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The mortality effect of $PM_{2.5}$ sources in the Greater Metropolitan Region of Sydney, Australia

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ABSTRACT

We describe an assessment of the impact on mortality of eight major sources of $PM_{2.5}$ in the Greater Metropolitan Region of Sydney, Australia (GMR). We modeled exposure to $PM_{2.5}$ for the year July 2010 to June 2011 and estimated the burden of current mortality attributable to these sources. We also estimated the number of life-years that would be produced if emissions from wood heaters and power stations, the two largest emissions sources, were reduced.

Wood heaters (assuming a real-world emissions factor of 11.4 g of $PM_{2.5}$ per kg of wood burned) were the most important source of $PM_{2.5}$ exposure, responsible for around 24.0% of the total anthropogenic $PM_{2.5}$ concentration. On-road sources and power stations were also important, responsible for 16.9% and 10.5% of anthropogenic $PM_{2.5}$ exposure respectively.

Around 1.2% of mortality (5,900 YLL) was attributable to long-term exposure to all anthropogenic $PM_{2.5}$, including 0.3% (1,400 YLL) attributable to wood heater–related $PM_{2.5}$, 0.2% (990 YLL) to on-road sources and 0.1% (620 YLL) to power stations.

Compared to ongoing emissions at 2010/11 levels, we estimated that a sustained reduction in emissions from wood heaters due to the introduction of an emissions standard of 1.5 g of $PM_{2.5}$ per kilogram of wood burned (real world emissions factor of 3.9 g of $PM_{2.5}$ per kg of wood burned) and the associated reduction in $PM_{2.5}$ population exposure would produce 90,000 life-years among the cohort of people alive in 2010/11. Complete removal of sulphur oxide emissions from power stations would produce 14,000 life-years and complete removal of nitrogen oxide emissions would produce 38,000 life-years. A range of sensitivity analyses indicate the true impact of $PM_{2.5}$ from these sources is likely to be at least as large as these estimates.

This assessment shows that eight sources are responsible for more than 60% of exposure to anthropogenic $PM_{2.5}$ in the Sydney GMR. Although the burden of mortality attributable to each source is relatively small, interventions that achieve sustained reductions in emissions could provide substantial health benefits, which are likely to far outweigh the costs.

1. Introduction

Long-term exposure to airborne particulate matter of less than 2.5 μm in diameter ($PM_{2.5}$) has been shown to shorten life by causing cardiovascular and respiratory disease (US EPA, 2009; WHO, 2005; WHO Regional Office for Europe, 2013b). Globally, outdoor air pollution is the environmental risk factor responsible for the greatest burden of disease (GBD, 2017). There is mounting evidence that reductions in $PM_{2.5}$ concentrations increase life expectancy (Pope et al., 2009).

Health impact assessments have been used to estimate the harm attributable to $PM_{2.5}$ and to quantify the benefit of actions to control air pollution (Cohen et al., 2005; COMEAP, 2010; Fann et al., 2012). Such assessments integrate evidence from epidemiological studies with local information about the $PM_{2.5}$ concentration, the size of the exposed population and its disease rates. Most commonly, impact assessments have quantified the burden of mortality attributable to long-term exposure to $PM_{2.5}$. That is, they have provided estimates of the effect on mortality in a specific year as a result of the exposure that occurred in recent years (Anenberg et al., 2010; Cohen et al., 2017). Less commonly, impact assessment methods have been used to predict how the

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life-extending effect of reducing $PM_{2.5}$ affects the demographic makeup of the population in the future (Miller and Hurley, 2003; Walton et al., 2015).

Interventions or actions to reduce $PM_{2.5}$ concentrations tend to target particular sources. Therefore, it is useful to attribute exposure and mortality to specific sources so that interventions can be given appropriate priority (Fann et al., 2013). Such source-specific attribution is becoming increasingly common, supported by advances in particle modeling. While modeled estimates of $PM_{2.5}$ concentrations suffer from uncertainties related to pollutant emissions, meteorology and atmospheric processes, they have the advantage of providing estimates of $PM_{2.5}$ concentration in areas where there are limited or no measurements from fixed site monitors. Particle models are also able to provide source-specific $PM_{2.5}$ concentration estimates that can be used for the attribution of health impacts (Lelieveld et al., 2015). For example, the GBD MAPS Working Group used a particle model to estimate that industrial sources were responsible for 27% of mortality attributable to $PM_{2.5}$ in China and that household solid fuel combustion was responsible for 19% (GBD MAPS Working Group, 2016; GBD MAPS Working Group, 2018). The same group estimated that residential biomass burning was responsible for 25% of deaths attributable to $PM_{2.5}$ in India, while coal combustion from industrial sources and power stations was responsible for around 16% of mortality (GBD MAPS Working Group, 2018).

The Sydney Greater Metropolitan Region of NSW (Sydney GMR) is on the east coast of Australia and has a population of around 5.6 million. It has relatively good air quality, with annual average $PM_{2.5}$ concentrations around 8 $\mu g/m^3$ which is below the World Health Organization guideline of 10 $\mu g/m^3$. However, there is increasing evidence that $PM_{2.5}$ has detrimental health effects at such low concentrations and so interventions to reduce air pollution are likely to be beneficial even in regions with relatively low air pollution concentrations (Crouse et al., 2012; Di et al., 2017; Hanigan et al., 2019; Pinault et al., 2016; Shi et al., 2016).

There are a number of important sources of $PM_{2.5}$ emissions in the Sydney GMR, including coal- and gas-fired power stations, motor vehicles, industrial vehicles, ships and domestic wood heaters. Domestic wood heaters are the largest source of primary $PM_{2.5}$, responsible for about 19% of total $PM_{2.5}$ emissions in the region, although just 4.4% of Sydney residents used wood as their primary source of heating in 2014 (Australian Bureau of Statistics, 2014b; NSW EPA, 2012). Coal was the fuel source for 83% of electricity generated in NSW in the 2010/11 financial year. Consistent with this, power stations are another important source of primary $PM_{2.5}$ and are also a significant source of the precursors of secondary particles that form in the atmosphere, sulfates and nitrates in particular (Cohen et al., 2012; Department and EnergyEnvironment and Energy, 2019; Malfroy et al., 2005). Vehicle ownership in Australia is among the highest in the Organisation for Economic Cooperation and Development (OECD), with 75 vehicles per 100 inhabitants in 2014 (OECD, 2015). In 2011, 91% of passenger vehicles used unleaded petrol (Australian Bureau of Statistics, 2011).

While $PM_{2.5}$ emissions from many sources are routinely estimated for the Syndey GMR, the contribution of source-specific emissions to human exposure and mortality is relatively unknown. Previous assessments have found that around 5,800 Years of Life Lost (YLL) annually were attributable to all anthropogenic $PM_{2.5}$ and that around 220 YLL were attributable to $PM_{2.5}$ from ships (Broome et al., 2015; Broome et al., 2016). The first of these assessments interpolated measured concentrations across the region and made assumptions about the proportion of $PM_{2.5}$ that was from anthropogenic sources. The second used a particle modeling framework to estimate the contribution of ships to total $PM_{2.5}$. To the best of our knowledge, there have been no health impact assessments that have applied a consistent modeling framework to multiple sources in this region.

This paper describes an assessment with two main parts. First, we estimated the existing burden of mortality attributable to long-term exposure to $PM_{2.5}$ from eight major sources in the Sydney GMR. Second, we estimated how much life would be produced by actions that reduce emissions from two major sources: wood heaters and power stations. For each part, a locally developed and validated particle modeling framework was used to estimate the $PM_{2.5}$ concentration attributable to each source.

2. Method

We used a three-stage impact assessment process that has been applied extensively in the literature (Fann et al., 2013; GBD MAPS Working Group, 2016). The first stage used a particle modeling framework to estimate the spatial distribution of source-specific $PM_{2.5}$ concentrations across the Sydney GMR for the baseline year, as well as future years under the selected air pollution control scenarios. The second stage combined the spatially resolved source-specific air pollution concentrations with population data to estimate a population-weighted concentration for the study region. The third stage used epidemiological evidence of the relationship between $PM_{2.5}$ and mortality to integrate the modeled population-weighted $PM_{2.5}$ concentration with local mortality estimates.

2.1. Air quality modeling

To estimate the burden of mortality attributable to specific sources of $PM_{2.5}$ emissions, we created a spatial grid of the modeled annual average (July 2010 to June 2011) ground-level $PM_{2.5}$concentration for the following sources:

1. Wood heaters
2. Coal- and gas-fired power stations
3. On-road sources
   (a) Petrol vehicles
   (b) Diesel vehicles
   (c) Non-exhaust emissions
4. Off-road mobile sources:
   (a) Industrial vehicles and equipment
   (b) Ships
   (c) Aircraft (air and land operations).

To estimate the benefit of actions that reduce emissions, we created spatial grids for each of the following scenarios:

1. Wood heater scenarios:
   (a) Wood heaters comply with an emissions standard of 2.5 grams of particles per kilogram of wood burned (g/kg)
   (b) Wood heaters comply with an emissions standard of 1.5 g/kg
   (c) Wood heaters are phased out
2. Power station scenarios:
   (a) All nitrous oxide emissions are prevented
   (b) All sulphur oxide emissions are prevented.

Real-world emissions from wood heaters are generally substantially larger than their technical specification because emissions are strongly influenced by factors outside the control of manufacturers, like fuel moisture content and airflow through the heater (Meyer et al., 2008). Therefore, the baseline 2010/11 wood heater emissions were modeled as 11.4 g of $PM_{2.5}$ per kilogram of wood burned and the 2.5 g/kg and 1.5 g/kg wood heater emissions standards were modeled as $PM_{2.5}$ emissions of 6.4 g/kg and 3.9 g/kg respectively.

We used a three-component particle modeling framework to produce gridded estimates of the daily average $PM_{2.5}$ concentration for each source and emissions-reduction scenario for the period July 2010 to June 2011. The framework is described in detail in Supplementary Appendix A. Briefly, the first component was the CSIRO Conformal Atmospheric Model (CCAM), which simulates meteorological

conditions (McGregor and Dix, 2008). The second component was the calculation and simulation of emissions of primary $PM_{2.5}$ and the gaseous precursors of secondary $PM_{2.5}$. Base emissions were taken from the NSW EPA 2008 air emissions inventory (NSW EPA, 2012), supplemented by a detailed inventory of ship emissions developed by Goldsworthy and Goldsworthy (2015). Emissions were adjusted for daily temperature and energy production in 2010/11. Modeled daily temperature for the study period was used to estimate daily Heating Degree Days (HDD) which was then used to scale the month-specific wood heater emissions to daily emissions that reflect local ambient temperatures throughout the Sydney GMR. Power station emissions were converted from generic hourly estimates to day specific emission estimates by using hourly power output data for the study period. The third component of the framework was the CSIRO chemical transport model, which calculates the transport, dispersion, chemical production and depositional loss of $PM_{2.5}$ (Cope et al., 2014).

Three grids were produced for each day and each scenario. A central 100 km by 100 km domain of 1 km resolution covered the Sydney conurbation. Around this, a 300 km by 300 km domain covered the majority of the rest of Sydney GMR at 3 km resolution. A small portion of the northern part of the study region (with low population density) lay outside this domain and was covered by a grid of 9 km resolution. All grids were combined to produce estimates of source-specific annual average concentrations over the entire study area. Finally, the annual average $PM_{2.5}$ grids were overlaid with a 1 km resolution population grid of Australia (Australian Bureau of Statistics, 2014a) to produce a population weighted-mean concentration for each source and scenario. These population weighted-means were our estimates of exposure (ΔX) for the health impact calculations.

2.1.1. Model verification

Specification of the meteorological model has a substantial effect on modeled $PM_{2.5}$ concentrations. We examined this effect by testing two configurations of CCAM (CCAM-A and CCAM-B). The configurations differed in the spectral nudging used to couple CCAM to global analysis fields and the prescription of surface roughness. We verified the predictions of each model against meteorological measurements and against fixed site monitor $PM_{2.5}$ concentrations using data provided by the NSW Office of Environment and Heritage. Details of the model verification process are provided in the Supplementary Appendix.

2.2. The concentration–response coefficient

Each health impact calculation relies on a concentration–response coefficient to relate the $PM_{2.5}$ concentration to a risk of death. Evidence from large multicity studies suggest that effects are similar across geographic areas. Therefore, we used a concentration–response coefficient derived from a meta-analysis of North American and European cohort studies (Hoek et al., 2013). The pooled estimate of relative risk from this study was 1.062 (95% confidence interval [CI] 1.041–1.084) for a 10 $\mu g/m^3$ increase in $PM_{2.5}$, giving a concentration–response coefficient (β-coefficient) of 0.006 (95% CI 0.004–0.008). The participants of cohort studies examining the relationship between $PM_{2.5}$ and mortality have generally been adults and so we assumed that the effect of $PM_{2.5}$ was limited to those aged 30 and older. Age-specific mortality and population data were obtained from the NSW Ministry of Health. We used the same concentration–response coefficient for each scenario. That is, we assumed that the mortality effect of $PM_{2.5}$ was the same, no matter it source.

There is some evidence that the concentration–response relationship steepens at lower $PM_{2.5}$ concentrations (Burnett et al., 2014; Crouse et al., 2012). Therefore, we conducted sensitivity analyses using an alternative β-coefficient derived from Roman et al. (2008) of 0.01.

2.3. Assessment of the burden of mortality

We described the burden of mortality related to specific sources of $PM_{2.5}$ using three metrics: The attributable number of deaths and YLL in the 2010/11 financial year, and the loss of life expectancy at birth.

The attributable number of deaths was calculated as:

$$\text{Attributable Number} = \sum_{i=30}^{85+} m_i(1 - e^{-\beta\Delta X})P_i$$

where m_i was the mortality rate in single-year age-group i in the year July 2010 to June 2011, β was a concentration–response coefficient that defines the relationship between the $PM_{2.5}$ concentration and mortality, ΔX was the population weighted-mean $PM_{2.5}$ concentration of interest and P_i was the size of the exposed population aged i.

Loss of life expectancy was calculated by subtracting 2010/11 life expectancy (calculated using standard life table methods) from the life expectancy there would have been if age-specific mortality rates among those aged 30 and over were reduced by $e^{-\beta\Delta X}$ (the reduction in risk associated to the source of interest).

YLL were calculated as:

$$\text{YLL} = \sum_{i=30}^{85+} \text{AN}_i \times \text{LE}_i$$

where AN_i was the attributable number of deaths among people aged i and LE_i was life expectancy of for age-group i.

2.4. Assessment of the benefit of $PM_{2.5}$ control strategies

We used a method adapted from Miller and Hurley (2003) to model the number of life-years produced by a sustained reduction in emissions over the lifetime of the Sydney GMR population alive in the baseline year. The method allows inclusion of a lag between reduced exposure to $PM_{2.5}$ and full attainment of the associated health benefits (a *cessation lag*) and a gradual reduction in exposure, as would be expected from an intervention that takes time to implement.

A series of matrices were used to predict the number of people surviving in each age-group and each future calendar year under a scenario of no change in mortality risk and scenarios where the mortality risk was reduced in line with reduced exposure to $PM_{2.5}$ (that is, by $e^{-\beta\Delta X}$). The number of life-years produced was calculated as the difference in population size between the baseline and reduced exposure scenarios in each future year. The calculations were conducted for the cohort of people alive in the baseline year and for an extended population, where it was assumed that the birth rate remained constant into the future and there was no migration.

The baseline year was July 2010 to June 2011 and the number of life-years produced were calculated over the subsequent 105 years. This is the approximate time to extinction of the cohort of people alive at baseline. It is important to include this length of time because the life-extending effects of reduced $PM_{2.5}$ exposure are only realised at the end of life.

We used a cessation lag structure previously recommended by the US EPA and applied in assessments by the UK's Committee on the Medical Effects of Air pollution (COMEAP, 2010; Walton, 2010). The structure assumes that 30% of the total health effect of reduced $PM_{2.5}$ exposure is achieved in the first year after the reduction, a further 12.5% in each of years two to five and the final 20% in years six to 20.

It has been estimated that the lifespan of a typical Australian wood heater is about 17 years so we assumed it would take this long to achieve the emissions reduction scenarios (AECOM, 2011). We assumed it would take 10 years to fully implement emissions controls from power stations.

Table 1
Predictions of the population-weighted $PM_{2.5}$ concentrations using the two models and the relative difference of CCAM-A compared to CCAM-B (which we have used for primary analyses).

Source ($\mu g/m^3$)	CCAM-A ($\mu g/m^3$)	CCAM-B	Relative difference (%)
Wood heaters	1.39	0.49	181
Power stations	0.19	0.22	−11
On-road mobile sources	0.61	0.35	76
Exhaust from petrol vehicle	0.13	0.08	55
Exhaust from diesel vehicles	0.30	0.16	89
Non-exhaust emissions	0.19	0.11	73
Off-road mobile sources	0.26	0.22	22
Industrial vehicles and equipment	0.07	0.06	6
Ships	0.14	0.12	21
Aircraft (flight and ground operations)	0.05	0.03	56
All anthropogenic sources	**3.50**	**2.06**	**70**

3. Results

3.1. Exposure assessment

3.1.1. Model verification

Estimates of population-weighted concentrations produced by the two model configurations are shown in Table 1. Estimates from the CCAM-B configuration were lower for all sources except power stations. The largest difference in source-specific estimates was for wood heaters.

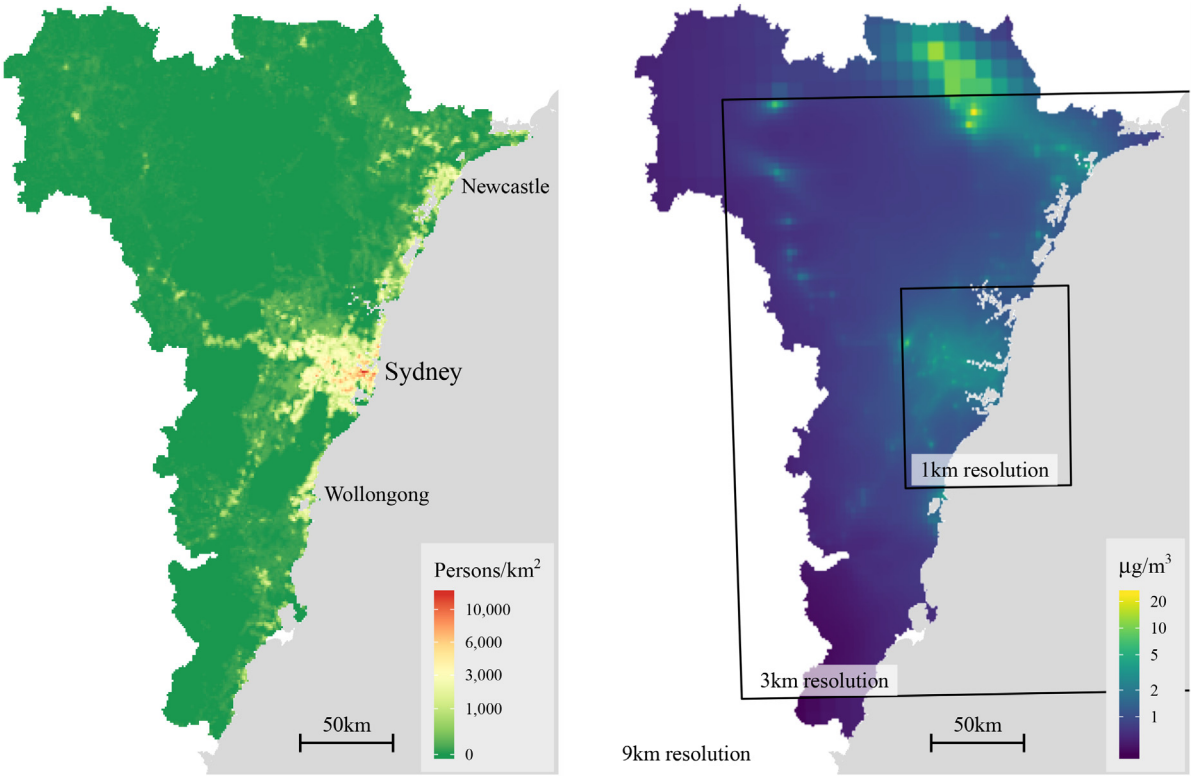
A detailed description of model performance and the model verification process is provided in the Supplementary Appendix. Verification of the output of the CCAM-A and CCAM-B model configurations against meteorological measurements found that both predicted temperature well, but that CCAM-A had a bias to under-predict wind speeds generally and CCAM-B had a bias to over-predict wind speeds at 10 m height. Both configurations modeled the wind direction probability density function well. Verification of modeled $PM_{2.5}$ concentrations against measured $PM_{2.5}$ concentrations showed that each configuration predicted day-to-day variations in $PM_{2.5}$ reasonably well, but that both were challenged to capture the most extreme $PM_{2.5}$ events. Statistical analysis suggested that the CCAM-B configuration was marginally better. The poorest performance of the CCAM-A configuration was during the winter months, with significant and regular over-prediction of daily $PM_{2.5}$. This over-prediction of $PM_{2.5}$ in winter is related to CCAM-A's tendency to under-predict minimum temperatures and to over-predict light winds, leading to increased wood heater emissions in the model and reduced atmospheric ventilation.

On the basis of this assessment, we selected CCAM-B as our primary model for assessment of health impacts. This choice adds conservatism to our approach, meaning we are more likely to have underestimated health impacts.

3.1.2. Modeled $PM_{2.5}$ concentrations

Fig. 1 shows the spatial relationship between the population of the Sydney GMR and anthropogenic $PM_{2.5}$. The highest concentrations were generally in areas of high population density. The notable exception was the northern part of the study region, which is an area of open-cut coal mining activity.

The spatial distribution of $PM_{2.5}$ varied by source (Fig. 2). $PM_{2.5}$ related to wood heaters and on-road vehicles was concentrated in more populated areas. $PM_{2.5}$ from power stations peaked in locations with low population density and was more evenly distributed across the entire region. $PM_{2.5}$ from ships was highest in coastal areas. $PM_{2.5}$ from



(a) Population density

(b) Anthropogenic $PM_{2.5}$ modeled using the CCAM-B configuration. The boxes show the modeling domains.

Fig. 1. The spatial distribution of (a) the population of the study region and (b) anthropogenic $PM_{2.5}$.

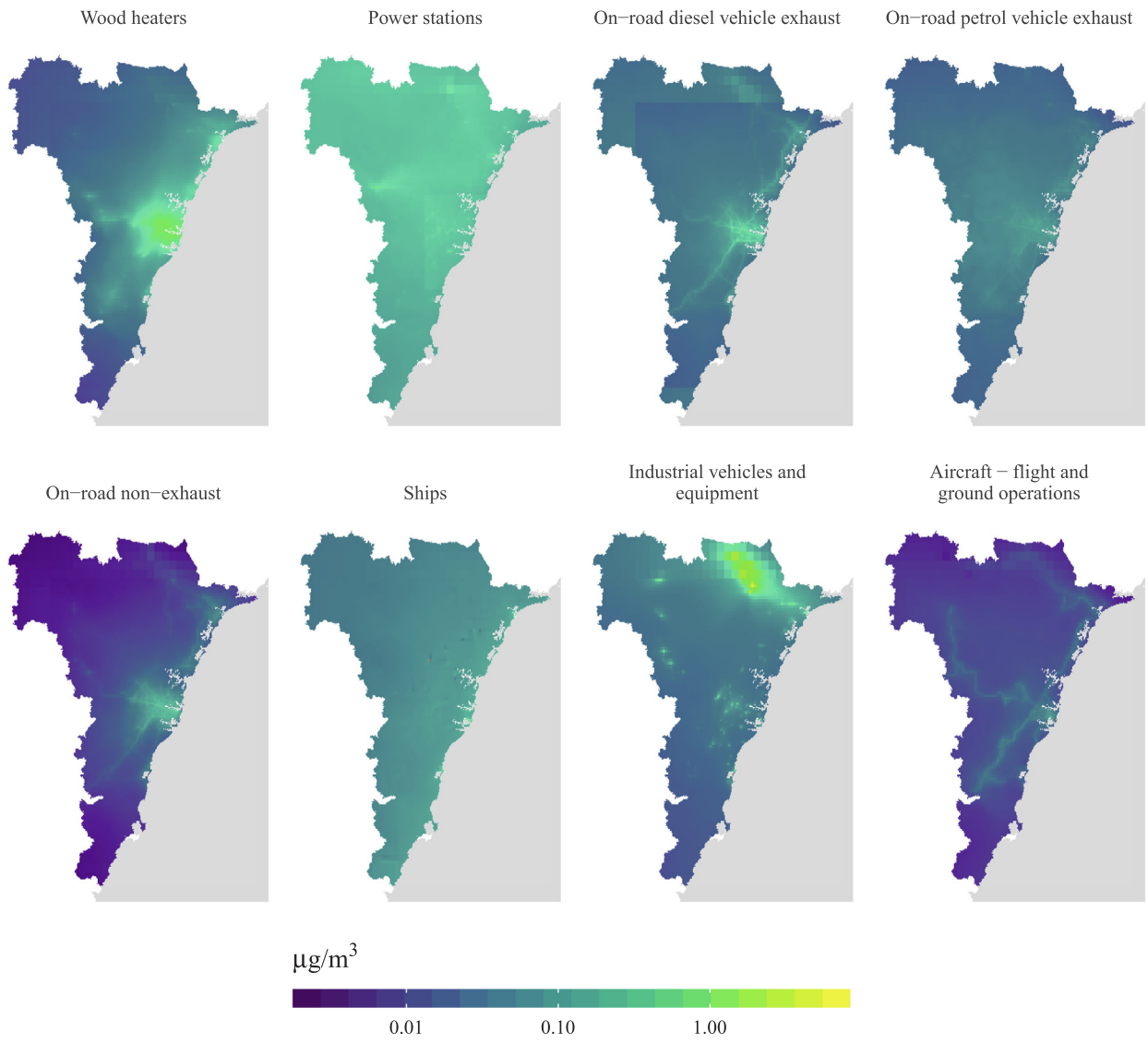


Fig. 2. The spatial distribution of source-specific $PM_{2.5}$.

Table 2
Primary $PM_{2.5}$ emissions from individual sources in 2008 and the modeled population-weighted annual average $PM_{2.5}$ (primary and secondary) in 2010/11, using the CCAM-B particle modeling framework.

Source	$PM_{2.5}$emission[a]		$PM_{2.5}$concentration[b]	
	Tonnes	%	µg/m^3	%
Wood heaters	7,400	19.0	0.49	24.0
Power stations	3,400	8.8	0.22	10.5
On-road mobile sources	2,100	5.3	0.35	16.9
Exhaust from petrol vehicle	180	0.5	0.08	4.1
Exhaust from diesel vehicles	1,100	2.9	0.16	7.6
Non-exhaust emissions	770	2.0	0.11	5.3
Off-road mobile sources	2,900	7.5	0.22	10.4
Industrial vehicles and equipment	2,000	5.2	0.06	3.0
Ships	850	2.2	0.12	5.7
Aircraft (flight and ground operations)	64	0.2	0.03	1.7
Other anthropogenic sources	16,000	41.0	1.28	38.1

[a] Emissions of primary $PM_{2.5}$ in 2008 (NSW EPA, 2012).
[b] Population-weighted annual average concentration.

industrial vehicles was heavily concentrated in the coal mining areas at the north of the Sydney GMR. These patterns were reflected in the population weighted-mean $PM_{2.5}$ concentrations (Table 2).

The total population weighted-mean annual average concentration of $PM_{2.5}$ was 5.54 µg/m^3. The anthropogenic component was 2.06 µg/m^3. The eight major sources considered in this assessment were responsible for about 61.9% of the anthropogenic concentration. Wood heaters were the most important single source of exposure (around 24.0% of the population weighted–mean anthropogenic $PM_{2.5}$ concentration), followed by on-road sources (16.9%) and power stations (10.5%).

3.2. The burden of mortality

Estimates of the burden of mortality attributable to each source are provided in Table 3. Long-term exposure to anthropogenic $PM_{2.5}$ at 2010/11 levels was responsible for around 1.2% of all mortality, equivalent to 420 premature deaths, 5,900 YLL and, if exposure were to persist at the same level over a life-time, a loss of life expectancy at birth of around 50 days. Wood-heaters were responsible for 1,400 YLL, on-road sources 990 YLL and power stations 620 YLL. The same concentration–response coefficient was used for each source, so the differences in burden are entirely related to differences in estimates of

Table 3
The annual burden of mortality related to long-term exposure to $PM_{2.5}$ from major sources in the Greater Metropolitan Region of NSW. The estimates use the Hoek et al. (2013) concentration–response coefficient and the CCAM-B particle model configuration.

Source	Attributable number of deaths		YLL		Loss of life expectancy[a]	
	N	95% CI	Years	95% CI	Days	95% CI
Wood heaters	100	67–140	1,400	920–1,900	12	8–16
Power stations	45	29–60	620	400–830	5	3–7
On-road mobile sources	72	47–97	990	650–1,300	8	6–11
Exhaust from petrol vehicle	17	11–23	240	160–320	2	1–3
Exhaust from diesel vehicles	32	21–43	440	290–600	4	2–5
Non-exhaust emissions	22	15–30	310	200–410	3	2–4
Off-road mobile sources	45	29–60	610	400–820	5	3–7
Industrial vehicles and equipment	13	8–17	180	120–240	2	1–2
Ships	25	16–33	340	220–450	3	2–4
Aircraft (flight and ground operations)	7	5–10	97	63–130	1	1–1
All anthropogenic sources	**420**	**280–570**	**5,900**	**3,800–7,900**	**50**	**33–67**

[a] Loss of life expectancy at birth.

exposure.

In sensitivity analyses, the higher concentration–response coefficient developed in Roman et al. (2008) resulted in an increase of around 58% for each measure of burden and for all sources. When the CCAM-A model was used to estimate the population-weighted concentration, our estimates of burden increased by 70% for all anthropogenic sources, 180% for wood heaters, 75% for on-road sources and 22% for off-road diesel sources. For power stations, our estimates were reduced by 11%.

3.3. Benefits of reduced emissions

Table 4 gives the estimated number of life-years produced by interventions to reduce $PM_{2.5}$ exposure compared to ongoing emissions at 2010/11 levels. Implementation of a 1.5 g/kg standard for wood heaters (real world emissions = 3.9 g/kg) would produce around 90,000 life-years among the 5.6 million people alive between July 2010 and June 2011 (95% CI 59,000–120,000), assuming that it would take 17 years to achieve the full reduction in emissions. People born in future years would also benefit from sustained reductions in emissions. When these people were taken into account (on the assumption of a constant birth rate), the number of life-years produced increases to 120,000 (95% CI 79,000–160,000). A phase-out of wood heaters over a 17 year period would produce 140,000 (95% CI 88,000–180,000) life-years among the population alive in 2010/11 and 180,000 (95% CI 120,000–250,000) life-years if those born in the future are also included.

Actions that reduce gaseous emissions from power stations would produce fewer life-years, but the potential benefits are still large. Compared to ongoing emissions at 2010/11 levels, elimination of NO_x emissions would produce 38,000 life years (95% CI 25,000–52,000) among those alive in 2010/11 and elimination of SO_x would produce 14,000 (95% CI 9,000–19,000).

4. Discussion

Our primary analysis shows that around 1.2% of all mortality in the Sydney GMR (equivalent to 5,900 YLL) is attributable to long-term exposure to anthropogenic $PM_{2.5}$. Wood heaters and power stations were responsible for the greatest burden, causing 0.3% and 0.1% of all mortality (1,400 and 620 YLL) respectively. Considered collectively, on-road sources were also important, responsible for around 0.2% of mortality (990 YLL).

The burden of mortality attributable to anthropogenic $PM_{2.5}$ and to each specific source is relatively small compared to other risk factors. The Global Burden of Disease study estimated that, for example, 14.8% of mortality in Australia in 2017 was attributable to dietary risk factors and that 11.7% was attributable to high body mass index (GBD, 2017). However, despite the relatively small burden attributable to outdoor $PM_{2.5}$, our analysis shows that interventions to reduce emissions from wood heaters and power stations could produce substantial benefit in the form of increased longevity.

Introduction of a 1.5 g/kg standard for wood heaters would produce 90,000 life-years among the 5.6 million residents of the Sydney GMR alive in 2010–11. The present monetary value of these additional 90,000 life-years is $4.1 billion (2017 Australian dollars), using the Australian Government Office of Best Practice Regulation's recommended value of a statistical life year, (OBPR, 2014) a discount rate of 3% and an assumption that it would take 17 years to achieve complete uptake of the standard. A sustained reduction in wood heater emissions would also benefit those born in the future, so the true benefit is likely to be larger. When benefits to future generations are taken into account, the present value of life-years produced rises to $4.5 billion. A previous study has estimated that it would cost government and industry $56 million to implement a 1.5 g/kg emissions standard, which suggests that introduction of a more stringent wood heater standards is likely to be highly cost-effective (Drew Collins, 2013). It should be noted that our assessment uses "real-world" estimates of wood heater emissions, which account for the fact that actual emissions from wood heaters are dependent on the manner in which they are operated.

Life-years produced by complete removal of nitrogen oxide and sulphur oxide emissions from power station have a present value of $1.8 billion and $0.66 billion respectively. These figures may underestimate of the full health benefits of power station emissions controls because exposure to NO_2 and SO_2 (components of NOx and SOx respectively) is associated with health effects that are independent of the health effects of $PM_{2.5}$ (US EPA, 2016; US EPA, 2017).

We population-weighted our modelled concentrations to account for the spatial relationship between source-specific $PM_{2.5}$ and populations. We show clearly that sources dispersed among populations have a larger health impact than those that are more distant. For example, on-road sources emitted 5.3% of primary anthropogenic $PM_{2.5}$, but were responsible for 17.0% of $PM_{2.5}$-related mortality. Conversley, industrial vehicles and equipment emitted 5.2% of primary $PM_{2.5}$, but caused 3.1% of mortality. The exception was power stations, which although generally sited outside large population centres, emitted 8.8% of primary anthropogenic $PM_{2.5}$, but were responsible for 10.5% of $PM_{2.5}$-related mortality, presumably because they are large emitters of precursors of secondary $PM_{2.5}$. This highlights the importance of health impact assessment for priority setting. In the absence of such assessment, emissions inventories may be used to prioritise intervention.

Our assessment used well established methods to predict the impact

Table 4
Life years produced by reductions in $PM_{2.5}$ emissions.

Source	Emissions scenario	Reduction in $PM_{2.5}$[a]	Life-years produced among those alive in 2011		Life-years produced among those alive in 2011 *and* people born in the future[b]	
		$\mu g/m^3$	N	95% CI	N	95% CI
Wood heaters[c]	Emissions standard of 2.5 g/kg (actual $PM_{2.5}$ emissions of 6.4 g/kg)	0.22	60,000	39,000–80,000	81,000	53,000–110,000
	Emissions standard of 1.5 g/kg (actual $PM_{2.5}$ emissions of 3.9 g/kg)	0.33	90,000	59,000–120,000	120,000	79,000–160,000
	No emissions	0.49	140,000	88,000–180,000	180,000	120,000–250,000
Power stations[d]	No nitrogen oxide emissions	0.14	38,000	25,000–52,000	52,000	34,000–69,000
	No sulphur oxide emissions	0.05	14,000	9,000–19,000	19,000	12,000–25,000

[a] Population weighted annual mean concentration.
[b] Up to 106 years in the future and assuming a constant number of births each year.
[c] Assuming a linear decline in emissions over 17 years and the US EPA cessation lag.
[d] Assuming a linear decline in emissions over 10 years and the US EPA cessation lag.

of $PM_{2.5}$ on mortality, (Broome et al., 2016; COMEAP, 2010; Fann et al., 2013; Miller and Hurley, 2003; WHO Regional Office for Europe, 2013a) but the results are affected by several sources of uncertainty. These include the particle modeling framework (illustrated by the differences in population-weighted concentrations between the CCAM-A and CCAM-B models) and the concentration–response function. There is also uncertainty in our estimates of the underlying population size and the age-specific mortality rates. It is important to note that the confidence intervals in this paper only account for the statistical imprecision in the concentration–response function.

In regard to uncertainty in the particle modeling framework, the source-specific proportions of population-weighted concentrations from our CCAM-B model are broadly consistent with estimates of source-specific concentrations from several source apportionment studies conducted in the Sydney GMR (Chan et al., 2008; Chan et al., 2011; Cohen et al., 2011; Cohen et al., 2012; Hibberd et al., 2016). These studies used chemical analysis of $PM_{2.5}$ and positive matrix factorisation to apportion particles to sources at specific monitoring locations. Collectively, they have reported that motor vehicles contributed between 8% and 33% to all $PM_{2.5}$ at the monitors and that biomass sources (including smoke for bushfires and planned hazard reduction burns, as well as smoke from solid fuel home heaters) contributed between 3% and 43%. The large variability in estimates for biomass sources in these previous studies may relate to the influence of major bushfires or hazard reduction smoke events during the monitoring period as well as spatial variation in wood-heater emissions. Estimates of the contribution of wood-heater specific emissions to total $PM_{2.5}$ during winter ranged from 11% to 45% (Cohen et al., 2011; Hibberd et al., 2016). Our study found that motor vehicles contributed around 16.9% to the population-weighted anthropogenic $PM_{2.5}$ concentration and wood heaters 24.0%. Cohen et al. (2012) estimated that power stations emissions were responsible for between 14% and 18% of all $PM_{2.5}$ at specific monitoring locations in the Sydney GMR, which was somewhat higher than our estimate of 11% of the population-weighted mean anthropogenic $PM_{2.5}$ concentration.

In general, the estimates of the absolute value of annual average anthropogenic $PM_{2.5}$ used in our primary analyses were lower than those from Sydney GMR source apportionment studies. For example, our population-weighted estimate of total anthropogenic $PM_{2.5}$ was 2.1 $\mu g/m^3$, compared to 3.9 $\mu g/m^3$ from both Cohen et al. (2011) and Cohen et al. (2012). The source-apportionment estimates are more similar to those produced by our CCAM-A model. However, as discussed in the previous section, the CCAM-B model performed better particularly in the winter months, and our population-weighted estimates have the advantage of taking into account the spatial variability of source-specific $PM_{2.5}$.

In regard to uncertainty in the concentration–response function, a recent study in Sydney found evidence of an association between long-term exposure to $PM_{2.5}$ and mortality. The magnitude of the concentration–response estimate from this study was substantially larger than the estimate we used in our primary and sensitivity analyses, but was imprecise and not statistically significant (Hanigan et al., 2019). Therefore, it was necessary to base our primary analysis on evidence from North American and European cohort studies (Hoek et al., 2013). The age structure of our study population and their pattern of mortality were similar to those of the countries where these cohort studies have been conducted and so it is reasonable to transfer the findings of these studies to our setting. One important difference is that $PM_{2.5}$ concentrations in the Sydney GMR are substantially lower than those observed in most other cities. However, there is little evidence to indicate a threshold of effect for $PM_{2.5}$, despite increasing numbers of studies with relatively low $PM_{2.5}$ concentrations (Di et al., 2017; Pinault et al., 2016). In fact, studies in regions with low $PM_{2.5}$concentrations, including the recent study in Sydney, suggest a steepening of the concentration–response curve at lower concentrations (Burnett et al., 2014; Crouse et al., 2012; Hanigan et al., 2019; Pinault et al., 2016). Our

sensitivity analysis using a concentration–response coefficient of 1% per μg of $PM_{2.5}$ shows how the choice of coefficient can have a large impact on results.

A quantitative evaluation of uncertainty was outside the scope of this project. Instead, we have aimed to be transparent about our modeling decisions and assumptions, which is relatively rare in the impact assessment literature. Overall, our approach for the primary analyses was to use assumptions that would lead to underestimation of impacts. Therefore, we can be reasonably confident that the true impacts are at least as large as those reported here. Our sensitivity analyses suggest that the true impacts could be substantially larger.

This paper quantifies the burden of mortality attributable to $PM_{2.5}$ from specific emission sources. It shows that wood heaters, on-road vehicles and power stations are collectively responsible for more than 50% of $PM_{2.5}$-related mortality. Even though the burden of mortality attributable to $PM_{2.5}$ is small relative to a number of other mortality risk factors, actions that achieve a sustained reduction in emissions from individual sources are likely to be highly beneficial.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.envint.2019.105429.

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