

Interaction Of Environmental And Dietary Factors In The Pathogenesis Of Cardiovascular Diseases

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Abstract: The article examines the relationship and interaction between environmental and dietary factors in the pathogenesis of cardiovascular diseases (CVD). Based on an analysis of current scientific sources, it emphasizes that environmental pollution (PM_{2.5}, NO₂, heavy metals, organic toxins) and unhealthy dietary patterns (excess trans fats, salt, and antioxidant deficiency) exert a synergistic effect on the development of inflammatory and degenerative changes in the cardiovascular system. Particular attention is given to the mechanisms of interaction between these factors, which include oxidative stress, endothelial dysfunction, and mitochondrial disturbances. The article substantiates the need for a comprehensive preventive approach based on improving diet quality and reducing exposure to environmental risks.

Keywords: Cardiovascular diseases, nutrition, environmental factors, oxidative stress, antioxidants, inflammation, synergism, prevention.

Introduction: Cardiovascular diseases (CVD) currently occupy a leading position among the primary causes of mortality and morbidity across the globe. According to data from the World Health Organization (WHO), over 17.9 million people succumb to heart and vascular-related conditions annually. This staggering figure represents approximately 32% of all deaths worldwide, underscoring the profound global health burden posed by these diseases. Historically perceived as ailments predominantly afflicting the elderly population, recent epidemiological trends reveal a concerning shift: cardiovascular diseases are increasingly being diagnosed in middle-aged individuals and, alarmingly, in younger age groups as well. This demographic transition in disease prevalence is closely linked to significant changes in modern lifestyles. Factors such as rapid urbanization, sedentary behavior, the adoption of Westernized dietary habits, growing exposure to environmental pollutants, and the escalating psychological stress associated with modern life all contribute to the increasing incidence of cardiovascular conditions. The fast-paced nature of contemporary living often results in limited physical activity, poor sleep patterns, high consumption of ultra-processed foods, and chronic exposure to stress—each of which

independently and synergistically heightens the risk of developing cardiovascular pathology.

METHODS

While classical risk factors such as genetic predisposition, arterial hypertension, hypercholesterolemia, smoking, obesity, diabetes mellitus, and physical inactivity remain highly relevant, recent scientific research has brought increased attention to the role of external and modifiable influences. In particular, two interconnected domains—environmental conditions and dietary habits—are emerging as critical contributors to cardiovascular risk. Traditionally, these factors have been studied in isolation within the scientific community. However, an expanding body of evidence highlights the necessity of examining them together due to their interactive and compounding effects on cardiovascular health. Environmental exposures encompass a wide range of physical, chemical, and biological stressors. These include air and noise pollution, heavy metal contamination, endocrine-disrupting chemicals, and climate-related factors such as extreme temperatures. Long-term exposure to air pollutants, especially fine particulate matter (PM_{2.5}), has been strongly associated with endothelial

dysfunction, increased arterial stiffness, systemic inflammation, and oxidative stress—hallmarks of atherosclerotic development. Noise pollution, often underestimated, can also lead to elevated blood pressure, disturbances in circadian rhythm, and increased sympathetic nervous system activity, thereby exacerbating cardiovascular strain. On the other hand, dietary patterns significantly influence the risk of CVD. Diets high in saturated fats, trans fats, added sugars, and sodium have been directly associated with the development of obesity, dyslipidemia, hypertension, and insulin resistance—all of which are key precursors to cardiovascular disease. Conversely, diets rich in fruits, vegetables, whole grains, lean proteins, and healthy fats, such as the Mediterranean or DASH diets, have shown substantial cardioprotective effects. However, food quality and nutritional value can also be indirectly compromised by environmental factors, such as contamination of crops and water sources with pesticides, heavy metals, and industrial chemicals, adding another layer of complexity to the diet-CVD relationship.

RESULTS

Importantly, environmental and dietary factors often do not act independently but rather interact in multifaceted and synergistic ways. For instance, populations residing in urban or industrialized regions may face higher levels of pollution and simultaneously have reduced access to fresh, nutritious foods, leading to a "double burden" of risk. This combined exposure can amplify pathological changes in the vascular endothelium, disrupt metabolic homeostasis, and impair myocardial function. Moreover, chronic inflammation and oxidative stress—common outcomes of both poor diet and environmental toxicity—serve as a shared biological pathway leading to accelerated cardiovascular aging and increased susceptibility to acute cardiac events such as myocardial infarction and stroke. Understanding the interplay between environmental and dietary influences is therefore essential for developing more effective prevention strategies. A comprehensive public health approach should include policy-level interventions to reduce pollution levels, ensure food safety, promote healthy eating habits, and encourage physical activity. Simultaneously, individual-level behavioral changes must be supported through education, access to healthcare, and community-based initiatives aimed at mitigating modifiable risk factors. Overall, cardiovascular diseases remain a pressing global health challenge, one that is no longer confined to older populations. The evolving landscape of risk—shaped by lifestyle transitions, environmental degradation, and dietary shifts—demands a holistic and integrative

approach. By recognizing and addressing the intricate connections between environmental and nutritional determinants of cardiovascular health, we can pave the way toward more effective prevention, earlier intervention, and improved outcomes for individuals across all age groups. Environmental pollution, particularly in the context of rapid industrial development and urban expansion, has become not only chronic but also increasingly widespread. Its impact is no longer confined to localized areas near industrial zones; rather, it has permeated every aspect of modern life, affecting populations across both developed and developing nations. Among the most hazardous pollutants are fine particulate matter (PM_{2.5} and PM₁₀), nitrogen dioxide (NO₂), ground-level ozone (O₃), sulfur dioxide (SO₂), and a range of heavy metals such as lead, cadmium, mercury, and arsenic. In addition, persistent organic pollutants (POPs), volatile organic compounds (VOCs), and various endocrine-disrupting chemicals contribute significantly to the toxic burden. These substances infiltrate the environment through industrial emissions, vehicle exhaust, agricultural runoff, and improper waste disposal, ultimately entering the air we breathe, the water we drink, and the food we consume. Prolonged exposure to such pollutants has been shown to induce systemic oxidative stress—a pathological condition in which the balance between free radicals and antioxidants in the body is disrupted. This oxidative burden leads to the activation of pro-inflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and C-reactive protein (CRP), which collectively contribute to chronic low-grade inflammation. Furthermore, these pollutants impair endothelial function by reducing nitric oxide (NO) bioavailability, altering vascular tone regulation, and increasing the permeability of blood vessels. The integrity of the endothelial lining is crucial for vascular homeostasis; when compromised, it initiates a cascade of deleterious events including leukocyte adhesion, smooth muscle cell proliferation, and lipid deposition—all of which are central mechanisms in the pathogenesis of atherosclerosis.

DISCUSSION

The clinical consequences of such environmental insults are profound. Endothelial dysfunction and chronic inflammation promote thrombogenesis by enhancing platelet aggregation and disrupting fibrinolytic balance. This, in turn, can lead to the formation of intravascular thrombi, impaired myocardial perfusion, and increased risk of acute coronary syndromes such as myocardial infarction and unstable angina. Over time, these processes contribute to the progression of ischemic heart disease,

cerebrovascular accidents (stroke), and peripheral artery disease. Landmark epidemiological studies by Pope and Dockery (2006), and later by Brook et al. (2010), have provided compelling evidence of a direct, dose-dependent relationship between air pollution levels and the incidence of cardiovascular events. Their findings underscore that even short-term increases in airborne pollutant concentrations can significantly elevate the risk of cardiac morbidity and mortality, especially in individuals with pre-existing cardiovascular conditions. In this context, modern dietary habits can either exacerbate or mitigate the adverse health effects of environmental pollutants. The typical Western diet—characterized by high intake of saturated fats, refined carbohydrates, processed meats, and sugary beverages—has been shown to potentiate oxidative stress and systemic inflammation. Such diets often lack essential micronutrients and bioactive compounds necessary for cellular defense mechanisms, rendering the body more vulnerable to the damaging effects of environmental toxins. Conversely, a balanced and nutrient-dense diet can serve as a powerful protective factor. Nutritional science increasingly supports the concept that diet acts not only as a source of energy but also as a modulator of gene expression, immune response, and oxidative stress. Diets rich in natural antioxidants and anti-inflammatory agents have demonstrated the ability to counteract the molecular damage caused by pollutants. Vitamins C and E, for example, are potent free radical scavengers that protect cellular membranes from lipid peroxidation. Omega-3 fatty acids, commonly found in fatty fish, flaxseed, and walnuts, have anti-inflammatory properties and are known to improve endothelial function and reduce triglyceride levels. Flavonoids and polyphenols, abundant in berries, green tea, dark chocolate, and red wine (in moderation), exert cardioprotective effects through their antioxidant, anti-inflammatory, and vasodilatory properties.

Additional micronutrients such as selenium, magnesium, and potassium play vital roles in cardiovascular health. Selenium, a component of glutathione peroxidase, aids in the detoxification of peroxides and other reactive oxygen species. Magnesium helps regulate vascular tone, supports myocardial electrical stability, and is inversely associated with the risk of hypertension and arrhythmias. Potassium contributes to the maintenance of normal blood pressure by counteracting the effects of sodium and aiding in fluid balance. Coenzyme Q10, a mitochondrial enzyme cofactor, is another compound of growing interest due to its role in cellular energy production and antioxidant

defense. It has been shown to improve endothelial function and may offer therapeutic benefits in patients with heart failure and hypertension. Substantial epidemiological data support the notion that adherence to a diet rich in whole, plant-based foods is associated with a markedly reduced risk of cardiovascular disease. Patterns such as the Mediterranean diet and the Dietary Approaches to Stop Hypertension (DASH) diet have consistently demonstrated reductions in cardiovascular events by as much as 30–40%. These benefits are attributed not only to the nutritional content but also to the synergistic interactions among various food components, which enhance their bioavailability and efficacy *in vivo*. Importantly, the protective effects of diet extend beyond individual nutrient supplementation. Whole-food-based dietary patterns emphasize the importance of food matrix, gut microbiota interactions, and long-term dietary behavior. Fermented foods, high-fiber fruits and vegetables, and prebiotic-rich grains can improve gut health, which in turn influences systemic inflammation and lipid metabolism—key factors in cardiovascular disease prevention.

In conclusion, the intersection between environmental pollution and modern dietary practices represents a critical area of concern in the field of cardiovascular medicine. While environmental exposures alone can initiate and exacerbate cardiovascular pathology, poor dietary habits can intensify their impact, creating a synergistic threat to public health. On the other hand, strategic dietary interventions offer a practical, accessible, and cost-effective means of mitigating these risks. Therefore, public health initiatives should prioritize both environmental policy reform and widespread nutrition education as complementary strategies in the fight against cardiovascular disease. Addressing these interconnected factors holistically is essential for reducing the global burden of heart and vascular diseases in the 21st century. On the other hand, diets rich in trans fats, saturated fats, added sugars, excessive sodium, and ultra-processed food ingredients have been shown to significantly disrupt lipid and carbohydrate metabolism, elevate systemic inflammation, and worsen endothelial dysfunction. These dietary components contribute to the development of insulin resistance, dyslipidemia, central obesity, and elevated blood pressure—core elements of metabolic syndrome, which is itself a major risk factor for cardiovascular diseases (CVD). Trans fats, in particular, are notorious for increasing low-density lipoprotein (LDL) cholesterol and reducing high-density lipoprotein (HDL) cholesterol, promoting the formation of atherosclerotic plaques and vascular stiffening.

Moreover, the food supply itself can serve as an additional source of ecotoxins, especially in regions with poor environmental regulation or contaminated agricultural systems. Crops and livestock may absorb toxic substances from polluted soil, water, or air, introducing harmful agents such as cadmium, mercury, arsenic, lead, and persistent organic pollutants (POPs) into the human food chain. Additionally, the improper storage of food products, especially grains and nuts, can lead to the proliferation of mold and the formation of mycotoxins, such as aflatoxins and ochratoxins, which are highly toxic and potentially carcinogenic. These substances have cumulative effects in the human body and exhibit a tendency to bioaccumulate in various tissues, including the myocardium and vascular walls, thereby compounding cardiovascular damage over time. The interaction between environmental pollutants and poor dietary quality is not merely additive but often synergistic, meaning that the combined effect is significantly greater than the sum of individual effects. This phenomenon can be understood through the lens of the synergism model, which posits that environmental toxicants initiate a cascade of molecular and cellular damage—particularly to vascular endothelium, cardiac muscle, and metabolic regulation—while an unhealthy diet fails to provide the necessary biological defenses for repair, detoxification, and recovery. In this setting, the organism becomes increasingly vulnerable to oxidative stress, mitochondrial dysfunction, and chronic inflammation. For instance, in the presence of air pollutants such as PM_{2.5}, nitric oxide bioavailability in endothelial cells is already diminished due to oxidative degradation. When compounded by a diet deficient in antioxidants like vitamins C and E, this condition worsens, allowing reactive oxygen species (ROS) to accumulate unchecked. Inadequate intake of essential nutrients such as magnesium, selenium, or omega-3 fatty acids further compromises myocardial energy metabolism and cellular resilience. The result is impaired regeneration of endothelial tissue, destabilized atherosclerotic plaques, and heightened susceptibility to cardiovascular events.

Moreover, toxic metals such as cadmium and mercury interfere with key enzymatic systems in the cardiovascular and nervous systems. Cadmium, for example, disrupts calcium signaling and induces apoptosis in vascular smooth muscle cells, contributing to arterial stiffness and hypertension. Mercury, on the other hand, can impair mitochondrial function and interfere with antioxidant enzymes such as glutathione peroxidase. In individuals who simultaneously consume diets high in sodium and low in potassium and antioxidants, the cardiovascular system is pushed

further into a state of pathological imbalance.

This compounded burden significantly increases the risk of developing a wide range of cardiovascular complications. Chronic exposure to this "dual stressor" scenario may contribute to the onset and worsening of conditions such as arrhythmias, left ventricular hypertrophy, myocardial fibrosis, heart failure, and even sudden cardiac death. Disrupted mitochondrial energy metabolism in cardiomyocytes, combined with persistent low-grade inflammation, can lead to impaired contractility and electrical conduction abnormalities, thereby fostering a more severe and treatment-resistant disease course. Modern biomedical research and epidemiological studies increasingly confirm these observations. Patients with pre-existing cardiovascular conditions who reside in environmentally disadvantaged areas—characterized by high levels of air, soil, or water pollution—and who simultaneously follow nutritionally inadequate diets tend to exhibit faster disease progression, more frequent and severe exacerbations, and a greater incidence of secondary complications such as thromboembolic events, arrhythmias, and chronic heart failure. These individuals often respond poorly to conventional treatment protocols, as their baseline systemic inflammation and oxidative stress reduce the efficacy of pharmacological interventions and delay recovery. For example, data from cohort studies such as the Multi-Ethnic Study of Atherosclerosis (MESA) and findings from global environmental health reports indicate that individuals in lower socioeconomic strata, who are more likely to be exposed to both environmental pollutants and poor-quality diets, experience a disproportionate burden of cardiovascular morbidity and mortality. This intersection of environmental injustice and nutritional vulnerability underscores the importance of addressing CVD not only from a clinical standpoint but also through public health and policy interventions. Furthermore, emerging research in the field of nutritional toxicology suggests that certain food components may influence the absorption, metabolism, and excretion of environmental toxicants. For instance, fiber-rich diets can bind some heavy metals in the gastrointestinal tract and reduce their systemic bioavailability, while high-fat diets may increase the intestinal absorption of lipophilic toxins. These interactions further illustrate the complexity of the diet-environment-health triad and highlight the potential for targeted dietary strategies to modulate toxic exposures.

In summary, the interaction between environmental and dietary factors plays a critical and often underappreciated role in the pathogenesis and progression of cardiovascular diseases. Environmental

pollutants initiate and perpetuate vascular and myocardial damage, while poor dietary patterns deprive the body of the molecular tools needed to combat these effects. The resulting synergistic burden accelerates cardiovascular deterioration and diminishes treatment effectiveness, particularly in socioeconomically vulnerable populations. Addressing this complex interplay requires a multidisciplinary approach that includes environmental regulation, food safety enforcement, nutritional education, and individualized medical care. Only through such integrated strategies can we hope to reduce the growing burden of cardiovascular disease in an increasingly polluted and nutritionally imbalanced world.

Preventive Recommendations

For effective prevention of cardiovascular diseases, it is essential to address both environmental and dietary risk factors simultaneously. This involves the following key strategies:

1. Developing a diet tailored to local conditions, with an emphasis on organic and minimally processed foods;
2. Increasing the intake of natural antioxidants through a diet rich in vegetables, fruits, berries, greens, fish, olive oil, and flaxseed oil;
3. Monitoring the quality of drinking water, air, and the surrounding environment, especially in industrial and densely populated areas;
4. Implementing cross-sectoral programs that include environmental education for the population, access to healthy food, monitoring of pollutants, and improvement of urban infrastructure.

Environmental and dietary factors are playing an increasingly significant role in the pathogenesis of cardiovascular diseases. Their effects are closely interconnected and are expressed through common mechanisms — such as inflammation, oxidative stress, and disruption of cellular metabolism.

Therefore, a modern strategy for the prevention and management of CVD should be based on an integrative approach, combining environmental medicine, nutrition science, and cardiology. Improving nutrition and reducing exposure to harmful environmental factors are not only critical tasks for the healthcare system, but also for society as a whole.

CONCLUSION

Cardiovascular diseases remain one of the foremost global health challenges, driven not only by traditional risk factors but increasingly by the complex interplay between environmental pollution and dietary habits. Industrial development and urbanization have led to

pervasive exposure to toxic pollutants, which induce oxidative stress, inflammation, and endothelial dysfunction—key mechanisms in cardiovascular pathology. Simultaneously, modern dietary patterns characterized by high intake of trans fats, saturated fats, sugars, and processed foods exacerbate these harmful effects by disrupting metabolism and limiting the body's capacity for repair and defense.

The synergistic relationship between environmental and dietary factors amplifies cardiovascular risk, accelerating disease progression and complicating treatment outcomes, particularly in vulnerable populations residing in polluted areas with limited access to nutritious food. This multifactorial burden highlights the urgent need for holistic public health strategies that integrate environmental regulation, food safety, nutritional education, and individualized healthcare interventions.

Addressing cardiovascular disease effectively in the 21st century demands recognition of the interdependent nature of these risk factors. By mitigating environmental exposures and promoting balanced, antioxidant-rich diets, it is possible to reduce systemic inflammation and oxidative damage, thereby improving vascular health and cardiac function. Ultimately, a coordinated approach targeting both environmental and lifestyle determinants holds the greatest promise for reducing cardiovascular morbidity and mortality worldwide.

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