse Effects Level (LOAEL). To derive air quality standards from these levels a range of uncertainty or 'safety' factors are 20 applied. This approach is commonly used to set air quality standards for a range of noncarcinogenic substances.

A quantitative risk assessment involves both exposure assessment and risk characterisation phases and leads to an estimate of either the risk to the population expressed, for example as 25 'one in a million' (as in carcinogenic risk assessment) or as the number of people affected eg 200 premature deaths per year. A full quantitative risk assessment requires extensive air monitoring data or appropriate modelling data to accurately assess the exposure of the population and the associated risk. All approaches require a judgement as to what is an 'acceptable' level of risk to the population. 30

#### 8.1.2 Benchmark Dose

The benchmark dose approach provides an alternative way of setting standards it sets a threshold level associated with a low level of increased risk above background. This approach is used by USEPA (2000) to set Reference dose concentrations (RfCs) for noncarcinogenic pollutants and by Environment Canada in deriving air quality guidelines/standards.

The NHMRC has developed guidelines for the assessment of carcinogenic soil contaminants which utilises a modified benchmark dose approach (NHMRC, 1999). These guidelines were 40 adopted by the NEPC in the contaminated sites NEPM.

The benchmark dose is usually associated with a 5% increase in risk of an adverse effect occurring. Uncertainty or 'safety' factors to take account of types of studies used in toxicity assessments and the data available (eg occupational exposures, laboratory animal 45 experiments, duration and levels of exposures) are then applied to derive a guideline or standard that would be protective of sensitive members of the general population.

The advantages of the benchmark dose approach include:

- it takes into account the entire dose response curve rather than a single dose as is done in 50 • the determination of the NOAEL:
  - the dose-response curve is not extrapolated to levels well below the experimental range as is done in the traditional carcinogenic risk assessments using unit risk factors;

35

- the approach is consistent across a range of studies and health endpoints as well as for carcinogens and non-carcinogens; and
  - it uses all relevant information.

If the experimental data available do not cover a sufficiently wide range of concentrations, it may not be possible to determine the shape of the dose-response curve.

#### 8.1.3 Carcinogenic risk assessment

Carcinogenic risk assessment is usually conducted using unit risk factors. A unit risk factor is the risk of the incidence of cancer resulting from a lifetime exposure (usually 70 years) to 1 µg/m<sup>3</sup> of a carcinogenic substance. In deriving air quality standards from this approach an 'acceptable' level of risk is specified and the concentration of the pollutant corresponding to that level of risk is determined. Bodies such as the World Health Organization (WHO), USEPA and California EPA Office of Environmental Health Hazard Assessment have all developed unit risk factors for carcinogens, however the values can vary by an order of magnitude depending on the studies used and the model chosen to derive the factor.

8.1.4 Summary

Each of the methods outlined above has advantages and disadvantages. In adopting overseas standards an assessment of the process undertaken to set the standards must be undertaken to ensure that the basis of the standard is appropriate for the Australian situation. In using risk assessment approaches, whether they are qualitative or quantitative, judgements must be made about the level of risk that is inherent in the resulting air quality standard. This may involve selection of appropriate 'safety' factors or defining explicit levels of 'acceptable' risk. Such judgements require input from a wide range of stakeholders and can be the subject of significant debate.

ean be the subject of significant debate.

#### 8.2 DERIVATION OF INVESTIGATION LEVELS

The goal of the proposed NEPM is focused on the collection of data in a nationally consistent manner to enable a better assessment of the situation with respect to air toxics in Australia in the future. The monitoring protocol associated with the proposed NEPM focuses monitoring at sites where significantly elevated levels of air toxics would be expected.

Given the lack of data for air toxics in Australia and the nature of the proposed NEPM, it is considered that it is not possible at this time to develop standards using any of the risk
assessment approaches outlined above. Therefore, an assessment has been made of overseas standards and guidelines for their applicability as investigation levels in the Australian context.

To assist in the assessment of ambient levels of air toxics, investigation levels have been based on overseas approaches and used as benchmarks. Only overseas standards or guidelines that had been derived for a similar purpose to that proposed in the Air Toxics NEPM were selected in order to avoid a level that may be too stringent or not stringent enough. In this context, it should be noted that investigation levels are levels that, if exceeded, may trigger some appropriate form of further investigation.

50

35

- In selecting overseas standards or guidelines for use as investigation levels for the air toxics 5 under consideration in the Air Toxics NEPM a range of criteria (including similarity of purpose) were applied. For each standard or guideline, the approach to setting the standard or guideline had to be identified and assessed for its suitability for Australia.
- The criteria used in the assessment included: 10
  - Evaluation of the studies used in the identification of health endpoints and dose response relationships (unit risk factors, NOAELs, LOAELs etc)
  - Assessment of the quality of the information, especially the quality of the exposure data
  - Were the health endpoints relevant for Australia? •
- What levels of risk are associated with the standards for carcinogens is this acceptable 15 • for Australia?
  - What uncertainty factors have been used and why?
  - Assessment of the Unit Risk factors and Dose response data used •
  - Comparison of data with those derived from any Australian studies ٠
  - Evaluation of the purpose of the standards do they fit with the intent of the NEPM?
    - Is the inhalation route the one that has been assessed?

These criteria are consistent with the approaches taken overseas in the evaluation of key studies and standards.

25

30

20

A review of overseas standards for the five air toxics under consideration has revealed that very few standards have been developed and those that have, have been derived for a The UK has standards for benzene and PAHs, the European variety of purposes. Commission has a standard for benzene and the WHO has guidelines for formaldehyde, toluene and xylenes. New Zealand has ambient air quality guidelines for three of the air toxics under consideration.

The Office for Environmental Health Hazard Assessment (OEHHA), part of the California EPA, has developed Reference Exposure Levels (RELs) for a range of pollutants, including the five air toxics under consideration in this NEPM. The RELs have been developed for use 35 in the California EPA's hot spots program. They are health-based numbers and have been derived through the use of No Observed Adverse Effects Levels (NOAEL) or Lowest Observed Adverse Effects Levels (LOAEL) with appropriate safety or uncertainty factors applied. In some cases the Bench Mark Dose (BMD) approach has also been used with appropriate uncertainty factors.

40

The RELs undergo extensive scientific peer review and public consultation processes before being adopted by OEHHA. A scientific advisory panel is set up to provide review and advice on the development of the RELs.

45

The Agency for Toxic Substances and Disease Registry (ATSDR) is part of the National Institute of Health in the US. ATSDR has developed Minimal Risk Levels (MRLs) for 118 substances including formaldehyde, toluene and xylenes. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of

adverse non-cancer health effects over a specified duration of exposure. They are intended to 50 serve as screening levels and are used by ATSDR health assessors and others to identify <sup>5</sup> contaminants and potential health effects that may be of concern at hazardous waste sites. MRLs have been determined for inhalation, ingestion and dermal routes of exposure.

ATSDR uses the NOAEL/uncertainty factor (NOAEL/UF) approach to derive MRLs for hazardous substances. They are set below levels that, based on current information, might cause adverse health effects in the people most sensitive to such substance-induced effects.

MRLs are derived for acute (1-14 days), intermediate (>14-364 days), and chronic (365 days and longer) exposure durations, and for the oral and inhalation routes of exposure. MRLs are generally based on the most sensitive substance-induced end point considered to be of relevance to humans. Exposure to a level above the MRL does not mean that adverse health effects will occur. MRLs are intended to serve as a screening tool to help public health professionals decide where to look more closely.

Proposed MRLs undergo a rigorous review process. They are reviewed by the Health Effects/MRL Workgroup within the Division of Toxicology and expert panel of external peer reviewers; the agency wide MRL Workgroup, with participation from other federal agencies, including EPA; and are submitted for public comment through the toxicological profile public comment period. Each MRL is subject to change, as new information becomes available, concomitant with updating the toxicological profile of the substance.

25

10

15

All of the standards and guidelines discussed above were assessed for their suitability for use in the Air Toxics NEPM.

#### 8.3 HEALTH ENDPOINTS

- <sup>30</sup> As part of the development of the proposed NEPM, extensive reviews of the health effects of the five air toxics under consideration were undertaken. These reviews, which highlight the key epidemiological and toxicological studies used as the basis for overseas standards, are available on the NEPC website. Representatives of the health sector in Australia appraised these reviews.
- 35

The health endpoints selected as being appropriate as the basis of air toxics standards in Australia are:

- Benzene cancer,
- PAHs cancer,
- Formaldehyde respiratory irritation,
  - Toluene respiratory irritation and central nervous system (CNS) effects, and
  - Xylenes respiratory irritation.

Consultation with the health sector indicated support for these health endpoints as appropriate as the basis for air quality standards for air toxics in Australia. They are also consistent with the health endpoints used in the derivation of overseas standards and guidelines for ambient air quality.

#### 8.4 ASSESSMENT OF OVERSEAS STANDARDS

<sup>50</sup> In reviewing the basis for the standards as a starting point for the proposed NEPM, it quickly became apparent that the process used to derive the overseas standards was, in many cases, not transparent. The exceptions to this were California EPA (OEHHA), ASTDR MRLs and

WHO Ambient Air Quality Guidelines (limited), European Commission Standards, and New 5 Zealand guidelines.

The UK standards for benzene and benzo(a)pyrene have been developed by an Expert Panel. The standards are based on expert judgement but the process used is not transparent. The WHO guidelines are based on the IPCS documents but the actual derivations of the standards are not transparent.

#### 8.4.1 California EPA RELs

The acute RELs developed by OEHHA for formaldehyde, xylenes and toluene are for a 1hour averaging time. They have been developed through an extensive assessment of the 15 epidemiological and toxicological literature and identification of NOAELs, LOAELs or in the case of formaldehyde, a benchmark concentration, and the application of uncertainty factors. A modified Haber's Law has been applied to convert averaging times from the experimental exposure times (30 minutes to 6 hours) to a consistent 1-hour averaging time (OEHHA, 1999; Rozman, 2000; Halmes et al., 2000). 20

A summary of the key studies and the process used to derive the individual RELs is outlined below.

*Formaldehyde* 25

10

The REL for formaldehyde is based on the protection of mild and moderate eye irritation which is the most sensitive health endpoint relevant to the human population. The key study used is that of Kulle et al., (1987) which looks at the results of a controlled exposure study of 19 non-asthmatic, non-smoking human subjects. This study was a key study in the health reviews and was highlighted by a number of agencies in the development of

30 guidelines and standards.

It should be noted that eye irritation was not the health endpoint identified by the health specialists for use in the proposed NEPM. However, it is more sensitive than the respiratory irritation endpoint originally identified.

OEHHA have used a benchmark concentration of 0.44 ppm (3-hour averaging time) based on the 95% lower confidence limit. An uncertainty factor of 10 has been applied to account for sensitive individuals within the general population. A factor of 10 has been used given the wide variability in response to formaldehyde that has been reported in the literature. The study by Pazdrak et al., (1993) was used as a supporting study as it also shows adverse effects associated with exposure to low levels of formaldehyde.

The resulting REL is 0.076 ppm for a 1-hour averaging time. This level is thought to be protective of the range of health effects associated with exposure to formaldehyde as it has 45 been derived for the most sensitive health endpoint.

#### Toluene

50

35

40

The acute REL for mild adverse effects of toluene is based on protection of CNS effects and eye and respiratory irritation. The key study on which the REL is based is that of Andersen et al (1983). This study, which was highlighted as a key study in the health reviews done as part of the development of this NEPM, was a controlled exposure study in which 16 young <sup>5</sup> healthy males were exposed to toluene. The exposure time was 6-hours and the LOAEL and NOAEL identified are 100 ppm and 40 ppm respectively.

OEHHA has used the NOAEL with an uncertainty factor of 10 applied to account for sensitive individuals within the general population. In addition to the REL for mild adverse effects, a REL has been derived for protection of serious adverse effects. OEHHA has determined that reproductive and developmental effects are the health endpoint to be protected. The basis for the serious adverse effects REL is an animal study showing toxicity to the foetus (IRDC, 1985). A NOAEL of 500 ppm (6 hour exposure) for fetotoxic effects has been determined. OEHHA has applied an uncertainty factor of 100 to account for both interspecies and intraspecies differences.

The resulting RELs are, for mild effects, 9.8 ppm for a 1-hour averaging time, and for severe effects, 5 ppm for a 6-hour averaging time. Converted to the same averaging time, the REL for mild effects is the more protective of the two values.

20

25

30

40

*Xylenes* 

The acute REL for xylenes is based on the protection of eye, nose and throat irritation. Irritation is considered to be the most sensitive human health effect occurring at exposure to low levels of xylenes. OEHHA has used the study of Hastings et al., (1984) with supporting studies by Carpenter et al., (1975) and Nelson et al., (1943).

The Hastings et al., (1984) study exposed 15 healthy human volunteers to 30 minutes exposures of xylenes. The subjects reported eye, nose and throat irritation. The NOAEL derived from this study was 100 ppm 30 minute averaging time). OEHHA applied an uncertainty factor of 10 to this value to account for sensitive individuals within the general population.

The resulting REL for a 1-hour averaging time is 5 ppm.

#### 35 8.4.2 ATSDR MRLs

The ATSDR MRLs are derived through a similar process to that of OEHHA in that NOAELs and LOAELs are determined and uncertainty factors applied. The acute RELs are derived for averaging periods of between 1 and 14 days, using the averaging period of the experimental exposure and adjusting for continuous exposure for 7 days per week over a lifetime. The MRLs are derived for the most sensitive human health effect for a given exposure route and duration.

#### Formaldehyde

The MRL for formaldehyde has been derived using the study by Pazdrak et al., (1993) as the key study. This study investigated the impact of formaldehyde exposure on the severity of symptoms of eye and nose irritation in occupationally exposed non-smoking adults. These are the same health endpoints used in the OEHHA REL development. The ATSDR identified a LOAEL of 0.4 ppm (2 hour averaging time) for these health effects. An uncertainty factor of 3 was used for the use of a LOAEL and a further factor of 3 to account for sensitive individuals within the general population.

The factor of 3 for the use of a LOAEL was considered to be justifiable as the symptoms were mild and reversible and the clinical significance of the changes in nasal lavage fluid is uncertain at present.

<sup>5</sup> The factor of 3 for sensitivity within the general population was considered to be justifiable as the study group was a potentially sensitive group as they displayed dermal (skin) sensitivity to formaldehyde.

The resulting MRL is 0.04 ppm for a 1-14 day exposure.

10

It should be noted that OEHHA used the Pazdrak et al., study as a supporting study rather than the key study due to uncertainties in the measures of exposure.

#### Toluene

- The MRL for toluene is based on the study of Andersen et al., (1983) as it was the only study in which a NOAEL has been identified. Studies by Baelum et al., (1985), Eccheveria et al., (1991) and Rahill et al., (1996) have been used as supporting studies. The key health endpoints identified in the Andersen study are CNS effects and respiratory irritation. A NOAEL of 40 ppm (1 hour averaging time) for CNS effects was identified. An uncertainty factor of 10 has been applied to account for sensitive individuals within the general
- population.

The resulting MRL is 1 ppm for 1-14 day exposures.

25 Xylenes

The MRL for Xylenes is based on CNS effects in an acute inhalation exposure study by Dudek et al (1990), with supporting studies by Gamberale et al (1978) and Carpenter et al (1975). The Dudek et al (1990) study used 10 volunteers exposed for xylenes at 100ppm for 4 hours and showed that subjects had prolonged reaction times (CNS depressant effect) following exposures. This was considered to be a LOAEL by the investigators. Gamberale et

- following exposures. This was considered to be a LOAEL by the investigators. Gamberale et al (1978) confirmed that the 100ppm is near the threshold for adverse effects as no effects on reaction times were seen in 15 volunteers exposed to xylenes at 100 and 299ppm for 70 minutes. However when subjects exercised for the first 30 minutes of the 70 minute exposure to 299ppm xylenes, reaction times were increased and short term memory was
- <sup>35</sup> impaired. The acute MRL of 1 ppm was derived from the LOAEL of 100ppm, 4 hour exposure. However no information is provided in derivation of uncertainty factors for the use of a LOAEL, intraspecies variability, or exposure time conversion from 4 hours to 1 hour.

### 8.4.3 WHO Ambient Air Quality Guidelines

- The WHO guidelines are based on the IPCS reviews developed primarily in the late 1980s and early 1990s. Although the guidelines have only just recently been released they are based on early data that are outdated in many cases. The ambient air quality guidelines are conservative and do not necessarily apply as trigger levels. They do not have any considerations of social and economic issues in their development. The standards do not in all cases apply to the health endpoints identified in the Health Specialist Workshop held in
- <sup>45</sup> all cases apply to the health endpoints identified in the Health Specialist Workshop held in Melbourne in June 2002.

For benzene and PAHs the WHO do not have air quality guidelines but sets unit risk factors. This means that a standard would have to be set from a predetermined level of risk. The health reviews conducted for benzene as the basis for the unit risk factors were done in 1993

and were reviewed in 1998. For benzo(a)pyrene the basis of the unit risk factor was a review

50

of the epidemiological data conducted in 1973.

<sup>5</sup> For formaldehyde, toluene and xylenes the ambient air quality guidelines that have been developed have been derived from the IPCS reviews published in 1989, 1985 and 1997 respectively.

The unit risk factor and health guidelines for non-carcinogenic effects for formaldehyde were derived in 1989 based on reviews of the health studies from the early 1980s. These reviews do not include recent evidence associated with the mechanism of action for formaldehyde. The inclusion of this data is critical in the development of any standards for formaldehyde and the lack of consideration in the development of the WHO guideline makes it unsuitable for adoption in the proposed NEPM.

15

20

25

In addition, it appears from the IPCS review that the guideline was developed as an indoor air quality guideline. No safety factors have been applied and there is no documentation as to why this has occurred and what this means for the protection of sensitive groups within the population. The basis of the standard is a NOAEL but the key study used as the basis for the guideline is not identified, making it hard to justify the use of this value.

A similar situation exists for the guidelines that have been developed for toluene and xylenes. The key studies have not been identified and the justification for the safety factors has not been provided. For toluene a safety factor of 1260 has been applied to a LOAEL. For xylenes a safety factor of 60 has been applied. The derivation of these safety factors is not

provided, making it difficult to assess the level of protection afforded these guidelines.

The WHO guidelines apply to a range of averaging times:

- Formaldehyde 30 minutes
- 30 Toluene 1 week
  - Xylenes 24 hours

### 8.4.4 UK Air Quality Standards

The UK air quality standard for benzene is derived from the results of occupational studies.
From the results of epidemiological studies, the expert panel estimated that exposure of workers to 500 ppb benzene over a working lifetime would not result in an increased risk of leukaemia that could be detectable by any feasible study. The basis for this judgement is not provided. A series of safety factors have then been applied – the justification for these is also not provided. The basis for the final recommendation of 5 ppb as a running annual average
is that it would provide an 'exceedingly small risk to health'. Again the basis for this statement has not been provided. This makes the use of this value difficult to justify for use in the NEPM.

For PAHs as benzo(a)pyrene, the standard has been derived from the results of an occupational exposure study of Canadian aluminium smelter workers (Armstrong et al 1994). A level of 10-99 µg/m<sup>3</sup> was identified as the LOAEL for a 50% increase in lung cancer over a working lifetime for total PAHs. The corresponding level of benzo(a)pyrene was estimated to be 0.25 to 2.5 µg/m<sup>3</sup>. The assumptions used are not provided. A number of safety factors were then applied. A factor of 10 was used in converting from a LOAEL to a NOAEL due to the fact that benzo(a)pyrene is a genotoxic carcinogen. Another factor of 10 was applied to go from a working lifetime to an entire lifetime of 70 years. Another factor of 10 was used in order to apply to the general population rather than workers. The resulting standard is 0.25 ng/m<sup>3</sup> as an annual average.

Impact Statement for the National Environment Protection (Air Toxics) Measure

5

The UK standard for PAHs is quite conservative and may be triggered in many areas of Australia that are impacted by woodsmoke. Many of the assumptions that have been made in the derivation of the standard are, as stated by the UK Department of Environment, quite arbitrary and may be difficult to defend.

10

45

The process used to derive the benzene standard is not as transparent as that for the derivation of the PAH standard.

#### 8.4.5 European Commission

- <sup>15</sup> The European Commission has established an air quality standard for benzene of  $5 \mu g/m^3$  as an annual average with a 100 percent tolerance level of 10  $\mu g/m^3$ . The  $5 \mu g/m^3$  level is a target to be met in 2010. The EC standard for benzene has been based on the protection of the general population from an increased risk of leukaemia. The standard was based on the recommendations of a Working Group made up of representatives from many European countries. A position paper developed by the Working Group was released in Sontember
- 20 countries. A position paper, developed by the Working Group, was released in September 1998 and provides the technical basis for the EC standards for benzene.

The EC Working Group on Benzene Position Paper (1998) stated that the Goodyear Pliofilm cohort was the most thoroughly studied group. For purposes of guideline derivation, the working group chose to use the risk calculation of Crump (1984) rather than to derive new estimates. It was considered to result in the highest plausible estimate of risk. A unit risk factor of 6 x 10<sup>-6</sup> was used as the upper bound risk calculation. This results in a lifetime risk of cancer of 2 in 100,000 associated with the EC standard of 5  $\mu$ g/m<sup>3</sup>.

- <sup>30</sup> The lowest unit risk factor which the group felt was likely to be plausible was in the order of 5 x 10<sup>-8</sup>, developed from a meta analysis of petroleum industry workers in UK and USA (Wong and Raabe 1995). However, the methodology for derivation of the unit risk factor is not explained in the position paper. For an excess lifetime risk of 1 in a million, the range of units converted into annual average concentrations is 0.21 to 20  $\mu$ g/m<sup>3</sup>. The Working Group
- recommended that this range should be taken as a starting point for developing proposals for a limit value, defined as an annual average concentration. The basis for the EC standard, as outlined in the Position Paper, is consistent with that used by other international bodies for developing guidelines/standards for benzene. The process is transparent and the assumptions are made clear. The level of scientific peer review and broad consultation is unknown.

The European Commission, in keeping with the process for benzene, have commissioned a working group to prepare a draft position paper for PAHs. This position paper is still in draft form and no resolution has been reached for a standard to be made. On this basis it is not appropriate to adopt the recommendations of the working group's draft report as an investigation level for the proposed NEPM.

### 8.4.6 New Zealand Ambient Air Quality Guidelines

The New Zealand Ministry for the Environment has adopted air quality guidelines for three of the air toxics under consideration. These guidelines are used as monitoring values and have been adopted from overseas standards. The draft guidelines were subject to scientific peer review and a broad consultative process to assess their suitability for application in New Zealand.

- <sup>5</sup> The New Zealand AAQ guidelines state that "the guideline values apply to outdoor air wherever a person might reasonably be expected to be exposed to the contaminant over the relevant averaging period. Where the guideline value applies therefore depends on the specific contaminant, the relevant averaging period, and the spatial extent over which the guideline is likely to be breached. This is a precautionary approach, because if pollution
- levels at a peak site where someone may be affected are within the guideline value, then it is expected that pollution levels at other sites will also be within the guideline value". This approach is consistent with the intent of the application of the proposed NEPM.

For formaldehyde, the New Zealand guideline value has been adopted from the WHO guidelines (1996). This value is based on an identified NOAELs from animal studies with no safety factor applied, similar to the approach used in the 2000 WHO guidelines.

For benzene the New Zealand value has been adopted through a combination of the EC and UK approaches. The current guideline value is  $10\mu g/m^3$  as an annual average with a target of 3.6  $\mu g/m^3$  to be achieved by 2010. Using the WHO risk factors the current guideline value equates to a risk of leukaemia of approximately 4 in 100,000.

For PAHs (as benzo(a)pyrene), the WHO unit risk factor has been used to estimate an annual average value of 0.3 ng/m<sup>3</sup> equating to a lifetime risk of lung cancer of 1 in 100,000. It is the
intent of the New Zealand Ministry to review this guideline in 2007 and move toward a toxic equivalence scheme for PAHs, similar to that used for organochlorines.

The process through which the New Zealand guideline values have been derived is transparent and all documentation is available from the Ministry for the Environment.

#### 30

45

20

#### **8.5 PROPOSED INVESTIGATION LEVELS**

In evaluating the most appropriate standards/guidelines for use as investigation levels in Australia the assessment criteria were applied. For formaldehyde, toluene and xylenes it is considered that the science behind the OEHHA RELs and the process of scientific peer review undertaken are the most sound of those assessed. It is proposed that they be adopted as investigation levels for the purposes of the Air Toxics NEPM. However, the averaging time for the RELs is 1 hour. This is impractical from a monitoring perspective. To derive the 1-hour values, OEHHA have applied a modified Haber's Law to convert from the experimental time (usually several hours) back to a consistent averaging time of 1-hour (OEHHA, 1999).

For the purposes of the proposed NEPM, monitoring will be conducted over 24-hour periods. Monitoring over shorter periods is unlikely to measure concentrations that would be detectable through the analytical methods. It is therefore proposed that, instead of applying Haber's law to the 1-hour values, it would be applied to the experimental times to convert to a consistent 24-hour averaging time. The process for the derivation of the proposed investigation levels is outlined below.

Impact Statement for the National Environment Protection (Air Toxics) Measure

Formaldehyde	
Key Study	Kulle et al., (1987)
Study population	19 asthmatic, non-smoking human subjects
Exposure method	0.5-3 ppm
Critical effects	mild and moderate eye irritation
LOAEL	1 ppm
NOAEL	0.5 ppm
Benchmark concentration	0.44 ppm (BC <sub>05</sub> )
Exposure duration	3 hours
Extrapolated 24-hour concentration	$0.15 \text{ ppm} (0.44^2 \text{ ppm x } 3 \text{ h} = \text{C}^2 \text{ x } 24 \text{ h})$
LOAEL uncertainty factor	not applicable
Interspecies uncertainty factor	1
	10
Intraspecies uncertainty factor	10
Cumulative uncertainty factor	
Proposed investigation level Conversion factor	0.015 ppm (24 hour averaging time)
	1 ppm = 1.24 mg/m <sup>3</sup> at 25°C
Toluene	
Key Study	Andersen et al., 1983
Study population	16 young healthy males
Exposure method	Inhalation
Critical effects	CNS effects (impaired reaction time and symptoms of
	headache, dizziness, a feeling of intoxication) and slight eye
	and nose irritation
LOAEL	100 ppm
NOAEL	40 ppm
Exposure duration	6 hours
Extrapolated 24-hour concentration	20 ppm ( $40^2$ ppm x 6 h = C <sup>2</sup> x 24 h)
LOAEL uncertainty factor	1
Interspecies uncertainty factor	1
Intraspecies uncertainty factor	10
Cumulative uncertainty factor	10
Proposed investigation level	2 ppm (24 hour averaging time)
Conversion factor	1 ppm = 3.75 mg/m³ at 25°C
Xylenes	
Key Study	Hastings et al., 1984 (with support from Carpenter et al., 1975;
	Nelson et al., 1943)
Study population	50 healthy human volunteers
Exposure method	30 minute exposures to 430, 860 or 1720 mg/m <sup>3</sup> xylene
	(technical grade)
Critical effects	subjective reports of eye, nose and throat irritation
LOAEL	860 mg/m <sup>3</sup>
NOAEL	430 mg/m <sup>3</sup> (100 ppm)
Exposure duration	30 minutes
Extrapolated 24-hour concentration	2 ppm (100 ppm x 0.5 h = C <sup>1</sup> x 24 h)
LOAEL uncertainty factor	1
Interspecies uncertainty factor	1
Intraspecies uncertainty factor	10
Cumulative uncertainty factor	10
Proposed investigation level	0.2 ppm (24 hour averaging time)
Conversion factor	1 ppm = 4.34 mg/m <sup>3</sup> at 25°C

For benzene, it is considered that the EC maximum tolerance level of 0.003 ppm should be adopted as an investigation level in the proposed NEPM. (This contrasts with the EC annual standard for benzene is 0.0015 ppm which applies everywhere including at peak sites.) The EC has the most transparent and extensive health review of any of the agencies that have developed standards for benzene. The level of protection offered by the EC maximum tolerance level is considered to be reasonable for the purpose of this NEPM.

The benzene Investigation Level is set to enable the accumulation of adequate data over the life of this NEPM to enable judgements to be made on the conclusion of this NEPM on the adoption of an Investigation Level of 0.0015ppm consistent with the approach adopted by the European Community, the United Kingdom and New Zealand, or some other level.

For benzo(a)pyrene (as a marker for PAHs) it is proposed to adopt the New Zealand air quality guideline value of  $0.3 \text{ ng/m}^3$  as an annual average. This is considered to be appropriate given the transparency in the process used to derive the number and the broad consultative and review processes that were undertaken.

Although the UK standard has a similar value, the process used to derive involves a number of unjustified assumptions that makes it difficult to adopt in Australia. In addition the application of the New Zealand Guidelines is consistent with the NEPM in that they apply where people live and are potentially exposed. As with the benzene investigation level, the level of protection afforded by the New Zealand Guideline is considered reasonable for the purposes of this NEPM.

The proposed investigation levels are summarised below:

30

35

15

20

25

Air Toxic	Averaging Time	Investigation Level
Benzene	Annual average	0.003ppm
BaP (as a marker for PAHs)	Annual average	0.3 ng/m <sup>3</sup>
Formaldehyde	24-hour	0.015 ppm
Toluene	24-hour	2 ppm
Xylenes	24-hour	0.2 ppm

 Table 8-2: Proposed investigation levels

#### **Conversion Factors**

Conversion factors for each air toxic are provided for convenience. The conversion factors are calculated from first principles assuming an ideal gas resulting in the equation.

Concentration in grams/m<sup>3</sup> at  $25^{\circ}$  Celsius and 101.3kPa = (Concentration in ppm) x 1000 x (273.15/298.15) x Molecular Weight

Pollutant	Conversion factor
Benzene	3190
Benzo[a]pyrene	10310
Formaldehyde	1230
Toluene	3770
Xylenes	4340

<sup>5</sup> Thus to convert from parts per million by volume to micrograms/cubic metre at **25° Celsius and 101.3 kPa**, use the equation below.

Concentration inµg/m<sup>3</sup> = (Concentration in ppm) x Conversion factor

10 Note:

As the standards have been developed using data at 25° Celsius and 101.3kPa then converted, data should be at this temperature and pressure to ensure relevance to health data.

#### 15 9 IMPACTS OF THE NEPM

#### 9.1 INTRODUCTION

It is important to recognise that the only obligations imposed by the proposed air toxics NEPM will be assessment, monitoring and reporting requirements placed on jurisdictions. It is further recognised that public concern may lead to pressure for accelerated and/or additional programs to control emissions of the five air toxics subject to the proposed NEPM, where monitoring reveals levels of air toxics above the proposed investigation values. Whilst this is not a direct consequence of the NEPM, it may result in a range of indirect effects. There is potential for these indirect effects to impact on industries due to air quality management activities by jurisdictions as a result of their assessment of monitoring data.

25

20

Under the proposed NEPM, all governments will be expected to initiate the assessment of Stage 1 sites at the commencement of the proposed NEPM. Monitoring at Stage 2 sites is expected to commence after the assessment is completed and as jurisdictional resources permit. There is no other requirement placed upon governments.

30

45

All state and territory governments have active air quality management programs to improve ambient air quality and, while these are expected to continue into the future, the proposed NEPM does not embody any extension or modification of such programs. Each government will continue to assess the priority to be given to air quality management initiatives in the context of overall government programs. However the proposed NEPM

initiatives in the context of overall government programs. However the proposed NEPM will provide a sound basis for the generation of a significantly improved national dataset that will greatly improve jurisdictional ability to assess the extent of any problems in the major airsheds in relation to air toxics. The proposed NEPM will therefore provide information that will assist governments in setting priorities for various air quality management programs.

Through the establishment of monitoring and reporting protocols for air toxics and investigation levels to assess the results of monitoring, the proposed NEPM provides a tool for communicating information to the public on ambient air quality in urban areas and for assessing the effectiveness of air quality management programs. It also provides a sound database for future studies on the risk posed by air toxics to the health of the Australian population.

This in turn should lead to more cost-effective programs, better priority setting by governments, improvements in infrastructure development planning, more informed choices by individuals and consequential reduction in the risk of adverse health effects posed by exposure to air toxics and possibly behavioural change. Overall, the adoption of the proposed NEPM should, through time, contribute significantly to improved protection of public health.

The proposed NEPM deals only with the assessment of air toxics by government, therefore 5 any direct costs are incurred only by governments. Jurisdictions will need to assess the most cost-effective means of complying with the monitoring protocol.

Programs to reduce emissions of air toxics will continue and the proposed NEPM will provide a better means of assessing the effectiveness of these programs and the targeting of 10 resources. The range of actions currently being undertaken in relation to industry and motor vehicle emissions, in particular, are outlined in this Impact Statement and are likely to continue. They are not to be construed as constituting actions that would flow from the adoption of the proposed NEPM.

15

20

30

#### 9.2 **COST OF DESKTOP ASSESSMENT AND MONITORING**

Jurisdictions already conduct routine air monitoring for the criteria pollutants but do not have similar extensive networks for air toxics. Most monitoring for air toxics has been on a campaign basis and has focussed on sites within urban areas. Very little monitoring of air toxics has been conducted in regional centres.

The methods available to jurisdictions for monitoring the selected air toxics are as follows:

- United States Environmental Protection Agency Compendium Method TO-11A. • Determination of Formaldehyde in Ambient Air Using Adsorbant Cartridge Followed by 25 High Performance Liquid Chromatography (HPLC) [Active Sampling Methodology] -January 1999,
  - United States Environmental Protection Agency Compendium Method TO-14A. Determination Of Volatile Organic Compounds (VOCs) In Ambient Air Using Specially Prepared Canisters With Subsequent Analysis By Gas Chromatography – January 1999,
  - United States Environmental Protection Agency Compendium Method TO-15. Determination Of Volatile Organic Compounds (VOCs) In Air Using Specially-Prepared Canisters And Analysed By Gas Chromatography/Mass Spectrometry (GC/MS) -January 1999,
- United States Environmental Protection Agency Compendium Method TO-13A. 35 Determination of Polycyclic Aromatic Hydrocarbons (PAHs) Using Gas Chromatography/Mass Spectrometry (GC/MS) – January 1999.
- Monitoring for the five air toxics designated in the NEPM can be carried out using the methods and protocols detailed in the NEPM and specified above. As well as benzene, 40 toluene and xylenes, formaldehyde can be determined using USEPA Method TO15 provided suitable canisters are used and method validation is carried out. If TO15 can be validated for formaldehyde then costs will reduced as jurisdictions will only have to set up for USEPA Method TO15 and USEPA Method TO13-A.
- 45

Jurisdictions are able to use a combination of the methods designated in the NEPM thus it is not possible to carry out precise costing as it will depend on the methods chosen, the costs of installation of individual sites and whether a jurisdiction can carry out its own analysis or must seek an external laboratory. Indicative costs for monitoring have been summarised in Table 9-1.

50

Some jurisdictions are already carrying out air toxics monitoring and analysis and monitoring costs to those jurisdictions could be significantly less than those indicated.

Table 9-1: In	dicative estimates of	the costs of monitor	oring of air toxics
---------------	-----------------------	----------------------	---------------------

	USEPA Method TO11-A for formaldehyde	USEPA Method TO14-A for benzene, toluene, and xylenes	USEPA Method TO15 for benzene, toluene, xylenes and formaldehyde	USEPA Method TO13- A for benzo(a)pyrene and other PAHs	Meteorological Equipment
Equipment	\$3,500 for TO11 sampler	\$16,500 per unit for AVOCS system and canisters	\$19,000 per unit for AVOCS system and canisters	\$11,000 per unit for PUF samplers	\$5,000 to \$10,000 depending on equipment chosen for a new site
Analysis	\$1,000 per site per annum internal analysis	\$5,000 per site per annum internal analysis \$21,600 per site per annum external laboratory analysis	<ul> <li>\$5,000 per site per annum internal analysis</li> <li>\$21,600 per site per annum external laboratory analysis</li> </ul>	\$9,000 external \$3,000 internal per site per annum	Data validation Time approximately ½ day per site per month
Infrastructure such as security cage, mains power connection, site rental	\$3,000 for external sampling unit (flow controller, timer, box)	\$6,000 per site then \$3,000 per installation	\$6,000 per site then \$3,000 per installation	\$2,000-\$6,000 per site then \$3,000 per installation	\$6,500 installation of meteorology equipment, includes allowance for soil test, engineering calculations, electrical power and mast installation
Maintenance	\$5,000 for calibration of flow meter, site visits	\$4,000 per annum (repairs, power, spare parts, vehicle mileage)	\$5,000-\$8,000 per annum (repairs, power, spare parts, vehicle mileage)	\$5,000-\$8,000 per annum (repairs, power, spare parts, vehicle mileage)	\$2000 per annum for calibration, data processing
Staff estimates as a fraction of full time per site per year	0.17fte	0.21fte	0.25fte	0.27fte	0.1fte

- <sup>5</sup> The above estimates have been developed with the assistance of jurisdictions with experience in the various monitoring techniques. As can be seen from cost estimates, internal analysis costs are much less than external ones but require laboratories to have suitable equipment and staff already set up. For example a GC/MS system could cost around \$140,000 and an HPLC system around \$50,000 along with infrastructure and staff.
  - Methods USEPA TO14-A and USEPA TO-15 have been costed with automatic sampling devices. A cheaper, simple system could be set up for about \$3,000 but this would then require more staff attention.
- <sup>15</sup> Infrastructure costs to support additional staff could vary from one jurisdiction to another depending on available laboratory space, and support resources.

Thus there are a variety of options open to jurisdictions depending on their current capabilities, and resources.

20

25

50

10

#### 9.2.1 Desktop Assessment & Reporting

Costs incurred in undertaking desktop assessments will vary according to the amount of information readily held by jurisdictions. The size of the jurisdiction, traffic conditions, industrial base and geography will all influence the scope of the desktop assessment. For example, the desktop assessments for smaller jurisdictions may be relatively straightforward due to limited number of sources of air toxics. This may be a larger task for bigger jurisdictions, but they are also more likely to have existing information readily accessible to assist in the desktop assessment.

- The desktop assessment is required, as a minimum, twice during the period of the NEPM. This requires the assembly of relevant data on population, industry, road traffic and other information into a comprehensive summary to enable the prediction of stage 1 sites and stage 2 sites. The time taken to carry out this task could vary from a number of weeks for one person in a smaller jurisdiction to a month or two for a small team in the larger jurisdictions and is largely dependent on the ease of availability of the relevant information
- jurisdictions and is largely dependent on the ease of availability of the relevant information in individual jurisdictions. Reporting on the desktop survey would follow on from the survey.

#### 9.2.2 Accreditation

<sup>40</sup> Provided that jurisdictions already have accreditation as required under the Ambient Air NEPM, they would then need to add the additional techniques chosen from the required methods as stipulated in the reference method Table 9.1.

This would involve setting up procedures, documentation and preliminary and accreditation45 assessment.

- Accreditation cost estimate \$5,000 per method
- Setting up and documentation and procedures approx 6 to 8 weeks work for one person (or \$6-8,000)
- Internal cost estimates: indicative costs for training of staff (where required), based on an estimate of 2 weeks per person for training in instrument operation, calibration, maintenance and NATA procedures, are \$2000 per person per method.

#### 5 9.2.3 Benefits of Monitoring

The benefits achieved by this NEPM will be:

- Identification of peak areas which will allow resources to be prioritised in the management of these air toxics;
- Generation of a nationally consistent data set for the identified air toxics;
- Provide information on which jurisdictions can base a "first approximation" of any health risk posed to identified exposed populations
  - Provide monitoring data which can assist in evaluation of the implementation of the Diesel NEPM and new national fuel quality standards;
  - Provide data to assist in the review of the NEPM.
- 15

10

Jurisdictions are required to carry out a desktop study which will uncover any areas where the selected air toxics may pose a problem. This in itself is a useful exercise to assist in air quality management and has not yet been undertaken in all jurisdictions.

<sup>20</sup> Monitoring data can be used by jurisdictions to provide useful information that will permit better understanding and management of air quality issues throughout jurisdictions. These include possible health problems with air toxics and whether sufficient monitoring is being carried out to enable the assessment of the effectiveness of the Diesel NEPM and changing fuel standards.

25

Monitoring data generated will be used as part of the review of this NEPM and should fill knowledge gaps currently preventing jurisdictions from carrying out a full assessment of their regions. Thus monitoring data could form a useful tool in an area of ambient air quality where there is currently a lack of information for a complete assessment.

30

35

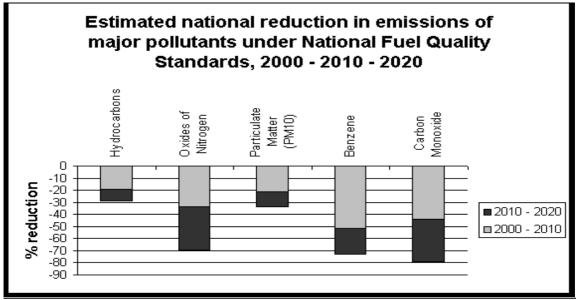
#### 9.3 MOTOR VEHICLE IMPACTS

#### 9.3.1 Motor Vehicle Design and Fuel Standards

Motor vehicles are a significant source of air toxics in urban areas and are estimated to contribute up to 58% of emissions of benzene, 50% of emissions of toluene, 56% of emissions of xylenes, 15% of emissions of PAHs and 34% of emissions of formaldehyde into ambient air in Australia (NPI 2000/2001).

The Australian Design Rules set emissions standards for motor vehicles. New Australian Design Rules have recently come into force with staged introduction of tighter emission standards. These standards are being introduced progressively from 2002 to 2006. To facilitate the introduction of new vehicle emission standards, national fuel quality standards have also been introduced through Commonwealth legislation. The national fuel quality standards set standards for maximum content of PAHs in diesel and benzene in petrol.

<sup>45</sup> The new vehicle emission standards and fuel quality standards will result in reduction of pollutants, including the air toxics under consideration, from the motor vehicle fleet. The estimated reductions for benzene and hydrocarbons (a composite category which includes toluene and xylenes) are identified in Figure-9.1. It is anticipated that the trends outlined in this figure are representative of all of the air toxics under consideration.



**Source**: SETTING NATIONAL FUEL QUALITY STANDARDS - A Review of Fuel Quality Requirements for Australian Transport Volume 2, March 2000 (scenario based on Euro 4 standards being introduced from 2008)

A review has commenced under the auspices of the Motor Vehicle Environment Committee to look at future vehicle emission standards (post 2006).

#### 15

10

#### 9.3.2 Motor Vehicle In-Service Performance

In addition to national measures such as motor vehicle design and fuel quality standards, individual jurisdictions employ other strategies to reduce emissions from motor vehicles, including emissions of the air toxics under consideration.

20

25

30

35

To improve the emissions profile of the in-service vehicle fleet, some jurisdictions have programs that encourage regular servicing and tuning of vehicles. Some jurisdictions also provide incentives to purchase cleaner new vehicles. The Diesel NEPM 1998 specifically addresses the issues of in-service performance of heavy duty diesel vehicles. Monitoring of air toxics under the proposed NEPM will provide information to the jurisdictions to assess the effectiveness of the implementation of the Diesel NEPM.

Additionally, programs targeting smoky vehicles have been introduced in most jurisdictions. The programs include community reporting and vehicle spotting by authorised officers via the 'ten second' rule. States also regulate aspects of vehicle operation including requirements for vertical exhausts for heavy-duty vehicles, and employ roadside inspections and enforcement provisions for poorly maintained or unroadworthy vehicles.

These strategies will facilitate management of all pollutants emitted from vehicles, including air toxics.

#### 5 9.3.3 Motor Vehicle Usage

The continued annual growth in vehicle kilometres travelled and fuel consumption by the motor vehicle fleet represents a significant challenge to urban air quality as motor vehicles are a major source of many pollutants including the air toxics under consideration.

- <sup>10</sup> Initiatives to manage the demand from travel include:
  - strategic transport and land use planning that encourages the integration of significant trip-generating land uses with non-motorised vehicular transport modes and public transport;
  - local traffic management initiatives, such as pedestrianisation;
- initiatives to manage travel demand, such as: providing infrastructure that supports alternatives to motor vehicles (such as bike paths), providing targeted information to people about their travel needs to help them make better decisions (such as the travel behaviour change programs currently being trialed in several Australian cities), and creating financial incentives that encourage the use of alternatives to the car; and
- education and general awareness raising campaigns to encourage motorists to minimise impacts associated with motor vehicle use.

The overall aim is to produce an urban form and transport system that support and encourage the use of alternatives to the motor vehicle, which in turn facilitates reduction in motor vehicle use.

25 motor vehicle use.

30

The introduction of the proposed Air Toxics NEPM will not lead to any direct impacts on motor vehicle manufacturers, retailers or fuel suppliers. The programs already in place at a national level relating to fuel quality and motor vehicle design rules will lead to reductions in the air toxics under consideration in this NEPM.

- If monitoring of air toxics near heavily trafficked roads indicates that the investigation levels contained in the Guideline in the proposed NEPM are exceeded, jurisdictions may undertake further investigation into the cause of the exceedance. This further investigation may include more extensive monitoring, and consideration of actions that may be appropriate to
- reduce exposure of the population to these levels of pollutants. This may involve a range of programs around motor vehicle usage and travel demand, a number of which are currently underway in a number of jurisdictions. This is not a direct impact of the proposed NEPM.

#### 40 9.4 PHOTOCHEMICAL SMOG IMPACTS

Benzene, toluene, xylenes and formaldehyde are part of a group of compounds known as volatile organic compounds (VOCs). In the presence of sunlight, VOCs and oxides of nitrogen react to form photochemical smog. These reactions require VOCs and oxides of nitrogen in sufficient concentrations, and exposure to sunlight for sufficient lengths of time,

- <sup>45</sup> before smog can form in significant quantity. Consequently, both smog formation potential and regional weather patterns play major roles in determining if, when and where elevated smog levels occur.
- Photochemical smog is usually monitored as ozone which is one of pollutants monitored under the Ambient Air Quality NEPM. Reductions in the gaseous air toxics under consideration in the proposed NEPM will help to reduce ozone levels and health impacts associated with exposure to ozone.

Individual VOCs vary greatly in their smog formation potential. The California Air Resources Board developed a scale called Maximum Incremental Reactivity (MIR) for ranking the smog formation potential of VOCs. Apace Research Ltd<sup>1</sup> reported MIRs for VOCs emitted by post-1986 Australian vehicles running on unleaded petrol ranging from 0.02 (for methane) to 10.1 (for 1,3,5-trimethylbenzene), with a weighted average of 3.4 for all VOCs. The following data were reported for air toxics:

Air Toxic	MIR	Total VOCs in exhaust emissions* (wt %)	Total VOCs in evaporative emissions* (wt %)
Benzene	0.42	4.9	1.1
Toluene	2.7	6.2	2.3
Xylenes	6.5 - 7.4	6.0	2.2
Formaldehyde	7.2	0.6	0

Table 9-2: VO	C emissions
---------------	-------------

Averaged across all test results (41 post-1986 Australian vehicles from 5 manufacturers, running on unleaded petrol)

15

Although not representative of the entire fleet, the above table indicates that toluene and xylenes have moderate to high MIRs, and make up a significant proportion of total VOC emissions from motor vehicles. Benzene and formaldehyde make smaller contributions. Taken as a group, these air toxics account for a significant proportion of the smog forming potential of VOC emissions from motor vehicles.

20

It is anticipated that actions to reduce emissions of benzene, toluene, xylenes and formaldehyde could flow from monitoring carried out under the proposed NEPM. Any such reductions in emissions will tend to reduce the potential to form photochemical smog.

#### 25

30

#### 9.5 INDUSTRY IMPACTS

The proposed NEPM does not apply as design ground level concentrations or emission limits for the control of industrial emissions. Industries large and small contribute to air pollution in general and are managed by States and Territories through a range of statutory and non-statutory measures including policies, licensing, regulations, and codes of practice. Existing programs aimed at managing general emissions from industry are likely to lead to reductions of emissions of air toxics.

For PAHs, the main source of industrial emissions is from combustion processes. These
include bitumen and asphalt production plants, petroleum refineries, power plants using
fossil fuels, coking ovens and aluminium refineries. Industrial emissions of benzene, toluene
and xylenes result from emissions associated with the use of solvents formulated with these
chemicals, as well as from combustion processes.

Industrial emissions of formaldehyde result from emissions associated with the manufacture of resins and textiles, disinfectants, fertilisers and the use of glues formulated with formaldehyde, as well as from combustion processes. The primary source of formaldehyde in ambient air in Australia is motor vehicles, through the incomplete combustion of fuel. NPI data indicate that the burning of solid fuel in homes can also be a significant source of benzene.

<sup>&</sup>lt;sup>1</sup> "Intensive Field Trial of Ethanol/Petrol Blend in Vehicles", Apace Research Ltd, ERDC Project No. 2511, December 1998, Volume 3, Appendix J

- Industry has improved its emissions performance over recent years as a result of increased 5 environmental awareness and interaction with environment protection agencies, which in their dealings with industry use a range of approaches. These include licensing of industrial premises, cleaner production partnerships, sustainable industry initiatives and, if required, enforcement activities such as pollution abatement notices and prosecutions. Whilst these
- programs are aimed at managing emissions from industry across the board, they clearly 10 have an impact on the air toxics considered under the proposed NEPM.

Emissions inventory information from the National Pollutant Inventory for the five most populous airsheds (Brisbane, Sydney, Melbourne, Adelaide and Perth) for the five air toxics dealt with under this NEPM is summarised in the table below:

15

Air Toxic	Industry emissions as a percentage of total emissions*	
	(range indicates variation between airsheds)	
PAHs	0.6 to 27	
Benzene	4 to 10	
Toluene	13 to 21	
Xylenes	13 to 27	
Formaldehyde	0.4 to 2.7	

\* Excluding bushfires, agricultural burning and hazard reduction burning

20

Unlicensed premises, including small to medium enterprises, may still produce significant emissions that may affect local air quality. This is particularly of concern when a number of these premises are clustered together near residential areas.

- The investigation levels for air toxics introduced through this NEPM will not apply to the 25 direct control of industrial emissions through the application as emission limits or design ground level concentrations. However, where the air toxics emissions from industry impact significantly on air quality where people live, jurisdictions may review licences and control practices to reduce ambient concentrations. Monitoring at Stage 2 sites will be conducted in areas where there are expected to be significantly elevated levels of air toxics and a 30 significant population exposed to those levels. This means that monitoring is unlikely to occur at the boundary of industrial premises. Where a site identified in the Stage 2 desk study is near an industrial premises already subject to jurisdictional regulatory control, the jurisdiction may decide that monitoring at such a site in accordance with the NEPM would not add value to existing actions. In such cases monitoring would not occur. This is entirely 35
  - at the discretion of individual jurisdictions.

It is considered that the introduction of the proposed Air Toxics NEPM will not have any direct costs to industry.

40

With the introduction of PM<sub>10</sub> and the proposed PM<sub>2.5</sub> standards in the Ambient Air Quality NEPM, jurisdictions have put in place, or are developing, a range of management strategies to control emissions of PM<sub>10</sub> and PM<sub>2.5</sub> from a range of sources including combustion processes at industrial sources. These strategies may also address emissions of air toxics from <sup>5</sup> these industrial processes, and it is unlikely that the introduction of this NEPM will significantly change these practices.

It may however lead to a greater focus on reducing emissions of air toxics through review of licences or processes that lead to emissions. The impact on individual companies is likely to be small as many of these strategies are already under consideration or in place.

#### 9.6 DOMESTIC SOLID FUEL BURNING IMPACTS

#### 9.6.1 Domestic wood heaters

10

25

A number of states and territories have recognised the use of domestic wood heaters can cause elevated levels of particles in urban, rural and regional areas, which can result in 'winter haze'. As domestic wood heaters are also a significant source of the air toxics addressed under the proposed NEPM, any existing programs aimed at managing emissions arising from their use will lead to reductions in emissions of air toxics.

- 20 A range of programs are currently being used by various jurisdictions to manage this issue, including:
  - Emission and efficiency standards for new wood heaters. Australian Standards have been developed for these parameters, and several jurisdictions have regulations that new wood heaters are to comply with the standard.
  - "Don't Light Tonight" campaigns. Meteorological modelling is used by some jurisdictions to predict nights on which dispersion is likely to be poor, thus resulting in prolonged exposure to wood smoke. When these conditions are forecast, the community is asked through media announcements to avoid lighting wood heaters.
- Wood heater replacement programs. Some jurisdictions offer subsidies to householders who permanently retire their wood heaters and replace them with cleaner burning appliances.
- Awareness and education campaigns. Some jurisdictions run public awareness campaigns to increase understanding of the community health problems associated with smoky wood heaters. These are generally supported by community education campaigns aimed at reducing wood heater emissions through better operation.
  - Firewood quality. A national program based on a voluntary code of practice is in place to improve the quality of firewood supplier by retail firewood merchants. This has the potential to reduce emissions as a result of burning unseasoned or contaminated firewood.
  - Enforcement measures. Some jurisdictions have developed, or are in the process of developing, regulations aimed at the prosecution of householders operating excessively smoky wood heaters. These measures, in combination with targeted community education, have the potential to significantly reduce wood smoke emissions with the threat of punitive action against offenders.
  - Whilst these programs are primarily aimed at managing winter haze and smoke issues associated with domestic wood heaters, they clearly have an impact on the air toxics considered under the proposed NEPM.
- 50

40

45

Smoke from solid fuel heating contains a range of pollutants. These include pollutants covered by the Ambient Air Quality NEPM (eg particulate matter, carbon monoxide and

Page 65

- nitrogen dioxide), as well as a broad range of air toxics. Air toxics found in woodsmoke 5 include benzene, toluene and xylenes, formaldehyde and PAHs. (Environment Australia 2002). Current measures being implemented to manage particle emissions from solid fuel heating may address air toxics under consideration.
- Air toxics emissions, as a result of woodsmoke, are subject to significant seasonal variation 10 associated with the greatly increased use of domestic solid fuel heating in the cooler months. NPI data (2000/2001) indicates solid fuel burning contributes approximately 53% of PAHs and formaldehyde<sup>2</sup> and 11% emissions of benzene in Australia. These contributions are significantly higher in some regional air sheds, where woodheaters are the major source of One such example is Launceston, where solid fuel heating accounts emissions. 15
- approximately for around 46% of emissions of benzene, 87% of formaldehyde, 91% of PAHs, 15% of toluene and 9% of xylenes emissions, annually.
- As a result of the significant contribution of woodheaters to the ambient load of the air toxics under consideration (and other pollutants), jurisdictions will likely consider how best to 20 augment the range of measures already being employed to reduce this load, particularly in airsheds where this is of concern. Any additional measures adopted may impact on woodheater manufacturers, fuel wood suppliers, enforcement agencies and users. It should be noted that similar actions might be required as a result through a variation to the Ambient
- Air Quality NEPM, the introduction of a standard for PM<sub>2.5</sub>. 25

#### 9.6.2 Woodheater Manufacturers

Woodheater manufacturers have been working towards reducing particle emissions for around two decades. Technological advances aimed at addressing particle emissions are likely to be effective in reducing emissions of air toxics, also (Environment Australia, 2002a). 30 Catalytic devices and 'high-tech' combustion chamber design are two examples of technologies aiming at reducing such emissions, with the latter favoured by most Other approaches, that have also proved successful, are high mass commentators. appliances where combustion takes place at high burn rates and heat is stored in appliance for gradual release into the living space and wood-pellet heaters where fuel is fed into the 35 combustion chamber continuously. The latter technology has only recently become available in Australia and has the potential to significantly reduce the influence of operator behaviour on wood heater emissions (Commonwealth 2002b).

- The Australian Standard for particle emissions from woodheaters (AS 4013) has been 40 adopted in legislation in most jurisdictions. This standard sets an emissions limit for particles from woodheaters of 4.0 gram particles per kilogram of wood burnt. Although the standard does not set emission standards for any of the air toxics under consideration, it is anticipated that a reduction in emissions of particulate matter will be accompanied by a reduction in emissions of these air toxics. 45

50

However, there is evidence to suggest that technological advances designed to reduce emissions from woodheaters within a test environment may not be as effective in the 'real world'. The Commonwealth State of the Environment Report (SOE) (2001) states that if improvement in winter air quality is to rely solely on the uptake of new woodheaters meeting tighter emission standards, it will probably take a long time for acceptable air

<sup>&</sup>lt;sup>2</sup> 2000/2001 NPI data (Note: this information may be incomplete as reporting on formaldehyde emissions is not required under the NPI until 2001/2002 reporting year)

<sup>5</sup> quality, in compliance with the Ambient Air Quality NEPM, to be achieved. The same situation is likely to apply to the air toxics under consideration.

Notwithstanding the influence of user behaviour on woodheater emissions, the adoption of standards for the air toxics under consideration may lead to an accelerated program to further reduce the particle emissions limit of new woodheaters, with product development costs to be borne by woodheater manufacturers.

Emissions of the air toxics under consideration from open fireplaces are similar in nature to those from other woodheaters and the discussion presented above is also applicable to open fireplaces. However, the extent to which emissions can be controlled through technology in the case of open fireplaces is limited, as there are currently no design criteria, construction standards or emissions standards applicable to these appliances.

#### 9.6.3 Firewood Suppliers

10

15

30

The combustion of firewood containing excessive moisture, in domestic solid fuel heaters, can lead to increased pollutant emissions, including the air toxics under consideration. To limit such emissions, some jurisdictions have introduced legislation aimed at regulating firewood quality. For example, WA and the ACT have regulations limiting the moisture content of firewood sold by merchants to 20%. However, such regulations do not address firewood quality when firewood is collected privately by users.

Should the results of monitoring undertaken for the proposed NEPM indicate levels significantly above the investigation levels, jurisdictions in which no regulations currently exist may initiate an accelerated program to regulate the firewood supply industry. This would lead to costs for government authorities, associated with regulation development and enforcement. It should be noted that the issue of firewood quality is addressed by the National Firewood Strategy (coordinated by the National Resource Management Ministerial

Council) possibly leading to complementary efforts in this area.

#### 35 9.6.4 Woodheater Users

The Commonwealth and a number of jurisdictions have implemented a range of community education programs to address woodheater emissions. These programs include:

*Improving operating behaviour*: Operator behaviour is the single most important factor
 influencing pollutant emissions from woodheaters. Even where properly seasoned firewood and best available technology is used, poor operating conditions can result in excessive emissions. For example, excessive firewood loading and restricting airflow in a woodheater will result in higher emissions of pollutants, including the air toxics under consideration. Encouraging the correct operation of woodheaters is therefore an effective strategy to reduce
 woodheater emissions. Evidence indicates that woodheater users are responsive to targeted

education and compliance activities.

Community awareness/education campaigns are based on a number of approaches. For example, the Commonwealth's *Breathe the Benefits* campaign included television advertisements, brochures and a website promoting the correct operation of woodheaters. In Victoria, volunteers in the *Community Access to Air Monitoring* program raise awareness in their community on the impacts of wood heating.

*Reducing the use of woodheaters.* If monitoring shows widespread and/or significant local exceedances of the investigation levels, jurisdictions may consider strategies to reduce the use of woodheaters in some areas. Strategies include community requests to restrict or reduce woodheater use when high air pollution is forecasted (eg the NSW EPA's 'Don't Light Tonight' campaign), programs designed to replace older, more polluting woodheaters, and more drastic measures such as the prohibition of woodheaters.

The extreme measure of prohibiting woodheater use, whether through the banning of the installation of new woodheaters or the prohibition of woodheater use, would have impacts on the community, particularly in the case of lower income groups located in regions where alternative forms of heating are more expensive. Other stakeholders likely to be impacted are woodheater manufacturers and retailers.

Restrictions on woodheater use can be implemented by various government agencies. This would have to be weighed up in the consideration of any action to address woodheater emissions in each jurisdiction. A small number of local councils in NSW have implemented a ban on the installation of new woodheaters.

The proposed NEPM will provide information that may lead to community acceptance of health problems resulting from woodheater use. This acceptance may assist in the implementation of measures to reduce woodheater emissions.

#### 9.6.5 Summary

15

20

40

50

There are several programs currently in place to address emissions from domestic solid fuel heating. It is most unlikely that the introduction of the monitoring and reporting requirements under the Air Toxics NEPM will induce further actions than those currently in practice or planned. However, widespread occurrence of NEPM monitoring results above the investigation levels in the proposed NEPM may be a driver for accelerated action and may assist in the targeting of programs.

### 35 9.7 PRESCRIBED BURNING IMPACTS

Prescribed burning is a management tool used by fire authorities and land managers to reduce the likelihood and impact of bushfires. The strategic use of prescribed burning for fuel reduction assists in the protection of human life, community assets, private property and habitats, and promotes biological diversity. Fire management practices in Australia have been developed in the context that fire is a natural and vital part of the landscape, and is required for the long-term survival of our unique flora and fauna.

- Prescribed burning is a term used to identify planned burning involving the controlled application of fire under specified environmental conditions to a predetermined area and at the time, intensity and rate of spread required to attain planned resource management
- 45 objectives. It covers a range of fire applications on both public and private land including:
  - Fuel reduction burning to prevent wildfires;
  - Burning to maintain ecosystem health;
  - Burning to remove crop wastes;
  - Burning to remove vegetation or other materials including pest plants and pest animal habitat; and
    - Burning to prepare seed beds for flora regeneration following timber harvesting.

- <sup>5</sup> Wildfire prevention through fuel reduction burning is a fundamental component of comprehensive fire prevention programs. These programs broadly aim to:
  - Reduce the number of unplanned fires;
  - Assist the rapid control of fires that do start by creating fuel reduced zones and increasing safety for fire fighters; and
- Assist the community to survive those fires that impact on them.

Bushfires, in addition to their potential immediate threat to life and property, can generate substantial air toxics emissions, with potential for exposure of communities. Prescribed burning provides a means, sometimes the only means available to modify the type and reduce the amount of fuel available for bushfires, particularly in forested areas or other areas where access is constrained. The resultant moderation of fire behaviour provides for safer and earlier control of bushfires, and therefore has the potential to reduce smoke emissions and community exposure, as well as reducing the direct threat to life and property. Air toxics emissions from strategically planned prescribed burning programs can, at times, be of significance. However they generally comprise only a small fraction of those produced during a single significant bushfire event.

In Australia, specific fire management laws have been developed in all jurisdictions. These laws place a clear statutory obligation on park and forest agencies, landowners and municipalities to institute effective fire protection measures. These laws include regulatory frameworks for the application of fire, recognising that strategically located burning is an essential component of park, forest and other land management. Prescribed fire is a key tool for these land managers to meet their community safety obligations.

- <sup>30</sup> While prescribed burning is important, the impacts on air quality can be significant in the areas surrounding the burns and, occasionally, on a wider regional scale. Concerns over health and visibility impacts associated with burning of large areas for fire risk management have led to pressure on fire management authorities to include smoke management considerations in planning and implementing prescribed burning. The challenge is to ensure
- <sup>35</sup> adequate levels of prescribed burning, under controlled conditions, to reduce the impacts of major bushfires on life and property, while minimising the exposure of communities to smoke impacts from planned burning activities. The aim must continue to be to find the balance between the risks of smoke impacts from prescribed burning on community health and the risk of major bushfires that threaten life and property.
- 40

15

20

Fire management authorities across Australia have increased their focus on the management of smoke impacts. Research into meteorological factors that influence the transport and dispersion of smoke plumes has led to advanced weather forecasting systems that are used for scheduling burns to avoid or reduce smoke impacts. In Western Australia, for example,

- <sup>45</sup> analysis of monitoring data provided by the Department of Conservation and Land Management indicates that haze events in Perth generally do not occur during the prescribed burning period (R Sneeuwjagt, WA CALM, 2002). In Victoria, smoke modelling research with the Bureau of Meteorology has enabled the Department of Sustainability and Environment to predict smoke plume dispersion from proposed burning operations to aid
- <sup>50</sup> decision making on burning operations near populated airsheds. Fire management and environmental protection authorities are now working together in most jurisdictions to address air quality issues associated with prescribed burning operations.

<sup>5</sup> The introduction of the PM<sub>10</sub> standards in the Ambient Air Quality NEPM has in part contributed to fire authorities shifting focus to the management of smoke impacts. The monitoring and reporting of air toxics required under the draft Air Toxics NEPM are unlikely to have any significant impact on fire management authorities as the current and proposed strategies to address PM<sub>10</sub> and PM<sub>2.5</sub> will also address air toxics.

10

# 9.8 AGRICULTURAL BURNING IMPACTS

Agricultural burning refers to the burning of crop stubble to prepare the land for re-sowing, or burning to remove pest plants or dead vegetation. Burning during fire restriction periods requires a permit, generally provided by local council officers. Burning is otherwise unregulated during non-permit periods except where local laws or other legislation come into effect.

15

20

Impacts of air toxics emissions from agricultural burning would generally be localised, however, a number of burns taking place at the same time within a region may have more widespread impacts. Also burning in unsuitable conditions (eg wet stubble) increases emissions of smoke.

During permit periods, landowners have a legal obligation to advise the local fire brigade or state fire authorities of their intention to burn and assess weather conditions forecast for the burn time. Other permit conditions include the estimation of certain fire parameters (eg rate of spread) to ensure the permit holder provides sufficient resources to control the fire and prevent it from spreading beyond designated control lines. Permits may in some circumstances also require an obligation to minimise impacts such that smoke does not enter populated areas for some time after the burn, based on weather forecasts.

30

Some local councils and state agricultural organisations have been proactive in reducing the potential for smoke impacts from agricultural burning. Activities include the promotion of alternative farming methods (eg minimum or zero tillage) and providing advice on optimum burn conditions.

35

40

It is considered that existing management systems for agricultural burning, and the commitment to further improve these systems are an adequate basis for the management of air toxics from this source. Accordingly, it is unlikely that the introduction of the proposed Air Toxics NEPM will significantly impact landowners or regulatory authorities overseeing burning operations.

# 9.9 CULTURAL ISSUES

Traditional cultural burning practices are carried out across extensive areas of northern Australia and are usually part of land management regimes designed to provide environmental resources for communities. However, little research has been undertaken to document the characteristics of cultural burning practices in different regions.

Burning of vegetation through cultural burns may be an important source of air toxics in rural areas and on the urban fringe. Emissions from these sources can be transported over
significant distances, impacting on both rural and urban areas. However, it is likely that emissions from cultural burns mostly take place in relatively remote areas and are therefore unlikely to significantly impact on populated areas.

- It is acknowledged that any management strategies that affect these practices may have 5 impacts on the communities by reducing the availability of particular resources, such as bush foods. However, it is considered that existing management systems for cultural burning are an adequate basis for the management of air toxics from this source. Accordingly, it is unlikely that the proposed Air Toxics NEPM would have a significant impact on cultural burning practices.
- 10

15

#### 9.10 WASTE BURNING AND LAND DEVELOPMENT IMPACTS

# 9.10.1 Background

Waste burning can take place in a variety of situations and locations, including:

- back-yard burning (open burning and incineration) of household rubbish and garden waste:
  - on-site burning (open burning and incineration) of commercial and industrial waste;
  - on-site incineration of clinical or related waste and quarantine waste;
  - incineration of waste at purpose built facilities; and •
- burning at landfills. 20 •

Back-yard burning is increasingly being viewed as unacceptable, and many jurisdictions have banned or severely restricted its use. Impacts of air toxics emissions from such small scale waste burning may be significant on a local scale (ie nuisance impacts on adjacent

- residents) but unless used widely would not result in widespread degradation of air quality. 25 Burning activities are often strictly controlled through regulation, local laws or licence conditions. Many local governments ban the use of backyard incinerators and the burning of waste.
- The predominant method of burning waste is through purpose-made incinerators. In most 30 jurisdictions, this activity must be licensed and air emissions are strictly controlled. Waste acceptance criteria are also established in licence conditions. Under most legislation or licence conditions, burning of waste at a landfill is a prohibited activity. Waste may also be burnt in co-generation plants, cement kilns and sugar mills as part of a beneficial use to
- recover energy or to form part of the manufacturing process. 35

Potential air toxics impacts from land development are associated with burning of cleared vegetation, which can take place in open piles or windrows, or in pit burners. Impacts are generally likely be confined to the local scale, however, development of large sites may result

- in broader impacts. Guidelines for managing impacts of land development operations are 40 established in most jurisdictions and appear to adequately address management of air toxics emissions. There may also be specific regulations in jurisdictions that restrict the use of burning in vegetation clearance. Many local governments also prohibit the open burning of vegetation that has been cleared for development, with some also banning the use of pit burners.
- 45

The States and Territories generally recognise burning as one of the least sustainable and least preferred methods for managing waste (particularly when waste heat is not recovered and utilised). The waste management hierarchy (which has been adopted in all States and

Territories) places disposal as the least preferred management option. This includes the 50 burning of waste without energy recovery. Details can be found in the following documents:

- 5 New South Wales
  - Clean Air (Plant and Equipment) Regulation 1997
  - Waste Avoidance and Resource Recovery Act 2001
  - State of Environment Report 1995, 1997

# 10 Victoria

- State Environment Protection Policy (Air Quality Management) 2001
- Industrial Waste Strategy 1998

#### Queensland

- 15 Waste Management Strategy for Queensland 1996
  - Environment Protection (Interim Waste) Regulation 1996
  - State Interest Planning Policy For Waste Management And Contaminated Land In Planning Schemes 2000
  - Environment Protection (Waste Management) Policy 2000
- 20 Environment Protection (Waste Management) Regulation 2000

#### Western Australia

- Management of Air Emissions from Biomedical Waste Incinerators 2000 Guidance for the Assessment of Environmental Factors
- 25 Towards Zero Waste 2001

#### South Australia

- Waste Management in South Australia: Discussion Paper leading to the development of an EP (Waste) Policy 2000
- 30

# Tasmania

- Draft Environmental Protection for Air Quality
- Environmental Management and Pollution Control Act 1994
- Environmental Management and Pollution Control (Waste Management) Regulations 2000

#### 35

40

# Australian Capital Territory

• No Waste by 2010: A Waste Management Strategy for Canberra

# Northern Territory

- Waste Management and Pollution Control Act 1998
  - Waste Management and Pollution Control (Administration) Regulations 2001

# 9.10.2 Impact of the proposed NEPM

45

Commercial and industrial incineration and incineration of clinical or related waste and other regulated or controlled wastes (such as solvents, quarantine waste, illicit substances) are generally controlled by State/Territory based licensing or approval systems. Licences/approvals are issued subject to conditions aimed at minimising emissions and 5 maximising combustion efficiency. Emissions of air toxics from these sources are reasonably well controlled.

Smoke from back yard burning, open burning of commercial and industrial waste, burning at landfills and burning associated with land development is widely recognised as an urban nuisance/public health issue. Management by local governments through local laws, town planning and development approval processes generally prevents or minimises impacts on urban areas. Monitoring of air toxics under the proposed NEPM is expected to put pressure on jurisdictions to reduce emissions if elevated levels are found in urban areas. However, given the pressure and control regimes that already exist for managing waste burning and land development in and around urban areas, it is considered unlikely that these activities

15 land development in and around urban areas, it is considered unlikely that these will be significantly influenced by the proposed NEPM.

# 9.11 TRANSPORT AND LAND USE PLANNING IMPACTS

30

Transport and land use planning processes are used by jurisdictions to manage the demand for transport by redesigning the urban form and transport infrastructure of our cities. The objective is to both minimise the need for transport services, and facilitate the most efficient forms of transport. The expected benefits would include reduced traffic congestion, lower emissions and noise levels, and deferral of the need to increase capacity of transport infrastructure. These processes are by necessity long term in nature because of the high cost and long life spans of buildings and transport infrastructure.

Jurisdictions already have programs in place to address compliance with the ambient air quality NEPM, traffic congestion and urban amenity issues. As the proposed NEPM is focused on the collection of data, it is unlikely that it will create direct pressure to change existing transport and land use planning activities. However, if investigation levels are exceeded in areas impacted by traffic emissions, this may provide incentive for jurisdictions to explore additional long term planning solutions.

#### 5 **REFERENCE LIST**

Abbate C, Giorgianni C, Munao F, et al. (1993). *Neurotoxicity induced by exposure to toluene. An electrophysiologic study.* Int Arch Occup Environ Health 64:389-392.

- <sup>10</sup> Agar JP, Gooding DA, Hartley MR, Hope LAE, Mitchell RM, Powell CB, (2000), *Air Quality Monitoring Report, Air Quality Monitoring at Richmond Primary School, Keswick*, Environment Protection Authority, Department for Environment & Heritage, SA.
- Agar JP, Gooding DA, Hartley MR, Hope LAE, Mitchell RM, Powell CB, (2001a), *Air Quality Monitoring at Hensley Foundry, Flinders Park*, Environment Protection Authority, Department for Environment & Heritage, SA.

Aksoy M (1989) *Hematotoxicity and carcinogenicity of benzene*. Environmental Health Perspectives, 82:193-197.

20

Aksoy M, Dincol K, Erdem S, Akgun T, and Dincol G. 1972. *Details of blood changes in 32 patients with pancytopenia associated with long-term exposure to benzene*. (Br. J. Ind. Med. 29:56-64).

<sup>25</sup> Albert RE, Sellakumar AR, Laskin S, et al. (1982). *Gaseous formaldehyde and hydrogen chloride induction of nasal cancer in the rat.* J Natl Cancer Inst 68:597-603.

Andersen I, Lundqvist GR, Molhave L, et al. (1983). *Human response to controlled levels of toluene in six-hour exposures.* Scand J Work Environ Health 9:405-418.

30

Andersen I, Molhave L. (1983). *Controlled human studies with formaldehyde*. In Gibson JE, (ed.) *Formaldehyde toxicity*. Washington, DC: Hemisphere Publishing Corporation, 154-165.

Aoyama K. (1986) *Effects of benzene inhalation on lymphocyte subpopulations and immune response in mice.* (Toxicol. Appl. Pharmacol. 85:92-101).

Armstrong B, Tremblay C, Baris D, Theriault G. (1994) *Lung cancer mortality and polynuclear aromatic hydrocarbons: a case-cohort study of aluminium production workers in Arvida, Quebec, Canada.* American Journal of Epidemiology, 139: 250-262.

40

ATSDR (Agency for Toxic Substances and Disease Registry) (1990a), *Toxicological Profile for Benzo(a)pyrene*, ATSDR/TP-88-05.

ATSDR (Agency for Toxic Substances and Disease Registry) (1990b), *Toxicological Profile for Polycyclic Aromatic Hydrocarbons*, ATSDR/TP-90-20

ATSDR (Agency for Toxic Substances and Disease Registry), (1995) *Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs) (Update)* 

50 ATSDR.. (Agency for Toxic Substances and Disease Registry) (1995). *Toxicological Profile for Xylenes (Update).* 

ATSDR (Agency for Toxic Substances and Disease Registry) (1999) *Toxicological Profile for Formaldehyde.* 

ATSDR (Agency for Toxic Substances and Disease Registry) (2000). *Toxicological Profile for Toluene* 

ATSDR Agency for Toxic Substances and Disease Registry) (2000). *Toxicological Profile for Xylenes* 

Baelum J, Andersen I, Lundqvist GR, et al. (1985). *Response of solvent-exposed printers and unexposed controls to six-hour toluene exposure*. Scand J Work Environ Health 11:271-280.

<sup>15</sup> Baelum J, Lundqvist L, Molhave L, Andersen NT. (1990). Human response to varying concentrations of toluene. Int Arch Occup Environ Health 62(1): 65-71.

Bardsley TB (1991). *Ambient Monitoring-58 Belmar Avenue, Altona,* Memorandum to Bruce Dawson (EPA Victoria), 12 MARCH, Ref No. Altona TB/GC/8

20

25

5

Bardsley TB (1996). *Evaluation of an OPSIS differential optical absorption spectrometer for ambient air monitoring*, Clean Air 30(2: 31)

Bardsley TB (1997). *Report on Huntsman Chemicals 1997*, Report prepared for EPA Victoria, West Metro Operations Unit

Bender JR, Mullin LS, Graepel GJ, et al. (1983). *Eye irritation response of humans to formaldehyde*. Am Ind Hyg Assoc J 44:463-465.

<sup>30</sup> Blair A, Stewart PA, Hoover RN. 1990. *Mortality from lung cancer among workers employed in formaldehyde industries.* Am J Ind Med 17:683-700.

Boey KW, Foo SC, Jeyaratnam J. (1997). *Effects of occupational exposure to toluene: a neuropsychological study on workers in Singapore.* Ann Acad Med Singapore 26(2):84-7.

35

Bond GG, McLaren EA, Baldwin CL, Cook RR (1986) *An update of mortality among chemical workers exposed to benzene*. British Journal of Industrial Medicine, 43:685-691 [erratum in 44:215].

40 Carpenter CP, Kinkead ER, Geary DL Jr, Sullivan LJ, & King JM (1975) *Petroleum hydrocarbon toxicity studies. V. Animal and human response to vapors of mixed xylenes.* Toxicol Appl Pharmacol, 33: 543-558.

Chang JCF, Gross EA, Swenberg JA, et al. (1983). Nasal cavity deposition, histopathology, and cell
 proliferation after single or repeated formaldehyde exposure in B6C3F1 mice and F-344 rats. Toxicol
 Appl Pharmacol 68:161-176.

CICAD (Concise International Chemical Assessment Document) (2002) *No. 40 Formaldehyde* , World Health Organization, Geneva

50

CIIT. (1998). Chemical Industry Institute of Toxicology. *Formaldehyde risk assessment meeting.* November 14, 1997. Research Triangle Park, NC.

Collins JJ, Acquavella JF, Esmen NA. (1997). An updated meta-analysis of formaldehyde exposure
 and upper respiratory tract cancers. J Occup Environ Med 39:639-651.

Collins JJ, Ireland BK, Easterday PA, Nair RS, Braun J. (1997). *Evaluation of lymphopenia among workers with low-level benzene exposure and the utility of routine data collection*. (J.Occup. Environ. Med. 39(3):232-237).

10 Commission of European Communities (1998), Council Directive on Ambient Air Quality: Assessment and Management Working Group on Benzene, Position paper.

Commission of European Communities (2001), European Commission. Polycyclic Aromatic Hydrocarbons (PAH) Position Paper (July 2001), Prepared by the Working Group On Polycyclic Aromatic Hydrocarbons

Commonwealth State of the Environment Report (SOE), (2001) Environment Australia, State of the Environment 2001 (Australia), Commonwealth of Australia.

20 Costantino JP, Redmond CK, Bearden (1995). *Occupationally related cancer risk among coke oven workers: 30 years of follow-up.* Journal of Occupational and Environmental Medicine; 37: 597-604.

Courtney KD, Andrews JE, Springer J, Menache M, Williams T, Dalley L, et al. (1986). *A perinatal study of toluene in CD-1 mice*. Fundam Appl Toxicol;6:145-154.

Crump KS (1994) *Risk of benzene-induced leukaemia: A sensitivity analysis of the Pliofilm cohort with additional follow up and new exposure estimates.* J Toxicol Environ Hjealth. 42:219-242.

- <sup>30</sup> Crump K, and Allen B. (1984). *Quantitative estimates of risk of leukemia from occupational exposure to benzene.* Occupational Safety and Health Administration; Docket H-059B. [As cited in: Cody RR, Strawderman WW, and Kipen HM. 1993. *Hematologic effects of benzene.* J.Occup. Med. 35(8):776-782.]
- <sup>35</sup> Deutsch-Wenzel RP, Brune H, Grimmer O, Dettbarn G and Misfeld J. (1983). *Experimental studies in rat lungs on the carcinogenicity and dose-response relationships of eight frequently occurring environmental polycyclic aromatic hydrocarbons.* JNCI 71:539-544.

Dudek B, Gralewicz K, Jakubowski M, Kostrzewski P, & Sokal J (1990) *Neurobehavioral effects of experimental exposure to toluene, xylene and their mixture.* Pol J Occup Med, 3: 109-116.

Eastmond DA, Smith MT, Irons RD (1987) *An interaction of benzene metabolites reproduces the myelotoxicity observed with benzene exposure*. Toxicology and Applied Pharmacology, 91:85-95.

45 ECETOC. 1995. Technical Report No. 65. *Formaldehyde and human cancer risk*. Brussels, Belgium:ECETOC.

Echeverria D, Fine L, Langolf G, et al. (1991). *Acute behavioural comparisons of toluene and ethanol in human subjects.* Br J Ind Med 48:750-761.

50

5

15

Environment ACT (2001). Personal communication to NEPCSC

Environment Australia, (2002a), *Technical Report No 5: Emissions from Domestic Solid Fuel Burning Appliances, March 2002* Commonwealth of Australia

5 Environment Canada, (1988). *Conservation and Protection, Pacific and Yukon Region, Vancouver, B.C.*, 48

Environment Canada, Health and Welfare Canada, (1992): *Canadian Environmental Protection Act. Priority Substances List assessment report— Report No. 4.* Ottawa, Ontario, Minister of Public Works and Government Services.

Environment Canada, Health and Welfare Canada (1993) *Canadian Environmental Protection Act. Priority Substances List assessment report— Benzene.* Ottawa, Ontario, Minister of Public Works and Government Services.

15

10

Environment Canada, Health and Welfare Canada, (1994): *Canadian Environmental Protection Act. Priority Substances List assessment report*—, Polycyclic *Aromatic Hydrocarbons (PAHs).* Ottawa, Ontario, Minister of Public Works and Government Services.

20 Environment Canada, Health and Welfare Canada (1999) *Canadian Environmental Protection Act* — *Priority Substances List* — *Supporting document for the environmental assessment of formaldehyde.* Hull, Quebec, Environment Canada

Environment Canada, Health Canada (2001) Canadian Environmental Protection Act. Priority
 Substances List assessment report— Formaldehyde. Ottawa, Ontario, Minister of Public Works and Government Services.

EPA Victoria (1998). Freeway air Quality Study 1996 Westgate Freeway Altona (Draft), Melbourne EPA(Victoria)

30

European Commission.(EC) (2001) Polycyclic Aromatic Hydrocarbons (PAH) Position Paper (July 2001), Prepared by the Working Group On Polycyclic Aromatic Hydrocarbons

Expert Working Party, (1996). *Report on an investigation by an expert working party into air pollution, environmental health and respiratory diseases: Launceston and upper Tamar valley Tasmania 1991-1994*.the Launceston City Council

Foo SC, Jeyaratnam J, Koh D. (1990). *Chronic neurobehavioural effects of toluene*. Br J Ind Med 47:480-484.

40

Foo,S.C., Ngim, C.H, Salleh I, Jeyaratnam J, Boey KW. (1993). *Neurobehavioural effects in occupational chemical exposure.* Environmental research, 60:267–273

Forkman, B.A, BA. Ljungberg T, Johnson AC, Nylen P, Stahle L, Hoglund G, Ungerstedt, U.
(1991). *Long-term effects of toluene inhalation on rat behaviour.* Neurotoxicology & Teratology. 13(5):475-81

Fox I (1999). *Particulate air pollution in the Canberra airshed (including PAHs).* Unpublished Master Degree's Thesis, Canberra University

50

55

Gamberale F, Annwall G, Hultengren M. (1978). *Exposure to xylene and ethylbenzene: III. Effects on central nervous functions.* Stand J Work Environ Health 4:204-211.

Gras JL, (1996). *The Perth haze study: A report to the Department of Environmental Protection of Western Australia on fine particle haze in Perth*. CSIRO Division of Atmospheric Research

Gras JL et al, (1992), *CSIRO-EPA Victoria, Melbourne aerosol study, Final report,* Aspendale Victoria, Australia, CSIRO Division of Atmospheric Research: 194

Gupta P, Banerjee DK, Bhargava SK, et al. (1993). Prevalence of impaired lung function in
 rubbermanufacturing factory workers exposed to benzo(a)pyrene and respirable particulate matter.
 IndoorEnviron 2:26-31.

Hake CLR, Stewart RD, Wu A, et al. (1981). *p-Xylene: Development of a biological standard for the industrial worker.* Report to the National Institute for Occupational Safety and Health, Cincinnati, OH, by the Medical College of Wisconsin, Inc., Milwaukee, WI. PB82-152844.

Halmes NC, Roberts SM, Tolson JK, Portier CJ. (2000) Re-evaluating cancer risk estimates for short-term exposure scenarios. Toxicological Sciences, 58 (1):32-42

20 Hass U, Lund SP, Simonsen L, & Schaich Fries A (1995) *Effects of prenatal exposure to xylene on postnatal development and behavior in rats.* Neurotoxicol Teratol, 17: 341-349.

Hass U, Lund SP, Simonsen L. (1997). *Long-lasting neurobehavioral effects of prenatal exposure to xylene in rats*. Neurotoxicology;18(2):547-551.

25

15

5

Hastings L, Cooper GP, & Burg W (1984) *Human sensory response to selected petroleum hydrocarbons.* Adv Med Environ Toxicol, 6: 255-270.

Hayes RB, Blair A, Stewart PA, et al. (1990). *Mortality of U.S. embalmers and funeral directors*. Am J Ind Med 18:641-652.

Hayes RB, Yin S-N, Dosemeci M, Li G-L, Wacholder S, Travis LB, Li C-Y, Rothman N, Hoover RN,Linet MS (1997) *Benzene and the dose-related incidence of hematologic neoplasms in China.* Journal of the National Cancer Institute, 89:1065-1071.

35

30

Heinrich, U., F. Pott, U. Mohr, R. Fuhst, and J. Konig, (1986), *Lung Tumours in Rats and Mice After Inhalation of PAH-rich Emissions.* Exp. Pathol, 29:29-34

Hudak A & Ungvary G (1978) *Embryotoxic effects of benzene and its methyl derivatives: Toluene, xylene.* Toxicology, 11: 55-63.

Hillefors-Berglund M, Liu Y, von Euler G. (1995). *Persistent, specific and dose-dependent effects of toluene exposure on dopamine D2 agonist binding in the rat caudate-putamen.* Toxicology 100:185-94.

45

Huntingdon Research Centre. (1992). *Toluene: Effect on pregnancy of the rat by inhalation (status report).* Huntingdon Research Centre, Ltd., Huntingdon, Cambridgeshire, England. APT 2/91279.

<sup>50</sup> IARC (International Agency for Research on Cancer). (1987). *IARC monographs on the evaluation of carcinogenic risk of chemicals to humans. Supp. 7: Overall evaluations of carcinogenicity: An updating of volumes 1 to 42.* World Health Organization, Lyon, France.

- <sup>5</sup> IARC (International Agency for Research on Cancer). (1995). *IARC monographs on the evaluation of carcinogenic risk of chemicals to humans. Vol. 62: Wood dusts and formaldehyde.* World Health Organization, Lyon, France.
- (IARC) International Agency for Research on Cancer (1983). *Benzo(a)]pyrene*. In: Polynuclear
   Aromatic Compounds, Part 1, Chemical, Environmental and Experimental Data. Vol. 32.
   Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. pp. 211-224.
- IARC (International Agency for Research on Cancer). (1982). IARC Monographs on the
   Evaluation of the Carcinogenic Risk of Chemicals to Humans: Some Industrial Chemicals and
   Dyestuffs. Volume 29. pp. 95-148 World Health Organization, Lyon, France.

Ikeda M, Koizumi A, Kasahara M, et al. (1986). *Combined effects of n-hexane and toluene on norepinephrine and dopamine levels in rat brain tissues after long-term exposure.* Bull Environ Contam Toxicol 36:510-517.

Infante PF, Rinsky RA, Wagoner JK, Young RJ. (1977). Leukemia in benzene workers. Lancet, 2:76-78.

<sup>25</sup> International Programme on Chemical Safety (IPCS),(1993). *Toluene. Environmental Health Criteria document No. 52.* World Health Organization, Geneva.

International Programme on Chemical Safety (IPCS),(1989). *Formaldehyde. Environmental Health Criteria document No 89.* World Health Organization, Geneva.

30

20

International Programme on Chemical Safety (IPCS), (1993). *Benzene. Environmental Health Criteria document No. 150.* World Health Organization, Geneva.

International Programme on Chemical Safety (IPCS), (1997). *Xylenes. Environmental Health Criteria Document 190* World Health Organization, Geneva.

International Programme on Chemical Safety (IPCS), (1998). *Selected Non-Heterocyclic Polycyclic Aromatic Hydrocarbons. Environmental Health Criteria document No. 202* World Health Organization, Geneva.

40

IRDC (International Research and Development Corporation), (1985). *Two-generation inhalation reproduction/fertility study on petroleum derived hydrocarbon with toluene*. API Medical Research Publication no. 32-32854. Washington (DC): American Petroleum Institute.

<sup>45</sup> Jenkins LJ, Jones RA, and Siegel J. (1970). *Long-term inhalation screening studies of benzene, toluene, o-xylene ,and cumene on experimental animals.* Toxicol. Appl. Pharmacol. 16:818-823.

Kamata E, Nakadate M, Uchida O, et al. (1997). *Results of a 28-month chronic inhalation toxicity study of formaldehyde in male Fischer-344 rats.* J Toxicol Sci 22:239-254.

50

Keller KA, and Snyder CA. (1978). *Mice exposed in utero to 20 ppm benzene exhibit altered numbers of recognizable hematopoietic cells up to seven weeks after exposure*. Fundam. Appl. Toxicol. 10:224-232.

5 Kerns WD, Pavkov KL, Donofrio DJ, et al. (1983). *Carcinogenicity of formaldehyde in rats and mice after long-term inhalation exposure*. Cancer Res 43:4382-4391.

Korsak Z, Sokal JA, Dedyk A, et al. (1988). *Toxic effects of combined exposure to toluene and xylene in animals: I. Acute inhalation study.* Pol J Occup Med 1:45-50.

Korsak Z, Swiercz R, & Jedrychowski R (1993) *Effects of acute combined exposure to* n-*butyl alcohol and* m-*xylene.* Pol J Occup Med Environ Health, 6: 35-41.

Kulle TJ. (1993). Acute odor and irritation response in health non-smokers with formaldehyde exposure. Inhal Toxicol 5:323-332.

Kulle TJ, Sauder LR, Hebel JR, et al. (1987). *Formaldehyde dose-response in healthy non-smokers.* J Air Pollut Control Assoc 37:919-924.

20 Kuna RA, and Kapp RW. (1981). *The embryotoxic/teratogenic potential of benzene vapor in rats.* Toxicol. Appl. Pharmacol. 57:1-7.

Laskin, S., M. Kuschner, and R.T. Drew (1970). *Studies in Pulmonary Carcinogenesis*, in: *Inhalation Carcinogenesis*, M.G. Hanna, Jr., P. Nettesheim, and J.R. Gilbert, (eds.), AEC Symposium Series No. 18, Oak Ridge, TN, Oak Ridge Division of Technical Information, U.S. Atomic Energy Commission, pp. 321-351.

McLaughlin JK. (1994). *Formaldehyde and cancer: a critical review*. Int Arch Occup Environ Health 66:295-301.

30

40

25

Mitchell R, Peat F, Caruso M. (1994), FOR Monitoring and Waste Water Branch of Office of the Environment Protection Authority, *Ambient monitoring at three sites in the Adelaide area with the POSIS Long Path Monitor*, Department of Environment and Natural Resources.

<sup>35</sup> Monticello TM, Morgan KT, Everitt JI, et al. (1989). *Effects of formaldehyde gas on the respiratory tract of Rhesus monkeys.* Am J Pathol 134:515-527.

Monticello TM, Miller FJ, Morgan KT. (1991). *Regional increases in rat nasal epithelial cell proliferation following acute and subchronic inhalation of formaldehyde*. Toxicol Appl Pharmacol 111:409-421.

Monticello TM, Swenberg JA, Gross EA, et al. (1996). *Correlation of regional and nonlinear formaldehyde-induced nasal cancer with proliferating populations of cells*. Cancer Res 56:1012-1022.

<sup>45</sup> Morata TC, Fiorini AC, Fischer FM, et al. (1997). *Toluene-induced hearing loss among rotogravure printing workers.* Scand J Work Environ Health 23(4):289-98.

Muller JF, Hawker DW, Connell DW. (1998). *Polycyclic aromatic hydrocarbons in the atmospheric environment of Brisbane, Australia.* Chemosphere 37:7 :1369-1383.

50

National Environment Protection Council (2000) National Environment Protection (Ambient Air Quality) Measure *Report of the Risk Assessment Taskforce*, NEPC October 2000.

Nelson KW, Ege JF Jr, Ross M, Woodman LE, & Silverman L (1943) Sensory response to certain
 *industrial solvent vapors.* J Ind Hyg Toxicol, 25: 282-285

<sup>10</sup> 

New Zealand Ministry for the Environment (2002), *Ambient air Quality Guidelines 2002*, Wellington New Zealand

NHMRC (National Health and Medical Research Council)(1999). *Toxicity assessment for carcinogenic soil contaminants* Canberra, Commonwealth of Australia

NICNAS (2001). National Industrial Chemicals Notification and Assessment Scheme, Benzene, Priority Existing Chemical Assessment Report No. 21 (2001)

<sup>15</sup> NPI (2000/2001) National Pollutant Inventory Database http://www.npi.gov.au/index.html

NSW EPA (New South Wales Environment Protection Authority) (1998) *Pilot air toxic project,* In *NSW air quality management plan: technical report,* Sydney, NSW EPA 98/21

20 NSW EPA (New South Wales Environment Protection Authority) (2002) Ambient air quality research project (1996-2001), 'Dioxins, organics, polycyclic aromatic hydrocarbons and heavy metals'.

NTP (National toxicology Program) (1998). *Eighth annual report on carcinogens.* Research Triangle Park, NC: US Department of Health and Human Services, Public Health Service, National Institute of Environmental Health Services.

Office of Environmental Health Hazard Assessment (OEHHA) (1993). *Benzo(a)pyrene as a Toxic Air Contaminant.* Part B. Health Effects of Benzo(a)pyrene. Air Toxicology and Epidemiology Section, Berkeley, CA.

30

45

55

25

5

Office of Environmental Health Hazard Assessment (OEHHA) Californian Environmental Protection Agency (1999) Air Toxics Hot Spots Program Risk Assessment Guidelines ,Part 11, Technical Support Document for Describing Available Cancer Potency Factors benzo[a]pyrene

<sup>35</sup> Office of Environmental Health Hazard Assessment (OEHHA) Californian Environmental Protection Agency, (1999), *Determination of Acute Reference Exposure Levels for Airborne Toxicants – Formaldehyde* 

 Office of Environmental Health Hazard Assessment (OEHHA) Californian Environmental
 Protection Agency, (1999), Determination of Acute Reference Exposure Levels for Airborne Toxicants – Toluene

Office of Environmental Health Hazard Assessment (OEHHA) Californian Environmental Protection Agency, (1999), *Determination of Acute Reference Exposure Levels for Airborne Toxicants – Xylenes* 

Ott G, Townsend JC, Fishbeck W, Langner RA (1978) *Mortality among individuals occupationally exposed to benzene*. Archives of Environmental Health, 33:3-10.

<sup>50</sup> Orbaek P, Nise G. (1989). *Neurasthenic complaints and psychometric function of toluene-exposed rotogravure printers.* Am J Ind Med 16:67-77.

Osborne MR and Crosby NT (1987). *Binding to proteins and nucleic acids*. In: *Benzopyrenes*. Cambridge, Cambridge University Press, pp 137-176 (Cambridge Monographs on Cancer Research).

Impact Statement for the National Environment Protection (Air Toxics) Measure

Panther BC, Hooper MA, Tapper NJ. (1999). A comparison of air particulate matter and associate polycyclic aromatic hydrocarbons in some tropical and temperate urban environments. Atmos Environ (in press)

<sup>10</sup> Partanen T. (1993). *Formaldehyde exposure and respiratory cancer - a meta-analysis of the epidemiologic evidence*. Scand J Work Environ Health 19:8-15.

Paustenbach DJ, Price PS, Ollison W, Blank C, Jernigan JD, Bass RD, Peterson HD (1992) *Reevaluation of benzene exposure for the Pliofilm (rubberworker) cohort (1936-1976).* Journal of Toxicology and Environmental Health, 36:177-231.

Pazdrak K, Gorski P, Krakowiak A, et al. (1993). *Changes in nasal lavage fluid due to formaldehyde inhalation*. Int Arch Occup Environ Health 64:515-519.

20 Pott F. Heinrich U.( 1990.) *Relative significance of different hydrocarbons for the carcinogenic potency of emissions from various incomplete combustion processes.* IARC Scientific Publications. (104):288-97.

Queensland EPA (2000) *Ambient Air Quality Monitoring in Queensland, 2000Annual Survey and trend Report.* Queensland Environment Protection Agency.

Rahill AA, Morrow PE, Frampton MW, Cox C, Gelein R, Speers D, Utell MJ. (1996). *Human performance during exposure to toluene.* Aviar Space Med 67:640-647.

Rickert DE, Baker TS, Bus JS, Barrow CS, Irons RD (1979) *Benzene disposition in the rat after exposure by inhalation*. Toxicology and Applied Pharmacology, 49:417-423.

Rinsky RA, Young RJ, Smith AB (1981). Leukemia in benzene workers. Am J Ind Med. 2: 217-245

<sup>35</sup> Rinsky RA, Smith AB, Hornung R,Fillonn TG, Young RJ, Landrigan PJ. (1987). Benzene and leukemia, an epidemiologic risk assessment. N Eng J Med, 316:1044-1050.

Ross D, Siegel D, Schattenberg DG, Moran JL (1996) *Cell-specific metabolism in benzene toxicity. A metabolic basis for benzene-induced toxicity at the level of the progenitor cell in human bone marrow.* Fundamental and Applied Toxicology, 30:339.

Rothman N, Li GL, Dosemeci M, Bechtold WE, Marti GE, Wang YZ, Linet M, Xi LQ, Lu W, Smith MT, Titenko-Holland N, Zhang LP, Blot W, Yin SN, Hayes RB. (1996). *Hematotoxocity among Chinese workers heavily exposed to benzene*. Am. J. Ind. Med. 29(3):236-246.

45

40

Rozman KK, (2000). The role of time in toxicology or Haber's  $c \times t$  product. Toxicology, 149 (1):35-42.

Shell Ltd (1995). *Ambient air monitoring: Benzene levels.* Shell Australia (1 Spring Street)
Internal Report.

Sneeuwjagt R (2002), Personal Communication, Western Australian Department of Conservation and Land Management.

5

15

<sup>5</sup> Snyder R (2000) *Recent developments in the understanding of benzene toxicity and leukemogenesis*. Drug and Chemical Toxicology, 23:13-25.

Snyder CA, Goldstein BD, Sellakamur A, Wolman S, Bromberg I, Erlichman MN, and Laskin S. (1978). *Hematotoxicity of inhaled benzene to Sprague-Dawley rats and AKR mice at 300 ppm.* J. Toxicol. Environ. Health 4:605-618.

Snyder R, Witz G, Goldstein BD (1993) *The toxicology of benzene*. Environmental Health Perspectives, 100:293-306.

<sup>15</sup> Snyder R, Hedli CC (1996) *An overview of benzene metabolism*. Environmental Health Perspectives, 104(suppl. 6):1165-1171.

Swenberg JA, Kerns WD, Mitchell RI, et al. (1980). *Induction of squamous cell carcinomas of the rat nasal cavity by inhalation exposure to formaldehyde vapor.* Cancer Res 40:3398-3402.

20

10

Tatrai E, Ungvary G, Cseh IR, et al. (1981). *The effects of long-term inhalation of ortho-xylene on the liver*. Ind Enviv Xenobiotica, *Proceedings of International Conference, Prague, Czechoslovakia, May 27-30, 1980.* New York, NY: Springer-Verlag, 293-300.

<sup>25</sup> Thiel R, Chahoud I. (1997). *Postnatal development and behaviour of Wistar rats after prenatal toluene exposure*. Arch Toxicol 71(4):258-65.

Thyssen, J., J. Althoff, G. Kimmerle and U. Mohr. (1981). *Inhalation studies with benzo(a)pyrene in Syrian golden hamsters.* J. Natl. Cancer Inst. 66: 575-577.

30

50

Torre P. (1995) *Some volatile organic compound analysis in Melbourne air environments,* EPA Scientific Advisory Panel Meeting, EPA Victoria

- Torre P, Bardsley TB Eriksen P. (1996) Volatile organic compound analysis in an industrial
   *location in Melbourne.* Proceedings of the 134<sup>th</sup> International Clean Air & Environment
   Conference, Adelaide, Australia, 22-25 September, Clean Air Society of Australia and New
   Zealand.
- Torre P and Bardsley TB et al. (1998) *Measuring volatile organic compounds during four mode of commuting in Melbourne.* Proceedings of the 14<sup>th</sup> International Clean Air & Environment
   Conference, Melbourne, Australia, 18-22 October, Clean Air Society of Australia and New
   Zealand.

Tsai SP, Wen CP, Weiss NS, Wong O, McClellan WA, and Gibson RL. (1983). *Retrospective mortality and medical surveillance studies of workers in benzene areas of refineries.* J. Occup. Med. 25(9):685-692.

Uchida Y, Nakatsuka H, Ukai H, Watanabe T, Liu Y-T, Huang M-Y, Wang Y-L, Zhu F-Z, Yin H, & Ikeda M (1993). *Symptoms and signs in workers exposed predominantly to xylenes*. Int Arch Occup Environ Health, 64: 597-605.

Ungvary G, Tatrai E, Hudak A, Barcza G, & Lorincz M (1980) *Studies on the embryotoxic effects of* ortho-, meta- *and* para-*xylene*. Toxicology, 18: 61-74

<sup>5</sup> Ungvary G & Tatrai E (1985) On the embryotoxic effects of benzene and its alkyl derivatives in mice, rats and rabbits. Arch Toxicol, 8(suppl): 425-430.

Ungvary G. and Tatrai E. (1985). *On the embryotoxic effects of benzene and its alkyl derivatives in mice,rats and rabbits.* Arch Toxicol (Suppl)8:425-430.

10

United Kingdom Expert Panel on Air Quality Standards (EPAQS) (1994). Benzene

United Kingdom Expert Panel on Air Quality Standards (EPAQS) (1999) Polycyclic Aromatic Hydrocarbons ,

15

U.S. Environmental Protection Agency (US EPA). (1984). *Health Effects Assessment for Benzo(a)pyrene*. EPA 540/1-86-022. Environmental Criteria and Assessment Office, Cincinnati, OH.

<sup>20</sup> US Environmental Protection Agency (US EPA. (1991). *Formaldehyde risk assessment update - final draft*. Washington, DC: U.S. Environmental Protection Agency, Office of Toxic Substances Disease Registry.

U.S. Environmental Protection Agency (US EPA). (2000) *Carcinogenic Effects of Benzene*: An Update (January 2000).

US Environmental Protection Agency (US EPA). (2002), *Integrated Risk Information System summary for Toluene* – (update)

<sup>30</sup> Vanderzalm LL, Hooper MA, Maenhaut W, Tapper NJ. (1998). *Particulate air quality and polycyclic aromatic hydrocarbons in regional Northwest Australia and Southeast Asia* In "14th International Clean Air and Environment Conference" Melbourne 18-22 October, pp433-438.

Vic Roads/EPA Victoria, (1991). PAH, Pm10, Total Particulate Matter and Lead Measurements at
 Debney's Park Estate, Flemington, 1990-1991, Vic Roads Study.

Vic Roads/EPA Victoria, (1994). Particle and carbonyl air monitoring, South Eastern Arterial Freeway, Malvern

40 von Euler G, Fuxe K, Hansson T, et al (1989b). *Persistent effects of neonatal toluene exposure on regional brain catecholamine levels and turnover in the adult male rat.* Toxicology 54:1-16.

Vrca A, Bozicevic D, Karacic V, et al (1995). *Visual evoked potentials in individuals exposed to long-term low concentrations of toluene*. Arch Toxicol 69(5):337-40.

45

Vrca A, Bozicevic D, Bozikov V, et al. (1997a). Brain stem evoked potentials and visual evoked potentials in relation to the length of occupational exposure to low levels of toluene. Acta Medica Croatica 51:215-219.

<sup>50</sup> Vrca A, Karacic V, Bozicevic D, et al. (1997b). *Cognitive evoked potentials VEP P300 in persons occupationally exposed to low concentrations of toluene.* Arh Hig Rada Toksikol 48:277-285.

Ward CO, Kuna RA, Snyder NK, Alsaker RD, Coate WB, and Craig PH. (1985). *Subchronic inhalation toxicity of benzene in rats and mice*. Am. J. Ind. Med. 7:457-473.

<sup>5</sup> Weber-Tschopp A, Fischer T, Grandjean E. (1977). *Irritating effects of formaldehyde on men.* Int Arch Occup Environ Health 39:207-218. (German)

WHO. (1989). World Health Organization. *Formaldehyde: Environmental health criteria*. Geneva: World Health Organization.

10

15

WHO. (1996). *Proposed Air Quality Guidelines for Europe*, -WHO Regional Office for Europe, Copenhagen

WHO. (2000). *Air Quality Guidelines for Europe, 2<sup>nd</sup> edition* -WHO Regional Office for Europe, Copenhagen.

Wong O. (1987a). An Industry-wide mortality study of chemical workers occupationally exposed to benzene I General Results. Br J Ind Med. 44:365-381.

20 Wong O. (1987a). An Industry-wide mortality study of chemical workers occupationally exposed to benzene II Dose-response analyses. Br J Ind Med. 44:382-395.

Wom O and Raabe K (1995). Cell-type-specific leukemia analyses in a combined cohort of more than 208,000 petroleum workers in the United States and the United Kingdom, 1037-1989. Reg Toxicol and Reg Pharmacol, 21:307-321.

Wouterson RA, van Garderen-Hoetmer A, Bruijntjes JP, et al. (1989). *Nasal tumors in rats after severeinjury to the nasal mucosa and prolonged exposure to 10 ppm formaldehyde*. J Appl Toxicol 9:39-46.

30

25

Yardley-Jones A, Anderson D, Parke DV (1991) *The toxicity of benzene and its metabolism and molecular pathology in human risk assessment*. British Journal of Industrial Medicine, 48:437-444.

Yin S, Li G, Hu Y, et al. (1987). *Symptoms and signs of workers exposed to benzene, toluene or the combination.* Ind Health 25:113-130.

Zavalic M, Mandic Z, Turk R, et al. (1998a). *Quantitative assessment of color vision impairment in workers exposed to toluene.* Am J Ind Med 33(3):297-304.

<sup>40</sup> Zavalic M, Mandic Z, Turk R, et al. (1998b). *Assessment of colour vision impairment in male workers exposed to toluene generally above occupational exposure limits.* Occup Med 48(3):175-180.

Zavalic M, Mandic Z, Turk R, et al. (1998c). *Qualitative color vision impairment in tolueneexposed workers*. Int Arch Occup Environ Health 71:194-200.

# **APPENDIX 1 - AIR TOXICS IN AUSTRALIA**

#### BENZENE

Sources of benzene in ambient air are natural processes and human activities that involve the combustion of organic material such as wood and coal and petroleum products. In the

industrial context benzene is used as a raw material to synthesise other chemicals. Nationally aggregated National Pollutant Inventory (NPI) data indicate that the major sources of ambient benzene emissions are transport related. Based on NPI data estimates other significant sources of ambient benzene include: domestic wood heaters; oil and gas extractive industries; lawn mowers; recreational boating; service stations; fires; iron and steel
 manufacturing; petroleum refining; and mineral/metal/chemical manufacturing and wholesaling.

As motor vehicles are currently a major source of benzene emissions, it is to be expected that benzene will be found throughout urban airsheds, with the highest relative concentrations likely to be associated with heavily trafficked roads. Industry related benzene emissions are likely to be in the areas close to the source rather than widely distributed across the airshed.

**Table A1.1** provides a summary table of jurisdictional monitoring data for benzene (all data are 24-hour averages unless otherwise indicated).

25

Jurisdiction	Monitoring site and characterisation	Number of samples	Mean (ppm)	Range (ppm)	Peak (ppm)
NSW (EPA 2002)	High traffic	24hr sample		(PP)	
	Sydney CBD	every sixth			0.0028
	General	day for five			
	Wollongong CBD	years			0.0007
	Newcastle CBD	5			0.0009
	Rozelle				0.0012
	St Mary's				0.00040 (ann average)
Victoria	High traffic				( C)/
(Vic EPA 1998)	Westgate Freeway &		0.0017	0.0002-	
	Altona (two sites)			0.0045	
Victoria	Industrial		0.0005 - 0.0014		0.0025
(Torre et al 1996)	Dandenong (six sites)				
Victoria (2001-2)	Traffic		0.0011 - 0.0019		0.0075
(Vic Roads 2001-2)					
Qld (2000-02)	High traffic				
(Qld EPA)	Brisbane CBD		0.001		0.0035
Qld (2000-02)	Industrial				
(Qld EPA)	Wynnum		0.0011		0.0024
Qld (2000-02)	General		0.0009		
(Qld EPA)	Springwood				0.0021
South Australia	High traffic				
(Agar 2000)	Keswick		0.009 (1hr av)		0.014 (1 hr av)
WA	High traffic				
(WA DEP 2002)	Duncraig	63	0.00168		0.01762
	Queen's Blvd	65	0.00202		0.0112
WA	Industrial				
(WA DEP 2002)	Hope Valley	33	0.00028		0.00105
	Kewdale	0.01	0.00043		0.00095
WA	General				
(WA DEP 2002)	Swanbourne	0.014	0.00039		0.0009
	Nth Fremantle	0.004	0.00037		0.00065
	Gooseberry Hill	0.004	0.00012		0.00021
	Joondanna	0.008	0.00037		0.00037

5 Note: the values shown for mean, range and peak levels are not comparable. The data are derived over different averaging times and the sample sized vary significantly

# FORMALDEHYDE

Formaldehyde is used for the manufacture of resins and textiles, disinfectants, fertilisers and
 as a laboratory fixative. It is also used in household products, such as pressed wood
 products, shampoo, nail polish and dishwashing liquids. Whilst formaldehyde vapours
 emitted from these products will impact on indoor air quality, the primary source of
 formaldehyde in ambient air in Australia is the incomplete combustion of fuel.

- <sup>15</sup> Nationally aggregated NPI data indicate that formaldehyde in Australia is emitted from a broad range of sources, with some significant sources including: the burning of solid fuel in homes; motor vehicles; lawn mowers; barbeques; and print shops.
- NPI data indicate that the main sources of formaldehyde in Australian airsheds are domestic
  and transport sources. The estimated attributed contribution from domestic sources ranges
  from 1% in South East Queensland to 95% in Hobart. The estimated attributed contribution
  from transport sources ranges from 4% in Hobart to 70% in Melbourne. Industry
  contributions are smaller, with estimated ranges from less than 1% in Perth, Adelaide,
  Canberra and Hobart to 39% in South East Queensland. Air monitoring data for
  formaldehyde are limited compared to other pollutants under consideration.

Jurisdiction	Monitoring site characterisation/l ocation	Number of samples	Mean (ppm)	Range (ppm)	Peak (ppm)
Victoria (Vic Roads, Vic EPA 1994)	<u>High traffic</u> Tooronga Rd CBD	4	0.00326	0.0004-0.0065 (19-17 hr av) 0.0021-0.0076 (20-26 hr av)	
Qld (Qld EPA)	<u>High traffic</u> Brisbane CBD		0.0032		0.0077
Qld (Qld EPA)	<u>Industrial</u> Wynnum		0.0057		0.0178
SA (Agar 2000)	<u>High traffic</u> Keswick		0.005–0.036 (30 min av)		
SA (Agar 2001)	<u>Industrial</u> Flinders Park		0.02 (30 min av)	0.005-0.03 (30 min av)	

**Table A1.2** provides a summary of jurisdictional monitoring data for formaldehyde (all data are 24-hour averages unless otherwise stated).

30

Note: the values shown for mean, range and peak levels are not comparable. The data are derived over different averaging times and the sample sized vary significantly

# POLYCYCLIC AROMATIC HYDROCARBONS(PAHS) MEASURED AS BENZO(A)PYRENE

<sup>35</sup> The main sources of ambient PAHs are combustion processes. These include motor vehicles, solid fuel heaters, tobacco smoke, cooking of food, bitumen and asphalt production plants, petroleum refineries, power plants using fossil fuels, coking ovens and aluminium refineries. Nationally aggregated NPI data indicate that PAHs in Australia are emitted predominantly from domestic solid fuel heating. Other sources include motor vehicles; lawn mowers; metal manufacturing; metal ore mining; recreational boating; chemical manufacturing; service stations; and petroleum refining.

10

5

NPI data indicate that the estimated attributable contribution from domestic sources (including bush fires, hazard reduction and agricultural burning) range from 23% in Darwin to 95% in Perth. The estimated attributable contribution from transport sources ranges from 8% in Hobart to 44% in Melbourne and South-East Queensland.

15

The estimated contribution from industry sources ranges from less than 1% in Hobart to 53% in Darwin. The data indicate that, with exception of Darwin, domestic sources are a major source of PAH emissions.

<sup>20</sup> Of all the air toxics under consideration in the proposed NEPM, PAHs are the most complex category as this group includes a large number of chemically varied compounds. The majority of studies have focussed on PAHs associated with particles, with data collected in areas near major roads or in wood smoke affected areas. Routine monitoring for PAHs is not conducted.

25

Given that the major source of PAHs is from combustion processes, emissions are generally widely distributed within the airsheds. Whilst industry contributes a small percentage of total PAH emissions, these impacts are likely to be in the areas close to the source rather than widely distributed across the airshed.

Table A1.3 provides a summary of jurisdictional monitoring data for benzo(a)pyrene (all data are 24-
hour averages unless otherwise stated

Jurisdiction	Monitoring site	Number of	Mean	Range (ng/m <sup>3</sup> )	Peak
	characterisation/location	samples	(ng/m³)		(ng/m <sup>3</sup> )
NSW (NSW EPA	Wood smoke- winter				
2002)	Sydney (av. of 6 sites)	52	0.46		2.25
	Wollongong (av. of 7 sites)	59	0.16		1.21
	Newcastle (av. of 3 sites)	31	0.30		1.92
	Tumut	14	1.12		3.06
	Cooma	13	1.12		3.02
	Armidale	5	1.3		3.77
	Lithgow	14	4.21		8.99
	_				
Victoria (1993)	High traffic		0.17		0.83
(Panther et al 1999)	Melbourne				
Victoria (Gras et al	Wood smoke - winter				
1992)	Alphington		13.33		
			(8 hr av)		
Qld (Muller et al	High traffic				
1998)	Woolloongabba	Averaging	1.5		2.0
	Fortitude Valley	time	0.27		0.33
	Mt. Gravatt	between two	0.14		0.23
	Dutton Park	and nine	0.21		0.44
	Graceville	days	0.013		
Qld (Muller et al	General				
1998)	Nathan	Averaging	0.10		0.22
	Redbank	time	0.008		
		between two			

Jurisdiction	Monitoring site	Number of	Mean	Range (ng/m <sup>3</sup> )	Peak
	characterisation/location	samples	(ng/m <sup>3</sup> )		(ng/m <sup>3</sup> )
		and nine			
		<u>days</u>			
ACT (Fox 1999)	High traffic				
	Civic	8		< 0.004-0.1222	
	Woden	9		< 0.004-0.095	
ACT (Fox 1999)	General				
	Gowrie	7		< 0.004	
WA (Gras 1996)	Wood smoke – winter				
	Duncraig	<u>(12 hr av.)</u>		1.011 - 19.701	
	_			0-6.809	
	Caversham	<u>(8-56 hr av)</u>			
		<u>(8-36 hr av)</u>		0-2.671	
	Swanbourne				
TAS (Expert Working	Wood smoke				
Party 1996)	Ti Tree Bend (1993)		1.24		7.46
1 urty 1000)	Newnham (1993)		1.29		9.67
	East Launceston (1993)		1.66		8.95
	Glen Dhu (1993)		1.18		7.15
	Newstead (1993)		1.10		4.57
NT Vanderzalm et al	General				
1998)	Darwin	51	0.09		
<i>'</i>	Jabiru East	70	0.02		

5 Note: the values shown for mean, range and peak levels are not comparable. The data are derived over different averaging times and the sample sized vary significantly

# TOLUENE

Toluene is synthesised in the process of refining petroleum and other fuels from crude oil, in
making coke from coal, and as a by-product in the manufacture of styrene. Toluene is used to manufacture paint, paint thinners, fingernail polish, lacquers, adhesives and rubber and in some printing and leather tanning processes. Toluene is significant in the aromatic component of petrol.

Nationally aggregated NPI data indicate that the primary source of toluene in ambient air is motor vehicle emissions. However there are a broad range of other sources including: architectural surface coatings; oil and gas extraction; domestic and commercial solvents/aerosols; lawn mowing; recreational boating; motor vehicle re-finishing; service stations; domestic solid fuel burning; motor vehicle manufacturing; petroleum refining; metal manufacturing and fires.

NPI data indicate that main sources of toluene in Australian air sheds are transport related with attributable contributions ranging from 20% in Darwin to 75% in Canberra. The contribution attributed to domestic sources range from 19% in Sydney to 42% in Darwin. Industry contributions are smaller ranging from 2% in Canberra to 38% in Darwin.

25

The data set for toluene is less comprehensive than for benzene. Air monitoring data for toluene have been collected from locations similar to those used for benzene monitoring, ie near major busy roads and industrial facilities.

<sup>30</sup> Given that the major source of toluene in Australian cities is motor vehicles, toluene is expected to be widely distributed within the airsheds, with highest relative concentrations likely to be associated with heavily trafficked roads. Whilst industry contributes a smaller <sup>5</sup> proportion to total toluene emissions, these impacts are likely to be experienced in areas close to the source rather than widely distributed across the airshed.

**Table A1.4** provides a summary of jurisdictional monitoring data for toluene (all data are 24-hour averages unless otherwise stated).

1	n
1	υ

Jurisdiction	Monitoring site characterisation/ location	Number of samples	Mean (ppm)	Range (ppm)	Peak (ppm)
NSW (NSW EPA 2002)	<u>High traffic</u> Sydney CBD <u>General</u>	24hr sample every sixth day for five	0.0042		0.01
	Wollongong CBD Newcastle CBD	years	0.001 0.0011		0.0034 0.006
Victoria (Bardsley 1997)	<u>High traffic</u> Geelong Rd Bunting Rd Judge St Fourth Ave Sunshine Rd Indwe St			$\begin{array}{c} 0.0009  0.0088\\ 0.0019  0.0183\\ 0.0014  0.0091\\ 0.0025  0.009\\ 0.0008  0.008\\ 0.0006  0.0089 \end{array}$	
Victoria (Torre et al 1996)	<u>Industrial</u> Dandenong 6 sites			0.0003-0.0048	
Victoria 2001-02 (Vic Roads 2002)	<u>Traffic</u>		0.0094		0.03
Queensland (Qld EPA)	<u>General</u> Springwood		0.0035		0.017
Queensland (Qld EPA)	<u>High traffic</u> Brisbane CBD		0.0041		0.035
Queensland (Qld EPA)	<u>Industrial</u> Wynnum		0.0031		0.0105
SA (Agar 2000)	<u>High traffic</u> Keswick			0 – 0.0277 (30 min av)	
SA (Agar 2001)	<u>Industrial</u> Flinders Park		0.005 (30 min av)	0-0.038	
WA (WA DEP)	<u>High traffic</u> Duncraig Queen's Bld	63 65	0.00297 0.00393	<0.0001-0.02996 <0.0001-0.01327	
WA (WA DEP)	<u>Industrial</u> Hope Valley Kewdale	33 10	0.00044 0.00164	<0.0001-0.00259 0.00056-0.00259	
WA (WA DEP)	<u>General</u> Swanbourne Nth Fremantle Gooseberry Hill Jondanna	14 4 4 8	0.00058 0.00066 0.00019 0.00109	<0.0001-0.00128 0.00042-0.00108 <0.0001-0.00028 <0.0001-0.00198	

Note: the values shown for mean, range and peak levels are not comparable. The data are derived over different averaging times and the sample sized vary significantly

# XYLENES

<sup>15</sup> Xylenes are synthesised in the petroleum refining process. Xylenes are used in the chemical industry for the production of paints, lacquers, insecticides, polyester fibre and dyes. Xylenes are also common in domestic products such as aerosol paints and lacquers, paints and thinners. Xylenes occur in petrol.

- <sup>5</sup> Nationally aggregated National Pollutant Inventory (NPI) data indicate the major sources of ambient xylene emissions are transport related. The NPI data indicate that other significant sources of ambient xylenes include: oil and gas extraction; lawn mowing, recreational boating; architectural surface coatings; motor vehicle refinishing; service stations; motor vehicle manufacturing; domestic solid fuel burning; and petroleum refining.
- 10

Based on NPI data estimates, the main sources of xylenes in Australian airsheds are transport related with attributable contributions ranging from 37% in Darwin to 83% in Canberra. The attributable contributions from domestic sources range from 12% in Sydney to 38% in Darwin. Industry contributions are smaller ranging from 1% in Canberra to 28% in Adelaide.

15 Adela

The air monitoring data set for xylenes is comparable to the data set for toluene. Air monitoring data for xylenes has been collected from similar locations to those used for toluene monitoring, being predominantly near major busy roads and industrial facilities. Given that the major source of xylenes in Australian cities is motor vehicles, xylenes would be expected to be widely distributed within airsheds. However, the highest relative concentrations are likely to be associated with heavily trafficked roads. Although industry contributes a lower proportion to total xylene emissions, these impacts are likely to be experienced in areas close to the source rather than widely distributed across the airshed.

25

20

**Table A1.5** provides a summary of jurisdictional monitoring data for xylenes (all data are 24-hour averages unless otherwise stated).

Jurisdiction	Monitoring site characterisation/location	Number of samples	Mean (ppm)	Range (ppm)	Peak (ppm)
NSW (NSW EPA 2002)	High traffic	24hr	(ppiii)		
	Sydney CBD	sample			
	(m,p xylene)	every sixth	0.0022		0.0062
	(o xylene)	day for	0.0008		0.0027
	General	five years	0.0000		0.0021
	Wollongong CBD	live years			
	(m,p xylene)		0.0005		0.0021
	(o xylene)		0.0002		0.0008
	Newcastle CBD		0.0002		0.0000
	(m,p xylene)		0.0006		0.0029
	(o xylene)		0.0002		0.0011
Victoria (Bardsley 1997)	High traffic				
	Geelong Rd				
	(m,p xylene)			0.0001-0.0027	
	(o xylene)			0.0001-0.0008	
	Bunting Rd				
	(m,p xylene)			0.0001-0.0019	
	(o xylene)			< 0.0001-0.0007	
	Judge St				
	(m,p xylene)			0.0001-0.0028	
	(o xylene)			0.0001-0.005	
	Fourth Ave				
	(m,p xylene)			0.0008-0.003	
	(o xylene)			0.0003-0.0009	
	Sunshine Rd				
	(m,p xylene)			0.0001-0.0026	
	(o xylene)			< 0.0001-0.0008	
	Indwe St				
	(m,p xylene)			< 0.0001-0.008	
	(o xylene)			< 0.0001-0.0014	

Jurisdiction	Monitoring site	Number of	Mean	Range (ppm)	Peak (ppm)
	characterisation/location	samples	(ppm)		
Victoria (Torre et al	Industrial				
1996)	Dandenong 6 sites				
	(m,p xylene)			<0.0002-0.0045	
	(o xylenes)			<0.0002-0.0008	
Victoria 2001-02 (Vic	Traffic				
Roads 2002)	(m, p xylene)		0.0055		0.018
	(o xylene)		0.0019		0.0062
Queensland (Qld EPA)	High traffic				
	Brisbane CBD				
	(p xylene)		0.0013		0.0023
Queensland (Qld EPA)	<u>Industrial</u>				
	Wynnum				
	(p xylene)		0.0014		0.003
Queensland (Qld EPA)	<u>General</u>				
	Springwood				
	(p xylene)		0.001		0.0022
SA (Mitchell et al 1994)	<u>High traffic</u>				
	North Terrace				
	(p xylene)		0.005 (1 hr		0.008 (1-hr
			av)		av)
WA (WA DEP)	<u>High traffic</u>				
	Duncraig				
	(m,p xylene)	0.063	0.00193	<0.0001-0.01672	
	(o xylene)	0.063	0.00078	<0.0001-0.00586	
	Queen's Blvd				
	(m,p xylene)	0.065	0.00238	<0.0001-0.01327	
	(o xylene)	0.065	0.00093	<0.0001-0.00476	
WA (WA DEP)	Industrial				
	Hope Valley				
	(m,p xylene)	0.033	0.00032	<0.0001-0.00286	
	(o xylene)	0.033	0.00011	<00.0001-	
	Kewdale			0.00055	
	(m,p xylene)	0.01	0.00104	0.00000.0.00100	
	(o xylene)	0.01	0.00036	0.00022-0.00186	
				<0.0001-0.00099	
WA (WA DEP)	<u>General</u>				
	Swanbourne	0.014	0.0004	.0.0001.0.00100	
	(m,p xylene)	0.014 0.014	0.0004 0.00018	<0.0001-0.00109	
	(o xylene) Nth Fremantle	0.014	0.00018	<0.0001-0.00057	
	(m,p xylene)	0.004	0.00014	0.000330.00051	
	(o xylene)	0.004	0.00014	0.000330.00031	
	Gooseberry Hill	0.004	0.00019	0.00014-0.00023	
	(m,p xylene)	0.004	0.00025	<0.0001-0.00043	
	(in, p xylene) (o xylene)	0.004	<0.00025	<0.0001-0.00043	
	Joondanna	0.004	~0.0001	~0.0001-0.00011	
	(m,p xylene)	0.008	0.00062	<0.0001-0.00145	
	(in, p xylene) (o xylene)	0.008	0.00082	<0.0001-0.00143	
	(O AYICHE)	0.000	0.00020	~0.0001-0.00000	
	for mean range and peak lay	1	1	data ana damirrad a	L

5

Note: the values shown for mean, range and peak levels are not comparable. The data are derived over different averaging times and the sample sized vary significantly

# **APPENDIX 2 - AUSTRALIAN PERSONAL EXPOSURE STUDIES**

#### **BTEX Personal Exposure Monitoring Study (October 2002)**

As part of the Commonwealth Government's Living Cities Air Toxics Program, the Department of Environmental Protection (WA) developed and coordinated a four city study which aimed to investigate the exposure of the urban population to benzene, toluene, ethylbenzene and xylene (BTEX). Participant in the study wore a passive BTEX sampler over 24 hours to monitor exposure to BTEX for five consecutive days.

Air Pollutant	Mean Exposure	Mean exposure
	[winter] (ppm)	[summer] (ppm)
Benzene	0.00125	0.00125
Toluene	0.00812	0.00529
Xylene	0.00623	0.00425
Ethylbenzene	0.00113	0.00081

- The mean BTEX personal exposure results were within the lower range of the personal 15 exposure measurements reported in the international literature. The lowest personal exposure measurement recorded for all the BTEX constituents were below the limit of detection. The most elevated exposure measurement recorded was 0.0238 ppm for benzene, 2.12ppm for toluene, 0.119 ppm for ethylbenzene and 0.697ppm for xylene. Elevated concentrations were found to be associated with non-occupational activities such as the use
- 20 of lacquer thinners, resins and house paints and exposure to spilt petrol.

The study used models to investigate the significant risk factors for increased BTEX exposure associated with specific activities and behaviours. Activities and behaviours found to significantly increase BTEX personal exposure included motor vehicle related activities such 25 as vehicle repair and machinery use refuelling of motor vehicles and time spent undertaking arts, crafts and woodwork. Time spent outdoors was found to decrease personal exposure to BTEX.

- For all of the BTEX constituents for both summer and winter, a highly significant difference 30 was found between the means exposure of participants in Perth and those in Adelaide, Melbourne and Sydney. Perth participant's BTEX exposures were significantly lower than the other three cities. This significant effect of the city of residence on participant exposure to BTEX is thought to be linked to more stringent fuel quality regulation in Western Australia.
- 35

10

#### 5

# APPENDIX 3 - CURRENT JURISDICTIONAL APPROACHES TO THE MANAGEMENT OF AIR TOXICS

# COMMONWEALTH

The Commonwealth has implemented a range of initiatives in recent years, which will impact on ambient levels of the air toxics under consideration. These initiatives focus on two general areas, namely: improved understanding of air toxics and source management.

# **IMPROVED UNDERSTANDING OF AIR TOXICS**

The Commonwealth has undertaken a number of investigations to improve our understanding of air toxics in the Australian context, particularly in terms of their ambient levels, sources, environmental fate and health effects. These projects include:

- development of the State of Knowledge Report on Air Toxics and Indoor Air Quality in Australia;
- funding a number of studies targeting air toxics (eg emissions from diesel vehicles,
- personal exposure monitoring of selected volatile organic compounds, determination of emission factors from in-service vehicles, characterisation of emissions from domestic solid fuel burning appliances); and
  - facilitating consultation at a national level and providing fora for information conduit through the Steering Group and Technical Advisory Group.

#### 25

20

# SOURCE MANAGEMENT

The Commonwealth has developed, implemented and promoted several measures to reduce emissions of air toxics and other pollutants from key sources, such as motor vehicles and domestic solid fuel heaters.

30

# Motor vehicle emissions

The Commonwealth has concentrated on improving the environmental performance of the transport sector, as it is a significant contributor to urban pollution, including air toxics. The following complementary strategies have been pursued:

- 35
- The progressive tightening of new vehicle emission standards. The *Motor Vehicle Standards Act 1989* provides the mechanism for setting national minimum standards for road vehicles, including standards for vehicle emissions. These standards are known as the Australian Design Rules. Australian Design Rule (ADR) 37/01 sets limits on exhaust and evaporative emissions of hydrocarbons from petrol operated passenger and light commercial. New ADRs have recently come into force with staged introduction of tighter emission standards. ADRs 79/00 and 79/01 set new emissions limits for light vehicles. ADR 80 sets new emission limits for total hydrocarbons from heavy duty diesel and LPG (liquefied petroleum gas) vehicles, and for methane and non-methane hydrocarbons from NG (natural gas) vehicles. These standards will be introduced
- 45

40

progressively from 2002 to 2006.

Li	Light vehicles (all fuels)		avy Vehicles (all fuels)
•	New ADR 79/00 adopts:	•	New ADR80/00 adopts:
•	Euro 2 in 2003/4 for petrol, NG and LPG	•	Euro 3 in 2002/3 (US MY2000 as alternative)
•	Euro 2 in 2002/3 for diesel	•	New ADR 80/01 adopts:
•	New ADR 79/01 adopts:	•	Euro 4 in 2006/7 (US 2004 as
•	Euro 3 in 2005/6 for petrol, NG and LPG		alternative)
•	Euro 4 in 2006/7 for diesel		

- Fuel quality standards to facilitate the introduction of new ADRs the Commonwealth Government has established legislation setting fuel quality standards. The *Fuel Quality Standards Act 2000* sets a legislative framework for managing, at a national level, fuel quality in Australia. The *Fuel Standard (Diesel) Determination 2001* sets fuel quality standards for diesel, the *Fuel Standard (Petrol) Determination 2001* sets fuel quality standards for petrol, and the *Fuel Standard (Liquefied Petroleum Gas) Determination* (under development) sets fuel quality standards for all of the air toxics under consideration they set standard for maximum content of PAHs in diesel and benzene in petrol (both to apply from 1 January 2006); and
  - The promotion of new, low emission, vehicle technologies, such as fuel cells. Funding of \$2 million is being provided to the Western Australian Department of Transport to trial the use of fuel cell technology in buses.

Combined, these actions will contribute to reduction in ambient levels of pollutants identified as posing a risk to the health of the Australian community.

# 25 Emissions from domestic solid fuel appliances

Domestic solid fuel appliances (such as woodheaters) have been found to significantly contribute to wintertime air pollution in some regions. While particle (principally  $PM_{2.5}$ ) pollution is often considered to be the main issue, solid fuel appliances are known also to contribute to ambient levels of the air toxics under consideration. To address these concerns, the Commonwealth has:

- encouraged all States and Territories to adopt the recently tightened Australian Standard for particle emissions from woodheaters (AS 4013);
- delivered a national awareness campaign aimed at reducing woodsmoke emissions by educating households in the correct operation of woodheaters;
- implemented a financial incentive scheme in Launceston to replace older woodheaters with heaters with reduced particle emissions this scheme has already been replicated by some State and Local governments in other woodsmoke-affected regions;
  - sponsored research to better understand the nature of woodsmoke emissions and ways to address these;
- delivered a training package for local government officers to deal with excessively smoky chimneys; and

10

15

20

 fostered complementary initiatives from State and Local Governments, equipment manufacturers and other stakeholders – these have included new woodsmoke regulatory measures, improved air monitoring facilities, the promotion of less-polluting technologies and community education about the dangers of woodsmoke exposure.

#### 10 NEW SOUTH WALES

# **NSW** MANAGEMENT STRATEGIES

A key strategy for NSW in managing air toxics was to undertake a broad program of monitoring from 1995 to 2001 to gather a better understanding of current levels of toxics in NSW. This included monitoring for a broad range of pollutants: dioxins, polycyclic aromatic hydrocarbons, 41 organic pollutants, including benzene, toluene and xylene; and some heavy metals.

In general, the levels of pollutants found were low. Dioxins were close to background levels indicating that controls on industrial sources have been effective. Levels of metals were also found to be low.

With regard to the pollutants being considered in this NEPM:

- Levels of benzene approached, but did not exceed, current international standards. Samples were collected at existing NEPM monitoring stations to represent general neighbourhood levels of exposure. The highest levels were found in the central business
- district and these are likely to be generated by emissions from motor vehicles.
- Limited monitoring for polycyclic aromatic hydrocarbons was undertaken, primarily in winter months. Whilst the health impact from PAHs being considered is chronic and therefore best represented by an annual average, only limited monitoring was undertaken in peak periods. Some towns in the Tablelands of NSW generated readings that were higher than current international annual average goals.

The management strategies for these pollutants focus on controls for each of the major sources: vehicles; industry; and solid fuel heaters.

35

40

30

15

20

25

# Motor vehicles

As motor vehicles are the major source of benzene (see predicted emissions for a model city in Table 1), strategies to reduce the contribution they make are vital. Significant gains to air quality have been achieved by improvements in fuel quality and vehicle emission performance.

Source of benzene emissions	%
Motor vehicles (petrol, diesel and LPG)	79
Industrial sources	11
Lawn mowers	6
Service stations	3
Domestic solid fuel burning	2
TOTAL	100

Table 1: Predicted daily releases of benzene within a model Australian urban centre

**Source:** *Draft Priority Existing Chemical Assessment Report—Benzene*, National Industrial Chemicals Notification and Assessment Scheme, Commonwealth of Australia, June 2001

While Table 1 only model emissions for a model Australian urban centre, actual emissions inSydney are expected to be similar to these figures.

Motor vehicles are also the major sources of other organic air toxics, such as toluene, xylene, ethylbenzene and styrene. Reducing emissions from motor vehicles will lower levels of these substances as well.

15

20

25

50

5

A broad reform program for vehicle performance will reduce these emissions over the next 10 years:

- **Fuel quality standards** (based on European regulations) will reduce emissions from vehicles. From 2006, the benzene content of petrol will be limited to a maximum of 1%, compared with the current legal maximum of 5%. This reduction will have an immediate and sustained impact on ambient benzene levels.
- New vehicle standards: European emission standards have been adopted and will dramatically reduce emissions from all new vehicles. The adoption of the European standards started in 2002 for diesel and commences in 2003 for petrol vehicles. Further emission reductions are required for new petrol vehicles from 2005 and new diesel vehicles from 2006. The NSW Government's Cleaner Vehicles Plan will promote the early uptake of vehicles meeting the even more stringent emission standards which will be required in Australia in the medium term, such as Euro 3 and Euro 4.

 Low volatility program: The NSW Government has previously established a Memorandum of Understanding with the oil industry to reduce the volatility of petrol during summer. This has resulted in less evaporation of volatile organic compounds, including benzene. The EPA is exploring the possibility of legally formalising the Regulation to ensure achievement of the desired reductions of volatile organic compounds and consistent compliance across the industry.

- **Emissions testing for cars:** The Roads and Traffic Authority has established two emissions testing stations at Botany and Penrith where owners can have their cars tested on a voluntary basis. The testing provides a short diagnostic report indicating the levels of emissions from each vehicle relative to all vehicles tested to date.
- Reduced vehicle use: The Government's Action for Air strategy contains a range of programs to address the issue of vehicle use. These include the promotion of appropriate land-use planning and infrastructure development to reduce reliance on motor vehicles and thus overall emissions. Actions agreed to at the Government-sponsored Clean Air Forum in November 2001 will further enhance existing programs. Key actions include the draft State Environmental Planning Policy (SEPP) on Integrating Land Use and Transport and the improvements and enhancements to the public transport system contained in Action for Transport 2010.
  - Vehicles using alternative fuels, such as hybrid electric vehicles and those running on compressed natural gas (CNG) and liquefied petroleum gas (LPG), will also reduce emissions. State Transit has already converted over 400 of its buses to CNG in the Sydney region.

Impact Statement for the National Environment Protection (Air Toxics) Measure

## 5 Industrial and commercial sources

Benzene has been eliminated in all but trace amounts in most industrial and domestic chemical products. The main individual industrial sources of benzene and related substances (such as toluene and xylene) are fuel refining, the manufacture of chemicals and combustion in cokeworks at steel plants.

10

15

PAHs are usually generated as a by-product of the incomplete combustion of organic material. The EPA regulates PAHs by controlling the effectiveness of combustion processes, as indicated by the carbon monoxide or particle emissions they produce. By controlling these pollutants, the PAHs produced are also controlled. Where an industrial process is particularly associated with PAH emissions, a specific monitoring program may be required to confirm that emission levels are acceptable.

to confirm that emission leve

A major element of the EPA's strategy to control recognised industrial emissions from existing sources has been through pollution reduction programs (PRPs) attached to 20 environment protection licences. Standards for new developments have also been strengthened over time as technology and knowledge have improved. Together, these have substantially reduced emissions from point sources.

- As one of the largest sources of benzene and other air toxics, BHP's Port Kembla steelworks has been covered by several five-year PRPs to reduce these emissions. This has included a \$93-million program to collect and reduce fugitive emissions from the coke ovens and \$2 million to control emissions from gas processing. The need for further controls on gas processing will be assessed after studies of emissions monitoring data.
- <sup>30</sup> In a comprehensive study in 2000–01, BHP assessed the sources and contribution of air toxics emissions from its Port Kembla steelworks. The study has allowed the identification of priorities for future reduction strategies which are currently being negotiated with BHP.
- Oil refineries are another industrial source of benzene and related air toxics. Almost 95% of emissions of benzene from refineries are fugitive or evaporative. Current reduction programs include detection of leaks and their repair to prevent the escape of benzene and other toxics from production processes; licensing fees based on pollutant loads to encourage better performance; and pollution reduction programs as part of environment protection licences.
- 40

45

50

Emissions from 'fuel marketing' are generally vapours lost from service stations and fuel depots, and transfers to bulk transport, such as road tankers and ships. The Clean Air (Plant and Equipment) Regulation 1997 requires vapour recovery systems to be attached to bulk fuel-handling facilities and Sydney service stations to reduce benzene emissions. The EPA is evaluating further controls on other fuel-handling equipment, such as controls at petrol

stations to collect fugitive vapours.

The use of licence fees based on the load of pollutants produced ('load-based licensing' or LBL) will continue to provide a financial incentive for licensed premises to find ways to reduce their emissions. The current LBL regime includes fees for a number of air toxics, such as benzene. The number of air toxics covered by LBL could be extended if necessary.

Current EPA cleaner production and education initiatives aimed at lowering emissions from dry-cleaning premises should reduce localised exposures to the organic air toxic tetrachloroethylene. Similar programs for spray painting and panel repair shops, and the

surface coating industry are expected to yield further localised reductions in emissions of 5 toluene and xylene. The fibreglass and composites industries also have a program to lower local exposure to styrene.

The EPA will continue to assess new industrial proposals for compliance with current goals and identify local air toxics issues in conjunction with local councils.

# **Solid fuel heaters**

Benzene and PAHs are emitted from solid fuel heaters through the combustion of carbonbased fuels, such as wood and coal. Poor combustion is the leading factor in emissions from these heaters.

15

10

20

These emissions have already been tackled on a number of fronts. A revised Australian Standard adopted in NSW sets emission limits for particles from new solid fuel heaters. Lower particle emissions will lead to a proportional reduction in emissions of PAHs. This standard has been formally adopted in the Clean Air (Domestic Solid Fuel Heaters) Regulation 1997.

The NSW Government committed \$1 million to the Clean Air Fund in 2002 to reduce smoke in the regional areas identified in the study as having high levels of pollution: Armidale, Cooma, Lithgow, Orange and Tumut (plus the Blue Mountains). Under the program, home 25 owners and businesses are offered a financial incentive to replace older, more polluting heaters with new, cleaner alternatives. The program is also supporting local council education and enforcement programs to ensure heaters are operated properly and do not emit excessive smoke. Another \$500,000 has been allocated to this program in 2003 which has been extended to include Wagga, Goulburn and Wingecarribee Councils. 30

The Government has actively promoted better use of solid fuel heaters through education campaigns, including publication of the EPA 1999 guideline, Selecting, Installing and Operating Domestic Solid Fuel Heaters. Other education programs include 'Don't light tonight, unless your heater's right' alerts when weather conditions threaten the dispersal of particle pollution in the metropolitan region; screening of information on better operation of heaters on regional television; and a comprehensive website on woodsmoke.

Under State planning legislation, local councils are able to tailor local planning instruments to prevent or restrict the installation of solid fuel heaters. Pittwater and Waverley Councils 40 have recently amended their Development Control Plans and Local Environmental Plans to restrict the installation of new heaters in their local areas. Other councils, such as Blue Mountains, Eurobodalla, Rockdale and Wollongong have published revised local approvals policies which clearly specify their requirements for installing heaters.

45

55

35

Local councils are also able to prevent owners allowing their heaters to emit excessive smoke by issuing notices under the Protection of the Environment Operations Act 1997.

The effectiveness of this comprehensive program on solid fuel heaters will continue to be monitored to determine whether additional initiatives are needed to achieve the necessary 50 reductions in woodsmoke and associated PAHs.

# VICTORIA

In Victoria the primary legislation that guides the approach to protection of the environment and EPA Victoria's environmental systems and practices is the Environment Protection Act

- 5 *(1970).* The Act allows for the development of a range of instruments that guide the protection of Victoria's air environment. This Act established the Environment Protection Authority (EPA) and defines its powers, duties and functions. The Act's provisions include statutory powers, instruments and measures to:
  - manage environmental quality;
- 10 establish environmental standards and criteria;
  - regulate emissions, discharges and wastes; and
  - prevent and clean up pollution.

The important instruments for environmental management include State Environment Protection Policies (SEPPs), industrial waste management policies, regulations, works approvals, licences and pollution abatement notices.

SEPPs establish a statutory framework for protecting the environment. The Governor in Council declares SEPPs, on the recommendation of EPA. These policies:

- identify the beneficial uses of the environment (including particular segments such as the air environment, or a particular water body or catchment) that are to be protected;
  - establish environmental indicators and associated environmental quality objectives to establish if the environment is being protected; and
  - define programs for attainment of these objectives so that identified beneficial uses are adequately protected.
  - Attainment programs usually specify a range of approaches, measures and instruments for policy implementation, and usually require the compliance and cooperation of government agencies, industry and the community to manage sources of pollution, reduce environmental
- <sup>30</sup> impacts and improve environmental quality. Cleaner production practices are a major focus of the approach to controlling emissions from industry in Victoria. The successful Environment Improvement Plans (EIPs) developed in partnership between industry and local communities have led to a greater understanding of industrial emissions in Victoria and continued improved industry performance. EIPs include action plans for continuous
- <sup>35</sup> improvement in the performance of individual industries and provide the communities with some confidence that emissions from the industries will reduce over time.

SEPPs provide the management approach and technical basis for the application of regulations, works approvals, licences and other statutory measures to manage the environment. The application of these instruments and measures must always be consistent

with the requirements of SEPPs.

#### **SEPP**S PROTECTING AIR QUALITY

Two SEPPs currently protect the air environment in Victoria. These were created in February 1999 by dividing the *State Environment Protection Policy (The Air Environment)* (made in 1981 and subsequently amended several times) into two policies:

- The State Environment Protection Policy (Ambient Air Quality) or SEPP (Ambient Air Quality); and
- The State Environment Protection Policy (Air Quality Management) or SEPP (AQM).

50

40

45

<sup>5</sup> The SEPP (Ambient Air Quality) contains the indicators, standards, goals and monitoring and reporting protocol of the Ambient Air Quality NEPM. The SEPP (Ambient Air Quality) also includes an ambient air objective for visibility reducing particles.

The SEPP (AQM) sets the framework for managing emissions to the air environment. These
 emissions are managed in such a way as to ensure that the air quality objectives of the SEPP (Ambient Air Quality) are met. In addition, a philosophy of continuous improvement is also pursued. The Principles of Environmental Protection contained in the *Environment Protection Act 1970* are explicitly stated in the SEPP (AQM) and guide the management of emissions to the air environment in Victoria. The focus is on the application of the waste hierarchy with avoidance being the primary aim rather than end-of pipe controls.

MANAGEMENT PRACTICES FOR AIR TOXICS

The SEPP (AQM) classifies pollutants into Class 1, 2 and 3 indicators. Pollutants are classified according to their toxicity, odorous properties and persistence in the environment.

- All generators of emissions of Class 1, 2 or 3 indicators must control their emissions by the application of best practice. For Class 3 indicators generators must control emissions to the maximum extent achievable (MEA). MEA is a move away from technological controls of pollution to application of the waste hierarchy. The focus of this approach is to avoid, reduce and reuse with the end-of-pipe controls being the last consideration. Design criteria
- have been set for these indicators and all applicants for Works Approval and licences must ensure that emissions of these substances are managed in such a way that the design criteria are not exceeded at ground level. Design criteria are modelling tools to be used in the design stage of an operation.
- <sup>30</sup> Formaldehyde, toluene and xylenes are classified as Class 2 indicators in the SEPP (AQM). Benzene and PAHs (BaP as a marker) due to their carcinogenic properties are classified as Class 3 indicators. The design criteria for toluene and xylenes are based on their odorous properties which is more stringent than the toxicity based values. The SEPP (AQM) also specifies intervention levels for benzene, formaldehyde, PAHs, toluene and xylenes. An
- <sup>35</sup> intervention level is a local air quality objective that can be used to assess the cumulative impacts of emissions in a local area. If an intervention level is exceeded then a Neighbourhood Environment Improvement Plan may be triggered.
- EPA Victoria has recently reviewed the Motor Vehicle Regulations in Victoria with the aim
   of reducing pollution, including air toxics, from motor vehicles. This review was part of the
   implementation of the Diesel NEPM. Other initiatives to implement the Diesel NEPM
   include the development of a diesel eco-maintenance program to improve the skills of
   existing and new diesel mechanics so that they are better equipped to recognise and address
   diesel vehicle emission issues. EPA Victoria is also investigating opportunities to develop an
   in-service audit capability for diesel vehicles.

EPA is currently finalising an Air Quality Improvement Plan for the Port Phillip Region that encompasses Melbourne and Geelong. AQIP contains a range of measures to ensure improvement in air quality in the region over the next 20 years. A range of measures that will lead to reductions in air toxics are included.

Victoria is also developing a Metropolitan Strategy, which aims to create a vision for retaining and enhancing Melbourne's livability over the next 30 years. The strategy will outline how the city can be shaped to better meet the needs of all people who live, work, visit and use services in Melbourne and the surrounding region. It will provide a framework for

Impact Statement for the National Environment Protection (Air Toxics) Measure

50

<sup>5</sup> local and state government to implement policies and plans that are responsive to the social, economic and environmental needs and challenges facing the metropolitan region over the next two to three decades.

Growth in motor vehicle kilometres travelled and associated emissions is one of the key challenges for the region and the Metropolitan Strategy will take this into account in developing a strategic integrated transport and land use planning framework that manages the demand for motor vehicle travel.

An Industrial Waste Management Policy for Domestic Solid Fuel Heating is also being introduced that will ensure that all wood heaters manufactured and installed in Victoria will comply with Australian Standard 4013. As domestic wood heating contributes up to 70% of PM<sub>2.5</sub> during winter in Melbourne, introduction of this standard will assist in reducing ambient particle levels and associated levels of PAHs. EPA Victoria is also working with the Fire Protection Agencies in Victoria (DNRE, CFA) to develop an approach to reduce the impact of hazard reduction burning and agricultural burning on air quality. A Protocol for Environmental Management will be developed and incorporated under the SEPP (AOM) to

Environmental Management will be developed and incorporated under the SEPP (AQM) to address these issues.

# QUEENSLAND

<sup>25</sup> Management of air toxics is achieved through the combined outcomes of a range of legislation, strategies and programs aimed at managing ambient air quality in general. These have been collected and prioritised for South East Queensland under a strategic plan entitled "South East Queensland Regional Air Quality Strategy", 1999, but the principles, as discussed below, apply throughout Queensland.

# **MOTOR VEHICLES**

30

National limits are set for hydrocarbon emissions (exhaust and evaporative) from new vehicles under Australian Design Rules. These limits have featured a series of reductions over the last decade, and further reductions will occur over the next five years. This results in on-going reductions in air toxic emissions from the Queensland vehicle fleet as

<sup>35</sup> in on-going reductions in air toxic emissions from the Queensland vehicle fleet as progressively less polluting vehicles gradually form greater proportions of the fleet due to vehicle write-offs after accidents and retirement of old vehicles.

Queensland Transport operates smoky vehicle and on-road vehicle emission random testing
 programs to encourage appropriate maintenance standards, and thus lower emissions, including air toxics.

Fuel quality specifications under the *Environmental Protection Regulations 1998* include a limit of 3.5 percent on the benzene content of petrol. Together with the national limit of 1 percent
<sup>45</sup> benzene due to commence in 2006, and the national limit on total aromatics, this contributes to significant on-going reductions in emissions of benzene, toluene and xylenes from petrol distribution and sales as well as vehicle operation.

# COMMERCIAL, INDUSTRIAL AND DOMESTIC SOURCES

- <sup>50</sup> Under the *Environmental Protection Act 1994*, "A person must not carry out an activity that causes, or is likely to cause, environment harm unless the person takes all reasonable and practicable measures to prevent or minimise the harm." A range of enforcement provisions is available including on-the-spot fines, orders to reduce or stop emissions, and prosecutions. The general provisions of the Act can be applied to air toxics sources with the aim of achieving best practice environmental management. Medium to large industrial activities
  - Impact Statement for the National Environment Protection (Air Toxics) Measure

<sup>5</sup> are also subject to licensing requirements, which could include conditions of approval aimed at minimising emissions of air toxics.

# HAZARD REDUCTION AND ECOLOGICAL BURNING

The organisations responsible for hazard reduction and ecological burning work closely with the Bureau of Meteorology and the Environmental Protection Agency to determine the most appropriate burning methods and the most suitable times for burning. The aim is ensure that all burning takes place in a way that minimises the impact of smoke, and any associated air toxics, on populated areas.

#### 15 **DOMESTIC WOOD HEATERS AND STOVES**

Under the *Environmental Protection (Air) Policy 1997*, all new domestic wood heaters and stoves sold in Queensland must comply with Australian Standard 4013, which sets limits on particle emissions. Provided these appliances are operated according to the manufacturers instructions, this has the effect of reducing all emissions, including air toxics. The Queensland government also supports the national voluntary code of practice for firewood merchants, which aims to improve the quality of retail firewood and thus reduce emissions when the fuel is burnt.

#### WESTERN AUSTRALIA

#### 25 **AIR TOXICS STUDIES**

20

A study of airborne Volatile Organic Compounds (VOCs) was undertaken by the Department of Environmental Protection in Perth from March 1997 to November 1998 to assess Perth's ambient air for VOC composition and concentration. Twenty three air toxics were detected in Perth, with toluene, benzene and the xylenes the most common air toxics found at all sites. The findings indicated that motor vehicles and domestic wood heaters

<sup>30</sup> found at all sites. The findings indicated that motor vehicles and domestic wood heaters were likely sources.

# LEGISLATION

The *Environmental Protection Act 1986* includes a number of statutory measures that can control air toxics in Western Australia.

A State Environmental Protection Policy is being developed under Part III of the Act that will implement the National Environment Protection Measure (NEPM) for Ambient Air. It is expected that the EPP would refer to the NEPM standards for criteria pollutants and any future air toxics standard. The EPP would allow for the development of monitoring programs and environmental management plans for control of air toxics.

Part IV of the *Environmental Protection Act 1986* deals with environmental impact assessment of significant proposals in Western Australia and provides an opportunity to assess and set conditions on proposals with the potential to emit air toxics. Part V of the Act covers licensing of existing industries and allows limits to be set on emissions.

A number of regulations exist under the *Environmental Protection Act 1986* which impact on air toxic emissions. These include the *Environmental Protection (Diesel and Petrol) Regulations 1999*, which set strict limits on levels of benzene in petrol, as well as on Reid vapour pressure and on aromatic and olefin content. The *Environmental Protection (Domestic Solid Fuel Burning Appliances and Firewood Supply) Regulations 1998* ban the sale of green firewood and wood heaters that do not comply with the Australian Standard (AS4013).

50

40

# 5 **PERTH AIR QUALITY MANAGEMENT PLAN**

The Perth Air Quality Management Plan (AQMP), released December 2000, and associated Implementation Strategy (released June 2002) include actions to reduce emissions from of air toxics from all sources, and in particular domestic wood heaters, motor vehicles and planned burning.

10

15

Smoke from domestic wood heaters is considered a significant source of air toxics during winter. In addition to the Regulations described above, the Perth AQMP includes programs aimed at education and behaviour change to reduce smoke from wood heaters, as well as the continued issuing of 'haze alerts' to warn wood heater owners when haze episodes are likely and encourage the careful operation of their wood heaters.

Motor vehicles are another significant source of air toxics in Western Australia. A number of behaviour change programs are continuing in Perth to encourage greater use of public transport and other alternatives to vehicle use. The Perth AQMP also includes actions to:

- Support the consistency and early adoption of Australian Design Rule emission standards with international best practice emission standards;
  - Evaluate LPG and CNG as fuel sources for the passenger and freight sectors;
  - Evaluate emissions testing options for introduction to Perth;
  - On-road enforcement of controls on excessive vehicle emissions.
- 25

Diesel vehicles are a source of PAHs and formaldehyde, so implementation of the Diesel NEPM will complement AQMP actions.

During spring and summer, a potential source of air toxics is smoke from biomass burning (controlled burns or bush fires). The Perth AQMP includes programs to reduce the impacts from these sources by improving co-ordination between agencies to ensure that smoke from controlled burning activities does not impact on population centres.

# DEVELOPMENT OF AMBIENT AIR QUALITY GUIDELINES FOR WESTERN AUSTRALIA

<sup>35</sup> In response to a growing need by industry and the community, guidelines are being developed for priority air toxics. As an interim position, World Health Organisation (WHO) Ambient Air Quality Guidelines are being used where available.

# SOUTH AUSTRALIA

<sup>40</sup> The current South Australian management strategy for air toxics stems from the *Environment Protection Act 1993*, which includes a general environmental duty for anyone undertaking any activity to avoid creation of environmental harm by all reasonable and practicable means. It also establishes a framework for development and application of specific controls on air pollutants including those classed as air toxics.

45

Priorities for management have been derived from air pollutant inventories that identified the significant sources, the most recent arising from the National Pollutant Inventory NEPM. The principles source classes are motor vehicles, industrial processes and domestic combustion sources.

50

55

Industrial sources have been subject to the longest program of emission control and management, through conditions attached to licences for prescribed activities with significant pollution potential. Activities not subject to the licence provisions of the legislation have been managed using Technical Bulletins that detail codes of practice for the activity or industrial process. Discharge concentration limits for air toxics have been set

- based upon international best practice, and these are combined with design maximum ground level concentrations published for a large range of substances.
   EPA conducts Pollution Prevention workshops for industry to promote replacement of substances and processes that result in air toxics release.
- <sup>10</sup> The EPA and its predecessors have conducted "hot spot" source related monitoring campaigns to investigate the potential for environmental harm and to validate predictions of ambient levels near potential significant sources. Four long path optical spectrometers have been commissioned in the past year to obtain continuous ambient concentration data that will help identify sources and characterise community exposure to BTEX and formaldehyde.
- 15 The instruments may be extended in their capability to measure other "toxics" compounds in future.

Motor vehicles are the most significant source of air toxics, and the fuel composition in SA is governed by the *Environment Protection (Motor Vehicle Fuel Quality) Policy 2002.* This Policy

- <sup>20</sup> was specifically designed to reduce emission of benzene and other health-related air toxics by 170 tonnes per year of benzene equivalent, as well as address air quality issues such as photochemical smog. Other fuel initiatives reducing air toxics emissions have included conversion of over 10% of the State government fleet to LPG; the metropolitan taxi fleet is also predominantly LPG fuelled. The Government bus fleet of approximately 650 includes
- 25 203 buses using compressed natural gas (CNG) to reduce emissions such as air toxics and Government has recently announced (August 2002) the purchase of another 100 CNG buses in its procurement program for the next 5 years.
- South Australia has been a strong advocate and supporter of Australian Standards for slow combustion heaters, and has pressured the manufacturers to improve their design and reduce emissions.

# TASMANIA

Activities taking place in Tasmania to manage air toxics include:

- Making of Air Quality Environment Protection Policy (scheduled for late 2002). This covers sources of air toxics and other pollutants and includes diffuse sources (eg domestic wood heating) and industry. A copy of the impact statement and draft policy may be found at <a href="http://www.dpiwe.tas.gov.au/inter.nsf/WebPages/CDAT-53M4U8?open">http://www.dpiwe.tas.gov.au/inter.nsf/WebPages/CDAT-53M4U8?open</a>
- Wood heater buy back and associated program in Launceston involving Environment Australia, Launceston City Council and Department of Primary Industries, Water and Environment. Wood heaters are considered to be a major source of particles and air toxics (PAHs, benzene and formaldehyde) in Tasmania.
- An education officer co-ordinates the Airwatch Tasmania program as well as being involved in community education on ways of reducing wood heater emissions.
  - Partnership agreements with Councils addressing local air quality issues.
  - After making of the Air Quality Policy, the development of a strategy to address other management instruments to assist the State in meeting the goals of the Ambient Air Quality NEPM. By reducing particle emissions from wood heaters, air toxics will also be reduced.
- 50
- Management strategies aimed at reducing PM10 in Tasmania should also effectively reduce air toxics.
- Monitoring of air toxics is not conducted in Tasmania at present.

# 5 AUSTRALIAN CAPITAL TERRITORY

#### **CURRENT MANAGEMENT STRATEGIES**

Air toxics are emitted from a number of sources but due to a lack of industry ACT emissions are dominated by transport emissions and during winter emissions from domestic wood heating.

10

The ACT Government has implemented a number of initiatives in recent years to target emissions from wood heaters including the ACT Firewood Strategy, the licensing firewood merchants (this replaced the previous voluntary code of practice) and the introduction of an air pollution warning system.

15

20

The ACT Government introduced the ACT Firewood Strategy in 1999, the first jurisdiction in Australia to develop such a program. The strategy aims to protect native forest and habitat without adversely affecting the air quality of the ACT and surrounds. The strategy has a strong ongoing public awareness campaign focussing on the correct operation and maintenance of wood heaters to reduce smoke emissions. This includes inspectors from Environment ACT targeting excessively smoky chimneys and providing advice on ways to minimise smoke.

- In April 2001 amendments to the Environment Protection Act 1997 (the Act) were enacted to introduce a mandatory licensing scheme for wood merchants. Merchants must now comply with a set of authorisation conditions including only selling dry seasoned timber and providing consumers with information on how to operate their wood heater correctly. Both of these conditions relate specifically to reducing air pollution.
- <sup>30</sup> Under Section 4 of the Act only heaters which are certified to Australian Standard 4013 (currently 4 grams of particle emissions per kilogram of fuel burnt) can be sold in the ACT. It is also an offence to modify a certified heater.
- In June 2001 the *"Don't Burn Tonight"* campaign was launched. The campaign operates over the winter months and aims to improve air quality by calling on Canberrans who use wood fired heaters to use alternative heating sources, if possible, on nights when atmospheric conditions will prevent the dispersion of wood smoke.

Emissions from motor vehicle have been addressed specifically at a national level through the progressive tightening of new vehicle emission standards and the introduction and enforcement of tighter fuel standards.

# **FUTURE MANAGEMENT STRATEGIES**

- Environment ACT is investigating the suitability of a woodheater replacement program similar to that implemented by the NSW EPA and the joint program being implemented in Launceston. The aim of this program would be to replace older more polluting heaters by offering a subsidy to install cleaner forms of heating. This, along with existing strategies should lead to reductions in PAHs and other air toxics associated with wood burning.
- <sup>50</sup> Given the small industry base in the ACT, industrial emissions are not a major source of air toxics. Any emissions are controlled through licensing of industry.

The introduction of the national fuel quality standards and the tightening of new vehicle emission standards will assist in the reduction of benzene and other motor vehicle pollutants over time.

55 over ti

## NORTHERN TERRITORY SOURCES OF AMBIENT AIR TOXICS

The sources, patterns and implications of ambient air toxic emissions are poorly defined in the Northern Territory. Monitoring of ambient air toxics is not conducted at present. Some emission estimation studies of air pollutants (including 8 air toxics) were carried out in 2000 as part of the National Pollutant Inventory. These studies showed that bushfires are the most significant source of benzene (83%) and total volatile organic compounds (46%). The transport sector produced the greatest proportion of arsenic (85%), chromium IV (67%) and lead (92%), although this sector is small and the amounts were low in comparison to other

<sup>15</sup> jurisdictions. Small proportions are generated by industrial, commercial and household activity. The production of dioxins and other air toxics from bushfires has not been assessed.

Emissions of air toxics from motor vehicles are low in comparison to other States and Territories. The Northern Territory has the youngest vehicle fleet of all jurisdictions, and the Australian Design Rules limiting emissions from new vehicles will continue to have particular benefit.

# LEGISLATION

The *Waste Management and Pollution Control Act* places a general environmental duty on anyone conducting a relevant activity to prevent or minimise pollution or environmental harm. The Act also contains general provisions for licensing and enforcement that can be applied to industry sources of air toxics.

#### RESEARCH

- <sup>30</sup> Research is needed to clarify the sources and levels of risk from air toxics. In particular, assessment of the generation of air toxics in tropical savanna fires would provide better assessment of emissions and indicate fire management strategies to predict and minimise those emissions. Such research is needed to clarify the effect of bushfire timing, frequency, extent, intensity and fuel types on the combustion products and the composition of smoke.
- <sup>35</sup> This will also help to define the exposure risk and health implications from dry season fires.

#### MONITORING AND REPORTING

Additional funds for monitoring and reporting of air toxics are unlikely to be available in the foreseeable future.

40

5

#### **APPENDIX 4 - MEMBERSHIP OF AIR TOXICS NEPM GROUPS**

The membership lists for some groups imply multiple membership for some jurisdictions. In such cases, membership was sequential and the jurisdiction concerned had only one representative at any given time.

10

5

# **PROJECT CHAIR**

Dr Brian Robinson/Mr Robert Joy EPA (Victoria)

#### **PROJECT MANAGER**

Mr Marc Thompson/Mr Haemish Middleton/ Ms Kerry Scott NEPC Service Corporation

#### **PROJECT TEAM**

Ms Sharon Ruffin/Ms Bronwen Harries/ Mr Leo Heiskanen Dr Lyn Denison Dr Michael Dean/Mr Tony Hodgson/ Mr Greg Davies/Mr Roger Bluett Mr Peter Nimmo/Mr Scott McDowell Mr Ziggy Durek/Mr Khokan Bagchi Mr Brian Roderick/Ms Uma Preston/ Mr Rob Mitchell National Health and Medical Research Council EPA (Victoria) NSW EPA

Department of Environment (Queensland) Environment Australia SA EPA

#### **ASSISTED BY**

Mr Haemish Middleton	NEPC Service Corporation
Ms Monina Gilbey	NEPC Service Corporation

#### JURISDICTIONAL REFERENCE NETWORK

Mr David Power	Environment ACT
Mr Greg Davies	NSW EPA
Mr Paul Dworjanyn /Mr Paul Kesby	Environment Australia
Mr Tom Whitworth	SA EPA
Mr Scott McDowell	Department of Environment (QLD)
Dr Bronwyn Burton/Ms Amanda Dawe	EPA (Victoria)
Ms Deanna Tuxford/Ms Sophie	Department of Environment Protection
Wallis/Mr Drew Farrer	(WA)
Dr Frank Carnovale/Ms Lynne Powell	Department Primary Industries, Water and Environment (TAS)
Mr Nigel Green/Mr Paul Lloyd	Department of Infrastructure, Planning and Environment (NT)

#### NON-GOVERNMENT ORGANISATIONS ADVISORY GROUP

Mr Mike Alston Ms Slawka Bell Dr Terry Bellair/Mr David Collins

Dr Jeff Bazelmans Mr Barry Bowden Dr Peter Brotherton

Ms Bronwyn Capanna

Mr Geoff Ereaut

Mr Michael Fagan Mr John Gras Dr Nigel Holmes/Mr Michael Bissell Mr Richard Hoy Ms Pieta-Rae Laut Mr Ewen Macpherson Ms Sharon Makin/Mr John Parsons Mr Lewis Mander Dr James Markos Mr Murray McCafferty/Mr Sean La Fontaine Mr Stewart McDonald Mr John Newton

Ms Elizabeth O'Brien

Mr Paul Orton Mr Matt Ruchel

Mr Bill Saxby Dr Jonathan Streeton Mr Peter Sturrock

Ms Kathryn Turner Mr Andrew Tytherleigh/Mr David Rynne Ms Anne Wilson Mr Bill Yeo Mr John Young/Mr David Grubits/ Ms Linda Perry Mr Mark Chladil The Australian Wood Panels Association **Environment Institute of Australia** Clean Air Society of Australia and New Zealand **Environment Business Australia Engineer Employers Association** National Environment Consultative Forum **Australian Chemical Specialties** Manufacturers' Association Plastics and Chemicals Industry Association Housing Industry Association **CSIRO** Atmospheric Research Minerals Council of Australia Electricity Supply Association of Australia Public Health Association Australian Institute of Petroleum Australian Industry Group Royal Australian Chemical Institute The Australian Lung Foundation Australian Institute of Environmental Health Ford Motor Company of Australia Australian Environment Business Network National Environment Consultative Forum Australian Business Ltd National Environment Consultative Forum Australian Gas Association Australian Medical Association Federal Chamber of Automotive Industries **Cement Industry Federation** Australian Chamber of Commerce and Industry Asthma Australia Australian Home Heating Association Australian Paint Manufacturers' Federation Australasian Fire Authorities Council