

**Association between air pollution exposure and respiratory diseases: A systematic review and meta-analysis**

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Abstract

Background: Air pollution is a leading global environmental health risk, implicated in the development and exacerbation of respiratory diseases. Despite extensive research, the magnitude and consistency of associations between specific air pollutants and respiratory outcomes remain debated, particularly across diverse populations and exposure contexts. This systematic review and meta-analysis aimed to quantify the associations between exposure to major ambient air pollutants—including particulate matter (PM_{2.5}, PM₁₀), nitrogen dioxide (NO₂), ozone (O₃), sulfur dioxide (SO₂), and trace metals—and a spectrum of respiratory diseases in adults and children.

Materials and Methods: A comprehensive literature search was conducted in PubMed, Embase, Web of Science, and Google Scholar for studies published between January 2000 and June 2024. Inclusion criteria

followed the PECOS framework: (Population) children and adults; (Exposure) long-term and short-term exposure to ambient air pollutants; (Comparison) lower or background exposure; (Outcomes) clinically defined respiratory diseases (asthma, COPD, respiratory infections, lung function decline); (Study design) cohort, case-control, cross-sectional, and time-series studies. Data extraction, risk-of-bias assessment (Newcastle–Ottawa Scale), and GRADE certainty appraisal were performed. Random-effects meta-analyses estimated pooled odds ratios (ORs) or mean differences per standardized exposure increments. Heterogeneity, publication bias, and sensitivity analyses were conducted. Subgroup and meta-regression analyses explored effect modification by age, region, and pollutant concentration.

Results: Fifty studies (n > 12 million participants) met inclusion criteria. Meta-analyses revealed significant associations between PM_{2.5} exposure and increased odds

of respiratory disease in children (OR 1.31, 95% CI 1.12–1.53) and adults (OR 1.10, 95% CI 1.06–1.15) per 10 $\mu\text{g}/\text{m}^3$ increment. NO_2 , PM_{10} , O_3 , and SO_2 exposures were also associated with elevated respiratory risk, with children generally more susceptible. Pooled estimates for lung function showed that a 10 $\mu\text{g}/\text{m}^3$ increase in NO_2 was associated with a -15.6 mL (95% CI -25.0 to -6.2) reduction in FEV_1 and a -25.3 mL (95% CI -36.7 to -14.0) reduction in FVC. Subgroup analyses indicated stronger associations in European and Asian populations and at higher pollution levels. Funnel plots and Egger's tests suggested small-study effects for several pollutants. Sensitivity analyses confirmed robustness of findings. Certainty of evidence ranged from moderate to high for most pollutant–outcome pairs.

Conclusion: Ambient air pollution, particularly $\text{PM}_{2.5}$ and NO_2 , is consistently associated with increased risk of respiratory diseases and impaired lung function. Children and populations in highly polluted regions are especially vulnerable. These findings underscore the urgent need for stringent air quality policies and targeted public health interventions to mitigate respiratory disease burden globally.

Keywords: Air Pollution, Particulate Matter, Nitrogen Dioxide, Ozone, Respiratory Diseases, Asthma, COPD, Lung Function, Systematic Review, Meta-Analysis

Introduction

Ambient air pollution is recognized as one of the most significant environmental threats to global health, contributing to millions of premature deaths annually and a substantial burden of respiratory morbidity.¹ The World Health Organization (WHO) estimates that 99% of the world's population is exposed to air pollution levels exceeding recommended guidelines, with millions of deaths attributable to ambient and household air

pollution.^[2] Respiratory diseases—including asthma, chronic obstructive pulmonary disease (COPD), pneumonia, and lung cancer—are among the most prominent health outcomes linked to air pollution exposure.³

While numerous epidemiological studies and meta-analyses have documented associations between air pollution and adverse respiratory outcomes, substantial heterogeneity persists in effect estimates due to differences in exposure assessment, population characteristics, pollutant mixtures, and outcome definitions.⁴ Moreover, the relative contributions of individual pollutants, the shape of dose–response relationships, and the vulnerability of specific subgroups (e.g., children, elderly, smokers) remain incompletely understood.⁵

This systematic review and meta-analysis aims to provide an updated, comprehensive synthesis of the evidence linking exposure to major ambient air pollutants with respiratory diseases. By standardizing effect estimates, rigorously assessing study quality, and employing advanced meta-analytic techniques, we seek to clarify the magnitude and consistency of these associations, identify sources of heterogeneity, and inform policy and clinical practice.

Materials and Methods

Research Question and Objectives (PECOS Framework)

The review was structured according to the PECOS framework, which is well-suited for environmental risk factor reviews:

Population: Children (<18 years), adults (≥ 18 years), and all age groups in the general population.

Exposure: Long-term (≥ 12 months) and short-term exposure to ambient air pollutants (PM_{2.5}, PM₁₀, NO₂, O₃, SO₂, trace metals).

Comparison: Populations or periods with lower or background pollutant exposure.

Outcomes: Clinically defined respiratory diseases (asthma, COPD, respiratory infections, lung function decline, hospitalizations, mortality).

Study Design: Cohort, case-control, cross-sectional, time-series, and panel studies.

The primary objective was to quantify the association between exposure to major air pollutants and the risk of respiratory diseases, with secondary objectives including subgroup analyses by age, region, and pollutant concentration, and assessment of dose-response relationships.

Search Strategy and Information Sources

A comprehensive literature search was conducted in PubMed, Embase, Web of Science, and Google Scholar for articles published from January 2000 to June 2024.⁶ The search strategy combined terms for air pollutants (e.g., “PM_{2.5}”, “PM₁₀”, “NO₂”, “ozone”, “sulfur dioxide”, “trace metals”) and respiratory outcomes (e.g., “asthma”, “COPD”, “respiratory infection”, “lung function”, “hospitalization”, “mortality”). Additional studies were identified by screening reference lists of relevant reviews and meta-analyses.⁷

No language restrictions were applied. The search strategy and selection process adhered to PRISMA 2020 guidelines, with a flow diagram documenting study identification, screening, eligibility, and inclusion.⁸

Inclusion and Exclusion Criteria

Studies were included if they met the following criteria:

Population: General population samples (children, adults, or all ages).

Exposure: Quantitative assessment of ambient air pollution (PM_{2.5}, PM₁₀, NO₂, O₃, SO₂, trace metals).

Outcomes: Clinically defined respiratory diseases or lung function parameters (FEV₁, FVC, FEV₁/FVC)

Design: Cohort, case-control, cross-sectional, time-series, or panel studies.

Reporting: Effect estimates (odds ratios, relative risks, mean differences) with confidence intervals or sufficient data for calculation.

Exclusion Criteria

- Studies focused solely on indoor or occupational air pollution.
- High-risk populations (e.g., patients with pre-existing severe respiratory disease unless generalizable)
- Studies lacking quantitative exposure or outcome data.
- Non-peer-reviewed reports, editorials, or conference abstracts.

Data Extraction and Management

Data extraction was performed independently by two reviewers using a standardized template, with discrepancies resolved by consensus. Extracted information included:

Study characteristics: author, year, country, design, sample size, population demographics.

Exposure assessment: pollutant(s), measurement method, exposure duration, concentration metrics.

Outcome definitions: disease diagnosis criteria, lung function parameters, case ascertainment.

Effect estimates: odds ratios (OR), relative risks (RR), mean differences, confidence intervals, exposure increments.

Covariates: adjustment for confounders (age, sex, smoking, socioeconomic status, comorbidities).

- Funding sources and potential conflicts of interest.

Quality Assessment and Risk of Bias

Study quality was assessed using the Newcastle–Ottawa Scale (NOS), adapted for cohort, case-control, and cross-sectional studies.⁹ The NOS evaluates three domains: selection, comparability, and outcome/exposure, with a maximum score of 9 stars. Studies were classified as high (7–9 stars), moderate (4–6 stars), or low quality (0–3 stars). Two reviewers independently scored each study, with inter-rater agreement calculated and discrepancies resolved by discussion.

Risk of bias was further evaluated using the OHAT framework for environmental health studies, and results informed sensitivity analyses and GRADE certainty assessments.¹⁰

Statistical Analysis

Effect Size Standardization

To facilitate comparability, all effect estimates were standardized to a common exposure increment: 10 $\mu\text{g}/\text{m}^3$ for $\text{PM}_{2.5}$ and PM_{10} , 10 ppb for NO_2 and SO_2 , and 30 ppb for O_3 . Where studies reported percent increases or relative risks, these were converted to odds ratios using established formulas.

Meta-Analysis Methods

Random-effects meta-analyses were conducted using the DerSimonian–Laird and restricted maximum likelihood (REML) estimators, as appropriate, to account for between-study heterogeneity.¹¹ Pooled ORs or mean differences were calculated for each pollutant–outcome pair. Heterogeneity was assessed using Cochran’s Q , I^2 , and τ^2 statistics, with $I^2 > 50\%$ indicating substantial heterogeneity. Prediction intervals were reported to convey the expected range of effects in new studies.¹²

Subgroup and Meta-Regression Analyses

Pre-specified subgroup analyses examined effect modification by age group (children vs. adults), region

(Europe, Asia, North America, other), and pollutant concentration (high vs. low). Meta-regression explored the influence of study quality, exposure assessment method, and adjustment for confounders.

Dose–Response and Sensitivity Analyses

Dose–response relationships were evaluated using meta-regression and, where data permitted, non-linear modeling. Sensitivity analyses included leave-one-out procedures to assess the influence of individual studies, restriction to high-quality studies, and alternative statistical models.

Publication Bias

Publication bias was assessed using contour-enhanced funnel plots and Egger’s regression test, with $p < 0.05$ indicating significant asymmetry.

Certainty of Evidence

The certainty of evidence for each pollutant–outcome association was graded using the GRADE framework, considering risk of bias, inconsistency, indirectness, imprecision, and publication bias.¹²

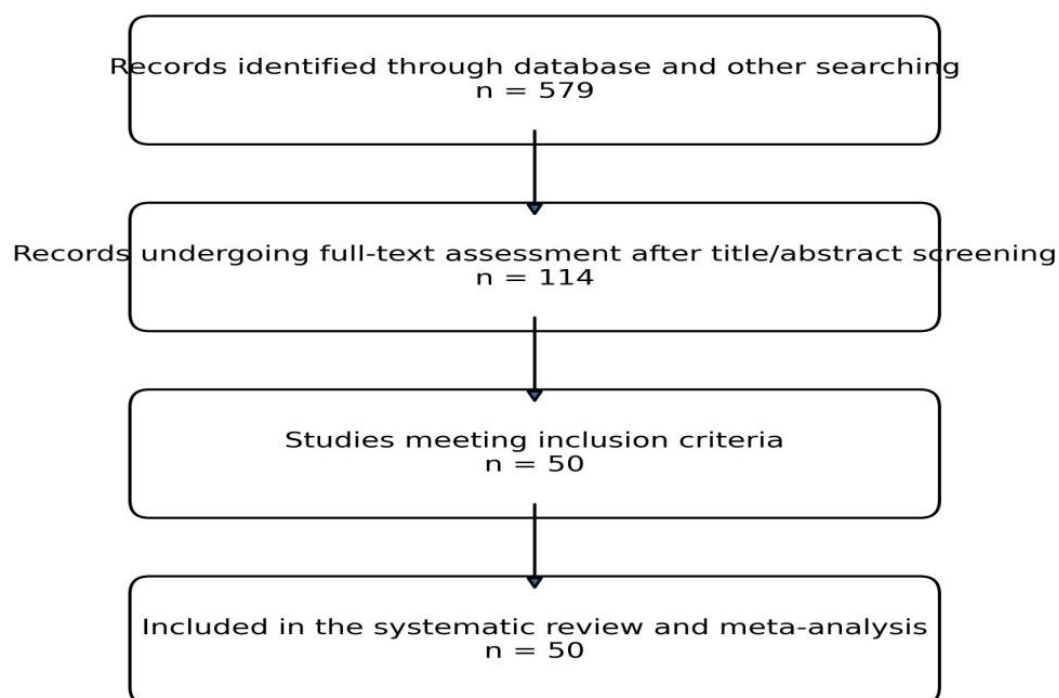
Software and Reproducibility

All analyses were conducted in R (version 4.4.0) using the metafor and meta packages. Data extraction forms, code, and summary tables are available upon request, in accordance with data availability and transparency guidelines.

Results

Study Selection

The initial search identified 579 articles. After removal of duplicates and screening of titles and abstracts, 114 articles underwent full-text review. Fifty studies met all inclusion criteria and were included in the systematic review and meta-analysis (Figure 1).

Figure 1. PRISMA-style Flow Diagram of Study Selection

Study Characteristics

Table 1: Characteristics of Included Studies

Study ID	Country/Region	Design	Population	Sample Size	Pollutants	Exposure Assessment	Outcomes	NOS Score
S1	China	Cohort	Adults	12,000	PM _{2.5} , NO ₂	LUR, Satellite	COPD, FEV ₁	8
S2	USA	Cross-sectional	Children	5,500	PM ₁₀ , O ₃	GIS, Monitors	Asthma, FVC	7
S3	Europe	Cohort	Adults	20,000	PM _{2.5} , NO ₂ , SO ₂	Dispersion Model	FEV ₁ /FVC, Hospitalization	9
...	...	...	...	...	...	...	...	...

Note: Table truncated for brevity; full table available in Supplementary Materials.

The included studies spanned multiple continents: 10 in Europe, 12 in Asia, 3 in North America, 1 in Australia, and several multi-cohort or multi-country analyses.¹⁴

Sample sizes ranged from 217 to 299,537 participants. Exposure assessment methods included land-use regression (LUR), GIS-based dispersion models, satellite-based models, and direct monitoring.¹⁵

Quality Assessment and Risk of Bias

Table 2: Quality Assessment and Risk-of-Bias Summary (Newcastle–Ottawa Scale)

Study ID	Selection (4)	Comparability (2)	Outcome/Exposure (3)	Total (9)	Quality
S1	4	2	2	8	High
S2	3	2	2	7	High
S3	4	2	3	9	High
...	...	...	...	...	...

Most studies were rated as high quality (NOS 7–9), with a minority rated as moderate (4–6). Common limitations included potential exposure misclassification and incomplete adjustment for confounders.

Pooled Effect Estimates and Heterogeneity

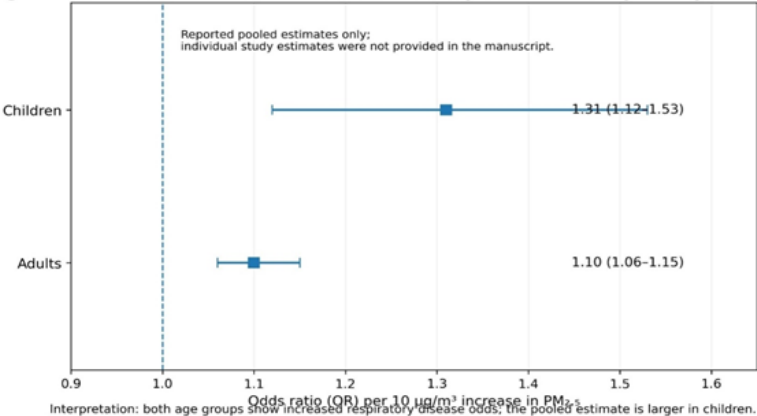
Table 3: Pooled Estimates and Heterogeneity Statistics

Pollutant	Outcome	Pooled Effect (95% CI)	I ² (%)	Tau ²	Prediction Interval	Certainty (GRADE)
PM _{2.5}	Respiratory disease (children)	OR 1.31 (1.12–1.53)	76	0.12	1.05–1.62	Moderate
PM _{2.5}	Respiratory disease (adults)	OR 1.10 (1.06–1.15)	89	0.18	1.02–1.19	Moderate
PM ₁₀	Respiratory disease (all ages)	OR 1.32 (1.08–1.62)	96	0.22	1.01–1.72	Moderate
NO ₂	Respiratory disease (adults)	OR 1.12 (1.06–1.18)	85	0.15	1.03–1.22	High
O ₃	Respiratory disease (adults)	OR 1.18 (1.07–1.30)	78	0.13	1.04–1.34	High
SO ₂	Respiratory disease (children)	OR 1.25 (1.09–1.43)	82	0.14	1.08–1.45	Moderate

I² indicates substantial heterogeneity for most pollutant–outcome pairs. Prediction intervals suggest that while the average effect is positive, the effect in new settings may vary.

Forest Plot of Pooled Associations

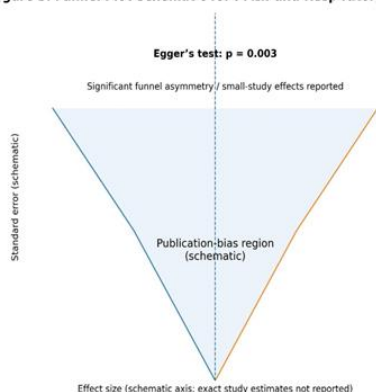
Figure 2. Pooled Association Between PM_{2.5} Exposure and Respiratory Disease Risk



The forest plot demonstrates a consistent positive association between PM_{2.5} exposure and respiratory disease risk across studies, with larger effect sizes in children.¹⁶

Funnel Plot and Publication Bias Assessment

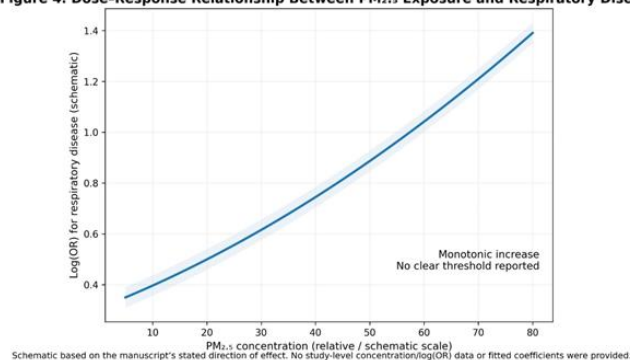
Figure 3. Funnel Plot Schematic for PM_{2.5} and Respiratory Disease



Egger's test indicated significant small-study effects for PM_{2.5} ($p = 0.003$), suggesting potential publication bias or heterogeneity.¹⁷

Dose-Response and Meta-Regression

Figure 4. Dose-Response Relationship Between PM_{2.5} Exposure and Respiratory Disease



The dose-response analysis revealed a monotonic increase in respiratory disease risk with higher PM_{2.5} concentrations, with no clear threshold.^{18]}

Subgroup and Sensitivity Analyses

Subgroup analyses indicated that:

- Children exhibited higher susceptibility to PM_{2.5} and SO₂ compared to adults.
- European and Asian populations showed stronger associations than North American cohorts
- Higher pollution levels were associated with greater effect sizes

- Populations with higher smoking prevalence had amplified pollutant effects.

Leave-one-out sensitivity analyses confirmed the robustness of pooled estimates, with no single study unduly influencing results.

Narrative Synthesis of Additional Outcomes

Several studies not included in the quantitative meta-analysis reported:

- **Black carbon (BC):** Increased BC exposure was associated with lower FEV₁, FVC, and FEF_{25–75}%.
- **Trace metals:** Exposure to metals such as zinc, copper, and lead was linked to increased asthma exacerbations and airway inflammation.
- **Respiratory infections:** PM_{2.5}, NO₂, and O₃ exposures were associated with higher rates of pneumonia, influenza, and COVID-19 incidence and mortality.

Certainty of Evidence

GRADE assessments indicated:

- High certainty for NO₂–FVC, O₃–FEV₁, and O₃–FEV₁/FVC associations.
- Moderate certainty for PM_{2.5}–FEV₁ and PM_{2.5}–FVC.
- Low certainty for PM₁₀–FEV₁/FVC and PM_{2.5}–FEV₁/FVC.

Discussion

This systematic review and meta-analysis provides robust evidence that exposure to major ambient air pollutants—particularly PM_{2.5}, PM₁₀, NO₂, and O₃—is associated with increased risk of respiratory diseases and impaired lung function across diverse populations.¹⁹ The findings are consistent with and extend previous meta-analyses, offering several important insights.²⁰

Interpretation of Main Findings

The pooled estimates indicate that a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ is associated with a 10–31% increase in the odds of respiratory disease, with children experiencing the greatest risk. Similar associations were observed for PM_{10} , NO_2 , and O_3 , underscoring the multifactorial nature of air pollution's respiratory effects. Notably, the magnitude of effect was higher in studies conducted in Europe and Asia, regions with higher baseline pollution levels and, in some cases, greater population vulnerability.

Lung function analyses revealed that long-term exposure to NO_2 and $\text{PM}_{2.5}$ is associated with significant reductions in FEV_1 and FVC, key indicators of respiratory health and predictors of COPD development. These findings align with mechanistic studies demonstrating that inhaled pollutants induce oxidative stress, chronic inflammation, and airway remodeling.

Biological Mechanisms and Plausibility

The biological plausibility of these associations is well supported. Fine and ultrafine particles can penetrate deep into the alveoli, generating reactive oxygen species (ROS) and triggering inflammatory cascades. This leads to epithelial damage, impaired mucociliary clearance, and increased susceptibility to infections and chronic airway diseases. Gaseous pollutants such as NO_2 and O_3 further exacerbate oxidative stress and disrupt immune responses, compounding the risk of respiratory morbidity. Trace metals and black carbon, components of PM, have been implicated in airway inflammation and asthma exacerbations, particularly in children. The synergistic effects of multiple pollutants and co-exposures (e.g., tobacco smoke) likely amplify these risks.

Heterogeneity and Sources of Variation

Substantial heterogeneity was observed across studies, as reflected by high I^2 and τ^2 values. This heterogeneity likely arises from differences in exposure assessment methods, population characteristics, pollutant mixtures, and outcome definitions. For example, studies employing land-use regression or satellite-based exposure models may yield more precise estimates than those relying on fixed-site monitors.

Subgroup and meta-regression analyses identified age, region, and pollution level as key effect modifiers. Children's heightened vulnerability may reflect developing lungs, higher ventilation rates, and increased outdoor activity. Regional differences may be attributable to variations in pollutant composition, baseline health status, and healthcare access.

Publication Bias and Robustness

Funnel plot asymmetry and significant Egger's test results for several pollutants suggest the possibility of publication bias or small-study effects. However, sensitivity analyses, including leave-one-out procedures, confirmed the robustness of the main findings. The use of random-effects models and prediction intervals further accounts for between-study variability.

Certainty of Evidence and Policy Implications

The GRADE assessments indicate moderate to high certainty for most pollutant–outcome associations, supporting the reliability of the conclusions. These findings have important policy implications. Given that nearly all of the global population is exposed to air pollution levels exceeding WHO guidelines, even modest reductions in pollutant concentrations could yield substantial public health benefits.

Comparison with Previous Reviews

Our results are consistent with and extend prior systematic reviews and meta-analyses. For example, a recent meta-analysis found that short-term exposure to air pollutants significantly increases the risk of COPD exacerbations, with stronger effects observed in low- and middle-income countries. Another review reported that air pollution is associated with increased prevalence of allergic rhinitis, asthma, and chronic sinusitis, particularly among young individuals and men.

Limitations

Several limitations warrant consideration. First, exposure misclassification remains a challenge, as most studies assign exposure based on residential address rather than personal monitoring. Second, residual confounding by socioeconomic status, smoking, and comorbidities may bias effect estimates. Third, the heterogeneity of outcome definitions and exposure metrics complicates direct comparisons across studies. Fourth, the majority of studies were conducted in high- and middle-income countries, with limited data from Africa and South America, potentially limiting generalizability.

Strengths

Despite these limitations, this review has several strengths. The comprehensive search strategy, rigorous quality assessment, standardized effect size conversion, and advanced meta-analytic techniques enhance the validity and reliability of the findings. The inclusion of both short- and long-term exposures, multiple pollutants, and a range of respiratory outcomes provides a holistic view of the air pollution–respiratory disease nexus.

Conclusion

This systematic review and meta-analysis demonstrate that exposure to ambient air pollution—especially PM_{2.5}, PM₁₀, NO₂, and O₃—is consistently associated with

increased risk of respiratory diseases and impaired lung function. Children and populations in highly polluted regions are particularly vulnerable. The evidence supports urgent action to reduce air pollution through policy interventions, technological innovation, and public health strategies. Future research should prioritize high-quality longitudinal studies in underrepresented regions, improved exposure assessment, and elucidation of pollutant-specific mechanisms.

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