Environmental Pollution and Cardiovascular Disease: Mechanisms, Risk Factors, and Policy Recommendations

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Abstract: Cardiovascular disease (CVD) results from a combination of genetic and environmental factors. Growing evidence shows that environmental pollution—such as air pollutants (PM2.5, NO2, O3), waterborne toxins (heavy metals, organic pollutants), and soil contaminants (phthalates, bisphenol A)—contributes significantly to CVD. These exposures can increase oxidative stress, activate immune cells, disrupt circadian rhythms, and trigger stress reactions. They also promote traditional cardio-vascular risk factors like obesity, diabetes, and hypertension. This review summarizes recent findings on the link between environmental contaminants and CVD. It proposes strategies for mitigation, including stricter emission standards, expanded monitoring, public health campaigns, and incorporating environmental exposure assessments into cardiovascular risk screening. These interventions are essential to reduce pollution-related cardiovascular morbidity and mortality.

1 INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of mortality worldwide, with its prevalence rising most sharply in low- and middle-income countries. From 1990 to 2019, the number of cardiovascular disease cases nearly doubled, the prevalence of cardiovascular disease increased from 271 million to 523 million worldwide, and the number of cardiovascular deaths increased from 12.1 million to 18.6 million, according to the Global Burden of Cardiovascular Disease report (Roth et al., 2020). Heart and vascular diseases, such as hypertension, coronary heart disease, cerebrovascular disease, peripheral vascular disease, heart failure, rheumatic heart disease, congenital heart disease, and cardiomyopathy, are collectively referred to as cardiovascular disease (CVD) by the World Health Organization (WHO). The escalating human and economic costs of CVD have made its prevention and management a critical public health priority (Liu et al., 2024).

Numerous risk factors are involved in the higher incidence and mortality of cardiovascular disease. These include individual habits like drinking and smoking, family dynamics like marital status, and environmental influences. The development of cardiovascular disorders, including hypertension, coronary heart disease, and stroke, has been demonstrated to be directly linked to environmental pollution, particularly contamination of the air, water, and soil. By altering vascular endothelial function and hastening atherosclerosis, for example, fine particulate matter ($PM_{2.5}$) in the atmosphere has been shown to trigger an inflammatory response, increase oxidative stress, and contribute to the development of cardiovascular disease. Furthermore, the cardiovascular system can be negatively impacted by toxic compounds found in soil and water pollution through alterations in hormone levels, immunological reactions, and other mechanisms. Pollutants can impact the cardiovascular system via a number of biological mechanisms, such as thrombosis, oxidative stress, inflammatory response, changed expression of genes encoding antioxidants, and extracellular vesicle release. The formation and progression of CVD are facilitated by the interactions and connections among these pathways (Thomas et al., 2025; Omar et al., 2024). As shown in Figure 1, these mechanisms elevate cardiovascular risk and contribute to adverse outcomes such as myocardial infarction, stroke, heart failure, and arrhythmias.

This review synthesizes recent experimental and epidemiological data on the links between environmental pollution and CVD, outlines key mechanistic insights across air, water, and soil contaminants, and proposes concise, evidence-based interventions to reduce exposure and cardiovascular risk.

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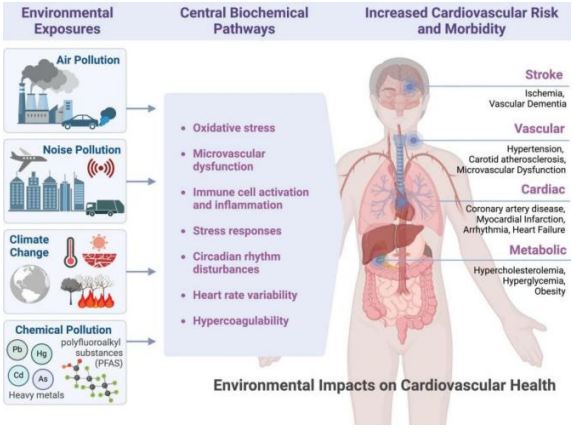

Figure 1. Environmental exposures and cardiovascular health (Jacob et al., 2024). Numerous harmful exposures have adverse effects on cardiovascular risk factors and outcomes via multiple overlapping pathways.

2 AIR POLLUTION

Based on their composition, outdoor air pollutants can be divided into two categories: particle pollutants and gaseous pollutants. Based on particle size, particulate matter (PM) can be categorized as fine particulate matter ($PM_{2.5}$), ultrafine particles (UFPs), inhalable particulate matter (PM_{10}), and others. Carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), ozone (O_3), and other gases are examples of gaseous pollution. Air pollutants can be produced and enter the human body from a variety of sources, including car exhaust, industrial emissions, forest fires, and other sources, due to the world's fast industrialization and urbanization (Yue et al., 2024). Hypertension is a significant risk factor for cardiovascular conditions such coronary heart disease, stroke, heart failure, atrial fibrillation, valvular heart disease, and aortic syndrome, according to large cohort studies. The risk of various conditions, including ischemic heart disease, cerebrovascular disorders, heart failure, and arrhythmias, may rise with either short-term or long-term exposure to $PM_{2.5}$. The damaging elements of PM, such as exposure to lead, cadmium, and arsenic, are particularly dangerous for the heart and vascular system and are linked to a higher risk of cardiovascular disorders. An elevated risk of stroke, atrial fibrillation, and atrial flutter is linked to prolonged exposure to NO_2. An elevated risk of cardiovascular death, mostly from ischemic heart disease, can result from prolonged exposure to O_3. Hospitalization for ischemic stroke can be considerably increased by short-term exposure to environmental SO_2, which is linked to cardiogenic embolism.

2.1 PM

Tiny solid or liquid particles that are suspended in the atmosphere are referred to as particulate matter (PM). Particles with a diameter of 10 micrometers or less are referred to as PM_{10}, while particles with a diameter of 2.5 micrometers or less are referred to as $PM_{2.5}$. $PM_{2.5}$ and UFPs can penetrate deeper into the respiratory tract and reach the alveoli because of their smaller size. This can impact gas exchange and perhaps have harmful effects on distant organs like the heart, brain, and placenta. According to research, exposure to $PM_{2.5}$ can raise oxidative stress levels and produce too many reactive oxygen species (ROS), which can cause cell damage and serious inflammatory reactions. These factors can then accelerate the development and progression of cardiovascular diseases (CVD). In particular, by increasing oxidative stress, $PM_{2.5}$ can activate cardiac ryanodine receptor 2 (Ryr2), resulting in intracellular calcium excess and cardiomyocyte death (Meng et al., 2022). The development of atherosclerotic plaques can also be accelerated by $PM_{2.5}$ exposure, which can also cause endoplasmic reticulum stress in macrophages, encourage Ca^{2+} accumulation in atherosclerotic plaques, raise ROS levels in the plaques, and increase apoptosis (He et al., 2020). Intercellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1) are endothelial function adhesion factors that are elevated in response to short-term exposure to $PM_{2.5}$, causing endothelial dysfunction. Heart disease or other cardiovascular problems may eventually arise from the buildup of arterial plaques brought on by prolonged exposure to $PM_{2.5}$, which can also cause vascular inflammation and atherosclerosis. According to epidemiological research, UFP exposure quickly lowers HRV, which has an impact on cardiac autonomic function (Zhang et al., 2022). Overall, oxidative stress, inflammatory reactions, endothelial dysfunction, and modulation of the autonomic nervous system are some of the ways that $PM_{2.5}$ and UFPs negatively impact the cardiovascular system. The development of cardiovascular disorders is facilitated by the interaction of these pathways.

2.2 Gaseous Pollutants

The cardiovascular system is largely harmed by gaseous pollutants through a variety of clinical and physiologic mechanisms. Atrial fibrillation, atrial

flutter, and stroke are among the cardiovascular events that are closely linked to prolonged exposure to nitrogen dioxide (NO_2). NO_2 can cause oxidative stress and systemic inflammation, which affects endothelial cell function and accelerates the development of atherosclerotic plaques. Arrhythmia risk may also be raised by the activation of inflammatory reactions, which may disrupt the regular activity of cardiomyocytes. Another prevalent gaseous pollutant, ozone (O_3), has a more intricate mechanism of harm. In addition to interfering with the metabolism of arachidonic acid, which leads to the production of thromboxane A2 and 20-hydroxy arachidonic acid, which activates platelets and causes oxidative damage, exposure to O_3 also increases the expression of coagulation biomarkers, including von Willebrand factor and clotting factors, to exacerbate blood coagulation tendencies and raise the risk of cardiovascular events (Taylor-Clark, 2020). Significantly, the association between exposure to air pollution and the emergence of cardiopulmonary disorders is mediated by extracellular vesicles (EVs). The pathophysiology of cardiovascular illnesses is significantly influenced by O_3 exposure, which modifies the release of EVs and the expression of the miRNAs they carry. Hemoglobin's ability to deliver oxygen is diminished by carbon monoxide's (CO) strong affinity binding to it, which causes tissue hypoxia and exacerbates cardiac hypoxia. In addition to hastening the development of atherosclerotic plaques, prolonged low-level CO exposure can cause acute cardiovascular events including myocardial infarction. Short-term exposure to sulfur dioxide (SO_2) induces oxidative stress and respiratory inflammation, which indirectly destabilize cardiovascular function and increase the risk of hospitalization for ischemic stroke and cardiogenic embolism.

3 WATER AND SOIL POLLUTION

The term "soil and water pollution" describes dangerous materials found in groundwater and water bodies that could have a negative impact on living things. Waste disposal, industrial emissions, and agricultural practices are the primary sources of contaminants. In addition to damaging aquatic life and lowering crop production, this pollution has a major negative impact on human health. The 2019 Global Burden of Disease Study estimates that water pollution from contaminated water sources killed about 1.23 million people. Pesticides, plastic debris, heavy metals, and persistent organic pollutants (POPs) are examples of common contaminants (Usman et al.,2024).

3.1 Plastics and Their Additives

Plastics are made of synthetic or organic polymers and are highly sought after worldwide due to their excellent qualities, which include flexibility, durability, and low production costs. Environmental pollution is caused by common plastic additives including phthalates, bisphenol A (BPA), and heavy metals. Phthalates may be linked to cardiovascular diseases (CVD), especially coronary artery disease (CAD), according to recent research. Increased carotid intima-media thickness (CIMT), atherosclerosis, increased risk of hypertension (HTN), metabolic syndrome (MetS), oxidative stress, and insulin resistance have all been linked to elevated phthalate levels. Polycarbonate plastics and epoxy resins are two common products made with bisphenol A (BPA). Numerous epidemiological studies have demonstrated a strong link between BPA exposure and an elevated risk of cardiovascular illnesses, primarily based on data from the National Health and Nutrition Examination Survey.

3.2 Pesticides and Persistent Organic Pollutants (POPs)

Because pesticides and persistent organic pollutants (POPs) are difficult for the environment to break down, they can linger and find their way into human bodies through soil and water. Furthermore, it has been discovered that pesticide exposure raises the incidence of certain cancers and has a negative impact on cardiovascular health. Increased oxidative stress, inflammation, and atherosclerosis could be the cause of these outcomes. Certain pesticides have the ability to directly harm cells' mitochondria, increasing reactive oxygen species (ROS), which in turn causes oxidative stress and inflammation, endothelial cell malfunction, and the acceleration of atherosclerosis. Meanwhile, there is a strong correlation between the incidence and mortality of cardiovascular illnesses, particularly with elevated risks of coronary artery disease (CAD), peripheral artery disease, and CIMT, and exposure to persistent organic pollutants, such as perfluoroalkyl compounds. The cardiovascular system is continuously threatened by prolonged exposure to pesticides and POPs, with possible pathogenic pathways including oxidative stress, inflammatory activation, and endocrine disruption.

3.3 Heavy Metal Pollution

It has been determined that exposure to heavy metal pollution increases the incidence of cardiovascular illnesses (Jacob et al., 2024). Although particulate air pollution also contains metals, ingestion is the main way that people are exposed to metals. Among the most extensively researched environmental hazardous metals linked to cardiovascular disease (CVD) are arsenic, cadmium, lead, copper, and mercury. Numerous studies show that exposure to metals, even at low levels, can raise the risk of cardiovascular illnesses and the mortality that goes along with them. The majority of metal contaminants cause cardiovascular illnesses by causing oxidative stress and systemic inflammation, which are prevalent pathophysiological routes. Atherosclerosis is facilitated by systemic inflammation, which in turn stimulates vascular inflammation, endothelial damage, and coagulation mechanism activation. Lipid peroxidation, an early indicator of atherosclerosis, has been discovered to be facilitated by a variety of hazardous metals. Usually, by competing with vital metals like calcium, iron, and zinc in the body, these harmful metals raise the risk of cardiovascular illnesses. For instance, lead's capacity to mimic the actions of vital elements like calcium and iron contributes to its cardiotoxicity. Because of their similar chemical structures, cadmium and lead can take the role of zinc in biological processes. Furthermore, by altering other risk factors, especially by raising the risk of obesity and hypertension (HTN), heavy metals may also make cardiovascular disorders worse (Usman et al.,2024; Jacob et al., 2024) (Figure 2).

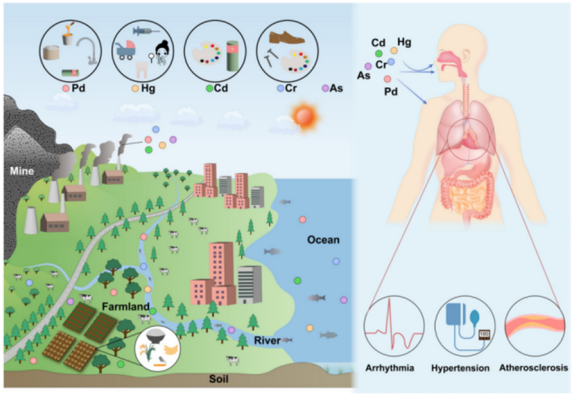

Figure 2. Distribution of heavy metals and their effects on the human cardiovascular system (Pan et al., 2024). The diagram depicts the distribution of arsenic (As), cadmium (Cd), lead (Pb), mercury (Hg), and chromium (Cr) in the environment (rivers, oceans, air, and soil) and the exposure source (food, drinking water, and industrial products). When exposure to heavy metals, they enter the body through inhalation, ingestion and dermal contact, eventually lead to cardiovascular abnormalities such as arrhythmia, hypertension and atherosclerosis.

4 POLICY RECOMMENDATIONS AND HEALTH INTERVENTION MEASURES

4.1 Strengthen Environmental Protection Policies

Stricter environmental protection laws should be developed and put into effect by the government to lessen the negative effects of pollution on cardiovascular health. Setting stronger emission limits, encouraging the use of clean energy, and bolstering the control of pollution in the air, water, and soil are some specific actions. For instance, lowering airborne concentrations of nitrogen oxides and fine particulate matter ($PM_{2.5}$) might lessen the direct damage they do to the cardiovascular system. At the same time, to guarantee the safety of drinking water and the health of the food supply chain, stringent management of soil contamination and water resources is required, preventing dangerous compounds from entering the human body through the food chain. The implementation of these measures requires coordination and cooperation among government departments, as well as active public participation, to achieve continuous improvement in environmental quality.

4.2 Promote Health Education and Behavioral Interventions

Preventing cardiovascular illnesses requires increasing public understanding of environmental protection and health literacy. The government should encourage the public to adopt healthy lifestyles that include balanced diets, regular exercise, quitting smoking, and consuming alcohol in moderation by using a variety of channels, including the media, community events, and online platforms, to spread information about the link between environmental pollution and cardiovascular diseases. To lessen the detrimental effects of environmental pollution, high-risk groups such as the elderly, children, and people with cardiovascular problems should receive personalized health management services. To improve the future generation's environmental awareness and health behavior abilities, educational

institutions should also include environmental health information in their curricula.

4.3 Enhance Monitoring and Data Collection

It is crucial to improve data collecting and environmental pollution indicator monitoring in order to more accurately evaluate the relationship between environmental pollution and cardiovascular illnesses. The government should assist the public in avoiding high-pollution areas, develop and enhance environmental monitoring networks, and rapidly disseminate information about pollution. Additionally, epidemiological research should be combined by health departments to continuously monitor and examine the relationship between environmental pollution and the incidence of cardiovascular disease, gathering pertinent data to support the development of policies. This data's sharing and public availability will also help research institutions carry out in-depth analyses to find successful intervention strategies.

4.4 Enhance Healthcare System Capacity

Hospitals and other community healthcare facilities should improve early cardiovascular disease screening, perform routine cardiovascular health examinations, promptly identify people who may have cardiovascular disease, and offer early therapies. In order to improve medical institutions' capacity to diagnose and cure cardiovascular diseases brought on by environmental pollution, the government should also invest more in equipment and technologies for cardiovascular disease diagnosis and treatment. To properly advise patients on their health, healthcare providers should undergo specific training to increase their knowledge of how environmental pollution affects cardiovascular health. In order to guarantee that more people, particularly those residing in highly polluted areas, have easy access to cardiovascular health management services, the government can simultaneously encourage changes to the health insurance system.

5 CONCLUSION

Numerous studies have demonstrated the strong correlation between environmental pollution (particularly air, water, and soil contamination) and the onset and progression of cardiovascular disorders. Through mechanisms like altering vascular endothelial function and speeding up atherosclerosis, fine particulate matter ($PM_{2.5}$) in the air can cause inflammatory reactions, raise oxidative stress, and encourage the development of cardiovascular illnesses. Furthermore, the cardiovascular system can be adversely affected by toxic compounds found in soil and water pollution through alterations in hormone levels, immunological reactions, and other routes. There are several biological ways in which these pollutants impact the cardiovascular system, but the main ones are oxidative stress, inflammation, thrombosis, alterations in the expression of genes that code for antioxidants, and the release of extracellular vesicles. These pathways are interrelated and interact with each other, promoting the onset and development of cardiovascular diseases.

The government should create and execute stronger environmental protection policies, such as raising emission standards, encouraging the use of clean energy, and bolstering the control of air, water, and soil pollution, in order to address the threat that environmental pollution poses to cardiovascular health. At the same time, it is crucial to promote healthy lifestyles, raise public knowledge of environmental protection and health literacy, and offer high-risk populations individualized health management services. Important steps to lessen the detrimental effects of environmental pollution on cardiovascular health include bolstering the monitoring of environmental pollution indicators, gathering pertinent data, and improving the capacity of the healthcare system to respond. In conclusion, it is important to consider how environmental contamination affects cardiovascular health. We can successfully reduce the detrimental effects of environmental pollution on cardiovascular health and raise the general level of cardiovascular health in the population by putting in place efficient policy measures, bolstering behavioral and health education initiatives, and improving the response capabilities of the healthcare system.

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