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Differential respiratory health effects from the 2008 northern California wildfires: A spatiotemporal approach

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ABSTRACT

We investigated health effects associated with fine particulate matter during a long-lived, large wildfire complex in northern California in the summer of 2008. We estimated exposure to $PM_{2.5}$ for each day using an exposure prediction model created through data-adaptive machine learning methods from a large set of spatiotemporal data sets. We then used Poisson generalized estimating equations to calculate the effect of exposure to 24-hour average $PM_{2.5}$ on cardiovascular and respiratory hospitalizations and ED visits. We further assessed effect modification by sex, age, and area-level socioeconomic status (SES). We observed a linear increase in risk for asthma hospitalizations (RR=1.07, 95% CI=(1.05, 1.10) per 5 $\mu g/m^3$ increase) and asthma ED visits (RR=1.06, 95% CI=(1.05, 1.07) per 5 $\mu g/m^3$ increase) with increasing $PM_{2.5}$ during the wildfires. ED visits for chronic obstructive pulmonary disease (COPD) were associated with $PM_{2.5}$ during the fires (RR=1.02 (95% CI=(1.01, 1.04) per 5 $\mu g/m^3$ increase) and this effect was significantly different from that found before the fires but not after. We did not find consistent effects of wildfire smoke on other health outcomes. The effect of $PM_{2.5}$ during the wildfire period was more pronounced in women compared to men and in adults, ages 20–64, compared to children and adults 65 or older. We also found some effect modification by area-level median income for respiratory ED visits during the wildfires, with the highest effects observed in the ZIP codes with the lowest median income. Using a novel spatiotemporal exposure model, we found some evidence of differential susceptibility to exposure to wildfire smoke.

1. Introduction

Wildfires have been increasing in frequency and severity in western North America, and this increase has been associated with earlier spring snowmelt and higher temperatures (Westerling et al., 2006). The risk of wildfires is projected to increase in California (Westerling and Bryant, 2008; Westerling et al., 2011) and in many parts of the world (Liu et al., 2010; Moritz et al., 2012) under probable future climate change scenarios.

Smoke from wildfires contains many pollutants of concern for public health including nitrogen dioxide, ozone, carbon monoxide, polycyclic aromatic hydrocarbons, aldehydes, and particulate matter less than 2.5 μm in aerodynamic diameter ($PM_{2.5}$) (Naeher et al., 2007). Previous epidemiological studies of wildfire smoke exposure have found consistent evidence of respiratory health effects in general and most specifically for exacerbations of asthma and chronic obstructive pulmonary disease (COPD). Findings for other health outcomes have been inconsistent across studies, and insufficient research has investigated whether particular

Abbreviations: AOD, aerosol optical depth; BRFSS, Behavioral Risk Factor Surveillance System; CI, confidence interval; COPD, chronic obstructive pulmonary disease; CV, cross-validated; ED, emergency department; GBM, generalized boosting method; GEE, generalized estimating equations; GOES, Geostationary Operational Environmental Satellite; IHD, ischemic heart disease; IQR, interquartile range; MODIS, MODerate resolution Imaging Spectroradiometer; PM, particulate matter; $PM_{2.5}$, particulate matter less than or equal to 2.5 μm in aerodynamic diameter; QICu, Quasi Information Criterion for GEE models; RH, relative humidity; RMSE, root mean squared error; RR, relative risk; RUC, Rapid Update Cycle model; SES, socio-economic status; WRF-Chem, Weather Research and Forecasting model with Chemistry; ZCTA, ZIP code tabulation area

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population subgroups are more susceptible to wildfire smoke exposure (Reid et al., 2016). Additionally, the existing literature lacks information on the shape of the exposure-response curve for wildfire smoke. Such information could be useful to decision-makers issuing health advisories during wildfire events.

This study investigates a particularly long-lived, large wildfire complex that occurred in northern California in the summer of 2008. A combination of meteorological conditions and difficulty with fire suppression contributed to very high air pollution levels throughout northern California (Reid et al., 2009). Smoke from the fires covered a large region with large population centers for almost six weeks, making this an important fire episode for analysis of public health effects.

We examined the effects of this relatively long exposure on cardiovascular and respiratory hospital admissions and emergency department (ED) visits within the population of northern and central California using a novel spatiotemporal exposure model. We aimed to assess if there were differential health effects of $PM_{2.5}$ during the wildfire compared to reference periods before and after the fires, to assess at what level of $PM_{2.5}$ the risk of adverse health effects starts to increase, and to identify population subgroups that were more susceptible to wildfire smoke during this event.

2. Methods

2.1. Study setting

The 2008 northern California wildfire complex consisted of thousands of wildfires ignited by a large lightning storm the weekend of June 20–21, 2008. Most of these fires were contained by the end of July 2008. We defined the pre-fire period as May 6 to June 19 (days=43), the fire period as June 20 to July 31 (days=42), and the post-fire period as August 1 to September 15 (days=46). These cut points were determined based on having similar numbers of days in the three time periods, the timing of the onset of the fires ignited by the lightning storm, and the designation that most of the fires had been contained by the end of July. The spatial confines of our analysis were the ZIP codes that fall within the following air basins: the Sacramento Valley, the San Francisco Bay Area, the Mountain Counties, Lake County, the North Central Coast, and the northern part of the San Joaquin Valley (Fig. 1). Most of the fires were located in mountainous regions that ringed the northern Central Valley: in the Trinity Alps west of Redding, the Sierra Nevada in the Mountain Counties to the east of Redding and Chico, and some fires near Big Sur, which is along the coast west of Fresno.

2.2. Exposure data

We estimated exposure to $PM_{2.5}$ for each day in each ZIP code using an exposure prediction model that was created from a large set of spatiotemporal data sets through data-adaptive machine learning methods. This method used 10-fold cross validation (CV) to select from within a large number of predictor variables and across many different statistical algorithms to optimize prediction of $PM_{2.5}$. The 24-hour average $PM_{2.5}$ values at 112 monitoring stations (Fig. 1) were used as the dependent variable. The predictor variables included aerosol optical depth (AOD) from the Geostationary Operational Environmental Satellite (GOES), output from the Weather Research and Forecasting coupled with Chemistry (WRF-Chem) model, various meteorological variables from the Rapid Update Cycle model, Julian date, weekend, amount of land use types within 1 km, the X-coordinate, the Y-coordinate, elevation, and traffic counts. In a previous paper (Reid et al., 2015), the generalized boosting model (GBM) predicted 24-hour average $PM_{2.5}$ better than the 10 other algorithms with a CV-R^2 of 0.80 using all of the predictor variables. In this analysis, we re-ran the GBM model and expanded the time period to include time periods before and after the fires. Accordingly, we removed predictor variables that were not available for the before and after time periods (e.g., local aerosol optical depth (AOD) and distance to the nearest fire cluster). In this modeling run, a GBM model containing 24 out of 25 possible predictor variables had an out-of-sample CV-R^2 of 0.79 and a CV root mean squared error (RMSE) of 1.44 $\mu g/m^3$, but including only the six most predictive variables resulted in almost equally good performance with a CV-R^2 of 0.78 and a CV-RMSE of 1.46 $\mu g/m^3$. The six most predictive variables were AOD from the GOES satellite, WRF-Chem output, Julian date, surface pressure, the X-coordinate and the Y-coordinate. The model predicted observed values better during the fires than before or after (Supplement Fig. S1).

We used this more parsimonious model to estimate exposures at the population-weighted centroid of each of 781 ZIP code tabulation areas (ZCTA), spatial constructs used by the US Census Bureau to create ZIP codes from census-area designations, using ArcGIS 10.1 (ESRI, 2012). The predictor variables in the exposure model are assigned to each ZCTA as the value of that input variable closest to the population-weighted centroid for that ZIP code. Predicted values for all ZIP codes in the study area over time are presented in Fig. 2.

2.3. Health data

We obtained daily counts of hospital admission visits (OSHPD, 2008b) and ED visits (OSHPD, 2008a) for each ZIP code in the study area for the following causes (ICD-9 code): asthma (493), COPD (496, 491–492), pneumonia (480–486), ischemic heart disease (IHD) (410–414), cardiac dysrhythmias and conduction disorders (426–427), heart failure (428), cerebrovascular disease (430–435, 437), and hypertension (401–405). The total population based on the 2010 US Census for all ZIP codes in the study area was 12.7 million.

2.4. Covariate data

Temperature and RH data are 24-hour averages taken from the Rapid Update Cycle (RUC) model from the National Climatic Data Center (http://ruc.noaa.gov/). We assigned the value from the grid cell that overlaid the population-weighted centroid of each ZIP code. We obtained estimates by ZIP code of population, median income, percent of the population over 65, percent of the population living in owner-occupied housing, and percent of the population with less than a high school diploma from the 2000 US Census. We used smoking prevalence estimates derived from Behavioral Risk Factor Surveillance System (BRFSS) data by ZIP code for the 2006–2010 time period based on the 2000 census ZIP codes (Ortega Hinojosa et al., 2014). For the ZIP codes (N=66, 8.5%) in our analysis that were created after 2000, we used county-level estimates. Daily 8-hour maximum ozone concentrations come from WRF-Chem.

2.5. Statistical analysis

We used Poisson generalized estimating equations (GEE) to calculate the population-averaged effect of exposure to $PM_{2.5}$ on cardiovascular and respiratory hospitalizations and ED visits during the summer of 2008 in northern California. We hypothesized that the effect of an increase in $PM_{2.5}$ during the wildfire period would be different than that in non-fire periods, and therefore included an interaction term indicating the time periods before, during, and after

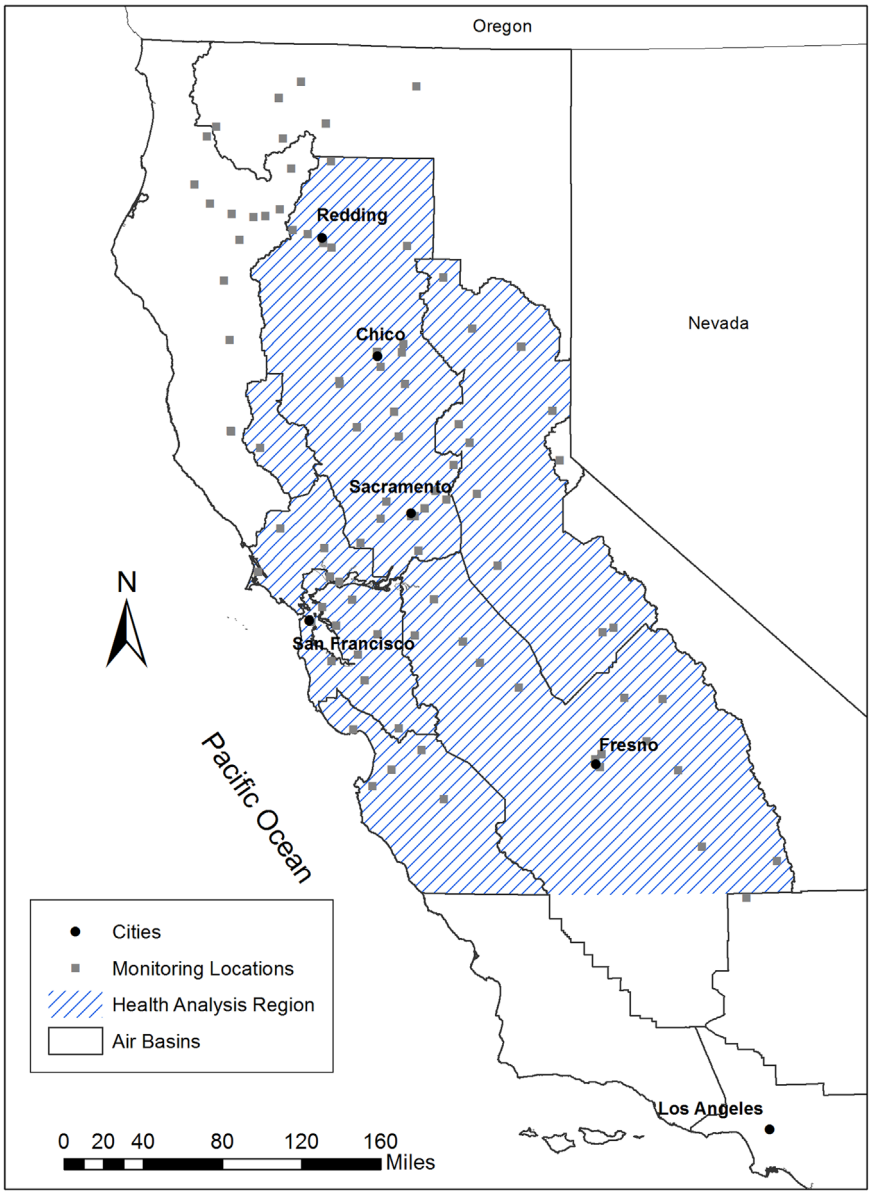

Fig. 1. Study region.

the fires. We used indicator variables to control for holiday and day of week effects. We assessed a variety of ways to control for temporal trend and found that a natural cubic spline on Julian date with 3 degrees of freedom (df) had the smallest Quasi Information Criterion for GEE models (QICu) (Hardin and Hilbe, 2003). To control for potential spatial confounding, we adjusted for smoking prevalence, median income as a measure of socio-economic status (SES), and percent of the population over 65 years of age (because elderly people may have increased susceptibility to wildfire health effects Reid et al., 2016) at the ZIP code level. Temperature and ozone are both spatiotemporal variables that could confound the $PM_{2.5}$-health relationship particularly during wildfires. We controlled for ozone and the heat index, a measure of apparent temperature that combines both temperature and RH, based on an algorithm used by the US National Weather Service that has been evaluated as the best of various apparent temperature metrics (Anderson et al., 2013). We found little difference between temperature and the heat index in our study domain (Pearson's r=0.995), but used heat index because other studies consider both temperature and RH to be confounders of the wildfire $PM_{2.5}$-health relationship (Delfino et al., 2009; Johnston et al., 2007). We also assumed *a priori* that the relationship between temperature and health would be linear, as the fires occurred only during the warm months. We used an exchangeable correlation structure with the sandwich estimator of the variance, which provides standard error estimates that are robust to misspecification of the covariance structure and also adjusts for any over-dispersion in the count data. We included the log of the size of the ZIP code population as an offset term.

Previous studies of the effects of $PM_{2.5}$ from wildfires on health have used various lags, mostly same-day, one-day, or two-day moving average. We initially investigated lags up to 28 days but did not find sustained effects (data not shown). We therefore applied a moving average of the two days prior to the date of hospital admission based on minimizing the QICu values. For the main analysis, we chose not to include same-day $PM_{2.5}$ in the moving average as we did not have access to the time of day of the hospitalization or ED visit and did not want to include counts of health outcomes that could have occurred before the exposure; however, we did a sensitivity analysis with the same-day data included (a three-day moving average).

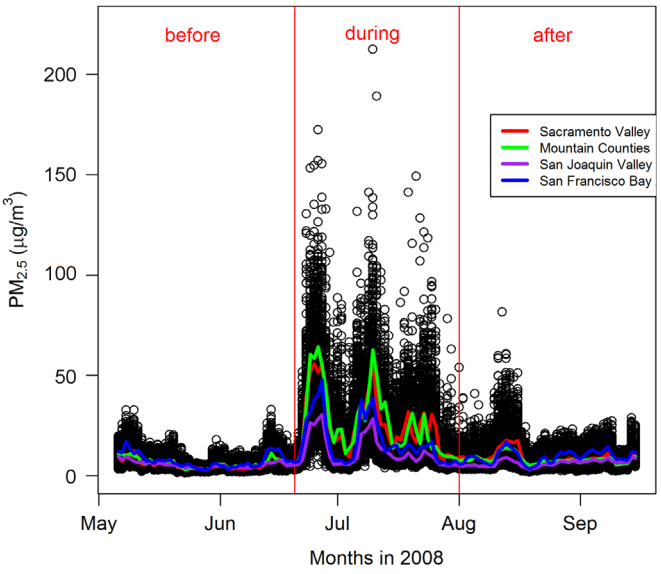

Fig. 2. $PM_{2.5}$ predictions by ZIP code for the before, during, and after fire periods with mean daily values for selected air basins. Open circles are predicted $PM_{2.5}$ values for each ZIP code for each day and colored lines represent the average value for all ZIP codes in that air basin.

Many epidemiological analyses of air pollution display their results as the effect for a change of 10 units or the interquartile range (IQR) in the pollutant of the exposures in the study. In an analysis over time, the effect estimates should represent the effect due to day-to-day differences in exposures, which may be much smaller (Snowden et al., 2015). We present our main findings as per 5 $\mu g/m^3$ $PM_{2.5}$ because our data had very few day-to-day changes of the IQR (6.7 $\mu g/m^3$) or higher, but 32% of ZIP code-days during the fire period and 11% overall experienced a day-to-day change of 5 $\mu g/m^3$or greater.

2.6. Exposure-response estimation

We evaluated the shape of the exposure-response function for wildfire smoke exposure by categorizing the continuously predicted 24-hour average $PM_{2.5}$ values to represent levels of the Air Quality Index (http://airnow.gov/index.cfm? action=aqibasics.aqi) updated for 2012. The categories we used were those considered to be good (< 12 $\mu g/m^3$), moderate (12.1–35.4 $\mu g/m^3$), unhealthy for sensitive groups (35.5–55.4 $\mu g/m^3$), and unhealthy, very unhealthy and hazardous ($> =55.5$ $\mu g/m^3$). We chose these cut-points because public health officials are given guidance on issuing advisories based on AQI levels as they get reported in the media (Lipsett et al., 2008). At this point, however, it is not clear that health effects increase with increasing values of $PM_{2.5}$ during wildfire episodes in the same way as they do for other forms of $PM_{2.5}$.

2.7. Identification of sensitive and vulnerable sub-populations

We evaluated the effect of $PM_{2.5}$ exposure during the fire period on hospitalizations and ED visits stratified by sex and age group (under 20 years old, 20–64 years old, and 65 and over). We also assessed effect modification by tertiles of ZIP code-level median income, percent of the population with less than a high school diploma, and percent of owner-occupied housing units. Counts of hospitalizations and ED visits by specific outcome and by these groups are presented in Supplemental Tables S1 and S2.

2.8. Sensitivity analyses

We performed the following sensitivity analyses: (1) using an exposure model that excluded variables that were highly correlated with those in the epidemiological models (i.e., Julian date, temperature, and RH), (2) including same day exposures along with the lag 1 and lag 2 exposures, thus a three-day moving average, (3) adjusting for temperature and relative humidity separately rather than combined in the heat index, and (4) including additional spatial covariates.

All statistical analyses were performed in R v. 2.15.3 Vienna, Austria (R Core Team, 2013). The Center for Protection of Human Subjects at the University of California, Berkeley deemed this work to be not human subjects research because the health data were administrative and not identifiable.

3. Results

3.1. Descriptive statistics

Daily $PM_{2.5}$ exposures were much higher during the fire period than in the periods before or after (Table 1 and Fig. 2). The heat index was much lower before the fires compared to during or after, likely due to seasonally lower temperatures in May and June compared to July, August, and September. Hospital and ED visits were highest in the before fire period.

Clear spatial differences in covariates existed by air basin (Table 2) demonstrating the need to control for purely spatial covariates that could confound the $PM_{2.5}$-health relationship. $PM_{2.5}$ from the wildfire was highest in the Sacramento Valley air basin, which was surrounded by fires, and the smoke funneled into the valley regardless of the wind direction.

3.2. Analyses by time period

During the fires, $PM_{2.5}$ was associated with both asthma hospitalizations (RR=1.07, 95% CI =(1.05, 1.10) per 5 $\mu g/m^3$ increase) and ED visits (RR=1.06, 95% CI =(1.05, 1.07) per 5 $\mu g/m^3$ increase); the association for ED visits was larger during than after the fires based on p-values of the interaction terms between $PM_{2.5}$ and time period (Tables 3 and 4). We also found a significant relationship between $PM_{2.5}$ and asthma ED visits and asthma hospitalizations before the wildfire period. ED visits for COPD were also associated with $PM_{2.5}$ during the fires (RR=1.02 (95% CI= (1.01, 1.04)) per 5 $\mu g/m^3$ increase); this was significantly different from effects found before but not after the fires. All-cause respiratory hospitalizations and ED visits were also associated with $PM_{2.5}$ during the fires, likely driven by asthma visits.

We found largely null results for cardiovascular disease outcomes related to $PM_{2.5}$ during the wildfires (Tables 3 and 4 and Supplement Figs. S2 and S3). Hypertension ED visits were associated with $PM_{2.5}$ after the fires but not before or during the fires. We also found an unanticipated protective association between $PM_{2.5}$ and congestive heart failure during the fire period. In both cases, rates were not significantly different from rates before the fire period.

3.3. Exposure-response analysis

For asthma hospitalizations and ED visits, the RR across exposure categories was not linear (Figs. 3 and 4). ED visits for COPD increased abruptly in the highest exposure category. We did not find differences in the shape of the exposure-response curves for the whole season compared to only the fire period (data not shown).

Table 1
Temporal descriptive statistics by time period.

	Full Season[a] N=102,311	Before Fires[a] N=33,583	During Fires N=32,802	After Fires N=35,926
Days (count)	131	43	42	46
Spatiotemporal Data – mean (SD)				
$PM_{2.5}$ (moving average of 24-hour average on lag days 1 and 2) ($\mu g/m^3$)	11.21 (10.78)	6.40 (3.17)	19.14 (15.48)	8.46 (3.99)
Temperature (°C)	21.29 (5.31)	18.77 (5.31)	22.48 (5.07)	22.57 (4.65)
RH (%)	52.75 (19.23)	52.50 (18.94)	54.67 (18.51)	51.24 (20.00)
Heat index moving average (°C)	21.78 (5.29)	18.83 (5.16)	23.25 (5.02)	23.18 (4.44)
Ozone (ppb)	54.40 (21.55)	47.63 (15.64)	59.69 (25.53)	55.92 (20.64)
Hospitalization Counts for whole area averaged by day: mean (min, max)				
All Respiratory	145.2 (92, 227)	174.7 (134, 227)	132.9 (92, 170)	128.9 (95, 165)
Asthma	26.6 (13, 49)	31.5 (17, 49)	23.4 (16, 38)	25.0 (13, 44)
COPD	34.6 (17, 52)	39.4 (24, 52)	32.2 (19, 46)	32.3 (13, 44)
Pneumonia	70.9 (33, 117)	86.8 (69, 117)	66.4 (33, 89)	60.1 (39, 83)
All CVD	390.9 (238, 509)	407.7 (254, 509)	387.7 (250, 480)	378.3 (238, 498)
Ischemic heart disease	125.1 (67, 176)	129.5 (71, 176)	124.4 (67, 169)	121.6 (67, 172)
Congestive heart failure	68.3 (39, 102)	73.9 (39, 102)	67.9 (45, 94)	63.6 (43, 85)
Dysrhythmias	63.3 (25, 98)	65.3 (39, 98)	63.0 (35, 86)	61.7 (25, 86)
Hypertension	19.6 (7, 33)	21.0 (9, 33)	18.4 (7, 27)	19.4 (7, 30)
Cerebrovascular disease	75.8 (45, 109)	78.2 (52, 109)	75.4 (51, 102)	78.8 (45, 100)
Emergency Department Visit Counts for whole area averaged by day: mean (min, max)				
All respiratory	752.0 (516, 1083)	883.6 (704, 1067)	665.6 (534, 852)	698.7 (516, 951)
Asthma	142.5 (83, 244)	169.1 (111, 244)	124.9 (85, 182)	133.7 (83, 211)
COPD	65.2 (41, 96)	71.3 (51, 94)	62.2 (41, 80)	61.3 (45, 78)
Pneumonia	113.6 (71, 176)	138.9 (112, 175)	103.9 (71, 134)	97.0 (73, 121)
All CVD	421.8 (342, 497)	430.6 (356, 493)	415.0 (364, 492)	414.5 (342, 490)
Ischemic heart disease	82.5 (60, 104)	84.3 (62, 103)	81.6 (62, 103)	81.1 (60, 101)
Congestive heart failure	77.2 (49, 110)	81.7 (48, 109)	76.5 (56, 101)	72.3 (55, 93)
Dysrhythmias	99.0 (63, 131)	99.6 (78, 128)	97.7 (63, 119)	98.2 (78, 128)
Hypertension	61.7 (39, 96)	61.7 (42, 81)	58.7 (39, 81)	63.5 (42, 96)
Cerebrovascular disease	78.5 (53, 103)	80.3 (63, 99)	78.0 (62, 95)	76.8 (53, 103)

N is the number of ZIP code-days, the unit of analysis.

[a] data as analyzed with two lagged days removed.

3.4. Sensitive and vulnerable populations

ED visits for asthma were significantly associated with $PM_{2.5}$ during the fire period for all age groups, with a nonsignificant increase in effect with increasing age (Supplement Table S3 and Fig. 5). Associations between $PM_{2.5}$ and hospitalizations for asthma were only present for ages 20–64 and ages ≥ 65 (Supplement Table S4). During the wildfires, individuals aged 20–64 had a significantly higher RR for COPD ED visits associated with $PM_{2.5}$ compared to those ≥ 65 (Fig. 5).

We also found some differences by sex. The association between $PM_{2.5}$ and ED visits for asthma and hypertension were significantly higher for females compared to males (Fig. 6 and Supplement Tables S5 and S6).

Asthma ED visits were significantly associated with $PM_{2.5}$ during the wildfires for all levels of SES (Supplement Fig. S4). The only consistent differential effects across tertiles of SES metrics were for ZIP-code level median income and respiratory ED visits. For asthma, COPD, pneumonia, and all-cause respiratory ED visits, there was a clear declining RR with increasing ZIP-code level median income (Fig. 7), but this was not observed for other respiratory outcomes (Supplement Tables S7 and S8).

3.5. Sensitivity analyses

Effect estimates were generally consistent across sensitivity analyses (Supplement Tables S9–S16) compared to our main model. Other formulations of the exposure model, inclusion of same-day hospitalizations and ED visits, and use of the heat index compared to temperature and relative humidity separately did not appreciably change the associations found with the main model.

4. Discussion

We found a significant relationship between $PM_{2.5}$ from wildfires and respiratory hospitalizations and ED visits. We used a sophisticated spatiotemporal exposure model with excellent performance in predicting $PM_{2.5}$ concentrations measured at air quality monitoring stations (out of sample CV-R^2 of 0.79), which may have enhanced our ability to detect subtle health effects. The most consistent effects were for asthma, with significant increases in hospitalizations and ED visits with a clear linear exposure-response relationship in categorical exposure models for ED visits. Regardless of level of SES for three measures of SES, there was a clear indication that increasing $PM_{2.5}$ levels during the wildfire events was associated with increased ED visits for asthma. We also observed some evidence that women were more susceptible than men to the effects of $PM_{2.5}$ during a wildfire on asthma. The finding of significant effects of asthma hospitalizations and ED visits before the fires as well as during and the lack of consistent interaction terms between time periods could imply that the effects of $PM_{2.5}$ on respiratory health outcomes are from $PM_{2.5}$ in general and not different by source of $PM_{2.5}$. This would imply that the risk associated with wildfires is due mainly to the heightened levels of exposure.

Our results are comparable to previous studies, particularly larger studies with spatiotemporal exposure assessments (Delfino et al., 2009; Henderson et al., 2011). The study most similar to ours found significant increases in respiratory hospitalizations associated with $PM_{2.5}$ during wildfire periods and also found few significant differences between the effects observed during the wildfires compared to after the wildfires (Delfino et al., 2009). Growing evidence suggests that wildfire smoke exposure is associated with exacerbation of COPD (Reid et al., 2016). Although we

Table 2
Spatial descriptive statistics by air basin.

	Full Area	Sacramento	San Joaquin	Mountain	Lake Tahoe	San Francisco	Lake County	North Coast	North Central Coast
Spatial Data – mean (SD)									
ZIP codes	781	173	168	108	8	248	13	17	46
Median income ($)	55,630 (26,510)	47,092 (18,956)	39,010 (13,359)	46,528 (17,057)	49,693 (21,897)	76,808 (28,923)	39,246 (10,525)	59,490 (27,546)	59,870 (18,446)
% less than HS education	12.88 (6.68)	13.80 (5.26)	10.65 (4.93)	17.59 (8.86)	6.71 (3.32)	11.87 (5.89)	20.95 (6.04)	13.12 (8.09)	10.69 (7.00)
% owner-occupied housing	63.90 (16.97)	67.74 (15.03)	60.18 (15.07)	74.76 (11.33)	64.58 (11.99)	59.57 (19.74)	72.22 (7.16)	63.06 (8.26)	58.7 (14.65)
Total Population	16,316 (18,150)	13,485 (16,471)	15,712 (16,997)	3716 (6121)	5825 (10,401)	26,254 (19,293)	4479 (4353)	2944 (4480)	15,293 (18,152)
Smoking prevalence	0.17 (0.04)	0.20 (0.03)	0.17 (0.04)	0.20 (0.02)	0.16 (0.03)	0.15 (0.04)	0.23 (0.01)	0.17 (0.03)	0.15 (0.03)
Spatiotemporal Data – mean (SD), [min, max]									
$PM_{2.5}$ moving average ($\mu g/m^3$)	11.21 (10.78) [1.62, 200.86]	13.16 (13.81) [1.82, 144.79]	7.92 (5.95) [2.91, 63.81]	13.29 (14.59) [1.62, 200.86]	15.18 (14.82) [1.96, 106.52]	11.27 (8.31) [2.39, 106.17]	14.72 (15.75) [2.42, 99.01]	13.54 (11.10) [2.22, 82.44]	8.29 (4.61) [2.08, 38.73]
Temperature (°C)	21.29 (5.31)	23.76 (4.23)	24.73 (4.09)	22.49 (5.14)	15.29 (4.59)	17.68 (4.22)	21.99 (3.76)	17.91 (4.11)	18.23 (4.45)
RH (%)	52.75 (19.23)	43.73 (12.77)	43.45 (11.12)	38.44 (14.48)	42.09 (16.37)	68.59 (16.3)	46.41 (14.43)	63.65 (17.28)	68.51 (17.24)
Heat index moving average (°C)	21.78 (5.29)	24.22 (4.2)	25.3 (4.18)	22.6 (5.13)	14.97 (4.58)	18.29 (4.17)	22.38 (3.7)	18.42 (4.06)	18.9 (4.51)
Ozone (ppb)	54.40 (21.55)	60.78 (18.16)	69.10 (16.96)	65.41 (17.50)	60.80 (15.64)	38.44 (17.33)	47.94 (16.95)	36.03 (11.76)	44.53 (15.60)

did not find significant increases in hospitalizations for COPD associated with $PM_{2.5}$ during the fire period, other studies have observed such effects (Delfino et al., 2009; Morgan et al., 2010). We did find significantly elevated ED visits for COPD during the fire period, which has been found in one other study (Rappold et al., 2011).

Our study, similar to many wildfire epidemiological studies, did not find evidence of significant effects of $PM_{2.5}$ from wildfires on hospitalizations or ED visits for cardiovascular disease (Hanigan et al., 2008; Henderson et al., 2011; Martin et al., 2013; Morgan et al., 2010). A few recent papers, however, have found significant effects for out-of-hospital cardiac arrests (Dennekamp et al., 2015; Haikerwal et al., 2015), hospitalizations for hypertension (Arbex et al., 2010), cardiovascular clinic visits (Lee et al., 2009), ED visits for congestive heart failure (Rappold et al., 2011), and hospitalizations for IHD (Johnston et al., 2007).

One important contribution to the literature on wildfire-health effects is our analysis of the exposure-response function for $PM_{2.5}$ during wildfires, as very few other studies have investigated this. Our findings are in line with Johnston et al. (2002) and Thelen et al. (2013) in finding increasingly significant respiratory health impacts with increasing concentrations of PM during wildfire periods.

Another important finding from our study relates to differential effects on certain populations. We found that women were more likely to be hospitalized and visit the ED for asthma and visit the ED for hypertension than men when exposed to high levels of $PM_{2.5}$ during wildfires. The only other study that investigated differential effects by sex on asthma hospitalization also found higher rates of asthma hospitalizations for women than men during the 2003 southern California wildfires (Delfino et al., 2009). To our knowledge, no other study has investigated differential gender effects of wildfire $PM_{2.5}$ on hypertension outcomes. It is not clear if women with asthma have greater biological susceptibility to wildfire smoke, if women are more likely to seek medical care for asthma exacerbations, if women are more likely to have uncontrolled asthma that would lead to heightened susceptibility, if it is some combination of these reasons, or due to chance.

We also found greater impact of wildfire air pollution on hospitalizations and ED visits for asthma among people aged 20–64 than those younger and older. Although this has been found in other previous wildfire studies in which middle-aged adults had higher odds of physician visits for asthma associated with PM_{10} during a wildfire than younger or older groups (Henderson et al., 2011), another study found the highest relative rates of asthma hospitalizations associated with $PM_{2.5}$ during a wildfire among people aged 65 and older (Delfino et al., 2009). In general, results on differential age effects have been inconsistent, and therefore this potential susceptibility factor should be further studied.

Interestingly, regardless of area-level SES - as measured by median income, high school graduation prevalence, prevalence of owner-occupied housing, or race - ED visits for asthma were significantly associated with $PM_{2.5}$ during the wildfire period (Supplement Fig. S4). Although we found consistent effects across tertiles of all measures of SES (Supplement Table S6), we did find some evidence of effect modification by ZIP code median income, with higher RRs with decreasing median income. Henderson et al. (2011) found no clear differences by neighborhood SES in associations between physician visits and various exposure metrics of wildfire smoke. Among counties affected by smoke from a peat fire in North Carolina, counties with lower SES had higher rates of ED visits for asthma and congestive heart failure compared to counties with higher SES (Rappold et al., 2012). Further research is needed to understand differential vulnerability to wildfire smoke exposure by SES.

This study made many comparisons to further understand population health effects and vulnerability to wildfire smoke, an

Table 3
Relative risks of hospitalization associated with $PM_{2.5}$ before, during, and after the 2008 northern California wildfires.

	RR for a 5 μg/m^3 change in $PM_{2.5}$			p-value comparing during to before	p-value comparing after to during
	Before Fires RR (95% CI)	During Fires RR (95% CI)	After Fires RR (95% CI)		
All respiratory	0.987 (0.946, 1.030)	1.018 (1.007, 1.029)	1.002 (0.959, 1.047)	0.165	0.473
Asthma	1.143 (1.042, 1.253)	1.073 (1.045, 1.101)	1.015 (0.928, 1.110)	0.185	0.227
COPD	0.890 (0.815, 0.971)	1.014 (0.992, 1.036)	1.048 (0.964, 1.140)	0.004	0.441
Pneumonia	0.966 (0.912, 1.024)	1.008 (0.991, 1.024)	1.001 (0.944, 1.062)	0.176	0.830
Cardiovascular disease	0.994 (0.968, 1.021)	0.995 (0.988, 1.002)	0.988 (0.965, 1.012)	0.969	0.577
Congestive disease	0.984 (0.925, 1.048)	0.987 (0.971, 1.003)	0.991 (0.935, 1.051)	0.943	0.878
Ischemic heart disease	1.003 (0.953, 1.055)	0.997 (0.984, 1.010)	0.986 (0.943, 1.030)	0.821	0.615
Dysrhythmias	1.013 (0.951, 1.079)	1.000 (0.984, 1.017)	1.022 (0.966, 1.082)	0.702	0.455
Cerebrovascular disease	0.980 (0.919, 1.046)	0.985 (0.970, 1.000)	0.974 (0.917, 1.033)	0.901	0.716
Hypertension	0.940 (0.840, 1.053)	1.002 (0.968, 1.037)	1.015 (0.905, 1.140)	0.290	0.825

All models are for the two-day moving average controlling for time trend, day of week, heat index, median income, percent of the population over 65, smoking prevalence, and ozone.

area that has not been sufficiently studied. We did not apply multiple testing corrections and thus p-values and confidence intervals should be interpreted accordingly. We do not claim that these results are definitive; rather they should be taken as part of a larger body of work on wildfire smoke exposure and health effects. This study used a novel spatiotemporal exposure model and the findings are generally in alignment with other studies of wildfire smoke exposure. Comparison of our results with those from future studies with spatiotemporal exposure modeling should provide better insight into the value of this approach.

We only investigated one air pollutant ($PM_{2.5}$) from these wildfires. Wildfires cause increases in other air pollutants of concern for public health. In ongoing research, we are modeling health effects of ozone from wildfires, which has been only minimally studied (Azevedo et al., 2011; Jalaludin et al., 2000). Our spatiotemporal modeling of ozone and $PM_{2.5}$ will allow assessment of effect modification and effect decomposition in meaningful ways.

Although exceptions exist (Szpiro et al., 2011), better exposure assessment can improve health effect estimation by decreasing exposure misclassification. Our exposure model predicted better during the fire period than in the before and after periods of the fires (Supplement Fig. S1). The health effects observed during the fires could be stronger than those before or after the fires because of better prediction by the exposure model, even though very few of the findings were significantly different between time periods in the main analysis. Indeed, the standard errors during the fire period are much smaller than those in the other two time periods. One of the likely reasons for better prediction during the fires is because satellite AOD, the strongest predictor in our exposure model, better predicts PM in the western US during high pollution events such as wildfires (Gupta et al., 2007).

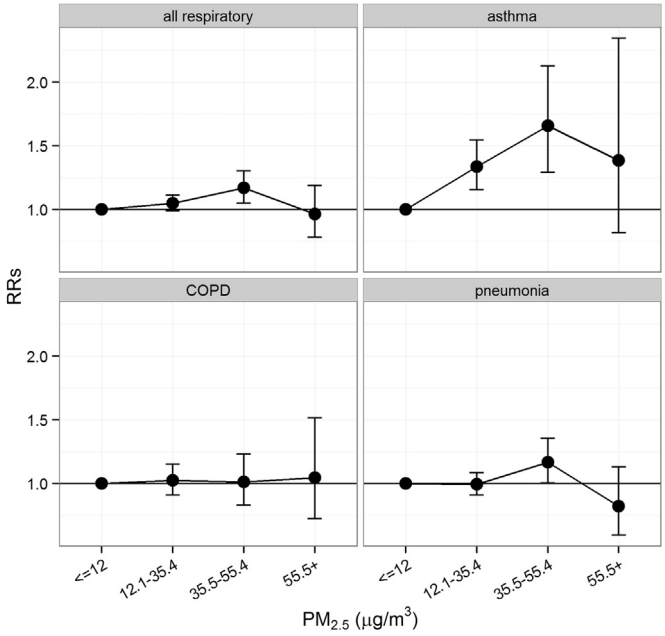

Fig. 3. Exposure-response for respiratory hospitalizations during the wildfire period.

Table 4
Relative risks of ED visits associated with $PM_{2.5}$ before, during, and after the 2008 northern California wildfires.

	RR for a 5 μg/m^3 change in $PM_{2.5}$			p-value comparing during to before	p-value comparing after to during
	Before Fires RR (95% CI)	During Fires RR (95% CI)	After Fires RR (95% CI)		
All respiratory	0.987 (0.968, 1.007)	1.015 (1.009, 1.020)	0.988 (0.967, 1.010)	0.008	0.019
Asthma	1.046 (1.000, 1.095)	1.056 (1.045, 1.068)	0.965 (0.925, 1.008)	0.682	0.000
COPD	0.959 (0.896, 1.027)	1.022 (1.006, 1.039)	1.043 (0.987, 1.102)	0.072	0.482
Pneumonia	0.939 (0.899, 0.980)	1.001 (0.989, 1.014)	0.99 (0.945, 1.036)	0.006	0.621
Cardiovascular Disease	1.003 (0.979, 1.028)	0.993 (0.987, 0.999)	1.000 (0.975, 1.026)	0.444	0.577
Congestive Heart Failure	0.980 (0.924, 1.040)	0.982 (0.967, 0.998)	1.033 (0.976, 1.092)	0.947	0.074
Ischemic Heart Disease	0.998 (0.946, 1.053)	0.997 (0.983, 1.011)	0.985 (0.931, 1.041)	0.965	0.654
Dysrhythmias	1.007 (0.961, 1.056)	0.995 (0.981, 1.010)	0.992 (0.939, 1.049)	0.649	0.916
Cerebrovascular Disease	0.988 (0.930, 1.051)	0.987 (0.973, 1.002)	0.979 (0.925, 1.037)	0.972	0.784
Hypertension	1.021 (0.953, 1.092)	1.012 (0.995, 1.029)	1.066 (1.008, 1.127)	0.818	0.080

All models are for the two-day moving average controlling for time trend, day of week, heat index, median income, percent of the population over 65, smoking prevalence, and ozone.

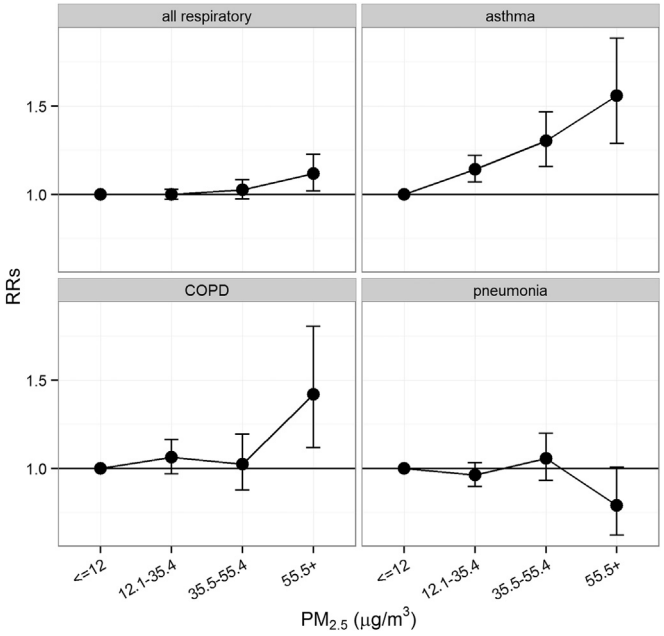

Fig. 4. Exposure-response for respiratory ED visits during wildfire period.

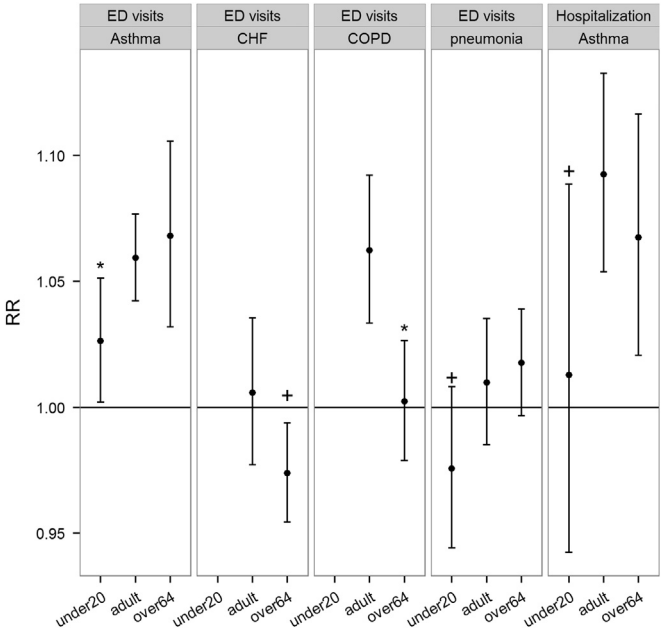

Fig. 5. Relative risks for a 5 μg/m^3 increase in $PM_{2.5}$ during the fire period by age group for respiratory hospitalizations. *denotes $p < 0.05$ level and + denotes $p < 0.10$ level for that age group compared to the adult (reference) age group during the fire period. No effect estimate is presented for the under20 age group for hospitalization for COPD because of so few observations of this health outcome in that group.

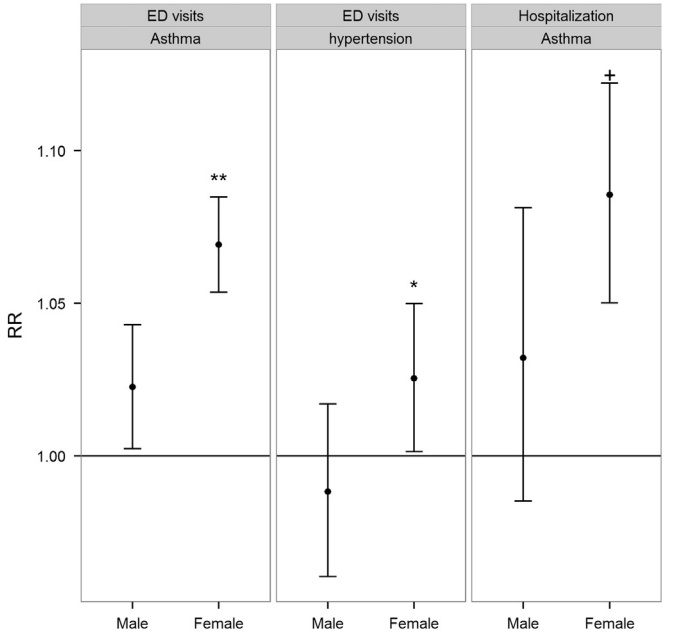

Fig. 6. Relative risks for a 5 μg/m^3 increase in $PM_{2.5}$ during the fire period by sex. **denotes $p < 0.01$, * denotes $p < 0.05$, and + denotes $p < 0.10$ for females compared to males during the fire period.

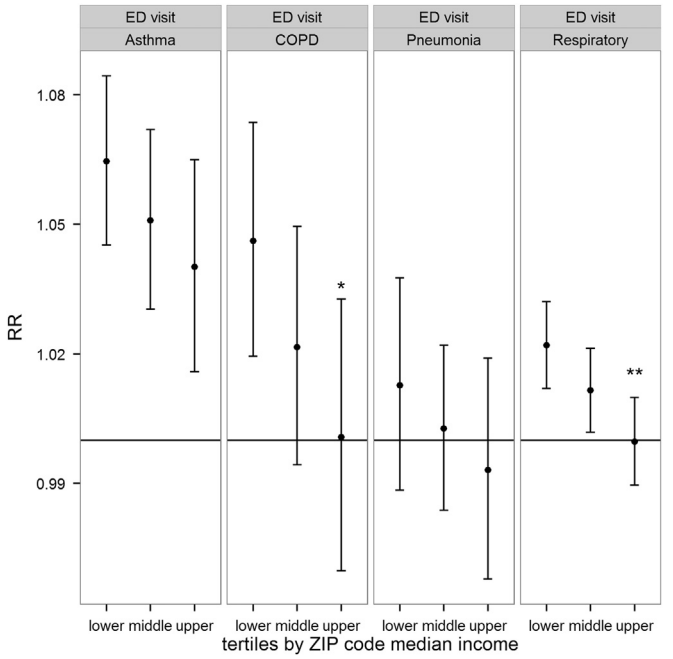

Fig. 7. Relative risks for a 5 μg/m^3 increase in $PM_{2.5}$ during the fire period by tertile of owner-occupied housing. **denotes $p < 0.01$, * denotes $p < 0.05$, and + denotes $p < 0.10$ compared to the lower tertile.

5. Conclusions

Using a novel spatiotemporal exposure model, we found that hospitalizations and ED visits for asthma were significantly associated with $PM_{2.5}$ during the 2008 northern California wildfires and that these effects increased with increasing $PM_{2.5}$ levels. Our results align with other studies that have used spatiotemporal exposure models (Delfino et al., 2009; Henderson et al., 2011) as well as more traditional exposure assignment methods (Johnston et al., 2007). We identified some differential effects by sex, age, and SES that should be further studied to determine if these groups are more vulnerable to wildfire smoke exposure. Our results add to the growing understanding of health risks associated with wildfire smoke, an exposure of increasing importance globally.

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The Center for Protection of Human Subjects at the University of California, Berkeley deemed this work to be not human subjects research because the health data were administrative and not identifiable.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.envres.2016.06.012.

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