1 Introduction

In recognition of the health impacts of air pollution, a range of standards have been introduced across Australia over the past two decades. Over this time, air quality reporting has improved greatly. Some degree of air pollution data is currently available for the capital cities and many regional areas. As yet, however, there is no direct linking of air pollution data with health outcomes. There is little active monitoring of the impact of air pollution on the health of asthma sufferers.

To bridge this gap, a way of monitoring the proportion of asthma exacerbations that is due to air pollution is needed. This would provide a clearer picture of the magnitude of the impact of air pollution on asthma, something that is likely to be of increasing importance as the world's climate changes (Capon & Hanna 2009). This information would have wide relevance, including informing asthma management strategies, assessing the impact of changes to environmental management policies and monitoring an important aspect of the health impacts of climate change.

To meet a similar need, agencies such as the United States Environment Protection Authority have developed systems like BenMAP, a software package for estimating the health impact of changes in air pollution levels (United States Environment Protection Authority 2009). Health impact functions used by these systems allow for the quantification of the impact of changes in air pollution levels on the health of a community.

However, the health impact functions of BenMAP and other systems must be modified if they are to be employed to monitor the impact of existing pollutant levels, rather than just assessing the likely impact of changes in pollutant levels.

Key components of a health impact function for monitoring the impact of air pollution on asthma in Australia might be:

- *Health effect estimate*: An estimate of the impact of a pollutant on a particular health outcome. This is usually in the form of a population attributable fraction (PAF). In the context of air pollution, PAFs are based on estimates of the relative risk[1] of exposure to the pollutant at a certain concentration in the atmosphere.
- *Pollutant level*: The concentration, or density, of the pollutant in the atmosphere.
- *Health outcome incidence*: The incidence of the chosen health outcome (in this case asthma hospitalisation).

Using these components, the health impact function for estimating the impact of a particular pollutant on asthma would be:

Health effect = effect estimate x pollutant level x outcome incidence

This report outlines the key challenges to be faced in conducting this type of health impact monitoring, summarises the main Australian research in this area and reviews the feasibility of applying this function to the monitoring of asthma in Australia. This includes identifying health effect estimate candidates from the available literature. The report also applies this methodology to hospitalisation data (the best available data source for estimating the incidence of severe asthma exacerbation).

[1] Relative risk is the risk of exposure to a certain stimulus leading to a defined outcome relative to non-exposure.

Components of air pollution

Air pollution arises from a range of sources, both natural and anthropogenic (of human origin). Natural sources of pollutants typically include bushfires, vegetation and dust storms, while industrial premises and road vehicles are examples of anthropogenic sources.

Pollutants directly emitted into the atmosphere, either from natural or anthropogenic sources, are known as *primary* pollutants. These may undergo chemical changes in the atmosphere forming *secondary* pollutants. For example, nitrogen oxide and volatile organic compounds (primary pollutants) react in the presence of sunlight to form ozone (a secondary pollutant).

The *Australia State of the Environment 2006* (Beeton et al. 2006) report presents the changes and improvements in the ambient air quality in the capital cities in Australia for the years 1991 to 2001 (Department of Environment and Climate Change 2007). In particular, the report compares the levels of each pollutant to the standards established by the National Environment Protection Council in 1998. These standards, established to ensure all Australians are guaranteed safe ambient air quality, can be found at Appendix A of this report.

The main pollutants routinely monitored in Australia are:

- ozone (O_3)
- nitrogen dioxide (NO_2)
- particulate matter of less than either 10 or 2.5 microns ($PM_{10 \text{ or } 2.5}$)
- visibility-reducing particles
- carbon monoxide (CO)
- sulfur dioxide (SO_2).

There are multiple ways of measuring the concentration of each pollutant. The methods differ in their accuracy, cost and ease of use. The choice of method depends on the purpose of investigation and on the data required. In general, measuring gaseous pollutants is easier than airborne particulate matter because the composition of particles is varied and cannot easily be characterised by any single physical or chemical property.

Appendix A provides a brief listing of the major pollutants, their main sources, how their concentrations are generally measured and how their contribution to air pollution in Australia has changed over recent years.

No distinction is made between natural and anthropogenic sources of pollution in this report and no attempt is made to estimate how much of the extant air pollution could be avoided through changes in policy or industry practice. This report has been produced as part of a broader asthma monitoring programme and the main interest is assessing the contribution of air pollution to the burden of asthma in Australia. For the gaseous pollutants the nature of the source is not crucial in this context as it has no impact on the pollutant itself and, therefore, the health impact. While the composition of particulates does vary depending on the source (Burgers & Walsh 2002), the effect of different particles on health is unclear and data for monitoring particulate composition are not readily available.

Health effect estimates

Figure 1.1 provides an illustration of the factors that contribute to asthma exacerbation.

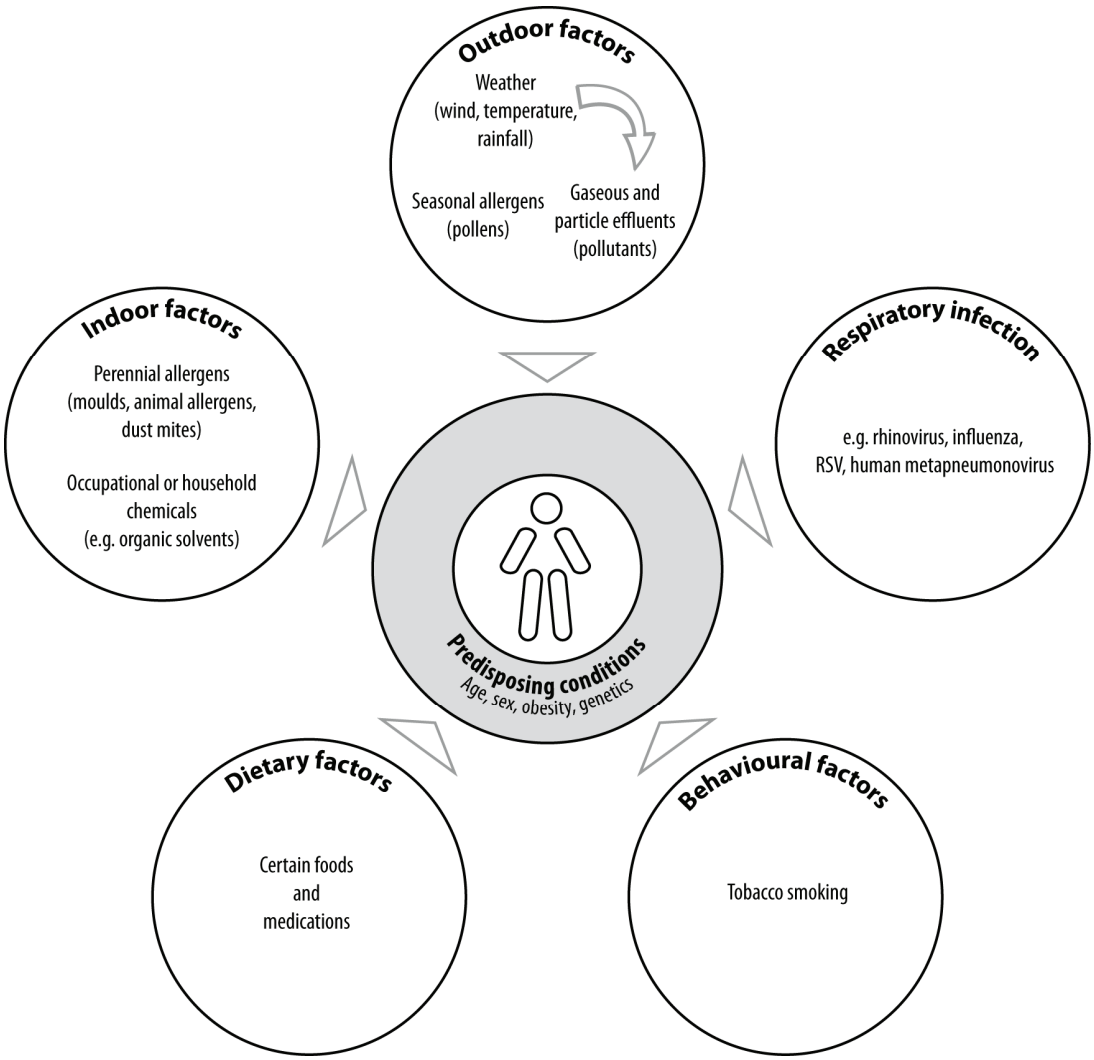

Figure 1.1: Asthma risk factors

Multiple contributors to risk make isolating the contribution of any single factor very difficult. To date, much of the air pollution research in Australia has attempted to quantify the relative risk to the health of a community caused by exposure to certain common pollutants (Anderson et al. 2004; Atkinson et al. 2001; Simpson et al. 2005b; EPA Victoria 2001). Generally the data are presented in the form of a relative risk estimate for exposure to one unit of a pollutant in the atmosphere (what is described as the exposure–response relationship).

Key variables for these studies include location studied, measuring technique for the pollutant, averaging time, ages of people affected and lag time between exposure and health outcome. In some cases, potential confounders such as temperature and season are also accounted for.

An example of relative risk research can be found in the 2001 study by the Environmental Protection Authority (EPA) of Victoria. This study found a relative risk estimate for asthma hospitalisation of 1.0072 per part per billion (ppb) of ozone in the atmosphere (measured as 1hr maximum for the day) during the warm season for children aged 0–14 years (EPA Victoria 2001). This suggests children aged 0–14 years are 1.0072 times more likely to be hospitalised for asthma for every ppb ozone increase in the atmosphere in the warmer months (using the 1hr maximum measure).

Presenting relative risk estimates such as this enhances our understanding of the contribution of outdoor air pollution to asthma outcomes. The crucial next step, which has only been taken in isolated cases in Australia (Burgers & Walsh 2002), is to convert these relative risk (RR) estimates to effect estimates, such as population attributable fractions (PAFs), and apply them to health outcomes data such as hospitalisation and mortality.

PAFs estimate the proportion of a particular outcome (in this case asthma hospitalisations) attributable to exposure to a certain factor (i.e. air pollution). They form the crucial health effect estimate needed for the health impact function described above. A common formula for calculating a PAF is:

$$\% \text{ population exposed} \times ((RR-1)/RR) = PAF$$

According to this formula, the PAF is modified by the proportion of the population exposed. This modification is included because relative risk research often compares a non-exposed control group (e.g. non-smokers) with an exposed group (smokers). The relative risk relates to the risk of a certain outcome arising from exposure to a particular factor versus non-exposure. For example, the risk of contracting lung cancer is greater if you smoke. The relative risk estimate in this case can't be directly translated into a PAF, though, because the PAF is influenced by how many people are exposed. To estimate the proportion of lung cancers within a population due to smoking, it is not enough to know that smokers have an increased risk of lung cancer; we need to know how many people within the population smoke.

In the case of air pollution, there is no identifiable group that could be described as exposed or non-exposed. Measurements of pollution are taken at certain geographical locations but it is not known exactly what proportion of the population is exposed to pollution at that level. Essentially, everyone is assumed to be exposed at whatever level the pollutant is measured. Relative risk in this context refers to the risk of increased hospitalisations arising from a per unit increase in pollution as measured at a particular geographic location. It is not a comparison between an exposed and a non-exposed group. The same group is being compared at different levels of pollution at different times. The effect of this on the PAF formula is to cancel out the influence of the proportion of the population exposed. If we assume that exposure is equal for all members of the population, there is no need to include the proportion of the population exposed. For this reason, the formula used here is:

$$(RR-1)/RR = PAF$$

Based on the above, the per unit PAF can be combined with the actual pollutant level to return an estimate of the proportion of hospitalisations due to that pollutant.

Using the EPA Victorian figures given above, for example, a relative risk figure of 1.0072 per ppb converts into a population attributable fraction of:

$$(1.0072-1)/1.0072 = 0.00715 \text{ or } 0.715\% \text{ per ppb}$$

In this case it could be argued that the proportion of asthma hospitalisations among 0–14 year olds in warm weather due to ozone increases by 0.715% for every ppb the 1hr

maximum of ozone goes up. Using the health impact function above, if the 1hr maximum for ozone reaches 100 parts ppb (the level set as the air quality target in Victoria) in a day, it is estimated that 100 x 0.00715= 71.5%, of asthma hospitalisations for 0–14 year olds, would be related to exposure to ozone on that day (assuming the weather is warm and, perhaps less likely, a linear exposure–response relationship exists between ozone levels and asthma hospitalisation (see Chapter 2 for more detail)).

The relative risk estimates provided by the available research, however, are not consistent. Many of these studies have used differing sampling strategies and data collection periods and have found varying results. The results also vary considerably from location to location as the surrounding atmospheric conditions differ. Another concern is that most of the relative risk research does not take into account threshold issues and the shape of the exposure–response relationship (Erbas & Hyndman 2005). It is unclear, for example, whether an increase from 0 to 1 ppb of a pollutant in the atmosphere has the same impact as an increase from 100 to 101 ppb.

Noting these and other limitations (see Chapter 2 for full discussion), this report identifies some potential health effect estimate candidates for use in future asthma monitoring and attempts to apply them to a single case study—Melbourne in 2006.

Air pollution and incidence data

The other components of the health impact function are input data that relate to a set geographical region. Air pollution data for the relevant pollutants must be matched with the source research for the health effect estimates in terms of pollutant measuring technique, averaging time, lag time and geographical region. Air pollution data should also be matched with the health outcome data for the same geographical region for the same period. And the health outcome data must match the health effect estimate research in relation to the age range.

Melbourne has been selected as the region for this report because of the relative abundance of air pollution research that has been conducted there compared with other areas (see Chapter 3). This research provides a greater depth of health effect estimates than is available for other regions, making the data matching task easier. The year 2006 was selected as a relatively recent year for which stable hospitalisation data is available.

The air pollution data for Melbourne was sourced from the Environment Protection Authority of Victoria.

The primary source of hospitalisation data is the National Hospital Morbidity Database (NHMD). The NHMD is compiled from data supplied by the state and territory health authorities. It is a collection of electronic, confidentialised summary records for separations (episodes of care) in public and private hospitals in Australia.

It is important to note here that hospitalisation is an extreme outcome for asthma. As a result, this data only includes the most visible of health outcomes. As to the effects of air pollution, hospitalisation and death likely represent the tip of a very large iceberg, as far as asthma exacerbation is concerned (Figure 1.2). For every asthma hospitalisation due to air pollution it is likely that many more people suffer asthma exacerbations of less severity, but there is little data available on this.

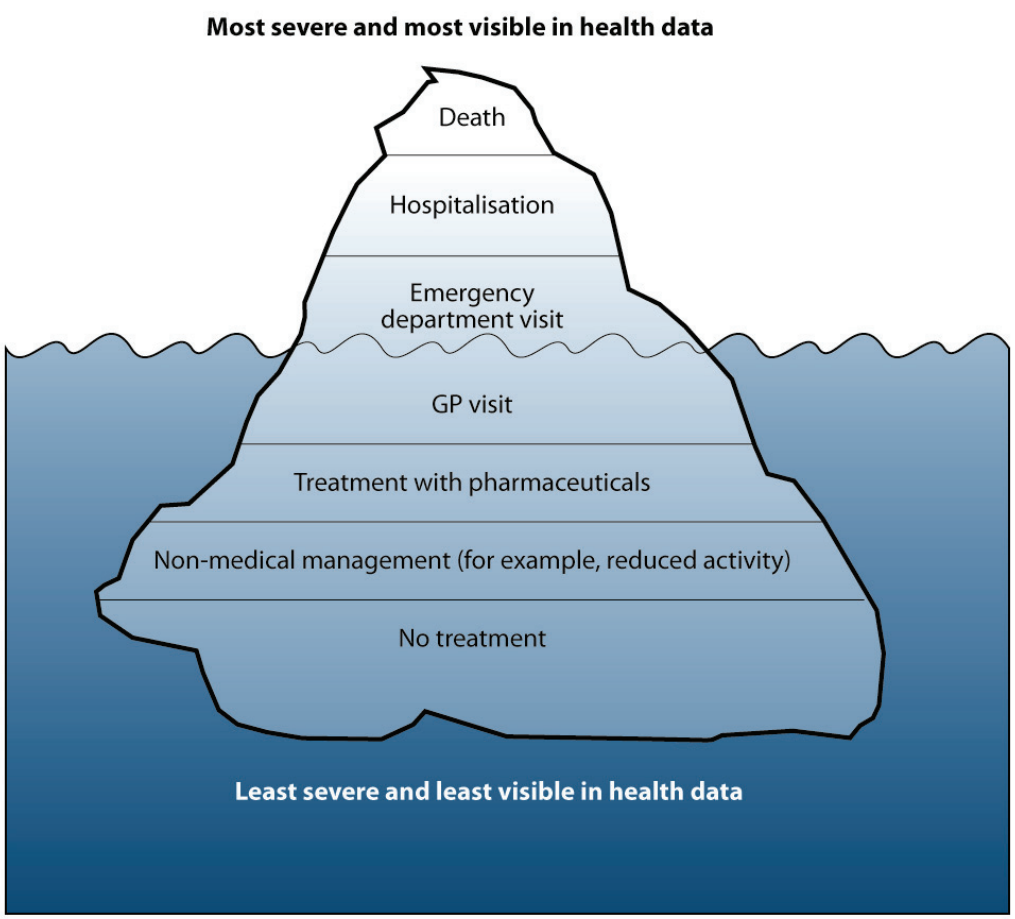

Figure 1.2: Asthma management iceberg

2 Key challenges

Linking air pollution to health outcomes is fraught with complexities. There are weaknesses in available data sources and confounders that make drawing conclusions difficult. Below is a discussion of some of the main challenges affecting air pollution monitoring in relation to asthma in Australia.

Regional variation

Monitoring stations only provide an indication of the level of pollution that people in the region are exposed to. Many monitoring stations are located away from densely populated zones and it is likely that the pollution levels recorded differ from the actual levels experienced by the population. There is likely to be significant variation between sites in the amount of time that people generally spend outside being exposed to air pollution; and, due to differences in planning history and population density, the effects of industrial pollution may be greater in some cities compared to others.

The relative altitude above sea level and the impact of local weather events can also affect the monitoring of air pollution (Cogo et al. 1997; Simpson et al. 2005b).

Other factors that lead to regional variations are population mix, possible variations in health practices and the composition of the particulate mix. There is also a genetic element to asthma (ACAM 2008) that contributes to variations in the prevalence of asthma between population groups. This means that the baseline prevalence and the exposure–response relationship are likely to be affected by the population.

Furthermore, health jurisdictions may have different approaches and policies for handling asthma exacerbations. Differences in policies regarding when an asthma sufferer should be admitted to hospital, for example, might confound results based on data relating to hospital admissions.

The composition of the particulate mix varies in time and location based on the source. The particulates produced by bushfires, for example, can be different in size and nature to those arising from industrial sources.

Time and lag effects

In many cases the relative risk research includes an estimate of the effect that occurs over the following one to five days after exposure to a pollutant (known as lag effect). There is an underlying assumption that daily episodes of pollution are closely linked in a temporal sense with asthma exacerbation. These studies identify asthma exacerbations that are linked to a given day's exposure to pollution. This approach does not take into account the effect of prolonged exposure to pollution such as the effect of exposure to a pollutant over a week, month or longer.

An acceptable methodology has not yet been identified to account for lag effects in the health impact function. This makes it necessary to apply the health effect formula on a day-by-day basis to the incidence data—creating daily estimates of the effect of a pollutant on asthma hospitalisations.

Measurement methods

Several different measurement approaches and averaging times have been used for each pollutant (see Chapter 3), both in the relative risk research as well as air pollution monitoring generally. Some approaches use 24 hour (daily) averages, whereas others use the daily 1 hour maximum approach or a number of other approaches. Technologies for measurement are also diverse, especially for particulates. To apply relative risk research to a health impact function, the measurement used to develop the effect estimate needs to be matched with measurement used to derive the air pollution data. Only air pollution data that was measured in the same way as in the effect estimate study can be used in the health impact function.

Age ranges

Many studies found positive relative risk estimates for pollutants only for certain age groups (Chapter 3). For example, the 0–14 years group tended to return more significant results than the 'all ages' group. This suggests that age has an effect on the interaction between pollutant level and outcome. Therefore, when applying effect estimates to hospitalisation data, the same age groups should be used.

Baseline incidence

Much of the Australian relative risk research used data from the 1990s (Chapter 3). The incidence of hospitalisation and mortality due to asthma has shown a general decline since that time (ACAM 2008). This means that the baseline incidence is now lower than during the source period for much of the air pollution research. The health effect of air pollution might be more pronounced in populations with lower baseline incidence, as it accounts for proportionately more of the total incidence. Alternatively, the impact of air pollution might be more pronounced in populations with higher baseline incidence. In this context, it is possible that the pollutants interact with other irritants in the environment to create relatively more exacerbations.

A further consideration is that baseline incidence may vary temporally (with season and time of day) and spatially (from city to city, and from suburb to suburb within a city), although typically incidence data is only available at higher levels of aggregation such as city-wide annual rates.

Data gaps

The air pollution research in Australia has tended to focus on hospitalisation and mortality (Naureckas et al. 2005). The most common reason people are admitted to hospital or die due to asthma is exacerbation of the condition (ACAM 2008). Outcomes such as hospitalisation, and especially death, represent serious events that occur only in the most severe asthma exacerbations. Due to a lack of data, it is not possible to explore the role of air pollution in less severe cases (Naureckas et al. 2005; ACAM 2008).

Alternate indicators of the rate of asthma exacerbation in the community include medication use and GP encounters. Asthma medications and their level of use vary widely between

patients and are also used for other conditions (such as Chronic Obstructive Pulmonary Disease), rendering trend data difficult to interpret. Similarly, examining GP visits data from the national data sets would have limited use, as there is no way to discern whether a GP visit due to asthma was due to an exacerbation or was simply a routine visit to renew a prescription.

Exposure–response relationship

The exposure–response relationship is the relationship between the pollutant level and the effect of exposure to the pollutant in terms of health outcome.

Many pollutants appear to have a non-linear exposure–response relationship (Vallero 2008; Daniels et al. 2000; Smith et al. 2000). That is, the effect estimate does not rise in linear proportion with the pollution level. While there may be no level at which exposure to air pollution is entirely harmless, there is some evidence to suggest that the effect of exposure to pollution increases more rapidly as the level of the pollutant increases in the atmosphere (Streeton 1997; US Environmental Protection Agency 1996; Bascom et al. 1996; Migliaretti et al. 2005; Dockery & Pope 1994). The implication of this for the health impact function is that the effect estimates, or PAFs, should not be applied equally at all pollution levels.

A linear relationship (model B in Figure 2.1) means that the effect increases proportionately with the pollutant level. Where there is an exposure–response curve, the effect increases either more or less rapidly than a linear relationship would predict. A pronounced rising curve (model C) indicates that the effect of the pollutant increases rapidly at higher pollutant levels. A curve that tapers off suggests that the effect of the pollution lessens at higher concentration levels (model A). And a curve that encompasses flat sections as well as steep inclines (model D) indicates an exposure–response relationship that changes considerably at different pollutant levels.

Even in situations of mild non-linearity in the exposure–response relationship, incorrectly concluding that a linear relationship exists can result in a large under- or over-estimation of the effects (Roberts & Martin 2006).

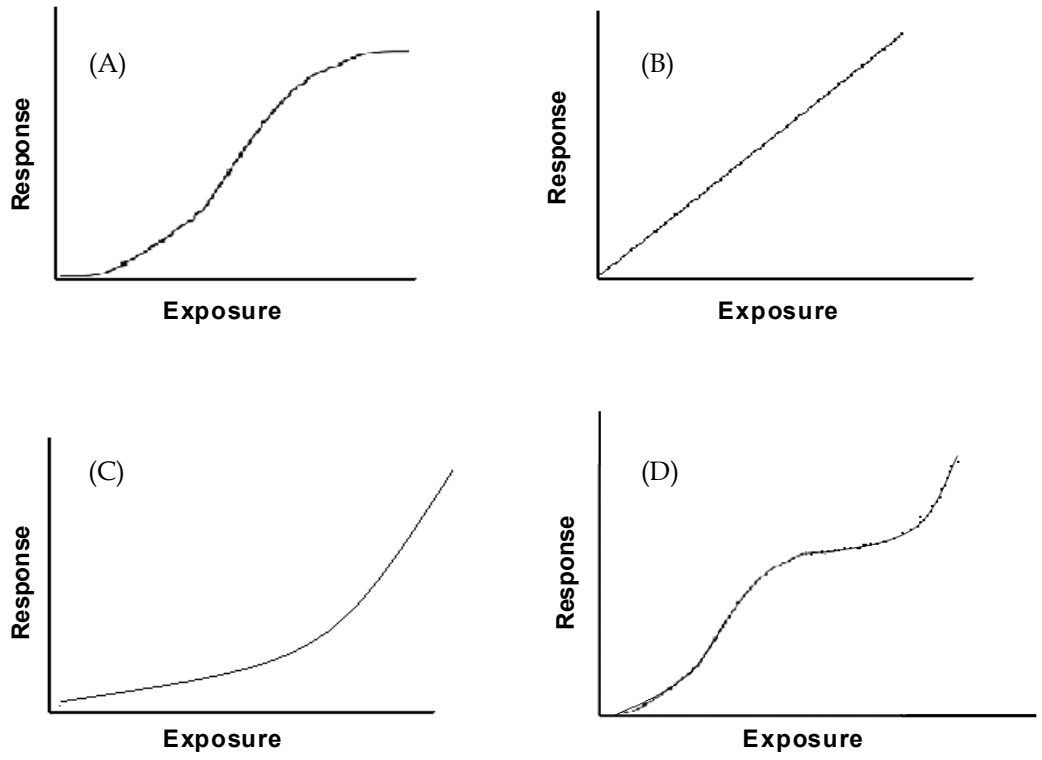

Figure 2.1. Possible exposure–response curves for the relationship between air pollution and asthma exacerbation

Co-linearity

Many pollutants share the same sources and their levels in the atmosphere often rise and fall in unison. For example, vehicle emissions cause a rise in NO_2 concentrations as well as a simultaneous rise in volatile organic compounds (VOCs). In this situation it can be difficult to determine what proportion of a particular health outcome can be attributed to an individual pollutant. In addition, pollutants can interact with each other at a chemical level to produce asthma exacerbations (e.g. NO_2 and VOCs combining to create O_3, which aggravates asthma).

Studies of the short-term effects of air pollution on health outcomes commonly adopt the Air Pollution and Health: A European Approach (APHEA) protocol to account for these issues. With the APHEA protocol, a model is usually built with an initial spectral analysis followed by regression analyses (generally based on generalised linear modelling or generalised additive modelling) that includes covariates and potential confounders, such as meteorological variables. Typically, Poisson regression is then used to produce estimates of associations between air pollution concentrations and health effect outcomes data.

Different studies on the same pollutant often report varying risk ratios because of factors such as errors in exposure assessment, errors in reporting the health outcome or the underlying indeterminacy of some of the associations of interest (Briggs et al. 2003).

The risk ratios adopted here are based on Australian research where an attempt has been made to account for co-linearity as well as controlling for other confounding factors such as weather. For the purposes of this report, we assume that the relative risk estimates relate to the independent effect of each pollutant. This potentially enables the health effect estimates for each pollutant to be added to provide the total effect of air pollution on asthma. In practice, however, insufficient research is available to determine if this approach would yield accurate results.

3 Previous research

Australian studies into the relative risk of exposure to air pollution have tended to focus on mortality and hospitalisation. Below is a brief summary of some of the main studies.

The reader will note each study measures pollutants differently and uses varying units of expression. For reference the main units of measurement referred to below are:

- ppb = parts per billion
- ppm = parts per million
- pphm = parts per hundred million
- $PM_{10 \text{ or } 2.5}$ = a measure of particulate density that involves weighing a filter, passing air through it and then weighing it again, to estimate the volume of particles over a certain size (either 10 or 2.5 microns) that were captured
- bscat = a measure of particulate density that that involves shining a light through a volume of air and measuring the total light scattering that occurs
- bsp = a measure of particulate density that is similar to bscat but that only measures the scattering of light caused by particles in the air (bscat includes light scattered by things such as gases and water).

The averaging period in the tables below refers to the period over which the air pollution readings are averaged. In the case of an averaging period of 1 hour, for example, data are grouped by the hour of the day in which they were recorded—for example, from 0100 to 0200 or from 1600 to 1700. The average reading is then calculated for each hour of the day. Where the table indicates 1 hr as the averaging period, the highest 1 hr reading was taken for each day in the study period. Where the averaging period was 24 hrs, the average of the readings over the entire day was taken.

Mortality

In Australia, the effect of air pollution on respiratory mortality has been studied in Melbourne by the EPA Victoria (2000) and in Sydney, Brisbane, Melbourne and Perth by Simpson et al. (2005b).

These studies have used statistical techniques such as generalised additive modelling to determine the relative risk of death caused by a respiratory condition occurring the same day or up to three days following a per unit increase of air pollutant.

The relative risk was calculated based on different time periods for death to occur following the air pollution event and different periods of time for averaging the measurement of air pollution.

These studies did not specifically identify asthma as an individual respiratory disease in the results so they are not considered in detail here. It is worth noting, however, that both studies found that air pollution had significant effects on respiratory mortality.

Table 3.1: Air pollutants, units of measurement and averaging periods, Melbourne (EPA Vic)

Pollutant	Unit of measurement	Averaging periods
Particles	bscat x 10^{-4}/m	1 hr, 24 hr
O_3	ppb	1 hr, 4 hr, 8 hr
NO_2	ppb	1 hr, 24 hr
CO	ppm	1 hr, 8 hr

Source: EPA Victoria (2000).

Table 3.2: Air pollutants, units of measurement and averaging periods, Sydney, Melbourne, Brisbane and Perth

Pollutant	Unit of measurement	Averaging periods
Particles	bscat x 10^{-4}/m	24 hr
O_3	ppb	1 hr, 4 hr
NO_2	ppb	1 hr

Source: Simpson et al. (2005b).

Hospitalisation

Six major studies have examined the effects of ambient air pollution on asthma-related hospitalisation in Australia.

Sydney

Morgan et al. (1998) studied the effects of ambient air pollution on daily hospital admissions for asthma in Sydney from January 1990 to December 1994. The study involved two age groups (1–14 years and 15–64 years), 3 pollutants (particles (bscat 10^{-4}/m), O_3 (ppb), and NO_2 (ppb)), two measuring periods (1 hour maximum and 24 hour average) and two time lags (same day and one day) (Table 3.4). The study adopted the APHEA approach using Poisson regression and generalised estimating equations (GEEs).

To control for the potential confounding effects of weather, temperature data were obtained from the Bureau of Meteorology. From these data daily mean temperature and dew point temperature were included in the model. Seasonal effects were also controlled.

Morgan et al. (1998) found NO_2 had a significant effect on asthma hospitalisations for 1 to 14 year olds (Table 3.3).

Table 3.3: Statistically significant relative risk estimates for NO_2 from Morgan (1998) and 95% upper and lower confidence intervals

				Relative risk			PAF		
Principal author	Age(yrs)	Period(hr)	Lag(d)	Mean	LCL	UCL	Mean	LCL	UCL
Morgan (1998)	1–14	1 max	0	1.053	1.011	1.097	5.024%	1.059%	8.826%

Table 3.4: Air pollutants, units of measurement and averaging periods for relative risk research

Pollutant	Unit of measurement	Averaging periods
Morgan et al. (1998)		
Particles	bscat x 10^{-4}/m	1 hr, 24 hr
O_3	ppb	1 hr
NO_2	ppb	1 hr, 24 hr
EPA Victoria (2001)		
O_3	ppb	1 hr, 4 hr, 8 hr
Particles	bsp x $10^{-4}/m^{-1}$	1 hr, 24 hr
NO_2	ppb	1 hr, 24 hr
CO	ppm	1 hr, 8 hr
Erbas & Hyndman (2005)		
NO_2	pphm	1 hr
SO_2	pphm	1 hr
O_3	pphm	1 hr
Particles	bsp x 10^{-4}/m	1 hr
Petroeschevsky et al. (2001)		
Particles	bsp x 10^{-5}/m	1 hr, 24 hr
O_3	pphm	1 hr, 8 hr
SO_2	pphm	1 hr, 24 hr
NO_2	pphm	1 hr, 24 hr
Department of Environment GoWA (2003)		
O_3	ppb	1 hr, 4 hr, 8 hr
NO_2	ppb	1 hr, 24 hr
CO	ppm	8 hr
Particles - bsp	10^{-4}/m	1 hr, 24 hr
Particles - $PM_{2.5}$	µg/m^3	24 hr
Simpson et al. (2005a)		
O_3	ppb	1 hr
NO_2	ppb	1 hr
Particles	bsp x 10^{-4}/m	24 hr
CO	ppm	8 hr

Melbourne

Melbourne: Study 1

Denison and colleagues (EPA Victoria 2001) studied the relationship between ambient air pollution and daily emergency admissions for asthma in Melbourne from July 1994 to December 1997. The study involved two age groups (all ages and 0–14 years), four pollutants (particles, O_3, NO_2 and CO), four measuring periods (Table 3.4) and five time lags (same day and one day, two days, three day average and five day average). The study adopted the APHEA approach using generalised additive models (GAM) and Poisson regression.

Table 3.5: Statistically significant relative risk estimates from EPA Victoria (2001) and 95% upper and lower confidence intervals

				Relative risk			PAF %		
Pollutant	Age(yrs)	Period(hr)	Lag(d)	Mean	LCL	UCL	Mean	LCL	UCL
CO	0–14	8	3 ave	1.061	1.027	1.095	5.714	2.667	8.659
CO	0–14	1	3 ave	1.031	1.010	1.052	3.007	0.990	4.979
CO	All	8	5 ave	1.064	1.036	1.092	6.006	3.503	8.442
CO	All	1	5 ave	1.040	1.022	1.058	3.828	2.172	5.455
NO_2	0–14	24	5 ave	1.012	1.006	1.018	1.166	0.577	1.739
NO_2	0–14	1	5 ave	1.005	1.001	1.008	0.478	0.130	0.833
NO_2	All	24	5 ave	1.015	1.010	1.019	1.429	0.980	1.874
NO_2	All	1	5 ave	1.006	1.003	1.009	0.587	0.319	0.862
Particulates	0–14	24	0	1.148	1.063	1.240	12.900	5.909	19.374
Particulates	0–14	1	0	1.059	1.020	1.100	5.589	1.932	9.107
Particulates	All	24	5 ave	1.139	1.058	1.227	12.235	5.500	18.487
Particulates	All	1	5 ave	1.077	1.037	1.117	7.115	3.605	10.491

To control for the potential confounding effects of weather, data on minimum, maximum and average temperature, minimum and maximum dew point temperature, and total daily rainfall were added to the model. Seasonal effects were also controlled by collating results for the whole year, the 'cool' season, and the 'warm' season.

This study found significant effects for CO, NO_2 and particulates for both the 0–14 years and all ages groups (Table 3.5).

Melbourne: Study 2

Erbas and Hyndman (2005) studied the effects of ambient air pollution on hospital admissions for asthma in Melbourne from 1989 to 1992. The study involved all ages, four pollutants (particles, O_3, SO_2 and NO_2 (all measured as parts per hundred million, pphm)), one measuring period (1 hour maximum) and two time lags (same day and one day) (Table 3.4). The study included a series of models using generalised linear models (GLM), GAM, parameter driven Poisson regression models (PDM) and, transitional regression models (TRM).

To control for the potential confounding effects of weather, data were obtained on daily levels of dry bulbs temperature and dew point temperature, and relative humidity. Seasonal effects were also controlled.

NO_2 was found to increase asthma hospitalisations using GLM, GAM, PDM and TRM for zero lag days (Table 3.6). In contrast, where a lag of 1 day was introduced, a significant negative effect was found using GAM. O_3 was also found to negatively affect asthma hospitalisations where a lag of 1 day was introduced into the analysis.

Table 3.6: Statistically significant relative risk estimates from Erbas and Hyndman (2005) and 95% upper and lower confidence intervals

				Relative risk			PAF %		
Pollutant	Age(yrs)	Period(hr)	Lag(d)	Mean	LCL	UCL	Mean	LCL	UCL
NO_2 (GLM)	All	1 max	0	1.050	1.010	1.080	4.762	0.990	7.407
NO_2 (GAM)	All	1 max	0	1.050	1.010	1.090	4.762	0.990	8.257
NO_2 (PDM)	All	1 max	0	1.040	1.010	1.080	3.846	0.990	7.407
NO_2 (TRM)	All	1 max	0	1.050	1.020	1.080	4.762	1.961	7.407
NO_2 (GAM)	All	1 max	1	0.960	0.920	0.990	–4.167	–8.696	–1.010
O_3 (GLM)	All	1 max	1	0.960	0.930	0.990	–4.167	–7.527	–1.010
O_3 (TRM)	All	1 max	1	0.970	0.950	0.990	–3.093	–5.263	–1.010

Brisbane

Petroeschecsky et al. (2001) studied the effects of ambient air pollution on hospital admissions for asthma in Brisbane from 1987 to 1994. The study involved three age groups (all ages, 0–14 years and 15–64 years), four pollutants (particles (bsp), O_3, SO_2 and NO_2), 3 measuring periods and five time lags (same day, 1 day, 2 days, 3 day average and 5 day average) (Table 3.4). The study adopted the APHEA approach using a series of models using generalised estimating equations (GEEs) and Poisson regression. Emergency admissions were the outcomes of interest; other modes of admission, such as transfers from other hospitals, scheduled admissions or admissions arranged through a general practitioner, were excluded.

Table 3.7: Statistically significant relative risk estimates from Petroeschevsky (2001) and 95% upper and lower confidence intervals.

				Relative risk			PAF %		
Pollutant	Age(yrs)	Period(hr)	Lag(d)	Mean	LCL	UCL	Mean	LCL	UCL
NO_2	15–64	1 max	0	0.941	0.900	0.984	–6.270	–11.111	–1.626
NO_2	All	1 max	3 ave	0.962	0.936	0.989	–3.950	–6.838	–1.112
O_3	0–14	8 ave	1	1.064	1.015	1.115	6.015	1.478	10.314
O_3	15–64	8 ave	2	1.084	1.037	1.133	7.749	3.568	11.739
O_3	All	8 ave	5 ave	1.090	1.042	1.141	8.257	4.031	12.358
Particulates	0–14	1 max	2	0.995	0.990	0.999	–0.503	–1.010	–0.100

To control for the potential confounding effects of weather, data were obtained from the Bureau of Meteorology on daily minimum and maximum temperature, rainfall and relative humidity. Seasonal effects were also controlled.

NO_2 and particulates were found to negatively affect asthma hospitalisations (Table 3.7). O_3 was found to have a positive effect on asthma hospitalisations for the 0–14 years, 15–64 years and all ages groups with lags of 1 day, 2 days and 5 days respectively.

Perth

Codde and colleagues (Department of Environment GoWA 2003) studied the relationship between changes in ambient air pollution and daily hospital admissions for asthma in Perth from January 1992 to December 1997. The study involved three age groups (all ages, 0–14 years and 65 years and over), five pollutants (particles (bsp and modelled $PM_{2.5}$), O_3, NO_2 and CO), four measuring periods (Table 3.4) and seven time lags (same day and 1 day, 2 days, 3 days, 2 day average, 3 day average and 4 day average). The study adopted the APHEA approach followed by a case-crossover design with logistic regression.

To control for the potential confounding effects of weather, data on daily temperature, dew point temperature, relative humidity, and wind speed and direction were added to the model. Seasonal effects were also controlled.

O_3 and particulates were found to have a positive effect on asthma hospitalisations (Table 3.8). O_3 was found to be significant for the 0–14 years group with zero lag, whereas, particulates were found to be significant for the 0–14 years and all ages groups where a lag of 2 days was included in the analysis.

Table 3.8: Statistically significant relative risk estimates from Department of Environment GoWA (2003) and 95% upper and lower confidence intervals

				Relative risk			PAF %		
Pollutant	**Age(yrs)**	**Period(hr)**	**Lag(d)**	**Mean**	**LCL**	**UCL**	**Mean**	**LCL**	**UCL**
O_3	0–14	1 max	0	1.003	1.000	1.006	0.309	0.030	0.577
Particulates	0–14	24 ave	2	1.003	1.001	1.006	0.339	0.080	0.596
Particulates	All	24 ave	2	1.002	1.000	1.004	0.239	0.040	0.438

Multiple cities

Simpson et al. (2005a) conducted a meta-analysis of the short-term effects of air pollution on hospital admissions in four cities: Brisbane, Melbourne, Perth and Sydney from 1996 to 1999. The study involved one age group (15–64 years), three pollutants (particles (bsp), O_3 (pphm), and NO_2 (pphm)), two measuring periods (1 hour maximum, and 24 hour average) and two time lags (2 days and 3 days) (Table 3.4).

The study used the APHEA 2 modelling approach to derive estimates and three different models to test the reliability of the findings: GLM, GAM and penalised spline models.

Only particulates were found to have a significant effect on asthma hospitalisations (Table 3.9). This effect was found for lags of 2 and 3 days.

Table 3.9: Statistically significant relative risk estimates from Simpson et al. (2005a) and 95% upper and lower confidence intervals

				Relative risk			PAF %		
Pollutant	**Age(yrs)**	**Period(hr)**	**Lag(d)**	**Mean**	**LCL**	**UCL**	**Mean**	**LCL**	**UCL**
Particulates	15–64	24 ave	2	1.064	1.001	1.132	6.024	0.060	11.622
Particulates	15–64	24 ave	3	1.089	1.024	1.159	8.198	2.344	13.696

4 Proposed methodology

The aim of this paper is to identify the best approach possible for estimating the impact of air pollution on asthma hospitalisations in a given year, given the currently available input research and data. The proposed methodology takes into account the challenges identified in Chapter 2 as far as possible. At the outset, however, it is acknowledged that the methodology will be imperfect and require further elaboration through targeted study.

In this context, it is proposed that a suitable health impact function to estimate the impact of a single pollutant would look like this:

Pollutant health effect (PHE) = effect estimate x pollutant level x outcome incidence

Calculating the total effect of air pollution using this base function might be a simple matter of adding the individual estimates as below:

Total health effect (all pollutants) = PHE1 + PHE2+…+PHEx

In terms of pollution levels, there are air pollution data available from most capital cities. Similarly, there are well established national data collections for both hospitalisations and deaths that enable detailed analysis. In this study, the daily pollution level was provided by the Environment Protection Authority of Victoria and the national morbidity data set was used to extract the hospitalisation data for the relevant region.

The selection of appropriate effect estimates is problematic. The effect estimates required here are often gained from research conducted into the relative risk of exposure to particular pollutants (United States Environment Protection Authority 2009). While there has been a body of such work conducted in Australia, the methodologies and results are inconsistent and difficult to interpret.

Due to the challenges described in more detail in Chapter 2, effect estimates must meet certain criteria before they can be used for the purposes identified here, including:

- The source research for the effect estimate is best drawn from the same geographical region (in this case Melbourne) as the air pollution and health incidence data.
- Results must account for the effect of a pollutant on the same day as exposure (zero lag), as the health impact function outlined above is not able to adequately account for lag times.
- The measurement technique and averaging time must match the available air pollution data.
- The age range must match the hospitalisation data.
- Adequate statistical controls must have been used in the studies to ensure the effect estimates are independent of any cumulative or interactive effects of other pollutants. This limits the impact of co-linearity and interactive effects and enables the addition of estimated health effects for individual pollutants as proposed above.

Very few studies currently meet all of these criteria, making the selection of suitable effect estimates limited. Furthermore, the fact that the exposure–response relationship might change at different levels of pollution needs to be accounted for (see *Exposure–response relationship* section in Chapter 2 for more detail).

Effect estimate selection

The Australian city that has returned findings that best meet these criteria is Melbourne. Erbas & Hyndman (2005) and EPA Victoria (2001) both conducted major studies into hospitalisations related to a variety of pollutants using data from the 1980s and 1990s.

In terms of hospitalisations, EPA Victoria (2001) found significant relative risk estimates for particulates (as measured by the scattering of light from the particles (bsp)) for zero lag for the 0–14 years age group, using both 24 hour average and daily maximum 1 hour readings (Table 4.1).

Table 4.1: Relative risk estimates for particulates in Melbourne

Age (yrs)	Period (hr)	Relative risk			PAF %		
		Mean	Lower	Upper	Mean	Lower	Upper
0–14	24	1.148	1.063	1.240	12.900	5.909	19.374
0–14	1	1.059	1.020	1.100	5.589	1.932	9.107

Source: EPA Victoria (2001)

Erbas & Hyndman (2005) found significant relative risk estimates for NO_2 for all age groups (zero lag) using 1 hour maximum readings (Table 4.2).

Table 4.2: Relative risk estimates for NO_2 in Melbourne

Age(yrs)	Period(hr)	Relative risk			PAF %		
		Mean	Lower	Upper	Mean	Lower	Upper
All	1 max	1.050	1.010	1.080	4.762	0.990	7.407
All	1 max	1.050	1.010	1.090	4.762	0.990	8.257
All	1 max	1.040	1.010	1.080	3.846	0.990	7.407
All	1 max	1.050	1.020	1.080	4.762	1.961	7.407

Source: Erbas and Hyndman (2005).

Findings for the other pollutants for Melbourne related to exposure responses that occurred 1 or more days after exposure. These could not be included here as no satisfactory way has been identified to account for lag effects in the health impact function.

Based on the above, the unmodified effect estimates selected for use in the test case presented here were:

- 12.9% per unit ($1x10^{-4}m^{-1}$) for 24 hour average bsp readings for $PM_{2.5}$ and 5.589% for 1 hour maximum readings. These were only applied to data relating to the 0–14 age group
- 4.762% per part per hundred million (pphm) for 1 hour maximum readings of NO_2. This was applied to all ages.

Data modifications to account for the unknown exposure–response relationship

The effect estimates used in the health impact function are essentially based on average responses over a period of time. If the exposure–response relationship is linear it does not matter if the overall pollution levels differ from the period in which the study was conducted. The per unit effect estimate is equally applicable at any pollutant level.

The background pollution levels during the period of study from which the effect estimate was derived are important where the exposure–response relationship is non-linear. Where a non-linear exposure–response relationship exists, the per unit effect estimate needs to be modified to account for the exposure–response relationship. Take the case where the effect of the pollutant starts at a very low level but increases quicker than the rate of increase of the pollutant. Changes in the pollution level at much higher levels than the period during which the effect estimate was developed will result in underestimation of the effect of the pollutant.

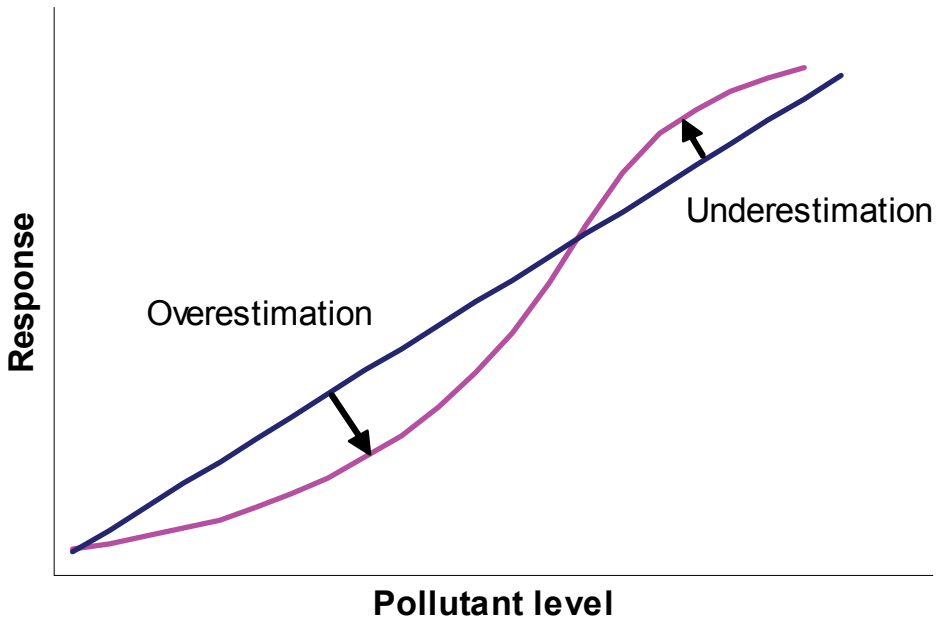

Figure 4.1. Impact of variation from effect estimate source pollution levels

In contrast, where changes in the pollution levels are at much lower levels than the period during which the effect estimate was developed, the results are likely to overestimate the effect of the pollutant.

Figure 4.1 illustrates this theoretical effect by plotting a linear exposure–response relationship against a curved relationship. For pollution levels where the two lines meet there will be no difference between the two effect estimates. Where the pollutant levels are lower, an assumed linear relationship will yield higher effect estimates than the curved relationship. And where they are higher, underestimation occurs.

There is some research to suggest that the relationship is linear for particulates (Schwartz 2004). Unfortunately, however, no research has been identified that conclusively quantifies the shape of the exposure–response curve for either NO_2 or particulates in Australia (Erbas & Hyndman 2005).

In the interests of ensuring the results presented here do not overstate the situation, two analyses are provided. The first presents the results using unmodified data. That is, the air pollution level data is incorporated into the health impact function without modification. The results, therefore, are subject to the exposure–response effect.

In the second analysis, a threshold has been introduced into the pollution data. Pollution levels below a set amount have been excluded from the analysis. More specifically, a modified pollution level has been calculated using the following formula:

Modified pollution level = actual pollution level – threshold level

This means that only the proportion of the pollution concentration above the threshold has been used in the calculations and zero pollution has been recorded for days where the pollution level was equal to or below the threshold.

The threshold was calculated based on the pollutant levels during the period in which the effect estimate was derived. It was set at one standard deviation below the mean. That is, the threshold was calculated by subtracting the standard deviation from the average level of the pollutant during the period in which the effect estimate was derived. Pollutant levels in 2006 that fell below this level were excluded from the analysis.

For example, the study providing the NO_2 effect estimate was conducted using data from July 1989 to December 1992. To calculate the minimum threshold the average level of NO_2 between July 1989 and December 1992 (2.301 pphm) was calculated, as was the standard deviation (0.968 pphm). One standard deviation was then subtracted from the average to derive the minimum threshold (1.333 pphm).

Days in 2006 where the NO_2 level was equal to or below this threshold were excluded from the analysis. For days where the NO_2 exceeded this level, only the proportion above the threshold was used in the modified calculations.

Take, for example, a day where the measure for NO_2 was 1.200 pphm. This is below the threshold of 1.333 pphm, so the pollution level would be treated as zero and zero hospitalisations would be attributed to NO_2 for that day. If NO_2 reached 1.750 pphm, however, then the modified pollution would be calculated as 1.750 – 1.333 = 0.417 pphm for that day and the hospitalisations calculated accordingly.

This approach has been taken on the assumption that the exposure–response curve is not steep enough to create significant overestimation within the range of one standard deviation below the average. As Figure 4.1 illustrates, if the exposure–response curve is non-linear, using pollution levels below the threshold level in the health impact formula becomes increasingly more likely to lead to overestimation as the pollutant levels get lower. Pollution levels higher than the average are allowed under the assumption that the effect on the results is one of under- rather than over-estimation.

Notes on the hospital data analysis

The hospitalisation data for Melbourne for the 2006 calendar year were derived from the National Hospital Morbidity Database using the Australian Standard Geographical

Classification system (Australian Bureau of Statistics 2005) state code 2 and statistical division code 05. The state code defined the state where the hospitalisation occurred, while the statistical division code defined the region of the patient's usual residence.

An asthma hospitalisation was defined as an admission where the principal diagnosis was identified as codes J45 and J46 according to the International Classification of Diseases and Related Health Problems, 10th revision. The principal diagnosis is the diagnosis established to be chiefly responsible for the patient's episode of care in hospital.

Only patients who underwent acute care were included.

The date of admission was used to identify the date when an asthma hospitalisation had occurred.

The hospitalisation data did not include patients who were treated in the emergency department.

5 Results

Table 5.1 presents summary descriptive data for the analysis conducted in relation to the pollutants studied. Overall, the levels of NO_2 in Melbourne were on average around 20% lower in 2006 than during the period within which the effect estimate was derived (July 1989 – December 1992). This suggests that the unmodified estimates for NO_2 are likely to overestimate the impact to some degree (see exposure–response discussion in Chapter 4).

The levels for particulates in Melbourne during the study period (2006) were approximately 25% higher than during the period in which the effect estimate was derived (July 1994 – December 1997). This was likely due to a particularly severe bushfire season experienced by Victoria in 2006. The impact of this is that the results for particulates are likely to underestimate the real impact of airborne particulates in that year.

Table 5.1: Summary descriptive data

Pollutant	Average	SD	Threshold
NO_2 pphm (Jul 89–Dec 92)	2.301	0.968	1.333
NO_2 pphm (2006)	1.833	0.822	.
bsp $1x10^{-4}m^{-1}$ (Jul 94–Dec 97)	0.550	0.480	0.070
bsp $1x10^{-4}m^{-1}$ (2006)	0.778	1.782	. .

Source: NO_2 data and bsp data for 2006 were provided by the Environment Protection Authority of Victoria. bsp data for Jul 94–Dec 97 were sourced from Denison et al (2001).

Table 5.2 presents the results for the unmodified data sets, including lower (LCL) and upper (UCL) 95% confidence levels for the estimates of the proportion of asthma hospitalisations due to air pollution.

The unmodified results suggest that approximately 549 (8.9%) of the 6,200 asthma hospitalisations in Melbourne were related to NO_2 in 2006; and 150 (4.2%) of the 3,533 asthma hospitalisations for 0–14 year olds were due to particulate exposure.

Table 5.2: Asthma hospitalisations due to NO_2 and particulates (unmodified)—Melbourne 2006

		Hospitalisations attributed to pollutant			% of total hospitalisations		
Pollutant	Total hospitalisations	Mean	LCL	UCL	Mean	LCL	UCL
NO_2 pphm (unmodified)	6200 (all ages)	549	114	855	8.9	1.8	13.8
bsp $1x10^{-4}m^{-1}$ (unmodified)	3533 (0–14yrs)	150	52	244	4.2	1.5	6.9

The modified calculations used thresholds based on the data presented in Table 5.1. The minimum threshold used to modify the data for NO_2 was 1.333 pphm. The threshold for the bsp readings was 0.070.

The modified data (Table 5.3) suggests that around 193 (3.1%) or between 0.6% and 4.8% of the 6,200 asthma hospitalisations in Melbourne in 2006 were related to exposure to NO_2. Of

the 3,533 asthma hospitalisations for children aged between 0–14 years, this data suggests that 3.9% (136) or between 1.3% and 6.3% were related to exposure to particulates.

Table 5.3: Asthma hospitalisations due to NO_2 and particulates (modified)—Melbourne 2006

Pollutant	Total hospitalisations	Hospitalisations attributed to pollutant			% of total hospitalisations		
		Mean	LCL	UCL	Mean	LCL	UCL
NO_2 pphm (modified)	6200 (all ages)	193	40	300	3.1	0.6	4.8
bsp $1x10^{-4}m^{-1}$ (modified)	3533 (0–14yrs)	136	47	222	3.9	1.3	6.3

6 Conclusions and future challenges

This report is designed to facilitate discussion on methods for monitoring the health impact of air pollution, given its importance for respiratory conditions and growing relevance in the context of climate change. It reviews the research that has been conducted in Australia and presents a methodology that can be used to enhance the monitoring of asthma as well as the health impact of air pollution more generally. It then applies this methodology to a real case—Melbourne, in the 2006 calendar year. The results serve to highlight key gaps in the available research as well as provide estimations of the impact of certain pollutants on asthma hospitalisations.

Due to a range of data limitations, analysis is only possible for certain pollutants. Presented here are estimations of the independent contributions of nitrogen dioxide (NO_2) and particulates to asthma hospitalisations in Melbourne in 2006. These estimates include an adjustment to account for an unknown exposure–response relationship between the pollutant and its impact on asthma hospitalisations. The adjusted results suggest that around 193 (3.1%) of the 6,200 asthma hospitalisations were related to exposure to NO_2 in Melbourne in 2006, and 136 (3.9%) of the 3,533 asthma hospitalisations of 0–14 year olds were related to particulates in the air.

Given that other pollutants have been found to be linked with asthma hospitalisations, these results suggest that the cumulative effect of air pollution on asthma hospitalisations is worth measuring. It is also worthwhile noting that hospitalisation is an extreme outcome for asthma and that many more people are likely to have had their daily lives interrupted as a result of air pollution.

Despite the reasonably large body of work that has been done on the health impact of air pollution in Australia and internationally, crucial gaps remain. A lack of conclusive research into the exposure–response relationship for each pollutant restricts the precision of the health impact function outlined here. More work is required to detail the nature of the relationship between exposure to a pollutant and asthma exacerbation, particularly in regard to the exposure–response curve and the degree of interaction between pollutants.

This is particularly relevant to the estimates presented regarding particulate exposure. Some modification has been made to limit overestimation resulting from the assumption of a linear exposure–response relationship. The particulate concentrates for 2006 were, however, much higher than during the period in which the effect estimates used in the health impact function were derived. In this case, it is possible that the results underestimate the actual impact of particulates on asthma hospitalisations during 2006 but, until the exposure–response relationship for particulates is clearly understood, it is difficult to be certain.

Another important factor for future monitoring efforts is the issue of co-linearity. Some statistical techniques (e.g. generalised additive modelling) were applied in the development of the effect estimates, to attempt to ensure that only the independent effect of the pollutant was being identified. There is still some question as to whether health impact estimates for multiple pollutants can be simply added together to develop an estimate of the total impact of air pollution. Insufficient information is available on this for any definitive conclusion to be drawn at this time.

These limitations aside, this report has identified a methodology and applied it to a typical scenario to estimate the impact of air pollution on asthma hospitalisations. This is both an

important step toward incorporating attributable fractions in asthma monitoring as well as a call for further research in this area. In particular, a coordinated series of studies are required to derive effect estimates using a consistent methodology nationally. More research is also required into the exposure–response relationship of each pollutant that is found to have a significant health effect. Development in these two fields would allow the methodology proposed here to be both refined to yield more accurate results and applied more broadly to other pollutants and locations.