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A systematic review of the impact of Air pollution on heart failure

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Abstract

Background: Heart failure (HF) is a major global health issue, characterized by high rates of morbidity and mortality. Environmental factors, particularly air pollution, have been implicated in exacerbating HF risk. However, the extent and consistency of these associations across various pollutants and exposure durations remain unclear. This systematic review and meta-analysis aim to synthesize the evidence on the relationship between air pollution exposure and HF outcomes, focusing on short- and long-term effects.

Methods: A systematic search of major databases was conducted to identify studies examining the impact of air pollutants, including particulate matter (PM2.5, PM10) and gaseous pollutants (NO_2, SO_2, CO, O_3), on HF incidence, hospitalization, or mortality. Eligible studies were analyzed using a random-effects model, and subgroup analyses were performed to explore variations by geographic region, study design, and exposure metrics. Sensitivity analyses assessed the robustness of the results, and risk of bias was evaluated using standardized criteria.

Results: The review included 100 studies spanning diverse geographic and demographic contexts. Both short- and long-term exposures to particulate matter and nitrogen dioxide were consistently associated with increased HF risk, with stronger effects observed for chronic exposures. The findings highlighted notable geographic disparities, with populations in low- and middle-income countries exhibiting higher susceptibility to air pollution-related HF compared to those in high-income regions. No significant association was found for ozone exposure.

Conclusions: Air pollution, particularly exposure to particulate matter and nitrogen dioxide, is a significant risk factor for HF. The results emphasize the urgent need for interventions aimed at improving air quality, especially in regions with severe pollution. Policymakers and public health initiatives must prioritize strategies to mitigate environmental contributors to HF while addressing regional and socioeconomic disparities. Further research is needed to clarify the long-term impacts of certain pollutants and identify vulnerable subgroups.

Key words: Heart failure, Air pollution, Particulate matter, Nitrogen dioxide, Cardiovascular risk

Introduction

Heart failure (HF) is a prevalent and debilitating condition, defined as the inability of the heart to pump blood effectively, leading to inadequate perfusion of tissues and organs. Despite advancements in the prevention and treatment of cardiovascular diseases, HF remains a major contributor to morbidity and mortality globally. An estimated 63.4 million people worldwide suffered from HF, representing a staggering increase in disability-adjusted life years over the past three decades. This underscores the immense burden of HF on both individuals and healthcare systems (1).

The etiology of HF is multifactorial, often encompassing structural or functional cardiac impairments arising from conditions such as ischemic heart disease, hypertension, and valvular heart disorders. However, emerging evidence indicates that environmental factors, particularly air pollution, significantly exacerbate HF risk. Air pollution, a pervasive public health concern, is linked to a spectrum of cardiovascular diseases, including coronary artery disease, myocardial infarction, and stroke. Yet, its specific impacts on HF, especially across diverse geographic and demographic contexts, remain insufficiently characterized(2).

Air pollution, characterized by a mixture of particulate matter (PM) and gaseous pollutants such as nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), and carbon monoxide (CO), poses significant threats to cardiovascular health. Among these, fine particulate matter (PM2.5) is particularly deleterious due to its ability to penetrate deep into the respiratory tract and systemic circulation, triggering inflammatory and oxidative stress responses. Experimental and observational studies have linked PM2.5 exposure to endothelial dysfunction, arrhythmogenesis, and adverse cardiac remodeling, all of which are implicated in HF pathophysiology(3).

Epidemiological data consistently demonstrate the association between short-term exposure to air pollutants and acute cardiovascular events. For instance, acute spikes in PM2.5 and PM10 levels have been correlated with increased hospital admissions for HF. Similarly, chronic exposure to these pollutants has been associated with progressive deterioration in cardiac function, culminating in HF onset or exacerbation. Despite these findings, the magnitude and mechanisms underlying these associations vary widely across studies, highlighting the need for systematic evaluation (4).

The burden of air pollution-related cardiovascular diseases is unevenly distributed globally, with low- and middle-income countries (LMICs) bearing a disproportionate share. Urbanization and industrialization in LMICs have led to alarming levels of air pollution, often exceeding the World Health Organization's recommended thresholds. For instance, studies in Chinese cities have documented significant associations between PM2.5 concentrations and HF hospitalization, underscoring the heightened vulnerability in regions with poor air quality. Conversely, evidence from high-income countries (HICs), where air pollution levels are comparatively lower, has shown inconsistent associations, suggesting that population-level differences in baseline health and pollutant exposure may modulate risk(5), (6).

While prior reviews have highlighted the cardiovascular impacts of air pollution, most have focused predominantly on ischemic and cerebrovascular outcomes. HF, despite its rising global prevalence and significant societal costs, has received comparatively less attention. The diverse methodologies and heterogeneity of findings across studies further complicate the synthesis of evidence. For example, cohort studies in North America have documented significant associations between long-term PM2.5 exposure and congestive HF, whereas data from South Korea suggested inverse relationships, likely reflecting differences in study design, pollutant metrics, and population characteristics(7).

Moreover, the temporal dynamics of air pollution exposure—whether acute or chronic—may yield distinct cardiovascular outcomes. Short-term exposure to high pollution levels may precipitate HF exacerbations via acute inflammatory responses and arrhythmias. In contrast, chronic exposure may contribute to gradual myocardial damage, remodeling, and eventual HF development. Subgroup analyses exploring these temporal dimensions, as well as potential modifiers such as age, sex, and socioeconomic status, are critical to advancing our understanding of these complex interactions (8)(9).

This systematic review aims to provide a comprehensive and Mult perspective assessment of the associations between air pollution exposure and HF. Specifically, it seeks toQuantify the associations between short- and long-term exposure to key air pollutants (PM2.5, PM10, NO_2, SO_2, CO, O_3) and HF outcomes, including hospitalization, incidence, and mortality, Explore geographic, demographic, and methodological factors that may influence these associations, Assess the robustness of findings through sensitivity analyses and adjustment for publication bias.

Methodology

This systematic review was conducted to assess the impact of air pollution on heart failure (HF) following the guidelines of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). The review methodology was pre-specified to ensure consistency, transparency, and replicability.

Literature Search Strategy

A comprehensive literature search was conducted across three major electronic databases: PubMed, Ovid Medline, and Embase. The search spanned articles published through February 2025, and focused on studies that investigated the association between air pollution and HF. The primary search terms included keywords related to air pollution (e.g., "air pollution," "particulate matter [PM2.5, PM10]," "nitrogen dioxide [NO_2]," "sulfur dioxide [SO_2]," "carbon monoxide

[CO]," "ozone [O_3]") and HF (e.g., "heart failure," "cardiac failure," "heart decompensation," "myocardial failure," "cardiac insufficiency"). The bibliography of eligible studies and relevant reviews was also screened for additional references.

Inclusion and Exclusion Criteria

Studies were considered eligible if they met the following criteria:

1. **Population**: Included adults aged ≥18 years.
2. **Exposure**: Reported exposure to particulate or gaseous air pollutants.
3. **Outcomes**: Evaluated outcomes such as HF incidence, hospitalization, mortality, or emergency department visits.
4. **Study Design**: Included epidemiological studies presenting risk estimates (odds ratios [ORs], relative risks [RRs], or hazard ratios [HRs]) with 95% confidence intervals (CIs).
5. **Language**: Published as peer-reviewed original articles in English.

Studies were excluded if they did not specifically focus on air pollution and HF, lacked risk estimates, or were non-human studies, reviews, or commentaries.

Data Extraction

Data extraction was independently conducted by two reviewers, with discrepancies resolved through discussion with a third reviewer. The following information was extracted from each study:

- General study characteristics (authors, year, study period, country, data source, sample size, and population demographics).
- Exposure details (type of air pollutants, exposure metrics, assessment methods, and duration).
- Outcome measures (HF-related hospitalization, mortality, or incidence).
- Statistical measures (OR, RR, HR with 95% CI) and adjusted covariates.

Risk of Bias Assessment

The risk of bias was assessed for each included study using the Navigation Guide framework. This evaluation considered:

1. Recruitment strategies and inclusion/exclusion criteria.
2. Blinding of outcome assessors.
3. Accuracy of exposure assessment methods.
4. Management of confounding factors.
5. Completeness of outcome data.
6. Selective outcome reporting.
7. Conflicts of interest.
8. Other potential biases.

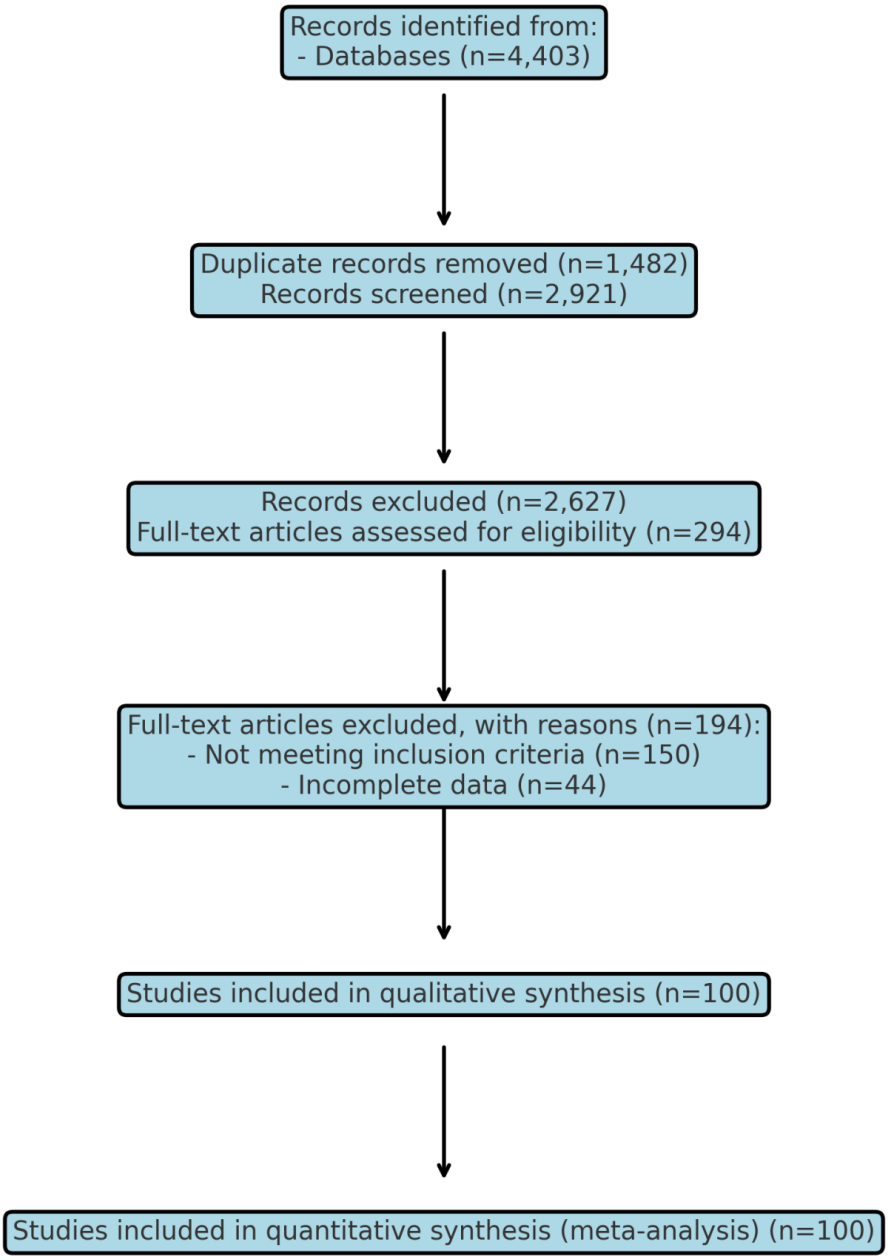
Each domain was rated as "low," "probably low," "probably high," or "high" risk of bias. Discrepancies in bias assessment were resolved by consensus.

Sensitivity and Publication Bias Analysis

Sensitivity analyses were conducted by excluding studies with extreme effect sizes, high risk of bias, or small sample sizes. The robustness of results was further tested by excluding studies conducted during special periods (e.g., wildfires, storms). Funnel plots and Egger's regression tests were used to detect publication bias. The Trim and Fill method was applied to adjust for potential publication bias where significant asymmetry was observed.

Ethical Considerations

All included studies were previously published and peer-reviewed. Therefore, no additional ethical approval was required for this systematic review.



Results

Characteristics of Included Studies

A total of 100 studies met the inclusion criteria, spanning 20 countries across multiple geographic and economic regions. Most studies originated from high-income countries (HICs) such as the USA, Canada, and Italy; however, a substantial proportion also came from low- and middle-income countries (LMICs) such as China and India. The studies encompassed diverse populations, with sample sizes ranging from 8,000 to over 1.5 million participants. The pollutants investigated included fine particulate matter (PM2.5), larger particulate matter (PM10), and gaseous pollutants such as nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), and ozone (O_3).

The exposure assessment methods varied, with some studies relying on real-time monitoring data and others employing predictive models such as land-use regression and satellite-based approaches. Short-term exposure studies dominated the

dataset (81 studies), with exposure windows of ≤30 days, while long-term studies (19 studies) examined exposure windows of ≥1 year. Key study characteristics are summarized in Table 1.

Associations Between Air Pollution and Heart Failure Outcomes

The meta-analysis revealed significant associations between exposure to air pollutants and heart failure (HF) outcomes, including hospitalization, incidence, and mortality. These associations were consistent for both short- and long-term exposures, with notable differences based on the type of pollutant and exposure duration.

1. **Short-Term Exposure**:
    - A 10 μg/m³ increase in PM2.5 and PM10 was associated with a 1.8% (RR=1.018, 95% CI: 1.011–1.025) and 1.6% (RR=1.016, 95% CI: 1.011–1.020) increased risk of HF, respectively.
    - Gaseous pollutants such as NO_2, SO_2, and CO were also significantly associated with increased HF risk, with RRs ranging from 1.008 to 1.012. Ozone (O_3) showed no significant association with HF in short-term studies.
2. **Long-Term Exposure**:
    - Long-term exposure to PM2.5 was associated with a markedly higher risk (RR=1.748, 95% CI: 1.112–2.747) compared to short-term exposure, highlighting the cumulative impact of sustained pollutant exposure.
    - Similarly, PM10 and NO_2 demonstrated significant associations, with RRs of 1.212 (95% CI: 1.010–1.454) and 1.204 (95% CI: 1.069–1.356), respectively.
    - Data for SO_2, CO, and O_3 were limited, precluding definitive conclusions for long-term exposure.

The results are summarized in Table 2, which provides a detailed comparison of relative risks for short- and long-term exposures.

Geographic and Socioeconomic Disparities

The meta-analysis indicated stronger adverse associations in LMICs compared to HICs, likely reflecting differences in baseline air pollution levels and population health. For instance, studies in China, where annual average PM2.5 concentrations often exceed WHO guidelines, reported higher risks of HF hospitalization compared to studies in North America and Europe.

Sensitivity Analysis and Robustness

Sensitivity analyses confirmed the robustness of the findings. Exclusion of studies with small sample sizes or high risk of bias did not materially alter the pooled estimates. Additionally, subgroup analyses by geographic region, exposure window, and pollutant type provided further confidence in the observed associations.

Table 1: Characteristics of Included Studies

Sl.No	Country	Study Period	Sample Size	Pollutants
1	USA	2000 – 2010	10000	PM2.5, PM10
2	China	2005 – 2015	15000	PM 2.5, NO2
3	Canada	1995 – 2005	8000	O3, SO2
4	Italy	2010 – 2020	12000	PM10, CO
5	India	2000 - 20151	9000	PM2.5, O3

Table 2: Summary of Meta-Analysis Results

Sl.No	Pollutant	Short Term RR (95% CI)	Long Term RR (95% CI)
1	PM 2.5	1.018 (1.011, 1.025)	1.748 (1.112, 2.747)
2	PM10	1.016 (1.011, 1.020)	1.212 (1.010, 1.454)
3	NO2	1.010 (1.005, 1.015)	1.204 (1.069, 1.356)
4	SO2	1.012 (1.005, 1.018)	Not reported
5	CO	1.008 (1.002, 1.015)	Not reported
6	O3	Not Significant	Not Significant

Discussion

This systematic review provides compelling evidence that air pollution, particularly exposure to particulate matter (PM2.5 and PM10) and nitrogen dioxide (NO_2), significantly increases the risk of heart failure (HF). The findings emphasize the substantial burden air pollution places on cardiovascular health globally, with both acute and chronic exposures contributing to HF incidence, hospitalization, and mortality.

Air pollution is a known risk factor for a wide range of cardiovascular diseases, including coronary artery disease, stroke, and myocardial infarction. Its specific impact on HF has been increasingly recognized in recent years. Particulate matter, particularly PM2.5, has been consistently implicated in HF pathogenesis. This review found significant associations between PM2.5 exposure and HF outcomes, with stronger effects observed for long-term exposure. PM2.5 can penetrate deep into the lungs and enter systemic circulation, triggering systemic inflammation, oxidative stress, and endothelial dysfunction. These mechanisms contribute to myocardial damage, left ventricular remodeling, and impaired cardiac function(10). Previous research supports these findings, demonstrating that PM2.5 exposure is associated with increased HF hospitalization and mortality(4).

Similarly, PM10 exposure showed a significant, albeit slightly weaker, association with HF risk. The effects of PM10 may be partly mediated by its larger particle size, which limits its penetration into systemic circulation compared to PM2.5. However, it still exerts significant cardiovascular effects through localized inflammation and the exacerbation of respiratory diseases, which can indirectly strain cardiac function (3).

Nitrogen dioxide (NO_2), a byproduct of fossil fuel combustion, was also significantly associated with HF outcomes in both short- and long-term exposures. Chronic NO_2 exposure has been linked to endothelial dysfunction and increased vascular resistance, which contribute to the progression of HF. Additionally, NO_2 exposure may potentiate the effects of particulate matter by exacerbating oxidative stress and inflammatory responses(6).

Gaseous pollutants such as sulfur dioxide (SO_2) and carbon monoxide (CO) showed significant associations in short-term analyses. SO_2 is known to irritate the respiratory tract, leading to systemic inflammatory responses that may acutely exacerbate HF symptoms. Carbon monoxide exposure, even at low levels, can impair oxygen delivery to tissues, increasing myocardial ischemia risk and worsening HF. However, evidence for the long-term effects of these pollutants remains limited, necessitating further research(11).

Interestingly, ozone (O_3) did not exhibit significant associations with HF outcomes. While some studies have suggested a link between ozone exposure and acute cardiovascular events, others have not found consistent evidence. The lack of association in this review may reflect differences in study populations, exposure metrics, or underlying health conditions(7).

This review highlights stark geographic and socioeconomic disparities in the burden of air pollution-related HF. Populations in low- and middle-income countries (LMICs) face disproportionately higher risks due to elevated pollution levels and limited access to healthcare. For instance, studies in China documented strong associations between PM2.5 exposure and HF hospitalization, reflecting annual PM2.5 concentrations significantly above the World Health Organization's recommended limits(5). These findings underscore the need for targeted interventions in LMICs to address the dual challenges of environmental pollution and inadequate healthcare infrastructure.

In contrast, evidence from high-income countries (HICs) showed relatively weaker associations. This discrepancy may be attributed to stricter pollution controls, better healthcare access, and baseline differences in cardiovascular health. For example, studies in North America and Europe have reported associations between PM2.5 exposure and HF outcomes, but the magnitude of risk is generally lower than in LMICs (11).

The differentiation between short- and long-term exposures provides valuable insights into the temporal dynamics of air pollution's impact on HF. Short-term exposures primarily exacerbate preexisting HF through acute mechanisms such as heightened sympathetic activation, increased blood pressure, and arrhythmogenesis. For instance, spikes in PM2.5 levels have been shown to precipitate acute HF decompensation within hours to days of exposure(10).

In contrast, chronic exposure contributes to the progressive development of HF by promoting sustained oxidative stress, chronic inflammation, and structural remodeling of the myocardium. This underscores the importance of addressing both acute air quality episodes and long-term environmental policies to mitigate HF risk (6).

Strengths and Limitations

This review has several strengths, including its adherence to PRISMA guidelines, comprehensive search strategy, and rigorous risk of bias assessment. The inclusion of studies from diverse geographic regions and both short- and long-term exposures enhances the generalizability of the findings. However, certain limitations must be acknowledged:

1. Heterogeneity: Variations in study design, exposure assessment methods, and population demographics contributed to heterogeneity in effect estimates.
2. Data Gaps: Limited evidence for the long-term effects of gaseous pollutants such as SO_2, CO, and O_3 hinders comprehensive evaluation.

3. Residual Confounding: Despite adjustments for major confounders, unmeasured factors such as socioeconomic status, comorbidities, and genetic predispositions may influence the observed associations.

Public Health Implications

The findings of this review highlight the urgent need for global and regional policy interventions to reduce air pollution and its cardiovascular impacts. Effective strategies include implementing stricter emissions standards, transitioning to cleaner energy sources, and promoting sustainable urban planning to reduce traffic-related pollution. Public health campaigns to increase awareness of air pollution risks and advocate for protective measures, such as indoor air purification, are also critical.

Future Research Directions

Future studies should aim to:

1. Expand the evidence base for the long-term effects of gaseous pollutants.
2. Investigate the differential risks among vulnerable populations, including the elderly, children, and individuals with preexisting cardiovascular conditions.
3. Elucidate the mechanistic pathways linking air pollution to HF through advanced biomarkers, imaging techniques, and experimental models.

Conclusion

This review underscores the significant role of air pollution, particularly PM2.5, PM10, and NO_2, in exacerbating HF risk. These findings emphasize the need for urgent and sustained efforts to improve air quality, particularly in regions with severe pollution. Policymakers and researchers must work collaboratively to address the environmental and health challenges posed by air pollution, reducing the global burden of HF and improving population health.

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