Environmental Research ▮ (▮▮▮▮) ▮▮▮–▮▮▮

Contents lists available at ScienceDirect

Environmental Research

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

Extreme air pollution events from bushfires and dust storms and their association with mortality in Sydney, Australia 1994–2007 ☆

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ARTICLE INFO

Article history:
Received 7 December 2010
Received in revised form
21 April 2011
Accepted 6 May 2011

Keywords:
Air pollution
Particulate matter
Bushfire smoke
Dust
Mortality

ABSTRACT

Introduction: Extreme air pollution events due to bushfire smoke and dust storms are expected to increase as a consequence of climate change, yet little has been published about their population health impacts. We examined the association between air pollution events and mortality in Sydney from 1997 to 2004.

Methods: Events were defined as days for which the 24 h city-wide concentration of PM_{10} exceeded the 99th percentile. All events were researched and categorised as being caused by either smoke or dust. We used a time-stratified case-crossover design with conditional logistic regression modelling adjusted for influenza epidemics, same day and lagged temperature and humidity. Reported odds ratios (OR) and 95% confidence intervals are for mortality on event days compared with non-event days. The contribution of elevated average temperatures to mortality during smoke events was explored.

Results: There were 52 event days, 48 attributable to bushfire smoke, six to dust and two affected by both. Smoke events were associated with a 5% increase in non-accidental mortality at a lag of 1 day OR (95% confidence interval (CI)) 1.05 (95%CI: 1.00–1.10). When same day temperature was removed from the model, additional same day associations were observed with non-accidental mortality OR 1.05 (95%CI: 1.00–1.09), and with cardiovascular mortality OR (95%CI) 1.10 (95%CI: 1.00–1.20). Dust events were associated with a 15% increase in non-accidental mortality at a lag of 3 days, OR (95%CI) 1.16 (95%CI: 1.03–1.30).

Conclusions: The magnitude and temporal patterns of association with mortality were different for smoke and dust events. Public health advisories during bushfire smoke pollution episodes should include advice about hot weather in addition to air pollution.

1. Introduction

Australia is an arid continent with an ancient history of dust storms and bushfire activity that predates human occupation (Bowman, 2000). Both events cause episodes of severe air pollution and both are set to increase in frequency and severity as global temperatures continue to warm (Confalonieri et al., 2007). The year of 2009 included Australia's worst bushfires and one of the most severe dust events on record, highlighting our particular vulnerability to these manifestations of climate change (Confalonieri et al., 2007). Severe air pollution events from bushfires and dust are usually short-lived, but the resulting particulate matter (PM) can reach extreme concentrations, travel vast distances, and affect densely populated areas far from their source.

Urban air pollution has been causally associated with non-accidental, respiratory and cardiovascular mortality (Pope and Dockery, 2006), yet data concerning association between mortality

☆ *Funding and Ethical Approvals*: The study was supported by an Australian Research Council linkage grant (LP0882048) with contributions from the following industry partners: The Tasmanian Department of Health and Human Services, Environment Protection Authority and Fire Service; the Western Australia Department of Health and Department of Environment and Conservation and the New South Wales Department of Health. Fay Johnston is supported by a fellowship from Australia's National Health and Medical Research Council (No. 490057). The funding agencies had no role in the analysis of data or preparing the manuscript. This research was approved by the human research ethics committees of the University of Tasmania and the Australian National University.

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0013-9351/$ - see front matter © 2011 Elsevier Inc. All rights reserved.
doi:10.1016/j.envres.2011.05.007

and severe dust or wildfire smoke events are sparse. A handful of toxicological studies suggests that smoke particles elicit a spectrum of effects similar to those of urban air pollution, including inflammation, oxidative stress and pro-coagulation (Jalava et al., 2006), but epidemiological studies are limited and have reported mixed outcomes. Of five previous studies of mortality and PM from wildfire smoke, only the two conducted during the extreme South East Asian wildfires of 1997–1998 reported a positive association (Jayachandran, 2005; Sastry, 2002). Two further studies from Finland and Sydney both reported increases in mortality similar in magnitude to studies of urban PM, but neither result was statistically significant (Hanninen et al., 2009; Morgan et al., 2010). A small study from Denver, USA found no association with mortality after a 2-day smoke pollution episode, but the statistical power of this study was limited (Hänninen, 2008; Vedal and Dutton, 2006). Studies of mortality in association with extreme dust events are also sparse. Perez et al. (2008) found an association between windblown dust from the Sahara desert and daily mortality in Barcelona, Spain. However, three studies of Asian dust storms had mixed results, with two finding small increases in non-accidental mortality that were not statistically significant (Chen et al., 2004; Hashizume et al., 2010; Kwon et al., 2002).

In summary, there is still insufficient evidence to determine whether the particulate matter from vegetation fire smoke or dust storm pollution carries a greater or lesser risk of mortality compared with PM from urban sources. The episodic nature of large events makes them difficult to study. It is challenging to predict precisely when and where air quality will be affected, so studies are generally retrospective and dependent upon routinely collected data. Further, the peak exposures may be too short-lived to detect all but the most sensitive health outcomes (Hänninen, 2008). Australia provides the opportunity to explore outcomes associated with both these exposures. Australian cities are frequently affected by bushfire smoke given their proximity to highly flammable native vegetation and hot dry weather conditions that favour combustion. The large arid interior of the continent is a major source of global atmospheric dust. Drought, overgrazing and bushfires all contribute to dust storms that occasionally affect Australian cities (Mitchell et al., 2010). Here we retrospectively assess the mortality associated with extreme air pollution events due to bushfire smoke and dust in Sydney (population 3,862,000) from January 1994 to June 2007.

2. Methods

2.1. Exposure definitions

Air quality data were provided by the New South Wales Department of Environment, Climate Change and Water. We obtained thirteen and a half years of daily average concentrations of particulate matter less than 10 μm in aeronautic diameter (PM_{10}). PM_{10} was measured at 7 monitoring stations across the city for which data were available for 70% or greater of days in the time series. All stations used tapered element oscillating microbalances for measuring PM concentrations. Missing data for each site were imputed using available data from other proximate monitoring sites in the network following the protocol of the Air Pollution and Health a European Approach studies in which the missing value is replaced with the weighted average of the values of the rest of the monitoring stations (Atkinson et al., 2001). We then calculated the daily city-wide average PM_{10} concentrations for the study period. Daily measures of 1 h maximum ozone (parts per billion) were provided for 9 stations. City-wide averages were calculated using the same methods described for PM above.

Smoke and dust events were identified from a validated database that is the subject of another paper (Johnston et al., 2011). In brief, an extreme pollution event was defined as any day on which the PM_{10} concentration exceeded the 99th percentile of the time series (47.3 $\mu g/m^3$), and multiple sources of information were used to evaluate the cause of each event. We began by searching news archives for stories related to dust and smoke pollution. Next we searched for evidence of smoke and dust plumes using readily-available remote sensing data from the moderate resolution imaging spectroradiometer and the total ozone mapping spectrometer provided by the US National Aeronautical and Space Administration (NASA, 2010). Finally, Government land management agencies provided us with information about dates of prescribed burning and wildfires, and the Dustwatch project of Griffith University, Brisbane provided information from their Dust Event Database. The latter uses computer simulations to estimate the origin and geographic distribution of Australia dust events (McTainsh, 1998; Raupach et al., 1994).

We selected the 99th percentile as the cut point for defining an extreme air pollution event because it corresponded closely with Australia's 24-h air quality standard for PM_{10} (50 $\mu g/m^3$) and the majority of air quality exceedences in Australia are due to these extreme events. This allows our results to be useful for the public health authorities that are required to act when air quality standards are breached. Previous bushfire smoke research in Sydney has also used the 99th percentile to define severe pollution events (Morgan et al., 2010). As a sensitivity analysis we also conducted analyses including less severe smoke and dust pollution events in which PM_{10} exceeded the 95th percentile (32 $\mu g/m^3$) (Fig. 2).

The 99th percentile was exceeded on 56 occasions in Sydney. All events were identified as being caused by either smoke or dust, with 52 validated by two or more separate sources. When the 95th percentile, was used for the cut point to define an extreme event, only 106 of the 204 exceedences could be confidently attributed to smoke or dust with 43 validated by two or more separate sources (Fig. 2) (Johnston et al., 2011). Days not identified as having bushfire smoke or dust events were classified as background. Sources of background particulate matter in Sydney include motor vehicle and industrial emissions, domestic wood smoke and crustal particles (NSW Department of Environment Climate Change and Water, 2010). In Sydney the vast majority of bushfire smoke is derived from wildfires in the eucalypt forests to the west of the city, while dust is transported from the arid interior of the continent (Fig. 1) (Johnston et al., 2011).

Daily average ambient temperature and humidity (as measured by dew point temperature) were provided by the Bureau of Meteorology and merged with the health data using a technique that weights observations from different weather stations according to population density (Hanigan et al., 2006). Epidemics of influenza were defined as days with hospital admission rates for influenza greater than the 90th percentile of the regional distribution (Touloumi et al., 2005).

2.2. Outcome data

Our study population was defined geographically according to their statistical local area of residence. We included all statistical local areas in the Sydney metropolitan area for which representative air quality monitoring data were available. Mortality data were provided by the Australian Bureau of Statistics coded according to the World Health Organisation's International Classification of Diseases (ICD) (Australian Bureau of Statistics; WHO, 2005). Until December 1996 the underlying cause of death was coded according to the ICD 9th revision (ICD-9) and thereafter according to the 10th revision (ICD-10). We followed the protocol of the National Casemix and Classification Centre for mapping from ICD-9 to ICD-10 (National Casemix and Classification Centre, 2010). We examined non-accidental mortality, (ICD-9 codes <800; ICD-10 codes A00-R99) cardiovascular mortality (ICD-9 codes 390-459; ICD-10 codes I00-I99 excluding 67.3, I68.0, I88, I97.8-98.0, G45 excluding G45.3, G46, M30, M31 and R58) and respiratory mortality (ICD-9 codes 460–519; ICD-10 codes J00-J99 excluding J95.4 to J95.9, R09.1, R09.8).

2.3. Statistical analyses

All analyses were conducted using the R statistical software package (R Foundation for Statistical Computing, 2006). We used a time-stratified case-crossover design in which the event status, coded as a binary indicator variable for smoke and dust, on (and up to three days before) the day of death was compared with the event status on control days matched by day of week, month and calendar year. By design this approach controls for the effects of day of week, season and long term trends on mortality (Maclure, 1991). We used conditional logistic regression models adjusted for meteorology and influenza epidemics to estimate the odds ratio (OR) for mortality associated with smoke and dust events compared with the background. To determine the optimal number of degrees of freedom for modelling the meteorological variables in the logistic models, we first conducted Poisson time-series generalised additive models using the mgcv package in R to display the concentration response function for each variable (Wood, 2006). All models included the following covariates: (1) influenza epidemics, a binary variable coded as 1 if influenza hospital admission rates were <90th percentile and otherwise 0; (2) average temperature (°C) for the previous 3 days (lags 1–3) modelled as a non-linear response with 2 degrees of freedom; (3) average dew point for the previous 3 days (lags 1–3) modelled as a non-linear response with 2 degrees of freedom; (4) same-day average temperature (°C) modelled as a non-linear response with 2 degrees of freedom; and (5) same-day average dew point (°C) modelled as a linear response.

Bushfires and dust storms tend to coincide with high ambient temperatures and higher maximum ozone concentrations, both of which have been independently associated with increased risk of mortality (Anderson and Bell, 2009; Ito et al., 2005). We therefore conducted sensitivity analyses to examine the contribution of temperature and 1 h maximum daily ozone to mortality during severe pollution events. We also examined the sensitivity of our results the cut point used to define a severe pollution event (99th vs 95th percentile).

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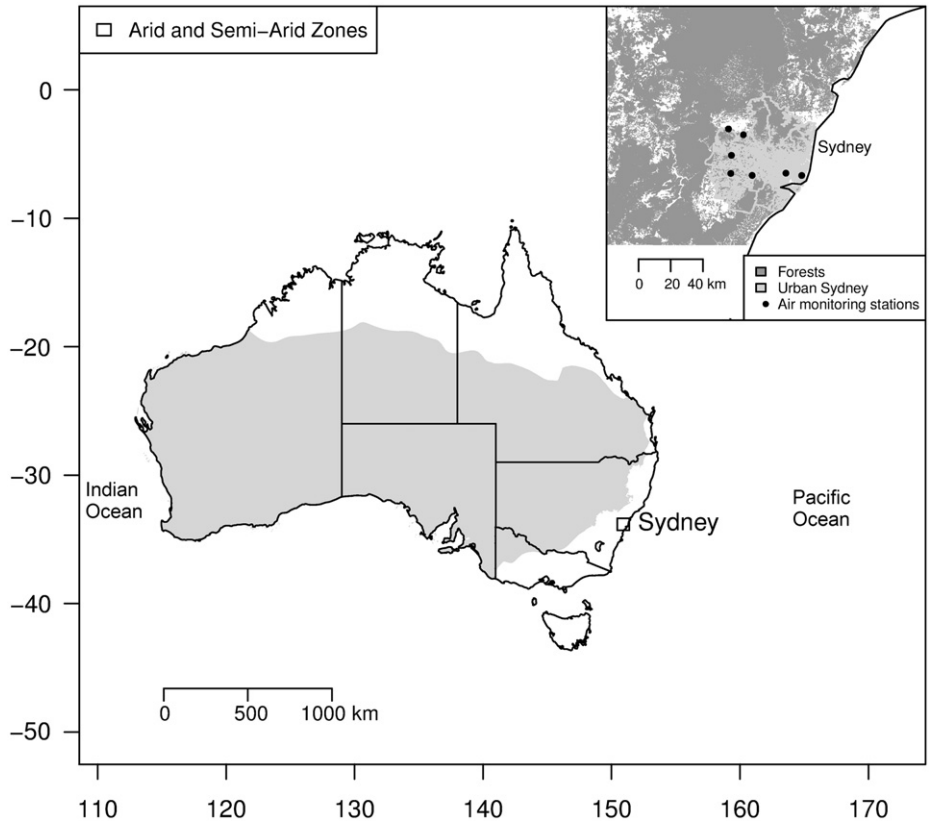

Fig. 1. Map of Australia showing the location of Sydney in relation to arid and semi-arid eco-climatic regions of Australia, the source of most atmospheric dust (based on Hutchinson et al., 2005). Inset shows the urban areas of Sydney, the location of air monitoring stations and adjacent forested areas, the source of most severe bushfire smoke events (Parsons et al., 2006).

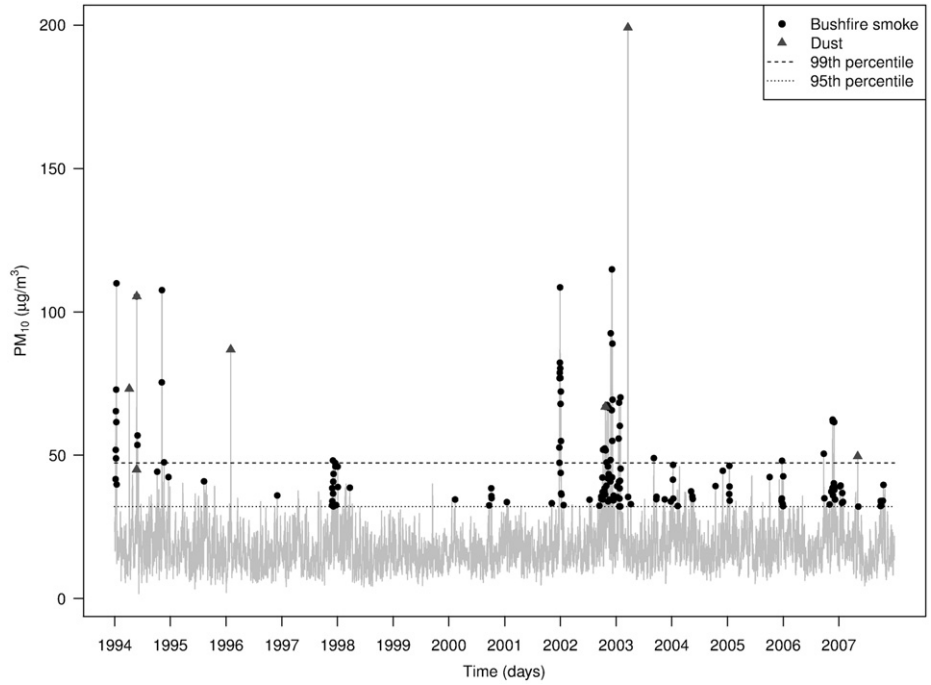

Fig. 2. Daily PM_{10} Sydney, Australia 1994–2007 showing identified bushfire smoke and dust pollution events. The 95th and 99th percentiles are indicated.

3. Results

There were 284,326 deaths during the 13.5 year study period. Nearly half were coded as cardiovascular while approximately 10% were coded as respiratory. There were 52 days on which PM_{10} exceeded the 99th percentile of its distribution (47.3 µg/m^3). All days were successfully identified as being smoke events ($N=46$), dust events ($N=4$), or both ($N=2$). Dust events, though far fewer

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in number, tended to have higher particle concentrations. Summary statistics for covariates are listed in Table 1 and results of the analyses are presented in Table 2.

Smoke events were associated with a 5% increase in non-accidental mortality at a lag of one day OR (95%CI) 1.05 (95%CI: 1.00–1.10). When same-day average temperature was excluded

Table 1
Summary statistics for daily pollutant, fire smoke, dust, meteorological and mortality, Sydney, 1994–2007.

Exposure or outcome	Number of observations	Mean	Minimum	First quartile	Median	Third quartile	Maximum
PM_{10} 24 h city wide average ($\mu g/m^3$)	5113	18.3	1.6	12.8	16.9	21.9	199.2
PM_{10} smoke days ($\mu g/m^3$)	48	67.3	47.3	52.2	63.9	75.8	114.8
PM_{10} dust days ($\mu g/m^3$)	6	96.8	49.6	68.4	80.01	100.8	199.2
PM_{10} background days ($\mu g/m^3$)	5061	17.8	1.64	12.8	16.8	21.7	47.3
24hr ave temp (°C), all days	5113	18.3	7.1	14.5	18.3	21.8	33.9
24 h ave Temp (°C), smoke days	48	23.8	12.8	21.0	23.9	26.8	32.8
24 h ave Temp (°C), dust days	6	22.3	17.0	20.6	23.0	24.8	25.5
24 h Dew Point (°C)	5113	10.6	−5.5	6.7	11.0	14.9	21.9
Ozone 1 h max (ppb)	5113	3.2	0.3	2.4	2.9	3.7	12.0
Non-accidental mortality	284,326	58	30	50	57	64	103
Cardiovascular mortality	120, 686	25	7	20	24	29	58
Respiratory mortality	24, 618	5	1	3	5	7	20

Table 2
Associations between non-accidental, cardiovascular, respiratory and combined cardiorespiratory mortality and severe pollution events due to bushfire smoke and dust for the same day (lag 0) and lags of 1–3 days, Sydney 1994–2007. Results of sensitivity analyses for meteorological covariates, same day maximum ozone, and cut point used to define pollution event are included.

Outcome	Model	Lag	BUSHFIRE SMOKE EVENT			DUST EVENT		
			Odds ratio	Lower 95%CI	Upper 95%CI	Odds ratio	Lower 95%CI	Upper 95%CI
Non-accidental mortality	Basic model[a]	0	1.02	0.98	1.05	1.04	0.92	1.18
		1	**1.05**	**1.00**	**1.10**	0.96	0.85	1.09
		2	1.00	0.95	1.04	0.96	0.85	1.09
		3	1.00	0.96	1.05	**1.16**	**1.03**	**1.30**
	Basic model[a] with exclusion of same day temperature	0	**1.05**	**1.00**	**1.09**	1.07	0.94	1.21
		1	**1.05**	**1.00**	**1.09**	0.97	0.86	1.10
		2	0.98	0.94	1.03	0.96	0.85	1.08
		3	1.00	0.95	1.04	**1.15**	**1.02**	**1.29**
	Basic model[a] with inclusion maximum ozone	0	1.02	0.97	1.07	1.04	0.92	1.17
		1	**1.05**	**1.01**	**1.10**	0.96	0.85	1.09
		2	0.99	0.95	1.04	0.90	0.85	1.09
		3	1.00	0.97	1.06	**1.16**	**1.03**	**1.30**
	Basic model[a] with 3 df for all metrological covariates	0	1.02	0.97	1.06	1.12	0.97	1.30
		1	**1.05**	**1.00**	**1.09**	1.03	0.89	1.19
		2	0.99	0.95	1.04	0.96	0.83	1.12
		3	1.01	0.96	1.06	**1.20**	**1.04**	**1.39**
	Basic model[a] 95th percentile used to define a pollution event	0	1.01	0.98	1.04	1.06	0.95	1.19
		1	1.00	0.98	1.04	1.01	0.90	1.13
		2	0.94	0.96	1.01	0.94	0.84	1.05
		3	1.00	0.97	1.02	**1.17**	**1.05**	**1.30**
Cardiovascular mortality	Basic model[a]	0	1.06	0.97	1.17	1.12	0.87	1.44
		1	1.07	0.98	1.18	0.99	0.76	1.30
		2	0.98	0.89	1.08	1.02	0.77	1.34
		3	1.01	0.92	1.11	1.10	0.85	1.42
	Basic model[a] with exclusion of same day temperature	0	**1.10**	**1.00**	**1.20**	1.14	0.89	1.47
		1	1.07	0.97	1.17	0.99	0.76	1.30
		2	0.96	0.88	1.06	1.02	0.77	1.34
		3	1.01	0.92	1.11	1.10	0.85	1.42
Respiratory mortality	Basic model[a]	0	1.00	0.79	1.25	0.89	0.49	1.63
		1	1.09	0.88	1.36	0.55	0.26	1.17
		2	0.96	0.77	1.20	1.02	0.54	1.91
		3	0.94	0.75	1.17	1.23	0.67	2.24
Combined cardiorespiratory mortality	Basic model[a]	0	1.02	0.97	1.07	1.05	0.88	1.24
		1	1.05	0.97	1.15	1.08	0.86	1.37
		2	0.98	0.89	1.02	1.07	0.79	1.31
		3	1.00	0.92	1.08	1.12	0.89	1.42

[a] Basic model covariates: (1) Flu epidemics, a binary variable coded as 1 if influenza hospital admission rates were $<$90th percentile and otherwise 0; (2) average temperature (°C) for the previous 3 days (lags 1–3) modelled as a non-linear response with 2 degrees of freedom; (3) average dew point for the previous 3 days (lags 1–3) modelled as a non-linear response with 2 degrees of freedom; (4) same-day average temperature (°C) modelled as a non-linear response with 2 degrees of freedom; and (5) same-day average dew point (°C) modelled as a linear response. Bold signifies $p \leq 0.05$.

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from the model, associations of a similar magnitude were also observed on the day of the event (lag 0) OR (95%CI) 1.05 (95%CI: 1.00–1.09). As there were fewer dust events, our effect estimates for this exposure were much less precise. We observed a 15% increase in non-accidental mortality three days following the event OR (95%CI) 1.16 (95%CI: 1.03–1.30). In contrast to smoke events, the outcomes for dust events were not sensitive to the inclusion of same-day temperature. Daily maximum ozone and the number of degrees of freedom used for the meteorological covariates did not influence results for smoke or dust events (Table 2). We did not find any associations with mortality when we included less severe smoke (and dust) pollution events in which PM_{10} exceeded the 95th percentile of 32.0 $\mu g/m^3$ (Table 2.) This is not surprising given that almost half of these events were not validated as being attributable to smoke or dust, and were therefore included as background concentrations.

A larger same day association was observed with smoke events and cardiovascular mortality, but only when same day temperature was excluded from the model OR (95%CI) 1.10 (95%CI: 1.00–1.20). Associations between same day dust events also had a larger point estimates for cardiovascular than for non-accidental mortality, but neither association approached statistical significance. Although we looked for associations between pollution events and respiratory mortality, our results were imprecise as deaths attributed to respiratory causes occur less frequently than those attributed to cardiovascular causes (Table 1). In all analyses of respiratory mortality the 95% confidence intervals were very wide and all included the null (Table 2).

4. Discussion

We have described the associations between non-accidental mortality and severe pollution events caused by bushfire smoke and dust. The impacts were greatest on the day-of and day following a smoke event, and three days following a dust event. During smoke events a high same-day temperature contributed to the same-day effect, but did not affect the lagged association with mortality. Cardiovascular mortality had a similar overall pattern to non-accidental mortality with greater effect size estimates for events, although the results did not reach statistical significance.

The strengths of this study include the long time period for which exposure and event data were available and the number of smoke events in the analyses. Many other studies have been limited to single event days or short-term episodes. However, the infrequent nature of dust storms resulted in very few events available for analysis and, consequently, the results were imprecise. Our research was limited by the need to use city-wide rather than individual level exposure assessment and not all regions of the city would have been equally affected by the smoke or dust events or other meteorological exposures.

Other studies on the association between bushfire smoke and mortality are limited, but their findings have been consistent with those reported here. For example, Morgan et al. (2010) found a 0.8% increase in mortality per 10 $\mu g/m^3$ rise in PM_{10} (95%CI= 0.24–1.86%) during 32 bushfire days in Sydney, but reported no association with cardiovascular mortality. Hänninen et al. (2009) and Sastry (2002) both found increases in mortality in the order of 1% per 10 $\mu g/m^3$ rise in PM_{10} associated with forest fires in Finland and Kuala Lumpur, respectively. Our results are broadly consistent with the current evidence concerning urban particulate air pollution which has been causally associated with daily non-accidental and cardiovascular mortality (Pope and Dockery, 2006).

Studies on the association between dust storms and mortality are even more limited than those on bushfire smoke. Perez et al. (2008) studied 90 Saharan dust intrusions in Barcelona over a two year period and observed a large and statistically significant increase in the mortality associated with coarse and fine PM during dust episodes. Mortality on dust days increased 8.4% per 10 $\mu g/m^3$ of particulate matter at a lag of 1 day. In a study of 39 Asian dust events in Taipei, Chen et al. (2004) observed a 5% increase in mortality at a lag of 2 days, but this did not reach statistical significance and their analyses were not adjusted for meteorology, other pollutants or influenza. Finally, Kwon et al. (2002) studied 28 Asian dust events that affected Seoul during 1995–1998. In a time series analysis (adjusted for meteorological variables but not influenza) they also found associations with non-accidental mortality (3.4% increase; 95%CI=0.5–7.4%) at a lag of 2 days.

The most notable difference between the dust and smoke events in our study was in the lag at which the associations were observed. Although the lag 3 association for dust events could be a chance finding (given the small number of events in the study), it is consistent with other available epidemiological evidence. Both Chen and Kwon reported lag times of 2 days until clinical outcomes were observed. Further, in their study of Asian dust events and hospital admissions in Taiwan, Bell and colleagues noted considerable temporal variation (1–3 days) in association within a single clinical outcome, depending upon which indicator of dust had been included in the analyses (Bell et al., 2008). It is plausible that the spectrum of health impacts associated with dust and smoke pollution events could be different from each other as they have markedly different compositions. Dust is derived from crustal particles, has a lower carbon content, a higher proportion of particles of larger size fractions and a large component of biological particles, such as bacteria, viruses and fungi (Griffin, 2007). It is plausible that longer lag times to adverse health outcomes following dust exposure could be explained by human patho-physiological responses to dust-borne micro-organisms. For example, the development of life-threatening sepsis takes longer than the precipitation of cardiac events, a well described outcome associated with smaller combustion-generated particles (Dennekamp et al., 2010). We were not able to explore this hypothesis in our analysis because there were too few daily deaths attributed to either respiratory or septic causes to enable a meaningful analysis.

The threshold for defining a severe pollution event in this study was the 99th percentile of the distribution of PM_{10} over the entire study period. This equated to 47.3 $\mu g/m^3$, just below Australia's 24-h air quality standard for PM_{10} (50 $\mu g/m^3$). However, in comparison to the air quality in many other countries, the pollution events we studied were relatively minor. Even during the most severe smoke event the city-wide average (114.8 $\mu g/m^3$) did not approach the current US air quality standard for PM_{10} (150 $\mu g/m^3$). Therefore, much higher mortality impacts might be evident in regions where large populations are recurrently exposed to very severe episodes of fire smoke, such as those sometimes cased by annual burning in South East Asia (van der Werf et al., 2010). We did not find associations with mortality when we included events in which PM_{10} exceeded the 95th percentile (32 $\mu g/m^3$) as a sensitivity analysis, which is not surprising. While including less severe pollution events provided additional data points for analysis, background pollution sources remain major contributors to ambient particle concentrations between the 95th and 99th percentiles (Johnston et al., 2011).

One limitation of these analyses is that we cannot determine which elements of the smoke and dust events were most important in the observed associations with mortality. Given that events were defined according to PM_{10} concentrations, we assume that particulate matter is responsible for some of the effect. We have also demonstrated that same-day temperature is an important contributor during bushfire events. However, it is possible that population-wide responses such as increased psychological stress also play a role. Similarly, other environmental

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exposures (e.g. co-pollutants in bushfire smoke or biological particles associated with dust events) could also contribute to adverse health outcomes (Jalava et al., 2006; Perez et al., 2008).

The long distance transport of dust and smoke and the associated health effects highlight the need to consider the nexus between the health of human populations and our landscapes. A hotter and drier climate favors more fire and drought, with resultant slower recovery of vegetation, and a higher risk of overgrazing from livestock (Mitchell et al., 2010; Wang and Hendon, 2007). Thus, the potential for harms from both smoke and dust events are likely to increase in Australia, and other regions with similar environments, as the global climate changes.

5. Conclusions

In summary, we have described associations between non-accidental mortality and severe pollution events caused by bushfire smoke and dust. During smoke events a high same-day temperature contributed to the same-day but not lagged associations with mortality. The implications of this study are threefold: (1) the need for better evidence to inform public health responses during dust storm and bushfire smoke events as both higher temperatures and pollution are likely to play a role in population health outcomes and need to be addressed as part of the public health response; (2) the need for greater acknowledgement of the linkages between the management of dust-prone rangelands and flammable vegetation with the wellbeing of rural and urban communities; and (3) the urgent need for mitigation of climate change to minimise its many inter-related consequences including severe bushfires, worsening air quality, heat stress and drought.

Acknowledgments

Grant McTainsh, John Leys, Grant Williamson and Talia Portner contributed to the identification of severe pollution episodes.

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Please cite this article as: Johnston, F., et al., Extreme air pollution events from bushfires and dust storms and their association with mortality in Sydney, Australia 1994–2007. Environ. Res. (2011), doi:10.1016/j.envres.2011.05.007