The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003

R J Delfino, S Brummel, J Wu, H Stern, B Ostro, M Lipsett, A Winer, D H Street, L Zhang, T Tjoa and D L Gillen

Occup. Environ. Med. 2009;66;189-197; originally published online 18 Nov 2008; doi:10.1136/oem.2008.041376

The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003

R J Delfino,[1] S Brummel,[2] J Wu,[1,3] H Stern,[2] B Ostro,[4] M Lipsett,[5] A Winer,[6] D H Street,[7] L Zhang,[5] T Tjoa,[1] D L Gillen[2]

► Additional information is published online only at http://oem.bmj.com/content/vol66/issue3

[1] Department of Epidemiology, School of Medicine, University of California, Irvine, California, USA; [2] Department of Statistics, School of Information and Computer Science, University of California, Irvine, California, USA; [3] Program in Public Health, University of California, Irvine, California, USA; [4] Air Pollution Epidemiology Section, California Office of Environmental Health Hazard Assessment, Oakland, California, USA; [5] Exposure Assessment Section, Environmental Health Investigations Branch, California Department of Health Services, Oakland, California, USA; [6] Department of Environmental Health Sciences, School of Public Health, University of California, Los Angeles, California, USA; [7] Independent consultant, Salem, Oregon, USA

Correspondence to: Dr Ralph J Delfino, Epidemiology Department, School of Medicine, University of California, Irvine, 100 Theory Dr., Suite 100, Irvine, CA 92617-7555, USA; rdelfino@uci.edu

Accepted 18 August 2008
Published Online First 18 November 2008

ABSTRACT

Objective: There is limited information on the public health impact of wildfires. The relationship of cardiorespiratory hospital admissions (n = 40 856) to wildfire-related particulate matter ($PM_{2.5}$) during catastrophic wildfires in southern California in October 2003 was evaluated.

Methods: Zip code level $PM_{2.5}$ concentrations were estimated using spatial interpolations from measured $PM_{2.5}$, light extinction, meteorological conditions, and smoke information from MODIS satellite images at 250 m resolution. Generalised estimating equations for Poisson data were used to assess the relationship between daily admissions and $PM_{2.5}$, adjusted for weather, fungal spores (associated with asthma), weekend, zip code-level population and sociodemographics.

Results: Associations of 2-day average $PM_{2.5}$ with respiratory admissions were stronger during than before or after the fires. Average increases of 70 μg/m^3 $PM_{2.5}$ during heavy smoke conditions compared with $PM_{2.5}$ in the pre-wildfire period were associated with 34% increases in asthma admissions. The strongest wildfire-related $PM_{2.5}$ associations were for people ages 65–99 years (10.1% increase per 10 μg/m^3 $PM_{2.5}$, 95% CI 3.0% to 17.8%) and ages 0–4 years (8.3%, 95% CI 2.2% to 14.9%) followed by ages 20–64 years (4.1%, 95% CI −0.5% to 9.0%). There were no $PM_{2.5}$–asthma associations in children ages 5–18 years, although their admission rates significantly increased after the fires. Per 10 μg/m^3 wildfire-related $PM_{2.5}$, acute bronchitis admissions across all ages increased by 9.6% (95% CI 1.8% to 17.9%), chronic obstructive pulmonary disease admissions for ages 20–64 years by 6.9% (95% CI 0.9% to 13.1%), and pneumonia admissions for ages 5–18 years by 6.4% (95% CI −1.0% to 14.2%). Acute bronchitis and pneumonia admissions also increased after the fires. There was limited evidence of a small impact of wildfire-related $PM_{2.5}$ on cardiovascular admissions.

Conclusions: Wildfire-related $PM_{2.5}$ led to increased respiratory hospital admissions, especially asthma, suggesting that better preventive measures are required to reduce morbidity among vulnerable populations.

The numbers of wildfires and their duration in the USA have increased over the past two decades due to warmer temperatures, earlier snowmelts and less rainfall, all of which are expected to worsen because of global warming.[1] These phenomena will likely impact public health. However, although the adverse effects of urban fine particulate air pollution ($PM_{2.5}$ or particles with an aerodynamic diameter of <2.5 μm) on cardiovascular and respiratory health have been well documented,[2] far fewer studies have evaluated the impacts of wildfire-generated $PM_{2.5}$. $PM_{2.5}$ is the air pollutant with the greatest increase in concentrations during fire events,[3] followed by particulate matter with an aerodynamic diameter of <10 μm (PM_{10}).[4] Studies that have evaluated the impacts of wildfire PM on hospital admissions, emergency department visits or clinic visits found associations with respiratory outcomes.[5–11] There is little research on the impact of wildfire smoke on cardiovascular outcomes; two studies have found no significant associations.[8 9] There have been conflicting reports on wildfire smoke and total mortality.[12 13] Several other studies have found adverse impacts of wildfire smoke on respiratory symptoms, medication use and lung function.[10 14–16]

We present here the largest study to date evaluating the relationships of hospital admissions for cardiorespiratory outcomes to wildfire-associated $PM_{2.5}$ using data from the catastrophic wildfires that struck southern California in the autumn of 2003. We linked $PM_{2.5}$ concentrations estimated at the zip code level[17] to a population-based dataset of hospital admissions using spatial time series analyses of data before, during and after the fires. Strong, dry winds from inland deserts fanned flames from nine distinct fires, which burned nearly three quarters of a million acres and destroyed approximately 5000 residences and outbuildings. The wildfires generated large amounts of dense smoke that covered much of urban southern California (2003 population of 20.5 million).[18] $PM_{2.5}$ and PM_{10} concentrations far exceeded US federal regulatory standards.[3 17] The goal of the present study is to assess the impact of this large wildfire event on serious morbidity.

METHODS

Hospital admission data

Hospital admission data for children and adults were obtained from the California State Office of Statewide Health Planning and Development (OSHPD). Specifically, we analysed 40 856 hospital admissions from the period before the wildfire episode (1–20 October), the episode period across southern California (21–30 October) and the period following the episode (31 October–15 November), for individuals who lived in affected counties and were diagnosed with the respiratory and cardiovascular illnesses listed in table 1. Other variables from OSHPD included in analyses were age, sex, race, ethnicity, five-digit zip code and

admission date. Patient zip code data from OSHPD were geocoded to zip code centroids and linked to air monitoring data and U.S. Census 2000 sociodemographic data. Institutional Review Board approvals were obtained from the California State Health and Human Services Agency, Committee for the Protection of Human Subjects, and from the University of California, Irvine Office of Research Administration.

Analyses were stratified by age groups: paediatric (0–4 and 5–19 years), adult (20–64 years) and elderly (65–99 years), except for chronic obstructive pulmonary disease (COPD, 20–64 and 65–99 years) and cardiovascular outcomes (45–99 years). Census demographic characteristics were missing for 474 admissions due to unmatched zip codes. We also analysed associations for asthma by gender because of differences in the age-dependent prevalence of asthma.

Exposures

We estimated daily PM_{10} and $PM_{2.5}$ concentrations at a zip code level from 1 October through 15 November 2003. These data are presented in more detail in our previous publication.[17] To our knowledge, this was the first study that systematically examined and estimated daily particle concentrations at such a fine spatial resolution over a relatively large study domain for this type of application. Spatially-resolved particle mass data are superior to using only the nearest available monitoring station data because they are expected to better represent personal exposures. We used available air pollution data from governmental network sites to build prediction models. Missing gravimetric PM concentrations from every 3rd or 6th day measurements or due to the incapacitation of monitors by the fires were estimated based on (1) temporal profiles of continuous hourly PM data at co-located or closely located sites and (2) light extinction from visibility data, meteorological conditions and smoke information extracted from moderate resolution imaging spectroradiometer (MODIS) satellite images at a 250 m resolution. Moderately strong prediction equations were developed for gravimetric PM mass at monitoring stations. Light extinction coefficient and MODIS satellite smoke data were the most important predictors of those measurements. Measured $PM_{2.5}$ was more accurately predicted in regression models compared with PM_{10} (R^2 0.78 vs 0.65, respectively). Therefore, the present analysis focuses only on $PM_{2.5}$.

Spatial interpolations of $PM_{2.5}$ concentrations were performed using inverse distance weighting, kriging or cokriging methods for the non-fire periods. Since the fire and smoke created highly heterogeneous pollution surfaces, typical inverse distance weighting and kriging were not suitable during the wildfire period. Therefore, polygons were created based on satellite images to represent each smoke-covered area under different smoke densities. $PM_{2.5}$ concentrations in each smoke-polygon were assigned separately, using measured or estimated concentrations from the predictive models (as described above). For each non-fire and fire day, the spatial $PM_{2.5}$ surfaces and zip code boundary map were overlaid and corresponding $PM_{2.5}$ concentrations were assigned to each zip code centroid (fig 1).

Measurements of daily airborne fungal spores (see online supplement) were carried out in another ongoing study in Riverside County.[19] Pollen concentrations were low and therefore were not included in the analysis. We assumed that Riverside ambient fungal data reflected region-wide trends.

Analysis

Outcomes were the total number of admissions for a diagnostic group within each zip code on each day of the study period. We hypothesised that associations between the wildfires and hospital admission rates would primarily be attributable to an increase in daily zip code-specific levels of $PM_{2.5}$ resulting from the fires. However, it is difficult to separate wildfire-generated PM from other PM sources in this heavily urbanised region. To this end, we constructed a wildfire indicator representing pre-wildfire, wildfire and post-wildfire periods, and tested the interaction between $PM_{2.5}$ and this indicator. We considered product terms to be significant at the $p<0.1$ level. Because dates of the wildfires varied throughout southern California, dates for the wildfire period indicator were defined to be county-specific based on MODIS satellite images of smoke covering any part of the county's urban areas (table 2).

The choice of adjustment covariates was motivated by biological plausibility that the covariate might confound the relationship between wildfire-related $PM_{2.5}$ and hospital admissions or an a priori belief that the variable could affect both $PM_{2.5}$ and admissions. Meteorological covariates from the National Climatic Data Center (http://www.ncdc.noaa.gov/oa/ncdc.html) included relative humidity, temperature and surface pressure gradient. So-called Santa Ana winds coming off the inland desert regions to the east (a large negative pressure gradient) are a strong determinant of wildfire events. There are few data on the effects of Santa Ana winds on asthma or other outcomes, but it is anticipated that hot dry desert winds associated with this weather pattern bring with them high concentrations of bioaerosols. Therefore, for asthma admissions, we also included fungal spores as a covariate. Deuteromycetes (eg, *Alternaria*) tend to increase during hot, dry windy periods.[20]

In addition, we decided a priori that spatial heterogeneity in census demographic factors at the aggregate zip code level (age, gender, race and income distributions) could confound associations. The distributions of each of these potential confounders were obtained at the zip code level from the 2000 U.S. Census (percentage of non-Caucasians, percentage of females, median household income and age distributions). Income was recoded into discrete variables by quartile. To control for zip code population age distribution, we first calculated the percentage of individuals in a zip code younger than 20 years and older than 65 years. Each zip code was then classified into one of four age categories by cross-classification of young (proportion of individuals <20 years old higher than the median proportion across all zip codes) and old (proportion of individuals>65 years old higher than the median proportion across all zip codes).

We also tested various functions of time including weekend versus weekday, day of the week and a smooth of time. In order to investigate residual confounding by date, we allowed for a flexible functional form (via smoothing splines, with degrees of freedom ranging from 1 to 10) (see online supplement). Controlling for day-of-week trend or the flexible time-adjusted models showed the $PM_{2.5}$ associations were robust with respect to these adjustments. We also tested various forms of temperature and relative humidity, including raw continuous scales, smoothed and categorical forms. Those models exhibiting the best fit with the fewest assumptions for functional form included weekend versus weekday, and temperature and relative humidity categorised into quartiles. The full set of adjustment covariates included these variables plus local pressure gradient, fungal spores (for asthma),

Table 1 Number of hospital admission by diagnostic* and age groups

Diagnosis	Total events	Events with U.S. Census 2000 defined population†
All respiratory‡		
Ages 0–4	2158	2143
Ages 5–19	1216	1205
Ages 20–64	8480	8314
Ages 65–99	9456	9357
Total	21 310	21 019
Asthma (ICD-9 493), primary		
Ages 0–4	606	600
Ages 5–19	739	733
Ages 20–64	1165	1151
Ages 65–99	543	538
Total	3053	3022
Acute bronchitis and bronchiolitis (ICD-9 466)		
Ages 0–4	354	353
Ages 5–19	23	23
Ages 20–64	108	106
Ages 65–99	137	136
Total	622	618
Chronic obstructive pulmonary disease (ICD-9 491, 492 and 496)		
Ages 20–64	927	910
Ages 65–99	1973	1950
Total	2900	2860
Pneumonia (ICD-9 480-87)		
Ages 0–4	542	537
Ages 5–19	298	293
Ages 20–64	1721	1686
Ages 65–99	3957	3924
Total	6518	6440
Upper respiratory infections (ICD-9 460–65)		
Ages 0–4	522	518
Ages 5–19	77	77
Ages 20–64	108	104
Ages 65–99	47	47
Total	754	746
All cardiovascular§		
Ages 45–99	27 486	27 170
Ages 65–99	19 380	19 197
Ischaemic heart disease (ICD-9 410–414)		
Ages 45–99	10 448	10 319
Ages 65–99	6491	6430
Cardiac dysrhythmias (ICD-9 426, 427)		
Ages 45–99	4051	4004
Ages 65–99	3048	3018
Congestive heart failure (ICD-9 402, 428)		
Ages 45–99	6202	6144
Ages 65–99	4750	4712
Cerebrovascular disease and stroke (ICD-9 430–438)		
Ages 45–99	5973	5908
Ages 65–99	4465	4422

*Principal cause of admission was coded by version 9 of the International Classification of Diseases (ICD-9); †population with available covariates for census population and census distribution of demographic characteristics used in the multivariate analysis. This excludes subjects aged ⩾100 years (48 (0.23%) respiratory and 51 (0.18%) cardiovascular admissions) because 2000 census age categories needed in the analysis stopped at 99 years; ‡includes all listed specific respiratory ICD-9 plus 7463 additional admissions for the following ICD-9 codes: 277 (cystic fibrosis), 490 (bronchitis NOS), 494 (bronchiectasis), 495 (extrinsic allergic alveolitis), 506 and 508 (other acute/subacute respiratory conditions due to fumes/vapours, or external agents, not separately analysed because n = 44), 786 (symptoms involving the respiratory system/other chest symptoms). §includes all listed specific cardiovascular ICD-9 codes plus 812 additional admissions for ICD-9 codes 440–459 (diseases of the peripheral circulation).

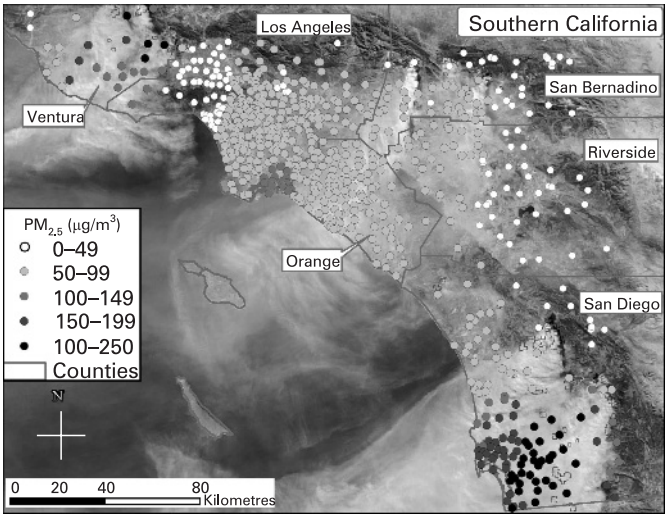

Figure 1 Interpolated $PM_{2.5}$ concentrations (μg/m³) at zip code centroids on 27 October 2003.

county, and zip code-level distributions of median household income, age, gender and race. Effects of covariates on point estimates of $PM_{2.5}$ were small.

Generalised estimating equations for Poisson data[21] were used to estimate the marginal association of daily hospital admission rates with daily $PM_{2.5}$ levels and presence of the wildfires. Log-transformed zip code-specific population estimates were used as the offset (denominator) term in all models. Age-specific population estimates were used as an offset term in the analysis of age group-specific outcomes. In order to obtain asymptotically valid inferences, covariate estimation was carried out using an independence working correlation structure in combination with empirical variance estimates clustering on zip code.[22 23] We note that the use of an independence working correlation structure was motivated by the desire to obtain consistent parameter estimates in the presence of time-varying covariates.[24]

Multiple lag models were considered to investigate associations between $PM_{2.5}$ and hospital admission rates, including a 7-day polynomial distributed lag,[25] and stratified analyses considering different lag associations. We found the 2-day moving average of $PM_{2.5}$ (average of today and yesterday) provided the best fitting model that adequately captured the association between $PM_{2.5}$ and admissions.

RESULTS

PM exposures

During the wildfires, smoke events dramatically increased local PM concentrations and created highly heterogeneous pollution surfaces.[17] For reference, the US National Ambient Air Quality Standard for 24 h average $PM_{2.5}$ is 35 μg/m³. The highest 24 h concentrations were ⩾240 μg/m³ at two sites in San Diego County. Table 2 contains county-level descriptive statistics for $PM_{2.5}$. As expected, average $PM_{2.5}$ concentrations during the wildfire period increased in all counties. Average PM levels during the period following the fires were observed to be lower in all counties relative to the period prior to the fires. This is because of the onshore flow that brought in the cool and moist clean air from the Pacific Ocean that helped end the wildfires.

Table 2 County-level mean particulate matter ($PM_{2.5}$) levels,* Southern California, 1 October–15 November 2003

Daily $PM_{2.5}$ levels ($\mu g/m^3$)	County					
	Los Angeles	Orange	Riverside	San Bernardino	San Diego	Ventura
Before fires						
Dates	01/10–23/10	01/10–23/10	01/10–20/10	01/10–20/10	01/10–24/10	01/10–22/10
Concentration (SD)	27.2 (12.4)	23.3 (9.6)	32.7 (14.7)	35.7 (16.6)	18.5 (6.7)	18.4 (8.3)
During fires						
Dates	24/10–29/10	24/10–28/10	21/10–29/10	21/10–30/10	25/10–30/10	23/10–30/10
Concentration (SD)	54.1 (21)	64.3 (26.5)	42.1 (25.5)	45.3 (28.7)	76.1 (66.6)	50.1 (50.5)
After fires						
Dates	30/10–15/11	29/10–15/11	30/10–15/11	31/10–15/11	31/10–15/11	31/10–15/11
Concentration (SD)	15.9 (5.5)	15.5 (10.2)	16.9 (8.6)	18.4 (8.3)	14.2 (7.2)	12.9 (4.3)

*$PM_{2.5}$ concentrations are calculated with equal weighting per zip code.

Spatial time series analysis of hospital admissions

$PM_{2.5}$ associations: interactions with wildfire period

We found that associations of 2-day lagged average of $PM_{2.5}$ with admissions for most respiratory outcomes were stronger during as compared with before or after the wildfires in models including a product term of wildfire period and $PM_{2.5}$, but the interaction was $p<0.1$ primarily for asthma.

Table 3 shows estimates for the relative change in rates for admissions in relation to a 10 $\mu g/m^3$ increase in $PM_{2.5}$. The table includes results for age and sex (asthma only) subgroups for the entire monitored period, and for wildfire periods. In product term models of $PM_{2.5}$ by wildfire period, $PM_{2.5}$ during the wildfire period was associated with combined respiratory admissions. Asthma admissions across all ages increased by 4.8% (95% CI 2.1% to 7.6%) in relation to $PM_{2.5}$ during the wildfire period, but there was no $PM_{2.5}$ association before or after the fires. The strongest wildfire-related $PM_{2.5}$ associations with asthma admissions were for the elderly, ages 65–99 years (10.1% increase), and children ages 0–4 years (8.3%), followed by adults ages 20–64 years (4.1%). There were no $PM_{2.5}$ associations in school aged children. Among women ages 20–64 years, the strongest asthma and $PM_{2.5}$ association was during the wildfires, but for men those ages it was after the wildfires. Among women ages 65–99, the strongest $PM_{2.5}$ association was after the wildfires, but for men those ages it was during the wildfires. Fungal spores were also significantly associated with asthma admissions in the adjusted model that included $PM_{2.5}$ (see online supplement).

The wildfires led to notably higher particle concentrations, so that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ used for effect estimates in table 3 represents only a small part of that increase. The overall population-weighted concentrations of predicted 24 h $PM_{2.5}$ at the zip code level were 90 $\mu g/m^3$ and 75 $\mu g/m^3$, under heavy and light smoke conditions, respectively, in contrast to concentrations of 20 $\mu g/m^3$ during the non-fire period.[17] Therefore, we rescaled effect estimates to represent the wildfire-related increases in $PM_{2.5}$. A 55 $\mu g/m^3$ increase in $PM_{2.5}$ during light smoke and a 70 $\mu g/m^3$ increase in $PM_{2.5}$ during heavy smoke conditions are predicted to lead to an adjusted 26% and 34% increase in asthma admissions for all ages, respectively.

For combined ages, acute bronchitis admissions increased more in relation to 10 $\mu g/m^3$ $PM_{2.5}$ during the wildfires (9.6%), but there was no association before or after the fires. In subgroup analyses, this association was still evident in children ages 0–4 years and the elderly.

COPD admissions for people ages 20–64 years significantly increased by 6.8% from 10 $\mu g/m^3$ $PM_{2.5}$ during the wildfires, but there was no association before or after the fires. The COPD increase with $PM_{2.5}$ during the fires was smaller for subjects ages 65–99 years (3.1%).

$PM_{2.5}$ was also associated with increased overall pneumonia admissions, both before (4.5%) and during the fires (2.8%). This was consistent across ages, except children ages 5–19 years showed an association only during the wildfires. There were no associations of $PM_{2.5}$ with admissions for upper respiratory infections (not shown).

There was a small relative increase in admission rates for total cardiovascular outcomes in people ages 45–99 years in relation to $PM_{2.5}$ during the fires. There were suggestions of a small increase in admissions for congestive heart failure in relation to $PM_{2.5}$ during the wildfires ($p<0.1$ compared with the pre-wildfire period), and an even smaller increase in admissions for ischaemic heart disease, but for both outcomes, the 95% confidence intervals crossed 1.0. $PM_{2.5}$ was inversely associated with cardiac dysrhythmia admissions across all periods. Admissions for cerebrovascular disease and stroke were positively associated with $PM_{2.5}$ (1.9%) across all periods.

Associations with wildfire period

In this analysis of the wildfire indicator variable, the pre-wildfire period is the referent time. Models were adjusted for the same covariates as $PM_{2.5}$ models, and are shown unadjusted and adjusted for $PM_{2.5}$ (table 4). Generally, there was little change in point estimates adjusting for $PM_{2.5}$. There were significantly increased risks for all respiratory hospital admissions after the fires compared with the pre-fire period. Admissions increased for all ages by 17% ($p<0.001$), and in age groups 5–19 years by 37% ($p<0.008$) and 65–99 years by 15% ($p<0.004$). Unexpected decreased risks of respiratory admissions were found during the fires compared with the pre-fire period in 0–4 year olds and elderly adults.

The period following the fires was associated with a 26% increase in the rate of asthma admissions for all ages. Asthma admissions were also increased during the fires among those aged 5–19 years (25%) and 20–64 years (27%), but associations for both groups were stronger after the fires (56% and 36%, respectively).

Increased risk of asthma admissions for the period during the wildfires was stronger in females ages 5–19 years (49%, $p<0.02$) than males (11%, $p = 0.5$) and in females ages 20–64 years (41%, $p<0.001$) than males (−7.6%, $p = 0.7$) (not shown). Increased risk of asthma admissions for the period after the wildfires was also stronger in females ages 5–19 years (81%, $p<0.01$) than males (39%, $p<0.11$) and in females ages 20–64 years (47%, $p<0.02$) than males (12%, $p = 0.7$).

Table 3 Relative rate of asthma admissions in relation to a 10 μg/m³ increase in 2-day moving average particulate matter ($PM_{2.5}$)

Hospital admissions outcome	All periods RR (95% CI)*	Pre-wildfire period RR (95% CI)	Wildfire period RR (95% CI)	p Value†	Post-wildfire period RR (95% CI)	p Value
All respiratory						
All ages	1.009 (0.999 to 1.018)	1.022 (1.004 to 1.040)	1.028 (1.014 to 1.041)	0.639	0.999 (0.968 to 1.031)	0.198
Ages 0–4	0.994 (0.967 to 1.021)	0.982 (0.921 to 1.046)	1.045 (1.010 to 1.082)	0.103	0.894 (0.807 to 0.991)	0.126
Ages 5–19	1.014 (0.983 to 1.046)	1.026 (0.946 to 1.113)	1.027 (0.984 to 1.076)	0.990	0.958 (0.852 to 1.077)	0.354
Ages 20–64	1.015 (1.002 to 1.029)	1.036 (1.007 to 1.066)	1.024 (1.005 to 1.044)	0.534	1.007 (0.960 to 1.056)	0.315
Ages 65–99	1.009 (0.996 to 1.022)	1.022 (0.994 to 1.050)	1.030 (1.011 to 1.049)	0.649	1.024 (0.976 to 1.074)	0.932
Asthma						
All ages						
Males and females	1.022 (1.001 to 1.042)	0.998 (0.949 to 1.050)	1.048 (1.021 to 1.076)	0.097	0.986 (0.910 to 1.068)	0.792
Males	1.010 (0.980 to 1.040)	1.021 (0.944 to 1.106)	1.031 (0.990 to 1.073)	0.848	1.063 (0.948 to 1.192)	0.553
Females	1.029 (1.001 to 1.058)	0.979 (0.913 to 1.050)	1.059 (1.022 to 1.097)	0.056	0.928 (0.829 to 1.037)	0.412
Ages 0–4						
Males and females	0.996 (0.947 to 1.048)	0.924 (0.824 to 1.035)	1.083 (1.021 to 1.149)	0.017	0.924 (0.767 to 1.113)	0.999
Males	1.018 (0.963 to 1.076)	0.942 (0.815 to 1.089)	1.086 (1.016 to 1.162)	0.101	1.057 (0.839 to 1.332)	0.380
Females	0.937 (0.845 to 1.040)	0.880 (0.706 to 1.099)	1.073 (0.965 to 1.194)	0.116	0.699 (0.515 to 0.949)	0.214
Ages 5–19						
Males and females	1.006 (0.966 to 1.048)	1.045 (0.936 to 1.167)	0.999 (0.935 to 1.068)	0.492	0.918 (0.788 to 1.069)	0.198
Males	0.991 (0.935 to 1.051)	1.034 (0.892 to 1.198)	0.969 (0.883 to 1.064)	0.462	0.979 (0.806 to 1.189)	0.671
Females	1.026 (0.964 to 1.092)	1.065 (0.901 to 1.260)	1.033 (0.943 to 1.132)	0.768	0.831 (0.640 to 1.079)	0.136
Ages 20–64						
Males and females	1.043 (1.012 to 1.076)	1.037 (0.957 to 1.123)	1.041 (0.995 to 1.090)	0.931	1.000 (0.882 to 1.132)	0.624
Males	1.013 (0.954 to 1.077)	1.159 (0.996 to 1.349)	0.939 (0.837 to 1.053)	0.026	1.275 (1.020 to 1.595)	0.486
Females	1.052 (1.015 to 1.090)	0.995 (0.904 to 1.096)	1.064 (1.014 to 1.116)	0.247	0.908 (0.780 to 1.056)	0.310
Ages 65–99						
Males and females	1.027 (0.974 to 1.082)	0.951 (0.849 to 1.064)	1.101 (1.030 to 1.178)	0.032	1.168 (0.967 to 1.412)	0.072
Males	1.046 (0.957 to 1.142)	0.948 (0.804 to 1.116)	1.185 (1.077 to 1.305)	0.029	0.902 (0.629 to 1.294)	0.804
Females	1.018 (0.958 to 1.081)	0.947 (0.813 to 1.102)	1.065 (0.977 to 1.162)	0.195	1.263 (1.024 to 1.557)	0.032
Acute bronchitis and bronchiolitis						
All ages	1.044 (0.990 to 1.102)	1.001 (0.890 to 1.126)	1.096 (1.018 to 1.179)	0.223	1.031 (0.870 to 1.222)	0.779
Ages 0–4	1.017 (0.949 to 1.089)	0.987 (0.847 to 1.149)	1.092 (0.997 to 1.195)	0.276	0.910 (0.700 to 1.183)	0.588
Ages 5–19	No convergence					
Ages 20–64	1.039 (0.912 to 1.183)	1.001 (0.792 to 1.266)	1.044 (0.872 to 1.252)	0.778	1.259 (0.921 to 1.722)	0.275
Ages 65–99	1.134 (1.039 to 1.238)	1.073 (0.764 to 1.505)	1.143 (1.032 to 1.265)	0.730	1.190 (0.865 to 1.638)	0.652
Chronic obstructive pulmonary disease						
Ages 20–99	1.018 (0.994 to 1.042)	1.007 (0.958 to 1.058)	1.038 (1.004 to 1.075)	0.320	1.024 (0.943 to 1.112)	0.728
Ages 20–64	1.022 (0.980 to 1.066)	0.995 (0.916 to 1.081)	1.068 (1.009 to 1.131)	0.161	1.015 (0.893 to 1.153)	0.781
Ages 65–99	1.019 (0.992 to 1.048)	1.014 (0.955 to 1.077)	1.031 (0.990 to 1.074)	0.660	1.023 (0.928 to 1.128)	0.878
Pneumonia						
All ages	1.009 (0.994 to 1.024)	1.045 (1.012 to 1.078)	1.028 (1.007 to 1.050)	0.420	0.980 (0.927 to 1.035)	0.045
Ages 0–4	0.995 (0.944 to 1.049)	1.048 (0.931 to 1.180)	1.018 (0.948 to 1.092)	0.691	0.823 (0.649 to 1.044)	0.089
Ages 5–19	1.030 (0.966 to 1.098)	1.017 (0.882 to 1.172)	1.064 (0.990 to 1.142)	0.586	1.017 (0.767 to 1.349)	0.998
Ages 20–64	1.008 (0.982 to 1.035)	1.041 (0.982 to 1.104)	1.032 (0.994 to 1.072)	0.823	1.013 (0.913 to 1.124)	0.633
Ages 65–99	1.011 (0.993 to 1.030)	1.050 (1.006 to 1.097)	1.029 (1.002 to 1.057)	0.445	0.985 (0.920 to 1.055)	0.127
All cardiovascular	0.996 (0.989 to 1.003)	0.992 (0.976 to 1.009)	1.008 (0.999 to 1.018)	0.104	0.991 (0.964 to 1.019)	0.955
Ischaemic heart disease	0.991 (0.980 to 1.003)	0.990 (0.963 to 1.017)	1.007 (0.990 to 1.024)	0.313	0.989 (0.950 to 1.030)	0.976
Congestive heart failure	0.989 (0.974 to 1.004)	0.978 (0.942 to 1.015)	1.016 (0.993 to 1.039)	0.096	0.969 (0.914 to 1.027)	0.791
Cardiac dysrhythmia	0.980 (0.962 to 0.998)	0.979 (0.935 to 1.025)	0.989 (0.961 to 1.017)	0.721	0.976 (0.912 to 1.044)	0.934
Cerebrovascular disease and stroke	1.019 (1.004 to 1.035)	1.015 (0.980 to 1.052)	1.016 (0.997 to 1.036)	0.971	1.044 (0.987 to 1.104)	0.379

*Rate ratio and 95% confidence interval per 10 μg/m³ increase in 2-day moving average $PM_{2.5}$, adjusted for fungal spore counts (asthma only), race, gender, county, median income, weekend, relative humidity, temperature, age and pressure gradient. RR×100 is the percentage increase in hospital admissions. Estimates for the three strata are derived from the product term models, while estimates for the full period are from a model without interaction terms; †the product term p value for the difference with the pre-fire period.

Admissions for acute bronchitis and bronchiolitis for combined ages were increased by 48% after the fires. The association for the post-fire period was seen in both ages 0–4 years (51%) and ages 20–64 years (137%). Pneumonia admissions for ages 0–4, 20–64 and 65–99 years were 46%, 30% and 27% higher during the period after the fires, respectively.

There was a 6.1% increased risk of combined cardiovascular admissions ($p<0.05$), and an 11.3% increased risk of congestive heart failure admissions after the fires ($p<0.06$). However, risk of cardiovascular admissions was lower during the fires by 4.4%. A relative increase in cerebrovascular disease and stroke admissions during the wildfires may have been attributable to

Table 4 Relative rate of respiratory admissions in relation to wildfire period

Hospital admissions outcome	n*	Pre-wildfire period (referent)	Wildfire period RR (95% CI)†		Post-wildfire period RR (95% CI)	
			Unadjusted for $PM_{2.5}$	Adjusted for $PM_{2.5}$	Unadjusted for $PM_{2.5}$	Adjusted for $PM_{2.5}$
All respiratory						
All ages	21 019	1.00	0.961 (0.916 to 1.008)	0.903 (0.850 to 0.960)	1.143 (1.072 to 1.219)	1.173 (1.097 to 1.253)
Ages 0–4	2143	1.00	0.865 (0.757 to 0.989)	0.842 (0.717 to 0.988)	1.152 (0.957 to 1.388)	1.162 (0.954 to 1.415)
Ages 5–19	1205	1.00	1.098 (0.910 to 1.324)	1.087 (0.863 to 1.370)	1.373 (1.089 to 1.732)	1.467 (1.142 to 1.883)
Ages 20–64	8314	1.00	0.991 (0.922 to 1.066)	0.923 (0.843 to 1.012)	1.074 (0.971 to 1.188)	1.104 (0.992 to 1.228)
Ages 65–99	9357	1.00	0.932 (0.867 to 1.003)	0.874 (0.795 to 0.959)	1.147 (1.045 to 1.259)	1.193 (1.084 to 1.313)
Asthma						
All ages	3022	1.00	1.088 (0.965 to 1.227)	0.992 (0.856 to 1.149)	1.264 (1.085 to 1.473)	1.336 (1.134 to 1.573)
Ages 0–4	600	1.00	0.806 (0.632 to 1.029)	0.714 (0.515 to 0.990)	1.092 (0.759 to 1.572)	1.133 (0.777 to 1.654)
Ages 5–19	733	1.00	1.254 (0.999 to 1.575)	1.282 (0.958 to 1.716)	1.564 (1.160 to 2.109)	1.629 (1.184 to 2.243)
Ages 20–64	1151	1.00	1.273 (1.067 to 1.518)	1.221 (0.979 to 1.524)	1.362 (1.043 to 1.779)	1.486 (1.111 to 1.987)
Ages 65–99	538	1.00	0.869 (0.657 to 1.151)	0.645 (0.450 to 0.925)	0.924 (0.606 to 1.408)	1.005 (0.650 to 1.552)
Acute bronchitis/ bronchiolitis						
All ages	618	1.00	1.143 (0.878 to 1.490)	0.959 (0.696 to 1.321)	1.482 (1.042 to 2.109)	1.580 (1.089 to 2.291)
Ages 0–4	353	1.00	1.128 (0.819 to 1.555)	0.899 (0.607 to 1.333)	1.520 (0.947 to 2.440)	1.547 (0.954 to 2.507)
Ages 5–19	23	1.00				
Ages 20–64	106	1.00	1.350 (0.688 to 2.648)	1.320 (0.608 to 2.863)	2.454 (1.068 to 5.640)	2.515 (1.055 to 5.998)
Ages 65–99	136	1.00	1.166 (0.643 to 2.115)	0.934 (0.422 to 2.066)	0.911 (0.428 to 1.942)	0.997 (0.439 to 2.262)
Chronic obstructive pulmonary disease						
Ages 20–99	2860	1.00	0.988 (0.875 to 1.115)	0.913 (0.779 to 1.069)	1.043 (0.885 to 1.228)	1.064 (0.897 to 1.262)
Ages 20–64	910	1.00	0.967 (0.779 to 1.201)	0.873 (0.660 to 1.156)	1.175 (0.862 to 1.601)	1.311 (0.954 to 1.802)
Ages 65–99	1950	1.00	1.002 (0.869 to 1.156)	0.926 (0.767 to 1.117)	0.985 (0.811 to 1.196)	0.981 (0.798 to 1.206)
Pneumonia						
All ages	6440	1.00	0.943 (0.868 to 1.025)	0.888 (0.799 to 0.986)	1.294 (1.158 to 1.446)	1.318 (1.174 to 1.479)
Ages 0–4	537	1.00	0.938 (0.705 to 1.247)	0.951 (0.678 to 1.333)	1.458 (0.974 to 2.182)	1.374 (0.885 to 2.133)
Ages 5–19	293	1.00	0.891 (0.604 to 1.312)	0.830 (0.541 to 1.272)	0.960 (0.588 to 1.569)	0.969 (0.578 to 1.624)
Ages 20–64	1686	1.00	0.927 (0.795 to 1.081)	0.837 (0.690 to 1.016)	1.314 (1.064 to 1.622)	1.300 (1.047 to 1.615)
Ages 65–99	3924	1.00	0.959 (0.861 to 1.068)	0.899 (0.782 to 1.033)	1.277 (1.102 to 1.481)	1.331 (1.142 to 1.552)
			Unadjusted for $PM_{2.5}$	Adjusted for $PM_{2.5}$	Unadjusted for $PM_{2.5}$	Adjusted for $PM_{2.5}$
All cardiovascular‡	27 170	1.00	0.958 (0.920 to 0.997)	0.947 (0.902 to 0.994)	1.061 (1.006 to 1.119)	1.053 (0.994 to 1.114)
Ischaemic heart disease	10319	1.00	0.913 (0.852 to 0.978)	0.905 (0.832 to 0.985)	1.029 (0.943 to 1.123)	1.029 (0.936 to 1.131)
Congestive heart failure	6144	1.00	0.891 (0.817 to 0.972)	0.911 (0.819 to 1.014)	1.113 (0.997 to 1.242)	1.105 (0.982 to 1.244)
Cardiac dysrhythmia	4004	1.00	0.968 (0.874 to 1.072)	0.964 (0.851 to 1.093)	1.089 (0.949 to 1.251)	1.057 (0.914 to 1.223)
Cerebrovascular disease and stroke	5908	1.00	1.066 (0.981 to 1.159)	1.017 (0.922 to 1.123)	1.013 (0.907 to 1.132)	1.013 (0.902 to 1.138)

*Number of hospital admissions for zip codes with defined populations; †adjusted for race, gender, county, median income, weekend, relative humidity, temperature, age and pressure gradient; ‡cardiovascular admissions were for subjects ages 45–99 years.
$PM_{2.5}$, particulate matter.

a cross-period effect of $PM_{2.5}$ (table 3) because this period association was confounded in the model adjusting for $PM_{2.5}$.

DISCUSSION

This is the first study to systematically examine and estimate the impacts on hospital admissions from wildfire-related $PM_{2.5}$ at such a fine spatial resolution (zip codes) over a large urban region. During the wildfire period, smoke events dramatically increased $PM_{2.5}$ compared to the preceding non-fire period. The wildfires and associated $PM_{2.5}$ were significantly associated with hospital admissions for respiratory illnesses, especially asthma, but also acute bronchitis and COPD. The impact on cardiovascular admissions was weaker.

Although product terms between $PM_{2.5}$ and the wildfire period indicator were not significant at the $p<0.1$ level in many models, we still observed a trend of stronger associations for $PM_{2.5}$ with respiratory admissions during the wildfire period. Some models showed increased admissions in relation to $PM_{2.5}$ before the wildfires, possibly due to the relatively high concentration of urban PM seen during this hot period (table 2). Some models also showed increased admissions in relation to $PM_{2.5}$ after the wildfires, despite much lower $PM_{2.5}$ concentrations. This may have been attributable to notable increases in respiratory admissions seen then, possibly due to a delayed impact of wildfire smoke.

Models with the wildfire period indicator support this possibility and suggest that some effects of wildfires are not entirely explained by $PM_{2.5}$ exposures. Results yielded inconsistencies for respiratory and cardiovascular admissions when comparing product term models for $PM_{2.5}$ by period to models using the period indicator alone. There were nominal associations of daily $PM_{2.5}$ during the wildfires with cardiovascular admissions, but the period indicator showed associations only after the wildfires. Non-asthma respiratory admission rates were also most strongly increased after the wildfires ended compared with the pre-fire period, while the $PM_{2.5}$ association was generally strongest during the wildfires. We also found the period following the wildfires was significantly associated with higher overall asthma admission rates. These associations were stronger among females. Asthma admissions were increased during the fires as well, but evident only among females ages 5–19 and ages 20–64. Possible reasons for stronger associations

among females include the differential impact of hormones and the menstrual cycle, airway function and structure, atopy and perception of symptoms.[26]

Although there was no association of asthma admissions with $PM_{2.5}$ in young people ages 5–19 years, the periods during and after the wildfires were significantly associated with increased admissions in this group. We speculate this may be attributable to unmeasured volatile (non-particulate) toxic air pollutants, including those associated with the more than 5000 buildings that burned. Alternatively, factors associated with the fires, such as psychosocial stress, could have led to effects that were independent of $PM_{2.5}$.

Associations with the post-wildfire period and wildfire-related $PM_{2.5}$ were also found for acute bronchitis and bronchiolitis, and pneumonia. This is the first report of wildfire associations with admissions for acute bronchitis and bronchiolitis, and pneumonia.

We also found a significantly increased risk of admissions for total cardiovascular outcomes and congestive heart failure after the fires. It is possible that systemic inflammation increases more strongly in relation to sustained multiday exposures to air pollutants than with acute single day exposures, as recently shown in our panel study of subjects with coronary artery disease.[27] Analyses of the London "killer smog" of 1952,[28] and recent analyses of particulate air pollution in Dublin, Ireland,[29] suggest that there may be delayed effects for weeks to months. The post-fire increases in cardiorespiratory admissions may be attributed to the following:

- 1) People may delay deciding to go to hospital until symptoms become too severe[30];
- 2) Cumulative biological effects of wildfire PM may culminate in severe symptoms many days after the initial cardiorespiratory impact. For example, most subjects with asthma show a progressive clinical and functional deterioration that takes place over hours to weeks[31];
- 3) Sustained effects of wildfire PM may lead to susceptibility to, or increased severity of, later respiratory infections, possibly through alterations in immune function or respiratory clearance mechanisms.

The strongest evidence for delayed effects in our study was the post-fire increase in asthma admissions combined with the association between asthma admission and $PM_{2.5}$ during the wildfires. However, given past annual trends (see online supplement), it is possible that asthma admissions following the wildfire period would have increased at this time of year anyway. This also applies to the post-fire increases in admissions for acute bronchitis and bronchiolitis, and pneumonia. Other limitations are that the period analysis does not have the temporal resolution of the daily time series analysis of $PM_{2.5}$. Therefore, differences in results of these analyses could result due to imprecision in the estimate for the non-quantitative indicator variable. Furthermore, power may be limited for specific outcomes subdivided by gender and age, which would apply to several nominally significant associations we found.

Our results for respiratory admissions are consistent with two other studies of the 2003 southern California wildfires using other less severe outcomes and focusing on particular regions, including emergency department visits in San Diego county[11 32] and respiratory symptoms in 16 towns in southern California.[16] Kunzli *et al*[16] reported results for school children in an ongoing cohort study who were potentially affected by the wildfires. They found parental self-reports of the smell of fire smoke indoors were associated with reported asthma attacks, wheezing, cough, bronchitis, colds, upper respiratory symptoms, medication usage and physician visits. Authors also analysed the impacts of between-community differences in PM_{10} using data from our study.[17] Changes in PM_{10} were associated with upper respiratory symptoms, cough and unspecified medication use.

Several investigations of wildfires have identified people with asthma as an especially sensitive subpopulation, using analyses of emergency department visits in California mountain counties during wildfires in 1987,[6] emergency department visits in eight Florida hospitals during wildfires in 1998,[5] and hospital admissions during the 1997 Indonesian wildfires.[9] A report from Australia examining smoke from bushfires and asthma emergency department visits found no association.[33]

Other time series studies have shown associations of asthma hospital admissions with urban air pollution.[34] However, the period of observation in our investigation is far shorter than most time series investigations, and thus statistical power is lower. Despite this, we found strong associations between $PM_{2.5}$ and hospital admissions. We attribute this to the large increase in wildfire-related PM, and the spatial time series approach, which likely reduced exposure error compared with the typical use of widely-dispersed regional PM data. Nevertheless, we are still limited by aggregate (not personal) exposure data.

This is the first report of associations of wildfire-related $PM_{2.5}$ with admissions for acute bronchitis and bronchiolitis, and for pneumonia. Our results showing increased COPD admissions in relation to $PM_{2.5}$ during the wildfires are consistent with a study of increased COPD hospital admissions during the 1997 Indonesian wildfires,[9] increased COPD emergency department visits during the 1987 wildfires in California mountain counties,[6] and respiratory symptoms in a panel of 21 patients with COPD associated with a forest fire near Denver, Colorado in June 2002.[35]

Total cardiovascular and congestive heart failure admissions increased only in the period following the wildfires. However, there was a small relative increase in admission rates for total cardiovascular outcomes in relation to $PM_{2.5}$ during the fires. Cerebrovascular disease and stroke were significantly increased in relation to $PM_{2.5}$ across the entire study period. Unexpected findings were the inverse associations for cardiac dysrhythmias and $PM_{2.5}$ across the whole period. While urban particles generally have been associated with a variety of adverse cardiovascular outcomes,[2] including stroke,[36] there is little research investigating the effects of smoke from wildfires or wood combustion on circulatory disease.[4] Our results can only be compared to null associations for cardiovascular hospital admissions during the 1997 Indonesian wildfires.[9] Moore *et al*[8] found that, although there was an excess of respiratory complaints, physician visits for cardiovascular illnesses in regions of British Columbia, Canada were not associated with wildfires.

The mechanisms explaining our findings for wildfire smoke are likely somewhat similar to those found for pollutant components from fossil fuel combustion. Evidence is mounting that urban air pollution triggers oxidative stress and inflammation.[2] A study of people exposed to forest fire smoke in Indonesia in 1997 showed increased circulating levels of interleukin-1β and interleukin-6 during the smoke period.[37] An experimental study of subjects exposed to clean air versus wood smoke in a chamber showed increased airway inflammatory responses (exhaled alveolar NO) and evidence of increased oxidative stress (malonadehyde in breath condensates).[38] An in

Main messages

- Wildfire-related $PM_{2.5}$ led to significantly increased asthma, bronchitis and COPD hospital admissions.
- Sensitive subgroups included young children and the elderly.

Policy implications

- In addition to advisories to avoid outdoor activities that increase exposure during wildfires, preventive measures need to be taken where possible to reduce exacerbations of asthma
- Preventive measures may include advisories for the early use of anti-inflammatory medications at the first sign of increasing asthma symptoms.
- The health impacts of wildfires reported here are anticipated to increase worldwide due to global warming, which has broad policy implications.

vitro study using mouse alveolar macrophages tested the effects of size-segregated PM from transported wildfire smoke collected in Helsinki, Finland.[39] Investigators showed that although the transported particles induced less cytokine production per unit mass compared with urban particles, they found enhanced inflammatory and cytotoxic activities per cubic meter of air due to the increased particulate mass concentration in the accumulation mode size range (0.1–2.5 μm in diameter). This might explain our finding of a larger asthma association per 10 $\mu g/m^3$ $PM_{2.5}$ during the wildfires as compared with the pre-wildfire period as simply due to the considerably higher concentrations rather than higher toxicity of wildfire smoke.

It is also possible that unmeasured volatile and semivolatile organic compound components are important in the effects of wildfire smoke, but such data are rarely available. In the present study, these include toxic gases emitted from synthetic materials in the approximately 5000 residences and outbuildings that burned.

Conclusions

We conclude the catastrophic wildfires that struck southern California in October of 2003 led to significantly increased hospital admissions for respiratory illnesses, especially asthma. Southern California experienced a second similar wildfire disaster in October 2007, yielding the two largest wildfire disasters in California's history within this recent 4-year period. A concern is that growing impacts of global warming on wildfire risk will continue to impact public health in similar regions across the globe.[1]

Given there were significant morbidity impacts associated with wildfire-related $PM_{2.5}$, we recommend that in addition to advisories to avoid outdoor activities that increase exposure during wildfires, preventive measures need to be taken where possible to reduce exacerbations of asthma. This may include the early use of anti-inflammatory medications at the first sign of increasing asthma symptoms. All of the health impacts identified in this study occurred in the face of numerous advisories by public health agencies and the media to avoid outdoor activities and to use air conditioning. Additional preventive measures in susceptible people including those with persistent asthma, such as the use of indoor air filters,[10 40] should be considered and then systematically evaluated in future wildfires.

Acknowledgements: We thank Joe Cassmassi and others at the South Coast Air Quality Management District for assistance with the meteorological and air pollutant data.

Funding: This study was funded by the South Coast Air Quality Management District contract no. 04182, and the National Institutes of Health, National Institute of Environmental Health Sciences grant no. ES-11615.

Competing interests: None.

REFERENCES

1. **Westerling AL,** Hidalgo HG, Cayan DR, *et al*. Warming and earlier spring increase western U.S. forest wildfire activity. *Science* 2006;**313**:940–3.
2. **Pope CA 3rd,** Dockery DW. Health effects of fine particulate air pollution: lines that connect. *J Air Waste Manag Assoc* 2006;**56**:709–42.
3. **Phuleria H,** Fine PM, Zhu Y, *et al*. Air quality impacts of the October 2003 Southern California wildfires. *J Geophys Res* 2005;**110**:D07S20.
4. **Naeher LP,** Brauer M, Lipsett M, *et al*. Wood smoke health effects: a review. *Inhal Toxicol* 2007;**19**:67–106.
5. **Centers for Disease Control and Prevention (CDC)**. Surveillance of morbidity during wildfires--Central Florida, 1998. *MMWR Morb Mortal Wkly Rep* 1999;**48**:78–9.
6. **Duclos P,** Sanderson LM, Lipsett M. The 1987 forest fire disaster in California: assessment of emergency room visits. *Arch Environ Health* 1990;**45**:53–8.
7. **Emmanuel SC.** Impact to lung health of haze from forest fires: the Singapore experience. *Respirology* 2000;**5**:175–82.
8. **Moore D,** Copes R, Fisk R, *et al*. Population health effects of air quality changes due to forest fires in British Columbia in 2003: estimates from physician-visit billing data. *Can J Public Health* 2006;**97**:105–8.
9. **Mott JA,** Mannino DM, Alverson CJ, *et al*. Cardiorespiratory hospitalizations associated with smoke exposure during the 1997, Southeast Asian forest fires. *Int J Hyg Environ Health* 2005;**208**:75–85.
10. **Mott JA,** Meyer P, Mannino D, *et al*. Wildland forest fire smoke: health effects and intervention evaluation, Hoopa, California, 1999. *West J Med* 2002;**176**:157–62.
11. **Viswanathan S,** Eris L, Diunugala N, *et al*. An analysis of effects of San Diego wildfire on ambient air quality. *J Air Waste Manag Assoc* 2006;**56**:56–67.
12. **Sastry N.** Forest fires, air pollution, and mortality in Southeast Asia. *Demography* 2002;**39**:1–23.
13. **Vedal S,** Dutton SJ. Wildfire air pollution and daily mortality in a large urban area. *Environ Res* 2006;**102**:29–35.
14. **Frankenberg E,** McKee D, Thomas D. Health consequences of forest fires in Indonesia. *Demography* 2005;**42**:109–29.
15. **Kunii O,** Kanagawa S, Yajima I, *et al*. The 1997 haze disaster in Indonesia: its air quality and health effects. *Arch Environ Health* 2002;**57**:16–22.
16. **Kunzli N,** Avol E, Wu J, *et al*. Health effects of the 2003 Southern California wildfires on children. *Am J Respir Crit Care Med* 2006;**174**:1221–8.
17. **Wu J,** Winer A, Delfino R. Exposure assessment of particulate matter air pollution before, during, and after the 2003 southern California wildfires. *Atmos Environ* 2006;**40**:3333–8.
18. **State of California**. *California county population estimates and components of change by year, July 1, 2000–2006*. Sacramento, CA: California Department of Finance 2006.
19. **Delfino RJ,** Zeiger RS, Seltzer JM, *et al*. The effect of outdoor fungal spore concentrations on asthma severity. *Environ Health Perspect* 1997;**105**:622–35.
20. **Weber RW.** Meteorologic variables in aerobiology. *Immunol Allergy Clin North Am* 2003;**23**:411–22.
21. **Zeger SL,** Liang KY. Longitudinal data analysis for discrete and continuous outcomes. *Biometrics* 1986;**42**:121–30.
22. **Huber PJ.** The behavior of maximum likelihood estimates under nonstandard conditions. In: Le Cam LM, Neyman J, eds. *Proceedings of the Fifth Berkeley Symposium on Mathematical Statistics and Probability*. Vol 1. Berkeley, CA: University of California Press, 1967:221–3.
23. **White H.** A heteroskedasticity-consistent covariance matrix estimator and a direct test for heteroskedasticity. *Econometrica* 1980;**48**:817–30.
24. **Pepe MS,** Anderson GL. A cautionary note on inference for marginal regression models with longitudinal data and general correlated response data. *Communications in Statistics - Simululation and Computation* 1994;**23**:939–51.
25. **Schwartz J.** The distributed lag between air pollution and daily deaths. *Epidemiology* 2000;**11**:320–6.
26. **Becklake MR,** Kauffmann F. Gender differences in airway behaviour over the human life span. *Thorax* 1999;**54**:1119–38.
27. **Delfino RJ,** Staimer N, Tjoa T, *et al*. Circulating biomarkers of inflammation, antioxidant activity, and platelet activation are associated with urban air pollution in elderly subjects with a history of coronary artery disease. *Environ Health Perspect* 2008;**116**:898–906.
28. **Bell ML,** Davis DL. Reassessment of the lethal London fog of 1952: novel indicators of acute and chronic consequences of acute exposure to air pollution. *Environ Health Perspect* 2001;**109**(Suppl 3):389–94.
29. **Goodman PG,** Dockery DW, Clancy L. Cause-specific mortality and the extended effects of particulate pollution and temperature exposure. *Environ Health Perspect* 2004;**112**:179–85.

30. **Janson S,** Becker G. Reasons for delay in seeking treatment for acute asthma: the patient's perspective. *J Asthma* 1998;**35**:427–35.
31. **Rodrigo GJ,** Rodrigo C, Hall JB. Acute asthma in adults: a review. *Chest* 2004;**125**:1081–1102.
32. **Johnson JM,** Hicks L, McClean C, *et al*. Leveraging syndromic surveillance during the San Diego wildfires, 2003. *MMWR Morb Mortal Wkly Rep* 2005;**54**(Suppl):190.
33. **Smith MA,** Jalaludin B, Byles JE, *et al*. Asthma presentations to emergency departments in western Sydney during the January 1994 bushfires. *Int J Epidemiol* 1996;**25**:1227–36.
34. **Sheppard L,** Levy D, Norris G, *et al*. Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987–1994. *Epidemiology* 1999;**10**:23–30.
35. **Sutherland ER,** Make BJ, Vedal S, *et al*. Wildfire smoke and respiratory symptoms in patients with chronic obstructive pulmonary disease. *J Allergy Clin Immunol* 2005;**115**:420–2.
36. **Wellenius GA,** Schwartz J, Mittleman MA. Air pollution and hospital admissions for ischemic and hemorrhagic stroke among Medicare beneficiaries. *Stroke* 2005;**36**:2549–53.
37. **van Eeden SF,** Tan WC, Suwa T, *et al*. Cytokines involved in the systemic inflammatory response induced by exposure to particulate matter air pollutants (PM_{10}). *Am J Respir Crit Care Med* 2001;**164**:826–30.
38. **Barregard L,** Sällsten G, Andersson L, *et al*. Experimental exposure to wood smoke: effects on airway inflammation and oxidative stress. *Occup Environ Med* 2008;**65**:319–24.
39. **Jalava PI,** Salonen RO, Halinen AI, *et al*. In vitro inflammatory and cytotoxic effects of size-segregated particulate samples collected during long-range transport of wildfire smoke to Helsinki. *Toxicol Appl Pharmacol* 2006;**215**:341–53.
40. **Henderson DE,** Milford JB, Miller SL. Prescribed burns and wildfires in Colorado: impacts of mitigation measures on indoor air particulate matter. *J Air Waste Manag Assoc* 2005;**55**:1516–26.