Air Pollution Economics

Health Costs of Air Pollution in the Greater Sydney Metropolitan Region

Department of
Environment and Conservation (NSW)

Acknowledgments

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CONTENTS

ABBREVIATIONS

AAQ NEPM	Ambient Air Quality National Environment Protection Measure
BaP	benzo-*a*-pyrene
BTCE/VEPA	Bureau of Transport and Communications Economics/ Victorian Environment Protection Authority
CBD	central business district
CNS	central nervous system
CO	carbon monoxide
COI	cost of illness
COPD	chronic obstructive pulmonary disease
CV	contingent valuation
DEC	Department of Environment and Conservation NSW
E-R	exposure-response
EU	European Union
GDP	gross domestic product
GMR	Greater Sydney Metropolitan Region—Sydney, Illawarra, lower Hunter
GSP	gross state product
IARC	International Agency for Research on Cancer
ICD	International Classification of Diseases
ICD9 460–519	statistical classification of diseases, injuries and causes of death
ICD9 390–459	based on the ICD 9th revision, 1975. The numbers refer to a type of disease. For example: • rheumatic fevers (390–392) • chronic rheumatic heart disease (393–398) • hypertensive disease (401–405) • ischaemic heart disease (410–414) • diseases of pulmonary circulation (415–417) • other forms of heart diseases (420–429) • cerebrovascular disease (430–438) • diseases of arteries, arterioles and capillaries (440–448) • diseases of veins and lymphatics, and other diseases of circulatory system (451–459) • acute respiratory infections (460–466), other diseases of upper respiratory tract (470–478) pneumonia and influenza (480–487) • chronic obstructive pulmonary disease and allied conditions (490–496) • pneumoconioses and other lung diseases due to external agents (500–509) • other diseases of respiratory system (510–519)
MAQS	Metropolitan Air Quality Study
NO_x	oxides of nitrogen
NSW EPA	New South Wales Environment Protection Authority (now DEC)
O_3	ozone
PAH	polycyclic aromatic hydrocarbons

PM	particulate matter
ppb	parts per billion
ppm	parts per million
QALY	quality adjusted life years
RfC	reference concentration
SO_2	sulfur dioxide
URF	unit risk factor
US EPA	United States Environment Protection Agency
VOC	volatile organic compounds
VOLY	value of life years
VOSL	value of statistical life
WTP	willingness to pay
$\mu g/m^3$	micrograms per cubic metre

SUMMARY

Air pollution is a persistent concern in the capital cities of Australia. Continued exposure to high levels of common air pollutants such as ozone (O_3), oxides of nitrogen (NO_x), carbon monoxide (CO) and particulate matter (PM) can result in serious health impacts, including premature death and cardiovascular and respiratory diseases. Those particularly susceptible are the very young, the elderly and those with pre-existing health conditions.

This study estimates the health cost of ambient air pollution in the Greater Sydney Metropolitan Region (GMR), which includes Sydney, Illawarra and the lower Hunter. This information has been prepared to assist decision-making on proposals that have the potential to affect the GMR's air quality.

The total health impact of air pollution can be considered the sum of:

- all independent effects of specific pollutants
- the effects of mixtures, and
- the additional effects (positive or negative) due to interactions between pollutants.

Epidemiological studies usually report the associations between one or more pollutants and health. However, pollutants such as PM, NO_2 (nitrogen dioxide) and CO are often strongly correlated and occur as components of the complex urban air pollution mix. This correlation makes it difficult to accurately determine the independent effects of specific pollutants.[1] Common ambient air pollutants have similar mechanisms and consequences on human health, further complicating the process of allocating the precise role each individual pollutant plays (neutral, additional or synergistic). Therefore, simply summing the specific impact of correlated individual pollutants (as reported by epidemiological studies) could lead to double counting and the overestimation of the total health impact.

The health impacts of a range of air pollutants are evaluated in this paper. However, to avoid double counting, it follows Kunzli *et al*. (1999) in using PM_{10} (particulate matter with an equivalent aerodynamic diameter of 10 µm or less) as the single indicator (the index pollutant) of the health impacts of common ambient air pollutants, and including non-overlapping health endpoints[2] only when calculating the total health impact of air emissions. These non-overlapping health outcomes are total mortality based on long-term exposure, respiratory hospital admissions, cardiovascular hospital admissions, incidence of chronic bronchitis in adults, acute bronchitis in children, restricted activity days in adults, asthma attacks in children and asthma attacks in adults. Not all PM_{10}-related health effects are quantified.

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[1] 'While some studies attempt to identify independent effects, the validity of the results is subject to substantial methodological and analytical limitations. A common approach is to include more than one pollutant in the same regression model, i.e: multipollutant models. However, the high correlations between pollutants often make the results of multipollutant models difficult to interpret. The degree of exposure measurement error for specific pollutants can also influence which of the pollutants is favoured in a multipollutant model.' (Morgan and Jalaludin, 2001, pages 30–31).

[2] A 'health endpoint' is a health effect that occurs as a result of the exposure to pollutants. 'Non-overlapping' means that health statistics are chosen so that they do not measure the same health effect (e.g. one would not count and value both 'pneumonia cases' and 'all cases of respiratory illness' that were attributed to a possible cause, as pneumonia is a subset of respiratory illness, and this would overestimate the impacts).

In this study the health costs of air pollution are estimated using two distinct thresholds. For the base case, the study adopts Kunzli *et al*.'s (1999) approach of estimating the impact of PM_{10} above a baseline of 7.5 µg/m^3. According to Kunzli *et al*. (1999), this threshold reflects the fact that currently available epidemiologic studies have not included populations exposed to levels below 5–10 µg/m^3 (mean 7.5 µg/m^3).

In a variation to this base case, costs are also calculated where the health effects of PM_{10} are estimated without a threshold. This variation provides a sensitivity analysis that shows how specifying a threshold affects total cost estimates.

As acknowledged by Kunzli *et al*. (1999), the approach of using one pollutant as an indicator of the air pollution mix and only estimating the impact of PM_{10} above a baseline will probably underestimate the impact of air pollution. In Sydney, ozone is a pollutant of particular concern, and epidemiological studies suggest that there are ozone health impacts additional to those accounted for in the index pollutant approach[3]. The index pollutant approach also does not account for the additional health effects of air toxics, such as extra cancer cases.

Nevertheless, the Kunzli *et al*. (1999) methodology used by this study is well-respected[4] and produces a conservative estimate of the health cost of Sydney's air pollution mix. The results are at an 'at least to be expected' level.

Estimates of the health costs of air pollution in the GMR are listed in Table S.1 for the base case and the above-mentioned variation to this base case. Results for Sydney, the Illawarra and the Hunter, aggregated to produce the GMR estimates, are presented in Chapter 6.

The high and low cost estimates listed in this study are based on:

- high and low exposure-response estimates for each health endpoint, derived from epidemiological studies showing the relative risk of health impacts from increased exposure to PM_{10} (see section 4.1); and
- high and low cost estimates for each health endpoint (sourced from the NSW Department of Health and willingness to pay estimates from previous studies). Health costs can be valued in terms of risk of premature death, quality of life impacts, health care costs and lost productivity (see section 5).

[3] Kunzli *et al*. (1999) acknowledge that 'In many countries, ozone may be a very important additional air pollution related health problem.'

[4] For instance, the Kunzli *et al*. (1999) methodology was used in a recent report by Fisher *et al*. (2002) to the New Zealand Ministry of Transport on the *Health effects due to motor vehicle air pollution in New Zealand*.

Table S.1: Health cost of air pollution in the GMR

Assumptions	Estimated annual health cost of 2000–2002 mean ambient pollution levels		
	Low	**High**	**Midpoint**
Cost based on PM_{10} indicator with threshold of 7.5 µg/m^3	$1.0 billion	$8.4 billion	$4.7 billion
Cost per capita	$192	$1,594	$893
Cost as percentage of gross state product	0.4%	3.4%	1.9%

Notes:

1. Costs are given in year 2003 dollars.
2. Costs primarily reflect long-term mortality, for which a value of statistical life of $1m to $2.5m is used.
3. Resident population of GMR for study period estimated at 5.27 million.
4. The range of costs shown in Table S.1 is calculated by multiplying low and high estimates of (a) the statistical likelihood of an adverse health outcome per unit increase in air pollution by (b) the economic cost estimated for each health endpoint.

Table S.1 shows that at the average levels of ambient particulate pollution that occurred across the GMR from 2000 to 2002, the total health costs of annual emissions of common ambient air pollutants from all sources in the GMR were conservatively estimated to be between $1 billion and $8.4 billion per annum. This is equivalent to between 0.4% and 3.4% of gross state product.

As discussed in Chapter 5, the cost estimates in this study are generally conservative. The value of statistical life (VOSL) is the main driver of total health costs, and the low and high estimates used for VOSL in this study reflect the lower range of values in the literature. Additionally, several health outcomes have been valued only in terms of cost of illness, which underestimates the total cost of a health outcome. For example, costs such as pain and suffering and loss of leisure are not comprehensively included in the analysis. The US EPA (2000b) reports that the cost of pain and suffering can be many times the cost of treatment.

The information in this report has been developed to provide a better understanding of the costs of air pollution. This information is intended to assist planners and policy makers in the development and consideration of programs and proposals that may affect air quality. For example, the information contained in this report could assist:

- the environmental impact assessment of major public infrastructure and industrial proposals
- valuation of options for transport planning in the implementation of the metropolitan development strategy
- the development and evaluation or review of practical measures or regulatory proposals to reduce pollutant emissions.

1. INTRODUCTION

1.1. Why calculate the health costs of air pollution?

Research over the last 30 years confirms that air pollution causes adverse effects on community health and the environment and imposes a real cost on the community.

Economic theory shows that for resources to be used and distributed efficiently, all costs and benefits of an activity need to be adequately considered. However, in many cases, the costs of air emissions are 'external' to the production and consumption decision-making processes, as they are imposed on the wider community rather than the polluter. The presence of external costs, or negative 'externalities', is a sign of 'market failure', and means that the social cost of an activity is greater than the private cost. In such instances, decision-making is not based on full costs, leading to inefficient use of resources.

Many of the costs associated with motor vehicle use, for instance, are external. Examples include the costs of congestion, and noise, water and air pollution. If users had to pay the full cost of road transport, including external costs, they might choose different forms of transport or decide to travel less.

Economic assessments of policy options must consider all costs and benefits of a proposal, including 'external' effects, such as air emissions. Failure to do so could mean that costs or benefits are significantly underestimated and the analysis is biased.

1.2. Purpose and scope

Given the need to identify and measure the external costs of air pollution, the NSW Department of Environment and Conservation (DEC) has undertaken this project to estimate the health costs of ambient air pollution in the Greater Metropolitan Region (GMR).[5] The primary goal is to provide robust information on the health costs of ambient air pollution to assist decision-making on proposals with the potential to affect Greater Sydney's air quality.

This report considers the health effects of a range of air pollutants, including:

- particulates (PM_{10})[6]
- carbon monoxide (CO)
- sulfur dioxide (SO_2)
- hydrocarbons
- nitrogen dioxide (NO_2)
- ozone (O_3)
- lead (Pb)
- air toxics (benzene and 1,3-butadiene).

[5] Which incorporates the airsheds of Sydney, Wollongong/Illawarra and Newcastle/Hunter, as previously defined by the Metropolitan Air Quality Study—MAQS.

[6] PM_{10} refers to particles with a diameter of 10 µm or less.

The analysis uses PM_{10} as an index pollutant to quantify the health costs of the ambient air pollution mix because, for PM_{10}, 'there exists a broad and sound epidemiological literature to extract effect estimates from' (Kunzli *et al*., 1999).

This project focuses on physical human health impacts from pollutant emissions. It does not provide a comprehensive examination of all impacts that emissions have on flora and fauna, climate, buildings and structures, and tourism.

1.3. Methodology

Calculating the health costs of emissions is a complex task that requires a systematic approach to modelling emissions, human exposure and adverse health costs. Two major tasks that emerged early in the process of this study were:

1. the need to identify appropriate exposure-response relationships for Sydney
2. the need to choose economic methodologies that accurately estimate the economic cost of relevant health endpoints.

DEC engaged two teams of consultants in mid-2001 to assist with these tasks.

The first team, from the Southern Cross Institute for Health Research (Morgan and Jalaludin, 2001), investigated the physical health impacts of air pollution. The team reviewed a large range of international and Australian studies that had applicability to the GMR airshed. From this review, Morgan and Jalaludin (2001) recommended exposure-response estimates for the common air pollutants[7] and air toxics.

A second team of consultants, the Centre for International Economics (CIE, 2001), recommended robust economic valuation methodologies and reviewed available literature on benchmark estimates for air pollution health costs. The results of both of these consultancies are reported in subsequent chapters of this report.

This project relies on the core findings of Morgan and Jalaludin (2001) and CIE (2001), together with recent health cost data and DEC estimates of pollutant loads. It follows Kunzli *et al*.'s (1999) approach of using PM_{10} as the index pollutant to calculate a conservative estimate of the health costs of the air pollution mixture in the GMR and of road transport emissions in Sydney.[8]

[7] Common air pollutants refer to carbon monoxide, nitrogen dioxide, ozone, PM_{10} and sulfur dioxide. This suite of pollutants is also frequently referred to as 'criteria pollutants' according to the Minnesota Pollution Control Agency. 'Criteria pollutants are air pollutants for which the US EPA has established National Ambient Air Quality Standards'.

[8] This approach was found to be appropriate by EPA Victoria, who reviewed a draft of this report.

2. AIR POLLUTION IN THE GMR

This section provides an overview of air quality in the GMR and estimates of the contribution of specific sources to the overall pollutant load.

2.1. Ambient air quality in the GMR

Currently, ambient air quality is usually judged by reference to the Ambient Air Quality National Environment Protection Measure (AAQ NEPM) standards. The AAQ NEPM standards are listed in Table 2.1.

Table 2.1: Ambient air quality NEPM standards and goals

Pollutant	Averaging period	Maximum concentration	Goal within 10 years—maximum allowable exceedences
Carbon monoxide	8 hours	9.0 ppm	1 day a year
Nitrogen dioxide	1 hour	0.12 ppm	1 day a year
	1 year	0.03 ppm	none
Photochemical oxidants (as ozone)	1 hour	0.10 ppm	1 day a year
	4 hours	0.08 ppm	1 day a year
Sulfur dioxide	1 hour	0.20 ppm	1 day a year
	1 day	0.08 ppm	1 day a year
	1 year	0.02 ppm	none
Lead	1 year	0.50 µg/m^3	none
Particles as PM_{10}	1 day	50 µg/m^3	5 days a year

The *NSW State of the Environment Report* 2003 (DEC, 2003) reported that urban air quality in NSW has improved significantly since the 1980s. A recent NSW EPA study (2002) of air toxics also found that most air toxic levels in NSW are low and well below current international standards and benchmarks.[9] However, emissions from industrial and transport activities in the GMR put pressure on maintaining air quality, with ozone (photochemical smog) and particle pollution (brown haze) of most concern in the GMR.

Ozone is formed through the reaction of oxides of nitrogen (NO_x—made up of both nitrogen dioxide and nitrogen oxide) and volatile organic compounds (VOCs—principally hydrocarbons) in the presence of sunlight and is of particular concern during the summer months. As shown in Table 2.1, the AAQ NEPM sets two standards for ozone, a 1-hour standard of 0.10 parts per million (ppm) and a 4-hour standard of 0.08 ppm.[10] Compliance with the AAQ NEPM goal requires that by 2008, the 1-hour and 4-hour standards be exceeded on no more than one day per year. The Sydney region faces a

[9] This report is available at www.environment.nsw.gov.au.

[10] 'These two standards offer similar levels of stringency. The 1-hour standard level is designed to protect the population from peak exposures, while the 4-hour averaging period reflects the potential for exposure during commonly observed ozone episodes. As a result of the interplay of emissions and meteorological conditions, elevated concentrations are generally seen only in daylight hours and during or after the warmest part of the day, and hence tend to be limited to periods of about 4 hours.' (NSW EPA, 2000)

significant challenge in complying with the NEPM goal for ozone. It experiences a number of exceedences of the 1- and 4-hour standards; for example, in the Sydney region in 2002, the 1-hour standard was exceeded on 9 days, and the 4-hour standard was exceeded on 15 days.

It is difficult to detect any clear trends in the number of exceedences of the AAQ NEPM standards for ozone and in annual maximum ozone concentrations in the GMR over the last decade. Peak ozone concentrations and the number of air NEPM exceedences can be partly attributed to variability in meteorological conditions—with hotter, drier weather associated with higher concentrations and a greater number of exceedences.

Although particle levels have declined substantially since the 1970s, particle pollution has shown no discernible trend over the last decade. The Air NEPM 1-day standard for PM_{10} is 50 $\mu g/m^3$ and is not to be exceeded more than five times a year at any one site. The number of allowed exceedences takes into account bushfires and similar natural events and necessary bushfire hazard reduction burning. In 2001, exceedences occurred on 8 days in Sydney and 5 days in both the Lower Hunter and the Illawarra. Bushfires are responsible for most occurrences of high levels of particulate pollution. Occasionally, widespread dust storms can also result in extreme particle levels. Apart from bushfires, hazard reduction burning, domestic wood heating and diesel vehicles are the major sources of particles in urban areas (DEC, 2003).[11]

The GMR experiences considerable variation in pollution levels, depending on seasonal factors and weather. Three other common ambient air pollutants are relevant here:

- *Carbon monoxide* levels have been declining since the early 1980s and have exceeded the NEPM 8-hour standard only a few times since 1995. Most CO pollution comes from motor vehicles in the GMR. The downward trends, in both the concentration of carbon monoxide and the number NEPM exceedences, reflect the introduction of successive emission controls on petrol-fuelled motor vehicles, with over 80% of petrol-fuelled vehicles now having some form of exhaust catalytic control (NSW EPA, 2000). 'Even in the Sydney CBD, where traffic densities are high, recent measurements indicate that carbon monoxide levels are now generally below the air NEPM standard of 9 ppm for an 8-hour average' (DEC, 2003).
- *Nitrogen dioxide*—'Exceedences of the Air NEPM standard of 0.12 ppm for a 1-hour average were commonly observed during the winter months of the early 1980s. Now exceedences are rare and for the last three years the highest value recorded in the Sydney region was 0.08 ppm. Over this period, maximum concentrations of 0.07 and 0.06 ppm were observed in the Illawarra and lower Hunter regions respectively' (DEC, 2003).
- *Sulfur dioxide (SO_2)* levels in Australian cities are generally low owing to the relatively low sulfur content of Australian fossil fuels. The NSW State of the Environment Report 2003 (DEC, 2003) reports that, overall, levels of SO_2 are low in the GMR and below ambient air quality guidelines: 'levels of sulfur dioxide are low with maximum hourly ambient concentrations in the Sydney region less than 25% of

[11] The Air NEPM has recently been amended to require monitoring of $PM_{2.5}$.

the AAQ NEPM standard of 0.20 ppm. Higher levels are observed in the more industrialised Illawarra and lower Hunter regions as a result of the influence of point sources. The GMR recorded no exceedences between 1994 and 2001.'

DEC monitors air pollution on a daily basis in the GMR. The ambient levels of common (or 'criteria') air pollutants averaged out over a recent period of 3 years are presented in Table 2.2.

Table 2.2. Average ambient pollutant levels in the GMR 2000–2002

Pollutant[1]	Region	Minimum	Median	Maximum	Units
Ozone	Sydney	0.0197	0.0470	0.1633	ppm
	Hunter	0.0143	0.0303	0.0790	ppm
	Illawarra	0.0163	0.0313	0.1147	ppm
NO_2	Sydney	0.0050	0.0170	0.0363	ppm
	Hunter	0.0017	0.0103	0.0227	ppm
	Illawarra	0.0013	0.0103	0.0267	ppm
CO	Sydney	0.16	0.83	3.97	ppm
	Hunter	0.07	0.43	3.20	ppm
	Illawarra	0.07	0.47	3.20	ppm
PM_{10}	Sydney	4.0	20.4*	119.6	$\mu g/m^3$
	Hunter	6.2	20.5*	100.3	$\mu g/m^3$
	Illawarra	4.0	19.1*	73.0	$\mu g/m^3$

1. Ozone—1 hour maximum.; NO2—24-hour average; CO—8-hour average; PM_{10}—24-hour average

* Annual average

2.2. Sources of pollution

The contribution of specific sources to total anthropogenic emissions of PM_{10}, NO_x and VOCs are shown in Tables 2.3 to 2.5. These figures are based on the MAQS inventory (Carnovale et al. 1996) updated to 2002 by DEC, accounting for factors such as growth in population, changes in industry mix and energy use, and changes in vehicle kilometres travelled and in vehicle emissions and fuel standards.

Table 2.3: Sources of PM_{10} emissions in GMR region, 2002

Sources	Annual tonnes (and % contribution)		
	Sydney	Wollongong Illawarra	Newcastle Hunter
Domestic fuel combustion	5 982 (23%)	220 (2%)	404 (1%)
Domestic lawn mowing	155 (1%)	8 (<1%)	15 (<1%)
Domestic natural gas combustion	41 (<1%)	0	0
Domestic waste combustion	21 (<1%)	922 (7%)	1 082 (4%)
Other (commercial & small industrial)	3 316 (13%)	324 (2%)	324 (1%)
Industrial facilities and power stations	11 896 (47%)	9 136 (65%)	22 278 (79%)
Motor vehicles	2 318 (9%)	107 (1%)	251 (1%)
Other mobile sources	1 739 (7%)	3 233 (23%)	3 795 (13%)
Total	25 467	13 951	28 149

Source: DEC Atmospheric Science emissions data, 2003.

Table 2.4: Sources of NO_x emissions in GMR region, 2002

Sources	Annual tonnes (and % contribution)		
	Sydney	Wollongong Illawarra	Newcastle Hunter
Domestic fuel combustion	642 (<1%)	23 (<1%)	43 (<1%)
Domestic lawn mowing	76 (<1%)	4 (<1%)	7 (<1%)
Domestic natural gas combustion	381 (<1%)	3 (<1%)	5 (<1%)
Domestic waste combustion	11 (<1%)	365 (2%)	428 (<1%)
Other (commercial & small industrial)	4 122 (4%)	220 (1%)	403 (<1%)
Industrial facilities and power stations	37 622 (35%)	8 126 (53%)	111 408 (90%)
Motor vehicles	62 806 (58%)	3 536 (23%)	7 246 (6%)
Other mobile sources	3 088 (3%)	2 988 (20%)	3 572 (3%)
Total	108 747	15 265	123 112

Source: DEC Atmospheric Science emissions data, 2003.

Table 2.5: Sources of VOC emissions in GMR region, 2002

Sources	Annual tonnes (and % contribution)		
	Sydney	Wollongong Illawarra	Newcastle Hunter
Domestic/commercial	51 591 (41%)	4 479 (50%)	6 998 (42%)
Industrial facilities and power stations	19 511 (15%)	804 (9%)	2 520 (15%)
Motor vehicles	48 632 (38%)	2 564 (29%)	5 705 (34%)
Other mobile sources	6 663 (5%)	1 103 (12%)	1 344 (8%)
Total	126 397	8 950	16 567

Source: DEC Atmospheric Science emissions data, 2003.
Note: Columns do not sum to total owing to rounding.

3. THE HEALTH EFFECTS OF AIR POLLUTANTS

Urban air pollution is a complex mixture of gases, compounds and particles that can have direct adverse impacts on human health. These impacts include respiratory diseases, asthma, heart disease, personal irritations and learning difficulties in children.

This section discusses epidemiological studies and the acute and chronic health effects of the air pollutants considered in this study.

3.1. Epidemiology and health risks

Epidemiology is the study of diseases in human populations. Epidemiological studies can identify correlations between air pollution and human health and characterise the relationship between the level of exposure and the response in the general population (and potentially susceptible segments of the population). Such studies can be used to help determine an acceptable level of exposure or risks.

Three components are required in order to estimate the number of people affected by air pollution in a given population:

- the exposure-response function (e.g. the relative risk)
- the frequency of the health outcome (i.e. the incidence or prevalence)
- the level of exposure.

The relative risk refers to the increase in risk for a specified change in the pollutant measure. The health risk due to a particular pollutant is related to its concentration and the duration of exposure. While the health risks due to air pollutants are generally low, the public health implications are potentially large when millions of residents of major cities are exposed to air pollution.

Epidemiological studies of the health effects of air pollution can be classified as investigating *acute effects* or *chronic effects*:

- Studies of **acute effects** assess relationships between day-to-day changes in mortality and morbidity and same day or previous day(s) air pollution levels. Such studies have found associations between air pollution and deaths, hospital usage, restricted activity days, exacerbations of asthma, minor changes in lung function, and other respiratory and cardiovascular symptoms.
- **Chronic effects** refer to health effects associated with long-term (i.e. repeated) exposure to ambient levels of air pollution. Chronic effects can also include effects such as increased hospital usage or premature death.

The fundamental strength of epidemiology is its ability to evaluate health outcomes in real people, living in normal environments and exposed to typical air pollution. However, limitations and uncertainties are also inherent in such observational studies, owing to potential confounding factors, time considerations in air pollution effects (e.g. lags and latencies), individual variations in air pollution exposure and exposure misclassification.

Air pollution is a complex mixture of many known and unknown substances. The total impact of air pollution on health is the sum of:

- all independent effects of specific pollutants
- the effects of mixtures, and
- the additional effects due to interactions between pollutants (that is, chemical reactions occurring in the air or in the course of inhalation, which may enhance or reduce the effects of individual pollutants (Kunzli *et al*., 1999).

The usual approach of epidemiological studies is to measure the association between at least one specific pollutant (e.g. PM, NO_x, CO or O_3) and health outcomes. These specific components are usually highly correlated with other pollutants and are considered indicative of the complex pollutant mixture. It is unclear how much the associations reported in epidemiological studies represent the independent effects of specific pollutants. This correlation means that simply summing the pollutant-specific impacts could lead to an overestimation of the overall impact of air pollution on health.

Because of the potential to overestimate the impact of air pollution on health, this study selected only one pollutant from the air pollution mix to avoid aggregating the effects of each pollutant separately. A similar approach is taken to other assessments of the health impacts of air pollution in the epidemiological literature (see Kunzli *et al*., 1999). Particulate matter was considered the best single pollutant to use as an 'index pollutant' for an assessment of the health effects of air pollution in the GMR.[12]

This approach is conservative. It produces an 'at least' estimate for the health cost of 'general ambient air pollution'. In addition, the approach does not quantify the additional health costs of ozone—an air quality concern in summer—or air toxics.[13]

3.2. Thresholds

Exposure-response functions may be estimated with and without a threshold. If there is assumed to be a threshold, air pollution levels below the threshold are assumed to have no associated health effects. Often in epidemiological studies no threshold is assumed: any exposure level is assumed to pose a non-zero risk of response to at least a sensitive subgroup of the population.

For many of the pollutants, a threshold exists at the *individual* level. Realistically, most people are not at risk of severe acute health effects at current background levels. However, substantial evidence indicates that there is no threshold at the *population*

[12] For PM_{10}, 'there exists a broad and sound epidemiological literature to extract effect estimates from' (Kunzli *et al.,* 1999).

[13] According to Kunzli *et al*. (1999), 'From a health point of view, "general ambient pollution" (characterised by pollutants such as PM2.5, PM10, total suspended particles, NO_2 and SO_2 and others) may be distinguishable from the additional air quality problem observed in summer only (i.e., oxidant pollution). We decided to estimate the impact for one single indicator of "urban air pollutant"' The impact of oxidant pollution—likely to cause at least in part additional and independent health effects—will not be quantified.'

level. That is, even at low background concentrations, some vulnerable people are exposed to concentrations that adversely affect health.

A United States Environmental Protection Agency (US EPA) assessment of the benefit and costs of the US *Clean Air Act* concluded that there is currently no scientific basis for selecting a threshold for the effects of the major air pollutants (including PM, CO, NO_2, O_3), if a threshold is defined as a level characterised by an absence of observable effects (US EPA, 1999).

Despite evidence that there is no population threshold, policy development often uses thresholds in exposure-response relationships. Even if an original study did not assume a threshold, simply truncating the exposure-response relationship imposes it. Possible threshold points include:

- the non-anthropogenic background pollutant level
- the lowest observed level in the study that estimated the exposure-response relationship
- a pollutant standard.

3.3. Physical effects of air pollution

The discussion below describes the physical effects of key air pollutants on human health. This discussion is mostly based on Morgan and Jalaludin's (2001) review.

Where possible, sensitive subgroups in the population have been identified, including people with existing disease (mainly respiratory and cardiovascular), people with infections such as influenza and pneumonia, asthmatics, the elderly and children.

Exposure-response estimates for the key pollutants are presented in Appendix 3. Risk estimates for benzene, 1,3-butadiene, polycyclic aromatic hydrocarbons (PAHs), toluene and xylene, sourced from the US EPA, the California Environmental Protection Agency, and the World Health Organisation, are also presented in Appendix 3.

Particulate matter (PM)

Particulate air pollution consists of minute solid and liquid particles directly emitted into the air, such as diesel soot, road and agricultural dust, and particles from manufacturing processes. Particles are also produced through photochemical reactions involving pollution gases, such as sulfur and nitrogen oxides, that are a by-product of fuel combustion (Ostro and Chestnut, 1998).

Particles are very diverse in their chemical composition and physical properties. The principal common feature is that they exist as discrete units ranging in size from 0.005 µm to about 100 µm in diameter—although they typically exhibit a bimodal size distribution with a peak in the range 0.1–2.5 µm and a second peak in the range 2.5–50 µm.

In discussing general findings of epidemiological studies conducted around the world, NEPC (1998) reported that:

- studies worldwide have shown that exposure to particulate matter is associated with a range of respiratory symptoms and conditions, as well as increased deaths from respiratory and cardiovascular disease
- there is no evidence that threshold concentrations can be identified for PM_{10} below which it is not possible to detect any population health impacts
- the elderly, children and people with respiratory infections or pre-existing heart or lung disease are particularly susceptible to the effects of particulates.

Statistical evidence suggests that the health effects of particulates can occur independently of the presence of other pollutants, such as ozone, NO_2 and SO_2. There is also increasing evidence that the adverse health effects of particulates are more closely associated with the $PM_{2.5}$ size fraction than with larger fractions (NEPC 1998).[14]

It is not yet clear how exposure to low ambient concentrations of particulates might produce the health effects observed in epidemiological studies and whether certain attributes of particles may be more closely associated to these health effects.

The characteristics of particles that are being investigated for their roles in causing health effects include metal content, particle size, and particles as carriers of other toxic compounds (such as gases or biological toxins from bacteria and pollens etc.).

Transition metals (such as Fe, Cu, Co, Mn) have been hypothesised to be associated with health effects, because they can cause the production of hydroxyl radicals, which are considered toxic to cells. Another hypothesis is that ultra-fine particles are more toxic than larger particles, because they can deposit effectively in the alveolar region and can penetrate the lung epithelium. It is also possible that particles can carry potentially toxic gases or toxins into the deep lung, thus increasing the risk of cellular damage (Health Effects Institute, 1999).

There is ongoing discussion over the most appropriate metric for particulate measurement and for ambient standards. In addition to particle mass, particle number and surface area may be relevant metrics for particulate matter. It is currently unclear whether certain characteristics of particulates are more closely associated with health effects than others, and regulatory action has focused on controlling particulate mass (Health Effects Institute, 1999).

Table 3.1 presents a summary of susceptibilities to various adverse health effects from PM exposure.

[14] There is a distinction in the health effects of different sized particles: particles up to 10 µm (PM_{10}) are inhaled into the airways, whereas larger particles are not; and when inhaled, particles with an aerodynamic diameter of less than 2.5 µm ($PM_{2.5}$) can penetrate deep into the lungs.

Table 3.1: Summary of adverse health effects from PM exposure and susceptible populations

Health effects	Susceptible groups	Notes
Acute (short-term) exposure		
Mortality	Elderly, infants, persons with chronic cardiopulmonary disease, influenza or asthma	How much life shortening is involved and how much is due to short-term mortality displacement is uncertain.
Hospitalisation / other health care visit	Elderly, infants, persons with chronic cardiopulmonary disease, pneumonia, influenza or asthma	Reflects substantive health impacts in terms of illness, discomfort, treatment costs, work or school time lost, etc.
Increased respiratory symptoms	Most consistently observed in people with asthma, and children	Mostly transient with minimal overall health consequences, although for a few there may be short-term absence from work or school due to illness.
Decreased lung function	Observed in both children and adults	For most, effects seem to be small and transient. For a few, lung function losses may be clinically relevant.
Chronic (long-term) exposure		
Increased mortality rates, reduced survival times, chronic cardiopulmonary disease, reduced lung function, lung cancer	Observed in broad-based cohorts or samples of adults and children (including infants). All chronically exposed are potentially affected	Long-term repeated exposure appears to increase the risk of cardiopulmonary disease and mortality. May result in lower lung function. Average loss of life expectancy in highly polluted cities may be as much as a few years.

Source: Adapted from Pope (2000) and Pope *et al.* (2002).

Ozone (O_3)

Ozone is an unstable blue gas with a pungent odour. Ozone molecules consist of three oxygen atoms. It is found both near the ground (troposphere) and in the stratosphere. Ozone formation and destruction occur naturally, particularly in the stratosphere, where the 'ozone layer' is found. As well as occurring naturally in the atmosphere, ozone can also form at ground level as a secondary pollutant formed by the reaction of oxides of nitrogen and VOCs in the presence of sunlight.[15] Ground-level ozone is a major constituent of photochemical smog.

[15] Non-methane hydrocarbons is a synonym for reactive organic compounds. This class of compounds is sometimes referred to as 'hydrocarbons'.

Ozone is a highly irritating gas that affects the respiratory tract. In experimental studies in humans, ozone toxicity occurs as a continuum in which higher concentrations, longer exposure duration and greater activity levels during exposure lead to greater adverse effects. Short-term acute effects include respiratory symptoms, increased respiratory rate, pulmonary function changes, increased airway hyper-responsiveness and increased airway inflammation (Morgan and Jalaludin, 2001). Epidemiological studies have also demonstrated adverse health effects, including decreases in lung function, increases in respiratory symptoms, increased emergency department attendances, increases in hospitalisations and increases in mortality. As many of the adverse health effects are observed both with exposures to ambient ozone (and co-pollutants) and in controlled experimental exposures (to ozone alone), it appears that the functional and symptomatic responses can be attributed primarily to ozone (Morgan and Jalaludin, 2001).

Table 3.2 lists the susceptibility of population subgroups to the health effects of ozone exposure.

Table 3.2: Susceptibility of population subgroups to health effects of ozone exposure

Group	Effect of ozone exposure
Smokers	The available evidence indicates that smokers may be less sensitive at concentrations of ozone equivalent to environmental exposure. The studies have been done on very few subjects only and their overall significance is open to question.
Age cohorts	Older people (over 40 years) may be less susceptible than young adults (18–26 years) when exposed to ozone in experimental chambers. Children are generally exposed to lower levels in chamber studies and therefore results are inconclusive. In Australian epidemiological studies, the population at highest risk for hospital admissions was children 0 to 14 years old, while for mortality it was adults over 65.
Asthmatics	In chamber studies, asthmatics are not more susceptible to ozone, but Australian epidemiological studies indicate that risk for hospitalisation is greater for asthmatics than for non-asthmatics.
Cardiorespiratory disease sufferers	Hospitalisations for cardiovascular or respiratory conditions have been found to be associated with high ozone concentrations in Australian epidemiological studies. It is therefore likely that people with these pre-existing conditions are more at risk.
Allergic rhinitis sufferers	Results for subjects with a history of allergic rhinitis are inconclusive.
Persons undertaking heavy exercise	Heavy exercise increases the inhibitory effects of ozone on respiratory function.

Nitrogen dioxide (NO_2)

NO_2 is a product of combustion. It is a precursor to ground-level ozone formation through photochemical reactions involving VOCs. NO_2 causes a brown colour in the atmosphere at elevated concentrations. It reacts in the atmosphere with ammonia to form fine particulates, which reduce visibility and increase $PM_{2.5}$ concentrations (Levelton Engineering Limited, 2000).

NO_2 irritates the mucous membranes in the respiratory tract. It impairs lung immunity mechanisms, increasing susceptibility to respiratory infections, especially in children and asthmatics, and reduces lung function at high levels. Asthmatics exposed either simultaneously or sequentially to NO_2 and an aeroallergen have an increased risk of an exaggerated response to the allergen (WHO, 2000). NO_2 enhances the effects of exposure to other known irritants, such as ozone, SO_2 and particulates.

Epidemiological studies indicate that NO_2 may increase respiratory illness in older children (5–15 years). These findings are of concern because of the potential long-term effects. Studies indicate that repeated respiratory illness in older children (independent of NO_2) is associated with increased lung damage in later life. Thus, any increases in such illnesses associated with NO_2 could have subsequent, as well as immediate, consequences (WHO, 2000).

The NO_2 provisions of the ambient air NEPM are based on evidence that important health effects of NO_2 occur above a threshold, and incorporate a safety factor accordingly (NEPC, 1998).

Asthmatics are likely to be the most sensitive subjects to NO_2 exposure. Health effects of NO_2 exposure in this group include decreased pulmonary function and increased bronchial reactivity.[16] The steady increase over time in the number of asthmatics in many countries enhances concerns about asthma. The mild asthmatics chosen for the controlled exposure studies do not represent all asthmatics, and there are likely to be some individuals with greater sensitivity to NO_2.

People with other pre-existing respiratory disease are also particularly susceptible to NO_2 exposure. Health effects for those in this group who are exposed to near-ambient concentrations of NO_2 include mild changes in airway responsiveness and in pulmonary function (US EPA, 1997).

Some mainly European epidemiological studies have shown associations between NO_2 and daily mortality and hospital admissions, although the results have been mixed (WHO, 2000). These conflicting results may reflect interactions between NO_2 and other pollutants (NEPC, 1998).

[16] Studies in asthmatics exposed to 0.3 to 0.6 ppm NO_2 indicate a change of about 5% in pulmonary function and increased airway responsiveness to bronchoconstrictors. Asthmatics have a higher baseline airway responsiveness, and NO_2-induced increases in airway responsiveness are expected to have clinical implications for exaggerated responses to a variety of provocative agents, such as cold air, allergies and exercise.

A recent review concluded that there is still insufficient evidence from epidemiological studies alone to establish whether the observed effects are causally related to NO_2 (Ackerman-Liebrich & Rapp, 1999). The review concluded that, combining all the evidence, it seems reasonable to assume that NO_2 is at least partially responsible for the observed effects of air pollution on health.

Carbon monoxide (CO)

Carbon monoxide is a clear, odourless gas produced by the incomplete combustion of organic compounds. It reduces the blood's capacity to carry oxygen. CO combines selectively with haemoglobin (the oxygen transport protein in red blood cells) to form carboxyhaemoglobin. CO impairs perception and judgment at low levels. Effects worsen as CO levels rise, leading ultimately to convulsions and coma at high concentrations. The CO provisions of the ambient air NEPM are based on evidence that a carboxyhaemoglobin threshold of 2.5% should not be exceeded, and incorporates a safety factor (NEPC, 1998).

Until recently, it was thought that current CO exposure levels were unlikely to produce serious health effects. However, recent studies have observed increases in daily mortality and hospital admissions for cardiovascular disease at levels below current ambient CO air quality standards (NEPC, 2000). It is possible that associations shown in these recent studies may be due to CO acting as an indicator of other pollutants, perhaps fine particles (Schwartz, 1995).

CO also participates in photochemical smog reactions, leading to increased ground-level ozone.

Experimental studies show that small changes in CO concentration aggravate angina. It is presumed that people with pre-existing ischaemic heart disease (coronary heart disease) are especially sensitive to interference with their oxygen supply and, hence, are a particularly susceptible subgroup. Other susceptible subgroups include pregnant women, fetuses and newborns.

Sulfur dioxide (SO_2)

SO_2 levels in Australian cities are generally low owing to the relatively low sulfur content of Australian fossil fuels, and rarely approach the current air NEPM standards. However, the potential impacts of SO_2 do mean that it is a pollutant of concern.

Exposure to ambient levels of SO_2 has been associated with reduced lung function, increased incidence of respiratory symptoms and diseases, irritation of the eyes, nose and throat, and premature mortality. Children, the elderly and those suffering respiratory ailments are particularly susceptible. Health impacts appear to be linked mostly to brief exposures to ambient concentrations above 1000 $\mu g/m^3$. However, some

epidemiological studies have shown an association between relatively low annual mean levels and excess mortality (World Bank Group, 1999).

In many instances, it is difficult to separate the adverse effects resulting from exposure to SO_2 from those resulting from concurrent exposure to mixtures including other known irritants pollutants such as ozone, NO_2 and, in particular, PM (NEPC, 1998).[17] However, results from controlled exposure studies support the epidemiological findings of exacerbation of asthma, increases in respiratory symptoms and decreases in lung function (NEPC, 1998).

Lead (Pb)

Lead is a soft bluish or silvery grey metal, which is naturally present in low concentrations in the Earth's crust.

Lead can be a very toxic element and is associated with a variety of health effects. According to the US EPA (1994):

> 'Brain damage, kidney damage, and gastrointestinal distress are seen from acute (short-term) exposure to high levels of lead in humans. Chronic (long-term) exposure to lead results in effects on the blood, central nervous system (CNS), blood pressure, kidneys, and Vitamin D metabolism. Children are particularly sensitive to the chronic effects of lead, with slowed cognitive development, reduced growth and other effects reported. Reproductive effects, such as decreased sperm count in men and spontaneous abortions in women, have been associated with high lead exposure. The developing fetus is at particular risk from maternal lead exposure, with low birth weight and slowed postnatal neurobehavioral development noted.'

The largest source of atmospheric lead has been the combustion of leaded petrol. Unleaded petrol was introduced into NSW in 1986, and lead in petrol has since been phased out. As a result, lead levels in the air have fallen dramatically, and recent research is showing a decline in lead in the blood of children in Sydney's urban areas to low levels. Since 1 January 2002, Commonwealth legislation has effectively banned the use of leaded petrol.

Air toxics

'Air toxics' is a general term referring to a broad range of pollutants that are believed to be highly toxic and pose significant health risks at low concentration levels. Air toxics exist at relatively low concentrations in urban airsheds, with significantly elevated levels occurring only near specific sources such as roads subject to heavy traffic, industrial sites and areas affected by wood smoke.

[17] SO_2 reacts with other substances in the atmosphere to form sulfate aerosols. Since most sulfate aerosols are part of $PM_{2.5}$, they may have an important role to play in the health impacts associated with PM (World Bank Group, 1999).

For example, a recent NSW EPA (2002) study of air toxics in the GMR found that 'most air toxic levels in NSW are low and well below current international standards and benchmarks.' This study also noted that concentrations of benzene and 1,3-butadiene at some sites, such as the Sydney central business district (CBD), are 'present at concentrations which approached, but did not exceed, the goals for these substances', and concentrations of PAHs in the Sydney CBD are 'in the vicinity of' proposed international goals for these pollutants (NSW EPA, 2002).

The health effects of air toxics, as discussed below, have been observed primarily in occupational and animal studies. Therefore, while there is considerable information about the toxicity and risk posed by the more common air toxics at the elevated concentrations found historically in some workplaces, much less is known about the potential risks of ongoing exposure to the low levels that occur in the general environment.

Benzene

Benzene is a colourless, liquid, flammable, aromatic hydrocarbon that is a component of petrol, or may result from incomplete combustion of fuels.

Benzene, a natural component of crude oil, is emitted from a range of industrial and combustion sources. The major source of benzene is motor vehicles—both vehicle exhaust (contributing approximately 75% to 80% of emissions) and evaporative emissions (including evaporation losses from motor vehicles and evaporation losses during the handling, distribution and storage of petrol).

Fuel quality standards imposing a benzene limit of 1% in petrol sold by 2006 should reduce benzene concentrations in the GMR significantly (petrol sold in NSW currently contains about 3% benzene).

Benzene is naturally broken down by chemical reactions within the atmosphere. The length of time that benzene vapour remains in the air varies between a few hours and a few days, depending on environmental factors, weather and the concentration of other chemicals in the air, such as nitrogen and sulfur dioxide.

Inhalation is the dominant pathway for benzene exposure in humans. Smoking is a large source of personal exposure. It is reported from various countries that extended travel in motorcars produces exposures that are second only to smoking as contributors to the intensity of overall exposure.

Current understanding of health effects of benzene is derived mainly from animal studies and human health studies in the occupational setting. Acute effects of benzene include skin and eye irritations, drowsiness, dizziness, headaches and vomiting. However, it is thought that at levels occurring in the ambient atmosphere, benzene does not have short-term or acute effects. The mechanisms of benzene toxicity are not well understood.

Health effects of chronic benzene exposure include:

- CNS depression
- chromosomal aberrations
- bone marrow toxicity (pancytopaenia)
- leukaemia (especially non-lymphocytic or myeloid)
- diminished immune function.

Benzene is carcinogenic, and long-term exposure can affect blood production and harm the immune system. It can cause cancers and leukaemia in laboratory animals and human populations exposed for long periods, and has been linked to birth defects in animals and humans (Environment Australia, 2001). The International Agency for Research on Cancer (IARC) has classified benzene in Group 1: 'human carcinogen' on the basis of sufficient evidence in humans (IARC, 1987).

All members of the population are susceptible to the adverse health effects of benzene. In addition, epidemiological studies cannot establish a threshold below which exposure to benzene is not linked to increased risk of adverse health effects.

Adverse health effects of both acute and chronic exposures to benzene have been documented. However, for the derivation of exposure-response functions, the main assessed health endpoint is leukaemia. Exposure-response estimates are not available for endpoints other than leukaemia.

1,3-Butadiene

1,3-Butadiene is a colourless gas, and is a major product of the petrochemical industry, used in the manufacture of synthetic rubber, latex paints and nylon.

The major source of 1,3-butadiene is incomplete combustion of petrol and diesel fuel. It is emitted from industrial facilities, tobacco smoke and motor vehicles. Workers in industries that produce 1,3-butadiene or who are exposed to motor vehicle exhaust are at risk of exposure. 1,3-Butadiene is highly reactive and can oxidise to form formaldehyde and acrolein, two toxic substances in their own right. The probable route of human exposure to 1,3-butadiene is through inhalation.

Exposure to 1,3-butadiene can irritate the eyes, nose and throat. Acute exposure to 1,3-butadiene can cause CNS damage, blurred vision, nausea, fatigue, headache, decreased pulse rate and pressure, and unconsciousness. Chronic exposure to lower levels can increase heart and lung damage. Although the human data (based on only a few occupational studies) is limited, there is sufficient animal data to suggest that 1,3-butadiene is a probable human carcinogen (US EPA, 2001).[18]

[18] The US EPA classified 1,3 butadiene in Group B2: probable human carcinogen (USEPA, 2001). IARC classifies 1,3 butadiene as a probable human carcinogen (IARC, 1987). The recent WHO revision of air quality guidelines concluded that 1,3 butadiene is probably carcinogenic to humans (Group 2A) (WHO, 2000).

Polycyclic aromatic hydrocarbons (PAHs)

PAHs are a group of several hundred organic chemicals made of only hydrogen and carbon and containing at least two fused aromatic rings (benzo-*a*-pyrene [BaP] is probably the most well known PAH). PAHs are a major component of polycyclic organic matter. Polycyclic organic matter is divided into PAH and PAH derivatives. PAH derivatives contain other elements in addition to carbon and hydrogen (e.g. nitro-PAH, amino-PAH and oxygenated-PAH).

PAHs are formed mainly as a result of pyrolytic processes, especially the incomplete combustion of organic materials during human activities such as processing of coal and crude oil, combustion of natural gas, combustion of refuse, vehicle traffic and tobacco smoke, and natural processes such as carbonisation (WHO, 2000). Occupational exposure to PAHs can occur in petroleum manufacture and use, or where coal, wood and other plant materials are burned (Environment Australia, 2001). In the air, PAHs are generally found attached to particulate matter.

Data from animal studies indicate that several PAHs may induce a number of adverse health effects, including immunotoxicity, genotoxicity, carcinogenicity and reproductive toxicity. BaP is the most intensively studied PAH in animals. BaP is the only PAH that has been tested for carcinogenicity following inhalation, and it produces lung cancer in animals. The lung carcinogenicity of BaP is enhanced by co-exposure to other substances, such as cigarette smoke and airborne particulates (WHO, 2000). Results from epidemiological studies indicate that an increase in lung cancer occurs in humans exposed to coke oven emissions, roofing tar emissions and cigarette smoke. Each of these contains a number of PAHs (CARB, 2000).

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 air is desirable. BaP has been suggested as the most appropriate indicator (WHO, 2000).

Toluene

Toluene is a colourless aromatic liquid derived from coal tar or from petroleum refining. Toluene is widespread in the environment owing to its use in a variety of commercial and household products. It is also found in tobacco smoke.

Air pollution from motor vehicles is a major source of exposure to toluene (WHO, 2000). Toluene is also emitted during crude petroleum and natural gas extraction, and in petroleum refining. Workers in industries exposed to motor vehicle exhaust are at greatest risk of exposure.

The CNS is the primary target for toluene toxicity in both animals and humans, for acute and chronic exposure. CNS dysfunction (often reversible) and narcosis occur in humans exposed to low or moderate levels of toluene (US EPA, 2001). Short-term exposure to high levels of toluene can result in light-headedness and euphoria (Environment

Australia, 2001). CNS depression has been shown to occur in chronic glue or paint abusers exposed to high levels. Symptoms include cerebral atrophy and impaired speech hearing and vision. Irritation of the upper respiratory tract is associated with chronic inhalation. Toluene does not appear to be carcinogenic (US EPA, 2001).

Xylene

Xylene is one of the family of isomeric, colourless, aromatic hydrocarbon liquids produced by the destructive distillation of coal and by refining of petroleum. It is used for high-octane and aviation gasoline, solvents, chemical intermediates, and the manufacture of polyester resins. Xylene is emitted during petroleum refining and solid fuel combustion, and is a component of vehicle exhaust. It is also a common component in many domestic products.

Acute exposure to xylene results in irritation of the respiratory tract, transient eye irritation and neurological effects. Chronic inhalation exposure results in CNS effects such as headaches, dizziness, fatigue, tremors and poor co-ordination. Other effects of chronic exposure include impaired pulmonary function and possible affects on the blood and kidneys (US EPA, 2001). The evidence of developmental or reproductive effects on humans in inconclusive. Xylene does not appear to be carcinogenic (US EPA, 2001).

3.4. Summary of health impacts

Health endpoints associated with selected air pollutants

Particulates	Nitrogen dioxide	Carbon monoxide	Ozone	Air toxics	Air toxics (PAHs)
• Increase in cardiac and respiratory mortality • Admissions to respiratory and cardiovascular casualty room and hospital • Increased incidence of acute bronchitis in adults and children. Increased prevalence and exacerbations of COPD in adults and children • Asthma attacks in adults and children • Cough • Restricted activity days • Reduced lung function	• Increased mortality • Impaired lung function • Impaired respiratory defence mechanisms, leading to increased susceptibility to infections • Increased respiratory disease in children	• Mortality, especially those with cardiovascular disease • Aggravation of cardiovascular disease & chest pain • Nausea • Headache • Fatigue	• Mortality • Acute respiratory problems • Chest constriction & pain • Increase in incidence and severity of asthma attacks • Increase in asthma and respiratory-related casualty room visits and hospitalisations • Coughing and wheezing • Eye irritation • Headache	**Benzene:** • Leukaemia • Long-term harm to immune system • Skin and eye irritations • Drowsiness • Dizziness • Headaches **Toluene:** • CNS dysfunction (often reversible) • Narcosis • Light-headedness **Xylene:** • Irritation of respiratory tract • Eye irritation • Headaches, dizziness, fatigue, tremors, coordination difficulties • Impaired pulmonary function **1,3-butadiene:** • Cancer • Eye, nose, throat irritation	• Cancer • Kidney & liver damage • Respiratory irritation • Exacerbation of asthma • Chronic bronchitis • Coughing & throat irritation

4. EXPOSURE-RESPONSE ESTIMATES FOR THE GMR

This section identifies exposure-response functions for PM_{10} and selected health endpoints in the GMR (exposure-response functions for other pollutants are listed in Appendix 3), and includes discussion of the uncertainty associated with quantifying health effects.

4.1. Exposure-response estimates for the GMR

This study is based on 'low' and 'high' PM_{10} exposure-response estimates in the GMR. These estimates were derived by Morgan and Jalaludin (2001) following a survey of peer-reviewed epidemiological studies. They are summarised in Table 4.1.

Morgan and Jalaludin's (2001) literature review was completed in September 2001. Therefore, only studies published before this date were considered in this report. More recent studies might show small differences in results, but there is no strong evidence that the results would fundamentally change the conclusions from the collective body of epidemiological literature.[19]

Meta-analysis

For each health outcome, Morgan and Jalaludin (2001) selected peer-reviewed studies from the international literature to derive a joint estimate and 95% confidence interval. These values represent the central, low and high estimates listed in Table 4.1 and in Appendix 2.

In deriving these exposure-response estimates, Morgan and Jalaludin (2001) used the approach of Kunzli *et al*. (1999) of using meta-analyses. That is, where available, meta-analytic estimates identified in the literature were used. If no such estimates could be found, Morgan and Jalaludin calculated their own combined estimates 'by variance weighted methods using either a fixed effect or random effects model.'[20]

Because uncertainty decreases as sample size increases, combining results from several studies may yield more reliable estimates of relative risk. Rather than estimates from one single study, meta-analytic point estimates can enhance the value of the available information and deal with heterogeneity between studies.

[19] Since Morgan and Jalaludin's (2001) literature review, several important epidemiological studies have been reanalysed. There has been controversy about the statistical methodology used in the time-series analysis of the acute effects of air pollution on daily mortality, resulting in a revision of the dose-response estimates. However, the revision does not affect the results of this study. This study estimates health costs using cohort studies of the effects of long-term exposure on mortality, rather than the controversial acute mortality studies. In March 2002, a reanalysis of one of the cohort studies used in this study to determine the effects of particulate pollution on mortality was published (Pope *et al.,* 2002). The results of this reanalysis are similar to the original analysis (Pope *et al.,* 1995), and their inclusion would not meaningfully change the conclusions of this report.

[20] This is a standard approach recently used by the WHO (2000) to calculate combined effects estimates for a range of health endpoints.

Uncertainty ranking scheme

To represent the inherent epidemiological uncertainties in the exposure-response estimates derived from meta-analyses, Morgan and Jalaludin (2001) developed a qualitative uncertainty ranking scheme. This scheme classifies the estimates as:

- 'A' (a very good level of certainty)—exposure-response estimates derived from joint estimates of several well-conducted studies.[21]
- 'B' (a good level of certainty)—exposure-response estimates derived from joint estimates of more than one well-conducted study.
- 'C' (a sufficient level of certainty)—exposure-response estimates derived from one well-conducted study.

Estimates derived from cohort studies were treated separately, owing to the 'high quality of data from such studies' (Morgan and Jalaludin, 2001).

'Generalisability' of exposure-response relationships

In reviewing the extent to which overseas exposure-response data can be generalised to Australia[22], NEPC (2000) concluded:

> 'Most information on the health effects of particles comes from epidemiological studies in the US. However, in recent years, there has been significant research conducted elsewhere, particularly in Europe. These European studies, while finding associations, can differ from the US studies in the strength of the association and the size of the effect estimates. Australian studies show associations between particles and daily mortality, hospital admissions and respiratory symptoms. However, Australian studies are currently insufficient to reliably establish specific Australian dose-response relationships. Therefore, overseas data must be used in health risk assessment and Australian studies [must be] used to support the overseas studies, while clearly identifying the uncertainties associated with using overseas data.'

To minimise the uncertainty associated with the application of exposure-response relationships derived from overseas studies to the GMR, Morgan and Jalaludin (2001):

- selected 'well conducted' epidemiological studies, which have been used in previous health impact assessments

[21] Morgan and Jalaludin (2001) used exposure-response estimates only from studies that satisfied the following criteria:

- peer-reviewed literature
- an adequate study design
- ecological studies excluded (ecological studies in this context refers to studies that do not, or cannot, control for major confounders)
- identified PM levels
- a reasonably complete analysis
- an exposure response function (or effect estimate) that expresses the relative increase in a selected health outcome for a given increment in air pollution, with 95% confidence limits.

Cross-sectional or cohort studies with exposure categorised into two or three levels are excluded (two or three exposure levels are considered too few for an epidemiologically robust assessment of exposure-response relationships, owing to the potential for uncontrolled confounding).

[22] For the purposes of this report, we refer to this as 'generalisability'.

- selected exposure-response relationships derived from studies conducted in more than one region (i.e., North America, Europe and Australia). Preference was given to studies conducted in North America and Western Europe, as the conditions at these study sites are generally more in line with the levels of exposure and the underlying population health status of people in NSW than studies conducted in other regions
- gave extra weight to exposure-response relationships with supporting evidence from Australian epidemiological studies. Morgan and Jalaludin took the view that an integrated exposure-response estimate from 'well conducted' studies in a selection of appropriate locations is likely to be closer to the 'true' exposure-response estimate than the results of one epidemiological study in the GMR.

The exposure-response relationships used in this study were derived from studies conducted in areas subject to a wide range of pollution levels. Pollution levels in Sydney fall at the lower end of this range. Under a linear exposure-response relationship (as is assumed by Morgan and Jalaludin, 2001), the health impacts and associated health costs at the lower range of exposure will be proportional to those throughout the range of exposure. However, there is always greater uncertainty surrounding estimates derived from data that fall at the lower or upper end of any exposure-response relationship.

From the above criteria, Morgan and Jalaludin (2001) developed a qualitative ranking scheme to indicate the strength of evidence supporting the generalisability of identified exposure-response estimates for the GMR. Three criteria were used to classify the generalisability of exposure-response estimates:

- +++, strong evidence of generalisability—exposure-response estimate derived from at least one study in more than one region, including supporting Australian data
- ++, good evidence of generalisability—exposure-response estimate derived from at least one study in more than one region
- +, sufficient evidence of generalisability—exposure-response estimate based on at least one study.

Again, exposure-response estimates derived from cohort studies (e.g. studies of the long-term effects of PM on mortality) are considered separately, 'due to the methodological advantages of such studies and the resources required to replicate some of these studies' (Morgan and Jalaludin, 2001).

Recommended PM_{10} exposure-response estimates for the GMR

The generalisability scheme can be combined with the uncertainty-ranking scheme to give a qualitative assessment of the confidence in the accuracy of an exposure-response estimate and the generalisability of this estimate to the GMR. For instance, the exposure-response estimate for respiratory hospital admissions for PM_{10} has a very good level of certainty (A) and strong evidence of generalisability (+++).

Table 4.1: Recommended PM_{10} exposure-response estimates for the GMR

Health endpoint	Average exposure-response estimates for 10 µg/m^3 change in PM_{10}	Uncertainty rank	Generalisability rank
Mortality (long-term)	low: 2.6% central: 4.3% high: 6.1%	B	++
Respiratory hospital admissions	low: 0.5% central: 0.8% high: 1.1%	A	+++
Cardiovascular hospital admissions	low: 0.6% central: 0.9% high: 1.3%	A	+++
Asthma attacks (<15 years)	low: 2.7% central: 4.4% high: 6.2%	A	++
Asthma attacks (>15 years)	low: 0.0% central: 0.4% high: 0.8%	A	++
Restricted activity days	low: 7.9% central: 9.4% high: 10.9%	C	+
Acute bronchitis (<15 years)	low: 13.5% central: 30.6% high: 50.2%	A	+++
Chronic bronchitis	low: 0.9% central: 9.8% high: 19.4%	B	+

4.2. Uncertainty in quantifying and applying exposure-response estimates

Epidemiological studies have some inherent uncertainties. Accurate estimation of both population exposure and response to a pollutant can be difficult, particularly as many pollutants occur as components of complex mixtures, and the extent of impact can depend on time considerations (e.g. lags and latencies) and variation in exposure. There are also potential confounding factors such as environmental agents or characteristics other than the pollutant of concern (e.g. cigarette smoking) and factors that influence the susceptibility of subjects (e.g. health status, medication).

The allocation of health impacts to specific sources adds to the uncertainty. Source-specific measures of pollutants have not been used in epidemiological studies, and there is little information providing direct estimates of the health impact of specific sources, such as traffic-related pollution. Although air pollution is associated with various health outcomes, it is not clear whether the exposure-response function related to traffic pollution, for example, differs from non-traffic sources.

The estimation of exposure-response functions is based on pollutant levels within a certain range in the location of a particular study. The fit between the estimated function and the pollutant data may become less certain towards the end of the pollutant data range, and the function is not normally extrapolated beyond the range of the data.

The uncertainty associated with the application of exposure-response functions from epidemiological studies is highlighted by the fact that results can vary both between and within countries. Potential sources of uncertainty associated with applying the results of epidemiological studies include:

- different methods of assessing exposure in epidemiological studies (that is, different approaches to assessing the spatial and temporal distributions of air pollutants by using fixed-site monitors)
- the variation of pollutant mixes and characteristics in different study areas—for example, differences in particulate effects between regions may be due to differences in local particulate size distributions, the chemical nature of particles and seasonal differences (Katsouyanni *et al.*, 1997)
- different methods of measuring health outcomes (differences in health care systems, lack of standardised health monitoring systems for morbidity)
- varying population characteristics in different study areas—that is, the extent to which cities have a larger or smaller proportion of people in highly susceptible subgroups.

5. HOW ARE HEALTH COSTS VALUED?

Health impacts give rise to a range of costs borne by both the individual and the wider community. Chapter 3 outlines a broad range of health endpoints that can be associated with each of the identified pollutants, and Chapter 4 identifies the health endpoints that can be quantified for the GMR.

This chapter identifies values that can be used to estimate the health costs associated with air pollution (based on use of PM_{10} as the index pollutant). The chapter begins with an overview of valuation methodologies. It reviews the appropriateness of methodologies and discusses the uncertainties surrounding these cost estimates. It concludes by discussing the estimates used in this study to value the cost of each health outcome and the implications of the adopted valuation methodology for the study's results. Chapter 6 outlines the potential problem of double counting and how it can be best avoided.

This chapter summarises CIE's (2001) report on *Economic Valuation Methodologies*.

5.1. Valuing health endpoints

The health endpoints assessed in this study fall into two general classes—mortality and morbidity. Mortality effects are those associated with changes in risk of premature mortality (death). Morbidity (illness) effects are associated with changes in the risk of acute and chronic cardiopulmonary and respiratory effects and illnesses.

Mortality and morbidity effects impose a range of costs on individuals. These costs include direct costs, such as payments for diagnosis and treatment, and indirect costs, such as lost productivity. In addition, adverse health effects can also impose costs that are less easily monetised, such as pain and suffering to individuals and their families.

Researchers have used a number of approaches to value these costs. They can be grouped into two broad approaches:

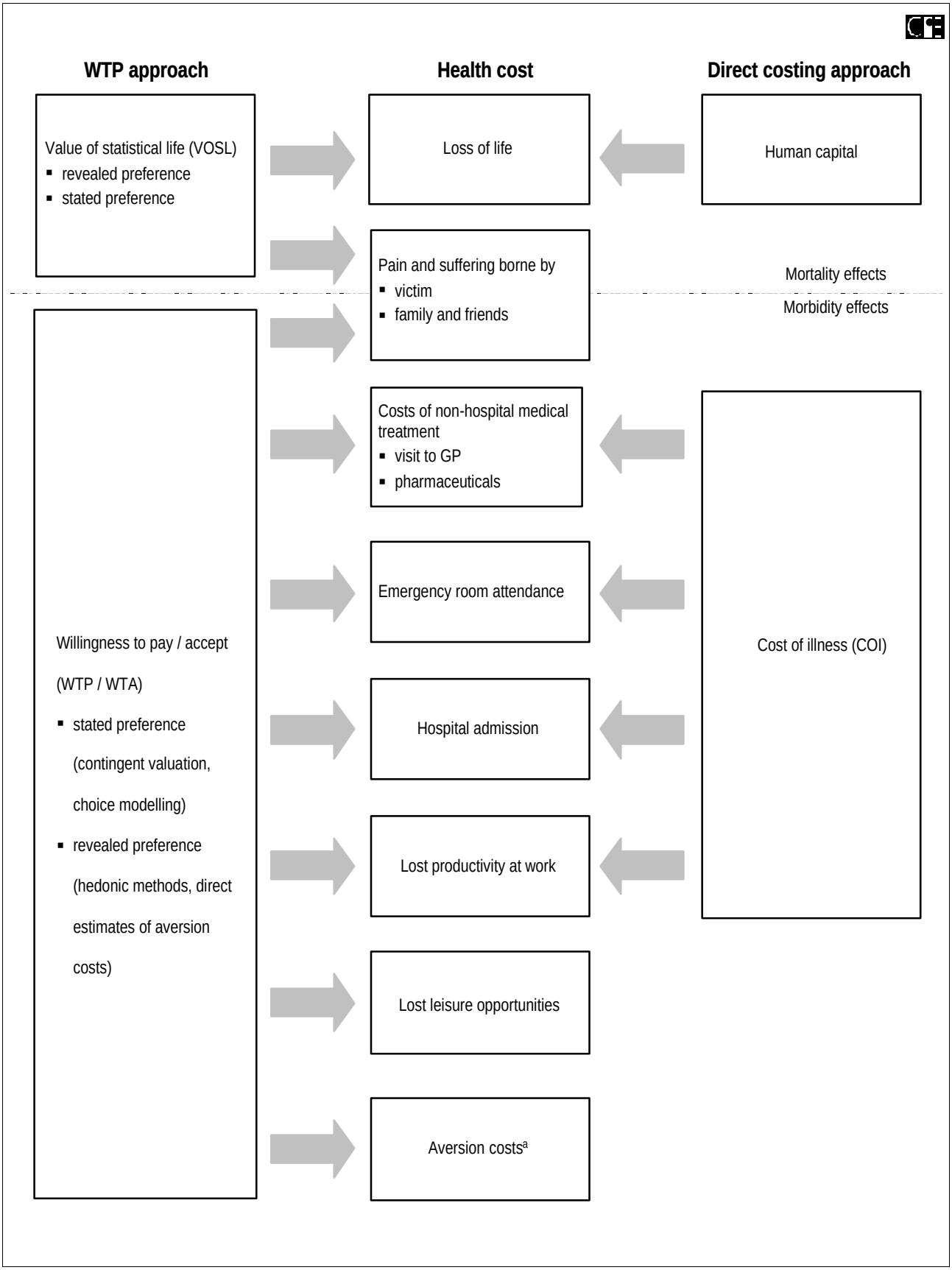
- **Cost of illness (COI)** or direct costing approaches use available information from existing markets to estimate actual costs generated by an illness. These methods require the presence of markets and market prices (or input costs) in order to value cost elements. In general, a COI estimate reflects expenditures on diagnosis and treatment (e.g. drugs, hospital services). It can also include lost productivity costs (e.g. forgone earnings as a result of hospitalisation). However, using this approach, it is difficult to value more intangible costs, e.g. the change in utility (or personal wellbeing) an individual experiences as a result of a change in health status.
- **Willingness to pay (WTP)** approaches typically survey individuals about their willingness to pay to avoid specified health risks. This approach provides an estimate that potentially captures a variety of cost components, including pain and

suffering, perceived quality of life and lost productivity. Two methods used in this category include:

- *stated preference methods*, which do not rely on the presence of an actual market, but instead elicit values based on an individual's stated or anticipated actions
- *revealed preference methods*, which rely on the assumption that an individual's WTP is reflected in their actual actions. This approach uses data from related markets, such as the real estate market to infer the community's WTP to avoid pollutant emissions, or the labour market to infer an individual's willingness to accept increased occupational risks of injury or death.

Figure 5.1 shows how WTP and COI methods compare in terms of their coverage of health costs associated with pollutant emissions. The estimates used for valuation of the health endpoints in this report do not necessarily reflect all of the cost components identified in Figure 5.1. Instead, Figure 5.1 reflects the cost components that could potentially be covered by the valuation methodology.

Figure 5.1: Valuation methodologies and coverage of health costs (CIE, 2001)



[a] Aversion costs can be associated with both mortality and morbidity effects.

Valuing morbidity impacts

Willingness to pay—stated preference techniques

Stated preference techniques can potentially be used to estimate the full range of costs identified in Figure 5.1. There are three broad approaches relevant to the estimation of the WTP to avoid pollution-related health outcomes:

- *Contingent valuation* (CV)—Value estimates are contingent on a hypothetical scenario that is presented to respondents. Early CV studies used open-ended questions, but respondents often find it difficult to nominate a maximum WTP for non-market goods that they are not accustomed to paying for. Open-ended CV surveys have been largely surpassed by referendum versions. Referendum versions ask respondents to make a discrete choice between alternatives and can be used to calculate the WTP to avoid a particular health outcome.
- *Choice modelling*—A survey presents respondents with a series of discrete choice sets (or questions) and requires them to select one option from three or more alternatives. One of the attributes must be in dollar terms to estimate the amount of money people are prepared to pay for improving a non-monetary attribute by one unit. This method essentially gets respondents to 'trade off' one attribute against another to determine the most preferred options.
- *Conjoint analysis*—Respondents are required to rate alternatives rather than make a commitment to a particular option. Conjoint analysis is one step removed from decisions that are made in a market environment. This remove has led to debate about the extent to which conjoint analysis can provide a valid measure of welfare. Given this uncertainty, only CV and choice modelling are considered in the discussion below.

Willingness to pay—revealed preference techniques

Estimates generated by revealed preference techniques potentially encompass the full range of costs identified in Figure 5.1. Two common approaches include hedonic pricing and averting behaviour.

- *Hedonic pricing method*—This assumes that a good or service can be viewed as a bundle of different attributes, and that its total price is an aggregate of the implicit prices that consumers place on these attributes. This method relies on the statistical analysis of market data to identify these implicit prices. For example, the wage an employer must offer to attract workers reflects a range of variables representing working conditions, such as the occupational risk of injury, and education and experience requirements. Labour market data can provide information on how a small change in the risk of an occupational injury is associated with a wage increase. This association can provide an estimate for WTP for reduced health risk. Another example is that house prices are a function of a host of variables, including land size, number of bedrooms, building quality and external features such as location. In the present context, the proximity to a major roadway or industry—a source of pollutant emissions to air—might be one of the explanatory variables.

- *Averting behaviour*—This describes actions by individuals to mitigate or avoid health impacts. In the case of air pollution, these might include 'defensive' expenditures such as purchases of air purifiers or air filters; or behavioural changes, such as changes in daily activities (e.g. staying indoors) or moving to another location to minimise incidence. This expenditure provides an indication of a person's willingness to pay to avoid a particular health outcome.

Direct costing method—cost of illness (COI)

The COI approach is generally used for the valuation of morbidity endpoints, rather than mortality. It is used to estimate health costs from direct and indirect costs. Direct costs generally include the prices of goods and services associated with the diagnosis and treatment of an illness, such as medical fees, drugs and hospital stay. Indirect costs are largely associated with lost productivity, estimated as forgone earnings. Its coverage is restricted to cost items for which price can be observed or readily calculated from known cost data. It does not include pain and suffering or the full value an individual places on avoiding the health effect (e.g. fear or dread).

Valuing mortality impacts

Willingness to pay—value of a statistical life (VOSL)

The value of a statistical life (VOSL) is an aggregate measure of a community's willingness to pay to reduce the risk of premature mortality. It is a WTP-based measure, so to the extent that people take into account the pain and suffering of family and relatives, these costs will be reflected in the WTP estimate that they report and therefore in the overall VOSL measure (CIE, 2001).

VOSL is the valuation of small changes in the *risk* of death. This is appropriate in that any change in public policy will generally result only in small changes in the health of an individual or in the risk of a major change in that health status. VOSL is calculated as follows:

$$\text{VOSL} = \frac{\text{Average individual WTP for risk reduction}}{\text{Observed change in risk}}$$

WTP for risk reduction can be estimated by either revealed preference or stated preference methods. Revealed preference methods include the compensating wage differential approach (hedonic approach that assumes that higher wages are paid for more risky occupations to compensate for the higher risk of injury or death) and averting behaviour studies. A large number of studies have attempted to estimate VOSL, and these estimates are commonly applied to the valuation of a fatal risk change.

Direct costing method—the human capital approach

The approach does not aim to capture the full costs associated with the death of a person—in particular, the pain, suffering, fear or dread felt by the individual or those left behind. The approach treats a life as though it were the same as productive capital and

estimates its value as the discounted sum of future earnings. In effect, the human capital approach uses the labour market as a surrogate for a non-existent market for human life (Commonwealth Department of Finance, 1991). However, this approach does not take account of the value that individuals place on their own lives, or the value of non-work activity and other non-tangible elements of human life. The approach therefore tends to underestimate what people are willing to pay for reducing the risk of premature death (CIE, 2001).

Other approaches to valuing mortality effects—VOLYs and QALYs

Other approaches attempt to adjust a VOSL estimate to reflect changes in life expectancy or effects on health status before death. The two most common approaches are the value of life years (VOLYs) and quality adjusted life years (QALYs). The VOLY method assesses the value that an individual or a group of individuals place on a change in life expectancy, i.e. a gain or loss in life years. The QALY approach allows information relating to the health state of the individual at risk to be included by weighting the life expectancy of an individual with a perceived health status.[23] These approaches, however, are reliant on a VOSL estimate.

VOLY and QALY approaches have some appeal in that they allow the valuation of a health risk to be differentiated according to the age and change in health status of those affected. However, converting a VOSL to a VOLY or QALY measure has been criticised because it suggests that WTP varies inversely with age. However, the literature suggests that WTP shows an inverted U-shape with age, with elderly people's WTP to reduce the risk of premature death significantly higher than that suggested by the VOLY approach (CIE, 2001).

Summary

Table 5.1 summarises the strengths and weaknesses of the various valuation methodologies. In general, WTP estimates are preferred for the valuation of both mortality and morbidity effects, because WTP offers:

- a direct estimate of impacts on individual welfare with respect to changes in health status
- superior coverage of the range of health costs associated with air pollution.

[23] The health status is ranked on a scale of zero to one, with zero equal to death and one equal to perfect health.

Table 5.1: Summary of valuation methodologies (CIE, 2001)

Methodology	Coverage	Source of data	Strengths	Weaknesses	Technical notes
Methodologies for valuing morbidity					
Cost of illness	Treatment costs. Lost productivity (cost items with observed market prices)	Health statistics. Data on treatment costs. Labour force data	Widely applied. Easily understood. Relative lower cost	Limited coverage. Lower bound estimate on WTP	Benefit transfer from older studies is less reliable if treatment or treatment costs are significantly different
Willingness to pay— stated preference	All cost elements, depending on survey design	Survey of affected population	Potentially full coverage of costs Direct measure of individual welfare	Significant resources required to implement. Subject to biases, since behaviour is hypothetical and not actual	Survey design is critical if potential bias problems are to be overcome
Willingness to pay— revealed preference	All cost elements, depending on measure	Data on property prices or wages. Survey of individuals to identify extent of averting behaviour	Based on observed behaviour, so may not be subject to biases associated with stated preference techniques. Lower cost than a customised survey if relevant data available	Scope and accuracy limited by data availability	
Methodologies for valuing mortality					
Human capital	Mortality only	Labour force data	Simple and easily understood	Under-estimates WTP	Superseded technique
VOSL	Mortality, pain and suffering of family and friends	Survey. Compensating wage data. Other hedonic price data	A fuller measure of the costs associated with mortality. Survey approach allows specific population sub-groups to be targeted	Potential for nature of measure to be misrepresented—not a measure of a specific life. Hedonic methods restrict	Benefit transfer based on this methodology is preferred to human capital approach
VOLY/QALY	Mortality—attempts to adjust WTP estimate for effect of age and quality of life	As for VOSL	Attempts to capture the effects of age and quality of life on WTP	Some doubts over methodology. Potential for underestimating WTP of older people	

5.2. Health cost of air pollution

Table 5.2.1 provides an overview of the range of cost estimates that exist for health endpoints associated with air pollution. The table show that valuation estimates vary greatly for a specific endpoint and across different endpoints. This variation is a function of several factors. In general, estimates vary with the endpoint's severity, duration and reversibility. Values are also influenced by how an individual perceives an endpoint, e.g. fear or dread.[24] In addition, the estimates in Table 5.2.1 were developed with different methodologies. Some are COI estimates, while others are WTP.

[24] Some studies suggest that individuals can view certain illnesses or conditions as worse than death.

Table 5.2.1: Summary of valuation estimates for various health endpoints

Health endpoint	Range of estimates $A (2000) prices	No. of studies
Premature mortality	0.025m–22.2m*	56
Respiratory hospital admission	4,200–48,500	5
Cardiac hospital admission	4,700–24,100	3
Asthma symptom days	18–87	3
Acute respiratory symptom days	5–25	2
Casualty room visits	381–18,000	2
Restricted activity day	23–154	8
Child bronchitis	202–717	2
Chronic bronchitis	0.196m–2.2m	2

Source: CIE (2001).

* Industrial Economics Incorporated (1993) concluded that a 'reasonable' estimate of the VOSL would fall within the bounds of US$1.6–4.8 million (1997 dollars) (cited in Pearce and Howarth, 2000).

The cost of pain and suffering appears to be significantly greater than the cost of treatment, depending on the health effect and population under consideration, as is evident from the results presented in Table 5.2.2. This table shows the difference between WTP and COI estimates for selected health effects (the difference between WTP and COI is a measure of the value people place on avoiding harm from illness, including pain and suffering). As noted by the US EPA (2000b), it is evident that:

> 'Survey respondents have ranked pain or discomfort, emotional distress, and the lost enjoyment of normal activities as more important effects of angina (Chestnut *et al*., 1988), asthma (Rowe and Chestnut, 1985), or light symptoms (Berger *et al*., 1987) than the medical expenses and lost income that are the focus of the COI approach.'

Table 5.2.2: Comparison of WTP and COI estimates

Study/health effect	WTP estimate ($US)[1]	COI estimate ($US)[1]	WTP/COI ratio
Berger *et al*. (1987), air-pollution-related symptoms (one symptom day)			
Cough	$115	$18	6.2
Sinus congestion	$41	$10	4.0
Throat congestion	$66	$22	3.1
Itchy eyes	$73	$22	3.3
Heavy drowsiness	$214	$3	78.9
Headache	$164	$5	31.5
Nausea	$72	$4	19.2
All symptoms	$121.76	$5.93	20.5
Chestnut *et al*. (1988, 1996), angina episodes (one episode)[2]	$54–$147	$19	2.9–8.0
Rowe and Chestnut (1985), asthma severity (50% reduction in 'bad asthma days')[3]	$632–$920	$71–$197	3.2–9.8
US EPA (1997), chronic bronchitis (one case)			
All ages	$260,000		
Age 30		$77,000	3.4
Age 40		$58,000	4.5
Age 50		$60,000	4.3
Age 60		$41,000	6.3

Source: US EPA (2000b).

Notes:

1 All values in 1996 dollars.

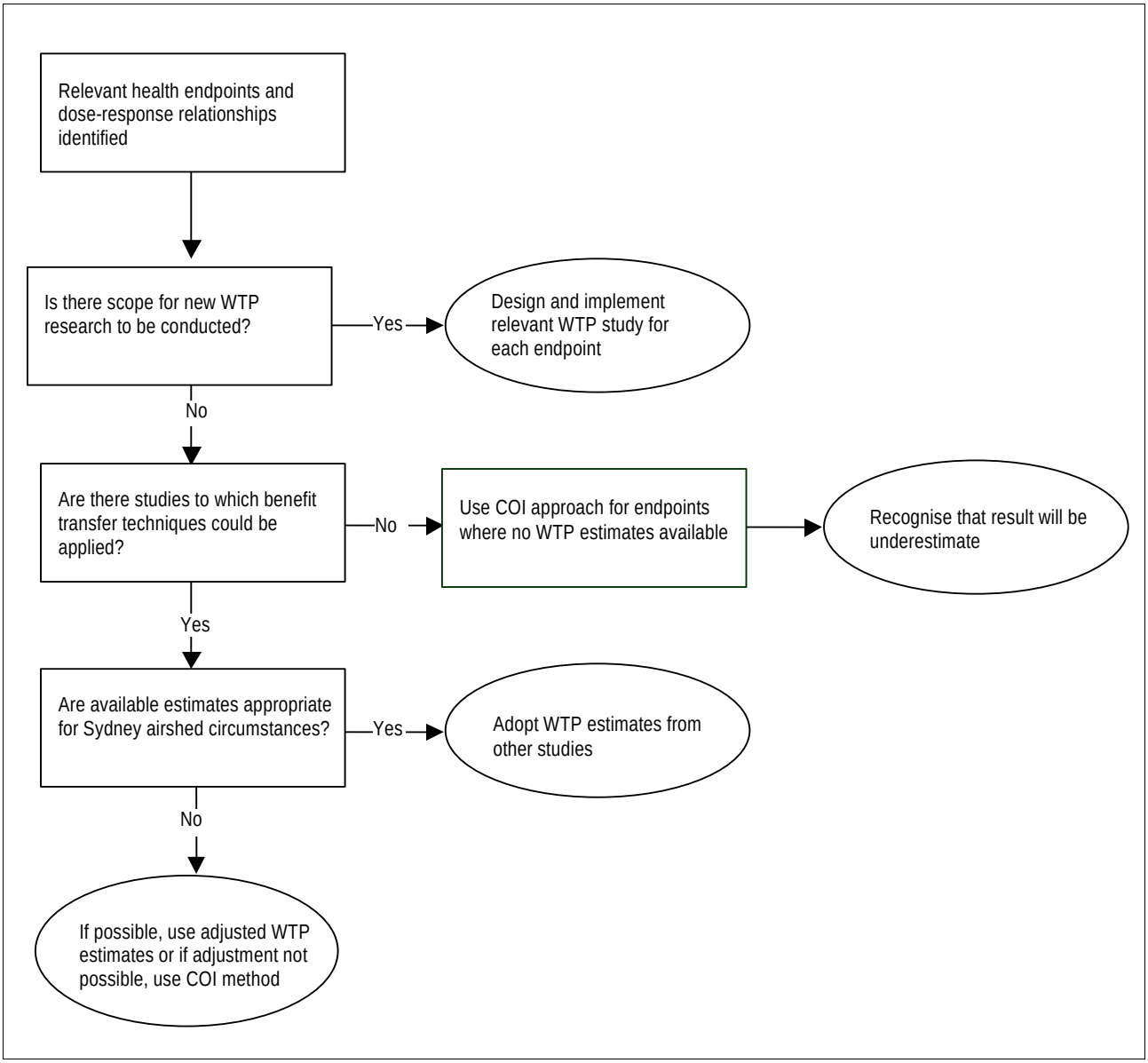
2 Estimates of WTP vary according to authors' treatment of high outlier estimates.

3 Estimates of WTP based on differing sample definitions.

5.3. Health cost valuation methods used in this study

In valuing the cost of health outcomes, this study followed the protocol in Figure 5.3 to assess the feasibility of valuing health endpoints. That is, the preferred approach was to use WTP to estimate costs of specific endpoints, as WTP measures provide more robust and complete coverage of the costs of a health outcome. However, COI was used where original research was infeasible or where WTP estimates were not available or appropriate to apply to this study.

Figure 5.3 Adopted approach for valuing health endpoints



The approach in Figure 5.3 was used to develop estimates for the valuation of health endpoints. Table 5.3.1 lists the methods and the resulting low and high cost estimates that were used to value each of the health endpoints relevant to this study.

Table 5.3.1: Valuation of health endpoints

Health outcome	Valuation methodology/ costs valued	Source	Low estimate (A$2003)	High estimate (A$2003)
Mortality (VOSL)	WTP	EU (2001) & EnHealth (2003)	1,004,000	2,500,000
Chronic bronchitis (adults)	WTP	Viscusi, Magat & Huber (1991) cited in OMA (1999)	214,000	569,000
Respiratory hospital admissions	COI*	NSW Health (2001)	3,880	4,660
Cardiovascular hospital admissions	COI*	NSW Health (2001)	7,000	8,400
Acute bronchitis (<15 years)	COI	Krupnick & Cropper (1989) cited in OMA (1999)	220	659
Asthma attacks (<15 years)	WTP	Rowe & Chestnut (1986) cited in OMA (1999)	22	97
Asthma attacks (>15 years)	WTP	As above	22	97
Restricted activity days (adults)	Lost productivity*	ABS (2003)	190	228

*Indicates that COI includes the value of lost productivity. For hospital admissions, lost productivity is calculated by multiplying the average length of stay by the average wage rate. For restricted activity days, lost productivity is the average daily wage rate.

As indicated in Table 5.2.1, numerous studies have attempted to estimate a VOSL. CIE (2001) noted that 56 studies gave a range of values between $0.025 and $22 million. However, there has been no major study of the value of a statistical life in Australia, and most road authorities in Australia base their costs of a fatality on the COI approach (EnHealth, 2003). For example, drawing on the COI method, the NSW RTA (1999) estimated a VOSL of $827,400 in 2000. According to EnHealth (2003), 'This approach underestimates the VOSL in Australia and it is proposed that the value of life should be based on international research until alternative Australian studies are available and agreed [on].'

The US EPA uses a VOSL of approximately US$6 million in most of its benefit–cost analysis (US EPA 2000a). This estimates represents the modal value of 26 VOSL estimates reviewed by Viscusi (1992).[25] An analysis of these values found the modal value to be US$4.5 million in 1997 dollars and concluded that a 'reasonable' estimate of the VOSL would fall within the bounds of US$1.6–$4.8 million (1997 dollars) (Pearce and Howarth, 2000). This translates into Australian dollars as approximately A$2.6–$7.6 million (adjusted for exchange rates and inflation).

Unlike the US EPA's approach, the EU argues that the VOSL is likely to decline with age. On the basis of its study of relevant values and an age adjustment, the EU (2001)

[25] The VOSL estimates come from 21 wage risks studies and five stated preference studies.

recommends a VOSL in the range of €0.65 million[26] to €2.5 million, with a 'best estimate' of €1 million (2000 prices), for mortality associated with environmental pollution. These estimates 'are applicable to deaths in a largely elderly population, where the reduction in life expectancy is likely to be short—maybe one year or less' (EU, 2001).

This study follows EnHealth's recommendation to use VOSL estimates rather than human capital estimates. It adopts low and high VOSL estimates of $1 million and $2.5 million. This range does not reflect the full range of estimates in the literature. The study uses the EU 'lower estimate' of €0.65 million (approximately A$1 million[27]) for the low estimate and the EnHealth (2003) recommended figure of $2.5 million for the high estimate.[28] Given the range of VOSL estimates cited in other studies, these figures are relatively conservative.

It is evident from comparing values in Tables 5.2.1 and 5.5.1 that this study has also been conservative in valuing respiratory and cardiovascular hospital admissions, acute bronchitis and restricted activity days in terms of medical treatment costs or lost productivity. That is, for these health outcomes, only COI estimates are used, and these estimates do not fully account for people's willingness to pay to avoid pain and suffering.[29]

[26] Euros.

[27] Converted to Australian dollars with Purchasing Power Parity rather than market exchange rates.

[28] 'In lieu of any agreed determination on VOSL, drawing on international studies, these Guidelines use an average value of A$2.5 million as a realistic figure for VOSL in Australia.' (EnHealth, 2003.)

[29] Where COI includes the cost of measures to limit pain, then *some* cost of pain and suffering is factored into the cost estimate. According to the US EPA (2000), no application of the cost-of-illness approach will account fully for pain and suffering (e.g. medication may only limit pain, not eliminate it), but illness costs may include a partial monetisation.

6. ESTIMATING THE HEALTH COST OF URBAN AIR POLLUTION

This section combines the exposure-response estimates listed in Table 4.1 with the baseline frequency of health outcomes (i.e. the expected number of health outcomes at the lowest assumed threshold assessed level of PM_{10} of 7.5 μg/m^3) to estimate the number of additional health outcomes attributable to air pollution. These values are then combined with the health cost estimates listed in Table 5.3.1 to calculate the (‘*at least*’) health cost of air pollution in the GMR.

6.1. The ‘at least’ approach

To estimate the health cost of air pollution, data are required on exposure-response relationships, the baseline population prevalence of health outcomes, ambient pollution levels for the region, and the economic valuation of quantified health endpoints attributable to air pollution. This study follows Kunzli *et al*. (1999) in using an ‘at least’ approach in calculating the health cost of ambient air pollution, ‘thus consistently selecting methodological assumptions in a way to get an impact which may be expected to be “at least” attributable to air pollution. Accordingly, the overall impact of air pollution is expected to be greater than the final estimates.’ This study also follows Kunzli *et al*. (1999) by reporting results as a range of impacts rather than an exact point estimate, to ‘unambiguously communicate’ the inherent uncertainty of such a calculation.

Components of the ‘at least’ approach used in this study are discussed below.

The index pollutant approach

As mentioned in Chapter 3, the total health effects of air pollution, which is a mixture of many known and unknown substances, can be considered the sum of:

1. all independent effects of specific pollutants
2. the effects of mixtures
3. the additional effects (positive or negative) due to interactions between pollutants.

The usual approach of epidemiological studies is to measure the association between at least one specific pollutant (e.g. PM, NO_x, CO or O_3) and health. These specific components, which are usually highly correlated with other pollutants, are considered indicative of the complex pollutant mixture. So it is unclear how much the associations reported in epidemiological studies represent the independent effects of specific pollutants. Therefore, simply summing the pollutant-specific impacts could lead to an overestimation of the overall impact of air pollution on health.

Accordingly, this study adopts Kunzli *et al*.’s (1999) approach of using PM_{10} as the sole indicator (‘index pollutant’) of the urban air pollution mix. This method is likely to *underestimate* the total health impact, particularly as it does not account for the independent effects of ozone. It also does not include the additional health effects of air toxics, such as excess cancer cases. Nevertheless, it overcomes any inherent

epidemiological uncertainty in determining the independent effects of correlated pollutants and provides an 'at least' health cost estimate.

Lowest assessed level of 7.5 µg/m^3

For its base case, this study follows the lead of Kunzli *et al*. (1999) in calculating health impacts of PM_{10} only above 7.5 µg/m^3. This is despite the World Health Organization's recommendation that no threshold for PM_{10} be used, meaning that even very low mean annual concentrations may have long-term effects on a population (WHO, 2000).

Kunzli *et al*. (1999) used this approach because then-available epidemiological studies had not included populations exposed to levels below 5–10 µg/m^3 (mean 7.5 µg/m^3). The use of this threshold also accounts for non-anthropogenic sources of PM_{10} emissions (i.e. it is sufficient to ensure that non-anthropogenic sources of PM_{10} are not included in the health cost of air pollution estimate).

Selecting non-overlapping health outcomes

DEC selected the following health endpoints to be quantified and monetised: total mortality (based on cohort or long-term studies), respiratory hospital admissions, cardiovascular hospital admissions, and incidences of chronic bronchitis in adults, acute bronchitis in children, restricted activity days in adults, asthma attacks in children, and asthma attacks in adults.

Several health outcomes were excluded from the assessment because they may be partially included in the above-mentioned health endpoints. This eliminated the risk of double counting (e.g. premature death due to short-term exposure, casualty room visits, respiratory symptoms in adults etc). Others were excluded because they are difficult to express in monetary terms (e.g. reduced lung function, school absenteeism, reduced physical performance, change in bronchial reactivity). Infant and intrauterine mortality were also excluded, as there has been limited research on each of these outcomes (Kunzli *et al*., 1999).

Therefore, the health outcomes included in the calculation do not include all outcomes for which there is an association with current or long-term ambient air pollution exposure.

Economic valuation

The valuation of mortality risk drives total health costs, and the low and high VOSL estimates used in this study are relatively conservative. Additionally, several health outcomes have been valued only in terms of COI. As described, the COI approach underestimates the total cost of a health outcome, as costs of pain and suffering and loss of leisure are not adequately factored into the analysis.

6.2. Steps in the estimation

Estimation of the health cost of ambient air pollution involved the following six steps.

1. Estimate average ambient PM_{10} levels

Table 6.2.1 presents average ambient PM_{10} levels in the GMR for 2000 to 2002. These are based on DEC monitoring data.

Table 6.2.1: Average ambient PM_{10} levels in the GMR 2000–2002

Area	Annual average[1]
Sydney	20.4 µg/m^3
Newcastle, Hunter	20.5 µg/m^3
Wollongong, Illawarra	19.1 µg/m^3

1. 24-hour average

The health cost calculation assumes that the entire population in each area is exposed to the average annual PM_{10} concentration for that area (as listed in table 6.2.1).[30]

2. Select relevant exposure-response estimates

The exposure-response estimates underlying this study are discussed and listed in section 4.1.

The epidemiology report of Morgan and Jalaludin (2001) provided exposure-response estimates expressed as 'relative risk', which is a common measure of effect used to report results in epidemiological studies. For example, a relative risk of 1.026 in chronic mortality for a 10 µg/m^3 increase in annual average PM_{10} means that a person exposed to such an increase has a 2.6% greater risk of dying than a person exposed to the lower pollution level.

Relative risk is not an absolute measure of risk, but a measure of the change in the level of risk. If those exposed and those unexposed to pollution have the same risk of a particular adverse health effect, then the relative risk equals 1. This study uses an additive risk function rather than a multiplicative function (as per Kunzli et al. 1999). This prevents unrealistic estimates of health effects in situations where the ambient concentration is higher than that used in this study. The additive risk function means that the number of additional cases is estimated for an increment in PM_{10} rather than a percentage increment in the health outcome.

[30] This follows the methodology of other similar studies conducted here and overseas (e.g. Rolfe *et al*., 2002, and Amoako *et al*., 2003).

3. Determine incidence or prevalence of health outcomes

For each of the health endpoints[31] identified by the epidemiology report, International Classification of Diseases (ICD) codes were used to derive relevant health costs.

Data on the incidence or prevalence of each health endpoint was obtained from NSW Health (2001), except for restricted activity days, asthma attacks and bronchitis. The number of restricted activity days was estimated from the ABS (1997) 1995 National Health Survey. Asthma attack data for adults were sourced from Public Health Division (2001), and for children from Robertson *et al.* (1998) and Woolcock *et al.* (2001). The prevalence of chronic and acute bronchitis was assumed to be 0.71% of adult population and 6.5% of child population. These figures on bronchitis prevalence were sourced from Kunzli *et al*.'s (1999) review of overseas studies.[32]

4. Estimate baseline frequency of health endpoints

Kunzli *et al*. used the following equation to estimate the proportion of the population that would experience an identified health outcome at the lowest assessed (or baseline) level of PM_{10}:

$$Po = \frac{Pe}{1 + [(RR - 1)(E - B)/10]}$$

where:

Pe = observed prevalence or incidence from studies and statistics
Po = baseline population frequency
E = observed average exposure level
B = baseline exposure level of 7.5 µg/m^3 of PM_{10}
RR = relative risk function for a 10 µg/m^3 increase in PM_{10}, derived from epidemiological studies.

To test the sensitivity of the results to this approach, cost estimates were also calculated without a PM_{10} threshold (see section 6.4).

5. Ascribe health cost estimate

Cost estimates (and methods of valuation) for each health outcome are listed in Table 5.3.1 in chapter 5.

[31] See Appendix 1, Table A.1.

[32] The childhood acute bronchitis prevalence figure of 6.5% is based on the work of Dockery *et al*. (1993, 1996). Dockery *et al*. (1993) reported data collected in 1980–81 (the Harvard 6-Cities study) from 5422 children aged 10 to 12, finding a prevalence of 6.3% bronchitis diagnosed by a doctor in the previous 12 months. For the extended Harvard 24-Cities study, the prevalence in 13 369 children aged 8 to 12 was between 3% and 10%. This compares with prevalence of 7.6% in lower Austria (Studnicka *et al.,* 1997); between 5.9% (Oberfeld & König. 1996) and 14.8% (Oberfeld *et al.,* 1997) in Salzburg; and 12.2% in Switzerland (Braun-Fahrländer *et al.* 1995).

The frequency figure of 0.71% for adult chronic bronchitis was obtained from Abbey *et al*. 1993. Notably, this is conservative, as it was obtained from a non-smoking population in southern California.

6. Estimation

To calculate the total health impact, the exposure-response estimates applicable to the GMR are combined with the population at risk. The number of cases (after adjustment for threshold effects) is then multiplied by the health cost estimate to give a total cost (within a low and high range).

In summary, the calculation is derived as follows:

- Prevalence data adjusted for baseline (or threshold) = population at risk
- Relative risk × adjusted prevalence data = number of people at risk
- Number at risk × per-unit health cost = health cost per health endpoint
- Sum each health impact to get regional impact
- Calculate cost per region at mean ambient level
- Divide total cost by total emissions = health cost per tonne

To demonstrate how the results were derived, the following example is shown for total PM_{10} impacts on respiratory hospital admissions in Sydney.

- A 10 µg/m^3 increase in PM_{10} above the threshold is associated with an increase in respiratory hospital admissions of between 0.5% and 1.1% (central estimate is 0.8%). (Table A.3.1)
- There were approximately 56 064 cases of respiratory hospital admissions in the Sydney region each year. Following the conservative approach adopted by Kunzli et al. (1999), we did not estimate any health effects below 7.5 µg/m^3 for the base case. Therefore, we adjust the number of people potentially affected, using the baseline prevalence equation, which gives an adjusted prevalence of around 55 280 people for the 'low' estimate and 55 705 people for the 'high' estimate, at the baseline ambient level. These figures are then multiplied by the high and low relative risk factors. The resulting estimate is that between 359 and 784 people are likely to be admitted to hospital for an increase of 10 µg/m^3 in PM_{10}).
- The average cost of a respiratory hospital admission in the Sydney region is estimated at between $3,884 and $4,660 per case (Table 5.3.1). The estimates of the number of cases likely to result from an increased exposure to PM_{10} (per 10 µg/m^3) are multiplied by these costs.
- The cost of respiratory hospital admissions in Sydney per 10 µg/m^3 increase in PM_{10} is therefore likely to be between $1.4 million (359 people × $3,884) and $3.56 million (784 people × $4,660).
- The same process is repeated for each health endpoint. The total annual health cost of air pollution in Sydney, per 10 µg/m^3 of PM_{10}, is estimated at between $547 million and $4.6 billion. For the GMR, this cost is estimated as between $791 million and $6.6 billion.
- The health cost at the mean ambient level is then calculated as follows: Mean ambient load for Sydney of 20.4 µg/m^3 less baseline level of 7.5 µg/m^3 = 12.9 µg/m^3. The cost per 10 µg/m^3 is then multiplied by 1.29 to calculate the total cost per region. The health cost of ambient pollution in Sydney is therefore estimated to be between

$706 million and $6 billion per annum (for the GMR, this range is between $1.01 billion and $8.4 billion per annum).

- Total regional health costs are then divided by the total anthropogenic emissions of PM_{10} in each region, to give an indication of the health cost associated with a tonne of PM_{10}. For Sydney, the health cost associated with a tonne of PM_{10} is therefore between approximately $28,000 and $235,000 (midpoint of $132,000).

6.3. Results

Table 6.3.1 present estimates of the health costs of air pollution in the GMR, using PM_{10} as the single indicator of the health effects of air pollution.

As previously mentioned, these are conservative estimates, as:

- PM_{10} costs were calculated using a threshold effect (i.e. assuming no costs up to the threshold)
- many additional chronic illnesses associated with air pollution were not included in the calculations
- seasonally limited health effects related to air pollution were not considered (e.g. ozone exposure in summer)
- the cost estimates of health endpoints used in this study are considered conservative (of particular significance is the cost estimate for mortality).

Table 6.3.1: Annual health costs of air pollution in the GMR (2003$)

Region	Low	High	Midpoint
	Total cost at mean ambient level ($ million)		
Sydney	706	5,994	3,350
Hunter	226	1,765	996
Illawarra	81	638	360
GMR	1,013	8,397	4,705
	Cost per tonne of PM_{10} ($ thousand)		
Sydney	28	235	132
Hunter	8	63	35
Illawarra	6	46	26

The estimated numbers of additional adverse health outcomes due to mean ambient pollution are listed in Table A.2 in Appendix 2.

Much of this study's methodology is based on the three-nation study in Austria, France and Switzerland, which was conducted on behalf of the Third European Ministerial Conference on Transport, Environment and Health (WHO, 1999).[33] Table 6.3.2 compares the results of our study with the results of the three-nation study in terms of

[33] The Kunzli *et al*. (1999) paper was key component of this three-nation study.

health cost per capita and health cost relative to those countries' gross domestic products (GDP) and to the NSW gross state product (GSP).

Table 6.3.2: Comparison of costs derived from this study and those of the three-nation study in Austria, France and Switzerland

Area	Health cost due to air pollution	
	% of GDP (or GSP)	Per capita
GMR	0.4% to 3.4% (NSW GSP)	$192 to $1,594
Austria	1.6% to 4.8%	$694 to $2,042
France	1.4% to 4.0%	$562 to $1,641
Switzerland	0.7% to 2.2%	$485 to $1,458

6.4. Sensitivity analysis

For the purposes of a sensitivity analysis, health costs were also estimated assuming no threshold for PM_{10}. Such a scenario is consistent with the World Health Organization's determination that there is no safe level of exposure to PM_{10}.[34]

The results of the sensitivity analysis are presented in Table 6.4.1. They show that without a PM_{10} threshold, the annual health costs of air pollution are significantly higher than if a threshold is assumed.[35] For instance, for the base case (that is, estimating the effects of PM_{10} only above a baseline of 7.5 $\mu g/m^3$), the 'low' estimate of the annual health cost of ambient pollution in the GMR is approximately $1.01 billion (Table 6.3.1). This compares with an estimate of $1.7 billion when the effects of PM_{10} are estimated without a threshold (Table 6.4.1). Corresponding 'high' estimates are $8.4 billion and $15.2 billion respectively.

Table 6.4.1: Annual health costs of air pollution in the GMR when effects of PM_{10} are estimated *without* a threshold (2003$)

Region	Low	High	Midpoint
	Total cost at mean ambient level ($ million)		
Sydney	1,153	10,872	6,012
Hunter	368	3,163	1,766
Illawarra	137	1,179	658
GMR	1,658	15,214	8,436
	Cost per tonne of PM_{10} ($ thousand)		
Sydney	45	427	236
Hunter	13	112	63
Illawarra	10	85	47

[34] The US EPA also concluded that there is currently no scientific basis for selecting a threshold for the effects of the major pollutants (including PM, CO and NO_2), if a threshold is defined as a level characterised by an absence of observable effects.

[35] If there is no threshold then health costs from PM_{10} pollution occur at any level of pollution.

6.5. Allocating health costs to specific sources

The section looks at some of the issues to consider, or limitations, in interpreting or applying the results of this study.

The index pollutant approach can be used to estimate the health cost of ambient air pollution in a given airshed. As shown in Table 6.3.1, the estimated health cost of air pollution (assuming a 7.5 μg/m^3 threshold) in Sydney is between approximately $700 million and $6 billion per annum. This reflects the effect of ambient air pollution levels averaged out over the entire metropolitan area.

Assigning health costs to specific sources is more problematic. Consider the case of emissions from motor vehicles. Table 2.3 in Chapter 2 shows that motor vehicles are responsible for approximately 9% of anthropogenic PM_{10} emissions in the Sydney airshed. This equates to a mean ambient PM_{10} level of 1.86 μg/m^3 (9.10% $\times$ 20.4 μg/m^3 = 1.86 μg/m^3). The annual health cost of this level of ambient PM_{10} in Sydney is estimated to range between $105 million and $990 million.[36]

However, for reasons discussed below, this range is likely to underestimate the health cost of motor vehicle emissions in Sydney:

Motor vehicles emit relatively low levels of PM_{10} but are still significant contributors to the ambient air pollution mix

This study uses PM_{10} as the indicator of the health impacts of the ambient air pollution mix because of the difficulty in determining the independent health effects of common air pollutants. PM_{10} is not the sole source of health impacts—other pollutants also contribute.

Tables 2.3 to 2.5 in Chapter 2 show that while motor vehicles contribute only about 9% of PM_{10} emissions in Sydney, they are significant contributors to the ambient air pollution mix. Motor vehicles are responsible for 38% of VOC emissions and 58% of NO_x emissions in Sydney. They are also by far the major source of CO emissions and a source of air toxics in urban areas.[37] This means that, by focusing on PM_{10} only, the index pollutant approach may underestimate the health impacts of air pollution from motor vehicles.

The conclusion from this is that while the indicator pollutant approach provides a useful technique for estimating health costs attributable to air pollution, policy action to control air pollution must still focus on a range of pollutants, not just PM_{10}.

[36] A threshold or baseline level of PM_{10} is not factored into these calculations. It is likely that such a baseline level of pollution would be reached via other sources, irrespective of the presence of road traffic pollution.

[37] Motor vehicles 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 (NEPC, 2003).

Motor vehicles' contributions to PM_{10} exposure levels are difficult to determine

Pollution from motor vehicles is emitted close to the ground, near large numbers of the population (e.g. in streets surrounded by residences), and consistently all year round. This may mean that motor vehicles contribute more than (the above-mentioned) 9% of ambient PM_{10} levels that the average Sydney resident is exposed to.

For example, Amoako *et al*. (2003) have related estimates of PM_{10} emissions from motor vehicles to population distribution, and estimate that motor vehicles are responsible for 43% of annual average PM_{10} exposure levels in Sydney. This equates to an ambient PM_{10} exposure level in Sydney of 8.77 $\mu g/m^3$ ($43\% \times 20.4\ \mu g/m^3 = 8.77$), or a health cost of motor vehicle emissions in Sydney of between $496 million and $4.7 billion per year.

More research is required on apportioning the health impact of air pollution to specific sources.

7. CONCLUSION

Few studies have evaluated the economic cost of air pollution in the GMR in monetary terms. Using PM_{10} as the index pollutant, this study has conservatively estimated the health cost of ambient air pollution in the GMR to be between $1.01 billion and $8.40 billion per annum. If the health impacts of PM_{10} are estimated without a threshold, the cost increases to between $1.66 billion and $15.21 billion per annum. The health costs of air pollution are real and substantial, and a reduction in air pollution would deliver long-term benefits from the population's improved health. These current estimates of the cost of air pollution will change over time with factors such population growth, changes to vehicle and fuel standards, and changes in production processes.

Estimating health costs from air pollution is an inherently uncertain process. However, these health cost estimates were derived using conservative valuation methods. Many health impacts were not costed; conservative estimates of the costs of health endpoints were used; and the index pollutant approach possibly undervalues a significant proportion of the health effects of other pollutants, particularly ozone.

Over time, these estimates can be refined as more epidemiological research is published, especially in regard to compounding effects. Potential areas of research include the following:

- the allocation of impacts to specific sources
- incorporating the effects of fine particulates ($PM_{2.5}$), especially given the serious health impacts identified in US research[38]
- quantification of the health effects of ozone, a pollutant of particular concern in Sydney during the warmer months
- the health effects of air toxics, particularly effects associated with exposure at levels found in the general environment
- assessment of the effects on special risk groups (e.g. air-pollution-related effects on infant birth weights, children's health etc.).

The results of this study are intended to inform planners and policy makers considering proposals that may reduce air quality, and developing and analysing measures to reduce emissions and protect the environment.

[38] US research suggests that $PM_{2.5}$ has possibly 4 times the effect of PM_{10} by concentration, although this is contentious.

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APPENDIX 1: HEALTH COSTS DUE TO AIR POLLUTION

Table A.1: Annual health costs per 10 µg/m^3 increase in particulate pollution (above 7.5 µg/m^3 threshold)

	Health costs (A$,000, 2003)			
Health endpoint	**Sydney low–high**	**Hunter low–high**	**Illawarra low–high**	**Total low–high**
Mortality (long-term effects) — >30 years	$500,640–$2,803,000	$162,000–$909,000	$65,200–$367,000	$728,000–$4,078,000
Respiratory hospital admissions	$1,082–$2,834	$270–$707	$131–$343	$1,480–$3,880
Cardiovascular hospital admissions	$3,047–$7,853	$898–$2,314	$439–$1,132	$4,380–$11,300
Acute bronchitis (<15 years)	$1,220–$9,680	$327–$2,590	$137–$1,120	$1,680–$13,400
Asthma attacks (<15 years)	$20–$196	$5.8–$56	$1.8–$18	$28–$270
Asthma attacks (>15 years)	$0–$77	$0–$20.5	$0–$6.1	$0–$104
Restricted activity days (>19 years)	$2,142–$3,430	$521–$833	$210–$337	$2,870–$4,600
Chronic bronchitis (adults)	$39,200–$1,820,000	$9,535–$443,000	$3,816–$181,000	$52,500–$2,440,000
Total costs	$547,000–$4,650,000	$174,000–$1,360,000	$69,900–$550,000	$791,000–$6,550,000

APPENDIX 2: ADDITIONAL HEALTH OUTCOMES DUE TO AIR POLLUTION

Table A.2: Annual number of additional health outcomes at mean ambient pollution level (effects of PM_{10} *with* threshold of 7.5 µg/m^3)

Health impacts	Sydney	Hunter	Illawarra	Total
	low–high (base incidence)	low–high (base incidence)	low–high (base incidence)	low–high (base incidence)
Mortality (long-term effects) — >30 years	643 to 1 446 (19 825)	210 to 473 (6 433)	75 to 170 (2 573)	929 to 2 089 (28 831)
Respiratory hospital admissions	359 to 784 (56 064)	90 to 197 (13 998)	39 to 85 (6 775)	489 to 1 067 (76 837)
Cardiovascular hospital admissions	561 to 1 206 (73 102)	167 to 358 (21 542)	73 to 156 (10 517)	801 to 1 720 (105 161)
Acute bronchitis (<15 years)	7 149 to 18 945 (48 200)	1 933 to 5 112 (12 944)	724 to 1 969 (5 349)	9 806 to 26 025 (66 493)
Asthma attacks (<15 years)	1 184 to 2 605 (35 182)	344 to 758 (10 159)	97 to 215 (3 201)	1 626 to 3 578 (48 542)
Asthma attacks (>15 years)	0 to 1 027 (100 507)	0 to 275 (26 757)	0 to 74 (8 022)	0 to 1 376 (135 286)
Restricted activity days (>19 years)	14 517 to 19 350 (156 966)	3 558 to 4 741 (38 203)	1 282 to 1 714 (15 270)	19 357 to 25 806 (210 439)
Chronic bronchitis (adults)	237 to 4 126 (20 611)	58 to 1 010 (5 016)	21 to 368 (2 005)	315 to 5 504 (27 632)

APPENDIX 3: EXPOSURE-RESPONSE ESTIMATES FOR SYDNEY

Table A.3.1: Recommended PM exposure-response estimates for Sydney

Health outcome	Source	Applied population	PM measure µg/m^3 (range)	E-R Estimate for a 10 µg/m^3 change in PM		Uncertainty rank	Generalis-ability rank
Mortality (long-term exposure effects): all deaths excluding violent deaths & accidents	PM_{10} Pooled estimate from Kunzli *et al*. (1999) (using Dockery *et al*. 1993 and Pope *et al*. 1995)	Adults >30 years	PM_{10} (annual mean) 18–47	low central high	1.026 1.043 1.061	B	++
	$PM_{2.5}$ Pooled estimate using Dockery *et al*. 1993 and Pope *et al*. 1995		$PM_{2.5}$ (annual mean) 9–34	low central high	1.041 1.066 1.092	B	++
Mortality (short-term exposure effects): all deaths excluding violent deaths & accidents	PM_{10} WHO 2000	All ages	PM_{10} 18–115	low central high	1.0062 1.0074 1.0086	A	+++
	$PM_{2.5}$ Schwartz *et al*. 1996	PM	$PM_{2.5}$ 11–30	low central high	1.011 1.015 1.019	B	
Respiratory hospital admissions (ICD9 460–519)	WHO 2000	All ages	PM_{10} 33–51 (10th–90th percentile of individual cities ranges from 14 to 120)	low central high	1.005 1.008 1.011	A_{+++}	+++
		PM					

Health outcome	Source	Applied population	PM measure µg/m^3 (range)	E-R Estimate for a 10 µg/m^3 change in PM		Uncertainty rank	Generalis-ability rank
Cardiovascular hospital admissions (ICD9 390–459)	Kunzli *et al*. 1999	All ages	PM_{10} 15–51	low central high	1.006 1.009 1.013	A	+++
Respiratory ED hospital attendance (ICD9 460–519)	WHO 2000 adjusted for Sydney ratio of respiratory hospital admissions to ED attendance	All ages	PM_{10} See respiratory admissions	low central high	Waiting on population data?	C	+
Cardiovascular hospital ED attendance (ICD9 390–459)	Kunzli *et al*. 1999 adjusted for Sydney ratio of cardiovascular hospital admissions to ED attendance	All ages	PM_{10} See cardiovascular admissions	low central high	Waiting on population data?	C	+
Acute bronchitis	Kunzli *et al*. 1999 Pooled analysis using Dockery *et al*. 1989, Dockery *et al*. 1996, Braun-Fahrlander *et al*. 1997	Children <15 years	PM_{10} Approx 15 to 59	low central high	1.135 1.306 1.502	A	+++
Asthma attacks	Kunzli *et al*. 1999						
	Joint estimate of studies in the Netherlands (Roemer 1993), Paris (Segala *et al.* 1998) and the USA (Pope *et al.* 1991)	Asthmatic children <15 years	PM_{10} and PM_{13}	low central high	1.027 1.044 1.062	A	++
	Joint estimate of studies in the Netherlands (Dusseldorp et al. 1995; Hilterman *et al.* 1998), Paris [Neukirch *et al.* 1998] and the USA (Pope *et al.* 1991) (Ostro *et al.* 1991)	Asthmatic adults ≥15 years	PM_{10} and PM_{13} PM ranges not explicitly stated in all studies. Sydney daily average PM_{10} within range of individual cities estimated from available data	low central high	1.000 1.004 1.008	A	++

Health outcome	Source	Applied population	PM measure µg/m^3 (range)	E-R Estimate for a 10 µg/m^3 change in PM		Uncertainty rank	Generalis-ability rank
Restricted activity days: respiratory-related restricted activity days per person per year, considered equivalent to 'absence from work'	Kunzli *et al*. 1999 based on Ostro 1990	Adults ≥20 years	PM_{10} (estimated from PM_{15}) PM ranges not explicitly stated in all studies. Sydney daily average PM_{10} within range of individual cities estimated from available data	low central high	1.079 1.094 1.109	C	+
Chronic bronchitis: symptoms of cough and/or sputum production on most days, for at least 3 months per year, and for 2 years or more	Kunzli *et al.* 1999 based on Abbey *et al*. 1993	Adults ≥25 years	PM_{10} (estimated from total suspended particles) PM ranges not explicitly stated in all studies. Sydney daily average PM_{10} within range of individual cities estimated from available data	low central high	1.009 1.098 1.194	B	+

Uncertainty rank: A, very good level of certainty; B, good level; C: sufficient level

Generalisability rank: +++, strong evidence of generalisability; ++, good evidence; +, sufficient evidence

Table A.3.2: Recommended ozone exposure-response estimates for Sydney

Health outcome	Source	Population	Ozone measure ppb (1-hour max.) (range)	E-R estimate for a 25 ppb change in ozone		Uncertainty rank	Generalis-ability rank
Mortality (short-term exposure effects)	Pooled estimate from US EPA 1997 report	All deaths (excluding violent deaths & accidents)	20–70* (26–319)	low	0.999	B	+++
				central	1.014		
				high	1.029		
All respiratory hospital admissions (ICD9 460–519)	Pooled estimate from Spix 1998	15–64 years	7–35	low	1.005	B	+
				central	1.019		
				high	1.033		
		65+ years		low	1.015	B	+
				central	1.031		
				high	1.047		
COPD hospital admissions (ICD9 490–492, 496)	Pooled estimate from Anderson *et al*. (1997)	All ages	19–39	low	1.011	B	+
				central	1.029		
				high	1.047		
Asthma hospital admissions (ICD9 493)	Pooled estimate from Sunyer *et al*. (1997)	All ages	14–36	low	1.011	B	+
				central	1.029		
				high	1.047		
Respiratory symptoms[†]	BTCE/VEPA 1994 (based on Krupnick *et al.* 1990)	All ages	98.6	low	$0.078 \times O \times pop$[‡]	C	+
				central	$0.153 \times O \times pop$		
				high	$0.229 \times O \times pop$		

Health outcome	Source	Population	Ozone measure ppb (1-hour max.) (range)	E-R estimate for a 25 ppb change in ozone		Uncertainty rank	Generalis-ability rank
Minor restricted activity day	BTCE/VEPA 1994 (based on Ostro and Rothschild 1989, Portney and Mullahy 1986)	All ages	23–42	low	1.93×10^{-2}§	B	+
				central	4.67×10^{-2}		
				high	7.40×10^{-2}		
Asthma attacks (daily risk of asthma attack per asthmatic)	BTCE/VEPA 1994 (based on Whittemore and Korn 1980, Holguin *et al.* 1985, Stock *et al.* 1988)	All people with asthma	23–150	low	$0.016 \times O \times pop$	C	+
				central	$0.188 \times O \times pop$		
				high	$0.520 \times O \times pop$		

Uncertainty rank: A, very good level of certainty; B, good level; C: sufficient level

Generalisability rank: +++, strong evidence of generalisability; ++, good evidence; +, sufficient evidence

* One-hour mean not available for one study.

† 19 respiratory symptoms were recorded, including chest discomfort, coughing, wheezing, sore throat, cold, and doctor-diagnosed flu.

‡ O = Highest daily one-hour ozone reading (in ppm) on days where maximum ozone level > 0.08 ppm

§ Daily risk for 'minor restricted activity day' per person for each ppm of high-hour ozone.

Table A.3.3: Recommended NO_2 exposure-response estimates for Sydney

Health outcome	Source	Applied Population	NO_2 ppb (Range)	E-R estimate for a 25 ppb change in NO_2		Uncertainty Rank	Generalisability Rank
COPD hospital admissions	Anderson *et al*. 1997	All ages	Median of 24 hr NO_2 range: 21 to 35	low	1.002*	B	+
				central	1.019*		
				high	1.047*		
Asthma hospital admissions	Sunyer *et al*. 1997	<15 years	Median of 24 hr NO_2 range: 18 to 35	low	1.006*	B	+
				central	1.026*		
				high	1.049*		
Lower respiratory tract symptoms	Hasselblad *et al*. 1992	5–12 years	2 week average NO_2 range: 0 to 75 ppb	low	1.17†	A	+++
				central	1.34†		
				high	1.50†		
Phlegm and sputum	Schwartz and Zeger 1990	Adults 18+ years	Mean of 24 hr NO_2: 130 ppb	low	1.004*	C	+?
				central	1.022*		
				high	1.041*		

* 24-hour average NO2; † 2-week average NO2

Uncertainty rank: A, very good level of certainty; B, good level; C: sufficient level

Generalisability rank: +++, strong evidence of generalisability; ++, good evidence; +, sufficient evidence

Table A.3.4: Summary of CO exposure-response estimates for selected health endpoints for Sydney

Health outcome	Source	Population	CO Measure (range)	E-R Estimate for a 1 ppm increase in daily CO		Uncertainty rank	Generalisability rank
Asthma hospital admissions (acute effect)	Sheppard *et al*. 1999	<65 years	Daily mean = 1.83 ppm (10th & 90th percentiles = 1.02 & 2.8 ppm)	low central high	1.017 1.054 1.093	C	+
All cardiovascular disease hospital admissions (acute effect)	Schwartz 1999	65+ years	Daily median ranged from 2 to 4.7 ppm (25th & 75th percentile = 2.0 & 6.4 ppm)	low central high	1.008 1.013 1.018	B	+

Uncertainty rank: A, very good level of certainty; B, good level; C: sufficient level

Generalisability rank: +++, strong evidence of generalisability; ++, good evidence; +, sufficient evidence

Table A.3.5: Summary of risk estimates for air toxics

Toxic	Source	Estimate	URF ($\mu g/m^3)^{-1}$ RfC (mg/m^3)	Uncertainty rank	Endpoint
Inhalation Unit Risk Factor (URF—cancer endpoints)					
Benzene	US EPA	low	2.2×10^{-6}	B	All cancer
	WHO	central	6.0×10^{-6}		
	Cal EPA	high	29.0×10^{-6}		
1,3-Butadiene	US EPA	low	1.7×10^{-4}	A	Leukaemia
	midpoint	central	2.3×10^{-4}		
	Cal EPA	high	2.8×10^{-4}		
PAH (BaP)	Cal EPA	low	1.1×10^{-3}	B	All cancer
	midpoint	central	44.0×10^{-3}		
	WHO	high	87.0×10^{-3}		
Reference exposure level (RfC—non-cancer endpoints)					
Toluene	Cal EPA	low	0.2	A	Neurological
	midpoint	central	0.3		
	US EPA	high	0.4		
Xylene	Cal EPA	central	0.3	C	Not available

US EPA 2001; WHO 2000; Cal EPA 2000

Uncertainty rank: A, very good level of certainty; B, good level; C: sufficient level

As the estimates in this table are based primarily on animal toxicological studies and occupational studies, they can be assumed to be generalisable to the Australian population