Health Effects and Costs of Vehicle Emissions

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Executive Summary

Air pollution has a very serious, but often under-estimated impact on the social and economic wellbeing of communities around the world. In many cities, motor vehicles are a major, and often the dominant source of this pollution.

Cancers and bronchial illnesses, stemming from inhalation of fine particles in motor vehicle exhaust, have been identified as being a health risk of great concern. Vehicle pollutants are also key constituents of photochemical smog, which has debilitating respiratory effects on the residents of many large urban areas.

Some transport fuels also contribute to a long list of highly toxic air contaminants which, although present in very low concentrations, include known carcinogens and are now suspected to have links with many "20th Century" illnesses, including higher incidence of asthma and allergies.

Numerous studies have explored the relationships that exist between human exposure to air pollutants and the cost of dealing with the health–related consequences.

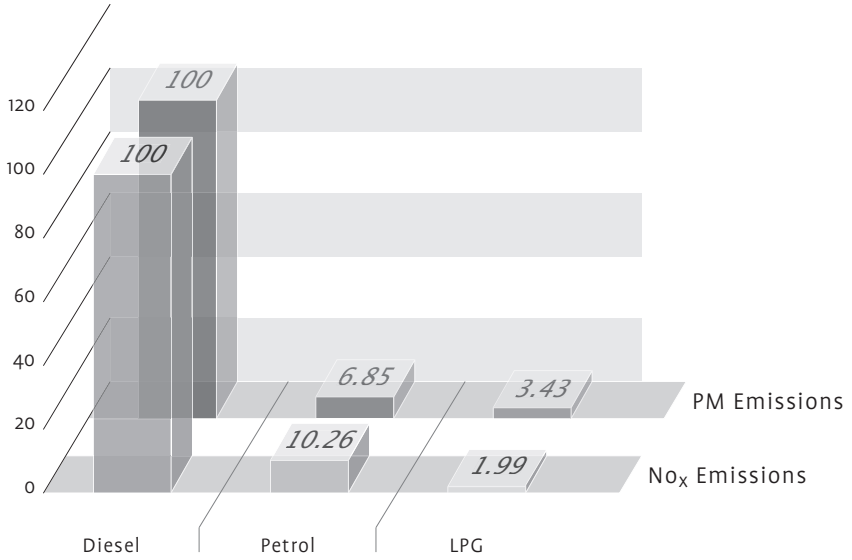
In 2000 the World Bank summarised the airborne particle problem as follows:

> *"High concentrations of suspended particulates adversely affect human health, provoking a wide range of respiratory diseases and exacerbating heart disease and other conditions. Worldwide, in 1995 the ill health caused by such pollution resulted in at least 500,000 premature deaths and 4-5 million new cases of chronic bronchitis."*

The European Union, in its wide ranging Extern-E study to quantify the economic effects of air pollution, concluded that the total social cost to EU member states was equivalent to between one and two per cent of GDP (85 to 170 billion Euros)

No single strategy will provide a solution to this pervasive problem, but policies and strategies that lead to a significantly increased uptake of cleaner fuels can greatly reduce levels of those pollutants that do most harm to humans.

Figure 1 Average Relative PM and No_X Emission Rates for Diesel, Petrol and LPG Variants of Same Vehicle Models



Of all commercially available transport fuels, diesel poses the greatest health hazard, with particle (PM) emission levels up to 50 times higher than petrol and 100 times higher than LPG.

In addition, diesel's very high emissions of oxides of nitrogen (NOx) are a major contributor to photochemical smog in many cities.

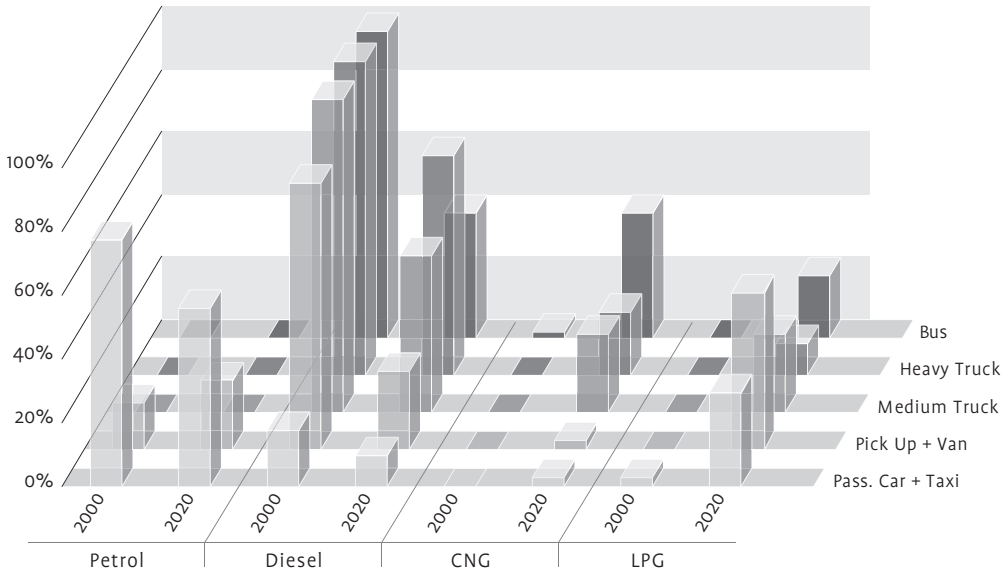
Although petrol has lower PM and NOx emissions than diesel, it releases high levels of "air toxics into the atmosphere. Given the scientific community's concerns about the effects of these substances on humans, switching from diesel to petrol is not the preferred option, especially as LPG emits relatively small amounts of air toxics compared to both petrol and diesel.

Hence, strategies that discourage the use of diesel and petrol, and encourage increased uptake of clean-burning gaseous fuels such as LPG (Autogas) and CNG, will deliver the greatest health benefits to the community.

The monetary cost of pollution-related sickness, social services, lost productivity and premature mortality has been extensively studied in many regions. Although the cost estimates vary according to local income levels and other economic factors, they almost invariably represent a significant, yet largely avoidable burden on the national economy.

A typical example of how fuel usage could be modified over a period of time is shown in the illustration below. The year 2000 fuel mix in each vehicle category is representative of many Asian cities.

Figure 2 Potential Shift from Diesel and Petrol to Gaseous Fuels over 20 Year Period



Apart from a predominance of petrol passenger cars the year 2000, fuel usage is heavily biased towards diesel, with a few LPG taxis and some CNG buses. The 2020 scenario has greatly reduced dependence on diesel, which can be achieved without adversely impacting on vehicle choice or functionality.

Translating this fuel shift into economic terms, the following table summarises health cost savings calculated on the basis of reduced exposure to particles generated by motor vehicles (additional, but smaller savings flow from reductions in other pollutants). It is also assumed that costs are given for the Asian city scenario illustrated above, and also for similar fuel use changes in hypothetical European and Australian cities. Note that the savings are US dollars per annum for each million vehicles.

Table 1 Estimated Reductions in PM-Related Health Costs for Increased Use of Gaseous Fuels

Region for City	**Cost ,Year 2000** per million vehicles	**Cost, Year 2020** per million vehicles	**Annual Health Cost Saving** per million vehicles
Asia	US$1.40 billion	US$0.24 billion	US$1.16 billion
Europe	US$2.80 billion	US$1.20 billion	US$1.60 billion
Australia	US$0.45 billion	US$0.19 billion	US$0.26 billion

The table clearly illustrates the very considerable benefits that flow from switching to cleaner fuels

Conclusions

Motor vehicle pollution has severe adverse health impacts on the community, especially for people living in urban areas or in locations close to busy roads. The resultant health care and lost productivity costs are very high, especially where there are large numbers of diesel vehicles.

For many countries, the net health costs of vehicle pollution have been estimated to exceed 2 per cent of national GDP.

The early adoption of rigorously implemented transport fuel policies, which lead to a strong uptake of cleaner gaseous fuels, can play a very significant role in reducing air pollution and its consequential harm to the community and the national economy.

Executive Summary

This report, commissioned by the World Liquid Petroleum Gas Association (WLPGA), reviews the health impacts and health costs of outdoor air pollution specifically particulates and air toxics.

The main objectives were to:

summarise the sources of ambient air pollution and the contribution of each of these sources to the overall levels of air pollutants;
summarise the health effects of particulates and air toxics;
summarise the exposure-response relationships between particulates and air toxics, and specific health effects (for example, mortality, hospital admissions); and, summarise the health costs attributable to particulates and air toxics.

Motor vehicles are important sources of both the criteria air pollutants and air toxics. Emission standards, fuel standards, types of fuel used, vehicle type mix and activity levels are important considerations when calculating the emissions from motor vehicles. Changes in fuel usage patterns and vehicle kilometres travelled will have implications for emission of pollutants. An increase in the proportion of light commercial vehicles using diesel fuel will lead to increases in emissions of fine particles and oxides of nitrogen. LPG and CNG fuels, compared to petrol and diesel fuels, have the lowest emission factor for PM10 and lower emission factors for oxides of nitrogen and the air toxics.

Concentrations of ambient air toxics are generally low and below the Air Toxics National Environment Protection Measure. Benzene, 1,3-butadiene and PAHs are known human carcinogens whereas toluene and xylenes are associated with adverse health effects. Motor vehicles are significant sources of air toxics.

Exhaust emissions for benzene and 1,3-butadiene are 85 to 100 percent lower for LPG fuel compared to petrol and diesel fuels and there are no exhaust emissions for PAHs, toluene and xylenes for LPG fuel. There are also no air toxics evaporative emissions from LPG fuel. Encouraging the use of LPG fuel can reduce motor vehicle emissions of air toxics.

Particulate matter remains a significant air pollution problem in all major cities around the world. Inhaled particulate matter induces inflammation in the lungs that results in increased respiratory symptoms, reduced lung function and changes to cardiac rhythm. The acute health effects of particulate matter are increased respiratory and cardiovascular morbidity and mortality whereas the long-term health effects of particulate matter are reduced lung function growth in children and increase risk of cancer in adults. Motor vehicles are a significant contributor to ambient concentrations of particulate matter. LPG has the lowest particulate emissions while diesel fuels have the highest particulate emissions. This is salient as the use of diesel fuels in light commercial vehicles is projected to increase substantially in the next 20 years.

A major proportion of the air pollution related health cost is attributable to fine particles and a very large component of the health costs are due to the cost of premature deaths. Hydrocarbons make a smaller contribution to the total air pollution health costs, and oxides of nitrogen and carbon monoxide make only very small contributions to the total air pollution health costs. Diesel fuel, and to a lesser extent petrol fuel, are significant contributors to ambient concentration of fine particles. The air pollution cost of diesel fuel is nearly eight times greater than the air pollution cost of petrol fuel. Reducing the ambient concentrations of fine particles will help avert the significant associated health costs. Use of LPG instead of diesel and petrol fuels could be an important strategy to reduce the ambient concentrations of fine particles and the associated health costs.

List of Abbreviations

AAQNEPM	Ambient Air Quality National Environment Protection Measure
ADR	Australian Design Rule
AHR	Airway hyperresponsiveness
APHEA	Air Pollution and Health: A European Approach
B[a]P	Benzo[a]pyrene
BSP	Back scatter particles
BTRE	Bureau of Transport and Regional Economics
CAAA	Clean Air Act Amendments
CI	Confidence interval
CNG	Compressed natural gas
COI	Cost of illness
CO	Carbon monoxide
COPD	Chronic obstructive pulmonary disease
EPA	Environment Protection Authority
EPHC	Environment Protection and Heritage Council
FEF75	Forced expiratory flow at 75 percent of forced vital capacity
FEV1	Forced expiratory volume in the first second
FVC	Forced vital capacity
g/km	Gram/kilometre
gm	Gram
HC	Hydrocarbon
HI	Hazard index
HQ	Hazard quotient
IARC	International Agency for Cancer and Research
Kt/yr	Kilotonne/year
LCV	Light commercial vehicle
LOAEL	Lowest observed adverse effect level
LPG	Liquefied petroleum gas
MEF25-75	Mid expiratory flow between 25 to 75 percent of forced vital capacity
mg	Milligram
MIL	Monitoring investigation level
MMEF	Maximum mid expiratory flow
MRAD	Minor restricted activity day
MRL	Minimal risk level
MTBE	Methyl tertiary butyl ether

MVEC	Motor Vehicle Environment Council
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
ng	Nanogram
NH&MRC	National Health and Medical Research Council
NNMAPS	National Morbidity Mortality and Air Pollution Study
NO	Nitric oxide
NO2	Nitrogen dioxide
NOx	Oxides of nitrogen
NOAEL	No observed adverse effect level
NSW EPA	New South Wales Environment Protection Authority
O3	Ozone
PAH	Polycyclic aromatic hydrocarbon
PEFR	Peak expiratory flow rate
PM	Particulate matter
PM2.5	Particulate matter less than 2.5 microns in diameter
PM10	Particulate matter less than 10 microns in diameter
POM	Polycyclic organic matter
PPB	Parts per billion
PPM	Parts per million
RAD	Restricted activity day
REL	Reference exposure level
RfC	Reference concentration
RR	Relative risk
SD	Standard deviation
SO2	Sulphur dioxide
SVOC	Semivolatile organic compound
TSP	Total suspended particulate
µg	Microgram
URF	Unit risk factor
US EPA	United States Environment Protection Agency
VKT	Vehicle kilometres travelled
VOC	Volatile organic compound
WHO	World Health Organization
WTP	Willingness to pay

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01.
Scope of the Study

This study has been commissioned by the World Liquid Petroleum Gas Association (WLPGA) to review the health impacts and health costs of outdoor air pollution specifically particulates (as measured by particulate matter less than 10 microns in aerodynamic diameter [PM10] and by particulate matter less than 2.5 microns in aerodynamic diameter [PM2.5] and air toxics (also known as hazardous air pollutants or HAPs).

The particular objectives are as follows:

1. Summarise the health effects of particulates and air toxics;
2. Summarise the exposure-response relationships between particulates and air toxics, and specific health effects (for example, mortality, hospital admissions);
3. Summarise the health costs attributable to particulates and air toxics; and
4. Summarise the sources of ambient air pollution and the contribution of each of these sources to the overall levels of air pollutants (for example, transport sector, industry).
5. Summarise the above information by four regions of the world - North America (United States of America and Canada), Europe and the United Kingdom, Asia and Australasia (Australia and new Zealand).

For each of the above objectives, information will be derived from published reports and reviews (both systematic and non-systematic reviews). These reports and reviews (including meta analyses) often use a range of study selection criteria to ensure the primary studies used are of high quality and that the results of these studies are epidemiologically robust. Although, there will be instances where information on particular issues will not be available, a review of the primary literature does not form the scope of the study.

An extensive search of the electronic scientific and medical databases for any published reviews and meta-analyses, for example, Medline and PubMed, as well as a search of relevant internet sites for reports, for example, EPHC, USEPA, HEI, COMEAP, CONCAWE, ExternE, AirImpacts.

Following the literature review, hard copies of all relevant reviews and reports will be obtained. The information will then be summarised and reported in both text and tabular form. Information on health effects and exposure-response relationships will be presented by region and air pollutant. Information relating to the other objectives will be presented by region. However, the layout of the presentation may vary according to the type and range of information that is available. In any case, the key conclusions will be presented as dot points.

The WLPGA will summarise existing mitigating policies and practices with regards to ambient particulates and air toxics.

02.

Sources and health costs of motor vehicle air pollution

This section of the report will discuss the sources of emission of the common air pollutants and the air toxics. Emphasis will be placed on particulates and the air toxics.

2.1 Motor vehicle related air pollution

Motor vehicles make a major contribution to urban air pollution (ASEC 2001). These include the common, or criteria, air pollutants as well as the air toxics.

A review of current air emissions inventory sources for Australian capital cities is summarised in Table 2.1 (Coffey Geosciences 2003). These results indicate that motor vehicle emissions make significant contributions to urban concentrations of carbon monoxide and particulates but not to sulphur dioxide or lead. In the cases of nitrogen dioxide and ozone, the concentrations of these pollutants in Australian capital cities are strongly influenced by motor vehicle emissions.

Motor vehicles are the dominant source of carbon monoxide emissions to urban air with recent emissions estimates indicating a contribution of 68 to 86 percent of the total for capital cities. Recent catalytic converters designed to remove hydrocarbon emissions from petrol motor vehicles are very effective in controlling carbon monoxide emissions and, as a result, carbon monoxide emissions for recent vintage vehicles are typically substantially lower than called for in the existing performance specifications. This effect is considered to be likely to have contributed to the gradual reduction in carbon monoxide concentrations recorded in capital cities over the period 1998 to 2001. It is clear from these data that carbon monoxide is not a problem in Australian metropolitan airsheds and is unlikely to be so in the foreseeable future (Coffey Geosciences 2003).

Nitrogen dioxide (NO_2) is a one of many species of oxides of nitrogen encountered in urban ambient air. Anthropogenic sources of oxides of nitrogen include industrial emissions associated with operation of burners, domestic fuel consumption and motor vehicles. Anthropogenic emissions of oxides of nitrogen are predominantly nitric oxide with typically only 5 to 10 percent nitrogen dioxide.

Table 2.1 Air Emissions Estimates – Australian Capital Cities

Parameter	Sydney[A]	Melbourne[B]	BrisbaneC	PerthD	Adelaide[E]	Canberra[F]	Hobart[F]	Darwin[F]
Total Anthropogenic Emissions (t/yr)								
Carbon monoxide	591,654	680,000	617,530	283,356	170,000	38,755	22,004	12,515
Oxides of nitrogen	89,232	86,000	97,385	67,830	30,000	5,845	3,319	1,887
Hydrocarbons	136,037	170,000	378,266	71,947	40,000	8,911	5,059	2,877
PM10	15,042	21,000	23,906	10,694	11,000	985	559	318
$PM_{2.5}$	7,075	11,400	10,462	4,834	4,620	463	262	150
Benzene	3,441	4,500	1,395	1,163	930	279	167	74
1,3-butadiene	592	600	240	200	160	48	29	13
Formaldehyde	3,663	1,300	1,485	1,238	990	297	178	79
Acetaldehyde	2,183	1,853	885	738	590	177	106	47
Percentage from Motor Vehicle Emissions								
Carbon monoxide	86.6%	82.4%	67.6%	80.0%	85.0%	86.6%	86.6%	86.6%
Oxides of nitrogen	72.3%	62.8%	62.2%	41.0%	60.0%	72.3%	72.3%	72.3%
Hydrocarbons	42.1%	37.1%	22.0%	31.0%	44.0%	42.1%	42.1%	42.1%
PM10	17.6%	16.7%	9.4%	13.0%	5.0%	17.6%	17.5%	17.6%
$PM_{2.5}$[G]	29.9%	24.6%	17.2%	23.0%	9.5%	29.8%	29.8%	30.0%
Benzene[H]	74.0%	77.8%	74.0%	73.9%	74.0%	73.8%	74.3%	74.3%
1,3-Butadiene[H]	81.3%	75.0%	81.3%	81.5%	81.3%	81.3%	79.3%	76.9%
Formaldehyde[H]	47.0%	63.1%	47.0%	46.9%	47.0%	47.1%	47.2%	46.8%
Acetaldehyde[H]	23.0%	23.0%	23.1%	23.0%	23.1%	23.2%	22.6%	23.4%

Notes

A 2000 data, projected from the 1992 Metropolitan Air Quality Study. Data supplied by NSW EPA (2003a).

B 1995-1996 Port Phillip Region data (EPA Victoria, 1998). Acetaldehyde based on Adelaide estimates, adjusted for population. PM_{10} and $PM_{2.5}$ emissions from road dust are excluded consistent with inventories for other cities.

C 2000 Southeast Queensland Region data (EPA Queensland, 2002).

D 1998-1999 data (Farrar et al, 2000).

E 1998-1999 aggregate data, plus 1999-2000 industry data (SA EPA, 2002).

F CO, NOx, hydrocarbons and PM10 based on Sydney estimates, adjusted based on population proportion in 2000.

G $PM_{2.5}$ based on PM_{10}:$PM_{2.5}$ ratio in Melbourne.

H Air toxic estimates based on Adelaide estimates, adjusted based on population proportion.

Source: (Coffey Geosciences 2003)

Ozone is mainly formed by chemical interaction between oxides of nitrogen and reactive organic compounds under the influence of sunlight. Ozone formation occurs more readily on warm sunny days and it is therefore predominantly a summer problem. Background concentrations of ozone in areas where human sources are largely absent are low. Ozone formation in ambient air under suitable conditions can proceed to a limit that is controlled by the concentration of oxides of nitrogen in the air. The rate at which the chemical reactions take place is affected by the concentration of reactive organic compounds, increasing as the concentration of these compounds increases. Thus ozone concentration is influenced by the concentrations of oxides of nitrogen and reactive organic compounds. Motor vehicle emissions are significant sources of both oxides of nitrogen and reactive organic compounds.

Motor vehicles are a significant contributor to urban particulate PM_{10} emissions to the atmosphere and are estimated to make up about 9 to 17 percent of emissions from all sources excluding dust. Motor vehicle emissions of particulates occur largely from heavy vehicles. Coffey Geosciences (Coffey Geosciences 2000) estimated that diesel vehicles accounted for approximately 60 percent of road vehicle emissions of PM_{10} in 2000.

$PM_{2.5}$ particulate emissions (particulate emissions with a characteristic dimension smaller than 2.5 microns) form a high proportion of motor vehicle particulate emissions. Motor vehicle emissions of $PM_{2.5}$ were taken as accounting for 80 percent of PM_{10} emissions in the development of 1995 emissions inventory for Melbourne. Motor vehicle emissions accounted for 25 percent of total $PM_{2.5}$ emissions (excluding road dust) in the Melbourne inventory (Coffey Geosciences 2003).

2.2 Factors impacting on motor vehicle emissions

Some of the major factors impacting motor vehicle emissions are:

2.2.1 Design standards

Vehicle emission standards are critical in influencing air quality. The vehicle emissions standards are given legal effect as Australian Design Rules (ADRs) under the Federal

Motor Vehicle Standards Act 1989. This Act is administered by the Department of Transport and Regional Services (DOTARS) and the standards are developed in a consultative process managed by the Motor Vehicle Environment Committee (MVEC).

Current ADRs are based upon European Standards, with United States standards for heavy vehicles adopted in relation to heavy vehicles fuelled by petrol. The current European standards do not cover these vehicles. The European standards in the form of United Nations Economic Commission for Europe (UNECE) standards are commonly referred to as the *Euro* standards.

There are currently five ADRs that set limits on vehicle emissions (Coffey Geosciences 2003):

- ADR30/01 which imposes limits on smoke emissions from diesel vehicles by adopting UN ECE R24/03 and allows US 94 smoke standards as an alternative;
- ADR79/00 implements Euro 2 standards for diesel passenger and light commercial vehicles (gross vehicle mass less than 3.5 tonnes) from 2002 for new models and from 2003 for all models and for petrol vehicles from 2003 (new models) or 2004 (all models);
- ADR79/01 implements Euro 3 standards for petrol passenger and light commercial vehicles from 2005 (new models) or 2006 (all models) and implements Euro 4 standards for diesel light vehicles from 2006 (new models) or 2007 (all models);
- ADR80/00 implements Euro 3 standards for heavy diesel vehicles (greater than 3.5 tonnes gross vehicle mass) from 2002 (new models) or 2003 (all models); and,
- ADR80/01 implements Euro 4 standards for heavy diesel vehicles from 2005 (new models) or 2006 (new models).

In the case of heavy duty diesel vehicles, United States and Euro emission standards are offered as alternatives, whereas, for heavy duty petrol vehicle only United States emission standards are offered. The emissions standards for petrol and LPG/CNG passenger vehicles are presented in Table 2.2 and those for heavy diesel vehicles are presented in Table 2.3. It is worth noting that emission standards for LPG/CNG vehicles are similar to petrol emission standards.

Table 2.2 ADR emission standards for petrol, LPG and CNG passenger cars

Standard	Application Date	Equivalent To	Exhaust Emission Limits			Evaporative Limit
			CO g/km	HC g/km	NOx g/km	HC g/test
Uncontrolled[A]	Pre-1972		39.9	3.7	2.1	
ADR 27A	1976	US 1973[B]	24.2	2.1	1.9	2[C]
ADR 27B	1978		22[D]	1.9[4]	1.9	6[E]
ADR 27C	1981[F]		18.6	1.75	1.9	2
ADR 37/00	1986	US 1975[G]	9.3	0.93	1.93	2
ADR 37/01	1997/99	US 1981	2.1	0.25	0.62	2
ADR 79/00	2003/04	Euro 2	2.2	$HC+NO_x$ 0.50		2
ADR 79/01	2005/06	Euro 3	2.3	0.2	0.15	2

Notes

A Emission levels from NSW EPA in MVEC 1997.

B CVS-C test procedure (cold start).

C Canister test procedure.

D Optional limits if no durability testing to establish deterioration factors.

E SHED test procedure adopted.

F 6 applied in NSW only.

G CVS-CH test procedure (weighted cold/hot start) adopted.

Source: (Coffey Geosciences 2003)

Table 2.3 ADR emission standards for heavy diesel vehicles

Standard	Application Date	CO (g/kWh)	HC (g/kWh)	NO_x (g/kWh)	PM (g/kWh)
ADR70/00[A]	1997/99	4.5	1.1	8.0	0.36
ADR80/00 (Euro 3)	2002/03	2.1	0.66	5.0	0.10
ADR80/01 (Euro 4)	2006/07	1.5	0.46	3.5	0.03

Notes

A ADR70/00 Allowed 3 alternate sets of standards – Euro 1 listed for illustration

Source: (Coffey Geosciences 2003)

2.2.2 Activity levels

Activity levels (passenger kilometres and tonne kilometres) are determined by the demand for transport services. Although emissions rates (per kilometre travelled) from motor vehicles may be falling, increasing use of motor vehicles and hence vehicle kilometres travelled (VKTs) may offset any gains made. Importantly, there is rapid growth of light urban commercial vehicles of about 6 percent per year (NEPC 1999).

In 2000, for the capital cities of Australia, the total VKT was 113,440 million. Of this figure, 91,240 VKTs were attributable to passenger vehicles, 15,880 to light commercial vehicles, 3,730 to rigid trucks, 1,020 to articulated trucks, 790 to buses and 790 to motorcycles (Coffey Geosciences 2003). The total VKTs was estimated to increase from 113,440 million in 2000 to 165,560 million in 2020, an increase of 46 percent (Coffey Geosciences 2003). The increase by motor vehicle category is shown in the following table (Table 2.4). The greatest percentage increases are for articulated trucks and light commercial vehicles. In absolute terms (number of motor vehicles) however, the greatest increase is for passenger vehicles.

Table 2.4 VKTs by motor vehicle category for the years 2000 and 2020

Vehicle category	2000 VKT (million)	2020 VKT (million)	% increase
Passenger	91,240	123,770	35.7
Light commercial	15,880	32,930	107.4
Rigid truck	3,730	4,690	25.7
Articulated truck	1,020	2,230	118.6
Bus	790	1,030	30.4
Motorcycle	790	910	15.2
Total	113,440	165,560	45.9

Source: (Coffey Geosciences 2003)

2.2.3 Vehicle efficiency

Vehicle efficiency is determined by rated fuel economy; actual on-road economy; in-service deterioration; and, congestion on the road system.

2.2.4 Mix of vehicles

The ages and modal split of the vehicles affects fuel economy and emissions. The modal split in passenger travel is moving towards cars, and in freight, the movement is towards light commercial vehicles. Although larger trucks and buses may emit more pollutants per VKT compared to passenger vehicles, the very large numbers of passenger cars compared to trucks and buses and their reliance on petrol engines ensures that they are the major contributors to carbon monoxide, hydrocarbons and oxides of nitrogen. Commercial vehicles are important contributors of oxides of nitrogen and particulates.

Table 2.5 presents percentage of emissions by vehicle category (Coffey Geosciences 2003). Compared to 2000, passenger vehicles will contribute smaller proportions of all four pollutant emissions in 2020, and particularly so for oxides of nitrogen and particulate matter. Light commercial vehicles, will increase their contributions to the emissions of hydrocarbons and particulate matter, whereas trucks will reduce their contribution to hydrocarbons and particles.

Table 2.5 Percentage of emissions by vehicle category

Vehicle Category	**Year 2000**				**Year 2010**				**Year 2020**			
	CO	**NO_x**	**HC***	**PM**	**CO**	**NO_x**	**HC***	**PM**	**CO**	**NO_x**	**HC***	**PM**
Passenger	82.6	60.8	71.3	37.0	79.3	54.2	67.4	36.9	75.5	35.8	68.4	20.4
Light commercial	13.9	12.2	11.5	12.3	14.2	13.8	14.0	17.2	12.7	13.5	18.4	33.0
Rigid truck	1.7	14.2	12.4	35.2	2.5	15.7	12.2	31.7	2.2	23.5	4.9	16.3
Articulated truck	0.5	6.9	1.2	8.6	0.8	10.7	1.7	8.3	2.0	21.2	3.1	23.7
Bus	0.3	5.7	2.2	6.3	0.4	5.2	1.9	4.6	0.4	5.3	0.8	2.5
Motorcycle	1.0	0.2	1.4	0.7	2.8	0.4	2.8	1.4	7.2	0.8	4.4	4.1
Total	100	100	100	100	100	100	100	100	100	100	100	100

Source: (Coffey Geosciences 2003)

2.2.5 Fuels used

Fuels vary in emission characteristics. For example, diesel fuels contribute a disproportionately high percentage of emissions in relation to their VKTs.

Therefore it is important to have information on the types of fuels in use, emissions factors for the different fuel types (petrol, diesel, LPG) and their pattern of usage within the transport sector.

There are now national fuel quality standards. Improving fuel quality allows the production of motor vehicles with improved efficiency and reduced tailpipe emissions. The Fuel Quality Standards Act 2000 provides the framework for the establishment of national fuel standards for automotive use and came into effect 1 January 2002 and will become progressively more stringent until 2006. In particular, the concentration of sulphur in petrol will be limited to 150ppm in petrol by 2005 and to 50ppm in diesel by 2006. The aim of the Act is to regulate the quality of fuel supplied in Australia in order to:

- Reduce the level of pollutants and emissions arising from the use of fuel that may cause environmental and health problems;
- Facilitate the adoption of better engine technology and emission control technology; and,
- Allow more effective operation of engines.

The legislation provides the framework for harmonizing Australian fuel quality standards with international standards. The first set of standards, for both petrol and diesel, came into effect on 1 January 2002. The standards bear similarities to the fuel standards adopted by the European Commission to accompany staged implementation of vehicle emissions standards. Australian and European fuel quality standards are presented in Table 2.6.

Table 2.6 Australian and European fuel quality standards

Parameter	Australia (2005/6)	Europe (2005)	Europe (2009)
Petrol			
Sulphur (ppm)	150 maximum	50 maximum	10 maximum
Olefins (% v/v)	18 maximum	18 maximum	18 maximum
Aromatics (% v/v)	42 pool average 45 maximum	35 maximum	35 maximum
Benzene (% v/v)	1 maximum	1 maximum	1 maximum
MTBE	1%*	15% maximum	15% maximum
Diesel			
Sulphur (ppm)	50 maximum	50 maximum	10 maximum
Polyaromatic hydrocarbons (%)	11 maximum	11 maximum	11 maximum
Density (kg/m^3)	820 to 850	845 maximum	845 maximum

Note

* A 1% limit is also referred to as an 'effective ban'. It allows for trace residual elements to be detectable, but is too low to be used effectively as an oxygenate in petrol.

Source: (Coffey Geosciences 2003)

A recent report (Coffey Geosciences 2003) provides some information on the fuel types being currently used in the transport sector and projections for 2020 (Table 2.7).

Table 2.7 Fuel usage distribution by VKT in 2000 and 2020

Vehicle Category	Fuel Usage Distribution by VKT 2000 Vehicles			Fuel Usage Distribution by VKT 2020 Vehicles		
	Petrol	Diesel	LPG/CNG	Petrol	Diesel	LPG/CNG
Passenger	95.3	2.5	2.2	88.8	5.0	6.2
Light commercial	72.7	23.0	4.3	41.0	45.0	12.3
Rigid trucks	0	100	0	0	100	0
Articulated trucks	0	100	0	0	100	0
Buses	0	99	1	0	95	5
Motorcycles	100	0	0	100	0	0

Source: (Coffey Geosciences 2003)

Coffey Geosciences (Coffey Geosciences 2003) estimated that, in 2000, petrol passenger vehicles contributed to 95.3 percent, diesel to 2.5 percent and LPG/CNG to 2.2 percent of all passenger car VKTs. For light commercial vehicles, these percentages were 72.7 percent, 23.0 percent and 4.3 percent respectively. Projections for 2020, demonstrated only small increases in the uptake of LPG/CNG for passenger vehicles, light commercial vehicles and buses (uptake at 6.2 percent, 12.3 percent and 5 percent for passenger vehicles, light commercial vehicles and buses respectively). It was also estimated that diesel use will significantly increase in light commercial vehicles. This will have implications for emissions of fine particles and oxides of nitrogen.

Therefore, motor vehicle emission control and fuel quality standards are important management strategies in controlling emissions from motor vehicles. With the implementation of new emission standards, particulates in urban areas from diesel vehicles will be about 26 percent less in 2015 compared to 2000 despite an expected growth of 60 percent in VKT (ASEC 2001). There will be decreases in oxides of nitrogen due to tighter emission standards for both petrol vehicles and heavy diesel vehicles. Decreases in hydrocarbons will be mainly due to more stringent emission standards for petrol vehicles (Coffey Geosciences 2003).

2.3 Emission factors for different fuel types

Estimating emissions from motor vehicles depend on a number of factors (NPI 2000):

- Traffic count data or spatially distributed VKT in an airshed;
- Relative VKT by motor vehicle type and road type in the airshed;
- VKT by motor vehicle and fuel type and year of manufacture of the motor vehicle;
- The number of vehicles by motor vehicle type, fuel type and year of manufacture;
- The average consumption rate of each motor vehicle/fuel type;
- The sulphur and lead contents of fuels and the relative vapour pressure of petrol used in an airshed; and'
- The average temperature an average daily maximum and minimum temperatures in an airshed.

2.3.1 Emission factors for PM_{10}, carbon monoxide, VOCs and oxides of nitrogen

Emission factors for PM_{10} and for carbon monoxide, VOCs and oxides of nitrogen published by the National Pollutant Inventory (NPI 2000) are presented in Tables 2.8 and 2.9. These tables present emission factors by motor vehicle type, fuel type and road type. Table 2.8 also presents emission factors for tyre wear and brake wear.

It is important to note that the lowest emissions for any of the motor vehicle categories are for LPG/CNG. Diesel fuels have the highest emissions of the three fuels. This is salient as the use of diesel fuels in light commercial vehicles is projected to increase (107 percent increase in VKT from 2000 to 2020) (Coffey Geosciences 2003).

Table 2.8 Emission factors for PM_{10} by motor vehicle, fuel and road type

Vehicle Type	Fuel	Emission Factor (g km^{-1})		
		Arterial	Freeway	Residential
Exhaust				
Passenger	Petrol	0.00932	0.00513	0.00932
	Diesel	0.148	0.0813	0.148
	LPG	0.00329	0.00181	0.00329
Light commercial	Petrol	0.0118	0.00649	0.0118
	Diesel	0.222	0.122	0.222
	LPG	0.00493	0.00271	0.00493
Heavy duty [a]	Petrol	0.120	0.0660	0.120
	Diesel	0.584	0.321	0.584
	LPG	0.0278	0.0153	0.0278
Bus	Petrol [b]	0.666	0.366	0.666
	Diesel[b]	0.666	0.366	0.666
	LPG	0.0317	0.0174	0.0317
Motorcycle	Petrol	0.0124	0.00684	0.0124
Tyre wear				
Passenger		0.00497	0.00497	0.00497
Light commercial		0.00497	0.00497	0.00497
Heavy duty		0.00746	0.00746	0.00746
Bus		0.00497	0.00497	0.00497
Motorcycle		0.00249	0.00249	0.00249
Brake wear				
All vehicles		0.00808	0.00808	0.00808

Notes

[a] Includes articulated, non-freight carrying and rigid truck.

[b] For buses, petrol and diesel emission factors are considered to be the same.

Source: (NPI 2000)

For both passenger cars and light commercial vehicles, LPG emissions for PM_{10} are between 35 percent to 42 percent of petrol emissions, and about 3 percent of diesel fuel. For heavy duty vehicles, LPG emissions are 23 percent and 5 percent of petrol and diesel fuels respectively, and for buses LPG emissions are about 5 percent of petrol and diesel fuels. The increasing use of LPG will obviously have an impact on particulate emissions and especially the very fine particles.

Emissions of carbon monoxide, VOCs (exhaust) and oxides of nitrogen are relatively similar for the three fuel types, although emissions of oxides of nitrogen are generally lower for LPG compared to petrol or diesel.

Table 2.9 Emission factors for carbon monoxide, oxides of nitrogen and VOCs by motor vehicle, fuel and road type

Vehicle type	Fuel	Emission Factor (g km^{-1})		
		Arterial	Freeway	Residential
CO				
Passenger	Petrol	19.3	18.8	22.3
	Diesel	0.637	0.516	1.13
	LPG	24.5	24.0	27.9
Light commercial	Petrol	17.2	13.9	30.6
	Diesel	0.810	0.656	1.44
	LPG	19.1	15.4	34.0
Heavy duty	Petrol	53.7	43.4	95.6
	Diesel	4.42	3.58	7.87
	LPG	59.7	48.3	106
Motorcycle	Petrol	9.04	7.32	16.1
NO_x				
Passenger	Petrol	1.58	1.98	1.78
	Diesel	0.785	1.33	1.02
	LPG	1.10	1.37	1.23
Light commercial	Petrol	1.32	2.24	1.73
	Diesel	1.03	1.75	1.35
	LPG	0.878	1.49	1.15
Heavy duty	Petrol	3.08	5.21	4.02
	Diesel	6.69	11.3	8.73
	LPG	2.04	3.46	2.66
Motorcycle	Petrol	0.428	0.724	0.558
VOCs (exhaust)				
Passenger	Petrol	1.26	1.24	1.45
	Diesel	0.331	0.310	0.513
	LPG	1.53	1.51	1.73
Light commercial	Petrol	1.64	1.53	2.53
	Diesel	0.554	0.517	0.857
	LPG	1.75	1.63	2.70
Heavy duty	Petrol	3.08	2.88	4.77
	Diesel	1.01	0.941	1.56
	LPG	3.29	3.07	5.09
Motorcycle	Petrol	1.23	1.15	1.90
VOCs (evaporative)				
Passenger	Petrol	0.535	0.241	0.535
	LPG	1.07	0.483	1.07
Light commercial	Petrol	0.586	0.275	0.586
	LPG	1.17	0.550	1.17
Heavy duty	Petrol	2.91	2.15	2.91
	LPG	5.81	4.29	5.81
Motorcycle	Petrol	0.803[a]	0.803	0.803

Source: (NPI 2000)

As a comparison, emission factors (g/km) for the various types of motor vehicles and fuels have also been estimated by Coffey Geosciences (Coffey Geosciences 2003). An adaptation is presented in Table 2.10.

Table 2.10 Emission factors by motor vehicle type and fuel type

Emission Factors (g/km)	CO	NOx	HC	PM
Petrol (Euro 3)				
Passenger	1.06	0.15	0.2	0.0007
Light commercial	0.76	0.18	0.25	0.0009
Rigid truck	25.1	3.36	1.59	0.235
Articulated truck	25.1	3.36	1.59	0.235
Buses	17.6	2.35	1.11	0.165
Motorcycle	18.4	0.53	2.9	0.064
Diesel (Euro 4)				
Passenger	0.16	0.27	0.05	0.025
Light commercial	0.29	0.33	0.06	0.04
Rigid truck	0.6	3.13	0.25	0.024
Articulated truck	1.61	6.71	0.64	0.064
Buses	0.6	3.13	0.25	0.024
Motorcycle	0	0	0	0
LPG/CNG (same as for petrol)				
Passenger	1.06	0.15	0.2	0.0007
Light commercial	0.76	0.18	0.25	0.0009
Rigid truck	25.1	3.36	1.59	0.235
Articulated truck	25.1	3.36	1.59	0.235
Buses	17.6	2.35	1.11	0.165
Motorcycle	18.4	0.53	2.9	0.064

Note

* Only tailpipe emissions considered for hydrocarbons

Source: (Coffey Geosciences 2003)

2.3.2 Exhaust emission factors for air toxics

Exhaust emission factors for arterial roads (major roads with moderate average speeds [30 km/hr] and moderate congestion levels [typically 20 percent idle time]) for five air toxics are presented in Table 2.11. These emission factors have been derived from published data using speciation factors (multiplying the emission factor for total VOCs by the speciated weight fractions for those species) (NPI 2000). LPG/CNG has lower emission factors for all the five air toxics. Of importance is the fact that the emission factors are much less for LPG/CNG for benzene, 1,3-butadiene and PAHs, which are all designated as human carcinogens.

Table 2.11 Exhaust emission factors for arterial roads for benzene, 1,3-butadiene, PAHs, toluene and xylenes

Air toxics	Passenger cars	Light commercial vehicles	Heavy duty vehicles
Benzene			
Petrol	0.08291	0.10791	0.20266
Diesel	0.00334	0.00560	0.01020
LPG/CNG	0.00001	0.00002	0.00003
1,3-butadiene			
Petrol	0.00818	0.01064	0.01999
Diesel	0.00038	0.00064	0.00116
LPG/CNG	0.00008	0.00010	0.00018
PAHs			
Petrol	0.00273	0.00356	0.00668
Diesel	0.00221	0.00370	0.00674
LPG/CNG	0.00000	0.00000	0.00000
Toluene			
Petrol	0.13230	0.17220	0.32340
Diesel	0.00487	0.00814	0.01485
LPG/CNG	0.00000	0.00000	0.00000
Xylenes			
Petrol	0.09563	0.12448	0.23377
Diesel	0.00387	0.00648	0.01182
LPG/CNG	0.00000	0.00000	0.00000

Source: (NPI 2000)

2.4 Health costs of motor vehicle emissions

Coffey Geosciences (Coffey Geosciences 2003) used emission data to calculate changes in ambient concentrations air pollutants for various emission and fuel quality scenarios.

Reductions in the rate of emissions will result in improvement in air quality. The magnitude of the resulting change in air quality change is affected by:

- The meteorological conditions within the urban airshed affected
- The magnitude of the change in emissions compared with background emission rates
- The time of the changes in emissions in relation of the temporal pattern of other sources
- Atmospheric chemical processes that act to alter the chemical character of emitted pollutants

Airshed modelling can be carried out to take account of the above factors but this is a complex task outside the scope of the present study. However this is a complex task, and an assessment of air quality impact can be made using the following simplifying assumptions (Coffey Geosciences 2003):

- The change in average concentration of particular air quality parameters above background is assumed to be proportional to the reduction in emission rate for that parameter as a fraction of the total emissions. For example, if the average concentration of carbon monoxide in an area is 5 ppm compared with a background concentration of 1 ppm (which is the average concentration which would be present if there were no emissions) and air emission control measures are put in place to reduce total air emissions by 25 percent then the average concentration of carbon monoxide could be expected to reduce by 1 ppm (25 percent of the level by which the average concentration exceeds the background concentration)
- Background concentration of air quality parameters is assessed on the basis of observations in areas where emissions from human sources are very low or taken as the lower five percentile of measurements in an area
- Total emissions within a city and the proportion attributable to road vehicles are assessed based on information obtained

from published reports or provided from the State environmental agencies

- In the case of parameters which are influenced by chemical processes, concentrations can be assessed on the basis of changes in a single precursor emission. In the cases of ozone and nitrogen dioxide it is assumed that changes in concentration are linearly related to emissions of oxides of nitrogen. This simplifying assumption can result in overestimation of the influence of emission changes

Assessment of the background concentration of air pollutants is important, as it is the difference between average concentration and the background concentration that is a measure of the influence of air emissions. The assessment of background concentration is uncertain due to the persistent influence of anthropogenic emissions on air quality. As a result the adopted background values may be too large resulting in underassessment of the beneficial impacts of air emission reductions (Coffey Geosciences 2003).

Table 2.12 presents the background emissions information for the eight Australian capital cities (Coffey Geosciences 2003). The emissions data are for total emissions for the four air pollutants and also for that attributable to motor vehicles.

Table 2.12 Background emissions information

			Emissions Inventory Data For City								
	VKT	Pop'n	Year	CO		NO_x		HC		PM_{10}	
	2000	2015		Total	Veh	Total	Veh	Total	Veh	Total	Veh
City	(Mkm)	('000)		(kt/y)	(kt/y)	(kt/y)	(kt/y)	(kt/y)	(kt/y)	(kt/y)	(kt/y)
Sydney[A]	36900	4729	2000	591.7	512.5	89.2	64.5	136.0	57.3	15.0	2.6
Melbourne[B]	33340	3970	1996	680.0	560.0	56.0	54.0	170.0	63.0	86.0	3.5
Brisbane[C]	14470	1952	2000	617.5	417.3	97.4	60.6	378.3	83.2	23.9	2.2
Perth[C]	12890	1627	1999	283.4	226.7	67.8	27.8	71.9	22.3	10.7	1.4
Adelaide[D]	9820	1186	1999	170.0	144.5	30.0	18.0	40.0	17.6	11.0	0.6
Canberra[E]	3463	310	2000	38.8	33.6	5.8	4.2	8.9	3.8	1.0	0.2
Hobart[E]	1735	176	2000	22.0	19.1	3.3	2.4	5.1	2.1	0.6	0.1
Darwin[E]	836	100	2000	12.5	10.8	1.9	1.4	2.9	1.2	0.3	0.1

Notes

A Sydney inventory data provided by NSW EPA – Based on extrapolation of 1996 MAQS inventory

B Melbourne inventory based on 1996 Port Phillip Bay inventory

C Farrer et al (2000) Perth Air Emissions Inventory Update

E EPA South Australia (2002) South Australia NPI Summary Report

F Extrapolated from Sydney scaled by population

Source: (Coffey Geosciences 2003)

Table 2.13 presents data on background air concentration for each of the eight capital cities as well the annual average concentration (Coffey Geosciences 2003).

Table 2.13 Background air quality information

City	VKT	Pop'n	Air Quality Data For City								
			Year	CO		NO_2		Ozone (O_3)		PM_{10}	
	2000	2015		B'gnd	Ave	B'gnd	Ave	B'gnd	Ave	B'gnd	Ave
	(Mkm)	('000)		(ppm)	(ppm)	(ppm)	(ppm)	(ppm)	(ppm)	(ppm)	(ppm)
Sydney	31305	4729	2000	0.1	0.4	0.005	0.011	0.005	0.014	8	16
Melbourne	30313	3970	2000	0.1	0.49	0.005	0.013	0.005	0.011	8	16
Brisbane	14929	1952	2000	0.1	0.36	0.005	0.007	0.005	0.016	8	17
Perth	11053	1627	2000	0.1	0.5	0.005	0.008	0.005	0.02	9	16
Adelaide	7866	1186	2000	0.1	1.86	0.005	0.011	0.005	0.017	9	16
Canberra	2451	310	2000	0.1	0.4	0.005	0.007	0.005	0.016	9	16
Hobart	1665	176	2000	0.1	0.5	0.005	0.007	0.005	0.016	9	16
Darwin	802	100	2000	0.1	0.4	0.005	0.007	0.005	0.016	9	16

Notes

Air quality data from draft National Ambient Air Quality Status and Trends Report 2001 (Environment Australia)

Background concentration for CO taken as 0.1ppm (see NEPC, 1997)

Background concentration for NO_2 taken as 0.001ppm, lower limit of monthly average values

Background concentration for O_3 taken as 0.001ppm, one quarter of the typical five percentile daily maximum

Background PM_{10} concentration taken as five percentile value in National Ambient Air Quality Status and Trends Report 2001 (Environment Australia)

Source: (Coffey Geosciences 2003)

Then using published dose-response relationships for various health outcomes, incidence of air pollution related health outcomes and cost per case for mortality and morbidity, they estimated the average health cost per tonne of emission for the eight major Australian cities (Table 2.14).

Table 2.14 Average cost per tonne of emission for eight Australian cities

Emission Type	Air Quality Impact	Health Savings ($/tonne)
Carbon monoxide	Carbon monoxide	$12.9
Oxides of Nitrogen	Nitrogen Dioxide	$58.7
Oxides of Nitrogen	Ozone	$8,500
Particulates (PM_{10})	Particulates (PM_{10})	$232,000
Hydrocarbons	Air toxic emissions	$2,200 [A]

Notes

A Health savings associated with hydrocarbon emissions based on US EPA (2003a) values.

Source: (Coffey Geosciences 2003)

2.5 Summary

- Motor vehicles are important sources of both the criteria air pollutants and air toxics
- Emission standards, fuel standards, types of fuel used, vehicle type mix and activity levels are important considerations when determining emissions from motor vehicles
- Activity levels as measured by VKT are projected to increase in the future
- Fuel usage pattern will change over time with a substantial increase in the proportion of light commercial vehicles using diesel fuel. This has implications for emissions of fine particles and oxides of nitrogen
- The proportion of vehicles using LPG/CNG is expected to increase only modestly
- LPG/CNG fuels, compared to petrol and diesel fuels, have the lowest emission factor for PM_{10}
- LPG/CNG fuels generally have lower emission factors for oxides of nitrogen compared to petrol or diesel fuels
- LPG/CNG fuels have substantially lower emission factors for air toxics compared to petrol or diesel fuels
- Health costs (per tonne of pollution) are greatest for particulate matter

2.6 References

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03.
Air Toxics

3.1 Background

The United States Environmental Protection Agency (US EPA) lists 188 air toxics that comprise a range of metals, fibres and chemicals. For the purposes of this report we will review the health effects of five air toxics - benzene, 1,3-butadiene, toluene, xylenes and PAHs (using benzo[*a*]pyrene as the marker). These five air toxics are ranked within the top seven for mass emissions (NEPC 2001).

There are many definitions for air toxics. The Australian Air Toxics Program (ATP) has defined air toxics as:

> *....gaseous, aerosol or particulate pollutants (other than the six criteria pollutants), which are present in the air in low concentrations with characteristics such as toxicity or persistence so as to be a hazard to humans, plant or animal life. The terms 'air toxics' and 'hazardous air pollutants' (HAPS) are used interchangeably.* (Environment Australia 2001)

The above definition is similar to that of the Organisation for Economic Co-operation and Development (OECD) except that the OECD definition also includes the criteria air pollutants (these are particles, photochemical oxidants as measured by ozone, nitrogen dioxide, lead, carbon monoxide and sulphur dioxide) (Environment Australia 2001).

The OECD has grouped the chemical air toxics as follows:

1. metals and metalloids (for example, cadmium, mercury, arsenic)
2. respirable mineral fibres (for example, asbestos, glass, microfibres)
3. inorganic gases (for example, fluorides, chlorine, cyanides)
4. halogenated organic compounds (for example, vinyl chloride, chlorobenzenes, dioxins).

On the other hand, the US EPA aggregates the air toxics into four general categories:

1. volatile organic compounds (VOCs)
2. metals
3. aldehydes
4. semivolatile organic compounds (SVOCs).

The air toxics of particular relevance to this report (that is, air toxics that result from motor vehicle exhausts) are the VOCs and polycyclic aromatic hydrocarbons (PAHs).

The VOCs are organic compounds in the boiling range of 50-260°C and include chemicals such as benzene, toluene and xylenes. VOCs are a concern because of their potential to contribute to the formation of ground level ozone and to global warming. Some of the VOCs can also have more direct effects on human health, for example, the link between benzene and leukaemia (Environment Australia 2001).

Burning fuels containing carbon (gasoline, oil, wood, coal, natural; gas), and using solvents, paints and glues releases VOCs. Motor vehicle emissions are an important source of VOCs.

PAHs are compounds that contain only hydrocarbon and carbon and are a group of over several hundred organic chemicals with two or more fused aromatic rings. Two ring PAHs are found in the vapour phase, two to five ring PAHs can be found in both the vapour and particulate phases and PAHs consisting of five or more rings tend to be solids adsorbed onto other particles in the atmosphere (Environment Australia 2001). Benzo-a-pyrene (B[*a*]P) is a five-ring compound and probably the most well known PAH. B[*a*]P is often used a marker for PAHs.

PAHs are a major component of polycyclic organic matter (POM). POM is divided into PAHs and PAH derivatives. PAH derivatives contain other elements (nitrogen, oxygen or sulphur) in addition to carbon and hydrogen (for example, nitro-PAH, amino-PAH and oxygenated-PAH).

PAHs are formed mainly as a result of incomplete combustion of organic materials during industrial and other human activities, such as processing of coal and crude oil, combustion of natural gas, combustion of refuse, wood burning stoves, motor vehicle exhaust, cooking, tobacco smoke, and natural processes such as carbonisation (WHO 2000). Occupational PAH exposure can occur in petroleum manufacture and use, coal production plants, coking plants, or where coal, wood or other plant materials are burned.

Of the hundreds of PAHs, toxicological endpoint and /or exposure data are available for only 33 PAHs (Environment Australia 2001).

3.2 Sources of air toxics

Air toxics are released from a variety of sources and activities. Most of the sources are anthropogenic sources (that is, related to human activities). Sources of air toxics can be divided into two main categories: point sources and diffuse sources. Point sources comprise industrial and other facilities that emit large amounts of air toxics in a localised area. Diffuse sources include mobile sources (motor vehicles and aircrafts) and area based sources (solid fuel combustion, dry-cleaning, building materials, and use of paints and thinners) (NEPC 2003).

Broadly, sources of air toxics include motor vehicles, industry, combustion of fossil fuels, cigarette smoking, household chemicals, and releases from carpets and furniture.

3.2.1 Motor vehicle emissions

This section will focus on motor vehicle emissions as most people will be expose to air toxics emitted from motor vehicles. Important air toxics emitted by motor vehicles are benzene, 1,3-butadiene, PAHs, formaldehyde and acetaldehyde. Most of the air toxics from cars arise from the by-products of the combustion process when fuel is burnt in the engine and then emitted via the exhaust system, and from evaporation of the fuel itself (NEPC 2003).

Incomplete combustion of fuel leads to emissions of VOCs (benzene, acetaldehyde, 1,3-butadiene) and formaldehyde. Evaporative emissions are mainly from petrol fuel and consists mainly of benzene and a small amount of lead. Diesel fuel has a lower vapour pressure and has negligible evaporative emissions compared to petrol fuel.

A number of factors will affect vehicle emissions. These are (NEPC 2003):

- vehicle type
- type and composition of fuel that is used
- age of vehicle
- types of roads on which a vehicle travels

Other factors include: road conditions, weather conditions, the proportions of hot and cold starts, and the use of air conditioners. In general, emissions are relatively insensitive to changes in fuel quality. Improvements in the emission rates of future vehicle fleets will be due primarily to advance in pollution control technology. The major advantage of cleaner fuels is to enable new pollution control technologies.

3.3 International guidelines/standards for air toxics

This section will give an overview of guidelines and standards for air toxics that are in use in different countries.

3.3.1 Australia

Australia has made a National Environment Protection Measure (NEPM) for four of the five air toxics discussed in this report (Table 3.1) (NEPC 2004). These are: benzene, PAHs, toluene and xylenes. The fifth air toxic for which a NEPM has been made, but which is not discussed in this report is formaldehyde.

These five air toxics were chosen on the following basis:

- pollutants that are primarily released from large point sources were excluded,
- adequate ambient monitoring data exist for the pollutants,
- the pollutant presents a significant risk to human health, and
- based on the National Pollutant Inventory data, the pollutant has highest mass emissions to the environment.

A second group of 12 air toxics (including 1,3-butadiene) was also identified for inclusion in the NEPM. This group of 12 compounds also met all the above criteria except that there were insufficient ambient monitoring data for the development of NEPMs.

The NEPM includes monitoring investigation levels (MILs) and are based on the protection of human health. MILs are not compliance standards. If MILs are exceeded then some form of further investigation may be appropriate (NEPC 2004).

The health endpoints selected as being appropriate as the basis for making NEPMs are as follows:

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

Table 3.1 Air toxics National Environment Protection Measure

Pollutant	Averaging period	Monitoring investigation level	Goal
Benzene	Annual average*	0.003ppm	For all the five pollutants, the 8-year goal is to gather sufficient data nationally to facilitate development of a standard
Benzo[*a*]pyrene (as a marker for PAHs)	Annual average	0.3ng/m3	
Formaldehyde	24 hours**	0.04ppm	
	Annual average		
Toluene	24 hours	1ppm	
	Annual average	0.1ppm	
Xylenes (as total of ortho, meta and para isomers)	24 hours	0.25ppm	
	Annual average	0.2ppm	

Note

* arithmetic mean concentrations of 24-hour monitoring results

** 24 hours measured from midnight to midnight

Source: (NEPC 2004)

Table 3.2 presents guidelines for the five air toxics discussed in this report that have been developed or used in some of the other countries or regions around the world.

Table 3.2 Air toxics guidelines/standards for the selected five air toxics

	Benzene	Benzo[*a*]pyrene	1,3-butadiene	Toluene	Xylenes
Australia	0.003ppm (annual average)	0.3ng/m^3 (annual average)	Nd[1]	0.1ppm (annual average)	0.2ppm (annual average)
United Kingdom air quality standards	5ppb (annual average)	0.25ng/m^3 (annual average)	Nd	Nd	Nd
New Zealand ambient air quality guidelines	10ug/m^3 (annual average)	0.3ng/m^3 (annual average)	Nd	Nd	Nd
European Commission air quality standard	5ug/m^3 (annual average)	Nd	Nd	Nd	Nd
World Health Organization	Nd	Nd	Nd	0.26mg/m^3 (weekly average)	Nd

Note

1 Not determined

Source: (NEPC 2003)

3.4 Assessing health effects

Air toxics can have both acute and chronic effects, as well as cancer and non-cancer effects.

Standard setting involves a range of environmental, health, technical, social, economic, political, legislative and cultural considerations. A health risk assessment (quantitative or qualitative) can be used to help in the standard setting.

3.4.1 Carcinogenic risk assessment

Cancer risk due to air pollutants is usually assessed using unit risk factors (URF). It is the risk of the incidence of cancer resulting from a lifetime exposure (usually 70 years) to 1ug/m^3 of a carcinogenic substance. The URF is also known as cancer unit risk estimate or inhalation potency factor (when inhalation is the pathway of exposure) (NEPC 2003).

Cancer risks are often expressed on a per million basis for comparative purposes. For example, a cancer risk of 100 in a million (that is, $100*10^{-6}$) at a location means that individuals resident at that location for 70 years have a 100 in a million chance of developing cancer. These estimates generally assume a non-threshold, low dose linearity, unless there is compelling evidence to the contrary, and are derived from occupational or animal studies.

Cancer risk can be estimated by multiplying the unit risk factor by the modelled ambient concentration to obtain a probability of cancer occurring in an exposed population.

$$Risk = concentration*URF$$

For example, if the unit risk is $1*10^6$ per 1ug/m^3 for chemical "Y", and the concentration of chemical "Y" is 5ug/m^3, then the risk is calculated as:

$$Risk = (1*10^{-6})*5 = 5*10^{-6}$$

This represents an upper estimate (usually a 95 percent upper confidence limit) of the potential cancer cases in a population exposed to chemical "Y" at 5 ug/m^3 over a lifetime. In other words, there is the potential for five extra cancer cases to occur over a 70-year period in a population of one million persons exposed to that level of chemical "Y".

In cases where there is exposure to multiple carcinogens, the cancer risk of each carcinogen is summed. The assumption implicit in this is that the effect on cancer risk in the population exposed to multiple pollutants is additive.

3.4.2 Non-cancer risk assessment

A number of measures have been used to assess the non-cancer risks of air pollutants. These are often known as investigation levels and are levels that, if exceeded, warrant further investigation (Table 3.3).

The Californian EPA has developed 'Reference Exposure Levels' (RELs) (OEHHA 2005a). These are health based levels and derived from No Observed Adverse Effect Level (NOAEL) or the Lowest Observed Adverse Effect Level (LOAEL) with uncertainty factors applied.

The Agency for Toxic Substances and Drug Registry (ATSDR) in the United States, has developed 'Minimal Risk Levels' (MRLs) (NEPC 2003; ATSDR 2005). A MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. MRLs are intended to be used as screening levels. They have been determined for inhalation, ingestion and dermal routes of exposure.

A 'Reference Concentration' (RfC) for chronic non-cancer effects is defined as the amount of toxicant, in ug/m^3, below which long term exposure to the general population, including sensitive subgroups, is not anticipated to result in any adverse effects. A central assumption underlying RfC is that a threshold exists below which no adverse effects will occur in the general population, although such a threshold may not be observable and can only be estimated. In general RfCs are derived from animal data through the application of extrapolation and uncertainty factors to NOAEL or LOAEL.

Non-cancer health endpoints are assumed to follow the concept of a threshold for effect. It is impossible to calculate the exact concentration at which anyone in a diverse population would respond. Inter-individual differences in response and the generally limited information on air toxics preclude such a determination. To quantify potential non-cancer health impacts the *Hazard Index* (HI) approach is recommended. While this method can quantify the increased risk due to air toxics, it cannot quantify the number of people affected.

The HI approach compares air pollutant concentrations with a corresponding hazard assessment benchmark such as the RfC or the REL. The Hazard Quotient (HQ) for a particular pollutant is the ratio of the concentration of the pollutant to the RfC or REL for that pollutant.

$$HQ_i = concentration_i / RfC_i$$

HQ_i . . . . . Hazard Quotient for pollutant i

$concentration_i$. . . . concentration of pollutant i

RfC_i . . . . . Reference Concentration (or REL) for pollutant i

A HQ>1 raises a "red flag" with regard to the exposure to that chemical and possible health impacts. Exceedance of a HQ of one does not necessarily mean that a health impact will in fact occur. It implies that the margin of safety built into the RfC is being eroded. The higher the ratio the closer the exposure to an adverse level. Uncertainty factors are included in the calculation of the RfCs to protect sensitive members of the population.

An indicator of total non-cancer hazard can be calculated by summing the HQs for each pollutant in order to derive a total HI.

$$HI_i = HQ_i$$

HI_i is the sum of the hazard quotients for all pollutants of interest. This measure assumes that multiple sub threshold exposures may result in an adverse health effect. Aggregate non-cancer hazards for specific target organ systems can also be evaluated by creating a separate total hazard index for several chronic non-cancer endpoints. These measures assume that, in the absence of comprehensive information, the effects of each pollutant are additive for a given organ system. The Californian EPA has developed toxicological endpoints in various target organ systems to be considered for a range of air toxics (OEHHA 2000).

Table 3.3 Investigation levels for the five selected air toxics

	Benzene	Benzo[*a*]pyrene	1,3-butadiene	Toluene	Xylenes
Australia	0.003 ppm (annual average)	0.3 ng/m^3 (annual average)	N/a[1]	0.1 ppm (annual average)	0.2 ppm (annual average)
California EPA (Inhalational reference exposure levels, RELs)	60 ug/m^3	N/a	260 ug/m^3	9.8 ppm (1 hour averaging)	5.0 ppm (1 hour averaging)
Agency for Toxic Substances and Drug Registry (Minimal risk levels, MRLs)	0.05 ppm (acute exposure)	N/a	N/a	1.0 ppm (acute exposure)	1.0 ppm (acute exposure)
US EPA (Reference concentration, RfC)	3*10^{-2} mg/m^3	N/a	2*10^{-3} mg/m^3	4*10^{-1} mg/m^3	0.1 mg/m^3

Notes

1 N/a=not assessed

3.5 Health effects of Air Toxics

National and international jurisdictions including Environment Australia, the US EPA, the Californian EPA and WHO have systematically reviewed the literature on the health effects of the five selected air toxics (benzene, 1,3-butadiene, PAH, toluene and xylenes) (Environment Australia 2001). A brief overview of the results of these reviews is presented.

Information on the health effects of air toxics is based mainly on cohort studies of workers exposed to high levels of air toxics and from animal toxicological studies. Our understanding of the health effects of air toxics is generally not as well developed as the common ambient air pollutants, primarily due to the lack of air toxics exposure data.

In theory, the process used to estimate the "acceptable" level of exposure requires that the entire slope of the dose-response curve relating exposure to a carcinogen to the likelihood of cancer developing in humans is known. In practice, only part of the dose response curve is likely to be known from epidemiological studies, or more often, from animal studies. Extrapolation from known risks at high levels to unknown risks at low levels of exposure requires the use of mathematical modelling of the dose response curve. These models extrapolate from actual effects at high exposure (from experimental and/or epidemiological data) to estimated effects at low exposures. Some low dose extrapolation models are more plausible than others, but it is generally impossible to test them either by epidemiological or experimental research designs. Depending on the assumptions made (for example, the source of the data used and the low dose model selected) the slope of the lines generated by different models and hence the estimates of risk, may vary substantially (Maynard, Cameron et al. 1997).

3.5.1 Benzene

Benzene is a natural component of crude oil. Almost all benzene found at ground level comes from human activities. It is emitted from industrial sources and a range of combustion sources including motor vehicle exhaust and solid fuel combustion. Benzene is also emitted from tobacco smoke. The major outdoor source is evaporative emissions and evaporation losses from motor vehicles, and evaporation losses during the handling, distribution and storage of petrol. Workers in industries exposed to motor vehicle exhaust are at risk of exposure.

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, climate and the concentration of other chemicals in the air, such as nitrogen and sulphur dioxide. It does not bio-accumulate in aquatic or terrestrial systems.

Inhalation is the dominant pathway for benzene exposure in humans. Smoking is an important source of personal exposure. Extended travel in motorcars also produces exposures that are second only to smoking as contributors to the intensity of overall exposure.

Current understanding of health effects of benzene are mainly derived 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. The most significant adverse effects of chronic benzene exposure are haematotoxicity, genotoxicity, carcinogenicity and can also lead to birth defects in humans and animals. There appears to be a dose-response relationship without any threshold effect. The mechanisms of benzene toxicity are not well understood.

Benzene is carcinogenic and long term exposure can affect normal blood production and can be harmful to the immune system. It can cause cancers and leukaemia (cancer of the tissues that form white blood cells) in laboratory animals and human populations exposed for long periods, and has been linked with birth defects in animals and humans.

Both the International Agency for Research and Cancer (IARC) and the US EPA have classified benzene as known human carcinogens (IARC 1987; US EPA 2005).

Although all in the population are susceptible to the adverse health effects of benzene, it is thought that at levels occurring in the ambient atmosphere, benzene does not have short-term or acute effects.

Even though adverse health effects have been documented with both acute and chronic exposures to benzene, for the purposes of the derivation of exposure-response functions, the main health endpoint that has been utilized is leukaemia.

Exposure response estimates are not available for non-leukaemia endpoints.

3.5.2 1,3-Butadiene

1,3-butadiene is emitted from oil refineries and chemical manufacturing plants. The major source of 1,3-butadiene is incomplete combustion of petrol and diesel fuel. 1,3-Butadiene is highly reactive and can oxidise to form formaldehyde and acrolin, two toxic substances in their own right. 1,3-Butadiene is emitted from industrial facilities, tobacco smoke and motor vehicle emissions. Workers in industries that use or produce 1,3-butadiene or are exposed to motor vehicle exhaust are at risk of exposure. 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 central nervous system damage, blurred vision, nausea, fatigue, headache, decreased pulse rate and pressure, and unconsciousness. Lung term exposure to lower levels has shown increases in heart and lung damage. There are inadequate human data (based on only a few occupational studies) but sufficient animal data to suggest that 1,3-butadiene is a human carcinogen. Chemical compounds closely related to 1,3-butadiene are known human carcinogens.

The US EPA classified 1,3-butadiene in Group B2: probable human carcinogen (US EPA 2005). 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).

3.5.3 Polycyclic Aromatic Hydrocarbons

PAHs contain only hydrocarbon and carbon and are a group of over several hundred organic chemicals with two or more fused aromatic rings (B[*a*]P is probably the most well known PAH). PAHs are formed mainly as a result of pyrolitic processes, especially the incomplete combustion of organic materials during industrial and other human activities, such as processing of coal and crude oil, combustion of natural gas, combustion of refuse, vehicle traffic, cooking, tobacco smoke, and natural processes such as carbonisation (WHO 2000). Occupational PAH exposure can occur in petroleum manufacture and use, or where coal, wood or other plant materials are burned. Most PAHs in air they are generally found attached to particulate matter. Occupation exposure to PAH may occur in coal production plants, coking plants and coal-gasification sites.

Data from animal studies indicate that several PAH may induce a number of adverse effects including immunotoxicity, genotoxicity, carcinogenicity and reproductive toxicity. B[*a*]P is by far the most intensively studied PAH in animals. B[*a*]P is the only PAH that has been tested for carcinogenicity following inhalation and it produces lung cancer in animals. The lung carcinogenicity of B[*a*]P is enhanced by co-exposure to other substances such as cigarette smoke and probably airborne particulates (WHO 2000). Results from epidemiological studies indicate 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 PAH.

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 PAH in air is desirable. B[*a*]P has been suggested as the most appropriate indicator (WHO 2000). The US EPA has classified B[*a*]P in Group B2, probably human carcinogen (US EPA 2005). IARC has classified B[*a*]P in Group 2A, human carcinogen (IARC 1987).

3.5.4 Toluene

Toluene is widespread in the environment due to its use in a variety of commercial and household products and it is found in tobacco smoke. Indoor toluene levels can be higher than outdoor levels during non-occupational exposure to paints and thinners, and also where tobacco smoke is present. Sniffing glue or paint can lead to high exposures. Air pollution from vehicles is a major source of exposure (WHO 2000). Toluene is emitted during crude petroleum and natural gas extraction, and petroleum refining. Workers in industries exposed to motor vehicle exhaust are at risk of exposure.

The central nervous system (CNS) is the primary target organ for toluene toxicity in both animals and humans for acute and chronic exposure. CNS dysfunction (often reversible) and narcosis are observed in humans exposed to low or moderate levels. Short term exposure to high levels of toluene can result in light-headedness and euphoria. CNS depression occurs in chronic 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. The US EPA has classified toluene in Group D, not classifiable as carcinogenic to humans (US EPA 2005). IARC has classified toluene in Group 3, not classifiable as a carcinogen (IARC 1987).

3.5.5 Xylenes

Xylenes are emitted during petroleum refining, solid fuel combustion, and is a component of vehicle exhaust. They are common components in numerous domestic products.

Acute exposure to xylenes 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 un-coordination. Other effects of chronic exposure include impaired pulmonary function, and possible affects on the blood and kidneys. The evidence of developmental or reproductive effects on humans in inconclusive. Xylenes do not appear to be carcinogenic. The US EPA has classified toluene in Group D, not classifiable as carcinogenic to humans (US EPA 2005). IARC has classified toluene in Group 3, not classifiable as a carcinogen (IARC 1987).

3.6 Cancer unit risk factors for the five selected air toxics

3.6.1 Benzene

The US EPA gives a range for the quantitative estimate of “Leukaemia” from inhalational exposure to benzene. The cancer URF is $2.2*10^{-6}$ per ug/m3 to 7.8*10-6 per ug/m3 (US EPA 2005).

WHO (WHO 2000) air quality guidelines concluded that benzene is carcinogenic to humans and no safe level of exposure can be recommended and gives the estimated excess lifetime risk of leukaemia as $6*10^{-6}$ per µg/m3.

The Californian EPA (OEHHA 2005b) has determined benzene is a carcinogen and identified an inhalation URF of $29*10^{-6}$ per µg/m3. The health endpoint for the exposure response relationship is “All Leukaemias”. It is assumed that there is no threshold for the exposure response relationship and that exposure is over a 70-year lifetime exposure.

Table 3.4 Cancer unit risk factors for benzene

Source	Cancer endpoint	Inhalation URF (per ug/m^3)
Californian EPA	Leukaemia in humans	$29.0*10^{-6}$
WHO	Leukemia in humans	$6.0*10^{-6}$
US EPA	Leukaemia in humans	$2.2*10^{-6}$ to $7.8*10^{-6}$
IARC	Not stated	$4.4*10^{-6}$ to $7.4*10^{-6}$

The above four reports present a range of cancer unit risk factors for inhalational benzene (Table 3.4). The California EPA estimate is about an order of magnitude greater than the other two estimates and the WHO estimate falls within the range of the US EPA estimates.

3.6.2 1,3-Butadiene

1,3-Butadiene may induce cancers at multiple sites in rodents including heart, lung, mammary gland, ovaries, liver, pancreas, thyroid, testes, and the hematopoietic system. Recent epidemiological evidence suggests an association between excess cases of leukaemia and lymphoma, and 1,3-butadiene exposure, although this provides only limited evidence to support the carcinogenic effects observed in experimental animals.

The US EPA (US EPA 2005) classified 1,3-butadiene as carcinogenic to humans by inhalation, and determined a cancer URF of $30*10^{-6}$ per ug/m^3.

WHO air quality guidelines (WHO 2000) concluded that 1,3-butadiene is probably carcinogenic to humans (Group 2A) but that there was insufficient evidence available to establish a lifetime risk estimate for 1,3-butadiene.

The Californian EPA has determined 1,3-butadiene is a carcinogen and identified an inhalation URF of $170*10^{-6}$ per ug/m^3 (US EPA 2005).This is an upper bound value for the risk. These URFs are presented in Table 3.5.

Table 3.5 Cancer unit risk factors for 1,3-butadiene

Source	Cancer endpoint	Inhalation URF (per ug/m^3)
US EPA	Leukaemia in humans	$30*10^{-6}$
Californian EPA	Mice studies	$1{,}700*10^{-6}$
WHO	-	Not available

3.6.3 Polycyclic Aromatic Hydrocarbons and benzo[*a*]pyrene

The California EPA has determined several PAHs are carcinogens, and identified an inhalation URF for B[*a*]P of $1100*10^{-6}$ per ug/m^3. This number is the upper risk limit. A number of other PAHs have been assigned relative URFs, with B[*a*]P as the reference compound.(36)

The US EPA has not established a URF for B[*a*]P as human data are lacking. However there are data from many animal studies demonstrating B[*a*]P to be carcinogenic. The US EPA and the IARC have classified B[*a*]P as a probable human carcinogen.

WHO air quality guidelines (WHO 2000) do not recommend any specific guideline values for PAH in air. PAH are typically constituents of complex mixtures and some PAH are potent carcinogens that may react with a number of other compounds. However, to facilitate the setting of control priorities, a unit risk factor for B[*a*]P as an indicator of PAH in air was estimated to be $87000*10^{-6}$ per ug/m^3 (Table 3.6).

Table 3.6 Cancer unit risk factors for benzo[*a*]pyrene

Source	Cancer endpoint	Inhalation URF (per ug/m^3)
US EPA	-	Not available
Californian EPA	Respiratory tract cancers in hamsters	$1{,}100*10^{-6}$
WHO	Not stated	$87{,}000*10^{-6}$

3.6.4 Toluene

The US EPA has not been able to assess the carcinogenicity of toluene as there are no data on the carcinogenicity of toluene in humans and the available animal data are inadequate (US EPA 2005). Both the US EPA (US EPA 2005) and IARC have defined toluene as “Not classifiable as a carcinogen”. Cancer URFs for toluene do not exist.

3.6.5 Xylenes

The US EPA has not been able to assess the carcinogenicity of xylenes as there are inadequate human data on the carcinogenicity of xylenes in humans and the available animal data are inconclusive. At this stage, xylenes are not classified as human carcinogens and quantitative URFs for cancer do not exist(US EPA 2005).

3.7 Contribution of fuels to air toxics emissions

It is recognized that the transport sector, and specifically motor vehicles, contributes significantly to the total emissions of air toxics. Motor vehicle emissions are even more important as large sections of the populations are exposed to motor vehicle emissions. This is unlike the case of occupational exposures where smaller numbers of people will be exposed to the pollutant.

Various types of pollutants are produced in the combustion process. A range of VOCs is produced because the fuel is not completely burnt (oxidised) during combustion. Oxides of nitrogen result from the oxidation of nitrogen at high temperature and pressure in the combustion chamber. Carbon monoxide occurs when carbon in the fuel is partially oxidised rather than fully oxidised to carbon dioxide. Sulphur dioxide and lead are derived from the sulphur and lead in fuels. Particulate matter is produced from the incomplete combustion of fuels, additives in fuels and lubricants, and worn material that accumulates in the engine lubricant. These additives and worn materials also contain trace amounts of various metals and their compounds which may be released as exhaust emissions.

Evaporative emissions come mainly from petrol (diesel fuel has a much lower vapour pressure) and consist of VOCs and small amounts of lead. These emissions may occur in several ways (NPI 2000):

Diurnal Losses:. . . . As the ambient air temperature rises during the day, the temperature of fuel in the vehicle's fuel system increases and increased vapour is produced.

Running Losses . . . . Heat from the engine and exhaust system can vaporize gasoline when the car is running.

Hot Soak Losses. . . . Because the engine and exhaust system remain hot for a period of time after the engine is turned off, gasoline evaporation continues when a car is parked.

Resting Losses. . . . . Vapour may be lost from the fuel system or the evaporative emission control system as a result of permeation through rubber components and other leaks.

Evaporative emissions also occur from vehicle refuelling at service stations or from fuel tanker loading and unloading, or when liquid fuel leaks or is spilt.

In 2003-2004, motor vehicle contributions to the total emissions of benzene, 1,3-butadiene, toluene and xylenes were 63 percent, 35 percent, 54 percent and 57 percent (NPI 2005). The motor vehicle contribution to PAHs was 8 percent with a greater proportion of emissions from the use of solid fuels (48 percent) (NPI 2005). However, ambient concentrations of ambient air toxics are generally very low.

The type of fuel that is used by the motor vehicle fleet has a bearing on the total emissions of air toxics. Fuels that are commonly used in motor vehicles are petrol, diesel and, to a lesser extent, LPG/CNG. Fuel usage varies by the category of motor vehicles. Passenger vehicles are mainly powered by petrol engines whereas a significant proportion of light commercial vehicles use diesel (and this proportion is projected to substantially increase over time) and heavy vehicles are mainly powered by diesel engines. LPG is used by some passenger vehicles (for example, taxis) light commercial vehicles and buses.

Table 3.7 presents fuel usage data by vehicle category as estimated by Coffey Geosciences in a report to Environment Australia (Coffey Geosciences 2003). It is estimated that in 2020, there will only be small increases in the uptake of LPG/CNG for passenger vehicles, light commercial vehicles and buses (uptake at 6.2 percent, 12.3 percent and 5 percent for passenger vehicles, light commercial vehicles and buses respectively). It was also estimated that there will be a substantial increase in diesel use in light commercial vehicles.

Table 3.7 Fuel usage distribution by VKT in 2000 and 2020

Vehicle Category	Fuel Usage Distribution by VKT 2000 Vehicles			Fuel Usage Distribution by VKT 2020 Vehicles		
	Petrol	Diesel	LPG/CNG	Petrol	Diesel	LPG/CNG
Passenger	95.3	2.5	2.2	88.8	5.0	6.2
Light Commercial	72.7	23.0	4.3	41.0	45.0	12.3
Rigid Trucks	0	100	0	0	100	0
Articulated Trucks	0	100	0	0	100	0
Buses	0	99	1	0	95	5
Motorcycles	100	0	0	100	0	0

Source: (Coffey Geosciences 2003)

Emission factors for air toxics for various types of fuels are presented in Table 3.8 (NPI 2000). Emission factors for LPG/CNG are similar or slightly higher than those for petrol. However it should be noted that evaporative emissions for LPG/CNG contain mainly propane and butane and do not contain any of the air toxics on the National Pollution Inventory (NPI 2000).

Table 3.8 Emission factors for VOCs by motor vehicle category and fuel type

Vehicle type	Fuel	Emission factor (g/km)		
Exhaust		Arterial	Freeway	Residential
Passenger	Petrol	1.26	1.34	1.45
	Diesel	0.331	0.310	0.513
	LPG/CNG	1.53	1.51	1.73
Light commercial	Petrol	1.64	1.53	2.53
	Diesel	0.554	0.517	0.857
	LPG/CNG	1.76	1.63	2.70
Heavy duty	Petrol	3.08	2.88	4.77
	Diesel	1.01	0.941	1.56
	LPG/CNG	3.29	3.07	5.09
Evaporative				
Passenger	Petrol	0.535	0.241	0.535
	LPG/CNG	1.07	0.483	1.07
Light commercial	Petrol	0.586	0.275	0.586
	LPG/CNG	1.17	0.550	1.17
Heavy duty	Petrol	2.91	2.15	2.91
	LPG/CNG	5.81	4.29	5.81

Source: (NPI 2000)

For passenger cars, emission factors for individual air toxics are presented using published speciation factors (multiplying the emission factor for total VOCs by the speciated weight fractions for those species) (NPI 2000). LPG/CNG has lower emission factors for all the five air toxics. Of importance is the fact that the emission factors are much less for LPG/CNG for benzene, 1,3-butadiene and PAHs, which are all designated as human carcinogens. Table 3.9 also presents the reduction in the emission factors for the five air toxics for petrol and diesel engines compared to LPG/CNG. It is important to note that there are significant reductions for all the five air toxics.

Table 3.9 Emission factors and percentage reduction in emission factors for individual air toxics for passenger cars

Passenger Cars	Emission factor (g/km)					
	Arterial		Freeway		Residential	
Exhaust	EF*	% reduction in EF**	EF	% reduction in EF	EF	**% reduction in EF**
Benzene						
Petrol	0.08291	100.0	0.08817	100.0	0.09541	100.0
Diesel	0.00334	99.7	0.00313	99.7	0.00518	99.6
LPG/CNG	0.00001		0.00001		0.00002	
1,3-butadiene						
Petrol	0.01064	99.1	0.00993	99.1	0.01642	99.1
Diesel	0.00064	84.4	0.00059	84.7	0.00099	84.8
LPG/CNG	0.00010		0.00009		0.00015	
PAHs						
Petrol	0.00668	100.0	0.00625	100.0	0.01035	100.0
Diesel	0.00674	100.0	0.00628	100.0	0.01041	100.0
LPG/CNG	0.00000		0.00000		0.00000	
Toluene						
Petrol	0.05618	100.0	0.02531	100.0	0.05618	100.0
Diesel	0.01573	100.0	0.00710	100.0	0.01573	100.0
LPG/CNG	0.00000		0.00000		0.00000	
Xylenes						
Petrol	0.08880	100.0	0.04175	100.0	0.08880	100.0
Diesel	0.03405	100.0	0.02516	100.0	0.03405	100.0
LPG/CNG	0.00000		0.00000		0.00000	

Notes

***** EF=emission factor

****** Percent reduction in emission factor compared to LPG/CNG

Source: (NPI 2000)

Table 3.10 presents the percentage reduction in emission factors for air toxics for light commercial vehicles. This is relevant as VKT projections indicate substantial increases for light commercial vehicles (107 percent increase in VKTs from 2000 to 2020) (Coffey Geosciences 2003). It is important to note that the use of LPG/CNG in motor vehicles will nearly completely eliminate emissions of air toxics.

Table 3.10 Changes in emission factors for air toxics for light commercial vehicles

Light commercial vehicles	% reduction in emission factors		
Exhaust	Arterial	Freeway	Residential
Benzene			
Petrol	100.0	100.0	100.0
Diesel	99.7	99.7	99.7
LPG/CNG	-	-	-
1,3-butadiene			
Petrol	99.1	99.1	99.1
Diesel	84.8	84.9	84.9
LPG/CNG	-	-	-
PAHs			
Petrol	100.0	100.0	100.0
Diesel	100.0	100.0	100.0
LPG/CNG	-	-	-
Toluene			
Petrol	100.0	100.0	100.0
Diesel	100.0	100.0	100.0
LPG/CNG	-	-	-
Xylenes			
Petrol	100.0	100.0	100.0
Diesel	100.0	100.0	100.0
LPG/CNG	-	-	-

Source: (NPI 2000)

3.8 Summary

- National guidelines have been established for five air toxics (benzene, benzo[a]pyrene [as a marker of PAHs], formaldehyde, toluene and xylenes).
- Concentrations of ambient air toxics are generally low and below the Air Toxics NEPM.
- Benzene, 1,3-butadiene and PAHs are human carcinogens.
- Toluene and xylenes are not human carcinogens but have human health effects.
- Motor vehicles are significant sources of air toxics.
- Type of fuel used in motor vehicles can significantly impact on air toxic emissions.
- Exhaust emissions (g/km) for total VOCs are highest for LPG/CNG and lowest for diesel fuel.
- Exhaust emissions (g/km) for benzene and 1,3-butadiene are 85 percent to 100 percent lower for LPG/CNG fuel compared to petrol and diesel fuels.
- There are no exhaust emissions for PAHs, toluene and xylenes for LPG/CNG fuel.
- There are no air toxics evaporative emissions from LPG/CNG fuel.
- Encouraging the use of LPG/CNG fuel can reduce motor vehicle emissions of air toxics and hence ambient concentrations of air toxics.

3.9 References

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04.
Particulate matter

4.1 Background

Particulate matter (PM) is a complex mixture of solids and/or liquids suspended in air. The size and composition of particulate matter is an important determinant of the health effects.

4.1.1 Size

Particles size is measured as the aerodynamic diameter of the particles and governs where the particles will be deposited in the respiratory tract and also the transport and removal of the particles from the air. The site of deposition in the respiratory tract influences the acute symptoms provoked, retention within the airways and possibly the long-term health consequences. Based on aerodynamic diameter particle pollution is divided into three groups PM_{10}, $PM_{2.5}$ and $PM_{0.1}$.

PM_{10} includes particles with an aerodynamic diameter 10 µm or less and therefore measures of PM_{10} include $PM_{2.5}$ and $PM_{0.1}$ (Figure 4.1). The precise definition of PM_{10} is particles that pass through a size selective inlet with a 50 percent efficiency cut-off at 10 µm aerodynamic diameter (European Commission 1997). Particles above 10 µm in diameter are predominantly deposited in the upper airways, such as in the nose, pharynx and trachea (Figure 4.2). Particles between 2.5-10 µm are inhalable and are deposited in the in the larger airways (Figure 4.2).

Figure 4.1 Categories of airborne particulate matter

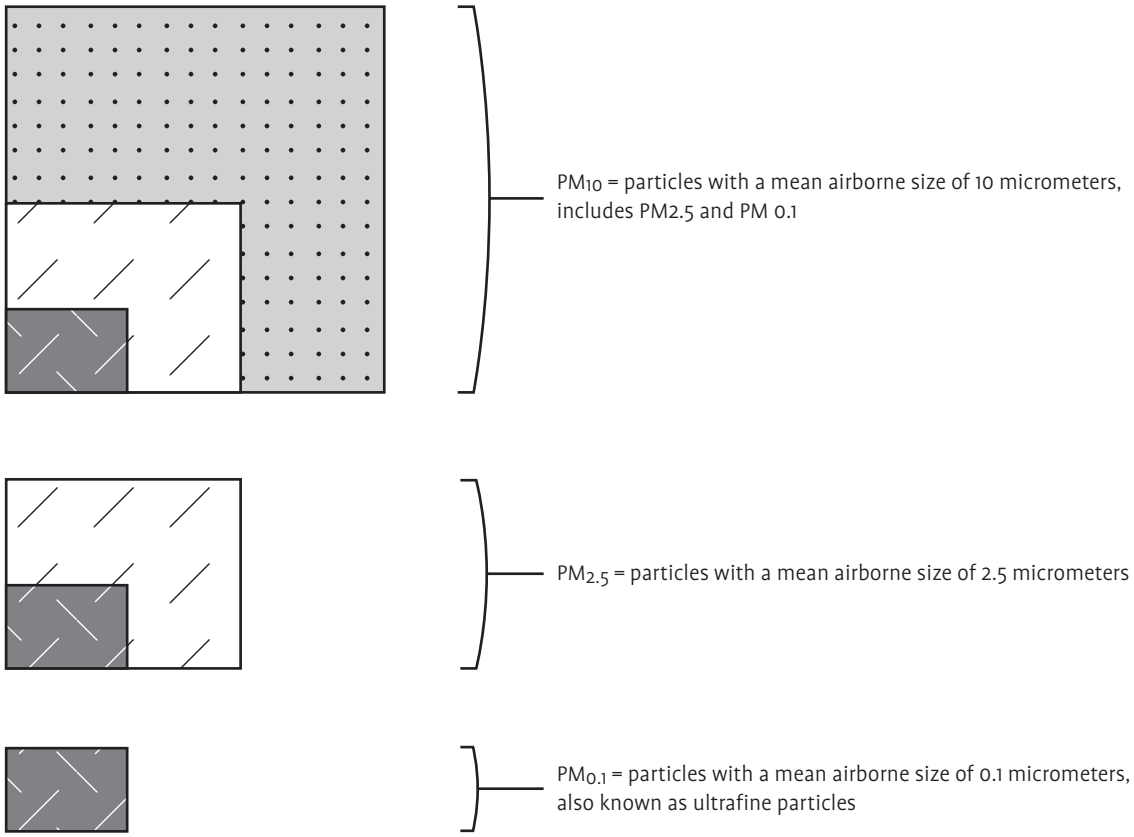
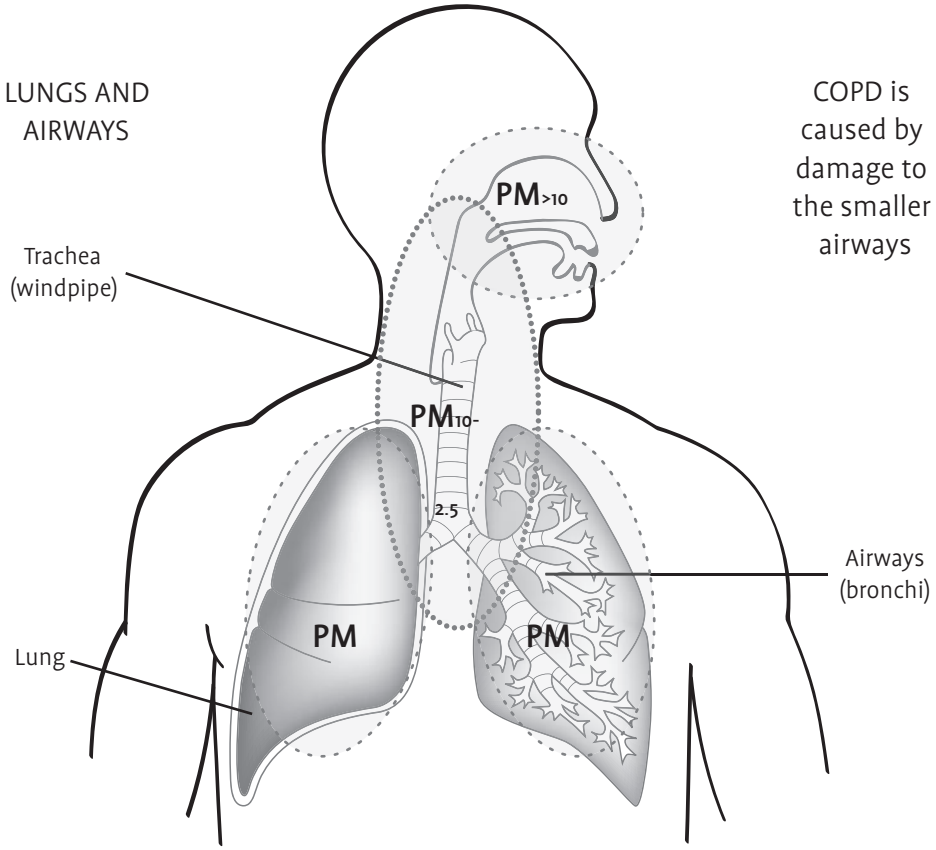


Figure 4.2 Penetration of particulate matter into the respiratory tract



$PM_{2.5}$ is all particles equal to or less than 2.5 µm in aerodynamic, or, more strictly, particles which pass through a size selective inlet with a 50 percent efficiency cut-off at 2.5 µm aerodynamic diameter. $PM_{2.5}$ is therefore a subset of PM_{10} (Figure 4.1). Smaller particles referred to as respirable, range in aerodynamic diameter from 0.1 to 2.5 µm and are deposited deeper into the respiratory tract, such as the bronchi and alveoli of the lungs (Figure 4.2) and have more adverse health consequences than larger particles. $PM_{2.5}$ are released as a result of combustion processes, such as motor vehicle exhausts and solid fuel heaters.

$PM_{0.1}$, referred to as ultrafine particles, have an aerodynamic diameter of less than 0.1 µm. $PM_{0.1}$ is therefore a subset of both $PM_{12.5}$ and PM_{10} (Figure 4.1). $PM_{0.1}$ account for the largest number of airborne particles, however the combined mass of these particles is often recorded as insignificant in comparison to the larger particles (Morawska, Moore et al. 2003). Due to limitation in methods for quantifying ultrafine particles, very few of the studies have examined the role of ultrafine particles in health outcomes (Morawska, Moore et al. 2003).

4.1.2 Composition

PM can be either primary or secondary in nature. Primary particles are emitted directly into the atmosphere either by natural or man-made processes, whereas secondary particles have a predominantly man made origin and are formed in the atmosphere from the oxidation and subsequent reactions of sulphur dioxide, nitrogen oxides and volatile organic compounds. The particles themselves are therefore a complex mixture of organic and inorganic compounds in solid or liquid states. Types of particulate pollution range from relatively large such as mineral dusts, such as occur during dust storms, to small particles released as a result of condensation of metals or organic compounds following high temperature combustion.

In addition to the different chemical composition of particles as a result of their formation process they may also be carriers of biological and non-biological mediators of inflammation. Biological mediators of mediators of inflammation include endotoxins and allergens, while non-biological mediators are some metal ions and polyaromatic hydrocarbons (WHO Europe 2003).

The ability of diesel particles to function as allergen carriers and to enhance the inflammatory response to allergens is well documented (WHO Europe 2003).

There also appears to be differences in the nature of the inflammatory response generated against smaller versus larger particles that are carrying allergen. While smaller particles carrying allergens may penetrate deeper into the respiratory tract they do not necessarily induce a greater immediate inflammatory response. In laboratory studies on cat allergic people with asthma, Lieutier-Colas et al. (Lieutier-Colas, Purohit et al. 2003) found that 10 micrometer droplets of cat allergen were 20 times more potent than 1.4 micrometer droplets for inducing immediate inflammatory responses, while the smaller particle were more potent at inducing late inflammatory responses (Lieutier-Colas, Purohit et al. 2003).

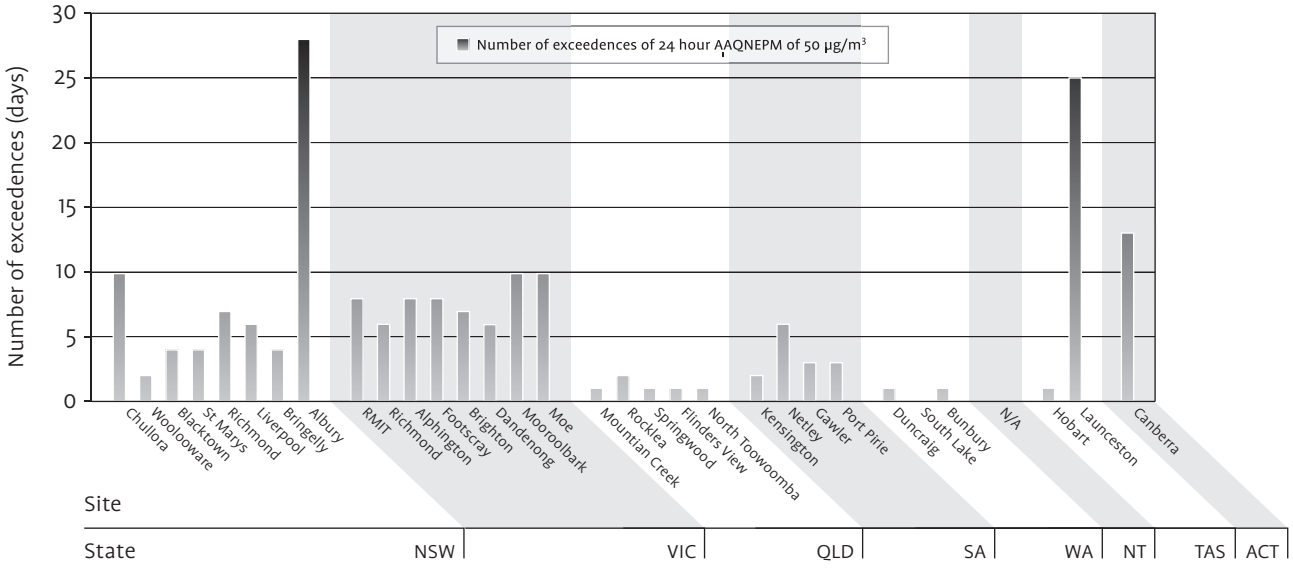
Metal ions have also been implicated in increasing the inflammatory properties of the inhaled particles. $PM_{2.5}$ particles rich in cadmium, nickel zinc and copper were found to induce almost twice the inflammatory response as particles with lower levels of these metal ions (Schaumann, Borm et al. 2004).

4.2 Exposure to PM

4.2.1 Ambient PM_{10}

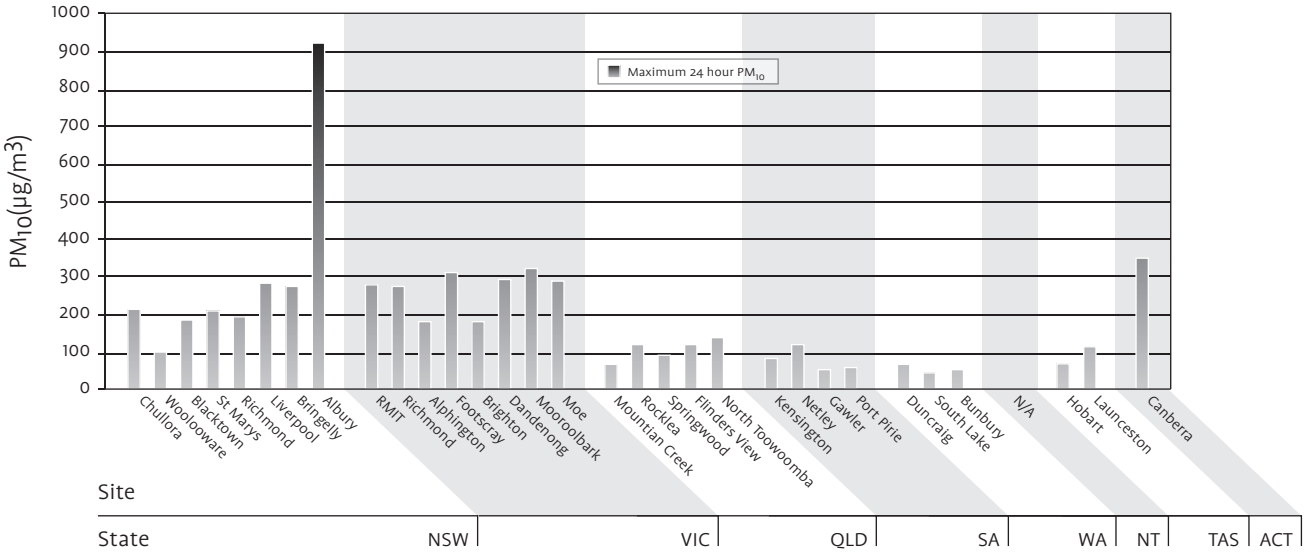
In most of the urban areas of Australia, where PM_{10} levels are monitored, they do exceed the current 1-day Air NEPM standard of 50 µg/m³ (Figure 4.3), however the highest levels recorded are often in rural locations and are associated with bushfires, wind-blown dust, and for Launceston, solid wood heaters (Figure 4.4). Exceedences in the major cities occur on 1-5 days per year and are normally also associated with bushfires and wind-blown dust. The median level of PM_{10} was from 13 to 21 µg/m³ in with higher median levels recorded at city locations (Figure 4.5).

Figure 4.3 Number of time the 24 hour PM_{10} concentration exceeded 50 µg/m³ in Australian Cities in 2003



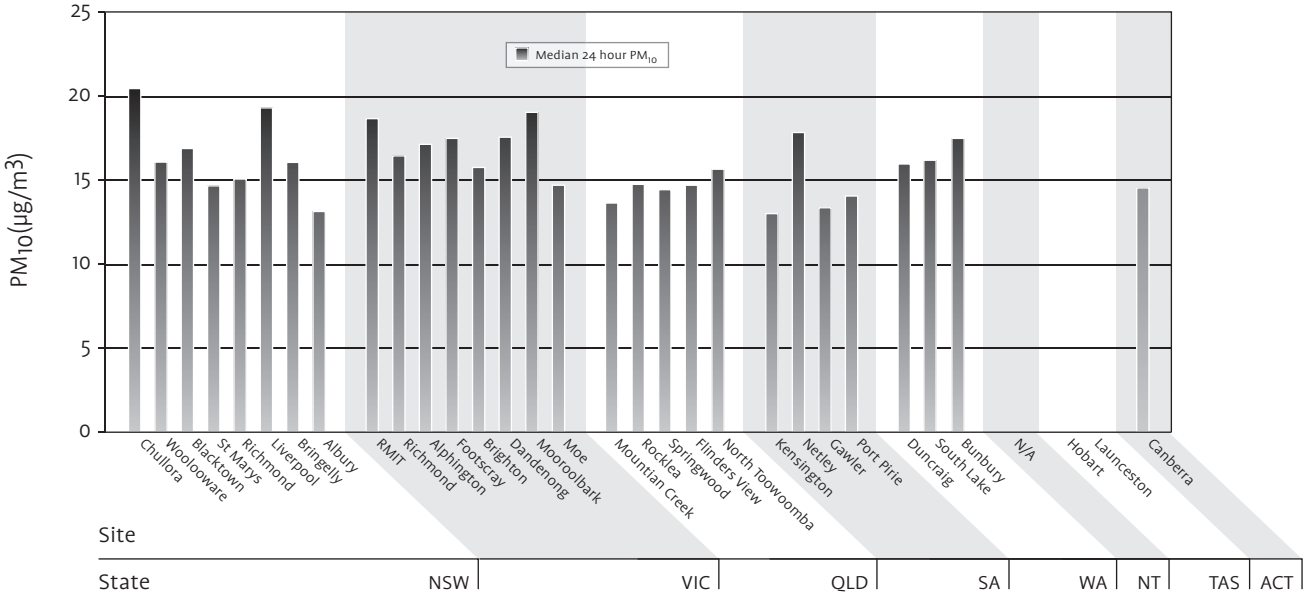
Source: (Department of Environment WA 2004; EPA ACT 2004; EPA NT 2004; EPA Queensland 2004; EPA SA 2004; EPA Tasmania 2004; EPA Victoria 2004; NSW Department of Environment and Conservation 2004)

Figure 4.4 Maximum 24 hour PM_{10} in Australian Cities in 2003



Source: (Department of Environment WA 2004; EPA ACT 2004; EPA NT 2004; EPA Queensland 2004; EPA SA 2004; EPA Tasmania 2004; EPA Victoria 2004; NSW Department of Environment and Conservation 2004)

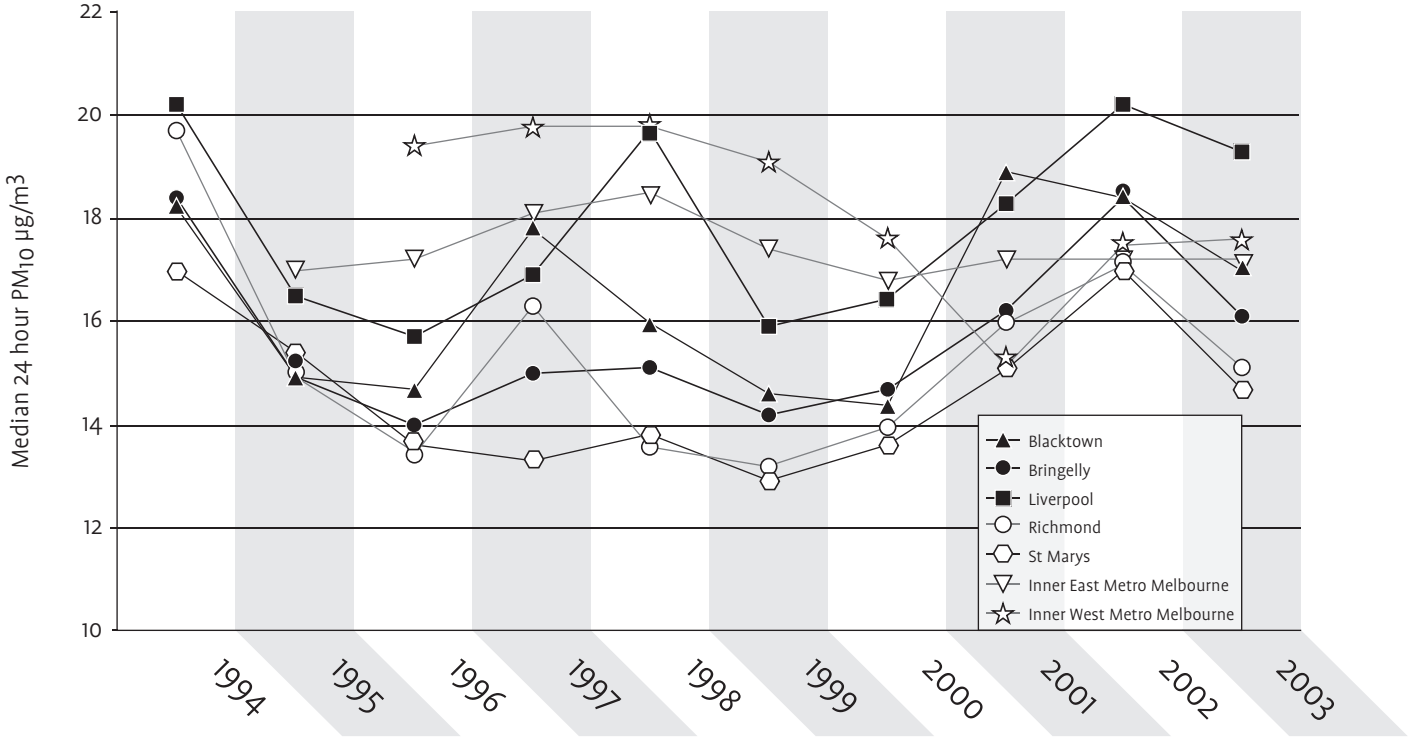
Figure 4.5 Median 24 hour PM_{10} in Australian Cities in 2003



Source: (Department of Environment WA 2004; EPA ACT 2004; EPA NT 2004; EPA Queensland 2004; EPA SA 2004; EPA Tasmania 2004; EPA Victoria 2004; NSW Department of Environment and Conservation 2004)

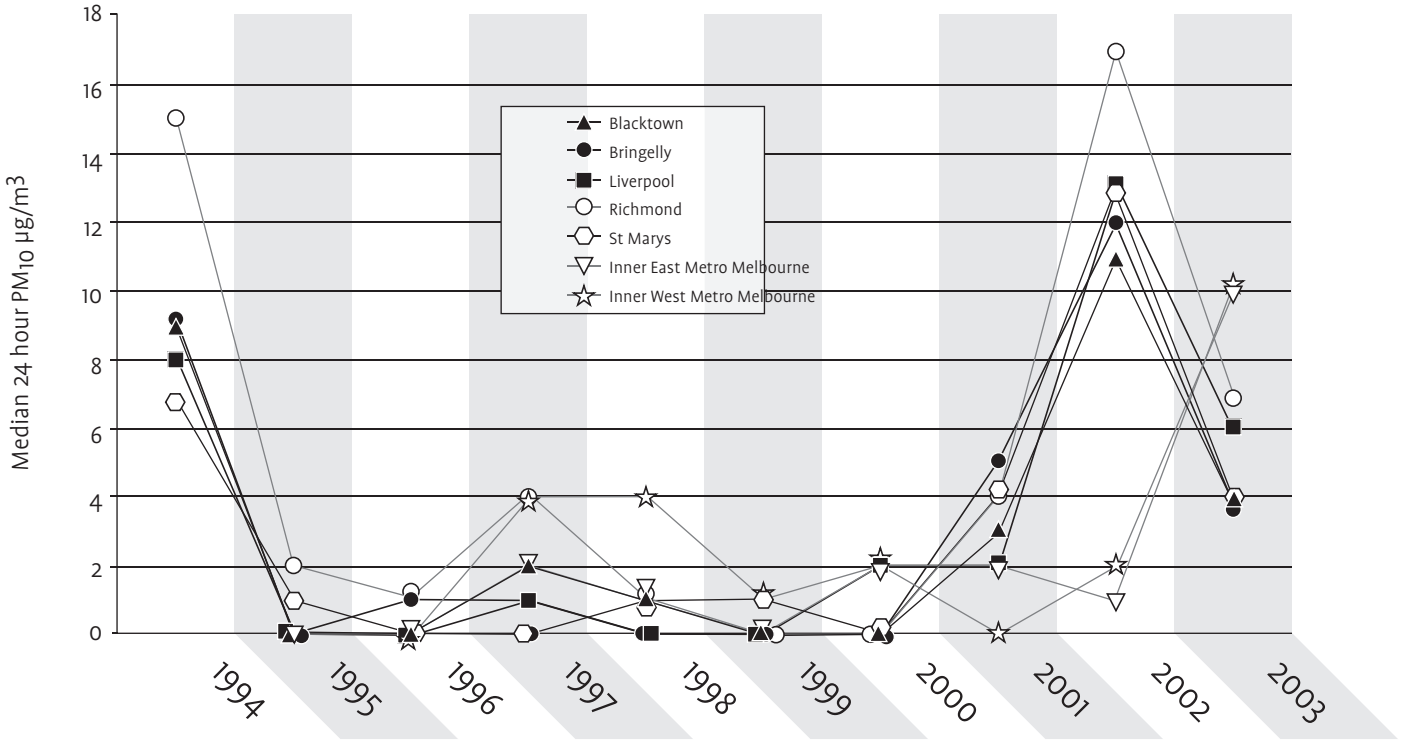
While median ambient PM_{10} levels in Australia are below the air quality NEPM, they have not decreased over time (for example, Figures 4.6 and 4.7), as has occurred for a number of other pollutants, such as lead, carbon monoxide and nitrogen dioxide. This has resulted in State EPA's expressing concern of the levels of PM_{10} in their major cities.

Figure 4.6 Median 24 hour PM_{10} (µg/m³) at sites around Sydney and Melbourne, over the period 1994-2003



Source: (EPA Victoria 2004; NSW Department of Environment and Conservation 2004)

Figure 4.7 Number of times in each year the maximum 24 hour PM_{10} exceeded 50 µg/m³ at sites around Sydney and Melbourne, over the period 1994-2003



Source: (EPA Victoria 2004; NSW Department of Environment and Conservation 2004)

4.2.2 Ambient $PM_{2.5}$

$PM_{2.5}$ levels in several Australian cities are given in Table 4.1. Although there is no Ambient Air Quality NEPMs for $PM_{2.5}$, the advisory reporting standard for 24 hours is 25 µg/m³. In the larger cities of Sydney and Melbourne, the 24 hour average concentration occasionally exceeds the advisory reporting standard of 25 µg/m³. In 2003 exceedences were often associated with bushfires.

Table 4.1 $PM_{2.5}$ concentration in Australian cities in 2003

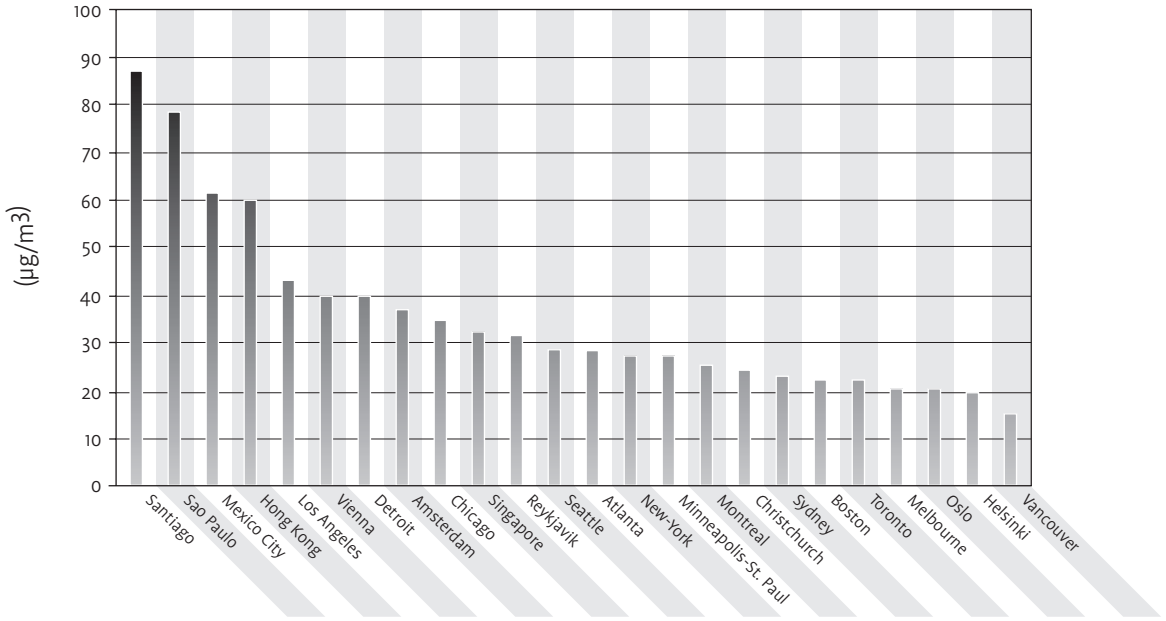
Location	Maximum (µg/m³)	Median (µg/m³)	Number of times exceeded 25 µg/m³
South-East Queensland			
Rocklea	33.1	3.9	1
Springwood	20.6	4.8	0
Victoria			
CBD South East	44.6	7.0	4
Inner East Metro	38.4	5.7	4
Inner West Metro	51.1	4.8	3
South Metro	39.4	5.1	2
Outer East Metro	33.5	7.6	3
Western Australia			
North East Metro	27.3	7.6	1
North Metro	25.2	8.3	1
South West Region	37.6	7.8	3
New South Wales			
Chullora	81.0	9.7	6
Earlwood	39.4	9.6	9
Wooloowaare	67.7	9.4	5
Richmond	61.9	8.1	10
Westmead	67.8	10.3	4
Liverpool	50.1	11.9	11

Source: (EPA Victoria 2004; NSW Department of Environment and Conservation 2004; Queensland 2004; EPA Tasmania 2004)

4.2.3 Australian versus overseas PM levels

Internationally, out of 24 cities considered, Melbourne and Sydney rank respectively 18th and 21st position for annual average PM_{10} concentration (Figure 4.8).

Figure 4.8 Annual average concentration of PM_{10} in selected cities in 1995

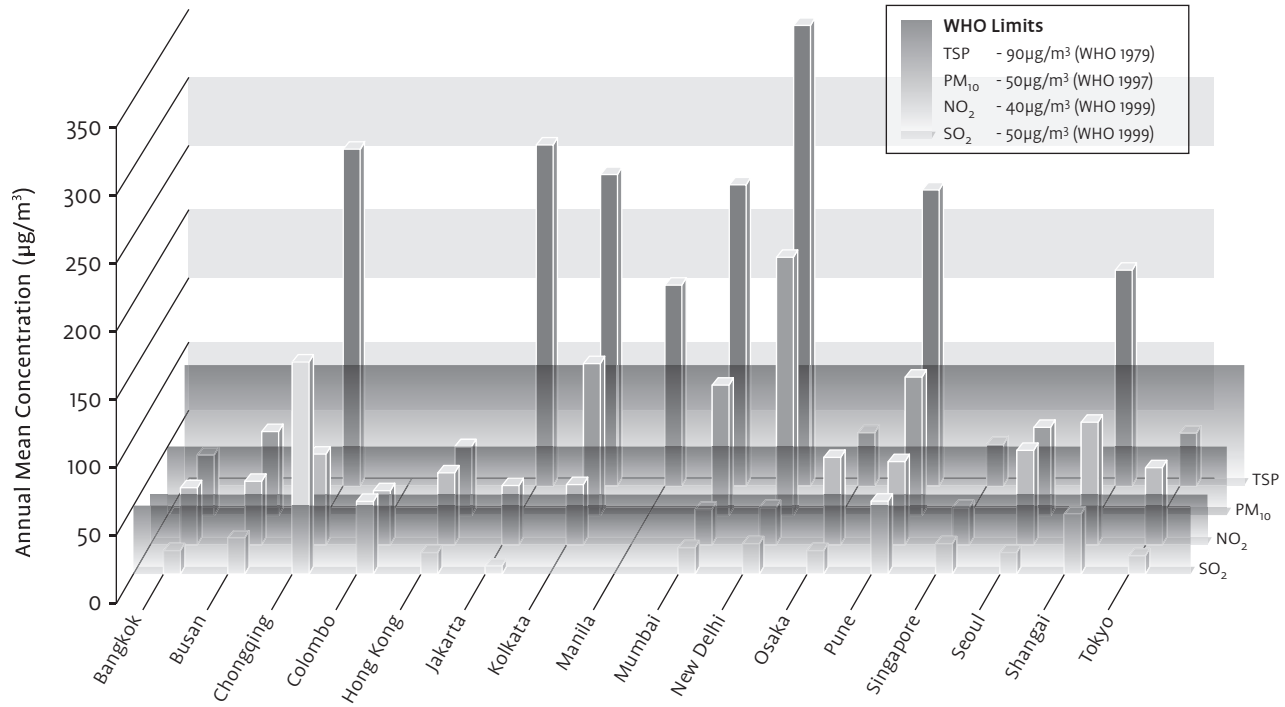


Source: (Manins, Allan et al. 2001)

4.2.4 PM exposure in Asia

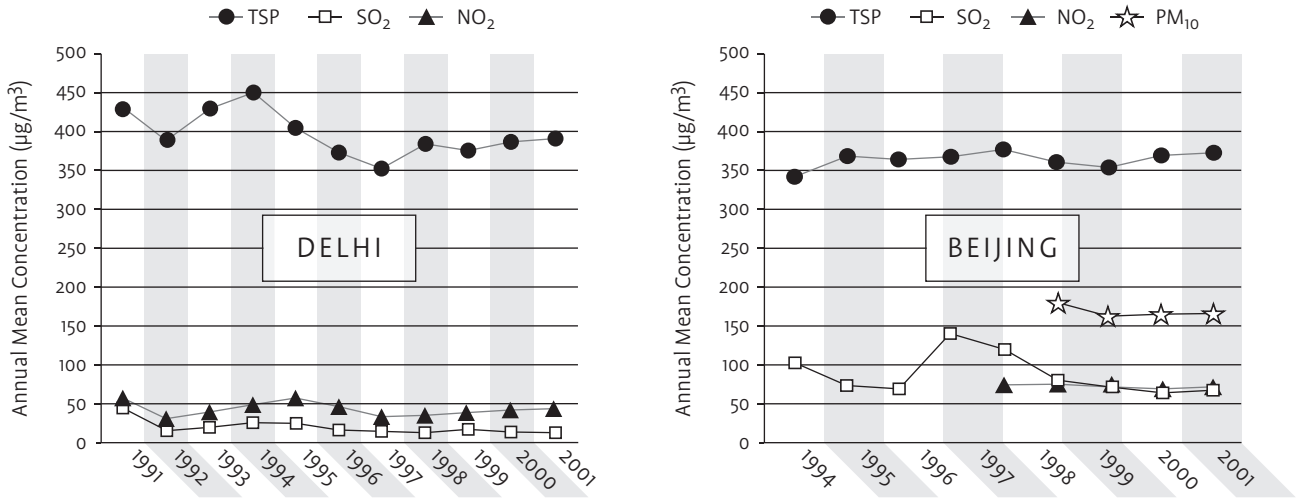
Some of the highest levels of outdoor air pollution in the world are found in Asian cities. Figure 4.9 shows the annual mean concentrations of suspended PM (TSP), PM_{10}, sulphur dioxide, and nitrogen dioxide in 2000 and 2001 in major Asian cities (Health Effects Institute 2004). Some cities in China and India have some of the world's highest recorded outdoor PM_{10} levels (Figure 4.10).

Figure 4.9 Annual mean concentrations of pollutants compared with their guidelines and standards in Asian cities, 2000 and 2001



Source: (Health Effects Institute 2004)

Figure 4.10 Annual mean concentrations of pollutants in residential areas of Delhi and Beijing



Source: (Health Effects Institute 2004)

4.3 International guidelines/standards for PM

4.3.1 Australia

The Ambient Air Quality National Environment Protection Measure (AAQNEPM) was introduced in 1998 by the National Environment Protection Council (NEPC) and provides national ambient air quality standards for the criteria air pollutants to which most Australians are exposed (Table 4.2). The AAQNEPM standards for PM_{10} were set after consideration of the health effects of these pollutants based on reports prepared by Streeton (Streeton 1997), the Technical Review Panel of the NEPC and the existing NH&MRC goals (National Environment Protection Council 1998). There is no current ambient air NEPM for $PM_{2.5}$ and in the past these finer particles have not been separately measured from PM_{10}. In 2003 the Environment Protection and Heritage Council (EPHC) introduced Advisory Reporting Standards for $PM_{2.5}$, which are:

25 µg/m³ averaged over one day (24 hours)

8 µg/m³ averaged over one year

The goal of the Advisory Reporting Standards are to gather sufficient data nationally to facilitate a review of the Advisory Reporting Standards as part of the development of a $PM_{2.5}$ ambient air NEPM, which is scheduled to commence in 2005. Monitoring of $PM_{2.5}$ commenced in all States and Territories in either January or July 2004.

Table 4.2 Australian Ambient Air Quality NEPM for PM_{10}

Pollutant	Averaging period	Maximum concentration
PM_{10}	1 day	50 µg/m³
($PM_{2.5}$)*	1 day	25 µg/m³
	1 year	8 µg/m³

Note

* Advisory Reporting Standard (see above)

Source: (National Environment Protection Council 1998)

4.3.2 International Standards

Australia's air quality standards are similar to overseas standards (Table 4.3), in addition some overseas standards have considered different time frames which are designed to reflect either the nature of the dose response relationship or the types of exposures encountered in the community.

Table 4.3 Summary of overseas air quality standards

Pollutant	Level	Time Period	Country	Institution
PM_{10}	No WHO standard, since the WHO were unable to identify a threshold concentration below which ambient PM has no effect on health.			WHO
	50 μg/m³	24 hr	Australia	NEPC
	150 μg/m³	24 hr	US	US EPA
	50 μg/m³	24 hr	US (California)	Southern California Air Quality Monitoring Department
	50 μg/m³	24 hr	UK	
	40 μg/m³	1 yr	UK	UK Dept. of Environment, Transport and the Regions
	150 μg/m³	24 hr	US	US EPA
	50 μg/m³	24 hr	NZ	New Zealand Ministry for the Environment
	75, 150 & 250 μg/m³	24 hr	China	Level depends on classification of area: Class 1 is tourist, historic and conservation areas. Class 2 is residential urban and rural areas. Class 3 is industrial areas and heavy traffic areas.
$PM_{2.5}$	No WHO Standard, since the WHO were unable to identify a threshold concentration below which ambient PM has no effect on health.			WHO
	25 μg/m³	24 hr	Australia	NEPC. Advisory reporting standard for non-peak sites.
	65 μg/m³	24 hr	US	US EPA
	15 μg/m³	1 yr	US	US EPA
	30 μg/m³	24 hr	Canada	Canadian Council of Ministers of the Environment.
	25 μg/m³	24 hr	New Zealand	New Zealand Ministry for the Environment, proposed interim guideline.

Note

1 Not determined

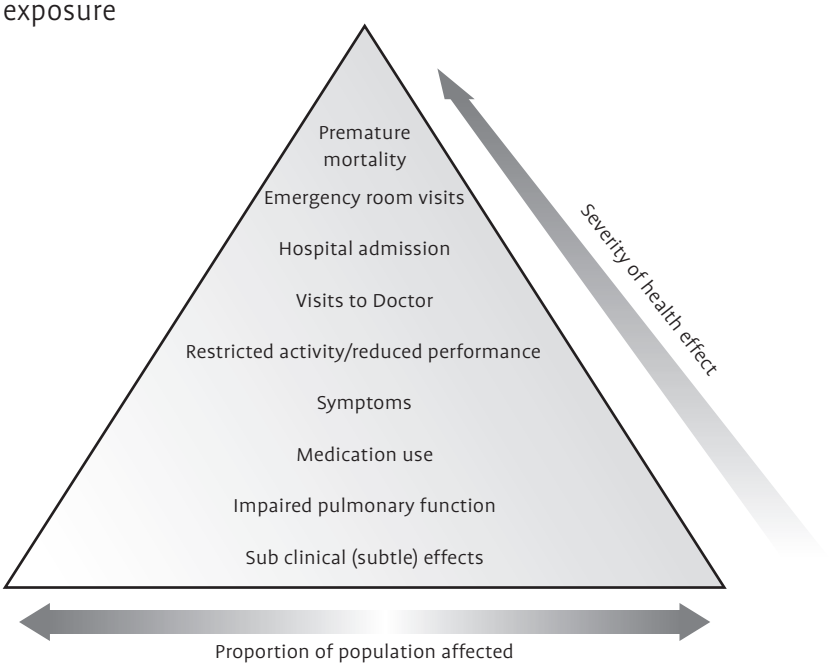
Source: (Cains, Cannata et al. 2003; O'Meara, Jalaludin et al. 2003)

4.4 Review of the health effects of PM exposure

4.4.1 Assessing the effect of air pollution on health

The adverse health effects as a result of ambient particulate matter exposure may range from the relatively mild sub clinical effects such as increased cough, clinical effects of reduction in lung function or increased medication usage, through to seeking medical attention by a general practitioner, emergency department attendances, hospital admission and premature mortality. Figure 4.11 illustrates the relationship between the frequency of an adverse health outcome and its severity. Less frequent events, such as death, are often a less sensitive marker of a health impact than, for example, change in respiratory symptoms.

Figure 4.11 The severity and frequency of an adverse health impact as a result of ambient air pollution exposure



Source: (WHO Europe 2001)

4.4.2 Types of Studies

There are three types of studies that examine the effects of air pollution on health. Each type of study has strengths and weaknesses and no single study can be interpreted as conclusive.

4.4.2.1 *Experimental Chamber Studies*

Challenge chamber or experimental studies involve the exposure of individuals to a known concentration of a single pollutant or mixtures of pollutants under experimental conditions. An individual or a small group of people are exposed to a certain concentration of a single pollutant for a defined period of time. During the experimental studies people are exercising at a moderate or high intensity for the majority of time they are exposed to the pollutant. Exercising increases the amount of pollutant inhaled and also deposits it further into the lower airways.

The advantages of chamber studies are that they are a controlled exposure study so that the dose of exposure can be accurately assessed. Chamber studies are extremely powerful for determination of dose response relationships, that is the impact of concentration and time on a health outcome. Challenge chambers also enable quantitative outcomes to be measured such as changes in lung function, which requires some interpretation. For assessing lung function, both the rate and total volume of air that can be forcibly exhaled after a full and deep inspiration are measured. The rate of expiration of air is often expressed as forced expiratory volume in the first second of expiration and termed FEV_1. FEV_1 is often reported as percentage of predicted FEV_1, which is based on established values that vary with sex, height and age. The maximum or peak expiratory flow rate (PEFR) is another measure of the rate expiration. PEFR is the maximum flow rate of forcibly exhaled air following a deep breath. The volume of expired air is termed the forced vital capacity (FVC). Another measure of lung function is airway resistance. Airway hyperresponsiveness (AHR) is a measure of the response of the lung to broncho-constricting stimuli.

Many studies examine the association between changes in FEV_1, or PEFR and levels of pollutants. In interpreting these studies it is necessary to assess the importance of percentage changes in FEV_1 or PEFR. Both FEV_1 and PEFR show diurnal variability in healthy subjects and in subjects with asthma and chronic obstructive pulmonary disease (COPD). The US EPA expert panel concluded that a greater than 10 percent fall in FEV_1 should be regarded as an adverse effect (U.S. Congress Office of Technology Assessment 1989). The American Thoracic Society (American Thoracic Society 1985; Crapo, Broaddus et al. 2000) concluded that reversible loss of lung function associated with symptoms should be considered as adverse. In this report we have accepted that a short-term reduction in FEV_1 of 10 percent or more, which is attributable to a pollutant exposure, does constitute an adverse health effect.

Much of the variation caused by pre-existing symptoms, personal exposure variations, level of activities, duration of exposure, local changes in concentration of pollutants, mixes of pollutants, medical practices or subjective assessment of outcomes can be minimised in chamber studies. Subjects in the studies are blinded to the concentration of pollutant at which they are exposed and when combined with objective assessment of lung function they are less influenced by participant's perceptions. Participants in the challenge chamber studies may be selected from certain at risk groups in the community, for example those with asthma or hypertension.

The disadvantages of challenge chamber studies are that the number of participants is limited and therefore the application of the data to the wider at-risk population is uncertain. Chamber studies do not replicate the range of environmental exposures and activities undertaken across the community and they do not account for the vast range of other environmental factors that may influence a response to a pollutant or a mix of pollutants. Environmental conditions within chambers such as temperature, humidity and the mixture of environmental pollutants may not depict the mixture of these variables that occurs in the ambient air. Furthermore, it is only feasible to measure short-term outcomes in chamber studies, that is symptoms and changes in lung function. The effect of pollutants on disease exacerbations and serious adverse outcomes cannot be assessed.

4.4.2.2 *Time series studies*

Time series studies are a powerful way to examine the influence of a pollutant upon a community. In these studies a community health outcome, such as deaths, emergency department attendances or hospital admissions are related to the ambient levels of pollutants. The data on air pollutants and health outcomes are collected retrospectively. The data are adjusted to take account of a variety of non-pollutant factors that may influence the health outcome, such as influenza epidemics or weather. From these studies two important outcomes can be assessed: which groups in the community are most sensitive to pollutants; and what is the relationship between an increase in the level of a pollutant and a community health outcome. The sensitivity of the health outcome to a change in pollutant level is normally expressed as an odds ratio or relative risk, which relates the increase in health outcome to a unit increase in pollutant. Odds ratios are often expressed as a percentage, for example, a 1.0 percent increase in hospital admissions for asthma and COPD in response to a 10 $\mu g/m^3$ increase in ambient PM_{10}.

4.4.2.3 *Panel or cohort studies*

A common form of observational study is the panel or cohort study. In short term studies, a group of volunteers (eg at a summer camp or a school) record symptoms and lung function measurements every day over a period of weeks or months during which time air pollutants and other environmental variables are measured at the site. This is a more powerful way of assessing air pollution effects than chamber studies since they are performed on a larger number of subjects and under real conditions, but it is often difficult to separate out the relative importance of specific pollutants and impossible to determine the actual level of exposure.

Long-term cohort studies are also used to assess the cumulative impact of air pollutants on health. In such studies groups of people living in different locations, which have different levels of air pollutants, are followed over periods of up to 20 years to assess the long term impact of air pollution on mortality and development of specific diseases such as lung cancer. The impact on children's health has also been assessed in these studies by examining changes in lung function or the development of diseases such as asthma or leukaemia.

4.4.3 Thresholds: Is there a safe level of particulate matter exposure?

A threshold is a level below which no adverse health effect will occur. The US EPA recently concluded that there was currently no scientific basis for selecting a threshold for the effects of the major air pollutants, including particulate matter, if a threshold is defined as a level characterised by an absence of observable effects (Committee on the Medical Effects of Air Pollutants 2002). The National Morbidity, Mortality and Air Pollution Study (NMMAPS) also recently examined whether a threshold existed for PM. Models were developed to test for thresholds for daily time-series data for the 20 largest US cities for 1987 through 1994, using concentration of PM_{10} as the exposure measure and mortality as the health outcome. These studies indicated that linear models without a threshold are appropriate for assessing the effect of particulate air pollution on daily mortality even at current ambient levels (Daniels, Dominici et al. 2004).

There is no threshold for PM_{10}.

4.4.4 At risk groups

Precisely defining the 'at risk' groups in the community is difficult and appears to depend on the nature of the study undertaken. For example, experimental challenge chamber studies suggest that individuals with asthma are not more susceptible to the effect of ozone than people without asthma, however asthmatics appear to be one group in the community who are more likely to be admitted to hospital on high ozone days. Generally those most at risk are children and people with respiratory diseases such as asthma and COPD, which includes chronic bronchitis and emphysema, as well as people with heart disease (Table 4.4).

It is generally acknowledged that children are more susceptible to the health effects of air pollutants than adults (Environment Australia 2003). The following reasons have been proposed:

- Infant's metabolic pathways may be insufficient to detoxify pollutants;
- Children are more metabolically active and therefore inhale more air than adults;
- Children also have smaller calibre airways that may more easily constrict; and
- Children spend more time outdoors.

Table 4.4 People who appear to be most at risk from the health effects of air pollutants

Pollutant	Group at risk	Impact on this group
CO	People with cardiovascular disease	Increase angina and increase risk of myocardial infarction
	Pregnant women and foetus	Reduced growth rate of the foetus
NO_2	People with asthma	Bronchoconstriction following exposure to levels of > 0.26 ppm and allergen challenge
		Increased risk of hospitalisation
	People with cardiovascular disease	Increased risk of hospitalisation
PM	Children	Reduced lung function growth
	People with chronic respiratory diseases (asthma and COPD)	Increased likelihood of hospitalisation
	People with cardiovascular or ischaemic heart disease	Increased likelihood of hospitalisation or death

4.4.5 Experimental studies on the acute effect of PM

Several experimental challenge chamber studies have measured the acute health effect of exposing individuals to high concentrations of particles generated from diesel engines or concentrated ambient pollutants (Table 4.5).

Exposure to concentrated particles or diesel particles results in lung inflammation that is dose dependant (Ghio, Kim et al. 2000). Exposure to levels of 100-300 µg/m^3 of PM_{10} has consistently been found to cause inflammation. This inflammatory response is characterised by neutrophil infiltration into the lungs and occurs in both people with and without asthma (Table 4.5).

Rudell et al. (Rudell, Ledin et al. 1996) exposed healthy non-smokers for one hour to diesel smoke that contained 2.6x10^6/cm^3 particles, 1.9 ppm nitrogen dioxide$_2$, 2.7 ppm nitric oxide, 30 ppm carbon monoxide and other pollutants. They also examined the effect of partial filtration of the smoke, which resulted in a 46 percent reduction in particles, but no change in the other pollutants. Compared with air, exposure to diesel smoke or partially filtered smoke, resulted in significant increases in unpleasant smells, irritation to the eyes and nose but varied enormously between the small number of individuals (n=12) tested (Rudell, Ledin et al. 1996). Diesel smoke or partially filtered smoke, when compared with air also resulted in a significant increase in airway resistance, but no change in FEV_1 or FVC.

Salvi et al. (Salvi, Blomberg et al. 1999) exposed 15 healthy human volunteers to air and diluted diesel exhaust under controlled conditions for 1 hour with intermittent exercise. The exposures were standardized by keeping the PM_{10} concentration at 300 µg/m^3 that was associated with 1.6 ppm nitrogen dioxide, 4.5 ppm nitric oxide, 7.5 ppm carbon monoxide; 4.3 ppm total hydrocarbons, 0.26 mg/m^3 formaldehyde, and 4.3x10^6 suspended particles/cm^3. Lung function (PEFR, FEV_1, FVC) was measured before and after each exposure and did not change following diesel exposure. Blood sampling and bronchoscopy were performed 6 hours after each exposure to obtain airway lavages and endobronchial biopsies. They found no changes in lung function but there were both systemic and pulmonary inflammatory responses. There was a significant increase in neutrophils and B lymphocytes in airway lavage, along with increases in histamine and fibronectin. The bronchial biopsies obtained 6 hours after diesel exposure showed a significant increase in neutrophils, mast cells, CD4+ and CD8+ T lymphocytes along with upregulation of the endothelial adhesion molecules ICAM-1 and VCAM-1, with increases in the numbers of LFA-11 cells in the bronchial tissue. Significant increases in neutrophils and platelets were observed in peripheral blood following diesel exposure.

Nordenhall et al. (Nordenhall, Pourazar et al. 2000) exposed fifteen healthy nonsmoking volunteers to 300 µg/m^3 of PM_{10} and air for 1 hour on two separate occasions. Analyses of sputum differential cell counts and soluble protein concentrations were performed 6 and 24 hours post exposure. Six hours after exposure to diesel exhaust a significant increase was found in the percentage of sputum neutrophils (37.7 versus 26.2 percent p=0.002) together with increases in the concentrations of interleukin-6 (12.0 versus 6.3 pg.mL^{-1}, p=0.006) and methylhistamine (0.11 versus 0.12 µg.L^{-1} , p=0.024). Irrespective of exposure, a significant increase was found in the percentage of sputum neutrophils at 24 hours as compared to 6 hours, indicating that the procedure of sputum induction itself may change the composition of sputum.

4.5 Combined exposures

The acute effect of motor vehicle exhaust on adults with asthma was assessed by Svartengren et al. (Svartengren, Strand et al. 2000). Twenty subjects with mild asthma were placed in a car inside a road tunnel for 30 minutes during peak-hour. This tunnel had a length of ~1,500m and was used by ~35,000 vehicles per day. The levels of nitrogen dioxide, PM_{10} and $PM_{2.5}$ were assessed inside the car, in the tunnel and at an urban location where control studies were performed. Four hours after tunnel or control exposures subjects were challenged with a mild dose of inhaled allergen (Table 4.5).

The median nitrogen dioxide concentration inside the car was 313 µg/m^3 (range: 203–462 µg/m^3) and was 28 times higher than the ambient exposure at the urban control site which was 11 µg/m^3 (range: 0–51 µg/m^3). The PM_{10} and $PM_{2.5}$ concentrations in the car were 170 µg/m^3 (range: 103–613 µg/m^3) and 95 µg/m^3 (range: 61–218 µg/m^3), respectively. PM_{10} and PM_2 levels at the control urban site were 14 times lower at 7 µg/m^3 (range: 2–17 µg/m^3) and 5 µg/m^3 (range: 2–11 µg/m^3), respectively.

Symptoms of noise, smell, cough irritation and self-perceived respiratory health were adversely affected by being in the tunnel. There were no differences in airway resistance or lung function during exposure to nitrogen dioxide, PM_{10} and $PM_{2.5}$ in the tunnel (Table 4.5).

Once people left the tunnel and were challenged with allergen their lungs were more reactive and a significant increase in airway resistance was found. Overall the effect of nitrogen dioxide across the entire group was not significant and there was no significant relationship between the level of nitrogen dioxide and change in airway resistance. However when the group was divided into those with exposures above 300 µg/m^3 (0.15 ppm) there was a significant increase in airway resistance and decrease

in lung forced expiratory flow in those exposed to >300 µg/m^3 compared with the control (unexposed group). The percentage reduction in FEV_1 was 8.5 percent for >300 µg/m^3 nitrogen dioxide versus 6.8 percent for the air control.

Subjects with the highest $PM_{2.5}$ exposure (>100 µg/m^3) had a marginally greater early reaction after allergen challenge compared with the control groups (no exposure).

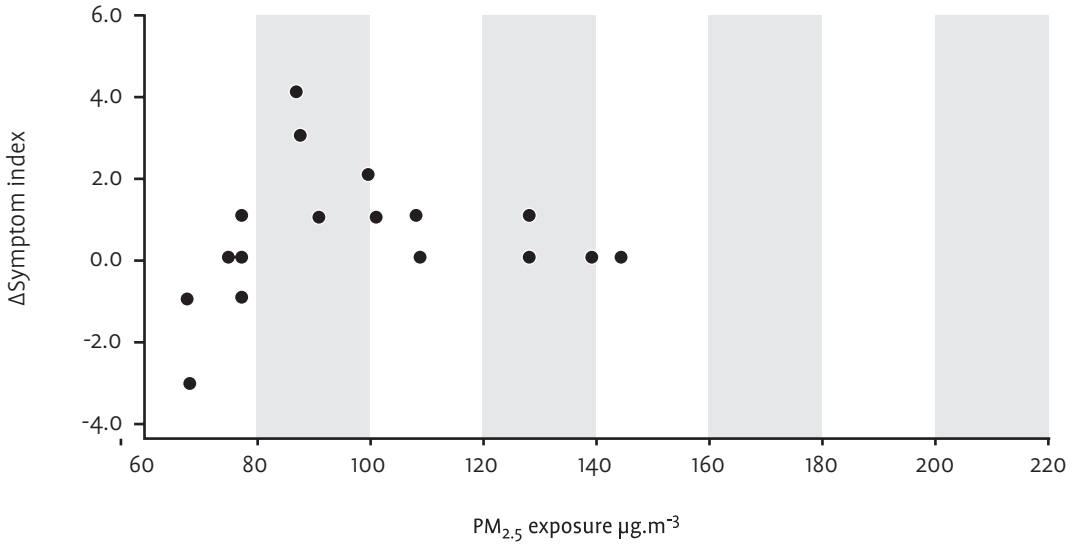
Tunnel exposure resulted in significantly more asthma symptoms, following allergen challenge, during the evening after the tunnel exposure than after the control exposure (p=0.016). Furthermore, the combined asthma symptoms during the evening, night and morning after the exposure tended to be worse (p=0.085). These symptoms were significantly related to nitrogen dioxide exposure. Asthma symptoms were significantly increased compared to control, when nitrogen dioxide exposure was >300 µg/m^3. The group with nitrogen dioxide exposure above 300 µg/m^3 had significantly more asthma symptoms during the exposure evening compared to control and more symptoms during the night after allergen inhalation compared to the group with those who were exposed to less than 300 µg/m^3.

Table 4.5 Challenge Chamber Studies with Particles

Study	Experimental group	Level and duration of exposure PM	Health outcomes measured and results.
Rudell et al. (1996)	n=12, healthy adults, non-asthmatics, non-smokers, exercising.	Partially filtered diesel exhaust for 1 hour, which contained: 1.4-2.6 x 10^5/cm^3 particles, 1.7-1.9 ppm NO_2, 2.6-2.7 ppm NO and 27-30 ppm CO, plus hydrocarbons and formaldehyde.	Symptoms, lung function and airway resistance. Symptoms of eye and nose irritation were increased although highly variable between individuals and therefore not significantly different. Significant increase in airway resistance.
Salvi et al. (1999)	n=15, healthy adults, non-asthmatics, non-smokers, exercising	300 µg/m^3 diesel particles (PM_{10}) for 1 hour on two occasions.	Lung function, inflammatory cells and mediators in bronchial lavage and serum. Significant increase in neutrophils, mast cells and lymphocytes in the lung and increases in neutrophils and platelets in blood.
Ghio et al. (2000)	n=38 (divided into 4 groups), healthy adults, non-asthmatics, non-smokers, exercising.	0, 48, 107 & 207 µg/m^3 for 2 hours.	Symptoms, lung function, inflammatory mediators and cells in the lungs (bronchial lavage). No change in symptoms, FEV_1 and FVC, PEF or airway resistance. Significant increases in neutrophils and no change in IL-8 or IL-6. Significantly increased fibrinogen in blood.
Nightingale et al. (Nightingale, Rogers et al. 2000)	n=10, healthy adults, non-asthmatics, non-smokers, at rest.	200 µg/m^3 diesel particles (PM_{10}) for 2 hours.	Lung function, AHR, Inflammatory cells and mediators in sputum. Significant increase in neutrophils and myeloperoxidase.
Nordenhall et al. (2000)	n=15, healthy adults, non-asthmatics, non-smokers, exercising.	300 µg/m^3 diesel particles (PM_{10}) for 1 hour on two occasions.	Inflammatory cells and mediators in sputum. Significant increase in neutrophils, IL-6 and methylhistamine.
Nordenhall et al. (Nordenhall, Pourazar et al. 2001)	n=14, atopic asthmatics, with stable asthma exercising.	300 µg/m^3 diesel particles (PM_{10}) for 1 hour on two occasions.	Lung function, airway resistance and AHR. Inflammatory cells and mediators in sputum. Significant increase in airway resistance, AHR and IL-6.
Svartengren et al. (2000)	n=20 adults with mild asthma. Exposed to pollutants within a road tunnel.	30 minute exposures to NO_2 313 µg/m^3 (range: 203–462 µg/m^3). PM_{10} & $PM_{2.5}$ 170 µg/m^3 (range: 103–613 µg/m^3) & 95 µg/m^3 (range: 61–218 µg/m^3) respectively.	Lung function, airway resistance, symptoms pre and post bronchial challenge. No effect on symptoms. Exposure to > 300 µg/m^3 NO_2 had a significantly greater early reaction, following allergen exposure, as well as lower lung function and more asthma symptoms during the late phase. Exposure to >100 µg/m^3 $PM_{2.5}$ increased early reaction.

The study by Svartengren et al (Svartengren, Strand et al. 2000) found that exposure to $PM_{2.5}$ in the road tunnels had no effect on symptoms (Figure 4.12), caused a slight increased in the early phase lung asthmatic reaction, but no change in the late phase reaction (Figure 4.13).

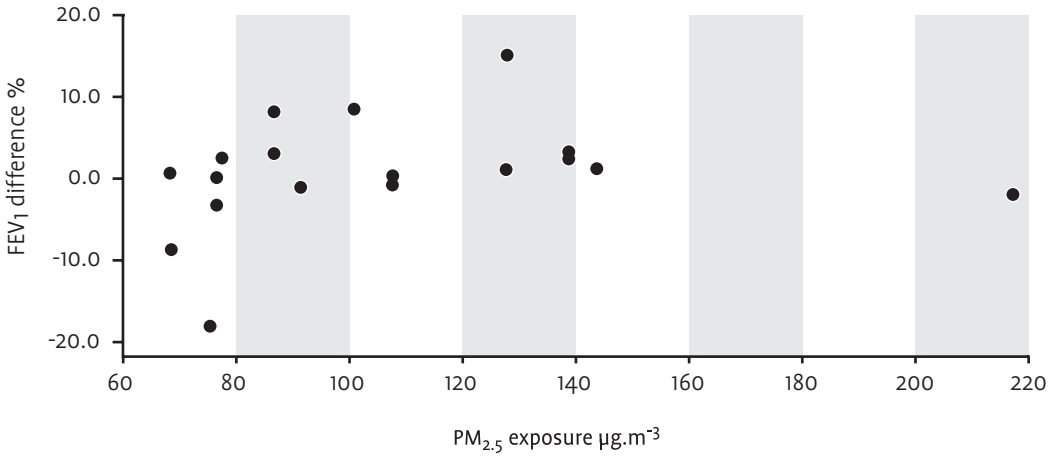
Figure 4.12 Effect of exposure to $PM_{2.5}$ for 30 minutes within the car in a road tunnel followed by allergen challenge on asthma symptoms index during the late phase



Source: (Svartengren, Strand et al. 2000)

Changes ( ) in symptom index during the evening, night and morning after allergen inhalation. Data are expressed as difference from the values obtained on the control day, which was exposure to air followed by allergen challenge. Negative values correspond to a greater effect on the control day compared to tunnel exposure. There was no significant relationship between individual differences between tunnel and control as a function of $PM_{2.5}$ exposure within the car (r^2=0.32, p=0.18) (Svartengren, Strand et al. 2000).

Figure 4.13 Asthmatic reaction during the late phase
Maximal late phase change in forced expiratory volume in one second (FEV_1)



Source: (Svartengren, Strand et al. 2000)

Data are expressed as the difference from the values obtained on the control day. Negative values correspond to a greater effect on the control day compared to tunnel exposure. There was no significant relationship between individual differences between tunnel and control as a function of $PM_{2.5}$ exposure within the car (r^2=0.18, p=0.46) (Svartengren, Strand et al. 2000).

Riediker et al. (Riediker, Cascio et al. 2004) reported on a study of occupationally exposed young, healthy, nonsmoking, male North Carolina highway patrol troopers. Nine troopers (age 23 to 30 years) were monitored on 4 successive days while working a 3 pm to midnight shift. Each patrol car was equipped with air-quality monitors. Blood was drawn 14 hours after each shift, and ambulatory monitors recorded the electrocardiogram throughout the shift and until the next morning. The average in vehicle pollutant concentrations were: $PM_{2.5}$ 24 µg/m^3 (range: 4.5-54.4); carbon monoxide 2.6 ppm (range: 0.9-5.9) and nitrogen dioxide 0.035 ppm (range: 0.002-0.213). There were significant associations for $PM_{2.5}$, but not carbon monoxide or nitrogen dioxide with inflammatory responses measured in blood and increased heart rate variability. Decreased lymphocytes (-11 percent per 10 µg/m^3 increase in $PM_{2.5}$) and increased red blood cell indices (1 percent mean corpuscular volume), neutrophils (6 percent), C-reactive protein (32 percent), von Willebrand factor (12 percent), next-morning heart beat cycle length (6 percent), next-morning heart rate variability parameters, and ectopic beats throughout the recording (20 percent). This small and recent study would suggest that increased exposure to $PM_{2.5}$ during a 9- hour shift does result in change in inflammation, coagulation and cardiac rhythm.

4.6 Panel studies of PM

4.6.1 PM and symptoms in Australia

Jalaludin et al (Jalaludin, Chey et al. 2000; Jalaludin, O'Toole et al. 2004) found no association between PM_{10} and lung function, wheeze, cough or medication use, but a significant association with doctor visits for asthma. A 12 µg/m^3 increase in 24 hour PM_{10} was associated with an 11 percent increase in visits to the doctor for asthma, which was statistically significant ($p<0.05$) (Jalaludin, O'Toole et al. 2004). The study followed 125 children living in southwestern Sydney, aged 10 years, with a history of wheeze for an 11 month period in 1994. A 0.0082 ppm increase in average daytime nitrogen dioxide was associated with an 5 percent increase in parent reported wet cough, which was statistically significant ($p<0.05$) (Jalaludin, O'Toole et al. 2004).

4.6.2 PM and symptoms in Europe

In panel studies of children there were significant associations between daily PM_{10} and both lower respiratory symptoms and peak expiratory flow rate, but not cough, upper respiratory symptoms or medication use (Table 4.6).

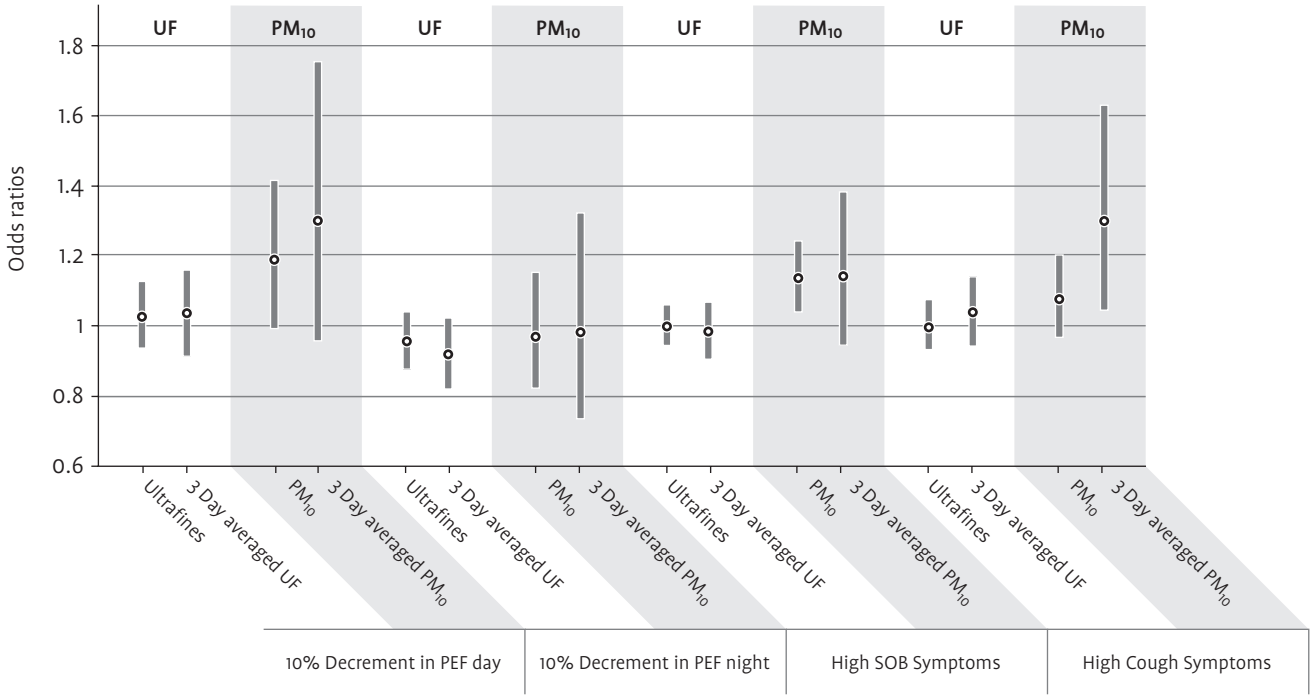
Table 4.6 Odds ratios, beta for PEFR, and (95 percent confidence intervals) estimates from meta-analyses for PM_{10} and respiratory function in children (Relative risks are for a 10 µg/m^3 increase in PM_{10})

Respiratory measure	Estimate (95%CI), # of studies
Peak expiratory flow rate	-0.085 (-0.136 - -0.033), 41 studies
Cough	0.999 (0.987-1.011), 34 studies
Lower respiratory symptoms	1.008(1.000-1.016), 39 studies
Upper respiratory symptoms	0.997 (0.994-0.999), 39 studies
Medication use in symptomatic children	1.005 (0.981-1.029), 31 studies

4.6.3 Particle size and symptoms

Osunyana et al (Osunsanya, Prescott et al. 2001) followed 44 people with COPD over a three month period and found significant associations with lung function and symptoms and both the concentration of PM_{10} and the number ultrafine particles (Figure 4.14). Both measures of particles were significantly associated with lung function measures, however there was no significant difference between PM_{10} and ultrafine particles. Other meta-analyses of PM_{10} versus $PM_{2.5}$ have found that there were too few studies where $PM_{2.5}$ was measured to provide reliable estimates (Anderson, Atkinson et al. 2004).

Figure 4.14 Effect of PM on peak flow in people with COPD. PM was measured as PM_{10} in µg/m³ and as the number of ultrafine particles (PM less than 0.1 micrometer)



Source: (Osunsanya, Prescott et al. 2001)

4.7 Hospital Admissions and PM

Time series studies examine associations between short term community health outcomes and fluctuations in ambient pollutants using regression models. In these studies the pollutant level is averaged over one to a few days and linked to a community health outcome such as hospital admissions or mortality. In assessing the impact of pollutants on the community, it is important to recognise that communities appear to differ considerably in their response to an elevated level of pollutant. The source of this variation is unknown, but may be related to climate, geography, source of pollutants, background level of pollutant, pollutant mixes and physiological differences in the population. It is therefore likely that the studies that are of most relevance to assessing the health impact of changes to air pollutants in Australian cities are likely to be studies that have been performed in Australian cities.

4.7.1 Dose Response Relationships for Australia

Five Australian studies have examined the acute effects of ambient pollutants on hospital admissions (Morgan, Corbett et al. 1998a; Denison, Simpson et al. 2001; Petroeschevsky, Simpson et al. 2001; Department of Environment WA 2003; Simpson, Williams et al. 2005b). The studies were published between 1997 and 2005 and the dose response relationships for each city are summarised in Table 4.7.

The most recent study included a meta-analysis of results for Brisbane, Melbourne, Sydney and Perth (Simpson, Williams et al. 2005b). The study extended from 1 January 1996 to 31 December 1999 for all four cities. Daily hospital admissions in each city and the concentration of ambient particles as measured by nephelometry (BSP; $10^{-4}.m^{-1}$, an indicator of concentrations of fine particles <2 µm in diameter) is given in Table A1, Appendix A. The total population examined for air pollution exposure across all cities was approximately 10 million (53 percent of the Australian population).

The hospital admissions for each disease category and for each city are given in Tables A1 and A2 (Appendix A). Cardiovascular disease admissions were the most common and ranged from a daily average of 14.1 in Brisbane to 84 in Melbourne. Asthma admissions accounted for about one third of all respiratory hospitalisations. Children 0-14 years old are the most likely group in the community to be hospitalised for asthma and account for between 2.5 and 15.5 hospital admissions per day across the four cities.

Ambient PM levels are given in Tables A1 and A2 (Appendix A). One hour maximum BSP was from 0.01-16.2 $10^{-4}m^{-1}$, while 24 hour PM was from 0.01-5.1 $10^{-4}m^{-1}$ (Tables A1 and A2, Appendix A). Sydney had the highest levels of PM, followed by Brisbane, Melbourne and Perth (Tables A1 and A2, Appendix A).

The meta-analysis of all four Australian cities indicated strong effects of increases in BSP on cardiac and respiratory disease hospital admission (Table 4.7) (Simpson, Williams et al. 2005b). For the meta-analyses there were no particle matter data common to all cities apart from the BSP data. However, PM_{10} datasets were available for Brisbane, Sydney and Melbourne and $PM_{2.5}$ datasets were available for Sydney, Perth, and Melbourne. A meta-analysis on these sets of three cities estimated that for a 10 $\mu g/m^3$ increase in PM_{10} concentration the increase in the daily number of cardiac admissions for all ages was 2.4 percent (95%CI: 1.5-3.4 percent) and increase in respiratory admissions in the elderly increased by 2.9 percent (95%CI: 1.3-4.4 percent). Also there was an increase of 5.1 percent (95%CI: 3.5-6.7 percent) in cardiac admissions for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration (Table 4.7).

Table 4. 7 Dose response relationships for increases in hospital admissions as a result of a 1 unit increase in BSP $10^{-4}m^{-1}$ or a 10 ug/m^3 increase in 24 hour PM_{10} or $PM_{2.5}$

City	Hospital admission	Size of effect (% increase)	Lower estimate (% increase)	Upper estimate (% increase)
All 4 cities for BSP (Simpson, Williams et al. 2005b)	Cardiac all ages	8.56	6.03	11.16
	Respiratory 65+ years	5.52	0.82	10.45
	Asthma 15-64 years	8.93	2.4	15.87
All 3 cities for PM_{10} (Simpson, Williams et al. 2005b)}	Cardiac all ages	2.4	1.5	3.4
	Respiratory 65+ years	2.9	1.3	4.4
All 3 cities for $PM_{2.5}$ (Simpson, Williams et al. 2005b)	Cardiac all ages	5.1	3.5	6.7
Sydney (Morgan, Corbett et al. 1998a)	Cardiovascular disease 65+ years	0.97	0.31	1.63
	Respiratory all ages	No effect		
Melbourne (Denison, Simpson et al. 2001).	Respiratory 65+ years	2.42	0.14	4.77
	Asthma all ages	4.45	1.90	7.05
	Cardiovascular disease 65+ years	1.83	0.69	2.99
Brisbane (Petroeschevsky, Simpson et al. 2001).	Respiratory all ages	1.8	0.72	2.77
	Cardiovascular disease all ages	No effect		
Perth (Department of Environment WA 2003).	Respiratory all ages	N/a*	N/a	N/a
	Cardiovascular disease all ages	N/a	N/a	N/a

Note

* n/a=Not available

4.7.1.1 *Sydney*

For Sydney, a 10 $\mu g/m^3$ increase in 24 hour PM_{10} resulted in a statistically significant increase of 0.97 percent in cardiovascular disease in people aged 65 years and over, the lower estimate was a 0.31 percent increase while the upper estimate was a 1.63 percent increase (Table 4.7). The lower and upper estimates are the 95% confidence intervals. There was no effect on respiratory admissions.

The summary estimates (Table 4.7) are based on more extensive analyses which are presented in Table A3 (Appendix A). An increase in 1 hour maximum BSP particulates from 0.25 to 1.48 $10^{-4}m^{-1}$ (~15 - 90 $\mu g/m^3$ PM_{10}) was associated with a 2.72 percent increase in admissions (Table A3, Appendix A). The results for 24-hour average particulates were similar (Table A3, Appendix A), with a 2.82 percent increase in heart disease admissions associated with an increase in particulates from 0.12-0.60 $10^{-4}m^{-1}$ (~7-36 $\mu g/m^3$ PM_{10}). COPD admissions also showed a trend towards increase with particulate levels, however it was not statistically significant.

4.7.1.2 *Melbourne*

The effects of increased PM in Melbourne are summarised in Table 4.7. A 10 µg/m^3 increase in 24 hour PM_{10} resulted in a statistically significant increases of 2.42 percent, 4.45 percent and 1.83 percent in respiratory admissions in people aged 65 years, asthma admissions in all ages and cardiovascular disease in people aged 65 years. All these effects were statistically significant ($p<0.05$).

Table A3 (Appendix A) also indicates fine particles were significantly ($p<0.05$) associated with admissions for respiratory disease (15-64 years, 65+ years), asthma (0-14 years, all ages), cardiovascular disease (65+ years, all ages) and ischaemic heart disease (all ages).

For respiratory admissions in the 15-64 year age group a unit ($1x10^{-4}m^{-1}$) increase in 1 hour BSP (equivalent to 15 µg/m^3 $PM_{2.5}$) was associated with a 3.8 percent increase in risk of admission (3-day lag). In the 65+ year age group, a one unit increase in the 24 hour (equivalent to ~ 4xSD) and maximum 1 hour (equivalent to ~ 2xSD) concentrations of BSP was associated with a 7.5 percent and 4.3 percent, respectively, increase in risk of admission. No significant associations were found for respiratory admissions in the 0-14 year or all age groups.

For asthma, strong associations were observed across most of the lag periods and averaging times examined in both the 0-14 year age group and in the all ages age group. In the 0-14 year age group, a unit increase in same day 24 hour BSP was associated with a 14.8 percent increase in risk of admission (Table A3, Appendix A). In the all ages category, the strongest associations were found with the 5-day cumulative average, where a unit increase in the 24 hour concentration was associated with a 13.9 percent increase in risk of admission for asthma.

Admissions for cardiovascular disease and ischaemic heart disease also showed strong, consistent associations with particles in Melbourne, in particular the 24 hour concentration (Table A3, Appendix A). While only marginally significant associations were found in the 0-64 year age group, admissions for cardiovascular disease in the 65+ years and all ages groups were consistently associated with BSP levels for both 1 hour maximum and 24 hour average. A unit increase in the 24 hour BSP concentration was associated with a 5.6 percent and 4.6 percent increase in risk of admission for cardiovascular disease in the 65+ years and all ages groups, respectively, per unit increase (equivalent to ~ 4xSD) in BSP. For the maximum 1 hour particle concentration a 3.5 percent and 2.7 percent increase in risk of admission per unit increase in BSP was found for cardiovascular disease in the 65+ years age group and all ages group respectively. Similar results were obtained for ischaemic heart disease (Table A3, Appendix A).

The results of the multi-pollutant analysis showed that many of the significant associations observed between hospital admissions and fine particles in the single pollutant analysis were reduced in size and significance after controlling for the effects of other pollutants, in particular nitrogen dioxide and carbon monoxide. Controlling for nitrogen dioxide in the model resulted in a reduction in the size and significance of the association between BSP and admissions for each of the outcomes. The one exception was cardiovascular admissions in the all ages group where the significance of the particle effect was retained after controlling for nitrogen dioxide (Table A3, Appendix A).

4.7.1.3 *Brisbane*

In Brisbane, a 10 µg/m3 increase in 24 hour PM was associated with a significant 1.8 percent increase in all respiratory, but not asthma only or cardiovascular admissions in single- and multi-pollutant models (Table 6 and Table A3, Appendix A) (Petroeschevsky, Simpson et al. 2001) .

For respiratory admissions in all ages (0-65+ years), a unit increase in 24 hour average concentration of BSP (5 day average) resulted in a 1.5 percent increase in hospital admission that was statistically significant ($p<0.05$). Within the 15-64 year old age group, a unit increase in maximum 1 hour BSP (5 day average) resulted in a statistically significant 0.5 percent increase in hospital admission for respiratory conditions. There was a trend toward increased respiratory admissions in 0-4 and 65+ year age groups, although this was not statistically significant (Table A3, Appendix A).

The effect was stronger for 24 hour BSP compared with 1 hour maximum BSP and in multi-pollutant models, after adjusting for high sulphur dioxide and high ozone, respiratory admissions in all ages remained significant (Petroeschevsky, Simpson et al. 2001). This study indicates that in Brisbane a one unit increase in 24 hour average BSP concentration across the range 0.30 –50.8 $10^{-4}m^{-1}$ resulted in a 1.5 percent increase in hospital admissions between 1987-1994 (Table A3, Appendix A).

For asthma only admissions, a subset of respiratory admissions, there were no significant increases in admissions in any of the age groups (Table A3, Appendix A).

For cardiovascular admissions, the most common reason for admission in the study (Table A3, Appendix A), there were no significant increases in admissions in any of the ages groups.

4.7.1.4 *Perth*

Respiratory hospitalisations in people over 65+ years and especially for COPD were significantly related to the level of PM (Table A3, Appendix A). In this study a case cross over analysis was used and the effects were large, however the effects for 24 hour PM were not reported.

4.7.2 Daily Hospital Admissions and PM_{10} in the US

WHO (Health Effects Institute 2004) recently provided summary estimate of the effect of particulate pollution on hospitalisations for respiratory diseases (Table 4.8) in the United States. A 10 $\mu g/m^3$ increase in 24 hour PM_{10} was associated with a 1.5 percent increase in hospital admission for COPD. The estimate was taken from the NMMAPS studies of hospital admissions that covered 10 large metropolitan areas in the United States of America with a combined population of 1,843,000 subjects over 65 years old.

Table 4.8 Estimated effects of air pollution on daily hospital admissions from the APHEA2 and NMMAPS

Study	Disease	Increase in disease admission per 10 $\mu g/m^3$ increase in PM_{10}
APHEA2	COPD and asthma	1.0% (0.4%–1.5%)
Europe	Respiratory, aged 65+ years	0.7% (0.2%-1.3%), 8 studies
NMMAPS	COPD	1.5% (1.0%–1.9%)

Source: (WHO Europe 2003)

4.7.3 Daily Hospital Admissions and PM_{10} in Europe

WHO also reported on the APHEA 2 hospital admission study covered a population of 38 million living in eight European cities, which where studied for three to nine years in the early-mid 1990s.

The Europe estimate (Table 4.8) is based on a later meta-analysis performed by WHO that includes the APHEA 2 studies (Anderson, Atkinson et al. 2004). WHO recently performed a meta-analyses of time series and panel studies (Anderson, Atkinson et al. 2004). Using studies catalogued in bibliographic databases up to February 2003, 629 ecological time series studies and 160 individual or panel studies were identified, and 286 time series and 124 panel studies provided usable data. The two databases contain over 11,700 and 6,400 effect estimates respectively. In WHO's meta-analysis for Europe, sufficient numbers of estimates (>3) for the effect of PM_{10} were available only for respiratory admissions in the 65+ year age group. The relative risk for a 10 $\mu g/m^3$ increase in PM_{10} was 1.007 (95%CI: 1.002, 1.013) and was based upon eight studies. Six of these eight estimates were provided by the APHEA 2 project (Atkinson, Anderson et al. 2001). Unfortunately much of the recently published data on particles and daily admissions for respiratory disease from APHEA 2 could not be used because this study did not report all respiratory admissions in the younger age groups.

According to WHO there were insufficient numbers of studies for other age groups or cardiovascular admissions (Anderson, Atkinson et al. 2004). For the age categories, ages 0–14 and 15–64 years, results were available from three studies conducted in London (Atkinson, Bremner et al. 1999), West Midlands (Bremner, Anderson et al. 1999) and Rome (Michelozzi, Forastiere et al. 2000). Together these cities represent a population in excess of 10 million people. A meta-analysis of results from these three cities gave summary estimates of 1.010 (95%CI: 0.998, 1.021) and 1.008 (95%CI: 1.001, 1.015) per 10 $\mu g/m^3$ increases in PM_{10} for respiratory admissions, ages 0–14 and 15–64 years respectively (Anderson, Atkinson et al. 2004).

4.7.4 Daily Hospital Admissions and $PM_{2.5}$ in Europe

WHO reported that few studies recorded $PM_{2.5}$ levels (Anderson, Atkinson et al. 2004). For fine and coarse particles only tone study provided results for the respiratory outcomes. The relative risks for $PM_{2.5}$ for each of the three age categories, 0–14, 15–64 and 65+ years were 1.091 (95%CI: 0.9994, 1.0391), 0.9881 (95%CI: 0.9633, 1.0135) and 0.9926 (95%CI: 0.9732, 1.0125) respectively. There were no estimates available from the 65+ years, cardiovascular admissions group. Results for coarse particles were similar to those for fine particles.

4.7.5 Daily Hospital Admissions and PM_{10} in Asia

The Health Effects Institute recently performed a meta-analysis of the effect of air pollution on health in Asia (Health Effects Institute 2004). The estimates for PM_{10} and respiratory hospital admissions were calculated for only two cities. Three separate studies in Hong Kong investigated the association between PM_{10} and respiratory hospital admissions for all respiratory diseases combined and for cause-specific admissions (asthma, COPD, and pneumonia). The only other city with a result for respiratory admissions was Seoul (asthma admissions). Each of the Hong Kong studies reported roughly the same increase in risk of hospital admissions for all respiratory causes (Figure 4.15). Only two studies, both from Hong Kong, reported positive estimates in all cardiovascular hospital admissions per unit increase in PM_{10} (Figure 4.16).

Figure 4.15 Percent change in mean number of respiratory hospital admissions per 10µg/m³ increase in 24-hour mean PM_{10} level

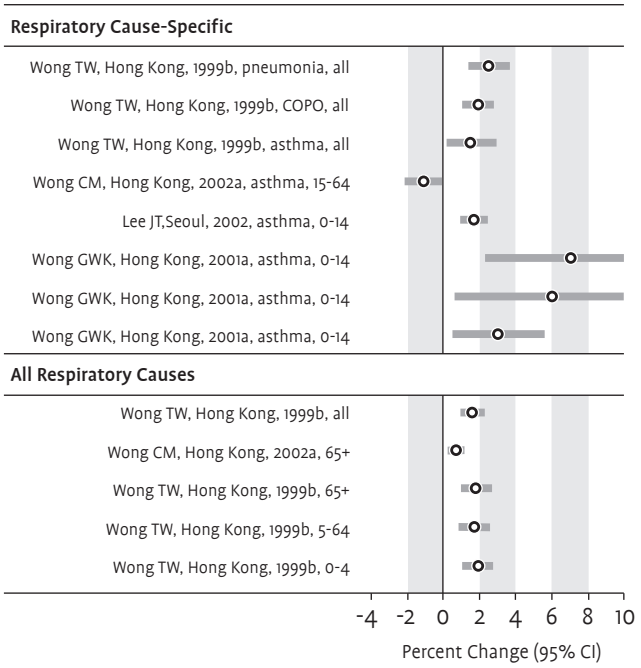
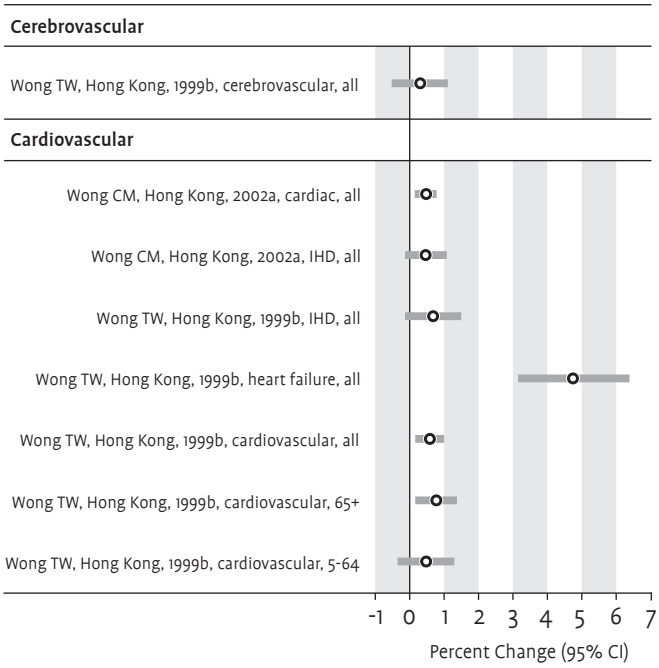


Figure 4.16 Percent change in mean number of cardiovascular hospital admissions per 10µg/m³ increase in 24-hour mean PM_{10} level



Source: (Health Effects Institute 2004)

4.8 Mortality

4.8.1 Dose Response Relationships for Australia

Five Australian studies have examined the relationship between air pollution and mortality and they are summarised in Table 4.9 and details provided in Tables B1- B4, Appendix B (Simpson, Williams et al. 1997; Morgan, Corbett et al. 1998b; Simpson, Denison et al. 2000a; Department of Environment WA 2003; Simpson, Williams et al. 2005a). The studies were published between1997-2005 and data were collected for periods of 4-6 years between 1987 and 1999.

The most recent study included a meta-analysis of results for Brisbane, Melbourne, Sydney and Perth (Simpson, Williams et al. 2005a). The study extended from 1 January 1996 to 31 December 1999 for all four cities. Daily mortality in each city and the concentration of ambient particles (light scattering by nephelometry, BSP $10^{-4}m^{-1}$, which is an indicator of concentrations of fine particles <2 µm in diameter) is given in Table B1, Appendix B. The total population examined for air pollution exposure across all cities was approximately 10 million (53 percent of the Australian population)

In the meta-analysis (Table B1, Appendix B) and the earlier studies (Table B2, Appendix B) cardiovascular disease mortality was on average five times more common than respiratory disease mortality (Tables B1 and B2). Brisbane had the lowest daily mortality, followed by Perth, Melbourne and Sydney. On each day, an average of five people died of respiratory illness in Melbourne or Sydney and 23-29 died from cardiovascular diseases (Tables B1 and B2).

Ambient PM levels are given in Table B1 and B2 (Appendix B). 1 hour maximum BSP was from 0.01-16.2 $10^{-4}m^{-1}$, while 24 hour PM was from 0.01-5.1 $10^{-4}m^{-1}$(Tables B1 and B3, Appendix B). Sydney recorded the highest levels of PM, followed by Brisbane, Melbourne and Perth (Tables B1 and B2, Appendix B).

For the meta-analysis, there were no PM data common to all cities apart from nephelometer data, which is recorded as BSP. When BSP was used as the measure of PM exposure, significant relationships were found for all cause and cardiovascular disease mortality (Table 4.8), but not respiratory disease mortality.

The relationships between BSP and $PM_{2.5}$ or PM_{10} data are not straightforward (Simpson, Williams et al. 2005a). However, there were datasets for PM_{10} for Brisbane, Sydney and Melbourne and $PM_{2.5}$ datasets for Sydney, Perth and Melbourne. The meta-analysis on these three cities estimated the increase in the daily number of deaths for all ages for a 10 µg/m³ increase in PM_{10} concentration to be 0.2 percent (95%CI: -0.8% to 1.2%), and estimated the increase in the daily number of deaths for all ages for a 10 µg/m³ increase in $PM_{2.5}$ concentration to be 0.9 percent (95%CI: -0.7% to 2.5%) (Table 4.9). Thus for BSP, but not PM_{10} or $PM_{2.5}$, there were significant associations between mortality and particulate air pollution in the four Australian cities.

Table 4.9 Dose response relationships for increases in daily mortality as a result of a 1 unit increase in BSP $10^{-4}m^{-1}$ or a 10 ug/m³ increase in 24 hour PM_{10} or $PM_{2.5}$

City	Cause of mortality	Size of effect (% increase)	Lower estimate (% increase)	Upper estimate (% increase)
All 4 cities for BSP (Simpson, Williams et al. 2005a)	All cause	2.84	0.15	5.60
	Cardiovascular	4.79	0.76	8.98
All 3 cities for PM_{10} (Simpson, Williams et al. 2005a)	All cause	0.2	-0.8	1.2
All 4 cities for $PM_{2.5}$ (Simpson, Williams et al. 2005a)	All cause	0.9	-0.7	2.5
Sydney (Morgan, Corbett et al. 1998b)	All cause	1.09	0.36	1.73
	Cardiovascular	1.11	0.10	2.09
Melbourne (Simpson, Denison et al. 2000a)	All cause (warm season only)	1.82	0.70	3.35
	Respiratory all ages (warm season only)	6.06	0.60	11.9
	Cardiovascular	No effect		
Brisbane (Simpson, Williams et al. 1997)	All cause	1.08	0.36	1.80
	Cardiovascular	No effect		
Perth (Department of Environment WA 2003)	All cause	No effect		
	Cardiovascular	No effect		

4.8.1.1 *Sydney*

A 10 µg/m³ increase in 24 hour PM_{10} resulted in a statistically significant increase of 1.09 percent in all cause mortality, which excludes accidental mortality. For all cause mortality the lower estimate was a 0.36 percent increase while the upper estimate was a 1.73 percent increase (Table 4.9). The lower and upper estimates are the 95% confidence intervals. This association with total mortality was significant after adjusting for nitrogen dioxide and ozone. In single pollutant models there was a similar size and significant effect on cardiovascular mortality (Table 4.9), however no significant effect on respiratory mortality (Table B3, Appendix B).

Results for an increase in 1 hour maximum particulates (BSP) from 0.23 to 1.42 $10^{-4}m^{-1}$ (~14-85 µg/m³ PM_{10}) were also significant and are given in Table B3 (Appendix B).

4.8.1.2 *Melbourne*

There were no significant associations between particulate pollution in Melbourne and mortality for the entire year, however there were associations in the warm period (Tables 4.9 and B3, Appendix B) (Simpson, Denison et al. 2000a; Simpson, Denison et al. 2000b).

4.8.1.3 *Brisbane*

In Brisbane there were significant associations between ambient particulate levels and all cause mortality (Tables 4.9 and B3, Appendix B). A 10 µg/m^3 increase in 24 hour PM_{10} resulted in a statistically significant increase of 1.08 percent in all cause mortality, which excludes accidental mortality. For all cause mortality the lower estimate was a 0.36 percent increase while the upper estimate was a 1.80 percent increase (Table 4.9). This association with all cause mortality was significant in single pollutant models only, while there were no significant effects on cardiovascular mortality or respiratory mortality (Table B3, Appendix B).

The effect of PM were also observed for 1 hour maximum BSP and the effect was strongest for people aged 65 years and older. A one unit increase in 1 hour daily maximum BSP resulted in a 0.2 percent increase in total mortality and a 0.4 percent increase in cardiovascular mortality in Brisbane between 1987 and 1993 (Table B3, Appendix B) (Simpson, Williams et al. 1997).

4.8.1.4 *Perth*

There were no significant associations between mortality and PM levels in Perth (Tables 4.9 and B3, Appendix B) (Department of Environment WA 2003).

4.8.2 Overseas studies

There have been a number of summary estimates of the effect of PM on mortality in the United States (Daniels, Dominici et al. 2004), Europe (WHO Europe 2003) and Asia (Health Effects Institute 2004).

4.8.2.1 *Daily Mortality and PM_{10} in Europe*

There were significant associations between PM_{10} and mortality in Europe (Table 4.10). The WHO meta-analysis (Anderson, Atkinson et al. 2004) found that a 10 µg/m^3 increase in 24 hour ambient PM_{10} was associated with significant increases in all-cause (0.6 percent), respiratory (1.0 percent) and cardiovascular (0.5 percent) mortality. These estimates were largely from the APHEA 2 study. The APHEA 2 mortality study covered a population of more than 43 million living in 29 European cities, which were all studied for > 5 years in the early-mid 1990s.

4.8.2.2 *Daily Mortality and PM_{10} in the US*

A recent reanalysis of a meta-analysis of the National Morbidity and Mortality Air Pollution Study (Daniels, Dominici et al. 2004) reported that a 10 µg/m^3 increase of PM_{10} was associated with a 0.34 percent (95% CI: 0.18%, 0.51%) increase in cardiovascular-respiratory mortality and a 0.28 percent (95% CI: 0.16%, 0.41%) for total mortality (Table 4.10). The NMMAPS examined the 20 largest metropolitan areas in the US with a population of more than 50 million people over the period 1987–1994.

Table 4.10 Relative risk estimates (95 percent confidence intervals) from meta-analyses for PM_{10} and all-cause and cause-specific mortality. Relative risks are for a 10ug/m^3 increase in daily PM_{10}

Region	Outcome and age	Summary estimate (95%CI), # of studies
Europe (Anderson, Atkinson et al. 2004)	All-cause mortality, all ages	1.006 (1.004-1.008), 33 studies
	Respiratory mortality, all ages	1.010 (1.001-1.018), 20 studies
	Cardiovascular mortality, all ages	1.005 (1.001-1.010), 23 studies
United States (Daniels, Dominici et al. 2004)	All-cause mortality, all ages	1.003(1.001-1.004), 20 cities
	Cardiovascular-respiratory mortality, all ages	1.003(1.002-1.005), 20 cities

4.8.2.3 *Daily Mortality and PM_{10} in Asia*

The Health Effects Institute recently performed a meta-analysis of the effect of air pollution on health in Asia (Health Effects Institute 2004). Eleven estimates for PM_{10} and daily mortality were reported from four cities: Bangkok, Seoul, Inchon, and Hong Kong. All estimates indicated increased risk for mortality at all ages. Only four estimates were available for the effect

of exposure to PM_{10} on daily all-cause, all-age mortality in different cities. The effect of a 10 μg/m³ increase in PM_{10} was a 0.49 percent (95%CI: 0.23,0.76) increase in risk of all-cause daily mortality.

Estimates for cause-specific mortality were too few to permit calculation of summary estimates. Nine reports of daily respiratory deaths were available from the same four cities: Bangkok, Seoul, Inchon, and Hong Kong. All estimates indicated an increased risk. The same four cities were the source of estimates of PM_{10} and daily cardiovascular deaths. Risk for all cardiovascular deaths from three cities was elevated; the estimates varied in size and precision.

4.8.3 *Daily Mortality and $PM_{2.5}$*

WHO presented a meta-analysis for the effect of $PM_{2.5}$ on mortality (Anderson, Atkinson et al. 2004) (Table 4.11). Too few studies have been performed in Europe for analysis, therefore the analysis was extended to include all studies, regardless of geographical location. For all cause mortality, 23 studies from around the world were considered, including 15 from the United States and Canada. In the global meta-analysis, a 10 ug/m3 increase in 24 hour $PM_{2.5}$ was associated with significant increases in daily: all cause mortality (0.9 percent); cardiovascular (1.3 percent); and respiratory mortality (1.1 percent) (Table 4.11). The effect estimates were slightly higher in the United States and Canada, although not significant for respiratory mortality. Only one to three European studies were considered in the analyses and there were no significant effects.

Table 4.11 Relative risk estimates (95 percent confidence intervals) from meta-analyses for $PM_{2.5}$ and daily mortality. Relative risks are for a 10 ug/m³ increase in $PM_{2.5}$

	All Cause (95%CI), # studies	**Cardiovascular (95%CI), # studies**	**Respiratory (95%CI), # studies**
US and Canada	1.013 (1.008-1.018), 15 studies	1.023 (1.003-1.044), 4 studies	1.016 (0.994-1.038), 4 studies
Global	1.009 (1.006-1.013), 23 studies	1.013 (1.005-1.022), 8 studies	1.011 (1.002-1.020), 8 studies
Europe	3 studies with no significant effect, RRs=1.003, 1.006 and 0.98 for the three studies	1.005 (0.998-1.022), 1 study	0.994 (0.969-1.031), 1 study

Source: (Anderson, Atkinson et al. 2004)

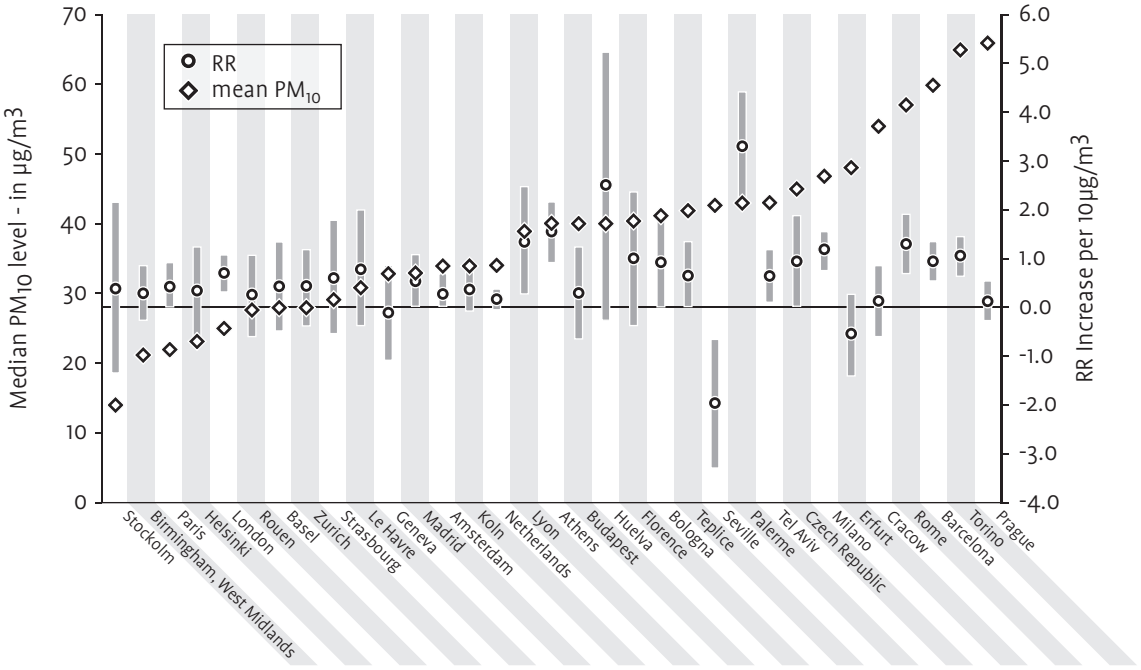
4.8.4 Variation Between Cities

While meta-analyses provide summary estimates for regions, it should be noted that considerable variation exists between cities. Figure 4.17 shows the individual variation in mortality associated with a 31.3 μg/m³ increase in $PM_{10,}$ which ranged from −4 to +6 percent. This meta-analysis of 48 studies found a 2 percent increase in mortality resulting from a 31.3 μg/m³ increase in $PM_{10.}$ Furthermore the increased relative risk for a city appears to be independent of the level of ambient PM_{10} (Figure 18) (Anderson, Atkinson et al. 2004).

Figure 4.17 The association between mortality and ambient PM_{10}

Source: (Stieb, Judek et al. 2002)

Figure 4.18 The association relative risk of all cause mortality for a 10 µg/m³ increase in PM_{10} and ambient annual average PM_{10}



Source: (Anderson, Atkinson et al. 2004)

4.9 Long term effects of ambient exposure

Elevated levels of air pollutants impact both acutely and in the longer term on health. The acute effects are easier to quantify and therefore provide more robust estimates of likely health outcomes. The chronic effects of air pollution are inherently more difficult to interpret than those reporting immediate or short-term effects. There is potential for confounding factors to yield spurious associations between average pollutant exposures and the incidence or prevalence of chronic disease. Some of the repeated associations that have been found are effects on lung function growth in children and adult mortality.

4.9.1 Lung function growth

4.9.1.1 *Southern Californian Children's Health Study*

The Southern Californian Children's Health Study (SCCHS) examined lung function growth in children over 8 years, between 1993 and 2001. The study examined lung growth and air pollution levels across each of 12 communities in Southern California. The primary source of pollutants was motor vehicles.

In the first cohort 3,035 children were tested in 1993 and subsequently on several occasions up to 1997. For each of the 12 areas, approximately 150 children in grade four, 75 in grade seven and 75 in grade 10 were selected from public schools. Over the study period, lung function was assessed on at least two occasions and on average 3.8 occasions. Three measures of lung function were made on each occasion and were: FVC, FEV_1, and maximal mid expiratory flow rate (MMEF). Levels of nitrogen dioxide, ozone, PM_{10}, $PM_{2.5}$ and inorganic acid vapour ($HCl+HNO_3$) were measured.

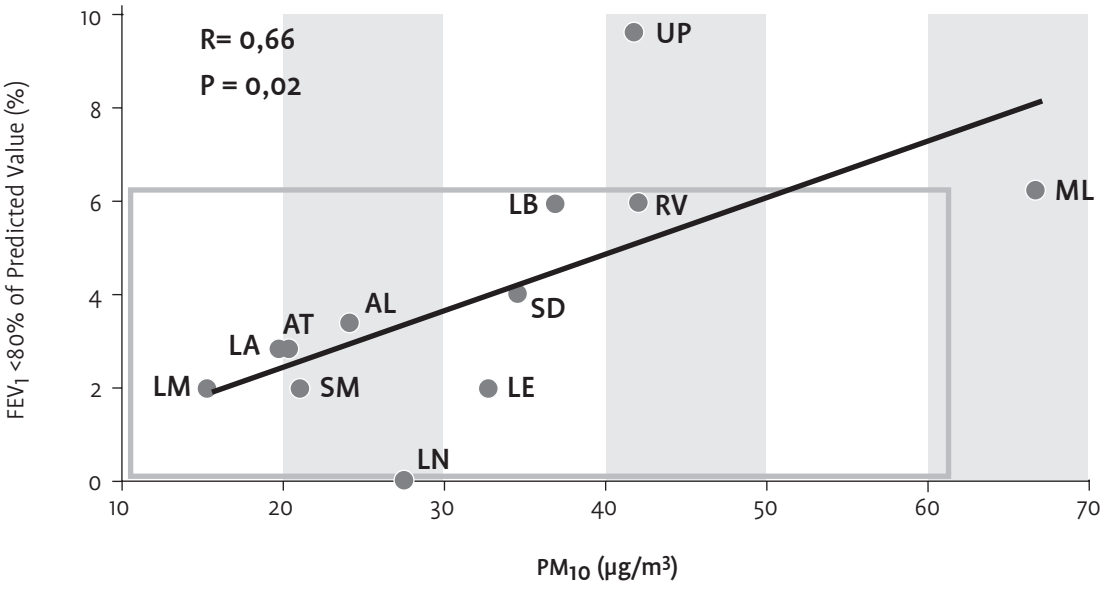
There were four-fold and five-fold differences, respectively in PM_{10} and $PM_{2.5}$ between the least and most polluted communities in the SCCHS. For PM_{10} the range in annual 24 hour average was 16.1 – 67.6 µg/m³. In the fourth grade cohort (1,498 children) annual lung function growth was from ~ 11 to 12.2 percent. Lung function growth was greatest in the communities with low particulate exposure and lowest in those with high particulate exposure. For both PM_{10} and $PM_{2.5}$, these relationship were statistically significant (R=-0.57, p=0.03 and R=-0.52, p=0.05, respectively). For the seventh and 10th grade cohorts, higher particulate exposure resulted in lower lung function growth, however in these cohorts the effect was not significant. A 51.5 µg/m³ increase in annual 24 hour PM_{10} exposure was associated with an annual decrease of 0.58 percent in FVC, 0.85 percent in FEV_1, 1.32 percent in MMEF and 1.36 percent in FEF_{75}, which were all significant ($p<0.05$). A 29.5 µg/m³ increase annual average two week $PM_{2.5}$ exposure was associated with an annual decrease of 0.47 percent in FVC, 0.64 percent in FEV_1, 1.03 percent in

MMEF and 1.31 percent in FEF_{75}, which was statistically significant for MMEF and FEF_{75} ($p<0.05$). When lung function growth was examined over a four year period those living in the highest PM_{10} exposure communities had a lung function growth that was 94-97.5 percent of those in low PM_{10} communities. Exposure to high levels of PM_{10} had a more adverse impact on lung function growth than exposure to environmental tobacco smoke (Gauderman, McConnell et al. 2000).

The findings were confirmed for $PM_{2.5}$ in a second cohort of 1,678 fourth grade children, although they were of a lesser magnitude than for the first cohort (Gauderman, Gilliland et al. 2002). A 22.2 µg/m³ increase in annual average $PM_{2.5}$ exposure was associated with an annual decrease of 0.14 percent in FVC, 0.39 percent in FEV_1 and 0.94 percent in MMEF which was statistically significant for MMEF ($p<0.05$).

Follow-up of the first SCCHS cohort from the age of 10 to 18 years, has confirmed the impact of high nitrogen dioxide and $PM_{2.5}$ on lung development (Gauderman, Avol et al. 2004). The significance of this study is that lung growth in females is almost completed by 18 years and in males growth has slowed considerably, thus it is less likely that lung function growth after these ages will compensate for low lung function at age 18 years. Over the eight-year period, deficits in the growth of FEV_1 were associated with exposure to nitrogen dioxide ($p=0.005$), acid vapor ($p=0.004$), $PM_{2.5}$ ($p=0.04$) and elemental carbon ($p=0.007$) (Table 4.16). Associations were also observed for other lung function measures. The net result of these changes in lung function over an eight year period was that for a 51.5 µg/m³ increase in annual average PM_{10} there was a 6 percent increase in the number of 18 year olds who had a clinically significant lower lung function (FEV_1 less than 80 percent of predicted) (Figure 4.19). These decreases in lung function growth were similar to those reported for maternal smoking, but smaller than those reported fro the effects of personal smoking (Gauderman, Avol et al. 2004).

Figure 4.19 The association between lung function growth in children and ambient PM_{10}



Source: (Gauderman, Avol et al. 2004)

In Figure 4.19, lung function growth is expressed as the community-specific proportion of 18-year-olds with a FEV_1 below 80 percent of the predicted value. Ambient PM_{10} is the annual average level between 1994 through 2000. The communities were: AL=Alpine, AT=Atascadero, LE=Lake Elsinore, LA=Lake Arrowhead, LN=Lancaster, LM=Lompoc, LB=Long Beach, ML=Mira Loma, RV=Riverside, SD=San Dimas, SM=Santa Maria, and UP=Upland.

Exposure to pollutants was associated with clinically and statistically significant deficits in the FEV_1 (<80 percent of predicted) attained at the age of 18 years (Table 4.12). For example, the estimated proportion of 18-year-old subjects with a low FEV_1 (defined as a ratio of observed to expected FEV_1 of less than 80 percent) was 4.9 times as great at the highest level of exposure to $PM_{2.5}$ as at the lowest level of exposure (7.9 percent versus 1.6 percent, $p=0.002$).

Table 4.12 Impact of ambient air pollution of lung function growth rate in children

Pollutant	FVC*		FEV1*		MMEF*	
	Difference (ml)	p value	Difference (ml)	p value	Difference (ml)	p value
NO_2	-95.0	0.05	-101.4	0.005	-211.0	0.02
Acid vapour	-105.2	0.03	-105.8	0.004	-165.0	0.07
PM_{10}	-60.2	0.33	-82.1	0.08	-154.2	0.16
$PM_{2.5}$	-60.1	0.24	-79.9	0.04	-168.9	0.06
Elemental carbon	-77.7	0.08	-87.9	0.007	-165.5	0.04

Note

* Values are the differences in the estimated rate of eight-year growth at the lowest and highest observed levels of the indicated pollutant. Differences are scaled to the range across the 12 study communities in the average level of each pollutant from 1994 through 2000 as follows: 37.5 ppb of O_3 (measured from 10 a.m. to 6 p.m.), 46.0 ppb of O_3 (the one-hour maximal level), 34.6 ppb of NO_2, 9.6 ppb of acid vapor, 51.4µg of PM_{10} per cubic meter, 22.8 µg of $PM_{2.5}$ per cubic meter, 1.2 µg of elemental carbon per cubic meter, and 10.5 µg of organic carbon per cubic meter.

Source: (Gauderman, Avol et al. 2004)

Avol et al. (Avol, Gauderman et al. 2001) investigated the effect of relocation on lung growth in 110 children who relocated at the age of 10 years and were followed up at age 15 years. Relocation of SCCHS children from an area with low PM_{10} to an area with 10 µg/m^3 higher PM_{10} would result in a 2.3 percent reduction in FEV_1 and a 5.7 percent reduction in MMEF. While this study demonstrated a negative effect of higher PM_{10}, it also demonstrated that the effect of PM_{10} was somewhat reversible, since in younger children who moved from a high PM_{10} exposure area to a low exposure area had an improvement in lung function.

Horak et al (Horak, Studnicka et al. 2002) followed on from earlier work by Frischer et al (Frischer, Studnicka et al. 1999) and examined lung function growth in 975 Austrian school children (grades 2-3) in eight communities with different levels of pollution. An increase in 10 µg/m^3 of PM_{10} was associated with a decrease in FEV_1 growth of 84 ml/year and also a decrease in MEF_{25-75}.

4.9.2 Mortality and lung cancer

There is evidence for an independent long-term PM effect on lung cancer mortality or total mortality (Health Effects Institute 2004).

Both the incidence and mortality for lung cancer was strongly associated with long-term concentrations of PM_{10} among males of the 6,338 non-smoking adults participating in the Adventist Health and Smog (AHSMOG) study and followed from 1977 to 1992. In both men and women, PM_{10} showed a strong association with mortality from non-malignant respiratory disease and lung cancer in males (Abbey, Nishino et al. 1999).

Pope et al (Pope, Burnett et al. 2002) found significant associations between long-term $PM_{2.5}$ exposure and lung cancer, cardiovascular or total mortality. This large study in the United States linked mortality and air pollution data for 500,000 people across 50 States, who were followed for 18 years from 1982 to 1998. Each 10 µg/m^3 increase in $PM_{2.5}$ was associated with a 4 percent, 6 percent and 8 percent increase in total, cardiopulmonary and lung cancer mortality. The effects were not consistent for PM_{10}, although there was a trend toward higher cardiopulmonary mortality with higher PM_{10}. There were no associations between long-term NO_2 levels and mortality.

4.10 Summary

- PM remains a significant air pollution problem in all major cities around the world
- Inhaled particulate matter induces inflammation in the lungs that results in increased respiratory symptoms, reduced lung function and changes to cardiac rhythm
- The health effects of particulate matter are manifest both acutely and in the long term
- The acute health effect of PM are increased respiratory and cardiovascular morbidity and mortality
- The long-term health effects of PM are reduced lung function growth in children and increase risk of cancer in adults

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05.
Health Cost of Ambient Air Pollution

The US EPA has been mandated to set air quality standards that protect public health, that is, the standards are based on the effects on health rather than on the basis of costs or other economic considerations (US EPA 1997). However, the US EPA does conduct cost benefit analyses to inform public debate.

> Valuing the health impacts of air pollution can be difficult. There are both methodological and ethical issues, some even arguing that it is not possible to place a monetary value on illness and death. But the fact remains that governments have to make choices about health interventions or investments. The omission of health costs can seriously affect the results of such decision-making processes.

A number of steps need to be taken when determining the health costs of air pollution (or conversely, the health benefits of reducing air pollution):

- Identifying the population's exposure to air pollutants
- Identification of the health impacts due to air pollution and associated exposure-response functions
- Determining the magnitude of the health impacts
- Estimating the monetary values for the associated morbidity and mortality impacts

Each of these steps is associated with a degree of uncertainty. However, the uncertainties in the estimates should not preclude the use of cost-benefit analyses in decision-making.

5.1 Australian studies of health costs of air pollution

5.1.1 Victorian Transport Externalities Study (RCG/Hagler Bailly 1994)

RCG/Hagler Bailly Inc, USA, was commissioned by the Victorian EPA to review the health effects and determine the health costs of ozone and air toxics in Melbourne that result from motor vehicles.

The estimates of health effects were sourced from the epidemiological literature. Health effects due to ozone that were quantified for this study were: premature mortality, asthma attacks in people with pre-existing asthma, minor restricted activity days and days with acute respiratory symptoms. Chronic effects and some other acute effects were not considered because of lack of evidence. Therefore, the estimates of health effects due to ozone will most likely be an underestimate.

The dose-response relationships in this report are derived from epidemiological studies conducted in the United States, on the assumption that the levels of ambient ozone in Melbourne and the United States are similar to each other. It was assumed that there was a threshold for ozone (0.08 ppm) below which they were no health effects. Here again, if there were to be health effects at ozone levels below 0.08 ppm, the health costs would be underestimated.

The only health endpoint for air toxics was cancer. Unit risk factors derived from the US EPA were used to estimate the number of cancers attributed to air toxics. It was assumed there was no threshold for carcinogens. The estimate of cancer cases attributable to air toxics were upper bound estimates as unit risk factors for cancers are based on upper 95 percent confidence levels.

A further two very important assumptions are made:

- The measured effect of ozone in the United States is not affected by any synergism with other pollutants that are present in the United States at different levels than in Melbourne.
- The Melbourne population has similar average sensitivities to ozone as the United States population.

The costs were based on willingness-to-pay (WTP) estimates except for non-fatal cancer where a cost-of-illness (COI) approach was used. A statistical life was valued at 1992A$5 million. Table 5.1 presents the cancer effects from exposure to air toxics due to motor vehicle emissions at 1990 levels and projected levels in 2005. Health costs are greater when a lower survival is assumed (11 percent versus 51 percent). Number of cancer cases attributable to air toxics are projected to decrease except for cancers attributable to diesel particulates, which are expected to increase (with an associated increase in health costs).

Table 5.1 Estimated cancer effects and monetary values of motor vehicle air toxic emissions

Hazardous emissions	Year	Estimated new cancer cases per year*	Estimated annual cancer monetary value (A$ million)	
			11% survival	51% survival
Diesel particulates	1990	1.6 to 8.1	$7.2 to $36.5	$4.0 to $20.3
	2005	2.6 to 13.0	$11.7 to $58.5	$6.5 to $32.5
Petrol particulates	1990	6.8	$30.6	$17.0
	2005	2.2	$9.9	$5.5
Benzene	1990	0.97	$4.4	$2.4
	2005	0.70 to 1.90	$3.2 to $8.6	$1.8 to $4.8
1, 3-butadiene	1990	0.47 to 1.3	$2.1 to $5.9	$1.2 to $3.3
	2005	0.37 to 1.6	$1.7 to $7.2	$0.9 to $4.0
Formaldehyde	1990	0.53	$2.4	$1.3
	2005	0.30 to 0.37	$1.4 to $1.7	$0.8 to $0.9
Benzo[*a*]pyrene	1990	0.03	$0.14	$0.08
	2005	0.01	$0.05	$0.03
Asbestos	1990	0.0001 to 0.3	$0.0005 to $1.4	$0.0003 to $0.8
Total	1990	10.4 to 18.0	$46.8 to $81.3	$26.0 to $45.2
	2005	6.2 to 19.1	$28.0 to $86.0	$15.5 to $47.7

Notes

***** Estimated cases from Hearn (Hearn 1992)

Source: (RCG/Hagler Bailly 1994)

Table 5.2 summarises the health costs for ozone and air toxics in 1992 Australian dollars. The health costs of air toxics are far greater than that due to ozone and are due to the fact that there were more deaths attributable to air toxics (10 to 18 deaths per year) compared to ozone (0.4 deaths per year), and that a statistical life was valued at A$5 million.

Table 5.2 Estimated annual monetary value for health effects due to ozone and air toxics in Melbourne (A$1992 millions)

Air Pollutant	Low estimate	Central estimate	High estimate
Ozone*			
	0.36	2.52	5.28
Air toxics	Estimated new cancer cases	Estimated annual monetary value (A$1992 millions)	
1990	10.4 to 18	26.0 to 45.2	

Notes

***** Estimates that 85 percent of Melbourne's ozone is from motor vehicle precursors.

Source: (RCG/Hagler Bailly 1994)

5.1.2 Review of health costs of road vehicle emissions (NRTC 1995)

This report was commissioned by the National Road Transport Commission (NRTC) and was prepared by L Segal Economic Consultants. The report reviews studies designed to quantify health impacts of motor vehicle emissions on the Australian community. Health costs of vehicle emissions are also presented.

In the current NRTC study, only two health points were taken into account - additional cancer (all cancers) risks due to air toxics and additional respiratory illness (all respiratory illness) on high ozone days. Additional cancer cases were derived from the work of Hearn (Hearn 1992) (see also the study by RCG/Hagler Bailly). However, it is not clear which air toxics have been included in this analysis. For ozone, a threshold of 0.08 ppm was assumed.

This report not only summarises a number of Australian that have determined the health costs for ozone and air toxics (Table 5.3) but also presents an independent assessment on health costs of road vehicle emissions.

Table 5.3 Estimated value of health impact of motor vehicle emissions, key studies from Australia

Pollutant	Estimated health costs of motor vehicle emissions A$ million	Source	Basis
Ozone	0.36 to 5.28	(RCG/Hagler Bailly 1994)	For Melbourne statistical division only. Based on USA health literature. Includes cost of health services, morbidity and mortality.
Air toxics	26.0 to 45.2		
Total	26.4 to 50.5		
All pollutants	787	(ISC 1990)	Estimate of motor vehicle costs for Australia, based on US Federal Highway Cost Allocation Study and reflects emissions of 1977 US road fleet. No discussion of validity of extrapolation to Australia.
Ozone	0.18 to 3.48	(Woodward, McMichael et al. 1993)	Estimate for Australia. Ozone only. Excess asthma morbidity postulated as the only health effect of ozone.
Ozone	14	(NRTC 1995)*	Estimate for Australia 1989-90. Based on 0.1% of cancers (for air toxics) and 0.1% of respiratory diseases (for ozone) attributed to road vehicle emissions. Includes cost of health services, notional values for loss of quality of life and premature mortality.
Air toxics	32		
Total	46		

Notes

* Current study

Source: (NRTC 1995)

5.1.3 An economic evaluation of the health impacts of air pollution in the BCC area (Simpson and London 1995)

The aim of this report was to estimate the public health costs of current levels of air pollution (PM_{10} and ozone) in the Brisbane City Council area. This study considers the health costs of all anthropogenic PM_{10} (motor vehicles and industrial emissions) and ozone rather that the specific contribution of motor vehicles.

The authors suggest that there is some question whether an ozone threshold exists. They consider the impact of not reaching the standard/guideline for a maximum 1-hour ozone level of 0.08 ppm, and therefore it is equivalent to the calculation of the impact of a relative change in ozone concentration.

The authors have relied heavily on previously published reports for the dose-response functions. A number of morbidity end points were also considered in this study. The authors note that the morbidity end points used in their study are not an exhaustive list of potential morbidity effects, but rather those for which reasonable information exists on dose-response relationships. Therefore, any analyses based solely on the morbidity effects used in their study would underestimate the true morbidity impacts.

For PM_{10}, no threshold or a threshold of 30 ug/m^3 was assumed and for ozone a threshold of 0.08 ppm was assumed. For this summary, the no threshold results are being reported. The value of a life was assumed to be A$5 million.

The health effects and health costs are given in Table 5.4. The total health cost of PM_{10} and ozone is between A$254.8 and A$462.3 (1994 dollars). The impact of particulates on mortality dominates the health costs, accounting for about 90 percent of the total costs. The costs of ozone are considerably less.

Table 5.4 Health costs associated with current air pollution in the City of Brisbane (values in millions of 1994 Australian dollars)

Health indicator	Air pollutant	Increase in health indicator per year	Value per unit (1994 A$)	Health costs (in 1994 A$ millions)	
				Low value	High value
Mortality	PM_{10}*	46-83 deaths	5,000,000.00	230	415
	Ozone	0 deaths	5,000,000.00	0	0
Morbidity					
Sore throat	Ozone	104,475-313,425 days	2.80	0.28	0.88
Cough	Ozone	68,510-205,533 days	2.10	0.14	0.43
Headache	Ozone	60,697-182,091 days	3.85	0.23	0.70
Chest discomfort	Ozone	33,702-101,106 days	4.55	0.15	0.46
Eye irritation	Ozone	0 days	2.45	0.00	0.00
Minimal restricted activity day	PM_{10}	264,492-476,085 days	32.15	8.5	15.4
	Ozone	0-1,423 days	32.15	0.00	0.05
Restricted activity day	PM_{10}	220,183-396,183 days	74.15	16.3	29.4
Asthma attack	Ozone	67-202 attacks	4480	0.003	0.009
Acute respiratory symptoms	Ozone	107-1,381 days	9.15	0.001	0.22
Total**				254.8	462.3

Notes

* Zero threshold assumed for PM_{10}

** The Low Total ignores the ozone symptoms contribution and the High Total ignores the ozone values for asthma attacks and acute respiratory symptoms as it is assumed that there is overlap in these data.

Source: (Simpson and London 1995)

5.1.4 Preliminary economic analysis of adopting new vehicle emission standards (NSW EPA 1999)

This aim of this report was to provide preliminary estimates of the costs and benefits of adopting more stringent emission standards for new motor vehicles in Australia. The costs of adopting Euro 2 and Euro 3 standards are calculated as well as the health benefits of reducing emissions.

The report investigated five scenarios:

1. Euro 2 standards introduced in 2002/2003
2. Euro 2 standards introduced in 2002/2003 and then Euro 3 standards introduced in 2005/2006
3. Euro 3 standards introduced in 2002/2003
4. Euro 3 standards introduced in 2005/2006
5. Petrol cars adopt Euro 2 in 2002/2003, then Euro 3 in 2005/2006, Heavy duty diesel vehicles adopt Euro 3 in 2002/2003

Motor vehicles are estimated to contribute up to 70 percent of total air pollution (80 percent of all oxides of nitrogen emissions, 50 percent of hydrocarbon emissions and 90 percent of carbon dioxide emissions).

The health costs of the air pollutants in this report were derived from the literature (Table 5.5). Estimates for ozone were not provided as it is a secondary pollutant and including the effects of ozone may lead to double counting of the overall effects of air pollutants.

In this report the value of a statistical life was estimated at A$7.38 million. This figure was adopted from the US EPA (US EPA 1997).

Table 5.5 Health costs per tonne of pollutant in Australian dollars (A$)

Pollutant	Median (A$)	Range (A$)
Particulates	$39,045	Not provided
Hydrocarbons	$1,440	$90 to $10,130
Nitrogen dioxide	$1,385	$3.50 to $6,000
Ozone	Not estimated	Not estimated
Carbon monoxide	$12	$6 to $45

Source: (NSW EPA 1999)

The costs and benefits, over 20 years, of adopting Euro standards in the five scenarios are given in Table 5.6.

Table 5.6 Net benefits* of Euro standards (A$NPV million 1999)

Scenario	1	2	3	4	5
	Euro 2 from 2002/03	**Euro 2 from 2002, then Euro 3 from 2005/06**	**Euro 3 from 2002/03**	**Euro 3 from 2005/06**	**Petrol cars-Euro 2 from 2002/03 and then Euro 3 from 2005/06; Heavy diesel vehicles –Euro 3 from 2002/03**
Costs					
Technology and hardware	662	831	803	623	807
Fuel reformulation	-	1,199	1,287	1,199	1,084
Laboratory upgrades	-	3	4	3	3
Certification	70	65	70	57	63
Total costs	732	2,098	2,164	1,882	1,957
Benefits*					
Hydrocarbons	80	701	892	611	630
Nitrogen dioxide	409	1,150	1,409	963	1,071
Carbon monoxide	38	793	882	621	884
Particulates	324	793	882	621	884
Unquantified benefits: these include personal and social costs avoided, investment opportunities, visual amenity, export potential, infrastructure damage avoided and reduced greenhouse emissions	Not quantified	Not quantified	Not quantified	Not quantified	Not quantified
Total benefits	851	2,716	3,523	2,423	2,762
Net Benefits	**119**	**618**	**1,359**	**541**	**804**

Notes

* All figures in Net Present Values (NPV) discounted at 7% over 20 years

Source: (NSW EPA 1999)

The report suggests that adopting Euro 3 standards for all vehicles in 2002/3 (scenario 3) would produce a net gain to the Australian community in excess of $1.3 billion over a twenty year period. The results can be considered conservative as a wide range of other benefits, and the health costs of ozone and benzene were not considered in the analyses.

5.1.5 Fuel quality and vehicle emissions standards: cost benefit analysis (Coffey Geosciences 2003)

Coffey Geosciences Pty Ltd. was commissioned by the Department of Environment and Heritage to undertake a cost benefit analysis of four options for future fuel quality.

This study addresses costs and benefits which would accompany implementation of further tightening standards in the second half of this decade. Costs considered include the costs of motor vehicle technology to improve emissions performance and fuel efficiency and capital and operating costs to provide improved motor vehicle fuels.

Benefits considered include the health benefits associated with improved air quality and the savings resulting from improved fuel efficiency. The study covered the period 2000 to 2020.

The cost benefit analysis considered four options for development of vehicle emissions and fuel quality standards in Australia:

- Option 1 - Status Quo – Implemented standards are considered to remain in place indefinitely;
- Option 2 – Mandate Euro 4 – The European standard known as Euro 4 is considered to apply to petrol vehicles from 2008 for new models and from 2009 for all models and to support this standard the sulphur content of petrol in premium unleaded petrol would be reduced to 50ppm from 2008;
- Option 3 – Mandate Euro 4/5 – As for Option 2 but with Euro 5 standards applying for heavy diesel vehicles from 2009 (new models) or 2010 (all models) with sulphur in diesel limited to 10ppm from 2009; and
- Option 4 – Mandate Euro 4/5 10ppm in petrol – As for Option 3 but with a 10ppm limit on sulphur applied to premium grades of petrol from 2010.

The results of this cost benefit analysis are intended to inform debate surrounding the nature and timing of introduction of vehicle emission and fuel quality standards after 2006, in particular, to support the Motor Vehicle Environment Committee's review of fuel quality and vehicle emissions standards for new vehicles.

With Options 2 to 4, the reductions anticipated from 2000 to 2010 for carbon monoxide were 15.3 percent for all three options, for oxides of nitrogen the reductions were 3.4 percent, 5.8 percent and 7.4 percent respectively, for hydrocarbons they were 4.6 percent, 4.7 percent and 6.5 percent respectively, and for PM_{10} they were 0 percent, 0.21 percent and 0.21 percent respectively.

The reductions anticipated from 2000 to 2020 for carbon monoxide were 69.9 percent for all three options, for oxides of nitrogen the reductions were 14.9 percent, 36.3 percent and 37.0 percent respectively, for hydrocarbons they were 21.4 percent, 21.9 percent and 22.7 percent respectively, and for PM_{10} they were 0 percent, 0.33 percent and 0.33 percent respectively.

Premature deaths and hospitalization were the only health effects that were taken into account. The value of a premature death was taken to be A$5 million.

Table 5.7 gives the estimates of the health costs for various air pollutants and Table 5.8 gives the health costs averted for the four options at baseline (2000), 2010 and 2020 for the eight capital cities of Australia.

Table 5.7 Average cost per tonne of emission for eight Australian cities

Emission Type	Air Quality Impact	Health Savings ($/tonne)
Carbon monoxide	Carbon monoxide	$12.9
Oxides of Nitrogen	Nitrogen Dioxide	$58.7
Oxides of Nitrogen	Ozone	$8,500
Particulates (PM_{10})	Particulates (PM_{10})	$232,000
Hydrocarbons	Air toxic emissions	$2,200 [A]

Notes

A Health savings associated with hydrocarbon emissions based on US EPA (2003a) values.

Source: (Coffey Geosciences 2003)

Table 5.8 Health savings projected for each option (A$ million)

Year	Option 2			Option 3			Option 4		
	Estimate	Lower bound	Upper bound	Estimate	Lower bound	Upper bound	Estimate	Lower bound	Upper bound
2000	0	0	0	0	0	0	0	0	0
2010	39	4	177	110	11	495	127	13	574
2020	176	18	790	437	44	1966	445	44	**2,000**

Source: (Coffey Geosciences 2003)

The health costs averted for each of the three scenarios were those health costs attributed to reductions in carbon monoxide, oxides of nitrogen and hydrocarbons. The reductions in PM_{10} concentrations were minimal. This is also reflected in the projected health savings (shown in Table 5.8), which is much less than savings demonstrated in other studies, for example, the BTRE (BTRE 2005) study. This is because the three scenarios have minimal effect on PM_{10} concentrations.

5.1.6 The economic consequences of the health effects of transport emissions in Australia (BTRE 2005)

This study on the health costs of transport emissions in Australian cities was conducted by the Bureau of Transport and Regional Economics in Canberra. It used epidemiological data to derive exposure-response functions and applied a refined human capital approach to derive economic costs.

Only the health effects of PM_{10} were included in this analysis and costs were calculated with a threshold (5 ug/m^3) and without a threshold for PM_{10}. Health endpoints used in this study were deaths (in those over the age of 30 years), hospital admissions for respiratory and cardiovascular disease, incidence of asthma attacks and incidence of bronchitis for the year 2000. In 2000, it was estimated that traffic related PM_{10} was responsible for 1,400 premature deaths, 2,400 hospital admissions and 21,000 days of asthma exacerbations.

The motor vehicle proportion of ambient PM_{10} ranged from 7 percent in Hobart to 43 percent in Sydney. The value of a statistical life was taken to be A$1.9 million (in 2002 dollars).

The health effects and health costs for mortality and morbidity due to traffic related PM_{10} in Australia are presented in Tables 5.9 and 5.10. The total health costs due to motor vehicle air pollution (using PM_{10} as the index pollutant) in Australia in 2000 was approximately A$2.7 billion. More than 85 percent of the cost was incurred in the capital cities. About 70 percent of the costs were attributed to mortality costs.

Table 5.9 Health events due to motor vehicle air pollution in Australia 2000

States/Territories	Mortality			Morbidity (except asthma attacks)			Asthma attacks		
	Central	Lower	Upper	Central	Lower	Upper	Central	Lower	Upper
NSW	638	394	888	1,208	415	1,981	419	221	609
VIC	393	243	574	747	262	1,220	628	321	922
QLD	182	113	253	371	129	607	215	110	348
SA	94	58	130	176	59	269	55	28	80
WA	89	54	123	183	61	301	55	29	80
TAS	13	8	18	16	6	34	1	0	1
NT	4	2	5	9	3	16	0	0	1
ACT	6	4	9	N/a*	N/a	N/a	N/a	N/a	N/a
Australia	1,420	876	1,970	2,710	934	4,449	1373	711	2,042

Note

***** Not available

Source: (BTRE 2005)

Table 5.10 Total economic costs of motor vehicle related air pollution in Australia 2000 (A$ million)

State	Mortality			Morbidity			Total		
	Central	Lower	Upper	Central	Lower	Upper	Central	Lower	Upper
NSW	829	512	1,151	363	194	530	1,192	706	1,682
VIC	512	316	711	230	123	335	741	439	1,045
QLD	236	146	328	113	60	164	349	206	492
SA	122	76	170	52	28	75	174	103	245
WA	115	71	160	53	28	77	169	100	237
TAS	17	10	23	5	3	10	22	13	33
NT	5	3	7	2	1	3	8	5	11
ACT	8	5	12	N/a*	N/a	N/a	8	5	12
Australia	1,846	1,140	2,562	817	438	1,195	2,663	1,577	3,757

Note

* Not available

Source: (BTRE 2005)

5.2 North American studies of health cost of air pollution

5.2.1 The economic value of quantifiable ozone and PM_{10} related health effects in the San Francisco Bay area (Hall, Brajer et al. 1994)

This study was aimed at documenting the economic value of avoiding health effects by attaining the Californian air quality standards for ozone and PM_{10}.

This study by Hall et al (Hall, Brajer et al. 1994) integrated human exposure modelling, health effects modelling and economic valuation methods to estimate the economic benefits of attaining the California air quality standards for ozone and PM_{10} in the San Francisco Bay Area.

Only a limited number of adverse health effects attributable to ambient ozone were addressed in this study. These health effects included: eye irritation, cough, throat irritation, chest discomfort, headache and minor restricted activity days (MRAD; days when normal activity is curtailed but not suspended). The authors state that these adverse effects were chosen because they could be reliably quantified (that is, exposure/dose, frequency of effect, value of each effect must all be able to be quantified).

The authors state that there are also a number of non-quantifiable health effects including the development of chronic respiratory diseases (for example, bronchitis, asthma, emphysema), and the decrements in lung function (as measured by spirometry).

A number of uncertainties are also acknowledged by the authors:

- The quantification is based on estimates only and hence they should be viewed as indications of the magnitude of effects and economic values and not as precise measurements
- May potential health effects cannot be quantified and therefore the benefits quantified are likely to be underestimates
- There is uncertainty regarding susceptible subgroups in the population.

The authors assumed the existence of a threshold effect at 0.08 ppm (that is, no effect below an ozone concentration of 0.08 ppm). The value of a life was placed at US$4.2 million (range: US$3.1 million to US$9.5 million).

The majority of health costs for ozone and PM_{10} were due to PM_{10} (at least 95 percent of costs), and most of the particulate cost was due to premature deaths. The health costs of ozone and PM_{10} are presented in Table 5.11.

Table 5.11 Economic value of reducing ozone and PM_{10} related health effects (annual millions in 1991US dollars)

Air Pollutant	Low	Mid	High
Ozone*			
Cough	0.10	0.30	0.86
Eye irritation	1.46	3.36	7.75
Throat irritation	0.34	0.73	1.53
Headache	0.16	0.44	1.19
Chest discomfort	0.11	0.25	0.52
Minimal restricted activity day	0.14	0.23	**0.37**
Total	2.32	5.29	12.22
PM_{10}			
Averted deaths	1,184	1,604	3,629
Restricted activity days	449	449	449
Total	1,633	2,053	4,078

Note

***** Assuming no overlap between person-days of symptoms

Source: (Hall, Brajer et al. 1994)

5.2.2 Costs and Benefits of the Clean Air Act, 1990 to 2010 (US EPA 1999)

This US EPA report to the US Congress assessed the benefits and costs of the Clean Air Act from 1990 to 2010. Amendments to the Clean Air Act were introduced in 1990 and represented an incremental improvement to the nation's clean air program. Therefore this report was designed to estimate the costs and benefits of the 1990 Amendments.

The steps taken in this analysis were:

- estimate of air pollution emissions in 1990, 2000 and 2010;
- estimate the cost of emission reductions arising from the Clean Air Act Amendments (CAAA);
- model air quality based on emissions estimate;
- quantify air quality related health and environmental effects; and,
- estimate the economic value of cleaner air; and, aggregate results and characterize uncertainty.

Six air pollutants were considered in this analysis: volatile organic compounds, oxides of nitrogen, sulphur dioxide, carbon monoxide, PM_{10} and $PM_{2.5}$. With the introduction of the Clean Air Act Amendments, by 2010, emissions for volatile organic compounds, oxides of nitrogen, sulphur dioxide, carbon monoxide, PM_{10} and $PM_{2.5}$ will have been reduced by 35 percent, 39 percent, 23 percent, 31 percent, 4 percent and 4 percent respectively. The reduction for particulates refers to emissions of primary particulates only. With the reductions in sulphur dioxide and oxides of nitrogen, which are precursors of fine particles, the expected reductions in all particulates (primary plus secondary) would be greater. The percent reductions in air pollutants due to motor vehicle emissions are shown in Table 5.12. It is important to note that motor vehicle particulate emissions are only a small component of total particulate emissions.

Table 5.12 National annual emission projections to 2010 (thousand tons)

	Source category	1990	2010 Pre-CAAA*	2010 Post-CAAA	% Change
Volatile organic compounds	Motor vehicle	6,800	7,300	3,900	-46%
	Total	23.000	28,000	18,000	-35%
Oxides of nitrogen	Motor vehicle	7,400	9,100	5,600	-39%
	Total	23,000	28,000	17,000	-39%
Carbon monoxide	Motor vehicle	62,000	66,000	42,000	-37%
	Total	94,000	107,000	82,000	-23%
Sulphur dioxide	Motor vehicle	570	770	410	-47%
	Total	22,000	26,000	18,000	-31%
Primary PM_{10}	Motor vehicle	360	300	210	-31%
	Total	28,000	29,000	28,000	-3%
Primary $PM_{2.5}$	Motor vehicle	290	230	140	-39%
	Total	7,100	7,700	7,400	-4%

Note

* CAAA=Clean Air Act Amendments

Source: (US EPA 1999)

One of the criteria used to select studies in this risk analysis was that the studies had to be conducted in the US or Canada (that is, studies conducted outside of the United States were not included). It was generally assumed that the annual benefits accrued in the same year as the costs are incurred. However, an exception was premature deaths, where it was modelled that deaths accrued over a period of five years from the time of exposure.

The range of benefits estimated incorporated both the quantified uncertainty associated with each of the health effect estimates and the quantified uncertainty associated with the corresponding economic valuation strategy. Cost of death was valued at US$4.8 million (1990 US dollars). The health effects valuations are presented in Table 5.13.

Table 5.13 Health effects unit valuation (1990 US dollars)

Endpoint		Pollutant	Mean Estimated Valuation per case
Mortality		PM_{10}	4,800,000
Chronic bronchitis		PM_{10}	260,000
Chronic asthma		O_3	25,000
Hospital admissions	All respiratory	SO_2, NO_2, PM_{10} and O_3	6,900
	All cardiovascular	CO, SO_2, NO_2, PM_{10} and O_3	9,500
Emergency room visits for asthma		PM_{10} and O_3	194
Respiratory illness and symptoms	Acute bronchitis	PM_{10}	45
	Asthma attack or moderate or worse asthma day	PM_{10} and O_3	32
	Acute respiratory symptoms	CO, SO_2, PM_{10} and O_3	18
	Upper respiratory symptoms	PM_{10}	19
	Lower respiratory symptoms	PM_{10}	12
	Shortness of breath, chest tightness or wheeze	PM_{10} and SO_2	5.30
Work loss days		PM_{10}	83
Mild restricted activity days		PM_{10} and O_3	38

Source: (US EPA 1999)

The monetized values of the health benefits are presented in Table 5.14. Note that the total benefits include some welfare benefits (about US$4,000 million; for example, improved visibility, reduced acidification). Morbidity benefits accounted for the other 60 percent of all non-mortality benefits.

The majority of the benefits were attributable to changes in particulate matter concentrations, and more specifically, to the effect of these ambient particulate changes on avoidance of premature mortality (about 90 percent of the benefits) and chronic bronchitis (the major morbidity benefit).

Importantly, motor vehicles contributed to 9 percent and 30 percent of the reductions in PM_{10} and $PM_{2.5}$ levels respectively. Therefore the benefits of motor vehicle related particulates could be about US$40,000 million. These particulate benefits are probably an underestimate as this report only considers the benefits of the reduction in primary particulates. With the reductions in oxides of nitrogen and sulphur dioxide, it would be expected that secondary particulate levels would also be reduced with the associated benefits.

Table 5.14 Criteria pollutant health benefits in 2010 (in millions of 1990 US dollars)

	Cost estimates		
	5th percenttile	Mean	95th percenttile
Mortality			
Ages 30+ years)	14,000	100,000	250,000
Chronic illness			
Chronic bronchitis	360	5,600	18,000
Chronic asthma	40	180	300
Hospitalisation			
All respiratory	76	130	200
Total cardiovascular	93	390	960
Asthma-related ED visits	0.1	1.0	2.8
Minor illness			
Acute bronchitis	0.0	2.1	5.2
Upper respiratory symptoms	4.2	19	39
Lower respiratory symptoms	2.2	6.2	12
Respiratory illness	0.9	6.3	15
Moderate-severe* asthma	1.9	13	29
Asthma attacks*	20	55	100
Chest tightness, shortness of breath or wheeze	0.0	0.6	3.1
Shortness of breath	0.0	0.5	1.2
Work loss days	300	340	380
Minimal restricted activity day/any of 19 respiratory effects	680	1,200	1,800
Total Benefits in 2010**	-	110,000	-

Notes

* Moderate-severe asthma and asthma attacks are included in the definition of minimal restricted activity day/any of 19 respiratory effects and should not be included in any total benefits to avoid the potential for double counting

** Summing the 5th and 95th percentile values would yield a misleading estimate of the 5th and 95th percentile estimate of total health benefits. Therefore only the mean value is presented

Source: (US EPA 1999)

5.2.3 The health costs of motor vehicle related air pollution (McCubbin and Delucchi 1999)

This study estimates the costs of all the health effects of all air pollutants from all emissions sources related to motor-vehicle use. The authors consider four sources of emissions from motor vehicle use: tailpipe and evaporative emissions from motor vehicles; emissions from upstream fuel and vehicle production processes; particulate dust emissions from paved roads; and, particulate dust emissions from unpaved roads.

Health costs of four criteria air pollutants (carbon monoxide, ozone, nitrogen dioxide and particulates) and six air toxics were considered (acetaldehyde, benzene, 1,3-butadiene, formaldehyde, gasoline particulates and diesel particulates).

Health effects considered in this analysis were premature deaths, cancer, acute morbidity (headache, sore throat, excess phlegm, eye irritation, non-asthma respiratory illness, asthma attack, respiratory restricted activity day) and chronic morbidity (asthma, chronic bronchitis, emphysema). A statistical life was valued at 1991 US$1 to US$4 million.

The costs of all anthropogenic air pollution was estimated to be between 1991US$55-672 billion and the cost of anthropogenic PM_{10} pollution was estimated to be between 1991US$52-652 billion. The costs of anthropogenic carbon monoxide, nitrogen dioxide and ozone were between 1991US$1-8 billion, 1991US$1-6 billion and 1991US$0.3-US$6 billion respectively. Of the total cost of anthropogenic PM_{10} pollution (between 1991US$52-652 billion), the cost of mortality predominated (between 1991US$40-470 billion). Table 5.15 presents the health costs of a 100 percent reduction in motor vehicle related emissions. Not surprisingly, the cost for urban areas is about 95 percent of the total costs.

Table 5.15 Cost of motor vehicle air pollution based on a 100 percent reduction in motor vehicle related emissions, (1991 US$ million)

Air pollutant	Motor vehicles		Motor vehicles+upstream		Motor vehicles+upstream+paved and unpaved road dust	
	Low	High	Low	High	Low	High
Nation						
PM_{10}	16,727	266,391	18,961	279,354	21,943	432,829
Ozone	214	1,899	228	1,945	228	1,945
Nitrogen dioxide	1,038	5,483	1,048	5,509	1,048	5,509
Carbon monoxide	919	8,085	921	8,092	921	8,092
Air toxics	87	1,622	87	1,623	87	**1,623**
Total	18,985	283,481	21,246	296,522	24,227	449,997
Urban						
PM_{10}	15,954	253,126	18,059	265,228	20,599	401,037
Ozone	196	1,730	209	1,771	209	1,771
Nitrogen dioxide	955	5,072	964	5,093	964	5,093
Carbon monoxide	829	7,089	831	7,094	831	7,094
Air toxics	76	1,411	76	1,412	76	1,412
Total	18,010	268,428	20,139	280,599	22,679	416,408

Source: (McCubbin and Delucchi 1999)

The costs per mile travelled for gasoline and diesel vehicles are given in Table 5.16. There are higher costs for diesel for the effects of PM, nitrogen dioxide and air toxics. The air pollution cost of using diesel is nearly eight times greater than the air pollution cost of using petrol.

The breakdown of these costs for particulate matter by vehicle type and emissions sources is shown in Table 5.17. For gasoline light and heavy duty vehicles, the cost per mile travelled was between 0.55 cents and 8.04 cents, whereas for diesel vehicles it was 3.48 cents to 64.86 cents.

Table 5.16 Costs per mile of motor vehicle travel based on a 10 percent reduction in motor vehicle emissions by type of fuel (cents per mile travelled in the USA in 1990)

Vehicle type	PM		Ozone		Carbon monoxide		Nitrogen dioxide		Air toxics		Total	
	Low	High	Low	High	Low	High	Low	High	Low	High	Low	High
Gasoline												
Motor vehicle emissions	0.55	8.04	0.01	0.08	0.05	0.42	0.05	0.25	0.0	0.06	0.65	8.83
Motor vehicle emissions+upstream	0.64	8.61	0.01	0.08	0.05	0.42	0.05	0.25	0.0	0.06	0.75	9.40
Diesel												
Motor vehicle emissions	3.48	64.86	0.01	0.15	0.00	0.05	0.12	0.78	0.01	0.27	3.62	65.85
Motor vehicle emissions+upstream	3.68	66.03	0.02	0.16	0.01	0.05	0.12	0.78	0.01	0.27	3.83	67.03
All												
Motor vehicle emissions	0.78	12.57	0.01	0.09	0.04	0.39	0.05	0.29	0.0	0.08	0.89	13.37
Motor vehicle emissions+upstream	0.89	13.17	0.01	0.09	0.04	0.39	0.05	0.29	0.0	0.08	1.00	**13.98**

Source: (McCubbin and Delucchi 1999)

Table 5.17 Costs of PM per mile of motor vehicle travel based on a 10 percent reduction in motor vehicle emissions by vehicle type (cents per mile travelled in the USA in 1990)

Vehicle type	**Motor vehicles**		Motor vehicles +upstream		Motor vehicles +upstream +paved road dust		Motor vehicles +upstream +paved road dust +unpaved road dust	
	Low	High	Low	High	Low	High	Low	High
LDGV*	0.48	7.02	0.56	7.50	0.60	10.90	0.65	12.18
LDGT	0.74	10.70	0.90	11.54	0.94	16.09	1.02	17.80
HDGV	1.56	30.28	1.78	31.53	1.90	42.55	2.07	46.69
Gasoline	0.55	8.04	0.64	8.61	0.68	12.33	0.74	13.74
LDDV**	1.47	18.49	1.50	18.70	1.53	21.34	1.57	22.34
LDDT	0.47	5.77	0.52	6.14	0.57	10.09	0.63	11.58
HDDV	4.18	79.93	4.43	81.37	4.75	110.91	5.21	122.01
Diesel	3.48	64.86	3.68	66.03	3.93	89.55	4.30	98.39
Total	0.78	12.57	0.89	13.17	0.94	18.47	1.02	20.46

Note

***** LDGV=light duty gasoline vehicle; LDGT= light duty gasoline truck; HDGV= heavy duty gasoline vehicle; **LDDV=light duty diesel vehicle; LDDT= light duty diesel truck; HDDV= heavy duty diesel vehicle

Source: (McCubbin and Delucchi 1999)

5.2.4 Illness costs of air pollution (DSS 2000)

This project was commissioned by the Ontario Medical Association, Canada, to document the data and methodology underlying the forecasts of health and economic damages attributable to air pollution (ozone and PM_{10}).

Health impacts assessed in this report were: premature deaths, hospital admission, emergency department visits, doctor's office visits and minor illnesses.

Health costs are provided for two time periods (2000 and 2015) and are presented for two scenarios:

1. Background ozone and PM_{10} values of 24 ppb and 0 ug/mg respectively
2. Status quo, that is, current air pollution levels for Ontario.

It is assumed that for the second scenario, the air pollution levels have remained constant, and the increased adverse health events are due to an increase in the population as well as to the aging of the population.

All premature deaths and most hospitalizations and minor illnesses are attributable to PM_{10}. Most of the emergency department visits are attributable to ozone. The value of a statistical life ranged between CAN$1.9 million to CAN$2.5 million. The health costs from this study are given in Table 5.18.

In 2000 in Ontario, with current air pollution levels, the total economic damages were estimated to be about CAN$10 billion. The great majority of these damages are attributable to PM_{10}. The annual damages for 2015 is in the order of CAN$12 billion. There is a significant increase due to premature mortality and this is due to the fact of an aging population and the increased vulnerability of the elderly to the effects of air pollution.

Table 5.18 Comparative economic damages due to air quality (in millions of Canadian dollars)

Scenario	Health care cost		Lost productivity		Increased pain and suffering		Loss of life		Total	
	2000	2015	2000	2015	2000	2015	2000	2015	2000	2015
Background	26.2	35.9	4.0	5.4	3.5	4.5	-	-	33.6	45.8
Status quo	601.5	696.3	560.1	626.3	4,758.2	5,367.5	4,058.4	5,365.7	9,979.0	12,055.9

Source: (DSS 2000)

5.3 European studies of health costs of air pollution

5.3.1 Health costs due to road traffic related air pollution (Seethaler 1999)

This was a trilateral project carried out by Austria, France and Switzerland. The aim was to estimate the health costs of traffic related air pollution by integrating data on air pollution, epidemiology and economy.

Air pollution exposure-response relationships were determined and number of excess health events attributable to road traffic was calculated. This report only assessed the effects of PM_{10} and assumed a threshold of 7.5 ug/m^3 (that is, there are no adverse health effects below the threshold level).

The weighted annual average PM_{10} concentrations for Austria, France and Switzerland were 26.0 ug/m^3, 23.5 ug/m^3 and 21.4 ug/m^3 respectively, and the road traffic related PM_{10} concentrations for the three countries were 8.0 ug/m^3, 8.9 ug/m^3 and 7.4 ug/m^3 respectively.

Health effects considered in this economic evaluation were premature deaths (in those 30+ years), hospital admissions for respiratory and cardiovascular diseases, incidence of chronic bronchitis in adults and bronchitis in children, asthma attacks and restricted activity days. A prevented death was valued at 0.9 million EUR.

The study reports that the sensitivity of the results presented in Table 5.19 is influenced by the assessment of PM_{10} exposure, the air pollution attributable health outcomes and the monetary valuation of the health effects. In general, for each sensitive assumption, an "at least" approach was adopted, that is, conservative values were chosen for each of the assumptions. Further, the results were also thought to underestimate the true costs since other health effects (for example, lung cancer, infant mortality) were not considered, additional effects of other air pollutants (for example, ozone) were not considered, and for the monetary valuation generally prudent cost factors were chosen.

Mortality costs were dominant in all three countries (about 70 percent of total costs). Within morbidity costs, chronic bronchitis (due to a high monetary value) and restricted activity days (due to the large number of restricted activity days) were the major contributors.

Table 5.19 Air pollution related health costs in Austria, France and Switzerland in 1996

	Austria		France		Switzerland	
	Costs attributable to total air pollution	Costs attributable to road traffic	Costs attributable to total air pollution	Costs attributable to road traffic	Costs attributable to total air pollution	Costs attributable to road traffic
Mortality	5,019	2,170	28,523	15,866	2,983	1,586
Morbidity	1,669	722	10,335	5,749	1,188	630
Total	6,687	2,892	38,848	21,615	4,170	2,216

Source: (Seethaler 1999)

5.4 Other studies of health costs of air pollution

5.4.1 Particulate matter in urban areas: health-based economic assessment (El-Fadel and Massoud 2000)

This study assessed the health impacts of PM_{10} air pollution in the urban areas of Lebanon and also estimated the economic benefits of a 10 ug/m^3 reduction in PM_{10} levels of ambient air pollution. The urban population of Lebanon was estimated at 1.64 million.

The authors assumed there was no threshold for PM_{10}, the whole population was equally sensitive to the effects of PM_{10} pollution and if age-specific dose response relationships were not available then the same dose response relationship was applied to each age-group.

Health endpoints considered were mortality, emergency department visits, and hospital admissions for pneumonia and chronic obstructive pulmonary disease. The value of a premature death was assumed to be between US$0.06 to US$1.35 million (willingness to pay approach).

Table 5.20 shows the annual number of health events avoided and the associated averted health costs for a 10 ug/m^3 reduction in PM_{10} levels. The majority of the costs are due to the cost of premature deaths. The willingness to pay approach estimates are an order of magnitude greater than the estimates derived from the cost of illness approach. This is because the cost of illness approach does not take into account inconvenience, suffering, losses in leisure and other intangibles.

Table 5.20 Economic benefit due to a 10 ug/m^3 reduction in PM_{10} levels

Health endpoint	Number of cases avoided	Total economic benefit (US$ million)	
		Cost of illness approach	Willingness to pay approach
Mortality	11-617	0.27-12.6*	3.5-157.9
COPD admissions	31-441	0.06-0.9	0.98-13.9
Pneumonia admissions	13-189	0.03-0.4	0.05-0.7
Emergency visits	609-25,578	0.05-1.9	Not available
Total		0.41-15.8	4.53-172.5

Note

* Human capital approach

Source: (El-Fadel and Massoud 2000)

5.4.2 Improving air quality in metropolitan Mexico City. An economic evaluation (Vergara 2002)

This study aimed to determine the health benefits of reducing ozone and PM_{10} concentrations in metropolitan Mexico City.

Although four air pollution reduction scenarios are presented in the report, only the health benefits of two scenarios are presented here. These are:

- 10 percent reduction in exposure (6.41 ug/m3 for PM10 and 0.0114 ppm reduction in ozone per person)
- Meeting air quality standards (14.06 ug/m3 for PM10 and 0.0702 ppm reduction in ozone per person)

The health impacts considered in this report are premature mortality, hospital admissions for respiratory and cardiovascular diseases, emergency department visits for respiratory disease, asthma symptoms, chronic bronchitis and restricted activity days. A number of methods were used to assess the economic costs of mortality and morbidity – cost of illness approach, willingness to pay approach and loss productivity approach. A combination of the above methods was used to derive the central estimate and the low and high estimates.

The averted annual health costs are presented in Table 5.21. It is salient to note that the health benefits of PM_{10} reduction are an order of magnitude higher than those of ozone.

Table 5.21 Benefits from reducing ozone and PM_{10} (US$ million 1999 dollars)

	Central estimate	Range
Ozone		
• **10% reduction**	75	**18-116**
• **Meet air quality standards**	465	**109-717**
PM_{10}		
• **10% reduction**	644	**96-1,451**
• **Meet air quality standards**	1,414	**210-3,186**

Source: (Vergara 2002)

5.4.3 Quantifying the human health benefits of curbing air pollution in Shanghai (Li, Guttikunda et al. 2004)

This study investigated the cost-benefits of PM_{10} reduction in the city of Shanghai due to the introduction of emission controls in two energy consumption sectors – in the power generation sector and in the industrial coal use sector. The base year (1995) was compared to 2020. It was hypothesized that in the power generation sector, all new power generating stations coming online from 2010 would adopt state of the art integrated gasification combined cycle technology for coal combustion, and that all industrial coal burning would be banned by 2020. The above two scenarios were compared to the business-as-usual scenario.

The health endpoints selected in this study were mortality, hospital admissions, hospital outpatient and emergency department visits and chronic bronchitis.

The value of an avoidable death was taken to be US$445,000. This figure is based on the American cost of US$4.2 million per avoidable premature death and adjusted by the purchasing power parity of USA and China. The number of health events avoided and the health costs are presented in Tables 5.22 and 5.23.

Table 5.22 Avoided health events under the two emission control scenarios (US$ millions)

Health Endpoint	Power Generation Control Scenario	Industrial Control Scenario
Mortality	2,808	1,790
Hospital admission	43,482	27,716
Hospital outpatient visit	96,293	61,379
Emergency room visit	48,506	30,918
Chronic bronchitis	1,753	1,117

Source: (Li, Guttikunda et al. 2004)

Table 5.23 Annual health benefits of emission control scenarios (US$ million per year in 1998 dollars)

Health Endpoint		Power Control Scenario	Industrial Control Scenario
Mortality	Low	139	88
	Central	347	221
	High	1,030	656
Mortality	Low	38	24
	Central	57	36
	High	119	76
Work day losses		13	8
Total health benefits (Central)		190-1,162 (417)	121-741 (266)

Source: (Li, Guttikunda et al. 2004)

In both the emission control scenarios, the health benefits are likely to be underestimates of the true health benefits as only one air pollutant and a limited number of health outcomes are considered. Nevertheless, the health costs averted by reducing PM_{10} pollution are considerable. In both scenarios, the majority of the averted costs are attributable to avoided premature mortality. Although this study does not directly address the health costs of traffic related particulate pollution, it does emphasis the point that reducing particulate pollution will save on health costs.

5.5 Summary of studies reporting the health costs of motor vehicle related air toxics

The following table (Table 5.24) summarises the health costs of motor vehicle related air toxics. It is important to note that it is difficult to directly compare the health costs across the various studies as the air toxic concentrations are likely to be different, the mix of air toxics may be different, the population at risk will vary and the monetary estimates are often expressed differently.

There are only a few reported studies that have investigated the health costs of ambient air toxics. Although air toxics have carcinogenic and other chronic health effects, their ambient concentrations are generally very low and the associated health costs of air toxics are also very low compared to the other air pollutants. For example, McCubbin (McCubbin and Delucchi 1999) estimated that the health costs of air toxics were only about 4-5 percent of the total health costs of all motor vehicle related air pollution.

Table 5.24 Summary of studies reporting the health costs of motor vehicle related air toxics

Study	Health Outcome	Central Estimate	Low Estimate	High Estimate	Notes
Australian Studies					
(RCG/Hagler Bailly 1994)	Cancer		1992 A$26.0 million	1992 A$45.2 million	Cost for Melbourne only
(NRTC 1995)	Cancer	1990 A$32 million			Costs for all Australia
Other studies					
(McCubbin and Delucchi 1999)	Cancer		1991 US$87 million	1991 US$1,622mmillion	Costs for all USA

5.6 Summary of studies reporting the health costs of motor vehicle related particulates

Table 5.25 summarises the health costs of motor vehicle related particulates. As with air toxics, it is important to note that it is difficult to directly compare the health costs across the various studies as the air toxic concentrations are likely to be different, the mix of air toxics may be different, the population at risk will vary and the monetary estimates are often expressed differently.

Few studies that have reported separately the health costs of particulates and even fewer studies have reported separately the health costs of motor vehicle related particulates. The health costs of particulates far surpass the health costs of the other criteria air pollutants (ozone, nitrogen dioxide, sulphur dioxide and carbon monoxide). McCubbin (McCubbin and Delucchi 1999) estimates between 88 percent to 94 percent of motor vehicle air pollution costs can be attributable to PM_{10}. Further, the majority of the health costs attributed to particulates are due to effects of particulates on premature mortality (between about 70 percent to 95 percent).

Table 5.25 Summary of studies reporting the health costs of motor vehicle related particulates

Study	Health Outcome	Central Estimate	Low Estimate	High Estimate	Notes
Australian Studies					
(Simpson and London 1995)	Mortality		1994 A$230 million	1994 A$415 million	Cost estimates are for Brisbane only. Both industrial and motor vehicle related PM_{10}.
	Minimal restricted activity days		1994 A$8.5 million	1994 A$15.4 million	
	Restricted activity days		1994 A$16.3 million	1994 A$29.4 million	
(BTRE 2005)	Mortality	2000 A$1,846 million	2000 A$1,140 million	2000 A$2,562 million	Cost estimates are for all Australia. Cost for hospitalizations and asthma attacks are presented as a combined figure.
	Hospitalizations and asthma attacks	2000 A$817 million	2000 A$438 million	2000 A$1,195 million	
Other studies					
(Hall, Brajer et al. 1994)	Mortality	1991 US$1,184 million	1991 US$1,164 million	1991 US$3,629 million	Costs for San Francisco Bay are only. All anthropogenic PM_{10}.
	Restricted activity days	1991 US$449 million	1991 US$449 million	1991 US$449 million	
(US EPA 1999)	Mortality	1990 US$100,000 million	1990 US$14,000 million	1990 US$250,000 million	Costs for all USA. Includes all sources of air pollutants (including motor vehicles). Mortality effects due to PM_{10} only. Morbidity effects due to all criteria air pollutants.
	Morbidity	1990 US$10,000 million	N/A	N/A	
(McCubbin and Delucchi 1999)	Mortality and morbidity		1991 US$16,727 million	1991 US$266,391 million	Costs for all USA and urban USA. PM_{10} from motor vehicles.
	All USA				
	Urban USA		1991 US$15,954 million	1991 US$253,126 million	
(Seethaler 1999)	Mortality	1996 Euro19,622			Costs for Austria, France and Switzerland. PM_{10} from motor vehicles.
	Morbidity	1996 Euro7,101			

5.7 Summary of health costs of air pollution

- A major proportion of the health cost is due to fine particles.
- A large component of the health costs are attributable to the cost of premature deaths.
- Reducing the ambient concentrations of fine particles will help avert the significant health costs attributable to fine particles.
- The air pollution cost of diesel fuel is nearly eight times greater than the air pollution cost of petrol fuel.
- Use of alternative fuels to diesel is one strategy to reduce the ambient concentrations of fine particles and the associated health costs.
- Ozone and air toxics do not contribute greatly to the overall health costs of air pollution.

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06.

Projected health costs of different fuel usage distribution scenarios

This section estimates the health costs of different fuel usage distribution scenarios for the eight Australian capital cities (Sydney, Melbourne, Brisbane, Adelaide, Perth, Hobart, Darwin and Canberra). In this section the focus is on the switch to alternate fuels (CNG and LPG) over time with the associated changes in emission patterns.

Fuel usage pattern for 2000 is taken to be the base scenario. Projections of likely fuel usage patterns for 2012 and 2020 have been made. For each projected fuel usage scenario, low, central and high LPG/CNG usage distributions are considered.

Then using information on total VKTs in the eight cities in 2000, the proportion of VKT by the different classes of vehicles (passenger vehicle, light commercial vehicle, rigid truck, bus and articulated truck) and by fuel types (petrol, diesel, CNG and LPG), emissions factors by the different classes of vehicles and by fuel types, and health costs associated per tonne of air pollutants (PM_{10}, hydrocarbons, oxides of nitrogen, carbon monoxide), the health costs for the three fuel usage distributions have been calculated.

The contribution of motorcycle VKTs and emissions have not been considered in these scenarios as they constitute only a very small proportion of total VKT.

6.1 Fuel usage distribution scenarios

Three scenarios were developed:

- Base case for 2000
- Fuel usage distribution for 2012
- Fuel usage distribution for 2020

The base case scenario was extracted from Coffey (Coffey Geosciences 2003) and projected estimates for 2012 and 2020 were developed by Air Quality Technologies Pty Ltd (Peter Anyon, 2005; personal communication). For each projected scenario (2012 and 2020), low, central and high CNG and LPG usage estimates are presented. These are presented in Tables 6.1, 6.2 and 6.3.

In 2012, it is estimated that diesel use would increase substantially for light commercial vehicles and moderate increase for passenger cars. There would be moderate increases in the use of LPG/CNG fuels for passenger cars, rigid trucks and buses. There would be minimal increases in the use of LPG/CNG for articulated trucks and motorcycles.

In 2020, more passenger cars would be using diesel and LPG/CNG compared to 2012. The trend to increasing LPG/CNG use for light commercial vehicles (at the expense of both diesel and petrol fuels), trucks and buses will continue compared to 2012.

Table 6.1 Projected fuel usage distribution scenario for 2000 (base case)

Vehicle Category	**VKT Distribution by Fuel Type** (%)			
	Petrol	Diesel	CNG	LPG
Passenger car and 4 wheel drive	93.5	2.5	0.0	4.0
Light commercial vehicle	72.7	23.0	0.0	4.3
Rigid truck	0.0	100.0	0.0	0.0
Bus	0.0	99.9	1.0	0.0
Articulated truck	0.0	100.0	0.0	0.0

Source: (Coffey Geosciences 2003)

Table 6.2 Projected fuel usage distribution scenario for 2012

Vehicle Category	**VKT Distribution by Fuel Type (%)**											
	Low Scenario				Central Scenario				High Scenario			
	Petrol	Diesel	CNG	LPG	Petrol	Diesel	CNG	LPG	Petrol	Diesel	CNG	LPG
Passenger car & 4 wheel drive	88.0	6.0	0.0	6.0	86.0	5.0	0.0	9.0	84.0	4.0	0.0	12.0
Light commercial vehicle	48.0	45.0	0.0	7.0	49.0	40.0	0.0	11.0	50.0	35.0	0.0	15.0
Rigid truck	0.0	95.0	3.0	2.0	0.0	92.0	5.0	3.0	0.0	89.0	6.0	5.0
Bus	0.0	94.0	5.0	1.0	0.0	90.0	7.0	3.0	0.0	84.0	10.0	6.0
Articulated truck	0.0	98.5	1.5	0.0	0.0	98.0	2.0	0.0	0.0	97.0	3.0	0.0

Source: (Peter Anyon, Air Quality Technologies Pty Ltd, 2005; personal communication)

Table 6.3 Projected fuel usage distribution scenario for 2020

Vehicle Category	**VKT Distribution by Fuel Type (%)**											
	Low Scenario				Central Scenario				High Scenario			
	Petrol	Diesel	CNG	LPG	Petrol	Diesel	CNG	LPG	Petrol	Diesel	CNG	**LPG**
Passenger car & 4 wheel drive	65.0	25.0	2.0	8.0	65.0	18.0	2.0	15.0	65.0	10.0	4.0	21.0
Light commercial vehicle	39.0	45.0	6.0	10.0	39.0	37.0	8.0	16.0	35.0	30.0	13.0	22.0
Rigid truck	0.0	90.0	6.0	4.0	0.0	82.0	10.0	8.0	0.0	73.0	15.0	12.0
Bus	0.0	87.0	8.0	5.0	0.0	79.5	12.0	8.5	0.0	70.0	18.0	12.0
Articulated truck	0.0	97.0	3.0	0.0	0.0	93.5	6.5	0.0	0.0	90.0	10.0	0.0

Source: (Peter Anyon, Air Quality Technologies Pty Ltd, 2005; personal communication)

6.2 Vehicle kilometres travelled

VKTs by the different types of vehicles for the base year (2000) were obtained from the Coffey report (Coffey Geosciences 2003) (Table 6.4). These data are for the eight capital cities (Sydney, Melbourne, Brisbane, Adelaide, Perth, Hobart, Darwin and Canberra). VKT projections for 2012 and 2020 were obtained from Air Quality Technologies Pty Ltd (Peter Anyon, 2005; personal communication) (Table 6.4).

Table 6.4 Vehicle kilometres travelled by type of vehicle, 2000, 2012 and 2020

Vehicle Category	Vehicle Kilometres Travelled (millions)
2000	
Passenger car and 4 wheel drive	91,240
Light commercial	15,880
Rigid truck	3,730
Bus	790
Articulated truck	1,020
Total	112,660
2012	
Passenger car and 4 wheel drive	110,758
Light commercial	26,110
Rigid truck	4,306
Bus	934
Articulated truck	1,746
Total	143,854
2020	
Passenger car and 4 wheel drive	123,770
Light commercial	32,930
Rigid truck	4,702
Bus	1,030
Articulated truck	2,230
Total	164,662

Source: (Coffey Geosciences 2003)

6.3 Total emissions of the four air pollutants

Total emissions for PM_{10}, oxides of nitrogen, hydrocarbons and carbon monoxide for the eight capital cities were provided by Air Quality Technologies Pty Ltd (Peter Anyon, 2005; personal communication) (Tables 6.5 to 6.12). Emission factors (grams per kilometre travelled; g/km) were derived from the 2003 Coffey report (Coffey Geosciences 2003).

The calculation of total emissions took into account vehicle categories, age profiles of each vehicle category, travel by vehicle age, year of manufacture, scrappage and new vehicle introductions. Emission factors were varied to take into account vehicle age, technological changes, ADR regulations and in-service deterioration.

Table 6.5 Total PM_{10} emission (tonnes/year) by vehicle and fuel type, 2000 and 2012

Vehicle Category	**Petrol**				**Diesel**			
	2000	**2012**			**2000**	**2012**		
	Base	**Low**	**Central**	**High**	**Base**	**Low**	**Central**	**High**
Passenger Car & 4 wheel drive	2,515	988	966	943	525	698	582	465
Light commercial vehicle	338	117	117	122	841	1,417	1,260	1,102
Rigid truck	0	0	0	0	2,278	1,176	1,139	1,102
Bus	0	0	0	0	516	211	202	189
Articulated truck	0	0	0	0	683	423	421	416
	CNG				**LPG**			
Passenger Car & 4 wheel drive	0	0	0	0	43	27	40	54
Light commercial vehicle	0	0	0	0	8	7	11	15
Rigid truck	0	8	13	16	0	5	8	13
Bus	0	2	3	4	0	0	1	2
Articulated truck	0	3	4	6	0	0	0	0

Source: (Peter Anyon, Air Quality Technologies Pty Ltd, 2005; personal communication)

Table 6.6 Total PM_{10} emission (tonnes/year) by vehicle and fuel type, 2000 and 2020

Vehicle Category	Petrol				Diesel			
	2000	2020			2000	2020		
	Base	Low	Central	High	Base	Low	Central	High
Passenger car & 4 wheel drive	2,515	116	116	116	525	927	668	371
Light commercial vehicle	338	15	15	13	841	606	499	404
Rigid truck	0	0	0	0	2,278	318	290	258
Bus	0	0	0	0	516	38	35	30
Articulated truck	0	0	0	0	683	121	116	112
	CNG				LPG			
Passenger car & 4 wheel drive	0	0	0	0	43	6	11	15
Light commercial vehicle	0	1	1	2	8	2	2	3
Rigid truck	0	13	22	33	0	9	18	27
Bus	0	2	4	5	0	1	3	4
Articulated truck	0	5	11	17	0	0	0	0

Source: (Peter Anyon, Air Quality Technologies Pty Ltd, 2005; personal communication)

Table 6.7 Total oxides of nitrogen emission (tonnes/year) by vehicle and fuel type, 2000 and 2012

Vehicle Category	Petrol				Diesel			
	2000	2020			2000	2020		
	Base	Low	Central	High	Base	Low	Central	High
Passenger car & 4 wheel drive	85,214	38,384	37,512	36,639	1,689	3,505	2,920	2,336
Light commercial vehicle	13,024	5,460	5,460	5,687	3,290	8,634	7,675	6,715
Rigid truck	0	0	0	0	26,482	19,812	19,187	18,561
Bus	0	0	0	0	9,685	5,120	4,903	4,576
Articulated truck	0	0	0	0	12,862	14,466	14,393	14,246
	CNG				LPG			
Passenger car & 4 wheel drive	0	0	0	0	2,442	1,753	2,630	3,507
Light commercial vehicle	0	0	0	0	516	533	838	1,143
Rigid truck	0	326	543	651	0	217	326	543
Bus	12	82	115	165	0	16	49	99
Articulated truck	0	0	0	0	0	0	0	0

Source: (Peter Anyon, Air Quality Technologies Pty Ltd, 2005; personal communication)

Table 6.8 Total oxides of nitrogen emission (tonnes/year) by vehicle and fuel type, 2000 and 2020

Vehicle Category	Petrol				Diesel			
	2000	2020			2000	2020		
	Base	Low	Central	High	Base	Low	Central	High
	85,214	11,364	11,364	11,364	1,689	7,713	5,554	3,085
	13,024	2,009	2,009	1,803	3,290	4,807	3,953	3,205
	0	0	0	0	26,482	10,032	9,141	8,137
	0	0	0	0	9,685	1,717	1,569	1,382
	0	0	0	0	12,862	7,301	7,038	6,774
	CNG				LPG			
Passenger car & 4 wheel drive	0	0	0	0	2,442	937	1,757	2,460
Light commercial vehicle	0	207	276	449	516	345	552	759
Rigid truck	0	556	927	1,391	0	371	742	1,113
Bus	12	98	147	220	0	61	104	147
Articulated truck	0	0	0	0	0	0	0	0

Source: (Peter Anyon, Air Quality Technologies Pty Ltd, 2005; personal communication)

Table 6.9 Total hydrocarbon emission (tonnes/year) by vehicle and fuel type, 2000 and 2012

Vehicle Category	Petrol				Diesel			
	2000	2020			2000	2020		
	Base	Low	Central	High	Base	Low	Central	High
Passenger car & 4 wheel drive	51,481	27,641	27,013	26,385	1,061	1,182	985	788
Light commercial vehicle	6,650	3,971	3,971	4,136	1,810	2,383	2,118	1,853
Rigid truck	0	0	0	0	12,737	6,307	6,108	5,909
Bus	0	0	0	0	2,003	858	821	766
Articulated truck	0	0	0	0	1,372	1,480	1,472	1,457
	CNG				LPG			
Passenger car & 4 wheel drive	0	0	0	0	2,643	2,262	3,392	4,523
Light commercial vehicle	0	0	0	0	472	695	1,092	1,489
Rigid truck	0	276	460	552	0	184	276	460
Bus	11	70	98	139	0	14	42	84
Articulated truck	0	0	0	0	0	0	0	0

Source: (Peter Anyon, Air Quality Technologies Pty Ltd, 2005; personal communication)

Table 6.10 Total hydrocarbon emission (tonnes/year) by vehicle and fuel type, 2000 and 2020

Vehicle Category	Petrol				Diesel			
	2000	2020			2000	2020		
	Base	Low	Central	High	Base	Low	Central	High
Passenger car & 4 wheel drive	51,481	12,799	12,799	12,799	1,061	1,427	1,027	571
Light commercial vehicle	6,650	2,403	2,403	2,157	1,810	847	697	565
Rigid truck	0	0	0	0	12,737	1,846	1,682	1,497
Bus	0	0	0	0	2,003	218	199	175
Articulated truck	0	0	0	0	1,372	950	916	882
	CNG				LPG			
Passenger car & 4 wheel drive	0	0	0	0	2,643	1,890	3,544	4,962
Light commercial vehicle	0	444	592	961	472	740	1,183	1,627
Rigid truck	0	472	786	1,179	0	314	629	943
Bus	11	83	124	186	0	52	88	124
Articulated truck	0	0	0	0	0	0	0	0

Source: (Peter Anyon, Air Quality Technologies Pty Ltd, 2005; personal communication)

Table 6.11 Total carbon monoxide emission (tonnes/year) by vehicle and fuel type, 2000 and 2012

Vehicle Category	Petrol				Diesel			
	2000	2020			2000	2020		
	Base	Low	Central	High	Base	Low	Central	High
Passenger car & 4 wheel drive	629,191	198,799	194,281	189,763	3,811	4,280	3,567	2,853
Light commercial vehicle	81,449	20,770	20,770	21,636	5,666	8,380	7,449	6,518
Rigid truck	0	0	0	0	18,153	9,811	9,501	9,191
Bus	0	0	0	0	4,733	2,021	1,935	1,806
Articulated truck	0	0	0	0	6,426	5,098	5,072	5,020
	CNG				LPG			
Passenger car & 4 wheel drive	0	0	0	0	30,955	15,588	23,381	31,175
Light commercial vehicle	0	0	0	0	5,540	3,483	5,474	7,464
Rigid truck	0	4,176	6,960	8,352	0	2,784	4,176	6,960
Bus	160	1,059	1,482	2,117	0	212	635	1,270
Articulated truck	0	0	0	0	0	0	0	0

Source: (Peter Anyon, Air Quality Technologies Pty Ltd, 2005; personal communication)

Table 6.12 Total carbon monoxide emission (tonnes/year) by vehicle and fuel type, 2000 and 2020

Vehicle Category	Petrol				Diesel			
	2000	2020			2000	2020		
	Base	Low	Central	High	Base	Low	Central	High
Passenger car & 4 wheel drive	629,191	68,360	68,360	68,360	3,811	4,953	3,566	1,981
Light commercial vehicle	81,449	7,610	7,610	6,830	5,666	3,682	3,027	2,455
Rigid truck	0	0	0	0	18,153	3,519	3,206	2,854
Bus	0	0	0	0	4,733	515	471	415
Articulated truck	0	0	0	0	6,426	2,495	2,405	2,315
	CNG				LPG			
Passenger car & 4 wheel drive	0	0	0	0	30,955	9,676	18,142	25,399
Light commercial vehicle	0	1,346	1,795	2,917	5,540	2,244	3,590	4,937
Rigid truck	0	7,133	11,889	17,833	0	4,755	9,511	14,266
Bus	160	1,259	1,888	2,832	0	787	1,338	1,888
Articulated truck	0	0	0	0	0	0	0	0

Source: (Peter Anyon, Air Quality Technologies Pty Ltd, 2005; personal communication)

6.4 Health costs for pollutants

The health cost data were obtained from the report by Coffey (Coffey Geosciences 2003). The health costs per tonne of air pollutant are shown in Table 6.13. The health costs are in 2003 Australian dollars.

Table 6.13 Health costs per tonne of air pollutant (A$2003)

Air pollutant	Cost per tonne (A$2003)
Carbon monoxide	12.90
NOx (as nitrogen dioxide)	58.70
NOx (as ozone)	8,500.00
PM_{10}	232,000.00
Hydrocarbon	2,200

Source: (Coffey Geosciences 2003)

6.5 Assumptions made in the health cost modelling

A number of assumptions have been made when calculating the health costs and these are as follows:

1. Hydrocarbon emissions were not been speciated in the analysis. However, a further analysis using emission fractions for certain air toxics has also been conducted.
2. Oxides of nitrogen exert their health effects through conversion to nitrogen dioxide.
3. As there is no simple method to estimate ozone concentrations from the primary precursor air pollutants, an analysis has been conducted assuming 20 percent of oxides of nitrogen emissions are converted to ozone.
4. Ozone is derived only from oxides of nitrogen and that hydrocarbons are not involved in ozone formation.
5. Health costs per tonne of air pollutant derived from the report by Coffey (Coffey Geosciences 2003).
6. VKTs for 2000 were derived from Coffey (Coffey Geosciences 2003).
7. Emission factors were derived from Coffey (Coffey Geosciences 2003).

6.6 Health costs of different fuel usage distribution

The health costs of emissions by different vehicle categories and fuel types are presented and discussed in the following sections.

6.6.1 Health costs of PM_{10}

Of the four air pollutants considered in this section, the health costs are greatest for PM_{10} (Tables 6.14 to 6.16). The cost of diesel emissions (in all three scenarios) far outweigh the cost of petrol or LPG/CNG emissions and are mainly due to emissions from trucks (both rigid and articulated) and buses. This is despite much lower VKT for trucks and buses compared to passenger cars. The health costs for both 2012 and 2020 are lower than the base scenario because of the increased projected use of CNG and LPG fuels.

Table 6.14 Estimated health costs for PM_{10} for 2000 (base case)

Vehicle Category	Health costs A$ millions			
	Petrol	Diesel	CNG	LPG
Passenger car and 4 wheel drive	583.6	121.8	0.0	10.0
Light commercial vehicle	78.3	195.1	0.0	1.9
Rigid truck	0.0	528.6	0.0	0.0
Bus	0.0	119.8	0.1	0.0
Articulated truck	0.0	158.5	0.0	0.0
Total	661.9	1,123.8	0.1	11.8

Table 6.15 Estimated health costs for PM_{10} for projected fuel usage distribution scenario for 2012

Vehicle Category	Health costs A$ millions											
	Petrol			Diesel			CNG			LPG		
	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	229.3	224.1	218.9	161.9	134.9	108.0	0.0	0.0	0.0	6.3	9.4	12.5
Light commercial vehicle	27.2	27.2	28.3	328.8	292.3	255.7	0.0	0.0	0.0	1.6	2.5	3.4
Rigid truck	0.0	0.0	0.0	272.9	264.2	255.6	1.8	3.0	3.6	1.2	1.8	3.0
Bus	0.0	0.0	0.0	49.0	47.0	43.8	0.5	0.6	0.9	0.1	0.3	0.6
Articulated truck	0.0	0.0	0.0	98.1	97.6	96.6	0.7	1.0	1.5	0.0	0.0	0.0
Total	256.5	251.3	247.2	910.7	836.0	759.8	3.0	4.6	6.0	9.1	14.0	19.5

Table 6.16 Estimated health costs for PM_{10} for projected fuel usage distribution scenario for 2020

Vehicle Category	Health costs A$ millions											
	Petrol			Diesel			CNG			LPG		
	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	27.0	27.0	27.0	215.1	154.9	86.1	0.0	0.0	0.0	1.3	2.5	3.5
Light commercial vehicle	3.5	3.5	3.1	140.7	115.7	93.8	0.2	0.3	0.5	0.4	0.6	0.8
Rigid truck	0.0	0.0	0.0	73.7	67.2	59.8	3.1	5.2	7.7	2.1	4.1	6.2
Bus	0.0	0.0	0.0	8.8	8.0	7.1	0.5	0.8	1.2	0.3	0.6	0.8
Articulated truck	0.0	0.0	0.0	28.0	27.0	26.0	1.2	2.5	3.8	0.0	0.0	0.0
Total	30.4	30.4	30.1	466.3	372.8	272.7	5.0	8.8	13.3	4.1	7.8	11.3

6.6.2 Health costs of oxides of nitrogen

6.6.2.1 *Health costs of nitrogen dioxide*

In this section it is assumed that all emissions of oxides of nitrogen exert their health effects as nitrogen dioxide. The health costs of nitrogen dioxide are given in Tables 6.17 to 6.19. The total health costs in the base scenario (2000) are only about $9 million, with most of the cost attributable to emissions from petrol powered vehicles. The LPG and CNG contribution to the health costs increases only marginally despite the greatly projected increase in the use of these fuels.

Table 6.17 Estimated health costs for nitrogen dioxide for 2000 (base case)

Vehicle Category	Health costs A$ millions			
	Petrol	Diesel	CNG	LPG
Passenger car and 4 wheel drive	5.0	0.1	0.0	0.1
Light commercial vehicle	0.8	0.2	0.0	0.0
Rigid truck	0.0	1.6	0.0	0.0
Bus	0.0	0.6	0.0	0.0
Articulated truck	0.0	0.8	0.0	0.0
Total	5.8	3.2	0.0	0.2

Table 6.18 Estimated health costs for nitrogen dioxide for projected fuel usage distribution scenario for 2012

Vehicle Category	Health costs A$ millions											
	Petrol			Diesel			CNG			LPG		
	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	2.3	2.2	2.2	0.2	0.2	0.1	0.0	0.0	0.0	0.1	0.2	0.2
Light commercial vehicle	0.3	0.3	0.3	0.5	0.5	0.4	0.0	0.0	0.0	0.0	0.0	0.1
Rigid truck	0.0	0.0	0.0	1.2	1.1	1.1	0.0	0.0	0.0	0.0	0.0	0.0
Bus	0.0	0.0	0.0	0.3	0.3	0.3	0.0	0.0	0.0	0.0	0.0	0.0
Articulated truck	0.0	0.0	0.0	0.8	0.8	0.8	0.0	0.0	0.0	0.0	0.0	0.0
Total	2.6	2.5	2.5	3.0	2.9	2.7	0.0	0.0	0.0	0.1	0.2	0.2

Table 6.19 Estimated health costs for nitrogen dioxide for projected fuel usage distribution scenario for 2020

Vehicle category	Health costs A$ millions											
	Petrol			Diesel			CNG			LPG		
	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	0.7	0.7	0.7	0.5	0.3	0.2	0.0	0.0	0.0	0.1	0.1	0.1
Light commercial vehicle	0.1	0.1	0.1	0.3	0.2	0.2	0.0	0.0	0.0	0.0	0.0	0.0
Rigid truck	0.0	0.0	0.0	0.6	0.5	0.5	0.0	0.1	0.1	0.0	0.0	0.1
Bus	0.0	0.0	0.0	0.1	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0
Articulated truck	0.0	0.0	0.0	0.4	0.4	0.4	0.0	0.0	0.0	0.0	0.0	0.0
Total	0.8	0.8	0.8	1.9	1.6	1.3	0.1	0.1	0.1	0.1	0.2	0.3

6.6.2.2 Health costs of ozone

In this section, it is assumed that 20 percent of all emissions of oxides of nitrogen are converted to ozone and the dollar costs for ozone are applied ($8,500/tonne ozone).

The health costs of ozone are shown in Tables 6.20 to6.22. In the base scenario, the overwhelming majority of the ozone health costs are attributable to the use of petrol and diesel fuels. Even in the high LPG use scenarios, the health costs of attributable to LPG and CNG fuels are very small.

Table 6.20 Estimated health costs for ozone for 2000 (base case)

Vehicle Category	Health costs A$ millions			
	Petrol	Diesel	CNG	LPG
Passenger car and 4 wheel drive	144.9	2.9	0.0	4.2
Light commercial vehicle	22.1	5.6	0.0	0.9
Rigid truck	0.0	45.0	0.0	0.0
Bus	0.0	16.5	0.0	0.0
Articulated truck	0.0	21.9	0.0	0.0
Total	167.0	91.8	0.0	5.0

Table 6.21 Estimated health costs for ozone for projected fuel usage distribution scenario for 2012

Vehicle Category	Health costs A$ millions											
	Petrol			Diesel			CNG			LPG		
	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	65.3	63.8	62.3	6.0	5.0	4.0	0.0	0.0	0.0	3.0	4.5	6.0
Light commercial vehicle	9.3	9.3	9.7	14.7	13.0	11.4	0.0	0.0	0.0	0.9	1.4	1.9
Rigid truck	0.0	0.0	0.0	33.7	32.6	31.6	0.6	0.9	1.1	0.4	0.6	0.9
Bus	0.0	0.0	0.0	8.7	8.3	7.8	0.1	0.2	0.3	0.0	0.1	0.2
Articulated truck	0.0	0.0	0.0	24.6	24.5	24.2	0.0	0.0	0.0	0.0	0.0	0.0
Total	74.5	73.1	72.0	87.6	83.4	78.9	0.7	1.1	1.4	4.3	6.5	9.0

Table 6.22 Estimated health costs for ozone for projected fuel usage distribution scenario for 2020

Vehicle Category	Health costs A$ millions											
	Petrol			Diesel			CNG			LPG		
	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	19.3	19.3	19.3	13.1	9.4	5.2	0.0	0.0	0.0	1.6	3.0	4.2
Light commercial vehicle	3.4	3.4	3.1	8.2	6.7	5.4	0.4	0.5	0.8	0.6	0.9	1.3
Rigid truck	0.0	0.0	0.0	17.1	15.5	13.8	0.9	1.6	2.4	0.6	1.3	1.9
Bus	0.0	0.0	0.0	2.9	2.7	2.3	0.2	0.2	0.4	0.1	0.2	0.2
Articulated truck	0.0	0.0	0.0	12.4	12.0	11.5	0.0	0.0	0.0	0.0	0.0	0.0
Total	22.7	22.7	22.4	53.7	46.3	38.4	1.5	2.3	3.5	2.9	5.4	7.6

6.6.3 Health costs of hydrocarbons

The health costs of hydrocarbons, although substantially less than the health costs of PM_{10}, are still not insignificant (Tables 6.23 to 6.25). Petrol powered engines (mainly passenger cars and light commercial vehicles) are the greatest contributors to the health costs of hydrocarbons. LPG and CNG fuels make greater contributions to the hydrocarbon health costs as the use of these fuels is projected to increase in 2012 and 2020. However, the costs are only marginally increased.

Table 6.23 Estimated health costs for hydrocarbons for 2000 (base case)

Vehicle Category	Health costs A$ millions			
	Petrol	Diesel	CNG	LPG
Passenger car and 4 wheel drive	113.3	2.3	0.0	5.8
Light commercial vehicle	14.6	4.0	0.0	1.0
Rigid truck	0.0	28.0	0.0	0.0
Bus	0.0	4.4	0.0	0.0
Articulated truck	0.0	3.0	0.0	0.0
Total	127.9	41.8	0.0	6.9

Table 6.24 Estimated health costs for hydrocarbons for projected fuel usage distribution scenario for 2012

Vehicle Category	Health costs A$ millions											
	Petrol			Diesel			CNG			LPG		
	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	60.8	59.4	58.0	2.6	2.2	1.7	0.0	0.0	0.0	5.0	7.5	10.0
Light commercial vehicle	8.7	8.7	9.1	5.2	4.7	4.1	0.0	0.0	0.0	1.5	2.4	3.3
Rigid truck	0.0	0.0	0.0	13.9	13.4	13.0	0.6	1.0	1.2	0.4	0.6	1.0
Bus	0.0	0.0	0.0	1.9	1.8	1.7	0.2	0.2	0.3	0.0	0.1	0.2
Articulated truck	0.0	0.0	0.0	3.3	3.2	3.2	0.0	0.0	0.0	0.0	0.0	0.0
Total	69.5	68.2	67.1	26.9	25.3	23.7	0.8	1.2	1.5	6.9	10.6	14.4

Table 6.25 Estimated health costs for hydrocarbons for projected fuel usage distribution scenario for 2020

Vehicle category	Health costs A$ millions											
	Petrol			Diesel			CNG			LPG		
	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	28.2	28.2	28.2	3.1	2.3	1.3	0.0	0.0	0.0	4.2	7.8	10.9
Light commercial vehicle	5.3	5.3	4.7	1.9	1.5	1.2	1.0	1.3	2.1	1.6	2.6	3.6
Rigid truck	0.0	0.0	0.0	4.1	3.7	3.3	1.0	1.7	2.6	0.7	1.4	2.1
Bus	0.0	0.0	0.0	0.5	0.4	0.4	0.2	0.3	0.4	0.1	0.2	0.3
Articulated truck	0.0	0.0	0.0	2.1	2.0	1.9	0.0	0.0	0.0	0.0	0.0	0.0
Total	33.4	33.4	32.9	11.6	9.9	8.1	2.2	3.3	5.1	6.6	12.0	16.8

6.6.3.1 *Health costs of air toxics*

The health costs of hydrocarbons presented above are for total hydrocarbon emissions and this may be an overestimate of the actual health costs attributable to hydrocarbons. There has been no attempt to speciate the air toxics. As the percentage of air toxics (of total hydrocarbon emission) varies with the fuel type, calculating only the total hydrocarbon emission health costs will not represent the true contribution of each of the different types of fuels to the total health costs.

In this section, the health costs of six specific air toxics are calculated. The six air toxics are: benzene, 1,3-butadiene, PAH, toluene, xylenes and formaldehyde. These six hydrocarbons have been selected because NEPMs have been made for five of them as they pose a risk to human health (benzene, PAH, toluene, xylenes and formaldehyde) and 1,3-butadiene is a known human carcinogen. The speciation factors for these six air toxics were obtained from the National Pollutant Inventory Manual (NPI 2000).

The speciation factors for these six air toxics are given in Table 6.26. For petrol, diesel and LPG hydrocarbon emissions, the percentage of these six air toxics are 27.1 percent, 12.7 percent and 0.18 percent respectively. Therefore, the total health costs, as presented in Table 6.23, can now be apportioned to each of the three fuels (Table 6.27).

Table 6.26 Hydrocarbon speciation factors for air toxics

	Petrol Exhaust	Diesel Exhaust	Lpg Exhaust
Benzene	0.0658	0.0101	0.00000943
1,3 butadiene	0.00649	0.00115	0.0000552
PAHs	0.00217	0.00667	
Toluene	0.105	0.0147	
Xylenes	0.0759	0.0117	
Formaldehyde	0.0156	0.0826	0.00178
Total	0.27096	0.12692	0.00184463

Source: (NPI 2000)

Table 6.27 Health costs of all hydrocarbon emission and six selected air toxic emissions

Vehicle Category	Health Costs A$ Millions			
	Petrol	Diesel	CNG	**LPG**
All Hydrocarbon Emissions	127.9	41.8	0.0	6.9
Six Selected Air Toxics	34.7	5.3	0.0	0.01

When only the costs of air toxics are calculated, not only is the overall health costs reduced (Table 6.27), but the health cost attributable to LPG use is negligible.

6.6.4 Health costs of carbon monoxide

The total health costs of carbon monoxide emissions are very small compared to the costs of PM_{10} and hydrocarbon emissions (Tables 6.28 to 6.30). The major health costs are attributable to emissions from petrol powered vehicles. Even in the high LPG use scenarios, the health costs of carbon monoxide due to LPG use are minimal.

Table 6.28 Estimated health costs for carbon monoxide for 2000 (base case)

Vehicle Category	Health costs A$ millions			
	Petrol	Diesel	CNG	LPG
Passenger car and 4 wheel drive	8.1	0.0	0.0	0.4
Light commercial vehicle	1.1	0.1	0.0	0.1
Rigid truck	0.0	0.2	0.0	0.0
Bus	0.0	0.1	0.0	0.0
Articulated truck	0.0	0.1	0.0	0.0
Total	9.2	0.5	0.0	0.5

Table 6.29 Estimated health costs for carbon monoxide for projected fuel usage distribution scenario for 2012

Vehicle Category	Health costs A$ millions											
	Petrol			Diesel			CNG			LPG		
	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	2.6	2.5	2.4	0.1	0.0	0.0	0.0	0.0	0.0	0.2	0.3	0.4
Light commercial vehicle	0.3	0.3	0.3	0.1	0.1	0.1	0.0	0.0	0.0	0.0	0.1	0.1
Rigid truck	0.0	0.0	0.0	0.1	0.1	0.1	0.1	0.1	0.1	0.0	0.1	0.1
Bus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Articulated truck	0.0	0.0	0.0	0.1	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0
Total	2.8	2.8	2.7	0.4	0.4	0.3	0.1	0.1	0.1	0.3	0.4	0.6

Table 6.30 Estimated health costs for carbon monoxide for projected fuel usage distribution scenario for 2020

Vehicle Category	Health costs A$ millions											
	Petrol			Diesel			CNG			LPG		
	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	0.9	0.9	0.9	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.2	0.3
Light commercial vehicle	0.1	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1
Rigid truck	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.2	0.2	0.1	0.1	0.2
Bus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Articulated truck	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total	1.0	1.0	1.0	0.2	0.2	0.1	0.1	0.2	0.3	0.2	0.4	0.6

6.7 Total health costs by vehicle type

The total health costs for each of the three fuel usage scenarios by vehicle type are given in Tables 6.31 to 6.34.

6.7.1 Total health costs of PM_{10}

The total health cost for 2000, 2012 and 2020 are given in Table 6.31. The health costs are greater for the base year (2000) because of the increased use of diesel. However, it is interesting to note that in the high LPG/CNG use estimate for 2020, the total PM_{10} health costs are lower than for the low and central LPG/CNG use estimates. This is due to the projected increase in the use of LPG/CNG fuels at the expense of diesel fuel.

Table 6.31 Total health costs of PM_{10} by vehicle type for the three projected fuel usage scenarios

Vehicle Category	Health costs A$ millions						
	2000	2012			2020		
	Base	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	715.3	397.5	368.4	339.3	243.5	184.4	116.5
Light commercial vehicle	275.2	357.6	321.9	287.4	144.7	120.0	98.1
Rigid truck	528.6	275.9	269.1	262.3	78.9	76.5	73.7
Bus	119.9	49.6	47.9	45.3	9.7	9.4	9.1
Articulated truck	158.5	98.8	98.6	98.1	29.2	29.5	29.8
Total	1,797.5	1,179.4	1,105.9	1,032.4	505.9	419.8	327.4

6.7.2 Total health costs of oxides of nitrogen

There are differences in health costs attributable to oxides of nitrogen (as nitrogen dioxide) between the three fuel usage scenarios (Table 6.32). However, the overall health costs attributable to oxides of nitrogen (as nitrogen dioxide) are insignificant compared to the health costs of PM_{10} and hydrocarbons.

Table 6.32 Total health costs of oxides of nitrogen by vehicle type for the three projected fuel usage scenarios

Vehicle Category	Health costs A$ millions						
	2000	2012			2020		
	Base	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	5.2	2.6	2.5	2.5	1.2	1.1	1.0
Light commercial vehicle	1.0	0.9	0.8	0.8	0.4	0.4	0.4
Rigid truck	1.6	1.2	1.2	1.2	0.6	0.6	0.6
Bus	0.6	0.3	0.3	0.3	0.1	0.1	0.1
Articulated truck	0.8	0.8	0.8	0.8	0.4	0.4	0.4
Total	9.1	5.8	5.7	5.6	2.8	2.6	2.5

6.7.3 Total health costs of hydrocarbons

Although there are differences in the total health costs attributable to hydrocarbons in 2000, 2012 and 2020 (Table 6.33), the differences between the low, central and high LPG scenarios are minimal.

Table 6.33 Total health costs of air toxics by vehicle type for the three projected fuel usage scenarios

Vehicle Category	**Health costs A$ millions**						
	2000	**2012**			**2020**		
	Base	**Low**	**Central**	**High**	**Low**	**Central**	**High**
Passenger car & 4 wheel drive	121.4	68.4	69.1	69.7	35.5	38.2	40.3
Light commercial vehicle	19.6	15.5	15.8	16.5	9.8	10.7	11.7
Rigid truck	28.0	14.9	15.1	15.2	5.8	6.8	8.0
Bus	4.4	2.1	2.1	2.2	0.8	0.9	1.1
Articulated truck	3.0	3.3	3.2	3.2	2.1	2.0	1.9
Total	176.5	104.1	105.3	106.8	53.9	58.7	63.0

6.7.4 Total health costs of carbon monoxide

The total health costs attributable to carbon monoxide are presented in Table 6.34. There are only small differences in the health costs among the three fuel usage scenarios and the absolute health costs are very small compared to the health costs of PM_{10}.

Table 6.34 Total health costs of carbon monoxide by vehicle type for the three projected fuel usage scenarios

Vehicle Category	Health costs A$ millions						
	2000	2012			2020		
	Base	Low	Central	High	Low	Central	High
Passenger car & 4 wheel drive	8.6	2.8	2.9	2.9	1.1	1.2	1.2
Light commercial vehicle	1.2	0.4	0.4	0.5	0.2	0.2	0.2
Rigid truck	0.2	0.2	0.3	0.3	0.2	0.3	0.5
Bus	0.1	0.0	0.1	0.1	0.0	0.0	0.1
Articulated truck	0.1	0.1	0.1	0.1	0.0	0.0	0.0
Total	10.1	3.6	3.7	3.8	1.5	1.8	2.0

6.8 Summary

- PM_{10} is the major contributor to the total health costs of air pollution
- Diesel fuel makes a disproportionate contribution to the PM_{10} health costs
- Hydrocarbons make a smaller contribution to the total air pollution health costs
- LPG emissions, due to its very low air toxic fraction, make a negligible contribution to the total health costs of air toxics
- Health costs attributable to ozone are mainly from the use of petrol and diesel fuels
- Oxides of nitrogen and carbon monoxide make only small contributions to the total air pollution health costs
- There are health costs savings to be made with increasing the use of alternate fuels, for example, LPG and CNG, at the expense of diesel fuel, especially for heavy trucks and buses

6.9 References

Coffey Geosciences (2003) . . . . . Fuel quality and vehicle emissions standards cost benefit analysis
Coffey Geosciences Pty Ltd.

NPI (2000). . . . . Emissions estimation technique manual
National Pollution Inventory, Department of Conservation and Heritage

Appendix A

Table A1 Summary statistics for daily hospital admissions and particulate air pollutant concentrations for the meta-analysis (years 1996-99) (Simpson, Williams et al. 2005b)

	Brisbane	Sydney	Melbourne	Perth
	mean (range)	mean (range)	mean (range)	mean (range)
Daily Hospital Admissions				
Cardiac				
All ages	17.7 (1-44)	68.8 (37-113)	55.7 (26-91)	24.6 (8-43)
≥65 years	12.6 (0-34)	44.8 (21-77)	37.1 (15-60)	17.1 (3-32)
15-64 years	5.1 (0-17)	23.7 (9-45)	18.5 (4-38)	7.3 (0-17)
Ischaemic heart disease				
All ages	9.6 (0-24)	33.8 (15-62)	30.2 (14-55)	12.9 (2-27)
≥65 years	6.6 (0-20)	20.8 (6-44)	18.9 (6-36)	8.5 (1-20)
Stroke				
≥65 years	3.1 (0-15)	12.2 (2-31)	11.0 (3-24)	4.4 (0-12)
Total Respiratory				
≥65 years	7.1 (0-28)	31.3 (9-88)	24.9 (4-66)	10.5 (1-32)
Asthma				
15-64 years	2.2 (0-15)	8.6 (0-29)	6.3 (0-24)	2.6 (0-13)
Asthma + COPD				
≥65 years	3.1 (0-15)	15.1 (1-41)	10.2 (0-30)	5.2 (0-18)
Pneumonia + Acute Bronchitis				
≥65 years	2.6 (0-12)	10.3 (0-33)	10.1 (0-30)	3.1 (0-12)
Daily PM Levels				
BSP ($10^{-4}m^{-1}$) 24 hour	0.3 (0.0-2.5)	0.3 (0.0-1.6)	0.3 (0.0-2.2)	0.3 (0.1-1.8)
PM_{10} ($\mu g/m^3$) 24 hour	16.5 (2.6-57.6)	16.3 (3.7-75.5)	18.2 (3.3-51.9)	
$PM_{2.5}$ ($\mu g/m^3$) 24 hour	7.5 (1.9-19.7)	9.0a (2.4-35.3)	9.3a (2.7-35.1)	9.0 (2.8-37.3)

Table A2 Daily hospitalisations – mean and range for the earlier studies on individual Australian cities

Outcome	ICD 9	Mean	Range
Brisbane (Petroeschevsky, Simpson et al. 2001)			
Asthma 0-14 years	493	2.6	0-18
Asthma – All	493	4.5	0-23
Respiratory – 65+ years	460-519	3.1	0-21
Respiratory – All	460-519	11.5	1-35
Cardiovascular - All	390-459	14.1	2-36
Sydney (Morgan, Corbett et al. 1998a)			
Asthma 0-14 years	493	15.5	1-76
Asthma – 15-64 years	493	9.0	0-27
COPD – 65+ years	490-492, 494, 496	9.7	0-33
Cardiovascular - All	410, 413, 427, 428	47.2	17-89
Melbourne (Denison, Simpson et al. 2001)			
Asthma 0-14 years	493	9.65	0-29
Asthma – All	493	18.47	3-52
Respiratory – 65+ years	460-519	24.08	8-68
Respiratory – All	460-519	65.87	21-132
Cardiovascular - All	390-459	84.04	47-129
Perth (Department of Environment WA 2003)			
Asthma 0-14 years	493	5.6	2-10 (10^{th}-90^{th} percentiles)
Asthma – All	493	8.8	4-15 (10^{th}-90^{th} percentiles)
Other COPD	490-492, 494, 496	3.3	1-6 (10^{th}-90^{th} percentiles)
Respiratory – All	460-519	25.3	14-38 (10^{th}-90^{th} percentiles)
Cardiovascular - All	390-459	26.5	19-35 (10^{th}-90^{th} percentiles)

Table A3 Particulate levels from the earlier time series hospitalisation studies conducted in four individual Australian cities

Location (study period)	Period of study	Averaging period	Particulate pollution levels[1] Mean (range)^		
			Whole Study Period	Cool Season	Warm Season
Brisbane (Petroeschevsky, Simpson et al. 2001)	1987-1994	1 hour maximum	7.01 (0.78-162.4) BSP $10^{-5}m^{-1}$	8.60 (1.10-162.4)# BSP $10^{-5}m^{-1}$	5.28 (0.78-81.0) BSP $10^{-5}m^{-1}$
Sydney (Morgan, Corbett et al. 1998a)	1990-1994	24 hour	2.74 (0.30-50.8) BSP $10^{-5}m^{-1}$	3.32 (0.47-50.8)# BSP $10^{-5}m^{-1}$	2.08 (0.42-15.9) BSP $10^{-5}m^{-1}$
		1 hour	0.76 (0.01-7.86) BSP $10^{-4}m^{-1}$	N/a*	N/a*
Melbourne (Denison, Simpson et al. 2001)	1994-1997	24 hour	0.32 (0.01-3.72) BSP $10^{-4}m^{-1}$	N/a*	N/a*
		1 hour	0.55 (0.07-3.26) BSP $10^{-4}m^{-1}$	0.66 (0.07-3.26) BSP $10^{-4}m^{-1}$	0.40 (0.08-2.73) BSP $10^{-4}m^{-1}$
Perth (Department of Environment WA 2003)	1992-1997	24 hour	0.24 (0.03-2.00) BSP $10^{-4}m^{-1}$	0.27 (0.03-2.00) BSP $10^{-4}m^{-1}$	0.19 (0.03-1.25) BSP $10^{-4}m^{-1}$
		1 hour	1.2 (0.3-2.6) BSP $10^{-4}m^{-1}$	1.61 (0.39-3.73) BSP $10^{-4}m^{-1}$	0.74 (0.25-1.39) BSP $10^{-4}m^{-1}$
		24 hour	0.25 (0.1-0.47) BSP $10^{-4}m^{-1}$	0.3 (0.12-0.57) BSP $10^{-4}m^{-1}$	0.2 (0.09-0.33) BSP $10^{-4}m^{-1}$

Notes

* Not available

\# Brisbane: Cool= Spring (September-November) since levels in Spring were slightly above Winter (June-August), Warm=December-February. Melbourne: Cool=April-October, Warm=November-March; Perth: Cool= May-October, Warm = November-April

^ 10-90th percentile for Perth.

[1] PM is reported as black smoke, BSP, PM_{10} and $PM_{2.5}$. The conversion factors for these are dependent on the city. To convert to ug/m^3 the conversion factors are dependent on the city. For Sydney the conversion factor is: $PM_{2.5}$ = 30 x BSP $10^{-4}m^{-1}$, PM_{10} = 2 x $PM_{2.5}$, therefore to convert PM_{10} in ug/m^3 to BSP $10^{-4}m^{-1}$ divide by 60. For Brisbane the conversion factor is: 1 x BSP $10^{-5}m^{-1}$= 0.3 x $PM_{2.5}$ and $PM_{2.5}$ ~ 0.4 x PM_{10}.

Table A4 Australian hospitalisation studies on the health effects of particulate pollution

Author, year and city	Type of hospital admission	Age (years)	Averaging period	RR (95%CI)
(Simpson, Williams et al. 2005b)	Cardiac	All	24 hour	1.0856 (1.0603-1.1116)
1996-1999	Cardiac	15-64	24 hour	1.0446 (1.0021-1.0889)
Brisbane, Melbourne, Sydney and Perth	Cardiac	>65	24 hour	1.1013 (1.0701-1.1334)
	Ischemic heart disease	All	24 hour	1.0872 (1.0516-1.1240)
	Ischemic heart disease	>65	24 hour	1.1029 (1.0591-1.1486)
	Respiratory	>65	24 hour	1.0552 (1.0082-1.1045)
	Asthma	15-64	24 hour	1.0893 (1.0240-1.1587)
	Asthma and COPD	>65	24 hour	1.0713 (1.0179-1.1276)
	Pneumonia and acute bronchitis	>65	24 hour	1.0769 (1.0046-1.1544)
	Asthma	15-64	24 hour	1.016 (0.994-1.038)
	Asthma	All	1 hour maximum	0.999 (0.995-1.002)
	Respiratory	0-4	1 hour maximum	1.002 (0.999-1.006)
	Respiratory	5-14	1 hour maximum	0.999 (0.995-1.005)
	Respiratory	15-64	1 hour maximum	1.005 (1.001-1.010)*
	Respiratory	65+	1 hour maximum	1.002 (0.999-1.005)
	Respiratory	All	24 hour	1.015 (1.006-1.023)*
	Cardiovascular	15-64	24 hour	0.995 (0.911-1.087)
	Cardiovascular	65+	1 hour maximum	0.998 (0.996-0.999)
	Cardiovascular	All	1 hour maximum	0.998 (0.997-0.999)
	Respiratory	15-64	1 hour maximum	1.002 (0.995-1.008)
	Respiratory	All	24 hour	1.015 (1.004-1.026)*
	Respiratory	All	24 hour	1.015 (1.007-1.024)*
(Morgan, Corbett et al. 1998a)	Asthma	1-14	1 hour maximum	1.008 (0.9726-1.0449)
1990-1994	Asthma	15-64	1 hour maximum	1.0225 (0.9864-1.0598)
Sydney	COPD	65+	1 hour maximum	1.031 (0.9962-1.0652)
	Heart Disease	All	1 hour maximum	1.0192 (1.0047-1.0339)*
	Heart Disease	65+	1 hour maximum	1.0272 (1.0077-1.047)*
	Heart Disease	0-64	1 hour maximum	1.0028 (0.9762-1.0295)
	Asthma	1-14	24 hour	0.9913 (0.9537-1.0302)
	Asthma	15-64	24 hour	1.0121 (0.9764-1.0510)
	COPD	65+	24 hour	1.0241 (0.9910-1.0584)
	Heart Disease	All	24 hour	1.0223 (1.0061-1.0388)
	Heart Disease	65+	24 hour	1.0282 (1.009-1.0477)*
	Heart Disease	0-64	24 hour	1.0102 (0.983-1.0381)*
	Heart Disease	65+	24 hour	1.0017 (0.982-1.0236)

Unit increase	Ambient particulate level mean (SD)	Adjusted confounders
1x0^{-4}m^{-1}	0.3 (0.0-2.5)	Season, influenza, day, long term, holiday, temperature, humidity, single pollutant models for BSP and significant for multi-pollutant models for cardiac all ages
	(0.78-162.4) 10^{-5}m^{-1}	
	2.74 for 24 hour (0.30-50.8) 10^{-5}m^{-1}	Single pollutant models for BSP only
		Not significant in a multi-pollutant model controlling for high ozone
		Significant in a multi-pollutant model controlling for high ozone
		Significant in a multi-pollutant model controlling for high SO_2
0.25-1.48 10^{-4}m^{-1}	0.76 for 1 hour max (0.01-7.86) 10^{-4}m^{-1}	Weather, season, long term trends, day, holidays, single pollutant
0.12-0.6 10^{-4}m^{-1}	0.32 for 24 hour average (0.01-3.72) 10^{-4}m^{-1}	Weather, season, long term trends, day, holidays, single pollutant
		Not significant in multi-pollutant models (above plus ozone and NO_2)

Table A4 Australian hospitalisation studies on the health effects of particulate pollution, continued

Author, year and city	Type of hospital admission	Age (years)	Averaging period	RR (95%CI)
(Denison, Simpson et al. 2001)	Respiratory	15-64	1 hour maximum	1.0383 (1.0063-1.0714)*
1994-1997	Respiratory	65+	1 hour maximum	1.043 (1.0088-1.0783)*
Melbourne	Respiratory	All	1 hour maximum	1.0119 (0.9967-1.0273)
	Asthma	0-14	1 hour maximum	1.0592 (1.0197-1.1002)*
	Asthma	All	1 hour maximum	1.0766 (1.0374-1.1172)*
	Cardiovascular	65+	1 hour maximum	1.0352 (1.0143-1.0565)*
	Cardiovascular	All	1 hour maximum	1.0274 (1.0104-1.0446)
	Ischaemic heart disease		1hour maximum	1.0297 (1.0090-1.0509)
	Respiratory	15-64	24 hour	1.0784 (1.0121-1.1491)*
	Respiratory	65+	24 hour	1.0745 (1.0041-1.1499)*
	Respiratory	All	24 hour	1.0239 (0.9927-1.0561)
	Asthma	0-14	24 hour	1.1481 (1.0628-1.2403)*
	Asthma	All	24 hour	1.1394 (1.0582-1.2268)*
	Cardiovascular	65+	24 hour	1.0560 (1.0208-1.0924)*
	Cardiovascular	All	24 hour	1.0461 (1.0174-1.0757)
	Ischaemic heart disease		24 hour.	1.0631 (1.0188-1.1093)*
	Respiratory	65+	1 hour maximum	1.0041 (1.0014-1.0069)*
	Respiratory	65+	1 hour maximum	1.0017 (1.0003-1.0031)*
	Cardiovascular	65+	1 hour maximum	1.0188 (1.0016-1.0362)*
	Cardiovascular	65+	24 hour	1.0021 (1.005-1.0037)*
	Cardiovascular	65+	24 hour	1.0172 (1.0045-1.0301)*
(Department of Environment WA 2003)	Respiratory	65+	1 hour maximum	1.0196 (1.0048-1.0347)*
1992-1997	COPD	All	1 hour maximum	1.0347 (1.0125-1.0573)*
Perth	COPD	65+	1 hour maximum	1.0492 (1.0239-1.0752)*
	COPD	65+	24 hour	1.2431 (1.0377-1.4892)*

	Unit increase	Ambient particulate level mean (SD)	Adjusted confounders
	$1x10^{-4}m^{-1}$	0.55 (0.48) x $10^{-4}m^{-1}$ for 1 hour max. (all year) 0.24 (0.23) x $10^{-4}m^{-1}$ for 24 hour (all year)	Weather, season, long term trends, day, holidays, single pollutant BSP and NO_2 in multi-pollutant models BSP and O_3 in multi-pollutant models BSP and CO in multi-pollutant models BSP and NO_2 in multi-pollutant models BSP and CO in multi-pollutant models
	$1x10^{-4}m^{-1}$	1.2 (0.3-2.6) $10^{-4}m^{-1}$ 0.25 (0.1-0.47) $10^{-4}m^{-1}$	Case cross-over analyses - current day with 1 week before and 1 week after. Adjusted for humidity, temperature, day and holidays.

Appendix B

Table B1 Descriptive data for the meta-analysis of four Australian cities for the period 1996-99 (Simpson, Williams et al. 2005a)

	Brisbane Mean (range)	**Sydney** Mean (range)	**Melbourne** Mean (range)	**Perth** Mean (range)
Mortality				
Total all-cause (all ages)	16.03 (5-33)	56.83 (31-103)	56.10 (30-90)	20.26 (5-40)
Respiratory disease (all ages)	1.51 (0-8)	5.43 (0-25)	4.92 (0-17)	1.96 (0-11)
Cardiovascular disease (all ages)	7.42 (1-18)	25.49 (10-56)	23.19 (8-41)	8.20 (0-20)
Cardiovascular disease (≥65 years)	6.78 (0-18)	22.78 (8-54)	20.91 (7-38)	7.38 (0-18)
Particulate Pollution				
BSP ($10^{-4}m^{-1}$)	0.26	0.25	0.26	0.25
24 hour	(0.02-2.49)	(0.04-1.58)	(0.03-2.23)	(0.07-1.76)
PM_{10} ($\mu g/m^3$)	16.50	16.30	18.20	
24 hour	(2.6-57.6)	(3.7-75.5)	(3.3-51.9)	
$PM_{2.5}$ ($\mu g/m^3$)	7.50b	9.00a	9.30a	9.0
24 hour	(1.9-19.7)	(2.4-35.3)	(2.7-35.1)	(2.8-37.3)

Table B2 Daily mortality – mean and range for earlier studies on individual cities

Outcome	ICD 9	Mean	Range
Sydney (Morgan, Corbett et al. 1998b)			
Asthma - All	N/a		
Respiratory - All	460-519	4.7	0-17
Cardiovascular - All	390-459	28.8	9-60
Brisbane (Simpson, Williams et al. 1997)			
Asthma - All	N/a		
Respiratory - All	460-519	1	N/a
Cardiovascular - All	393-399, 402, 404, 410-416, 420, 429	5	N/a
Melbourne (Simpson, Denison et al. 2000a)			
Asthma - All	N/a		
Respiratory - All	460-519	4.5	0-16
Cardiovascular - All	390-459	24.3	8-43
Perth (Department of Environment WA 2003)			
Asthma - All	N/a		
Respiratory - All	460-529	2.4	0-5 (10-90 percentiles)
Cardiovascular - All	390-459	11.6	7-17 (10-90 percentiles)

Note

N/a not available

Table B3 Particulate pollution levels from time series mortality on earlier studies conducted in four individual Australian cities

Location (study period)	Period of study	Averaging period	Particulate pollution levels[1] mean (range)#		
			Whole Study Period	**Cool Season**	**Warm Season**
Brisbane (Simpson, Williams et al. 1997)	1987-1993	1 hour maximum	6.68 (0.78-77.3) BSP $10^{-5}m^{-1}$	7.70 (0.95-77.73) BSP 10-5m-1	4.89 (0.78-50.88) BSP $10^{-5}m^{-1}$
Sydney (Morgan, Corbett et al. 1998b)	1989-1993	24 hour	2.59 (0.3-15.16) BSP $10^{-5}m^{-1}$	2.93 (0.3-15.16) BSP $10^{-5}m^{-1}$	2.27 (0.42-14.17) BSP $10^{-5}m^{-1}$
		1 hour maximum	0.70 (0.08-6.16)	N/a	N/a
Melbourne (Simpson, Denison et al. 2000a)	1991-1996	24 hour	0.30 (0.03-1.85) BSP $10^{-4}m^{-1}$	N/a	N/a
		1 hour maximum	0.60 (0.08-4.98) BSP $10^{-4}m^{-1}$	0.70 (0.1-4.36) BSP 10-4m-1	0.46 (0.08-4.98) BSP 10-4m-1
Perth (Department of Environment WA 2003)	1992-1997	24 hour	0.26 (0.04-2.52) BSP $10^{-4}m^{-1}$	0.29 (0.04-2.52) BSP 10-4m-1	0.22 (0.04-2.07) BSP 10-4m-1
		1 hour maximum	1.2 (0.3-2.6)	1.61 (0.39-3.73)	0.74 (0.25-1.39)
		24 hour	0.25 (0.1-0.47) BSP 10-4m-1	0.3 (0.12-0.57) BSP 10-4m-1	0.2 (0.09-0.33) BSP 10-4m-1

Note

N/a = Not available

Brisbane: Cool=June-August, Warm=December-February; Melbourne: Cool=April-October, Warm=November-March; Perth: Cool= May-October, Warm = November-April.

10-90th percentile in Perth

[1] PM is reported as black smoke, BSP, PM_{10} and $PM_{2.5}$. The conversion factors for these are dependent on the city. To convert to ug/m^3 the conversion factors are dependent on the city. For Sydney the conversion factor is: $PM_{2.5}$ = 30 x BSP $10^{-4}m^{-}$, PM_{10} = 2 x $PM_{2.5}$, therefore to convert PM_{10} in ug/m^3 to BSP $10^{-4}m^{-}$divide by 60. For Brisbane the conversion factor is: 1 x BSP $10^{-5}m^{-1}$ = 0.3 x $PM_{2.5}$ and $PM_{2.5}$ ~ 0.4 x PM_{10}.

Table B4 Summary of the Australian studies of ambient particulate pollution and mortality

Author, Year and City	Type of Mortality	Age (years)	Averaging Period	RR (95%CI)
(Simpson, Williams et al. 2005a)	Total	All	24 hour	1.0284 (1.0015-1.0560)
1996-1999	Respiratory	All	24 hour	1.0690 (0.9814-1.1645)
Brisbane, Melbourne, Sydney and Perth	Cardiovascular	All	24 hour	1.0479 (1.0076-1.0898)
	Cardiovascular	>65	24 hour	1.0337 (0.9918-1.0774)
(Simpson, Williams et al. 1997)	Total	All	1 hour maximum	1.002 (1.000-1.004)*
1987-1993	Total	>65	1 hour maximum	1.002 (1.000-1.004)*
Brisbane	Cardiovascular	All	1 hour maximum	1.004 (1.001-1.008)*
	Cardiovascular	>65	1 hour maximum	1.005 (1.001-1.009)*
	Respiratory	All	1 hour maximum	1.001 (0.997-1.005)
	Total	All	24 hour	1.009 (1.003-1.015)*
	Total	>65	24 hour	1.010 (1.002-1.018)*
	Cardiovascular	All	24 hour	1.010 (0.998-1.02)
	Cardiovascular	>65	24 hour	1.011 (0.999-1.023)
(Morgan, Corbett et al. 1998b)	Total	All	1 hour maximum	1.0253 (1.0087-1.042)*
1989-1994	Cardiovascular	All	1 hour maximum	1.0296 (1.0082-1.0514)*
Sydney	Respiratory	All	1 hour maximum	1.041 (0.991-1.0930)
	Total	All	24 hour	1.0263 (1.0087-1.044)*
	Cardiovascular	All	24 hour	1.0268 (1.0025-1.0516)*
	Respiratory	All	24 hour	1.0334 (0.9787-1.0911)
	Total	All	24 hour	1.0229 (1.002-1.044)*
	Cardiovascular	All	24 hour	1.021 (0.9925-1.0497)
	Respiratory	All	24 hour	1.007 (0.9446-1.0747)
(Simpson, Denison et al. 2000a)	Total	All	1 hour maximum or 24 hour	no significant effect (all year)
1991-1996	Respiratory	All	1 hour maximum or 24 hour	no significant effect (all year)
Melbourne	Cardiovascular	All	1 hour maximum or 24 hour	no significant effect (all year)
	Total	All	24 hour $PM_{2.5}$	1.0038 (1.0006-1.007)*
	Respiratory	All	24 hour $PM_{2.5}$	1.0118 (1.0003-1.0232)*
	Respiratory	65+	24 hour $PM_{2.5}$	1.0127 (1.0009-1.0246)*
	Total	All	24 hour PM_{10}	1.0018 (1.0007-1.0033)*
	Respiratory	All	24 hour PM_{10}	1.0059 (1.0006-1.0113)
	Respiratory	65+	24 hour PM_{10}	1.0065 (1.0012-1.0119)
(Department of Environment WA 2003)	No significant associations with mortality			
1992-1997				
Perth				

	Unit Increase	Adjusted Confounders
	$1x10^{-4}m^{-1}$	Weather, long term trends, day, season, holidays, influenza Single pollutant model only, not significant when NO_2 was included in the model
	$1x10^{-5}m^{-1}$ $1x10^{-5}m^{-1}$	Weather, long term trends, day, season, holidays, influenza Single pollutant model only
	$0.23\text{-}1.42\ 10^{-4}m^{-1}$ $0.10\text{-}0.5\ 10^{-4}m^{-1}$	Weather, long term trends, day, holidays, influenza epidemic In multi-pollutant models (NO_2 and ozone)
	$1\ ug/m^3$	Weather, long term trends, day, season, holidays, influenza Single model only, no significant effect in multi-pollutant models Warm season Warm season
		Case cross-over analyses - current day with 1 week before and 1 week after. Adjusted for humidity, temperature, day and holidays.