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Air particulate pollution due to bushfires and respiratory hospital admissions in Brisbane, Australia

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Abstracts

A generalised linear model with the negative binomial distribution was used to examine the impact of bushfire smoke on respiratory hospital admissions in Brisbane, Australia from 1 July 1997 to 31 December 2000. The results of this study show that daily respiratory hospital admission rates consistently increased with increasing levels of particles of 10 microns or less in aerodynamic diameter (PM_{10}) for both bushfire and non-bushfire periods. This relationship appeared stronger during bushfire periods than non-bushfire periods, especially for the current day. Compared with the lower level of PM_{10} (i.e. $<15 \text{ ug/m}^3$), the relative risk (RR) for respiratory hospital admissions increased by 9 % and 11% for the medium level (i.e. $15\text{-}20 \text{ ug/m}^3$) and by 19% and 13% for higher level (i.e. $>20 \text{ ug/m}^3$) during bushfire and non-bushfire periods, respectively. The findings suggest that bushfire smoke was associated with an increased risk of respiratory hospital admissions in Brisbane. The health impact assessment needs to be considered in the control and management of bushfires.

Running Title: Bushfire smoke and respiratory hospital admissions

Keywords: Air pollution, bushfire, particulate matter, respiratory hospital admissions, generalised linear model, negative binomial distribution, relative risk

Introduction

Since the infamous London smog event of 1952 (Ministry of Health 1954), many epidemiological studies have demonstrated that exposure to particle air pollution has been linked with reduced pulmonary function, increased respiratory symptoms, increased hospital admissions, increased emergency department visits and even increased mortality (Thurston et al. 1994; Brunekreef et al. 1995; Schwartz 1996; Moolgavkar et al. 1997; Peters et al. 1997; Timonen & Pekkanen 1997; Wordley et al. 1997; Morgan et al. 1998; Atkinson et al. 1999; Jedrychowski et al. 1999; Braga et al. 2001; Michelle et al. 2001; Petroeschevsky et al. 2001; Wong et al. 2001; Kuo 2002). Although the effects of particle air pollution on the risk of respiratory diseases are small, the burden of disease attributable to air pollution may be substantial, as the populations exposed are large and a threshold has not been unequivocally established to date (Schwartz et al. 1994; Wordley et al. 1997).

A number of studies (Brunekreef et al. 1995; Timonen & Pekkanen 1997; Morgan et al. 1998; Atkinson et al. 1999; McGowan et al. 2002) have also indicated the existence of detrimental effects at exposure levels below the current national and international air quality guidelines (ie: 24-hour $PM_{10} \leq 50/ \mu g/m^3$) (NEPC 2004). In general, the health effect estimates observed in the United Kingdom and Europe are lower than those in the United States, and the lowest effects are observed in Australia (Wordley et al. 1997; Petroeschevsky et al. 2001). This may be explained by exposure to different levels of particle air pollution, various compositions of air pollutants and other factors that may influence the association of particle air pollution with respiratory diseases, such as the

susceptibility of the population. These results also suggest that the dose-response relationships determined within a particular country or region may not be readily transferable to other areas.

Although there has been a consistent association between particle air pollution and respiratory diseases, the nature of this association remains unclear. For example, is there any difference in the effects of particulates from traffic or bushfires? If so, which source of particulates, from motor vehicles or bushfires, has a greater impact on respiratory diseases (Pope et al. 1995; Aditama 2000; Jalaludin et al. 2000; Wrobel et al. 2000; Burr et al. 2004)? A few investigations have examined the relationship between exposure to particulate pollution from bushfires and respiratory diseases, but the results were inconsistent (Duclos et al. 1990; Churches & Corbett 1991; Cooper et al. 1994; Smith et al. 1996; Emmanuel 2000; Jalaludin et al. 2000; Johnson et al. 2002). For example, the study by Duclos et al. (1990) found an increase in hospital emergency visits for asthma and chronic obstructive pulmonary disease following the 1997 Californian forest fire. A study in Singapore found an increase in respiratory hospital admissions from smoke haze with an increase in PM₁₀ levels from 50 ug/m³ to 150 ug/m³ (Emmanuel 2000). In Australia, only two of five studies have shown an association between bushfire smoke and certain health outcomes (e.g. asthma) (Churches & Corbett 1991; Cooper et al. 1994; Smith et al. 1996; Jalaludin et al. 2000; Johnson et al. 2002). The study by Churches & Corbett (1991) showed a weak link between particulate air pollution and asthma attendances for a short period (a month) in Sydney during the 1991 bushfire. Subsequently, three studies were conducted in Sydney during the 1994 bushfire. Among these, a study by Cooper et al. (1994) did not

find any increase in asthma presentations after the bushfire event, compared with those before the bushfire. Another study by Smith et al. (1996) used more detailed and complex analysis strategy and concluded that particulate air pollution generated from bushfires did not increase asthma presentations to the emergency departments in Western Sydney. A study by Jalaludin et al. (2000) used a time series analysis to examine the acute effects of bushfire on peak expiratory flow rates (PEFR) in children over a period of one week, and found no association. However, a recent time series study, conducted by Johnson et al (2000) in Darwin showed a significant increase in asthma with each 10 ug/m^3 increase in PM_{10} over 7 months with almost continuous bushfire activity. When the range of studies is considered there appeared to be an impact of bushfire smoke on respiratory diseases, even though many of the previous studies do not report statistically significant findings. Therefore, more research on this issue is needed (Lewis & Corbett 2002).

To assess the possible impact of particle air pollution from bushfires on respiratory disease, this study examined the relationship between exposure to particulate matter and respiratory hospital admissions during bushfire events in Brisbane between 1997 and 2000.

Methods

Air pollution and hospital admissions

Daily data on particulate matter smaller than $10\mu\text{m}$ (PM_{10}) were obtained from the Queensland Environmental Protection Agency for the period from 1 July 1997 to 31 December 2000. There were five monitoring sites to measure PM_{10} concentration in

Brisbane during this period. The monitoring site with the most complete records on PM₁₀ (4.5% missing value) was chosen to be used for this study, which is located at the Central Business District (CBD), near Southeast Freeway. The other four monitoring sites were not included because they have incomplete data on PM₁₀ (missing values 7% - 88%) (Figure 1). The PM₁₀ concentration was measured by a Tapered Element Oscillating Microbalance (TEOM) method. Data on bushfire events during this period were obtained from the Department of Emergency Service, Brisbane City Council. The data included all bushfires from the Brisbane Region, which covers approximately 5,160 square kilometres and includes the local government areas of Brisbane and surrounding areas such as Caboolture, Kilcoy, Pine Rivers, Redcliffe and Redlands. Any events of less or equal to 1 hectare burnt area were excluded from the analysis. This analytical protocol was recommended by the Department of Emergency Service (Angela Higginson, personal communication, April 2004), because such small fires were unlikely to significantly increase the levels of particulate air pollution.

“[Insert Figure 1 about here]”

Routinely collected data on daily respiratory hospital admissions in Brisbane were obtained from the Queensland Department of Health. The sample included all patients with respiratory diseases admitted to both public and private hospitals, from 1 July 1997 to 31 December 2000. All the cases were diagnosed on discharge. The data were categorised using International Classification of Diseases, 9th (ICD-9) and 10th revisions (ICD-10). Selected principal diagnosis for respiratory disease (ICD-9: 460-519 or ICD-

10: J00-599) excluding influenza (ICD-9 487 or ICD-10 J11-11) (National Centre for Health Statistics 1980; WHO 1992) was drawn from the original data set.

Potential confounding variables

Data on meteorological variables were obtained from the Australian Bureau of Meteorology including daily average maximum and minimum temperatures, relative humidity (at 9am), rainfall and wind direction (at 3pm). Other potential confounders such as seasonality, day of the week, holidays, long term trends and influenza were also considered.

Statistical analysis

Missing daily PM₁₀ values were interpolated with the mean values of the same weeks and months. The student t tests were used to compare the difference between the datasets before and after missing values were replaced. There were 1222 days during 1997-2000 recorded with PM₁₀ data. After replacing the missing PM₁₀ values, the mean daily PM₁₀ for the study period was 16.11 µg/m³ compared with 16.08 µg/m³ before replacing missing values. There was no significant difference between the datasets with and without the missing values (p = 0.90), and therefore, a completed set of data with PM₁₀ values was used in this study.

The goodness of fit for both the Poisson and negative binomial distributions of daily hospital admission counts was tested (Johnson et al. 2002; Academic Technology Services 2005). The results indicated that the negative binomial distribution provided a better fit than Poisson distribution (further data are available from the corresponding

author). Therefore, a generalised linear model with the negative binomial distribution was used to estimate the effects of bushfire smoke on respiratory hospital admissions.

To estimate the association between PM₁₀ concentration generated from bushfire smoke and respiratory hospital admissions, PM₁₀ data were categorised in three levels: <15, 15-20, and > 20 µg/m³. These cut-off points were selected for two major reasons: firstly, to ensure reasonable sample size at each level; and secondly, to make the results of this study comparable to other studies. Relative risks (RRs) of respiratory hospital admissions with 95% confidence intervals for increasing levels of PM₁₀ were calculated by comparing the incidence rate of respiratory hospital admissions among subjects in medium and higher levels of PM₁₀ to that in lower levels of PM₁₀. The linear trends were also tested across three PM₁₀ levels.

Multivariate negative binomial models were used to compare daily hospital admissions across the three PM₁₀ levels. Potential confounding factors were taken into account in the multivariate models (Katsouyanni et al. 1996). Only those confounding variables which had a significant impact on the estimates of the RRs (i.e. ≥10% of change in the effect estimate) were included in the final multivariate model. The pattern of residuals by time and the goodness of fit of the model were considered. In addition, a possible lagged effect of one, three and five days was also examined.

The associations between PM₁₀ concentration and respiratory hospital admissions during non-bushfire and overall periods were also examined compared with the bushfire period.

All the data analyses were conducted using the SAS statistical software package version 8.2 (SAS Institute Inc 2002).

Results

Daily respiratory hospital admissions and PM₁₀ concentration

Overall. There were 1280 records of daily hospital admissions during the study period. A total of 42268 patients were admitted for respiratory diseases, with a median of 33 patients per day (range: 6 – 91).

The mean daily PM₁₀ was 16.08 µg/m³ (range: 4.90-60.60 µg/m³) for the study period. There were four days when the PM₁₀ exceeded the National Environment Protection Council standard for maximum mean 24-hour PM₁₀ of 50 µg/m³ during the study period (NEPC 2004). Figure 2 shows the variation of respiratory hospital admissions in relation to PM₁₀ concentrations in Brisbane, 1997-2000.

“[Insert Figure 2 about here]”

Bushfire & non-bushfire periods. A total of 452 bushfire days (35% of the total days) was recorded during the study period (Table I). Among the bushfire periods, the total number of patients with respiratory hospital admission in Brisbane was 15308, with a median of 34 patients per day (range: 9-76). The mean daily PM₁₀ for bushfire period was 18.28 µg/m³ (range 7.50-60.60 µg/m³). There were in total two days when the PM₁₀

exceeded the National Environment Protection Council standard for maximum mean 24-hour PM₁₀ of 50 µg/m³.

“[Insert Table I about here]”

Likewise, for non-bushfire periods, the total number of respiratory hospital admissions in Brisbane was 26960, with a median of 32 patients per day (range: 7-91). The mean daily PM₁₀ was 14.91 µg/m³ (range: 4.90-58.10 µg/m³).

Daily patients by PM₁₀ level during bushfire and non-bushfire days are shown in Figure 3. It appeared that the median number of patients slightly increased with the increasing levels of PM₁₀, and daily number of patients during bushfire days was greater than that during non bushfire days when PM₁₀ levels increased, especially PM₁₀ levels were > 20 µg/m³.

“[Insert Figure 3 about here]”

Relative risk for respiratory hospital admissions

Table II shows the results of the negative binomial regressions of respiratory hospital admissions by PM₁₀ category for the same day, at lags of one, three and five days.

Overall. For the current day, there was a statistically significant linear trend for an increased risk of respiratory hospital admissions with increasing levels of PM₁₀ (p<0.01). This association also appeared at lagged days. However, all the RRs at lagged days were generally smaller than those on the current day (Table II).

“[Insert Table II about here]”

The plot of residuals by time shows that the residuals in the model appeared to fluctuate randomly around zero with no obvious trend and seasonality (Figure 4), which suggests that the model fitted the data reasonably well.

“[Insert Figure 4 about here]”

Bushfire vs non-bushfire periods. During the bushfire periods, statistically significant trends were observed for the RRs of respiratory hospital admissions across different PM₁₀ levels on the current day. For an increase in PM₁₀ from low (<15 µg/m³) to medium (15-20 µg/m³) or high (>20 µg/m³) level, there was an increase in respiratory hospital admissions by 9% to 19% (p<0.01) after adjustment for potential confounders. For lagged days, statistical linear trends were only observed at lags of one and five days (p≤0.03). However, the effects of PM₁₀ at lagged days were generally smaller than those on the current day.

Compared with the lower PM₁₀ level (i.e. <15ug/m³), there was an increase in respiratory hospital admissions by 11% and 13% for the medium (i.e.15-20ug/m³) and higher PM₁₀ level (i.e. >20ug/m³) during non-bushfire periods on the current day, respectively. There were similar associations between PM₁₀ and respiratory hospital admissions at lagged days.

Discussion

The results of this study show that the overall mean PM₁₀ level in Brisbane was 16.1 ug/m³, which was lower than the mean PM₁₀ levels in most cities in North America (Samet et al. 2000), Europe (Ponce de Leon et al. 1996), Hong Kong (Wong et al. 2001), Taiwan (Kuo et al. 2002) and Bangkok (Preutthipan et al. 2004). It was also lower than the mean PM₁₀ level in Sydney (Morgan et al. 1998), Melbourne (Simpson et al. 2000) and Brisbane during the period 1987-1993 (Simpson et al. 1997). The mean PM₁₀ concentration during the bushfire periods was 18.28 ug/m³, which was higher than the PM₁₀ levels (14.91 ug/m³) in non-bushfire periods, but much lower than those in Sydney (Cooper et al. 1994), Darwin (Johnson et al. 2002), Indonesian (Aditama et al. 2000) and Singapore (Emmanuel 2000) during bushfire events. The lower level of PM₁₀ in Brisbane may be explained by its general decline of particulate air pollution and the location of monitoring site used in this study. The monitoring site located at the Central Business District (CBD), near the Southeast Freeway, was within neither the industrial nor bushfire areas. The PM₁₀ in this site was slightly lower than the other four monitoring sites that were not included due to incomplete data on PM₁₀. Moreover, the predominant winds were in an east and north-easterly direction in Brisbane. The monitoring site was upwind of locations where many fires occurred. Therefore, the actual PM₁₀ levels which the population exposed to (particularly during bushfire periods) may be higher than that measured.

During the bushfire periods, the median daily respiratory hospital admissions were also higher than those in non-bushfire days when PM₁₀ concentration increased, particularly when PM₁₀ level was greater than 20 ug/m³. A statistically significant

relationship between PM₁₀ and respiratory hospital admissions was observed for both bushfire and non-bushfire periods. RRs for daily respiratory hospital admissions increased significantly with rising concentrations of PM₁₀ from low (<15 µg/m³) to medium (15-20 µg/m³) or high level (>20 µg/m³), particularly on the current day (RR_{bushfire} =1.09 to 1.19 vs. RR_{non-bushfire}=1.11 to 1.13). The results provide compelling support for the findings in Darwin (Johnson et al. 2002), California (Duclos et al. 1990) and Singapore (Emmanuel 2000), but not for the studies in Sydney (Jalaludin et al. 2000; Smith et al. 1996; Cooper et al. 1994). Compared with previous studies, caution should be exercised due to differences in study designs and statistical methods. For example, many previous bushfire studies (Jalaludin et al. 2000; Smith et al. 1996; Cooper et al. 1994) used indirect measures of particulate pollution, and most of them did not use appropriate time series methods or adequately accounted for confounders (Cooper et al. 1994; Lewis et al. 2002). Moreover, the majority of the previous studies only focused on the association between bushfire events and asthma (Churches & Corbett 1991; Smith et al. 1996; Cooper et al. 1994; Johnson et al. 2002). In addition, most of these studies covered shorter time periods with small sample sizes (Jalaludin et al. 2000; Lewis et al. 2002). It seems that the potential effects of PM₁₀ on respiratory hospital admissions if any would be quite small. And to detect such small effects, a large sample size may be required. Thus, the size of study sample in some previous studies may be not sufficient to detect a weak relationship between air pollution arising from bushfires and respiratory hospital admissions (Duclos et al. 1990; Emmanuel 2000).

Overall, RRs for daily respiratory hospital admissions rose (RR=1.11 to 1.16) significantly with increasing levels of PM₁₀, which is consistent with other studies (Morgan et al. 1998; McGowan et al. 2002; Oftedal et al. 2003; Hagen et al. 2000; Braga et al. 2001). The results of this study also indicate that there appeared to be a dose-response relationship between PM₁₀ concentrations and respiratory hospital admission rates.

The comparison of the health impact of PM₁₀ between bushfire and non-bushfire periods indicates that RRs for respiratory hospital admissions on the current day increased by up to 19% and 13% during bushfire and non-bushfire periods, respectively, when PM₁₀ concentration rose from a low level (<15 µg/m³) to a high level (>20 µg/m³). It seems that air pollution from bushfire events may have a greater impact on respiratory hospital admissions than that from other sources. However, these effects were not apparent at any lagged days. The reason why air pollution from bushfires exhibited a greater impact on respiratory hospital admissions than other sources may be explained by differences in the composition of PM₁₀ and in particular the particle size (PM_{2.5}) distribution between bushfire and non-bushfire days (Emmanuel 2000). The results of this study also suggest that the effect of PM₁₀ generated from bushfires on respiratory diseases is likely to be immediate and short term. Therefore, further research is clearly needed to fully understand the mechanisms of the health effects of bushfires.

This study has four major strengths. Firstly, according to our knowledge, this is the first study that compares the differences in the possible health impact of PM₁₀ between bushfire and non-bushfire periods. Secondly, databases used in this study were quite

comprehensive. For example, only 4.5% of daily PM₁₀ data were missing during the study period. The mean daily PM₁₀ values were similar between the original data and those after replacing the missing data. Therefore, this allowed the linkage and analysis of all relevant data including daily PM₁₀ values, daily counts of respiratory hospital admissions and confounding variables during 1997 - 2000 in this study. Thirdly, the study covered a longer time period than previous bushfire studies (Lewis et al. 2002). Finally, a sophisticated time series analysis methodology was applied. The goodness-of-fit of the model was also examined, which indicates that the model fitted the data reasonably well (Figure 3).

This study also has four limitations. Firstly, PM₁₀ data from a single monitoring site may be not representative of the whole city and may also underestimate the actual air pollution levels in the region. Secondly, we did not have information on any transfers and re-admissions of patients during the study period, which may bias the results. Thirdly, the categorization of PM₁₀ used in this study may be too narrow. This narrow categorisation of PM₁₀ may not be easy to generalise to other regions and study populations. Therefore, caution must be taken when interpreting the findings from this study. Finally, we did not have information about personal exposure; non-differential misclassification is likely to occur although it possibly biases the results towards null.

In conclusion, bushfires may be associated with an increased risk of respiratory hospital admission. The findings of this study, if confirmed, may have implications for the control and management of bushfires. Since the possible health impact of bushfires is of immense public health significance, given global climate change will increase the

frequency and intensity of bushfires in many parts of the world (IPCC 2001), further research on this issue is clearly warranted.

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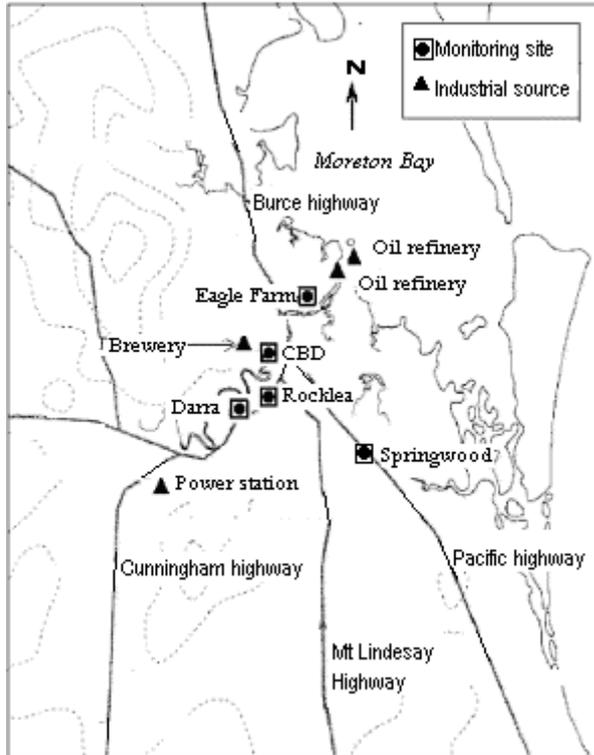
Legends for Figures 1-4:

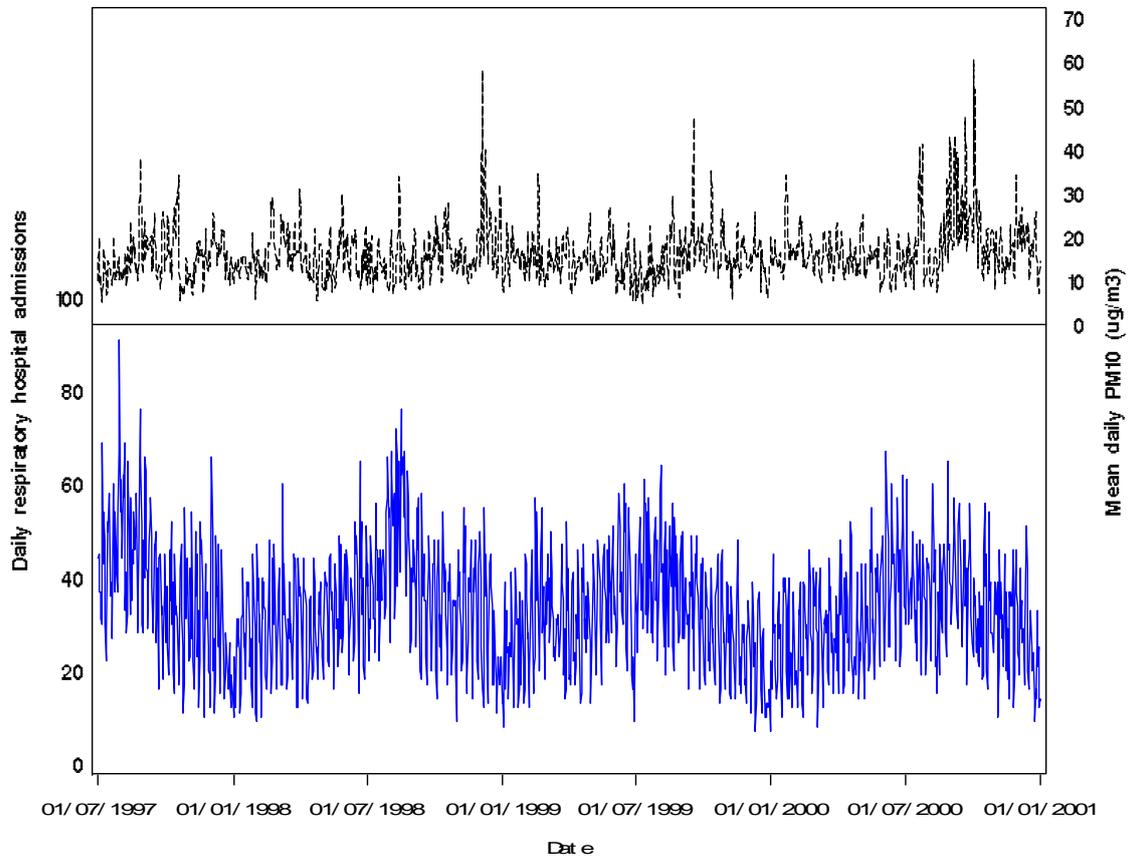
Figure 1. Map of Brisbane region, showing location of air monitoring stations for PM_{10} measurement and pollution sources

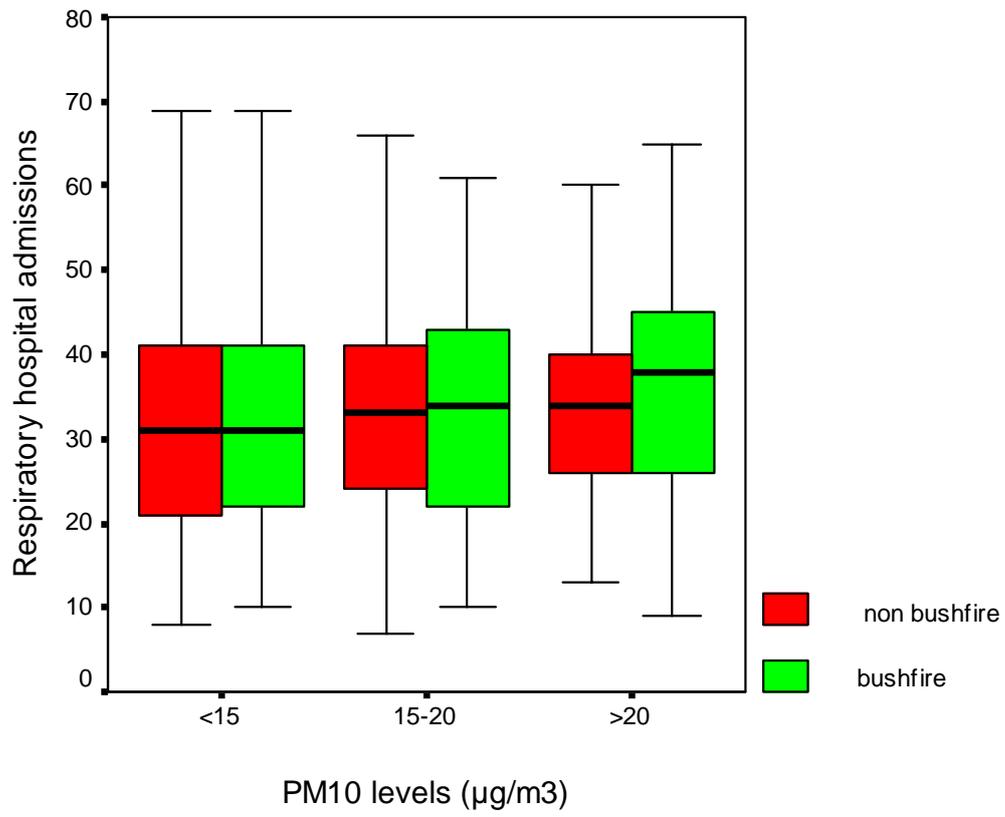
Figure 2. Respiratory hospital admissions and PM_{10} concentrations in Brisbane, 1997-2000

Figure 3. Average number of daily cases by PM_{10} level during bushfire and non-bushfire periods, 1997-2000

Figure 4. Plot of residuals by day with respiratory hospital admissions in multivariate negative binomial regression model







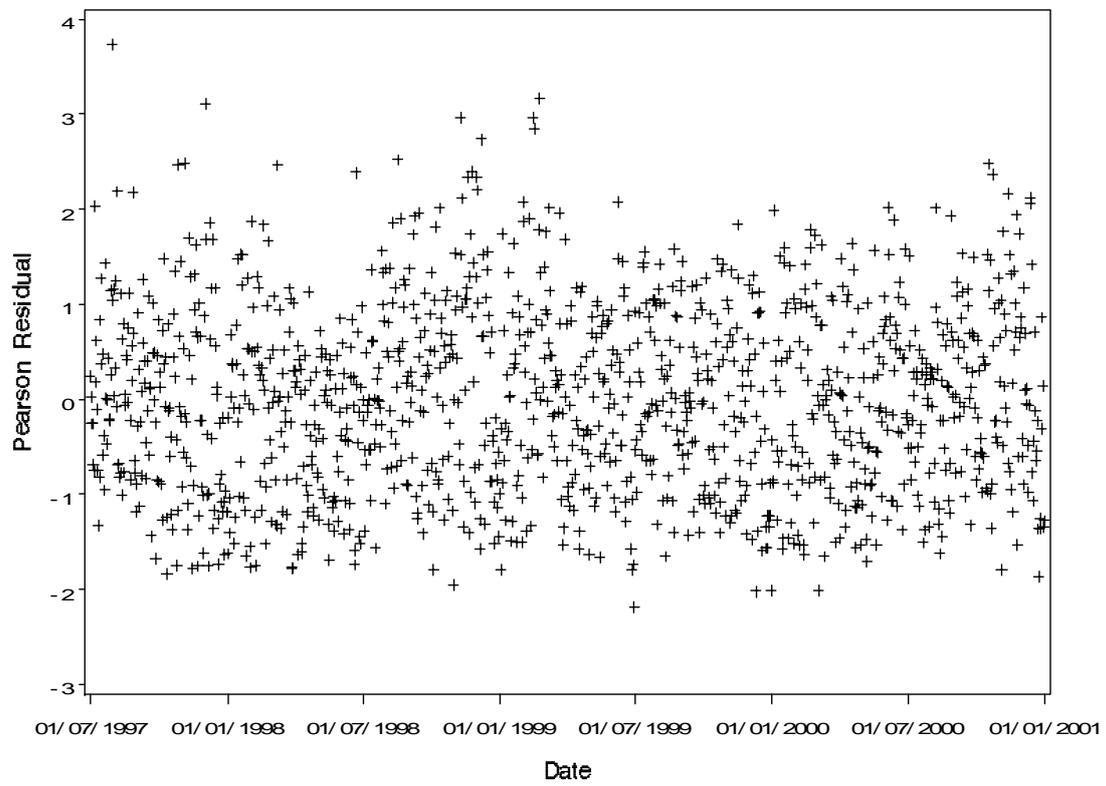


Table I. Cases of respiratory hospital admissions during bushfire and non bushfire periods

PM₁₀ (µg/m³)	Bushfire			Non bushfire			Total		
	Number of days	Total cases	Average daily cases	Number of days	Total cases	Average daily cases	Number of days	Total cases	Average daily cases
<15	130	4252	31±14	389	12358	31±13	519	16610	31±13
15 -20	193	6385	34±12	329	10888	33 ±13	522	17273	33 ±13
>20	129	4671	38±13	110	3714	34±11	239	8385	35±12
Total	452	15308	34±13	828	26960	32±13	1280	42268	33 ±13

Table II. Adjusted relative risk (95% CI) for respiratory hospital admissions, by levels of PM₁₀ (µg/m³) during bushfire and non-bushfire periods, 1997-2000

	PM ₁₀ Value (µg/m ³)	Current day	Lag 1 day	Lag 3 day	Lag 5 day
Over all	<15	1.00	1.00	1.00	1.00
	15-20	1.11 (1.05-1.15)	1.10 (1.05-1.15)	1.11 (1.06-1.16)	1.08 (1.03-1.13)
	> 20	1.16 (1.10-1.23)	1.14 (1.08-1.20)	1.09 (1.03-1.15)	1.13 (1.07-1.19)
	P value for trend	<0.01	<0.01	<0.01	<0.01
Bushfire	<15	1.00	1.00	1.00	1.00
	15-20	1.09 (1.01-1.18)	1.09 (1.00-1.18)	1.11 (1.03-1.21)	1.07 (0.99-1.16)
	> 20	1.19 (1.09-1.30)	1.13 (1.04-1.24)	1.09 (0.99-1.19)	1.13 (1.03-1.23)
	P value for trend	<0.01	<0.01	0.07	0.03
Non Bushfire	<15	1.00	1.00	1.00	1.00
	15-20	1.11 (1.05-1.17)	1.09 (1.00-1.18)	1.10 (1.05-1.16)	1.08 (1.03-1.14)
	> 20	1.13 (1.06-1.23)	1.13 (1.04-1.24)	1.09 (1.00-1.17)	1.12 (1.04-1.21)
	P value for trend	<0.01	<0.01	<0.01	<0.01

* Adjusted for the confounding effects of average temperature, day of the week, (sinusoidal term), long term trends (years) and influenza.

