International Journal of Epidemiology, 2024, **53(3)**, dyae066
https://doi.org/10.1093/ije/dyae066
Original article

Original article

Ambient fine particulate matter and daily mortality: a comparative analysis of observed and estimated exposure in 347 cities

Wenhua Yu [1], Wenzhong Huang,[1] Antonio Gasparrini [2], Francesco Sera [3], Alexandra Schneider,[4] Susanne Breitner,[4] Jan Kyselý,[5,6] Joel Schwartz,[7] Joana Madureira,[8,9] Vânia Gaio [10], Yue Leon Guo,[11,12,13] Rongbin Xu [1], Gongbo Chen,[1] Zhengyu Yang,[1] Bo Wen,[1] Yao Wu,[1] Antonella Zanobetti,[7] Haidong Kan [14], Jiangning Song [15], Shanshan Li[1] and Yuming Guo[1,*], Multi-City Multi-Country Collaborative Research Network

[1]Climate, Air Quality Research Unit, School of Public Health and Preventive Medicine, Monash University, Melbourne, Australia, [2]Department of Public Health Environments and Society, London School of Hygiene & Tropical Medicine, London, UK, [3]Department of Statistics, Computer Science and Applications 'G. Parenti', University of Florence, Florence, Italy, [4]Institute of Epidemiology, Helmholtz Zentrum München—German Research Center for Environmental Health (GmbH), Neuherberg, Germany, [5]Department of Climatology, Institute of Atmospheric Physics, Academy of Sciences of the Czech Republic, Prague, Czech Republic, [6]Faculty of Environmental Sciences, Czech University of Life Sciences, Prague, Czech Republic, [7]Department of Environmental Health, Harvard T.H. Chan School of Public Health, Boston, MA, USA, [8]Department of Environmental Health, Instituto Nacional de Saúde Dr Ricardo Jorge, Porto, Portugal, [9]EPIUnit—Instituto de Saúde Pública, Universidade do Porto, Porto, Portugal, [10]Department of Epidemiology, Instituto Nacional de Saúde Dr Ricardo Jorge, Lisboa, Portugal, [11]Department of Environmental and Occupational Medicine, National Taiwan University (NTU) College of Medicine and NTU Hospital, Taipei, Taiwan, [12]National Institute of Environmental Health Sciences, National Health Research Institutes, Miaoli, Taiwan, [13]Institute of Environmental and Occupational Health Sciences, NTU College of Public Health, Taipei, Taiwan, [14]Department of Environmental Health, School of Public Health, Fudan University, Shanghai, China and [15]Monash Biomedicine Discovery Institute, Department of Biochemistry and Molecular Biology, Monash University, Melbourne, Australia

*Corresponding author. School of Public Health and Preventive Medicine, Monash University, Melbourne, VIC 3004, Australia.
E-mail: yuming.guo@monash.edu

Abstract

Background: Model-estimated air pollution exposure products have been widely used in epidemiological studies to assess the health risks of particulate matter with diameters of ≤2.5 μm ($PM_{2.5}$). However, few studies have assessed the disparities in health effects between model-estimated and station-observed $PM_{2.5}$ exposures.

Methods: We collected daily all-cause, respiratory and cardiovascular mortality data in 347 cities across 15 countries and regions worldwide based on the Multi-City Multi-Country collaborative research network. The station-observed $PM_{2.5}$ data were obtained from official monitoring stations. The model-estimated global $PM_{2.5}$ product was developed using a machine-learning approach. The associations between daily exposure to $PM_{2.5}$ and mortality were evaluated using a two-stage analytical approach.

Results: We included 15.8 million all-cause, 1.5 million respiratory and 4.5 million cardiovascular deaths from 2000 to 2018. Short-term exposure to $PM_{2.5}$ was associated with a relative risk increase (RRI) of mortality from both station-observed and model-estimated exposures. Every 10-$\mu g/m^3$ increase in the 2-day moving average $PM_{2.5}$ was associated with overall RRIs of 0.67% (95% CI: 0.49 to 0.85), 0.68% (95% CI: –0.03 to 1.39) and 0.45% (95% CI: 0.08 to 0.82) for all-cause, respiratory, and cardiovascular mortality based on station-observed $PM_{2.5}$ and RRIs of 0.87% (95% CI: 0.68 to 1.06), 0.81% (95% CI: 0.08 to 1.55) and 0.71% (95% CI: 0.32 to 1.09) based on model-estimated exposure, respectively.

Conclusions: Mortality risks associated with daily $PM_{2.5}$ exposure were consistent for both station-observed and model-estimated exposures, suggesting the reliability and potential applicability of the global $PM_{2.5}$ product in epidemiological studies.

Keywords: Short-term exposure, fine particulate matter, model estimation, air monitoring station observation, mortality risk comparison.

Key Messages

- This study investigated the disparities in health effects between station-observed and model-estimated daily mean fine particulate matter ($PM_{2.5}$) and 15.8 million all-cause, 1.5 million respiratory and 4.5 million cardiovascular deaths across 15 countries from 2000 to 2018.
- Results found consistent mortality risks associated with daily $PM_{2.5}$ exposure from both station-observed and model-estimated data, with every 10-$\mu g/m^3$ increase in daily mean $PM_{2.5}$ associated with relative risk increase for mortality.
- The findings highlight the reliability and potential applicability of model-estimated $PM_{2.5}$ exposure assessments in epidemiological studies, supporting their use when direct measurement data are unavailable.

Received: 11 June 2023. **Editorial Decision:** 25 March 2024. **Accepted:** 13 April 2024

Introduction

Both acute and chronic exposure to particulate matter with diameters of ≤2.5 μm ($PM_{2.5}$) are associated with a range of adverse health outcomes, including cardiovascular and respiratory diseases, as well as premature deaths.[1–3] Ambient air quality monitoring station data provide precise exposure assessment for areas close to the monitoring stations and have been widely used in epidemiological studies to estimate the health impacts of air pollution.[4,5] However, reliance on a limited number of monitoring sites for exposure assessment may not adequately capture population exposure in large-scale air pollution epidemiological studies. Single station sites cannot represent air quality within whole cities, let alone across broader regions.[6]

Model-estimated air quality products play crucial roles in addressing the limitations of monitoring stations. They enable exposure estimates for populations far from stations or in regions where monitoring stations are unavailable.[7] Numerous air pollution modelling studies have been emerging to estimate both long-term (monthly to yearly) and short-term (hourly to daily) $PM_{2.5}$ exposure on regional and global scales.[8,9] These air quality models use either traditional statistical approaches or intricate hybrid machine-learning and deep-learning algorithms to provide accurate air pollution estimations with enhanced spatial coverage and spatiotemporal resolution.[5,9,10]

Increasing epidemiological studies have used estimated air pollution levels to assess the association between $PM_{2.5}$ exposure and adverse health outcomes.[3,11–13] However, to date, only a few studies have compared the difference in the exposure–response (E–R) relationships derived from station-observed air quality concentrations and model-estimated exposure.[6,13,14] Even though most studies found a consistent positive E–R association, health effect estimates varied across exposure assessment approaches.[6,15] Additionally, previous comparative studies have primarily focused on specific regions or cities.[7,12,15,16] To the best of our knowledge, no study has examined the difference in E–R with station-observed and model-estimated daily $PM_{2.5}$ in multiple countries and cities on a global scale. This study aimed to evaluate the reliability of employing model-estimated daily $PM_{2.5}$ in mortality risk assessment, using daily mortality data from 347 cities across 15 countries and regions worldwide with average period of 8.3 years from 2000 to 2018.

Methods

Data collection

The daily mortality data were collected from the Multi-City Multi-Country collaborative research network (MCC)—an international collaboration of research teams that seeks to produce epidemiological evidence on associations between environmental exposure and health.[4] We incorporated daily mortality statistics occurring from January 2000 through to December 2018. The daily counts of deaths resulting from all-cause diseases, cardiovascular disease [International Classification of Diseases, 10th revision (ICD-10) codes I00–I99] and respiratory disease (ICD-10 codes J00–J99) were obtained for MCC cities with available data during the study period. When all-cause mortality data were unavailable, non-accidental deaths (CD-10 codes A00–R99) were used to represent all-cause mortality.

The global station-observed daily mean $PM_{2.5}$ concentration data were obtained from official monitoring stations through multiple data sources worldwide, including the national and regional environmental protective agencies from the USA, Australia,[17] China, the European Environmental Agency[18] and other national government agencies.[9] To ensure the comparability of health risks estimated from station-observed and model-estimated $PM_{2.5}$ sources, our study was limited to MCC cities in which both ground-based $PM_{2.5}$ observations and model-estimated values were available during the study period. As a result, 1710 stations in 347 MCC cities from 15 countries and regions were ultimately included in the analysis. Figure 1 demonstrates the geographical distribution of the 347 locations and their observed mean daily $PM_{2.5}$ concentrations during the study time frame. A detailed description of the included monitoring stations in MCC cities and climate data collection can be found in the Supplementary material (available as Supplementary data at *IJE* online).

The grid-based model-estimated daily $PM_{2.5}$ data were retrieved from a global modelling product.[9] This product estimated global surface-level daily $PM_{2.5}$ concentrations at a high spatial resolution of 0.1°×0.1° from 2000 to 2019, using an innovative machine-learning framework. In brief, an innovative Deep Ensemble Machine Learning model was employed by integrating measurements in 5446 ground-based monitoring stations,[19] GEOS-Chem Chemical Transport Model simulations, satellite-based data, and meteorological and land cover information. The model achieved high consistency compared with the ground monitoring measurements, with a cross-validation R^2 of 0.91 and a root mean square error of 7.86 μg/m^3.[9] We calculated the average of the daily $PM_{2.5}$ concentrations from grid cells at the same centroid as the monitoring stations within a 10-km buffer in 347 MCC cities to compare the disparities in E–R associations. Data with missing values were excluded from the matched data set.

Data analysis

We used a two-stage analytical approach to estimate the associations of both station-observed and model-estimated daily $PM_{2.5}$ exposure with all-cause, respiratory and cardiovascular mortality separately.[20] This approach has been widely employed in previous multi-country studies.[4,21,22] In the first stage, the city-specific daily $PM_{2.5}$ E–R associations for each of the 347 cities were estimated using a quasi-Poisson regression. In line with previous short-term $PM_{2.5}$ mortality association studies,[4,23] we used a natural cubic spline function of calendar day with seven degrees of freedom (df) per year to control the seasonality and long-term trends; 'day of the week' was included in the model to account for the weekly variations. We included daily temperature and relative humidity using the natural cubic spline functions with three df and a maximum lag of 3 days to control for non-linear and delayed confounding impacts of weather conditions. Consistently with previous studies,[4,23] we assumed a linear E–R association and used a 2-day moving average (lag 0–1) of $PM_{2.5}$ concentrations (the mean of current and previous days) in our main analyses. We also explored the current-day (lag0) and previous-day (lag1) exposures to examine the delayed pattern of the mortality risks associated with short-term exposure to $PM_{2.5}$.

Figure 1. The distribution of ground-based daily mean fine particulate matter ($PM_{2.5}$) in 347 cities during the study period

In the second stage, we performed a random-effects meta-analysis to pool the city-level estimates obtained from the first stage to achieve the overall E–R associations.[20] We assessed the heterogeneity of effect estimates across cities using Cochran's Q-test and the I^2 statistic.[24] The results were reported as the relative risk increase (RRI) percentage for deaths associated with a 10-μg/m^3 increase in daily mean $PM_{2.5}$ concentrations: RRI = (Relative Risk – 1) ×100%. We also estimated the pooled nation-specific RRIs for all-cause, cardiovascular and respiratory deaths. The multivariate meta-regression method was used to examine the statistical differences in the RRIs from station-observed and model-estimated daily $PM_{2.5}$ data. We calculated the *P*-value to assess the magnitude of the difference between these two exposure sources. The details about the meta-regression analysis can be found in the Supplementary material (available as Supplementary data at *IJE* online).

Several sensitivity analyses were conducted to test the robustness of the E–R estimations. We examined potential non-linearity in the E–R relationships between short-term $PM_{2.5}$ exposure and mortality. Furthermore, we used different knots and df for temperature and $PM_{2.5}$ concentrations, as well as extending the maximum lag days from 2 to 5 days to examine the robustness of our estimations. We also examine the disparities in model-estimated $PM_{2.5}$ for mortality risk assessment by comparing them with an external data source from MCC monitoring stations.[4] Specifically, we exclusively selected MCC monitoring stations that were not used in the establishment of the Deep Ensemble Machine Learning model. Further details regarding the sensitivity analyses are presented in the Supplementary material (available as Supplementary data at *IJE* online).

Results

In total, 15.8 million all-cause or non-accidental deaths, 1.5 million deaths due to respiratory diseases, and 4.5 million cardiovascular deaths across 347 cities in 15 countries and regions were included in the study. Table 1 displays a summary of the mortality counts, daily mean $PM_{2.5}$ concentrations and correlation between station-observed and model-estimated $PM_{2.5}$ exposures in the MCC countries. The mean daily $PM_{2.5}$ concentration was 12.79 μg/m^3 (SD: 9.95) from station-based $PM_{2.5}$ observations and 12.82 μg/m^3 (SD: 8.32) from model-estimated exposure. The details about the comparison between station-observed and model-predicted daily $PM_{2.5}$ in the study period are presented in Supplementary Figure S1 and Supplementary Table S2 (available as Supplementary data at *IJE* online).

Figure 2 shows the comparison of the RRIs of all-cause, respiratory and cardiovascular mortality associated with station-observed and model-estimated daily mean $PM_{2.5}$ concentrations. Both station-observed and model-estimated $PM_{2.5}$ revealed positive relationships with all-cause, respiratory and cardiovascular mortality. The pooled RRIs for all-cause, respiratory and cardiovascular mortality were 0.67% (95% CI: 0.49 to 0.85), 0.68% (95% CI: –0.03 to 1.39) and 0.45% (95% CI: 0.08 to 0.82) per 10-μg/m^3 increase in the 2-day moving average $PM_{2.5}$ (lag0–1) from ground-based stations and 0.87% (95% CI: 0.68 to 1.06), 0.81% (95% CI: 0.08 to 1.55) and 0.71% (95% CI: 0.32 to 1.09) from model-estimated $PM_{2.5}$ data. Although the mortality effects for the model-estimated exposure were slightly higher than those for the observed exposure, no notable difference was observed ($P = 0.122$). Additionally, the RRIs for current (lag0) and previous-day (lag1) $PM_{2.5}$ exposure showed no difference between station-observed and model-estimated data. Figure 3 displays the nation-specific analysis of the RRIs in all-cause, respiratory and cardiovascular mortality associated with daily ground-observed and model-estimated $PM_{2.5}$ concentrations. We observed considerable variations in the estimated associations across study countries, with RRIs for all-cause deaths ranging from –1.06% (Norway) to 2.07% (Italy) for a 10-μg/m^3 increase in station-observed daily $PM_{2.5}$ concentrations (Supplementary Table S3, available as Supplementary data at *IJE* online). However, the nation-specific RRIs did not reveal an obvious difference between the station-observed and model-estimated $PM_{2.5}$ for the majority of the countries in the study.

We also explored the non-linear associations between $PM_{2.5}$ and all-cause, respiratory and cardiovascular mortality. As a result, no differences were found between the mortality effects based on station-observed and model-estimated $PM_{2.5}$ data, with the corresponding CIs largely overlapping with each other (Supplementary Figure S2, available as Supplementary data at *IJE* online). Although the overall difference was not substantial, the cardiovascular mortality estimates displayed a slightly pronounced variation between model-estimated and

Table 1. Descriptive statistic for the mortality counts, daily mean fine particulate matter ($PM_{2.5}$) concentrations and correlations from both ground-based observations and corresponding model-estimated values in study regions

Country/region	No. of cities	Mortality counts[a]			Observed $PM_{2.5}$ ($\mu g/m^3$)[b]		Estimated $PM_{2.5}$ ($\mu g/m^3$)[c]		r[e]
		All-cause	Respiratory	Cardiovascular	Mean	SD[d]	Mean	SD	
Australia	40	191 776	17 540	55 684	7.22	4.74	7.38	3.22	0.73
China	3	175 220	21 695	73 126	55.66	42.14	55.51	38.64	0.99
Czechia	4	375 938	22 922	176 795	23.49	19.85	22.92	14.92	0.87
Finland	1	103 183	5 470	37 115	8.88	5.28	9.70	4.82	0.86
France	19	545 339	34 963	NA	16.78	11.22	16.24	9.18	0.90
Germany	8	1 051 633	NA	NA	16.10	11.07	16.83	9.96	0.92
Italy	3	54 653	NA	NA	19.04	14.30	19.47	10.01	0.80
Netherlands	5	75 527	NA	NA	13.37	9.44	13.91	9.12	0.98
Norway	1	69 577	5544	17 477	10.85	6.07	10.70	4.96	0.89
Portugal	2	601 001	68 113	184 571	13.16	12.29	12.98	8.07	0.81
Spain	16	641 412	83 598	193 961	11.32	7.71	11.45	6.03	0.80
Sweden	3	398 460	28 389	156 802	10.22	6.43	10.71	5.64	0.86
Taiwan	6	227 233	28 194	52 641	23.40	11.99	24.09	10.17	0.92
United Kingdom	30	2 806 736	407 292	909 634	12.38	9.03	12.28	7.67	0.90
USA	206	8 510 553	840 970	2 648 452	12.89	8.35	12.90	6.70	0.89
Total	347	15 828 241	1 564 690	4 506 258	12.79	9.95	12.82	8.32	0.90

[a] NA indicates that the data on respiratory or cardiovascular deaths are unavailable for the country or region.
[b] Observed $PM_{2.5}$ refers to the daily average concentration of fine particulate matter with a diameter of <2.5 μm, as measured by monitoring stations.
[c] Estimated $PM_{2.5}$ denotes the daily $PM_{2.5}$ concentrations estimated by using a Deep Ensemble Machine Learning model at the same locations as the monitoring stations.
[d] SD refers to the standard deviation of the daily average $PM_{2.5}$ observed or estimated during the study period.
[e] 'r' indicates the correlation coefficient based on Spearman correlation analysis.

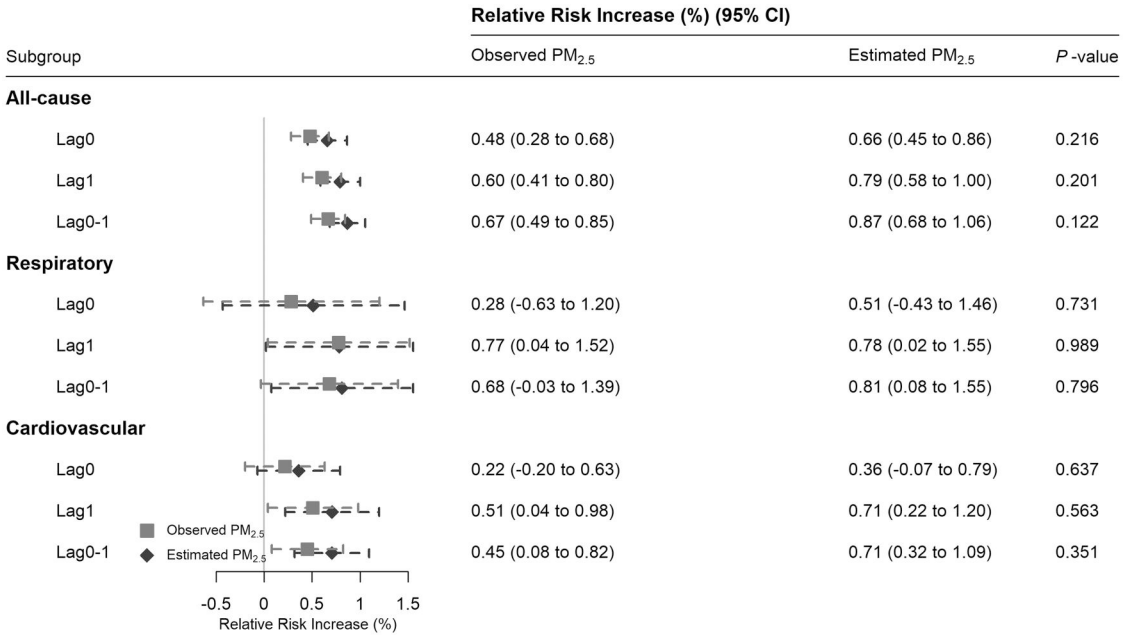

Figure 2. Comparison of the relative risk increase (%) for all-cause, respiratory and cardiovascular mortality associated with a 10-$\mu g/m^3$ increase in station-observed and model-estimated daily mean fine particulate matter ($PM_{2.5}$) with different lags. [a]95% CI of the relative risk increase. [b]*P*-value was calculated using the multivariate meta-regression method to indicate the statistical differences in the pooled relative risk increases from station-observed and model-estimated daily fine particulate matter ($PM_{2.5}$) data

station-observed $PM_{2.5}$ data, particularly at around 20 $\mu g/m^3$ (Supplementary Figure S2, available as Supplementary data at *IJE* online). Furthermore, after modifying the maximum lag days and parameters of the spline functions, the estimated RRIs from the station observations and the model-estimated $PM_{2.5}$ did not show obvious disparities (Supplementary Tables S4–S6, available as Supplementary data at *IJE* online). In terms of comparison with MCC monitoring stations, we found positive adverse E–R association for both station-observed and model-estimated $PM_{2.5}$ exposure, with overall RRIs of 0.98% (95% CI: 0.72 to 1.25) and 0.46% (95% CI: 0.19 to 0.73) for all-cause deaths separately (Supplementary Table S7, available as Supplementary data at *IJE* online).

Discussion

In this study, we used both station-observed and model-estimated $PM_{2.5}$ concentrations to investigate the

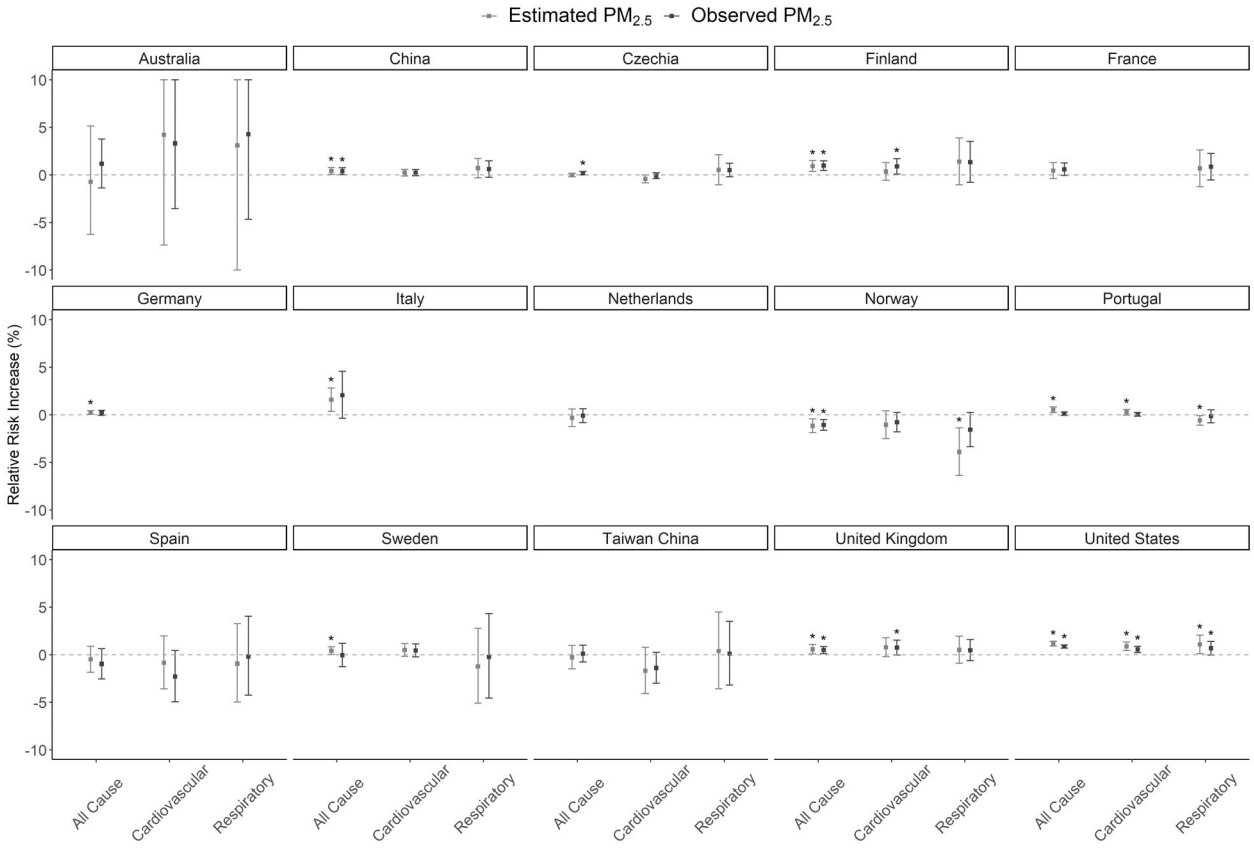

Figure 3. Comparison of the relative risk increase (%) for mortality associated with a 10-$\mu g/m^3$ increase in station-observed and model-estimated daily mean fine particulate matter ($PM_{2.5}$) by country. [a]* indicates that the CIs of the relative risk increase do not encompass zero, suggesting that the fine particulate matter ($PM_{2.5}$) exposure is positively or negatively associated with an increased risk of mortality

associations between daily $PM_{2.5}$ exposure and mortality. The results indicated that short-term exposure to $PM_{2.5}$ has consistent impacts on mortality, irrespective of the distinct $PM_{2.5}$ exposure assessment sources. Our findings underscored the reliability and potential applicability of the global model-estimated $PM_{2.5}$ product in health risk assessment.

The association between short-term exposure to $PM_{2.5}$ and mortality has been extensively studied. A recent systematic review synthesized the evidence on the effects of short-term exposure to air pollutants on all-cause, cardiovascular and respiratory mortality.[25] The authors identified positive associations between $PM_{2.5}$ and mortality, with RRIs of 0.65% (95% CI: 0.44 to 0.86) for all-cause deaths, 0.92% (95% CI: 0.61 to 1.23) for cardiovascular deaths and 0.73% (95% CI: 0.29 to 1.16) for respiratory deaths, respectively. Another study estimated short-term associations between daily $PM_{2.5}$ and mortality using data from 652 cities in the MCC network.[4] The results demonstrated RRIs of 0.68% (95% CI: 0.59 to 0.77) in all-cause deaths, 0.55% (95% CI: 0.45 to 0.66) in cardiovascular mortality and 0.74% (95% CI: 0.53 to 0.95) in respiratory mortality. Consistently with these studies, our research revealed similar positive associations between short-term daily $PM_{2.5}$ exposure and all-cause mortality, with RRIs of 0.67% (95% CI: 0.49 to 0.85) for station-observed $PM_{2.5}$ and 0.87% (95% CI: 0.68 to 1.06) for model-estimated $PM_{2.5}$.

Previous studies have examined the accuracy and reliability of model-estimated $PM_{2.5}$ concentrations in mortality risk assessment by comparing the effect estimates with observations from monitoring stations.[12,13,26–29] For example, Di *et al.* evaluated the disparities of an artificial neural network $PM_{2.5}$ estimation model prediction in assessing the associations of both short-term and long-term $PM_{2.5}$ exposure with all-cause mortality in older American adults by matching the data with the nearest air quality monitoring stations within 50 km.[12,13] The comparative analyses yielded consistent conclusions for the use of model-estimated exposure data in mortality risk assessment.

However, differences in health risk estimations exist between station-observed and model-estimated $PM_{2.5}$ exposure mainly due to exposure measurement errors. A recent review study revealed that measurement error could lead to an underestimation of E–R associations for time-series studies, resulting from the negative bias introduced by time-activity and spatial errors.[30] Feng *et al.* investigated the consequences of measurement errors in the associations between station-observed and model-estimated daily $PM_{2.5}$ exposures and all-cause mortality in Medicare beneficiaries in the USA.[14] They found an approximately 7% effect underestimation when using an ensemble machine-learning model-estimated $PM_{2.5}$. He *et al.* compared the estimated associations between cardiovascular admissions in New York and daily $PM_{2.5}$ exposures from five different exposure data sources.[6] The results showed that, despite consistently positive associations, the health impact assessments in different exposure models exhibited considerable disparities, with the highest E–R estimates originating from monitoring stations. Jerrett *et al.* also found that remote sensing data demonstrated notable associations between $PM_{2.5}$ and mortality, whereas the E–R estimates derived from ground-based data were generally higher

than those from remote sensing alone.[15] In our study, we observed slightly higher E–R estimates, especially for cardiovascular mortality in model estimations, compared with those from monitoring stations. Various factors could account for these elevated effect estimates. First, the comparison of the overall E–R estimates could be influenced by the location of the monitoring stations in certain countries. For example, consistently with previous study,[4] we observed an RRI of 1.17% for the model-estimated data in the USA, which was double that for monitoring stations (0.86%). Given the high proportion of monitoring stations in the USA (with 206 out of 347 MCC cities), the high RRI in the USA may contribute to a higher overall RRI for model-estimated $PM_{2.5}$ exposure than the monitoring stations. In addition, we recognize that taking averages of grid cells in each monitoring station may introduce uncertainties in the model-estimated exposure, subsequently influencing the RRI estimates in cities. Ground-based monitoring station data, especially within urban areas, usually exhibit higher spatiotemporal variations compared with the average of the model-estimated grid-based data.[6] However, the discrepancies in overall and national E–R estimates between station-observed and model-estimated exposures in our study were not statistically different ($P = 0.122$).

In general, the disparities in health effect estimates from monitoring stations and model-estimated $PM_{2.5}$ products originate from two primary components.[6] One stems from the model prediction errors, whereas the other arises from variations attributed to spatial aggregation.[6] Even though many air pollution modelling products have reported moderate or satisfied consistency with monitoring station observations, model prediction errors may arise because of the inherent assumptions embedded in the models and the inadequate control of confounding variables. The modelling accuracy varies across different spatial regions. For example, high spatiotemporal variations in measurement errors may exist especially in regions with certain specific features or areas without monitoring stations. Feng *et al.* observed that the areas with high elevation in East North and West South-Central US regions suffered higher biases in the model-estimated E–R estimation.[14] Many modelling studies—including the one currently employed—have shown that the model uncertainties exhibited spatial and temporal variations, especially in areas with limited or no monitoring stations.[9,31] Moreover, the distances between individuals and monitoring stations may also impact the accuracy of population exposure estimations. Lee *et al.* compared the Kriging model with satellite-based estimations and found that the satellite-based model achieved more accurate population exposure estimates for areas that were >100 km from monitoring stations.[32]

On the other hand, exposure measurement disparities could be introduced due to spatial aggregation. In this regard, He *et al.* found a huge difference in health effect estimates by comparing the E–R associations from four $PM_{2.5}$ estimation products with distinct spatial resolutions.[6] Kelly *et al.* concluded that finer-scale models would have high variations by comparing nine $PM_{2.5}$ exposure models.[33] Bai *et al.* found that spatial resolution greatly influenced the model-estimated $PM_{2.5}$ concentrations, with an overestimation of $PM_{2.5}$ occurring with coarser resolutions.[34] Wei *et al.* posited that $PM_{2.5}$ products estimated by models with finer spatial resolution are likely to yield reduced bias in assessing mortality risk.[35] However, further research is required to explore how spatial aggregation from different spatial resolutions impacts the estimates of the E–R association.

Numerous studies have reported distinct air pollution health effects in different countries, cities, and urban–rural regions.[4,6,14] In accordance with previous studies,[4,21] our findings show that the estimated national-level pooled E–R associations varied across countries. The observed disparities may be potentially explained by factors such as varied population susceptibility, socio-economic disparities, and the availability and quality of collected data. Additionally, it is essential to recognize that the data from the monitoring stations might not capture the complete spectrum of $PM_{2.5}$ concentrations across nations. However, the analyses based on data from 1710 monitoring stations in 347 MCC cities revealed that the effect estimates from both station-observed and model-estimated $PM_{2.5}$ exhibited consistent directions, suggesting marked reliability of our model-estimated $PM_{2.5}$ product in E–R assessments across the studied countries.

This study has several limitations. First, our comparative analysis is based on the city level and the pooled country-level effect estimates cannot be directly compared with each other due to the uneven distribution of included cities. The pooled country-level effect estimations also cannot fully represent the air pollution mortality risks across the entire country, given that the monitoring stations are predominantly located in urban environments. Similarly, the city-level exposure cannot completely represent the population exposure in the whole city due to the limited number of monitoring stations in cities. Additionally, we should caution that the E–R estimates were based on monitoring station data rather than actual personal exposures. The exposure misclassifications may impact the effect estimates due to population mobility and individual proactive protective behaviours against air pollution. However, our primary aim in this study is to assess the accuracy and reliability of the global $PM_{2.5}$ product in health risk estimates. This trade-off is worthwhile considering the great potential of deploying the global $PM_{2.5}$ product.

In conclusion, this study represents the first multi-country analysis to estimate the mortality effect of short-term $PM_{2.5}$ exposure from both station-observed and model-estimated daily $PM_{2.5}$ from 347 cities across 15 countries worldwide. We observed consistently positive adverse health effects of short-term $PM_{2.5}$ on deaths, regardless of whether the exposure assessment was derived from monitoring stations or the model-estimated global product. Consequently, these findings provide crucial evidence to support the reliability of the proposed high-resolution global daily $PM_{2.5}$ product for mortality risk assessment.

Notes

The Multi-City Multi-Country collaborative research network (MCC): Shilu Tong (National Institute of Environmental Health, Chinese Center for Disease Control and Prevention, Beijing, China; School of Public Health and Social Work, Queensland University of Technology, Brisbane, Australia), Mathilde Pascal (Santé Publique France, Department of Environmental and Occupational Health, French National Public Health Agency, Saint Maurice, France), Susana das Neves Pereira da Silva (Department of Epidemiology, Instituto Nacional de Saúde Dr Ricardo Jorge, Lisboa, Portugal), Aurelio Tobias [Institute of Environmental Assessment and Water Research (IDAEA), Spanish Council

for Scientific Research (CSIC), Barcelona, Spain], Carmen Íñiguez (Department of Statistics and Computational Research. Universitat de València, València, Spain; CIBERESP, Madrid. Spain), Shih-Chun Pan (National Institute of Environmental Health Science, National Health Research Institutes, Zhunan, Taiwan), Aleš Urban (Institute of Atmospheric Physics, Academy of Sciences of the Czech Republic, Prague, Czech Republic), Jouni J.K. Jaakkola [Center for Environmental and Respiratory Health Research (CERH), University of Oulu, Oulu, Finland; Medical Research Center Oulu, Oulu University Hospital and University of Oulu, Oulu, Finland], Niilo Ryti [Center for Environmental and Respiratory Health Research (CERH), University of Oulu, Oulu, Finland; Medical Research Center Oulu, Oulu University Hospital and University of Oulu, Oulu, Finland], Caroline Ameling [National Institute for Public Health and the Environment (RIVM), Centre for Sustainability and Environmental Health, Bilthoven, Netherlands], Danny Houthuijs [National Institute for Public Health and the Environment (RIVM), Centre for Sustainability and Environmental Health, Bilthoven, Netherlands], Shilpa Rao (Norwegian institute of Public Health, Oslo, Norway), Bertil Forsberg (Department of Public Health and Clinical Medicine, Umeå University, Sweden), Matteo Scortichini (Department of Epidemiology, Lazio Regional Health Service, Rome, Italy), Massimo Stafoggia (Department of Epidemiology, Lazio Regional Health Service, Rome, Italy), Pierre Masselot (Department of Public Health Environments and Society, London School of Hygiene & Tropical Medicine, London, UK).

Ethics approval

This study was approved by Monash University Human Research Ethics Committee (#24439).

Data availability

The mortality data used in this study were collected by the Multi-City Multi-Country collaborative research network (MCC) with a data-sharing agreement and cannot be openly available publicly. The daily station-observed and model-estimated data sets are available on request from the corresponding authors.

Supplementary data

Supplementary data are available at *IJE* online.

Author contributions

Y.G. and S.L. obtained funding. Y.G., J.S. and S.L. are the study guarantors. Y.G. conceived the study. W.Y. wrote both the original draft and performed the reviewing and editing under the supervision of Y.G., J.S. and S.L. W.Y. also performed the literature search, model establishment and analysis. W.H., A.G., F.S., A.S., B.S., J.K., J.S., J.M., V.G., YL.G., R.X., A.Z., Z.Y., B.W., Y.W., H.K. and other members of the MCC collaborative network mentioned in the notes contributed to the data collection and cleaning. W.H. and G.C. verified the analytical methods. W.Y. and G.C. contributed to the interpretation of parts of the results and code improvement. A.G., F.S., A.S., B.S., J.K., J.S., J.M., V.G. and YL.G. provided the edits in writing; Y.G., J.S. and S.L. supervised the project. Y.G. and S.L. accessed and verified the data and are responsible for the decision to submit the manuscript. All authors discussed the results and provided critical feedback and helped shape the final manuscript.

Funding

This work was supported by the Australian Research Council (grant number DP210102076); the Australian Medical Research Future Fund (grant number 2015916); the Australian National Health and Medical Research Council (grant numbers APP2000581, APP1163693 to Y.G., APP2008813 to Y.G. and APP2009866 to S.L.); Monash Graduate Scholarship and Monash International Tuition Scholarship (grant number 29790980 to W.Y.) and China Scholarship Council (grant number 202006380055 to W.H., 202006010043 to B.W. and 202006010044 to Y.W.).

Conflict of interest

None declared.

References

1. Cohen AJ, Brauer M, Burnett R *et al.* Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. *Lancet* 2017;**389**:1907–18.
2. Han C, Xu R, Ye T *et al.* Mortality burden due to long-term exposure to ambient PM2. 5 above the new WHO air quality guideline based on 296 cities in China. *Environ Int* 2022;**166**:107331.
3. Yu W, Xu R, Ye T *et al.* Estimates of global mortality burden associated with short-term exposure to fine particulate matter (PM2·5). *Lancet Planet Health* 2024;**8**:e146–55.
4. Liu C, Chen R, Sera F *et al.* Ambient particulate air pollution and daily mortality in 652 cities. *N Engl J Med* 2019;**381**:705–15.
5. Jin X, Fiore AM, Civerolo K *et al.* Comparison of multiple PM2. 5 exposure products for estimating health benefits of emission controls over New York State, USA. *Environ Res Lett* 2019; **14**:084023.
6. He MZ, Do V, Liu S *et al.* Short-term PM2. 5 and cardiovascular admissions in NY State: assessing sensitivity to exposure model choice. *Environ Health* 2021;**20**:93–11.
7. Bravo MA, Fuentes M, Zhang Y, Burr MJ, Bell ML. Comparison of exposure estimation methods for air pollutants: ambient monitoring data and regional air quality simulation. *Environ Res* 2012; **116**:1–10.
8. Di Q, Kloog I, Koutrakis P *et al.* 5 exposures with high spatiotemporal resolution across the continental United States. *Environ Sci Technol* 2016;**50**:4712–21.
9. Yu W, Ye T, Zhang Y *et al.* Global estimates of daily ambient fine particulate matter concentrations and unequal spatiotemporal distribution of population exposure: a machine learning modelling study. *Lancet Planet Health* 2023;**7**:e209–18.
10. Van Donkelaar A, Hammer MS, Bindle L *et al.* Monthly global estimates of fine particulate matter and their uncertainty. *Environ Sci Technol* 2021;**55**:15287–300.
11. Veronica A Southerland M, Michael Brauer M, Arash Mohegh P *et al.* Global urban temporal trends in fine particulate matter (PM2·5) and attributable health burdens: estimates from global datasets. *Lancet Planet Health* 2021;**6**:e139–46.
12. Di Q, Wang Y, Zanobetti A *et al.* Air pollution and mortality in the Medicare population. *N Engl J Med* 2017;**376**:2513–22.
13. Di Q, Dai L, Wang Y *et al.* Association of short-term exposure to air pollution with mortality in older adults. *JAMA* 2017; **318**:2446–56.

14. Feng Y, Wei Y, Coull BA, Schwartz JD. Measurement error correction for ambient PM2. 5 exposure using stratified regression calibration: effects on all-cause mortality. *Environ Res* 2023; **216**:114792.
15. Jerrett M, Turner MC, Beckerman BS *et al.* Comparing the health effects of ambient particulate matter estimated using ground-based versus remote sensing exposure estimates. *Environ Health Perspect* 2017;**125**:552–59.
16. McGuinn LA, Ward-Caviness C, Neas LM *et al.* Fine particulate matter and cardiovascular disease: comparison of assessment methods for long-term exposure. *Environ Res* 2017;**159**:16–23.
17. Riley M, Kirkwood J, Jiang N, Ross G, Scorgie Y. Air quality monitoring in NSW: From long term trend monitoring to integrated urban services. *Air Qual Clim Chang* 2020;**54**:44–51.
18. European Environment Agency. *Air Quality e-Reporting (AQ e-Reporting)*. 2022. https://www.eea.europa.eu/data-and-maps/data/aqereporting-9 (15 April 2023, date last accessed).
19. Yu W, Li S, Ye T, Xu R, Song J, Guo Y. Deep ensemble machine learning framework for the estimation of PM 2.5 concentrations. *Environ Health Perspect* 2022;**130**:37004.
20. Sera F, Gasparrini A. Extended two-stage designs for environmental research. *Environ Health* 2022;**21**:1–13.
21. Chen G, Guo Y, Yue X *et al.* Mortality risk attributable to wildfire-related PM2· 5 pollution: a global time series study in 749 locations. *Lancet Planet Health* 2021;**5**:e579–87.
22. Gasparrini A, Guo Y, Sera F *et al.* Projections of temperature-related excess mortality under climate change scenarios. *Lancet Planet Health* 2017;**1**:e360–67.
23. Chen R, Yin P, Meng X *et al.* Fine particulate air pollution and daily mortality. A nationwide analysis in 272 Chinese cities. *Am J Respir Crit Care Med* 2017;**196**:73–81.
24. Gasparrini A, Armstrong B, Kenward M. Multivariate meta-analysis for non-linear and other multi-parameter associations. *Stat Med* 2012;**31**:3821–39.
25. Orellano P, Reynoso J, Quaranta N, Bardach A, Ciapponi A. Short-term exposure to particulate matter (PM10 and PM2. 5), nitrogen dioxide (NO2), and ozone (O3) and all-cause and cause-specific mortality: systematic review and meta-analysis. *Environ Int* 2020;**142**:105876.
26. Son J-Y, Sabath MB, Lane KJ *et al.* Long-term exposure to PM2. 5 and mortality for the older population: effect modification by residential greenness. *Epidemiology* 2021;**32**:477–86.
27. Wang Y, Shi L, Lee M *et al.* Long-term exposure to PM2. 5 and mortality among older adults in the southeastern US. *Epidemiology* 2017;**28**:207–14.
28. Hart JE, Liao X, Hong B *et al.* The association of long-term exposure to PM 2.5 on all-cause mortality in the Nurses' Health Study and the impact of measurement-error correction. *Environ Health* 2015;**14**:38–39.
29. Yu W, Song J, Li S, Guo Y. Is model-estimated PM2. 5 exposure equivalent to station-observed in mortality risk assessment? A literature review and meta-analysis. *Environ Pollut* 2024; **348**:123852.
30. Richmond-Bryant J, Long TC. Influence of exposure measurement errors on results from epidemiologic studies of different designs. *J Expo Sci Environ Epidemiol* 2020;**30**:420–9.
31. Di Q, Amini H, Shi L *et al.* An ensemble-based model of PM2. 5 concentration across the contiguous United States with high spatiotemporal resolution. *Environ Int* 2019;**130**:104909.
32. Lee S-J, Serre ML, van Donkelaar A, Martin RV, Burnett RT, Jerrett M. Comparison of geostatistical interpolation and remote sensing techniques for estimating long-term exposure to ambient PM2. 5 concentrations across the continental United States. *Environ Health Perspect* 2012;**120**:1727–32.
33. Kelly JT, Jang C, Timin B *et al.* Examining PM2. 5 concentrations and exposure using multiple models. *Environ Res* 2021; **196**:110432.
34. Bai H, Shi Y, Seong M, Gao W, Li Y. Influence of spatial resolution on satellite-based PM2. 5 estimation: implications for health assessment. *Remote Sens* 2022;**14**:2933.
35. Wei Y, Qiu X, Yazdi MD *et al.* The impact of exposure measurement error on the estimated concentration–response relationship between long-term exposure to PM 2.5 and mortality. *Environ Health Perspect* 2022;**130**:77006.

International Journal of Epidemiology, 2024, 53, 1–8
https://doi.org/10.1093/ije/dyae066
Original article