Air pollution from bushfires and their association with hospital admissions in Sydney, Newcastle and Wollongong, Australia 1994–2007

Abstract

Objective: We examined the association between validated bushfire smoke pollution events and hospital admissions in three eastern Australian cities from 1994 to 2007.

Methods: Smoke events were defined as days on which bushfire smoke caused the 24-hour citywide average concentration of airborne particles to exceed the 99th percentile of the daily distribution for the study period. We used a time-stratified case-crossover design to assess the association between smoke events and hospital admissions. Odds ratios (OR) and 95% confidence intervals (CI) were estimated for cardiovascular and respiratory conditions on event days compared with non-event days. Models were adjusted for daily meteorology, influenza epidemics and holidays.

Results: Smoke events occurred on 58 days in Sydney (population: 3,862,000), 33 days in Wollongong (population: 406,000) and 50 days in Newcastle (population: 278,000). In Sydney, events were associated with a 6% (OR=1.06, 95%CI=1.02-1.09) same day increase in respiratory hospital admissions. Same day chronic obstructive pulmonary disease admissions increased 13% (OR=1.13, 95%CI=1.05-1.22) and asthma admissions by 12% (OR=1.12, 95%CI=1.05-1.19). Events were also associated with increased admissions for respiratory conditions in Newcastle and Wollongong.

Conclusions: Smoke events were associated with increased hospital admissions for respiratory but not cardiovascular conditions. Large populations are needed to assess the impacts of brief exposures.

Implications: Public health impacts from bushfire pollution events are likely to increase in association with a warming climate and more frequent severe fire weather.

Key words: vegetation fires, bushfire smoke, hospital admissions, particulate air pollution, respiratory disease, cardiovascular disease

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Bushfires are an ever-increasing problem in Australia. The bushfire season is getting longer and episodes of severe fire weather are becoming more frequent.[1-3] As a result, costs from infrastructure losses and deaths have also been increasing.[4] From 1967 to 1999 insurance claims from bushfires averaged $77 million per year. The bushfires in January 2003 caused more than $300 million in claims, and those from the 2009 Victorian bushfires were estimated to be in excess of $4.4 billion.[3,5] In an attempt to reduce fuel loads and the risk of hazardous fires, prescribed burning on public lands has been recommended by The Royal Commission into the Victorian Bushfires of 2009. However, they acknowledge that prescribed burning is a controversial practice as it is risky and resource intensive, and has a range of adverse effects on local communities, especially with respect to air quality.[3]

A management constraint is the relative lack of good-quality evidence specifically concerning the health impacts of bushfire smoke, and uncertainty about the applicability of findings from the vast literature on urban air pollution, most of which concerns pollution derived from industry and transport, rather than vegetation fires. A growing body of research has consistently shown that elevated concentrations of particulate matter (PM) from vegetation fire smoke are associated with respiratory morbidity, especially related to adult asthma and chronic obstructive pulmonary disease (COPD).[6-8] Fewer studies of mortality have been published, with only two[9,10] of five studies[11-13] reporting a significant association with smoke-related PM. Similarly, studies of associations with cardiovascular disease outcomes are limited and report inconsistent findings.[11,14-19] Dennekamp et al.[20] showed an increased risk of out-of-hospital cardiac arrests on days affected by bushfire smoke in Melbourne, Australia. Another study on peat bog smoke found associations with cardio-pulmonary symptoms and heart

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failure in North Carolina, US.[21] However, other studies have been less conclusive. Johnston et al.[16] and Hanigan et al.[15] found no associations with admissions for cardiovascular conditions, with the exception of a significant positive association for Indigenous Australians, a high-risk group. Similarly, Henderson et al.[22] found associations for respiratory physician attendances and hospital admissions, but found no associations for cardiovascular outcomes.

Our aim was to investigate the link between PM related to bushfire smoke (PM_{10} or $PM_{2.5}$) and morbidity as measured by overall hospital admissions, and specifically for respiratory and cardiovascular diseases, in a multi-centre study of three eastern Australian cities intermittently affected by bushfire smoke. These were Sydney, Wollongong and Newcastle, with populations of about 3,862,000; 278,000; and 406,000, respectively.[23] All three cities lie on a coastal lowland plain surrounded by elevated sandstone tablelands largely covered with thick eucalypt forests (Figure 1). The main sources of background PM include motor vehicle and industrial emissions, domestic wood smoke and crustal particles.[24] The majority of fire smoke is derived from prescribed and wild fires in the eucalypt forests in the Blue Mountains to the west of the region.[25] Although fire activity is highly variable, these cities are affected by severe pollution events from bushfires for an average of three days each year.[25]

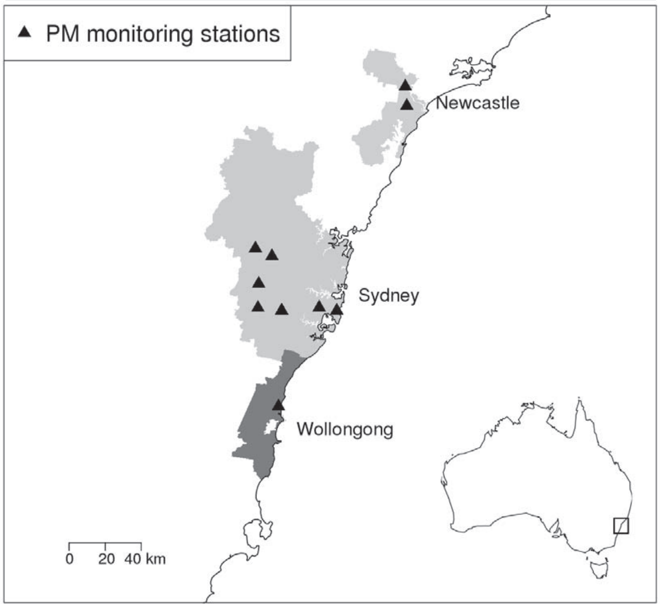
Methods

Exposure measurement

Air quality data were obtained from the NSW Office of Environment and Heritage (www.environment.nsw.gov.au/AQMS/search.htm). They provided 13.5 years of daily average concentrations of PM less than 10 microns (PM_{10}) and less than 2.5 microns ($PM_{2.5}$) in aeronautic diameter. Both PM fractions were measured by tapered element oscillating microbalances at seven monitoring stations in Sydney, one in Wollongong and two in Newcastle (Figure 1). Data were only included if it were available for at least 70% of the days in the time series. We calculated daily city-wide averages of PM_{10} concentrations from 1994 to 2007, and $PM_{2.5}$ concentrations from 1996 to 2007 (1998–2007 in Wollongong). Missing values were imputed using the weighted average values from all proximal monitoring sites in accordance with the protocol from the APHEA (Air Pollution and Health: a European Approach) study.[26]

A database of validated vegetation fire smoke pollution events for this region from 1994-2007 has been previously described.[25] In brief, smoke events were defined as those days when the PM_{10} and/or $PM_{2.5}$ exceeded the 99th percentile of the entire time series and the elevated PM could be attributed to vegetation fires (including bushfires, forestry regeneration and agricultural burning) using media sources, government records and/or remote sensing data. Days above the 99th percentile, but not conclusively validated as being affected by vegetation fires, were classified as background PM days. The 99th percentile was chosen as it corresponds closely to Australian air quality standards and enables a clear delineation between background and bushfire days. Bushfires close to the study populations have occurred since 2007. However the extent to which

Figure 1: Map of the PM monitoring stations in the cities of Sydney, Wollongong and Newcastle, Australia.



these influenced city-wide mean concentrations of particulate matter has not been evaluated.

The Australian Bureau of Meteorology provided daily average ambient temperature and humidity, as measured by dew point temperature.[27] The inverse distance weighted averages of all valid weather station observations within 50 kilometres of the population weighted centres of each city were calculated and merged with the health data.[28] Epidemics of influenza were defined as days with hospital admission rates for influenza (ICD9 code 487 and ICD10 codes J10-J11) greater than the 90th percentile of the distribution for greater Sydney metropolitan region.[29] Public and school holidays for the NSW region were collated from a comprehensive listing of events[30] and diaries and calendars.

Outcome data

The study population was defined geographically according to statistical local area of residence. We included all statistical local areas in the Sydney, Newcastle and Wollongong metropolitan areas for which representative air quality monitoring data were available. Hospital admissions data were provided by the Department of Health in NSW, coded according to the World Health Organization's International Classification of Diseases (ICD).[31] Until December 1996, the primary diagnosis for admissions to hospital was coded according to the ICD 9th revision (ICD-9) and thereafter according to the 10th revision (ICD-10). To map from ICD-9 to ICD-10 codes we followed the protocols of the National Casemix and Classification Centre.[32] The association between smoke events and hospital admissions was examined for all non-trauma admissions (ICD9 <800, ICD10 A00-R99), and specifically for respiratory (ICD9 = 460-519; ICD10 = J00-J99 {excluding J95.4-J95.9} R09.1, R09.8) and cardiovascular (ICD9 = 390-459; ICD10 = I00-I99 {excluding I67.3, I68, I88, I97.8, I97.9, I98}, G45 {excluding G45.3}, G46, M30, M31, R58) diagnoses. Respiratory subgroups included asthma (ICD9 = 493; ICD10 = J45-J46); chronic obstructive pulmonary

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disease (ICD9 = 490-492,494-496; ICD10 = J40-J44, J47, J67); and pneumonia and acute bronchitis (ICD9 = 466,480-486; ICD10 = J12-J17, J18.0, J18.1, J18.8, J18.9, J20, J21). Cardiovascular subgroups included arrhythmia (ICD9 = 427; ICD10 = I46-I49); cardiac failure (ICD9 = 428; ICD10 = I50); cerebrovascular disease (ICD9 = 430-438; ICD10 = I60-I66, I67); and ischemic heart disease (ICD9 = 410-413; ICD10 = I20-I25).

Statistical analyses

The study followed a time-stratified case-crossover design in which the event status (smoke event or background day: a binary variable 1,0) on the day of hospitalisation (or up to three days before) was compared with the event status on control days matched by day of week, month and calendar year. This design controls for the potential effects of day of week, season and long term trends on hospital admissions.[33] We used conditional logistic regression models adjusted for temperature, dew point, influenza epidemics and public holidays to estimate the odds ratio (OR) for hospital admissions associated with smoke events compared with the background. For asthma, we also included school holidays as a covariate in the models.[34] As just 58 bushfire smoke days were available for analysis in Sydney, and slighter fewer in the other two centres, our sample size was limited, and for this reason we did not stratify analyses by gender or age.

To determine the optimal number of degrees of freedom for modelling the meteorological covariates we fit natural cubic splines with varying degrees of freedom for each temperature and humidity variable and assessed the difference in the Akaike Information Criterion (AIC). The models included the covariates as follows: average temperature and humidity (ºC – same day average temperature/humidity {measured as dew point temperature} modelled as a non-linear variable with 3 degrees of freedom, df); lagged temperature and humidity (ºC – average temperature/humidity for the previous 3 days {lags1-3} modelled as a non-linear variable with 3 df); and a binary variable for influenza epidemic (if influenza hospital admissions rates were >90th percentile), public holidays and school holidays (for asthma only). A sensitivity analysis was undertaken to ascertain whether Ozone changed the associations. All analyses were conducted using the R statistical software package.[35]

Ethical approval

This study was approved by the Tasmanian Human Research Ethics Committee (H0010047) and the Human Research Ethics Committee of the Australian National University (2008/199).

Results

During the study period there were 58 days validated smoke event days in Sydney, 33 in Wollongong and 50 in Newcastle. The vast majority were due to bushfires, rather than agricultural or other types of planned vegetation fires. Summary statistics for daily PM_{10} and $PM_{2.5}$ and meteorological variables for each city are shown in Table 1. In Sydney, there were a total of 3,141,017 non-trauma hospital admissions, 273,034 in Wollongong and 345,736 in Newcastle. Of those, between 17% and 19% were coded as cardiovascular admissions and between 13% and 15% as respiratory admissions, depending on the city.

In Sydney, smoke events were significantly (p<0.05) associated with a 2% increase in all non-trauma hospital admissions on the same day as the event (OR=1.02, 95%CI=1.00–1.03). Smoke events were not associated with the total number of same day hospital admissions in Wollongong or Newcastle. These results were all independent of temperature (same day and previous three days), humidity (same day and previous three days), influenza epidemics, and public holidays.

The associations between smoke events and hospital admissions for all respiratory conditions are shown in Figure 2 and associations

Table 1: Summary statistics for particulate concentration-meteorological variables and hospital admissions – Greater NSW – 1994-2007.

	Sydney			Wollongong			Newcastle		
	N	Mean	Range	N	Mean	Range	N	Mean	Range
PM10 24hr city-wide average µg/m³	4,929	18.4	1.6-199.2	4,624	19.0	2.5-280.5	4,800	19.4	2.0-160.9
PM2.5 24hr city-wide average µg/m³	4,006	10.2	2.1-100.2	3,359	9.4	1.6-112.2	3,868	9.7	1.8-61.9
PM10 smoke days µg/m³*	48	67.3	47.3-114.8	31	68.8	50.9-280.5	34	67.1	49.9-160.9
PM2.5 smoke days µg/m³ *	36	43.9	27.4-100.2	31	38.3	24.7-112.2	37	34.5	25.2-61.9
24hr avg Temp ˚C-all days	4,929	18.3	7.2-33.9	4,929	17.5	8.0-32.4	4,929	18.3	8.7-33.1
24hr Dew Point ˚C	4,929	10.7	–5.5-21.9	4,929	11.4	–6.2-23.5	4,929	12.5	–3.7-23.7
Hospital Admissions:									
Total Admissions	3,141,017	2770	1690-4893	273,034	241	78-421	345,736	305	117-515
Cardiovascular admissions	519,621	105	14-169	51,833	46	15-85	60,522	56	8-104
Arrhythmia	72,198	15	1-38	7,456	2	0-9	7,577	2	0-11
Cardiac Failure	71,352	14	1-39	6,739	1	0-8	9,530	2	0-9
Cerebrovascular Disease	89,819	18	2-36	9,563	2	0-9	11,205	2	0-9
Ischemic Heart Disease	180,852	37	5-66	18,824	4	0-15	24,369	5	0-19
Respiratory admissions	483,028	98	29-215	35,796	32	6-83	46,394	41	11-93
Asthma	76,668	21	1-87	4,823	1	0-10	5,704	2	0-9
COPD†	89,030	18	2-55	9,141	2	0-11	10,021	2	0-11
Pneumonia and Acute Bronchitis	142,634	29	1-87	10,530	2	0-12	14,607	3	0-16

*N – number of observations; * - Total number of days in excess of PM10 and/or PM2.5 = 58; † - Chronic Obstructive Pulmonary Disease*

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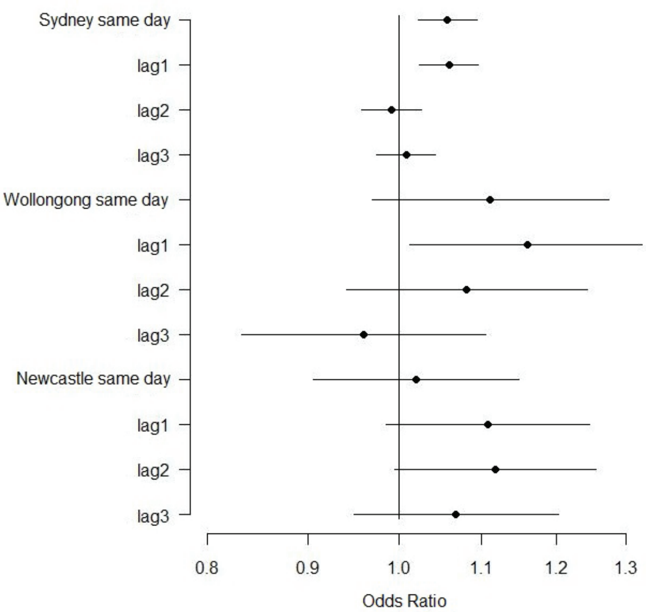
Figure 2: Associations between respiratory hospital admissions and pollution events from bushfire smoke for the same day and at lags of 1-3 days in Sydney, Wollongong and Newcastle, 1994-2007.



with specific diagnoses are listed in Table 2. In Sydney, associations with respiratory outcomes of similar magnitude were generally observed on the day of a smoke event and the day after a smoke event. The same day increase was 5% (OR=1.05, 95%CI=1.02-1.09) for all respiratory admissions, 12% (OR=1.12, 95%CI=1.05-1.19) for asthma admissions, and 13% (OR=1.13, 95%CI=1.05-1.22) for COPD admissions. In the other cities with much smaller populations, associations with all respiratory admissions were more variable and tended to be greatest on the day after the smoke event. In the smaller cities, there was a trend towards a lagged association with pneumonia and bronchitis, which was statistically significant in Newcastle (Figure 2). Associations with specific respiratory diagnoses, although they tended to be positive, were less consistent and lacked precision (Table 2).

Smoke events were not associated with cardiovascular hospital admissions (data not shown) nor any subgroup of cardiovascular disease in any city (Table 3). None of these results were sensitive to the inclusion of Ozone, a pollutant known to have potential health impacts.

Discussion

We found that smoke events were associated with increased hospital admissions for respiratory but not cardiovascular diseases. Our findings are consistent with previous work reporting associations with respiratory outcomes, particularly chronic lower respiratory conditions such as asthma and COPD.[20] We found similar results in all three cities, although the results for the two smaller cities were less precise than those for Sydney. Our findings highlight the difficulty of studying population level impacts of exposure to landscape fire smoke. Despite a long time series, the population centres of fewer than 500,000 people did not provide enough power

Table 2: Associations between respiratory hospital admissions and severe pollution events due to bushfire smoke, for the same day (lag 0) and lags of 1 to 3 days, in greater Sydney 1994-2007.

Outcome	City	Lag	Odds ratio	95% Confidence Interval (Lower,	Upper)
Asthma					
	Sydney	0	1.12	(1.05,	1.19)
		1	1.10	(1.04,	1.17)
		2	1.12	(1.05,	1.19)
		3	1.06	(0.99,	1.12)
	Newcastle	0	0.97	(0.75,	1.26)
		1	1.02	(0.80,	1.29)
		2	1.06	(0.84,	1.35)
		3	1.10	(0.88,	1.37)
	Wollongong	0	1.03	(0.80,	1.33)
		1	1.04	(0.82,	1.33)
		2	1.07	(0.83,	1.38)
		3	1.06	(0.83,	1.34)
Chronic Obstructive Pulmonary Disease					
	Sydney	0	1.13	(1.05,	1.22)
		1	1.12	(1.04,	1.21)
		2	1.02	(0.95,	1.10)
		3	1.05	(0.97,	1.13)
	Newcastle	0	1.18	(0.94,	1.48)
		1	1.20	(0.96,	1.51)
		2	1.09	(0.86,	1.37)
		3	1.35	(1.08,	1.69)
	Wollongong	0	1.25	(0.98,	1.60)
		1	1.09	(0.84,	1.40)
		2	1.31	(1.02,	1.67)
		3	1.01	(0.78,	1.29)
Pneumonia and Acute Bronchitis					
	Sydney	0	1.05	(0.98,	1.12)
		1	1.05	(0.98,	1.12)
		2	0.98	(0.92,	1.05)
		3	1.00	(0.94,	1.07)
	Newcastle	0	1.04	(0.84,	1.27)
		1	1.24	(1.01,	1.52)
		2	1.26	(1.03,	1.55)
		3	1.02	(0.82,	1.26)
	Wollongong	0	1.07	(0.81,	1.40)
		1	1.28	(0.97,	1.68)
		2	1.04	(0.78,	1.40)
		3	1.07	(0.80,	1.42)

Model covariates: (1) same-day average temperature (ºC) modelled as a non-linear response with 3 degrees of freedom; (2) same-day average humidity (as dew point) (ºC) modelled as a non-linear response with 3 degrees of freedom; (3) average temperature (ºC) for the previous 3 days (lags1-3) modelled as a non-linear response with 3 degrees of freedom; (4) average dew point for the previous 3 days (lags1-3) modelled as a non-linear response with 3 degrees of freedom; (5) influenza epidemics, a binary variable coded as 1 if influenza hospital admission rates were < 90th percentile and otherwise 0; and (6) public holidays, a binary variable coded as 1 or 0.

Additionally for asthma: school holidays were also modelled as a binary variable (1,0).

Bold p≤0.05

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Table 3: Associations between cardiovascular hospital admissions and severe pollution events due to bushfire smoke, for the same day (lag 0) and lags of 1 to 3 days, in three NSW cities 1994-2007.

Outcome	City	Lag	Odds ratio	95% Confidence Interval Lower	Upper
Arrhythmia					
	Sydney	0	0.95	0.88	1.03
		1	0.93	0.86	1.01
		2	0.96	0.88	1.04
		3	0.95	0.88	1.03
	Newcastle	0	1.11	0.84	1.47
		1	0.91	0.68	1.21
		2	0.86	0.64	1.14
		3	1.06	0.79	1.41
	Wollongong	0	0.99	0.92	1.07
		1	1.01	0.94	1.09
		2	0.97	0.90	1.04
		3	1.02	0.95	1.10
Cardiac Failure					
	Sydney	0	1.01	0.93	1.11
		1	0.92	0.84	1.00
		2	0.98	0.90	1.07
		3	1.05	0.96	1.14
	Newcastle	0	1.03	0.78	1.36
		1	1.08	0.82	1.42
		2	1.06	0.81	1.39
		3	1.06	0.81	1.39
	Wollongong	0	0.79	0.57	1.09
		1	0.98	0.70	1.35
		2	1.01	0.72	1.42
		3	0.86	0.62	1.20
Cerebrovascular Disease					
	Sydney	0	0.99	0.92	1.07
		1	1.01	0.94	1.09
		2	0.97	0.90	1.04
		3	1.02	0.95	1.10
	Newcastle	0	0.93	0.74	1.17
		1	0.96	0.77	1.21
		2	0.90	0.71	1.14
		3	1.02	0.82	1.28
	Wollongong	0	0.92	0.71	1.20
		1	0.99	0.77	1.27
		2	1.09	0.84	1.40
		3	1.08	0.84	1.40
Ischemic Heart Disease					
	Sydney	0	0.99	0.94	1.05
		1	1.02	0.97	1.08
		2	1.03	0.98	1.08
		3	0.98	0.93	1.03
	Newcastle	0	1.02	0.87	1.19
		1	0.93	0.79	1.10
		2	0.95	0.81	1.12
		3	1.02	0.87	1.20
	Wollongong	0	0.97	0.81	1.16
		1	0.92	0.76	1.10
		2	1.12	0.94	1.34
		3	0.97	0.81	1.17

Table 3 cont. : Associations between cardiovascular hospital admissions and severe pollution events due to bushfire smoke, for the same day (lag 0) and lags of 1 to 3 days, in three NSW cities 1994-2007.

Model covariates: (1) same-day average temperature (ºC) modelled as a non-linear response with 3 degrees of freedom; (2) same-day average humidity (as dew point) (ºC) modelled as a non-linear response with 3 degrees of freedom; (3) average temperature (ºC) for the previous 3 days (lags1-3) modelled as a non-linear response with 3 degrees of freedom; (4) average dew point for the previous 3 days (lags1-3) modelled as a non-linear response with 3 degrees of freedom; (5) influenza epidemics, a binary variable coded as 1 if influenza hospital admission rates were < 90th percentile and otherwise 0; and (6) public holidays, a binary variable coded as 1 or 0.

Additionally for asthma: school holidays were also modelled as a binary variable (1,0).

Bold p≤0.05

to reliably detect associations with well-established respiratory outcomes when compared with Sydney, which has a population of approximately 4 million. We did not find any association with cardiovascular admissions, even in the large population centre of Sydney. One explanation is that there is truly no association or it could reflect the heterogeneity of the underlying causes of each outcome we examined. However, several lines of evidence suggest that adverse cardiovascular outcomes from exposure to vegetation fire smoke are probable. For example: (1) Acute exposures to elevations in urban PM have been associated with cardiovascular morbidity and mortality[36] and toxicological studies suggest that smoke particles elicit similar effects to those of urban particles;[37,38] (2) Associations with bushfire smoke and out-of-hospital cardiac arrests has been documented;[20] (3) Cardiovascular admissions have been associated with PM in cities in which the PM is largely derived from biomass combustion;[39,40] and (4) An intervention to reduce levels of indoor wood smoke showed significant improvements in risk factors for cardiovascular disease.[41]

However, despite this fragmentary evidence, just a handful of epidemiological studies examining cardiovascular disease outcomes and fire smoke have been published to date. Most have had either null or inconclusive results[11,14,16,18,22] with the notable exception of a recent study of exposure to peat fire smoke.[21] This could be because health outcomes associated with exposure to fire smoke are challenging to study. The episodic nature of large events makes it difficult to know precisely when and where air quality will be affected and many affected areas do not have routine air quality monitoring programs.[42] Occasionally, large fires affect urban areas where fixed site air quality monitoring networks measure ambient concentrations in conjunction with those from all other sources. However, as discussed above, the peak exposures may be too short-lived to detect all but the most sensitive health outcomes with adequate statistical power.[43] Although we were able to study a large population and relatively large number of vegetation fire

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smoke events, our results generally had wide confidence intervals and lacked precision.

Moreover, some degree of exposure misclassification is inevitable and this would likely have biased our results towards the null. Other possible explanations for our null results for cardiovascular diseases include there being a true difference in the toxicology of vegetation fire smoke compared with background urban air pollution, with a smaller or absent association with cardiovascular diseases. Further, brief exposures of just one or two days as commonly experienced by southern Australian populations could be less hazardous than chronic exposures. Another potential limitation, as with all studies of this type, is the use of fixed site monitors. Our aim was to evaluate the impact of severe city-wide smoke events. However, it is possible that on some occasions smaller smoke plumes were not detected by available monitors and these would not have been classified as severe smoke events.

Bushfires are a major population hazard in Australia and monitoring of PM levels should be available in fire-prone areas, especially in regional and remote areas which are currently unmonitored.

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