Article

Avoidable Mortality Attributable to Anthropogenic Fine Particulate Matter (PM$_{2.5}$) in Australia

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Abstract: Ambient fine particulate matter <2.5 μm (PM$_{2.5}$) air pollution increases premature mortality globally. Some PM$_{2.5}$ is natural, but anthropogenic PM$_{2.5}$ is comparatively avoidable. We determined the impact of long-term exposures to the anthropogenic PM component on mortality in Australia. PM$_{2.5}$-attributable deaths were calculated for all Australian Statistical Area 2 (SA2; n = 2310) regions. All-cause death rates from Australian mortality and population databases were combined with annual anthropogenic PM$_{2.5}$ exposures for the years 2006–2016. Relative risk estimates were derived from the literature. Population-weighted average PM$_{2.5}$ concentrations were estimated in each SA2 using a satellite and land use regression model for Australia. PM$_{2.5}$-attributable mortality was calculated using a health-impact assessment methodology with life tables and all-cause death rates. The changes in life expectancy (LE) from birth, years of life lost (YLL), and economic cost of lost life years were calculated using the 2019 value of a statistical life. Nationally, long-term population-weighted average total and anthropogenic PM$_{2.5}$ concentrations were 6.5 μg/m^3 (min 1.2–max 14.2) and 3.2 μg/m^3 (min 0–max 9.5), respectively. Annually, anthropogenic PM$_{2.5}$-pollution is associated with 2616 (95% confidence intervals 1712, 3455) deaths, corresponding to a 0.2-year (95% CI 0.14, 0.28) reduction in LE for children aged 0–4 years, 38,962 (95%CI 25,391, 51,669) YLL and an average annual economic burden of $6.2 billion (95%CI $4.0 billion, $8.1 billion). We conclude that the anthropogenic PM$_{2.5}$-related costs of mortality in Australia are higher than community standards should allow, and reductions in emissions are recommended to achieve avoidable mortality.

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1. Introduction

Long-term exposure to ambient air pollution is an established risk factor for a range of cardiovascular and respiratory diseases, contributing to premature mortality and reductions in life expectancy (LE) [1]. Demonstrated nonfatal health associations of air pollution include increased rates of hospitalisation [2], birth defects [3], impaired cognitive function [4], and increased medication usage [5].

Air pollutants that affect health include fine particulate matter (PM) < 2.5 μm ($PM_{2.5}$) in aerodynamic diameter [1]. $PM_{2.5}$ can be emitted from combustion or other processes (primary $PM_{2.5}$) or can be produced via chemical reactions of precursor emissions (secondary $PM_{2.5}$). As an ambient exposure, $PM_{2.5}$ can reach indoor environments and, under certain atmospheric conditions, can travel long distances over several days [6]. Current evidence suggests that there is no safe lower threshold of exposure to $PM_{2.5}$ for mortality because the exposure–response relationship is approximated by a linear function even at very low concentrations [7]. Nonetheless, this remains a strong assumption due to the lack of knowledge about the shape of the exposure–response association at these lower levels [8].

Natural $PM_{2.5}$ includes wind-blown dust, sea salt, organic aerosol from biogenic sources, and emissions from volcanoes and landscape fires. Given the spatial heterogeneity of PM from these sources and the influence of rainfall and wind on their local concentrations, natural $PM_{2.5}$ concentrations vary widely between locations and over time. Anthropogenic sources of $PM_{2.5}$ are responsible for substantial human exposure, emanating from transport and industrial processes such as mining and power generation. Residential wood heaters are also a major source of PM, for example they accounted for 19% of anthropogenic $PM_{2.5}$ emissions and 24% of $PM_{2.5}$ concentrations in Sydney, Australia, during 2010 and 2011 [9]. Although these sources of $PM_{2.5}$ might be expected to increase with population growth and increased economic activity [10], air-pollution control policies have effectively reduced $PM_{2.5}$ concentrations in some high-income countries, as shown by Carnell et al. in the United Kingdom [11]. Several studies show health benefits of reducing anthropogenic $PM_{2.5}$, as reviewed by Rich [12].

Only one study has related all-cause mortality to long-term exposure to $PM_{2.5}$ in the general population in Australia [7], although it was not possible to distinguish between risks from anthropogenic and natural PM sources. Health-impact assessments quantifying the effects on mortality of $PM_{2.5}$ from shipping emissions [13] as well as from wood heaters, traffic, and industrial activities [9] have demonstrated that years of life lost (YLL) and LE offer sensitive indicators of health burdens in Australia. These statistics can also be used to calculate economic costs [14].

Given the ubiquitous but modifiable nature of exposures to anthropogenic $PM_{2.5}$, robust estimates of human-health impacts could be used to inform air-pollution control policies. Herein, we quantified the effect of current levels of anthropogenic $PM_{2.5}$ on mortality in Australia in terms of $PM_{2.5}$-attributable mortality, changes in LE for children, and the economic costs of the associated YLL.

2. Materials and Methods

2.1. Study Region and Period

We calculated the effect of anthropogenic $PM_{2.5}$ on mortality in all 2310 Statistical Area level 2 (SA2) geographical areas in Australia with age-specific population counts from the Australian Bureau of Statistics (ABS) dataset "Population by Age and Sex, Regions of Australia, Estimated Residential Population 2006–2016" from ABS-TableBuilder (cat. no. 3235.0). We then aggregated attributable numbers of premature deaths, YLL and changes of LE for the entire population of Australia. We chose to start the study period in

2006 to coincide with that census year. $PM_{2.5}$ levels were relatively stable in most states and territories except NT and QLD for the study period 2006–2016 (Figure 1).
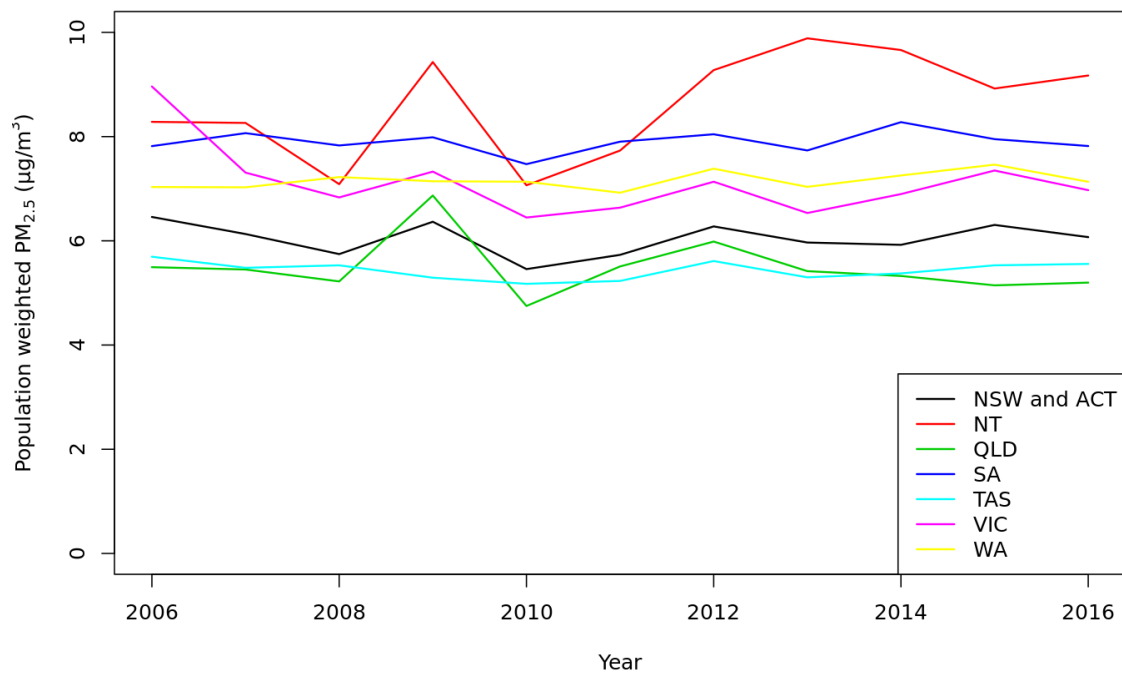

Figure 1. Time-series of population weighted ambient particulate matter ($PM_{2.5}$) concentrations ($\mu g/m^3$) across the years 2006–2016 for all Australian states—NSW, New South Wales; ACT, Australian Capital Territory; NT, Northern Territory; QLD, Queensland; SA, South Australia; TAS, Tasmania; VIC, Victoria; WA, Western Australia.

2.2. Assessment of Anthropogenic vs. Non-Anthropogenic $PM_{2.5}$ Concentrations

Annual average $PM_{2.5}$ concentrations were obtained from a validated satellite-based land-use regression (LUR) model, as described by Knibbs et al. [15]. The regression model uses satellite imagery, chemical-transport model (CTM) simulations and land-use data as predictors, and incorporates direct $PM_{2.5}$ measurements from ambient-air monitoring agencies in Australia [15]. The data are available on request from the Australian Centre for Air pollution, energy and health Research (CAR) https://cloudstor.aarnet.edu.au/plus/f/2454567279. The model was estimated for each mesh-block (MB), which is the smallest area in the Census geography (Figure 2). It is not possible to show MBs in Figure 2 due to the difference in spatial scale. Instead, we have added a small area map to the Supporting Information (Figure S1) to demonstrate the small sizes of MB regions, which enable high spatial resolution in our exposure assessment. Anthropogenic $PM_{2.5}$ was defined as the difference between estimated $PM_{2.5}$ concentrations and the 5th percentile of concentrations for all MBs in each state/territory per year. This definition accommodates differences in natural background concentrations between states/territories due to localised influences such as bushfire, dust, and sea salt in the diverse landscapes across the country. This approach is consistent with that taken by the global burden of disease study for estimating the counterfactual level of $PM_{2.5}$ [1].

To validate our state/territory estimates of natural $PM_{2.5}$, we performed sensitivity analyses using 5th percentile $PM_{2.5}$ concentrations for all MBs in Bureau of Meteorology climate zones (Supporting Information S8). This approach gave similar results.
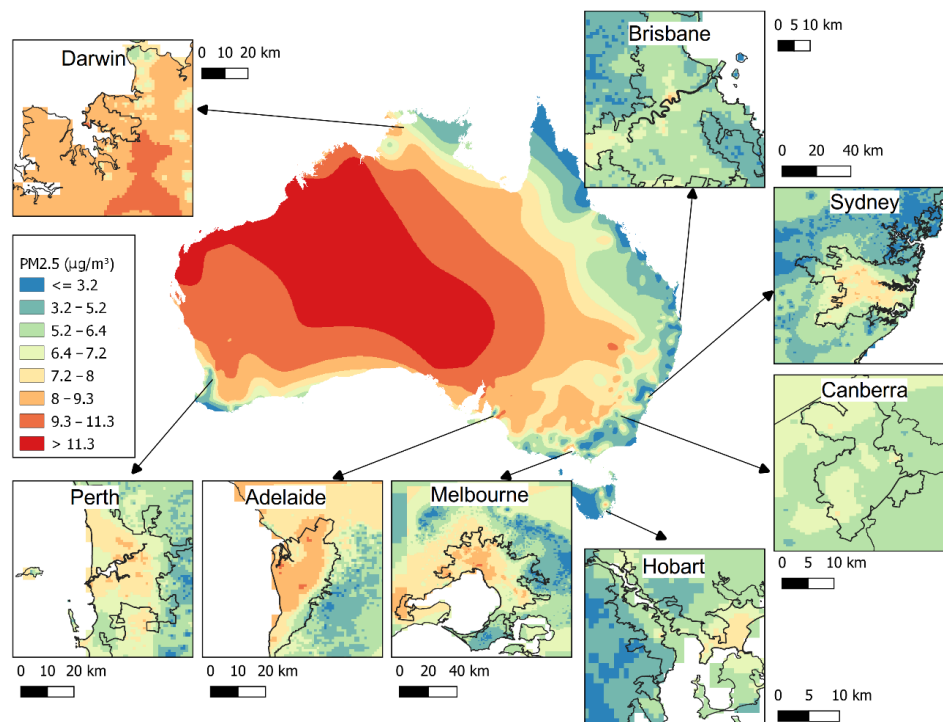
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Figure 2. Map of Australia showing modelled estimates of annual average PM$_{2.5}$ (μg/m^3) concentrations in 2015.

2.3. Health Outcomes

Mortality data for years 2006–2016 by age and state/territory and corresponding population data were accessed from the Australian Bureau of Statistics (Cat. No. 3302.0—Deaths, Australia, available from the ABS.Stat website: http://stat.data.abs.gov.au). Further information about these data sources is presented in the supporting information. No ethics approvals were needed because we used aggregated data from the public domain.

Age-specific death rates for each state/territory and year were linked with the age-specific populations by year within 2016 ABS SA2 geographical boundaries to calculate baseline mortality levels in each subpopulation. To smooth excess variability in annual deaths, we used three-year rolling average annual age-specific rates.

2.4. Quantification of Mortality Attributable to Anthropogenic PM$_{2.5}$

Due to the limited number of Australian epidemiological studies of long term PM$_{2.5}$ air pollution exposure and mortality, we used a relative risk (RR) function estimated from a meta-analysis of European and North American studies [16], as recommended by the World Health Organization (WHO) [17]. A pooled RR of 1.062 (95% CI 1.041, 1.084) per 10-μg/m^3 increments in long term annual average PM$_{2.5}$ exposures of people aged ≥30 years is recommended for health-impact assessments of PM$_{2.5}$ [16,17]. The reviewed studies were performed in countries with similar levels of economic development, similar demographic characteristics, and similar patterns of mortality as those in Australia, albeit with higher air-pollution concentrations [16]. We used this RR to estimate the attributable numbers (AN) of deaths caused by long-term PM$_{2.5}$ exposure for each SA2. We calculated AN based on estimates of local anthropogenic PM$_{2.5}$ and then aggregated to a national total using the following equation:

$$AN = \sum \left(1 - e^{(-\beta \Delta X_{ij})}\right) \times Expected_{ij} \tag{1}$$

where $Expected_{ij}$ is the death count estimated by applying mortality rate in age-group i by age-specific population counts in SA2 2016 census area j, $\beta = \log(RR)/10$ and ΔX_{ij} is the annual anthropogenic PM$_{2.5}$ concentration in SA2 j.

2.5. Life Expectancy Calculations

Life tables were generated for each year in each SA2, and LE at birth was calculated for 5-year age groups up to age 85-plus. To quantitatively assess the health impact of anthropogenic $PM_{2.5}$, LE for a hypothetical counterfactual population without anthropogenic $PM_{2.5}$ was calculated by subtracting $PM_{2.5}$-attributable numbers of deaths in each age group from expected numbers of deaths, as described by Miller and Hurley [18]. See supporting information for more details (Supporting information Sections S5 and S6).

2.6. Economic Valuation

To determine the economic value of removing all anthropogenic $PM_{2.5}$ in Australia, we discounted the 2019 willingness-to-pay value of a statistical life year (VSLY = $213,000) by 3% annually [19] and summed for each of the remaining potential life years in each age group. The resulting age-specific value of statistical life (VSL) estimates were then multiplied by corresponding attributable numbers of deaths (averaged for the years 2006–2016) and were summed across all age groups. For more details see Supporting information Section S7.

Data preparation and analyses were performed using the R language and environment for statistical computing (version 3.4.4, R Core Team Vienna, R Foundation for Statistical Computing, Vienna, Austria) and MS Excel (Microsoft, Redmond, Washington, DC, USA).

3. Results

3.1. Exposure Assessment

Nationally, the long-term population weighted average $PM_{2.5}$ concentration across the years 2006–2016 was 6.5 μg/m^3 (MB min 1.2–max 14.2), and the anthropogenic component was 3.2 μg/m^3 (MB min 0–max 9.5). Figure 1 shows the modelled 2015 estimates of the annual average $PM_{2.5}$ in ABS MBs across the country and in the major cities. $PM_{2.5}$ concentrations clearly vary across Australia, reflecting the various natural and anthropogenic contributors to ambient PM, such as dust, sea salt, bushfire smoke, and emissions from transport, industry, agriculture, and residential wood heaters.

Table 1 shows estimated average anthropogenic, non-anthropogenic and total $PM_{2.5}$ concentrations in Australia based on the 5th percentile MB level of each state/territory for each year. Average total $PM_{2.5}$ for the entire country varied little between years (Table 1) but differed markedly between states (Figure 2), reflecting diverse ecological conditions across the country. In Queensland and the Northern Territory, $PM_{2.5}$ estimates varied considerably over the study period, due to droughts, floods, and landscape fires (dust storms, bushfire, and controlled burns). In contrast, population-weighted anthropogenic $PM_{2.5}$ concentrations varied little between years, indicating similar pollution sources in Australian capital cities, where most people live.

Table 1. Estimated population-weighted national anthropogenic, non-anthropogenic and total $PM_{2.5}$ (μg/m^3) by year.

Year	Anthropogenic $PM_{2.5}$	Natural $PM_{2.5}$	Total $PM_{2.5}$
2006	3.2	3.9	7.1
2007	3.1	3.5	6.6
2008	3.2	3.1	6.3
2009	3.2	3.7	6.9
2010	3.1	2.8	5.9
2011	3.1	3.1	6.2
2012	3.2	3.6	6.7
2013	3.2	3.1	6.3
2014	3.2	3.2	6.4
2015	3.2	3.4	6.6
2016	3.1	3.3	6.4

3.2. Mortality Burden

We estimate an average annual mortality burden of 38,962 (95%CI 25,391, 51,669) YLL among people aged 30+ years attributed to anthropogenic $PM_{2.5}$ pollution in Australia between 2006–2016. This is approximately 2% of all mortality or 2616 (95%CI 1712, 3455) deaths. In Table 2, we present annual average mortality burdens in each Australian state and territory. These analyses show that more than 80% of premature deaths occurred in the more populous eastern states New South Wales (NSW), Australian Capital Territory (ACT), Victoria (VIC), and Queensland (QLD).

Table 2. Average annual mortality burden; Attributable Number (AN) of premature deaths and Years of Life Lost (YLL) in each Australian state and territory; NSW, New South Wales; ACT, Australian Capital Territory; VIC, Victoria' QLD, Queensland; WA, Western Australia; SA, South Australia; NT, Northern Territory.

Region	AN (95%CI)	YLL (95%CI)
NSW and ACT	940 (615–1241)	13,956 (9094–18509)
VIC	650 (425–858)	9366 (6103–12421)
QLD	517 (338–682)	7925 (5165–10509)
WA	197 (129–260)	3178 (2072–4213)
SA	188 (123–249)	2653 (1729–3518)
TAS	102 (67–135)	1419 (925–1882)
NT	21 (14–28)	464 (303–615)
National	2616 (1712–3455)	38,962 (25391–51669)

Based on ANs among Australians of 30+ years-of-age, we estimate that LE among children <5 years-of-age was reduced by 76 (95%CI 50, 101) days due to anthropogenic $PM_{2.5}$ (assuming lifelong exposures). Using the 2019 VSLY of $213,000 with an annual social discount rate of 3% [19], we calculated the value of a statistical life (VSL) for each age group based on remaining LE and estimated an average annual mortality-related cost of anthropogenic $PM_{2.5}$ of $6.2 billion nationally (95%CI $4.0 billion, $8.1 billion).

4. Discussion

In this study, we estimate that the mortality burden of anthropogenic emissions of $PM_{2.5}$ in Australia was 2616 excess deaths per year on average (approximately 2% of total mortality), and 38,962 YLL were attributable. In an Australian study from 2016 [13], $PM_{2.5}$ from shipping activities, which use low-quality diesel fuel, were responsible for the loss of 220 years of life among people who died in 2010/11 in the greater metropolitan area (GMR) of Sydney. In a more recent study, 1.2% of all-cause mortality in the greater Sydney metropolitan area was attributed to $PM_{2.5}$ from all anthropogenic sources, corresponding with 5900 YLL annually [9]. In the same study, $PM_{2.5}$ concentrations were estimated using a chemical-transport model (CTM) of eight anthropogenic sources; the total population weighted $PM_{2.5}$ concentration was 5.5 μg/m^3 with an anthropogenic component of 2.1 μg/m^3. Our estimates for Sydney are consistent with this anthropogenic proportion, and our estimates of YLL were comparable (data not shown).

Our estimated annual mortality-related cost of anthropogenic $PM_{2.5}$ in Australia was $6.2 billion (95%CI $4.0 billion, $8.1 billion) nationally in 2019 dollars. This is supported by a recent estimate for the special report of the MJA-Lancet Countdown [20], which found that urban $PM_{2.5}$ costs equated to $5.3 billion in 2015 dollars. This is similar to our estimate after adjusting for inflation; however, our methods for exposure assessment and economic valuation with discounting were different. Therefore, comparisons of estimated health-cost estimates should be made with caution. Despite the relatively low levels of air pollution in Australia, the substantial health burden is of public health concern, both in societal and economic terms.

Increased anthropogenic emissions have been associated with increased industrial and economic activities [10], suggesting that concentrations of many pollutants will increase

globally over the coming decades without substantial decreases in fossil fuel and biomass combustion [21]. However, economic development has been decoupled from increasing anthropogenic air-pollution emissions in some countries [22] where clean-air policies have been implemented [23]. Moreover, we found no increases in anthropogenic $PM_{2.5}$ over the period 2006–2016 in Australia.

Lelieveld, et al. [8] assessed global $PM_{2.5}$ concentrations and found global mean LE would increase by 1.7 (1.4–2.0) years if all potentially controllable anthropogenic emissions were removed. They estimated total lost LE from air pollution was 2.9 years, exceeded that of smoking (2.2 years of lost LE) [8]. Our estimated loss of life expectancy of 76 days (0.2 years) is similar to that found in [8] for Australia/Oceania combined, but is lower than the global average estimate (1.7 years), due to the lower exposure levels and related mortality rates in Australia. In another worldwide study by the Global Burden of Disease (GBD) 2019 project [24], 1625 (95%UI 508, 2877) deaths were attributed to ambient particulate air pollution in Australia in 2016, whereas our estimate was 2616 (95%CI 1712, 3455) for the period 2006–2016 (data available from Institute for Health Metrics and Evaluation (IHME) website http://ghdx.healthdata.org/gbd-results-tool). Our estimate is 60% higher than that from the GBD study. This difference can be explained by differing datasets used for exposure and death rates, and different exposure–response risk functions and counterfactual exposure.

For context, in a study of Australian smokers in NSW, individual LE of heavy smokers was reduced by 10 years [25]. Given the widespread exposure to anthropogenic $PM_{2.5}$, compared with that of heavy smoking, the population impact may be substantial. For example, the GBD report from 2020 ranked air pollution as the 4th highest risk factor for mortality, with 6.67 million attributable deaths during the period 1990–2019 [24].

We used high-resolution air-pollution models that were informed by monitor data, land-use data and satellite imaging. However, among limitations of this study, we did not analyse regions within states because mortality rates for small areas were not publicly available. Moreover, $PM_{2.5}$ is associated with a broad range of health effects, such as low birth weight and respiratory illnesses, that increase hospitalisation and general-practice visits. The costs of these are not captured by our VSLY estimates, thus only calculating the mortality burden will underestimate the overall impacts of $PM_{2.5}$ pollution on public health and health services. The present study is also limited by the absence of locally derived RR with only one cohort study published [7], and so we applied the RR from a meta-analysis as recommended by the WHO "Health risks of air pollution in Europe—HRAPIE project" recommendations [17]. However, a new meta-analysis has recently been published that found support for a higher RR (1.08), which we have included as a sensitivity analysis [26]. As expected, this showed an increase to our estimated health burden and costs, and supported our conclusion that the burden is substantial. In addition, the global exposure mortality model (GEMM), a non-linear exposure–response function that employs a low minimum-risk threshold was used by Lelieveld, et al. [8]. However, the two meta-analyses [17,26] support our application of a linear RR in the Australian context. A further limitation to our study was the lack of estimates of natural $PM_{2.5}$. We approximated these using the 5th percentile threshold, which is likely to vary less than in reality, but probably overestimates baseline $PM_{2.5}$. This limitation will only be addressed when natural $PM_{2.5}$ estimates from a suitable model are available for the entire country.

5. Conclusions

Our findings present some clear implications for policymakers. The estimated burden of premature death attributable to anthropogenic $PM_{2.5}$ shows that this environmental risk factor has a significant impact on public health in Australia, and the health benefits of exposure reductions have been demonstrated in multiple studies. Although ambient annual average $PM_{2.5}$ concentrations have remained relatively stable in major cities in Australia over the past several years, the exposure level is increasing due to increases in the population. Hence, given that the $PM_{2.5}$ exposure–response relationship appears

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to be linear at the low levels found in Australian cities, $PM_{2.5}$ reporting standards that prioritise continual reductions in $PM_{2.5}$ pollution are urgently required. These are likely to require reductions in emissions of primary $PM_{2.5}$ and of secondary $PM_{2.5}$ precursors across multiple sectors (road transport, domestic heating, industry, and agriculture). Health impact assessments such as this can inform decision-making for urban developments, the energy system, and future studies that assess the costs and benefits of anthropogenic air pollution.

Supplementary Materials: The following are available online at https://www.mdpi.com/1660-460 1/18/1/254/s1, Figure S1: Average $PM_{2.5}$ in 2015 across the country and inset maps of the Sydney region and the small case study region in Western Sydney. Figure S2: Climate zones from Bureau of Meteorology rainfall levels. Figure S3: Map of Australia showing modelled estimates of annual average PM2.5 (µg/m3) concentrations in 2015. Climate boundaries are marked.

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References

1. Cohen, A.J.; Brauer, M.; Burnett, R.; Anderson, H.R.; Frostad, J.; Estep, K.; Balakrishnan, K.; Brunekreef, B.; Dandona, L.; Dandona, R.; et al. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: An analysis of data from the Global Burden of Diseases Study 2015. *Lancet* **2017**, *389*, 1907–1918. [CrossRef]
2. Shah, A.S.V.; Langrish, J.P.; Nair, H.; A McAllister, D.; Hunter, A.L.; Donaldson, K.; E Newby, D.; Mills, N.L. Global association of air pollution and heart failure: A systematic review and meta-analysis. *Lancet* **2013**, *382*, 1039–1048. [CrossRef]
3. Hansen, C.A.; Barnett, A.G.; Jalaludin, B.B.; Morgan, G.G. Ambient Air Pollution and Birth Defects in Brisbane, Australia. *PLoS ONE* **2009**, *4*, e5408. [CrossRef] [PubMed]
4. Clifford, A.; Lang, L.; Chen, R.; Anstey, K.J.; Seaton, A. Exposure to air pollution and cognitive functioning across the life course—A systematic literature review. *Environ. Res.* **2016**, *147*, 383–398. [CrossRef] [PubMed]
5. Peters, A.; Dockery, D.W.; Heinrich, J.; E Wichmann, H. Medication use modifies the health effects of particulate sulfate air pollution in children with asthma. *Environ. Health Perspect.* **1997**, *105*, 430–435. [CrossRef]
6. Kollanus, V.; Tiittanen, P.; Niemi, J.V.; Lanki, T. Effects of long-range transported air pollution from vegetation fires on daily mortality and hospital admissions in the Helsinki metropolitan area, Finland. *Environ. Res.* **2016**, *151*, 351–358. [CrossRef]
7. Hanigan, I.; Rolfe, M.I.; Knibbs, L.D.; Salimi, F.; Cowie, C.T.; Heyworth, J.; Marks, G.B.; Guo, Y.; Cope, M.; Bauman, A.; et al. All-cause mortality and long-term exposure to low level air pollution in the '45 and up study' cohort, Sydney, Australia, 2006–2015. *Environ. Int.* **2019**, *126*, 762–770. [CrossRef]
8. Lelieveld, J.; Pozzer, A.; Pöschl, U.; Fnais, M.; Haines, A.; Münzel, T. Loss of life expectancy from air pollution compared to other risk factors: A worldwide perspective. *Cardiovasc. Res.* **2020**, *116*, 1910–1917. [CrossRef]
9. Broome, R.; Powell, J.; Cope, M.E.; Morgan, G.G. The mortality effect of PM2.5 sources in the Greater Metropolitan Region of Sydney, Australia. *Environ. Int.* **2020**, *137*, 105429. [CrossRef]

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10. Kulkarni, S.; Sobhani, N.; Miller-Schulze, J.P.; Shafer, M.M.; Schauer, J.J.; Solomon, P.A.; Saide, P.; Spak, S.N.; Cheng, Y.; Van Der Gon, H.A.C.D.; et al. Source sector and region contributions to BC and PM2.5 in Central Asia. *Atmos. Chem. Phys. Discuss.* **2014**, *14*, 11343–11392. [CrossRef]

11. Carnell, E.J.; Vieno, M.; Vardoulakis, S.; Beck, R.C.; Heaviside, C.; Tomlinson, S.J.; Dragosits, U.; Heal, M.R.; Reis, S. Modelling public health improvements as a result of air pollution control policies in the UK over four decades—1970 to 2010. *Environ. Res. Lett.* **2019**, *14*, 074001. [CrossRef]

12. Rich, D.Q. Accountability studies of air pollution and health effects: Lessons learned and recommendations for future natural experiment opportunities. *Environ. Int.* **2017**, *100*, 62–78. [CrossRef] [PubMed]

13. Broome, R.A.; Cope, M.E.; Goldsworthy, B.; Goldsworthy, L.; Emmerson, K.; Jegasothy, E.; Morgan, G.G. The mortality effect of ship-related fine particulate matter in the Sydney greater metropolitan region of NSW, Australia. *Environ. Int.* **2016**, *87*, 85–93. [CrossRef] [PubMed]

14. Robinson, L.A.; Hammitt, J.K.; O'Keeffe, L. Valuing Mortality Risk Reductions in Global Benefit-Cost Analysis. *J. Benefit-Cost Anal.* **2019**, *10*, 15–50. [CrossRef] [PubMed]

15. Knibbs, L.D.; Van Donkelaar, A.; Martin, R.V.; Bechle, M.J.; Brauer, M.; Cohen, D.D.; Cowie, C.T.; Dirgawati, M.; Guo, Y.; Hanigan, I.C.; et al. Satellite-Based Land-Use Regression for Continental-Scale Long-Term Ambient $PM_{2.5}$ Exposure Assessment in Australia. *Environ. Sci. Technol.* **2018**, *52*, 12445–12455. [CrossRef] [PubMed]

16. Hoek, G.; Krishnan, R.M.; Beelen, R.; Peters, A.; Ostro, B.; Brunekreef, B.; Kaufman, J.D. Long-term air pollution exposure and cardio- respiratory mortality: A review. *Environ. Health* **2013**, *12*, 43. [CrossRef]

17. WHO. *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: Geneva, Switzerland, 2013.

18. Miller, B.G.; Hurley, J. Life table methods for quantitative impact assessments in chronic mortality. *J. Epidemiology Community Health* **2003**, *57*, 200–206. [CrossRef]

19. Australian Government Office of Best Practice Regulation. Best Practice Regulation Guidance Note Value of a statistical life Guidance Note 2019. Available online: https://www.pmc.gov.au/sites/default/files/publications/value-of-statistical-life-guidance-note_0_0.pdf (accessed on 31 June 2020).

20. Zhang, Y.; Beggs, P.; McGushin, A. The 2020 special report of the MJA-Lancet Countdown on health and climate change: Lessons learnt from Australia's 'Black Summer'. *Med. J. Aust.* **2021**, *213*, 490–492. [CrossRef]

21. Van Vuuren, D.; Edmonds, J.; Kainuma, M.; Riahi, K.; Thomson, A.; Hibbard, K.; Hurtt, G.C.; Kram, T.; Krey, V.; Lamarque, J.-F.; et al. The representative concentration pathways: An overview. *Clim. Chang.* **2011**, *109*, 5–31. [CrossRef]

22. Zheng, Y.; Xue, T.; Zhang, Q.; Geng, G.; Tong, D.; Li, X.; He, K. Air quality improvements and health benefits from China's clean air action since 2013. *Environ. Res. Lett.* **2017**, *12*, 114020. [CrossRef]

23. Zhao, Y.; Nielsen, C.P.; Lei, Y. Quantifying the uncertainties of a bottom-up emission inventory of anthropogenic atmospheric pollutants in China. *Atmos. Chem. Phys.* **2011**, *46*, 3973–3980. [CrossRef]

24. Abbafati, C.; Machado, D.B.; Cislaghi, B. Global burden of 87 risk factors in 204 countries and territories, 1990–2019: A systematic analysis for the Global Burden of Disease Study 2019. *Lancet* **2020**, *396*, 10258.

25. Banks, E.; Joshy, G.; Weber, M.F.; Liu, B.; Grenfell, R.; Egger, S.J.; Paige, E.; Lopez, A.D.; Sitas, F.; Beral, V. Tobacco smoking and all-cause mortality in a large Australian cohort study: Findings from a mature epidemic with current low smoking prevalence. *BMC Med.* **2015**, *13*, 1–10. [CrossRef] [PubMed]

26. Chen, J.; Hoek, G. Long-term exposure to PM and all-cause and cause-specific mortality: A systematic review and meta-analysis. *Environ. Int.* **2020**, *143*, 105974. [CrossRef]