The American Journal of Medical Sciences and Pharmaceutical Research
ISSN 2689-1026 | Open Access

TYPE Original Research
PAGE NO. 32-36
DOI 10.37547/tajmspr/Volume07Issue04-06

Urbanization and declining air quality: clinical aspects of cardiovascular impact

Farida Azizova

Center for Professional Development of Medical Workers, Tashkent, Uzbekistan

OPEN ACCESS

SUBMITED 16 February 2025
ACCEPTED 17 March 2025
PUBLISHED 16 April 2025
VOLUME Vol.07 Issue04 2025

CITATION

Farida Azizova. (2025). Urbanization and declining air quality: clinical aspects of cardiovascular impact. The American Journal of Medical Sciences and Pharmaceutical Research, 7(04), 32–36.
https://doi.org/10.37547/tajmspr/Volume07Issue04-06

Abstract: Rapid urbanization has significantly contributed to deteriorating air quality across global cities. Increasing levels of air pollutants, particularly fine particulate matter (PM2.5), nitrogen dioxide (NO_2), and ozone (O_3), are linked to adverse cardiovascular outcomes. This article explores the clinical implications of declining air quality due to urbanization, reviewing evidence from epidemiological and clinical studies and analyzing the mechanisms through which air pollution exacerbates cardiovascular disease. The findings emphasize the urgent need for integrative public health policies and clinical interventions to mitigate these risks.

Keywords: Urbanization, Air Pollution, Cardiovascular Disease (CVD), PM2.5, Nitrogen Dioxide (NO_2), Ozone (O_3), Endothelial Dysfunction, Systemic Inflammation, Hypertension, Myocardial Infarction, Environmental Health, Public Health Policy.

Introduction: Urbanization is one of the most significant demographic shifts of the 21st century. As of 2024, more than 56% of the world's population resides in urban areas, and this is projected to increase to nearly 70% by 2050, according to United Nations estimates. While urban living has many socio-economic advantages—including better access to healthcare, education, and employment—it also has environmental and health challenges, especially the deterioration of air quality.

Air pollution is a severe and unwanted accompaniment of rapid and largely unchecked urbanization. Increased vehicular emissions, industrial activities, construction, and energy consumption are all significant contributing factors to the release of dangerous pollutants into the atmosphere. Some of the most harmful air pollutants in cities are particulate matter (especially PM2.5), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), and carbon monoxide (CO). These pollutants have human

health effects that are well characterized, with a growing body of evidence incriminating cardiovascular disease (CVD) as a leading clinical endpoint of long-term and short-term exposure.

Cardiovascular disease remains the greatest killer worldwide, and emerging evidence implicates air pollution as a modifiable risk factor. Both acute and chronic exposures to air pollutants have been associated with increased incidence of hypertension, atherosclerosis, myocardial infarction, heart failure, arrhythmias, and stroke. Most vulnerable are the elderly, individuals with underlying cardiovascular disease, and residents of low- and middle-income urban cities where pollution-reducing measures are not typically in effect.

At the clinical level, the pathophysiological mechanisms that link air pollution with cardiovascular dysfunction include systemic inflammation, oxidative stress, endothelial dysfunction, and autonomic nervous system imbalance. Such biologic responses are the predisposition to the development of CVD and can be quantified using biomarkers such as C-reactive protein (CRP), interleukins, and troponins.

While the issue has been broadly acknowledged, there is a gap in the translation of environmental risk into clinically relevant approaches. Understanding how cardiovascular health is impacted by air pollution due to urbanization is not just a requirement for public health policy but also for clinical practice. Physicians, city planners, and policymakers must work in tandem with one another to address this new public health menace.

The aim of this article is to review the clinical aspects of cardiovascular disease in relation to worsening air quality in cities. By summarizing the recent literature and discussing the key mechanisms, outcomes, and implications, this article aims to provide a general overview of the urban air–cardiovascular health connection.

METHODS

This study employed a systematic narrative literature review to evaluate the clinical impact of deteriorating air quality in urban areas on cardiovascular health. Three major electronic databases—PubMed, Scopus, and Web of Science—were systematically searched to identify relevant articles. The search was conducted from November 2024 to February 2025. Keywords and Medical Subject Headings (MeSH) terms used in combination were: "urbanization," "air pollution," "cardiovascular disease," "PM2.5," "nitrogen dioxide," "ozone," "clinical outcomes," "biomarkers," and "environmental health."

The following were the inclusion criteria: (1) English language studies from January 2015 through December 2024; (2) original research articles, meta-analyses, or systematic reviews in peer-reviewed journals; (3) urban population studies; (4) articles that provided cardiovascular outcomes for ambient air pollution exposure; and (5) studies on human subjects. Editorials, opinion pieces, conference abstracts, and studies with a primary outcome of interest as non-cardiovascular health effects (e.g., respiratory or neurological effects only) were excluded.

The search strategy followed the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines where applicable, although the present study is not a formal systematic review (Page et al., 2021). Duplicates were removed, and titles and abstracts were screened independently by two reviewers. Full-text papers of possibly relevant studies were obtained and were reviewed in-depth to assess their applicability to the study objective.

Selected studies were reviewed systematically for the following major parameters:

• Type and concentration of air pollutants measured, such as PM2.5, PM10, nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), and ozone (O_3), by standard environmental monitoring techniques.

• Study population and setting population characteristics, with urban-rural comparisons where applicable, and subgroup analyses by age, sex, socioeconomic status, and pre-existing medical conditions (Brook et al., 2010; Kaufman et al., 2016).

• Cardiovascular impacts, both acute (myocardial infarction, arrhythmias, hypertensive crisis, etc.) and chronic (heart failure, atherosclerosis, hypertension, etc.), determined by validated clinical criteria and hospital or public health databases (Rajagopalan et al., 2018).

• Cardiovascular disease-relevant clinical biomarkers, such as high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), B-type natriuretic peptide (BNP), cardiac troponins, and blood pressure or heart rate variability alterations (Pope et al., 2016).

• Mechanistic support from cohort studies, controlled exposure studies, and population-based investigations of inflammation, endothelial dysfunction, oxidative stress, and autonomic imbalance as mediating pathways of pollution-attributable cardiovascular damage (Newby et al., 2015; Münzel et al., 2017).

RESULTS

67 related studies were considered relevant to the research question with observational cohorts, cross-sectional data, and controlled exposure trials conducted

in diverse urban environments. A common and necessary finding in much of the literature is that the urban air pollution—particularly low- and middle-income country locations—is being observed at levels way beyond World Health Organization's established standards. Year-round average concentrations of fine particulate matter (PM2.5) often varied between two and ten times the safe level of 5 μg/m³, particularly in megacities such as Delhi, Beijing, and Los Angeles (World Health Organization, 2021; Brauer et al., 2016). These elevated concentrations are mainly attributed to motor vehicle emissions, industrial processes, and fossil fuel burning for energy, compounded in densely populated and rapidly urbanizing regions.

A number of large-scale epidemiological studies have established a strong association between urban air pollution exposure and increased rates of cardiovascular events. For instance, findings from the Framingham Offspring Study demonstrated that short-term exposure to high concentrations of PM2.5 considerably increased the risk of myocardial infarction, with effects being observed within hours to days following exposure (Wilker et al., 2013). Similarly, Multi-Ethnic Study of Atherosclerosis (MESA Air) also revealed that long-term exposure to nitrogen dioxide (NO_2) and traffic-related air pollution was associated with the fast progression of coronary artery calcification, indicating long-term vascular burden (Kaufman et al., 2016).

Hypertension has also emerged as a critical outcome linked to urban air pollution. Several studies observed that chronic exposure to NO_2 and PM2.5 is associated with both increased systolic and diastolic blood pressure, suggesting vascular dysfunction and increased peripheral resistance as underlying mechanisms (Fuks et al., 2014; Yang et al., 2018). These associations were more pronounced in older adults and individuals with pre-existing cardiometabolic risk factors.

Air pollution exposure also appears to exacerbate heart failure and contribute to the onset of cardiac arrhythmias. Emergency department visits and hospitalizations for heart failure exacerbations have been positively correlated with elevated levels of ozone (O_3) and particulate matter, particularly during heat waves or pollution spikes (Shah et al., 2013; Bell et al., 2014). Arrhythmogenic effects, especially among the elderly, have been linked to short-term fluctuations in PM2.5 and ultrafine particles, likely mediated through autonomic imbalance and increased myocardial irritability.

At the molecular and clinical biomarker level, studies consistently reported elevated concentrations of high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), fibrinogen, and cardiac troponins among individuals exposed to high levels of ambient air pollution. These biomarkers reflect a systemic inflammatory state and endothelial activation, which are pivotal in the development and destabilization of atherosclerotic plaques (Brook et al., 2010; Pope et al., 2016). Mechanistic insights from controlled human exposure studies and animal models further support the notion that air pollutants trigger oxidative stress, impair endothelial nitric oxide signaling, and induce autonomic dysregulation, ultimately increasing cardiovascular vulnerability (Münzel et al., 2017; Newby et al., 2015).

Taken together, the evidence supports a strong and biologically plausible link between urban air pollution and adverse cardiovascular outcomes. The relationship spans both acute and chronic exposures, involves multiple pollutant types, and affects diverse urban populations worldwide.

DISCUSSION

This review underscores the mounting evidence of a significant and growing association between urban air pollution and adverse cardiovascular outcomes. Multiple studies from diverse geographical regions have consistently shown that exposure to pollutants such as particulate matter (PM2.5) and nitrogen dioxide (NO_2) not only exacerbates pre-existing cardiovascular conditions, but also contributes to the onset and progression of cardiovascular disease. These findings are supported by extensive epidemiological data and experimental studies that indicate how chronic exposure to these pollutants may influence cardiovascular health on both acute and long-term scales. In particular, fine particulate matter (PM2.5), which penetrates deeply into the respiratory system and enters the bloodstream, has been implicated in increasing the risk of conditions like myocardial infarction, stroke, and heart failure (Pope et al., 2016; Wilker et al., 2013). Nitrogen dioxide (NO_2) exposure, often associated with traffic-related pollution, has been found to contribute significantly to the development of hypertension and atherosclerosis, both of which are critical risk factors for cardiovascular diseases (Brook et al., 2010; Yang et al., 2018).

The pathophysiological mechanisms behind these associations are complex and involve several interconnected biological pathways. Endothelial dysfunction, a hallmark of cardiovascular diseases, is a key mechanism by which air pollutants influence vascular health. Systemic inflammation, which is triggered by exposure to airborne pollutants, has been shown to initiate and exacerbate atherosclerotic processes, leading to plaque formation and instability,

which can result in acute events like heart attacks and strokes (Brook et al., 2010; Münzel et al., 2017). Moreover, the dysregulation of autonomic functions and oxidative stress further compounds the cardiovascular risks associated with air pollution (Newby et al., 2015). The evidence also points to the fact that these harmful effects are not confined to individuals with existing cardiovascular disease; even healthy individuals exposed to high levels of pollution show signs of vascular and systemic dysfunction.

Urban populations are particularly vulnerable to the harmful effects of air pollution, especially in low- and middle-income countries where pollution control measures are often inadequate and access to healthcare services may be limited. In these regions, rapid urbanization, coupled with industrialization and vehicle emissions, has resulted in alarming levels of air pollutants, exacerbating public health concerns. Furthermore, socioeconomic disparities often mean that marginalized communities bear a disproportionate burden of exposure, with limited resources for mitigation or treatment of the resulting health conditions (Brauer et al., 2016; WHO, 2021).

As clinicians, it is critical to recognize environmental exposure as a modifiable cardiovascular risk factor. The medical community should incorporate environmental history into routine cardiovascular assessments, especially for patients with pre-existing cardiovascular conditions. Providing guidance to patients regarding the avoidance of outdoor activities during periods of high pollution is important, particularly for vulnerable populations such as the elderly and those with pre-existing cardiovascular disease. Additionally, patients living in highly polluted urban areas may benefit from the use of air purifiers and protective masks, which can help reduce indoor pollution exposure (Pope et al., 2016; Bell et al., 2014). These interventions are simple yet effective strategies to help mitigate the cardiovascular risks posed by environmental pollutants.

On a broader scale, urban planning and public health policies must prioritize air quality as a critical component of cardiovascular health. Transitioning to cleaner energy sources and electric transportation options is essential in reducing the emissions that contribute to urban air pollution. Expanding green spaces and urban vegetation can also help improve air quality by naturally filtering pollutants. In addition, the implementation of real-time air monitoring systems, along with public alert systems that inform residents of pollution levels, can enable individuals to take protective actions during periods of high pollution exposure (Shah et al., 2013; WHO, 2021). Moreover, stronger regulatory measures to limit emissions from industrial and vehicular sources are crucial to reducing the overall burden of air pollution and its associated health risks. Public health campaigns focusing on the cardiovascular risks of air pollution can also help raise awareness and encourage behavior changes at the individual and community levels.

Ultimately, addressing the intersection of urbanization, air pollution, and cardiovascular disease requires a multi-faceted approach that includes clinical awareness, public health initiatives, and policy interventions. The evidence reviewed in this article reinforces the importance of integrating air quality concerns into cardiovascular risk assessments, healthcare practice, and urban policy, as these strategies will be essential in mitigating the harmful effects of pollution on cardiovascular health.

CONCLUSION

Urbanization is inextricably linked to air pollution, which poses a significant and growing threat to cardiovascular health globally. As cities continue to expand and populations increase, the environmental challenges associated with urban living, particularly poor air quality, intensify. The effects of air pollution, especially particulate matter (PM2.5) and nitrogen dioxide (NO2), are well-documented in their capacity to contribute to various cardiovascular conditions, including hypertension, coronary artery disease, heart attacks, strokes, and even heart failure.

Clinical evidence strongly underscores the need for a concerted healthcare response to address the widespread impact of urban air pollution on heart health. Studies show that individuals living in polluted urban environments are at a higher risk of developing cardiovascular diseases, and this risk is exacerbated for vulnerable populations such as children, the elderly, and those with pre-existing heart conditions. Moreover, the synergistic effect of air pollution and other lifestyle factors, such as physical inactivity and poor diet, compounds the risk of cardiovascular diseases in urban settings.

To mitigate these threats, it is imperative that both healthcare professionals and policymakers work together in a multifaceted approach. Healthcare providers must be proactive in screening for cardiovascular risk factors in individuals living in high-pollution areas, adopting preventive measures, and educating the public about the dangers of air pollution on heart health. On the policy front, there is a critical need for more stringent air quality regulations and urban planning strategies that promote green spaces, reduce traffic emissions, and prioritize sustainable public transport. Additionally, promoting the use of cleaner technologies and renewable energy sources will

play a pivotal role in reducing the long-term exposure to harmful air pollutants.

Furthermore, the call for further longitudinal studies is essential to better understand the long-term effects of urban air pollution on cardiovascular health. Such research will provide deeper insights into the mechanisms by which air pollution influences heart disease and will guide future clinical practices and public health initiatives. Integrative strategies, including the collaboration of medical professionals, environmental scientists, urban planners, and policymakers, are crucial for developing comprehensive solutions that address the dual challenges of urbanization and air pollution.

In conclusion, as urbanization continues to shape the global landscape, the cardiovascular burden posed by air pollution requires immediate attention. By adopting a holistic approach that encompasses healthcare interventions, effective policy measures, and sustained research, we can significantly mitigate the harmful effects of air pollution on heart health and work towards creating healthier urban environments for future generations.

REFERENCES

Bell, M. L., Ebisu, K., Leaderer, B. P., Gent, J. F., Lee, H. J., Koutrakis, P., & Peng, R. D. (2014). Associations of PM2.5 constituents and sources with hospital admissions: analysis of four counties in Connecticut and Massachusetts (USA) for persons ≥65 years of age. Environmental Health Perspectives, 122(2), 138–144.

Brauer, M., Freedman, G., Frostad, J., et al. (2016). Ambient air pollution exposure estimation for the Global Burden of Disease 2013. Environmental Science & Technology, 50(1), 79–88.

Brook, R. D., Rajagopalan, S., Pope, C. A. III, et al. (2010). Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. Circulation, 121(21), 2331–2378.

Fuks, K. B., Weinmayr, G., Basagaña, X., et al. (2014). Long-term exposure to ambient air pollution and traffic noise and incident hypertension in seven cohorts of the European study of cohorts for air pollution effects (ESCAPE). European Heart Journal, 38(13), 983–990.

Kaufman, J. D., Adar, S. D., Allen, R. W., et al. (2016). Association between air pollution and coronary artery calcification within six metropolitan areas in the USA (the MESA Air study): A longitudinal cohort study. The Lancet, 388(10045), 696–704.

Münzel, T., Hahad, O., Kuntic, M., et al. (2017). Effects of air pollution on the cardiovascular system. European Heart Journal, 38(8), 555–561. https://doi.org/10.1093/eurheartj/ehw139

Newby, D. E., Mannucci, P. M., Tell, G. S., et al. (2015). Expert position paper on air pollution and cardiovascular disease. European Heart Journal, 36(2), 83–93b.

Page, M.J., McKenzie, J.E., Bossuyt, P.M., et al. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. BMJ, 372, n71. https://doi.org/10.1136/bmj.n71

Pope, C. A. III, Turner, M. C., Burnett, R. T., et al. (2016). Relationships between fine particulate air pollution, cardiometabolic disorders, and cardiovascular mortality. Circulation Research, 119(11), 1204–1215.

Rajagopalan, S., Al-Kindi, S.G., & Brook, R.D. (2018). Air pollution and cardiovascular disease: JACC state-of-the-art review. Journal of the American College of Cardiology, 72(17), 2054–2070. https://doi.org/10.1016/j.jacc.2018.07.099

Shah, A. S. V., Lee, K. K., McAllister, D. A., et al. (2013). Short term exposure to air pollution and stroke: systematic review and meta-analysis. BMJ, 346, f1295.

Wilker, E. H., Mittleman, M. A., Coull, B. A., et al. (2013). Long-term exposure to ambient air pollution and subclinical measures of cardiovascular disease: The Framingham Heart Study. Environmental Health Perspectives, 121(5), 639–645.

World Health Organization (2021). WHO global air quality guidelines: Particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide.