Health Effects of Wildfire Smoke Exposure

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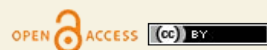
Keywords

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Abstract

We review current knowledge on the trends and drivers of global wildfire activity, advances in the measurement of wildfire smoke exposure, and evidence on the health effects of this exposure. We describe methodological issues in estimating the causal effects of wildfire smoke exposures on health and quantify their importance, emphasizing the role of nonlinear and lagged effects. We conduct a systematic review and meta-analysis of the health effects of wildfire smoke exposure, finding positive impacts on all-cause mortality and respiratory hospitalizations but less consistent evidence on cardiovascular morbidity. We conclude by highlighting priority areas for future research, including leveraging recently developed spatially and temporally resolved wildfire-specific ambient air pollution data to improve estimates of the health effects of wildfire smoke exposure.

INTRODUCTION

In recent years, headlines reading "The World Is on Fire" have been run in newspapers globally as high-profile wildfires burned in Australia, the Amazon rainforest, Chile, Russia, southern Europe, and western North America, accompanied by striking satellite imagery of wide swaths of the world blanketed in smoke. It seems clear that wildfires negatively impact health: The air is toxic and the blazes destroy property and traumatize communities. And yet, unlike typical ambient air pollution, the impacts of wildfire smoke exposures on health remain incompletely understood. Given that climate change is projected to increase the frequency and size of wildfires in many parts of the world in coming decades, an improved understanding of the health impacts of wildfires is an urgent public health priority.

The purpose of this review is to discuss current knowledge on the trends and drivers of global wildfire activity, techniques and recent advances in the measurement of wildfire smoke exposure, and available evidence on the myriad health effects of wildfire smoke exposure. We define wildfires as uncontrolled fires that occur in a natural environment, such as forests, grasslands, or prairies. These fires can have both proximate and distant direct and indirect health impacts, ranging from injury from fires, heat, and property damage to respiratory impacts from smoke inhalation to trauma-related mental health harm. We critically review empirical methods for assessing the health effects of wildfire smoke exposure, offering suggestions for future studies to improve methodological consistency and rigor. Then, in a meta-analysis, we quantitatively synthesize existing literature on the effects of ambient wildfire smoke on same-day all-cause mortality, respiratory-related emergency department (ED) visits and hospitalizations, and cardiovascular-related ED visits and hospitalizations. We conclude by commenting on gaps in our knowledge of the health effects of wildfires and potential solutions for addressing their impacts.

WILDFIRE ACTIVITY: TRENDS AND DRIVERS

Wildfire activity is driven by a complex combination of climate, ecological, and human factors. Available evidence suggests climate-related factors like temperature and precipitation are the most important drivers of large-scale patterns of wildfire activity (1). Climate-induced warming and drying have increased the frequency and severity of fire-conducive weather conditions in nearly all global regions over at least the last 50 years (1–3). A growing population, particularly in the wildland–urban interface, can also lead to more frequent human-caused ignitions. This combination of factors is likely to generate widespread risk of increasing wildfire activity in the coming decades (1, 2, 4, 5). Despite these patterns, observed trends in wildfire burned area have varied across regions, and global average burned area actually declined between 2000 and 2020 (3), in large part driven by human factors, such as land use transition, forest management, and other land management practices (3, 6). Irrespective of longer-term trends, fire activity is highly variable year-to-year, and extreme fire activity years are observed even in regions with decreasing trends.

WILDFIRE SMOKE

Composition

Wildfires emit a mixture of particles and gaseous pollutants that are known to negatively impact human health, including particulate matter (PM), carbon monoxide, nitrogen oxides, and volatile organic compounds (7–10). Depending on the materials burned, heavy metals like lead and mercury can also be emitted. Wildfire smoke has also been documented to contain toxic carcinogens—not unlike cigarette smoke—such as benzene, benzo[a]pyrene, and

dibenz[a,h]anthracene. Wildfire-specific PM likely has a different toxicological profile from PM originating from other sources (7, 10); however, the relative toxicity of wildfire-derived PM compared to PM from other sources remains uncertain. The amount and composition of pollution emitted from a specific fire vary depending on the fire's size, temperature of combustion, materials burned (e.g., grasses, tree species, buildings, vehicles), distance the smoke has traveled, and environmental conditions like wind speed, temperature, and humidity (9, 10).

Measurement

To enable studies focused on the health impacts of wildfires, approaches to estimating wildfire smoke exposures must be able to both separate wildfire smoke from other pollution sources and estimate exposures everywhere people live in a temporally and spatially disaggregated manner. Multiple approaches to estimating wildfire-specific ambient air pollution concentrations and exposures have been employed in the literature, each with strengths and limitations (11). (**Supplemental Table 1** summarizes available data products and approaches.)

Ground monitors can accurately estimate surface pollutant concentrations, but they do not distinguish between pollution from wildfires and that from other sources, and they are sparsely located in most regions (12, 13). Aside from ground monitors, studies have employed atmospheric chemical transport models (CTMs) (14, 15), dispersion models, and statistical models including machine learning approaches (16, 17) to estimate ambient wildfire smoke concentrations. CTMs are complex numerical models that simulate atmospheric chemistry dynamics and directly model the movement and evolution of wildfire emissions; frequently, CTMs are run with and without emissions from fires to estimate wildfire-specific pollution concentrations. CTMs are computationally intensive and thus hard to run for large spatial areas at high resolution over meaningful time scales; they are also sensitive to uncertain inputs (e.g., emissions from a given fire) (18). Dispersion models use meteorology and simplified physics to model the transport of pollution emissions; they are less computationally intensive than CTMs but may fail to capture certain complexities. In contrast to CTMs and dispersion models, statistical models do not attempt to model atmospheric chemistry and instead characterize the direct relationship between wildfires (i.e., the presence of smoke plumes) and surface pollution concentrations measured at ground monitors. They often incorporate remotely sensed measures of atmospheric aerosols, meteorology, and other factors that influence smoke concentrations. However, these models are typically trained on imperfect proxies for surface-level wildfire smoke pollution. Hybrid approaches incorporating outputs from multiple modeling frameworks (i.e., CTMs used as an input into statistical models) are also increasingly common (14, 19).

Recently, high-resolution wildfire-specific surface $PM_{2.5}$ concentration estimates have been developed for California (17) and the contiguous United States (14, 16). Daily estimates of total $PM_{2.5}$ that incorporate, but do not distinguish, wildfire-specific $PM_{2.5}$ are available at various spatial and temporal resolutions (see **Supplemental Table 1**). While the choice of wildfire smoke metric may be dictated by availability, existing evidence suggests that this choice affects estimated associations between wildfire smoke and health outcomes (20, 21).

Trends

Due to the methodological challenges discussed above, reliable global data on wildfire-specific pollution are not currently available. Furthermore, the distinction between wildfires and other types of landscape fire (e.g., crop burning) is unclear in many regions. Available estimates of global air quality impacts associated with all landscape fire suggest that fire is responsible for about 14% of ambient $PM_{2.5}$ in Africa, 9% in South America, 7% in North America, 4% in Asia, and 2% in

Europe in recent years (22). Collectively, 43 million people live in areas where the air quality is "unhealthy" ($PM_{2.5} > 55 \, \mu g \, m^{-3}$) because of wildfires at least once yearly (22).

In North America, where air quality monitoring is more comprehensive, evidence suggests that wildfires have played an increasingly important role in determining overall air quality levels over the last several decades (23). Smoke's impact on surface average and extreme $PM_{2.5}$ concentrations is now observed throughout much of the western United States (16, 24) and as far away as the East Coast (24, 25). In 2020 alone, an estimated >25 million people in the United States were exposed to at least 1 day with wildfire $PM_{2.5} > 100 \, \mu g \, m^{-3}$ (16).

SUMMARY OF HEALTH IMPACTS OF WILDFIRES AND SMOKE EXPOSURE

Similar to air pollution from other sources, observational evidence has linked exposure to wildfire smoke with a wide range of human health outcomes (**Supplemental Figure 1**) and has been the subject of previous reviews (8, 9, 26–31).

Substantial literature documents wildfire smoke's impacts on respiratory health, measured in increased respiratory-related mortality (32) and morbidity (33), declines in lung function, asthma exacerbations (34), respiratory medication dispensations (35), and respiratory infections including COVID-19 (36).

Despite established links between all-source PM and cardiovascular and cerebrovascular health, evidence related to wildfire smoke exposure has been mixed (37). Some studies have reported positive associations between wildfire pollution and cardiovascular mortality (32) and morbidity (38, 39), e.g., heart attacks (40). However, a number of other studies have reported non–statistically significant differences and even declines in cardiovascular outcomes (e.g., 41, 42).

Studies have also examined a range of other outcomes. A growing number of studies have identified an association between wildfire smoke and adverse pregnancy and birth outcomes, namely preterm birth and low birthweight (43–46), potentially through the effects of both exposure to wildfire smoke and maternal stress associated with wildfire occurrence. Recent literature has documented worsened cognitive outcomes (47, 48) and declines in mental health (49) associated with exposure to wildfire smoke. For example, Cleland et al. (47) assessed cognitive performance among adults and found that the presence of wildfire smoke plumes was negatively associated with the estimated attention score on the same day and one week later. Emerging literature also links wildfire smoke exposure with skin diseases (50), eye conditions (51), and cancer (52, 53).

Some studies have investigated the biological mechanisms through which wildfire smoke exposure negatively impacts health (e.g., 54, 55). It is likely that the mechanisms parallel those established in the broader air pollution literature, namely oxidative stress and inflammation, impaired nervous system function, vascular dysfunction, direct damage when particulates and chemicals enter the bloodstream, and epigenetic alterations, among others (10, 37, 56).

Firefighters

Studies have documented a range of occupation-related health impacts among front-line wildland firefighters (35, 57, 58). Wildland firefighters face multiple health hazards, including exposure to smoke, intense heat, low oxygen conditions, excess noise, physical hazards like falling trees, burning debris, and ash, as well as long working hours with minimal rest and protections (57, 58). These hazards can negatively impact respiratory health, cardiovascular health, and mental health, and lead to dehydration, malnutrition, and acute physical injuries (35, 57, 58). Typically, evidence for these impacts has come from case studies or assessments of within-subject changes before and after work shifts or across fire seasons, e.g., declined acute lung function after work shifts (59).

Long-term health impacts from occupational exposures among wildland firefighters are less well-documented, though emerging literature suggests elevated risks for cancer (60), cardiovascular disease (61), and biomarkers of aging (62).

Nearby Communities

Wildfires can affect local communities through multiple channels, including physical damage to infrastructure, air quality degradation, loss of livelihoods, and disruption of local ecosystems (63). A large body of evidence has documented negative mental health outcomes during and after wildfire events (64), including elevated rates of post-traumatic stress disorder (65), depression (66), anxiety (67), and substance use (65). Wildfires can impact mental health through several pathways, including reduced sleep duration and quality, reduced physical activity, increased perceptions of risk and anxiety, isolation from others, forced evacuations and/or relocations, reduced access to livelihoods, and loss of nature (49, 64, 68). Recent work emphasizes that measurement of this broad array of potential impacts—rather than counting buildings burned or resources spent on suppression efforts—is needed to quantify the impact of wildfires on communities (69).

Given the vulnerability of some communities to the impacts of environmental hazards (70), community resilience is a critical factor in mitigating the health effects of wildfires (71, 72). Recent work discusses adaptive and transformative social-ecological resilience, where communities and social systems adapt to new dynamics (e.g., increased wildfire activity) by changing aspects of the system (e.g., land use planning) and intentionally transform to acknowledge the role of fire in social-ecological systems (73). As more of us live in wildfire-prone regions, it is increasingly important to develop and implement policies that anticipate and plan for fires—as we do for other natural hazards like floods, earthquakes, and hurricanes (71).

META-ANALYSIS OF EFFECTS OF WILDFIRE SMOKE EXPOSURE ON MORTALITY AND ON RESPIRATORY AND CARDIOVASCULAR MORBIDITY

To quantitatively assess the impact of wildfire smoke on a range of health outcomes, we conducted a meta-analysis of the existing empirical literature. We focused our attention on studies with research designs and statistical methods that used variation in smoke exposure that is plausibly uncorrelated with other drivers of health risk; these tended to be studies that utilized within-location variation in smoke exposure and health outcomes over time to identify the impact of smoke on health.

Methods

We used a two-level strategy to search for studies evaluating the effect of ambient wildfire smoke (specifically, $PM_{2.5}$) on five health outcomes: all-cause mortality, respiratory-related ED visits and hospitalizations, and cardiovascular-related ED visits and hospitalizations. First, we conducted a literature search of the National Library of Medicine's PubMed database (see **Supplemental Section 2** for the search string). Second, we identified additional articles using search techniques such as backward and forward citation chasing, and included references cited in previous systematic and narrative reviews on wildfires and health (i.e., 8, 9, 26–32). All the studies we included were (*a*) published in a peer-reviewed journal, (*b*) human subject studies of the general population, and (*c*) studies of exposures to wildfire smoke. We excluded articles that did not generate original effect estimates, that evaluated the effects of smoke from other types of fires (e.g., mine fires), and that documented chronic health effects of wildfire smoke pollution. Titles and abstracts were screened by one author (C.F.G.) using the Covidence online platform so that duplicate papers

were automatically removed. C.F.G. reviewed the full texts of all potentially eligible studies and determined inclusion. Three authors (C.F.G., S.H.N., and M.B.) reviewed included studies. (See **Supplemental Section 2** and **Supplemental Table 2** for more details.)

We extracted risk estimates from all studies and their corresponding confidence intervals for the association between a measure of wildfire smoke exposure and the outcome of interest. When studies presented cumulative and contemporaneous effects of wildfire smoke exposure on health, the contemporaneous (i.e., same-day or lag 0) effect was selected. In the case that lagged effects were presented and/or modeled separately, we extracted the same-day effect estimate. Study-specific estimates were pooled using a random-effects maximum likelihood (REML) estimation approach implemented using the "metafor" package (version 2) (74) in R (version 4.2.2) (75). We generated study-specific pooled estimates using REML in the case that studies provided multiple effect estimates (e.g., one estimate per geographic unit).

Between-study heterogeneity was assessed using I^2, which is estimated as the fraction of total heterogeneity explained by between-study heterogeneity. Publication bias was assessed using the Egger's regression test for funnel plot asymmetry, which tests for the presence of a relationship between observed effect sizes and standard errors.

Results

The search yielded 1,283 articles (**Supplemental Figures 2–4**). After applying exclusion/inclusion criteria, 153 were eligible for full-text review. Of these, some studies were excluded because they did not evaluate the appropriate outcome, were not of the general population (e.g., included older adults or children only), did not generate original effect estimates, had an inadequate study design (e.g., inadequate control for confounders), did not plausibly estimate the effect of wildfire-specific pollution (e.g., studies that estimated the effect of wildfire smoke plumes, event studies), or did not focus on wildfires (e.g., studies of mine fires). We included 8 studies in our meta-analysis of all-cause mortality, 10 for respiratory hospitalizations, 9 for cardiovascular hospitalizations, 5 for respiratory ED visits, and 4 for cardiovascular ED visits. (These studies are summarized in **Supplemental Tables 3–7**; the excluded studies are summarized in **Supplemental Tables 8–10**.)

Meta-Analysis

Figure 1 summarizes included studies and pooled estimates. Same-day all-cause mortality increased by 0.15% [95% confidence interval (CI) 0.01–0.28%] per 1–μg m^{-3} increase in wildfire-specific PM$_{2.5}$. There were robust positive associations between wildfire PM$_{2.5}$ and same-day respiratory outcomes: Respiratory hospitalizations increased by 0.25% (95% CI 0.09–0.52%) and respiratory ED visits increased by 0.36% (95% CI 0.19–0.53%) per additional 1–μg m^{-3} increase in ambient wildfire smoke PM$_{2.5}$. We found a non–statistically significant 0.06% (95% CI 0.00–0.12%) increase in same-day cardiovascular hospitalizations and no meaningful change in same-day cardiovascular ED visits (-0.03%; 95% CI -0.18–0.12%) per additional 1–μg m^{-3} increase in ambient wildfire smoke PM$_{2.5}$. For all outcomes except respiratory hospitalizations and cardiovascular ED visits, there was evidence of heterogeneity in effects across studies (i.e., Q-statistic $p < 0.05$). Egger's tests did not indicate evidence of publication bias for any outcome ($p > 0.05$) (see **Supplemental Figure 5** for funnel plots).

Limitations

There are limitations of our analysis worth discussing. First, as additional studies are published that meet high empirical standards, this meta-analysis should be updated. Second, while we
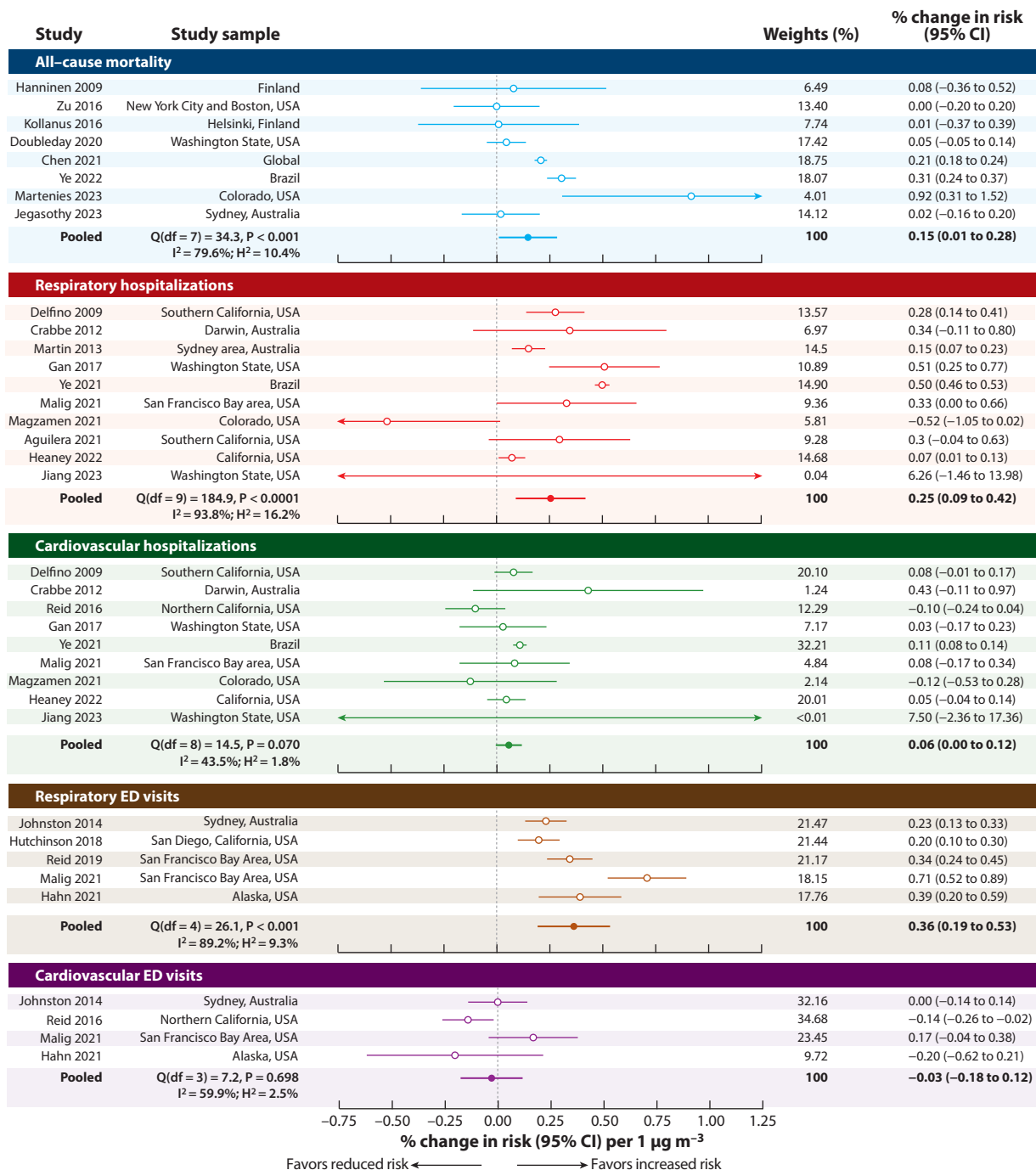
Figure 1

Study	Study sample	Weights (%)	% change in risk (95% CI)
All-cause mortality			
Hanninen 2009	Finland	6.49	0.08 (−0.36 to 0.52)
Zu 2016	New York City and Boston, USA	13.40	0.00 (−0.20 to 0.20)
Kollanus 2016	Helsinki, Finland	7.74	0.01 (−0.37 to 0.39)
Doubleday 2020	Washington State, USA	17.42	0.05 (−0.05 to 0.14)
Chen 2021	Global	18.75	0.21 (0.18 to 0.24)
Ye 2022	Brazil	18.07	0.31 (0.24 to 0.37)
Martenies 2023	Colorado, USA	4.01	0.92 (0.31 to 1.52)
Jegasothy 2023	Sydney, Australia	14.12	0.02 (−0.16 to 0.20)
Pooled	Q(df = 7) = 34.3, P < 0.001 I^2 = 79.6%; H^2 = 10.4%	100	**0.15 (0.01 to 0.28)**
Respiratory hospitalizations			
Delfino 2009	Southern California, USA	13.57	0.28 (0.14 to 0.41)
Crabbe 2012	Darwin, Australia	6.97	0.34 (−0.11 to 0.80)
Martin 2013	Sydney area, Australia	14.5	0.15 (0.07 to 0.23)
Gan 2017	Washington State, USA	10.89	0.51 (0.25 to 0.77)
Ye 2021	Brazil	14.90	0.50 (0.46 to 0.53)
Malig 2021	San Francisco Bay area, USA	9.36	0.33 (0.00 to 0.66)
Magzamen 2021	Colorado, USA	5.81	−0.52 (−1.05 to 0.02)
Aguilera 2021	Southern California, USA	9.28	0.3 (−0.04 to 0.63)
Heaney 2022	California, USA	14.68	0.07 (0.01 to 0.13)
Jiang 2023	Washington State, USA	0.04	6.26 (−1.46 to 13.98)
Pooled	Q(df = 9) = 184.9, P < 0.0001 I^2 = 93.8%; H^2 = 16.2%	100	**0.25 (0.09 to 0.42)**
Cardiovascular hospitalizations			
Delfino 2009	Southern California, USA	20.10	0.08 (−0.01 to 0.17)
Crabbe 2012	Darwin, Australia	1.24	0.43 (−0.11 to 0.97)
Reid 2016	Northern California, USA	12.29	−0.10 (−0.24 to 0.04)
Gan 2017	Washington State, USA	7.17	0.03 (−0.17 to 0.23)
Ye 2021	Brazil	32.21	0.11 (0.08 to 0.14)
Malig 2021	San Francisco Bay area, USA	4.84	0.08 (−0.17 to 0.34)
Magzamen 2021	Colorado, USA	2.14	−0.12 (−0.53 to 0.28)
Heaney 2022	California, USA	20.01	0.05 (−0.04 to 0.14)
Jiang 2023	Washington State, USA	<0.01	7.50 (−2.36 to 17.36)
Pooled	Q(df = 8) = 14.5, P = 0.070 I^2 = 43.5%; H^2 = 1.8%	100	**0.06 (0.00 to 0.12)**
Respiratory ED visits			
Johnston 2014	Sydney, Australia	21.47	0.23 (0.13 to 0.33)
Hutchinson 2018	San Diego, California, USA	21.44	0.20 (0.10 to 0.30)
Reid 2019	San Francisco Bay Area, USA	21.17	0.34 (0.24 to 0.45)
Malig 2021	San Francisco Bay Area, USA	18.15	0.71 (0.52 to 0.89)
Hahn 2021	Alaska, USA	17.76	0.39 (0.20 to 0.59)
Pooled	Q(df = 4) = 26.1, P < 0.001 I^2 = 89.2%; H^2 = 9.3%	100	**0.36 (0.19 to 0.53)**
Cardiovascular ED visits			
Johnston 2014	Sydney, Australia	32.16	0.00 (−0.14 to 0.14)
Reid 2016	Northern California, USA	34.68	−0.14 (−0.26 to −0.02)
Malig 2021	San Francisco Bay Area, USA	23.45	0.17 (−0.04 to 0.38)
Hahn 2021	Alaska, USA	9.72	−0.20 (−0.62 to 0.21)
Pooled	Q(df = 3) = 7.2, P = 0.698 I^2 = 59.9%; H^2 = 2.5%	100	**−0.03 (−0.18 to 0.12)**

% change in risk (95% CI) per 1 µg m^{-3}

Favors reduced risk ← → Favors increased risk

Figure 1

Meta-analysis of the associations between ambient wildfire-specific fine particulate matter and same-day health outcomes per 1 µg m^{-3}. Pooled responses are derived from random effects meta-analysis and estimated via restricted maximum likelihood.

analyze respiratory- and cardiovascular-specific outcomes, these categories are still broad, and there could be meaningful cause-specific heterogeneity in responses (e.g., respiratory tract infections versus chronic respiratory disorders). Third, we only extracted estimates from the general population; further investigation into heterogeneous effects across subpopulations (i.e., sex, age, or socioeconomic and demographic characteristics) is warranted. Fourth, we extracted same-day effects, which were the most common outcomes reported in the literature, and did not include lagged or cumulative effects as they were not sufficiently consistently reported. However, as more work emerges, including lagged and cumulative effects in meta-analyses will be critical in order to completely quantify the impacts of wildfire smoke on health.

CRITICAL REVIEW OF METHODS FOR ASSESSING HEALTH EFFECTS OF WILDFIRE SMOKE EXPOSURE

Here we describe important methodological issues in assessing the health effects of wildfire smoke and quantify their importance. Differences in methodological choices could in part explain the substantial heterogeneity in estimated effect sizes observed for some health outcomes (**Figure 1**), and we provide guidance on a set of methodological choices that we hope will guide future research in this area.

The central empirical challenge in estimating the effect of wildfire smoke on a chosen health outcome, as in other environmental health settings, is in isolating variation in wildfire smoke exposure from variation in other correlated factors that could also affect health outcomes. Absent this ability, measured smoke–health linkages are associational and cannot reliably inform quantitative estimates of the overall health burden of smoke exposure. However, compared to most other sources of variation in air pollution, the plausibly random temporal variation in wildfire smoke (i.e., variation unlikely to be correlated with confounders) offers unique opportunities to separate pollution exposure from other sources of correlated health risk and, thus, to establish plausibly causal concentration–response relationships. However, given difficulty in measuring smoke exposure at broad temporal and spatial scales, these opportunities have not always been exploited in the existing literature.

For instance, one common approach to quantifying smoke–health relationships has been to relate spatial or spatiotemporal variation in health outcomes to similar variation in wildfire smoke at a chosen spatial scale (e.g., zip code), and to adjust directly for variables (e.g., income) that could be correlated with both differences in average smoke exposure and in average health outcomes across locations. This approach is challenging because average smoke exposure is correlated in a statistically significant way with a very large set of measurable covariates (**Supplemental Figure 6**), and controlling completely for these measured covariates, and for the plausible set of additional unmeasured covariates also correlated with both smoke and health outcomes, becomes exceedingly challenging. As a result, these regression-adjustment approaches are unlikely to reliably isolate the causal effect of wildfire smoke on health. Rather than attempting regression adjustment, alternative approaches have instead utilized temporal variation in smoke exposure, comparing individuals to themselves (as in a case-crossover design) or locations to themselves (as in a time series or panel fixed-effects design) over time as smoke concentrations fluctuate. Conditional on seasonal controls and longer-term time trends, such temporal variation is plausibly random—an idiosyncratic function of where exactly a given fire starts and which way the smoke is blown. These designs can best measure the causal effect of short-term variation in smoke (subdaily to annual), periods over which temporal variation is plausibly random. As desired exposure windows get longer (multiple years or longer), temporal variation is reduced, and these designs become more challenging.

A second important issue is in accurately estimating the potential nonlinear shape of the smoke–health concentration–response curve. Recent work shows striking nonlinearities in the responsiveness of ED visits to daily wildfire smoke exposure (76), with increases in total visits at moderate exposures and substantial decreases at high exposures—the latter likely a result of behavioral changes during extreme exposures, such as reduced driving and traffic accidents, that reduce nonrespiratory morbidity. Failure to account for these potential nonlinearities could lead to inaccurate assessments of the overall contribution of smoke exposure to a given health outcome.

A final critical issue is in adequately accounting for the possibility of temporal lags between exposure and outcome. These lagged effects could amplify the total effect of a given smoke exposure, for instance if smoke exacerbates a respiratory infection that leads to an ED visit days after the exposure, or they could lead to offsetting effects, for instance by accelerating the speed with which a respiratory infection requires a hospital visit but not increasing the total number of hospital visits. This latter phenomenon, in which contemporaneous and lagged exposures have opposite signs, is often referred to as displacement or harvesting in the literature. Ex ante, it is unknowable whether either amplification or displacement (or some combination of the two) is occurring. The standard approach to calculating the cumulative (time-integrated) effect of a given exposure increase is to estimate distributed lag models, where the health outcome is modeled as a function of contemporaneous and temporally lagged values of wildfire smoke exposure, and then calculate the cumulative effect as the sum of effects across the contemporaneous and lagged variables. Estimating a distributed lag model but not summing the coefficients, as is sometimes done in the literature, does not yield consistent estimates of the total effect of an increase in smoke exposure.

To quantitatively illustrate the importance of these two concerns—nonlinear effects and lags—we revisit earlier work (76) and estimate the effect of daily wildfire smoke exposure on ED visits, using the universe of cause-coded ED visits aggregated to the zip code level in California during 2006–2017, gridded daily estimates of ambient wildfire $PM_{2.5}$, and panel fixed-effect regression models that flexibly account for location and time trending unobservables. In a linear model, the relationship between smoke exposure and ED visits is negative and would lead us to estimate that a total of 1,300 ED visits per year attributable to wildfire $PM_{2.5}$ were averted across California in 2006–2017 (**Figure 2a**). In contrast, a nonlinear model (a fourth-degree polynomial) indicates that ED visits increase at low to medium smoke $PM_{2.5}$ concentrations (<25 µg m^{-3}) but decline at higher concentrations. These declines in total ED visits at high daily smoke levels are likely driven by protective behavior that reduces, among other things, accidental injuries (76). Because most wildfire smoke days have low to medium $PM_{2.5}$ concentrations, using the quartic model we estimate an excess of 3,300 ED visits per year in California attributable to wildfire smoke. When examining respiratory diseases, we can see that the total respiratory ED visits model is a combination of asthma and respiratory tract infections, which respond differently to increasing wildfire $PM_{2.5}$ (**Figure 2b**). Failure to account for lags also alters inference of the total effect of wildfire $PM_{2.5}$ on changes to ED visit rates (**Figure 2c,d**). Accounting for lagged impacts increases the estimated effect of low to medium concentrations and also enhances the declines at higher concentrations, with the net effect of increasing excess ED visits by an order of magnitude (from 300 to 3,300 per year). Future studies on the health impacts of wildfire smoke should make sure to assess the importance of both delayed effects and potential nonlinearities.

KNOWLEDGE GAPS

A central unanswered question in our understanding of the health effects of wildfires is, "How different is wildfire pollution from pollution due to other sources in ways that matter for human
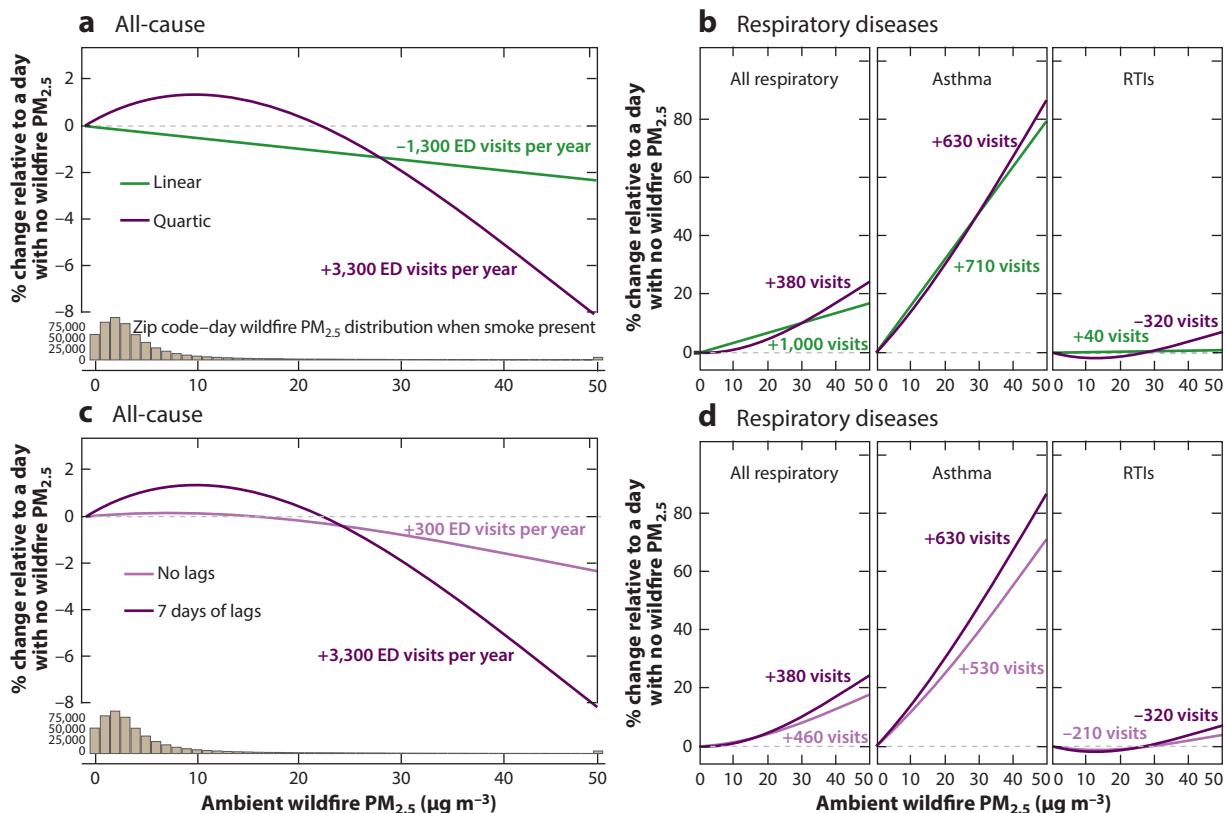
Figure 2

Differing response of daily zip code–level ED visit rates to wildfire $PM_{2.5}$ concentrations according to modeling linear and nonlinear effects and lagged effects in California, 2006–2017. Curves measure the percentage change in ED visits for a given daily smoke $PM_{2.5}$ concentration. Annotations indicate the total number of ED visits to public facilities attributable to wildfire smoke, with positives indicating excess ED visits and negatives indicating averted ED visits. Panels *a–b* show estimates under linear or nonlinear models; panels *c–d* show nonlinear estimates with and without lagged effects. Histograms in panels *a* and *c* indicate the distribution of zip code–smoke days on days with any wildfire smoke in that zip code. Abbreviations: ED, emergency department; PM, particulate matter; RTI, respiratory tract infection.

health?" One key element in this question is whether the composition and toxicity of wildfire smoke differ from other sources of air pollution. To investigate the toxicological profile of wildfire pollution compared to other common sources of pollution, future research could investigate whether a given unit of $PM_{2.5}$ from wildfire smoke is more toxic for human health than the average unit of $PM_{2.5}$ from other sources. The answer will likely vary across contexts (e.g., forest type, soil type, whether buildings burned, smoke distance traveled since emission) and exposure pathways. Ability to understand and predict the toxicity of smoke emitted from specific fires could shape long-term and immediate public health response and fire management.

A second key element is the episodic nature of wildfires. Fires do not steadily emit pollution at a fixed level, which distinguishes them from other sources of pollution like transportation and industry. Yet, we do not know whether varying patterns of exposure to wildfire smoke have differential impacts on human health. For example, wildfires may lead to ground-level $PM_{2.5}$ upwards of 100 $\mu g\, m^{-3}$ for only a day. Other times, ambient wildfire $PM_{2.5}$ might remain at 10 $\mu g\, m^{-3}$ but

persist for ten days. The cumulative dose in both scenarios would be $100\,\mu\mathrm{g\,m^{-3}}$, but nonlinearities described above for ED visits suggest that the health impacts could be quite different.

Another theme for future investigation is the extent to which human behavioral responses to ambient wildfire smoke shape health outcomes and healthcare utilization. Given the salience of wildfire smoke and that existing public health strategies rely on individuals undertaking self-protective behaviors when thick smoke is present, understanding the extent to which these behaviors actually protect health and alter public and individual health responses is critical for informing future resource allocation and policy. As the above nonlinear ED results suggest, the mixed evidence on the health impacts of wildfires may be partially explained by the combination of limited changes in behavior on moderate smoke days and protective behavior on high smoke days. Studies of smoke's cumulative effects on healthcare utilization could be averaging the positive and negative effects of wildfire smoke at different exposure levels, leading to null results.

The long-term health impacts of wildfire smoke exposures also remain poorly understood (77). These analyses are empirically challenging, as discussed above (see the section titled Critical Review of Methods for Assessing Health Effects of Wildfire Smoke Exposure), because disentangling variations in wildfire smoke exposure from factors correlated with health outcomes is increasingly difficult over longer time periods. However, as wildfires are a seasonal phenomenon that can contribute up to half of all ambient air pollution in some regions (16), they are worth quantifying to understand optimal investments in control measures.

Finally, there are important additional uncertainties in our understanding of the health impacts of wildfire smoke, including (but certainly not limited to) the imperfect measurement of wildfire smoke pollution discussed in previous sections, the extent to which personal exposures (including indoor exposures) deviate from the ambient concentrations used in most health studies, the extent to which prescribed burns are harmful for health and how these negative health impacts are offset by the reduced risk of more harmful fires in the future, the extent to which variations in chemical species present in wildfire smoke are captured in existing environmental epidemiological studies of ambient wildfire-specific pollution by being correlated with total $PM_{2.5}$, the potentially synergistic negative health impacts of hot and smoky days, the extent to which wildfire smoke waves are worse than periodic single-day episodes (e.g., 4 consecutive days versus 4 nonconsecutive days in the same month) due to biological or behavioral change, and how the salience of wildfires varies across contexts and the extent to which these differences affect health outcomes.

STRATEGIES FOR ADDRESSING HEALTH EFFECTS OF WILDFIRE SMOKE

We highlight three broad solution areas for addressing the human health effects of wildfires and smoke: those that aim to limit (*a*) the ignition of health-harming wildfires, (*b*) the damage from already ignited wildfires, and (*c*) the health harm from wildfire smoke.

Addressing the health impacts of wildfires begins with the upstream determinants of wildfire activity. Broadly speaking, the recent increase in wildfire activity in North America has been driven by the combined effect of a century of fire suppression that left an accumulation of fuels, a warming climate that has made these fuels drier and more flammable, and increased human activity in the wildland–urban interface that has made ignitions more likely. Reducing the likelihood of future extreme wildfires and the smoke they cause will thus require addressing these interacting factors. Such efforts are critical but may not be easy: Global climate change must be slowed or reversed, incentives to build houses in the wildland–urban interface reduced, and a century of accumulated fuels cleared from fire-prone areas using a variety of fuel management techniques. At scale, such efforts will likely require decades or longer to fully take effect.

When fires are already ignited, difficult decisions must be made as to how to manage them. Historically, efforts have focused on quickly suppressing fires, with suppression activity and costs focused on preventing incursion of fires into human inhabited areas. Wildfires near urban areas undoubtedly threaten lives, but fires distant from inhabited areas can generate large downwind smoke exposures that also threaten lives; these more distant fires may receive less suppression effort, even if they are potentially more costly from a public health perspective (78). There is also growing recognition that low-intensity fire, when left to burn or—in the case of prescribed fire—purposefully ignited, plays a critical ecological function and can reduce the likelihood of future extreme wildfire. Formally quantifying these trade-offs is a critical area for future work.

Given that increasingly extreme wildfire activity and smoke generation is, unfortunately, likely in the near term, efforts to protect public health in the face of growing exposures will be critical. One such effort will be ensuring that the public is informed of when wildfire smoke exposures are expected and how people can protect themselves from smoke. Systems can be developed that forecast wildfire activity (e.g., 15, 79) and that can be used to warn the public of imminent smoke exposures to encourage health-protective behaviors that limit exposure (e.g., running air purifiers, wearing respiratory protection, leaving the area) (30, 35, 80, 81). However, observational evidence on the effectiveness of these risk mitigation strategies remains limited. Small-sample and modeling evidence does suggest that running air purifiers and correctly wearing high-quality respiratory protection could reduce exposures and health-related risks (80, 82–84), but further randomized evaluation is warranted to guide policy and personal investments. Still, information alone is unlikely to be sufficient for self-protection (85), and thus communities will likely need to take direct preventive actions (e.g., subsidy or short-term rentals of air purifiers, access to clean air spaces, shelters in cleaner-air regions) targeted at those most vulnerable, including pregnant individuals, young children with asthma, older adults with chronic lung disease, and outdoor laborers. Physicians can play a role in facilitating preventive action, especially by (*a*) encouraging at-risk patients to stay at home, run air filters, and take other actions to protect themselves from wildfire smoke and (*b*) prophylactic prefilling of relevant prescriptions and increasing telemedicine opportunities. Conditional on a given level of smoke exposure, increased healthcare access can also help mitigate the severity of health impacts.

CONCLUSIONS

Wildfires are projected to increase in frequency and size in many regions globally because of a combination of climatic and human behavioral factors; the impacts of these fires on air quality and human health are also likely to grow. While accumulating evidence makes it clear that inhaled wildfire smoke negatively impacts human health, it is also increasingly clear that wildfire smoke is different from pollution from other sources in ways that likely matter for human health. For example, wildfire smoke may have a different toxicological profile from pollution from other sources; wildfires produce different patterns of exposure; and the salience of wildfire smoke can induce behavioral changes that alter health impacts, at least in some contexts. Analyses of the health effects of wildfire smoke exposures, including when distant from fires, should take advantage of recently developed smoke-specific ambient $PM_{2.5}$ data sets and the replicable approaches employed in the production of these data. Future work should aim to leverage and understand the unique dynamics of wildfire smoke: (*a*) Temporal variation in ambient wildfire smoke concentrations is frequently idiosyncratic, enabling causal interpretations when appropriately controlling for area characteristics and time trends; (*b*) healthcare utilization may respond nonlinearly to increasing pollution levels due to a combination of individual-level pathophysiological impacts and changing behaviors at the individual and population level; and (*c*) healthcare utilization and health impacts

can have varied lagged effects according to the outcome and location of interest. Better understanding these dynamics will be critical for understanding and mitigating the health impacts of wildfires in a changing climate.

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