Associations of wildfire smoke PM$_{2.5}$ exposure with cardiorespiratory events in Colorado 2011–2014

Jennifer D. Stowell$^{a,1}$, Guannan Geng$^{a,1}$, Eri Saikawa$^{a,b}$, Howard H. Chang$^c$, Joshua Fu$^d$, Cheng-En Yang$^d$, Qingzhao Zhu$^d$, Yang Liu$^{a,*}$, Matthew J. Strickland$^{e,**}$

$^a$Department of Environmental Health, Rollins School of Public Health, Emory University, 1518 Clifton Road NE, Atlanta, GA 30322, USA

$^b$Department of Environmental Sciences, Emory University, 201 Dowman Drive, Mailstop 1131-002-1AA, Atlanta, GA 30322, USA

$^c$Department of Biostatistics and Bioinformatics, Rollins School of Public Health, Emory University, 1518 Clifton Road NE, Atlanta, GA 30322, USA

$^d$Department of Civil and Environmental Engineering, University of Tennessee Knoxville, 851 Neyland Drive, Knoxville, TN 37996, USA

$^e$School of Community Health Sciences, University of Nevada Reno, 1664 N. Virginia Street, Reno, NV 89557, USA

Abstract

Background: Substantial increases in wildfire activity have been recorded in recent decades. Wildfires influence the chemical composition and concentration of particulate matter ≤2.5 μm in aerodynamic diameter (PM$_{2.5}$). However, relatively few epidemiologic studies focus on the health impacts of wildfire smoke PM$_{2.5}$ compared with the number of studies focusing on total PM$_{2.5}$ exposure.

Objectives: We estimated the associations between cardiorespiratory acute events and exposure to smoke PM$_{2.5}$ in Colorado using a novel exposure model to separate smoke PM$_{2.5}$ from background ambient PM$_{2.5}$ levels.

Methods: We obtained emergency department visits and hospitalizations for acute cardiorespiratory outcomes from Colorado for May-August 2011–2014, geocoded to a 4 km geographic grid. Combining ground measurements, chemical transport models, and remote sensing data, we estimated smoke PM$_{2.5}$ and non-smoke PM$_{2.5}$ on a 1 km spatial grid and aggregated to match the resolution of the health data. Time-stratified, case-crossover models were used to estimate the associations.

This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

*Correspondence to: Y. Liu, Department of Environmental Health, Rollins School of Public Health, Emory University, 1518 Clifton Road NE, Atlanta, GA, USA. yang.liu@emory.edu. **Correspondence to: M.J. Strickland, School of Community Health Sciences, University of Nevada Reno, 1644 N Virginia Street, Reno, NV, USA. mstrickland@unr.edu.

Declaration of competing interest

The authors declare they have no actual or potential competing financial interests.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2019.105151.
fit using conditional logistic regression to estimate associations between fire smoke PM$_{2.5}$ and nonsmoke PM$_{2.5}$ for overall and age-stratified outcomes using 2-day averaging windows for cardiovascular disease and 3-day windows for respiratory disease.

**Results:** Per 1 μg/m$^3$ increase in fire smoke PM$_{2.5}$, statistically significant associations were observed for asthma (OR = 1.081 (1.058, 1.105)) and combined respiratory disease (OR = 1.021 (1.012, 1.031)). No significant relationships were evident for cardiovascular diseases and smoke PM$_{2.5}$. Associations with non-smoke PM$_{2.5}$ were null for all outcomes. Positive age-specific associations related to smoke PM$_{2.5}$ were observed for asthma and combined respiratory disease in children, and for asthma, bronchitis, COPD, and combined respiratory disease in adults. No significant associations were found in older adults.

**Discussion:** This is the first multi-year, high-resolution epidemiologic study to incorporate statistical and chemical transport modeling methods to estimate PM$_{2.5}$ exposure due to wildfires. Our results allow for a more precise assessment of the population health impact of wildfire-related PM$_{2.5}$ exposure in a changing climate.

**Keywords**

Particulate matter; Wildfire; Air quality; Epidemiology; Climate change; Respiratory; Cardiovascular

1. **Introduction**

Climate change, defined as the long-term change in global and regional weather patterns, has been extensively documented since the mid-to-late 20th century (Boudes, 2011; Incropera, 2016; The Environmental Pollution Panel, 1965; United States Environmental Protection Agency, 2017b). Despite politically charged debates regarding the cause of the change, it is clear that climate change and its resulting extreme weather events could severely impact the health and well-being of populations across the globe (Berrang-Ford et al., 2015; Kjellstrom et al., 2016; Thornton et al., 2014; Wu et al., 2016). One area that reflects the synergistic impact of climate change and human activity is the occurrence of wildfires. Notably, the Western US has seen consistent and rapid increases in wildfire activity since the 1980s. This increase has been characterized by rises in the frequency, severity, size, and total burned area associated with wildfires (Liu et al., 2013; Westerling, 2016; Westerling et al., 2006). Fire effects are often seen at great distances from the events due to large smoke plumes, sometimes extending across multiple counties or states. States in the Rocky Mountain region continue to exhibit climatic factors conducive to fire activity—including high temperatures, low soil moisture, decreased rainfall, and increased solar radiation (Crockett and Westerling, 2018; Dawson et al., 2014; Griffin and Anchukaitis, 2014; Leung and Gustafson, 2005; Penrod et al., 2014). Conditions may become more suitable to large wildfires over time due to climate change (Keeley and Syphard, 2016; Keywood et al., 2013; Stavros et al., 2014). Consequently, wildfires place significant burdens on the human, economic, and environmental systems in areas surrounding and downwind from the burn zone. This is of particular concern given the impact that wildfire events can have on regional air quality and, subsequently, human health (Liu et al., 2015; Reid, Jerrett et al., 2016).
Wildfire smoke can produce significantly higher exposures to harmful compounds than are normally found in non-fire urban settings (Alves et al., 2011; Kim et al., 2018; Na and Cocker, 2008). Fine particulate matter (PM$_{2.5}$, airborne particles < 2.5 μm in aerodynamic diameter) is of particular concern due to its ability to travel deep into the human respiratory system and enter the blood stream (Dockery and Pope, 1994; Hong et al., 2017; Kim et al., 2015; Liu et al., 2015; Park and Wexler, 2008; Reid, Jerrett et al., 2016; United States Environmental Protection Agency, 2017a). Smoke particles differ in both size and composition from particles found in typical ambient PM from non-wildfire sources. It has been shown that organic compounds, such as methanol or formaldehyde, make up a significantly higher proportion of smoke PM$_{2.5}$ when compared with ambient PM (X.X. Liu, 2017; Na and Cocker, 2008). These distinctions could have differing effects on human health outcomes and may vary by fuel source. This has been shown in both in vivo and in vitro studies using human cells and mice (Kim et al., 2019; Shin et al., 2017; Xu et al., 2019). While much is left to be understood about the toxicological differences, current literature has begun to elucidate potential differences between smoke and ambient PM sources. It is, therefore, important to differentiate between smoke and non-smoke PM$_{2.5}$ when assessing the health impact of wildfires.

While numerous epidemiological studies have established the associations between ambient PM$_{2.5}$ and human health (Brook et al., 2010; Di et al., 2017; Pope and Dockery, 2006), relatively few studies have focused specifically on wildfire smoke (Rappold et al., 2017). For example, Reid et al. published a study showing a significant result for asthma during fire events (previous 2-day moving average) for a 5 μg/m$^3$ change in PM$_{2.5}$ concentration (Reid, Jerrett et al., 2016). While Reid et al. included satellite and chemical transport data, they were limited to the use of fire day and fire distance parameters to account for smoke PM instead of directly estimating smoke PM concentrations. Additionally, many studies are restricted to the use of ambient urban air pollution measurements, coupled with fire day indicators, to represent fire-related exposures. In addition, current guidelines for public health response to wildfire events rely heavily on changes of ambient total PM measurements due to a lack of information in wildfire-specific air quality (Lipsett et al., 2016). A few studies have distinguished among sources on larger scales (Hutchinson et al., 2018; J.C. Liu, 2017; Thelen et al., 2013). For example, Liu et al. derived metrics of smoke waves for distinguishing fire activity and evaluated the health impacts of smoke PM$_{2.5}$ (J.C. Liu, 2017). Their chemical transport model simulations, however, were on a spatial grid of 0.5 × 0.67 degrees, which may be too coarse to capture finer-scale spatial gradients of exposure, see Supplemental Fig. 1.

Though there is consistent evidence for associations between wildfire events and disease, questions remain regarding the relationship between wildfire smoke PM$_{2.5}$ and both respiratory and cardiovascular outcomes given the difficulty in estimating smoke PM$_{2.5}$ exposure. Developing robust methods for understanding this complex relationship is vital to understand the potential future impacts of climate and wildfire events on human health. Building upon previous studies, the goal of our study is to estimate the associations for multiple respiratory and cardiovascular acute health events in relation to wildfire smoke PM$_{2.5}$ in Colorado during the fire seasons of 2011–2014 using novel, high-resolution methods to separate wildfire smoke PM$_{2.5}$ from background ambient PM$_{2.5}$. 
2. Methods

2.1. Health data

We obtained individual-level health data on daily hospitalizations and emergency
department (ED) visits at all public and private hospitals for the fire seasons (May-August)
of 2011–2014 from the Colorado Department of Public Health and Environment.
Information included in the patient records are dates of admission, residential address, age,
sex, payer information and International Classification of Diseases version 9 (ICD9) codes
for primary and secondary diagnoses. Patients admitted to the hospital through the ED were
only counted once, and those with elective hospitalizations were excluded from analysis.
We analyzed multiple endpoints for primary cardiovascular and respiratory diagnoses.
Respiratory outcomes include asthma (ICD9: 493), bronchitis (ICD9: 490), chronic
obstructive pulmonary disease (ICD9: 491, 492, and 496), upper respiratory infection
(ICD9: 460–465 and 466.0), and combined respiratory disease (ICD9: 460–465, 466.0,
466.1, 466.11, 466.19, 480–486, 487, 488, 490, 491, 492, 496, and 493). Cardiovascular
outcomes include ischemic heart disease (ICD9: 410–414), acute myocardial infarction
(ICD9: 410), congestive heart failure (ICD9: 428), dysrhythmia (ICD9: 427), peripheral/
cerebrovascular disease (ICD9: 433–437, 440, 443, 444, 451–453), and combined
cardiovascular disease (ICD9: 410–414. 427, 428, 433–437, 440, 443, 444, 451–453). Due
to inadequate numbers, events in children were not analyzed for COPD or any
cardiovascular outcomes.

2.2. PM$_{2.5}$ and meteorological data

We sought to separate smoke PM$_{2.5}$ from ambient sources. To accomplish this, daily mean
PM$_{2.5}$ concentrations were adopted and improved from our previous study by adding new
data (Geng et al., 2018). Briefly, mean concentrations were estimated using a two-model
approach to combine information from high-resolution satellite AOD derived from the
Multi-angle Implementation of Atmospheric Correction (MAIAC) algorithm, model
simulations from the Community Multiscale Air Quality Modeling System (CMAQ), and
ground measurements obtained from the U.S. Environmental Protection Agency (USEPA)
for fire seasons in the state of Colorado (April-September 2011–2014). The first model (i.e.
AOD model) utilized random forest modeling to incorporate MAIAC AOD, smoke mask,
meteorological fields and land-use variables. The second model (i.e. CMAQ model) utilized
statistical downscaling to calibrate the CMAQ PM$_{2.5}$ simulations. Additional exposure
modeling specifics can be found in Supplemental 2 and Supplemental Fig. 2. The output
exposure data have full coverage in space and time and are able to capture the large fire
events at a resolution of 1 km × 1 km (CV $R^2 = 0.81$ and RMSE = 1.85 μg/m$^3$). Compared
to Geng et al. (2018), major improvements include new observation data from the National
Park Service to capture PM$_{2.5}$ enhancement near wildfires, allowing for a better
representation of high values found during fire events (Supplemental 2 and Supplemental
Fig. 2) (Benedict et al., 2017; Martin et al., 2013). Additionally, a random forest approach
was utilized instead of the original statistical downscaler for the AOD model. This improved
the $R^2$ of the AOD model from 0.65 to 0.92 and the gap-filled $R^2$ from 0.66 to 0.81 (Geng et
PM$_{2.5}$ exposure values were then aggregated to a 4 km × 4 km grid to match the resolution of the health data.

Fire count data were obtained using the MODIS fire count product to specify fire days for each grid cell (NASA, 2018). Wildfire and prescribed fire emissions were obtained from the US EPA emissions inventory for the study period. To calculate the wildfire smoke PM$_{2.5}$ fractions, we used two CMAQ model scenarios—with and without smoke and dust particles. The differences between these scenarios were then divided by the total PM$_{2.5}$ scenario to calculate the smoke PM$_{2.5}$ fractions. The smoke PM$_{2.5}$ fractions were then multiplied by the total satellite-based PM$_{2.5}$ exposure to get the smoke PM$_{2.5}$ concentrations.

### 2.3. Epidemiological modeling methods

We estimated associations between short-term changes in air quality and ED visits and hospital admissions using a case-crossover study design (Maclure, 1991). Each individual’s event day (i.e., date of ED visit or hospitalization) was matched with up to four non-event days, with matching based on grid location, day of week, and calendar month (Levy et al., 2001). Exposure and meteorology were assigned to each event day and corresponding non-event days based on the 4 km × 4 km grid cell in which the patient’s address is located. The 4 km grid was chosen a priori through collective agreement between the researchers and the Colorado State Health Department. This resolution was deemed the finest resolution we could use while still conserving confidentiality. We then used conditional logistic regression to estimate the associations between ED visits and hospitalizations for each outcome and exposure to non-smoke PM$_{2.5}$ and smoke PM$_{2.5}$. The final models for respiratory outcomes are shown in model specification 1 & 2 below:

\[
\logit P(Y) = \beta(\text{total}_{3\text{day}} \, \text{PM}_{2.5}) + \beta(\text{temp}_{3\text{day}}) + ns(\text{doy})
\]  

\[
\logit P(Y) = \beta(\text{smoke}_{3\text{day}} \, \text{PM}_{2.5}) + \beta(\text{nonsmoke}_{3\text{day}} \, \text{PM}_{2.5}) + \beta(\text{temp}_{3\text{day}}) + ns(\text{doy})
\]

where $\text{total}_{3\text{day}} \, \text{PM}_{2.5}$ represents the 3-day moving average for total PM$_{2.5}$ (i.e., smoke + non-smoke), $\text{temp}_{3\text{day}}$ is the 3-day moving average temperature, $ns(\text{doy})$ is a spline for day of year (two internal nodes per year), $\text{smoke}_{3\text{day}} \, \text{PM}_{2.5}$ represents the three-day moving average smoke PM$_{2.5}$; and $\text{nonsmoke}_{3\text{day}} \, \text{PM}_{2.5}$ denotes three-day moving average PM$_{2.5}$ not related to wildfires. Cardiovascular outcome models were conducted using the same models shown in model specifications 1 and 2, but with 2-day averaging windows. Exposure windows of 3-day average PM for respiratory outcomes and 2-day average PM for cardiovascular outcomes were decided a priori based on published studies and consensus information found in the latest Integrated Science Assessment from the USEPA (Analitis et al., 2012; Delfino et al., 2009; Kunzli et al., 2006; J.C. Liu, 2017; Rappold et al., 2011; Reid, Jerrett et al., 2016; Strickland et al., 2010; USEPA, 2019). Sensitivity analyses were conducted using lag 0, lag 0–1 and seven-day exposure windows for respiratory outcomes and lag 0 and three-day exposure windows for cardiovascular outcomes.
Other potential confounders were assessed (relative humidity, boundary layer height, heat index, wind speed). However, these parameters did not influence the results and were omitted in the final model. Analyses to examine the presence of potential effect modification were completed using sex and age-stratification. Age-stratified categories included children (0–18 years), adults (19–64 years), and older adult (65+ years). We conducted all analyses in R 3.4.3 (2017) and SAS© 9.4.

3. Results

3.1. Exposure modeling and smoke contribution to PM$_{2.5}$ levels

A time series plot for modeled statewide daily mean PM$_{2.5}$ concentrations is shown in Fig. 1. Modeled total PM$_{2.5}$ values ranged from close to 0 to 47.48 μg/m$^3$, with an overall mean value of 4.67 μg/m$^3$. The exposure model was also used to separate smoke PM$_{2.5}$ from nonsmoke PM$_{2.5}$. This separation is based on the CMAQ fraction, with total PM$_{2.5}$ equal to the sum of non-smoke PM$_{2.5}$ and smoke PM$_{2.5}$. Ratios of smoke PM$_{2.5}$ to total PM$_{2.5}$ ranged from 0 to 99.56% (mean = 0.006%), with smoke PM$_{2.5}$ levels ranging from 0 to 37.34 μg/m$^3$. The statewide daily mean smoke vs. total PM$_{2.5}$ ratio is also shown for the entire study period (See Fig. 2). As shown, concentrations varied year-to-year and between stations. This is likely due to the spatial variability of wildfires and varied smoke plume behavior due to factors such as prevailing wind speed and direction. To illustrate PM$_{2.5}$ concentrations and ratios attributable to fire, Fig. 3 shows the domain-wide average total PM$_{2.5}$ on fire days (smoke PM$_{2.5}$ > 1%) compared with the domain-wide average ratio of smoke PM$_{2.5}$. For the entire study period, total PM$_{2.5}$ averaged 7.87 μg/m$^3$ with average fire PM$_{2.5}$ ratios at 28%. Fig. 4 shows locations on a fire day near two major fires that occurred during our study period. As shown in Fig. 4A, high levels of smoke PM$_{2.5}$ can be seen despite more moderate total PM$_{2.5}$ concentrations. Fig. 4B depicts a fire day with much higher total PM$_{2.5}$ concentrations and the subsequent contributions of smoke PM$_{2.5}$. Additional analysis showed relatively little correlation between smoke PM$_{2.5}$ and non-smoke PM$_{2.5}$ (Pearson correlation coefficient $r = 0.11$, $p < 0.0001$). The peaks of highest smoke PM$_{2.5}$ ratios tended to correspond with active fire days. Fig. 5 illustrates the modeled total PM$_{2.5}$ and smoke PM$_{2.5}$ ratio for June 22, 2013, a peak fire day during the West Fork Fire Complex. As depicted, when compared to satellite imaging, the modeled smoke PM$_{2.5}$ appears to capture the apparent visible smoke plume adequately.

3.2. Epidemiological modeling

After excluding duplicate events and events with non-geocoded addresses, 44,262 of 490,368 (9%) of cases were excluded from the analysis. A total of 446,106 ED visit and hospitalization events were analyzed from the Colorado Department of Public Health and Environment. Of those included, there were 204,823 male and 241,283 female cases. The lowest case count occurred in 2011 (n = 102,318), with the highest number of cases in 2014 (n = 129,477). While many reasons could exist, the large increase seen in 2014 could be explained by changes in health seeking behavior due to wider Medicaid coverage resulting from the implementation of the Affordable Care Act (Singer et al., 2019). Other summary statistics on age groups and events per year are found in Table 1.
Using conditional logistic regression models, we estimated the odds ratio for exposure to smoke PM$_{2.5}$ and individual health outcomes. As shown in Fig. 6 and Supplemental Table 1, we observed significant positive associations between 1 μg/m$^3$ increases in 3-day moving average fire exposures and both asthma (OR 1.081, 95% CI (1.058, 1.105)) and combined respiratory disease (OR 1.021, 95% CI (1.012, 1.031)) in a model that adjusted for PM$_{2.5}$ from other sources. There were no significant positive associations linked to cardiovascular outcomes and 2-day smoke PM$_{2.5}$ exposures (see Fig. 7 and Supplemental Table 2). However, some inverse associations were shown to be protective for cardiovascular outcomes. This could possibly be due to random error, or it may be that individuals with pre-existing cardiovascular disease stay indoors on days with fire activity.

The models were also run using total PM$_{2.5}$ for both cardiovascular and respiratory outcomes. Overall, the majority of the respiratory odds ratios for 3-day average total PM$_{2.5}$ were either null or trending to positive (Supplemental Table 3). The odds ratios for ischemic heart disease, acute myocardial infarction, and dysrhythmia also suggest a trend toward a positive association (see Supplemental Table 4). The cardiovascular results for total PM$_{2.5}$ included significant negative results for congestive heart failure, peripheral/cerebrovascular disease, and cardiovascular disease.

We conducted sensitivity analyses for additional exposure windows. Using lag 0 for both respiratory and cardiovascular outcomes, similar results were seen with smoke PM$_{2.5}$ exposure, with notable differences in overall upper respiratory infection (OR 1.015, 95% CI (1.005, 1.026)) and upper respiratory infection in children (OR 1.018, 95% CI (1.004, 1.003), see Supplemental Figs. 3 and 4). Using lag 0–1 for all respiratory outcomes, the results were again similar to the initial analysis with changes for overall and child-only upper respiratory infections; see Supplemental Fig. 5. Using a 7-day averaging window for respiratory outcomes, asthma was the only outcome to have a significant positive association with smoke PM$_{2.5}$ exposure (OR 1.081, 95% CI (1.051, 1.112), see Supplemental Table 5). The associations for asthma, upper respiratory infection, bronchitis, and combined respiratory disease trended positive but not significant for 7-day averaged total PM$_{2.5}$ exposure (see Supplemental Table 6). A 3-day averaging window used for cardiovascular outcomes also yielded either null or negative results (Supplemental Tables 7 and 8).

### 3.3. Stratified analysis

To investigate potential effect modification of the relationship between exposures and respiratory outcomes, we conducted stratified analyses based on sex and age. While most sex-stratified total PM$_{2.5}$ results were null, an association was seen in females for bronchitis (OR 1.007, 95% CI (1.001, 1.013), see Supplemental Table 9), however, no significant results were observed for cardiovascular outcomes and both 2-day total and smoke PM$_{2.5}$ (Supplemental Tables 10 and 11). Associations for both female and male asthma cases and 3-day average smoke PM$_{2.5}$ were significant, with higher odds shown in female cases (OR 1.096, 95% CI (1.064, 1.128)) than in male cases (OR 1.063, 95% CI (1.029, 1.098)). Female bronchitis cases (OR 1.054, 95% CI (1.010, 1.101)) and female total respiratory cases (OR 1.027, 95% CI (1.015, 1.040)) were also positively associated with smoke PM$_{2.5}$. 

_Environ Int. Author manuscript; available in PMC 2021 May 28._
Additional sex-stratified, 3-day average smoke PM$_{2.5}$ results can be found in Supplemental Table 12.

Additionally, some outcomes exhibited differences when stratified on age. After age-stratification, there were no patterns found linking respiratory outcomes and total PM$_{2.5}$ with any specific age group (Supplemental Table 13). Regarding smoke PM$_{2.5}$, Fig. 6 also depicts the ORs and associated confidence intervals for each of the respiratory outcomes by age group. In children ages 0 to 18 years, significant positive associations were seen for asthma (OR 1.075, 95% CI (1.035, 1.116)). Adults aged 19 to 64 years of age exhibited positive associations for asthma (OR 1.091, 95% CI (1.060, 1.122)), bronchitis (OR 1.044, 95% CI (1.005, 1.085)), COPD (OR 1.056, 95% CI (1.015, 1.100)), and combined respiratory disease (OR 1.030, 95% CI (1.017, 1.044)) (see also Supplemental Table 14). For individuals 65 and older, there were no significant positive associations seen for respiratory outcomes. We found no positive associations for age-stratified total or smoke PM$_{2.5}$ and any of the cardiovascular outcomes (See Fig. 7 and Supplemental Tables 15 and 16). Additional results for stratification analyses using a 7-day averaging window for respiratory outcomes and a 3-day averaging window for cardiovascular outcomes can be found in Supplemental Tables 17–24. Of note, associations for both childhood and adult asthma, adult COPD, and adult combined respiratory disease events were positively associated with 7-day average smoke PM$_{2.5}$ (see Supplemental Table 17).

4. Discussion

In this study, we estimated associations between various health outcomes and acute exposure to non-smoke PM$_{2.5}$ and smoke PM$_{2.5}$ in the state of Colorado over a four-year period (2011–2014). The design of this study is centered on smoke PM$_{2.5}$ contributions to health outcomes. This work builds on our previous work by improving exposure data metrics and expanding from a 1-month pilot study (Alman et al., 2016). The exposure data considers both spatial and temporal variability by including the use of satellite data to enhance the exposure estimates on an improved spatial scale of 4 km × 4 km. Another unique aspect of our exposure assessment is that we were able to separate smoke PM$_{2.5}$ from non-smoke sources and estimate risks attributable to wildfire smoke distinct from those due to PM$_{2.5}$ exposures from other sources.

As we hypothesized, many of the respiratory disease outcomes increased during periods of wildfire activity. For respiratory outcomes, we estimated an increase (OR = 1.036 (95% CI: 1.022, 1.050%)) in ED/hospitalizations per 1 µg/m$^3$ increase in fire smoke PM$_{2.5}$ exposure. The magnitude of the association was largest for asthma (OR = 1.081 (95% CI: 1.058, 1.105)). Additionally, we observed heterogeneity in the association estimates when stratifying by age group. Positive associations were observed for asthma events, where ED/hospitalizations increased significantly in children (OR = 1.075 (95% CI: 1.035, 1.116)) and in adults (OR = 1.091 (95% CI: 1.060, 1.122)) whereas the association estimate was lower in magnitude and was less precise for older adults (OR = 1.009 (95% CI: 0.920, 1.106)). Similarly, an increase was seen for combined respiratory diseases with increases in ED/hospitalizations and adults (OR = 1.030 (95% CI: 1.017, 1.044)). Specifically, in the adult group, increases were also shown for both bronchitis (OR = 1.044 (95% CI: 1.005, 1.085))
and COPD (OR = 1.056 (95% CI: 1.015, 1.100)). As opposed to other studies, there was no association shown for respiratory diseases when stratified for the older adult age group.

Unlike respiratory outcomes, we did not see a strong link between smoke PM$_{2.5}$ and cardiovascular outcomes. Results for combined cardiovascular disease yielded null results (OR = 0.998 (95% CI: 0.984, 1.011)). Similar results were shown for both the adult and older adult age groups. This is not wholly surprising given differing results in current literature regarding the links between cardiovascular outcomes and wildfire events. There are fewer examples of cardiovascular associations with wildfire smoke exposure compared to respiratory outcomes. Additionally, associations with cardiovascular outcomes tended to be substantially lower in magnitude than for the respiratory outcomes. These differences are consistent with published studies on both types of outcomes (Cascio, 2018; Deflorio-Barker et al., 2019; Dennekamp et al., 2011; Dennekamp et al., 2015; Johnston et al., 2014; Liu et al., 2015; Reid, Brauer et al., 2016; Wettstein et al., 2018). For example, in Deflorio-Barker et al. (2019), most cardiovascular outcomes were not significant with fire day PM$_{2.5}$ using lag0-2. They also found similar results for smoke day all-cause cardiovascular outcomes were very similar to non-smoke days (OR 1.06 for smoke days vs OR 1.07 for non-smoke days (Deflorio-Barker et al., 2019)).

Our high-resolution epidemiological study furthers the current knowledge in the field by incorporating random forest modeling methods combining information from MAIAC AOD, CMAQ simulations, and ground measurements to elucidate the portion of PM2.5 present in the air due to wildfire smoke. Previous work has been done to enhance the spatial coverage and resolution of total PM2.5 estimates during wildfire events (Reid et al., 2019). While most work compared smoke and non-smoke days using various fire indicators, our study particularly focuses on the separation of smoke PM2.5 from other sources. In most work, researchers compared smoke and non-smoke days using a variety of methods different from our study (Reid et al., 2019; Reid, Jerrett et al., 2016). For example, satellite measurements are increasingly used to augment the spatially sparse ground monitoring for PM. However, this remains a relatively new approach to capturing the smoke PM concentrations. A study by Liu et al. looked at the entire Western US at the county-level using combined satellite and ground data (J.C. Liu, 2017). They defined a fire indicator variable, or “smoke wave,” which includes periods of at least two days of high pollution from wildfire smoke. Using this method, Liu et al. found associations between wildfire smoke exposure and various respiratory illnesses, but no associations with cardiovascular outcomes. Reid et al. (2015) used a machine learning approach to integrate multiple data sources including smoke indicators such as the distance to the nearest fire cluster and a smoke intensity calculation. The use of more advanced methods for predicting PM$_{2.5}$ exposure enhanced the exposure estimations, however, the PM$_{2.5}$ concentrations were not separated into smoke and non-smoke concentrations (Reid et al., 2015).

Other work has utilized methods combining wildfire emissions and smoke plume modeling. For example, Hutchinson et al. examined similar epidemiological questions using exposure data derived from a model that combined the Wildland Fire Emissions Information System and the Hybrid Single-Particle Lagrangian Integrated Trajectories (Hutchinson et al., 2018). Their study found increases in respiratory events with null cardiovascular results. However,
the methods denoted fire-specific emissions due to fire location and progression from modeled progression maps and may not capture exposures as well as the use of chemical transport models. Ultimately, while our results carry similar interpretations to both studies, subtle dissimilarities may be seen as we utilize different air quality evaluation products and higher-resolution meteorological and epidemiological data to better-define the local exposures for each event.

The asthma association found in our study is substantially larger than those shown in previous publications. In addition to Reid, Brauer et al. (2016), other studies found significant associations between smoke PM and health outcomes. Delfino et al. reported significant associations of OR = 1.043 between asthma and 2-day moving average smoke exposure for 10 μg/m3 increase in total PM2.5 concentration (Delfino et al., 2009). In a more recent study, Reid et al. also found a significant association for asthma and previous 2-day moving average smoke exposure, with an OR of 1.050 during fire events for a 10 μg/m3 increase in PM2.5 (Reid et al., 2019). Factoring in the domain-wide average smoke PM2.5 ratio for the study period (~28% for days with > 1% smoke PM), our result per 1 μg/m3 roughly translates to 1.08 per 4 μg/m3 of total PM2.5. This converted result is more aligned with previously reported values, and the larger effect estimate is likely due to improved exposure assessment. It is also important to remember that our methods are unlike the majority of previous literature. Namely, the general approach in previous studies is to model smoke exposure using smoke day indicators. Our approach differed in that we sought to isolate the actual concentration of PM2.5 directly from smoke. We originally hypothesized that there may be a difference in toxicity of smoke PM2.5 compared to non-smoke PM2.5.

When compared with other literature, our findings suggest that smoke PM2.5 may actually be more damaging to human health. Aside from asthma outcomes, the majority of the health associations in this study fall in line with those found in previous literature. For example, Deflorio-Barker et al. (2019) also demonstrated stronger associations with respiratory outcomes than those with cardiovascular disease; with asthma exhibiting the largest OR of 1.06 (Deflorio-Barker et al., 2019).

While we did not investigate physiological mechanisms, these results may be explained by the toxicity of smoke PM2.5. Since different chemical compositions of PM2.5 may affect the body differently, it has been suggested that toxicological differences may play a role in how wildfire smoke PM affects the human anatomy and physiology. Multiple toxicological studies have shown differences in the composition and effects of wildfire smoke compared to ambient air (Franzi et al., 2011; Kim et al., 2018; Wegesser et al., 2010; Wegesser et al., 2009; Wong et al., 2011). It has been shown that the small particles found in wildfire smoke may be responsible for stimulation of mechanisms that lead to increased oxidative stress at the cellular level. Wegesser et al. (2009) observed significant changes in macrophage and neutrophil counts in mouse lung samples exposed to wildfire smoke PM compared to ambient air. An additional study by the same group, expanded on these findings to show that substances such as polycyclic aromatic hydrocarbons (PAH) can be present in much higher concentrations in smoke versus levels detected in ambient air (Wegesser et al., 2010). Franzi et al., (2011) looked specifically at the inflammatory responses due to wildfire smoke PM exposure. PM from wildfire smoke exhibited approximately five times more toxicity to lung macrophages than nonsmoke exposure. This study also showed significant changes in
reactive oxygen species and subsequent oxidative stress, leading to higher cell degeneration and potential apoptosis. Similarly, Kim et al. (2018) found significant increases in mouse lung neutrophils after exposure and that levels of lung toxicity were significantly associated with fuel type (Kim et al., 2018).

Despite the strengths of our study, some limitations remain. While we sought to enhance the exposure estimates for individual cases, some exposure misclassification is still possible given the assumption that the location of a person’s address is a good representation of their short-term exposures to smoke PM. An additional limitation exists due to the use of modeled exposure data. However, as stated previously and despite this uncertainty, the model accurately captures the temporal and spatial trends of PM$_{2.5}$ measured by ground monitors and, thus give an accurate representation of overall trends. Additionally, several health events were left out of the analysis due to issues with address geocoding or non-Colorado residency. However, the exclusions were relatively small with only 9% of cases not used in the final analyses. Additionally, our analyses lacked the ability to differentiate chemical compositions of PM$_{2.5}$. Thus, we cannot link toxicological effects to our exposure metrics. Finally, the selection of averaging window size, though based on current literature, may also introduce error into the analysis.

Notwithstanding these limitations, our methods lend insight into important challenges that remain in the wildfire smoke exposure and health effects literature. The use of higher resolution enhanced exposure data provides a new approach to assigning exposure to individual events. Using multiple data products, our method aids in distinguishing wildfire smoke PM$_{2.5}$ from background PM$_{2.5}$. Unlike ground monitors that provide spatially sparse measurements, the exposure model used here provides daily concentrations for each 4 km × 4 km grid cell in our epidemiological study.

5. Conclusions

Supported by high-resolution PM$_{2.5}$ exposure estimates, we found significant associations between wildfire smoke and acute respiratory outcomes in Colorado, despite an absence of association with total PM$_{2.5}$ concentrations. Our findings point to potential toxic differences between smoke and non-smoke PM$_{2.5}$ exposure; suggesting that PM$_{2.5}$ from wildfire smoke could pose a significant threat to public health. This is especially true given the expected climate change-related impacts on wildfire incidence. It is, therefore, important to derive more accurate concentration-response relationships specific to wildfire smoke in order to develop a better understanding of future potential health risks based on increased wildfire activity. Taken together, the current analysis can inform public health agencies and healthcare systems regarding the potential future burden of wildfire smoke PM$_{2.5}$ exposure within the context of climate change. This information may be a key element in evaluating and enhancing current preparations aimed at wildfire-event response readiness.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.
Acknowledgments

The authors would like to thank Kirk Bol and the Colorado Department of Public Health and Environment for providing the health data used in this analysis.

Funding sources

The work of J. Stowell, G. Geng, Y. Liu, and H. Chang was partially supported by the NASA Applied Sciences Program (grant NNX16AQ28G, PI: Y. Liu). This publication was developed under assistance agreement no. 83586901 awarded by the U.S. Environmental Protection Agency (PI: Y. Liu). It has not been formally reviewed by EPA. The views expressed in this document are solely those of the authors and do not necessarily reflect those of the Agency. EPA does not endorse any products or commercial services mentioned in this publication.

References

Alman BL, Pfister G, Hao H, Stowell J, Hu X, Liu Y, Strickland MJ, 2016. The association of wildfire smoke with respiratory and cardiovascular emergency department visits in Colorado in 2012: a case crossover study. Environ. Health 15 (1), 1–9. 10.1186/s12940-016-0146-8. [PubMed: 26739281]

Alves CA, Vicente A, Monteiro C, Goncalves C, Evtyugina M, Pio C, 2011. Emission of trace gases and organic components in smoke particles from a wildfire in a mixed-evergreen forest in Portugal. Sci. Total Environ. 409 (8), 1466–1475. 10.1016/j.scitotenv.2010.12.025. [PubMed: 21277615]

Analisis A, Georgiadis I, Katsouyanni K, 2012. Forest fires are associated with elevated mortality in a dense urban setting. Occup. Environ. Med. 69 (3), 158–162. 10.1136/oem.2010.064238. [PubMed: 21849344].

Benedict KB, Prenni AJ, Carrico CM, Sullivan AP, Schichtel BA, Collett JL, 2017. Enhanced concentrations of reactive nitrogen species in wildfire smoke. Atmos. Environ. 148, 8–15. 10.1016/j.atmosenv.2016.10.030.

Berrang-Ford L, Pearce T, Ford JD, 2015. Systematic review approaches for climate change adaptation research. Reg. Environ. Chang. 15 (5), 755–769. 10.1007/s10113-014-0708-7.

Boudes P, 2011. United Nations Conference on the Human Environment. In. pp. 410–413.

Brook RD, Rajagopalan S, Pope CA, Brook JR, Bhatnagar A, Diez-Roux AV, … Council Nutr Phys Activity, M, 2010. Particulate matter air pollution and cardiovascular disease an update to the scientific statement from the American Heart Association. Circulation 121 (21), 2331–2378. 10.1161/CIR.0b013e3181dbece1. [PubMed: 20458016]

Cascio WE, 2018. Wildland fire smoke and human health. Sci. Total Environ. 624, 586–595. 10.1016/j.scitotenv.2017.12.086. [PubMed: 29272827]

Crockett JL, Westerling AL, 2018. Greater temperature and precipitation extremes intensify western us droughts, wildfire severity, and Sierra Nevada tree mortality. J. Clim. 31 (1), 341–354. 10.1175/jcli-d-17-0254.1.

Dawson JP, Bloomer BJ, Winner DA, Weaver CP, 2014. Understanding the meteorological drivers of us particulate matter concentrations in a changing climate. Bull. Am. Meteorol. Soc. 95 (4), 520–532. 10.1175/bams-d-12-00181.1.

Deflorio-Barker S, Crooks J, Reyes J, Rappold AG, 2019. Cardiopulmonary effects of fine particulate matter exposure among older adults, during wildfire and non-wildfire periods, in the United States 2008–2010. Environ. Health Perspect. 127 (3). 10.1289/ehp3860.

Delfino RJ, Brummel S, Wu J, Stern H, Ostro B, Lipsett M, … Gillen DL, 2009. The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003. Occup. Environ. Med. 66 (3), 189–197. 10.1136/oem.2008.041376. [PubMed: 19017694]

Dennekamp M, Erbas B, Sim M, Glass D, Keyword M, Abramson M, Tonkin A, 2011. Air pollution from bushfires and out-of-hospital cardiac arrests in Melbourne, Australia. Epidemiology 22 (1). 10.1097/01.ede.0000391824.65847.98. (S53–S53).

Dennekamp M, Straney LD, Erbas B, Abramson MJ, Keyword M, Smith K, … Tonkin AM, 2015. Forest fire smoke exposures and out-of-hospital cardiac arrests in Melbourne, Australia: a case-crossover study. Environ. Health Perspect. 123 (10), 959–964. 10.1289/ehp.1408436. [PubMed: 25794411]
Di Q, Wang Y, Zanobetti A, Wang Y, Koutrakis P, Choirat C, … Schwartz JD, 2017. Air pollution and mortality in the medicare population. N. Engl. J. Med. 376 (26), 2513–2522. 10.1056/NEJMoa1702747. [PubMed: 28657878]

Dockery DW, Pope CA, 1994. Acute respiratory effects of particulate air-pollution. Annu. Rev. Public Health 15, 107–132. 10.1146/annurev.pu.15.050194.000543. [PubMed: 8054077]

Franzi LM, Bratt JM, Williams KM, Last JA, 2011. Why is particulate matter produced by wildfires toxic to lung macrophages? Toxicol. Appl. Pharmacol. 257 (2), 182–188. 10.1016/j.taap.2011.09.003. [PubMed: 21945489]

Geng G, Murray NL, Tong D, Fu JS, Hu X, Lee P, … Liu Y, 2018. Satellite-based daily pm2.5 estimates during fire seasons in Colorado. Journal of Geophysical Research: Atmospheres 0 (ja). 10.1029/2018JD028573.

Griffin D, Anchukaitis KJ, 2014. How unusual is the 2012–2014 California drought? Geophys. Res. Lett. 41 (24), 9017–9023. 10.1002/2014gl062433.

Hong KY, King GH, Saraswat A, Henderson SB, 2017. Seasonal ambient particulate matter and population health outcomes among communities impacted by road dust in British Columbia, Canada. J. Air Waste Manage. Assoc. 67 (9), 986–999. 10.1080/10962247.2017.1315348.

Hutchinson JA, Vargo J, Milet M, French NH, Billmire M, Johnson J, Hoshiko S, 2018. The San Diego 2007 wildfires and medi-cal emergency department presentations, inpatient hospitalizations, and outpatient visits: an observational study of smoke exposure periods and a bidirectional case-crossover analysis. PLoS Med. 15 (7). 10.1371/journal.pmed.1002601.

Incropera FP, 2016. Climate Change: A Wicked Problem. Cambridge University Press, New York City, New York.

Johnston FH, Purdie S, Jalaludin B, Martin KL, Henderson SB, Morgan GG, 2014. Air pollution events from forest fires and emergency department attendances in Sydney, Australia 1996–2007: a case-crossover analysis. Environ. Health 13. 10.1186/1476-069x-13-105.

Keeley JE, Syphard AD, 2016. Climate change and future fire regimes: examples from California. Geosciences 6 (3), 14. 10.3390/geosciences6030037.

Keywood M, Kanakidou M, Stohl A, Dentener F, Grassi G, Meyer CP, … Burrows J, 2013. Fire in the air: biomass burning impacts in a changing climate. Crit. Rev. Environ. Sci. Technol. 43 (1), 40–83. 10.1080/10643389.2011.6043389.

Kim KH, Kabir E, Kabir S, 2015. A review on the human health impact of airborne particulate matter. Environ. Int. 74, 136–143. 10.1016/j.envint.2014.10.005. [PubMed: 25452430]

Kim YH, Warren SH, Krantz QT, King C, Jaskot R, Preston WT, … Gilmour MI, 2018. Mutagenicity and lung toxicity of smoldering vs. flaming emissions from various biomass fuels: implications for health effects from wildland fires. Environ. Health Perspect. 126 (1). 10.1289/ehp2200.

Kim YH, King C, Krantz T, Hargrove MM, George JJ, McGee J, … Gilmour MI, 2019. The role of fuel type and combustion phase on the toxicity of biomass smoke following inhalation exposure in mice. Arch. Toxicol. 93 (6), 1501–1513. 10.1007/s00204-019-02450-5. [PubMed: 31006059]

Kjellstrom T, Briggs D, Freyberg C, Lemke B, Otto M, Hyatt O, 2016. Heat, human performance, and occupational health: a key issue for the assessment of global climate change impacts. In: Fielding JE (Ed.), Annual Review of Public Health. vol 37. pp. 97–112 Vol. 37.

Kunzli N, Avol E, Wu J, Gauderman WJ, Rappaport E, Millstein J, … Peters JM, 2006. Health effects of the 2003 southern California wildfires on children. Am. J. Respir. Crit. Care Med. 174 (11), 1221–1228. 10.1164/rccm.200604-519OC. [PubMed: 16946126]

Leung LR, Gustafson WI, 2005. Potential regional climate change and implications to us air quality. Geophys. Res. Lett. 32 (16). 10.1029/2005gl022911.

Levy D, Lumley T, Sheppard L, Kaufman J, Checkoway H, 2001. Referent selection in case-crossover analyses of acute health effects of air pollution. Epidemiology 12 (2), 186–192. [PubMed: 11246579]

Lipsett M, Materna B, Stone SL, Therriault S, Blaisdell R, Cook J, 2016. Wildfire smoke: a guide for public health officials (revised May 2016). Retrieved from. https://www3.epa.gov/airnow/wildfire_may2016.pdf.
Liu JC, Pereira G, Uhl SA, Bravo MA, Bell ML, 2015. A systematic review of the physical health impacts from non-occupational exposure to wildfire smoke. Environ. Res. 136, 120–132. 10.1016/j.envres.2014.10.015. [PubMed: 25460628]

Liu JC, Wilson A, Mickley LJ, Dominici F, Ebisu K, Wang Y, … Bell ML, 2017. Wildfire-specific fine particulate matter and risk of hospital admissions in urban and rural counties. Epidemiology 28 (1), 77–85. 10.1097/ede.0000000000000556. [PubMed: 27648592]

Liu XX, Huey LG, Yokelson RJ, Selimovic V, Simpson JJ, Muller M, … Wolfe GM, 2017. Airborne measurements of western us wildfire emissions: comparison with prescribed burning and air quality implications. J. Geophys. Res.-Atmos. 122 (11), 6108–6129. 10.1002/2016jd026315.

Liu YQ, Goodrick SL, Stanturf JA, 2013. Future us wildfire potential trends projected using a dynamically downscaled climate change scenario. For. Ecol. Manag. 294, 120–135. 10.1016/j.foreco.2012.06.049.

Maclure M, 1991. The case-crossover design: a method for studying transient effects on the risk of acute events. Am. J. Epidemiol. 133 (2), 144–153. [PubMed: 1985444]

Martin MV, Heald CL, Ford B, Prenni AJ, Wiedinmyer C, 2013. A decadal satellite analysis of the origins and impacts of smoke in Colorado. Atmos. Chem. Phys. 13 (15), 7429–7439. 10.5194/acp-13-7429-2013.

Na K, Cocker DR, 2008. Fine organic particle, formaldehyde, acetaldehyde concentrations under and after the influence of fire activity in the atmosphere of riverside, California. Environ. Res. 108 (1), 7–14. 10.1016/j.envres.2008.04.004. [PubMed: 18538316]

NASA, 2013. Worldview earthdata. Retrieved from. https://worldview.earthdata.nasa.gov/.

NASA, 2018. Fire Information for Resource Management System (Firms). Retrieved from. https://earthdata.nasa.gov/earth-observation-data/near-real-time/firms.

Park SS, Wexler AS, 2008. Size-dependent deposition of particles in the human lung at steady-state breathing. J. Aerosol Sci. 39 (3), 266–276. 10.1016/j.aerosci.2007.11.006.

Penrod A, Zhang Y, Wang K, Wu SY, Leung LR, 2014. Impacts of future climate and emission changes on us air quality. Atmos. Environ. 89, 533–547. 10.1016/j.atmosenv.2014.01.001.

Pope CA, Dockery DW, 2006. Health effects of fine particulate air pollution: lines that connect. J. Air Waste Manage. Assoc. 56 (6), 709–742. 10.1080/10473229.2006.10464485.

Rappold AG, Stone SL, Cascio WE, Neas LM, Kilariu JV, Carraway MS, … Devlin RB, 2011. Peat bog wildfire smoke exposure in rural North Carolina is associated with cardiopulmonary emergency department visits assessed through syndromic surveillance. Environ. Health Perspect. 119 (10), 1415–1420. 10.1289/ehp.1003206. [PubMed: 21705297]

Rappold AG, Reyes J, Pouliot G, Cascio WE, Diaz-Sanchez D, 2017. Community vulnerability to health impacts of wildland fire smoke exposure. Environmental Science & Technology 51 (12), 6674–6682. 10.1021/acs.est.6b06200. [PubMed: 28493694]

Reid CE, Jerrett M, Petersen ML, Pfister GG, Morefield PE, Tager IB, … Balmes JR, 2015. Spatiotemporal prediction of fine particulate matter during the 2008 northern California wildfires using machine learning. Environmental Science & Technology 49 (6), 3887–3896. 10.1021/es505846r. [PubMed: 25648639]

Reid CE, Brauer M, Johnston FH, Jerrett M, Balmes JR, Elliott CT, 2016. Critical review of health impacts of wildfire smoke exposure. Environ. Health Perspect. 124 (9), 1334–1343. 10.1289/ehp.1409277. [PubMed: 27082891]

Reid CE, Jerrett M, Tager IB, Petersen ML, Mann JK, Balmes JR, 2016. Differential respiratory health effects from the 2008 northern California wildfires: a spatiotemporal approach. Environ. Res. 150, 227–235. 10.1016/j.envres.2016.06.012. [PubMed: 27318255]

Reid CE, Considine EM, Watson GL, Telesca D, Pfister GG, Jerrett M, 2019. Associations between respiratory health and ozone and fine particulate matter during a wildfire event. Environ. Int. 129, 291–298. 10.1016/j.envint.2019.04.033. [PubMed: 31146163]

Shin HJ, Cho HG, Park CK, Park KH, Lim HB, 2017. Comparative in vitro biological toxicity of four kinds of air pollution particles. Toxicological Research 33 (4),305–313. 10.5487/tr.2017.33.4.305. [PubMed: 29071015]
Singer AJ, Thode HC, Pines JM, 2019. Us emergency department visits and hospital discharges among uninsured patients before and after implementation of the affordable care act. JAMA Netw. Open 2 (4), 8. 10.1001/jamanetworkopen.2019.2662.

Stavros EN, McKenzie D, Larkin N, 2014. The climate-wildfire-air quality system: interactions and feedbacks across spatial and temporal scales. Wiley Interdisciplinary Reviews-Climate Change 5 (6), 719–733. 10.1002/wcc.303.

Strickland MJ, Darrow LA, Klein M, Flanders WD, Samat JA, Waller LA, … Tolbert PE, 2010. Short-term associations between ambient air pollutants and pediatric asthma emergency department visits. Am. J. Respir. Crit. Care Med. 182 (3), 307–316. 10.1164/rccm.200908-1201OC. [PubMed: 20378732]

The Environmental Pollution Panel, 1965. Restoring the Quality of Our Environment. (Retrieved from Washington, D.C.).

Thelen B, French NHF, Koziol BW, Billmire M, Owen RC, Johnson J, … Wu SL, 2013. Modeling acute respiratory illness during the 2007 San Diego wildland fires using a coupled emissions-transport system and generalized additive modeling. Environ. Health 12. 10.1186/1476-069x-12-94.

Thornton PK, Ericksen PJ, Herrero M, Challinor AJ, 2014. Climate variability and vulnerability to climate change: a review. Glob. Chang. Biol. 20 (11), 3313–3328. 10.1111/gcb.12581. [PubMed: 24668802]

United States Environmental Protection Agency, 2017a. Particulate matter (pm) basics. Retrieved from. https://www.epa.gov/pm-pollution/particulate-matter-pm-basics.

United States Environmental Protection Agency, 2017b. Stratospheric Ozone Protection: 30 Years of Progress and Achievements. (Retrieved from Washington, D.C.).

USEPA, 2019. Integrated science assessment for particulate matter. Retrieved from. https://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=341593.

Wegesser TC, Pinkerton KE, Last JA, 2009. California wildfires of 2008: coarse and fine particulate matter toxicity. Environ. Health Perspect. 117 (6), 893–897. 10.1289/ehp.0800166. [PubMed: 19590679]

Wegesser TC, Franzi LM, Mitloehner FM, Figueroa-Fernandez A, Last JA, 2010. Lung antioxidant and cytokine responses to coarse and fine particulate matter from the great California wildfires of 2008. Inhal. Toxicol. 22 (7), 561–570. 10.3109/08958370903571849. [PubMed: 20388000]

Westerling AL, 2016. Increasing western us forest wildfire activity: sensitivity to changes in the timing of spring (vol 371, 20150178, 2016). Philosophical Transactions of the Royal Society B-Biological Sciences 371 (1707). 10.1098/rstb.2016.0373.

Westerling AL, Hidalgo HG, Cayan DR, Swetnam TW, 2006. Warming and earlier spring increase western us forest wildfire activity. Science 313 (5789), 940–943. 10.1126/science.1128834. [PubMed: 16825536]

Wettstein ZS, Hoshiko S, Fahimi J, Harrison RJ, Cascio WE, Rappold AG, 2018. Cardiovascular and cerebrovascular emergency department visits associated with wildfire smoke exposure in California in 2015. J. Am. Heart Assoc. 7 (8), 10.1161/jaha.117.007492.

Wong LSN, Aung HH, Lame MW, Wegesser TC, Wilson DW, 2011. Fine particulate matter from urban ambient and wildfire sources from California’s San Joaquin valley initiate differential inflammatory, oxidative stress, and xenobiotic responses in human bronchial epithelial cells. Toxicol. in Vitro 25 (8), 1895–1905. 10.1016/j.tiv.2011.06.001. [PubMed: 21703343]

Wu XX, Lu YM, Zhou S, Chen LF, Xu B, 2016. Impact of climate change on human infectious diseases: empirical evidence and human adaptation. Environ. Int. 86, 14–23. 10.1016/j.envint.2015.09.007. [PubMed: 26479830]

Xu HM, Leon JF, Liouasse C, Guinot B, Yoboue V, Akpo AB, … Cao JJ, 2019. Personal exposure to pm2.5 emitted from typical anthropogenic sources in southern west Africa: Chemical characteristics and associated health risks. Atmos. Chem. Phys. 19 (10), 6637–6657. 10.5194/acp-19-6637-2019.

Environ Int. Author manuscript; available in PMC 2021 May 28.
Fig. 1.
Daily mean modeled PM$_{2.5}$ from fire seasons 2011–2014 in Colorado. State-averaged time series data for fire seasons (May-August) 2011–2014 show total modeled PM$_{2.5}$ levels by day, month, and year.
Fig. 2.
Daily mean ratio of PM$_{2.5}$ attributed to wildfire. State-averaged time series data for fire seasons (May-August) 2011–2014 depicting ratio of modeled smoke PM$_{2.5}$ to total modeled PM$_{2.5}$. 
Fig. 3.
Domain-wide daily mean total PM$_{2.5}$ and mean ratio of PM$_{2.5}$ on fire days (fire PM > 1%).
Time series depicting both total and ratio of modeled smoke PM$_{2.5}$ to total modeled PM$_{2.5}$. 
Fig. 4. Daily mean total PM$_{2.5}$ and mean ratio of PM$_{2.5}$ attributed to wildfire at two locations. Time series depicting both total and ratio of modeled smoke PM$_{2.5}$ to total modeled PM$_{2.5}$. A) Location near the High Park Fire (June 9–30, 2012) and B) Location near Waldo Canyon Fire (June 23-July 10, 2012). Red boxes indicate active fire days. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)
Fig. 5. 
Satellite smoke plume, modeled total PM$_{2.5}$ and smoke PM$_{2.5}$ for west fork fire complex, June 22, 2013. Modeled data corresponds to visible smoke plume as shown in A-C. A) Satellite image from June 22, 2013 with active West Fork Complex Fire (NASA, 2013). B) Total PM$_{2.5}$ for Colorado on June 22, 2013. C) Amount of PM$_{2.5}$ attributed to fire on June 22, 2013.
### Fig. 6.
Wildfire smoke PM$_{2.5}$ exposure and respiratory outcomes. Odds ratios for both total and age-stratified respiratory outcomes per 1 μg/m$^3$ increase in wildfire smoke PM$_{2.5}$ exposure, arranged by outcome and age group.

| Health Outcome                  | Age    | Case Count | Fire PM (1μg/m$^3$) 3-day average OR & 95% CI |
|---------------------------------|--------|------------|-----------------------------------------------|
|                                 |        |            | OR                                            |
| Asthma                          | Child  | 10,184     | 1.075 (1.035,1.115)                           |
|                                 | Adult  | 10,448     | 1.091 (1.060,1.122)                           |
|                                 | Elderly| 1,286      | 1.009 (0.920,1.106)                           |
|                                 | All    | 21,918     | 1.081 (1.058,1.105)                           |
| Upper Respiratory Infection     | Child  | 41,710     | 1.010 (0.991,1.029)                           |
|                                 | Adult  | 26,718     | 1.005 (0.984,1.026)                           |
|                                 | Elderly| 2,286      | 1.004 (0.940,1.072)                           |
|                                 | All    | 70,714     | 1.008 (0.995,1.021)                           |
| Bronchitis                      | Child  | 1,403      | 0.971 (0.890,1.060)                           |
|                                 | Adult  | 6,772      | 1.044 (1.005,1.085)                           |
|                                 | Elderly| 1,223      | 0.908 (0.805,1.024)                           |
|                                 | All    | 9,398      | 1.018 (0.984,1.052)                           |
| Chronic Obstructive Pulmonary Disease | Child  | 22         | --                                            |
|                                 |Adult   | 4,491      | 1.056 (1.015,1.100)                           |
|                                 | Elderly| 5,486      | 0.989 (0.951,1.030)                           |
|                                 | All    | 9,999      | 1.020 (0.991,1.049)                           |
| Respiratory Disease             | Child  | 61,713     | 1.016 (1.001,1.032)                           |
|                                 | Adult  | 56,529     | 1.030 (1.017,1.044)                           |
|                                 | Elderly| 17,121     | 1.000 (0.976,1.024)                           |
|                                 | All    | 135,363    | 1.021 (1.012,1.031)                           |
| Health Outcome                  | Age    | Case Count | Fire PM (1μg/m³) 2-day average OR & 95% CI |
|--------------------------------|--------|------------|----------------------------------------|
| Acute Myocardial Infarction    | adult  | 3,228      | 1.011 (0.963,1.062)                     |
|                                | elderly| 3,633      | 1.002 (0.954,1.052)                     |
|                                | all    | 6,862      | 1.006 (0.972,1.042)                     |
| Congestive Heart Failure       | adult  | 2,335      | 0.962 (0.896,1.032)                     |
|                                | elderly| 6,087      | 0.993 (0.957,1.029)                     |
|                                | all    | 8,450      | 0.999 (0.955,1.018)                     |
|                                |        |            |                                        |
|                                | adult  | 5,799      | 0.993 (0.956,1.030)                     |
|                                | elderly| 8,877      | 1.010 (0.980,1.040)                     |
|                                | all    | 14,946     | 1.005 (0.982,1.028)                     |
| Dysrhythmia                    |        |            |                                        |
|                                | adult  | 4,762      | 0.999 (0.957,1.043)                     |
|                                | elderly| 5,592      | 1.022 (0.982,1.063)                     |
|                                | all    | 10,358     | 1.011 (0.982,1.041)                     |
| Ischemic Heart Disease         |        |            |                                        |
|                                | adult  | 7,638      | 0.964 (0.928,1.002)                     |
|                                | elderly| 8,713      | 0.978 (0.945,1.013)                     |
|                                | all    | 16,471     | 0.971 (0.946,1.000)                     |
| Peripheral/Cerebrovascular     |        |            |                                        |
|                                | adult  | 71,690     | 0.986 (0.965,1.008)                     |
|                                | elderly| 115,842    | 1.004 (0.987,1.021)                     |
|                                | all    | 189,632    | 0.998 (0.984,1.000)                     |

Fig. 7.
Wildfire smoke PM$_{2.5}$ exposure and cardiovascular outcomes. Odds ratios for both total and age-stratified cardiovascular outcomes per 1 μg/m$^3$ increase in wildfire smoke PM$_{2.5}$ exposure, arranged by outcome and age group.
### Table 1

Epidemiologic data descriptive statistics.

|                        | Case count |
|------------------------|------------|
| Total records          | 490,368    |
| Geocoded addresses     | 446,106    |
| Non-geocoded addresses | 44,262     |
| Year of event          |            |
| 2011                   | 102,318    |
| 2012                   | 102,574    |
| 2013                   | 111,737    |
| 2014                   | 129,477    |
| Age ranges             |            |
| 0–18 y                 | 94,022     |
| 19–64 y                | 202,665    |
| 65+ y                  | 149,419    |
| Sex                    |            |
| Female                 | 241,282    |
| Male                   | 204,823    |