



Long-term exposure to wildfire smoke and mortality: Heterogeneous effects by exposure metric and across subpopulations

Lara Schwarz^a , Timothy B. Frankland^b , Sara Y. Tartof^c , Gina S. Lee^c , Yuqian M. Gu^c, Elizabeth Rose Mayeda^d , David J. X. González^a , Tarik Benmarhnia^{e,f} , Joan A. Casey^{g,h,1} , and Chen Chen^{e,i}

Affiliations are included on p. 10.

Edited by Drew Shindell, Duke University, Durham, NC; received April 25, 2025; accepted November 17, 2025

Wildfire smoke, once rare, is a hazard that populations across the globe are increasingly exposed to repeatedly. Evidence of acute health effects of wildfire particulate matter (PM_{2.5}) is growing, but less is known about long-term effects related to repeated exposures. Using a cohort of 1,250,083 Kaiser Permanente Southern California members aged ≥60, we estimated the association between all-cause mortality and 3-y exposure to five different census tract-level wildfire smoke metrics (mean daily wildfire-specific PM_{2.5}, mean daily wildfire-specific PM_{2.5} during the peak wildfire week, number of days with daily wildfire-specific PM_{2.5} > 0 μg/m³, number of weeks with average wildfire-specific PM_{2.5} > 5 μg/m³, and number of smoke waves). We applied a discrete-time approach with pooled logistic regressions, adjusting for sex, age, race and ethnicity, marital status, smoking status, requiring an interpreter, calendar year, and census tract-level poverty and population density. When comparing those highly exposed (95th percentile) to those minimally exposed (5th percentile), we found an increased odds of mortality across all five wildfire smoke metrics. Mean daily wildfire PM_{2.5} was the metric most strongly associated with mortality (odds ratio: 1.07; 95% CI: 1.05, 1.09). We observed greater vulnerability to the long-term effects of smoke for individuals under age 75, or with Black or Other racial/ethnic identity, or living in a census tract with higher poverty. Identifying the most harmful long-term wildfire smoke metric and most-at-risk populations can help focus attention for developing effective adaptation strategies in a changing climate.

wildfire smoke | particulate matter | wildfires | mortality | vulnerability

Climate-driven environmental changes, such as droughts and rising temperatures, have led to longer fire seasons and increasing wildfire frequency, severity, and duration (1, 2). Wildfires are also becoming increasingly destructive (2) while exposing large populations to harmful air pollution (3), as observed with the destructive January 2025 wildfires in Los Angeles. Fine particulate matter (PM_{2.5}) is a major pollutant produced by wildfires and one of the criteria air pollutants recognized as particularly detrimental to human health (4). When inhaled, these particles' small size increases their ability to enter deeply into the lung alveoli and bloodstream and cause harm to biological systems (5). Air pollution from wildfire smoke has reversed decades of progress in air pollution reduction around the world (6, 7). In recent years, wildfires have accounted for up to 25% of PM_{2.5} across the United States and up to half in some Western states, including California (8).

Growing evidence demonstrates that short-term exposure to wildfire smoke has acute health effects (3, 9–11). Wildfire PM_{2.5} is associated with higher risk of acute cardiovascular and respiratory disease hospitalizations and deaths (3, 10, 12), and these associations may be stronger than those with PM_{2.5} from other sources, such as traffic or industry-related pollutants (13). However, while wildfires were previously exceptional and localized events with public health concern focused on acute impacts for relatively few, their increasing occurrence and geographic spread from smoke traveling long distances, has made wildfire smoke a repeated and long-term exposure for many. Although it is well known that long-term exposure to all-source PM_{2.5} increases morbidity and mortality (14), the long-term impacts of wildfire PM_{2.5} on chronic health outcomes is not well understood.

Prior studies that have looked at long-term effects of wildfire smoke considered the association between average smoke exposure over one or several years and mortality, with none considering census tract-level wildfire exposure using longitudinal data in the United States (15–17). Being able to characterize individual-level residential exposure and follow-up at-risk populations through multiple years is important to understand long-term

Significance

As wildfires become more frequent and severe, the harmful effects of wildfire smoke are reaching beyond immediate impacts. Effects on mortality risk are persisting years after the initial exposure, leading to long-term consequences on affected populations. We found long-term exposure to wildfire smoke to be associated with higher mortality risk across various metrics of smoke exposure, with 3-y mean wildfire-related PM_{2.5} concentration showing the strongest effect. Individuals under age 75 and who identified as Black or in the Other race and ethnic group were most at risk. Wildfire-smoke related deaths are preventable, making it crucial to focus on actions that protect the most vulnerable and limit public health harms.

Author contributions: T.B.F., S.Y.T., G.S.L., E.R.M., T.B., J.A.C., and C.C. designed research; L.S., T.B.F., S.Y.T., G.S.L., Y.M.G., E.R.M., D.J.X.G., T.B., J.A.C., and C.C. performed research; L.S., T.B.F., J.A.C., and C.C. analyzed data; and L.S., T.B.F., Y.M.G., D.J.X.G., T.B., J.A.C., and C.C. wrote the paper.

The authors declare no competing interest.

This article is a PNAS Direct Submission.

Copyright © 2025 the Author(s). Published by PNAS. This article is distributed under [Creative Commons Attribution-NonCommercial-NoDerivatives License 4.0 \(CC BY-NC-ND\)](#).

¹To whom correspondence may be addressed. Email: jacasey@uw.edu.

This article contains supporting information online at <https://www.pnas.org/lookup/suppl/doi:10.1073/pnas.2509173122/-/DCSupplemental>.

Published December 15, 2025.

effects (17). Additionally, while average exposure is a reasonable metric, it may not fully capture the specific aspects of prolonged smoke exposure that contribute most significantly to health impacts. Researchers have highlighted the importance of using multiple exposure metrics to evaluate differences in the duration, frequency, and severity of wildfire smoke and its associated health impacts (18, 19). Also, all-source $PM_{2.5}$ has a known nonlinear exposure–response relationship with mortality, which could also extend to wildfire $PM_{2.5}$. Previous work has found different associations between long-term exposure to 12-mo average county-level wildfire $PM_{2.5}$ and monthly mortality rates across a range of concentrations based on eight categories with the strongest associations observed above $5 \mu\text{g}/\text{m}^3$ (16), but the shape of this continuous curve has not been described.

Southern California is a unique context to study the health effects of wildfire smoke due to its high wildfire risk and sociodemographic diversity. California is highly affected by wildfires and this is only increasing; the total burned area from wildfires in the state doubled from 1984 to 2017 (20). Wildfire smoke is not equally harmful to all populations, with individuals in older age, minoritized, or disadvantaged groups potentially at elevated risk (19, 21, 22). This may be driven by differential access to the goods, services and opportunities of society, psychological stress, and subsequently differential internal dose and susceptibility toward wildfire smoke (23–26). Despite the growing evidence of the harmful impacts of long-term exposure to wildfire smoke, a lot is unknown about how smoke exposure over the long-term drives higher burden and what populations are most susceptible (12, 19, 20). Identifying which populations are most at-risk to the long-term effects of wildfire smoke is useful to inform targeted health-protective actions. Older adults are likely more vulnerable to the adverse health effects of wildfire smoke due to higher prevalence of preexisting health conditions, mobility limitations, decreased social support, and reduced access to information (27, 28). Prior wildfire and health studies have considered those above 60 y of age as older adults (29).

The objective of this study was to evaluate the long-term effects of wildfire smoke on all-cause mortality in a Southern California cohort of older persons followed from 2008 to 2019. By using a unique longitudinal dataset and census tract–level individual wildfire exposure, we expand on previous studies on the long-term effects of wildfire smoke on mortality by improving spatial resolution of exposure and allowing the study of different exposure profiles and vulnerability factors. Specifically, we explored differences in mortality effects across five different tract-level metrics of wildfire smoke meant to capture different exposure regimes (i.e., duration, frequency, and intensity) of wildfire $PM_{2.5}$ exposure to understand which wildfire smoke exposures best explain the long-term health burden. We were specifically interested in comparing concentration metrics, which use a continuous level of exposure, and exceedance metrics, which use counts of days or weeks over certain thresholds. Subsequently, we considered nonlinearity in these effects and identified particularly vulnerable subgroups.

Results

Of the 1,640,220 eligible Kaiser Permanente Southern California (KPSC) members aged ≥ 60 between January 1, 2008, and January 1, 2019, we included 1,250,083 members in our open cohort study population. The catchment area for our study population was Imperial, Kern, Los Angeles, Orange, Riverside, San Bernardino, San Diego, San Luis Obispo, Santa Barbara, and Ventura Counties. We excluded 245,389 members who did not meet continuous enrollment criteria, 134,111 who were aged < 60

during the baseline year (defined as the year 2008 or the first year of enrollment if the member enrolled after 2008), 10,271 who did not have residence in KPSC's catchment area during the follow-up period, and 366 who had missing rural-urban area code or sex data (*SI Appendix, Fig. S1*).

Approximately half of the study population were women (53.2%). The majority race and ethnicity group was non-Hispanic White (49.3%), followed by Hispanic (26.0%), and Asian (10.4%) (Table 1). The majority of individuals in the sample were married or partnered (53.9%), and most never smoked (61.5%). Approximately one-tenth of the sample (10.9%) required an interpreter when receiving medical care. Across an average 23 quarters (SD: 15 quarters) of follow-up, we observed 172,196 deaths (13.8%) in this cohort, with 162,504 individuals lost to follow-up (13%). The corresponding cumulative incidence rates for death and loss-to-follow-up are similar across sociodemographics, except for higher censoring in the Other race and ethnicity group and higher death for those above or equal to 75 y (*SI Appendix, Table S1*).

Capitalizing on an existing dataset of wildfire-specific $PM_{2.5}$, we utilized five metrics to quantify the long-term exposure to wildfire smoke based on members' residential census tracts: the mean 3-y rolling average of daily wildfire-specific $PM_{2.5}$ (subsequently referred to as mean wildfire $PM_{2.5}$), the mean 3-y rolling average of daily wildfire-specific $PM_{2.5}$ during the peak wildfire week (subsequently referred to as mean peak week), the 3-y rolling sum of days with daily wildfire-specific $PM_{2.5}$ above zero (subsequently referred to as nonzero days), the 3-y rolling sum of weeks with average wildfire-specific $PM_{2.5}$ above five $\mu\text{g}/\text{m}^3$ (subsequently referred to as weeks over five), and the 3-y rolling sum of two consecutive days with an average daily wildfire $PM_{2.5}$ concentration $> 15 \mu\text{g}/\text{m}^3$ (subsequently referred to as smoke waves). These metrics were moving averages and sums updated every quarter based on the residential census tract of KPSC members. Mean wildfire $PM_{2.5}$ and mean peak week are continuous metrics, while nonzero days, weeks over five and smoke waves are discrete metrics that capture the number of events that reached each threshold in the 3-y period. Areas with the highest average exposures for five wildfire metrics were near the Los Padres National Forest and Sequoia National Forest (Fig. 1). Several of the wildfire metrics were positively correlated with each other, ranging from 0.24 to 0.8 with mean wildfire $PM_{2.5}$ showing the highest correlation with weeks over five (Fig. 1).

To understand the direction of association between wildfire metrics and risk of mortality, a discrete-time approach with pooled logistic regression was applied to evaluate the association between each of the five metrics of long-term exposure to wildfire smoke and risk of all-cause mortality. When comparing those highly exposed (95th percentile) to those minimally exposed (5th percentile), we found increased odds of all-cause mortality for those highly exposed across all long-term wildfire smoke metrics (Fig. 2). The 95th and 5th percentiles of each wildfire smoke metrics are shown in *SI Appendix, Table S2*. The corresponding odds ratios (ORs) of mortality and wildfire metrics were 1.07 (95% CI: 1.05 to 1.09) for mean wildfire $PM_{2.5}$, 1.06 (95% CI: 1.04 to 1.09) for mean peak week, 1.05 (95% CI: 1.02 to 1.09) for nonzero days, 1.04 (95% CI: 1.02 to 1.05) for weeks above five, and 1.03 (95% CI: 1.01 to 1.04) for smoke waves (*SI Appendix, Table S3*).

We subsequently modeled the potentially nonlinear exposure–response relationship for the five wildfire smoke metrics with all-cause mortality using penalized splines with up to five degrees of freedom. We observed ORs above one comparing most exposure values of the five wildfire metrics to zero exposure (Fig. 3).

Table 1. Summary statistics of baseline characteristics of the study population and overall environmental exposure of Kaiser Permanente Southern California members, 2009 to 2019

Overall N = 1,250,083	
Individual-level characteristics	
Sex–N (%)	
Female	665,440 (53.2)
Male	584,643 (46.8)
Age at cohort entry–Years, median (IQR)	63 (60, 69)
Race and ethnicity–N (%)	
Non-Hispanic	
Asian	130,388 (10.4)
Black	118,027 (9.4)
White	616,660 (49.3)
Other*	59,629 (4.8)
Hispanic	325,379 (26.0)
Marital status–N (%)	
Married or Partnered	673,557 (53.9)
Divorced or Separated	121,075 (9.7)
Never married	149,429 (12.0)
Widowed	173,455 (13.9)
Other or Unknown	132,567 (10.6)
Smoking status–N (%)	
Never, Passive, or Unknown	768,853 (61.5)
Former Smoker	418,378 (33.5)
Current Smoker	62,852 (5.0)
Required interpreter–N (%)	135,641 (10.9)
Community-level characteristics	
Poverty (%)† – mean (SD)	12 (9)
Population density– mean (SD)	2,998 (2,599)
Environmental exposures during the study period – median (95th - 5th percentile)	
3-y mean wildfire PM _{2.5} (μg/m ³)	0.087 (0.23)
3-y mean wildfire PM _{2.5} during peak exposure week (μg/m ³)	2.3 (12.5)
3-y number of days with PM _{2.5} > 0 μg/m ³	37 (87)
3-y number of weeks with wildfire PM _{2.5} > 5 μg/m ³	0 (2)
3-y number of smoke waves	0 (1)
3-y mean nonwildfire PM _{2.5} (μg/m ³)	11.2 (7.4)

IQR, interquartile range; KPSC, Kaiser Permanente Southern California; SD, standard deviation.
 *Other includes individuals of multiple races, Native American and Alaskan Native, Pacific Islander, other, and unknown race and ethnicity.
 †Percentage of census tract living below the federal poverty threshold

We observed little deviation from linearity for mean daily wildfire PM_{2.5} and smoke waves. The three other metrics exhibited non-linearity, where ORs relative to zero exposure increased and then plateaued and decreased at the highest levels of exposure. For these three metrics, the highest OR relative to zero exposure occurred for the study population exposed to 20 μg/m³ at mean peak week, 160 d for nonzero days, and 7 wk for weeks over five. Numerical results are shown in *SI Appendix, Table S4*.

To understand differential vulnerability to wildfire exposure, we conducted stratified analysis by individual and community-level factors: sex, age group, race and ethnicity, and census tract–level poverty. No strong differences were observed by sex (Fig. 4). When stratified by age, persons under 75 y showed higher risk, and they had the highest risk for mean wildfire PM_{2.5} (OR = 1.18, 95% CI: 1.14, 1.22) contrasting 5th to 95th percentile of exposure in the total population while persons over age 75 did not show elevated risk (OR = 1.01, 95% CI: 0.99, 1.03) (Cochran Q: $P < 0.001$). This difference was consistent across wildfire metrics, with varying levels of precision. When comparing differences across wildfire metrics within each of the race and ethnicity groups, non-Hispanic Asian and non-Hispanic Black persons showed the strongest risk to the mean peak week exposure metrics with an OR of 1.16 [95% CI: 1.03, 1.30] and 1.18 [95% CI: 1.08, 1.28], respectively (Cochran Q: $P < 0.001$) (Fig. 4). In contrast, no risk was found for non-Hispanic White individuals for this exposure metric. Out of all five metrics, non-Hispanic White individuals showed the highest risk for mean wildfire PM_{2.5} exposure (OR = 1.04, 95% CI: 1.02, 1.07). Hispanic individuals had imprecise estimates across all wildfire metrics. When looking at differences by poverty level, those who lived in higher poverty census tracts had higher ORs of mortality from wildfire smoke than those that live in lower poverty census tracts, although the differences were not statistically significant for any exposure metric (Cochran Q P -values from 0.2 to 0.65) (*SI Appendix, Table S3*).

We conducted three secondary analyses, four sensitivity analyses, and one post hoc analysis to further explore our results. First, we conducted the same analyses using nonwildfire PM_{2.5} exposure as a secondary aim. The mean 3-y rolling average for mean nonwildfire PM_{2.5} concentration was 11.10 μg/m³ (IQR: 9.88 to 12.64). We observed an OR of 1.12 (95% CI: 1.10 to 1.14, comparing 5th to 95th percentile of exposure) in the total population for nonwildfire PM_{2.5} and mortality. We also allowed non-linearity and observed a U-shaped relationship between nonwildfire PM_{2.5} and mortality, with a small inverse association observed around 8.5 μg/m³, a stronger association at the lowest levels of exposure, and the strongest adverse association at the highest levels of exposure (*SI Appendix, Fig. S2*). We also observed similar effect modification results for age, sex, and poverty as for the wildfire metrics (*SI Appendix, Fig. S3*). Although the Other group had the highest OR among all race and ethnicity groups for nonwildfire PM_{2.5} and five wildfire metrics, those identifying as Black race experienced the lowest OR for nonwildfire PM_{2.5}, which differed from this group having the second highest ranking in wildfire metrics. For our sensitivity analysis, we added 3-y rolling average nonwildfire PM_{2.5} in our main model for each wildfire metrics to account for potential residual confounding and found minimal change in results (*SI Appendix, Fig. S4*). Similarly, we added 3-y rolling average wildfire PM_{2.5} in the nonlinear model for nonwildfire PM_{2.5} and found similar results (*SI Appendix, Fig. S5*). We also added a season indicator in the main model to account for potential temporal confounding and found similar results as the main model (*SI Appendix, Fig. S6*). In the final sensitivity analysis, we accounted for potential census tract clustering with generalized estimating equations models and found very similar results (*SI Appendix, Fig. S7*). Finally, in the post hoc analysis, we further stratified the Other group into two groups with known/unknown race and ethnicity. The Other-unknown group accounted for 62.8% of the Other race and ethnicity group and experienced earlier and higher loss to follow-up (mean follow-up time of 5.5 y and 47.3% of loss-to-follow-up individuals). The exceptionally high ORs among the Other race and ethnicity group were driven

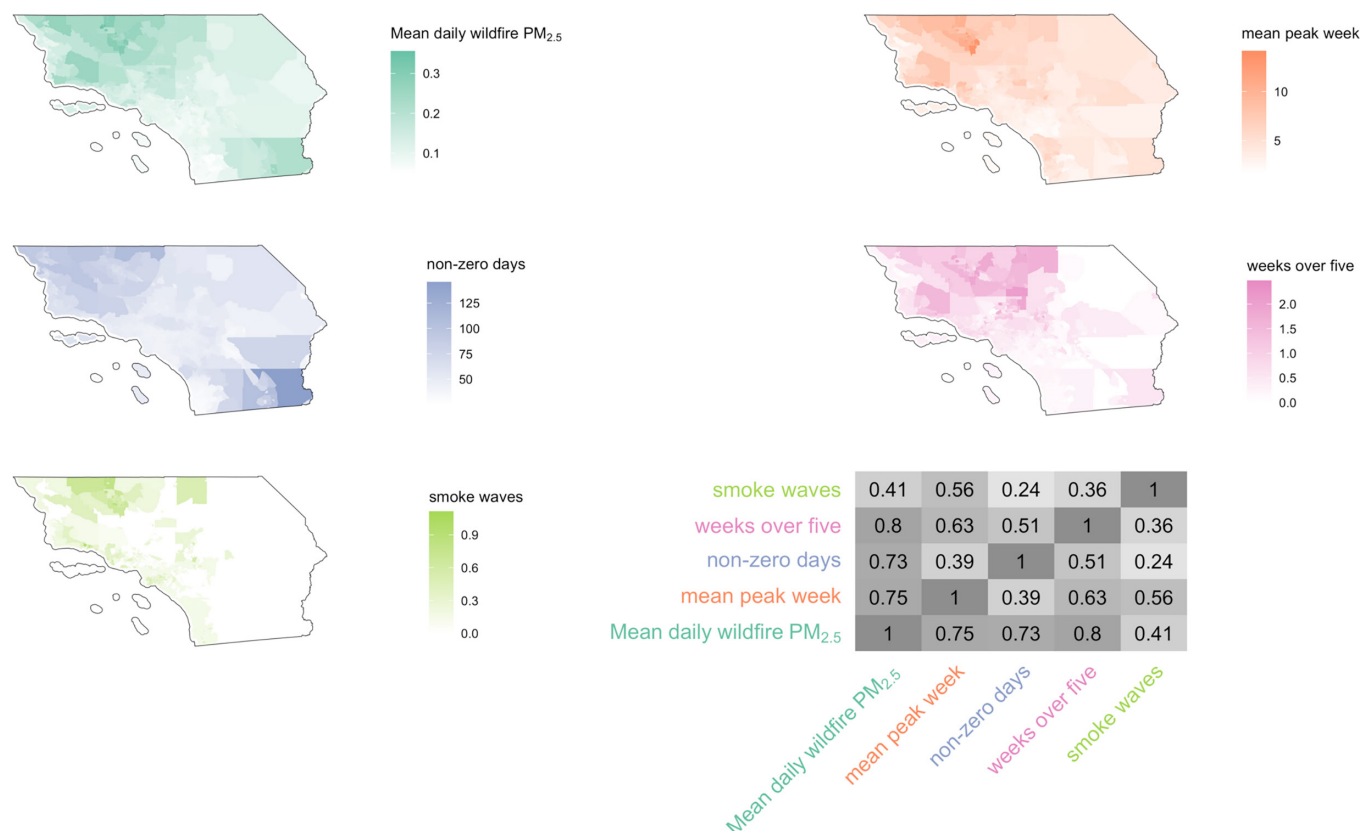


Fig. 1. Census tract-level exposures for five wildfire exposure metrics and their correlations, 2009 to 2019. The figure displays quarterly updated 3-y rolling averages or sums of wildfire exposure metrics for census tracts within Kaiser Permanente Southern California's catchment area. Metrics are mean 3-y rolling average of daily wildfire-specific PM_{2.5} (mean wildfire PM_{2.5}), mean 3-y rolling average of daily wildfire-specific PM_{2.5} during the peak wildfire week (mean peak week), 3-y rolling sum of days with daily wildfire-specific PM_{2.5} above zero (nonzero days), 3-y rolling sum of weeks with average wildfire-specific PM_{2.5} above five $\mu\text{g}/\text{m}^3$ (weeks over five), and 3-y rolling sum of two consecutive days with an average daily wildfire PM_{2.5} concentration $>15 \mu\text{g}/\text{m}^3$ (smoke waves).

by the subgroup of participants with unknown race and ethnicity (*SI Appendix, Fig. S8*).

Discussion

Using KPSC electronic health record (EHR) data including longitudinal census tract of residence information on members and state-of-the-art daily census tract-level wildfire-specific PM_{2.5} concentration, we observed positive associations between five long-term wildfire exposure metrics and risk of mortality, with the highest effect estimate observed for the mean wildfire PM_{2.5} metric. We observed a linear relationship for mean wildfire PM_{2.5} and smoke wave metrics, with some evidence suggesting a plateau in effect estimates at higher values for mean peak week, nonzero days, and weeks over five metrics. When stratified by individual and community characteristics, we observed stronger associations for persons below 75 y of age, Asian and Black individuals, and those living in higher poverty census tracts than their counterparts.

This study adds to a growing body of research showing the harmful effects and health burden of wildfire smoke on mortality. Our results on the effects of mean wildfire smoke on mortality risk are consistent with existing estimates from other contexts and regions. We found an OR of 1.07 (95% CI: 1.05 to 1.09), suggesting a 7% higher odds of mortality for those highly exposed as compared to those minimally exposed to 3-y mean wildfire PM_{2.5} (corresponding to an exposure change of $0.23 \mu\text{g}/\text{m}^3$). In comparison, existing estimates using longitudinal data from the United Kingdom looking at cumulative exposure translate to hazard ratio of 1.11 (95% CI: 1.03, 1.16) per $0.23 \mu\text{g}/\text{m}^3$ increase in 3-y mean

wildfire PM_{2.5} (17). A similar study in Brazil focused on cardiovascular mortality found that populations in the highest quartile of annual mean exposure to wildfire smoke (4.22 to $17.12 \mu\text{g}/\text{m}^3$) had a 2.2% higher mortality risk compared with those in the lowest quartile ($\leq 1.82 \mu\text{g}/\text{m}^3$) (15). Although these are not directly comparable to our estimates as they used different risk estimands and exposure metrics, the results suggest similar elevated risks in Southern California as what was observed in the United Kingdom and Brazil. In an ecological study in the United States, Ma et al. found that counties with 12-mo moving average smoke exposure ranging from 0.3 to $0.4 \mu\text{g}/\text{m}^3$ (most similar to our exposure change of $0.23 \mu\text{g}/\text{m}^3$) had 0.35 (95% CI: 0.21, 0.49) higher monthly nonaccidental mortality rate per 100,000 people compared to counties with exposure lower than $0.1 \mu\text{g}/\text{m}^3$ (16). Unfortunately, we cannot directly compare their absolute estimates to our estimates at relative scale.

Using five long-term wildfire exposure metrics allowed us to evaluate differential health impacts driven by different patterns of exposure to smoke in a region highly exposed to wildfires (18, 19). For example, considering two individuals with the same mean wildfire PM_{2.5} over 3-y but with different exposure profiles (for example, one was exposed to $100 \mu\text{g}/\text{m}^3$ 1 d during the period and another exposed to $1 \mu\text{g}/\text{m}^3$ for 100 d over the study period), the four other wildfire metrics will capture exposure variations not captured by mean wildfire PM_{2.5}. Our results suggested that metrics based on wildfire PM_{2.5} concentrations (mean wildfire PM_{2.5}, which essentially integrates all exposure but does not differentiate between intensity, frequency and duration of wildfire smoke, and mean peak week representing peak intensity of wildfire smoke)

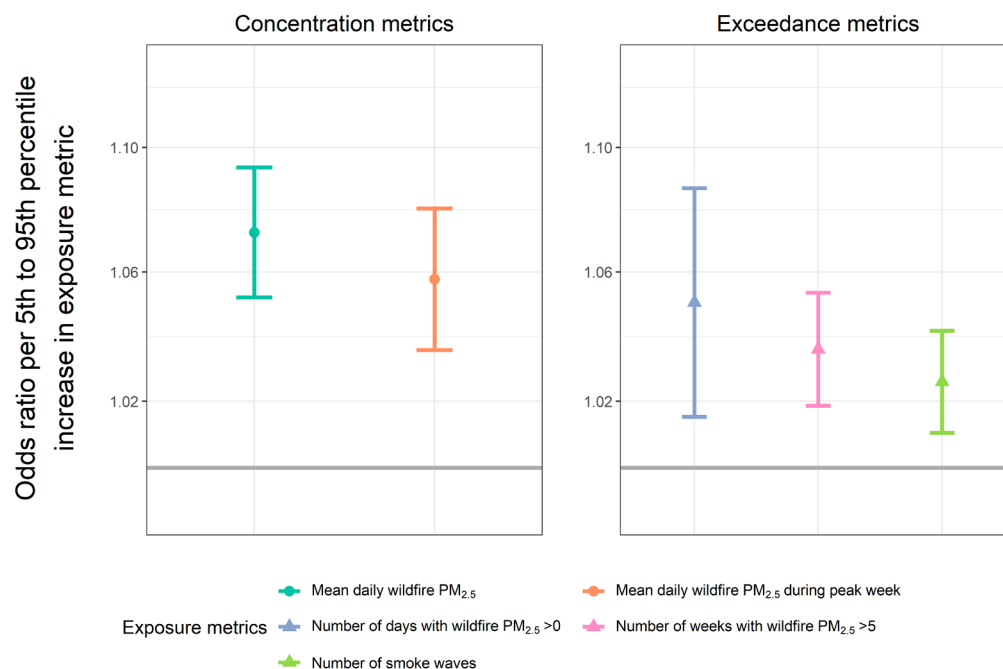


Fig. 2. Association of five wildfire PM_{2.5} 3-y exposure metrics and mortality among Kaiser Permanente Southern California members aged ≥ 60 , 2009 to 2019. Concentration metrics are mean wildfire PM_{2.5} and mean peak week and exceedance metrics are nonzero days, weeks over five and smoke waves. Odds ratios with 95% CI come from pooled logistic regression models and represent the contrast between the 5th and 95th percentile of exposure for each metric. Models were adjusted for sex, age, race and ethnicity, marital status, smoking status, and interpreter status, census tract-level poverty and population density, and indicator variables for each year, while including inverse probability censoring weights to account for differential loss to follow-up. The odds ratio displayed in the y-axis uses the natural logarithm scale, where equal distances represent equal multiplicative changes rather than additive ones. The gray line indicates null value for odds ratio at 1.

show higher risks than exceedance metrics based on the frequency of high wildfire smoke periods. This may indicate that duration and intensity of wildfire smoke, as compared to frequency, drove the greatest burden, suggesting that cumulative exposure to wildfire smoke has important health impacts.

Our results have implications for public health measures. For example, clean air centers are a public health measure to provide improved air quality to the public during a wildfire smoke event and limit the health burden of smoke. The majority of Clean Air Centers in California are activated only when the air quality exceeds a certain threshold, such as a daily air quality index of 100 to 150 (30). Our results suggest that activation of public health measures such as clean air centers should not only depend on intensity of smoke, as the long-term effects can be harmful even at low levels. Other public health measures such as Cal/OSHA's Wildfire Smoke Protection regulation, which addresses workers' health relating to harmful air pollution exposure and risk communication, are typically based on short-term health risks but public health measures should take into account that even low-level exposure can have long-term health impacts (31). Besides, low-cost filtration systems have been shown to improve indoor air quality during wildfire smoke and can be an effective strategy especially for individuals with limited material resources (32). Ensuring access to multilingual health communications and recommendations is important to ensure protection of those that are most vulnerable (33).

We evaluated whether the different metrics showed a linear exposure–response relationship with mortality in order to better understand which types of smoke exposure are worse for health, potentially due to behavior change at the highest levels of exposure. Previous evidence on linearity of the exposure–response relationship between wildfire smoke and mortality is inconclusive. In their ecological study, Ma et al. observed similar relationships

between 12-mo moving average smoke exposure and monthly nonaccidental mortality rate across exposure values below $5 \mu\text{g}/\text{m}^3$, with a substantially higher but less precise association observed above $5 \mu\text{g}/\text{m}^3$ (16). Previous studies exploring nonlinearity of the effect of long-term all-source PM_{2.5} on mortality also found inconsistent results, some suggesting a near-linear relationship across the exposure spectrum while others reporting associations only above $11 \mu\text{g}/\text{m}^3$ (14). We observed little deviation from linearity for mean daily wildfire PM_{2.5} and number of smoke waves, while the other metrics showed different effects across the values of metrics observed, generally increasing over the exposure values with a plateau or a decrease at the higher exposure values. This could potentially be due to behavioral changes such as better self-protection like closing windows or wearing masks to reduce internal dose of wildfire PM_{2.5} in the most exposed population. The deviation from linearity occurred when fewer observations existed and had wider CI. However, as wildfires worsen, these exposures will likely become more common, and it is important to do follow-up work to determine whether these nonlinearities persist.

Overall, mean wildfire PM_{2.5} drove the greatest effect across the entire study population, but we observed differences by subpopulation. We observed strong associations among relatively younger individuals (age <75) compared with older individuals (age ≥ 75) across all metrics, particularly for mean wildfire PM_{2.5}, where the younger population was highly impacted and there was no association for the older age group. This could be driven by differences between age groups, as the most susceptible persons 75 and over may have died sooner, therefore selecting a healthier subset within this age group. This disparity could also be driven by behavioral differences, as those 75 and over may stay indoors more often and, as a result, have lower wildfire PM_{2.5} exposure than those <75. Besides, the cumulative mortality rate among individuals with age

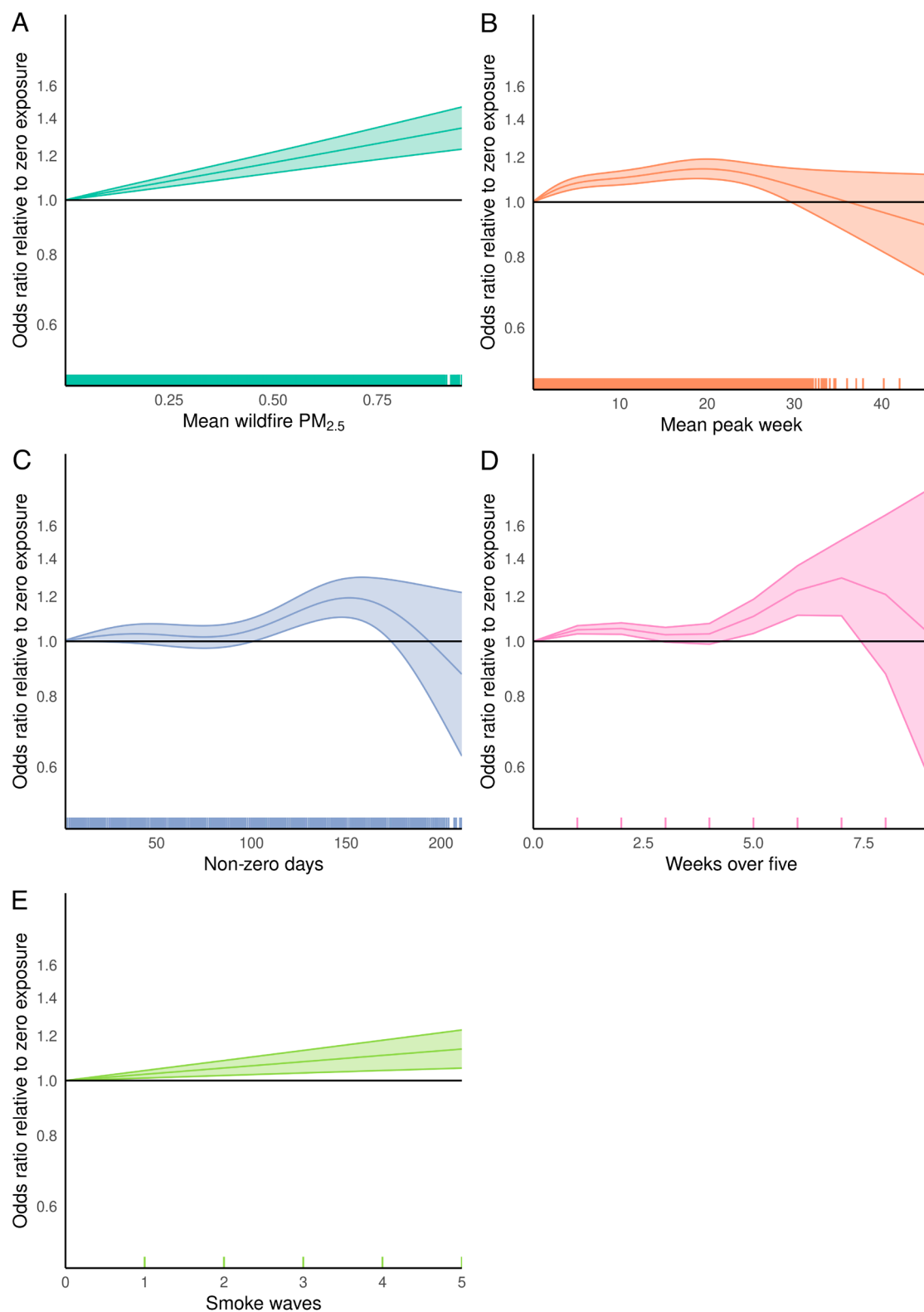


Fig. 3. Nonlinear exposure-response relationship between wildfire $PM_{2.5}$ 3-y exposure metrics, (A) mean wildfire $PM_{2.5}$, (B) mean peak week, (C) non-zero days (D) weeks over five and (E) smoke waves and mortality among Kaiser Permanente Southern California members aged ≥ 60 , 2009 to 2019. Odds ratios with 95% CI come from the pooled logistic model utilizing a penalized spline function of the exposure metric with thin plate regression splines and a maximum of five degrees of freedom. Models were adjusted for sex, age, race and ethnicity, marital status, smoking status, and interpreter status, census tract-level poverty and population density, and indicator variables for each year, while including inverse probability censoring weights to account for differential loss to follow-up. The odds ratio displayed in the y-axis is on the natural logarithm scale, where equal distances represent equal multiplicative changes rather than additive ones. The black line indicates null value for odds ratio at 1.

at entry above 75 was 6.6 times of the younger population (*SI Appendix, Table S1*), and a small effect on relative scale (OR) can still lead to a large increase in cumulative mortality rate and

a considerable public health burden. Gao et al. (2023) found no effect modification related to age comparing those below 60 and those 60 y and older, and Ma et al. (2024) found substantially

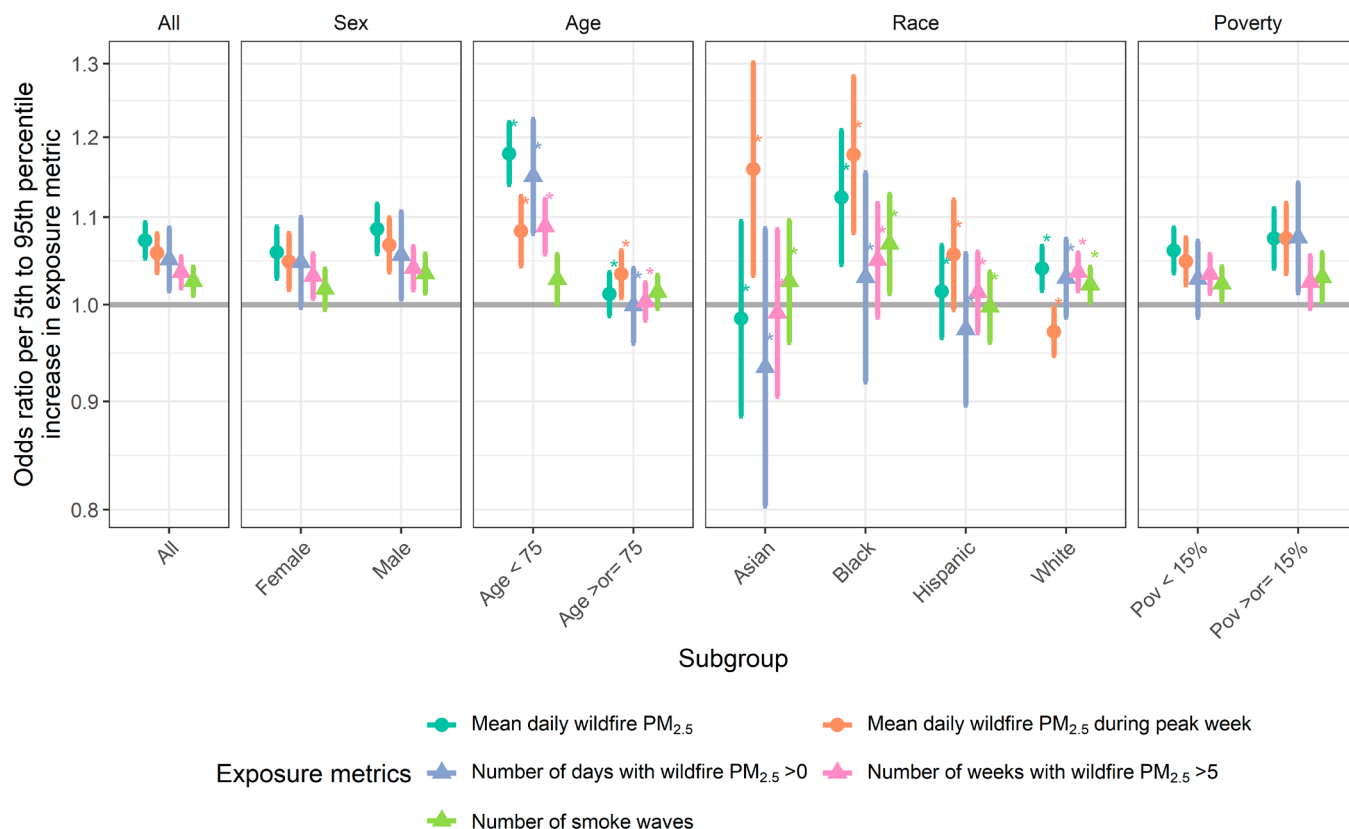


Fig. 4. Association between five wildfire $PM_{2.5}$ 3-y exposure metrics and mortality by subgroup among Kaiser Permanente Southern California members aged ≥ 60 , 2009 to 2019. The pooled logistic model adjusted for sex, age, race and ethnicity, marital status, smoking status, and interpreter status, census tract-level poverty (over 15% of population below federal poverty threshold) and population density, and indicator variables for each year (excluding the variable being stratified), while including inverse probability censoring weights to account for differential loss to follow-up. The 5th and 95th percentiles from the overall population were used to calculate the corresponding odds ratio. The odds ratio displayed in the y-axis uses the natural logarithm scale, where equal distances represent equal multiplicative changes rather than additive ones. Estimates with significant heterogeneity (Cochran's Q test) across subgroups within each effect modifier and wildfire metric are denoted with a star. The gray line indicates null value for odds ratio at 1. Note: We excluded the Other race and ethnicity group for easier visualization (wide CI and high effect estimates as shown in *SI Appendix, Fig. S4*).

higher risks among individuals ≥ 65 compared to those < 65 (16, 17). Notably, these earlier studies did not examine heterogeneity among older adults, as done in the present study. Additional research to further understand the modifiable risk factors in age-related vulnerability to wildfire smoke is needed to inform targeted warnings and communications.

Smoke exposure metrics driving the greatest health impact varied by race and ethnicity group. When comparing differences across exposure metrics within race and ethnicity subgroups, Asian and non-Hispanic Black individuals appeared to be most affected by mean peak week, while this metric was not associated with mortality for non-Hispanic White individuals in our study population. We observed the highest risk of mortality in the Other race and ethnicity group across all metrics, driven by those with unknown race or ethnicity. This is likely due to different health profiles of those with unknown race and ethnicity, who had an earlier loss to follow-up than the rest of our cohort, which can coincide with lower socioeconomic stability and render them particularly susceptible to wildfire smoke. Besides this group, the greatest impact of mean wildfire $PM_{2.5}$ was observed in non-Hispanic Black individuals, which is consistent with studies showing that Black populations are particularly at risk to the effect of wildfire smoke on incident dementia (34) and adverse respiratory health outcomes (22). Psychosocial stressors due to marginalization and discrimination may contribute to increased susceptibility toward long-term exposures (35, 36). Similarly, peak-week exposure showed a stronger association with

mortality for Black and Asian individuals than for other racial and ethnic groups. Mean wildfire $PM_{2.5}$ was also more strongly associated with mortality among Black individuals, which may reflect differences in resources and capacity to modify behavior during major smoke events. For example, Black and Hispanic individuals are more likely to live in poorer-quality housing than white individuals due to historical policies such as redlining (37), which could contribute to disproportionately high indoor wildfire $PM_{2.5}$ due to high permeability to outdoor air pollutants. There are many potential underlying drivers of wildfire smoke-related health disparities, and it is difficult to examine them given constraints in the way racial and ethnic identity are reported. Further work should examine social determinants related to structural racism, which could contribute to observed disparities.

Our results suggest that high-poverty census tracts may be more vulnerable to the effects of wildfire smoke, although these differences were not statistically significant. This is consistent with previous studies considering poverty as an effect modifier of long-term effects of wildfire smoke on mortality. In the United Kingdom, no differences appeared in the association between long-term wildfire-related $PM_{2.5}$ exposure and all-cause mortality by household income or socioeconomic status (17). Previous work in California also did not find community-level poverty to be an important predictor in the association between acute wildfire smoke and respiratory emergency department visits and hospitalizations (38). However, understanding community-level

vulnerability in addition to individual risk factors can be critical in effective prioritization of regions for public health actions and protections. Communities with higher poverty rates might also have limited resources to protect themselves from wildfire smoke, such as living in lower-quality housing with poor insulation, and are more likely to experience a high internal dose of smoke exposure (25). Providing risk communications that are accessible and adapted to most at-risk populations will be important to protect those that are most vulnerable (39).

Results of the secondary analysis evaluating the association between nonwildfire $PM_{2.5}$ and mortality suggested that the long-term effects of nonwildfire $PM_{2.5}$ show a U-shaped curve. The shape remained unchanged after further adjusting for long-term wildfire $PM_{2.5}$ (SI Appendix, Fig. S5). This exposure–response relationship differed from most previous research on the mortality effects of long-term all-source $PM_{2.5}$, and additional research is needed to understand the possible reason for this finding. When stratified by race and ethnicity, those in the Other race and ethnicity group show a higher risk of mortality, similar to our findings on wildfire $PM_{2.5}$. The odds of mortality for nonwildfire $PM_{2.5}$ is higher for Asian, non-Hispanic White, and Hispanic populations than the Black race and ethnicity group, with this group suggesting a protective association. Previous work considering long-term effects of all-source $PM_{2.5}$ on Black persons in the United States found the non-Hispanic Black group is at higher-risk than the non-Hispanic White population, even at low levels of exposure (40). However, race as an effect modifier on the association between all-source $PM_{2.5}$ exposure and mortality is inconsistent across studies, some showing higher risk for the Black population when compared to White populations and others showing a lower risk (41). Additionally, we are considering non-wildfire $PM_{2.5}$ which may show different relationships than all-source $PM_{2.5}$. More research is needed to further understand population vulnerabilities to all-source, wildfire-specific, and non-wildfire $PM_{2.5}$ in the Southern California region.

A strength of this study is its use of five wildfire smoke metrics. We utilized detailed EHR data from a region highly impacted by wildfire smoke, allowing for longitudinal analyses and exposure assessments, an advantage over prior studies. Limitations of the study include that wildfire $PM_{2.5}$ data underestimate smoke exposure due to the limitations of visible satellite imagery used for the estimations (42). We use 3-y estimates to characterize long-term exposure as it corresponds to the time frame used by the US Environmental Protection Agency for air pollution regulation compliance, but earlier wildfires may also increase mortality risk. An extension of this research could evaluate different averaging periods to more fully characterize long-term exposure. Second, we cannot rule out the possibility of residual biases. For example, the lack of information on individual-level socioeconomic status can cause residual confounding beyond our adjustment for census tract-level poverty, while behavior patterns and current or prior occupational exposures to wildfire $PM_{2.5}$ might produce differential exposure misclassification since personal exposure may differ from ambient wildfire $PM_{2.5}$. Although it is hard to gauge the direction of residual bias from such factors, evidence suggests that individuals with lower socioeconomic status tend to experience higher levels of indoor wildfire $PM_{2.5}$ compared to those in higher-cost housing, when the ambient levels are the same (25), suggesting a potential higher internal exposure dose among such subpopulations. Also, we observed about 13% loss to follow-up in our study population, but 31% for the most vulnerable Other-unknown race and ethnicity group. We used inverse probability censoring weight (IPCW) in our models to account for

differential censoring related to measured confounders, though it does not address unobserved potential confounders. Last, we only included KPSC members with more than 1 y of enrollment and probably missed individuals with extremely low income, who might be the most vulnerable.

Results of this study demonstrate the importance of going beyond one wildfire metric to understand the health effects of wildfire smoke. Future work can build on this by considering additional chronic health endpoints beyond all-cause mortality, such as lung cancer and brain tumors, which may be more strongly associated with wildfire smoke (43, 44). Also, wildfire smoke has been shown to be associated with increases in suicides in rural counties in the United States (45). Understanding what metrics of wildfire smoke are driving specific causes of death could provide insight into the mechanisms and inform potential interventions. A global analysis of landscape fires in low and middle-income countries found that each $1 \mu\text{g}/\text{m}^3$ increase in monthly $PM_{2.5}$ from landscape fires increased risk of child mortality by 2.31% (95% CI; 1.50, 3.13) (46). Expanding this work to a younger population could also be important, as they may have unique vulnerability factors. We hope this study will encourage more research to further understand the long-term effects of smoke, what metrics are driving these effects and vulnerability factors across different regions and sociodemographic groups.

Methods

Study Population. We used EHR data spanning 2008 to 2019 from KPSC, which serves 4.7 million people across 10 counties (47) (Fig. 1). KPSC members are similar to the demographics of Southern California residents, with minor underrepresentation of individuals with extremely low income and those with high education (48). The KPSC EHR contains longitudinal data on members' residential addresses and sociodemographic characteristics, updated at healthcare encounters or by the member. We created an open cohort of KPSC members who were enrolled continuously for at least 1 y (allowing 90-d enrollment gap) starting on January 1, 2008, or their corresponding baseline year, enrolled at least 1 d in the year following their baseline year (e.g., 1 d in 2009 if the baseline year was 2008), aged ≥ 60 in their baseline year, and living in a KPSC catchment census tract (SI Appendix, Fig. S1). The date of eligibility, cohort entry, and start of follow-up was the first day of the year after the baseline year for each cohort member. The earliest follow-up date was January 1, 2009, and the follow-up extended through the date of death, loss-to-follow-up, or administrative censoring on December 31, 2019.

Data used in this study were exempt from patient consent. The study protocol was approved by WCG Institutional Review Board (IRB), the IRB for KPSC, Columbia University, and the University of Washington.

Exposure Assessment. We utilized a previously published dataset of daily census-tract wildfire $PM_{2.5}$ concentrations in California from 2006 to 2019 (42). Briefly, Aguilera et al. estimated the daily census tract-specific total $PM_{2.5}$ with an ensemble machine learning approach incorporating outdoor $PM_{2.5}$ measurements from the US Environmental Protection Agency Air Quality System, aerosol optical depth, smoke plume and wildfire data, meteorological variables, and land use characteristics. The R^2 value is 0.78 for total $PM_{2.5}$ using holdout cross-validation test. They then estimated the daily nonwildfire $PM_{2.5}$ concentrations by removing census-tract days exposed to wildfire based on smoke plume data and imputing the missing values with multiple imputation via random forest. The corresponding daily census-tract wildfire $PM_{2.5}$ concentrations were the difference between the total and nonwildfire $PM_{2.5}$ concentrations. Spearman correlations were estimated between exposure metrics.

Following Casey et al. 2024, we quantified the long-term exposure to census tract-level wildfire $PM_{2.5}$ using 3-y moving averages, updated quarterly, of five metrics based on members' residential census tracts in the 3-y prior to the quarter of outcome ascertainment (19). Quarter is defined as January to March, April to June, July to September, and October to December. Out of the five metrics, two were measured based on concentration, or a continuous level of exposure, and

three were measured based on exceedance, or a count of days or weeks that had an exposure over a certain threshold. The two concentration metrics were the mean daily wildfire PM_{2.5} and mean daily wildfire PM_{2.5} during peak exposure week. The three exceedance metrics were the number of days with wildfire PM_{2.5} > 0 µg/m³, number of weeks with wildfire PM_{2.5} > 5 µg/m³, and number of smoke waves (defined as ≥2 consecutive days with >15 µg/m³ wildfire PM_{2.5}). We assigned exposure to each KPSC member with a 3-y rolling exposure for each metric, updating their residential census tract at the beginning of each quarter.

Outcome Ascertainment and Covariates. We obtained the date of death from any cause for cohort members through the KPSC Mortality Data Mart, which incorporated deaths sourced from the California state death files, National Death Index data, the Social Security Administration, KP internal systems, and non-KP claims. We aggregated deaths to the quarterly level to correspond with the temporal resolution of the wildfire smoke exposure variables.

We also derived individual-level characteristics of cohort members based on information from the EHR, including sex (male and female), age, self-reported race and ethnicity (non-Hispanic Asian or Pacific Islander, non-Hispanic Black, non-Hispanic White, Hispanic, and Other [other race and ethnicity included individuals identifying with multiple races, Native American and Alaskan Native, other races and ethnicity, or unknown races and ethnicity]), smoking status (current, former, and never smoker), relationship status (married, domestic partner, or common law; divorced or separated; widowed; single; and other or unknown), and whether an interpreter was required at any healthcare encounter. For analysis, we used baseline values of individual sociodemographics except for age, which was updated yearly along with community-level characteristics.

We also linked the Integrated Public Use Microdata Series census tract-level community characteristics (percent of households living below the federal poverty threshold and population density) summarized from the 2010 US Census to each cohort member via their census tract of residence at the beginning of each quarter (49). We categorized a census tract as high poverty if ≥15% of its population lived below the federal poverty threshold based on this census.

Individual-level and community-level characteristics were selected as confounders in our analysis based on prior knowledge of their relationship with wildfire smoke exposure and mortality. We considered individual race and ethnicity as indicators of exposure to stressors related to racism, which in this context could be interpersonal, institutional, and structural (23, 26, 50). Similarly, although the other individual-level characteristics might not directly affect the wildfire smoke hazard, they are known risk factors for death and can affect the actual exposure and internal dose of wildfire smoke via behavior patterns and quality of their living environment (25). We considered the census tract-level poverty as a proxy for individuals' socioeconomic position and their community-level living environment, which may affect their exposure and vulnerability to wildfire smoke (51). Population density was also used as a surrogate for the community-level living environment (38).

Statistical Analysis. We used a discrete-time approach (quarterly time interval) with pooled logistic regression to evaluate the association between each of five metrics of long-term exposure to wildfire smoke and risk of all-cause mortality. In the model for each wildfire metric, we controlled for individual sociodemographics, including sex, age at corresponding quarter (modeled with natural cubic spline function with one internal knot), race and ethnicity, marital status, smoking status, interpreter status, and community-level characteristics including population density and poverty, and indicator variables for each year. We also accounted for differential loss-to-follow-up by including a stabilized IPCW. Specifically, we modeled the probability of remaining in the cohort (i.e., not censored) accounting for individual sociodemographics, community-level characteristics, exposure, indicators for year since the start of follow-up, and interaction terms between exposure and indicators for year since the start of follow-up (full censoring model). To create the stabilized weight that reduces the influence of extreme weights, we also modeled the probability of remaining in the cohort (i.e., not censored) accounting for baseline confounders, exposure, indicators for year since the start of follow-up, and interaction terms between exposure and indicators for year since the start of follow-up (empty censoring model including exposure and baseline confounders). We then calculated the stabilized weight as the yearly predicted probability of not being censored for every individual and time point based on the empty censoring model over the same value based on

the full censoring model for every member. The final stabilized IPCW for each individual is the cumulative product of these stabilized weights up to the time point of interest after setting the value to 0 when the individual was censored. We reported the odds ratio of mortality comparing those highly exposed (95th percentile) to those minimally exposed (5th percentile).

We further explored possible nonlinearity in the exposure-response relationship between each wildfire metric and risk of all-cause mortality. We replaced the wildfire metric with a penalized spline function of the metric with thin plate regression splines and a maximum of five degrees of freedom. This allowed the algorithm to decide where and how flexible the spline should be for the nonlinear association explored.

Based on previous evidence of differential effect and potential mechanisms leading to differential vulnerability discussed above, we also explored effect modification by sex (female and male), age at corresponding quarter when outcome was ascertained (below 75 y of age and above or equal to 75 y of age), race and ethnicity (non-Hispanic Asian or Pacific Islander, non-Hispanic Black, non-Hispanic White, Hispanic, and other), and poverty level (poverty rate below 15% and poverty rate above or equal to 15%) through stratified analysis. To ensure that person-years from the same individual remain in the same poverty category, we used baseline poverty status of participants in the stratified analysis. We used the same model and confounders in the stratified analysis, while excluding the variable being stratified and using IPCW calculated for each stratum. We also used the 5th to 95th percentiles from the overall population to calculate the corresponding odds ratio. We conducted Cochran's Q test to evaluate the heterogeneity across subgroup-specific estimates, using *P*-value of 0.1 as the threshold to detect significant heterogeneity to account for low power of detecting heterogeneity in this test (52).

Secondary and Sensitivity Analyses. We conducted three secondary analyses and four sensitivity analyses to test robustness of our results. For our secondary analyses, we evaluated the association between 3-y rolling average nonwildfire PM_{2.5} and risk of all-cause mortality, with and without considering nonlinearity and subgroups. For our sensitivity analyses, we included 3-y rolling average nonwildfire PM_{2.5} for each quarter as a covariate in our main model for each wildfire metrics to account for potential residual confounding. We also add another sensitivity analysis to include quarterly 3-y rolling average of wildfire PM_{2.5} in the nonlinear model for nonwildfire PM_{2.5}, to test the robustness of our results toward residual confounding from wildfire PM_{2.5}. Third, although our exposure metrics aim to capture wildfire exposure in the previous 3 y, updating them quarterly might introduce temporal confounding by season as both wildfire activity and mortality have seasonal trends. To test the robustness of our results toward seasonal confounding, we add a season indicator in our main model (April to September vs. October to March). Finally, we conducted a sensitivity analysis using the generalized estimating equations model, including census tracts as clusters with an exchangeable covariance matrix to account for potential within-census tract clustering.

As a post hoc analysis to explore the high estimates among the Other race and ethnicity category, we further stratified the Other group into those with unknown race and ethnicity information and those with known race and ethnicity information (individuals self-reported as identifying with multiple races, Native American and Alaskan Native, other races and ethnicity).

Analyses were conducted using R Statistical Software version 4.3.0 (R Foundation for Statistical Computing, Vienna, Austria). The code is provided at the following link: https://github.com/benmarhnia-lab/long_term_wildfire_mortality.

Data, Materials, and Software Availability. Original, diagnosis-level data tied to individuals, locations, and time are considered personally identifiable health information. These data cannot be shared owing to risks of breaching patient confidentiality. Anonymized data that support the findings of this study may be made available from the investigative team upon agreement to abide by the terms outlined in data use agreement. The daily census-tract-level wildfire PM_{2.5} and nonwildfire PM_{2.5} data are available at: <https://doi.org/10.7910/DVN/CICODO> (53).

ACKNOWLEDGMENTS. Drs. S.Y.T., E.R.M., T.B., and J.A.C. and Mr. T.B.F. were supported by the US NIH National Institute on Aging grant R01-AG071024. Dr. J.A.C. was also supported by the NIH National Institute for Environmental Health Sciences grant P30-ES007033.

Author affiliations: ^aDivision of Environmental Health Sciences, School of Public Health, University of California Berkeley, Berkeley, CA 94704; ^bData Reporting and Analytics, Kaiser Permanente Hawaii Center for Integrated Health Care Research, Honolulu, HI 96819; ^cDepartment of Research and Evaluation, Kaiser Permanente Southern California, Pasadena, CA 91101; ^dDepartment of Epidemiology, University of California Los Angeles Fielding School of Public Health, University of California Los Angeles, Los Angeles, CA 90095; ^eClimate, Atmospheric Sciences, and Physical Oceanography,

Scripps Institution of Oceanography, University of California San Diego, La Jolla, CA 92037; ^fIrset Institut de Recherche en Santé, Environnement et Travail, UMR-S 1085, Inserm, University of Rennes, EHESP, Rennes 35000, France; ^gDepartment of Environmental and Occupational Health Sciences, University of Washington School of Public Health, Seattle, WA 98195; ^hDepartment of Epidemiology, University of Washington School of Public Health, Seattle, WA 98195; and ⁱDepartment of Population Health Sciences, University of Utah, Salt Lake City, UT 84108

1. J. G. Pausas, J. E. Keeley, Wildfires and global change. *Front. Ecol. Environ.* **19**, 387–395 (2021).
2. K. Shi, Y. Touge, Characterization of global wildfire burned area spatiotemporal patterns and underlying climatic causes. *Sci. Rep.* **12**, 644 (2022).
3. G. Chen *et al.*, Mortality risk attributable to wildfire-related PM2.5 pollution: A global time series study in 749 locations. *Lancet Planet. Health* **5**, e579–e587 (2021).
4. P. Orellano, J. Reynoso, N. Quaranta, A. Bardach, A. Ciapponi, Short-term exposure to particulate matter (PM10 and PM2.5), nitrogen dioxide (NO2), and ozone (O3) and all-cause and cause-specific mortality: Systematic review and meta-analysis. *Environ. Int.* **142**, 105876 (2020).
5. Q. Liu *et al.*, Effect of exposure to ambient PM2.5 pollution on the risk of respiratory tract diseases: A meta-analysis of cohort studies. *J. Biomed. Res.* **31**, 130–142 (2017).
6. W. Knorr *et al.*, Air quality impacts of European wildfire emissions in a changing climate. *Atmos. Chem. Phys.* **16**, 5685–5703 (2016).
7. R. R. Buchholz *et al.*, New seasonal pattern of pollution emerges from changing North American wildfires. *Nat. Commun.* **13**, 2043 (2022).
8. M. Burke *et al.*, The changing risk and burden of wildfire in the United States. *Proc. Natl. Acad. Sci. U.S.A.* **118**, e2011048118 (2021).
9. F. Hughes *et al.*, Impact of wildfire smoke on acute illness. *Anesthesiology* **141**, 779–789 (2024).
10. J. C. Liu, G. Pereira, S. A. Uhl, M. A. Bravo, M. L. Bell, A systematic review of the physical health impacts from non-occupational exposure to wildfire smoke. *Environ. Res.* **136**, 120–132 (2015).
11. C. F. Gould *et al.*, Health effects of wildfire smoke exposure. *Annu. Rev. Med.* **75**, 277–292 (2024).
12. A. I. Chen, K. Ebisu, T. Benmarhnia, R. Basu, Emergency department visits associated with wildfire smoke events in California, 2016–2019. *Environ. Res.* **238**, 117154 (2023).
13. R. Aguilera, T. Corringham, A. Gershunov, T. Benmarhnia, Wildfire smoke impacts respiratory health more than fine particles from other sources: Observational evidence from Southern California. *Nat. Commun.* **12**, 1493 (2021).
14. J. Chen, G. Hoek, Long-term exposure to PM and all-cause and cause-specific mortality: A systematic review and meta-analysis. *Environ. Int.* **143**, 105974 (2020).
15. Y. Gao *et al.*, Wildfire-related PM2.5 and cardiovascular mortality: A difference-in-differences analysis in Brazil. *Environ. Pollut.* **347**, 123810 (2024).
16. Y. Ma *et al.*, Long-term exposure to wildland fire smoke PM2.5 and mortality in the contiguous United States. *Proc. Natl. Acad. Sci. U.S.A.* **121**, e2403960121 (2024).
17. Y. Gao *et al.*, Association between long-term exposure to wildfire-related PM2.5 and mortality: A longitudinal analysis of the UK Biobank. *J. Hazard. Mater.* **457**, 131779 (2023).
18. J. D. Sacks, C. T. Migliaccio, C. E. Reid, L. Montrose, Shifting the conversation on wildland fire smoke exposures: More smoke within and across years requires a new approach to inform public health action. *ACS EST Air* **2**, 122–129 (2025), 10.1021/acestair.4c00236.
19. J. A. Casey *et al.*, Measuring long-term exposure to wildfire PM2.5 in California: Time-varying inequities in environmental burden. *Proc. Natl. Acad. Sci. U.S.A.* **121**, e2306729121 (2024).
20. L. Dong *et al.*, Meteorological environments associated with California wildfires and their potential roles in wildfire changes during 1984–2017. *J. Geophys. Res.* **126**, e2020JD033180 (2021).
21. J. Vargo, B. Lappe, M. C. Mirabelli, K. C. Conlon, Social vulnerability in US communities affected by wildfire smoke, 2011 to 2021. *Am. J. Public Health* **113**, 759–767 (2023).
22. J. C. Liu *et al.*, Who among the elderly is most vulnerable to exposure to and health risks of fine particulate matter from wildfire smoke? *Am. J. Epidemiol.* **186**, 730–735 (2017).
23. G. C. Gee, D. C. Payne-Sturges, Environmental health disparities: A framework integrating psychosocial and environmental concepts. *Environ. Health Perspect.* **112**, 1645–1653 (2004).
24. D. C. Payne-Sturges *et al.*, Defining and intervening on cumulative environmental neurodevelopmental risks: Introducing a complex systems approach. *Environ. Health Perspect.* **129**, 35001 (2021).
25. M. Burke *et al.*, Exposures and behavioural responses to wildfire smoke. *Nat. Hum. Behav.* **6**, 1351–1361 (2022).
26. C. P. Jones, Levels of racism: A theoretic framework and a gardener's tale. *Am. J. Public Health* **90**, 1212–1215 (2000).
27. S. L. Vega, M. L. Childs, S. Aggarwal, R. C. Nethery, Wildfire smoke exposure and cause-specific hospitalization in older adults. *JAMA Netw. Open* **8**, e257956 (2025).
28. W. E. Cascio, Wildland fire smoke and human health. *Sci. Total Environ.* **624**, 586–595 (2018).
29. C. C. Melton, C. M. De Fries, R. M. Smith, L. R. Mason, Wildfires and older adults: A scoping review of impacts, risks, and interventions. *Int. J. Environ. Res. Public Health* **20**, 6252 (2023).
30. R. J. Treves, E. Liu, S. L. Fischer, E. Rodriguez, G. Wong-Parodi, Wildfire smoke clean air centers: Identifying barriers and opportunities for improvement from California practitioner and community perspectives. *Soc. Nat. Resour.* **36**, 1078–1097 (2023).
31. K. Layton, Proposed federal OSHA standards for wildfire smoke. *Seattle J. Tech. Environ. Innovat. L.* **10**, 5 (2020).
32. N. W. May, C. Dixon, D. A. Jaffe, Impact of wildfire smoke events on indoor air quality and evaluation of a low-cost filtration method. *Aerosol Air Qual. Res.* **21**, 210046 (2021), 10.4209/aaqr.210046.
33. Y. Ornelas Van Horne, L. M. Diaz, J. Trowbridge, C. S. Alcala, D. J. X. González, Toward language justice in environmental health sciences in the United States: A case for Spanish as a language of science. *Environ. Health Perspect.* **131**, 85001 (2023).
34. H. Elser *et al.*, Wildfire smoke exposure and incident dementia. *JAMA Neurol.* **82**, 40–48 (2025).
35. G. S. Smith, E. Anjum, C. Francis, L. Deanes, C. Acey, Climate change, environmental disasters, and health inequities: The underlying role of structural inequalities. *Curr. Environ. Health Rep.* **9**, 80–89 (2022).
36. A. G. Berberian, D. J. X. Gonzalez, L. J. Cushing, Racial disparities in climate change-related health effects in the United States. *Curr. Environ. Health Rep.* **9**, 451–464 (2022).
37. S. Friedman, E. Rosenbaum, Nativity status and racial/ethnic differences in access to quality housing: Does homeownership bring greater parity? *Hous. Policy Debate* **15**, 865–901 (2004).
38. V. Do, C. Chen, T. Benmarhnia, J. A. Casey, S. Diego, Spatial heterogeneity of the respiratory health impacts of wildfire smoke PM2.5 in California. *GeoHealth* **8**, e2023GH000997 (2024).
39. M. H. Vien, S. L. Ivey, H. Boyden, S. Holm, L. Neuhauser, A scoping review of wildfire smoke risk communications: Issues, gaps, and recommendations. *BMC Public Health* **24**, 312 (2024).
40. K. P. Josey *et al.*, Air pollution and mortality at the intersection of race and social class. *N. Engl. J. Med.* **388**, 1396–1404 (2023).
41. M. T. Hicken, D. Payne-Sturges, E. McCoy, Evaluating race in air pollution and health research: Race, PM2.5 air pollution exposure, and mortality as a case study. *Curr. Environ. Health Rep.* **10**, 1–11 (2023).
42. R. Aguilera *et al.*, A novel ensemble-based statistical approach to estimate daily wildfire-specific PM2.5 in California (2006–2020). *Environ. Int.* **171**, 107719 (2023).
43. J. Korsiak *et al.*, Long-term exposure to wildfires and cancer incidence in Canada: A population-based observational cohort study. *Lancet Planet. Health* **6**, e400–e409 (2022).
44. P. Yu *et al.*, Exposure to wildfire-related PM2.5 and site-specific cancer mortality in Brazil from 2010 to 2016: A retrospective study. *PLoS Med.* **19**, e1004103 (2022).
45. D. Molitor, J. T. Mullins, C. White, Air pollution and suicide in rural and urban America: Evidence from wildfire smoke. *Proc. Natl. Acad. Sci. U.S.A.* **120**, e2221621120 (2023).
46. T. Xue *et al.*, Associations between exposure to landscape fire smoke and child mortality in low-income and middle-income countries: A matched case-control study. *Lancet Planet. Health* **5**, e588–e598 (2021).
47. Kaiser Permanente Southern California Research Program, *About Us: Fast Facts* (2023). <https://www.kp-scalresearch.org/aboutus/fast-facts/>. Accessed 14 January 2025.
48. C. Koebnick *et al.*, Sociodemographic characteristics of members of a large, integrated health care system: Comparison with US Census Bureau data. *Perm. J.* **16**, 37–41 (2012).
49. S. Manson, J. Schroeder, D. V. Riper, S. Ruggles, Data from "IPUMS National Historical Geographic Information System: Version 12.0". University of Minnesota. <https://doi.org/10.18128/D050.V12.0>. Accessed 1 February 2024.
50. Z. D. Bailey *et al.*, Structural racism and health inequities in the USA: Evidence and interventions. *Lancet* **389**, 1453–1463 (2017).
51. C. Reid *et al.*, Effect modification of the association between fine particulate air pollution during a wildfire event and respiratory health by area-level measures of socio-economic status, race/ethnicity, and smoking prevalence. *Environ. Res. Health* **1**, 025005 (2023).
52. J. S. Kaufman, R. F. MacLehose, Which of these things is not like the others?: Subgroup effects in cancer research. *Cancer* **119**, 4216–4222 (2013).
53. J. Casey, T. Benmarhnia, R. Aguilera, Data from "Daily census tract-level wildfire fine particulate matter concentrations for California, 2006–2020". Harvard Dataverse. <https://doi.org/10.7910/DVN/CICODO>. Deposited 9 January 2024.