DLA Environmental Services
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Appendix B: Health risk assessment

Document control				
Version	Date	Comment	Prepared by	Reviewed by
1	070/4/2017	Draft final	Lyn Denison	Deanna Tuxford
2	12/03/2018	Revision following client comments	Paul Boulter, Kapil Kulkarni	Damon Roddis
3	30/05/2018	Revision following client comments	Paul Boulter, Kapil Kulkarni	Damon Roddis
4	26/06/2018	Revision following client comments	Paul Boulter, Kapil Kulkarni	Damon Roddis
5 (Final)	06/07/2018	Revision following client comments	Paul Boulter, Kapil Kulkarni	Damon Roddis

Contents

1 Introduction

1.1 Overview

This Health Risk Assessment (HRA) informed the review of the air quality standards for nitrogen dioxide (NO_2), ozone (O_3) and sulfur dioxide (SO_2) in the National Environment Protection (Ambient Air Quality) Measure (AAQ NEPM). The HRA provided supporting health evidence for the review, and was an input to the cost-benefit analysis (CBA). The HRA was conducted broadly in accordance with the methodology for setting air quality standards in Australia NEPC (2011a).

For each pollutant, the main steps in the HRA were as follows:

- A **literature review** of the health evidence for SO_2, NO_2 and O_3. This involved a detailed, up-to-date summary of the literature to inform the HRA methodology.
- An **exposure assessment** to quantify the concentrations of SO_2, NO_2 and O_3 that have historically (2010-2014) been observed in Australian cities and large regional centres, as well as the projected concentrations for the future years of 2021, 2031 and 2040.
- A **risk characterisation**, which involved:
    - Estimating the historical and future attributable cases of various health outcomes due to short-term and long-term exposure to SO_2, NO_2 and O_3. The projected outcomes for future years were determined for scenarios with and without a single package of abatement measures.
    - Determination of the health outcomes avoided by compliance[1] with various proposed air quality standards.

It is worth noting that, as far as possible, pollutant concentrations in this Appendix are referred to in the units of parts per billion (ppb). Occasionally, references are made to mass concentrations (e.g. $\mu g/m^3$). Conversion factors for these are provided at the start of the main Impact Statement.

1.2 Current and proposed air quality standards

The HRA examined both the current AAQ NEPM standards for SO_2, NO_2 and O_3, and a number of proposed (more stringent) alternative standards for potential future implementation. The numerical values of the current and proposed standards for the three pollutants are shown in Tables 1-1, 1-2 and 1-3, with the current standards being highlighted with light blue shading.

The proposed standards were identified through a review of the international literature and regulations, including the WHO Air Quality Guidelines, the United States Environmental Protection Agency (USEPA) National Ambient Air Quality Standards (NAAQS), and the standards that have been adopted in other leading countries[2]. The proposed standards were then endorsed for assessment by the (then) Air Thematic Oversight Group (Air TOG), comprising members from all Australian jurisdictions.

[1] Where reference is made to 'compliance' in this Appendix, unless stated otherwise it refers to the comparison between measured/predicted pollutant concentrations and current/proposed air quality standards. It does not refer to the requirements of the AAQ NEPM, such as monitoring or reporting.

[2] The term 'leading countries' has two meanings in the context of this report: 1) Countries that are viewed internationally as leaders in managing air quality, and 2) Countries that have the most stringent standards.

Table 1-1: Current AAQ NEPM standards and proposed standards for SO_2

Pollutant	Averaging period	Concentration (ppb)	Source
SO_2	1-hour	75	Air TOG
		100	Air TOG
		150	Air TOG
		200	AAQ NEPM
	24-hour	7	Air TOG
		20	Air TOG
		40	Air TOG
		80	AAQ NEPM
	Annual	10	Air TOG
		20	AAQ NEPM

Table 1-2: Current AAQ NEPM standards and proposed standards for NO_2

Pollutant	Averaging period	Concentration (ppb)	Source
NO_2	1-hour	40	Air TOG
		80	Air TOG
		97	Air TOG
		120	AAQ NEPM
	Annual	10	Air TOG
		19	Air TOG
		30	AAQ NEPM

Table 1-3: Current AAQ NEPM standards and proposed standards for O_3

Pollutant	Averaging period	Concentration (ppb)	Source
O_3	1-hour	70	Air TOG
		85	Air TOG
		100	AAQ NEPM
	4-hour	60	Air TOG
		70	Air TOG
		80	AAQ NEPM
	8-hour	47	Air TOG
		55	Air TOG
		60	Air TOG
		70	Air TOG

2 Methodology

2.1 Literature review of the health effects of sulfur dioxide, nitrogen dioxide and ozone

The first step of the HRA was a detailed, up-to-date review of the health effects of SO_2, NO_2 and O_3. This focussed on publications by key agencies, including USEPA, WHO, and the UK Committee Medical Effects of Air Pollution (COMEAP). A literature search was also conducted through PubMed and Medline to identify any additional significant new research. An important objective of the literature review was to identify any recent research undertaken in Australia that could inform the review of the air quality standards in the AAQ NEPM. The main findings of the literature review are presented in Section 3 for SO_2, Section 4 for NO_2 and Section 5 for O_3.

2.2 Exposure assessment

The exposure assessment quantified the concentrations of SO_2, NO_2 and O_3 that have historically (2010-2014) been experienced in Australian cities and large regional centres, and the estimated concentrations for the future years 2021, 2031 and 2040. The work concentrated mainly on NSW (Sydney, Wollongong and Newcastle) and Victoria (Melbourne and Latrobe Valley), as detailed air quality modelling could be conducted for these airsheds. For the modelled concentration projections (see Appendix A – air quality study), the health risk was assessed both with, and without, abatement measures. In addition, the numbers of health outcomes avoided if the proposed standards could be met were determined by 'rolling back' the daily and annual concentrations to correspond to the standards. This was consistent with the NEPC standard-setting methodology (NEPC, 2011b).

To assess the exposure of the population to historical levels of SO_2, NO_2 and O_3, ambient air quality monitoring data collected by the jurisdictional environment agencies were used. All data were obtained from monitoring stations that were established to meet the requirements of the AAQ NEPM, so as to provide concentrations that were generally representative of population exposure. These stations are listed in Annexure A of Appendix A (air quality study). No monitoring data were available for Tasmania, as the pollutants being considered in this review are not routinely monitored in Tasmania. In most jurisdictions NO_2 and O_3 are not monitored in regional centres. SO_2 data are collected in some regional centres with large industrial sources. As the main influence in these areas was usually a single industrial source, which was subject to jurisdictional air quality management and licensing requirements, the areas were considered not to be the focus of the AAQ NEPM and were therefore excluded from the assessment.

To assess population exposure and changes in short-term concentrations (1-hour, 4-hour or 8-hour maximum, or 24-hour average), the measurements for all relevant monitoring stations across the network in each airshed were averaged to obtain values that were representative of the total population within the airshed. This was conducted for each day of the year. Annual average values were calculated in a similar manner, with the annual average for each site within the available dataset being averaged across all sites to give a network average value. This approach was consistent with the NEPC standard-setting methodology (NEPC, 2011b). It should be noted that this method could lead to an overestimate of the risk to the population in airsheds where some of the SO_2 monitoring stations are in locations that are influenced by emissions from industrial sources.

The concentration-response functions (CRFs) used in this HRA have been derived from population-based epidemiological studies. As recommended by NEPC, the approach used in the HRA was consistent with that used to assess exposure in the epidemiological studies from which the CRFs

used in the risk characterisation have been derived. Epidemiological studies conducted in Australia and internationally average the monitoring data across the available air pollution monitors to obtain measures of the exposure of the population as a whole.

For future projections under different emission scenarios, air dispersion modelling was conducted. The emission scenarios and modelling approaches are described in detail in Appendix A (air quality study). Two main scenarios were assessed:

- A '**Business-as-Usual**' (BAU) scenario, which included actions already agreed to be implemented that may lead to emission reductions, and therefore improvements in air quality, and projected out to 2040.
- An '**Abatement Package**' scenario, which included a single package of potential emission-reduction measures that could lead to improvements in air quality, again projected out to 2040.

Air dispersion modelling was only conducted for NSW (Sydney, Wollongong and Newcastle) and Victoria (Melbourne and Latrobe Valley), as these jurisdictions had the most comprehensive emission inventories to support the modelling. The air quality model predictions for the two scenarios in 2021, 2031 and 2040 were extracted for the same locations as the monitoring stations. The resulting health outcomes for these locations were assessed for each scenario. The assumptions and modelling methods for projected air quality are provided in Chapter 2 of the Impact Statement and Appendix A (air quality study).

Golder Associates has previously provided estimates of background concentrations of NO_2 and O_3 in Australian cities (Frangos and DiMarco, 2013). No estimates were provided for SO_2 background concentrations, as it was considered that all the sources of SO_2 in Australian airsheds are anthropogenic. For consistency with the Golder Associates analysis, and in the absence of alternative data, the background concentrations for NO_2 (2.1 ppb) and O_3 (8.7 ppb) cited in their report were used for the purposes of this risk assessment. Further discussion on the selection of background data is provided in the Golder Associates report. These values were subtracted from the network-average concentrations to provide values that were representative of anthropogenic sources of NO_2 and O_3 in Australian airsheds.

2.3 Risk characterisation

2.3.1 Overview

The risk characterisation estimated the number of attributable cases for each health outcome due to air pollution. The number of health outcomes avoided – if the alternative standards could be met – were calculated relative to historical air quality (2010 to 2014) and relative to the BAU future projections (2021, 2031 and 2040).

The approach used was again consistent with the NEPC methodology. Air quality and health data for 2015 and 2016 were not available at the time that the review commenced, and were not included in the HRA. However, the air quality data for these years were included in the air quality study (Appendix A).

Health outcomes avoided were calculated using the general approach used in previous studies, such as the one by Golder Associates (Frangos and DiMarco, 2013). That is, the change in health outcomes (denoted Δy) is expressed as a function of:

- The change in pollutant concentrations (denoted Δx)

- A baseline incidence rate (denoted y_0)
- A concentration-response function (CRF), which defines the increase in a health outcome per unit increase in pollutant concentration

The general equation is provided below:

$$\Delta y = f(\Delta x, y_0, CRF)$$

Golder Associates expressed the CRF as a 'beta' (β) coefficient, which results in the following equation (Frangos and DiMarco, 2013, p.9):

$$\Delta y = \left(e^{\beta \Delta x} - 1\right) y_o$$

In this Appendix, the CRF is expressed as a relative risk (RR), which is the form normally used by the source literature to express CRFs[3]. The relationship between RR and β is presented in the equation below (Golder Associates, 2013, p.12):

$$\beta = {\log RR}/{\Delta x}$$

This results in the use of the following equation, which is a transformed version of the Golder Associates equation:

$$\Delta y = \Delta x . (RR - 1) . y_o$$

A sensitivity analysis was conducted using the 95% confidence limits of the various CRFs to provide not only a central estimate but also the upper- and lower-bound estimates of the attributable health outcomes.

2.3.2 Concentration-response functions

The NEPC (2011b) standard-setting methodology recommends that CRFs should be selected:

- Following the collation and review of relevant studies considered to be of acceptable quality.
- By giving priority to studies that are based on the same exposure metrics as that used in the study for which the risk characterisation is required.
- By considering the characteristics of the study population (e.g. socio-economic factors, susceptibility of subgroups etc.) that influence exposure effects, and how these differ from the population of the studies being reviewed.

NEPC also recommends that results of individual studies may only be combined following formal meta-analytic methods, pooled analyses or expert judgment.

A review by Australian epidemiologists prepared for EPA Victoria, entitled *Health Risk Assessment – Preliminary Work to Identify Concentration-Response Functions for Selected Ambient Air Pollutants,* identified the CRFs to be used in the setting of air quality standards in Australia (Jalaludin and Cowie, 2012). Their report identified the range of health endpoints having CRFs that are robust enough to be

[3] The RR is provided by the source literature for a corresponding change in concentration (Δx). In this study, the RR is 'normalised' into a per unit change (e.g. relative risk per ppb).

used in the quantification of the health effects attributable to air pollution. However, two recent international projects coordinated by the World Health Organization (WHO) Regional Office for Europe have significantly updated the health evidence and the basis for assessment:

- *Review of evidence on health aspects of air pollution* (REVIHAAP) (WHO, 2013a)
- *Health risks of air pollution in Europe* (HRAPIE) (WHO, 2013b)

The REVIHAAP and HRAPIE projects addressed a list of key policy-relevant questions on the health aspects of air pollution, and provided advice based on a review of the latest scientific evidence. The HRAPIE project defined the pollutants, health outcomes and CRFs for health impact assessments. For NO_2 and O_3 the HRAPIE CRFs were considered to be the most robust available in the literature, and incorporated more extensive and more up-to-date information than the Australian CRFs.

Based on the considerations above, three groups of CRFs were defined for used in the HRA, and these are summarised below. Further details are provided in the pollutant-specific sections (3, 4 and 5) of this Appendix.

- **Group 1 CRFs**. This group included the recommendations from the HRAPIE project for NO_2 and O_3, and drew upon various studies for SO_2.
    - For NO_2 and O_3 the CRFs from HRAPIE were used in preference to the ones developed previously for Australia. In line with the recommendation by WHO (2013b), the Group 1 CRFs applied the HRAPIE cut-off[4] for annual average NO_2 of 20 µg/m^3, and the cut-off for daily maximum 8-hour O_3 of 35 ppb. This is because the shape of the CRF curves below these cut-off values is highly uncertain.
    - For SO_2 the CRFs for 1-hour and 24-hour exposures were taken from various sources, as recommended by Jalaludin and Cowie (2012). No studies investigating the long-term effects of exposure to SO_2 on health were identified.
- **Group 2 CRFs**. This group used the same CRFs as Group 1. However the 20 µg/m^3 and 35 ppb cut-offs for mortality from long-term NO_2 and short-term O_3 exposure were not applied. The rationale for excluding the cut-offs was that, while the shape of the CRFs below these levels is unclear, this does not necessarily mean that no health outcomes can be assumed. The data used to derive the CRFs recommended by WHO (2013b) were from Europe, where concentrations in major airsheds rarely exceed these levels. However, concentrations of these pollutants in major Australian airsheds are different to those in Europe. In particular, NO_2 concentrations in Sydney and Melbourne are below those in many of the major European cities.
- **Group 3 CRFs**. This group included only the Australia-specific CRFs. The CRFs for NO_2 and O_3 were taken from Jalaludin and Cowie (2012). The SO_2 CRFs were the same as those in Group 1.

NB: The HRA was based on the Group 1 CRFs. The Group 2 and Group 3 CRFs were included in the work to examine the sensitivity of the HRA (and CBA) results to CRF selection. The specific differences between the Group 1 and Group 3 CRFs are detailed in the assessment section for each pollutant.

[4] In this report the term 'cut-off' is used where a lower limit is applied to a CRFs because there are insufficient data to characterise the CRF at lower concentrations. It should be noted that this is not the same as the threshold for the health effects of air pollution.

In addition, for each CRF the upper and lower 95% confidence limits have been used in addition to the central estimate to provide a range of potential health effects.

2.3.3 Baseline health and population data

To calculate the attributable health outcomes, baseline health data were required. The baseline mortality data (rate/100,000 population) were obtained from ABS for 2010, 2011, 2012, 2013 and 2014, and for all airsheds assessed in the HRA. Hospital admissions data and emergency department data for each outcome and age group considered were obtained from the relevant State/Territory Health Departments. Data for 2014 were not available for NSW at the time the assessment began, so the 2013 data were used to calculate the attributable health outcomes for 2014. For all future projections, the 2014 baseline health data (the most recent data) were used.

The population data used in the risk characterisation were obtained from the most recent ABS estimates for 2010-2014, as well as for the projections out to 2040.

2.3.4 Other aspects of calculations

To calculate the number of attributable cases, the risk per 100,000 people was multiplied by the population obtained from ABS (divided by 100,000). For short-term effects associated with daily changes in each pollutant, the number of cases for each day of the year was calculated, and the results were summed over all days to give the annual total. For the assessment of long-term mortality for NO_2, the annual average concentrations were used in the calculations. For O_3 and NO_2 the pollutant concentration was adjusted for background concentrations, as described in Section 2.2.

For the future projections, estimates of attributable and avoided outcomes were determined for each of the modelled years. The attributable (and avoided) health outcomes for existing air quality are reported as the total for each year. It is important to note that the air dispersion modelling assumed a gradual uptake of the abatement measures from 2021 to 2040, with minimal uptake in 2021 and maximum uptake in 2040. Therefore, the benefits achieved by the implementation of these measures differed for each of the years considered. The assumptions around the uptake of the abatement measures are described in Appendix A (air quality study).

NB: Attributable health outcomes have been calculated independently for each pollutant. These attributable health outcomes for the different pollutants are not additive.

3 Health risk assessment for sulfur dioxide

3.1 Review of health effects

In recent years the health effects of exposure to SO_2 in ambient air have been well studied and reviewed by agencies such as USEPA (2008), WHO (2006) and California EPA (OEHHA, 2000, 2011). The findings of recent studies have strengthened the evidence that the main health effects associated with SO_2 are short-term effects on the respiratory system.

3.1.1 International studies

3.1.1.1 Short-term effects

Population-based epidemiological studies have reported a link between short-term SO_2 exposure and daily mortality, respiratory effects, and cardiovascular effects. The associations persist when other pollutants, such as particles, are controlled for. The epidemiological evidence is supported by controlled human exposure studies and animal toxicology studies. The strongest evidence comes from controlled human exposure studies examining short-term exposure to SO_2 and respiratory effects. These studies exposed volunteers to SO_2 for periods ranging from 5–10 minutes up to one hour. Adverse effects, such as sneezing or shortness of breath, occurred within the first few minutes after inhalation and were not changed by further exposure. The effects were greater when the person was exercising and were most pronounced in people with asthma and other respiratory conditions such as chronic obstructive pulmonary disease (COPD), and particularly in exercising asthmatics.

The REVIHHAP study (WHO, 2013a) found that there is no new evidence since the WHO (2006) review regarding the adverse health outcomes of SO_2 linked to 10 minute exposures. There is some new evidence for increases in hospital admissions for asthma associated with 24-hour SO_2 concentrations. The associations that have been observed with all-cause[5], cardiovascular, respiratory and cardiac mortality, and short-term exposures are suggestive of a causal relationship between SO_2 and these outcomes.

There is also new evidence which strengthens previous findings of hospital admissions for respiratory disease and exposure to short-term SO_2 (WHO, 2013a). Strong associations have been observed for increases in hospital admissions and emergency department attendances for children with asthma and short-term SO_2 exposures. Associations have also been observed for increases in hospital admissions for cardiovascular disease in adults and exposure to SO_2.

The epidemiological evidence, supported by controlled human exposure studies and a limited number of animal toxicological studies conducted at near-ambient concentrations, indicate an association between short-term exposure to SO_2 and several measures of respiratory health, including respiratory symptoms, inflammation, and airway hyper-responsiveness (USEPA, 2008; WHO, 2013a).

The epidemiological evidence further indicates that the SO_2-related respiratory effects (≥ 1-hour, generally 24-hour average) are more pronounced in asthmatic children and older adults (65+ years). In the limited number of studies that examined potential confounding by co-pollutants through multi-pollutant models, the SO_2 effect was generally found to be robust after adjusting for particles and other co-pollutants (USEPA, 2008).

[5] 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

A large number of epidemiological studies in cities in various parts of the world, including the United States, Canada and Europe, have reported associations between exposure to ambient levels of SO_2 and increases in all-cause (non-accidental) and respiratory and cardiovascular mortality, often at 24-hour average levels of less than 10 ppb (Biggeri et al., 2005; Samet et al., 2000a,b; Dominici et al., 2003a,b; Burnett et al., 1998a, 2000, 2004; Katsouyanni et al., 1997, 2003; Samoli et al., 2001, 2003; USEPA, 2008; Stieb et al. 2002, 2003). The mortality effect estimates from the multi-pollutant models in the multi-city studies suggest some confounding between SO_2 and particles and/or NO_2 (USEPA, 2008).

A large number of epidemiological studies generally report consistent and robust associations between ambient SO_2 concentrations and emergency department visits and hospitalisations for all respiratory causes, particularly among children and older adults (65+ years), and for asthma and COPD (USEPA, 2008). Some studies report greater increases in emergency department visits and hospitalisations with season (Schouten et al., 1996; Spix et al., 1998; Wong et al., 1998) and others found the associations, with similar increases in SO_2, to be greater in winter (Castellsague et al., 1995; Tenias et al., 1998; Wong et al., 2002a,b; Vigotti et al., 1996; Walters et al., 1994). Warmer months were more likely to show evidence of an association with adverse respiratory outcomes in children, while older adults appeared to be more likely to be affected during the cooler months.

Intervention studies provide further evidence of an association between SO_2 and respiratory morbidity (USEPA, 2008). The Hong Kong intervention study compared the effects of reducing SO_2 (up to 80% in polluted districts) and sulfate (38% in polluted districts) levels on bronchial responsiveness in primary school children living in two districts (polluted and less polluted). The authors found a greater decrease in bronchial hyper-reactivity and bronchial reactivity in schoolchildren in the polluted district than in the less polluted district (Wong et al. 1998). Another study reported a significant decline in symptoms of cough, sore throat, phlegm, and wheezing in children from the polluted district compared with the unpolluted district in Hong Kong (Peters et al. 1996).

The strongest evidence for a causal relationship between respiratory morbidity and short-term exposure to SO_2 comes from human clinical studies reporting respiratory symptoms and decreased lung function following peak exposures to SO_2 of 5–10-minute duration (WHO, 2006). The exact duration is not critical, however, because responses occur very rapidly, within the first few minutes from commencement of inhalation; continuing the exposure further does not increase the effects. These effects have been observed consistently across studies involving mild to moderate asthmatics during exercise. Statistically significant decrements in lung function accompanied by respiratory symptoms, including wheeze, chest tightness and shortness of breath, have been clearly demonstrated following exposure to 0.4–0.6 ppm SO_2.

Although studies have not reported statistically significant respiratory effects following exposure to 0.2–0.3 ppm SO_2, some asthmatic subjects (5–30%) have been shown to experience moderate to large decrements in lung function at these concentrations (WHO, 2006). Such effects are enhanced by exercise, which increases the volume of air inspired, thereby allowing SO_2 to penetrate further into the respiratory tract. An acute effect of short-term exposure at rest to 0.2 ppm SO_2 is a change in heart rate variability, in which normal young adults responded with small but statistically significant increases in both high and low frequency power, while asthmatic subjects responded with decreases in these parameters of comparable magnitude. A wide range of sensitivity has been demonstrated, both among normal individuals and among those with asthma, who form the most sensitive group for pulmonary function changes. Continuous exposure–response relationships, without any clearly defined threshold, are evident (WHO, 2006).

From the information published to date, the overall conclusion is that the minimum concentration evoking changes in lung function in exercising asthmatics is of the order of 400 ppb, although there is

one example of small changes in airway resistance in two sensitive subjects at 100 ppb (WHO, 2006). In evaluating this further, judgements are required regarding the clinical significance of such effects, the extent to which particularly sensitive subjects have been represented in the studies, the practical relevance of the enforced exercise required to enhance the effects, and how to relate the short (10- to 15 minute) exposures to the more usual hourly average monitoring data (WHO, 2006).

Epidemiological studies have examined the association between air pollution and cardiovascular effects, including increased heart rate, reduced heart rate variability (HRV), incidence of ventricular arrhythmias, changes in blood pressure, incidence of myocardial infarctions (MI), and emergency department visits and hospitalisations due to cardiovascular causes. The epidemiologic evidence from studies of the effect of SO_2 on implantable cardioverter-defibrillator (ICD) recorded arrhythmias, blood pressure and blood markers of cardiovascular risk failed to provide consistent evidence to suggest a role for SO_2 in cardiovascular disease development (USEPA, 2008).

Although biologically plausible modes of action that could explain short-term SO_2 effects on the cardiovascular system have been identified, consideration of these modes of action in light of findings from additional animal toxicological, human clinical and epidemiological studies led the USEPA to the conclusion that the evidence as a whole is inadequate to infer a causal relationship (USEPA, 2008). Specifically, evidence from human clinical and epidemiological studies of HRV in healthy persons as well as persons with asthma or cardiovascular disease was inconsistent and did not support an effect of SO_2 on the autonomic nervous system, despite some positive findings.

Several studies have observed positive associations between ambient SO_2 concentrations and emergency department visits or hospital admissions for cardiovascular diseases (e.g. all cardiovascular diseases, cardiac diseases and cerebrovascular diseases), particularly among individuals of 65+ years of age. However, the results are not consistent across studies. The strongest evidence comes from a large multi-city study conducted in Spain (Ballester et al., 2006) that observed statistically significant positive associations between ambient SO_2 and cardiovascular disease admissions, although the SO_2 effect was found to diminish by half with PM_{10} and CO adjustment.

3.1.1.2 Long-term effects

Epidemiological evidence on the effect of long-term exposure to SO_2 on mortality is limited and, according to the USEPA (2008), is inadequate to infer a causal relationship. Overall, a reanalysis of results from two major US epidemiological studies (Pope et al. 1995; Dockery et al., 1993) observed an association between long-term exposure to SO_2 or sulfur-containing particulate matter (PM) and mortality (Pope et al. 2002; Burnett et al., 2000; Jerrett et al., 2003; Elliott et al. 2007). However, several other US and European cohort studies did not observe an association (Abbey et al. 1999; Lipfert et al. 2000a,b; Nafstad et al. 2004; Filleul et al. 2005; Beelen et al. 2008a,b). The lack of consistency across studies, the inability to distinguish potential confounding by co-pollutants (especially PM), and uncertainties regarding the geographic scale of analysis, limit the interpretation of a causal relationship (USEPA, 2008).

Studies identified by the USEPA (2008) which examined the effects of long-term exposure to SO_2 on asthma, bronchitis, and respiratory symptoms observed positive associations in children. In the limited number of studies examining the SO_2 associations with lung function, the results were generally mixed. The USEPA (2008) concluded in its review that the overall epidemiological evidence on the respiratory effects of long-term exposure to SO_2 is inadequate to infer a causal relationship. The available toxicological and epidemiological evidence on the effect of long-term exposure to SO_2 on cardiovascular health is also too limited to make any conclusions.

A number of studies have reported associations between exposure to SO_2 and low birth weight and premature birth (Sram et al., 2005; Dugandzic et al., 2006). A Canadian study found that first trimester

exposures in the highest quartile for SO_2 and PM_{10} suggested an increased risk of delivering a low-birth-weight infant (Dugandzic et al., 2006). In Korea, Leem et al. (2006) also found an association between low birth weight and low levels of air pollutants, including SO_2. In the USA, a time-series study undertaken by Sagiv et al. (2005) found evidence of an increase in pre-term birth risk with exposure to PM_{10} and SO_2 which were consistent with prior investigations of spatial contrasts. However, toxicological studies provide very little biological plausibility for the effects. The USEPA found that limited number of studies, inconsistent results across trimesters of pregnancy, and the lack of evidence regarding confounding by co-pollutants limits the interpretation of these studies and makes it difficult to draw conclusions regarding the effect of SO_2 on birth outcomes.

In 2011 the Californian Office of Environmental Health Hazard Assessment (OEHHA) evaluated the reproductive and developmental effects of SO_2 and found that exposure to SO_2 has effects on both male and female reproductive health as well as developmental toxicity including pre-term birth, offspring growth, pregnancy loss and congenital malformations. There are few studies of the relationship between SO_2 exposure and female reproductive toxicity that have been conducted. One retrospective cohort study of females undergoing the first cycle of in vitro fertilisation found that SO_2 concentrations were consistently, though not statistically significantly, associated with decreased odds of live births. Associations with other pollutants were stronger. The potential female reproductive toxicity of SO_2 via inhalation in rats was also investigated. The authors reported effects on oestrous cycle length, pregnancy frequency and duration and offspring growth (OEHHA, 2011). Although these effects have been identified, no CRFs for these outcomes are available at this time.

Numerous studies in humans and animals provide evidence that exposure to episodes of relatively high air pollution causes male reproductive toxicity manifested as adverse effects on semen quality, sperm chromatin integrity and biochemical parameters in the testis (OEHHA, 2011). In addition to direct evidence of male reproductive toxicity, the studies on semen quality and sperm chromatin integrity also provide mechanistic evidence consistent with decreased time to conception for males. Exposure to SO_2 is associated with increased DNA damage which occurs in sperm cells of humans and animals exposed to SO_2. Three occupational studies reporting an association between exposure to SO_2 and increased DNA damage in lymphocytes of workers provide supporting evidence.

Human epidemiological studies examining the association between SO_2 exposure and pre-term births reported a statistically significant higher risk of preterm birth associated with exposure to SO_2 (USEPA, 2008; OEHHA, 2011). Studies with higher levels of exposure were more likely to report significantly increased risk of pre-term birth. A significant dose-response relationship was observed between SO_2 and total suspended particulates (TSP) and gestational age in a prospective cohort study that examined the highest exposure levels. In this study, the gestation age distribution curve was more skewed toward the left (shorter gestation) on high pollution days, suggesting that pregnancies at high risk for pre-term delivery may be particularly susceptible to adverse effects of air pollution.

Epidemiological studies that evaluated foetal growth in association with SO_2 exposures reported that ambient SO_2 exposure was statistically significantly associated with indicators of foetal growth restriction or increased risk of low birth weight. OEHHA (2011) noted that two studies reported mixed findings for foetal growth, i.e., SO_2 exposure was significantly associated with indicators of both reduced and increased birth weight, depending on the exposure levels, exposure periods, and populations. Two studies reported SO_2 was associated with decreased risk of low birth weight or increased birth weight.

Yang M and Chou S-Y (2017) found that the closure of a coal-fired power plant in New Jersey reduced the likelihood of having a low birth weight baby and having a preterm birth by 15 percent and 28 percent, respectively, in areas downwind of the power plant. However, this study was based on limited data, and this association has not yet been confirmed by WHO or USEPA.

Overall, the majority of studies that had measured SO_2 concentrations well above limits of detection found that SO_2 was associated with increased risk of low birth weight or other measures of foetal growth restriction. The studies found different susceptible exposure periods. The role of co-pollutants in relation to the potential effects of SO_2 on birth weight is unclear, but several well-conducted studies suggest that SO_2 is associated with reduced birth weight, independent of co-pollutants.

3.1.2 Australian studies

3.1.2.1 Short-term effects

In an Australian study, Jalaludin et al. (2006) reported a 3% excess risk in cardiovascular disease hospital admissions per 0.75 ppb incremental change in 24-hour average SO_2 in single-pollutant models, which was nullified when CO was included.

In a case-crossover study of air pollution and child respiratory health undertaken in five Australian and two New Zealand cities, Barnett et al. (2005) found a statistically significant increase in hospital admissions and SO_2 for 1-hour SO_2. The ambient levels recorded during the study for 1-hour SO_2 ranged from 3.7 to 10.1 ppb, and for the 24 hour mean form 0.9 to 4.3 ppb. In the 1–4 year age group there was evidence of seasonal impacts on pneumonia and acute bronchitis admissions for SO_2 with higher effects observed in the warmer months (Barnett et al., 2005).

A study of 123,840 births of over 20 weeks' gestation in Sydney, between 1998 and 2000, found that 4.9% of babies were born at less than 37 weeks gestation. The mean of the 1-hour maximum SO_2 levels was 3.6 ppb. SO_2 levels in early pregnancy had a large adverse impact on gestational age in those infants conceived in autumn and winter for a 1 ppb increase in SO_2. The authors noted that SO_2 appears to be an important pollutant, despite SO_2 levels in Sydney being well below the national standard, with vehicular traffic being the primary source and it is conceivable that SO_2 is a marker for traffic related air pollutants in the study (Jalaludin et al., 2007).

3.1.2.2 Long-term effects

The results of studies examining the association between long-term exposure to SO_2 and respiratory morbidity are generally inconsistent. Cross-sectional studies conducted in New South Wales in the Hunter and Illawarra regions found no association between annual average levels of SO_2 and prevalence of asthma in children (Henry et al, 1991) and chest colds and respiratory symptoms such as cough and wheeze (Lewis et al, 1998).

3.2 Exposure assessment

To assess the exposure of the population to recent historical levels of SO_2, ambient air monitoring data obtained by the jurisdictional environment agencies between 2010 and 2014 were analysed. A summary of the data used for each airshed is shown in Table 3-1. In general, the data available for SO_2 were more limited than the data for the other pollutants, and no data were available for Tasmania or the ACT. Moreover, some of the SO_2 monitoring stations are in locations that are influenced by emissions from industrial sources, and this may lead to an overestimate of the risk to the population in the corresponding airsheds. For example, this is particularly the case in the Melbourne airshed where estimated health outcomes are significantly above those of other pollutants and this is not considered to be reflective of current SO_2 levels in this airshed, which are well below AAQ NEPM standards.

To assess exposure to daily 1-hour maximum and daily average concentrations, the values for all sites were averaged across the network to obtain a value that was representative of total population within the airshed. This was done for each day of the year.

Table 3-1: Summary of monitoring data used in exposure assessment for SO_2

Location	Period covered	Number of monitoring locations
NSW: Sydney	2010 - 2014	8
NSW: Newcastle	2010 - 2014	2
NSW: Wollongong	2010 - 2014	3
VIC: Melbourne	2010 - 2014	3
VIC: Latrobe Valley	2010 - 2014	1
QLD: Brisbane (SEQ)	2010 - 2014	2
SA: Adelaide	2010 - 2014	1
WA: Perth	2010 - 2014	3
NT: Darwin	2011 - 2014	2 (2012-13), 1 (2011-14)

3.3 Risk characterisation

3.3.1 Concentration-response functions

The results of epidemiological studies have shown that a wide range of health effects are associated with exposure to SO_2. Australian studies (NEPC, 2011a; EPHC 2005) have found associations between SO_2 levels currently experienced in Australian cities and the following health outcomes:

- Increases in daily mortality
- Increases in hospital admissions for:
    - Respiratory disease
    - Cardiovascular disease
- Increases in emergency room attendances for asthma

The health outcomes for which CRFs were recommended by Jalaludin and Cowie (2012) have been assessed in this health risk assessment for the relevant age groups. The groups that were identified as being susceptible to the effects of SO_2 are:

- The elderly
- People with existing cardiovascular and respiratory disease
- People with asthma
- Children

The CRFs for SO_2 are presented in Table 3-2. These were taken from various sources, as recommended by Jalaludin and Cowie (2012). The SO_2 CRF for hospital admission for respiratory disease was taken from an Australian multicity meta-analysis (Simpson et al., 2005a,b). The CRF for daily all-cause mortality for SO_2 was taken from the large meta-analysis conducted by Anderson et al. (2007) for the Department of Health in the UK. The meta-analysis was based on all peer-reviewed papers with quantitative results from time series and panel studies ambient air pollution published up to 2006, and was based on 144 estimates. This study covered a number of cities and provides a robust exposure-response function in the absence of Australian data. A study by Jalaludin et al. (2008) examining the association between emergency department attendances for asthma in children

conducted in Sydney has been used as the basis for the SO_2 CRF for this outcome. No studies investigating the long-term effects of exposure to SO_2 on health were identified.

Table 3-2: Health outcomes and CRFs for SO_2 (Group 1)

Averaging period/statistic	Health outcome (age group)	Group 1 CRFs	
		CRF[a, b]	Source
1-hour maximum	Hospital admissions for respiratory disease (65+ years)	0.52 (0.19 - 0.87)	Simpson et al. (2005a,b)
24-hour average	Daily all-cause[c] mortality (all ages)	0.17 (0.13 - 0.20)	Anderson et al. (2007)
24-hour average	Emergency department visits for asthma (<15 years)	2.00 (0.88 - 3.00)	Jalaludin et al. (2008)

(a) Percentage increase in health outcome per 1 ppb increase in SO_2 concentration
(b) Central estimate and 95% confidence interval. The range of these uncertainties have been derived directly from the source studies and reflects the uncertainty in the statistical analysis of the epidemiological data.
(c) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

The air quality data were combined with the Group 1 CRFs in Table 3-2 and baseline health statistics for each location to calculate the number of attributable health outcomes due to SO_2.

3.3.2 Health outcomes for Business-as-Usual and Abatement Package scenarios

The estimates for the number of historical and projected attributable health outcomes due to SO_2 in the modelled airsheds (NSW and Victoria) are shown in Table 3-3. The projected outcomes are presented for both the BAU and Abatement Package scenarios, and the health outcomes avoided in the latter are also stated[6]. Table 3-4 gives the historical results for the non-modelled airsheds.

In Sydney and Melbourne the health burden is projected to increase substantially in the future under the BAU scenario. However, future emissions and concentrations of SO_2 from industry[7] (and hence the attributable and avoided health outcomes) are likely to be overestimated in the BAU scenario.

In the Abatement Package scenario there are marked reductions in the incidence of the health outcomes, such that by 2040 all-cause mortality and respiratory illness due to SO_2 are generally approaching the 2010-2014 average, with the exception of Melbourne where they are predicted to still be significantly higher. For Melbourne the results indicate that the Abatement Package would not lead to sufficient reductions in SO_2 levels to offset the predicted increase in emissions in future years.

[6] Where the health outcomes avoided do not exactly equal the outcomes in the BAU scenario minus the outcomes in the Abatement Package scenario, this is due to rounding.

[7] Several industrial sources are scheduled to close prior to 2040, including old, coal-fired power stations in NSW and Victoria. These closures were not all included in the projections. The main effect of this will be that SO_2 emissions and concentrations from industry in the BAU scenario, and the effects of any associated abatement measures, are overestimated in the Impact Statement.

Table 3-3: Historical and projected health burden attributable to SO_2 in Australian airsheds (NSW and Victoria airsheds)

Airshed	Annual average 2010-2014	Scenario	Number of attributable health outcomes 2021	2031	2040	Health outcomes avoided 2021	2031	2040
Daily mortality, all-cause[(a)]								
NSW: Sydney	19	*BAU*	38	47	59			
		Abatement Package	32	29	18	6	18	40
NSW: Newcastle	6	*BAU*	17	21	25			
		Abatement Package	14	11	5	3	9	20
NSW: Wollongong	2	*BAU*	3	4	3			
		Abatement Package	3	2	1	0	1	2
VIC: Melbourne	42	*BAU*	93	107	131			
		Abatement Package	62	76	64	31	31	68
VIC: Latrobe Valley	1	*BAU*	1	1	2			
		Abatement Package	1	1	1	0	0	1
Hospital admissions for respiratory disease (65+ years)								
NSW: Sydney	293	*BAU*	701	893	1102			
		Abatement Package	589	511	338	113	382	765
NSW: Newcastle	75	*BAU*	203	246	284			
		Abatement Package	168	137	70	35	110	215
NSW: Wollongong	30	*BAU*	46	54	62			
		Abatement Package	38	30	14	8	24	48
VIC: Melbourne	664	*BAU*	1390	1594	1807			
		Abatement Package	921	1119	923	469	475	884
VIC: Latrobe Valley	15	*BAU*	15	16	17			
		Abatement Package	11	12	10	4	4	7
Emergency department visits for asthma (<15 years)								
NSW: Sydney	49	*BAU*	96	118	143			
		Abatement Package	82	73	44	15	45	99
NSW: Newcastle	9	*BAU*	24	29	34			
		Abatement Package	20	16	7	4	13	27
NSW: Wollongong	3	*BAU*	5	6	5			
		Abatement Package	4	4	2	1	2	3
VIC: Melbourne	183	*BAU*	423	489	571			
		Abatement Package	283	347	278	139	142	294
VIC: Latrobe Valley	5	*BAU*	5	6	6			
		Abatement Package	4	4	4	2	1	3

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(b) The method for calculating the health outcomes is based on averaging concentrations across an airshed. As some of the SO_2 monitoring stations are in locations that are influenced by emissions from industrial sources, this may lead to an overestimate of the risk to the population in the corresponding airshed. For example, this is particularly the case in the Melbourne airshed where estimated health outcomes are significantly above those of other pollutants and this is not considered to be reflective of current SO_2 levels in this airshed, which are well below AAQ NEPM standards.

Table 3-4: Historical health burden attributable to SO_2 in Australian airsheds (other airsheds)

Airshed	Number of attributable health outcomes (annual average 2010-2014)		
	Daily mortality, all-cause[(a)]	Hospital admissions for respiratory disease (65+ years)	Emergency department visits for asthma (<15 years)
QLD: Brisbane (SEQ)	17	147	36
SA: Adelaide	2	44	9
WA: Perth	18	279	52
NT: Darwin	0	0	0

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

3.3.3 Health outcomes for compliance with standards

3.3.3.1 1-hour standards

The proposed 1-hour standard of 75 ppb has only been exceeded in Perth and the Latrobe Valley (see Appendix A – air quality study, Section 2.2). These locations are dominated by large industrial sources, and the exceedances have been infrequent. The health outcomes associated with 1-hour SO_2 concentrations are therefore low, and for the majority of the Australian population meeting the proposed standards would not lead to a significant health benefit. The health outcomes for compliance with the proposed 1-hour standards have therefore not been presented here.

3.3.3.2 24-hour standards

The numbers of health outcomes associated with meeting the SO_2 24-hour standards of 7 ppb and 20 ppb are given in Annexure A, Section A.2.

Table 4-4 presents the numbers of health outcomes avoided in the NSW and Victoria airsheds by compliance with the proposed 24-hour standards for SO_2 of 7 ppb and 20 ppb. The corresponding results for the other airsheds (2010-2014 average only) are given in Table 4-5. The proposed 24-hour standard of 7 ppb has historically been exceeded in most Australian cities in some years (see Appendix A – air quality study, Section 2.2). It is clear that meeting the proposed 24-hour standard of 7 ppb would have a significant health benefit, especially in Melbourne. The 20 ppb standard is met in most airsheds, therefore the health benefits that would be achieved by meeting the proposed standard are smaller.

Table 3-5: Health outcomes avoided if proposed 24-hour SO_2 standards of 7 ppb and 20 ppb are met (NSW and Victoria airsheds)

Airshed	24-hour SO2 standard of 7 ppb				24-hour SO2 standard of 20 ppb			
	Health outcomes avoided				Health outcomes avoided			
	Average 2010-2014[a]	2021	2031	2040	Average 2010-2014	2021	2031	2040
Daily mortality, all-cause[b]								
NSW: Sydney	- [c]	- [c]	- [c]	- [c]	- [c]	- [c]	- [c]	- [c]
NSW: Newcastle	0	10	13	17	- [c]	- [c]	0	3
NSW: Wollongong	0	1	1	1	- [c]	- [c]	- [c]	- [c]
VIC: Melbourne	22	40	43	52	2	- [c]	- [c]	- [c]
VIC: Latrobe Valley	1	1	0	- [c]	- [c]	- [c]	- [c]	- [c]
Hospital admissions for respiratory disease (65+ years)								
NSW: Sydney	- [c]	- [c]	- [c]	- [c]	- [c]	- [c]	- [c]	- [c]
NSW: Newcastle	-2	0	0	0	- [c]	- [c]	0	0
NSW: Wollongong	3	0	0	10	- [c]	- [c]	- [c]	- [c]
VIC: Melbourne	353	596	634	718	238	- [c]	- [c]	- [c]
VIC: Latrobe Valley	-2	0	0	- [c]	- [c]	- [c]	- [c]	- [c]
Emergency department visits for asthma (<15 years)								
NSW: Sydney	- [c]	- [c]	- [c]	- [c]	- [c]	- [c]	- [c]	- [c]
NSW: Newcastle	0	14	18	23	- [c]	- [c]	0	4
NSW: Wollongong	1	1	1	1	- [c]	- [c]	- [c]	- [c]
VIC: Melbourne	97	181	194	227	7	- [c]	- [c]	- [c]
VIC: Latrobe Valley	3	2	2	- [c]	- [c]	- [c]	- [c]	- [c]

(a) Small negative values can occur where there are slight differences in the years with data.

(b) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(c) For these conditions, either the standard was already met in the airshed, or data were not available.

(d) The method for calculating the health outcomes is based on averaging concentrations across an airshed. As some of the SO_2 monitoring stations are in locations that are influenced by emissions from industrial sources, this may lead to an overestimate of the risk to the population in the corresponding airshed. For example, this is particularly the case in the Melbourne airshed where estimated health outcomes are significantly above those of other pollutants and this is not considered to be reflective of current SO_2 levels in this airshed, which are well below AAQ NEPM standards.

Table 3-6: Health outcomes avoided if proposed 24-hour SO_2 standards of 7 ppb and 20 ppb are met (other airsheds)

Airshed	24-hour SO2 standard of 7 ppb				24-hour SO2 standard of 20 ppb			
	Health outcomes avoided[a]				Health outcomes avoided[a]			
	Average 2010-2014	2021	2031	2040	Average 2010-2014	2021	2031	2040
Daily mortality, all-cause[b]								
QLD: Brisbane (SEQ)	- [c]	n/a	n/a	n/a	- [c]	n/a	n/a	n/a
SA: Adelaide	- [c]	n/a	n/a	n/a	- [c]	n/a	n/a	n/a
WA: Perth	3	n/a	n/a	n/a	- [c]	n/a	n/a	n/a
NT: Darwin	- [c]	n/a	n/a	n/a	- [c]	n/a	n/a	n/a
Hospital admissions for respiratory disease (65+ years)								
QLD: Brisbane (SEQ)	- [c]	n/a	n/a	n/a	- [c]	n/a	n/a	n/a
SA: Adelaide	- [c]	n/a	n/a	n/a	- [c]	n/a	n/a	n/a
WA: Perth	1	n/a	n/a	n/a	- [c]	n/a	n/a	n/a
NT: Darwin	- [c]	n/a	n/a	n/a	- [c]	n/a	n/a	n/a
Emergency department visits for asthma (<15 years)								
QLD: Brisbane (SEQ)	- [c]	n/a	n/a	n/a	- [c]	n/a	n/a	n/a
SA: Adelaide	- [c]	n/a	n/a	n/a	- [c]	n/a	n/a	n/a
WA: Perth	9	n/a	n/a	n/a	- [c]	n/a	n/a	n/a
NT: Darwin	- [c]	n/a	n/a	n/a	- [c]	n/a	n/a	n/a

(a) 'n/a' = not available (not modelled)

(b) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(c) For these conditions, either the standard was already met in the airshed, or data were not available.

3.3.4 Sensitivity tests

As only the Group 1 CRFs were used for SO_2, no sensitivity tests were conducted.

3.4 Summary

3.4.1 Literature review

There have been few new studies on the health effects of SO_2 since the NEPM review (NEPC, 2011a). The findings of the REVIHHAP study and the 2008 USEPA review concluded that the associations previously reported for short and long-term exposures to SO_2 and adverse health outcomes, such as increases in mortality and morbidity primarily from respiratory causes, remain valid. Asthmatics are still the most vulnerable group within the population for adverse effects from SO_2 exposure. The evidence of long-term effects associated with SO_2 is weak. No CRFs are available for long-term effects that would enable a health risk assessment to be conducted for these outcomes.

The 2011 OEHHA review provided evidence of reproductive and developmental effects associated with exposure to SO_2. These effects were not evaluated in the REVIHAAP report (WHO, 2013a).

The review of the recent health evidence supports the findings of the NEPM review (NEPC, 2011a). The lack of strong evidence for long-term health effects from exposure to SO_2 has led international agencies to revoke annual average standards and focus on implementing short-term standards for the protection of human health.

3.4.2 Exposure and risk characterisation

Exposure to recent historical levels of SO_2 in some Australian airsheds is associated with significant adverse health outcomes, and the health burden is projected to increase substantially in the future under the BAU scenario. In the Abatement Package scenario there are marked reductions in the incidence of the health outcomes, such that by 2040 all-cause mortality and respiratory illness due to SO_2 are generally approaching the 2010-2014 average, with the exception of Melbourne.

The proposed 1-hour standard of 75 ppb has only been exceeded in locations that are dominated by large industrial sources, and the exceedances have been infrequent. The health outcomes associated with 1-hour SO_2 concentrations are therefore low, and meeting the proposed standards would not lead to a significant health benefit for the majority of the Australian population.

The proposed 24-hour standard of 7 ppb has historically been exceeded in most airsheds, and compliance with the proposed 24-hour standard of 7 ppb would have a significant health benefit. The 20 ppb standard is met in most airsheds, therefore the health benefits that would be achieved by meeting the proposed standard are smaller.

4 Health risk assessment for nitrogen dioxide

4.1 Review of health effects

In recent years there has been a significant increase in the number of studies that have investigated the effects of NO_2 on health. The results of these studies have strengthened the evidence base for independent effects of NO_2 on health, and for long-term effects. These effects have been the subject of several reviews, and have led to the lowering of air quality standards for NO_2 in the USA and by the WHO. NO_2 is of concern not only because of the health effects associated with exposure directly to NO_2, but also because NO_2 is a precursor to both O_3 and secondary PM.

The REVIHHAP study (WHO, 2013a) examined the new studies of both long-term and short-term exposure to NO_2, and associations with mortality, hospital admissions and respiratory symptoms. It concluded that these new studies show that short-term exposure to NO_2 is associated with increases in these outcomes. Studies of the long-term effects of exposure to NO_2 have shown associations with both mortality and morbidity outcomes. The effects that have been observed for both long-term and short-term exposure are occurring below the current WHO air quality guidelines for NO_2 (97 ppb as a 24-hour average, and 19 ppb as an annual mean). The most recent review from the USEPA (2016) came to the same conclusion on the evidence for both short-term and long-term health effects attributable to NO_2. The USEPA concluded that the strongest associations were found with respiratory outcomes, and in particular asthma.

Controlled human exposure and toxicological studies have provided support for biological mechanisms for the effects that are observed in epidemiological studies, and provide evidence for a causal relationship between exposure to NO_2 and these outcomes (USEPA, 2016; WHO, 2013a). The most recent studies have provided evidence that NO_2 has an independent effect from other pollutants.

4.1.1 International studies

4.1.1.1 Short-term effects

Short-term exposure to NO_2 has been linked to increases in all cause, cardiovascular and respiratory mortality (WHO, 2013a; NEPC, 2010: USEPA, 2016). The effects have been shown to be greater in people 65 years of age and older, and for respiratory mortality. Epidemiological studies have provided no evidence of a threshold for the effect. The recent studies have provided evidence that has strengthened the association with hospital admissions and emergency department visits for respiratory disease, including all respiratory causes, asthma and COPD (WHO, 2013a). Strong associations have been observed for all respiratory causes in people 65 years and older, and for children with asthma. The effects are not as strong for cardiovascular causes and, in some cases, there is no consistent effect observed with cardiovascular effects. There is some evidence for an association with cardiac hospital admissions, but the findings are not consistent across studies.

Panel studies of children with asthma show associations between NO_2 and reductions in lung function, increases in cough, night-time asthma, and school absenteeism (WHO, 2013a; USEPA, 2016). There is also an increase in symptoms in asthmatic children and changes in lung function observed, as well as increases in airway inflammation and hyper-responsiveness. Controlled human exposure and animal toxicology support the findings of the epidemiological and panel studies.

Controlled human exposure studies show increased inflammation of the airways and airway hyper-responsiveness at NO_2 levels down to 0.2 ppm in healthy individuals. As previously mentioned, the

general population includes sensitive populations, and effects are likely to occur at lower levels of NO_2 than those for which adverse effects have been observed in controlled human exposure studies.

Epidemiological studies have found NO_2-related increases in airway inflammation in children with asthma and in several study populations that have a high prevalence of allergies (USEPA, 2016). These epidemiological associations were found in several studies characterised as having a strong exposure assessment, with school, personal monitoring or time weighted estimates of exposure. The observations of NO_2-related increases in allergic inflammation support the findings that NO_2 is associated with increases in airway responsiveness in adults with asthma, and increases in respiratory symptoms in children with asthma and allergy.

NO_2-related decreases in lung function have not been found consistently in adults with asthma in controlled human exposure studies, but have been observed in recent epidemiological studies of children with asthma (USEPA, 2016). In these studies several of the study populations of children with asthma also had high prevalence of allergy.

The USEPA (2016) concluded that the evidence indicates that there is a causal relationship between short-term NO_2 exposure and respiratory effects based on the consistency, coherence, and biological plausibility of findings from the effects of exacerbations on asthma. There is some evidence of relationships of NO_2 exposure with impaired host defence, COPD exacerbations, and respiratory mortality. Previous uncertainty regarding co-pollutant confounding has been reduced, with recent epidemiological studies showing that associations between ambient NO_2 concentrations and respiratory effects remain positive in co-pollutant models with $PM_{2.5}$, PM_{10}, SO_2, and O_3. Evidence for NO_2-induced increases in airway responsiveness in adults with asthma, from controlled human exposure studies, provides biological plausibility for effects on asthma exacerbation. Further evidence of NO_2-related oxidative stress and inflammation, including allergic inflammation, describes the biological processes and the modes of action for exacerbations of asthma. This epidemiological and experimental evidence together demonstrate the independent effects of short-term exposure to NO_2, and together form the basis of the USEPA concluding that a causal relationship exists.

4.1.1.2 Long-term effects

Epidemiological studies of long-term effects of NO_2 exposure on mortality (both respiratory and cardiovascular causes), and with children's respiratory symptoms and lung function, also support the conclusion that NO_2 has an independent effect on health (WHO, 2013a; COMEAP, 2014).

However, unlike particulate air pollution, which has been studied in detail, there is much greater uncertainty as to the quantitative health impact on large populations of long-term exposure to NO_2. The evidence is growing, but there is still uncertainty regarding long-term mortality impacts of NO_2 compared with those associated with particulate matter. There is only a limited number of large epidemiological studies which have estimated CRFs for mortality associated with long-term exposure to NO_2. Hence, there is much greater uncertainty associated with determining the appropriate CRF for long-term NO_2 exposure and mortality to use in an assessment of population health outcomes from exposure to NO_2. This uncertainty is reflected by WHO HRAPIE report, which classes the long-term mortality CRF for NO_2 as a Group B CRF "…. for which there is more uncertainty about the precision of the data used for quantification of effects" (WHO, 2013b).

In addition, there is likely to be considerable overlap between the health effects of long-term exposure to NO_2 and $PM_{2.5}$, as they are highly correlated. Any method to assess the number of health outcomes associated with NO_2 should include and adjustment to remove the contribution of $PM_{2.5}$.

Recent epidemiological studies do provide new evidence for an association between long-term exposure to NO_2 and respiratory effects in adults, with the most consistent findings for asthma (USEPA, 2016). Associations are less consistent for hospital admissions for COPD or asthma in adults. The results of studies in adults are also inconsistent for respiratory mortality associated with long-term NO_2 exposure. A recent study found higher NO_2 exposures among adults with hospital admissions and emergency department visits for pneumonia. Since the review of the AAQ NEPM there have been a number of studies examining the long-term effects of NO_2 on both mortality and morbidity. Several meta-analyses have been conducted to provide pooled estimates of the long-term effects of NO_2 on mortality and morbidity including the incidence of asthma (Faustini et al., 2014; Hamra et al., 2015).

The study by Faustini et al. (2014) conducted a meta-analysis of the long-term studies that were conducted between 2004 and 2013 evaluating the relationship between NO_2 and mortality. The effects of $PM_{2.5}$ were also available. Of 23 papers that were selected for review 19 were carried through to the meta-analysis. These studies were undertaken in various parts of the world including Europe, North America and Asia. The study found that the long-term effects of NO_2 on mortality were as great as that for $PM_{2.5}$. A review of the multi-pollutant models found that the effect of NO_2 was an independent effect. The pooled effect estimates were Relative Risk (RR) 1.04 (95% CI 1.02 – 1.06) for all cause (non-traumatic) mortality, 1.13 (95% CI 1.09 – 1.18) for cardiovascular mortality and 1.02 (95% CI 1.02 – 1.03) for respiratory mortality. For all cause (non-traumatic) and cardiovascular mortality the effect estimates based on interquartile ranges were greater for NO_2 compared with $PM_{2.5}$ – 6% NO_2 compared with 3% $PM_{2.5}$ for all-cause mortality and 29% for NO_2 compared with 16% for $PM_{2.5}$ for cardiovascular mortality.

A meta-analysis conducted to evaluate the association between NO_2 and lung cancer (Hamra et al., 2015) found that a 10 $\mu g/m^3$ increase in annual average NO_2 is associated with a 4% (95% CI 1% to 8%) increase in lung cancer. The meta-analysis included 20 studies from various parts of the world including North America, Europe and Japan. Restricting the analysis to studies that adjusted for confounding due to smoking status, socioeconomic status/income, education and/or occupation found that the effect estimates remained largely unchanged.

Hoek et al. (2013) conducted a review and meta-analysis of studies that had been conducted to evaluate the association between cardio-respiratory mortality and air pollution. The studies were drawn from Europe, North America, and Asia. The pooled effect estimate for NO_2 for all-cause mortality was a 5% increase (95% CI 3%, 8%) per 10 $\mu g/m^3$ increase in annual average NO_2. This finding is similar to that from the meta-analysis conducted by Faustini et al. (2014).

A recent meta-analysis conducted by Barone-Adesi et al. (2015) looked at the effects of long-term exposure of traffic related pollutants, including NO_2, and lung function in children. The meta-analysis included 9 studies including the Child Heart and Health Study in England (CHASE). The results of the meta-analysis showed that a 10 $\mu g/m^3$ increase in long-term NO_2 exposure was associated with a 0.7% decrease in FEV_1 in children. The authors concluded that this would translate to an increase of 7% (95% CI: 4% to 12%) in children with abnormal lung function. The observed effect was not modified by a reported asthma diagnosis.

Long-term exposure to NO_2 has been linked to deficits in lung function growth (USEPA, 2016). These findings have been found in studies in California, Mexico and Sweden. In these studies the effects on NO_2 were greater than those observed for other pollutants including $PM_{2.5}$. There is also strong evidence of an association between long-term exposure to NO_2 and the incidence of asthma and wheeze. This new evidence suggests that NO_2 exposure may actually cause asthma rather than just exacerbate existing asthma. There have also been studies showing increases in mortality with long-term exposure to NO_2 including all-cause, cardiovascular (especially ischaemic heart disease) lung

cancer and respiratory mortality. These effects are similar to those observed for $PM_{2.5,}$ if not greater, and are independent of $PM_{2.5}$.

The strongest evidence indicating that there is likely to be a causal relationship between long-term NO_2 exposure and respiratory effects comes from the associations consistently found between ambient NO_2 concentrations and asthma incidence in children in diverse geographical locations (USEPA, 2016; WHO, 2013a). This recent evidence is provided by several high quality single and multicity studies characterised by prospective follow-up of children, in several cases from birth to ages up to 12 years. Associations were found after adjustment for potential confounding by socio-economic status, smoking exposure, housing characteristics and the use of gas stoves. Associations were found with NO_2 obtained from central site monitors and more spatially resolved residential outdoor NO_2 estimated exposures. Asthma incidence was associated with the average NO_2 from the first year of life and NO_2 averaged over multiple years with study mean concentrations from 14 to 21 ppb. The relationship between long-term NO_2 exposure and asthma is supported by evidence of respiratory effects associated with short-term exposure to NO_2 (USEPA, 2016). Several epidemiological studies found increases in respiratory symptoms and pulmonary inflammation in children in the general population associated with short-term increases in ambient NO_2 concentrations. An effect on asthma incidence is also supported by evidence from prospective studies showing increases in respiratory symptoms in children with asthma associated with long-term NO_2 exposure (USEPA, 2016).

The USEPA (2016) concluded that the new evidence indicates that there is likely to be a causal relationship between long-term NO_2 exposure and respiratory effects based primarily on recent epidemiological findings in children for increases in asthma incidence and collective results showing decreases in lung function and partially irreversible decreases in lung function growth. Supporting evidence includes NO_2 related increases in respiratory symptoms in children with asthma, allergic sensitisation in children, asthma in adults and impaired host defence in animal models. NO_2 associations with respiratory effects remain positive in co-pollutant models and findings of NO_2 induced airway responsiveness and immune responses in experimental studies provide biological plausibility.

4.1.2 Australian studies

4.1.2.1 Short-term effects

Australian studies have reported similar associations between hospitalisation for respiratory effects, including asthma, and daily NO_2 as overseas studies (Morgan et al., 1998a; Barnett et al., 2005; Erbas B and Hyndman, 2005; Jalaludin et al. 2004; Rodriguez et al., 2007), although the effect estimates have been mixed, and a few studies reported no associations (e.g. Petroeschevsky et al., 2001). In a meta-analysis of results from 5 Australian and 2 New Zealand cities Barnett et al. (2005) analysed hospital admissions for 3 age groups of children. Significant increases in hospital admissions for respiratory disease (1–4 and 5–14 years) and asthma (5–14 years) were associated with interquartile range increases in either 1-hour or 24-hour NO_2. The largest association reported was a 6.0% increase in asthma admissions with a 5.1 ppb increase in 24- hour NO_2. The effect was not reduced by inclusion of PM_{10} in the analysis.

In the ACHAPS panel study (SCEW, 2012) the most consistent adverse effect was that increased NO_2 exposure was associated with an increased risk of cough and wheezing during the day and night, and increased use of bronchodilators for symptom relief. Relationships between NO_2 and night symptoms and effects were greater for NO_2 24-hour than for NO_2 1-hour and were more consistent. Effects on symptoms occurring during the day were strongest for same day exposures.

A meta-analysis of the associations between pollutants and cardiovascular hospital admissions in the elderly in Brisbane, Canberra, Melbourne, Perth, Sydney, Auckland and Christchurch found significant associations between CO, NO_2, and particles and five categories of cardiovascular disease admissions. The two largest statistically significant increases were for cardiac failure, with a 6.9% increase for a 5.1 ppb increase in NO_2 and a 6.0% increase for a 0.9 ppm increase in CO (Barnett et al., 2006).

4.1.2.2 Long-term effects

The ACHAPS cross-sectional study shows consistent evidence of respiratory adverse effects of NO_2 for both recent and life-time exposure (SCEW, 2012). These adverse effects are manifested as increased risk of asthma-like symptoms (in particular, wheeze), increased airway inflammation and reduced lung volumes. Airways inflammation, as measured by exhaled nitric oxide (NO), increased by 3% (1-5%) and lung volume as measured by pre-bronchodilator forced expiratory volume (FEV_1) and forced vital capacity (FVC) decreased by 7.1 ml (2.8-11.4) and 6.8 ml (2.7-10.9) per ppb respectively. The observed effects were slightly smaller for lifetime exposure.

There was no evidence that the effects were stronger in atopic subjects (people whose asthma is triggered by exposure to allergens). The absence of a greater effect in atopic subjects, the finding that lung volumes, rather than airway calibre (reflected in FEV_1/FVC ratio), and persistence of the effect after bronchodilator, imply that the consequence of NO_2 exposure is not typical asthma; instead, more non-specific lung effects are implicated (SCEW, 2012).

4.2 Exposure assessment

As with SO_2, to assess the exposure of the population to recent historical levels of NO_2, ambient air monitoring data obtained by the jurisdictional environment agencies between 2010 and 2014 were analysed. A summary of the data used for each airshed is shown in Table 4-1. No data were available for Tasmania, and in most jurisdictions NO_2 is not monitored in regional centres. Again, for the modelled projections, the health risk was assessed both with and without abatement measures, and the roll-back approach was used to determine the health outcomes avoided by compliance with the proposed standards.

Table 4-1: Summary of data used in exposure assessment for NO_2

Location	Period covered	Number of monitoring locations
NSW: Sydney	2010 - 2014	7
NSW: Newcastle	2010 - 2014	2
NSW: Wollongong	2010 - 2014	3
VIC: Melbourne	2010 - 2014	5
VIC: Latrobe Valley	2010 - 2014	1
QLD: Brisbane (SEQ)	2010 - 2014	5
SA: Adelaide	2010 - 2014	5
WA: Perth	2010 - 2014	7
NT: Darwin	2011 - 2014	2 (2012-13); 1 (2011, 2014)
ACT: Canberra	2010 - 2013	2

Airshed-average concentrations were determined using the approach described earlier for SO_2. The background value of 2.1 ppb cited by Frangos and DiMarco (2013) was used as the background NO_2 concentration for the purposes of this risk assessment. This value was subtracted from the measured airshed-average concentrations to provide NO_2 values which were representative of anthropogenic sources of NO_2.

As both short-term and long-term (annual average) standards are being proposed for NO_2 the effect of changing the concentration for one averaging period on the other had to be considered in the exposure assessment. For example, if the 1-hour data were adjusted to meet a proposed 1-hour standard, then the same roll-back was applied to the annual average data. This did not always result in the meeting of the proposed annual average standards. If the proposed 1-hour standards were met but the annual average standards were not, then the 1-hour data were adjusted by the amount needed to meet the annual average standards.

4.3 Risk characterisation

4.3.1 Concentration-response functions

NO_2 levels currently experienced in Australian cities have been associated with the following health outcomes:

- Increases in daily mortality
- Increases in hospital admissions for:
    - Respiratory disease
    - Cardiovascular disease
- Increases in emergency room attendances for asthma

In this health risk assessment, the health outcomes have been assessed for the relevant age groups.

At the time of conducting the literature review no studies investigating the long-term effects of exposure to NO_2 on health had been conducted in Australia. However, there have been several international studies that have shown strong associations between long-term exposure to NO_2 and increases in mortality. On the basis of the findings of these studies, long-term mortality has also been assessed.

The groups that were identified as being susceptible to the effects of NO_2 are:

- Elderly
- People with existing cardiovascular and respiratory disease
- People with asthma
- Low socioeconomic groups
- Children

The Group 1 and Group 3 (sensitivity test) CRFs for NO_2 are summarised in Table 4-2.

Table 4-2: Health outcomes and CRFs for NO_2 (Group1 and Group 3)

Averaging period/statistic	Health outcome (age group in years)	Group 1 CRFs		Group 3 CRFs	
		CRF [a, b]	Source	CRF [a, b]	Source
Annual average	Annual all-cause[c] mortality (30+)	0.94[d] (0.53 - 1.37)	WHO (2013b)	-	-
	Annual cardiovascular mortality (30+)	-	-	-	-
	Annual respiratory mortality (30+)	-	-	-	-
24-hour maximum 1-hour	Daily all-cause[c] mortality (all)	0.06 (0.03 - 0.08)	WHO (2013b)	-	-
	Daily cardiovascular mortality (30+)	-	-	-	-
	Daily respiratory mortality (30+)	-	-	-	-
24-hour average	Daily all-cause[c] mortality (all)	-	-	0.21 (0.06 - 0.74)	EPHC (2005)
	Daily cardiovascular mortality (30+)	-	-	0.21 (0.10 - 0.62)	EPHC (2005)
	Daily respiratory mortality (30+)	-	-	0.47 (0.14 - 1.64)	EPHC (2005)
	Hospital admissions for cardiovascular disease (65+)	0.30 (0.07 - 0.53)	EPHC (2005)	0.30 (0.07 - 0.53)	EPHC (2005)
	Hospital admissions for respiratory disease (65+)	0.37 (0.24 - 0.50)	WHO (2013b)	0.62 (0.25 - 1.10)	EPHC (2005)
	Hospital admissions for respiratory disease (15-64)	0.37 (0.24 - 0.50)	WHO (2013b)	0.21 (0.12 - 0.62)	EPHC (2005)
	Emergency department visits for asthma (<15)	0.12 (0.12 - 0.33)	EPHC (2005)	0.12 (0.12 - 0.33)	EPHC (2005)

(a) Percentage increase in health outcome per 1 ppb increase in NO_2 concentration

(b) Central estimate and 95% confidence interval. The range of these uncertainties have been derived directly from the source studies and reflects the uncertainty in the statistical analysis of the epidemiological data.

(c) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months (for daily all-cause mortality) and approximately 14 years (for annual all-cause mortality).

(d) The HRAPIE cut-off of 20 $\mu g/m^3$ for this CRF was applied in Group 1. The only difference between Group 2 and Group 1 is that a cut-off was not applied to this CRF in Group 2.

The CRFs used in Group 1 incorporated the recommendations of the HRAPIE project for the following (WHO, 2013b):

- All-cause mortality for ages 30+ from long-term exposure to NO_2.
- All-cause mortality for all ages from short-term exposure to NO_2.
- Hospital admissions due to respiratory disease for all ages 65+ from short-term exposure to NO_2.
- Hospital admissions due to respiratory diseases for ages 15-64 from short-term exposure to NO_2.

The Group 1 CRFs also excluded the application of a CRF for mortality due to cardiovascular and respiratory diseases for the following reasons:

- No corresponding recommendations were available from WHO (2013b).
- Including the CRFs recommended by Jalaludin and Cowie (2012) would result in an estimate of mortality from cardiovascular and respiratory disease that would be higher than total mortality, which is not meaningful.

As noted earlier, the Group 2 CRFs (also for sensitivity tests) were the same as those in Group 1, but without the application of the 20 μg/m^3 cut-off for mortality from long-term (annual) NO_2 exposure. The Group 3 CRFs for short-term (daily) effects were taken from EPHC (2005). No CRFs for long-term (annual) exposure to NO_2 were applied.

The numbers of attributable cases were calculated as described in Section 2.3.

For the purposes of the CBA, the HRAPIE recommendation to reduce the estimated mortality outcomes due to exposure to NO_2 by up to 33 per cent, to avoid double-counting with mortality associated with $PM_{2.5}$ exposure, was also adopted. A reduction of 16.6 per cent is applied, consistent with the guidance by Defra (2015). However, for the purposes of the HRA, which does not consider $PM_{2.5}$, no such reduction is applied.

4.3.2 Health outcomes for Business-as-Usual and Abatement Package scenarios

The estimates of the historical and projected health outcomes due to NO_2 in the modelled airsheds (NSW and Victoria) – based on the Group 1 CRFs – are shown in Table 4-3. The projected outcomes are presented for both the BAU and Abatement Package scenarios, and the health outcomes avoided in the latter are also stated. Table 4-4 gives the historical results for the non-modelled airsheds.

Table 4-3: Historical and projected health burden attributable to NO_2 in Australian airsheds (NSW and Victoria)

Airshed	Annual average 2010-2014	*Scenario*	Number of attributable health outcomes 2021	2031	2040	Health outcomes avoided 2021	2031	2040
Long-term, all-cause mortality (30+ years)								
NSW: Sydney	0[c]	*BAU*	0[c]	0[c]	0[c]			
		Abatement Package	0[c]	0[c]	0[c]	0[c]	0[c]	0[c]
NSW: Newcastle	0[c]	*BAU*	0[c]	0[c]	0[c]			
		Abatement Package	0[c]	0[c]	0[c]			
NSW: Wollongong	0[c]	*BAU*	0[c]	0[c]	0[c]			
		Abatement Package	0[c]	0[c]	0[c]	0[c]	0[c]	0[c]
VIC: Melbourne	0[c]	*BAU*	0[c]	0[c]	0[c]			
		Abatement Package	0[c]	0[c]	0[c]			
VIC: Latrobe Valley	0[c]	*BAU*	-[a]	-[a]	-[a]			
		Abatement Package	-[a]	-[a]	-[a]	-[a]	-[a]	-[a]
Daily mortality, all-cause[b]								
NSW: Sydney	42	*BAU*	35	39	46			
		Abatement Package	34	31	35	1	8	12
NSW: Newcastle	4	*BAU*	3	4	4			
		Abatement Package	3	3	3	0	1	1
NSW: Wollongong	2	*BAU*	1	1	1			
		Abatement Package	1	0	0	0	0	0
VIC: Melbourne	33	*BAU*	21	18	31			
		Abatement Package	19	17	21	2	1	10
VIC: Latrobe Valley	1	*BAU*	-[a]	-[a]	-[a]			
		Abatement Package	-[a]	-[a]	-[a]	-[a]	-[a]	-[a]
Hospital admissions, cardiovascular (65+ years)								
NSW: Sydney	1298	*BAU*	1704	1902	2246			
		Abatement Package	1647	1503	1690	58	399	556
NSW: Newcastle	101	*BAU*	117	132	150			
		Abatement Package	112	101	101	6	31	48
NSW: Wollongong	47	*BAU*	27	28	33			
		Abatement Package	24	18	17	2	10	16
VIC: Melbourne	884	*BAU*	869	735	1319			
		Abatement Package	784	702	901	85	33	417

Airshed	Annual average 2010-2014	Scenario	Number of attributable health outcomes 2021	2031	2040	Health outcomes avoided 2021	2031	2040
VIC: Latrobe Valley	14	*BAU*	-[a]	-[a]	-[a]			
		Abatement Package	-[a]	-[a]	-[a]	-[a]	-[a]	-[a]
Hospital admissions, respiratory (65+ years)								
NSW: Sydney	735	*BAU*	965	1077	1271			
		Abatement Package	932	851	957	33	226	315
NSW: Newcastle	49	*BAU*	57	65	73			
		Abatement Package	55	50	50	3	15	24
NSW: Wollongong	26	*BAU*	15	15	18			
		Abatement Package	13	10	9	1	5	9
VIC: Melbourne	507	*BAU*	498	421	756			
		Abatement Package	450	403	517	48	19	239
VIC: Latrobe Valley	9	*BAU*	-[a]	-[a]	-[a]			
		Abatement Package	-[a]	-[a]	-[a]	-[a]	-[a]	-[a]
Hospital admissions, respiratory (15-64 years)								
NSW: Sydney	583	*BAU*	420	469	561			
		Abatement Package	406	371	422	14	98	139
NSW: Newcastle	34	*BAU*	25	28	32			
		Abatement Package	24	22	22	1	7	10
NSW: Wollongong	21	*BAU*	8	8	10			
		Abatement Package	7	5	5	1	3	5
VIC: Melbourne	528	*BAU*	280	237	431			
		Abatement Package	253	226	295	27	11	137
VIC: Latrobe Valley	11	*BAU*	-[a]	-[a]	-[a]			
		Abatement Package	-[a]	-[a]	-[a]	-[a]	-[a]	-[a]
Emergency department visits, asthma (<15 years)								
NSW: Sydney	42	*BAU*	35	39	44			
		Abatement Package	34	31	33	1	8	11
NSW: Newcastle	2	*BAU*	2	2	2			
		Abatement Package	2	2	2	0	0	1
NSW: Wollongong	1	*BAU*	0	0	1			
		Abatement Package	0	0	0	0	0	0
VIC: Melbourne	57	*BAU*	37	31	53			
		Abatement Package	33	30	37	4	1	17
VIC: Latrobe Valley	1	*BAU*	-[a]	-[a]	-[a]			
		Abatement Package	-[a]	-[a]	-[a]	-[a]	-[a]	-[a]

(a) No projects for Latrobe Valley were available.

(b) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(c) The health burden is zero because average airshed long-term NO_2 concentrations were lower than that the HRAPIE cut-off of 20 μg/m^3, even though concentrations at some monitoring stations (e.g. in Sydney) were above this value.

Table 4-4: Historical health burden attributable to NO_2 in Australian airsheds (other airsheds)

Airshed	Number of attributable health outcomes (annual average 2010-2014) Long-term, all-cause[a] mortality (30+ years)	Daily mortality, all-cause[a]	Hospital admissions, cardiovascular (65+ years)	Hospital admissions, respiratory (65+ years)	Hospital admissions, respiratory (15-64 years)	Emergency department visits, asthma (<15 years)
QLD: Brisbane (SEQ)	0[b]	12	288	160	168	10
SA: Adelaide	0[b]	7	210	123	114	11
WA: Perth	0[b]	6	152	90	98	7
NT: Darwin	0[b]	0	0	0	3	0

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(b) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 μg/m^3.

In recent years (2010-2014) levels of NO_2 in Australian airsheds are likely to have had a substantial impact on public health, especially in the more urbanised airsheds of Sydney and Melbourne. To some extent, this has been masked by the application of the HRAPIE cut-off for annual mean NO_2 in the Group 1 CRFs as, with this cut-off not applied, mortality outcomes due to long-term exposure would be considerably larger (see, for example, the results for the Group 2 CRFs in Figure 4-1 in section 4.3.4). In the BAU scenario, for most of the short-term outcomes and locations the health burden in 2040 was projected to increase compared with 2021. Although the Abatement Package scenario generally led to some reductions in the incidence of the health outcomes, in some cases there was little improvement (and even some increases) relative to the 2010-2014 average.

4.3.3 Health outcomes for compliance with standards

4.3.3.1 1-hour standards

It was shown in the air quality study (Appendix A, Section 2.3) that the current AAQ NEPM standard for 1-hour NO_2 (120 ppb) is met in all airsheds. The proposed 1-hour standards of 80 ppb and 97 ppb are also currently met. However, there have historically been exceedances in the most airsheds of the proposed 1-hour standard of 40 ppb. All the proposed standards are predicted to be met in all modelled locations except Sydney. The 1-hour 40 ppb standard is the only standard predicted to be exceeded in Sydney.

The numbers of health outcomes associated with meeting the NO_2 1-hour standard of 40 ppb are given in Annexure B, Section B.2. Table 4-5 presents the numbers of health outcomes avoided in the NSW and Victoria airsheds by compliance with the proposed 1-hour standard for NO_2 of 40 ppb. The corresponding results for the other airsheds (20140-2014 average only) are given in Table 4-6. A significant number of attributable health outcomes would be avoided in Sydney if the proposed 1-hour standard for NO_2 of 40 ppb could be met.

Table 4-5: Health outcomes avoided if the proposed 1-hour NO_2 standard of 40 ppb is met (NSW and Victoria airsheds)

Airshed	1-hour NO_2 standard of 40 ppb Health outcomes avoided			
	Annual average 2010-2014[a]	2021	2031	2040
Long-term, all-cause[b] mortality (30+ years)				
NSW: Sydney	0[c]	0[c]	0[c]	0[c]
NSW: Newcastle	0[c]	- [d]	- [d]	- [d]
NSW: Wollongong	0[c]	- [d]	- [d]	- [d]
VIC: Melbourne	0[c]	- [d]	- [d]	- [d]
VIC: Latrobe Valley	- [d]	- [d]	- [d]	- [d]
Daily mortality, all-cause[b]				
NSW: Sydney	12	18	4	10
NSW: Newcastle	-1	- [d]	- [d]	- [d]
NSW: Wollongong	0	- [d]	- [d]	- [d]
VIC: Melbourne	6	- [d]	- [d]	- [d]
VIC: Latrobe Valley	- [d]	- [d]	- [d]	- [d]
Hospital admissions, cardiovascular (65+ years)				
NSW: Sydney	394	848	208	490
NSW: Newcastle	-50	- [d]	- [d]	- [d]
NSW: Wollongong	-5	- [d]	- [d]	- [d]
VIC: Melbourne	204	- [d]	- [d]	- [d]
VIC: Latrobe Valley	- [d]	- [d]	- [d]	- [d]
Hospital admissions, respiratory (65+ years)				
NSW: Sydney	223	480	118	277
NSW: Newcastle	-24	- [d]	- [d]	- [d]
NSW: Wollongong	-3	- [d]	- [d]	- [d]
VIC: Melbourne	117	- [d]	- [d]	- [d]
VIC: Latrobe Valley	- [d]	- [d]	- [d]	- [d]
Hospital admissions, respiratory (15-64 years)				
NSW: Sydney	167	209	51	122
NSW: Newcastle	-6	- [d]	- [d]	- [d]
NSW: Wollongong	-4	- [d]	- [d]	- [d]
VIC: Melbourne	94	- [d]	- [d]	- [d]
VIC: Latrobe Valley	- [d]	- [d]	- [d]	- [d]
Emergency department visits, asthma (<15 years)				
NSW: Sydney	12	18	4	10
NSW: Newcastle	-1	- [d]	- [d]	- [d]
NSW: Wollongong	0	- [d]	- [d]	- [d]
VIC: Melbourne	11	- [d]	- [d]	- [d]
VIC: Latrobe Valley	- [d]	- [d]	- [d]	- [d]

(a) Small negative values occur where there are slight differences in the years with data.

(b) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(c) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

(d) For these conditions, either the standard was already met in the airshed, or data were not available.

Table 4-6: Health outcomes avoided if the proposed 1-hour NO_2 standards of 40 ppb is met (other airsheds)

Airshed	1-hour NO2 standard of 40 ppb Health outcomes avoided[a]			
	Annual average 2010-2014	2021	2031	2040
Long-term, all-cause[b] mortality (30+ years)				
QLD: Brisbane (SEQ)	0[c]	n/a	n/a	n/a
SA: Adelaide	0[c]	n/a	n/a	n/a
WA: Perth	0[c]	n/a	n/a	n/a
NT: Darwin	-	n/a	n/a	n/a
Daily mortality, all-cause[b]				
QLD: Brisbane (SEQ)	10	n/a	n/a	n/a
SA: Adelaide	12	n/a	n/a	n/a
WA: Perth	9	n/a	n/a	n/a
NT: Darwin	-	n/a	n/a	n/a
Hospital admissions, cardiovascular (65+ years)				
QLD: Brisbane (SEQ)	244	n/a	n/a	n/a
SA: Adelaide	351	n/a	n/a	n/a
WA: Perth	216	n/a	n/a	n/a
NT: Darwin	-	n/a	n/a	n/a
Hospital admissions, respiratory (65+ years)				
QLD: Brisbane (SEQ)	136	n/a	n/a	n/a
SA: Adelaide	206	n/a	n/a	n/a
WA: Perth	128	n/a	n/a	n/a
NT: Darwin	-	n/a	n/a	n/a
Hospital admissions, respiratory (15-64 years)				
QLD: Brisbane (SEQ)	141	n/a	n/a	n/a
SA: Adelaide	191	n/a	n/a	n/a
WA: Perth	139	n/a	n/a	n/a
NT: Darwin	1	n/a	n/a	n/a
Emergency department visits, asthma (<15 years)				
QLD: Brisbane (SEQ)	8	n/a	n/a	n/a
SA: Adelaide	19	n/a	n/a	n/a
WA: Perth	10	n/a	n/a	n/a
NT: Darwin	-	n/a	n/a	n/a

(a) 'n/a' = not available (not modelled)

(b) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(c) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

4.3.3.2 Annual average standards

It was shown in the air quality study (Appendix A, Section 2.3) that the current NEPM standard for annual average NO_2 of 30 ppb and the proposed standard of 19 ppb are met in all airsheds. There have historically been exceedances of the proposed standard of 10 ppb in Sydney, the Port Phillip Region and Wollongong. In Sydney and the Port Phillip Region the exceedances of the 10 ppb standard have occurred at multiple stations and in multiple years.

Because average airshed concentrations were below the HRAPIE cut-off for long-term NO_2 concentrations of 20 $\mu g/m^3$ (around 10 ppb – i.e. the lowest proposed standard), no health benefits of meeting the standards could be determined.

It is worth noting that reducing the maximum 1-hour values to meet 40 ppb alone does not lead to the meeting of the 10 ppb annual average standard. However, in all cases assessed, meeting the 10 ppb annual standard leads to the 40 ppb 1 hour standard being met.

4.3.4 Sensitivity tests

For NO_2, the sensitivity testing examined the health outcomes in NSW and Victoria when the different CRF groups were used. In each state, and for each health outcome and period, the total number of health outcomes was summated across all airsheds for the BAU scenario. No projections were available for the Latrobe Valley, and therefore the future year results for Victoria only include Melbourne.

The results for NSW and Victoria are shown in Figures 4-1 and 4-2 respectively. Note that the difference in the number of health outcomes between the Group 1 and Group 2 CRFs for long-term all-cause mortality (30+ years) is due to the application of the 20 µg/m^3 cut-off (as recommended by HRAPIE). The HRAPIE recommended 16.6% adjustment to account for $PM_{2.5}$ does not contribute to this result as it has not been applied in the HRA (as discussed in Section 4.3.1).

It can be seen that:

- In the case of long-term all-cause mortality, the HRAPIE cut-off for NO_2 of 20 µg/m^3 that was applied in Group 1 meant that there were zero health outcomes, as annual mean concentrations in the airsheds were lower than this value. In Group 2 the cut-off was not applied, and it can be seen that this resulted in considerable numbers of outcomes. However, there is also uncertainty in terms of the health response below the cut-off. CRFs for this health outcome were not included for Group 3.
- The Group 1 CRFs used in the HRA gave much lower estimates than the Australian Group 3 CRFs for daily all-cause mortality and hospital admissions for respiratory disease (65+ years). Conversely, for hospital admissions for respiratory disease (15-64 years) the Group 3 CRFs gave lower results than the Group 1 CRFs.
- For hospital admissions for cardiovascular disease and emergency department visits for asthma, the Group 1 and Group 3 CRFs gave similar results.

When considering any differences between the groups, it should be borne in mind that level of confidence for the Group 1 CRFs is higher than that for the Group 3 CRFs, given that they are derived from a comprehensive and up-to-date meta-analysis. There are uncertainties associated with the CRFs adopted from some of the Australian studies. For example, the EPHC study from which the Group 3 CRFs for hospital admissions from short-term exposure to NO_2 were derived acknowledged that the results probably referred to impacts from a mixture of gases and particles.

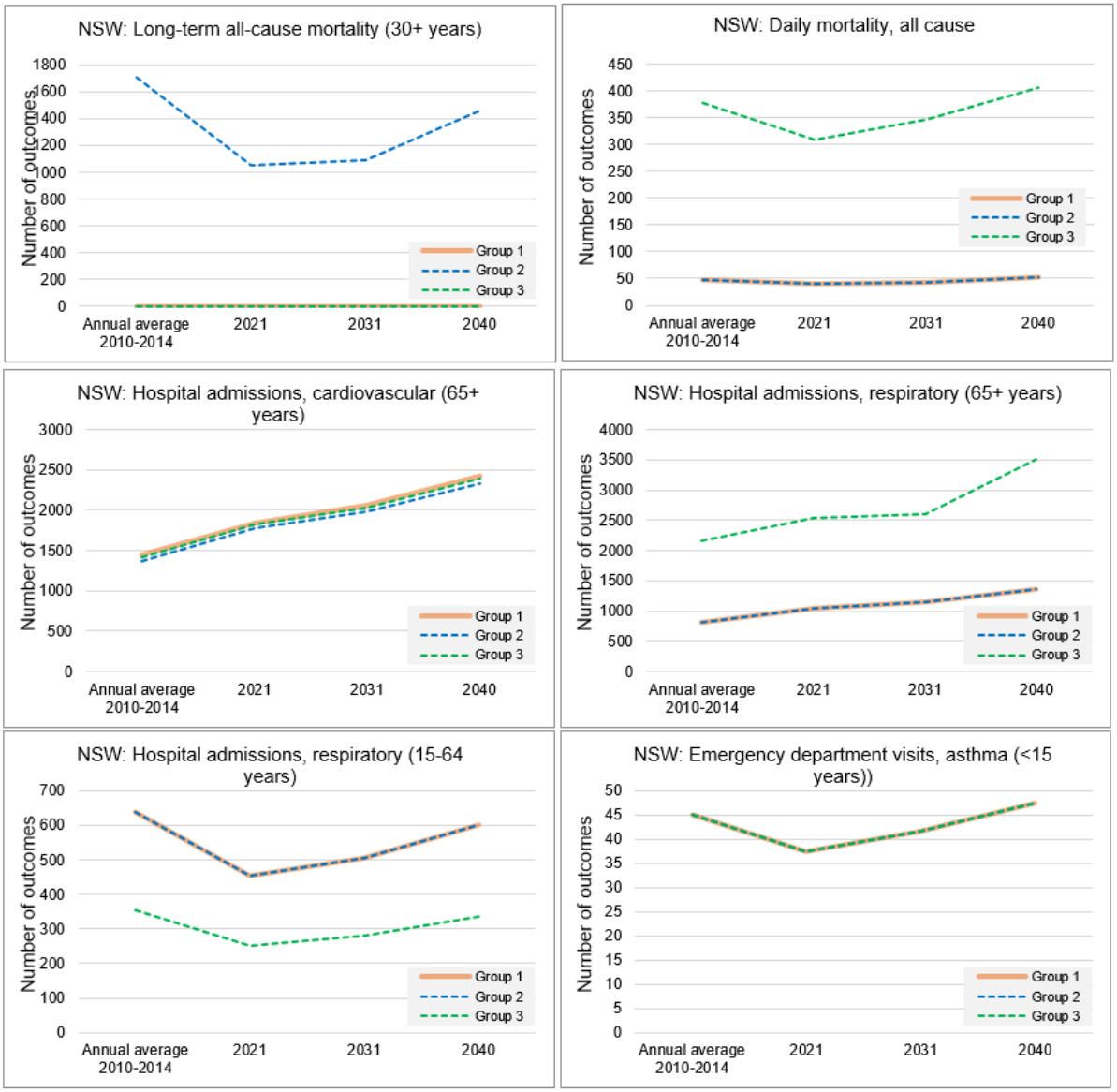

Figure 4-1: Comparison between NO_2 health outcomes in NSW for different CRF groups

As discussed above, the difference between the Group 1 and Group 2 results is due to the cut-off but not the 16.6% adjustment to account for $PM_{2.5}$, which has not been applied in the HRA.

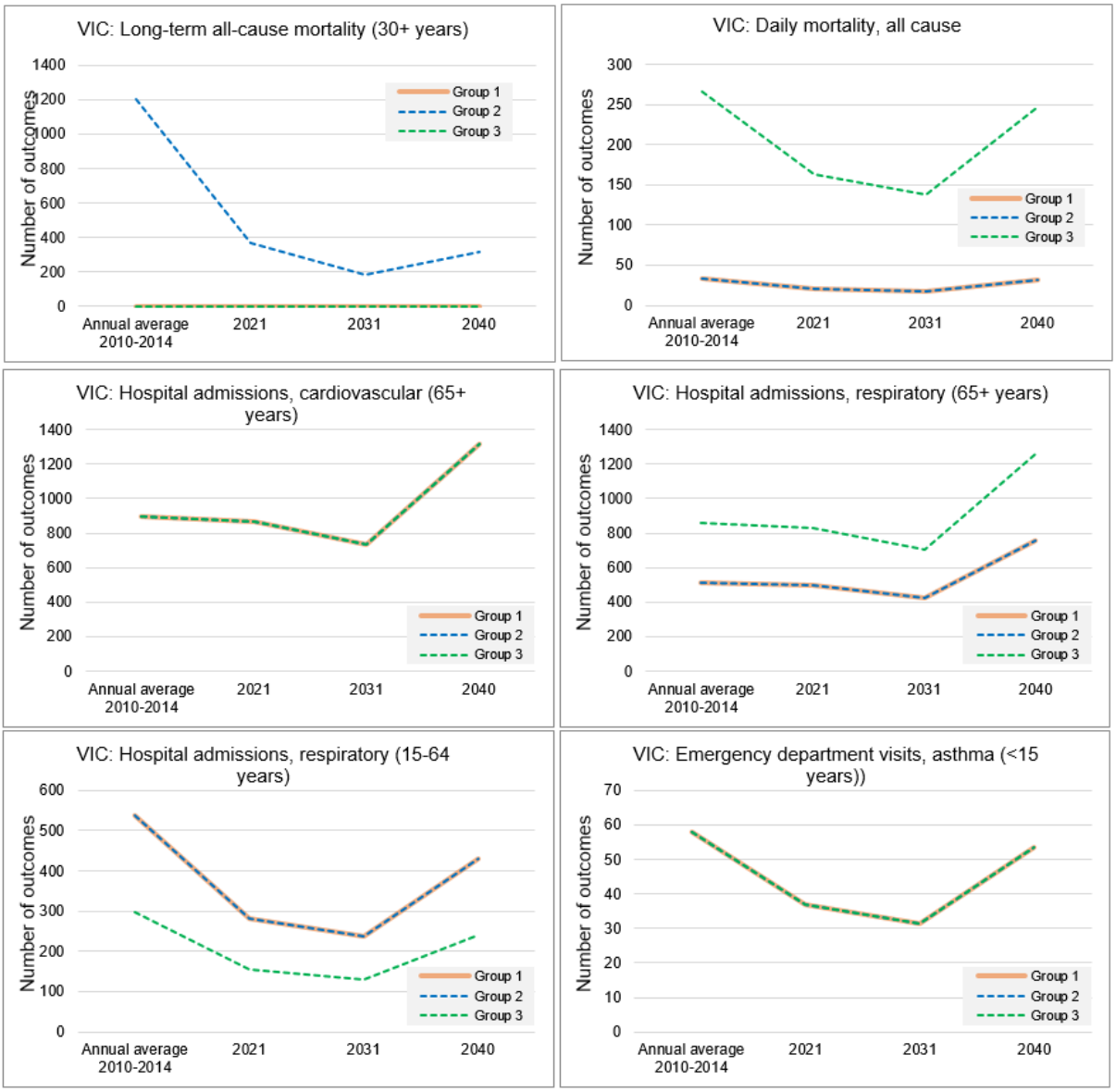

Figure 4-2: Comparison between NO_2 health outcomes in Victoria for different CRF groups

As discussed above, the difference between the Group 1 and Group 2 results is due to the cut-off but not the 16.6% adjustment to account for $PM_{2.5}$, which has not been applied in the HRA.

4.4 Summary

4.4.1 Literature review

The international reviews have found that there is considerable new evidence on the health effects of NO_2, and that these effects are independent of other pollutants including PM. In Australian studies the strongest and most consistent associations between mortality and hospital admissions and air pollution are found for NO_2. These effects are stronger than those observed for PM_{10} and $PM_{2.5}$. Long-term studies have found that exposure to NO_2 is causally linked to respiratory outcomes including asthma incidence and reduced lung function growth.

The review of the current literature strengthens the findings of the NEPM review (NEPC, 2011a).

4.4.2 Exposure and risk characterisation

The current AAQ NEPM standard for 1-hour NO_2 (120 ppb) is met in all airsheds. All the proposed standards are predicted to be met in all modelled locations except Sydney. The proposed 1-hour 40 ppb standard is the only standard predicted to be exceeded in Sydney. The current AAQ NEPM standard for annual average NO_2 (30 ppb) is met in all airsheds. The proposed annual average standards of 19 ppb and 10 ppb are also currently met.

However, in recent years levels of NO_2 in Australian airsheds have still had a significant impact on public health, especially in the more urbanised airsheds of Sydney and Melbourne. The health burden is also projected to increase in these cities in the future. The Abatement Package scenario generally led to relatively small reductions in health outcomes.

A significant number of attributable health outcomes would be avoided in Sydney if the proposed 1-hour standard for NO_2 of 40 ppb could be met.

5 Health risk assessment for ozone

5.1 Review of health effects

O_3 is a secondary pollutant and is formed from precursors such as oxides of nitrogen and VOCs (volatile organic compounds). Ambient monitoring shows that there is significant year-to-year variability in the O_3 levels observed in our cities. O_3 levels are influenced by hot summers (temperature) and bushfires. The main health effects associated with exposure to O_3 are on the respiratory tract. The mechanism by which O_3 affects the respiratory tract include the formation of secondary oxidation products in the lung, activation of neural reflexes, initiation of inflammation, alterations of epithelial barrier function, sensitisation of bronchial smooth muscle, changes in immunity and airway remodelling. Systemic inflammation and oxidative stress may also be critical to the effects of O_3 on the cardiovascular system, as observed in some studies.

The most recent reviews conducted by the international agencies including WHO (2013b), USEPA (2016) and COMEAP (2015) have investigated the evidence on both long-term and short-term effects of O_3 on health. The reviews of the short-term evidence have found that there is new evidence on the short-term effects associated with daily maximum 1-hour and 8-hour O_3 concentrations with all cause, cardiovascular and respiratory mortality as well as cardiovascular and respiratory hospital admissions (WHO, 2013; USEPA, 2013; COMEAP, 2015). The evidence base for long-term effects of O_3 has also strengthened, in particular, for increases in asthma incidence and impacts on lung function growth.

5.1.1 International studies

5.1.1.1 Short-term effects

In a review of the O_3 standards in the United States, the USEPA concluded that there was clear, consistent evidence of a causal relationship between short-term exposure to O_3 and respiratory health effects (USEPA, 2006). This finding was supported by the coherence of effects across a range of epidemiological, controlled human exposure and toxicological studies. These findings indicated that the effects of short-term exposure to O_3 can impact on a range of respiratory health endpoints. This ranges from respiratory tract inflammation to respiratory related emergency department visits and hospital admissions.

There was strong evidence that short-term O_3 exposures induced or were associated with statistically significant declines in lung function (WHO, 2006). An equally strong body of evidence from controlled human exposure and toxicological studies demonstrated that O_3 induced inflammatory responses, increased epithelial permeability and airway hyper-responsiveness. These findings supported the outcomes of epidemiological studies which showed that short-term increases in O_3 concentrations were consistently associated with increases in respiratory symptoms and asthma medication use in children with asthma, respiratory-related hospital admissions and asthma-related emergency department visits.

In the 2013 review, the USEPA concluded that more recent studies built on the findings of the previous review and strengthened the evidence that short-term exposure to O_3 is causally associated with respiratory health effects. Recent controlled human exposure studies have shown that O_3 levels as low as 60-70 ppb are associated with statistically significant group mean decreases in pulmonary function in young healthy adults. These results are supported by the findings of epidemiological studies that provide strong evidence of associations between O_3 exposure and respiratory hospital admissions and emergency department visits across the US, Canada and Europe (USEPA, 2013; WHO, 2013a). Several multicity studies and multi-continent study reported associations between

short-term increases in ambient O_3 concentrations and increases in respiratory mortality. A large body of individual level epidemiological panel studies have demonstrated associations between exposure to O_3 and respiratory symptoms in children with asthma. The findings of these studies are supported by recent studies that found O_3-associated increases in indicators of airway inflammation and oxidative stress in children with asthma.

The USEPA (2013) concluded that the evidence from toxicological studies including O_3 induced airways hyper-responsiveness, decreased pulmonary function, allergic responses, lung injury, impaired host defence and airway inflammation, have characterised modes of action and provided biological plausibility for the associations observed in epidemiological studies of ambient O_3 concentrations with lung function and respiratory symptoms, hospital admissions and emergency department visits and mortality. They further concluded that the evidence integrated across the controlled human exposure, epidemiological and toxicological studies and across the spectrum of respiratory health endpoints continues to demonstrate that there is a causal relationship between short-term O_3 exposure and respiratory health effects.

Although there is evidence for adverse respiratory health effects associated with long and short-term exposure to O_3, the USEPA (2013) identified that the evidence for an association between exposure to O_3 and cardiovascular effects is not as strong. Controlled human exposure studies have shown increases and decreases in high frequency HRV following relatively low [120 ppb during rest] and high [300 ppb with exercise] O_3 exposures, respectively (USEPA, 2013). The USEPA (2013) noted that animal toxicology studies, although limited in number, suggest that short-term O_3 exposure induces vascular oxidative stress and release of pro-inflammatory mediators, alters heart rate (HR) and heart rate variability (HRV), and disrupts the regulation of the pulmonary endothelin system (USEPA, 2013). It was noted that the changes in cardiac function observed in animal and human studies provided preliminary evidence for O_3-induced modulation of the autonomic nervous system through the activation of neural reflexes in the lung. Controlled human exposure studies also support the animal toxicology studies by demonstrating O_3-induced effects on blood biomarkers of systemic inflammation and oxidative stress as well as changes in biomarkers suggestive of a pro-thrombogenic response to O_3.

In 2008 the WHO published a report on the long-range transport of air pollution. The study focused on the health effects associated with O_3 exposures related to long-term transport of O_3 across Europe (WHO, 2008). The WHO identified that in terms of short-term exposures to O_3, the recent epidemiological studies had strengthened the evidence that daily exposures to O_3 increased mortality and respiratory morbidity rates. Studies on pulmonary function, lung inflammation, lung permeability, respiratory symptoms, increased medication usage, morbidity and mortality, indicated that O_3 appears to have an effect independent of other pollutants including particulate matter. The WHO concluded that evidence that O_3 may act independently of other pollutants is supported by the results of controlled human exposure studies and toxicological studies showing the potential of O_3 per se to cause adverse health effects especially in vulnerable people.

In regard to the cardiovascular effects of O_3, the USEPA (2016) concluded that the animal toxicological studies demonstrate O_3-induced cardiovascular effects, and support the strong body of evidence indicating O_3-induced cardiovascular mortality. Animal toxicological and controlled human exposure studies provide evidence for biologically plausible mechanisms underlying these O_3-induced cardiovascular effects. However, a lack of coherence with epidemiologic studies of cardiovascular morbidity remains an important uncertainty. Taken together, the overall body of evidence across disciplines led the USEPA to determine that there is sufficient evidence to conclude that there is likely to be a causal relationship between relevant short-term exposures to O_3 and cardiovascular effects. The WHO (2013b) considered that the toxicological data from animal and human exposure studies

provided ample support for the short-term effects of O_3 on a range of pulmonary and vascular health-relevant endpoints (WHO, 2013a).

5.1.1.2 Long-term effects

The results of recent studies have strengthened the evidence base for long-term impacts of O_3 on health (WHO, 2013a; USEPA, 2013). Studies have shown that long-term exposure to O_3 has an impact on people with existing disease particularly in those with COPD, diabetes, congestive heart failure and myocardial infarction. Long-term exposure to O_3 has also been associated with an increase in asthma incidence, asthma severity, hospital care for asthma and lung function growth (USEPA, 2013; WHO, 2013a). The toxicological data supports the findings of epidemiological studies and has strengthened in recent years. The toxicological studies show evidence of chronic injury and long-term structural change to the airways of animals exposed to prolonged periods of O_3 as well as O_3 and allergens combined. New human epidemiological studies and animal toxicological studies suggest an effect of O_3 exposure on cognitive development and reproductive health including preterm birth (USEPA, 2013). The strongest evidence for a relationship between long-term exposure to O_3 and respiratory morbidity comes from studies that demonstrate long-term exposures to O_3 are associated with new onset asthma in children as well as increases in respiratory symptom effects in children with asthma (WHO, 2013a; USEPA, 2013). However, recent animal toxicological studies have demonstrated O_3-induced cardiovascular effects, specifically enhanced ischemia/reperfusion injury, disrupted NO-induced vascular reactivity, decreased cardiac function and increased heart rate variability (HRV). These effects are consistent with cardiovascular system effects observed after long-term O_3 exposure, such as increased vascular disease. These effects may in part correspond to the alteration of the autonomic nervous system or to the development and maintenance of systemic oxidative stress and a pro-inflammatory environment that can result from pulmonary inflammation (USEPA, 2013).

The USEPA (2013) found that the evidence from long-term studies of O_3 exposure shows associations with respiratory health effects including new-onset asthma and respiratory mortality. A US multi-community prospective cohort study demonstrated that asthma risk is affected by interactions between genetic variability, environmental O_3 exposure and behaviour. The evidence relating long-term O_3 exposure to new-onset asthma is supported by toxicological studies of asthma in monkeys (USEPA, 2013). The results of this study support the biological plausibility of long-term exposure to O_3 contributing to the effects of asthma in children. The epidemiological studies showing an association between new-onset asthma and O_3 showed that the associations were observed in people living in areas where the mean annual 8-hour maximum O_3 concentration was 55.2 ppb compared with those who lived where it was 38.4 ppb. Early life O_3 exposure can alter airway development and lead to the development of asthma. The USEPA (2013) concluded that other recent epidemiological studies provide coherent evidence for long-term O_3 exposure and respiratory effects such as first asthma hospitalisation, respiratory symptoms in asthmatics and respiratory mortality. These studies have been conducted in areas with mean 8-hour maximum O_3 less than 63 ppb. Generally the epidemiological and toxicological evidence provides a compelling case that supports the hypothesis that a relationship exists between long-term exposure to O_3 and measures of respiratory health and mortality. The evidence for short-term exposure to O_3 and effects on the respiratory system provides coherence and biological plausibility for the effects of long-term exposure to O_3. The USEPA (2013) concluded that there is biologically plausible evidence of a likely causal relationship between long-term exposure to O_3 and respiratory effects.

The USEPA (2013) conducted a synthesis of new and old research on the association between long-term exposure to O_3 and mortality and found that the strongest evidence for an association is with respiratory mortality after adjusting for the effects of $PM_{2.5}$. There was inconsistent evidence for an

association between long-term exposure to ambient O_3 and cardiopulmonary mortality. Several analyses from the American Cancer Society study reported some positive associations while other studies reported none. There is generally limited evidence for an association between long-term exposure to ambient O_3 and total mortality (USEPA, 2013). The findings for respiratory mortality are consistent and coherent with the evidence from epidemiological studies, controlled human exposure and animal toxicological studies for the effects of short-term and long-term exposure to O_3 on respiratory effects. The USEPA (2013) concluded that the overall evidence is suggestive of a causal relationship between long-term exposure to O_3 and total (non-accidental) mortality.

The USEPA (2013) also concluded that the existing evidence is suggestive of a causal relationship between long-term exposures to O_3 and cardiovascular effects. This is based on the results of recent studies, in particular animal toxicological studies that have shown that long-term exposure to O_3 leads to cardiovascular morbidity including increased vascular disease. Overall the evidence from the toxicological studies provide some evidence for O_3-induced cardiovascular effects but the effects observed were not consistently supported by controlled human exposure studies or epidemiological studies.

The USEPA (2013) also concluded that evidence from recent studies is suggestive of a causal relationship between long-term exposure to O_3 and reproductive and developmental effects. This was based on limited evidence of an association between long-term exposure to O_3 and decreased sperm concentration as well as reduced birth weight and restricted foetal growth. Studies on the association of O_3 exposure and central nervous system effects have provided evidence that is suggestive of a causal relationship between O_3 exposure and central nervous system effects (USEPA, 2013). These effects include alterations in neurotransmitters, motor activity, short and long-term memory, sleep patterns and histological signs of neurodegeneration.

5.1.1.3 Evidence of a threshold for effect

Studies that have investigated the presence of a threshold for the effects of O_3 have found no evidence of a threshold for long-term effects, in particular mortality. Controlled human exposure studies show that O_3 at 60 ppb causes impaired lung function and inflammation of the airways in healthy adults. It is expected that effects would occur at lower levels in susceptible groups however controlled exposure studies cannot include these groups as only healthy volunteers or people with mild disease can be included. Studies conducted at children's summer camps have seen adverse effects at lower concentrations of O_3 than those observed in controlled human exposure studies (USEPA, 2013; WHO, 2013a). This is thought to reflect the response of susceptible groups in the general population. The multi-city studies on mortality and hospital admission outcomes have shown no evidence for a threshold for adverse effects. The WHO (2013b) concluded that if a threshold does occur that it is below 45 ppb 1-hour maximum O_3.

5.1.2 Australian studies

Australian studies on the health effects of O_3 have shown mixed results. Early studies conducted in Sydney, Melbourne, Perth and Brisbane found mixed results. Associations between 1, 4 and 8-hour O_3 levels and total mortality were reported in Brisbane (Simpson et al., 1997), Melbourne (Simpson et al. 2001) and Sydney (Morgan et al., 1998b), but a later multi-city study reported no association (Simpson et al., 2005b). Associations between 4-hour and 8-hour O_3 levels were reported in studies in Perth (Hinwood et al., 2004) and the multi-city and Melbourne studies mentioned above, but not in the Brisbane and Sydney studies. Only the Perth study reported a significant association between O_3 exposure and cardiovascular mortality.

A more recent study conducted in Adelaide (Chen et al., 2016) used air pollution data from 2003 – 2013 to examine the association between air pollution and hospital admissions for asthma. The study found that the largest effect for O_3 was in the warm season with the 5 day cumulative effect of an 11.7% increase in the risk of asthma hospital admissions in children (0-17 years) per 10 ppb increment in O_3. For all other pollutants considered the strongest effects were observed in the cool season.

Mixed results have also been reported for associations between O_3 levels and hospitalisation for respiratory disease. Two multi-city studies (Barnett et al. 2005, Simpson et al. 2005a), reported positive associations between increases in O_3 levels of 1 ppb and respiratory admissions. This association was only demonstrated in one of the studies in the warm season (Barnett et al. 2005). EPA Victoria (2001) reported increases in hospital admissions for respiratory and asthma with increases in O_3 levels, with larger increases reported in the warm season. A study of childhood emergency presentations for asthma in Melbourne regions reported mixed results with a positive significant (non-linear) relationship with O_3 concentrations in the Western and South/South Eastern regions, but not in other regions (Erbas et al., 2005). An 11 month longitudinal panel study of 125 primary school children with a history of wheeze in Sydney, reported no associations between ambient O_3 concentrations and respiratory symptoms, asthma medication use, and doctor visits for asthma (Jalaludin et al., 2004). Another panel study in Perth found an association between O_3 and raised body temperature on the day of onset of upper respiratory illness. Incidence of cough was also increased, but not significantly (Rodriguez et al. 2007).

Mixed results were also reported for associations between O_3 levels and hospitalisation for cardiovascular disease, with only Simpson et al. (2005a, 2005b) reporting an association between O_3 levels and hospital admissions for cardiovascular causes. A study of emergency department attendances in Sydney found no effect of O_3 on the rate of attendances for cardiovascular conditions in the elderly (Jalaludin et al., 2006).

Of the birth studies undertaken in Australia, a study in Brisbane reported exposure to O_3 during trimester one was associated with an increased risk of pre-term birth with an odds ratio of 1.26 (95%, 1.10-1.45%) (Hansen et al. 2006). A similar finding, limited to pregnancies conceived in the spring, was demonstrated in Sydney (Jalaludin et al., 2007). Another study in Brisbane reported that there was no strong evidence of an association between O_3 levels during pregnancy and sub-optimal foetal growth (Hansen et al., 2007). A study of 'small for gestational age' babies over a two year period in Sydney did not report any association between birth weight and O_3 levels (Mannes et al. 2005).

In summary, some studies show no impacts even when the analyses are restricted to the warm months, while others show a protective effect, that is, that O_3 actually has a health benefit (SCEW, 2012). The reason for the observed protective effects is unclear. The reactions that lead to the formation of O_3 require UV radiation and are temperature dependent with higher O_3 concentrations associated with higher temperatures. The complex photochemistry between NO_2 and O_3 is a further confounding factor that may influence the associations observed between O_3 and adverse health effects. This is an issue raised by COMEAP (2015) that needs further consideration in conducting health impact assessments.

5.2 Exposure assessment

The HRA has been undertaken to assess the risk from current levels of O_3 experienced in Australian cities and large regional centres, and for the years 2021, 2031 and 2040 for Melbourne, Latrobe Valley, Sydney, Wollongong and Newcastle. For the modelled projections, the health risk was assessed both with, and without, abatement measures. The number of health outcomes avoided if the proposed standards could be met was determined by rolling back the daily 1-hour maximum O_3

concentrations so that the alternate standards could be met. This is consistent with the NEPC standard-setting methodology (NEPC, 2011b).

To assess the exposure of the population to current levels of O_3, ambient air monitoring data obtained by the jurisdictional environment agencies was used. Data from 2010-2014 was generally available. All data was obtained from monitoring locations established to meet the requirements of the NEPM to provide data that is generally representative of population exposure. A summary of the data used for each location is shown in Table 5-1. No data were available for Tasmania.

To assess exposure to daily 1-hour maximum concentrations, the daily 1-hour maximums for all sites were averaged across the network to obtain a value that was representative of total population within the airshed. This was done for each day of the year.

The use of daily maximum 8-hour concentrations for O_3 was required for the exposure assessment using the Group 1 and 2 CRFs. However, the air quality modelling did not provide outputs for all the scenarios relating to compliance with proposed air quality standards. Therefore, to estimate maximum 8-hour O_3 concentrations a ratio between closely-related metrics was calculated using regression analysis of modelled data from Sydney and Melbourne in 2010 and 2014. Maximum 8-hour O_3 concentrations were estimated by applying a ratio of maximum 1-hour and 8-hour O_3 concentrations, derived from regression analysis of the sample data. The regression analysis showed a strong fit to the sample data, with an adjusted R^2 value of 99%.

The background value of 8.7 ppb cited by Frangos and DiMarco (2013) was used as the background O_3 concentration for the purposes of this risk assessment. This value was subtracted from the network average datasets to provide O_3 data representative of anthropogenic sources of O_3 in Australian cities.

Table 5-1: Summary of monitoring data used in exposure assessment for O_3

Location	Period	Number of monitoring locations
NSW: Sydney	2010 - 2013	7
NSW: Newcastle	2010 - 2013	2
NSW: Wollongong	2010 - 2013	3
VIC: Melbourne	2010 - 2014	5
VIC: Latrobe Valley	2010 - 2014	1
QLD: Brisbane (SEQ)	2010 - 2014	5
SA: Adelaide	2010 - 2014	5
WA: Perth	2010 - 2014	7
NT: Darwin	2011 - 2014	2 (2012-13); 1 (2011, 2014)
ACT: Canberra	2010 - 2013	2

5.3 Risk characterisation

5.3.1 Concentration-response functions

The results of epidemiological studies have shown that a range of health effects are associated with exposure to O_3. International studies have found associations between O_3 levels currently experienced in Australian cities and the following health outcomes:

- Increases in daily mortality – all causes (non-traumatic), respiratory and cardiovascular disease
- Increases in hospital admissions for:
    - Respiratory disease
    - Cardiovascular disease
- Increases in emergency room attendances for asthma

These health outcomes have been assessed in this health risk assessment for the relevant age groups.

The groups that were identified as being susceptible to the effects of O_3 are:

- Elderly
- People with existing cardiovascular and respiratory disease
- People with asthma
- Children

The Group 1 and Group 3 (sensitivity test) CRFs for O_3 are summarised in Table 5-2. For Group 1, the CRF for emergency department visits was based on the recommendations of the HRAPIE project covering studies in 32 European cities (WHO, 2013b). Group 1 also included a CRF for all-cause mortality (all ages) from 8-hour exposure to O_3 from EPHC (2005). As in the case of NO_2, the Group 1 CRFs excluded mortality due to cardiovascular and respiratory disease. The reasons for this were explained in the corresponding section of the Appendix for NO_2. As noted earlier, the Group 2 CRFs were the same as those in Group 1, but without the application of the 35 ppb cut-off for mortality from short-term O_3 exposure. The Group 3 CRFs were taken from EPHC (2005).

Table 5-2: Health outcomes and CRFs for O_3 (Group1 and Group 3)

Averaging period/statistic	Health outcome (age group in years)	Group 1 CRFs		Group 3 CRFs	
		CRF [a, b]	Source	CRF [a, b]	Source
Daily maximum 1-hour	Daily all-cause[c] mortality (all)	-	-	0.14 (0.03 - 0.24)	EPHC (2005)
	Daily cardiovascular mortality (all)	-	-	0.21 (0.11 - 0.32)	EPHC (2005)
	Daily respiratory mortality (all)	-	-	0.23 (0.01 - 0.48)	EPHC (2005)
	Emergency department visits for asthma (<15)	0.10 (0.08 - 0.18)	EPHC (2005)	0.10 (0.08 - 0.18)	EPHC (2005)
Daily maximum 8-hour	Daily all-cause[c] mortality (all)	0.06[d] (0.03 - 0.09)	WHO (2013b)	-	-
	Daily cardiovascular mortality (all)	-	-	-	-
	Daily respiratory mortality (all)	-	-	-	-

(a) Percentage increase in health outcome per 1 ppb increase in O_3 concentration

(b) Central estimate and 95% confidence interval. The range of these uncertainties have been derived directly from the source studies and reflects the uncertainty in the statistical analysis of the epidemiological data.

(c) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months (for daily all-cause mortality) and approximately 14 years (for annual all-cause mortality).

(d) The HRAPIE cut-off of 35 ppb for this CRF was applied in Group 1. The only difference between Group 2 and Group 1 is that a cut-off was not applied to this CRF in Group 2.

5.3.2 Health outcomes for Business-as-Usual and Abatement Package scenarios

The estimates for the number of historical and projected attributable health outcomes due to O_3 in the modelled airsheds (NSW and Victoria) are shown in Table 5-3. As before, the projected outcomes are presented for both the BAU and Abatement Package scenarios, and the health outcomes avoided in the latter are also stated. Table 3-4 gives the historical results for the non-modelled airsheds. The results show that there is a significant health burden associated with exposure to recent historical levels of O_3, particularly in the main cities. In Sydney and Melbourne the health burden is projected to increase substantially in the future under the BAU scenario.

Table 5-3: Historical and projected health burden attributable to O_3 in Australian airsheds (NSW and Victoria)

Airshed	Annual average 2010-2014	Number of attributable health outcomes				Health outcomes avoided		
		Scenario	2021	2031	2040	2021	2031	2040
Daily mortality, all-cause[(a)]								
NSW: Sydney	152	*BAU*	54	67	78			
		Abatement Package	50	60	72	4	7	6
NSW: Newcastle	13	*BAU*	5	6	6			
		Abatement Package	5	5	6	-	-	-
NSW: Wollongong	5	*BAU*	1	1	1			
		Abatement Package	1	1	2	-	-	-
VIC: Melbourne	46	*BAU*	50	59	70			
		Abatement Package	42	54	63	8	5	7
VIC: Latrobe Valley	1	*BAU*	1	1	1			
		Abatement Package	1	1	1	-	-	-
Emergency department visits, asthma (<15 years)								
NSW: Sydney	122	*BAU*	93	111	123			
		Abatement Package	93	111	122	-	-	1
NSW: Newcastle	8	*BAU*	5	6	6			
		Abatement Package	6	6	6	-	-	-
NSW: Wollongong	4	*BAU*	3	3	3			
		Abatement Package	3	3	3	-	-	-
VIC: Melbourne	129	*BAU*	160	200	221			
		Abatement Package	183	197	218	-23	2	4
VIC: Latrobe Valley	3	*BAU*	3	4	4			
		Abatement Package	3	4	4	-	-	-

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

Table 5-4: Historical health burden attributable to O_3 in Australian airsheds (other airsheds)

Airshed	Number of attributable health outcomes (annual average 2010-2014)	
	Daily mortality, all-cause[(a)]	Emergency department visits, asthma (<15 years)
QLD: Brisbane (SEQ)	53	46
SA: Adelaide	25	63
WA: Perth	49	54
NT: Darwin	3	1

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

Overall, the Abatement Package scenario did not have a large effect on health outcomes. The abatement measures resulted in only small reductions in health outcomes in 2031 and 2040. In Melbourne in 2021, the year in which it was assumed that the abatement measures would be implemented, there was a predicted increase in the attributable health effects due to O_3. The reasons for this are unknown, but are likely to be due in part to the complex photochemistry between NO_2 and O_3. The Abatement Package included measures for both NO_2 and VOCs which are precursors of O_3. NO_2 also reacts to remove O_3 from the atmosphere. Decreasing NO_2 may have an unwanted impact of increasing O_3 concentrations.

5.3.3 Health outcomes for compliance with standards

It was shown in the air quality study that the implementation of the Abatement Package did not lead to the meeting of the proposed 1-hour standard for O_3 of 70 ppb standard. The 85 ppb and 100 ppb proposed standards were met with the Abatement Package.

The numbers of health outcomes associated with meeting the O_3 standards are given in Annexure C, Section C.2. Table 5-5 presents the numbers of health outcomes avoided in the NSW and Victoria airsheds by compliance with the proposed 1-hour standards for O_3 of 70, 85 and 100 ppb. The corresponding results for the other airsheds (20140-2014 average only) are given in Table 5-6.

NB: Although there was no Group 1 CRF for daily, all-cause mortality associated with 1-hour O_3, it is still appropriate to present the results of compliance with 1-hour standards based on the CRF for 8-hour O_3. In other words, although the health benefits are for compliance with a 1-hour standard, reducing maximum 1-hour concentrations also leads to reductions in other concentration metrics (in this case, 8-hour).

In some cases, the modelling suggested that health outcomes would increase in NSW for compliance with the 70 ppb and 85 ppb standards (i.e. the health outcomes avoided are negative).

Table 5-5: Health outcomes avoided if the proposed 1-hour standards for O_3 of 70 ppb, 85 ppb and 100 ppb are met (NSW and Victoria airsheds)

Airshed	1-hour O_3 standard of 70 ppb				1-hour O_3 standard of 85 ppb				1-hour O_3 standard of 100 ppb			
	Health outcomes avoided				Health outcomes avoided				Health outcomes avoided			
	Annual average 2010-2014	2021	2031	2040	Annual average 2010-2014	2021	2031	2040	Annual average 2010-2014	2021	2031	2040
Daily mortality all causes[(a)] (all ages)												
NSW: Sydney	105	28	36	44	52	10	15	19	9	- [(b)]	- [(b)]	- [(b)]
NSW: Newcastle	4	-66	-80	-82	- [(b)]	-81	-97	-106	- [(b)]	- [(b)]	- [(b)]	- [(b)]
NSW: Wollongong	4	-4	-4	-4	- [(b)]	- [(b)]	- [(b)]	- [(b)]	2	- [(b)]	- [(b)]	- [(b)]
VIC: Melbourne	19	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	6	- [(b)]	- [(b)]	- [(b)]
VIC: Latrobe Valley	0	-153	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]
Emergency Department visits asthma (<15 years)												
NSW: Sydney	37	21	26	35	19	7	8	11	4	- [(b)]	- [(b)]	- [(b)]
NSW: Newcastle	1	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]
NSW: Wollongong	2	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	1	- [(b)]	- [(b)]	- [(b)]
VIC: Melbourne	15	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	12	- [(b)]	- [(b)]	- [(b)]
VIC: Latrobe Valley	0	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]	- [(b)]

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(b) For these conditions, either the standard was already met in the airshed, or data were not available.

Table 5-6: Health outcomes avoided if the proposed 1-hour standards for O_3 of 70 ppb, 85 ppb and 100 ppb are met (other airsheds)

Airshed	1-hour O_3 standard of 70 ppb				1-hour O_3 standard of 85 ppb				1-hour O_3 standard of 100 ppb			
	Health outcomes avoided[(a)]				Health outcomes avoided[(a)]				Health outcomes avoided[(a)]			
	Annual average 2010-2014	2021	2031	2040	Annual average 2010-2014	2021	2031	2040	Annual average 2010-2014	2021	2031	2040
Daily mortality all causes[(b)] (all ages)												
QLD: Brisbane (SEQ)	-	n/a	n/a	n/a	-	n/a	n/a	n/a	-	n/a	n/a	n/a
SA: Adelaide	-	n/a	n/a	n/a	-	n/a	n/a	n/a	-	n/a	n/a	n/a
WA: Perth	-	n/a	n/a	n/a	-	n/a	n/a	n/a	-	n/a	n/a	n/a
NT: Darwin	-	n/a	n/a	n/a	-	n/a	n/a	n/a	-	n/a	n/a	n/a
Emergency Department visits asthma (<15 years)												
QLD: Brisbane (SEQ)	1	n/a	n/a	n/a	-	n/a	n/a	n/a	-	n/a	n/a	n/a
SA: Adelaide	-	n/a	n/a	n/a	-	n/a	n/a	n/a	-	n/a	n/a	n/a
WA: Perth	7	n/a	n/a	n/a	-	n/a	n/a	n/a	7	n/a	n/a	n/a
NT: Darwin	-	n/a	n/a	n/a	-	n/a	n/a	n/a	-	n/a	n/a	n/a

(a) 'n/a' = not available (not modelled)

(b) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

Note (1): For these conditions, either the standard was already met in the airshed, or data were not available.

5.3.4 Sensitivity tests

For O_3, the sensitivity testing examined the health outcomes in NSW and Victoria when the different CRF groups were used. In each state, and for each health outcome and period, the total number of health outcomes was summated across all airsheds for the BAU scenario. The results for NSW and Victoria are shown in Figures 5-1 and 5-2 respectively. It can be seen that:

- For all-cause mortality the Group 1 CRFs resulted in much lower estimates than the Group 3 CRFs. In addition, the removal of the O_3 cut-off in Group 2 had a significant effect on the estimates.
- For emergency department visits for asthma, all CRF Groups gave the same results.

As with NO_2, when considering any differences between the groups for O_3 it should be borne in mind that level of confidence for the Group 1 CRFs is higher than that for the Group 3 CRFs.

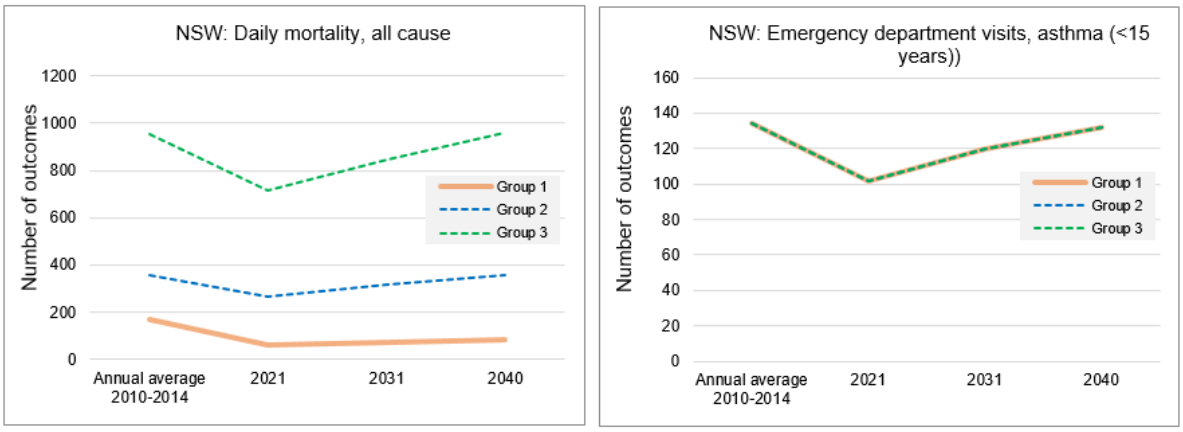

Figure 5-1: Comparison between O_3 health outcomes in NSW for different CRF groups

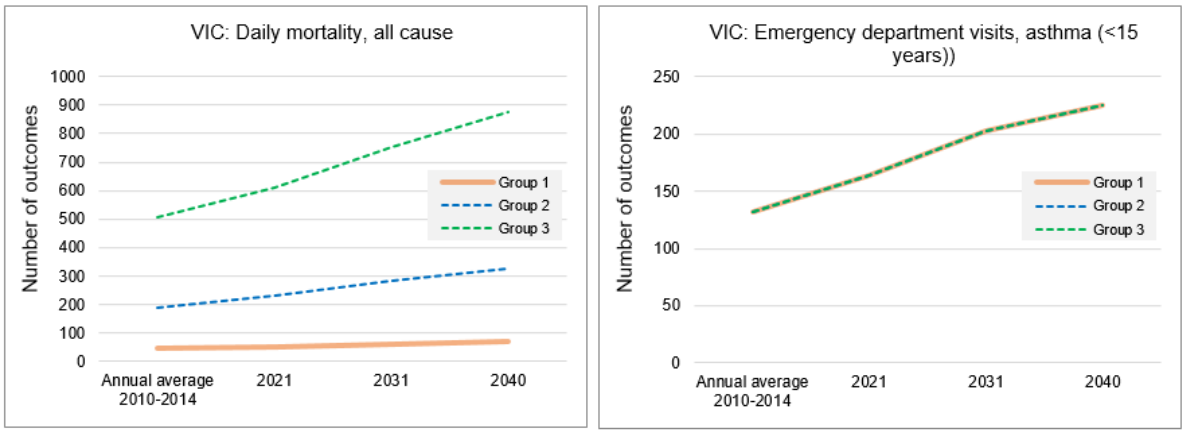

Figure 5-2: Comparison between O_3 health outcomes in Victoria for different CRF groups

5.4 Summary

5.4.1 Literature review

International studies have provided evidence that exposure to O_3 is causally linked to mortality and morbidity primarily for respiratory causes. There is some evidence that O_3 exposures are also associated with cardiovascular outcomes, but the evidence isn't as strong as for respiratory outcomes.
Long-term exposure to O_3 is linked to the incidence of asthma, and not just the exacerbation of existing asthma. O_3 was also found to be associated with a range of reproductive and developmental effects that had not previously been linked with O_3 exposure.

There is currently no convincing evidence of a threshold for short-term exposure to daily maximum 1 hour or 8-hour O_3 concentration, or of a non-linear relationship at low concentrations.

The evidence with regard to long-term effects of O_3 led the USEPA (2013) to consider the establishment of a long-term seasonal average for O_3. Other agencies, such as the WHO (2013b), have noted the emerging evidence around long-term effects, but concluded that the evidence is not sufficient to establish a guideline/standard. The studies are not sufficient at this time to enable determination of a robust CRF to use in a health risk assessment.

5.4.2 Exposure and risk characterisation

The HRA has shown that there is a substantial public health burden due to current O_3 levels in Australian cities.

Overall, the Abatement Package scenario did not have a large effect on health outcomes. The abatement measures resulted in only small reductions in health outcomes in 2031 and 2040. In Melbourne in 2021, the year in which it was assumed that the abatement measures would be implemented, there was a predicted increase in the attributable health effects due to O_3. This is likely to be due in part to the complex photochemistry of O_3 formation and removal.

The projected O_3 concentrations for Sydney, Wollongong, Newcastle, Melbourne and Latrobe Valley will exceed the 85 ppb and 70 ppb standards. If the standards could be met, there would be a public health benefit.

6 Uncertainties and limitations

There were a number of uncertainties in the HRA process. These included:

- Air quality data were not available for all airsheds and all years. No data were available for Tasmania, as the levels of O_3, NO_2 and SO_2 fall below the screening level for monitoring in the NEPM. Given that the levels are so low that they do not require monitoring, the associated health effects will also be low.
- The population projections used in the HRA were obtained from ABS. There is uncertainty in these projections that may lead to an over prediction or under prediction of the estimated health effects. This cannot be quantified.
- Dispersion modelling uncertainty. The models may have over-predicted or under-predicted the exposure data used in the HRA, and hence health outcomes. As discussed in Appendix A, the analysis of the predicted O_3 levels in Sydney suggested that the model was under - predicting. This means that the predicted health effects and avoided health effects if alternative standards were met may also have been underestimated.
- The use of current baseline health data for future projections. Changes in age distributions and health status over time may either increase or decrease the predicted risk which cannot be quantified. The projected ABS population data shows an ageing population with a greater percentage of people over 65 years of age in the future.
- The use of overseas CRFs to assess health outcomes may have resulted in uncertainty due to differences in population, air pollution levels and climatic conditions that may change the risk to the population in Australian cities.

7 References

Abbey D E, Nishino N, McDonnell W F, Burchette R J, Knutsen S F, Lawrence Beeson W and Yang J X (1999). Long-term inhalable particles and other air pollutants related to mortality in nonsmokers. *Am. J. Respir. Crit. Care Med*., Vol. 159(2), pp. 373-382.

Anderson H R, Atkinson R W, Bremner S A, Carrington J and Peacock J (2007). Quantitative systematic review of short term associations between ambient air pollution (particulate matter, ozone, nitrogen dioxide, sulphur dioxide and carbon monoxide), and mortality and morbidity. Division of Community Health Sciences, St George's, University of London, London Available at: https://www.gov.uk/government/publications/quantitativesystematic-review-of-short-term-associations-between-ambient-air-pollutionparticulate-matter-ozone-nitrogen-dioxide-sulphur-dioxide-and-carbon-monoxideand-mortality-and-morbidity.

Ballester F, Rodriguez P, Iniguez C, Saez M, Daponte A, Galan I, Taracido M, Arribas F, Bellido J, Cirarda F B, Canada A, Guillen J J, Guillen-Grima F, Lopez E, Perez-Hoyos S, Lertxundi A and Toro S (2006). Air pollution and cardiovascular admisisons association in Spain: results within the EMECAS project. *J. Epidemiol. Community Health*, Vo. 60, pp. 328-336.

Barnett A G, Williams G M, Schwartz J, Best T L, Neller A H, Petroeschevsky A L and Simpson R W (2006). The Effects Of Air Pollution On Hospitalizations For Cardiovascular Disease In Elderly People In Australian And New Zealand Cities. *Environmental Health Perspectives*, Vol. 114, pp. 1018-1023.

Barnett A G, Williams G M, Schwartz J, Neller A H, Best T L, Petroeschevsky A and Simpson R W (2005). Air Pollution And Child Respiratory Health: A Case-Crossover Study In Australia And New Zealand. *American Journal of Respiratory & Critical Care Medicine*, Vol. 171, pp. 1272-8.

Barone-Adesi F, Dent J E, Dajnak D, Beevers S, Anderson H R, Kelly F J, Cook D G and Whincup P (2015). Long-Term Exposure to Primary Traffic Pollutants and Lung Function in Children: Cross Sectional Study and Meta-Analysis. *PLOS ONE 10(11); e0142565*.

Beelen R, Hoek G, van den Brandt P A, Goldbohm R A, Fischer P, Schouten L J, Armstrong B, and Brunekreef B (2008a). Long-term exposure to traffic-related air pollution and lung cancer risk. *Epidemiology*, Vol. 19(5), pp. 702-10.

Beelen R, Hoek G, van den Brandt P A, Goldbohm R A, Fischer P, Schouten L J, Jerrett M, Hughes E, Armstrong B and Brunekreef B (2008b). Long-term effects of traffic-related air pollution on mortality in a Dutch cohort (NLCS-AIR study). *Environ. Health Perspect*., Vol.116(2), pp.196-202.

Biggeri A, Baccini M, Bellini P and Terracini B (2005). Meta-analysis of the Italian studies of short-term effects of air pollution (MISA), 1990-1999. *Int. J. Occup. Environ. Health*. Vol. 11(1), pp. 107-22.

Burnett R T, Cakmak S, Brook J R (1998a). The effect of the urban ambient air pollution mix on daily mortality rates in 11 Canadian cities. *Can. J. Public Health*. Vol. 89(3), pp.152-6.

Burnett R T, Cakmak S, Raizenne M E, Stieb D, Vincent R, Krewski D, Brook JR, Philips O and Ozkaynak H (1998). The association between ambient carbon monoxide levels and daily mortality in Toronto, Canada. *J. Air Waste Manag. Assoc*., Vol. 48(8), pp. 689-700.

Burnett R T, Brook J, Dann T, Delocla C, Philips O, Cakmak S, Vincent R, Goldberg M S and Krewski D (2000). Association between particulate- and gas-phase components of urban air pollution and daily mortality in eight Canadian cities. *Inhal. Toxicol.,* Vol. 12, Suppl 4, pp. 15-39.

Burnett R T, Stieb D, Brook J R, Cakmak S, Dales R, Raizenne M, Vincent R and Dann T (2004). Associations between short-term changes in nitrogen dioxide and mortality in Canadian cities. *Arch. Environ. Health,* Vol. 59(5), pp. 228-36.

Castellsague J, Sunyer J, Sáez M and Antó J M (1995). Short-term association between air pollution and emergency room visits for asthma in Barcelona. *Thorax*, Vol. 50(10), pp. 1051-6.

Cesaroni G, Badaloni C, Gariazzo C, Stafoggia M, Sozzi R, Davoli M and Forastiere F (2013). Long-Term Exposure to Urban Air Pollution and Mortality in a Cohort of More than a Million Adults in Rome. *Environmental Health Perspectives*, Vol. 121(3), pp. 324-331.

Chen K, Glonek G, Hansen A, Williams S, Tuke J, Salter A and Bi P (2016). The effects of air pollution on asthma hospital admissions in Adelaide, South Australia, 2003-2013: time-series and case-crossover analyses. *Clin. Exp. Allergy*, Vol. 46(11), pp.1416-1430.

COMEAP (2014). Considering the Evidence for the Effects of Nitrogen Dioxide on Health. Committee on the Medical Effects of Air Pollution, Public Health England, London.

COMEAP (2015). Quantification of Mortality and Hospital Admissions Associated with Ground Level Ozone. Committee on the Medical Effects of Air Pollution, Public Health England, London.

Defra (2015). Valuing impacts on air quality: Updates in valuing changes in emissions of oxides of nitrogen (NO_X) and concentrations of nitrogen dioxide (NO_2). Department of the Environment, Food and Rural Affairs, London.

Dockery DW , Pope C A 3rd, Xu X, Spengler J D, Ware J H, Fay M E, Ferris B G Jr and Speizer F E (1993). An association between air pollution and mortality in six U.S. cities. *N. Engl. J. Med.*, Vol. 329(24), pp. 1753-9.

Dominici F, McDermott A, Zeger S L, Samet J M (2003a). Airborne particulate matter and mortality: timescale effects in four US cities. *Am. J. Epidemiol.*, Vol. 157(12), pp.1055-65.

Dominici F, McDermott A, Zeger S L and Samet J M (2003b). National maps of the effects of particulate matter on mortality: exploring geographical variation. Environ. Health Perspect., Vol. 111(1), pp. 39-44.

Dugandzic R, Dodds L, Stieb D and Smith-Doiron M (2006). The association between low level exposures to ambient air pollution and term low birth weight: a retrospective cohort study. *Environ. Health.* Feb 17;5:3.

Elliott P, Shaddick G, Wakefield J C, de Hoogh C and Briggs D J (2007). Long-term associations of outdoor air pollution with mortality in Great Britain. *Thorax*. Vol. 62(12), pp.1088-94.

EPA Victoria (2001). Ambient Air Pollution and Daily Hospital Admissions in Melbourne 1994-1997, EPA Publication 789.

EPHC (2005). Multicity Mortality and Morbidity Study, available at www.ephc.gov.au.

Erbas B and Hyndman R J (2005). Sensitivity of the estimated air pollution-respiratory admissions relationship to statistical model choice. *Int. J. Environ. Health Res.,* Vol. 15(6), pp. 437-48.

Erbas B, Kelly AM, Physick B, Code C and Edwards M (2005). Air pollution and childhood asthma emergency hospital admissions: estimating intra-city regional variations. *Int. J. Environ. Health Res.*, Vol. 15(1), pp.11-20.

Faustini A, Rapp R and Forastieri F (2014). Nitrogen Dioxide and Mortality: Review and Meta-Analysis of Long-Term Studies. *European Respiratory Journal*, Vol. 44(3), pp. 744-53.

Filleul L, Rondeau V, Vandentorren S, Le Moual N, Cantagrel A, Annesi-Maesano I, Charpin D, Declercq C, Neukirch F, Paris C, Vervloet D, Brochard P, Tessier JF, Kauffmann F and Baldi I (2005). Twenty five year mortality and air pollution: results from the French PAARC survey. *Occup. Environ. Med.* Vol. 62(7), pp. 453-60.

Frangos J and DiMarco P (2013). 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-Rev0. Golder Associates, Richmond, Victoria.

Hamra G B, Laden F, Cohen A J, Raaschou-Nielsen O, Brauer M and Loomis D (2015). Lung Cancer and Exposure to Nitrogen Dioxide and Traffic: A Systematic Review and Meta-Analysis, *Environ. Health Perspect*., Vol. 123, pp. 1107-1112.

Hansen C, Neller A, Williams G and Simpson R (2006). Maternal exposure to low levels of ambient air pollution and preterm birth in Brisbane, Australia. *BJOG*. 2006 Aug;113(8), pp. 935-41.

Hansen C, Neller A, Williams G and Simpson R (2007). Low levels of ambient air pollution during pregnancy and fetal growth among term neonates in Brisbane, Australia. *Environ. Res*. Vol. 103(3), pp. 383-9.

Henry R L, Bridgman H A, Wlodarczyk J, Abramson R, Adler J A and Hensley M J (1991). Asthma in the vicinity of power stations: II. Outdoor air quality and symptoms. *Pediatr. Pulmonol*.,, Vol. 11(2), pp. 134-40.

Hinwood A L, de Klerk N, Rodriguez C, Runnion T, Jacoby P, Landau L, Murray F, Feldwick M and Spickett J (2004). Changes in daily air pollution and mortality in Perth: A case crossover study, *Environmental Health*, Vol. 4(3), pp. 13-22.

Hoek G, Krishnan R M, Beelen R, Peters A, Ostro B, Brunekreef B and Kaufman J D (2013). Long-Term Air Pollution Exposure and Cardio-Respiratory Mortality: A Review. *Environmental Health*, 12:43.

Jalaludin B, O'Toole B I, Leeder S R (2004). Acute effects of urban ambient air pollution on respiratory symptoms, asthma medication use, and doctor visits for asthma in a cohort of Australian children. *Environ. Res*. May; 95(1), pp. 32-42.

Jalaludin B, Morgan G, Lincoln D, Sheppeard V, Simpson R and Corbett S (2006). Associations between ambient air pollution and daily emergency department attendances for cardiovascular disease in the elderly (65+ years), Sydney, Australia. *J. Expo. Sci. Environ. Epidemiol*., Vol. 16(3), pp. 225-37.

Jalaludin B, Mannes T, Morgan G, Lincoln D, Sheppeard V and Corbett S (2007). Impact of ambient air pollution on gestational age is modified by season in Sydney, Australia. *Environ Health*. Jun 7;6:16.

Jalaludin B, Khalaj B, Sheppeard V and Morgan G (2008). Air pollution and ED visits for asthma in Australian children: a case-crossover analysis. *Int. Arch. Occup. Environ. Health*, Vol. 81(8), pp. 967-74.

Jalaludin B and Cowie C (2012). Health Risk Assessment – Preliminary Work to Identify Concentration Response Functions for Selected Ambient Air Pollutants. www.nepc.gov.au/nepms/ambient-air-quality/proposed-variation/consultation.

Jerrett M, Burnett RT, Willis A, Krewski D, Goldberg MS, DeLuca P and Finkelstein N (2003). Spatial analysis of the air pollution-mortality relationship in the context of ecologic confounders. *J. Toxicol. Environ. Health A*. Aug 22-Oct 10;66(16-19), pp. 1735-77.

Katsouyanni K, Touloumi G, Samoli E, Petasakis Y, Analitis A, Le Tertre A, Rossi G, et al. (2003). Sensitivity analysis of various models of short-term effects of ambient particles on total mortality in 29 cities in APHEA2. In special report: Revised analyses of time-series studies of air pollution and health. Special Report 886. Health Effects Institute, Boston MA.

Katsouyanni K, Zmirou D, Spix C, Sunyer J, Schouten JP, Ponka A, Anderson H R, Le Moullec Y, Wojtyniak B, Vigotti M A, Bacharova L and Schwartz J (1997). Short-term effects of air pollution on health: a European approach using epidemiologic time series data. The APHEA Project. Air Pollution Health Effects - A European Approach. *Public Health Rev*., 1997;25(1), pp. 7-18; discussion 19-28.

Leem J H, Kaplan BM, Shim YK, Pohl H R, Gotway C A, Bullard S M, Rogers J F, Smith M M, Tylenda C A (2006). Exposures to air pollutants during pregnancy and preterm delivery. *Environ Health Perspect*. 2006 Jun;114(6), pp. 905-10.

Lewis P R, Hensley M J, Wlodarczyk J, Toneguzzi R C, Westley-Wise V J, Dunn T and Calvert D (1998). Outdoor air pollution and children's respiratory symptoms in the steel cities of New South Wales. *Med. J. Aust.*, 1998 Nov 2;169(9), pp. 459-63.

Lipfert F W, Morris S C and Wyzga R E (2000a). Daily mortality in the Philadelphia metropolitan area and size-classified particulate matter. *J. Air Waste Manag. Assoc.*, Aug;50(8), pp.1501-13.

Lipfert F W, Zhang J and Wyzga R E (2000b). Infant mortality and air pollution: a comprehensive analysis of U.S. data for 1990. J. Air Waste Manag Assoc. 2000 Aug;50(8), pp. 1350-66.

Mannes T, Jalaludin B, Morgan G, Lincoln D, Sheppeard V and Corbett S (2005). Impact of ambient air pollution on birth weight in Sydney, Australia. *Occup. Environ. Med*., 2005 Aug;62(8), pp. 524-30.

Morgan G, Corbett S, Wlodarczyk J and Lewis P (1998a). Air pollution and daily mortality in Sydney, Australia, 1989 through 1993. *Am. J. Public. Health.* May;88(5), pp. 759-64.

Morgan G, Corbett S and Wlodarczyk J (1998b). Air pollution and hospital admissions in Sydney, Australia, 1990 to 1994. Am. J. Public Health. Dec; 88(12), pp. 1761-6.

Nafstad P, Håheim L L, Wisløff T, Gram F, Oftedal B, Holme I, Hjermann I and Leren P (2004). Urban air pollution and mortality in a cohort of Norwegian men. *Environ. Health Perspect*., Apr;112(5), pp. 610-5.

NEPC (2010). Review of the National Environment Protection (ambient air quality) measure. Discussion paper. Air quality standards. Prepared for the National Environment Protection Council. http://www.ephc.gov.au/sites/default/files/AAQ_DiscPpr__Review_of_the_AAQ_NEPM_Discussion_Paper_AQ_Standards_Final_201007.pdf.

NEPC (2011a). National Environment Protection (Ambient Air Quality) Measure Review. National Environment Protection Council Service Corporation, Level 5 81 Flinders Street, Adelaide, South Australia. http://www.ephc.gov.au/sites/default/files/AAQ%20AAQ NEPM%20review%20report_0.pdf

NEPC (2011b). Methodology for Setting Air Quality Standards in Australia, Part A, February 2011. National Environment Protection Council, Canberra.

NHMRC (2006). Ambient Air Quality Standard Setting: An Approach to Health-Base Hazard Assessment.

OEHHA (2000). Sulfur dioxide: evaluation of current California air quality standards with respect to protection of children. Available at oehha.ca.gov/media/downloads/crnr/oehhaso2.pdf

OEHHA (2011). Evidence on the developmental and reproductive toxicity of Sulfur Dioxide, Reproductive and Cancer Hazard Assessment Branch Office of Environmental Health Hazard Assessment, California Environmental Protection Agency.

Peters J, Hedley A J, Wong C M, Lam T H, Ong S G, Liu J and Spiegelhalter D J (1996). Effects of an ambient air pollution intervention and environmental tobacco smoke on children's respiratory health in Hong Kong. *Int. J. Epidemiol*, Aug;25(4), pp. 821-8.

Petroeschevsky A, Simpson RW, Thalib L and Rutherford S (2001). Associations between outdoor air pollution and hospital admissions in Brisbane, Australia. *,* 2001 Jan-Feb;56(1), pp. 37-52.

Pope CA 3rd, Thun M J, Namboodiri M M, Dockery D W, Evans J S, Speizer F E and Heath CW Jr. (1995). Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am. J. Respir. Crit. Care Med*., Mar;151(3 Pt 1), pp.669-74.

Pope, C A III, Burnett R T, Thun M J, Calle E E, Krewski D, Ito K and Thurston G D (2002). Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution. *Journal of the American Medical Association,* Vol. 287, pp. 1132-1141.

Rodriguez C, Tonkin R, Heyworth J, Kusel M, De Klerk N, Sly P D, Franklin P, Runnion T, Blockley A, Landau L and Hinwood A L (2007). The relationship between outdoor air quality and respiratory symptoms in young children. *Int. J. Environ. Health Res*., Vol. 17(5), pp. 351-60.

Sagiv S K, Mendola P, Loomis D, Herring A H, Neas L M, Savitz D A and Poole C (2005). A time-series analysis of air pollution and preterm birth in Pennsylvania, 1997-2001. *Environ. Health Perspect.*, Vol. 113(5), pp. 602-6.

Samet J M, Dominici F, Curriero F C, Coursac I and Zeger S L (2000a). Fine particulate air pollution and mortality in 20 U.S. cities, 1987-1994. *N. Engl. J. Med*., 2000 Dec 14;343(24), pp.1742-9.

Samet J M, Zeger S L, Dominici F, Curriero F, Coursac I, Dockery D W, Schwartz J and Zanobetti A (2000b). The National Morbidity, Mortality, and Air Pollution Study. Part II: Morbidity and mortality from air pollution in the United States. *Res. Rep. Health Eff. Inst.*, Jun;94(Pt 2), pp. 5-70; discussion pp. 71-9.

Samoli E, Schwartz J, Wojtyniak B, Touloumi G, Spix C, Balducci F, Medina S, Rossi G, Sunyer J, Bacharova L, Anderson H R and Katsouyanni K (2001). Investigating regional differences in short-term effects of air pollution on daily mortality in the APHEA project: a sensitivity analysis for controlling long-term trends and seasonality. *Environ Health Perspect*., Vol. 109(4), pp. 349-53.

Samoli E, Touloumi G, Zanobetti A, Le Tertre A, Schindler C, Atkinson R, Vonk J, Rossi G, Saez M, Rabczenko D, Schwartz J and Katsouyanni K (2003). Investigating the dose-response relation between air pollution and total mortality in the APHEA-2 multicity project. *Occup Environ Med.*, Vol. 60(12), pp. 977-82.

SCEW (2012), Australian Child Health and Air Pollution Study (ACHAPS). Available at www.scew.gov.au/air.

Schouten J P, Vonk J M and de Graaf A J (1996) .Short term effects of air pollution on emergency hospital admissions for respiratory disease: results of the APHEA project in two major cities in The Netherlands, 1977-89. *Epidemiol. Community Health.,* Apr;50, Suppl 1:s22-9.

Simpson R W, Williams G, Petroeschevsky A, Morgan G and Rutherford S. (1997). Associations between outdoor air pollution and daily mortality in Brisbane, Australia. *Arch. Environ. Health*. Vol. 52(6), pp. 442-54.

Simpson R, Denison L, Petroeschevsky A, Thalib L and Williams G (2001). Effects of ambient particle pollution on daily mortality in Melbourne, 1991-1996. *J. Expo. Anal. Environ. Epidemiol*., 2000 Sep-Oct;10(5), pp. 488-96.

Simpson R, Williams G, Petroeschevsky A, Best T, Morgan G, Denison L, Hinwood A, Neville G and Neller A (2005a). The short-term effects of air pollution on daily mortality in four Australian cities. *Aust. NZ J. Public Health,* Vol. 29(3), pp. 205-12.

Simpson R, Williams G, Petroeschevsky A, Best T, Morgan G, Denison L, Hinwood A and Neville G (2005b). The short-term effects of air pollution on hospital admissions in four Australian cities. *Aust. NZ J. Public Health,* Vol. 29(3), pp. 213-21.

Simpson R, Williams G, Petroeschevsky A, Best T, Morgan G, Denison L, Hinwood A, Neville G and Neller A (2005). The Short-Term Effects Of Air Pollution On Daily Mortality In Four Australian Cities. *Australian & New Zealand Journal of Public Health*, Vol. 29, pp. 205-12.

Spix C, Anderson H R, Schwartz J, Vigotti M A, LeTertre A, Vonk J M, Touloumi G, Balducci F, Piekarski T, Bacharova L, Tobias A, Pönkä A and Katsouyanni K (1998). Short-term effects of air pollution on hospital admissions of respiratory diseases in Europe: a quantitative summary of APHEA study results. Air Pollution and Health: a European Approach. *Arch. Environ. Health*. Vol. 53(1), pp. 54-64.

Sram R J, Binková B, Dejmek J and Bobak M (2005). Ambient air pollution and pregnancy outcomes: a review of the literature. *Environ. Health Perspect.*, Vol. 113(4), pp. 375-82.

Stieb D M, Judek S and Burnett RT (2002). Meta-analysis of time-series studies of air pollution and mortality: effects of gases and particles and the influence of cause of death, age, and season. *J. Air Waste Manag. Assoc*., Vol. 52(4), pp. 470-84.

Stieb D M, Judek S and Burnett RT (2003). Meta-analysis of time-series studies of air pollution and mortality: update in relation to the use of generalized additive models. *J. Air Waste Manag. Assoc*., Vol. 53(3), pp. 258-61.

Tenías J M, Ballester F and Rivera M L (1998). Association between hospital emergency visits for asthma and air pollution in Valencia, Spain. *Occup. Environ. Med*., Vol. 55(8), pp. 541-7.

USEPA (2008). Integrated Science Assessment Sulfur Dioxide. U.S. Environmental Protection Agency, Washington, DC.

USEPA (2006). Air Quality Criteria for Ozone and Related Photochemical Oxidants (Final Report). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-05/004aF-cF.

USEPA (2016). Integrated Science Assessment of Oxides of Nitrogen. U.S. Environmental Protection Agency, Washington, DC.

USEPA (2013). Integrated Science Assessment of Ozone. U.S. Environmental Protection Agency, Washington, DC. First External Draft.

Vigotti M A, Rossi G, Bisanti L, Zanobetti A and Schwartz J (1996). Short term effects of urban air pollution on respiratory health in Milan, Italy, 1980-89. *J. Epidemiol. Community Health*., Vol. 50, Suppl. 1: s71-5.

Walters S, Griffiths R K and Ayres J G (1994). Temporal association between hospital admissions for asthma in Birmingham and ambient levels of sulphur dioxide and smoke. *Thorax*, Vol. 49(2), pp. 133-40.

WHO (2006). Air Quality Guidelines. Global Update. 2005. Particulate Matter, Ozone, Nitrogen Dioxide and Sulfur Dioxide. World Health Organization Regional Office for Europe, Copenhagen, Denmark.

WHO (2008). Health Risks of Ozone from Long Range Transboundary of Air Pollution. World Health Organization Regional Office for Europe, Copenhagen, Denmark.

WHO (2013a). Health risks of air pollution in Europe – HRAPIE project. Recommendations for concentration–response functions for cost–benefit analysis of particulate matter, ozone and nitrogen dioxide. World Health Organization Regional Office for Europe, Copenhagen, Denmark.

WHO (2013b). Review of Evidence of Health Effects of Air Pollution Project: REVIHAPP. World Health Organization Regional Office for Europe, Copenhagen, Denmark.

Wong C M, Lam T H, Peters J, Hedley A J, Ong S G, Tam A Y, Liu J and Spiegelhalter D J (1998). Comparison between two districts of the effects of an air pollution intervention on bronchial responsiveness in primary school children in Hong Kong. *J. Epidemiol. Community Health*., Vol. 52(9), pp. 571-8.

Wong C M, Atkinson R W, Anderson H R, Hedley A J, Ma S, Chau PY and Lam T H (2002a). A tale of two cities: effects of air pollution on hospital admissions in Hong Kong and London compared. *Environ. Health Perspect*., Vol. 110(1), pp. 67-77.

Wong T W, Tam W S, Yu T S and Wong A H (2002b). Associations between daily mortalities from respiratory and cardiovascular diseases and air pollution in Hong Kong, China. *Occup Environ Med*. Vol. 59(1), pp. 30-35. Erratum in: Occup. Environ. Med., 2002 Sep;59(9): p. 650.

Yang M and Chou S-Y (2017). The Impact of Environmental Regulation on Fetal Health: Evidence from the Shutdown of a Coal-Fired Power Plant Located Upwind of New Jersey. *Journal of Environmental Economics and Management*. In press.

Annexure A: Detailed risk characterisation for SO_2

A.1 Historical and projected health burden

Table A-1: Historical and projected health burden attributable to 1-hour SO_2 (NSW)

Health outcome	Historical					Projected							
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
NSW: Sydney												
Hospital admissions for respiratory disease (65+ years)	273 (97-457)	231 (82-387)	237 (84-398)	332 (118-557)	391 (139-656)	*BAU*	701 (250-1176)	893 (318-1498)	1102 (393-1850)			
						Abatement Package	589 (210-987)	511 (182-857)	338 (120-566)	113 (40-189)	382 (136-641)	765 (273-1284)
NSW: Newcastle												
Hospital admissions for respiratory disease (65+ years)	66 (23-111)	66 (23-110)	71 (25-119)	74 (26-124)	98 (34-163)	*BAU*	203 (72-340)	246 (88-413)	284 (101-477)			
						Abatement Package	168 (59-281)	137 (48-229)	70 (24-116)	35 (13-59)	110 (39-184)	215 (77-360)
NSW: Wollongong												
Hospital admissions for respiratory disease (65+ years)	30 (10-49)	28 (9-46)	22 (7-37)	29 (10-47)	40 (14-67)	*BAU*	46 (16-77)	54 (19-90)	62 (22-104)			
						Abatement Package	38 (13-63)	30 (10-50)	14 (5-24)	8 (3-14)	24 (9-40)	48 (17-80)

Table A-2: Historical and projected health burden attributable to 1-hour SO_2 (Victoria)

Health outcome	Historical					Projected						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
VIC: Melbourne												
Hospital admissions for respiratory disease (65+ years)	695 (248-1166)	472 (168-792)	645 (230-1083)	599 (213-1004)	907 (323-1522)	*BAU*	1390 (496-2333)	1594 (569-2675)	1807 (645-3032)			
						Abatement Package	921 (329-1546)	1119 (399-1878)	923 (329-1549)	469 (167-787)	475 (170-797)	884 (316-1483)
VIC: Latrobe Valley												
Hospital admissions for respiratory disease (65+ years)	13 (4-22)	12 (4-20)	13 (4-22)	18 (6-29)	18 (6-30)	*BAU*	15 (5-25)	16 (5-26)	17 (6-29)			
						Abatement Package	11 (3-18)	12 (4-19)	10 (3-16)	4 (2-7)	4 (1-7)	7 (3-12)

Table A-3: Historical and projected health burden attributable to 1-hour SO_2 (other airsheds)

Health outcome	Historical					Projected						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
QLD: Brisbane (SEQ)												
Hospital admissions for respiratory disease (65+ years)	183 (65-308)	181 (64-303)	136 (48-227)	126 (44-210)	112 (39-187)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
SA: Adelaide												
Hospital admissions for respiratory disease (65+ years)	44 (15-74)	50 (17-83)	47 (16-79)	41 (14-68)	36 (12-60)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
WA: Perth												
Hospital admissions for respiratory disease (65+ years)	256 (91-430)	236 (84-396)	235 (83-393)	336 (120-564)	330 (117-554)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
NT: Darwin												
Hospital admissions for respiratory disease (65+ years)	No data	- -	- -	- -	- -	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a

Table A-4: Historical and projected health burden attributable to 24-hour SO_2 (NSW)

Health outcome	Historical					Projected						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
NSW: Sydney												
Daily mortality, all-cause[(a)]	21 (16-25)	17 (13-20)	19 (15-23)	21 (16-25)	18 (14-21)	*BAU*	38 (30-45)	47 (37-56)	59 (46-70)			
						Abatement Package	32 (25-38)	29 (22-34)	18 (14-21)	6 (5-7)	18 (14-21)	40 (32-49)
Emergency department visits for asthma (<15 years)	53 (23-79)	44 (19-66)	48 (20-71)	53 (23-79)	46 (19-68)	*BAU*	96 (42-144)	118 (51-177)	143 (62-214)			
						Abatement Package	82 (35-122)	73 (31-109)	44 (19-66)	15 (6-22)	45 (20-68)	99 (43-148)
NSW: Newcastle												
Daily mortality, all-cause[(a)]	7 (5-8)	6 (5-7)	7 (5-8)	6 (4-6)	6 (4-6)	*BAU*	17 (13-20)	21 (16-25)	25 (19-29)			
						Abatement Package	14 (11-17)	11 (9-13)	5 (4-6)	3 (2-4)	9 (7-11)	20 (16-24)
Emergency department visits for asthma (<15 years)	10 (4-14)	9 (4-14)	10 (4-14)	8 (3-11)	8 (3-11)	*BAU*	24 (10-36)	29 (12-42)	34 (14-50)			
						Abatement Package	20 (8-30)	16 (6-23)	7 (3-10)	4 (2-6)	13 (6-19)	27 (12-40)
NSW: Wollongong												
Daily mortality, all-cause[(a)]	2 (1-2)	2 (1-2)	1 (1-1)	2 (1-2)	2 (1-2)	*BAU*	3 (2-3)	4 (2-4)	3 (2-3)			
						Abatement Package	3 (2-3)	2 (1-2)	1 (0-1)	- (0-1)	1 (1-2)	2 (1-2)
Emergency department visits for asthma (<15 years)	4 (1-5)	3 (1-4)	2 (0-3)	3 (1-4)	3 (1-4)	*BAU*	5 (2-7)	6 (2-8)	5 (2-7)			
						Abatement Package	4 (1-6)	4 (1-5)	2 (0-3)	1 (0-1)	2 (1-3)	3 (1-4)

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

Table A-5: Historical and projected health burden attributable to 24-hour SO_2 (Victoria)

Health outcome	Historical					Projected						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
VIC: Melbourne												
Daily mortality, all-cause[(a)]	53 (41-63)	40 (31-48)	45 (35-54)	32 (25-38)	39 (30-47)	*BAU*	93 (73-112)	107 (84-129)	131 (104-158)			
						Abatement Package	62 (49-75)	76 (60-91)	64 (50-77)	31 (24-37)	31 (25-38)	68 (54-82)
Emergency department visits for asthma (<15 years)	235 (102-351)	177 (77-264)	195 (85-292)	139 (60-208)	171 (74-255)	*BAU*	423 (124-425)	489 (213-732)	571 (249-856)			
						Abatement Package	283 (184-634)	347 (213-732)	278 (249-856)	139 (61-209)	142 (62-213)	294 (128-441)
VIC: Latrobe Valley												
Daily mortality, all-cause[(a)]	2 (1-1)	2 (1-1)	2 (1-2)	1 (0-1)	1 (0-1)	*BAU*	1 (1-1)	1 (1-1)	2 (1-1)			
						Abatement Package	1 (0-1)	1 (0-1)	1 (0-1)	- -	- -	1 (1-1)
Emergency department visits for asthma (<15 years)	6 (2-9)	6 (2-9)	7 (3-10)	4 (1-5)	4 (1-5)	*BAU*	5 (2-7)	6 (2-8)	6 (2-9)			
						Abatement Package	4 (1-5)	4 (1-6)	4 (1-5)	2 (1-2)	1 (1-2)	3 (1-4)

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

Table A-6: Historical and projected health burden attributable to 24-hour SO_2 (other airsheds)

Health outcome	Historical					Projected						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
QLD: Brisbane (SEQ)												
Daily mortality, all-cause[(a)]	16 (12-19)	21 (16-25)	12 (9-13)	21 (16-25)	15 (11-17)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits for asthma (<15 years)	36 (15-53)	46 (20-69)	25 (10-36)	44 (19-66)	31 (13-47)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
SA: Adelaide												
Daily mortality, all-cause[(a)]	4 (2-4)	2 (1-2)	2 (1-2)	3 (2-3)	1 (1-1)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits for asthma (<15 years)	15 (6-22)	8 (3-11)	9 (3-13)	11 (4-15)	5 (2-7)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
WA: Perth												
Daily mortality, all-cause[(a)]	12 (9-14)	15 (11-17)	15 (12-18)	23 (18-28)	25 (19-29)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits for asthma (<15 years)	35 (15-52)	44 (19-65)	44 (19-65)	67 (29-100)	71 (31-106)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
NT: Darwin												
Daily mortality, all-cause[(a)]	No data	- -	- -	- -	- -	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits for asthma (<15 years)	No data	1 (0-1)	1 -	1 -	- -	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

A.2 Health outcomes - compliance with standards

Table A-7: Health outcomes for compliance with proposed 24-hour standard for SO_2 of 7 ppb

Health outcome	Historical					Projected		
	Number of health outcomes							
	2010	2011	2012	2013	2014	2021	2031	2040
NSW: Sydney								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Hospital admissions respiratory (65+ years)	-	-	-	-	-	-	-	-
Emergency Dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
NSW: Newcastle								
Daily mortality all causes[(a)] (all ages)	6 (4-7)	5 (4-6)	-	-	6 (4-7)	7 (5-8)	8 (6-9)	8 (6-9)
Hospital admissions respiratory (65+ years)	65 (23-108)	67 (23-112)	-	-	100 (35-168)	203 (72-340)	246 (88-413)	284 (101-477)
Emergency Dept. visits asthma (<15 years)	9 (3-13)	7 (3-11)	-	-	9 (3-13)	10 (4-15)	11 (4-15)	11 (4-16)
NSW: Wollongong								
Daily mortality all causes[(a)] (all ages)	1 (1-1)	1 (0-1)	1 (1-1)	2 (1-1)	-	2 (1-2)	3 (2-3)	2 (1-2)
Hospital admissions respiratory (65+ years)	29 (10-49)	26 (9-44)	21 (7-36)	28 (9-46)	-	46 (16-77)	54 (19-90)	52 (18-87)
Emergency Dept. visits asthma (<15 years)	2 (0-3)	2 (0-2)	2 (0-3)	3 (1-4)	-	4 (1-5)	5 (2-7)	4 (1-5)
VIC: Melbourne								
Daily mortality all causes[(a)] (all ages)	14 (10-16)	24 (18-28)	17 (13-20)	23 (18-27)	21 (16-24)	53 (42-64)	65 (51-77)	79 (62-95)
Hospital admissions respiratory (65+ years)	148 (52-248)	240 (85-402)	243 (86-408)	456 (162-765)	466 (166-781)	794 (283-1333)	960 (342-1611)	1088 (388-1826)
Emergency Dept. visits asthma (<15 years)	62 (26-92)	105 (45-157)	75 (32-112)	101 (44-151)	90 (39-135)	242 (105-362)	294 (128-441)	344 (150-516)
VIC: Latrobe Valley								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	1 -	1 -	1 (0-1)	-
Hospital admissions respiratory (65+ years)	-	-	-	-	17 (6-28)	15 (5-25)	16 (5-26)	-
Emergency Dept. visits asthma (<15 years)	-	-	-	-	2 (1-3)	3 (1-4)	4 (1-6)	-
QLD: Brisbane (SEQ)								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Hospital admissions respiratory (65+ years)	-	-	-	-	-	-	-	-
Emergency Dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
SA: Adelaide								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Hospital admissions respiratory (65+ years)	-	-	-	-	-	-	-	-
Emergency Dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
WA: Perth								
Daily mortality all causes (all ages)	-	-	13 (10-15)	-	17 (13-20)	-	-	-
Hospital admissions respiratory (65+ years)	-	-	230 (82-386)	-	326 (116-546)	-	-	-
Emergency Dept. visits asthma (<15 years)	-	-	38 (16-56)	-	49 (21-73)	-	-	-
NT: Darwin								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Hospital admissions respiratory (65+ years)	-	-	-	-	-	-	-	-
Emergency Dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

Table A-8: Health outcomes for compliance with proposed 24-hour standard for SO_2 of 20 ppb

Health outcome	Historical					Projected		
	Number of health outcomes avoided							
	2010	2011	2012	2013	2014	2021	2031	2040
NSW: Sydney								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Hospital admissions respiratory (65+ years)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
NSW: Newcastle								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	21 (16-24)	22 (17-26)
Hospital admissions respiratory (65+ years)	-	-	-	-	-	-	246 (88-413)	284 (101-477)
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	28 (12-42)	30 (13-44)
NSW: Wollongong								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Hospital admissions respiratory (65+ years)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
VIC: Melbourne								
Daily mortality all causes[(a)] (all ages)	40 (31-47)	-	-	-	-	-	-	-
Hospital admissions respiratory (65+ years)	425 (151-714)	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	177 (77-265)	-	-	-	-	-	-	-
VIC: Latrobe Valley								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Hospital admissions respiratory (65+ years)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
QLD: Brisbane (SEQ)								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Hospital admissions respiratory (65+ years)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
SA: Adelaide								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Hospital admissions respiratory (65+ years)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
WA: Perth								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Hospital admissions respiratory (65+ years)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
NT: Darwin								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Hospital admissions respiratory (65+ years)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

Annexure B: Detailed risk characterisation for NO_2

B.1 Historical and projected health burden

Table B-1: Historical and projected health burden attributable to NO_2 (NSW: Sydney)

Health outcome	Historical					Projected						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
Long-term mortality, all cause[a] (30+ years)	0[b]	0[b]	0[b]	0[b]	0[b]	*BAU*	0[b]	0[b]	0[b]			
						Abatement Package	42 (19-76)	31 (14-56)	35 (16-64)	- -	12 (6-22)	23 (11-42)
Daily mortality, all-cause[a]	43 (25-60)	41 (24-57)	44 (26-62)	43 (25-60)	41 (24-57)	*BAU*	35 (20-49)	39 (23-55)	46 (27-65)			
						Abatement Package	34 (20-47)	31 (18-43)	35 (20-49)	1 (1-2)	8 (5-12)	12 (7-16)
Hospital admissions, cardiovascular (65+ years)	1135 (252-2008)	1043 (241-1844)	1147 (265-2029)	1411 (326-2496)	1755 (405-3106)	*BAU*	1704 (393-3015)	1902 (439-3365)	2246 (518-3974)			
						Abatement Package	1647 (380-2913)	1503 (347-2659)	1690 (390-2990)	58 (13-102)	399 (92-706)	556 (128-984)
Hospital admissions, respiratory (65+ years)	642 (410-874)	590 (377-803)	649 (414-883)	799 (510-1086)	994 (634-1352)	*BAU*	965 (616-1312)	1077 (687-1465)	1271 (812-1730)			
						Abatement Package	932 (595-1268)	851 (543-1158)	957 (611-1302)	33 (21-44)	226 (144-307)	315 (201-429)
Hospital admissions, respiratory (15-64 years)	609 (389-829)	579 (369-787)	635 (405-864)	581 (371-790)	512 (327-696)	*BAU*	420 (268-571)	469 (299-638)	561 (358-763)			
						Abatement Package	406 (259-552)	371 (236-504)	422 (269-574)	14 (9-19)	98 (63-134)	139 (89-189)
Emergency department visits, asthma (<15 years)	43 (42-114)	40 (40-107)	43 (42-114)	42 (41-111)	40 (40-107)	*BAU*	35 (35-93)	39 (39-104)	44 (44-118)			
						Abatement Package	34 (34-90)	31 (30-82)	33 (33-89)	1 (1-3)	8 (8-22)	11 (11-29)

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(b) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

Table B-2: Historical and projected health burden attributable to NO_2 (NSW: Newcastle)

Health outcome	Historical					Projected						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
Long-term mortality, all cause[a] (30+ years)	0[b]	0[b]	0[b]	0[b]	0[b]	*BAU*	0[b]	0[b]	0[b]			
						Abatement Package	3 (1-5)	2 (1-4)	2 (1-4)	- -	1 (1-3)	2 (1-4)
Daily mortality, all-cause[a]	5 (2-6)	4 (2-5)	4 (2-6)	4 (2-5)	4 (2-5)	*BAU*	3 (2-4)	4 (2-5)	4 (2-6)			
						Abatement Package	3 (1-4)	3 (1-4)	3 (1-4)	- -	1 (1-1)	1 (1-2)
Hospital admissions, cardiovascular (65+ years)	100 (23-177)	91 (21-160)	96 (22-171)	98 (23-174)	119 (27-210)	*BAU*	117 (27-208)	132 (30-233)	150 (35-265)			
						Abatement Package	112 (26-197)	101 (23-179)	101 (23-180)	6 (1-11)	31 (7-54)	48 (12-85)
Hospital admissions, respiratory (65+ years)	49 (31-66)	44 (28-60)	47 (30-64)	48 (30-65)	58 (37-79)	*BAU*	57 (36-78)	65 (41-87)	73 (46-99)			
						Abatement Package	55 (34-74)	50 (31-67)	50 (31-67)	3 (2-4)	15 (10-20)	24 (15-32)
Hospital admissions, respiratory (15-64 years)	40 (25-54)	35 (22-47)	37 (23-50)	31 (19-41)	26 (16-35)	*BAU*	25 (16-34)	28 (18-38)	32 (20-43)			
						Abatement Package	24 (15-32)	22 (13-29)	22 (13-29)	1 (1-2)	7 (4-9)	10 (7-14)
Emergency department visits, asthma (<15 years)	3 (2-7)	2 (2-6)	2 (2-6)	2 (2-5)	2 (1-5)	*BAU*	2 (1-5)	2 (2-5)	2 (2-6)			
						Abatement Package	2 (1-4)	2 (1-4)	2 (1-4)	- -	- (0-1)	1 (1-2)

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(b) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

Table B-3: Historical and projected health burden attributable to NO_2 (NSW: Wollongong)

Health outcome	Historical					Projected						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
Long-term mortality, all cause[a] (30+ years)	0[b]	0[b]	0[b]	0[b]	0[b]	*BAU*	0[b]	0[b]	0[b]			
						Abatement Package	2 (1-4)	2 (0-3)	2 (0-3)	- -	1 (0-2)	2 (1-3)
Daily mortality, all-cause[a]	2 (1-2)	2 (1-2)	2 (0-2)	2 (0-2)	1 (0-2)	*BAU*	1 (0-0)	1 (0-0)	1 (0-1)			
						Abatement Package	1 (0-0)	0 (0-0)	0 (0-0)	- -	- -	- (0-1)
Hospital admissions, cardiovascular (65+ years)	45 (10-80)	49 (11-87)	44 (10-77)	45 (10-80)	52 (12-92)	*BAU*	27 (6-47)	28 (6-49)	33 (8-59)			
						Abatement Package	24 (6-43)	18 (4-32)	17 (4-30)	2 (0-4)	10 (2-17)	16 (4-29)
Hospital admissions, respiratory (65+ years)	25 (15-33)	27 (17-36)	24 (15-32)	25 (15-33)	29 (18-38)	*BAU*	15 (9-19)	15 (9-20)	18 (11-24)			
						Abatement Package	13 (8-18)	10 (6-13)	9 (6-12)	1 (1-2)	5 (3-7)	9 (6-12)
Hospital admissions, respiratory (15-64 years)	23 (14-31)	25 (15-33)	21 (13-29)	20 (12-26)	17 (11-23)	*BAU*	8 (4-10)	8 (5-11)	10 (6-13)			
						Abatement Package	7 (4-9)	5 (3-7)	5 (3-6)	1 (0-1)	3 (2-4)	5 (3-6)
Emergency department visits, asthma (<15 years)	1 (1-3)	1 (1-3)	1 (1-2)	1 (0-2)	1 (0-2)	*BAU*	- (0-1)	- (0-1)	1 (0-1)			
						Abatement Package	- (0-1)	- -	- -	- -	- -	- (0-1)

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(b) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 μg/m^3.

Table B-4: Historical and projected health burden attributable to NO_2 (VIC: Melbourne)

Health outcome	Historical					Projected						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
Long-term mortality, all cause[a] (30+ years)	0[b]	0[b]	0[b]	0[b]	0[b]	*BAU*	0[b]	0[b]	0[b]			
						Abatement Package	17 (7-30)	7 (3-12)	11 (5-20)	- -	1 (1-2)	3 (1-6)
Daily mortality, all-cause[a]	32 (18-44)	35 (20-48)	30 (18-42)	34 (20-47)	35 (21-49)	*BAU*	21 (12-29)	18 (10-24)	31 (18-44)			
						Abatement Package	19 (11-26)	17 (9-23)	21 (12-30)	2 (1-3)	1 (0-1)	10 (6-14)
Hospital admissions, cardiovascular (65+ years)	684 (158-1211)	753 (174-1333)	667 (154-1180)	966 (223-1709)	1349 (311-2386)	*BAU*	869 (201-1537)	735 (170-1300)	1319 (304-2333)			
						Abatement Package	784 (181-1388)	702 (162-1242)	901 (208-1594)	85 (20-149)	33 (8-58)	417 (96-739)
Hospital admissions, respiratory (65+ years)	392 (250-534)	432 (275-587)	382 (244-520)	554 (353-753)	773 (493-1052)	*BAU*	498 (318-678)	421 (269-573)	756 (482-1028)			
						Abatement Package	450 (287-612)	403 (257-547)	517 (330-703)	48 (31-66)	19 (12-26)	239 (153-326)
Hospital admissions, respiratory (15-64 years)	523 (334-711)	574 (366-780)	507 (323-689)	531 (339-723)	506 (323-688)	*BAU*	280 (179-381)	237 (151-321)	431 (275-586)			
						Abatement Package	253 (161-344)	226 (144-307)	295 (188-401)	27 (17-37)	11 (7-14)	137 (87-186)
Emergency department visits, asthma (<15 years)	55 (55-146)	60 (60-160)	52 (51-138)	58 (58-154)	61 (60-161)	*BAU*	37 (36-98)	31 (31-83)	53 (53-142)			
						Abatement Package	33 (33-88)	30 (29-79)	37 (36-97)	4 (4-10)	1 (1-4)	17 (17-45)

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(b) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

Table B-5: Historical and projected health burden attributable to NO_2 (VIC: Latrobe Valley)

Health outcome	Historical					Projected						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
Long-term mortality, all cause[a] (30+ years)	0[b]	0[b]	0[b]	0[b]	0[b]	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Daily mortality, all-cause[a]	1 (0-0)	1 (0-1)	1 (0-0)	1 (0-0)	- -	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, cardiovascular (65+ years)	13 (3-24)	14 (3-25)	13 (3-23)	17 (4-30)	14 (3-25)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, respiratory (65+ years)	9 (5-11)	9 (5-12)	8 (5-11)	11 (6-14)	9 (5-12)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, respiratory (15-64 years)	12 (7-16)	12 (7-16)	12 (7-15)	10 (6-13)	7 (4-9)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits, asthma (<15 years)	1 (1-2)	1 (1-3)	1 (1-2)	1 (0-2)	1 (0-1)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(b) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

Table B-6: Historical and projected health burden attributable to NO_2 (QLD: Brisbane)

Health outcome	Historical					Projected[a]						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
Long-term mortality, all cause[b] (30+ years)	0[c]	0[c]	0[c]	0[c]	0[c]	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Daily mortality, all-cause[b]	11 (6-15)	13 (7-17)	11 (6-16)	13 (7-17)	11 (6-16)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, cardiovascular (65+ years)	272 (63-481)	302 (70-535)	273 (63-483)	310 (71-548)	283 (65-501)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, respiratory (65+ years)	151 (96-205)	168 (107-228)	152 (96-206)	172 (109-234)	157 (100-213)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, respiratory (15-64 years)	160 (102-217)	178 (113-241)	161 (102-219)	179 (114-244)	160 (102-218)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits, asthma (<15 years)	10 (9-25)	11 (10-28)	9 (9-25)	11 (10-28)	10 (9-25)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a

(a) 'n/a' = not available (not modelled).

(b) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(c) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

Table B-7: Historical and projected health burden attributable to NO_2 (SA: Adelaide)

Health outcome	Historical					Projected[a]						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
Long-term mortality, all cause[b] (30+ years)	0[c]	0[c]	0[c]	0[c]	0[c]	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Daily mortality, all-cause[b]	8 (4-11)	6 (3-9)	6 (3-9)	6 (3-8)	8 (4-11)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, cardiovascular (65+ years)	240 (55-424)	190 (44-337)	191 (44-338)	188 (43-333)	241 (56-426)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, respiratory (65+ years)	141 (90-191)	112 (71-152)	112 (71-152)	111 (70-150)	142 (90-192)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, respiratory (15-64 years)	131 (83-177)	104 (66-141)	104 (66-141)	103 (65-139)	131 (83-178)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits, asthma (<15 years)	13 (12-34)	10 (10-27)	10 (10-27)	10 (10-26)	13 (12-34)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a

(a) 'n/a' = not available (not modelled).

(b) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(c) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

Table B-8: Historical and projected health burden attributable to NO_2 (WA: Perth)

Health outcome	Historical					Projected[a]							
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
Long-term mortality, all cause[b] (30+ years)	0[c]	0[c]	0[c]	0[c]	0[c]	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Daily mortality, all-cause[b]	7 (4-9)	6 (3-7)	7 (4-9)	6 (3-8)	6 (3-7)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, cardiovascular (65+ years)	165 (38-292)	136 (31-240)	165 (38-291)	154 (36-273)	139 (32-246)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, respiratory (65+ years)	98 (62-132)	80 (51-109)	97 (62-132)	91 (58-124)	82 (52-112)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, respiratory (15-64 years)	107 (68-145)	87 (55-118)	107 (68-145)	99 (63-134)	88 (56-120)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits, asthma (<15 years)	8 (7-21)	6 (6-17)	8 (7-20)	7 (7-19)	6 (6-17)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a

(a) 'n/a' = not available (not modelled).

(b) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(c) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

Table B-9: Historical and projected health burden attributable to NO_2 (NT: Darwin)

Health outcome	Historical					Projected[a]						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
Long-term mortality, all cause[b] (30+ years)	No data	0[c]	0[c]	0[c]	0[c]	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Daily mortality, all-cause[b]	No data	- -	- -	- -	- -	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, cardiovascular (65+ years)	No data	- -	- -	- -	- -	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, respiratory (65+ years)	No data	- -	- -	- -	- -	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Hospital admissions, respiratory (15-64 years)	No data	3 (2-4)	2 (1-3)	5 (3-7)	3 (1-3)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits, asthma (<15 years)	No data	- -	- -	- -	- -	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a

(a) 'n/a' = not available (not modelled).

(b) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(c) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

B.2 Health outcomes - compliance with standards

Table B-10: Health outcomes for compliance with proposed 1-hour standard for NO_2 of 40 ppb (NSW)

Health outcome	Historical				Projected			
	Number of health outcomes							
	2010	2011	2012	2013	2014	2021	2031	2040
NSW: Sydney								
Long-term mortality, all cause[a] (30+ years)	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]
Daily mortality, all-cause[a]	32 (19-45)	35 (20-49)	31 (18-43)	26 (15-35)	26 (15-36)	18 (10-24)	35 (20-49)	36 (21-51)
Hospital admissions, cardiovascular (65+ years)	858 (198-1517)	910 (210-1610)	798 (184-1412)	842 (194-1490)	1112 (257-1967)	856 (197-1514)	1694 (391-2996)	1757 (405-3108)
Hospital admissions, respiratory (65+ years)	485 (310-660)	515 (329-701)	452 (288-614)	477 (304-648)	629 (402-856)	484 (309-659)	959 (612-1304)	994 (635-1353)
Hospital admissions, respiratory (15-64 years)	460 (294-626)	505 (322-687)	442 (282-601)	347 (221-472)	324 (207-441)	211 (134-286)	418 (266-568)	439 (280-597)
Emergency department visits, asthma (<15 years)	32 (32-86)	35 (35-94)	30 (29-79)	25 (24-66)	25 (25-67)	18 (17-47)	35 (34-92)	35 (34-92)
NSW: Newcastle								
Long-term mortality, all cause[a] (30+ years)	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]
Daily mortality, all-cause[a]	-	-	-	5 (3-7)	5 (2-6)	-	-	-
Hospital admissions, cardiovascular (65+ years)	-	-	-	140 (32-248)	161 (37-285)	-	-	-
Hospital admissions, respiratory (65+ years)	-	-	-	69 (43-93)	79 (50-107)	-	-	-
Hospital admissions, respiratory (15-64 years)	-	-	-	44 (27-59)	36 (22-48)	-	-	-
Emergency department visits, asthma (<15 years)	-	-	-	3 (3-8)	3 (2-7)	-	-	-
NSW: Wollongong								
Long-term mortality, all cause[a] (30+ years)	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]
Daily mortality, all-cause[a]	2 (1-2)	2 (1-2)	2 (1-2)	2 (1-2)	-	-	-	-
Hospital admissions, cardiovascular (65+ years)	46 (11-81)	51 (12-91)	51 (12-91)	58 (13-102)	-	-	-	-
Hospital admissions, respiratory (65+ years)	25 (16-34)	28 (18-38)	28 (18-38)	32 (20-43)	-	-	-	-
Hospital admissions, respiratory (15-64 years)	23 (14-31)	26 (16-34)	25 (16-34)	25 (16-34)	-	-	-	-
Emergency department visits, asthma (<15 years)	1 (1-3)	1 (1-3)	1 (1-3)	1 (1-3)	-	-	-	-

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(b) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

Table B-11: Health outcomes for compliance with proposed 1-hour standard for NO_2 of 40 ppb (Victoria)

Health outcome	Historical				Projected			
	Number of health outcomes							
	2010	2011	2012	2013	2014	2021	2031	2040
VIC: Melbourne								
Long-term mortality, all cause[a] (30+ years)	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]
Daily mortality, all-cause[a]	26 (15-36)	-	-	28 (16-39)	-	-	-	-
Hospital admissions, cardiovascular (65+ years)	564 (130-997)	-	-	797 (184-1410)	-	-	-	-
Hospital admissions, respiratory (65+ years)	323 (206-439)	-	-	457 (291-621)	-	-	-	-
Hospital admissions, respiratory (15-64 years)	431 (275-586)	-	-	438 (280-596)	-	-	-	-
Emergency department visits, asthma (<15 years)	45 (45-120)	-	-	48 (47-127)	-	-	-	-
VIC: Latrobe Valley								
Long-term mortality, all cause[a] (30+ years)	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]	0[b]
Daily mortality, all-cause[a]	-	-	-	-	-	-	-	-
Hospital admissions, cardiovascular (65+ years)	-	-	-	-	-	-	-	-
Hospital admissions, respiratory (65+ years)	-	-	-	-	-	-	-	-
Hospital admissions, respiratory (15-64 years)	-	-	-	-	-	-	-	-
Emergency department visits, asthma (<15 years)	-	-	-	-	-	-	-	-

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(b) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

Table B-12: Health outcomes for compliance with proposed 1-hour standard for NO_2 of 40 ppb (other airsheds)

Health outcome	Historical					Projected[a]		
	Number of health outcomes							
	2010	2011	2012	2013	2014	2021	2031	2040
QLD: Brisbane (SEQ)								
Long-term mortality, all cause[b] (30+ years)	0[c]	0[c]	0[c]	0[c]	0[c]	n/a	n/a	n/a
Daily mortality, all-cause[b]	-	-	16 (9-22)	18 (10-25)	16 (9-22)	n/a	n/a	n/a
Hospital admissions, cardiovascular (65+ years)	-	-	377 (87-667)	446 (103-790)	398 (92-704)	n/a	n/a	n/a
Hospital admissions, respiratory (65+ years)	-	-	209 (133-285)	248 (158-337)	221 (141-300)	n/a	n/a	n/a
Hospital admissions, respiratory (15-64 years)	-	-	223 (142-303)	258 (165-351)	226 (144-307)	n/a	n/a	n/a
Emergency department visits, asthma (<15 years)	-	-	13 (13-34)	15 (15-40)	13 (13-35)	n/a	n/a	n/a
SA: Adelaide								
Long-term mortality, all cause[b] (30+ years)	0[c]	0[c]	0[c]	0[c]	0[c]	n/a	n/a	n/a
Daily mortality, all-cause[b]	12 (7-16)	9 (5-12)	13 (7-18)	13 (7-17)	13 (7-17)	n/a	n/a	n/a
Hospital admissions, cardiovascular (65+ years)	353 (81-624)	272 (63-481)	382 (88-676)	377 (87-668)	371 (86-656)	n/a	n/a	n/a
Hospital admissions, respiratory (65+ years)	207 (132-282)	160 (102-217)	225 (143-305)	222 (141-301)	218 (139-296)	n/a	n/a	n/a
Hospital admissions, respiratory (15-64 years)	192 (122-261)	148 (94-201)	208 (132-283)	206 (131-279)	202 (129-274)	n/a	n/a	n/a
Emergency department visits, asthma (<15 years)	19 (18-50)	15 (14-38)	20 (20-54)	20 (20-53)	20 (19-52)	n/a	n/a	n/a
WA: Perth								
Long-term mortality, all cause[b] (30+ years)	0[c]	0[c]	0[c]	0[c]	0[c]	n/a	n/a	n/a
Daily mortality, all-cause[b]	10 (5-14)	-	11 (6-15)	13 (7-18)	11 (6-14)	n/a	n/a	n/a
Hospital admissions, cardiovascular (65+ years)	241 (56-426)	-	267 (62-472)	314 (73-556)	259 (60-458)	n/a	n/a	n/a
Hospital admissions, respiratory (65+ years)	142 (90-193)	-	158 (100-215)	186 (118-253)	153 (97-208)	n/a	n/a	n/a
Hospital admissions, respiratory (15-64 years)	156 (99-212)	-	173 (110-235)	202 (128-274)	164 (104-223)	n/a	n/a	n/a
Emergency department visits, asthma (<15 years)	12 (11-30)	-	12 (12-33)	15 (14-38)	12 (11-31)	n/a	n/a	n/a
NT: Darwin								
Long-term mortality, all cause[b] (30+ years)	0[c]	0[c]	0[c]	0[c]	0[c]	n/a	n/a	n/a
Daily mortality, all-cause[b]	-	-	-	-	-	n/a	n/a	n/a
Hospital admissions, cardiovascular (65+ years)	-	-	-	-	-	n/a	n/a	n/a
Hospital admissions, respiratory (65+ years)	-	-	-	-	-	n/a	n/a	n/a
Hospital admissions, respiratory (15-64 years)	-	3 (1-3)	-	-	-	n/a	n/a	n/a
Emergency department visits, asthma (<15 years)	-	-	-	-	-	n/a	n/a	n/a

(a) 'n/a' = not available (not modelled).

(b) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

(c) The health burden is zero because average airshed concentrations are lower than that the HRAPIE cut-off of 20 $\mu g/m^3$.

Annexure C: Detailed risk characterisation for O_3

C.1 Historical and projected health burden

Table C-1: Historical and projected health burden attributable to O_3 (NSW)

Health outcome	Historical					Projected						
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
NSW: Sydney												
Daily mortality, all-cause[(a)]	137 (66-203)	119 (57-175)	138 (66-204)	181 (87-268)	185 (89-274)	*BAU*	54 (26-80)	67 (32-99)	78 (37-115)			
						Abatement Package	50 (24-74)	60 (28-89)	72 (34-106)	4 (2-6)	7 (3-10)	6 (3-9)
Emergency department visits for asthma (<15 years)	118 (94-211)	111 (89-200)	116 (92-208)	129 (102-231)	137 (109-246)	*BAU*	93 (74-167)	111 (88-199)	123 (98-220)			
						Abatement Package	93 (74-167)	111 (88-199)	122 (97-219)	0 (0-0)	0 (0-0)	1 (1-1)
NSW: Newcastle												
Daily mortality, all-cause[(a)]	13 (6-19)	11 (5-16)	12 (5-17)	17 (8-24)	14 (6-20)	*BAU*	5 (2-8)	6 (2-8)	6 (2-8)			
						Abatement Package	5 (2-8)	5 (2-8)	6 (2-8)	0 (0-0)	0 (0-0)	0 (0-0)
Emergency department visits for asthma (<15 years)	8 (6-14)	7 (5-13)	8 (6-13)	8 (6-14)	8 (6-14)	*BAU*	5 (4-9)	6 (4-10)	6 (5-11)			
						Abatement Package	6 (4-9)	6 (4-10)	6 (5-11)	0 (0-0)	0 (0-0)	0 (0-0)
NSW: Wollongong												
Daily mortality, all-cause[(a)]	5 (2-7)	5 (2-7)	4 (1-6)	6 (3-9)	6 (2-8)	*BAU*	1 (0-1)	1 (0-1)	1 (0-1)			
						Abatement Package	1 (0-1)	1 (0-2)	2 (0-2)	0 (0-0)	0 (0--1)	0 (0--1)
Emergency department visits for asthma (<15 years)	4 (3-7)	4 (3-7)	4 (3-7)	5 (3-8)	5 (3-8)	*BAU*	3 (2-5)	3 (2-5)	3 (2-6)			
						Abatement Package	3 (2-5)	3 (2-5)	3 (2-6)	0 (0-0)	0 (0-0)	0 (0-0)

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

Table C-2: Historical and projected health burden attributable to O_3 (Victoria)

Health outcome	Historical					Projected							
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
VIC: Melbourne												
Daily mortality, all-cause[(a)]	42 (20-62)	10 (4-14)	8 (3-12)	82 (39-122)	85 (41-126)	*BAU*	50 (23-73)	59 (28-88)	70 (33-104)			
						Abatement Package	42 (20-62)	54 (26-80)	63 (30-93)	8 (4-11)	5 (3-8)	7 (4-11)
Emergency department visits for asthma (<15 years)	135 (107-242)	90 (72-162)	79 (62-141)	167 (133-300)	173 (138-311)	*BAU*	160 (128-288)	200 (159-359)	221 (177-398)			
						Abatement Package	183 (146-330)	197 (157-354)	218 (174-391)	-23 (-18--42)	2 (2-4)	4 (3-7)
VIC: Latrobe Valley												
Daily mortality, all-cause[(a)]	1 (0-1)	0 (0-0)	1 (0-0)	2 (0-2)	1 (0-1)	*BAU*	1 (0-1)	1 (0-1)	1 (0-1)			
						Abatement Package	1 (0-1)	1 (0-1)	1 (0-1)	- -	- -	- -
Emergency department visits for asthma (<15 years)	3 (2-5)	3 (2-4)	3 (2-5)	3 (2-6)	2 (1-4)	*BAU*	3 (2-5)	4 (2-6)	4 (3-7)			
						Abatement Package	3 (2-5)	4 (2-6)	4 (3-6)	- -	- -	- -

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

Table C-3: Historical and projected health burden attributable to O_3 (other airsheds)

Health outcome	Historical					Projected[(a)]							
	Number of attributable health outcomes					*Projection scenario*	Number of attributable health outcomes			Number of health outcomes avoided with abatement		
	2010	2011	2012	2013	2014		2021	2031	2040	2021	2031	2040
QLD: Brisbane (SEQ)												
Daily mortality, all-cause[(b)]	31 (15-46)	40 (19-59)	62 (29-92)	65 (31-96)	67 (32-99)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits for asthma (<15 years)	39 (30-69)	43 (34-76)	49 (39-87)	49 (39-88)	53 (42-94)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
SA: Adelaide												
Daily mortality, all-cause[(b)]	22 (10-32)	26 (12-38)	22 (10-32)	24 (11-36)	30 (14-43)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits for asthma (<15 years)	59 (47-105)	62 (49-111)	63 (50-114)	63 (50-114)	66 (52-119)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
WA: Perth												
Daily mortality, all-cause[(b)]	47 (22-69)	45 (21-67)	47 (22-70)	58 (27-85)	49 (23-71)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits for asthma (<15 years)	50 (40-90)	51 (40-91)	55 (44-99)	59 (46-105)	57 (45-102)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
NT: Darwin												
Daily mortality, all-cause	No data	4 (1-5)	6 (2-8)	3 (1-4)	3 (1-4)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a
Emergency department visits for asthma (<15 years)	No data	1 (0-2)	2 (1-2)	1 (0-2)	1 (1-2)	*BAU*	n/a	n/a	n/a			
						Abatement Package	n/a	n/a	n/a	n/a	n/a	n/a

(a) 'n/a' = not available (not modelled).

(b) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

C.2 Health outcomes - compliance with standards

Table C-4: Health outcomes for compliance with proposed 1-hour standard for O_3 of 70 ppb

Health outcome	Historical					Projected		
	Number of health outcomes							
	2010	2011	2012	2013	2014	2021	2031	2040
NSW: Sydney								
Daily mortality all causes[(a)] (all ages)	-	-	53 (25-78)	40 (19-60)	-	27 (12-39)	31 (15-46)	34 (16-50)
Emergency dept. visits asthma (<15 years)	-	-	87 (69-157)	82 (65-148)	-	71 (57-128)	85 (68-153)	88 (70-157)
NSW: Newcastle								
Daily mortality all causes[(a)] (all ages)	7 (3-11)	11 (5-16)	9 (4-12)	9 (4-13)	-	3 (1-4)	3 (1-4)	3 (1-4)
Emergency dept. visits asthma (<15 years)	7 (5-12)	7 (5-13)	7 (5-11)	7 (5-12)	-	5 (3-8)	5 (3-8)	5 (3-8)
NSW: Wollongong								
Daily mortality all causes[(a)] (all ages)	2 (1-3)	1 (0-1)	-	1 (0-1)	-	-	-	-
Emergency dept. visits asthma (<15 years)	3 (2-5)	3 (2-4)	-	3 (2-4)	-	-	-	-
VIC: Melbourne								
Daily mortality all causes[(a)] (all ages)	-	38 (18-56)	29 (13-42)	27 (12-39)	13 (6-19)	48 (23-71)	-	-
Emergency dept. visits asthma (<15 years)	-	135 (108-243)	124 (98-222)	115 (92-207)	81 (65-146)	154 (123-277)	-	-
VIC: Latrobe Valley								
Daily mortality all causes[(a)] (all ages)	-	-	-	1 (0-1)	1 (0-1)	1 (0-1)	1 (0-1)	1 (0-1)
Emergency dept. visits asthma (<15 years)	-	-	-	3 (2-4)	3 (2-5)	3 (2-5)	3 (2-5)	3 (2-6)
QLD: Brisbane (SEQ)								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	3 (2-5)	-	-	-	-	-	-	-
SA: Adelaide								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
WA: Perth								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	7 (5-13)	10 (8-18)	2 (1-4)	8 (6-13)	7 (5-12)	-	-	-
NT: Darwin								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

Table C-5: Health outcomes for compliance with proposed 1-hour standard for O_3 of 85 ppb

Health outcome	Historical					Projected		
	Number of health outcomes							
	2010	2011	2012	2013	2014	2021	2031	2040
NSW: Sydney								
Daily mortality all causes[(a)] (all ages)	-	-	108 (52-160)	92 (44-136)	-	44 (21-65)	52 (24-76)	59 (28-87)
Emergency dept. visits asthma (<15 years)	-	-	106 (84-190)	100 (80-180)	-	86 (68-154)	103 (82-185)	111 (89-200)
NSW: Newcastle								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
NSW: Wollongong								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
VIC: Melbourne								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
VIC: Latrobe Valley								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
QLD: Brisbane (SEQ)								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
SA: Adelaide								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
WA: Perth								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
NT: Darwin								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.

Table C-6: Health outcomes for compliance with proposed 1-hour standard for O_3 of 100 ppb

Health outcome	Historical					Projected		
	Number of health outcomes							
	2010	2011	2012	2013	2014	2021	2031	2040
NSW: Sydney								
Daily mortality all causes[(a)] (all ages)	-	-	-	143 (68-211)	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	118 (94-212)	-	-	-	-
NSW: Newcastle								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
NSW: Wollongong								
Daily mortality all causes[(a)] (all ages)	-	3 (1-4)	-	3 (1-4)	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	4 (2-6)	-	4 (2-6)	-	-	-	-
VIC: Melbourne								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	39 (18-58)	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	116 (93-209)	-	-	-
VIC: Latrobe Valley								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
QLD: Brisbane (SEQ)								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
SA: Adelaide								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	-	-	-	-	-	-
WA: Perth								
Daily mortality all causes[(a)] (all ages)	-	-	-	-	-	-	-	-
Emergency dept. visits asthma (<15 years)	-	-	9 (7-15)	26 (20-46)	-	-	-	-
NT: Darwin								
Daily mortality all causes (all ages)	-	-	-	-	-	-	-	-

(a) 'All-cause mortality' refers to acute mortality events that are estimated to shorten life by 6 months.