International Journal of Environmental Health Research
December 2006; 16(6): 391 – 404

Taylor & Francis
Taylor & Francis Group

Vegetation fires, particulate air pollution and asthma: A panel study in the Australian monsoon tropics

FAY H. JOHNSTON[1], ROSALIND J. WEBBY[2], LOUIS S. PILOTTO[3], ROSS S. BAILIE[4], DAVID L. PARRY[5], & STEPHEN J. HALPIN[6]

[1]*Menzies School of Health Research and School for Environmental Research, Institute of Advanced Studies, Charles Darwin University, Northern Territory,* [2]*Northern Territory Department of Health and Community Services and National Centre for Epidemiology and Population Health, Australian National University,* [3]*Faculty of Medicine, University of New South Wales,* [4]*Menzies School of Health Research, Institute of Advanced Studies,* [5]*Faculty of Education Health and Science, Charles Darwin University,* [6]*Menzies School of Health Research, Institute of Advanced Studies, Charles Darwin University, and Flinders University of South Australia, Australia*

Abstract
We examined the relationship between particulate matter (PM) <10 and <2.5 microns in diameter (PM_{10} and $PM_{2.5}$) generated by vegetation fires and daily health outcomes in 251 adults and children with asthma over a 7-month period. Data were analysed using generalized estimating equations adjusted for potential environmental confounders, autocorrelation, weekends and holidays. PM_{10} ranged from 2.6 – 43.3 $\mu g\ m^{-3}$and was significantly associated with onset of asthma symptoms, commencing oral steroid medication, the mean daily symptom count and the mean daily dose of reliever medication. Similar results were found for $PM_{2.5}$. No associations were found with the more severe outcomes of asthma attacks, increased health care attendances or missed school/work days. These results help fill a gap in the evidence about the population health impacts of lower levels of pollution characteristic of deliberate landscape burning to control fuel loads versus the better documented risks of more intense and severely polluting wildfires.

Keywords: *Asthma, air pollution, particulate matter, vegetation fires, prescribed burning*

Introduction

The increasing frequency of severe vegetation fire events throughout the world is just one example of global environmental change that is affecting human well-being (Schwela 2001). For example, fire-prone regions of the world such as Australia have documented major and increasing economic and human costs from loss of infrastructure, fire-fighting, deaths and the precipitation of physical and mental illnesses, often with long-lasting

Correspondence: Dr Fay Johnston, Menzies School of Health Research and the School for Environmental Research, Charles Darwin University. PO Box 3185, West Hobart, Tas 7000, Australia. Tel: +614 07612247. E-mail: fay.johnston@cdu.edu.au

ISSN 0960-3123 print/ISSN 1369-1619 online © 2006 Taylor & Francis
DOI: 10.1080/09603120601093642

morbidity (Clayer et al. 1985; McFarlane et al. 1997). In addition to these direct effects, major vegetation fires usually push particulate air pollution concentrations well beyond background levels and can affect population centres far from the fires themselves with measurable impacts on morbidity and mortality (Sastry 2002; Sapkota et al. 2005; Sutherland et al. 2005). This has created an increasing dilemma for park and property managers who recognize that the management of highly fire adapted and fire-prone vegetation requires deliberate burning (Ellis et al. 2004). While nearly every major inquiry into fire disasters in Australia has called for much more prescribed burning to reduce the risk of disasters, there has been falling community tolerance of the smoke pollution these fires generate, even though this is usually far less than that generated by wild fires (Crossett 2004).

There is currently insufficient evidence to determine if there is a safe or acceptable level for deliberate population exposure to vegetation smoke or to assess the population health trade-offs between more frequent exposure to low levels of smoke, compared with the less frequent occurrence of severely polluting and dangerous wild fires. Large multi-centre studies from around the world have clearly established a relationship between airborne particulate matter (PM) and several indices of morbidity and mortality with no apparent lower threshold (Dockery et al. 1993; Pope 2000; Samet et al. 2000; Atkinson et al. 2001). However most studies have been conducted in urban settings in which particulate matter (PM) largely arises from industrial and motor vehicle emissions rather than biomass combustion. The tropical Australian city of Darwin provides a useful setting to examine the health impacts of low levels of vegetation fire smoke. Deliberate landscape burning is a well established management practice and approximately half of the surrounding savanna is burnt during the six-month dry season each year (Russell-Smith et al. 2003). Other sources of air pollution are minimal. During the dry season it has been calculated that 95% of atmospheric PM is derived from vegetation fire smoke while industrial and motor vehicle emissions together contribute just 5% (Gras et al. 2001).

Our study aimed to gather evidence concerning the safety of exposure to vegetation fire smoke for people with asthma. We examined the relationship between PM and asthma symptoms and medication use in a panel of 251 adults and children over a seven-month period in 2004. The study was approved by the human research ethics committees of Charles Darwin University and the joint committee of the Menzies School of Health Research and the Northern Territory Department of Health and Community Services.

Methods

Participants and study setting

Those eligible for inclusion were adults and children with all of the following: asthma diagnosed by a doctor, presence of asthma symptoms or use of medication for asthma in the last year, resident in the Darwin urban region and access to a telephone. Several methods were used for recruitment. All 44 primary and secondary schools, all 16 pharmacies and 16 of the 29 general practices located in the study region agreed to distribute leaflets inviting their clients with asthma to participate in the study.

In addition we contracted a market research company to undertake telephone recruitment using their pre-existing database of residents in the study region supplemented by random dialling from the telephone directory. Participants were informed that many environmental factors, not specifically vegetation fire smoke, were being examined for their effect on asthma.

Exposure measures

We measured the daily mean mass of particulates per cubic meter of air with diameters of <10 microns (PM_{10}) and <2.5 microns ($PM_{2.5}$). Our primary monitoring site was located close to the main residential areas of the study region. We used a Rupprecht and Patashnick Partisol plus Model 2025 sequential air sampler which provided 24-hour gravimetric measures of PM_{10} and $PM_{2.5}$. These data were validated by inter-laboratory comparison gravimetric analyses conducted with the Marine and Atmospheric Research division of Australia's Commonwealth Scientific and Industrial Research Organization (CSIRO). A secondary monitoring site was established in another major residential area approximately 22 km from the primary site to confirm the regional nature of air pollution in Darwin and provide a back up for equipment failure at the primary site. Here we used a Rupprecht and Patashnick Tapered Element Oscillating Microbalance (TEOM) series 1400a which provided continuous PM_{10} loadings with 30-min time resolution. Data from this site were validated by testing in parallel with a co-located Partisol sampler. All equipment was calibrated and maintained according to the manufacturer's specifications.

The daily concentration of pollen and spores in air was measured using a Burkard® volumetric spore trap co-located with the air quality monitoring equipment at the primary monitoring site. The spore trap collects airborne particles as they settle onto an adhesive tape on a slowly rotating drum. The seven-day tapes were cut into daily lengths, stained and mounted on glass microscope slides. Each slide was viewed by Zeiss light microscope at a magnification of $\times 400$ and pollen counted along four full lengthwise transects. Due to the much greater abundance of spores the spore counts were completed on two separate full lengthwise transects. Counts were then converted to correspond to grains per cubic meter of air as a daily mean value.

Records of the maximum and minimum air temperature, relative humidity, dew point and rainfall measured at Darwin airport were provided by the Bureau of Meteorology.

Weekly consultation rates for influenza-like illness from 14 sentinel general practitioners (GPs) located in Darwin and Palmerston were provided by the NT Department of Health and Community Services. The sentinel GPs use a standard case definition for influenza and record their total weekly numbers of patients. The rate is reported as weekly number of influenza cases per 1000 consultations. This information was used as a marker of respiratory viral activity in the community.

Outcome measures

Demographic, clinical, social and environmental data were collected using a questionnaire completed at the time of enrolment. Clinical questions were adapted from the protocol of the International Study on Asthma and Allergy in Children (Asher et al. 1995; Jenkins et al. 1996). The questionnaire also included a validated asthma severity scale which identifies participants at high risk of hospital attendance over a 12-month period (Wakefield et al. 1997). Spirometry was performed on adults and children greater than 8 years of age following the guidelines of the National Asthma Council of Australia (2002) and Pierce and Johns (1995). Reversible airflow obstruction was defined as an increase of 15% or more in forced expiratory volume in 1 second (FEV_1) following inhaled bronchodilator. Salivary samples were collected, centrifuged, frozen and forwarded to the toxicology department of the Adelaide Women and Children's Hospital for cotinine estimation by micro-plate enzyme immunoassay to test for tobacco smoke exposure.

Participants used a daily diary to record their asthma symptoms, medication use, missed school or work days and health care visits for asthma. These data were collected and entered in an electronic database via a fortnightly telephone call. We recorded presence or absence of asthma attacks and individual symptoms of cough, breathlessness, wheezing and chest tightness for both day and night. Asthma attacks were defined as any asthma episode involving breathlessness and/or wheezing and/or chest tightness and/or coughing that interrupts ongoing activities or requires some procedures, such as resting or using a nebuliser to resume normal and comfortable breathing. Each medication, strength, dose and number of applications taken each day was recorded individually and classified as follows: (i) short acting inhaled bronchodilator (reliever), (ii) inhaled steroids, cromoglycates and oral montelukast (preventers), (iii) long acting bronchodilators (symptom controllers) and (iv) oral steroids. The main outcome measures examined were the daily proportion of the group that: (i) had any symptoms attributable to asthma, (ii) became symptomatic after being symptom free for at least 7 days, (iii) used reliever medication, (iv) commenced a reliever after at least 7 days without any reliever, (v) commenced a course of oral steroids, (vi) experienced exercise induced asthma, (vii) experienced an asthma attack (viii) missed school or work due to asthma and (ix) saw a health profession about their asthma. We also examined the mean number of symptoms present and the mean number of times a reliever medication was used each day.

Statistical analysis

Data were analysed using Stata8 statistical software package (StataCorp 2003). We used generalized estimating equations to generate population averaged models adjusted for minimum daily air temperature, relative humidity, pollen and spore counts, the weekly rate of consultations to general practitioners for influenza-like illness, temporal autocorrelation of outcomes, weekends and holiday periods. We examined the relationship between same day exposures and lags of up to 5 days to both PM_{10} and $PM_{2.5}$. Logistic regression models were used to calculate odds ratios (OR) for dichotomous outcomes and negative binomial regression models were used to calculate incidence rate ratios (IRR) for count outcomes. Subgroup analyses were conducted on adults and children separately and on participants who reported either moderate or severe asthma, using a preventer, or meeting criteria for being high risk of hospital attendance. Subgroup analyses were further adjusted for age (adult vs. child), self-reported severity, use of a preventer, smoking status (smoker vs. non-smoker) indigenous status, household crowding (ratio of people to bedrooms) and education level (highest qualification of any adult in the household).

Missing PM data

For single missing values of just one day, data were replaced by the average of the readings before and after the missing value. For gaps of two or more days PM_{10} data from the secondary site were used to estimate values for the primary site as results from the two sites were correlated. The nine missing values from the primary site between 7 April and 7 June were calculated by multiplying the values recorded at the secondary site by 2.1, the ratio of the mean values at the two sites for that period. For the remainder of the study the mean PM_{10} readings were very similar (ratio = 1.03) and missing data at the primary site were directly replaced with the readings from the secondary site. Missing $PM_{2.5}$ data were calculated by multiplying the PM_{10} value for that day by 0.56 as this ratio remained consistent throughout the study period.

We examined the sensitivity of our results to the imputed values by conducting additional analyses using imputed data values set 25% higher and 25% lower than in our original estimation. We also examined outcomes against PM_{10} data from the secondary site only.

Results

Participants

A total of 251 people started the study of whom 235 (94%) contributed data for the full duration of the study. An average of 210 people contributed data each day. Of the 16 who dropped out 14 relocated away from the study site and two relocated and become uncontactable. Their data were included for the time they participated. An average of 5% (range 2–14%) were absent from the study region each day. The proportion of absent participants was higher during school holidays (mean = 8%) and peaked at 14% over the Easter break. Individual data were excluded from the analysis for the days in which participants were absent. The mean number of days contributed per participant was 186 out of a maximum possible of 214 (87%).

Approximately half the group were less than 18 years of age (Figure 1) and 57% identified vegetation fire smoke as a trigger of their asthma symptoms. 74% classified their asthma as being mild and 26% met criteria for being at high risk of a hospital attendance within 12 months. At enrolment 63% said they used regular inhaled steroids, however the average daily proportion of participants that actually used an inhaled preventer during the study period was 39% (range 36–43%). The demographic and clinical characteristics reported by participants at the time of enrolment are summarized in Table I.

Our group had similar characteristics to people with asthma in the general Australian population reported by the National Health Survey in 2001 (Australian Bureau of Statistics, 2004). We had a higher proportion who initially reported regular use of inhaled steroids

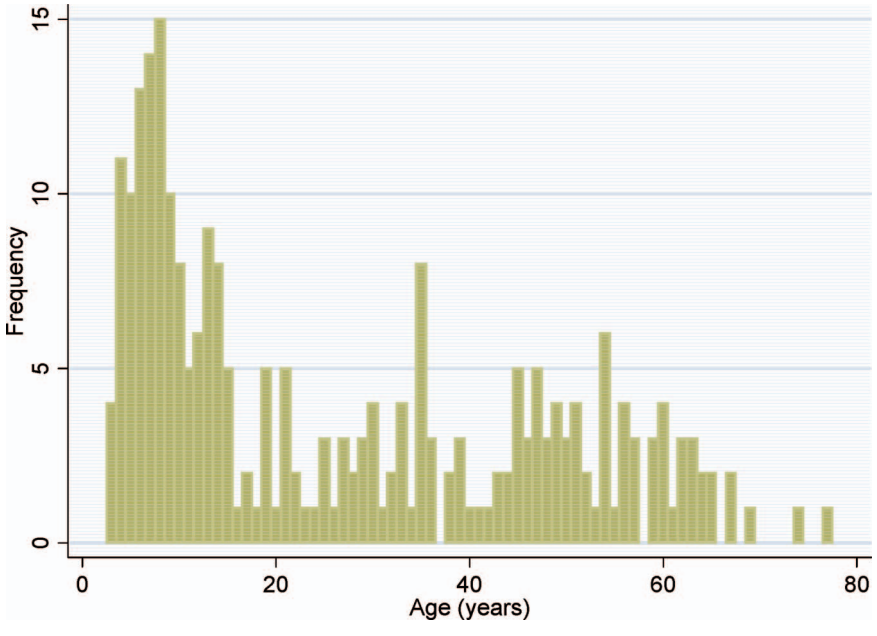

Figure 1. Age distribution of participants.

Table I. Characteristics of study participants at baseline.

Socio/demographic indicators	Adults $n=130$ (%)	Children $n=121$ (%)	Total $n=251$ (%)
Male	28	63	45
Female	72	37	55
Indigenous	7	19	13
Smoker	22	0	11
Exposure to environmental tobacco smoke at home	26	35	30
Ratio of people: bedrooms <1	46	7	28
Ratio of people: bedrooms $=1$	25	17	21
Ratio of people: bedrooms >1	29	76	27
Adult in house with a tertiary qualification	72	70	70
Clinical features			
Using regular inhaled steroids	68	57	63
Mild asthma in the last month	76	70	73
Moderate asthma in the last month	19	27	23
Severe asthma in the last month	5	3	4
Used oral steroids in the last month	12	17	14
Woken from sleep with asthma in the last month	34	40	37
Admission to hospital for asthma in last 12 months	5	16	10
Saw more than 1 GP for asthma in last 12 months	22	31	26
High risk of hospital attendance*	19	33	26
History of eczema or hay fever	79	51	65
Positive cotinine in non-smoker	5	7	6
Reversible airflow obstruction** (of 109 adults and 42 children able to perform spirometry)	11	16	13
Reported triggers of asthma			
Cold or respiratory viral infection	58	59	59
Change in weather	68	63	65
Bushfire smoke	62	52	57
Exercise	57	48	53
Dust	65	38	52
Pollen	56	31	43
Cigarette smoke	58	24	41
Animal hair/fur	41	20	31
Foods	27	11	19
Swimming pools	3	12	12
Drugs	10	2	6

*According to the severity scale developed by Wakefield et al (Wakefield et al. 1997); **FEV1 increase of 15% following inhaled bronchodilator.

(63% vs. 39%), a slightly lower proportion of adult smokers (22% vs. 26%), a greater predominance of males among children (63% vs. 56%) and females among adults (72% vs. 58%). We also had a higher proportion of indigenous participants compared with the 2001 census count for Darwin (13% vs. 9%) and a high proportion of participants living in a house in which any adult had a tertiary or trade qualification (72%). In the 2001 census, 37% of adults in Darwin and 35% adult Australians reported holding a tertiary qualification (Australian Bureau of Statistics 2002, 2003).

PM data quality and availability

PM_{10} and $PM_{2.5}$ data measured at the primary site were found to be valid and reliable on independent quality control checks. Data at this site were available for 166 of the 215 days of

the study period (77%) with gaps ranging from 1 – 12 days in length. Data from the TEOM also met all quality control checks and were more complete (87%).

Environmental conditions during the study

Metrological conditions were unusual during the study period. Rainfall, usually a rare event in the tropical dry season, persisted well into June and delayed the curing of the savanna grasses. This resulted in lower than usual fire hazard conditions and lower than expected air pollution during the study. Environmental data are summarized in Table II.

Particulate levels fluctuated with several peaks and troughs throughout the study period. The PM_{10} often climbed or fell by 15 – 20 $\mu g/m^3$ over periods of just a few days. Australia's national air quality target for PM_{10} of 50 $\mu g/m^3$ was exceeded once at the secondary monitoring site only. $PM_{2.5}$ exceeded the national advisory reporting standard of 25 $\mu g/m^3$ on 5 days. $PM_{2.5}$ was highly correlated with PM_{10} at the primary site ($r = 0.9$) with a mean value of 56% of PM_{10}.

For the first 3 months of operation PM_{10} data from the TEOM at the secondary site was consistently half that recorded by the Partisol sampler at the primary site. There was no obvious explanation for this difference. Thereafter readings from the two sites remained similar apart from two episodes in which PM_{10} peaked at a higher level at the secondary site. Both these episodes could be attributed to local fires closer to the secondary site. Measurements from the two sites were correlated ($r = 0.7$).

Pollen and fungal counts were low compared with other Australian capital cities (Rutherford et al. 2000). The weekly proportion of GP consultations for influenza-like illnesses fluctuated with two small peaks during March and June.

Health outcomes

The frequency of health outcomes among participants are shown in Table III. Significant first order autocorrelation was found for all outcomes except for the less frequent events of commencing reliever, commencing oral steroids, missing school or work due to asthma, and seeing a health professional about asthma.

Table II. Description of measured environmental variables, 7 April to 7 November, Darwin 2004.

Exposure	Mean	Standard Deviation	Range
PM_{10} at primary monitoring site ($\mu g/m^3$)	20.0	6.4	2.6 – 43.3
$PM_{2.5}$ at primary monitoring site ($\mu g/m^3$)	11.1	5.4	2.2 – 36.5
PM_{10} at secondary monitoring site ($\mu g/m^3$)	18.0	8.4	2.9 – 53.7
Total pollen count (grains/m^3)	16.4	9.4	0.5 – 60.5
Grass pollen count (grains/m^3)	3.5	5.0	0 – 22.2
Fungal spore count (spores/m^3)	1843	1043	0 – 6049
Daily maximum temperature (°C)	31.8	2.1	25.7 – 36.1
Daily minimum temperature (°C)	21.3	3.2	13.3 – 27.5
Relative humidity at 9:00 a.m. (%)	64.8	14.8	9 – 96
Weekly GP consultation rate for influenza-like illness (rate/1000 consultations)	12.2	6.8	3.3 – 27.3

Table III. Summary of outcomes measures.

Outcome	Mean daily percentage	Standard deviation	Range
Wheeze	9.7	2.5	3.4–18.4
Breathlessness	10.6	2.4	6.0–17.4
Cough	20.0	3.9	11.1–31.6
Chest tightness	9.8	2.4	4.7–16.1
Any symptom(s) attributable to asthma	25.7	4.5	15.8–37.7
Become symptomatic (after at least one symptom free week)	1.8	1.2	0–5.7
Exercise induced asthma (of those who exercised)	6.8	2.0	2.1–13.0
Asthma attack	3.0	1.2	0.9–8.4
Used reliever	23.2	3.0	16.1–31.2
Commenced reliever (after at least one week without using reliever)	1.45	1.1	0–5.45
Used any preventer (including inhaled steroids, cromoglycates and montelukast)	44.7	1.9	39.8–48.9
Used inhaled steroids	39.5	1.6	36–42.8
Used combination preventer/symptom controller	25.7	1.4	22.7–29.5
Used leukotriene antagonists	4.8	0.6	3.2–6.0
Used inhaled cromoglycates	2.3	0.5	1.4–3.9
Used symptom controller	2.6	0.4	1.8–4.1
Used oral steroids	1.1	0.6	0–3.0
Commenced oral steroids (after at least one week without oral steroids)	0.12	0.2	0–0.9
Saw a health professional about asthma	0.6	0.5	0–2.3
Missed school or work due to asthma	0.8	0.9	0.3–9.3
Counted outcomes	*Mean daily count*		
Daily number of asthma symptoms (maximum of 8)	0.8	1.7	0–8
Daily number of times reliever was used	0.6	1.4	0–22

Relationship between PM and health outcomes

We found small associations between PM_{10} and $PM_{2.5}$ and the proportion of participants starting a course of oral steroids and becoming symptomatic following a symptom free period. Additionally there were associations between $PM_{2.5}$ and starting to use reliever medication and between PM_{10} and the average number of asthma symptoms and the mean number of times reliever medication was used by participants each day. Most associations were of slightly greater magnitude in adults compared with children and in those using a preventer (Table IV). We did not find any associations between pollution levels and exercise induced asthma, asthma attacks, missed work or school due to asthma or health care attendances for asthma. When we examined outcomes for time lags of up to 5 days associations were identified at lags of one day between PM_{10} and onset of symptoms and $PM_{2.5}$ with both onset of symptoms and commencing a reliever (data not shown). Lags of 2–5 days were not associated with any of the health outcomes measured. These outcomes were not substantially altered by using the 25% higher or lower estimations for the imputed PM data. When we examined outcomes against PM_{10} recorded from the secondary monitoring site we also found very similar results. The main differences with the latter analysis were that the association between PM_{10} and commencement of reliever medication achieved statistical significance while the association between PM_{10} and commencement of oral steroids lost statistical significance.

Table IV. Relationships between PM and health outcomes.

	PM_{10} (rise of 10 μg/m^3)			$PM_{2.5}$ (rise of 5 μg/m^3)		
Outcome	IRR	95% CI	*p*	OR	95% CI	*p*
Symptoms attributable to asthma						
Overall	1.010	0.98 – 1.04	0.401	1.000	0.98 – 1.01	0.848
*Adults	1.027	0.987 – 1.068	0.183	1.000	0.976 – 1.026	0.944
*Children	0.930	0.966 – 1.057	0.641	1.008	0.980 – 1.037	0.558
*Using preventer	1.022	0.985 – 1.060	0.243	1.013	0.990 – 1.037	0.249
Became symptomatic						
Overall	**1.240**	**1.106 – 1.39**	**0.000**	**1.150**	**1.07 – 1.23**	**0.000**
*Adults	**1.277**	**1.084 – 1.504**	**0.003**	**1.165**	**1.058 – 1.284**	**0.002**
*Children	**1.247**	**1.058 – 1.468**	**0.008**	**1.148**	**1.042 – 1.264**	**0.005**
*Using preventer	**1.317**	**1.124 – 1.543**	**0.001**	**1.181**	**1.076 – 1.296**	**0.000**
Used reliever						
Overall	1.010	0.99 – 1.04	0.264	1.000	0.98 – 1.02	0.788
*Adults	1.026	0.990 – 1.063	0.147	1.007	0.980 – 1.035	0.573
*Children	1.006	0.960 – 1.055	0.779	1.002	0.972 – 1.034	0.861
*Using preventer	**1.035**	**1.004 – 1.06**	**0.024**	**1.020**	**1.000 – 1.042**	**0.050**
Commenced reliever						
Overall	1.132	0.99 – 1.29	0.065	**1.120**	**1.03 – 1.210**	**0.005**
*Adults	1.199	0.994 – 1.446	0.57	**1.141**	**1.021 – 1.275**	**0.019**
*Children	1.093	0.906 – 1.319	0.93	1.112	0.994 – 1.243	0.061
*Using preventer	1.194	0.996 – 1.432	0.55	**1.129**	**1.013 – 1.257**	**0.028**
Commenced oral steroids						
Overall	**1.540**	**1.01 – 2.34**	**0.046**	**1.310**	**1.03 – 1.66**	**0.023**
*Adults	**1.752**	**1.008 – 3.045**	**0.047**	**1.601**	**1.192 – 2.150**	**0.002**
*Children	1.292	0.682 – 2.448	0.431	0.995	0.625 – 1.459	0.832
*Using preventer	1.430	0.888 – 2.304	0.141	**1.350**	**1.040 – 1.752**	**0.024**
Asthma attack						
Overall	1.030	0.95 – 1.12	0.434	0.980	0.94 – 1.04	0.639
*Adults	1.08	0.976 – 1.202	0.129	1.026	0.962 – 1.095	0.424
*Children	0.861	0.710 – 1.044	0.129	**0.832**	**0.731 – 0.946**	**0.005**
*Using preventer	1.051	0.939 – 1.175	0.382	1.002	0.934 – 1.075	0.937
Exercise induced asthma						
Overall	0.980	0.92 – 1.05	0.667	0.990	0.95 – 1.03	0.741
*Adults	0.988	0.902 – 1.081	0.793	0.998	0.943 – 1.056	0.95
*Children	0.972	0.844 – 1.119	0.696	0.982	0.899 – 1.071	0.684
*Using preventer	1.026	0.928 – 1.134	0.608	1.002	0.942 – 1.067	0.929
Saw a health professional for asthma						
Overall	1.030	0.85 – 1.26	0.742	1.030	0.91 – 1.16	0.652
*Adults	1.064	0.794 – 1.424	0.676	1.079	0.899 – 1.296	0.412
*Children	0.998	0.749 – 1.328	0.989	1.003	0.841 – 1.195	0.973
*Using preventer	0.924	0.731 – 1.169	0.513	0.980	0.847 – 1.133	0.803
Missed school or work due to asthma						
Overall	1.102	0.941 – 1.290	0.226	1.025	0.9284 – 1.131	0.628
*Adults	1.135	0.897 – 1.435	0.290	1.077	0.923 – 1.247	0.323
*Children	1.073	0.862 – 1.333	0.527	1.000	0.873 – .1458	0.998
*Using preventer	1.025	0.857 – 1.228	0.783	1.005	0.897 – 1.124	0.936

(*continued*).

Table IV. (*Continued*).

Outcome	PM_{10} (rise of 10 μg/m^3)			$PM_{2.5}$ (rise of 5 μg/m^3)		
	IRR	95% CI	*p*	OR	95% CI	*p*
Mean daily number of asthma symptoms						
Overall	**1.020**	**1.001 – 1.031**	**0.014**	1.003	0.99 – 1.01	0.594
*Adults	**1.027**	**1.005 – 1.049**	**0.017**	0.998	0.984 – 1.012	0.799
*Children	1.016	0.986 – 1.047	0.304	1.004	0.985 – 1.023	0.669
*Using preventer	**1.034**	**1.011 – 1.058**	**0.004**	1.013	0.999 – 1.028	0.067
Mean daily number of applications of reliever						
Overall	**1.020**	**1.00 – 1.030**	**0.032**	1.002	0.993 – 1.010	0.697
*Adults	**1.032**	**1.008 – 1.057**	**0.007**	1.001	0.986 – 1.016	0.876
*Children	1.002	0.969 – 1034	0.909	1.000	0.980 – 1.021	0.933
*Using preventer	**1.022**	**1.001 – 1.043**	**0.039**	1.005	0.994 – 1.017	0.315

All analyses adjusted for minimum air temperature, humidity, rainfall, pollen count, spore count, influenza rates, weekends, holiday periods and temporal autocorrelation. OR, odds ratio; IRR, interval rate ratio; CI, confidence interval; **Bold** = $p < 0.05$. *Subgroup analyses additionally adjusted for age, smoking, education status, indigenous status, household crowding, self reported severity and current use of preventer.

Relationship between other environmental exposures and health outcomes

We found small associations between the following environmental exposures and health outcomes: (i) minimum air temperature and the prevalence of asthma symptoms (an inverse association OR = 0.98, 95% CI = 0.97 – 0.99, $p = 0.03$), (ii) GP consultation rates for influenza and the prevalence of asthma symptoms (OR = 1.06, 95% CI = 1.01 – 1.12, $p = 0.025$) and (iii) the fungal spore count and both onset of asthma symptoms (OR = 1.0001, 95% CI = 1.00002 – 1.0002, $p = 0.011$) and commencement of reliever medication (OR = 1.0002, 95% CI = 1.000014 – 1.0007, $p = 0.020$). We did not find any health outcomes associated with pollen levels, rainfall, relative humidity, dew point or maximum air temperature.

In absolute terms most of the associations we identified were due to response variation in just 5% of the group or 10 – 11 participants.

Discussion

In this study we identified associations between low levels of PM_{10} and $PM_{2.5}$ derived from vegetation fire smoke and asthma symptoms and medication use, but not asthma attacks, healthcare attendances or missed school or work due to asthma.

These findings are consistent with many epidemiological studies that have described linear relationships with no apparent lower threshold between particulate air pollution and a wide range of adverse health outcomes including all cause mortality and exacerbations of respiratory and cardiovascular diseases (Daniels et al. 2000; WHO 2000; Schwartz et al. 2002; Katsouyanni 2003; Ward & Ayres 2004). They are also consistent with the current state of knowledge on the pulmonary toxicology of the components of vegetation smoke particularly carbon-based PM generated by combustion (Schwela 2001; Donaldson 2004).

Our results are largely consistent with the few previously published epidemiological studies specifically examining the health impacts of particulate pollution from vegetation fires. Of the twelve published studies we identified, nine found associations with a range of health

outcomes including hospital or clinic attendances for respiratory conditions (Duclos et al. 1990; Churches & Corbett 1991; Centers for Disease Control and Prevention 1999; Emmanuel 2000; Johnston et al. 2002; Mott et al. 2002) exacerbation of symptoms in a cohort of people with chronic obstructive pulmonary disease (Sutherland et al. 2005) and hospital admissions and all cause-mortality (Sastry 2002; Mott et al. 2005). The remaining three, all of which studied fires near Sydney in 1994, were negative (Cooper et al. 1994; Smith et al 1996; Jalaludin et al 2000). Jalaludin et al. (2000) were recruiting children for a longitudinal study of lung function when the fires occurred. Their analysis was based on data from just 32 participants and might have lacked sufficient statistical power. The other two studies, which examined hospital presentation data for asthma, were subject to the inherent limitations of retrospective studies. Relying on the examination of routinely collected data limits the ability to adjust for important confounders such as weather, pollen levels and community activity of respiratory viral illnesses (Anderson 1999). Moreover, pollution from fires is often subject to considerable media attention which may influence the medication use and health-seeking behaviour of people with diseases such as asthma.

Our study was able to overcome many of these limitations because it was located where a prolonged period of fluctuating smoke haze is a predictable annual phenomenon and other sources of air pollution are minimal (Gras et al. 2001). Data were collected prospectively over a 7-month period during which the day to day variations in pollution were not obvious to the general public and received little media attention. We were also able to control for pollen counts, fungal spores, dew point, humidity, temperature, rainfall, influenza rates, days of the week, public holidays and school holidays all of which could confound the association between PM and health outcomes.

There are several potential explanations why we did not find any associations between particulate levels and the more severe outcomes of asthma attacks, missed school or work, and attending a health service for asthma. The impacts of air pollution might be less severe at relatively lower levels of pollution, we might not have had sufficient power to detect associations with the outcomes that were infrequent in this study, or this could have partly been due to the particular characteristics of our participants. Although their age and gender distributions were broadly similar to people with asthma in the general Australian population (Australian Bureau of Statistics 2004), we may have had a greater proportion of people competent in managing their (or their children's) asthma as evidenced by the higher proportion of households containing a person with a tertiary qualification and the higher proportion of participants using regular preventers. The latter observation might also reflect a group with more severe disease. Subgroup analysis by use of preventers had very similar outcomes to the overall findings with just a slightly greater magnitude in the association of PM with some symptoms and medication use.

An important contribution of our study is that we were able to examine PM derived from vegetation fire smoke at levels considerably lower than those examined in previously published epidemiological studies and well below Australia's air quality standards for PM_{10} and $PM_{2.5}$ (National Environment Protection Council [NEPC] 2003). This helps to fill an important gap in the evidence concerning the public health consequences of lower levels of pollution generated by smaller fires deliberately lit for land management purposes (U.S. Environmental Protection Agency [USEPA] 1998; Lewis & Corbett 2002). The risks of this practice need to be put in context with the risks of failing to manage fuel loads, particularly the economic, health and ecological costs of uncontrolled wild fires (NEPC 2002; Ellis et al. 2004). Pollution generated by major fires is usually considerably greater than that produced by prescribed burns and important public health impacts can be expected if large populations are exposed. For example, although there were no direct deaths from the Sydney fires of 2001

in which the PM_{10} remained above 150 $\mu g/m^3$ for 10 days, Australia's National Environment Protection Council (NEPC) estimated the excess pollution would have been responsible for 16 deaths and at least 30 hospital admissions (NEPC 2002).

Conclusion

We have identified several clinically important associations between symptoms and medication use for asthma and particulate air pollution (PM_{10} and $PM_{2.5}$) at levels below current national air quality targets. Our findings add to the growing literature about the adverse health impacts of population exposure to smoke from vegetation fires. The public health risks of deliberate burning for fuel management should be examined in relation to the better documented, more severe risks of large uncontrolled fires. While deliberate burning may be justifiable in this context, minimization of population exposure to particulate pollution should remain a high priority.

Acknowledgements

Thanks are due to our research assistants Anne Myerscough, Judy Manning and Francoise Foti, and collaborators Michael Foley, Simon Haberle, David Bowman and John Gras. This study was funded by a linkage grant from the Australian Research Council with cash and in kind support from the Northern Territory Government, Bureau of Meteorology and Asthma NT.

References

Anderson HR. 1999. Methodological aspects of the evaluation of the health effects of air pollution episodes. In: Holgate ST, Samet JM, Koren HS, Maynard RL, editors. Air pollution and health. London: Academic Press. pp 476–481.

Asher MI, Keil U, Anderson HR, Beasley R, Crane J, Martinez F, Mitchell EA, Pearce NE, Sibbald B, Stewart AW, et al. 1995. International study of asthma and allergies in childhood (ISAAC): Rationale and methods. Eur Respir J 8:483–491.

Atkinson RW, Anderson HR, Sunyer J, Ayers J, Baccini M, Vonk JM, Boumghar A, Forastiere F, Forsberg AB, Touloumi G, Schwartz J, Katsouyanni K. 2001. Acute effects of particulate pollution on respiratory admissions. Results from the APHEA 2 project. Am J Respir Crit Care Med 164:1860–1866.

Australian Bureau of Statistics. 2002. Basic Community Profile and Snapshot 705 Darwin, Australia, Northern Territory. 2001 Census. Catalog No. 2001.0.

Australian Bureau of Statistics. 2003. Census of Population and Housing: Selected Education and Labour Force Characteristics Australia. 2001 Census Catelog No. 2017.0.

Australian Bureau of Statistics. 2004. Asthma in Australia: A Snapshot. 2001 National Health Survey. Catalog No. 4819.0.55.001.

Centers for Disease Control and Prevention. 1999. Surveillance of morbidity during wildfires – Central Florida, 1998. Morbid Mortal Weekly Rev 48:78–79.

Churches T, Corbett S. 1991. Asthma and air pollution in Sydney. NSW Public Health Bulletin 8:72–73.

Clayer JR, Bookless-Pratz C, Harris RL. 1985. Some health consequences of a natural disaster. Med J Australia 143:182–184.

Cooper C, Mira M, Danforth M, Abraham K, Fasher B, Bolton P. 1994. Acute exacerbations of asthma and bushfires. Lancet 343:1509.

Crossett G. 2004. Advocates for Clean Air Australia. Submission to the Council of Australian Governments Bushfire Inquiry. Available: www.coagbushfireenquiry.gov.au/subs_html/gayle_crossett.html. Accessed October 2005.

Daniels MJ, Dominici F, Samet JM, Zeger SL. 2000. Estimating particulate matter mortality dose-response curves and threshold levels: an analysis of daily time-series for the 20 largest US cities. Am J Epidemiol 152:397–406.

Dockery D, Pope CA, Xu X, Spengler JH, Ware ME, Ferris BG, Speizer FE. 1993. An association between air pollution and mortality in six US cities. N Engl J Med 329:1753–1759.

Donaldson K. 2004. The biological effects of coarse and fine particulate matter. Toxicol Appl Pharmacol 195:35–44.
Duclos P, Sanderson LM, Lipsett M. 1990. The 1987 forest fire disaster in California: Assessment of emergency room visits. Arch Environ Health 45:53–58.
Ellis S, Kanowski P, Whelan R. 2004. National Inquiry on Bushfire Mitigation and Management. Commonwealth of Australia, Canberra.
Emmanuel SC. 2000. Impact to lung health of haze from forest fires: The Singapore experience. Respirology 5:175–182.
Gras J, Parry D, Jong T, Mungksgaad N, Ayers G, Keywood M, Boast K, Powell J, Selleck P, Firestone T. 2001. A pilot study of the air quality in Darwin NT. Department of Infrastucture Planning and Environment. Northern Territory Government, Darwin, pp 1–17.
Jalaludin B, Smith M, O'Toole B, Leeder S. 2000. Acute effects of bushfires on peak expiratory flow rates in children with wheeze: a time series analysis. Australian NZ J Public Health 42:174–177.
Jenkins M, Clarke J, Carlin J. 1996. Validation of questionnaire and bronchial hyper-responsiveness against repiratory physician assessment in the diagnosis of asthma. Int J Epidemiol 25:609–616.
Johnston FH, Kavanagh AM, Bowman DM, Scott RK. 2002. Exposure to bushfire smoke and asthma: An ecological study. Med J Aust 176:535–538.
Katsouyanni K. 2003. Ambient air pollution and health. In: Briggs DJ, Joffe M, Elliott P, editors. Impact of environmental pollution on health. Glasgow, UK: British Medical Bulletin Vol. 68. pp 143–157.
Lewis P, Corbett S. 2002. Bushfires, air pollution and asthma. M J Australia 176:517.
McFarlane AC, Clayer JR, Bookless CL. 1997. Psychiatric morbidity following a natural disaster: An Australian bushfire. Soc Psychiatry Psychiatric Epidemiol 32:261–268.
Mott JA, Meyer P, Mannino D, Redd SC, Smith EM, Gotway-Crawford C, et al. 2002. Wildland forest fire smoke: Health effects and intervention evaluation, Hoopa, California, 1999. Western J Med 176:162–163.
Mott JA, Mannino DM, Alverson CJ, Kiyu A, Hashim J, Lee T, et al. 2005. Cardiorespiratory hospitalizations associated with smoke exposure during the 1997, Southeast Asian forest fires. Int J Hygiene Environ Health 208(1–2):75–85.
National Asthma Council of Australia. 2002. Asthma Management Handbook. National Asthma Council Australia, Melbourne.
National Environment Protection Council (NEPC). 2002. National Environment Protection (Ambient Air Quality) Measure impact statement for PM2.5 variation. Setting a PM2.5 standard in Australia. Environment Protection and Heritage Council, Adelaide.
National Environment Protection Council (NEPC). 2003. National Environment Protection (Ambient Air Quality) Measure as amended. Environment Protection and Heritage Council, Adelaide.
Pierce R, Johns D. 1995. Spirometry, the measurement and interpretation of ventilatory function in clinical practice, National Asthma Council of Australia. Melbourne.
Pope CA. 2000. Epidemiology of fine particulate air pollution and human health: Biologic mechanisms and who's at risk? Environ Health Perspect 108:713–723.
Russell-Smith J, Yates C, Edwards A, Allan GE, Cook GD, Cooke P, Craig R, Heath B, Smith R. 2003. Contemporary fire regimes of northern Australia, 1997–2001: Change since Aboriginal occupancy, challenges for sustainable management. Int J Wildland Fire 12(4):283–297.
Rutherford S, Simpson R, Williams G, Mitchell C, McCall B. 2000. Relationships between environmental factors and lung function of asthmatic subjects in south east Queensland, Australia. J Occup Environ Med 42:882–891.
Samet JM, Zeger S, Dominici F, Dockery D, Schwartz J. 2000. Fine particulate air pollution and mortality in 20 US cities, 1987–1994. Cambridge, Mass: Health Effects Institute.
Sapkota A, Symons JM, Kleissl J, Wang L, Parlange MB, Ondov J, Breysse PN, Diette GB, Eggleston PA, Buckley TJ. 2005. Impact of the 2002 Canadian forest fires on particulate matter air quality in Baltimore city. Environ Sci Technol 39:24–32.
Sastry N. 2002. Forest fires, air pollution, and mortality in Southeast Asia. Demography 39:1–23; 158:133–138.
Schwartz J, Laden F, Zanobetti A. 2002. The concentration-response relation between PM(2.5) and daily deaths. Environ Health Perspect 110:1025–1029.
Schwela D. 2001. Fire disasters: The WHO-UNEP-WMO health guidelines for vegetation fire events. Ann Burns Fire Disasters XIV.
Smith MA, Jalaludin B, Byles JE, Lim L, Leeder SR. 1996. Asthma presentations to emergency departments in western Sydney during the January 1994 bushfires. Int J Epidemiol 25:1227–1236.
StataCorp. 2003. Stata Statistical Software: Release 8. StataCorp LP, College Station TX.
Sutherland ER, Make BJ, Vedal S, Zhang L, Dutton SJ, Murphy JR, Silkoff PE. 2005. Wildfire smoke and respiratory symptoms in patients with chronic obstructive pulmonary disease. J Allergy Clin Immunol 115:420–422.

USEPA. 1998. Interim air quality policy on wildland and prescribed fires. U.S. Environmental Protection Agency. Available: http://www.epa.gov/ttn/oarpg/t1/memoranda/firefnl.pdf. Accessed March 2006.

Wakefield M, Ruffin R, Campbell D, Staugas R, Beilby J, McCaul K. 1997. A risk screening questionnaire for adult asthmatics to predict attendance at hospital emergency departments. South Australian Asthma Reference Panel. Chest 112:1527–1533.

Ward DJ, Ayres JG. 2004. Particulate air pollution and panel studies in children: A systematic review. Occup Environ Med 61:e13.

WHO. 2000. Air Quality Guidelines for Europe. World Health Organisation. Geneva. Available: www.euro.who.int/document/e71922.pdf. Accessed October 2005.