

## The health costs of traffic related air pollution in Perth

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### Abstract

Not all costs connected with transport are fully covered by the purchase or running costs of the vehicle. There are numerous external costs resulting from air pollution, traffic accidents and noise that result in a measurable financial burden to society. The costs associated with the health effects of air pollution make up the majority of this financial burden. Society effectively subsidises the cost of transport through increased hospital admissions, lost productivity, chronic illnesses and early mortality.

If a government is to make informed transport policy decisions it is important that they have an understanding of the health effects of transport emissions and how these effects add to the real cost of a particular transport option.

This paper will examine several different methods of calculating the health costs of air pollution. It will compare the results that are obtained by these different methods and discuss the advantages and disadvantages of each. A cost per kg of air pollutants in Perth will be developed and the magnitude of these costs over the life of a vehicle will be illustrated by comparing them to the private operating costs of a diesel and a CNG bus.

### Introduction

This paper describes part of a larger research effort being conducted under the Western Australian Governments Sustainable Transport Energy for Perth (STEP) program that seeks to analyse different transport options by comparing the social and private through life costs. The primary difference between social and private costs is that social costs include external costs that result from the particular activity but that are not paid directly by the operator. The external costs of transport include air pollution effects, traffic accidents, noise etc.

Air pollutants from vehicles are a complex mix of chemicals that change as they travel from the source of the emission. They include particulates, oxides of sulphur, oxides of nitrogen, CO, CO<sub>2</sub>, ozone and hydrocarbons such as methane. These pollutants impose a financial burden on society through increased health costs and damage to buildings, crops and natural fauna. CO<sub>2</sub> and methane also contribute to global greenhouse gases.

European studies (Bickel, Schmid et al. 1997) have concluded that the damages related to transport emissions are dominated by the cost of adverse health impacts. The real monetary costs of these damages needs to be known if they are to be included in the life cycle costs of different vehicles.

Three methods of calculating the health costs are discussed in this paper. All three follow similar steps in that they first prioritise pollutants according to greatest health impact, then define each impact in terms of its health outcome and responsible pollutant and finally quantify the economic costs.

The approaches discussed in this paper are:

- a top down approach that identifies the total cost of several pollution related health outcomes and then allocates the cost of those outcomes to each type pollutant based on the amount of each pollutant emitted into the atmosphere;
- a bottom up approach that uses the relationship between the atmospheric concentrations of different pollutants and various health outcomes to determine economic costs; and
- a second bottom up approach that uses a basic model of the behaviour of different pollutants in the atmosphere and concentration-response relationships to estimate the health cost of each kilogram of pollutant.

## **Emissions**

Road transport is responsible for a range of emissions that adversely affect human health. Air contaminants are produced primarily through the combustion of fuel but also originate from other vehicle components such as brake and clutch linings and pads, tyres and fuel tanks, and from road-surface wear.

The European commission has conducted a series of studies to determine the external costs of energy (ExternE) including that used in transport. These studies have shown that social costs of transport emissions are dominated by health impacts, particularly due to primary (PM<sub>2.5</sub>) and secondary particulates (sulphates and nitrates). Carcinogens such as benzene and toluene have a high specific toxicity but proved to be of much lower importance because they are emitted only in very small quantities (Bickel, Schmid et al. 1999).

When considering the emissions that result from transport it is critical that the complete fuel life cycle is considered. Beer on behalf of the AGO has conducted a comprehensive study of life-cycle emissions of alternative fuels for heavy vehicles (Beer, Grant et al. 2001). In this study Beer considers greenhouse gases, air pollutants, and air toxics that are produced not only as direct emissions from vehicles but also those associated with the fuel's extraction, production, transport, processing, conversion and distribution. Beer identifies CO, NO<sub>x</sub>, SO<sub>2</sub>, non-methane volatile organic compounds (NMVOC) and particulates as the key air pollutants that impact on human health.

The National Environment Protection Council identifies particulates, CO, NO<sub>2</sub>, SO<sub>2</sub>, ozone and lead as the six criteria air pollutants primarily responsible for Australian health effects such as daily mortality, hospital admissions and emergency department attendances (National Environment Protection Council 2003, p.7). Of these six, motor vehicles are the key sources of CO and NO<sub>2</sub> in urban areas. Motor vehicles are no longer a major source of lead due to the introduction of unleaded fuels (National Environment Protection Council 1998, p.7).

## **Health effects**

Air pollutants cause adverse effects on human health if they are present in air at sufficient concentrations and for a sufficient length of time. Health effects associated with the six common pollutants include respiratory effects, ranging from minor symptoms such as a cough to the more serious, eg chest congestion and asthma, to the very serious such as chronic illness and possibly death. Where a relatively minor symptom occurs the aggregate effect can often be very debilitating, particularly for susceptible subgroups (National Environment Protection Council 1998).

The health effects of the air pollutants are quantified mainly through population-based epidemiological and controlled human exposure studies as well as animal toxicological studies. A World Health Organisation (WHO) review of these studies indicates that transport-related air pollution contributes to an increased risk of death, particularly from cardiopulmonary causes. It also increases the risk of respiratory symptoms and diseases that are not related to allergies. European estimates indicate that the number of deaths per year attributable to transport-related air pollution is similar to the death toll from traffic accidents (Krzyzanowski 2005).

The National Environment Protection Council provides details of the health effects of each of the six key pollutants (National Environment Protection Council 1998). A summary of the relevant pollutants, and their effects, is provided in the following paragraphs.

## **Carbon Monoxide**

Inhaled Carbon Monoxide (CO) combines with haemoglobin, the blood's oxygen-carrying molecule, inhibiting the molecules ability to carry oxygen. If sufficient haemoglobin molecules are affected the ability of the blood to carry oxygen is impaired. The brain, nervous tissues, heart muscle and some other specialised tissues which require large amounts of oxygen may not receive enough to function optimally, and may suffer temporary or permanent damage. In healthy adults exposure to CO can cause reduced work capacity and a significant

decrease in work time. It can also have neurobehavioral effects such as decreased visual perception, manual dexterity and an ability to learn. CO exposure has also been linked to decreased birth weight in non-smoking mothers.

### **Nitrogen dioxide**

Nitrogen dioxide (NO<sub>2</sub>) affects health directly by causing inflammation within the lungs; and indirectly by impairing the immune defence mechanisms in the lungs. Young children are especially susceptible to the indirect effects, which potentially increase respiratory infections.

NO<sub>2</sub> appears to contribute both to morbidity and to mortality particularly in young children, asthmatics, and in individuals with chronic inflammatory airway disease. Studies carried out in Sydney have shown an association between hospital admissions for respiratory and cardiac conditions and ambient NO<sub>2</sub> levels.

### **Ozone**

Ozone (O<sub>3</sub>) is the principal component of photochemical smog which is a complex mix of chemicals produced in the atmosphere by the action of sunlight on other pollutants such as oxides of nitrogen (NO<sub>x</sub>) and other reactive organic compounds (ROC). Up to 80% of the NO<sub>x</sub> emissions in Australian cities come from motor vehicles.

Measurements of the photochemical oxidants that make up smog are usually referenced to ozone i.e. although measurements are specifically of ozone they are taken to be representative of all photochemical oxidants. Also present are peroxyacetyl nitrate (PAN) and aldehydes such as formaldehyde.

Ozone is a strong, highly reactive, oxidising agent that can cause significant irritation of the respiratory tract and mucous membranes. The range and severity of the effects on health are dependent on the pollutant concentration, exposure duration, and individual sensitivity. Exposure to ozone can cause minor changes in lung function and increased symptoms consistent with airway irritation, leading to increased requirement for medication and medical and hospital services. There is also some evidence that ozone may be associated with increased mortality from cardiovascular causes, especially in the elderly. Exercise enhances the effects of ozone on lung function.

Since ozone is a secondary pollutant formed by the action of sunlight on primary pollutants, the control of ozone requires management strategies that reduce in precursor primary pollutants such as NO<sub>x</sub> and ROCs.

### **Sulphur dioxide**

Sulphur dioxide (SO<sub>2</sub>) is a colourless, pungent, irritating and reactive gas which is soluble in water. SO<sub>2</sub> acts directly on the upper airways (ie nose, throat, trachea and major bronchi) resulting in symptoms such as wheezing, chest tightness, shortness of breath or coughing. Exposure to SO<sub>2</sub> can result in reduced lung volume (National Environment Protection Council 1998).

Sulphur dioxide is rarely of concern in urban Australia except near petrochemical industrial areas (BTRE 2005).

### **Particulates**

Airborne particles are very diverse in their chemical composition and physical properties. They can be emitted directly from the exhaust as a primary pollutant or develop in the atmosphere as a secondary pollutant from the reaction of other emissions.

Particles can be referred to as total suspended particles (TSP) or as black smoke (soot), but most commonly particulates are classified according to their size which ranges from 0.005 micrometre (µm) to about 100 µm in diameter. Common size-related terms are PM<sub>10</sub> for particles less than 10µm, PM<sub>2.5</sub> for particles less than 2.5µm and 'inhalable' or 'respirable' particles.

Respirable particles are smaller than inhalable and can penetrate more deeply into the lungs. These smaller or fine particles have been associated with a wide range of respiratory symptoms. Long and short term exposure

to such particles has been linked to increased deaths from heart and lung disease. No safe minimum exposure for the effects of particles has been identified.

Particles can also carry carcinogens, such as polycyclic aromatic hydrocarbons, into the lungs. The elderly, children, and people with respiratory infections or lung or cardiovascular disease are particularly susceptible to the effects of airborne particles (National Environment Protection Council 1998).

Secondary particulates such as sulphates, which derive primarily from SO<sub>2</sub> emissions, nitrates, which derive primarily from NO<sub>2</sub> emissions and organic aerosols, which derive primarily from VOC emissions can also contribute significantly to particle levels (National Environment Protection Council 1998).

A summary of the health effects of major air pollutants is provided in Table 1.

Primary Pollutants	Secondary Pollutants	Impacts
Particulates (Black smoke or soot, PM <sub>10</sub> , PM <sub>2.5</sub> )		cardio-pulmonary <b>morbidity</b> (cerebrovascular hospital admissions, congestive heart failure, chronic bronchitis, chronic cough in children, lower respiratory symptoms, cough in asthmatics, <b>mortality</b> : reduction in life expectancy due to short and long term exposure
CO		<b>morbidity</b> (cardio-vascular), <b>mortality</b> (congestive heart failure)
SO <sub>2</sub>		cardio-pulmonary <b>morbidity</b> (hospitalization, consultation of doctor, asthma, sick leave, restricted activity), <b>mortality</b>
SO <sub>2</sub>	sulphates	as for particulates
NO <sub>x</sub>		<b>morbidity</b> (respiratory, eye irritation)
NO <sub>x</sub>	nitrates	as for particulates
NO <sub>x</sub> +VOC	ozone	<b>morbidity</b> (respiratory hospital admissions, restricted activity days, asthma attacks, symptom days), <b>mortality</b>
PAH (polycyclic aromatic Hydrocarbons) includes diesel soot, benzene, 1,3,-butadiene		cancers
dioxins		cancers
As, Cd, Cr, Ni		cancers and other morbidity
Hg, Pb		<b>morbidity</b> (neurotoxic)

**Table 1 - Health effects of air pollution (Friedrich, Rabl et al. 2001)**

Often, the effects observed in epidemiological studies cannot be attributed to the specific pollution indicator used in the study, but can be attributed to a mixture of pollutants. Fine particulates and ozone are associated with increased risks of mortality and respiratory morbidity, while allergic responses have been linked to exposure to nitrogen dioxide, ozone and particulates.

This accumulated evidence allows the hazards of transport related air pollution to be identified, but makes only a limited contribution to the quantitative assessment of the adverse effects on health and to the prediction of the benefits of reducing a particular part of the total air-pollution mix. More research is needed on the patterns and the adverse health effects of population exposure and on the role due to the different components of the pollution mix (Krzyzanowski 2005).

## Health Impacts

The aim of this economic valuation is to determine the financial cost to society of health effects of pollution. The choice of which health outcomes to include in a cost study is based on whether the impact can be assigned a monetary value and whether the impact can be reasonably attributed to a single pollutant. The second condition is important to avoid double counting of impacts which would cause an over estimation of the costs.

The economic value can be determined by using a top down approach which takes the total cost of a particular health impact, such as early mortality, and calculates what portion of that cost can be attributed to a particular pollutant. An alternative to this is a bottom up approach which uses the relationship between the ambient concentration of a pollutant and a particular health outcome to estimate the number of cases of that outcome that can be linked to pollution levels. The cost to society is then determined by multiplying the predicted number of cases by the cost of each case. This is repeated for each identified health outcome.

An example of a top down approach is that used by Beer in his paper presented to the 16<sup>th</sup> International Clean Air and Environment Conference in 2002 (Beer 2002). Beer's approach was to first identify the key pollutants that present the greatest risk to human health and the total cost, across Australia, of the different health impacts. The pollutants and health impacts that Beer included in his study were taken from the National Environment Protection Council's Ambient Air Quality National Environment Protection Measure (NEPM) (National Environment Protection Council 1998) and are listed below:

- CO – Loss of 1 day's earning for 50,000 people at a cost of A\$6 million.
- NO<sub>2</sub> – 10 to 15% of the population display respiratory symptoms at a cost of A\$5 million.
- O<sub>3</sub> – Up to 10 deaths per year in Australia, with total (mortality and morbidity) costs of A\$810 million.
- PM<sub>10</sub> - Up to 2,400 deaths per year in Australia, with an associated health cost of A\$17.2 billion.
- SO<sub>2</sub> – 20% of susceptible individuals (15% of the population) display respiratory symptoms at a cost of A\$1.4 million.

These total health costs are the result of pollution from all sources including power generation, industrial processes, air, sea and land transport etc. Beer used the ratio of transport emissions to total emissions to calculate the proportion of the total health costs that can be attributed to road transport.

He then used the mass of each pollutant produced by road transport each year to calculate the health costs associated with each tonne of pollutant. The results are provided in Table 2.

	PM <sub>10</sub>	NMHC	NO <sub>x</sub>	CO
Upper Bound	221,143	72,482	900	9
Best Estimate	147,429	19,331	870	3
Lower Bound	108,296	11,665	277	2

**Table 2 - Top down valuation of pollutants (\$/tonne)**

The advantage of this approach is that the total cost of each health affect is accounted for, but there can be a large degree of uncertainty associated with the total health costs and the proportion that can be attributed to an individual pollutant. The estimates are also based on historical data and may not accurately represent the marginal cost of current or future emissions.

A further disadvantage is that local factors such as population density, weather, geography and even local health care costs are not considered. European studies have found that the damages per tonne of pollutant were very case specific and were dependant on the location of the plant and the height of the stack (Pingoud, Malkki et al. 1997). Spadaro also highlighted these local effects and found that the effects of weather, stack height, flue gas exit temperature, exit diameter and exit speed all affected the damages from a particular site (Spadaro and Rabl 1998). Spadaro also notes that receptor density (i.e. population density) had the greatest impact on damages and that changes in population density can cause the damages due to primary pollutants

to vary by an order of magnitude. By comparison, for a given population stack height could vary the damages by a factor of up to two.

Bottom up or impact pathway studies attempt to quantify the damage cost of each pollutant based by tracing the pollutant from where it is emitted to the affected receptors, in this case the population. This approach determines the relationship between micro level burdens of pollutant and environmental impacts through the application of damage functions.

The relationship between concentrations of air pollutants and a particular health impact are determined primarily through epidemiological studies but are also drawn from controlled human exposure studies, and animal toxicology. Epidemiological studies examine the relationship between air pollution exposure and health effects in the community which can be acute or chronic (long-term) effects. Several types of epidemiological studies are used worldwide to examine the associations between exposure to air pollution and adverse health effects. Much of the data that exists has arisen from studies looking at short-term changes in air pollution and daily changes in health outcomes such as premature mortality and hospital admissions (National Environment Protection Council 2003, p.4).

There are several areas of uncertainty involved in epidemiological studies including:

- accurate estimation of exposure to a pollutant;
- the extent of potential confounding factors (eg cigarette smoking, health status);
- time considerations in air pollution effects such as lags and latencies between exposure and the effect; and
- individual variation in air pollution exposure (National Environment Protection Council 2003, p.4).

Epidemiological studies commonly use SO<sub>2</sub>, NO<sub>2</sub>, PM<sub>10</sub>, CO and total suspended particulates as indicators of exposure. However, these pollutants are highly correlated and epidemiological studies cannot strictly allocate the observed effects to single pollutants. The repetition of symptoms in Table 1 gives an indication of the difficulty in attributing a particular health outcome to a single pollutant. The WHO advocates a least cost approach where a single pollutant is used as a 'surrogate pollutant', with the aim of capturing the effects of all other pollutants, not just the one being measured (Kunzli, Kaiser et al. 1999). This approach may be overly conservative but a pollutant-by-pollutant approach has the potential to grossly overestimate the impact (Kunzli, Kaiser et al. 2000). The WHO recommends the use of PM<sub>10</sub> as a surrogate indicator for air pollution (Sommer, Seethaler et al. 1999). This approach may be broadened in future to include a precursor to ozone such as NO<sub>2</sub>. There are indications, but currently no conclusive evidence, that ozone is not significantly correlated with particulates (BTRE 2005).

The choice of PM<sub>10</sub> as the representative pollutant is supported by Beers results in Table 2 which show PM<sub>10</sub> to have the greatest associated health costs per tonne of pollutant.

The Bureau of Transport and Regional Economics (BTRE) used an impact pathway approach in 2005 to determine the economic costs of transport emissions in Australia using PM<sub>10</sub> as an indicator of air pollution (BTRE 2005). The health outcomes used in that study are listed in Table 3.

<b>Health outcome</b>	<b>Relative risk estimate associated with a 10µg/m<sup>3</sup> increase in PM<sub>10</sub></b>	<b>95 per cent confidence interval</b>
		<i>Central Lower and Upper</i>
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) <sup>a</sup>	1.094	1.079-1.502
Asthma attacks (children <15 years) <sup>b</sup>	1.044	1.027-1.062
Asthma attacks (adults ≥15 years) <sup>b</sup>	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

**Table 3 - Health outcomes and relative risks of PM<sub>10</sub> exposure (BTRE 2005, p.86)**

These particular outcomes were chosen based on the criteria discussed earlier i.e. a financial cost could be assigned to each outcome and each outcome is independent of the others. The relative risks (RR) figures are derived through epidemiological studies and indicate the relative increase in cases, of each outcome, for a 10µg/m<sup>3</sup> increase in concentration of PM<sub>10</sub>. For example premature mortality has a relative risk of 1.043 which indicates that for every 10µg/m<sup>3</sup> increase in PM<sub>10</sub> mortality will increase by 1.043 to 1 or 4.3%.

RR figures are used to calculate the number of cases that can be attributed to PM<sub>10</sub> exposure for a given population. The figures in Table 3 are based on a WHO review of European and US research (Kunzli, Kaiser et al. 1999).

BTRE used these health outcomes and associated RR in combination with Australian population density data, ambient concentrations of PM<sub>10</sub>, and health care costs data to determine the mortality and morbidity effects of motor vehicle air pollution in regional and urban Australia. A summary of the results is presented in Table 4 which is the combined totals for both regional and urban Australia.

	<b>Mortality</b>	<b>Morbidity</b>	<b>Asthma attacks</b>
Attributable Cases in 2000	1420	2710	1373
Economic Valuation (\$M)	1,846 <sup>a</sup>	817 (including asthma attacks)	

<sup>a</sup>Based on a value of statistical life (VOSL) of A\$1.3M

**Table 4 - BTRE health impacts and economic valuation**

These results allow estimates to be made of the health cost savings that would result from emissions reduction strategies. The BTRE results don't, however, produce a marginal cost per kg of pollutant i.e. the health costs associated with each additional kilogram of pollutant. A marginal cost is necessary to calculate the health costs associated with the operation of different vehicles under different conditions. To calculate this it is necessary to ascertain how each additional kilogram of emissions will effect atmospheric concentrations of that pollutant.

The atmospheric concentration of a pollutant is dependant on the amount of pollutant being added through emissions and the amount being removed by interaction with other chemicals and through wet and dry deposition.

The ExternE project uses a damage estimation method called an impact-pathway approach that determines damages based on the rate of emission of a pollutant e.g. tonnes per year (Bickel and Friedrich 2005) from a particular source. The methodology has been widely recognised as the most developed methodology to account for the externalities of power generation (Pingoud, Malkki et al. 1997) and has been used in many European countries as part of the ExternE project and in other non-ExternE studies (Melichar, Havranek et al. 2004). ExternE is specifically looking at the externalities of electricity generation but the approach has also been modified and applied to transport (Bickel, Schmid et al. 1997).

An implementation of the ExternE impact-pathway approach is the Simplified Uniform World Model (SUWM). The description of the SUWM presented in this paper is derived from two papers by Joseph Spadaro (Spadaro and Rabl 2002) (Spadaro 2002).

The SUWM is similar to the BTRE approach in that it uses concentration response functions (CRFs) to relate emissions to impacts. The difference in the SUMW approach is that instead of measured atmospheric concentrations it uses the rate of emission i.e. tonnes per annum and the rate of removal (depletion velocity) to determine the magnitude of an effect. This is shown in Equation 1.

$$SUWM \text{ _ Damage} = \frac{Emission \text{ _ rate} \times receptor \text{ _ density} \times CRF}{Depeletion \text{ _ velocity}} \times Unit \text{ cost } t$$

**Equation 1**

The term “Receptor density” in Equation 1 is the population density assuming a total surface area bounded by a circle with a radius between 500 and 1000 km.

The SUWM is derived from more complex representations which are described in Spadaro’s papers. The application of the SUWM is based on the following assumptions:

1. constant emission rate,
2. uniform population distribution,
3. uniform atmospheric transport parameters (i.e. constant depletion velocity) and
4. linear concentration response functions with no threshold.

This method can be applied to primary and secondary pollutants and Spadaro treats each pollutant separately. In this case the WHO and BTRE least cost approach will be applied and only the damages for PM<sub>10</sub> are calculated.

The SUWM formula for primary pollutants is:

$$D = D_{uni} = \frac{f_{CR} \rho_{uni}}{k_{uni}} Q$$

**Equation 2 - (Spadaro and Rabl 2002)**

Where:

- $D$  = damage (e.g. in years of life lost (YOLL)/yr) due to emission  $Q$
- $\rho_{uni}$  = uniform receptor density (population density) in people/m<sup>2</sup>
- $f_{CR}$  = slope of the concentration-response (CR) curve in YOLL/(pers.yr.µg/m<sup>3</sup>)
- $k_{uni}$  = uniform depletion velocity for emission  $Q$  in m/s
- $Q$  = emission rate of primary pollutant in µg/sec

The damage figure,  $D$ , represents the annual incidence of a particular health impact. This is then multiplied by the unit cost of that impact to determine the cost per annum. To determined the number of cases per kilogram of pollutant the emissions rate,  $Q$ , is set to 1kg per year (Spadaro 2002, p.11).

According to Australian Bureau of Statistics survey data (City of Perth 2004), Perth's population in June 2003 was 1.4 million people occupying an area of 5,386 km<sup>2</sup>. This equates to a population density in the Perth greater metropolitan area of 259 people per km<sup>2</sup> or 2.59 x 10<sup>-4</sup> people per m<sup>2</sup>.

$f_{CR}$  is a product of the nominal incidence or the baseline in cases per year of the particular disease ( $I_{ref}$ ), and the increased risk ratio (RR) which represents the change in prevalence of the disease due to an increase in pollutant concentration (Bickel and Friedrich 2005, p. 269). The health outcomes and relevant RRs used in this study are those identified in Table 3. The RRs are normalised to represent the increased percentage risk per 1µg/m<sup>3</sup> of pollutant to determine the relevant  $f_{CR}$ .

The depletion velocity,  $k_{un}$ , is a combination of the wet and dry deposition rates of the pollutant and is dependant on local conditions such as rain-fall, average wind speeds and atmospheric stability. Depletion velocities for PM<sub>10</sub> can range from 0.53 (Jinan, China) to 2.86 (North Amazonas, Brazil), the USA adopts a depletion rate of 1.0 for all pollutants (Spadaro 2002, p.23). A depletion velocity specific to conditions in Perth wasn't available so the depletion rate of 0.84 from Yantai, China was used since Yantai is a coastal city at similar latitude to Perth and with a similar average wind speed (4.3m/s for Yantai, 3.5m/s for Perth<sup>1</sup>) and annual rain fall (73cm/yr for Yantai, 79cm/yr for Perth<sup>2</sup>).

Health costs per outcome were taken from the BTRE study (BTRE 2005, p.117). Spadaro suggests that SUMW damage costs for particulates and other primary pollutants need to be scaled by a factor of 10 to account for ground level emissions (Spadaro 2006).

The resulting health costs for Perth are provided in Table 5.

	PM <sub>10</sub> \$/kg	PM <sub>10</sub> \$/tonne
Upper Bound	11.6	11,600
Best Estimate	7.86	7,860
Lower Bound	4.28	4,280

**Table 5 - Perth health costs**

These results are an order of magnitude less than those suggested by Beer in Table 2 but are similar to the value of €15,400/tonne used in Europe (Spadaro and Rabl 2002, Table 2).

Beer's estimate for PM<sub>10</sub> is based on a value of statistical life (VSOL) of \$7.4M whereas the figure used in this study was \$1.3M as suggested by BTRE (BTRE 2005, p.97). If a VSOL of \$7.4M had been used the best estimate would have been \$24,600 per tonne.

### Conclusion

PM<sub>10</sub> is used as a surrogate pollutant in this paper to represent the impact of a highly correlated set of pollutants to remove the chance of double counting particular impacts. This doesn't imply that the goal is only to reduce PM<sub>10</sub>. All emissions need to be addressed in any reduction strategy even though PM<sub>10</sub> appears to be the major contributor to transport emissions related health costs.

There is increasing evidence that O<sub>3</sub> needs to be accounted for separately. Further study needs to undertaken to quantify these damages and to assess the relevance of O<sub>3</sub> and its precursors separately.

Perth's population was considered to be uniformly distributed but this is not the case. Over 98,000 people work in the CBD every day which covers an 8.75 km<sup>2</sup>. This equates to a population density forty times greater than the one used in this study meaning that a kilogram of pollutants emitted in the CBD would carry a much greater health cost than a kilogram emitted in suburban Perth. Quantifying the magnitude of this effect would require

<sup>1</sup> The Australian Bureau of Meteorology ([www.bom.gov.au](http://www.bom.gov.au))

<sup>2</sup> The Australian Bureau of Meteorology ([www.bom.gov.au](http://www.bom.gov.au))

knowledge of several factors including what proportion of time people spend out side during the day, how long pollutants persist in the CBD and how well pollutant concentrations penetrate into office areas.

The costs of each health impact were based on costs incurred in Adelaide. Data was available for Melbourne, Sydney, Brisbane and Adelaide but not for Perth (BTRE 2005, p.117-118). The variance in costs between cities was small so it is not expected that this has had a significant affect on the outcome. However, future research should include Perth specific health costs.

The calculated damage cost is relatively sensitive to  $k_{uni}$ . A change in  $k_{uni}$  causes a proportional change in the total damage cost i.e. a 10% change in the value of  $k_{uni}$  causes a 10% change in the damage cost. Further research is required to determine the depletion velocity of pollutants in Australian cities; it would also be useful to determine the effect of seasonal changes on depletion velocity considering its dependence on rain and wind.

There are potentially very diverse estimates for the costs of damages to health from road transport related air pollution. The values can vary greatly depending on the method used and the assumptions made. There is also uncertainty inherent in the results of epidemiological studies and their applicability to Australian conditions. Assumptions made about the relationship between ambient concentrations and the actual dose received by each person also contributes to uncertainty. This begs the question of whether it is valid to even attempt to quantify these costs. However, it is critical from a policy perspective to consider the complete cost of different options. If that cost is uncertain it can still be contained within known bounds to provide best case, worst case and most likely costs.

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