

Mortality and long-term exposure to source-specific PM_{2.5}: evidence from a national cohort study in China

Xia Meng*, Yuchang Zhou*, Su Shi*, Shuxiao Wang, Maryam Zaid, Hongliang Zhang†, Jianlin Hu†, Gang Li, Haidong Kan, Maigeng Zhou†



Summary

Background The hazardous effects of fine particulate matter (PM_{2.5}) could vary by source. However, evidence regarding the long-term effects of source-specific PM_{2.5} on mortality is scarce, particularly in countries with high levels of pollution. We aimed to assess the associations between long-term exposure to source-specific PM_{2.5} and mortality in China, a low-income and middle-income country with high concentrations of PM_{2.5}.

Methods This nationwide cohort study used data from the 2010–11 wave of the China Chronic Disease and Risk Factor Surveillance (CCDRFS) project, which surveyed a geographically and socioeconomically representative sample of participants aged 18 years and older across mainland China. Participants were followed up through mortality information from the national Disease Surveillance Point system. Source-specific PM_{2.5} concentrations were estimated by combining high-resolution concentration predictions generated by a random forest model with estimates of the proportions contributed by each of six PM_{2.5} sources—industry, energy, transportation, residential, agriculture, and other—derived from the Community Multiscale Air Quality model. A Cox proportional hazards model, in which exposures were treated as time-varying covariates, was used to evaluate the associations between long-term exposure to source-specific PM_{2.5} and mortality from total non-accidental causes, cardiopulmonary disease, and lung cancer.

Findings 98 058 participants were followed from the date of their CCDRFS survey, conducted between January, 2010, and December, 2011, until death or Dec 31, 2020, whichever was earliest. Of these 98 058 participants, 96 955 were included in the analyses. The mean age was 46.5 years (SD 15.0); 52 631 (54.3%) participants were female and 44 324 (45.7%) were male. Almost all anthropogenic sources of PM_{2.5} were significantly associated with mortality. For each IQR increase, PM_{2.5} from industry (hazard ratio 1.079 [95% CI 1.024–1.137]), transportation (1.076 [1.034–1.119]), and residential (1.075 [1.029–1.124]) sources had the strongest associations with mortality from total non-accidental causes. For mortality from cardiopulmonary diseases, PM_{2.5} from transportation (1.106 [1.049–1.165]), industrial (1.085 [1.011–1.163]), and residential (1.070 [1.012–1.130]) sources had the strongest associations, and for mortality from lung cancer, the strongest associations were observed for PM_{2.5} from agricultural (1.256 [1.089–1.449]), industrial (1.235 [1.049–1.454]), and transportation (1.204 [1.048–1.384]) sources.

Interpretation We show that the mortality risks of PM_{2.5} exposure can vary according to the source. These findings could contribute evidence for the development of health-oriented policies to enhance the effectiveness of clean air initiatives.

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Introduction

According to estimates from the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2021, fine particulate matter (particles with an aerodynamic diameter $\leq 2.5 \mu\text{m}$; PM_{2.5}) contributed to approximately 4.72 million deaths globally in 2021, ranking as the second leading risk factor for mortality worldwide.¹ Over the past few decades, policies and technologies have resulted in substantially improved air quality in some areas.^{2–4} However, despite this progress, 99% of people worldwide are living in areas with unsafe concentrations of PM_{2.5}, and 34% are living in places where air pollution exceeds even the least strict interim

air quality target proposed by WHO.⁵ Strengthening interventions to further improve air quality is therefore crucial to protect public health globally.

PM_{2.5} is emitted from various sources, including vehicles, agricultural and industrial activities, and energy generation. Developing effective policies to reduce PM_{2.5} pollution ultimately requires the targeted management of specific emission sources. Currently, air quality guidelines set by WHO and assessments from GBD studies are mainly derived from exposure–response relationships that focus on total mass concentrations of PM_{2.5}.⁶ These approaches implicitly assume that all fine particles have the same

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*Contributed equally

†Joint senior authors

School of Public Health, Shanghai Institute of Infectious Disease and Biosecurity, Key Laboratory of Public Health Safety of the Ministry of Education and Key Laboratory of Health Technology Assessment of the Ministry of Health, Fudan University, Shanghai, China

(Prof X Meng PhD, S Shi MS, Prof H Kan PhD); National Center for Chronic and Noncommunicable Disease Control and Prevention, Chinese Center for Disease Control and Prevention, Beijing, China (Y Zhou PhD, Prof M Zhou PhD); State Key Joint Laboratory of Environmental Simulation and Pollution Control, School of Environment, Tsinghua University, Beijing, China (Prof S Wang PhD); Department of Epidemiology, School of Public Health, Fudan University, Shanghai, China (Prof M Zaid PhD); Department of Environmental Science and Engineering, Fudan University, Shanghai, China (Prof H Zhang PhD); Collaborative Innovation Center of Atmospheric Environment and Equipment Technology, Jiangsu Key Laboratory of Atmospheric Environment Monitoring and Pollution Control, Nanjing University of Information Science & Technology, Nanjing, China (Prof J Hu PhD); Department of Institute of Information and Statistics, Beijing Centre for Disease Prevention and Control, Beijing, China (Prof G Li PhD); Children's Hospital of Fudan University, National Center for Children's Health, Shanghai, China (Prof H Kan)

Correspondence to: Prof Haidong Kan, School of Public Health, Shanghai Institute

of Infectious Disease and Biosecurity, Key Laboratory of Public Health Safety of the Ministry of Education and Key Laboratory of Health Technology Assessment of the Ministry of Health, Fudan University, Shanghai 200032, China
 kanh@fudan.edu.cn
 or
 Prof Gang Li, Department of Institute of Information and Statistics, Beijing Centre for Disease Prevention and Control, Beijing 100013, China
 ligangcn@126.com

Research in context

Evidence before this study

We searched PubMed and Web of Science with the keywords ("PM_{2.5}" OR "fine particulate matter" OR "fine particles") AND ("source") AND ("long-term") AND ("total non-accidental" OR "cardiopulmonary disease" OR "lung cancer") for all studies published in English from database inception to Feb 28, 2025. We found few epidemiological studies reporting associations between long-term exposure to source-specific PM_{2.5} and mortality. However, the existing studies were mainly conducted in high-income countries (eg, the USA or countries in Europe) and mainly focused on only a few sources of PM_{2.5} (eg, transportation and industry). To date, no nationwide cohort study to our knowledge has investigated such associations in low-income and middle-income countries (LMICs), where PM_{2.5} concentrations and emission characteristics, as well as population demographic characteristics and socioeconomic status, differ those in from high-income countries.

Added value of this study

To the best of our knowledge, this is the first nationwide cohort study to examine the effects of long-term exposure to source-specific PM_{2.5} on mortality in an LMIC with high pollution levels. In this cohort study of 96 955 adults aged 18 years and older in China, we found that almost all anthropogenic sources of PM_{2.5} were significantly associated with mortality, with the strongest associations observed for PM_{2.5} from transportation, industry, and agricultural sources.

Implications of all the available evidence

Our findings show that the mortality risks of PM_{2.5} exposure vary by source. Identifying the health effects of PM_{2.5} from different sources is fundamentally important for policy making and for cost-benefit analyses of air pollution control. We highlight the necessity of considering source-specific health effects rather than assuming uniform toxicity of particulate matter, providing evidence for better targeted and more effective strategies for air quality management.

toxicity, without accounting for variations in their source or chemical composition.⁷ However, previous studies have suggested that different components of PM_{2.5} have differing toxicities, and because the composition depends on the source, the hazardous effects of PM_{2.5} from different sources should theoretically differ. Because the toxicity of various PM_{2.5} components is still inconclusive, and few studies have directly evaluated the hazardous effects of PM_{2.5} from different sources, little evidence is available from which to assess the disease burden of source-specific PM_{2.5} considering their unequal toxic effects. Understanding how the hazardous effects of PM_{2.5} differ across sources could support the development of better-targeted and more effective strategies for emission reduction, ultimately maximising public health benefits.

Evidence on the hazardous effects of PM_{2.5} from different sources is scarce. First, most studies focused on the short-term effects of exposure,^{8,9} with relatively few cohort studies providing long-term exposure-response functions from which the disease burden attributable to source-specific PM_{2.5} could be calculated. Second, the few cohort studies that have reported the hazardous effects of PM_{2.5} from different sources on mortality were conducted in high-income countries, such as the USA and countries in Europe;¹⁰⁻¹² evidence from low-income and middle-income countries (LMICs), where PM_{2.5} concentrations are higher and emission characteristics differ from those in high-income countries, is scarce. Finally, previous studies mainly focused on PM_{2.5} from only a few sources (eg, transportation and industry),¹⁰⁻¹² with little attention given to energy, residential, and agricultural sources, even though they are also major contributors to PM_{2.5}.

Using data from a nationwide cohort study, we aimed to investigate the associations between long-term exposure to

source-specific PM_{2.5} and mortality from total non-accidental causes, cardiopulmonary disease, and lung cancer.

Methods

Study design and participants

This national cohort study used data derived from the 2010-11 waves of the China Chronic Disease and Risk Factors Surveillance (CCDRFS) project, a nationally representative survey conducted in China between January, 2010, and December, 2011. Details of the CCDRFS design and sampling methods have been published elsewhere.^{13,14} In brief, participants aged 18 years and older were randomly selected using a multistage stratified sampling strategy to ensure geographical and socioeconomic representativeness across mainland China (appendix p 4).¹⁴ Participants were followed up through mortality information from the national Disease Surveillance Point (DSP) system. 161 sites were included in this study (figure 1). The study was approved by the ethical review committee of the Chinese Center for Disease Control and Prevention (201010), and all participants provided written informed consent.

Health data

Baseline data at the individual-participant level were obtained by the 2010-11 CCDRFS project through face-to-face interviews, with questions on personal characteristics, lifestyle risk factors, and medical history. The covariates were age, sex, BMI, educational level, marital status, smoking status and intensity, passive smoking exposure, alcohol consumption status and intensity, exercise intensity, daily fruit and vegetable intake, and history of stroke and acute myocardial infarction. In addition, gross

See Online for appendix

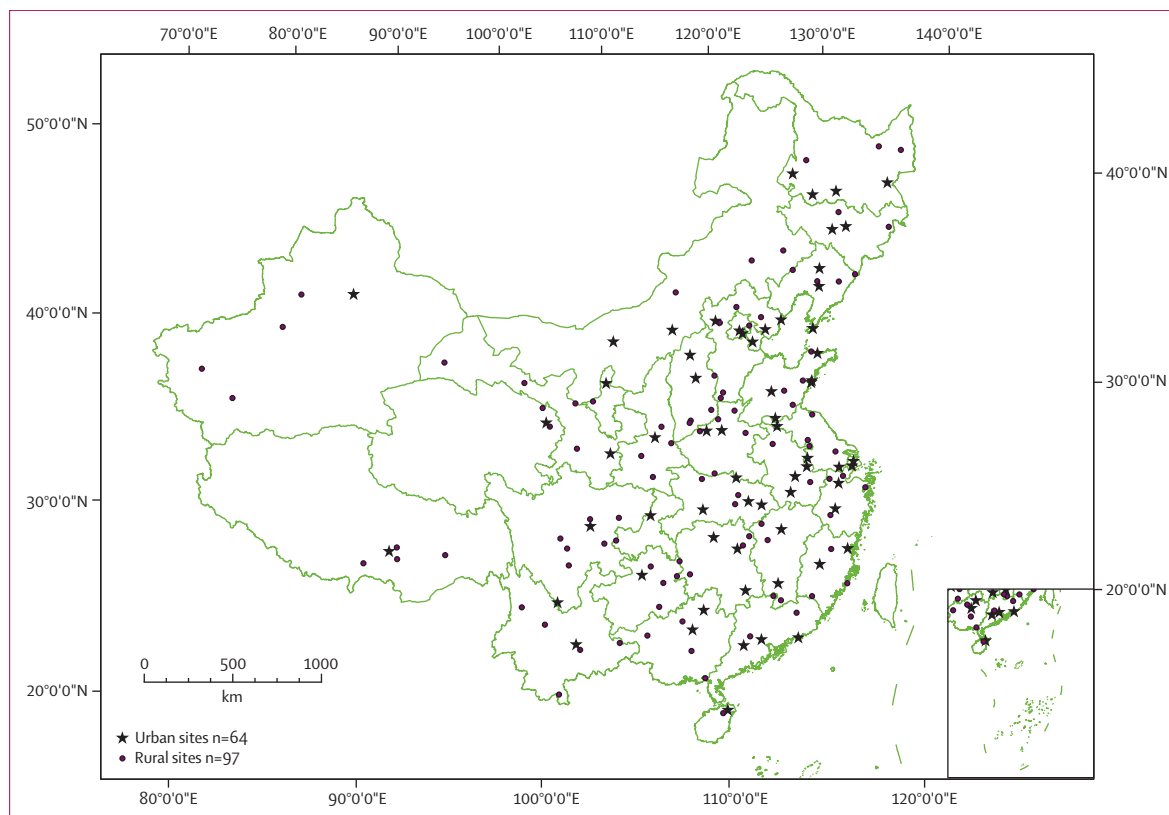


Figure 1: Spatial distribution of the 161 sites in this study

domestic product (GDP) and the number of hospital beds per 1000 people were also included (appendix p 5).

Each participant was tracked from enrolment until death or Dec 31, 2020, whichever was earliest, through linkage between the CCDRFS and DSP systems using unique identification numbers. Causes of death were determined and categorised by trained staff according to the ICD-10. Rigorous quality control procedures were implemented throughout the annual reviews of all mortality records. Deaths from total non-accidental causes (A00–R99), cardiopulmonary diseases (I00–I99 and J00–J99), and lung cancer (C34) were included in the analyses.

Exposure assessment

Source-specific $PM_{2.5}$ concentrations were estimated using a three-step approach that integrated machine learning and chemical transport modelling to leverage their complementary strengths (appendix p 27). Full details are given in the appendix (pp 6–7). In summary, daily $PM_{2.5}$ concentrations at a 1 km resolution were first predicted using random forest models incorporating ground measurements, satellite aerosol optical depth, and meteorological and land-use data,^{15,16} providing high spatiotemporal coverage and accuracy even in areas or periods without monitoring data. Second, the source-oriented Community Multiscale Air Quality (CMAQ) model (version 5.2) was applied to simulate the relative contributions of nine

sources of $PM_{2.5}$ (industry, energy, transportation, residential, agriculture, biogenic, sea salt, dust, and secondary organic aerosol) on the basis of emission inventories and meteorological fields.^{17–19} Although the CMAQ model can distinguish between sources, its spatial resolution is coarser and uncertainties higher than those of machine learning models. Therefore, in a third step, the source-specific proportions simulated by the CMAQ model were interpolated to the 1 km grid and multiplied by the high-resolution predictions of $PM_{2.5}$ concentration to generate daily, source-specific $PM_{2.5}$ concentrations. Exposure was assessed at an individual-participant level by linking the annual averaged source-specific $PM_{2.5}$ concentrations to each participant's residential address recorded during the baseline survey. To ensure the validity of effect estimates, $PM_{2.5}$ from sources contributing less than 5% of the total $PM_{2.5}$ concentration were grouped into a category denoted other.

Statistical analysis

We used a Cox proportional hazards model to evaluate the associations between total and source-specific $PM_{2.5}$ concentrations and mortality from total non-accidental causes, cardiopulmonary diseases, and lung cancer—the main outcome of the study. Separate models were fitted for total and source-specific $PM_{2.5}$ concentrations. To account for temporal variations during follow-up, exposures were

treated as time-varying covariates at annual intervals. Data were structured in a person-year format, with each participant contributing one record per calendar year. Exposure was evaluated annually from baseline to the end of follow-up. For mortality from total non-accidental causes and cardiopulmonary diseases, the annual mean PM_{2.5} concentration in the previous year ($t-1$) was assigned as the exposure for the year t . For lung cancer mortality, an 8-year moving average ($t-8$ to $t-1$) was used to account for the time lag between the exposure in year t and the development of lung cancer.²⁰ Exposures in the current year were excluded to avoid temporal misclassification or reverse-timing bias.

To investigate the independent effects of PM_{2.5} concentration on mortality, we constructed four models that systematically addressed confounding factors: individual baseline characteristics, community socioeconomic factors, lifestyle factors, and pre-existing medical conditions. Model 1 featured a basic adjustment for baseline characteristics. Model 2 was based on model 1 but further adjusted for GDP and the number of hospital beds per 1000 people, to address area-level socioeconomic disparities that could modify associations between PM_{2.5} exposure and mortality through environmental policies or health-care access. Model 3 was based on model 2 but also included additional lifestyle factors—fruit and vegetable consumption and exercise habits—to further control for behavioural confounders. Finally, model 4 included all variables in model 3 but further adjusted for pre-existing medical conditions, which could influence an individual's susceptibility to PM_{2.5}. Age (in 1-year intervals) and sex were included as stratification factors. The results from model 4 are reported in this study. For further details of the four models, see the appendix (p 13).

To estimate the shape of the associations between total and source-specific PM_{2.5} concentrations and mortality, concentration–response curves were plotted using the approach described in a previous study.¹³ In brief, a penalised spline function was applied in place of a linear term in model 4.

To investigate whether component-specific toxicity could explain the source-dependent effects of PM_{2.5}, we further assessed the associations between long-term exposure to five major PM_{2.5} components (black carbon, organic matter, and sulphate [SO₄²⁻], nitrate [NO₃⁻], and ammonium [NH₄⁺] ions) and mortality from total non-accidental causes, cardiopulmonary diseases, and lung cancer. Concentrations of the five major PM_{2.5} components were estimated using multiple predictors and XGBoost models, which has been reported in previous publications.^{21,22}

We conducted stratified analyses to explore the effects of age, sex, residential area, region, GDP, and the number of hospital beds per 1000 people. Several sensitivity analyses were conducted to assess the robustness of the results. Detailed methods for the stratified and sensitivity analyses are shown in the appendix (pp 8–9).

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

98 058 participants aged 18 years or older were followed up from the date of their CCDRFS survey, conducted between January, 2010, and December, 2011, until death or Dec 31, 2020, whichever was earliest. After excluding 1103 individuals with missing baseline data, 96 955 participants were included in the analyses. There were no significant differences between the excluded and included participants in terms of key demographic and lifestyle characteristics (appendix pp 14–15). The demographic characteristics of the eligible participants at baseline have been described in detail in previous studies¹³ and are summarised in table 1. The mean age of the 96 955 participants was 46.5 years (SD 15.0); 52 631 (54.3%) were female and 44 324 (45.7%) were male. During the follow-up period, 6418 (6.6%) of 96 955 participants died from total non-accidental causes, including 3640 (3.8%) from cardiopulmonary disease and 498 (0.5%) from lung cancer.

PM_{2.5} from anthropogenic sources was the primary contributor to total PM_{2.5} concentrations at the residences of participants at baseline. The highest PM_{2.5} fraction was attributed to industry (mean concentration 22.86 µg/m³ [SD 13.97]), followed by residential (11.41 µg/m³ [5.99]) and agricultural (7.20 µg/m³ [3.67]) sources (table 2). The concentrations of total and source-specific PM_{2.5} were highly correlated (appendix p 16).

The associations between long-term exposure to PM_{2.5} and increased risks of mortality from the three studied causes were generally significant across the four models. For model 4, hazard ratios (HRs) per IQR (30.90 µg/m³) increase in total mass concentrations of PM_{2.5} were 1.078 (95% CI 1.028–1.131) for total non-accidental causes, 1.070 (1.007–1.137) for cardiopulmonary disease, and 1.161 (1.004–1.343) for lung cancer (appendix p 17).

PM_{2.5} from almost all anthropogenic sources studied was significantly associated with the three causes of mortality (figure 2, appendix p 18). For mortality from total non-accidental causes, PM_{2.5} from industrial (HR 1.079 [95% CI 1.024–1.137]; IQR 16.84 µg/m³), transportation (1.076 [1.034–1.119]; 3.07 µg/m³), and residential (1.075 [1.029–1.124]; 7.36 µg/m³) sources showed the highest effect estimates per IQR increase in mass concentration. For mortality from cardiopulmonary diseases, PM_{2.5} from transportation (1.106 [1.049–1.165]; 3.07 µg/m³), industrial (1.085 [1.011–1.163]; 16.84 µg/m³), and residential (1.070 [1.012–1.130]; 7.36 µg/m³) sources had the strongest effect estimates. For mortality from lung cancer, PM_{2.5} from agricultural (1.256 [1.089–1.449]; 4.07 µg/m³), industrial (1.235 [1.049–1.454]; 16.84 µg/m³), and transportation (1.204 [1.048–1.384]; 3.07 µg/m³) sources had the highest effect estimates. The HR estimates for a 1 µg/m³ increase in PM_{2.5} concentration showed similar patterns (appendix p 18).

	Overall (n=96 955)	PM _{2.5} exposure (µg/m ³)			
		Q1 (14·00–33·15; n=24 595)	Q2 (33·15–52·40; n=23 814)	Q3 (52·40–75·90; n=23 808)	Q4 (75·90–124·65; n=24 738)
Age (years)	46·5 (15·0)	44·3 (14·6)	46·2 (14·7)	47·6 (15·1)	48·0 (15·1)
Sex					
Female	52 631 (54·3%)	12 860 (52·3%)	12 766 (53·6%)	13 295 (55·8%)	13 710 (55·4%)
Male	44 324 (45·7%)	11 735 (47·7%)	11 048 (46·4%)	10 513 (44·2%)	11 028 (44·6%)
BMI (kg/m ²)	23·9 (3·6)	23·4 (3·6)	23·5 (3·5)	24·3 (3·6)	24·5 (3·6)
Educational attainment*					
Elementary school or below	42 303 (43·6%)	13 800 (56·1%)	11 553 (48·5%)	7738 (32·5%)	9212 (37·2%)
Middle school	30 791 (31·8%)	7056 (28·7%)	7806 (32·8%)	7693 (32·3%)	8236 (33·3%)
High school	15 823 (16·3%)	2649 (10·8%)	3138 (13·2%)	5172 (21·7%)	4864 (19·7%)
Above high school	8038 (8·3%)	1090 (4·4%)	1317 (5·5%)	3205 (13·5%)	2426 (9·8%)
Marital status					
Never married	8300 (8·6%)	2151 (8·7%)	1845 (7·7%)	2292 (9·6%)	2012 (8·1%)
Married or cohabiting	78 768 (81·2%)	20 205 (82·2%)	19 124 (80·3%)	18 877 (79·3%)	20 562 (83·1%)
Divorced, separated, or widowed	9680 (10·0%)	2216 (9·0%)	2745 (11·5%)	2590 (10·9%)	2129 (8·6%)
Other	207 (0·2%)	23 (0·1%)	100 (0·4%)	49 (0·2%)	35 (0·1%)
Ethnicity					
Han ethnicity	82 480 (85·1%)	19 405 (78·9%)	20 360 (85·5%)	21 141 (88·8%)	21 574 (87·2%)
Ethnic minorities	14 475 (14·9%)	5190 (21·1%)	3454 (14·5%)	2667 (11·2%)	3164 (12·8%)
Smoking status					
Non-smoker	65 821 (67·9%)	16 305 (66·3%)	16 124 (67·7%)	16 494 (69·3%)	16 898 (68·3%)
Former smoker	5326 (5·5%)	1331 (5·4%)	1198 (5·0%)	1348 (5·7%)	1449 (5·9%)
Current smoker (≤1 pack per day)	11 000 (11·3%)	2727 (11·1%)	2542 (10·7%)	2839 (11·9%)	2892 (11·7%)
Current smoker (>1 pack per day)	2875 (3·0%)	738 (3·0%)	756 (3·2%)	634 (2·7%)	747 (3·0%)
Unknown	11 933 (12·3%)	3494 (14·2%)	3194 (13·4%)	2493 (10·5%)	2752 (11·1%)
Passive smoking					
No	6 399 (47·9%)	11 331 (46·1%)	11 020 (46·3%)	11 464 (48·2%)	12 584 (50·9%)
Yes	50 556 (52·1%)	13 264 (53·9%)	12 794 (53·7%)	12 344 (51·8%)	12 154 (49·1%)
Alcohol consumption status					
No	62 958 (64·9%)	15 054 (61·2%)	16 125 (67·7%)	15 375 (64·6%)	16 404 (66·3%)
Yes	33 997 (35·1%)	9541 (38·8%)	7689 (32·3%)	8433 (35·4%)	8334 (33·7%)
Daily intake of alcohol (g/day)	6·9 (20·9)	8·1 (23·0)	6·2 (20·0)	5·9 (18·5)	7·2 (21·7)
Daily intake of vegetables (g/day)	330·3 (269·0)	316·1 (296·3)	333·9 (265·8)	328·9 (252·6)	342·5 (258·1)
Daily intake of fruit (g/day)	85·1 (144·9)	70·7 (180·3)	75·0 (122·5)	102·4 (140·6)	92·6 (125·8)
Exercise intensity					
High-intensity exercise	4818 (5·0%)	1091 (4·4%)	967 (4·1%)	1417 (6·0%)	1343 (5·4%)
Medium-intensity exercise	11 327 (11·7%)	2166 (8·8%)	1747 (7·3%)	3880 (16·3%)	3534 (14·3%)
Absence of regular exercise	80 810 (83·3%)	21 338 (86·8%)	21 100 (88·6%)	18 511 (77·8%)	19 861 (80·3%)

Data are mean (SD) or n (%). Q=quartile. *Typical ages of attendance are ≤12 years for elementary school, 12–15 years for middle school; 15–18 years for high school; and ≥18 years for above high school.

Table 1: Characteristics of the study population by quartiles of PM_{2.5} exposure averaged from 2010–11 at baseline

For all three mortality outcomes, the highest effect estimates per 1 µg/m³ increase in mass concentration were seen for transportation-related PM_{2.5}, with HRs of 1·024 (1·011–1·037) for total non-accidental causes, 1·033 (1·016–1·051) for cardiopulmonary disease, and 1·062 (1·015–1·112) for lung cancer, followed by PM_{2.5} from energy and agricultural sources. No significant associations were observed between PM_{2.5} from other sources (biogenic sources, sea salt, dust, and secondary organic aerosols) and mortality.

The exposure–response curves for the associations between PM_{2.5} concentrations and mortality from total

non-accidental causes showed increased mortality at higher concentrations for total PM_{2.5} and all source-specific PM_{2.5}, with the curves characterised by an initial increase followed by a plateau (figure 3).

Five major PM_{2.5} components—black carbon, organic matter, and SO₄²⁻, NO₃⁻, and NH₄⁺ ions—were significantly associated with mortality from all three studied causes (appendix p19). For each IQR increase in component-specific PM_{2.5} concentration, organic matter, SO₄²⁻ ions, and black carbon showed the strongest association with mortality from total non-accidental causes and

	PM _{2.5} concentrations (µg/m ³)						
	Mean	SD	Minimum	25th percentile	Median	75th percentile	Maximum
Total PM _{2.5}	55.81	24.77	14.00	33.15	52.40	75.90	124.65
Industry	22.86	13.97	0.18	10.48	20.14	33.67	61.77
Energy	5.36	2.75	0.06	3.38	5.18	6.99	14.20
Transportation	2.85	1.96	0.04	1.49	2.19	3.93	9.29
Residential	11.41	5.99	0.45	6.66	10.22	15.31	32.34
Agriculture	7.20	3.67	0.74	4.25	6.65	10.26	16.89
Other	6.12	3.29	2.48	4.37	5.25	6.51	27.02

Other comprises PM_{2.5} from biogenic sources, sea salt, dust, and secondary organic aerosols, each contributing less than 5% of the total PM_{2.5} concentration.

Table 2: Total and source-specific PM_{2.5} concentrations at baseline (averaged from 2010–11) at the residences of participants

cardiopulmonary diseases, whereas SO₄²⁻, NH₄⁺, and NO₃⁻ ions had the highest effect estimates for lung cancer mortality. For each 1 µg/m³ increase in mass concentration, black carbon had the highest effect estimates for the three mortality outcomes, followed by NH₄⁺ and SO₄²⁻ ions.

Stratified analyses indicated significant effect differences between age subgroups and rural–urban subgroups for PM_{2.5} from some sources, whereas between-strata differences were not significant in other analyses (appendix pp 10–11, 30). The results remained robust across multiple sensitivity analyses (appendix pp 20–26, 31).

Discussion

To the best of our knowledge, this is the first nationwide cohort study to examine the effects of long-term exposure to source-specific PM_{2.5} on mortality in an LMIC with high pollution levels. Our analysis revealed that PM_{2.5} from different sources has different hazardous effects. Specifically, PM_{2.5} from anthropogenic sources—such as transportation, industry, and agriculture—showed the largest associations with mortality.

Our study contributes to the scarce evidence on the hazardous effects of source-specific PM_{2.5}. Previous studies are sparse and have predominantly focused on only a few specific sources.^{10–12} For example, the Cancer Prevention Study II, the California Teachers Study, and the ELAPSE Project mainly focused on PM_{2.5} from transportation and industrial sources.^{10–12} Our study also included PM_{2.5} from energy, residential, and agricultural sources and found significant associations with mortality, providing a comprehensive examination of the mortality risks associated with each sector. Moreover, previous studies were mainly conducted in high-income countries with low PM_{2.5} concentrations, such as the USA and countries in Europe, and the findings of these studies might not be applicable to areas with high PM_{2.5} concentrations and different emission characteristics. According to evidence from studies focusing on PM_{2.5} total mass concentrations, the effect estimates obtained from a relative risk model known as the global exposure mortality model differed markedly from estimates in the integrated exposure–response model, due

in part to the addition of data from a Chinese cohort exposed to high levels of pollution.^{23,24} As such, our study, which is based on data from a general population cohort in China, where PM_{2.5} concentrations are high, could provide crucial evidence to refine exposure–response curves for estimating the effects of exposure to total and source-specific PM_{2.5} on mortality.

On the basis of convergent evidence from previous studies and our component-based analysis, the differences in hazardous effects of PM_{2.5} from different sources can be attributed mainly to variations in the toxicity of the chemical components, the particle size distribution, and the emission characteristics. Regardless of whether exposure was assessed as a per-unit (1 µg/m³) increase or per IQR increase, PM_{2.5} from transportation sources produced among the strongest hazardous effects on population mortality, which can be explained as follows. First, as a result of incomplete combustion and inadequate exhaust treatment, vehicle emissions—particularly from diesel engines—release large amounts of black carbon, which is considered to be the most toxic component of PM_{2.5}.^{10,25,26} We also identified black carbon as having the strongest associations with mortality from total non-accidental causes and cardiopulmonary disease. Second, combustion conditions within vehicle engines facilitate the formation of high concentrations of ultrafine particles²⁷ that can penetrate deep into the lungs and enter the bloodstream,²⁸ exacerbating systemic inflammation.^{29,30} Finally, transportation is a major source of heavy metals and polycyclic aromatic hydrocarbons in PM_{2.5}, both of which could pose substantial risks to health.^{31,32}

We found that industrial, energy, and residential sources of PM_{2.5} were major contributors to total PM_{2.5} concentrations and posed significant risks to health. Industrial and energy-related emissions—primarily from power generation and the production of steel, cement, and chemicals—largely result from the combustion of fossil fuels,⁷ particularly coal, which releases abundant carbonaceous matter, sulphates, and nitrates.²⁵ Our component-based analysis corroborated the high toxicity of carbonaceous matter, whereas sulphate and nitrate ions showed weaker but still significant associations with total non-accidental and cardiopulmonary mortality. Residential sources of PM_{2.5}—including biomass and coal combustion, waste incineration, and cooking emissions—also contributed notably to mortality risks. These sources emit large amounts of organic matter and elemental carbon and generate ultrafine particles from incomplete combustion, which can enter the bloodstream and induce systemic inflammation and oxidative stress.^{29,30,33,34} Although industrial and residential sources were the largest contributors to total PM_{2.5} concentrations, their per-unit (1 µg/m³) effects were smaller than those of transportation sources. PM_{2.5} from agricultural sources was also significantly associated with mortality, consistent with a previous study.¹⁰ Agricultural activity—such as fertiliser use and livestock emissions—releases large amounts of ammonia,²⁵ which forms

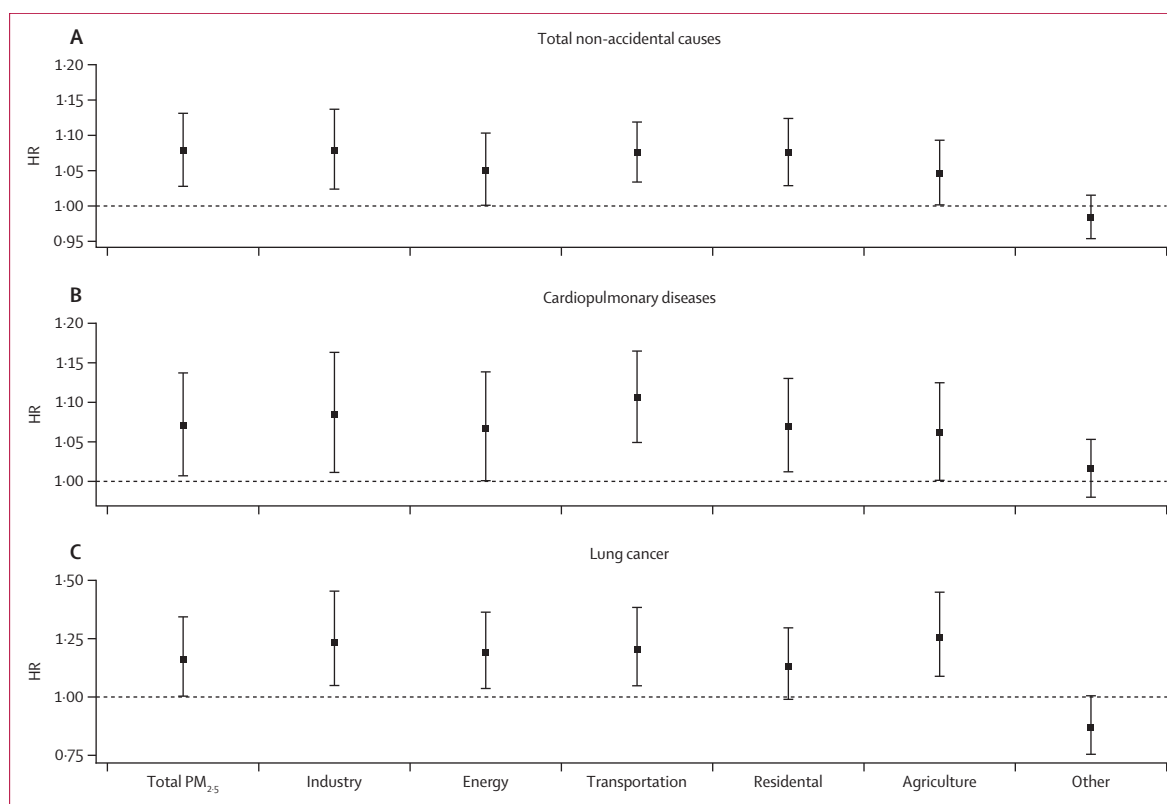


Figure 2: HRs for the associations between total and source-specific PM_{2.5} concentrations and mortality

Associations between an IQR increase in the concentrations of total and source-specific PM_{2.5} and mortality from total non-accidental mortality (A), mortality from cardiopulmonary diseases (B), and mortality from lung cancer (C). Data are HR (95% CI). The dotted lines indicate an HR of 1.000. All HRs were estimated using model 4. Other comprises PM_{2.5} from biogenic sources, sea salt, dust, and secondary organic aerosols, each contributing less than 5% of the total PM_{2.5} concentration. IQRs are 30.90 µg/m³ for total PM_{2.5}, and 16.84 µg/m³, 3.04 µg/m³, 3.07 µg/m³, 7.36 µg/m³, 4.07 µg/m³, and 2.16 µg/m³ for PM_{2.5} from industrial, energy, transportation, residential, agricultural, and other sources, respectively. HR=hazard ratio.

secondary compounds such as ammonium sulphate and ammonium nitrate through atmospheric reactions, both of which are harmful to health.^{33,34} Our component-based analysis further supported this concept, showing that sulphate and ammonium ions were associated with total non-accidental, cardiopulmonary, and lung cancer mortality per 1 µg/m³ increase in concentration. Moreover, agricultural emissions contain organic matter such as endotoxins and microbial fragments, which could induce inflammation and aggravate respiratory diseases.^{35–37}

We did not find significant associations between PM_{2.5} from other sources (biogenic sources, sea salt, dust, and secondary organic aerosols) and mortality. PM_{2.5} from these sources is generally present in low concentrations and shows little variability, limiting the capacity of this study to identify any associations. Further studies are needed to investigate the hazardous effects of PM_{2.5} from these sources.

This study provides evidence for policy-making efforts aimed at analysing the costs and benefits of pollution control strategies and further reducing PM_{2.5} concentrations to maximise public health benefits. Over the past few decades, many countries have made notable progress in improving

air quality through strict air pollution control measures.^{2–4} Governments have invested substantial funds in implementing air pollution control policies, with varying levels of expenditure and regulatory stringency across different emission sectors. Our results show that the mortality risk associated with PM_{2.5} varies by emission source, which could support more accurate cost–benefit assessments of air pollution control measures. However, despite these improvements in air quality, global PM_{2.5} concentrations remain considerably higher than suggested by WHO air quality guidelines.⁶ In China, where PM_{2.5} concentrations remain particularly high despite substantial previous improvements, achieving further improvements has become increasingly challenging,³⁸ highlighting the urgent need for more effective and targeted air pollution control strategies. Although many studies have examined the hazardous effects of PM_{2.5} components,^{39,40} effective air pollution control ultimately requires the management of emission sources rather than a sole focus on individual components. Our study provides a scientific basis for considering both the concentrations and the hazardous effects of pollutants when making air quality control policies for various emission sectors, in order to maximise health

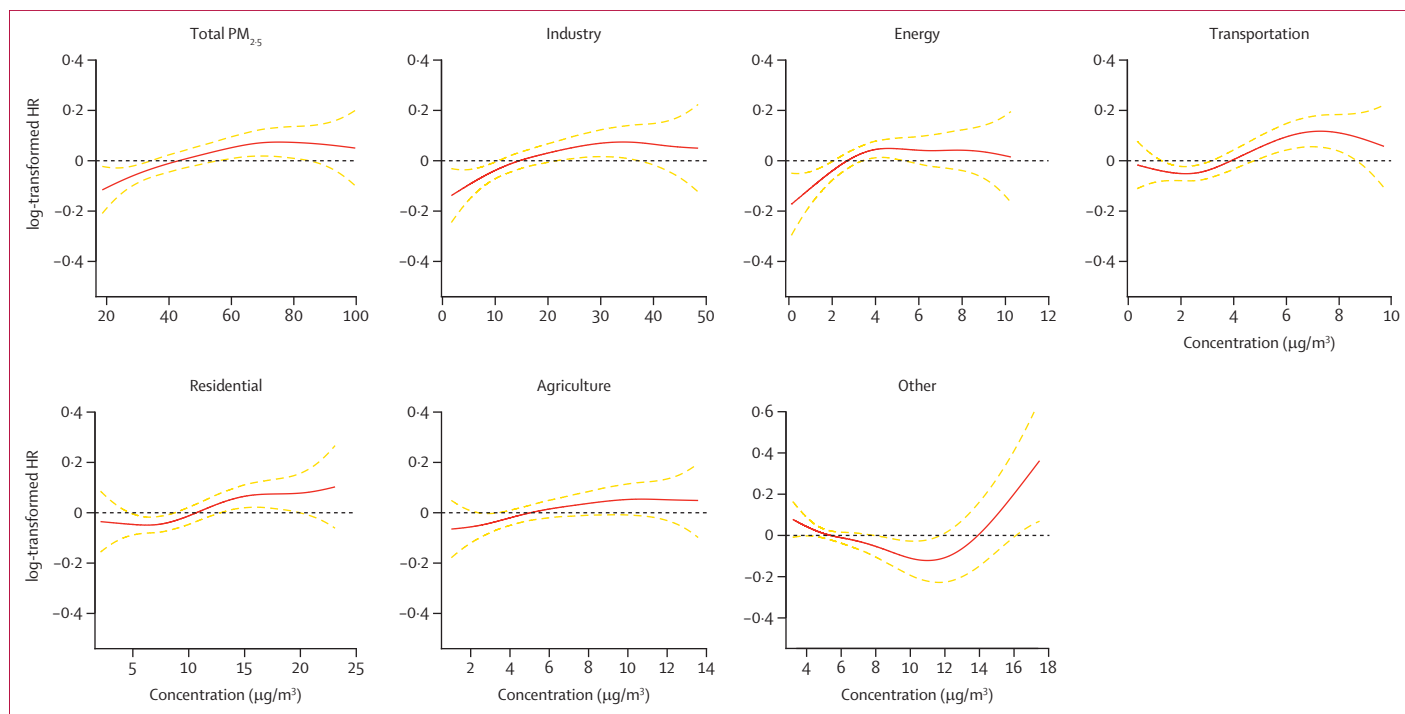


Figure 3: Exposure-response curves showing the associations between total and source-specific PM_{2.5} concentrations and mortality from total non-accidental causes

Data are log-transformed HR (95% CI). The dotted black lines indicate a value of zero. All HRs were estimated using model 4. Other comprises PM_{2.5} from biogenic sources, sea salt, dust, and secondary organic aerosols, each contributing less than 5% of the total PM_{2.5} concentration. HR=hazard ratio.

benefits. We found that PM_{2.5} from transportation, energy, and agricultural sources had the greatest hazardous effects for a per-unit (1 µg/m³) increase in concentration, whereas PM_{2.5} from transportation, industrial, and agricultural sources had the largest effects per IQR increase in concentration. Future air pollution control strategies in China could therefore prioritise addressing anthropogenic sources of PM_{2.5}, such as transportation, industry, and agriculture.

This study has several strengths. First, the cohort design enables us to obtain strong evidence on the differential mortality risks of source-specific PM_{2.5}. Second, this study was conducted in regions with high PM_{2.5} concentrations, addressing the limitations of previous research that mainly focused on high-income countries with low pollution levels; these results could therefore help to refine exposure-response relationships in the case of high pollutant concentrations. Third, this study comprehensively assessed the risk of mortality on exposure to PM_{2.5} from multiple sources using a unified method, providing valuable insights into targeted air pollution control strategies. Fourth, our study used unified datasets and methods to simultaneously analyse the differential health effects of major PM_{2.5} components and source-specific PM_{2.5} emissions. This component-based assessment provided crucial mechanistic evidence that corroborates the observed disparities in the health effects of source-specific PM_{2.5}, establishing a dual-validation framework for pollution source apportionment in epidemiological studies.

This study has several limitations. First, we used the CMAQ model to estimate source-specific PM_{2.5} concentrations. Although adjustments were made to improve accuracy, potential bias in the classification of emissions could have been introduced. Nevertheless, the model was driven by the Multi-resolution Emission Inventory Model for Climate and Air Pollution Research, which integrates data from various sources—including Chinese Energy Statistics and the Ministry of Ecology and Environment, among others—and is considered one of the most accurate emission inventories available in China.^{41,42} Second, as with most registration databases, under-reporting could exist in the DSP system. However, the under-reporting rate is likely to be low and is considered acceptable, as the reporting of death is legally mandated in China and is supported by comprehensive quality-control measures. Additionally, the DSP system has been widely used in governmental and international reports, showing its reliability for research.^{13,43} Third, because follow-up data were not available, PM_{2.5} exposure was assessed on the basis of participants' residential addresses at baseline, potentially leading to the misclassification of exposure. However, this limitation is unlikely to substantially affect the results, given that frequent relocation is uncommon in Chinese culture. Additionally, previous studies have indicated that residential mobility has a minimal effect on the observed associations between long-term or lifetime exposure to pollutants and mortality.^{44,45} Fourth, individual-level covariates—such as smoking, diet, and physical activity—were self-reported

and assessed only at baseline, which could introduce recall error and non-differential misclassification that typically bias the observed associations towards the null.⁴⁶ Fifth, indoor air pollution and the use of mitigation measures (eg, range hoods and air purifiers) were not assessed; these factors could influence personal PM_{2.5} exposure and should be examined in future research. Finally, air pollution concentrations were assessed at a 1 km resolution, which could lead to non-differential exposure misclassification—particularly in urban areas with a high spatial heterogeneity of PM_{2.5} pollution. However, such a Berkson error would be likely to bias effect estimates towards the null,⁴⁶ and therefore would not change the conclusions derived from this study. In the future, large cohort studies with source apportionment of PM_{2.5} at the individual-participant level could help to validate the findings from this study.

To our knowledge, this is the first cohort study conducted in an LMIC with high pollution concentrations to examine the effects of source-specific PM_{2.5} on mortality. The results showed that risks from PM_{2.5} vary by source, with PM_{2.5} from transportation, industry, and agriculture having the largest effects on population mortality. Our study provides valuable evidence for developing health-oriented policies to further reduce PM_{2.5} concentrations.

Contributors

XM, SS, and HK contributed to the conceptualisation of the study and the development of the methods. XM, YZ, SS, HZ, JH, GL, HK, and MZh contributed to data collection. XM and SS contributed to the visualisation of the figures and tables. XM, SS, MZa, SW, and HK prepared the draft of the manuscript, which was revised by XM, YZ, SS, MZa, SW, HK, and MZh. GL, HK, and MZh supervised the data analysis and writing of the manuscript. XM and SS accessed and verified the data. All authors had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Declaration of interests

We declare no competing interests.

Data sharing

The source-specific PM_{2.5} dataset is available from the corresponding authors upon reasonable request.

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