Mortality risk attributable to wildfire-related $PM_{2·5}$ pollution: a global time series study in 749 locations

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Summary

Background Many regions of the world are now facing more frequent and unprecedentedly large wildfires. However, the association between wildfire-related $PM_{2·5}$ and mortality has not been well characterised. We aimed to comprehensively assess the association between short-term exposure to wildfire-related $PM_{2·5}$ and mortality across various regions of the world.

Methods For this time series study, data on daily counts of deaths for all causes, cardiovascular causes, and respiratory causes were collected from 749 cities in 43 countries and regions during 2000–16. Daily concentrations of wildfire-related $PM_{2·5}$ were estimated using the three-dimensional chemical transport model GEOS-Chem at a $0·25° \times 0·25°$ resolution. The association between wildfire-related $PM_{2·5}$ exposure and mortality was examined using a quasi-Poisson time series model in each city considering both the current-day and lag effects, and the effect estimates were then pooled using a random-effects meta-analysis. Based on these pooled effect estimates, the population attributable fraction and relative risk (RR) of annual mortality due to acute wildfire-related $PM_{2·5}$ exposure was calculated.

Findings 65·6 million all-cause deaths, 15·1 million cardiovascular deaths, and 6·8 million respiratory deaths were included in our analyses. The pooled RRs of mortality associated with each 10 μg/m³ increase in the 3-day moving average (lag 0–2 days) of wildfire-related $PM_{2·5}$ exposure were 1·019 (95% CI 1·016–1·022) for all-cause mortality, 1·017 (1·012–1·021) for cardiovascular mortality, and 1·019 (1·013–1·025) for respiratory mortality. Overall, 0·62% (95% CI 0·48–0·75) of all-cause deaths, 0·55% (0·43–0·67) of cardiovascular deaths, and 0·64% (0·50–0·78) of respiratory deaths were annually attributable to the acute impacts of wildfire-related $PM_{2·5}$ exposure during the study period.

Interpretation Short-term exposure to wildfire-related $PM_{2·5}$ was associated with increased risk of mortality. Urgent action is needed to reduce health risks from the increasing wildfires.

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Introduction

Recently, large and unprecedented wildfires have been occurring frequently across the world. During the past 3 years, wildfires have been observed in many locations of the world, including Australia, British Columbia in Canada, the western USA, and the Amazon rainforest.[1] For example, since the start of 2019, wildfires in California have burned more than 3 million acres, resulting in thousands of destroyed homes and businesses.[2] The wildfires in Australia have affected every state and destroyed more than 2000 homes and burned millions of acres.[3] Wildfires have both direct and indirect effects on health with potentially lasting consequences. Beyond direct injury, mental health can be harmed by the risks fires pose and loss of possessions and housing. The pollution from wildfire smoke can spread as far as 1000 km away and risk of wildfires is projected to keep increasing as climate change worsens.[1]

Wildfire smoke is a complex mixture of particulate matter (PM) and gaseous pollutants.[4] Among the various air pollutants emitted by wildfires, fine particulate matter ($PM_{2·5}$) is of great concern, as particles in this size range

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Research in context

Evidence before this study

Many regions of the world are now facing more frequent and unprecedentedly large wildfires. Wildfire-related air pollution has become a major public health concern, as it can travel widely and cause various adverse health effects. Previous studies have found wildfire-related air pollution to be significantly associated with increased mortality risk. We searched PubMed, Web of Science, Google Scholar, and China National Knowledge Infrastructure using the terms "wildfire", "bushfire", "fine particulate matter", "fine particles", "$PM_{2·5}$", "death", and "mortality" in English and Chinese for studies published up to Dec 25, 2020. We identified several studies exploring the impact of wildfire-related $PM_{2·5}$ on mortality. These studies showed that wildfire-related $PM_{2·5}$ had adverse effects on all-cause, cardiovascular, and respiratory mortality. However, the existing evidence comes from single-city or single-region studies, and not from studies with global reach.

Added value of this study

To the best of our knowledge, this is the largest study evaluating associations between acute wildfire-related $PM_{2·5}$ and mortality, and the first to do so comprehensively across various regions of the world, using daily death count data between 2000 and 2016 from 749 cities in 43 countries and regions. We found that the pooled relative risks of mortality associated with a 10 μg/m³ increase in the 3-day moving average of wildfire-related $PM_{2·5}$ concentrations were 1·019 (95% CI 1·016–1·022) for all-cause mortality, 1·017 (1·012–1·021) for cardiovascular mortality, and 1·019 (1·013–1·025) for respiratory mortality. Overall, 0·62% (95% CI 0·48–0·75) of all-cause deaths, 0·55% (0·43–0·67) of cardiovascular deaths, and 0·64% (0·50–0·78) of respiratory deaths were attributable to the acute impacts of wildfire-related $PM_{2·5}$ exposure during the study period.

Implications of all the available evidence

This study provides robust epidemiological evidence of the acute effects from wildfire-related $PM_{2·5}$ exposure on mortality, based on a large multicountry dataset and standard statistical method. Policy makers and public health professionals should raise awareness of wildfire pollution to prompt public responses and take actions to avoid exposure.

enter into the lungs and reach the alveoli where the small particles can translocate through the alveolar epithelium and enter the circulation.[5,6] Compared with $PM_{2·5}$ from urban sources, wildfire-related $PM_{2·5}$ tends to be more toxic due to its chemical composition and smaller particle size, and is often accompanied by co-exposure to other harmful environmental factors, particularly high temperatures.[1]

By contrast with numerous studies on total or urban background $PM_{2·5}$, far fewer studies have focused on health effects of wildfire-related $PM_{2·5}$ specifically, although some previous studies do suggest harm to public health.[7,8] Wildfire-related $PM_{2·5}$ exposure has been found to be associated with adverse health outcomes, such as premature mortality, asthma, and reduced lung function.[9–12] Studies examining the health effects of wildfire smoke in the USA, Canada, Australia, and Europe have found adverse health effects.[9,13–15] However, existing evidence mainly comes from single-city or single-region studies, and not from global studies. One study estimated that 339 000 deaths could have been attributable to global landscape fire smoke annually during 1997–2006,[16] but updated evidence from global-scale studies has not been subsequently reported.

In this study, associations between daily exposure to wildfire-related $PM_{2·5}$ and mortality were evaluated using the Multi-City Multi-Country (MCC) Collaborative dataset for 749 cities from 43 countries and regions.

Methods

Mortality and socioenvironmental data

Mortality data in this study were obtained from the MCC Collaborative Research Network, an international collaboration of research teams established to perform epidemiological studies on associations between environmental stressors and health.[17,18] The current MCC Network covers 750 cities from 43 countries and regions (appendix pp 6–7). Daily counts of all-cause deaths were collected from relevant authorities of each country or region. Mortality data for non-external causes (International Classification of Diseases [ICD] 9th Revision codes 0–799 or 10th Revision [ICD-10] codes A0–R99) were alternatively collected if all-cause mortality data were unavailable. In addition, mortality counts were collected specifically for cardiovascular (ICD-10 codes I00–I99) and respiratory (ICD-10 codes J00–J99) causes. Mortality data for all causes or non-external causes were available for 749 cities during the study period, while cardiovascular mortality data were available for 629 cities in 28 countries and respiratory mortality data for 647 cities in 29 countries. Other location-specific information was also collected: meteorological parameters (daily mean temperature and relative humidity) and gross domestic product (GDP) per capita.

Estimation of wildfire-related $PM_{2·5}$

From our previous work on global fire air pollution,[19] daily concentrations of wildfire-related $PM_{2·5}$ from Jan 1, 2000, to Dec 31, 2016, were estimated at a $0·25° \times 0·25°$ resolution. Briefly, the three-dimensional chemical transport model GEOS-Chem (version 12.0.0) was used to estimate global fire-induced perturbations in $PM_{2·5}$. A biomass burning inventory was adopted from the Global Fire Emissions Database (GFED; version 4.1), which estimated emissions based on satellite retrieval of burn area and active fire information. The GFED detected fires from five sources, including agricultural waste burning; boreal forest fires;

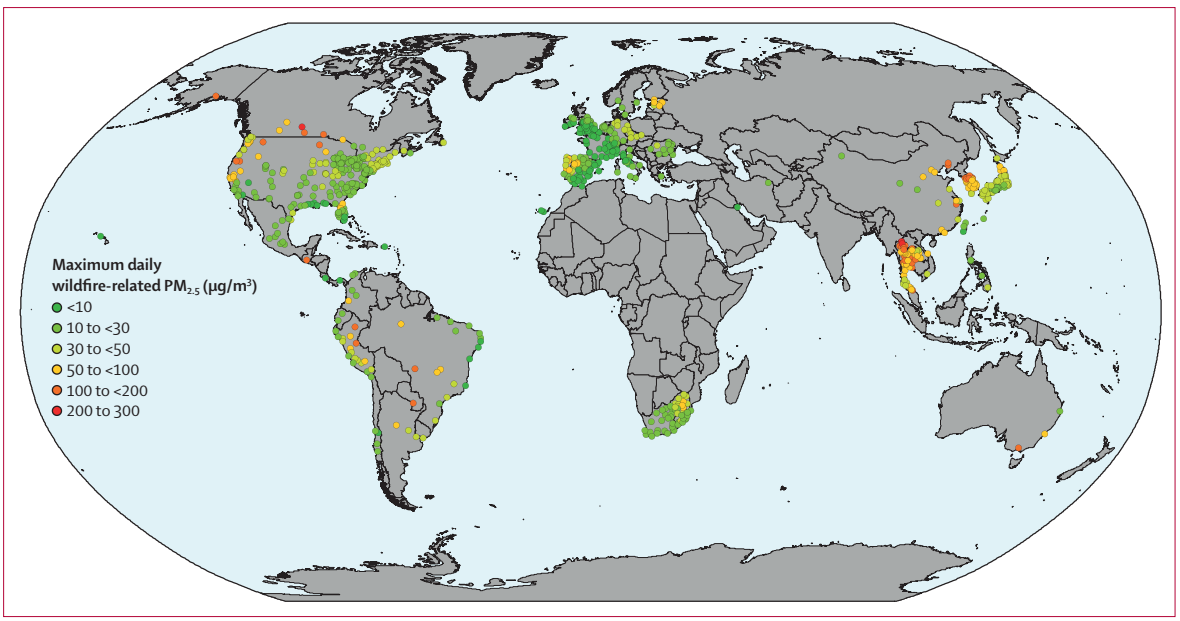

Figure 1: **Maximum levels of estimated daily wildfire-related $PM_{2·5}$ in study locations during 2000–16**

tropical forest fires; savanna, grassland, and shrubland fires; and temperate forest fires.[20]

Daily enhancements of $PM_{2·5}$ concentrations by fires during the study period were estimated as the differences between simulations with and without fire emissions. Daily concentration of wildfire-related $PM_{2·5}$ was first estimated globally using GEOS-Chem at a spatial resolution of 2·0°×2·5°, and then was adjusted and downscaled at a spatial resolution of 0·25°×0·25° using ground-level measurements of $PM_{2·5}$ and other predictors (eg, temperature, precipitation, wind speed, and day of the week). As wildfire-related $PM_{2·5}$ was not routinely monitored, the GEOS-Chem-derived estimates of all-source $PM_{2·5}$ were compared with ground-level measurements and their differences were further used to adjust the GEOS-Chem-derived wildfire-related $PM_{2·5}$. Results of a ten-fold cross-validation method showed that the adjusted all-source daily $PM_{2·5}$ concentrations derived from GEOS-Chem explained 86·5% of the variability of ground-level measurements. Details of model validation, adjustment, and downscaling are shown in the appendix (pp 3–5). Based on the raster data on estimation of global wildfire-related $PM_{2·5}$ at a spatial resolution of 0·25°×0·25° (roughly 28 km² at the equator), the concentration of the pollutant in each city on each day was assigned as the average of all the cell values that fell at least partly in each city.

Statistical analysis

To examine the association between exposure to daily wildfire-related $PM_{2·5}$ and mortality, a two-stage analytical approach was adopted.[21,22] In the first stage, a quasi-Poisson regression was employed to examine the city-specific association between daily concentration of wildfire-related $PM_{2·5}$ and death counts. Based on our previous work,[23,24] the single-day effect of wildfire-related $PM_{2·5}$ exposure on mortality on the current day and its lagged effects up to 7 days (from lag 0 to lag 7 days) were considered in city-specific models. Moving average lag models (eg, lag 0–1 and lag 0–2) were also implemented to examine cumulative effects of wildfire-related $PM_{2·5}$ exposure.[25] The seasonality and long-term trends were controlled using a natural cubic spline of time with 7 degrees of freedom per year.[21] The moving averages of temperature (for all cities) and relative humidity (applied to 556 out of 749 cities with available humidity data) during lag 0–7 days were controlled using natural cubic splines with 4 degrees of freedom.[26] Additionally, categorical variables for day of the week were included in the model.

In the second stage, the effect estimates from the city-specific models were pooled to derive overall effect estimates at the global and national levels using a random-effects meta-analysis.[27] The pooled $PM_{2·5}$–mortality association was shown as relative risk (RR) of death associated with a 10 μg/m³ increase in wildfire-related $PM_{2·5}$. The heterogeneity of effect estimated across cities was tested using the Cochran Q test and I^2 statistic.[28] To check for non-linear associations, the moving average of wildfire-related $PM_{2·5}$ was fitted using a B-spline function and two knots placed at the 25th and 75th percentiles of mean $PM_{2·5}$ concentration across all cities.[18] Then concentration–response relationships between wildfire-related $PM_{2·5}$ exposure and mortality were pooled at the global level.

Our initial analyses showed moderate heterogeneity in effect estimates across cities for all-cause mortality

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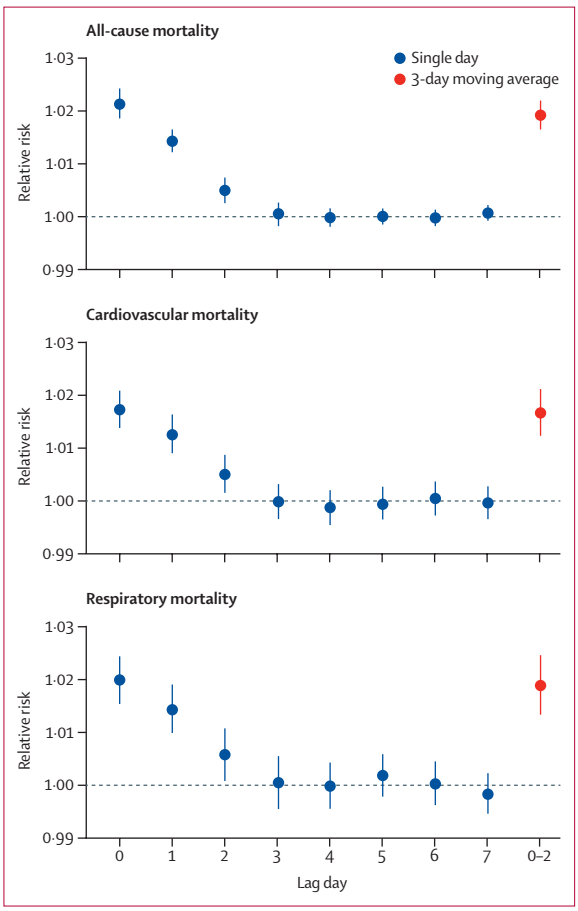

Figure 2: **Pooled relative risks of mortality associated with a 10 μg/m³ increase in wildfire-related $PM_{2·5}$ during lag 0–7 days**
Estimates show the single-day effects or 3-day moving average effect of wildfire-related $PM_{2·5}$ on mortality, with bars representing 95% CIs.

(I^2 13–50%), and almost no heterogeneity for cardiovascular (I^2: 0–10%) and respiratory (I^2: 0–18%) mortality (appendix p 8). Therefore, based on the pooled global-level risk estimates and assuming that the observed relationship was causal, the population attributable fraction (PAF) of annual deaths due to short-term exposure to wildfire-related $PM_{2·5}$ was calculated.[24] First, the number of annual deaths attributable to wildfire-related $PM_{2·5}$ was calculated for each city using pooled global-level effect estimates. Then, the total number of attributable deaths was divided by the total number of deaths across all cities to derive the pooled PAF at a global level. Additional analyses were done by pooling city-specific results at country, WHO region, and GDP levels. If only one city of a country was included in this study, the results for that city were used to represent its country. These analyses were done separately for all-cause, cardiovascular, and respiratory mortality. The specific formulas used are shown in the appendix (pp 5–6).

To examine the potential confounding effects of $PM_{2·5}$ from other sources, the results controlling for other-source $PM_{2·5}$ were compared with those that did not, using data from cities with available ground-measured $PM_{2·5}$. To test whether 7 days were sufficient to capture the lag effects of $PM_{2·5}$, sensitivity analyses were done by extending the maximum lag time from 7 to 10 days. To test the robustness of the results, the degrees of freedom for meteorological variables were changed to 3, 5, and 6, and lag times up to 10 days were considered for these variables. The city-specific models were also checked by only controlling for ambient temperature. All analyses were done using R software (version 4.0.1) and the mvmeta R package.[28]

Role of the funding source
The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results
A summary of study locations, periods, and number of deaths is shown in the appendix (pp 6–7). In total, 65·6 million all-cause deaths, 15·1 million cardiovascular deaths, and 6·8 million respiratory deaths were included in the analyses. Countries and regions contributed a median of 14·0 years (IQR 6·5). The maximum concentrations of estimated daily $PM_{2·5}$ induced by wildfires varied substantially by study location (figure 1). The highest daily concentrations of wildfire-related $PM_{2·5}$ (>100 μg/m³) were mainly estimated for cities in North America and east Asia, such as Saskatoon (Canada), Spokane (USA), Regina (Canada), and Chuncheon and Icheon (South Korea), whereas the lowest concentrations (<10 μg/m³) were mainly observed in Europe, such as Rennes and Paris (France), Bern (Switzerland), and Turin (Italy). 665 (89%) of the 749 cities had a mean concentration of estimated daily wildfire-related $PM_{2·5}$ of less than 2 μg/m³, with IQRs of less than 5 μg/m³ across all cities (appendix pp 17–18). Additional statistical information of wildfire-related $PM_{2·5}$ in study locations are shown in the appendix (pp 9–11).

When considering pooled associations between daily exposure to wildfire-related $PM_{2·5}$ and daily mortality during lag 0–7 days, we found that the effects tended to disappear after lag 2 days (figure 2); we thus focused on the effect estimates during lag 0–2 days. Wildfire-related $PM_{2·5}$ exposure was significantly associated with all-cause mortality at lag between 0 and 2 days, with the greatest risk at lag 0 days (RR 1·021 [95% CI 1·018–1·024] per 10 μg/m³ increase), followed by lag 1 day (1·014 [1·012–1·016]) and lag 2 days (1·005 [1·002–1·007]). Similar results were seen for cardiovascular mortality (1·017 [1·014–1·021] at lag 0 days, 1·013 [1·009–1·016] at lag 1 day, and 1·005 [1·001–1·009] at lag 2 days) and respiratory mortality (1·020 [1·015–1·024] at lag 0 days, 1·014 [1·010–1·019] at lag 1 day, and 1·006 [1·001–1·011] at lag 2 days).

The 3-day moving average of wildfire-related $PM_{2·5}$ (lag 0–2 days) was significantly associated with the three causes of mortality: RR 1·019 (95% CI 1·016–1·022) for all-cause mortality, 1·017 (1·012–1·021) for cardiovascular mortality, and 1·019 (1·013–1·025) for respiratory mortality. The pooled results for 3-day moving average of wildfire-related $PM_{2·5}$ at the country level are shown in table 1 and those pooled by WHO region and by GDP level are presented in the appendix (p 12). The highest unit RRs for all-cause mortality were observed in Europe, particularly in France, Italy, Germany, and Romania. The highest unit RRs for cardiovascular mortality were observed in Europe (including Portugal, Spain, and the Czech Republic) and the highest RRs for respiratory mortality were observed in Europe and Asia (including the Philippines, Sweden, and Kuwait; table 1).

When assessing the pooled concentration–response relationships between mortality and the 3-day moving average of wildfire-related $PM_{2·5}$, RRs initially increased with respect to concentrations for both all-cause and cardiovascular mortality, levelling out at around 20 μg/m³ (figure 3). For respiratory mortality, the same relationship occurred at lower concentrations, with RRs levelling out at around 15 μg/m³; however, a marked increase in RR was observed at concentrations greater than 30 μg/m³ (figure 3).

Based on the pooled global associations between mortality and the 3-day moving average of wildfire-related $PM_{2·5}$, an estimated 33 510 all-cause deaths (95% CI 26 204–40 763), 6993 cardiovascular deaths (5466–8510), and 3503 respiratory deaths (2739–4259) were attributable to acute wildfire-related $PM_{2·5}$ exposure annually on average, corresponding to PAFs of 0·62% (95% CI 0·48–0·75) for all-cause mortality, 0·55% (0·43–0·67) for cardiovascular mortality, and 0·64% (0·50–0·78) for respiratory mortality. PAFs are shown by country or region in table 2, and by WHO region and GDP level in the appendix (p 13), alongside the corresponding attributable numbers of deaths (p 16). The highest PAFs for all-cause mortality due to acute wildfire-related $PM_{2·5}$ exposure were observed in Thailand, Guatemala, Mexico, Paraguay, and Peru. WHO regions showing the highest PAFs for all-cause mortality were Central America (1·73%, 1·35–2·10), South-East Asia (1·63%, 95% CI 1·29–1·97), and South Africa (0·99%, 0·78–1·21); these three regions, alongside South America, also showed the highest PAFs for cardiovascular and respiratory mortality, with PAFs greater than 1·00% (appendix p 13).

Sensitivity analyses showed that the pooled results did not change substantially by further controlling for other-source $PM_{2·5}$ (appendix pp 19–20). The pooled results using adjusted and unadjusted wildfire-related $PM_{2·5}$ were consistent, although greater uncertainties were observed for results using unadjusted data (appendix pp 20–21). Lags of up to 2 days were sufficient to capture the lag effects of $PM_{2·5}$, as no significant associations of wildfire-related $PM_{2·5}$ exposure were observed during lags 3–10 days (appendix p 22). The results did not change substantially with use of 3, 5, or 6 degrees of freedom and 10-day lag effects for meteorological variables, or with controlling only for temperature in city-specific models (appendix pp 23–27).

	All-cause mortality	Cardiovascular mortality	Respiratory mortality
Argentina	1·040 (1·017–1·063)	NA	NA
Australia	1·002 (0·991–1·012)	NA	NA
Brazil	1·011 (0·998–1·024)	NA	NA
Canada	0·992 (0·978–1·007)	0·997 (0·974–1·021)	1·023 (0·994–1·053)
Chile	1·033 (1·010–1·056)	NA	NA
China	1·030 (1·005–1·055)	1·026 (0·982–1·073)	1·006 (0·977–1·036)
Colombia	1·028 (1·011–1·046)	1·023 (0·992–1·055)	1·002 (0·975–1·029)
Costa Rica	1·069 (1·032–1·106)	1·061 (0·995–1·131)	1·000 (0·994–1·006)
Czech Republic	1·073 (1·009–1·142)	1·070 (0·954–1·200)	1·039 (0·951–1·137)
Ecuador	1·010 (0·935–1·091)	1·033 (0·911–1·172)	1·066 (0·886–1·281)
Estonia	0·995 (0·955–1·036)	NA	NA
Finland	1·001 (0·997–1·005)	1·006 (0·999–1·012)	0·974 (0·869–1·070)
France	1·253 (1·079–1·455)	NA	1·022 (0·989–1·056)
Germany	1·126 (1·068–1·188)	NA	NA
Greece	1·009 (1·002–1·016)	1·015 (1·005–1·024)	1·010 (0·991–1·030)
Guatemala	0·996 (0·990–1·002)	NA	NA
Iran	1·014 (0·998–1·030)	1·030 (1·006–1·055)	1·006 (0·987–1·024)
Ireland	0·891 (0·751–1·057)	1·059 (0·713–1·573)	1·033 (0·997–1·070)
Italy	1·139 (1·081–1·199)	NA	NA
Japan	1·027 (1·022–1·032)	1·027 (1·017–1·036)	1·022 (1·005–1·040)
Kuwait	1·017 (0·989–1·045)	1·044 (1·006–1·083)	1·105 (0·973–1·239)
Mexico	1·002 (0·997–1·007)	1·001 (0·991–1·011)	1·072 (0·943–1·218)
Moldova	1·041 (0·916–1·182)	NA	NA
Netherlands	0·990 (0·842–1·164)	NA	NA
Norway	1·016 (0·995–1·036)	1·034 (1·000–1·067)	1·006 (0·987–1·024)
Panama	1·011 (0·918–1·111)	0·989 (0·839–1·161)	1·035 (0·949–1·126)
Paraguay	1·001 (0·999–1·003)	1·000 (0·997–1·003)	1·004 (0·999–1·009)
Peru	0·975 (0·957–0·994)	NA	NA
Philippines	1·008 (0·983–1·034)	1·000 (0·946–1·058)	1·171 (0·963–1·423)
Portugal	1·062 (0·999–1·130)	1·101 (0·967–1·254)	1·009 (0·975–1·043)
Puerto Rico	1·053 (1·003–1·106)	NA	NA
Romania	1·116 (1·075–1·158)	NA	NA
South Africa	1·018 (1·011–1·024)	1·016 (1·005–1·027)	1·016 (1·003–1·029)
South Korea	1·012 (1·000–1·024)	0·992 (0·974–1·010)	1·003 (0·991–1·015)
Spain	1·066 (1·030–1·104)	1·074 (1·026–1·126)	1·015 (0·994–1·038)
Sweden	0·946 (0·868–1·032)	1·007 (0·882–1·149)	1·145 (0·986–1·330)
Switzerland	1·026 (0·827–1·274)	1·059 (0·765–1·464)	0·926 (0·843–1·017)
Taiwan	1·034 (1·013–1·055)	1·009 (0·966–1·053)	1·035 (0·958–1·118)
Thailand	1·016 (1·013–1·020)	1·012 (1·005–1·018)	1·005 (0·996–1·015)
UK	1·023 (0·955–1·095)	1·061 (0·960–1·174)	1·022 (1·007–1·038)
Uruguay	1·019 (1·008–1·029)	NA	NA
USA	1·010 (1·001–1·020)	1·014 (0·998–1·031)	1·023 (1·015–1·030)
Vietnam	1·009 (0·950–1·071)	1·006 (0·953–1·062)	0·990 (0·786–1·246)

Data are RR (95% CI). RRs were associated with per 10 μg/m³ increase in moving average of wildfire-related $PM_{2·5}$ during lag 0–2 days. NA=not available. RR=relative risk.

Table 1: **Relative risks of mortality associated with exposure to wildfire-related $PM_{2·5}$ during lag 0–2 days in 43 countries and regions.**

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For more on the **MCC Collaborative Research Network** see http://mccstudy.lshtm.ac.uk

See Online for appendix

For more on the **GEOS-Chem model** see http://wiki.seas.harvard.edu/geos-chem

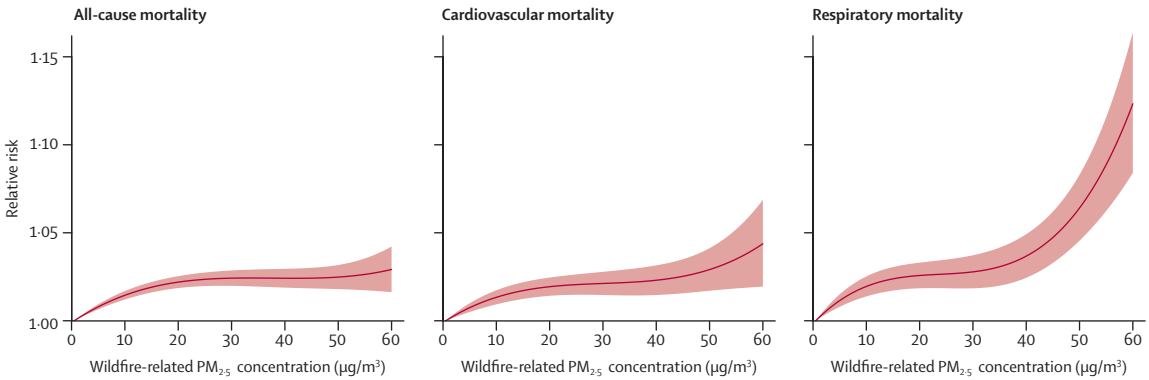

Figure 3: **The pooled concentration–response relationships between mortality and the 3-day moving average of wildfire-related $PM_{2·5}$ during lag 0–2 days**
Shaded areas represent 95% CIs.

Discussion

To our knowledge, this is the largest study evaluating associations between acute wildfire-related $PM_{2·5}$ and mortality, and the first to do so comprehensively across various regions of the world. We found that exposure to wildfire-related $PM_{2·5}$ was significantly associated with increased all-cause, cardiovascular, and respiratory mortality at a global level, but the associations varied across countries and regions.

The wildfire-related $PM_{2·5}$–mortality associations were assessed across various geographical regions and populations during a relatively long study period, based on the largest mortality dataset covering 43 countries and regions worldwide. With the use of a two-stage design, all city-specific associations between wildfire-related $PM_{2·5}$ and mortality were analysed in the same way, facilitating the comparison of results across different populations and regions.[17,18] The second stage random-effects meta-analysis has been widely used to examine both within-city and between-city variations regarding risk estimates.[29] The PAF was estimated using the pooled effect estimates with the same lag structure for each location, which provides essential information for public health planning and potential interventions.[30]

The results of our study are consistent with those of previous investigations, despite different effect estimates and exposure periods. However, previous studies were mainly restricted to a single study area or country, or a particular fire season. For example, a study in 27 countries in Europe estimated that 1483 premature deaths in 2005 and 1080 in 2008 could be attributable to vegetation fire-related $PM_{2·5}$.[15] Another study in Canada found that 54–240 premature deaths were attributable to wildfire-related $PM_{2·5}$ annually between 2013–15 and 2017–18.[9] Fixed and temporary ground monitors and satellite-based data have alternatively been used to estimate exposure to wildfire-related air pollutants, but these methods provide limited spatiotemporal coverage, low data quality of surface pollution level, and cannot quantify the contribution of fire smoke.[31] The GEOS-Chem model can address these problems by considering both non-fire and fire emissions. However, uncertainty in emissions data might affect the accuracy of estimation. For example, a study in North America reported that GFED-driven estimates matched well with observations, but showed overestimates and underestimates in some species and regions.[32]

Wildfire-related $PM_{2·5}$ undergoes long-range transport and continues to contribute to poor air quality even after fire seasons.[33] Therefore, evaluating health effects of wildfires should not be restricted to areas and time periods where and when wildfires occur. The pooled PAF of mortality attributable to acute wildfire-related $PM_{2·5}$ might seem low in terms of relative increase (<0·7%). This is caused by the special distribution of concentrations of wildfire-related $PM_{2·5}$ over time. Extremely high concentrations of wildfire-related $PM_{2·5}$ only occurred during fire seasons, which constituted a very short period relative to the whole study period, while wildfire-related $PM_{2·5}$ remained at a very low level during the long periods between fire seasons, with nearly 90% of cities having a mean concentration of estimated daily wildfire-related $PM_{2·5}$ of less than 2 μg/m³. However, the overall health impacts of wildfire-related $PM_{2·5}$ would be generally underestimated by this study. Wildfire-related $PM_{2·5}$ has both short-term and long-term health effects, but our study only focused on its short-term effects on mortality. More studies are needed in future to systematically examine its long-term effects on various health outcomes.

Our previous work on ambient $PM_{2·5}$ (mainly urban background $PM_{2·5}$) and daily mortality in 652 cities showed that all-cause mortality increased by 0·44% (95% CI 0·39–0·50), cardiovascular mortality by 0·36% (0·30–0·43), and respiratory mortality by 0·47% (0·35–0·58) with every 10 μg/m³ increase in $PM_{2·5}$ at lag 0–1 days.[18] By comparison, we found that wildfire-related $PM_{2·5}$ exposure had stronger effects on mortality (higher RRs) and a longer lag time than urban $PM_{2·5}$. The potential greater toxicity of wildfire $PM_{2·5}$ could reflect its higher fractions of small particles (eg, sub-micrometre particles and ultrafine particles) and

	All-cause mortality	Cardiovascular mortality	Respiratory mortality
Argentina	0·77% (0·60–0·93)	NA	NA
Australia	0·88% (0·70–1·07)	NA	NA
Brazil	0·70% (0·54–0·85)	NA	NA
Canada	0·33% (0·26–0·41)	0·33% (0·26–0·40)	0·32% (0·25–0·39)
Chile	0·43% (0·33–0·52)	NA	NA
China	0·66% (0·51–0·80)	0·67% (0·52–0·81)	0·65% (0·50–0·79)
Colombia	0·97% (0·76–1·18)	0·97% (0·76–1·18)	0·95% (0·74–1·16)
Costa Rica	0·94% (0·73–1·14)	0·92% (0·72–1·13)	0·92% (0·71–1·12)
Czech Republic	0·14% (0·11–0·17)	0·13% (0·10–0·16)	0·13% (0·10–0·16)
Ecuador	0·98% (0·76–1·19)	0·94% (0·74–1·15)	0·99% (0·77–1·20)
Estonia	0·17% (0·13–0·21)	NA	NA
Finland	0·14% (0·11–0·18)	0·15% (0·12–0·18)	0·13% (0·10–0·16)
France	0·12% (0·09–0·14)	NA	0·11% (0·09–0·14)
Germany	0·13% (0·10–0·16)	NA	NA
Greece	0·33% (0·26–0·40)	0·33% (0·25–0·40)	0·34% (0·26–0·41)
Guatemala	3·04% (2·39–3·68)	NA	NA
Iran	0·34% (0·26–0·41)	0·33% (0·26–0·41)	0·28% (0·22–0·34)
Ireland	0·09% (0·07–0·11)	0·09% (0·07–0·11)	0·08% (0·06–0·10)
Italy	0·30% (0·23–0·36)	NA	NA
Japan	0·63% (0·49–0·76)	0·61% (0·47–0·74)	0·61% (0·47–0·74)
Kuwait	0·37% (0·29–0·46)	0·37% (0·29–0·46)	0·36% (0·28–0·44)
Mexico	1·72% (1·35–2·09)	1·69% (1·32–2·05)	1·77% (1·39–2·15)
Moldova	0·27% (0·21–0·32)	NA	NA
Netherlands	0·13% (0·10–0·15)	NA	NA
(Continued from previous column)			
Norway	0·09% (0·07–0·12)	0·09% (0·07–0·12)	0·09% (0·07–0·11)
Panama	0·37% (0·29–0·45)	0·38% (0·29–0·46)	0·34% (0·26–0·41)
Paraguay	2·09% (1·64–2·54)	2·10% (1·65–2·55)	2·19% (1·72–2·65)
Peru	1·61% (1·26–1·96)	NA	NA
Philippines	0·79% (0·62–0·97)	0·80% (0·63–0·98)	0·78% (0·61–0·95)
Portugal	0·28% (0·22–0·34)	0·26% (0·20–0·31)	0·27% (0·21–0·33)
Puerto Rico	0·27% (0·21–0·34)	NA	NA
Romania	0·35% (0·27–0·42)	NA	NA
South Africa	0·99% (0·78–1·21)	1·00% (0·78–1·21)	1·10% (0·86–1·34)
South Korea	0·53% (0·41–0·64)	0·53% (0·41–0·64)	0·52% (0·41–0·63)
Spain	0·19% (0·15–0·23)	0·19% (0·14–0·23)	0·18% (0·14–0·22)
Sweden	0·10% (0·08–0·12)	0·10% (0·08–0·12)	0·10% (0·07–0·12)
Switzerland	0·15% (0·12–0·18)	0·15% (0·12–0·18)	0·15% (0·12–0·18)
Taiwan	0·58% (0·45–0·70)	0·57% (0·44–0·69)	0·57% (0·45–0·70)
Thailand	2·32% (1·83–2·80)	2·43% (1·92–2·93)	2·44% (1·92–2·94)
UK	0·09% (0·07–0·11)	0·09% (0·07–0·11)	0·08% (0·07–0·10)
Uruguay	0·51% (0·40–0·62)	NA	NA
USA	0·26% (0·20–0·32)	0·26% (0·20–0·31)	0·26% (0·20–0·31)
Vietnam	0·99% (0·78–1·21)	1·04% (0·81–1·27)	0·97% (0·76–1·18)

Data are PAF (95% CI). PAFs were calculated using the pooled global-level risk estimates. The corresponding number of deaths is shown in the appendix (pp 14–15). NA=not available. PAF=population attributable fraction.

Table 2: **PAF of annual mortality due to exposure to wildfire-related $PM_{2·5}$ during lag 0–2 days in 43 countries and regions**

more oxidative and proinflammatory components, such as polycyclic aromatic hydrocarbons and aldehydes.[34] Moreover, the joint effects of wildfire-related $PM_{2·5}$ and other pollutants, such as oxidant gases, might result in amplified health effects.[1]

Several limitations of this study should be noted. Although our MCC mortality data covered 43 countries and regions, they were not evenly distributed on every continent. The pooled mortality risk should not be interpreted as providing global results with high representativeness, as the analyses were mainly performed for urban populations. Some country-specific results might not fully represent the health effects for those countries owing to the small number of cities included in this study; in particular, 11 countries only had data for one city (appendix pp 6–7). Moreover, due to missing values or unavailability of data, the mortality data in some locations did not cover the full study period. Fire emissions generate a dynamic mixture of air pollutants that varies over space and time and that cannot be fully captured by the GEOS-Chem model.[35] We did not consider other air

pollutants from wildfires including carbon monoxide, carbon dioxide, or ozone. Moreover, the spatial resolution of estimation is coarse, which might underestimate the spatial variations of exposure and introduce exposure misclassification. The accuracy and spatial resolution of estimated wildfire-related $PM_{2·5}$ can be improved in future by including more detailed exposure data (eg, satellite-based data and weather data) with novel models. Finally, we did not analyse the association between wildfire-related $PM_{2·5}$ and mortality in susceptible subgroups of the populations (eg, by age or sex) owing to unavailability of individual information. If possible, such stratified analyses should be done in future studies to identify subpopulations vulnerable to wildfire air pollution.

This study provides robust epidemiological evidence for acute effects of wildfire-related $PM_{2·5}$ exposure on mortality, based on a large multicountry dataset and standard statistical method. Policy makers and public health professionals should raise awareness of wildfire pollution to guide prompt public responses and take actions to reduce exposure. Effective wildland management policies and practices should be implemented to manage vegetation and mitigate climate change as far as possible.

Contributors

YG, AG, MH, and BAr set up the collaborative network. YG and SL conceived, designed, and coordinated the study. YG, SL, and GC developed the statistical methods, took the lead in drafting the manuscript and interpreting the results, and verified the underlying data. YG and XY did the exposure assessment of wildfire-related $PM_{2·5}$. Other authors provided the data on mortality and temperature, and contributed to the interpretation of the results and to the submitted version of the manuscript. All authors had full access to all data and final responsibility to submit this paper for publication.

Declaration of interests

We declare no competing interests.

Data sharing

Data used in this study were collected by collaborators within the MCC Network under a data sharing agreement and cannot be made available publicly.

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