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The odds of developing asthma and wheeze among children and adolescents exposed to particulate matter: a systematic review and meta-analysis

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Abstract

Background Exposure to air pollution specifically particulate matter causes significant health risk to children which increases their susceptibility to respiratory diseases.

Objectives This review aimed to pool the association between particulate matter exposure and childhood asthma and wheeze among children and adolescents.

Methods This review included observational study articles retrieved from electronic data bases such as PubMed, Google Scholar, Hinari, Science Direct, and Semantic Scholar from 1996 to June 17, 2024. Data were extracted and analyzed using Microsoft Excel 16 and STATA version 17, respectively. Joanna Briggs Institute evaluation criteria and I^2 test statistics were used for quality and heterogeneity assessment, respectively.

Results Fourty seven studies with a total of 417,874 of children and adolescents met the inclusion criteria. The pooled odd ratio (OR) of the association between Particulate Matter with a diameter of 10 micrometers or less (PM10) and Particulate Matter with a diameter of 2.5 micrometers or less (PM2.5) with asthma were 1.04 (95% CI: 1.03–1.06, $p < 0.001$) with significant extreme heterogeneity ($I^2 = 82.7\%$, $p < 0.001$) and 1.05 (95% CI 1.04–1.07, $p < 0.001$) with high heterogeneity ($I^2 = 80.6\%$, $p < 0.001$) among the included studies, respectively. The overall pooled estimate indicates a statistically significant association between PM10 and wheeze, with OR of 1.06 (95% CI: 1.05, 1.07) and moderate heterogeneity among included studies (I^2=57.5%, $p < 0.007$) where as more association was observed between PM2.5 and wheeze with OR of 1.15. (95% CI: 1.10, 1.20) with an (I^2 =72.8%, $p < 0.001$).

Conclusion The findings of this systematic review and meta-analysis demonstrated a statistically significant association between exposure to both PM10 and PM2.5 and the occurrence of asthma and wheezing in children and adolescents. Both PM10 and PM2.5 are associated with increased odds of asthma and wheezing, with PM2.5 showing a stronger relationship. The significant levels of heterogeneity observed suggest variations across studies, which may

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be due to differences in study designs, exposure level and outcome measurement types. These findings indicate the need for strategies to reduce particle air pollution to mitigate its adverse effects on children's respiratory health.

Keywords Children, Adolescent, Asthma, Wheezing, Particulate matter, Air pollution, Systematic review and Meta-analysis

Introduction

Globally, air pollution, particularly exposure to particulate matter, is the most serious public health concern. Particulate matter(PM), which is made up of small solid or liquid particles suspended in the atmosphere, is the leading cause of air pollution. Particulate matteris divided into two categories: PM2.5 (particles with an aerodynamic diameter of less than 2.5 μm) and PM10 (particles with an aerodynamic diameter of less than 2.5 μm) [1, 2]. Since their size is very small, they can penetrate deeply into the lungs and bloodstream, causing adverse health effects [3].

Previous epidemiological studies have indicated a strong association between PM exposure and the development of respiratory diseases, such as asthma and wheezing among children and adolescents [1]. A study reported in 2015 shows that PM2.5 was ranked as the fifth-highest mortality risk factor that significantly increased the worldwide burden of disease [2]. WHO and EPA recognize the noteworthy contribution of PM to air pollution, its detrimental impact on respiratory well-being, and the aggravation of asthma and wheeze in this population [3, 4].

Asthma is the most prevalent inflammatory disease of the airways, characterized by recurring episodes of wheezing, shortness of breath, chest tightness, and coughing. Exposure to air pollutants, particularly PM, has been identified as a major environmental factor that contributes to the onset and worsening of asthma and wheezing among children and adolescents [5]. PM influences respiratory health through different mechanisms, such as exacerbating airway inflammation, oxidative stress, and altered immune responses, potentially leading to the onset and exacerbation of asthma [6].

Children and adolescents are more prone to asthma from PM exposure [7] because of greater breathing rates, lesser nasal particle filtration efficiency, growing immune systems, longer outdoor exposure, narrower airways, and immature lung tissue, making them more susceptible to allergens and irritants [8, 9]. Numerous studies around the globe confirmed that both long-term and short-term exposure to PM cause an increased risk of asthma [10–15].

Wheezing and asthma are two of the most prevalent respiratory conditions impacting children and adolescents globally, significantly impairing their health and overall quality of life [16]. Asthma has become a leading chronic allergic respiratory disease that affects children globally and has caused significant health challenges in recent years [17]. Asthma puts the greatest burden on children, rapid increases in global morbidity, mortality and reduces productivity including the school's absenteeism. Their growing vulnerability to particulate matter pollution and other triggers exacerbates asthma and wheeze affecting long-term health and development [18]. The increasing burden of asthma among children populations is becoming great concern, with a notable increase in magnitude.

The 2015 global burden of disease study estimates that 358.2 million people worldwide suffer from asthma, 12.6% rise from 1990 [19]. According to the estimates of the global burden of ambient PM 2.5 and asthma in 2015, around 50 million children worldwide were believed to have asthma [20]. This trend has been attributed to various environmental, genetic, and lifestyle factors, among which exposure to PM [21] has gained significant attention [15]. Existing body of scientific evidence suggests that children and adolescents who had early life exposure (before and within the first year of birth) to air pollution due to PM2.5 have a high risk of asthma [22, 23]. The level of PM (PM10, PM2.5) which usually exceed the WHO safety threshold, were associated with asthma [24, 25]. Asthmatic symptoms due to PM exposure are documented in 14% of children worldwide [26]. Mothers with particulate exposure during their pregnancy are associated with an increased risk of children developing respiratory illnesses such as asthma and wheezing. This relationship has been mostly observed in industrialized nations [27–29]. Asthmatic symptoms due to PM exposure are documented in 14% of children worldwide [26]. Other study indicated that the overall prevalence of childhood asthma increased from 8.7% in 2001 to 9.7% in 2009 [30].

Despite numerous individual studies that have investigated the association between PM exposure and asthma/wheeze among children and adolescents, the reported findings have been inconsistent with varying effect sizes and statistical significances. This inconsistency needs a comprehensive synthesis of the existing literature to clarify the extent of pooled association. Therefore, this systematic review and meta-analysis aims to provide a robust and up-to-date assessment of the odds of developing asthma and wheeze among children and adolescents exposed to different PM size fractions (PM2.5 and PM10) [31, 32].

Table 1 Searching strategies used for each types of data base for observational studies with english Language,2024

S/*N*	Database types	Searching strategies for each types of data base	Number of studies
1	PubMed	((((((((((((((((((((((asthma [Title/Abstract]) OR (wheeze [Title/Abstract])) OR (wheezing [Title/Abstract])) AND (children [Title/Abstract])) OR (adolescent [Title/Abstract])) OR (youth [Title/Abstract])) OR (teenager [Title/Abstract])) OR (pediatric [Title/Abstract])) AND ("particulate matter"[Title/Abstract])) OR (PM[Title/Abstract])) OR (PM10[Title/Abstract])) OR (PM2.5[Title/Abstract])) OR ("air pollution"[Title/Abstract])) OR ("airborne particle"[Title/Abstract])) AND (odd ratio[Title/Abstract])) OR (odds[Title/Abstract])) OR (association[Title/Abstract])) OR (relationship[Title/Abstract])) AND (exposure[Title/Abstract])) OR ("environmental exposure"[Title/Abstract])) OR ("indoor air exposure"[Title/Abstract])) OR ("ambient air exposure"[Title/Abstract])) OR ("air quality"[Title/Abstract])	3789
2	Hinari	((Abstract: (asthma)) OR (Abstract: (wheeze)) OR (Abstract: (wheezing))) AND ((Abstract: (children)) OR (Abstract: (adolescent)) OR (Abstract: (youth)) OR (Abstract: (teenager)) OR (Abstract: (pediatric))) AND ((Abstract: ("particulate matter")) OR (Abstract: (PM)) OR (Abstract: (PM10)) OR (Abstract: (PM2.5)) OR (Abstract: ("air pollution"[)) OR (Abstract: ("airborne particle"[))) AND ((Abstract: (odd ratio)) OR (Abstract: (odds)) OR (Abstract: (association)) OR (Abstract: (relationship)) OR (exposure) OR ("environmental exposure"))	1843
3		**((**Abstract: (asthma)) OR (Abstract: (wheeze)) OR (Abstract: (wheezing))) AND ((Abstract: (children)) OR (Abstract: (adolescent)) OR (Abstract: (youth)) OR (Abstract: (teenager)) OR (Abstract: (pediatric))) AND ((Abstract: ("particulate matter")) OR (Abstract: (PM)) OR (Abstract: (PM10)) OR (Abstract: (PM2.5)) OR (Abstract: ("air pollution"[)) OR (Abstract: ("airborne particle"[))) AND ((Abstract: (odd ratio)) OR (Abstract: (odds)) OR (Abstract: (association)) OR (Abstract: (relationship)) OR (exposure) OR ("environmental exposure"))	265
4	**Science Direct**	**(**Asthma OR Wheeze) AND [37] AND (Particulate Matter OR PM2.5 OR Air Pollution) AND (Odds Ratio OR Association OR Exposure)	7,703
5	**Semantic Scholar**	(Asthma OR Wheeze) AND [37] OR (Adolescents) OR (Youth) OR (teenager) OR (pediatric) AND (Particulate Matter OR PM2.5 OR Air Pollution) AND (Odds Ratio OR Association OR Exposure)	255

Methods

Reporting system and registration

We conducted a systematic review focusing on primary studies that investigated the relationship between PM2.5 and/or PM10 exposure and asthma and/or wheezing in children from prenatal exposure to 18 years across the globe. Our review adhered to the core principles outlined in the Centre for Reviews and Dissemination's (CRD) guidance for healthcare reviews and followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The review protocol was registered with the International Prospective Register of Systematic Reviews (PROSPERO: CRD42024562670), which can be accessed at https://www.crd.york.ac.uk/PROSPERO/.

Data sources, searching strategies, and study selection

A thorough literature search was performed across multiple electronic databases, including PubMed, Google Scholar, Hinari, Semantic Scholar, and Science Direct. We included studies published from the inception of these databases in 1976 up to June 17, 2024, by seven authors independently (AK, ETA, CD, YM, AE, YT, and AEB). Studies from previous systematic reviews were reassessed and incorporated into this meta-analysis. Additionally, we reached out to experts in the field to gather more information on both published and unpublished research. To ensure a comprehensive search, we also meticulously reviewed the references in selected studies to identify any related studies that may have been missed. The MeSH and search filters were included in the search strategies (Table 1).

Beyond the primary keywords, we employed synonyms, abbreviated symbols, and additional free-text keywords to enhance the search. Only full-text articles published in English were included in the review. It was made due to resource constraints in translation and to ensure consistency in data extraction and interpretation. All included and excluded studies were screened using EndNote 20 and the Rayyan automation tool. Manual verification was performed to mitigate the risk due to algorism error, but the potential for oversight remains a limitation in the study selection process. The screening process began with an independent review of titles and abstracts, followed by a full-text screening of the selected studies by three authors. Any disagreements were resolved through consensus. The selection process was meticulously documented to enable the completion of a PRISMA 2020 flow diagram.

Inclusion and exclusion criteria

This review focused on cohort, case-control, and cross-sectional studies that examined the relationship between PM exposure and asthma or wheezing in children under

18 years of age including prenatal exposure. We included studies without restrictions on the study period, sample size, study setting, or publication status, encompassing both published and unpublished research. Observational studies assessing the association of PM2.5 and PM10 exposure and asthma or wheeze on children were eligible, with comparisons made between exposed and less or non-exposed children. Studies involving children exposed to PM from indoor exposure, and ambient exposure were included. However, studies that involved populations other than children were excluded. The primary outcomes considered were adjusted odd ratio reported on the association between PM exposure and asthma or wheezing in children and adolescents. We excluded qualitative studies, studies that could not be retrieved, editorial letters, studies with poor methodological quality based on JBI (Joanna Briggs Institute) criteria, and studies did not report relevant outcomes from the meta-analysis.

Exposure assessment

The included studies assessed PM pollution exposure using two main methods including land-use regression (LUR) models and ground-based monitoring data with cumulative PM exposure estimates. LUR models used geographic, meteorological, and traffic variables for spatially resolved data, while ground-based monitors provided high temporal resolution but limited spatial coverage. Cumulative PM exposure captured multi-day effects to account for delayed health impacts. This study used cumulative effects for studies reporting lagged effects, as this approach provides a more comprehensive measure of exposure.

Outcome assessment

This review included questionnaire assessments and/or physician diagnoses of asthma and wheeze [33] to estimate the association between PM2.5 and PM10 exposure, expressed as an adjusted odd ratio. Due to variability in methods, effect estimates were retained in their original form.

Data extraction and quality assessment

After all articles were exported into EndNote 20 and the Rayyan automation tool, duplicate entries were removed. The remaining data were extracted using a standardized form, which was initially piloted on two included studies. This form captured study characteristics, outcomes, and risk of bias and was implemented in Microsoft Excel 2016. All authors (AK, ETA, CD, AE, YT, YM, and AEB) were responsible for extracting data from cohort, case-control, and cross-sectional studies. The recording details includes name of authorship, publication year, country, study design, sample size, type of PM exposure, exposure metrics, exposure assessment methods, years of outcome measurement, outcome assessment method, and the association of PM exposure with asthma [34] and wheezing among children and adolescents.

Following the screening of relevant articles for eligibility by the three reviewers, the quality of each study was assessed using the Joanna Briggs Institute (JBI) critical appraisal checklist. Each reviewer independently evaluated the risk of bias for the studies, with the results expressed on a 100% scale. Articles with a quality score above 50% were included in the subsequent qualitative and quantitative analyses. In cases where discrepancies arose during the quality assessment, the mean score from all reviewers was calculated to resolve any differences.

Data synthesis and analysis

To estimate the pooled effect sizes for the association between PM exposure and the occurrence of asthma and wheezing, we employed random effects models after excluding studies with risk of bias from the review based on their JBI scores. An outlier analysis was conducted to identify studies with extreme effect sizes that could potentially skew the overall conclusions. Additionally, a series of sensitivity analyses were performed to assess the validity and robustness of the summary measures and none of a single study did not unduly influence overall findings.

Subgroup analyses were conducted to explore potential sources of heterogeneity, focusing on factors such as regions, study design, exposure assessment methods, outcome measurement methods and exposure level for association between PM and asthma. The heterogeneity of the included studies was assessed using I^2 statistic, where values below 50%, 51–75%, 76–85% and 86 – 00% represented low, moderate, high and extreme heterogeneity respectively [35].

We considered a 95% confidence interval (CI) and a p-value of less than 0.05 as statistically significant for associations, presence of publication bias, and heterogeneity. For the summary measure, we utilized a random effects model, assuming that the included studies represent a random sample of all possible results using Der Simonian-Laird estimator. All analyses were performed using Stata version 17 (Stata Corp LLC, College Station, TX, USA) and "metan" package from Stata (https://www.stata.com).

Result

Included studies and baseline characteristics

Based on the search study stated above, 13,855 studies from databases, 43 from websites, and 36 from citations were identified. A total of 502 studies from the database were discarded due to duplication. About 218 discarded studies were excluded via EndNote 20, and the remaining

5972 studies were excluded using the Rayyan automation tool. Title and abstract parts of the remaining 7665 studies were reviewed, of which 7245 studies were excluded due to irrelevance. Out of the 420 studies that were sought to be retrieved, 92 could not be retrieved, and 328 were eligible for full-text screening. Finally, 14 studies from the new database, 5 studies from the website and citation, and 28 articles screened and reevaluated from previous studies (Fig. 1).

A total of 47 articles [21, 36–81] were included in this systematic review and meta-analysis of which 31 were used to determine the association between exposure to PM10, and asthma whereas 29 articles were used for association between PM2.5 and asthma (Table 2) whereas 18 studies were used to determine association between exposure to PM10, PM2.5 and wheeze (Table 3) among children and adolescents. In this meta-analysis, a total 417,874 of children as study subjects were included. In this meta-analysis, 6 studies were carried out in United states of America [36, 57, 62, 68, 73, 80], ten studies from China [44–47, 55, 58–60, 79, 81], five studies from Taiwan [42, 43, 54, 76, 78], four from France [37, 56, 71, 72],

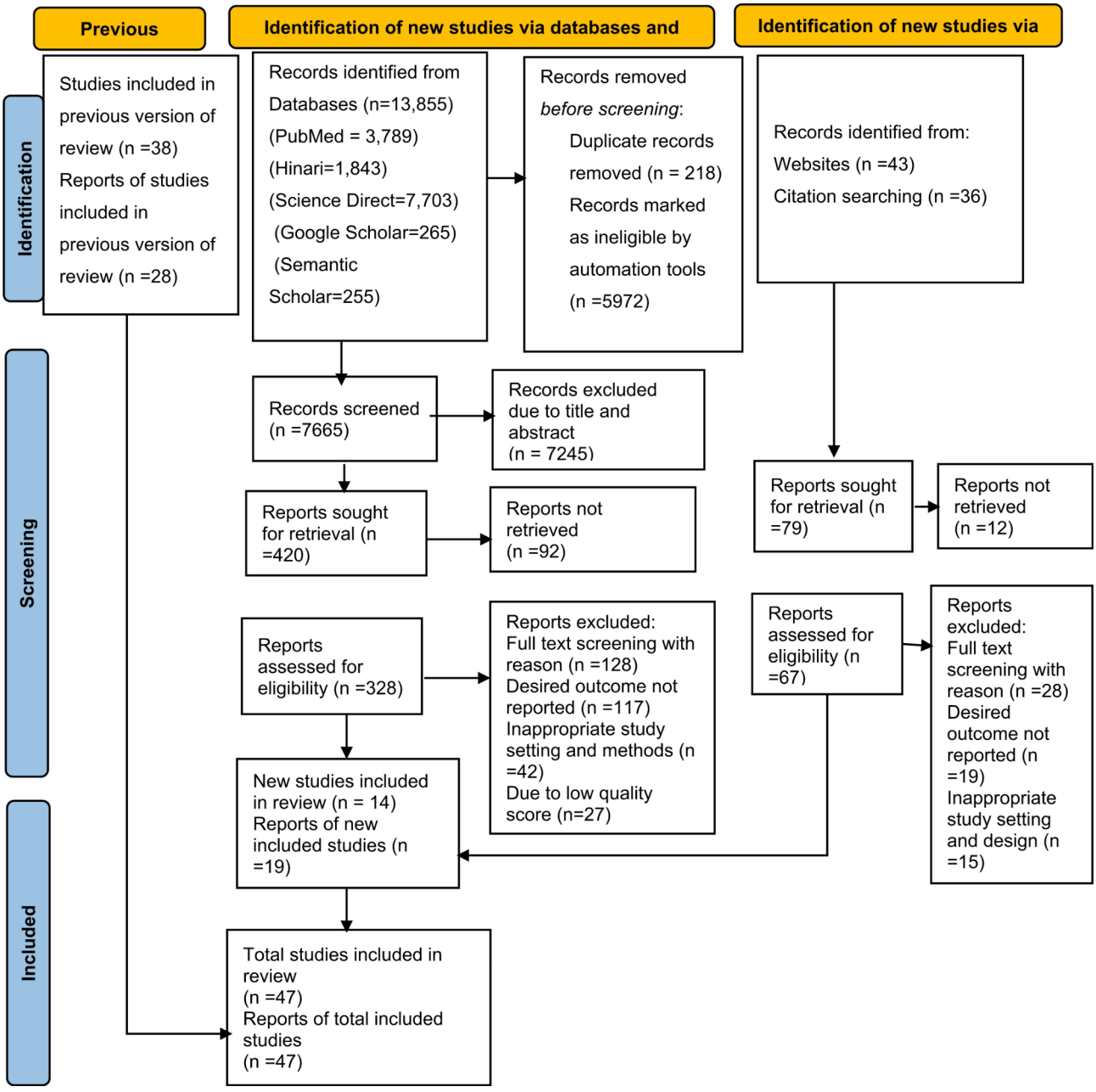

Fig. 1 PRISMA flow diagram of the included studies for the systematic review and meta-analysis of association of particulate matter exposure and asthma and wheeze, 2024

Table 2 Characteristics of all included study for the association between PM10, PM2.5 and asthma, 2024

Author	Year	Country	Study Design	Sample size	Pollutant type	Exposure (µg/m3)	Exposure assessment	Age intervals	Assessment tool	PM10 OR(95%CI)			PM2.5 OR(95%CI)			status
Dockery et al.	1996	USA & Canada	cross sectional	13,369	PM2.5,PM10	Per unit increase	LUR modeling	8–12 years	ISAAC	0.77	0.44	1.35	0.72	0.4	1.28	Low risk
Braun-Fahrlander et al.	1997	Switzerland	cross sectional	4,470	PM10	Per unit increase	Monitoring data	6–15 years	ISAAC	0.81	0.43	1.56				Low risk
Peters et al.	1999	USA	cohort	3,676	PM10	Per IQR increase	Monitoring data	school age	ISAAC	0.93	0.76	1.13				Low risk
Brauer et al. a	2002	Netherland	cohort	2,989	PM2.5	Per IQR increase	LUR modeling	0–2 years	ISAAC				1.12	0.74	2.02	Low risk
Gehring et al.	2002	Germany	cohort	1,756	PM2.5	Per unit increase	LUR modeling	0–2 years	ISAAC				0.98	0.8	1.2	Low risk
Just et al.	2002	France	cohort	82	PM10	Per unit increase	Monitoring data	7–15 years	doctor diagnosed	1.16	0.63	2.13				Low risk
Shima et al.	2002	Japan	cohort	3,048	PM10	Per unit increase	Monitoring data	6–12 years	ISAAC	2.84	0.84	9.58				Low risk
Zhang et al.	2002	China	cross sectional	7,621	PM10,PM2.5	Per unit increase	Monitoring data	5–17 years	ISAAC	1.33	0.8	2.19	1.22	0.74	2.02	Low risk
Kim et al.	2004	USA	cross sectional	1,109	PM2.5,PM10	Per unit increase	Monitoring data	school age	ISAAC	1.06	0.97	1.16	1.04	0.96	1.12	Low risk
Hwang et al.	2005	Taiwan	cross sectional	32,672	PM10	Per unit increase	Monitoring data	school age	ISAAC	0.94	0.90	0.98				Low risk
Penard-Morand et al.	2005	France	cross sectional	6,672	PM10	Per unit increase	Monitoring data	9–11 years	ISAAC	0.54	0.27	1.10				Low risk
Annesi-Maesano et al.	2007	France	cross sectional	5,338	PM2.5	Per unit increase	Monitoring data	school age	ISAAC				1.09	0.89	1.33	Low risk
Brauer et al.	2007	Netherland	cohort	2,826	PM2.5	Per IQR increase	LUR modeling	0–4 years	ISAAC				1.32	1.04	1.69	Low risk
Morgenstern et al.	2007	Germany	cohort	3,577	PM2.5	Per unit increase	LUR modeling	0–2 years	ISAAC				1.10	0.96	1.26	
Morgenstern et al.	2008	Germany	cohort	2,436	PM2.5	Per unit increase	LUR modeling	4–6 years	ISAAC				1.12	0.96	1.31	
Meng et al.	2010	USA	Cross sectional	1502	PM10, PM2.5	Per unit increase	Monitoring data	1–17 Years	Doctor diagnosed	1.31	0.89	1.93	1.48	0.62	3.52	
Akinbami et al.	2010	USA	cross sectional	9,891	PM10,PM2.5	Per unit increase	Monitoring data	3–17 years	NHIS	0.99	0.9	1.09	1.03	0.98	1.08	Low risk
Carlsten et al.	2010	Canada	cohort	184	PM2.5	Per IQR increase	LUR modeling	0–7 years	Doctor diagnosed				3.10	1.30	7.40	Low risk
Clark et al.	2010	Canada	cohort	37,401	PM2.5,PM10	Per unit increase	Monitoring data	0–4 years	Doctor diagnosed	1.07	1.03	1.12	1.01	0.99	1.03	Low risk
Gehring et al.	2010	Netherland	cohort	3,143	PM2.5	Per IQR increase	LUR modeling	0–8 years	ISAAC				1.26	1.04	1.52	Low risk
Penard-Morand et al.	2010	France	cross sectional	6,683	PM10	Per unit increase	Dispersion model	9–11 years	ISAAC	1.28	1.06	1.51				Low risk
Portnov et al.	2012	Israel	cross sectional	3,922	PM10	Per unit increase	Monitoring data	6–14 years	HSD	1.11	1.05	1.17				Low risk
Gruzieva et al.	2013	Sweden	cohort	3,633	PM10	Per unit increase	Dispersion model	0–12 years	Doctor diagnosed	3.8	0.9	16.2				Low risk
Liu et al.	2013	China	cross sectional	6,730	PM10	Per IQR increase	Monitoring data	3–7 years	ATS	1.1	0.85	1.42				Low risk
Nishimura et al.	2013	USA	case control	4,320	PM10,PM2.5	Per unit increase	Monitoring data	8–21 years	Doctor diagnosed	1.13	0.97	1.31	1.02	0.93	1.12	Low risk
Liu et al.	2014	China	cross sectional	23,326	PM10	Per IQR increase	Monitoring data	6–13 years	ATS	1.34	1.24	1.45				Low risk
Molter et al.	2014	England	cohort	10,377	PM10,PM2.5	Per unit increase	LUR modeling	0–10 years	ISAAC questionnaire	0.88	0.63	1.23	1.23	0.78	1.94	Low risk
Susana et al.	2014	Italy	cohort	693	PM10	Per unit increase	Monitoring data	2–18 years	Doctor diagnosed	1.79	1.13	2.84				Low risk
Gehring et alb	2015	Sweden Germany Netherland	cohort	3,702	PM10,PM2.5	Per IQR increase	LUR modeling	0–14 years	ISAAC questionnaire	1.06	0.97	1.15	1.12	1.02	1.24	Low risk
Gehring et ala	2015	Netherland	cohort	14,126	PM10,PM2.5	Per unit increase	LUR modeling	0–16 years	ISAAC questionnaire	1.25	0.94	1.66	1.03	0.95	1.11	Low risk

Table 2 (continued)

Author	Year	Country	Study Design	Sample size	Pollutant type	Exposure (μg/m3)	Exposure assessment	Age intervals	Assessment tool	PM10 OR(95%CI)			PM2.5 OR(95%CI)			status
Wang et al.	2015	Taiwan	cohort	2,661	PM10,PM2.5	Per unit increase	Monitoring data	Birth-KG	doctor diagnosed	1.39	1.03	1.87	1.45	1.07	1.97	Low risk
Deng et al.	2016	China	cross sectional	2,598	PM10	Per unit increase	Monitoring data	3–6 years	ISAAC questionnaire	1.1	0.95	1.27				Low risk
Kim et al.	2016	Korea	cross sectional	1,828	PM10	Per unit increase	Monitoring data	6–7 years	ISAAC questionnaire	1	0.71	1.42				Low risk
Liu et al.	2016	China	cross sectional	3,358	PM10	Per unit increase	Monitoring data	4–6 years	ISAAC questionnaire	1.38	1.02	1.87				Low risk
Chen F et al.	2017	China	cross sectional	30,759	PM2.5	Per unit increase	Monitoring data	School	ISAAC questionnaire				1.10	1.03	1.18	Low risk
Chen BY et ala	2019	China	cross sectional	6346	PM10,PM2.5	Per unit increase	Monitoring data	preschool children	ISAAC questionnaire	1.48	0.93	2.36	1.90	1.41	2.57	Low risk
Chen BY et alb	2019	China	cross sectional	11,585	PM10,PM2.5	Per unit increase	Monitoring data	preschool children	Doctor diagnosed	1.37	0.92	2.04	1.24	1.04	1.48	Low risk
Olaniyan et al.	2020	South Africa	cohort	590	PM2.5	Per IQR increase	Monitoring data	school age	Doctor diagnosed				0.86	0.32	2.3	Low risk
Ortega-Garcia et al.	2020	Spain	cross sectional	12,354	PM10	Per unit increase	Monitoring data	less than 19 years	Doctor diagnosed	1.02	1.01	1.04				Low risk
Wen Zeng et al.	2021	China	cross sectional	59,754	PM10,PM2.5	Per unit increase	LUR modeling	2–18 years	ATS	1.29	1.2	1.38	1.49	1.34	1.67	Low risk
Wu et al.	2022	China	cross sectional	29,418	PM10,PM2.5	Per unit increase	Monitoring data	4–6 years	ISAAC questionnaire	1.11	1.02	1.2	1.14	1.03	1.26	Low risk
Chen J et al.	2023	China	cohort	4,864	PM2.5	Per unit increase	LUR	6–16 years	ISAAC questionnaire				1.07	0.93	1.23	Low risk
Giien Tsai et al.	2024	China	cohort	4,736	PM2.5	Per unit increase	LUR modeling	6–18 years	Doctor diagnosed				1.60	1.31	1.96	Low risk
Chen T et al.	2024	China	cross sectional	11,825	PM10,PM2.5	Per IQR increase	Monitoring data	Preschool	Doctor diagnosed	1.53	1.27	1.85	1.39	1.24	1.56	Low risk
Zanobeti et al.	2024	USA	cohort	5,279	PM2.5	Per unit increase	Monitoring data	1–12 years	Doctor diagnosed				1.30	1.03	1.65	Low risk

Table 3 Characteristics of all included study for the association between PM10, PM2.5 and wheeze,2024

Author	Year	Country	Study Design	Sample size	Exposure	Exposure mean concentration	Exposure assessment method	Age of assessment (years)	Outcome assessment	OR, PM10, 95% CI			OR, PM2.5,95%CI:			Quality status
Brauer et al.	2007	Netherlands	Cohort	4,000	PM2.5	> 10 µg/m3	Monitoring data	0–4 years	ISAAC questionnaire				1.22	1.06	1.41	Low risk
Braun-Fahrlander	1997	Switzerland	cross sectional	4470	PM10	> 10 µg/m3	Monitoring data	6–15 years	ISAAC questionnaire	0.92	0.47	1.78				Low risk
Dockery et al.	1996	USA & Canada	cross sectional	13,369	PM2.5,PM10	> 10 µg/m3	Monitoring data	8–12 years	ISAAC questionnaire	0.79	0.57	1.08	0.82	0.58	1.15	Low risk
Gehring et al.	2002	Germany	Cohort	1756	PM2.5.	≤ 10 µg/m3	LUR modeling	0–2 years	ISAAC questionnaire				0.91	0.76	1.09	Low risk
Gehring et al.	2010	Netherlands	Cohort	3863	PM2.5	≤ 10 µg/m3	LUR modeling	0–8 years	ISAAC questionnaire				1.2	1.08	1.33	Low risk
Giien Tsai et al.	2024	Taiwan	Cohort	4,736	PM2.5	≤ 10 µg/m3	LUR modeling	6–18 years	Doctor diagnosed				1.73	1.4	2.1	Low risk
Linares et al.	2010	Mexico	Cohort	464	PM10	≤ 10 µg/m3	Monitoring data	6–14 years	ATS questionnaire + spirometry	1.05	1.03	1.06				Low risk
Liu et al. a	2013	China	cross sectional	6730	PM10	> 10 µg/m3	Monitoring data	3–7 years	ATS	1.35	1.15	1.57				Low risk
Liu et al. b	2014	China	cross sectional	23,326	PM10	> 10 µg/m3	Monitoring data	6–13 years	ATS	1.16	1.07	1.27				Low risk
Morgenstern et al.	2007	Germany	Cohort	3,577	PM2.5	> 10 µg/m3	Monitoring data	0–2 years	ISAAC questionnaire				1.1	0.94	1.28	Low risk
Naidoo et al.	2013	South Africa	Cohort	792	PM10	≤ 10 µg/m3	Monitoring data	2–14 years	Doctor diagnosed	1.27	0.75	2.15				Low risk
Olaniyan et al.	2020	South Africa	Cohort	590	PM2.5	> 10 µg/m3	Monitoring data	school age	Doctor diagnosed				1.08	0.71	1.66	Low risk
Penard-Morand et al.	2005	France	cross sectional	6672	PM10	> 10 µg/m3	Monitoring data	9–11 years	ISAAC questionnaire	1.05	0.72	1.54				Low risk
Peters et al.	1999	USA	Cohort	3,676	PM10	> 10 µg/m3	Monitoring data	school age	ISAAC questionnaire	1.05	0.89	1.25				Low risk
Velicka et al.	2015	Czech Republic	Cohort	147	PM10	≤ 10 µg/m3	Monitoring data	6–18 years	Doctor diagnosed	1.07	1.04	1.11				Low risk
Wen Zeng et al.	2021	China	cross sectional	59,754	PM10,PM2.5	≤ 10 µg/m3	LUR modeling	2–18 years	ATS	1.14	1.07	1.21	1.24	1.12	1.37	Low risk
Wu et al.	2022	China	cross sectional	29,418	PM2.5	> 10 µg/m3	Monitoring data	4–6 years	ISAAC questionnaire	1.03	0.98	1.09	1.08	1.01	1.16	Low risk
Zhang et al.	2002	China	cross sectional	7,621	PM10,PM2.5	> 10 µg/m3	Monitoring data	school age	ISAAC questionnaire	1.12	0.58	2.15	1.05	0.58	1.92	Low risk

four from Netherland [21, 39, 50, 52], three from Germany [49, 64, 65], two from Canada [41, 67], two from South Africa [66, 69], one each from Italy [75], Sweden [53], Czech Republic [77], England [82], Japan [61], Israel [74], Switzerland [40], Spain [70] and Mexico [38] where two combined from USA and Canada [48] as well as Sweden, Netherland and Germany [51].

PM10 exposure and childhood asthma

A meta-analysis based on 31 studies was conducted using a random effects model to investigate the association between childhood asthma and PM10 exposure. The pooled odds ratio (OR) of 1.04 (95% CI: 1.03–1.06, $p < 0.001$) showed a statistically significant association between PM10 exposure and the risk of childhood asthma. However, considerable heterogeneity was detected (I^2 = 82.7%, $p < 0.001$) indicating variability between odd ratio across included studies (Fig. 2).

Subgroup analysis showed different heterogeneity in different WHO regions ($p = 0.037$). Non-significant low heterogeneity was detected in the Americas (1.05, 95% CI: 1.01–1.10, I^2 = 13.0%, $p = 0.331$) and significant moderate heterogeneity in the European region (1.09, 95% CI: 0.99–1.21, I^2 = 59.6%, $p = 0.008$). The Western Pacific showed strong association and extreme heterogeneity (1.27, 95% CI: 1.12–1.45, I^2 = 90.0%, $p < 0.05$) highlighting extreme variability across regions (Fig. 3).

The forest plot for subgroup analysis by study design indicated that the pooled OR of cross-sectional studies was 1.14 (95% CI: 1.07–1.22), showed a significant association. However, there is high variability among the included studies (I^2 = 87.5%, $p < 0.001$). Cohort studies found an effect size of 1.10 (95% CI: 1.00-1.21) with low variability (I^2 = 48.9%, $p = 0.04$) that indicating a persistent but significantly weaker association. The overall effect size from all research types is 1.13 (95% CI: 1.08–1.19), demonstrating that PM10 exposure is associated with childhood asthma. However, the analysis revealed no significant heterogeneity ($p = 0.828$) in effect estimates across different study designs, suggesting consistent conclusions (Fig. 4).

The meta-regression analysis also demonstrated a significant upward trend in the coefficients for asthma related to PM10 exposure over time, indicating that the impact of PM10 on asthma has increased across the studied years (Fig. 5).

The association between PM2.5 exposure and childhood asthma

A meta-analysis using data from 29 primary observational studies showed a combined odd ratio of 1.05 (95% CI 1.04–1.07, $p < 0.001$). This suggests that exposure to PM2.5 increases the likelihood of acquiring asthma in childhood. The analysis revealed high heterogeneity among the included studies ($I^2 = 80.6\%$, $p < 0.001$), which implies that effect estimates vary among studies, as shown in Fig. 6.

Subgroup analysis by regions showed significant variability in effect sizes between geographic regions ($p = 0.001$). Significant association was observed only in the Western Pacific OR = 1.31, (95% CI: 1.18–1.46, I^2 = 48.6%, $p = 0.059$), while homogeneity was observed in European region (OR = 1.09, 95% CI: 1.04–1.14) and in America (1.03, 95% CI: 0.99–1.08, I^2 = 0.0%, $p = 0.482$) (Fig. 7).

Subgroup analysis was conducted based on exposure assessment methods, revealing no significant variability in effect sizes across different methods. This indicates that the choice of exposure assessment method does not significantly influence the overall findings ($p = 0.189$). Specifically, studies utilizing monitoring data reported a cumulative effect size of 1.12 (95% CI: 1.05–1.19), with high heterogeneity observed among the included studies (I^2 = 79.7%, $p < 0.001$). Similarly, studies employing land use regression modeling demonstrated a cumulative odds ratio of 1.20 (95% CI: 1.10–1.31), also with significant heterogeneity (I^2 = 73.3%, $p < 0.001$) (Fig. 8).

The forest plot groups studies according to PM2.5 exposure levels, distinguishing between those per unit increases and per IQR increases in $\mu g/m^3$. Both per unit and per IQR increases in PM exposure show significant health risks, with high overall heterogeneity. The lack of significant between-group variability ($p = 0.222$) suggests that the choice of exposure measurement method does not significantly impact the findings (Fig. 9).

Subgroup analysis based on outcome assessment indicated that absence of significant variation between asthma cases reported by parents and identified by clinician ($P = 0.290$). Studies based on parent reports of asthma exhibited a cumulative effect size of 1.14 (95% CI: 1.08–1.21) with a significant variability (I^2 = 71.9%, $p < 0.001$), indicating high heterogeneity among the included studies.

On the other hand, studies employing physician diagnoses for asthma had a slightly larger pooled effect size of 1.23 (95% CI: 1.08–1.41) but extreme variability (I^2 = 85.9%, $p < 0.001$). Overall, the analysis across both categories produced an effect size of 1.16 (95% CI: 1.10–1.22) with significant high variability (I^2 = 80.6%, $p < 0.000$). These findings indicate a significant association between PM2.5 exposure and childhood asthma, observed in both parent-reported and physician-diagnosed cases (Fig. 10).

Additionally, meta-regression analysis revealed a statistically significant increase in the coefficient of asthma related to PM2.5 over time ($\beta = 0.012$, 95% CI: 0.004–0.019, $p = 0.006$), with the year of study explaining a substantial portion of the between-study variance. However, sample size did not contribute to the heterogeneity

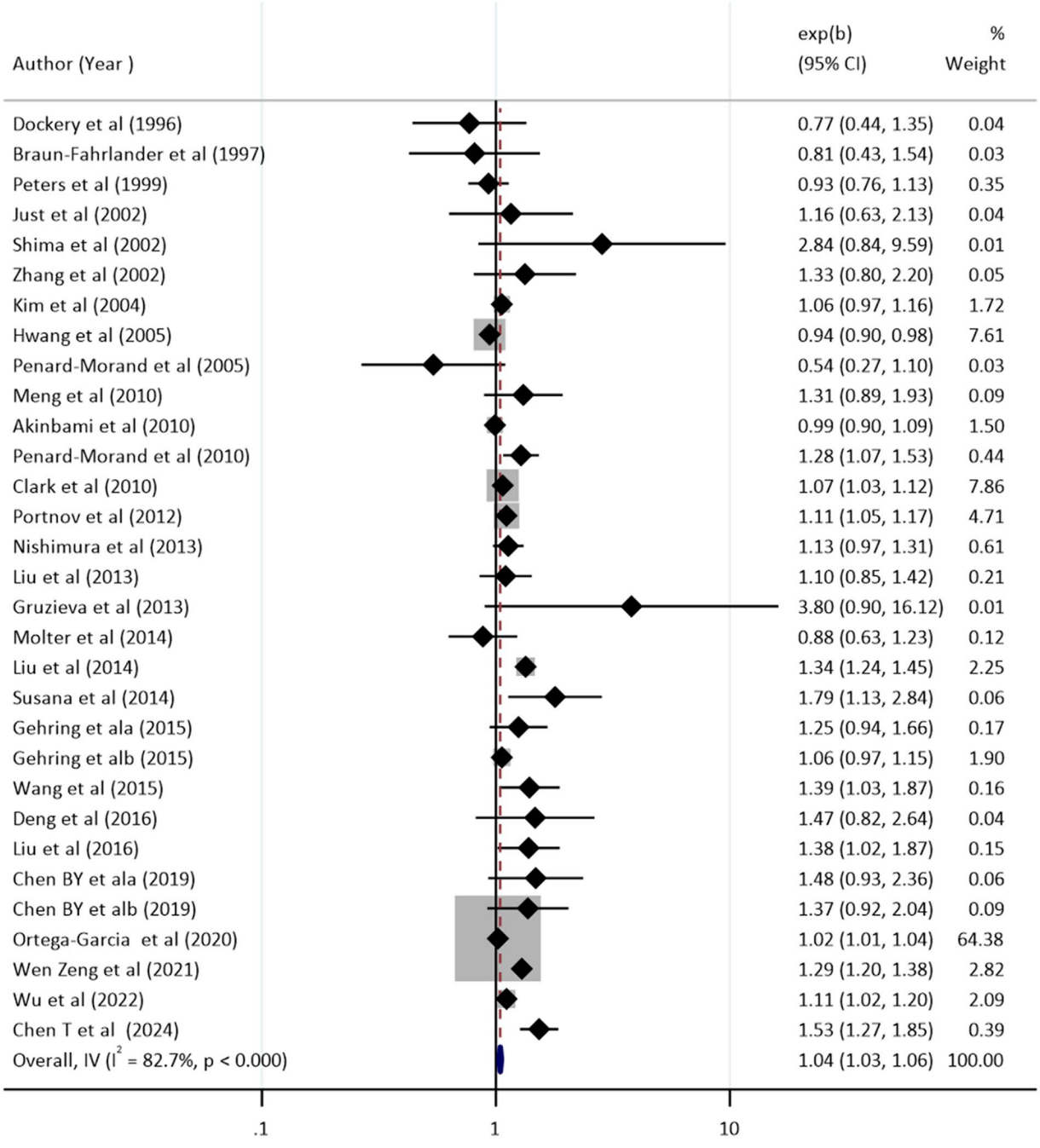

Fig. 2 Forest plot of odd ratios for the association of particulate matter (PM10) exposure and asthma, 2024

among the included studies. These results suggest that the impact of PM2.5 on asthma has been progressively increasing over the years examined in the studies (Fig. 11).

Particulate matter exposure and wheezing

The first plot indicates the correlation between PM10 exposure and wheeze. The overall pooled odd ratio indicates a significant association between PM10 and wheeze with an odds ratio of 1.06 (95% CI: 1.05, 1.07) with moderate heterogeneity between studies (I^2=57.5%, p<0.007). This result indicates exposure to PM10 increases the likelihood of wheezing (Fig. 12).

The second plot examines the relationship between PM2.5 exposure and wheeze. It also showed a significant association with a pooled odds ratio of 1.15. (95% CI: 1.10, 1.20). This implies that PM2.5 exposure has a stronger association with wheeze than PM10 exposure.

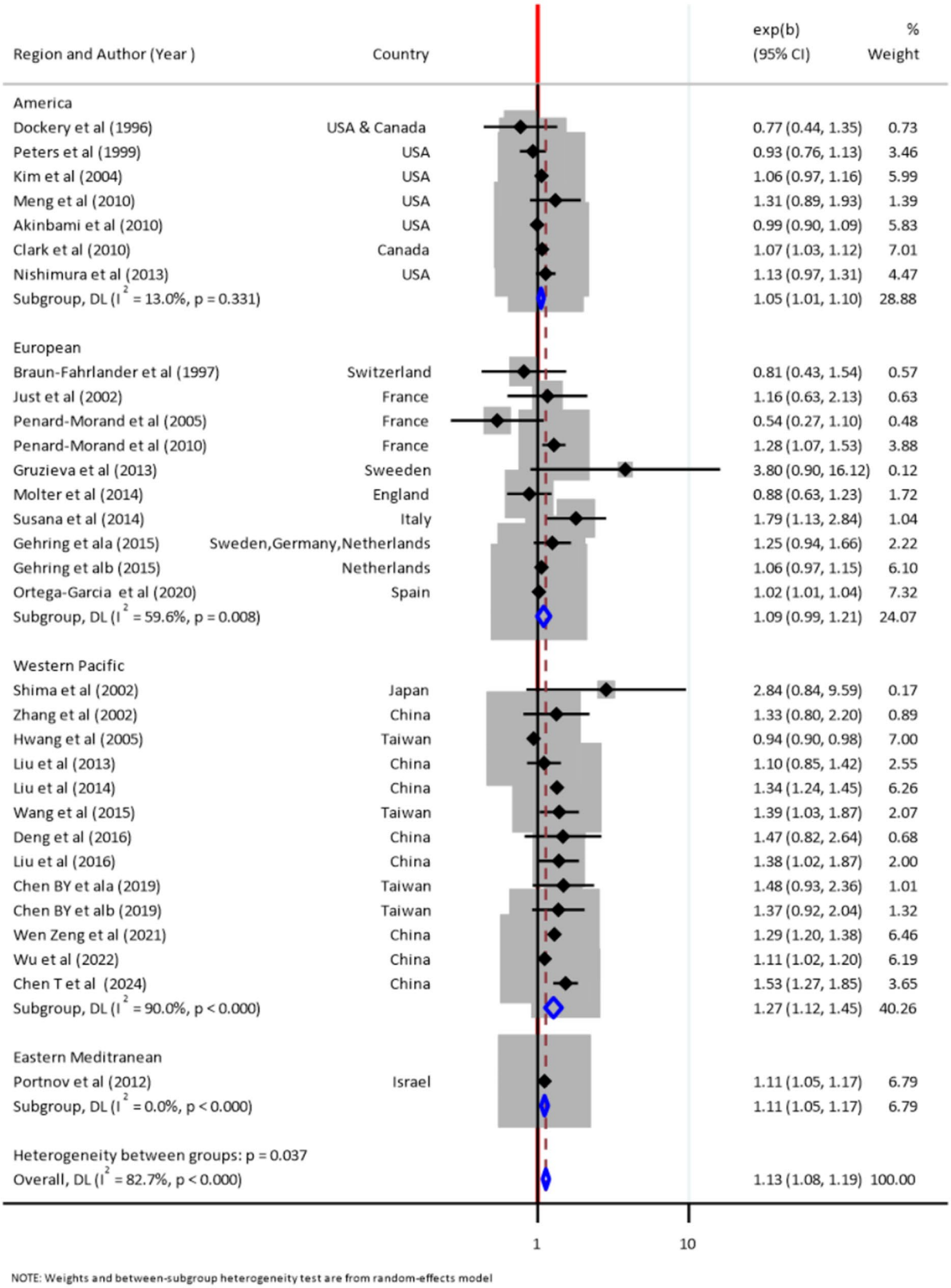

Fig. 3 Subgroup analysis of association between PM10 exposure and asthma using WHO regions, 2024

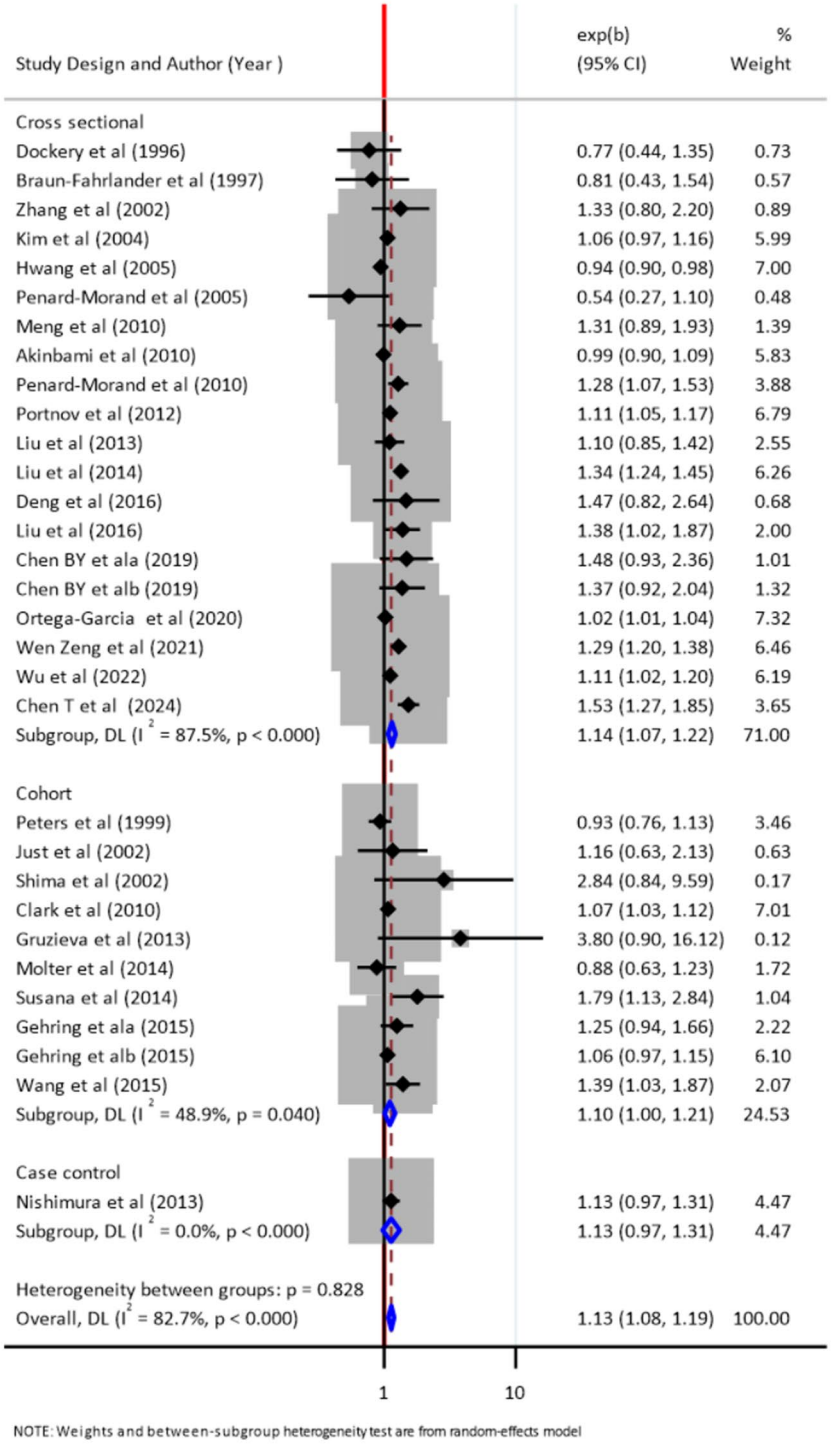

Fig. 4 Subgroup analysis of association between PM10 exposure and asthma using study design, 2024

The heterogeneity for PM2.5 exposures is higher than for PM10 exposure, with an I^2 of 72.8%, indicating significant variation among the included studies. Overall meta-add ratio results indicate that PM2.5 exposures are associated to an elevated risk of wheezing compared with PM10 exposure (Fig. 12).

Publication bias assessment

The included studies were evaluated for publication bias. It arises when research with significant outcomes is more likely to be published than research without significant results. To reduce publication bias, a comprehensive literature search using several databases was conducted

```
. metareg logORPM10 Samplesize , wsse(selogORPM10 )

Meta-regression                                         Number of obs   =        31
REML estimate of between-study variance                 tau2            =    .01555
% residual variation due to heterogeneity               I-squared_res   =    82.55%
Proportion of between-study variance explained          Adj R-squared   =   -10.09%
With Knapp-Hartung modification
```

logORPM10	Coefficient	Std. err.	t	P>|t|	[95% conf.	interval]
Samplesize	-2.99e-07	2.11e-06	-0.14	0.888	-4.61e-06	4.01e-06
_cons	.1369557	.0478321	2.86	0.008	.039128	.2347833

```
. metareg logORPM10 Year , wsse( selogORPM10 )

Meta-regression                                         Number of obs   =        31
REML estimate of between-study variance                 tau2            =    .01025
% residual variation due to heterogeneity               I-squared_res   =    83.09%
Proportion of between-study variance explained          Adj R-squared   =    27.45%
With Knapp-Hartung modification
```

logORPM10	Coefficient	Std. err.	t	P>|t|	[95% conf.	interval]
Year	.0122317	.0042821	2.86	0.008	.0034739	.0209895
_cons	-24.49413	8.619154	-2.84	0.008	-42.12228	-6.865984

Fig. 5 Meta regression analysis of association between PM10 exposure and asthma using sample size and study year, 2024

from different databases. An egger's test and funnel plot were employed to evaluate publication bias. Egger's test was found to be non-significant for small study effects ($p > 0.05$), although the funnel plot revealed an asymmetrical distribution of studies. Egger's test results and funnel plot asymmetry may be due to sample size, heterogeneity, or method sensitivity differences, rather than true bias (Fig. 13).

Discussion

Air pollution, which is influenced by the unique nature and characteristics of particulate matter, has become a serious global public health issue. Children and adolescents who are exposed to particle matters, particularly PM10 and PM2.5, cause significantly respiratory diseases and symptoms, including asthma and wheezing. As a result, we performed a comprehensive systematic review and meta -analysis, and analyzed 47 studies published between 1996 and June 17, 2024. The purpose of the study was to evaluate the relationship between PM exposure (PM2.5 and PM10) and asthma/wheeze development among children and adolescents.

We generated statistically significant random-effect estimates for PM2.5 and PM10 exposures using both general and group-specific meta-analyses. The sensitivity analyses confirmed the robustness of our conclusions. Despite the strength of the overall findings, substantial variability was detected, notably for PM2.5. The highest heterogeneity was noted in the association between PM10 exposure and asthma development. This study finding showed a significant association between PM10 and asthma, PM2.5 and asthma, PM10 and wheeze as well as PM2.5 and wheeze among children and adolescents. Our study finding supports the previous findings [5, 31, 83–85] in which indicated a significant association between PM exposure (PM2.5 and PM10) and asthma development among children and adolescents.

Both the present study and earlier systematic review and meta-analysis emphasized the significance of associations between PM exposure and respiratory diseases and symptoms to highlight the impact of air quality on

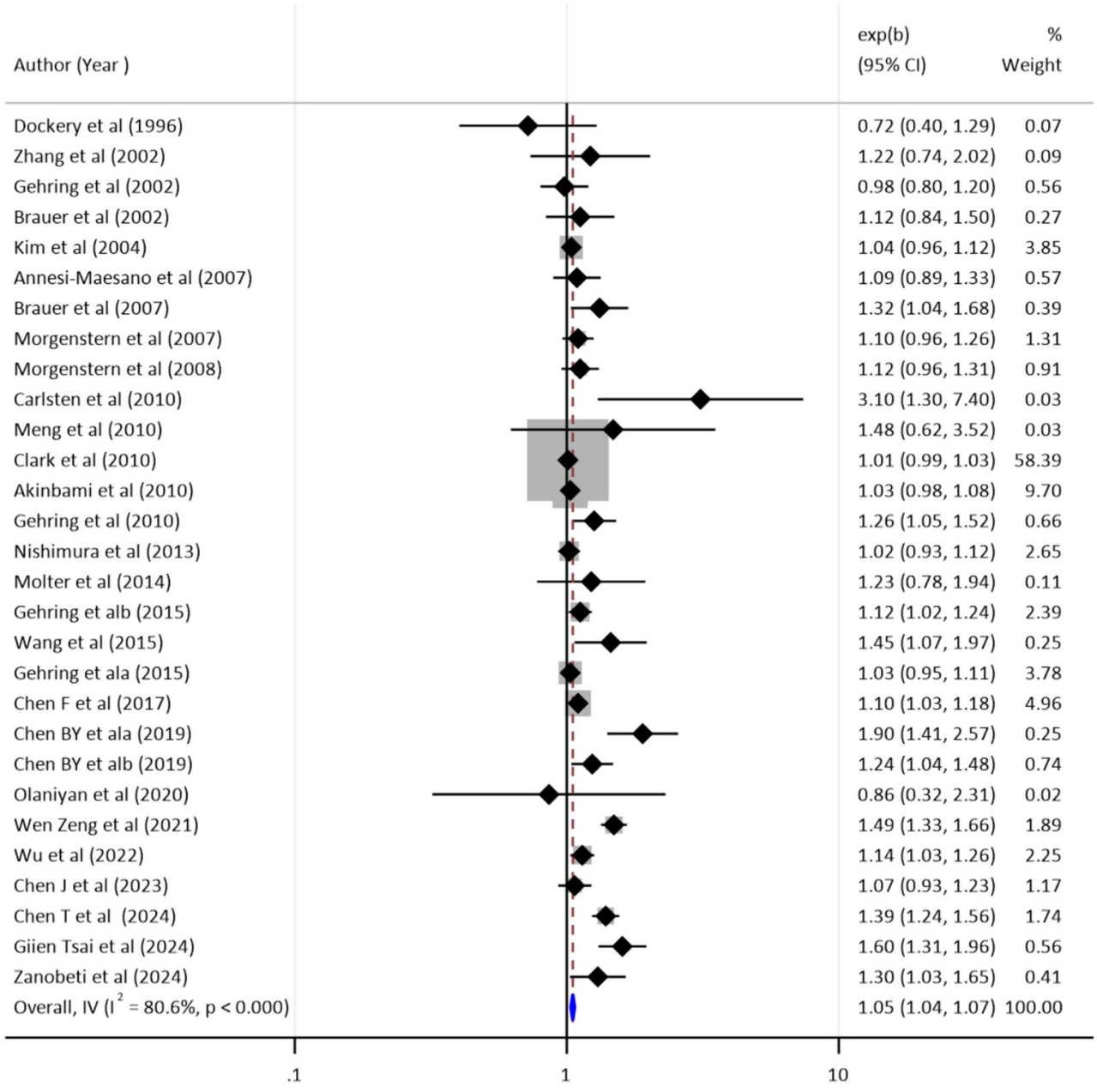

Fig. 6 Forest plot of odd ratios for the association between PM2.5 exposure and asthma, 2024

respiratory health [83]. However, another systematic review and meta-analysis conducted on the association between air pollution exposure and childhood asthma reported no significant association, although a significant association between PM and wheeze was reported [63, 86].

This disparity on association between PM10 and/or PM2.5 exposure and childhood asthma across systematic reviews and meta-analyses undertaken at different times can be attributable to several variables. One of the primary cause might be the collection of new evidence along time. As more studies become published and included in meta-analysis, the statistical power to detect a significant association increases particularly those using advanced exposure assessment techniques, the evidence base become more robust, resulting in more conclusive findings [87, 88].

Improvements in study methodology may also help to explain the discrepancy. Earlier studies might have used less precise methods for measuring PM exposure

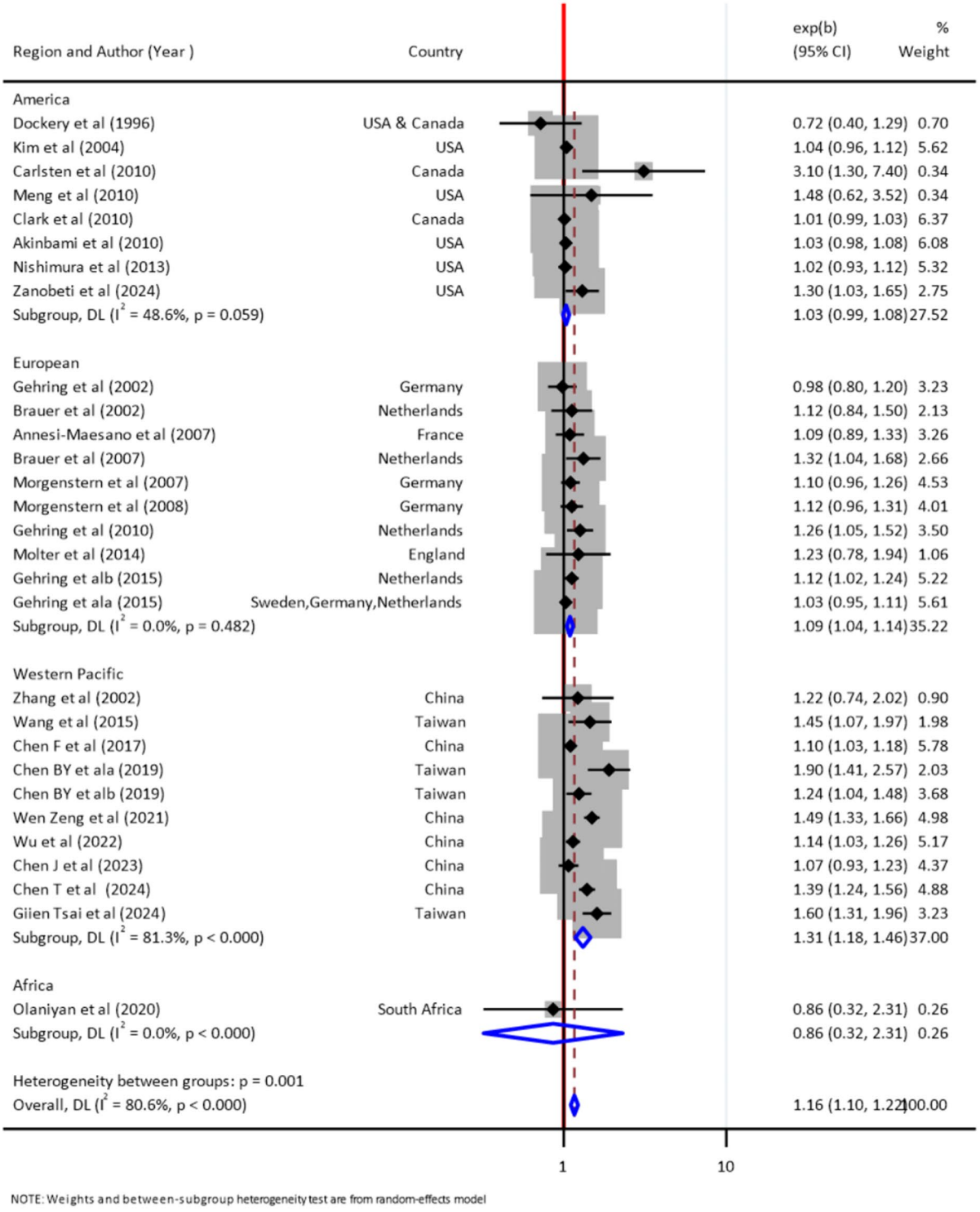

Fig. 7 Subgroup analysis of association between PM2.5 exposure and asthma using WHO regions, 2024

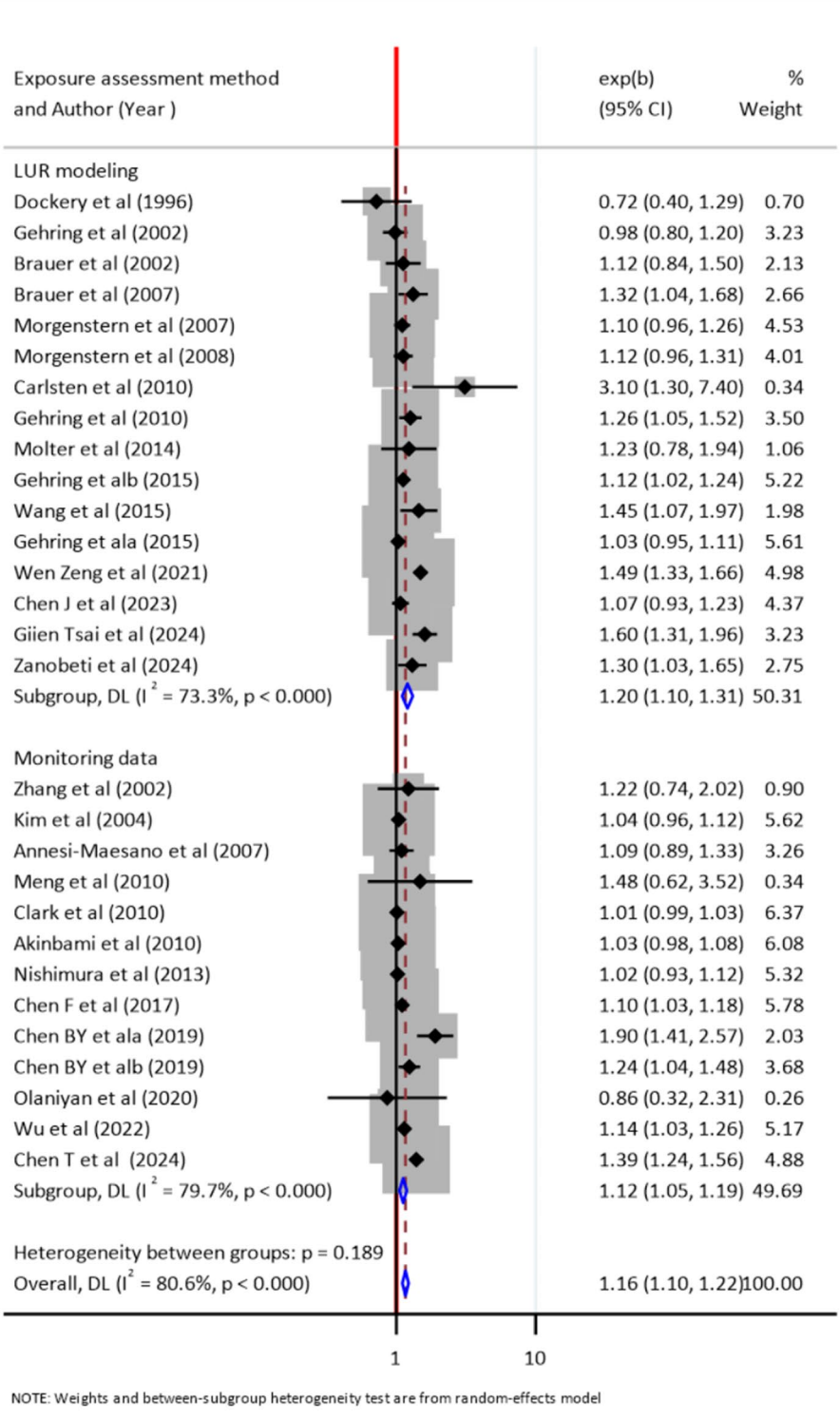

Fig. 8 Subgroup analysis of PM2.5 and asthma using exposure assessment methods, 2024

or smaller sample sizes, making it difficult to identify a significant association [89]. Later studies frequently utilize more sophisticated tools, like personal monitoring devices, to quantify exposure more accurately, resulting in stronger and more reliable findings [90].

Changes in environmental conditions over time are also important factor for this discrepancy [91]. In recent years, stricter environmental regulations and better technologies may have decreased exposure to PM [92]. This reduction may enable researchers to better uncover the

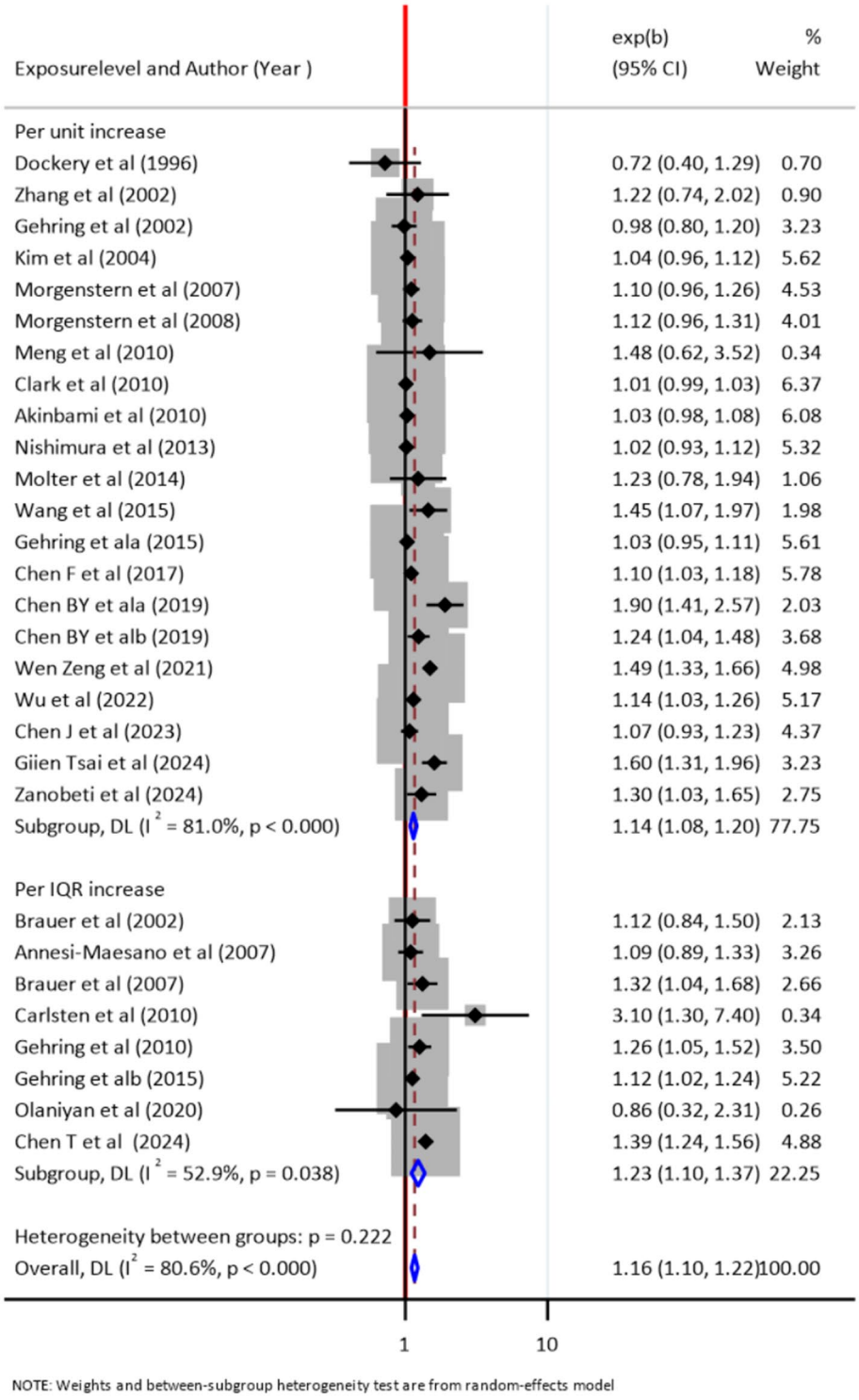

Fig. 9 Subgroup analysis of PM2.5 and asthma using exposure mean concentration, 2024

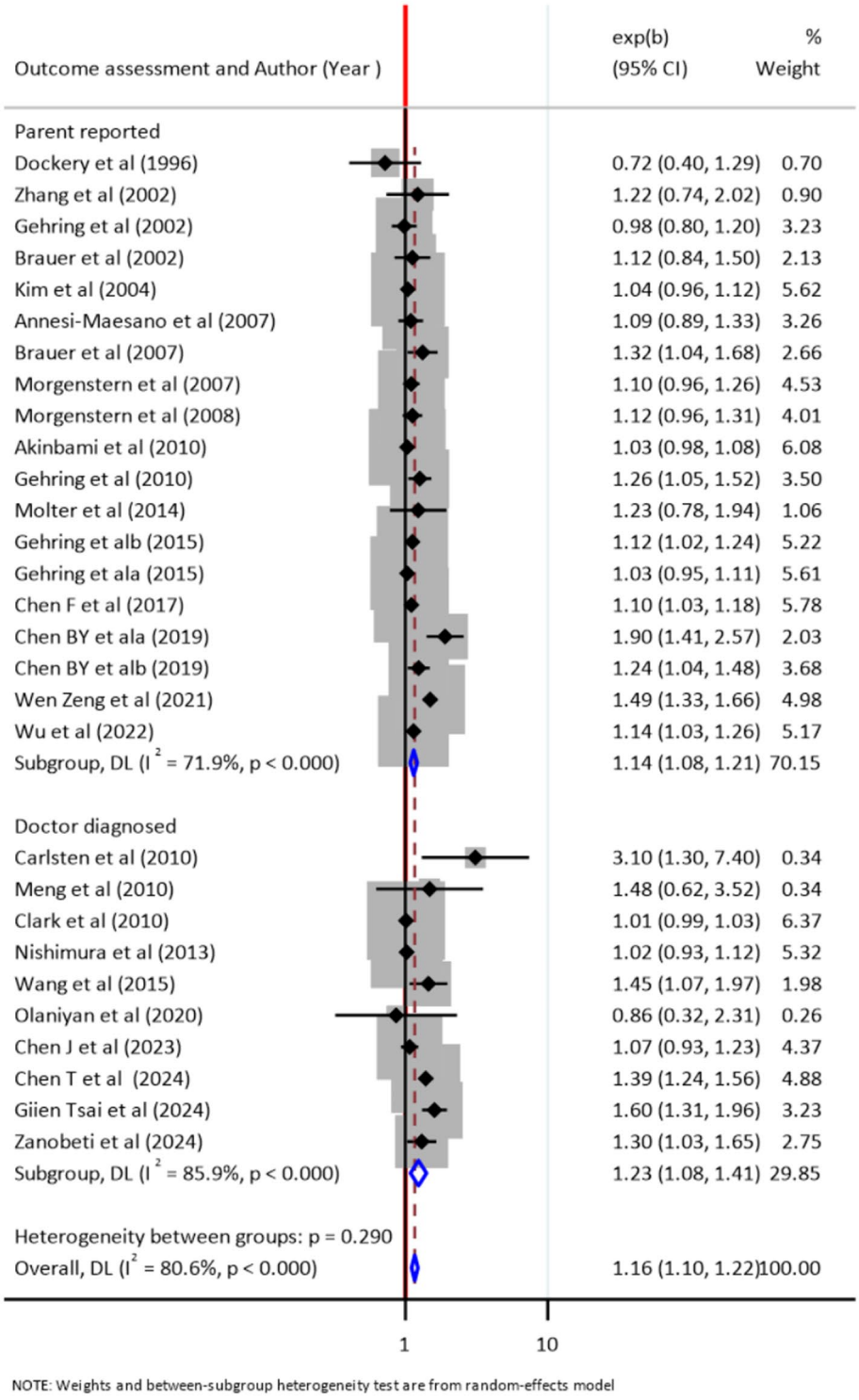

Fig. 10 Sub group analysis of association between PM2.5 exposure and asthma using types of outcome assessment, 2024

```
. metareg logORPM255 Samplesize , wsse( selogORPM25 )

Meta-regression                                 Number of obs   =        29
REML estimate of between-study variance         tau2            =    .01718
% residual variation due to heterogeneity       I-squared_res   =    80.03%
Proportion of between-study variance explained  Adj R-squared   =    -7.06%
With Knapp-Hartung modification
```

logORPM255	Coefficient	Std. err.	t	P>|t|	[95% conf.	interval]
Samplesize	4.02e-07	2.16e-06	0.19	0.854	-4.02e-06	4.83e-06
_cons	.1519883	.044007	3.45	0.002	.0616933	.2422833

```
. metareg logORPM255 Year , wsse( selogORPM25 )

Meta-regression                                 Number of obs   =        29
REML estimate of between-study variance         tau2            =   .009422
% residual variation due to heterogeneity       I-squared_res   =    67.85%
Proportion of between-study variance explained  Adj R-squared   =    41.28%
With Knapp-Hartung modification
```

logORPM255	Coefficient	Std. err.	t	P>|t|	[95% conf.	interval]
Year	.0116933	.0038808	3.01	0.006	.0037305	.019656
_cons	-23.39892	7.81497	-2.99	0.006	-39.43391	-7.363923

Fig. 11 Meta regression analysis of association between PM10 exposure and asthma using sample size and study year, 2024

association between even lower amounts of PM and asthma, as the consequences of chronic, low-level exposure become obvious over time. Furthermore, this discrepancy could also be influenced by changes in public health practices, such as improved asthma management, higher indoor air quality standards, and increased public awareness [93].

As asthma symptoms improve and other environmental factors improve, epidemiological studies will be able to identify the particular function of particulate matter in initiating asthma. The study's findings on PM exposure's association with childhood asthma and wheeze are highly heterogeneous, possibly due to regional differences, population characteristics, study year, demographics, environmental conditions, and confounding variables [94].

The current study found higher diversity by study design across the included studies. The investigation of the association between PM exposure and childhood asthma yields more strong relationship from cross-sectional than cohort studies. This difference could be attributed to the fact that cross-sectional studies examine data at a particular point in time, allowing for the detection of immediate association but restricting causal inferences [95]. This design may overstate the strength of the relationship, particularly because it frequently measures short-term exposures, capturing peak exposure levels that amplify the reported association. Finally, cross-sectional studies may include populations more susceptible to PM exposure, further strengthening the observed associations.

On the other hand, cohort studies track people over time, offering a more accurate picture of cause-and-effect association. However, the averaging of long-term exposure data can result in weaker associations [96]. Furthermore, cohort studies often account for confounding factors more successfully and demonstrate exposure

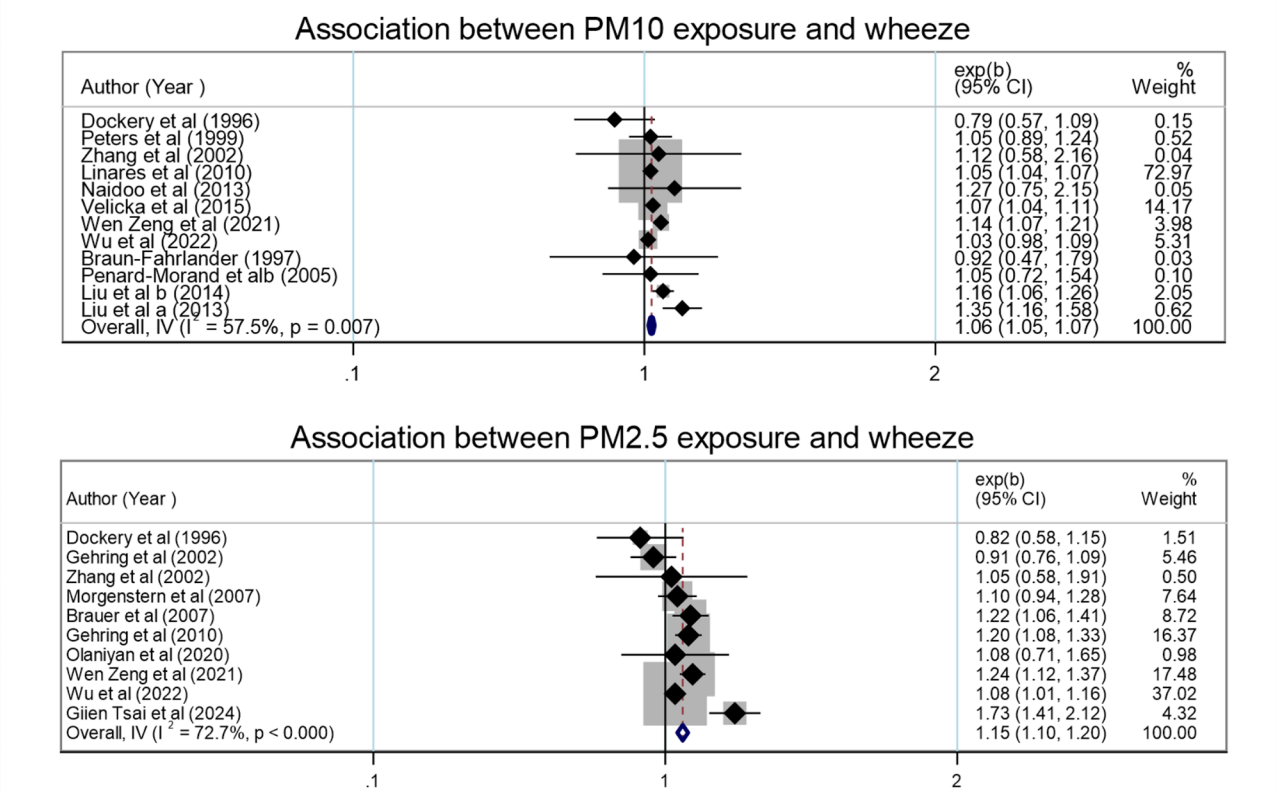

Fig. 12 Forest plot of subgroup odd ratios for the association between PM10 and PM2.5 exposure and wheeze, 2024

before to asthma development, reducing difficulties of reverse causation [97].

The study revealed a strong significant association between the development of childhood asthma and PM2.5 exposures at per inter quartile range (IQR) increase compared to per unit increase with lower heterogeneity. This variance may be because of exposure at IQR increase representing a wide range of exposure covering 50% of data that better reflects the exposure than per-unit increase. It can identify all substantial changes at high level of exposure, leading to a strong association with the outcome.

Another key reason might be the non-linear exposure-response relationship, where the IQR increase might better reflect the overall effect of PM2.5 exposure as it encompasses a wider span of exposure level [98–100]. In this case IQR can reflect better general effects, as it contains a wide ranges of exposure values. Per unit increase can only capture small, step by step changes that do not fully represent widespread trends in contrast to IQR increase exposure [101].

For individuals potentially exposed to unit levels of pollution, their vulnerability might be lower [102], on the other hand, individuals with wider pollution range may readily acquire the health outcome because of the increased effect of pollution [103, 104]. Another reason could be exposure misclassification in studies with a broad range of exposure, where range pollution data might not reflect individual exposure which leads to dilute the observed association [105]. Moreover, per-unit exposure assessments might be more sensitive to measurement errors in individual studies [106].

Finally, there may be a threshold effect at which the association between PM2.5 and asthma development becomes weaker beyond a certain concentration, indicating a saturation point in the exposure-response relationship [104]. Such factors together explain why stronger associations with asthma are observed using exposure at the IQR increase rather than the exposure at per unit increase. The reason for higher heterogeneity in studies with PM2.5 exposures at per unit increase and asthma may be due to differences in study design, population characteristics, exposure assessments and outcome measurements [107].

Studies relying on association between PM2.5 and parent-reported asthma exhibited a cumulative effect size of 1.15 with significant high heterogeneity. In contrast, studies that employed PM2.5 exposure and physician diagnosed asthma showed a slightly higher pooled effect size of 1.25 but with significant extreme heterogeneity. This differences may be because of the subjective nature of parent-reported outcomes, wherein flaws like as recall

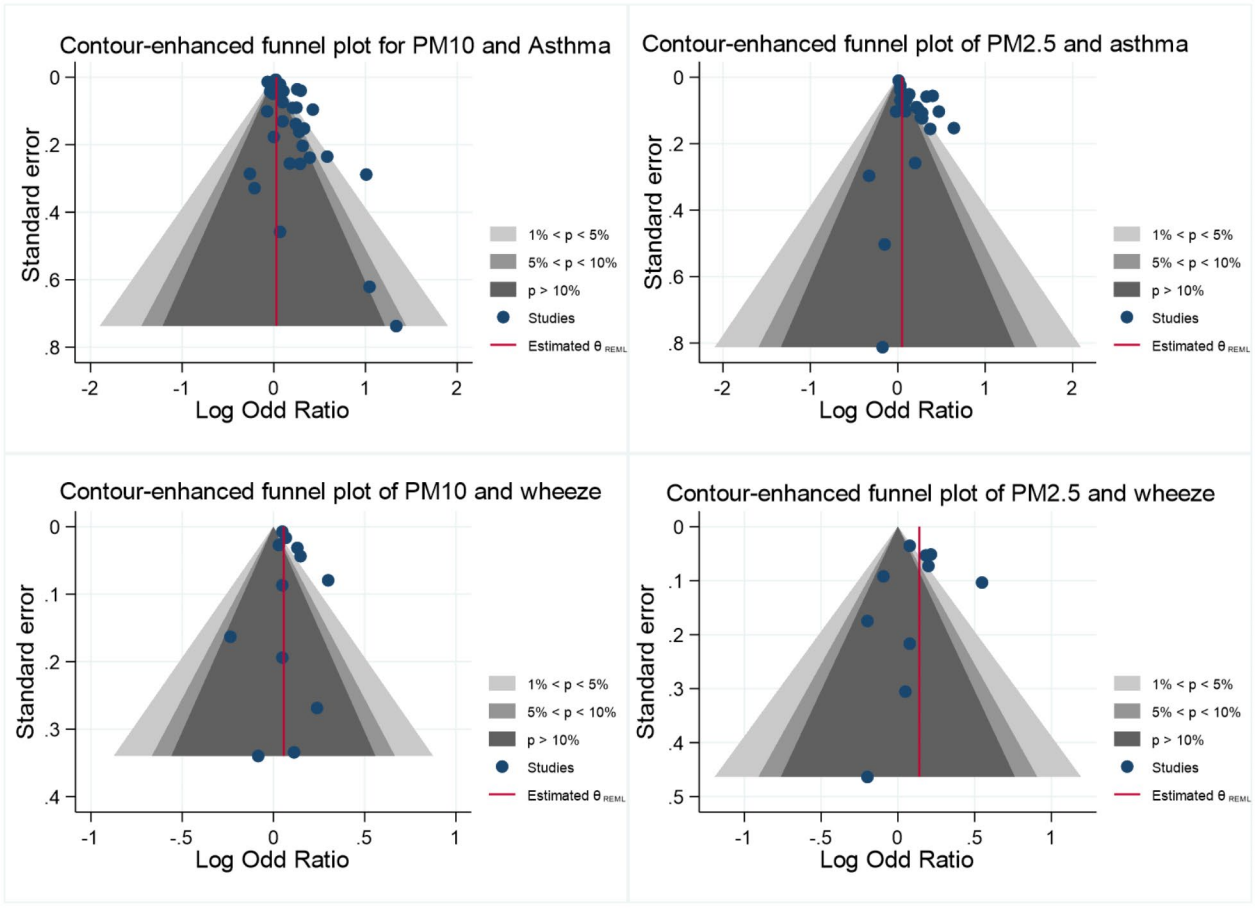

Fig. 13 Funnel plot of log odd ratios for the association between PM2.5 and PM10 exposure versus asthma and wheeze, 2024

bias, misinterpretation of signs and symptoms, or misclassification of different respiratory diseases as asthma or wheeze might add inconsistencies among studies leads high variability in effect estimates among studies [108].

Although clinician-diagnosed asthma is likely more reliable and based on defined diagnostic criteria, the heterogeneity could come from changes in diagnostic techniques, types of healthcare setup, clinician/physician variation in diagnosis, and population characteristics across different areas [109]. Furthermore, variations in the measurement or assessment of PM exposure among studies may contribute to the observed variability in both groups [110]. Overall, the differences between parent-reported and physician-diagnosed asthma indicates the need of using standardized, objective outcome measures to reduce bias and improve the consistency of findings in epidemiological studies of PM and asthma.

Generally, a detailed evaluation of this review revealed a relatively strong association between PM2.5 exposures and development of asthma and wheezing in children, as opposed to PM10 exposure. This discrepancy may be attributable to the fact that PM2.5 particles are significantly smaller than PM10 particles, allowing them to penetrate deeper into the lungs, reaching the bronchioles and alveoli where they might induce inflammation, whereas PM10 particles remain trapped in the upper respiratory tract. PM2.5 comprise more harmful components including combustion particles, heavy metals, and organic compounds which can cause oxidative stress and inflammation in lung tissues that will increase the development of asthma [101, 111–113].

In contrast, PM10 is composed of larger, less hazardous particles like dust and pollen. Furthermore, PM2.5 can stay in the air for longer periods of time, the duration of exposure rises, increasing the likelihood of inhalation [114]. Children's respiratory systems are still developing, and they breathe more air than their body weight, making them especially exposed to the effects of PM2.5 [101]. These features, including PM2.5's deeper lung penetration, greater toxicity, and more prominent inflammatory responses, explain why it has a stronger association with the onset of asthma and wheezing in children than PM10.

Conclusion

This review found a significant association between exposure to PM and asthma and wheezing in children. Both PM10 and PM2.5 are associated with increased odds of asthma and wheezing, with PM2.5 showing a stronger relationship. The significant levels of heterogeneity observed suggest variations across studies due to differences in study designs, exposure level and outcome measurement type. Further research is required to investigate the remaining elements that contribute to variability in these associations. These findings indicate the need for strategies to reduce particle air pollution to mitigate its adverse effects on children's respiratory health.

Strength and limitation of the study

The major limitation of this review is the inconsistent classification of exposure duration and age intervals among study participants across included studies, which did not allow for meaningful subgroup analysis or meta-regression based on exposure duration and age. Standardization of age stratification and exposure time for the assessment of developmental-stage-specific susceptibility should be a priority in future studies. This study is focused on PM10 and PM2.5 exposures and does not differentiate between interactions with other pollution such as NO_2, O_3 or SO_2, restricting from interaction or synergistic effects. Multi-pollutant models are needed for a comprehensive understanding of interactions effects. The exclusion of studies with low quality, as assessed through JBI criteria, may additionally introduce systematic bias. Additionally, non-English studies were excluded, probably affecting this review comprehensiveness.

The majority of the studies used questionnaires and self-reported exposure, which introduced recall bias and increased the likelihood of misclassification. To account possible causes of heterogeneity, subgroup analyses was conducted based on regions, research design, exposure assessment methods, exposure level, outcome assessment methods and meta regression using sample size and study years. Furthermore, this systematic review and meta-analysis followed the most recent PRISMA guidelines, ensuring the review process was transparent and rigorous.

All relevant cases of respiratory outcomes (asthma and wheeze) caused by exposure to PM10 and PM2.5 were carefully evaluated. The inclusion of a diverse set of studies enriched the analysis by presenting a comprehensive picture of the respiratory health effects of PM exposure. This combination of subgroup analysis and robust methodology provides valuable insights for future study and reinforces the importance of the findings for public health interventions.

Study implications for intervention

This finding has great implications for educating parents and caregivers about reducing children's exposure to PM2.5 and PM10 and for community health programs by increasing air quality monitoring around residential areas, schools, and strengthening policies to reduce industrial and vehicular emissions. Furthermore, healthcare providers should integrate air quality data into clinical assessments to identify children at higher risk of asthma exacerbations due to particulate matter (PM) exposure. Screening tools can include questions about proximity to pollution sources, such as busy roads or industrial areas. Clinicians and public health experts should educate families on minimizing outdoor activities during high pollution days, using air purifiers, and improving home ventilation. Personalized asthma action plans, tailored to local air quality, can guide medication use during poor air quality periods, reducing emergency visits. Healthcare providers can also advocate for stronger air quality policies and engage in community initiatives, such as reducing emissions near schools. Integrating real-time air quality alerts into clinical practice enables families to take preventive measures, improving outcomes and reducing the asthma burden linked to air pollution. This study also shared robust data globally which has a significant policy implication for public health, air quality standards and interventions to protect this vulnerable group of population from the respiratory health effects of PM exposure among children and adolescents.

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Author contributions
AK conceptualized, designed, software, analyzed, interpreted the data, and drafted, wrote and edited the manuscript. AEB conceptualized, designed, software, interpreted the data, and edited the manuscript. ETA, AE, YT, GA, and YM conceptualized, software, performed writing and editing of the manuscript. All authors read and approved the final manuscript.

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Data availability
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
Ethical approval and informed consent were not applicable. However, the review protocol was registered with the International Prospective Register of Systematic Reviews (PROSPERO: CRD42024562670).

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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