



The Impact of Fine Particulate Matter on Cardiovascular Events: A Global Perspective

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Fine particles, particularly those with an aerodynamic diameter $\leq 2.5 \mu\text{m}$ (PM_{2.5}), have emerged as one of the dominant risk factors for cardiovascular disease (CVD), as demonstrated in numerous studies worldwide (1-5). Cardiovascular epidemiology indicates that air pollution is linked to approximately 6.7 million premature deaths annually, with most occurring in low- and middle-income countries that still rely heavily on fossil fuels and biomass and have limited access to green energy (4, 6, 7). In 2019 alone, outdoor air pollution was estimated to cause approximately 4.2 million premature deaths, and two-thirds of these deaths were due to cardiovascular causes, such as ischemic heart disease and stroke (6, 7).

Among the various pollutants, PM_{2.5} is most consistently associated with CVD (1-5, 8). Owing to its small size, PM_{2.5} can penetrate deeply into the distal airways, reach the alveoli, and partially enter the circulation, where it interacts directly with vascular and cardiac tissues (1-3, 5). Contemporary reviews in cardiology and internal medicine report that both long-term and short-term exposure to PM_{2.5} is associated with a higher incidence of ischemic heart disease, acute myocardial infarction (AMI), stroke, heart failure, arrhythmias, and cardiovascular mortality (1-5). A recent state-of-the-art review concluded that PM_{2.5} is now the most important environmental cardiovascular risk factor and that it often acts synergistically with hypertension, diabetes, dyslipidemia, and smoking in determining overall risk (3, 4).

Epidemiologic and clinical studies have quantified these associations. Large cohorts and meta-analyses

indicate that each $10 \mu\text{g}/\text{m}^3$ increase in PM_{2.5} is associated with approximately a 2% higher risk of myocardial infarction, even after adjustment for traditional risk factors and copollutants (3, 9). Time-series and case-crossover analyses show that short-term increases in PM_{2.5} and PM₁₀ (particles $\leq 10 \mu\text{m}$) over hours to days are followed by increased hospital admissions and deaths due to acute coronary syndromes, particularly among older adults and patients with preexisting heart disease (2, 4, 5, 8). These studies support the concept that chronic exposure to PM_{2.5} and PM₁₀ creates a vulnerable substrate, including accelerated atherosclerosis, arterial stiffness, and endothelial dysfunction, on which acute pollution peaks can trigger plaque instability and thrombosis, resulting in ST-elevation myocardial infarction or other acute coronary events.

Arrhythmias represent another major manifestation of air pollution-related cardiovascular harm. Particulate matter (PM_{2.5}, PM₁₀, and ultrafine particles) and gaseous pollutants are associated with a wide spectrum of cardiac rhythm disturbances, including atrial fibrillation, supraventricular tachycardia, ventricular arrhythmias, premature beats, increased implantable cardioverter-defibrillator discharges, and higher arrhythmia-related mortality (1, 3, 5). Across multiple epidemiologic designs, including analyses of implantable cardioverter-defibrillator data, Holter monitoring, emergency visits, and mortality registries, PM_{2.5} has emerged as the pollutant with the strongest and most consistent relationship with arrhythmias, both as a short-term trigger and as a contributor to

long-term cardiovascular mortality (3, 5). Mechanistic work summarized in this and related reviews indicates that PM_{2.5}-induced oxidative stress and systemic inflammation can alter cardiomyocyte ion-channel function and calcium handling (1-3, 5). Concurrent autonomic imbalance and prothrombotic changes further lower the threshold for both atrial and ventricular arrhythmias.

Heart failure is closely intertwined with these pathways. PM_{2.5} has been described as contributing to both the development and progression of heart failure through sustained systemic inflammation, endothelial dysfunction, accelerated coronary atherosclerosis, elevated blood pressure, and repeated episodes of ischemia and arrhythmia (1-5). Meta-analytic evidence indicates that short-term increases in PM_{2.5} and nitrogen dioxide are associated with higher rates of heart failure hospitalization and death, with particularly pronounced effects in older adults and in those with existing CVD (1, 3, 4). For many patients in rapidly urbanizing regions who already carry multiple risk factors, PM_{2.5} therefore acts as both a chronic accelerator of ventricular dysfunction and an acute trigger of decompensation.

These risks are not distributed equally across the globe. Data from the Global Burden of Disease project and subsequent analyses indicate that approximately 89% of deaths attributable to outdoor air pollution occur in low- and middle-income countries (4, 6, 7). The highest exposures and burdens are observed in regions where urban growth is rapid, coal and other fossil fuels dominate power generation, and investment in clean transport and renewable energy lags behind (1, 4, 6, 7). In many such cities, daily PM_{2.5} levels frequently exceed World Health Organization guideline values by severalfold for large parts of the year, leaving hundreds of millions of people chronically exposed to unhealthy air from childhood (1, 4, 6, 7). In these populations, high baseline exposure accelerates atherosclerotic cardiovascular disease and elevates background risk, while transient pollution surges act as final hits that precipitate acute coronary syndromes, arrhythmias, and episodes of heart failure.

From a mechanistic standpoint, the central role of fine particles is increasingly well understood. Inhaled PM_{2.5} and ultrafine particles deposit in the distal airways and alveoli, where they generate reactive oxygen species and activate resident immune cells, initiating local and systemic oxidative stress and inflammation (1-3, 5). Circulating cytokines, acute-phase

proteins, and oxidized lipids then act on the vascular endothelium, reducing nitric oxide bioavailability, promoting vasoconstriction, and increasing the expression of adhesion molecules (1-3, 5). Over time, these processes accelerate atherosclerosis, increase arterial stiffness, and raise blood pressure. In parallel, PM_{2.5} exposure promotes a prothrombotic state, with higher fibrinogen levels, altered plasminogen activator inhibitor 1 and tissue plasminogen activator, and increased platelet reactivity, all of which favor thrombus formation on vulnerable plaques (1-3, 5). Autonomic imbalance and direct myocardial oxidative injury complete the picture, linking PM_{2.5} exposure to both electrical instability, including arrhythmias, and mechanical dysfunction, including heart failure.

For clinicians, these data have clear implications. Reviews from cardiology societies and expert groups argue that air pollution, particularly PM_{2.5}, should be recognized as a major modifiable cardiovascular risk factor, similar to hypertension, dyslipidemia, and smoking. In practice, cardiologists and primary care physicians should consider air quality when assessing cardiovascular risk. They should also recognize that days with high PM_{2.5} levels may increase the short-term risk of acute coronary syndromes, arrhythmias, and worsening heart failure, and they should explain this risk to patients with established cardiovascular disease or multiple risk factors.

Individual-level measures, such as advising vulnerable patients to limit intense outdoor exercise and avoid traffic hotspots on days with severe pollution, can help reduce some of the excess risk, although they cannot fully offset the impact of sustained exposure. Ultimately, the largest cardiovascular gains will come from structural changes, including stricter emission standards for vehicles and industry, improved fuel quality, expansion of clean public transport, promotion of active and low-emission mobility, and accelerated investment in renewable energy.

Footnotes

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