Government of **Western Australia**
Department of **Health**

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Earth, wind and fire

Using satellite imagery to map the health effects of landscape fire smoke on Perth metropolitan residents

Main Report July 2021

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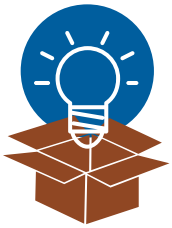
Courtesy of Department of Fire and Emergency Services Incident photographer Morten Boe.

Report overview

What is the health impact of smoke exposure due to landscape fires?

Landscape fires (LFs) are controlled (prescribed burns) or uncontrolled fires (e.g. wild fires) that occur in forest grass, scrub, bush or grasslands. Landscape fires are an important source of short-term air pollution. Information on the impact of acute exposure to landscape fires on members of the general population is currently limited. Findings from this study have relevance to health policy, partnerships, spatial application and policy.

Study aim



To expand understanding of:

- appropriate methods to measure population LF smoke exposure
- the impact of landscape fires on the general population, including the identification of vulnerable groups.

Methods

Satellite image analysis of smoke plumes via earth observation data.

Earth observation data linked to air quality and climate data to model and validate smoke exposure.

Air quality measure $PM_{2.5}$ linked to health utilisation data (i.e.hospital admissions, emergency department attendance and ambulance callouts).

Vulnerable groups

Older adults (60+ years), children, low socioeconomic areas, people with heart or lung conditions.

Key findings

LF smoke-related particulate matter ($PM_{2.5}$) was significantly associated with previous day $PM_{2.5}$ levels, venting index, fire radiative power, aerosol optical depth, fire danger rating and smoke plume masks.

A significant association was found between LF smoke-related $PM_{2.5}$ and emergency department attendances and hospital admissions for respiratory and cardiovascular conditions.

Health service utilisation peaked on the same day and 1, 2, or 3 days after exposure to landscape fire smoke.

Recommendations

Health professionals and policy makers should enhance education programs about the harms associated with landscape fires.

Programs should emphasise medical conditions involved and possible delayed smoke effects.

Regular and real time fire data should be collected to determine population and geographical areas at risk.

Project partners:

Government of **Western Australia** Department of **Health**

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Abbreviations

AC	Ambulance Callouts
ACS	Acute Coronary Syndrome
ALRTI	Acute Lower Respiratory Tract Infections
AOD	Aerosol Optical Depth
BOM	Australian Bureau of Meteorology
COPD	Chronic Obstructive Pulmonary Disease
CVD	Cardiovascular Diseases
CI	Confidence Interval
EDA	Emergency Department Attendances
FDR	Fire Danger Rating
FRP	Fire Radiative Power
GIS	Geographical Information System
GWRR	Geographically Weighted Ridge Regression
HA	Hospital Admissions
HMDS	Hospital Morbidity Data System
IDW	Inverse Distance Weighted
LF	Landscape Fire
PC	Postcode
$PM_{2.5}$	Particulate Matter with a diameter of < 2.5 micrometres
$PM_{2.5}$lag0	$PM_{2.5}$ in the current day
$PM_{2.5}$lag1	$PM_{2.5}$ in the previous day
RR	Risk Ratio
SA2	Statistical Area Level 2
SEIFA	Socioeconomic Indexes for Areas
SPM	Smoke Plume Mask
TIA	Transient Ischemic Attack
VI	Venting Index
WA	Western Australia

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Executive summary

In this study, we aimed to expand the understanding of acute exposure to smoke from landscape fires (LFs) in relation to a range of respiratory and cardiovascular health effects using three health care utilisations datasets (emergency department attendances (EDA), hospital admissions (HA), and ambulance callouts (AC)). We modified an established smoke optimised empirical $PM_{2.5}$ exposure model and used earth observation data from fine spatial-temporal resolution satellite images in assessing the population's exposure to landscape fires smoke in the Perth metropolitan area of Western Australia. We used estimated daily LF smoke related $PM_{2.5}$ concentrations from the model to evaluate the effects of LFs on health outcomes. We found that the methods were useful in evaluating the effects of LFs smoke on a wide range of adverse respiratory and cardiovascular diseases and in identifying vulnerable populations due to landscape fires.

The key findings of the study are categorised in the following three areas.

1. Image analysis to identify smoke plumes and affected areas

- Smoke plume identification and spatial analysis were useful and effective tools in more precisely identifying the movement of smoke and affected geographical areas, thus producing better population exposure estimates.
- Through this study a systematic way of identifying, digitalising, and rasterising smoke plumes into spatial grid cells was developed. Such a system could be potentially used in other similar studies.

2. An empirical LF smoke exposure model

- A systematic methodology was employed in this study to model and validate LF smoke related $PM_{2.5}$ concentrations in the Perth metropolitan area that was not evident from the air quality monitoring network.
- A series of air quality, earth observation and climate data were used in such modelling processes. The parameters included $PM_{2.5}$, remotely sensed fire radiative power (FRP), aerosol optical depth (AOD), smoke plume masks (SPM), venting index (VI) and fire danger rating (FDR).
- The exposure estimates from the model were also used for Statistical Area level 2 (SA2) geographical units, so that the geographical variation analysis of the health impacts of LF smoke became possible.
- The LFs smoke related $PM_{2.5}$ for a day was significantly associated with all independent variables in the established model ($P<0.05$). Such variables included observed $PM_{2.5}$ concentrations in previous day ($R^2=0.175$), followed by VI ($R^2=0.036$), FRP ($R^2=0.031$), AOD ($R^2=0.015$), FDR ($R^2=0.006$), and SPM ($R^2=0.002$). The model explained a total of 24% variance in the PMlag0 values.

3. Assessment of smoke related $PM_{2.5}$ and health utilisation relationship

- There was a strong link between smoke related $PM_{2.5}$ and health utilisation for a wide range of respiratory and cardiovascular responses related emergency department attendances and hospital admissions. There was weaker evidence of an association with ambulance callouts.
- Landscape fire smoke exposure significantly increases general emergency department visits and general hospital admissions, but with no significant increase in general ambulance callouts.
- There was a significant dose-response association in delayed effects of lag 1 and lag 3 with 8% to 19% increased risk for acute lower respiratory tract infections attending emergency departments.
- There was also a non-significant 25% increase in ambulance callouts due to respiratory arrest.
- There was a strong effect for cardiovascular diagnosis with a 2% to 7% significant increased risk at the high exposure level to smoke related $PM_{2.5}$ in both EDA and hospital admissions in the general cardiovascular category.
- There was a significant increased EDA due to diagnosis related to transient ischemic attack with a significant dose-response increased risk up to 25%.
- Older people aged 60 years and above, people living in low socioeconomic areas and those with heart or lung problems are more susceptible to LF smoke.
- There was a non-significant dose-response effect for the impact of LF smoke-related $PM_{2.5}$ on respiratory effects for asthma with a 3% to 10% non-significant increased risk seeking emergency department and 2% to 18% non-significant increased risk in hospital admissions, and up to 11% non-significant increased risk in ambulance callouts on the same day and in almost all delayed lag effects.

Based on the study findings we recommend the following two categories of recommendations.

Policy recommendations

- Policy makers and health professionals should initiate and enhance community education programs about the harms caused by the landscape fires. Community education programs should also provide practical advice on actions that can be taken by individuals to minimise exposure and potential harm including the emphasis of the main respiratory and cardiovascular conditions identified in the study.
- Health education programs should focus on fire prevention, management and general safety, and adopting personal protective behaviours during a smoke episode such as taking reasonable precautions to avoid bushfire smoke. The program should also include the emphasis of possible delayed smoke effects on individuals in the affected areas. The programs should start prior to the LF seasons (i.e. September to June).
- Elderly people, children and populations living in lower socio-economic areas should be made aware of the effects of air pollution including landscape fire smoke in health promotion programs. As part of overall hazards planning, common locations for the elderly and children (e.g. nursing homes, child-care centres and schools) should be spatially identified and pre-warning information be sent to specific locations via different media.
- The resources should be increased to establish more air quality stations than the current number, especially in the southern, eastern and northern outskirts of the Perth metropolitan area and Southwest areas where LFs occur frequently. Immediate mobile monitoring of particulate matter concentrations (in particular, $PM_{2.5}$) and data on routine ambient air quality monitoring in a local region to reflect community exposures are recommended.

Technical recommendations

- Regular and real time capturing of landscape fire data should be implemented to determine population/geographical areas at risk. Smoke plumes identified via the satellites and other earth observation data collected should be used to assist in capturing LFs and/or monitoring their movement for improving early warning systems.
- Spatial services could assist with mapping smoke plumes and at-risk populations in the affected area. The existing spatial service at jurisdictions should be expanded to accommodate the increased need if required. In particular, the development of automatic mapping/digitalisation of smoke plumes should be considered so that timeliness and efficiency of tracking the trajectory of smoke plumes can be realised.
- Mobile apps such as AirRater (https://airrater.org/what-does-it-monitor/) or mobile messaging services such as asthma alert that are being developed in WA may incorporate such LF data so that LF exposed vulnerable populations can be informed and preventative measures can be taken in a timely manner.

1. Introduction and Background

Landscape fires (LFs) include wildfires (WFs) and prescribed/planned burns (PBs) and are defined as fires that occur in forest, scrub, or grassland (bushfires). These fires are an important source of short-term increases in particulate air pollution. Prescibed burning is the process of planning and applying controlled fires to predetermined areas, under specific environmental conditions to reduce the fuel load available for bushfires (Haikerwal et al., 2015). Landscape fires, particularly WFs, are a growing concern globally as they are expected to increase in frequency and intensity due to changes in our climate (Fried et al., 2008, Spracklen et al., 2009, Westerling et al., 2006). In a preliminary analysis of landscape fires project in Western Australia, there were approximately 8,000 LFs during 2015-2017, including approximately 1,773 (22%) LFs in Perth metropolitan and South West regions. Of these, 377 (21.2%) were PBs and 1,396 (78.7%) were WFs (internal unpublished government data) (Clappinson et al., 2019).

The effects of LF smoke on air quality and consequently human health depend greatly on factors such as the existing health condition of individuals, length of exposure, concentration and size of air pollutants (Stephenson, 2010). The extensive fires across Australia in the 2019/20 fire season are a clear, recent example of these impacts, with significant health concerns and protective equipment shortages experienced across Western Australia, Australian Capital Territory, Victoria and New South Wales (Arriagada et al., 2020). Short-term increases in particulate air pollution has been associated with a wide range of health effects, including exacerbations of respiratory symptoms, impaired lung function, medication use, physician visits, EDA, hospital admissions, paramedic services, cardiovascular effects and premature mortality (Dennekamp and Abramson, 2011, Haikerwal et al., 2015, Johnson et al., 2019, Liu et al., 2015, Liu et al., 2017, Pope and Dockery, 2006, Pope et al., 2006).

Like background urban air pollution, LF smoke contains a complex mixture of particulate matter, water vapour and gases, many of which are known to be air pollutants or greenhouse gases and can affect the health of human communities (Malilay, 1999). This occurs because bushfires smoke often disperses over long distances, hundreds of kilometres and may persist for days or even weeks. Severe bushfires are capable of causing widespread economic, social and environmental impacts across spatial and temporal dimensions and can be responsible for periods of extremely poor air quality (Stephenson, 2010). Therefore, it is important to assess the impact of fires on a wide population scale, rather than focusing on communities in the immediate vicinity of fires. Even small increases in risk could cause large public health impacts (Stephenson, 2010).

The pollutant that is most consistently elevated due to LF smoke is particulate matter (PM) in the air, which can reach exceedingly high levels, and is considered dangerous to human health. PM is measured according to its diameter: coarse particles are between 2.5 and 10μm (micrometres) (PM10) and fine particles are between 1 and 2.5μm ($PM_{2.5}$). Both size fractions have been associated with adverse health effects but $PM_{2.5}$ is of particular concern and has the strongest effect (Tham and Bell, 2008). Airborne PM, particularly $PM_{2.5}$, has been associated with respiratory tract symptoms, asthma exacerbations, and lung cancer, as well as irregular heartbeats, heart attacks, and premature death (Pope, 2000). It has been hypothesized that fine particulate air pollution may provoke the release of harmful inflammatory cytokines, increased blood coagulability, changes in blood viscosity, thrombus formation, plague erosion, increase in heart rate and decrease in heart rate variability, leading

to cardiac death (Gold et al., 2000, Haikerwal et al., 2015, Pope, 2000). 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 (Cohen et al., 2005).

According to Environmental Protection Measure for Ambient Air Quality, the target for maximum mean PM10 over 24 hour period is 50 ug/m3 (Luhar et al., 2006). Studies have shown that during fires, it exceeded 50 ug/m3 during several days (2007, Pio et al., 2008), which indicates that PM concentrations several times above background urban concentrations can occur. Reisen et al (2011) provide data on PM exposure for sites in rural Australia including locations affected by the major Victorian Alpine bushfires of 2006/07. These are likely to be more representative than European or US studies due to differences in fuel type and consumption, terrain features and weather patterns. Given the increase in PM concentrations during smoke events and its impact on the local air quality, the need to understand the influence of LFs smoke exposure on human health is important. Understanding the patterns of the dose-response relationship between PM concentrations and health impact is also an important aspect of the damage caused by the population exposure to landscape fires. To date, there is a lack of evidence on the population' exposure level (dose response relationship) of landscape fire pollutions and their adverse health consequences.

There has been a wide variety of research on the impact of LFs smoke on physical health, for instance on asthma sufferers (Johnston et al., 2002), or firefighters (Aisbett et al., 2007), and mental health problems including anxiety, depression or post-traumatic stress disorder for a number of years (Epa Victoria, 2007, Stephenson, 2010). However, studies of the excessive levels of particulate matters (PM) in the atmosphere during severe bushfires and their impact on human health have not been conclusive and have shown mixed results with more consistent evidence for respiratory outcomes, with limited and inconsistent evidence on cardiovascular disease. (Cooper et al., 1994, Delfino et al., 2009, Dennekamp and Abramson, 2011, Dennekamp et al., 2015, Elliott et al., 2013, Hanigan et al., 2008, Henderson et al., 2011, Henderson and Johnston, 2012, Johnston et al., 2011, Johnston et al., 2002, Johnston et al., 2019, Morgan et al., 2010, Rappold et al., 2012, Rappold et al., 2011, Smith et al., 1996, Stephenson, 2010). The inconsistencies in results of cardiovascular effects are unexpected as daily $PM_{2.5}$ is associated with cardiovascular effects in urban environments (Brook et al., 2010, Martinelli et al., 2013). The discrepancies between smoke-related and urban $PM_{2.5}$ could be due to differences in the constituents of the particulate matter, or limited statistical power to detect the effect of LFs, or both (Yao et al., 2016). Hence, to detect small increases in health outcomes associated with LFs smoke, there is a need for further epidemiological studies with more accurate exposure modelled for large populations.

Few studies assessed the effect of bushfire smoke as measured by $PM_{2.5}$ on daily EDA, hospital admissions and ambulance callouts for cardio-respiratory outcomes (Dennekamp et al., 2015, Johnston et al., 2019, Rappold et al., 2012). There is also a lack of understanding of the health impacts of bushfire smoke related $PM_{2.5}$ on specific determinants of community health such as age, sex, socioeconomic status, and geographical variations as not all communities are affected equally. Elderly people, children and populations with lower socio-economic backgrounds share larger health burden in response to the environmental exposures as they are more susceptible to LFs smoke-related diseases compared to others (Miller et al., 2017, Molitor et al., 2011, Morello-Frosch et al., 2002, Rappold et al., 2012)

The overall aim of this study was to expand the understanding of acute exposure to LFs smoke in relation to a range of cardio-respiratory health effects using three health care datasets (EDA, hospital admissions, and AC). As such, we have modified and improved a recently developed empirical model, modelled air exposure data to estimate smoke-related $PM_{2.5}$ levels, especially in areas without any air-quality monitors, and evaluated the impact of short-term exposure to smoke-related $PM_{2.5}$ on adverse health outcomes such as increases in health care utilisations (emergency department attendances, hospitalisations, and ambulance callouts) for selected respiratory and cardiovascular morbidities. We also estimated lag effects of 1 to 3 days between exposure and selected adverse health outcomes and evaluated specific measures of community health such as age, sex and socio-economic area of residence as modifiers of risk for cardio-respiratory outcomes following an episode of acute exposure to LFs.

1.1 Study aim and objectives

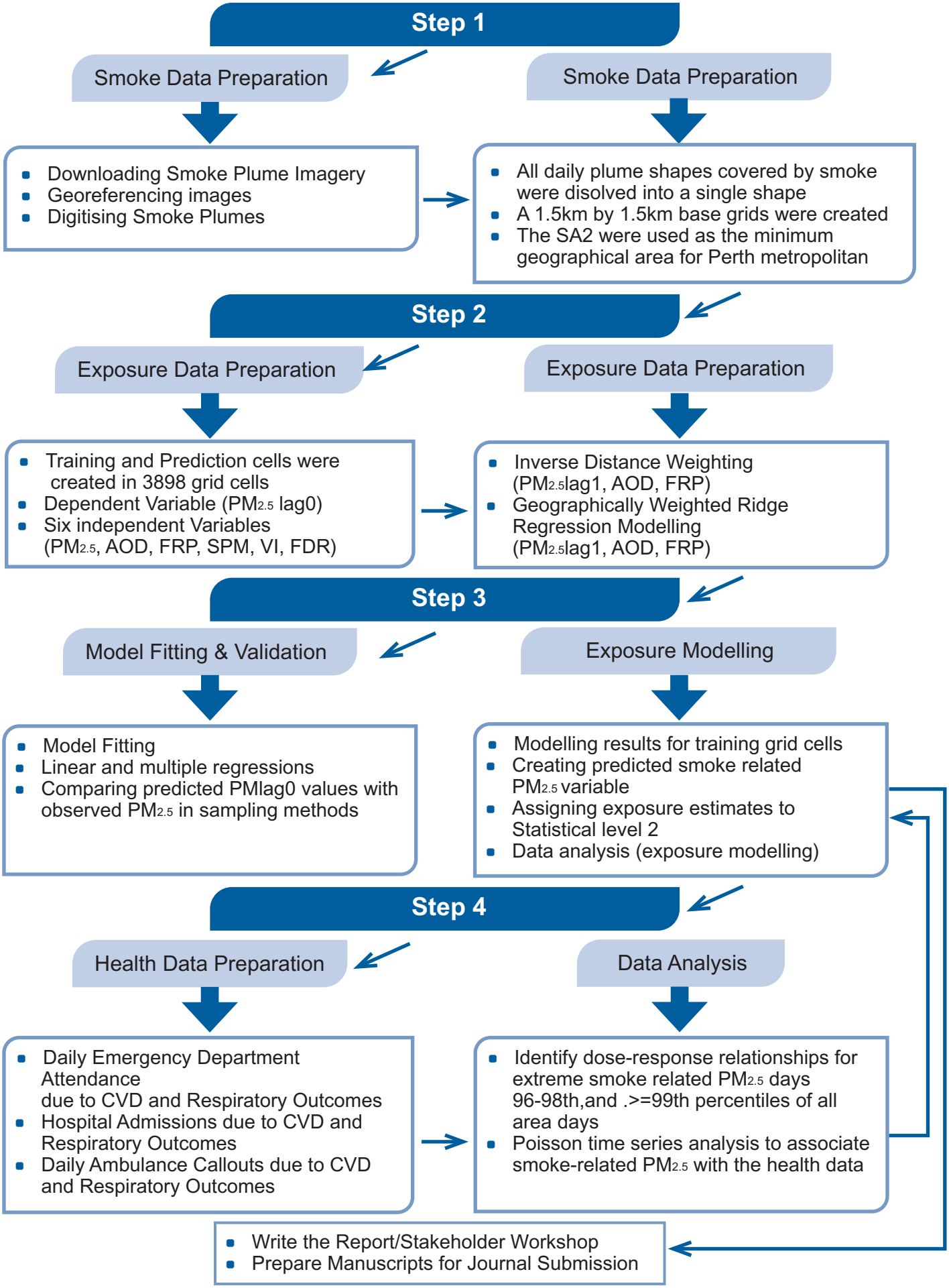
1.1.1 General aim

The aim of this project was to use earth observation data and other measures to develop a fire smoke-optimised empirical exposure model to assess whether LFs had significant adverse health effects on the general population in the Perth metropolitan area.

1.1.2 Specific objectives

1. To develop a smoke-optimised empirical fine particulate matter ($PM_{2.5}$) exposure model using earth observation data from satellite images and other related parameters to assess the population's exposure to LFs smoke in the Perth metropolitan area of Western Australia.
2. To assess the effects of LFs smoke including WFs and PBs on health service utilisations including hospital admission (HA), emergency department attendance (EDA) and ambulance callout (AC) for a wide range of cardio-respiratory conditions in the Perth metropolitan area.
3. To identify high risk areas and vulnerable populations more likely to be affected by LFs smoke.
4. To provide recommendations to inform policy development in reducing and/or eliminating the impacts of LFs including WFs and PBs using the project outcomes.

Figure 1. Overview of the landscape fire project design and analytical processes



2. Statistical and Spatial Analysis Methods

The statistical and spatial analyses were conducted in four steps as shown below.

Step 1: Image analysis to identify smoke plumes and affected areas

Step 2: An empirical smoke exposure model

Step 3: Air quality relationship assessment (model fitting)

Step 4: LF smoke related $PM_{2.5}$ and health utilisation relationship assessment

Flow chart in Figure 1 provides an overview of research plan design and analytical process of the landscape fire project.

2.1. Step 1: Image analysis to identify smoke plumes and affected areas

This step was to use satellite image analysis to identify smoke plumes and affected areas as smoke plume was one of the important potential predictors for the air quality modelling in steps 2 and 3 described below. Tracings of smoke plumes were made using hourly true-colour imagery from Himawari 8 satellite image as background. True-colour imagery was downloaded in a Portable Network Graphics (PNG) format, from Himawari 8 website (https://himawari8.nict.go.jp). To ensure proper alignment, images were georeferenced to the Map Grid of Australia Zone 50 (MGA50) coordinate reference system. Georeferencing was based on a second-order polynomial transform using 6 well-spaced control points.

Visual interpretation and digitisation of smoke plumes were done by GIS experts, using ArcGIS Desktop (version 10.6) software. Historical burnt-area data were used to guide the capture of smoke plumes. Smoke plumes caused by bushfires managed by local government or Department of Fire Emergency Services, agricultural burning, burning of debris associated with land development activities were excluded as there was no relevant/complete data available for the project. Digitisation was restricted to days of recorded fire. Burnt-area fire data for the study period (July 2015 – December 2017) were obtained from the Department of Biodiversity, Conservation and Attractions (DBCA).

To facilitate analyses on the daily timescale, hourly smoke plume tracings were grouped by calendar date, and then dissolved shapefiles of daily smoke plumes were analysed to characterise the spatial pattern of smoke plumes; analyses were conducted at both the grid cell level and SA2 administration unit level. To allow combination with other gridded climate products, daily smoke plume masks were further rasterized to 1.5 km x 1.5 km grids of proportional grid cell coverage. The grid size was chosen to match that of model outputs from the Australian Bureau of Meteorology (BOM), such as Venting Index (VI) and Fire Danger Rating (FDR).

2.2. Step 2: An empirical exposure model

The objectives for Step 2 of Landscape Fire (LF) project were

1) To model and validate LF smoke related (in short, smoke related) $PM_{2.5}$ concentrations in the Perth metropolitan area that was not covered by the air quality monitoring network.
2) To assign the exposure estimates from the model to Statistical Area level 2 (SA2) geographical units.

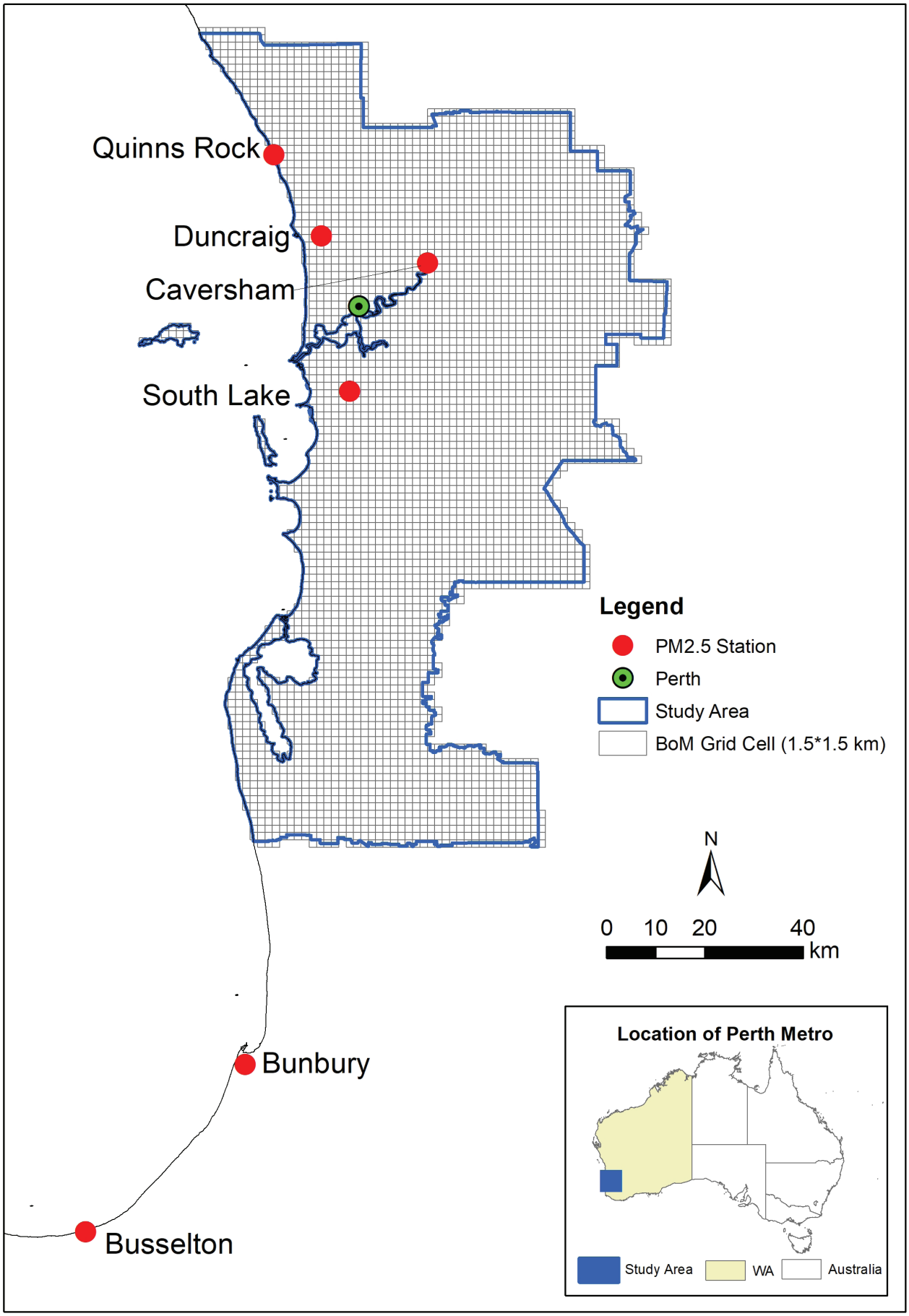
2.2.1 Materials and methods

We modified and developed an established smoke-optimised empirical model that estimated $PM_{2.5}$ concentrations by combining multiple data sources that reflect aerosol measurements, fire information and atmospheric conditions by adopting the general approach of Yao and Henderson (Yao and Henderson, 2014) to the Western Australian study area, i.e., Perth metropolitan area as shown in Figure 2.

a. Training and prediction grid cells

A total number of 3,898 grid cells with a resolution of 1.5km by 1.5km over the Perth metropolitan area were created as the base grids for modelling. This resolution was chosen to be consistent with the resolution for the VI and FDR data from Bureau of Meteorology (BOM). Here we refer to the 3,898 cells as “prediction cells”; and “training cells” refer to the subset of those prediction cells that contained at least one surface $PM_{2.5}$ monitoring station operated by Department of Water and Environmental Regulation during the study period (Figure 2). There were 4 training cells in the study and in total there were 915 days during the study period. Thus, there were 3,660 training cell records (=4 X 915) for the whole study period.

Figure 2. Study area and model estimate base grid cells (training & prediction cells) in Perth metropolitan area. Inset map showing the study area and the State of Western Australia with respect to Australia



b. Data sources, preparation, and process (exposure data)

In order to produce the empirical model, we constructed a multiple linear regression model where the $PM_{2.5}$ concentrations on a day for a grid cell ($PM_{2.5}$lag0) were used as a dependent variable and six independent variables included the observed $PM_{2.5}$ value on the previous day for that grid cell ($PM_{2.5}$lag1), remotely sensed fire radiative power (FRP), aerosol optical depth (AOD), whether smoke plume covered the centroid of a grid cell obtained in Step 1 above (also termed as smoke plume mask (SPM)) which is similar with the well-known HMS data product over North America[1], venting index (VI), and fire danger rating (FDR) that indicates pollutant dispersion potential (Yao and Henderson, 2014). Please see the full description of these variables and their sources below.

Population Data: Population data for a SA2 level estimated resident populations (ERPs) by age group, gender and SA2 for the Perth metropolitan area was sourced from the Australian Bureau of Statistics. The monthly populations were computed using a linear interpolation method, interpolated based on mid-year ERPs and such populations were then applied to all the days in the month.

Air Quality Data and Inverse Distance Weighted (IDW) Method: Air quality data was sourced from the Department of Water and Environmental Regulation for the whole study period (1 July 2015 to 31 December 2017). The data was from 10 stations for 4 main pollutants (NO, NO2, O3 and $PM_{2.5}$) covering the Perth metropolitan area. The average of maximum 8 hourly values of each air pollutant was calculated and represented as daily measures. In this report, the results of particulate matter with aerodynamic diameter ≤2.5 micro metres ($PM_{2.5}$) were presented as it is the small fine particulate matter that could penetrate deep into the alveolar region of the lung and enter the bloodstream, leading to a variety of manifestations of cardio-respiratory outcomes. The data for $PM_{2.5}$ was available for six stations as described in Table 1.

[1] https://www.ospo.noaa.gov/Products/land/hms.html

Table 1. Perth metro air quality stations, pollutants measured and station latitude and longitude

Station	Pollutants measured	Latitude	Longitude
Bunbury	$PM_{2.5}$	-33.3416	115.6433
Busselton	$PM_{2.5}$	-33.6482	115.352
Duncraig	$NO/NO_2/PM_{2.5}$	-31.8264	115.7829
Quinns Rock	$NO/NO_2/O_3/ PM_{2.5}$	-31.6779	115.6961
Rockingham	$NO/NO_2/O_3$	-32.2627	115.7485
Rolling Green	$NO/NO_2/O_3$	-31.6953	116.3186
Southlake	$NO/NO_2/O_3/PM_{2.5}$	-32.1106	115.8348
Swanbourne	$NO/NO_2/O_3$	-31.956	115.762
Wattleup	NO/NO_2	-32.1778	115.798

The value of each air pollutant for each BOM grid cell in the study area (totally 3,898) was calculated using inverse distance weighted (IDW) method, a type of deterministic method for multivariate interpolation with a known scattered set of points in the geospatial analysis (Wong et al., 2004). The inverse power used in this study was the number two. The allocated values to unknown points were calculated with a weighted average of the air quality values available at the known stations. IDW assigns more weight to closer known points than distant ones when estimating data at an unknown point.

Dependent Variable

Daily $PM_{2.5}$lag0: Hourly day measurements of $PM_{2.5}$ from the six air monitoring stations in the study area were used to calculate daily average ($PM_{2.5}$lag0) in each available training cell as the response variable. Four stations were in the Perth metropolitan area and two stations (Bunbury and Busselton) were in the Southern rural area where in the case of Bunbury there is also a substantial port and industrial hub (i.e., Kemerton and Dardanup) that could be generating various forms of emissions. The distance from Bunbury and Busselton to the nearest grid is about 45km and 85km, respectively. As the two rural stations were in areas with frequent landscape fire and close to the Perth metropolitan area, the measurements from these two stations were also included to estimate the $PM_{2.5}$ in the metropolitan area.

Multiple Imputations of Missing Values: The average of maximum eight hourly value of $PM_{2.5}$ for a day was calculated and represented as daily measures. As the four air quality measures had some missing values (5.17%), such missing values were estimated by using multiple imputation (MI) procedure in SAS. The MI method's main principle is to allow for the uncertainty about the missing values by providing different imputed datasets and combining results from each of those imputed datasets (Sterne et al., 2009). The imputation procedure is based on the concept that the missing values are filled by values that are taken from the distribution estimate of the non-missing dataset. Five imputation values were estimated and the average of those five estimated values was used to replace missing values.

Independent Variables

$PM_{2.5}$lag1: This was the daily average $PM_{2.5}$ from the previous day measurements. Each prediction cell was assigned the $PM_{2.5}$lag1 value using Inverse Distance Weighted (IDW) modelling as described above.

Smoke Plume Mask (SPM): These are represented by manually digitized smoke plume areas from satellite images from the Himawari 8 geostationary satellite operated by the Japanese Meteorological Agency (Japanese Meterological Agency, 2020). This is similar with the well-known HMS data product over North America (Noaa, 2020). Potential smoke days for the study area were identified using the Department of Biodiversity, Conservation, and Attractions state-wide fire history database. The Australian Bureau of Meteorology (BOM) assisted in downloading the relevant satellite image data. Images were then processed and georeferenced prior to use. We inspected each image for evidence of smoke plumes within a 100km buffer of the edge of the study area. Identified smoke plumes were then manually digitized and stored in a shapefile vector data format. For each hourly image containing smoke plumes, files containing one or more traced plumes were created. Identifiable LFs were selected to assist in defining the smoke plumes from satellite images and affected geographical areas. The trajectories of smoke plumes were obtained hourly from 9am-5pm (Western Standard Time) for each day in the study period. Training and prediction cells that had their centres covered by the plume were assigned a value of 1, otherwise a 0 (for both training and prediction cells). Further details on the use of training and prediction cell information can be found on Page 16 of the report. Hourly shape files were collated to provide daily estimates of the proportion of each of the metropolitan SA2s covered by the smoke plume. For each day on which smoke was observed, additional metadata was collected. These metadata indicate whether smoke was identified and digitised anywhere within the 100km buffer at any time of a day (yes/no), and if cloud was present in the buffer at any time of the day (yes/no). All image processing and digitisation was completed using ESRI ArcGIS (Version 10.7.1).

There were 237 days within the study period (915 days) where smoke plume data was captured. This variable SPM was used in exposure modelling to derive a predicated smoke related $PM_{2.5}$.

AOD (Aerosol Optical Depth): Remotely sensed AOD is a unitless measure of light absorption and extinction in the entire atmospheric column that correlates with on-ground particulate concentrations. Analyses were based on the blue band (0.47 μm) layer from the MCD19A2 Version 6 data product from NASA[2]. Data from multiple orbits were aggregated through simple pixel-based averaging. Although quality control flags are provided with the MCD19A2 data product, we ignored them in our analyses as quality filtering of data can adversely impact findings in smoke-related applications[3]. Finally, the one km resolution gridded data were resampled, using nearest neighbour interpolation, to a 1.5 km x 1.5 km spatial resolution to match the resolution of other variables used in regression analyses.

FRP (Fire Radiative Power): The FRP measures the intensity of landscape fire points, is proportional to their aerosol emissions. FRP data were extracted from the Geoscience Australia online grid[4]. The days with most severe fire activity was identified by sum of FRP values. FRP is the rate of energy emitted from the fire in Gigawatts (GW) within the one grid cell[5]. The WA Fire Power data from Geoscience Australia was used to calculate the FRP value. FRP is a point dataset. During a day, the same location can have multiple FRP

[2] https://doi.org/10.5067/MODIS/MCD19A2.006
[3] https://lpdaac.usgs.gov/documents/110/MCD19_User_Guide_V6.pdf
[4] https://sentinel.ga.gov.au/#/
[5] http://cedadocs.ceda.ac.uk/770/1/SEVIRI_FRP_documentdesc.pdf

values; the mean FRP value for the same location on the same day was used. According to the literature (Price et al., 2012), the FRP values within 400 km buffer distance were used and recommended for the Perth metropolitan area and the IDW methods was used to estimate the grid cell FRP values in the study area based on the distance of each grid cell to the point FDR values from Geoscience Australia.

Venting Index (VI): It measures daily forecast of VI in m2/s at all stations across Australia. The VI was calculated from hourly data and then averaged for the day. The VI is equal to the depth of the atmospheric boundary layer times by the mean wind speed in the boundary layer. The VI ranges from 0 to 7050, indicating the potential for the atmosphere to disperse airborne pollutants, based on the wind speed in the mixed layer and the thickness of the mixed layer.

The formulas and the scale used to calculate VI in statistical exposure modelling is as below:

$$\text{VI} = \text{wind speed (m/s)} \times \text{boundary layer depth (m)}$$

The scale (dispersion class) for the VI is the same as used by the US Forest Services as below:

Very poor (0-2350), Poor (2350-4700), Fair – marginal (4700-7050), Good (>7050).

The VI and FDR data were sourced from BOM for the whole study period as the Network Common Data Form (NetCDF) file, a file format for storing multidimensional scientific data (variables). Each of these variables was displayed through a dimension (time) in GIS software and then the values for those variables were extracted to BoM grid cells for the study area.

Fire Danger Rating (FDR): The FDR, sourced from BOM, is a numeric index between 0 to 100 that indicates the fire risk in an area based on meteorological conditions, fuel availability, fuel moisture, and other indicators[6]. Table 2 indicates the range of GFDI/FFDI typically associated with each FDR as used in Western Australia[7].

Table 2. Fire danger rating and corresponding fire danger index ranges

Fire Danger Rating	Fire Danger Index Range
Low-Moderate	0 - 11
High	12 - 31
Very High	32 - 49
Severe	50 – 74
Extreme	75 - 99
Catastrophic	100 and greater

[6] http://www.bom.gov.au/weather-services/fire-weather-centre/fire-weather-services/
[7] DFES can modify the FDR based on other meteorological factors (such as dry lightning) and other non-meteorological factors.

Weather Station Data: Weather data such as temperature and humidity were sourced from the BOM from seven weather stations in the Perth metropolitan area. The weather data as additional adjustment risk factors were used also in the smoke health impact analysis. The weather data of each SA2s (totally 174 SA2s in the study area) were calculated using IDW method.

Table 3. Perth metropolitan weather stations used for the study

Station ID	Station Name	Latitude	Longitude
9053	Pearce	-31.67	116.02
9021	Perth airport	-31.93	115.98
9225	Perth city	-31.92	115.87
9240	Bickley	-32.01	116.14
9977	Mandurah	-32.52	115.71
9172	Jandakot	-32.10	115.88
9215	Swanbourne	-31.96	115.76

C. Geographically weighted ridge regression (GWRR)

In addition to the IDW method described above, geographically weighted ridge regression (GWRR) method was also used to explore spatial heterogeneity in the relationship between fine particulate matter and known covariates of air pollution. GWRR is a local regression technique that reduces the impact of multicollinearity in predictors on estimates of regression coefficients (Wheeler, 2007). GWRR was preferred to GWR (Brunsdon et al., 2007) due to its useful feature of penalising regression coefficients to minimise the adverse impact of multicollinearity on model parameter estimates.

In this study, a variant of GWRR known as the locally compensated ridge geographically weighted (LCR-GW) regression was used, and processing has been done in the R software package (Lu et al., 2014). In the LCR-GW approach, in contrast to other implementations of GWRR, local compensation of regression (or ridge regression) is only performed when a pre-specified local condition number (CN) threshold is exceeded. The condition number is a measure of the extent to which local data is impacted by multicollinearity, with higher values implying higher levels of multicollinearity. The LCR-GW method uses a local ridge parameter (λ) instead of a global one. The lambda (λ) parameter represents the amount of penalty applied to local regression coefficients. In this study, a condition number threshold of 30 was used, meanwhile local lambdas were estimated adaptively via cross validation. Choice of distance bandwidth, that controls the level of smoothing in estimates, is an important aspect of geographically weighted regression. Here, we used an adaptive Gaussian kernel of size equal to the total number of observations (~ 174 km). A large bandwidth size was chosen to guard against model failure due to spatial clustering of categories of the categorical predictors. The choice of a large bandwidth is consistent with that of related studies (Fann et al., 2018, Lassman et al., 2017).

Overall, we found the results from the LCR-GW were similar to those from the IDW method. The full results from the IDW method are presented and explained in the main body of the report; and the results from the LCR-GW method are presented in the Appendix GWRR Statistical Analysis Results section without further explanation but to demonstrate the validity of the analyses.

2.3 Step 3: Air quality relationship assessment (model fitting)

We applied to the empirical model results from training cells from Step 2 to the prediction cells and further validate the model and assess the suitability of the model in this step.

2.3.1. Exposure modelling and validation

We first used simple linear regression to assess the associations between the $PM_{2.5}$lag0 (or PMlag0) response variable and each of the six potentially predictive covariates ($PM_{2.5}$lag1, AOD, FRP, SPM, FDR, and VI) in the training cells (3,660 records). Next, multiple linear regression models shown below were fitted with all potentially predictive covariates that were associated with $PM_{2.5}$lag0 in the multiple linear regressions, using a forward stepwise approach to maximise the adjusted R-squared of the model. In our model, all covariates were significantly associated with an increased $PM_{2.5}$lag0.

$PM_{2.5}$lag0~ $PM_{2.5}$lag1 + SPM+AOD+ FRP+VI+FDR

In addition, we tried to use a similar approach as Yao and Henderson (2014) and selected cells with FRP values in three categories for the degree of smokiness: i) greater than the 80th percentile to form a training dataset (i.e., high-smoke days), ii) the medium (FRP within 40th to 60th percentile) and iii) low (FRP less than 20th percentile) smoke days. We then used the training dataset to generate prediction models and assessed each model's goodness of fit by measuring the root mean square error (RMSE). The smaller RMSE, the better goodness of fit is. Because the high-smoke days were used to train the model, we also used a leave-one-year-out approach to evaluate its performance. This leave-one-year-out approach has been used in other researches in this area including in Yao exposure modelling (Yao and Henderson, 2014). However due to small sample size, we could not obtain meaningful results from this sampling method.

Therefore, we used a sampling method which was different from Yao and Henderson (2014) and conducted a random sample to obtain 80% of the whole sample in the training cells (2,928 out of 3,660 records). Multiple regression models were then fitted with all potentially predictive covariates that were associated with the $PM_{2.5}$lag0 in those 80% sampling records in the training database. All covariates were significantly associated with the $PM_{2.5}$lag0. We then applied the model from the 80% sample of station based $PM_{2.5}$ measures to the remaining 20% and examined the RMSE to assess model performance. Goodness of fit statistics including Pearson correlation coefficients with the observed station-based measurements was also calculated. The RMSE is a measure of the average difference between predicted and observed $PM_{2.5}$ values, with smaller RMSE values indicating better prediction accuracy. With a satisfactory goodness of fit, we then applied the model from 80% of the sample to 100% of the station-based sample in training cells (3,660 records).

In preliminary analyses, we found that the highest $PM_{2.5}$lag0 original observed value was 67.70 mg/m3 and a small proportion (5.17%) of predicted values were greater than 67.70 mg/m3, thus all predicted $PM_{2.5.}$ over 67.70 mg/m3 were set to 67.70 mg/m3 for fitting the full data model (training and prediction). This assisted to produce a more reliable model as we did not have these values in the original $PM_{2.5}$. We also used a leave-one-year-out approach to evaluate its performance in this range where the model established with the July 2015-December 2016 data was applied to the 2017 data to assess the suitability of the model.

In our study sample, the 80% sample produced a model with the best goodness of fit. Therefore, we selected that model for the final 100% sample analysis in training cells and evaluated the predicted smoke related $PM_{2.5}$lag0 with the original observed $PM_{2.5}$lag0. We then applied the final 100% sample model derived from training cells (full training cells) to the full database (3,649,935 records). Evaluation of the model in full database produced the adjusted R-squared (R^2) =0.207 with a root mean square (RMSE) of 1.78 (Table 4). Table 4 presents the model evaluation results for the selected final models.

Table 4. Model evaluation results

Dataset	Statistical Performance			Difference (Pred-Obs)		Obs $PM_{2.5}$	
	R^2	Corr	RMSE	Median	Mean	Median	Mean
Training cells (100%)	0.245	0.495	3.140	0.4	0	7.46	8.090
Full database (100%)	0.207	0.455	1.780	-2.1	-2.51	5.82	6.320

Pred=predicted, Obs=observed, Corr=Correlation, RMSE= root mean square error. Training cells (100%) =3,660 records; Full database (100%) = 3,649, 935 records.

2.3.2. Modelling results for training grid cells

The total number of observations in training model was 3,660 records. Simple linear regressions between PMlag0 and each of the six potentially predictive covariates all resulted in significant associations at the P<0.05 level (Table 5). The $PM_{2.5}$lag1 had the highest variance explained (R^2=0.175), followed by VI (R^2=0.036), FRP (R^2=0.031), AOD (R^2=0.015), FDR (R^2=0.006) and SPM (R^2=0.002). The best multiple regression model included all candidate variables (Table 5). The model explained a total of 24% variance for the PMlag0 values. The mean (standard deviation) daily $PM_{2.5}$ concentration was 8.09 (3.6) mg/m^3, which was similar with the mean of smoke related $PM_{2.5}$ in predicted training model of 8.08 (1.8).

Table 5. Multiple linear regression results of predictors of smoke related $PM_{2.5}$ (Final training model summary)

Predictor	Estimate	SE[a]	T Value	P value	Importance[b]
$PM_{2.5}$lag1[c]	0.380	0.040	7.790	<0.001	0.175
AOD[d]	4.404	0.860	5.110	<0.001	0.015
FRP[e]	1.760	0.460	3.790	<0.001	0.031
FDR[f]	0.524	0.080	6.550	<0.001	0.006
SPM[g]	0.990	0.440	2.220	0.026	0.002
VI[h]	0.830	0.050	16.040	<0.001	0.036

N=3,660, R square = 0.245, RMSE= 3.14; [a] SE=standard error; [b]Importance calculated as the proportion of variance explained attributable to the variable; [c]Previous day $PM_{2.5}$ (mg/m^3) based on IDW method at grid cell level; [d]Aerosol Optical Depth index value at grid cell level; Fire Radiative Power in Gigawatts at grid cell level; [f]Fire Danger Rate at grid cell level; [g]Smoke Plume Mask at grid cell level; [h]Venting Index (m^2/s) value at grid cell level

2.3.3. Modelling results for training and prediction cells (full database)

The total number of observations in both training and prediction cells (full database) was 3,649,935 records. Simple linear regressions between $PM_{2.5}$lag0 and each of the six potentially predictive covariates all resulted in significant associations at the P<0.05 level. The best multiple regression model included all candidate variables (Table 6). The $PM_{2.5}$lag1 had the highest variance explained (R2=0.397), followed by FRP (R2=0.168), SPM (R2=0.059), AOD (R2=0.049), FDR (R2=0.012), and VI (R2=0.000). PMlag1 was the most important variable for explaining the variance in $PM_{2.5}$, followed by FRP, SPM, AOD, FDR, and VI. The mean (standard deviation) daily smoke related $PM_{2.5}$ was 6.32 (2.8) mg/m3.

Table 6. Multiple linear regression results of predictors of smoke related PM2.5 (Final full data summary)

Predictor	Estimate	SE[a]	T Value	P value	Importance[b]
$PM_{2.5}$lag1[c]	0.410	0.000	793.580	<0.001	0.397
AOD[d]	4.960	0.020	170.150	<0.001	0.049
FRP[e]	0.140	0.000	24.330	<0.001	0.168
FDR[f]	0.524	0.080	6.550	<0.001	
SPM[g]	2.870	0.020	99.620	<0.001	0.059
VI[h]	0.830	0.000	27.030	<0.001	0.036

N= 3,649, 935, R square = 0.606, RMSE= 1.78; a SE=standard error; b Importance calculated as the proportion of variance explained attributable to the variable; c Previous day PM2.5 (mg/m3) based on IDW method at grid cell level; d Aerosol Optical Depth (a unitless measure) index value at grid cell level; e Fire Radiative Power in Gigawatts at grid cell level; f Fire Danger Rate (a numeric index between 0 to 100) at grid cell level; g Smoke Plume Mask (whether smoke plume covered the centroid of a grid cell: 0=no covered and 1=covered) at grid cell level; h Venting Index (m2/s) value at grid cell level.

2.3.4. Assigning the exposure estimates to Statistical Area level 2 (SA2) geographical units

We calculated the median of $PM_{2.5}$ values from the gridded cells corresponding to a specific SA2. The database with SA2 codes and the predicted smoke related $PM_{2.5}$ was used for health data analysis in Section 2.4.

2.3.5. Data analysis (exposure modelling and validation)

In model fitting, we firstly adopted the regression model used in Yao and Henderson (2014). Our air pollution model comprised $PM_{2.5}$ for the day ($PM_{2.5}$lag0) as the outcome variable, and six explanatory variables: three categorical variables (i.e. SPM, VI and FDR) and three continuous variables (i.e. the first lag of $PM_{2.5}$ ($PM_{2.5}$lag1), FRP and AOD.

2.4 Step 4: LF smoke-related PM2.5 and health utilisation relationship assessment

2.4.1. Objectives

There were two objectives for Step 4 of the LF project.

1) To estimate the association between exposure to smoke related $PM_{2.5}$ and selected adverse health outcomes including cardiovascular diseases (CVD) and respiratory diseases for hospitalisations, emergency department attendances (EDA), and ambulance callouts (AC).
2) To estimate lag effects of 1 to 3 days between exposure to smoke related $PM_{2.5}$ and selected adverse health outcomes including hospitalisations due to CVD and respiratory diseases for hospitalisations, EDA and AC as the smoke impacts might occur on the same day of a LF event, or 1 to 3 days after a LF event.

2.4.2. Study design, setting and participants

A population-based time series design was used to estimate the association between daily exposures to smoke-related $PM_{2.5}$ with the daily health outcome rates. Time series designs are suitable for environmental epidemiological studies investigating short term associations between exposure variables and health outcomes.

We constructed a daily time series by gender, age group (0-14, 15-60 and 60+ years) and statistical area level 2 (SA2) for the Perth metropolitan area from 1 July 2015 to 31 December 2017.

2.4.3. Data preparation (health data)

We used de-identified (anonymised) information on the selected health outcomes using International Classification of Diseases -10th Revision - Australian Modification (ICD-10-AM) codes to classify admissions by principal diagnosis of interest (Table 7). The ambulance data had its own specific disease codes.

Table 7. ICD-10-AM codes or problem codes used for identifying conditions for the project

Heath Outcome	ICD-10-AM Codes (EDA & Hospitalisation) Ambulance (Problem codes)
Respiratory (EDA) Respiratory (Hosp) Respiratory (AC)	MDB=3B or MDC=4 J00–J99 Probcode=240-249
Asthma (EDA) Asthma (Hosp) Asthma (AC)	J45-J46 J45-J46 Probcode=241
Acute Lower Respiratory Tract Infections (EDA) Acute Lower Respiratory Tract Infections (Hosp) Respiratory Tract Infections (AC)	J20- J22, J10-J11, J16.8, J17-J18, J12, J15-J16, J1.4, J1.3, A48.1-A48.2, B59, J20- J22, J10-J11, J16.8, J17-J18, J12, J15-J16, J1.4, J1.3, A48.1-A48.2, B59, Probcode=245
Chronic Obstructive Pulmonary Disease (ED) Chronic Obstructive Pulmonary Disease (Hosp) Chronic Obstructive Pulmonary Disease (AC_Not Available)	J40-J44, J47 J40-J44, J47 -
Croup (ED) Croup (Hosp) Croup (AC_Not Available)	J05.0 J05.0 -
Respiratory Arrest (EDA_Not Available) Respiratory Arrest (Hosp_Not Available) Respiratory Arrest (AC)	- - Probcode=248
Cardiovascular (EDA) Cardiovascular (Hosp) Cardiovascular (AC)	MDB=3A or MDC=5 G45, I00-I99 Probcode= 312-419
Arrhythmia (EDA) Arrhythmia (Hosp) Arrhythmia (AC)	I44-I45, I47, I48.0-I48.4, I48.9, I49, R00, R94.3 I44-I45, I47, I48.0-I48.4, I48.9, I49, R00, R94.3 Probcode=417
Angina (EDA) Angina (Hosp) Angina (AC)	I20 I20 Probcode=412
Stroke (EDA) Stroke (Hosp) Stroke (AC)	I60-I64 I60-I64 Probcode=312
Heart Failure (EDA) Heart Failure (Hosp) Heart Failure (AC_Not Available)	I50 I50 -
Acute Coronary Syndrome (EDA) Acute Coronary Syndrome (Hosp) Acute Coronary Syndrome (AC_Not Available)	I21-I22 I21-I22 -

Heath Outcome	ICD-10-AM Codes (EDA & Hospitalisation) Ambulance (Problem codes)
Transient Ischemic attack (EDA) Transient Ischemic attack (Hosp) Transient Ischemic attack (AC_Not Available)	G45 G45 -
Cardiac Arrest (EDA_Not Available) Cardiac Arrest (Hosp_Not Available) Cardiac Arrest (AC)	- - Probcode=418

EDA: emergency department attendance data; Hosp: hospitalisation data; AC: ambulance callout data. Probcode: problem code for ambulance data (paramedic-determined on scene); MDB: Major Diagnostic Block; MDC: Major Diagnostic Category.

These data were sourced from three administrative data sets in Western Australia including:

1) Daily EDA count of total and selected respiratory diseases and CVD from the WA Emergency Department Data Collection (EDDC). Respiratory diseases include asthma, acute lower respiratory tract infections, chronic obstructive pulmonary disease (COPD), and croup. Cardiovascular diseases include arrhythmia, angina, stroke, heart failure, acute coronary syndrome, and transient ischemic attack.
2) Number of total hospital admissions and admissions due to selected respiratory diseases and CVD from the WA Hospital Morbidity Data Collection. Respiratory diseases include asthma, acute lower respiratory tract infections, COPD, and croup. Cardiovascular diseases include arrhythmia, angina, stroke, heart failure, acute coronary syndrome, and transient ischemic attack. Only admissions to hospitals in the metropolitan area classified as emergency admissions were included and elective admissions were excluded from the analysis.
3) Daily AC due to CVD and respiratory disease from WA St John Ambulance Data Collection.

2.4.4. Air quality data (exposure measurement)

As described in Section 2.2, gridded daily average exposure estimates for $PM_{2.5}$ were produced at a resolution of 1.5km x 1.5km by adapting the empirical model of Yao and Henderson (Yao and Henderson, 2014) to estimate of daily fire smoke exposure over the Perth metropolitan area using air quality, meteorological and remote sensing data. The model included an estimation of smoke related $PM_{2.5}$ concentrations in populated areas of the Perth Metropolitan area that were not covered by the air quality monitoring network as well as those areas that are covered by air quality monitoring network. The model inputs were multiple sources of data including $PM_{2.5}$lag1 monitor measurements, remotely sensed fire radiative power, aerosol optical depth, smoke plume images, fire danger rating, and a venting index that indicated pollutant dispersion potential.

The values for estimated $PM_{2.5}$ concentrations were categorized into three levels based on their distribution percentiles, i.e., low (<=95th percentile, i.e. <=8.73), middle (96th-98th percentile, >8.73 and <12.60) and high level (>=99th percentile, i.e. >=12.60). In brief, smoke events were defined as those days when the $PM_{2.5}$ was equal or exceeded the 99th percentile of the entire time series. The 99th percentile was chosen as it corresponds closely to Australian air quality standards and enables a clear delineation between background and bushfire days. The 99th percentile cut off has also been used in other Australian studies investigating the effect of bush fires on health outcomes (Johnston et al., 2011, Johnston et al., 2014).

2.4.5. Population data

Population data for a statistical area level 2 (SA2 level) estimated resident populations by age group, gender and SA2 for the Perth metropolitan area were sourced from the Australian Bureau of Statistics. The monthly populations were computed using a linear interpolation method, interpolated based on mid-year and such populations were then applied to all the days in the month. The daily estimates then served as the estimated populations at risk for calculation of rates in the analysis. For the ambulance callouts database, the population at risk was defined to be WA residents with a residential postcode within the Perth metropolitan area. The population counts were sourced from the estimated resident population from the Australian Bureau of Statistics for the study period.

2.4.6. Potential confounders and effect modifiers

We considered other independent risk factors as potential confounding factors in the statistical analysis. These factors are as below.

- Age where we categorised it into three levels (0-14, 15-59 and >60 years), where the age group of 15-59 was considered as the reference group in the analysis.
- Sex (Male, Female), where males were considered as the reference group in the analysis.
- Socioeconomic status was measured by Socio-Economic Index for Area (SEIFA) which is a product developed by the ABS that ranks areas in Australia according to relative socio-economic advantage and disadvantage. The SEIFA indexes are based on information from the five-yearly Census. We used the SEIFA 2011 version, and categorised it based on three levels (advantaged, middle, and disadvantaged), where we used the advantaged group as the reference group in the analysis.
- Meteorological data: Australian Bureau of Meteorology provided daily average ambient temperature and humidity, as measured by dew point temperature calculated using the formula below.

Dew temperature (in ºC) = observed temperature (in ºC) - (100- relative humidity (in %)/5)

More details can be found in Lawrence et al (Lawrence, 2005). The values for dew point temperature, were categorized into three levels based on their distribution percentiles, i.e., low (<95th percentile, i.e. <16.32), middle (95th-97th percentile, i.e. >=16.32 &<17.96) and high level (>=98th percentile, i.e. >=17.96). The dew point is the temperature to which air must be cooled to become saturated with water vapour. When further cooled, the airborne water vapor will condense to form liquid water (dew). The measurement of the dew point is related to humidity. A higher dew point means there is more moisture in the air. In normal conditions, the dew point temperature will not be greater than the air temperature since relative humidity cannot exceed 100%. For example, at a higher dew point of, around 70F (210C), most people feel hot or sticky because the amount of water vapour in the air shows the evaporation or perspiration and keeps the body from cooling (https://www.e-education.psu.edu/meteo3/l4_p7.html). The 98th percentile was chosen as it corresponds closely to 21C 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.

Humidity were classified as low (<95 percentile, i.e. <87.32), medium (95th-98th percentile, i.e. >=87.32 &<92.25) and high level (>=99th percentile, i.e. >=92.25).

As described in Step 2, the inverse distance weighted averages of all valid weather station observations were calculated.

- Residential wood fire use level: we categorised it into three levels of wood fire use based on month (infrequent = December, January, February and March; less frequent = April, October and November; frequent = May – September in a year), where we considered the infrequent use as the reference group in the analysis.
- Seasonal factors include holiday (public holiday, not public holiday), weekend (weekday, weekend) and season (summer, autumn, winter, spring). Public and school holidays for the Perth region were collated from a comprehensive listing of events, diaries, and calendars.

2.4.7. Statistical analysis

We conducted univariate and multivariate Poisson Regressions to assess the health risks associated with the smoke related $PM_{2.5}$. In the multivariate regression models, we included all confounding factors as described above including socio-demographic factors such as age, gender, SEIFA, seasonal factors such as public holiday, weekend, season and other environmental exposures such as dew point temperature, humidity and wood fire use period to assess the potential health risks associated with $PM_{2.5}$.

The multivariate models were run on lag effects of smoke related $PM_{2.5}$ concentrations on the same day (lag0), 1 day (lag1), 2 days (lag2) and 3 days (lag3) after a LF event. For instance, cumulative 3-day data was the sum of current day count and population with subsequent two days' counts and population. The model with the greatest risk ratio (RR) for the variable smoke-related $PM_{2.5}$ was considered as the best model where the strong lag effect of smoke-related $PM_{2.5}$ was demonstrated. RR was calculated to assess the difference in health outcome measures associated with smoke related $PM_{2.5}$. The comparison between different levels for a risk factor was considered as statistically significant if the P-value is less than 0.05. For brevity, we used the term 'significant' indicates a difference was statistically significant. The data analysis was conducted using SAS Enterprise Guide 5.1.

2.4.8. Ethics approval

This study was approved by the Western Australia Department of Health Human Research Ethics Committee (No. 2018/31), the Curtin University Ethics Committee (No. HRE2019-0029) and WA St John Ambulance Research Advisory Group.

3. Results

The total number of ED attendances, hospital admissions and ambulance callouts for the study were 1 543 222, 535 175 and 78 441, respectively. The number and percentage of records for each health condition, environmental exposures, socio-demographic factors, and seasonal factors are presented in Table 8.

Table 8. Number (N) and Percentage (%) of Socio-Demographic Characteristics, Environmental Exposures, and Health Outcomes for Study Participants, Perth Metropolitan Area, WA, July 2015-2017

Health Outcomes	EDA[a]		Hosp[b]		AC[c]	
	N	%	N	%	N	%
Total Number	1543222	100	535175	100	78441	100
Respiratory	114160	7.40	54995	10.28	33171	42.29
Asthma	10196	0.66	4370	0.82	2950	3.76
Chronic Obstructive Pulmonary Disease	10902	0.71	11686	2.18		
Acute Lower Respiratory Tract Infections	36859	2.39	24898	4.65	11417	14.55
Croup	10322	0.67	1008	0.19		
Respiratory Arrest					431	0.55
Cardiovascular	130466	8.45	56879	10.63	45598	58.13
Arrhythmia	11515	0.75	10537	1.97		
Dysrhythmia					8219	10.48
Heart Failure	6745	0.44	10037	1.88		
Acute Coronary Syndrome	6447	0.42	8367	1.56		
Angina	6263	0.41	5050	0.94	611	0.78
Stroke	2194	0.14	4659	0.87	3581	4.57
Transient Ischemic Attack	3886	0.25	2554	0.48		
Cardiac Arrest					3914	4.99
Socio-Demographic						
Age						
0-14	376554	24.40	71931	13.44	3769	4.80
15-59	798877	51.77	225701	42.17	19661	25.06
60+	367791	23.83	237543	44.39	55011	70.13
Gender						
Male	768766	49.82	259862	48.56	39457	50.30
Female	774456	50.18	275313	51.44	38984	49.70
SEIFA						
Disadvantaged	526956	34.15	179797	33.60	26888	34.28
Middle	313968	20.34	112983	21.11	18444	23.51
Advantaged	702298	45.51	242395	45.29	33109	42.21

Health Outcomes	EDA[a]		Hosp[b]		AC[c]	
	N	%	N	%	N	%
Environmental Exposures						
Smoke-related $PM_{2.5}$						
No smoke/Low (<8.73ug/m3)	1465782	94.98	508463	95.01	74709	95.24
Medium (8.73 -12.60ug/m3)	61400	3.98	21338	3.99	2988	3.81
High (≥12.60ug/m3)	16040	1.04	5374	1.00	744	0.95
Woodfire						
Infrequent	459767	29.79	156916	29.32	20419	26.03
Less frequent	409684	26.55	141581	26.46	20016	25.52
Frequent	673771	43.66	236678	44.22	38006	48.45
Dewpoint Temperature (^{0}C)						
Low (<16.32°C)	1465086	94.94	508674	95.05	74854	95.43
Medium (16.32 -17.96°C)	46680	3.02	15410	2.88	2119	2.70
High(>17.96°C)	31456	2.04	11091	2.07	1468	1.87
Relative humidity (%)						
Low (<87.32)	1497554	97.04	519276	97.03	75991	96.88
Medium (87.32-92.25)	30707	1.99	10789	2.02	1768	2.25
High (>92.25)	14961	0.97	5110	0.95	682	0.87
Seasonal and holiday Factors						
Holiday						
Not Holiday	1496078	96.95	522091	97.56	76469	97.49
Holiday	47144	3.05	13084	2.44	1972	2.51
Weekend						
Weekday	1087816	70.49	403615	75.42	57533	73.35
Weekend	455406	29.51	131560	24.58	20908	26.65
Season						
Summer	353934	22.93	120423	22.50	15729	20.05
Autumn	310212	20.10	107483	20.08	14831	18.91
Winter	414500	26.86	146331	27.34	23603	30.09
Spring	464576	30.10	160938	30.07	24278	30.95

[a]Emergency Department Admissions; [b]Hospital Admission; [c]Ambulance callout.

3.1. Emergency department attendance (EDA)

3.1.1. Association of total number of EDA with LF smoke related $PM_{2.5}$

In the multivariate analysis of all-cause EDA outcomes, we found a dose response relationship on the same day smoke related $PM_{2.5}$ (lag 0), and on all lag effects of 1 to 3 days (Table 9). For example, on the same exposed day, there was a 2% significant increase in EDA where the smoke-related $PM_{2.5}$ was at the medium level (96-98 percentile) and was 5% significant increase in RR where the smoke-related $PM_{2.5}$ was at the high level (>=99 percentile).

In an interaction analysis with age, sex, and SEIFA, we found that the EDA rates significantly increased in those aged >=60 years risk in all lag effects. There was also an increased risk in lag 3 for those in disadvantaged groups.

Table 9. Multivariate Poisson Regressions for the Association between Emergency Department Attendance (all-cause) and LF Smoke-related PM2.5 for Same Day (Lag0) and lags of 1 to 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)	1.00				1.00				1.00				1.00			
96-98 (Med)	1.02	1.01	1.03	<.0001	1.02	1.01	1.03	<.0001	1.02	1.01	1.02	<.0001	1.01	1.01	1.02	<.0001
> = 99 (High)	1.05	1.03	1.06	<.0001	1.04	1.03	1.05	<.0001	1.03	1.02	1.04	<.0001	1.03	1.03	1.04	<.0001
Dewpoint Temperature																
<=95 (Low)	1.00				1.00				1.00				1.00			
96-97 (Med)	1.03	1.02	1.04	<.0001	1.03	1.02	1.04	<.0001	1.02	1.01	1.03	<.0001	1.02	1.01	1.02	<.0001
>=98 (High)	1.03	1.02	1.04	<.0001	1.02	1.01	1.03	<.0001	1.01	1.00	1.02	0.0015	1.01	1.000	1.011	0.0137
Wood Fire																
Infrequent	1.00				1.00				1.00				1.00			
Less frequent	0.98	0.97	0.99	<.0001	0.98	0.97	0.98	<.0001	0.98	0.97	0.98	<.0001	0.98	0.97	0.98	<.0001
Frequent	1.00	0.99	1.01	0.9911	1.00	1.00	1.01	0.5246	1.00	1.00	1.01	0.9027	1.00	0.99	1.00	0.5712
Socio-Demographic																
Age																
15-59	1.00				1.00				1.00				1.00			
0-14	0.65	0.65	0.65	<.0001	0.65	0.65	0.65	<.0001	0.65	0.65	0.65	<.0001	0.65	0.65	0.65	<.0001
60+	1.01	1.01	1.02	<.0001	1.01	1.01	1.02	<.0001	1.01	1.01	1.02	<.0001	1.01	1.01	1.02	<.0001

	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
Gender																
Male	1.00				1.00				1.00				1.00			
Female	1.00	1.00	1.01	0.2278	1.00	1.00	1.00	0.0847	1.00	1.00	1.00	0.0333	1.00	1.000	1.000	0.0119
SEIFA																
Advantaged	1.00				1.00				1.00				1.00			
Middle	1.19	1.18	1.19	<.0001	1.19	1.18	1.19	<.0001	1.19	1.18	1.19	<.0001	1.19	1.18	1.19	<.0001
Disadvantaged	1.50	1.49	1.50	<.0001	1.50	1.49	1.50	<.0001	1.50	1.49	1.50	<.0001	1.50	1.49	1.50	<.0001
Seasonal factors																
Holiday																
Not Holiday	1.00				1.00				1.00				1.00			
Holiday	1.09	1.08	1.10	<.0001	1.09	1.09	1.10	<.0001	1.07	1.06	1.08	<.0001	1.05	1.05	1.06	<.0001
Weekend																
Weekday	1.00				1.00				1.00				1.00			
Weekend	1.05	1.04	1.05	<.0001	1.07	1.06	1.07	<.0001	1.06	1.05	1.06	<.0001	1.03	1.03	1.03	<.0001
Season																
Summer	1.00				1.00				1.00				1.00			
Autumn	1.02	1.02	1.03	<.0001	1.02	1.02	1.03	<.0001	1.02	1.02	1.03	<.0001	1.03	1.02	1.03	<.0001
Winter	1.03	1.02	1.04	<.0001	1.03	1.02	1.04	<.0001	1.03	1.03	1.04	<.0001	1.03	1.03	1.04	<.0001
Spring	1.05	1.04	1.06	<.0001	1.05	1.04	1.05	<.0001	1.05	1.04	1.05	<.0001	1.05	1.04	1.05	<.0001

*Adjusted for all variables in the table and Humidity.

3.1.2. Association of total number of EDA with other risk factors

There was a 3% significant increase for the association between EDA with dew point temperature at both medium and high levels compared to low level dew point temperature. The pattern of association of EDA rates by dew point temperature was also significant on all lag effects of 1to 3 days (Table 9).

EDA were less frequent in children 1 to 14 years age compared to adults 15-59 years age. There was no significant difference between males and females attending the ED department.

There was a dose response relationship for socio-economic status of participants measured as SEIFA in this study, where middle and disadvantaged people had significantly 19% and 50% increase in RR compared with advantaged people, respectively. The pattern of association of EDA rates by SEIFA was also significant on all lag effects of 1 to 3 days.

There were also significant increases of 9% and 5% in those attending EDA in public holidays and weekend, respectively. The pattern of associations of EDA rates by public holidays and weekend were also both significant on all delayed lag effects of 1to 3 days.

There were 2%, 3%, and 5% significant increases in EDA in autumn, winter and spring respectively as compared to summer. These patterns of associations of EDA rates by season were all also significant on all delayed lag effects of 1to 3 days.

3.1.3. Association of EDA for selected adverse respiratory health outcomes with smoke related $PM_{2.5}$

Our study indicated that there was 1% to 19% increased risk of EDA for individuals with respiratory diseases including asthma (non-significant increase on all lag effects with dose-response effect), and acute lower respiratory tract infections (significant increase on lags 1 to 3 at high level smoke with dose-response effects). See the Table 10 and Figure 3 for the full results.

In an interaction analysis with age, sex, and SEIFA, we found that the EDA rates on lag0 for asthma significantly increased about 85% in disadvantaged areas. There was also 20% increased risk of EDA due to acute lower respiratory tract infections in lag3 for those in disadvantaged groups.

3.1.4. Association of EDA for selected adverse cardiovascular health outcomes with smoke related $PM_{2.5}$

There was a significant 5% to 7% increase at high level smoke with dose response effect on the same day smoke related $PM_{2.5}$ (lag 0), and on all lag effects of 1 to 3 days for total cardiovascular health outcomes. There was 1% to 25% increased risk for individuals with underlying cardiovascular diseases including transient ischemic attack (significant increase at high level smoke in lag 1 & 2 with dose-response effect), and Acute Coronary Syndrome (significant increase on lag0 at medium level and non-significant increase in lag1). See the Table 10 and Figure 3 for the full results.

In an interaction analysis with age, sex and SEIFA, we found that the ED attendance rates on all lag effects for total cardiovascular diseases significantly increased about 5% to 8% in those aged above 60 years and above.

3.1.5.Temporal and spatial distribution of smoke related $PM_{2.5}$ on EDA rates

Figure 3 shows the SA3-based geographical variations of the effects of smoke-related $PM_{2.5}$ on the ED presentation rates by season. Maps in Figure 4 were created based on the results of rate ratio values via the assessment of interaction effects between smoke-related $PM_{2.5}$ categories and SA3 on ED attendance rates after adjusting for age, sex, SEIFA, public holiday, weekend, season, wood fire, geographical areas and temperature dew point in Poisson regression models. In deriving crude ED attendance rates by SA3, we found that the Claremont-Cottesloe areas had the lowest ED attendance rates that were associated with smoke-related $PM_{2.5}$ in all 4 seasons. Thus, we selected this area as the reference group when comparing with other SA3 areas in joint effect analysis (interaction effect).

In the Poisson regression models, for each SA3, the ED attendance rate ratio (RR) between high levels (ie, high and medium) and low level of $PM_{2.5}$ was first calculated, resulting two RRs for comparing high versus low and medium versus low level of $PM_{2.5}$. The ratio between the RR (high or medium) for each SA3 and the respective RR for the Claremont-Cottesloe areas was then computed, resulting in a rate ratio of two RRs that reflected how many times the RR in each SA3 was compared to that of the Claremont-Cottesloe areas for respective $PM_{2.5}$ levels. Such a RR was used to indicate the geographical variation of the pure effects of $PM_{2.5}$.

As shown in Figure 4, most areas with high level smoke related $PM_{2.5}$ had a higher risk of ED presentation when compared with low level. In Season 1 (December to February), all areas had higher rates except Wanneroo, Gosnells and Swan, although the results were not statistically significant. In season 2 (March to May), most areas had lower rates except Armadale, Mundaring, Belmont-Victoria park, and South Perth, although the results were not statistically significant. Wanneroo had statistically significant lower rates. In season 3 (June to August), all areas had higher rates except South Perth, but the results were statistically significant for Stirling only. In Season 4 (September to November), all areas had higher rates except Kwinana and Serpentine-Jarrahdale, although the results were only statistically significant for Armadale, Wanneroo, Fremantle, and Kalamunda.

There were no significant differences between geographical areas when we compared medium level smoke related $PM_{2.5}$ with low level. In Season 1, the results were statistically significant for Armadale, Kwinana, Mandurah, Wanneroo, Rockingham, Gosnells, Stirling, and Perth City.

In Season 2, the result for Kalamunda was significant. In Season 3, the results for Armadale, Mandurah, Serpentine-Jarrahdale, Wanneroo, Rockingham, Stirling, Cockburn, and Canning were significant. In Season 4, the result was only significant for South Perth.

Figure 4 shows the distribution of fires by fire type, month and season over the study period. The largest number of wild fires 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), although, the number of wildfires appeared to have decreased from September 2016. This figure helps us interpret differences between WFs and PBs in different seasons

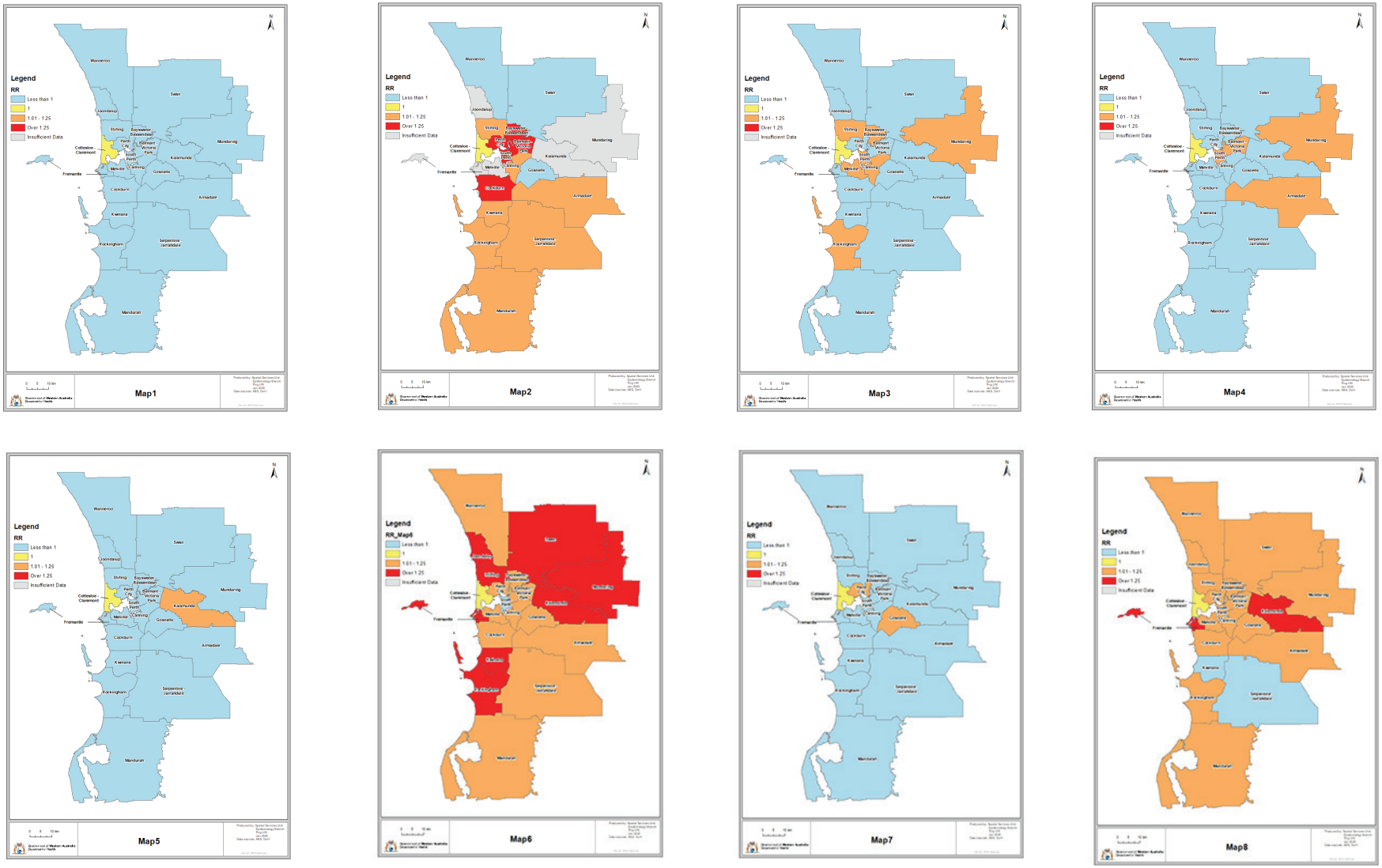


Figure 3. Temporal and spatial distribution of the SA3 based geographical variations of the effect of LF smoke-related $PM_{2.5}$ on the EDA rate, Perth, WA, July 2015-December 2017

(Notes: 1. Adjusted for the age, sex, SEIFA, public holiday, weekend, season, wood fire season, geographical areas and temperature dew point. 2. Effect expressed as risk ratio compared with low level of smoke related PM2.5 exposure. 3. Maps 1, 3, 5 and 7 at the top showing the effect of medium level of smoke related PM2.5 on emergency department attendances for seasons 1 to 4, respectively. 4. Maps 2, 4, 6 and 8 at the bottom showing the effect of high level of smoke related PM2.5 on ED attendances for seasons 1 to 4, respectively.)

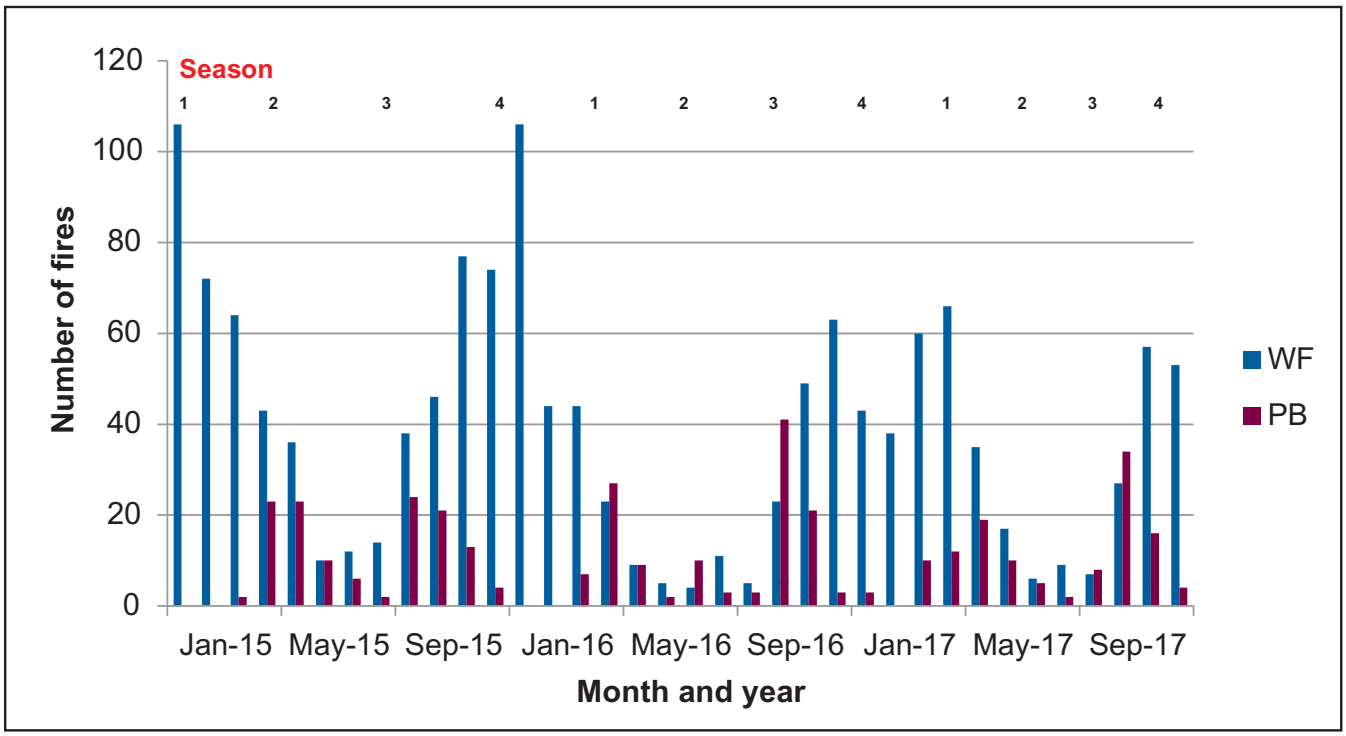


Figure 4. Temporal distribution of monthly fire events, by burn type and season, Perth metropolitan and South West regions, 2015 to 2017, WA

(Season1 = summer, Season 2= autumn, Season 3 = winter, and Season 4= spring)

Table 10. Multivariate Poisson Regressions for the association between health conditions for emergency department attendance (EDA), hospital admissions, and ambulance callout (AC) and LF smoke related $PM_{2.5}$ for same day (lag0) and lags of 1 to 3 days

Health Outcomes	Lag	EDA				Hosp				AC			
		RR[a]	95%CI		P-Value	RR[a]	95%CI		P-Value	RR[a]	95%CI		P-Value
Total	0 (M[b])	1.02	1.01	1.03	<.0001	1.02	1.01	1.04	0.000	0.99	0.95	1.02	0.462
	0 (H[c])	1.05	1.03	1.06	<.0001	1.02	1.00	1.05	0.101	0.94	0.87	1.01	0.097
	1(M)	1.02	1.01	1.03	<.0001	1.02	1.01	1.03	0.001	0.98	0.95	1.00	0.092
	1(H)	1.04	1.03	1.05	<.0001	1.03	1.01	1.05	0.002	0.99	0.94	1.04	0.584
	2(M)	1.02	1.01	1.02	<.0001	1.01	1.00	1.02	0.002	0.98	0.96	1.00	0.127
	2(H)	1.03	1.02	1.04	<.0001	1.02	1.01	1.04	0.003	0.98	0.94	1.02	0.243
	3(M)	1.01	1.01	1.02	<.0001	1.01	1.01	1.02	<0.0001	0.98	0.97	1.00	0.092
	3(H)	1.03	1.03	1.04	<.0001	1.02	1.00	1.03	0.008	0.97	0.94	1.01	0.103
Respiratory (Total)	0 (M)	0.96	0.93	0.99	0.021	0.98	0.94	1.03	0.715	1.00	0.94	1.05	0.869
	0 (H)	0.96	0.90	1.02	0.144	1.02	0.94	1.11	0.579	0.92	0.82	1.02	0.122
	1(M)	0.96	0.94	0.99	0.001	0.98	0.94	1.01	0.127	0.98	0.94	1.02	0.278
	1(H)	0.94	0.91	0.99	0.008	0.97	0.91	1.03	0.255	0.99	0.92	1.07	0.833
	2(M)	0.97	0.95	0.99	0.000	0.97	0.95	1.00	0.055	0.97	0.94	1.00	0.069
	2(H)	0.94	0.90	0.97	0.000	0.96	0.91	1.01	0.100	0.96	0.90	1.03	0.239
	3(M)	0.97	0.95	0.99	0.000	0.97	0.95	1.00	0.026	0.97	0.94	1.00	0.025
	3(H)	0.95	0.92	0.98	0.000	0.96	0.92	1.01	0.092	0.94	0.89	1.00	0.037
Asthma	0 (M)	1.03	0.93	1.14	0.604	1.16	1.00	1.35	0.052	1.11	0.92	1.33	0.270
	0 (H)	1.07	0.89	1.29	0.464	1.06	0.80	1.42	0.403	0.93	0.64	1.35	0.708
	1(M)	1.03	0.96	1.11	0.442	1.05	0.94	1.17	0.410	1.03	0.90	1.17	0.702
	1(H)	1.10	0.96	1.25	0.167	1.18	0.97	1.43	0.101	1.01	0.78	1.30	0.961
	2(M)	1.02	0.96	1.08	0.517	1.02	0.93	1.11	0.737	1.02	0.91	1.13	0.758
	2(H)	1.07	0.96	1.19	0.219	1.16	0.99	1.35	0.072	1.05	0.86	1.29	0.624
	3(M)	1.03	0.98	1.08	0.268	1.02	0.94	1.10	0.674	1.02	0.93	1.12	0.692
	3(H)	1.04	0.94	1.14	0.446	1.12	0.98	1.29	0.098	1.00	0.84	1.20	0.967

Health Outcomes	Lag	EDA				Hosp				AC			
		RR[a]	95%CI		P-Value	RR[a]	95%CI		P-Value	RR[a]	95%CI		P-Value
ALRTI	0 (M)	0.91	0.86	0.96	0.001	0.98	0.91	1.05	0.539	0.98	0.89	1.08	0.704
	0 (H)	0.91	0.82	1.02	0.102	0.98	0.87	1.12	0.800	0.87	0.72	1.06	0.169
	1(M)	1.08	0.99	1.18	0.070	0.98	0.93	1.02	0.324	0.96	0.90	1.04	0.310
	1(H)	1.19	1.10	1.29	<.0001	0.86	0.78	0.94	0.001	0.91	0.80	1.04	0.179
	2(M)	0.93	0.90	0.96	<.0001	0.96	0.93	1.00	0.070	0.96	0.91	1.02	0.210
	2(H)	0.83	0.78	0.89	<.0001	0.87	0.80	0.94	0.000	0.92	0.83	1.03	0.151
	3(M)	1.08	1.02	1.15	0.015	0.97	0.94	1.00	0.063	0.96	0.91	1.01	0.102
	3(H)	1.17	1.10	1.23	<.0001	0.90	0.84	0.96	0.001	0.88	0.80	0.97	0.013
COPD	0 (M)	1.03	0.93	1.13	0.613	1.00	0.91	1.10	0.940				
	0 (H)	0.99	0.82	1.19	0.883	0.96	0.80	1.16	0.685				
	1(M)	1.00	0.94	1.08	0.913	0.98	0.91	1.05	0.524				
	1(H)	0.98	0.86	1.12	0.747	0.98	0.86	1.11	0.727				
	2(M)	1.00	0.94	1.06	0.961	0.98	0.93	1.03	0.451				
	2(H)	0.96	0.86	1.07	0.438	0.99	0.89	1.10	0.845				
	3(M)	0.98	0.94	1.03	0.513	0.98	0.93	1.03	0.362				
	3(H)	0.98	0.89	1.08	0.674	0.96	0.87	1.05	0.330				
Croup	0 (M)	0.86	0.76	0.96	0.006	0.98	0.91	1.05	0.539				
	0 (H)	1.10	0.91	1.33	0.310	0.98	0.87	1.12	0.800				
	1(M)	0.89	0.83	0.96	0.004	0.98	0.93	1.02	0.324				
	1(H)	0.97	0.84	1.11	0.644	0.86	0.78	0.94	0.001				
	2(M)	0.88	0.82	0.94	<.0001	0.96	0.93	1.00	0.070				
	2(H)	0.91	0.81	1.03	0.133	0.87	0.80	0.94	0.000				
	3(M)	0.89	0.84	0.94	<.0001	0.97	0.94	1.00	0.063				
	3(H)	0.89	0.80	0.99	0.029	0.90	0.84	0.96	0.001				

Health Outcomes	Lag	EDA				Hosp				AC			
		RR[a]	95%CI		P-Value	RR[a]	95%CI		P-Value	RR[a]	95%CI		P-Value
Respiratory Arrest	0(M)									1.25	0.78	2.00	0.348
	0(H)									0.26	0.04	1.82	0.173
	1(M)									1.11	0.78	1.57	0.551
	1(H)									0.88	0.42	1.86	0.745
	2(M)									1.07	0.80	1.42	0.669
	2(H)									1.09	0.63	1.89	0.759
	3(M)									1.13	0.89	1.44	0.321
	3(H)									1.20	0.76	1.89	0.433
Cardiovascular (Total)	0(M)	1.02	0.99	1.05	0.154	1.04	1.00	1.09	0.047	0.98	0.93	1.03	0.378
	0(H)	1.07	1.01	1.12	0.019	1.05	0.96	1.13	0.282	0.96	0.88	1.06	0.430
	1(M)	1.02	1.00	1.04	0.051	1.04	1.01	1.07	0.019	0.98	0.94	1.01	0.186
	1(H)	1.06	1.03	1.11	0.001	1.07	1.01	1.13	0.027	0.99	0.92	1.05	0.676
	2(M)	1.01	1.00	1.03	0.118	1.02	1.00	1.05	0.051	0.99	0.97	1.02	0.588
	2(H)	1.06	1.02	1.09	0.001	1.05	1.00	1.10	0.035	0.99	0.94	1.04	0.717
	3(M)	1.01	0.99	1.02	0.230	1.02	1.00	1.04	0.122	1.00	0.97	1.02	0.755
	3(H)	1.05	1.02	1.07	0.001	1.04	1.00	1.09	0.034	1.00	0.95	1.04	0.839
Arrhythmia	0(M)	1.08	0.98	1.18	0.107	1.12	1.02	1.24	0.015	0.94	0.84	1.06	0.327
	0(H)	1.05	0.87	1.26	0.639	1.07	0.89	1.30	0.454	0.82	0.64	1.04	0.100
	1(M)	1.01	0.95	1.08	0.740	1.04	0.97	1.12	0.218	0.93	0.86	1.01	0.082
	1(H)	1.03	0.90	1.17	0.686	1.10	0.96	1.25	0.172	0.92	0.78	1.08	0.319
	2(M)	1.00	0.95	1.06	0.990	1.04	0.98	1.10	0.219	0.93	0.87	0.99	0.027
	2(H)	0.99	0.89	1.11	0.887	1.05	0.94	1.17	0.423	0.91	0.79	1.03	0.138
	3(M)	1.00	0.95	1.05	0.902	1.00	0.95	1.05	0.975	0.95	0.90	1.01	0.075
	3(H)	0.96	0.87	1.06	0.422	1.01	0.92	1.11	0.808	0.95	0.85	1.06	0.326

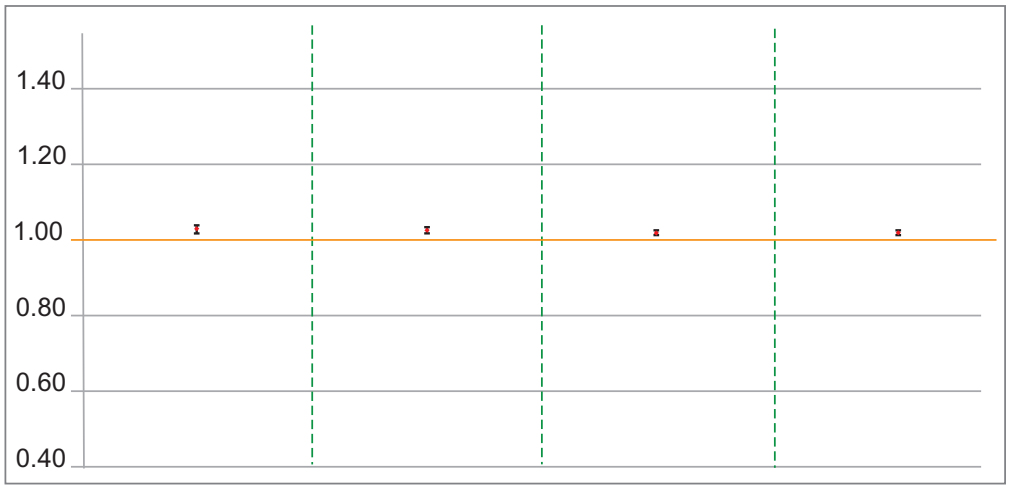
Health Outcomes	Lag	EDA				Hosp				AC			
		RR[a]	95%CI		P-Value	RR[a]	95%CI		P-Value	RR[a]	95%CI		P-Value
Angina	0(M)	0.90	0.78	1.03	0.116	1.07	0.93	1.23	0.355	0.82	0.52	1.30	0.398
	0(H)	0.89	0.68	1.17	0.401	0.83	0.61	1.12	0.225	0.67	0.25	1.79	0.425
	1(M)	0.85	0.77	0.94	0.002	1.06	0.96	1.17	0.284	0.99	0.73	1.33	0.922
	1(H)	0.93	0.77	1.11	0.411	1.06	0.87	1.28	0.584	0.85	0.46	1.58	0.608
	2(M)	0.87	0.80	0.94	0.001	1.03	0.95	1.12	0.495	1.12	0.89	1.41	0.339
	2(H)	0.89	0.76	1.04	0.132	1.12	0.96	1.30	0.151	1.20	0.78	1.85	0.404
	3(M)	0.90	0.84	0.96	0.002	1.04	0.97	1.12	0.282	1.05	0.85	1.29	0.651
	3(H)	0.88	0.77	1.01	0.066	1.11	0.98	1.27	0.113	1.11	0.76	1.64	0.588
Stroke	0(M)	0.97	0.77	1.21	0.754	1.05	0.90	1.21	0.535	0.89	0.74	1.06	0.194
	0(H)	0.79	0.49	1.28	0.346	1.06	0.80	1.41	0.684	0.86	0.61	1.23	0.416
	1(M)	0.92	0.79	1.08	0.327	1.05	0.95	1.17	0.337	0.95	0.84	1.08	0.435
	1(H)	0.93	0.68	1.27	0.642	1.17	0.97	1.42	0.104	0.78	0.60	1.02	0.065
	2(M)	0.93	0.81	1.05	0.246	1.01	0.92	1.10	0.886	1.01	0.92	1.12	0.825
	2(H)	1.06	0.84	1.35	0.623	1.09	0.93	1.28	0.292	0.90	0.73	1.10	0.284
	3(M)	0.96	0.86	1.07	0.469	1.03	0.96	1.11	0.408	1.05	0.97	1.15	0.224
	3(H)	1.04	0.85	1.28	0.690	1.07	0.93	1.24	0.315	0.90	0.76	1.08	0.253
Heart Failure	0(M)	0.92	0.81	1.05	0.231	1.02	0.92	1.13	0.763				
	0(H)	0.87	0.67	1.12	0.286	1.03	0.85	1.25	0.779				
	1(M)	0.93	0.85	1.02	0.161	1.00	0.93	1.07	0.975				
	1(H)	0.89	0.75	1.07	0.242	1.01	0.88	1.16	0.867				
	2(M)	0.95	0.88	1.02	0.182	0.99	0.93	1.05	0.695				
	2(H)	0.92	0.80	1.06	0.301	1.02	0.91	1.14	0.709				
	3(M)	0.96	0.90	1.03	0.317	0.99	0.94	1.04	0.740				
	3(H)	0.93	0.82	1.05	0.256	1.04	0.94	1.14	0.438				
ACS	0(M	1.13	1.00	1.28	0.045	1.01	0.90	1.13	0.849				
	0(H)	1.02	0.80	1.30	0.896	1.04	0.84	1.29	0.696				
	1(M)	1.06	0.98	1.16	0.163	1.03	0.95	1.12	0.447				
	1(H)	1.05	0.89	1.25	0.569	1.05	0.90	1.21	0.559				

Health Outcomes	Lag	EDA				Hosp				AC			
		RR[a]	95%CI		P-Value	RR[a]	95%CI		P-Value	RR[a]	95%CI		P-Value
ACS (continued)	2(M)	1.03	0.95	1.10	0.474	1.03	0.97	1.10	0.323				
	2(H)	1.00	0.87	1.15	0.995	0.95	0.84	1.08	0.433				
	3(M)	1.00	0.93	1.06	0.891	1.01	0.96	1.07	0.638				
	3(H)	1.00	0.88	1.13	0.990	0.98	0.88	1.10	0.756				
TIA	0 (M)	1.01	0.86	1.19	0.876	1.07	0.87	1.30	0.536				
	0 (H)	1.14	0.84	1.54	0.391	1.06	0.72	1.57	0.751				
	1(M)	1.02	0.91	1.15	0.685	1.06	0.92	1.22	0.445				
	1(H)	1.25	1.02	1.53	0.034	1.04	0.79	1.37	0.781				
	2(M)	1.02	0.93	1.12	0.681	1.11	0.99	1.25	0.064				
	2(H)	1.20	1.01	1.42	0.039	1.18	0.95	1.46	0.137				
	3(M)	1.02	0.94	1.11	0.660	1.08	0.98	1.19	0.128				
	3(H)	1.14	0.98	1.32	0.095	1.16	0.97	1.40	0.109				
Cardiac Arrest	0(M)									1.04	0.88	1.22	0.674
	0(H)									0.88	0.63	1.23	0.470
	1(M)									1.01	0.90	1.13	0.871
	1(H)									0.97	0.78	1.22	0.805
	2(M)									1.04	0.95	1.15	0.364
	2(H)									0.93	0.77	1.12	0.437
	3(M)									1.06	0.98	1.15	0.155
	3(H)									0.96	0.82	1.12	0.593

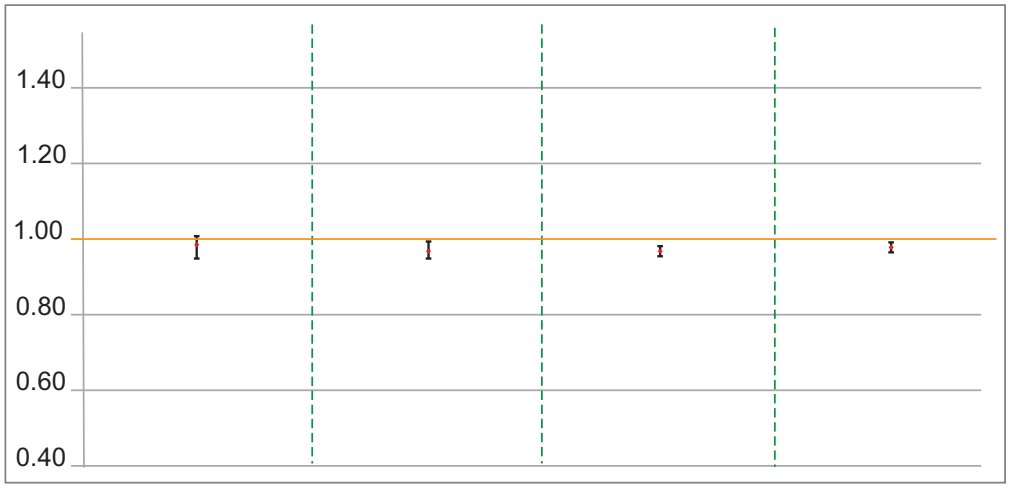
ALRTI = Acute Lower Respiratory Tract Infections; COPD = Chronic Obstructive Pulmonary Disease; ACS = Acute Coronary Syndrome; TIA = Transient Ischemic Attack; a Adjusted for dew temperature, humidity, SEIFA, age, sex, weekend, public holiday, wood fire use &season; [b] M=Medium $PM_{2.5}$; [c]H=High $PM_{2.5}$; [d]Total EDA: Interactions with those aged above 60 years with significant 3% to 5% increase. There was also an interaction with SEIFA in lag3 with disadvantaged group; e Asthma: Interactions with SEIFA with 85% increase at high smoke level for medium disadvantaged group; [f]ALRTI: Interactions with sex with 20% increase at high smoke level for females; [g]Total Cardiovascular: Interactions with age above 60 years old with significant 5% to 8% increase. An interaction analysis in hospital admissions, elderly people had significant 11% to 25% increased risk in total respiratory hospital admissions. Children had significant 36% to 93% increased risk for asthma, significant 31% to 52% increased risk for asthma in disadvantaged group, and about 5 times increased risk for COPD. Disadvantaged group had significant 7% to 12% increased risk in heart failure, and about two-fold increased risk on the same day for angina. An interaction analysis in ambulance callouts (AC), elderly people had significant increased risk of 19% to 45% for total respiratory diseases, 18% to 42% for respiratory infections, 14% for CVD, 75% for angina, and 45% to 2-fold for cardiac arrest. Disadvantaged group had 26% to 72% for total respiratory diseases, 58% to 68% for asthma, 27% to 2-fold increase for respiratory infections, 15% to 25% for CDV total, 59% for cardiac arrest, 2-fold for dysrhythmia and 50% for stroke.

Figure 5 below presents the ED results in Table 10 visually, showing the risk ratios and 95% confidence intervals for the effects (expressed as risk ratio) of LF smoke at high level on daily EDA for cardio-respiratory conditions.

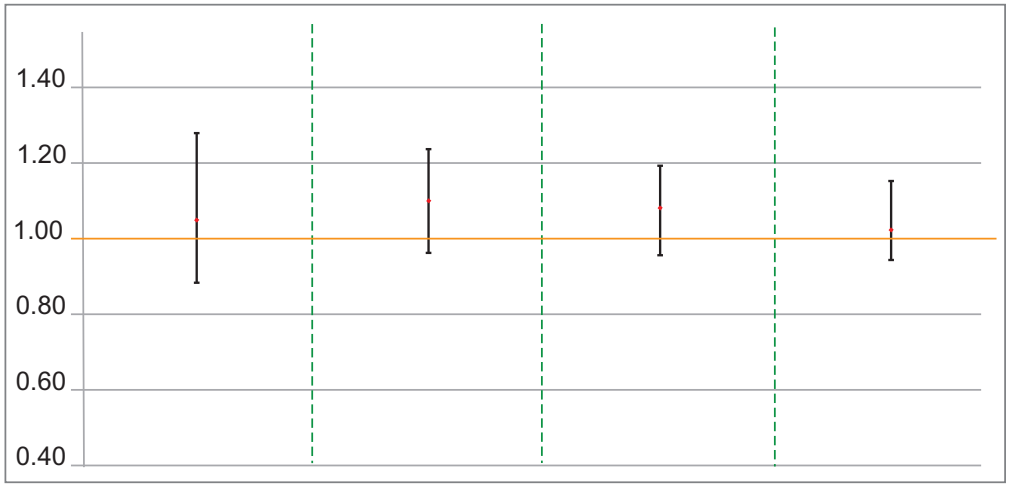
a) Total EDA



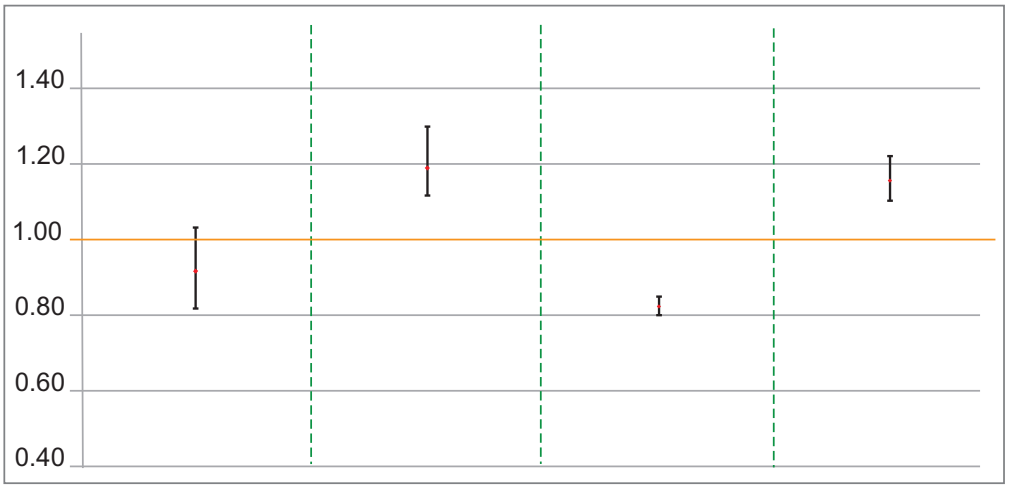
b) All respiratory



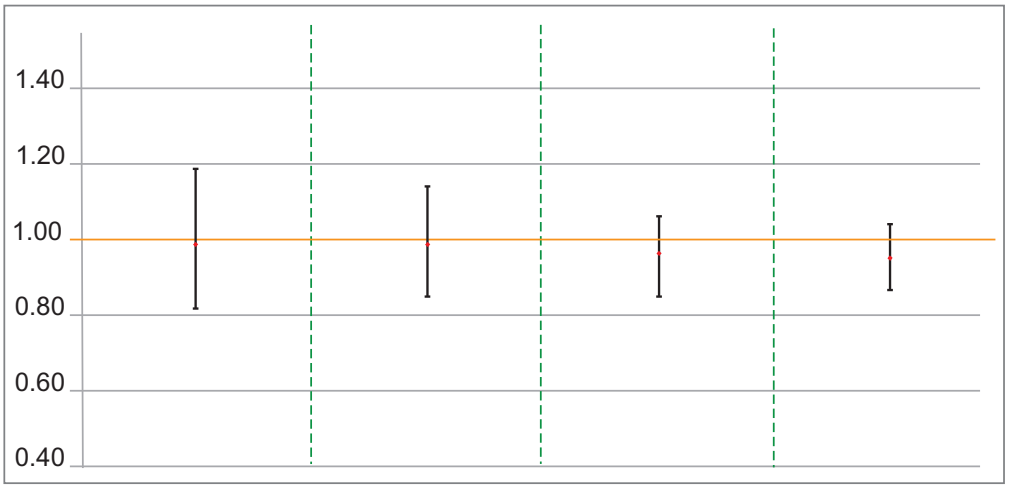
c) Asthma



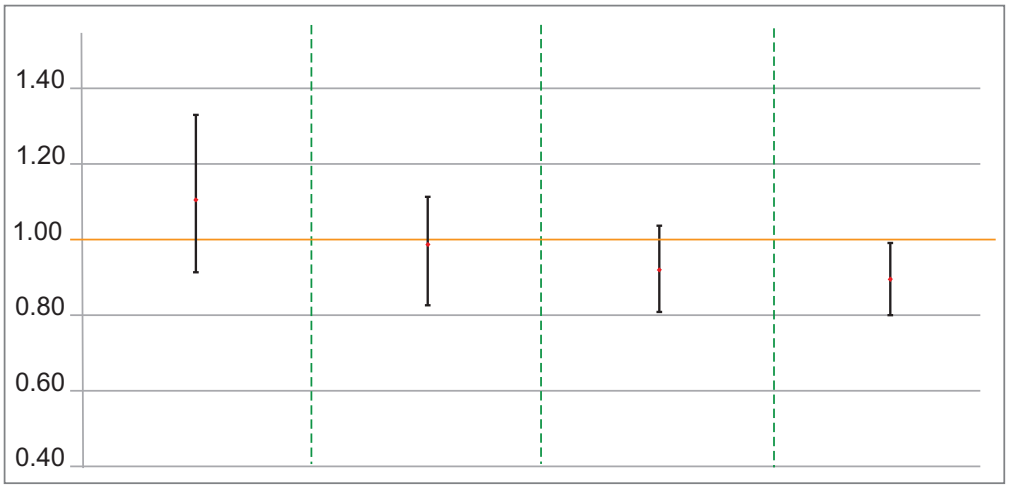
d) ALTRI



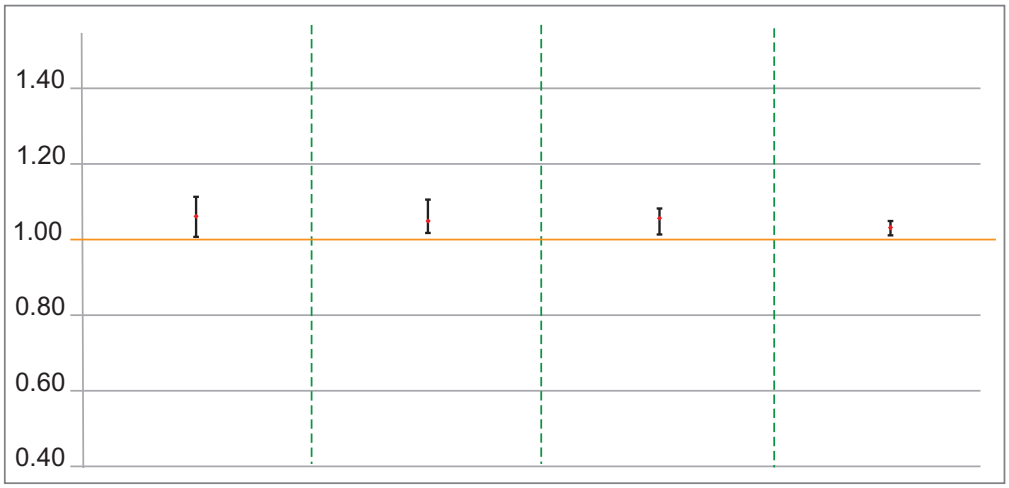
e) COPD



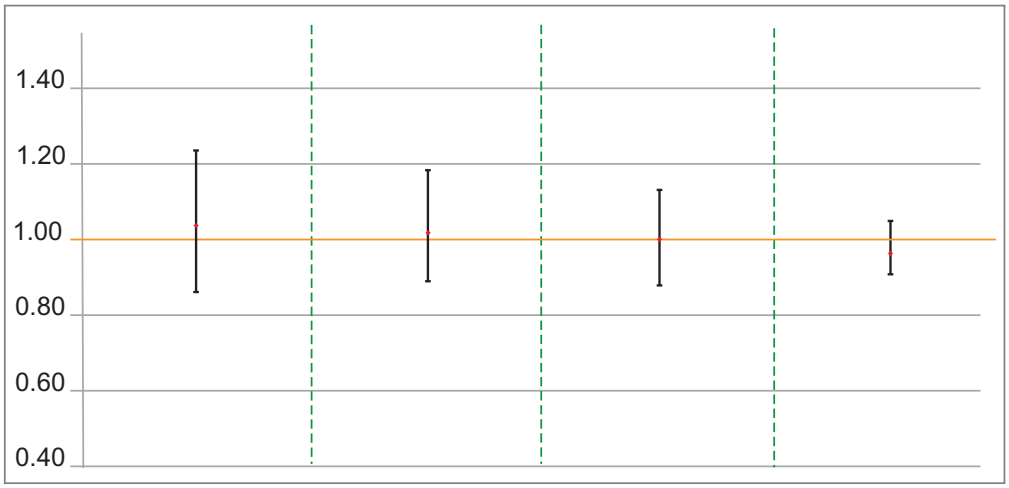
f) Croup



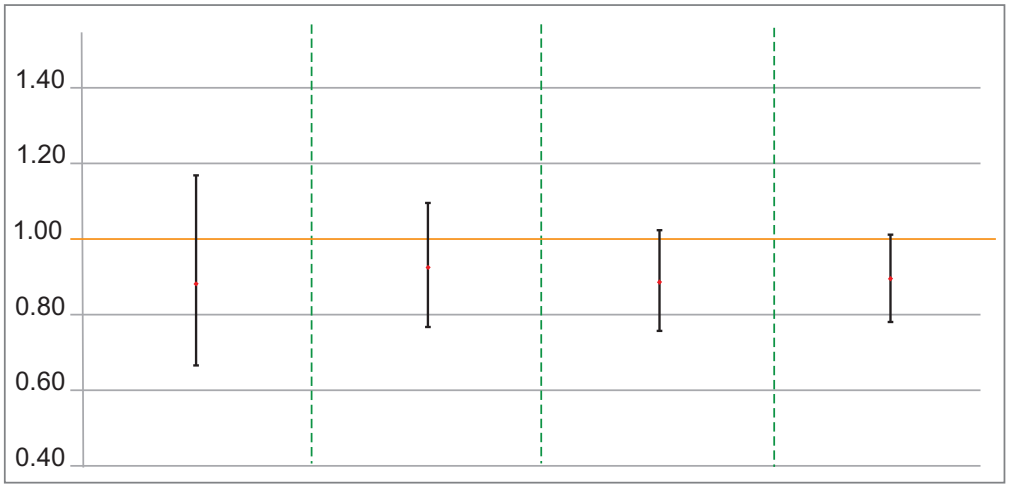
g) All cardiovascular



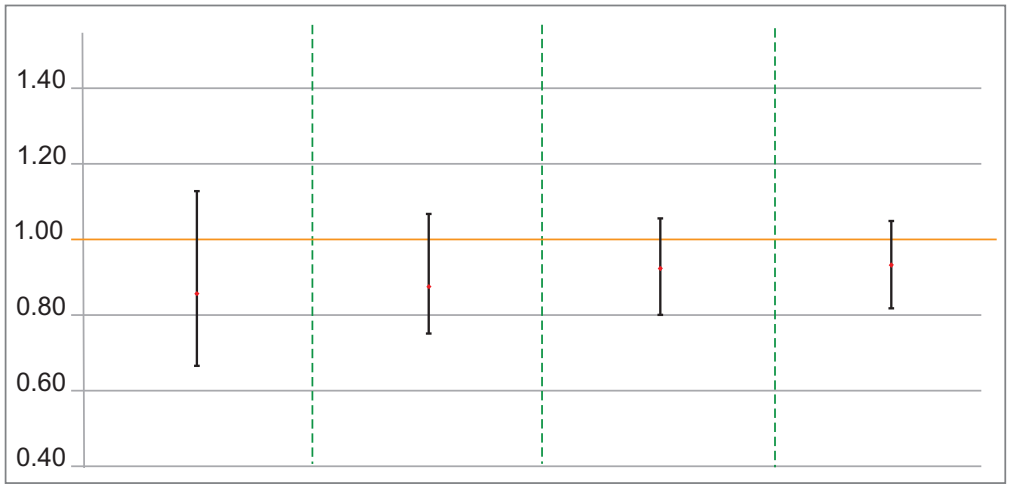
h) Arrhthmia



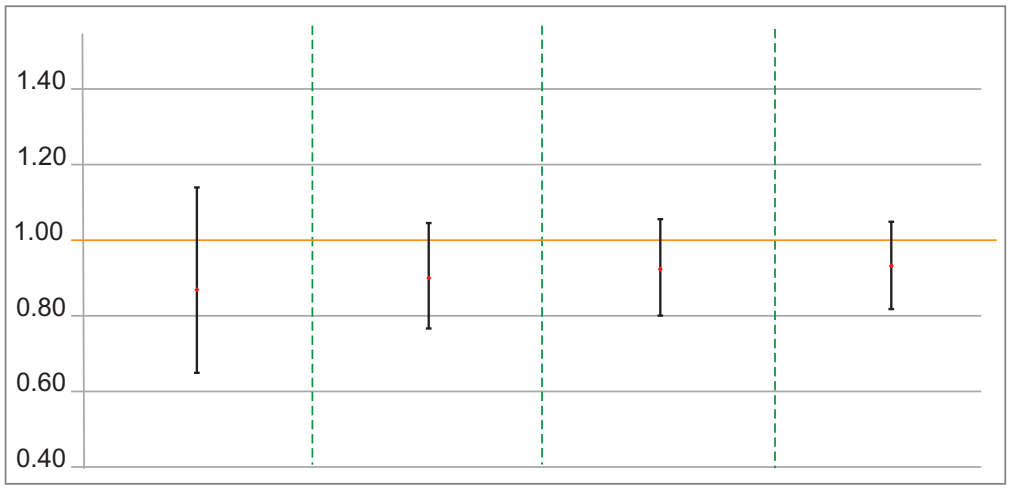
i) Angina



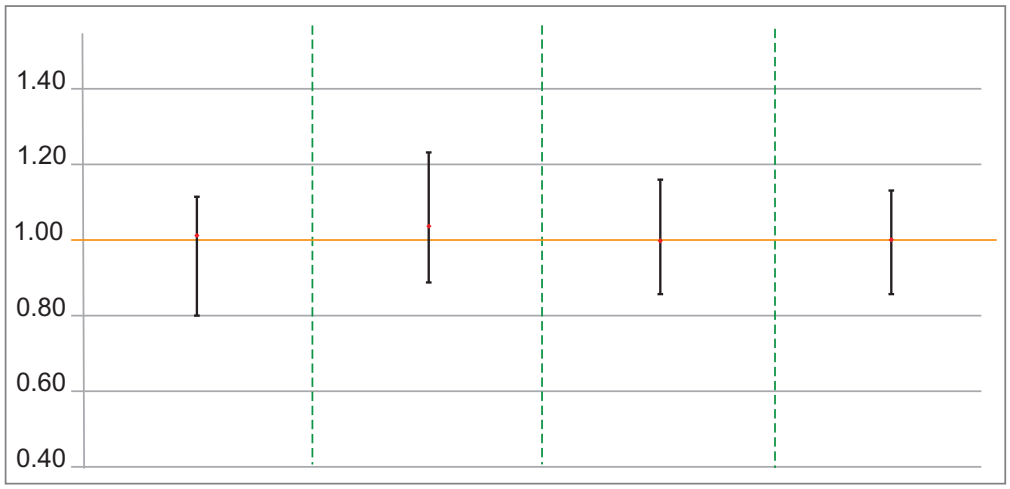
j) Stroke



k) Heart failure



l) ACS



m) TIA

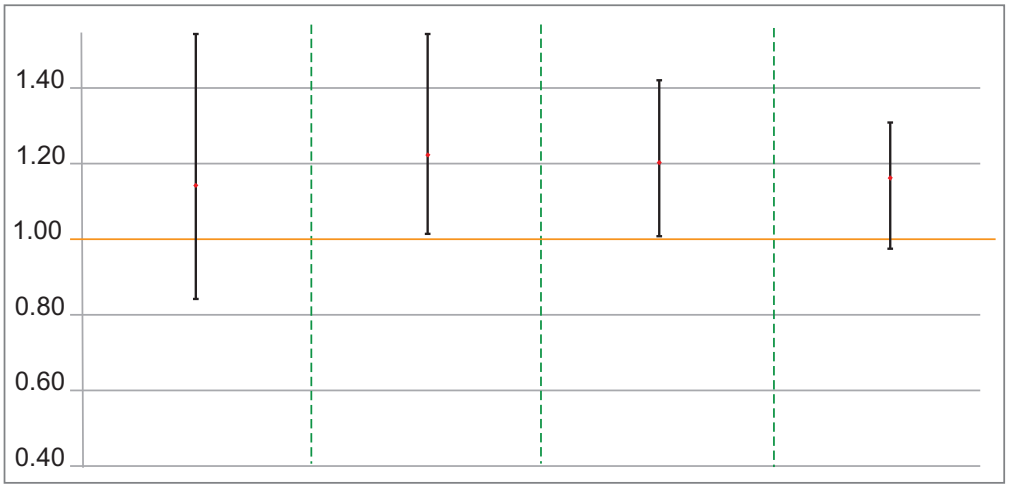


Figure 5. Risk ratio and 95% confidence intervals for assessment of effects of LF smoke at high level on daily EDA for cardio-respiratory conditions

In each graph, 1st to 4th bars show risk ratios and 95% CIs for lag 0 on the left and to lag 3 on the right, respectively. Y axis denotes risk ratios. Horizontal yellow line indicates reference group (ie, low level fire smoke) risk ratio (eg, a value of 1). ALRTI = Acute Lower Respiratory Tract Infections; COPD = Chronic Obstructive Pulmonary Disease; ACS = Acute Coronary Syndrome; TIA = Transient Ischemic Attack

3.2. Hospital admissions

3.2.1 Association of total number of hospital admissions with smoke related $PM_{2.5}$

In the multivariate analysis of total hospital admission outcomes, we found a 2% significant increased risk on the same day smoke-related $PM_{2.5}$ (lag 0) where the smoke-related $PM_{2.5}$ was at both the medium level (96-98 percentile) and high level (>=99 percentile). There were significant dose response relationships on delayed lag effects of 1 to 3 days in medium and high-level smoke related $PM_{2.5}$ compared to low level (Table 10).

In an interaction analysis with age, sex and SEIFA, we found that the total hospital admissions rates significantly increased with dose response effects for 4% (RR: 1.04, CI: 1.01-1.07) on medium level smoke, 5% (RR: 1.05, CI: 1.01-1.10), 4% (RR: 1.04, CI: 1.01-1.08), 5% (RR: 1.05, CI: 1.02-1.08) on lags effects 0 to 3 respectively after exposure to smoke-related $PM_{2.5}$ at high-level smoke in those aged >=60 years risk as compared to the people aged 15 to 59 years old. There was also 3% increase (RR: 1.03, CI: 1.01-1.05) risk in children age under 14 years old on lag effects 2 and 3 after exposure to high level smoke.

3.2.2. Association of hospital admissions for selected adverse respiratory health outcomes with smoke related $PM_{2.5}$

This study indicated that there was 1% to 18% non-significant increased risk with dose-response effect for individuals with underlying respiratory disease of asthma (Table 10).

In an interaction analysis with age, sex and SEIFA, we found that the hospital admission rates due to respiratory outcomes significantly increased with dose response effect (not shown here) for 25% (RR: 1.25, CI: 1.02-1.53), 20% (RR: 1.20, CI: 1.04-1.39), 11% (RR: 1.11, CI: 1.00-1.24) on lags effects 0, 1 and 3 respectively after exposure to smoke-related $PM_{2.5}$ at high-level smoke in those aged >=60 years risk as compared to the people aged 15 to 59 years old.

In an interaction analysis with age, sex and SEIFA, we found that the hospital admission rates for asthma significantly increased about 59% in those children aged <=14 years (RR: 1.59, CI: 1.04-2.43) on the same day at medium level smoke, and 93% (RR: 1.93, CI: 1.05-3.56) on lag1 at high level smoke, and 36% (RR: 1.36, CI: 1.06-1.74) on lag2 medium smoke level, and 39% (RR: 1.39, CI: 1.11-1.72) on lag3 medium smoke level. Hospital admission rates due to asthma also increased in those mid disadvantaged areas (RR: 1.31, CI: 1.01-1.71) 46% (RR: 1.46, CI: 1.17-1.81) on lag1 and 2 at medium-level smoke, and with significant dose response of 38% (RR: 1.38, CI: 1.13-1.67) and 52% (RR: 1.52, CI: 1.07-2.15) on lag3 at both medium- and high-level smoke.

Hospital admission rates due to COPD also increased significantly more than five-fold in those children aged <=14 years (RR: 5.43, CI: 1.29-22.85), 54% (RR: 1.54, CI: 1.09-2.19), 37% (RR: 1.37, CI: 1.06-1.78) on the same day and on lag1 and 3, all at high-level smoke.

3.2.3. Association of hospital admissions for selected adverse cardiovascular health outcomes with smoke related $PM_{2.5}$

There was a significant 2% to 7% increase at medium or high-level smoke with dose response effect on the same day smoke related $PM_{2.5}$ (lag 0), and on all lag effects of 1 to 3 days for total cardiovascular health outcomes. There was 1% to 18% increased risk for individuals with underlying cardiovascular diseases including Arrhythmia (12% significant increase at medium-level smoke and 1 to 10% non-significant increase on lags 1 to 3 with dose-response effect), angina (3% to 12 % non-significant increase risk on lags 1 to 3 with dose response effects), stroke (1 to 17% non-significant increase on all lags effects with dose-response effect), heart failure (1% to 4% non-significant increase risk on all lags effects with dose response effects), Acute Coronary syndrome (1% to 5% non-significant increase risk on lags 0 to 2 with dose response effects), transient ischemic attack (4% to 18% non-significant increase risk on all lag effects with dose response effects on lags 2 and 3).

In an interaction analysis with age, sex and SEIFA, we found that the hospital admission rates for total cardiovascular diseases and for those with underlying heart failure significantly increased 7% (RR: 1.07, CI: 1.02-1.13) to 12% (RR: 1.12, CI: 1.00-1.26) in people living in disadvantaged areas after exposure to medium level smoke on lag3.

Hospital admission due to angina also increased significantly about two and half-fold (RR: 2.54, CI: 1.15-5.60) on the same day, (RR: 2.10, CI: 1.24-3.56) on lag 1, and lag2 (RR: 1.24, CI: 1.03-1.49) in people living in disadvantaged areas after exposure to medium level smoke.

3.3. Ambulance callouts

3.3.1. Association of total number of ambulance callouts with smoke related $PM_{2.5}$

Smoke-related $PM_{2.5}$ was not associated with the total number of ambulance callouts on same day or delayed effects in Perth metropolitan area (Table 10).

In an interaction analysis with age, sex and SEIFA, we found that the total ambulance callout rates significantly increased with dose response effect for 22% (RR: 1.22, CI: 1.04-1.44), 15% (RR: 1.15, CI: 1.03-1.29), 14% (RR: 1.14, CI: 1.04-1.25), 16% (RR: 1.16, CI: 1.07-1.26) on lags effects 0 to 3 respectively after exposure to smoke-related $PM_{2.5}$ at high-level smoke in those aged >=60 years risk as compared to the people aged 15 to 59 years old. We also found that those living in disadvantaged areas had significant increased risk about 26% (RR: 1.25, CI: 1.06-1.49) on the same day and to 12% (RR: 1.12, CI: 1.00-1.26), 16% (RR: 1.16, CI: 1.05-1.27), 11% (RR: 1.11, CI: 1.02-1.21) on lags 1 to 3 with dose response effect after exposure to high level smoke-related $PM_{2.5}$ as compared to the socio-economically least disadvantaged areas. There was also a similar significant dose response effects on all lag effects for mid disadvantaged group as compared to advantaged group. Females as compared to males had about 10% (RR: 1.10, CI: 1.00-1.22) significant increase on lags 1 to 3 after exposure to high level smoke related $PM_{2.5}$.

3.3.2. Association of ambulance callouts for selected adverse respiratory health outcomes with smoke related $PM_{2.5}$

Smoke-related $PM_{2.5}$ was not significantly associated with the total number of same day or delayed effects for total number of respiratory outcomes in Perth metro. There was 1% to 11% increased risk for individuals with underlying respiratory disease of asthma (non-significant increase on all lag effects). There was also 7% to 25% increased risk for individuals with underlying respiratory arrest (non-significant increase on all lag effects with dose response effect on lags 2 & 3). The associations between smoke related $PM_{2.5}$ and respiratory conditions are shown in Table 10.

In an interaction analysis with age, sex and SEIFA, we found that the ambulance callout rates due to respiratory outcomes significantly increased with dose response effect (not shown here) for 45% (RR: 1.45, CI: 1.11-1.89), 34% (RR: 1.34, CI: 1.21-1.61), 23% (RR: 1.23, CI: 1.05-1.43), 19% (RR: 1.19, CI: 1.04-1.36) on lags effects 0 to 3 respectively after exposure to smoke-related $PM_{2.5}$ at high-level smoke in those aged >=60 years risk as compared to the people aged 15 to 59 years old. We also found that those living in disadvantaged had significant increased risk about 72% (RR: 1.72, CI: 1. 31-2.25) on the same day and to 43% (RR: 1.43, CI: 1.20-1.71), 37% (RR: 1.37, CI: 1.18-1.59), 26% (RR: 1.26, CI: 1.11-1.43) on lags 1 to 3 with dose response effect after exposure to high level smoke-related $PM_{2.5}$ as compared to those living in the socio-economically advantaged areas. There was also a similar significant dose response effects on all lag effects for mid disadvantaged areas as compared to advantaged areas. Females as compared to males had about 13% (RR: 1.13, CI: 1.01-1.26) significant increase on lag 3 after exposure to high level smoke related $PM_{2.5}$.

There was also significant 58% to 68% increase in all lag effects for asthma in those mid disadvantaged areas after exposure to mid-level smoke related $PM_{2.5}$.

Those elderly people aged >=60 years had about 42% increase risk of respiratory infection after exposure to high-level smoke in lag 1 and in lag 2 with less extent of 18% increased risk. There was also about two-fold significant increase (RR: 1.96, CI: 1. 22-3.16) in those with respiratory infection condition on the same day and to 42% (RR: 1.42, CI: 1.03-1.94), 39% (RR: 1.39, CI: 1.08-1.80), 27% (RR: 1.27, CI: 1.01-1.59) on lags 1 to 3 with dose response effect in those disadvantaged areas after exposure to high level smoke-related $PM_{2.5}$.

3.3.3. Association of ambulance callouts for selected adverse cardiovascular health outcomes with smoke related $PM_{2.5}$

Smoke-related $PM_{2.5}$ was not associated with the total number of same day or delayed effects for total number of cardiovascular health outcomes in Perth metro. There was 5% to 20% increased risk for individuals with underlying angina (non-significant increase on lag effects 2 & 3 with dose response effect). There was also 1% to 6% increased risk for individuals with underlying cardiac arrest (non-significant increase on all lag effects at medium level). The associations between smoke related $PM_{2.5}$ and respiratory conditions are shown in Table 10.

In an interaction analysis with age, sex and SEIFA, we found that the ambulance callout rates due to total cardiovascular outcomes significantly increased with dose response effect (not shown here) for 14% (RR: 1.14, CI: 1.03-1.26), on lag effects 3 after exposure to smoke-related $PM_{2.5}$ at high-level smoke in those aged >=60 years risk as compared to the people aged 15 to 59 years old. We also found that those living in disadvantaged areas had significant increased risk about 19% (RR: 1.19, CI: 1. 01-1.40), 15% (RR: 1.15, CI: 1.00-1.32), and 25% (RR: 1.25, CI: 1.11-1.40) on lags 1 to 3 with a dose response effect after exposure to high level smoke-related $PM_{2.5}$ as compared to the socio-economically advantaged areas.

There was also about two-fold significant increase risk (RR: 2.06, CI: 1.05-4.01) on the same day and to 45% (RR: 1.45, CI: 1.04-2.02) on lags 3 with a dose response effect in those elderly people age 60 years and above with cardiac arrest condition after exposure to high level smoke-related $PM_{2.5}$. There was also significant 59% increase in lag effect 3 in those people with cardio arrest condition in mid-disadvantaged areas after exposure to mid-level smoke related $PM_{2.5}$.

Elderly people aged 60 years and above also had 75% (RR:1.75, CI: 1.10-2.78) increase risk of having angina after exposure to high level smoke in lag3.

People in mid disadvantaged group had about two-fold increase risk of dysrhythmia (RR:1.99, CI: 1.10-3.61) on the same day exposure to high level smoke.

People in disadvantaged areas had about 50% (RR:1.50, CI: 1.00-2.25) increase risk of stroke (RR:1.50, CI: 1.00-2.25) on lag3 after exposure to high level smoke.

4. Discussion

4.1. Statement of principal findings

For this study we modified and validated a model developed by Yao and Henderson (2016) to determine exposure to LFs smoke related $PM_{2.5}$. Using this model, we observed a dose-response association with 2% to 5% significant increased risk of total emergency attendance and total hospital admission rates on same day and all delayed lag effects for those people who exposed to landscape fire smoke as measured by smoke-related $PM_{2.5}$ at medium level (95-98th percentile) and high level (>=99th percentile) compared to the low level (<95th percentile). Exposure to landscape fire smoke at high levels (>=99th percentile) was associated with a wide range of adverse respiratory, and cardiovascular diseases in all health care utilisations.

4.2 Assessing population exposure to landscape fire smoke

Assessment of population exposure to smoke from LFs has been a key challenge in health studies. There was a range of approaches applied in previous studies. Five categories of exposure assessment tools were considered on this topic of research including routine air quality monitoring, fire smoke proxy such as burned area after landscape fires, remote sensing products, forecasting modelling and retrospective modelling which includes mechanistic air quality models and the empirical models using regressions with visibility, meteorological data, monitoring measurements and remote sensing data, to estimate PM concentrations during wildfire smoke events. All these tools have strengths and limitations, which has been discussed in an evidence review (Yao et al, 2014). Briefly, studies used indicators such as ground-based air monitors, or area burnt. These are indirect proxies and their accuracy can be influenced by other environmental factors, so it is difficult to ascertain the fraction of health morbidity due to bushfire smoke. Moreover, ground-based air pollution monitors are not located in all places or time periods with affected populations. Remote sensing based on satellite data provides enough spatial coverage, but measures smoke at all attitudes, not always representative of ground-level exposure, particularly in relation to the diurnal and seasonal weather patterns that influence the Perth air-shed. As advised by DBCA, these include the predictable summer sea-breeze, and thermal inversions common in the cooler months.

There are also missing data due to cloud cover because of using only satellite data. Forecasting models have been recommended to predict the future development of the smoke events, but not to determine actions according to the exact predicted concentrations (Yao et al, 2014). There is uncertainty in model performance and establishing models require expertise and resource to run the model. Both remote sensing and forecasting models are suitable for qualitative but not quantitative use.

With consideration of available resources and the aims of this project, we selected an empirical model for this project developed in Canada (Yao and Henderson, 2014). This approach is simple to operate and has high spatial and temporal resolution which suits the aim of this project. It is suitable for monitoring landscape fire smoke from long-ranged transportation and improving the spatial resolution of existing monitoring networks.

Chemical transport models, such as Global Earth Observing System (GEOS-Chem) models, a global three-dimensional (3-D) model of tropospheric chemistry (Brey et al., 2018), Weather Research and Forecasting Model with Chemistry (WRF-Chem) (Grell et al., 2005) or chemical transport model (CTM) (Cope et al., 2004) can estimate air pollutants specifically from bushfires. These methods were previously used in other studies (Alman et al., 2016, Haikerwal et al., 2015, Liu et al., 2017). These models also have limitations. For example, one limitation of using CTM is that the bushfire-specific pollutant estimates may be difficult to validate. Modelled data could also be computationally expensive and requires collaborative efforts of atmospheric scientists.

4.3 Emergency department attendances associated with cardio-respiratory morbidity

We found 1% to 5% significant increased risk in total emergency attendance rates and up to 25% increased risk for individuals with underlying cardio-respiratory diseases including asthma, acute lower respiratory tract infections, transient ischemic attack and acute coronary syndrome after exposure to landscape fire smoke-related $PM_{2.5}$. There were large differences in study design, air pollution type, time periods and statistical methods used in literature. For Australia, studies of the effect of LFs smoke on respiratory health related emergency visits have been done in Melbourne (Tham et al., 2009), Darwin (Johnston et al., 2002), Sydney (Cooper et al, 1994, Smith et al, 1996). Tham et al (2009) used a time series approach to investigate the effects of PM10, and other pollutants on respiratory-related EDA during a 6-month bushfire season in 2002-2003 and found a 9.1ug/m3 increase in PM10 was non-significantly associated with a 1.8% (0.4-3.3%) increase in respiratory-related EDA presentations in Melbourne. Johnson found a significant 20% increase in asthma visits per 10mg/m3 increase in PM10 and that risk of emergency contact for asthma in Darwin were 2.4 times greater on fire days with PM10>10ug/m3 compared with days with PM10<10mg/m3 (Johnston et al., 2002). Exposure to particulate air pollution and recorded asthma attacks or increased bronchodilator use were also reported in literature (Pope, 2000). Our results on acute lower respiratory tract infections are consistent with large studies in literature when they reported associations with lower respiratory symptoms and cough and were usually statistically significant. An increased risk in selected respiratory health effects related emergency visits was also observed in North America. Miller in a study of the health impacts of wildfire smoke in US for the period of 2006 to 2013 found evidence that increase in respiratory outcomes are due mainly to lower respiratory tract infections (Miller et al., 2017), which is consistent with what we found in this study. Studies in Sydney by Cooper et al and Smith et al (Cooper et al., 1994, Smith et al., 1996) didn't detect an adverse association on respiratory health related emergency visits.

This is the first comprehensive study in Australia to evaluate the relationship between landscape fire smoke-related $PM_{2.5}$ with a wide range of cardiovascular morbidity related to EDA and the first to study a dose-response relationship on this association. We found statistically significant associations at the high level smoke-related $PM_{2.5}$ with total cardiovascular outcomes, which is consistent with the wider evidence (Brook et al., 2010, Nawrot et al., 2011, Pope et al., 2006). We also found 1% to 25% significant increase risk with dose response relationship for transient ischemic attack on lag effects of 1 and 2, which is consistent with a study in Victoria, Australia which found an increase of ischemic heart disease (IHD) by 2.07% at lag 2 after exposure to wildfires. Studies have reported a 2% to 20% increase in risk of acute IHD-related morbidity for a 10mg/m3 increase in $PM_{2.5}$ levels (Peters et al., 2001, Pope and Dockery, 2006, Pope et al., 2006). The sustained effects of

wildfire smoke exposure and cumulative biological effects could be responsible for a delayed effect of $PM_{2.5}$ exposure on acute CHD events (Delfino et al., 2009, Haikerwal et al., 2015). The influence of individual perceptions, severity of symptoms, environmental conditions and decisions to seek medical care during bushfire episodes have been discussed in literature as possibilities for the delayed impact of wildfire $PM_{2.5}$ exposure, and needs further exploration (Delfino et al., 2009, Haikerwal et al., 2015, Rappold et al., 2012).

4.4 Hospital admissions associated with cardio-respiratory morbidity

Our study indicated that there was 2% to 7% significant increase risk in total hospital admission and total cardiovascular diseases and up to 18% increased risk for individuals admitted to hospital with underlying respiratory disease of asthma, arrhythmia, angina, stroke, acute coronary syndrome, and transient ischemic attack. Australian studies of the effects of bushfire smoke on hospital admissions for respiratory disease have been done in Sydney (Morgan et al., 2010), Brisbane (Chen et al., 2006), Darwin (Hanigan et al., 2008, Johnston et al., 2007) and in Sydney, Newcastle, and Wollongong (Martin et al., 2013). Hanigan (Hanigan et al., 2008) used a time series analysis and found an increase of 10ug/m3 same day PM10 was associated with a non-significant 4.81% increase in total hospital admissions which was similar with this WA study. Johnson (Johnston et al., 2007) used a case-crossover analysis and found an increase of 10ug/m3 in PM10 was non-significantly associated with hospital admissions for all respiratory conditions (OR: 1.08, 95% CI: 0.98-1.18) and significantly associated with COPD hospital admissions (OR: 1.21, 95% CI: 1.00-1.47). Martin (Martin et al., 2013) used a time-stratified case-crossover design to assess the association between smoke events and hospital admissions and found 6%, 12% and 13% same day increase in respiratory hospital admissions. Similarly, studies in US found exposure to wildfire smoke was associated with hospital admissions for cardiovascular disease such as cardiac arrests, or symptoms such as chest pain (Azevedo et al., 2011, Delfino et al., 2009, Rappold et al., 2012, Rappold et al., 2011). Our positive associations for arrhythmia, acute coronary syndrome, angina, stroke, and transient ischemic attack are also consistent with the wider evidence in studies of urban air pollution (Brook et al., 2010, Martinelli et al., 2013).

4.5 Ambulance callouts associated with cardio-respiratory morbidity

We found 1% to 25% increased risk for individuals with underlying cardio-respiratory diseases including asthma, respiratory arrest, angina, and cardiac arrest after exposure to landscape fire smoke-related $PM_{2.5}$. Our results for ambulance callouts were not as strong as the results from emergency department and hospital admissions. One possibility is that EDA data is a better indicator of LF impacts on human bodies as the LFs mainly cause acute diseases. Ambulance callouts are a source of population-level health information that has been less extensively evaluated with respect to air pollution. Johnson 2019 (Johnston et al., 2019) evaluated 394, 217 paramedic assessments and daily $PM_{2.5}$ concentrations from three states in South-Eastern Australia and used a time-stratified, case-crossover analysis and found 6% to 12% increased odds of paramedic assessments with a range of respiratory and cardiovascular outcomes, which is similar to what we found in this study and a study in Melbourne (Dennekamp et al., 2015).

4.6 Vulnerable sub-populations

Although there are good data on differential effects of air pollution on vulnerable sub-populations, there is limited research to address the question of who is at risk to adverse health effects from landscape fire smoke related $PM_{2.5}$. Our findings in relation to all health care utilisations due to cardio-respiratory diseases are consistent with some of available evidence that the elderly adults (Delfino et al., 2009, Henderson et al., 2011, Morgan et al., 2010, Mott et al., 2005, Rappold et al., 2011); and people living in low-socio-economic areas (Hanigan et al., 2008, Johnston et al., 2007, Rappold et al., 2012) are most likely to be susceptible to this relatively short-term exposure to smoke-related $PM_{2.5}$. Men and women may also have different health risks when exposed to bushfire smoke. In our study, a statistically significant difference was only observed for acute lower respiratory tract infections where women had 20% increased risk compared to men in lag effect of 1 day. We didn't find any differences in smoke-related $PM_{2.5}$ effect estimates between men and women in respiratory and cardiovascular emergency attendances and persons with existing health conditions such as chronic cardio-pulmonary disease, influenza, and asthma although we didn't assess the effect of precondition of diseases in this study. These results are consistent with other studies such as assessing respiratory diseases physician visits (Henderson et al., 2011).

4.7 Strengths and limitations

This is the first study in Western Australia to evaluate the effect of landscape bushfire smoke on a wide range of adverse respiratory and cardiovascular events using three large WA health care datasets. The main strength of the study was the use of earth observation data including digitalised smoke plumes for wildfires and prescribed burns identified via NASA's satellite, aerosol optical depth from NASA, fire radiative power from the Geoscience Australia online grid, and venting index and fire danger rating from BOM. Furthermore, we used temporally and spatially resolved modelled air exposure data from a wider Perth metropolitan area including areas with no monitoring facilities. We modified a well-established smoke-optimised empirical exposure model for the Perth metropolitan area to estimate fire smoke-related $PM_{2.5}$ concentrations. A further strength was that we utilised three comprehensive state-wide health service datasets (EDA, hospital admissions and ambulance callouts) to obtain information about socio-demographic, socio-economic, clinical health data and other information relevant to aim of this study. We were able to assess specific conditions including a wide range of respiratory and cardiovascular diseases. Finally, we were able to assess the dose-response relationship which is an important aspect of the epidemiological evidence in relation to harms caused by landscape fire air pollution exposure.

As similar with other studies, there were some limitations, although we tried our best to address most of these limitations. One limitation was that we could not distinguish between the effects of wildfires and prescribed burns on daily smoke related $PM_{2.5}$ count as there was a possibility of observing WFs and PBs within a geographical proximity and within the days of each other. It was impossible to identify the type of fire if two types were reported at similar time and proximity, especially when fire plumes of the two types were mixed due to wind. However, one thing to note is that wildfires were more common than prescribed burns and accounted for about 80% of the total number of landscape fires in the study area during the study period.

There were limited numbers of air quality monitoring stations in the Perth metropolitan area to estimate smoke related $PM_{2.5}$. To address this limitation, we tried GWRR and IDW modelling

and finally selected IDW method to derive estimation on air quality for the whole Perth metropolitan area including the area where we didn't have air quality monitoring stations as well as those areas without $PM_{2.5}$ monitoring results. Despite this, there might be a possibility of some degrees of uncertainty on the accuracy of the predicted measure of smoke related $PM_{2.5}$.

There was also a possibility of exposure misclassification, as all persons may not have been exposed to the same levels of $PM_{2.5}$ (ecological fallacy). To address this limitation, we designed air pollution modelling with considerable spatial variation within a 1.5 by 1.5 km scale, so the smoke related $PM_{2.5}$ can be captured in our exposure modelling with spatial variation. We believe this would reduce the exposure misclassification as compared with other studies where they used at least 5 km by 5 km. We also believe that although it is likely that a large amount of smoke-related $PM_{2.5}$ was due to bushfire smoke, the impact of it on health service found in this study could not be attributed solely to bushfire $PM_{2.5}$. There are only a few studies in Australia that did attempt to separate bushfire PM10 from background PM10 (Chen et al., 2006, Morgan et al., 2010), but none for assessing $PM_{2.5}$.

Another limitation was that the smoke episodes were of short duration. We investigated the lag effects of 3 days and the population of about 1.5 million emergency attendances, more than half-million hospital admissions and about 80,000 ambulance callouts for two and half years. However, statistical power could be increased by studying larger populations and over longer periods.

We had a lack of information on personal risk factors, such as alcohol consumption, smoking and underlying personal health conditions. However, we were able to include important potential risk factors such as socio-demographic factors, socio-economic factors, area-level environmental exposures, and seasonal factors in our analytical modelling. Information on these confounding variables would be important for health planning programs and interventions for the vulnerable people.

We also had a lack of information on smoke plumes caused by bushfires managed by local government or Department of Fire Emergency Services, agricultural burning, burning of debris associated with land development activities as there was no relevant/complete data available for the project.

There were also limitations in statistical data analysis of this study. Modelling of counts using a Poisson distribution may be limited as our data had zero inflation with excess of zero counts in the data. To address this limitation, we also used Zero-inflated Poisson (ZIP) regression, but there was no any improvement in the results. The number of observations in a few of the models was small, so we could not obtain the reliable results in those models. Multi-pollutant models were not included in the modelling, so the effects could be due to other air pollutants or their combined effects. There is some evidence that other pollutants such as O3 might have adverse health effects (Azevedo et al., 2011). We included the effect of wood fire use in the modelling, although the effect was not significant. On the other hand, studies from Australian wildfire events reported that $PM_{2.5}$ was the most significantly elevated pollutant and its level exceeded the regulatory air quality standards as compared to other pollutants (Dennekamp et al., 2015, Reisen et al., 2011). A study on biomass burning emissions over northern Australia reported that 87% of PM10 due to wildfire consists of $PM_{2.5}$ (Luhar et al., 2006).

5. Conclusions, Significance, and Recommendations

In this study, we used earth observation data from satellite images and modified a smoke optimised empirical $PM_{2.5}$ exposure model in assessing the population's exposure to landscape fire smoke in Western Australia. We found that the method was useful in evaluating the effect of LF smoke on a wide range of adverse respiratory and cardiovascular diseases.

Our study showed a strong link between smoke related $PM_{2.5}$ and adverse health effects for a wide range of respiratory and cardiovascular related emergency department attendance and hospital admissions but with less evidence in ambulance callouts. We have shown that landscape fire smoke exposure is associated with increases in general EDA and general hospitalisation admissions, but with no significant increase in general ambulance callouts. Our results on the impact of LF smoke-related $PM_{2.5}$ on respiratory effects showed a significant dose-response association in delayed effects of lag 1 and lag 3 days with 8% to 19% increased risk for acute lower respiratory tract infection EDAs. Our results on acute lower respiratory tract infections are consistent with large studies in literature when they reported associations with lower respiratory symptoms and cough, and were usually statistically significant. In addition, there was a 3% to 10% non-significant dose-response effect for asthma increased risk for emergency department attendances and 2% to 18% non-significant increased risk in hospital admissions, and up to 11% non-significant increased risk in ambulance callouts on the same day and in almost all delayed lag effects. There was also a non-significant 25% increase in ambulance callouts due to respiratory arrest.

Our results suggest stronger effects for cardiovascular diagnosis. We provided evidence of a 2% to 7% significant increased risk at the high exposure level to smoke related $PM_{2.5}$ in both EDA and hospital admissions in the general cardiovascular category, but with no significant increase in general ambulance callouts. The significant increase in general cardiovascular EDA to a large extent is explained by diagnosis related to transient ischemic attack. Transient ischemic attack had a significant increased dose-response risk up to 25% in lag1 and lag 2 at the high level. This study is the first to find a significant dose-response relationship between exposure to landscape fire $PM_{2.5}$ and an increase in the risk for EDA due to transient ischemic attack. There was also nonsignificant increase risk of up to 25% in all health care utilisations due to a variety of cardio diseases such as arrhythmia, angina, stroke, acute coronary syndrome, and cardiac arrest.

The results suggest that, among various measures of health, older people aged 60 years and above, people from low socioeconomic areas and those with heart or lung problems are more susceptible community members to LFs smoke.

This is the first WA study to examine the effects of LFs on a large population, covering the whole metropolitan area of Perth. The findings of this project would provide an evidence base to inform policy development in reducing and/or eliminating the impacts of LFs including WFs and PBs. It would also provide evidence for any future studies (e.g., development of spatial applications) to inform health promotion planners, health services, clinicians, patients, and the public before the planned LFs events and after uncontrolled LFs events.

Based on the study findings we recommend the following two categories of recommendations.

Policy recommendations

- Policy makers and health professionals should initiate and enhance community education programs about the harms caused by the landscape fires. Community education programs should also provide practical advice on actions that can be taken by individuals to minimise exposure and potential harm including the emphasis of the main respiratory and cardiovascular conditions identified in the study.
- Health education programs should focus on fire prevention, management and general safety and adapting personal protective behaviours during a smoke episode and to take reasonable precautions to avoid bushfire smoke inhalation. The program should also include the emphasis of possible delayed smoke effects on individuals in the affected areas. The programs should start prior to the LF seasons (i.e., September to June).
- Elderly people, children and populations with lower socio-economic areas should be made aware of the effects of air pollution including landscape fire smoke in health promotion programs. As part of overall hazards planning, common locations for the elderly and children (e.g., nursing homes, child-care centres and schools) should be spatially identified and pre-warning information be specifically sent to such locations via different media.
- The resources should be increased to establish more air quality stations than the current number, especially in the southern, eastern and northern outskirts of the Perth metropolitan area and Southwest areas where LFs occur frequently. Immediate mobile monitoring of particulate matter concentrations (in particular, $PM_{2.5}$) and data on routine ambient air quality monitoring in a local region to reflect community exposures are recommended (Public Health Response to Prolonged Smoke Events, NSW Health, 2017).

Technical recommendations

- Regular and real time capturing of landscape fire data should be implemented to determine population/geographical areas at risk. Smoke plumes identified via the satellites and other earth observation data collected should be used to assist in capturing LFs and/or monitoring their movement for improving early warning systems.
- Spatial services could assist with mapping smoke plumes and at-risk populations in the affected area. The existing spatial service at jurisdictions should be expanded to accommodate the increased need if required. In particular, the development of automatic mapping/digitalisation of smoke plumes should be considered so that timeliness and efficiency of tracking the trajectory of smoke plumes can be realised.
- Mobile apps such as AirRater (https://airrater.org/what-does-it-monitor/) or mobile messaging services such as asthma alert that are being developed in WA may incorporate such LF data so that LF exposed vulnerable populations can be informed and preventative measures can be taken in a timely manner. Mobile messaging services for alerting people with other relevant respiratory and cardiovascular conditions identified in the study should be considered at the same time during the design phase.

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Appendix

GWRR Statistical Analysis Results

We conducted univariate and multivariate Poisson Regressions to assess the health risks associated with the smoke related PM2.5 using GWRR method. In the multivariate regression models, we included all confounding factors as described above including socio-demographic factors such as age, gender, SEIFA, seasonal factors such as public holiday, weekend, season and other environmental exposures such as dew point temperature, humidity and wood fire use period to assess the potential health risks associated with associated with $PM_{2.5}$.

Multivariate Poisson Regressions for the Association between Health Conditions for Emergency Attendance (EDA), Hospital Admissions, and Ambulance Callout (AC) and LF Smoke-related PM2.5 for Same Day (Lag0) and lags of 1 to 3 Days

Health Outcomes	Lag	EDA				Hosp				AC			
		RR	95%CI		P-Value	RR	95%CI		P-Value	RR	95%CI		P-Value
Total	0(M)	1.01	1.00	1.01	0.131	1.01	1.00	1.03	0.123	0.95	0.91	0.98	0.006
	0(H)	1.03	1.01	1.05	0.000	1.02	0.99	1.05	0.181	0.97	0.90	1.04	0.384
	1(M)	1.02	1.01	1.02	<.0001	1.01	1.00	1.02	0.247	0.95	0.92	0.98	0.000
	1(H)	1.03	1.02	1.05	<.0001	1.02	1.00	1.04	0.033	0.95	0.90	1.00	0.052
	2(M)	1.02	1.02	1.03	<.0001	1.00	0.99	1.00	0.236	0.95	0.93	0.97	<.0001
	2(H)	1.03	1.03	1.04	<.0001	1.03	1.01	1.04	0.002	0.98	0.94	1.02	0.419
	3(M)	1.02	1.02	1.03	<.0001	0.99	0.99	1.00	0.110	0.96	0.94	0.98	<.0001
	3(H)	1.03	1.02	1.04	<.0001	1.02	1.00	1.03	0.009	0.97	0.94	1.01	0.154
Respiratory	0(M)	0.92	0.89	0.96	<.0001	0.94	0.89	0.98	0.007	0.92	0.86	0.97	0.005
	0(H)	0.93	0.88	0.99	0.022	0.96	0.88	1.05	0.381	1.00	0.89	1.11	0.936
	1(M)	0.94	0.92	0.96	<.0001	0.94	0.91	0.98	0.001	0.93	0.89	0.97	0.001
	1(H)	0.90	0.86	0.94	<.0001	0.95	0.89	1.01	0.089	0.93	0.86	1.01	0.067
	2(M)	0.95	0.93	0.97	<.0001	0.94	0.91	0.97	<.0001	0.94	0.90	0.97	0.000
	2(H)	0.90	0.87	0.93	<.0001	0.94	0.89	0.99	0.013	0.96	0.90	1.02	0.206
	3(M)	0.96	0.94	0.97	<.0001	0.95	0.93	0.97	<.0001	0.95	0.92	0.98	0.001
	3(H)	0.91	0.88	0.93	<.0001	0.94	0.90	0.98	0.005	0.94	0.89	1.00	0.037
Asthma	0(M)	0.85	0.76	0.95	0.005	1.00	0.85	1.17	0.986	1.04	0.86	1.26	0.674
	0(H)	1.12	0.93	1.34	0.219	1.09	0.82	1.44	0.567	1.31	0.96	1.80	0.088
	1(M)	0.91	0.84	0.98	0.017	1.06	0.95	1.19	0.269	1.07	0.94	1.22	0.329
	1(H)	1.03	0.90	1.18	0.688	1.03	0.84	1.27	0.775	1.11	0.88	1.42	0.378
	2(M)	0.96	0.91	1.02	0.222	1.03	0.94	1.12	0.595	1.05	0.94	1.17	0.361
	2(H)	1.05	0.94	1.17	0.426	0.99	0.84	1.18	0.918	1.09	0.89	1.33	0.407
	3(M)	0.96	0.91	1.01	0.128	1.03	0.95	1.12	0.452	1.06	0.97	1.17	0.192
	3(H)	1.03	0.94	1.14	0.492	1.03	0.89	1.19	0.737	1.06	0.89	1.26	0.533

Health Outcomes	Lag	EDA				Hosp				AC			
		RR	95%CI		P-Value	RR	95%CI		P-Value	RR	95%CI		P-Value
Acute Lower Respiratory Track Infections*	0(M)	0.90	0.85	0.95	0.001	0.93	0.87	1.00	0.044	0.88	0.79	0.98	0.023
	0(H)	0.82	0.73	0.91	0.000	0.90	0.79	1.03	0.125	0.95	0.79	1.14	0.560
	1(M)	0.88	0.84	0.92	<.0001	0.92	0.87	0.97	0.001	0.93	0.86	1.00	0.042
	1(H)	0.82	0.76	0.89	<.0001	0.93	0.85	1.02	0.113	0.85	0.74	0.97	0.020
	2(M)	0.89	0.86	0.92	<.0001	0.92	0.89	0.96	0.000	0.91	0.86	0.97	0.003
	2(H)	0.79	0.74	0.85	<.0001	0.89	0.83	0.96	0.003	0.88	0.79	0.98	0.022
	3(M)	1.11	1.04	1.18	0.001	0.93	0.90	0.97	0.000	0.92	0.87	0.97	0.001
	3(H)	1.24	1.17	1.31	<.0001	0.89	0.83	0.95	0.000	0.89	0.81	0.98	0.019
Chronic Obstructive Pulmonary Disease	0(M)	0.92	0.83	1.02	0.121	1.07	0.98	1.18	0.141				
	0(H)	0.97	0.81	1.17	0.787	1.00	0.84	1.20	0.992				
	1(M)	0.92	0.85	0.99	0.027	1.02	0.95	1.09	0.617				
	1(H)	0.89	0.77	1.02	0.090	0.93	0.82	1.06	0.285				
	2(M)	0.92	0.87	0.98	0.009	0.99	0.94	1.05	0.728				
	2(H)	0.90	0.80	1.00	0.060	0.94	0.85	1.05	0.269				
	3(M)	0.92	0.88	0.97	0.003	0.98	0.94	1.03	0.507				
	3(H)	0.89	0.80	0.98	0.015	0.96	0.87	1.05	0.322				
Croup	0(M)	0.85	0.76	0.95	0.005	0.71	0.49	1.04	0.079				
	0(H)	0.79	0.63	0.98	0.032	0.48	0.20	1.17	0.106				
	1(M)	0.88	0.81	0.95	0.001	0.72	0.55	0.94	0.017				
	1(H)	0.95	0.82	1.09	0.466	0.58	0.33	1.03	0.061				
	2(M)	0.86	0.81	0.92	<.0001	0.74	0.60	0.92	0.007				
	2(H)	0.93	0.83	1.05	0.228	0.69	0.45	1.06	0.093				
	3(M)	0.85	0.81	0.90	<.0001	0.73	0.60	0.88	0.001				
	3(H)	0.93	0.84	1.03	0.159	0.72	0.50	1.04	0.076				

Health Outcomes	Lag	EDA				Hosp				AC			
		RR	95%CI		P-Value	RR	95%CI		P-Value	RR	95%CI		P-Value
Respiratory Arrest	0(M)									1.28	0.80	2.05	0.294
	0 (H)									0.53	0.13	2.12	0.367
	1(M)									1.22	0.88	1.71	0.237
	1(H)									0.52	0.19	1.39	0.193
	2(M)									1.14	0.86	1.51	0.355
	2(H)									0.95	0.53	1.73	0.878
	3(M)									1.10	0.86	1.41	0.437
	3(H)									1.11	0.68	1.79	0.679
Cardiovascular	0(M)	0.97	0.94	1.00	0.050	1.00	0.95	1.04	0.847	0.97	0.93	1.02	0.257
	0(H)	1.06	1.01	1.12	0.030	1.06	0.98	1.15	0.145	0.95	0.86	1.05	0.296
	1(M)	1.00	0.98	1.02	0.681	0.96	0.90	1.02	0.213	0.96	0.93	1.00	0.030
	1(H)	1.05	1.01	1.09	0.008	0.95	0.90	1.01	0.103	0.97	0.91	1.04	0.388
	2(M)	1.00	0.98	1.01	0.749	0.99	0.97	1.02	0.647	0.96	0.93	0.98	0.002
	2(H)	1.06	1.02	1.09	0.001	1.05	1.00	1.10	0.051	1.01	0.95	1.06	0.832
	3(M)	1.00	0.99	1.02	0.759	1.00	0.97	1.02	0.676	0.97	0.95	1.00	0.020
	3(H)	1.05	1.02	1.08	0.001	1.03	0.99	1.08	0.117	1.00	0.96	1.05	0.888
Arrhythmia	0(M)	0.99	0.90	1.09	0.801	1.06	0.96	1.17	0.226	0.91	0.81	1.02	0.098
	0(H)	1.01	0.84	1.22	0.888	1.26	1.05	1.50	0.011	0.95	0.76	1.20	0.684
	1(M)	0.99	0.93	1.06	0.789	1.05	0.98	1.12	0.169	0.89	0.82	0.97	0.005
	1(H)	1.02	0.89	1.16	0.767	1.19	1.05	1.36	0.006	0.88	0.75	1.04	0.142
	2(M)	0.97	0.92	1.03	0.342	1.01	0.95	1.07	0.762	0.90	0.84	0.96	0.002
	2(H)	0.98	0.88	1.10	0.735	1.15	1.04	1.28	0.008	0.94	0.83	1.07	0.375
	3(M)	0.97	0.93	1.02	0.273	1.01	0.96	1.06	0.609	0.90	0.85	0.95	0.000
	3(H)	0.99	0.90	1.09	0.830	1.11	1.01	1.21	0.033	0.94	0.84	1.06	0.304

Health Outcomes	Lag	EDA				Hosp				AC			
		RR	95%CI		P-Value	RR	95%CI		P-Value	RR	95%CI		P-Value
Angina	0(M)	0.91	0.80	1.04	0.176	1.02	0.88	1.17	0.804	1.22	0.83	1.80	0.305
	0 (H)	0.80	0.60	1.07	0.128	1.00	0.76	1.33	0.982	0.17	0.02	1.18	0.073
	1(M)	0.91	0.82	1.00	0.042	1.04	0.94	1.15	0.406	1.12	0.84	1.49	0.443
	1(H)	0.89	0.73	1.07	0.217	0.93	0.75	1.14	0.468	0.51	0.23	1.15	0.105
	2(M)	0.90	0.84	0.98	0.011	1.06	0.97	1.14	0.188	1.07	0.84	1.36	0.575
	2(H)	0.92	0.79	1.07	0.275	0.99	0.84	1.17	0.901	1.04	0.65	1.66	0.864
	3(M)	0.92	0.86	0.98	0.011	1.06	0.99	1.13	0.114	1.16	0.95	1.41	0.151
	3(H)	0.89	0.78	1.02	0.089	1.01	0.88	1.16	0.891	1.14	0.77	1.68	0.508
Stroke	0(M)	0.87	0.69	1.10	0.231	1.13	0.97	1.30	0.107	0.94	0.79	1.12	0.500
	0(H)	0.89	0.57	1.40	0.619	0.91	0.67	1.24	0.564	0.84	0.59	1.21	0.360
	1(M)	0.91	0.78	1.07	0.259	1.04	0.93	1.15	0.479	0.91	0.80	1.03	0.149
	1(H)	0.78	0.55	1.10	0.151	1.07	0.87	1.31	0.509	0.83	0.64	1.07	0.153
	2(M)	0.92	0.80	1.05	0.193	1.05	0.96	1.14	0.267	0.93	0.84	1.03	0.187
	2(H)	0.78	0.59	1.03	0.080	1.04	0.88	1.23	0.653	0.81	0.66	1.01	0.057
	3(M)	0.94	0.84	1.05	0.265	1.04	0.97	1.12	0.283	0.97	0.89	1.06	0.551
	3(H)	0.87	0.69	1.10	0.241	0.95	0.82	1.10	0.496	0.86	0.72	1.03	0.107
Heart Failure	0(M)	0.88	0.77	1.00	0.054	1.00	0.90	1.11	0.988				
	0(H)	0.99	0.78	1.26	0.958	1.07	0.89	1.29	0.481				
	1(M)	0.93	0.84	1.02	0.102	1.02	0.94	1.09	0.689				
	1(H)	0.95	0.80	1.13	0.552	1.06	0.92	1.21	0.428				
	2(M)	0.92	0.85	0.99	0.036	1.00	0.94	1.06	0.983				
	2(H)	0.98	0.85	1.12	0.744	1.02	0.91	1.14	0.739				
	3(M)	0.94	0.88	1.00	0.050	0.98	0.93	1.04	0.568				
	3(H)	0.95	0.84	1.07	0.424	1.03	0.93	1.13	0.593				

Health Outcomes	Lag	EDA				Hosp				AC			
		RR	95%CI		P-Value	RR	95%CI		P-Value	RR	95%CI		P-Value
Acute Coronary Syndrome	0(M)	1.00	0.88	1.14	0.951	0.99	0.89	1.11	0.911				
	0(H)	0.94	0.73	1.21	0.639	1.03	0.83	1.27	0.800				
	1(M)	1.03	0.95	1.13	0.471	1.01	0.93	1.09	0.883				
	1(H)	0.99	0.83	1.18	0.907	1.01	0.86	1.17	0.938				
	2(M)	1.02	0.95	1.10	0.629	0.99	0.93	1.06	0.806				
	2(H)	1.00	0.87	1.16	0.974	1.06	0.94	1.20	0.341				
	3(M)	1.00	0.94	1.07	0.925	0.94	0.94	0.94	0.750				
	3(H)	0.98	0.87	1.11	0.776	0.94	0.94	0.94	0.543				
Transient Ischemic Attack	0(M)	0.91	0.77	1.08	0.278	1.04	0.85	1.27	0.708				
	0(H)	0.88	0.62	1.24	0.465	1.10	0.75	1.61	0.618				
	1(M)	0.88	0.78	1.00	0.044	0.96	0.83	1.12	0.608				
	1(H)	1.03	0.82	1.29	0.816	1.10	0.84	1.44	0.496				
	2(M)	0.90	0.82	1.00	0.044	0.96	0.85	1.09	0.545				
	2(H)	1.07	0.89	1.28	0.456	1.04	0.83	1.31	0.703				
	3(M)	0.95	0.87	1.03	0.234	1.03	0.93	1.14	0.583				
	3(H)	1.04	0.89	1.22	0.600	1.05	0.87	1.28	0.610				
Cardiac Arrest	0(M)									1.06	0.90	1.24	0.490
	0(H)									0.89	0.64	1.25	0.511
	1(M)									1.07	0.95	1.20	0.259
	1(H)									0.79	0.61	1.01	0.061
	2(M)									1.05	0.96	1.16	0.285
	2(H)									0.86	0.71	1.05	0.137
	3(M)									1.07	0.98	1.16	0.113
	3(H)									0.88	0.74	1.04	0.120

* In ambulance data, Respiratory Tract Infections.

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