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Forest Fire Smoke Exposures and Out-of-Hospital Cardiac Arrests in Melbourne, Australia: A Case-Crossover Study

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Running title: Does forest fire smoke increase cardiac arrests?

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Abstract

Background: Millions of people can potentially be exposed to smoke from forest fires, making this an important public health problem in many counties.

Objective: This study aims to measure the association between out-of-hospital cardiac arrest (OHCA) and forest fire smoke exposures in a large city during a severe forest fire season, and estimate the excess OHCA's due to the fire smoke.

Methods: The association between particulate matter (PM) and other air pollutants and OHCA was investigated using a case-crossover study of adults (>35 years) in Melbourne, Australia. Conditional logistic regression models were used to derive estimates of the percent change in the rate of OHCA associated with an IQR increase in exposure. From July 2006 to June 2007 OHCA data were collected from the Victorian Ambulance Cardiac Arrest Registry. Hourly air pollution concentrations and meteorological data were obtained from a central monitoring site.

Results: There were 2046 OHCAs with presumed cardiac aetiology in our study period. Among men during the fire season, greater increases in OHCA were observed with interquartile increases in the 48-hour lagged $PM_{2.5}$ (8.05%; 95%CI: 2.30, 14.13%; IQR=6.1 $\mu g/m^3$), PM_{10} (11.1%; 95%CI: 1.55, 21.48%; 13.7 $\mu g/m^3$), and CO (35.7%; 95%CI: 8.98, 68.92%; 0.3 ppm). There was no significant association between the rate of OHCA and air pollutants among women. 174 'fire-hours' (i.e. hours in which Melbourne's air quality was affected by forest fire smoke) were identified during 12 days of the 2006/2007 fire season and 23.9 (95% CI: 3.1, 40.2) excess OHCA were estimated to occur due to elevations in $PM_{2.5}$ during these 'fire-hours'.

Conclusions: This study found an association between exposure to forest fire smoke and an increased in the rate of OHCA. These findings have implications for public health messages to raise community awareness and for planning of emergency services during forest fire seasons.

Introduction

Millions of people worldwide can potentially be exposed to seasonal high levels of smoke from forest (bush or wild) fires, making this an important public health problem. As forest fires are predicted to increase in frequency and severity (Confalonieri et al. 2007) and smoke from these fires can travel long distances, it is important to understand the impact of these seasonal high peak smoke concentrations

The smoke from forest fires consists of many different constituents, but the pollutant most significantly increased during smoke episodes is $PM_{2.5}$ – particles with an aerodynamic diameter smaller than 2.5 micrometres (μm) (Reisen et al. 2011). On days without forest fire smoke, $PM_{2.5}$ makes up approximately 40% of PM_{10} (particles with an aerodynamic diameter smaller than 10 μm) (Chan et al. 2008). During forest fires, this proportion increases dramatically(Reisen et al. 2013). Studies around the world have observed particulate matter (PM) concentrations during forest fires well above the recommended air quality standards (Johnston et al. 2011; Morgan et al. 2010; Reisen et al. 2011; Reisen et al. 2013; Sapkota et al. 2005; Schranz et al. 2010). The WHO 24 hour average air quality guideline for PM_{10} is 50 $\mu g/m^3$ and for $PM_{2.5}$ 25 $\mu g/m^3$. Other pollutants that are increased during forest fire smoke episodes, but not to the extent of $PM_{2.5}$, are Ozone (O_3) and Carbon monoxide (CO) (Dutkiewicz et al. 2011; Reisen et al. 2011).

A recent review concluded that several studies have found associations between forest fire smoke and respiratory morbidity (Dennekamp and Abramson 2011), however only a few studies have investigated cardiovascular health outcomes. Of those studies investigating cardiovascular outcomes and forest fire smoke, most of them investigated hospital admissions (Delfino et al. 2009; Hanigan et al. 2008; Henderson et al. 2011; Johnston et al. 2007; Martin et al. 2013;

Morgan et al. 2010; Mott et al. 2005). These studies either showed no association or inconsistent results. Two of the studies have found a weak association with hospital admissions, but only for indigenous people (Hanigan et al. 2008; Johnston et al. 2007). A comprehensive study from Sydney found a small increase in non-accidental mortality at a lag of 1 day after exposure to forest fire smoke (OR 1.05; 95% CI: 1.00, 1.10) (Johnston et al. 2011).

We have previously shown an association between urban $PM_{2.5}$ concentrations and out-of-hospital cardiac arrest (OHCA) (Dennekamp et al. 2010; Straney et al. 2014). This poses the question: what are the health effects of exposure to episodes of forest fire smoke, where the $PM_{2.5}$ concentrations may be many times higher than urban background concentrations and when air quality standards are regularly exceeded?

To our knowledge, this is the first study to investigate the association between OHCA and forest fire smoke exposure. OHCA is potentially a better outcome to investigate than hospital admissions, as most patients who present with a cardiac arrest with presumed cardiac aetiology die before reaching hospital (Stub et al. 2011). As a result, it is likely that hospital studies will miss a substantial number of relevant cases of acute cardiac events.

The South-East of Australia experienced a very severe forest fire season in the summer of 2006/2007, and over 1 million hectares of land were burnt. Smoke from the fires travelled long distances and covered the city of Melbourne on several days. This, together with a detailed ambulance registry on out-of-hospital cardiac arrests in Melbourne, provided a unique opportunity to investigate the association between forest fire smoke and cardiac arrests in a large urban population during a severe forest fire season.

Methods

Study population and outcome data

OHCA data covering July 2006 to June 2007 were drawn from Ambulance Victoria's Victorian Ambulance Cardiac Arrest Registry (VACAR) (http://www.ambulance.vic.gov.au/Research/Latest-Research.html). Ambulance Victoria follow the Utstein criteria, an established a set of common definitions for cardiac arrest (Cummins et al. 1991; Jacobs et al. 2004). The VACAR captures all cardiac arrests attended by the ambulance service (Fridman et al. 2007) and is one of the largest and most comprehensive cardiac registries in the world and includes data on age, sex and exact time of the emergency call on an individual basis.

OHCAs were included if they occurred in Metropolitan Melbourne, had presumed cardiac aetiology and occurred in those 35 years or older. We excluded those younger than 35 because it was more difficult to determine possible cardiac aetiology (due to for example genetic diseases) (Deasy et al. 2011). Of the total number of OHCAs attended by ambulance personnel, about 80% had presumed cardiac aetiology and were included in the analysis (Dennekamp et al. 2010). Reasons for exclusions have been detailed elsewhere (Dennekamp et al. 2010), but included OHCAs due to road traffic accidents or other trauma, overdoses, terminal illness or an underlying respiratory cause.

Ambient air pollution and meteorology data

Hourly average $PM_{2.5}$, PM_{10}, carbon monoxide (CO), ozone (O_3), nitrogen dioxide (NO_2) and sulphur dioxide (SO_2) were obtained from the Environment Protection Agency (EPA) Victoria (www.epa.vic.gov.au) using a central monitoring station in inner suburban Melbourne. Hourly

average observations of temperature and relative humidity were obtained from the Bureau of Meteorology monitoring site at Melbourne Airport.

Fire season and 'fire-hours'

The fire season for the purposes of this paper was defined as the period from November 2006 to March 2007 because this is the annual fire danger season in Victoria. This period has the highest 'Fire Danger Ratings' (which is an indication of how dangerous a fire would be if it started) and the vast majority of days on which total Fire Bans were declared in Victoria occurred within this period (30 Total Fire Ban days were declared in 2006 and 2007, and only 3 fell outside this period).

'Fire-hours' are defined as the periods when the Melbourne population was most likely to have been impacted by forest fire smoke and they can be identified by a combination of chemical transport modelling and observed increases in particle and gas concentrations during forest fires. Details of the criteria for the identification of fire-hours are presented in Supplemental Material (see Supplemental Material, Criteria for the identification of fire-hours, and Supplemental Material, Figures S1 and S2 and Table S1). In summary, fire-hours were identified as those when the hourly $PM_{2.5}$ concentration was greater than 50 $\mu g/m^3$ *and* the hourly carbon monoxide concentration was greater than 50 ppm *and* the back trajectories for air masses at 1000m elevation were in the north west to north east sector (315° to 45°) where the forest fires were occurring.

Statistical analysis

Case-crossover analysis

A case-crossover analysis was conducted using a time-stratified referent period to select control exposures associated with each index case, where case exposure was the exposure in the hour the OHCA occurred. The reference exposures were the exposures in the day and hour of the case on all days falling within the same month and on the same day of the week as the case. This approach eliminated confounding by hour of the day, day of the week and monthly trends, and also of seasonal and long term trends in the exposure variables (Bateson and Schwartz 1999, 2001; Maclure 1991).

OHCA was the binary outcome (dependent) variable in the analysis. The exposure variables were hourly average pollutant concentrations. Analyses were done for lag 0 (hour of arrest), lag 1 (hour prior to arrest), lag 2 etc, and average concentrations of lag 0-2 (average of hour of arrest, lag 1 and lag 2), lag 0-3, lag 0-4, lag 0-8, lag 0-12, lag 0-24 and lag 0-48. In addition, analyses were done using the whole year from 01/07/2006 to 30/06/2007 and for the fire season only (01/11/2006 to 31/03/2007). Stratification was done by sex, age group (35-64, 65-74, 75 years and over), and both age and sex.

Temperature and relative humidity were included as potential confounders. Conditional logistic regression models were used to evaluate the association between the pollutants and OHCA. The parameter estimates from these models may be interpreted as proportional changes in the odds (also referred to as rate in this manuscript), calculated from the odds ratios for the interquartile range of the pollutant. The percentage difference in the odds (rate) was calculated from the OR using the formula: (OR-1) * 100.

As a first step, single pollutant models were developed, followed by multi- pollutant models which included those pollutants that showed associations.

Calculation of excess OHCAs attributable to exposure to $PM_{2.5}$ due to forest fire smoke

Using the 'fire-hour' data, we merged the hourly pollution data such that each pollutant recording had a binary variable indicating whether that hour was associated with a forest fire period. We constructed a second binary variable with a default value of 0, but equal to 1 where the current or any of the preceding 47 hours included 'fire-hours' periods. We calculated the total number of hours where at least one hour in the 48 hours prior was associated with forest fire smoke. This represented the Risk period.

We used two approaches for estimating the number of OHCA attributable to the forest fires: i) a model derived estimate, using the odds ratio for and interquartile range increase of $PM_{2.5}$ levels in the 48 hours preceding the arrest, and ii) a direct calculation based on the difference in the rates between the 'fire-hours and non-'fire-hours'. Details of these two approaches can be found in the Supplemental Material, Methodology: Calculation of excess out-of-hospital cardiac arrests.

All analyses were conducted using Stata (version 12.1, Stata Corp, College Station, TX, USA). P-values <0.05 were considered statistically significant.

Results

Study population and exposure description

A total of 2046 OHCA occurred during the study period (July 2006 to June 2007) in metropolitan Melbourne, 64% were men and the mean age was 71.8 ± 14.2 years. Of these, 783

(38%) occurred during the fire season (November 2006 to March 2007). Men were significantly younger than women when an OHCA occurred, 69.6 vs 75.8 years respectively (p<0.01)

Average hourly concentrations of air pollutants and weather data for the fire season and for the whole year are displayed in Table 1. $PM_{2.5}$ was the pollutant that showed the greatest increase when comparing concentrations between the fire season and non-fire season. Figure 1 presents the hourly $PM_{2.5}$ concentrations in December 2006 and January 2007 and clearly shows the high peak exposures that occurred during exposure to forest fire smoke. The highest hourly $PM_{2.5}$ exposure was 247.2μg/m^3 and it occurred at 3pm on 20/12/2006.

A total of 174 'fire-hours' were identified during the 2006/2007 fire season, over 12 days (Figure 1). The number of 'fire-hours' per day ranged from 4 to 21 hours. The 12 days fell in the period between 09/12/2006 to 10/01/2007. The average $PM_{2.5}$ concentration during the 'fire-hours' was 106 μg/m^3.

Association between air pollution exposure and OHCA

Table 2 displays the results of a percentage increase in the rate of OHCA for an interquartile increase in airborne PM and CO, both for the entire study year (June 2006-July 2007) and for the fire season (November 2006 to March 2007). The results for all analyses can be found in the Supplemental Material, Table S2. An increased risk of OHCA was observed for an interquartile increase in the 48-hour lagged $PM_{2.5}$, both overall (4.4%; 95% CI: 0.2, 8.7%) and among men (7.8%; 95% CI: 2.5–13.3%). Interquartile increases in 24-hour and 48-hour CO levels were also associated with increased risks of OHCA among men. During the fire season, the 48-hour estimated effects of $PM_{2.5}$ remained significant, at 5.4% (95% CI: 0.9, 10.1%). In addition, the 48-hour interquartile increase in CO became significantly associated with the risk of OHCA

(10.0%; 95% CI: 0.6, 20.2%). Among men in the fire season, greater increases in OHCA were observed with interquartile increases in the 48-hour lagged $PM_{2.5}$, PM_{10}, and CO. There was no significant association between the risk of OHCA and any of the air pollutants among women.

The hour of the reported arrest and the interquartile change in ozone for the whole year lagged 2 and 4 hours were associated with increases in OHCA in 65 to 74 year olds for the entire duration of the study (Table 3). During the fire season period, similar results for ozone and OHCA were observed among 65 to 74 year olds, although the confidence intervals were much wider. In addition, among those over-75 years old, 8- and 12-hour lags were associated with an increased risk. For women, 8- and 12-hour lags of an interquartile change in ozone were associated with OHCA during the fire season.

Two pollutant models were developed for 48 hour $PM_{2.5}$ with O_3 and CO. For ozone both the 48 hour lag and the 2 hour lags were investigated (as significant associations were seen in the O_3 single pollutant model for the 2 hour lag) and $PM_{2.5}$ remained significant (4.7%; 95% CI: 0.4, 9.3% and 7.5%; 95% CI: 2.0, 13.3 respectively). However O_3 became non-significant (-4.7%; 95% CI: -18.4, 11.3% and 2.5%; 95% CI: -10.2, 17.0% respectively). The two pollutant models for $PM_{2.5}$ and CO (48 hour average) resulted in both associations becoming non-significant (3.71%; 95% CI: -1.2, 8.9% and 2.0%; 95% CI -6.3, 10.9% respectively). However restricting this analysis to males only resulted in a significant association with $PM_{2.5}$ but not CO (6.7%; 95% CI: 0.5, 13.3% and 3.3%; 95% CI: -7.11, 14.9 respectively). The correlation between $PM_{2.5}$ and O_3 was 0.24 and with $PM_{2.5}$ and CO was 0.37.

Excess arrests attributable to forest fire smoke

Two methods were used to estimate the number of excess arrests that were attributable to forest fire smoke (see Supplemental Material, Methodology: Calculation of excess out-of-hospital cardiac arrests)). Using the model-derived calculation, it was estimated that 23.9 (95% CI: 3.1-40.2) excess arrests were associated with the 'fire-hours' and using the direct calculation it was estimated that 28.9 (95% CI: 3.8, 52.9) excess arrests were associated with the 'fire-hours'.

The mean temperature was higher in the hours that were identified as being affected by forest fire smoke when compared to the other hours throughout the year (21.65 vs 14.53º). However each degree increase in temperature was only associated with a 0.02% increase in risk of OHCA and after adjusting for this, there was no change in the estimated number of excess arrests.

Discussion

This study shows that exposure to forest fire smoke in the 2006/2007 Victorian fire season was associated with an increased risk of having an out-of-hospital cardiac arrest in Melbourne, its capital city with a population of about 4 million. In addition 24 - 29 excess OHCA were estimated to have occurred in Melbourne due to the air quality being affected by smoke from the forest fires.

Although hourly air quality data were available, and several short term averages (including 1, 2, 4, 8 and 12 hours) were investigated, the strongest association was found with the 24 hour and 48 hour rolling averages for $PM_{2.5}$ and CO prior to the OHCA occurring, suggesting that there was a delayed or cumulative association. This association was predominantly seen in men. We are not aware of any plausible reasons why the associations were seen particularly in men and did not have data concerning recognised cardiac risk factors for coronary artery disease. However almost

two thirds of the OHCA during the 2006 / 07 fire season occurred in men, and men having OHCA were significantly younger than women. Our observations could possibly reflect only the higher age-related incidence of coronary artery disease and OHCA, and effects on the background of greater abnormalities of other risk factors in men. Perhaps it is related to confounding factors like smoking or blood pressure, or even other factors like behavioural differences, such as time spent outside could play a role. There are no other studies to date that we are aware of that have investigated OHCA and $PM_{2.5}$ during forest fire smoke events. However of the studies investigating urban $PM_{2.5}$ and OHCA, some have reported a strong association for men (Dennekamp et al. 2010; Ensor et al. 2013) while others did not find this (Rosenthal et al. 2008; Silverman et al. 2010; Sullivan et al. 2003). A study from Launceston looking at cardiovascular mortality found similar gender specific observations. The air pollutiont there was mainly from biomass combustion (residential woodsmoke), but this study found a significant improvement in cardiovascular mortality when air pollution decreased. This was not found in women (Johnston et al. 2013).

For ozone, when analysing all OHCAs, the coefficients were not significantly elevated. Only when stratified by age and sex were significant positive associations observed, and only at rolling averages of 12 hours or less. In the literature, the studies investigating the association between urban O_3 and OHCA show inconsistent results. Some did not find associations (Dennekamp and Abramson 2011; Silverman et al. 2010) and some very recent studies did (Ensor et al. 2013; Raza et al. 2014; Rosenthal et al. 2013). Of the latter, one study found statistical associations with O_3 exposure 2 hours, 24 hours and 72 hours prior to an OHCA (Raza et al. 2014), another only found significant associations with lag 2 days exposures and not hourly

lagged exposures (Rosenthal et al. 2013) and another found associations with both hourly and daily (max 8-hour average) exposure (Ensor et al. 2013; Raza et al. 2014; Rosenthal et al. 2013).

The $PM_{2.5}$ associations found here are relatively similar to our previous study where we investigated the association between urban air pollution and OHCA in Melbourne (Dennekamp et al. 2010). A 4.25 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a a 3.61% increase in risk of OHCA (95% CI: 1.29, 5.99%) in our urban air pollution study and of 3.75% (95% CI: 0.60, 7.00%) during the fire season in the present paper. However even though the estimated effects may be similar the change in air quality concentrations is much larger during forest fire episodes compared to non-forest fire episodes hence resulting in a measureable excess in OHCAs during relatively short smoke episodes.

The estimated effects for $PM_{2.5}$, PM_{10}, O_3 and CO were almost all larger and stronger in the fire season despite the smaller sample size. This association was confirmed by the analysis using 'fire-hours' which showed a significant increase in the number of OHCAs.

Our findings suggest that $PM_{2.5}$ seems to be the key pollutant associated with excess OHCA during forest fires. First of all in our study $PM_{2.5}$ increases the most during a forest fire season compared to any of the other pollutants that were monitored (including PM_{10}). And even with introduction of O_3 and CO, the $PM_{2.5}$ association remained consistent. However this should be interpreted with caution due to the high correlation between the pollutants, in particular between $PM_{2.5}$ and CO (*R=0.37)*.

Previous studies have not shown consistent associations between exposure to forest fire smoke and cardiovascular outcomes (Delfino et al. 2009; Hanigan et al. 2008; Henderson et al. 2011;

Johnston et al. 2011; Johnston et al. 2007; Martin et al. 2013; Morgan et al. 2010; Mott et al. 2005; Schranz et al. 2010). Most of these studies investigated hospital admissions for cardiovascular disease in general or for a specific disease type like myocardial infarction, while ours to our knowledge is the first study to clearly show an association between ambulance data for OHCA and forest fire smoke. Perhaps we find an association here because the vast majority of OHCAs are fatal and therefore these cases do not appear in hospital emergency presentations or hospital admission records. From July 2006 to June 2007 the proportion of OHCA attended by Ambulance in Melbourne (aged ≥35 and presumed cardiac) who were declared deceased at the scene and not transported to hospital was 78.4%. The severe outcome of arrest that could be associated with air pollution would result in a selection bias for studies that relied on hospital admissions.

Although no studies have previously investigated the association between OHCA and forest fire smoke, several studies have investigated urban $PM_{2.5}$ and OHCA with inconsistent findings. No significant associations were found in some (Levy et al. 2001; Raza et al. 2014; Sullivan et al. 2003), another study found null results overall except for a sub-group that had arrests that were witnessed by bystanders (Rosenthal et al. 2008), and a few large studies did find associations between urban $PM_{2.5}$ and OHCAs (Dennekamp et al. 2010; Ensor et al. 2013; Silverman et al. 2010). Even though the present study was rather small, it is likely we found a significant association due to the large $PM_{2.5}$ concentrations that occur during a forest fire season, and hence we were able to detect a significant increase in excess OHCA's.

Our data could not provide further insights into the underlying mechanisms involved. However, these have been extensively reviewed elsewhere (Brook et al. 2010). We have hypothesised that

PM exposure may cause systemic inflammation that can lead to an increase in blood coagulability with resultant coronary thrombosis (Seaton and Dennekamp 2003). In addition the risk of potentially lethal cardiac arrhythmias and cardiac arrest may be increased, possibly partly because of impaired cardiac autonomic control mechanisms. (Brook et al. 2010; Luttmann-Gibson et al. 2010), but this hypothesis is not supported by two large studies with patients with implantable defibrillators that did not find associations between PM levels and tachyarrhytmic events. (Anderson et al. 2010; Metzger et al. 2007)Studies have shown ozone exposure to be associated with disturbed heart rate variability (Kop et al. 2001; Park et al. 2005; Utell et al. 2002)

Strengths and limitations

A major strength of this study is the health outcome data. The Victorian Ambulance Cardiac Arrest Registry is comprehensive as it covers close to 100% of the OHCAs that occur in Melbourne and are attended by ambulance.

A limitation of this study (and with most other air pollution studies) is the use of one central monitoring location in Melbourne for the air pollutant concentrations that was used to represent exposure for the whole of Metropolitan Melbourne. At the time of the study there were only 2 EPA Victoria monitors measuring $PM_{2.5}$, both in inner Melbourne. One of the sites has a significant amount of data missing during the study period whereas the other site had near complete data for all pollutants. Where data were available, we found that the correlation between the two monitors was very high (R=0.95) and therefore the data from one monitoring station was used in this study. The exposure misclassification resulting from this is likely to underestimate the association and the number of attributable arrests. The 'fire-hour' data were

derived from a location 30 km from the EPA monitoring station. This would explain the fact that in Figure 1 it can be seen that on 2 occasions the concentration increases sharply and the 'fire-hour' starts a couple of hours later. However considering our analysis used the 48 hours prior to an OHCA, this is unlikely to have a measurable effect on our results. Modelled data which would more accurately reflect probable exposure in areas that do not have monitoring are recommended for future studies.

Another limitation is that it is not possible to draw conclusions regarding susceptible sub-groups in the community, as we do not have detailed information on individual risk factors and co-morbidities. This is not a problem statistically, as in a case-crossover design these factors are adjusted for by design. However the utility of the findings are reduced as advice can only be provided in general terms, rather than being specific to different risk groups.

In conclusion, the results suggest that exposure to forest fire smoke is associated with the occurrence of out-of-hospital cardiac arrests in men. It is estimated that in the 2006/2007 forest fire season the smoke was responsible for 24 - 29 excess arrests in Melbourne. The impact of this is likely to increase in the future, as forest fires are likely to increase in frequency and severity in many countries where forest fires occur in close proximity to large population centres. These findings have implications for public health messages to raise community awareness and for planning of emergency services during forest fire seasons.

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Table 1. Average of hourly air pollution and meteorological data for the whole year (01/07/2006 to 30/06/2007), for the risk period[a] and for the non-risk period.[b]

Exposure	N whole year	Mean whole year	Mean Risk period	Mean Non-Risk period	Ratio Risk /Non-Risk period	Percentile cutpoint whole year			
						25%	50%	75%	IQR[c]
$PM_{2.5}$ ($\mu g/m^3$)	8590	7.6	32.4	6.3	5.2	2.4	4.8	8.5	6.1
PM_{10} ($\mu g/m^3$)	8618	21.0	55.2	19.2	2.9	11.3	16.9	25.0	13.7
CO (ppm)	8200	0.42	0.51	0.42	1.2	0.2	0.3	0.5	0.3
O_3 (ppb)	8201	17.1	33.3	16.2	2.1	6	16	23	17
NO_2 (ppb)	8226	11.3	8.8	11.4	0.8	5	9	16	11
SO_2 (ppb)	8177	0.84	0.6	0.86	0.7	0	1	1	1
Temperature (ºC)	8708	14.9	21.6	14.5	1.5	10.2	13.9	18.3	8.1
Relative Humidity (%)	8708	64.8	45.6	65.8	0.7	50	68	82	32

[a]Risk period = those hours where at least 1 'fire-hour' occurred in the previous 48 hours. [b]Non-Risk period = Those hours in the whole year except for the Risk period. [c]IQR, interquartile range.

Table 2. Estimated percentage difference in the rate of out-of-hospital cardiac arrest for an interquartile increase in each air pollutant[a] using conditional logistic regression models.[b,c]

Study population	Hourly Lags	$PM_{2.5}$ (%, 95% CI)	PM_{10} (%, 95% CI)	CO (%, 95% CI)
Whole year July 2006 - June 2007				
Total (N = 2046)	0	1.3 (-1.0-3.8)	-0.2 (-4.1-3.8)	-0.6 (-4.8-3.9)
	0-24	3.0 (-0.3-6.5)	3.9 (-1.5-9.6)	2.7 (-3.3-9.2)
	0-48	4.4 (0.2-8.7)*	4.0 (-2.4-10.8)	5.6 (-1.6-13.2)
Men (N = 1311)	0	2.2 (-0.7-5.3)	1.4 (-3.5-6.5)	1.1 (-4.3-6.9)
	0-24	4.9 (0.7-9.3)*	6.6 (-0.2-13.9)	8.0 (0.1-16.6)*
	0-48	7.8 (2.5-13.3)**	8.4* (0.1-17.3)	10.0 (0.6-20.2)*
Women (N = 735)	0	-0.2 (-4.2-3.9)	-2.9 (-9.3-3.9)	-4.1 (0.6-20.2)
	0-24	-0.4 (-6.0-5.5)	-0.9 (-9.6-8.6)	-6.0 (-15.1-4.1)
	0-48	-1.8 (-8.6-5.4)	-3.4 (-13.3-7.7)	-1.4 (-15.1-4.1)
Fire Season (Nov 06 - March 07)				
Total (N = 783)	0	1.9 (-0.6-4.5)	3.0 (-1.4-7.5)	3.9 (-6.0-14.8)
	0-24	3.5 (-0.1-7.3)	7.0 (0.8-13.6)*	16.5 (-0.1-35.8)
	0-48	5.4 (0.9-10.2)*	7.7 (0.3-15.8)*	24.6 (4.5-48.0)*
Men (N = 500)	0	2.5 (-0.7-5.7)	4.5 (-1.0-10.3)	6.9 (-5.8-21.3)
	0-24	4.7 (0.1-9.4)*	8.3 (0.6-16.6)*	24.6 (2.9-50.8)*
	0-48	8.1 (2.3-14.1)**	11.1 (1.6-21.5)*	35.7 (9.0-68.9)**
Women (N = 283)	0	0.9 (-3.3-5.2)	0.4 (1.5-21.5)	-1.3 (-16.2-16.2)
	0-24	1.1 (-4.9-7.6)	4.1 (-6.0-15.3)	0.9 (-22.4-31.4)
	0-48	0.2 (-7.2-8.2)	1.3 (-10.4-14.4)	4.3 (-22.1-39.8)

[a]IQR is based on the distribution of the whole year. IQR are as follows: 6.1 $\mu g/m^3$ ($PM_{2.5}$), 13.7 $\mu g/m^3$ (PM_{10}) and 0.3 ppm for CO. [b]No significant results for less than 24 hour rolling average. [c]Adjusted for temperature and relative humidity.

*$p<0.05$; **$p<0.01$.

Table 3. Estimated percentage difference in the rate of out-of-hospital cardiac arrest for an interquartile increase in ozone (O_3)[a] of 17 ppb using conditional logistic regression models.

	Age groups				Sex	
Hourly Lags	**>35 years**	**35-64 years**	**65-74 years**	**≥ 75**	**Men**	**Women**
Whole year (01/07/06 - 30/06/07)						
0	5.9 (-4.6-17.5)	-7.9 (-23.2-10.3)	43.8 (10.8-86.5)**	7.5 (-7.6-25.0)	6.5 (-6.6-21.3)	6.8 (-10.3-27.0)
0-2	4.3 (-6.1-15.9)	-10.5 (-25.6-7.7)	42.8 (10.4-84.8)**	5.6 (-9.3-23.0)	4.4 (-8.5-19.2)	5.7 (-11.1-25.7)
0-4	3.4 (-7.0-15.1)	-10.7 (-26.2-8.2)	36.1 (5.2-76.0)*	4.9 (-10.0-22.2)	3.9 (-9.1-18.8)	4.2 (-12.8-24.5)
0-8	6.9 (-4.2-19.2)	-5.4 (-22.3-15.2)	25.2 (-3.6-62.6)	10.5 (-5.2-28.8)	3.8 (-9.4-19.0)	13.9 (-5.2-36.8)
0-12	7.9 (-4.1-21.3)	-5.5 (-23.6-16.9)	20.0 (-8.5-57.1)	13.9 (-3.5-34.4)	3.6 (-10.5-19.9)	17.1 (-3.8-42.4)
0-24	4.3 (-8.7-19.2)	-6.8 (-26.9-18.9)	23.7 (-9.1-68.4)	7.3 (-11.0-29.3)	1.8 (-13.8-20.2)	9.6 (-12.3-36.9)
0-48	-0.3 (-14.2-15.9)	-1.8 (-25.0-28.5)	2.4 (-28.2-46.2)	0.6 (-18.6-24.3)	0.7 (-16.5-21.5)	-2.5 (-24.2-25.4)
Fire season (01/11/06 - 31/03/07)						
0	9.1 (-4.7-25.0)	2.4 (-17.6-27.3)	48.9 (2.4-116.6)*	8.5 (-11.4-33.0)	5.4 (-11.2-25.2)	17.8 (-5.8-47.3)
0-2	8.6 (-5.3-24.5)	-2.5 (-22.3-22.4)	57.0 (8.3-127.6)*	9.3 (-10.8-33.9)	4.7 (-12.1-24.7)	17.3 (-5.9-46.3)
0-4	11.5 (-3.0-28.0)	0.1 (-20.9-26.8)	56.2 (8.3-125.3)*	11.8 (-8.6-36.9)	7.9 (-9.6-28.7)	19.4 (-4.7-49.7)
0-8	20.0 (3.8-38.6)	9.6 (-14.6-40.5)	45.5 (0.7-110.3)*	23.4 (0.5-51.5)*	11.2 (-7.2-33.4)	38.8 (9.0-76.7)**
0-12	24.1 (5.4-46.2)	12.0 (-15.5-48.4)	20.9 (-18.1-78.5)	38.1 (9.0-75.1)**	12.4 (-8.5-38.1)	49.0 (13.4-95.7)**
0-24	15.2 (-5.2-40.1)	14.8 (-18.2-61.1)	-8.7 (-43.2-46.8)	30.3 (-1.4-72.2)	5.9 (-17.2-35.5)	33.7 (-3.2-84.6)
0-48	8.1 (-12.5-33.5)	21.2 (-15.3-73.4)	-25.9 (-57.0-27.8)	13.9 (-15.6-53.7)	4.8 (-19.5-36.5)	14.1 (-19.8-62.3)

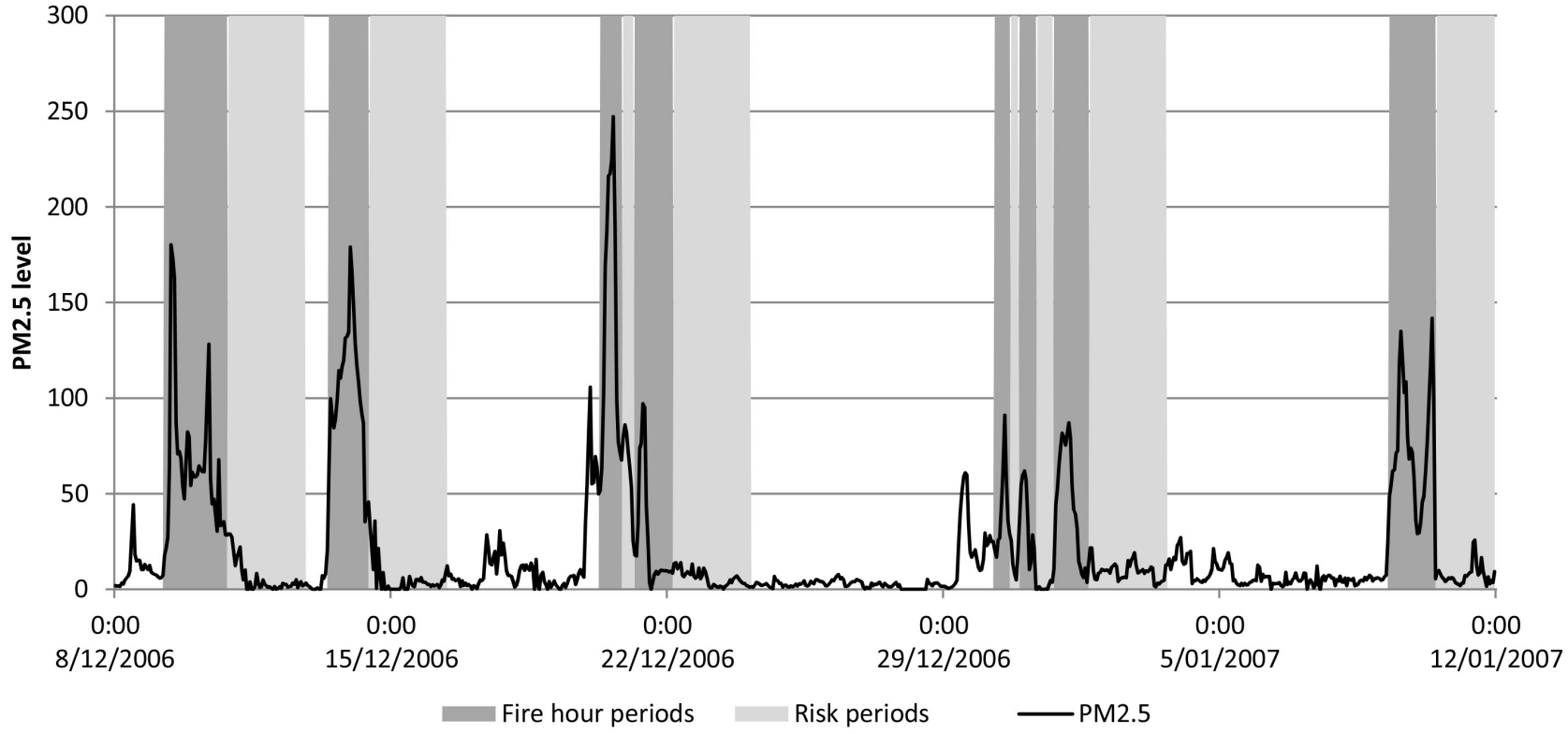
[a]Adjusted for temperature and relative humidity.

*p<0.05; **p<0.01.

Figure Legend

Figure 1. Hourly average $PM_{2.5}$ concentration in Melbourne from 08/12/2006 to 12/01/2007. The dark grey areas represent the 'fire hours' (periods with forest fire smoke), and the light grey areas represent the 'risk period' (at least 1 'fire-hour' in the previous 48 hours)

Figure 1.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Fire hour periods
Risk periods
PM2.5