

ORIGINAL ARTICLE

Fine particulate matter (PM_{2.5}) exposure during a prolonged wildfire period and emergency department visits for asthma

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ABSTRACT

Background and objective: The 2006–2007 wildfire period was one of the most extensive and long lasting fires in Australian history with high levels of fine particulate matter (PM_{2.5}). Large populations were exposed to smoke for over 2 months. The study aimed to investigate the association between wildfire-related PM_{2.5} exposure and emergency department (ED) visits for asthma.

Methods: A time-stratified case-crossover design was used to investigate associations between daily average PM_{2.5} and ED attendances for asthma from December 2006 to January 2007. ED data were obtained from the Victorian Emergency Minimum Dataset. Smoke dispersion during the wildfire event was modelled using a validated chemical transport model. Exposure data (daily average PM_{2.5}, temperature and relative humidity) were modelled for the study period. Various lag periods were investigated.

Results: There were 2047 ED attendances for asthma during the study period. After adjusting for temperature and relative humidity, an interquartile range increase in PM_{2.5} levels of 8.6 µg/m³ was associated with an increase in ED attendances for asthma by 1.96% (95%CI: 0.02, 3.94) on the day of exposure. Lag periods up to 2 days prior did not show any association. A strong association was observed among women 20 years and older (5.08% 95%CI: 1.76, 8.51).

Conclusions: Wildfire-related PM_{2.5} was associated with increased risk of ED attendance for asthma during the wildfire event. It is important to understand the role of wildfire PM_{2.5} as a trigger for asthma presentations.

Key words: asthma, emergency services/hospitals, fires, particulate matter, smoke.

SUMMARY AT A GLANCE

This time-stratified case cross-over study investigated the association between fine particulate (PM_{2.5}) exposure from wildfire smoke and emergency department (ED) visits for asthma during the 2006–2007 wildfires in Victoria, Australia. Modelled air exposure data were used. Wildfire PM_{2.5} was associated with increased risk of ED attendance for asthma.

Abbreviations: ABS, Australian Bureau of Statistics; CI, confidence interval; COPD, chronic obstructive pulmonary disease; CTM, chemical transport model; ED, emergency department; ICD, International Classification of Diseases; ICD-10AM code, ICD 10th revision; IQR, interquartile range; PM_{2.5}, particulate matter with aerodynamic diameter less than 2.5 µm; TAPM, the air pollution model; VEMD, Victorian Emergency Minimum Dataset.

INTRODUCTION

Asthma remains a significant global health problem affecting around 334 million individuals worldwide and increasing in developing countries.^{1,2} Asthma is characterized by recurrent episodes of airway obstruction associated with bronchial hyperresponsiveness and airway inflammation.³ It is a heterogeneous condition caused by a combination of genetic and environmental factors. Particulate air pollutant is an important environmental factor that can trigger asthma.⁴ Fine particulate matter (PM_{2.5}) (aerodynamic diameter <2.5 µm) pollutant is particularly harmful, as it is easily inhaled deep into the lungs and embeds in the alveoli. It is capable of promoting inflammatory and oxidative stress leading to pulmonary damage and exacerbations of various respiratory conditions including asthma.⁵

PM_{2.5} is a major component of wildfire smoke known to disperse widely and affect large populations far from the fire source.⁶ These fine particles are significantly elevated during wildfire episodes.⁷ Given the increase in frequency, intensity and duration of

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Received 21 April 2015; invited to revise 22 May and 2 July 2015; revised 24 June and 5 July 2015; accepted 7 July 2015 (Associate Editor: Giorgio Piacentini).

wildfires in recent times,⁸ smoke exposure from these fires is a major public health concern.

Studies investigating the association between particulate matter air pollution during wildfires and respiratory health endpoints including asthma have commonly used fixed air quality monitors to estimate pollutant concentrations.^{9–11} These air quality monitors are usually sparsely located and limited in number. The lack of spatially resolved particulate pollutant data restricts most studies to areas surrounding monitoring sites, mostly urban centres that may not be truly representative of the entire population affected during smoke events. Rural/regional areas often experience very high PM_{2.5} levels due to their close vicinity to wildfires; however these areas lack routine monitoring facilities.

The objective of this study was to investigate the association between PM_{2.5} exposure during a prolonged period of wildfire smoke and emergency department (ED) visits for asthma during the 2006–2007 wildfires in Victoria, Australia, utilizing modelled estimates of wildfire-related PM_{2.5} pollutant data.

METHODS

Study period

The study period included 2 months of intense wildfires (1st Dec 2006–31st Jan 2007). The 2006/2007 wildfire period was the longest running collection of fires recorded in Victoria and burnt approximately 1 million hectares of land. The long duration of these wildfires led to smoke dispersion over a wide geographic area. The air quality during this period was substantially diminished with elevated surface concentrations of PM_{2.5} found for most of the wildfire event. The maximum daily PM_{2.5} concentration recorded across large parts of Victoria was greater than 200 µg/m³ (Supplementary Fig. S1). This greatly exceeded the allowable air quality standards for PM_{2.5} as set by the regulatory bodies worldwide (in Australia the advisory standard is 25 µg/m³ for a 24-h period, in the United States it is 35 µg/m³ for a 24-h period). There were around 15–20 days when PM_{2.5} level was greater than 50 µg/m³ (twice the advisory standard).

Health and population outcome data

ED attendance data were obtained from the Victorian Emergency Minimum Dataset (VEMD). VEMD is a comprehensive administrative dataset maintained by the Department of Health and Human Services containing demographic and clinical information on all ED visits to Victorian hospitals.¹² The health data were coded according to the World Health Organization's International Classification of Diseases (ICD). De-identified information included: event date, home postcode, age, gender, principal diagnosis according to ICD-10th revision (ICD-10AM code) and a unique event identifier. While our primary outcome was ED attendances for asthma (ICD10 J45–J46), we also analysed for chronic obstructive pulmonary disease (COPD) (ICD10 J41–J44) as there is substantial diagnostic overlap with asthma, especially in adults.¹³

According to the Australian Bureau of Statistics the estimated resident population of Victoria in 2006 was 5.1 million. Around 3.6 million people resided in metropolitan Melbourne and 1.5 million in rural/regional areas. We included cases whose residential postcodes fell within the state of Victoria.

Air pollution data

Smoke dispersion modelling for the 2006/2007 Victorian wildfire was undertaken using the atmospheric dispersion air pollution model (TAPM) coupled with a chemical transport model (CTM).¹⁴ The modelling technique was developed by the Commonwealth Scientific and Industrial Research Organisation (CSIRO) Oceans and Atmosphere Flagship located in Victoria. CTM is a three dimensional model of emissions, transport, chemical transformation and deposition of gaseous and aerosol species. It predicts regional air quality from a defined inventory of pollutant sources.

For this study TAPM–CTM was configured with a complete emissions inventory which included industrial, domestic, transport and natural sources of aerosols in addition to the smoke from the wildfire event. However, only wildfires contributed significant amounts of PM_{2.5} above the natural background concentration (5–10 µg/m³). Mean daily surface PM_{2.5} concentrations were computed for each 9 km × 9 km cell in an 80 × 80 grid domain centred on Melbourne (Fig. 1). This covered all of Victoria except for small areas at the extreme eastern and western boundaries. Model validation, accuracy and spatial variation have been extensively tested and discussed in detail elsewhere.^{15–17}

Comprehensive observations of smoke composition (including aerosol chemical composition, plume ageing and secondary aerosol formation) were also made at the CSIRO air quality monitoring station in Melbourne during the wildfire event. The size, characterization and chemical composition of smoke aerosol during the wildfire event have been detailed in a recent study.¹⁸ Both the aerosol composition and detailed trajectory analysis confirmed that the only significant source of PM_{2.5} during the pollution events was from the wildfires. PM_{2.5} contribution from other non-wildfire sources (industries, transport, sea salt, windblown dust) during this wildfire period was negligible. Modelled daily average temperature, relative humidity and ground-level PM_{2.5} for the study period were matched by residential postcodes.

Statistical analysis

A time-stratified case-crossover analysis was conducted to investigate associations between wildfire-related PM_{2.5} and respiratory outcomes using conditional logistic regression models. In this design, PM_{2.5} exposure on the day of the health event, for example, asthma presentation (case day), was compared with PM_{2.5} exposure on several non-event days (control days). The control days were selected by time-stratified sampling and matched on day of the week within the same month, and year as the health event.¹⁹ This design automatically controlled for time-

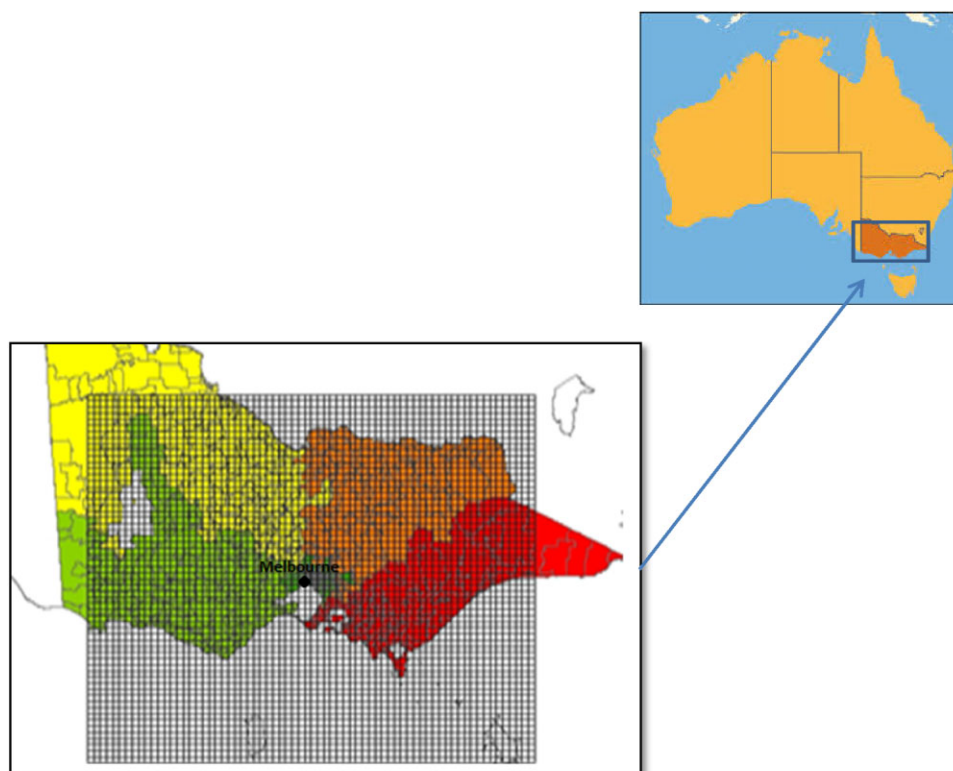


Figure 1 Map of Victoria with grid domain centred on Melbourne (9 km × 9 km cell in an 80 × 80 grid domain).

dependent risk factors such as daily, monthly, seasonal and long-term trends. Moreover since the individuals who experienced the health event also served as their own controls, the time independent factors such as age, gender, smoking, socio-economic status, pre-existing health conditions and other individual risk factors were controlled by this approach.¹⁹

We conducted subgroup analysis by age and gender and investigated lag periods up to 2 days before the event. The analysis of different lag periods was performed individually. The models were adjusted for lagged temperature and relative humidity. Minimal rainfall was recorded during the study period (Bureau of Meteorology, Victoria); we therefore did not include rainfall in the analysis. The overall results are presented as the percentage change in risk of ED attendance for asthma for an interquartile range (IQR) increase in daily average PM_{2.5} concentrations after controlling for temperature and humidity at various lag periods. Analyses were conducted in Stata (Version 12.1, StataCorp, College Station, TX, USA).²⁰

The study was approved by the Monash University Human Research Ethics Committee (Approval number: CF12/0076 – 201200011).

RESULTS

Table 1 provides a description (numbers, percentages) of ED attendances for asthma for the 2-month study period. There were 2047 ED attendances for

Table 1 Characteristics of emergency department visits for asthma in Victoria (1st December 2006–31st January 2007)

Asthma	<i>n</i> = 2047
Gender	
Men	984 (48)
Women	1063 (52)
Age (years)	
≤19	1053 (51)
Men	647 (61)
Women	406 (39)
≥20	994 (49)
Men	337 (34)
Women	657 (66)

Values are *n* (%).

asthma, with individuals 20 years and older accounting for 49% of cases, and women accounting for a majority of cases (66%) compared with men in this age group. The ED attendance data also indicated 927 cases of COPD. Table 2 provides an overview of the distributions of modelled daily average PM_{2.5} concentration, temperature and relative humidity.

An IQR increase in PM_{2.5} levels of 8.6 µg/m³ was associated with an increase in ED attendances for asthma by 1.96% (95%CI: 0.02, 3.94) at lag 0 (same day exposure) after adjusting for temperature and relative

Table 2 Descriptive summary of modelled daily average air exposure data (Dec 2006–Jan 2007)

Variable	Mean	Maximum	Percentiles			IQR
			25%	50%	75%	
PM _{2.5} (µg/m ³)	15.81	294.95	5.58	7.18	14.19	8.60
Temperature (°C)	18.91	32.46	15.10	18.01	22.99	7.89
RH (%)	58.35	96.62	47.77	60.74	68.96	21.18

RH, relative humidity; IQR, interquartile range (e.g. values are calculated for a change in PM_{2.5} levels from the 25th to the 75th percentile).

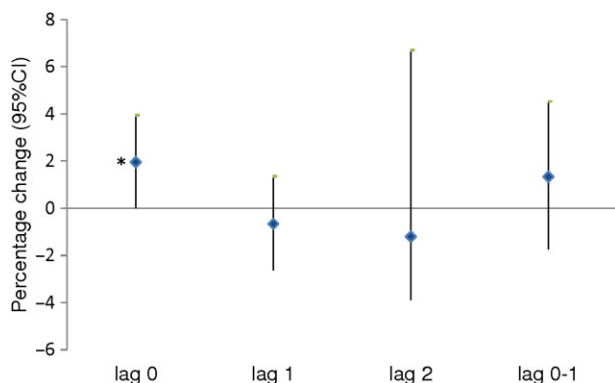


Figure 2 Percentage change (95%CI) in risk of emergency department attendance for asthma for an IQR increase in PM_{2.5} by 8.6 µg/m³ after adjusting for temperature and relative humidity at various lag days (asterisk indicates significant association).

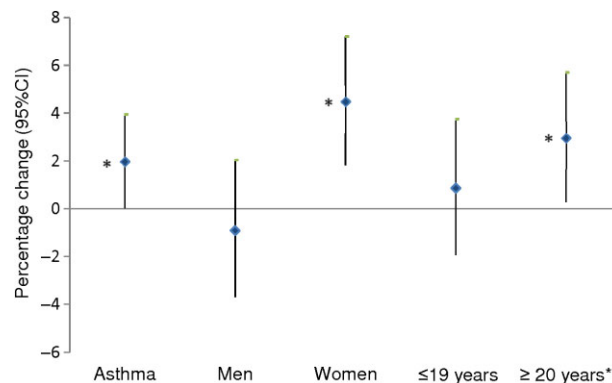


Figure 3 Percentage change (95%CI) in risk of emergency department attendance for asthma (by age and by gender) for an IQR increase in PM_{2.5} by 8.6 µg/m³ after adjusting for temperature and relative humidity at lag 0 (same day exposure). (Asterisks indicate significant association.)

humidity. None of the other lag periods showed any significant association (Fig. 2). No association was found for COPD (−1.71%; 95%CI: −4.73, 1.39) and combined asthma and COPD (0.87%; 95%CI: −0.76, 5.30) at lag 0 or for any other lag periods (data not shown).

The subgroup analyses for asthma presentations demonstrated significant associations for women (4.47%; 95%CI: 1.81, 7.20) and for people 20 years and older (2.95%; 95%CI: 0.28, 5.69%) (Fig. 3).

Figure 4 shows that the association with age group (20 years and older) was confined to women (5.08%; 95%CI: 1.76, 8.51).

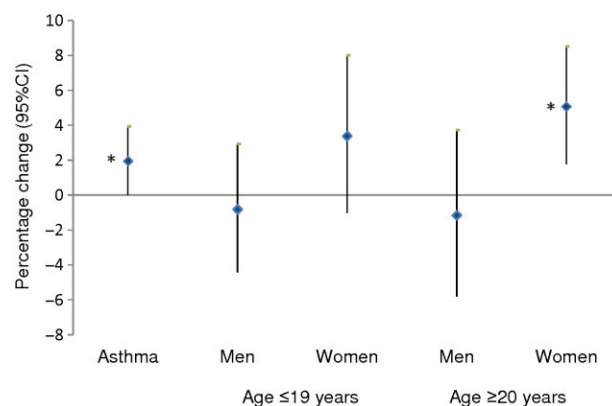


Figure 4 Percentage change (95%CI) in risk of ED attendance for asthma in men and women by age groups for an IQR increase in PM_{2.5} by 8.6 µg/m³ after adjusting for temperature and relative humidity at lag 0 (same day exposure). (Asterisks indicate significant association).

DISCUSSION

This study found a positive association between wildfire-related PM_{2.5} and ED attendances for asthma during the 2006/2007 fire period in Victoria. The increase in risk was strongest in adult women. The association was strong on the same day of exposure to wildfire-related PM_{2.5} concentrations. Other lag periods did not show any association.

The finding of an increase in risk of ED attendances for asthma on the same day of exposure could be due to various postulated mechanistic pathways by which acute exposure to fine particulate matter may lead to asthma. While the detailed mechanism remains to be

elucidated, acute short-term exposure to PM is known to initiate local and systemic responses via the release of pro inflammatory factors (cytokines, transcription factors) from the airway epithelial cells. This response follows from oxidative stress, which causes depletion of anti-oxidants and may cause pulmonary

tissue damage and exacerbations of asthma.^{5,21} Apart from pathophysiological mechanisms, certain non-biological effects such as social factors, economic factors and environmental conditions could determine use of health care services during extreme episodes.²²

In relation to our finding that adult women were at increased risk for asthma-related ED attendance, there is another study²³ that showed women 20 years and older to be at higher risk of asthma due to PM_{2.5} exposure from wildfire smoke episodes. However this study reported an increase in risk for hospital-related admissions (reflecting more severe exacerbations of asthma)²³ unlike our study where we reported an increase in risk for ED-related attendances (reflecting moderate or poorly controlled asthma).

This is an important consideration as all levels of asthma presentations (milder symptoms to severe attacks) are captured during ED visits, unlike hospital admissions where typically only severe cases are admitted. Another study²⁴ also reported adult women to be at increased risk of asthma-related ED attendances, although this study examined exposures to peat bog fire smoke (different chemical composition to wildfire emissions).

Possible reasons for a stronger association in women might include inherent structural differences between the genders (lung size and growth), differences in deposition of fine particles, differences in airway hyperresponsiveness or the potential effect of female hormones on airway cells.^{25–27} Younger males have smaller airways relative to their lung volume than females, thus asthma is more prevalent in boys than girls.²⁶ Other possibilities may be due to differences in perception of symptoms and healthcare usage for asthma, socio-cultural factors or gender differences in indoor/outdoor exposures.^{27,28}

Epidemiological studies have consistently reported positive associations between urban PM_{2.5} exposures (traffic emissions, industrial emissions) and respiratory morbidity.^{29–37} However the difference in toxicity between wildfire PM_{2.5} and urban PM_{2.5} is not yet known. It has been hypothesized that variation in the chemical composition of PM_{2.5} derived from different sources^{7,38} could be responsible for differences in toxicity. Wegesser *et al.*³⁹ compared particulates derived from wildfires and from background urban sources and demonstrated greater pulmonary toxicity of wildfire smoke particulates in an animal model. Such studies are limited and current knowledge does not allow quantification of the health effects of particulate composition from different sources.^{40,41} The difference in biological response could also be explained due to variation in the duration and intensity of exposure to particulate pollutants. Unlike PM_{2.5} derived from urban pollution, wildfire-related PM_{2.5} can reach extreme concentrations with variations in the duration of exposure to smoke events.^{6,38} While certain highly polluted megacities in the world can record particulate concentrations⁴² as high as those observed during wildfire events, the duration and frequency of exposure to elevated PM level citywide is relatively constant. These variations in pollutant exposure make any direct comparison difficult to interpret.

The main strength of our study was the use of spatially resolved modelled air exposure data from wider geographical areas including areas with no monitoring facilities. Monitored data do not adequately represent the smoke impacts in areas most affected by wildfires (rural/regional areas) thus limiting the ability to detect associations that might be present.⁴³ Furthermore unlike most other wildfires that are short lived (lasts for few days) and sporadic in nature, the 2006/2007 wildfires were widespread and lasted for a long period of time. Thus large populations were exposed to PM_{2.5} levels for a long duration during this wildfire event. This allowed analysis of a larger sample size thereby increasing the statistical power of our study.

A further strength was that we utilized a comprehensive state-wide health dataset (VEMD) to obtain information relevant to the aims of the study. Access to population-based data reduced the risk of selection bias and strengthened case ascertainment. In adults, while physicians are able to distinguish between asthma and COPD, some diagnostic overlap does occur.^{13,44} To evaluate the extent that such overlap affects our presented estimates, we investigated the association between wildfire-related PM_{2.5} and ED presentations for COPD and combined asthma and COPD conditions. We found no significant associations suggesting our presented estimates for asthma attendances to be true and not due to methodological artefact.

One limitation of our study was the lack of information on personal risk factors, such as socio-economic status, smoking, obesity and underlying health conditions. However the case cross-over design controls for such confounding factors as individuals serve as their own controls.¹⁹ Nonetheless, information on these confounding variables would be important for formulating targeted health messages and interventions for at-risk groups of people. We also lacked data on indoor PM_{2.5} concentrations during wildfire episodes although research has shown near equal indoor–outdoor concentration ratio of particulates during wildfire smoke events.^{45,46} We were also unable to account for repeated respiratory presentations by the same individual since we obtained only de-identified ED data.

We also acknowledge that we only investigated the impact of wildfire PM_{2.5} and did not include other criteria pollutants (e.g. ozone, nitrogen dioxide, sulphur dioxide) in our study. Therefore we cannot infer direct causality between wildfire PM_{2.5} and asthma attendance. However during the 2006/2007 wildfire event PM_{2.5} was the pollutant most significantly elevated and levels exceeded the regulatory air quality standards. Other pollutants were also reported to be elevated but not to the same magnitude as seen for PM_{2.5} levels.^{18,46,47}

Robust evidence-based research is required to fill the knowledge gaps which currently exist in this area of public health importance. Novel air exposure modelling techniques to improve spatial coverage, forecasting tools to improve predictions of air quality, health impact assessment of at-risk groups (women, children, elderly), targeted health advice and

improvement of emergency health services are areas of need. Pathophysiological pathways which trigger acute asthma due to fine PM exposure still remain uncertain and need further research. Improving understanding of these key priority areas is needed so that effective and timely adaptive strategies can be developed and implemented to mitigate respiratory health risks due to wildfire smoke exposure in the community.

Overall this study showed a strong association between exposure to wildfire PM_{2.5} pollutant and ED attendances for asthma during the 2006–2007 wildfire period in Victoria. Wildfires are likely to become an increasing problem worldwide in the context of warming climate. This will increase environmental triggers of asthma and other respiratory conditions, resulting in more severe and frequent disease exacerbations. Given that the burden of asthma remains high, it is important to understand the role of wildfire PM_{2.5} as a trigger for acute asthma presentations. This may have important implications to reduce the burden of disease in the community.

Acknowledgement

This research was funded by the Bushfire Cooperative Research Centre now a part of Bushfire & Natural Hazards Cooperative Research Centre, Melbourne, Victoria, Australia.

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Supplementary Information

Additional Supplementary Information can be accessed via the *html* version of this article at the publisher's website.

Figure S1 Maximum PM_{2.5} concentration (24 h) across Victoria during 2006–2007 wildfires.

SUPPLEMENTARY INFORMATION

Fine particulate matter (PM_{2.5}) exposure during prolonged wildfire period and Emergency

Department visits for asthma.

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Figure S1: Maximum PM_{2.5} concentration (24 hour) across Victoria during 2006-2007 wildfires.

