CORONAVIRUS

Excess of COVID-19 cases and deaths due to fine particulate matter exposure during the 2020 wildfires in the United States

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The year 2020 brought unimaginable challenges in public health, with the confluence of the COVID-19 pandemic and wildfires across the western United States. Wildfires produce high levels of fine particulate matter ($PM_{2.5}$). Recent studies reported that short-term exposure to $PM_{2.5}$ is associated with increased risk of COVID-19 cases and deaths. We acquired and linked publicly available daily data on $PM_{2.5}$, the number of COVID-19 cases and deaths, and other confounders for 92 western U.S. counties that were affected by the 2020 wildfires. We estimated the association between short-term exposure to $PM_{2.5}$ during the wildfires and the epidemiological dynamics of COVID-19 cases and deaths. We adjusted for several time-varying confounding factors (e.g., weather, seasonality, long-term trends, mobility, and population size). We found strong evidence that wildfires amplified the effect of short-term exposure to $PM_{2.5}$ on COVID-19 cases and deaths, although with substantial heterogeneity across counties.

INTRODUCTION

According to the National Interagency Fire Center, approximately 7 million acres of land burn every year in the United States (*1*). As of December 2020, more than 10 million acres were burnt in the western United States alone. In 2020, California and Washington both recorded their largest wildfires in state history (*1*, *2*). The warming climate is expected to increase wildfire risk and, consequently, exposure to smoke (*3*, *4*). In the last 4 years, the United States has experienced record-breaking wildfires, leading to an increase of more than 470,000 daily exposures per year and 1.85 billion more person-days of exposure to high wildfire risk compared to 2001–2004 (*5*). Wildfire smoke contains high levels of fine particulate matter ($PM_{2.5}$) (*4*), the pollutant in smoke that poses the greatest risk to health (*2*, *6*). Short-term exposure to $PM_{2.5}$ is associated with adverse health outcomes (*6*–*9*). According to recent research by Burke *et al.* (*2*), wildfires contribute to up to 25% of the $PM_{2.5}$ concentration in the atmosphere in the United States and up to half of $PM_{2.5}$ in some regions of the western United States.

Exposure to $PM_{2.5}$, specifically from wildfires, has been associated with negative health outcomes (*3*, *4*, *10*–*16*), including all-cause mortality and respiratory morbidity, as well as asthma, chronic obstructive pulmonary diseases, and others (*11*, *12*, *17*, *18*). In particular, studies have shown that short-term wildfire-specific $PM_{2.5}$ exposure is linked to increases in asthma symptoms, emergency department visits for respiratory symptoms, and respiratory hospital admissions, as well as increases in risk and severity of respiratory viral infections (*4*, *19*–*21*). Certain populations are at higher risk from exposure to $PM_{2.5}$ from wildfires, including people with heart or lung disease, the elderly, children, and fetuses (*11*, *18*, *19*, *22*).

Between March and December 2020, the western United States was afflicted by two natural disasters: wildfires burning through millions of acres and the coronavirus disease 2019 (COVID-19) pandemic. Recent studies have provided preliminary evidence of an association between short- and long-term exposure to $PM_{2.5}$ and COVID-19 health outcomes [see, for example, (*23*–*25*)]. A study by Pozzer *et al.* (*25*) estimated that 17% of COVID-19 mortality in North America could be attributed to exposure to particulate air pollution. Another study by Wu *et al.* (*23*) found that only an increase of 1 $\mu g/m^3$ in the long-term average $PM_{2.5}$ concentration is associated with an 11% increase in COVID-19 mortality. The U.S. Centers for Disease Control and Prevention (CDC) (*26*) states that "wildfire smoke can irritate your lungs, cause inflammation, affect your immune system, and make you more prone to lung infections, including COVID-19." Henderson (*27*) urged greater recognition of the potential for a dangerous interaction between SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2; the virus that causes COVID-19) and smoke pollution. Regardless of the clear threat, no study to date has quantified the degree to which the increases in $PM_{2.5}$ during the 2020 wildfires exacerbated the severity of the COVID-19 pandemic in the United States in terms of excess cases and deaths.

Supported by biological plausibility (*28*, *29*), we hypothesize that short-term exposure to $PM_{2.5}$ might increase the likelihood of (i) more severe infection so that an asymptomatic infection becomes symptomatic and is detected as a case and (ii) more severe infection that leads to death. To gather evidence for these hypotheses, we acquired, harmonized, linked, and analyzed publicly available daily time series data for 92 counties in the states of California, Washington, and Oregon, where most of the wildfires between 15 March and 16 December 2020 occurred. Our goal was to quantify the potential association between short-term exposure to $PM_{2.5}$ during the wildfires and the epidemiological dynamics of COVID-19 cases and deaths. More specifically, we estimated the percentage increase in COVID-19 cases and deaths associated with a daily increase of 10 $\mu g/m^3$ in $PM_{2.5}$ for 28 days for each county and pooled across all counties. We also conducted sensitivity analyses using 14 and 21 lag days. We also estimated the percentage of COVID-19 cases and deaths

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attributable to exposure to high levels of $PM_{2.5}$ during the 2020 wildfires for each county. In addition to analyzing the distributed lag effects using 14 and 21 lag days, we also evaluated several sensitivity analyses with respect to confounding adjustment for seasonality, long-term trends, weather, and mobility. All data and software are publicly available at https://github.com/xiaodan-zhou/covid_wildfire. In addition, infographics with detailed results for each county are available at https://analysis-1.maps.arcgis.com/apps/dashboards/c0df43781aeb4085954676f4e9ca9bf9.

RESULTS

We assembled a multisite time series study of 133 counties in three states (California, Washington, and Oregon) for the period from 15 March to 16 December 2020 (a total of 277 days). We excluded from the analysis 41 counties that had missing $PM_{2.5}$ daily values during wildfire days in the study period, as it would not be appropriate to impute these missing values using historical data. The remaining 92 counties cover 95.1% of population of the three states (48.8 million), with a total of 25,484 daily records at the county level.

Definition of a wildfire day

In National Oceanic and Atmospheric Administration's Hazard Mapping System (HMS), human analysts use the Geostationary Operational Environmental Satellite (GOES) images to delineate smoke-affected areas and to qualitatively categorize each polygon as light, medium, or heavy smoke based on visual inspection of the apparent opacity (*30*–*32*). The smoke categories roughly correspond to $PM_{2.5}$ ranges based on the now discontinued GOES Aerosol Smoke Product: light (0 to 10 $\mu g/m^3$), medium (10 to 21 $\mu g/m^3$), and heavy (21 to 32 $\mu g/m^3$). Because light and medium smoke often reflect smoke aloft rather than at the surface (section S1.3), we used only the heavy smoke category to differentiate wildfire from non-wildfire days for a given location. Any day that did not satisfy this definition was defined as a non-wildfire day. Figure 1 shows the 92 counties that were included in the analysis for each of the three states. The color code denotes the percentage of wildfire days during the study period, which ranged from 3 to 29%.

Table 1 summarizes the daily $PM_{2.5}$ level and the number of COVID-19 cases and deaths on wildfire and non-wildfire days during 15 August and 15 October 2020, when 88% of the wildfires occurred. The daily levels of $PM_{2.5}$ during wildfire days was higher than those on non-wildfire days, with a median of 31.2 versus 6.4 $\mu g/m^3$. In some counties, the levels of $PM_{2.5}$ on wildfire days reached extremely high levels. For example, from 14 to 17 September 2020, Mono County, CA, experienced four sequential days with $PM_{2.5}$ levels higher than 500 $\mu g/m^3$ as a result of the Creek Fire. The daily COVID-19 case rate and the daily COVID-19 death rate are higher on wildfire days compared to non-wildfire days.

Daily increase in $PM_{2.5}$ attributable to a wildfire day

For each wildfire day, in each county, we estimated the increase in the daily level of $PM_{2.5}$ attributable to wildfires by implementing a counterfactual analysis (*33*). More specifically, we assumed that the factual time series data are the observed county-specific daily levels of $PM_{2.5}$. For wildfire day and for each county, we estimated the counterfactual value, that is, the level of $PM_{2.5}$ that would have occurred for the same day and the same county under the hypothetical scenario in which a wildfire did not occur. We estimated these counterfactual values by taking the median of the daily levels of $PM_{2.5}$ on the same day of the year and on the same county for the previous years of 2016–2019. For each county, we calculated the daily increase in $PM_{2.5}$ attributable to wildfires as the difference between the daily observed value and the daily counterfactual value. Figure S1 shows an example of this calculation for a single time series for Los Angeles County, CA.

Figure 2 shows the daily time series data for the daily levels of $PM_{2.5}$ (blue), daily number of COVID-19 cases (red), and daily number of COVID-19 deaths (black) for the six most populated counties. The orange vertical bars in the $PM_{2.5}$ time series denote daily increases in $PM_{2.5}$ attributable to wildfire days. Figure S2 shows boxplots of the distribution of weekly $PM_{2.5}$ levels ($\mu g/m^3$) across counties, separately for each state during the study period. We also show the distribution of $PM_{2.5}$ for the same weeks and the same counties but averaged across previous years (2016–2019). The distribution of the weekly $PM_{2.5}$ averages for the year 2020 and for the years 2016–2019 were similar, except for the period from 17 August to 5 October 2020, in California, and from 1 to 21 September 2020, in Oregon and Washington. These are the periods when wildfires mostly occurred in these states.

The statistical analysis of the multisite time series data must account for several challenges apparent in the data: (i) Many counties

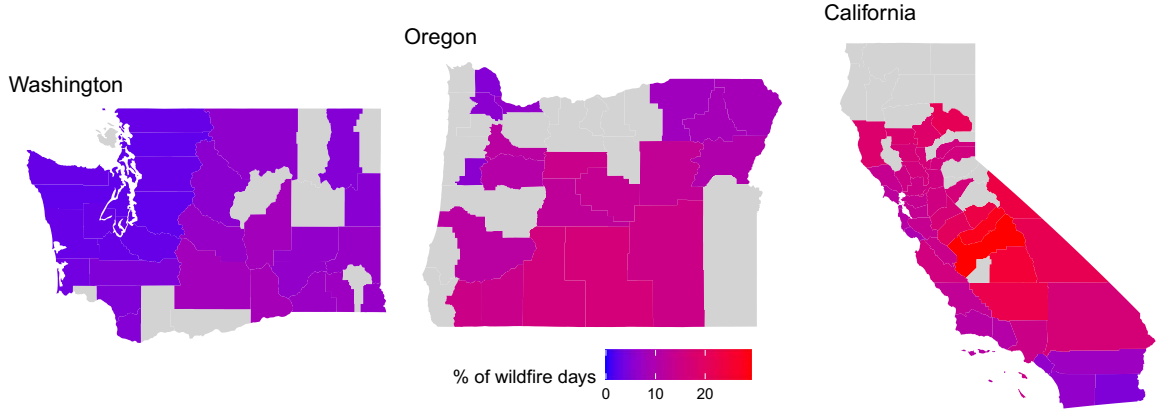

Fig. 1. Maps of the 92 counties included in the analysis. The color code denotes the percentage of wildfire days during the study period (15 March to 16 December 2020). Counties in gray were excluded from the analysis. The percentage of wildfire days ranges from 3 to 29%.

Table 1. Summary of COVID-19 cases, deaths, and $PM_{2.5}$ for 92 counties in the western United States during the wildfire season from 15 August to 15 October 2020 (61 days). We report the median, minimum, and maximum among the 92 counties. A day is defined as a wildfire day if the daily levels of smoke density on that day are higher than 21 μg/m^3.

	Total	Wildfire days	Non-wildfire days
Number of days	61	28 (7, 56)	33 (5, 54)
Daily $PM_{2.5}$ (μg/m^3)	11.7 (0.0, 696.7)	31.2 (1.5, 696.7)	6.4 (0.0, 322.6)
Days with zero COVID-19 cases (%)	24 (2, 97)	24 (0, 100)	22 (0, 97)
Days with zero COVID-19 deaths (%)	84 (5, 100)	85 (9, 100)	83 (0, 100)
Daily COVID-19 case rate (per 100,000)	7.5 (0.4, 47.9)	8.0 (0.0, 44.3)	6.9 (0.5, 49.3)
Daily COVID-19 death rate (per 1,000,000)	1.12 (0.00, 12.01)	1.23 (0.00, 11.09)	1.00 (0.00, 16.63)

have several days with a zero count of COVID-19 cases (or deaths) (zero inflation) (see Table 1); (ii) because of the SARS-CoV-2 incubation period, it is expected that a change in COVID-19 cases and deaths might occur up to at most 4 weeks after exposure to $PM_{2.5}$ (delayed effects); (iii) the relationship between daily exposure to $PM_{2.5}$ and COVID-19 cases (or deaths) is expected to be heterogeneous across counties due to differences in the stage of the pandemic, testing practices, and vulnerability of the population (heterogeneity); and (iv) we must control for potential nonlinear confounding effects of both measured confounders (e.g., weather and mobility) and unmeasured confounders (e.g., seasonality and trend). To overcome these challenges, we developed and implemented a Bayesian hierarchical zero-inflated negative binomial distributed lag (BH-ZINB-DL) model to estimate the association between daily changes in $PM_{2.5}$ and the percentage increase in the risk of COVID-19 cases and deaths up to 28 days after exposure. A distributed lag model is a popular approach for the analysis of time series data and has been implemented previously to estimate the association between an increase in exposure to air pollution on a given day and the percentage increase in the risk of a health outcome on the same day (lag 0), the day after (lag 1), and up to several days after exposure (*34*, *35*).

For each county, we estimated the following parameters: (i) lag-specific effects, defined as the percentage increase in COVID-19 cases (or deaths) associated with a daily increase of 10 μg/m^3 in $PM_{2.5}$ separately for each lag, and (ii) cumulative effects across all lags, the percentage increase in COVID-19 cases (or deaths) associated with a daily increase of 10 μg/m^3 in $PM_{2.5}$ for 28 subsequent days, cumulatively at 4 weeks. We also estimated the percentage of the total number of COVID-19 cases (or deaths) attributable to the daily increases in $PM_{2.5}$ levels on wildfire days. Table S1 summarizes the parameters of interest and their mathematical formulations. We fit the same model for COVID-19 cases and COVID-19 deaths. The statistical model is described in greater detail in Materials and Methods.

Figure 3 shows the posterior distributions of the county-specific cumulative effects at 4 weeks, representing the percentage increase in COVID-19 cases and deaths associated with a daily increase of 10 μg/m^3 in $PM_{2.5}$ for 28 subsequent days. The posterior distribution of the same cumulative effect, but pooled across all the counties, is shown in the red boxplot. We highlight an association when the 95% credible intervals (CIs) do not include 0. For COVID-19 cases, we found that 52 of 92 counties had strong evidence of a positive association between exposure to $PM_{2.5}$ and increased risk of COVID-19 cases 4 weeks later. Pooled across counties, we found that a daily increase of 10 μg/m^3 in $PM_{2.5}$ for 28 subsequent days was associated with a 11.7% (95% CI, 8.2 to 16.0) increase in COVID-19 cases. The counties of Sonoma, CA, and Whitman, WA, had the largest effect: We found that a daily increase of 10 μg/m^3 in $PM_{2.5}$ for 28 subsequent days was associated with a 65.3% (95% CI, 41.9 to 88.2) and 71.6% (95% CI, 47.5 to 94.8) increase in COVID-19 cases, respectively. For COVID-19 deaths, we found that 17 of 92 counties had strong evidence of a positive association between exposure to $PM_{2.5}$ and increased risk of COVID-19 death 4 weeks later. While pooling the distributed lag effects across counties, we found that a daily increase of 10 μg/m^3 in $PM_{2.5}$ for 28 subsequent days was associated with a 8.4% (95% CI, 2.1 to 15.3) increase in COVID-19 deaths. Calaveras, CA, and San Bernardino, CA, had the largest effect: We found that a daily increase of 10 μg/m^3 in $PM_{2.5}$ for 28 subsequent days was associated with a 52.8% (95% CI, 18.4 to 87.0) and 65.9% (95% CI, 22.8 to 105.3) increase in COVID-19 deaths, respectively. The orange triangles represent the percentage of wildfire days in that county. Results for the cumulative effects at 3 and 2 weeks are shown in figs. S6 and S9. Note that we also found strong evidence of heterogeneity in these associations. We identify a negative association for six and three counties for COVID-19 cases and deaths, respectively.

Figure S3 shows the posterior distributions of the lag-specific effects pooled across all counties, representing the percentage increase in COVID-19 cases and deaths associated with a daily increase of 10 μg/m^3 in $PM_{2.5}$ separately for each lag. Results for the cumulative effects at 3 and 2 weeks are shown in figs. S8 and S11, respectively.

Figures S4 and S5 show the percentage of total COVID-19 cases and death, respectively, attributable to observed high levels of $PM_{2.5}$ on wildfire days in the states of Washington, Oregon, and California. Figure 4 shows the posterior distributions of the percentage of total COVID-19 cases and deaths attributable to the higher than expected levels of $PM_{2.5}$ on wildfire days for each county. These posterior distributions take into account of the strength of the evidence regarding the association between short-term exposure to $PM_{2.5}$ and COVID-19 cases and deaths for each county (see Fig. 3) and increases in $PM_{2.5}$ attributable to wildfires (see fig. S1). The counties of Butte, CA, and Whitman, WA, showed the largest effect on COVID-19 cases: We found that the percentage of total COVID-19 cases attributable to high levels of $PM_{2.5}$ on wildfire days was 17.3 (95% CI, 13.9 to 20.7) and 18.2 (95% CI, 14.6 to 21.2), respectively. The counties of Butte, CA, and Calaveras, CA, showed the largest effect on COVID-19 deaths: The percentage of total COVID-19 deaths attributable to high levels of $PM_{2.5}$ on wildfire days was 41.0 (95% CI, 24.6 to 56.7) and 137.4 (95% CI, 62.2 to 212.9), respectively. The overall number of COVID-19 cases and deaths attributable to daily increase in $PM_{2.5}$ from wildfires is 19,742 (95% CI, 7062 to 31,310) and 748 (95% CI, 398 to 1102), respectively. Results for

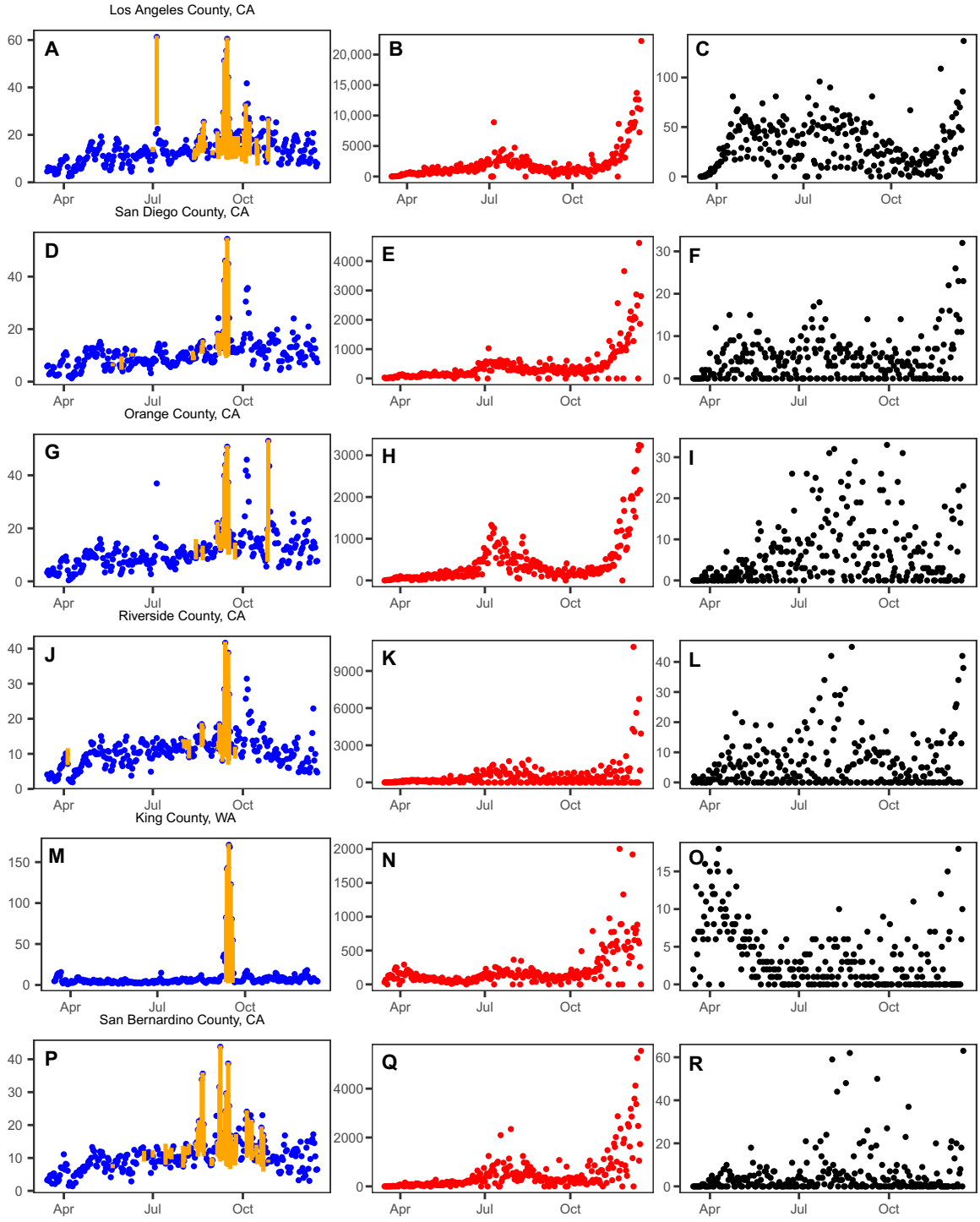

Fig. 2. Time series of $PM_{2.5}$ levels, COVID-19 positive cases and deaths in the six most populated counties in our analysis. Daily $PM_{2.5}$ levels ($\mu g/m^3$, in blue) (**A**, **D**, **G**, **J**, **M**, and **P**), daily COVID-19–positive cases (in red) (**B**, **E**, **H**, **K**, **N**, and **Q**), and daily COVID-19 deaths (in black) (**C**, **F**, **I**, **L**, **O**, and **R**). Data are shown for the top six counties in terms of population size and for the study period (15 March to 16 December 2020). Orange vertical bars [in (A), (D), (G), (J), (M), and (P)] denote daily increases in $PM_{2.5}$ attributable to each wildfire day. Data visualization for all the counties is available at https://analysis-1.maps.arcgis.com/apps/dashboards/c0df43781aeb4085954676f4e9ca9bf9.

cumulative effects up to 3 and 2 weeks after $PM_{2.5}$ exposure are reported in figs. S7 and S10, respectively.

Figure 5 summarizes the results of our comprehensive sensitivity analysis. We analyzed the posterior distribution of the cumulative effects and pooled across the counties in our original analysis (A) compared to the following scenarios: B, decreasing the number of lag days under consideration from 4 to 3 weeks along with a decrease in number of spline bases for approximating the distributed

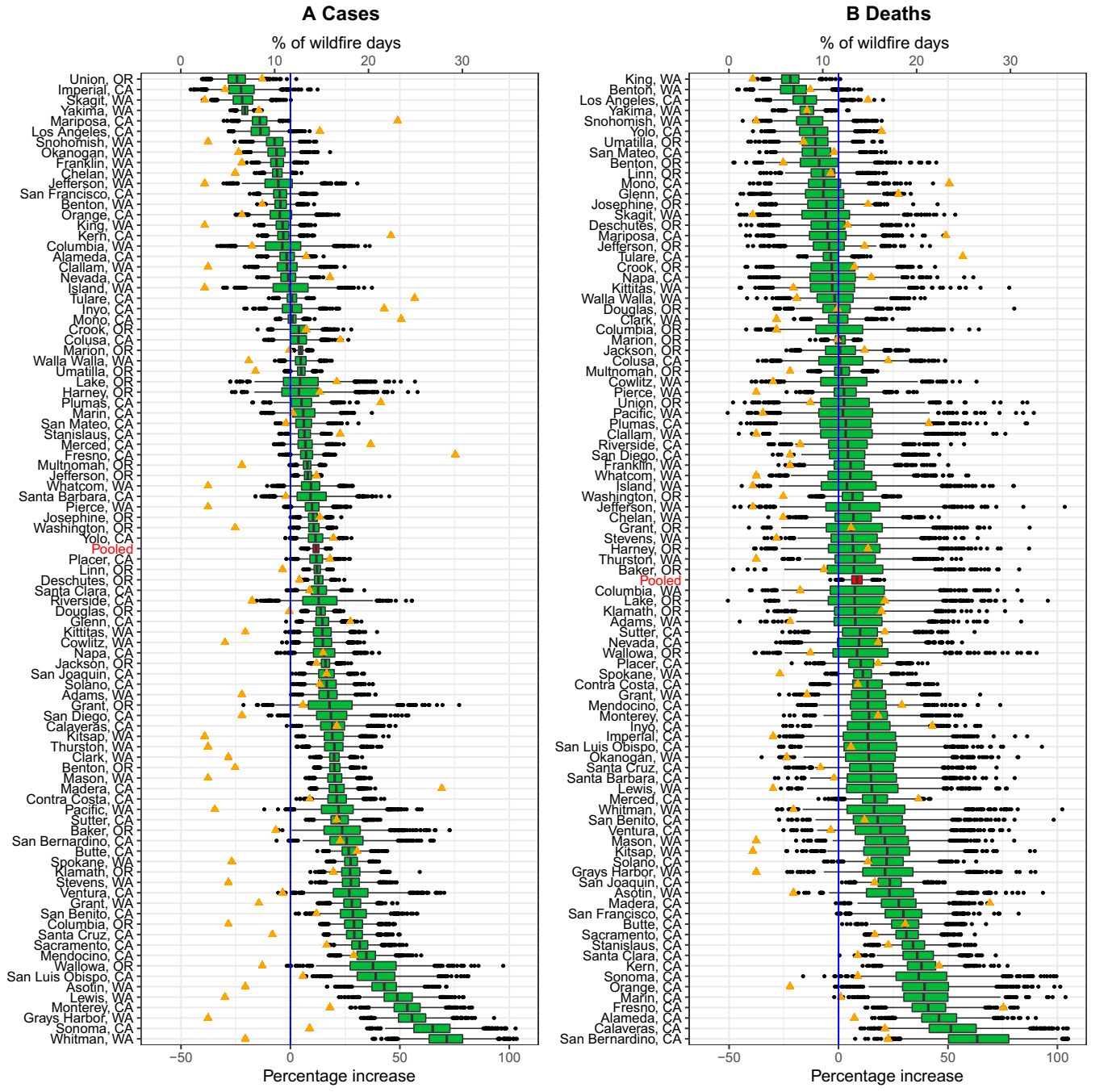

Fig. 3. Cumulative effects of $PM_{2.5}$ exposure up to 4 weeks for each county, and pooled across the counties for COVID-19 cases and COVID-19 deaths. The percentage increase in COVID-19 cases (**A**) and COVID-19 deaths (**B**) associated with a daily increase of 10 $\mu g/m^3$ in $PM_{2.5}$ for 28 subsequent days, cumulatively at 4 weeks. Orange triangles represent the percentage of wildfire days (out of 277 days in the study period) for each county.

lag function from six to five; C, decreasing the number of lag days in consideration to 2 weeks along with a decrease in the number of natural spline basis functions to four; D, adjusting for mobility data, which consequently omits six counties; E, increasing the number of spline basis functions for the distributed lag function from six to eight; F, more aggressively adjusting for temperature, humidity, and seasonality trends; G, omitting Calaveras, CA, and Mono, CA, counties; and H, dropping adjustments for the day of week. Results were consistent across the scenarios. However, we found smaller effects when we reduced the analysis to 21 lags (3 weeks, scenario B) and was further reduced when we used 14 lags (2 weeks, scenario C). We found slightly attenuated effects when we adjust for mobility data (scenario D). The posterior probabilities that the cumulative effect is positive are always larger than 0.8 across every sensitivity scenario.

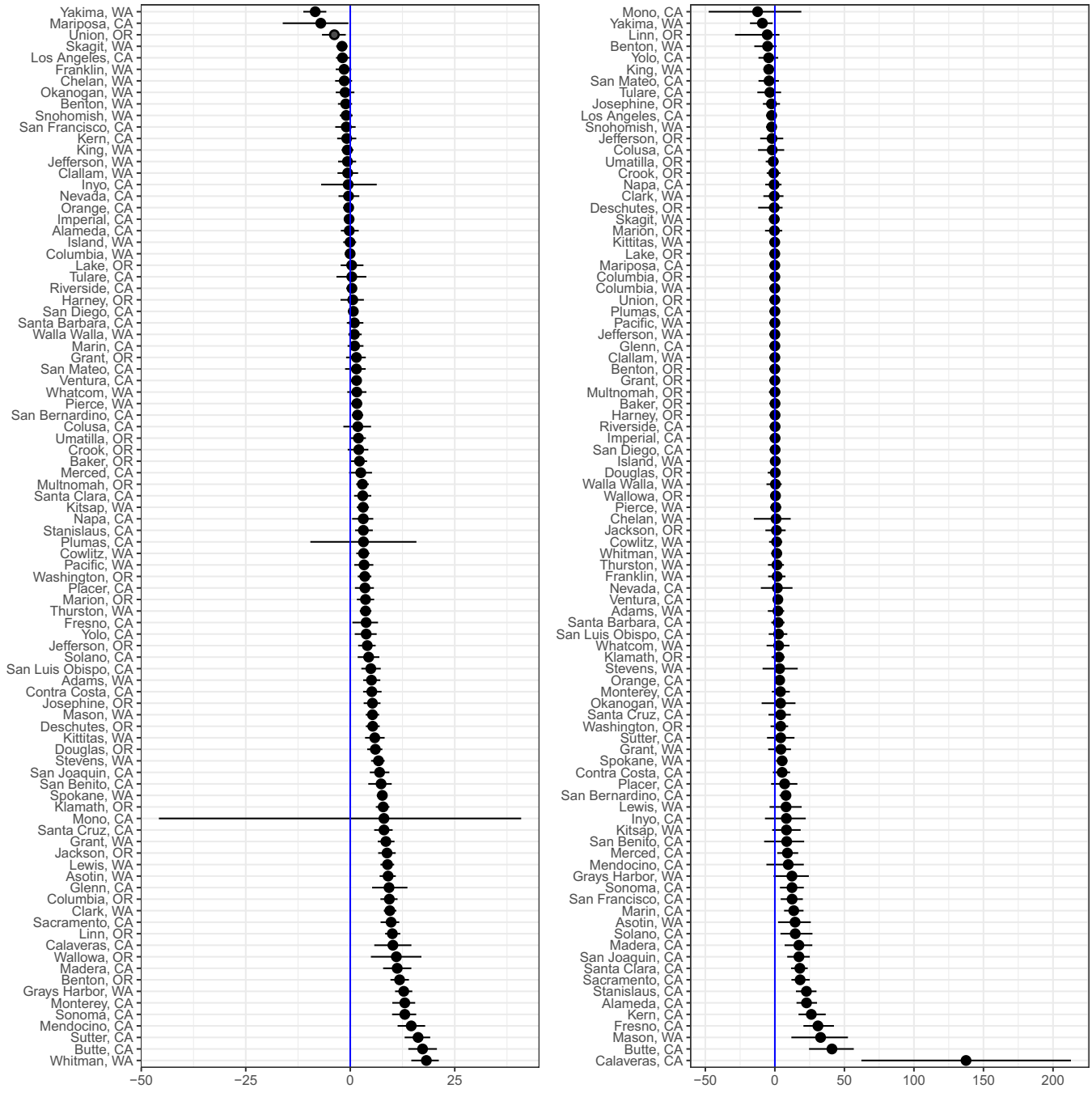

Fig. 4. Point estimate and 95% CI of the percentage of total COVID-19 cases and deaths attributable to high levels of $PM_{2.5}$ on wildfire days during the period from 15 March to 26 November 2020. Error bars represent 95% CIs.

This suggests that, overall, there is strong evidence of an association between short-term exposure to $PM_{2.5}$ and the dynamic of COVID-19 cases and deaths (see table S3).

DISCUSSION

We estimated the association between daily changes in levels of $PM_{2.5}$ and the percentage increase in COVID-19 cases and deaths for the counties in the western United States that were affected by the 2020 wildfires. In addition, we estimated the percentage of the total number of COVID-19 cases and deaths that were attributable to exposure to high levels of $PM_{2.5}$ during the wildfires for each of the counties.

While pooling across all counties, we found strong evidence of a positive associations between daily increases in $PM_{2.5}$ and increased risks of COVID-19 cases and deaths, cumulatively up to 4 weeks. We found that, in some of the counties, the percentage of the total number of COVID-19 cases and deaths attributable to the high

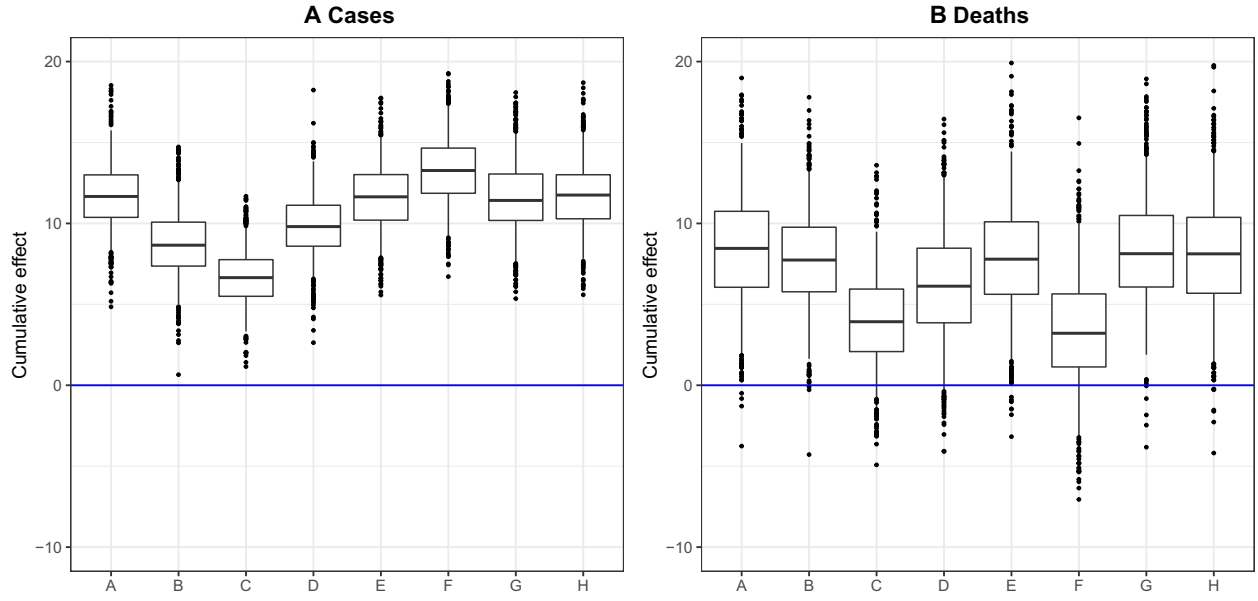

Fig. 5. Cumulative effect of $PM_{2.5}$ exposure in six sensitivity analysis with respect to confounding adjustment for seasonality, long-term trends, weather, and mobility. These are posterior distribution of the percentage increase in COVID-19 cases (**A**) and COVID-19 deaths (**B**) associated with a daily increase of 10 μg/m^3 in $PM_{2.5}$ for 28 subsequent days, cumulatively for 4 weeks, and pooled across counties. A, original analysis; B, decreasing the number of lag days under consideration from 4 to 3 weeks along with a decrease in number of spline bases for approximating the distributed lag function from six to five; C, decreasing the number of lag days in consideration to 2 weeks along with a decrease in the number of natural spline basis functions to four; D, adjusting for mobility data, which consequently omits six counties; E, increasing the number of spline basis functions for the distributed lag function from six to eight; F, more aggressively adjusting for temperature, humidity, and seasonality trends; G, omitting Calaveras, CA, and Mono, CA, counties; H, dropping adjustments for the day of week.

levels of $PM_{2.5}$ was substantial. However, we also found evidence of large heterogeneity across counties, including evidence of protective effects in a small number of counties. These results provide strong evidence that, in many counties, the high levels of $PM_{2.5}$ that occurred during the 2020 wildfires substantially exacerbated the health burden of COVID-19.

Our results have biological plausibility. A recent review by Navarro *et al.* (*28*) described that the co-occurrence of SARS-CoV-2 infection and wildfire smoke inhalation may present an increased risk for COVID-19 illness. Woodby *et al.* (*29*) suggested that exposure to air pollution skews the adaptive immune response toward bacterial/allergic immune responses, as opposed to an antiviral response, which may affect COVID-19 severity and outcomes. Exposure to air pollutants could also predispose exposed populations toward developing COVID-19–associated immunopathology, enhancing virus-induced tissue inflammation and damage (*28*, *29*).

Many recent studies have illustrated the biological plausibility between short-term exposure to air pollution and COVID-19 cases and deaths [see, for example, (*36*)]. As reported in (*37*, *38*), COVID-19 could have an air transmission and $PM_{2.5}$ could create a suitable environment for transporting the virus at greater distances than those considered for close contact. Moreover, $PM_{2.5}$ induces inflammation in lung cells, and exposure to $PM_{2.5}$ could increase the susceptibility and severity of the COVID-19 patient symptoms.

It is important to interpret our results in the context of the time course of COVID-19 infection and deaths. The CDC website (*39*) reports that the incubation period for COVID-19 is thought to extend to 14 days, with a median time of 4 to 5 days from exposure to symptom onset. Recently, the World Health Organization reported that the time between symptom onset and death ranged from about 2 to 8 weeks (*40*). This is consistent with our results of findings delayed effects between daily increases in $PM_{2.5}$ and COVID-19 cases and deaths up to 4 weeks later.

The key strengths of this study are the following: (i) the representativeness of the study population; (ii) the multisite time series study design; (iii) the use of satellite data to identify wildfire days and their validation with airport data; (iv) statistical modeling that accounts for delayed effects, heterogeneity across counties, and days with zero events; (v) the use of counterfactual analysis to estimate the levels of $PM_{2.5}$ on wildfire days compared to the levels that would have occurred in the absence of wildfires; and (vi) the numerous sensitivity analyses conducted to increase confidence in the results.

To our knowledge, ours is the first study to use data from multiple states and counties affected by the 2020 wildfire season in the United States, covering 95% of the population of Washington, Oregon, and California. Meo *et al.* (*41*) conducted a similar study in 10 counties in California. They reported that, after the wildfires, $PM_{2.5}$ concentrations increased by 220.71% and the numbers of COVID-19 cases and deaths increased by 56.9 and 148.2%, respectively (*41*). However, a key limitation of that study was that the statistical modeling did not account for the many challenges inherent in these data, such as confounding factors, delayed effects, overdispersion, and heterogeneity across counties. The study also did not attempt to distinguish wildfire $PM_{2.5}$ from other types of particles. Leifer *et al.* (*42*) also conducted a similar study focusing on three wildfire events in Orange County, CA, and have the same limitations.

The multisite time series study design allowed us to estimate, separately for each county, the association between daily changes in

$PM_{2.5}$ and the percentage increases in COVID-19 cases and deaths for every single lag and up to 4 weeks (2 or 3 weeks) later. In our model, we adjusted for temperature, humidity, and mobility and used satellite data to identify wildfire days. We also attempted to account for other unmeasured confounders by including a smooth function of time to control for seasonality and temporal trends. By modeling the temporal dynamic between exposure to $PM_{2.5}$ and the delayed effects on COVID-19 cases and deaths separately within each county, our approach takes advantage of differences in the timing of wildfires across counties. Moreover, the time series nature of the study is such that the only potential confounders of the association between $PM_{2.5}$ and COVID-19 are factors that vary temporally in a similar manner as $PM_{2.5}$. Thus, because of the sudden spikes in the level of $PM_{2.5}$ on wildfire days (see Fig. 2 for Los Angeles County), it is hard to hypothesize a factor that would be correlated with these $PM_{2.5}$ increases and, at the same time, affect COVID-19 cases and deaths. Nevertheless, as mentioned below, potential unmeasured confounding cannot be fully ruled out.

New COVID-19 cases and deaths can fluctuate for other reasons beyond short-term exposure to $PM_{2.5}$, including changes in temperature, humidity, societal patterns of social distancing and mass gatherings, or adherence to wearing masks. We adjusted for both measured and unmeasured confounding factors using nonlinear modeling of temperature variables and smooth functions of time to control for seasonality and trends. Furthermore, we purposely estimated the dynamic relationship between exposure to $PM_{2.5}$ and COVID-19 cases and deaths separately within each county in an attempt to account for heterogeneity across counties in many potential confounding factors that were unmeasured and can vary temporally, such as social distancing, mass gatherings, and testing practices. Still, residual unmeasured confounding could lead to bias. It is possible that the protective effects that we observed in a small number of counties could be the result of unmeasured confounding bias and/or a very small number of events.

Beyond simply providing evidence of a short-term association between daily changes in ambient levels of $PM_{2.5}$ and COVID-19 cases and deaths, we used historical median levels of $PM_{2.5}$ to estimate the increases in the levels of $PM_{2.5}$ attributable to wildfires in the 2020 season and, by extension, the excess COVID-19 burden. On the wildfire days, we found greater $PM_{2.5}$ levels in 2020 compared to the same days in previous years, and this led to extra COVID-19 cases and deaths in many of the counties included in the study. This illustrates the systemic and contingent nature of crises and how the effects of one global crisis (climate change) can have cascading effects on concurrent global crises (the COVID-19 pandemic) that play out in location-specific ways (increased COVID-19 cases and deaths due to wildfire).

We conducted numerous sensitivity analyses to check the robustness of our findings. For example, we varied the degrees of freedom of the smooth functions to adjust more or less aggressively for seasonality and temporal trends. We also conducted analyses that allow for delayed effects under various distributed lag time frames and assessed the sensitivity of the results to the adjustment for mobility data. In addition, we ran simulations of the data-generating process assumed by our model. Our data and results are fully reproducible. As shown in Fig. 4, in Calaveras, CA, we found a very large estimate of the percentage of total COVID-19 deaths attributable to $PM_{2.5}$ levels on the wildfire days (77.6%; 95% CI, 32.6 to 128.1), even after having accounted for many potential confounders. This is due to the fact that 77% (17 of 22) of the total number of COVID-19 deaths occurred during or near wildfire days with very high levels of $PM_{2.5}$.

Our study has several limitations: (i) We treated all sources of $PM_{2.5}$ as having the same effect on health, when emerging evidence is beginning to show that $PM_{2.5}$ from wildfire smoke may actually be more damaging to health than $PM_{2.5}$ from other sources (*20*); (ii) we did not investigate whether $PM_{2.5}$ from wildfire smoke is more toxic than $PM_{2.5}$ from other sources (*4*, *43*, *44*); rather, we determined whether the higher levels of $PM_{2.5}$ (regardless of the source) that occurred on wildfire days further exacerbated the burden of COVID-19; (iii) observations of $PM_{2.5}$ on wildfire days were missing for 41 of the 133 counties, leading us to drop these counties from the analysis, as imputing these missing observations using historical values of $PM_{2.5}$ would not have been appropriate; (iv) the HMS smoke product is based on an analyst's qualitative interpretation of satellite images, which may not accurately reflect surface smoke conditions, especially when smoke plumes are aloft or cloud cover interferes with smoke detection. HMS also does not quantify the fraction of wildfire-specific $PM_{2.5}$ and so may not necessarily reflect the human exposure to smoke alone; (v) our model assumes linear exposure response curve for each lag (we recognize that nonlinear extensions would have been desirable); (vi) the reported date of the COVID-19 cases or deaths used in the analysis might be delayed by a few days with respect to the date when the event occurred (we adjusted for day of the week and smoothed the lag-specific coefficients across lags to overcome for this issue); (vii) data on the testing rates are not available, and therefore, the results for the COVID-19 cases should be interpreted with caution, as they might be affected by unmeasured confounding bias; and (viii) our analysis did not account for exposure uncertainty, as we assume that daily exposure to $PM_{2.5}$ is the same for all the subjects living in the same county.

The year 2020 has presented unimaginable challenges due to the COVID-19 pandemic and the wildfires in the western United States (*45*). As the states of Washington, Oregon, and California were trying to contain the pandemic, they were afflicted by wildfires of unprecedented intensity. In some counties, the levels of $PM_{2.5}$ were higher than 500 $\mu g/m^3$ for several consecutive days. Climate change has been a key factor in increasing the risk and extent of wildfires in the western United States. Robust projections indicate that the risk of wildfires will continue to increase in most areas of the world as climate change worsens (*16*) and that the fires will increase excess mortality and morbidity from burns, wildfire smoke, and mental health effects (*11*, *16*, *19*, *46*–*51*). For example, in a previous study, we estimated that, between 2046 and 2051, more than 82 million people will likely be affected by "smoke waves," defined as two or more days with unhealthy levels of $PM_{2.5}$ from fire, in northern California, western Oregon, and the Great Plains (*4*). Wildfire risk depends on a number of factors, including temperature, soil moisture, and the presence of trees, shrubs, and other potential fuel. All of these factors have strong direct or indirect ties to climate variability and climate change (*52*). Wildfire smoke pollution is of growing importance due to climate change, with similarities in composition and health effects of anthropogenic and wildfire air pollution. Wildfire smoke pollution has the potential to increase COVID-19 transmission due to acute wildfire PM exposure (*27*). Notably, wildfires during a pandemic create a cascading disaster with disruption to directly affected communities that challenge infection mitigation

practices, such as social distancing in evacuation shelters, while local disaster responses (including healthcare) are multiply stressed. These synergies likely increased COVID-19 cases and negative outcomes (*45*).

The world has been in pandemic mode for more than a year. International divergence in vaccine distribution has slowed the pace of inoculations. Although their forecasts and timelines vary, modelers agree that COVID-19 is here to stay (*53*). In this study, we have identified high levels of $PM_{2.5}$ during wildfire days in the western United States in 2020 as a key factor in exacerbating the severity of the COVID-19 pandemic in affected counties across three states.

MATERIALS AND METHODS

Table S2 summarizes the sources of the publicly available data used in this study. We conducted a multisite time series study by harmonizing and linking daily data for the outcome, exposure, and confounders at the county level for 92 U.S. counties. We analyzed daily time series data for the period from 15 March to 16 December 2020. We included 92 of 133 U.S. counties located in the three U.S. states that were affected by the 2020 wildfires (Washington, Oregon, and California). We excluded from the analysis 41 counties that had missing daily $PM_{2.5}$ data during wildfire days, as it would have not been appropriate to impute these missing daily $PM_{2.5}$ levels using historical data. These 92 counties cover 95.1% of the population of three states (48.8 million).

Missing data

Missing data for the COVID-19 cases and deaths are both 0.65% of the total number of observations. We excluded these records from the analysis. Mobility data were missing for six counties, which cover 0.1% of the study population. We excluded these six counties in sensitivity analyses that adjusted for mobility. There were 250 missing $PM_{2.5}$ values on non-wildfire days. We imputed these missing data using the historical median value for the same day during 2016–2019. The replacement had a mean of 4.6 $\mu g/m^3$, close to the median on non-wildfire days in 2020 (5.0 $\mu g/m^3$). We collected daily smoke density data up to 26 November 2020 from NOAA Hazard Mapping System (*54*); thus, our estimates of excess COVID-19 cases and deaths are based on the time period from 15 March to 26 November 2020.

Statistical methods

We developed and implemented a BH-ZINB-DL model to estimate the association between daily changes in $PM_{2.5}$ and the percentage increase in the risk of COVID-19 cases and deaths up to 28 days after exposure. A distributed lag model is a popular approach for the analysis of time series data and has been implemented to estimate the association between an increase in exposure to air pollution on a given day and the percentage increase in the risk of a health outcome on the same day (lag 0), the day after (lag 1), and up to several days after exposure (*34*, *35*). We fit the same model for COVID-19 cases and COVID-19 deaths. The model can be described as a two-stage model for illustrative purposes, but in practice, the entire model is fit jointly. More specifically, in the first stage of the model, we specify a zero inflated negative binomial model for the daily time series data for each county. We assume county-specific random intercepts and county-specific random effects for the vector of the distributed lag coefficients denoted by θ_{ik}, where i denotes the county and k denotes the lag. In the main analysis, we considered lags up to 28 days, whereas in sensitivity analyses, we considered lags up to 21 and 14 days. The daily event rates are adjusted for the day of the week, for nonlinear confounding effects of temperature and humidity, for trend and seasonality, and for county-specific population size. Note that we considered a constrained distributed lag model where we assumed that the lag-specific coefficients θ_{ik} are a smooth function of the k lagged measurements. In the second stage of the model, we introduce random effects distributions to combine information across counties and estimate lag-specific coefficients (η_k), which are the pooled effect estimates estimated across counties.

For each county, we estimated the following parameters: (i) lag-specific effects, defined as the percentage increase in COVID-19 cases (or deaths) associated with a daily increase of 10 $\mu g/m^3$ in $PM_{2.5}$ separately for each lag, and (ii) cumulative effects across all lags, the percentage increase in COVID-19 cases (or deaths) associated with a daily increase of 10 $\mu g/m^3$ in $PM_{2.5}$ for 28 subsequent days, cumulatively across all lags. Table S1 summarizes the parameters of interest and their mathematical formulations.

We fit the models via Markov chain Monte Carlo and obtained posterior samples of all the unknown parameters. The data and statistical models fit with R 4.0.4 and JAGS 4.3.0 have been made available with the paper. The technical details of the BH-ZINB-DL model are described in section S2. We tested the performance of the model to simulated data, and details are included in section S2.2.

Estimating the percentage of total COVID-19 cases and deaths attributable to the observed high levels of $PM_{2.5}$ on the wildfire days

To describe our approach, it is useful to consider the factual and the counterfactual scenarios. In the factual scenario, we observed levels of $PM_{2.5}$ on wildfire days. We denote these values as PM_{ij}, where j indexes the days after 15 March 2020. We then considered the counterfactual scenario where wildfires did not occur. Under this counterfactual scenario, we estimated the daily levels of $PM_{2.5}$ that we would have observed for the same days and for the same county using historical data. We denote these values as $\widetilde{PM}_{ij}$. See above for details and also fig. S1.

The overall goal of this counterfactual analysis is to estimate the number of COVID-19 cases and deaths in 2020 had there been no wildfires. In other words, we are estimating counterfactual outcomes for COVID-19 cases/deaths had the $PM_{2.5}$ measurements been the same as those observed in past years without wildfires. More specifically, we denote by λ_{ij}, and ρ_{ij}, the expected number of COVID-19 cases (deaths) on a day j in county i estimated in correspondence of the factual and counterfactual values of $PM_{2.5}$. The posterior samples of ρ_{ij} represent predictions of λ_{ij} while substituting the factual $PM_{2.5}$ with the counterfactual PM_{ij} into eq. S6, which appears in section S1. The same parameter draws used to generate λ_{ij} are also used to generate ρ_{ij}. First, we calculate the excess number of COVID-19 cases (or deaths) as

$$G_i = \sum_{j=1}^{277} Y_{ij} \times \left(1 - \frac{\rho_{ij}}{\lambda_{ij}}\right) \quad (1)$$

where Y_{ij} is the observed daily number of COVID-19 cases (or deaths) and 277 is the total number of days. Second, we estimate the percentage of the total number of cases (or deaths) as

$$H_i = 100\,\% \times \frac{G_i}{N_i - G_i} \quad (2)$$

where N_i is the number of COVID-19 cases or deaths observed in county i for the time period between 15 March and 16 December 2020. Note that because the number of wildfire days, the levels of $PM_{2.5}$, and the association between exposure to $PM_{2.5}$ and COVID-19 cases (or deaths) vary across counties, we decided not to pool this information across counties. The overall number of COVID-19 cases and deaths attributable to observed high levels of $PM_{2.5}$ on the wildfire days is estimated as $\sum_{i=1}^{92} G_i$.

SUPPLEMENTARY MATERIALS

Supplementary material for this article is available at http://advances.sciencemag.org/cgi/content/full/7/33/eabi8789/DC1

REFERENCES AND NOTES

1. National Fire News: National Interagency Fire Center (2020).
2. M. Burke, A. Driscoll, J. Xue, S. Heft-Neal, J. Burney, M. Wara, The changing risk and burden of wildfire in the United States. *Proc. Natl. Acad. Sci. U.S.A.* **118**, e2011048118 (2021).
3. C. Liu, R. Chen, F. Sera, A. M. Vicedo-Cabrera, Y. Guo, S. Tong, M. S. Z. S. Coelho, P. H. N. Saldiva, E. Lavigne, P. Matus, N. V. Ortega, S. O. Garcia, M. Pascal, M. Stafoggia, M. Scortichini, M. Hashizume, Y. Honda, M. Hurtado-Díaz, J. Cruz, B. Nunes, J. P. Teixeira, H. Kim, A. Tobias, C. Íñiguez, B. Forsberg, C. Åström, M. S. Ragettli, Y.-L. Guo, B.-Y. Chen, M. L. Bell, C. Y. Wright, N. Scovronick, R. M. Garland, A. Milojevic, J. Kyselý, A. Urban, H. Orru, E. Indermitte, J. J. K. Jaakkola, N. R. I. Ryti, K. Katsouyanni, A. Analitis, A. Zanobetti, J. Schwartz, J. Chen, T. Wu, A. Cohen, A. Gasparrini, H. Kan, Ambient particulate air pollution and daily mortality in 652 cities. *N. Engl. J. Med.* **381**, 705–715 (2019).
4. J. C. Liu, A. Wilson, L. J. Mickley, F. Dominici, K. Ebisu, Y. Wang, M. P. Sulprizio, R. D. Peng, X. Yue, J.-Y. Son, G. B. Anderson, M. L. Bell, Wildfire-specific fine particulate matter and risk of hospital admissions in urban and rural counties. *Epidemiology* **28**, 77–85 (2017).
5. N. Watts, M. Amann, N. Arnell, S. Ayeb-Karlsson, J. Beagley, K. Belesova, M. Boykoff, P. Byass, W. Cai, D. Campbell-Lendrum, S. Capstick, J. Chambers, S. Coleman, C. Dalin, M. Daly, N. Dasandi, S. Dasgupta, M. Davies, C. D. Napoli, P. Dominguez-Salas, P. Drummond, R. Dubrow, K. L. Ebi, M. Eckelman, P. Ekins, L. E. Escobar, L. Georgeson, S. Golder, D. Grace, H. Graham, P. Haggar, I. Hamilton, S. Hartinger, J. Hess, S.-C. Hsu, N. Hughes, S. J. Mikhaylov, M. P. Jimenez, I. Kelman, H. Kennard, G. Kiesewetter, P. L. Kinney, T. Kjellstrom, D. Kniveton, P. Lampard, B. Lemke, Y. Liu, Z. Liu, M. Lott, R. Lowe, J. Martinez-Urtaza, M. Maslin, L. M. Allister, A. M. Gushin, C. M. Michael, J. Milner, M. Moradi-Lakeh, K. Morrissey, S. Munzert, K. A. Murray, T. Neville, M. Nilsson, M. O. Sewe, T. Oreszczyn, M. Otto, F. Owfi, O. Pearman, D. Pencheon, R. Quinn, M. Rabbaniha, E. Robinson, J. Rocklöv, M. Romanello, J. C. Semenza, J. Sherman, L. Shi, M. Springmann, M. Tabatabaei, J. Taylor, J. Triñanes, J. Shumake-Guillemot, B. Vu, P. Wilkinson, M. Winning, P. Gong, H. Montgomery, A. Costello, The 2020 report of the lancet countdown on health and climate change: Responding to converging crises. *Lancet* **397**, 129–170 (2020).
6. T. Deryugina, G. Heutel, N. H. Miller, D. Molitor, J. Reif, The mortality and medical costs of air pollution: Evidence from changes in wind direction. *Am. Econ. Rev.* **109**, 4178–4219 (2019).
7. J. M. Samet, F. Dominici, F. C. Curriero, I. Coursac, S. L. Zeger, Fine particulate air pollution and mortality in 20 U.S. cities, 1987–1994. *N. Engl. J. Med.* **343**, 1742–1749 (2000).
8. F. Dominici, R. D. Peng, M. L. Bell, L. Pham, A. McDermott, S. L. Zeger, J. M. Samet, Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *JAMA* **295**, 1127–1134 (2006).
9. Q. Di, Y. Wang, A. Zanobetti, Y. Wang, P. Koutrakis, C. Choirat, F. Dominici, J. D. Schwartz, Air pollution and mortality in the Medicare population. *N. Engl. J. Med.* **376**, 2513–2522 (2017).
10. H. Chen, J. M. Samet, P. A. Bromberg, H. Tong, Cardiovascular health impacts of wildfire smoke exposure. *Part. Fibre Toxicol.* **18**, 2 (2021).
11. C. E. Reid, M. Brauer, F. H. Johnston, M. Jerrett, J. R. Balmes, C. T. Elliott, Critical review of health impacts of wildfire smoke exposure. *Environ. Health Perspect.* **124**, 1334–1343 (2016).
12. W. E. Cascio, Wildland fire smoke and human health. *Sci. Total Environ.* **624**, 586–595 (2018).
13. J. A. Casey, M.-A. Kioumourtzoglou, H. Elser, D. Walker, S. Taylor, S. Adams, R. Aguilera, T. Benmarhnia, R. Catalano, Wildfire particulate matter in Shasta County, California and respiratory and circulatory disease-related emergency department visits and mortality, 2013–2018. *Environ. Epidemiol.* **5**, e124 (2021).
14. O. Adetona, T. E. Reinhardt, J. Domitrovich, G. Broyles, A. M. Adetona, M. T. Kleinman, R. D. Ottmar, L. P. Naeher, Review of the health effects of wildland fire smoke on wildland firefighters and the public. *Inhal. Toxicol.* **28**, 95–139 (2016).
15. C. J. Matz, M. Egyed, G. Xi, J. Racine, R. Pavlovic, R. Rittmaster, S. B. Henderson, D. M. Stieb, Health impact analysis of $PM_{2.5}$ from wildfire smoke in Canada (2013–2015, 2017–2018). *Sci. Total Environ.* **725**, 138506 (2020).
16. R. Xu, P. Yu, M. J. Abramson, F. H. Johnston, J. M. Samet, M. L. Bell, A. Haines, K. L. Ebi, S. Li, Y. Guo, Wildfires, global climate change, and human health. *N. Engl. J. Med.* **383**, 2173–2181 (2020).
17. C. E. Reid, M. Jerrett, I. B. Tager, M. L. Petersen, J. K. Mann, J. R. Balmes, Differential respiratory health effects from the 2008 northern California wildfires: A spatiotemporal approach. *Environ. Res.* **150**, 227–235 (2016).
18. J. C. Liu, G. Pereira, S. A. Uhl, M. A. Bravo, M. L. Bell, A systematic review of the physical health impacts from non-occupational exposure to wildfire smoke. *Environ. Res.* **136**, 120–132 (2015).
19. C. Black, Y. Tesfaigzi, J. A. Bassein, L. A. Miller, Wildfire smoke exposure and human health: Significant gaps in research for a growing public health issue. *Environ. Toxicol. Pharmacol.* **55**, 186–195 (2017).
20. R. Aguilera, T. Corringham, A. Gershunov, T. Benmarhnia, Wildfire smoke impacts respiratory health more than fine particles from other sources: Observational evidence from southern California. *Nat. Commun.* **12**, 1493 (2021).
21. J. L. Domingo, J. Rovira, Effects of air pollutants on the transmission and severity of respiratory viral infections. *Environ. Res.* **187**, 109650 (2020).
22. S. M. Holm, M. D. Miller, J. R. Balmes, Health effects of wildfire smoke in children and public health tools: A narrative review. *J. Expo. Sci. Environ. Epidemiol.* **31**, 1–20 (2020).
23. X. Wu, R. C. Nethery, M. B. Sabath, D. Braun, F. Dominici, Air pollution and covid-19 mortality in the United States: Strengths and limitations of an ecological regression analysis. *Sci. Adv.* **6**, eabd4049 (2020).
24. T. Benmarhnia, Linkages between air pollution and the health burden from covid-19: Methodological challenges and opportunities. *Am. J. Epidemiol.* **189**, 1238–1243 (2020).
25. A. Pozzer, F. Dominici, A. Haines, C. Witt, T. Münzel, J. Lelieveld, Regional and global contributions of air pollution to risk of death from COVID-19. *Cardiovasc. Res.* **116**, 2247–2253 (2020).
26. U.S. Centers for Disease Control and Prevention, Wildfire Smoke and COVID-19, 23 June 2021 (2021); https://www.cdc.gov/disasters/covid-19/wildfire_smoke_covid-19.html [accessed 2 August 2021].
27. S. B. Henderson, The COVID-19 pandemic and wildfire smoke: Potentially concomitant disasters. *Am. J. Public Health* **110**, 1140–1142 (2020).
28. K. M. Navarro, K. A. Clark, D. J. Hardt, C. E. Reid, P. W. Lahm, J. W. Domitrovich, C. R. Butler, J. R. Balmes, Wildland firefighter exposure to smoke and covid-19: A new risk on the fire line. *Sci. Total Environ.* **760**, 144296 (2020).
29. B. Woodby, M. M. Arnold, G. Valacchi, Sars-cov-2 infection, covid-19 pathogenesis, and exposure to air pollution: What is the connection? *Ann. N. Y. Acad. Sci.* **1486**, 15–38 (2020).
30. G. D. Rolph, R. R. Draxler, A. F. Stein, A. Taylor, M. G. Ruminski, S. Kondragunta, J. Zeng, H.-C. Huang, G. Manikin, J. T. McQueen, P. M. Davidson, Description and verification of the NOAA smoke forecasting system: The 2007 fire season. *Weather Forecast.* **24**, 361–378 (2009).
31. S. J. Brey, E. V. Fischer, Smoke in the city: How often and where does smoke impact summertime ozone in the United States? *Environ. Sci. Technol.* **50**, 1288–1294 (2016).
32. S. J. Brey, M. Ruminski, S. A. Atwood, E. V. Fischer, Connecting smoke plumes to sources using hazard mapping system (HMS) smoke and fire location data over north America. *Atmos. Chem. Phys.* **18**, 1745–1761 (2018).
33. F. Dominici, C. Zigler, Best practices for gauging evidence of causality in air pollution epidemiology. *Am. J. Epidemiol.* **186**, 1303–1309 (2017).
34. A. Gasparrini, B. Armstrong, M. G. Kenward, Distributed lag non-linear models. *Stat. Med.* **29**, 2224–2234 (2010).
35. L. J. Welty, R. D. Peng, S. L. Zeger, F. Dominici, Bayesian distributed lag models: Estimating effects of particulate matter air pollution on daily mortality. *Biometrics* **65**, 282–291 (2009).
36. N. Ali, F. Islam, The effects of air pollution on covid-19 infection and mortality–A review on recent evidence. *Front. Public Health* **8**, 580057 (2020).
37. N. T. Tung, P.-C. Cheng, K.-H. Chi, T.-C. Hsiao, T. Jones, K. BéruBé, K.-F. Ho, H.-C. Chuang, Particulate matter and sars-cov-2: A possible model of covid-19 transmission. *Sci. Total Environ.* **750**, 141532 (2021).
38. S. Comunian, D. Dongo, C. Milani, P. Palestini, Air pollution and covid-19: The role of particulate matter in the spread and increase of covid-19's morbidity and mortality. *Int. J. Environ. Res. Public Health* **17**, 4487 (2020).

39. U.S. Centers for Disease Control and Prevention, Management of patients with confirmed 2019-ncov, updated 12 February 2021 (2021); https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-guidance-management-patients.html [accessed 2 August 2021].
40. D. Baud, X. Qi, K. Nielsen-Saines, D. Musso, L. Pomar, G. Favre, Real estimates of mortality following COVID-19 infection. *Lancet Infect. Dis.* **20**, 773 (2020).
41. S. A. Meo, A. A. Abukhalaf, A. A. Alomar, O. M. Alessa, W. Sami, D. C. Klonoff, Effect of environmental pollutants pm-2.5, carbon monoxide, and ozone on the incidence and mortality of sars-cov-2 infection in ten wildfire affected counties in California. *Sci. Total Environ.* **757**, 143948 (2021).
42. I. Leifer, M. T. Kleinman, D. Blake, D. Tratt, C. Marston, Wildfire smoke exposure: Covid19 comorbidity? *J. Respir.* **1**, 74–79 (2021).
43. J. D. Stowell, G. Geng, E. Saikawa, H. H. Chang, J. Fu, C.-E. Yang, Q. Zhu, Y. Liu, M. J. Strickland, Associations of wildfire smoke pm2. 5 exposure with cardiorespiratory events in Colorado 2011–2014. *Environ. Int.* **133**, 105151 (2019).
44. F. J. Kelly, J. C. Fussell, Global nature of airborne particle toxicity and health effects: A focus on megacities, wildfires, dust storms and residential biomass burning. *Toxicol. Res.* **9**, 331–345 (2020).
45. R. N. Salas, J. M. Shultz, C. G. Solomon, The climate crisis and covid-19—A major threat to the pandemic response. *N. Engl. J. Med.* **383**, e70 (2020).
46. S. E. Finlay, A. Moffat, R. Gazzard, D. Baker, V. Murray, Health impacts of wildfires. *PLOS Curr.* **4**, e4f959951cce2c (2012).
47. J. E. Neumann, M. Amend, S. C. Anenberg, P. L. Kinney, M. Sarofim, J. Martinich, J. Lukens, J.-W. Xu, H. Roman, Estimating pm2.5-related premature mortality and morbidity associated with future wildfire emissions in the western us. *Environ. Res. Lett.* **16**, 035019 (2021).
48. D. M. J. S. Bowman, C. A. Kolden, J. T. Abatzoglou, F. H. Johnston, G. R. van der Werf, M. Flannigan, Vegetation fires in the Anthropocene. *Nat. Rev. Earth Environ.* **1**, 500–515 (2020).
49. M. D. Hurteau, S. Liang, A. L. Westerling, C. Wiedinmyer, Vegetation-fire feedback reduces projected area burned under climate change. *Sci. Rep.* **9**, 2838 (2019).
50. M. Turco, J. J. Rosa-Cánovas, J. Bedia, S. Jerez, J. P. Montávez, M. C. Llasat, A. Provenzale, Exacerbated fires in Mediterranean Europe due to anthropogenic warming projected with non-stationary climate-fire models. *Nat. Commun.* **9**, 3821 (2018).
51. Q. Sun, C. Miao, M. Hanel, A. G. Borthwick, Q. Duan, D. Ji, H. Li, Global heat stress on health, wildfires, and agricultural crops under different levels of climate warming. *Environ. Int.* **128**, 125–136 (2019).
52. P. E. Dennison, S. C. Brewer, J. D. Arnold, M. A. Moritz, Large wildfire trends in the western United States, 1984–2011. *Geophys. Res. Lett.* **41**, 2928–2933 (2014).
53. M. Scudellari, How the pandemic might play out in 2021 and beyond. *Nature* **584**, 22–25 (2020).
54. NOAA Office of Satellite and Product Operations, NOAA hazard mapping system fire and smoke product, updated 5 August 2020 (2020); https://www.ospo.noaa.gov/Products/land/hms.html#about [accessed 2 August 2021].

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