Environmental Research 143 (2015) 19–25

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

The health benefits of reducing air pollution in Sydney, Australia

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ARTICLE INFO

Article history:
Received 19 May 2015
Received in revised form
11 August 2015
Accepted 7 September 2015
Available online 27 September 2015

Keywords:
$PM_{2.5}$
Ozone
Sydney
Australia
Health impact
Environmental benefits mapping andanalysis program – community edition
Fine particles

ABSTRACT

Among industrialised countries, fine particle ($PM_{2.5}$) and ozone levels in the Sydney metropolitan area of Australia are relatively low. Annual mean $PM_{2.5}$ levels have historically remained below 8 μg/m^3 while warm season (November–March) ozone levels occasionally exceed the Australian guideline value of 0.10 ppm (daily 1 h max). Yet, these levels are still below those seen in the United States and Europe. This analysis focuses on two related questions: (1) what is the public health burden associated with air pollution in Sydney; and (2) to what extent would reducing air pollution reduce the number of hospital admissions, premature deaths and number of years of life lost (YLL)? We addressed these questions by applying a damage function approach to Sydney population, health, $PM_{2.5}$ and ozone data for 2007 within the BenMAP-CE software tool to estimate health impacts and economic benefits. We found that 430 premature deaths (90% CI: 310–540) and 5800 YLL (95% CI: 3900–7600) are attributable to 2007 levels of $PM_{2.5}$ (about 2% of total deaths and 1.8% of YLL in 2007). We also estimate about 630 (95% CI: 410–840) respiratory and cardiovascular hospital admissions attributable to 2007 $PM_{2.5}$ and ozone exposures. Reducing air pollution levels by even a small amount will yield a range of health benefits. Reducing 2007 $PM_{2.5}$ exposure in Sydney by 10% would, over 10 years, result in about 650 (95% CI: 430–850) fewer premature deaths, a gain of 3500 (95% CI: 2300–4600) life-years and about 700 (95% CI: 450–930) fewer respiratory and cardiovascular hospital visits. These results suggest that substantial health benefits are attainable in Sydney with even modest reductions in air pollution.

1. Introduction

The risks to human health of even low levels of ambient air pollution are well established in the epidemiological, clinical and toxicological literature (Burnett et al., 2014; Lim et al., 2012; US EPA, 2009). Exposure to particles less than 2.5 μm in diameter ($PM_{2.5}$) and photochemical oxidants, measured as ozone (O_3), are of particular concern as these pollutants are linked to increased risk of premature death and acute and chronic morbidity. Epidemiological studies have shown that long-term exposure to $PM_{2.5}$ is associated with increased cardiopulmonary mortality (Cesaroni et al., 2013; Krewski et al., 2009; Pope et al., 2004; Pope, 2002; Schwartz et al., 2008) and short-term exposure is associated with increased daily mortality and hospital admissions (Katsouyanni et al., 2009; Simpson et al., 2005). More recently, reductions in $PM_{2.5}$ have been associated with longer life-expectancy at birth in the U.S. (Correia et al., 2013; Pope et al., 2009). Exposure to ozone has been associated with increased daily mortality, reduced survival and morbidity (Bell et al., 2004; Ito et al., 2005; Jerrett et al., 2009; Levy et al., 2005a).

Compared to cities in other industrialised countries, air pollution levels in Sydney (population approximately 4.6 million) are relatively low. Monitored annual mean $PM_{2.5}$ concentrations are generally below the 8 μg/m^3 Australian annual advisory standard, while the 1-h ozone standard (0.10 ppm) was exceeded an average of eight days each year between 1994 and 2011 and the 4-h standard (0.08 ppm) was exceeded an average of 11 days each year. Although Sydney levels of $PM_{2.5}$ and ozone are below both European the United States air quality standards, air quality in Europe and the USA has been improving in recent decades, while $PM_{2.5}$ and ozone levels in Sydney have remained relatively static (European Environment Agency, 2014; Pope et al., 2009).

In Sydney, domestic solid fuel burning was the largest source of

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http://dx.doi.org/10.1016/j.envres.2015.09.007
0013-9351/

$PM_{2.5}$ emissions in 2008, responsible for 51% of all emissions. The second and third largest sources were non-exhaust PM from on-road mobile sources (5.5%) and on-road heavy duty diesel exhaust (5.3%). In regard to precursors of ozone, exhaust from on-road vehicles (gasoline and diesel) accounted for 58% of emissions while domestic and commercial solvents were the largest source of volatile organic compounds (20%) (NSW EPA, 2008). Estimates of the burden of disease provide an indication of the scale of the air pollution problem. Estimates of the benefits associated with reductions in ambient $PM_{2.5}$ and ozone support the design of air pollution control strategies. One such strategy currently under review is the Australian National Environment Protection (Ambient Air Quality) Measure (NEPM) for outdoor air pollution. As part of this review, the government is considering a compliance standard for $PM_{2.5}$ and a framework to encourage reductions in population exposure, even where $PM_{2.5}$ levels are below the standard. Specific control strategies that could reduce air pollution in Sydney include increased regulation of non-road diesel engines and wood-burning heaters (NSW Environment Protection Authority, 2010; National Environment Protection Council, 2013a).

In this assessment, we quantify: (1) the burden of disease attributable to recent levels of anthropogenic (human made) $PM_{2.5}$ and ozone in Sydney in terms of hospital admissions, mortality and years of life lost (YLL); and (2) the potential health benefits to the Sydney population of reductions in $PM_{2.5}$ and ozone.

Estimates of the burden of disease provide an indication of the scale of the air pollution problem. Estimates of the benefits associated with reductions in ambient $PM_{2.5}$ and ozone support the design of air pollution control strategies. One such strategy currently under review is the Australian National Environment Protection Measure (NEPM) for outdoor air pollution. As part of this review, the government is considering a compliance standard for $PM_{2.5}$ and a framework to encourage reductions in population exposure, even where $PM_{2.5}$ levels are below the standard. Specific control strategies that could reduce air pollution in Sydney include increased regulation of non-road diesel engines and wood-burning heaters (NEPC, 2013a; NSW Department of Environment, Climate Change and Water, 2010).

2. Methods

We followed the well-established "damage function" approach to relate anthropogenic air pollution in Sydney to hospital admissions and premature deaths in 2007. 2007 was chosen because this was the most recent year for which mortality data were available. This approach has been used to estimate the burden of disease associated with air pollution in the US and Europe (Berman et al., 2012; Davidson et al., 2007; Hubbell et al., 2005; Künzli et al., 2000) and employs a health impact function (HIF), defined as:

$$\Delta y = (1 - e^{\beta \Delta x}) \cdot y_0 \cdot Pop$$

where Δy is the change in the number of cases of the health outcome of interest, Δx is the change in air pollution exposure, β is a risk coefficient of the health outcome of interest drawn from an epidemiological study, y_0 is the baseline incidence rate of the health outcome and *Pop* is the size of the exposed population.

We used the open source Environmental Benefits Mapping and Analysis Programme – Community Edition v1.06 (BenMAP-CE)[28] to quantify the numbers of premature deaths and hospital admissions attributable to $PM_{2.5}$ and ozone. BenMAP-CE is a tool developed for the US Environment Protection Authority (EPA) that systematises air pollution health impact assessment, incorporating user defined inputs. YLL estimates were calculated outside BenMAP-CE using the procedures described in the relevant sections below.

To minimise the risk of double-counting health effects related to both $PM_{2.5}$ and ozone exposure, or to both long- and short-term exposure to $PM_{2.5}$, we limited our analysis to the following exposure-outcome pairs: (1) long-term exposure to $PM_{2.5}$ and all-cause mortality; (2) short-term exposure to $PM_{2.5}$ and admission to hospital with cardiovascular and respiratory disease; (3) short-term exposure to ozone and non-trauma mortality; and (4) short-term exposure to ozone and respiratory hospital admission. Each selected pair is supported by strong evidence of a causal association (US EPA, 2009, WHO Regional Office for Europe, 2013). Analysis of YLL was limited to long-term exposure to $PM_{2.5}$ and all-cause mortality because the β-coefficient for this pair is derived from a survival model.

2.1. Inputs

2.1.1. Exposure to $PM_{2.5}$ and ozone

We estimated population exposure to $PM_{2.5}$ and ozone in Sydney from measurements taken at NEPM performance monitoring stations. The number of NEPM monitoring stations and their location is chosen to ensure they contribute to a representative measure of the air quality experienced by the general population of Sydney. We began with daily observations of 24 h average $PM_{2.5}$ and maximum one hour average ozone for the year 2007, provided by the NSW Office of Environment and Heritage. $PM_{2.5}$ was measured using a tapered element oscillating microbalance (TEOM) at four monitoring sites in the Sydney Metropolitan Area (Fig. 1). One monitoring site (Richmond) had only recorded 42% of daily $PM_{2.5}$ observations in 2007, and was excluded from further analyses of $PM_{2.5}$ to avoid biasing our estimates of population exposure. Ozone was measured at eleven monitoring sites in the Sydney Metropolitan Area in 2007 (Fig. 1). One site (Lindfield) had just 55% of daily ozone observations recorded, and was excluded from further analyses of ozone. Summary statistics of the monitoring stations used in the assessment are provided in Supplemental Tables 1 and 2.

Population exposure was assigned at the Local Government Area (LGA) level by interpolating the annual averages of measured daily $PM_{2.5}$ and 1 h-maximum ozone concentrations to Sydney

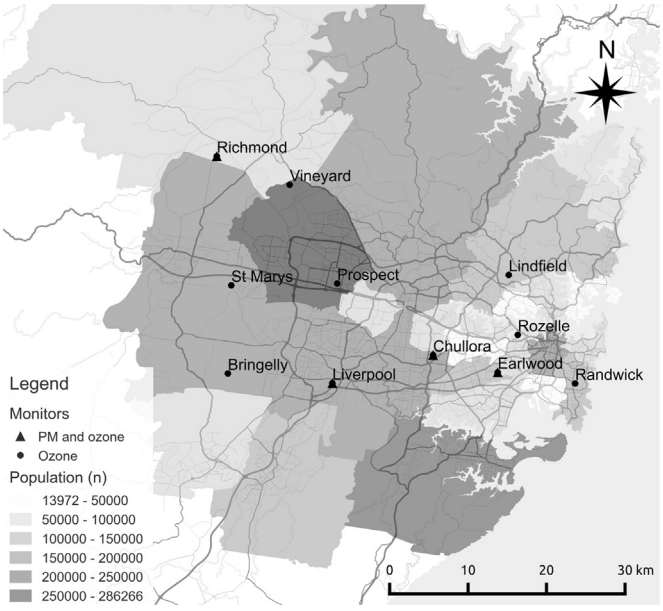

Fig. 1. Sydney LGAs, their population size and the location air pollution monitors.

Metropolitan LGAs (2008 boundaries) using Voronoi Neighbourhood Averaging (VNA) ($n=39$ LGAs). The VNA algorithm calculates an inverse-distance-weighted air quality value (Chen et al., 2004). LGAs included in the Sydney Metropolitan Area air shed were those defined by the NSW *Protection of the Environment Operations (Clean Air) Regulation 2010*.

For both pollutants, an air pollution surface interpolated from 2007 monitor levels was used to represent recent exposure.

There are currently no modelled estimates of the contribution of non-anthropogenic sources to ambient $PM_{2.5}$ and ozone in Australia. In the absence of such estimates, a previous Australian risk assessment has assumed the fifth percentile of daily average $PM_{2.5}$ and ozone levels represented background concentrations (National Environment Protection Council, 2013b) – 2.1 μg/m^3 and 8.6 ppb respectively. For this assessment, we assumed that, at each monitoring site, 40% of measured $PM_{2.5}$ and 30% of ozone was from non-anthropogenic sources. After interpolation to LGAs, these levels equated to population-weighted average concentrations of 2.4 μg/m^3 and 9.6 ppb respectively. Thus, while still somewhat arbitrary, our approach uses background concentrations similar to those of the earlier risk assessment.

For both pollutants, we also "rolled back" 2007 levels in 10% increments to the assumed background concentration. We used these incremental reductions to estimate the potential benefits that might accrue from strategies that reduce exposure.

2.1.2. Risk coefficients

Risk coefficients (β) for the selected pollutant-outcome pairs were identified from previously published epidemiological studies. For our primary analyses, we selected coefficients from large, well-conducted U.S. multicity studies that have previously been employed in comparable health impact assessments (Table 1).

The coefficients for the effect of ozone exposure are derived from studies that used maximum 8-h averages. A conversion factor of 1.33 (Thurston and Ito, 2001) was used to convert these to coefficients for 1-h averages.

2.1.3. Baseline incidence rates and LGA population size

The Australian Bureau of Statistics (ABS) provided data on population size and cause-specific deaths in 2007. The NSW Ministry of Health provided data from the NSW Admitted Patient Data Collection (NSW APDC) on hospital visits. ICD-10-Australian Modification (ICD10-AM) codes were used to identify all-cause, non-trauma (ICD10: A-R, Z35.5, Z35.8), cardiopulmonary (ICD10-AM: J00-J99, I10–I15, I20–I25, I26–I28, I30–I52, I60–I69), cardiovascular (ICD10-AM: I00-I99 [excluding I67.3, I68.0, I88, I97.8, I97.9, I98.0], G45 [excluding G45.3], G46, M30, M31, R58) and respiratory deaths (ICD10-AM: J00-J99 [excluding J95.4 to J95.9], R09.1, R09.8), and hospitalisations with a primary diagnosis of cardiovascular (ICD10-AM: I00-I99 [excluding I67.3, I68.0, I88, I97.8, I97.9, I98.0], G45 [excluding G45.3], G46, M30, M31, R58) or respiratory disease (ICD10-AM: J00-J99 [excluding J95.4 to J95.9], R09.1, R09.8). Using these data, age-specific mortality and hospital admission rates in 2007 were calculated for each LGA, providing spatially resolved baseline health data appropriate for an urban-level analysis.

2.2. Estimating the burden of disease attributable to exposure to anthropogenic $PM_{2.5}$ and ozone

2.2.1. Estimating the incidence of premature deaths and episodes of illness

We used the HIF defined above to calculate the number of premature deaths and hospital admissions attributable to anthropogenic $PM_{2.5}$ and ozone in 2007 in each LGA. These numbers were aggregated to produce estimates for the whole of Sydney. The effects of exposure to $PM_{2.5}$ were calculated using annual average levels. The effects of short-term exposure to ozone were calculated using the seasonal average of maximum daily one hour average levels.

2.2.2. Estimating YLL

Our assessment of YLL is limited to all-cause mortality from long-term exposure to $PM_{2.5}$. We assumed that each $PM_{2.5}$-attributable death is responsible for a number of YLL equal to life expectancy at age of death. Age-specific life expectancies of a population with no exposure to anthropogenic $PM_{2.5}$ were calculated by adjusting mortality rates from a standard NSW life table by the relative risk associated with a reduction in $PM_{2.5}$ exposure from 2007 levels to background.

2.3. Estimating the benefit of a reduction in exposure to $PM_{2.5}$ and ozone

2.3.1. Morbidity

To estimate the number of cardiovascular and respiratory hospital admissions that might be avoided by reductions in air pollution, we first estimated the number that would be avoided in 2007. We then assumed this number remains constant in all future years.

Table 1
Effect coefficients applied in this analysis.

Endpoint	Pollutant	Study	Population	Risk estimate (95th percentile)
Premature death				
Cohort study, all-cause	$PM_{2.5}$ (annual avg)	Krewski et al. (2009)	> 29 years	RR = 1.06 (1.04–1.06) per 10 μg/m^3
Daily time series	O3 (Daily 8 h Max)	Levy et al. (2005b)	All ages	β = 0.001119 (0.000179)
Respiratory hospital admissions				
	$PM_{2.5}$ (24-h avg)	Zanobetti and Schwartz (2006)	> 64 years	β = 0.00207 (0.00446)
	O3 (Daily 8 h Max)	Schwartz (1995)	> 64 years	RR = 1.20 (1.06–1.37) per 50 μg/m^3
		Burnett (2001)	< 2 years	Increase = 33.0 (t-statistic 3.44) per 45.2 ppb
Cardiovascular hospital admissions		*Pooled using equal weights:*		
	$PM_{2.5}$ (24-h avg)	Zanobetti and Schwartz (2009)	> 64 years	β = 0.00189 (0.000283)
		Peng et al. (2009)		β = 0.00068 (0.000214)
		Peng et al. (2008)		β = 0.00071 (0.00013)
		Bell et al. (2008)		β = 0.0008 (0.000107)
		Moolgavkar (2000)	20–64 years	RR = 1.04 (t statistic: 4.1) per 10 μg/m^3

2.3.2. Mortality

The primary benefit of a reduction in a population's exposure to $PM_{2.5}$ is an increase in the average length of survival, which results in an increase in the size of the population. We quantify this benefit using a method developed by Miller and Hurley (Miller, 2003). This involves modelling the future mortality experience of the cohort of people alive in 2007 under two scenarios – one where $PM_{2.5}$ exposure remains unchanged, the other where exposure is reduced. At the same time, it is assumed that the annual number of births and the baseline mortality rate remains constant and that there is no migration. The number of additional life-years in the less exposed population in a specific year is the difference in the size of the two populations in that year. We modelled the effect over 105 years – the time to extinction of the large majority of people alive in 2007.

For our main analysis, we assume that an intervention to reduce $PM_{2.5}$ causes an immediate reduction in concentration and that this has and immediate effect on health. However, it is likely that there is, in fact, a "cessation lag" between the initial reduction in exposure and the realisation of the full health benefit of that reduced exposure. While there is little evidence to support the use of any particular cessation lag for $PM_{2.5}$ impact assessment, we conducted sensitivity analyses to examine the effect of various linear lags from five to 20 years (Schwartz et al., 2008; Walton, 2010).

3. Results

The three monitoring sites used for analysis of $PM_{2.5}$ each had over 90% of daily observations recorded in 2007, with annual average $PM_{2.5}$ levels for each monitor ranging from 5.9 μg/m^3 (Earlwood) to 7.1 μg/m^3 (Liverpool). The LGA interpolated $PM_{2.5}$ levels ranged from 5.4 μg/m^3 in Hawkesbury LGA to 6.5 μg/m^3 in Liverpool LGA (Fig. 2). The average $PM_{2.5}$ concentration across the Sydney metropolitan area was approximately 6 μg/m^3 on a population-weighted basis.

We estimate that exposure to 2007 levels of anthropogenic $PM_{2.5}$ had a mortality effect equivalent to 430 (95% CI: 310–540) premature deaths, with a total loss of life of approximately 5,800 years (95% CI: 3900–7600) (Table 2). This represents approximately 2.1% of all deaths and 1.8% of all YLL in Sydney in 2007.

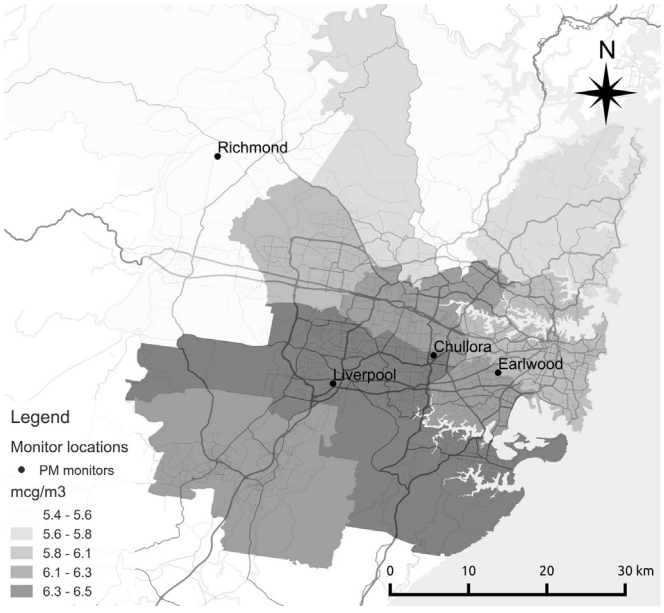

Fig. 2. Interpolated baseline annual average $PM_{2.5}$ concentrations (μg/m^3) in 2007 by LGA.

Table 2
The burden of disease in 2007 attributable to recent levels of anthropogenic $PM_{2.5}$ and ozone.

Outcome	Age	Number	95% CI	Attributable fraction (%)
$PM_{2.5}$-related impacts				
Premature deaths	30+	430	310–540	2.1
YLL	30+	5800	3900–7600	1.8
Cardiovascular hospital admissions	18+	270	180–370	0.3
Respiratory hospital admissions	65+	150	94–200	0.3
Ozone-related impacts				
Premature deaths	0+	160	120–210	0.8
Respiratory hospital admissions	0–2 and >64	760	130–1700	1.5

We also estimated that 2007 levels of anthropogenic $PM_{2.5}$ in Sydney caused 270 (95% CI: 180–370) cardiovascular hospital admissions among populations 18 years and older and 150 (95% CI: 94–200) respiratory hospital admissions among populations 65 and older (Table 2).

Assuming no cessation lag, reducing 2007 $PM_{2.5}$ levels by 10% (0.6 μg/m^3) would yield 71 (95% CI: 52–91) fewer premature deaths in people 30 years and older in the first year after the reduction (Table 3). If the 10% reduction was sustained, we estimate there would be about 640 (95% CI: 430–850) fewer premature deaths and 3500 additional life-years (95% CI: 2300–4600) during the first 10 years (Fig. 3 and Supplemental Table 3) of the intervention. The mortality effect of the six $PM_{2.5}$ rollback scenarios over 100 years is illustrated in Fig. 3. The results are sensitive to the assumed cessation lag (Supplemental Table 4). For example, if it were to take five years to achieve the full effect of reduced exposure, a 10% reduction in $PM_{2.5}$ would, over 10 years, result in an estimated mortality benefit equivalent to 420 fewer premature deaths and 1700 additional life years. The effect of various cessation lags is illustrated in Supplemental Fig. 1.

We also estimate approximately 46 (95% CI: 30–62) cardiovascular hospital admissions in people aged 18 year and over, and 25 (95% CI: 16–33) respiratory hospital admissions in people aged 65 years and over would be avoided each year with a 10% reduction in short term $PM_{2.5}$ exposure (Table 3).

The ten monitoring sites used for analysis of ozone all had over 90% of daily observations recorded in 2007, with the exception of Prospect, which had 81%. The highest daily 1 h maximum ozone

Table 3
Estimated number of deaths and hospital admissions in Sydney in that would have been avoided if $PM_{2.5}$ and ozone levels had been 10% lower.

Outcome	Age	Number	95% CI	Attributable fraction (%)
$PM_{2.5}$-related impacts				
Premature deaths	30+	71	52–91	0.4
Cardiovascular hospital admissions	18+	46	30–62	0.06
Respiratory hospital admissions	65+	25	16–33	0.04
Ozone-related impacts				
Premature deaths	0+	24	17–30	0.27
Respiratory hospital admissions	0+	110	17–230	0.19

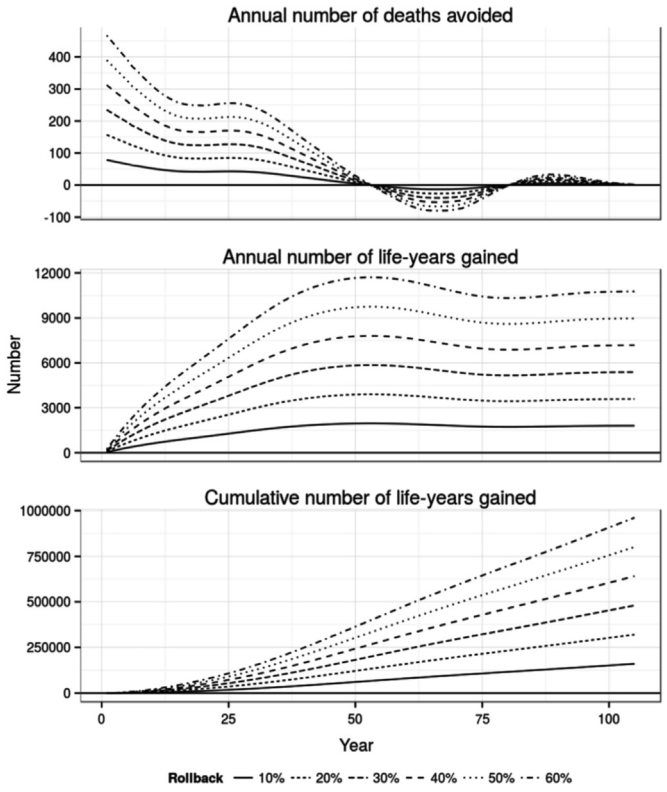

Fig. 3. The effect of each $PM_{2.5}$ rollback scenario on the avoided premature deaths and additional life-years. The graphs show the annual number of deaths avoided and life-years gained in a less exposed population compared to baseline.

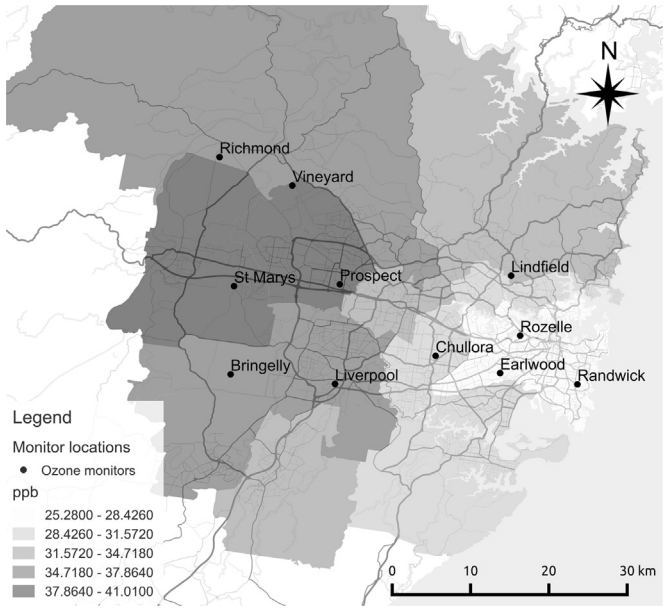

Fig. 4. Interpolated baseline seasonal average of maximum daily one hour average ozone concentrations (ppb) in 2007 by LGA.

recorded was 134 ppb at Richmond (Fig. 4).

We estimated total anthropogenic ozone caused 160 deaths (95% CI: 120–210) and 760 (95% CI: 130–1,700) respiratory hospital admissions in 2007 (Table 2). Equivalent analyses using the Australian HIF produced an estimate of 240 (95% CI: 91–390) premature deaths.

If daily ozone levels had been 10% lower (3 ppb) than those observed in 2007, it is estimated that 24 (95% CI: 17–30) premature deaths from non-trauma related causes and 110 respiratory hospital admissions (95% CI: 17–230) could have been avoided (Table 3).

Sensitivity analyses showed that the health benefits estimates for a change in $PM_{2.5}$ and ozone for the Sydney Metropolitan Area are broadly scalable to different changes in pollutant levels (Supplemental Table 5).

4. Discussion

Assessments such as this inform the public and policy makers of the health risks associated with air pollution and the relative benefits of emission control options. While Sydney enjoys $PM_{2.5}$ and ozone levels that are comparatively low among industrialised countries, this analysis suggests that these pollutants are responsible for an important disease burden, and that relatively modest air quality improvements would yield substantial health benefits. It also suggests that, even though the ozone standard is exceeded more frequently than the $PM_{2.5}$ standard, $PM_{2.5}$ has a larger adverse impact.

We found that, in 2007, Sydney residents' exposure to anthropogenic $PM_{2.5}$ had a mortality effect equivalent to 430 premature deaths and 5800 YLL and was responsible for approximately 320 hospital admissions with cardiovascular and respiratory illness. In the same year, exposure to anthropogenic ozone had a mortality effect equivalent to 160 premature deaths and was responsible for approximately 760 respiratory hospital admissions.

If we assume there is no cessation lag and that the full health benefits of reduced $PM_{2.5}$ are realized immediately, reducing 2007 annual mean $PM_{2.5}$ levels in Sydney by a small amount (10%) would, over 10 years, prevent approximately 650 premature deaths with a gain of about 3500 life-years in that time period. Over 20 years, an additional 12,000 life-years would be lived. Since 2007, $PM_{2.5}$ and ozone concentrations have remained relatively unchanged while the population of Sydney has grown by approximately 12%. Therefore, it can be reasonably be expected that the burden of disease associated with these pollutants will have increased.

Translation of the current mortality burden into monetary terms may be useful for decision makers. Using a value of statistical life of $6 million (2006 AUD) (ACSC, 2007) indexed to 2007, the cost of premature deaths attributable to air pollution in 2007 is approximately $2.6 billion. While we lacked the appropriate cost of illness measures to assess the economic value of the estimated 270 cardiovascular and 150 respiratory hospital admissions, prior analyses (EPA, 2011) suggest that the value of avoided premature deaths account for over 95% of the total value associated with mortality and morbidity endpoints.

Our assessment of the benefits of reduced $PM_{2.5}$ concentrations takes into account the demographic changes that such a reduction would bring about. As shown in Fig. 3, there is a fall in the initial number of avoided premature over time. This occurs because, as $PM_{2.5}$-related risk is reduced, more people survive to future years, increasing the size and average age of the population. All other things being equal, more deaths occur in a larger and older population, which offsets the reduction in $PM_{2.5}$-related risk. After about 50 years, the larger population size and older average age more than offset the reduction in $PM_{2.5}$-related risk and there are more deaths in the less exposed population. Despite this, the population continues to experience more life-years by virtue of its larger size.

Correlation between ambient $PM_{2.5}$ and ozone concentrations means that the effects of each pollutant observed in epidemiological studies of the two pollutants may not be entirely

independent. Because of this, there may be some overlap between our estimated $PM_{2.5}$- and ozone-related premature deaths and respiratory hospital admissions. To avoid double-counting, the estimates from the two pollutants should not be added together.

Our results suggest that approximately 2.1% of deaths and 1.8% of YLL are attributable to $PM_{2.5}$ in Sydney. A recent US study attributed 4–7% of premature deaths to $PM_{2.5}$ levels above non-anthropogenic background levels (US background range: 0.62–1.72 $\mu g/m^3$), equivalent to an approximate 80–90% reduction in $PM_{2.5}$ (Fann et al., 2011). Thus, our Sydney estimate of avoided premature deaths is comparable to the US estimate.

Estimates of the health benefits associated with a change in air pollution are dependent on the risk coefficients chosen. We chose to use a risk coefficient for long-term exposure to $PM_{2.5}$ derived from the very large American Cancer Society cohort because this coefficient has been commonly used in important risk assessments (US EPA, 2010; COMEAP, 2010) and because it incorporated a large number of cities with differing sources of $PM_{2.5}$. In support of this, a recent meta-analysis of 13 cohort studies of long-term exposure to $PM_{2.5}$ produced a very similar effect estimate (Hoek et al., 2013). However, it should be noted that studies with better-quality exposure assessment have tended to produce larger effect estimates and a process of expert elicitation produced a coefficient of 10% per 10 $\mu g/m^3$ (Roman et al., 2008). Use of a larger coefficient would produce higher estimates of both burden and the potential benefits of $PM_{2.5}$ reductions.. It should also be noted that there is some uncertainty extrapolating a risk estimate from the US to Australia, where the mix of $PM_{2.5}$ sources is likely to be different. Coefficients for the effects of ozone have varied more widely. Our sensitivity analysis using a risk coefficient from an Australian multi-city study produced an estimate of non-trauma mortality approximately 50% higher than the estimates produced using coefficients from time-series studies conducted in the US and Europe (Bell et al., 2004). This highlights the importance of selecting an appropriate coefficient and assessing the sensitivity of results to different coefficients.

A strength of our analysis is the use of LGA-based population and baseline health data to account for the spatial distribution of air pollution and population within Sydney. However, when interpreting estimates of the potential health benefits associated with a change in air pollution, the uncertainty in such estimates should be considered. Our estimates have accounted for statistical imprecision around the risk coefficients selected from epidemiological studies, as inferred from their confidence intervals, and our sensitivity analyses address uncertainty around coefficient selection and the length of any cessation lag. Other sources of uncertainty not accounted for include the uncertainty around incidence and population estimates, and estimated $PM_{2.5}$ and ozone exposures derived from fixed site monitors. In addition, $PM_{2.5}$ exposures estimates for the Sydney Metropolitan Area are at the lower end of the exposure input range for the Krewski et al. (2009) risk coefficient for long-term exposure to $PM_{2.5}$. However, the epidemiological literature offers little evidence of a threshold in the concentration–response functions (World Health Organisation Regional Office for Europe, 2013). While recent studies have found that the concentration–response curve flattens at higher concentrations, a recent cohort study in Canada found associations between $PM_{2.5}$ and mortality at concentrations comparable to those in Sydney (Burnett et al., 2014; Crouse et al., 2012).

5. Conclusion

We have shown the magnitude of effect of a reduction in air pollutants in the Sydney Metropolitan Area, represented by the estimated number of premature deaths avoided due to long-term exposure to $PM_{2.5}$, the additional life-years that would be gained and the estimated number of hospitalisations avoided due to reduced short-term exposure to $PM_{2.5}$. For comparison purposes we have also provided estimates of the effect on mortality of a reduction in ozone of similar proportions. This work shows that current levels of air pollution in Sydney make a substantial contribution to the burden of disease, and reducing air pollution would provide important public health benefits.

Acknowledgements

Suzanne Quigley, NSW Office of Environment and Heritage, who assisted with provision of air quality data.

Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.envres.2015.09.007.

References

Australian Compensation and Safety Council, 2007. The Health of Nations: The Value of a Statistical Life. Commonwealth of Australia, Canberra.

Bell, M.L., Ebisu, K., Peng, R.D., Walker, J., Samet, J.M., Zeger, S.L., et al., 2008. Seasonal and regional short-term effects of fine particles on hospital admissions in 202 US counties, 1999–2005. Am. J. Epidemiol. 168. http://dx.doi.org/10.1093/aje/kwn252 1301–10.

Bell, M.L., McDermott, A., Zeger, S.L., Samet, J.M., Dominici, F., 2004. Ozone and short-term mortality in 95 US urban communities, 1987–2000. JAMA J. Am. Med. Assoc. 292, 2372–2378. http://dx.doi.org/10.1001/jama.292.19.2372.

Berman, J.D., Fann, N., Hollingsworth, J.W., Pinkerton, K.E., Rom, W.N., Szema, A.M., et al., 2012. Health benefits from large-scale ozone reduction in the United States. Environ. Health Perspect. 120. http://dx.doi.org/10.1289/ehp.1104851 1404–10.

Burnett, R.T., 2001. Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. Am. J. Epidemiol. 153, 444–452. http://dx.doi.org/10.1093/aje/153.5.444.

Burnett, R.T., Pope, C.A., Ezzati, M., Olives, C., Lim, S.S., Mehta, S., 2014. An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure. Environ. Health Perspect.

Cesaroni, G., Badaloni, C., Gariazzo, C., Stafoggia, M., Sozzi, R., Davoli, M., et al., 2013. Long-term exposure to urban air pollution and mortality in a cohort of more than a million adults in Rome. Environ. Health Perspect. 121. http://dx.doi.org/10.1289/ehp.1205862 324–31.

Chen, J., Zhao, R., Li, Z., 2004. Voronoi-based k-order neighbour relations for spatial analysis. ISPRS J. Photogramm. Remote Sens. 59, 60–72. http://dx.doi.org/10.1016/j.isprsjprs.2004.04.001.

Committee on the Medical Effects of Air Pollutants, 2010. The mortality effect of long-term exposure to particulate matter in the United Kingdom.

Correia, A.W., Pope, C.A., Dockery, D.W., Wang, Y., Ezzati, M., Dominici, F., 2013. Effect of air pollution control on life expectancy in the United States: an analysis of 545 U.S. counties for the period from 2000 to 2007. Epidemiology 24, 23–31. http://dx.doi.org/10.1097/EDE.0b013e3182770237.

Crouse, D.L., Peters, P.A., van Donkelaar, A., Goldberg, M.S., Villeneuve, P.J., Brion, O., et al., 2012. Risk of nonaccidental and cardiovascular mortality in relation to long-term exposure to low concentrations of fine particulate matter: a Canadian national-level cohort study. Environ. Health Perspect. 120. http://dx.doi.org/10.1289/ehp.1104049 708–14.

Davidson, K., Hallberg, A., McCubbin, D., Hubbell, B., 2007. Analysis of PM2.5 using the environmental benefits mapping and analysis program (BenMAP). J. Toxicol. Environ. Health A 70. http://dx.doi.org/10.1080/15287390600884982 332–46.

European Environment Agency, 2014 “Air Quality in Europe-2014 Report” EEA Copenhagen. ⟨http://www.eea.europa.eu/publications/air-quality-in-europe-2014⟩ (accessed 11.08.15).

Fann, N., Lamson, A.D., Anenberg, S.C., Wesson, K., Risley, D., Hubbell, B.J., 2011. Estimating the national public health burden associated with exposure to ambient PM(2.5) and ozone. Risk Anal. 32, 81–95. http://dx.doi.org/10.1111/j.1539-6924.2011.01630.x.

Hoek, G., Krishnan, R.M., Beelen, R., Peters, A., Ostro, B., Brunekreef, B., Kaufman, J. D., 2013. Long-term air pollution exposure and cardio- respiratory mortality: a review. Environ. Health 12 (1), 43. http://dx.doi.org/10.1186/1476-069X-12-43.

Hubbell, B.J., Hallberg, A., McCubbin, D.R., Post, E., 2005. Health-related benefits of attaining the 8-h ozone standard. Environ. Health Perspect. 113, 73–82.

Ito, K., De Leon, S.F., Lippmann, M., 2005. Associations between ozone and daily mortality: analysis and meta-analysis. Epidemiology 16, 446–457.
Jerrett, M., Burnett, R.T., Pope, C.A., Ito, K., Thurston, G., Krewski, D., et al., 2009. Long-term ozone exposure and mortality. N. Engl. J. Med. 360. http://dx.doi.org/10.1056/NEJMoa0803894 1085–95.
Katsouyanni, K., Samet, J.M., Anderson, H.R., Atkinson, R., Le Tertre, A., Medina, S., et al., 2009. Air pollution and health: a European and North American approach (APHENA). Res. Rep. Health Eff. Inst., 5–90.
Krewski, D., Jerrett, M., Burnett, R.T., Ma, R., Hughes, E., Shi, Y., et al., 2009. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Res. Rep. Health Eff. Inst., 5–114 (discussion 115–136).
Künzli, N., Kaiser, R., Medina, S., Studnicka, M., Chanel, O., Filliger, P., et al., 2000. Public-health impact of outdoor and traffic-related air pollution: a European assessment. Lancet 356, 795–801. http://dx.doi.org/10.1016/S0140-6736(00)02653-2.
Levy, J.I., Chemerynski, S.M., Sarnat, J. a, 2005a. Ozone exposure and mortality. Epidemiology 16, 458–468. http://dx.doi.org/10.1097/01.ede.0000165820.08301.b3.
Levy, J.I., Chemerynski, S.M., Sarnat, J.A., 2005b. Ozone exposure and mortality: an empiric bayes metaregression analysis. Epidemiology 16, 458–468.
Lim, S.S., Vos, T., Flaxman, A.D., Danaei, G., Shibuya, K., Adair-Rohani, H., et al., 2012. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the global burden of disease study 2010. Lancet 380. http://dx.doi.org/10.1016/S0140-6736(12)61766-8 2224–60.
Miller, B.G., 2003. Life table methods for quantitative impact assessments in chronic mortality. J. Epidemiol. Community Health 57, 200–206. http://dx.doi.org/10.1136/jech.57.3.200.
Moolgavkar, S.H., 2000. Air pollution and hospital admissions for diseases of the circulatory system in three U.S. metropolitan areas. J. Air Waste Manag. Assoc. 50, 1199–1206. http://dx.doi.org/10.1080/10473289.2000.10464162.
National Environment Protection Council, 2013a. Consultation Regulation Impact Statement for Reducing Emissions from Wood Heaters. NEPC. Canberra.
National Environment Protection Council, 2013b. Exposure Assesment and Risk Characterisation to Inform Recommendations for Updating Ambient Air Quality Standards for $PM_{2.5}$, PM_{10}, NO_2, SO_2. NEPC. Canberra.
NSW Environment Protection Authority, 2010. Air Emissions Inventory for the Greater Metropolitan Region in New South Wales. 2008 Calendar Year. Consolidated Natural and Human-Made Emissions: Results. vol. 1. NSW Environment Protection Authority, Sydney.
Peng, R.D., Bell, M.L., Geyh, A.S., McDermott, A., Zeger, S.L., Samet, J.M., et al., 2009. Emergency admissions for cardiovascular and respiratory diseases and the chemical composition of fine particle air pollution. Environ. Health Perspect. 117. http://dx.doi.org/10.1289/ehp.0800185 957–63.
Peng, R.D., Chang, H.H., Bell, M.L., McDermott, A., Zeger, S.L., Samet, J.M., et al., 2008. Coarse particulate matter air pollution and hospital admissions for cardiovascular and respiratory diseases among Medicare patients. J. Am. Med. Assoc. 299. http://dx.doi.org/10.1001/jama.299.18.2172 2172–9.
Pope, C.A., Burnett, R.T., Thurston, G.D., Thun, M.J., Calle, E.E., Krewski, D., et al., 2004. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. Circulation 109. http://dx.doi.org/10.1161/01.CIR.0000108927.80044.7F 71–7.
Pope, C.A., Ezzati, M., Dockery, D.W., 2009. Fine-particulate air pollution and life expectancy in the United States. N. Engl. J. Med. 360. http://dx.doi.org/10.1056/NEJMsa0805646 376–86.
Pope III, C. a, 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA J. Am. Med. Assoc. 287, 1132–1141. http://dx.doi.org/10.1001/jama.287.9.1132.
Roman, H.A., Walker, K.D., Wash, T.L., et al., 2008. Expert judgment assessment of the mortality impact of changes in ambient fine particulate matter in the U.S. Environ. Sci. Technol. 42 (7), 2268–2274 http://doi.org/10.1021/es0713882.
Schwartz, J., Coull, B., Laden, F., Ryan, L., 2008. The effect of dose and timing of dose on the association between airborne particles and survival. Environ. Health Perspect. 116. http://dx.doi.org/10.1289/ehp.9955 64–9.
Simpson, R., Williams, G., Petroeschevsky, A., Best, T., Morgan, G., Denison, L., et al., 2005. The short-term effects of air pollution on daily mortality in four Australian cities. Aust. N. Z. J. Public Health 29, 205–212. http://dx.doi.org/10.1111/j.1467-842X.2005.tb00758.x.
Thurston, G.D., Ito, K., 2001. Epidemiological studies of acute ozone exposures and mortality. J. Expo. Anal. Environ. Epidemiol. 11 (4). http://dx.doi.org/10.1038/sj.jea.7500169 286–94.
U.S. EPA, 2009. Integrated Science Assessment for Particulate Matter (Final Report).
US EPA National Center for Environmental Assessment RTPNEMAG, Brown J. Integrated Science Assessment of Ozone and Related Photochemical Oxidants (Final Report).
U.S. EPA, 2010. Our Nation's Air Status and Trends Through 2008. EPA-454/R. doi: EPA-454/R-09-002.
US EPA, 20111. The benefits and costs of the Clean Air Act from 1990 to 2020 (final report).
Walton, Heather A., 2010. Working Paper: Development of Proposals for Cessation Lag(s) for Use in Total Impact Calculations. Supporting Paper to COMEAP 2010 Report: The Mortality Effects of Long Term Exposure to Particulate Air Pollution in the United Kingdom. Health Protection Agency, Didcot.
World Health Organization Regional Office for Europe, 2013. Review of Evidence on Health Aspects of Air Pollution-REVIHAAP. WHO, Copenhagen.
Zanobetti, A., Schwartz, J., 2006. Air pollution and emergency admissions in Boston, MA. J. Epidemiol. Community Health 60. http://dx.doi.org/10.1136/jech.2005.039834 890–5.
Zanobetti, A., Schwartz, J., 2009. The effect of fine and coarse particulate air pollution on mortality: a national analysis. Environ. Health Perspect. 117, 898–903. http://dx.doi.org/10.1289/ehp.0800108.