PUBLIC HEALTH

# Mortality attributable to $PM_{2.5}$ from wildland fires in California from 2008 to 2018

**Rachel Connolly[1,2]*, Miriam E. Marlier[1], Diane A. Garcia-Gonzales[1], Joseph Wilkins[3], Jason Su[4], Claire Bekker[1], Jihoon Jung[5], Eimy Bonilla[3], Richard T. Burnett[6,7], Yifang Zhu[1], Michael Jerrett[1]***



**In California, wildfire risk and severity have grown substantially in the last several decades. Research has characterized extensive adverse health impacts from exposure to wildfire-attributable fine particulate matter ($PM_{2.5}$), but few studies have quantified long-term outcomes, and none have used a wildfire-specific chronic dose-response mortality coefficient. Here, we quantified the mortality burden for $PM_{2.5}$ exposure from California fires from 2008 to 2018 using Community Multiscale Air Quality modeling system wildland fire $PM_{2.5}$ estimates. We used a concentration-response function for $PM_{2.5}$, applying ZIP code–level mortality data and an estimated wildfire-specific dose-response coefficient accounting for the likely toxicity of wildfire smoke. We estimate a total of 52,480 to 55,710 premature deaths are attributable to wildland fire $PM_{2.5}$ over the 11-year period with respect to two exposure scenarios, equating to an economic impact of $432 to $456 billion. These findings extend evidence on climate-related health impacts, suggesting that wildfires account for a greater mortality and economic burden than indicated by earlier studies.**

## INTRODUCTION

Wildfire risk and severity have grown in the past several decades across the western United States. Climate change (*1*–*3*), an expansion of the wildland-urban interface (*4*, *5*), and questionable wildfire management practices emphasizing fire suppression have all contributed to this increased risk (*6*). In California, the traditional wildfire season has lengthened, causing peak impacts to occur in earlier months (*7*). California's recent wildfire seasons have caused extensive environmental, health, and economic damages within and outside of the state (*6*, *8*).

Wildfire smoke contributes to fine particulate matter ($PM_{2.5}$) pollution, with recent studies finding that smoke can account for one-quarter to one-half of $PM_{2.5}$ throughout the United States, with particularly high levels in western regions (*5*, *9*). Recent studies analyzing decadal air pollution trends through 2016 have found that $PM_{2.5}$ levels generally improved throughout the country over previous decades except for fire-prone regions in the northwest United States (*10*) and the western United States more broadly, which have experienced increases in summer smoke $PM_{2.5}$ (*11*). A recent study extending these analyses through 2022, however, found that since approximately 2016, progress has slowed or reversed in most states throughout the United States, with wildfire smoke influencing almost all states experiencing changes in trends (*12*).

Scholars use various methods for estimating air quality during wildfires, including chemical transport models (CTMs), machine learning algorithms, in situ monitoring data and satellite data, and combinations of these tools and datasets (*5*, *8*, *9*, *11*, *13*–*18*). Several of these methods have the ability to distinguish wildfire smoke from undifferentiated $PM_{2.5}$, with various strengths and limitations associated with each approach. In situ air quality monitoring is often sparse in fire-affected areas, and even with dense coverage, monitoring alone cannot isolate smoke $PM_{2.5}$ concentrations from undifferentiated $PM_{2.5}$ from all sources. Consequently, analyses modeling wildfire smoke remain vital for characterizing the spatial distribution, magnitude, and temporal trends of wildfires as well as understanding population exposures to smoke $PM_{2.5}$, which adversely affect public health (*19*–*23*).

Exposure to $PM_{2.5}$ in urban air is associated with a multitude of health risks, including premature mortality and respiratory and cardiovascular morbidity outcomes (*24*). In terms of wildfire-associated $PM_{2.5}$ specifically, relatively well-established evidence exists on the impact of wildfire smoke exposure on morbidity, such as respiratory illness and hospitalizations (*20*–*22*, *25*). Evidence for mortality resulting from $PM_{2.5}$ exposure during wildfire events is more mixed (*19*, *21*, *22*, *26*), although recent studies have quantified the relationship between short-term exposure to wildfire smoke and mortality (*27*, *28*) [as well as potentially fatal conditions (*29*)] and estimated health impacts during wildfire events, applying both wildfire-specific $PM_{2.5}$ dose-response coefficients and undifferentiated $PM_{2.5}$ dose-response coefficients to concentration changes to calculate premature deaths (*30*, *31*).

Such studies have largely found that exposure to $PM_{2.5}$ due to wildfires has substantial impacts on mortality and resulting economic burdens, with adverse effects reported in North America more broadly, the western United States, and California specifically, which is the study area for this analysis. One long-term analysis in Canada found that the estimated economic impact for chronic health effects over a 5-year period was between $4 billion and $19 billion annually, associated with 570 to 2500 annual attributable premature deaths across the population of more than 35 million individuals (*31*). An analysis across the United States, with a population of ~330 million (~310 million with respect to the time frame of the cited study), estimated wildfire impacts from a 5-year period to result in tens of thousands of deaths annually and a total of hundreds of billions of dollars for chronic impacts over the entire period

[1]Department of Environmental Health Sciences, Fielding School of Public Health, University of California, Los Angeles, Los Angeles, CA, USA. [2]Luskin Center for Innovation, University of California, Los Angeles, Los Angeles, CA, USA. [3]Department of Earth, Environment and Equity, Howard University, Washington, DC, USA. [4]Department of Environmental Health Sciences, School of Public Health, University of California, Berkeley, Berkeley, CA, USA. [5]Department of City and Regional Planning, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA. [6]Institute of Health Metrics and Evaluation, University of Washington, Seattle, WA, USA. [7]Population Studies Division, Environmental Health Science and Research Bureau, Health Canada, Ottawa, Canada.
*Corresponding author. Email: mjerrett@ucla.edu (M.J.); rachelconnolly@g.ucla.edu (R.C.)

(*32*). Another recent study analyzed mortality impacts from April to October in 2012, 2013, and 2014 and found 4000 annual deaths attributable to wildfires, alongside an economic valuation of $36 billion, with substantial air quality impacts and mortality burden in the western states (*33*). In a western United States–focused study, a short-term analysis examining a specific wildfire event in the fall of 2020 in Washington state found that, for the population of around 7.7 million, a 13-day period of increased $PM_{2.5}$ exposure from smoke was associated with more than 1000 premature deaths from the marginal contribution of wildfire smoke to chronic exposures and ~90 deaths from short-term exposures (*30*). Last, a recent study focused on 2018 California wildfires found that fires contributed to more than 3600 deaths and more than $148 billion in total damages from health costs and capital and other indirect losses (*8*).

While the California population of nearly 40 million is at a heightened risk of wildfire exposure, no long-term epidemiological studies have directly assessed the mortality impacts resulting from years of increasing wildfire exposures within the state. Existing studies are also limited by the use of county-level health data. Furthermore, no studies apply a chronic dose-response coefficient developed specifically for wildfire exposures; for long-term evaluations beyond a specific fire event, existing research solely uses undifferentiated $PM_{2.5}$ concentration-response coefficients, which do not capture differences in the characteristics of wildfire-specific $PM_{2.5}$ that could affect the dose-response effect (*25*, *34*).

To bridge these knowledge gaps, we use modeled wildland fire-associated $PM_{2.5}$ concentrations, high-resolution California Department of Public Health (CDPH) mortality data, and a calculated chronic dose-response coefficient for wildfire $PM_{2.5}$ exposures and mortality to estimate premature deaths due to wildland fires over an 11-year period from 2008 to 2018. The importance of wildfire management will only grow in the coming decades as aridification intensifies with climate change and more regions become susceptible to fires. Growing the evidence on health impacts from wildfires and potential health savings from wildfire management will be critical in ensuring the mitigation of wildfire impacts throughout the state and other regions.

## RESULTS

### Overview of modeled wildland fire $PM_{2.5}$ data

Here, we present a summary of the temporal, spatial, and overall distribution of the Community Multiscale Air Quality (CMAQ) modeled $PM_{2.5}$ concentrations at the 12-km grid scale. "All sources $PM_{2.5}$" refers to total, undifferentiated $PM_{2.5}$ concentrations, "nonfire $PM_{2.5}$" refers to concentrations excluding wildland fires, and "fire-only $PM_{2.5}$" describes the difference between those two simulations, the latter of which is the focus of our analysis. A model validation analysis at the monthly scale using several established model evaluation metrics is included in the Supplementary Materials (Supplementary Text).

Table 1 presents a summary of the modeled $PM_{2.5}$ estimates, which includes concentrations from the entire state, including rural areas with minimal background pollution. As shown in Table 1, fire-only $PM_{2.5}$ contributes between 6.9 and 49% of $PM_{2.5}$ from all sources, depending on the severity of the fires in each particular year. In 2008, 2017, and 2018, years where California fires burned between 1.5 million and nearly 2 million acres (*35*), fire-only $PM_{2.5}$ was responsible for almost half of all sources $PM_{2.5}$. The all sources $PM_{2.5}$ concentrations (which include fires) were considerably higher in those years as well.

Expanded summary statistics for the independent grid cells (minimum, mean, and maximum annual concentrations at the grid cell level) for all 11 years are provided in table S1. Elevated maximum fire-only concentrations exist for several years due to extreme wildland fire events, and there are also low minimum annual concentrations from grid cells with little to no fire activity.

To visually review model outputs, we examine fire-only concentrations for the entire time period (Fig. 1) as well as compare (i) all sources, (ii) nonfire, and (iii) fire-only concentrations at the grid cell

**Table 1. Summary of averaged modeled $PM_{2.5}$ (μg/m$^3$) values and acres burned by year (2008 to 2018) statewide in California.** Notes: All sources includes both fire and nonfire sources; fire-only includes wildland fire sources only; nonfire includes nonfire sources only. Acres burned were extracted from CAL FIRE Redbooks for each year (https://fire.ca.gov). N.A., not applicable to present a sum of acres burned alongside temporal averages.

| Year | All sources $PM_{2.5}$ (SD, μg/m$^3$)* | Fire-only $PM_{2.5}$ (SD, μg/m$^3$) | Nonfire $PM_{2.5}$ (SD, μg/m$^3$) | Percent of $PM_{2.5}$ attributable to fire | Total acres burned |
|---|---|---|---|---|---|
| 2008 | 8.83 (5.49) | 4.33 (5.04) | 4.51 (3.34) | 49.0% | 1,593,690 |
| 2009 | 4.78 (3.03) | 0.60 (0.39) | 4.18 (3.00) | 12.6% | 451,969 |
| 2010 | 4.61 (3.21) | 0.32 (0.29) | 4.30 (3.21) | 6.9% | 134,462 |
| 2011 | 3.91 (2.23) | 0.49 (0.34) | 3.42 (2.25) | 12.6% | 228,599 |
| 2012 | 3.83 (2.10) | 0.69 (0.74) | 3.14 (2.14) | 18.1% | 829,224 |
| 2013 | 3.88 (2.36) | 1.17 (1.26) | 2.70 (2.17) | 30.3% | 601,635 |
| 2014 | 4.74 (3.95) | 1.24 (3.73) | 3.49 (2.06) | 26.2% | 625,540 |
| 2015 | 5.32 (4.85) | 1.95 (4.75) | 3.37 (1.93) | 36.7% | 880,899 |
| 2016 | 4.11 (2.37) | 1.00 (1.46) | 3.10 (1.76) | 24.4% | 669,534 |
| 2017 | 6.76 (5.50) | 3.04 (5.28) | 3.72 (1.85) | 44.9% | 1,548,429 |
| 2018 | 7.65 (4.68) | 3.47 (4.42) | 4.18 (1.78) | 45.3% | 1,975,086 |
| All years | 5.31 (4.16) | 1.66 (3.47) | 3.65 (2.44) | 31.3% | N.A. |

*Includes total land area with rural locations with lower $PM_{2.5}$, which results in lower modeled values; see table S2 for a breakdown by metropolitan statistical area.

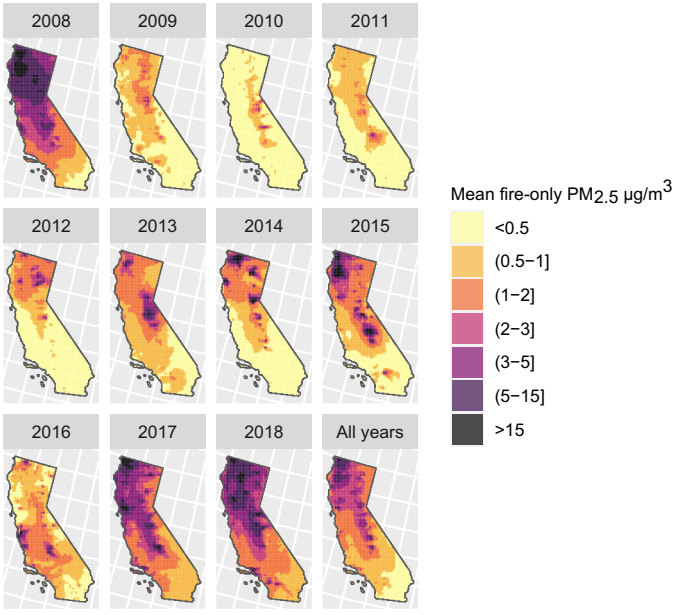


**Fig. 1. CMAQ average daily fire-only $PM_{2.5}$ concentrations (µg/m$^3$).** The data are at a 12-km resolution for 2008 to 2018, and the average value for all years is also presented. These are computed as the average over all days in each 12-km grid cell in each time period.

level for mean $PM_{2.5}$ across the 11-year period (fig. S1). We also visualize locations with daily $PM_{2.5}$ concentrations greater than the U.S. Environmental Protection Agency's (EPA) 24-hour (daily) National Ambient Air Quality Standards (NAAQS) of 35 µg/m$^3$ and annual NAAQS of 12 µg/m$^3$ over the entire 11-year period (fig. S2, A and B, respectively) and daily $PM_{2.5}$ concentrations greater than 35 µg/m$^3$ for each individual year (fig. S3) (this analysis was completed prior to the annual NAAQS update to 9 µg/m3 announced in February 2024) . These figures demonstrate spatial and temporal trends in elevated $PM_{2.5}$ concentrations but do not represent actual exceedances of the NAAQS standards or indicate nonattainment.

Figure 1 shows fire-only concentrations by year for all 11 years of data, with notable regional variation in fire impacts over the long-term period (see fig. S4 for the locations of fires greater than 300 acres in each year). Average annual fire-only concentrations exceed 15 µg/m$^3$ in several locations throughout the state in the high-fire years. In contrast, during the least affected year, 2010, the fire-only concentrations were less than 0.5 µg/m$^3$ throughout most of the state. The spatial distribution of all sources, nonfire, and fire-only concentrations (fig. S1) vary as anticipated due to differing pollution sources in different regions. Generally, wildfire smoke appears to expand the geographic areas affected by higher $PM_{2.5}$. The nonfire modeled values demonstrate higher pollution throughout two regions also prone to temperature inversions: LA County, a region known for extensive traffic and industrial pollution, and the San Joaquin Valley, with two large highways running north-south and pollution from agricultural sources. The fire-only concentrations affect more rural, forested areas throughout the state on average, especially in the northern and eastern parts of the state, although substantial regional variations not captured by these annual averages also exist (Fig. 1).

Most modeled concentrations higher than the 35 µg/m$^3$ NAAQS threshold over the 11-year period are due to fire-only $PM_{2.5}$ (fig. S2A). The most fire-affected regions in the state, mostly in the vicinity of national forests in northwest California and east of the San Joaquin Valley, have grid cells with close to or more than 100 days with modeled concentrations higher than the 24-hour NAAQS threshold over the 11-year period. The high-fire years contribute a large portion of these elevated values over much of the state, with more than 25 days greater than the daily NAAQS threshold within a given year (fig. S3). With respect to the annual averages of the modeled values, concentrations greater than the annual NAAQS in the more populated, urban regions of the state (such as Los Angeles) are primarily due to nonfire sources, with fire-only sources accounting for values higher than the NAAQS thresholds in the more rural regions in the northern part of the state (fig. S2B). These fire-only sources are responsible for average concentrations greater than the annual thresholds in several regions and for multiple years during the 11-year period, which demonstrates the magnitude of air pollution impacts during fire events.

## Mortality and economic valuation impacts of wildland fires

The total mortality burden for exposure to $PM_{2.5}$ due to wildfires in California, estimated for two exposure scenarios using a calculated chronic wildfire-specific dose-response value ($\beta_{WL}$), is presented in Fig. 2 (table S3), along with 95% confidence intervals (CIs). In the base case scenario, no outliers are removed to characterize the potential impact of extremely high wildfire concentrations on mortality. In the modified cap (mod cap) scenario, fire-only $PM_{2.5}$ concentrations falling outside of the 99.9th percentile of modeled values are excluded (capped) to account for potentially skewed concentrations produced by the CMAQ model simulations. The chronic wildfire-specific dose-response value applied here, $\beta_{WL}$, was derived to account for the potential increased toxicity of wildfire smoke (*25*, *36*) using pre-existing dose-response values from primary literature (including two wildfire-specific short-term dose-response values and a chronic, undifferentiated dose-response value for $PM_{2.5}$ from all sources; see Materials and Methods). We also include results using the preexisting chronic undifferentiated $PM_{2.5}$ dose-response value ($\beta_L$) (*37*) for comparison.

For the base case, including all of the original modeled fire-only values for all 11 years and applying $\beta_{WL}$, annual mortality impacts due to fire-only $PM_{2.5}$ exposure range from a low of ~1240 deaths (95% CI: 110 to 2370) in 2010 to a high of 12,850 (95% CI: 1150 to 23,730) in 2018 (Fig. 2); the latter of which is the year with the highest number of wildfire acres burned during our analysis period. This equates to a total of ~55,710 (95% CI: 4960 to 103,410) for the base case over the 11-year period and 52,480 (95% CI: 4610 to 98,340) for the mod cap (see table S4 for a by-county breakdown of base case mortality results alongside total valuation).

As previously mentioned, we also present estimated mortality impacts using an undifferentiated chronic $PM_{2.5}$ dose-response value not specific to wildfire smoke exposures, $\beta_L$ (*37*), to compare to our estimates using the calculated chronic wildfire-specific dose-response value (Fig. 2). When using $\beta_L$, the total estimated mortality attributable to fire-only $PM_{2.5}$ is ~36,140 (95% CI: 24,740 to 44,320) for the base case and 33,890 (95% CI: 23,130 to 41,650) for the mod cap. These estimates are ~35% less than the projected mortality impacts when using the $\beta_{WL}$ dose-response value accounting for wildfire-specific impacts, although the magnitude of these estimated

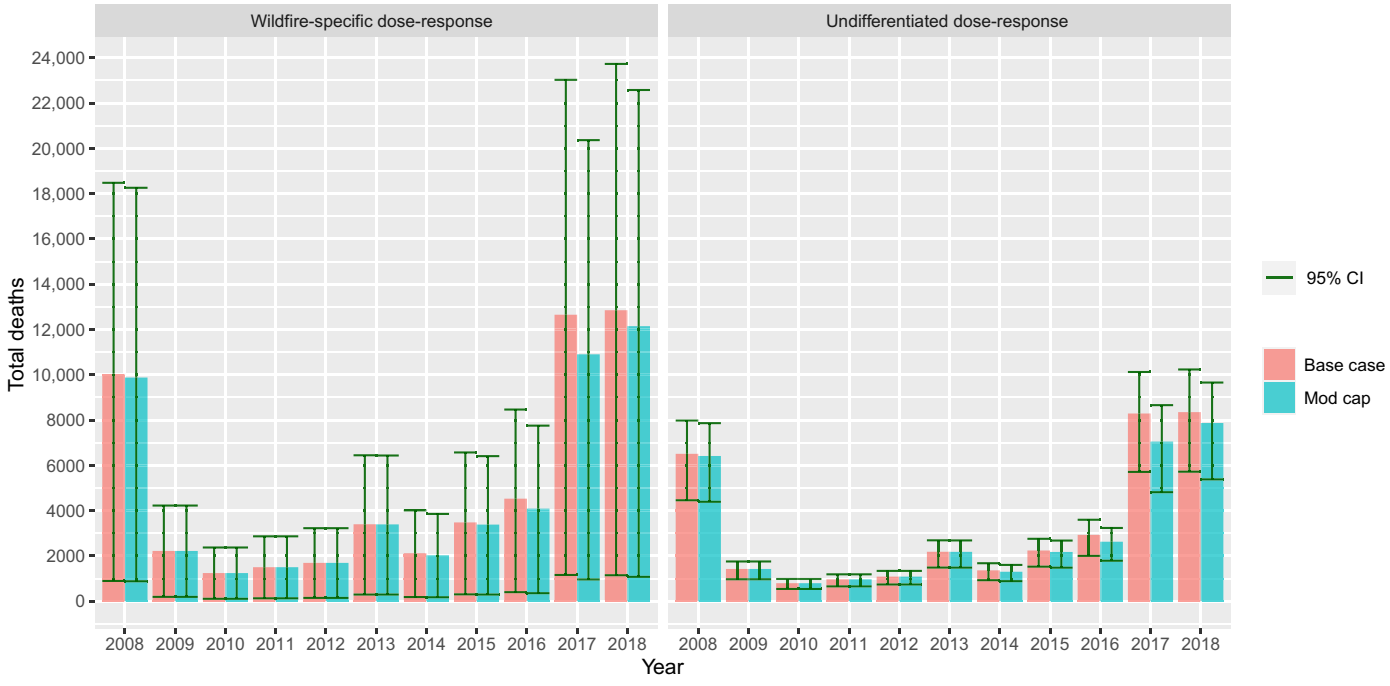


**Fig. 2. Summary of long-term mortality impacts across California due to fire-only $PM_{2.5}$.** This represents total deaths attributable to fire-only $PM_{2.5}$ for adults ages 25+. These were estimated using wildfire-specific (**left**) and undifferentiated (**right**) chronic dose-response values and are presented for 2008 to 2018. Base case, no modeled $PM_{2.5}$ concentrations are capped; mod cap, modeled $PM_{2.5}$ concentrations are capped at the 99.9th percentile value of all fire-only concentrations.

premature deaths demonstrates that, regardless of the added wildfire toxicity assumption (see Materials and Methods), mortality impacts from wildland fire smoke are substantial.

Figure 3 depicts base case mortality impacts across California for the year with the lowest number of deaths attributable to wildland fire (2010), highest number (2018), and the average over the 11-year period (see fig. S5 for the full by-year breakdown for all years and fig. S6 for the spatial distribution of total mortality impacts over the 11-year period). In 2010, a low-fire year with the least number of attributable deaths, ~90% of all ZIP codes were estimated to experience between 0 and 2 deaths. In 2018, the highest fire year with the largest number of deaths attributable to fire-only $PM_{2.5}$, almost 10% of ZIP codes experienced more than 15 deaths.

The elevated number of fires in 2008, 2017, and 2018—along with a large increase in mortality impacts, represented by dark blue on the maps—are particularly notable, and visible temporal and spatial trends exist (fig. S5). In 2008, the largest fires were clustered in northern California, with more statewide spread of fires throughout 2017 and 2018. Although fires throughout 2008 contributed a higher percent of all sources $PM_{2.5}$ than in 2017 and 2018 (Table 1), the attributable deaths were higher for the later years because the fires and smoke exposure in those years expanded to more to high population areas.

Although the fires are in more rural, forested regions (fig. S4 and Fig. 1), the mortality impacts are more widespread throughout population centers such as Los Angeles County in southern California, the San Joaquin Valley in central California, and the Bay Area in northern California as smoke can be transported to these areas and fewer individuals live in forested regions. For example, the Rough Fire of 2015 burned more than 150,000 acres in a more rural area of Fresno County, but most mortality impacts (represented by dark blue on the map) are west of the fire in a more populated area of the county and throughout the San Joaquin Valley more broadly.

These mortality impacts can be viewed in the context of two supplemental analyses. First, the mortality attributable to all sources $PM_{2.5}$ is presented in table S5. Premature deaths attributable to all sources $PM_{2.5}$ are five times larger than the mortality impacts from solely wildland fire impacts, with a total of 290,540 deaths attributable to undifferentiated $PM_{2.5}$ from all sources over the 11-year period of the analysis. Such results demonstrate that, while other sources of $PM_{2.5}$ may dominate in urban population centers and therefore result in disproportionately higher attributable mortality as compared to the overall contribution of wildland fire smoke to all sources $PM_{2.5}$ (Table 1), wildland fires are still responsible for ~19% of $PM_{2.5}$-associated deaths overall and up to 42% in high-fire years.

Second, the mortality attributable to fire-only $PM_{2.5}$ estimated using three different short-term dose-response values to estimate $\beta_{WL}$ [as opposed to the variance-weighted average of the U.S. estimate from the same global study and the Washington wildfires study (*27*)] results in higher estimates (tables S6 to S8). When the global dose-response value (based on a 0- to 2-day moving average of $PM_{2.5}$) is applied (*38*), the estimated mortality impacts are approximately twice as high as the results presented in Fig. 2, with a total estimate of 103,930 for all 11 years (table S6) versus 55,710 for our primary results. This is reflective of a larger short-term dose-response value for wildfire impacts for the global estimate as compared to the two United States–specific values used in the primary analysis. In addition, when lag-specific estimates from both the global study and the Washington study are pooled each based on the matching 0- and 1-day lags (tables S7 and S8), total deaths of 108,920 and 76,920 attributable to fire-$PM_{2.5}$ are estimated for the two lags, respectively. It is worth noting that the global dose-response values

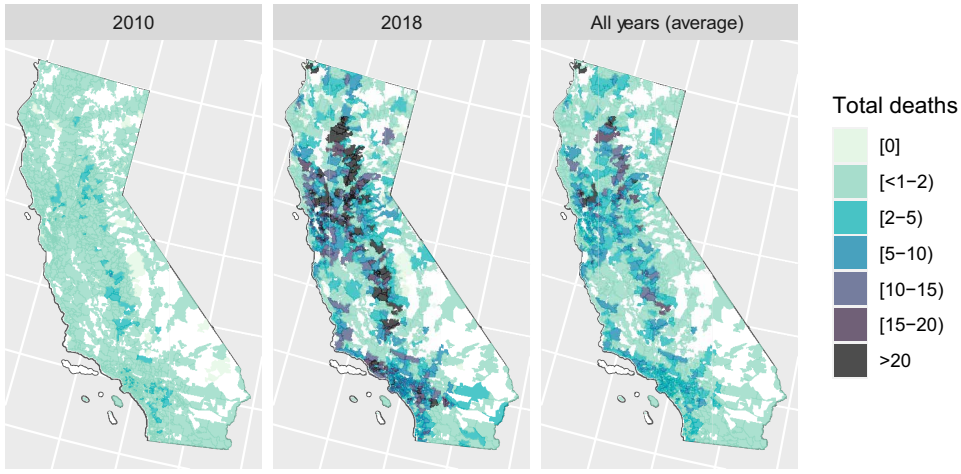


**Fig. 3. Total deaths attributable to fire-only $PM_{2.5}$ (base case).** This is depicted for the year with the fewest deaths attributable to wildland fire (2010), most deaths attributable to wildland fire (2018), and the annual average over the 11-year period (2008 to 2018). Darker colors indicate that more deaths occurred in a given ZIP code, and white areas are outside of ZIP code designations.

have much smaller standard errors than the estimates provided in the Washington state study and therefore are provided a substantially larger weight in the pooled dose-response estimates.

Last, the valuation estimates for the base case and the mod cap (and CIs), using only the primary wildfire-specific dose-response value, are presented in Fig. 4 and table S9. The net present value of the estimates for all years is ~$456 billion (95% CI: $40.6 billion to $847 billion) for the base case and $432 billion (95% CI: $37.9 billion to $808 billion) for the mod cap.

## DISCUSSION

Here, we report on modeled wildland fire $PM_{2.5}$ estimates at the 12-km grid scale for 2008 to 2018, estimate associated premature mortality using a chronic dose-response value for wildfire exposure, and calculate the associated economic valuation. We find that the modeled wildland fire-only $PM_{2.5}$ estimates follow anticipated spatial and temporal trends with respect to the patterns of fire activity in the state. An estimated 52,480 (95% CI: 4610 to 98,340) to 55,710 (95% CI: 4960 to 103,410) premature deaths are attributable to fire-only $PM_{2.5}$ in California from 2008 to 2018 with respect to two exposure scenarios, with an associated economic valuation of $432 billion and $456 billion (2015 dollars), respectively. These deaths account for nearly 19% of total deaths attributable to all sources $PM_{2.5}$ in the state during this 11-year period. This analysis characterizes mortality impacts in the state over a long 11-year period, applies a chronic dose-response value for wildfire-specific $PM_{2.5}$ exposure, and uses highly resolved health data in concert with a CTM (CMAQ) capable of isolating wildfire-related fine particle concentrations. These findings add to a growing body of literature on California-specific wildfire health effects (*39*–*41*) and more broadly to evidence on past and projected wildfire and other climate-related health impacts occurring in California, the United States, and globally (*42*–*47*). The large, growing impacts of wildfires on air pollution along with the mortality and economic burden presented here raise questions about societal investments in wildfire prevention and management. The state and federal governments have committed to a multiyear increase of about $6.7 billion for wildfire mitigation (*48*), but such investments fall well below the projected cost savings if greater investments were made to prevent and manage wildfire impacts.

### Modeled fire-only $PM_{2.5}$ estimates

The spatial distribution of fire-only $PM_{2.5}$ from our CMAQ model outputs aligns with general trends observed in analyses of historical fire records (*3*, *7*) and other environmental health–focused studies using modeled data (*49*), although the model can overpredict concentrations in the high-fire years [other studies have reported similar CMAQ tendencies toward overprediction during wildfire events due to challenges in modeling the distribution of fire emissions (*16*, *50*, *51*)]. As anticipated, the high-fire years of 2008, 2017, and 2018 demonstrated elevated $PM_{2.5}$ concentrations, with many daily and annual values greater than the associated NAAQS thresholds (Table 1 and figs. S2, A and B, and S3). A recent wildland fire modeling analysis by Koman *et al.* (*49*) used CMAQ to evaluate modeled exposure to wildland fire smoke from 2007 to 2013 in California and estimated all sources and fire-only $PM_{2.5}$ concentrations consistent with the results we present in Table 1 for the years overlapping with our analysis. This was expected considering the data inputs were similar, including the use of the BlueSky framework and SMARTFIRE2 to develop emissions to use within CMAQ. In addition, studies incorporating machine learning algorithms in estimating wildfire $PM_{2.5}$ are becoming more common as an alternative to CTMs (*9*, *13*, *15*); two recent studies have used machine learning techniques to parse out wildfire smoke $PM_{2.5}$ across the contiguous United States. Childs *et al.* (*9*) found that smoke $PM_{2.5}$ can contribute approximately half of annual all sources $PM_{2.5}$ in certain high-fire locations in the western United States (equating to an increase in annual $PM_{2.5}$ of 5 $\mu g/m^3$ in certain regions). This aligns with our modeled results for the high-fire years of 2008, 2017, and 2018 (Table 1) (*9*).

### Mortality impacts of exposure to wildland fire $PM_{2.5}$

We present a range of potential mortality impacts from two exposure scenarios [one with no modeled values altered (base case) and

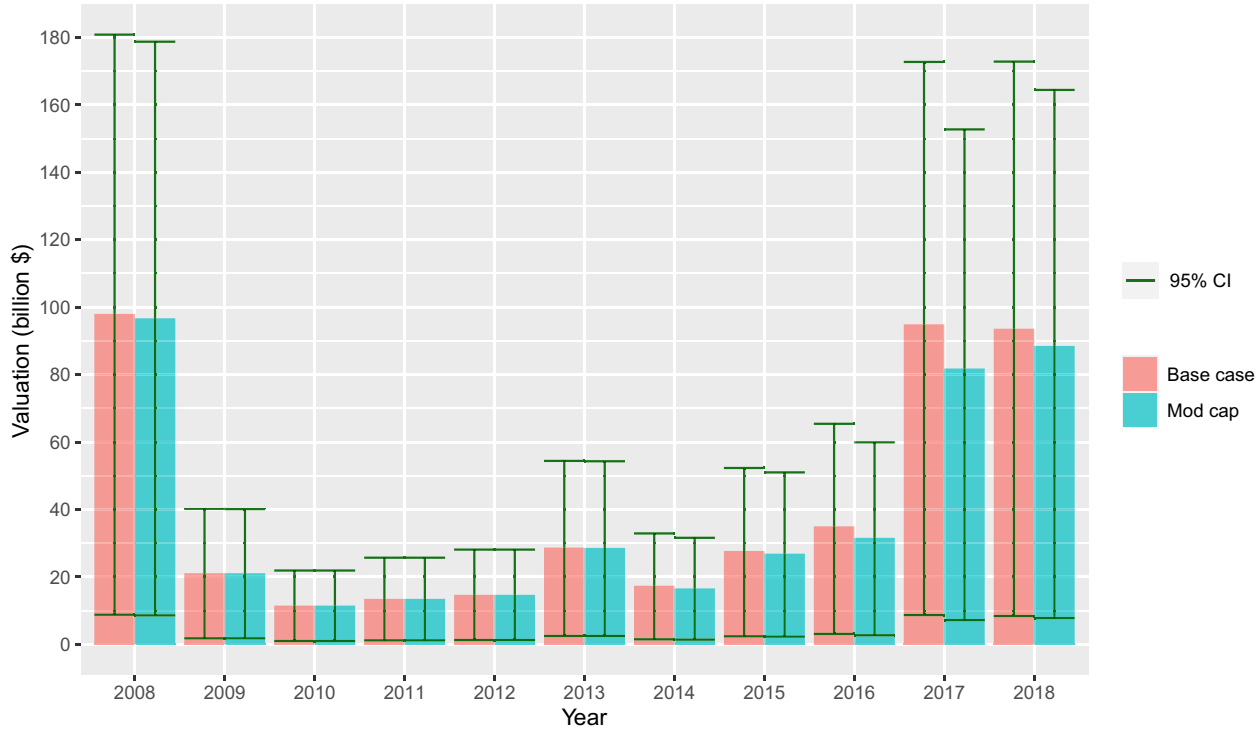


**Fig. 4. Economic valuation of mortality impacts from wildland fires.** These are presented alongside 95% CIs for the base case and mod cap scenarios, using the wildfire-specific dose-response value ($\beta_{WL}$; 2015 dollars, 3% discount rate, and 2015 income year).

one with modeled values capped (mod cap)] to account for uncertainties in the modeled $PM_{2.5}$ estimates. Our use of a wildfire-specific chronic dose-response value (as opposed to an undifferentiated dose-response value, which we also present as a sensitivity analysis) results in an increase in the magnitude of our findings, as is shown in the comparison to the premature mortality estimated using a chronic undifferentiated $PM_{2.5}$ dose-response value from Pope *et al.* (*37*) (Fig. 2). We selected the Pope *et al.* study because it is based on a recent, representative U.S. sample.

Several studies quantify health impacts from exposure to $PM_{2.5}$ during wildfires, but few examine mortality in California specifically. A recent study by Wang *et al.* (*8*) evaluating the economic footprint of the 2018 California wildfires conducted a health impact assessment for one portion of the analysis. They estimated 3652 premature deaths associated with wildfire $PM_{2.5}$ exposure (*8*), which is substantially lower than our estimates of 12,150 to 12,850 for 2018. While the discrepancy is likely partially due to varying modeled $PM_{2.5}$ exposure used in the two studies, it is primarily due to the use of differing dose-response values. Wang *et al.* estimated mortality using a combination of a 2013 California-specific dose-response estimate (*52*) and a well-established U.S. dose-response value from 2009 (*53*) commonly used in U.S. health impact analyses. Their analysis used the U.S. EPA's Environmental Benefits Mapping and Analysis–Community Edition (BenMAP-CE), which uses county-level health estimates. Our study builds on this California-specific analysis by (i) using more highly resolved health data, which can reduce potential misclassification of exposures associated with using spatially coarse health data; (ii) extending the temporal period of the health analysis (analyzing 11 years of data, instead of just 1); and (iii) applying a chronic wildfire-specific dose-response value.

Fann *et al.* (*32*) quantified long-term mortality and morbidity impacts throughout the entire country for 2008 to 2012, using the same commonly used U.S. dose-response value mentioned previously and the same CMAQ simulation we apply in this study (*53*). Although results for California are not explicitly presented, the authors reported that California is one of the several states in the country with the most notable mortality and respiratory morbidity impacts over the 5-year period (*32*). They estimated 14,000 premature deaths in the United States for the high-fire year of 2008 as compared to our estimates of ~10,000 (for both scenarios) in California alone. Again, our use of the wildfire-specific dose-response coefficient has also increased the magnitude of our results. In addition, like the California economic footprint study discussed previously, the U.S. study was limited by the use of county-level health data, which is again less spatially resolved than the ZIP code–level data used here.

## Implications of using modeled air quality estimates for health impact assessment

The scenario-specific analysis has several implications as well. We find that capping fire-only concentrations at the 99.9th percentile (exceeding 143 $\mu g/m^3$; see table S10) of values results in several hundreds to thousands of fewer fire-only $PM_{2.5}$ attributed deaths per year, but the overall magnitude of impacts is still substantial with the peak concentrations capped. The results vary little between the base case and mod cap scenarios in the lower fire years (especially 2009 to 2014), which indicates that these higher concentrations are occurring primarily in the high-fire years and are likely driven by severe fire events. As it is certainly possible for concentrations to reach and exceed 143 $\mu g/m^3$ (the 99.9th percentile value) during fire

events, capping these values would lead to an underestimate for the mod cap. In addition, the observed CMAQ model overprediction during fire events would lead to an overestimate for the base case. This is an uncertainty in using modeled data for health impact assessment, particularly for analyses in which the results can be affected by high concentration averages applied in dose-response analysis.

This variation in results between the base case and the mod cap and the differing magnitudes of our findings with the wildfire-specific versus undifferentiated dose-response value (Fig. 2) highlights several considerations and challenges associated with using modeled data for health studies. The implications and sensitivity associated with the choice of wildfire smoke exposure data and potential misclassification in relation to quantifying health impacts have been discussed in recent studies (*20*, *54*–*56*). One study found differing odds ratios for morbidity outcomes using three different methods of wildfire smoke estimation (Weather Research and Forecasting coupled with Chemistry, kriging, and geographically weighted ridge regression) (*55*). Another analysis that was focused on acute health impacts during the 2017 California wildfires used varying dose-response values and exposure surfaces to test the sensitivity of results (*54*). The authors found that there were no statistically significant differences in results for the variation in either input, but the differing magnitudes in outcomes resulting from the use of a range of dose-response values supported the use of context-specific dose-response values, as we have applied in this study (*54*).

### Contribution, strengths, and limitations

This study has several strengths and presents a unique contribution to the literature. The use of 11 years of CMAQ data enabled us to report on a long-term period of wildfire impacts in California, with several high-fire years with substantial impacts. The use of fire-only $PM_{2.5}$ estimates from the CMAQ model is a distinct strength of this study. Although recent machine learning analyses have parsed out wildfire-specific $PM_{2.5}$ at slightly more spatially resolved levels than our 12-km grid [10 km (*9*) and ZIP code (*15*)], uncertainty exists in these estimates due to a series of assumptions in the methodology. Both studies intersect the Hazard Mapping System Fire and Smoke Product (HMS Smoke) hand-drawn smoke plumes from satellite imagery with the various grids as a primary method of identifying smoke days. The HMS Smoke product, however, characterizes the density of smoke plumes in the atmospheric column and accordingly is not precisely aligned with ground-level $PM_{2.5}$ concentrations (*57*). Furthermore, the studies characterize the fire-only concentrations using undifferentiated $PM_{2.5}$ concentrations (from all sources) and the binary smoke day classification, which again requires several assumptions to extract fire-only $PM_{2.5}$ using counterfactual nonsmoke concentrations (*9*, *15*). The CMAQ modeled estimates applied in this study are subject to typical limitations associated with use of a CTM, but these values are based on actual all sources and nonsmoke modeled $PM_{2.5}$ and do not involve the use of imputation. The use of highly resolved health data at the ZIP code level is another key contribution of this study. Less spatially resolved county-level mortality rates are used in BenMAP-CE (*58*) and many existing health impact assessments, which can result in potential exposure misclassification, as mentioned previously. We also apply a fire-specific dose-response coefficient accounting for increased toxicity of wildfire smoke, which gives a first estimate of chronic wildfire-specific mortality impacts. In addition, the inclusion of two exposure scenarios enables us to evaluate the sensitivity of the magnitude of health impacts to high $PM_{2.5}$ concentrations from severe wildfire events.

Several limitations deserve mention. The CMAQ model is affected by typical challenges associated with the use of data inputs and procedures for modeling wildfire smoke using CTMs (*32*, *59*, *60*). We address model overprediction concerns by including mod cap, in which we remove modeled data outside of the 99.9th percentile of all values and develop a second set of mortality and valuation estimates to consider and discuss. In addition, the CMAQ model runs do not isolate wildfire emissions from prescribed burns. Therefore, the results presented here include mortality associated with all wildland fires (not including agricultural burns, which are not incorporated in the isolated fire-only fraction) and do not solely represent wildfires. Prescribed burns in California, however, account for a very small proportion of the total acres burned (*61*), although this may change in the future with ambitious targets for increased land management practices (*62*). For this study period, we do not anticipate a substantial portion of the mortality impacts to be attributable to prescribed burning.

In addition, we estimated a wildfire dose-response value, which enables us to account for the potentially increased toxicity of wildfire smoke. Some uncertainty exists with this approach because this dose-response value was not established through primary research (a long-term wildfire-specific estimate has not yet been developed) but instead was calculated using existing dose-response values; rationale for this approach is described in detail in the subsection Rationale for the dose-response function. With respect to the short-term wildfire-specific dose-response function used to estimate the final coefficient, we chose to use the variance-weighted average of two dose-response values for the short-term wildfire-specific dose-response coefficient, one from a Washington wildfires study (representative of wildfire conditions, $PM_{2.5}$ composition, and population in the western United States). We drew the second from a global study that estimated short-term mortality risk attributable to wildfire smoke exposures in 749 cities and provided a supplemental estimate for 210 U.S. cities (*38*). The main dose-response value presented in the global study, however, found mortality risk estimates of a higher magnitude than the Washington study and its own United States–based estimate; results using this dose-response value are included as a sensitivity analysis (table S6) and demonstrate approximately twice the mortality impact, with more than 100,000 deaths attributable to fire-$PM_{2.5}$ over the 11-year period (*38*). While the application of two U.S. dose-response values—as we have done here—is the most appropriate approach for this analysis, the inconsistencies between global and U.S. estimates highlight the need for further analysis to characterize the relationship between wildfire $PM_{2.5}$ exposure and both acute and chronic mortality impacts. Last, we used total mortality from all causes of death in our analysis, but one of the wildfire-specific studies from which we drew a short-term dose-response value only considered nontraumatic mortality (*27*), which accounts for ~94% of mortality across the state (*63*); however, the two other dose-response values included in our derived dose-response value used all-cause mortality to develop their estimates, so we do not anticipate that this would notably affect the results (*37*, *38*).

#### *Rationale for the dose-response function*

We created a dose-response function that mathematically combines dose-response functions from existing studies of long-term effects from undifferentiated $PM_{2.5}$ and acute studies of wildfire $PM_{2.5}$ and

undifferentiated $PM_{2.5}$. Although periodic and seasonal, wildfire smoke is now an ongoing exposure that affects much of the population on a regular basis (our analysis found that all ZIP codes in California are exposed to smoke each year). We were able to separate annual wildfire $PM_{2.5}$ concentrations from those generated by other sources such as traffic and industry by using a CTM for exposure assignment. This enabled us to estimate and compare the magnitude of effects attributable to wildfires and undifferentiated $PM_{2.5}$. Ideally, we would have been able to refer to studies that directly estimated the long-term mortality risk specific to wildfire $PM_{2.5}$; however, as previously mentioned, such estimates are not available in the literature. This approach raises three questions. First, could wildfire smoke elicit similar effects as undifferentiated $PM_{2.5}$, which would lead to long-term health impacts? Second, does sufficient evidence of differential toxicity exist to justify adjusting estimates for heightened toxicity of wildfire smoke given growing evidence in the epidemiological and toxicological literature (*25*, *27*, *38*)? Third, does the dosing regimen matter or are acute higher exposures over a few days equal to moderate exposures over a longer period of weeks or months if the total effective dose is the same?

Regarding the application of evidence on long-term effects, a limited number of studies suggest that wildfire smoke can be associated with other health effects, which could reduce survival chances. One study showed that lung function was reduced for 2 to 4 years after major wildfire smoke exposure (*64*). Studies of adolescent rhesus macaque monkeys who were housed outdoors during the massive 2008 wildfires in Northern California showed associations between early life exposures (7 to 8 weeks of age) and subsequent respiratory and immunological abnormalities in adolescence, including reduced lung function measures (*65*). Lung function and other measures of respiratory health have been associated with decreased survival (*66*, *67*). Moreover, a cohort study from Canada detected associations between exposure to wildfires over 10 to 20 years and cancer incidence (*68*). These findings suggest that wildfire $PM_{2.5}$ can generate long-term health effects capable of reducing survival chances, which would result in higher mortality for those living in areas with chronically higher wildfire $PM_{2.5}$ than those in areas with lower levels.

Studies on the chronic effects of undifferentiated $PM_{2.5}$ consistently show a larger dose-response function per unit of $PM_{2.5}$ mass than time series studies of acute effects (*69*). This likely occurs because the cohort study design with longer follow-up (multiple years) than time series studies (typically 0 to 3 days) captures both the long-term contribution of air pollution to disease formation and the acute mortality that occurs when susceptible individuals experience higher $PM_{2.5}$ exposure over periods of days (*69*). Under specific plausible conditions, an earlier study demonstrated that results from time series studies equal estimates from a dynamic population, similar to a cohort study, when each individual's survival experience is summarized as the daily number of deaths (*70*). These findings further emphasize two considerations: (i) short- and long-term effects are quantitatively similar and variations in observed effects are likely the result of differences in follow-up times between the time series and cohort designs and (ii) relying solely on the time series estimates of mortality (evaluating just a few days of exposure) to estimate the burden from wildfire $PM_{2.5}$ will likely underestimate the effects because the dose-response function fails to include the accumulated contribution of $PM_{2.5}$ to chronic diseases such as atherosclerosis, asthma, lung function decrements, and diabetes (*40*, *71*–*74*), all of which could contribute to higher mortality risk and push some individuals into the susceptible ranges where acute events would accelerate their death.

Following this and addressing the second question, we have thus modified existing estimates of long-term exposure to undifferentiated $PM_{2.5}$ with adjustment for potentially different dose-response functions of wildfires compared to undifferentiated $PM_{2.5}$. Evidence remains formative on the relative toxicity of $PM_{2.5}$ from wildfires versus other sources, but some toxicological evidence supports our approach because many of the same sub-acute mechanisms (very short term—usually less than a day) that have been documented for undifferentiated $PM_{2.5}$ show similar modes of action with wildfire smoke (*75*). Although mechanisms appear similar, some studies suggest that wildfire smoke may have higher toxicity than undifferentiated $PM_{2.5}$. Using in vivo mouse bioassays, a study on the toxicity of 2008 California wildfire particulate matter emissions found that exposures were more toxic to the lungs compared to particulate matter collected from ambient air not affected by wildfires (*36*). Wildfire $PM_{2.5}$ may have a greater potential to cause inflammatory and oxidative stress responses seen 6 to 24 hours after exposure, despite containing lower levels of polycyclic aromatic hydrocarbons than urban particulate matter concentrations (*76*, *77*). Compared to post-fire periods, samples collected during the 3 days of the October 2007 Southern California wildfires were found to have a higher number of particles larger than 0.1 μm in diameter as well as elevated levels of levoglucosan, water-soluble organic carbon, minerals (magnesium, phosphorus, potassium, and manganese), carbon monoxide, and nitrogen monoxide (*78*). The redox activity of $PM_{2.5}$ during the wildfire, measured using dithiothreitol (DTT) and macrophage reactive oxygen species (ROS) assays, indicated higher DTT activity, but ROS activity remained relatively similar to undifferentiated $PM_{2.5}$. In sum, wildfire $PM_{2.5}$ elicits many of the same sub-acute biological processes as undifferentiated $PM_{2.5}$, with some studies suggesting heightened toxicity in the lung, possibly due to differences in the physical-chemical composition of the particles. In attempting to estimate the health burden of wildfire $PM_{2.5}$, then, studies that rely solely on acute estimates from undifferentiated $PM_{2.5}$ may underestimate effects if wildfire $PM_{2.5}$ exhibits heightened toxicity. To overcome this limitation, we have included estimates of both the adjusted (estimated wildfire-specific chronic dose-response value, Eq. 2, under the assumption of increased toxicity) and unadjusted (undifferentiated chronic dose-response value) long-term mortality associated with fire-only $PM_{2.5}$ in Fig. 2, which shows the likely increases in mortality burden.

Epidemiological analyses based on respiratory hospital admissions in Southern California support the toxicological evidence, with findings indicating that wildfire smoke is up to 10 times more harmful to human health than PM from other sources (*25*, *79*). In addition, wildfire $PM_{2.5}$ appears to have larger effects on mortality than undifferentiated $PM_{2.5}$ (*38*, *80*). These studies provide valuable insights into the comparative toxicology of wildfire and undifferentiated $PM_{2.5}$, but the actual degree of increased toxicity is not fully resolved and likely complicated by the large variability in the fuel type and wildfire emissions, weather patterns, combustion profiles, and resulting physical-chemical composition, among others (*19*, *75*, *81*). Viewed together, however, these research findings indicate that wildfire $PM_{2.5}$ may have unique physical-chemical components that amplify its toxicity and therefore are potentially more harmful to human respiratory health compared to undifferentiated $PM_{2.5}$ from

nonwildfire sources. Again, current research supports our approach of adjusting for the higher dose-response observed from wildfire $PM_{2.5}$ exposure versus undifferentiated $PM_{2.5}$.

On the third question relating to the difference in the periodicity of wildfires compared to undifferentiated $PM_{2.5}$ from other sources, we do not have direct evidence of wildfires contributing to longer-term effects or those that are similar to more regular exposures from other sources. By analogy, however, we can consider major acute periods such as the London Fog, which had severely elevated levels of $PM_{2.5}$. Reanalyses of the London Fog data showed that the highly elevated exposures continued to exact a toll on mortality for months after the acute event had subsided (*82*). As with undifferentiated $PM_{2.5}$, acute exposure to particulate matter today can prompt a biological response, which alters the probability of survival, potentially several months or years after exposure. That is, the date of a person's death could be affected by their exposure several years ago. Purely acute time series studies cannot capture this type of long-term effect.

In sum, to estimate mortality impacts, we have assumed that particulate matter-associated health effects from any source are proportional to total inhaled dose of particles, a similar assumption made by the Global Burden of Disease program to establish the Integrated Exposure-Response model (*83*). Furthermore, we have assumed that particulate matter toxicity is independent of the dosing regimen (i.e., a few days of high exposure is equivalent to several days of moderate exposure) and only depends on total inhaled dose. Last, burden estimates are independent of the differences in the shape of the concentration-response function between undifferentiated $PM_{2.5}$ and wildfire $PM_{2.5}$, only depending on the magnitude of the slope of the log-linear model used to capture toxicity in time series studies. On the basis of the above reasoning, these assumptions appear to be supported by empirical findings and toxicological investigations. We recognize, however, that our approach to the dose-response function does not replace the need for future studies to directly investigate the chronic mortality effects of wildfire $PM_{2.5}$. Lacking this direct knowledge, our approach appears to be a more viable and accurate way to estimate the mortality burden from wildfire $PM_{2.5}$ than simply relying on time series studies, which likely would lead to an underestimate of the effects.

### Key areas for future study

Further study on these topics will be crucial as policymakers make efforts to reduce the widespread impacts of climate change on the environment and human health. Future work on air pollution modeling to parse out wildfire concentrations will enable more precision in health impact assessments. While a growing number of machine learning analyses discuss all sources (undifferentiated) $PM_{2.5}$ results in the context of wildfire smoke (*14*, *84*, *85*), only recently have models isolating fire-specific $PM_{2.5}$ been built (*9*, *15*). This is an area for research and development, including further comparison against typical CTMs to determine the best approaches to develop exposure surfaces for health analyses. Last, evaluating the equity dimensions of exposure and health outcomes is an area for future study. Another key implication of the substantial health and associated economic impacts from wildfires presented in this study is the importance of cultivating community resilience (*23*, *86*) and protecting vulnerable populations, who have less access to wildfire mitigation resources and reduced adaptive capacity (*23*, *87*). While many wildfire-prone regions are home to communities with lower social vulnerability (*88*), the intersection of wildfire health effects and equity will continue to grow in importance in the coming years as wildfires increase in severity and populations become more vulnerable to subsequent impacts. Considering the magnitude of the mortality impacts estimated here and the diverse population living in California, including many communities with preexisting vulnerability, this presents an opportunity for future research and evidence-based policy action to protect public health and promote equity.

In conclusion, this analysis characterizes the harmful impacts of $PM_{2.5}$ from wildland fire smoke on the health of the California population during the 11-year period of 2008 to 2018. This health impact analysis applies CTM estimates of wildland fire $PM_{2.5}$ to estimate mortality outcomes using high-resolution health data. This analysis is also innovative with respect to the long-term nature of the evaluation over an 11-year period and estimation and application of a chronic dose-response value for wildfire-specific $PM_{2.5}$ exposure. We estimate that between 52,480 and 55,710 premature deaths are attributable to fire-only $PM_{2.5}$ exposures, with an associated economic valuation of $432 billion to $456 billion. These findings have direct implications for California, a state at the forefront of climate policy development with many fire-prone regions and a diverse population to protect. Growing the evidence base on health impacts from wildfires and other climate-related exposures is critical in motivating future investments to mitigate the impacts of climate change and protect vulnerable populations.

## MATERIALS AND METHODS

### Data

#### *Modeled wildland fire $PM_{2.5}$ concentrations*

We used daily modeled $PM_{2.5}$ concentrations for 2008 to 2018 for the state of California at a 12-km grid spatial resolution, estimated using the U.S. EPA's CMAQ (version 5.0.1 to 5.3; see table S11) modeling system.

These wildland fire emissions estimates [which include wildfires and prescribed burns (but exclude agricultural burns), hereafter referred to as simply "fire"] incorporate multiple sources of fire activity (see table S11 for a full list of all data sources and specifications). SMARTFIRE2 (*89*) was used to reconcile the sources of fire activity data. Fuel consumption was calculated using the U.S. Forest Service's CONSUME version 3.0 fuel consumption model and the Fuel Characteristic Classification System fuel-loading database in the BlueSky framework (*90*). Emission factors were taken from the Fire Emission Production Simulator model. Nonfire emissions sources are from the National Emissions Inventory. The model was run with all emissions (fire and nonfire sources) and again without fires. The calculated difference between these simulations (all sources $PM_{2.5}$ and nonfire $PM_{2.5}$) isolates the fire contribution, or fire-only $PM_{2.5}$. The model simulations for 2008 to 2012 are the same as those used by Rappold *et al.* (*91*) and Fann *et al.* (*32*).

The first 5 years of data from 2008 to 2012 have been published by Wilkins *et al.* (*51*) and compared to other models in the literature (*5*); the remaining 6 years of data for 2013 to 2018 have not yet been reported in published studies. Therefore, we present a summary of all 11 years of data alongside the mortality and valuation analysis in this study. We compiled descriptive statistics for all 11 years of data, comparing all sources, fire-only, and nonfire $PM_{2.5}$ concentrations throughout the state and estimating the contribution of fires to all sources $PM_{2.5}$. We also investigate the impacts on air quality from fires within the context of days exceeding the NAAQS of daily

$PM_{2.5}$ > 35 μg/$m^3$ and years exceeding the annual NAAQS of 12 μg/$m^3$ (*51*).

In addition, a supplemental validation analysis comparing monthly average modeled concentrations to observed concentrations from ground station data is included in the Supplementary Materials (Supplementary Text) (*92*).

***Mortality data***

Statewide annual mortality data (total number of deaths) by ZIP code and age for all 11 years are managed by the CDPH and are publicly available on the California Health and Human Services Open Data Portal website (*63*). For several ZIP code and age categories, the count of deaths is suppressed for confidentiality reasons (i.e., counts <11). Therefore, we implemented substitution procedures to fill in the missing deaths. First, because we only apply the dose-response values to ages 25+ (due to the nature of the epidemiological analysis from which the dose-response values were derived), we calculated the percentage of deaths in people over 25 for the entire state for each year, which is ~98%. For the ZIP codes where the total number of deaths was available, but the total number of deaths by age group were suppressed due to low counts in each group, we multiplied that percentage (98%) by the total number of deaths in the ZIP code to estimate the number of deaths for the applicable age group. For ZIP codes where even the total number of deaths are suppressed, we conservatively assume that the ZIP code contains one-half of the suppression threshold and applied the percentage (98%) to that estimated value. We compared our final death count to the total reported deaths in the state (from the same CDPH data source) as a metric of quality assurance, and the total estimates varied by less than 0.35%.

**Mortality and associated economic valuation calculations**

We quantified the total mortality burden for exposure to $PM_{2.5}$ due to wildfires in California at the ZIP code level, using 11 years of CMAQ data (2008 to 2018). Based on the evaluation of the modeled data shown in the Supplementary Materials, we found that the highest modeled fire-only $PM_{2.5}$ values skew the correlations between the modeled and observed concentrations; thus, there is more uncertainty associated with those high concentrations. Therefore, we conducted two mortality analyses: (i) base case, with no outliers removed, to characterize the potential impact of extremely high wildfire concentrations on mortality; and (ii) mod cap, capping fire-only $PM_{2.5}$ concentrations falling outside of the 99.9th percentile of modeled values (at 143 μg/$m^3$; see table S11), considering that the model is expected to perform less reliably far outside of the dataset.

We averaged the daily fire-only $PM_{2.5}$ values to develop estimates for each year and grid cell and assigned exposures in each year to each ZIP code in California, using zonal statistics to estimate area-weighted averages for each ZIP code polygon from the annual $PM_{2.5}$ raster grid files for each year.

Then, we developed a wildfire-specific chronic dose-response coefficient (Eqs. 1 and 2 and see the Rationale for the dose-response function section in Discussion); the term "chronic" is also referred to as "long term" by some studies in the literature [e.g., Fann *et al.* (*32*)]. As mentioned previously, while there is substantial evidence regarding the impacts of exposure to wildfire-specific $PM_{2.5}$ on morbidity, such as respiratory outcomes (*21*, *39*), long-term mortality impacts from exposure to $PM_{2.5}$ from wildfire smoke—including how these impacts differ from exposure to ambient urban $PM_{2.5}$—are not established and identified as a substantial knowledge gap in the literature (*19*, *21*, *34*). To our knowledge, no existing studies have attempted to characterize the dose-response between chronic wildfire $PM_{2.5}$ exposure and mortality. A limited number of studies focus on characterizing the short-term (or acute) wildfire-$PM_{2.5}$ mortality relationship (*27*, *38*), with one study focused on the west coast of the United States evaluating short-term impacts from days with heavy ground-level smoke from wildfire events in Washington state (*27*) and another global study, which presents a United States–specific dose-response estimate along with the main global estimate (*38*). In addition, while there are no studies quantifying the relationship between chronic wildfire smoke exposure and mortality, several well-established dose-response values for the mortality impact of both chronic and short-term $PM_{2.5}$ exposures from undifferentiated (all sources) ambient $PM_{2.5}$ have been estimated. Existing short-term wildfire $PM_{2.5}$ dose-response values (*27*, *38*) demonstrate a more substantial impact on mortality than short-term undifferentiated dose-response values (*80*), providing evidence of potential increased toxicity of wildfire smoke. In addition, a small but growing evidence base has identified potential mechanisms for differential toxicity (*36*, *76*, *77*), and recent evidence from California has found differential increased impacts of wildfire $PM_{2.5}$ on human health outcomes as compared to ambient $PM_{2.5}$ (*25*). See the Rationale for the dose-response function section in Discussion for further details and a comprehensive discussion of evidence.

Therefore, the application of an undifferentiated dose-response value to wildland fire-specific $PM_{2.5}$ exposures may underestimate mortality impacts. To address this concern, and to avoid statistical shortcomings (e.g., mortality displacement) associated with quantifying mortality impacts from short-term, daily $PM_{2.5}$ exposures (*93*), we calculated a chronic dose-response value using Eqs. 1 and 2 below, which account for potential added toxicity of wildfire smoke as is suggested in several California-specific analyses (*25*, *36*). First, in Eq. 1, we pooled the two dose-response values: one from the previously mentioned Washington-based study and the other from the global study, which presented a U.S. estimate that was developed using data from 210 cities. Then, in Eq. 2, we calculated the adjusted wildfire-specific $PM_{2.5}$ dose-response value

$$\beta_{WS} = \frac{\left(\beta_{WA} \times \frac{1}{SE_{WA}{}^2}\right) + \left(\beta_{US} \times \frac{1}{SE_{US}{}^2}\right)}{\frac{1}{SE_{WA}{}^2} + \frac{1}{SE_{US}{}^2}} \quad (1)$$

$$\beta_{WL} = \frac{\beta_{WS}}{\beta_S} \times \beta_L \quad (2)$$

where $\beta_{WA}$ is the short-term wildfire $PM_{2.5}$ dose-response value from the Washington study (*27*), $\beta_{US}$ is the short-term wildfire $PM_{2.5}$ dose-response value for the United States from the global study (*38*), and $SE_{WA}$ and $SE_{US}$ are the two standard errors for those dose-response values, respectively. The result of Eq. 1, $\beta_{WS}$ is the variance-weighted average of the two short-term wildfire $PM_{2.5}$ dose-response values [Washington and United States; from the Washington state study, the dose-response estimate for lag day 1 (versus 0) was chosen because the only available U.S. estimate from the global study was a 0- to 2-day moving average] (*27*, *38*), $\beta_S$ is a short-term undifferentiated

$PM_{2.5}$ dose-response value from a recent meta-analysis (*80*), $\beta_L$ is a chronic (annual) undifferentiated $PM_{2.5}$ dose-response value from a recent country-wide cohort study (*37*), and $\beta_{WL}$ is the result: a chronic wildfire-specific $PM_{2.5}$ dose-response value (see table S12 for a list of the dose-response values used in our analysis, as extracted directly from the studies and standardized to the one-unit pollutant increment). We used a Monte Carlo distribution to estimate the final dose-response value used. We calculated a 95% CI for the estimated dose-response value.

Then, we calculated the mortality burden from exposure to $PM_{2.5}$ due to wildland fire smoke in the state of California using Eq. 3 below (*58*)

$$\Sigma\Delta m_{ij} = \left[1 - \frac{1}{e^{(\beta_{WL} \times \Delta PM_{2.5ij})}}\right] \times d_{ij} \quad (3)$$

where $\beta_{WL}$ is the result of Eq. 2 (dose-response value), $\Delta PM_{2.5ij}$ represents the change in $PM_{2.5}$ concentration from wildland fire smoke in year $i$ and ZIP code $j$, $d_{ij}$ represents the total deaths in adults ages 25 and up, and $\Delta m_{ij}$ represents the total mortality burden from wildland fires.

We also replicated the mortality calculations in Eq. 3 using solely the chronic undifferentiated $PM_{2.5}$ dose-response value from the U.S. national study conducted by Pope *et al.* (*37*) to characterize the differences when the dose-response value is not adjusted for the potential added toxicity of wildfire smoke (as we did in Eq. 2).

Last, we apply the EPA's value of a statistical life (VSL) to these mortality impacts to estimate the total valuation of the health burden, using Eq. 4 below

$$\text{Economic valuation} = \Sigma\Delta m_{ij} \times V \quad (4)$$

where $\Delta m_{ij}$ is the result of Eq. 3 (mortality burden from wildland fires) and $V$ is the EPA's VSL, which is $8.7 million in 2015 dollars (inflation year). We accounted for income growth to the year 2015 using publicly available income growth factors used in the U.S. EPA's BenMAP-CE tool (*58*) because changes in income can affect willingness to pay for reduced risk of mortality. Last, we applied a 3% discount rate over the 11-year period to estimate the net present value of our economic estimates (*94*).

We also conducted several supplemental mortality analyses to further contextualize our primary results. First, we developed mortality estimates associated with all sources $PM_{2.5}$ exposure, using $\beta_L$, the same chronic undifferentiated $PM_{2.5}$ dose-response value used in our primary analysis (*37*). Second, we estimated $\beta_{WL}$ using three alternative short-term wildfire $PM_{2.5}$ dose-response values: first, the primary dose-response result from the recent global study from which we extracted the U.S. value (used in our main estimate) (*38*); second, a calculated variance-weighted average combining a lag-based analysis presented in the global dose-response study and the Washington state study, using lag day 0 from both studies; and third, the same calculated variance-weighted average but using lag day 1 from both analyses.

## Supplementary Materials

**This PDF file includes:**

Supplementary Text
Figs. S1 to S9
Tables S1 to S15

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**Acknowledgments:** We acknowledge C. Garcia, N. Hernandez, B. Holmes-Gen, and B. Weller from the California Air Resources Board as well as K. Wyat Appel and N. Fann from the U.S. EPA for their advisement. **Funding:** This work was supported by the California Air Resources Board [grant no. 19RD015 (M.J.)], National Aeronautics and Space Administration (NASA) [grants nos. 80NSSC22K1684 and 80NSSC22K1480 (J.W. and M.E.M.)], DOE (BER) Award Research Development and Partnership (RDPP) [grant no. DE-SC0023511 (J.W.)], and UCLA Center for Healthy Climate Solutions (M.J.). **Author contributions:** Conceptualization: M.J., M.E.M., R.C., J.S., D.A.G.-G., R.T.B., and J.W. Methodology: M.J., M.E.M., R.C., and R.T.B. Formal analysis: R.C. Investigation: R.C. and J.W. Visualization: R.C. Supervision: M.J. and M.E.M. Writing—original draft: R.C. Writing—review and editing: R.C., M.J., M.E.M., D.A.G.-G., J.S., C.B., E.B., J.J., J.W., Y.Z., and R.T.B. Project administration: D.A.G.-G. **Competing interests:** The authors declare that they have no competing interests. **Data and materials availability:** The modeled air quality data that support the findings of this study are available at the following link: https://doi.org/10.5061/dryad.sxksn03b3. Publicly available mortality data for California are found here: https://cal-vida.cdph.ca.gov/ and https://data.chhs.ca.gov/dataset/death-profiles-by-zip-code. All data needed to evaluate the conclusions in the paper are present in the paper and/or Supplementary Materials.

Submitted 29 September 2023
Accepted 6 May 2024
Published 7 June 2024
10.1126/sciadv.adl1252