

Wildfire smoke PM_{2.5} and mortality rate in the contiguous United States: A causal modeling study

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Abstract

The relationships between chronic exposure to wildfire smoke PM_{2.5} (particulate matter with aerodynamic diameter of $\leq 2.5 \mu\text{m}$) and mortality remain poorly understood, with causal evidence being particularly scarce. In this ecological study, we used a doubly robust method, incorporating flexible generalized propensity score estimation that captured potential nonlinearity and interactions among confounders and relaxed the distribution form assumption for exposure, to estimate the effects of annual exposure to wildfire smoke PM_{2.5} on all-cause and cause-specific mortality in the contiguous United States from 2006 to 2020. We found that wildfire smoke PM_{2.5} was associated with increased mortality rate for all studied outcomes, except for deaths from transport accidents or falls, which served as negative outcome controls. Wildfire smoke PM_{2.5} was responsible for ~24,100 all-cause deaths per year in the contiguous United States. The exposure-response curve for all-cause mortality increased monotonically, with no evidence of a “safe” threshold. Among the six cause-specific outcomes, mortality from neurological disease showed the greatest increase per 0.1 $\mu\text{g}/\text{m}^3$ increase in smoke PM_{2.5} exposure. Our study provided robust evidence for the chronic effect of wildfire smoke PM_{2.5} on mortality, underscoring the urgent need for targeted measures to mitigate the substantial and escalating burden of wildfires.

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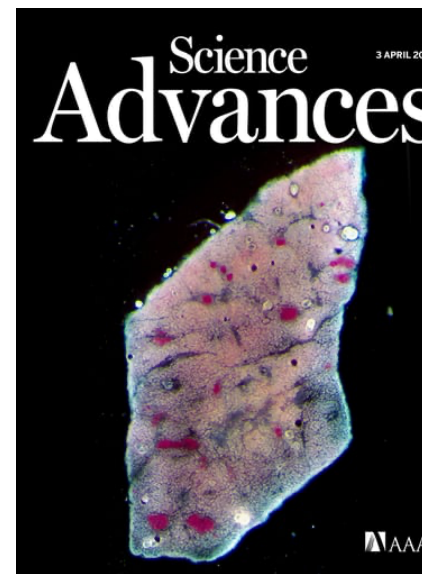
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INTRODUCTION

Wildfires, a major environmental hazard, are expected to intensify due to climate change (1), primarily driven by human activities (2, 3). Over recent decades, their mag-

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nitude, intensity, and frequency have risen sharply worldwide, with projections showing a global increase of extreme fires up to 50% by 2100 (4). Wildfires can cause large amounts of air pollutants in a short period of the onset and jeopardize air quality in places thousands of kilometers away through the migration of smoke. Of wildfire emissions, fine particulate matter (PM_{2.5}; particulate matter with aerodynamic diameter of ≤2.5 μm) is a primary pollutant, which can penetrate deep into the respiratory system and pass through the alveoli into the bloodstream (5). Wildfire smoke PM_{2.5} is considered more toxic to humans than nonsmoke PM_{2.5} due to its higher contents of carbonaceous and polar organic compounds, which can lead to respiratory illness, cardiovascular disease, neurological disease, and premature death (1, 6).

Existing epidemiological studies have documented that exposure to wildfire smoke PM_{2.5} is associated with an increased risk of all-cause mortality, but the findings regarding cause-specific mortality were mixed (7–12). In addition, most existing studies have focused on acute effects of wildfire smoke PM_{2.5}, with limited evidence available on chronic effects. Moreover, prior studies primarily fitted traditional regression models with the consideration of limited potential confounders, which is subject to confounding bias. These studies were observational and did not use statistical methods designed for addressing residual confounding and informing policy development (13). In recent years, causal modeling methods have been developed to address confounding in observational human studies by establishing a counterfactual framework in which confounders are balanced and exposure is independent of the outcome (14). To our knowledge, only two prior studies have used causal modeling, but they were specific to cardiovascular and cancer mortality, respectively (15, 16). Robust evidence regarding the impact of wildfire smoke PM_{2.5} on all-cause mortality and other cause-specific mortality within a causal modeling framework is largely lacking.

In this study, we examined the association between annual exposure to wildfire smoke PM_{2.5} and county-level mortality in the contiguous US. We used a doubly robust method that incorporated flexible generalized propensity score (GPS) estimation strategies to account for nonlinearities and interactions among measured confounders and relaxed the assumption for the distributional form of exposure. Furthermore, we used mortality from transport accident or falls as two negative outcome controls to test the presence of uncontrolled confounding. The exposure-response curve was plotted to visualize these relationships. Subgroup analyses were conducted to identify potential vulnerable groups. Our findings provided robust evidence for the impact of wildfire smoke PM_{2.5} on mortality and highlighted the urgency of implementing strategies to reduce wildfire-related mortality from increasing wildfire incidents.

RESULTS

From 2006 to 2020, an annual average population of 293,803,431 was recorded from 3068 counties across the contiguous US (Table 1). The annual average mortality rate for deaths from all causes, circulatory diseases, endocrine, nutritional and metabolic diseases, mental and behavioral disorders, neurological diseases, neoplasms, respiratory diseases, and transport accidents were 1077.2, 339.2, 49.4, 55.4, 72.3, 237.5, 114.1, and 16.0 per 100,000 persons, respectively.

Annual mortality rate per 100,000 persons	Min	Mean ± SD	Max
All causes	100.2	1077.2 ± 288.2	3498.7

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Table 1. County-level annual mortality rate in the contiguous US, 2006–2020.
The county-level annual mortality rates were obtained from CDC WONDER.

The distributions of annual county-level characteristics are listed in [Table 2](#). Over the study period from 2006 to 2020, the annual concentrations of wildfire smoke PM_{2.5} at the county-level averaged 0.4 µg/m³, with a median (interquartile range) of 0.3 (0.2, 0.6) µg/m³. The nonsmoke PM_{2.5} concentrations and normalized difference vegetation index (NDVI) were, on average, 7.9 µg/m³ and 0.5, respectively. The average temperature was 29.9°C for summer and 7.5°C for winter. Summer precipitation averaged 3.4 mm, and winter precipitation averaged 2.7 mm.

Characteristic	Min	10th per- centile	25th per- centile	Mean	Median	75th per- centile	90th per- centile	Max
Wildfire smoke	0.0	0.1	0.2	0.4	0.3	0.6	0.8	18.6

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Table 2. Distribution of annual county-level characteristics in the contiguous US, 2006–2020.

- * The average concentrations of smoke PM_{2.5} and nonsmoke PM_{2.5} were calculated based on the whole-year daily value.
- † The seasonal average temperature and precipitation based on 3-month daily averages (June, July, and August for summer, and December, January, and February for winter).
- ‡ The annual average NDVI data were calculated from the monitoring MODIS data at 16-day intervals per year.
- § The population characteristics are neighborhood-level statistics from the US Decennial Census and American Community Survey.

On average each year, 83.2% of the population was under 65 years old, 83.4% were white, 62.1% were female Medicare enrollees who had a mammogram, and 81.2% had a blood lipids test ([Table 2](#)). The annual average percentage of women and the population who graduated high school as their highest level of education was 50.1 and 51.1%, respectively. In contrast, the annual average percentage of Black individuals and the population living below the poverty line were 9.4% and 15.9%, respectively.

The effect estimates associated with annual wildfire smoke PM_{2.5} exposure were presented as change in mortality rate per 0.1 µg/m³ increase in wildfire smoke PM_{2.5}, as a 0.1 µg/m³ increase in annual wildfire smoke PM_{2.5} is more realistic and likely to occur. Shown in [Fig. 1](#), each 0.1 µg/m³ increase in annual wildfire smoke PM_{2.5} exposure was associated with an increase of 1.904 [95% confidence interval (CI): 1.616 to 2.192] for all-cause deaths per 100,000 persons. We also observed positive associations between annual wildfire smoke PM_{2.5} exposure and annual mortality from neurological diseases, circulatory diseases, neoplasms, endocrine, nutritional and metabolic diseases, respiratory diseases, and mental and behavioral disorders. Among these cause-specific deaths, wildfire smoke PM_{2.5} had the most substantial impact on mortality from neurological diseases, with an estimated rate difference of 0.334 deaths per 100,000 persons (95% CI: 0.288 to 0.380), followed by circulatory diseases (0.186, 95% CI: 0.080 to 0.293) and endocrine, nutritional and metabolic diseases (0.181, 95% CI: 0.144 to

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0.217). Furthermore, an increase in wildfire smoke PM_{2.5} concentrations by 0.1 µg/m³ was associated with an increase of 0.167 (95% CI: 0.092 to 0.242) deaths per 100,000 persons for neoplasms, 0.082 (95% CI: 0.035 to 0.128) deaths per 100,000 persons for mental and behavioral disorders, and 0.080 (95% CI: 0.027 to 0.133) deaths per 100,000 persons for respiratory diseases. The results from our negative outcome control analyses showed null associations between wildfire smoke PM_{2.5} and mortality from transport accidents or falls, with estimated rate differences of -0.005 (95% CI: -0.022 to 0.013) and 0.003 (95% CI: -0.021 to 0.027) deaths per 100,000 persons per 0.1 µg/m³ increase in wildfire smoke PM_{2.5}, respectively.

Fig. 1. Absolute increase in mortality rate 100,000 persons per 0.1 µg/m³ increase in annual wildfire smoke PM_{2.5}.

The Bonferroni correction was used to adjust 95% CIs. The model was adjusted for nonsmoke PM_{2.5}, year, summer temperature, winter temperature, summer precipitation, winter precipitation, percentage of age under 65 years old, percentage of women, percentage of white, percentage of Black, percentage of the population who graduated high school as their highest level of education, percentage of the population living below the poverty line, percentage of women who had a mammogram, percentage of people who had a blood lipids, NDVI, and average RUCA score.

The estimated annual attributable mortality burden by wildfire smoke PM_{2.5} is shown in [Table 3](#). Each 0.1 µg/m³ increase of annual wildfire smoke PM_{2.5} concentrations was responsible for about 5594 (95% CI: 4749 to 6440) all-cause deaths per year. This translated to a total of 24,054 all-cause deaths per year (95% CI: 20,421 to 27,520) attributable to wildfire smoke PM_{2.5} in the contiguous US. For other specific causes, there are ~981 (95% CI: 846 to 1115) deaths from neurological diseases, 547 (95% CI: 234 to 859) deaths from circulatory diseases, 530 (95% CI: 424 to 637) deaths from endocrine, nutritional and metabolic diseases, 490 (95% CI: 269 to 712) deaths from neoplasms, 240 (95% CI: 103 to 377) deaths from mental and behavioral disorders, and 235 (95% CI: 80 to 391) deaths from respiratory diseases attributable to wildfire smoke PM_{2.5} per year for each 0.1 µg/m³ increase in wildfire smoke PM_{2.5} concentrations.

Mortality rate	Annual attributable deaths (95% CI)
All causes	5594 (4749, 6440)

Table 3. The attributable annual deaths in the contiguous US by each 0.1 $\mu\text{g}/\text{m}^3$ increase in wildfire smoke $\text{PM}_{2.5}$ exposure.
Total attributable deaths were calculated by rate difference $\times$ total population.

The estimated exposure-response curves between wildfire smoke $\text{PM}_{2.5}$ and mortality rate can be seen in Fig. 2. We observed a monotonically increasing relationship between wildfire smoke $\text{PM}_{2.5}$ and all-cause mortality, with slightly steeper increases as exposure levels rose and no evidence of a “safe” threshold below which the effect was absent. The relationships between wildfire smoke $\text{PM}_{2.5}$ and mortality from neurological diseases and endocrine, nutritional and metabolic diseases were nearly linear. As for circulatory diseases, neoplasms, mental and behavioral disorders, and respiratory diseases, we observed J-shaped exposure-response relationships, where mortality initially decreased slightly before rapidly increasing as exposure levels increased.

Fig. 2. Exposure-response curves between annual wildfire smoke $\text{PM}_{2.5}$ and mortality rate (per 100,000 persons).

The solid lines denote point estimates and shaded areas denote the corresponding 95% CIs. The y axis is the estimated value of mortality rate per 100,000 persons. For uniformity, the x axis of all graphs was constrained to 8.30 $\mu\text{g}/\text{m}^3$; see the graph without constraints in the fig. S2.

As a sensitivity analysis, the positive associations between wildfire smoke $\text{PM}_{2.5}$ and mortality rate persisted when we trimmed the stabilized inverse probability weighting (IPW) at the 2.5th and 97.5th percentiles, instead of first and 99th percentiles for the main analysis, with slight increases of the effect estimates in all models, except for mortality from endocrine, nutritional and metabolic diseases (table S1). Furthermore, the estimated exposure-response patterns remained largely unchanged, except that the decline in mortality rates for neoplasms, mental and behavioral disorders, and respiratory diseases with increasing exposure became negligible at a lower concentration range in the J-shaped exposure-response curve (fig. S1). Positive associations with most outcomes remained when using a linear regression model to estimate GPS, although the effect estimates became smaller (table S2). An exception was mortality from circulatory diseases, for which negative association was observed. The associations with mortality from transport accidents or falls were still statistically nonsignificant in these sensitivity analyses.

In subgroup analyses, we observed that the effect of wildfire smoke $\text{PM}_{2.5}$ on mortality rate from all causes, circulatory diseases, neoplasms, mental and behavioral disorders, and endocrine, nutritional and metabolic diseases was more pronounced among communities with a higher percentage of the population under 65 years old (percentage of age under 65 years old > 75th percentile) (Fig. 3; numeric data in the Supplementary Materials). Similarly, significantly higher effect estimates for mortality rate from all causes, neoplasms, and endocrine, nutritional and metabolic diseases were observed among those living in more rural areas [average rural-urban commuting area (RUCA) score > 75th percentile; RUCA reflects population density, degree of urbanization, and

commuting patterns, which together represent the rural-urban characteristics of a region]. Furthermore, the associations between wildfire smoke $PM_{2.5}$ and mortality rate were stronger during periods of lower temperatures in both summer and winter (summer temperature and winter temperature $\leq$ 75th percentile).

Fig. 3. Subgroup analyses of the associations between annual wildfire smoke $PM_{2.5}$ and mortality rate (per 100,000 persons).

The P values between groups were calculated by pairwise comparisons of coefficients, and statistically significant differences are indicated using asterisks, with one, two, and three asterisks representing P values less than 0.05, 0.01, and 0.001, respectively. Numerical data are shown in tables S3 to S6.

DISCUSSION

Despite the increasing wildfires driven by the rapidly changing climate, the shift in US climate policy starting in 2025 is halting federal climate action and jeopardizing global mitigation efforts, posing a critical and underappreciated risk to climate progress (17, 18). Our findings on the chronic effects of wildfire smoke $PM_{2.5}$ on mortality in the contiguous US highlights the serious threat to human health and the urgent need for effective mitigation strategies. We used a doubly robust approach with flexible GPS estimation strategies to account for nonlinearities and interactions among measured confounders, while relaxing the distributional assumption for exposure. Null associations between wildfire smoke $PM_{2.5}$ and two negative outcome controls provided reassurance that the effects were not substantially biased from uncontrolled confounding. We observed that exposure to wildfire smoke $PM_{2.5}$ was associated with increased mor-

tality from all causes, neurological diseases, circulatory diseases, neoplasms, endocrine, nutritional and metabolic diseases, respiratory diseases, and mental and behavioral disorders. This increase corresponded to ~5594 (95% CI: 4749 to 6440) all-cause deaths per year for each 0.1 $\mu\text{g}/\text{m}^3$ increase in annual wildfire smoke $\text{PM}_{2.5}$ concentrations. This translated to a total of 24,054 all-cause deaths attributable to wildfire smoke $\text{PM}_{2.5}$ in the contiguous US per year. For all studied outcomes, the exposure-response curves generally showed an increasing trend as exposure levels rose, especially at higher exposure levels. In addition, greater effects of wildfire smoke $\text{PM}_{2.5}$ were observed in communities with a higher percentage of the population under 65 years old, in more rural areas, and during lower summer and winter temperatures.

Although existing epidemiological studies support a consistent association between wildfire smoke $\text{PM}_{2.5}$ and all-cause mortality (19–24), many were time-series studies that did not capture long-term effects, and the findings about the affected causes of mortality remained inconsistent. Jegasothy *et al.* (10) found a significant association between exposure to wildfire smoke $\text{PM}_{2.5}$ and an increased risk of all-cause death in Sydney, Australia. Both a global and a Brazil-based time-series study reported that higher wildfire smoke $\text{PM}_{2.5}$ exposure was associated with increased risks of all-cause, cardiovascular, and respiratory mortality (7, 12). However, using a Cox proportional hazards regression model based on the UK Biobank cohort, Gao *et al.* (8) found that wildfire smoke $\text{PM}_{2.5}$ was associated with increased risks of all-cause, nonaccidental, and neoplasm mortality, but not cardiovascular-, respiratory-, or mental health-related mortality. More recently, Ma *et al.* (11) used a panel fixed-effects model to explore the impact of annual exposure to wildfire smoke $\text{PM}_{2.5}$ on mortality in the contiguous US from 2007 to 2020. They observed that nonaccidental, cardiovascular, endocrine, and mental mortality rates increased to varying degrees during months in which the 12-month moving average of smoke $\text{PM}_{2.5}$ concentrations exceeded 0.1 $\mu\text{g}/\text{m}^3$, relative to months with average concentrations below this threshold. In contrast, no associations were observed with neurological or respiratory mortality rates (11). The nuances across these prior findings may be explained by the difference in study population, exposure levels, the method for estimating $\text{PM}_{2.5}$ concentrations from wildfire origin, and outcome assessment. Furthermore, the aforementioned studies only considered limited covariates (e.g., temperature and humidity) and did not fully address the nonlinearities and interactions among measured confounders or potential bias from uncontrolled confounders.

The doubly robust method used in our study combined the outcome regression and IPW, offering the advantage that the estimator remained valid even if either the GPS model or the outcome regression model was misspecified (25, 26). This offered two opportunities to correctly, or at least approximately correctly, specify one of the two models, instead of relying solely on a correctly specified outcome regression model. Consistent with our findings, the only two causal modeling studies to date using the variant difference-in-differences method have reported that wildfire smoke $\text{PM}_{2.5}$ was associated with cardiovascular mortality (15) and neoplasm mortality (16) in Brazil. Furthermore, to detect the presence of uncontrolled confounding, we performed analyses using two negative outcome controls, which explored the relationships of the exposure with two independent outcomes that should theoretically be unaffected by smoke $\text{PM}_{2.5}$ (27). We found little evidence of the associations between wildfire smoke $\text{PM}_{2.5}$ and the negative outcome controls of mortality from transport accident or falls, which further suggests that our findings for studied outcomes are reliable, with little biases from uncontrolled factors. Together, our findings suggest chronic effects of wildfire smoke $\text{PM}_{2.5}$ exposure and all-cause and six cause-specific mortality outcomes. In contrast, when GPS was estimated using linear regression, the sensitivity analysis showed a protective effect of wildfire smoke $\text{PM}_{2.5}$ and circulatory mortality.

This may partly reflect the limitations of traditional linear regression model in capturing nonlinearities and interactions among confounders, as well as their reliance on strict distributional assumptions for the independent variable.

We found that ~5600 all-cause deaths per year are attributable to each 0.1 $\mu\text{g}/\text{m}^3$ increase in annual wildfire smoke $\text{PM}_{2.5}$ in the contiguous US. A 0.1 $\mu\text{g}/\text{m}^3$ increase in annual wildfire smoke $\text{PM}_{2.5}$ is entirely plausible, as the national average during the study period was 0.43 $\mu\text{g}/\text{m}^3$ with a standard deviation of 0.48 $\mu\text{g}/\text{m}^3$. This is particularly concerning when compared to our previous studies, which found that each 0.1 $\mu\text{g}/\text{m}^3$ increase in annual all-sourced $\text{PM}_{2.5}$ concentrations contributed to ~1154 all-cause deaths among US Medicare participants (26, 28). This suggests that wildfire smoke $\text{PM}_{2.5}$ was approximately five times more toxic than general $\text{PM}_{2.5}$. The estimated total all-cause mortality burden associated with wildfire smoke $\text{PM}_{2.5}$ was 24,054 deaths per year (95% CI: 20,421 to 27,520) in the contiguous US, which is broadly comparable to the 11,415 nonaccidental deaths per year (95% CI: 6754 to 16,075) reported by Ma *et al.* (11), considering differences in modeling methods and the fact that all-cause mortality generally exceeds nonaccidental mortality. Moreover, the estimated exposure-response curves for all-cause mortality showed rapid increases with rising exposure, while the curves for other cause-specific mortality exhibited a monotonic increase at the higher exposure level, despite slight fluctuations at lower levels. From a long-term public health perspective, these results highlighted the critical need to control wildfire smoke $\text{PM}_{2.5}$ levels to prevent premature deaths and mitigate public health risks. Despite this urgency, wildfire smoke $\text{PM}_{2.5}$ is excluded from regulatory attainment determinations under the US Environmental Protection Agency (EPA), as wildfires are classified as natural disasters and thus fall outside the control of local authorities. However, most wildfires are initiated by human activities, and prescribed burns are known to limit the risk. Such procedures should be more widely used, which require local control and regulatory authority.

We observed that wildfire smoke $\text{PM}_{2.5}$ had the strongest effect on neurological disease mortality, followed by circulatory mortality and cancer mortality. With an aging population, the prevalence of neurological disease, particularly stroke and dementia, is likely to increase substantially in the next two decades. If so, that wildfire impacts will grow. $\text{PM}_{2.5}$ can initiate oxidative stress and inflammation in the nervous system (29). Studies of dogs and deceased children in Mexico have found $\text{PM}_{2.5}$ to be present in postmortem brain tissue, with evidence of inflammation surrounding the particles (30–32). Compared to nonsmoke $\text{PM}_{2.5}$, wildfire smoke $\text{PM}_{2.5}$ has a smaller average particle size, which may contribute to its unique toxic effects on the nervous system as smaller particles are more likely to cross the blood-brain barrier (5). This is further supported by the findings of Elser *et al.* (33), who reported that a 1 $\mu\text{g}/\text{m}^3$ increase in the 3-year average of wildfire smoke $\text{PM}_{2.5}$ exposure was associated with an 18% increase in the odds of incident dementia, whereas only a 1% increase in dementia risk was observed for a 1 $\mu\text{g}/\text{m}^3$ increase in nonsmoke $\text{PM}_{2.5}$ exposure among 1.2 million older residents from Southern California. Another plausible explanation is that individuals with neurological disorders represent a vulnerable population due to limited capacity to reduce exposure and comorbidities that are common with such disorders. Our findings deserve further study given that existing studies suggest that wildfire smoke $\text{PM}_{2.5}$ typically has a greater effect on the respiratory and cardiovascular diseases (34, 35). More research examining the relationship between wildfire smoke $\text{PM}_{2.5}$ and mortality due to neurological conditions is needed, especially dementia (36).

Our subgroup analyses indicated a stronger mortality effect associated with wildfire smoke $\text{PM}_{2.5}$ among communities with a higher percentage of population under 65 years old. This finding may be partly explained by the fact that older adults (≥ 65 years)

are likely to spend considerably more time indoors compared to younger individuals (e.g., those in their 20s or younger) (37), resulting in lower levels of exposure. Moreover, they are more likely to adopt healthier lifestyles, such as lower rates of smoking and binge drinking (38, 39), which may attenuate mortality risk caused by smoke PM_{2.5}. Circulatory mortality was the leading cause-specific mortality related to wildfires smoke PM_{2.5} in these younger communities. One possible explanation is that young adults are experiencing a significantly increased burden of cardiovascular disease (40), which could interact with wildfire smoke PM_{2.5} exposure and amplify its effects. However, this indicator (percentage of population under 65 years old) is collected at the neighborhood level, rather than the individual level; thus, the possibility that the true vulnerable population was obscured by neighborhood-level factors cannot be ruled out. In addition, we observed that people living in more rural areas were at a higher risk of death from wildfire smoke PM_{2.5}. These areas may experience higher concentrations of wildfire smoke due to their proximity to the wildfire sources. Furthermore, rural areas often have fewer medical resources, lower health awareness, and limited economic resources, which may lead to delayed or inadequate treatment and, consequently, higher mortality. Similar findings have been observed in the previous studies on general air pollution and mortality (41, 42). In the temperature-stratified analyses, larger effects of wildfire smoke PM_{2.5} on mortality were observed during lower temperatures in both summer and winter. Evidence has found that temperature is an important risk factor of mortality, with lower temperatures associated with greater mortality risks compared to higher temperatures (43). Moreover, people are more likely to exercise outdoors during cooler summer temperatures, while colder winter temperatures are more prone to temperature inversions that hinder the dispersion of wildfire smoke PM_{2.5} (44). Both factors may contribute to increased exposure, which further exacerbate mortality effects. We observed subtle differences in exposure distribution across subgroups (figs. S3 to S6), which may also support the interpretation of the heterogeneity in subgroup-specific effects. Identifying susceptible groups is crucial for addressing health equity and developing effective mitigation strategies. For example, enhancing air quality monitoring and health resources in rural areas, providing smoke-day warning in communities with a younger population, and implementing air purification during periods of lower winter and summer temperatures may help mitigate the risk of premature death from wildfire smoke PM_{2.5}. Nevertheless, caution is warranted when extrapolating these results, particularly in subgroups with small sample sizes, despite good covariate balance achieved in most subgroups (figs. S7 to S10).

One of the major strengths of this study is the use of an advanced doubly robust model combined with random forests and a kernel density estimator, along with adjustment for the extensive set of confounders, which enhances the control of confounding bias and yields less biased effect estimates. Our model has effectively balanced the relationship between the confounders and the exposure, as evidenced by the majority of the correlation coefficients between them becoming smaller when weighted by the estimated GPS or the stabilized IPW and falling within a narrow range of ± 0.1 (figs. S7 to S11). Kernel density estimator is particularly effective for skewed distributions, capturing the underlying structure of right-skewed exposure, while the exposure distribution after IPW resembles the original exposure distribution, further supporting that we were successful in eliminating covariate bias on exposure (fig. S12). Another strength is that the potential bias from uncontrolled confounders was tested using two negative outcome controls. Moreover, to the best of our knowledge, our results fill the gaps in existing studies on exposure-response curves as well as subgroup analyses. Our results provide direct and robust evidence for mortality related to wildfire smoke PM_{2.5}, which could inform public health recommendations to reduce wildfire smoke PM_{2.5} exposures for preventing premature death and regulatory considerations for wildfire occurrence.

Our study also has limitations. First, the daily estimates of wildfire smoke $PM_{2.5}$ primarily relied on satellite-based smoke plume identification. As a result, this approach may underestimate wildfire smoke $PM_{2.5}$ concentrations on days when plumes are not visible to satellites, such as during long-range transport or when smoke is aged and diffused. Furthermore, although the prediction of daily concentrations of wildfire smoke $PM_{2.5}$ was at 10-km grid cells, the exposure measures used in this study were annual county-level averages. The large temporal and spatial span of aggregated exposure measures may raise the issue of nondifferential misclassification of exposure, thereby potentially underestimating the effect of wildfire smoke $PM_{2.5}$ (45). Second, death records of fewer than 20 per year were excluded from our analysis because the US Centers for Disease Control and Prevention Wide-ranging Online Data for Epidemiologic Research (CDC WONDER) flagged them as unreliable. This exclusion may limit the generalizability of our findings to less populated counties. Third, given the J-shaped associations between wildfire smoke $PM_{2.5}$ and several cause-specific mortality outcomes, our reported effect estimates may simplify the true relationships and should be interpreted with caution. Last, besides $PM_{2.5}$, other pollutants derived from wildfire smoke (e.g., organic gases and NO_2) and mental health impacts related to wildfires (e.g., psychological trauma from experiencing wildfires) were not considered in this study, nor were their potential interactions. Alternatively, there is a possibility that the associations we observed for wildfire smoke $PM_{2.5}$ might be a proxy of these pollutants or mental health impacts.

In summary, our study characterized the detrimental effects of chronic exposure to wildfire smoke $PM_{2.5}$ on mortality in the contiguous US. By leveraging a causal modeling method and incorporating double-negative outcome control analyses, we improved covariate balance and detected potential bias from unmeasured confounders, thereby enhancing the robustness of our results. As wildfires become more frequent and widespread, our findings point to the need for targeted interventions to reduce the long-term mortality burden of wildfire smoke $PM_{2.5}$, particularly in rural areas, younger communities, and during cooler periods in both summer and winter, where the effects appear to be more pronounced.

MATERIALS AND METHODS

Study population and mortality data

Mortality data were extracted from CDC WONDER, covering 3068 counties in the contiguous US from 2006 to 2020. The annual county-level crude mortality rate per 100,000 persons was collected for analyses, with mortality rate based on death count fewer than 20 excluded due to their classification as unreliable. The primary causes of death were coded based on the International Statistical Classification of Diseases and Related Health Problems—10th Revision (ICD-10). Specifically, the mortality outcomes of interest included all causes (A00–Y89), neoplasm causes (C00–D48), endocrine, nutritional and metabolic diseases (E00–E88), mental and behavioral disorders (F01–F99), diseases of the neurological system (G00–G98), diseases of the circulatory system (I00–I99), and diseases of the respiratory system (J00–J98). In addition, mortality from transport accidents (V01–V99) or falls (W00–W19) was selected as negative outcome controls, as they are likely to share some of the same potential sources of bias with the exposure and outcome of interest, such as socioeconomic status and psychosocial stress (46, 47), but are not plausibly related to wildfire smoke $PM_{2.5}$ (27).

Exposure data

The annual mean concentrations of county-level wildfire smoke $PM_{2.5}$ during the study period were calculated based on daily local-level estimates developed by Stan-

ford's Environmental Change and Human Outcomes (ECHO) Laboratory (48). In brief, smoke-affected days were identified using satellite imagery-based plume classification and simulated air trajectories from fire locations. On these smoke-affected days, anomalous increases in PM_{2.5} concentrations relative to background levels were attributed to wildfire smoke. For locations without monitors, a machine learning combining additional available data and reanalysis datasets was built to predict daily wildfire smoke PM_{2.5} estimates. Last, daily ground-level wildfire-driven PM_{2.5} concentrations at a spatial resolution of 10 km by 10 km across the contiguous US were estimated. The model showed reasonable predictive performance with a spatial overall out-of-sample R^2 of 0.67 and an in-sample (within) R^2 of 0.65. The intersection-weighted wildfire smoke PM_{2.5} concentrations for population and area were assigned to each spatial scale. The spatial-scale predictions of wildfire smoke PM_{2.5} were aggregated to the county level, and daily predictions for each calendar year were averaged to obtain the annual concentrations.

Confounders

A wide range of variables including calendar year, neighborhood-level statistics, primary health care access, rural-urban commuting, nonsmoke PM_{2.5}, meteorological factors, and NDVI were considered as potential confounders. Among these variables, calendar year and rural-urban commuting accounted for temporal variations and rural-urban differences, respectively. Neighborhood-level statistics, meteorological factors (i.e., seasonal temperature and precipitation), and NDVI (an indicator of greenness exposure) were related to pollution levels and/or mortality (49, 50). In addition, primary health care access is a well-established predictor of mortality and was closely related to socioeconomic status.

Neighborhood-level statistics, including percentage of age under 65 years old, percentage of women, percentage of white, percentage of Black, percentage of the population who graduated high school as their highest level of education, and percentage of the population living below the poverty line, were drawn from the US Decennial Census for the years 2000 (51, 52) and 2010 (53) and American Community Survey for the years 2011–2019 (54). Missing values for 2001–2009 are estimated using linear interpolation between data from 2000 and 2010.

Primary health care access was assessed using two indicators: the annual percentage of women Medicare enrollees aged 67 to 69 who had a mammogram over a 2-year period, and the annual percentage of Medicare enrollees with diabetes aged 65 to 75 who had a blood lipids test in a year. These data from 2006 to 2020 were obtained from Dartmouth Health Atlas (55). The 2010 RUCA code, obtained from US Department of Agriculture, Economic Research Service, was used to classify US census tracts on the basis of urbanization, population density, and daily commuting (56). The primary RUCA code is integers from 1 to 10, with higher values indicating a shift from metropolitan to rural areas. The county-weighted average RUCA score was calculated by aggregating and averaging the primary RUCA code for each county, weighted by the population of each census tract.

Nonsmoke PM_{2.5} concentrations were calculated by subtracting wildfire smoke PM_{2.5} from the all-sourced PM_{2.5}, with the calculated negative values (accounting for 4.16% of the data) excluded from our analyses. The daily ground-level all-sourced PM_{2.5} data were obtained from the US High Air Pollutants (USHAP) (57). This database used a deep learning model that combines big data from satellite remote sensing products, ground-based observations, model simulations, and atmospheric reanalysis to estimate all-sourced PM_{2.5} data in the US from 2000 to 2020 at 1-km² grid cells, and the

yielded estimates align well with ground-based measurements [average cross-validation: $R^2 = 0.82$, normalized root mean square error (RMSE) = 0.40]. Consistent with wildfire smoke PM_{2.5}, the annual averages of county-level all-sourced PM_{2.5} concentration were calculated.

The meteorological factors, including temperature and precipitation, were considered for both summer and winter. Daily data on minimum temperature, maximum temperature, and precipitation were obtained from Daymet Version 4 database developed by Oak Ridge National Laboratory, which estimated weather parameters over North America at a 1 km-by-1 km spatial resolution (58). These daily gridded data were aggregated to county level by averaging values from grids whose centroids fell within county boundaries. The daily mean temperature was the average of minimum and maximum temperatures for each day, and the seasonal average temperatures for winter (January and February at the beginning of the year, and December at the end of the year) and summer (June, July, and August) were calculated. The average precipitation in summer and winter is also calculated by taking seasonal averages.

The NDVI data, monitored every 16 days at 250-m grid cells, were obtained from the Terra Moderate Resolution Imaging Spectroradiometer (MODIS) Vegetation Indices Version 6.121 (59). We aggregated the gridded NDVI data to annual county-level averages and adjusted them in all analyses.

Statistical analysis

We used a two-stage doubly robust analysis approach to estimate the effect of wildfire smoke PM_{2.5} exposure on mortality rate. In the first stage, we predicted the expected level of exposure given all measured confounders using random forests, which captured all potential nonlinearities and complex interactions among confounders (45). To tune the random forest model effectively and mitigate overfitting, fivefold cross-validation was conducted, and the configuration with the lowest RMSE was selected (see table S7 for full hyperparameter details). Then, considering the highly skewed distribution for wildfire smoke PM_{2.5} concentrations (Table 2), we used kernel density estimation to estimate the probability density of the difference between the expected and observed exposure levels. The smoothed residual from kernel density estimation was interpolated into a function and thus relaxed the key assumption about the form of the distribution of exposure residuals. The formula for calculating GPS is as follows: $GPS_i = \hat{f}_e[Y_i - f(X_i)]$, where $\hat{f}_e(\cdot)$ is the residual-based kernel density estimation function, Y_i is the observed exposure level for county year i , and $f(X_i)$ is the expected exposure level predicted from random forests. This estimated GPS reflects the probability of being exposed to the observed smoke PM_{2.5} level after accounting for measured confounders (60).

In the second stage, we fitted a generalized additive model incorporating IPW to obtain an unbiased estimate. The *stabilized IPW* was calculated using the following formula

$$stabilized\ IPW = \frac{K(x_i - \bar{x}_i)}{x_i}$$

in which $K(\cdot)$ is the interpolation function of a smoothed estimate of the distribution of observed exposure level around its mean from kernel density estimator, x_i represents the estimated GPS for the wildfire smoke PM_{2.5} exposure for county year i , and $\bar{x}_i$ represents the average estimated GPS for exposure. To avoid the effect of extreme IPW on

analysis results, the *stabilized IPW* value was trimmed to the range of first and 99th percentile (26). If the IPW model was correctly specified, we created a pseudopopulation through weighting in which the exposure was independent of all measured confounders, which eliminated the potential for confounding by those covariates. Last, we fitted a generalized additive linear regression model for annual county-level mortality rate against wildfire smoke PM_{2.5} exposure with adjustment of all measured confounders, weighted by the stabilized IPW. If the GPS model was misspecified, the adjustment of all confounders in the outcome regression would still provide valid health effect estimates. Our effect estimates were presented as absolute changes in mortality rate per 100,000 persons for each 0.1 µg/m³ increase in exposure level of wildfire smoke PM_{2.5}. This increment aligns more closely with typical exposure variations and is therefore more plausible and meaningful for interpretation. To account for multiple testing of nine mortality outcomes (one all-cause of death, six specific causes of death, and two negative outcome controls), we applied the Bonferroni correction to adjust 95% CIs. This is conservative because the tests are not independent (i.e., if wildfire smoke PM_{2.5} is associated with deaths from all-cause, it is likely associated with deaths from some specific causes). We further calculated the number of annual deaths attributable to a 0.1 µg/m³ increase in exposure using the formula: annual attributable deaths = rate difference × annual average population.

To better understand the relationships between wildfire smoke PM_{2.5} and mortality rate, the exposure-response curves were estimated based on our generalized additive model with thin plate regression splines. The basis dimension for the curve was set to 5 to allow sufficient nonlinearity while avoiding overfitting, and smoothing parameters that determine the effective degrees of freedom were automatically selected via generalized cross-validation. The results from model diagnostics indicated appropriate balances between bias and variance in the model (table S8). To obtain the predicted mortality rates, all other covariates were fixed at their mean values. The Bonferroni correction was also performed in the calculation of 95% CIs.

Age and seasonal temperature are well-known predictors of mortality (61, 62), and significant differences in wildfire smoke PM_{2.5} levels and mortality rates were observed between rural and urban areas (63). Therefore, we further performed stratified analyses to test whether the relationships differed across subgroups defined by upper or lower quartiles of percentage of population under 65 years old, average RUCA score, summer temperature, and winter temperature. A two-sample *t* test was used to compare the difference of coefficients across subgroups. To assess the robustness of our results, we trimmed the IPW_{st} at the 2.5th and 97.5th percentiles and repeated the analyses. In addition, we estimated the GPS using a parametric linear regression model. All data cleaning, merging, and statistical analyses were performed in R software (version 4.4.2) using packages such as “mgcv” and “dplyr.” Example code used for analysis was provided in the Supplementary Materials.

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Author contributions:

M.Z.: Writing—original draft, Conceptualization, Writing—review and editing, Methodology, Validation, Formal analysis, and Visualization. E.C.: Writing—review and editing, Resources, and Data curation. A.S.: Writing—review and editing, Methodology, and Formal analysis. A.A.P.: Investigation and Writing—review and editing. M.D.Y.: Writing—review and editing. X.W.: Writing—review and editing and Funding acquisition. J.D.S.: Conceptualization, Writing—review and editing, Methodology, Resources, Funding acquisition, and Formal analysis. R.O.W.: Writing—original draft, Writing—review and editing, and Supervision. Y.W.: Conceptualization, Writing—review and editing, Methodology, Resources, Funding acquisition, Data curation, Supervision, Software, and Project administration.

Competing interests:

X.W. participated in the US EPA Workshop to Inform Review of the Ozone National Ambient Air Quality Standards in 2024. X.W. has been employed at Meta since 2025. The work presented in this manuscript was conducted prior to joining the company. The other authors declare that they have no competing interests.

Data and materials availability:

Mortality data can be obtained from US Centers for Disease Control and Prevention WONDER (<https://wonder.cdc.gov/mcd-icd10.html>). The originally predicted, grid-level PM_{2.5} from all-sourced data are available at <https://zenodo.org/records/7884640>. Meteorological data are available at https://daac.ornl.gov/DAYMET/guides/Daymet_-_Daily_V4.html. All data and code needed to evaluate and reproduce the results in the paper are present in the paper and/or the Supplementary Materials.

Supplementary Materials

This PDF file includes:

Example R code
Figs. S1 to S12
Tables S1 to S8

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