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Health impacts of transport emissions in Australia: Economic costs

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HEALTH IMPACTS OF TRANSPORT EMISSIONS IN AUSTRALIA: ECONOMIC COSTS

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FOREWORD

Several studies have been initiated in recent years in Australia, and elsewhere in the developed world, that aim to cost the health and environmental effects of ambient concentrations of air pollution. These studies are largely in response to the mounting epidemiological evidence that exposure to air pollutants can be harmful to humans. This report is part of a series of Bureau of Transport and Regional Economics research on the socioeconomic costs of transport impacts. Previous research has concentrated on transport accidents. It is the Bureau's first study on the socioeconomic cost of the health effects of transport emissions.

The Bureau acknowledges with appreciation the assistance of the state and territory environmental protection agencies (EPAs). It particularly wishes to acknowledge the help provided by Sean Walsh (Victorian EPA), Don Neale (Queensland EPA), David Power (Environment ACT) and Alan Betts (New South Wales EPA). Thanks are also due to Dr Bill Physick from the Commonwealth Scientific and Industrial Research Organisation, Professor Tord Kjellstrom from the Australian National University—for advice on methodology—Dr Geoff Morgan, from the Southern Cross Institute of Health Research and Associate Professor Bin Jalaludin, Epidemiology Unit at Liverpool Hospital, for their comments on our initial scoping paper.

The study was undertaken by Johnson Amoako, Madhumita Lodh and Tim Risbey. The project team was led by Christine Williams during the project scoping and subsequently by Tim Risbey. The study was initially directed by Dr Anthony Ockwell and later by Phil Potterton. Dr David Cosgrove provided comments.

Phil Potterton
Executive Director
June 2005

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- Epidemiological research in the 1990s showed that people's health may be affected by exposure to much lower levels of some common air pollutants than previously believed. Health effects range from mild respiratory effects, through to asthma, cardiovascular conditions and premature mortality. While elderly people are most at risk, the very young are also at risk.
- Despite substantial reductions in the levels of many ambient air pollutants, several remain of policy concern. These are particulates and the precursors to photochemical smog—nitrogen oxides and volatile organic compounds.
- In aggregate terms, industrial, electricity generation and wood burning sources, rather than motor vehicles, are the principal emission sources of particulate matter of less than 10 microns in diameter. However, levels of human exposure to motor vehicle sources tend to be comparatively higher. Motor vehicles are the major source of the precursors to photochemical smog in capital cities.
- This study provides some quantitative estimates of the economic costs of the health effects of motor vehicle pollution in Australia. It focuses solely on motor vehicles because the data on emissions sources is not sufficiently comprehensive and is not nationally consistent for other forms of transport. These limitations mean that it is impossible to make reliable estimates of the whole of transport share of any major ambient air pollutant. However, it is clear from the available estimates that aircraft, shipping, boating and rail contribute a comparatively small proportion of total airshed emissions.
- This study estimates that in 2000 motor vehicle-related ambient air pollution accounted for between 900 and 4500 morbidity cases—cardio-vascular and respiratory diseases and bronchitis—and between 900 and 2000 early deaths.
- The economic cost of morbidity ranges from $0.4 billion to $1.2 billion, while the economic cost of mortality ranges from $1.1 billion to $2.6 billion.
- The value of a statistical life used was $1.3 million—a discount of 30 per cent on the Bureau's costing of transport accident fatalities. This reflects the older age profile of air pollution-related early deaths.
- These estimates are derived using the results of a recent international study which estimated the long-term health impacts of ambient air pollution using particulate matter of less than 10 microns as a surrogate for all air pollutants.

EXECUTIVE SUMMARY

This study provides some quantitative estimates of the economic costs of the health effects of motor vehicle pollution in Australia.

AIR POLLUTION IN CONTEXT

Systematic policy responses to air pollution around the world date back more than half a century. Environmental regulation has dramatically reduced the levels of sulfur dioxide, lead and carbon monoxide in most developed countries, including Australia. Several remain the subject of ongoing policy concern including particulate matter of less than 10 microns (PM_{10}) and nitrogen oxides. Both are principally associated with respiratory conditions. Although it is not a directly emitted pollutant, the concentration of ozone in local air is a key indicator of the amount of photochemical smog. Ozone is formed by a reaction between volatile organic compounds and nitrogen oxides under sunlight.

Sulfur dioxide is rarely of concern in urban Australia except near petrochemical industrial areas.

AIR POLLUTION AND MOTOR VEHICLES

Motor vehicles are the major emitters of ambient air pollutants in urban Australia. However, rural and regional Australia face issues of managing windblown dust from mining and agriculture, smoke and agricultural sprays.

Air quality reporting in Australia is an evolving science and is subject to uncertainties. These uncertainties affect estimates of both the absolute levels and the shares contributed by different sources, including motor vehicles. Nevertheless, it is clear that motor vehicles are the principal source of nitrogen oxides and carbon monoxide in the capital city airsheds. In 2000–01, the reference year for this study, motor vehicles are estimated to have contributed 47 per cent of nitrogen oxide levels in Perth and 82 per cent in Southeast Queensland. Motor vehicles are estimated to be the source of more than 60 per cent of carbon monoxide levels for all capital city airsheds other than Darwin.

By contrast, electricity generation and industrial production are the main sources of both sulfur dioxide and PM_{10} emissions in the capital city airsheds. However, the motor vehicle share of PM_{10} emissions is higher in central city

areas—for example, 55 per cent in the Sydney central business district, compared with 30 per cent across the entire Sydney–Newcastle–Wollongong airshed. A high proportion of motor vehicle particulate emissions are very fine particles—particles smaller than one micron (PM_1). Particles smaller than 2.5 microns ($PM_{2.5}$)—including PM_1—are now known to be more highly correlated with cardiopulmonary disease and lung cancer mortality.

AIR POLLUTION AND TRANSPORT

This study focuses on motor vehicles because the data are not sufficiently comprehensive and are not nationally consistent for other forms of transport. These limitations mean that currently it is not possible to make reliable estimates of the whole of transport share of any major pollutant.

Where estimates are available, they indicate that aircraft emissions are small in relation to total airshed emissions. The highest aircraft contribution is estimated to be five per cent—for PM_{10} emissions—in the Adelaide airshed.

Similarly, where estimated, rail emissions were negligible for all capital city airsheds other than Perth—with less than two per cent of total PM_{10}—and Adelaide—with less than two per cent of total nitrogen oxides.

Due to lack of data, estimates for shipping and boating are less reliable than for other transport sources but the magnitudes are clearly small.

Road dust has been excluded due to uncertainty about its contribution, and significance, to total PM_{10} emissions. This implies a possible underestimation of the size of the health effect of transport emissions. However, emissions have not been estimated for some naturally occurring sources of PM_{10} . This will over-estimate the transport share of PM_{10}—and the number of health cases and economics costs—attributed to human activity such as transport.

MEASURING THE HEALTH IMPACT OF AIR POLLUTION

Epidemiological research in the 1990s showed that people's health may be affected by exposure to much lower levels of some common air pollutants than previously believed. Thus, even though Australia may be regarded as a country of comparatively low pollution levels, and despite improvements in recent years, potential health risks remain. Health effects range from subtle or mild effects, through to asthma and premature mortality. While elderly people are most at risk, the very young are also at risk.

The health impacts of pollution are estimated by calculating the relationship between population exposure to outdoor pollution and different health outcomes, while at the same time controlling for factors such as age, prior health status and weather conditions. It is known that almost all pollutants are correlated with each other in their health impacts.

This study therefore follows Künzli et al[1] (the Künzli study) and Fisher et al[2] in using PM_{10} as a 'surrogate pollutant'. Surrogate pollutants aim to capture the effects of all other pollutants—not solely the one being measured. It is possible that this approach may be overly conservative. There are indications, but currently no conclusive evidence, that ozone is not significantly correlated with particulates.

The study also adopts the Künzli study's estimates of the long-term health impact of pollution, which is typically larger than the short-term or immediate impact. The estimates in the Künzli study are based on meta-analysis of cohort studies undertaken in the United States of America (United States). Most Australian epidemiological studies to date have been based on short-term time series data only and have not had the benefit of cohort data. Australian studies, however, have been used to confirm that differences between conditions in Australia, America and Europe—in weather, traffic density, demographic characteristics and population health status—do not affect the general applicability of the estimated exposure–response relationships.

MOTOR VEHICLE POLLUTION AND ITS HEALTH IMPACT COSTS

Estimating the economic cost of these health effects is dependent on estimates of loss of quality of life due to ill health—the morbidity effect—and the mortality effect. The mortality effect is the extent of premature death due to air pollution. Studies indicate that the life expectancy lost due to premature mortality can range from a few months to 10 years.

This study estimates that in 2000 motor vehicle pollution accounted for between 900 and 4500 morbidity cases—cardio-vascular disease, respiratory disease, and bronchitis—and for between 900 and 2000 early deaths.[3] More than 85 per cent of these early deaths would have occurred in the capital cities, where over 80 per cent of Australians live. The ranges reflect the uncertainty bound—or confidence interval—surrounding the specific epidemiology estimates of health effects found in the Künzli study.

The economic cost of this premature mortality was between $1.1 billion and $2.6 billion (central estimate $1.8 billion). In addition, the estimated economic cost of morbidity was between $0.4 billion to $1.2 billion (central estimate $0.8 billion).

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1 Künzli N, Kaiser R, Medina S, Studnicka M, Chanel O, Filliger P, Herry M, Horak F Jr, Puybonnieux-Texier V, Quenel P, Schneider J, Seethaler R, Vergnaud JC, Sommer H 2000, 'Public health impact of outdoor and traffic-related air pollution: a European assessment', *The Lancet*, vol. 356

2 Fisher GW, Rolfe KA, Kjellstrom T, Woodward A, Hales S, Sturman AP, Kingham S, Petersen J, Shrestha R, King D 2002, *Health effects due to motor vehicle air pollution in New Zealand, a report submitted to the Ministry of Transport*, Wellington, New Zealand.

3 Early deaths are estimated using all cause mortality data, other than external causes.

The combined economic cost of motor vehicle-related mortality and morbidity was between $1.6 billion and $3.8 billion (central estimate $2.7 billion).

In deriving these health cost estimates, the value of a statistical life has been assumed to be $1.3 million—a discount of approximately 30 per cent on the value used to estimate rail crash costs.[4] The morbidity estimates are based on estimates of equivalent years of healthy life lost due to disability. They are informed by Australian estimates of the quality of life impact of bronchitis, respiratory illnesses and cardiovascular conditions. The assumed value per healthy year of life lost due to disability is $50 000.

HEALTH IMPACT COSTS AND AIR POLLUTION TRENDS

During the 1990s there were dramatic reductions in lead emissions for motor vehicles and significant reductions in other pollutants such as volatile organic compounds and carbon monoxide. These reductions are attributable to emissions control technology—notably catalytic converters—and new fuel standards.

Further measures to reduce pollution are currently under way, with more stringent new vehicle emissions standards being implemented between 2002 and 2007. Tighter standards for heavy vehicles and diesel-fuelled light vehicles will also reduce particulate emissions from new vehicles.

Looking forward, the Bureau has projected that motor vehicle emissions of nitrogen oxides, carbon monoxide and sulfur dioxide will decline significantly by 2020. It expects that PM_{10} emissions will be stable. These projections take account of changes to new vehicle emission standards and fuel standards, and a projected 46 per cent increase in total fleet vehicle kilometres travelled.[5]

These developments suggest that the health effects—and economic costs—of transport are likely to reduce to some extent over time. However, this may be offset in part by an ageing population.

SENSITIVITY TESTING OF THE ECONOMIC COST ESTIMATES

The total cost estimate of $2.7 billion (central value) varies substantially with changes to key assumptions.

Due to current data limitations, there is uncertainty about the proportion of PM_{10} concentrations in different locations that is attributable to motor vehicles. If the motor vehicle share is increased to 45 per cent—from approximately 35

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4 Bureau of Transport and Regional Economics 2003c, *Rail accident costs in Australia, report 108*, BTRE, Canberra.

5 Bureau of Transport and Regional Economics 2003b, *Urban air pollution from motor vehicles: Australian trends to 2020*, BTRE, Canberra.

per cent—in capital cities and increased to 20 per cent elsewhere, total estimated costs increase to $3.7 billion (+ 37 per cent).

Conversely, if the motor vehicle share of PM_{10} is reduced to 20 per cent in capital cities and elsewhere kept at base values, then total estimated costs are reduced to $2.1 billion (– 23 per cent).

Generally, pollutants cause negligible effects at very low levels. However, it is unclear whether this is the case for particulates. If the minimum exposure for PM_{10} mortality effects was reduced from 5 μm^3 to zero then the total estimated costs would increase to $3.3 billion (+ 25 per cent).

ECONOMIC COSTS AND EXTERNAL COSTS

These estimates of economic cost should not be interpreted as an external cost that motorists are not currently paying for. This is a separate issue outside the scope of this study, and requires consideration of a range of factors. These factors include taxes and charges already borne by motorists and the extent to which costs imposed on the community vary by class of vehicle, location, time of day and other factors.

Rather, the purpose of the estimates is to provide a comparative point of reference when assessing the effect of specific initiatives and, where appropriate, to assist in refining policy priorities.

FUTURE DIRECTIONS

The health impact of pollution is an evolving area of scientific research. Future estimates of the economic consequences of the health impact of motor vehicle pollution may be affected by new research results with respect to:

- the health impact of very fine particles—PM_1 and $PM_{2.5}$
- threshold levels above which pollutants are damaging to human health
- measurement of indoor pollution and its contributory sources
- more robust estimates of particulate emissions from road dust sources
- more accurate measurement of the motor vehicle share of local air pollution
- the appropriateness of using surrogate pollutants to measure the combined impact of pollutants on health
- the extent of length of life loss that is associated with premature death from pollution causes.

Economic research into the community's willingness-to-pay to reduce the risk of pollution-related morbidity and mortality should also improve future estimates of the economic costs of these health effects.

CHAPTER 1 INTRODUCTION

There is an increasing recognition that we need to know more about the economic and social costs resulting from transport impacts if we are to develop effective strategies to address both social and economic efficiency objectives.

Several studies have been undertaken in recent years in Australia and elsewhere in the developed world to cost the health and environmental impacts of ambient concentrations of air pollution (Segal 1999; Beer 2002; Fisher et al 2002; Künzli et al 2000; Burgers et al 2002; and AEA Technology 2002).

These studies are largely in response to the mounting epidemiological evidence that exposure to ambient air pollutants can be harmful to humans (Morgan et al 1998a and 1998b; Pope et al 1992; Pope et al 1995a and 1995b). Epidemiological studies attempt to show statistically significant links between ambient air pollutants and the extent of life lost in the general population. Estimates of the economic costs of ambient air pollution have been derived using the results of these statistical analyses.

This research is an attempt to quantify the economic costs to Australia of the health effects of transport emissions of ambient air pollutants. It is part of the Bureau of Transport and Regional Economics' ongoing work on quantifying the economic costs associated with transport impacts. The Bureau's previous work concentrated on the economic cost of transport accidents (Bureau of Transport Economics 2000).

SCOPE

Human activity produces a wide range of air pollutants contributing to global warming—carbon dioxide, methane, and nitrogen monoxide; acid rain—ammonia and sulfur dioxide; and local or ambient air pollutants.

This study focuses on major ambient air pollutants, the local effects of which have been statistically linked to adverse human health impacts.

The ambient air pollutants of most concern in Australia are listed in the 1998 Ambient Air Quality National Environmental Protection Measure (Ambient Air Quality NEPM). They are:

- carbon monoxide
- nitrogen oxides

- volatile organic compounds
- lead
- sulfur dioxide
- particulate matter of less than 10 microns—PM_{10}.

Nitrogen oxides and volatile organic compounds—also commonly known as hydrocarbons—are precursors to ozone.

The estimates in this study relate solely to outdoor ambient air pollution and do not consider indoor air quality. Despite the amount of time that most people spend indoors, relatively little research has been done on the quality of indoor air. Indoor air pollution is a major area of ongoing research.

Acid rain is produced when air pollutants react with water to form weak acids which then fall as rain. Like ambient air pollution, acid rain is localised. However, acid rain is not considered here as the principal impact is environmental—it includes damage to trees, plants, waterlife, and degradation of drinking water supplies.

Greenhouse gas emissions may ultimately have consequences for human health—for example, from higher average ambient temperatures. However, these are also outside the scope of this study because there are no direct links between local greenhouse gas emissions and local public health.

This report focuses on the economic costs of transport-sourced ambient air pollution, although transport is only one of many anthropomorphic sources. Transport sources in urban airsheds include motor vehicles, aircraft, shipping and boating, rail, paved and unpaved roads (dust), and related industrial activity.

In defining the transport share of ambient air pollution, the Bureau has limited the estimates to motor vehicle sources. This is due to current data limitations for other transport sources.

APPROACH

This study uses epidemiology-based exposure–response functions to estimate the number of health cases—mortality and morbidity—attributable to ambient air pollution. The Bureau has applied a BTRE-refined human-capital approach (Bureau of Transport Economics 2000) to derive the economic costs of transport-sourced ambient air pollution.

Given the uncertainties in both modelling and attributing the health costs of air pollution, the Bureau has taken an 'at least' approach in selecting data and parameters. This approach provides some degree of assurance that the health costs have not been overestimated.

It is important to note that the study estimates the economic cost of the health impact of motor vehicle pollution—not the external cost of motor vehicle

pollution. Externalities refer to interactions among economic agents that are not adequately reflected in markets. Externalities can be addressed by a range of policy measures—including taxation and regulation. Motor vehicle pollution in Australia is regulated through vehicle emission and fuel standards, under the Australian Design Rules. The cost of these regulations is reflected in the cost of purchasing and operating vehicles. This study does not assess these costs. Therefore, the Bureau is not in a position to determine whether the estimated cost of the health impact of pollution is external, or whether it is fully or partly internalised—for example, through taxes and charges that motorists pay to operate vehicles.

REPORT STRUCTURE

This report comprises six chapters—including this one.

Chapter 2 discusses the issues of air quality and associated health effects, and trends in air pollution.

Chapter 3 discusses the transport sources of ambient air pollutants, the transport share of major ambient air pollutants emissions in capital city airsheds, and the Bureau of Transport and Regional Economics's projections to 2020 of motor vehicle emissions.

Chapter 4 examines, and presents an overview of, the current literature on air quality and health.

Chapter 5 outlines the analytical approach used for the assessment and chapter 6 contains the calculations and conclusions.

CHAPTER 2 AIR QUALITY IN AUSTRALIA

This chapter focuses on outdoor air quality in Australia with reference to the capital cities. It presents historical trend analysis of major air pollutant concentration levels in selected cities. The final section discusses indoor air quality in Australia.

Air quality became a prominent global issue in the 1990s when community concerns about human health increased. This increase was primarily due to the emergence of a large body of epidemiological research showing adverse health effects of air pollution. However, researchers in Europe had noticed adverse health affects from air pollution as far back as the 1920s—for example, the Meuse Valley episode in Belgium in 1930. Most notable among these studies was the London episode of 1952, where the originally estimated death toll—since increased—was 4000. Based on these research findings, environmental regulatory authorities in Europe started implementing stringent air quality measures in the early 1960s. This resulted in a reduction of air pollution levels.

The most pronounced effect was observed for sulfur dioxide. Total emissions were reduced by about 50 per cent in the European region between 1980 and 1995. The reduction in nitrogen oxide emissions was smaller and was observed only after 1990. Between 1990 and 1995 total emissions were reduced by approximately 15 per cent (European Environment Agency 1998).

The mandatory use of unleaded petrol has seen a remarkable reduction of atmospheric lead content in Europe (World Health Organisation (WHO) 1999b) and in Australia (Manins et al 2001).

Trends in concentrations of other pollutants in urban air—such as nitrogen dioxide or particulate matter—are less clear. International organisations still consider these pollutants a risk to human health (European Environment Agency 2004).

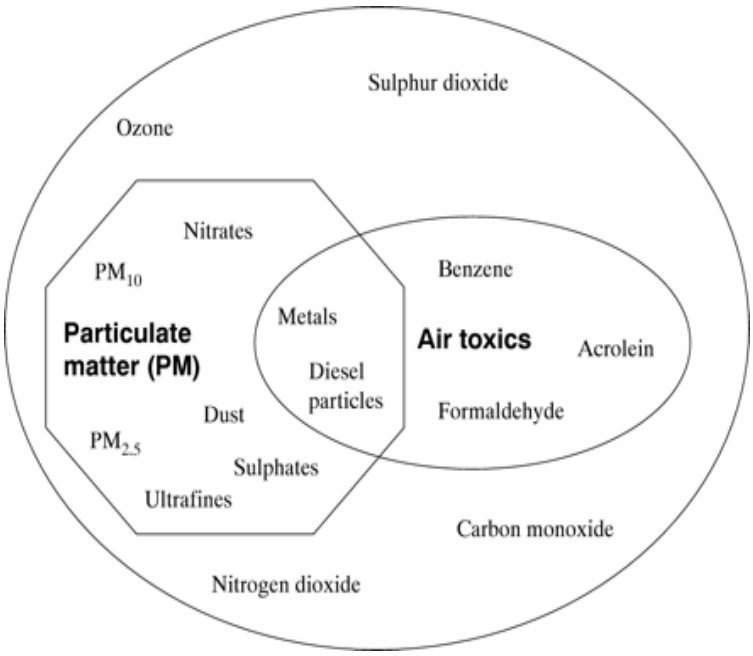
Air pollutant levels in Australian urban areas are not generally considered to be high relative to other world cities—see figures 2.1 and 2.2. The 2001 state of the environment them report states that that there is little evidence in the capital cities of Australia of air pollution problems arising from sulfur dioxide, nitrogen dioxide or lead (Manins et al 2001). However, it suggests that monitoring of these and other pollutants continues because of the strong epidemiological evidence linking exposure to particles and mortality.

Further, it is still necessary to research the extent of pollution, and to quantify the economic costs of health effects due to existing air pollution levels in Australian cities. Australian researchers have only focused on this complex issue in the 1990s and significant work is currently in progress.

AIR POLLUTANTS

Air pollutants are usually classified into suspended particulate matter—dusts, fumes, mists, smokes; gaseous pollutants—gases and vapours; and odours. Figure 2.1 represents the mix of major ambient air pollutants.

FIGURE 2.1 MAJOR POLLUTANTS IN THE AIR POLLUTION MIX



Source Grigg 2002 (adapted from Health Effects Institute)

Suspended particulate matter in the air can be categorised into:

- total suspended particles
- particles of less than 10 microns—PM_{10}
- particles of less than 2.5 microns—PM2.5.

Ten microns (micrometres, μm) are about one-tenth the width of a human hair. Particles of less than 10 microns (PM_{10}) correspond to the inhalable fraction of suspended particulate matter, with $PM_{2.5}$ the sub-fraction most likely to penetrate deepest into the airways (Grigg 2002).

Types of suspended matter include:

- diesel exhaust particles, fly-ash, mineral dusts—for example, coal, asbestos, limestone, cement
- metal dusts and fumes—for example, zinc, copper, iron, lead
- acid mists—for sulfuric acid
- fluoride particles
- paint pigments
- pesticide mists
- carbon black
- oil smoke.

Suspended particulate matter also includes secondary pollutants generated by chemical reactions in the atmosphere. For example, sulphate and nitrate aerosols formed from sulfur dioxide and nitrogen oxides.

Suspended particulate pollutants exacerbate respiratory diseases and may cause cancers, as well as corrosion and destruction to plant life. They can also constitute a nuisance—for example, the accumulation of dirt; interfere with sunlight—for example, light scattering from smog and haze; and the particle surface can act as a catalyst for the reaction of adsorbed chemicals.

Gaseous pollutants include:

- sulfur compounds—for example, sulfur dioxide and sulfur trioxide
- carbon monoxide
- nitrogen compounds—for example, nitric oxide, nitrogen dioxide and ammonia
- organic compounds—for example, volatile organic compounds, polycyclic aromatic hydrocarbons and halogen derivatives, aldehydes
- halogen compounds—for example, halogen fluorine and halogen chlorine
- odorous substances.

Volatile organic compounds are released from burning fuel—for example, petrol, oil, wood, coal and natural gas—evaporation of solvents, paints, glues and other products used at work or at home. They include benzene, toluene, methylene chloride and methyl chloroform. Some volatile organic compounds have been shown to cause serious health problems including cancer.

Nitrogen oxides and volatile organic compounds—also known as hydrocarbons—react with sunlight to eventually form ozone, which, when concentrated, provides an estimate of photochemical smog. Ozone is a form of molecular oxygen that consists of three oxygen atoms linked together. Ozone in the upper atmosphere—the ozone layer—occurs naturally and protects life on earth by filtering out ultraviolet radiation from the sun. But ozone at ground level is a noxious pollutant. The smog that occurs in Australian cities is

photochemical in origin and the concentrations of ozone provide an estimate of photochemical smog (Manins et al 2001).

Specific chemicals cause some odours—for example, hydrogen sulphide, carbon disulphide and mercaptans. However, some sources of odour are difficult to define chemically. Offensive odours are a major source of complaints to environment protection agencies. Even at low concentrations, odours may be offensive to humans and cause some people to feel unwell.

AIR POLLUTANTS AND HUMAN HEALTH

Ambient air pollutants pose different levels of risk to humans and the environment. Research in the 1990s revealed that human health may be affected by exposure to much lower levels of some common air pollutants than previously believed (WHO 1999b).

Evidence is emerging also that long-term exposure to low concentrations of particulate matter in air is associated with mortality and other chronic effects, such as increased rates of bronchitis and reduced lung function (WHO 2000b). For pollutants like sulfur dioxide and nitrogen dioxide, there is no evidence suggesting any threshold levels for health effects. However, they are unlikely to affect people's health at very low levels. Recent research studied the effect of long-term air pollution exposure in urban areas on lung cancer. The results suggested that carcinogenic chemicals in the smallest air particles and carcinogenic gases—like benzene and benzopyrene—could be the possible causal agents (Kjellstrom et al 2002). The health effects of air pollutants are discussed in detail in chapter 4.

How these pollutants affect people's health depends on the pollutants' characteristics and the level of people's exposure to them. The specific characteristics of the pollutant, and the circumstances in which exposure occurs, are crucial for assessing the health effects of the pollutants. The National Environment Protection Council Technical Advisory Panel—formed to recommend substances for inclusion in the National Pollutant Inventory[6]—assessed the risks to human health and the environment from exposure to a

[6] The National Pollutant Inventory is an internet database that provides information on the type and amount of pollution emitted to the air, land and water for a wide range of major airsheds across Australia. This inventory gives information on the pollutants emitted and the magnitude of emissions from facilities like manufacturing sites and from other sources such as households and transport. Facilities estimate their own emissions and government agencies estimate aggregated emissions from households and other sources. The national pollutant inventory provides the best available source of Australia-wide emissions data for major air pollutants. However, inventory data does not provide a direct link between emissions and people's *exposure* to air pollutants (Australian Government. Department of the Environment and Heritage 2003).

substance listed in the inventory. The Panel's risk assessments for the major air pollutants are summarised in table 2.1.

TABLE 2.1 NATIONAL POLLUTANT INVENTORY RISK RANKING OF SELECTED AIR POLLUTANTS

Pollutant	*Health hazard*	*Environmental hazard*	*Combined rank*	*National Pollution Inventory rank*[1]
Nitrogen oxides—N_2O NO NO_2	1.5	3.0	4.5	1
Carbon monoxide—CO	2.0	0.8	2.8	3
Sulfur dioxide—SO_2	1.5	1.3	2.8	3
PM_{10}	1.2	1.3	2.5	7
Lead and compounds	1.7	1.5	3.2	11
Non-methane volatile organic compounds				
Xylene	1.3	1.0	2.3	9
Benzene	2.3	1.0	3.3	14
Toluene	1.3	1.3	2.6	33

Notes The approach used to assess pollutants results in some substances receiving the same National Pollution Inventory rank. This ranking also considers human exposure to the pollutant. Excluded from the National Environment Protection Council list of approximately 400 substances were substances that had been banned or scheduled for phase-out, agriculture and veterinary chemicals, and those substances where other reporting was in place because of their ozone depleting or greenhouse effects.

Source National Pollution Inventory Technical Advisory Panel (1999); National Pollution Inventory substance profiles (Environment Australia undated).

CURRENT AIR QUALITY IN AUSTRALIA

Background

Australia is a highly urbanised country with over 80 per cent of the population living in cities. Each city has expanded rapidly in the past 30 years and relies heavily on private rather than public transport. As well as benefits, the continuing growth of cities has brought costs such as congestion and air pollution. Air pollution causes damage to health, degradation of the built and natural environment, and loss of amenities.

According to the Australian Academy of Technological Sciences and Engineering (1997a), air quality in all major Australian cities generally meets standards established to protect human health. However, relatively high concentrations of some pollutants are occasionally experienced in the larger cities. Consequently, it is a national priority to monitor the state of air quality in the major cities, where photochemical smog and airborne particles are the most serious urban air quality hazards.

Institutional arrangements

In Australia, the Ambient Air Quality National Environment Protection Measure (Ambient Air Quality NEPM), implemented in June 1998, sets standards—see table 2.2—for six air pollutants:

- carbon monoxide
- ozone
- nitrogen dioxide
- sulfur dioxide
- lead (Pb)
- particulate matter less than 10 microns (PM_{10}).

Also in 1998, the National Environment Protection Council established a goal—the national achievement of the standards by 2008—and a reporting protocol to measure progress towards the goal. States and territories are required to monitor their air quality in accordance with this protocol.

TABLE 2.2 NATIONAL AMBIENT AIR QUALITY STANDARDS

Pollutant	*Averaging Period*	*Maximum Concentration*	*Goal within 10 years (Max allowable excedences)*
Carbon monoxide	8 hours	9.0 ppm	1 day a year
Nitrogen dioxide	1 hour	0.12 ppm	1 day a year
	1 year	0.03 ppm	None
Photochemical oxidant (as ozone)	1 hour	0.10 ppm	1 day a year
	4 hours	0.08 ppm	1 day a year
Sulfur dioxide	1 hour	0.20 ppm	1 day a year
	1 day	0.08 ppm	1 day a year
	1 year	0.02 ppm	None
Lead	1 year	0.50 $\mu g/m^3$	None
Particles as PM_{10}	1 day	50 $\mu g/m^3$	5 days a year

Source Manins et al 2001

In addition, each state in Australia has made substantial efforts to analyse pollution sources and trends, and to formulate policies to manage air quality at acceptable levels. For example, in 1996 the New South Wales Environment Protection Authority published the *Metropolitan Air Quality Study* (*MAQS*) and *Developing an Air Quality Management Plan for Sydney, the Illawarra and the Lower Hunter: a New South Wales Government Green Paper*. In 1999 Queensland published the *South-East Queensland Regional Air Quality Strategy*.

Particulate matter of less than 2.5 microns was not included in the standards set by the 1998 Ambient Air Quality NEPM. Following the increasing evidence on

the health effects of $PM_{2.5}$, the Environment Protection and Heritage Council approved advisory reporting standards for $PM_{2.5}$ on 23 May 2003. These standards support the national collection of data on fine particles for the 2005 review of the Ambient Air Quality NEPM (Environment Protection and Heritage Council 2003).

Trends in concentrations of major pollutants in Australian cities

The purpose of long-term monitoring is to assess whether air quality is improving or deteriorating. Air quality monitoring by state and territory environment protection authorities began in the late 1970s. This section examines trends in the 1980s and 1990s.

Air quality trends in the last 25 years have been affected by many initiatives to reduce air pollutant emissions. These include:

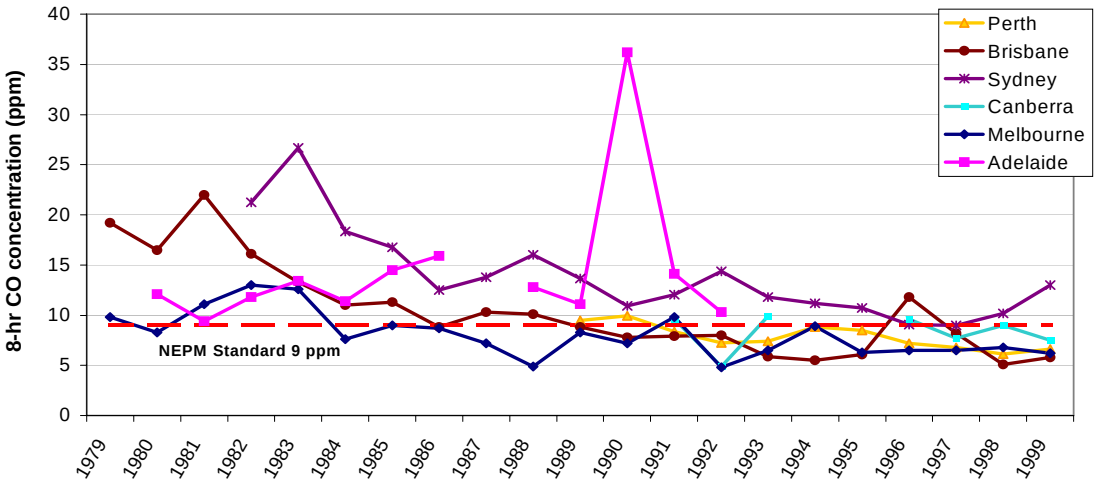
- reducing the sulfur content and eliminating lead from transport fuels
- the fitting of control equipment in older industrial facilities
- placing restrictions on backyard burning in some local authority areas.

Against that, climate change can have a serious impact on the extent of the vegetation burning and the incidence of bushfires (Selvey and Sheridan 2002; McMichael et al 2002; Cheney 1995). These can cause variations in pollutant concentration levels in the air from year–to–year. Seasonal variations in the concentration levels of air pollutants are also an important influencing factor. However, they are not discussed in this section due to lack of data.

Figures 2.2 to 2.8 depict trends in annual average concentration of major air pollutants in selected Australian capital cities during the 1980s and 1990s.

Figure 2.2 shows that eight-hour average carbon monoxide concentrations reduced substantially in most cities. Except in Sydney and Adelaide, where they remained below, or a little above, the Ambient Air Quality NEPM standard in the 1990s. The increased concentration in 1999 for Sydney might have been caused by bushfires.

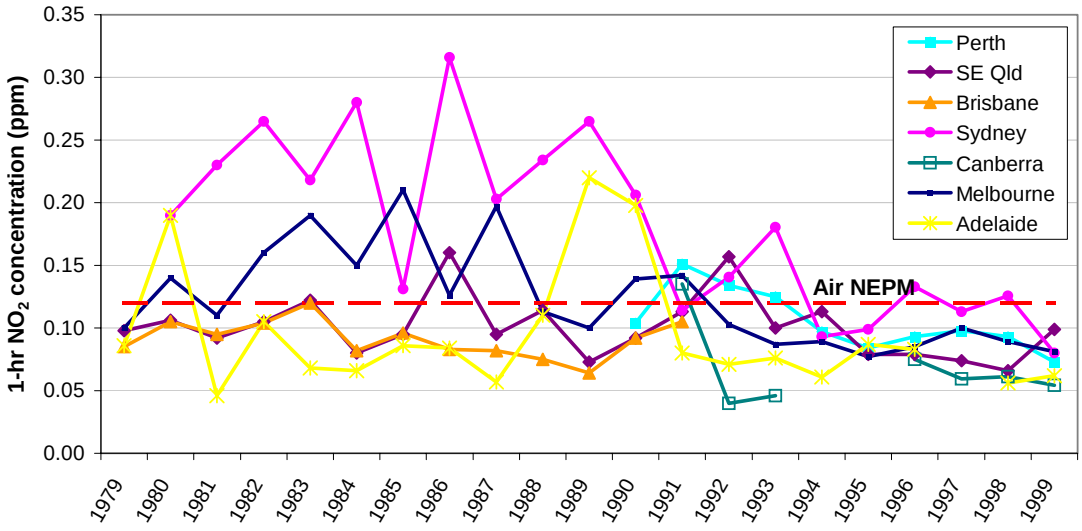
FIGURE 2.2 HIGHEST EIGHT-HOUR AVERAGED CARBON MONOXIDE CONCENTRATIONS FOR SELECTED CITIES



Source State and territory environmental authorities, Manins et al 2001

Figure 2.3 depicts the long-term trend in one-hour average concentration of nitrogen dioxide in selected capital cities from 1979 to 1999. In the 1980s, there was high variability in concentrations in Sydney and Melbourne. Brisbane was the only city where the nitrogen dioxide concentration level did not exceed the Ambient Air Quality NEPM standard. The concentration decreased substantially for Sydney and Melbourne during the 1990s. After 1994, it remained below the Ambient Air Quality NEPM standard of 0.12 parts per million for all cities except Sydney.

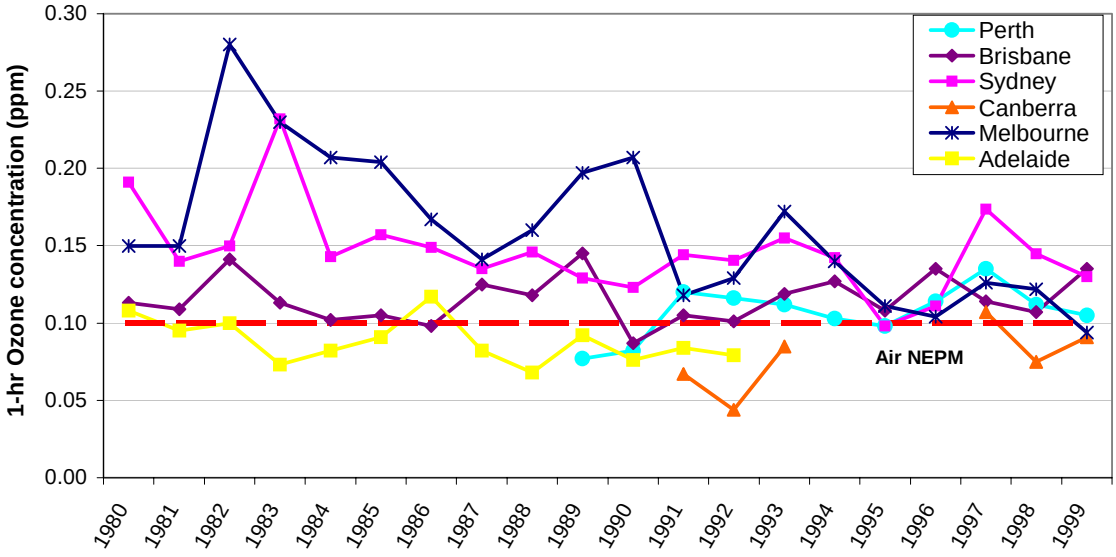
FIGURE 2.3 HIGHEST ONE-HOUR NITROGEN DIOXIDE CONCENTRATIONS FOR SELECTED CITIES



Source State and territory environmental authorities, Manins et al 2001

Figure 2.4 presents the trend in one-hour average concentration for ozone from 1980 to 1999. Sydney and Melbourne showed high variability in the ozone concentration from 1980 to 1996, although the concentration has declined over the period for these two cities. Canberra and Adelaide were close to the level of the Ambient Air Quality NEPM standard for ozone for most of the observed period. However, for the maximum amount averaged over four hours, there has not been much decline. This possibly reflects greater traffic activity outside the peak periods and hence, the level of photochemical smog has remained constant.

FIGURE 2.4 MAXIMUM ONE-HOUR AVERAGE OZONE CONCENTRATIONS IN SELECTED CITIES, 1980 TO 1999



Source Commonwealth, state and territory environment authorities, Manins et al 2001

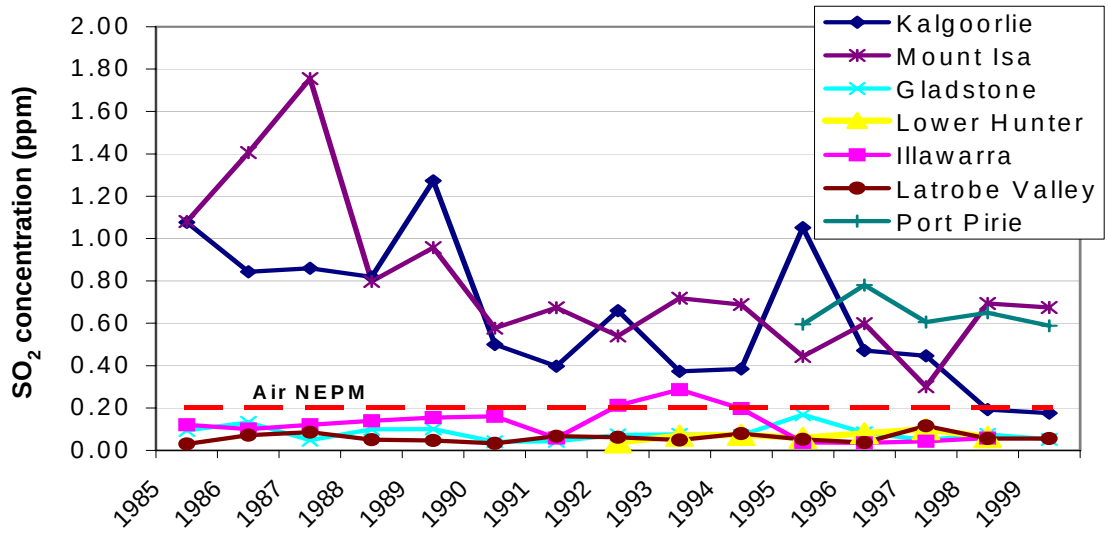
Figures 2.5 and 2.6 illustrate trends in average concentrations of sulfur dioxide and airborne lead respectively from 1985 to 1999.

In urban Australia, sulfur dioxide levels are not normally monitored in the major cities where emissions are usually negligible. Ambient sulfur dioxide concentrations rarely exceed Ambient Air Quality NEPM values except near petroleum refineries or petrochemical or chemical factories (Manins et al 2001).

In regional areas sulfur dioxide concentration levels are monitored where there is significant industrial activity. Figure 2.5 indicates that although sulfur dioxide emissions declined significantly in the 1990s, they remained above the Ambient Air Quality NEPM standard in Kalgoorlie (Western Australia), Mount Isa (Queensland) and Port Pirie (South Australia). In contrast, concentrations conformed to the Ambient Air Quality NEPM standard in the Lower Hunter

and the Illawara (New South Wales) and the La Trobe Valley (Victoria) throughout most of the observation period.

FIGURE 2.5 HIGHEST ONE-HOUR AVERAGE SULFUR DIOXIDE CONCENTRATIONS IN REGIONAL AREAS, 1985 TO 1999



Note Lower Hunter from 1992; Port Pirie from July 1995.

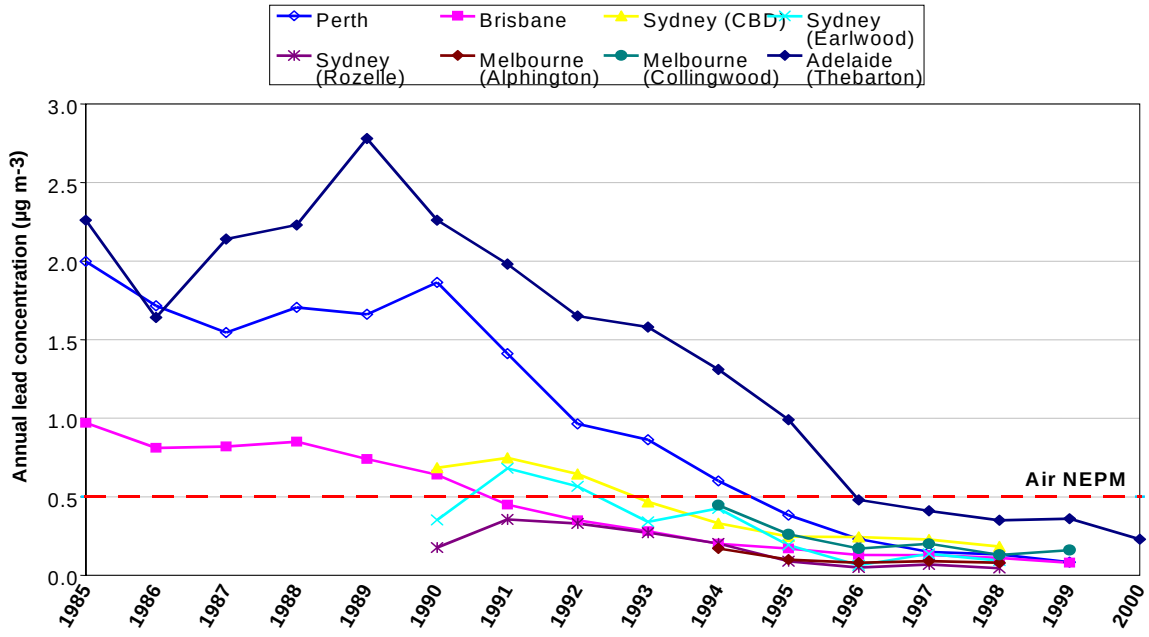
Source Data from State environment protection authorities, Western Australian Department of Environment Protection, Manins et al 2001

Since 1986, recorded lead levels in ambient air have dropped to the point that lead in air is no longer an issue in metropolitan Australia (Manins et al 2001).

The phase-out of leaded petrol has resulted in a sustained decline in ambient lead levels in capital cities (see figure 2.6). Petrol engine cars sold in Australia after 1985 were required to have catalytic converters and run on unleaded petrol. Leaded fuel was phased out on 1 January 2002.

In Adelaide lead levels have fallen from 3.7 µg/m^3 in 1982 to 0.02 µg/m^3, prompting the South Australian Environmental Protection Agency to cease airborne lead monitoring from the end of June 2003 (Environmental Protection Agency South Australia 2003).

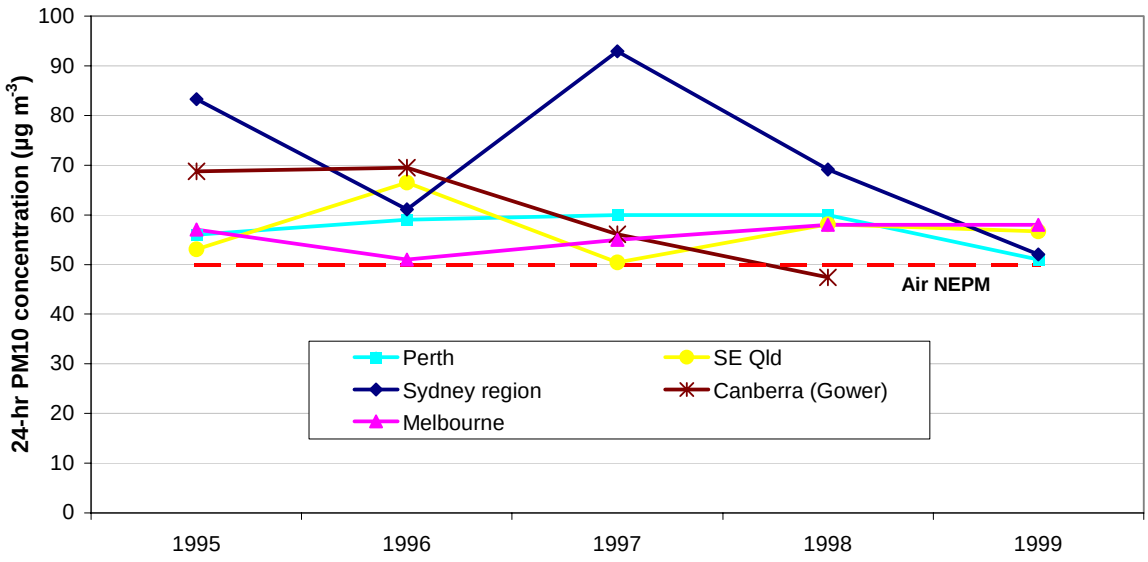
FIGURE 2.6 TRENDS IN AIRBORNE LEAD LEVELS, 1985 TO 2000



Source State environmental authorities, Manins et al 2001

Figure 2.7 depicts the maximum 24-hour concentration level in PM_{10} in selected Australian cities. It suggests that Melbourne registered an increase in PM_{10} concentrations from 1995 to 1999. In the same period, Sydney, Perth and Canberra showed reduced concentrations.

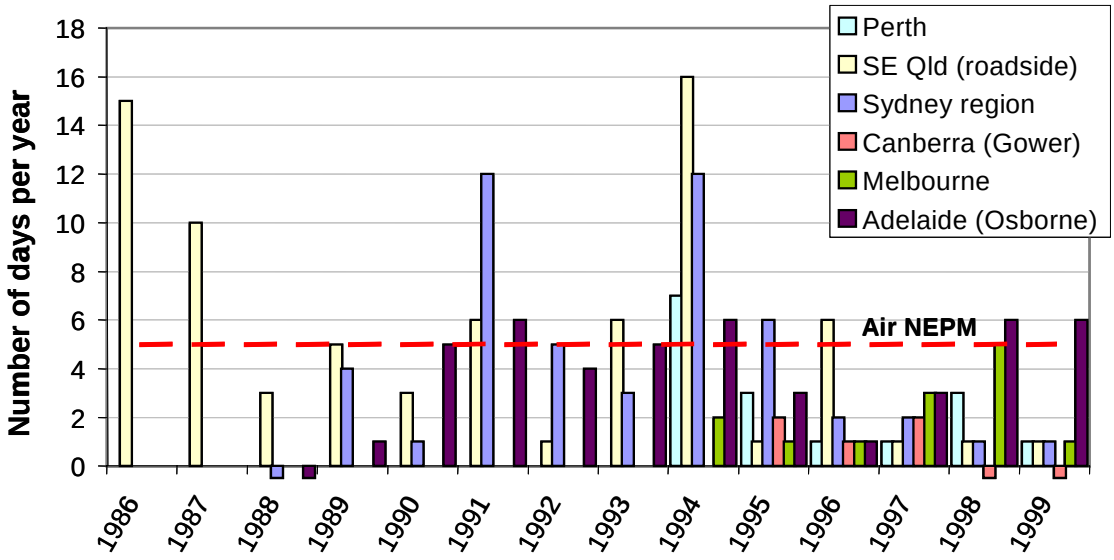
FIGURE 2.7 MAXIMUM 24-HOUR PM_{10} CONCENTRATIONS FOR SELECTED CITIES



Source State and territory environmental authorities, Manins et al 2001

Figure 2.8 displays the number of days annually from 1985–86 to 1999 when 24-hour average PM_{10} concentrations exceeded the Ambient Air Quality NEPM standard. Except for 1994, 1991, 1987 and 1986, most cities met, or were close to meeting, the Ambient Air Quality NEPM standard.

FIGURE 2.8 NUMBER OF DAYS ANNUALLY WHEN 24 HOUR PM_{10} CONCENTRATIONS EXCEED AIR NEPM



Notes PM10 24-hour standard 50 µg/m3, 10-year goal maximum—PM10 not to exceed the Ambient Air Quality NEPM more than 5 times per year. Negative values indicate no occasions in the period where the standard was exceeded. Zero values indicate no data.

Source State and territory environmental authorities, Manins et al 2001

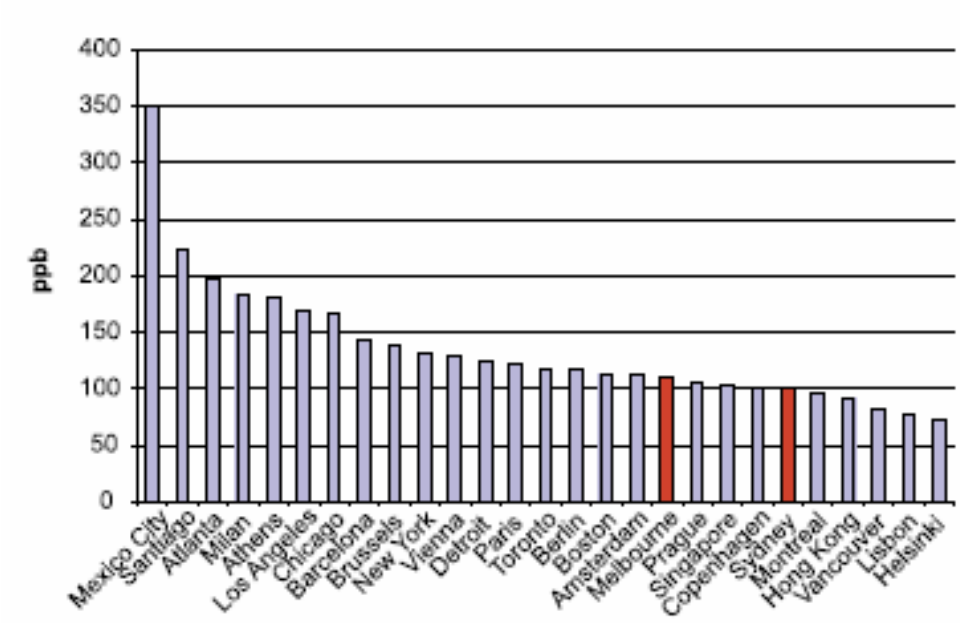
Instances of higher PM_{10} levels in the major urban areas can occur during bushfires or controlled burning of excess vegetation on the outskirts of urban areas. Fires are the likely cause of the peaks shown in figures 2.7 and 2.8 for Sydney and south-east Queensland.

International comparisons

Australia has relatively limited air pollution problems compared with cities such as Los Angeles, Mexico City and Athens. This is partially attributable to Australia having fewer sources of pollution—because of less heavy industrialisation. In addition, local winds tend to rapidly disperse pollution over the cities. Other factors in Australia's favour are its relatively small population, it is surrounded by oceans and it does not receive a high volume of polluted air from neighbouring countries. Consequently, Australian cities are less densely populated and have generally lower concentrations of ambient air pollution.

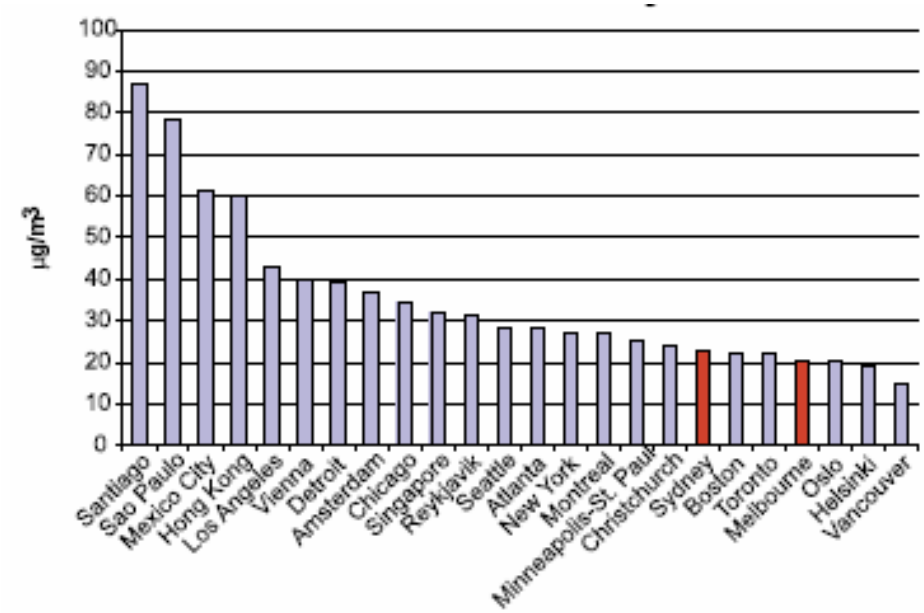
However, Sydney, Melbourne and other large Australian cities experience days of high concentration of major air pollutants each year. This increased air pollution level is usually season specific. Summer and autumn tend to be the worst times of the year with increased incidences of hospitalisation and lost working days.

FIGURE 2.9 CONCENTRATIONS OF OZONE (MEASURED FOR MAXIMUM ONE-HOUR OZONE CONCENTRATIONS) IN SELECTED CITIES IN 1995



Source Mannins et al 2001, p.101 Figure 122

FIGURE 2.10 CONCENTRATIONS OF PM_{10} (MEASURED AS ANNUAL AVERAGE OF PM10 CONCENTRATIONS) IN SELECTED CITIES IN 1995



Source Mannins et al 2001, p.101 Figure 122

Figures 2.9 and 2.10 present the annual average concentration level of two major air pollutants, ozone and particulate matter (PM_{10}), in different cities in the world. Out of 27 cities considered, Melbourne and Sydney rank respectively in 18th and 22nd position for maximum one-hour ozone concentration. For annual average PM_{10} concentration, Sydney is ranked 18th and Melbourne is ranked 21st, out of 24 cities studied internationally.

The nature of air pollution in Australia is often quite different from that in other countries. Sulfur dioxide, for example, accounts for a far lower share of emissions in Australia than in Europe. In Australia, ambient $PM_{2.5}$ concentrations—especially in summer—compare quite favourably to other parts of the world. On a per capita basis, pollution rates in Australian cities are relatively high compared with cities overseas. However, from a health effects perspective, it is emissions per unit area that count, not emissions per capita.

The air pollutants of current concern in Australia are particles—PM_{10} and $PM_{2.5}$, ozone, nitrogen dioxide, benzene, air toxics and, to a lesser extent, carbon monoxide.

AIR QUALITY ISSUES

Photochemical smog

Photochemical smog is formed on days when the sun shines on air containing volatile organic compounds and nitrogen oxides. Motor vehicles, industry and bushfires are major sources of volatile organic compounds and nitrogen oxides.

Ozone concentrations provide an estimate of photochemical smog in cities (Manins et al 2001). The Ambient Air Quality NEPM states:

> In general, high levels of ozone are only a problem for major cities where emissions from concentrated urban activities can accumulate to high levels if the meteorology is favourable for pollution build up and for smog formation. Melbourne, Sydney, Perth, Brisbane and Adelaide, are the main Australian cities of sufficiently large size and favourable meteorology for significant ozone formation. Most rural areas and other cities have either populations which are too small and dispersed and/or meteorology that does not favour ozone production. (National Environment Protection Council 1998)

Particulate matter

The main concern in Australian cities is with the concentration of particulate matter (PM_{10}, $PM_{2.5}$). Research since 2000 from the United States reveals that daily mortality is linked both to PM_{10} and $PM_{2.5}$. The health effects of air pollutants are discussed in detail in chapter 4. Particle pollution is associated with significant health impacts, including exacerbation of asthma and an

increased risk of premature mortality—especially in the very young and the elderly.

Table 2.3 shows some recent sampled PM_{10} in Australian capital cites. The annual mean shows that all cities are significantly below the one-day national standard shown in table 2.2. The standard is daily samples, and there are some periods where sampled PM_{10} exceeds the one day Ambient Air Quality NEPM standard.

TABLE 2.3 MAXIMUM AND ANNUAL AVERAGE LEVELS OF PM_{10}

City	*Year*	*Maximum PM_{10} in $\mu g/m^3$*	*Annual average PM_{10} in $\mu g/m^3$*
Sydney–Earlwood	2001	130	21
Melbourne–Alphington	2001	72.6	18.9
Brisbane–Rocklea	2001	70.8	16.6
Adelaide–North Plympton	2001	43.8	19.3
Perth	2001	Na	20
Hobart	2001	71	16
Canberra–Monash	2001	70.6	19.2
Darwin	2000	54	14.9

Note Na - not available

Source Various environment protection agency web sites and unpublished data

Road transport

In Australia there is community concern that the increasing number of diesel vehicles in Australian cities may be increasing particle emissions. Diesel exhaust has been linked in numerous scientific studies to cancer, the exacerbation of asthma and other respiratory diseases (American Lung Association 2000). In August 1998, the State of California decided that there was enough evidence to list particulate matter in diesel exhaust as a toxic air contaminant. That is, a probable carcinogen requiring action to reduce public exposure and risk (California Air Resources Board 1998). In 2002, the United States Environment Protection Authority released its final report into the health effects of diesel exhaust. The reports' health assessment was that:

> … long-term (i.e., chronic) exposure to [diesel exhaust] is likely to pose a lung cancer hazard as well as damage the lung in other ways depending on exposure. The health assessment's conclusions are based on exposure to exhaust from diesel engines built prior to the mid-1990s. Short-term (i.e., acute) exposures can cause transient irritation and inflammatory symptoms, although the nature and extent of these symptoms are highly variable across the population. The assessment also states that evidence is emerging that diesel

> exhaust exacerbates existing allergies and asthma symptoms (United States Environment Protection Authority 2002, p. xiv-xv).

The report states that it is the particulate fraction of diesel exhaust that is the key to its understanding of the health issues and that future cleaner diesel engines will reduce particulate emissions by 90 per cent from current levels. The USEPA believes it will need to re-evaluate the general applicability of its conclusions as these cleaner engines replace those in current use (United States Environment Protection Authority 2002, p. xv).

Most trucks, buses and other large commercial vehicles use diesel engines. Diesel engines produce a higher proportion of fine particles compared to petrol using passenger cars—although actual emissions depend on a vehicle's emission control technology. In addition, sports utility vehicles are an increasing proportion of the passenger vehicle fleet—this may be an issue to the extent that they are more likely than cars to use diesel fuel. However, new vehicle emissions standards, more stringent fuel standards and control technology in vehicles could reduce $PM_{2.5}$ emissions from the diesel fleet more than 85 per cent by 2009–10 (Bureau of Transport and Regional Economics 2003a). Transport and air pollution are discussed in chapter 3.

Emission control technology and changes in fuel standards have reduced motor vehicle air pollution emissions. But emissions of fine particles and volatile organic compounds from road vehicles remain the main concern for transport-related health risks. Health effects of air pollutants are discussed in chapter 4.

INDOOR AIR POLLUTION

Pollution levels are higher indoors than they are outside and people generally spend more time inside. Thus, most of a person's daily exposure to many air pollutants comes through inhaling indoor air.

Australians spend 90–96 per cent of their time indoors.[7] Despite the long periods people spend indoors, relatively little research has been done on the quality of air in homes, schools, recreational buildings, restaurants, public buildings and offices, or inside cars. In recent years, the United States Environmental Protection Agency and its Science Advisory Board have conducted comparative risk studies. These studies have consistently ranked indoor air pollution among the top five environmental risks to public health.

The most important compounds in indoor air environments include suspended particulate matter, sulfur dioxide, nitrogen oxides, carbon monoxide,

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[7] The Australian Bureau of Statistics' national time use surveys indicate that on average Australians spent about four per cent of their day, or one hour, outdoors and seven per cent —or 24 minutes—of their recreation time outdoors (ABS 1994, ABS 1996, ABS 1998). Thus, the quality of the indoor air environment is a very important issue of growing community significance (see *Part B: Indoor air quality*, Manins et al 2001).

photochemical oxidants (as ozone), and lead. Moreover, indoor sources may lead to an accumulation of some compounds rarely present in ambient air. However, because there is continuous air exchange between indoors and outdoors, most pollutants present in outdoor air are also found indoors. Indoor concentrations of air pollutants are influenced by:

- outdoor levels
- indoor sources
- the rate of exchange between indoor and outdoor air
- the characteristics and furnishings of buildings.

In developed countries, pollutant concentrations indoors are similar to outdoor concentrations, with the ratio of indoor to outdoor concentration ranging from 0.7 to 1.3. Concentrations of combustion products in indoor air can be substantially higher than outdoor concentrations when heating and cooking appliances are used. This is particularly true in developing countries where ovens and braziers are used with imperfect kitchen and stove designs.

Poor indoor air quality can result in significant adverse effects on our health and environment. Moreover, these effects carry a significant cost burden to the economy. The Commonwealth Scientific and Industrial Research Organisation estimates that the cost of poor indoor air quality in Australia may be as high as $12 billion per year (Brown 1998).

Australia's National Health and Medical Research Council (1996) has recommended interim national indoor air quality goals for common indoor air pollutants (table 2.4).

According to WHO Air Quality Guidelines (WHO 1999a), in five developed European countries formaldehyde concentrations in indoor air were reported to range from 9–70 mg/m^3. Higher values are occasionally encountered, especially in dwellings with urea-formaldehyde foam insulation (European Collaborative Action 1993).

Radon—a chemically inert gas—is an important background source of exposure to radiation. Being inert it does not combine with the atoms of its host material. Instead, it works its way through the tiny cracks and voids in the ground and into the atmosphere, where it can be inhaled in the air we breathe. The concentration of radon can be higher indoors as buildings have the effect of trapping radon within. At a natural threshold level, radon levels in the air range from 8 to 20 Bq/m^3[8] (Hocking and Joyner 1994; Langroo et al 1990; Solomon 1990). The Australian Radiation Protection and Nuclear Safety Agency has conducted a nationwide survey of radon in Australian homes. The results show that the average concentration of radon is about 12 Bq/m^3—less than in many

[8] Bq/m^3: Becquerel per cubic metre is a measure of the rate of radioactive decay present in air.

other countries and is not much higher than levels in outside air (Australian Radiation Protection and Nuclear Safety Agency, undated). In general, average indoor levels of radon are 20–70 Bq/m^3 in Europe (European Collaborative Action 1995), although they may be 10 times higher in certain areas.

TABLE 2.4 INTERIM NATIONAL INDOOR AIR QUALITY GOALS

Pollutant	*Goals for maximum permissible levels of pollutants in air*[a]		*Measurement criteria*	*Comments*
	µg/m^3	ppm		
Carbon monoxide	10 000	9	8-hour average not to be exceeded more than once a year	This is not a threshold limit value
Formaldehyde[b]	120	0.1	Not to be exceeded	For dwellings and schools
Lead	1.5	–	3-month average	
Ozone	210	0.10	Maximum hourly average not to be exceeded more than once a year	
	170	0.08	Four hour average	
Radon[b]	200 Bq/m^3	–	Annual mean	Action level
Sulfates	15	–	Annual mean	
Sulfur dioxide	700	0.25	10-minute average	Levels may not be low enough to protect the most sensitive individuals
	570	0.20	Hourly mean	
	60	0.02	Annual mean	
Particles	90	–	Annual mean	
Total volatile organic compounds	500	–	Hourly average	A single compound shall not contribute more than 50 per cent of the total

a. At 0° C and 101.3 kPa.

b. Final National Health and Medical Research Council goals. - = no goals set in those units

Source National Health and Medical Research Council 1996

Exposure to naturally occurring radiation materials is often increased by human activities—for example, burning coal, making and using fertilisers, oil and gas production.

Exposure to environmental tobacco smoke is an important factor in indoor air quality assessment, as tobacco smoke is linked to increased concentration of PM_{10}.

CHAPTER 3 TRANSPORT'S CONTRIBUTION TO AMBIENT AIR POLLUTION

This chapter focuses on the ambient air pollutants of most concern in Australia—those pollutants for which standards have been set in the Ambient Air Quality National Environment Protection Measure (Ambient Air Quality NEPM). This was discussed in chapter 2. These are:

- carbon monoxide
- particulate matter emissions—PM_{10} and $PM_{2.5}$
- nitrogen dioxide
- sulfur dioxide
- lead
- volatile organic compounds—including hydrocarbons.

This chapter uses National Pollution Inventory emissions data by source to estimate the transport share, whereas the emissions projections report the results of the Bureau of Transport and Regional Economics' own modelling of emissions by vehicle type.

Because National Pollution Inventory data is lacking for many regional areas, this chapter focuses on transport emissions in capital cities. Where inventory data are available it indicates that transport is generally a minor source of particulate matter emissions in regional areas.

The chapter looks first at how transport generates emissions and the major transport sources in urban areas. It then describes the major ambient pollutants in capital city airsheds using 2000–01 National Pollution Inventory data. The last section outlines the Bureau's own projections of motor vehicle emissions of major ambient air pollutants.

TRANSPORT AND AMBIENT AIR POLLUTION

The power to operate most vehicles, whether on road or rail, in the air or at sea, comes from burning fuel—for example, petrol, diesel, liquified petroleum gas—in an engine—see figure 3.1.

As the fuel vaporises into the hot air in the combustion chambers it starts to oxidise. As more fuel vaporizes and mixes with air, the number and rate of the

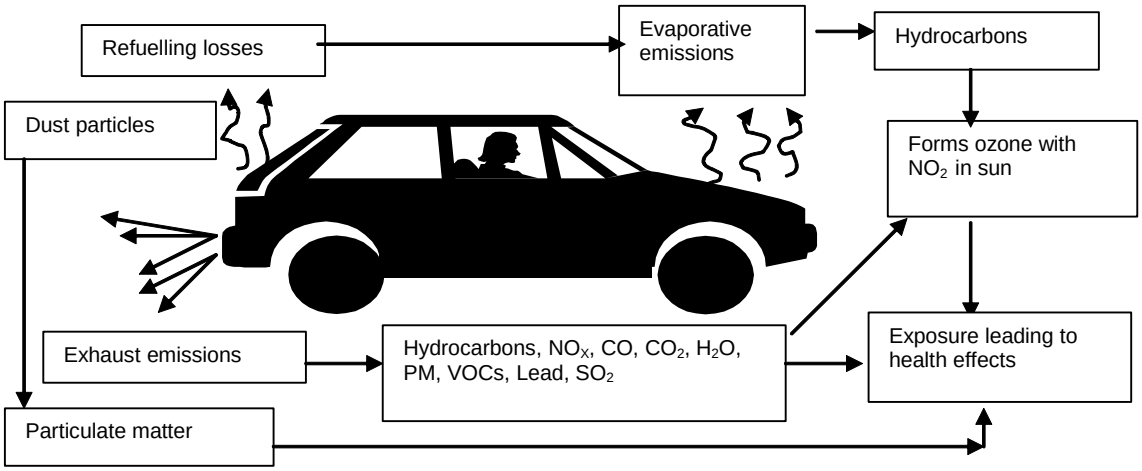
oxidation reactions increase until the end of the ignition delay period. At this point ignition occurs at many locations independently where fuel and air ratios are in the combustible range. Combustion then propagates very rapidly. This initial combustion is called the pre-mixed combustion phase.

At the end of the pre-mixed combustion phase, most of the fuel has yet to be injected or is still in a region that is too rich to burn. But injection continues and fuel continues to vaporise and mix with air—helped by the heat release and turbulence generated by the initial combustion. This is called the diffusion controlled, or mixing controlled, phase of combustion. Ideally, this phase consumes all of the remaining fuel.

Pollutants are formed during the combustion process. But there is no direct relationship between regulated pollutants produced by the engine, tailpipe emissions of these pollutants, and vehicle mass or size.

Non-combustion sources of ambient air pollution can include the evaporation of the fuel itself and vehicle movement can generate emissions of road dust and brake lining dust.

FIGURE 3.1 TRANSPORT EMISSIONS AND HUMAN HEALTH EFFECTS



COMBUSTION POLLUTANTS

In a complete combustion process, the hydrocarbons in fuel and oxygen in air are converted to water and carbon dioxide. In reality, the combustion process is not perfect and engines produce various local air pollutants. They include:

Nitrogen oxides

These are gaseous pollutants produced under very high pressure and temperature conditions in an engine. Nitrogen and oxygen in the air combine to form nitrous oxide, nitrogen dioxide and nitrogen oxide. Their production

increases with excess oxygen—which occurs under lean fuel conditions—and higher combustion temperatures.

Carbon monoxide

This gaseous pollutant is produced when there is not enough oxygen present in the combustion chamber. As a result the fuel is partially, rather than fully, oxidised. High levels of carbon monoxide can be caused by a too rich fuel mixture, incorrect idle speed, faulty air cleaner or positive crankcase ventilation (PCV) valve, incorrect fuel pressure or faulty carburettor/injection system.

Volatile organic compounds

These consist mainly of unburned hydrocarbons but may include partially burned compounds such as aldehydes. While most fuel is burned, a small fraction—typically one to five per cent—remains unburned. Production of volatile organic compounds can be many times higher than normal where a malfunction like a bad spark plug inhibits proper combustion.

Particulate matter of less than 10 microns

Particulate matter of less than 10 microns is a heterogenous blend of different size particles that can carry a range of toxic trace substances depending on the source. Combustion sources such as engines produce proportionally more of the finer $PM_{2.5}$ fraction than other sources of particulate matter.

While many pollutants are created during combustion, actual emissions from the vehicle's tailpipe depend on a wide range of factors. These include:

- how the vehicle is driven, its age and how well it is maintained.
- The pollution control technologies fitted to the vehicle. While pollutants are produced in the combustion process, emissions control technologies—such as catalytic converters and particle traps—can substantially limit tailpipe emissions of specific pollutants.
- The type of fuel. Different fuels produce different pollution mixes, or emission specifications. Petrol engines produce significant levels of hydrocarbons, carbon monoxide and nitrogen oxides. Diesel engines produce low levels of carbon monoxide and hydrocarbons compared with petrol engines. But they produce relatively high levels of nitrogen oxides and particulate matter—including diesel particles.[9] Liquified petroleum gas and compressed natural gas engines produce very low levels of particulate matter and negligible evaporative hydrocarbon emissions.

[9] These consist of a carbonaceous core onto which over 18 000 different compounds may be adsorbed (Grigg 2002).

- The amount of sulfur in fuel. Sulfur directly contributes to air pollution as some is converted to sulfate particles. Reducing sulfur decreases particulate matter production linearly in almost all engines. More importantly, reducing sulfur enables the adoption of better engine technologies and helps manufacturers meet stricter emissions standards. The European Programme on Emissions, Fuels and Engine Technologies (1995, pp. 11–12) found that reducing sulfur reduced exhaust emissions of hydrocarbons, carbon monoxide and nitrogen oxides generally linearly, by approximately eight to 10 per cent.

NON-COMBUSTION POLLUTANTS

Transport also produces non-combustion ambient air pollutants including evaporative emissions and particulate matter.

Evaporative emissions consist of volatile organic compounds and—from leaded fuel—small amounts of lead.[10] Emissions occur when fuel vapour escapes from the vehicle fuel, engine or exhaust systems, and during refuelling or fuel tanker loading and unloading.

Evaporative volatile organic compound emissions come mainly from petrol as diesel has a much lower vapour pressure. Fuel parameters such as Reid Vapour Pressure, distillation range, benzene and aromatic content may significantly affect evaporative emissions (Environment Australia 2000).

Particulate matter is generated by the suspension or re-suspension of loose material on the road surface when a vehicle travels over a road (Environment Australia 1999a). The main sources of road particulate matter are silt and soil. Particle size is therefore relatively coarse and typical trace pollutants in the PM_{10} fraction differ from combustion sources. Measured concentrations tend to be highest within two metres of the roadway and emissions do not typically disperse widely from the source.

TRANSPORT SOURCES IN URBAN AREAS

This section describes the major transport sources of ambient air pollution in urban areas. They include motor vehicles, aircraft, rail, shipping and road dust. Related industrial sources are also discussed briefly.

Motor vehicles

Motor vehicles are a major source of ambient air pollution in urban Australia. Traffic contributes more than 75 per cent of carbon monoxide emissions and

[10] Lead has been phased-out of motor vehicle fuel but is still used in some other transport fuels. These include Avgas which is used in piston engine planes.

most emissions of nitrogen oxides. It is a major contributor to volatile organic compounds and particulate matter emissions (Manins et al 2001).

Petrol and diesel engines produce a very high proportion of very fine particles ($PM_{2.5}$). Cadle et al (1999) found that 91 per cent of the petrol vehicle PM_{10} mass was smaller than 2.5 μm and at that least 97 per cent of the diesel and smoking vehicle PM_{10} mass was smaller than 2.5 μm. Cadle et al (1999) also found that the mass median diameter of particles was 0.12 μm for petrol vehicles and 0.18μm for diesel and smoking petrol vehicles.

According to Beer et al (2001 p. 18), diesel engines are presently a major source of fine particles. Diesel engines produce particles at about 20 times the rate of petrol engines.

For heavy vehicles powered by liquefied petroleum gas and compressed natural gas, nitrogen oxides emissions are lower than diesel while methane emissions are higher than low sulfur diesel.[11]

In Australia performance-based standards limit the amount of specific air pollutants that may be emitted by new vehicles. Each new vehicle model is required to comply with Australian Design Rules[12] before it is supplied to the Australian market. However, Australian Design Rules do not currently apply to petrol vehicle PM_{10} emissions. Manufacturers can choose from a wide range of technologies to meet Australian Design Rules and, for commercial reasons, may incorporate the minimum controls needed to meet the standard. As a result there is no direct relationship between regulated emissions and vehicle mass or size—see figure 3.2. It is therefore possible for a large six-cylinder car with highly effective emissions control technology to have lower emissions than a small four cylinder car with minimal emission controls.

The catalytic converter is a key technology that has already had a major effect on motor vehicle emissions of carbon monoxide, nitrogen oxides and volatile organic compounds (Australian Government Department of Transport and Regional Services 2001). However, about 60–80 per cent of all non-methane hydrocarbon emissions and carbon monoxide emissions[13] are generated in the first few minutes before the converter reaches normal operating temperatures (Laining 1994, quoted in Biel et al 1996).

New motor vehicles' emission rates are typically well below the regulated limits to allow for production variability and in-service deterioration. A vehicle's emissions increase over its operating life. This is due to:

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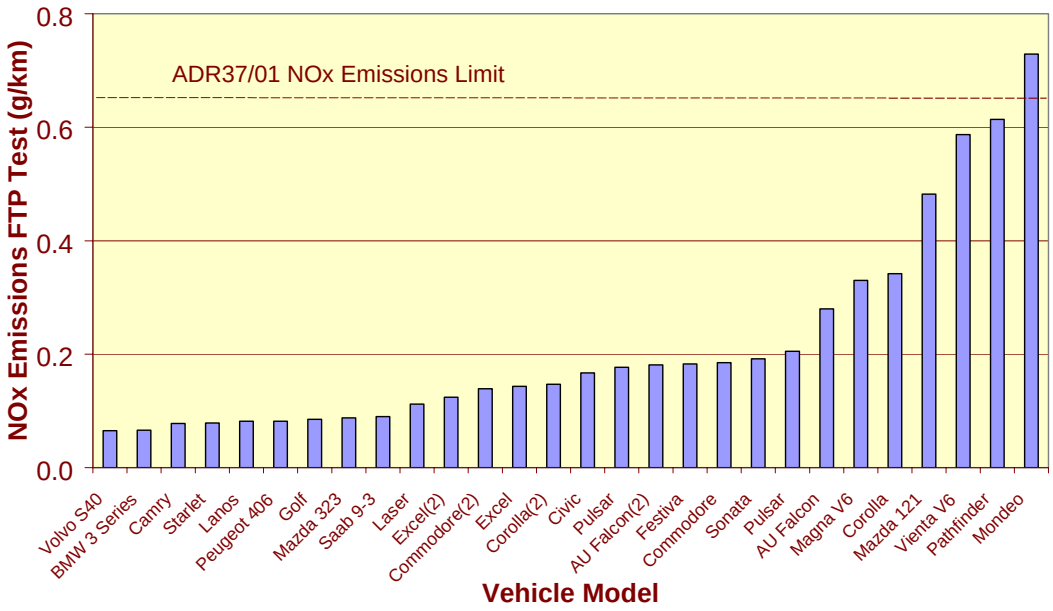
[11] However, methane is an issue in greenhouse gas terms and dedicated catalysts with a high loading of active components are needed to maximise methane oxidation (Beer et al 2001).

[12] Australian Design Rules are national standards under the *Motor Vehicle Standards Act 1989*.

[13] Where there are no additional treatments to reduce cold start emissions.

- wear and tear
- failure of vehicle components
- lower levels of vehicle maintenance
- the reduced efficiency of emission controls—for example, the conversion efficiency of the catalysts in catalytic converters deteriorates with distance (Environment Australia 2000).

FIGURE 3.2 NITROGEN OXIDE EMISSIONS FROM ADR37/01 VEHICLES ON THE ADR37/01 TEST



Note Most vehicles in the Comparative Vehicle Emissions Study were designed to comply with ADR37/01.

Source Australian Government Department of Transport and Regional Services 2001

When a sample of essentially new vehicles in the Australian fleet was tested, average emission rates were around 60 per cent below the nominated limits (Australian Government Department of Transport and Regional Services 2001).

In a study of light duty vehicles in Denver, Colorado, Cadle et al (1999) found that new vehicles have very low emission rates. The study reported that emission rates for smoking gasoline vehicles and older light-duty diesel averaged 100 times higher than the newer vehicles.

Motor vehicle emissions vary significantly with vehicle and engine operation. These in turn are strongly related to road types, and hence vehicle speeds and driving patterns. The road types considered in the National Pollution Inventory estimates for motor vehicles are arterial, freeway and residential. Each has a different assumed average speed and congestion pattern. Factors that are not considered in deriving inventory estimates include:

- road conditions and grade
- weather conditions
- the proportions of hot and cold starts
- the use of air conditioners (Environment Australia 2000).

Aircraft

Aircraft emissions of ambient air pollutants are small where National Pollution Inventory emission estimates are available. The exceptions to this are Canberra and Adelaide. Aircraft generate about three per cent of total sulfur dioxide emissions in Canberra and almost five per cent of total PM_{10} emissions—excluding road dust—in Adelaide.

There are no National Pollution Inventory estimates for aircraft in the Sydney–Newcastle–Wollongong or southeast Queensland airsheds. According to the Australian Academy of Technological Sciences and Engineering (1997a) however, aircraft in the Sydney–Newcastle–Wollongong airshed contributed around 3.2 per cent of nitrogen oxides but considerably less carbon monoxide and volatile organic compounds.

Aircraft produce substantially more emissions per movement than motor vehicles. But aircraft spend so little of their operational time in airsheds that they make only a small contribution to total airshed pollutant load (Australian Academy of Technological Sciences and Engineering 1997a, p. 50).

Estimating aircraft emissions in an airshed is a complex task and there is a lack of data compared with motor vehicle emissions.[14] According to Environment Australia, two actions are needed to improve the accuracy of estimates:

- More work is required on the emission profiles of different engines under differenet operating modes.
- The serious lack of data on emissions of particulate matter from aircraft engines must be rectified (Environment Australia 2003b).

Unlike motor vehicles, most aircraft emissions are unregulated although the International Civil Aviation Organisation has standards to regulate hydrocarbons (Environment Australia 2003b).

[14] The National Pollution Inventory techniques used to estimate aggregate aircraft emissions include estimating for landings, taxi/idle, takeoff and climb out up to 1000 metres. Information is needed about engine emission speciation and rates for different operating modes, as well as the number of movements by aircraft type and flight paths.

Shipping and boating

Where estimated, shipping and boating emissions of carbon monoxide, nitrogen oxides, PM_{10} and sulfur dioxide were small in most capital city airsheds in 2000–01.

The National Pollution Inventory method provides techniques for estimating emissions from commercial shipping and boating, and recreational boating sources (Environment Australia 1999c). Commercial and recreational boating emissions arise from boat engines while the boats are travelling. While both inboard and outboard engines are used in commercial boats, most recreational boats use outboard engines. All outboard engines use petrol and most inboard engines use diesel.

Commercial ships are driven primarily by large slow-speed and medium-speed diesel engines, and occasionally by steam turbines and gas turbines—these latter excluded from the approved estimation method due to limited usage. Ships emit air pollutants while underway, and at berth where they use diesel-powered generators to furnish auxiliary power. Emissions underway come from a ship's engine exhaust and are influenced by engine size, the fuel used—residual oil or diesel oil, operating speed and load. In addition to engine exhaust emissions, there are fugitive emissions from the loading and ballasting of petroleum tankers in port.

The information needed to estimate commercial shipping emissions includes:

- composition of the fleet using a port
- engine types and fuel types of each craft
- time spent at berth
- time spent from the airshed perimeter to engine shut-down and vice versa
- the number of movements per year.

There are usually no controls on exhaust emissions from commercial ships, commercial boats and recreational boats.

Estimates for shipping and boating are less reliable than for other transport sources due to lack of data. Given the complexity of the estimation process and the fact that much of this detail is less available than for aviation, most estimates in the literature are regarded as 'order-of-magnitude' (Australian Academy of Technological Sciences and Engineering 1997b).

Rail

Rail emissions of carbon monoxide, nitrogen oxides, sulfur dioxide, PM_{10} were negligible in all capital city airsheds in 2000–01 where National Pollution Inventory emission estimates are available. The exceptions to this are Perth and Adelaide with less than two per cent of total PM_{10} and less than two percent of nitrogen oxides respectively.

Where available, National Pollution inventory emission estimates for rail include diesel-electric locomotives—which use a diesel engine and an alternator or generator to power their traction motors—but exclude electric and steam trains. Also excluded are emissions from small engines used on refrigerated and heated railcars, as well as brake dust particulate matter from trains. There are usually no specific emission devices fitted to locomotive engines.

While the National Pollution Inventory aggregate emissions method for rail includes both line haul and railyard sources, the latter may be included in facilities estimates. Locomotive emission factors are derived from the United States national locomotive fleet. The areas of greatest uncertainty for rail are the exhaust profiles for volatile organic compounds and particulate matter where diesel truck engine profiles are used (Environment Australia 1999b).

Road dust

When a vehicle travels over a paved road, particulate emissions are generated by the suspension or re-suspension of loose material on the road surface. Field studies have found that roads are major sources of airshed particulate matter. The main source of road dust particles is local soil and other sources of loose material (Environment Australia 1999a; Environment Australia 2000).

Road dust may comprise a large proportion of PM_{10} by mass—as measured for inventory purposes. But motor vehicles and other combustion sources produce a much higher proportion of $PM_{2.5}$. Cadle et al (1999) found that more than 90 per cent of motor vehicle PM_{10} is $PM_{2.5}$. This difference is important. Pope et al (2002) have shown stronger associations between all-cause, cardiopulmonary and lung cancer mortality and fine particle exposure, including $PM_{2.5}$, than with the coarser PM_{10} and PM_{15} fractions. The health effects of air pollutants are discussed in detail in chapter 4.

There is significant uncertainty about the order of magnitude of the Australian road dust estimates. A National Pollution Inventory method has been agreed to estimate emissions from paved and unpaved road sources and South Australia, Victoria and Tasmania have produced estimates. Major sources of uncertainty include the sparseness of Australian emissions data and year-to-year variation in weather conditions. In deriving its assessment of the state of ambient air quality, the 2001 State of the Environment Report stated:

> … authorities often stressed the uncertainties associated with the data and were reluctant to agree to their processing and dissemination (e.g. data associated with road dust emissions of particulate matter…). The data associated with road dust emissions highlights our lack of knowledge in relation to the sources of exposure to particulate matter. (Manins et al 2001)

This study has excluded road dust due to the lack of National Pollution Inventory data for most capital cities and uncertainty about the order of magnitude of the estimates.

Related industry sources

Transport-related industry emission sources range from service facilities such as ports and airports to transport vehicle maintenance, repair and manufacturing. Fuel refining is not a transport-related industry source.

Under the National Pollution Inventory, industry emissions comprise a mix of self-reporting—where companies report when emissions are above certain thresholds—and aggregated emissions estimates for small industrial sources. There is significant variation in industry emissions across states and territories.

Where they have been estimated, transport-related industry sources make only a small contribution to total airshed emissions of nitrogen oxides, carbon monoxide, PM_{10} and sulfur dioxide. For example, emissions from non-aircraft service operations are much smaller than aircraft exhaust emissions (Australian Academy of Technological Sciences and Engineering 1997a, p. 50). Non-aircraft industrial sources of PM_{10} are only significant in the Sydney–Newcastle–Wollongong and Port Phillip Bay airsheds. Reported non-aircraft emissions in the Sydney–Newcastle–Wollongong airshed comprised 432 tonnes,[15] or two per cent, of total PM_{10} emissions in 2000–01.

An exception was for particulate matter in the Port Phillip Bay airshed where 2000–01 emissions, largely sourced from vehicle manufacturing, comprised about six per cent—excluding road dust.

TRANSPORT SHARE OF MAJOR AIR POLLUTANTS IN CAPITAL CITIES

This section describes transport's contribution to ambient air pollution in Australian capital cities using the National Pollution Inventory database—the best available source of emissions estimates for a range of major airsheds and major ambient air pollutants. However, it does not cover the whole of Australia—there are 32 airsheds including urban and rural locations. The inventory is still being developed and there are many issues that introduce uncertainty into the total magnitude of emission estimates, transport's contribution to the total, and state and territory comparisons. They include:

- uncertainty about the size of road dust particulate emissions
- lack of inventory estimates for rail, aircraft, shipping and boating sources for the Sydney–Newcastle–Wollongong and Southeast Queensland airsheds
- non-capital city transport shares cannot be estimated because aggregated inventory emission estimates are unavailable for many regional areas
- states and territories may have used different approved methodologies to estimate aggregated emissions

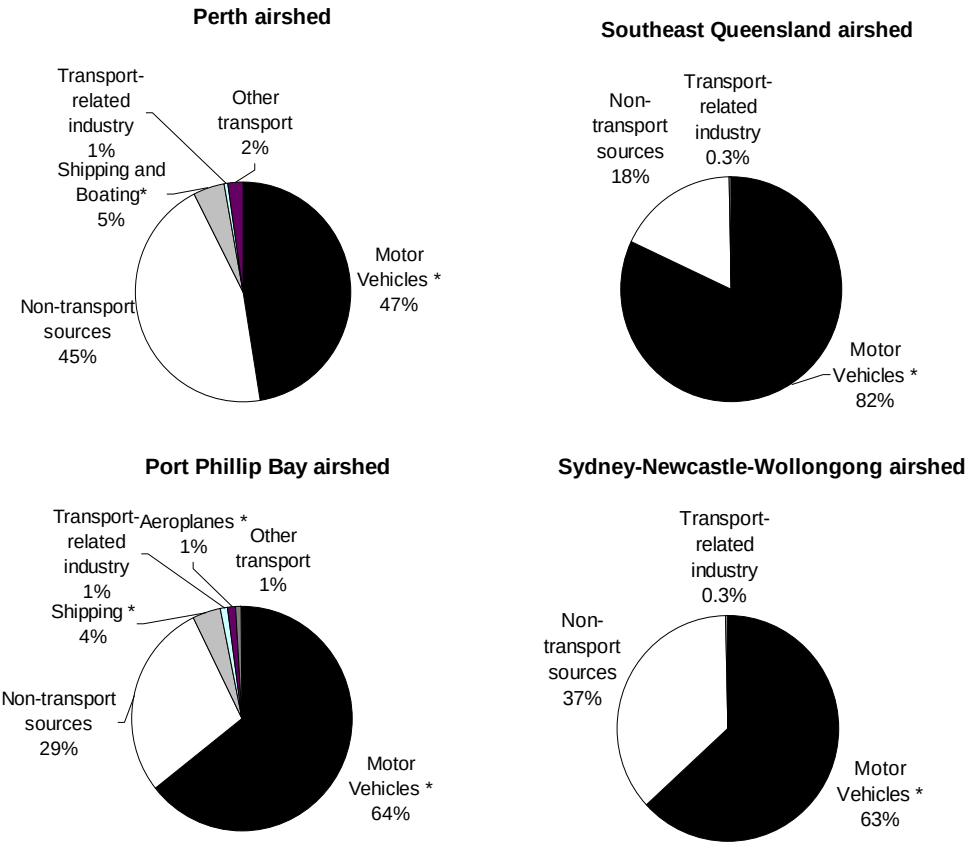
[15] Ansett produced most of these emissions. Following Ansett's collapse in September 2001, non-aircraft emissions reduced to 76 tonnes or 0.35 per cent of 2001–02 PM_{10} emissions.

- under-reporting of facility (industry) emissions will overstate the transport share of aggregated emissions
- the choice of airshed boundary affects total emissions and transport share.[16]

Nitrogen oxides

The transport share of nitrogen oxides emissions in 2000–01 ranged from more than 50 per cent in the Perth airshed to more than 90 per cent in the Canberra airshed (Figure 3.3).

FIGURE 3.3 SOURCES OF NITROGEN OXIDES FOR SELECTED CAPITAL CITY AIRSHEDS 2000–01



Note Excludes rail, aircraft, shipping and boating sources for the Sydney–Newcastle–Wollongong and Southeast Queensland airsheds.

Source Bureau of Transport and Regional Economics estimates using National Pollution Inventory data

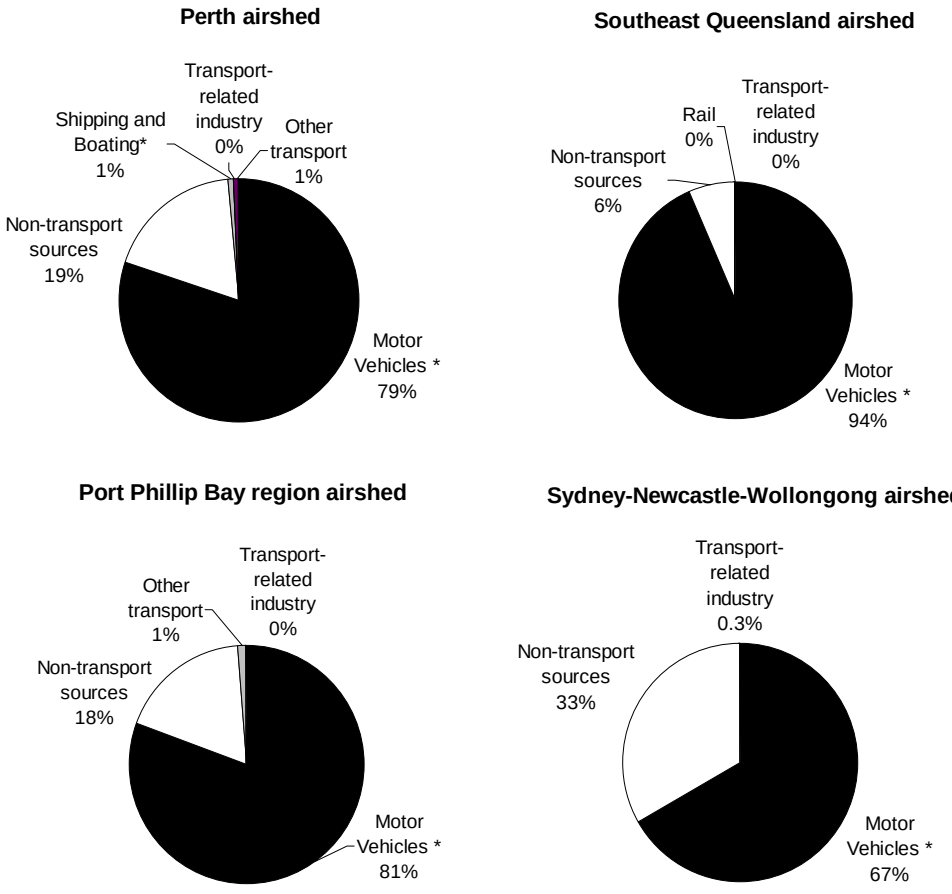
16 For example, Greater Sydney includes emissions from the Hunter Valley, the electricity generating area to the north of Sydney. Data for the Port Phillip Bay airshed excludes the analogous area, the Latrobe Valley, to the east of Melbourne (Manins et al 2001). Size and location of airsheds are determined by stakeholder priorities and the reasons for selection.

In terms of the transport share, Figure 3.3 indicates motor vehicles are the major transport source in all capital cities and that aviation, shipping and boating—where estimated—are relatively minor sources.

Carbon monoxide

In 2000–01, between 70 and 90 per cent of carbon monoxide emissions for all capital city airsheds, except Darwin, were attributed to motor vehicles. Bushfires were the major source of carbon monoxide in Darwin—motor vehicles accounted for only 20 per cent of carbon monoxide emissions. Figure 3.4 shows the relative transport and related industry shares of carbon monoxide emissions for selected capital city airsheds.

FIGURE 3.4 SOURCES OF CARBON MONOXIDE FOR SELECTED CAPITAL CITY AIRSHEDS 2000–01



Note Excludes rail, aircraft, shipping and boating sources for the Sydney–Newcastle–Wollongong and Southeast Queensland airsheds.

Source Bureau of Transport and Regional Economics estimates using National Pollution Inventory data

Volatile organic compounds (hydrocarbons)

Major volatile organic compounds emitted by motor vehicles include benzene, toluene, and xylenes. The National Pollution Inventory requires individual reporting for specific compounds as well as total volatile organic compounds. The National Pollution Inventory definition specifically excludes methane, carbon monoxide, carbon dioxide, carbonic acid, metallic carbides and carbonate salts.

The inventory substance-reporting list (table 1 and 2 substances) contains 42 volatile organic compounds. These include aliphatic hydrocarbons—for example hexane; aromatic hydrocarbons—for example benzene, toluene, and xylenes; and oxygenated compounds—such as acetone and similar ketones. Some compounds are included as individual substances due to their toxicity to plants, animals and humans—not because of their activity as a precursor to the formation of smog (Environment Australia 2003a).

Total volatile organic compounds—including benzene, toluene, and xylenes—is a National Pollution Inventory reportable substance because it captures the combined effect of compounds contributing to smog where individual substances do not meet a usage threshold.

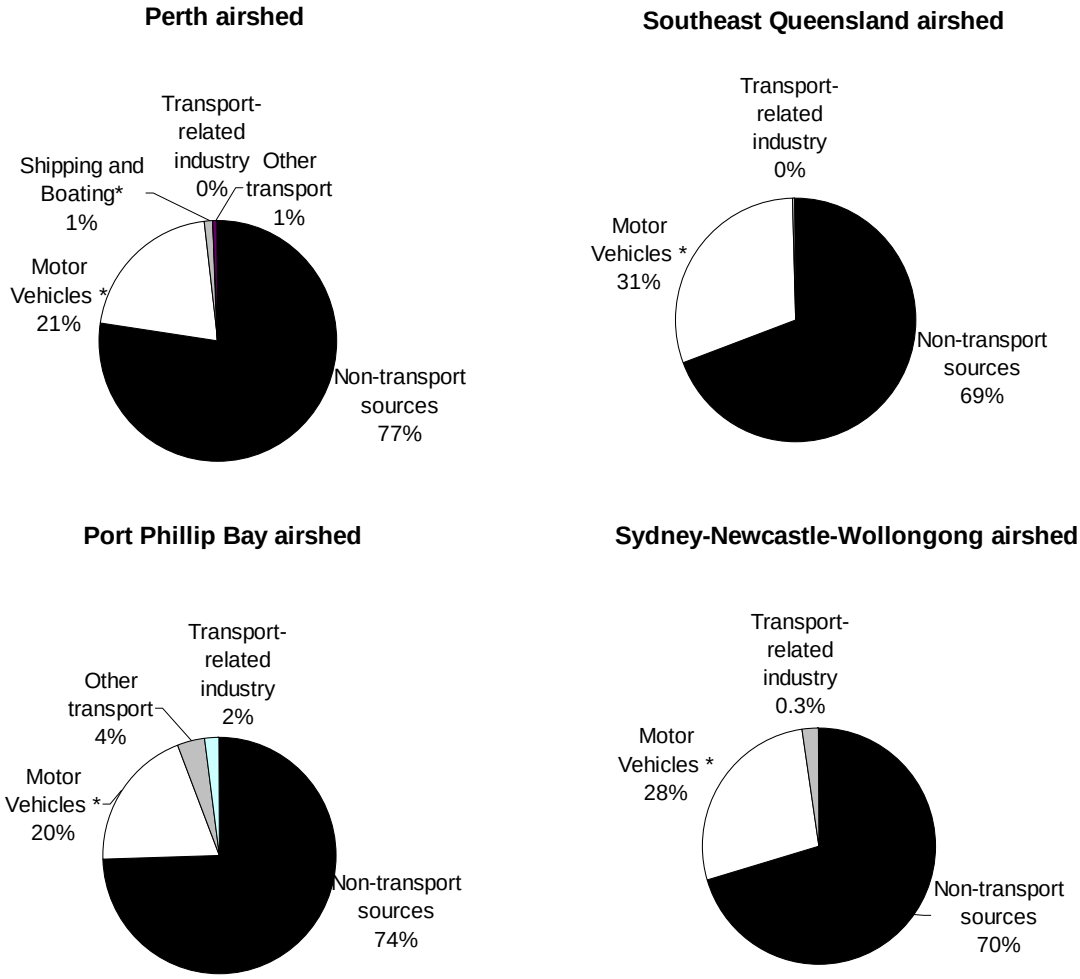
In 2000–01 motor vehicle sources in the Sydney–Newcastle–Wollongong airshed contributed:

- 2.9 million kilograms of benzene—75 per cent of total airshed emissions. Service stations accounted for an additional five per cent.
- 6.1 million kilograms of toluene—67 per cent of total airshed emissions. Service stations accounted for an additional six per cent.
- 6 million kilograms of xylene—75 per cent of total airshed emissions. Service stations accounted for an additional seven per cent.
- 96 million kilograms of total volatile organic compounds—almost two-thirds of total airshed emissions. Service stations accounted for a further four per cent.

Particulate matter of less than 10 microns (PM_{10})

Transport is a significant source of PM_{10} in most major capital city airsheds—see figure 3.5—with motor vehicles the largest single source of transport PM_{10} in these cities. However, when compared to nitrogen oxides and carbon monoxide—see figures 3.3 and 3.4—the estimated transport share of PM_{10} in the major capital city airsheds was low at between 20 and 30 per cent. For Darwin, Canberra and Hobart the estimated transport share of PM_{10} was 10 per cent or less.

FIGURE 3.5 SOURCES OF PM_{10} (EXCLUDING ROAD DUST) FOR SELECTED CAPITAL CITY AIRSHEDS 2000–01



Note Excludes rail, aircraft, shipping and boating sources for the Sydney–Newcastle–Wollongong and Southeast Queensland airsheds. Transport sources of ambient pollution excludes both road dust and secondary sources of pollution such particles generated in the atmosphere by chemical reactions involving transport emissions (such as sulphates from sulfur dioxide and nitrates from nitrogen oxides emissions).

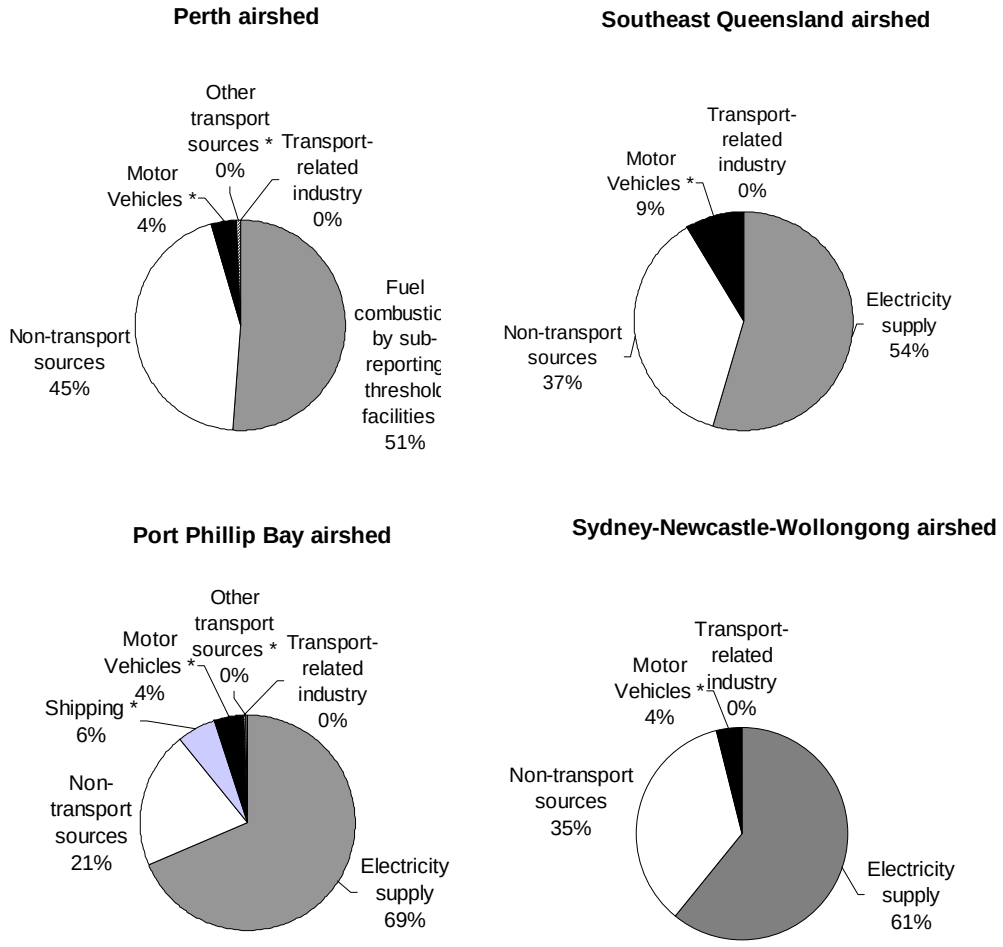
Source Bureau of Transport and Regional Economics estimates using National Pollution Inventory data.

However the transport share of PM_{10} varies widely across cities and is much higher in central city areas. For example, motor vehicles' share of PM_{10} in the Sydney central business district is approximately 55 per cent. In the Sydney–Newcastle–Wollongong airshed it is less than 30 per cent. These airshed emission estimates exclude PM_{10} from paved and unpaved roads. Where estimates have been made, road dust emissions as a proportion of airshed PM_{10} —including road dust—range from 63 per cent in the Adelaide airshed to 27 per cent in the Port Phillip Bay airshed.

Sulfur dioxide

Transport is a significant source of sulfur dioxide emissions in Adelaide, Canberra and Darwin and a minor source in other capital city airsheds—see figure 3.6.

FIGURE 3.6 SOURCES OF SULFUR DIOXIDE IN SELECTED CAPITAL CITY AIRSHEDS, 2000–01



Note Excludes rail, aircraft, shipping and boating sources for the Sydney–Newcastle–Wollongong and Southeast Queensland airsheds

Source Bureau of Transport and Regional Economics estimates using National Pollution Inventory data.

Power generation sources dominated sulfur dioxide airshed emissions in the Sydney–Newcastle–Wollongong, Port Phillip Bay, and Southeast Queensland airsheds. The major sources of sulfur dioxide in the Adelaide and Perth airsheds are industry facilities. In the Canberra and Hobart airsheds domestic fuel burning for heating is a significant source of sulfur dioxide emissions.

Transport's contribution to sulfur dioxide emissions is relatively small. However, motor vehicles are the largest transport source of sulfur dioxide in all capital city airsheds except in the Darwin and Port Phillip Bay airsheds. In these airsheds shipping is the major source.

Overall, as discussed in Chapter 2, ambient sulfur dioxide levels are generally not considered a problem in urban areas. However, the move to lower sulfur standards for fuel will reduce motor vehicles' contribution to urban sulfur dioxide pollution.

Lead

As discussed in Chapter 2, lead levels in ambient air have dropped to the point that lead in air is no longer an issue in metropolitan Australia (Manins et al 2001). In 2000–01 motor vehicles contributed more than 90 per cent of residual lead emissions in all capital city airsheds except Adelaide.

All major pollutants by airshed

Table 3.1 summarises indicative transport and related industry contributions by city airshed for major ambient pollutants for 2000–01. The National Pollution Inventory is continuing to evolve and data in several areas are subject to a high degree of uncertainty. Consequently, estimated shares by source are indicative of the likely order of magnitude.

TABLE 3.1 INDICATIVE TRANSPORT AND RELATED INDUSTRY SHARES OF MAJOR POLLUTANTS FOR MAJOR CITY AIRSHEDS 2000–01

per cent

Airshed	*Nitrogen oxides*	*Carbon monoxide*	*Sulfur dioxide*	*PM_{10}*	*Lead*
Adelaide	64	89	32	24	66
Canberra	93	76	32	10	93
Darwin	63	20	65	2	92
Hobart	84	68	6	7	96
Port Phillip Bay region	71	82	11	30	96
Perth	54	82	4	16	95
Southeast Queensland	82	93	9	31	Nc
Sydney–Newcastle–Wollongong	63	67	4	30	nc

a. Total PM_{10} airshed estimates exclude paved and unpaved road sources and include transport-related industrial sources.

b. National Pollution Inventory data comprise reported emissions by industrial facilities and emissions estimates for smaller companies and a range of mobile and non-industrial sources.

c. Proportions rounded to the nearest whole per cent.

Source Bureau of Transport and Regional Economics estimates using National Pollution Inventory data

Because the National Pollution Inventory is still evolving, it has many shortcomings. For example, it only provides data on known pollutant sources where they have been reported by industry or estimated by the responsible jurisdiction.

There are also several other important issues and caveats concerning inventory data that may affect the transport share.

For example, the number of industrial facilities reporting emissions is increasing as the reporting mechanism becomes better established. A total of 2948 facilities reported to the National Pollution Inventory in 2001–02 compared with 2374 the previous year (Kemp 2003). Better reporting may in part explain the increase in PM_{10} facility emissions in 2001–02. Total reported PM_{10} emissions increased by 80 million kilograms or 21 per cent when compared with 2000–01.

An important caveat is that aggregated National Pollution Inventory emission estimates including transport are not updated annually. The 2000–01 inventory estimates for transport and other aggregated sources were based on results of studies conducted during the 1990s. It is possible to update estimates using recent vehicle movement data. But it is important to note that the emission factors used to estimate emissions also change over time. Older airshed studies would be expected to overstate the motor vehicle share of emissions particularly for carbon monoxide and volatile organic compounds. Reductions in emissions from these sources are due in large part to the removal of non-catalytic converter, pre-1986 vehicles from the vehicle fleet.

It is not possible to use the National Pollution Inventory data to estimate the transport share in regional areas. Aggregated inventory emission estimates for transport sources—including motor vehicles, aircraft and rail—are not available for many regional areas (including regional cities such as Albury and Cairns).

As previously noted, the major issue in regional areas is particulate matter. In most regional areas the main contributors of particulate matter are bushfires, mining and agriculture, and windblown particles. Where National Pollution Inventory data are available for regional areas it suggests that motor vehicle exhaust emissions—as opposed to road dust—are a minor source of particulate matter.

Other important caveats apply when interpreting emission inventory data. A major caveat is that there is not a direct link between emissions and human exposure. Another caveat is that the National Pollution Inventory, like most emission inventory data, records mass concentration data—presented as kilograms of specific pollutants. For fine particles, particle diameter and trace contaminants—which can both vary widely by source—may be more important in terms of human health (Ayers et al 1999).

Despite these shortcomings, the National Pollution Inventory represents the best available data on emissions of ambient air pollutants in Australian cities.

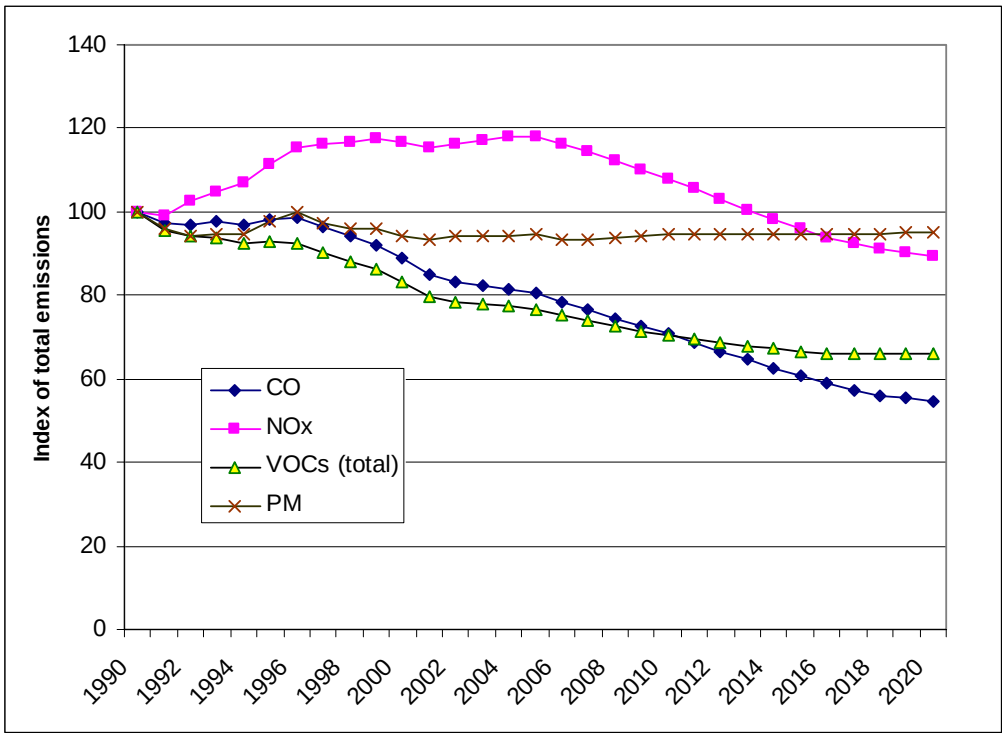
Ultimately it is emissions inventories upon which management measures or options must be based (Australian Academy of Technological Sciences and Engineering 1997a, p. 32).

MOTOR VEHICLE EMISSION TRENDS AND PROJECTIONS

Since 1990 motor vehicle emissions of carbon monoxide and total volatile organic compounds have decreased—see figure 3.7. At the same time, total nitrogen oxides emissions have increased and PM_{10} emissions have been relatively stable (Bureau of Transport and Regional Economics 2003b).

Over the same period, lead emissions decreased due to the phasing-out of leaded fuel and removal of vehicles using leaded fuel from the fleet.

FIGURE 3.7 PROJECTED GROWTH IN MAJOR POLLUTANT EMISSIONS FROM MOTOR VEHICLES FOR AUSTRALIAN METROPOLITAN AREAS, 1990–2020 (INDEX WITH 1990 = 100)



Source Bureau of Transport and Regional Economics 2003b

In 2000, the total vehicle kilometres travelled was 113 billion kilometres. Passenger cars travelled the greatest number of vehicle kilometres in 2000. There are fewer trucks and other commercial vehicles, and they travel fewer kilometres in the urban environment.

Total vehicle kilometres travelled are expected to increase to 166 billion kilometres by 2020—an overall increase of 46 per cent, or 1.9 per cent annually

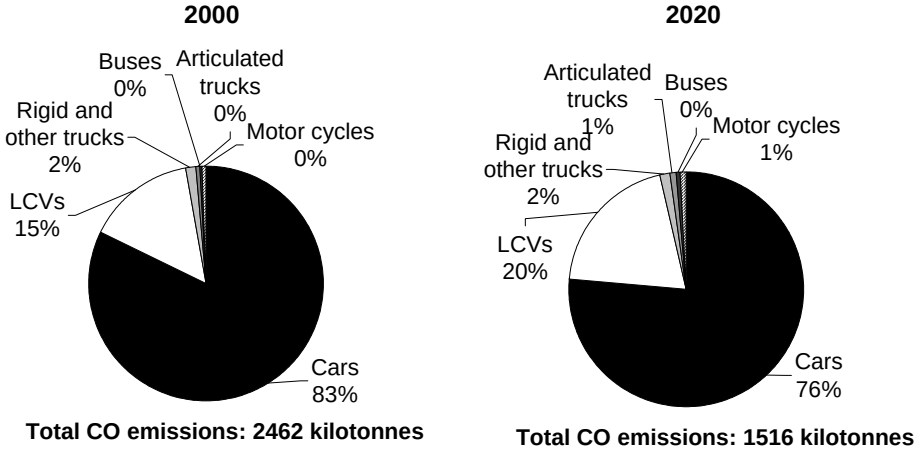
(Bureau of Transport and Regional Economics 2003b). However, there are significant differences in the projections by vehicle type. By 2020 total vehicle kilometres travelled are expected to increase by:

- 36 per cent by cars—including four-wheel drive passenger vehicles
- 107 per cent by light commercial vehicles
- 26 per cent by rigid trucks
- 120 per cent by articulated trucks
- 31 per cent by buses
- 16 per cent by motorcycles.

Balancing the increase in total vehicle kilometres travelled, the average vehicle emission performance will continue to improve as new, more efficient vehicles replace older, pre-1986 vehicles—those without catalytic converters.

Total metropolitan carbon monoxide emissions are projected to decrease substantially by 2020. However, light commercial vehicles' share increases substantially, while cars' relative share declines—see figure 3.8.

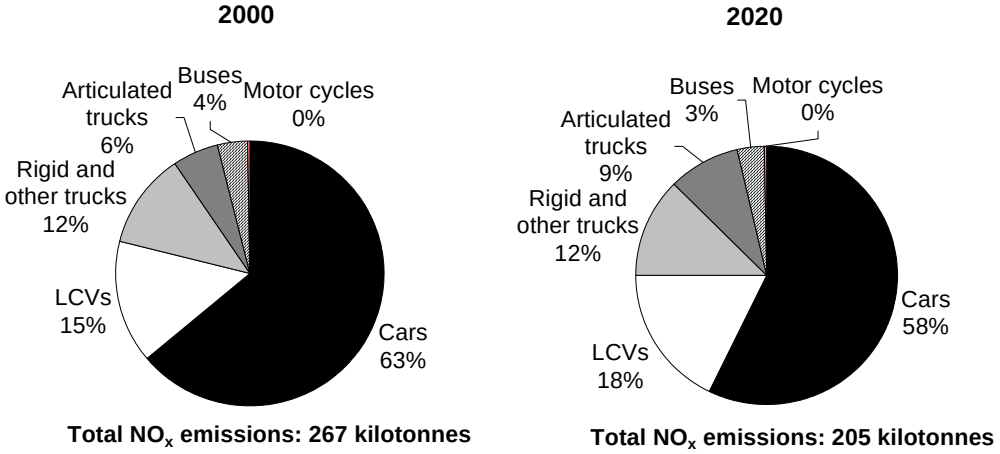
FIGURE 3.8 METROPOLITAN CARBON MONOXIDE EMISSION PROJECTIONS: SHARES BY VEHICLE TYPE



Source Bureau of Transport and Regional Economics 2003b

Metropolitan motor vehicle emissions of nitrogen oxides are also projected to decrease substantially by 2020. However, light commercial vehicles and articulated trucks' shares of total nitrogen oxides emissions are expected to increase. Cars' share is expected to decrease—see figure 3.9.

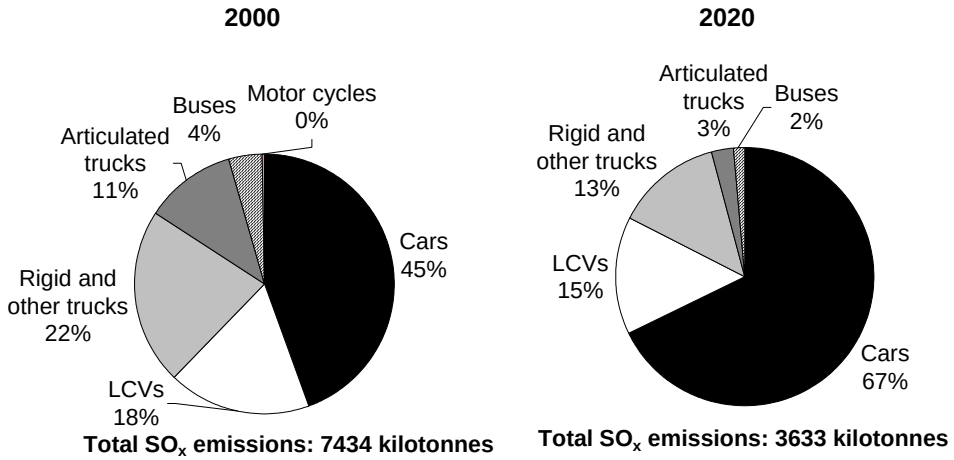
FIGURE 3.9 METROPOLITAN NITROGEN OXIDE EMISSION PROJECTIONS: SHARES BY VEHICLE TYPE



Source Bureau of Transport and Regional Economics 2003b

Total metropolitan sulfur dioxide emissions are projected to decrease by more than 50 per cent by 2020. By 2020, the relative shares of all truck categories decreases substantially and the share of light commercial vehicle emissions decreases slightly—see figure 3.10. For the same period, cars' share of sulfur dioxide emissions is expected to increase from 45 per cent to 67 per cent.

FIGURE 3.10 METROPOLITAN SULFUR DIOXIDE EMISSION PROJECTIONS: SHARES BY VEHICLE TYPE



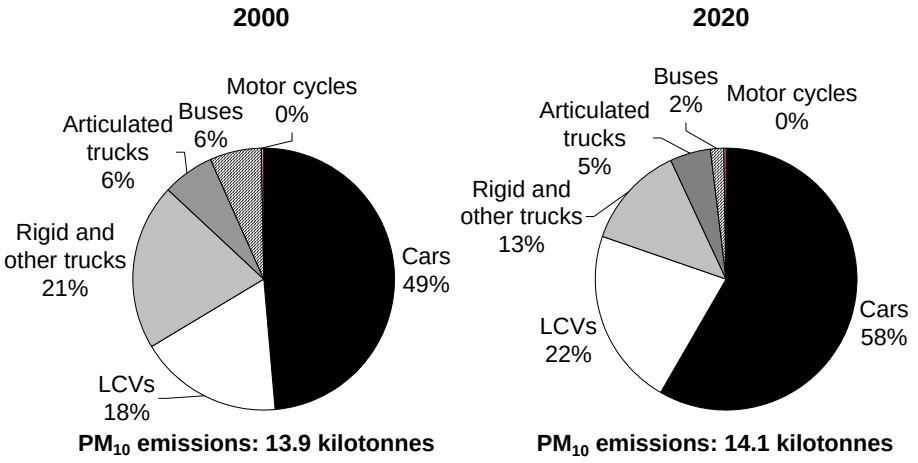
Source Bureau of Transport and Regional Economics 2003b

Metropolitan PM_{10} emissions are projected to remain virtually unchanged until 2020, despite an expected 46 per cent increase in projected total fleet vehicle

kilometres travelled. By 2020, cars' relative share of metropolitan motor vehicle PM_{10} emissions increases from 49 to 58 per cent, light commercial vehicles' share increases marginally and trucks' share declines in all categories—see figure 3.11.

Cars' share of total PM_{10} emissions is projected to increase due to the sheer number of cars and the fact that particulate matter emissions by petrol cars—unlike diesel passenger vehicles—are not subject to Australian Design Rules.

FIGURE 3.11 METROPOLITAN PM_{10} EMISSION PROJECTIONS: SHARES BY VEHICLE TYPE



Source Bureau of Transport and Regional Economics 2003b

Measures for a Better Environment and projected motor vehicle emissions

The projected reductions in motor vehicle emissions of carbon monoxide, nitrogen oxides and volatile organic compounds are due to:

- the removal of older, particularly non-catalyst, vehicles
- the introduction of new vehicles subject to progressively more stringent emissions standards
- the progressive introduction of low-sulfur fuel standards for both diesel and petrol.

The Australian Government's Tax Package Agreement announced on 28 May 1999 included a broad range of environmental initiatives entitled Measures for a Better Environment. One of these measures was the introduction of a package of stringent new-vehicle emission standards for petrol and diesel vehicles to take effect between 2002 and 2007. These new standards are based on European

standards commonly referred to as Euro 2, 3 and 4 (Australian Government Department of Transport and Regional Services 2002).

For diesel vehicles the package included the adoption of:

- Euro 2 in 2002–03 for all new light diesel vehicles
- Euro 3 in 2002–03 for all new medium and heavy duty diesel vehicles
- Euro 4 in 2006–07 for all new diesel vehicles.

For petrol vehicles the package included the adoption of:

- Euro 2 in 2003–04 for all new petrol vehicles
- Euro 3 in 2005–06 for all new petrol vehicles.

New Australian Design Rules announced on 20 December 1999 give effect to the new vehicle standards in the Measures for a Better Environment package. These new standards progressively reduce the limits for carbon monoxide, hydrocarbons and nitrogen oxides. They also subject new vehicles to more stringent emission testing. These new vehicle standards also reduce the emission limit for diesel particulate matter by 80 to 90 per cent.

The Measures for a Better Environment package of new motor vehicle emission standards will lead to significant reductions in emissions from new motor vehicles. The most significant reductions will be in the PM_{10} and nitrogen oxides limits—see table 3.2. The new standards will also include tighter evaporative hydrocarbon standards for petrol vehicles.

TABLE 3.2 REDUCTIONS IN VEHICLE EMISSION LIMITS

Standards	*Cars (percentage reduction)*		*Four wheel drive and light commercial vehicles (percentage reduction)*		*Heavy duty vehicles (percentage reduction)*	
	Petrol	Diesel	Petrol	Diesel	Diesel	Diesel
From	NO_x	PM	NO_x	PM	NO_x	PM
Euro 2 [a] to Euro 3	30	40	30-35[b]	40	30	35
Euro 3 [b] to Euro 4	50	50	50	50	30	80

Note Percentage reductions have been rounded to the nearest five per cent.

a. For Euro 2 standards there is a combined regulated limit for hydrocarbons and nitrogen oxides, EU assume a ratio of 55 per cent evaporative hydrocarbons to 45 per cent nitrogen oxides.

b. Range reflects differing reductions depending on the mass of the vehicle.

Source Australian Government Department of Transport and Regional Services 1999

New fuel standards are being progressively introduced for both diesel and petrol as part of the Measures for a Better Environment package. This includes measures to ensure low-sulfur diesel is available within the timeframe of the new vehicle standards. The Australian Government has already announced

increases in excise and customs duty that will fund grant payments to producers and importers of premium unleaded petrol from 1 January 2006. The grants will be available to producers and importers of premium unleaded petrol with less than 50 parts per million sulfur—this complies with the Euro 4 standard. Similar arrangements will also be implemented from 1 January 2007 for diesel with less than 10 parts per million sulfur—this complies with the Euro 5 standard (Costello and Kemp 2003).

Unlike the changes in vehicle standards, these reductions in the sulfur content of fuel will enhance the reductions in total ambient air pollution emissions. They will also reduce emissions across the entire fleet—not just in vehicles meeting the new emission standards (Australian Government Department of Transport and Regional Services 1999, p. 12).

The principal benefit of reducing sulfur is to enable the use of better emissions technology. With diesel vehicles there would be some direct benefit from a reduction from 50 parts per million sulfur to 10 parts per million. One estimate projects that particulate matter emissions are likely to be reduced by approximately five per cent (Motor Vehicle Environment Committee 2003, p. 38).

Motor vehicle projections to 2020

The Bureau of Transport and Regional Economics projects that the phase out of older vehicles, combined with new Australian Design Rules to 2006, will deliver overall reductions in total metropolitan motor vehicle emissions to 2020. For the major ambient air pollutants:

- Carbon monoxide emissions are projected to decline by approximately 38 per cent.
- Nitrogen oxides emissions are projected to decline by approximately 23 per cent.
- Volatile organic compound emissions—including exhaust and evaporative emissions—are projected to decline by approximately 20 per cent.
- Sulfur dioxide emissions are projected to decline strongly following the reductions in fuel sulfur content with the phased introduction of the new fuel standards.
- Projected total transport particulate matter emissions—excluding road dust—are projected to remain stable over the period. (Bureau of Transport and Regional Economics 2003b).

Even though total fleet emissions are expected to decline, emissions of most pollutants will remain significant—with the exception of lead. These projections do not include re-fuelling and other service station volatile organic compound emissions, road dust or secondary particles formed by chemical reactions in the atmosphere.

Several points should be noted about the projections. Firstly, the long life of large diesel commercial vehicles means that new, less polluting vehicles are likely to comprise a relatively small proportion of the total fleet by 2020. In its major air pollutants projections, the Bureau has assumed low penetration rates for hybrid fuel vehicles (Bureau of Transport and Regional Economics 2003b).

Secondly, the fastest growing segment of the vehicle market is sports utility vehicles (Australian Bureau of Statistics 2004). This may be expected to disproportionately increase PM_{10} emissions to the extent that sports utility vehicles are more likely to use diesel fuel than cars. According to the Australian Bureau of Statistics Motor Vehicle Census, the total number of diesel passenger vehicles increased more than 73 000, or almost 37 per cent, between 1997 and 2002 (ABS 2002). However as a proportion of the passenger-vehicle fleet, diesel vehicles comprised only 2.7 per cent at 31 March 2002—272 072 vehicles, up from 2.2 per cent in 1997—198 615 vehicles. Further, the Measures for a Better Environment package is expected to substantially reduce particle emissions across the diesel fleet.

As with all projections derived by aggregate modelling techniques, these projections have reasonably high levels of uncertainty—notably for particulate matter. Key assumptions—about which relatively little is known—include rates of engine degradation and emission equipment failure.

CONCLUDING REMARKS

Transport is the major source of carbon monoxide, nitrogen oxides and a significant source of PM_{10} in Australian capital cities' airsheds. Motor vehicles are the major transport source of these pollutants in capital cities. Unlike aircraft, locomotives or ships, new motor vehicles are subject to performance-based standards that regulate exhaust emissions of most major pollutants.

When it estimated the transport shares of urban air pollution, this study used the National Pollution Inventory database of emissions by source. However, data issues limited the estimation to health costs to motor vehicle sources of PM_{10}.

A key issue is that the National Pollution Inventory does not provide estimates for other transport sources for all capital city airsheds. However, where inventory data are available, it indicates that emissions from other transport modes and related industry sources are generally minor.

Another key issue is the uncertainty about the contribution, and significance of, road dust to total PM_{10} emissions. Unfortunately, estimates of road dust emissions are not available for all capital city airsheds. Where estimates were available, they were not sufficiently robust to include in this analysis. This implies an under-estimation of transport's share of PM_{10} in Australian capital city airsheds, and a possible underestimation of the size of the health effect of transport.

However, there is also uncertainty about the composition of road dust. It is likely to contain fewer trace pollutants than combustion sources and the average size of the road dust fraction is larger than particulate matter from combustion sources.

Emissions from some naturally occurring sources of PM_{10} have not been estimated for the National Pollution Inventory. Natural sources of PM_{10} include sea salt aerosols—chloride concentration, wind-blown mineral dust, and biological debris such as pollen. This will over-estimate the share—and the number of health cases and economics costs—attributed to human activity such as transport.

Looking forward, the Bureau has projected that motor vehicle emissions of nitrogen oxides, carbon monoxide and sulfur dioxide will decline significantly by 2020. It expects that PM_{10} emissions will be stable. These projections consider:

- the Australian Government's current policies including changes to Australian Design Rule new vehicle emission standards and fuel standards in the Measures for a Better Environment tax package
- a projected 46 per cent increase in total fleet vehicle kilometres travelled.

CHAPTER 4 HEALTH EFFECTS OF AIR POLLUTANTS

This chapter presents selected evidence of the health effects of air pollutants with reference to the existing body of research literature. The first section discusses health effects of individual major pollutants as indicated by the existing research studies. The second section addresses selected important issues of current research focus. The final section presents a brief literature review of the studies—most are epidemiological—on the health effects of air pollution covering Australian and overseas studies. The studies are presented by the method or approach used, and the findings and conclusions reached.

Extensive research on the health effects of air pollution has been carried out in different parts of the world characterised by:

- differing air quality
- different pollutant mixes and levels
- climate
- available civic amenities
- differing socioeconomic status of the population.

Most of these studies observed that air pollution has an association with both short-term and long-term mortality as well as morbidity effects on the exposed population

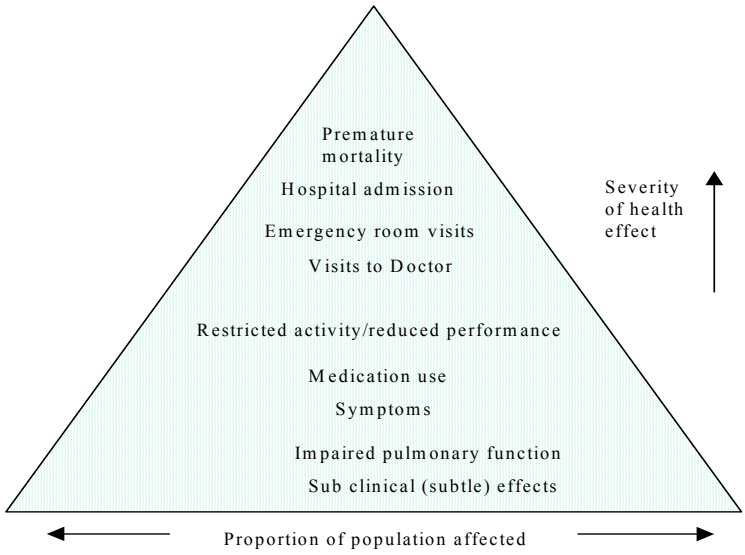
The focus on health effects of air pollutants dates back to the early and mid-20th century. At the time, a series of episodes showed that air pollution could have acute adverse effects on health. Since then, a vast amount of literature has emerged focusing on the damaging effects of air pollutants on public health.

Epidemiological studies during the 1990s revealed that people's health may be affected by exposure to much lower levels of some common air pollutants than believed even a few years ago (Fisher et al 2002). This suggests that despite significant reductions in the concentrations of many pollutants, adverse health effects still occur in most countries (World Health Organization (WHO) 2000b). Hence, even though Australia may be regarded as a country of low pollution levels, a potential health risk remains.

HEALTH EFFECTS OF INDIVIDUAL POLLUTANTS

The health effects can manifest in many ways. Figure 4.1 shows the path or the various stages of the health effects. These range from mild or subtle health effects to premature mortality.

FIGURE 4.1 THE SEQUENCE OF HEALTH IMPACT



Source World Health Organization 2000a

The effects of air pollutants on health vary depending on several factors. These include the level of exposure and the susceptibility of the exposed population. The susceptibility of the population is affected by factors such as the numbers of young children and older people, and the proportion of people suffering from asthma and other chronic respiratory conditions. In addition, sources and patterns of exposure—for example, indoor and outdoor exposures—are likely to differ substantially from region to region. This variation is partly dependent on weather conditions. The duration and type of exposure are also important.

Currently, researchers are interested in the health effects of these pollutants:

- fine particles
- ground-level ozone
- nitrogen oxides
- sulfur dioxide
- carbon monoxide
- benzene.

Table 4.1 summarises the health effects of these major pollutants.

TABLE 4.1 MAJOR AIR POLLUTANTS AND THEIR HEALTH EFFECTS

Pollutant	*Sources*	*National Pollution Inventory Ranking*	*Health effects*	*Where important*
Carbon monoxide	Motor vehicles, burning of fossil fuels	3	Blood absorbs carbon monoxide more readily than oxygen, reducing the amount of oxygen being carried through the body. Carbon monoxide can produce tiredness and headaches. People with heart problems are particularly at risk	Indoors, localised[a]
Sulfur dioxide	Coal and oil burning power stations, mineral ore processing and chemical manufacture	3	Attacks the throat and lungs. People with breathing problems can suffer severe illness	Localised
Nitrogen dioxide	Fuel combustion	1	Affects the throat and lungs	Indoors, urban and regional
Volatile organic compounds	Motor vehicles, fuel combustion, solvent use		Some volatile organic compounds cause eye and skin irritation, headaches or nausea, while some are classed as carcinogens	Indoors, urban
Ozone	Formed from nitrogen oxides and hydrocarbons reacting together in sunny conditions. These chemicals are released by motor vehicles and industry		Ozone attacks the tissue of the throat and lungs	Urban and regional
Lead	Exhaust gases from motor vehicles that use leaded petrol, smelters, old, leaded paints	11	Particles containing lead in the air can enter the lungs. The lead can then be absorbed into the blood stream. Over a period lead can affect the nervous system and the body's ability to produce blood	Localised, urban
Particles (PM_{10})	Motor vehicles, domestic fires, burning of plant materials, bushfires, indoor activities	7	May cause breathing difficulties and worsen respiratory diseases. Some particles contain cancer-producing materials	Indoors, urban, regional and localised
Fluoride	Coal combustion and aluminium processing		Vegetation damage, fluorosis of teeth of cattle	Localised

a. Localised refers to a specific agricultural activity, industrial activity or location.

Source Manins et al (2001)

Particulate matter

Suspended particulate matters (SPM) are considered to be the main cause of the excess mortality observed during the London and New York smog episodes of the 1950s and 1960s. 'Excess mortality for exposures to these pollutants is estimated to be one in 10 000 for current levels of PM_{10} in Los Angeles (Mage and Zali (eds) 1992).

Three pioneering United States studies (Dockery et al 1993; Pope et al 1995a and 1995b) found strong associations between PM_{10} and mortality.

The Six Cities study (Dockery et al 1993) found that each additional 10 $\mu g/m^3$ of fine particles ($PM_{2.5}$) pollution increased mortality by 14 per cent. This is seven times greater than the immediate short-term effects observed in Sydney (Robinson 1999).

Pope et al (1995b) conducted another cohort study of over 500 000 adults in 151 cities to examine the mortality effects of long term or chronic exposure to particulate air pollution. The results suggest that a 10 $\mu g/m^3$ increase in average PM_{10} exposure was associated with an increase in daily mortality of 3 per cent or more. The strongest association was with cardiopulmonary disease and lung cancer deaths with only small, insignificant associations with other causes.

More recent research from the United States has found that daily mortality is significantly linked to PM_{10} and $PM_{2.5}$ pollution. Generally, particles pollution is known to have significant health impacts. These include exacerbation of asthma and increased risk of premature mortality—especially in the very young and the elderly. These studies showed that people living in less polluted cities live longer than those living in more polluted cities. Subsequent reanalysis of these studies, published by the Health Effects Institute (Krewski et al 2000) confirmed these earlier findings.

Ozone

Ozone is a severe irritant that is responsible for the choking, coughing, and stinging eyes associated with smog. It damages lung tissue, aggravates respiratory disease, and makes people more susceptible to respiratory infections. Children are especially vulnerable to ozone's harmful effects, as are adults with existing diseases, such as respiratory problems. Even otherwise healthy individuals may experience impaired health from breathing ozone-polluted air.

Thurston et al (2001) found that there is a possible association between brief ozone exposure episodes and increased mortality—particularly in the elderly. Ozone can increase the sensitivity of the airways to allergic triggers in people with asthma (Holz et al 2002 and Schelegle et al 2003). However, the concurrence of high ozone levels with hot weather makes it difficult to separate

the effect of heat from that of ozone on mortality (Kjellstrom et al 2002). At levels of 200–400 μg/m^3 for a one-hour average, its health effects include:

- lung inflammation
- reduced pulmonary function
- decreased resistance to pulmonary infections.

However, a WHO working group (2003) accepted the evidence that—in the short-term and at low ozone concentrations—ozone can increase mortality and respiratory morbidity.

Several studies conducted in Mexico City demonstrated the association of peak daily ozone concentration with respiratory health. A study conducted among children reported both acute and subacute effects of ozone on lung function (Castillejos et al 1992). Another study conducted among preschool children found an increase in school absenteeism for respiratory illnesses among those exposed to higher ozone concentrations (Romieu et al 1992).

Asthmatic children may be more susceptible than others to the effects of ozone exposure. Studies conducted in Mexico City have demonstrated that asthma-related emergency department visits increased by 43 per cent with an increase of 50 parts per billion in the daily one-hour maximum ozone level, with a one-day lag (Romieu et al 1995).

Morgan et al (1998b) found that, between 1989 and 1993, an increase in the daily maximum one-hour ozone concentration in Sydney was associated with a 2.45 per cent increase in heart disease admissions among people 65 years and older. Another study of daily mortality in the Brisbane region (Simpson et al 1997) indicated that ozone levels were significantly associated with total daily mortality when maximum daily ozone levels were approximately 240 μg/m^3.

Nitrogen oxides

The smog haze of polluted cities is caused by nitrogen dioxide. This leads to respiratory problems in sensitive individuals—particularly asthmatics and young children. Choi et al (1997) reported that an interaction between air pollution—especially nitrogen dioxide and high temperature—may synergistically increase lung cancer mortality rates. The study also found that the interaction between nitrogen dioxide and temperature explained regional differences in age-adjusted lung cancer rates. Nitrogen dioxide may:

- increase susceptibility to infection
- worsen asthma symptoms
- act with other pollutants such as sulfur dioxide to impair lung function.

A 1998 study reported on the health effects on 60 healthy Beijing children aged 9–11 years exposed to nitrogen dioxide for two months. It reported a negative correlation between peak expiratory flow rates and nitrogen dioxide

concentration (Xu Xiping et al 1998). The results indicate that increased nitrogen dioxide levels could affect children's respiratory function, aggravate air duct blocking and subsequently reduce peak expiratory flow rates. Long-term exposure to 50-100 $\mu g/m^3$ nitrogen dioxide may significantly affect children's respiratory and immunity systems. It may also have similar effects on sensitive adults.

Morgan et al (1998b) conducted a time series analysis of daily hospital admissions and outdoor air pollutants in Sydney between 1990 and 1994. An increase in the daily maximum one-hour concentration of nitrogen dioxide—from the 10^{th} to the 90^{th} percentile—was associated with increases in childhood asthma and chronic obstructive pulmonary disease admissions. They increased by 5.29 per cent and 4.6 per cent respectively.

Several studies have examined the relationship between respiratory symptoms and distance from roads with heavy traffic. The studies showed that the prevalence of respiratory symptoms, such as chronic cough and wheezing, was higher in residents who lived near to roads (Nitta et al 1993; Ono et al 1990). When there were no indoor nitrogen dioxide sources—except for gas cooking stoves—nitrogen dioxide levels were attributable primarily to automobile exhaust (Nakai et al 1995).

Sulfur dioxide

Sulfur dioxide exposure can have both short-term and long-term health effects. At excessive levels it can attack the respiratory tract directly, aggravating pre-existing lower airway inflammatory conditions and increasing susceptibility to respiratory tract infection. In heavily industrialised areas, sulfur dioxide combined with particles can increase the incidence of chronic lung disease—for example, chronic bronchitis—especially in children. An individual's response to sulfur dioxide exposure depends on the concentration and duration of exposure. Studies of human subjects have indicated that minimum concentrations of sulfur dioxide can cause adverse health effects. These studies were performed on asthmatics—among the most sensitive to sulfur dioxide. Subjects were placed in an exposure chamber for approximately 15 minutes and their responses to sulfur dioxide were measured both at rest and under exercise conditions.

Reist et al (1998) found that sulfur dioxide could cause lung injury—both by enhancing proteolysis and by creating new antigens that could trigger an immune response.

Carbon monoxide

Carbon monoxide reduces the amount of oxygen carried by the blood to body tissue. High concentrations may cause permanent damage to the central nervous and cardiovascular systems. Traffic-congested areas can generate

carbon monoxide levels as high as 20 to 30 mg/m^3. This leads to carboxyhaemoglobin levels of three per cent, 'which produces cardiovascular and neuro-behavioural effects and seriously aggravates the condition of individuals with ischaemic heart disease' (Mage and Zali (eds) 1992).

In Cairo, carbon monoxide concentrations greater than the WHO Guidelines for Air Quality values were recorded in streets having moderate-to-heavy traffic densities in residential areas and in the city centre (Nasralla 1997). These concentrations resulted in high levels—sometimes more than 10 per cent of carboxyhaemoglobin—in the blood of traffic policemen. This study also found a significant direct relationship between ischaemic heart disease and carboxyhaemoglobin levels in Cairo traffic policemen (Salem 1990).

Benzene

Animal studies have shown that benzene exposure can lead to leukaemia through damage to genetic material in cells. The only epidemiological studies of the health effects of benzene in Australia, as overseas, have been of people exposed to benzene in the workplace (Peach 1997). The current understanding of the health effects of benzene is 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 exposure are haematotoxicity, genotoxicity and carcinogenicity (Morgan and Jalaludin, 2001).

According to Duarte-Davidson et al (2001), the major health risk associated with low concentrations of exposure to benzene has been shown to be leukaemia, in particular acute non-lymphocytic leukaemia. The lowest concentration of exposure at which an increased incidence of acute non-lymphocytic leukaemia among occupationally exposed workers has been reliably detected has been estimated to be in the range of 32–80 μg/m3. The authors conclude that, overall, the evidence from human studies suggests that any risk of leukaemia at exposure concentrations three orders of magnitude less than the occupational lowest observed effect level—is likely to be exceedingly small and probably not detectable with current methods (Duarte-Davidson et al 2001).

The United Kingdom Department of the Environment's expert panel on air quality standards was unable to define an absolutely safe exposure level for benzene (United Kingdom Department of the Environment 1994). For practical purposes the panel believed that an annual average concentration of five parts per billion could be proposed. This was based on data from cohort studies of workers exposed to benzene.

REVIEW OF KEY INTERNATIONAL AND AUSTRALIAN AIR POLLUTION STUDIES

This section reviews the existing Australian and overseas literature on the health effects of air pollution. The researchers used several different study designs in this area:

- time series/correlational analysis
- cohort studies
- cross-sectional analysis
- case control studies
- panel studies
- chamber studies.

The review of existing literature shows that studies conducted in Australia were limited in number and coverage till the late 1990s. But overseas studies are extensive in both size and coverage. Most of the major Australian studies available are included in this review. However, for reasons of space and scope, not all major overseas studies have been covered. Also, there are several extensive literature reviews (Holland et al 1979; Romieu 1992; Flachsbart 1992; Peach 1997; Goldsmith and Kobzik 1999; Denison et al 2000; Morgan and Jalaludin 2001; Brunekreef and Holgate 2002).

A review of the literature suggests that epidemiological studies formed a major part of the research on air quality and health in the 1990s. However, in a more recent trend, meta-analysis and chamber studies have become quite popular with researchers. These analyses are a step forward from conventional epidemiological studies.

Epidemiological studies focus on the evidence of association between the incidence of diseases—called the effects—and risk factors and the level of pollutants in the air. These studies do not address the issue of causality and mechanisms of the effect.

Chamber studies are designed to find the biological mechanisms by which air pollutants may cause increased morbidity and mortality as established by epidemiological studies. Extensive research using chamber studies is currently underway in different parts of the world.

Gene Glass proposed a new method, meta-analysis, to integrate and summarise the findings from a body of research in 1976. Meta-analysis refers to the analysis of analyses. It is the statistical analysis of a large collection of analysis results from individual studies for the purpose of integrating the findings (Glass 1976, p. 3).

Meta-analysis can help provide new insights, explain differences between results of similar studies, or determine useful directions of research. The

appropriate application of meta-analysis in environmental economics provides several benefits. They include:

- a better use of existing information and knowledge
- removal of some of the subjectivity from analysis and forecasting
- greater clarity as to where future efforts in environmental economic analysis can be focused most effectively.

Early studies

Between 1930 and 1950, there were many studies examining exposure to high peaks of air pollutant concentrations in many cities in the West. These studies consistently found a strong association between high air pollution exposure and excess mortality rates. Goldsmith (1990) called this the 'era of air pollution disasters'. He cited incidents such as the 1952 London smog, pollution episodes in the Meuse Valley of Belgium in 1931, and Donora in Pennsylvania in 1948. Stricter regulations and monitoring in the aftermath of such tragedies significantly decreased the number of deaths attributable to air pollutant exposures.

In the 1952 London air pollution episode, it was estimated that 4000 extra deaths occurred as a result of a smog consisting largely of high concentrations of sulfur dioxide and particulate matter (Brimblecombe 1987). Also, in the Donora episode in 1948, about 43 per cent of the total population was affected by symptoms including headache, eye irritation, breathing difficulties and vomiting.

Analysis of the London episode showed that the elderly—especially those suffering from pre-existing cardiorespiratory disorders—and the very young were at greatest risk. Later studies demonstrated a decline in urban levels of chronic bronchitis as concentrations of air pollutants fell (Chinn et al 1981). A recent reanalysis of the 1952 London episode data indicated that the estimated excess death toll due to increased concentrations of air pollutants was about 12 000 (Bell and Davis 2001).

Time series analysis

A new database of epidemiological studies emerged in the late 1980s and 1990s. This database of time series studies was developed first in the United States and later in Europe and other areas (Katsouyanni et al 1995; Schwartz et al 1996; Anderson et al 1996; Touloumi et al 1996; Burnett et al 1998a and b).

In essence, the time series approach takes the day as the unit of analysis and routinely collected pollution data is then analysed for adverse health effects over a period of time. These studies attempt to correlate daily changes in pollution—average concentrations of pollutants—with changes in health—for example, hospital admissions—or death rates. At the same time, the analysis

considers carefully confounding factors such as season, temperature and day of the week.

The results of these time series studies have been significantly consistent and have withstood critical examination well (Samet et al 1995). The disadvantage is that the time series approach cannot establish the causal nature of the associations demonstrated. However, detailed examination of the data—and application of the usual statistical tests for likelihood of causality—generally supported the findings.

The time series studies showed epidemiological evidence that indicates a positive association between daily mortality from:

- respiratory and cardiovascular causes and exposure to ozone—Touloumi et al 1997; Sartor et al 1997; Borja-Arburto et al 1997; Simpson et al 1997
- nitrogen dioxide—United Kingdom Committee on the Medical Effects of Air Pollutants 1998; Streeton 1997; Bascom et al 1996
- fine particles: total suspended particles, PM_{10}, and $PM_{2.5}$—Samet et al 1997 and 1998; Wordley et al 1997; United Kingdom Committee on the Medical Effects of Air Pollutants 1998; Morgan et al 1998a; Simpson et al. 1997; Denison et al 2000.

The meta-analysis of the Air Pollution and Health: European Approach studies used a variety of measures of particles. It revealed a 0.4 per cent increase in daily mortality per 10 $\mu g/m^3$ increase in PM_{10}. It has been estimated that in Great Britain there are 8100 premature deaths per year that can be attributed to exposure to PM_{10} (United Kingdom Committee on the Medical Effects of Air Pollutants 1998).

In 1998, Prescott et al published the results of a fourteen-and-a-half year time series study on urban air pollution and health in Edinburgh. The study showed that there were statistically significant associations—after adjustment for confounding factors—between particulate air pollution and adverse health effects in the elderly—that is, people aged over 65 years. Prescott et al (1998) concluded that a $10mg/m^3$ increment in black smoke—another index of particulate pollution—was associated with an approximate four per cent increment in mortality from respiratory diseases. The study also found a five per cent increment in emergency hospital admissions for cardiovascular disease such as heart attacks.

Many studies have shown adverse health effects—both mortality and morbidity—associated with carbon monoxide exposure. Several studies (Sheppard et al 1999; Schwartz et al 1999; Morris and Naumova 1998; Yang et al 1998) have found a statistically significant association between low-level ambient concentrations of carbon monoxide—at the daily one-hour maximum—and all age group hospital admissions for cardiovascular disease.

Morgan et al (1998b) published the results of a time series analysis of counts of daily hospital admissions and outdoor air pollutants between 1989 and 1993 in Sydney. They showed that increases in heart disease, chronic obstructive pulmonary disease and childhood asthma were associated with increased ambient nitrogen dioxide levels. An increase in the daily maximum one-hour concentration of nitrogen dioxide was associated with an increase of 5.3 per cent in childhood asthma admissions and 4.6 per cent in chronic obstructive pulmonary disease admissions. A similar increase in daily maximum one-hour particle concentration was associated with a three per cent increase in chronic obstructive pulmonary disease admissions.

Another study on daily mortality in the Brisbane region (Simpson et al 1997) suggested that ozone levels were significantly associated with total daily mortality. At that time, maximum daily ozone levels were approximately 240 $\mu g/m^3$. This study indicated that the associations found between total daily mortality and particle levels in the United States and other European countries might also be applicable in Brisbane. The evidence of interaction between the ozone effects—mainly in summer—and either particles, sulfur dioxide, or nitrogen dioxide was insignificant in this study. The associations between ozone and daily mortality were significant only for individuals who were older than 65 years. Positive associations between ozone and daily mortality were also found with cardiovascular disease categories. The regression coefficients, when significant, were higher than those for total mortality. The study results indicated a possible threshold for ozone levels, but no threshold for particle levels.

The Melbourne Mortality Study (Denison et al 2000) attempted to identify the impacts of ambient air pollution on daily mortality in Melbourne between 1991 and 1996. This study found positive associations between daily mortality and major pollutants like nitrogen dioxide, carbon monoxide, ozone, and fine particles—PM_{10} and $PM_{2.5}$. The latter was estimated from backscatter of particles data—a proxy measure of particulate matters.

The study results showed that hospital admissions for children with asthma were affected by air pollution—in particular fine particles and ozone. A 10 $\mu g/m^3$ increase in daily average $PM_{2.5}$ was associated with a 1.4 per cent increase in daily all-cause mortality. This result is similar to that observed in the Six Cities Study (Schwartz et al 1996) and the Wordley et al (1997) study for Birmingham in the United Kingdom. Admissions for the elderly with existing heart disease were strongly associated with carbon monoxide. The observed associations were seen to be stronger during warm seasons for all pollutants. The strongest, or most statistically significant, associations were observed for ozone and nitrogen dioxide. Their effects—in terms of size and significance—varied depending on the methods used to control for the highly confounding effects of weather in the analysis. The methods used included generalised additive models and trigonometric models. However, the effects associated

with carbon monoxide, nitrogen dioxide and particles were difficult to separate from each other (Denison et al 2000).

A recent time series study (Petroeschevsky et al 2001) examined the relationship between air pollution and hospital admissions in Brisbane between 1987 and 1994. They used the Air Pollution and Health: European Approach protocol approach to analyse the effects of four pollutants—ozone, sulfur dioxide, nitrogen dioxide, and particles measured by backscatter particles. These four pollutants were found to have the greatest impact on respiratory health. Controlling for weather conditions—such as high temperature and humidity—in the model had no impact on the size or significance of the effects. The study found strong, positive associations between ozone, particles, sulfur dioxide and hospital admissions. Seasonal associations were found for nitrogen dioxide.

However, the most significant finding of Petroeschevsky et al (2001) was the strong and consistent relationship observed between ozone and admissions for asthma. Ozone was associated with between four and 10 per cent of asthma admissions, depending on the age group considered. However, they did not find an increasing linear dose-response relationship between PM_{10} and hospital admissions (Pope et al 1995b; Wordley et al 1997). This finding contrasts with Schwartz (1997), who found significant positive associations between PM_{10} and cardiovascular admissions in Tucson in the United States.

Cohort studies

Cohort studies carefully follow clearly defined populations—where detailed information is known about individuals—over a period of time. They provide the most complete estimates of the numbers of deaths, and the average reduction in lifespan, attributable to air pollution. Such studies include not only those whose deaths were advanced by recent exposure to air pollution, but also those who died from chronic disease caused by long-term exposure. These studies are best suited to investigate the common health effects of air quality that will accrue in sufficiently large numbers of population over a reasonably short period. Examples include respiratory illness, cardiac symptoms and asthma attacks. Unfortunately, these studies are very expensive and time consuming.

Henry et al (1991b) investigated the impact of maximal daily sulfur dioxide and nitrogen oxide concentrations in the air on asthmatic children in selected Australian towns near power stations. But the study results did not show any association between pollutants and asthma symptoms in children.

Two important cohort studies (Dockery et al 1993; Pope et al 1995a) were based on observations from the late 1970s to the late 1980s. The results of these studies suggested an association between exposure to fine particulate matter in the air and mortality in selected United States cities. The Six Cities study (Dockery et al 1993) found that each additional 10 $\mu g/m^3$ of fine particle ($PM_{2.5}$) pollution increased mortality by 14 per cent.

However, these two studies did not sufficiently account for confounding socioeconomic factors. In these studies, particle levels were measured over only a few years. So the studies cannot consider different exposure durations or differences in historical levels of pollution in cities.

Another study (Abbey et al 1999) reported an observed strong association between PM_{10} and non-malignant respiratory deaths in men and women, and on lung cancer mortality in non-smoking non-Hispanic, white males. In this cohort study, researchers used data from 348 monitoring stations to estimate monthly ambient concentrations of PM_{10}, ozone, sulfur dioxide and nitrogen dioxide. They gathered mortality data from 1977 until 1992.

The Health Effects Institute conducted a reanalysis of these studies (Krewski et al 2000). The reanalysis examined a wider range of factors, such as poverty and unemployment, and found no marked impact on the all-cause mortality results. Level of education was a strong effect modifier. The effect of particles on mortality decreased with increasing education. Regional correlations were found between mortality rates in cities. This suggested that unknown factors were also affecting mortality.

A more recent study in selected United States cities (Pope et al 2002) found that lung cancer mortality and non-malignant cardiopulmonary deaths were more consistent with the mortality effect of PM_{10}. A European study conducted in the Netherlands (Brunekreef and Holgate 2002) supported the United States evidence and suggested that traffic-related air pollution is associated with cardiorespiratory deaths.

Cross-sectional studies

Cross-sectional studies compare the frequencies of ill health of people in various localities. Some studies may involve relatively small numbers of people, but with more sophisticated methods of measuring personal exposure and health effects. Others are not strictly epidemiological but may help to corroborate, or supplement, the findings from epidemiological investigations.

Most of the cross-sectional studies investigated the health effects of children in different localities. Ware et al (1986) and Dockery et al (1989) reported a positive association between respiratory symptoms among United States metropolitan school children and air pollutants including:

- particles—total suspended particles, PM_{10} and $PM_{2.5}$
- sulfur dioxide
- nitrogen dioxide
- ozone
- ferrous sulphate.

Studies conducted on school children in England (Brabin et al 1994) and Italy (Corbo et al 1993; Forastiere et al 1994) suggested a greater prevalence of respiratory symptoms in children living in polluted and/or industrialised areas.

Halliday et al (1993) and Henry et al (1991a) examined two groups of Australian schoolchildren: those who lived near power stations and those who did not. They investigated the prevalence of respiratory symptoms, lung function and bronchial reactivity. Both studies found a greater incidence of colds and flu among children living near power stations (as cited in Peach 1997).

Studies of children living in 24 United States and Canadian communities reported significant associations between exposure to fine particles and their acidity and lung function. In addition, those children exposed to fine particles had symptoms of bronchitis but not asthma (Brunekreef and Holgate 2002).

Panel studies

Panel studies are short-term studies conducted on volunteers on a daily or weekly basis. They provide data on health endpoints—for example, respiratory or cardiovascular symptoms—and objective measures of lung or cardiac function. These studies are usually large and collaborative, and generally take a multi-country analytical approach.

In Europe, the Air Pollution and Health: European Approach studies are leading panel studies. The Air Pollution and Health: European Approach–1 (APHEA–1) studies were based on data from the 1980s to the early 1990s, whereas Air Pollution and Health: European Approach–2 (APHEA–2) studies used recent data. The Air Pollution and Health: European Approach–2 Mortality study (Atkinson et al 2001) covered a population of more than 43 million people living in 29 European cities, for more than five years in the first half of the 1990s. The combined effect estimate of this study—from 21 cities' data—showed that all-cause daily mortality increased by 0.6 per cent for each 10 mg/m^3 increase in PM_{10}, with some heterogeneity.

The National Mortality, Morbidity and Air Pollution Studies (2000) investigated the pollution effect in the 20 largest metropolitan areas in the United States. It studied 50 million people between 1987 and 1994. Their finding was quite close to the APHEA–2—an all-cause mortality increase of 0.5 per cent for each 10mg/m^3 increase in PM_{10}. Results from a panel study conducted in Punchucavi, Chile, indicated a five per cent increase in coughing among children with chronic respiratory symptoms. This increase was associated with an increase of 30 μg/m^3 in the 24-hour average levels of PM_{10} (Sanchez-Cortez 1997).

Case control studies

In this type of study, a group of subjects with a particular health effect—for example, asthma patients—is compared with a group of subjects without any health effect. Both groups are exposed to the same air pollution levels and pollutant effects are investigated as an exposure–response function.

Nyberg et al (2000) provided evidence of a link between air pollution—particularly from traffic—and lung cancer. The literature shows mixed results for asthma. Studies conducted in Mexico among asthmatic children have documented an increase in respiratory symptoms and a decrease in lung function related to PM_{10} exposure. The results suggested that an increase of 10 $\mu g/m^3$ in PM_{10} levels was associated with a four per cent increase in minor respiratory symptoms, and a 0.35 per cent decrease in peak expiratory flow rate (Romieu et al 1996). It is important to note that the results of this study suggest a synergistic effect of PM_{10} and ozone exposure on the incidence of lower respiratory tract symptoms in these children.

A study of the morbidity of allergic rhinitis based on Japan National Health Insurance records showed a three-fold increase in the rate of allergic rhinitis over 10 years (Miyao et al 1993). Additionally, results suggested possible correlations between the morbidity of allergic rhinitis and the mean yearly levels of suspended particulate matter and nitrogen dioxide.

Chamber studies

Chamber studies analyse the mechanism by which exposure to individual air pollutants affect human and animal health—for example, lung, cardiovascular, brain and stomach functions. They do not examine either the mixtures or temporal variation that occur in natural exposures.

A general finding is that individual air pollutants can exert their own specific individual toxic effects on the respiratory and cardiovascular systems. But ozone, nitrogen oxides and suspended particulate matter are all potent oxidants. Several studies have been conducted to explain the effects of exposure to ozone (Samet et al 2001; Schelegle et al 2003; Holz et al 2002). Diesel particles and ozone exposure have been found to increase the sensitisation to common allergens (Nel et al 1998).

A review of the chamber studies shows that the mechanism of ozone-induced health effect has been explained. But little is known about the dynamics of the long-term effects of nitrogen dioxide on human health. However, studies have found that short-term exposure to nitrogen dioxide can enhance airway responses to inhaled allergens in asthmatic individuals (Brunekreff and Holgate 2002).

Concerning particles—PM_{10}, $PM_{2.5}$ and total suspended particles—knowledge of the respiratory actions of particles has grown, although an understanding of how particles increase the risk of cardiovascular events is not clear.

Meta-analysis

A health impact assessment by Ostro and Chestnut (1998) estimated the health benefits of reducing particulate matter air pollution in the United States. The epidemiological studies included in the analysis used several alternative measures of particulate matter including total suspended particles, $PM_{2.5}$, and sulphate aerosols as well as PM_{10}. This study identified high, low and central estimates from selected epidemiological studies.

The United Kingdom Committee on the Medical Effects of Air Pollutants (1998) quantified the effects of air pollution on health in the United Kingdom. This study collected exposure–response estimates from a selection of European studies and estimates from the few available United Kingdom studies. Estimates for mortality were examined from three sources including an Air Pollution and Health: European Approach meta-analysis, a WHO meta-analysis, and a time series study in Birmingham by Wordley et al (1997). The Committee concluded that the WHO meta-analysis was appropriate for quantifying United Kingdom health effects.

The APHEA studies are a series of daily mortality studies and daily hospital studies conducted in various European cities using a common methodology. In the APHEA study meta-analysis (Touloumi et al 1997), results from four site studies—London, Paris, Athens and Barcelona—were included in the analysis. All four of these studies were also included in the mortality meta-analysis presented in the report (United Kingdom Committee on the Medical Effects of Air Pollutants 1998).

A report by Thurston and Ito (1999) included several meta-analyses of ozone's effect on a range of health outcomes. Results from five studies conducted in Canada and New York provide the data for the pooling of the estimates. The mean daily one-hour ozone concentration ranged from 32 to 75 parts per billion.

Künzli et al (2000) estimated the public health impact of traffic-related ambient air pollution in France, Switzerland and Austria. This study used long-term epidemiological estimates of the increase in risk for a specified change in ambient pollutant concentrations. Short-term effects were not considered as the authors thought this was inappropriate when they assessed the annual effect on mortality. In an earlier study, Künzli et al (1999) identified a range of morbidity and mortality health outcomes associated with particulate matter in Switzerland, France and Austria. Künzli et al (1999) combined selected exposure–response estimates into a pooled estimate using meta-analysis techniques.

Morgan and Jalaludin (2001) identified exposure–response assessment for the effects of the identified air pollutants in Sydney on various health endpoints. They followed Künzli's approach (Künzli et al 1999) and their assessment used exposure–response estimates derived from meta-analyses of several studies. Where available, meta-analysis estimates from the literature were used. However, because estimates were not available in the literature, researchers calculated combined estimates by variance-weighted methods using either a fixed effect or random effects model.

A report prepared for the New Zealand Ministry of Transport by Fisher et al (2002) estimated the health effects of motor vehicle pollution in New Zealand. This study used the European approach developed by Künzli et al (2000) and covered about 80 per cent of the total population. It concluded that overseas results were applicable, and the methodologies valid, for making an assessment in New Zealand. This study estimated that the total annual mortality for people aged over 30 using PM_{10} as the proxy indicator for all ambient air pollutants. The results indicated that annual traffic-related air pollution mortality was 'around 399'—almost double the annual traffic accident road toll.

A WHO task group (WHO 2004) prepared the most recent meta-analysis. In this study, a quantitative meta-analysis of peer-reviewed studies was conducted to obtain summary estimates for certain health effects linked to exposure to particulate matter and ozone. The data for these analyses came from a database of time series studies using data, both ecological and individual, for several European cities. The results of this analysis confirmed statistically significant relationships between particulate matter and ozone in ambient air with mortality.

MAJOR CURRENT RESEARCH ISSUES

Asthma and Air Pollution

For a long time researchers thought that air pollution could not cause asthma to develop in previously healthy people. Some recent experiments challenge this notion. Epidemiological and chamber studies show that a combination of air pollution and allergens could cause some cases of asthma. Most major ambient air pollutants have significant effects on lung function. However, other factors—including allergens, colds, cigarette smoking and exposure to fumes and dust—are probably more important in causing asthma attacks.

Recent research has implicated ozone, nitrogen dioxide and particulate matter—all pollutants formed from vehicle exhausts—as contributing factors to asthma-related hospital admissions and mortality. Human and animal exposure studies and laboratory-based studies have demonstrated that diesel particles, ozone and nitrogen dioxide induce an inflammatory response in the respiratory system thereby contributing to allergies (Salvi 2001; Barck et al 2002; Vagaggini et al 2002). A study by Davies et al (1993) showed that both ozone and nitrogen

dioxide damage the lining of the respiratory system. This damage allows substances to penetrate the lining of the respiratory system, impairs the operation of cilia and leads to the chemical cascade that brings on asthma attacks.

Associations have been observed between airborne particulate matter (PM_{10} and $PM_{2.5}$) and restricted activity days and the exacerbation of asthma and chronic obstructive pulmonary disease. Other clinical results show associations with decreases in lung function, increased use of asthma medication, decreased heart rate variability, and the triggering of myocardial infarction (Buckeridge et al 2002; von Klot et al 2002; Peters et al 2001). In 1994, Harvard researchers (Dockery and Pope 1994) reviewed the combined results of several studies. They concluded that each 10 $\mu g/m^3$ increase in PM_{10} was associated with an approximate three per cent increase in asthma attacks, bronchodilator use, and lower respiratory symptoms.

A recent study of patients suffering from severe asthma found significant associations between PM_{10} and ozone and asthma attacks (Desqueyroux et al 2002). Another recent study found that nitrogen oxides increased the risk of death in patients with more than one emergency room admission for asthma (Sunyer et al 2002). In this study, ozone was also found to increase the risk of death in asthmatic patients during spring and summer. Another review by Koren (1995) showed that ozone is associated with increased hospitalisation rates for asthma. However, it was difficult to separate the effects of ozone from those of other pollutants such as acid aerosols.

Air pollutants may not only increase the frequency and intensity of symptoms in allergic patients but may sensitise airways to airborne allergens in predisposed individuals (Bucchieri et al 2002). One possible mechanism is that air pollutants may adhere to the surface of pollen grains—thereby changing the morphology of these grains—and inducing airway inflammation (Glikson et al 1995; D'Amato, 2002). There is Australian evidence of both these effects, especially in Brisbane where fungi are also prominent aero-allergens (Rutherford et al 2000; Glikson et al 1995).

Abramson and Forbes (2001) conducted a case crossover study in Melbourne between 1994 and 1996. Their study aimed to estimate the association between ambient particulate or ozone levels and the risk of death or hospital admission for asthma. This study could not establish any significant relationship between air pollution and asthma-related death or morbidity. According to researchers, the likely explanation in this instance was that the study had insufficient statistical power—particularly for the examination of asthma deaths. It was also possible that the results were biased by unmeasured time varying confounding factors—such as activity patterns.

Children's health and air pollution

Researchers have identified several health effects of air pollution on both adults and children. However, because children's physiological responses to air pollution are quite different from that of adults, they are more susceptible to the effects of air pollution. Children differ from adults in their activities, their rate of breathing, their lung anatomy and physiology, and their organ maturity. For example, children take in more air per unit body weight at a given level of exertion than do adults. In a comparable activity children may take in 20–50 per cent more air, and hence more air pollution, than an adult. The air pollutants that are of special concern for children include airborne particles, carbon monoxide, ozone, nitrogen oxides, sulfur dioxide, and acid aerosols (Mathieu-Nolf 2002).

Children are more vulnerable to ambient air pollutants due to greater relative exposure. While adults typically spend 5–15 per cent of their time outdoors, children may spend more than 20 percent of their time outdoors. There are definite health benefits to having children participate in outdoor activities. However, scientific evidence suggests that air pollution exposures can injure children's lungs and other organs during their physiological growth period (Kleinman 2000).

Children are often outdoors during periods when air pollution is at its highest. This implies maximum exposures of developing lungs and other organs to air pollution. Air pollution can change the cells in the developing lung by damaging those that are most susceptible. This means the lung may not achieve its full growth and function as a child matures to adulthood.

The 10-year Children's Health Study (Gauderman et al 2002) is one of the most comprehensive studies to date of the long-term effects of smog on children. This University of Southern California led study has tracked major pollutant levels and monitored the respiratory health of more than 6000 children in a dozen Southern Californian communities since 1993. Its findings examine the development of lung function in children and show that lung function of children in polluted areas lags behind that of children in areas with cleaner air. The study found that, for children with asthma, their symptoms increased when they were exposed to higher levels of ozone and particulate matter. Similarly, children without pre-existing symptoms were found to be more likely to develop asthma when they played three or more competitive sports and lived in communities with higher levels of ozone and particulate matter.

Another study by McConnell et al (2002) which is also based on the University of Southern California Study, showed that children that play sports in areas with high levels of air pollution are three to four times more likely to develop asthma than other children.

A cross-sectional survey of children's health was undertaken in New South Wales between October and December 1993 to investigate the relationship

between outdoor air pollution and the respiratory health of children aged 8–10 years (Lewis et al 1998). This cross-sectional study of primary school children showed an important association between relatively low levels of particulate air pollution and respiratory symptoms. This is consistent with similar cross-sectional studies from other countries.

There is a higher prevalence of respiratory symptoms among children living near motorways or freeways, and also a higher prevalence of chronic coughing, wheezing, asthma attacks and rhinitis in areas with higher truck traffic density (Oosterlee et al 1996; van Vliet et al 1997; van Der See et al 1999; Venn et al 2001; Lin et al 2002). Other studies have also found a strong association between decreased lung function of children living near motorways and increased air pollution levels from truck and motor vehicle traffic (Brunekreef et al 1997; Nakai et al 1999). Findings from the international collaborative study on the impact of Traffic-Related Air Pollution on Childhood Asthma (commonly known as TRAPCA) confirmed the association between traffic-related air pollution and coughing in children under two years of age (Gehring et al 2002).

A study of asthmatic children in Taiwan who were exposed to high levels of traffic-related air pollution—identified using carbon monoxide and nitrogen dioxide as marker compounds—reported more respiratory symptoms than children with lower exposures. The incidence of asthma in children has also been associated with traffic-related air pollutants such as nitrogen dioxide and $PM_{2.5}$, particularly for children under one year of age (Brauer et al 2002; Zmirou et al 2002).

The role of ambient air pollution as a possible risk factor for Sudden Infant Death Syndrome has received little attention and is controversial (Dales et al 2004). Some studies have found an association between air pollution and Sudden Infant Death Syndrome—for example, Woodruff et al (1997) found an association with PM_{10} exposure. However, this study did not adjust for birth weight or income, and gaseous pollutants were not considered.

There are now many identified and well-publicised risk factors for Sudden Infant Death Syndrome. Known risk factors include the winter season, low socioeconomic status, environmental tobacco smoke, and the prone position (Dales et al 2004). However, Dales et al found that Sudden Infant Death Syndrome was associated with air pollution—specifically the effects of sulfur dioxide and nitrogen dioxide. These effects seemed to be independent of socio-demographic factors, temporal trends, and weather. The results of their study also indicated that the effects of carbon monoxide, ozone and particulate matter were not significant. However, one weakness of the study was the lack of daily data on $PM_{2.5}$ (Dales et al 2004).

Carbon monoxide may also have prenatal effects. Pregnant women who were exposed to high levels of ambient carbon monoxide—five to six parts per million—were at increased risk of having low birth-weight babies. Babies

exposed to carbon monoxide during the maturation of their organs may suffer permanent changes to those organs.

However, nitrogen dioxide seems to be the most important nitrogen compound known to have acute adverse health effects on children. It causes significant lung irritation and inflammation. Nitrogen dioxide differs from ozone in that it suppresses the immune system to a much greater degree. Epidemiological studies have shown that children exposed to high levels of ambient nitrogen dioxide may be at increased risk of respiratory infections (Kleinman 2000). These studies suggest that children with asthma are more likely than children without asthma to have reduced lung function and symptoms of respiratory irritation—such as coughing and sore throats—when outdoor average nitrogen dioxide concentrations exceed about 0.02 parts per million.

Some studies have also suggested that children younger than five may be more severely affected by nitrogen dioxide than older children. Several epidemiological studies have suggested that, for children, the most important effect of ambient exposure to nitrogen dioxide might be increased susceptibility to respiratory infections and increased severity of responses to inhaled allergens.

There is a strong body of evidence that some children are also adversely affected by particulate matter. A review of health effects literature by the American Thoracic Society's Environmental and Occupational Health Assembly (American Thoracic Society 1996) reported that daily fluctuations in PM_{10} levels were related to:

- acute respiratory hospital admissions in children
- school and kindergarten absences
- decreases in peak lung air flow rates in normal children
- increased medication use in children and adults with asthma.

Thresholds in air pollutant levels

Recent research evidence suggests that threshold levels of air pollutants—in terms of their impact on health—are difficult to measure because adverse health effects can happen at very low levels. The WHO working group (2003) accepted that, even at low ozone concentrations, there are short-term ozone effects on mortality and respiratory morbidity. Particulate matter has been identified as having both premature mortality and morbidity effects at very low levels in various parts of the world with differing climates, socioeconomic status and pollution levels (Fisher et al 2002).

In the Horstman et al (1995) clinical study, asthmatic subjects exposed to ozone levels of 0.16 parts per million were found to be significantly more sensitive to ozone exposure than non-asthmatics. This level exceeds the current United States 24-hour average standard of 0.12 parts per million. However, a review by

Lebowitz (1996) indicates that the lowest effect level for asthma can occur with levels as low as 0.08 parts per million.

Koren (1995) summary of clinical studies indicated that asthmatics may develop significant increases in airway resistance with brief exposures to sulfur dioxide at concentrations as low as 0.25 parts per million. Gong et al (2003) studied asthmatics exposed to sulfur dioxide while exercising. They found increasing symptoms and lung function responses in concentrations above 0.5 parts per million. However, Lebowitz (1996) reports that the lowest effect level from two epidemiological studies was 200 $\mu g/m^3$—or 0.08 parts per million.

Extent of loss of life

Researchers have attempted to estimate the public health impact of air pollution on the basis of both cohort studies and time series studies. On the basis of the cohort study by Pope et al (1995b), Brunekreef (1997) estimated that current air pollution exposure levels in the Netherlands may lead to an average reduction in longevity of one year. Studies conducted in the United States report suggest slightly larger effects: perhaps 2.5 to 3.1 years are lost by people living in the most polluted cities compared with people not exposed to urban air pollution:

> … [these] estimates of population average life lost from the pollution are probably a worst-case scenario for the United States. They use relatively large estimates of excess risk from chronic exposure to pollution from the recent prospective cohort studies of adults and the postneonatal infant mortality study. They assume that the risk effects are cumulative for all or a large part of persons' lives, and they assume lifelong residency in one of the most polluted U.S. cities. Loss of life estimates due to pollution exposure of 1-3 years for lifelong residents of highly polluted cities, however, is not unreasonable, especially in some of the more polluted cities in the world. (Pope 2000)

According to Künzli et al (1999), assuming an average reduction across the whole population presumes that air pollution has a similar impact for all members of the population. Each person therefore has a very small reduction in life expectancy. That is, it is assuming that air pollution constitutes a worsening risk factor for the whole population and not only for a specific subgroup.

Alternatively, the impact of air pollution on the life expectancy may be estimated from the cases of premature death—that is, of real but anonymous cases. This approach assumes that there are susceptible subgroups that experience premature death due to air pollution. Conceptually, the interpretation of epidemiological findings favour the latter scenario:

> The disposition for certain diseases is not randomly distributed, but shows significant differences instead. [For example] the epidemiological findings show that the impact of air pollution on respiratory mortality is higher than on total mortality, or that the increase in hospital admissions also depends on the disease. Thus, according to susceptibility status, effect estimates may be different. Under the whole population scenario effect estimates are assumed to be identical for the whole population. In theory there are arguments in favour

> for both scenarios. The discussion on [which is] the more realistic scenario is still in progress. (Künzli et al 1999).

For the purposes of this study, it is the extent of life lost in the susceptible subgroups that is relevant. In general, studies show that pollution-induced premature deaths occur mostly in people over the age of 60. For example, Künzli et al (1999) show that the average age of people dying from cancer, cardiovascular and respiratory diseases in Switzerland is 80 years, compared with 71.9 for other non-violent causes. Künzli (2002) notes the calculation of annual years of life lost are sensitive to the age structure of the premature deaths and that:

> So far, published studies did not provide estimates of the years of life lost nor the age structure of the premature death, thus, indirect estimates and assumptions must be relied upon, using life tables of the population.

Quantifying the health effect of air pollution

In developed countries quantifying the health effects of air pollution is an increasingly important public policy issue. However, the issue is complex and involves both methodological issues and considerable uncertainty.

A WHO working group (2000a) examined methodological issues related to the health impact assessment of air pollution. The group concluded that cohort studies provide the most complete estimates of both the number of deaths attributable to air pollution and the average reduction in life span associated with air pollution exposure. However, the group agreed that time series studies would continue to play an important role in understanding exposure–response relationships. It also identified sensitivity analysis as having a critical role in bringing across the uncertainty of the impact estimates.

Epidemiological studies have quantified various health effects—for example, eye irritation, cough, throat irritation, chest discomfort, headache and minor restricted activity days (Utell and Frampton 2000; Künzli 2000; Hall et al 1994). These studies also found several unquantified health effects. These included the development of chronic respiratory diseases—such as bronchitis, asthma and emphysema—and gradual decreases in lung function. However, cohort studies have now quantified some of these health effects (Pope et al 2002).

The United Kingdom Department of Health's Committee on the Medical Effects of Air Pollutants worked on quantifying the health effects of air pollution in the United Kingdom (United Kingdom Committee on the Medical Effects of Air Pollution 1998). It noted that the effects on mortality had not been fully quantified. The Committee concluded that the quantitative impact of pollution could vary between countries with different cultures and lifestyles and that this variation could not be predicted. Accordingly, it developed a range of estimates and commented on its confidence in them. The uncertainties in this process resulted in a wide range of estimates—from £2600 to £1.4 million—to avoid a premature air pollution-related death.

WHO (1999c) estimated the health costs of transport-related air pollution in Austria, France and Switzerland at 27 billion Euros in 1996. This study followed the willingness-to-pay approach, which included the material costs as well as intangible cost elements—such as pain, suffering and the loss of quality of life. Economic approaches to valuing life are discussed in more detail in chapter 5.

CONCLUDING REMARKS

This chapter discusses in detail the evidence of the serious health effects of air pollution. The existing literature shows that long-term exposures have more adverse health effects and hence higher cost implications for the community. Researchers have identified the major air pollutants with adverse health effects and their sources. However, the exact nature of the association between air pollutants and the exposed population—given different attributes such as age and existing health conditions—is still a matter of further research.

Most of these air pollutants are present in the air at the same time and researchers are yet to find a way to isolate their individual impacts on human health. Chamber studies have made some progress on this issue, notably by identifying the short-term health effects of ozone.

Another unresolved issue is the minimum threshold levels of air pollutants, beyond which health effects occur. The obvious questions are: what is the threshold level of each air pollutant and how is that level calculated? Any calculation must allow for different climates and different levels of air pollution across countries. The WHO has presented guidelines on the likely threshold levels (WHO 2000b) for the major air pollutants, but these may not be universally applicable.

Finally, approaches to quantifying health effects pose a major challenge for researchers. However, before researchers can estimate the health effects of air pollution, they must answer two important questions. They are: how do we quantify and value the loss of life?

CHAPTER 5 HEALTH EFFECTS ASSESSMENT APPROACH

This chapter discusses the overall approach used to quantify and value motor vehicle emission health effects in this study. As discussed in chapter 3, data limitations preclude estimation of the health impact of emissions from other transport modes.

The approach follows the European concept often referred to as the 'dose–response' or 'impact pathway' (AEA Technology 2002; World Health Organization (WHO) 2000a).

Primarily the approach involves the following steps:

- assessing and quantifying emissions from the pollutant source—in this case from motorised vehicles
- assessing the resulting air pollution concentrations in the surrounding area
- assessing the receptor (people) weighted pollution increases
- deriving exposure–response functions that link health cases—such as respiratory and cardiovascular disease—to pollutant increments
- valuing health cases using economic values of human life derived by either the willingness–to–pay or human-capital approach.

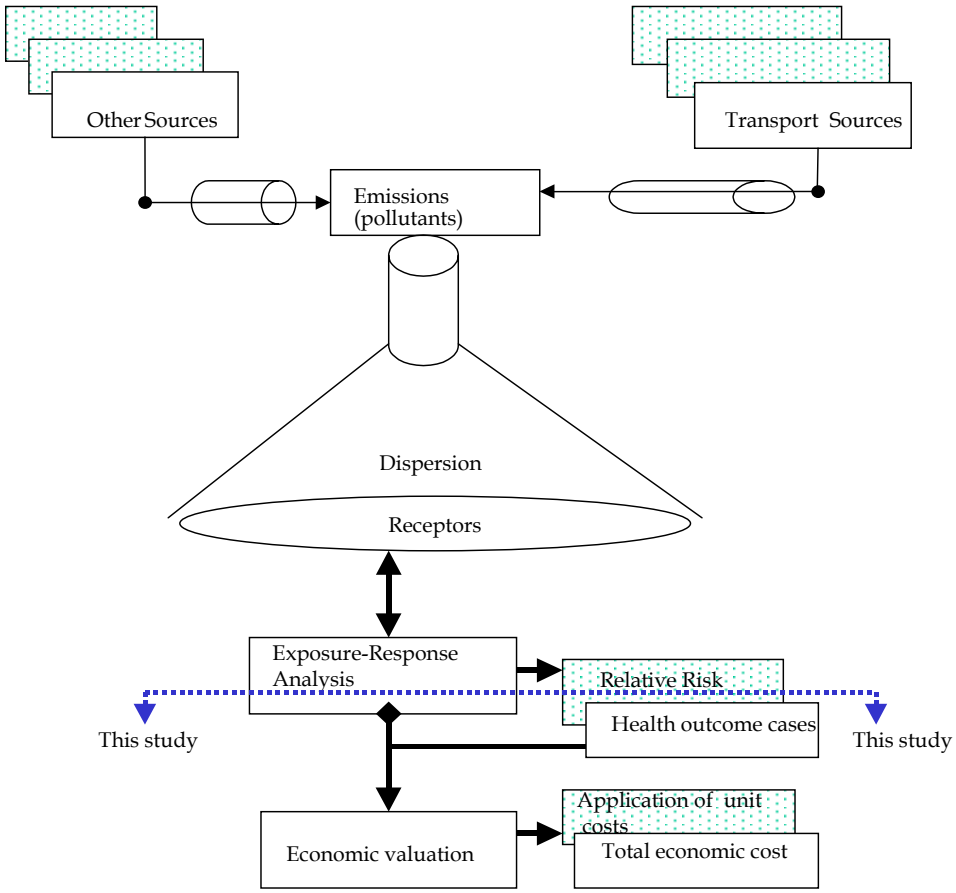
These steps are illustrated in figure 5.1. This study's assessment of the economic costs starts from the derived relative risk stage. Hence it does not include the detailed analysis and valuation of specific emission sources, dispersion modelling or impact analysis in terms of deriving dose–response regression coefficients—that is, relative risk ratios.[17] As illustrated in figure 5.1, the first step in assessing traffic pollution costs is quantifying the amount and type of pollutants that transport emits.

As outlined below, particulate matter (PM_{10}) is used in the assessment as a surrogate, or index, for all ambient air pollutants. Particulate matter is usually monitored and measured in terms of the mass concentration of particles of 10 microns or less (PM_{10}) and 2.5 microns or less in diameter ($PM_{2.5}$). The reasons

[17] The relative risk ratio is the slope of a regression line and it can be defined in this case as the proportion by which a change in health effect—the number of cases—occurs for a given increment in the ambient concentration of a pollutant.

for choosing particulate matter instead of any other air pollutant are discussed in the next section.

FIGURE 5.1 ASSESSMENT FLOW CHART



Source Bureau of Transport and Regional Economics

CHOICE OF SURROGATE POLLUTANT

The major transport pollutants for which epidemiological studies have derived relative risk ratios are PM_{10}, nitrogen dioxide, sulfur dioxide and carbon monoxide. Ideally, the best approach for choosing a surrogate pollutant would involve two steps. The first is to estimate the number of people affected using each pollutant's derived relative risk ratios.

Then the outcomes would be summed to obtain the total number of health cases—that is, the mortality and morbidity.

Unfortunately, this would grossly overestimate the number of health cases. The reason is that almost all air pollutants are correlated with each other (Morgan et al 1998a). That is, they affect, and interact with, each other. Therefore, it is not

always possible to separate or isolate the effects of a single pollutant. Hence, epidemiological studies—which estimate the association between pollutants and health effects—cannot strictly allocate the observed effects to individual pollutants.

The literature suggests that the statistical analysis underpinning epidemiological studies has established a strong correlation between various pollutants and health outcomes. But the strong correlations themselves do not necessarily imply causation. In the words of Kendall and Stuart (1961):

> A statistical relationship, however strong and however suggestive it may be, can never establish causal connection: our ideas of causation must come from outside statistics, ultimately from some theory or other.

The explanation of causation comes from epidemiology and toxicology and this is an area of intense and ongoing research. For example, in 1964, when the United States Surgeon General (Hauer 1997) asserted that smoking causes cancer, the following ad hoc rules for judging causality were used:

- strength of association—meaning some statistical measure of association is strong
- dose-response effect—the more of the causal factor, the larger the effect
- no temporal ambiguity—disease follows exposure to the risk factor
- consistent findings—several studies produce similar results
- biological plausibility—the hypothesis make sense in view of what is known in biology
- coherence of evidence—some combination of the previous two points
- specificity—causal factor causes this disease, and this disease is due to this causal factor.

A pollutant-by-pollutant assessment of health effects will grossly overestimate the health outcomes. Given this, there has to be a surrogate pollutant that can capture some or all of the effects of all the other pollutants. In 2000, the WHO and a body of other international studies suggested that PM_{10} or sulfur dioxide are useful indicators of the health risk of transport sources of ambient air pollution. The WHO stated that epidemiological analyses are best viewed as surrogates for mixtures of pollutants emitted from particular sources. They suggest that:

- Impact assessments should not simply add estimates of effects of individual pollutants derived from single-pollutant statistical models. However, multi-pollutant models may produce unstable estimates as the number of pollutants they include increases. Adding pollutant-specific effects may be justified when levels of the specific pollutants are clearly not correlated. For example, the overall impact of pollution in some locations in Europe might be estimated by summing the impacts of particles and ozone. This should be done cautiously. Firstly, in some cities particulate matter and ozone levels

may well be correlated. Secondly, the possibility of synergy, or antagonism, of pollutants cannot be excluded with confidence.

- Despite growing evidence from toxicological and epidemiological research that particulate air pollution per se is harmful, other pollutants—for example sulfur dioxide—should not be ignored. There may, in some settings, be better surrogates for specific pollutant sources than some indices of particulate matter. For example, it may be more appropriate to use carbon monoxide or nitrogen dioxide for mobile combustion sources, and sulfur dioxide for the combustion of home heating oil. In some cities the impact of these other pollutants on health may be substantial as well. More attention needs to be paid to the analysis of multi-city data to derive reliable coefficients for these pollutants.
- The health impact of ambient air pollution may depend on the mixture of pollutants. Therefore, there may be merit in adjusting a given city's effect estimate for PM_{10}. This adjustment could be done according to the local concentrations of other pollutants—for example nitrogen dioxide—identified in multisite studies as modifiers for the effect of particles (WHO 2000a).

Given the available epidemiological and monitoring data, this study has used PM_{10} as the main indicator of ambient air pollution. The Bureau acknowledges that selecting only one pollutant may underestimate the health effects. This is consistent with an 'at least' approach to quantifying the health impacts of ambient air pollution—as used by Künzli et al (2000).

One way to minimise the degree to which the health effects are underestimated would be to include estimates of the health costs of ozone. This is an approach that merits consideration, because the correlation between particulate and ozone has been found to be not statistically significant. Even so, this finding of non-correlation may be location specific. Hence, caution is required in the addition of particulate and ozone health outcomes (WHO 2000a). Given this uncertainty, the health impact of ozone was not included in this analysis.

DETERMINING THE MOTOR VEHICLE PROPORTION OF PARTICLE EMISSIONS

As discussed in chapter 3, transport sources produce a range of different ambient air pollutants. However, not all are monitored and measured, or are believed to be important in terms of their health impact. Major pollutants with known health effects are discussed in detail in chapter 4. This analysis uses particulate matter—measured as PM_{10}—as a surrogate for all ambient air pollutants. Thus, the transport contribution to PM_{10} is an important input into the modelling of the health effects and their valuation.

Ideally the best data on transport pollution to use in the modelling is that measured directly from the atmosphere at the height where people are exposed.

However, measuring the proportion of particulate matter in the atmosphere that is attributable to transport emissions is almost impossible because of multiple sources of emissions. Unlike individual gaseous pollutants, suspended particulate matter is composed of a wide range of materials and particle sizes arising from a variety of sources. This was discussed in detail in chapter 2.

The relative contribution of each source and type of pollutant can vary every day, depending on meteorological conditions and the quantity from mobile and static sources. In practice, environmental protection authority air quality monitoring stations sample or capture PM_{10} from all sources including transport. The transport proportion is determined from this sampled mass concentration of PM_{10}. The derivation of the transport proportion is, therefore, fraught with difficulties. Fisher et al (2002) highlight some of these difficult issues as including:

- emissions from different sources occur at different times of the day—vehicle contributions might dominate during peak hours but be negligible at night.
- Emissions vary during day of the week—vehicle emissions tend to be lower on Sundays, but perhaps population exposure is greater on the weekends as more people are outdoors.
- The fractions will definitely vary through the season—particularly in regions with home heating emissions.
- Vehicle emissions tend to occur near to the ground, so perhaps they have more of an effect than industrial emissions occurring well above the ground.
- Particles of soot from vehicles—potentially carrying traces of toxic substances—may have a greater effect than those from other sources.
- Some people spend a significant amount of time in or around vehicles, whereas others may spend almost no time in significantly exposed situations.

Notwithstanding these difficulties and uncertainties, researchers have attempted to estimate the transport proportion of emissions (Bureau of Transport and Regional Economics 2002a, 2002b and the National Pollutant Inventory 2003). The inventory web site (www.npi.gov.au) provides details on the methodology used by some jurisdictions in Australia to estimate pollutant emissions by transport sources.

The Bureau has excluded non-motor vehicle transport emission sources due to the lack of National Pollution Inventory data for key capital city airsheds. Where estimates are available for other modes, they suggest that the effects of excluding aircraft, rail, shipping/boating and related industry sources are likely to be relatively minor in most capital city airsheds. This issue is discussed in more detail in chapter 3.

In this study, the Bureau estimates the motor vehicle proportion of PM_{10} using the National Pollution Inventory mass concentration data (in kilograms) for

PM_{10}. It assumes that these are applicable to the city-by-city aggregate samples (in $\mu g/m^3$) at the respective monitoring stations.

The motor vehicle proportion may be estimated as the particles emitted through motor vehicle exhaust pipes or may include particles stirred up from road surfaces as vehicles move along. There is considerable uncertainty about the magnitude of road dust emissions and National Pollution Inventory estimates are not available for all states and territories. To ensure consistency across all cities, the Bureau has re-estimated the motor vehicle proportions by excluding road particulate matter. The National Pollution Inventory is discussed in more detail in chapter 3.

As shown in table 5.1, there is wide variation in the estimated motor vehicle proportion of PM_{10}. This is largely due to the differing sources of PM_{10} that have been reported by each jurisdiction, population densities and methods of estimation. Therefore, caution is required when comparing these proportions.

It should be noted that the motor vehicle proportions of particulate emission shown in table 5.1, and used for the calculations, differ from those shown for capital cities in table 3.2. The proportions shown in table 5.1 were estimated for areas that closely match the population and PM_{10} sampled district boundaries. Appendix 7 contains examples of how the Sydney and Melbourne vehicle proportions were estimated. Table 3.2 presents motor vehicle proportions based on known emissions within defined airshed boundaries. These airshed boundaries may extend beyond capital city boundaries—for example, the Sydney airshed boundary includes Newcastle and Wollongong.

TABLE 5.1 MOTOR VEHICLE SHARE OF PM_{10}

City	*Vehicles %*	*State/Territory wide*[a]	*Vehicles %*
Sydney	43	NSW	10
Melbourne	33	Vic	12
Brisbane	31	Qld	8
Adelaide	19	SA	6
Perth	20	WA	8
Hobart	10	Tas	7
Darwin	37	NT	1
Canberra	12	ACT	Na

Note na =not applicable. a= state and territory-wide motor vehicle proportions exclude capital cities.

Source Bureau of Transport and Regional Economics estimates using National Pollutant Inventory data (www.npi.gov.au)

POLLUTANT DISPERSION (CONCENTRATION)

Usually atmospheric researchers use a dispersion function to calculate the concentration of particles. This approach may assume mean dispersion conditions. The Commonwealth Industrial and Scientific Research Organisation airshed model, *The Air Pollution Model*, is an approach used in Australia to predict air pollutant concentration in the atmosphere. The Air Pollution Model (Hurley 2002) solves the fundamental fluid dynamics and scalar transport equations. It then uses these solutions to predict meteorology and ambient concentrations for a range of important reactive and non-reactive pollutants. Unlike pollutant monitoring—which cannot be carried out at all locations—the modelling provides data on ambient concentrations over a wide area.

From points of emission discharge, pollutants are dispersed into the atmosphere by wind. This suggests concentration and personal exposure at a spatial level will vary, and the literature tends to support this assumption (Kingham et al 2000; Fischer et al 2000). However, available data—for example, 2000 and 2001 annual average monitoring data for Sydney—indicates that mass particulate matter concentrations are distributed fairly uniformly in the metropolitan area. While this is the case for the annual average data it is not necessarily the case for the daily data (see for instance Neale 2003; Environmental Protection Agency South Australia 2002).

Some studies have suggested that particulate concentration can vary according to height above ground. Micallef and Collis (1998) even suggest that vertical variation can also lead to different height groups of the population being exposed to different pollutant concentrations. These studies suggest that the levels at which pollutants are monitored are an important factor in determining personal exposure levels. It may also have implications for heavily trafficked metropolitan cities since motor vehicle emissions usually occur closer to the ground.

In this study, sample particulate concentrations, measured at monitoring stations, are used as a surrogate for the average personal exposure. This assumes that the individuals are exposed to a quantity of pollution as measured at the monitoring sites.

In the capital cities this assumption may not be a major problem as the cities are relatively uniformly covered by monitoring stations. Spatial variability in pollution levels may be a major issue in regional Australia and is likely to lead to greater variability in personal exposure levels. However, given that approximately 80 per cent of Australia's population live in the capital cities, this potential over or underestimation is unlikely to bias the national estimates.

SELECTION OF RELATIVE RISK ESTIMATES

There have been numerous epidemiological studies—see chapter 4—on the health effects of air pollution. These studies demonstrate that the effects of

pollutants can be quantified using statistical techniques to analyse dose–response relationships. Despite this body of evidence, the estimated exposure-response functions are not usually consistent across studies. Researchers must decide which relative risk ratio, or exposure–response function, to use. There is also the issue of whether to use relative risk ratios derived from single pollutant equations or those derived from multiple regression analysis in which all suspected pollutants are modelled together.

Concerning the appropriate relative risk ratio to use to calculate health effects, several organisations and studies have recommended a series of exposure–response functions. Examples of this approach include a study of three European countries—France, Switzerland and Austria (Künzli et al 2000); the WHO air quality guidelines (WHO 1999a, 2000b); and the European Commission's ExternE Study (European Commission 1999).

In this study, the Bureau has used relative risk ratios for particulate matter from Künzli's et al three European country study results—the Künzli approach. There are two reasons for adopting this approach.

First, it is a state-of-the-art approach and builds on the advances made in the European Commission's ExternE Study. The approach has been used to independently value transport emission health costs in France, Switzerland, Austria and New Zealand.

Second, the approach relies extensively on epidemiological studies conducted and corroborated in the United States and Europe. It also adopts the WHO methodological recommendations.

The choice of overseas-derived relative risk ratios ultimately raises further questions. For example, are these overseas relative risk ratios appropriate for Australia, and why haven't Australian relative risk ratios been used?

APPLICABILITY OF OVERSEAS STUDIES

Several epidemiological studies have been undertaken in Australia using air pollution data sampled in major urban centres, such as Sydney, Melbourne and Brisbane. The results of these studies are robust and they are consistent—despite different relative risk estimates—with overseas studies (Denison et al 2001; Morgan et al 1998a).

However, most Australian studies are based on short-term time series data. That is, they examine the association between daily variation in pollution and health effects. Thus the regression coefficient that is obtained measures the short-term health impact. That is, the proportional increase in daily health effect—for example, the death rate—attributable to recent exposure to air pollution. These short-term studies are likely to capture only part of the pollutant-related cases, such as occurrences where exposure and health outcome, for example, death, are closely connected in time.

Time series analysis—as reported in the literature—is unlikely to capture increases in long-term morbidity and reduced life expectancy. There are advanced econometric techniques that can be used on time series data to derive both short-term and long-term coefficients. But the epidemiology literature reveals a limited application of these techniques for estimating regression coefficients for ambient air pollution (Zeger et al 1999; Schwartz 2000). Künzli estimates that using short-term time series-based regression coefficients is likely to underestimate mortality rates by four to five times. Hence, conceptually it is inappropriate to use short-term relative risk estimates to estimate the long-term impacts of air pollution on morbidity and mortality. Advanced time series analysis may improve the applicability of the results. After all, when econometric techniques are applied, short-term and long-term coefficients are routinely derived from the same time series data.

The Künzli approach adopted for this study is based on meta-analysis of cohort-based long-term United States studies. Cohort studies, by design and approach, track large populations over time and thus, by implication, through different levels of personal pollution exposure. Cohort studies include people whose deaths were advanced by recent exposure to air pollution, and those who died from chronic disease caused by long-term exposure. The relative risk ratio derived from cohort studies therefore captures both the short- and long-term health impact. Expert opinion converges on using results of cohort studies—at least until further advances are made in the analysis of time series data (WHO 2000a).

Perhaps the key issue is not whether air pollution studies are time series or cohort based. Rather, it is whether United States and European conditions—weather, traffic density, demographic characteristics and population health status—are similar to those in Australia. This is very important. Firstly these conditions affect the magnitude of the relative risk ratio. Secondly, the hypothesis underpinning the statistical analysis assumes that the characteristics of the study population—and the mixture of pollutants used to derive the regression coefficients—are identical to those in the general population to which the analysis is applied. For this assumption to be valid, the Australian conditions need to be sufficiently similar to the conditions that influence the magnitude of the regression coefficients in Europe and the United States. Estimated relative risk ratios from Australian studies provide evidence that this is the case (Denison et al 2001).

And the statistical techniques employed in dose–response analysis ensure that other confounding factors—such as temperature, age, gender and smoking—are accounted for in the modelling. In practice, however, all confounding factors may not be captured in the modelling. Nevertheless, the consistencies among studies from different countries suggest that uncontrolled confounding factors may not be a major issue.

This suggests that the relative risk ratios used by Künzli et al (2000) can be applied appropriately in the Australian context. It does not mean the number of health cases will be identical: this to some extent depends on age structure of the population and other factors. For example, an older population has a higher mortality rate than a younger population. The Australian median age is 35, Germany's is 40.1, France's is 37.6, the United Kingdom's is 37.7 and Switzerland's is 40.2 (United Nations 2002). Other health differences, such as in care systems and eating habits, may also influence the number of health cases.

The next chapter discusses the Künzli approach, its underlying assumptions, and the approach used to value the health outcomes in detail.

CHAPTER 6 ECONOMIC COST OF AMBIENT AIR POLLUTION FROM MOTOR VEHICLES

The analysis and calculation of economic values basically falls into two phases. The first is an impact assessment—that is, the derivation of the attributable number of health cases. The second is the calculation of the value of statistical life, the valuation of life quality and the direct cost of resources. These are then used to estimate total economic costs.

DERIVATION OF ATTRIBUTABLE NUMBER OF HEALTH CASES

The model used to derive the number of attributable health cases from all sources of pollutants is that used by Künzli et al (2000). The mathematical expression of the model is:

$$P_o = \frac{P_e}{1 + [(RR - 1)(Eo - B)/10]}$$

where:-

P_o = mortality in the base
P_e = the observed mortality in the population (age > 30)
E_o = observed mean PM_{10} exposure level
B = PM_{10} exposure level for mortality effect
RR = relative risk for a 10$\mu g/m^3$ increment of PM_{10}

The increased mortality attributable to a 10 $\mu g/m^3$ increase in PM_{10} is then calculated:

$$D_{10} = P_o * (RR - 1)$$

where:-

D_{10} = the number of additional deaths per one million people to the base level of mortality for a 10 $\mu g/m^3$ increase in PM_{10}
RR = relative risk for a 10$\mu g/m^3$ increment of PM_{10}

Next:

$$N_c = D_{10} * P_c * (E_o - B) / 10$$

where:-

N_c = the number of deaths due to PM_{10} for the population
D_{10} = the number of additional deaths per one million people to the base level of mortality for a 10 μg/m^3 increase in PM_{10}
P_c = the population
E_o = observed mean PM_{10} exposure level
B = PM_{10} exposure level for mortality effect

THE MODEL'S KEY ASSUMPTIONS

Although there is no empirical evidence on a PM_{10} threshold level above which health effects start to occur, Künzli et al state that:

> The increase in mortality cases is only considered from the exposure class of 5-10 μg/m^3 onwards (average 7.5 μg/m^3). It is to emphasize that this can not be considered as a level of no effect. So far, epidemiologic studies give no indication for a 'no-effect threshold' for PM_{10}, although some studies like those conducted in Switzerland include regions with rather low annual mean PM_{10} levels. However, studies did not include population living in regions with PM_{10} levels below 5-10 mg/m^3. Therefore, we did not extrapolate the risk function down to zero. This is again in line with the "at least" approach (Künzli et al 2000)

This study assumes that a minimum exposure level of PM_{10} is 5 μg/m^3. Other studies have used values between zero and 7.5 μg/m^3 as a minimum exposure level. A sensitivity analysis was conducted to assess the impact of a zero exposure class.

It is further assumed that all the population in each city is exposed to the weighted-average annual PM_{10} concentration estimated for the area. This issue is discussed further below.

Overall, the approach to modelling is to estimate the impacts that may be expected to be at least attributable to air pollutants.

DATA

The analysis in this study is based on year 2000 data. The choice of 2000 is primarily to ensure data consistency across all states and territories and also with data collected by the Australian Bureau of Statistics (ABS) and the Australian Institute of Health and Welfare. Typically, some ABS and Australian Institute of Health and Welfare data sets have a lag of one to two years. Although 2000 is the base year for sampled PM_{10}, where there is evidence of wide variability the weighted annual average or annual average has been estimated over the 2000 and 2001 data sets. This has been done to smooth or reduce variability in the data.

The primary sources of the data used in this study are Environment Australia and the various state environmental protection agencies, the ABS, the

Australian Institute of Health and Welfare, and the international literature for the estimated relative risk ratios.

Airborne particles are generally measured or sampled in two ways—by the use of a Tapered Element Oscillating Microbalance or High Volume Sampler. With the Tapered Element Oscillating Microbalance, the oscillating unit's frequency changes as particles are deposited on a heated filter.[18] The high volume sampler operation involves passing air through a size selective screen to first remove the larger-sized particles from the air stream, followed by particle collection on a filter. The concentration of respirable particles in the sampled air is calculated by measuring the weight gained by the filter, and the volume of air passing through the sampler.

Four data components are required to assess the number of health cases attributable to air pollutants:

- exposure–response functions
- the frequency of health outcome—that is, the prevalence
- the level of exposure
- the population.

EXPOSURE–RESPONSE FUNCTIONS

As discussed in chapter 5, the exposure–response functions were those used by Künzli et al (2000). They were calculated as the variance weighted average across a number of studies. Table 6.1 shows the exposure–response functions expressed as relative risk per 10 $\mu g/m^3$. A relative risk estimate of 1.043—that is, 1.043-1—or 4.3 per cent for total mortality can be interpreted as: for every 10 $\mu g/m^3$ increment in PM_{10}, the number of cases of premature mortality increases by 4.3 per cent. The theoretical basis of this interpretation is explained in Künzli et al (2000) and Pope et al (1995b). The number of health cases attributable to air pollutants has been estimated using the relative risk estimates in table 6.1.

An important point to note is that the relative risk estimates for mortality used by Künzli et al (2000) and this analysis are limited to adults 30 years and older. The estimated number of premature deaths that may be associated with air pollution will therefore exclude any deaths in potentially sensitive groups younger than 30—including infants and young children. For morbidity, the analysis excludes the number of restricted activity days lost to persons under the age of 20.

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[18] With the Tapered Element Oscillating Microbalance, the filter is heated and a significant proportion of volatile particles can be boiled-off. This can result in an underestimation of PM_{10} concentrations under some conditions. For this reason the PM_{10} measurements are adjusted to account for the loss of volatile particulate matter. The magnitude of the adjustment varies with the nature of the particulate matter and with ambient conditions. These adjustments are made before comparison with the national standard.

TABLE 6.1 HEALTH OUTCOME RELATIVE RISK ESTIMATES PM_{10}

Health outcome	*Relative risk estimate associated with a 10 $\mu g/m^3$ increase in PM_{10}*	*95 per cent confidence interval*
	Central	Lower and upper
Total mortality (adults ≥30 years, excluding traumatic or external causes)	1.043	1.026-1.061
Respiratory hospital admissions (all ages)	1.013	1.001-1.025
Cardiovascular hospital admissions (all ages)	1.013	1.007-1.019
Chronic bronchitis incidence (adults ≥25 years)	1.098	1.009-1.194
Bronchitis episodes (children <15 years)	1.306	1.135-1.502
Restricted activity days (adults ≥20 years)[a]	1.094	1.079-1.502
Asthma attacks (children <15 years)[b]	1.044	1.027-1.062
Asthma attacks (adults ≥15 years)[b]	1.039	1.019-1.059

a. Total person-days per year—any days where persons were forced to alter normal activity, due to respiratory disease

b. Total person-days per year with asthma attacks

Source Künzli et al 2000

FREQUENCY OF HEALTH OUTCOME

Data on the frequency of health-related cases were obtained from the Australian Institute of Health and Welfare. Table 6.2 shows the data for each health outcome for the year 2000.

For total deaths from non-external causes, some experts (World Health Organization (WHO) 2000a) believe that data on all-cause mortality are more reliable than data on cause-specific mortality because of issues with both classification and registration. Examples include cardiovascular disease, and asthma. Also, there is the possibility that there may be causes of death that are related to air pollution that have not yet been identified. Hence, it is appropriate to estimate pollution-related deaths from an all-deaths register other than from external causes. For these reasons, the WHO suggests that relative risk estimates for all-cause mortality should always be used when available (WHO 2000a). This is the approach adopted in this study. It is also important to note that the mortality data used in this analysis include only people aged 30 or more.

TABLE 6.2 HEALTH OUTCOME CASES IN CAPITAL CITIES AND REGIONAL AREAS 2000

Health outcome	Number of cases	
	Capital cities	Regional areas[a]
Total mortality (adults ≥30 years, Excluding traumatic or external causes)	70 568	47 066
Respiratory hospital admissions (all ages)	160 961	126 420
Cardiovascular hospital admissions (all ages)	224 820	164 183
Chronic bronchitis incidence (adults ≥25 years)	990	1 675
Bronchitis episodes (children <15 years)	250	686
Asthma attacks (children <15 years)[b]	11 937	7 636
Asthma attacks (adults ≥15 years)[b]	11 884	9 357

Note na not applicable

a. Regional areas are defined as all areas outside of capital cities.

b. Total person-days per year with asthma attacks.

Source Australian Institute of Health and Welfare

LEVEL OF EXPOSURE

Exposure is a measure of the overall time of pollutant contact by humans and the severity of that contact. Exposure assessment is a complex issue. Walsh and Beer (1998) outlined some of the factors that may require consideration in the assessment of personal exposure levels. These may include:

- time spent in different situations—such as home, work, in-transit—and travel routes taken
- the air quality in each micro-environment—usually resulting in exposure to multiple pollutants
- variations in breathing rate
- local variations in air quality—near roads and factories
- the general variation of ambient air quality in time and space—for example, the number of poor visibility days per year is typically higher in the city centre compared with the outer suburbs
- the duration of pollution events
- the general spatial pattern of residential population.

However, like the European and the New Zealand studies referred to in this report, the exposed population is the total number of persons in a city or town where monitoring occurs.

Levels of particulate exposure were obtained from States' and Territories' environmental protection agencies—either directly or from their respective web sites. Since all areas do not have monitoring stations, estimates were made of

exposure levels for capital city and regional areas. The total population were assumed to be exposed to this aggregate exposure levels.

An alternative approach for capital cities would to determining exposure levels would be to divide a city into many grids and estimate the population and exposure across the city on a grid-by-grid basis. This approach is more precise. But the Bureau considers that it is unlikely to improve the cost estimates given the degree of uncertainty attached to other key parameters—most notably, the transport share of ambient air pollution.

Because there are relatively few monitoring stations outside of the major capital cities, it is more difficult to accurately estimate particle levels in regional areas. Cities and towns where there are monitoring stations could be assessed individually. The results could then be applied to districts with identical average daily traffic and population. However, if monitoring sites were selected to monitor specific local pollution sources—for example, a major factory—the result could be biased.

Even if it was possible to estimate particle levels in regional area, the comparable health data—that is, hospital separations—are unlikely to be made available at this small demographic level. Health authorities are reluctant to provide health data for smaller cities and towns because the numbers are so small that individual patients could be identified.

Given these limitations, state- and territory-wide particulate matter exposure levels were assumed to be the minimum annual average sample recorded at a regional monitoring station. This is in accordance with the at least approach as it assumes everyone in a given population receives at least this minimum exposure to particulate matter. This approach should also minimise possible selection bias—that is, where monitors are being placed. Table 6.3 summarises the data on PM_{10} exposure levels.

TABLE 6.3 ANNUAL AVERAGE PM_{10} EXPOSURE LEVELS—ALL SOURCES, CAPITAL CITIES AND REGIONAL AREAS

Capital city	*PM_{10} $\mu g/m^3$*	*Regional areas*[a]	*PM_{10} $\mu g/m^3$*
Sydney	18.0	NSW	17.2
Melbourne	18.0	VIC	15.2
Brisbane	18.7	QLD	13.0
Adelaide	19.0	SA	15.0
Perth	18.8	WA	16.0
Hobart	16.0	TAS	16.0
Canberra	16.3	ACT	16.3
Darwin	14.9	NT	16.0

a. Regional areas are defined as all areas outside of capital cities.

Source Bureau of Transport and Regional Economics and state and territory environment protection agencies

CALCULATING HEALTH CASES

The Künzli model discussed above was programmed into a spreadsheet and the data sets discussed above used as input.

Tables 6.4 and 6.5 show the number of people dying prematurely that may be associated with motor vehicle pollution in capital cities and regional areas respectively. Table 6.6 presents combined mortality estimates for Australia.

For 2000 this study estimates that motor vehicle pollution accounted for between approximately 900 and 2000 premature deaths in Australia. More than 85 per cent of these early deaths would have occurred in capital cities and the rest in regional areas. Sydney and Melbourne—the cities with the largest populations and highest traffic levels—accounted for the most premature deaths.

For all capital cities the central estimate of approximately 1200 premature deaths attributed to motor vehicle air pollution represented about 1.7 per cent of all non-traumatic deaths for adults aged 30 or over.

TABLE 6.4 HEALTH CASES OUTCOME—MOTOR VEHICLE AIR POLLUTION–ASSOCIATED MORTALITY IN CAPITAL CITIES 2000

Capital Cities	*Central estimate*	*Lower*	*Upper*
Sydney	549	339	762
Melbourne	344	213	478
Brisbane	151	94	210
Adelaide	87	54	120
Perth	80	49	111
Hobart	6	4	9
Darwin	4	2	5
Canberra	6	4	9
All capital cities	1228	758	1703

Note Totals may not add due to rounding.

Source Bureau of Transport and Regional Economics estimates

TABLE 6.5 HEALTH CASES OUTCOME: MOTOR VEHICLE AIR POLLUTION—ASSOCIATED MORTALITY IN REGIONAL AREAS 2000

States and Territories	*Central estimate*	*Lower*	*Upper*
NSW	89	55	124
VIC	49	30	69
QLD	31	19	43
SA	7	4	10
WA	9	5	12
TAS	7	4	9
NT	0	0	0
ACT	na	na	na
Australia[a]	192	118	267

Note na not available. Totals may not add due to rounding.

a. Regional areas are defined as all areas outside of capital cities.

Source Bureau of Transport and Regional Economics estimates

TABLE 6.6 HEALTH CASES OUTCOME—MOTOR VEHICLE AIR POLLUTION–ASSOCIATED MORTALITY IN AUSTRALIA 2000

States and Territories	*Central estimate*	*Lower*	*Upper*
NSW	638	394	886
VIC	393	243	547
QLD	182	113	253
SA	94	58	130
WA	89	54	123
TAS	13	8	18
NT	4	2	5
ACT	6	4	9
Australia[a]	1420	876	1970

Note Totals may not add due to rounding.

a. Regional areas are defined as all areas outside of capital cities.

Source Bureau of Transport and Regional Economics estimates

In addition to premature deaths, the results show significant morbidity. Motor vehicle air pollution is estimated to have accounted for between approximately 900 and 4500 morbidity cases—cardio-vascular disease, respiratory disease, and bronchitis. Similarly, motor vehicle-related air pollution is estimated to have contributed to between 700 and 2050 asthma attacks in Australia (table 6.9).

The next section discusses the approach used for the economic valuation and the derivation of the economic costs associated with these health cases.

TABLE 6.7 HEALTH CASES OUTCOME—MOTOR VEHICLE AIR POLLUTION–ASSOCIATED MORBIDITY IN CAPITAL CITIES 2000

	Morbidity cases			*Asthma attacks*		
City	*Central estimate*	*Lower*	*Upper*	*Central estimate*	*Lower*	*Upper*
Sydney	1071	368	1756	392	208	570
Melbourne	682	240	1113	593	303	870
Brisbane	321	112	524	181	93	265
Adelaide	168	57	275	52	27	76
Perth	165	56	271	50	26	73
Hobart	10	4	16	0	0	0
Darwin	9	3	15	0	0	1
Canberra	na	na	na	na	na	na
All capital cities	2425	839	3970	1269	657	1855

Note Na not available. ACT morbidity data was not available. Totals may not add due to rounding.

Source Bureau of Transport and Regional Economics estimates

TABLE 6.8 HEALTH CASES OUTCOME—MOTOR VEHICLE AIR POLLUTION–ASSOCIATED MORBIDITY IN REGIONAL AREAS[A] 2000

	Morbidity cases			*Asthma attacks*		
Regional areas in:	*Central estimate*	*Lower*	*Upper*	*Central estimate*	*Lower*	*Upper*
NSW	137	47	226	27	14	39
VIC	65	22	107	36	18	53
QLD	50	17	83	34	17	83
SA	8	3	14	3	1	4
WA	18	5	30	5	3	7
TAS	6	2	18	1	0	1
NT	0	0	1	0	0	0
ACT	na	na	na	na	na	na
All regional areas	285	96	479	105	54	187

Note Na not available. ACT morbidity data was not available. Totals may not add due to rounding.

a. Regional areas are defined as all areas outside of capital cities.

Source Bureau of Transport and Regional Economics estimates

TABLE 6.9 HEALTH CASES OUTCOME—MOTOR VEHICLE AIR POLLUTION–ASSOCIATED MORBIDITY IN AUSTRALIA 2000

	Morbidity cases			*Asthma attacks*		
States/ Territories	*Central estimate*	*Lower*	*Upper*	*Central estimate*	*Lower*	*Upper*
NSW	1208	415	1981	419	221	609
VIC	747	262	1220	628	321	922
QLD	371	129	607	215	110	348
SA	176	59	289	55	28	80
WA	183	61	301	55	29	80
TAS	16	6	34	1	0	1
NT	9	3	16	0	0	1
ACT	na	na	na	na	na	na
Australia	2710	934	4449	1373	711	2042

Note na not available. Totals may not add due to rounding.

Source Bureau of Transport and Regional Economics estimates

ECONOMIC VALUATION

Society imputes economic values to premature deaths and disability or impairment caused by human activities such as traffic pollution or road crash injuries. The conceptual basis is that premature death or impairment deprives the community of the services of these people. Also, these people suffer loss of life quality. Thus, the consequences of premature death or impairment caused by human activities extend far beyond the immediate financial costs. The concept is used extensively in the valuation of economic losses associated with transport accident fatalities—see for example, Bureau of Transport and Regional Economics (2000 and 2003c). It can also be used to value the health impact of ambient air pollution, or any premature death or impairment.

Economists value these economic losses using two conceptual approaches—human capital and willingness-to-pay. There are other variants of these two approaches—see for example Abelson (2003). These two approaches are mutually exclusive, even though they are significantly complementary.

NOTION OF VALUE OF LIFE

People generally value their own lives—and those of their family and close friends—very highly. Indeed, life can be said to be priceless, as without life money would be of no use. This view is supported by the many publicised incidents where large amounts of money have been spent to save the life of an identified person. However, at the other end of the spectrum, much lower implicit values are placed on lives every day. Life has an assumed implicit value. This is demonstrated whenever funding is allocated to health and emergency services, to occupational health and safety projects, or to any activity that aims to save lives or prevent injuries.

Therefore, whereas a particular life may be regarded as priceless, relatively low implicit values may be assigned to life because of the distinction between identified and anonymous, or statistical, lives. When a value of life estimate is derived, it is not any particular person's life that is valued, but that of an unknown, or statistical, individual. The concept relates to the probability of a fatality in a given population. This is an important distinction when examining premature death caused by human activities such as road accidents or traffic pollution. It is important because it is not known which particular lives will be saved due to a change in the statistical probability of a death or injury in a crash or by pollutants. Valuing an unknown, or statistical, life eliminates subjective assessments of the worth of particular individuals. In practical terms, the distinction means that much smaller sums are allocated to saving statistical lives than may be spent in saving identified lives.

Human-capital method

The human-capital approach seeks to measure the economic impact of death and injury through the loss of output or productivity. This is generally done by calculating the present value of a persons' potential future output, as measured by their discounted anticipated stream of earnings. Essentially, this method calculates and values the years of life lost due to mortality. To this base value are added other costs which may include:

- monetary estimates of lost quality of life
- the value of non-market output—such as the services of those involved in household and community duties
- resource costs including medical and hospital costs.

An advantage of this approach to calculating years of life lost is that it avoids double counting for lost quality of life.

Willingness-to-pay method

The willingness-to-pay method estimates the value of life according to the amounts that individuals are prepared to pay to reduce risks to their lives. This approach attempts to capture trade-offs between wealth and risk. It also reflects the value of intangible elements such as quality of life and joy of living.

This approach uses people's preferences, either stated or revealed, to ascertain the value they place on reducing risk to life. To this base cost are added the net lost output, medical and administrative costs. These are human capital values.

Generally, the willingness-to-pay approach yields values far higher than those based solely on the human-capital approach.

The choice of approach

Both the human-capital and willingness-to-pay approaches are imperfect means of estimating the value of statistical life. Fundamentally, there are theoretical problems with the human-capital approach's application to the economic valuation of life. However, the willingness-to-pay approach involves various empirical difficulties.

The two approaches have some common deficiencies. Both involve a partial equilibrium approach in the sense that they ignore the wider consequences of extending life. For example, both approaches ignore intergenerational costs, such as a heavier social security burden on younger members of society. In other words, current members of the labour force must finance the consumption of a significant proportion of the elderly. Although these can be adjusted for in the human-capital approach, in practice they are usually not adjusted.

A basic criticism of the willingness-to-pay approach is that it fails to provide a market value. The estimate of value solely reflects what people are prepared to pay to reduce the risk of being injured or killed. It does not provide a comparison, as in a market, with a composite supply of health or safety-related goods, which would produce an equilibrium value. Willingness-to-pay estimates often have wide variation in the value of statistical lives. This is due, in part, to the fact that the value depends on circumstances and individual preferences in avoiding physical risk. This can be viewed in two ways:

- as a complicating and inconsistent factor
- as a strength of the willingness-to-pay approach in more precisely recognising people's preferences for particular preventive activities.

The variation is also partly due to country differences. Willingness-to-pay is country-specific. Inter-country comparisons of willingness-to-pay values are difficult to make as social, cultural and income factors confuse the picture.

In addition, reliability can be a problem when estimates are based on stated preferences—for example, survey responses—rather than on market transactions. This makes it difficult to know if the same values would be forthcoming if actual, rather than hypothetical, dollars were at stake.

There are two basic criticisms of the human-capital approach:

- It assumes full employment in the economy in that it attributes expected future income to all individuals. To counter this, in 1998 the Bureau of Transport Economics introduced the concept of the labour force participation rate. This ensures that the probability of people being in future formal employment or unemployed is considered.
- The approach does not provide an accurate measurement of the intrinsic value in cases where there is loss of life or suffering. To counter this, the Bureau of Transport and Regional Economics estimates life quality values using non-economic Australian court awards as a proxy. Court awards are seen as reflecting society's preferences. This is particularly so given the public reaction in recent years to court awards perceived as excessive. This has led to a change in the amounts being awarded. By excluding economic awards, double counting is avoided and the residual amount can be added to the present value of the productivity losses and other resource costs.

The human-capital approach produces lower bound but relatively consistent estimates to the extent that the results are easily replicated. The values are sensitive to the choice of discount rate. The choice of an appropriate discount rate and growth factors can bridge the gap between willingness-to-pay and human-capital estimates. A review of the literature by a European expert group (European Conference of Ministers of Transport 2000) could not settle firmly on either method. A review by Trawen et al (2002) found the application of these two methods is fairly even in the developed world. However, the trend is

shifting towards the willingness-to-pay approach due to recent advances in the technique.

Given the refinements the Bureau has made in its application of the human-capital approach and the need for consistency, the human-capital method is used in this study. This human capital method is also arguably the appropriate one for a higher level study of this type, in contrast to the evaluation of specific initiatives where it may be more practical proposition to assess the willingness-to-pay to reduce the risk of death. A detailed example of the human-capital method is discussed in appendix II.

VALUE OF STATISTICAL LIFE

Mortality

A recent Bureau study (Bureau of Transport and Regional Economics 2003c) for rail crash fatalities used an estimated VOSL of A$1.9 million.[19] The recommended VOSL for Europe is 1.1 to 1.3 million euros in 2000 values (European Conference of Ministers of Transport 2000). At the exchange rate of €1=A$1.7437 this is equivalent to A$1.9 to A$2.3 million. Drawing on a review of the literature and international guidelines for life and health values, Abelson (2003) recommended that Australian public agencies adopt a VOSL of $2.5 million for a healthy individual of about 40 years to avoid immediate death.

The Bureau's VOSL estimate of A$1.9 million for rail accident deaths is based on the age profile of these fatalities. Transport accident fatalities tend to be in the younger age group—particularly motor vehicle fatalities. However, pollution-related premature deaths are disproportionately in older age groups. The over 60 age group accounts for more than 60 per cent of pollution-related premature deaths, compared with less than 30 per cent of motor vehicle accident fatalities. Given this age disparity, it may be inappropriate to estimate the cost of air pollution-related mortality using an unadjusted VOSL derived for transport accident fatalities or to directly compare the number of attributed deaths from air pollution to transport accident fatalities (Rabl 2003).

In estimating the economic cost of air pollution, it is more appropriate to use an estimate that reflects the reduced life expectancy for affected individuals—rather than an average across the whole population. Whereas the average age of transport accident fatalities can be calculated, the long term cohort studies on which the mortality estimates are based do not provide estimates of the years of life lost for the affected groups or the age structure of the premature deaths. Thus, indirect estimates must be relied upon—these can be estimated using life tables (Künzli 2002). According to Sommer et al (1999):

[19] The BTRE has used the human-capital method to separately estimate VOSL for aviation, road, rail and maritime accident fatalities. The results by mode are of similar magnitude, reflecting the similar average age profiles for transport accident victims.

> The victims of road accidents typically have an average age of 30-40 years. The remaining average life expectancy for this age group is 35 to 45 years. On the contrary, the victims of air pollution have a much higher average age of about 70-80 years. The remaining life expectancy in this age group is about 10 to 15 years.

This analysis assumes that the reduction in life expectancy of pollution-related premature deaths is approximately 10 years. Using the human-capital approach, the estimated productivity loss for a death at age 60 is A$843 000 (in 2000 dollars). That is, about 30 per cent less than the average productivity loss value per transport accident fatality ($1.2 million) across the general population. A productivity loss of $843 000 per fatality translates into a value of about $60 000 per life year loss. When quality of life is adjusted for, the VOLY for mortality is $93 000. This is comparable with Abelson's estimate of $108 000 for the non-age dependent VOLY (Abelson 2003).

The Bureau has therefore used a VOSL of A$1.3 million (in 2000 dollars) in estimating of the cost of air pollution-related premature mortality—a 30 per cent reduction in the Bureau's rail accident VOSL. This is based on the estimated value of a life year lost (VOLY), adjusted for quality of life lost, and the average reduction in life expectancy for pollution-related premature deaths.

However, it is possible that a reduction of 30 per cent in the VOSL may still be an overestimation—this would be the case if lives were being shortened by only two to three years. The average reduction in life expectancy of pollution-related premature deaths could be as low as one year. This was discussed in more detail in chapter 4. At this extreme of one year, the mortality cost would be A$132 million and the total economic cost of air pollution—including morbidity—would be A$0.94 billion. This value is below the lower value estimated in the base—see table 6.12. This would also be the case if Abelson's VOLY was used—with a total economic cost of air pollution of $0.96 billion.

The human-capital method generally produces values at the lower end of the scale. Thus, if the willingness-to-pay method was used, the calculated values would be higher.

Another related issue is that people with pollution-related illnesses may suffer over longer periods than road crash fatalities. The costs associated with long-term illnesses—together with medical and hospital costs—are addressed and captured in the morbidity costs.

Morbidity

In addition to causing premature death, air pollution exposure also impairs or reduces affected people's life quality and productive capacity. The economic cost of morbidity comprises the temporary loss of productivity, the loss of life quality and the resource costs such as hospital and medical costs.

Several techniques can be used to assess the burden of diseases. These include:

- the use of years of life lost (YLL)
- equivalent healthy years of life lost due to disability (Brunekreef 1997; Robins and Greenland 1991; Mathers et al 1999)
- disability or quality adjusted life years (DALYs or QALYs) (Mathers 1999).

The DALY extends the concept of potential years of life lost due to premature death. It also includes equivalent healthy years of life lost by a person not being in optimal good health. One DALY is a lost year of healthy life. It is calculated as the sum of years of life lost due to premature mortality (YLL) and equivalent healthy years of life lost due to disability (YLLD). That is:

DALY =YLL+YLLD

In this study, the economic costs of mortality are estimated separately. Therefore, the use of DALY to estimate the burden of morbidity presents the potential for double counting since the concept includes the estimate of premature mortality. Because of this potential for double counting this analysis uses the YLLD approach for the estimation and costing of morbidity. The Bureau used a variant of the YLLD when it estimated the burden of long-term disability resulting from transport accident injuries (Bureau of Transport Economics 2000).

Years lost due to disability may be calculated as:

YLLD= I*D*[1-exp(-r*L)]/r

where:

YLLD is years of healthy life lost due to disability
I is the number of incident cases in a reference period—for example, cases of respiratory hospital admissions
D is the disability weight in the range of 0–1
L is the average duration of disability—measured in years
r is the discount rate

Mathers et al (1999) discuss this approach in detail and the Bureau has drawn on these results in this study. They report that in 1996 approximately nine per cent of total life expectancy at birth was lost due to disability for both males and females in Australia. The same report indicates that major air pollution related disease burdens—as a percentage of total years of life lost due to disability—were:

- asthma—4.8 per cent
- bronchitis—3.4 per cent
- respiratory disease—1.2 per cent
- cardiovascular disease—8.9 per cent.

The years of life lost due to disability were estimated using these percentages—as per Mathers et al (1999)—the ABS Australia 1999 life expectancy tables, and hospital admission data.[20]

The YLLD analysis requires separate individual admission records. The data was adjusted to reflect these definitional differences—see Mathers et al 1999. Also, the median age for each group was used: for example, age group 5–9 would have a median age of seven and a male life expectancy of 69.81 years. For asthmatic patients this life expectancy will be reduced by 4.8 per cent.

The economic cost of morbidity is estimated as:

M_d= YLLD*VLY

where

M_d is the morbidity cost
VLY is the value per healthy life year lost

This approach implicitly assumes that YLLD is associated with zero productivity—that is, time lost due to illness is assumed to produce no economic value. By implication this accounts for productivity losses due to restricted activity days. For this reason the value associated with restricted activity days has not been analysed independently of YLLD. Either restricted activity days—converted to years—or YLLD can be used in this approach but not both. While restricted activity days include non-hospital losses, assuming 100 per cent productivity losses through YLLD may account for any differences. The estimated YLLD is adjusted to reflect the respective motor vehicle pollution proportions.

The value per healthy year of life lost due to disability—using the human-capital approach to estimate the VOSL as discussed in this chapter—is estimated at $50 000.[21] The total economic cost of morbidity was in the order of $0.8 billion in 2000. This calculation assumes a VLY of $50 000.

[20] Hospital admissions data are, in effect, hospital separations. Admissions and separations are not necessarily the same thing. Separation data counts the number of times a particular health case is presented at a hospital irrespective of the patient. This means that the same person can be counted several times within a year.

[21] For unit costs used in the calculation of the total economic costs see appendix III, table III-1.

ESTIMATED ECONOMIC COST OF AIR POLLUTION-RELATED MORTALITY AND MORBIDITY

The combined economic cost of motor vehicle-related mortality and morbidity—the central estimates—was approximately $2.7 billion in 2000. More than 85 per cent of this cost is incurred in capital cities, where more than 80 per cent of Australians live. The cost range is between a lower value of $1.6 billion and an upper value of $3.8 billion. This range in values reflects the uncertainty bound or confidence interval surrounding the specific epidemiology estimates of health effects found by Künzli et al (2000).

The total economic cost of air pollution-related mortality is in the order of $1.8 billion. This figure is based on estimated motor vehicle pollution-related deaths of 1420—tables 6.4 and 6.5—and a VOSL of $1.3 million. The total economic costs associated with morbidity are in the order of $0.8 billion—see tables 6.11.

Tables 6.10, 6.11 and 6.12 show the total economic losses attributed to premature deaths and morbidity likely to have been caused by motor vehicle-related air pollution in Australia in 2000.

As indicated in chapter 1, this estimate should not be interpreted as an estimate of the external cost of motor vehicle pollution. This is a separate issue outside the scope of this study. However, it is possible that part or all of these costs are already internalised by motorists—for example, through taxes and charges that motorists pay to operate vehicles.

TABLE 6.10 TOTAL ECONOMIC COSTS OF MOTOR VEHICLE–RELATED POLLUTION ($M) IN AUSTRALIAN CAPITAL CITIES, 2000

Capital Cities	*Mortality*			*Morbidity*			*Total*		
	Base	*Lower*	*Upper*	*Base*	*Lower*	*Upper*	*Base*	*Lower*	*Upper*
Sydney	713	441	990	323	173	472	1,036	613	1,462
Melbourne	448	276	621	211	113	307	658	389	928
Brisbane	197	122	273	98	52	142	295	174	415
Adelaide	113	70	156	49	26	72	162	96	228
Perth	104	64	144	49	26	71	153	90	215
Hobart	8	5	11	3	2	5	11	7	16
Darwin	5	3	7	2	1	3	7	4	10
Canberra	8	5	12	-	-	-	8	5	12
All capital cities	1,596	986	0	735	394	1,072	2,330	1,380	3,286

Note na not available. Totals may not add due to rounding. Mortality cost estimated using a VOSL of A$1.3 million.

Source Bureau of Transport and Regional Economics estimates

TABLE 6.11 TOTAL ECONOMIC COSTS OF MOTOR VEHICLE–RELATED AIR POLLUTION ($M)—REGIONAL AREAS[A], 2000

	Mortality			*Morbidity*			*Total*		
Regional areas[a] in	*Base*	*Lower*	*Upper*	*Base*	*Lower*	*Upper*	*Base*	*Lower*	*Upper*
NSW	116	72	161	40	21	58	156	93	220
VIC	64	39	90	19	10	28	83	50	117
QLD	40	24	55	15	8	22	54	32	77
SA	10	6	13	2	1	3	12	7	16
WA	12	7	16	4	2	6	16	9	22
TAS	8	5	12	2	1	5	10	6	17
NT	0	0	0	0	0	0	0	0	1
ACT	na	na	na	na	na	na	na	na	na
All regional areas	250	154	348	82	44	123	332	198	470

Note na not available. Totals may not add due to rounding. Mortality cost estimated using a VOSL of A$1.3 million.

a Regional areas are defined as all areas outside of capital cities.

Source Bureau of Transport and Regional Economics estimates

TABLE 6.12 TOTAL ECONOMIC COSTS OF MOTOR VEHICLE–RELATED AIR POLLUTION ($M)–AUSTRALIA, 2000

	Mortality			*Morbidity*			*Total*		
State	*Base*	*Lower*	*Upper*	*Base*	*Lower*	*Upper*	*Base*	*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	nc	nc	nc	8	5	12
Australia	1,846	1,140	2,562	817	438	1,195	2,663	1577	3,757

Note na not available. Totals may not add due to rounding. Mortality cost estimated using a VOSL of A$1.3 million.

a. Regional areas are defined as excluding capital cities and towns with less than 5000 people.

Source Bureau of Transport and Regional Economics estimates

SENSITIVITY ANALYSIS

Sensitivity analysis was carried out to test the robustness of the key assumptions underpinning this analysis. The variations were done individually. That is, when one variable was changed all other variables were held constant at the base values. The sensitivity analysis results are presented in Table 6.13.

Addressing uncertainty in the transport share

Due to current data limitations, there is uncertainty about the proportion of PM_{10} concentrations attributable to motor vehicles in different locations.

If the motor vehicle share of PM_{10} is increased to 45 per cent—from approximately 35 per cent—in capital cities and increased to 20 per cent elsewhere, total estimated costs increase from the central estimate of $2.7 billion to $3.7 billion (+ 37 per cent).

Conversely, if the motor vehicle share of PM_{10} is reduced to 20 per cent in capital cities and elsewhere kept at base values, total estimated costs are reduced to approximately $2.1 billion (– 23 per cent).

Testing the effect of a zero threshold

Generally, pollutants cause negligible effects at very low levels. However, it is unclear whether this is the case for particulate matter. Künzli et al (2000) assume a dose–response threshold on the basis that there were no populations in their study exposed to ambient particulate matter concentrations between 0 and 5 μm^3. However, this should not be taken to mean there is no health effect below the threshold. As discussed in chapter 4, the question of a dose–response threshold—if any—is still an important research issue.

If the minimum exposure for PM_{10} mortality effects was reduced from 5 μm^3 to zero then the total estimated costs would increase to $3.3 billion (+ 25 per cent).

How different approaches to valuing statistical life affect the results

The value of statistical life used to derive the central estimates was $1.3 million—approximately 30 per cent less than the VOSL used for rail accident fatalities (Bureau of Transport and Regional Economics 2003c). This reflects the older age profile of air pollution-related early deaths.

TABLE 6.13 SENSITIVITY TESTING RESULTS: ECONOMIC COSTS OF MOTOR VEHICLE–RELATED AIR POLLUTION ($M)–AUSTRALIA, 2000

State	*Central estimate*	*Increased motor vehicle share*	*Decreased motor vehicle share*	*Zero threshold*	*VOSL A$2.5m; VOLY A$108,000*
NSW	1192	1381	810	1491	2361
VIC	741	960	565	931	1470
QLD	349	520	279	439	693
SA	174	356	180	216	344
WA	169	322	169	208	334
TAS	22	70	30	29	43
NT	8	13	5	10	15
ACT	8	31	14	12	16
Australia	2663	3653	2052	3335	5277

Note na not available. Totals may not add due to rounding.

Source Bureau of Transport and Regional Economics estimates

HEALTH COST ESTIMATES: COMPARISON WITH OTHER STUDIES

The health costs of air pollution due to the impact of vehicle emissions on the community have been estimated in a range of studies. However, all studies have limitations. They reflect the uncertainty about the exposure–response relationship between air quality and health, and the lack of consensus about the appropriate methods for placing dollar values on health effects. This section compares estimates from a range of relevant studies with this study.

In 1999, using an approach similar to the one used in this report, Filliger et al (1999) estimated health costs due to transport-related air pollution in Austria, France and Switzerland. They presented their findings in a paper prepared for the WHO Ministerial Conference for Environment and Health in London. The estimated health costs for the 1996 amounted to approximately 27 billion Euro across three countries:

- Austria—total vehicle pollution A$5.2 billion
- France—A$38.5 billion
- Switzerland—A$4 billion.

Across the three countries, the average road traffic-related share of gross domestic product amounted to 1.7 per cent (Filliger et al 1999).

Small and Kazimi (1995) estimated unit costs for the health effects of exhaust particulate emissions and motor vehicle-related ozone for the 1992 vehicle fleet in the Los Angeles region. These were approximately US$0.04 per kilometre for light vehicles and US$0.55 per kilometre for heavy vehicles. On the basis of urban vehicle kilometres travelled, these figures mean that exhaust particulate

emissions and motor vehicle-related ozone cost Australia approximately $7.4 billion per year. Using this study's estimation of the unit costs for vehicles with improved emission control gives a lower value of approximately $4 billion for future traffic streams.

A study conducted by the Australian Road Research Board (Brindle et al 1999) estimated that the health costs of vehicle emissions for Australia range from a low of A$20 million to a high of A$5.3 billion per annum. This wide range of estimated cost is attributed to differing assumptions and assertions about the:

- value of life
- valuation of medical costs
- degree of causative relationship between vehicles, pollution and health effects.

According to a report submitted to the National Road Transport Commission in 1995 (Segal 1999), the total value of the health effects of road vehicle emissions in Australia was estimated to be between A$20 million and A$100 million. The estimates for health costs were based on the respiratory health effects of ozone and cancer due to air toxics. The health effects of fine particles on the cardiopulmonary system were not included. They were excluded because the report concluded that there was no evidence of health effects at the low levels of fine particles typical of Australian cities.

Beer (2002) estimated the health costs of air pollution caused by vehicle emissions in Australia. Australian health impacts were estimated to cost A$30.4 billion comprising:

- particulate matter of less than 10 microns in diameter—A$17.2 billion
- non-methane hydrocarbons—A$12.8 billion
- nitrogen oxides—A$0.41 billion
- carbon monoxide—A$0.006 billion
- sulfur dioxide—A$0.001 billion.

If ozone is excluded, the figures decrease to A$12.4 billion for non-methane hydrocarbons and A$0.005 billion for nitrogen oxides. The total Australian health impact cost is reduced to A$29.6 billion. The study used a value of statistical life of US$6 million, as employed by the United States Environmental Protection Agency (personal communication).

A study conducted for the Australian Government Fuel Taxation Inquiry (AEA Technology 2002) assessed the potential air pollution costs from road transport in Australia using a dose–response approach (European Commission 2000). These costs were estimated by quantifying and valuing the potential health effects from vehicles. The study used data from the international literature, but adjusted it to reflect Australian conditions. A preliminary estimate of the potential air pollution costs from all road traffic in Australia indicated a damage

cost of approximately A$3 billion per year. The analysis of different technologies and fuels showed that measures to increase fleet turnover and introduce cleaner fuels would have a significant effect in reducing these costs. Of all previous Australian studies—these were discussed in detail in chapter 4—this is perhaps the most comprehensive.

However, previous Australian studies relied on a value of statistical life derived from overseas studies. These values tend to be very high when compared to the VOSL used in this study, and are particularly sensitive to the exchange rates chosen for conversion into Australian currency.

Another factor accounting for the higher economic cost of pollution in some of these studies is the inclusion of more than one pollutant. As discussed in chapter 5, using more than one pollutant usually leads to overestimation.

The central estimate of $2.7 billion in this report is comparable with the results obtained by the most comprehensive Australian study and with the overseas studies that have adopted a similar approach.

CONCLUDING REMARKS

The analysis shows that in Australia, in 2000, probable motor traffic pollution-related premature deaths ranged from a low of 900 to a high of 2000. In addition, the study estimates that between approximately 900 and 4500 hospital cases were attributable to motor traffic pollution. Also in 2000, between approximately 1400 and 2000 asthma attacks may have been attributable to motor vehicle emissions.

The Bureau estimates that the total annual economic burden for 2000 amounts to approximately $2.7 billion—that is, between a lower value of $1.6 billion and an upper value $3.8 billion.

The health impact of pollution is an evolving area of scientific research. Future estimates of the economic consequences of the health impact of motor vehicle pollution may be affected by new research into the:

- threshold levels above which pollutants are damaging to human health
- measurement of indoor pollution and its contributory sources
- more robust estimates of particulate emissions from road dust sources
- more accurate measurement of the motor vehicle share of local air pollution
- appropriateness of using surrogate pollutants to measure the combined impact of pollutants on health
- length of life loss associated with premature death from pollution causes.

Economic research into the community's willingness-to-pay to reduce the risk of pollution-related morbidity and mortality would also improve future estimates of the economic costs of these health impacts.

APPENDIX I TRANSPORT SHARES OF PARTICULATE MATTER IN CAPITAL CITY AIRSHEDS

This appendix provides a detailed breakdown of PM_{10} emissions from motor vehicles and other transport modesfor each capital city airshed as summarised in chapter 3. Appendix IV describes the methodology—not airshed-based—for estimating the motor vehicle proportion of PM_{10} for the capital cities, as per the analysis in chapter 6.

TABLE I.1 MOTOR VEHICLE PM_{10} EMISSION ESTIMATES BY AIRSHED 2000–01

National Pollution Inventory airshed description	*2000–01 PM_{10} emissions (kilograms)*	*Proportion of total[a] PM_{10} (per cent)*
Adelaide SA	570 000	17
Canberra ACT	92000	10
Darwin NT	69000	1
Hobart TAS	180 000	6
Perth WA	1 600 000	15
Port Phillip Region VIC	3 500 000	20
Southeast Queensland	790 000	31
Sydney–Newcastle–Wollongong NSW	5 800 000	28

Note nc Not estimated or not applicable. Proportions rounded to the nearest whole number.

a. Where total PM_{10} emissions exclude dust from paved and unpaved roads for all airsheds.

Source Bureau of Transport and Regional Economics estimates using National Pollution Inventory data (www.npi.gov.au)

TABLE I.2 PAVED AND UNPAVED ROAD PM_{10} EMISSION ESTIMATES BY AIRSHED, 2000–01

National Pollution Inventory airshed description	*2000–01I PM_{10} emissions (kilograms)*
Adelaide SA	5 600 000
Canberra ACT	na
Darwin NT	na
Hobart TAS	1 000 000
Perth WA	na
Port Phillip Region VIC	6 400 000
Southeast Queensland	na
Sydney–Newcastle–Wollongong NSW	na

Note nc Not estimated or not applicable.

Source Bureau of Transport and Regional Economics estimates using National Pollution Inventory data (www.npi.gov.au)

TABLE I.3 AIRCRAFT PM_{10} EMISSION ESTIMATES BY AIRSHED 2000–01

National Pollution Inventory airshed description	*2000–01 PM_{10} emissions (kilograms)*	*Proportion of total[a] PM_{10} (per cent)*
Adelaide SA	160 000	4.8
Canberra ACT	nc	nc
Darwin NT	25 000	0.4
Hobart TAS	2500	0.1
Perth WA	47 000	0.4
Port Phillip Region VIC	250 000	1.4
Southeast Queensland	nc	Nc
Sydney–Newcastle–Wollongong NSW	nc	Nc

Note nc Not estimated or not applicable.

a. Where total PM_{10} emissions exclude dust from paved and unpaved roads for all airsheds.

Source Bureau of Transport and Regional Economics estimates using National Pollution Inventory data (www.npi.gov.au)

TABLE I.4 SHIPPING PM_{10} EMISSION ESTIMATES BY AIRSHED 2000–01

National Pollution Inventory airshed description	*2000–01 PM_{10} emissions (kilograms)*	*Proportion of total[a] PM_{10} (per cent)*
Adelaide SA	23 110	0.7
Canberra ACT	nc	nc
Darwin NT	29 000	0.5
Hobart TAS	16 500	0.6
Perth WA	120 000	1.1
Port Phillip Region VIC	440 000	2.5
Southeast Queensland	nc	nc
Sydney–Newcastle–Wollongong NSW	nc	nc

Note nc Not estimated or not applicable.

a. Where total PM_{10} emissions exclude dust from paved and unpaved roads for all airsheds.

Source Bureau of Transport and Regional Economics estimates using National Pollution Inventory data (www.npi.gov.au)

TABLE I.5 RAIL PM_{10} EMISSION ESTIMATES BY AIRSHED 2000–01

National Pollution Inventory airshed description	*2000–01 PM_{10} emissions (kilograms)*	*Proportion of total[a] PM_{10} (per cent)*
Adelaide SA	13 000	0.4
Canberra ACT	nc	nc
Darwin NT	nc	nc
Hobart TAS	790	0.0
Perth WA	19 000	1.7
Port Phillip Region VIC	160	0.0
Southeast Queensland	26 000	0.4
Sydney–Newcastle–Wollongong NSW	nc	nc

Note nc Not estimated or not applicable.

a. Where total PM_{10} emissions exclude dust from paved and unpaved roads for all airsheds.

Source Bureau of Transport and Regional Economics estimates using National Pollution Inventory data (www.npi.gov.au)

APPENDIX II DERIVING THE VALUE OF A STATISTICAL LIFE—HUMAN-CAPITAL APPROACH

PRODUCTIVITY LOSSES IN THE WORKPLACE

When an individual dies prematurely or suffers from disability/impairment due to avoidable human activity, their potential labour output over their expected remaining years of life is lost to society or is reduced. Only the value of the labour is considered foregone. Income from non-labour sources—such as dividends and rental income—will continue regardless of the mortality or health of individuals. As the loss of labour services is felt in the workplace, the household and the community and their worth to these areas is calculated.

Working life tables have been used to derive potential workplace productivity losses and the average retirement period. The retirement period is calculated by working out the difference between life expectancy and expected working life. Others have used different age cut-offs, for example 65, 70, 75 and 85 years to represent either working life or potential productive years. Usually the ages between 15 and 70 years are referred to as the potentially productive years of life lost on the assumption that these are the productive years of life. Another measure, years of accumulated ability lost, weights the number of deaths by the ages at which they occur. This approach assumes that peoples' potential contributions increase with age and experience (Hahn RA 1995). Where a cut-off age is used to represent working life, it implicitly assumes that there is no working life thereafter.

Potential working life or productive years are derived from life expectancy tables. As described by the Australian Bureau of Statistics, life tables depict the mortality experience of a hypothetical cohort of newborn babies throughout their entire lifetime. They are based on the assumption that this cohort is subject to the age-specific mortality rates of the reference period. Typically this hypothetical group comprises 100 000 people. Life tables have many uses—for example, estimating the probability of living or surviving or dying. While these are not always published, they can easily be computed with the base table.

In computing the workplace productivity loses, neither expected working life tables nor a cut-off age have been used. The approach is based on the probability of a person surviving or dying. For example, suppose six people, all aged 44, died prematurely as a result of air pollution. The next step would be to

calculate the likelihood that those six people would have survived to age 45, 46, 47 et cetera. As the six progressively survive to the next age, the likelihood of their surviving or dying changes to reflect the specific age they have reached. At a point in time, between age 44 and 99, the hypothetical six people would have nil chance of living. It is at that point that all six, or some, would have naturally died.

Since the life tables are constructed with actual death rates at each single age the probability of surviving or dying ratios are weighted to reflect the high-risk age groups. This is an important factor to model into premature death cost analysis as some age groups have associated high risk factors.

The next step in the computation is to apply the Australian workforce participation rate at single age and by gender to those six people. This determines whether the people would have been employed. Next, the mean annual potential earnings at single age and by gender, are applied to each surviving person. Table II.1 shows an example of this hypothetical model computation. The computation formula is thus:

$$P_l = L_{X-t}\left(l_{x+t+1} \Big/ l_{x+t}\right)$$

where:

P_l is productivity losses

A_x is number of aviation accident deaths which would have survived to the next age x+1

l_x is number of people who survive to age x in a cohort of 100 000 (Australian Bureau of Statistics life tables 1994-96)

V_t is employment participation rate multiplied by potential (age-specific) earnings.

Since the expected losses occur in the future, the values are not comparable to today's values. Hence, the result (P_l) for each surviving year—table II.1 last column—is discounted to present value. That is:

$$PV = \sum_{x=i}^{\infty} \frac{P_x}{(1+rate)^{x-i}}$$

where:

PV is the present value of productivity losses over the relevant period and rate is the discount rate.

The base year in which the deaths occur, P_l, is halved to reflect the fact that deaths will occur randomly throughout the year. This avoids over or underestimation. P_l is computed for each single age that death occurs, and is summed after discounting.

Table II.1 Valuation of productivity losses

The base year in which the deaths occur	*Persons surviving out of 100 000*	*Fatality*	*Likely to survive*	*Mean annual earnings*	*Workforce participation rate*	*Value of Productivity Lost*
43	95 478	0	0		0.87	0
44	95 281	6	0	65 565	0.87	171 557
45	95 071	0	6	61 862	0.86	315 043
46	94 845	0	6	69 322	0.85	347 044
47	94 600	0	6	69 193	0.85	342 713
48	94 334	0	6	82 402	0.85	400 225
49	94 044	0	6	71 851	0.85	349 282
50	93 726	0	6	77 072	0.82	356 854
51	93 378	0	6	77 820	0.82	354 041
52	92 996	0	6	92 265	0.80	407 408
53	92 576	0	6	87 823	0.80	383 980
54	92 113	0	6	71 246	0.80	308 459
55	91 601	0	6	70 227	0.73	277 213
56	91 035	0	6	70 223	0.72	269 539
57	90 407	0	6	73 975	0.65	253 978
58	89 710	0	6	85 048	0.64	283 936
59	88 937	0	6	69 065	0.55	196 383
60	88 079	0	6	78 642	0.52	210 385
61	87 129	0	5	**122 936**	0.48	302 731
62	86 077	0	5	92 620	0.41	192 297
63	84 917	0	5	87 091	0.37	158 160
64	83 639	0	5	65 131	0.32	101 311
65	82 238	0	5	50 444	0.23	55 869
66	80 704	0	5	51 373	0.20	48 629
67	79 034	0	5	52 301	0.17	41 289
68	77 222	0	5	53 230	0.13	31 467
69	75 264	0	5	54 158	0.10	24 064
70	73 160	0	5	55 086	0.09	21 473
71	70 907	0	*4*	56 015	0.07	16 511
72	68 510	0	4	56 943	0.08	18 596
73	65 971	0	4	57 872	0.06	13 699
74	63 296	0	4	58 800	0.06	13 406
75	60 484	0	4	59 729	0.06	13 067
76	57 532	0	4	60 657	0.05	10 565
77	54 436	0	3	61 585	0.03	6118
78	51 195	0	3	62 514	0.03	5868
79	47 821	0	3	63 442	0.03	5591

The base year in which the deaths occur	Persons surviving out of 100 000	Fatality	Likely to survive	Mean annual earnings	Workforce participation rate	Value of Productivity Lost
80	44 332	0	3	64 371	0.02	3525
81	40 752	0	3	65 299	0.03	4957
82	37 118	0	2	66 228	0.02	4606
83	33 467	0	2	67 156	0.03	4236
84	29 847	0	2	68 084	0.04	5137
85	26 303	0	2	69 013	0.02	2309
86	22 885	0	1	69 941	0.00	0
87	19 639	0	1	70 870	0.00	0
88	16 608	0	1	71 798	0.00	0
89	13 825	0	1	72 727	0.00	0
90	11 318	0	1	73 655	0.00	0
91	9110	0	1	74 583	0.00	0
92	7211	0	0	75 512	0.00	0
93	5617	0	0	76 440	0.00	0
94	4313	0	0	77 369	0.00	0
95	3272	0	0	78 297	0.00	0
96	2459	0	0	79 225	0.00	0
97	1826	0	0	80 154	0.00	0
98	1341	0	0	81 082	0.00	0
99	973	0	0	82 011	0.00	0
						6 518 834

Source Bureau of Transport and Regional Economics estimates

To demonstrate the sensitivity of the present values, this study uses discount rates four, seven and eight per cent. Four per cent is the rate used by the Bureau for estimating the cost of road crashes (Bureau of Transport Economics 2000).

People's potential earnings from potential work are based on mean weekly earnings by single age and disaggregated by gender. The mean earnings best represent the market replacement wage for future potential labour. The weekly earnings are annualised, and factored by 25.4 per cent to account for labour on-costs—leave loading, superannuation et cetera. Finally a two per cent annual growth rate is applied to the earnings stream to account for real increases in productivity over time.

The workforce participation rate was based on single age and disaggregated by gender (Australian Bureau of Statistics unpublished data). It is generally assumed that ages 0 to 14 are not participants in the workforce. Any fatality in

this age bracket is assigned to age 15. That is, no productivity values are assigned until they are assumed to have reached age 15.

LOSSES IN HOUSEHOLD AND COMMUNITY PRODUCTION

When a person dies prematurely, productive activities in the home and elsewhere outside the formal work environment are lost. These losses are associated both with the employed and unemployed. This study used the market replacement method as opposed to the opportunity cost method. This involves estimating the average hours of work outside the formal workplace and applying a wage rate—that is, the expected or assumed cost of labour to perform the same function. The level of earnings per hour for those in the formal workforce was assumed to be equivalent to the labour cost to perform the household functions.

The Australian Bureau of Statistics estimates that employed males will, on average, devote 14.5 hours per week in household and other productive activities, compared with 22.1 hours per week for unemployed males. The rates for employed and unemployed females are 28.4 hours per week and 38.2 hours per week respectively. These values are annualised using 52 weeks, on the assumption that household works are carried out almost everyday of the week. As described earlier in this section, for computational simplicity the productive values—outside the formal workplace—during a person's employment period were incorporated in the estimates for workplace productivity. That is, values associated with 14.5 hours males and 28.4 hours females.

The same computational method used for the workplace productivity losses and described above is used for the estimation of household and other non-formal workplace productivity while a person is out of work.

Direct costs and quality of life values are added to the values derived using the above approach. For details see Bureau of Transport Economics (2000) and Bureau of Transport and Regional Economics (2003c).

APPENDIX III HEALTH CASE DATA USED IN THE ANALYSIS

TABLE III.1 HEALTH CASE DATA BY CAPITAL CITY 2000

Australia	*Hospital admissions/separation*	*No of days*	*Unit costs $*
Value of statistical life			1 900 000
Value per equivalent healthy years of life lost due to disability (YLLD)			50 000
Value per equivalent healthy life year lost due to mortality (YLL)			93 000
Sydney			
Respiratory hospital admissions (all ages)	62 635		336 832
Cardiovascular hospital admissions (all ages)	85 313		537 371
Chronic bronchitis incidence (adults >25 years)	np		274 369
Bronchitis episodes (children <15 years)	np		274 369
Restricted activity days (adults >20 years)*	na	Na	na
Asthma attacks (children <15 years)+	4947	8614	195 954
Asthma attacks (adults >15 years)+	4692	15 494	195 954
Melbourne			
Respiratory hospital admissions (all ages)	49 876		351 976
Cardiovascular hospital admissions (all ages)	72 981		531 596
Chronic bronchitis incidence (adults >25 years)	np		274 457
Bronchitis episodes (children <15 years)	np		274 457
Restricted activity days (adults >20 years)*	na	Na	na
Asthma attacks (children <15 years)+	3129	4527	210 983
Asthma attacks (adults >15 years)+	3668	12 859	210 983

Brisbane			
Respiratory hospital admissions (all ages)	24 213		329 678
Cardiovascular hospital admissions (all ages)	34 172		514 485
Chronic bronchitis incidence (adults >25 years)	np		292 197
Bronchitis episodes (children <15 years)	np		292 197
Restricted activity days (adults >20 years)*	na	Na	na
Asthma attacks (children <15 years)+	1751	3 189	206 634
Asthma attacks (adults >15 years)+	1987	8 274	206 634

Adelaide			
Respiratory hospital admissions (all ages)	20 999		338 478
Cardiovascular hospital admissions (all ages)	27 418		509 593
Chronic bronchitis incidence (adults >25 years)	np		290 153
Bronchitis episodes (children <15 years)	np		290 153
Restricted activity days (adults >20 years)*	na	Na	Na
Asthma attacks (children <15 years)+	1941	3711	190 571
Asthma attacks (adults >15 years)+	1312	5191	190 571

APPENDIX IV MOTOR VEHICLE SHARE OF PARTICULATE MATTER USED IN THE ANALYSIS

Estimates of the motor vehicle share of PM_{10} emissions are available for each city by area, as reported in the National Pollution Inventory.

Tables IV.1 and IV.2 show how the shares for Sydney and Melbourne were estimated for use in the analysis in chapters 5 and 6.

The tables show:

- the motor vehicle proportion—not including road dust—as estimated for each capital city by area
- the absolute level of PM_{10} emissions (in kilograms) from motor vehicles that this implies
- the weighting assigned to each city area (in micrograms), based on total pollution from all sources for each area.

TABLE IV.1 SYDNEY MOTOR VEHICLE PM_{10} SHARES

Sydney	*Veh %*	*PM_{10} (Kilograms)*	*Weighting (Micrograms*
Ashfield	51.5	45 000	2 317 500
Bankstown	57.2	250 000	14 300 000
Blacktown	39.1	260 000	10 166 000
Botany Bay	19.0	71 000	1 349 000
Camden	27.0	40 000	1 072 000
Campbelltown	17.0	140 000	2 338 000
Canterbury	50.0	150 000	7 425 000
Concord	56.8	43 000	2 442 400
Fairfield	17.2	180 000	3 096 000
Hawkesbury	28.2	56 000	1 579 200
Hurstville	48.9	71 000	3 471 900
Kogarah	57.5	64 000	3 680 000
Lane Cove	56.3	46 000	2 589 800
Leichhardt	51.3	71 000	3 642 300
Liverpool	55.9	21 000	11 739 000
Marrickville	55.5	100 000	5 555 000
North Sydney	57.0	71 000	4 047 000
Parramatta	33.7	25 000	8 425 000
Penrith	22.4	150 000	3 360 000
Pittwater	41.2	50 000	2 060 000
Randwick	30.8	76 000	2 340 800
Rockdale	20.1	130 000	2 613 000
Ryde	59.9	160 000	9 584 000
South Sydney	60.8	130 000	7 904 000
Strathfield	66.5	75 000	4 987 500
Sydney	68.1	64 000	4 358 400
Warringah	53.0	170 000	9 010 000
Waverley	26.8	22 000	589 600
Willoughby	48.9	96 000	4 694 400
Sum		3 241 000	140 731 800
Average	44 %		
Weighted average			43.4 %

TABLE IV.2 MELBOURNE MOTOR VEHICLES PM_{10} SHARES

Melbourne	*Total PM_{10} (Kg)*	*Vehicle share*	*Paved Road share*	*Total less paved Rd*	*Veh %*	*Weighting*
Banyule	420 000	63 000	180 000	240 000	0.26	16 538
Bayside	570 000	84 000	290 000	280 000	0.30	25 200
Boroondara	580 000	120 000	290 000	290 000	0.41	49 655
Brimbank	620 000	110 000	170 000	450 000	0.24	26 889
Cardinia	630 000	84 000	22 000	608 000	0.14	11 605
Casey	900 000	110 000	140 000	760 000	0.14	15 921
Darebin	390 000	76 000	140 000	250 000	0.30	23 104
Frankston	280 000	31 000	190 000	90 000	0.34	10 678
Greater Dandenong	880 000	55 000	510 000	370 000	0.15	8176
Hobsons Bay	690 000	140 000	160 000	530 000	0.26	36 981
Hume	900 000	240 000	75 000	825 000	0.29	69 818
Kingston	860 000	96 000	370 000	490 000	0.20	18 808
Knox	580 000	56 000	190 000	390 000	0.14	8041
Manningham	490 000	85 000	130 000	360 000	0.24	20 069
Maribyrnong	260 000	44 000	110 000	150 000	0.29	12 907
Maroondah	250 000	36 000	55 000	195 000	0.18	6646
Melbourne	510 000	96 000	190 000	320 000	0.30	28 800
Melton	410 000	200 000	140 000	270 000	0.74	148 148
Monash	820 000	69 000	510 000	310 000	0.22	15 358
Moonee Valley	270 000	66 000	120 000	150 000	0.44	29 040
Moreland	360 000	81 000	100 000	260 000	0.31	25 235
Mornington Peninsula	970 000	75 000	380 000	590 000	0.13	9534
Nillumbik	340 000	110 000	140 000	200 000	0.55	60 500
Stonnington	290 000	73 000	130 000	160 000	0.46	33 306
Whitehorse	540 000	63 000	300 000	240 000	0.26	16 538
Whittlesea	450 000	190 000	47 000	403 000	0.47	89 578
Wyndham	680 000	23 000	35 000	645 000	0.04	820
Yarra	230 000	46 000	96 000	134 000	0.34	15 791
Glen Eira	500 000	53 000	300 000	200 000	0.27	14 045
Sum		2 575 000				847 729
Average					29%	
Weighted average						33%

GLOSSARY

Acute	Sharp, severe, having rapid onset, severe symptoms, and a relatively short duration. Contrast with chronic.
Aerosol	A suspension of particles or liquid droplets—other than water or ice—in the atmosphere that can remain in air for long periods of time because of their small size and light weight.
Airshed	A body of air bounded by topography and meteorology in which a pollutant, once emitted, is contained for a period of time
Air toxics	Gaseous, aerosol or particulate pollutants—other than the six criteria pollutants—present in the air in low concentrations. These pollutants are toxic or persist so as to be a hazard to human, plant or animal life. The terms 'air toxics' and 'hazardous air pollutants' (HAPS) are often used interchangeably.
Ambient air	As defined in the Ambient Air Quality National Environment Protection Measure: 'ambient air means the external air environment, it does not include the air environment inside buildings or structures.'
Carcinogen	A substance, factor or situation that causes or induces cancer.
Case	As used in epidemiology, this is a countable instance in the population or study group of a disease, health disorder, or condition. Sometimes used to refer to an individual with the particular disease.
Case-control study	A type of observational analytic study. A study in which the risk factors of people with a disease are compared with those without a disease. Enrolment into the study is based on presence (case) or absence (control) of disease. Characteristics such as previous exposure are then compared between cases and controls.

Causality	The relating of causes to the effects they produce. It must be emphasized that epidemiological evidence by itself is insufficient to establish causality—although it can provide powerful circumstantial evidence.
Cohort	A well-defined group of people who have had a common experience or exposure, who are then followed up for the incidence of new diseases or events, as in a cohort or prospective study. A group of people born during a particular period or year is called a birth cohort.
Cohort study	A type of observational analytic study. Enrolment into the study is based on exposure characteristics or membership in a group. Disease, death, or other health-related outcomes are then ascertained and compared.
Concentration	The quantative expression of the presence of a pollutant, but there is no exposure unless there is physical contact.
Confidence interval	A range of values for a variable of interest, constructed so that this range has a specified probability of including the true value of the variable. The specified probability is called the confidence level, and the end points—the minimum or maximum values—of the confidence interval are called the confidence limits.
Confounding factor	A variable that can cause or prevent the outcome of interest, is not an intermediate variable, and is associated with the factor under investigation. A confounding variable may be due chance or bias. Unless it is possible to adjust for confounding variables, their effects cannot be distinguished from those of factor(s) being studied.
Chronic	Occurring over a long period of time, either consistently or intermittently. Contrast with acute.
Chronic effects	Effects that last a long time even if caused by a single acute exposure.
Criteria pollutants	In Australia six criteria pollutants have been identified: carbon monoxide, lead, nitrogen dioxide, photochemical oxidants (measured as ozone), particles as PM_{10} (particles of 10 micrometres or less) and sulfur dioxide.
Incidence (of a disease)	Rate of new occurrences of a disease.
Disability-adjusted life years (DALYs)	An indicator developed to assess the global burden of disease. DALYs are computed by adjusted age-specific life expectancy for loss of health life due to disability. The value of a year of life at each age is weighted, as are

	decrements to health from disability from specified diseases and injuries.
Discounting	The process of converting future dollars and future health outcomes to their present value. See also Discount rate.
Discount rate	The interest rate used to compute present value, or the interest rate used in discounting future sums.
Dose	A level of exposure which is a function of pollutant concentration, length of time a subject is exposed, and the amount of the pollutant absorbed.
Dose-response	The relationship between the dose of a pollutant and the response (or effect) it produces on a biological system.
Emission factor	For stationary sources, the relationship between the amount of pollution produced and the amount of raw material processed or burned. For mobile sources, the relationship between the amount of pollution produced and the number of vehicle miles travelled. Emissions for a given source can be calculated from the emission factor of a pollutant and specific data regarding quantities of materials used by the source. This approach is used in preparing an emissions inventory.
Emissions	Releases of contaminants from a facility, area or other source.
Epidemiology	The study of the occurrence and distribution of disease within a population. In other words, the core public health science, investigating the causes and risk factors of disease and injury in populations and the potential to reduce such disease burdens.
Evaporative emissions	Emissions of hydrocarbons and trace metals from evaporating fuel.
Exposure	Contact with a chemical or physical agent that can occur through breathing, drinking, eating and by direct skin contact.
Exposure-response	The relationship between exposure level and incidence of adverse effects.
Exposure assessment	Determination of the sources, environmental transport and modification, and fate of pollutants and contaminants, including the conditions under which people or other target species, could be exposed and the doses that could result in adverse effects.

Hazard	An intrinsic capacity to cause harm associated with an agent or process. Alternatively, a source of possible damage or injury.
Human capital (approach)	A theoretical approach to valuing human life that seeks to measure the economic impact of death and injury through the loss of output or productivity. It does this by using the market value of the output produced by an unknown individual over an expected lifetime.
Hydrocarbons	Compounds containing various combinations of hydrogen and carbon atoms. Sources include trees, fuels and solvents. See also volatile organic compounds.
Incidence	In the case of a health event, the number of new cases during a certain time period.
Indoor air	The NHMRC defines indoor air as any non-industrial indoor space where a person spends a period of an hour or more in any day. This can include the office, classroom, motor vehicle, shopping centre, hospital and home.
Life expectancy	The average length of life of individuals in a population.
Life tables	Tables derived using a procedure by which the mortality (or morbidity) of a fixed population is evaluated within successive small time intervals so that the time dependence of mortality can be determined.
Longitudinal study	A study taking place over time. If individuals are followed, this is a longitudinal cohort study. If individuals are not followed, but classes (usually age classes) are restudied, this is a longitudinal cross-sectional study.
Morbidity	Refers to ill health in an individual and to levels of ill health in a population or group.
Mortality	Death.
Mortality rate	A measure of the frequency of occurrence of death in a defined population during a specified interval of time.
Organic compounds	Chemical compounds containing mainly carbon, hydrogen, nitrogen, and oxygen.
Particulate matter (PM)	Any material, except pure water, that exists in the solid or liquid state in the atmosphere.
Polycyclic aromatic hydrocarbons (PAHs)	Organic compounds that include only carbon and hydrogen with a fused ring structure containing at least two benzene (six-sided) rings. PAHs may also contain additional fused rings that are not six-sided. Combustion

	of organic substances is a common source of PAHs.
Prevalence	The number or proportion of cases or events or conditions in a given population.
Quality-adjusted life year (QALY)	A common measure of health improvement used in cost-utility analysis, it measures life expectancy adjusted for quality of life.
Quality of life	A broad construct reflecting subjective or objective judgment concerning all aspects of an individual's existence, including health, economic, political, cultural, environmental, aesthetic, and spiritual aspects.
Respirable suspended particles	The proportion of total suspended particles of a size smaller than 10 micrometres. They have the ability to penetrate deeply into the lungs.
Relative risk (RR)	An estimate of the magnitude of an association between exposure and disease which also indicates the likelihood of developing the disease among persons who are exposed relative to those who are not. It is defined as the ratio of incidence of disease in the exposed group divided by the corresponding incidence of disease in the non-exposed group.
Risk factor	A characteristic (eg age, sex, race) or variable (eg smoking, occupational exposure) associated with increased probability of an adverse health effect.
Secondary air pollutants	Products of atmospheric chemical reactions involving one or more other air pollutants.
Sensitivity	The ability of a system to detect epidemics and other changes in disease occurrence. The proportion of persons with disease who are correctly identified by a screening test or case definition as having disease.
Sensitivity analysis	Calculations that isolate factors involved in a analysis to indicate the degree of influence each factor has on the outcomes of the entire analysis.
Smog	A combination of smoke and other particles, ozone, hydrocarbons, nitrogen oxides, and other chemically reactive compounds which, under certain conditions of weather and sunlight, may result in a murky brown haze that causes adverse health effects.
Source	Any place or object from which air pollutants are released. Sources may be either stationary or mobile.

Synergistic	The interaction of two or more chemicals resulting in an effect greater than the sum of their separate effects.
Time-series analysis	Statistics using multiple observations of an individual or group over time; statistical analysis of the dynamics of change.
Total suspended particles (TSPs)	Particles of solid or liquid matter - such as soot, dust, aerosols, fumes, and mist - up to approximately 30 micrometres in size.
Toxicity	The quality or degree of being poisonous or harmful to plant, animal or human life.
Uncertainty	A situation in which an individual has incomplete information as to what is going to happen in the future.
Value of statistical life (VOSL)	The monetary value assigned for economic costing purposes to the life of an unknown individual.
Volatile organic compounds (VOC)	Released from burning fuel and the evaporation of solvents, paints, glues and other products used at work or at home. Consist mainly of unburned hydrocarbons but includes partially burned compounds such as aldehydes.
Willingness-to-pay (approach)	A theoretical approach to valuing human life that seeks to determine the maximum amount of money that an individual is prepared to give up to ensure an outcome—such as avoiding premature death or disability.
Years of life lost (YLL)	A measure of the impact of premature mortality on a population, calculated as the sum of the differences between some predetermined minimum or desired life span and the age of death for individuals who died earlier than that predetermined age.
Years of life lost due to disability (YLLD)	Equivalent healthy years of life lost due to disability. This is the same as years lost due to disability (YLD) as used by Mathers et al 1999 and others—however, it has been defined as YLLD in this study to avoid confusion with the term 'years lived with a disability' (also YLD) as more generally used in the literature.

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