Article

Did Fine Particulate Matter from the Summer 2016 Landscape Fires in Tasmania Increase Emergency Ambulance Dispatches? A Case Crossover Analysis

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Abstract: During summer in early 2016, over 70 landscape fires in Tasmania (Australia) caused several severe episodes of fire smoke across the island state. To assess the health impact of the fire smoke, a case crossover analysis was performed, which measured the association between increased concentrations of $PM_{2.5}$ and emergency ambulance dispatches (EAD) from 1 January to 31 March 2016. Control days were matched by latitude and longitude, day of the week and calendar month. Exposure data were obtained from air quality monitoring stations at lag times of 1–48 h and for the 24-h mean on the same day and 1-day lag. Positive associations were observed between an increase of 10 $\mu g/m^3$ in $PM_{2.5}$ and EAD for stroke on the same day (OR 1.10, 95% CI 1.02–1.19) and at 1-day lag (OR 1.10, 95% CI 1.02–1.18). Furthermore, there were non-significant increases in breathing problems (OR 1.04, 95% CI 1.00–1.08) and diabetic problems (OR 1.11, 95% CI 0.99–1.22) at 1-day lag. The EAD for all causes were not increased. These findings will be used for ambulance service planning and public health risk communication in future landscape fire events.

Keywords: particulate matter; landscape fire smoke; emergency ambulance dispatches

1. Introduction

During an unusually hot and dry summer in early 2016, over 70 landscape fires within a 60-day period in Tasmania (Australia) caused severe episodes of fire smoke across the island state. The concentrations of fine particulate matter (microscopic particles with diameters of less than 2.5 micrometers that are suspended in the air; $PM_{2.5}$) were recorded to have an average of up to 500 $\mu g/m^3$ over 24 h, which are far higher than the Australian National Environment Protection Measure (NEPM) Standard of an average 24-h concentration of 25 $\mu g/m^3$. Given the emerging evidence for acute health effects of landscape fire smoke exposure [1], the aim of this study was to investigate if smoke from the Tasmanian landscape fires caused an increase in acute health events.

The health effects of air pollution are predominantly due to $PM_{2.5}$ generated during combustion. These include acute effects, such as asthma exacerbations, myocardial infarction and stroke; and chronic

effects, such as chronic obstructive pulmonary disease (COPD) and lung cancer. Globally, ambient air pollution is the leading environmental cause of death, resulting in 4.2 million deaths each year (7.6% of all deaths) [2]. The major global causes of death that are attributable to ambient air pollution include COPD (27.1%), lung cancer (16.5%), ischemic heart disease (17.1%) and cerebrovascular disease (14.2%) [2]. Among the global burden of disease from ambient air pollution, 340,000 deaths each year have been attributed directly to landscape fire smoke [3].

The previous studies in the setting of landscape fire smoke exposure have shown an increase in acute respiratory conditions [4,5], cardiac arrest [6], cardiovascular mortality [1], non-traumatic emergency department attendances [7] and all-cause mortality [1,8,9], with the elderly and people with pre-existing health conditions being most at risk [5].

Few epidemiological studies have assessed the health effects of air pollution in Australia. One study in Sydney, the largest city in Australia, attributed 2.1% of all deaths to $PM_{2.5}$ and ozone exposure [10]. The Global Burden of Disease Study estimated that ambient air pollution causes 12.7 deaths per 100,000 per year in Australia, including 5.8% of deaths due to COPD, 5.6% of deaths from ischemic heart disease, 3.8% of stroke deaths, 4.4% of lower respiratory infection deaths and 2.5% of lung cancer deaths [11]. Acute health conditions are often recorded in hospital or emergency department settings. However, these data are limited to the location at which the patient is treated and do not provide information on the exposure of cases to $PM_{2.5}$.

Emergency ambulance dispatches (EAD) provide information on acute health events and offer an advantage in the studies of environmental exposures by providing geospatial information for cases. The studies of fire smoke exposure and EAD have shown an increase in cardiovascular events, including out-of-hospital cardiac arrest [6,12–14] and chest pain [15]; respiratory problems [16,17]; diabetic symptoms [18]; syncope/fainting [18] and all non-traumatic causes combined [19,20].

The aim of this study was to determine if the increased concentrations of $PM_{2.5}$ from fire smoke in Tasmania from 1 January to 31 March 2016 were associated with an increase in all-cause, respiratory-related or cardiovascular-related EAD.

2. Materials and Methods

2.1. Setting

Tasmania is an island state in Southern Australia with a land mass of 68,000 km^2 and an estimated resident population of 518,000 in 2016. Tasmania has low concentrations of air pollution during summer with occasional peaks during landscape fires. The NEPM standards are exceeded periodically during winter months in the towns situated in plateaus or valleys due to domestic woodfire emissions. Very little air pollution is generated from industrial sources or traffic [21].

2.2. Study Design

A time-stratified case crossover design was used to measure the association between exposure to $PM_{2.5}$ and EAD. The case crossover design is used in environmental epidemiology to measure the effect of short-term environmental exposures on acute health conditions [22]. In this methodology, the cases also act as their own control. The cases included all individuals who required an EAD during the study period of 1 January to 31 March 2016. The cases were matched to controls according to the day of the week within the same calendar month, which resulted in three or four controls per case.

2.3. Exposure Data

Air pollution data were obtained from the Base Line Air Network of EPA Tasmania (BLANKeT), which is a network of 29 stations established to measure $PM_{2.5}$, along with meteorological variables obtained in the major cities and towns of Tasmania (Figure 1). BLANKeT data provides a good estimate of the population exposure to $PM_{2.5}$ with 95% of the Tasmanian population residing within 20 km of a station [15]. Hourly exposure data of $PM_{2.5}$, air temperature and relative humidity were extracted

from the location (latitude and longitude) of the cases and controls. Exposures between the monitoring stations were estimated using inverse distance weighted (IDW) interpolation of the BLANKeT network data, using the *gstat* packaging in *R* 3.4 [23]. Exposure values were calculated at lag times of 0–48 h prior to the EAD and the 24-h average on the same day (0–24 h prior) and at 1-day lag (24–48 h lag).
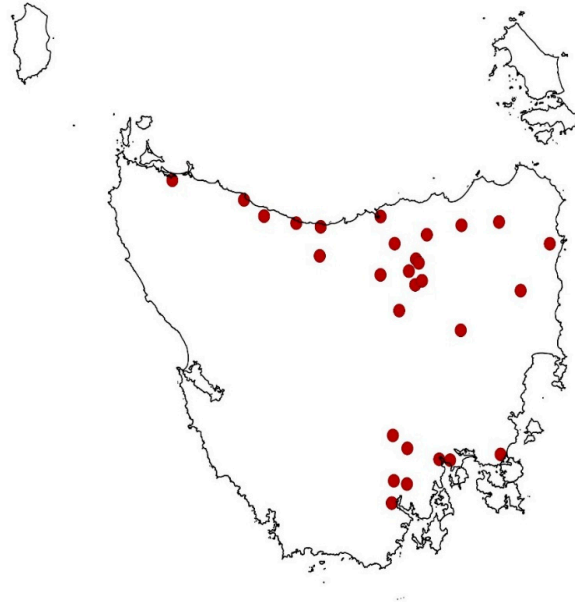

Figure 1. Location of the 21 Base Line Air Network of EPA Tasmania station. Most stations are located in the towns and cities in northern coastal, northeastern and southeastern Tasmania.

2.4. Outcome Data

The primary endpoint of interest was EAD. An EAD was defined as an ambulance dispatched within Tasmania during the study period with information recorded on the dispatch category. EAD data were received from the Tasmanian Ambulance Service Computer Aided Dispatch System (CAD), which uses trained operators to answer emergency calls and dispatch ambulances. CAD is based on the 37 Medical Priority Dispatch System (MPDS) categories. The MPDS categories, which were developed by the International Academies of Ambulance Dispatch, are used throughout Australia and in comparable countries, such as the United Kingdom and the United States [15,24]. The data that were available included the time of the call, location of the incident, age and sex of the case and the MPDS category. Data on inter-facility transfers or motor vehicle crash notifications were not received. Cases that were categorized as "Standby" or that did not have an MPDS category were excluded. All-causes, all non-trauma causes and the MPDS categories that have previously been associated with acute health effects from PM [15,20,25] were analyzed. The categories included: chest pain; heart problems (non-chest pain); stroke; cardiac or respiratory arrest or death; breathing problems; diabetic problems; and unconscious or fainting.

2.5. Statistical Analysis

Population data from the Australian Bureau of Statistics (June 2015) were used to calculate age-specific and regional rates of ambulance usage. Statistical analyses were performed using Stata version 14.2 (Statacorp). A conditional multivariate logistic regression was performed to calculate adjusted odds ratios for the association between an increase of 10 $\mu g/m^3$ in $PM_{2.5}$ and EAD at the time of the event and at 1-h lag periods from 1–47 h prior to the EAD and for the same day (mean of 0–24 h prior to EAD) and at a 1-day lag (mean of 24–48 h prior to EAD). Covariates were the preceding 24-h

mean temperature and 24-h mean relative temperature. Odds ratios were considered to be significant if the 95% confidence interval did not cross 1 and *p*-values were <0.05.

2.6. Ethics

The study was approved by the Tasmanian Health and Medical Human Research Ethics Committee (Reference number H0012974) and the Australian National University Human Research Ethics Committee (Protocol 2016/603).

3. Results

Data were received on 14,512 EAD, of which 36 were excluded (those categorized as "Standby" or did not have an MPDS category). This resulted in a final total of 14,476 cases. The matching of cases to controls according to the day of the week within the month resulted in 48,984 controls. Males and females were equally represented (approximately 50% each). About half (7513; 52%) were from Southern Tasmania, which corresponds to a similar proportion of the Tasmanian population (262,500; 51%) (Table 1).

The average daily frequency of EAD across the study period was 160. Age-specific EAD was highest among the 85 and older age group with an average of 144 EAD per 100,000 population per day during the 91-day study period. This was 16-fold higher than the lowest age group of 5–14 years, which had an average of 9 EAD per 100,000 population per day over the study period. The most common MPDS categories were sick person (1925; 13%), chest pain (non-traumatic) (1788; 12%) and falls (1585; 11%) (Table 2).

Table 1. Demographic features, location and timing of the cases and the Tasmanian population.

	Cases	Tasmania
	Number (%)	**Number (%)**
Total EAD	14,476 (100)	NA
Median Age (Years)	57	42
Sex		
Female	7216 (49.8)	255,728 (49.4)
Male	7174 (49.6)	261,860 (50.6)
Unspecified	86 (0.6)	
Region		
North	3747 (26.0)	143,537 (27.7)
Northwest	3145 (21.8)	111,566 (22.0)
South	7513 (52.1)	262,485 (50.7)

Table 2. Number and proportion of EAD by MPDS category in the period of 1 January to 31 March 2016.

MPDS Category	Number (%)
Sick person	1925 (13.3)
Chest pain (non-traumatic)	1788 (12.4)
Falls	1585 (10.9)
Breathing problems	1292 (8.9)
Unconscious/fainting	961 (6.6)
Abdominal pain/problems	913 (6.3)
Evaluation—interfacility	743 (5.1)
Traffic/transportation accidents	579 (4.0)
Traumatic injury	485 (3.4)
Convulsions/fitting	446 (3.1)
Hemorrhage/lacerations	421 (2.9)
Overdose/poisoning	406 (2.8)
Stroke	365 (2.5)

Table 2. *Cont.*

MPDS Category	Number (%)
Psychiatric/abnormal behavior or suicidal	350 (2.4)
Back pain	333 (2.3)
Heart problems (non-chest pain)	325 (2.2)
Allergies/envenomation	323 (2.2)
Headache	236 (1.6)
Unknown problem	194 (1.3)
Assault/sexual attack	187 (11.3)
Diabetic problems	157 (1.1)
Cardiac or respiratory arrest/death	128 (0.9)
Pregnancy/childbirth/miscarriage	127 (0.9)
Other *	207 (1.5)
Total	14,476

* Choking; burns/explosion; eye problems/injury; bite/attack; Stab/gunshot/penetrating trauma; electrocution/lightning; heat/cold exposure; carbon monoxide/inhalation/hazmat; drowning/diving/scuba accident; inaccessible incident/entrapment.

During the study period, BLANKeT stations in northern Tasmania recorded two distinct periods of $PM_{2.5}$ that were higher than the NEPM standard (Figure 2). These periods corresponded with the landscape fires, of which most were in northern and northwestern Tasmania. Across the three major urban centers in Tasmania, the NEPM Standard was exceeded on two days in Hobart (southern Tasmania), six days in Launceston (northern Tasmania) and seven days in Devonport (northwestern Tasmania).
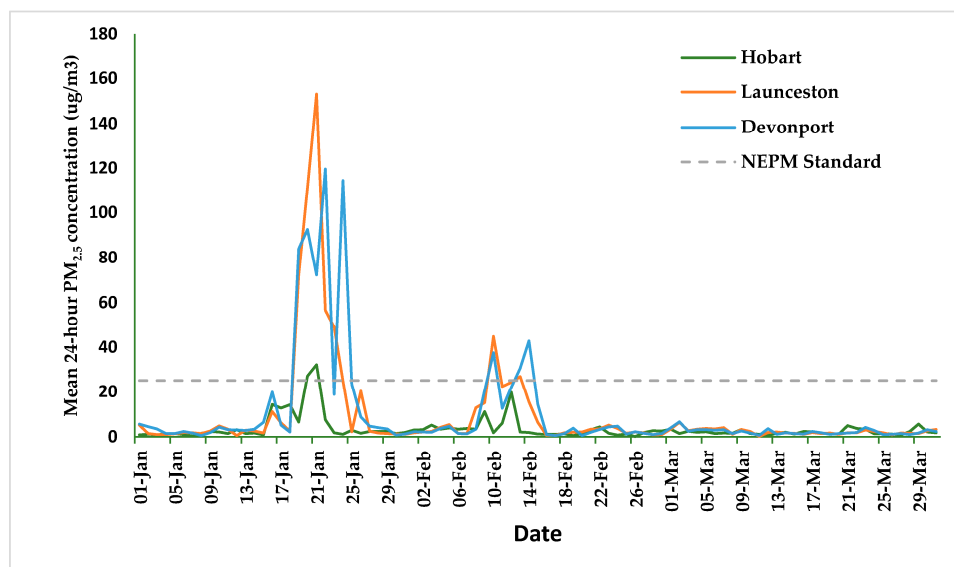

Figure 2. Mean 24-h $PM_{2.5}$ concentration at Devonport (northwest Tasmania), Launceston (northern Tasmania), Hobart (southern Tasmania) and the NEPM 24-h standard in the period of 1 January to 31 March 2016.

In the multivariable analysis, there was no association between an increase of 10 $\mu g/m^3$ in $PM_{2.5}$ and all-cause EAD (same day OR 1.00, 95% CI 0.90–1.01, *p* = 0.88; 1-day lag OR 1.00, 95% CI 0.99–1.02, *p* = 0.59) (Table 3). In the analysis conducted according to selected MPDS dispatch categories, EAD for breathing problems were increased at lag times of 40 and 42 h (Figure 3) with a non-significant positive association at 1-day lag (OR 1.04, 95% CI 1.00–1.08, *p* = 0.06) (Table 3).

EAD for stroke were increased at lag times of 12–16, 19–22, 26–31 and 35 h (Figure 3) with significant positive associations on the same day (OR 1.10, 95% CI 1.02–1.19, *p* = 0.01)) and at 1-day lag

times (OR 1.10, 95% CI 1.02–1.18, *p* = 0.02) (Table 3). Diabetic problems were increased at lag times of 26, 36, 38–40, 45 and 46 h (Figure 3) and there was a non-significant positive association at 1-day lag (OR 1.11, 95% CI 0.99–1.22, *p* = 0.08) (Table 3).

No associations were observed for an increase of 10 μg/m^3 in PM$_{2.5}$ and EAD for cardiac or respiratory arrest, unconscious/fainting, heart problems (non-chest pain) or non-trauma causes (Table 3).

Table 3. Adjusted odds ratio, 95% confidence interval and *p*-value for an increase of 10 μg/m^3 in PM$_{2.5}$ for all causes and selected EAD dispatch categories at same day and 1-day lag.

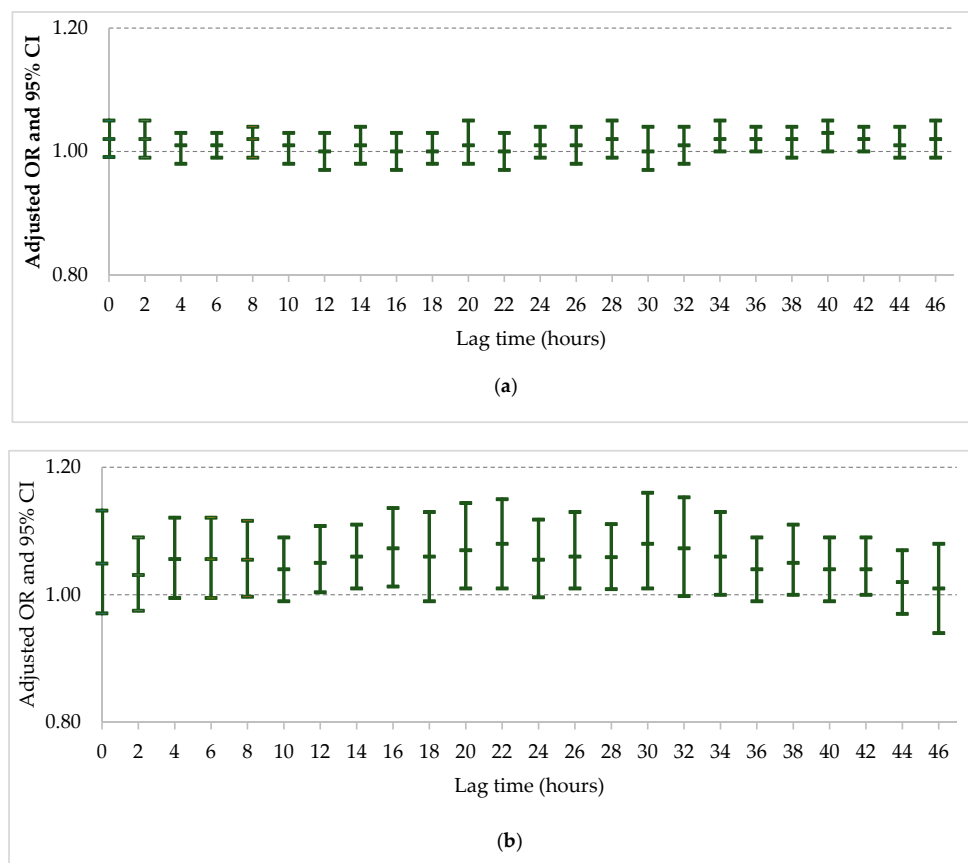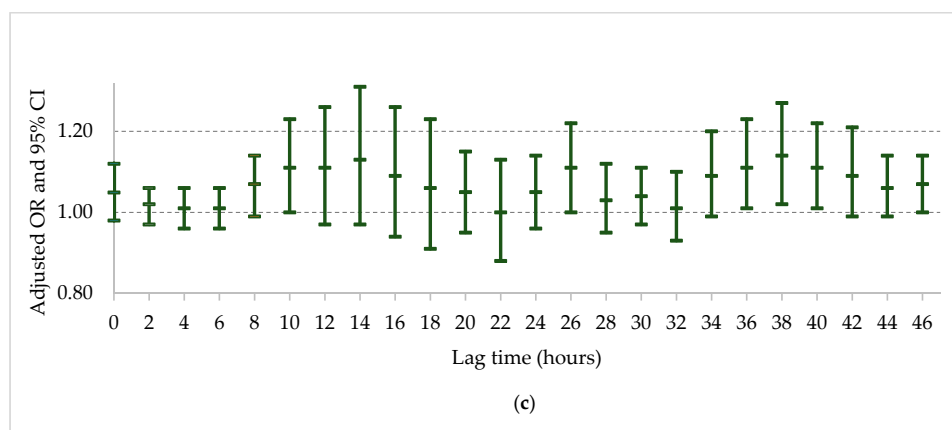| Dispatch Category	Number (%)	Same Day Lag (0–24 h)			1 Day Lag (24–48 h)		
		Odds Ratio	95% CI	*p*-Value	Odds Ratio	95% CI	*p*-Value
All causes	14,476 (100)	1.00	0.99–1.01	0.88	1.00	0.99–1.02	0.59
Non-trauma causes	9219 (63.7)	0.99	0.98–1.01	0.37	1.00	0.98–1.01	0.77
Chest pain (non-traumatic)	1788 (12.4)	0.98	0.95–1.02	0.34	0.99	0.95–1.03	0.49
Breathing problems	1292 (8.9)	1.02	0.98–1.05	0.36	1.04	01.00–1.08	0.06
Unconscious/fainting	961 (6.6)	0.99	0.95–1.03	0.59	0.99	0.95–1.04	0.70
Stroke	**364 (2.5)**	**1.10**	**1.02–1.19**	**0.01**	**1.10**	**1.02–1.18**	**0.02**
Heart problems (non-chest pain)	325 (2.2)	0.99	0.91–1.08	0.84	0.95	0.86–1.06	0.37
Diabetic problems	157 (1.1)	1.07	0.96–1.18	0.21	1.10	0.99–1.22	0.08
Cardiac or respiratory arrest/death	128 (0.9)	0.98	0.83–1.14	0.76	1.00	0.88–1.13	0.98

(a)

(b)

Figure 3. *Cont.*

Figure 3. Adjusted odds ratios and 95% confidence intervals for an increase of 10 μg/m^3 in PM$_{2.5}$ and EAD for (**a**) breathing problems, (**b**) stroke and (**c**) diabetic problems with 0–46 h lag periods.

4. Discussion

In this study, the elevations in PM$_{2.5}$ concentration were associated with significant increases in EAD for the dispatch categories of breathing problems, diabetes and stroke at hourly lag times of 12–46 h. In the analysis over 24-h time periods, an increase of 10 μg/m^3 in PM$_{2.5}$ was associated with a significant increase in EAD for stroke on the same day and at 1-day lag. There were positive associations between an increase of 10 μg/m^3 in PM$_{2.5}$ and the dispatch categories of diabetic problems and breathing problems at 1-day lag although these associations did not reach statistical significance.

The lag time between the exposure to elevated concentrations of PM$_{2.5}$ and the acute health event is consistent with findings in other studies as the health effects of landscape fire smoke are usually delayed by hours up to several days. The physiological mechanisms for the lag time is not completely understood but has been shown to occur as a consequence of the direct irritant and pro-inflammatory effects of PM. These effects trigger metabolic processes, involving cytokine release, oxidative stress, thrombosis and coagulation, which take time to have actual health consequences [26–29].

The finding of increased concentrations of PM$_{2.5}$ not being associated with the dispatch category of chest pain is consistent with some, but not all, other studies [15,18]. This may be because the dispatch category of chest pain is not specific and has been shown to have a wide range of causes, including cardiovascular, gastrointestinal and psychological problems [15]. Only a certain proportion of these are likely to be affected by PM$_{2.5}$ exposure. The finding of increased PM$_{2.5}$ concentration not being associated with cardiac arrest contrasts with the results of several other studies [12–14,26,30]. However, the confidence intervals for this category were wide and therefore, the results should be interpreted with caution.

At a population level, an odds ratio of 1.1 for the dispatch category of stroke at 1-day lag corresponds to an increase of 10% in EAD for stroke for every 10 μg/m^3 increase in PM$_{2.5}$. This would equate to a 100% increase—or doubling—of stroke-related EAD following an increase of 100 μg/m^3 in PM$_{2.5}$. During the study period, 24-h average PM$_{2.5}$ concentrations of up to 300 μg/m^3 were recorded (highest single reading of 2000 μg/m^3) by the BLANKeT network air monitoring stations, which were 30-fold higher than an increase of 10 μg/m^3 in PM$_{2.5}$. These findings could have an impact on ambulance services in Tasmania during severe smoke events. Notwithstanding, the daily average number of EAD for stroke in Tasmania is small and an increase in all-cause EAD was not found, which may have been expected. This suggests that other factors may also contribute. In comparison, a study investigating the impact of smoke-related PM$_{2.5}$ on all-cause mortality and hospitalizations for cardiovascular and respiratory conditions in Sydney (population of approximately 5 million, which is 10-fold greater than Tasmania) estimated that there were 29 cardiovascular hospitalizations,

58 respiratory hospitalizations and 14 deaths due to hazard reduction burns during a single month in 2016 [31].

A major strength of this study was that the $PM_{2.5}$ concentration was matched geospatially with acute health events as measured by EAD. This information is not available in other settings, such as emergency departments or hospitals, and it provides a more precise estimate of the association between exposure to $PM_{2.5}$ and EAD. Another strength of this study was the time-stratified case crossover design where cases act as their own controls, thus removing confounding factors, such as age, sex and comorbidities.

A limitation of the study was that it was undertaken over a relatively short time-frame compared with other environmental epidemiological studies, which resulted in small sample sizes within MPDS categories. Another limitation is that MPDS clinical categories are designed to facilitate rapid ambulance dispatch and do not consistently correspond with a final diagnosis that corresponds with the International Classification of Diseases coding system [15]. Therefore, it cannot be concluded that an increase in the dispatch category of stroke, diabetic problems or breathing problems represents a definite increase in these conditions.

This study did not distinguish between the sources of $PM_{2.5}$ or other air pollutants but it is unlikely that sources other than fire smoke significantly contributed to exposures because of the low population density and limited number of industrial and traffic sources of air pollution in Tasmania. The Tasmanian Environmental Protection Agency report that ambient levels of industrial and vehicle generated pollutants (ozone, nitrogen dioxide, carbon monoxide and sulfur dioxide) are very low, while smoke from landscape fires and domestic wood heaters is the major contributor to air pollution in Tasmania [21]. Screening for these pollutants does take place and concentrations are consistently found to be well below national standards. Fire smoke is a mixture of hundreds of different aerosols and gases. In the context of open burning, PM can be considered as a marker of this mixture and it is recognized as the primary driver of adverse health impacts from fire smoke [1,8].

Finally, a type 1 statistical error may have occurred in the logistic regression at hourly lag times across multiple ambulance dispatch categories. To reduce the type 1 error, the results of each dispatch category analysis were interpreted at hourly lag times in the context of the same day and 1-day lag periods, which has been done in previous studies.

Landscape wildfires, which are the greatest natural disaster risk posed to Tasmania, are expected to increase in frequency and severity due to global warming [32,33]. In addition to the ecological and economic impact of landscape fires, the contribution of landscape fire smoke to the global burden of disease via acute cardiovascular and respiratory events is increasingly being recognized. Preparing for landscape fire events and promoting methods to reduce the harms of smoke exposure, such as through the use of air filters, are important areas for public health officials to focus on into the future.

5. Conclusions

In this study, an increase of 10 $\mu g/m^3$ in $PM_{2.5}$ was positively associated with an increase in EAD for the dispatch categories of stroke, breathing problems and diabetic problems at lag times of 12–47 h. There was also an increase in stroke on the same day and at 1-day lag and non-significant associations between breathing problems and diabetic problems at 1-day lag. There was no increase in all-cause EAD. This study indicates that increased concentrations of $PM_{2.5}$ are associated with an increased demand for selected ambulance dispatch categories. However, the absolute increase is small and may not have an impact on ambulance services unless the $PM_{2.5}$ concentration increase is very high and is sustained for several days within or adjacent to a large population, such as in a capital city in mainland Australia.

These findings, which were obtained from a relatively small and dispersed population during a period of multiple landscape fires, add to the expanding literature on the acute health effects of exposure to landscape firesmoke. In particular, we found that increased concentrations of $PM_{2.5}$ from fire smoke are associated with a range of acute health effects and have a significant impact on public

health at a population level. These findings will be used for ambulance service planning and by public health professionals in developing communication plans to prepare for and respond to landscape fires into the future.

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