Exposure to fine particulate matter (PM2.5) during landscape fire events and the risk of cardiorespiratory emergency department attendances: a time-series study in Perth, Western Australia

Adeleh Shirangi ,[1,2,3] Ting Lin,[1] Ivana Iva'nova',[4] Grace Yun,[1] Grant J Williamson,[5] Peter Franklin,[6] Le Jian,[1] Rowena Burch,[1] Ashraf Dewan,[4] Bradley Santos,[7] Nathan Eaton,[8] Jianguo Xiao[1]

For numbered affiliations see end of article.

Correspondence to
Dr Adeleh Shirangi, Epidemiology Branch, Department of Health, Government of Western Australia, East Perth, Western Australia, Australia; shirangi.adeleh@curtin.edu.au; a.shirangi@murdoch.edu.au

Received 21 October 2021
Accepted 8 June 2022

To cite: Shirangi A, Lin T, Iva'nova' I, *et al*. *J Epidemiol Community Health* Epub ahead of print: [*please include* Day Month Year]. doi:10.1136/jech-2021-218229

ABSTRACT

Background Landscape fires (LFs) are the main source of elevated particulate matter ($PM_{2.5}$) in Australian cities and towns. This study examined the associations between daily exposure to fine $PM_{2.5}$ during LF events and daily emergency department attendances (EDA) for all causes, respiratory and cardiovascular outcomes.

Methods Daily $PM_{2.5}$ was estimated using a model that included $PM_{2.5}$ measurements on the previous day, remotely sensed aerosols and fires, hand-drawn tracing of smoke plumes from satellite images, fire danger ratings and the atmosphere venting index. Daily $PM_{2.5}$ was then categorised as high (≥99th percentile), medium (96th–98th percentile) and low (≤95th percentile). Daily EDA for all-cause and cardiorespiratory conditions were obtained from the Western Australian Emergency Department Data Collection. We used population-based cohort time-series multivariate regressions with 95% CIs to assess modelled daily $PM_{2.5}$ and EDA associations from 2015 to 2017. We estimated the lag-specific associations and cumulative risk ratios (RR) at lags of 0–3 days, adjusted for sociodemographic factors, weather and time.

Results All-cause EDA and overall cardiovascular presentations increased on all lagged days and up to 5% (RR 1.05, 95% CI 1.03 to 1.06) and 7% (RR 1.07, 95% CI 1.01 to 1.12), respectively, at the high level. High-level exposure was also associated with increased acute lower respiratory tract infections at 1 (RR 1.19, 95% CI 1.10 to 1.29) and 3 (RR 1.17, 95% CI 1.10 to 1.23) days lags and transient ischaemic attacks at 1 day (RR 1.25, 95% CI 1.02 to 1.53) and 2 (RR 1.20, 95% CI 1.01 to 1.42) days lag.

Conclusions Exposure to $PM_{2.5}$ concentrations during LFs was associated with an increased risk of all-cause EDA, overall EDA cardiovascular diseases, acute respiratory tract infections and transient ischaemic attacks.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Research into the health effects of excessive levels of PM2.5 during severe landscape fires (LFs) has consistently found evidence for respiratory morbidity but has been limited and inconsistent for cardiovascular disease. Few studies assessed the effect of exposure to PM2.5 concentrations during LF events on cardiorespiratory emergency department attendances (EDA).

WHAT THIS STUDY ADDS

⇒ This study is the first to find a significant dose–response relationship between exposure to elevated PM2.5 during LF events, and an increased risk of EDA due to transient ischaemic attacks. The largest effects occurred 1 and 2 days after exposure. We also found a dose–response relationship in most lag-specific effect estimates, which adds to the limited evidence in the studies on the acute health effects from daily exposure to PM2.5 during LF events.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study has implications for practical public health response actions for the appropriate preparedness prior to LF events and for health service and clinician alerts during LF events.

INTRODUCTION

Landscape fires (LFs), which include wildfires (WFs) and prescribed/planned burns (PBs), are an important source of short-term increases in particulate air pollution.[1] LFs, particularly WFs, are a growing concern globally as they are expected to increase in frequency and intensity due to climate change.[2 3] Studies from Australian WF events reported that $PM_{2.5}$ was the pollutant most significantly elevated during LFs, and its levels exceeded the regulatory air quality standards compared with other contaminants.[4 5] A study on biomass burning emissions over northern Australia reported that 87% of PM_{10} due to WF consists of $PM_{2.5}$.[6] PM in the air, particularly $PM_{2.5}$, is of particular concern as it can penetrate deep into the alveolar region of the lung and enter the bloodstream, leading to a variety of manifestations of cardiorespiratory outcomes.[7 8] An analysis of the global burden of disease due to outdoor air pollution estimated that $PM_{2.5}$ causes about 3% of mortality from cardiopulmonary disease.[9]

Short-term increases in particulate air pollutants have been associated with many adverse health effects, including exacerbations of acute cardiorespiratory morbidity.[2 8 10–12] Research into the health effects of excessive levels of PM during severe LFs has consistently found evidence for respiratory morbidity but has been limited and inconsistent for cardiovascular disease (CVD).[2 4 11 13–24] The discrepancies between LF-related and urban $PM_{2.5}$ could be due to differences in the constituents of the PM, limited power due to the small population size to detect the effect of LFs or difficulties in measuring exposure to LF smoke.[25]

Few studies assessed the effect of elevated $PM_{2.5}$ during LF events on cardiorespiratory outcomes using routinely collected administrated data.[8 12 14] In addition, the dose–response relationships between the effect of $PM_{2.5}$ during LF events and adverse health effects of CVD are rarely investigated. So far, most epidemiological studies on LFs have focused on estimating the population exposure to smoke-related $PM_{2.5}$ with a prediction of grid resolution of at least 5 km × 5 km over the locations of interest.[26] Thus, there is the possibility of uncaptured exposure variability within the prediction grid cells. There is also room for improvement in estimating the values of LF indicators more precisely using advanced methodologies such as inverse distance weighted (IDW) modelling. Hence, to detect changes in health outcomes associated with LF smoke, there is a need to address these questions through the improved exposure assessment on previously exposure modelling.

This study aimed to expand the understanding of acute exposure to $PM_{2.5}$ concentrations that are predominately elevated during LF events to a range of cardiorespiratory health effects. We used a recently developed empirical exposure model with some modifications to improve estimating $PM_{2.5}$ concentrations exposure levels during LF events on emergency department attendances (EDA) for a range of respiratory and cardiovascular outcomes. We also assessed age, sex and socioeconomic status as modifiers of risk for cardiorespiratory effects following acute exposure to LFs.

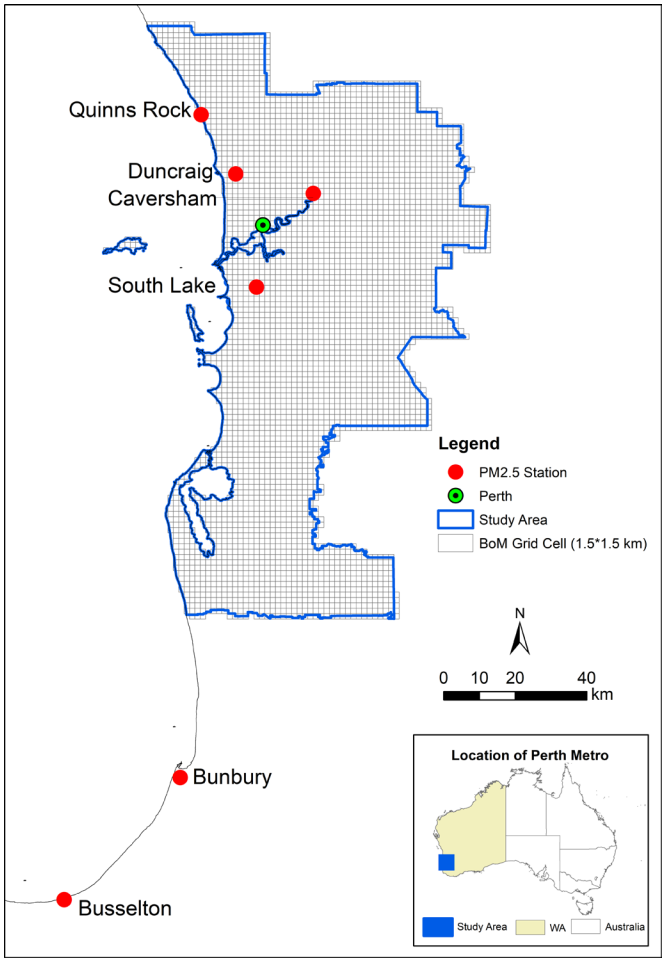

Figure 1 Study area and model estimate base grid cells. Inset map showing the study area and the state of Western Australia with respect to Australia. WA, Western Australia.

MATERIALS AND METHODS

Study design, setting, period and participants

A population-based cohort time series design was used to estimate the association between exposures to daily $PM_{2.5}$ exposure during LF events and daily variations in health outcome rates in Perth, Western Australia (WA). Population data by 5-year age group, gender and Statistical Area 2 (SA2) for the study areas were sourced from the Australian Bureau of Statistics.[27] SA2 is a medium-sized geographical area in the Australian Statistical Geography Standard[28] and represents a community that interacts together socially and economically. SA2s generally have a population range of 3000–25 000 persons, with an average population of 10 000 persons.

The monthly populations were computed using a linear interpolation of mid-year populations, and such populations were then applied to all days in the month. The daily estimates then served as the estimated populations at risk for calculating rates in the analysis. The study participants were individuals living in SA2. The analysis unit was SA2 with different population sizes.

The population cohort was assembled from all residents (N=1 543 222) who attended WA emergency departments over the study period. A daily time series by gender, age group (0–14, 15–60 and 60+ years) and SA2 for the Perth metropolitan area from 1 July 2015 to 31 December 2017 was constructed. The time-series database was then joined to environmental exposures, including the modelled $PM_{2.5}$, seasonal and public holiday factors, and health outcomes of interest.

Air quality data

Air quality data were sourced from Western Australian Department of Water and Environmental Regulation for the whole study period (1 July 2015 to 31 December 2017). The data for particulate matter with aerodynamic diameter ≤2.5 micrometres ($PM_{2.5}$) pollutants was available for all six stations, as shown in figure 1. Although two air quality stations (Bunbury and Busselton) were located outside the study area, their data were still used in spatial interpolation to ensure robust estimates at unsampled locations. The average of the top 8 hourly values of $PM_{2.5}$ for a day was calculated and represented as a daily measure. We selected this approach for two main reasons: (1) the distribution of $PM_{2.5}$ was skewed, and most values were minimal; (2) the top eight readings usually occurred during the working 8 hours of a day when people were exposed to air pollution, and during other time in a day, people would have much less chance to be exposed.Almost all variables in the model had zero or 0.1% missing. However, some air quality data had a low percentage of missing values (5.17%), such missing values were imputed by using the multiple imputation procedure in SAS enterprise Guide V.5.1 (SAS Institute). This low percentage of missing patterns was probably mainly due to measuring periodic equipment

maintenance. We also examined the patterns of missing values that followed a missing at the random way.

Exposure assessment

$PM_{2.5}$ concentrations were used as a proxy measure for exposure to LF smoke. We hypothesised that PM2.5 concentrations are consistently elevated during LFs smoke events at locations near and far from the fire. To estimate $PM_{2.5}$ concentrations over the study area, specifically $PM_{2.5}$ associated with smoke from LFs, we adopted an empirical exposure model previously developed and described elsewhere,[11 25 26] with some modifications to improve the modelling. Briefly, we used various observation data to evaluate the elevated $PM_{2.5}$ concentrations during LF events. We built an empirical exposure model to estimate $PM_{2.5}$ concentrations in the areas without the air quality monitoring stations. The model inputs included $PM_{2.5}$ measurements from monitoring air quality stations, $PM_{2.5}$lag1 ($PM_{2.5}$ daily average from the previous day's monitor measurements), remotely sensed fire radiative power (FRP),[29] aerosol optical depth,[30] smoke plume mask (SPM),[31] which is similar with the well-known hazard mapping system data product over North America,[32] fire danger rating and a Venting Index that indicated pollutant dispersion potential. All image processing and digitisation was completed using ESRI ArcGIS (V.10.7.1).

Similar to a previously developed model,[26] we used a training dataset comprising grid cells containing air quality stations to generate prediction models, and assessed the goodness of fit of each model by calculating the root mean square error (a measure of the difference between predicted and observed $PM_{2.5}$values) and Pearson's correlation coefficient (r). However, we used hold-out cross-validation, with an 80/20 train/test split of the whole sample, instead of FRP distribution due to different FRP distribution in this study. A leave one out approach was used to evaluate its performance. The model prediction had a root mean squared error of 3.14 mg/m^3 (table 1). There were positive correlations between the predicted $PM_{2.5}$ and LF predictors, including the SPM from observed values, indicating high $PM_{2.5}$ is primarily due to LFs.

We used a resolution of 1.5 km by 1.5 km for grid cells instead of 5 km by 5 km.[26] As such, we had 3898 grid cells as the base grids for modelling. These grid cells were defined by the Australian Bureau of Meteorology. The study location, model estimate base grid cells and the place of air quality monitoring stations are shown in figure 1. Another modification was the use of IDW modelling, a type of deterministic method for multivariate interpolation with a known scattered set of points throughout the study area to estimate the values of LF indicators,[33] instead of using the nearest monitoring site. IDW assigns more weight to closer known points than distant ones when estimating data at an unknown point. The IDW was conducted using ArcGIS Desktop V.10.7.1. The inverse power used in this study was two. The allocated values to unknown points were calculated with a weighted average of the air quality values available at the known stations.[34] We used the predicted $PM_{2.5}$ concentrations during LF events to examine the adverse health effects on EDA.

Table 1 Multiple linear regression results of LF predictors for PM2.5 (final training model summary)

Predictor**†	Estimate	SE	T value	P value	Partial R^{2**}
PM2.5lag1	0.380	0.040	7.790	<0.001	0.175
AOD	4.404	0.860	5.110	<0.001	0.015
FRP	1.760	0.460	3.790	<0.001	0.031
FDR	0.524	0.080	6.550	<0.001	0.006
SPM	0.990	0.440	2.220	0.026	0.002
VI	0.830	0.050	16.040	<0.001	0.036

N=3660, R square = 0.245, RMSE (root mean square error) = 3.14.
**Importance calculated as the proportion of variance explained attributable to the variable without adjusting for other factors.
†** All predictor measures are at grid-cell level.
AOD, aerosol optimal depth; FDR, fire danger rating; FRP, fire radiative power in Gigawatts; PM2.5lag1, Previous day PM2.5 (mg/m3); SPM, smoke plume mask (whether smoke plume covered the centroid of a grid cell: 0=not covered and 1=covered) ; VI, Venting Index (m^2/s).

Health outcome data

Daily EDA count for total presentations and selected respiratory and CVD were retrieved from the WA Emergency Department Data Collection. We used the major diagnostic block (MDB), major diagnostic category (MDC) and International Classification of Diseases-10th Revision to classify EDA. Respiratory diseases included the following conditions: all respiratory (MDB=3B, MDC=4), asthma (J45–J46), acute lower respiratory tract infections (ALRTI) (J20–J22, J10–J11, J16.8, J17–J18, J12, J15–J16, J1.4, J1.3, A48.1–A48.2, B59), chronic obstructive pulmonary disease (J40–J44, J47) and Croup (J05.0). CVDs include the following conditions: all CVDs (MDB=3A, MDC=5), arrhythmia (I44–I45, I47, I48.0–I48.4, I48.9, I49), angina (I20), stroke (I60–I64), heart failure (I50), acute coronary syndrome (I21–I22) and transient ischaemic attack (TIA) (G45).

Statistical analysis

The values for estimated $PM_{2.5}$ concentrations were categorised into three levels based on their distribution percentiles, that is, low (≤95th percentile, ie, ≤8.73), middle (96th–98th percentile, >8.73 and <12.60) and high level (≥99th percentile, ie, ≥12.60) to assess dose–response relationships. The high level (99th percentile) was chosen as it corresponds closely to Australian air quality standards and enables a clear delineation between background air quality and LFs. The 99th percentile cut-off has also been used in other Australian studies investigating the effect of bush fires on health outcomes.[20 35] LF-related days were defined as those days when the predicated $PM_{2.5}$ was equal or exceeded the 99th percentile of the entire time series.

The associations of estimated $PM_{2.5}$ exposure on health service utilisation was also examined on the same day and cumulative for 1–3 days. We used distributed non-linear lag modelling for modelling lag structures. For instance, cumulative 3-day data were the sum of current day counts and population with subsequent 3 'days' counts. The above classifications captured periods of high smoke days of varying intensity and lag effects relative to the expected $PM_{2.5}$ concentrations in each of 3898 grid cells. We calculated the median of $PM_{2.5}$ values from the gridded cells corresponding to a specific SA2. The database with the predicted $PM_{2.5}$ during LFs at the SA2 level was used for health data analysis. Several independent risk factors were considered potential confounders, including age, sex, SEIFA, dewpoint temperature, woodfire, holiday, weekend and season (table 2). We also investigated interactions with effect modifiers of age, sex and socioeconomic status as a priori (additional risk due to interaction).

Univariate statistical analysis and multivariate Poisson regressions were conducted to assess the health risks associated with elevated $PM_{2.5}$. The Poisson model was applied to aggregated data at the SA2 level. We included all confounding factors in the multivariate regression models as described above. All variables were categorical except humidity, which we used as a continuous

Table 2 Number (N) and percentage (%) of sociodemographic characteristics, environmental exposures and health outcomes for study participants, Perth metropolitan area, WA, July 2015–2017

Health outcomes	N	%
Total no of EDA*	1 543 222	100.00
Total respiratory	114 160	7.40
Asthma	10 196	0.66
Chronic obstructive pulmonary disease	10 902	0.71
Acute lower respiratory tract infections	36 859	2.39
Croup	10 322	0.67
Total cardiovascular	130 466	8.45
Arrhythmia	11 515	0.75
Heart failure	6745	0.44
Acute coronary syndrome	6447	0.42
Angina	6263	0.41
Stroke	2194	0.14
Transient ischaemic attack	3886	0.25
Sociodemographic conditions		
Age		
0–14	376 554	24.40
15–59	798 877	51.77
60+	367 791	23.83
Gender		
Male	768 766	49.82
Female	774 456	50.18
SEIFA**		
Disadvantaged	526 956	34.15
Middle	313 968	20.34
Less disadvantage	702 298	45.51
Smoke-related PM2.5		
No smoke/low (<8.73 µg/m^3)	1 465 782	94.98
Medium (8.73–12.60 µg/m^3)	61 400	3.98
High (≥12.60 µg/m^3)	16 040	1.04
Woodfire		
Infrequent	459 767	29.79
Less frequent	409 684	26.55
Frequent	673 771	43.66
Dewpoint temperature***		
Low (<16.32°C)	1 465 086	94.94
Medium (16.32°C–17.96°C)	46 680	3.02
High (>17.96°C)	31 456	2.04
Relative humidity		
Low (<87.32)	1 497 554	97.04
Medium (87.32–92.25)	30 707	1.99
High (>92.25)	14 961	0.97
Seasonal/time and holiday factors		
Public holiday		
No	1 496 078	96.95
Yes	47 144	3.05
Weekend		
No	1 087 816	70.49
Yes	455 406	29.51
Season		
Summer	353 934	22.93
Autumn	310 212	20.10
Winter	414 500	26.86
Spring	464 576	30.10

*EDA, emergency department attendances;
**SEIFA, Socio-economic indexes for area;
***Dew temperature selected as an indicator of temperature. Dew temperature (in °C)=observed temperature (in °C) – (100- relative humidity (in %)/5. The values for dew point temperature were categorised into three levels based on their distribution percentiles, as shown in the table. The 98th percentile was chosen as it corresponds closely to the 21°C dew point in our database, where it has been used as a guide for human comfort (most people tend to feel uncomfortable when dew points get well into the 60s or 70s.

variable due to collinearity. The goodness-of-fit measure was used to identify the most optimal models. We also conducted a zero-inflated regression to control for the overdispersion issue.

The multivariate models were run on lagged effects of LF related to daily exposure to $PM_{2.5}$ concentrations on the same day (lag0), cumulative 1 day (lag1=lag0+lag1), cumulative 2 days (lag2=lag0+lag1+lag2) and cumulative 3 days (lag3=lag0+lag1+lag2+lag3) following exposure to estimated $PM_{2.5}$. We considered lagged periods of 1, 2 and 3 days, as these periods are the most effects days consistent with previous studies. The lagged effect approach has been applied in similar studies previously.[11 17 18 23 36] The risk ratio (RR) was calculated to assess the difference in health outcome measures associated with $PM_{2.5}$. The model with the greatest RR for the $PM_{2.5}$ exposure was considered the best model where the strong lagged effect of $PM_{2.5}$ was demonstrated. The comparison between different levels for a risk factor was considered statistically significant if the p value was less than 0.05. The statistical analysis was conducted using SAS Enterprise Guide V.5.1.

RESULTS

The total number of EDA during the study period was 1 543 222. The number and percentage of records for each health condition, environmental exposures, sociodemographic factors and time/seasonal factors are presented in table 2. Acute lower respiratory tract infections (ALRTI) (N=36 859) from respiratory outcomes and arrhythmia (N=11 515) from cardiovascular outcomes were the most common cause of EDA.

In the multivariate analysis of total EDA (table 3), an increase of 1% to 5% was observed in all lagged effects with a maximum of 5% significant increase (RR 1.05, 95% CI 1.03 to 1.06), where the $PM_{2.5}$ was at the high level ≥99 percentile) on the same exposed day (lag 0). In an interaction analysis with age, sex and SEIFA (Socio-Economic Indexes for Areas), the total EDA rates increased by 3% to 5% ($p<0.05$) in those aged 60 years and above in all lagged effects. There was also an increased risk of lagged effect within 3 days for those in disadvantaged groups.

As demonstrated in table 4, the rate of asthma increased with the dose–response effect on all lagged days with a maximum cumulative risk of 10% within 1 day at a high level (RR 1.10, 95% CI 0.96 to 1.25).The rate of acute lower respiratory tract infections increased within one and 3 days, with a maximum cumulative risk of 19% within 1 day (RR 1.19, 95% CI 1.10 to 1.29) and 17% within 3 days (RR 1.17, 95% CI 1.10 to 1.23 (table 4 and figure 2). In an interaction analysis with age, sex and SEIFA, the EDA rates on the same day for asthma significantly increased by about 85% (RR 1.85, 95% CI 1.14 to 3.00) in the disadvantaged group. There was also a 20% increased risk (RR 1.20, 95% CI 1.02 to 1.41) of EDA due to acute lower respiratory tract infections in lagged effect within 3 days for disadvantaged groups.

The rate of overall CVD increased by 5% to 7% ($p<0.05$) at high-level $PM_{2.5}$ exposure with dose–response effect at all lagged effects with maximum risk on the same day (RR 1.07, 95% CI 1.01 to 1.12). TIA rate increased by 25% (RR 1.25, 95% CI 1.02 to 1.53) and 20% (RR 1.20, 95% CI 1.01 to 1.42) with dose–response effect within 1 and 3 days, respectively (table 4 and figure 2).In an interaction analysis with age, sex and SEIFA, the EDA rates on all lagged effects for total CVDs increased about 5%–8% ($p<0.05$) in those aged 60 years and above.[37]

Table 3 Multivariate Poisson regressions for the association between emergency department attendance and exposure to estimated PM2.5 for same day (Lag0) and lags of 1–3 days

	Lag 0			Lag 1			Lag 2			Lag 3		
	RR*	95% CI	P value	RR*	95% CI	P value	RR*	95% CI	P value	RR*	95% CI	P value
Environmental Exposures												
Smoke-related $PM_{2.5}$												
≤95 (low)	1			1			1			1		
96–98 (medium)	1.02	1.01 to 1.03	<0.0001	1.02	1.01 to 1.03	<0.0001	1.02	1.01 to 1.02	<0.0001	1.01	1.01 to 1.02	<0.0001
≥99 (high)	1.05	1.03 to 1.06	<0.0001	1.04	1.03 to 1.05	<0.0001	1.03	1.02 to 1.04	<0.0001	1.03	1.03 to 1.04	<0.0001
Dewpoint temperature												
≤95 (low)	1			1			1			1		
96–97 (medium)	1.03	1.02 to 1.04	<0.0001	1.03	1.02 to 1.04	<0.0001	1.02	1.01 to 1.03	<0.0001	1.02	1.01 to 1.02	<0.0001
≥98 (high)	1.03	1.02 to 1.04	<0.0001	1.02	1.01 to 1.03	<0.0001	1.01	one to 1.02	0.0015	1.01	one to 1.01	0.0137
Wood fire												
Infrequent	1			1			1			1		
Less frequent	0.98	0.97 to 0.99	<0.0001	0.98	0.97 to 0.98	<0.0001	0.98	0.97 to 0.98	<0.0001	0.98	0.97 to 0.98	<0.0001
Frequent	1	0.99 to 1.01	0.9911	1	one to 1.01	0.5246	1	one to 1.01	0.9027	1	0.99 to 1	0.5712
Sociodemographic												
Age												
15–59	1			1			1			1		
0–14	0.65	0.65 to 0.65	<0.0001	0.65	0.65 to 0.65	<0.0001	0.65	0.65 to 0.65	<0.0001	0.65	0.65 to 0.65	<0.0001
60+	1.01	1.01 to 1.02	<0.0001	1.01	1.01 to 1.02	<0.0001	1.01	1.01 to 1.02	<0.0001	1.01	1.01 to 1.02	<0.0001
Gender												
Male	1			1			1			1		
Female	1	one to 1.01	0.2278	1	one to 1	0.0847	1	one to 1	0.0333	1	one to 1	0.0119
SEIFA												
Advantaged	1			1			1			1		
Middle	1.19	1.18 to 1.19	<0.0001	1.19	1.18 to 1.19	<0.0001	1.19	1.18 to 1.19	<0.0001	1.19	1.18 to 1.19	<0.0001
Disadvantaged	1.5	1.49 to 1.5	<0.0001	1.5	1.49 to 1.5	<0.0001	1.5	1.49 to 1.5	<0.0001	1.5	1.49 to 1.5	<0.0001
Seasonal factors												
Holiday												
Not holiday	1			1			1			1		
Holiday	1.09	1.08 to 1.1	<0.0001	1.09	1.09 to 1.1	<0.0001	1.07	1.06 to 1.08	<0.0001	1.05	1.05 to 1.06	<0.0001
Weekend												
Weekday	1			1			1			1		
Weekend	1.05	1.04 to 1.05	<0.0001	1.07	1.06 to 1.07	<0.0001	1.06	1.05 to 1.06	<0.0001	1.03	1.03 to 1.03	<0.0001
Season†												
Summer	1			1			1			1		
Autumn	1.02	1.02 to 1.03	<0.0001	1.02	1.02 to 1.03	<0.0001	1.02	1.02 to 1.03	<0.0001	1.03	1.02 to 1.03	<0.0001
Winter	1.03	1.02 to 1.04	<0.0001	1.03	1.02 to 1.04	<0.0001	1.03	1.03 to 1.04	<0.0001	1.03	1.03 to 1.04	<0.0001
Spring	1.05	1.04 to 1.06	<0.0001	1.05	1.04 to 1.05	<0.0001	1.05	1.04 to 1.05	<0.0001	1.05	1.04 to 1.05	<0.0001

*Risk Ratio, Adjusted for all variables in the table and humidity. The goodness-of-fit measures indented including dew temperature as well as humidity (continuous variable) as an optimal final model.
†The largest number of wildfires occurred in spring (ie, season 4, September to November) and summer (season1, December to February), while prescribed burns had the largest numbers in autumn (season 2, March to May) and winter (season 3, June to August). We could not distinguish between the landscape fires of wildfires and controlled burns for this study.

DISCUSSION

We estimated $PM_{2.5}$ concentrations exposure during LF events for Perth residents based on their area of residence. Exposure to elevated $PM_{2.5}$ during LF events at high levels (≥99 th percentile) was associated with increased EDA related to a range of adverse respiratory and CVDs. The largest point estimates were observed within 1 day after exposure. We also found a dose–response relationship in most lag-specific effect estimates, which adds to the limited evidence on the studies on the acute health effects from daily exposure $PM_{2.5}$ during LF events. Older adults aged 60 years and above and populations with lower socioeconomic backgrounds were more vulnerable to the impact of air pollution due to LF smoke.

We found significant associations between elevated $PM_{2.5}$ during LF events with ALRTI and non-significant increased risk with asthma. A recent US study found evidence that an increase in respiratory outcomes is mainly due to lower respiratory tract infections, which is consistent with what we found in this study.[38] An ecological study[39] found a significant 20% increase in asthma visits per 10 $\mu g/m^3$ increase in PM_{10} and that risk of an emergency contact for asthma in Darwin was 2.4 times greater on days with PM_{10} >10 $\mu g/m^3$ compared with days with PM_{10} <10 $\mu g/m^3$. Some studies found no associations in asthma ED presentations.[16 24] Studies using ambulance and hospital admission data have investigated the association between daily elevated $PM_{2.5}$ during LF events and selected adverse respiratory outcomes.[11 18 36 40 41]

Few studies have found a relationship between exposure to $PM_{2.5}$ during LF events and a wide range of cardiovascular morbidity associated with EDA. We found dose–response

Table 4 Multivariate poisson regression results for the association between EDA, a range of respiratory and cardiovascular diseases, and exposure to estimated PM2.5 for same-day (Lag0) and lags of 1–3 days

Health outcomes	Lag	RR*	95% CI	P value
Total EDA	0(M†)§	1.02	1.01 to 1.03	<0.0001
	0(H‡)§	1.05	1.03 to 1.06	<0.0001
	1(M)§	1.02	1.01 to 1.03	<0.0001
	1(H)§	1.04	1.03 to 1.05	<0.0001
	2(M)§	1.02	1.01 to 1.02	<0.0001
	2(H)§	1.03	1.02 to 1.04	<0.0001
	3(M)§	1.01	1.01 to 1.02	<0.0001
	3(H)§	1.03	1.03 to 1.04	<0.0001
Total respiratory	0(M)	0.96	0.93 to 0.99	0.021
	0(H)	0.96	0.9 to 1.02	0.144
	1(M)	0.96	0.94 to 0.99	0.001
	1(H)	0.94	0.91 to 0.99	0.008
	2(M)	0.97	0.95 to 0.99	<0.0001
	2(H)	0.94	0.9 to 0.97	<0.0001
	3(M)	0.97	0.95 to 0.99	<0.0001
	3(H)	0.95	0.92 to 0.98	<0.0001
Asthma	0(M)	1.03	0.93 to 1.14	0.604
	0(H)¶	1.07	0.89 to 1.29	0.464
	1(M)	1.03	0.96 to 1.11	0.442
	1(H)	1.1	0.96 to 1.25	0.167
	2(M)	1.02	0.96 to 1.08	0.517
	2(H)	1.07	0.96 to 1.19	0.219
	3(M)	1.03	0.98 to 1.08	0.268
	3(H)	1.04	0.94 to 1.14	0.446
ALRTI	0(M)	0.91	0.86 to 0.96	0.001
	0(H)	0.91	0.82 to 1.02	0.102
	1(M)	1.08	0.99 to 1.18	0.07
	1(H)**	1.19	1.1 to 1.29	<0.0001
	2(M)	0.93	0.9 to 0.96	<0.0001
	2(H)	0.83	0.78 to 0.89	<0.0001
	3(M)	1.08	1.02 to 1.15	0.015
	3(H)	1.17	1.1 to 1.23	<0.0001
COPD	0(M)	1.03	0.93 to 1.13	0.613
	0(H)	0.99	0.82 to 1.19	0.883
	1(M)	1	0.94 to 1.08	0.913
	1(H)	0.98	0.86 to 1.12	0.747
	2(M)	1	0.94 to 1.06	0.961
	2(H)	0.96	0.86 to 1.07	0.438
	3(M)	0.98	0.94 to 1.03	0.513
	3(H)	0.98	0.89 to 1.08	0.674
Croup	0 (M)	0.86	0.76 to 0.96	0.006
	0 (H)	1.1	0.91 to 1.33	0.31
	1(M)	0.89	0.83 to 0.96	0.004
	1(H)	0.97	0.84 to 1.11	0.644
	2(M)	0.88	0.82 to 0.94	<0.0001
	2(H)	0.91	0.81 to 1.03	0.133
	3(M)	0.89	0.84 to 0.94	<0.0001
	3(H)	0.89	0.8 to 0.99	0.029
Total cardiovascular	0(M)††	1.02	0.99 to 1.05	0.154
	0(H)	1.07	1.01 to 1.12	0.019
	1(M)	1.02	one to 1.04	0.051
	1(H)††	1.06	1.03 to 1.11	0.001
	2(M)††	1.01	one to 1.03	0.118
	2(H)††	1.06	1.02 to 1.09	0.001
	3(M)††	1.01	0.99 to 1.02	0.23
	3(H)††	1.05	1.02 to 1.07	0.001

Continued

Table 4 Continued

Health outcomes	Lag	RR*	95% CI	P value
Arrhythmia	0(M)	1.08	0.98 to 1.18	0.107
	0(H)	1.05	0.87 to 1.26	0.639
	1(M)	1.01	0.95 to 1.08	0.74
	1(H)	1.03	0.9 to 1.17	0.686
	2(M)	1	0.95 to 1.06	0.99
	2(H)	0.99	0.89 to 1.11	0.887
	3(M)	1	0.95 to 1.05	0.902
	3(H)	0.96	0.87 to 1.06	0.422
Angina	0(M)	0.9	0.78 to 1.03	0.116
	0(H)	0.89	0.68 to 1.17	0.401
	1(M)	0.85	0.77 to 0.94	0.002
	1(H)	0.93	0.77 to 1.11	0.411
	2(M)	0.87	0.8 to 0.94	0.001
	2(H)	0.89	0.76 to 1.04	0.132
	3(M)	0.9	0.84 to 0.96	0.002
	3(H)	0.88	0.77 to 1.01	0.066
Stroke	0(M)	0.97	0.77 to 1.21	0.754
	0(H)	0.79	0.49 to 1.28	0.346
	1(M)	0.92	0.79 to 1.08	0.327
	1(H)	0.93	0.68 to 1.27	0.642
	2(M)	0.93	0.81 to 1.05	0.246
	2(H)	1.06	0.84 to 1.35	0.623
	3(M)	0.96	0.86 to 1.07	0.469
	3(H)	1.04	0.85 to 1.28	0.69
Heart failure	0(M)	0.92	0.81 to 1.05	0.231
	0(H)	0.87	0.67 to 1.12	0.286
	1(M)	0.93	0.85 to 1.02	0.161
	1(H)	0.75	1.07 to 1.04	0.242
	2(M)	0.95	0.88 to 1.02	0.182
	2(H)	0.92	0.8 to 1.06	0.301
	3(M)	0.96	0.9 to 1.03	0.317
	3(H)	0.93	0.82 to 1.05	0.256
ACS	0(M)	1.13	One to 1.28	0.045
	0(H)	1.02	0.8 to 1.3	0.896
	1(M)	1.06	0.98 to 1.16	0.163
	1(H)	1.05	0.89 to 1.25	0.569
	2(M)	1.03	0.95 to 1.1	0.474
	2(H)	1	0.87 to 1.15	0.995
	3(M)	1	0.93 to 1.06	0.891
	3(H)	1	0.88 to 1.13	0.99
TIA	0(M)	1.01	0.86 to 1.19	0.876
	0(H)	1.14	0.84 to 1.54	0.391
	1(M)	1.02	0.91 to 1.15	0.685
	1(H)	1.25	1.02 to 1.53	0.034
	2(M)	1.02	0.93 to 1.12	0.681
	2(H)	1.2	1.01 to 1.42	0.039
	3(M)	1.02	0.94 to 1.11	0.66
	3(H)	1.14	0.98 to 1.32	0.095

*Adjusted for dew temperature, humidity, SEIFA, age, sex, weekend, public holiday, wood fire use and season.
†M=Medium $PM_{2.5}$.
‡H=High $PM_{2.5.}$
§Total ED: Interactions with those aged above 60 years with significant 3%–5% increase. There was also an interaction with SEIFA in lag3 with the disadvantaged group.
¶Asthma: Interactions with SEIFA with 85% increase at a high smoke level for the medium disadvantaged group.
**ALRTI: Interactions with sex with 20% increase at a high smoke level for females.
††Total cardiovascular: Interactions with age above 60 years old with significant 5%–8% increase.
ACS, acute coronary syndrome; ALRTI, acute lower respiratory tract infections; COPD, chronic obstructive pulmonary disease; EDA, emergency department attendance; TIA, transient ischaemic attacks.

associations between exposure to elevated $PM_{2.5}$ at high concentration (≥12.60 μg/m^3) with total CVD outcomes within all lagged effects. This is consistent with the evidence for urban exposure to $PM_{2.5}$.[10 42 43] We also found up to a 25% increase in risk with dose–response relationship for the TIA within 1–2 days after exposure, which is consistent with a study in Victoria,

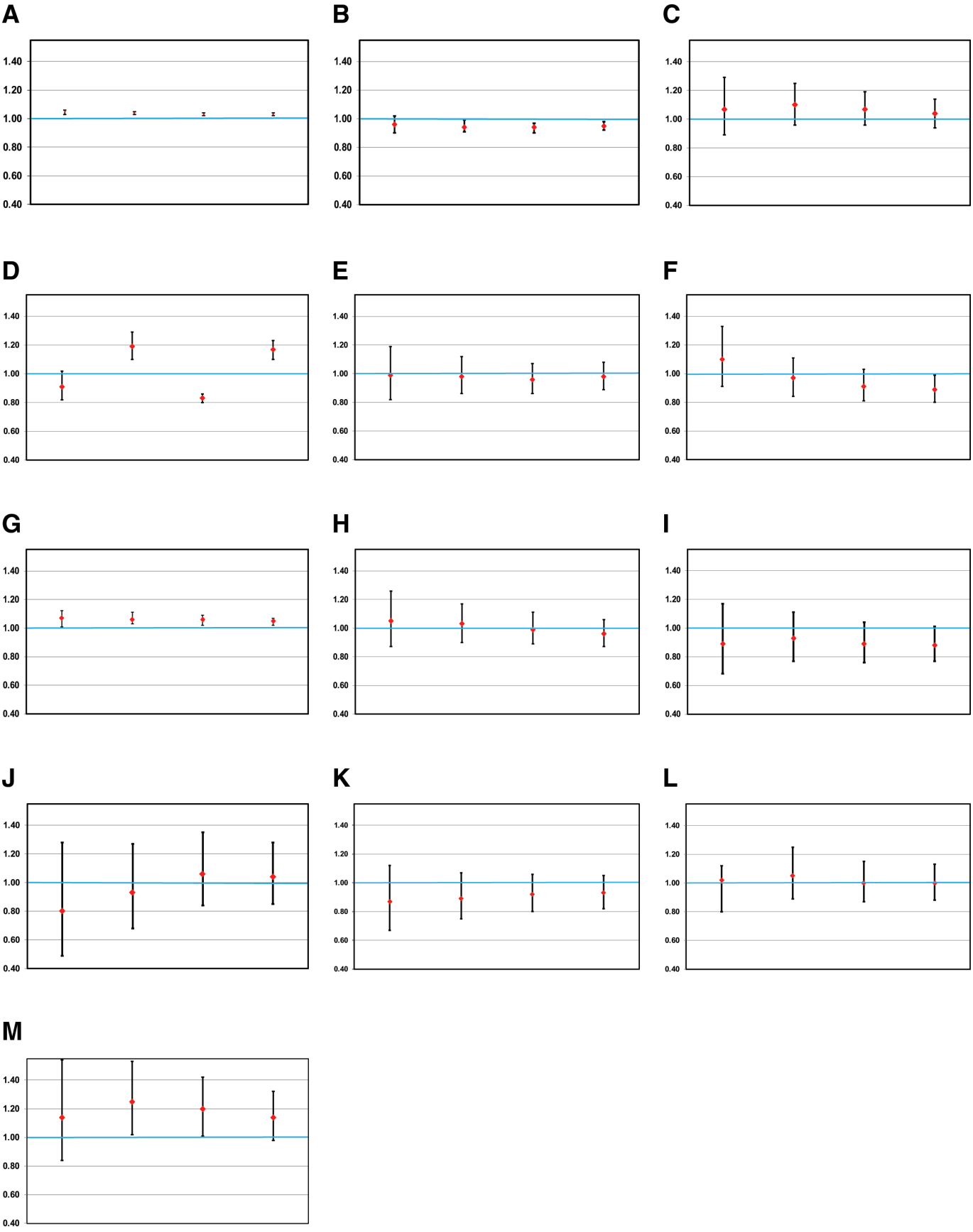

Figure 2 Risk ratio (RR) and 95% CIs for assessing of the effects of estimated PM2.5 at a high level on daily EDA for cardiorespiratory outcomes. In each graph, first to fourth bars show RR and 95% CI for lag 0 on the left to lag four on the right, respectively.
A;Total EDA; B, All respiratory; C, Asthma; D, ALTRI; E, COPD; F, CROUP; G, All cardiovascular; H, Arrhythmia; I, Angina; J, Stroke; K, Heart failure; I, ACS; M, TIA.

Australia, which found an increase in ischaemic heart disease (IHD) by 2.07% within 2 days after exposure to WFs. Studies of urban $PM_{2.5}$ have reported a 2%–20% increase in the risk of acute IHD-related morbidity for a 10 mg/m^3 increase in $PM_{2.5}$ levels.[10 44 45] The sustained effects of elevated $PM_{2.5}$ exposure during LF events and cumulative biological effects could be responsible for a delayed effect of $PM_{2.5}$ exposure on acute CHD events.[8 17] The influence of individual perceptions, the severity of symptoms, environmental conditions and decisions to seek medical care during LF events have been discussed in the literature as possible explanations for the delayed impact of elevated $PM_{2.5}$ during LF events, which needs further exploration.[8 14 17]

There is limited research to address the question of population subgroups at greater risk of adverse health effects from elevated $PM_{2.5}$ during LF events. Our findings about cardiorespiratory related EDA are consistent with available evidence that the elderly[15 17] and people from low socioeconomic backgrounds[14 22 36] are most likely to be susceptible to elevated $PM_{2.5}$. We also reported that women had a 20% increased chance of ALRTI compared with men, consistent with a previous study.[18]

This study has several strengths. The main strength was using temporally and spatially resolved modelled air pollution exposure data for an entire city, including areas with no monitoring facilities. We designed air pollution with finer spatial resolution within 1.5 km by 1.5 km compared with 5 km by 5 km used by other studies using a similar methodology.[11 26 46] This finer spatial resolution would reduce uncaptured variability within the prediction grid cells with greater accuracy and reduce exposure misclassification. We used the IDW method, which is a more reliable method to calculate the allocated values of LF indicators, compared with the nearest monitoring site used by previous studies.[11 26] We used a comprehensive state-wide health dataset (EDA) with a wide range of respiratory and CVDs. We also obtained sociodemographic, socioeconomic, clinical data and other information relevant to this study. Finally, we assessed the dose–response relationship, which is a crucial aspect of the damage caused by pollution exposure.

There were also several limitations. It included a limited number of air quality monitoring stations in the Perth metropolitan area to establish a more robust model. We used the IDW to spatially interpolate air quality measures to address this limitation. Despite this, there might be a possibility of some degrees of uncertainty in the accuracy of the predicted estimate of $PM_{2.5}$ during LF events. Second, we could not distinguish between the effect of WFs and PBs on daily smoke count as there was a possibility of having both types of fire within the same day. However, WFs were more common than prescribed burns and accounted for about 80% of the total LFs in the study area during the study period. Thirdly, despite the modelling exposure to $PM_{2.5}$ during LF events, there might be still likely to have exposure misclassification, as all persons would not have been exposed to the same levels of $PM_{2.5}$, and there might be some missed periods/grids of elevated PM. However, our finer spatial resolution of 1.5 km by 1.5 km grid cell would reduce this exposure misclassification.

Finally, we were unable to examine all confounding sources of $PM_{2.5}$ that might exist inside people's homes and immediate environments. However, we did consider the woodfire use in the analysis to capture it as one of the home air pollution sources.

CONCLUSION

We used an established empirical $PM_{2.5}$ exposure model to assess the population's exposure to LFs in WA. This study is the first to find a significant dose–response relationship between exposure to elevated $PM_{2.5}$ during LF events and an increased risk of EDA due to TIA. The high $PM_{2.5}$ during LF events is also accompanied by an increase in the number of patients seeking emergency services, total CVD and some common health problems in the community such as asthma and acute lower respiratory infections. The results are consistent with the literature suggesting that older people aged 60 years and above, people with disadvantaged status and those with heart or lung problems are more susceptible in the affected communities to LF smoke. This study has implications for practical public health response actions for the appropriate time scale during LF events.

Author affiliations
[1]Epidemiology Branch, Department of Health, Government of Western Australia, East Perth, WA, Australia
[2]School of Population Health, Curtin University, Bentley, WA, Australia
[3]College of Arts, Business, Law, and Social Sciences, Murdoch University, Murdoch, WA, Australia
[4]Department of Spatial Sciences, School of Earth Sciences, Curtin University, Bentley, WA, Australia
[5]School of Natural Sciences, University of Tasmania, Hobart, Tasmania, Australia
[6]School of Population Health, University of Western Australia, Crawley, WA, Australia
[7]Environmental Prediction Services – Severe Weather, Bureau of Meteorology (WA Office), West Perth, WA, Australia
[8]NGIS, West Perth, WA, Australia

Twitter Adeleh Shirangi @AdelehShirangi

Acknowledgements The authors are grateful to the Data Linkage Unit at the WA Department of Health for providing health utilisation data. The authors would acknowledge Dr Emmanuel Ongee for his excellent work on smoke plume shape digitalisation, Ms Laura Clappinson for her help in applying data and ethics for the project, and Professor Fay Johnston's advice on the early stage of the project. The authors also would aknowledge Professor Bert Veenendaal (Professor of spacial sciences at Curtin University), who took part in the initial part of the funding idea.

Contributors All authors contributed to the project's conceptualisation, research design and implementation. All authors reviewed, co-authored, and approved the manuscript. AS led the development of the concept and design for this paper, performed the literature review, led the selection, design and arrangement of the exposure assessment methodology, performed epidemiological analysis, including exposure modelling and health risk assessment, and wrote the paper; JX contributed to project administration, funding acquisition, and supporting role in the development of methodology and creation of models; TL, GY undertook spatial IDW modelling and creating the map; II contributed to project administration and supervision of digitalised smoke plume shape process; AD contributed to supervision of digitalised smoke plume shape process; GW advised on spatial exposure modelling; RB contributed to digitalisation of smoke plume shapes; BS, NE contributed to the provision of resource materials for the exposure assessment. PF reviewed the paper; LJ contributed to ethics application and reviewed the paper; JX and AS are responsible for the overall content as guarantors.

Funding The research leading to these results received funding from FrontierSI (CRCSI, Cooperative Research Centre for Spatial Information) under a subcontract agreement between Curtin University and AS (Curtin University Reference: RES-60362/CTR-12933 FrontierSI Project 5H02). While conducting this research project, AS was an employee at the WA Department of Health.

Disclaimer The views expressed in this publication are those of the authors and not necessarily those of the FrontierSI, Curtin University, or the WA Department of Health.

Map disclaimer The depiction of boundaries on this map does not imply the expression of any opinion whatsoever on the part of BMJ (or any member of its group) concerning the legal status of any country, territory, jurisdiction or area or of its authorities. This map is provided without any warranty of any kind, either express or implied.

Competing interests None declared.

Patient consent for publication Not applicable.

Ethics approval This study was approved by the Western Australia Department of Health Human Research Ethics Committee (No. 2018/31) and the Curtin University Ethics Committee (No. HRE 2019-0029).

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement No data are available.

ORCID iD
Adeleh Shirangi http://orcid.org/0000-0002-4842-5513

REFERENCES

1 Pio CA, Legrand M, Alves CA, *et al*. Chemical composition of atmospheric aerosols during the 2003 summer intense forest fire period. *Atmos Environ* 2008;42:7530–43.
2 Dennekamp M, Abramson MJ. The effects of bushfire smoke on respiratory health. *Respirology* 2011;16:198–209.
3 Spracklen DV, Mickley JA, Logan JA, *et al*. Impacts of climate change from 2000 to 2050 on wildfire activity and carbonaceous aerosol concentrations in the Western United States. *Journal of Geophysical Research Atmospheres* 2009;14:1–17.
4 Dennekamp M, Straney LD, Erbas B, *et al*. Forest fire smoke exposures and out-of-hospital cardiac arrests in Melbourne, Australia: a case-crossover study. *Environ Health Perspect* 2015;123:959–64.
5 Reisen F, Hansen D, Meyer CPM. Exposure to bushfire smoke during prescribed burns and wildfires: firefighters' exposure risks and options. *Environ Int* 2011;37:314–21.
6 Luhar A, Galbally I, Keywood M. Modelling PM10 concentrations and carrying capacity associated with woodheater emissions in Launceston, Tasmania. *Atmos Environ* 2006;40:5543–57.
7 Englert N. Fine particles and human health--a review of epidemiological studies. *Toxicol Lett* 2004;149:235–42.
8 Haikerwal A, Akram M, Del Monaco A, *et al*. Impact of fine particulate matter (PM2.5) exposure during Wildfires on cardiovascular health outcomes. *J Am Heart Assoc* 2015;4:e001653.
9 Cohen AJ, Ross Anderson H, Ostro B, *et al*. The global burden of disease due to outdoor air pollution. *J Toxicol Environ Health A* 2005;68:1301–7.
10 Pope CA, Muhlestein JB, May HT, *et al*. Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. *Circulation* 2006;114:2443–8.
11 Johnston FH, Salimi F, Williamson GJ, *et al*. Ambient particulate matter and Paramedic assessments of acute diabetic, cardiovascular, and respiratory conditions. *Epidemiology* 2019;30:11–19.
12 Haikerwal A, Akram M, Sim MR, *et al*. Fine particulate matter (PM2.5) exposure during a prolonged wildfire period and emergency department visits for asthma. *Respirology* 2016;21:88–94.
13 Rappold AG, Stone SL, Cascio WE, *et al*. Peat bog wildfire smoke exposure in rural North Carolina is associated with cardiopulmonary emergency department visits assessed through syndromic surveillance. *Environ Health Perspect* 2011;119:1415–20.
14 Rappold AG, Cascio WE, Kilaru VJ, *et al*. Cardio-Respiratory outcomes associated with exposure to wildfire smoke are modified by measures of community health. *Environ Health* 2012;11:71.
15 Morgan G, Sheppeard V, Khalaj B, *et al*. Effects of bushfire smoke on daily mortality and hospital admissions in Sydney, Australia. *Epidemiology* 2010;21:47–55.
16 Cooper CW, Mira M, Danforth M, *et al*. Acute exacerbations of asthma and bushfires. *Lancet* 1994;343:1509.
17 Delfino RJ, Brummel S, Wu J, *et al*. The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003. *Occup Environ Med* 2009;66:189–97.
18 Henderson SB, Brauer M, Macnab YC, *et al*. Three measures of forest fire smoke exposure and their associations with respiratory and cardiovascular health outcomes in a population-based cohort. *Environ Health Perspect* 2011;119:1266–71.
19 Henderson SB, Johnston FH. Measures of forest fire smoke exposure and their associations with respiratory health outcomes. *Curr Opin Allergy Clin Immunol* 2012;12:221–7.
20 Johnston FH, Hanigan IC, Henderson SB, *et al*. Creating an integrated historical record of extreme particulate air pollution events in Australian cities from 1994 to 2007. *J Air Waste Manag Assoc* 2011;61:390–8.
21 Johnston FH, Kavanagh AM, Bowman DMJS, *et al*. Exposure to bushfire smoke and asthma: an ecological study. *Med J Aust* 2002;176:535–8.
22 Hanigan IC, Johnston FH, Morgan GG. Vegetation fire smoke, Indigenous status and cardio-respiratory hospital admissions in Darwin, Australia, 1996–2005: a time-series study. *Environ Health* 2008;7:12.
23 Elliott CT, Henderson SB, Wan V. Time series analysis of fine particulate matter and asthma reliever dispensations in populations affected by forest fires. *Environ Health* 2013;12:11.
24 Smith MA, Jalaludin B, Byles JE, *et al*. Asthma presentations to emergency departments in Western Sydney during the January 1994 bushfires. *Int J Epidemiol* 1996;25:1227–36.
25 Yao J, Eyamie J, Henderson SB. Evaluation of a spatially resolved forest fire smoke model for population-based epidemiologic exposure assessment. *J Expo Sci Environ Epidemiol* 2016;26:233–40.
26 Yao J, Henderson SB. An empirical model to estimate daily forest fire smoke exposure over a large geographic area using air quality, Meteorological, and remote sensing data. *J Expo Sci Environ Epidemiol* 2014;24:328–35.
27 Australian Bureau of Statistics. 3218.0-Regional population growth, Australia, 2016. Available: https://www.abs.gov.au/AUSSTATS/abs@.nsf/ProductsbyReleaseDate/7B33A7E366915C49CA258291001DFE75?OpenDocument
28 Australian Bureau of Statistics. *2016 Census QuickStats - Greater Perth*. Commonwealth of Australia, 2017.
29 Australian Government Geoscience Australia. *Digital earth Australia hotspot*, 2020.
30 EARTH DATA. MCD19A2v006, 2020. Available: https://lpdaac.usgs.gov/products/mcd19a2v006/
31 Japan Meterological Agency. Himawari 8, 2020. Available: https://www.jma.go.jp/jma/indexe.html
32 NOAA. Hazard mapping system fire and smoke product. office of satellite and product Oerations, 2020. Available: https://www.ospo.noaa.gov/Products/land/hms.html
33 Wong DW, Yuan L, Perlin SA. Comparison of spatial interpolation methods for the estimation of air quality data. *J Expo Anal Environ Epidemiol* 2004;14:404–15.
34 GIS Geography. *Inverse distance weighting (IDW) Interplolation*, 2020.
35 Johnston FH, Purdie S, Jalaludin B, *et al*. Air pollution events from forest fires and emergency department attendances in Sydney, Australia 1996-2007: a case-crossover analysis. *Environ Health* 2014;13:105.
36 .Johnston FH, Bailie RS, Pilotto LS, *et al*. Ambient biomass smoke and cardio-respiratory hospital admissions in Darwin, Australia. *BMC Public Health* 2007;7:240.
37 Shirangi A, Xiao J, Ongee E. Earth, wind and fire: using satellite imagery to map the health efforts of landscape fire smoke on Perth metropolitan residents. main report in: department of health GoWA, 2021. Available: https://ww2.health.wa.gov.au/Reports-and-publications/Earth-Wind-and-Fire-Health-effects-of-landscape-fire-smoke
38 Miller N, Molitor D, Zou E. Blowing smoke: health impacts of wildfire plume dynamics. corpus ID: 52839640 [1-35], 2017. Available: https://www.semanticscholar.org/paper/Blowing-Smoke-%3A-Health-Impacts-of-Wildfire-Plume-1-Miller/365190c5bfc20e3e02d5e4b771d42f11fd1c1241
39 Johnston FH, Henderson SB, Chen Y, *et al*. Estimated global mortality attributable to smoke from landscape fires. *Environ Health Perspect* 2012;120:695–701.
40 Salimi F, Henderson SB, Morgan GG, *et al*. Ambient particulate matter, landscape fire smoke, and emergency ambulance dispatches in Sydney, Australia. *Environ Int* 2017;99:208–12.
41 Hanigan IC, Johnston FH. Respiratory hospital admissions were associated with ambient airborne pollen in Darwin, Australia, 2004-2005. *Clin Exp Allergy* 2007;37:1556–65.
42 Brook RD, Rajagopalan S, Pope CA, *et al*. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American heart association. *Circulation* 2010;121:2331–78.
43 Nawrot TS, Perez L, Künzli N, *et al*. Public health importance of triggers of myocardial infarction: a comparative risk assessment. *Lancet* 2011;377:732–40.
44 Peters A, Liu E, Verrier RL, *et al*. Air pollution and incidence of cardiac arrhythmia. *Epidemiology* 2000;11:11–17.
45 Pope CA, Dockery DW. Health effects of fine particulate air pollution: lines that connect. *J Air Waste Manag Assoc* 2006;56:709–42.
46 .Yao J, Brauer M, Wei J, *et al*. Sub-Daily exposure to fine particulate matter and ambulance dispatches during Wildfire seasons: a case-crossover study in British Columbia, Canada. *Environ Health Perspect* 2020;128:67006.