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# Associations between respiratory health and ozone and fine particulate matter during a wildfire event



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#### ABSTRACT

Wildfires have been increasing in frequency in the western United States (US) with the 2017 and 2018 fire seasons experiencing some of the worst wildfires in terms of suppression costs and air pollution that the western US has seen. Although growing evidence suggests respiratory exacerbations from elevated fine particulate matter (PM<sub>2.5</sub>) during wildfires, significantly less is known about the impacts on human health of ozone (O<sub>3</sub>) that may also be increased due to wildfires. Using machine learning, we created daily surface concentration maps for PM<sub>2.5</sub> and O<sub>3</sub> during an intense wildfire in California in 2008. We then linked these daily exposures to counts of respiratory hospitalizations and emergency department visits at the ZIP code level. We calculated relative risks of respiratory health outcomes using Poisson generalized estimating equations models for each exposure in separate and mutually-adjusted models, additionally adjusted for pertinent covariates. During the active fire periods, PM2.5 was significantly associated with exacerbations of asthma and chronic obstructive pulmonary disease (COPD) and these effects remained after controlling for O3. Effect estimates of O3 during the fire period were non-significant for respiratory hospitalizations but were significant for ED visits for asthma (RR = 1.05 and 95% CI = (1.022, 1.078) for a 10 ppb increase in  $O_3$ ). In mutually-adjusted models, the significant findings for PM<sub>2.5</sub> remained whereas the associations with O<sub>3</sub> were confounded. Adjusted for O<sub>3</sub>, the RR for asthma ED visits associated with a  $10\,\mu\text{g/m}^3$  increase in PM<sub>2.5</sub> was 1.112 and 95% CI = (1.087, 1.138). The significant findings for PM<sub>2.5</sub> but not for O<sub>3</sub> in mutually-adjusted models is likely due to the fact that PM<sub>2.5</sub> levels during these fires exceeded the 24-hour National Ambient Air Quality Standard (NAAQS) of 35 µg/m<sup>3</sup> for 4976 ZIP-code days and reached levels up to 6.073 times the NAAQS, whereas our estimated O3 levels during the fire period only occasionally exceeded the NAAQS of 70 ppb with low exceedance levels. Future studies should continue to investigate the combined role of O3 and PM2.5 during wildfires to get a more comprehensive assessment of the cumulative burden on health from wildfire smoke.

## 1. Introduction

In the western United States and elsewhere, the frequency of large wildfires, their duration, and the length of the wildfire season have all increased (Westerling et al., 2006). Likely climate change scenarios suggest even higher fire risks in the future (Moritz et al., 2012). Given the observed impacts of wildfires on ambient fine particulate matter (PM $_{2.5}$ ) (McClure and Jaffe, 2018) and ozone (O $_3$ ) (Gong et al., 2017; Lu et al., 2016), an understanding of the population health impacts from exposure to these air pollutants from fires is a critical concern for

public health protection.

Some of the difficulty in studying the health impacts of air pollution from wildfires could be attributable to difficulties in exposure assessment to air pollution during the fires. The monitoring networks for  $O_3$  and  $PM_{2.5}$  are spatially sparse, and many PM monitors measure only every three or six days. The use of monitoring data alone for exposure assessment requires averaging across space, time, or both; thus exposure estimates fail to capture the true distribution of wildfire-attributable air pollution exposures that vary over small areas and short time periods. Spatial and temporal averaging of exposure leads to

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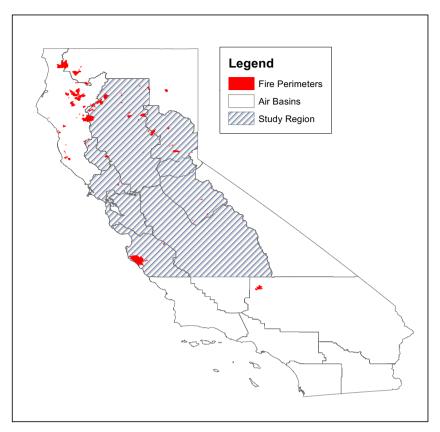


Fig. 1. Study area.

exposure misclassification which, depending on the error type, can bias effect estimates towards the null, increase variance, or both (Zeger et al., 2000).

Researchers are increasingly combining output from chemical transport models and satellite data to estimate air pollution exposures across space and time because each alone has its limitations. Chemical transport models are continuous in space and time, but are physical models rather than measurements and are dependent on various inputs, many of which, such as the fire emissions, contain large uncertainties. Satellite data are full-column measurements, not just at ground-level where people breathe and also often do not have enough time slices to understand the diurnal profile in air pollution levels. "Blended" models that combine information from these two sources, sometimes with other ancillary datasets, are increasingly being used in air pollution exposure estimates and in air pollution epidemiology in general (Bellinger et al., 2017). However, only a few studies of wildfire smoke and health have implemented such models (e.g., Reid et al., 2015; Gan et al., 2017).

A recent review documented increasing evidence of respiratory health effects from exposure to particulate matter (PM) from wildland fires, particularly for asthma, but to date only two studies have examined health effects of O<sub>3</sub> from wildland fires (Tham et al., 2009; Azevedo et al., 2011). Azevedo et al. (2011) found a significant correlation between O3 peaks during a wildfire and hospitalizations for cardiovascular disease but not respiratory disease. These analyses, however, were just correlations and did not adjust for any confounding variables. Tham et al. (2009) found no association between wildfireassociated O<sub>3</sub> and respiratory ED visits. This is despite documentation of high O<sub>3</sub> from many wildfires (Gong et al., 2017). Moreover, a large literature exists on the health effects from ambient O<sub>3</sub> (Chen et al., 2007), demonstrating that acute exposure to ambient O<sub>3</sub> is associated with adverse respiratory health effects (e.g. Stieb et al., 2012), increased mortality (Thurston and Ito, 2001), and some (although mixed) evidence of impacts on cardiovascular disease (Shah et al., 2013; Devlin

et al., 2012; Mirowsky et al., 2017).

Our study uses previously-developed statistical spatiotemporal model estimates of population exposure to  $PM_{2.5}$  (Reid et al., 2015) and  $O_3$  from the 2008 northern California wildfires. These models use the gradient boosting machine learning algorithm to combine data from various sources, such as output from chemical transport models (CTMs), satellite products of atmospheric composition such as aerosol optical depth (AOD), meteorological data, and other relevant data. Each model performed well for predicting out-of-sample air pollution concentrations. The  $PM_{2.5}$  model had a 10-fold  $CV-R^2$  of 0.78 (Reid et al., 2016), whereas the  $O_3$  model had a 10-fold  $CV-R^2$  of 0.73 [see Supplementary material for more information]. In this study, we used these spatiotemporal models to estimate population exposure to  $O_3$  and  $PM_{2.5}$  and evaluated how these exposures were associated with respiratory health during the 2008 northern California wildfires.

## 2. Methods

# 2.1. Study site

The 2008 northern California wildfires were ignited on the weekend of June 20–21, 2008 by over 6000 lightning strikes during a dry lightning storm which lit thousands of fires in 26 counties in California (CARB, 2008). Air pollution levels remained elevated throughout the period from June 20 until the end of July, at which point most of the fires were contained (CARB, 2008). The smoke covered large areas including the San Francisco Bay and the Sacramento metropolitan areas. Thus, large populations (estimated at over ten million people) had relatively long exposures, making this an important fire episode for analysis of public health effects.

We define the fire period as June 20–July 31, 2008; however, our analyses also include modeling of exposures and health effects from May 6 through September 26, 2008 to account for periods both before

and after the fires (May-September 2008).

The spatial limits of the study area include all ZIP codes (N = 753) within the following affected air basins in northern California: 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 shows the fire boundaries and study region for this study. Smoke from these fires traveled throughout the study region. We dropped some ZIP codes from our analysis because their populations were less than 100 people. This left 751 ZIP codes for our analysis. The census-estimated median population size of these ZIP codes was 9745 but ranged from 104 to 90879. The total population of the 751 ZIP codes in our study area is estimated at over 12.7 million. The average size of these ZIP codes was 157.8 km².

# 2.2. Hospitalization and emergency department (ED) visits

We obtained daily counts of hospitalizations and emergency department (ED) visits by residential ZIP code from the California Department of Public Health Environmental Health Investigations Branch for the state of California for respiratory outcomes that are hypothesized to be related to acute PM exposures. These include asthma (ICD-9 code 493), COPD (496,491-492), pneumonia (480-486), acute bronchitis (466), and acute respiratory infections (460-465). We also created a combined respiratory outcomes category which is a sum of all of these ICD-9 codes by day and by ZIP code.

# 2.3. Exposure assessment

We used previously-created spatiotemporal exposure models to estimate 24-hour average PM<sub>2.5</sub> and 8-hour maximum O<sub>3</sub> concentrations on each day for every ZIP code in the study area. Each pollutant model used observed monitoring data as the outcome variable and a variety of potential input variables that included satellite air pollution measures, chemical transport model output, meteorological data, land use information, and temporal and spatial variables as the predictor variables. To obtain accurate estimates of pollutant exposure, we evaluated a variety of machine learning algorithms, including models that accommodate nonlinear and interacting predictor effects. Because we didn't hypothesize that the relationships between the predictor variables and the pollutant observations would be linear, we tested a variety of machine learning algorithms that allow for non-linear relationships between input and output variables including: generalized additive models (GAM), generalized boosting model (GBM), k-nearest neighbor regression, lasso regression, linear models, multivariate adaptive regression splines (MARS), neural networks, random forest, and support vector machines with a radial basis kernel (SVM). We used 10-fold cross-validation (CV) to assess performance across each algorithm using the cross-validated root mean squared error (CV-RMSE) and the cross-validated  $R^2$  (CV- $R^2$ ). More specifics on the methodology can be found at Reid et al. (2015, 2016) and in the Supplementary material. The dependent variables were daily observations from monitors for each pollutant. For both pollutants, the best-fitting statistical algorithm was the GBM model. The  $PM_{2.5}$  model had a 10-fold  $CV-R^2$  of 0.78 (Reid et al., 2016), whereas the  $O_3$  model had a 10-fold CV- $R^2$  of 0.73 [see Supplemental Information].

We used these models to estimate exposures at each ZIP code centroid in the study area. This assumes that the relationships from the machine-learning algorithm between the exposure covariates and the monitored air pollution levels hold during times and locations where there were no measurements of levels of these air pollutants.

# 2.4. Covariate information

Because temperature and relative humidity are known to influence respiratory health and can often be high during wildfires, we took 24hour average temperature and relative humidity variables from the Rapid Update Cycle (RUC) model from the National Climatic Data Center (http://ruc.noaa.gov/). Studies of the health effects of extreme heat note that it is not only temperature but rather heat and relative humidity combined that affect how humans experience heat and how it impacts their health (Anderson et al., 2013). There are many ways to combine temperature and relative humidity, however, research has shown that most of these indices are highly correlated (Anderson et al., 2013). Therefore we chose to use the heat index equation used by the U.S. National weather service.

Our unit of analysis is the ZIP code-day, which means that we have an observation (counts of hospitalizations or ED visits for each specific health outcome linked to daily measures of  $PM_{2.5}$  and  $O_3$  and other covariates) for each ZIP code for each day. Therefore, we had to adjust for variables that could differ by ZIP code that could influence our health outcome and could potentially be statistically related to  $PM_{2.5}$  and  $O_3$  concentrations. 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). We also obtained estimates by ZIP code of total population, percent of the population over 65, % of the ZIP code aged 5 or younger, % non-White, and median household income from the 2000 US Census by ZIP-code tabulation areas (ZCTAs) which approximate ZIP codes to spatial census units.

## 2.5. Statistical analysis

The outcome variable in our analysis was the number of hospitalizations or ED visits for each respiratory health outcome listed above for each ZIP code-day during our study time period and spatial area. We used Poisson generalized estimating equations (GEE) to account for the repeated measures at the ZIP code level, with an exchangeable correlation structure and an offset of the log-transformed population of each ZIP code. To obtain standard errors which are robust to covariance matrix misspecification and overdispersion, we used the Huber-White sandwich estimator. We performed single pollutant models for both PM<sub>2.5</sub> and O<sub>3</sub>, as well as multipollutant models with both pollutants included. We adjusted for the following confounding variables: daily heat index, temporal trend modeled with a natural cubic spline with 3 degrees of freedom, estimated ZIP code-level smoking prevalence, % of the ZIP code aged 65 or older, % of the ZIP code aged 5 or younger, % non-White, ZIP code level median income, and day of week and holidays modeled as dummy variables. Total population at the ZIP code was used as an offset term. These were all done using the R package geepack (Højsgaard et al., 2006).

We did a preliminary analysis of lags of up to seven days (data not shown), and found that most of the health impacts for all health endpoints analyzed were due to exposures 1 to 2 days prior to the health event. We therefore modeled exposures to both  $PM_{2.5}$  and  $O_3$  as moving averages of the two days prior to the event. This is in line with findings from other studies of wildfire smoke exposure and health (Henderson et al., 2011; Delfino et al., 2009; Elliott et al., 2013).

We present our results as the relative risks associated with  $PM_{2.5}$  and  $O_3$  in periods before, during, and after the wildfires separately to assess if there are differential associations between each pollutant during the wildfires as compared to before or after the wildfires because our exposure estimates measure total  $PM_{2.5}$  and  $O_3$  rather than just the fraction of each pollutant that was due to the wildfires.

Because our models assumed linear associations between each pollutant and respiratory health outcomes, we also did a sensitivity analysis of each pollutant cut into five quintiles for the period during the fires. We then reran the single pollutant and mutually-adjusted models with these categorized pollutant concentrations.

All analyses were done using R version 3.5.1 (R Core Team, 2018). This research was approved by the University of Colorado Boulder Institutional Review Board, protocol #17-0417.

**Table 1**Total counts of daily ZIP code hospitalizations and emergency department visits summed across all 753 ZIP codes by before, during, and after fire periods.

	Hospitalizations		ED visits			
Health outcomes	Before	During	After	Before	During	After
All respiratory Asthma COPD Pneumonia Acute bronchitis Acute respiratory infections	7908 1438 1777 3924 260 264	5579 980 1352 2788 119	5922 1150 1485 2759 148 228	40,420 7736 3259 6351 3127 16,038	27,934 5244 2610 4359 1911 11,346	32,112 6145 2815 4453 2318 13,659

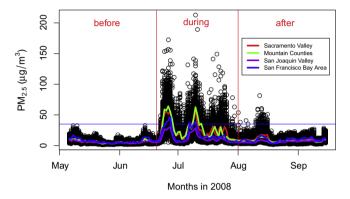


Fig. 2.  $PM_{2.5}$  levels by ZIP-code day during the study period with averages for some air basins.

#### 3. Results

There were more ED visits than hospitalizations during our study period. Table 1 shows the total number of hospitalizations and ED visits for the before fire, during fire, and after fire periods by respiratory health outcome.

Although the PM $_{2.5}$  levels were much higher during the fire period than before or after (Fig. 2), the  $O_3$  levels during the fire were not as high relative to normal background levels (Fig. 3). For only 848 ZIP-code days did the  $O_3$  levels exceed the EPA National Ambient Air Quality Standards (NAAQS) daily standard for  $O_3$  of 70 ppb, whereas the daily PM $_{2.5}$  NAAQS standard of  $35\,\mu\text{g/m}^3$  was exceeded on 4976 ZIP-code days.

# 3.1. Ozone only models

Associations between O<sub>3</sub> and respiratory hospitalizations were mostly null, except for a protective (negative) association between O<sub>3</sub>

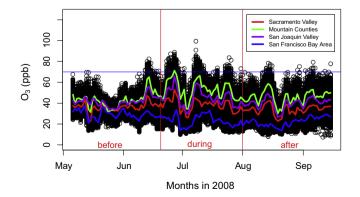


Fig. 3. Ozone levels by ZIP-code day during the study period with averages for some air basins.

and pneumonia hospitalizations during the fires (Table 2).

Ozone during the fires, not adjusted for  $PM_{2.5}$ , was significantly associated with ED visits for asthma (RR = 1.05 and 95% CI = (1.022, 1.078) for a 10 ppb increase in  $O_3$ ) and combined respiratory (RR = 1.013 and 95% CI = (1, 1.027) for a 10 ppb increase in  $O_3$ ). The association between  $O_3$  and combined respiratory remained after the fires, and we also observed a significant association between  $O_3$  and ED visits for acute respiratory infections after the fires, when  $O_3$  levels were still somewhat elevated (Table 3).

# 3.2. PM<sub>2.5</sub> only models

In models with only PM<sub>2.5</sub>, we found significant associations for hospitalizations for asthma (RR = 1.14 and 95% CI = (1.082, 1.201) for a  $10\,\mu\text{g/m}^3$  increase in PM<sub>2.5</sub>), COPD (RR = 1.042 and 95% CI = (1.002, 1.084) for a  $10\,\mu\text{g/m}^3$  increase in PM<sub>2.5</sub>) and combined respiratory outcomes (RR = 1.039 and 95% CI = (1.017, 1.061) for a  $10\,\mu\text{g/m}^3$  increase in PM<sub>2.5</sub>) but not for pneumonia or acute respiratory infections (Table 4). There was also a significant association between PM<sub>2.5</sub> and asthma hospitalizations before the fires, as we had found in our previous analysis which did not include these updated estimates of ozone (Reid et al., 2016).

 $PM_{2.5}$  not adjusted for  $O_3$  was significantly associated with asthma (RR = 1.115 and 95% CI = (1.09, 1.14) for a  $10\,\mu\text{g/m}^3$  increase in  $PM_{2.5}$ ) and COPD ED visits (RR = 1.054 and 95% CI = (1.023, 1.085) for a  $10\,\mu\text{g/m}^3$  increase in  $PM_{2.5}$ ), as well as combined respiratory outcomes during the fires but not before or after the fires. The relative risk of asthma ED visits associated with  $PM_{2.5}$  during the fires was also significantly different from those after the fires. We also found protective associations between  $PM_{2.5}$  and pneumonia and acute respiratory infections before the fires (Table 5).

## 3.3. Mutually-adjusted models

In mutually-adjusted models,  $O_3$  was not significantly associated with any respiratory hospitalization before, during, or after the fires. However, asthma and combined respiratory hospitalizations were associated with  $PM_{2.5}$  after adjustment for  $O_3$  during the fires and associations were suggestive for COPD and pneumonia. The significant association between  $PM_{2.5}$  and asthma hospitalizations before the fires remained after adjustment for  $O_3$ , and we found a protective association of  $PM_{2.5}$  and COPD before the fires (Fig. 4).

The significant relationships between  $O_3$  and ED visits during the fires appeared confounded by  $PM_{2.5}$  as those associations went away in mutually-adjusted models (Fig. 5). The correlation between the two-day moving average of  $O_3$  and  $PM_{2.5}$  was only 0.195, therefore this was likely not due to collinearity. The significant associations after the fire between  $O_3$  and combined respiratory and acute respiratory infections, however, remained.

 $PM_{2.5}$  during the wildfires was associated with increased risk of an ED visit for asthma (RR = 1.112 and 95% CI = (1.087, 1.138) for a  $10 \,\mu\text{g/m}^3$  increase in  $PM_{2.5}$ ) and COPD (RR = 1.05 and 95% CI = (1.019, 1.082) for a  $10 \,\mu\text{g/m}^3$  increase in  $PM_{2.5}$ ), as well as for combined respiratory visits (RR = 1.035 and 95% CI = (1.023, 1.046) for a  $10 \,\mu\text{g/m}^3$  increase in  $PM_{2.5}$ ). There was a suggestive association between  $PM_{2.5}$  during the wildfires and acute bronchitis ED visits. There were no significant associations between  $PM_{2.5}$  and respiratory ED visits before the fires when adjusted for  $O_3$ , but there were significant protective associations for pneumonia and acute respiratory infections before the fires (Fig. 5).

The results of our sensitivity analysis on quintiles of each pollutant are shown in the Supplementary material. For  $PM_{2.5}$ , we found increasing risk of asthma hospitalizations with increasing quintiles of exposure in both the  $PM_{2.5}$ -only model and the mutually adjusted model. Similarly, ED visits for combined respiratory, asthma, and COPD increased with increasing quintiles of  $PM_{2.5}$  exposure. We found no

**Table 2**Relative risks (and 95% Confidence Intervals) of hospitalization associated with a 10 ppb increase in ozone before, during, and after the 2008 northern California wildfires additionally adjusted for % non-white and % under age 5.

Health outcome	Before fires	During fires	After fires
Combined respiratory	0.992 (0.956, 1.030)	0.986 (0.961, 1.012)	0.989 (0.958, 1.021)
Asthma	0.939 (0.862, 1.023)	1.014 (0.956, 1.076)	0.995 (0.923, 1.072)
COPD	0.987 (0.919, 1.060)	1.018 (0.968, 1.071)	0.981 (0.929, 1.035)
Pneumonia	1.001 (0.953, 1.052)	0.964 (0.930, 0.999)	0.992 (0.950, 1.036)
Acute bronchitis	1.123 (0.921, 1.369)	0.976 (0.831, 1.147)	0.963 (0.795, 1.167)
Acute respiratory infections	1.008 (0.847, 1.201)	0.894 (0.778, 1.027)	0.956 (0.810, 1.129)

Table 3
Relative risks (and 95% CIs) of ED visits associated with a 10 ppb increase in ozone before, during, and after the 2008 northern California wildfires.

Health outcome	Before fires	During fires	After fires
Combined respiratory	0.986 (0.968, 1.005)	1.013 (1.000, 1.027)	1.046 (1.029, 1.063)
Asthma	0.971 (0.934, 1.008)	1.050 (1.022, 1.078)	1.030 (0.997, 1.064)
COPD	0.985 (0.930, 1.043)	1.031 (0.998, 1.065)	1.010 (0.964, 1.058)
Pneumonia	0.984 (0.946, 1.023)	0.992 (0.965, 1.019)	1.011 (0.975, 1.048)
Acute bronchitis	0.945 (0.878, 1.017)	1.008 (0.966, 1.052)	1.006 (0.950, 1.065)
Acute respiratory infections	0.994 (0.962, 1.026)	0.998 (0.976, 1.020)	1.083 (1.057, 1.109)

Table 4
Relative risks (and 95% CIs) of hospitalizations associated with a  $10 \,\mu\text{g/m}^3$  increase in  $PM_{2.5}$  before, during, and after the 2008 northern California wildfires.

Health outcome	Before fires	During fires	After fires
Combined respiratory	0.979 (0.901, 1.064)	1.039 (1.017, 1.061)	1.013 (0.929, 1.105)
Asthma	1.228 (1.025, 1.472)	1.140 (1.082, 1.201)	0.989 (0.821, 1.192)
COPD	0.831 (0.698, 0.989)	1.042 (1.002, 1.084)	1.123 (0.953, 1.323)
Pneumonia	0.940 (0.838, 1.055)	1.018 (0.986, 1.051)	1.020 (0.908, 1.145)
Acute bronchitis	1.061 (0.665, 1.692)	0.887 (0.746, 1.055)	1.098 (0.695, 1.734)
Acute respiratory infections	0.862 (0.545, 1.363)	0.922 (0.812, 1.047)	0.612 (0.389, 0.962)

significant associations between any quintile of  $O_3$  exposure and any respiratory hospitalizations in individual pollutant or mutually-adjusted models. However, the highest quintile of  $O_3$  was associated significantly with combined respiratory, asthma, and acute bronchitis ED visits. The associations for combined respiratory and acute bronchitis, but not asthma ED visits, held in models mutually adjusted for  $PM_{2.5}$ . We consider the results from the models for quintiles of air pollutant exposures to be preliminary as cutting the during fire period pollutant exposures into five equal groupings left many groups with small numbers of ZIP code-days.

# 4. Discussion

Our paper is one of the few epidemiological papers to investigate the role of  $O_3$  during wildfires on respiratory health. Using spatio-temporal models of  $O_3$  and  $PM_{2.5}$  based on machine-learning algorithms with good out-of-sample performance to estimate air pollution exposures during a wildfire event in 2008 in northern California, we found significant associations between  $O_3$  and respiratory ED visits, but not respiratory hospitalizations, when  $PM_{2.5}$  was not included in the model. When we adjusted  $O_3$  levels for  $PM_{2.5}$ , we found that the associations for  $O_3$  during the fires became null, however there were some

significant associations in the period after the fires for ED visits. Notably,  $O_3$  levels were still elevated during this time.

Associations between PM $_{2.5}$  exposures and respiratory ED visits and hospitalizations remained significant when mutually adjusted for  $O_3$  during the wildfire time period. The stronger association of PM $_{2.5}$  and respiratory health during the wildfires is likely due to the fact that the PM $_{2.5}$  levels during the fires (mean =  $19.202\,\mu\text{g/m}^3$ ) were much higher than before (mean =  $6.445\,\mu\text{g/m}^3$ ) or after the fires (mean =  $8.555\,\mu\text{g/m}^3$ ). The PM $_{2.5}$  levels during the fire were up to 6.073 times higher than the 24-hour NAAQS of  $35\,\mu\text{g/m}^3$ . However, the  $O_3$  levels during the fire (mean =  $36.093\,\text{ppb}$ ) were not much higher than before (mean =  $35.74\,\text{ppb}$ ) or after the fires (mean =  $32.331\,\text{ppb}$ ). Very few values were much higher than the 8-hour max NAAQS for  $O_3$  of  $70\,\text{ppb}$ .

The stronger associations between  $O_3$  and ED visits, not adjusted for PM<sub>2.5</sub>, compared to hospitalizations for asthma and COPD could be related to the severity of the health outcome. A health concern that results in a visit to the ED is normally less severe than one that results in a hospitalization. In our data, different outcomes were more likely to result in a visit to the ED or being hospitalized (Table 1). In our study population, we observed many more asthma ED visits than hospitalizations for asthma.

We had done a previous study investigating the association of PM<sub>2.5</sub>

Table 5 Relative risks (and 95% CIs) of ED visits associated with a  $10 \,\mu\text{g/m}^3$  increase in  $PM_{2.5}$  before, during, and after the 2008 northern California wildfires.

Health outcome	Before fires	During fires	After fires
Combined respiratory	0.990 (0.953, 1.029)	1.035 (1.024, 1.045)	0.985 (0.943, 1.029)
Asthma	1.072 (0.980, 1.172)	1.115 (1.090, 1.140)	0.921 (0.845, 1.005)
COPD	0.953 (0.833, 1.091)	1.054 (1.023, 1.085)	1.110 (0.999, 1.235)
Pneumonia	0.907 (0.834, 0.988)	1.010 (0.985, 1.035)	1.013 (0.925, 1.110)
Acute bronchitis	1.132 (0.980, 1.307)	1.035 (0.997, 1.074)	1.066 (0.937, 1.213)
Acute respiratory infections	0.928 (0.870, 0.990)	0.997 (0.980, 1.015)	0.952 (0.889, 1.020)

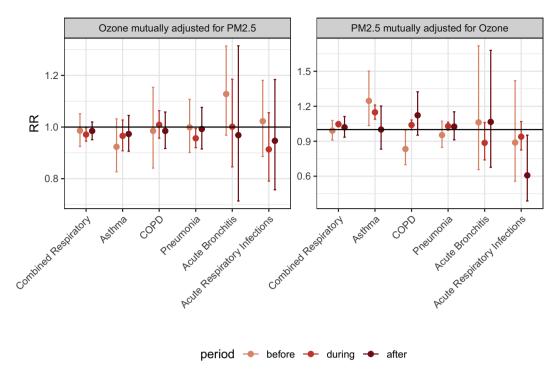


Fig. 4. Relative Risks (RRs) for hospitalizations in mutually-adjusted models for before, during, and after the wildfires.

during these fires with respiratory and cardiovascular hospitalizations and ED visits. In that study, we used an estimate of  $O_3$  from a chemical transport model rather than our blended spatiotemporal estimate of  $O_3$ . The correlation between these two estimates of daily 8-hour maximum  $O_3$  levels was 0.7, however our results for PM<sub>2.5</sub> adjusted for ozone are similar between the two papers with significant associations between PM<sub>2.5</sub> and asthma hospitalizations, and for asthma and COPD ED visits during the fires (Reid et al., 2016).

This study furthers our analysis of the health impacts of the 2008 northern California wildfires by investigating the role of  $O_3$  during the

fires on respiratory health but also expanding our respiratory health outcomes to include acute bronchitis and acute respiratory infections, which we had not analyzed in the previous study (Reid et al., 2016). We did find a suggestive association between  $PM_{2.5}$  during the fires and acute bronchitis ED visits that remained after adjustment for  $O_3$ .

To our knowledge, only two previous studies have attempted to assess the effects of  $O_3$  during wildfires on population health. Tham et al. (2009) studied the associations between  $PM_{10}$  or  $O_3$  with respiratory hospitalizations and ED visits during a bushfire in Victoria, Australia in 2003. They found no significant associations with  $O_3$  but

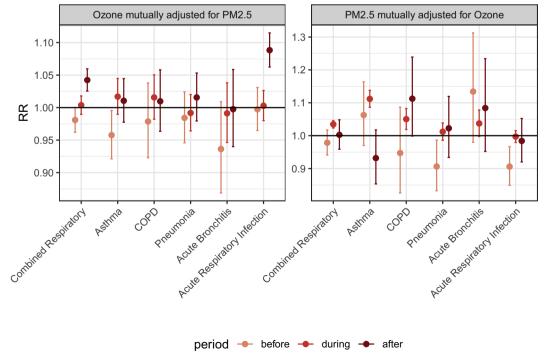


Fig. 5. Relative Risks (RRs) for ED visits in mutually-adjusted models for before, during, and after the wildfires.

did find a significant relationship between PM10 and daily respiratory ED visits and hospitalizations. The authors did not investigate a model with both pollutants. The primary goal of a study by Azevedo et al. (2011) was to understand the long-range transport of O<sub>3</sub> in rural and remote areas of Portugal and assess its impacts on health. Although this study reported associations between O3 related to wildfire events and cardiac diseases, they did not adjust for important confounders such as temperature, humidity, day of week effects, or seasonality. We therefore question the validity of these results until more robust epidemiological studies are done that control for these likely time-varying confounders. Despite the lack of evidence of a relationship between O<sub>3</sub> during wildfires and respiratory health from our study and the Tham study, we note that there is sufficient evidence that O<sub>3</sub> can be heightened during wildfires (Jaffe et al., 2008; Pfister et al., 2008), and it is possible that the lack of evidence shown here could be context specific. O3 is much more challenging to link to other covariates such as fire counts compared to, e.g. PM<sub>2.5</sub>. A large part of the PM<sub>2.5</sub> burden is directly emitted from the fires and the maximum impact occurs right over and nearby the source region. O<sub>3</sub>, however, is chemically produced from fire-emitted precursors and has a complex non-linear chemistry. As a result, the region of highest impact is shifted downwind of the source region with the distance dependent on transport times and chemical regime. We recommend that more studies of the health impacts of wildfire smoke begin to incorporate exposures to O<sub>3</sub> as well as PM to get a better perspective on the complete health impacts of wildfires.

While our study has strengths in its spatiotemporal air pollution exposure assessment and being one of the only papers to estimate the association between O3 during wildfires and respiratory health, it is not without its limitations. The results of our study are dependent on the models used to assess exposure to  $PM_{2.5}$  and  $O_3$ . Our exposure models had good out-of-sample performance yielding CV-R<sup>2</sup> of 0.78 and 0.73 for PM<sub>2.5</sub> and O<sub>3</sub>, respectively, but there is still the possibility of exposure measurement error. We are investigating the effect of exposure measurement error on the results obtained in this study in our on-going research. The key limitation of our study is that the results can only be interpreted for this fire in this location and this population. Differences in fuels burned, combustion conditions, meteorology, and topography can all influence the concentration and composition of the smoke to which a population is exposed, which can affect whether and to what extent health harms occur. For O<sub>3</sub> in particular, different fire dynamics, such as concentrations of precursor pollutants and how much sunlight can penetrate the smoke plume, can influence how much O<sub>3</sub> is formed, which would impact the results of an analysis of the effects of O<sub>3</sub> from wildfires on health. Additionally, the underlying susceptibility of a population, such as its age structure and prevalence of pre-existing respiratory diseases such as asthma and COPD, can influence how strongly smoke from wildfires will impact the health of that population.

# 5. Conclusions

To our knowledge, this is the first study to investigate the association between both  $PM_{2.5}$  and  $O_3$  during a wildfire, separately and in mutually-adjusted models, and respiratory health. Using spatiotemporal exposure models for  $PM_{2.5}$  and  $O_3$ , we found significant associations between each pollutant separately and respiratory health outcomes. However, these associations only remained for  $PM_{2.5}$  in mutually-adjusted models. We hypothesize that this is due to the fact that  $PM_{2.5}$  levels increased to levels up to 6 times higher than the EPA's  $PM_{2.5}$  daily NAAQS, whereas  $O_3$  levels during these fires increased only a bit above the EPA's daily 8-hour max  $O_3$  NAAQS value. Further research is needed to better understand the role of  $O_3$  on respiratory health during other wildfires and in other contexts.

## Competing interests

The authors have no competing interests to declare.

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#### Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2019.04.033.

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