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THE ECONOMIC CONSEQUENCES OF THE HEALTH EFFECTS OF TRANSPORT EMISSIONS IN AUSTRALIAN CAPITAL CITIES

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Abstract

There is increasing recognition that we need to know more about the economic and social costs resulting from transport externalities if we are to develop effective strategies to address both societal requirements and institutional efficiency. It is desirable that all externalities be fully costed and for these costs to be included in transport project evaluations, and for transport users to face the full cost (social and private) of their transport choices. A number of studies have been initiated in recent years in Australia and elsewhere in the developed world with the objective of costing the health and environmental impacts 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. Many of these studies have attempted to link the extent of life lost and the socioeconomic characteristics of those at greatest risk to air pollution. From that analysis, estimates of the economic cost have been derived.

This paper is largely drawn from a forthcoming Bureau of Transport and Regional Economics (BTRE) study and is an attempt to quantify the economic costs of the health effects of transport emissions in Australian capital cities. It uses epidemiology-based exposure-response functions to derive an attributable number of health cases, and applies a BTRE-refined human capital approach to derive economic costs.

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1 INTRODUCTION

The purpose of this paper is to quantify the economic costs of the health effects of transport emissions in Australian capital cities. It uses epidemiology-based exposure-response functions to derive an attributable number of health cases and applies a BTRE-improved human capital approach to derive economic costs.

For many years now, epidemiologists and toxicologists have been reporting on the scientific evidence showing adverse health effects from air pollution. This work can be traced back to the early and mid 20th century through a series of air pollution episodes in developed countries, which had acute adverse effects on public health. The Meuse Valley in Belgium (1930), Donora in the USA (1948) and London in England (1952) all experienced air pollution episodes, which were investigated in detail *(WHO, 1999*). In the 1952 London air pollution episode, it was estimated that 4000 extra deaths occurred as a result of a smog largely consisting of high concentrations of sulphur dioxide (SO_2) and particulate matter (Brimblecombe, 1987). Since then, a vast amount of literature has emerged focusing on the damaging effects of air pollutants on public health.

Based on these research findings, environmental regulatory authorities in Europe started implementing stringent air quality measures in the early 1960s, resulting in a reduction of air pollution levels. The most pronounced effect was observed for sulphur dioxide: total emission was reduced by about 50% in the European region during the period 1980–1995. Reduction of emission of nitrogen oxides was smaller and was observed only after 1990: total emissions declined by about 15% in the period from 1990 to 1995 (EEA, 1998). Implementation of mandatory use of unleaded petrol to minimise lead concentration levels in the air has seen a remarkable reduction of lead content in the atmosphere in Europe (WHO 1999) and in Australia (Environment Australia 2001). Trends in concentrations of other pollutants in urban air, such as nitrogen dioxide or particulate matter, are less clear and it is considered that these pollutants still constitute a risk to human health (WHO 1998, EEA 2003).

However, epidemiological research over 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 developed countries (WHO 2000). Hence, even though Australia may be regarded as a country of low pollution levels, a potential health risk remains.

1.1 Current Air Quality In Australia

Australia is a highly urbanised country with over 80 per cent of the population living in the cities. Each city has expanded rapidly in the past 30 years and relies heavily on private rather than public transport. The continuing growth of cities has imposed increasing costs through transport externalities, in particular congestion and pollution. In addition to health effects, air pollution causes degradation of the built and natural environment and loss of amenity. Hence, it is a national priority to monitor the state of air quality in the major cities. The air pollutants of current concern in Australia are particulates (PM10 and PM2.5), ozone, nitrogen dioxide, benzene and to a lesser extent, carbon monoxide (CO).

The nature of air pollution in Australia is often quite different from that in other countries. Sulphur dioxide, for example, accounts for a far lower share of emissions in Australia than in Europe. However, local ozone is an issue in Australia. The most important area of concern in Australia is the increased concentration of particulate matters (PM10, PM2.5) in Australian cities. Epidemiological evidence indicates that particulate pollution results in premature mortality as well as morbidity effects.

Table 1 shows the Australia National Environment Protection Measure for Ambient Air Quality (NEPM) standards set for six air pollutants. Table 2 shows some recorded or sampled maximum and annual average PM10 in Australian capital cities.

TABLE 1: 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
Ozone	1 hour	0.10 ppm	1 day a year
	4 hours	0.08 ppm	1 day a year
Sulphur 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 PM10	1 day	50 $\mu g/m^3$	5 days a year

Source: Environmental Protection and Heritage Council (EPHC) http://www.ephc.gov.au

Table 2: Maximum and Annual Average levels of PM10

City	Year	Maximum PM10 $\mu g/m^3$	Average PM10 $\mu g/m^3$
Sydney- Earlwood	2001	130	21
Melbourne -Alphington	2001	72.6	18.9
Brisbane- Rocklea	2001	70.8	16.6
Adelaide- North Plymton	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

Na not available

Source: Various EPA web sites and unpublished data

1.2. Pollution and Health Effects

The effects of air pollutants on health vary depending on several factors including the level of exposure and the susceptibility of the exposed population. The susceptibility of

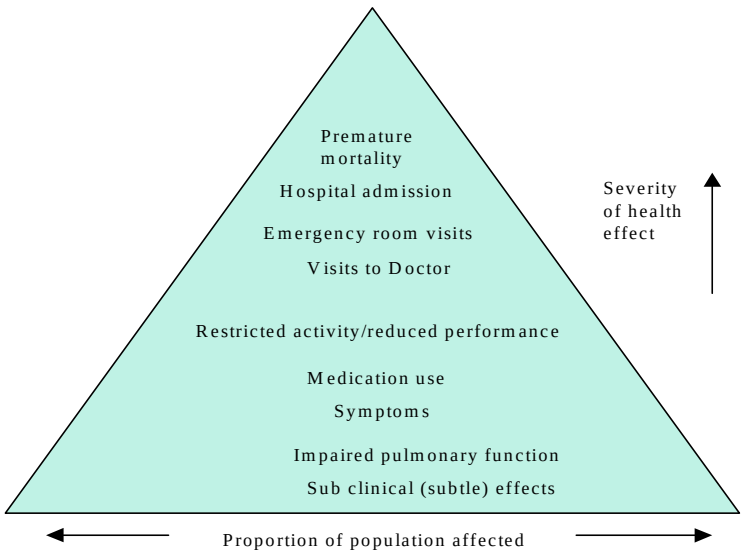
the population is affected by factors such as the number of young children and older people as well as the proportion of people suffering from asthma and other chronic respiratory conditions. Epidemiological studies reflect this variation in sensitivity by showing different associations between levels of exposure and health effects for different sub-populations. In addition, sources and patterns of exposure (for example, indoor and outdoor exposures) are likely to differ substantially from region to region, which is partly dependent on the weather conditions. Major air pollutants and their health effects are summarised in table 3.

TABLE 3: MAJOR AIR POLLUTANTS AND HEALTH EFFECTS

Pollutant	*Health effects*
Carbon monoxide	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.
Sulphur dioxide	Affects the throat and lungs. People with breathing problems can suffer severe illness.
Nitrogen dioxide	Affects the throat and lungs.
Volatile organic compounds	Some VOCs cause eye and skin irritation, headaches or nausea, while some are classed as carcinogens.
Ozone	Ozone Affects the tissue of the throat and lungs.
Lead	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.
Particles	May cause breathing difficulties and worsen respiratory diseases. Some particles contain cancer-producing materials.

Source: Australian State of the Environment Report 2001

FIGURE 1: THE SEQUENCE OF HEALTH IMPACT



Source: WHO 2000

Figure 1 shows the stages of the various health effects. These stages can range from subtle/mild health effects to premature mortality.

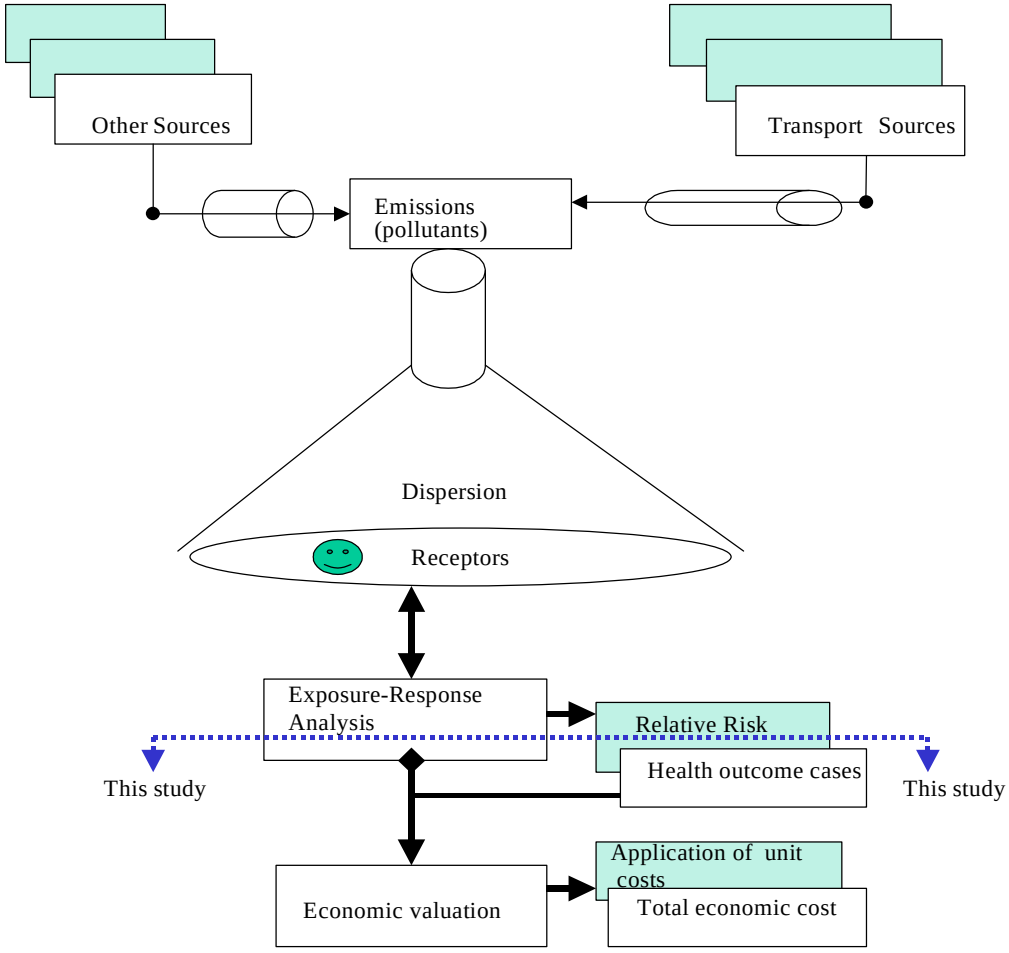
1.3 Motor Vehicles and Emissions

Motor vehicle emissions consist of a mixture of many compounds however, not all of these compounds are monitored and measured, and they are not all believed to be important in terms of their health impact. The most commonly cited vehicle pollutants include, particulate matter (PM), oxides of nitrogen (NO_x), volatile organic compounds (VOCs) or hydrocarbons, sulphur dioxide (SO_2) and carbon monoxide (CO). Passenger cars in urban areas are the major contributors to motor vehicle emissions. However, trucks and other commercial vehicles, while fewer in number and kilometres travelled, are nevertheless a significant source of NOx and particulate emissions in urban areas.

2 ASSESSING HEALTH CASES –ANALYTICAL APPROACH

The approach we have adopted follows the European concept often referred to as the 'dose response' or 'impact pathway' approach (AEA Technology Environment2002, WHO 2000). Primarily, the approach involves the steps shown in figure 2. This assessment takes its start from the derived relative risk stage of figure 2.

Figure 2: ASSESSMENT FLOW CHART



2.1 Conceptual Issues

There have been numerous epidemiological studies (for example, Denison et al 2001, Morgan et al 1998, Pope et al 1992 and 1950 on the health effects of air pollution. These studies have demonstrated 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. The issue arises as to which exposure-response function / relative risk (RR) ratios to use. There is also the issue of whether to use RR ratios derived from single pollutant equations or to use RR ratios derived from multiple regression analysis in which all suspected pollutants are modelled together. This raises the additional issue of which pollutant should be chosen to represent all other possible pollutants if a single pollutant equation is the basis of calculation.

On the issue of the appropriate RR ratio to use to calculate health effects, a number of organisations/studies have recommended a series of exposure-response functions. Examples of this approach include, a (Künzli et al 2000) study of three European countries (France, Switzerland and Austria), the World Health Organisation air quality guidance (WHO 1999, 2000) and the European Commission's ExternE Study (EC 2000). In this paper, RR ratios associated with particulates have been adopted from the Künzli's et al three European country study.

The choice of overseas-derived RR ratios ultimately raises two key questions. Are these overseas RR ratios appropriate for Australia, and why have Australian RR ratios not been used? These questions will be discussed after we address the issue of choice of pollutant.

PM_{10}, NO_2, SO_2 and CO are among the major transport emissions for which epidemiological studies have derived RR ratios. Ideally, the best approach would be to estimate the number of persons affected using each pollutant's derived RR ratios and then sum the outcomes to obtain the total number of health outcome cases (mortality and morbidity). Unfortunately, it has been shown that such an approach would grossly overestimate the heath effect or number of cases. The reason is that almost all the pollutants are found to be correlated with each other (Morgan et al 1998). That is, they affect and interact with each other and 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 observed effects to single pollutants.

Although the literature suggests that the statistical analysis underlying epidemiological studies has established strong correlations between various pollutants and health outcomes, 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/toxicology and it is an area of intense and on-going research. Given that a pollutant by pollutant assessment of health effects would grossly overestimate the outcome, there has to be a surrogate pollutant that can capture some or all of the effects of all the other pollutants. WHO (2000) recommends PM_{10}. or sulphur dioxide as a useful indicator of the health risk of transport emission pollution. Given the available epidemiological and monitoring data, PM_{10} has been used in this paper as the main indicator of air pollution.

2.2 Applicability of Overseas Studies

A number of epidemiological studies have been undertaken in Australia using air pollution data sampled in major urban centres, including, Sydney, Melbourne and Brisbane. The results of these studies are robust and they are consistent with other studies carried out overseas (Denison et al 2001 and Morgan et al 1998). However, all of these 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 obtained (regression coefficient is synonymous with RR ratio), measures the short-term health impact - the proportional increase in the daily health effect (for example, 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 (say, death) are closely connected in time. In the case of reduced life expectancy due to long term morbidity advanced by air pollution, time-series analysis as reported in the literature is unlikely to capture this effect. Although there are advanced econometric techniques that can be used on time-series data to derive both short-term and long-term coefficients, the epidemiology literature reveals a limited application of these techniques for the estimation of regression coefficients (Zeger et al 1999, and Schwartz 2000). Künzli et al (2000) estimated that the use of short-term time -series based regression coefficients is likely to underestimate mortality by about 4-5 times. Hence, conceptually, it is inappropriate to use short-term relative risk estimates for the analysis of long-term impacts.

On the other hand, the Künzli-derived RR ratios adopted for this study are based on meta analysis of cohort-based long-term US 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 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. The RR ratios derived from cohort studies, therefore, capture both the short-term and long-term health impacts.

Perhaps the key issue is not whether air pollution studies are time-series or cohort but rather whether US 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 RR ratio. Second, the hypothesis underlying the statistical analysis assumes that the characteristics of the study population and the mixture of pollutants used to derive the regression coefficient 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 US. Estimated RR ratios from Australian studies provide evidence that this is the case (Denison et al 2001).

The statistical techniques employed ensure that other confounding factors such as temperature, age, sex, smoking etc are accounted for in the modelling. In other words, the modelling attempts to isolate the effects of the pollutant from all known confounding factors. In practice, 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 RR ratios used by Künzli et al (2000) can be appropriately applied in the Australian context.

3 DATA

In this paper the analysis is based on 2000 data. The choice of 2000 is primarily to ensure consistency of the data 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 (AIHW). Typically, some ABS and AIHW data sets have a lag of 1 to 2 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.

Table 4 shows the exposure-response functions expressed as relative risk (RR) per $10\mu g/m^3$ (Künzli et al 2000). A relative risk estimate of 1.043 or 4.3 per cent (ie 1.043-1) for total mortality can be interpreted as follows: for every $10\mu g/m^3$ increment in PM_{10}, the number of cases of premature mortality increases by 4.3 per cent. For the theoretical basis of this interpretation, see for instance Künzli et al 2000, Morgan et al 1998 and Pope et al 1995.

Measurement of the proportion of particles in the atmosphere that is attributable to motor vehicle emissions is almost impossible because of multiple sources of particle emissions. The relative contribution of each source/type varies from day to day, depending on meteorological conditions and the quantity of emissions from mobile and static sources (see Fisher et al 2002). In practice, air quality monitoring stations sample or capture total PM_{10} from all sources including transport. It is from this measured total particulate that the transport proportion has to be determined. The derivation of the transport proportion is, therefore, fraught with difficulties.

Notwithstanding these difficulties and uncertainties, attempts have been made to estimate the transport proportion of emissions (BTRE 2002, 2003 and National Pollution Inventory 2003). The National Pollution Inventory (NPI) web site (www.npi.gov.au) provides details on the methodology used by some jurisdictions to estimate transport related proportions of pollutants. In this paper, we have assumed that the proportions shown in the NPI data (in kg) for particulate matter 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 (see table 6 for motor vehicle proportions).

For consistency across all cities and also because paved road particles consist largely of wind-blown dust, the motor vehicle proportions have been re-estimated by excluding paved road particles from the aggregate estimates provided at the NPI web site. As shown in table 5, there is wide variation in the motor vehicle proportion of PM_{10}. This is largely due to the differing sources used by each jurisdiction and variation in population densities and methods of estimation. Therefore, caution is required when comparing these proportions.

TABLE 4: HEALTH OUTCOME RELATIVE RISK RATIOS PM_{10}

Health outcome	*Relative risk estimate associated with a 10 µg/m³ increase in PM_{10}*	*95% confidence interval*
	Base Case	Lower - 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

3.1 PM_{10} 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 (1997) outlined some of the factors that may require consideration in the assessment of personal exposure levels. The populations affected by particulate levels measured at each monitoring station were estimated using the ABS CDATA software. The software uses a GIS base data linked to the Australian census data. Using information on longitude and latitude for each monitoring station, the estimated population within a 2-kilometre radius was collected and assumed to be the directly affected population. To obtain citywide population particulate exposure levels, the populations within a 2-kilometre radius of each monitoring station were used as weights in the following formula:

$$PM_{10wa} = \frac{\sum_i PM_{10i}(pop_i)}{\sum_i pop_i}$$

where,
PM_{10wa} is PM_{10} weighted annual average, PM_{10i} is PM_{10} concentration in cell (area) i and pop_i is the population in cell (area) i.

The weighted average for all monitoring stations in a city was then assumed to be the citywide population exposure level.

3.2 Health Outcome Cases

Data on frequency of health-related cases were obtained from the Australian Institute of Health and Welfare. Table 6 shows the data for each health outcome for the year 2000. For total deaths from non-external causes, some experts (WHO 2000) believe that data

on all-cause mortality are more reliable with respect to both classification and registration than data on cause-specific mortality. 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, it has been suggested that relative risk estimates for all-cause mortality should always be used when available (WHO 2000). This is the approach adopted in this paper. It is also important for the reader to note that the mortality data used in this analysis include only people aged 30 or more.

TABLE 5: ANNUAL AVERAGE PM_{10} EXPOSURE LEVELS - ALL SOURCES AND VEHICLE SHARES[1]

Location	*PM_{10} $\mu g/m^3$*	*Vehicle proportion %*
Sydney	18.0	43
Melbourne	18.0	33
Brisbane	18.7	31
Adelaide	19.0	19
Perth	18.8	20
Hobart	16.0	7
Canberra	16.3	12
Darwin	14.9	37

Sources: BTRE estimate derived from NPI data

TABLE 6: HEALTH OUTCOME CASES IN CAPITAL CITIES (2000)

Health outcome	Cases
Total mortality (adults ≥30 years, excluding traumatic or external causes)	70 568
Respiratory hospital admissions (all ages)	160 961
Cardiovascular hospital admissions (all ages)	224 820
Chronic bronchitis incidence (adults ≥25 years)	990
Bronchitis episodes (children <15 years)	250
Asthma attacks (children <15 years)[a]	11 937
Asthma attacks (adults ≥15 years)[a]	11 884

[a] Total person-days per year with asthma attacks

Source: AIHW

4 CALCULATIONS

In the assessment of the number of health cases attributable to air pollutants, four data components are required:

1. Exposure - response functions
2. The frequency of health outcome (ie the prevalence)

[1] Details on the vehicle proportion estimate will be provided in the forthcoming BTRE study. report. All published BTRE reports are available on the web at www.btre.gov.au.

3. Level of exposure
4. Population.

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:

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

where,

Po = baseline mortality
Pe = the observed mortality in the population (age > 30)
Eo = observed mean PM_{10} exposure level
B = threshold PM_{10} exposure level for mortality effect
RR = relative risk for a 10μg/m^3 increment of PM_{10},

The increased mortality attributable to a 10μg/m^3 increase in PM_{10} is then calculated:
D_{10} = Po * (RR – 1)
where,
D_{10} = the number of additional deaths per 1 million people to the baseline mortality for a 10 μg/m^3 increase in PM_{10}.

Finally, we get the total numbers of PM_{10} induced deaths:
Nc = D_{10} * Pc * (Eo – B) / 10
where,
Nc = the number of deaths due to PM_{10} for the population
Pc = the population.

4.1 Model's Key Assumptions

Although there is no empirical evidence on a PM_{10} threshold level beyond which health effects start to occur, it may be expected that there is a natural PM_{10} which is emitted independent of human activities. Natural sources of PM_{10} include sea salt aerosols (chloride concentration), wind-blown mineral dust, and biological debris such as pollen. Most wind-blown dust and pollen is likely to be in excess of 10 μm in diameter and is unlikely to be sampled. It has been assumed that the natural level of PM_{10} is 5 μg/m^3. However, to address the other view that there is no threshold, a sensitivity analysis has been carried out with no threshold. Other studies, such as those referenced in this work, have used values between 0 and 7.5 μg/m^3 as thresholds. The threshold implies that the attributable level of PM_{10} has to be net of the respective level of natural PM_{10} to accurately measure the health effect of motor vehicle emissions. 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. Overall, the approach to modelling assumes the 'at least' approach: that is, calculations are aimed at estimating the impacts which may be expected to be 'at least' attributable to air pollutants.

The model was programmed into a spreadsheet and the data sets discussed above were used as inputs. The results of the calculations are shown in tables 7 and 8 and are the respective number of people in Australia dying or suffering adverse health effects from non-external causes that may be associated with motor vehicle pollution. The total number of deaths estimated for all capital cities is around 1200 in the year 2000. Sydney recorded the greatest number of deaths followed by Melbourne. This result was

expected, as Sydney and Melbourne have the largest populations and highest traffic levels. In addition, motor vehicle pollution accounted for around 2400 hospital admissions and 21000 days of asthma attacks in the same year.

TABLE 7: HEALTH CASES OUTCOME- MORTALITY (VEHICLE SHARE)

Capital Cities	*Base Case*	*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	1 228	758	1 703

Note: lower and upper are 95 per cent confidence intervals

TABLE 8: HEALTH CASES OUTCOME –MORBIDITY (VEHICLE SHARE)

	Morbidity Cases			*Asthma Attacks (No of days)*		
City	Base case	Lower	Upper	Base case	Lower	Upper
Sydney	1 085	373	1 778	7 441	4 230	10 591
Melbourne	693	244	1 129	9 025	4 833	13 074
Brisbane	326	114	532	2 810	1 513	4 060
Adelaide	170	57	280	922	513	1 318
Perth	167	57	274	890	500	1 270
Hobart	10	4	16	3	2	5
Darwin	9	3	15	5	3	8
Canberra	na	na	na	na	na	na
All capital cities	2 460	851	4024	21 095	11 594	30 325

na= not available. ACT morbidity data was not available

5 ECONOMIC VALUATION

Society imputes economic values to premature deaths or disability/impairment caused by human activities such as traffic pollution or road crashes injuries. The conceptual basis is that premature death or impairment deprives the community of the services of the victims. Also the victims 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 victims (see, for example, BTRE 2000 and 2003). The impact of transport emission pollutant, or indeed any premature death or impairment, is equally subject to the same concept. Economists value these economic losses using two conceptual approaches, namely ‘human capital’ and ‘willingness to pay’. These two approaches are mutually exclusive, even though they are significantly complementary.

5.1 Human Capital Method

The human capital approach seeks to measure the economic impact through the loss of output or productivity of victims. This is generally done by calculating the present value of the victim's potential future output, as measured by the victim's discounted anticipated stream of earnings. Essentially, this method calculates and values the years of life lost due to mortality (YLL). 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, and resource costs including medical and hospital costs. The inclusion of lost quality of life is done in such a way that double counting is avoided.

5.2 Willingness to Pay Method

The willingness to pay method attempts to capture trade-offs between wealth and risk. In other words, it estimates the value of life in terms of the amounts that individuals are prepared to pay to reduce risks to their lives. The approach uses people's preferences (either stated or revealed) to ascertain the value they place on reducing risk to life and reflects the value of intangible elements, such as quality of life and joy of living. To this base cost are added net lost output, medical costs, administrative costs, etc, which are values of human capital. Generally, the willingness to pay approach yields values far higher than those based solely on the human capital approach.

5.3 Assessing the Two Approaches

Both the human capital and willingness-to-pay approaches are imperfect in estimating the value of 'statistical life'. 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 in order to reduce the risk of being injured or killed and 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.

The human capital approach has been criticised on two basic grounds:

1. It assumes full employment in the economy, in that it attributes expected future income to all victims. To counter this, criticism, the BTE (1998) introduced the concept of probability and labour force participation rate that ensures that the probability of victims being in future formal employment or unemployed is taken into account.
2. It does not provide an accurate measurement of the intrinsic value in cases where there is loss of life or suffering, and involves an accounting approach. To counter this, the BTRE estimated 'quality of life' values using 'non-economic' Australian court awards as a proxy. Court awards are seen as reflecting society preferences. This is particularly so, given the recent public outcry over perceived excessive court awards. The public reaction appears to have led to a reduction in the amounts being awarded. By excluding economic awards, double counting is avoided and the non-economic values based on court awards 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 and the results are easily replicated. The values are sensitive to the discount rate. A review of the literature by a European expert group (ECMT 2001) could not settle firmly on either method. A review by Trawen et al (2002) found that the application of these two methods is fairly even in developed countries, although there is a shift towards the willingness to pay approach due to recent advances in the technique.

In this paper the human capital method has been applied on the basis that these costs can be compared with existing estimates of road trauma (BTRE 2000).

A recent BTRE study (BTRE 2003) estimated the value of a statistical life (VOSL) at $A1.9 million in 2000 values. The recommended VOSL for Europe is 1.1 to 1.3 million euros in 2000 values (ECMT 2001). At the current exchange rate of (€ 1=$A1.7437) these are equivalent to $A1.9 million to $A2.3 million. The estimate of VOSL of $A1.9 million is based on the profile of transport accident victims (in this case, rail accident victims). Transport accident victims tend to be in the younger age group, particularly motor vehicle victims. For these and other reasons, it has been argued that the estimated VOSL may not be representative of pollution victims as these victims tend to be in the age bracket of the over 60's. Even in the situation where pollution-related deaths occur at younger ages, some research suggests that the pollution effect has probably advanced an existing health condition and, thus, life is shortened by a few years or months.

However, it is important to note that premature pollution-related deaths are anonymous: that is, they involve statistical cases which have been drawn from the total Australian non-traumatic deaths register. Therefore, individual persons with their unique characteristics can not be identified and, so, their precise years of life lost can not be determined. It is also worth pointing out that the sampling for pollution mortality in this study is from age 30. Although road deaths involving the 17 to 25 age bracket receive more media attention due to their large numbers relative to their population size, the dominant age group for motor vehicle accident deaths in terms of absolute numbers is the 26s to 59s (ATSB 2003).

The BTRE has used the human capital method to separately estimate VOSL for aviation, road and railway and maritime accident victims. All of the results are consistent and similar. The profiles of all transport accident victims extend across the spectrum of the Australian population. Notwithstanding these issues, a close examination of motor vehicle crash-related deaths indicates that over 60 years olds' accounts for less than 30 per cent of total accident victims; while in the case of pollution-induced deaths, the same group accounts for more than 60 per cent. In order to address this apparent difference in the age distribution, the estimated VOSL of $A1.9 million has been adjusted down to $A 1.3 million. This, we believe, at least partially addresses the different age distribution of the exposed population. This is an approximate adjustment because there is no precise way to address the issue. Even using the years-of-life-lost approach does not address it adequately. Depending on how the issue is conceptualised, this adjustment may still result in an over estimation for example, if it is considered that lives are being shortened by a few years (say 2-3). On the other hand, the human capital method generally produces values at the lower end of the scale and thus, if the willingness to pay method were the basis of estimation, the calculated values would have been much higher. Another related issue is that pollution victims may suffer over longer periods than road crash victims. The costs associated with long term illnesses are addressed and captured in the morbidity costs. The medical and hospital costs are calculated over the expected life span of victims.

5.4 Application to mortality cases

To obtain the economic costs of the health outcome, the VOSL ($A1.3 million) is applied to mortality cases estimated as shown in table 7. The results of this calculation, (that is,

the total economic losses attributable to deaths in 2000 likely to have been caused by traffic emissions in Australia) are shown in table 9.

5.5 Application to morbidity cases

In the case of morbidity, it is implied that death does not occur as a result of air pollution exposure. Rather, the quality of life and productive capacity of the victims are impaired or reduced. The economic costs, therefore, arise as a result of temporal loss of productivity, loss of quality of life and the resource costs (such as hospital and medical costs). A number of techniques have been used for the assessment of the burden of diseases. These include the use of 'years of life lost' (YLL), equivalent 'healthy' years of life lost due to disability' (YLD) (Brunekreef 1997, Robins et al 1999, Mathers et al 1999), and 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 to include equivalent 'healthy' years of life lost by virtue of being in states other than good health. One DALY is a lost year of 'healthy' life and is calculated as the sum of years of life lost due to premature mortality (YLL) and equivalent 'healthy' years lost due to disability (YLD). That is, DALY =YLL+YLD.

In this paper, the economic costs of mortality are estimated separately from morbidity. Therefore, the use of DALY to estimate the burden of morbidity presents the potential for double counting since DALY by concept includes the estimate of premature mortality. Because of this potential for double counting, we have adopted the YLD approach for the estimation and costing of morbidity. Mathers et al (1999) discussed this approach in detail and we have drawn on these results in this paper.

Mathers et al (1999) reported that in 1996 approximately 9 per cent of total life expectancy at birth is 'lost' due to disability for both males and females in Australia. The same report indicated that asthma is responsible for 4.8 per cent, bronchitis 3.4 per cent, respiratory illnesses 1.2 per cent and cardiovascular 8.9 per cent of years of life lost due to disability in Australia. Using the ABS Australia 1999 life expectancy tables, these percentages, and the hospital admission data[2] summarised in table 5 (the full hospital admission data is presented by age group eg 0-4, 5-9 etc), the years of life lost due to disability are estimated. The YLD analysis requires separate individual admissions records. The data were 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 7) and a male life expectancy of 69.81 year was used; for asthmatic patients, this will be further reduced by 4.8 per cent.

The economic cost of morbidity is then simply estimated as
$M_d = YLD * V_{ly}$
where, M_d *=morbidity cost and* V_{ly} *is value per healthy life year lost*

This approach implicitly assumes YLD is associated with zero productivity (that is, time lost due to illness is assumed to produce no economic value) and by implication accounts for productivity losses due to 'Restricted Activity Days' (RAD). For this reason, the values associated with 'Restricted Activity Days' have not been analysed

[2] Hospital admission data are in effect hospital separations. Admissions and separations are not necessarily the same thing. Separation counts the number of times a particular health case is presented at a hospital irrespective of the patient. In other words, the same person can be counted several times within a year

independently of YLD. Either RAD (converted to years) or YLD can be used in this approach but not both. The estimated YLDs are adjusted to reflect the respective transport proportions. Using the human capital approach (discussed earlier), it is estimated that the value per 'healthy' year of life lost is $A50,000. The total economic costs associated with morbidity are shown in table 9.

The economic analysis shows that, overall, the economic burden resulting from traffic pollution in Australian capital cities amounts to about $A3.3 billion. The burden range, within a 95 per cent confidence interval, is between a lower value of $A2.7 billion and an upper value of $A3.9 billion.

TABLE 9: TOTAL ECONOMIC COSTS –(MOTOR VEHICLES SHARE $A MILLION)

Capital Cities	*Mortality*			*Morbidity*		
	Base case	*Lower*	*Upper*	*Base case*	*Lower*	*Upper*
Sydney	713	441	990	785	782	788
Melbourne	448	276	621	466	465	468
Brisbane	197	122	273	223	222	223
Adelaide	113	70	156	109	109	110
Perth	104	64	144	110	110	111
Hobart	8	5	11	7	7	7
Darwin	5	3	7	10	10	10
Canberra	8	5	12	na	na	na
All capital cities	1 596	986	2 214	1 712	1 705	1 718

5.6 Sensitivity analysis

To test the robustness of the key assumptions underlying this analysis, sensitivity analysis was carried out by varying the key assumptions under different scenarios. The base case includes an analysis of lower and upper cases at 95 per cent confidence intervals. The variations were done one at a time; that is, when one variable is changed all other variables are held constant at the base case values. The key sensitivity assumptions are:

- PM_{10} threshold levels varied from the base case of 5 µg/m^3 to 0 µg/m^3
- PM_{10} motor vehicle proportion are varied from the base case upward to equal 45 per cent and a lower scenario by varying all proportions upward or downward to equal 20 per cent.
- The value of a statistical life (VOSL) remains unadjusted at $A1.9m.

Overall, the sensitivity analysis suggests that, when no threshold is assumed, the economic cost is increased by about 18 per cent from $A3.3 billion to about $A3.9 billion. Setting traffic pollution proportions for all cities to 45 per cent resulted in an increase of about 39 per cent in the total economic costs: that is, from $A3.3 billion to $A4.6 billion. Setting traffic pollution proportions for all cities to 20 per cent results in about a 39 per cent decrease in the total economic costs: that is, from $A3.3 billion to $A2 billion. When the VOSL remains unadjusted at $A1.9 million the economic cost totals $A4 billion that is, about 21 per cent above the base case.

Table 10 shows the population used in the analysis and the attributable transport related deaths for all capital cities for the same year (2000). It shows that the estimates of pollution-induced deaths in capital cities exceed the number of capital city road fatalities. For Australia as a whole, road fatalities in 2000 totalled 1 800.

TABLE 10: CAPITAL CITY POPULATION VEHICLE POLLUTION INDUCED DEATHS AND ROAD FATALITIES (2000)

City	*Total population*	*Pollution induced deaths (range)*		*Road fatalities*
Sydney	3 502 301	339	762	267
Melbourne	3 160 171	213	478	230
Brisbane	1 508 161	94	210	44
Adelaide	1 002 127	54	120	70
Perth	1 176 542	49	111	91
Hobart	126 048	4	9	5
Darwin	71 347	2	5	19
Canberra	309 799	4	9	14
All Capital Cities	11 856 496	758	1 703	740

Sources: BTRE and ATSB

6 CONCLUSIONS

The analysis in this paper has shown that the probable motor traffic pollution related deaths in Australia capital cities in 2000 amounted to about 1200 persons or between a lower figure of 758 and an upper figure of 1700. In addition, there were 2400 hospital cases and 21000 days of asthma attacks. The economic analysis shows that, overall, the economic burden resulting from the health effects of traffic pollution in Australian capital cities in 2000 amounted to about $A3.3 billion. The burden range, within a 95 per cent confidence interval, is between a lower value of $A2.7 billion and an upper value of $A3.9 billion.

Obviously, there are some uncertainties about the key assumptions underpinning the analysis. Although attempts have been made to address the limitations (sensitivity analysis), some observers may be tempted to argue that perhaps it is better not to calculate the costs until the methods are better defined. However, the overwhelming scientific evidence is that current levels of air pollution have adverse health effects. Thus, the impact cannot be zero. Desisting from this type of analysis, under the pretext of uncertainties, would promote decisions without consideration of aspects of public health. This is particularly true for environmentally sensitive decisions. For example, transport infrastructure investment analysis would continue to under-estimate the cost of externalities in cost-benefit studies.

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