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THE ROLE OF AMBIENT PARTICULATE MATTER IN CARDIOVASCULAR DISEASE PATHOGENESIS

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ABSTRACT

Background: Ambient particulate matter (PM) is a well-established risk factor for cardiovascular disease (CVD), yet regional differences in exposure, susceptibility, and health outcomes remain underexplored. Central and Eastern Europe, including Poland, experience some of the highest PM concentrations in the European Union, but are underrepresented in global analyses.

Objective: This narrative review aims to synthesize current evidence on the pathophysiological mechanisms linking PM exposure to CVD, while also highlighting epidemiological trends and risk differentials specific to Poland in comparison to Western European countries.

Methods: A comprehensive literature search was performed in PubMed and Google Scholar using keywords such as "air pollution","particulate matter", "cardiovascular disease", "Poland" and "Europe". Studies published between 2016 and 2024 were considered. Epidemiological data from Polish and European public health sources were also analyzed.

Results: PM exposure (notably PM2.5 and PM10) is strongly associated with hypertension, atherosclerosis, stroke, heart failure, acute coronary syndromes, and arrhythmias. Recent findings underscore heightened vulnerability among older adults and urban populations in Poland. Compared to Western Europe, Poland shows persistently higher average annual PM2.5 levels and related CVD mortality, suggesting disparities in environmental regulation and preventive strategies.

Conclusions: This review contributes region-specific insight into the cardiovascular impact of air pollution and underlines the need for targeted public health interventions in Poland. Strengthening air quality policies and integrating environmental risk into cardiovascular prevention guidelines are essential steps toward reducing the regional health burden.

Keywords: air pollution, particulate matter (PM2.5, PM10), cardiovascular disease, stroke, heart failure

INTRODUCTION

Air pollution is a global health and environmental concern, encompassing gaseous, liquid, and solid substances—including particulate matter (PM)—that pose substantial risks to human health. These pollutants originate from both natural processes, such as wildfires and volcanic eruptions, and anthropogenic sources, including industrial emissions, vehicular traffic, and residential heating. The most harmful components include nitrogen oxides (NOx), sulfur dioxide (SO_2), carbon monoxide (CO), and volatile organic compounds (VOCs), as well as suspended solid particles categorized by size: PM10, PM2.5, PM1.0, and PM0.1. The smaller the particle, the deeper it penetrates the respiratory tract; ultrafine particles (PM0.1) can cross the alveolar barrier and enter the bloodstream, exerting systemic effects.

Air pollution is responsible for more than 300,000 premature deaths annually in Europe, primarily due to respiratory and cardiovascular diseases. Despite efforts by the European Union to implement stricter environmental regulations since the 1980s, many countries—including Poland—continue to record particulate matter concentrations that exceed WHO recommendations. The 2019 Global Burden of Disease (GBD) study identified air pollution as the fourth leading environmental risk factor for morbidity and mortality globally.

Cardiovascular diseases (CVDs) remain the leading cause of death worldwide. A growing body of epidemiological and experimental evidence indicates a strong association between PM exposure and CVD pathogenesis, including hypertension, atherosclerosis, stroke, heart failure, and arrhythmias. However, much of the existing literature focuses on Western Europe, North America, and China, whereas Central and Eastern European countries—such as Poland—are underrepresented, despite experiencing disproportionately high levels of air pollution. Poland is among the EU countries with high air pollution levels, especially during the winter season (due to coal heating). Poland has the highest annual mean concentrations of PM2.5 and PM10. Regions such as Kraków, Katowice, and Nowy Sącz frequently exceed both EU and WHO air quality limits, particularly during the heating season. These elevated pollution levels coincide with high rates of cardiovascular morbidity and mortality, suggesting a possible compounding effect that has yet to be fully explored.

AIMS

Therefore, this review aims to synthesize recent evidence on the pathophysiological mechanisms linking ambient particulate matter with cardiovascular disease, while contextualizing the findings within the Polish epidemiological and regulatory background.

MATERIALS AND METHODS

The authors conducted a comprehensive review of recent scientific literature using databases from PubMed and Google Scholar. To identify reliable information, the following search terms were utilized: "air pollution," "cardiovascular disease," "particulate matter," "atherosclerosis," "ozone," "heart failure," and "congenital heart disease." Only sources published from 2016 onward were included in the review.

RESULTS

An analysis of the reviewed studies indicates that various air pollutants are differentially associated with specific cardiovascular conditions. Table 1 summarizes the key pollutants—PM2.5, PM10, NO_2, O_3, and CO—along with their most consistently reported cardiovascular outcomes and underlying pathophysiological mechanisms. Notably, PM2.5 has the broadest range of associations, affecting hypertension, ischemic heart disease, arrhythmias, and stroke. Gaseous pollutants such as NO_2 and ozone also demonstrate distinct mechanistic pathways, including inflammation and oxidant-antioxidant imbalance, particularly in vulnerable populations. These findings reinforce the multifactorial nature of air pollution–related cardiovascular morbidity and underscore the need for pollutant-specific mitigation strategies.

Air Pollutant	Associated Cardiovascular Diseases	Mechanism of Action	Selected References
PM2.5	Hypertension, Ischemic heart disease, Atrial fibrillation, Stroke	Inflammation, oxidative stress, endothelial dysfunction	[3], [6], [27], [28]
PM10	Atherosclerosis, Acute coronary syndromes, Stroke	Lipid imbalance, vascular dysfunction	[9], [16], [25]

NO_2	Acute coronary syndromes, Atrial fibrillation, Stroke	Vasoconstriction, inflammation	[21], [24], [28]
O_3	Stroke (notably in men <60 y.o.)	Oxidant-antioxidant imbalance	[14], [15]
CO	Atherosclerosis	Tissue hypoxia, ischemic damage	[9], [29]

HYPERTENSION

Hypertension is diagnosed when a sustained systolic blood pressure of at least 140 mmHg and/or a diastolic blood pressure of at least 90 mmHg is observed. The pathogenesis of this disorder is attributed to reduced vascular compliance and increased arterial wall stiffness, which result from endothelial cell damage and impaired repair mechanisms. Studies indicate that exposure to airborne particulate matter can significantly increase the risk of developing hypertension [3,4]. It has also been demonstrated that the health effects related to air pollution exposure depend on gender, age, and geographical location [5]. Moreover, a meta-analysis of 20 studies revealed a correlation between long-term exposure to PM2.5 and the development of hypertension [6]. In addition, research has shown that particulate matter such as PM2.5, PM10, and ozone negatively impacts arterial elasticity, while the adverse effects of air pollution are less pronounced among individuals residing in green areas [7].

Adherence to a healthy, balanced diet may serve as an important protective factor against the development of hypertension resulting from exposure to particulate matter of varying sizes. The consumption of foods such as fish, whole-grain products, eggs, fruits, mushrooms, and root vegetables has been shown to exert a protective effect against air pollution-induced hypertension. Similar protective effects were observed for vitamins B2, B3, and E, with the beneficial impact of vitamin E being more pronounced than that of the B vitamins. Adopting a vitamin-rich, healthy diet produces antioxidant and anti-inflammatory effects, which may mitigate the deleterious impact of particulate matter on the organism, thereby reducing the risk of developing air pollution-related hypertension [3].

Several studies indicate that specific dietary components may mitigate the cardiovascular effects of air pollution. As summarized in Table 2, foods rich in antioxidants and anti-inflammatory compounds—such as fish, eggs, mushrooms, and whole grains—are associated with reduced risk of air pollution–related hypertension and lipid disorders. Among micronutrients, vitamin E has shown a particularly strong protective effect through its capacity to neutralize reactive oxygen species, while B-group vitamins support endothelial function and vascular integrity. These findings suggest that dietary modification may serve as a practical and low-cost adjunct strategy in reducing pollution-related cardiovascular risk, particularly in high-exposure populations.

Component	Protective Effect	Mechanism of Action
Fish, eggs, mushrooms	Reduced risk of hypertension	Anti-inflammatory and antioxidant action
Vitamin E	Strong antioxidant protection	Neutralization of free radicals
Vitamins B2, B3	Moderate vascular protection	Support of vascular function
Whole grain products	Improved lipid profile	Regulation of cholesterol levels

DYSLIPIDEMIA AND ATHEROSCLEROSIS

Dyslipidemia is a well-established risk factor for the development of atherosclerosis, which is closely associated with cardiovascular diseases [8]. Scientific studies have demonstrated that air pollution significantly contributes to alterations in blood lipid levels. It has been shown that long-term exposure to atmospheric aerosols (PM1, PM2.5, and PM10) as well as carbon monoxide results in elevated total cholesterol levels. In particular, exposure to PM10 is positively correlated with increased serum levels of triglycerides and LDL-C, whereas HDL-C—an important protective factor against cardiovascular diseases—is reduced with higher concentrations of PM1.0, PM10, carbon monoxide, and sulfur dioxide [9, 10].

Research has also identified specific groups at increased risk for developing dyslipidemia due to air pollution

exposure, including older individuals [10] and those consuming high-calorie, high-fat diets [11]. Moreover, alterations in blood lipid profiles have been observed in young, healthy subjects subjected to prolonged air pollution exposure. For example, in a study involving young Korean men, increased levels of PM2.5 were associated with a reduction in HDL-C, while elevated NO_2 concentrations correlated with increased total cholesterol levels [12].

STROKE

Depending on its etiology, stroke can be classified as either ischemic stroke—associated with insufficient blood and oxygen delivery to the brain—or hemorrhagic stroke, which results from the extravasation of blood within the brain [13]. It is estimated that 14% of all stroke-related deaths are correlated with air pollution [14], a category that includes ozone, which disrupts the oxidant-antioxidant balance in the human body. Studies have demonstrated that even short-term exposure to ozone may be linked to acute stroke events, with this association being notably more pronounced among men, individuals under 60 years of age, and patients with hypertension [15].

Other studies report that short-term exposure to PM2.5 particulate matter at levels exceeding 10 µg/m³ is correlated with an increased incidence and mortality from ischemic stroke [16]. Associations have also been identified between short-term elevations in PM2.5 levels and hemorrhagic stroke. In a study conducted in Algarve, Portugal—a region characterized by low ambient pollution—an increase in PM2.5 of 10 µg/m³ was associated with a 5.7% higher probability of intracerebral hemorrhage [17]. Furthermore, another study involving 15 million participants found a correlation between elevated levels of nitrogen dioxide and atmospheric soot and an increased frequency of stroke events [18]. Finally, short-term exposure to PM10 has been shown to result in a higher rate of hospitalizations among stroke patients [19].

HEART FAILURE

In studies investigating the association between air pollution and cardiovascular diseases, the impact of atmospheric aerosols on hospitalization rates and mortality due to heart failure has been observed. Long-term exposure to PM2.5, PM2.5–10, and PM10 has been implicated in the development of heart failure, with similar long-term effects noted for NO_2 and NO_x gases. Individuals with a genetic predisposition to this condition appear to be more susceptible to the harmful effects of air pollution [20].

In another study, a positive association was reported between exposure to NO_x and road traffic-related air pollutants and the occurrence of heart failure [21]. Additionally, one investigation described the influence of both physical activity and exposure to PM2.5 on the risk of developing heart failure. Physical activity was identified as a protective factor, whereas exposure to PM2.5 was positively correlated with an increased incidence of heart failure events. The highest risk was observed among patients with low levels of physical activity who were exposed to elevated concentrations of PM2.5 [22].

Moreover, short-term exposure to PM2.5 particulate matter was associated with an increased frequency of hospital readmissions due to exacerbation of heart failure. This frequency was found to depend on the time elapsed since the initial diagnosis of the condition and the initiation of treatment [23].

ACUTE CORONARY SYNDROMES

Exposure to PM2.5 and PM10 in ambient air correlates with an increased risk of developing acute coronary syndromes. Short-term exposure to these particulate matters has been associated with higher rates of hospitalization for ST-elevation myocardial infarction (STEMI) and increased mortality. A similar effect has been observed with exposure to nitrogen dioxide (NO_2) [24].

The risk of developing an acute coronary syndrome resulting in death was found to increase with exposure to nitrogen oxides, especially among elderly individuals, males, current or former smokers, and patients with pre-existing atherosclerotic plaques in the carotid artery identified before the study commenced [21]. It has been demonstrated that particulate matter exerts a stronger influence on the development of acute coronary syndromes than gaseous pollutants [25].

Furthermore, it is recognized that elderly patients [26] and individuals with pre-diagnosed cardiovascular diseases are more susceptible to the effects of PM2.5 than healthy individuals. Additionally, studies indicate that patients with a history of stroke or myocardial infarction are at a heightened risk of experiencing acute coronary syndromes and cardiovascular mortality, even at PM2.5 levels below 12 µg/m³. This suggests that current PM2.5 standards may be insufficient to reduce the incidence and mortality of cardiovascular diseases among susceptible populations [27].

CARDIAC ARRHYTHMIAS

Epidemiological studies have examined the relationship between the incidence of cardiac arrhythmias and exposure to PM2.5 in ambient air. One four-year study conducted in South Korea, which involved 432,587

participants, confirmed this association and identified atrial fibrillation as the most common arrhythmia induced by PM2.5 exposure. An increased predisposition to develop arrhythmias due to air pollution was observed with rising PM2.5 levels compared to PM10. Moreover, the risk of atrial fibrillation was higher in individuals over 60 years of age, those with obesity, coexisting arterial hypertension, a history of myocardial infarction, and in males [28].

Franco Folino and colleagues evaluated a cohort of 281 patients with either an implantable cardioverter-defibrillator (ICD) or a cardiac resynchronization therapy device with defibrillator function (CRT-D). The aim of their study was to assess the impact of short-term exposure to polluted air on the occurrence of ventricular arrhythmias. The researchers analyzed ambient concentrations of particulate matter (PM10 and PM2.5) alongside gaseous pollutants, including carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and ozone (O_3). They demonstrated a significant correlation between PM2.5 exposure and the occurrence of ventricular arrhythmias. Furthermore, a link was established between particulate matter exposure and episodes of ventricular arrhythmias manifested as ventricular tachycardia or ventricular fibrillation in patients with a prior myocardial infarction, whereas no association was found between the aforementioned gaseous pollutants and the development of ventricular arrhythmias [29].

The pathogenesis of air pollution-induced cardiac arrhythmias involves a systemic inflammatory response, which leads to the release of proarrhythmic inflammatory cytokines. Other contributing mechanisms include prothrombotic pathways [30]. Particulate matter that enters the bloodstream exerts a direct effect on cardiomyocytes by rapidly inducing the production of reactive oxygen species (ROS), which in turn alter ion channel function. Moreover, long-term exposure to PM2.5 results in cardiac remodeling—structural changes in the walls of the cardiac chambers and atria—that leads to increased left ventricular end-systolic and end-diastolic volumes [31]. This remodeling promotes fibrosis and the formation of reentrant circuits [32]. Additionally, autonomic nervous system dysfunction, as evidenced by a reduction in heart rate variability (HRV), further contributes to the development of arrhythmias [33].

DISCUSSION

Environmental pollution has a significant impact on human health, particularly in the development of cardiovascular diseases. Polluted air is a global issue affecting all inhabitants of the Earth. According to a report by the World Health Organization (WHO), as much as 90% of the global population lives in areas where the permissible air quality standards have been exceeded. Breathing exposes individuals continuously to both particulate and gaseous pollutants—such as NO_2, SO_2, O_3, H_2S, and CO—which adversely affect the entire organism, including the cardiovascular system. These pollutants contribute to the prevalence of so-called "civilizational diseases," namely arterial hypertension and atherosclerosis, posing significant challenges for healthcare systems.

The number of deaths attributable to air pollution continues to rise. In 2021, the WHO implemented stricter guidelines concerning the allowable concentration of ambient PM2.5. Under the current standards, the maximum annual concentration of PM2.5 is 5 µg/m³, while the daily limit is set at 15 µg/m³. For comparison, the reference ranges for PM10 have also been revised, with an annual limit of 15 µg/m³ and a daily limit of 45 µg/m³ [34].

Furthermore, choosing to engage in outdoor physical activity in highly urbanized regions should be based on up-to-date air pollution forecasts, which are routinely made available online. It is essential to undertake actions aimed at reducing the emission of harmful substances, improving air quality, and implementing pro-environmental policies.

To strengthen the epidemiological relevance of particulate matter exposure, Table 3 compares average annual PM2.5 concentrations and age-standardized cardiovascular mortality rates across selected European countries. Poland stands out with some of the highest PM2.5 levels in the EU and a markedly elevated cardiovascular mortality rate, exceeding 450 deaths per 100,000 population. In contrast, countries such as Sweden and France report significantly lower pollution levels and better cardiovascular outcomes. This disparity highlights both the environmental burden in Central and Eastern Europe and the potential public health gains achievable through targeted emission reductions and air quality interventions in regions such as Poland.

Country	Annual Mean PM2.5 (µg/m³)	CVD Mortality per 100,000 (age-standardized)	WHO Guideline Exceeded?
Poland	~18	>450	Yes
Germany	~12	~300	Partially
France	~11	~250	Partially

Sweden	~6	~200	No
WHO Guidelines	≤5	—	—

CONCLUSIONS

This review confirms that ambient particulate matter, particularly PM2.5 and PM10, plays a significant role in the development and exacerbation of cardiovascular diseases, including hypertension, atherosclerosis, stroke, heart failure, acute coronary syndromes, and arrhythmias. These findings are consistent with existing literature from Western Europe, North America, and East Asia.

However, this study emphasizes a critical knowledge gap: the underrepresentation of Central and Eastern Europe in global air pollution and CVD data syntheses. Poland, in particular, demonstrates some of the highest PM concentrations in the European Union. Despite regulatory efforts, many regions routinely exceed WHO- recommended thresholds for air quality. This is paralleled by persistently high cardiovascular mortality rates, suggesting a potential link between environmental exposure and regional health disparities.

PRACTICAL IMPLICATIONS

Clinicians should consider air pollution exposure as a modifiable cardiovascular risk factor, especially for elderly patients and those with pre-existing conditions. Routine patient education on minimizing exposure—such as avoiding outdoor physical activity during high-pollution days and using indoor air filtration—should be incorporated into cardiovascular care.

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