Journal of the American Heart Association

ORIGINAL RESEARCH

Wildland Fire–Related Smoke $PM_{2.5}$ and Cardiovascular Disease Emergency Department Visits in the Western United States

Linzi Li, MPH, MSPH; Wenhao Wang, MPH; Howard H. Chang, PhD; Alvaro Alonso, MD, PhD; Yang Liu, PhD

BACKGROUND: The impact of short-term exposure to fine particulate matter with a diameter ≤2.5 μm ($PM_{2.5}$) due to wildland fire smoke on the risk of cardiovascular disease (CVD) remains unclear. We investigated the association between short-term exposure to wildfire smoke $PM_{2.5}$ and emergency department visits for acute CVD in the western United States from 2007 to 2018.

METHODS: We analyzed 49 759 958 emergency department visits for primary or secondary diagnoses of atrial fibrillation (AF), acute myocardial infarction, heart failure, stroke, and total CVD across 5 states. Daily smoke, nonsmoke, and total $PM_{2.5}$ were estimated using a 1-km resolution satellite-driven multistage model and were aggregated to the zip code level. A case-crossover study design was used, adjusting for temperature, relative humidity, and day of the year.

RESULTS: The mean smoke $PM_{2.5}$ was 1.27 (interquartile range, 0–1.29) μg/m^3. A 10-μg/m^3 increase in smoke $PM_{2.5}$ was associated with a minuscule decreased risk for AF (odds ratio, 0.994 [95% CI, 0.991–0.997]), heart failure (odds ratio, 0.995 [95% CI, 0.992–0.998]), and CVD (odds ratio, 0.997 [95% CI, 0.996–0.998]) but not for acute myocardial infarction and stroke. Adjusting for nonsmoke $PM_{2.5}$ did not alter these associations. A 10-μg/m^3 increase in total $PM_{2.5}$ was linked to a small increased risk for all outcomes except stroke (odds ratio for CVD, 1.006 [95% CI, 1.006–1.007]). Associations were similar across sex and age groups.

CONCLUSIONS: Short-term wildfire smoke $PM_{2.5}$ exposure was unexpectedly associated with a slightly lower risk of CVD emergency department visits. Whether these findings are due to methodological issues, behavioral changes, or other factors requires further investigation.

Key Words: air pollution ■ cardiovascular disease ■ $PM_{2.5}$ ■ wildland fire

The surge in wildfire occurrences globally in recent decades has raised alarms about the role of climate change.[1] The repercussions of climate change include exacerbating fire frequency and endangering human livelihoods, ecosystems, and public health.[1–3] Coupled with warming and drought climatic conditions, severe fire events can catalyze vegetation changes, implicating rapid ecosystem shifts and reductions in valued resources.[1,4,5] In the United States, the wildfire-burned area has increased ≈4-fold over the past 4 decades, and the trend is expected to continue as a result of climate warming, earlier spring, and other factors.[6] The western United States, an area prone to wildfire disasters, often faces consecutive years marked

Correspondence to: Yang Liu, PhD, Gangarosa Department of Environmental Health, 1518 Clifton Road NE, Claudia Nance Rollins Bldg., Room 7011, Atlanta, GA 30322. Email: yang.liu@emory.edu

This manuscript was sent to William W. Aitken, MD, Assistant Editor, for review by expert referees, editorial decision, and final disposition.

Supplemental Material is available at https://www.ahajournals.org/doi/suppl/10.1161/JAHA.124.040080

For Disclosures and Sources of Funding, see page 8.

JAHA is available at: www.ahajournals.org/journal/jaha

RESEARCH PERSPECTIVE

What Is New?

- Our study found a small decrease in cardiovascular disease emergency department visit risk associated with short-term exposure to wildfire smoke fine particulate matter with a diameter ≤2.5 μm ($PM_{2.5}$), using >49 million cardiovascular disease emergency department visit records and high-resolution satellite-based $PM_{2.5}$ data.
- A consistent risk increase was observed with total $PM_{2.5}$ exposure, suggesting that smoke-related $PM_{2.5}$ may have different temporal dynamics.

What Question Should Be Addressed Next?

- Future research focusing on short-term wildfire smoke $PM_{2.5}$ exposure on cardiovascular disease emergency department visit should consider behavioral factors and copollutant influences and adopt prospective study designs.

Nonstandard Abbreviations and Acronyms

PM	particulate matter
$PM_{2.5}$	fine particulate matter with a diameter ≤2.5 μm

by both drought and heightened fire weather.[7] Since 1986, the frequency of large wildfires in the western United States has increased 4-fold, and the total area burned has expanded to 6.5 times the average levels recorded between 1970 and 1986.[8] Smoke from wildfires significantly contributes to fine particulate matter in the atmosphere, a form of air pollution consisting of particles with a diameter of <2.5 μm ($PM_{2.5}$).[9,10] The contribution of wildfire smoke to $PM_{2.5}$ concentrations across the United States has increased since the mid-2000s, now representing up to half of the total $PM_{2.5}$ in western regions, a stark increase from <20% around 2015.[6,11] Additionally, smoke influences the trends in extreme $PM_{2.5}$ days in western and midwestern states. Evidence shows that since 2012, the rise in the number of days above 35 μg/m^3 of $PM_{2.5}$ would have been lower without wildfire smoke, and the days surpassing 35 μg/m^3 of $PM_{2.5}$ would not have exceeded the threshold absent wildfire smoke on that day.[12]

Long-term exposure to ambient $PM_{2.5}$ is linked with several adverse cardiometabolic health outcomes and cardiopulmonary disease, including increased blood glucose, endothelial dysfunction, incident cardiovascular disease (CVD) events, chronic obstructive pulmonary disease, pneumonia, and all-cause death.[13–15] Recognized as a substantial contributor to ambient $PM_{2.5}$ levels, wildfire smoke has been identified as a potential environmental risk factor for CVD, which includes a range of heart and blood vessel disorders and stroke.[10,16–19] Between 2017 and 2020, nearly 10% of US adults, totaling 28.6 million individuals, experienced some form of CVD, including coronary artery disease, heart failure (HF), and stroke.[20] Moreover, CVD complications significantly compound the burden for both individuals and the health care system worldwide. CVD can exacerbate or increase the risk of other chronic conditions, such as diabetes, kidney disease, and functional decline, further impairing the quality of life and potentially leading to early death.[20] Globally, CVD is the primary cause of death, responsible for 19.9 million (95% uncertainty interval, 18.4–21.2) deaths in 2021 with 13.3% of these cardiovascular deaths attributable to ambient $PM_{2.5}$ exposure.[20,21] In the United States, among the Medicare enrollees, a 10 μg/m^3 increase in exposure to a 12-month moving average $PM_{2.5}$ was associated with 1.56-fold (95% CI, 1.55–1.57) increase in cardiovascular death.[14]

Previous literature has documented the potential pathophysiologic mechanisms through which wildfire smoke can contribute to CVD. Inhaled particulate matter (PM) that accumulates in the alveoli may be a trigger of a series of adverse reactions that lead to CVDs, including inflammation, oxidative stress, thrombosis and coagulation, and vascular dysfunction.[22–24] Individual susceptibility conditions and concomitant existing air copollutants, such as preexisting coronary artery disease and ozone, could worsen these effects.[22,25] Numerous epidemiological studies have investigated the cardiovascular effects of wildfire smoke. However, the transient nature of wildfire events introduces challenges in defining the exposure time window and ensuring consistency in the measurement of PM during wildfire events. There are discrepancies in whether the acute PM exposure should be based on 3-day averages or 1-hour daily peak averages, and whether chronic PM exposure should be quantified using annual averages or extreme daily wildfire PM. Although certain past studies have indicated that wildfire smoke $PM_{2.5}$ increased CVD morbidity and death, these associations have not been consistent across regions, populations, and periods, partially due to different wildfire smoke $PM_{2.5}$ concentration measures.[26,27] Measuring smoke $PM_{2.5}$ have challenges, including limited spatial monitoring coverage, aerosol optical depth data availability, model performance in extreme concentrations, temporal and spatial variability, and so on. In a global time series study in 749 locations, short-term wildfire smoke $PM_{2.5}$ did not have a substantial impact

on cardiovascular death in the United States (relative risk, 1.014 [95% CI, 0.998–1.031]), while wildfire-related $PM_{2.5}$ was associated with a higher risk of cardiovascular death in the pooled result of all the areas.[28]

Therefore, we aim to add to the evidence by exploring the association between exposure to short-term wildfire smoke $PM_{2.5}$ exposure and emergency department (ED) visits for CVD in the western United States from 2007 to 2018, using satellite-driven exposure measures.

METHODS

In accordance with the agreement with each state, the ED visit data are prohibited from sharing to protect health information. The Daymet daily 1-km meteorological data are available in the Oak Ridge National Laboratory data archive (https://doi.org/10.3334/ORNLDAAC/2129). The zip code–level wildfire smoke $PM_{2.5}$ data are available on Figshare (https://doi.org/10.6084/m9.figshare.25016510).

ED Visit Data

ED visits for CVDs in 5 western US states were obtained from hospital associations or state health departments. The states and corresponding data ranges are California (2007–2018), Arizona (2010–2018), Nevada (2009–2016), Oregon (2014–2018), and Utah (2007–2016). An ED visit was classified as either outpatient or inpatient care directly from the ED. The ED visit record contains service date, age in years, sex, race, zip code, and *International Classification of Diseases* (*ICD*) diagnosis codes. Visits before October 1, 2015, used *ICD*, *Ninth Revision* (*ICD-9*) and visits afterward used *ICD*, *Tenth Revision* (*ICD-10*). The CVDs of interest in this study were acute myocardial infarction (AMI); stroke; HF; atrial fibrillation (AF); and total CVD, which was defined as total circulatory disease. We included both the primary and secondary diagnoses of CVDs. The *ICD* codes used for identification were AMI (*ICD-9*: 410; *ICD-10*: I21, I22), stroke (*ICD-9*: 430, 431, 434, 436; *ICD-10*: I60, I61, I62), HF (*ICD-9*: 428; *ICD-10*: I50), AF (*ICD-9*: 427.3x; *ICD-10*: I48), and total circulatory diseases (*ICD-9*: 390–459; *ICD-10*: I00-I99). This study received approval from the Institutional Review Board at Emory University (STUDY00004823), which also granted an exemption from informed consent requirements due to the impracticability of obtaining consent from each individual patient and the minimal risk associated with the study.

Exposure Measures

Daily wildfire smoke $PM_{2.5}$ was estimated using a multistage, chemical transport modeling-based framework at a high spatial resolution of 1 km. This model incorporates advanced air quality $PM_{2.5}$ simulations, various satellite remote sensing products, meteorological analyses, land use information, and comprehensive ground-level observations. The total $PM_{2.5}$ model was developed using data from areas impacted by wildfire smoke, while the background $PM_{2.5}$ model was based on data from areas without wildfire smoke. Both models were applied to predict $PM_{2.5}$ concentrations in various scenarios. The difference between total $PM_{2.5}$ and background $PM_{2.5}$ was calculated to isolate the $PM_{2.5}$ contribution from wildfire smoke. Detailed modeling system information has been published elsewhere.[29] The wildfire smoke, total, and nonsmoke $PM_{2.5}$ data were further aggregated to the patients' zip code level of each day. For all ED records in Nevada, the aggregation was performed by matching the first 4 digits of the zip codes due to the unavailability of the full zip codes in this state. Additionally, the contribution of smoke $PM_{2.5}$ to total $PM_{2.5}$ were modeled in sensitivity analysis.

Covariates

In this study, relative humidity, average temperature, and day of year were accounted for as confounding variables. Daily average temperature was calculated by averaging the minimum and maximum temperatures, and daily relative humidity was estimated using the Magnus formula.[30] Daymet data on daily minimum and maximum temperatures in degrees Celsius and vapor pressure in pascals, with a resolution of 1 km from 2007 to 2018, were sourced from the Oak Ridge National Laboratory Distributed Active Archive Center for Biogeochemical Dynamics.[31] The Daymet meteorological data were also aggregated to each patient's zip code level each day.

Statistical Analysis

We used a case-crossover study design to investigate the associations between short-term smoke $PM_{2.5}$ exposure and ED visits for CVD in the western United States.[32] The time-stratified approach was used in the control selection: Each ED visit (case) was matched with up to 4 nonevent days based on the day of the week in the same calendar month.[33,34] Because the selection of possible control dates that form a stratum do not depend on the event date, the time-stratified approach is not subject to bias resulting from the time trend.[34] Additionally, stable characteristics at the individual level (eg, sex, age, socioeconomic status) are automatically controlled for in the control selection phase. We then performed conditional logistic regression to estimate the associations between smoke $PM_{2.5}$ and total $PM_{2.5}$ with CVD ED visits. We adjusted

for nonsmoke $PM_{2.5}$ in the model examining the effect of smoke $PM_{2.5}$ because wildfire smoke $PM_{2.5}$ can interact with ambient air pollutants. The model specifications are shown below:

$$logit(P_{ED}) = \beta_0 + \beta_1 smoke\ PM_{2.5} + ns(RH) + ns(temp) + ns(doy)$$

$$logit(P_{ED}) = \beta_0 + \beta_{1a} smoke\ PM_{2.5} + \beta_{1b} non\_smoke\ PM_{2.5} + ns(RH) + ns(temp) + ns(doy)$$

$$logit(P_{ED}) = \beta_0 + \beta_1 total\ PM_{2.5} + ns(RH) + ns(temp) + ns(doy)$$

P_{ED} represents the probability of occurrence of the ED visits for CVD events. For the primary analyses, the outcomes were any primary and secondary diagnosis of the ED visits for CVD events. Relative humidity (*RH*), average temperature (*temp*), and day of the year (*doy*) were modeled using natural splines (*ns*). The degrees of freedom for the *ns* function were 3. For wildfire smoke, nonsmoke, and total $PM_{2.5}$ concentrations and covariates, we used 1-, 2-, and 3-day averages leading up to and including the day of the ED visit. For sensitivity analysis, we conducted the following: (1) stratified the study population by sex (male and female) and age (<65 years and ≥65 years); (2) examined the effect of the ratio of $\frac{smoke\ PM_{2.5}}{total\ PM_{2.5}}$ and tested an interaction between the ratio and total $PM_{2.5}$ as following:

$$logit(P_{ED}) = \beta_0 + \beta_{1a} \frac{smoke\ PM_{2.5}}{total\ PM_{2.5}} + ns(RH) + ns(temp) + ns(doy) + \beta_{1b} \frac{smoke\ PM_{2.5}}{total\ PM_{2.5}} * total\ PM_{2.5};$$

(3) estimated the associations between smoke $PM_{2.5}$ and total $PM_{2.5}$ with primary CVD ED visits; (4) examined the impact of 7-day average wildfire smoke, nonsmoke, and total $PM_{2.5}$ concentrations leading up to and including the day of the ED visit. The ratio $\frac{smoke\ PM_{2.5}}{total\ PM_{2.5}}$ serves as an indicator of the relative contribution of wildfire smoke $PM_{2.5}$ to overall $PM_{2.5}$ on a given day. By examining the relationship between the ratio of smoke $PM_{2.5}$ to total $PM_{2.5}$ and CVD ED visits, we aimed to assess whether the relative dominance of smoke $PM_{2.5}$, as opposed to background $PM_{2.5}$, has a differential impact on health outcomes given the same level of total $PM_{2.5}$ exposure. Odds ratios (ORs) and their corresponding 95% CIs were reported. All the analyses were conducted in R 4.2.2 (2022-10-31; available at: https://cran.r-project.org/bin/windows/base/old/4.2.2/).

RESULTS

In total, 49 759 958 ED visits with a primary or secondary CVD diagnosis were available. Among those, 6 808 839 (14%) were visits for AFs, 1 222 053 (2%) were visits for AMIs, 7 194 474 (14%) were visits for HFs, and 808 396 (2%) were visits for strokes. The average age at the time of the visit was 64±17.4 years, and 61% of the patients were women. Table 1 shows the characteristics of those who were included in this study with ED visits for primary or secondary diagnoses of CVD. Over the study period from January 1, 2007, to December 31, 2018, the average wildfire smoke, nonsmoke, and total $PM_{2.5}$ were 1.11 (interquartile range, 1.29) μg/m^3, 7.64 (interquartile range, 5.15) μg/m^3, and 8.77 (interquartile range, 5.77) μg/m^3, respectively. The distributions of smoke $PM_{2.5}$, total $PM_{2.5}$, nonsmoke $PM_{2.5}$, and the ratio of smoke $PM_{2.5}$ and total $PM_{2.5}$ over the study period are shown in the Figure.

Wildfire Smoke $PM_{2.5}$ and CVD ED Visits in the Western US

During the study period, 10 μg/m^3 higher smoke $PM_{2.5}$ on the day of the ED visit was associated with a small decrease in the risk of ED visits for AF (OR, 0.994 [95% CI, 0.991–0.997]), HF (OR, 0.995 [95% CI, 0.992–0.998]), and CVD (OR, 0.997 [95% CI, 0.996–0.998]). However, there was no association observed with AMI and stroke. The same results were found between 2-day and 3-day moving average smoke $PM_{2.5}$

Table 1. Characteristics of Subjects With Primary or Secondary Diagnosis of Total CVD (N=49 759 958)

	Statistics	
Age, y	64±17.4	
Female sex	30 095 611 (60.5)	
Race		
White	39 128 609 (78.6)	
Black	6 685 553 (13.4)	
American Indian or Alaska Native	467 695 (0.9)	
Asian/Pacific Islander	3 964 921 (8.0)	
Other race	800 727 (1.6)	
Cardiovascular outcomes	Primary or secondary diagnosis	Primary diagnosis
AF	6 808 839 (13.7)	867 623 (9.6)
AMI	1 222 053 (2.5)	786 445 (8.7)
HF	7 194 474 (14.5)	1 172 596 (13.0)
Stroke	808 396 (1.6)	635 225 (7.0)
Total CVD	49 759 958 (100)	9 022 749 (100)

Data are shown as mean±SD or n (%). Percentages of race do not add up to 100% due to missing data and overlapping categories. AF indicates atrial fibrillation; AMI, acute myocardial infarction; CVD, cardiovascular disease; and HF, heart failure.

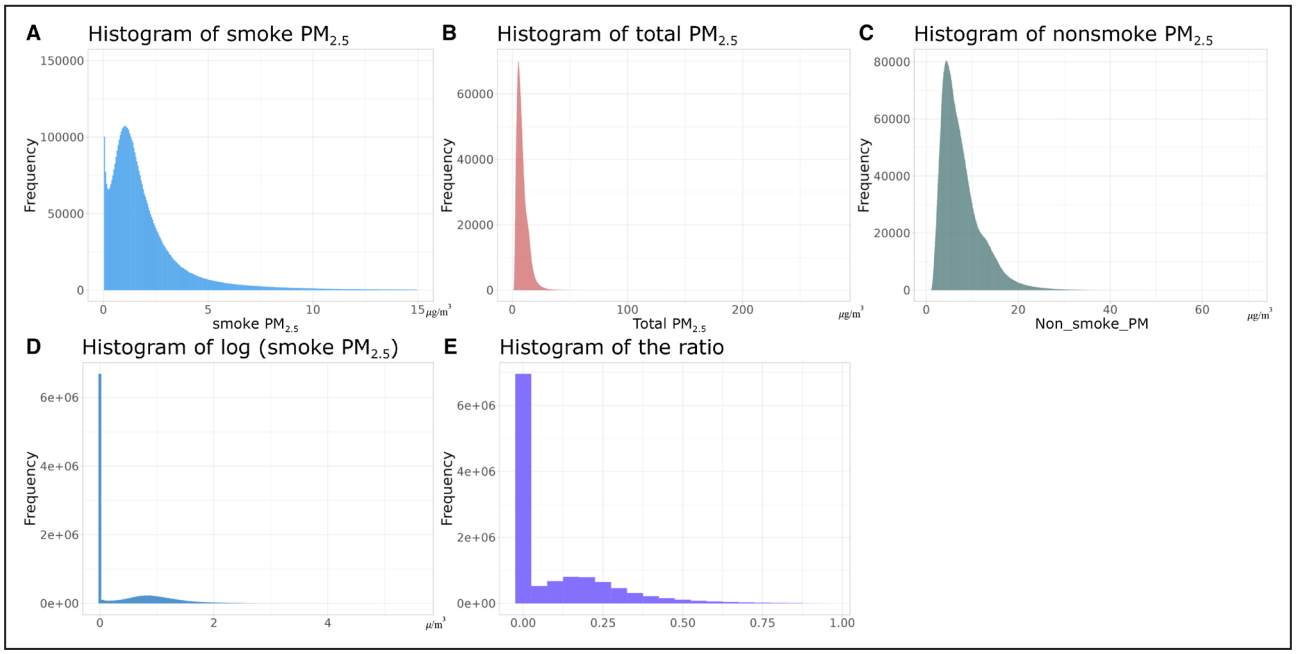

Figure. Histograms of smoke $PM_{2.5}$ within 15 μg/m³ and the frequency within 150000 (A), total $PM_{2.5}$ (B), nonsmoke $PM_{2.5}$ (C), log (smoke $PM_{2.5}$+1) (D), and the ratio of smoke $PM_{2.5}$/total $PM_{2.5}$ (E).
Period: January 1, 2007, to December 31, 2018. $PM_{2.5}$ indicates fine particulate matter with a diameter ≤2.5 μm.

and each outcome (Table 2). Further adjusting for nonsmoke $PM_{2.5}$ in the model did not change the associations substantively for all cardiovascular outcomes. Total $PM_{2.5}$ was associated with an increased risk of ED visits for CVDs. A 10 μg/m³ higher total $PM_{2.5}$ on the day was associated with a slightly increased risk of ED visits for AF (OR, 1.005 [95% CI, 1.004–1.007]), AMI (OR, 1.010 [95% CI, 1.006–1.015]), HF (OR, 1.008 [95% CI, 1.006–1.010]), and CVD (OR, 1.006 [95% CI, 1.006–1.007]), but not for stroke (OR, 1.001 [95% CI, 0.994–1.007]). Similar associations were observed with the 2-day and 3-day moving average total $PM_{2.5}$ across all cardiovascular outcomes (Table 2).

Stratified Analysis

The analysis stratified by age (<65 and ≥65 years) provided similar results to the main analysis. In both age groups, an increased 1-, 2-, and 3-day smoke resulted in a slight decrease in the risk or no risk change of

Table 2. Effect of 1-, 2-, and 3-Day Smoke $PM_{2.5}$ and Total $PM_{2.5}$ on CVD ED Visits

	AF	AMI	HF	Stroke	CVD
Smoke $PM_{2.5}$					
Not adjusting for nonsmoke					
1-d	0.994 (0.991–0.997)	0.999 (0.992–1.006)	0.995 (0.992–0.998)	0.996 (0.985–1.008)	0.997 (0.996–0.998)
2-d	0.992 (0.989–0.995)	0.997 (0.990–1.005)	0.994 (0.991–0.997)	0.996 (0.984–1.008)	0.996 (0.993–0.999)
3-d	0.992 (0.989–0.995)	0.998 (0.990–1.006)	0.994 (0.991–0.997)	0.993 (0.980–1.006)	0.996 (0.995–0.997)
Adjusting for nonsmoke $PM_{2.5}$					
1-d	0.991 (0.988–0.994)	0.997 (0.989–1.004)	0.992 (0.989–0.995)	0.996 (0.985–1.007)	0.995 (0.994–0.996)
2-d	0.989 (0.986–0.992)	0.994 (0.987–1.002)	0.990 (0.987–0.993)	0.996 (0.984–1.008)	0.994 (0.993–0.995)
3-d	0.989 (0.986–0.992)	0.994 (0.986–1.002)	0.990 (0.987–0.993)	0.993 (0.980–1.006)	0.993 (0.992–0.995)
Total $PM_{2.5}$					
1-d	1.005 (1.004–1.007)	1.010 (1.006–1.015)	1.008 (1.006–1.010)	1.001 (0.994–1.007)	1.006 (1.006–1.007)
2-d	1.005 (1.003–1.007)	1.010 (1.005–1.010)	1.008 (1.006–1.010)	0.999 (0.991–1.006)	1.006 (1.005–1.007)
3-d	1.005 (1.003–1.007)	1.010 (1.005–1.016)	1.008 (1.006–1.010)	0.995 (0.988–1.003)	1.006 (1.005–1.007)

Odds ratios and 95% CIs are presented. AF indicates atrial fibrillation; AMI, acute myocardial infarction; CVD, cardiovascular disease; ED, emergency department; HF, heart failure; OR, odds ratio; and $PM_{2.5}$, fine particulate matter with a diameter ≤2.5 μm. ORs correspond to every 10 μg/m³ increase in $PM_{2.5}$. All the models were adjusted for daily relative humidity, average temperature, and day of the year. Exposure and covariates were modeled as the value on the day of ED visit, and 2- and 3-d moving averages leading up to and including the day of the ED visit.

all cardiovascular outcomes. Adjusting for nonsmoke $PM_{2.5}$ in addition did not change the results significantly. Higher 1-, 2-, and 3-day total $PM_{2.5}$ were associated with higher risks of ED visits for all cardiovascular outcomes (Table S1). Sex minimally modified the association between smoke $PM_{2.5}$ and HF, but not for AF, AMI, stroke, and CVD. Similar to the results in the total population, higher total $PM_{2.5}$ was associated with a marginally elevated risk of ED visits for all cardiovascular outcomes except stroke among both men and women (Table S2).

Ratio of Smoke $PM_{2.5}$ and Total $PM_{2.5}$ With CVD ED Visits

In the model where the ratio of smoke $PM_{2.5}$ to total $PM_{2.5}$ was modeled as a continuous variable, a 10% increase in the ratio was related to a slightly lower risk of ED visits for AF (1-day OR, 0.998 [95% CI, 0.997–0.997]), HF (1-day OR, 0.997 [95% CI, 0.996–0.998]), and CVD (1-day OR, 0.999 [95% CI, 0.998–0.999]). No significant association was found between the ratio and the risk of ED visits for AMI and stroke. The quartiles of the ratio were not consistently associated with any of the outcomes across models using different days of exposure (Table S3).

Wildfire Smoke $PM_{2.5}$ and Primary CVD ED Visits

A 10-$\mu g/m^3$ higher smoke $PM_{2.5}$ in the model of 1-, 2-, and 3-day averages was associated with slightly lower risks of HF and total CVD but not with the risks of AF, AMI, and stroke consistently. Further adjusting for nonsmoke did not modify the results. Increases in total $PM_{2.5}$ were associated with higher odds of all the primary cardiovascular end points, although statistical significance was observed only for total CVD (Table S4).

Seven-Day Wildfire Smoke $PM_{2.5}$ and CVD ED Visits

Consistent results were observed between 7-day moving average smoke $PM_{2.5}$ and cardiovascular outcomes, aligning with the findings from the 1-, 2-, and 3-day windows. Further adjusting for nonsmoke $PM_{2.5}$ did not change the association. Increased total $PM_{2.5}$ was associated with higher odds of all the outcomes except for stroke, for which slightly decreased odds were observed (OR, 0.987 [95% CI, 0.978–0.997]; Table S5).

DISCUSSION

This study explored the association between short-term $PM_{2.5}$ from wildland fires and ED visits for CVDs in 5 states in the western United States from 2007 to 2018. With a large sample size of >49 million, we were able to detect very small effect sizes. Consistent with prior research, total $PM_{2.5}$ was associated with an increased risk of ED visits for CVD.[14,35] Unexpectedly, though, we found small but statistically significant reductions in risk of ED visits for CVD associated with higher exposure to smoke $PM_{2.5}$. The results were not substantially different across different sex and age (<65 and ≥65 years) groups.

Current evidence from epidemiological or environmental exposure studies regarding the links between short-term smoke $PM_{2.5}$ and CVDs is inconsistent but in general does not support increased risk of CVD with greater short-term exposure to smoke $PM_{2.5}$. A recent review of the impact of wildfires on cardiovascular health highlighted that the existing evidence linking CVD with $PM_{2.5}$ and other air pollutants is mixed.[36] In a US study from 2008 to 2010, increases in $PM_{2.5}$ in days with wildfire smoke exposure (smoke days) were similarly associated with all-cause cardiovascular hospitalizations as similar increases in non–wildfire smoke days.[37] A similar study in Colorado reported null results regarding the link between smoke $PM_{2.5}$ and any CVD (OR, 0.998 [95% CI, 0.984–1.011]) or specific types of CVD including AMI, HF, dysrhythmia, ischemic heart disease, and peripheral or cerebrovascular disease, with small effect sizes.[27] In a study investigating source-apportioned $PM_{2.5}$ and ED visits for CVD in Atlanta, Georgia, biomass burning $PM_{2.5}$ with lag 0 did not present a significant effect on the risk of CVD ED visits; total ambient $PM_{2.5}$ with lag 0 was not associated with most cardiovascular outcomes except for ischemic stroke.[38] During the wildfire period in Canada, no increased risk of physician visits for CVD was observed related to an elevated 2-day $PM_{2.5}$, but in the post-wildfire period, an increased risk of physician visits for HF (11% [95% CI, 3%–21%]) and ischemic heart disease (19% [95% CI, 7%–33%]) among seniors was found.[17] It has been reported that the wildfire-related $PM_{2.5}$ in California from 2015 to 2017 was unfavorably associated with a higher risk of out-of-hospital cardiac arrest across multiple lag days (2-day OR, 1.7 [95% CI, 1.18–2.13]).[39] A study in 204 US counties found short-term exposure to ambient $PM_{2.5}$ to be associated with an increased risk of CVD hospital admission rates without statistical significance.[40] However, the measurements of $PM_{2.5}$ were in 3 density categories, which reduced statistical power and precision. It is important to note that the cardiovascular associations with $PM_{2.5}$ vary depending on the duration of exposure (short term versus long term), the source of $PM_{2.5}$, and the air pollution regulations in the area where $PM_{2.5}$ levels are high. Furthermore, different study designs, air quality measurements and monitoring, lack of long-term follow-up, and neglect

of gaseous species and other hazardous pollutants also contribute to the controversial findings between smoke $PM_{2.5}$ and CVD ED visits.

Several factors may explain the lack of increased CVD risk associated with short-term exposure to smoke $PM_{2.5}$. First, the impact of smoke $PM_{2.5}$ within a short window of exposure might be too subtle to affect acute ED visits for CVD compared with ambient $PM_{2.5}$. While fire smoke is becoming a major contributor of $PM_{2.5}$ pollution in the United States, the estimated contribution was up to 25% of total $PM_{2.5}$ in the contiguous United States,[12] and the long-term effect of wildfire smoke $PM_{2.5}$ on CVD might be limited compared with ambient $PM_{2.5}$.[41] Smoke plumes are often localized and episodic, causing significant variations in $PM_{2.5}$ levels in short durations, while nonsmoke $PM_{2.5}$ is more widespread and persistent. The stable temporal and spatial patterns of nonsmoke $PM_{2.5}$ might have led to a broader and consistent population impact on CVD risk. In the case that the short-term total $PM_{2.5}$ serves as a proxy for long-term exposure, the observed impact of total $PM_{2.5}$ may be attributed to prolonged exposure. Moreover, the progression of each type of CVD is complex and multifactorial. The precipitating conditions involved in the hypothesized mechanisms of CVD may require a long time to develop, such as arterial occlusion and ischemic necrosis for AMI and stroke,[42] left ventricular diastolic or systolic dysfunction for HF,[43] and vulnerable atrial structural and functional substrates for AF.[44] The risk of acute CVD attributable to several days of exposure to wildfire smoke $PM_{2.5}$ may be diminutive compared with other well-established risk factors. Second, public health advisories and regulations during wildfire seasons and events and awareness of susceptibility to air pollution may mitigate the adverse health effects of smoke $PM_{2.5}$, as people might stay indoors or seek shelter during wildfires, thereby reducing their true exposure to high levels of smoke $PM_{2.5}$. Moreover, the pattern of behavior change might be different among younger and older adults.[45] Additionally, it was possible that people reduced health care–seeking behaviors during wildfire episodes, but the impact of these behavioral changes was not expected to be significant and bias the results in this study. The presence of elevated smoke $PM_{2.5}$ may not always correspond with visible smoke that would trigger behavioral avoidance in the general population. On most days during the study period, smoke $PM_{2.5}$ accounts for only <10% of total $PM_{2.5}$ (Figure [E]). The relatively small proportion influences both the visibility of the smoke and its overall contribution to total $PM_{2.5}$-related CVD ED visits risk. People are more likely to change their behaviors when the wildfire smoke is visible or olfactible on any given day. Moreover, CVD ED visits generally reflect more severe and acute cardiovascular events, while individuals are less likely to delay seeking care even during disruptive conditions. Third, although the case-crossover study design can naturally control for confounding by non–time-dependent factors, time-varying factors and other behaviors subject to change might still confound the associations. For example, some people might stop engaging in outdoor physical activities and stay indoors during the wildfire episode. Changes in behavioral risk factors in days with high smoke $PM_{2.5}$ could even explain the small but significant reduction in CVD risk with higher short-term smoke $PM_{2.5}$ exposure. Fourth, the presence of other air pollutants, such as CO, SO_2, and O_3, due to wildfires may contribute to the risk of acute CVD, but these were not fully accounted for in this study.

This study has several strengths. This study included a large number of ED visits for cardiovascular outcomes across 5 US states over 12 years, enabling us to detect the small effect of wildfire smoke $PM_{2.5}$. Additionally, the exposure was measured using a high-performance satellite-driven approach with high spatial resolution, offering complete coverage and greater accuracy than monitor-based data. However, several limitations should be acknowledged. First, ED visit data capture only acute CVD events. CVD events requiring long-term medical attention were not included. Second, the exposure and ED visit data were at the zip code level. Misclassification of the patient's true residential zip code and mismeasurement of the smoke $PM_{2.5}$ exposure may lead to information bias. Third, as mentioned above, residual time-dependent confounding was possible due to the case-crossover study design and unmeasured air pollutants.

CONCLUSIONS

In conclusion, our study examined the relationship between short-term wildfire $PM_{2.5}$ exposure and ED visits for CVD across 5 Western US states from 2007 to 2018. Although we found statistical significance between smoke $PM_{2.5}$ and total $PM_{2.5}$ with ED visits for some cardiovascular outcomes and total CVD, the effect sizes were small and their public health and clinical significance remains uncertain. Further studies with more advanced study designs and more accurate study measures are warranted to investigate the effect of wildfire smoke $PM_{2.5}$ on cardiovascular health.

ARTICLE INFORMATION

Received November 18, 2024; accepted July 28, 2025.

Affiliations

Department of Epidemiology, Rollins School of Public Health (L.L., A.A.), Gangarosa Department of Environmental Health, Rollins School of Public Health (W.W., Y.L.) and Department of Biostatistics, Rollins School of Public Health (H.H.C.), Emory University, Atlanta, GA.

Acknowledgments

We are grateful for the support of the health data sources listed in the following sentence and their contributing hospitals. The ED visits data used to produce this publication were acquired from the Arizona Department of Health Services; California Office of Statewide Planning and Development, now California Department of Health Care Access and Information; Nevada Division of Health Care Financing and Policy, released through the Center for Health Information Analysis of the University of Nevada, Las Vegas; Oregon Healthcare Enterprises, Inc., Apprise Health Insights, a subsidiary of the Oregon Association of Hospitals and Health Systems; and Utah Department of Health, Office of Health Care Statistics. The authors thank The Environmental Exposures and Emergency Department Visits Across the Nation (ENVISION) investigators for their enthusiastic collaboration and effort. The analysis, interpretation, conclusions, and views presented in this article are solely those of the authors and do not reflect the official positions or conclusions of the listed data sources. The authorization to release this information does not imply any endorsement of the study or its findings by these data sources. The data sources, along with their employees, officers, and agents, do not make any representations, warranties, or guarantees regarding the accuracy, completeness, timeliness, or suitability of the information presented here.

Sources of Funding

This work was supported by the National Institutes of Environmental Health Services under Award Number 1R01ES032140 (Dr Liu) and R01ES027892 (Dr Chang), and the American Heart Association under Award Number 23PRE1020888 (L. Li). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Disclosures

None.

Supplemental Material

Tables S1–S5

REFERENCES

1. Jones MW, Abatzoglou JT, Veraverbeke S, Andela N, Lasslop G, Forkel M, Smith AJP, Burton C, Betts RA, van der Werf GR, et al. Global and regional trends and drivers of fire under climate change. *Rev Geophys*. 2022;60:e2020RG000726. doi: 10.1029/2020RG000726
2. Ellis TM, Bowman DM, Jain P, Flannigan MD, Williamson GJ. Global increase in wildfire risk due to climate-driven declines in fuel moisture. *Glob Chang Biol*. 2022;28:1544–1559. doi: 10.1111/gcb.16006
3. Watts N, Amann M, Arnell N, Ayeb-Karlsson S, Belesova K, Boykoff M, Byass P, Cai W, Campbell-Lendrum D, Capstick S, et al. The 2019 report of the lancet countdown on health and climate change: ensuring that the health of a child born today is not defined by a changing climate. *Lancet*. 2019;394:1836–1878. doi: 10.1016/S0140-6736(19)32596-6
4. Prichard SJ, Hessburg PF, Hagmann RK, Povak NA, Dobrowski SZ, Hurteau MD, Kane VR, Keane RE, Kobziar LN, Kolden CA, et al. Adapting western North American forests to climate change and wildfires: 10 common questions. *Ecol Appl*. 2021;31:e02433. doi: 10.1002/eap.2433
5. Brown T, Leach S, Wachter B, Gardunio B. The extreme 2018 northern California fire season. *Bull Am Meteorol Soc*. 2020;101:S1–S4. doi: 10.1175/BAMS-D-19-0275.1
6. Burke M, Driscoll A, Heft-Neal S, Xue J, Burney J, Wara M. The changing risk and burden of wildfire in the United States. *Proc Natl Acad Sci*. 2021;118:e2011048118. doi: 10.1073/pnas.2011048118
7. Richardson D, Black AS, Irving D, Matear RJ, Monselesan DP, Risbey JS, Squire DT, Tozer CR. Global increase in wildfire potential from compound fire weather and drought. *Npj Clim Atmos Sci*. 2022;5:23. doi: 10.1038/s41612-022-00248-4
8. Westerling AL, Hidalgo HG, Cayan DR, Swetnam TW. Warming and earlier spring increase Western U.S. Forest wildfire activity. *Science*. 2006;313:940–943. doi: 10.1126/science.1128834
9. Zhang D, Wang W, Xi Y, Bi J, Hang Y, Zhu Q, Pu Q, Chang H, Liu Y. Wildland fires worsened population exposure to $PM_{2.5}$ pollution in the contiguous United States. *Environ Sci Technol*. 2023;57:19990–19998.
10. Liu JC, Pereira G, Uhl SA, Bravo MA, Bell ML. A systematic review of the physical health impacts from non-occupational exposure to wildfire smoke. *Environ Res*. 2015;136:120–132. doi: 10.1016/j.envres.2014.10.015
11. O'Dell K, Ford B, Fischer EV, Pierce JR. Contribution of wildland-fire smoke to US PM2.5 and its influence on recent trends. *Environ Sci Technol*. 2019;53:1797–1804. doi: 10.1021/acs.est.8b05430
12. Burke M, Childs ML, de la Cuesta B, Qiu M, Li J, Gould CF, Heft-Neal S, Wara M. The contribution of wildfire to PM2.5 trends in the USA. *Nature*. 2023;622:761–766. doi: 10.1038/s41586-023-06522-6
13. Erqou S, Clougherty JE, Olafiranye O, Magnani JW, Aiyer A, Tripathy S, Kinnee E, Kip KE, Reis SE. Particulate matter air pollution and racial differences in cardiovascular disease risk. *Arterioscler Thromb Vasc Biol*. 2018;38:935–942. doi: 10.1161/ATVBAHA.117.310305
14. Pun VC, Kazemiparkouhi F, Manjourides J, Suh HH. Long-term PM2.5 exposure and respiratory, cancer, and cardiovascular mortality in older US adults. *Am J Epidemiol*. 2017;186:961–969. doi: 10.1093/aje/kwx166
15. Pope Iii CA, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA*. 2002;287:1132–1141. doi: 10.1001/jama.287.9.1132
16. Hadley MB, Henderson SB, Brauer M, Vedanthan R. Protecting cardiovascular health from wildfire smoke. *Circulation*. 2022;146:788–801. doi: 10.1161/CIRCULATIONAHA.121.058058
17. Mahsin M, Cabaj J, Saini V. Respiratory and cardiovascular condition-related physician visits associated with wildfire smoke exposure in Calgary, Canada, in 2015: a population-based study. *Int J Epidemiol*. 2022;51:166–178. doi: 10.1093/ije/dyab206
18. Hahn MB, Kuiper G, O'Dell K, Fischer EV, Magzamen S. Wildfire smoke is associated with an increased risk of cardiorespiratory emergency department visits in Alaska. *GeoHealth*. 2021;5:e2020GH000349. doi: 10.1029/2020GH000349
19. Aguilera R, Corringham T, Gershunov A, Benmarhnia T. Wildfire smoke impacts respiratory health more than fine particles from other sources: observational evidence from Southern California. *Nat Commun*. 2021;12:1493. doi: 10.1038/s41467-021-21708-0
20. Martin SS, Aday AW, Almarzooq ZI, Anderson CAM, Arora P, Avery CL, Baker-Smith CM, Barone Gibbs B, Beaton AZ, Boehme AK, et al. 2024 heart disease and stroke statistics: A report of US and global data from the American Heart Association. *Circulation*. 2024;149:e347–e913. doi: 10.1161/CIR.0000000000001209
21. Murray CJ, Aravkin AY, Zheng P, Murray CJL, Abbafati C, Abbas KM, Abbasi-Kangevari M, Abd-Allah F, Abdelalim A, Abdollahi M, et al. Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the global burden of disease study 2019. *Lancet*. 2020;396:1223–1249. doi: 10.1016/S0140-6736(20)30752-2
22. Brook RD, Rajagopalan S, Pope CA, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F, Hong Y, Luepker RV, Mittleman MA, et al. Particulate matter air pollution and cardiovascular disease. *Circulation*. 2010;121:2331–2378. doi: 10.1161/cir.0b013e3181dbece1
23. Rajagopalan S, Al-Kindi SG, Brook RD. Air pollution and cardiovascular disease. *J Am Coll Cardiol*. 2018;72:2054–2070. doi: 10.1016/j.jacc.2018.07.099
24. Rajagopalan S, Brauer M, Bhatnagar A, Bhatt DL, Brook JR, Huang W, Münzel T, Newby D, Siegel J, Brook RD, et al. Personal-level protective actions against particulate matter air pollution exposure: a scientific statement from the American Heart Association. *Circulation*. 2020;142:e411–e431. doi: 10.1161/CIR.0000000000000931
25. Bhatnagar A. Cardiovascular pathophysiology of environmental pollutants. *Am J Phys Heart Circ Phys*. 2004;286:H479–H485. doi: 10.1152/ajpheart.00817.2003
26. Heaney A, Stowell JD, Liu JC, Basu R, Marlier M, Kinney P. Impacts of fine particulate matter from wildfire smoke on respiratory and cardiovascular health in California. *GeoHealth*. 2022;6:e2021GH000578. doi: 10.1029/2021GH000578
27. Stowell JD, Geng G, Saikawa E, Chang HH, Fu J, Yang CE, Zhu Q, Liu Y, Strickland MJ. Associations of wildfire smoke PM2.5 exposure with cardiorespiratory events in Colorado 2011–2014. *Environ Int*. 2019;133:105151. doi: 10.1016/j.envint.2019.105151
28. Chen G, Guo Y, Yue X, Tong S, Gasparrini A, Bell ML, Armstrong B, Schwartz J, Jaakkola JJK, Zanobetti A, et al. Mortality risk attributable to wildfire-related PM2.5 pollution: a global time series study in 749 locations. *Lancet Planet Health*. 2021;5:e579–e587. doi: 10.1016/S2542-5196(21)00200-X

29. Zhang D, Wang W, Xi Y, Bi J, Hang Y, Zhu Q, Pu Q, Chang H, Liu Y. Wildland fires worsened population exposure to PM(2.5) pollution in the contiguous United States. *Environ Sci Technol*. 2023;57:19990–19998. doi: 10.1021/acs.est.3c05143
30. Lawrence MG. The relationship between relative humidity and the dewpoint temperature in moist air: a simple conversion and applications. *Bull Am Meteorol Soc*. 2005;86:225–234. doi: 10.1175/BAMS-86-2-225
31. Thornton M, Shrestha R, Wei Y, Thornton P, Kao S, Wilson B. *Daymet: Daily Surface Weather Data on a 1-km Grid for North America, Version 4 R1*. Oak Ridge, Tennessee, USA: ORNL DAAC; 2022.
32. Maclure M. The case-crossover design: A method for studying transient effects on the risk of acute events. *Am J Epidemiol*. 1991;133:144–153. doi: 10.1093/oxfordjournals.aje.a115853
33. Levy D, Lumley T, Sheppard L, Kaufman J, Checkoway H. Referent selection in case-crossover analyses of acute health effects of air pollution. *Epidemiology*. 2001;12:186–192. doi: 10.1097/00001648-200103000-00010
34. Janes H, Sheppard L, Lumley T. Case-crossover analyses of air pollution exposure data: referent selection strategies and their implications for bias. *Epidemiology*. 2005;16:717–726. doi: 10.1097/01.ede.0000181315.18836.9d
35. Krittanawong C, Qadeer YK, Hayes RB, Wang Z, Virani S, Thurston GD, Lavie CJ. PM2.5 and cardiovascular health risks. *Curr Probl Cardiol*. 2023;48:101670. doi: 10.1016/j.cpcardiol.2023.101670
36. Williams VA, Perreault LR, Yazbeck CT, Micovic NA, Oakes JM, Bellini C. Impact of wildfires on cardiovascular health. *Circ Res*. 2024;134:1061–1082. doi: 10.1161/CIRCRESAHA.124.323614
37. DeFlorio-Barker S, Crooks J, Reyes J, Rappold AG. 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*. 2019;127:037006. doi: 10.1289/EHP3860
38. Pennington AF, Strickland MJ, Gass K, Klein M, Sarnat SE, Tolbert PE, Balachandran S, Chang HH, Russell AG, Mulholland JA, et al. Source-apportioned PM2.5 and cardiorespiratory emergency department visits: accounting for source contribution uncertainty. *Epidemiology*. 2019;30:789–798. doi: 10.1097/EDE.0000000000001089
39. Jones CG, Rappold AG, Vargo J, Cascio WE, Kharrazi M, McNally B, Hoshiko S; with the CARES Surveillance Group. Out-of-hospital cardiac arrests and wildfire-related particulate matter during 2015–2017 California wildfires. *J Am Heart Assoc*. 2020;9:e014125. doi: 10.1161/JAHA.119.014125
40. Dominici F, Peng RD, Bell ML, Pham L, McDermott A, Zeger SL, Samet JM. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *JAMA*. 2006;295:1127–1134. doi: 10.1001/jama.295.10.1127
41. Gao Y, Huang W, Xu R, Gasevic D, Liu Y, Yu W, Yu P, Yue X, Zhou G, Zhang Y, et al. Association between long-term exposure to wildfire-related PM2.5 and mortality: A longitudinal analysis of the UK biobank. *J Hazard Mater*. 2023;457:131779. doi: 10.1016/j.jhazmat.2023.131779
42. Widimský P, Kožnar B, Vaško P, Peisker T, Štětkářová I. Acute myocardial infarction and acute stroke: what are the differences? Focus on reperfusion therapy. *Cor Vasa*. 2013;55:e111–e116. doi: 10.1016/j.crvasa.2013.02.002
43. Arrigo M, Jessup M, Mullens W, Reza N, Shah AM, Sliwa K, Mebazaa A. Acute heart failure. *Nat Rev Dis Primers*. 2020;6:16. doi: 10.1038/s41572-020-0151-7
44. Nattel S, Burstein B, Dobrev D. Atrial remodeling and atrial fibrillation. *Circ Arrhythm Electrophysiol*. 2008;1:62–73. doi: 10.1161/CIRCEP.107.754564
45. Laumbach R, Meng Q, Kipen H. What can individuals do to reduce personal health risks from air pollution? *J Thorac Dis*. 2015;7:96–107. doi: 10.3978/j.issn.2072-1439.2014.12.21