RESEARCH REPORT

Evaluating the impact of air pollution on the incidence of out-of-hospital cardiac arrest in the Perth Metropolitan region: 2000-2010.

Lahn Straney[1], Judith Finn[1,2,3], Martine Dennekamp[1], Alexandra Bremner[4], Andrew Tonkin[1], Ian Jacobs [2,3]

1. School of Public Health and Preventive Medicine, Monash University, Melbourne, Victoria, Australia;
2. Faculty of Health Science, Curtin University, Perth, Western Australia, Australia;
3. St John Ambulance Western Australia, Perth, Western Australia, Australia;
4. School of Population Health, The University of Western Australia, Perth, Western Australia, Australia.

*** Corresponding author**
Lahn Straney, PhD
Monash University
Level 6, 99 Commercial Road,
Melbourne, Vic 3004 AUSTRALIA
Tel: +613 9903 0317
Fax: +613 9903 0705
Cell: +614 31 374 384
Email: lahn.straney@monash.edu

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Competing Interest
None to declare.

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ABSTRACT

Background: Out-of-hospital cardiac arrest (OHCA) remains a major public health issue. Several studies have found that an increased level of ambient particulate matter smaller than 2.5 microns (PM2.5) is associated with an increased risk of OHCA. We investigated the relationship between air pollution levels and the incidence of OHCA in Perth, Western Australia.

Methods: We linked St John Ambulance OHCA data of presumed cardiac etiology with Perth air pollution data from 7 monitors which recorded hourly levels of particulate matter smaller than 2.5 and 10 microns (PM2.5/PM10), carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen dioxide (NO_2) and ozone (O_3). We used a case-crossover design to estimate the strength of association between ambient air pollution levels and risk of OHCA.

Results: Between 2000 and 2010 there were 8,551 OHCAs that met the inclusion criteria. Of these, 5,624 (65.8%) occurred in men. An interquartile range (IQR) increase in the 24 hour and 48 hour averages of PM2.5 was associated with 10.6% (OR 1.106; 95% CI: 1.038-1.180) and 13.6% (OR 1.136; 95% CI: 1.051-1.228) increases, respectively, in the risk of OHCA. CO showed a consistent association with increased risk of an OHCA. An IQR increase in the 4 hour average concentration of CO was associated with a 2.2% (OR 1.022; 95% CI: 1.002-1.042) increase in risk of an OHCA. When we restricted our analysis of CO to arrests occurring between 6am and 10am we found a 4.4% (95% CI: 1.1%-7.8%) increase in risk of an OHCA.

Conclusion: Elevated ambient PM2.5 and CO are associated with an increased risk of OHCA.

What is already known on this subject?
There is increasing evidence that elevated PM2.5 is associated with an increased risk of out-of-hospital cardiac arrest. However studies of other pollutants have yielded inconsistent results.

What this study adds?
This study provides additional evidence that elevated PM2.5 is associated with an increased risk of OHCA and is the first study that has shown a significant association between carbon monoxide and OHCA risk. In addition, the study demonstrates that the result of misclassification of personal exposure, a problem common to most studies of air pollution, will result in the effect size being biased toward the null.

INTRODUCTION

Out-of-hospital cardiac arrest (OHCA) remains a major public health issue[1] despite the decreasing incidence of coronary artery disease[2]. Although it is nearly 50 years since the advent of cardiopulmonary resuscitation[3], the high case fatality rate of OHCA (>90%) in most communities[4,5], indicates the importance of understanding the immediate precipitants of OHCA, with a view to developing appropriate preventive strategies [6]. It is believed that cardiac arrest is typically caused by a transient factor which often acts on an abnormal myocardial substrate and triggers life-threatening ventricular arrhythmias and possible coronary thrombosis that typically characterise cardiac arrest[7,8]. Various potential triggers have been mooted[8–11]. Among these, there is, growing interest in the possible relationship between ambient air pollution levels and onset of OHCA.

The recently published Global Burden of Disease study reveals that in 2010, ambient particulate matter pollution accounted for 3.1 million deaths and 3.1% of global disability-adjusted life years lost[12]. Ambient air pollution, which ranked as the eighth most significant risk factor for disease burden, is considered to be a risk factor for respiratory tract infections and cancers, ischemic heart disease (IHD), and cerebrovascular disease[13]. Given that IHD is the predominant cause of cardiac arrest[14], it is reasonable to hypothesise an association between OHCA onset and air pollution levels.

Several studies have found that increased levels of ambient particulate matter smaller than 2.5 microns (PM2.5) are associated with increased risk of OHCA [13,15,16], although other studies have reported no association [17,18]. Studies of other air pollutants, such as particulate matter smaller than 10 microns (PM10), carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen

dioxide (NO_2) and ozone (O_3), and risk of OHCA have also reported inconsistent results[16,19–22].

A recent study conducted in Melbourne, the capital of Victoria in Australia [13], reported that an interquartile range increase of 4.26 μg/m^3 in PM2.5 over the 2 days prior to the OHCA was associated with an increase in OHCA risk of 3.6% (Odds ratio (OR) 1.036, 95% confidence interval (CI) 1.013-1.060). However, a limitation of the Melbourne study was that data from one central monitoring station were used to estimate exposure for the entire population. Although Perth, the capital of Western Australia, is a smaller city, data are available from seven air quality monitoring stations. Therefore in the present study, we investigated the relationship between air pollution levels and the incidence of out-of-hospital cardiac arrest (OHCA) in Perth, with arguably a more accurate estimate of individual exposure.

METHODS

Study Population

The study population included all adult OHCA patients in Perth (Western Australia) who were attended by St John Ambulance Western Australia SJA(WA) paramedics between 2000 and 2010. The study cohort was restricted to those over 35 years of age who experienced an OHCA with a presumed cardiac etiology, i.e. cases where the cause of arrest was a known alternative precipitant such as trauma, drowning or a drug overdose were excluded. The date and time of the emergency call to SJA(WA) was taken as the 'date and time of arrest'. Patients who were subsequently identified as having a date of death (as recorded in the WA death register) prior to the date of the emergency call were excluded because of difficulty in

accurately determining the 'time zero' for estimation of exposure. Cases excluded on this basis were most likely to be people who had died hours or even days before being found and the emergency call made.

Setting

Perth is located in the south-western corner of Australia. It is the capital city of the state of Western Australia and has a land area of 5400km^2 [23]. The estimated population of the Perth Statistical Division in 2005 was 1 485 823 persons [24], of whom 49.8% were men and 52% were aged 35 years or older.

Ethics

This study received approval from the University of Western Australia (RA/4/1/2567) and Monash University Human Research Ethics Committees (CF12/3390 – 2012001603); and approval from the St John Ambulance (Western Australia) Research Advisory Group.

OHCA data

All road-based emergency ambulance services in WA are provided by a single ambulance service; SJA(WA). Emergency calls for an ambulance are received by the ambulance service operations centre and computer-aided dispatch (CAD) is used to assign calls to ambulance paramedic crews. During the study period, patient care data was recorded by the paramedics using a paper-based patient care record (PCR). The PCR data were manually entered into the SJA(WA) server and linked to the CAD data for each event.

Clinical and CAD data for all OHCAs are routinely downloaded from the SJA(WA) server, and comprise the SJA(WA) OHCA research database, which is securely housed in the

Discipline of Emergency Medicine at The University of Western Australia in Perth. The date of death from the WA death register is manually added to the records. For this study, the (de-identified) data items extracted from the SJA(WA) database were as follows: patient age, sex and residential postcode; the date and time (to the minute) of the emergency call; whether the arrest was witnessed by bystanders or paramedics; the initial arrest rhythm (e.g. ventricular fibrillation); the arrest location (postcode); the ambulance response time; and whether or not the date of death was prior to the date of the emergency call.

Air Pollution data

The WA Department of Environment and Conservation collect data hourly on air pollution levels, temperature and relative humidity from monitoring stations throughout the Perth metropolitan region. We used data from seven monitors that fell within the metropolitan area serviced by SJA(WA). The location of these monitors is shown on a map provided in the supplementary material (Figure S1). Detailed information on data collection methods and data handling has been published elsewhere [25,26]. Tapered element oscillating microbalance is used for measuring PM2.5 and PM10 levels, gas filter correlation spectrophotometry for CO, ultraviolet absorption for O_3, ultraviolet fluorescence for SO_2, and chemiluminescence for NO_2.

Average hourly concentrations of PM2.5, PM10, O_3, NO_2, SO_2 and CO were collected by at least one of the monitoring stations. Table 1 shows the data collected by each monitor. We compared pollutant levels to the acceptable limits specified in the National Environment Protection Measures (NEPM) using continuous moving averages of pollutant levels, such that in a complete 48 hour period a 24 hour average occurs 25 times (i.e. 1-24, 2-25, 3-26, …, 25-48). This is distinct from their typical application where a 48 hour period would be considered as two 24 hour periods.

(Table 1 here)

We used a commercial database [27] to evaluate distances between the arrest location and the monitoring stations. The database is a matrix of distances in metres between the centres of pairs of Australian postcode areas. We configured this dataset so that it contained the postcode for the area in which each of the seven monitors was located and the Perth postcode for the area in which an arrest may have occurred for each observation.

Given large seasonal variations in temperature and humidity, simple outlier detection was not useful for identifying potential data errors. To deal with this, we developed a temperature prediction model using linear regression. We used hour of the day, month, and year as well as an interaction between month and year to predict hourly temperature. We manually assessed each observation with a residual of >15°C by comparing the observed and recorded values in the preceding and following hours, as well as the temperatures recorded at other monitoring stations at the same time. The value was considered erroneous if it did not fall between the preceding and following recorded values or if there was large inconsistency (>10°C) between the recorded value and those recorded at other monitors. Erroneous values were treated as missing.

Imputation

To deal with missing temperature data we implemented multiple imputation using the *ice* package in Stata (version 12, Stata Corp, College Station, Texas, USA). We validated this approach by recoding a random 10% sample of known values (stratified by monitor) as missing. We repeated the imputation process and compared the imputed values to the observed values using the concordance correlation coefficient [28].

Data linkage

We linked each occurrence of OHCA in the SJA(WA) data to hourly pollution data, matched for day of the week, hour of the day, month and year. This meant that each case was linked to four or five pollution recordings depending on the number of times the day of the week occurred within that month. Case exposure was based on the pollution data recorded on the date of the arrest with the pollution recordings from the other matching days within the month serving as controls. For example, for a case that occurred on a Monday in September at 3pm, the control exposures were all other Mondays at 3pm in September of the same year.

We linked the postcode distance dataset to the arrest data using the location of the arrest. Using this data, each observation was assigned one temperature value and one value for each pollutant using the closest monitor for which data was available. For example, we created a PM2.5 variable that was populated with data from the closest monitor based on the location of the arrest. If those data were missing or not collected, then data from the second closest monitor were used, and so on.

Study design

We used a case-crossover design to estimate the strength of association between ambient air pollution levels and risk of OHCA. The case-crossover study design, originally described by Maclure, estimates the effects of transient changes in an exposure on the risk of a rare acute-onset disease [29]. In this design each index case serves as its own referent. As originally conceived, exposures close in time to the event were contrasted with exposures at a previous time when, typically, the event had not occurred. Selecting referent exposure periods in studies of air pollution poses specific considerations, including seasonal trends, and effects are related to time of day and day of the week. Previous methodological work with air pollution indicates that the least biased method is a time stratified approach with the referent

exposures selected from those that occurred on the same day of the week within the same time strata (e.g. month) [30,22,18].

Statistical Analysis

We used conditional logistic regression models to investigate the relationship between air pollution and the occurrence of an OHCA. The primary outcome was defined as the occurrence of an OHCA attended by SJA(WA) paramedics. Exposure variables were the lagged and pooled average values of PM2.5, PM10, NO_2, O_3, CO and SO_2. We controlled for the mean hourly temperature and relative humidity as potential confounders.

We considered the potential for latency between the exposure and outcome by investigating lagged exposure values of one to three hours, where '3' represents the pollutant value three hours preceding the arrest. In addition we considered averages for different time periods preceding the arrest up to and including the hour of arrest (hour '0'), and created pooled averages for the current hour to four, eight, 12, 24 and 48 hours.

We stratified models by sex, time of day (2am- 6am, 6am- 10am, 10am- 2pm, 2pm- 6pm, 6pm- 10pm, 10pm- 2am), age in three groups (35-64 years, 65-74 years, 75 years and older) and initial arrest rhythm (ventricular fibrillation/ventricular tachycardia (VF/VT) vs non-VF/VT). In addition to the single pollutant models we ran saturated models including all pollutants. In total 1,176 models were run (168 multiple pollutant and 1,008 single pollutant). All statistical tests were two tailed and a p value <0.05 was considered to be statistically significant.

Where we found significant associations, we quantified the impact of the pollutant on the risk of the incidence of OHCA by calculating the percentage increase in risk that would be observed with an interquartile range increase in the concentration of the pollutant.

All analyses were conducted using Stata 12 (StataCorp, College Station, Texas, SA).

Exposure bias evaluation

We tested the impact of using data from multiple monitors to evaluate the association between pollutant concentration and the risk of an OHCA event. We postulated that using data from the closest monitor, when multiple monitors were available in a city, would provide a more accurate measure of exposure than that from a single monitor. We compared the strength and significance of associations between pollutant concentration and the risk of an OHCA event where the monitor was assigned to the case based on the closest distance to the arrest (our default) versus random assignment where the recording was randomly assigned from a single monitor irrespective of distance.

RESULTS

Between 2000 and 2010 there were 8551 OHCAs which met the inclusion criteria (see Figure 1). Of these, 5624 (65.8%) were experienced by men and 2927 (34.2%) by women. The mean and median ages for men were 68.7 and 74.0 years respectively, and 73.7 and 77.0 years for women.

The distribution of ambulance call times is shown in Figure 2. There are two peaks in number of emergency calls, with the primary peak around 10am and the secondary peak at about 6pm.

The percentiles of the average hourly concentrations of the pollutants monitored are presented in Table 2. Using continuous moving averages, between 2000 and 2010, there were 1,464 occasions, (PM2.5, 958; PM10, 501; O_3, 5) when recorded concentrations at the Perth monitors used in this study exceeded the limits specified as acceptable by the National Air

Quality Standards [31]. A breakdown of the number of occasions by monitor and standard has been provided in the supplementary material (Table S1).

(Table 2 here)

Ambient temperature data were missing for 9.7% across all monitors and ranged from 0.6% missing at Wattleup to 48.9% missing at Rockingham. There were no missing data for ambient temperature after running the multiple imputation and the concordance correlation coefficient measuring agreement between our known and imputed values was 0.96. Data for relative humidity were only available from the Caversham monitor and missing for 0.4% of hourly readings and for 0.4% of the matched observations in the SJA(WA) data.

We linked 8551 OHCAs to the pollution data such that the dataset used for the analysis contained 37 689 observations. The mean number of controls per case was 3.4. After assigning pollutant values to each observation, PM2.5 values were missing for 0.27% of observations, 0.06% observations were missing values for O_3, NO_2, CO and SO_2, and 1.06% of observations were missing values for PM10. Correlations between the levels of each pollutant are provided in the supplementary material (Table S2).

The relative odds of cardiac arrest among all observations for each unit increase in pollution are summarised in Table 3. The data values of 0, 1, 2 and 3 represent lagged pollutant values, and the ranges (0-*n*) represent pooled averages from *n* hours before the arrest to the hour of the arrest. The pooled averages of PM2.5 8, 12, 24 and 48 hours prior to the arrest were significantly associated with increased odds of cardiac arrest. Likewise, the pooled averages of CO 1, 2, 3 and 4 hours prior to the arrest were significantly associated with increased odds of cardiac arrest.

(Table 3 here)

We stratified our analysis by time of day, VF/VT vs non VF/VT arrests, sex and age. Although there were some significant associations, there were no more than would be expected to occur by chance. Full details of all single pollutant models have been provided in the supplementary material (Table S3).

In the multi-pollutant models, the 24 hour average of PM2.5 showed a significant positive association at the 5% level among all arrests (Figure 3). An interquartile range (IQR) increase in the 48 hour average of PM2.5 was associated with a 13.6% (OR 1.136; 95% CI: 1.051-1.228) increase in the risk of OHCA. The multi-pollutant models also showed a significant association between risk of an OHCA and the pooled averages of CO 1, 2, 3 and 4 hours prior to the arrest. We also found that the magnitude and significance of these findings were strengthened when we restricted our analysis to only those arrests that occurred between 6am and 10am.

Figure 4 shows the relative odds of an OHCA event for an IQR increase in CO using our multi-pollutant models. Results are stratified by whether the dataset included all arrests or only those occurring between 6am and 10am, the lag (0-1 to 0-4 hour means), and the method for assigning the monitor to the case. Based on these results, an IQR increase in the four hour average concentration of CO was associated with a 2.2% (OR 1.022; 95% CI: 1.002-1.042) increase in the risk of OHCA. When we restricted our analysis to arrests occurring between 6am and 10am we found a 4.4% (OR 1.044; 95% CI: 1.011-1.078) increase in the risk of an OHCA (Figure 4). All models, by lag and strata, have been provided in the supplementary material (Table S4).

In instances where the CO pollutant reading was assigned from a monitor randomly (rather than from the monitor closest to the location of the arrest), effects were attenuated and all associations became non-significant (Figure 4). For PM2.5, the increase in OHCA risk

associated with an IQR increase in remained significant at 10.6% (OR 1.106; 95% CI: 1.038-1.180), but reduced to 7.3% (OR 1.073; 95% CI: 1.013-1.136) when we assigned the monitor randomly.

DISCUSSION

The temporal pattern of emergency calls was similar to a previous report, with most notably an elevated incidence of OHCA in the early hours of the morning [6]. Results of our analysis also imply that there is increased risk of OHCA in conditions of elevated ambient CO and PM2.5. The association between OHCA and CO was no longer statistically significant when we assigned the CO exposure randomly from one of three monitors rather than using the monitor closest to the site of the arrest. This finding has several implications. Firstly, other studies that have relied on a single monitor may not have detected a relationship due to poor estimation of personal exposure. Secondly, personal exposure may differ from ambient levels depending on distance. Thirdly, if we were able to capture true personal exposure the association would be likely to be higher. Fourthly, the increased magnitude of the associations between CO and arrests occurring between 6 and 10 am suggest either that the ambient levels measured by the monitors may better reflect personal exposure during this time or that there may be a threshold level such that a minimum concentration is necessary to see an effect.

Although not directly comparable, our results are consistent with earlier work on PM2.5 by Dennekamp et al in Melbourne, (who analysed the mean PM2.5 level on the day of and day prior to the arrest rather than the 24 hours preceding the hour of arrest) [13]. Our findings suggest a larger increase in risk associated with an IQR increase in the 24 hours average of PM2.5 (10.6% vs 3.6%). Part of the difference in risk (25.4%) is explained by the fact that the

PM2.5 IQR was larger in Perth than Melbourne (5.08 vs 4.26). Median levels of PM2.5 were also higher in Perth than Melbourne (6.80 vs 4.77 $\mu g/m^3$) and this might suggest that there is a threshold effect where the relationship is moderated by the increase and the absolute level of PM2.5. The associations between OHCA and PM10 were attenuated in the multi-pollutant models. This is probably due to the correlation between PM2.5 and PM10 levels (R^2=0.623).

To our knowledge only one study has investigated the association between OHCA and hourly CO concentrations [16]. This study, set in Copenhagen, had a higher hourly median CO concentration (0.25 ppm vs 0.11ppm in Melbourne) though it did not find a significant association with OHCA risk. One possible reason for this may be that the pollutant values were sourced from one central monitor, and it's possible that this estimated personal exposure less accurately. In addition, the Copenhagen study had about half the number of participants as the current study and given the small effect size it's possible that the study was underpowered to detect a significant association.

There are a few other studies that have investigated the association between air pollution and OHCA or cardiac deaths [15–18,32,33]. The results of these studies are not consistent. Two studies in Seattle and another in Copenhagen did not find significant associations between increased levels of air pollution (including PM2.5 and CO) and OHCA [16,17,22]. A study in New York found a significant association between daily PM2.5 and OHCA in the warm season, but the association with CO was weaker (and not significant). On the other hand, a study in Rome reported an association between daily PM10 and CO (PM2.5 was not measured) with out-of hospital coronary deaths. Although a study in Indianapolis did not find any association with daily PM2.5, it did find an association with the PM2.5 concentration during the hour of the OHCA, but only if this was witnessed by bystanders. The study did not investigate other pollutants.

The present study had several unique features that may have had important implications for these results. Firstly, we found that the hourly distribution of PM2.5 levels was different at Quinn's Rock (the most northern monitor and located on the coast) compared with other monitors (appendix figures). Given its proximity to the ocean, we considered that the difference may be partly due to sea salt being recorded at this monitor. We tested the sensitivity of our results by excluding those observations (120 arrests) where the PM2.5 measure was assigned from the Quinn's Rock monitor, but found no significant difference in the results to those reported. Secondly, previous reports have not been able to identify death dates and thus could not exclude observations where the death occurred >24 hours preceding the emergency call. We compared our results including these data and found that the key results highlighted in this report were not sensitive to their exclusion. Thirdly, we note that previous reported associations of PM10 and cardiovascular risk were moderated by high temperature [34,35]. We tested this relationship by restricting our analysis to cases and controls with temperatures in the highest 5%, 10%, 15% and 20% and found no evidence of effect modification in our multi-pollutant models, (the results have been provided in the supplementary material).

Case-crossover studies are known to be subject to bias depending on the method by which referent cases are selected. However, as others have reported, the bias is smallest when selected using a time-stratified approach [22,36]. One of the strengths of our paper was the use of multiple monitors to assign exposure to individual cases. However, while we used data from the closest monitor available, it is possible that this may not accurately reflect true personal exposure. It is likely that environmental factors such as wind and rain, as well as individual variability in exposure due to personal activity and time spent indoors, will influence the true measure of exposure. Nevertheless, as Levy et al noted, while this methodological challenge

is pervasive in research on PM-related health effects, exposure misclassification is unlikely to mask an important exposure-outcome relationship [18]. Similarly we had no data about other risk factors such as smoking, nor any information about existing cardiovascular disease or other comorbidities. However, it is important to note that in up to 50% of OHCA patients, cardiac arrest is the first presenting 'symptom' [37]. Our study population only included OHCA patients attended by SJA(WA) paramedics in response to an emergency call and therefore may not include all OHCAs. For example, patients who arrest en route to the emergency department by private transport are not included. However, previous research has found the frequency of such events is very low [38], and there is no reason to expect that the relationship between air pollution and risk of an OHCA event would be confounded by the mode of transportation.

CONCLUSION

These findings support previous research which indicates that elevated PM2.5 is associated with an increased risk of OHCA. We also found that elevated CO is associated with a smaller but statistically significant increase in the risk of OHCA and that this association is stronger in the morning. Further research is required to more fully understand the association between pollutant levels and personal exposure, the putative biological mechanisms and the subsequent risk of cardiac arrest.

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Table 1 Data collected by each of the monitoring stations in Metropolitan Perth

Station	$PM_{2.5}$	PM_{10}	O_3	NO_2	CO	SO_2	Ambient Temperature	Relative Humidity
CA	●	●	●	●	●		●	●
DU	●	●	●	●	●		●	
QR	●		●	●			●	
RO			●	●		●	●	
SL	●	●	●	●	●	●	●	
SW			●	●			●	
WT						●	●	

Caversham (CA), Duncraig (DU), Quinns Rock (QR), Rockingham (RO), South Lake (SL), Swanbourne (SW), Wattleup (WT)

Table 2. Descriptive Hourly Air Pollution Data (01/01/2000 to 31/12/2010)across all 7 monitors in Perth

		Percentile cut point					
	N[a]	5%	25%	50%	75%	95%	IQR[b]
$PM_{2.5}$ ($\mu g/m^3$)	245,664	2.30	4.72	6.80	9.80	17.70	5.08
PM_{10} ($\mu g/m^3$)	246,210	4.99	9.90	14.60	21.00	36.38	11.10
CO (ppm)	275,917	0.00	0.07	0.11	0.23	0.90	0.16
NO_2 (ppb)	647,975	0.00	1.00	3.00	8.10	19.80	7.10
O_3 (ppb)	566,711	1.90	12.80	20.00	27.30	35.00	14.50
SO_2 (ppb)	283,316	0.00	0.10	0.40	0.90	3.50	0.80

[a]Number of observations across all monitors [b]IQR, interquartile range

Table 3 Relative odds of OHCA per unit increase in pollutant concentration among all arrests in single pollutant models

	$PM_{2.5}$			PM_{10}			CO		
Data	**OR**	**95% CI**		**OR**	**95% CI**		**OR**	**95% CI**	
0	0.999	(0.995-1.004)		1.000	(0.997-1.002)		1.095	(1.013-1.183)	**
1	1.001	(0.997-1.005)		1.000	(0.998-1.002)		1.061	(0.981-1.147)	
2	1.001	(0.997-1.005)		0.999	(0.997-1.001)		1.067	(0.985-1.156)	
3	1.002	(0.998-1.006)		0.999	(0.997-1.002)		1.081	(0.996-1.172)	*
0-1	1.000	(0.996-1.005)		1.000	(0.997-1.002)		1.089	(1.003-1.183)	**
0-2	1.001	(0.996-1.005)		0.999	(0.997-1.002)		1.092	(1.001-1.191)	**
0-3	1.001	(0.996-1.006)		0.999	(0.996-1.002)		1.096	(1.001-1.199)	**
0-4	1.003	(0.997-1.008)		0.999	(0.996-1.002)		1.100	(1.002-1.208)	**
0-8	1.006	(1.000-1.011)	**	1.000	(0.997-1.004)		1.090	(0.989-1.201)	*
0-12	1.007	(1.001-1.013)	**	1.002	(0.999-1.006)		1.114	(1.010-1.228)	**
0-24	1.009	(1.002-1.016)	**	1.002	(0.998-1.006)		1.058	(0.938-1.192)	
0-48	1.010	(1.001-1.018)	**	1.001	(0.996-1.006)		1.040	(0.904-1.197)	

	O_3		NO_2		SO_2	
Data	**OR**	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
0	1.000	(0.996-1.003)	1.002	(0.998-1.007)	0.997	(0.988-1.007)
1	0.999	(0.996-1.003)	1.000	(0.995-1.005)	1.001	(0.991-1.010)
2	1.000	(0.997-1.003)	0.997	(0.992-1.001)	0.995	(0.985-1.005)
3	1.000	(0.997-1.004)	0.998	(0.993-1.003)	0.997	(0.987-1.008)
0-1	0.999	(0.996-1.003)	1.001	(0.997-1.006)	0.999	(0.988-1.009)
0-2	1.000	(0.996-1.003)	1.000	(0.994-1.005)	0.996	(0.984-1.007)
0-3	1.000	(0.996-1.004)	0.999	(0.994-1.004)	0.995	(0.983-1.008)
0-4	1.000	(0.997-1.004)	0.999	(0.993-1.004)	0.995	(0.982-1.009)
0-8	1.000	(0.996-1.004)	0.999	(0.993-1.005)	0.991	(0.974-1.009)
0-12	1.000	(0.996-1.005)	0.999	(0.993-1.006)	0.988	(0.967-1.010)
0-24	1.002	(0.997-1.007)	0.997	(0.990-1.004)	0.991	(0.967-1.016)
0-48	1.001	(0.995-1.006)	1.000	(0.992-1.008)	0.9859	(0.957-1.015)

* p<0.1, ** p<0.05, *** p<0.01

FIGURE LEGENDS

Figure 1 Flowchart of patients who met the inclusion criteria

Figure 2 The distribution of time that emergency calls were received for OHCAs attended by SJA(WA) paramedics by hour for the study period 2000-2010

Figure 3 Odds ratio for an interquartile range increase in the 8, 12, 24 and 48 hour mean average concentration of $PM_{2.5}$, PM_{10} and CO among all arrests (multi-pollutant models).

Figure 4 Odds ratio for an interquartile range increase in the 0-1, 0-2, 0-3 and 0-4 hour mean average CO concentration among all arrests and arrests occurring between 6-10am (multi-pollutant models).