Acute effects of bushfires on peak expiratory flow rates in children with wheeze: a time series analysis

Abstract

Objectives: To determine the effects of the January 1994 Sydney bushfire on evening peak expiratory flow rates (PEFR) in children with wheeze.

Methods: Children with a history of wheeze were enrolled in the longitudinal study and completed a daily asthma diary. We obtained daily air pollution, meteorological, pollen and alternaria data. We then used generalised estimating equation techniques to determine associations between the bushfire period and particulate matter less than 10 microns (PM_{10}) and PEFR.

Results: The maximum daily PM_{10} level peaked at 210 ug/m^3, which was nearly seven times the usual PM_{10} level for the rest of January and February 1994. There was no significant association between mean PM_{10} and PEFR (beta-coefficient = -0.009, p=0.86). Children without bronchial hyper-reactivity had a significant negative association between PEFR and PM_{10} (beta-coefficient = -0.1029, p=0.03). The bushfire period was not significant in any of the models.

Conclusions: We did not find an association between the bushfire period or PM_{10} and evening PEFR, although in a subgroup of children without bronchial hyper-reactivity, a significant negative association was present between PM_{10} and evening PEFR.

Implications: We conclude that the high levels of particulate pollution caused by the Sydney bushfires did not lead to any clinically significant reductions in PEFR in symptomatic children. Our results have implications for community risk communication during future bushfires.

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Although there are some reported studies on the health effects of bushfires particles,[1-6] there are no reported studies that have investigated the effects of particulate pollution from bushfires on lung function in children.

In January 1994, in Sydney, Australia, there were major bushfires on the outskirts of the city. These bushfires encircled the city and were the worst seen in the 20th Century. A haze of smoke hung over the entire city for about one week. A few days before the bushfires we had commenced recruiting children for a longitudinal study to examine the relationship between daily peak expiratory flow rates (PEFR) and air pollution. This environmental disaster provided an opportunity to study the effects of bushfire smoke in children with a history of wheezing. In particular, we aimed to determine the effects, if any, of the bushfire period and particulate matter less than 10 microns (PM_{10}) on PEFR.

Methods

We obtained ethics approval from all relevant institutional ethics committees prior to commencement of the study. In January 1994, following a cross-sectional study of children in six primary schools in western Sydney, children with a reported history of wheezing in the previous 12 months were enrolled in a longitudinal study. Each child in the study was visited and they and their parents were instructed in the use of a mini-Wright peak flow meter (Clement Clarke International Ltd, United Kingdom) and a daily asthma diary. At subsequent home visits we observed, and corrected if necessary, the child's PEFR measurement technique.

Air pollution, meteorological and pollen data

We obtained daily air pollution and meteorological data (temperature and humidity) for 1994 from six ambient air quality monitoring stations in western and south-western Sydney. The monitoring stations continuously measured ambient ozone (Monitor Lab Ozone Analyzer Model 8810), PM_{10} (TEOM[7]Series 1400A, Rupprecht & Pataschnick Co, Inc., USA) and nitrogen dioxide (Monitor Lab Nitrogen Oxides Analyzer Model 8840). Air pollution and meteorological data were provided as daily maximum, daily maximum for daytime period (between 0600 hours to 2100 hours), daily arithmetic average and daily arithmetic

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average for daytime period. Total pollen and alternaria counts were obtained from two sites using Burkard seven-day spore traps.

Statistical methods

We report data from 1 January 1994 to 31 January 1994 which included the bush fire period (7 January to 14 January 1994). Only children recruited by the 12 January 1994 were eligible for this analysis. We used the evening recorded PEFR as the outcome variable. We excluded any PEFR values that were greater than 120% of the child's mean observed PEFR (PEFRs recorded in the presence of a research assistant) and diary records for when children were out of metropolitan Sydney. We commenced recruiting children for the longitudinal study on 6 January 1994. By 12 January 1994 we had recruited 32 children whose data form the basis for this analysis.

We used the mean daily values for air pollution and meteorological variables in our analyses except for ozone where the maximum daily values were used. These values were only calculated if at least 80% of hourly values were present. Each child's air pollution and meteorological exposures were derived from the monitoring station closest to that child's school. Data from two monitoring stations were subsequently not utilised because of incomplete data collection.

We used generalised estimating equation (GEE) techniques[7] to determine associations between PM_{10} levels and PEFR. PM_{10} levels were also lagged for up to four days and averaged over two and five days. All GEE models included a first order autoregression correlation structure, indicator variables for the bushfire period period (7 January to 14 January 1994) and for asthma medications, linear term for time trend, mean temperature, mean humidity, number of hours spent outdoors, and the log transformed total pollen and alternaria counts. All analyses were performed using SAS statistical software.[8,9]

Results

The mean age of the 32 children was 9.2 years (standard deviation=0.8), 60% were boys (n=19), 38% (n=12) had four or more attacks of wheezing in the past 12 months, 38% (n=12) had bronchial hyper-reactivity on histamine challenge and 78% (n=25) had a doctor diagnosis of asthma.

The maximum daily PM_{10} level increased on 7 January, peaked on 13 January and was down to baseline levels by 15 January 1994 (Figure 1). The peak level (210ug/m^3) was nearly seven times the usual PM_{10} level for the rest of January and February. PEFRs were recorded by most children for each day of the study period (range: 23-32 PEFR records/day). There are no obvious trends in PEFRs (Figure 1). Figure 2 presents time series plots for mean daily PM_{10}, maximum daily ozone, mean daily nitrogen dioxide and mean daily temperature. There were moderate positive correlations between mean PM_{10} and maximum ozone (r=0.67), mean nitrogen dioxide (r=0.51) and mean temperature (r=0.39). There were negative correlations between mean PM_{10} and mean humidity (r=-0.15) and alternaria count (r=-0.56).

The indicator variable for the bushfire period did not make a significant contribution to any of the GEE models. After adjusting for the bushfire period and other potential confounders, there was no significant association between mean PM_{10} and PEFR (beta-coefficient = -0.009) (see Table 1). Lagging the PM_{10} values by up to four days or averaging the PM_{10} values over two and five days did not appreciably change the association. A one day lag produced the largest negative, but non-significant, association (beta-coefficient = -0.09). Using the maximum daily PM_{10} levels instead of the mean daily PM_{10} levels as the dependent variable gave similar results (beta-coefficient = -0.0158, p=0.57).

Results of the regression modelling for subgroups of children with and without bronchial hyper-reactivity, and for children with a doctor diagnosis of asthma are also presented in Table 1. Children without bronchial hyper-reactivity had a significant

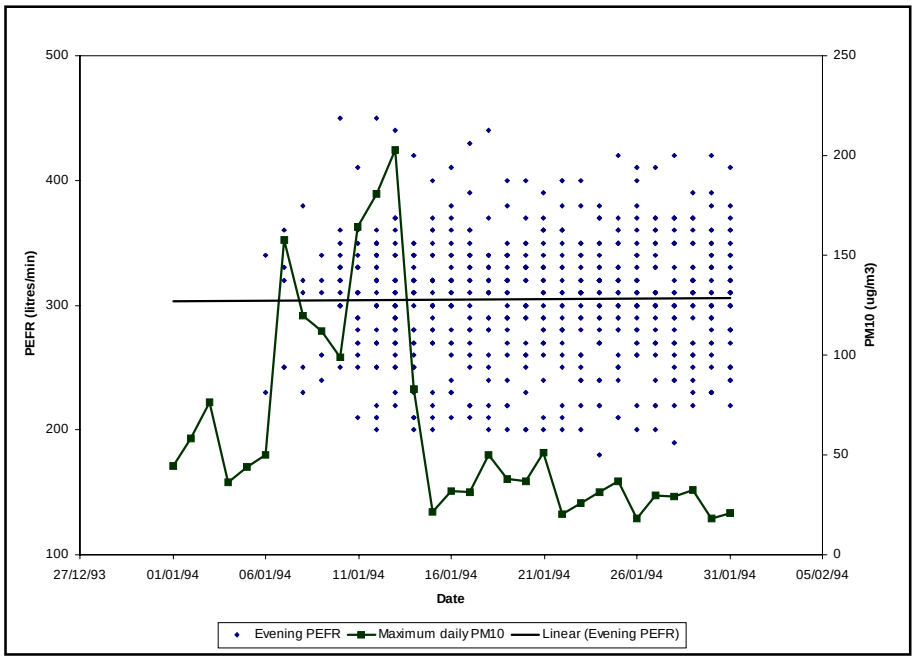

Figure 1: Daily evening peak expiratory flow rates and maximum daily PM_{10} levels, January 1994 (each PEFR point may represent one or more children).

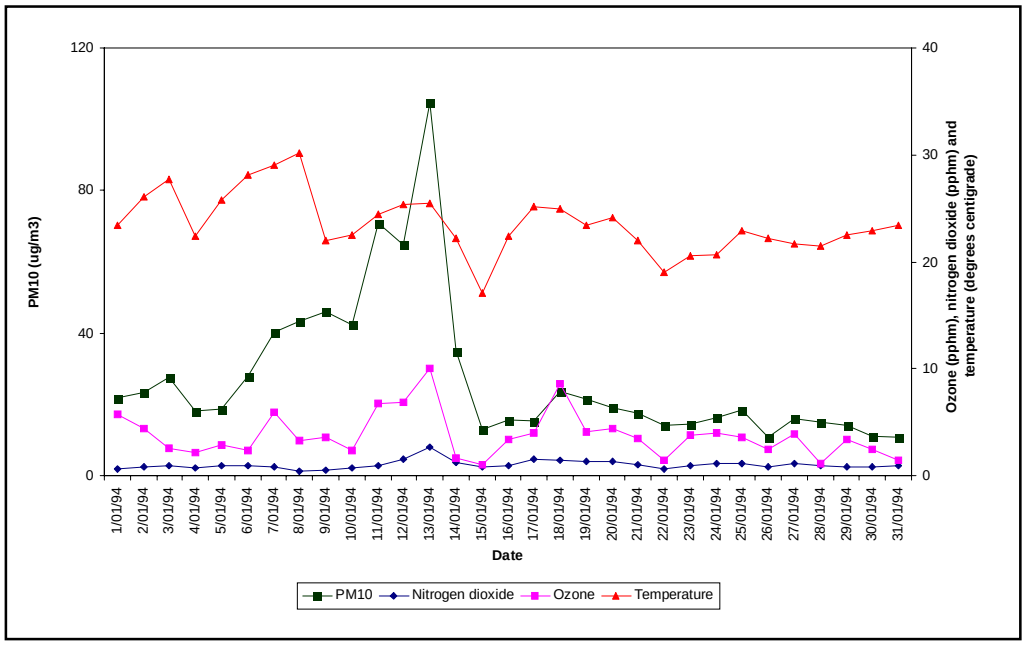

Figure 2: Time series plot of mean daily PM_{10}, mean daily nitrogen dioxide, mean daily temperature and maximum daily ozone, January 1994.

negative association between PEFR and PM_{10} (beta-coefficient = -0.1029, p=0.03).

Discussion

The Sydney bushfires of 1994 afforded us an opportunity to study the acute effects of particulate pollution due to bushfires on lung function. We did not find an association between the bushfire period and evening PEFR nor between PM_{10} levels and evening PEFR. In a subgroup of children without bronchial hyper-reactivity, but not in children with bronchial hyper-reactivity or doctor diagnosed asthma, there was a significant negative association between between PM_{10} levels and evening PEFR.

Table 1: Beta-coefficients for the association between PM_{10} and evening PEFR from GEE models[a].

	Beta-coefficient	Standard error	*p*-value
All children (n = 32)			
Lag 0[b] PM_{10}	-0.0094	0.0549	0.86
Lag 1 PM_{10}	-0.0913	0.0845	0.28
Lag 2 PM_{10}	0.0196	0.0642	0.76
Lag 3 PM_{10}	0.0160	0.0441	0.72
Lag 4 PM_{10}	0.0308	0.0437	0.48
2-day average PM_{10}	-0.0049	0.1506	0.92
5-day average PM_{10}	0.1200	0.1253	0.34
Children with bronchial hyper-reactivity (n = 12)			
Lag 0 PM_{10}	0.1562	0.1120	0.15
Children without bronchial hyper-reactivity (n = 20)			
Lag 0 PM_{10}	-0.1029	0.0470	0.03
Children with a doctor diagnosis of asthma (n = 25)			
Lag 0 PM_{10}	0.0178	0.0612	0.77

Notes:
(a) Time trend, mean daily temperature, mean daily humidity, daily log total pollen, daily log alternaria, asthma medication use and indicator variable for bushfire period included in all models.
(b) Lag 0 = same day value.

We adjusted for potential confounders that could have had an impact on the results. Including a variable for time trend and number of hours spent outdoors in the model accounted for lung growth over time and for differences in the degree of exposure to bushfire smoke respectively. Indicator variables for salbutamol and inhaled corticosteroid use ensured that medication use did not lead to confounding. We also adjusted for temperature and humidity, as well as for pollen and alternaria.

Our findings are consistent with two other studies that did not demonstrate increased emergency department attendances for respiratory diseases attributable to bushfires.[1,6] We also did not find differences in the proportion of child-days with wheeze and dry cough between the bushfire period and non-bushfire period (data not shown). However, repeat cross-sectional studies in bushfire firefighters have demonstrated small decreases in lung function as measured by spirometry.[4,10] In these studies, however, there was no direct measurement of particulate pollution. Further, others have found increased emergency department attendances for respiratory diseases attributable to bushfires.[3,5]

Contrary to our findings, small but significant associations between urban particulate pollution, unrelated to bushfires, and lung function in children have been reported.[11-13] There may be a number of reasons for this. The children in our study were not a homogenous group. Subgroup analysis demonstrated inconsistencies in the associations between PM_{10} levels and PEFR. The association between PM_{10} and PEFR in children with and without bronchial hyper-reactivity is interesting, but in view of the small numbers of children in the study, these associations need to be confirmed in larger studies. Many children may have stayed indoors during the bushfire period to reduce their exposure to the bushfires. However, the fact that we included covariates for time spent outdoors and the bushfire period, and that indoor particulate pollution is about half the outdoor particulate pollution[14,15] suggests that children staying indoors is unlikely to bias our findings. Despite appropriate adjustment for confounding, it is still

possible that our effect estimates may be biased because the outcome may both affect and be affected by time-dependent covariates such as medication use and time spent outdoors.[16] We suspect that any such bias would be towards the null. Lastly, it is possible that PM_{10} derived from bushfires has different bronchial irritant characteristics to PM_{10} derived from urban sources of particulate pollution.

We also found a significant negative association between log of the daily alternaria count and PEFR (beta-coefficient = -2.88, p=0.006). This relationship was robust over a range of models and in subgroup analyses. However, when a separate analysis was performed over a two-month period, the association was no longer significant.

In summary, we investigated the association between particulate pollution due to bushfires and evening PEFR in children with a history of wheezing. We did not find a significant association between the bushfire episode or PM_{10} and evening PEFR, although in a subgroup of children without bronchial hyper-reactivity, the association between PM_{10} levels and evening PEFR was significant. We can conclude, from a public health point of view, that particulate pollution from bushfire does not lead to any clinically significant reductions in PEFR in symptomatic children. Our results have implications for community risk communication during future bushfires.

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