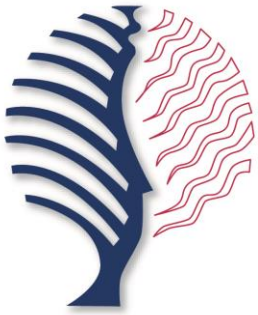

UNIVERSITY CENTRE FOR RURAL HEALTH
NORTH COAST
education research workforce

A collaboration between The University of Sydney, Southern Cross University, The University of Western Sydney, The University of Wollongong

Summary for Policy Makers of the Health Risk Assessment on Air Pollution in Australia

Prepared for:
National Environment Protection Council

November 2013

Authors:

A/Prof Geoff Morgan

Associate Professor – Environmental Health
University Centre for Rural Health – North Coast
Sydney Medical School
University of Sydney

Dr Richard Broome

Deputy Director
Environmental Health Branch
Health Protection NSW

Prof Bin Jalaludin

Director, Centre for Research, Evidence Management and Surveillance, Sydney and South Western Sydney Local Health Districts
Conjoint Professor
School of Public Health and Community Medicine
University of New South Wales

ISBN 978-1-921733-88-8
November 2013

Contents

Glossary

Anthropogenic air pollution	Air pollution resulting from human activities
Attributable cases	The number of avoided (or additional) cases in the population if the exposure was reduced (or increased)
Attributable proportion (or percentage)	The proportion (or percentage) of avoided (or additional) cases in the population if the exposure was reduced (or increased)
Background air pollution	The concentration of air pollution from natural sources, e.g. dust, sea salt, gaseous emissions from vegetation, bushfire smoke
Burden of disease	The impact of disease on a population
Concentration response function (CRF)	An estimate of the change in health outcome (e.g. mortality or hospitalisation) related to a change in air pollution exposure
Health outcome	A measure of health status, e.g. mortality, hospital admission, symptoms
Health risk assessment (HRA)	A formal process to quantify the health impacts of an exposure in an exposed population
Life expectancy	The average number of years a person of specified age and sex can expect to live. Calculated from the age- and sex-specific probability of death at each age
Micrograms per cubic metre ($\mu g/m^3$)	Particle pollution concentration unit of measure
Micrometre (µm)	A unit of measure equivalent to 1×10^{-6} metre, or one millionth of a metre
National Environment Protection (Ambient Air Quality) Measure (the Air NEPM)	A national objective to assist in protecting air quality that includes the current Australian standards for ambient air pollution, as set by the National Environment Protection Council (NEPC)
Particle pollution / particulates / PM	Particle pollution, also referred to as particulate matter, is a mixture of small particles and liquid droplets suspended in air, made up of a complex range of components including nitrates, sulphates, organic chemicals, metals, as well as soil and dust
$PM_{2.5}$	Particulate matter with an aerodynamic diameter less than or equal to 2.5 micrometres
PM_{10}	Particulate matter with an aerodynamic diameter less than or equal to 10 micrometres
Years of life lost (YLL)	The sum, over all persons in the exposed population, of the years those persons would have lived had they experienced normal life expectancy

Executive summary

In 2011 the Council of Australian Governments (COAG) identified air quality as a *Priority Issue of National Significance* and agreed that the COAG Standing Council on Environment and Water would develop a National Plan for Clean Air to improve air quality, and community health and well being. One of the first deliverables identified for the first stage of the National Plan for Clean Air was a health risk assessment of airborne particles, ozone, nitrogen dioxide and sulfur dioxide.

The report on the outcomes of this health risk assessment, referred to in this summary document as the HRA Report 2013, estimated the health impacts of air pollution in Australia for the review of national ambient air quality standards as part of the National Plan for Clean Air (Frangos & DiMarco 2013). This summary for policy makers outlines the key findings of the HRA Report 2013.

Pollutants in the air can be solid or liquid particles in suspension, or gases. The common airborne particles of primary concern are 10 µm or less in size, referred to as PM_{10}, and a subgroup of finer particles referred to as $PM_{2.5}$, which are less than 2.5 µm in size. Gaseous pollutants include ozone (O_3), nitrogen dioxide (NO_2) and sulfur dioxide (SO_2).

The health effects associated with exposure to these pollutants range from small, temporary changes in the respiratory tract and impaired lung function, to symptoms so serious they can lead to death. The consequences for people's health include restricted activity or reduced performance, hospital emergency department visits, or hospital admissions. There is strong evidence for important air pollution effects on both the respiratory and cardiovascular systems from both short-term (24-hours) and long-term (annual) exposures. The most severe health effects include a significant reduction in life expectancy of the average population, linked to long-term exposure to particles. There is no evidence of a threshold concentration below which adverse health effects of particulates are not observed.

The HRA Report 2013 applied a health risk assessment (HRA) methodology consistent with the National Environment Protection Council's framework for setting air quality standards in Australia (NEPC 2011). HRAs can address two separate but related questions:

- What is the annual 'burden' of current exposure to air pollution on health in a population, expressed as the annual percentage (and number) of attributable (or additional) deaths and hospitalisations?
- What is the 'impact' on health of increasing or decreasing air pollution under scenarios that could result from different policy settings?

The health 'burden' assessment estimates the scale of the current air pollution problem, while the health 'impact' assessment provides information about how health outcomes vary with potential changes to air pollution levels, such as changes due to policy measures to reduce air pollution. The concept of 'attributable cases' due to air pollution exposure is central to estimating its health burden and health impact. Unlike deaths due to traffic accidents, the impacts of air pollution on health cannot be directly counted, and must be evaluated from estimates of health risk based on scientific research.

The HRA Report 2013 estimated the number of attributable cases due to air pollution at *current exposures* and for three *scenario exposures*, which were hypothetical situations representing either an increase or decrease in exposures to air pollution, some long-term and some short-term. These scenarios provide information on the magnitude of potential changes to health impacts under hypothetical air pollution exposures, but they do not

directly relate to possible alternative air quality standards and are not equivalent to achieving a standard at the scenario target value.

Interpreting the estimated number of deaths attributable to air pollution exposure is difficult, and measures of total population survival time such as changes in life expectancy or the total number of years of life lived in the population are considered more meaningful. To provide an illustration of these measures and to aid interpretation of the overall results of the HRA Report 2013, additional analyses were conducted using Sydney as a case study, given its large urban population and availability of air quality data. These analyses estimated the loss of life expectancy and the number of years of life lost among residents of Sydney as a result of exposure to $PM_{2.5}$.

Air pollution is a mixture of many different pollutants and levels of common air pollutants can be linked so that they increase or decrease together, making it difficult to separate out the independent health effects due to each specific pollutant. Simply adding together the attributable cases of specific air pollutants as a means of capturing their total health burden would result in double counting and an overestimation of the total health burden. To overcome this, HRAs generally only estimate attributable health effects for one or possibly two 'index' pollutants and for non-overlapping health outcomes. The 'index' pollutant approach still includes the majority of effects of all other correlated pollutants and avoids the issue of double counting. Estimates of attributable cases due to air pollution are most robust for large population metropolitan centres. Therefore, this summary for policy makers focuses on the combined results for four major Australian cites: Sydney, Melbourne, Brisbane (including south-east Queensland) and Perth, which captures most of Australia's urban population.

Key results from the HRA Report 2013 for the four major cities were:

- From 2006 to 2010 the average annual $PM_{2.5}$ exposure ranged from 5–8 $\mu g/m^3$, while the average annual daily PM_{10} exposures ranged from 16–20 $\mu g/m^3$.
- Annual mortality attributable to long-term $PM_{2.5}$ exposures above background is equivalent to approximately 1590 deaths at typical ages (2.2%).
- Approximately 2070 cardiovascular hospital admissions across all ages (1.4%) were attributable to short-term $PM_{2.5}$ exposures above background.
- Approximately 120 hospital emergency department attendances for childhood asthma (0.6%) were attributable to short-term $PM_{2.5}$ exposure above background.
- Approximately 1130 respiratory hospital admissions in 0–14 year olds (2.2%) were attributable to short-term PM_{10} exposures above background.
- Approximately 530 pneumonia and acute bronchitis hospital admissions at ages 65+ (2.5%) were attributable to short-term PM_{10} exposures above background.

Application of the three hypothetical scenarios to data for the four cities also revealed:

- Increasing the average annual $PM_{2.5}$ exposure from 5–8 $\mu g/m^3$ to 10$\mu g/m^3$ led to an estimated 48% increase in attributable mortality at typical ages, while decreasing it to 6 $\mu g/m^3$ led to a decrease in attributable mortality of 34%.
- All three scenarios related to short-term $PM_{2.5}$ exposures reflected policy settings that would reduce current exposure levels. A potential reduction in annual attributable cardiovascular hospital admissions of up to 58%, and childhood asthma hospital emergency department visits of up to 59%, were projected under the scenario with the greatest reduction in daily $PM_{2.5}$ exposures.
- The scenarios related to short-term PM_{10} exposure all represented a decrease in exposure compared to current exposure levels, and revealed potential reductions

of approximately 65% in attributable hospital admissions for both childhood respiratory ailments and adult pneumonia and acute bronchitis, for the scenario with the largest decrease in exposure levels.

While a reduction in attributable mortality may provide an indication of the immediate benefit in the first year after a reduction in $PM_{2.5}$ exposure, alternative measures that reflect overall population survival time are required to quantify the long-term benefits. Changes in population survival time may be presented in two ways: the change in life expectancy and the change in the number of years of life lived across the whole population. The additional analysis for Sydney (based on 2008 exposure levels) revealed the following:

- The annual number of deaths in Sydney attributable to long-term $PM_{2.5}$ exposure above background is equivalent to approximately 520 deaths at typical ages.
- Approximately 6300 life years were estimated to have been lost in 2008 due to all human-made $PM_{2.5}$, and the total number of life years that could be saved over the next 100 years by reducing long-term exposure down to background levels was estimated to be 916,000. Increasing long-term $PM_{2.5}$ exposure by 2.2 $\mu g/m^3$ could lead to an additional 560,000 life years being lost over the next 100 years. (The 100 year period was selected to better capture the years of life lost (or saved) over the lifetime of those born in 2008).
- It was estimated that long-term $PM_{2.5}$ exposure in 2008 (annual average $PM_{2.5}$ of 6.3 $\mu g/m^3$) above background is responsible for a reduction in life expectancy at birth of approximately 72 days for males and 65 days for females. The three scenarios were found to reduce life expectancy further if they involved an increase in long-term $PM_{2.5}$ exposure, and increase life expectancy if long-term $PM_{2.5}$ exposure was reduced.

There is no doubt that air pollution affects health and concerns about these effects have driven improvements in air pollution around the world (COMEAP 2010). The health risk assessment summarised in this summary for policy makers describes the large magnitude of the public health burden of current air pollution exposures in four Australian cities.

The magnitude of the effects of air pollution reported here are estimates that come with a range of uncertainties intrinsic to assessments of this kind. The assumptions and methods used to estimate the health impacts of air pollution reported here are likely to have underestimated the total health benefits of reducing air pollution. While the HRA Report 2013 includes estimates of the health impacts of particulate air pollution on mortality and hospitalisation, it does not include estimates of a range of less severe health effects also associated with air pollution, including cough and other respiratory symptoms, increased medication use, and school or work absence (US EPA 2012). This report only used particulates to quantify the health effects of exposure to the complex mixture of air pollution present in Australian cities. Other pollutants such as ozone can have independent health effects, or may interact with particulates to enhance their effects on health.

The results reported here show that decreasing air pollution exposures in Australia would lead to substantial health benefits. Research suggests that the acute effects of air pollution on health (e.g. hospitalisation) would be reduced in parallel with reductions in air pollution. Reduction in the health effects of long-term exposure to air pollution (e.g. mortality) would require sustained reductions in air pollution over longer time periods and the health benefits would accrue over a longer time period. A multi-sectoral approach, engaging all relevant sectors such as transport, housing, energy production and industry, is needed to develop and effectively implement long-term policies that reduce the risks of air pollution to health (WHO 2013).

Table 1: Health effects of ambient air pollution

Air pollutant	Effects related to short-term exposure	Effects related to long-term exposure
Particulate matter	Lung inflammation	Increased lower respiratory symptoms
	Respiratory symptoms	Reduced lung function in children
	Adverse effects on the cardiovascular system	Increased chronic obstructive pulmonary disease
	Increased medication use	Reduced lung function in adults
	Increased hospitalisations	Reduced life expectancy, mainly due to cardiopulmonary mortality and probably to lung cancer
	Increased mortality	
Ozone	Adverse effects on lung function	Reduced lung function development
	Lung inflammatory reactions	
	Adverse effects on the respiratory system	
	Increased medication use	
	Increased hospitalisations	
	Increased mortality	
Nitrogen dioxide (an indicator of traffic related air pollution)	Effects on lung function, especially in asthmatics	Reduced lung function
	Increased airway allergic inflammatory reactions	Increased probability of respiratory symptoms
	Increased hospitalisations	
	Increased mortality	
Sulfur dioxide	Effects on lung function, especially in asthmatics	Probable reduced life expectancy
	Increased hospitalisations	
	Increased mortality	

Source: WHO Europe 2004

1 Introduction

In 2011 the Council of Australian Governments (COAG) identified air quality as a *Priority Issue of National Significance* and agreed that the COAG Standing Council on Environment and Water would develop a National Plan for Clean Air to improve air quality, and community health and well being. One of the first deliverables identified for the first stage of the National Plan for Clean Air was a health risk assessment of airborne particles, ozone, nitrogen dioxide and sulfur dioxide.

This summary for policy makers draws on the results of the recent health risk assessment (HRA) of air pollution in Australia (Frangos & DiMarco 2013) – referred to as the HRA Report 2013 – and is for a non-specialist audience. More detailed information on the methods and results, including assumptions and uncertainties inherent in the HRA, can be obtained from the full HRA Report 2013.

1.1 Health effects of air pollution

The common ambient (or outdoor) air pollutants of primary concern to health in Australia are:

- **particles** – particles with an aerodynamic diameter of <10 µm – referred to as PM_{10}. PM_{10} includes a subgroup of finer particles – referred to as $PM_{2.5}$ (particles with an aerodynamic diameter of <2.5 µm)
- **gases** – including ozone (O_3), nitrogen dioxide (NO_2) and sulfur dioxide (SO_2).

The health effects associated with exposure to ambient air pollution range from small transient changes in the respiratory tract and impaired lung function, to mortality, and can result in restricted activity/reduced performance, hospital emergency department visits and hospital admissions. There is strong evidence for important air pollution effects on both the respiratory and cardiovascular systems for both short-term and long-term exposures. The range of health effects associated with air pollution exposures are summarised in Table 1. The most severe health effects include a significant reduction in life expectancy of the average population due to long-term exposure to particles.

A recent United States Environmental Protection Agency (US EPA) report summarised the strength of the evidence for $PM_{2.5}$ effects on various health outcomes ranging from causal (the evidence is strong enough to conclude that $PM_{2.5}$ exposure causes the health outcome) to suggestive (the evidence suggests that $PM_{2.5}$ might cause the health outcome) and this is summarised in Table 2. $PM_{2.5}$ is now generally believed to have a *causal* or *likely causal* relationship with a small number of health outcomes and a *suggestive* relationship with many other health outcomes (US EPA 2012). The International Agency for Research on Cancer (IARC) recently classified outdoor air pollution as a mixture, and particulate matter specifically, as carcinogenic to humans (Loomis et al. 2013).

There is no evidence of a threshold concentration below which adverse health effects of PM are not observed (Pope & Dockery 2006; COMEAP 2009; Brook et al. 2010). A recent World Health Organization (WHO) review of the scientific literature since 2005 found strong evidence that the effects of $PM_{2.5}$ on a wide range of adverse health outcomes occurred at levels below those experienced in Australia, and followed a mostly linear concentration response relationship. The review also found that particles in the PM_{10} size fraction have health effects independent of $PM_{2.5}$. There is increasing evidence of the adverse effects on health of coarse particles in the size range from $PM_{2.5}$ to PM_{10}, and studies show associations between long-term exposure to PM_{10} and health, especially for respiratory outcomes. Coarse and fine particles deposit at different locations in the respiratory tract, have different sources and composition, act through partly different biological mechanisms, and result in different health outcomes. The WHO review concluded that there is good evidence to support maintaining independent short-term and long-term standards for ambient PM_{10} in addition to $PM_{2.5}$ to protect against the health effects of both fine and coarse particles (WHO 2013).

Table 2: Causal determinants for short- and long-term exposure to particulates

Long-term exposure to $PM_{2.5}$		
Size fraction	**Outcome**	**Causality determination**
$PM_{2.5}$	Cardiovascular effects	Causal
	Respiratory effects	Likely to be causal
	Mortality	Causal
	Reproductive and developmental effects	Suggestive
	Cancer, mutagenicity, and genotoxicity	Causal[a]
Short-term exposure to PM		
Size fraction	**Outcome**	**Causality determination**
$PM_{2.5}$	Cardiovascular effects	Causal
	Respiratory effects	Likely to be causal
	Mortality	Causal
$PM_{10-2.5}$	Cardiovascular effects	Suggestive
	Respiratory effects	Suggestive
	Mortality	Suggestive

Source: US EPA 2012

[a] $PM_{2.5}$ causality determination for cancer, mutagenicity, and genotoxicity amended from 'suggestive' to 'causal' based on recent International Agency for Research on Cancer (IARC) classification of outdoor air pollution as a mixture, and particulate matter specifically, as carcinogenic to humans (Loomis et al. 2013).

Some population groups are more susceptible to the effects of air pollution. Table 3 identifies groups that are susceptible to various adverse health effects from particulate exposure and the overall health relevance of these effects. Associations between mortality and $PM_{2.5}$ have been observed in a number of subpopulations including those with pre-existing respiratory disease, cardiovascular disease, diabetes, people aged over 65 years, females, deaths out of hospital and those of lower socio-economic status (Pope & Dockery 2006).

Table 3: Summary of groups susceptible to adverse health effects from particulate exposure

Health effects	Who is susceptible?	Overall health relevance
Acute (short-term) exposure		
Mortality	Elderly, infants, persons with chronic cardiopulmonary disease, influenza or asthma	Obviously relevant. How much life shortening is involved and how much is due to short-term mortality displacement (harvesting) is uncertain.
Hospitalisation / other health care visit	Elderly, infants, persons with chronic cardiopulmonary disease, pneumonia, influenza or asthma	Reflects substantive health impacts in terms of illness, discomfort, treatment costs, work or school time lost, etc.
Increased respiratory symptoms	Most consistently observed in people with asthma and children	Mostly transient with minimal overall health consequences, although for a few there may be short-term absence from work or school due to illness.
Decreased lung function	Observed in both children and adults	For most, effects seem to be small and transient. For a few, lung function losses may be clinically relevant.
Plasma viscosity, heart rate variability, pulmonary inflammation	Observed in both healthy and unhealthy subjects. No studies of children	Effects seem to be small and transient. Overall health relevance is unclear, but may be part of pathophysiologic pathway linking PM with cardiopulmonary mortality.
Chronic (long-term) exposure		
Increased mortality rates, reduced survival times, chronic cardiopulmonary disease, reduced lung function	Observed in broad-based cohorts or samples of adults and children (including infants). All chronically exposed are potentially affected	Long-term repeated exposure appears to increase the risk of cardiopulmonary disease and mortality. May result in lower lung function. Average loss of life expectancy in highly polluted cities may be as much as a few years.

Source: Pope & Dockery 2006

1.2 Health risk assessment and Australian air quality standards

The HRA Report 2013 was undertaken to provide estimates of the health impacts of air pollution in Australia for the review of national ambient air quality standards as part of the development of the National Plan for Clean Air. HRA is a pillar of the National Environment Protection Council's (NEPC) framework for setting air quality standards in Australia (NEPC 2011). The current Australian standards for ambient air pollution are set out in the National Environment Protection (Ambient Air Quality) Measure (the 'Air NEPM'), and are summarised in Table 4.

Table 4: Australian standards and guidelines for ambient air pollution

Pollutant	Averaging period	Maximum concentration	Maximum number of exceedances
Carbon monoxide	8 hours	9 ppm	1 day a year
Nitrogen dioxide	1 hour 1 year	0.12 ppm 0.03 ppm	1 day a year none
Ozone	1 hour 4 hours	0.10 ppm 0.08 ppm	1 day a year 1 day a year
Sulfur dioxide	1 hour 1 day 1 year	0.20 ppm 0.08 ppm 0.02 ppm	1 day a year 1 day a year none
Lead	1 year	0.5 µg/m^3	none
PM_{10}	24 hours	50 µg/m^3	5 days a year
$PM_{2.5}$	24 hours 1 year	25 µg/m^3 8 µg/m^3	N/A

Source: Department of the Environment 2013

HRAs can address two separate but related questions:

- What is the annual 'burden' of current exposure to air pollution on health in a population, expressed as the annual percentage (and number) of attributable (or additional) deaths and hospitalisations?
- What is the 'impact' on health of increasing or decreasing air pollution under scenarios that could result from different policy settings?

The health 'burden' assessment estimates the scale of the current air pollution problem, while the health 'impact' assessment provides information about how health outcomes vary with potential changes to air pollution levels, including those due to policy measures to reduce air pollution. The concept of 'attributable cases' due to air pollution exposure is central to estimating the health burden and health impact of air pollution. Unlike deaths due to traffic accidents the impacts of air pollution on health cannot be directly counted, and must be measured in an exposed population by applying estimates of health risk from scientific research to estimate the attributable cases.

Key steps in the health risk assessment included:

- **Hazard assessment**: A key piece of information required for quantifying the health effects of air pollution is an estimate of the change in health outcome (e.g. mortality or hospitalisation) related to a change in air pollution exposure, known as a 'concentration response function' (CRF). An expert review of epidemiology studies on air pollution and health identified CRFs to be applied in the HRA Report 2013 (Jalaludin & Cowie 2012).
- **Exposure assessment**: Estimates of the exposure of the population to air pollutants. The HRA Report 2013 estimated population exposures to outdoor air pollution from 2006 to 2010 for 32 Australian cities including the major metropolitan areas of Sydney, Melbourne, Brisbane and Perth, which captured most of the Australian urban population, referred to as *current exposures*. The HRA Report 2013 also estimated the increase or decrease in exposures to air pollution under three scenarios – referred to as *scenario exposures*.
- **Risk characterisation**. Risk characterisation integrates, evaluates and summarises the *hazard assessment* and the *exposure assessment.* It also evaluates the overall quality of the assessment and the degree of confidence in the results. For each of the 32 Australian cities, the HRA Report 2013 estimated the number of deaths and hospitalisations attributable to air pollution from *current exposures*, and the three *scenario exposures*. An additional analysis was conducted to provide additional detail for Sydney as a case study on the long-term mortality benefits of pollution reduction in terms of increased life expectancy ('life-years'), rather than in terms of the number of deaths avoided.

2 Hazard assessment – expert review of scientific evidence

While the common air pollutants are associated with a broad range of health outcomes, it is clear that evidence for some of these outcomes is either inadequate or inconsistent, leading to difficulty in establishing robust CRFs for use in HRA. The CRF determined by a particular epidemiological study will be influenced by the study methods as well as factors related to the study location, including the sources of air pollution and patterns of exposure, climate, the population and their health. This can lead to differences in CRFs between studies from different locations. Typically such inconsistencies are resolved through a review of the overall evidence by an expert panel. The expert review of epidemiological studies of air pollution and health (Jalaludin & Cowie 2012) identified clear scientific evidence for a range of mortality and hospitalisation outcomes associated with exposure to air pollutants ($PM_{2.5}$, PM_{10}, O_3, NO_2, SO_2) and these CRFs were applied in the HRA Report 2013.

Estimates of the attributable cases due to air pollution exposure can vary depending on the CRFs that are used in the calculations. Therefore the selection of the appropriate CRFs (from among the hundreds of published CRFs) is of utmost importance. The selected CRFs should be applicable to the populations living in Australian cities and towns. CRFs that are derived from overseas epidemiological studies may not always be the most appropriate as Australian geography, climate, air pollutant sources and patterns of exposure may be quite different compared to the areas where the original epidemiological studies were undertaken.

The CRFs used in the HRA Report 2013 are based on epidemiological studies set in large cities in the USA or Europe, as well as a study set in major Australian cities (including Sydney, Melbourne, Brisbane and Perth). Due to the inherent uncertainties of generalising these CRFs to relatively small cities and towns, estimates of attributable cases due to air pollution are most robust for large metropolitan centres. Therefore, this summary report focuses on the combined results for the four major Australian cites of Sydney, Melbourne, Brisbane (including south-east Queensland) and Perth.

3 Exposure assessment – current exposures to air pollution and possible future exposure scenarios

The HRA Report 2013 estimated exposure to air pollution by averaging air pollutant concentrations at all available ambient air pollution monitoring sites within a city. The city-wide average approach to estimating air pollution exposure is consistent with the methods generally used in epidemiological studies. However, the *current exposures* and *scenario exposures* estimated using this approach cannot be directly compared with the Australian Air NEPM standards as the standards are assessed at specific air monitoring sites, rather than the average of several sites.

Ambient air pollution is produced from both human activities (also referred to as anthropogenic air pollution) and natural background sources (including wind blown dust, fires, sea salt and gaseous emissions from vegetation). The HRA Report 2013 estimated the number of attributable cases (as well as the attributable proportion) at *current exposures* and the three hypothetical *scenario exposures*. *Scenario exposures* were obtained by adjusting all *current exposures* above background proportionally, so that the maximum exposure from 2006 to 2010 is set equal to the scenario level. This can involve scaling current exposures up or down, depending on whether the scenario level represents an exposure above or below the maximum city-wide average exposure for the location of interest. The various scenario exposures and their interpretation for long-term $PM_{2.5}$, short-term $PM_{2.5}$, and short-term PM_{10} exposures are shown in Table 5. While long-term PM_{10} exposure is associated with mortality, evidence indicates that long-term exposure to $PM_{2.5}$ has the strongest association with mortality. Hence, to avoid double counting of effects, long-term PM_{10} exposure is not assessed in this report. It is important to note that the scenarios provide information on the magnitude of health impacts associated with potential changes to average city-wide air pollution exposures. The scenarios do not directly relate to possible alternative air quality standards and are not equivalent to achieving a standard at the scenario target value given that compliance with standards is assessed at each monitoring site separately (and not as a city-wide average exposure).

All estimates of attributable mortality and hospitalisation were for exposures above average national background exposure. The average national background exposure was estimated to be 2.7 $\mu g/m^3$ for $PM_{2.5}$ and 7.5 $\mu g/m^3$ for PM_{10}. From 2006 to 2010, the average annual $PM_{2.5}$ *current exposures* in the four major Australian cities studied ranged from 5–8 $\mu g/m^3$, while the average annual daily PM_{10} *current exposures* ranged from 16–20 $\mu g/m^3$.

Two different analyses were conducted depending on the health outcome being assessed:

- **'Daily' or 'short-term' exposure effects on health**: This analysis uses daily exposures as an estimate of short-term population exposure. Daily attributable cases (deaths, hospitalisations) were estimated and then an annual change was obtained by summing the daily changes. Thus the results, although described as 'daily', estimate annual attributable cases due to daily (short-term) air pollution exposure.
- **'Annual' or 'long-term' exposure effects on health**: This analysis uses annual average exposures as an estimate of long-term population exposure. Thus the results estimate annual attributable cases due to annual (long-term) air pollution exposure.

Annual deaths and hospitalisations attributable to *current exposures* were averaged over a five-year period (2006–2010) for each of the four cities. Averaging over this period is intended to be representative of a range of environmental conditions (such as dry and wet years) and capture the variability in baseline mortality and hospitalisation and also air pollution exposure for those years.

Table 5: Summary of current and scenario exposures from 2006 to 2010 in Sydney, Melbourne, Brisbane and Perth, and their interpretation

Scenario	Maximum exposure (2006–2010)	Comments/interpretation
$PM_{2.5}$ – long-term exposure		
Current	Maximum of annual average exposure[1]: range = 7.1 µg/m^3 to 8.3 µg/m^3	Attributable cases estimated for *current exposures* above a background
Scenario One	Maximum annual average exposure: 10 µg/m^3	*Scenario exposures* increased resulting in increased attributable cases compared to *current exposures*
Scenario Two	Maximum annual average exposure: 8 µg/m^3	*Scenario exposures* marginally increased resulting in marginally increased attributable cases compared to *current exposures*
Scenario Three	Maximum annual average exposure: 6 µg/m^3	*Scenario exposures* decreased resulting in decreased attributable cases compared to *current exposures*
$PM_{2.5}$ – short-term exposure		
Current	Maximum of daily exposure[2]: range = 26 µg/m^3 to 34 µg/m^3	Attributable cases estimated for *current exposures* above background
Scenario One	Maximum daily exposure: 25 µg/m^3	*Scenario exposures* decreased resulting in decreased attributable cases compared to *current exposures*
Scenario Two	Maximum daily exposure: 20 µg/m^3	*Scenario exposures* decreased resulting in decreased attributable cases compared to *current exposures*
Scenario Three	Maximum daily exposure: 15 µg/m^3	*Scenario exposure*s decreased resulting in decreased attributable cases compared to *current exposures*
PM_{10} – short-term exposure		
Current	Maximum of daily exposure[3]: range =56 µg/m^3 to 70 µg/m^3	Attributable cases estimated for *current exposures* above a background
Scenario One	Maximum daily exposure: 50 µg/m^3	*Scenario exposure* decreased resulting in decreased attributable cases compared to *current exposures*
Scenario Two	Maximum daily exposure: 40 µg/m^3	*Scenario exposure* decreased resulting in decreased attributable cases compared to *current exposures*
Scenario Three	Maximum daily exposure: 30 µg/m^3	*Scenario exposure* decreased resulting in decreased attributable cases compared to *current exposures*

[1] Annual attributable cases associated with long term-$PM_{2.5}$ exposure are derived from an annual average $PM_{2.5}$ exposure for the four cities (5–8 µg/m^3) calculated by averaging daily exposures over each year. Maximum annual exposures over the four cities range from 7.1–8.3 µg/m^3.

[2] Annual attributable cases associated with short-term $PM_{2.5}$ exposure are calculated as the sum of daily attributable cases derived from daily $PM_{2.5}$ exposures. Maximum daily exposures over the four cities range from 26–34 µg/m^3.

[3] Annual attributable cases associated with short term PM_{10} exposure are calculated using averaged daily PM_{10} exposures for the four cities (16–20 µg/m^3). Maximum daily exposures over the four cities range from 56–70 µg/m^3.

4 Risk characterisation – the health effects of air pollution in four Australian cities

4.1 Measures of effect used to summarise health impacts

The HRA Report 2013 characterises the risk associated with air pollution by combining information from both the hazard assessment and the exposure assessment. The results are provided as:

- the proportion (and annual number) of hospitalisations and deaths attributable to current exposures to air pollution
- the proportion (and annual number) of hospitalisations and deaths attributable to air pollution under three alternative scenarios.

It is common for air pollution HRAs to estimate the number of 'attributable' cases. While the interpretation of attributable hospitalisation is relatively straightforward, interpretation of attributable deaths is less clear. Because of these difficulties, some groups, most notably the UK Committee on the Medical Effects of Air Pollution (COMEAP), have argued that measures of total population survival time provide more meaningful information about the mortality impacts of exposure to air pollution (COMEAP 2010). Such measures include the change in life expectancy associated with a change in exposure to PM and the change in the total number of years of life lived in the population.

To provide an illustration of these measures and to aid interpretation of the overall results of the HRA Report 2013, additional analyses were conducted using Sydney as a case study, given its large urban population and availability of air quality data, to estimate the loss of life expectancy and the number of years of life lost among Sydney residents as a result of exposure to $PM_{2.5}$. These additional analyses used methods adapted from those described in a recent COMEAP report (COMEAP 2010). A detailed description of the purpose, methods and results of this additional analysis is provided in Appendix 1 of this report.

4.2 Index pollutant approach to avoid double counting

While HRAs can quantify the health effects of all the common air pollutants they usually focus on one or two pollutants. Air pollution is a mixture of many different pollutants and exposure to the common air pollutants can be correlated so that they increase or decrease together. This makes it difficult for epidemiological studies to separate out the independent health effects of each specific pollutant. Therefore, simply adding the attributable cases due to each specific air pollutant as a means of capturing the total health burden attributable to all the common air pollutants would result in double counting and an overestimation of the total health burden.

Double counting can also occur as $PM_{2.5}$ is not only correlated with PM_{10} but is also a subset of PM_{10}. Therefore, adding the attributable cases due to PM_{10} and $PM_{2.5}$ exposures to obtain an overall health burden attributable to total particulate exposure would also result in double counting.

Epidemiological research has demonstrated associations between $PM_{2.5}$ and *all-cause mortality* as well as *cardiopulmonary mortality*. Again, adding these estimates would overestimate the attributable mortality due to $PM_{2.5}$ as *cardiopulmonary mortality* is a subset of *all-cause mortality*. $PM_{2.5}$ exposure is associated with short-term mortality and long-term mortality. However, it is generally accepted that estimates of attributable mortality due to long-term exposure include at least some of the attributable mortality due to short-term $PM_{2.5}$ exposure. Because the mortality effect of long-term $PM_{2.5}$ exposures is far greater, it is given precedence, and to avoid double counting, attributable mortality due to short-term $PM_{2.5}$ exposure is not included with long-term estimates.

HRAs generally take a conservative approach and only estimate attributable health effects for one or possibly two 'index' pollutants and for non-overlapping health outcomes. Currently, the US EPA (US EPA 2011b) and the European Commission (European Commission 2005) focus only on $PM_{2.5}$ and O_3 in their HRAs due to the greater weight of evidence for health effects for these pollutants. The 'index' pollutant approach still includes the majority of effects of all other correlated pollutants and avoids the issue of double counting.

As with any statistical analysis and modelling, each step of the HRA includes a range of data analysis decisions and assumptions resulting in uncertainties and limitations in the conclusions that can be drawn. These uncertainties are generally addressed in the HRA by making conservative assumptions that tend to underestimate the attributable cases due to air pollution, and by conducting sensitivity analyses.

This report focuses on the attributable mortality and hospitalisation due to particulate exposure for the following particulate measures and non-overlapping health outcomes:

- long-term $PM_{2.5}$ exposure and all-cause mortality in those aged 30+ years
- short-term $PM_{2.5}$ exposure and all cardiovascular hospital admissions in all ages
- short-term $PM_{2.5}$ exposure and asthma hospital emergency department (ED) attendance in children 1–14 years
- short-term PM_{10} exposure and all respiratory hospital admissions in children aged 0–14 years
- short-term PM_{10} exposure and pneumonia and acute bronchitis hospital admissions in those aged 65+ years.

Table 6 summarises the estimates of $PM_{2.5}$ and PM_{10} attributable annual mortality and hospitalisation due to *current exposure* and *scenario exposures* for Sydney, Melbourne, Brisbane and Perth. The results presented below are for exposure data that excluded days where pollutant concentrations were influenced by extreme events such as dust storms and bushfires. However, the results for the health burden of air pollution above background were similar if the extreme days were included in the analyses.

The estimates of attributable mortality and hospitalisation due to particles reported here are generally consistent with previous Australian and US HRA estimates (Burgers & Walsh 2002; Begg 2007; US EPA 2010).

Figure 1 is a series of charts illustrating the changes brought about by application of the three scenarios to each of the health outcomes listed in Table 6.

Table 6: Estimates of $PM_{2.5}$ and PM_{10} attributable annual mortality and hospitalisation due to *current exposure* and *scenario exposures* in Sydney, Melbourne, Brisbane and Perth, averaged over the period 2006 to 2010

Health outcome	Pollutant	Time period	Scenario	Number of attributable cases (% increase / decrease compared to current)	Proportion of attributable cases (95% confidence interval)
All-cause mortality 30+ years	$PM_{2.5}$	Long-term exposure	Current	1586 cases	2.2% (1.4% to 3.0%)
			Scenario 1: 10 µg/m^3	+760 (+48%)	
			Scenario 2: 8 µg/m^3	+110 (+7%)	
			Scenario 3: 6 µg/m^3	-533 (-34%)	
Cardiovascular hospital admissions all ages	$PM_{2.5}$	Short-term exposure	Current	2067 cases	1.4% (0.6% to 2.1%)
			Scenario 1: 25 µg/m^3	-481 (-23%)	
			Scenario 2: 20 µg/m^3	-837 (-40%)	
			Scenario 3: 15 µg/m^3	-1189 (-58%)	
Asthma hospital emergency department attendance 1–14 years	$PM_{2.5}$	Short-term exposure	Current	124 cases	0.6% (0.4% to 0.8%)
			Scenario 1: 25 µg/m^3	-34 (-27%)	
			Scenario 2: 20 µg/m^3	-54 (-43%)	
			Scenario 3: 15 µg/m^3	-74 (-59%)	
Respiratory hospital admissions 0–14 years	PM_{10}	Short-term exposure	Current	1130 cases	2.2% (0.2% to 4.3%)
			Scenario 1: 50 µg/m^3	-373 (-33%)	
			Scenario 2: 40 µg/m^3	-588 (-49%)	
			Scenario 3: 30 µg/m^3	-733 (-65%)	
Pneumonia and acute bronchitis hospital admissions 65+ years	PM_{10}	Short-term exposure	Current	529 cases	2.5% (0.3% to 5.0%)
			Scenario 1: 50 µg/m^3	-175 (-33%)	
			Scenario 2: 40 µg/m^3	-255 (-49%)	
			Scenario 3: 30 µg/m^3	-344 (-65%)	

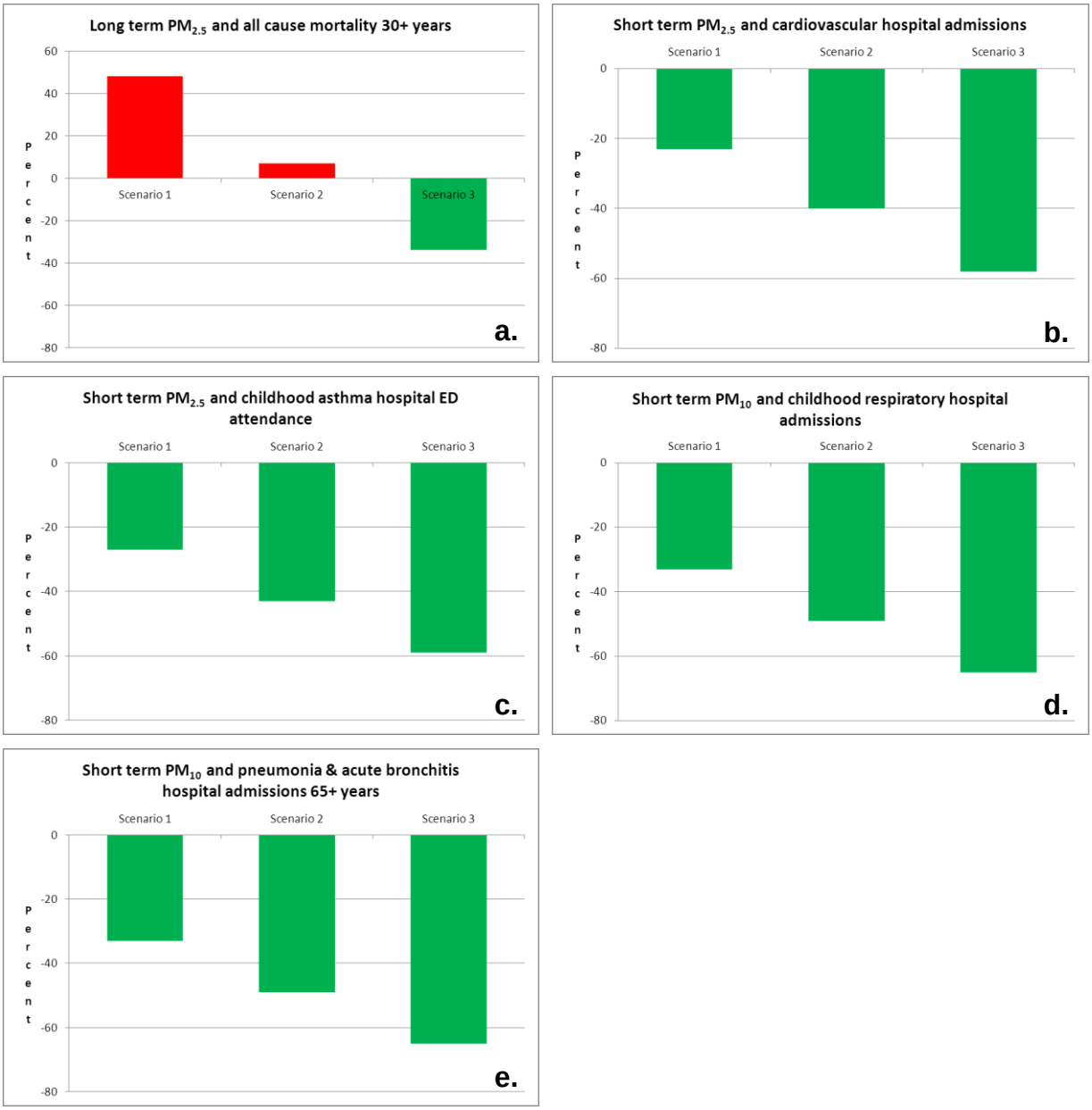

Figure 1: Percentage change in attributable annual mortality and hospitalisations due to Scenario One, Scenario Two and Scenario Three exposures compared to current particulate pollution exposures over the four cities.
Note: Red (positive) indicates increase compared to current exposure, green (negative) indicates decrease compared to current exposure.

4.3 Long-term $PM_{2.5}$ exposure – attributable mortality

Current annual average $PM_{2.5}$ exposure in Sydney, Melbourne, Brisbane and Perth ranged from 5.1 μg/m^3 to 7.8 μg/m^3. In these four major cities annual mortality attributable to current long-term $PM_{2.5}$ exposures above background is estimated to be equivalent to approximately 1590 deaths (2%) at typical ages. This estimate of attributable (or additional) deaths due to long-term $PM_{2.5}$ exposure does not provide information on the ages at which the deaths occur nor the fact that the loss of life associated with the deaths varies with age.

These results describe the current burden of mortality associated with past and present exposure to human-made $PM_{2.5}$. In addition to understanding this burden, it is also useful to consider the impact of scenarios that would result in sustained changes to $PM_{2.5}$ exposure. This was done by assessing three different scenarios based on increasing / decreasing current $PM_{2.5}$ exposures.

The current maximum annual $PM_{2.5}$ exposures in the four cities ranged from 7.1 to 8.3 $\mu g/m^3$, so compared to current exposures Scenario One (10 $\mu g/m^3$) reflects a situation where exposure to $PM_{2.5}$ is generally increased, while Scenario Two (8 $\mu g/m^3$) would result in similar $PM_{2.5}$ exposures, and Scenario Three (6 $\mu g/m^3$) would result in decreased $PM_{2.5}$ exposures (Figure 1a). Under Scenario One attributable mortality at typical ages would increase by 48% (760 additional deaths) while Scenario Three exposures would reduce attributable mortality at typical ages by 34% (approx. 530 fewer deaths) compared to *current exposures*. Scenario Two exposures would result in a marginal increase in attributable mortality at typical ages.

4.4 Short-term $PM_{2.5}$ exposure – attributable cardiovascular hospital admissions

Current annual average daily $PM_{2.5}$ exposure in the four cities ranged from 5.1 $\mu g/m^3$ to 7.8 $\mu g/m^3$. Annual cardiovascular hospital admissions attributable to current short-term $PM_{2.5}$ exposure above background is estimated to be about 2070 cases, or 1.4% (95% confidence interval: 0.6% to 2.1%) over the four cities.

The current maximum daily $PM_{2.5}$ exposures in the four cities ranged from 26 to 34 $\mu g/m^3$ and so all three scenarios (Scenario One: 25 $\mu g/m^3$; Scenario Two: 20 $\mu g/m^3$; Scenario Three: 15 $\mu g/m^3$) reflect policy settings that would reduce *current exposures* to short-term $PM_{2.5}$. Compared to current exposures in the four cities Scenario One exposures would reduce annual attributable cardiovascular hospital admissions by 23% (about 480 admissions), while the larger reductions to exposure under Scenario Three would result in a reduction of 58% (approximately 1190 admissions) (Figure 1b).

4.5 Short-term $PM_{2.5}$ exposure – attributable childhood asthma hospital emergency department attendance

Annual childhood asthma hospital emergency department attendance attributable to current short-term $PM_{2.5}$ exposure above background is estimated to be about 120 cases (0.6%; 95% confidence interval: 0.4% to 0.8%) over the four cities.

All three short-term $PM_{2.5}$ scenarios (Scenario One: 25 $\mu g/m^3$; Scenario Two: 20 $\mu g/m^3$; Scenario Three: 15 $\mu g/m^3$) decreased exposures compared to *current exposures* in the four cities. Therefore, reducing *current exposures* under Scenarios One, Two and Three would reduce annual attributable childhood asthma hospital emergency department attendance by 27% (approximately 30 cases), 43% (about 50 cases) and 59% (about 70 admissions), respectively, over the four cities (Figure 1c). Because hospital emergency department treatment only forms a small proportion of childhood asthma treatment in the population, it is likely that the actual improvement in asthma incidence would be greater than that represented solely by asthma hospital emergency department attendance (AIHW 2003, 2010).

4.6 Short-term PM_{10} exposure – attributable respiratory hospital admissions

Current annual average daily PM_{10} exposure in the four cities ranged from 16.4 $\mu g/m^3$ to 19.7 $\mu g/m^3$. Respiratory hospitalisation attributable to current short-term PM_{10} exposure

above background is estimated to be about 1130 cases, or 2% of annual respiratory 0–14 years hospital admissions (95% confidence interval: 0.2% to 4.3%), and approximately 530 cases, or 2.5% of annual pneumonia and acute bronchitis 65+ years hospital admissions (95% confidence interval: 0.3% to 5.0%).

The current maximum daily PM_{10} exposures in the four cities ranged from 56 $\mu g/m^3$ to 70 $\mu g/m^3$ and so all three short-term PM_{10} scenarios (Scenario One: 50 $\mu g/m^3$; Scenario Two: 40 $\mu g/m^3$; Scenario Three: 30 $\mu g/m^3$) decreased exposures compared to *current exposures* in the four cities. Therefore, reducing *current exposures* under Scenario One would reduce attributable childhood respiratory hospital admissions by 33% (approximately 370 admissions; Figure 1d) and adult pneumonia and acute bronchitis hospital admissions by 33% (approximately 180 admissions; Figure 1e) compared to current exposures. The larger decrease in PM_{10} exposures under Scenario Three would reduce attributable childhood respiratory hospital admissions by 65% (about 730 admissions) and attributable adult pneumonia and acute bronchitis hospital admissions by 65% (approximately 340 admissions) compared to current exposures.

5 Additional analysis for Sydney on long-term exposure to particles and life expectancy

The HRA Report 2013 presents changes in the number of 'attributable' deaths related to changes in the level of $PM_{2.5}$ exposure. It is tempting to conclude from this that these changes in number of 'attributable' deaths would remain constant in each year that the change in $PM_{2.5}$ exposure persisted. This is not the case. While 'attributable' deaths provide an indication of the immediate benefit **in the first year** after a reduction in $PM_{2.5}$, alternative measures that reflect **overall population survival time** are required to quantify the long-term benefits. Changes in population survival time may be presented in two ways:

1. the change in life expectancy – the average length of life of an individual in the population of interest
2. the change in the number of years of life lived in the whole population.

The relationship between the number of attributable deaths presented in the HRA Report 2013 and information about the overall change in population life expectancy, is illustrated in additional analyses for Sydney.

5.1 Sydney analysis – long-term $PM_{2.5}$ exposure and years of life lost

The annual number of deaths in Sydney attributable to long-term $PM_{2.5}$ exposure in 2008 (annual average $PM_{2.5}$ of 6.3 µg/m^3) above background is equivalent to approximately 520 deaths at typical ages (or 2.0% of annual deaths, 95% confidence interval: 1.2% to 2.7%). To illustrate what is meant by 'equivalent to deaths at typical ages', Figure 2 shows the age distribution of the $PM_{2.5}$ related mortality effect as well as the age distribution of all deaths that occur in Sydney. The two distributions have a very similar shape. Note that no $PM_{2.5}$ related mortality effect is recorded among people aged less than 30 years because the effect estimate used for risk assessment is derived from a study that was limited to participants aged over 30 years.

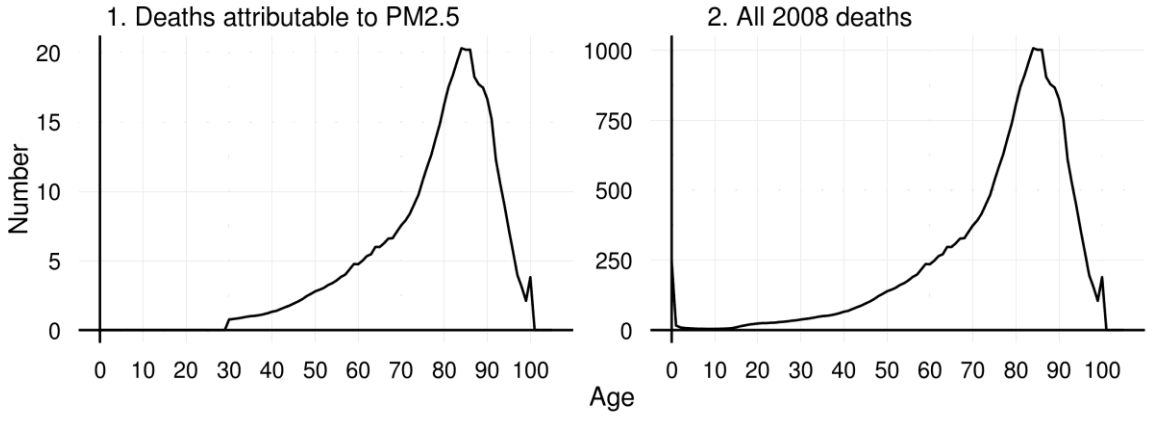

Figure 2: Age distribution of deaths in Sydney (note different y-axis scales)

Estimation of the number of years of life that were lost in 2008 as a result of long-term exposure to human-made $PM_{2.5}$ is relatively straightforward. Each attributable death is multiplied by the relevant age-specific life expectancy. For example, a 40 year old male

has a remaining life expectancy of approximately 42 years. Therefore, each death of a 40 year old male is responsible for a loss of 42 years of potential life.

The number of years of life lost in 2008 due to long-term $PM_{2.5}$ in Sydney above background (that is, the years of life lost due to all human-made $PM_{2.5}$) is estimated to be 6300 life years.

Estimating the impact over time of increasing or reducing exposure to $PM_{2.5}$ requires somewhat more complicated calculations because a change in $PM_{2.5}$ levels has an impact on both the size and age distribution of the impacted population. The analysis assesses the impact of reducing exposure to $PM_{2.5}$ by 1 $\mu g/m^3$ in Sydney (equivalent to Scenario Three in 2008) over the next 100 years from 2008, by examining the difference between the Sydney population exposed to 1 $\mu g/m^3$ less $PM_{2.5}$, compared to the Sydney population with no change in exposure.

In the first year of reduced exposure, there would be approximately 140 fewer deaths in Sydney compared to a situation with no reduced exposure. In the same year, 70 additional years of life would be lived because each of the 140 people who avoided death in that year gain on average 6 months of life. After 50 years, the less exposed population has accrued an additional 100,000 years of life. After 100 years, this has increased to approximately 250,000 years of life. If exposure was reduced down to background levels, after 100 years an estimated 916,000 life years would be saved.

If long-term $PM_{2.5}$ exposure in Sydney were increased by 2.2 $\mu g/m^3$ compared to 2008 exposures (equivalent to Scenario One) an estimated additional 560,000 life years would be lost over the next 100 years. If long-term $PM_{2.5}$ exposure in Sydney were increased by 0.6 $\mu g/m^3$ compared to 2008 exposures (equivalent to Scenario Two) an estimated additional 153,000 life years would be lost over the next 100 years.

5.2 Sydney analysis – long-term $PM_{2.5}$ exposure and change in life expectancy

Based on additional analyses it is estimated that in Sydney long-term $PM_{2.5}$ current exposure in 2008 (annual average $PM_{2.5}$ of 6.3 $\mu g/m^3$) above background is responsible for a **reduction** in life expectancy at birth of approximately 72 days for males and 65 days for females.

Compared to current exposures, Scenario One equivalent exposure would **reduce** life expectancy by an estimated 43 days for males and 39 days for females. Compared to current exposures, Scenario Two equivalent exposure would **reduce** life expectancy by 11 days for males and 11 days for females. Compared to current exposures, Scenario Three equivalent exposures would **increase** life expectancy by 20 days for males and 18 days for females.

6 Wider questions about exposure to particle pollution

6.1 Are infrequent peaks in pollution more harmful than ongoing mid-range pollution?

Epidemiological evidence indicates that the adverse health effects of short-term $PM_{2.5}$ exposure are driven primarily by the numerous middle range exposure days within the air pollution distribution and not by the peak exposure days. For example, HRAs conducted by the US EPA found that most of the risk associated with short-term exposures results from the large number of days during which the 24-hour average concentrations are in the low to mid range, below the peak 24-hour concentrations (US EPA 2011a). The US EPA also states that there is no evidence suggesting that risks associated with long-term $PM_{2.5}$ exposures are likely to be disproportionately driven by peak 24-hour concentrations (US EPA 2011a).

Therefore, control strategies that focus primarily on reducing extreme days are less likely to achieve reductions in $PM_{2.5}$ exposures that contribute most to health effects, when compared to an approach that focuses on reducing the middle range of the $PM_{2.5}$ exposure distribution (US EPA 2011a).

6.2 Is there a safe level of particle pollution?

There is no evidence of a threshold concentration below which adverse health effects of PM are not observed (Pope & Dockery 2006; COMEAP 2009; Brook et al. 2010). Therefore, in cities where PM exposures are generally low compared to air quality standards, there is still a health benefit gained by further reductions in PM, especially in areas of high population density (WHO 2013).

The HRA findings confirm that current exposures to outdoor air pollution contribute substantially to mortality and hospitalisation in Australia. A recent UK report estimated the total population survival time lost due to particulate exposure was larger than the mortality impacts of environmental tobacco smoke or road traffic accidents. Correspondingly, reductions in population exposure to air pollution expressed as annual average $PM_{2.5}$ can have appreciable benefits in terms of reduced death rates and the associated increase in life expectancy, and in terms of the total years lived by the population as a whole (COMEAP 2010).

7 Conclusions

There is no doubt that air pollution affects health and concerns about these effects have driven improvements in air pollution around the world (COMEAP 2010).

The health risk assessment summarised in this report describes the large magnitude of the public health burden of current air pollution exposures in four Australian cities. From 2006 to 2010 the average annual $PM_{2.5}$ in the four major cities ranged from 5.1 to 7.8 $\mu g/m^3$. The annual mortality attributable to these current long-term $PM_{2.5}$ exposures above background in the four cities is estimated to be equivalent to around 1590 deaths (2.2%) at typical ages.

The report also describes the substantial reductions in the impact on health that could result from reducing air pollution in these cities under three different scenarios. Under Scenario One $PM_{2.5}$ exposures would increase in the four cities so that average annual $PM_{2.5}$ would range from 7.0 to 9.7 $\mu g/m^3$, increasing attributable mortality at typical ages by 48% (equivalent to 760 deaths at typical ages in the first year after the increase) compared to current long-term $PM_{2.5}$ exposures. Under Scenario Three $PM_{2.5}$ exposures would decrease in the four cities so that average annual $PM_{2.5}$ levels would range from 4.7 to 5.9 $\mu g/m^3$, decreasing attributable mortality at typical ages by 34% (equivalent to 533 deaths at typical ages in the first year after the decrease) compared to current long-term $PM_{2.5}$ exposures.

The relationship between attributable deaths and the overall change in population life expectancy was illustrated by an analysis of $PM_{2.5}$ in Sydney. The mortality effects of long-term exposure to human-made $PM_{2.5}$ in Sydney in 2008 (annual average of 6.3 $\mu g/m^3$) is estimated to be equivalent to 522 deaths at typical ages. The years of life lost in 2008 in Sydney is estimated to be 6300 life years. The loss of life expectancy related to long-term exposure to 2008 levels of human-made $PM_{2.5}$ is 72 days for males and 65 days for females.

The Sydney analysis also assessed the impact of reducing annual average $PM_{2.5}$ in Sydney by 1 $\mu g/m^3$ from 2008 annual average $PM_{2.5}$ exposures (equivalent to Scenario Three) over the next 100 years. In the first year of reduced exposure, there would be a mortality effect equivalent to approximately 140 fewer deaths in Sydney compared to a situation with no reduced exposure. In the same year, 70 additional years of life would be lived. After 50 years, the less exposed population has accrued an additional 100,000 years of life. After 100 years, this has increased to approximately 250,000 years of life.

The magnitudes of the effects of air pollution reported here are estimates that come with a range of uncertainties intrinsic to assessments of this kind. The assumptions and methods used here to estimate the health impacts of air pollution are likely to have underestimated the total health benefits of reducing air pollution. While the HRA Report 2013 includes estimates of the health impacts of particulate air pollution on mortality and hospitalisation, it does not include estimates of a large range of less severe health effects also associated with air pollution including cough and other respiratory symptoms, increased medication use, and school or work absence (US EPA 2012). This report focused on PM_{10} and $PM_{2.5}$ pollution to quantify the health effects of exposure to the complex mixture of air pollution present in Australian cities. Other pollutants such as ozone can have independent health effects, or may interact with particulates to enhance their effects on health.

The results reported here show that decreasing air pollution exposures in Australia would lead to substantial health benefits. Research suggests that the acute effects of air pollution on health (e.g. hospitalisation) would be reduced in parallel with reductions in air pollution. Reduction in the health effects of long-term exposure to air pollution (e.g. mortality) would require sustained reductions in air pollution over longer time periods and the health benefits would accrue over a longer time period. A multi-sectoral approach, engaging such relevant sectors as transport, housing, energy production and industry, is needed to develop and effectively implement long-term policies that reduce the risks of air pollution to health (WHO 2013).

8 References

AIHW (2003), *Asthma in Australia*, Australian Centre for Asthma Monitoring, Australian Institute for Health and Welfare, Canberra.

AIHW (2010), *Monitoring the impact of air pollution on asthma in Australia: a methods paper*, Asthma Series, Cat. no. ACM 18, Australian Institute for Health and Welfare, Canberra, viewed 18 December 2012.

Begg, S, Vos, T, Barker, B, Stevenson, C, Stanley, L and Lopez, A (2007), *The burden of disease and injury in Australia 2003*, Cat. no. PHE 82, Australian Institute for Health and Welfare, Canberra.

Brook, RD, Rajagopalan, S, Pope, CA, III, Brook, JR, Bhatnagar, A and Diez-Roux, AV (2010) 'Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association', *Circulation* 121(21):2331–2378.

Burgers, M and Walsh, S (2002), *Exposure Assessment and Risk Characterisation for the Development of a PM2.5 standard, Melbourne*, report prepared for the National Environmental Protection Council Service Corporation, Environment Protection Authority of Victoria, Melbourne.

COMEAP (2009), *Long-Term Exposure to Air Pollution: Effect on Mortality*, a report by the Committee on the Medical Effects of Air Pollutants, Chilton, UK.

COMEAP (2010), *The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom*, a report by the Committee on the Medical Effects of Air Pollutants, Chilton, UK.

Department of the Environment (2013), *Air quality standards*, Australian Department of the Environment, Canberra, updated July 2013, viewed November 2013, at www.environment.gov.au/topics/environment-protection/air-quality/air-quality-standards#air.

European Commission (2005), *ExternE, Externalities of Energy: Methodology 2005 Update*, P Bickel and R Friedrich (eds), European Commission, Luxemburg.

Frangos, J and DiMarco, P (2013), *Draft exposure assessment and risk characterisation to inform recommendations for updating ambient air quality standards for $PM_{2.5}$, PM_{10}, O_3, NO_2, SO_2*, Report number 127643066-001-R-RevA, Golder Associates, Richmond, Victoria.

Jalaludin, B, and Cowie, C (2012), *Health Risk Assessment – Preliminary Work to Identify Concentration Response Functions for Selected Ambient Air Pollutants*, report prepared for EPA Victoria, Respiratory and Environmental Epidemiology, Woolcock Institute of Medical Research, Glebe, 30 June 2012.

Loomis, D, Grosse, Y, Lauby-Secretan, B, El Ghissassi, F, Bouvard, V, Benbrahim-Tallaa, L, Guha, N, Baan, R, Mattock, H and Straif, K, on behalf of the International Agency for Research on Cancer Monograph Working Group IARC, Lyon, France (2013), 'The carcinogenicity of outdoor air pollution', *The Lancet Oncology*, *Early Online Publication*, 24 October 2013 (Article in Press DOI: 10.1016/S1470-2045(13)70487-X).

NEPC (2011), *An Australian Approach to Setting Air Quality Standards in Australia (Part A)*, National Environmental Protection Council, Canberra.

Pope, CA, III and Dockery, DW (2006), 'Health effects of fine particulate air pollution: lines that connect', *Journal of the Air & Waste Management Association* 56(6): 709–742.

US EPA (2010), *Quantitative Health Risk Assessment for Particulate Matter*, Office of Air Quality Planning and Standards, US Environmental Protection Agency, EPA-452/R-10-005, Washington DC, USA.

US EPA (2011a), *Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards*, US Environmental Protection Agency, EPA 452/R-11-003, Washington DC, USA.

US EPA (2011b), *The benefits and costs of the Clean Air act from 1990 to 2020*, US Environmental Protection Agency, Washington DC, USA.

US EPA (2012), *Provisional assessment of recent studies of health effects of particulate matter exposure*, US Environmental Protection Agency, Washington DC, USA.

WHO Europe (2004), *Health aspects of air pollution*, results from the WHO project 'Systematic review of health aspects of air pollution in Europe', World Health Organization, Regional Office for Europe, Copenhagen, Denmark.

WHO (2013) *Review of evidence on health aspects of air pollution – REVIHAAP Project Technical Report*, World Health Organization, Regional Office for Europe, Copenhagen, Denmark.

Appendix 1: Additional Sydney analysis – purpose, methods and descriptive results

The mortality results of the HRA Report 2013 are presented in terms of 'attributable' deaths. When results are presented in this way, it is tempting to think that a reduction in air-pollution levels will 'save' the lives of an identifiable group of people. This is not the case.

We know from epidemiological studies of long-term exposure to $PM_{2.5}$ that people who live in less polluted cities tend, on average, to live longer. However, we do not know how this average increase in length of life is distributed among the population. It is likely that there is variation in effect between individuals. That is, exposure to $PM_{2.5}$ will affect the health of some people more than others.

Epidemiological studies present the observed change in survival time as a *relative risk* of mortality. Calculation of 'attributable' deaths is one way that risk assessors may apply the relative risks from epidemiological studies to alternative populations. The results of this calculation are an aggregation across the whole population of age-specific risks attributable to $PM_{2.5}$. They should not be interpreted to mean that $PM_{2.5}$ only contributes to a specific number of 'attributable' deaths. In reality, $PM_{2.5}$ will likely contribute in some way to the shortening of life of a great many more people. In this section, we attempt to clarify what is meant by the number of 'attributable' deaths by referring to that number, *XX*, as *a mortality effect equivalent to XX deaths at typical ages*. This is discussed in more detail later.

The HRA Report 2013 also presents changes in the number of 'attributable' deaths related to changes in the level of $PM_{2.5}$ exposure. It is tempting to conclude from this that these changes in number of 'attributable' deaths would remain constant in each year that the change in $PM_{2.5}$ exposure persists. Again, this is not the case. For example, the HRA Report 2013 estimates that, in the four Australian cities, 533 'attributable' deaths would be avoided in the first year of Scenario 3. It should not be concluded from this that 5330 'attributable' deaths would be avoided in the first 10 years. In fact, the number is far less. The reasons for this are as follows.

We know that, on average, people live longer if their exposure to $PM_{2.5}$ is reduced. If we assume that a reduction in exposure has an immediate health impact, in the first year after a reduction in $PM_{2.5}$, there will be fewer deaths and more people will survive to the next year, becoming older at the same time. As a result, the population will increase in size and average age; in each subsequent year there will be more people exposed to $PM_{2.5}$ and these people will, on average, be older. So even though the level of $PM_{2.5}$ is reduced and people are living longer, the number of 'attributable' deaths will increase each year due to the increased population size and age. Therefore, while 'attributable' deaths provide an indication of the immediate benefit in the first year after a reduction in $PM_{2.5}$, alternative measures that reflect overall population survival time are required to quantify the long-term benefits. Changes in population survival time may be presented in two ways:

1. the change in life expectancy – the average length of life of an individual in the population of interest
2. the change in the number of years of life lived in the whole population.

To illustrate the relationship between the number of attributable deaths presented in the HRA Report 2013 and alternative metrics that provide information about the overall change in population survival time, additional analyses were conducted for Sydney. For the purposes of describing the concepts of these additional analyses we attempt to answer two questions:

1. What is the effect on mortality of current levels of $PM_{2.5}$ in Sydney?
2. What is the impact of reducing exposure to $PM_{2.5}$ by 1 $\mu g/m^3$ in Sydney (equivalent to Scenario 3 in the HRA Report 2013)?

The impacts of the other HRA Report 2013 scenarios were also assessed and the results presented in the main body of this report.

Question 1: What is the effect on mortality of current levels of $PM_{2.5}$ in Sydney?

To answer this question, we used an estimate of the 2008 Sydney population, the 2008 level of $PM_{2.5}$ exposure and the average NSW age-specific death rates between 2007 and 2009. 2008 was chosen because it was the centre of the five years analysed in the HRA Report 2013 and the $PM_{2.5}$ exposures were typical of $PM_{2.5}$ exposures in Sydney in that timeframe. As with the HRA Report 2013, background $PM_{2.5}$ was assumed to be 2.7 $\mu g/m^3$.

The mortality effect of long-term exposure to human-made $PM_{2.5}$ in Sydney in 2008 (annual average $PM_{2.5}$ of 6.3 $\mu g/m^3$) is estimated to be *equivalent to 522 deaths at typical ages*. To illustrate what is meant by 'equivalent to deaths at typical ages', Figure A1 shows the age distribution of the $PM_{2.5}$ related mortality effect as well as the age distribution of all deaths that occurred in Sydney. It should be noted that the two distributions have a very similar shape. There is no $PM_{2.5}$ related mortality effect shown among people aged less than 30 years because the effect estimate used for risk assessment is derived from a study that was limited to participants aged over 30 years.

Estimation of the number of years of life that were lost in 2008 as a result of long-term exposure to human-made $PM_{2.5}$ is relatively straightforward. Each attributable death is multiplied by the relevant age-specific life expectancy. For example, a 40 year old male has a life expectancy of approximately 42 more years. Therefore, each death of a 40 year old male is responsible for a loss of 42 years of potential life.

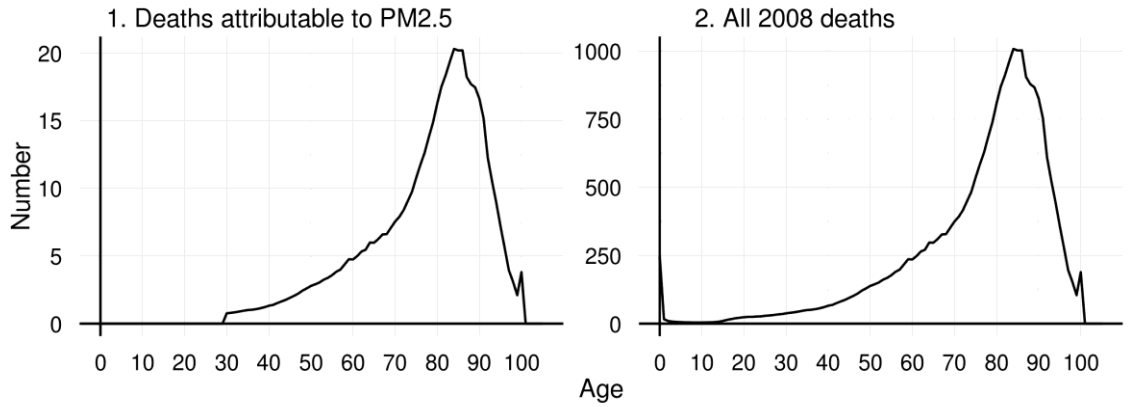

Figure A1: Age distribution of deaths in Sydney (note different y-axis scales)

Approximately 6300 years of life were lost in 2008 in Sydney as a result of exposure to human-made $PM_{2.5}$. Figure A2 shows the age distribution of these years. While the distribution is less peaked than the distribution of attributable deaths, it is still the case that the majority of years of life are lost among people aged 50 years or more.

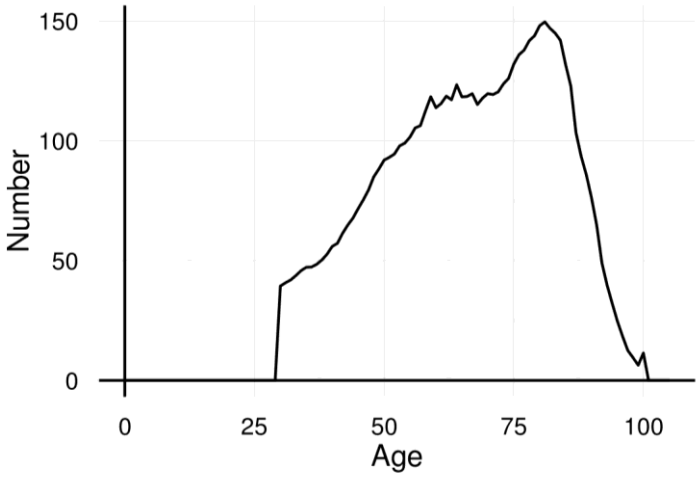

Figure A2: Age distribution of years of life lost in Sydney in 2008 attributable to exposure to human-made $PM_{2.5}$

The loss of life expectancy associated with long-term exposure to 2008 levels of human-made $PM_{2.5}$ is 72 days for males and 65 days for females.

Question 2: What is the impact over time of reducing exposure to $PM_{2.5}$ by 1 $\mu g/m^3$ in Sydney (the HRA Report 2013 Scenario 3)?

This question is related to Question 1, but requires somewhat more complicated calculations. This is because a change in $PM_{2.5}$ levels has an impact on both the size and age distribution of the impacted population.

To answer Question 2, we used methods adapted from those described by COMEAP (COMEAP 2010). As with Question 1, the 2008 Sydney population and $PM_{2.5}$ levels were used, as were the NSW 2007 to 2009 average age-specific mortality rates. A number of simplifying assumptions have been made. Importantly, we have assumed that the birth rate remains constant and there is no migration into or out of the population. We have also assumed that the full health benefit of a reduction in $PM_{2.5}$ exposure occurs immediately.

Figure A3 illustrates the impact of this intervention over the next 105 years. Each line represents the difference between the Sydney population exposed to 1 $\mu g/m^3$ less $PM_{2.5}$ compared to the Sydney population with no change in exposure. The solid line shows the effect on a population where a new birth-cohort is introduced each year. The dashed line illustrates what happens to the cohort of people alive in 2008 only. For the purposes of description, we will focus on the *extended* population (the solid line), that is, the population into which new people are born. The top panel shows the annual difference in number of deaths. The middle panel shows the annual difference in the number of years of life lived. The bottom panel shows the cumulative difference in the number of years of life lived.

In the first year of reduced exposure, there would be approximately 140 fewer deaths in Sydney compared to a situation with no reduced exposure (Figure A3, top panel). In the same year, 70 additional years of life would be lived (Figure A3, middle panel). This is because each of the 140 people who avoided death in that year gain on average six months of life.

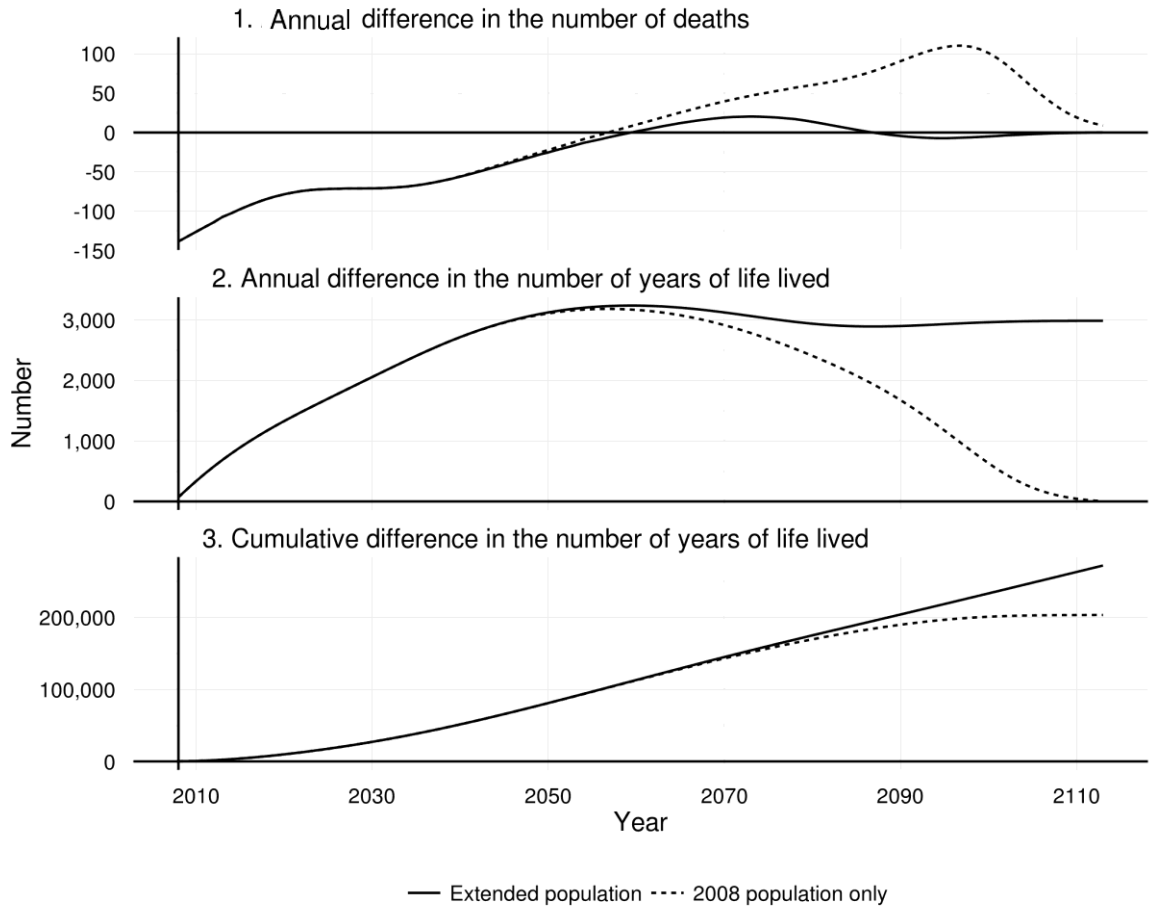

Figure A3: The difference in deaths and life years between the Sydney population with a reduction in $PM_{2.5}$ exposure of 1 µg/m^3 compared to the Sydney population with no change in exposure

Figure A3, top panel shows that the difference in the number of deaths avoided because of reduced $PM_{2.5}$ exposure falls each year. This is because, as people live longer, the less exposed population increases in size and average age. All other things being equal, there will be more deaths in a larger and older population. However, as Figure A3, middle panel shows, the number of years of life lived increases rapidly. Again, this is because the less exposed population is increasing in size. Each additional person alive in the less exposed population contributes a single year of life to the annual total, so a larger population gains more years of life each year.

After approximately 50 years a new equilibrium is reached (although true equilibrium is not achieved until extinction of the entire 2008 cohort in about 2113). At this point, the annual number of deaths in both populations is roughly the same. However, by this time the less exposed population contains approximately 3000 more people and on average these people live longer. In fact, the reduction in exposure to $PM_{2.5}$ results in an increase in life expectancy at birth of approximately 20 days for males and 18 days for females.

Figure A3, bottom panel shows the cumulative total of the number of years of life lived. After 50 years, the less exposed population has accrued an additional 100,000 years of life. After 100 years, this has increased to approximately 250,000 years of life.

Appendix 2: Attributable mortality and hospitalisation due to ozone, nitrogen dioxide and sulfur dioxide

The HRA Report 2013 (Frangos & DiMarco 2012) estimates the health burden and health impacts of particulates ($PM_{2.5}$ and PM_{10}) as well as nitrogen dioxide (NO_2), ozone (O_3) and sulfur dioxide (SO_2). As discussed in the HRA supplementary report, simply adding the attributable cases due to NO_2, O_3 and SO_2 to the attributable cases due to particulates, as a means of capturing the total health burden attributable to all the common air pollutants, would result in double counting and an overestimation of the total health burden.

HRAs generally take a conservative approach and only estimate attributable health effects for one or possibly two 'index' pollutants and for non-overlapping health outcomes. The index pollutant approach still includes the majority of effects of all other correlated pollutants and avoids the issue of double counting. The supplementary report focuses on the attributable mortality and hospitalisation due to particulate exposure. This appendix and Table A1 summarises the HRA Report 2013 results for NO_2, O_3 and SO_2.

Ozone

The health effects of exposure to ambient ozone are summarised in Table 1 of the supplementary report. A recent World Health Organization (WHO) review of the scientific literature since 2005 found (WHO 2013):

- additional supporting evidence that the acute effects of O_3 on health are independent of particulates
- new evidence that long-term exposure to O_3 effects cardiorespiratory mortality
- further evidence that O_3 is associated with the new onset of asthma, asthma severity, hospitalisation for asthma, and reduced lung function
- suggestive new evidence of O_3 effects on cognitive development and reproductive health, including preterm birth.

From 2006 to 2010 the annual median daily maximum 1 hr O_3 exposures varied across the four cities from around 35 ppb in Sydney to around 30 ppb in Melbourne. However, during this same period there was large variation in maximum daily 1 hr O_3 exposures in the four cities from around 110 ppb in Sydney to around 60 ppb in Brisbane.

The large variation in the maximum daily 1 hr O_3 concentrations between the four cities resulted in very different changes in O_3 and associated attributable percentages of cases for each city under the three scenarios. Sydney and Melbourne had similar ozone exposures for Scenario One (100 ppb) and improved (lower) exposures for Scenario Two (85 ppb) and Scenario Three (70 ppb) compared to current exposure. However, Brisbane and Perth had worsening (higher) exposures for Scenario One (100 ppb) and Scenario Two (85 ppb), and similar exposures for Scenario Three (70 ppb) compared to current exposure.

Short-term O_3 exposure – attributable all-cause mortality (non-trauma) all ages

Annual mortality attributable to current short-term O_3 exposure above background is estimated to be equivalent to approximately 2240 deaths or 3.4% of deaths at typical ages (95% confidence interval: 0.7% to 5.8%) over the four cities. The percentage annual deaths ranged from 3.7% in Sydney to 3.0% in Melbourne.

Because of the large variations in maximum daily 1 hr O_3 concentrations between the cities it is not appropriate to calculate an averaged percentage change over the four cities for the scenario exposures. Over the four cities, when compared to current exposures, Scenario One (100 ppb) produced an increase equivalent to approximately 230 deaths at typical ages, Scenario Two (85 ppb) produced a decrease equivalent to approximately 180 deaths at typical ages, while Scenario Three (70 ppb) produced a decrease equivalent to approximately 590 deaths at typical ages.

Short-term O_3 exposure – attributable childhood hospital emergency department attendance

Annual childhood hospital emergency department attendance attributable to current short-term O_3 exposure above background is estimated to be 660 or 3.1% of cases (95% confidence interval: 1.9% to 4.4%) over the four cities. The percentage annual cases ranged from 3.4% in Sydney to 2.8% in Melbourne.

Because of these large variations between the cities it is not appropriate to calculate an averaged percentage change over the four cities for the scenario exposures. Over the four cities, when compared to current exposures, Scenario One (100 ppb) produced an increase of 40 childhood hospital emergency department attendances, Scenario Two (85 ppb) produced a decrease of approximately 80 attendances, while Scenario Three (70 ppb) produced a decrease of approximately 190 attendances.

Nitrogen dioxide

The health effects of exposure to ambient nitrogen dioxide are summarised in Table 1 of the supplementary report. A recent World Health Organization (WHO) review of the scientific literature since 2005 found (WHO 2013):

- NO_2 is associated with increased mortality, hospital admissions and respiratory symptoms
- chamber and toxicological evidence provides some mechanistic support for a causal interpretation of respiratory effects
- sensitivity to NO_2 differs across subjects, i.e. lowest health-relevant concentrations are not the same for all
- NO_2 short-term effects in many studies remain after adjustment for co-pollutants (including PM_{10}, $PM_{2.5}$, black smoke)
- consistent short-term epidemiological evidence and some mechanistic support for causality that NO_2 has some direct acute effects and is an excellent marker of pollutants not characterised by particulates and O_3, such as traffic related pollution.

From 2006 to 2010 the annual median daily maximum 1 hr NO_2 exposures varied across the four cities from around 20 ppb in Melbourne and Sydney to around 15 ppb in Brisbane and Perth. However, during this same period there was large variation in maximum daily 1 hr NO_2 exposures in the four cities from around 60 ppb in Melbourne to around 30 ppb in Brisbane.

The large variation in the maximum daily 1 hr NO_2 concentrations between the four cities resulted in different changes in NO_2 for each city under the three scenarios. However, Scenario One (120 ppb) and Scenario Two (80 ppb) produced worsening (higher) exposures in all four cities. Scenario Three (40 ppb) produced similar exposures in Sydney and Perth, higher exposures in Brisbane and lower exposures in Melbourne.

Short-term NO_2 exposure – attributable all-cause mortality (non-trauma) all ages

Annual mortality attributable to current short-term NO_2 exposure above background is estimated to be equivalent to 2.8% of deaths at typical ages (95% confidence interval: 0.5% to 5.3%) over the four cities. The percentage annual deaths ranged from 3.4% in Melbourne to 2.3% in Brisbane.

Averaged over the four cities, increasing exposure under Scenario One (120 ppb) and Scenario Two (80 ppb) increased estimated equivalent all-cause mortality all ages by around 190% and 90% respectively compared to current exposures. Adjusting NO_2 exposure to Scenario Three (40 ppb) produced a small (10%) decrease in equivalent all-cause mortality compared to current exposures.

Short-term NO_2 exposure – hospital admissions

Averaged over the four cities, annual cardiovascular hospital admissions attributable to current short-term NO_2 exposure above background is estimated to be 4.3% of cardiovascular admissions 65+ years (95% confidence interval: 3.0% to 5.5%), and 2.1% of cardiovascular admissions 15–64 years (95% confidence interval: 0.5% to 3.8%).

Averaged over the four cities, annual respiratory hospital admissions attributable to current short-term NO_2 exposure above background is estimated to be 2.4% of respiratory admissions 65+ years (95% confidence interval: 0.9% to 3.9%), 2.6% of respiratory admissions 15–64 years (95% confidence interval: 0.8% to 4.6%), and 6.7% of respiratory admissions 1–14 years (95% confidence interval: 1.8% to 12.0%).

Averaged over the four cities, increasing exposure under Scenario One (120 ppb) and Scenario Two (80 ppb) increased estimated cardiovascular and respiratory hospital admissions outcomes by around 190% and 90% respectively compared to current exposures. Adjusting NO_2 exposure to Scenario Three (40 ppb) produced a small (10%) decrease in estimated cardiovascular and respiratory hospital admissions outcomes compared to current exposures.

Short-term NO_2 exposure – asthma hospital emergency department attendance

Averaged over the four cities, annual asthma hospital emergency department attendance attributable to current short-term NO_2 exposure above background is estimated to be 6.7% of cases (95% confidence interval: 1.8% to 12.0%).

Averaged over the four cities, increasing exposure under Scenario One (120 ppb) and Scenario Two (80 ppb) increased estimated asthma hospital emergency department attendance by around 190% and 90% respectively compared to current exposures. While adjusting NO_2 exposure to Scenario Three (40 ppb) produced a small (9%) decrease in estimated asthma hospital emergency department attendance compared to current exposures.

Sulfur dioxide

The health effects of exposure to ambient sulfur dioxide are summarised in Table 1 of the supplementary report. A recent World Health Organization (WHO) review of the scientific literature since 2005 (WHO 2013) found little new evidence on the health effects of short-term SO_2 exposures that would lead to a change in current WHO air quality guidelines.

Current short-term exposure to daily maximum 1 hr SO_2 in Sydney, Melbourne and Perth (no data for Brisbane or south east Queensland) is generally well below 100 ppb.

Short-term SO_2 exposure and respiratory hospital admissions

Averaged over Sydney, Melbourne, Brisbane and Perth annual respiratory hospital admissions 65+ years attributable to current short-term daily 1 hr SO_2 exposure above background is estimated to be 2.3% of cases (95% confidence interval: 0.8% to 5.9%).

As current exposure to SO_2 in Sydney, Melbourne and Perth is generally well below 100 ppb Scenario One (200 ppb), Scenario Two (150 ppb) and Scenario Three (100 ppb) all substantially increased attributable respiratory hospital admissions 65+ years compared to current exposures.

Table A1: Attributable mortality and hospitalisation due to current exposure and scenario exposures to ozone, nitrogen dioxide and sulfur dioxide

Pollutant	Time period	Outcome	Scenario	Number of attributable cases % increase/ decrease compared to current	Proportion of attributable cases and increase/ decrease compared to current (95% CI)
Ozone	Short-term	All-cause mortality (non-trauma) all ages	Current	2243 cases	3.4% (0.7%–5.8%)
			Scenario 1 (100 ppb)	+232%	[a]
			Scenario 2 (85 ppb)	-183%	[a]
			Scenario 3 (70 ppb)	-594%	[a]
	Short-term	Asthma hospital emergency department attendance 1–14 years	Current	660 cases	3.1% (1.9%–4.4%)
			Scenario 1 (100 ppb)	+40%	[a]
			Scenario 2 (85 ppb)	-77%	[a]
			Scenario 3 (70 ppb)	-194%	[a]
Nitrogen dioxide	Short-term	All-cause mortality (non-trauma) all ages	Current	[b]	2.8% (0.5%–5.3%)
			Scenario 1 (120 ppb)	[b]	+191%
			Scenario 2 (80 ppb)	[b]	+90%
			Scenario 3 (40 ppb)	[b]	-10%
	Short-term	Cardiovascular hospital admissions 65+ years	Current	[b]	4.3% (3.0%–5.5%)
			Scenario 1 (120 ppb)	[b]	+195%
			Scenario 2 (80 ppb)	[b]	+90%
			Scenario 3 (40 ppb)	[b]	-10%

Nitrogen dioxide, continued	Short-term	Cardiovascular hospital admissions 15–64 years	Current	[b]	2.1% (0.5%–3.8%)
			Scenario 1 (120 ppb)	[b]	+188%
			Scenario 2 (80 ppb)	[b]	+88%
			Scenario 3 (40 ppb)	[b]	-10%
	Short-term	Respiratory hospital admissions 65+ years	Current	[b]	2.4% (0.9%–3.9%)
			Scenario 1 (120 ppb)	[b]	+189%
			Scenario 2 (80 ppb)	[b]	+88%
			Scenario 3 (40 ppb)	[b]	-10%
	Short-term	Respiratory hospital admissions 15–64 years	Current	[b]	2.6% (0.8%–4.6%)
			Scenario 1 (120 ppb)	[b]	+189%
			Scenario 2 (80 ppb)	[b]	+88%
			Scenario 3 (40 ppb)	[b]	-10%
	Short-term	Respiratory hospital admissions 1–14 years	Current	[b]	6.7% (1.8%–12.0%)
			Scenario 1 (120 ppb)	[b]	+203%
			Scenario 2 (80 ppb)	[b]	+92%
			Scenario 3 (40 ppb)	[b]	-10%
	Short-term	Asthma hospital emergency department attendance 1–14 years	Current	[b]	1.7% (0.9%–2.5%)
			Scenario 1 (120 ppb)	[b]	+187%
			Scenario 2 (80 ppb)	[b]	+88%
			Scenario 3 (40 ppb)	[b]	-9%
Sulfur dioxide	Short-term	Respiratory hospital admissions 65+ years	Current	[b]	2.3% (0.8%–5.9%)
			Scenario 1 (200 ppb)	[b]	+741%
			Scenario 2 (150 ppb)	[b]	+504%
			Scenario 3 (100 ppb)	[b]	+286%

[a] Proportion of attributable cases not given due to large variation between the four cities
[b] Number of attributable cases not provided as summary tables in HRA Report 2013