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Review Article

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A global meta-analysis of particulate and gaseous air pollutants in relation to COVID-19 mortality and hospitalization

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ABSTRACT

A growing body of evidence implicates ambient air pollution in the exacerbation of clinical outcomes after SARS-CoV-2 infection. To synthesize this evidence, we performed a global systematic review and meta-analysis to precisely quantify the associations between exposure to specific atmospheric contaminants and the subsequent risks of COVID-19-related mortality and hospital admission.

Our methodology adhered to the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) framework, involving a comprehensive search of scientific databases for literature published until the end of August 2025. From this search, 44 publications were deemed eligible for inclusion. We employed random-effects models to compute summary Risk Ratios (RRs) representing the change in health risk per 1 µg/m³ increment in atmospheric pollutant concentration. Our findings indicate that long term exposure to fine Particulate Matter ($PM_{2.5}$), coarse Particles (PM_{10}), Nitrogen dioxide (NO_2), and Sulfur dioxide (SO_2) significantly increased the likelihood of fatal outcomes from COVID-19.

The respective pooled RRs were 1.046 (95% CI: 1.031–1.062), 1.079 (95% CI: 1.005–1.154), 1.017 (95% CI: 1.004–1.029), and 1.077 (95% CI: 1.021–1.133). Acute exposures to ambient $PM_{2.5}$ and NO_2 concentrations were similarly associated with increased mortality, demonstrating risk ratios of 1.043 (95% CI: 1.033–1.053) and 1.033 (95% CI: 1.019–1.048) respectively per 1 µg/m³ increment. Additionally, both acute and chronic exposures to $PM_{2.5}$, PM_{10}, and NO_2 showed significant associations with higher COVID-19 hospitalization rates.

This meta-analysis provides robust quantitative suggestion that ambient $PM_{2.5}$, $PM_{10,}$ NO_2, and SO_2 are significant and modifiable risk factors for severe COVID-19 outcomes. These results emphasize the critical need for enhanced air quality standards as a fundamental element of public health policy to alleviate the impact of COVID-19 and bolster defenses against forthcoming respiratory epidemics.

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Review

Ambient air pollution remains one of the most significant environmental determinants of global morbidity and mortality, presenting a major challenge to public health systems worldwide [1]. Exposure to ambient pollutants is a recognized risk factor for various infectious and respiratory diseases [2, 3]. Particulate Matter (PM)—especially coarse (PM_{10}) and fine ($PM_{2.5}$) fractions—represents a major threat to population health due to its capacity for deep respiratory penetration [4]. Epidemiological studies have consistently linked PM exposure to pathologies including asthma, chronic obstructive pulmonary disease, and neurological dysfunctions [5, 6]. The Global Burden of Disease report attributed 6.67 million premature deaths to PM exposure. Chronic exposure to $PM_{2.5}$ contributes to a measurable decline in life expectancy, a consequence largely driven by its established role in elevating the incidence of respiratory illness, cardiovascular disease, and specific cancers [7-9]. This considerable public health burden is further compounded by other hazardous agents, including Sulfur dioxide (SO_2), Carbon monoxide (CO), Nitrogen dioxide (NO_2), and Polycyclic Aromatic Hydrocarbons (PAHs) [10].

Beyond its established effects on chronic diseases, accumulating evidence indicates that air pollution may influence the transmission and severity of infections via immunological and cellular mechanisms [11]. Chronic exposure appears to compromise immune function and alter host responses to pathogens [12]. Furthermore, fine aerosols can act as viral vectors, facilitating pathogen penetration into epithelial cells and potentially exacerbating respiratory tract infections [13]. Taken together, these mechanisms offer plausible explanations for epidemiological findings linking air pollution exposure with increased susceptibility to viral and bacterial respiratory infections [14], beyond its established impact on cardiopulmonary morbidity and mortality [15].

The COVID-19 pandemic offers a critical case study of this interaction. Caused by SARS-CoV-2, it has resulted in over 6.5 million reported deaths globally, placing extraordinary strain on healthcare systems and economies [16, 17]. While person-to-person contact is the primary transmission route, respiratory aerosol droplets (<5 μm) play a key role in airborne spread [18]. Several biological mechanisms are thought to explain how air pollution exacerbates severity of COVID-19. The presence of particulate in the respiratory system promotes the generation of hydroxyl radicals, a process that subsequently triggers the release of pro-inflammatory signaling molecules, including interleukin-6 (IL-6). This cascade of inflammatory activity can increase an individual's susceptibility to more severe disease manifestations [19, 20]. Laboratory investigations further indicate that exposure to $PM_{2.5}$ induces an overexpression of ACE2. As this transmembrane receptor functions as the principal binding site for SARS-CoV-2 viral entry, its elevated expression could potentially enhance cellular susceptibility to infection [21].

Consequently, numerous epidemiological investigations have investigated the relationships between specific pollutants ($PM_{2.5}$, PM_{10}, NO_2, SO_2) and COVID-19 outcomes [22, 23]. Their findings, however, show substantial heterogeneity, reporting diverse effect estimates—including Odds Ratios (OR), Rate Ratios (RR), and regression coefficients with 95% Confidence Intervals (CI) [24-27]. This heterogeneity largely

stems from methodological differences, particularly in confounder adjustment for socioeconomic factors [28], and limitations in exposure assessment, such as the use of aggregate data that may not reflect individual-level exposures [29]. Regional variations in pollution profiles, healthcare infrastructure, and pandemic dynamics further complicate these associations.

Given these inconsistencies, synthesizing global evidence through a comprehensive meta-analysis is essential. Accordingly, this investigation was designed to synthesize epidemiological data through a meta-analysis, with the primary objective of deriving precise, pooled effect estimates for the connection between critical air pollutants and patient outcomes of COVID-19 mortality and hospitalization. With its comprehensive scope, methodological transparency, and analytical rigor, this study provides evidence-based insights to guide targeted public health strategies, shape environmental policy, and strengthen pandemic preparedness [30].

Definitions of terms

To ensure clarity and consistency throughout the manuscript, the following abbreviations and technical terms are defined: ACE2: Angiotensin-Converting Enzyme 2; CO: Carbon Monoxide; COVID-19: Coronavirus Disease 2019; IL-6: Interleukin-6; IL-8: Interleukin-8; NO_2: Nitrogen Dioxide; NOQAS: Newcastle-Ottawa Quality Assessment Scale; OHAT: Office of Health Assessment and Translation; OR: Odds Ratio; PAHs: Polycyclic Aromatic Hydrocarbons; $PM_{2.5}$: Fine Particulate Matter (aerodynamic diameter ≤2.5 μm); PM_{10}: Coarse Particulate Matter (aerodynamic diameter ≤10 μm); PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses; SO_2: Sulfur Dioxide.

Protocol and registration

This research was performed as a systematic review and meta-analysis, following the methodological standards outlined in the PRISMA statement. The analytical protocol received prospective registration in the PROSPERO international database under identifier CRD42022336143.

Literature search and study selection

We performed an extensive literature retrieval across four electronic databases—Scopus, PubMed, Web of Science, and Google Scholar—to recognize relevant articles published before September 1, 2025. The search syntax integrated controlled vocabulary, including MeSH terms, and keywords pertaining to two core concepts: atmospheric pollutants (e.g., "nitrogen dioxide," "$PM_{2.5}$") and severe COVID-19 sequelae (e.g., "hospital admission," "fatality"). These concepts were logically combined using AND/OR Boolean operators. To minimize the risk of omitting pertinent research, we also manually examined the bibliographies of all selected articles.

Two reviewers conducted the study selection process, which involved a sequential assessment of titles, abstracts, and finally, the full-text manuscripts. Studies were included if they were original, peer-reviewed epidemiological reports providing an adjusted quantitative estimate—such as an Hazard Ratio (HR), Odds Ratio (OR), or Rate Ratio (RR) accompanied by a 95% confidence interval (CI)—for the association between community-level air pollution and either COVID-19-associated mortality or hospitalization. We excluded non-English publications, reviews, editorials, conference abstracts, and studies that focused exclusively on indoor air pollution or tobacco smoke.

Data extraction

Two investigators (M.M. and B.K.) independently employed a standardized protocol to systematically extract relevant data from all included studies. The data collection protocol captured multiple study characteristics including: primary author identification, publication year, geographical location, research methodology, cohort dimensions, exposure quantification techniques, targeted air contaminants, reported effect estimates with corresponding 95% confidence intervals, and key covariates incorporated into the analytical models.

The methodological rigor and potential for bias in the included studies were evaluated by two tools: the Office of Health Assessment and Translation (OHAT) framework and the Newcastle-Ottawa Scale (NOS). The OHAT tool facilitated a domain-based evaluation, rating potential bias in areas such as participant selection, exposure determination, and control for confounding as "low," "probably low," "probably high," "high," or "not applicable." Concurrently, the NOS was applied to gauge study quality across three core domains: study group selection (capped at 4 stars), intergroup comparability (capped at 2 stars), and outcome verification (capped at 3 stars). A study's total NOS score determined its quality classification: ≤4 for low, 5–6 for moderate, and ≥7 for high quality. Any disagreements arising during the screening, data extraction, or appraisal phases were reconciled through discussion between the reviewers, with unresolved issues adjudicated by a third researcher.

Statistical synthesis

Pooled Risk Ratios (RRs) with 95% Confidence Intervals (CIs) were calculated to estimate the change in health risk associated with each 1 μg/m³ increment in atmospheric pollutant concentration. To accommodate expected methodological and clinical variation among the included studies, the DerSimonian and Laird random-effects model served as the primary analytical framework for pooling data. For comparative purposes, a fixed-effect model was also implemented. To harmonize effect measures, ORs and HRs were converted to RRs using established formulas [31], with baseline outcome rates sourced from the Human Mortality Database where required [32]. For studies that reported effect estimates for increments other than 1 μg/m³, the estimates were standardized using a validated methodology [33].

The degree of inconsistency in effect sizes among the incorporated investigations, referred to as between-study heterogeneity, underwent formal statistical assessment. This assessment employed the I^2 index, measuring the proportion of total variance attributable to systematic heterogeneity rather than sampling error, alongside Cochran's Q test for statistical significance. An I^2 value exceeding 70%, coupled with a Q-test p-value below 0.10, was interpreted as indicating considerable heterogeneity. To investigate the potential for publication bias, we employed both visual and statistical methods. Visual evaluation of potential bias was conducted through funnel plot symmetry analysis, while quantitative assessment employed both Egger's linear regression method and Begg's rank correlation procedure; for these tests, a p-value of less than 0.10 was considered indicative of potential bias. The influence of any potentially missing studies on the overall results was estimated using the trim-and-fill method. The stability and reliability of the pooled estimates were further examined through a leave-one-out sensitivity analysis, which systematically recalculates the summary estimate after omitting one study at a time. All statistical computations were

performed using STATA 15 and R statistical environment 4.4.2.

Results

Study identification and characteristics

Our systematic search of electronic databases yielded an initial 2,262 records, supplemented by 12 further publications identified through manual review of references. After the removal of duplicate entries, 1,162 unique citations underwent preliminary evaluation based on their titles and abstracts. From this, 125 articles were selected for a comprehensive full-text evaluation, of which 44 studies ultimately satisfied all pre-defined criteria for inclusion in the quantitative synthesis. A complete depiction of the selection workflow, including the specific reasons for excluding studies at the full-text stage, is showed in the PRISMA diagram (Fig. 1).

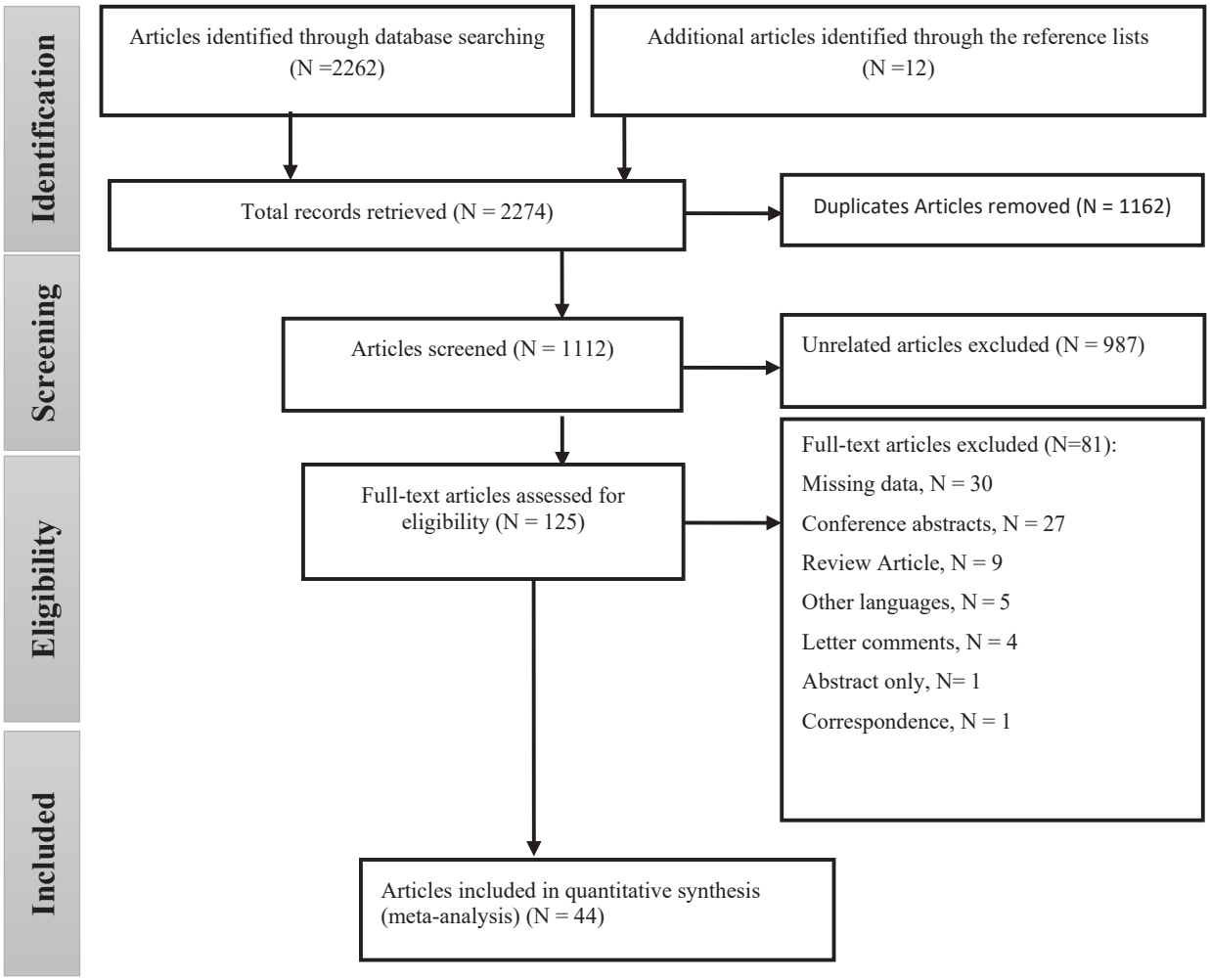


Fig. 1. Selection and summarizing of the studies

The 44 included studies, published between 2019 and 2022, encompassed 9,036,240 COVID-19 cases and 2,204,178 deaths across 95 countries. Geographically, the studies were distributed as follows: North America (n=12), South America (n=12), Asia (n=13), and Europe (n=15), with one global study spanning 730 regions in 63 countries [34] (Fig. 2a). This highlights a substantial geographic evidence gap, as no studies were identified from Africa, Russia, or Australia.

Study designs were predominantly ecological or time-series (n=29), with eight cohort studies and seven cross-sectional studies (Fig. 2b–c). Short-term and long-term exposures were defined in the original studies, typically as ≤14 days and >30 days, reflecting common epidemiological conventions. Most studies were conducted in urban or industrial regions, representing populations with elevated baseline exposures and increased susceptibility to air pollution. $PM_{2.5}$ was the most frequently investigated pollutant, followed by PM_{10}, NO_2, O_3, SO_2, and CO (Table 1). The baseline concentrations of the principal air pollutants varied considerably across the geographic regions represented in the included studies (Fig. 3). Median levels of $PM_{2.5}$ were highest in studies from Asia, while levels of NO_2 were more uniformly distributed across North America, Europe, and Asia. The distributions of PM_{10} and SO_2 also showed significant regional heterogeneity, with the latter exhibiting the most pronounced variability and several outlier regions. Most investigations incorporated statistical adjustments for crucial confounding variables such as patient age, biological sex, and pre-existing medical conditions.

Table 1. Details of the included studies in the systematic review

#	Study	Location	Outcome	Type	Exposure	Pollutants	Effect (NOS)	Ref.
1	Travaglio et al., 2021	England	COVID-19 Mort./Infect.	Time Series	Short-term	NO, NO_2, O_3, $PM_{2.5}$, PM_{10}	RR (7)	[35]
2	Sanchez-Piedra et al., 2021	Spain	COVID-19 Mort.	Time Series	Short-term	NO_2, $PM_{2.5}$	OR (7)	[36]
3	Rodriguez-Villamizar et al., 2021	Colombia	COVID-19 Mort.	Ecological	Long-term	$PM_{2.5}$	RR (6)	[25]
4	Mendy et al., 2021	USA	COVID-19 Hosp.	Ecological	Long-term	$PM_{2.5}$	OR (5)	[37]
5	Konstantinoudis et al., 2021	England	COVID-19 Mort.	Ecological	Long-term	$PM_{2.5}$, NO_2	RR (8)	[38]
6	Bowe et al., 2021	USA	COVID-19 Hosp.	Cohort	Long-term	$PM_{2.5}$	RR (6)	[39]
7	Tchicaya et al., 2021	France	COVID-19 Mort.	Cross-sectional	Long-term	$PM_{2.5}$	RR (6)	[40]
8	Zheng et al., 2021b	China	Cases, Hosp.	Time Series	Long-term	NO_2, $PM_{2.5}$ PM_{10}	RR (8)	[41]
9	Norouzi et al., 2022	Iran	COVID-19 Mort.	Ecological	Short-term	NO_2, $PM_{2.5}$	RR (7)	[42]
10	Hutter et al., 2020	Austria	COVID-19 Mort.	Cohort	Short-term	NO_2, PM_{10}	HR (6)	[43]
11	Wu et al., 2020b	USA	COVID-19 Mort.	Ecological	Long-term	$PM_{2.5}$	RR (7)	[26]
12	Bozack et al., 2022	USA	Mort., ICU, Intub.	Cohort	Long-term	$PM_{2.5}$, NO_2	RR (7)	[44]
13	Marquès et al., 2022	Spain	Mort., Severity	Cohort	Long-term	PM_{10}, NO_2, NO_2	OR (8)	[45]
14	Garcia et al., 2022	USA	COVID-19 Mort.	Ecological	Long-term	$PM_{2.5}$, PM_{10}, NO_2, O_3	RR (8)	[46]

Table 1. Continued

#	Study	Location	Outcome	Type	Exposure	Pollutants	Effect (NOS)	Ref.
15	Scalsky et al., 2022	UK	COVID-19 Infect.	Cohort	Long-term	$PM_{2.5}$, PM_{10}, NOx, NO_2, NO	OR (7)	[47]
16	Aloisi et al., 2022	Italy	COVID-19 Mort.	Ecological	Short-term	$PM_{2.5}$	RR (8)	[48]
17	Stieb et al., 2020	Canada	Hosp. & Incidence	Ecological	Long-term	$PM_{2.5}$	RR (6)	[49]
18	Zhu et al., 2020	China	COVID-19 Infect.	Time Series	Short-term	$PM_{2.5}$, PM_{10}, NO_2, O_3, SO_2, CO	RR (9)	[50]
19	De Angelis et al., 2021	Italy	Incidence & Mort.	Ecological	Long-term	$PM_{2.5}$, PM_{10}, NO_2	RR (9)	[27]
20	Dales et al., 2021	Chile	COVID-19 Mort.	Time-series	Short-term	CO, NO_2, $PM_{2.5}$, O_3	RR (9)	[51]
21	Ibarra-Espinosa et al., 2022	Brazil	Cases & Deaths	Time-series	Short-term	$PM_{2.5}$, O_3	RR (8)	[52]
22	Yao et al., 2020b	China	Case Fatality	Cross-sectional	Long-term	$PM_{2.5}$, PM_{10}	RR (7)	[53]
23	Xu et al., 2022	USA	COVID-19 Infect.	Ecological	Short-term	$PM_{2.5}$, O_3	RR (6)	[54]
24	Becchetti et al., 2022	Italy	COVID-19 Deaths	Cross-sectional	Long-term	$PM_{2.5}$, PM_{10}	RR (6)	[55]
25	Meo et al., 2021	UK	Incidence & Mort.	Time-series	Short-term	$PM_{2.5}$, CO, O_3	RR (5)	[56]
26	Liang et al., 2020	USA	Fatality & Mort. Rate	Cross-sectional	Long-term	NO_2, $PM_{2.5}$, O_3	RR (6)	[57]
27	Tian et al., 2021	China	Case Fatality	Cohort	Short-term	$PM_{2.5}$, PM_{10}, O_3, NO_2, SO_2	HR (7)	[58]
28	Veronesi et al., 2022	Italy	COVID-19 Cases	Time Series	Long-term	$PM_{2.5}$, PM_{10}, NO_2, NO, O_3	RR (7)	[59]
29	Wang et al., 2020	China	COVID-19 Hosp.	Time Series	Short-term	$PM_{2.5}$, PM_{10}	RR (6)	[60]
30	Coker et al., 2020	Italy	COVID-19 Mort.	Ecological	Long-term	$PM_{2.5}$	RR (7)	[61]
31	Zhang et al., 2021	China	Confirmed Cases	Time Series	Short-term	CO, NO_2, O_3, $PM_{2.5}$, PM_{10}, SO_2	RR (8)	[62]
32	Jiang et al., 2020	China	COVID-19 Incidence	Cohort	Short-term	$PM_{2.5}$, PM_{10}, SO_2, CO, NO_2, O_3	RR (7)	[23]
33	Wu et al., 2020d	USA	COVID-19 Mort.	Cross-sectional	Long-term	$PM_{2.5}$	RR (6)	[63]
34	Gujral and Sinha, 2021	USA	SARS-CoV-2 Cases	Ecological	Short-term	$PM_{2.5}$, PM_{10}, O_3	RR (6)	[64]
35	Hadei et al., 2021	Iran	Mort. & Morbidity	Time Series	Short-term	$PM_{2.5}$, PM_{10}, NO_2, O_3	RR (7)	[65]
36	Jiang and Xu, 2021	China	COVID-19 Deaths	Time Series	Short-term	$PM_{2.5}$, PM_{10}, SO_2, CO, NO_2, O_3	(6)	[66]

Table 1. Continued

#	Study	Location	Outcome	Type	Exposure	Pollutants	Effect (NOS)	Ref.
37	Lu et al., 2021	China	COVID-19 Infect.	Time-series	Short-term	$PM_{2.5}$, O_3, SO_2, NO_2	RR (5)	[67]
38	Solimini et al., 2021	63 Countries	COVID-19 Cases	Ecological	Long-term	$PM_{2.5}$, PM_{10}	RR (7)	[34]
39	Sahoo, 2021	India	Infected Cases	Time Series	Short-term	$PM_{2.5}$, PM_{10}, NO_2, SO_2	RR (6)	[68]
40	Saez et al., 2020	Spain	Incidence & Death	Ecological	Long-term	NO_2, PM_{10}	RR (7)	[69]
41	Karimi et al., 2022	Iran	Mort. & Hosp.	Ecological	Long-term	PM_{10}, $PM_{2.5}$, SO_2, CO, O_3, NO_2	RR (8)	[70]
42	Petroni et al., 2020	USA	COVID-19 Mort.	Time Series	Long-term	$PM_{2.5}$, O_3	RR (7)	[71]
43	Shim et al., 2022	South Korea	COVID-19 Incidence	Cross-section	Short-term	SO_2, CO, NO_2, $PM_{2.5}$, PM_{10}	OR (7)	[72]
44	Sheridan et al., 2022	England	Hosp. & Mort.	Cohort	Long-term	PM_{10}	OR (6)	[73]

Abbreviations: Mort.: Mortality; Hosp.: Hospitalization; Infect.: Infection; Intub.: Intubation; NOS: Newcastle-Ottawa Scale; RR: Risk Ratio; OR: Odds Ratio; HR: Hazard Ratio.

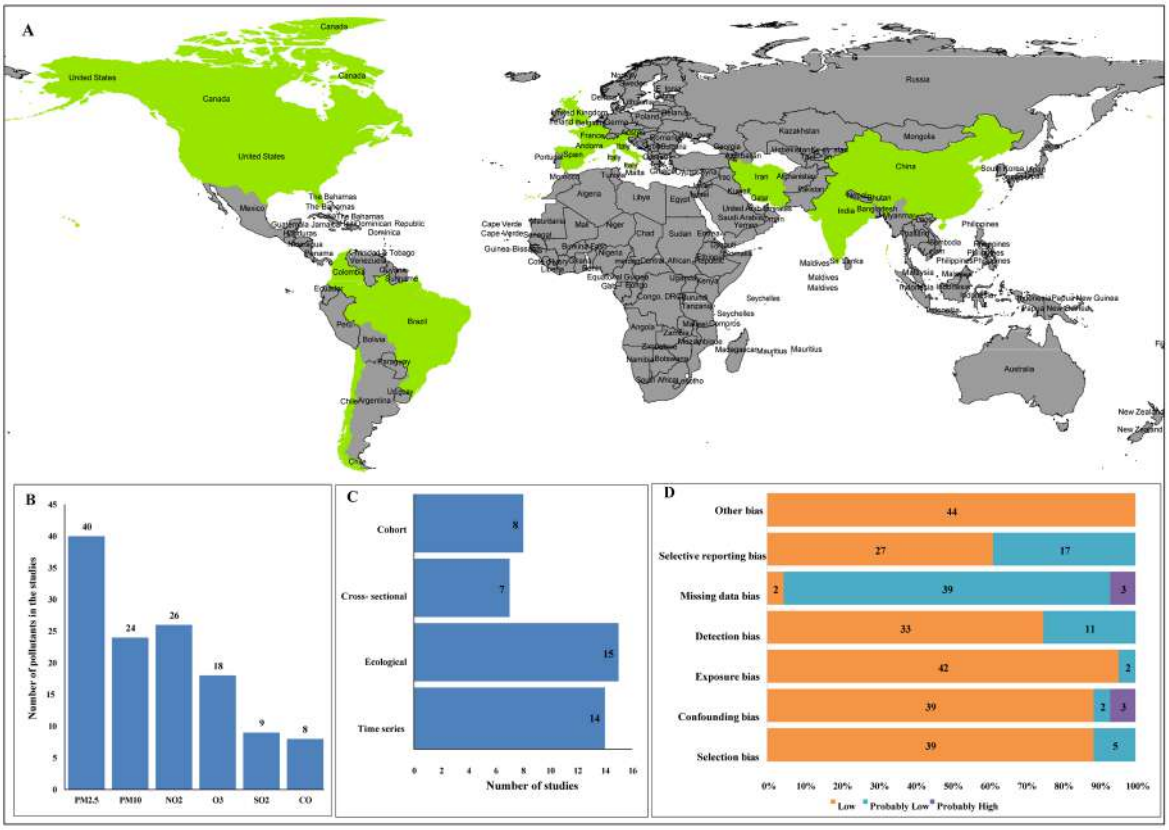


Fig. 2. (A) a global map indicating the geographical distribution of included studies (highlighted in green); (B) a frequency distribution of publications by pollutant category; (C) a classification of studies by methodological design; and (D) a summary of risk-of-bias assessments using a color-coded system (orange: low risk, blue: probably low risk, dark blue: probably high risk)

Table 2. Risk of bias assessment for selected studies

Study	Selection bias	Confounding bias	Exposure bias	Detection bias	Missing data bias	Selective reporting bias	Other bias
Travaglio et al., 2021	⊕	⊕	⊕	⊕	⊗	⊖	⊕
Sanchez-Piedra et al., 2021	⊕	⊕	⊕	⊕	⊗	⊖	⊕
Rodriguez-Villamizar et al., 2021	⊖	⊕	⊕	⊕	⊗	⊖	⊕
Mendy et al., 2021	⊕	⊖	⊕	⊖	⊖	⊖	⊕
Konstantinoudis et al., 2021	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Bowe et al., 2021	⊕	⊕	⊕	⊕	⊖	⊖	⊕
Tchicaya et al., 2021	⊖	⊕	⊕	⊖	⊖	⊖	⊕
Zheng et al., 2021b	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Norouzi et al., 2022	⊕	⊕	⊕	⊕	⊖	⊖	⊕
Hutter et al., 2020	⊕	⊕	⊕	⊖	⊖	⊖	⊕
Wu et al., 2020b	⊕	⊕	⊕	⊖	⊖	⊖	⊕
Bozack et al., 2022	⊕	⊕	⊕	⊕	⊖	⊖	⊕
Marquès et al., 2022	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Garcia et al., 2022	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Scalsky et al., 2022	⊕	⊕	⊖	⊕	⊖	⊕	⊕
Aloisi et al., 2022	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Stieb et al., 2020	⊕	⊕	⊕	⊖	⊖	⊖	⊕
Zhu et al., 2020	⊕	⊕	⊕	⊕	⊖	⊕	⊕
De Angelis et al., 2021	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Dales et al., 2021	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Ibarra-Espinosa et al., 2022	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Yao et al., 2020b	⊕	⊕	⊕	⊖	⊖	⊕	⊕
Xu et al., 2022	⊕	⊕	⊕	⊖	⊖	⊖	⊕
Becchetti et al., 2022	⊕	⊕	⊕	⊕	⊖	⊖	⊕
Meo et al., 2021	⊕	⊗	⊖	⊖	⊖	⊖	⊕
Liang et al., 2020	⊕	⊕	⊕	⊕	⊖	⊖	⊕
Tian et al., 2021	⊕	⊕	⊕	⊕	⊖	⊖	⊕
Veronesi et al., 2022	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Wang et al., 2020	⊖	⊕	⊕	⊖	⊖	⊖	⊕
Coker et al., 2020	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Zhang et al., 2021	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Jiang et al., 2020	⊕	⊖	⊕	⊕	⊖	⊕	⊕
Wu et al., 2020d	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Gujral and Sinha, 2021	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Hadei et al., 2021	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Jiang and Xu, 2021	⊖	⊗	⊕	⊖	⊖	⊕	⊕
Lu et al., 2021	⊖	⊗	⊕	⊖	⊖	⊕	⊕
Solimini et al., 2021	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Sahoo, 2021	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Saez et al., 2020	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Karimi et al., 2022	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Petroni et al., 2020	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Shim et al., 2022	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Sheridan et al., 2022	⊕	⊕	⊕	⊕	⊖	⊕	⊕
Risk of bias rating	**Low** ⊕		**Probably Low** ⊖		**Probably high** ⊗	**High** ?	

Table 3. The pooled RR of COVID-19 mortality and hospitalization per 1µg/m^3 increment in air pollutants by the random effect model

Outcome	Pollutant	Exposure	Number of studies	Pooled RR (95% CI)			Cochran's Q	p-value	I^2 (%)
Mortality	$PM_{2.5}$	Short-term	16	1.046	1.031	1.062	1064.51	0.000	98.6%
		Long-term	10	1.043	1.033	1.053	64.99	0.000	86.2%
	PM_{10}	Short-term	6	1.00	0.988	1.002	201.32	0.000	97.5%
		Long-term	5	1.079	1.005	1.154	29.52	0.000	86.4%
	NO_2	Short-term	15	1.036	1.020	1.053	902.38	0.000	98.2%
		Long-term	7	1.017	1.004	1.029	22.61	0.002	69.0%
	O_3	Short-term	7	0.980	0.964	0.995	179.21	0.000	96.7%
		Long-term	5	1.036	1.005	1.067	38.90	0.000	89.7%
	SO_2	Short-term	5	1.051	0.970	1.131	139.23	0.000	97.1%
		Long-term	1	1.077	1.021	1.133	-	-	-
	CO	Short-term	6	0.730	0.302	1.157	4633.81	0.000	99.9%
		Long-term	1	1.004	0.998	1.010	-	-	-
Hospitalization	$PM_{2.5}$	Short-term	5	1.015	1.00	1.035	144.96	0.000	97.2%
		Long-term	17	1.028	1.022	1.034	1092.49	0.000	98.5%
	PM_{10}	Short-term	8	1.013	1.001	1.026	130.63	0.000	94.6%
		Long-term	7	1.011	1.004	1.018	29.99	0.000	80.0%
	NO_2	Short-term	6	1.032	1.017	1.047	90.33	0.000	94.5%
		Long-term	11	1.040	1.024	1.056	82.63	0.000	87.9%
	O_3	Short-term	8	1.007	1.003	1.011	32.89	0.000	78.7%
		Long-term	2	0.992	0.969	1.014	10.16	0.001	90.2%
	SO_2	Short-term	9	1.019	1.002	1.036	289.70	0.000	97.2%
		Long-term	1	1.045	1.00	1.091	-	-	-
	CO	Short-term	6	0.861	0.389	1.334	7703.77	0.000	98.9%
		Long-term	1	1.066	0.981	1.151	-	-	-

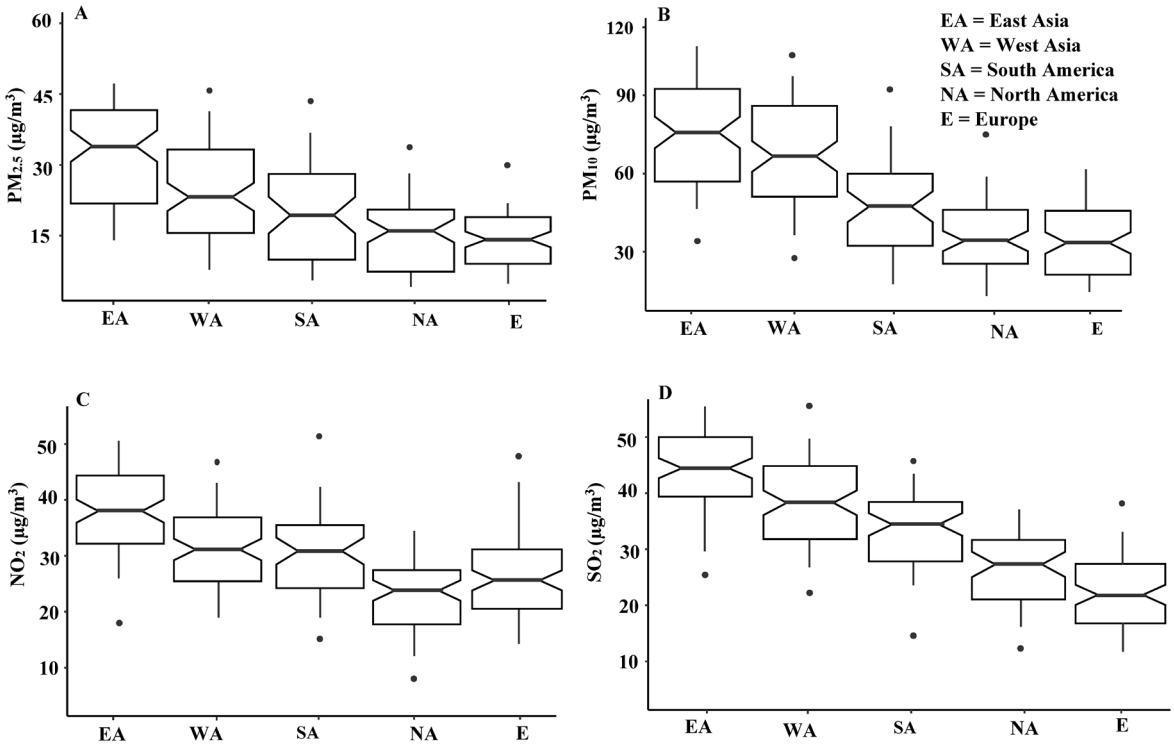
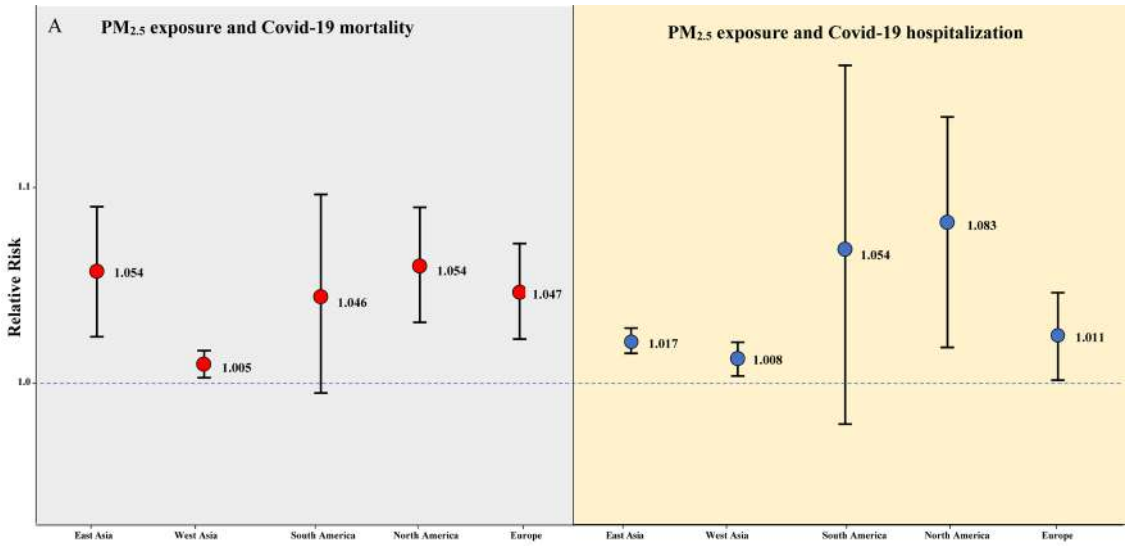


Fig. 3. The regional distributions of four principal air pollutants—$PM_{2.5}$, PM_{10}, NO_2, and SO_2. The boxplot visualization displays the data distribution through median values, interquartile ranges, and extreme values, with outliers explicitly represented



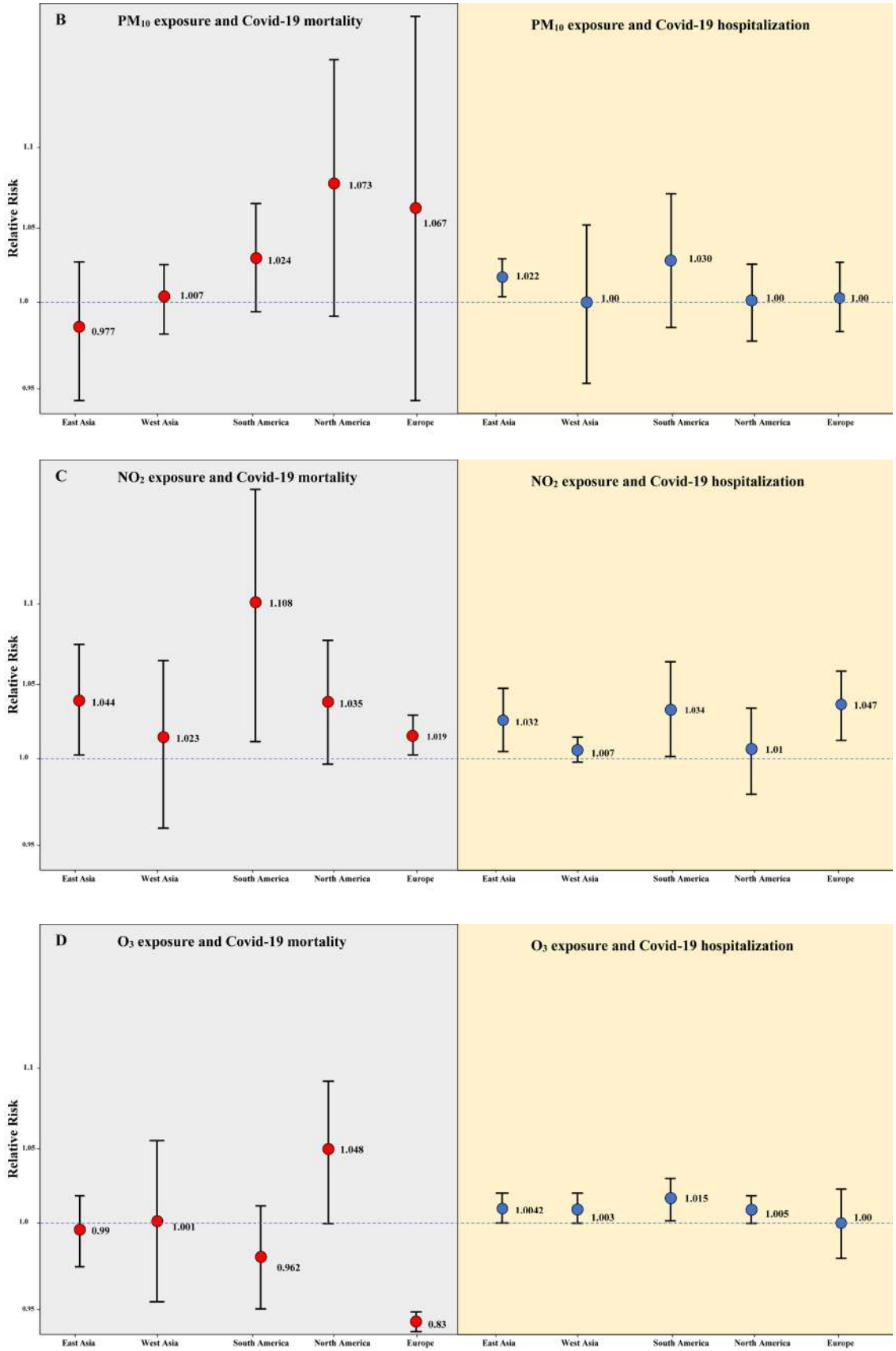


Fig. 4. Pooled risk estimates for COVID-19 mortality (red circles) and hospital admission (blue circles) with corresponding confidence intervals, stratified by geographical region, for $PM_{2.5}$, PM_{10}, NO_2, and O_3 exposures per 1 µg/m³ increment

Risk of bias

Risk of bias, evaluated by the OHAT tool, was generally low across most domains and studies (Table 2, Fig. 2d). Most studies demonstrated low risk of confounding and detection bias, while a few exhibited probable high risks in specific domains, such as missing data. Assessment of methodological quality using the Newcastle-Ottawa Scale (NOS) specified that 28 of the included investigations (59.5%) were of high quality, achieving a score of seven or higher (Table 1). Overall, the studies showed high methodological rigor.

Relationships of airborne pollutants with COVID-19 severity

Pooled Relative Risks (RRs) for a 1 μg/m³ increment in pollutant concentration are summarized in Table 3. All odds ratios and hazard ratios were converted to RRs to ensure comparability.

Mortality: Elevated concentrations of $PM_{2.5}$ demonstrated a significant association with COVID-19 mortality. For each incremental rise of 1 μg/m³ in $PM_{2.5}$ levels, the relative risk for mortality was 1.043 (95% CI: 1.033–1.053) for short-term exposure and 1.046 (95% CI: 1.031–1.062) for long-term exposure.

Sustained exposure to coarse Particulate Matter (PM_{10}) was related to a 7.9% increment in mortality risk per 1 μg/m³ (RR: 1.079, 95% CI: 1.005–1.154). Similarly, chronic exposure to nitrogen dioxide (NO_2) and sulfur dioxide (SO_2) showed significant associations with elevated mortality, with RRs of 1.017 (95% CI: 1.004–1.029) and 1.077 (95% CI: 1.021–1.133) per 1 μg/m³ increment, respectively. Long-term ozone (O_3) exposure demonstrated a modest positive relationship with risk of mortality (RR: 1.036, 95% CI: 1.005–1.067), whereas short-term O_3 exposure was inversely associated with mortality, potentially reflecting residual confounding, measurement error, or short-term physiological adaptation, warranting further investigation. CO showed inconsistent associations, with non-significant effects for short-term exposure and wide confidence intervals pointing to limited statistical power (Fig. 4).

Hospitalization: Both short- and long-term exposures to $PM_{2.5}$ and PM_{10} were significantly related to COVID-19 hospitalization. Associations for NO_2 (short-term RR, 1.032; long-term RR, 1.032) and O_3 (short-term RR, 1.007; long-term RR, 1.000) were generally consistent, with long-term O_3 exposure showing no measurable effect. For long-term exposures, NO_2 and SO_2 remained associated with hospitalization, although the association for SO_2 was of borderline significance, indicating a less conclusive relationship (RR, 1.045; 95% CI, 1.000–1.091). CO was not positively related to hospitalization in either timeframe (Fig. 4).

Additional analyses

Subgroup analyses by geographic region indicated that the positive association between $PM_{2.5}$ and COVID-19 mortality was consistent and significant in Europe, North America, West Asia, and East Asia, but not in South America. Regional analyses for PM_{10} did not show significant associations in any subgroup. Statistical evidence from Egger's test ($t = 3.45$, $p = 0.041$) suggested possible publication bias in the studies examining $PM_{2.5}$ and mortality. Despite this indication, subsequent sensitivity analyses demonstrated that the aggregated risk estimates remained stable, thereby confirming the fundamental reliability of this key finding.

This meta-analysis of 44 studies across 30 countries provides robust evidence that exposure to air pollutants—specifically $PM_{2.5}$, PM_{10}, NO_2, and SO_2—significantly increases the risk of severe COVID-19 outcomes. These results underline the

role of air pollution as a critical environmental determinant of pandemic severity.

The analysis revealed distinct exposure-response patterns. $PM_{2.5}$ elevated mortality risk from both long-term (RR, 1.046; 95% CI, 1.031–1.062) and short-term exposure (RR, 1.043; 95% CI, 1.033–1.053), implicating both acute inflammatory pathways and cumulative physiological damage. In contrast, the mortality risks for PM_{10} and SO_2 were specific to long-term exposure (RR, 1.079 and 1.077, respectively), suggesting their adverse effects manifest through sustained processes that progressively compromise cardiopulmonary resilience. NO_2 exhibited consistent risks across timeframes (short-term RR, 1.033; long-term RR, 1.017), aligning with its ability to provoke both immediate airway inflammation and chronic vascular endothelial dysfunction [74]. For hospitalization, significant associations were confirmed for long-term exposures to $PM_{2.5}$ (RR, 1.028), PM_{10} (RR, 1.011), and NO_2 (RR, 1.032), while SO_2 showed a borderline effect (RR, 1.045; 95% CI, 1.000–1.091).

The observed population-level relationships find support in recognized pathophysiological pathways. Sustained inhalation of particulate matter and nitrogen dioxide (NO_2) initiates a state of chronic inflammation and oxidative stress, principally mediated through mitochondrial dysfunction that generates Reactive Oxygen Species (ROS) at concentrations that surpass the protective threshold of cellular antioxidant systems [75]. This primed pro-inflammatory state may exacerbate the cytokine release syndrome characteristic of severe COVID-19. Furthermore, long-term $PM_{2.5}$ exposure upregulates ACE2 receptor expression in respiratory epithelia, enhancing cellular susceptibility to SARS-CoV-2 infection and potentially accelerating viral replication [22].In combination, these mechanisms of heightened host vulnerability offer a biologically plausible framework for understanding the elevated rates of hospital admission and fatality documented in the epidemiological data. Evidence suggesting particles act as viral vectors remains limited and inconsistent across studies [76, 77].

The null findings for O_3 and CO are mechanistically plausible. The inverse association with short-term O_3 may stem from its complex atmospheric chemistry and substantial exposure misclassification, as fixed-site monitors often fail to capture concentration gradients across heterogeneous urban landscapes [13, 75]. For CO, the lack of association likely reflects ambient concentrations below thresholds for systemic inflammatory effects, combined with a primary toxicity mechanism—carboxyhemoglobin formation—that is less directly involved in COVID-19 immunopathology than the pathways activated by other pollutants [22, 23].

Our pooled estimates are generally more conservative than those in some previous meta-analyses, potentially due to broader inclusion criteria and rigorous handling of substantial heterogeneity (e.g., for long-term $PM_{2.5}$: I^2 = 89%). This heterogeneity arose from variations in study design, population characteristics, and exposure assessment methods. Nevertheless, the consistent direction of effects across diverse settings strengthens the generalizability of the findings. The stability of these findings was verified through sensitivity testing. Although Egger's regression test indicated minor publication bias specifically for European $PM_{2.5}$ analyses (p=0.041), application of the trim-and-fill method demonstrated that this bias did not substantially affect the overall conclusions.

The interpretation of these findings should consider several methodological constraints. The ecological design of many studies limits causal inference at the individual level and

raises potential for residual confounding. Exposure misclassification remains a concern, particularly for spatially variable pollutants. The geographic scope is limited, with a notable underrepresentation of Low- and Middle-Income Countries (LMICs). Future cohort studies in these highly polluted regions are essential to validate these associations and guide public health interventions. Finally, the focus on ambient pollution excludes indoor exposures, a significant risk factor in many settings.

Conclusion

This meta-analysis identifies ambient $PM_{2.5}$, PM_{10}, NO_2, and SO_2 as significant and modifiable determinants of severe COVID-19 outcomes. The strength of association varies by pollutant and exposure duration, reflecting distinct toxicological profiles. These findings indicate that rigorous air quality management is a critical component of pandemic preparedness. Integrating air pollution control into public health policy represents a strategic, non-pharmaceutical intervention to mitigate severe respiratory outcomes, strengthen population resilience, and yield substantial co-benefits for cardiopulmonary health.

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Competing interests

The authors declare that they have no competing interests

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Ethical consideration

"Ethical issues (Including plagiarism, Informed Consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc) have been completely ob-served by the authors."

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