ORIGINAL RESEARCH ARTICLE

Diponegoro International Medical Journal 2025 December, Vol 6, No.2: 54-60
e-ISSN: 2745-5815

The Impact of Particulate Matter Air Pollution on Coronary Heart Disease: A Systematic Review

Gabriela Valencia Putri Husodho[1], Muhammad Abi Ghoffari Siregar[2]*, Agyta Hanifa Faiza[1]

[1]Faculty of Medicine, Universitas Diponegoro, Semarang, Indonesia
[2]Biomedical Science Masters Program, Faculty of Medicine, Universitas Andalas, Padang, Indonesia

Keywords:

Air pollution
Coronary heart disease
Particulate matter
Quality of life

*) Correspondence to:
abisiregaar@gmail.com

Article history:

Received 17-10-2025
Accepted 22-12-2025
Availableonline 23-12-2025

ABSTRACT

Background: Coronary heart disease remains a major global health burden. Ambient particulate matter air pollution is a modifiable exposure, and particles smaller than 2.5 micrometers and particles smaller than 10 micrometers have been linked to cardiovascular morbidity and mortality. This systematic review synthesized recent observational evidence on particulate matter exposure and coronary heart disease.
Objective: To assess the impact that PM2.5 and PM10 have on coronary heart disease.
Methods: PubMed, ScienceDirect, and EBSCO were searched for English-language observational studies published from 2020 to 2025 that evaluated ambient particulate matter exposure and coronary heart disease outcomes. Case reports and studies with insufficient extractable data were excluded. Reporting followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guideline.
Results: Forty-six studies were included. Twenty-four evaluated particles smaller than 2.5 micrometers only, and twenty-two evaluated both size fractions; none assessed particles smaller than 10 micrometers alone. Most studies were conducted in China. Higher particulate matter exposure was generally associated with higher risk of coronary heart disease and poorer clinical outcomes, including increased hospitalization and mortality. Effect estimates were typically larger for particles smaller than 2.5 micrometers.
Conclusion: Observational evidence published from 2020 to 2025 indicates that particulate matter exposure, particularly particles smaller than 2.5 micrometers, is associated with higher risk of coronary heart disease and adverse clinical outcomes.

DIMJ, 2025, 6(2), 54-60 DOI: https://doi.org/10.14710/dimj.v6i1.29648

1. Introduction

Coronary heart disease (CHD) remains the leading cause of death worldwide and a major source of long-term disability. In 2022, an estimated 315 million people were living with CHD, corresponding to an age-standardized prevalence of 3,605 per 100,000 population. Although age-standardized prevalence has declined since 1990, the absolute number of affected individuals continues to rise, largely driven by population growth and ageing. In 2021, CHD was responsible for approximately 9 million deaths globally, and projections suggest that the overall cardiovascular burden may increase substantially by mid-century if current trajectories persist.[1,2]

Beyond established clinical risk factors, environmental exposures have emerged as important and potentially modifiable determinants of CHD, with ambient air pollution receiving particular attention. Fine particulate matter, especially particles smaller than 2.5 micrometers and particles smaller than 10 micrometers, is biologically plausible as a contributor to atherothrombotic disease through pathways involving systemic inflammation, oxidative stress, autonomic imbalance, and endothelial dysfunction. In 2019, air pollution was associated with 4.2 million deaths worldwide, many from cardiovascular causes.[3] Observational evidence from diverse settings has linked particulate matter exposure with higher rates of CHD-related hospitalization, mortality, and years of life lost, underscoring the relevance of air quality as a population-level target for prevention.[4–6]

The socioeconomic implications of CHD extend far beyond clinical outcomes, inflicting a heavy toll on national health systems, productivity, and social equity. The United States projects a threefold increase in cardiovascular-related economic burden by 2050, with healthcare costs alone expected to quadruple. Similar trends are evident across Europe, where annual CHD-related expenditures are estimated at €282 billion.[7] In parallel, CHD amplifies health inequities. Communities facing socioeconomic disadvantage often experience higher exposure to environmental hazards, greater barriers to preventive care, and delayed access to effective treatment, resulting in disproportionate risk and poorer outcomes.[8] In Indonesia, CHD-related productivity losses have been estimated to exceed 1.5 million productivity-adjusted life years,

translating into economic losses of more than US$33 billion, highlighting the tangible consequences for households and national development.[9]

Effective reduction of CHD burden requires integrated strategies that combine clinical prevention across the life course with policies that address upstream determinants. While individual-level interventions remain essential, population-level measures that improve air quality, including emissions control and urban planning that supports healthier environments, offer broad and durable cardiovascular benefits.[10,11] Modelling suggests that relatively modest reductions in particulate matter concentrations could avert large numbers of cardiovascular events, reinforcing the potential value of environmental policy reform.[12]

These approaches are especially important in low- and middle-income countries, where most cardiovascular deaths occur and health systems may face persistent gaps in diagnostics, essential medicines, and workforce capacity.[13,14] In this context, scalable, evidence-based packages and coordinated government action have demonstrated promise in improving cardiovascular outcomes.[15]

Against this background, this study examines the impact of ambient particulate matter exposure on the risk of coronary heart disease to inform prevention strategies and policy responses that are clinically meaningful, equitable, and feasible across settings.

2. Methods

Study Design

This study was a systematic review conducted to synthesize existing evidence on the effect of particulate matter pollution on the occurrence of coronary heart disease or ischemic heart disease. This design was chosen as it allows for comprehensive identification, analysis, and evaluation of relevant studies, providing a robust foundation for understanding the topic. We conducted this systematic review from May 2025 to July 2025.

Research question

Our primary research question was formulated using the Population, Exposure, Comparison, and Outcome (PECO) framework.[16] Population: Patients with Coronary Heart Disease (CHD) or Ischemic Heart Disease (IHD), Exposure: Exposure to particulate matter (PM) air pollution as an external environmental factor, Comparison: Patients with CHD who are not or have low exposure to air pollution, or have good air quality conditions (if applicable), Outcome: mortality, morbidity, and risk of development of CHD/IHD. Our study was guided by the following research question: how does particulate matter air pollution affect the occurrence of coronary heart disease or ischemic heart disease?

Search strategy

A comprehensive search strategy was developed to capture a wide array of studies addressing this topic. Search terms were constructed using the keywords ("Air pollution" or "airborne pollution" or particulate matter") and ("cardiovascular" or "heart function" or "cardiac function" or “coronary heart disease” or “ischemic heart disease”). Boolean operators AND/OR were utilized to ensure precision and inclusivity. We searched three databases, including PubMed, ScienceDirect and EBSCO, to identify relevant literature. We employed the following inclusion and exclusion criteria in choosing the articles:

- Inclusion criteria
  - Articles using English and limited to Scopus-indexed journals from 2020-2025.
  - The search engine databases used were PubMed, ScienceDirect and EBSCO
  - Full-text articles
- Exclusion criteria
  - Articles solely comprised of abstracts
  - Studies that do not align with the investigation of particulate matter air pollution in coronary heart disease
  - Replication of articles
  - Articles containing inadequate data

Screening of the article

All articles retrieved through database searches were imported into Rayyan.ai for consolidation and deduplication. Subsequently, independent reviewers among MAS, AHF, and GVPH conducted a three-step screening process. 1) Title Screening: Titles were screened for relevance; 2) abstract screening: Abstracts were evaluated to confirm alignment with the inclusion criteria; 3) full-text screening: Eligible studies underwent thorough full-text analysis to finalize the selection. Discrepancies were resolved through discussion and consultation

Data extraction

A standardized data extraction form agreed upon by the authors was used to systematically collect from the included studies. Data collected from the articles included the following: author, year of publication, type of particulate matter pollution, country, risk of development of CHD/IHD, morbidity data, and mortality data. Morbidity data included quality of life parameters including YLLs (years of life lost), DALYs (disability adjusted life years), and YLDs (years lived with disability).

Quality appraisal of the included studies

The risk of bias was assessed using the Risk of Bias in Non-randomized Studies of Exposures (ROBINS-E) tool.[17] Authors independently assessed the studies based on the tool's criteria. Any discrepancies were resolved by discussion. In each study, bias was assessed in seven domains: confounding, measurement of the exposure, selection of participants, deviations from intended interventions, missing data, outcome measurement, and selection of reported results. Bias in each domain was categorized as low, some concerns, high, or very high. Finally, the prior assessment results were used to determine the overall risk of bias in each study.

Data analysis

A conventional inductive content analysis approach was employed in the data analysis process, resulting in the decision to categorize results based on particulate matter pollution type and its corresponding disease risk, morbidity, and mortality data. A formal meta-analysis or quantitative analysis was not done due to the heterogeneity of outcomes in the studies, hence the results were presented in a descriptive manner.

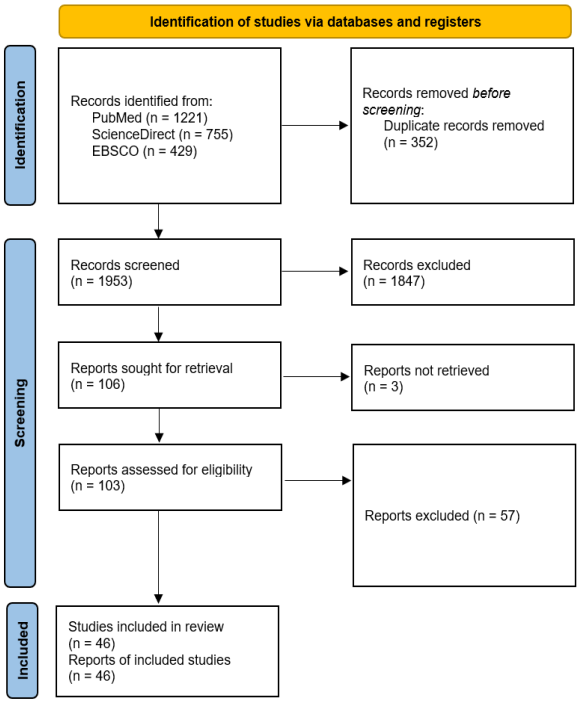

Figure 1. PRIMA Flowchart for Selection of Articles

3. Result

Forty-six studies were included in the final synthesis. (Figure 1.) Quality assessment using the ROBINS-E tool showed thirty-six articles having low risk of bias, six with some concerns, three with high risk of bias, and one with very high risk of bias. (Figure 2.) Even though quality assessment results did not affect exclusion of certain articles, it is still important to note each studies' risk of bias.

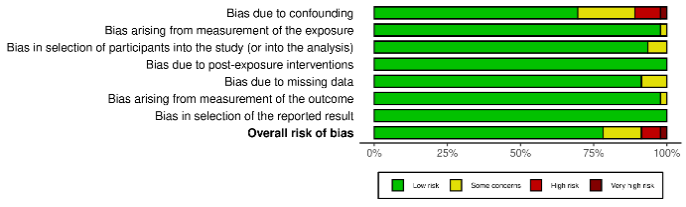

Figure 2. Summary plot of ROBINS-E quality assessment

24 studies discussed PM2.5, 22 studies discussed both PM2.5 and PM10, while none of the studies discussed only PM10. Lack of research on only PM10 is most likely due to less evidence on PM10 having an impact on cardiovascular disease, as smaller particles are more commonly seen in CHD pathogenesis. The majority of the studies were conducted in China (21 studies). Other countries in which the research was conducted in include: Canada (1), Germany (1), India (1), Iran (1), Korea (1), Libya (1), Mexico (1), Peru (1), Poland (2), Serbia (1), South Korea (2), Sweden (2), United Kingdom (5), United States (4), and Taiwan (1). Odds ratio, hazards ratio, and relative risk of CHD were higher overall in PM2.5 exposures compared to PM10. Higher mortality and hospitalization rates were also seen with higher rates of PM exposure, where exposure rates for the majority of studies were categorized into low-moderate (≤25 μg/m³) and high (>25 μg/m³). Burden of disease parameters namely YLL, DAYL, and DYL also showed high significant results, indicating high morbidity for CHD patients due to PM exposure.

4. Discussion

Particulate Matter 2.5 (PM2.5)

Particulate matter with an aerodynamic diameter of ≤2.5 μm (PM2.5) is a major component of ambient air pollution. These particles are small enough to penetrate deep into the lungs and enter the bloodstream. Both acute and chronic exposure of PM 2.5 can trigger pulmonary oxidative stress, systemic inflammation, endothelial dysfunction, vasoconstriction, cardiac electrical alterations, and thrombosis. All of which are linked to an increased risk of cardiovascular diseases, including coronary heart disease (CHD). Due to these significant health implications, the relationship between air pollution and cardiovascular risk has gained considerable attention in both scientific research and public media.[18–22]

Increased levels of PM2.5, particularly above 5.48 μg/m³, have been linked to a higher risk of developing ischemic heart disease.[23] A 10 μg/m³ rise in PM2.5 levels corresponds to a 10% increase in IHD occurrence.[24] Among patients with lower respiratory tract infections (LRTIs), 9.13% developed coronary heart disease (CHD), and those individuals exhibited higher PM2.5 exposure, elevated inflammatory markers, older age, lower BMI, and a greater prevalence of atrial fibrillation. Notably, LRTI patients also had a significantly increased risk of coronary artery thrombosis (OR: 2.49; CI: 1.35–4.60), suggesting a synergistic effect between respiratory infections and air pollution in the development of cardiovascular complications.[25] In environments with higher walkability, the adverse effects of PM2.5 on the risk of coronary heart disease (CHD) may be mitigated.[3] This aligns with evidence showing that increased physical activity is associated with a reduced risk of ischemic heart disease (IHD) among patients with dyslipidemia.[26]

Air pollution is widely recognized as a major global health issue, with fine particulate matter (PM2.5) being the pollutant most consistently linked to increased morbidity and mortality.[27–29] Two main forms of $PM_{2.5}$ have been quantified in the Global Burden of Disease (GBD) 2019, namely, ambient particulate matter (APM, exposure to $PM_{2.5}$ in the outdoor air) and household particulate matter (HPM, exposure to $PM_{2.5}$ due to solid fuel use). They present correlations with socio-demographic development, in which APM exposure increases in the low-to-moderate

socio-demographic index (SDI), while HPM steadily decreases with socioeconomic development.[30]

In Southeastern China, the number of CHD deaths due to APM exposure from 1990 to 2019 increased by 4.2 times. CHD mortality due to APM and HPM exposures were higher in men than women. CHD mortality also increased in the elderly, especially in those over 60 years.[30] Fine particulate matter (PM2.5) components generated by fossil fuel combustion have been more associated with increased CVD mortality compared to those derived from soil-derived $PM_{2.5}$.[31] Based on Global Burden of Disease estimates in 2019, such exposure was responsible for around 4.1 million deaths with respiratory and cardiovascular diseases, cancer, and diabetes being the main contributors in 2019.[10] PM2.5 was also contributed to approximately 2.1 million premature cardiovascular deaths and 48.5 million lost years of healthy life in 2017.[28]

PM 2.5 exposure was also contributed to increased risk of morbidity and hospitalization rates. Older adults and women appear to be more vulnerable, particularly during the colder seasons.[32] Short term exposure of ambient PM2.5 is associated with increases in emergency room visits for CHD.[29]

Fine particulate matter (PM2.5) is a major environmental risk factor for coronary heart disease (CHD), capable of initiating and worsening cardiovascular pathology through several interrelated mechanisms. Due to their small size (≤2.5 μm), these particles can penetrate deep into the lungs and enter the bloodstream via the alveolar-capillary barrier. Once in circulation, PM2.5 interacts directly with vascular tissues, promoting endothelial injury and systemic inflammation.[33]

Studies show that ultrafine particles rapidly translocate into the blood through processes like macropinocytosis and endocytosis. Within minutes, PM2.5 can be found in systemic organs such as the liver, accumulating in vascular endothelial cells, damaging mitochondria and lysosomes. This penetration is facilitated by increased permeability of the alveolar barrier, allowing direct entry into the vascular system where particles contribute to endothelial dysfunction, platelet activation, and myocardial injury. [34]

A key mechanism of toxicity is excessive production of reactive oxygen species (ROS). PM2.5 triggers oxidative stress via mitochondrial dysfunction, NADPH oxidase activation, and particle-surface interactions. This overwhelms cellular antioxidant defences and damages vascular components.[35] ROS also activates inflammatory pathways like NF-κB, elevating cytokines such as IL-6, TNF-α, and C-reactive protein. Endothelial cells respond by releasing microparticles (markers of apoptosis) and suppressing angiogenic signals, creating an environment that promotes vascular inflammation and dysfunction.[36] Additionally, TLR4-mediated pathways amplify this response, promoting macrophage activation and foam cell formation.[37]

Particulate matter (PM2.5) also accelerates atherosclerosis by impairing endothelial nitric oxide production, increasing vascular permeability, and enhancing lipid infiltration. It promotes foam cell formation from macrophages and smooth muscle cells via the TLR4/NF-κB pathway, encouraging plaque growth.[37] These plaques are often unstable due to reduced collagen and thinner fibrous caps. Long-term exposure is linked to increased coronary artery calcification, with even small PM2.5 increments significantly advancing calcium scores.[38] Pro-thrombotic effects are also evident, including elevated tissue factor, reduced thrombomodulin, and increased platelet aggregation.[39]

Finally, PM2.5 causes direct myocardial toxicity. It damages cardiomyocytes through oxidative stress, membrane disruption, and subclinical ischemia, as reflected by elevated troponin levels in exposed individuals. The combined impact of vascular inflammation, atherosclerosis progression, thrombosis, and cardiac cell injury greatly elevates the risk for CHD events such as myocardial infarction, unstable angina, and sudden cardiac death.[40]

Particulate Matter 10 (PM10)

Particulate matter with aerodynamic diameter ≤10 μm (PM10) exposure poses a significant and well-established risk for coronary heart disease (CHD) through multiple interconnected pathophysiological mechanisms that manifest both acutely and chronically. A 10 μg/m³ rise in PM10 is linked to a 1.69% increase in CHD hospitalizations, 1% rise in myocardial infarction, and 0.23% higher cardiovascular mortality.[41] PM10 triggers systemic inflammation (via cytokines like IL-6, TNF-α), oxidative stress (causing endothelial dysfunction and plaque progression), and autonomic imbalance (e.g., reduced heart rate variability and elevated blood pressure).[42] It also promotes thrombosis and impairs fibrinolysis. These effects are more pronounced in vulnerable groups (e.g., elderly, diabetics) and occur even at levels below WHO guidelines, with both short-term and long-term exposures showing a linear dose-response relationship. This underscores PM10 as a critical and modifiable environmental risk factor for CHD.[43,44]

PM10 exposure is a well-established environmental risk factor for coronary heart disease (CHD), contributing to increased morbidity and mortality through multiple interconnected mechanisms. These include systemic inflammation, endothelial dysfunction, autonomic imbalance, and prothrombotic changes. Inhalation of PM10 triggers the release of inflammatory cytokines (e.g., IL-6, IL-8, IL-1β), impairs nitric oxide–mediated vasodilation, disrupts heart rate variability, and promotes coagulation via tissue factor activation.[45] Epidemiological studies show that every 10 μg/m³ increase in PM10 is associated with a 1.2–1.8% rise in coronary events and up to a 1.97% increase in cardiovascular mortality.[41,46] These effects occur even at PM10 levels below current regulatory standards, especially in vulnerable populations, underscoring the need for urgent public health measures to mitigate this modifiable risk factor.[41,43]

PM10 tends to stay in upper airways, unlike PM2.5, due to its larger size, hence it most likely affects the cardiovascular system indirectly, such as by inducing

pulmonary oxidative stress and inflammatory response, as well as interacting with autonomic nervous system receptors.[45] Its effects are also most likely to be observed after long-term exposure as it accumulates in the cardiorespiratory system; previous research showed that long term PM exposure was associated with inflammation and fibrinolysis markers.[47] Out of all the studies included in this review's result synthesis, it is interesting to note that all articles discussing PM10 also include PM2.5 in their research, as a comparison to PM2.5 is most likely important due to PM2.5 playing a more significant role in CHD pathogenesis.

Limitations and Recommendations for Future Research

As the majority of studies were conducted in China, it is recommended for studies to be conducted in other countries, especially those with high industrialization levels. Research specifically on PM10 exposure was also limited; hence, more studies should also focus on deepening understanding on its PM10 on cardiovascular diseases, especially CHD. The review protocol was not registered in PROSPERO, which is a limitation and may introduce risks of reporting bias.

5. Conclusion

An increase in both particulate matter of size 2.5 and 10 µm exposure is associated with increased risk of coronary heart disease occurrence (odds ratio, relative risk, and hazard ratio), mortality, and hospitalization rates, and decreases quality of life (YLLs, DALYs, and YLDs) in patients. PM2.5 was found to exhibit a more significant and worse effect in comparison to PM10, most likely due to its smaller size.

Ethical Approval

There is no ethical approval.

Conflicts of Interest

The authors declare no conflict of interest.

Funding

No specific funding was provided for this article.

Author Contributions

Conceptualization, GVPH and MAGS; methodology, GVPH; software, GVPH; validation, GVPH, MAGS, and AHF; formal analysis, GVPH, MAGS, and AHF; investigation, GVPH, MAGS, and AHF; resources, MAGS; data curation, GVPH and AHF; writing—original draft preparation, GVPH, MAGS, and AHF; writing—review and editing, GVPH, MAGS, and AHF.

Acknowledgments

This work was supported by the Medical Faculties of Diponegoro University and Andalas University.

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