GLOBAL HEART

Impact of Ambient Air Pollution on Cardiovascular Diseases in Low- and Lower-Middle-Income Countries: A Systematic Review and Meta-Analysis

ORIGINAL RESEARCH

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ABSTRACT

Air pollution contributes to over 8.1 million deaths annually, predominantly from cardiovascular causes. The burden of air pollution is significantly higher in low-income countries (LICs) and lower middle-income countries (LMICs), yet most air pollution research is performed in higher-income countries. Our objective was to systematically review the association between exposure to ambient air pollution and cardiovascular disease (CVD) in LICs and LMICs. PubMed and Global Health databases were systematically searched for studies that explore associations between daily increases in gaseous (SO_2, NO_2, CO, O_3) and particulate matter (PM) air pollutants with CVD mortality and hospital admission in adults. Studies were assessed for risk of bias based on outcome validity, exposure measurement quality, and confounder adjustment. A random-effects model was used to estimate overall and per-pollutant risks from short-term exposure studies, standardised to 10 μg/m³ increments. Of 1329 articles screened, 48 met the inclusion criteria, of which 22 included a measure of relative risk suitable for meta-analysis. Short-term exposure to $PM_{2.5}$ and PM_{10} were associated with increased combined mortality and hospital admission with 0.53% (95% CI: 0.31%–0.75%) and 1.68% (95% CI: 0.17%–3.21%) increase per 10 μg/m³, respectively. NO_2 showed a 0.66% increase (95% CI: 0.36%–0.97%) per 10 μg/m³. This systematic review highlights the limited evidence on air pollution and CVD in LICs and LMICs. Nonetheless, this meta-analysis found positive associations between several ambient air pollutants and cardiovascular hospital admissions and mortality. There is the vital need for further research in underrepresented regions, particularly on the effects of long-term exposure, in order to establish the true burden of air pollution on cardiovascular health in regions where air pollution is frequently high, or access to healthcare is limited.

KEYWORDS:

particulate matter; nitrogen dioxide; hospital admissions; mortality; developing countries; LMICs

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1. INTRODUCTION

Air pollution (both indoor and outdoor), which contributed to over eight million deaths globally in 2021, is considered one of the most worrying global health emergencies (1, 2). It is both the second largest modifiable risk factor for mortality and the greatest risk factor for disability-adjusted life years (DALYs) (3). Almost 90% of global ambient air pollution related deaths occur in low- and lower-middle-income countries (LICs and LMICs) (4). The disproportionate burden of air pollution-related cardiovascular disease in LICs and LMICs stems from multiple factors. While higher ambient pollution concentrations play a significant role, there are also important considerations regarding potential differences in air pollution composition and population demographics that may not be fully captured in current global models. Understanding whether the same exposure produces similar or different cardiovascular effects across diverse settings is critical for accurate burden assessment.

Ambient (outdoor) air pollution can arise from many sources and consequently contains a large mixture of thousands of chemicals, which vary spatially and temporally depending on source, dispersion, atmospheric reactions and meteorological conditions (5). Criteria air pollutants that affect health include particulate matter (PM) of varying size, nitrogen dioxide (NO_2), ozone (O_3), sulfur dioxide (SO_2) and carbon monoxide (CO). Fine particulate matter (PM with aerodynamic diameter ≤2.5 μm; '$PM_{2.5}$') is the air pollutant that is most consistently associated with adverse health effects especially in organ systems beyond the lung.

In 2021, cardiovascular diseases (CVDs) were responsible for 52% of the total DALYs attributable to ambient $PM_{2.5}$ exposure (3). Both short- and long-term exposure to air pollution is associated with increased risk of CVD (6, 7), including cardiovascular outcomes such as myocardial infarction (MI) (8–10), stroke (11, 12), and heart failure (HF) (13, 14). The causality of these observations are supported by a substantial body of mechanistic research that show that inhaled air pollutants, especially PM, induce cardiovascular dysfunction through multiple mechanisms, such as oxidative stress, inflammation, endothelial dysfunction, thrombosis, autonomic imbalance, and acceleration of the growth and instability of atherosclerosis plaques (15, 16).

The majority of epidemiological studies on air pollution have been conducted in high-income countries (HICs) (17, 18), and may not be generalisable to LICs and LMICs where sources of air pollution and population demographics and diseases differ. For example, current global estimates of the health impact of air pollution in LICs and LMICs, including those from the Global Burden of Disease (GBD) study, rely heavily on exposure-response functions derived predominantly from HICs. These estimates often apply concentration-response relationships from HICs to pollution levels and demographic profiles in LMICs, with limited validation from local studies. This extrapolation approach, while practical given data limitations, introduces uncertainty in burden estimates. Removing or lowering risk factors is key to the prevention of CVD (19, 20) and is especially important in LMIC settings given that the availability of treatment may be limited.

Despite recognition of the disproportionate burden of both air pollution exposure and CVD suffered by LICs and LMICs, the evidence base remains relatively limited (21, 22). Existing reviews group LMICs with upper-middle-income countries (7, 18) or focus only on particulate matter (23). Another notable gap is the absence of pooled estimates of exposure-response relationships, which hinders an adequate burden assessment and means to direct public health policy. Here, we aimed to systematically review and synthesise the evidence on associations between ambient air pollution exposure and cardiovascular outcomes in, specifically, LICs and LMICs. Our review has been designed to include all criteria air pollutants ($PM_{2.5}$, PM_{10}, SO_2, NO_2, CO, and O_3) and both cardiovascular mortality and hospitalisation. The review aims not only to establish and quantify these effects of air pollution in these regions, but also to identify research gaps that are needed to ascertain full burden on health and help guide air quality interventions that could improve cardiovascular health.

2. METHODS

2.1 SEARCH STRATEGY AND SELECTION CRITERIA

The systematic review and meta-analysis protocol was registered with PROSPERO (registration CRD42023484929) and adhered to the Preferred Reporting Items of Systematic Reviews and Meta-Analysis (PRISMA) guidelines (24).

PubMed and Global Health databases were systematically searched for relevant resources. The search terms were a pairwise combination of 'air pollution' (including particulate matter ($PM_{2.5}$ and PM_{10} (particles with aerodynamic diameter ≤10 μm)), SO_2, NO_2, CO, O_3) and 'cardiovascular disease', 'myocardial infarction', 'stroke', and 'heart failure' (HF), selected to capture both overall CVD outcomes and specific CVDs with well-established links to ambient air pollution exposure. These terms were combined using AND, and OR, and some were expanded, especially those with multiple synonyms, to include all pertinent articles. Adjustments to the search were made as necessary, by the specific requirements of the database used. The search spanned the inception of the database to June 2024 (for full search strategy, see **Supplementary Material**).

Studies were included if published in English, conducted in LICs or LMICs as from the 2024 World Bank definition (**Figure S1**), and reported associations between ambient air pollution and the aforementioned CVD endpoints. We included studies of all age groups with no restrictions on population demographics. This review focused on ambient air pollution; studies examining indoor air pollution or non-ambient occupational air pollution exposure were excluded. We included both short-term exposure studies (examining acute exposure over days to weeks) and long-term exposure studies (examining chronic exposure over months to years). Various methodological approaches were eligible—including time-series, case-crossover, cohort studies, cross-sectional, ecological, cost analysis, comparative risk assessment designs, and studies using the World Health Organization (WHO) Health Impact Assessment model (e.g., AirQ+), which estimates health impacts of air pollution using standardised assumptions and software tools—as long as they reported quantitative associations between ambient air pollution and cardiovascular outcomes. Summaries, editorials, short commentaries, conference proceedings, non-peer-reviewed studies, and full texts that were not available online were all excluded.

2.2 SCREENING AND DATA EXTRACTION

Results from the search were entered into Endnote, and duplicates removed. Rayyan was used to screen the papers. Titles/abstracts and full text-screening, and data extraction process were conducted independently by M.A. and S.R. For each article, meta-data information was extracted via a predefined data-extraction tool previously used (22). In summary, the following were extracted: (1) study characteristics (first author, publication year, study design, country, and data source); (2) ambient air pollution measurement and measurement tools (e.g., fixed monitoring stations, satellite-derived estimates, or personal monitors) and period of exposure (short-term or long-term); and (3) CVD admission and mortality (22).

Any disparities between the reviewers were deliberated, and if necessary, resolved by a third reviewer (S.S.).

2.3 RISK OF BIAS ASSESSMENT

The risk of bias assessment tool adapted for this study has been previously reported (9, 22, 25). This evaluation covers three components: validity of the occurrence of CVD (rated 0 or 1), quality of air pollutant measurements (rated 0 or 1), and the degree of confounder adjustment (rated 0, 1, 2, or 3). Studies were rated as good quality (best scores in all domains), average (mixed scores), or low quality (any domain scored zero). (For full description, see **Supplementary Material**.)

2.4 DATA ANALYSIS

For the meta-analysis, we included only studies reporting relative risk (RR), hazard ratio (HR), or odds ratio (OR) for lag zero or lag one day. Only one study provided cumulative lags (0–1) which was used as the shortest lag. RR were pooled for a standardised increment in pollutant concentration, with all concentrations standardised to a 10 $\mu g/m^3$ increment.

For this standardisation, we used the following conversions specific for each pollutant: one part per billion (ppb) was converted to 1.88 μg/m³ for NO_2, 2.62 μg/m³ for SO_2, and 1.96 μg/m³ for O_3. One part per million (ppm) was converted to 1145 μg/m³ for CO (under the assumption of standard temperature and pressure of 25 °C and 1 atm) (26).

In line with established methodological practices in air pollution meta-analyses (14), we assumed a linear relationship between exposure and outcome for standardisation purposes. Standardised risk estimates were calculated for each study using the following equation (14):

$$\mathbf{RR}_{(\text{standardised})} = \mathbf{RR}^{\text{Increment (10)/Increment(original)}} \quad (1)$$

To ensure consistency across studies and reduce heterogeneity from differing lag structures, we extracted effect estimates for the shortest lag available (typically lag zero or lag 1) to assess overall risk estimates.

In studies reporting multiple health CVD outcomes (e.g., stroke, MI, HF) under exposure to the same pollutant and categorised by the same outcome type (admission, mortality), we conducted an internal meta-analysis to derive a single pooled effect. This pooled estimate was then used as a representative measure for that study in the overall meta-analysis of CVD admissions and mortality. For studies reporting both specific CVD outcomes and a broader overall CVD outcome for the same pollutant and endpoint, we exclusively used the overall CVD admission or mortality outcome to avoid double-counting and ensure consistency in the pooled analysis.

Funnel plots were used to examine publication bias and assessed for asymmetry using Egger's regression test; correction for asymmetry was performed using the trim and fill method (27).

A sensitivity analysis was conducted excluding 1) ORs; 2) studies from Iran (which represented the majority of included studies).

Because of the diversity in study designs, methods, lag exposures, and geographical and population differences, we used a random-effects model to account for within and between study heterogeneity. Heterogeneity was examined using the standard I^2 test. Statistical significance was taken as two-sided $p < 0.05$. Analysis was performed using R Software (version 4.4.2).

3. RESULTS

3.1 STUDY OVERVIEW

The initial literature search identified 1474 research articles. After removing duplicates, the articles were reduced to 1329. Following titles/abstracts and full-text review, 48 studies met the criteria for inclusion (Figure 1 and Table 1). The majority of studies were conducted in Iran (n = 38), followed by Vietnam (n = 3) and Bangladesh (n = 3). Indonesia, India, Nepal, and Lebanon had one study each (**Figure S2**). This includes one multi-country study (28) covering France, Iran, and Italy, from which only the Iran data were extracted. There were more short-term studies (n = 39) than long-term studies (n = 9). Time series and case-crossover studies were the most frequently used study designs (n = 23), followed by studies using the WHO Health Impact Assessment model (n = 14), cross-sectional studies (n = 5) and cohort studies (n = 3). Furthermore, three studies used cost analysis (n = 1), ecological (n = 1), and comparative risk assessment (n = 1) designs. Of the 48 studies included in the review, the majority (31; 64.6%) were of good or average methodological quality, with the most common limitations being inadequate confounder adjustment and insufficient air pollution measurement quality.

3.2 META-ANALYSIS

Overall CVD effects were obtained for four studies (**Figure S3**) through internal meta-analyses combining HF admission and MI admission for $PM_{2.5}$ (29); ischemic heart disease (IHD) mortality and stroke mortality for $PM_{2.5}$ (30); IHD admission and MI admission for CO, NO_2, O_3, and SO_2 separately (31); HF admission, IHD admission and stroke admission for CO (32). Overall CVD admission was provided as an outcome and used instead of other CVD specific outcomes for two studies (31, 33). **Figure S3** presents these combined within-study effect estimates that were subsequently used in our main meta-analysis.

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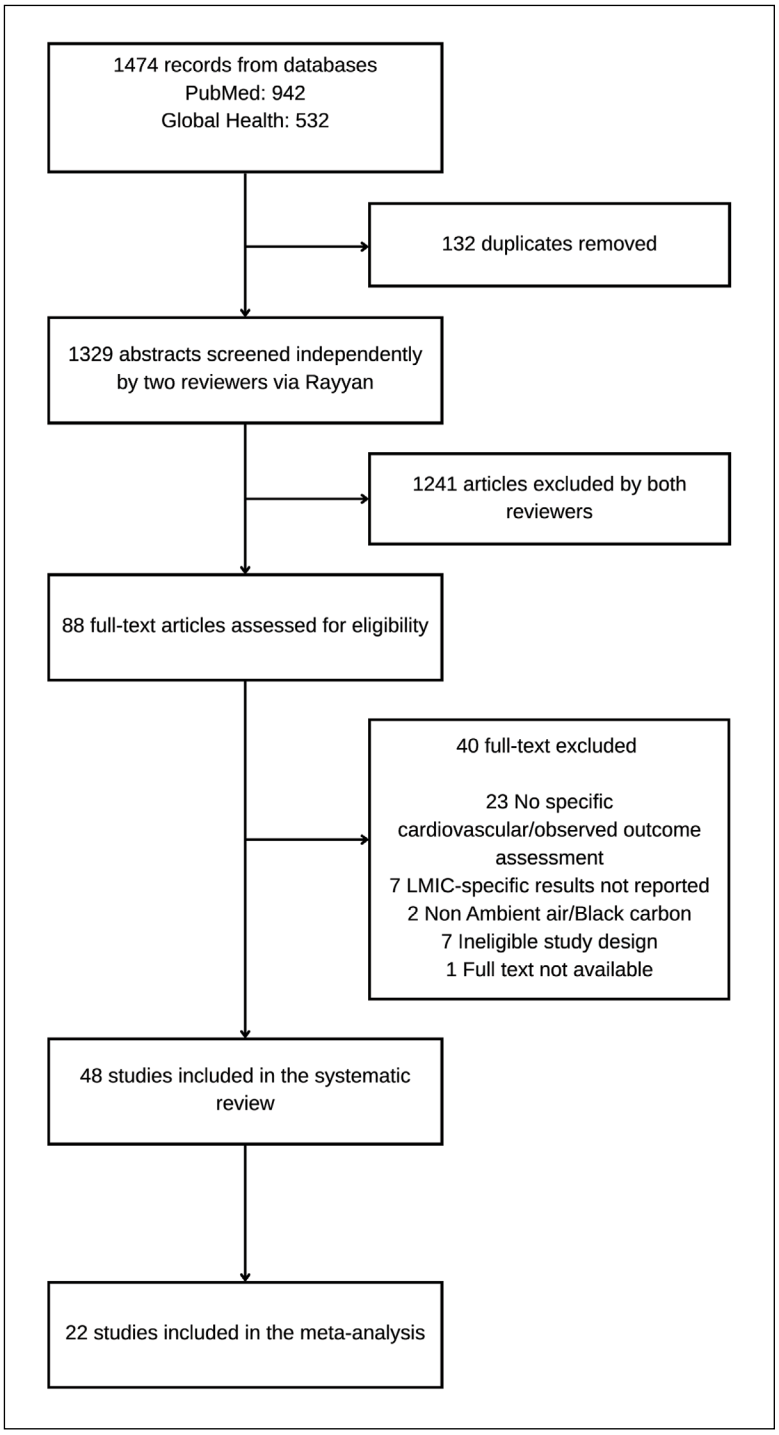

Figure 1 PRISMA flow chart.

The detailed pooled effect size RRs of pollutants for a 10 μg/m^3 increase along with p-values and heterogeneity are given in Table 2. The meta-analysis included a total of 22 studies and 101 data points investigating air pollutant-outcome pairs. Most of the studies (21; 88 estimates) examined the association between air pollutants and hospital admission for CVD, while only five studies (13 estimates) investigated associations with mortality.

Daily exposure to $PM_{2.5}$ and PM_{10} were both associated with increased combined hospital admission and CVD mortality with an RR of 1.0053 (95% Confidence Interval (CI): 1.0031–1.0075; I^2 = 74.3%) and 1.0168 (95% CI: 1.0017–1.0321; I^2 = 88.1%) per 10 μg/m^3 increase in daily concentrations respectively (Figure 2). When stratified by outcome (admission or mortality), there was strong evidence of association between exposure to 10 μg/m^3 of $PM_{2.5}$ and hospital admissions for CVD with an RR of 1.0046 (95% CI: 1.0025–1.0067; I^2 = 72.1%), while the same level of exposure to PM_{10} showed strong evidence of association with an RR of 1.019 (95% CI: 1.0017–1.0368; I^2 = 87.2%) for hospital admission. The RR for mortality was larger for $PM_{2.5}$ (RR = 1.0384; 95% CI: 0.9813–1.0988; I^2 = 86.8%) compared to PM_{10} (RR = 1.0032; 95%

Table 1 Baseline characteristics of included studies.

Note: (*) studies included in the meta-analysis, Air quality modelling software (WHO AirQ). WHO, World Health Organization; HIA, Health Impact Assessment; AirQ, Air Quality Health Impact Assessment tool.

AUTHOR	YEAR	COUNTRY	DATA SOURCE	DURATION	DESIGN	OUTCOME	POLLUTANTS	EXPOSURE ASSESSMENT	ROB ASSESSMENT
(63) (*)	2017	Iran	Iran statistical centre	Short term	Cross-sectional	admission	SO_2	Ground monitoring stations	Low
(64) (*)	2017	Iran	Hospital database	Short term	Cross-sectional	admission	SO_2, NO_2, NO, NO_x, CO, O_3, PM_{10}	Ground monitoring stations	Average
(33) (*)	2019	Iran	Hospital database	Short term	Time series	admission	NO_2, NO, O_3	Ground monitoring stations	Good
(65) (*)	2005	Iran	Hospital database	Long term	Time series	admission	SO_2, NO_2, CO, O_3, PM_{10}	Ground monitoring stations	Good
(66)	2022	Vietnam	Hospital database	Short term	Time series	admission	PM_{10}, NO_2	Ground monitoring stations	Average
(37) (*)	2016	Vietnam	Hospital database	Short term	Time series	admission	NO_2, PM_{10}, SO_2	Ground monitoring stations	Average
(67) (*)	2017	Iran	Hospital database	Short term	Time series	admission	SO_2, CO_2, NO_2, O_3, $PM_{2.5}$, PM_{10}	Ground monitoring stations	Good
(32) (*)	2023	Iran	Clinical registry	Short term	Time series	admission	CO	Ground monitoring stations	Good
(34) (*)	2019	Iran	Hospital database	Short term	Case crossover	admission	O_3, SO_2, NO_2, CO, PM_{10}, $PM_{2.5}$	Ground monitoring stations	Average
(68)	2022	Indonesia	Survey data	Long term	Cross-sectional	admission	$PM_{2.5}$	Satellite monitoring	Low
(38) (*)	2019	Bangladesh	Hospital database	Short term	Case crossover	admission	$PM_{2.5}$	Ground monitoring stations	Average
(35) (*)	2023	Iran	Hospital database	Short term	Case crossover	admission	$PM_{2.5}$, PM_{10}	Ground monitoring stations	Average
(69)	2017	Iran	Hospital database	Short term	HIA, WHO AirQ	mortality	O_3	Ground monitoring stations	Low
(70)	2015	Iran	WHO AirQ	Short term	HIA, WHO AirQ	mortality	O_3	Ground monitoring stations	Low
(39) (*)	2021	Bangladesh	Hospital database	Short term	Time series	mortality, admission	$PM_{2.5}$	Ground monitoring stations	Average
(71)	2017	Iran	WHO AirQ	Short term	HIA, WHO AirQ	mortality	$PM_{2.5}$, PM_{10}	Ground monitoring stations	Low
(72)	2016	Iran	WHO AirQ	Short term	HIA, WHO AirQ	mortality	$PM_{2.5}$	Ground monitoring stations	Low
(73)	2020	Iran	WHO AirQ	Long term	HIA, WHO AirQ	mortality	$PM_{2.5}$	Ground monitoring stations	Low
(36) (*)	2019	Iran	Hospital database	Short term	Case crossover	admission	$PM_{2.5}$, PM_{10}, SO_2, O_3 CO, NO_2	Ground monitoring stations	Average
(28) (*)	2019	France, Iran, Italy	WHO AirQ	Short term	HIA, WHO AirQ	mortality, admission	PM_{10}, $PM_{2.5}$, O_3	Ground monitoring stations	Low
(74) (*)	2023	Iran	Hospital database	Short term	Time series	admission	O_3, CO, NO_2, SO_2, PM_{10}, $PM_{2.5}$	Ground monitoring stations	Average
(75) (*)	2022	Iran	Hospital database	Short term	Time series	admission, mortality	SO_2, NO_2, CO, O_3	Ground monitoring stations	Good
(76)	2021	Iran	Hospital database	Short term	Time series	admission	CO, O_3, $PM_{2.5}$, NO_2, SO_2	Ground monitoring stations	Good

(Contd.)

AUTHOR	YEAR	COUNTRY	DATA SOURCE	DURATION	DESIGN	OUTCOME	POLLUTANTS	EXPOSURE ASSESSMENT	ROB ASSESSMENT
(77) (*)	2012	Iran	Hospital database	Short term	Cross-sectional	admission	SO_2, CO, NO	Ground monitoring stations	Average
(30) (*)	2021	Iran	WHO AirQ	Long term	Time series	mortality	$PM_{2.5}$	Ground monitoring stations	Low
(78)	2018	Iran	WHO AirQ	Short term	HIA, WHO AirQ	mortality	PM_{10}, $PM_{2.5}$	Ground monitoring stations	Low
(79)	2020	Vietnam	Hospital database	Short term	Case crossover	admission	PM_{10}, $PM_{2.5}$, SO_2, CO, NO	Ground monitoring stations	Good
(80)	2016	Iran	WHO AirQ	Short term	HIA, WHO AirQ	mortality	PM_{10}, NO_2, O_3	Ground monitoring stations	Low
(81)	2019	Iran	Municipal records	Long term	Cost analysis	mortality	$PM_{2.5}$	Ground monitoring stations	Low
(82)	2014	Iran	WHO AirQ	Short term	HIA, WHO AirQ	admission	PM_{10}	Ground monitoring stations	Average
(83)	2022	Iran	WHO AirQ	Short term	HIA, WHO AirQ	Mortality, admission	$PM_{2.5}$, PM_{10}	Ground monitoring stations	Low
(84)	2017	Iran	WHO AirQ	Short term	HIA, WHO AirQ	mortality	PM_{10}	Ground monitoring stations	Low
(85) (*)	2018	Iran	Hospital database	Short term	Ecological	mortality	PM_{10}, O_3, NO_2, SO_2, CO	Ground monitoring stations	Good
(86)	2024	Bangladesh	Registry data	Long term	Cohort	mortality	$PM_{2.5}$	Satellite monitoring	Average
(87)	2021	Iran	Cohort data	Long term	Cohort	mortality	$PM_{2.5}$	Satellite monitoring	Average
(88)	2015	Iran	National registry	Short term	Cross-sectional	mortality	O_3, CO, NO_2, SO_2, PM_{10}	Ground monitoring stations	Average
(89)	2018	Iran	WHO AirQ	Short term	HIA, WHO AirQ	mortality, admission	O_3	Ground monitoring stations	Low
(90)	2016	Iran	WHO AirQ	Short term	HIA, WHO AirQ	mortality	SO_2, NO_2	Ground monitoring stations	Low
(91)	2022	India	Survey/registry data	Long term	Cohort	mortality	$PM_{2.5}$	Ground monitoring stations	Good
(92)	2017	Iran	Registry data	Long term	Comparative risk assessment	mortality	$PM_{2.5}$	Ground monitoring stations	Average
(93)	2017	Nepal	Hospital database	Short term	Case crossover	admission	PM_{10}	Ground monitoring stations	Good
(94)	2020	Iran	Hospital database	Short term	Time series	admission	CO, O_3, SO_2, NO_2, PM_{10}	Ground monitoring stations	Good
(29) (*)	2021	Iran	Hospital database	Short term	Time series	admission	$PM_{2.5}$	Ground monitoring stations	Average
(31) (*)	2019	Iran	Hospital database	Short term	Time series	admission	PM_{10}, O_3, NO_2	Ground monitoring stations	Average
(40) (*)	2015	Lebanon	Hospital database	Short term	Time series	admission	$PM_{2.5}$, PM_{10}	Ground monitoring stations	Good
(95)	2024	Iran	WHO AirQ	Short term	HIA, WHO AirQ	mortality	NO_2, O_3, SO_2	Ground monitoring stations	Low
(96) (*)	2018	Iran	Hospital database	Short term	Case crossover	admission	CO, O_3, NO_2, PM_{10}, SO_2	Ground monitoring stations	Good
(97)	2020	Iran	Municipal records	Short term	Time series	mortality	$PM_{2.5}$	Ground monitoring stations	Good

CATEGORY	RR	95% CI LOWER	95% CI UPPER	NUMBER OF STUDIES	NUMBER OF DATA POINTS	I^2
CO						
mortality + admission	1.0001	1.0001	1.0001	12	14	84.8%
admission	1.0001	1.0001	1.0001	11	13	86.0%
mortality	–	–	–	–	–	–
NO_2						
mortality + admission	1.0066	1.0036	1.0097	12	15	56.1%
admission	1.0062	1.0026	1.0099	11	13	51.0%
mortality	1.0108	1.0008	1.021	2	2	85.0%
NO						
mortality + admission						
admission	1.002	0.9987	1.0053	3	3	28.5%
mortality	–	–	–	–	–	–
O_3						
mortality + admission	0.9995	0.9988	1.0002	12	15	84.3%
admission	0.999	0.9985	0.9995	10	12	83.5%
mortality	1.0038	1.0007	1.0069	3	3	47.7%
PM_{10}						
mortality + admission	1.0168	1.0017	1.0321	13	19	88.1%
admission	1.0191	1.0017	1.0368	12	17	87.2%
mortality	1.0032	0.9984	1.008	2	2	95.1%
$PM_{2.5}$						
mortality + admission	1.0053	1.0031	1.0075	12	18	74.3%
admission	1.0046	1.0025	1.0067	11	15	72.1%
mortality	1.0384	0.9813	1.0988	3	3	86.8%
SO_2						
mortality + admission	1.0015	0.9986	1.0044	13	16	34.4%
admission	1.0022	0.9991	1.0053	12	14	34.3%
mortality	0.9976	0.9909	1.0044	2	2	25.6%

Table 2 Meta-Analysis results.

CI: 0.9984–1.008; I^2 = 95.1%), although neither were statistically significant due to the high variability of risk estimates, especially for $PM_{2.5}$ mortality.

Exposure to gaseous pollutants was reported in approximately half of the studies included in the analysis. NO_2 generally had the strongest positive associations with an overall RR of cardiovascular outcomes of 1.0066 (95% CI: 1.0036–1.0097; I^2 = 56.1%), 1.0062 (95% CI: 1.0026–1.0099; I^2 = 51.0%) for admission alone and 1.0108 (95% CI: 1.0008–1.0210; I^2 = 85.0%) for mortality alone, per 10 μg/m^3 increase in NO_2. O_3 was associated with an increased RR of mortality of 1.0038 (95% CI: 1.0007–1.0069; I^2 = 47.7%) per 10 μg/m^3, however there was no association with CVD admission or the overall CVD outcome. Increases in levels of CO were associated with an RR of 1.0001 (95% CI: 1.0001–1.0001; I^2: 86%) in hospital admissions per 10 μg/m^3. No significant associations were detected for SO_2 or NO with either CVD mortality or admission.

Publication bias (Egger's test for asymmetry $p < 0.05$) was observed only for NO_2 ($p = 0.0083$) and CO ($p < 0.001$) (**Table S1**). Adjustment for asymmetry with the trim and fill method did not alter the effect direction or effect size.

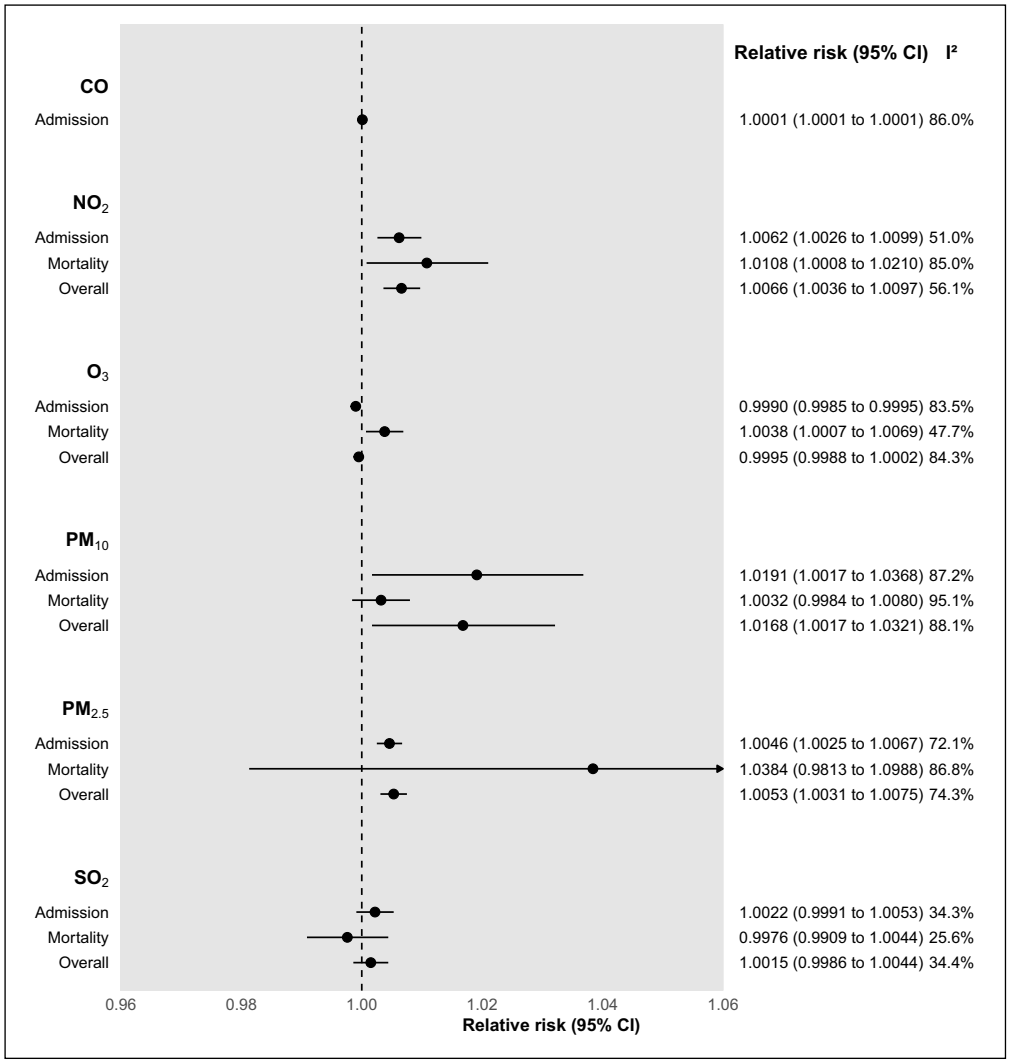

Figure 2 Forest plot of pooled relative risks for cardiovascular outcomes (per 10 μg/m³ increment).

3.3 SENSITIVITY ANALYSIS

A sensitivity analysis was conducted excluding three studies (34–36) which reported OR for hospital admission rather than RR, to account for any bias introduced with the assumption OR approximation to RR. While the pattern of effects remained the same, the effect size for daily exposure to PM_{10} and $PM_{2.5}$ were both associated with reductions in the RR of combined CVD mortality and hospital admissions in the sensitivity analysis, with an RR of 1.0065 (95% CI: 0.9941–1.0189; I^2 = 87.1%) and 1.0056 (95% CI: 1.0026–1.0087; I^2 = 48.8%) per 10 μg/m³ increase in daily concentrations, respectively (**Figure S4**). These changes were driven by reductions in the effect size for hospital admissions, bringing those associations closer to the null. For the gaseous pollutants, the only effect sizes impacted by this sensitivity analysis were related to NO_2, where the RR for combined mortality and hospital admissions reduced slightly to 1.0065 (95% CI: 1.0037–1.0094; I^2 = 58.1%). No other effect sizes were impacted by this sensitivity analysis.

An additional analysis was performed to assess whether the predominance of Iranian studies (38 out of 48 studies) might have biassed our overall findings, and to determine if the observed associations were consistent across different geographical settings. This meta-analysis was conducted on five studies (28, 37–40) (17 data points) after excluding all data from Iran (**Figure S5**). PM_{10} showed a smaller but less heterogeneous effect in the non-Iranian studies (RR = 1.0062; 95% CI: 1.0042–1.0081; I^2 = 0%) versus Iranian studies (RR = 1.0221; 95% CI: 1.0022–1.0424; I^2 = 87.5%). For $PM_{2.5}$, the non-Iranian studies had a lower but still significant effect estimate (RR = 1.0041; 95% CI: 1.0020–1.0061; I^2 = 0%) compared to Iranian studies (RR = 1.0091; 95% CI: 1.0041–1.0142; I^2 = 81.5%). Notably, the non-Iranian studies demonstrated markedly lower heterogeneity (I^2 = 0–6.4%) compared to the Iranian studies (I^2 = 79.5–87.5%). Due to the limited number of non-Iranian studies, separate analyses by outcomes were not feasible. In many cases, only one or two studies contributed data, although most of these

individual study findings were consistent with the overall associations observed in the pooled analyses.

4. DISCUSSION

This systematic review and meta-analysis find that short-term exposure to ambient air pollution, particularly for $PM_{2.5}$, PM_{10}, and NO_2, is associated with CVD outcomes in LICs and LMICs. These results complement global findings that air pollution is linked to cardiovascular morbidity and mortality, extending these findings beyond these data, which were predominantly from middle- to high-income countries, to lower income nations. While statistically significant associations were found, this review highlights the paucity of data from different countries that is needed to ascertain the true burden of air pollution in low-income nations.

Our findings build on the previous global meta-analysis by Shah et al. in 2013 and 2015 (12, 14), which linked air pollution to HF and stroke, by focusing specifically on LICs and LMICs. For CVD hospital admissions, the pooled $PM_{2.5}$ effect observed here (0.46% increase per 10 μg/m³) is lower than the 1.01% increase reported by Shah et al., but slightly higher than 0.26–0.29% reported from studies using data from more than 180 cities in China, an upper-middle income country (41, 42). By comparison, data from the USA, a high income country, found that a 10 μg/m³ increase in $PM_{10-2.5}$ was associated with a 0.69% increase in same-day cardiovascular hospital admissions (43). When examining mortality outcomes, our estimates (3.84% increase per 10 μg/m³) exceed those reported in previous short-term exposure systematic reviews from global (0.84%) and China-specific (0.63%) settings (44, 45).

Larger effects are typically observed for long-term cohort studies in HICs. For instance, in a US cohort study (7.5 million person-years) there was a considerably higher increase (14%, 95% CI: 2–27%) in stroke mortality and in IHD mortality (16%, 95% CI: 9–22%) per 10 μg/m³ $PM_{2.5}$ (46). This greater risk ratio in long-term studies reflects that admissions and mortality outcomes will likely capture any long-term effects of air pollution on disease progression as well as the acute effects of air pollution on exacerbation of CVD that leads to a cardiovascular event. Longitudinal cohort studies are more common in HICs due to factors that include the availability of long-term air pollution monitoring data from stationary monitoring networks, comprehensive medical records data systems, and the greater availability of resources required for prospective participant follow-up over many years. The limited number of long-term exposure studies in LICs/LMICs represents a critical research gap that needs to be addressed to establish the full burden of chronic air pollution exposure in these settings.

We observed that NO_2 generally had the strongest positive associations among gaseous pollutants. NO_2 is a gas with oxidative properties that induces pulmonary inflammation and oxidative stress; effects that can be transmitted to the circulation to impair cardiovascular function through multiple mechanisms, especially with long-term exposure (47). NO_2 is also a marker of traffic-related air pollution, emissions of which also include semi-volatile species and particulate matter, especially ultrafine particles, which are infrequently assessed in epidemiological studies; there may therefore be a degree of 'effect transfer' between closely correlated pollutants (48). Nonetheless, rapidly urbanising LMIC settings will see increasing vehicle numbers and, frequently, an older vehicle fleet that generates significant emissions with less stringent emission controls. A greater commitment towards targeted interventions to reduce traffic emissions in LICs/LMICs could therefore lead to significant health benefits, not only for respiratory conditions but also for cardiovascular outcomes. Other gaseous pollutants, such as O_3 and CO, showed varying degrees of association with cardiovascular outcomes. The weaker associations observed for O_3 might reflect its different spatial distribution and atmospheric processes (including reactions that lead to an inverse relationship with NO_2), in addition to the relatively limited monitoring of this pollutant. Nonetheless, O_3 should not be overlooked, as associations have been observed between O_3 and cardiovascular disease in other regions of the world (49). Marked seasonal variations in this pollutant are observed and levels of this pollutant are likely to rise as global temperatures increase. For CO, our results here are consistent with previous meta-analyses linking short-term CO exposure to risk of myocardial infarction (50).

The effect of air pollutant exposure on mortality was stronger than that on hospital admissions. This pattern remained consistent across pollutants; mortality outcomes generally exceeded admission outcomes, especially for $PM_{2.5}$ (3.84% vs 0.46% increase in risk) and NO_2 (1.08% vs 0.62% increase in risk). Our observations may reflect several LIC and LMIC-specific challenges, including delayed healthcare-seeking behaviour driven by financial constraints and limited healthcare access (51), and the limited availability of advanced cardiac care facilities (52, 53). However, the larger mortality effects observed in our analysis are based on considerably fewer studies (typically 2–3 studies per pollutant) compared with hospital admission outcomes (10–12 studies per pollutant), which could influence the reliability and precision of the mortality estimates. For $PM_{2.5}$ mortality in particular, high heterogeneity was largely driven by a single Iranian study that reported substantially larger effects than others, meaning the pooled estimate should be interpreted with caution until further studies are available. The higher mortality effects might also indicate that air pollution in LMICs has more severe cardiovascular consequences, possibly due to a more susceptible population or synergistic interactions with other risk factors such as household air pollution, occupational exposures, climate factors and co-morbidities (54).

An interesting finding from our analysis was the differences in risk of hospital admissions between the two-size metrics of particulate matter, with PM_{10} demonstrating stronger associations with cardiovascular admissions (RR: 1.0191; 95% CI: 1.0017–1.0368) compared to $PM_{2.5}$ (RR: 1.0046; 95% CI: 1.0025–1.0067) per 10 μg/m³ increase. Typically, $PM_{2.5}$ shows stronger associations with cardiovascular outcomes than PM_{10} (55), in principle due to the greater penetration of these particles into the lung, their larger particle surface area and, in general, the higher content of harmful constituents of the sources that $PM_{2.5}$ arises from. A few factors might explain the pattern observed. First, when standardising to equivalent mass increments (10 μg/m³), the relative toxicity comparison is influenced by the typical concentration ranges of these pollutants. Given that PM_{10} concentrations are generally higher and more variable in LMIC urban settings, a 10 μg/m³ increment represents a smaller proportional change in exposure for PM_{10} than for $PM_{2.5}$, potentially making direct comparison of effect sizes challenging. Also, in many LMIC settings, the proportion of PM_{10} derived from sources such as road dust, traffic and construction activities may be greater than in HICs, reflecting industrial emissions that are prevalent in rapidly urbanising environments, although it should be recognised that these sources will also contribute to particles in the $PM_{2.5}$ size range. The relative toxicity of the sources of PM in LICs are less well determined than that of urban PM in HIC settings and laboratory sources of combustion-derived PM (55). Thus, there is a need for better attribution of different source contributions, and atmospheric reactions that affect the air pollution mixture people are exposed to, to link to health outcomes, and subsequently support targeted air quality interventions.

Studies performed in Iran constituted the majority of the dataset and were concentrated in urban areas. However, this dominance raises questions about generalisability. Non-Iranian studies, though fewer, showed similar directions of association but tended to report slightly weaker effects for $PM_{2.5}$ and PM_{10}. Iran is also frequently affected by transboundary desert dust storms, which contribute substantially to ambient particulate matter and differ in toxicity from combustion-derived particles more typical of other urban environments (56). WHO has highlighted desert dust as a pollutant warranting separate long-term assessment (57). While the data from Iran is informative, it highlights the paucity of data from other regions. In particular, South Asia and sub-Saharan African regions, home to some of the world's most polluted cities, are underrepresented. This geographic imbalance parallels gaps identified in the GBD study, which notes that 90% of air pollution-related deaths occur in LMICs, yet there is often a dearth of air pollution monitoring stations in low-income regions (58), especially sub-Saharan Africa (22). The few studies from these potentially high-burden regions, where air pollution characteristics and healthcare infrastructure could differ markedly, represent a critical data gap. Our review focused specifically on LICs and lower-middle-income countries, excluding upper-middle-income countries such as China, South Africa, and Brazil, where much of the recent growth in air pollution–CVD research has occurred. This deliberate distinction means that the geographic distribution of studies observed here reflects evidence gaps specific to the lowest-income settings, which previous reviews combining all low and middle-income countries may not have revealed. Also, our review focused on incident (hospitalisation and

mortality) rather than prevalence of cardiovascular risk factors such as hypertension and diabetes. Since these risk factors are themselves influenced by air pollution, through both shared and independent mechanisms, including them alongside incident CVD events would risk double-counting of effects in a pooled analysis. Our focus on incident events also reflects a stronger causal inference framework, with clear temporal associations between air pollutant exposure and cardiovascular outcomes, and reduced potential for confounding.

The observed associations between air pollution and cardiovascular disease are supported by mechanistic studies (15, 59). All the criteria air pollutants investigated can induce oxidative stress and inflammation in the lung, both of which can be propagated to the systemic circulation (15) Several pollutants, but most notably PM, induce a range of cardiovascular impairments including endothelial dysfunction, increased blood pressure, changes to heart rhythm, increased cardiac susceptibility to ischaemia, enhanced blood clotting, impaired fibrinolysis, to name but a few (47). Exposure to air pollution also promotes lipid peroxidation, vascular inflammation and accelerates atherosclerosis, and ultrafine particles may promote atherothrombotic vascular disease by directly accessing the blood and accumulating at sites of vascular inflammation (60). All these mechanisms will increase the risk of developing CVD but also increase the risk of cardiovascular events that lead to the outcomes studied in the current review. CO, a product of incomplete combustion, binds to haemoglobin, reducing oxygen delivery and exacerbating cardiac ischemia (50, 61), a mechanism that may be particularly detrimental in populations with pre-existing anaemia, prevalent in LICs (62). Future studies should ascertain the extent to which these mechanisms contribute to differences in the profile of cardiovascular diseases between LICs, LMICs and HICs, and if this may engender differences in susceptibility to specific air pollutants.

4.1 LIMITATIONS

4.1.1 Limitations of meta-analysis

While our study provides much needed insights from LICs and LMICs, several limitations warrant consideration. First, significant heterogeneity among the included studies owing to varying methodologies contributes substantially to the variability of pooled estimates and suggests the need for caution in generalising findings. Second, we assumed many of the included studies used generalised linear models, which assume a linear relationship between exposure and outcome. This assumption may not always hold true, as the exposure-response relationship is often reported to be supralinear (less steep at very high pollution levels). Third, our standardisation to a 10 $\mu g/m^3$ increment for all pollutants, while facilitating comparisons, may not optimally represent the typical exposure contrasts for each pollutant in LMIC settings. Finally, the predominance of short-term exposure studies in our analysis prevents an adequate assessment of the long-term cardiovascular consequences of chronic air pollution in these settings.

4.1.2 Limitations of systematic review

Our systematic review also has limitations. Although we used comprehensive search strategies, we may have missed relevant studies published in non-English languages or in journals not indexed in the databases searched. The substantial geographic concentration of studies in Iran limits generalising of our findings to other LICs and LMICs with different pollution profiles, population characteristics, and healthcare systems. The relatively small number of studies reporting certain outcomes (particularly mortality) resulted in less precise pooled estimates for these endpoints. This review is limited to incident events (hospitalisation and mortality) rather than prevalence and risk factors to ensure results reflect a causal inference framework (as from study protocol).

4.2 FUTURE RESEARCH DIRECTIONS

Advancing research on air pollution and cardiovascular disease in LICs and LMICs requires a paradigm shift toward longitudinal, context-specific, and interdisciplinary approaches. Prioritising longitudinal cohort studies in regions with a high pollution burden will help to clarify the cumulative risks of chronic exposure in settings with a different socioeconomic status and healthcare disparities. It is highly likely that air pollution sources and PM composition will differ

from HICs, yet assessment of this in LICs and LMICs (and between locations with different degrees of urbanisation) has been minimal. Source apportionment methodologies would help disentangle region-specific contributors such as industrial emissions, traffic, or agricultural burning, although such studies would require sufficient support to perform adequately. Not only would this information provide insight into which air pollutants are most harmful, but it would help in the design of targeted mitigation strategies to maximise potential health benefits for given resources. Population characteristics will differ between nations, including demographics that may be more vulnerable to air pollution exposure and health statuses that incur biological susceptibility to air pollutants. Mapping population demographics, even as far as profiling of gene variants that confer greater susceptibility, would contextualise air pollutant-outcome risk scores by clarifying the extent to which certain populations or individuals are biologically more susceptible to air pollution, thus enabling targeted public health interventions and more precise risk assessments.

5. CONCLUSION

This systematic review and meta-analysis found significant associations between ambient air pollution and CVD in LICs and LMICs, complementing the findings of studies in higher income regions. High variability was observed for risk ratios due to the relatively limited number of studies identified and the different approaches used within them. However, given the overall positive associations between several air pollutants and cardiovascular mortality and hospital admission for CVD, the high levels of air pollution, as well as the structural and health inequities observed in many LICs and LMICs, suggest that the burden of air pollution on cardiovascular health is likely to be substantial. While a growing body of evidence on air pollution and cardiometabolic risk factors exists in broader LMIC settings, substantial gaps remain in studies reporting clinical CVD events in the LIC. Thus, there is a clear need for action on air pollution in LICs and LMICs, in terms of providing the evidence base to establish the risks of different sources of air pollution that exert the greatest effect on health. Doing so would greatly aid the justification of resources to improve air quality through policies that will provide a concerted approach of ground-level intervention, public awareness and appropriate health care provision.

DATA ACCESSIBILITY STATEMENT

Data extracted for this meta-analysis, including summary estimates and data dictionary, will be available upon request to the corresponding author following publication. Requests should be accompanied by a research proposal detailing the intended use and require approval from the study authors, along with a signed data-access agreement. The study protocol is publicly available via PROSPERO (registration number: CRD42023484929).

ADDITIONAL FILE

The additional file for this article can be found as follows:

- **Supplementary Materials.** Full search strategy, supplementary figures (S1–S5), supplementary table (S1), and risk of bias assessment details. DOI: https://doi.org/10.5334/gh.1545.s1

ETHICS AND CONSENT

Ethical approval was not required for this study as it is a systematic review and meta-analysis of previously published data. No individual participant data were collected or used.

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COMPETING INTERESTS

Pablo Perel is Executive Editor of Global Heart. We confirm that all other authors are not current or former (within the past three years) members of the Global Heart editorial team or board. All authors have no competing interests to declare.

AUTHOR CONTRIBUTIONS

MA, MRM, and MDC conceived and designed the study. MA and SR performed the literature searches and independently screened studies, with discrepancies resolved by SS. MA, NE, SR and SS extracted data, conducted the statistical analysis, and drafted the initial manuscript. NE, SR, ST, SS, PP, AS, MDC, and MRM contributed substantially to interpreting results, critically revised the manuscript for important intellectual content, and provided methodological guidance. No authors were prohibited from accessing the data. All authors reviewed and approved the final manuscript, and MRM and MDC had final responsibility for the decision to submit the manuscript for publication.

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