Effects of Bushfire Smoke on Daily Mortality and Hospital Admissions in Sydney, Australia

Author(s): Geoffrey Morgan, Vicky Sheppeard, Behnoosh Khalaj, Aarthi Ayyar, Doug Lincoln, Bin Jalaludin, John Beard, Stephen Corbett and Thomas Lumley

Source: *Epidemiology*, Vol. 21, No. 1 (January 2010), pp. 47-55
Published by: Lippincott Williams & Wilkins
Stable URL: http://www.jstor.org/stable/25662805
Accessed: 14-03-2017 00:58 UTC

REFERENCES

Effects of Bushfire Smoke on Daily Mortality and Hospital Admissions in Sydney, Australia

Geoffrey Morgan,[a,b] *Vicky Sheppeard,*[c] *Behnoosh Khalaj,*[d] *Aarthi Ayyar,*[d] *Doug Lincoln,*[a] *Bin Jalaludin,*[e] *John Beard,*[a,f] *Stephen Corbett,*[g] *and Thomas Lumley*[h]

Background: Little research has investigated the health effects of particulate exposure from bushfires (also called wildfires, biomass fires, or vegetation fires), and these exposures are likely to increase, for several reasons. We investigated associations of daily mortality and hospital admissions with bushfire-derived particulates, compared with particulates from urban sources in Sydney, Australia from 1994 through 2002.

Methods: On days with the highest particulate matter $(PM)_{10}$ concentrations, we assumed PM_{10} was due primarily to bushfires. We calculated the contribution of bushfire PM_{10} on these days by subtracting the background PM_{10} concentration estimated from surrounding days. We assumed PM_{10} on the remaining days was from usual urban sources. We implemented a Poisson model, with a bootstrap-based methodology, to select optimum smoothed covariate functions, and we estimated the effects of bushfire PM_{10} and urban PM_{10}, lagged up to 3 days.

Results: We identified 32 days with extreme PM_{10} concentrations due to bushfires or vegetation-reduction burns. Although bushfire PM_{10} was consistently associated with respiratory hospital admissions, we found no consistent associations with cardiovascular admissions or with mortality. A 10 $\mu g/m^3$ increase in bushfire PM_{10} was associated with a 1.24% (95% confidence interval = 0.22% to 2.27%) increase in all respiratory disease admissions (at lag 0), a 3.80% (1.40% to 6.26%) increase in chronic obstructive pulmonary disease admissions (at lag 2), and a 5.02% (1.77% to 8.37%) increase in adult asthma admissions (at lag 0). Urban PM_{10} was associated with all-cause and cardiovascular mortality, as well as with cardiovascular and respiratory hospital admission, and these associations were not influenced by days with extreme PM_{10} concentrations.

Conclusions: PM_{10} from bushfires is associated primarily with respiratory morbidity, while PM_{10} from urban sources is associated with cardiorespiratory mortality and morbidity.

(*Epidemiology* 2010;21: 47–55)

Most epidemiologic studies of the health effects of air pollution have been conducted in urban settings where particulate pollution is derived from a variety of sources. This complicates efforts to disentangle the health effects of particulates from various sources and size fractions, and remains a major gap in current evidence.[1] Particulate matter from bushfires (also called wildfires, vegetation fires, or biomass fires) is an increasing and unregulated source of air pollution. Such fires generate pollution episodes across wide geographic areas and frequently affect major population centers. Bushfire activity is likely to increase with global warming and with associated changes in vegetation burning practices, resulting in increased population exposures to pollution.[2] The increasing use of deliberate fuel-reduction burns to avert major fire disasters, especially where expanding urban populations encroach on bushland around the fringes of cities, is becoming more controversial in light of the evidence of adverse health impacts of particulate air pollution.[3] Wood burning for domestic heating is also increasing in several countries.[4] Therefore, understanding the possible health effects of biomass combustion smoke is becoming increasingly important.

Ambient particles are associated with daily mortality and hospital admissions in many cities with a range of pollutant concentrations and mixtures.[1] The association appears to be linear and no clear threshold concentration has been identified.[1] Several Sydney studies have found similar associations with particulate matter less than 10 μm diameter (PM_{10}) even at relatively low particulate concentrations.[5–10]

Associations of biomass smoke with respiratory problems have been documented in Australia,[11–14] Europe,[15] the United States,[16–20] and South East Asia.[21–23] However, associations

Submitted 20 May 2008; accepted 1 April 2009; posted 10 November 2009.
From the [a]Northern Rivers University, Department of Rural Health, University of Sydney, New South Wales, Australia; [b]North Coast Area Health Service, New South Wales, Australia; [c]Environmental Health Branch, New South Wales Health Department, Sydney, Australia; [d]Centre for Epidemiology and Research, New South Wales Health Department, New South Wales, Australia; [e]Centre for Research, Evidence Management and Surveillance, Sydney South West Area Health Service, and School of Public Health and Community Medicine, University of New South Wales, Australia; [f]Center for Urban Epidemiologic Studies, New York Academy of Medicine, New York, NY; [g]Western Sydney Area Health Service, and School of Public Health, University of Sydney, Sydney, Australia; and [h]Department of Biostatistics, University of Washington, Seattle, WA.
Supported by the Australian Research Council Linkage Grant (LP0882048), SPIRT Grant (2049570), and the New South Wales Department of Health Biostatistical Officer Training Program (to D.L., A.A., and B.K.).

SDC Supplemental digital content is available through direct URL citations in the HTML and PDF versions of this article (www.epidem.com).
Correspondence: Geoff Morgan, Northern Rivers University, Department of Rural Health, University of Sydney, PO Box 3074, Lismore, New South Wales 2480, Australia. E-mail: geoff.morgan@ncahs.health.nsw.gov.au.

ISSN: 1044-3983/10/2101-0047
DOI: 10.1097/EDE.0b013e3181c15d5a

with cardiovascular outcomes are inconsistent.[18,24] A recent review found that wood smoke particles are at least as toxic for respiratory disease as other major categories of combustion-derived particles, but, there is too little evidence to make a judgment about the relative toxicity of wood smoke particles with respect to cardiovascular outcomes.[4]

Despite Sydney's generally low concentrations of particulate matter, the Sydney basin occasionally experiences extreme particulate concentrations due to bushfires in the surrounding bushland.[25,26] Regular fuel-reduction burns are conducted to reduce fuel load and minimize the risk to life and property from uncontrolled bushfires. Occasionally these reduction burns also result in high particulate pollution in Sydney. Sydney's large population exposure to occasional extreme episodes of particulates from bushfires and fuel-reduction burns, against a background of generally low particulate concentration, provides a useful setting in which to investigate the health effects of biomass combustion particulates.

The aims of our study are: to assess the health effects of bushfire particulate compared with urban particulate; to assess the effects of urban particulate while controlling for extreme bushfire events, not controlled for in previous Sydney studies; and to implement a time-series approach that assesses the results' sensitivity to model specification.

Sydney has good daily data on PM_{10} from 1994. A proxy measure of particulates as measured by light scattering, called back scatter particle, is also monitored in Sydney. Back scatter particle is considered to be a good correlate for $PM_{2.5}$ in Sydney and so we also used this proxy as a particulate measure in our analysis. Ozone (O_3) concentrations may be elevated during bushfire episodes and ozone is treated as a potential confounder in this study.

METHODS

We constructed daily time series of mortality, hospital admissions, meteorologic factors, and air pollutants for the Sydney metropolitan area from 1 January 1994 to 30 June 2002, following the APHEA2 protocol.[27–29] Meteorologic data were obtained from the Australian Bureau of Meteorology, and the New South Wales Department of Environment and Climate Change provided the air pollution data. Mortality and population data were obtained from the Australian Bureau of Statistics, and the New South Wales Department of Health provided data on hospital admissions. The specific *International Classification of Diseases—9th Revision* (ICD9) and *10th Revision* (ICD10) codes for the various mortality and hospital admission outcomes assessed are summarized in the online appendix (eTable, http://links.lww.com/EDE/A344). The age groupings are defined as all ages, childhood (1–14 years), adult (15–64 years), and elderly (65+ years). Daily hospital admission for influenza [ICD9: 487; ICD10: J10, J11] were used to identify influenza epidemic days according to the APHEA2 protocol.

Particulate data for the study period was available for PM_{10} measured by tapered element oscillating microbalance (8 monitoring sites), and particulate as measured by the coefficient of light scattering by fine particles in the size range 0.01–2 μm in back scatter particle units per 10 kilometers (8 monitoring sites). We calculated same-day (lag 0) and lagged (lag 1, lag 2, lag 3 days) 24-hour average PM_{10} ($\mu g/m^3$), 24-hour average back scatter particle (10^{-4} m^{-1}), and daily 1-hour maximum ozone (ppb) (10 monitoring sites). $PM_{2.5}$, measured by tapered element oscillating microbalance, was available only from 1997 (3 monitoring sites) and so we were not able to investigate the health effects of $PM_{2.5}$ directly in this study. We also calculated daily $PM_{2.5}$ 24-hour average ($\mu g/m^3$) from January 1997 for comparison purposes.

No registries identifying days affected by bushfires or fuel-reduction burns in Sydney were available for the study period. We identified bushfire days as days with city-wide 24 hours average PM_{10} concentrations greater than the 99th percentile for the study period. We verified that bushfires or fuel-reduction burns occurred on or immediately prior to these bushfire days by checking newspaper archives and other sources.

We assumed PM_{10} on nonbushfire days was derived from miscellaneous urban sources, including vehicles, industry, domestic wood smoke and crustal particles, which we defined as background PM_{10}. We assumed PM_{10} on bushfire days (with PM_{10} above the 99th percentile), was composed primarily of particles from bushfires and reduction burns, but also include background PM_{10} from the usual urban sources. We estimated what background PM_{10} would have been on bushfire days if the bushfire had not occurred, as the 30-day moving average of PM_{10}, with PM_{10} on bushfire days set to missing. Bushfire PM_{10} on bushfire days is the difference between total PM_{10} and background PM_{10}. We took a similar approach to categorizing black scatter particles into bushfire particles and background particles.

We followed a bootstrap-based methodology developed by Dominici[30] to select optimum smooth covariate functions. This approach addresses concerns that the magnitude and statistical uncertainty of the pollutant effect estimates are sensitive to the degree of adjustment for covariate functions, controlled by the number of degrees of freedom (*df*) in the smooth function of time and other covariates. Equation 1 describes the overdispersed Poisson semiparametric model with smoothing splines and asymptotically exact standard errors we implemented using the GAM exact function (http://ihapss.biostat.jhsph.edu/software/gam.exact/gam.exact.htm, accessed 18 March 2004). All analysis was conducted using S-Plus Version 6.1.[31]

$$\begin{aligned}\log E[Y_t] = {} & \beta_1(\alpha)\text{Bushfire } PM_{10} \\ & + \beta_2(\alpha)\ \text{Background } PM_{10} + s(\text{Time}, 7\times\text{Year}\times\alpha) \\ & + s(\text{Temp}_t, 3\times\alpha) + s(\text{TempLag1–3}_t, 3\times\alpha) \\ & + s(\text{RHumid}_t, 3\times\alpha) + s(\text{RHumidLag1–3}_t, 3\times\alpha) \\ & + \text{DayOfWeek} + \text{FluEpi}\end{aligned} \quad (1)$$

Where, Y_t = number of daily deaths, Time = day of study, Year = number of years in study, $Temp_t$ = daily average temperature, $TempLag1–3_t$ = daily temperature average (lag 1+lag 2+lag 3), $RHumid_t$ = daily average relative humidity, $RHumidLag1–3_t$ = daily relative humidity average (lag 1+lag 2+lag 3), DayOfWeek = day of week dummy variable, FluEpi = flu epidemic dummy variable, s represents a smoothing function, and α is a factor multiplying the degree of smoothing (α = 0.2, 0.4, 0.6, 0.8,…, 3) that takes the same value for all variables in the same model.

The weather and other covariates in this model are similar to those identified using the APHEA2 time series approach in recent Sydney studies.[7,8] For each outcome we first identified the background PM_{10} and bushfire PM_{10} lag with the largest t value (positive or negative) for α = 1. We then investigated the sensitivity of the PM_{10} effect estimates ($\beta_1[\alpha]$ and $\beta_2[\alpha]$) to model choice, for each value of α from 0.2 to 3. There is less bias in the effect estimates with smoothing at higher values of α, but the standard errors are larger. For each outcome we assessed the effects of each pollutant using the α that minimized the mean square error (MSE) of $\beta_1(\alpha)$ and $\beta_2(\alpha)$ in Equation 1. Where this optimum α was different for $\beta_1(\alpha)$ and $\beta_2(\alpha)$, we ran Equation 1 twice and present the results using the optimum α for $\beta_1(\alpha)$ and the optimum α for $\beta_2(\alpha)$. Single-pollutant models include both bushfire PM_{10} and background PM_{10} in the same model. We used a similar approach for the back scatter particle measures of background and bushfire particles. We used the approach outlined above to investigate the association between ozone and each outcome. We included ozone in multipollutant models to assess the independent effects of background PM_{10} and bushfire PM_{10} (ozone, bushfire PM10 and background PM10 in the same model).

RESULTS

Data for daily average temperature and relative humidity were missing for only 6 days of the 8.5-year study period. Daily pollutant concentrations were missing on 1 day for PM_{10} and BSP, and no days for ozone. We identified 32 bushfire days with PM_{10} concentrations greater than or equal to the 99th percentile (PM_{10} >42 $\mu g/m^3$). The 32 bushfire days consisted of 2 major bushfire events lasting several days each, 3 events lasting 2 days each, and 9 single-day events. Figure 1 shows a graph of both daily PM_{10} and daily hospital admissions for all respiratory conditions at all ages during the 2 major bushfire events in January 1994 and December 2001. This illustrates the extreme nature of the events and their well-defined period.

All 17 bushfire days in the 2 major events were also described in firefighter publications.[25,26] These 2 major events, 3 other events each lasting 2 days, and 4 single-day events were confirmed by unpublished data from the New South Wales National Parks and Wildlife Service (Ross Bradstock, University of Wollongong, 26 October 2006, written communication). Of the 5 remaining single- day events, 3 were confirmed by newspaper articles.[32–34] PM_{10} concentrations at all monitoring sites throughout the city were extreme on the 2 unconfirmed bushfire days. The lowest concentration at any one site on each day was 67 $\mu g/m^3$, which is indicative of a city-wide fire event in Sydney on both days.

The following results are based on the 32 bushfire days. We conducted several sensitivity analyses including: using

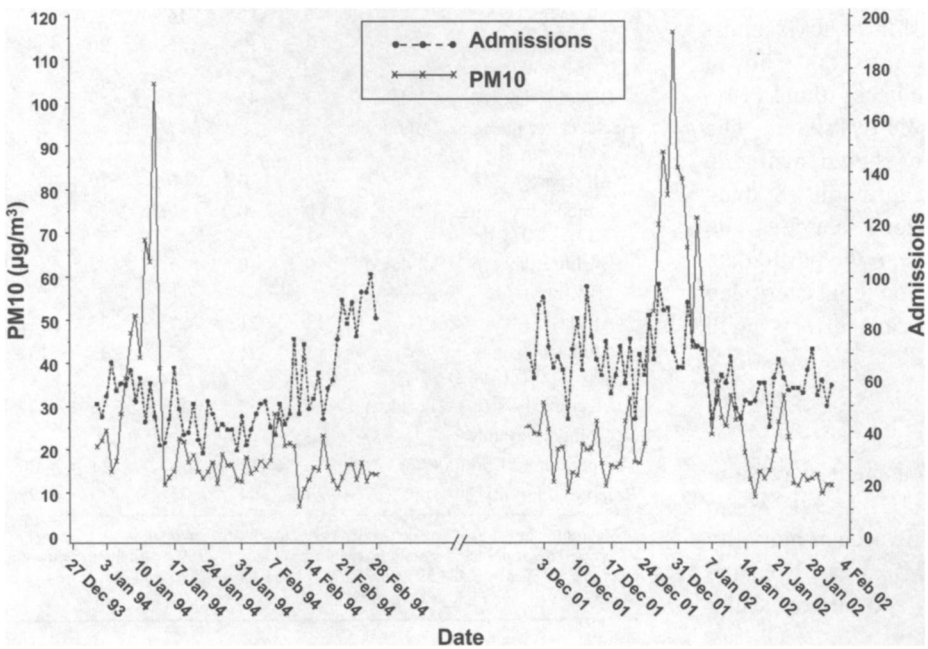

FIGURE 1. Daily respiratory hospital admissions and daily PM_{10} during the 2 major Sydney bushfire events in January 1994 and December 2001.

only those 30 verified bushfire days; using alternative methods to categorize daily PM_{10} into bushfire PM_{10} and background PM_{10}; not categorizing daily PM_{10} into bushfire PM_{10} and background PM_{10}; excluding from the analysis both bushfire PM_{10} and background PM_{10} on bushfire days; and investigating bushfire PM_{10} effects up to lag 7 days. For all these sensitivity analyses (available on request), we found little or no difference to the results for background PM_{10} and bushfire PM_{10} as reported below.

Descriptive statistics for disease outcomes, meteorologic factors and pollutants are summarized in Table 1. Correlations between pollutant and meteorologic parameters are presented in Table 2. From 1997, the mean ratio of $PM_{2.5}$ to PM_{10} on nonbushfire days was 0.58 compared with 0.69 on bushfire days (n = 17 days), indicating that the $PM_{2.5}$ proportion of PM_{10} is generally higher during bushfire days.

The optimum α that minimized the MSE of $\beta_1(\alpha)$ and $\beta_2(\alpha)$ in Equation 1 for all health outcomes generally ranged from 0.8 to 2.0. Some respiratory hospital admission outcomes had higher optimum α values and in these cases the confidence intervals were generally consistently above 1, or consistently below 1 irrespective of α. In such cases, we took a conservative approach and selected a large optimum α value with wider confidence intervals (all results available on request). We used the results from the optimal alpha single-pollutant models to estimate the percentage change and 95% confidence interval (CI) in each health outcome for a 10 $\mu g/m^3$ increase in bushfire PM_{10} and background PM_{10}. The associations of bushfire PM_{10} and background PM_{10} (lags 0–3) with mortality are given in Table 3 and with hospital admissions in Table 4. Plots of these results are included in eFigure (http://links.lww.com/EDE/A344). The background back-scatter-particulate and bushfire back-scatter-particulate single-pollutant model results (not shown, available on request) were similar to the background PM_{10} and bushfire PM_{10} results unless explicitly stated. The ozone single-pollutant model results (not shown, available on request) were similar to those of previous Sydney time-series studies,[5–10] and including ozone as a covariate in the PM_{10} models made little difference to the particulate results. Our results summary focuses on consistent lag effects rather than statistically significant effects with erratic lag structures.

Mortality

Bushfire PM_{10} was associated with a small increase in all-cause mortality at lag 0 days (0.80% [CI = −0.24% to 1.86%]), but was not associated with cardiovascular mortality or respiratory mortality. Background PM_{10} was associated with small increases in all-cause mortality and cardiovascular mortality but not with respiratory mortality.

TABLE 1. Summary Statistics for Daily Mortality and Hospital Admission Counts, Air Pollution Concentrations, and Meteorologic Variables, Sydney, January 1994–June 2002

	Min.	25%	50%[a]	75%	Max.
Mortality (no.)[b]					
All-cause, all ages (n = 177,141)	30	49	56	64	103
Cardiovascular					
All ages (n = 79,057)	8	21	25	30	57
65+ years (n = 70,618)	6	18	22	27	56
Respiratory, all ages (n = 15,981)	0	3	5	7	25
Hospital admissions (no.)[b]					
Cardiovascular					
All ages (n = 287,327)	10	82	92	103	148
65+ years (n = 191,684)	5	53	61	70	110
Cardiac					
All ages (n = 210,379)	9	59	67	76	113
65+ years (n = 137,887)	4	38	44	50	81
Ischemic heart disease					
All ages (n = 101,134)	4	28	32	37	62
65+ years (n = 62,761)	3	17	20	23	44
Stroke, 65+ years (n = 37,692)	1	10	12	15	31
Respiratory					
All ages (n = 273,197)	23	68	84	104	188
65+ years (n = 93,905)	3	22	28	36	88
COPD, 65+ years (n = 36,772)	1	8	11	15	39
Pneumonia and acute bronchitis, 65+ years (n = 29,447)	0	6	9	12	33
Asthma					
1–14 years (n = 40,213)	0	8	11	16	63
15–64 years (n = 25,235)	0	5	8	10	29
Pollutants					
PM_{10} 24-h ($\mu g/m^3$)					
All days	3	12	16	21	117
Nonbushfire days (n = 3071 days)	3	12	16	20	42
Bushfire days (n = 32 days)	43	47	62	80	117
Back scatter particles 24 hours ($10^{-4}m^{-1}$)					
All days	0	0.2	0.2	0.4	4.1
Nonbushfire days (n = 3071 days)	0	0.2	0.2	0.3	1.7
Bushfire days (n = 32 days)	0.2	0.6	1.3	2.5	4.1
O_3 1-h (ppb)					
All days	3	21	27	35	132
Nonbushfire days (n = 3071 days)	3	21	27	35	132
Bushfire days (n = 32 days)	8	35	56	78	124
Meteorologic variables					
Temperature 24-hours (°C)	8	14	18	21	30
Relative humidity 24-hours (%)	24	64	71	79	99

[a]Mean not shown as it was generally very close to the median.
[b]Sydney metropolitan area study region populations (1997 estimated residential population): all ages = 3,482,395 persons; 0–14 years = 687,968 persons; 15–64 years = 2,396,127 persons; 65+ years = 398,300 persons.

TABLE 2. Correlation for Pollutant and Meteorologic Parameters. Correlations for Bushfire Days Only Are Included Below the Diagonal

	PM_{10}	Back Scatter Particles	O_3	$PM_{2.5}$[a]	Temperature	Relative Humidity
PM_{10} 24-hours ($\mu g/m^3$)		0.72	0.43	0.86	0.27	−0.15
Back scatter particles[b] ($10^{-4}m^{-1}$)	0.47		0.23	0.89	−0.02	0.13
O_3 1-h (ppb)	0.28	0.69		0.40	0.56	−0.22
$PM_{2.5}$[a] 24-h ($\mu g/m^3$)	0.84	0.76	0.52		0.10	−0.02
Temperature (°C)						−0.01
Relative humidity (%)						

[a]$PM_{2.5}$ data available only from 1 January 1997 to 30 June 2002 and includes 17 bushfire days.
[b]Back Scatter Particles defined as the coefficient of light scattering by fine particles in the size range 0.01–2 μm.

TABLE 3. Percentage Change and 95% Confidence Interval in Mortality for a 10 $\mu g/m^3$ Increase in Bushfire PM_{10} and Background PM_{10} (up to Lag 3 Days)

	Bushfire PM_{10}		Background PM_{10}	
	α[a]	% Change (95% CI)	α[a]	% Change (95% CI)
All-cause, all ages	0.8		1.2	
Lag 0[b]		0.80 (−0.24 to 1.86)		0.22 (−0.74 to 1.18)
Lag 1		0.18 (−0.84 to 1.22)		1.35 (0.38 to 2.32)
Lag 2		0.15 (−0.88 to 1.19)		1.07 (0.14 to 2.00)
Lag 3		0.30 (−0.74 to 1.34)		0.56 (−0.32 to 1.45)
Cardiovascular, all ages	0.8		1.6	
Lag 0		0.76 (−0.76 to 2.30)		0.36 (−1.09 to 1.82)
Lag 1		0.30 (−1.20 to 1.82)		1.42 (−0.04 to 2.90)
Lag 2		−0.18 (−1.68 to 1.34)		0.51 (−0.89 to 1.93)
Lag 3		0.21 (−1.30 to 1.73)		0.10 (−1.22 to 1.44)
Cardiovascular, 65+ years	0.8		1.6	
Lag 0		0.84 (−0.78 to 2.48)		0.36 (−1.17 to 1.91)
Lag 1		0.35 (−1.23 to 1.97)		1.39 (−0.15 to 2.96)
Lag 2		−0.25 (−1.83 to 1.37)		0.75 (−0.73 to 2.27)
Lag 3		0.29 (−1.30 to 1.91)		0.30 (−1.10 to 1.72)
Respiratory, all ages	0.8		1.4	
Lag 0		−0.32 (−3.70 to 3.18)		0.32 (−3.11 to 3.88)
Lag 1		0.25 (−3.11 to 3.72)		−0.25 (−3.66 to 3.28)
Lag 2		−0.77 (−4.13 to 2.70)		2.06 (−1.32 to 5.54)
Lag 3		0.22 (−3.18 to 3.74)		2.19 (−1.02 to 5.51)

[a]α defined as factor multiplying the degree of smoothing for all variables in the model.
[b]Lag 0 defined as the concentration measured during the 24-hour period from midnight to midnight at the day of death, lag 1 as the previous 24-hour period, and so on.

Hospital Admission

Bushfire PM_{10} was not associated with all cardiovascular disease admissions, cardiac admissions or ischemic heart disease admissions. However, bushfire particles were associated with a small and sustained increase in elderly ischemic heart disease admissions for lag 0 (1.20 [0.34 to 2.07]), lag 1 and lag 2 days, with similar though less precise associations in all-age heart disease admissions. In contrast, background PM_{10} lag 0 was consistently associated with small increases in both all-age and elderly cardiovascular admissions, cardiac admissions and admissions, for ischemic heart disease.

Bushfire PM_{10} was associated with a small and sustained increase in elderly respiratory admissions at lag 0 (1.72 [0.12 to 3.34]), lag 1, and lag 2 days. Although background PM_{10} was also associated with a small increase in elderly respiratory admissions at lag 0 days, the effect decreased quickly at longer lags. Both bushfire PM_{10} and background PM_{10} were associated with small increases in all-age respiratory admissions at lag 0 days.

Bushfire PM_{10} was associated with a moderate and sustained increase in elderly chronic obstructive pulmonary disease (COPD) admissions at lag 0 (3.29 [0.86 to 5.78]), lag 1, lag 2, and lag 3 days. However, background PM_{10} were not associated with elderly COPD admissions.

Bushfire PM_{10} was associated with a moderate increase in elderly pneumonia and acute bronchitis admissions at lag 1 day (2.81 [0.19 to 5.50], but the pattern of lag effects was erratic and the association was not reflected in bushfire back scatter particles. Background PM_{10} was associated with elderly pneumonia and acute bronchitis admissions.

Bushfire PM_{10} was associated with a large increase in admissions for adult asthma at lag 0 days (5.02 [1.77 to 8.37]). In contrast, bushfire PM_{10} was associated with a moderate decrease in childhood asthma admissions at lag 3 days (−3.10 [−6.18 to 0.07]). Background PM_{10} was not associated with an increase in childhood or adult asthma admissions.

DISCUSSION

We found consistent associations in older persons between bushfire particulate and respiratory hospital admissions, beyond the underlying effect of exposure to particulates from urban sources. The strongest association with bushfire particulate was for COPD and other respiratory conditions. These results are consistent with clinical studies of biomass derived particulates[35,36] and with the limited previous population-based research.[14] We did not identify a consistent association between bushfire-generated particulates and cardiovascular admissions or mortality. Extending our analysis to include lag effects up to 7 days indicated that the effects of bushfire particulates on

TABLE 4. Percentage Change and 95 Confidence Interval in Hospital Admissions for a 10 $\mu g/m^3$ Increase in Bushfire PM_{10} and Background PM_{10} (up to Lag 3 Days)

	Bushfire PM_{10}		Background PM_{10}	
	α[a]	% Change (95% CI)	α[a]	% Change (95% CI)
Cardiovascular, all ages	1.8		1.0	
Lag 0[b]		−0.29 (−1.18 to 0.61)		1.22 (0.41 to 2.03)
Lag 1		−0.44 (−1.34 to 0.47)		0.38 (−0.42 to 1.18)
Lag 2		0.08 (−0.82 to 1.00)		−0.11 (−0.87 to 0.67
Lag 3		−0.06 (−0.96 to 0.84)		−0.53 (−1.26 to 0.21)
Cardiovascular, 65+ years	2.0		2.0	
Lag 0		−0.30 (−1.39 to 0.79)		1.30 (0.32 to 2.28)
Lag 1		−0.51 (−1.61 to 0.60)		0.49 (−0.48 to 1.46)
Lag 2		0.52 (−0.59 to 1.65)		−0.01 (−0.93 to 0.93)
Lag 3		0.01 (−1.08 to 1.12)		−0.48 (−1.36 to 0.41)
Cardiac, all ages	1.4		1.0	
Lag 0		−0.50 (−1.49 to 0.51)		1.48 (0.55 to 2.41)
Lag 1		−0.58 (−1.58 to 0.44)		0.82 (−0.09 to 1.74)
Lag 2		0.26 (−0.75 to 1.28)		−0.04 (−0.91 to 0.84)
Lag 3		−0.17 (−1.16 to 0.84)		−0.58 (−1.41 to 0.26)
Cardiac, 65+ years	1.2		1.0	
Lag 0		−0.44 (−1.65 to 0.80)		1.41 (0.27 to 2.56)
Lag 1		−0.77 (−2.00 to 0.48)		0.92 (−0.20 to 2.06)
Lag 2		0.51 (−0.73 to 1.77)		0.14 (−0.93 to 1.23)
Lag 3		−0.16 (−1.38 to 1.07)		−0.42 (−1.45 to 0.61)
Ischemic heart disease, all ages	1.4		0.8	
Lag 0		0.34 (−1.06 to 1.76)		1.17 (−0.09 to 2.45)
Lag 1		0.03 (−1.37 to 1.46)		1.07 (−0.17 to 2.33)
Lag 2		0.29 (−1.13 to 1.73)		0.58 (−0.62 to 1.79)
Lag 3		−0.66 (−2.06 to 0.76)		−0.25 (−1.40 to 0.92)
Ischemic heart disease, 65+ years	0.8		1.0	
Lag 0		1.17 (−0.58 to 2.94)		0.62 (−0.99 to 2.24)
Lag 1		0.89 (−0.86 to 2.67)		1.56 (−0.05 to 3.19)
Lag 2		0.81 (−0.94 to 2.58)		0.96 (−0.59 to 2.53)
Lag 3		−0.49 (−2.21 to 1.25)		−0.19 (−1.65 to 1.29)
Stroke, 65+ years	0.6		1.4	
Lag 0		−0.45 (−2.71 to 1.86)		0.06 (−2.10 to 2.27)
Lag 1		0.26 (−2.05 to 2.62)		−0.34 (−2.49 to 1.85)
Lag 2		0.81 (−1.50 to 3.17)		−0.38 (−2.45 to 1.75)
Lag 3		0.68 (−1.61 to 3.02)		−0.14 (−2.11 to 1.87)
Respiratory, all ages	3.0		1.0	
Lag 0		1.24 (0.22 to 2.27)		1.04 (0.02 to 2.07)
Lag 1		0.72 (−0.32 to 1.78)		0.44 (−0.57 to 1.47)
Lag 2		0.75 (−0.30 to 1.81)		0.37 (−0.61 to 1.36)
Lag 3		−0.20 (−1.23 to 0.85)		0.66 (−0.28 to 1.61)
Respiratory, 65+ years	2.6		1.0	
Lag 0		1.72 (0.12 to 3.34)		2.34 (0.83 to 3.87)
Lag 1		1.67 (0.05 to 3.31)		0.85 (−0.63 to 2.36)
Lag 2		2.31 (0.69 to 3.96)		0.26 (−1.18 to 1.72)
Lag 3		1.36 (−0.22 to 2.96)		0.25 (−1.13 to 1.65)
COPD, 65+ years	0.8		1.2	
Lag 0		3.29 (0.86 to 5.78)		0.57 (−1.74 to 2.93)
Lag 1		3.65 (1.18 to 6.19)		−0.23 (−2.52 to 2.12)
Lag 2		3.80 (1.40 to 6.26)		−0.62 (−2.85 to 1.67)
Lag 3		2.87 (0.51 to 5.28)		−0.09 (−2.23 to 2.10)
Pneumonia and acute bronchitis, 65+ years	0.8		1.0	
Lag 0		1.86 (−0.72 to 4.52)		2.81 (0.16 to 5.53)
Lag 1		2.81 (0.19 to 5.50)		2.14 (−0.49 to 4.84)
Lag 2		0.23 (−2.26 to 2.78)		1.96 (−0.60 to 4.59)
Lag 3		1.93 (−0.55 to 4.48)		0.62 (−1.81 to 3.11)
Asthma, 1–14 years	1.6		1.0	
Lag 0		−1.85 (−5.00 to 1.42)		−0.62 (−3.42 to 2.25)
Lag 1		0.63 (−2.41 to 3.77)		−1.13 (−3.91 to 1.74)
Lag 2		1.13 (−1.95 to 4.30)		0.61 (−2.12 to 3.42)
Lag 3		−3.10 (−6.18 to 0.07)		1.45 (−1.21 to 4.18)
Asthma, 15–64 years	2.8		0.8	
Lag 0		5.02 (1.77 to 8.37)		−0.94 (−3.62 to 1.82)
Lag 1		2.50 (−0.69 to 5.78)		−1.11 (−3.76 to 1.61)
Lag 2		0.86 (−2.26 to 4.08)		0.24 (−2.36 to 2.91)
Lag 3		0.63 (−2.48 to 3.84)		0.67 (−1.86 to 3.26)

[a]α defined as factor multiplying the degree of smoothing for all variables in the model.

[b]Lag 0 defined as the concentration measured during the 24-hour period from midnight to midnight at the day of hospital admission, lag 1 as the previous 24-hour period, and so on.

respiratory admissions did not extend more than 3 days after the bushfire exposure.

During periods of almost continuous bushfire activity in northern Australia, Johnston[11] found an increased risk of asthma emergency department attendance. During the January 1994 Sydney bushfire, Jalaludin[12,13] observed only minor effects on respiratory symptoms and lung function in a cohort of 32 children with wheeze, whereas Smith[37] found no impact on children's emergency attendances for asthma. Several studies have been conducted after wildfire events in California. Kunzli[16] carried out a questionnaire-based survey to assess the effects of the 2003 wildfires on the health of children and adolescents, and found substantial effects of wildfire smoke exposure on eyes as well as upper and lower respiratory symptoms of children and adolescents. Mott[18] found that medical

visits for respiratory illness increased, and that people experienced worsening of lower-respiratory-tract symptoms during a Californian wildfire. This was most pronounced in people with pre-existing cardiopulmonary conditions.

Domestic use of wood for heating or cooking creates a particulate exposure similar to bushfires. In Christchurch, New Zealand, winter particulate is primarily derived from domestic wood smoke, and the composition of particles is likely to be similar to that from bushfire smoke. Like our results for Sydney, a recent study in Christchurch found no association of PM_{10} with cardiovascular admission.[10] An earlier study in Christchurch found an association of PM_{10} with admissions for heart failure, but not with other cardiac diagnosis. This earlier study also found associations with respiratory admissions, but in contrast to our Sydney findings the largest effects were observed in children.[24] A recent review found that in modern societies the effects on asthma of residential wood smoke were stronger than the effects of particulate from other sources.[38]

Although our observed protective effect of bushfire particulate on childhood asthma contrasts with the observations from a number of wood-smoke-affected locations, it is consistent with Smith's study of the 1994 bushfire event in Sydney.[37] This may be due to modifying factors during the extreme events we studied, compared with the more familiar exposures of domestic wood smoke or the regular bushfire activity studied by Johnston in northern Australia.[11] Parents of children with asthma may take extra precautions with their care during days when PM levels are noticeably extreme. Children with asthma are usually treated and may have had better access to medical treatment. In California, Kunzli[16] found that individuals with asthma were more likely to take preventive action such as wearing masks or staying indoors during wildfires, and a surveillance report by the US Centers for Disease Control and Prevention found increased over-the-counter sales of medication after wildfires.[39] As with other time-series studies of childhood asthma, there was more difficulty in our study modeling the series for children due to the complex seasonal cycles.[6,40] This may be because children are more likely to experience frequent respiratory epidemics and because school holidays and midterm holidays influence hospital admission rates.[41,42]

Since the late 1990s, the New South Wales Health Department has issued warnings during extreme bushfire events in Sydney to advise people with asthma and chronic cardiopulmonary conditions to stay indoors, reduce exercise, and use preventive medications.[43] The observed protective effect of bushfire particulate on childhood asthma may be partly due to the influence of these health warnings in modifying behavior of children with asthma. The warnings may not be as effective for adults with asthma and COPD. A study of the public health impact of interventions on the Hoopa Valley Indians in the United States found that recollection of public service announcements about avoiding smoke were associated with decreased lower-respiratory-tract effects.[18]

Although we found consistent associations of bushfire particulate with respiratory admissions, we did not find consistent evidence of an effect on cardiovascular admissions. However, the association between bushfire particulates (as measured by a proxy measure of particulate) and admissions for ischemic heart disease suggests that an effect of bushfire particulate on cardiovascular morbidity requires further investigation. Any such effect on cardiovascular morbidity may be driven by an association with $PM_{2.5}$ rather than PM_{10}, as the $PM_{2.5}$ proportion of PM_{10} is generally higher during bushfire days than nonbushfire days, and the correlations of PM_{10} and $PM_{2.5}$ with back scatter particles suggest that the latter is a better proxy for $PM_{2.5}$ than for PM_{10} during bushfire periods. Research into the effects of biomass smoke on nonrespiratory (including cardiovascular) outcomes has been highlighted as requiring further work.[4]

The association we found between hospital admissions and bushfire particulate was not reflected in all-cause mortality. We found no evidence of an association between respiratory mortality and bushfire particulate. However, the number of daily deaths ascribed to respiratory mortality in Sydney is low, with a median of 5 (4 during bushfire days) and this limits the power of the analysis to find an effect even if one were present.

The associations we found between background PM_{10} and cardiorespiratory admissions and mortality are similar to those in previous Sydney studies,[5–10] suggesting that the effects of nonbushfire particulate are not driven by high-particulate days. The model-building we performed to select the optimum smooth covariate functions indicates that the magnitude of the PM effect estimates for some outcomes are sensitive to the degree of covariate smoothing. The potential for exposure misclassification is a limitation of our study, as it is possible we missed some bushfire days due to a lack of historical information identifying bushfire days in Sydney. We ensured as far as practicable that only actual bushfire days are included in the PM_{10}-bushfire analysis. Missing some bushfire days would reduce the power of this analysis to find effects (if one is present), but it would be unlikely to bias the results. While Sydney does experience occasional extreme concentration of PM_{10} due to bushfires and fuel-reduction burns, these incidents are rare and PM_{10} is usual relatively low in Sydney. For this entire period, PM_{10} was low (median PM_{10} = 16 $\mu g/m^3$, 75th percentile PM_{10} = 20 $\mu g/m^3$). It is possible that we included a small number of extra bushfire days with days categorized as background days in our analysis of background PM_{10}. Any such inclusions would be unlikely to influence the background PM_{10} results due to the

large number of nonbushfire days in our 8.5-year study period. The sensitivity analysis we conducted that did not categorize daily PM_{10} into bushfire PM_{10} and background PM_{10} found results similar to those reported here for background PM_{10}. This suggests that including additional bushfire days with nonbushfire days in our background PM_{10} analysis would not bias these PM_{10} results.

In summary, bushfire particulate in Sydney was consistently associated with respiratory morbidity but not cardiovascular morbidity. In contrast, particulate derived from urban sources was associated with cardiovascular and respiratory mortality and morbidity, and these associations were not driven by occasional extreme particulate concentrations due to smoke from bushfires or fuel-reduction burns. For uncontrolled events such as bushfires, improving communication through public health messages may assist in reducing health effects. The population health effects of particulates from fuel-reduction burns may be reduced by conducting such burns in conditions that minimize smoke exposure to populated locations.

ACKNOWLEDGMENTS

We thank the Australian Bureau of Statistics for providing the mortality data, the New South Wales Department of Health for providing the hospital admissions data via the HOIST data access, analysis and reporting facility, Alan Betts from the New South Wales Department of Environment and Climate Change for providing the air pollution data, and the Australian Bureau of Meteorology for the meteorological data. We also acknowledge collaborators on the SPIRT project, including Gail Williams from the School of Population Health, University of Queensland, Queensland, Australia. Special thanks to Ross Bradstock, University of Wollongong, for his invaluable assistance verifying bushfire pollution affected days in Sydney, and to Bert Brunekreef, Universiteit Utrecht, for his thoughtful comments on an earlier draft of the paper.

REFERENCES

1. Pope CA, Dockery DW. Health effects of fine particulate air pollution: lines that connect. *J Air Waste Manage Assoc.* 2006;56:709–742.
2. Westerling AL, Hidalgo HG, Cayan DR, Swetnam TW. Warming and earlier spring increases western U.S. forest wildfire activity. *Science.* 2006;313:940–943.
3. Ellis S, Kanowski P, Whelan R. *National inquiry on bushfire mitigation and management (Canberra, ACT).* Canberra, Australia: Commonwealth of Australia; 2004.
4. Naeher LP, Brauer M, Lipsett M, et al. Woodsmoke health effects: a review. *Inhal Toxicol.* 2007;19:67–106.
5. Morgan G, Corbett S, Wlodarczyk J, Lewis P. Air pollution and daily mortality in Sydney, Australia, 1989 through 1993. *Am J Public Health.* 1998;88:759–764.
6. Morgan G, Corbett S, Wlodarczyk J. Air pollution and daily hospital admissions in Sydney, Australia, 1990 to 1994. *Am J Public Health.* 1998;88:1761–1766.
7. Simpson R, Williams G, Petroeschevsky A, et al. The short-term effects of air pollution on hospital admissions in four Australian cities. *Aust N Z J Public Health.* 2005;29:213–221.
8. Simpson R, Williams G, Petroeschevsky A, et al. The short-term effects of air pollution on mortality in four Australian cities. *Aust N Z J Public Health.* 2005;29:205–212.
9. Barnett AG, Williams GM, Schwartz J, et al. Air pollution and child health. *Am J Respir Crit Care Med.* 2005;171:1272–1278.
10. Barnett AG, Williams GM, Schwartz J, et al. The effects of air pollution on hospitalisations for cardiovascular disease in elderly people in Australian and New Zealand cities. *Environ Health Perspect.* 2006;114:1018–1023.
11. Johnston FH, Kavanagh AM, Bowman DMJS, Scott RK. Exposure to bushfire smoke and asthma: an ecological study. *Med J Aust.* 2002;176:535–538.
12. Jalaludin B, Smith M, O'Toole B, Leeder S. Acute effects of bushfires on peak expiratory flow rates in children with wheeze: a time series analysis. *Aust N Z J Public Health.* 2000;24:174–177.
13. Jalaludin B, O'Toole B, Morgan G, Leeder S. Acute effects of bushfires on respiratory symptoms and medication use in children with wheeze in Sydney, Australia. *Environ Health.* 2004;4:20–29.
14. Chen L, Verral K, Tong S. Air particulate pollution due to bushfires and respiratory hospital admissions in Brisbane, Australia. *Int J Environ Health Res.* 2006;16:181–191.
15. Ovadnevaite J, Kvietkus K, Marsalka A. 2002 summer fires in Lithuania: impact on the Vilnius city air quality and the inhabitants health. *Sci Total Environ.* 2006;356:11–21.
16. Kunzli N, Avol E, Wu J, et al. Health effects of the 2003 Southern California wildfires on children. *Am J Respir Crit Care Med.* 2006;174:1221–1228.
17. Duclos P, Sanderson LM, Lipsett M. The 1987 forest fire disaster in California: assessment of emergency room visits. *Arch Environ Health.* 1990;45:53–58.
18. Mott JA, Meyer P, Mannino D, Redd SC. Wildland forest fire smoke: health effects and intervention evaluation, Hoopa, California, 1999. *West J Med.* 2002;176:157–162.
19. Viswanathan S, Eria L, Diunugala N, Johnson J, McClean C. An analysis of effects of San Diego wildfire on ambient air quality. *J Air Waste Manage Assoc.* 2006;56:56–67.
20. Moore D, Copes R, Fisk R, Joy R, Chan K, Brauer M. Population health effects of air quality changes due to forest fires in British Columbia in 2003: estimates from physician-visit billing data. *Can J Public Health Revue Canadienne de Sante Publique.* 2006;97:105–108.
21. Kunii O, Kanagawa S, Yajima I, et al. The 1997 haze disaster in Indonesia: its air quality and health effects. *Arch Environ Health.* 2002;57:16–22.
22. Emmanuel S. Impact to lung health of haze from forest fires: the Singapore experience. *Respirology.* 2000;5:175–182.
23. Aditama T. Impact of haze from forest fire to respiratory health: Indonesian experience. *Respirology.* 2000;5:169–174.
24. McGowan J, Hider R, Chacko E, Town G. Particulate air pollution and hospital admissions in Christchurch, New Zealand. *Aust N Z J Public Health.* 2002;26:23–29.
25. Crombie-Brown J, ed. *A State Ablaze—The January 1994 Fires.* Rosehill, New South Wales: New South Wales Rural Fire Service; 1998.
26. New South Wales Fire Brigade. Christmas 2001/2002 Bushfires. *Fire News.* Autumn 2002;3.
27. Atkinson RW, Anderson HR, Sunyer J, et al. Acute effects of particulate air pollution on respiratory admissions. *Am J Respir Crit Care Med.* 2001;164:1860–1866.
28. Katsouyanni K, Touloumi G, Samoli E, et al. Confounding and effect modification in the short-term effects of ambient particles on total mortality: results from 29 European cities within the APHEA2 project. *Epidemiology.* 2001;12:521–531.
29. Le Tertre A, Medina S, Samoli E, et al. Short-term effects of particulate air pollution on cardiovascular diseases in eight European cities. *J Epidemiol Community Health.* 2002;56:773–779.
30. Dominici F, McDermott A, Hastie T. Improved Semi-Parametric Time Series Models of Air Pollution and Mortality. *J Am Stat Assoc.* 2004;468:938–948.
31. Insightful Corporation. *S-Plus 6.1 for Windows Professional Edition (Release 1).* Seattle, WA: Insightful Corporation; 2002.
32. Pollution sparks fire ban. *The Sydney Morning Herald.* 30 May 1994.
33. Miller G, Molitorisz S. Three hurt as fierce winds cut a swathe of damage. *The Sydney Morning Herald.* 7 October 1994.

34. Brown M. Heat-hit firefighters hang in for a cooler Tuesday. *The Sydney Morning Herald*. 23 March 1998.
35. Orozco-Levi M, Garcia-Aymerich J, Villar J, Ramirez-Sarmient A, Anto JM, Gea J. Wood smoke exposure and risk of chronic obstructive pulmonary disease. *Eur Respir J*. 2006;27:542–546.
36. Dennis RJ, Malonado D, Baena E, et al. Wood smoke exposure and risk for obstructive airways disease among women. *Chest*. 1996;109(suppl 3):55S–56S.
37. Smith M, Jalaludin B, Byles J, Lim L, Leeder SR. Asthma presentations to emergency departments in western Sydney during the January 1994 Bushfires. *Int J Epidemiol*. 1996;25:1227–1236.
38. Boman B, Forsberg A, Jarvholm B. Adverse health effects from ambient air pollution in relation to residential wood combustion in modern society. *Scand J Work Environ Health*. 2003;29:251–260.
39. Johnson JM, Hicks L, McClean C, Ginsberg M. Leveraging syndromic surveillance during the San Diego wildfires, 2003. *Morb Mortal Wkly Rep*. 2003;54(suppl):190.
40. Schwartz J, Spix C, Touloumi G, et al. Methodological issues in studies of air pollution and daily counts of deaths or hospital admissions. *J Epidemiol Community Health*. 1996;50(suppl 1):S3–S11.
41. Storr J, Lenney W. School holidays and admissions with asthma. *Arch Dis Child*. 1989;64:103–107.
42. Lincoln D, Morgan G, Sheppeard V, Jalaludin B, Corbett S, Beard J. Return to school after term holidays is associated with increased hospital admissions for childhood asthma in Sydney, Australia. *Public Health*. 2006;120:854–862.
43. New South Wales Health. *Air Pollution Health Alerts*. Sydney, Australia: NSW Health; 2005.