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ORIGINAL PAPER

Risk of respiratory and cardiovascular hospitalisation with exposure to bushfire particulates: new evidence from Darwin, Australia

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Abstract The risk of hospitalisation from bushfire exposure events in Darwin, Australia, is examined. Several local studies have found evidence for the effects of exposure to bushfire particulates on respiratory and cardiovascular hospital admissions. They have characterised the risk of admission from seasonal exposures to biomass air pollution. A new, unanalysed data set presented an additional chance to examine unique exposure effects, as there are no anthropogenic sources of particulates in the vicinity of the exposure monitor. The incidence of daily counts of hospital admissions for respiratory and cardiovascular diagnoses was calculated with respect to exposures of particulate matter (PM_{10}), course particulate matter, fine particulate matter (FPM) and black carbon composition. A Poisson model was used to calculate unadjusted (crude) measures of effect and then adjusted for known risk factors and confounders. The final model adjusted for the effects of minimum temperature, relative humidity, a smoothed spline for seasonal effects, 'date' for a linear effect over time, day of the week and public and school holidays. A subset analysis adjusted for an influenza epidemic in a particular year. The main findings suggest that respiratory admissions were associated with exposure to PM_{10} with a lag of 1 day when adjusted for flu and other confounders (RR = 1.025, 95 % CI 1.000–1.051, $p < 0.05$). This effect is strongest for exposure to FPM concentrations (RR = 1.091, 95 % CI 1.023–1.163, $p < 0.01$) when adjusted for flu. Respiratory admissions were also associated with black carbon concentrations recorded the previous day (RR = 1.0004, 95 % CI 1.000–1.0008, $p < 0.05$), which did not change strength when adjusted for flu. Cardiovascular admissions had the strongest association with exposure to same-day PM and highest RR for exposure to FPM when adjusted for confounders (RR = 1.044, 95 % CI 0.989–1.102). Consistent risks were also found with exposure to black carbon with lags of 0–3 days.

Keywords Particulates · Bush fires · Cardiovascular and respiratory hospital admissions · Health risk

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Introduction

Many studies have shown that exposure to atmospheric pollutants, particularly airborne particulates, increases the risk of respiratory and cardiovascular presentations and admissions to emergency departments. Most studies have focused on the response to anthropogenic emissions in urban areas (e.g. EPA Victoria 2001). However, occasionally, natural sources are responsible for elevated levels of particulates arising from natural fires and biomass burning. Effects have been found in

North America, Indonesia and Australia, where seasonal bush fires occur regularly, and populations are repeatedly exposed to elevated particulates from this source (Tham et al. 2009; Delfino et al. 2009; Morgan et al. 2010; Hanigan et al. 2008; Johnston et al. 2007). The repetition of events allows opportunities to test this association.

Exposure to Australian bushfire smoke has been the subject of several previous studies. Recent ecological studies conducted in Darwin, Northern Territory (NT), Australia, found significant increases in asthma hospital presentations to the Royal Darwin Hospital, with increasing PM_{10} concentrations measured in Darwin (Johnston et al. 2002a; Bowman and Johnston 2005). A stronger association than previously reported (a 26 % increase) was found which could be due to the fact that Darwin has no significant sources of atmospheric particulates other than bush fires. Current day PM_{10} and lags of 1, 3 and 5 days were modelled with asthma presentations. A significant increase ($p < 0.05$) in the risk of asthma presentation occurred when PM_{10} was greater than 40 μg/m^3 compared with the baseline category of less than 10 μg/m^3, for same-day and all lagged-day exposures. The unadjusted rate ratio (RR) of 2.21 (95 % CI 1.40–3.52) increased to 2.39 (95 % CI 1.46–3.90) when adjusted for day of week and weekly rates of influenza for same-day exposures. The highest effect was found with a lag of 5 days for concentrations >40 μg/m^3, RR = 2.56 (95 % CI 1.60–4.09) (Johnston et al. 2002a). Following the initial results of this study, Lewis and Corbett (2002) called for further research on the health effects of bushfire smoke for public health interest, in order to enhance warning systems for susceptible populations such as asthmatics.

A further investigation into cardio-respiratory hospital admissions to the same hospital over the three fire seasons of 2000, 2004 and 2005 found a larger measure of effect of PM_{10} on all respiratory admissions in the local indigenous population, (Odds Ratio (OR) 1.17 95 % CI 0.98–1.40, $n = 567$) compared with the general population (OR 1.08 95 % CI 0.98–1.18, $n = 2{,}466$). Likewise for cardiovascular admissions, for ischaemic heart disease, the indigenous population had a strong positive association with exposure to PM_{10}, strongest with a lag of 3 days (OR 1.71 95 % CI 1.14–2.55), whilst overall for the general population, there was no association between same-day PM_{10} and a lag of 3 days and admissions (Johnston et al. 2007).

The availability of additional data sets to test this association in Darwin arose and is the focus of this paper. Using a previously unanalysed exposure data set, from the 1990s at an additional semi-rural monitoring site, similar epidemiological analysis of the relationship between bushfire smoke and hospital admissions can add to the body of evidence regarding this particular exposure. The analysis of the data took place more recently. Data from this site are representative of the semi-urban background particulate levels that a large proportion of the population is exposed to, but has never been subject to epidemiological analysis. By establishing the relative risk (RR) of outcomes from this new data set of exposure during this previous monitoring period, we would be able to test this association and add to the evidence to cover a longer time period from more recent studies (2000–2005).

Methodology

For this analysis, an ecological time series analysis was conducted involving a multivariate analysis using regression modelling, exploring the relationship between exposures and outcomes whilst adjusting for confounders. The relative risk ratio (RR) of an increase in admission counts in relation to increases in the value of the exposure, compared with the baseline or minimum value, is reported.

Exposure measures

An air quality monitoring campaign was conducted by the Commonwealth Scientific and Industrial Research Organisation (CSIRO) and Northern Territory University (now Charles Darwin University) during the years 1993–1998 at Charles Point. The monitoring site was located 25 km west of the city of Darwin on the Cox Peninsula (12°25′S, 130°37′E) (Fig. 1). Particulate pollution in Darwin is regional with high correlation between readings monitored up to 25 km away (Bowman et al. 2007).

Particulate concentrations were measured using stacked filter units with an inlet that provided a 50 % cut-off diameter of 10 μm. The sample was split into the course mode aerosol for particles of between 2 and 10 μm, and a fine mode aerosol of <2 μm. Black carbon (BC) concentrations were measured using a photometer reflectance technique. The sum of the fine

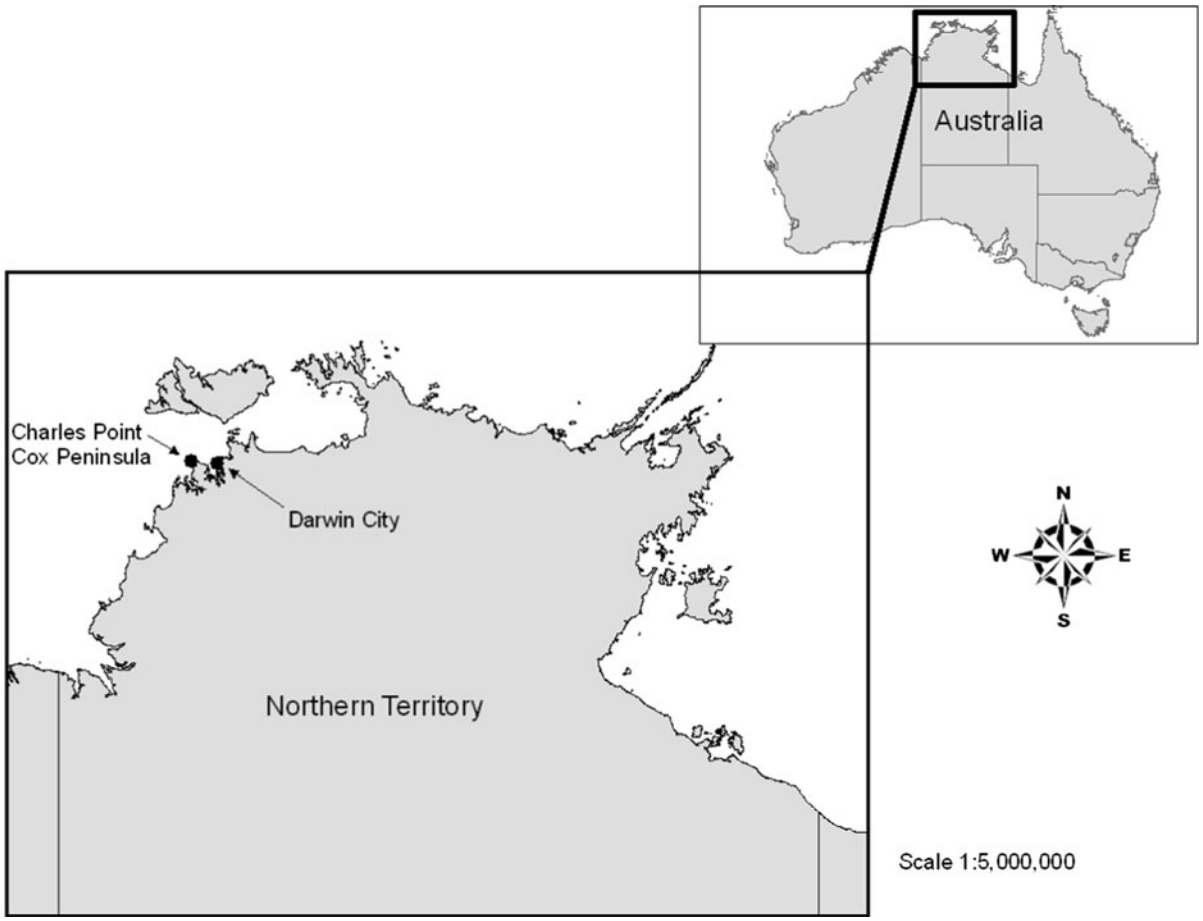

Fig. 1 Study area showing the position of the air quality monitoring station and Darwin City

(FPM) and course mode (CPM) aerosol gives the concentration of particulate matter with less than 10 μm in aerodynamic diameter (PM_{10}), as a 10-μm size inlet was used (Artaxo et al. 1995).

Particulate concentrations from this campaign were provided electronically by Charles Darwin University (CDU), for a period between 1993 and 1996. The data are owned by CDU, CSIRO and Paulo Artaxo, University Sao Paulo, Brazil. Meteorological data were provided by the Bureau of Meteorology, Northern Territory region for Darwin airport.

Electronic records of pollution variables were available from 1 July 1993 to 31 December 1995. Data on CPM, FPM and fine soot or black carbon were retained, and PM_{10} was calculated by the addition of the course and fine fraction (CPM + FPM).

The measurements taken at Charles Point were highly correlated with measurements at Charles Darwin University, in central Darwin for 2005 ($r^2 = 0.74$) (Bowman et al. 2007), suggesting that these measurements are a good proxy for the exposure of the surrounding Darwin population during this study period.

Outcome measures

Hospital emergency admission counts for the Royal Darwin Hospital during the period 1993–1998 were provided by the Northern Territory Government's Department of Health and Community Services. Data were provided for admissions coded according to the International Classification of Diseases version 9 (ICD9). This consisted of emergency admissions with a principle diagnosis of any respiratory and cardiovascular conditions, coded 460–519 and 390–459, respectively. Data for all patients were given, regardless of place of residence, selected for the number of separations (discharge). Emergency department presentations data were not available for this time period.

By sorting on location, the patient's place of residence was reviewed and admissions for patients from non-Darwin locations were eliminated. Patients from other states, overseas or other NT locations were discarded, so that the retained data set represented people that were thought to be exposed to the pollution levels recorded in the Darwin area. This discarded a sizable number of people who are brought to the hospital by air ambulance/flying doctors on the day of admission as their exposure is unknown. Data were then sorted into admission date order and restricted to the study time period. The sum of the number of admissions per day was calculated separately for respiratory and cardiovascular conditions.

Information on periods of flu epidemics (available for 1995 only) and public and school holidays was provided by the Menzies School of Health Research, Charles Darwin University.

Data preparation

The electronic data files were manipulated in MS Excel, and the raw data imported into Stata v8.2 for further manipulation, cleaning and analysis. Data files were merged on the date field so that a value of each variable was linked on date.

Delayed effects, lag structure and categorical data

Hospital admissions are often related to exposure on a previous day to the admission day. Lagged variables were, therefore, generated for all four exposure variables, with a lag of 1, 2 and 3 days.

Continuous variables were reclassified into categories, chosen in bands of increasing 10 $\mu g/m^3$ for PM_{10}, to be consistent and comparable with other studies. The course and fine fraction were classified into three categories based on their distributions. The following categories were chosen for each exposure variable:

- PM_{10} categories: <10 $\mu g/m^3$ (category 1), 10–20 (2), 20–30 (3), >30 (4).[1]
- CPM: <10 (1), 10–20 (2), >20 (3).
- FPM: <5 (1), 5–10 (2), >10 (3).
- BC: <100 ng/m^3 (1), 100–500 (2), 500–1,000 (3), $>1{,}000$ (4).

[1] Only one value of daily PM_{10} was recorded over 40 $\mu g/m^3$.

Both the continuous and the categorical variables were retained and used in the regression modelling process.

Potential confounders and effect modifiers

Meteorological data were selected for the study period obtaining a daily average value for each variable. Table 1 below shows the variables, measurement units and the categories used for continuous variables or codes for dichotomous variables. Categories were chosen in consideration of the distribution of the individual values and in consultation with similar studies (Jalaludin et al. 2002; Johnston et al. 2002a; Schwartz et al. 1996).

Data on other a priori confounders were collected, and variables generated to represent the presence or value of that variable. A list of the dates of public and school holidays in the NT during the study period was used to generate two variables representing the presence of public or school holidays, respectively, coded 0 or 1, as it is known that the occurrence of holidays can influence the number of people being admitted to hospital.

A year variable was generated to identify a linear trend over time in the outcome variable. The day of the week is also known to influence the number of admissions, as there are more admissions on weekdays than weekends, and Mondays are often the highest (Schwartz et al. 1996). 'Day of week' and 'month' variables were created and added to the model to control for weekly and seasonal trends.

Periods of influenza epidemics can influence respiratory admissions, so to control for flu, a variable was created to represent a period of a flu epidemic for 2 months in 1995 based on the number of weekly emergency department consultations for flu or viral illness (F. Johnston, personal communication). The variable was created with data coded 0 or 1 for the days in 1995 only, as flu epidemic data were only available for this year.

Other variables created for time series analysis purposes

As admissions are known to vary with season and also with year, but the relationship may not be linear, semi-parametric approaches can be used to model these variations over time, among other approaches such as

Table 1 Meteorological variables obtained for the analysis and classification of categories

Meteorological variables	Unit of measure	Categories
Daily maximum temperature	Degrees celsius (°C)	<28 (1), 28–31 (2), 31–34 (3), >34 (4)
Daily minimum temperature	Degrees celsius (°C)	<18 (1), 18–22 (2), 22–26 (3), >26 (4)
Dew point temperature	Degrees celsius (°C)	<10 (1), 10–20 (2), 20–25 (3), >25 (4)
Relative humidity	%	<50 (1), 50–80 (2), 80–100 (3)
Precipitation	mm/day	0 (1), 0–20 (2), >20 (3)
Visibility	km	<15 (1), 15–25 (2), >25 (3)
Occurrence of haze, mist or smoke	Y/N	0 (N) or 1 (Y)

using sinusoidal terms to model wavelengths, filtering, diagnostic residual plots and smoothing, for example, by using moving averages (Schwartz et al. 1996). The simplest method for regression modelling is to fit a spline function to model the periods of fluctuations over time and season, so that the 'noise' over this can be detected as the daily fluctuation effect due to pollution and weather. A cubic polynomial smoothing spline was fitted to time intervals to capture local patterns using the 'spbase' function in Stata.

Results

The distributions of daily respiratory and cardiovascular counts, respectively, for admissions to the Royal Darwin Hospital for the study period are explained in Table 2. As the outcome variables are counts per day, being a non-negative integer, a Poisson distribution was assumed for the counts and overdispersion does not occur. A test for this is that the mean and variance are approximately equal. This gives a relative risk ratio (RR) of the counts in relation to increases in the value of exposure or confounder concerned, compared with the baseline or minimum value.

Univariate analysis was first conducted to examine variations in the outcomes with increasing values of exposures and confounder variables. Adjusted measures of effect were then modelled by multivariable analysis for each combination of exposure and outcome. The final adjusted regression model included a smoothing spline with 16 degrees of freedom, to control for season, 'date' to control for linear trends in time, minimum temperature, relative humidity, public and school holidays and day of the week. Other confounding factors did not improve the model. Table 3 gives the results of the adjusted RR using continuous variables of the selective data sets.

The influenza epidemic may have had additional effects on respiratory counts, so a subset analysis on 1995 data for respiratory counts was undertaken. Adjusted RRs of effect were remodelled for this year only, with and without controlling for the flu epidemic (Table 4).

Exposure metrics measured over the study period

Table 5 shows the variations in particulate matter and size fractions over the study period. Table 6 shows summary statistics for each potential confounder measured over the study period. The continuous variables showed clear distinct seasonal patterns (PM_{10}, CPM, FPM, BC, maximum temperature, minimum temperature, dew point temperature, relative humidity, precipitation and visibility). PM_{10} measurements were relatively low during the study period with a peak daily average of 42.5 $\mu g/m^3$, mean of 16.9 $\mu g/m^3$ ($n = 184$).

Particulate matter of all size fractions (PM_{10}, CPM and FPM) was elevated in the dry seasons (April to October). Concentrations of black carbon were consistently low during the wet season (November to March) and higher during the dry season (Fig. 2). This would correspond to the fire season showing continuous bushfire activity in the area over the dry season. PM in Darwin is regional with little spatial variation; levels are driven by emissions from bushfire activity, showing the dominant source of particulate matter in the area.

Temperatures follow a seasonal pattern with maximum temperatures peaking at the end of the dry season/beginning of the wet season, and a secondary peak at the end of the wet season. Minimum temperatures showed a clear seasonal effect with maximums

Table 2 Summary statistics for outcome variables, daily respiratory and cardiovascular counts

Outcomes	*n*	Mean	SD	Variance	Min	Max
Respiratory counts	925	1.80	1.44	2.08	0	8
Cardiovascular counts	925	1.01	1.00	1.00	0	5

n number of observations

Table 3 Adjusted measures of effect for each continuous exposure variable and lagged exposures on both outcomes (to 3 d.p. except where relevant)

Exposure	*n*	Respiratory counts			Cardiovascular counts		
		RR	95 % CI		RR	95 % CI	
			Lower	Upper		Lower	Upper
PM_{10}	182	1.006	0.988	1.024	1.020	0.997	1.043
PM_{10} lag 1	183	1.014	0.996	1.031	0.998	0.976	1.021
PM_{10} lag 2	183	0.996	0.979	1.015	0.993	0.969	1.017
PM_{10} lag 3	183	0.992	0.974	1.009	0.993	0.967	1.018
CPM	181	1.000	0.978	1.022	1.019	0.991	1.048
CPM lag 1	182	1.016	0.993	1.038	0.991	0.963	1.021
CPM lag 2	182	0.998	0.975	1.021	0.980	0.949	1.013
CPM lag 3	182	0.999	0.977	1.021	0.983	0.950	1.017
FPM	181	1.035	0.989	1.083	1.044	0.989	1.102
FPM lag 1	182	1.022	0.981	1.065	1.017	0.968	1.070
FPM lag 2	182	0.985	0.941	1.032	1.021	0.966	1.078
FPM lag 3	182	0.948*	0.903	0.995	1.013	0.955	1.075
BC	182	1.000	1.000	1.000	1.000	1.000	1.000
BC lag 1	183	1.000	1.000	1.000	1.000	1.000	1.0005
BC lag 2	183	1.000	1.000	1.000	1.000	1.000	1.0005
BC lag 3	183	1.000	0.9994	1.000	1.000	1.000	1.0003

*Significant at $p < 0.05$

occurring during the wet season and minimums during the dry season. Dew point temperatures showed the same pattern, but with a larger variation in the minimums during the dry seasons. Relative humidity has a maximum of 100 % during the wet seasons. Visibility, recorded in 5-km bands, is higher in wet seasons than dry, reflecting the higher visibility when particulate matter is lower during this time. Precipitation is zero for most of the year, showing consistently dry periods and monsoonal wet seasons. These meteorological variables all show characteristics of a monsoon climate with periods of dry and wet seasons.

Unadjusted or crude measures of effect were modelled using Poisson regression for the association of each exposure on each outcome variable. This gives the risk ratio (RR) of the increase in counts compared with a one-unit increase in the exposure and the 95 % CI for the true population RR. For continuous data, this represents a risk of hospital admission count for each 1 $\mu g/m^3$ increase in PM_{10}, CPM or FPM, or 1 ng/m^3 increase for BC. For categorical data, this represents an increase in one category (e.g. 10 $\mu g/m^3$ increase in PM_{10}). Both continuous and categorical data were used in the regression models giving the results for only the continuous exposure data for the same-day exposure and lags of 1–3 days for simplicity.

Adjusted measures of effect were modelled for each combination of exposure and outcome. From the univariate analysis, variables found to be associated with both the outcomes and the exposures were retained and adjusted for in the model. Known confounders or risk factors were also retained. As time series studies

Table 4 Adjusted measures of effect for respiratory counts for 1995 only with and without controlling for flu (to 3 d.p. except where relevant)

Exposure	*n*	Respiratory counts without flu			Respiratory counts with flu		
		RR	95 % CI		RR	95 % CI	
			Lower	Upper		Lower	Upper
PM_{10}	67	1.010	0.983	1.038	1.010	0.984	1.040
PM_{10} lag 1	67	1.023	0.998	1.049	1.025*	1.000	1.051
PM_{10} lag 2	67	0.999	0.975	1.025	1.000	0.975	1.025
PM_{10} lag 3	67	0.998	0.973	1.023	0.999	0.974	1.025
CPM	67	1.008	0.975	1.043	1.009	0.976	1.043
CPM lag 1	67	1.020	0.988	1.054	1.022	0.989	1.056
CPM lag 2	67	1.003	0.971	1.040	1.004	0.971	1.038
CPM lag 3	67	1.009	0.977	1.042	1.011	0.979	1.045
FPM	67	1.036	0.964	1.113	1.036	0.964	1.112
FPM lag 1	67	1.086**	1.019	1.156	1.091**	1.023	1.163
FPM lag 2	67	0.984	0.921	1.051	0.984	0.921	1.051
FPM lag 3	67	0.934	0.862	1.012	0.936	0.863	1.014
BC	67	1.000	1.000	1.000	1.000	1.000	1.000
BC lag 1	67	1.0004*	1.000	1.0008	1.0004*	1.000	1.0008
BC lag 2	67	1.000	1.000	1.000	1.000	1.000	1.0005
BC lag 3	67	1.000	0.9992	1.000	1.000	1.000	1.0002

*Significant at $p < 0.05$

**Significant at $p < 0.01$

Table 5 Summary statistics for the exposures measured over the study time period

Exposure (units)	*n*	Mean	SD	Min	Max
PM_{10} ($\mu g/m^3$)	184	16.9	7.7	0.52	42.5
CPM ($\mu g/m^3$)	183	11.6	5.7	0.44	32.3
FPM ($\mu g/m^3$)	183	5.3	3.7	0.08	20.7
BC (ng/m^3)	184	616.9	586.6	3.4	3,384.7

n number of observations

show systematic variations in time, the most important effects to be controlled are season and trend. For short time periods, a linear time trend may be approximated (Schwartz et al. 1996). Meteorological, pollution and health variables will often show systematic variation with seasons. Therefore, year, month and date were initially included in the model to control for trends in time and season. The use of the smoothing spline improved the model, so year and month were substituted, but date retained to control for time.

Meteorological effects are the next largest effects, effecting day-to-day variations. Temperature and relative humidity are widely agreed to be plausibly associated with mortality and morbidity (Schwartz et al. 1996). Minimum temperature was thought to be the most appropriate predictor here, as in hot climates, people's ability to cope with the minimum temperature (i.e. ability to cool down) can determine health effects. Therefore, minimum temperature and relative humidity were retained for the model.

Day of the week, holidays and influenza epidemics are also known to be risk factors in air pollution epidemiology. Day of the week was shown to affect the rates of both outcome counts in the univariate analysis. Public and school holidays can influence the number of people presenting to hospital and therefore need to be retained. Jalaludin et al. (2002) suggest that school holidays in the NT in June and July can

Table 6 Summary statistics for each potential confounder measured over the study time period

Variables (units)	*n*	Mean	SD	Min	Max
Maximum temperature (°C)	925	32.1	1.67	25.4	37.1
Minimum temperature (°C)	922	23.1	2.82	13.1	28.6
Dew point temperature (°C)	925	20.3	5.69	−2	28
Relative humidity (%)	925	70.5	15.29	22	100
Visibility (km)	924	24.4	6.49	0.5	35
Precipitation (mm/day)	925	5.0	16.08	0	178.2
Occurrence of haze, mist or smoke (y/n) (0–1)	886	–	–	0	1
Year (1–3)	925	–	–	1	3
Month (1–12)	925	–	–	1	12
Day of week (1–7)	925	–	–	1	7
Public holiday (y/n) (0–1)	925	–	–	0	1
School holiday (y/n) (0–1)	925	–	–	0	1
Flu epidemic (y/n) (0–1)	365	–	–	0	1

n number of observations

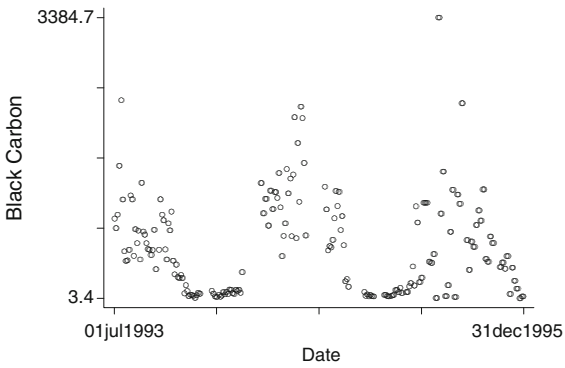

Fig. 2 Values of black carbon (*BC*) measured over the study time period (ng/m^3)

influence hospital admission rates. In reply, Johnston et al. (2002b) found that school holiday periods did not appreciably alter the RR for asthma presentations to hospital in Darwin.

A fully adjusted complex model was reduced to a simpler model by removal of variables that did not improve the model.

Findings and discussion

The levels of PM during the study period were relatively low for an urban population exposure, with a mean daily PM_{10} value of 16.9 μg/m^3 and maximum daily value of 42.5 μg/m^3. This reflects the position of the monitoring site at Charles Point, being remote and reflective of semi-urban exposures of the population in the Darwin area. The values of PM_{10} are below the Australian National Environment Protection Measure (NEPM) standard of 50 μg/m^3 as a 24-h average, designed for the protection of health, suggesting that little or no health effects would be detected. However, WHO (2000) suggest that there is no threshold for PM effects.

Due to the seasonality of PM, and near absence of BC concentrations during the winter season (Fig. 2), bushfire activity is driving the level of PM concentrations at Charles Point. As there is a lack of heavy industry or other significant PM sources in Darwin, the exposure measured here is fairly unique coming predominately from bush fires in the locality. It is important to test health responses in relation to these biogenic emissions. Similar studies of bushfire PM at low concentrations have found significant effects on health (Johnston et al. 2002a, b, 2007; Bowman and Johnston 2005).

Regarding lag exposure effects, crude measures of effect showed that risk of cardiovascular admission significantly increased with exposures of FPM with a lag of 1 day, which was reduced but still significant with a lag of 2 days. Exposure to BC with a lag of 1 day also showed a significant increase in risk of cardiovascular counts.

Adjusted measures of effect

When the crude measures of effect were adjusted for the effects of confounders, the RRs for admission counts were still very close to one (Table 3). After adjustment, a decreased risk of RC and exposure to FPM with a lag of 3 days were significant (RR = 0.948, 95 %CI 0.903–0.995, $p < 0.05$).

The subset analysis of respiratory counts adjusted for flu was more consistent with the previous crude measures of effect (Table 4). The risk of respiratory admissions increased from 1.023 to 1.025 (95 %CI 1.000–1.051) $p < 0.05$, for exposure to PM_{10} with a lag of 1 day. This was consistent across size fractions of PM, with higher RR on exposures with a lag of 1 day. This higher risk was also significant for exposures to FPM with a lag of 1 day (RR = 1.086, 95 %CI 1.019–1.156, $p < 0.01$), and when adjusted for flu, the RR increased and was still significant (RR = 1.091, 95 %CI 1.023–1.163, $p < 0.01$). Exposure to BC with a lag of 1 day resulted in a significant increased risk (RR = 1.0004, 95 %CI 1.000–1.0008, $p < 0.05$) and remained the same after adjustment for flu.

A model of expected counts, comparison to previous studies and mechanism of effect

The equation for modelling the expected counts, based on a Poisson distribution of the outcome variable, is as follows:

$$\text{Log } (\text{E}(Y)) = \beta_0 + \beta_1 X_1 + \beta_p X_p \quad \text{(after Schwartz et al. 1996)},$$

where

- Y is daily respiratory or cardiovascular admission counts
- E(Y) is the expected value of Y on that day
- $X_1,\ldots.X_p$ are the predictors of daily counts, here being exposure, minimum temperature, relative humidity, a smoothing spline, date, day of week, presence of public or school holiday and flu where appropriate.
- $\beta_0 \ldots\ldots \beta_p$ are the regression coefficients for these predictors.

In comparison with the previous air pollution epidemiological study in Darwin for the year 2000 Johnston et al. (2002a), similar elevated risks were found for respiratory admissions to the Royal Darwin Hospital with PM exposures with a lag of 1 day after adjustment for flu. Johnston et al. (2002a) reported the strongest effects for asthma presentations to the same hospital occurred after a lag time of 5 days for exposure to PM_{10} over 40 μg/m^3, measured in Darwin city. Lower concentrations of PM recorded at the Charles Point monitoring site used in this analysis restrict the comparison of these findings. The outcomes measured also differ, being total daily respiratory and cardiovascular admissions, although Johnston's outcome of asthma presentations to the same hospital is likely to be related to the total number of respiratory admissions. Although this study was conducted for a different time period and used exposure data from a different monitoring site, it is comparable to Johnston's previous study as health effects are detected at low PM exposures to the same hospital.

A later, but longer time period study (1996–2005) in the same study area found that the exposure-associated cardio-respiratory hospital admissions varied. Total respiratory admissions were associated with same-day PM_{10}, with a 4.81 % increase per 10 μg/m^3, although the range was very wide (95 % CI−1.04 to 1.01 %). There were generally negative non-significant associations for cardiovascular outcomes. The investigators separated out the effects on non-indigenous and indigenous populations, and after adjusting for ethnicity, indigenous cardiovascular admissions had a positive non-significant effect with lags of 2 and 3 days (Hanigan et al. 2008).

Indigenous admissions were also elevated in a subgroup population analysis over three fire seasons (2000, 2004 and 2005) in Darwin. A case–crossover analysis calculated odd ratios for step increases of 10 μg/m^3 in PM_{10}. Like this study, the same confounders impacted on the rates, including weekly influenza rates and public and school holiday periods, but rainfall was also adjusted for. For a higher exposure rate, with maximum PM_{10} of 70 μg/m^3, a positive relationship was found for respiratory admissions (OR 1.08, 95 %CI 0.98–1.18) with an elevated impact on the indigenous subpopulation (OR 1.17, 95 %CI 0.98–1.40). However, there was no overall relationship with cardiovascular admissions, but indigenous people had positive but non-significant associations with lags of 1–3 days. In general, ischaemic heart disease admissions were significantly decreased overall with same-day exposure (OR 0.82,

95 %CI 0.68–0.98) for the non-indigenous population, but in contrast, were raised in indigenous people, with a significantly different effect with a lag of 3 days (OR 1.71, 95 % CI 1.14–2.55) (Johnston et al. 2007).

In other parts of Australia, the occurrence of bush fires in Victoria in 2003 was found to have a stronger effect on respiratory emergency department attendances ($p < 0.001$) and less effect on hospital admissions ($p = 0.06$). Researchers related the levels of PM_{10}, Airborne Particle Index (API), a measure of fine particulate 0.1–1 μm in diameter and ozone (O_3, daily maximum 4 h averages). A fully adjusted model found the strongest association with PM_{10} and daily respiratory emergency department attendances in Melbourne (RR = 1.018, 95 % CI 1.004–1.033, $p = 0.01$). The association between API and ozone was non-significant. Separate cross-correlations with exposures and admissions showed no significant lag effects; however, cross-correlations with emergency department attendances suggested a 1-day lag effect (Tham et al. 2009).

Delfino et al. (2009) suggested that few studies have evaluated the impacts of wildfire-generated $PM_{2.5}$, which increases the most in concentrations in fire events. They stated that there is little research on the impact of wildfire smoke on cardiovascular outcomes, with two studies finding no significant associations, and conflicting reports on wildfire smoke and total mortality. Whilst $PM_{2.5}$ exposures were modelled from spatial proxies, Delfino et al. found significant respiratory admission associations, notably for asthma, acute bronchitis, COPD and pneumonia, from Southern California wildfires in 2003. Admissions increased at varying ages (most notably in the very young and old) and were strongest with a 2-day average lag. There was limited evidence of a small relative impact of wildfire-related $PM_{2.5}$ on cardiovascular admissions in people aged 45–99, which increased in the post-wildfire period (RR = 1.053, CI 0.994–1.114), possibly due to a delayed impact of the wildfire smoke. This comprised of a small increase in congestive heart failure admissions ($p < 0.01$) and a smaller increase in ischaemic heart disease (both non-significant). Cerebrovascular disease and stroke admissions were positively associated (+1.9 %) both during and after the wildfires (n.s.).

Morgan et al. (2010) on examining bushfire impacts in Sydney found no consistent associations with cardiovascular admissions or with mortality. During two major bushfire events in 1993/1994 and 2001/2002, bushfire PM_{10} was associated with a small increase in all-cause mortality on the same-day of exposure (0.80 % CI −0.24 to 1.86 %), but not cardiovascular or respiratory mortality. Hospital admissions for all cardiovascular diseases, cardiac or ischaemic heart diseases were not associated with bushfire PM_{10}, whereas background PM_{10} concentrations (from urban and other sources) had an association with small increases in both all-age and elderly cardiovascular, cardiac and ischaemic admissions. A small and sustained increase in elderly ischaemic heart disease admissions with same-day bushfire particles (1.20 % CI 0.34–2.07 %) was found and also with lags of 1 and 2 days. Consistent effects were found in the elderly (65+) from exposure to bushfire particulates and respiratory hospital admissions (strongest for COPD and other respiratory conditions) above the underlying effect of exposure from urban sources.

In Europe, a study of the impacts of one of the worst episodes of the decade, from wildfires of Russia, Belarus, Ukraine and the Baltic countries, examined the population effects on 11 provinces in Southern Finland. Hanninen et al. (2009) estimated the burden of the additional fine particulates ($PM_{2.5}$) of 15.7 μg/m^3 during 2 weeks of the summer in 2002 resulted in a 1 % excess mortality (CI 0.5–2 %) increase per 10 μg/m^3 rise, meaning an additional 17 (CI9-34) cases of additional mortality over the 2 weeks. All relative risk regression coefficients were statistically non-significant, but consistent results over various lags suggested an association but are limited due to the short time period of the episode.

Naeher et al. (2007) conducted a systematic review of woodsmoke studies, including studies of forest and brush fires. In general, they concluded that the limitation of all wildfire studies was to reliably characterise population exposures. They suggest that it is possible that there is less respiratory toxicity from bushfire smoke than from forest fire smoke due to chemical and physical differences. However, the outcomes could be influenced by exposures of pollen or mould, and smoke exposures should be analysed for these bio-aerosols. They found, however, that the consistency of effects indicates that the associations between PM_{10} and respiratory health admissions on bushfire days are at least as great as on those days when other sources of particulates dominate.

For this study period, in comparison with studies of urban pollution exposure, the results of our study are similar in strength to a study of hospital admissions in

Melbourne during 1994–1997 (EPA Victoria 2001). Cardiovascular admissions of all ages were found to have a strongest association of a relative risk of 1.0560 (95 % CI 1.0208–1.0924) for a 1 μg/m^3 increase in particles (black smoke particles (BSP)) measured as a 24-h average with a lag of 1 day. Respiratory admissions for all ages had the strongest association with the same-day 24-h average BSP with a relative risk of 1.0239 (95 % CI 0.9927–1.0561).

Some thoughts on the biological plausibility of the results presented here turn our attention to the mechanism of effects. Stronger increased risks were found here with exposure to FPM than PM_{10}, for example, for RC adjusted for flu, RR = 1.025 (95 % CI 1.000–1.051) for PM_{10} and RR = 1.091 (95 % CI 1.023–1.163) for FPM, both with a lag of 1 day. This could reflect the respiratory health response to increased FPM concentrations, as this size aerosol represents the inhalable fraction (<2 μm). Cardiovascular admission counts had a relatively larger and consistent response to the black carbon levels, perhaps reflecting some kind of cardiovascular mechanism occurring with exposure to black carbon. This could have implications for public health in areas where populations are exposed to bushfire smoke, as black carbon emissions are higher from this source of particulates.

Limitations

A number of biases and uncontrolled confounding could have influenced the results presented here. The outcome variables represent all respiratory and all cardiovascular admissions to the Royal Darwin Hospital. They were not broken down to specific diagnosis, such as upper respiratory infections, chronic obstructive pulmonary disease (COPD), bronchitis, asthma or ischaemic heart disease. Previous studies have shown responses of these health outcomes to atmospheric pollution exposure. Using a generalised outcome was necessary for initial analysis for maximum power.

The outcome variable was limited to people with a place of residence in the Darwin area. Non-Darwin locations were omitted, as their exposure could not be quantified. The result was a lowering of the daily counts used in the analysis, and exposure misclassification could arise, as all persons may not have been exposed to these levels of PM (ecological fallacy).

Modelling of cardiovascular counts using a Poisson distribution may be limited as there could be evidence of zero inflation with excess zeros. A zero-inflated Poisson model may have been more appropriate to use. Serial correlation was also not considered here. A number of exposures and their lags were modelled on the two outcomes. Multiple testing problems could arise making significant results by chance due to the number of tests being produced. Caution should, therefore, be taken in interpreting any significant result.

Multipollutant models were not attempted, and effects could be due to other pollutants or their combined effects. The numbers of observations in some of the models are small and reduce the power and reliability of these results. Flu data were only available for 1995 and when adjusted for in the models, made a significant difference to some RRs. It is not known how much influence flu had in other study years.

Conclusion

This study adds to the body of evidence on a unique but important exposure and examined the effects of bushfire particulates on respiratory and cardiovascular hospital admissions. Strong evidence (e.g. Fig. 2) shows that air quality in the Darwin area is driven by bushfire smoke, and this study has shown that hospitalisation may increase at these times, even at low levels of PM exposure. These results for an earlier study period are consistent with the subsequent study period and other similar studies.

Several areas of the world are affected by wildfires, affecting neighbouring densely populated areas, often attracting media attention. The increased pressure of growing populations and expanding urban areas means residential dwellings will continue to encroach into marginal bush land. Climate change also increases this risk with increasing temperatures and vegetation fuel loads. The study presented here helps to quantify this current risk and can be used for modelling future impacts under climate change scenarios.

This study did not consider the effects on vulnerable age groups (under the age of 5 and over the age of 65, etc.), gender, indigenous status and length of hospital stay, to examine variability between groups. Further analysis could also include data on the elemental and ionic composition of the fine and course

PM, to identify larger effects due to specific composition of the particles. The USEPA (2004) reviewed studies where biomass burning emissions were characterised and showed that proportionally higher levels of elemental and organic carbon, chlorine, potassium and zinc emissions arose compared to emissions from combustion sources, for example, from burning diesel and petrol fuel. Differential health responses may occur from exposure to these components, bringing profound implications for controlled bush burning and public health responses. Further epidemiological studies of the effect of bushfire smoke on health outcomes may prove the case for fire management practices (e.g. Bowman and Johnston 2005), for example, in the frequency of prescribed burning to reduce fuel load and wildfire damage.

As there is only one major hospital in the area and only one emergency room, the Darwin study area represents a good area to study effects of atmospheric pollution on health. Any respiratory or cardiovascular patient with severe symptoms in a large catchment area is presented to the Royal Darwin Hospital. Therefore, the health outcomes cannot be diluted as a choice of hospitals does not exist.

Implications for research mirror those arguments presented in Lewis and Corbett (2002) that more studies need to focus on time series analysis on repeatable health outcomes, for example, presentations to ER for asthma or admissions for respiratory or cardiovascular diagnoses. Implications for public health practice recommend preparing medical resources for elevated presentations during bushfire smoke episodes and possible evacuations, health warnings for susceptible populations and development of early warning systems.

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Conflict of interest The author declares that they have no conflict of interest. The guest editors/author declares that they have no conflict of interest with the conference sponsors.

Ethics approval As hospital admission count data are not publicly available, ethics approval was sought and gained from the London School of Hygiene and Tropical Medicine (LSHTM) Ethics Committee for the MSc thesis. Ethics approval was also gained from Charles Darwin University and the Royal Darwin Hospital Ethics Committees.

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