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The health benefits of reducing air pollution in Sydney, Australia

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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/m³ 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.

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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 (O₃), 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/m³ 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 and 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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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.

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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

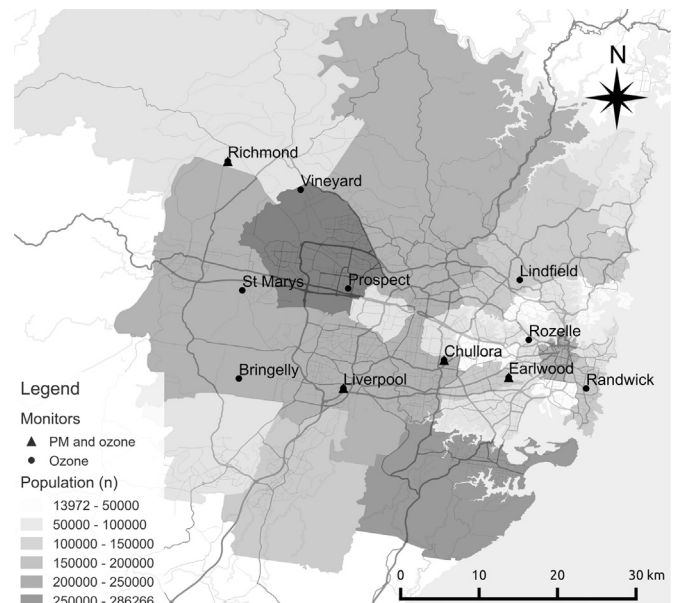


Fig. 1. Sydney LGAs, their population size and the location air pollution monitors.

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