J Korean Med Sci. 2025 Jul 14;40(27):e156
https://doi.org/10.3346/jkms.2025.40.e156
eISSN 1598-6357·pISSN 1011-8934

JKMS

Original Article

Public Health & Preventive Medicine

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A Systematic Review and Meta-Analysis on Long-Term Exposure to Particulate Matter and All-Cause and Cause-Specific Mortality in the Asia-Pacific States

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OPEN ACCESS

Received: Aug 6, 2024
Accepted: Jan 2, 2025
Published online: May 12, 2025

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ABSTRACT

Background: Global Burden of Disease (GBD) studies have proposed integrated exposure-response models primarily based on North American and European data, which may not be directly applicable to the Asia-Pacific region. Through a systematic review and meta-analysis, we aimed to explore the association between long-term exposure to ambient particulate matter (PM) and mortality in the Asia-Pacific states.

Methods: We searched 3 databases (PubMed [n = 8,326], Embase [n = 4,709], and Cochrane Library [n = 357]) between 1st January 1990 and 31st July 2023. Our search focused on studies examining the associations between long-term exposure to PM with an aerodynamic diameter < 2.5 μm ($PM_{2.5}$) and 10 μm (PM_{10}) and all-cause (or non-accidental) and cause-specific mortality, including cardiovascular disease (CVD), ischemic heart disease (IHD), stroke, acute lower respiratory infection, chronic obstructive pulmonary disease, and lung cancer deaths in the Asia-Pacific states. We conducted a meta-analysis to pool the estimates in the studies.

Results: We identified 71 articles investigating the association between long-term exposure to PM and all-cause and cause-specific mortality. For a 10 μg/m^3 increase in $PM_{2.5}$, the pooled relative risk (RR) with 95% confidence intervals (CI) for all-cause mortality was 1.11 (95% CI, 1.05–1.17), 1.13 (95% CI, 1.06–1.21) for CVD, 1.13 (95% CI, 1.02–1.25) for IHD, 1.12 (95% CI, 1.02–1.24) for stroke, and 1.12 (95% CI, 1.08–1.16) for lung cancer. For a 10 μg/m^3 increase in PM_{10}, the pooled RR for all-cause mortality was 1.12 (95% CI, 1.00–1.24) and 1.33 (95% CI, 1.28–1.38) for IHD.

Conclusion: Our analysis revealed positive associations between long-term exposure to PM and all-cause and cause-specific mortality for CVD, IHD, stroke, and lung cancer in the Asia-Pacific states.

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Trial Registration
PROSPERO Identifier: CRD42023441916

Funding
This study was partially supported by the project titled "Institute of Ewha-SCL for Environmental Health (IESEH)" and Research of Environmental Examination Model for Children and Women (No. 1-2022-0205-001-3). Additional support was provided by the Korea Environment Industry & Technology Institute (KEITI) through the Digital Infrastructure Building Project for Monitoring, Surveying, and Evaluating Environmental Health, funded by the Korea Ministry of Environment (MOE) (grant No. RS-2021-KE001615).

Disclosure
The authors have no potential conflicts of interest to disclose.

Data Sharing Statement
Data sharing statement is provided in **Supplementary Material 1**.

Data Availability Statement
All meta-analyses described in this study can be obtained from the corresponding author upon request, including the data and analytic code used in the analyses.

Author Contributions
Conceptualization: Ha E, Lim YH. Data curation: Oh J, Hevia-Ramos G. Formal analysis: Oh J, Hevia-Ramos G. Investigation: Oh J, Hevia-Ramos G, Ha E. Methodology: Oh J, Hevia-Ramos G, Ha E, Hong YC, Kim H. Supervision: Lim YH. Writing - original draft: Oh J, Hevia-Ramos G, Ha E, Hong YC, Kim H, Lim YH.

Trial Registration: PROSPERO Identifier: CRD42023441916

Keywords: Asia-Pacific States; Ambient; Particulate Matter; Mortality; Systematic Review; Meta-Analysis

INTRODUCTION

In recent decades, escalating levels of air pollution have merged as a significant global health concern, particularly in densely populated and economically developing regions such as the Asia-Pacific states.[1] According to the State of Global Air 2020, unlike trends in other nations, Asian countries observed a consistent annual increase in the number of deaths linked to exposure to fine particulate matter (PM) with an aerodynamic diameter of less than 2.5 μm ($PM_{2.5}$) between 2010 and 2019.[2] This rising burden of air pollution, characterized by the accumulation of PM in the atmosphere, has been linked to adverse health outcomes, highlighting the critical need for comprehensive investigations into the long-term impacts on all-cause and cause-specific mortality.

Health impact assessments are crucial for understanding the relationship between air quality and public health. Previous studies have explored the methodological complexity of such assessments, using integrated models[3] and the Global Exposure Mortality Model (GEMM)[4] to establish a robust framework for analyzing the long-term consequences of PM exposure. However, current Global Burden of Disease (GBD) studies have mainly relied on data from North America and Europe, which may not directly apply to the Asia-Pacific states due to differences in exposure levels, pollutant compositions, and population characteristics. For example, in the case of GEMM, only 3 of the 41 cohort studies included cohorts from Asia (China, Hong Kong, and Taiwan). Moreover, Asian regions generally experience higher $PM_{2.5}$ concentrations compared to western countries, alongside notable differences in racial, geographical, lifestyle, and social determinants of health. Addressing the complexities of air pollution in Asia requires a nuanced understanding of these various factors. Different pollutants have distinct characteristics that can impact various health outcomes.[5] Therefore, our research aimed to enhance understanding of the associations between PM exposure and various causes of mortality in the Asia-Pacific states. By providing valuable insights, this research can guide the development of policies and interventions to mitigate the adverse health effects of air pollution in the region.

METHODS

We used the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 checklist for systematic review (**Supplementary Table 1**).

Search strategy

The study protocol was registered with the International Prospective Register of Systematic Reviews (PROSPERO) under the registered ID, CRD42023441916. Following the formulation of the Population, Exposure, Comparator, Outcome, and Study Design framework, specific eligibility criteria for study inclusion and exclusion were applied.

Population

This study included human populations exposed to $PM_{2.5}$ and PM with an aerodynamic diameter < 10 μm (PM_{10}) through ambient air in the Asia-Pacific states as defined by the United Nations (https://www.un.org/dgacm/en/content/regional-groups). This encompassed developed and developing areas, both urban and rural, with a focus on sub-groups at risk, such as children, the elderly, and patients with specific conditions. Populations exposed to $PM_{2.5}$ and PM_{10} in occupational or indoor settings were excluded.

Exposure

We included studies that analyzed the exposure to long-term (ranging from months to years) ambient $PM_{2.5}$ and PM_{10} expressed in concentration units of micrograms per cubic meter ($\mu g/m^3$). Additionally, studies reporting total suspended particulate (TSP) and suspended particulate matter (SPM) expressed in ($\mu g/m^3$) were also included.

Comparator

The comparison in this analysis was made between individuals exposed to lower ambient $PM_{2.5}$ and PM_{10} concentrations in the same or a control population.

Outcomes

Our study focused on health outcomes based on evidence suggesting causality from long-term exposure to $PM_{2.5}$ and PM_{10}.[6,7] The following cause-specific mortality outcomes were included: all-cause, cardiovascular diseases (CVD), ischemic heart diseases (IHD), stroke, acute lower respiratory infection (ALRI), chronic obstructive pulmonary diseases (COPD), lung cancer mortality, type 2 diabetes mellitus (T2DM) and chronic kidney disease (CKD). Non-accidental mortality was considered equivalent to all-cause mortality, as both definitions are used in studies. Detailed International Classification of Disease, 9th revision (ICD-9) and 10th revision (ICD-10) codes are described in **Supplementary Table 2**.

Study design

The study included epidemiological studies that employed major study designs, including retrospective and prospective cohorts, case-control, and nested case-control study designs.[8] Journal-published articles in any language with an abstract in English were included. Exclusion criteria comprised qualitative studies, studies lacking individual-level data (e.g., fully ecological outcomes), studies with no original data, reviews and methodological papers, and non-human studies. Additionally, conference papers, abstracts, letters, notes, and grey literature were excluded.

A systematic search of eligible studies was conducted comprehensively using the databases MEDLINE through PubMed (n = 8,326), Embase through Embase.com (n = 4,709), and Cochrane Library through Cochranelibrary.com (n = 357). The search was conducted between 1st January 1990 and 31st July 2023. Population, exposure, and study design were included in the search terms, while health outcome specifications were not included in the search strategy to broaden the search scope. Detailed search strategies for each database can be found in **Supplementary Table 3**. After excluding 2,267 duplicates, 6,059 articles remained for review.

Study selection and data collection

The *Rayyan* software (https://www.rayyan.ai/) was used to manage all the screened articles extracted from the search strategies. Initially, 2 researchers (JO and GHR) independently screened 6,059 articles based on title and abstract, excluding 5,978 irrelevant articles and leaving

81 articles. Subsequently, a comprehensive evaluation of the selected articles was conducted through full-text assessment. Disagreements in each step of the eligibility criteria were resolved through discussion with a third reviewer (YL). Finally, ten additional articles (pre-print: 1, conference abstract: 5, irrelevant studies: 3, and cross-sectional study: 1) were excluded.

The following characteristics were extracted from included studies: citation details (title, authors, date of publication), study design and study location (country/city), characteristics of the study population, follow-up period(s), mean or median age, percentage of males and females, details on exposure (pollutant, unit of measurement, concentration including 5th–95th or 25th–75th percentiles (whichever available) of population exposure, mean/median and min-max, exposure assessment/measurement methods, period of the year of exposure assessment), co-pollutants, outcome assessment, confounders adjusted for in the model, estimated associations and their confidence intervals (CIs), methods and results of the assessment of the shape of the exposure-response function, including association estimates for subsets of the data where the highest levels have been excluded from the analysis, and conflicts of interest.

In cases where a single publication reported multiple health outcomes of interest, each outcome was recorded. When multiple publications reported multiple effect estimates for a particular health outcome derived from a single pollutant model, data for the crude model, the most adjusted model, and the author's favored model were extracted. Additionally, 2 pollutant models were extracted if available.

Meta-analysis

The meta-analysis was used for three or more studies with the same pollutant ($PM_{2.5}$ or PM_{10}) and health outcome, considering both the general population and the elderly population. In cases where more than one study reported the same outcome variable within the same population, the most recently published outcome was included in the meta-analysis. For example, the study by Yang et al.[9] on the Hong Kong elderly cohort was included in the meta-analysis examining the associations between $PM_{2.5}$ and non-accidental mortality, while a similar study conducted by Wong et al.[10] was excluded.

Among the selected studies, we unified exposure units in $PM_{2.5}$ or PM_{10} using the converted values ($PM_{2.5}$ = 0.7 × SPM; PM_{10} = 0.55 × TSP).[11,12]

Considering several heterogeneities between studies (e.g., study design, covariates, exposure range), a pooled effect was estimated while incorporating a random effect assumption. The relative risk (RR) was estimated as a hazard ratio with 95% CI per 10 μg/m^3 increase in $PM_{2.5}$ or PM_{10} by back-transforming the hazard ratio and CI as follows:

$$\beta = \frac{\ln(RR_o)}{\text{increment}}$$

$$\text{Standard error (SE)} = \{\ln(RR_{o_{high}}) - \ln(RR_{o_{low}})\}/(2 \times 1.96 \times increment)$$

$$RR_c = \exp(\beta \times 10)$$

$$RR_{c_{low}} = \exp(\beta \times 10 - 1.96 \times SE \times 10)$$

$$RR_{c_{high}} = \exp(\beta \times 10 + 1.96 \times SE \times 10)$$

https://doi.org/10.3346/jkms.2025.40.e156

Here, the β and RR_c indicate the log RR and RR per unit change in each study, respectively. The RR_c indicates the RR per 10 μg/m^3 increase in $PM_{2.5}$ or PM_{10}. $RR_{c_{low}}$ and $RR_{c_{high}}$ indicate the lower and upper bounds of the 95% CI for the RR_c, respectively.

An 80% prediction interval (PI) was presented, considering the statistical heterogeneity of the effect estimates between studies and the uncertainty associated with small studies.[13,14] To evaluate the statistical heterogeneity between studies, the I^2 and Q-statistics of effects estimates were used.[15]

Risk of bias (RoB) assessment

A RoB assessment was conducted specifically for observational studies using the RoB assessment tool developed by a panel of experts assembled by the World Health Organization (WHO) (https://www.who.int/europe/publications/i/item/WHO-EURO-2020-5556-45321-64864). The tool consists of 6 domains: 1) confounding, 2) selection bias, 3) exposure assessment, 4) outcome measurement, 5) missing data, and 6) selective responding. Each domain encompasses subdomains, as detailed in **Supplementary Text 1**.

The RoB assessment was independently performed by 2 researchers (JO and GHR) and checked by the third reviewer (YL). Based on the WHO RoB assessment tool, the criteria for defining critical and additional confounders were followed, with "smoking" defined as a critical confounder instead.[8,16] For the assessment of each domain, the guidelines outlined in the WHO RoB assessment tool were followed: If any subdomain has a rating indicating a high risk of bias, the entire domain receives a 'high'; If all subdomains have ratings indicating a low risk of bias, the entire domain is rated as 'low'; If at least one subdomain has a moderate risk of bias rating and none have a high risk, the entire domain is rated as 'moderate.'

Subgroup analyses

Two subgroup analyses were conducted, one by sex (men vs. women) and the other by age group (excluding vs. limiting to the elderly). Due to the limited number of studies conducted on the elderly group, only $PM_{2.5}$ and all-cause mortality were considered for this subgroup analysis.

Sensitivity analyses

For PM and all-cause mortality, 3 sensitivity analyses were conducted, including studies in 1) infants and children, 2) patients with cause-specific diseases, and 3) Chinese men.

To assess the reliability and publication bias of each meta-analysis result, the funnel plot and Egger's test were performed. The funnel plot is commonly used to visually assess publication bias, while Egger's test statistically evaluates asymmetry in the distribution of effect sizes (i.e., RR) in systematic reviews.[17,18]

All data analyses and visualizations were conducted using R (version 4.3.1; R Foundation for Statistical Computing, Vienna, Austria). The "*dplyr*" and "*metafor*" packages in the R programming language were used for the analyses. Statistical significance was set at a *P* value of less than 0.05.

https://doi.org/10.3346/jkms.2025.40.e156

RESULTS

Summary of articles

A total of 71 articles of cohort studies were included in this systematic review (**Fig. 1**). Regarding the population type, 38 studies were conducted on the general population, ten on the elderly population, two on Chinese men, one for civil service employees and teachers, two for infants and one for children, and 17 for the patients-based population. Among 71 studies, 50.7% of studies (n = 36) were conducted in China, followed by South Korea (n = 13; 18.3%), Japan (n = 6; 8.5%), and Taiwan (n = 6; 8.5%). The mean $PM_{2.5}$ concentrations were the lowest in Japan (11.6 μg/m^3) and highest in China (80.6 μg/m^3) (**Table 1**).[9,10,12,19-86] For PM_{10}, studies from three countries (China, South Korea, and Japan) were included, with the mean PM_{10} concentrations being the lowest in Japan (17.2 μg/m^3) and the highest in China (144.34 μg/m^3). In the meta-analysis, 51 studies were included by excluding studies focusing on infants, children, and patients.

Meta-analyses

$PM_{2.5}$ and mortality

Of the 71 articles, 51 studies examined the associations between long-term exposure to $PM_{2.5}$ and all-cause or cause-specific mortality in general or elderly populations. Specifically, 19 studies on all-cause mortality, 15 on CVD, 11 on IHD, 12 on stroke, 5 on respiratory, 7 on COPD, 5 on ALRI, and 8 on lung cancer were used for meta-analysis for $PM_{2.5}$. However, no meta-analysis was performed for T2DM and CKD due to insufficient articles analyzing the associations with $PM_{2.5}$ and PM_{10} exposure.

For a 10 μg/m^3 increase in $PM_{2.5}$ concentrations, the pooled effects (i.e., RR) with 95% CI for all-cause mortality was 1.11 (95% CI, 1.05–1.17) (**Fig. 2**). Long-term exposure to $PM_{2.5}$ was positively associated with cause-specific mortality with RR of 1.13 (95% CI, 1.06–1.21) for

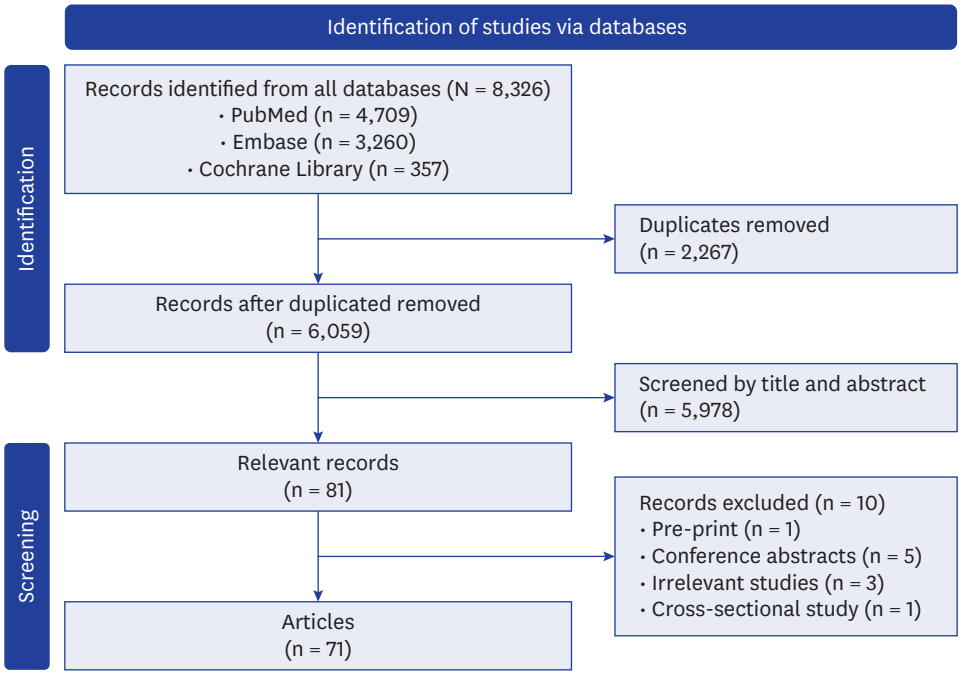


Fig. 1. The flow diagram of the study selection of this study.

https://doi.org/10.3346/jkms.2025.40.e156

Table 1. The characteristics of 71 studies examining the association between long-term ambient $PM_{2.5}$ exposure and cause-specific mortality

Author and year	Study/Data sources	Study period	Study design	Study location	Study population	Mean age	Age group	Study size			Exposure			Outcomes
								Total	Male	Female	Categories	Mean	Median	
Brown et al., 2022[33]	MDS	2004–2013	Cohort	India	General population	27.0[a]	15–69	678,889,985	51%	49%	$PM_{2.5}$		25.5	All-cause, non-accidental, stroke, IHD
Cao et al., 2011[26]	China National Hypertension Survey	1991–2000	Cohort	China	General population	55.8	15+	70,947	52%	48%	TSP	289		All-cause, CVD, respiratory, LC
Chen et al., 2019[34]	China National Stroke Registry Cohort	2007–2008	Cohort	China	Ischemic stroke patients	65.5	N/A	12,291	62%	38%	$PM_{2.5}$ PM_{10}	80.6 (deaths), 79.5 (survivals) 134.9 (deaths), 135.8 (survivals)		All-cause
Chen et al., 2023[19]	NHIRD	2000–2013	Cohort	Taiwan	Dialysis patients	58.0	18+	34,088	49%	51%	$PM_{2.5}$		31.19	All-cause
Chen et al., 2023[35]	Major Projects of Science Research for the 11th and 12th Five-Year Plans of China	2009–2020	Cohort	China	General population	48.3	N/A	580,757	47%	53%	$PM_{2.5}$ PM_{10}	37.34 62.05		Stroke
Chen et al., 2017[36]	Northern China	1999–2009	Cohort	China	General population	44.3	23+	39,054	50%	50%	PM_{10}	144.3		Respiratory, COPD
Chen et al., 2016[37]	Northern China	1998–2009	Cohort	China	General population	44.3	23+	39,054	50%	50%	PM_{10}	144.34		All-cause, LC
Chen et al., 2021[24]	RDCS	2006–2014	Cohort	China	General population	57.1	18+	28,793	44%	56%	$PM_{2.5}$	52.08	52.42	Non-accidental, CVD, respiratory
Choi et al., 2022[38]	KAMIR and KAMIR-NIH registry	2006–2015	Cohort	South Korea	Acute myocardial infarction patients	63.8	N/A	46,263	72%	28%	$PM_{2.5}$ PM_{10}	25.56 50.26		All-cause
Cui et al., 2023[39]	Third Central Clinical College of Tianjin Medical University	2013–2018	Cohort	China	Hepatocellular carcinoma patients	60.6	N/A	1,440	74%	26%	$PM_{2.5}$	58.56		All-cause
Dong et al., 2012[40]	Northern China	1998–2009	Cohort	China	General population	58.1	35+	9,941	49%	51%	PM_{10}	154		Respiratory
Eom et al., 2022[41]	CBRCR	2005–2019	Cohort	South Korea	Cancer patients	58.1	N/A	44,432	49%	51%	PM_{10}	49		All-cause, LC
Guo et al., 2022[42]	MJ Health Management Institution	2001–2019	Cohort	Taiwan	General population	39.4	18+	400,459	49%	51%	$PM_{2.5}$	26.4		All-cause, non-accidental, CVD, pneumonia
Guo et al., 2021[43]	MJ Health Management Institution	2001–2019	Cohort	Taiwan	General population	39.2	18+	384,130	49%	51%	$PM_{2.5}$	26.4		Non-accidental
Guo et al., 2022[44]	MJ Health Management Institution	2001–2019	Cohort	Taiwan	General population	N/A	18+	384,130	-	-	$PM_{2.5}$	26.3		Pneumonia
Hadley et al., 2022[45]	GCS	2004–2008	Cohort	Iran	General population	52.1	40–75	50,045 (derivation cohort: 45,052)	43%	57%	$PM_{2.5}$	33.5		All-cause, CVD

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Table 1. (Continued) The characteristics of 71 studies examining the association between long-term ambient $PM_{2.5}$ exposure and cause-specific mortality

Author and year	Study/Data sources	Study period	Study design	Study location	Study population	Mean age	Age group	Study size			Exposure			Outcomes
								Total	Male	Female	Categories	Mean	Median	
Hu et al., 2023[46]	Peritoneal dialysis cohort	2013–2018	Cohort	China	Dialysis patients	56.9	18+	886	55%	45%	$PM_{2.5}$ PM_{10}	72.52 116.67	58 101	All-cause
Huang et al., 2023[47]	Northern China	1998–2019	Cohort	China	General population	43.5	N/A	37,442	49%	51%	$PM_{2.5}$ PM_{10}	70.2 136.5		Non-accidental, CVD, LC
Hwang and Hu 2020[48]	Taiwan's National Health Insurance	2001–2016	Cohort	Taiwan	Elderly	68.0[a]	60–79	1.91 million	-	-	$PM_{2.5}$		32	All-cause
Jalali et al., 2021[49]	Isfahan Cohort Study	2001–2016	Cohort	Iran	General population	49.6	35+	3,081	48%	52%	$PM_{2.5}$	45.08		All-cause, CVD
Ji et al., 2022[50]	CLHLS	2008–2018	Cohort	China	Elderly	86.9	65+	11,835	43%	57%	$PM_{2.5}$	52.8		All-cause
Ji et al., 2020[51]	CLHLS	2008–2014	Cohort	China	Elderly	87.0	65–79	12,873	43%	57%	$PM_{2.5}$	49.63		All-cause
Jung et al., 2020[52]	CRC-ESRD Clinical Research Center for End-Stage Renal Disease	2008–2015	Cohort	South Korea	Dialysis patients	60.5	N/A	5,041	59%	41%	PM_{10}	50.38 to 55.22		All-cause
Jung et al., 2021[53]	Korean National Institute of Environmental Research and Seoul Research Institute of Public Health and Environment	2001–2015	Cohort	South Korea	CKD patients	60.5	N/A	29,602	48%	52%	$PM_{2.5}$ PM_{10}	24.84 (deaths), 24.37 (survivals) 47.55 (deaths), 46.30 (survivals)	24.86 (deaths), 24.34 (survivals) 46.44 (deaths), 45.55 (survivals)	All-cause
Kang et al., 2023[28]	NHID	2009–2013	Cohort	South Korea	COPD patients	58.0	40+	121,423	63%	37%	PM_{10}	49.9		All-cause, LC, pneumonia, COPD, CVD, stroke, DM
Katanoda et al., 2011[12]	Three-prefecture Cohort Study	1983–1995	Cohort	Japan	General population	N/A	40+	63,520	47%	53%	SPM $PM_{2.5}$			Respiratory, Pneumonia, COPD, LC
Kim et al., 2017[54]	NHIS-NSC	2007–2013	Cohort	South Korea	General population	57.0	18+	136,094	49%	51%	$PM_{2.5}$	25.03	21.92	All-cause, CVD
Kim et al., 2020[55]	Korean NHIS-NSC	2009–2013	Cohort	South Korea	General population	42.0	18+	436,933	50%	50%	$PM_{2.5}$	18.8		All-cause, CVD
Kim et al., 2017[56]	NHIS-NSC	2002–2013	Cohort	South Korea	General population	47.8	20–65	275,337	54%	46%	PM_{10}	56		Non-accidental, CVD, stroke, respiratory, LC
Lee et al., 2020[57]	NHIS-NSC	2009–2013	Cohort	South Korea	COPD patients	N/A	40+	6,313	54%	46%	PM_{10}	1-month: 46.4, 3-month: 39.5, 6-month: 41.2, 12-month: 48.2		Non-accidental
Li et al., 2023[58]	DHS conducted in 17 LMICs	2017–	Cohort	16 Asian and African LMICs (AA-LMICs)	Children	N/A	< 5	79,995	-	-	$PM_{2.5}$	50.85		Under-five death
Li et al., 2020[59]	China-PAR	1992–2015	Cohort	China	General population	51.0	18+	118,551	41%	59%	$PM_{2.5}$	65		LC

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Table 1. (Continued) The characteristics of 71 studies examining the association between long-term ambient $PM_{2.5}$ exposure and cause-specific mortality

Author and year	Study/Data sources	Study period	Study design	Study location	Study population	Mean age	Age group	Study size			Exposure			Outcomes
								Total	Male	Female	Categories	Mean	Median	
Li et al., 2018[60]	CLHLS	2008–2014	Cohort	China	Elderly	89.0	65+	13,344	42%	58%	$PM_{2.5}$		50.7	All-cause
Liang et al., 2020[61]	China-PAR	2000–2015	Sub cohorts	China	General population	51.2	18+	116,972	41%	59%	$PM_{2.5}$	67.4		CVD, IHD, Stroke
Liang et al., 2022[62]	CCDRFS	2010–2017	Cohort	China	General population	46.2	18+	90,672	43%	57%	$PM_{2.5}$	46.1	48.4	CVD, IHD, Stroke
Liao et al., 2022[63]	DHS/NFHS-4	2009–2016	Cohort	India	Mother-child pairs	N/A	< 5	252,788	52%	48%	$PM_{2.5}$	9-month in utero average: 71.1, post-delivery lifetime average: 73.7		All-cause
Lim et al., 2020[22]	NHID	2008–2016	Cohort	South Korea	Elderly	N/A	65+	1,720,230	43%	57%	$PM_{2.5}$		27.4	All-cause, CVD, IHD, Stroke, Respiratory, COPD, LC
Liu et al., 2022[64]	CFPS project	2010–2017	Cohort	China	General population	45.7	16–110	30,524	48%	52%	$PM_{2.5}$	47.1	43.8	All-cause
Liu et al., 2023[65]	133 hospitals across 11 cities in Hebei Province	2010–2019	Cohort	China	Lung cancer patients	66.9	N/A	6,429	66%	34%	$PM_{2.5}$		102.1	All-cause
Nakharutai et al., 2022[66]	Chiang Mai Cancer Registry	2003–2018	Cohort	Thailand	Lung cancer patients	64.0[a]	N/A	9,820	60%	40%	$PM_{2.5}$ PM_{10}	37.4 52.1		All-cause
Nishiwaki et al., 2013[67]	JPHC study	1990–2008	Cohort	Japan	General population	48.3	40–59	78,057	47%	53%	PM_{10}	17.2–43.7		IHD, Stroke, CVD, LC
Oh et al., 2023[68]	NHID	2008–2016	Cohort	South Korea	IHD patients	76.8	65+	306,418	43%	57%	$PM_{2.5}$	27.9		All-cause, CVD, Stroke, IHD
Peng et al., 2017[29]	Chinese tuberculosis cohort	2003–2013	Cohort	China	Tuberculosis diagnosed patients	N/A	N/A	4,444	74%	26%	$PM_{2.5}$	53.53		All-cause, tuberculosis, respiratory cancer, DM
Ran et al., 2020[31]	Elderly Health Service cohort	1998–2010	Cohort	Hong Kong	CKD patients	72.8	65+	902	42%	58%	$PM_{2.5}$	37.8	37.8	All-cause, CVD, IHD, stroke, respiratory, pneumonia, CKD
Ran et al., 2020[32]	Elderly Health Service Cohort	1998–2010	Cohort	China	Elderly	72.0	65+	61,447	34%	66%	$PM_{2.5}$		35.78	CKD
Sawada et al., 2022[25]	JPHC study	1990–2013	Cohort	Japan	General population	51.9	40–69	87,385	47%	53%	$PM_{2.5}$	11.6		All-cause, CVD, IHD, stroke, respiratory, pneumonia, COPD, LC
Shan et al., 2020[30]	Northern China	1998–2009	Cohort	China	General population	44.1	N/A	38,529	50%	50%	PM_{10}	143.36		DM
Shin et al., 2022[69]	NHIS-NSC	2005–2015	Cohort	South Korea	General population	46.6	N/A	87,608	54%	46%	$PM_{2.5}$ PM_{10}	26 48		LC
Son et al., 2011[70]	Korean National Statistical Office	2004–2007	Cohort	South Korea	Births	NA	< 1	359,459	51%	49%	TSP $PM_{2.5}$ PM_{10}	98 31.5 62.2		All-cause, respiratory
Takeuchi et al., 2021[71]	IPHS	1993–2010	Cohort	Japan	General population	58.8	40–79	91,808	34%	66%	SPM (less than 6.5–7 um)	32.7		CVD, IHD

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Table 1. (Continued) The characteristics of 71 studies examining the association between long-term ambient $PM_{2.5}$ exposure and cause-specific mortality

Author and year	Study/Data sources	Study period	Study design	Study location	Study population	Mean age	Age group	Study size			Exposure			Outcomes
								Total	Male	Female	Categories	Mean	Median	
Tseng et al., 2015[20]	Civil servants cohort	1989–2008	Cohort	Taiwan	Civil service employees and teachers	41.2	30+	43,227	57%	43%	$PM_{2.5}$		27.3–30.9 (20–80th)	All-cause, CVD, IHD, Stroke
Ueda et al., 2012[72]	NIPPON DATA	1980–2004	Cohort	Japan	General population	48.9	30+	7,250	43%	57%	PM		33.1–36.6	All-cause, CVD, IHD, Stroke
Wang et al., 2023[73]	Major Projects of Science Research - Guangzhou sub-cohort	2009–2020	Cohort	China	General population	48.3	18+	580,757	47%	53%	$PM_{2.5}$ PM_{10}	37.34 62.05		COPD
Wang et al., 2023[74]	Major Projects of Science Research - Guangzhou sub-cohort	2009–2015	Cohort	China	General population	48.3	18+	580,757	47%	53%	PM_1	19.56		COPD
Wang et al., 2023[23]	Major Projects of Science Research for the 11th (2006–2010) and 12th (2012–2017) five-year plans of China	2009–2020	Cohort	China	General population	48.3	18+	580,757	47%	53%	PM_1 $PM_{2.5}$ PM_{10}	2009 yr: 25.30, 2020 yr: 15.89 2009 yr: 50.09, 2020 yr: 23.97 2009 yr: 77.75, 2020 yr: 43.30		All-cause
Wong et al., 2015[10]	Hong Kong elderly	1998–2011	Cohort	Hong Kong	Elderly	72.0	65+	66,820	34%	66%	$PM_{2.5}$		35.3	Non-accidental, CVD, IHD, stroke, respiratory, oneumonia, COPD
Wong et al., 2016[75]	Hong Kong elderly	1998–2011	Cohort	Hong Kong	Elderly	N/A	65+	66,820	-	-	$PM_{2.5}$	33.7	33.2	LC
Xia et al., 2023[76]	PURE-China	2005–2021	Cohort	China	General population	50.6	35–75	42,160	42%	58%	$PM_{2.5}$	45.2		All-cause, CVD
Xu et al., 2021[77]	Seven hospitals in Beijing	2012–2016	Cohort	China	CVD patients	67.6	N/A	5,143	60%	40%	$PM_{2.5}$	78.85	81.2	All-cause, CVD, IHD
Yang et al., 2023[78]	BCR	2001–2018	Cohort	China	Lung cancer patients	69.2	N/A	129,856	62%	38%	$PM_{2.5}$	80.1		All-cause
Yang et al., 2020[79]	China-PAR	2000–2015	Cohort	China	General population	51.6	18+	116,821	41%	59%	$PM_{2.5}$	64.9		All-cause, CVD
Yang et al., 2021[80]	Northern China	2000–2009	Cohort	China	General population	44.0	N/A	38,140	50%	50%	$PM_{2.5}$	66.3		Stroke
Yang et al., 2018[9]	Hong Kong elderly	1998–2011	Cohort	Hong Kong	Elderly	70.2	65+	60,548	33%	67%	$PM_{2.5}$		42.2	Non-accidental, CVD, IHD, stroke, respiratory, pneumonia, COPD
Yin et al., 2017[21]	Chinese men	1990–2006	Cohort	China	Men	54.8	40+	189,793	100%	0%	$PM_{2.5}$	43.7		Non-accidental, CVD, IHD, stroke, COPD, LC

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Table 1. (Continued) The characteristics of 71 studies examining the association between long-term ambient $PM_{2.5}$ exposure and cause-specific mortality

Author and year	Study/Data sources	Study period	Study design	Study location	Study population	Mean age	Age group	Study size			Exposure			Outcomes
								Total	Male	Female	Categories	Mean	Median	
Yorifuji et al., 2019[81]	Basic Health Checkups Follow-up	2006–2016	Cohort	Japan	General population	69.9	40+	75,531	32%	68%	$PM_{2.5}$	14		Non-accidental, IHD, stroke, pneumonia, COPD, LC
Zhang et al., 2011[82]	Northern China	1998–2009	Cohort	China	General population	58.1	N/A	9,941	49%	51%	PM_{10}	154		All-cause, CVD, stroke
Zhang et al, 2014[83]	Northern China	1998–2009	Cohort	China	General population	44.3	N/A	39,054	50%	50%	PM_{10}	144		All-cause, CVD, IHD, stroke
Zhang et al., 2021[84]	CFPS project	2010–2018	Cohort	China	General population	45.7	16–110	30,946	49%	51%	$PM_{2.5}$	47.5		All-cause
Zhang et al., 2023[85]	Major Projects of Science Research for the 11th and 12th Five-year Plans of China	2009–2020	Cohort	South China	General population	48.3	N/A	580,757	47%	53%	$PM_{2.5}$ PM_{10}	37.3 62.1		CVD, IHD
Zhang et al., 2023[86]	CLHLS	2005–2018	Cohort	China	Elderly	87.1	65+	20,352	42%	58%	$PM_{2.5}$	65.5		All-cause
Zhou et al., 2014[27]	Chinese men	1990–2006	Cohort	China	Men	53.4	40+	71,431	100%	0%	PM_{10}	104		All-cause, CVD, respiratory, LC

IHD = ischemic heart disease, MDS = Million Death Study, $PM_{2.5}$ = particulate matter with an aerodynamic diameter < 2.5 μm, TSP = total suspended particulate, CVD = cardiovascular disease, LC = lung cancer, NA = not available, NHIRD = National Health Insurance Research Database, PM_{10} = particulate matter with an aerodynamic diameter <10 μm, COPD = chronic obstructive pulmonary disease, RDCS = Rural Deqing Cohort Study, KAMIR = Korea Acute Myocardial Infarction, NIH = National Institutes of Health, CBRCR = Chungbuk Regional Cancer Registry, GCS = Golestan Cohort Study, CLHLS = Chinese Longitudinal Healthy Longevity Survey, CRC-ESRD = Clinical Research Center for End-Stage Renal Disease, CKD = chronic kidney disease, NHID = National Health Insurance Database, DM = diabetes mellitus, NHIS-NSC = National Health Insurance Service-National Sample Cohort, DHS = Demographic and Health Surveys, NFHS-4 = National Family Health Survey 2015–2016, LMICs = low and middle income countries, China-PAR = Prediction for Atherosclerotic Cardiovascular Disease Risk in China, CCDRFS = China Chronic Disease and Risk Factors Surveillance, CFPS = China Family Panel Studies, JPHC = Japan Public Health Center-based Prospective Study, IPHS = Iberaki Prefectural Health Study, NIPPON DATA = National Integrated Project for Prospective Observation of Non-communicable Diseases and its Trends in the Aged, PURE-China = Prospective Urban and Rural Epidemiology Study conducted in China, BCR = Beijing Cancer Registry.
[a]Median age.

CVD, 1.13 (95% CI, 1.02–1.25) for IHD, 1.12 (95% CI, 1.02–1.24) for stroke, and 1.12 (95% CI, 1.08–1.16) for lung cancer (**Table 2**, **Supplementary Figs. 1-4**). No statistically significant associations were found for respiratory diseases (RR, 1.02; 95% CI, 0.93–1.12), COPD (RR, 1.19; 95% CI, 0.99–1.44) and ALRI (RR, 1.16; 95% CI, 0.99–1.36) (**Table 2**, **Supplementary Figs. 5-7**). The meta-analysis estimates for the association between long-term exposure to $PM_{2.5}$ and cause-specific mortality, excluding lung cancer (I^2 = 12.35%; **Supplementary Fig. 4**), showed high heterogeneity (I^2 > 60%; **Table 2**). Additionally, the predicted intervals of the pooled estimates of the associations between long-term exposure to $PM_{2.5}$ and lung cancer were statistically significant (RR, 1.12; 80% PI, 1.08–1.16).

PM_{10} and mortality

Among the general or elderly population studies (n = 51), 5 studies examined the associations of long-term exposure to PM_{10} with all-cause mortality: 6 for CVD, 3 for IHD, 4 for stroke, 4 for respiratory, and 5 for lung cancer. No meta-analysis was performed for COPD, ALRI, T2DM, and CKD due to insufficient articles analyzing their association with PM_{10} exposure.

Supplementary Table 4, **Supplementary Figs. 8** and **9** show that long-term exposure to PM_{10} was positively associated with all-cause mortality with RR of 1.12 (95% CI, 1.00–1.24) and IHD mortality with RR of 1.33 (95% CI, 1.28–1.38). However, no associations were found for CVD (RR, 1.05; 95% CI, 0.92–1.22), stroke (RR, 1.33; 95% CI, 1.28–1.38), respiratory disease

https://doi.org/10.3346/jkms.2025.40.e156

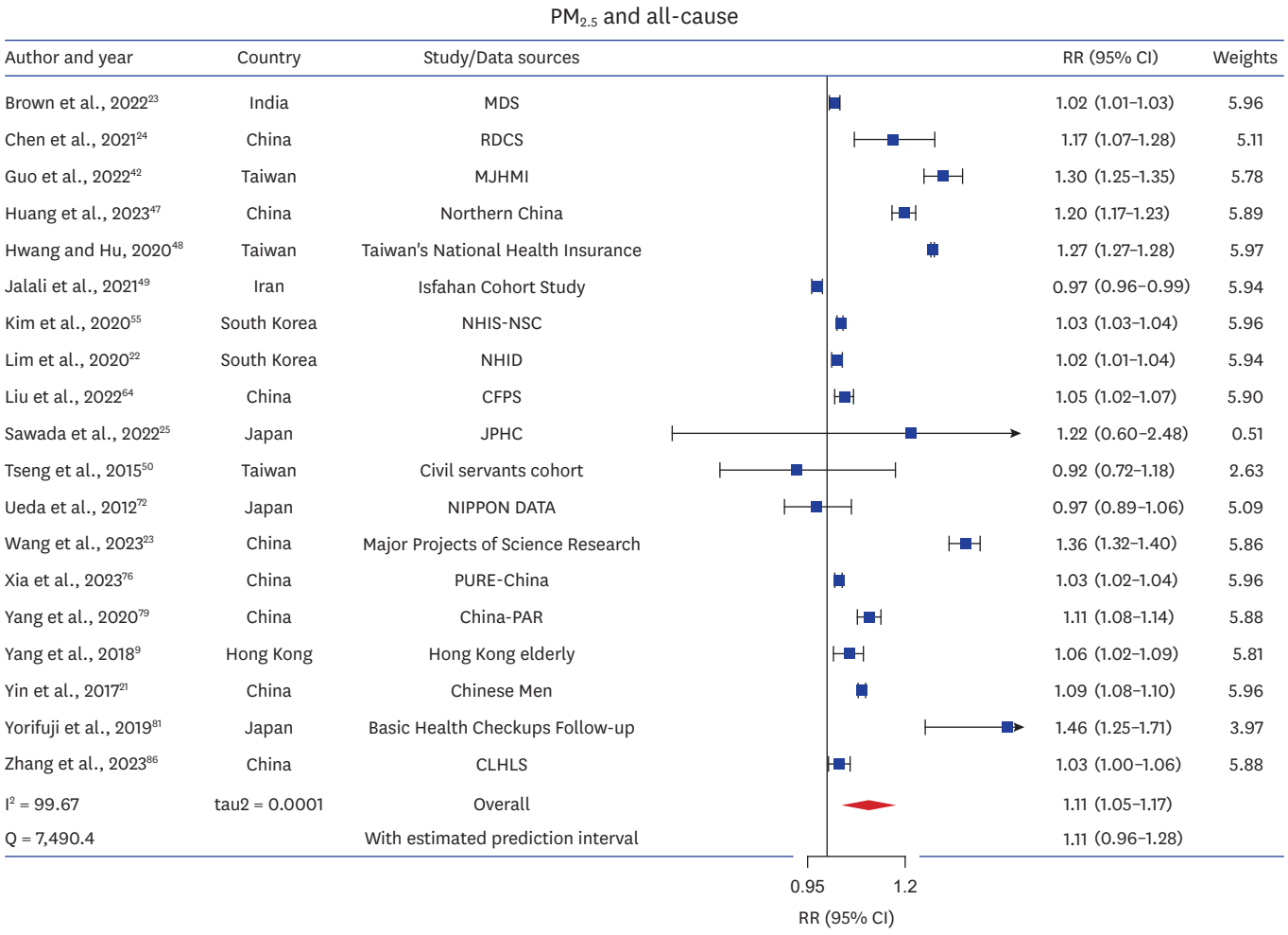


Fig. 2. Meta-analysis of the RR and 95% CIs of all-cause mortality per 10 μg/m³ increase in long-term $PM_{2.5}$ exposure combining the effects of 19 studies.
RR = relative risk, CI = confidence interval, $PM_{2.5}$ = particulate matter with an aerodynamic diameter < 2.5 μm, MDS = Million Death Study, RDCS = Rural Deqing Cohort Study, MJHMI = MJ Health Management Institution, NHIS-NSC = National Health Insurance Service-National Sample Cohort, NHID = National Health Insurance Database, CFPS = China Family Panel Studies, JPHC = Japan Public Health Center-based Prospective Study, NIPPON DATA = National Integrated Project for Prospective Observation of Non-communicable Diseases and its Trends in the Aged, PURE-China = Prospective Urban and Rural Epidemiology Study conducted in China, China-PAR = Prediction for Atherosclerotic Cardiovascular Disease Risk in China, CLHLS = Chinese Longitudinal Healthy Longevity Survey.

Table 2. Pooled association between long-term exposure to per 10 μg/m³ increase in long-term $PM_{2.5}$ and cause-specific mortality in Asia-Pacific states

Cause of death	$PM_{2.5}$				
	No.	RR	95% CI	80% PI	I^2
CVD	15	1.13	1.06–1.21	0.96–1.34	99.25%
IHD	11	1.13	1.02–1.25	0.93–1.37	95.67%
Stroke	12	1.12	1.02–1.24	0.92–1.36	97.18%
Respiratory	5	1.02	0.93–1.12	0.91–1.14	63.68%
COPD	7	1.19	0.99–1.44	0.89–1.60	92.74%
ALRI	5	1.16	0.99–1.36	0.95–1.42	76.07%
LC	8	1.12	1.08–1.16	1.08–1.16	12.35%

$PM_{2.5}$ = particulate matter with an aerodynamic diameter < 2.5 μm, RR = relative risk, CI = confidence interval, PI = prediction interval, CVD = cardiovascular disease, IHD = ischemic heart disease, COPD = chronic obstructive pulmonary disease, ALRI = acute lower respiratory infection, LC = lung cancer.

(RR, 1.10; 95% CI, 0.92–1.31), and lung cancer (RR, 1.04; 95% CI, 0.98–1.10) (**Supplementary Figs. 10-13**).

https://doi.org/10.3346/jkms.2025.40.e156

Risk of bias

A summary of the RoB for all exposure-outcome pairs (n = 109, including 82 and 27 pairs associated with $PM_{2.5}$ and PM_{10}, respectively) was presented in **Supplementary Tables 5** and **6**, **Fig. 3**. In the domain of confounding, most studies were assessed as having a moderate risk, followed by a high risk. For the exposure assessment domain, most studies were evaluated as having a moderate or low risk. In the domain of selection bias, most studies were assessed as having a low risk. The outcome measurement and selective responding domains were all indicated as having a low risk in all included studies. For missing data domains, only one study[19] was rated as a high risk, and the remaining studies were rated as a low risk.

Subgroup analyses

No differences were observed in the meta-analysis for long-term exposure to $PM_{2.5}$ and all-cause mortality according to sex (**Supplementary Table 7**, **Supplementary Figs. 14**-**20**). However, women showed slightly higher RR compared to men for cause-specific mortality of CVD, IHD, Stroke, and COPD. For lung cancer, men showed a statistically significant association (RR, 1.15; 95% CI, 1.03–1.29); however, women did not show a strong association (RR, 1.02; 95% CI, 0.89–1.17) (**Supplementary Table 7**, **Supplementary Fig. 20**).

For PM_{10}, CVD did not show different associations by sex. However, for IHD mortality, women showed a higher RR compared to men (**Supplementary Table 8**, **Supplementary Figs. 21** and **22**).

In addition, in **Supplementary Fig. 23**, the pooled effect of meta-analysis on the association between $PM_{2.5}$ and all-cause mortality excluding the elderly population was similar to the main findings (RR, 1.11; 95% CI, 1.04–1.19) (**Fig. 2**). However, the pooled effect of meta-analysis on the association between $PM_{2.5}$ and all-cause mortality in the elderly population was marginally significant (RR, 1.09; 95% CI, 0.99–1.21).

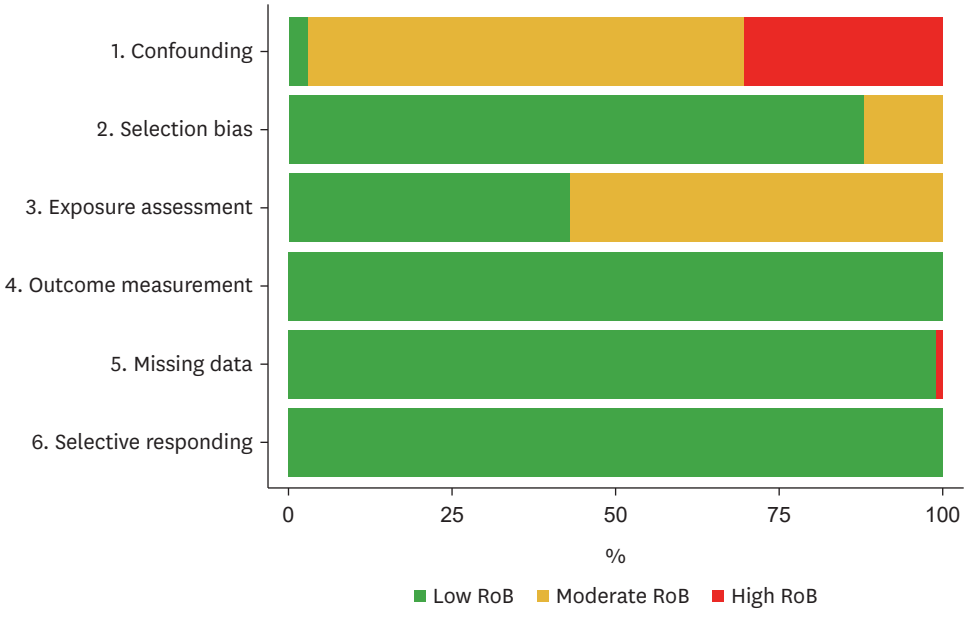


Fig. 3. Summary of RoB for all exposure-outcome pairs (n = 109) of meta-analysis.
RoB = risk of bias.

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Sensitivity analyses

For $PM_{2.5}$ and all-cause mortality, including infants and children studies, the pooled effect was similar to the main findings (RR, 1.11; 95% CI, 1.05–1.17) (**Supplementary Fig. 24**). However, the pooled effect of meta-analysis on the association between PM_{10} and all-cause mortality, including infants and children studies, was slightly higher (RR, 1.15; 95% CI, 1.02–1.29) (**Supplementary Fig. 25**).

For all-cause mortality, including patient-based studies, the pooled effects were higher than pooled effects excluding patient studies (RR of $PM_{2.5}$, 1.15; 95% CI, 1.09–1.22; RR of PM_{10}, 1.19; 95% CI, 0.97–1.46) (**Supplementary Figs. 26** and **27**).

For $PM_{2.5}$ and all-cause mortality excluding Chinese men study, the pooled effect was similar to the pooled effect including Chinese men study (RR, 1.11; 95% CI, 1.05–1.17) (**Supplementary Fig. 28**). However, the pooled effect of meta-analysis on the association between PM_{10} and all-cause mortality excluding Chinese men study was slightly higher (RR, 1.14; 95% CI, 1.01–1.29) (**Supplementary Fig. 29**) than pooled effects including Chinese men study (RR, 1.12; 95% CI, 1.00–1.24) (**Supplementary Fig. 8**).

The funnel plot and Egger's test indicate no publication bias for PM and cause-specific mortality (**Supplementary Figs. 30** and **31**).

DISCUSSION

This systematic review examined the association between long-term exposure to $PM_{2.5}$ and PM_{10} in ambient air and cause-specific mortality in the Asia-Pacific states. The review identified 71 relevant studies and included 51 studies focusing on the general or elderly population in the meta-analysis. Our findings suggest that increased ambient $PM_{2.5}$ exposure in the region is associated with higher risks of all-cause, CVD, IHD, stroke, and lung cancer mortality. Ambient PM_{10} exposure was associated with all-cause and IHD mortality.

Our $PM_{2.5}$ and all-cause and cause-specific mortality findings were similar to previous systematic review studies.[8,87] While Chen and Hoek[8] evaluated 25 cohort studies mostly conducted in North American and European countries and 3 studies in the Asia region[9,20,21] and estimated RR of 1.08 (95% CI, 1.06–1.09), which is slightly lower than our estimate in the Asian-Pacific states (RR, 1.11; 95% CI, 1.05–1.17), our findings for PM_{10} and all-cause mortality are also higher (RR, 1.12; 95% CI, 1.00–1.24) compared to the Chen and Hoek study (RR, 1.04; 95% CI, 1.03–1.06).[8] The recent global systematic review meta-analysis conducted by Orellano et al.[87] also demonstrated similar results (RR, 1.095; 95% CI, 1.064–1.127). Another systematic review evaluated 60 cohort studies on the association between long-term exposure to $PM_{2.5}$ and cause-specific mortality and disease incidence, including all-cause-, CVD, respiratory, renal disease, and cancer in Asia-Pacific region, including Australia, from 2000 to 2020.[88] The study reported higher heterogeneities among studies and did not conduct a meta-analysis. In contrast, we specifically focused on the Asia-Pacific states, excluding Australia and New Zealand, to reduce heterogeneity in the meta-analysis.

For CVD, IHD, and stroke mortality, we consistently found adverse associations with long-term exposure to $PM_{2.5}$, whereas PM_{10} was only positively associated with IHD. Similarly, Chen and Hoek[8] found a strong association between PM_{10} and IHD, but not with stroke. Due to the

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limited number of articles included in the meta-analysis with PM_{10} and IHD (n = 3), caution is needed to interpret the findings.

Regarding respiratory, COPD, and ALRI mortality, we observed a marginal association with an increase in $PM_{2.5}$, although it did not reach statistical significance. However, the Chen and Hoek[8] review found a significant association between long-term exposure to $PM_{2.5}$ and respiratory (RR, 1.10; 95% CI, 1.03–1.18), COPD (RR, 1.11; 95% CI, 1.05–1.17), and ALRI (RR, 1.16; 95% CI, 1.01–1.34) mortality. This discrepancy may be attributed to the weight of different studies focusing on respiratory-related outcomes in our meta-analysis, leading to higher heterogeneity when compared with other mortality outcomes. Researchers have suggested that the number of respiratory deaths might not have been sufficient to detect significant associations.[22] Furthermore, Lim et al.,[22] reported that the duration of exposure windows may have an important influence when analyzing different diseases. More substantial effects were found when using 36-month moving averages on COPD and lung cancer compared with shorter moving averages (12 to 24 months), which can be explained by the chronic nature of diseases with slow progression (cumulative effect over the years).[22] Additionally, differences in the composition of PM and levels of exposure concentration to which different populations were exposed might have played a role in the impact of air pollution on health outcomes.[23]

When classifying diseases related to respiratory conditions, we encountered a lack of consensus in the literature regarding which ICD-9 or ICD-10 codes to consider. For example, 4 of 5 studies used the ICD-9 or ICD-10 classifications (460–519 or J00–J99),[12,22,24,25] and one study used some of the ICD-10 codes for respiratory diseases (J00–J47 and J80–J99).[9] Moreover, some studies did not specify the ICD-10 codes used in their studies.[26,27] Nevertheless, we cannot rule out discrepancies in the diagnosis of diseases across countries.

We found a significant association between long-term exposure to $PM_{2.5}$ and lung cancer mortality, but not PM_{10}. Our findings were consistent with previous studies.[8,87,89,90] The pooled estimate per 10 $\mu g/m^3$ increase in long-term exposure to $PM_{2.5}$ in our study (RR, 1.12; 95% CI, 1.08–1.16) was slightly higher compared to those reported in the studies by Cui et al.[89] (RR, 1.05; 95% CI, 1.03–1.07) and Yang et al.[90] (RR, 1.07; 95% CI, 1.01–1.13), but similar to those reported in the Chen and Hoek[8] review (RR, 1.12; 95% CI, 1.07–1.16) and Orellano et al.,[87] review (RR, 1.09; 95% CI, 1.05–1.14).

Some studies have reported an association between exposure to $PM_{2.5}$ or PM_{10} and T2DM[28-30] and CKD.[31,32] However, we were not able to pool the estimates in a meta-analysis due to the small number of research results and large heterogeneity.

We observed heterogeneity of effect sizes in the meta-analyses, excluding the result derived from $PM_{2.5}$ and lung cancer. This discrepancy may stem from geographical location, exposure concentration, composition of PM, exposure measurement, exposure allocation, and potential confounders in each study. For all-cause mortality, we noted distinct associations with long-term exposure to $PM_{2.5}$ by sex and age group, consistent with previous studies (**Supplementary Figs. 32** and **33**).[8] Our review, which included data from Asia-Pacific states, incorporated a broader age range with a minimum age of 15 years and a range of mean or median ages spanning from 27 to 87.1 years (**Table 1**). In contrast, the review by Chen and Hoek[8] included studies with a minimum age of 25 years and a narrower range of mean or median ages, from 41 to 70.2 years. The effect sizes and their variability were notably larger in our analysis compared to studies from Western regions. This observation suggests greater

https://doi.org/10.3346/jkms.2025.40.e156

heterogeneity among the studies included in our systematic review compared to the previous systematic review.[8] This difference may provide a more comprehensive view but also indicate the need for caution in direct comparisons with Western estimates.

For CVD, IHD, stroke, and COPD, we found slightly higher effect sizes in women compared to men in the Asia-Pacific states. These results may be attributed to genetic, physiological, and behavioral differences between men and women regarding exposure to air pollution.[91,92]

The WHO RoB guidelines specify certain confounding factors that should be controlled for to ensure reliable results. These include critical potential confounders such as age, sex, and socioeconomic status indicators—like education level, employment status, individual income, mean income, and deprivation index—as well as body mass index. Additional recommended confounders are enrollment year, ethnicity, diet, physical activity, marital status, and smoking status. Although the WHO RoB guidelines classify smoking status as a recommended confounder, in our review, smoking was considered a critical confounder, consistent with prior review studies.[8] However, we recognize that it can be challenging for all studies to adequately account for every recommended confounder. In our review, some studies that did not control for critical potential confounders (e.g., socioeconomic status, body mass index, and smoking status) were assessed as having a high risk of bias, highlighting the importance of these variables in minimizing confounding and ensuring the validity of the findings.

The U.S. Environmental Protection Agency (EPA) has reported that long-term exposure to PM is associated with mortality, based on a compilation of research findings.[92] PM encompasses organic chemicals, metals, soil, acids, and dust, classified based on size, with smaller particles potentially penetrating deeper into the body than large particles. PM can impact all-cause mortality and cause-specific (CVD, respiratory, and lung cancer) mortality through direct mechanisms such as systemic inflammation, oxidative stress, epigenetics, DNA damage activation of coagulation pathways, and systemic circulation.[93,94]

We conducted a comprehensive study on the association between long-term exposure to PM and all-cause and cause-specific mortality reported in the Asia-Pacific states. Our findings suggest that the Asia-Pacific states with moderate to high PM concentration levels exhibit stronger associations between PM exposure and all-cause and cause-specific mortality compared to global estimates, which included an extensive number of countries with low PM levels.

However, our systematic review may have several limitations. Firstly, the inclusion/exclusion criteria may have inadvertently excluded certain articles, potentially limiting the comprehensiveness of our review. Additionally, we only included articles with abstracts in English, which may have excluded relevant studies published in other languages. Secondly, our literature search targeted 55 countries corresponding to the Asia-Pacific states. While these countries exhibit a diverse range of exposure levels and lifestyle patterns (e.g., diet and smoking), our included studies were mainly conducted in a few states, including China, Japan, India, Iran, South Korea, and Thailand (alphabetically ordered). This may introduce a bias when applying findings to the Asia-Pacific states. Lastly, global studies reported a supra-linear (i.e., non-linear) relationship characterized by a steep increase at low concentrations and a diminishing association at high concentrations. However, our evidence was derived assuming a linear relationship, which may not fully capture the complexities of the relationship between PM exposure and mortality.[4,95,96]

Our study reveals significant positive associations between long-term exposure to $PM_{2.5}$ and all-cause and cause-specific mortality, including CVD, IHD, stroke, and lung cancer, in the Asia-Pacific states. Additionally, long-term exposure to PM_{10} shows positive associations with all-cause and IHD mortality in the same region. These findings contribute to a deeper understanding of the health impacts of $PM_{2.5}$ and PM_{10} in the Asia-Pacific states, where air pollution levels are generally higher than those in North America and European countries.

SUPPLEMENTARY MATERIALS

Supplementary Text 1
Each subdomain of the 6 domains for risk of bias assessment

Supplementary Table 1
PRISMA 2020 checklist

Supplementary Table 2
ICD-10 and ICD-9 codes were reported by outcome in selected articles

Supplementary Table 3
Search database and search query for the systematic review

Supplementary Table 4
Pooled associations between long-term exposure to per 10 μg/m³ increase in long-term PM_{10} and cause-specific mortality in Asia-Pacific states

Supplementary Table 5
Risk of bias for included articles in meta-analysis between long-term exposure to $PM_{2.5}$ and cause-specific mortality

Supplementary Table 6
Risk of bias for included articles in meta-analysis between long-term exposure to PM_{10} and cause-specific mortality

Supplementary Table 7
Pooled associations between long-term exposure to per 10 μg/m³ increase in long-term $PM_{2.5}$ and cause-specific mortality stratified by sex in Asia-Pacific states

Supplementary Table 8
Pooled associations between long-term exposure to per 10 μg/m³ increase in long-term PM_{10} and cause-specific mortality stratified by sex in Asia-Pacific states

Supplementary Fig. 1
Meta-analysis of the RR and 95% CIs of CVD per 10 μg/m³ increase in long-term $PM_{2.5}$ exposure combining the effects of 15 studies.

Supplementary Fig. 2
Meta-analysis of the RR and 95% CIs of IHD per 10 μg/m³ increase in long-term $PM_{2.5}$ exposure combining the effects of 11 studies.

https://doi.org/10.3346/jkms.2025.40.e156

Supplementary Fig. 3
Meta-analysis of the RR and 95% CIs of stroke per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects of 12 studies.

Supplementary Fig. 4
Meta-analysis of the RR and 95% CIs of lung cancer per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects of 8 studies.

Supplementary Fig. 5
Meta-analysis of the RR and 95% CIs of respiratory disease per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects of 5 studies.

Supplementary Fig. 6
Meta-analysis of the RR and 95% CIs of COPD per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects of 7 studies.

Supplementary Fig. 7
Meta-analysis of the RR and 95% CIs of ALRI per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects of 5 studies.

Supplementary Fig. 8
Meta-analysis of the RR and 95% CIs of all-cause mortality per 10 μg/m^3 increase in long-term PM_{10} exposure combining the effects of 5 studies.

Supplementary Fig. 9
Meta-analysis of the RR and 95% CIs of IHD per 10 μg/m^3 increase in long-term PM_{10} exposure combining the effects of 3 studies.

Supplementary Fig. 10
Meta-analysis of the RR and 95% CIs of CVD per 10 μg/m^3 increase in long-term PM_{10} exposure combining the effects of 6 studies.

Supplementary Fig. 11
Meta-analysis of the RR and 95% CIs of stroke per 10 μg/m^3 increase in long-term PM_{10} exposure combining the effects of 4 studies.

Supplementary Fig. 12
Meta-analysis of the RR and 95% CIs of respiratory disease per 10 μg/m^3 increase in long-term PM_{10} exposure combining the effects of 4 studies.

Supplementary Fig. 13
Meta-analysis of the RR and 95% CIs of lung cancer per 10 μg/m^3 increase in long-term PM_{10} exposure combining the effects of 5 studies.

Supplementary Fig. 14
Meta-analysis of the RR and 95% CIs of all-cause mortality per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects stratified by sex.

https://doi.org/10.3346/jkms.2025.40.e156

Supplementary Fig. 15
Meta-analysis of the RR and 95% CIs of CVD per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects stratified by sex.

Supplementary Fig. 16
Meta-analysis of the RR and 95% CIs of IHD per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects stratified by sex.

Supplementary Fig. 17
Meta-analysis of the RR and 95% CIs of stroke per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects stratified by sex.

Supplementary Fig. 18
Meta-analysis of the RR and 95% CIs of respiratory per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects stratified by sex.

Supplementary Fig. 19
Meta-analysis of the RR and 95% CIs of COPD per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects stratified by sex.

Supplementary Fig. 20
Meta-analysis of the RR and 95% CIs of lung cancer per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects stratified by sex.

Supplementary Fig. 21
Meta-analysis of the RR and 95% CIs of CVD per 10 μg/m^3 increase in long-term PM_{10} exposure combining the effects stratified by sex.

Supplementary Fig. 22
Meta-analysis of the RR and 95% CIs of IHD per 10 μg/m^3 increase in long-term PM_{10} exposure combining the effects stratified by sex.

Supplementary Fig. 23
Meta-analysis of the RR and 95% CIs of all-cause mortality per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects stratified by age group.

Supplementary Fig. 24
Meta-analysis of the RR and 95% CIs of all-cause mortality per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects including infants and children studies.

Supplementary Fig. 25
Meta-analysis of the RR and 95% CIs of all-cause mortality per 10 μg/m^3 increase in long-term PM_{10} exposure combining the effects including infants and children studies.

Supplementary Fig. 26
Meta-analysis of the RR and 95% CIs of all-cause mortality per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects including cause-specific patient studies.

https://doi.org/10.3346/jkms.2025.40.e156

Supplementary Fig. 27
Meta-analysis of the RR and 95% CIs of all-cause mortality per 10 μg/m^3 increase in long-term PM_{10} exposure combining the effects including cause-specific patient studies.

Supplementary Fig. 28
Meta-analysis of the RR and 95% CIs of all-cause mortality per 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure combining the effects excluding Chinese men study.

Supplementary Fig. 29
Meta-analysis of the RR and 95% CIs of all-cause mortality per 10 μg/m^3 increase in long-term PM_{10} exposure combining the effects excluding Chinese men study.

Supplementary Fig. 30
Funnel plot for meta-analysis on the associations between $PM_{2.5}$ and cause-specific mortality.

Supplementary Fig. 31
Funnel plot for meta-analysis on the associations between PM_{10} and cause-specific mortality.

Supplementary Fig. 32
Age-specific comparison of effect sizes for the associations between long-term exposure to $PM_{2.5}$ and all-cause mortality per 10 μg/m^3 increase.

Supplementary Fig. 33
Age- and sex-specific comparison of effect sizes for the associations between long-term exposure to $PM_{2.5}$ and all-cause mortality per 10 μg/m^3 increase.

Supplementary reference

Supplementary Material 1
Data sharing statement

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