Mortality risk attributable to wildfire-related $PM_{2.5}$ pollution: \uparrow 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 PM2.5 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 wildfirerelated PM_{2.5} were estimated using the three-dimensional chemical transport model GEOS-Chem at a $0.25^{\circ} \times 0.25^{\circ}$ resolution. The association between wildfire-related PM_{3.5} 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_{1.5} 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", "PM25", "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}

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 PM2.5, far fewer studies have focused on health effects of wildfire-related PM2.5 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 339000 deaths could have been attributable to global landscape fire smoke annually during 1997-2006,16 but updated evidence from globalscale 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

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 μ g/m³ increase in the 3-day moving average of wildfire-related PM_{2.5} concentrations were 1·019 (95% Cl 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% Cl 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 $_{25}$ 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.

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 locationspecific 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°×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;

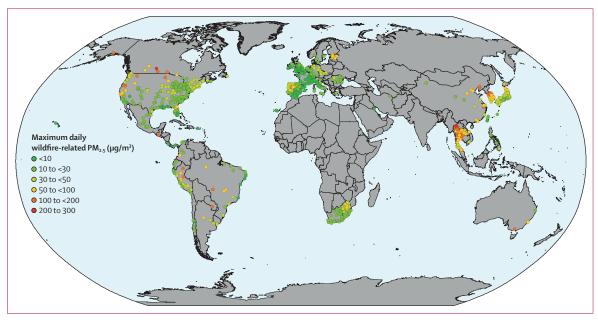


Figure 1: Maximum levels of estimated daily wildfire-related PM₂₅ in study locations during 2000-16

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

Daily enhancements of PM2.5 concentrations by fires during the study period were estimated as the differences between simulations with and without fire emissions. Daily concentration of wildfire-related PM2.5 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 $0.25^{\circ} \times 0.25^{\circ}$ using ground-level measurements of $PM_{2.5}$ and other predictors (eg, temperature, precipitation, wind speed, and day of the week). As wildfire-related PM2.5 was not routinely monitored, the GEOS-Chem-derived estimates of all-source PM2.5 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 PM2.5 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 PM2.5 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_{3.5} and death counts. Based on our previous work, ^{23,24} the single-day effect of wildfire-related PM2.5 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 cityspecific 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 I² 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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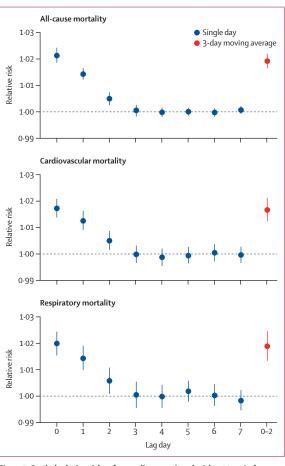


Figure 2: Pooled relative risks of mortality associated with a 10 μ g/m³ increase in wildfire-related PM₂₅ during lag 0-7 days Estimates show the single-day effects or 3-day moving average effect of wildfire-related PM₂₅ on mortality, with bars representing 95% Cls.

(I2 13-50%), and almost no heterogeneity for cardiovascular (I2: 0-10%) and respiratory (I2: 0-18%) mortality (appendix p 8). Therefore, based on the pooled globallevel 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 PM2.5 was calculated.24 First, the number of annual deaths attributable to wildfirerelated PM2.5 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 cityspecific 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 mymeta 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_{3.5} 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 PM2.5 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 $\mu g/m^3$ (figure 3). For respiratory mortality, the same relationship occurred at lower concentrations, with RRs levelling out at around 15 $\mu g/m^3$; however, a marked increase in RR was observed at concentrations greater than 30 $\mu g/m^3$ (figure 3).

Based on the pooled global associations between mortality and the 3-day moving average of wildfire-related PM_{2.5}, an estimated 33510 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 PM2.5 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 PM2.5 exposure were observed in Thailand, Guatemala, Mexico, Paraguay, and Peru. WHO regions showing the highest PAFs for allcause 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 othersource 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

	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	1.019 (1.008-1.029)	1.014 (0.998–1.031)	1.023 (1.015–1.030)
UJA	1.010 (1.001-1.020)	1.014 (0.330-1.031)	1.052 (1.012-1.030)

Data are RR (95% CI). RRs were associated with per 10 μ g/m³ increase in moving average of wildfire-related PM₂₅ during lag 0–2 days. NA=not available. RR=relative risk.

Table 1: Relative risks of mortality associated with exposure to wildfire-related PM $_{25}$ during lag 0–2 days in 43 countries and regions.

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).

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

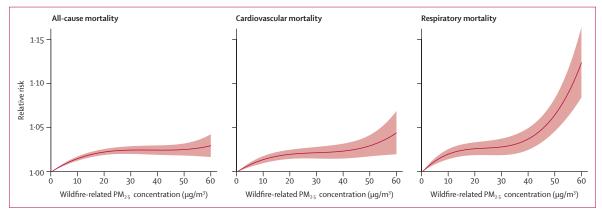


Figure 3: The pooled concentration-response relationships between mortality and the 3-day moving average of wildfire-related PM₂₅ during lag 0-2 days Shaded areas represent 95% Cls.

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 PM2.5.15 Another study in Canada found that 54-240 premature deaths were attributable to wildfire-related PM2.5 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 PM2.5 might seem low in terms of relative increase (<0.7%). This is caused by the special distribution of concentrations of wildfire-related PM2.5 over time. Extremely high concentrations of wildfire-related PM2.5 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 PM2,5 would be generally underestimated by this study. Wildfire-related PM2.5 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 $10 \,\mu\text{g/m}^3$ increase in $PM_{2.5}$ at lag 0–1 days. 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	Cardiovascular	Respiratory
	mortality	mortality	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%	0·33%	0·32%
	(0·26–0·41)	(0·26–0·40)	(0·25–0·39)
Chile	0·43% (0·33-0·52)	NA	NA
China	0.66%	0·67%	0·65%
	(0.51-0.80)	(0·52–0·81)	(0·50–0·79)
Colombia	0·97%	0·97%	0·95%
	(0·76–1·18)	(0·76–1·18)	(0·74–1·16)
Costa Rica	0·94%	0·92%	0·92%
	(0·73–1·14)	(0·72–1·13)	(0·71–1·12)
Czech Republic	0·14%	0·13%	0·13%
	(0·11-0·17)	(0·10-0·16)	(0·10–0·16)
Ecuador	0·98%	0·94%	0·99%
	(0·76–1·19)	(0·74–1·15)	(0·77-1·20)
Estonia	0·17% (0·13-0·21)	NA	NA
Finland	0·14%	0·15%	0·13%
	(0·11-0·18)	(0·12-0·18)	(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%	0·33%	0·34%
	(0·26-0·40)	(0·25-0·40)	(0·26-0·41)
Guatemala	3·04% (2·39–3·68)	NA	NA
Iran	0·34%	0·33%	0·28%
	(0·26-0·41)	(0·26-0·41)	(0·22-0·34)
Ireland	0·09%	0·09%	0·08%
	(0·07–0·11)	(0·07-0·11)	(0·06–0·10)
Italy	0·30% (0·23–0·36)	NA	NA
Japan	0·63%	0·61%	0·61%
	(0·49-0·76)	(0·47-0·74)	(0·47-0·74)
Kuwait	0·37%	0·37%	0·36%
	(0·29-0·46)	(0·29-0·46)	(0·28–0·44)
Mexico	1·72%	1·69%	1·77%
	(1·35–2·09)	(1·32–2·05)	(1·39-2·15)
Moldova	0·27% (0·21–0·32)	NA	NA
Netherlands	0·13% (0·10–0·15)	NA	NA
		(Table 2 continues	in next column

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

	All-cause	Cardiovascular	Respiratory
	mortality	mortality	mortality
(Continued from p	revious column)		
Norway	0·09%	0·09%	0·09%
	(0·07–0·12)	(0·07–0·12)	(0·07–0·11)
Panama	0·37%	0·38%	0·34%
	(0·29–0·45)	(0·29–0·46)	(0·26-0·41)
Paraguay	2·09%	2·10%	2·19%
	(1·64–2·54)	(1·65–2·55)	(1·72–2·65)
Peru	1·61% (1·26–1·96)	NA	NA
Philippines	0·79%	0·80%	0·78%
	(0·62–0·97)	(0·63-0·98)	(0·61–0·95)
Portugal	0·28%	0·26%	0·27%
	(0·22-0·34)	(0·20–0·31)	(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%	1·00%	1·10%
	(0·78–1·21)	(0·78–1·21)	(0·86–1·34)
South Korea	0·53%	0·53%	0·52%
	(0·41-0·64)	(0·41–0·64)	(0·41–0·63)
Spain	0·19%	0·19%	0·18%
	(0·15-0·23)	(0·14-0·23)	(0·14-0·22)
Sweden	0·10%	0·10%	0·10%
	(0·08–0·12)	(0·08–0·12)	(0·07–0·12)
Switzerland	0·15%	0·15%	0·15%
	(0·12-0·18)	(0·12-0·18)	(0·12-0·18)
Taiwan	0·58%	0·57%	0·57%
	(0·45-0·70)	(0·44-0·69)	(0·45–0·70)
Thailand	2·32%	2·43%	2·44%
	(1·83-2·80)	(1·92-2·93)	(1·92-2·94)
UK	0·09%	0·09%	0·08%
	(0·07-0·11)	(0·07–0·11)	(0·07-0·10)
Uruguay	0·51% (0·40-0·62)	NA	NA
USA	0·26%	0·26%	0·26%
	(0·20–0·32)	(0·20–0·31)	(0·20-0·31)
Vietnam	0·99%	1·04%	0·97%
	(0·78–1·21)	(0·81–1·27)	(0·76–1·18)
stimates. The corresp	onding number of o	ed using the pooled gl deaths is shown in the ation attributable frac	appendix

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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