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Epidemiology, Mechanisms and Prevention in the Etiology of Environmental Factor-Induced Cardiovascular Diseases

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Abstract

Cardiovascular diseases are a significant cause of mortality worldwide, and their prevalence can be amplified by a range of environmental factors. This review article critically evaluated the published information on the epidemiology and pathophysiological mechanisms of various environmental factors such as air indoor and outdoor air pollution, water pollution, climate change, and soil pollution. Preventative measures to mitigate these effects including public health responses are discussed with gaps in our knowledge for future studies.

Keywords: Air pollution; Climate change; Indoor air pollution; Outdoor air pollution; Soil pollution; Water pollution

Indoor Air Pollution

Background

Indoor air pollution has been categorized as a significant contributing factor to the development of cardiovascular disease (CVD). It is composed of particulate matter which is typically classified into three different sizes: ultrafine particles, fine particles (PM2.5), and coarse particles (PM10) [1] (Figure 1). Globally, the smoke from cooking indoors using biomass fuels such as wood and crop waste is the primary source of indoor air pollution. These indoor stoves produce high PM values and increased carbon monoxide (CO) exposure which is detrimental to an individual's health. Due to the customary nature of women tending to the needs of the household and young children being around their mothers, these population groups tend to be the most affected. In a study completed by the International Respiratory Society, it was found that developing countries in Asia produced elevated levels of indoor pollution. Over a span of 24 hours, the average PM10 was between 300 to 3,000 $\mu g/m^3$, which can reach up to 30,000 $\mu g/m^3$ during periods of cooking [2]. Furthermore, over the same span, the average CO level is between 2-50 ppm and can go as high as 500 ppm during cooking [2]. According to the United States Environmental Protection Agency, a healthy average PM10 and CO level over a 24-hour span is 150 $\mu g/m^3$ and 9 ppm, respectively [3], which is 200 times less than the PM10 and about six times less the CO levels you would find during a cooking period in a developing country.

Environmental tobacco smoke (ETS) is another major source of indoor air pollution as well. Worldwide, approximately 40% of children, 35% of women, and 32% of men are exposed to ETS [4]. ETS is composed of toxic levels of nicotine, ammonia, and carbon monoxide; all of which have been shown to have a correlation with cardiovascular mortality.

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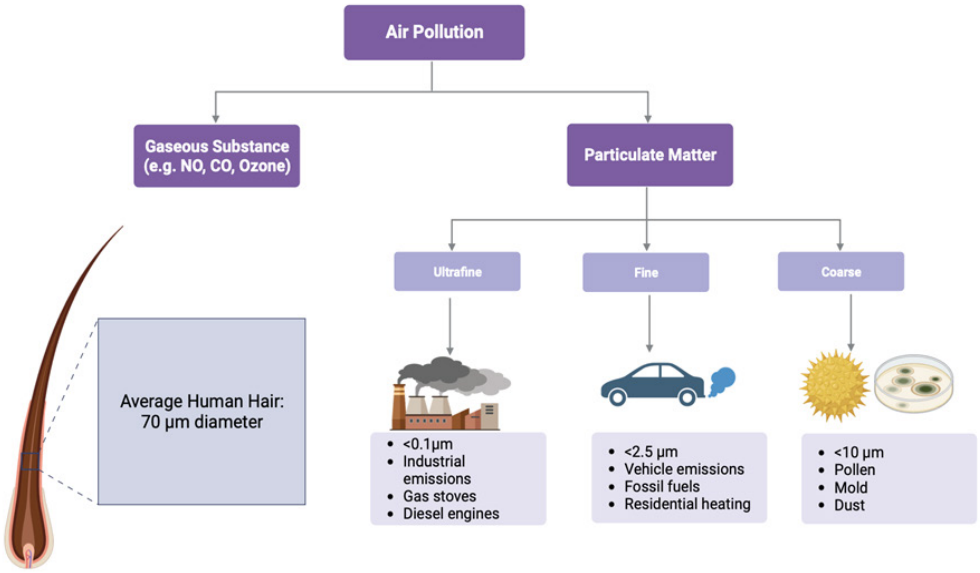

Figure 1: Air pollution consists of a combination of gaseous substances and particulate matter. Particulate matter can be categorized into three different sizes and from various sources.

Epidemiology

Cardiovascular Mortality

The particulate matter from household air pollution is generated from the inefficient use of solid fuels that are utilized for the operation of open fires and stoves in developing countries. According to the World Health Organization (WHO), approximately 2.4 billion people across the globe use these inefficient methods of cooking, putting them at risk for heart disease due to the elevated levels of indoor air pollution [5]. The household air pollution created from this accounts for 3.2 million deaths in 2020 and more than 237,000 deaths for children below the age of 5 [5]. Among the 3.2 million deaths, 32% are from ischemic heart disease and 23% are from stroke.

Furthermore, current evidence shows that non-smokers who live with smokers and are exposed to secondhand smoke are at greater risk for developing coronary heart disease compared to non-smokers who are not exposed to any smoke particulates. In a recent Center for Disease Control study, secondhand smoke increases the risk of developing coronary heart disease by about 25-30% and it is directly responsible for 35,000 deaths each year in the United States. Additionally, secondhand smoke increases the risk of stroke by 20-30% and causes about 8000 deaths annually [6].

Coronary Heart Disease

A 20-year prospective study published in 2004 identified the correlation between secondhand smoke and coronary heart disease (CHD) and the risk associated with it. The cardiac risk was between 1.45 (95% CI: 1.10 -2.08) and 1.57 (95% CI: 1.08 - 2.28), depending on the level of passive smoke exposure [7]. In a meta-analysis of 29 epidemiological studies that examined the risk of CHD in individuals exposed to passive smoke, a pooled risk of 1.31(95% CI: 1.21-1.41) was identified [8]. These risk values were just as large as those seen with light active smoking. Furthermore, in another of 18 epidemiological studies, it was found that non-smokers exposed to secondhand smoke had a CHD risk of 1.25 (95% CI: 1.17-1.32) as compared to non-smokers not exposed to smoke [9]. Thus, it was concluded that ETS is associated with an increase in CHD.

Stroke

In 2015, a study was conducted to analyze the relationship between environmental smoke exposure and the risk of the different subtypes of stroke among non-smokers. The risk for ischemic stroke was 1.29 (95% CI: 1.00-1.68). However, no significant correlation was found between environmental smoke exposure and hemorrhagic stroke [10]. Therefore, the overall risk of stroke was increased by 30% in individuals exposed to secondhand smoke, and according to the data, the only connection is with ischemic stroke.

Pathophysiology

Platelet Function

Platelets, or thrombocytes, are small cell fragments that circulate throughout the blood and play an integral role in clot formation. When vascular endothelium becomes impaired, platelets become activated and begin to coagulate by repairing the injured tissue. Elevated levels of platelets can damage the linings of coronary arteries which can develop into atherosclerosis, increase thrombus formation, and are correlated with an increased risk of ischemic heart disease [11]. In an experiment evaluating the connection between

platelet activity and passive smoke, non-smokers were placed in a room for 20 minutes where cigarettes had just been smoked [11]. By using platelet sensitivity as a marker for platelet aggregation, it was found that the non-smokers had increased platelet levels after being exposed to passive smoke.

Oxidative Stress

Oxidative stress is a physiological phenomenon that refers to the imbalance of the production and accumulation and reactive oxygen species (ROS) and the body's ability to detoxify these chemically reactive products [13]. This type of stress where there is an overwhelming amount of ROS, such as free oxygen radicals, can be detrimental to the human body by damaging cellular function and disrupting homeostasis [11]. Free oxygen radicals are typically produced during aerobic processes and under normal conditions, biological antioxidants protect the body's vasculature from oxidative stress damage. However, in addition to the free oxygen radicals that are produced in the body, passive smoke serves as another source of ROS that can cause oxidative stress and deplete the levels of antioxidants within the body [14]. When passive smoke enters the body, nitric oxide (NO) production in the endothelium is ceased. The ROS chemically react with NO and produce peroxide, increasing the number of stressors within the body. Furthermore, secondhand smoke decouples NO synthase, which ultimately creates superoxide instead of NO in the body. Additionally, passive smoke increases ROS production by activating NADPH oxidase [12].

Public Health Response

A smoking ban is a public health measure that is implemented to facilitate improvements in overall cardiovascular health. In a 2002 study, the city of Helena, Montana instituted a smoking ban on public and workspaces from June 5- December 3, 2002 [15]. It was reported that there was a significant decrease in the number of cardiovascular hospital admissions, from an average of 40 between June-December in the previous years compared to 24 during the time in which the ban was in place (16 fewer admissions; 95% CI: 0.3 -31.7) [15]. Moreover, another study presented at the American Heart Association Conference in 2005 analyzed the city-wide smoking ban in all public areas in Pueblo, Colorado. Implementing this policy helped reduce the myocardial infarction rate by 27% within a year and a half [16]. From these two cases, a smoking ban appears to be a viable option that can help improve cardiovascular health.

Outdoor Air Pollution

Background

Outdoor air pollution is a major public health concern and a significant environmental risk to cardiovascular health. In a recent study by the Global Burden of Disease in 2015, it was reported that ambient air pollution led to 4.2 million deaths (7.6% of the total global mortality) and 103.1 million disability-adjusted life-years (DALYs) (4.2% of global DALYs) [17]. Furthermore, WHO notes that in 2019, 99% of the global population did not reside in locations that met the WHO regulations for air quality (18). Due to this environmental health issue, WHO reports that 37% of ambient air pollution deaths were caused by ischemic heart disease and stroke. Thus, it is evident that air pollution plays an integral role in cardiovascular mortalities (Figure 2).

As mentioned previously, air pollution is composed of particulate matter (PM) and gaseous particles that are detrimental to cardiovascular health. PM is categorized into three different sizes: ultrafine particles, fine particles ($PM_{2.5}$), and coarse particles (PM_{10}). PM also has a variety of compositions depending on the original source. Carbonaceous particles originate from the combustion of fossil fuel sources and biomass. Some sources of combustion are residential heating, industrial power plants, and road traffic emissions. These carbon-based particles contain organic compounds such as polycyclic aromatic hydrocarbons and other reactive metals [19]. On the other hand, inorganic compounds are typically found on sedimentary rocks and mineral grains in agriculture. Moreover, Gaseous substances include nitrogen dioxide, ozone, carbon monoxide, sulfur dioxide, and other volatile organic compounds [20]. Nitrogen oxides lead to the development of toxic secondary particles by undergoing a series of photochemical reactions with the atmosphere and ammonia from agriculture [12]. One example of a secondary pollutant is ozone which is formed from the reaction between sunlight and nitrogen oxides.

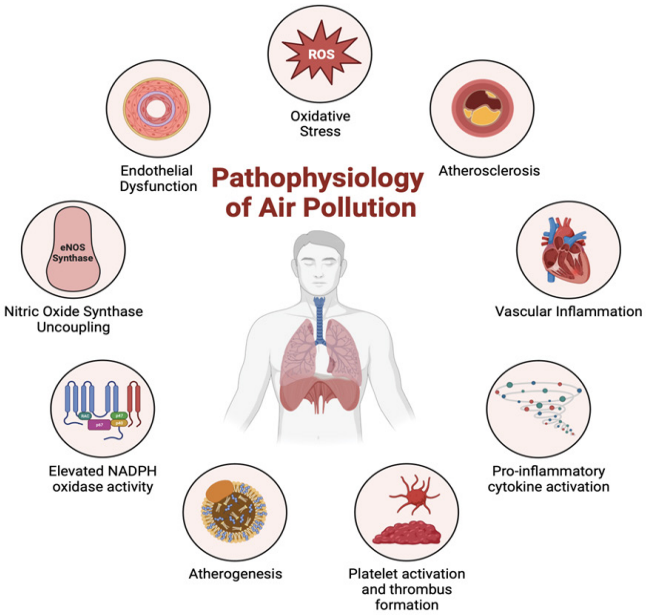

Figure 2: In addition to the detrimental impacts on cardiovascular health, air pollution can affect other organ systems, as shown in this figure.

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Epidemiology

Long-Term Cardiovascular Mortality

In a meta-analysis conducted in 2013, it was shown that there is an 11% (95% CI: 6–16%) significant increase in cardiovascular mortality for a 10 μg/m^3 increase in annual $PM_{2.5}$ atmospheric concentrations [21]. Even after considering socioeconomic status and cardiovascular factors, the strongest relationship was observed by ischemic heart disease mortality. Although a clear association can be visualized between $PM_{2.5}$ levels and cardiovascular-related deaths, it is important to note that some studies suggest that the adverse effects of $PM_{2.5}$ were higher in individuals with low levels of education [21]. Moreover, it was observed that only fine and ultrafine particles had a significant impact on cardiovascular mortality. Coarse particles did not have a similar effect.

Another meta-analysis of 19 studies was carried out to highlight the adverse effect of long-term exposure to nitrogen dioxide (NO_2) on cardiovascular mortality. It was reported that there is a 13% significant increase in cardiovascular mortality for a 10 μg/m^3 increase in annual NO_2 atmospheric concentrations [22]. For long-term ozone(O_3) exposure, a strong association was seen with cardiovascular-related deaths. The hazard ratio per 10 μg/m^3 O_3 increase was 1.22 (95% CI: 1.13–1.33) for overall CVD mortality [24]. Finally, the impact of long-term exposure to sulfur dioxide (SO_2) has not been well-established, but there is a positive association with respiratory mortality [23].

Short-Term Cardiovascular Mortality

In terms of immediate effects on cardiovascular mortality, associations have been developed as well. In a meta-analysis from 2014, it was documented that there is a 0.84% (95% CI: 0.41%-1.28%) significant increase in cardiovascular mortality for a 10 μg/m^3 increase in daily $PM_{2.5}$ concentrations [25]. However, this association varies depending on the location in the world. The association between short-term NO_2 exposure and cardiovascular-related deaths is also regionally dependent. In a study completed in 2021, it was observed that there is a 1.72% (95% CI: 1.41%-2.04%) significant increase in cardiovascular mortality for an 18.8 μg/m3 increase in daily NO_2 concentrations [26]. For short-term exposure to O_3, there is a 0.7% significant increase in cardiovascular mortality for a 10 μg/m3 increase in daily O_3 concentrations [27]. In a single pollutant model, it was noted that there is a 1.30% (95% CI: 0.27%-2.35%) significant increase in cardiovascular mortality for a 10 μg/m^3 increase in daily SO_2 concentrations [28].

Long-Term Coronary Artery Disease

Throughout the current literature, there are multiple studies that discuss the positive association between long-term exposure to air pollution and coronary artery disease (CAD). The Escape project was a 10-year European study that investigated the association between air pollutants and coronary events using a pool of over 100,000 participants [29]. From this study, there was a 13% (hazard ratio 1.13, 95% CI: 0.98-1.30) significant increase in the risk of myocardial infarction with an annual increase of 5 μg/m^3 in $PM_{2.5}$ concentrations. Furthermore, there was a 12% (hazard ratio 1.12, 95% CI: 1.01-1.25) significant increase in the risk of myocardial infarction with an annual increase of 10 μg/m^3 in PM_{10} concentrations [29]. In another study conducted in China, it was found that long-term exposure to toxic air pollutants, such as $PM_{2.5}$ and NO_2, is strongly associated with coronary artery calcium (CAC) [30]. Additionally, another Chinese study analyzed the relationship between long-term sulfur dioxide exposure and CHD. It was documented that there is a 2.5% significant increase in the risk of developing CHD for every 10 μg/m^3 in SO_2 concentrations annually [31].

Short-term Coronary Artery Disease

It is well known that short-term exposure to air pollutants can trigger a cascade of acute coronary artery events. A recent meta-analysis noted that air pollution and traffic emissions were the two strongest factors that can trigger myocardial infarction (MI) [32]. As it pertains to ST-elevated myocardial infarction (STEMI) specifically, a case-crossover study performed in 2013 explored the connection between air pollution and STEMI. This study indicated that for every 10 μg/m3 increase in $PM_{2.5}$ and NO_2, there was a 2.8% and 5.1% significant increase in the risk of STEMI for each respective pollutant [33]. Moreover, it appeared the risk related to NO_2 was greater among younger individuals, while the elderly population was more susceptible to PM exposure [33]. As for short-term ozone exposure, one study found that there was a 6.3% (95% CI: 1.2%-11.7%) significant increase in out-of-hospital coronary deaths for a 10 μg/m^3 increase in O_3 [34]. Higher risks were noted for females who had a past medical history of being hospitalized for coronary artery events.

Heart Failure

One meta-analysis explored the relationship between air pollutants and congestive heart failure (CHF). A strong, positive association was found between short-term increases in toxic gaseous substances (such as CO, SO_2, NO_2) and PM and increased risk of hospitalization or death from CHF [35]. The strongest association was seen on the day of pollutant exposure, with more persistent effects for $PM_{2.5}$ [35].

Pathophysiology

Oxidative Stress

As per multiple studies, it is well known that a sudden influx of toxic gaseous substances coupled with particulate matter causes a severe oxidative stress reaction. A study in 2013 explored the oxidative stress reaction enzymatic pathway by obtaining serum from individuals who have been exposed to diesel exhaust voluntarily. After incubating the endothelial

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cells with the serum from these participants, ROS, such as superoxide anion, production was visualized following a dose-response pattern [36]. Furthermore, the production of ROS was directly linked to the amount of $PM_{2.5}$ that was inhaled [36]. Other in vitro studies support the instrumental role of ROS generation. These reports state that the enzyme, superoxide dismutase, can reverse the detrimental effect caused by diesel exhaust exposure [37]. Moreover, oxidative stress reactions are directly related to the compounds that coat the surface of diesel particles such as transition metals, quinones, and polycyclic aromatic hydrocarbons [37].

Endothelial Dysfunction

The decrease in nitric oxide (NO) availability is another pathway of the endothelial oxidative stress reaction. NO is a key compound found within the human body that regulates vascular tone and blood flow. In a recent study, subjects were exposed to either diesel exhaust or filtered air for five hours. The results showed that exposure to diesel exhaust led to the uncoupling of nitric oxide synthase; thus, impairing endothelial function and vasoconstriction and increasing the amount of ROS [38,39].

Endothelial dysfunction is an early marker for atherosclerosis as well. In another study, patients with a previous history of myocardial infarction were exposed to diesel exhaust. It was observed that diesel exhaust exposure triggered myocardial ischemia within these patients, suggesting that air pollution has an adverse influence on the regulation of myocardial blood flow [40]. Moreover, it was documented that short-term exposure to diesel exhaust is associated with arterial vasoconstriction and endothelial response [41].

Atherogenesis

In addition to endothelial dysfunction, the oxidative stress caused by air pollution results in the alteration of lipids that are circulating throughout the body [42]. When air pollution enters the body, it generates oxidized low-density lipoproteins (LDLs) and other highly oxidized phospholipids [42]. Proatherogenic molecules, such as oxidized phospholipids and LDLs, activate endothelial cells by diffusing into the subendothelial space [43]. This specific type of activation leads to the release of monocyte chemotactic protein-1 and vascular cell adhesion molecule-1, which are pro-inflammatory molecules. These molecules facilitate monocyte recruitment and differentiation into macrophages [43]. Ultimately, this process accelerates plaque buildup and triggers vascular inflammation (Figure 3).

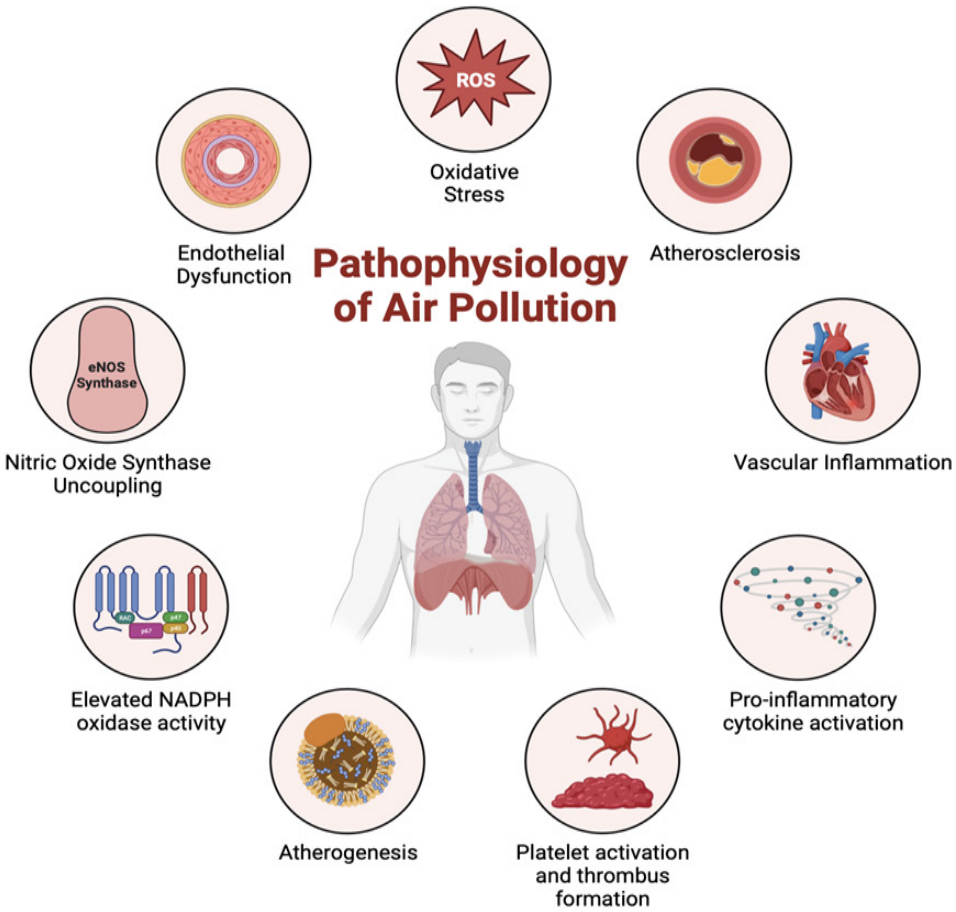

Figure 3: Pathophysiological effects of air pollution.

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Public Health Response

A recent study examined the downstream effects of reducing air pollution in patients with coronary heart disease. The results from this study showed that using a highly protective face mask reduced cardiovascular symptoms and improved health measures in this highly susceptible patient population [44]. Furthermore, during a coal sale ban in Dublin, Ireland, there was a 70% reduction in the amount of black smoke within six years. Due to this, the number of cardiovascular-related deaths significantly decreased by 10.3% (approximately 243 fewer deaths/year) [45]. From these independent cases, it is evident that air pollution is directly related to cardiovascular mortality and specific public health measures can be implemented to help alleviate this issue (Figure 4).

Climate Change

Background

The drastic changes between extreme hot and cold temperatures pose a significant risk to cardiovascular health. To understand which of the two climates has a stronger impact on CVD, researchers compared the number of cardiovascular-related deaths on the coldest and hottest 2.5% of days with cardiovascular-related deaths on days that had optimal temperatures [46]. The data were collected from hundreds of cities all over the world. It was reported that for every 1000 cardiovascular-related death, extreme hot days accounted for 2.2 additional deaths and extreme cold days accounted for 9.1 additional deaths [46]. Therefore, although both extreme temperatures contribute to adverse effects on cardiovascular health, extreme cold temperature appears to have a more detrimental effect.

Epidemiology

Cardiovascular Mortality

In a study conducted between 2006 and 2010 in thirty-six cities across China, it was found that the average temperature during cold-spell days was 2-4°C lower compared to the rest of the year. During the 2008 cold spell, the mortality from CVD increased by 52.9% (95% CI: 42.1%-64.5%) [47]. In a recent meta-analysis containing 159 studies, it was reported that with every 1°C decrease in temperature, cardiovascular mortality increased by 1.6% (95% CI: 1.015%-1.018%) and morbidity increased by 1.2% (95% CI: 1.010-1.014) [48].

Coronary Heart Disease

Cold weather plays an integral role in the development of myocardial infarction. In a Canadian study performed over a period of six years, it was noted that with every 10°C decrease in temperature, the risk of STEMI increased by 7% [49]. Additionally, no significant association was observed between STEMI and a range of warmer temperature. This clear connection between cold temperature and STEMI can

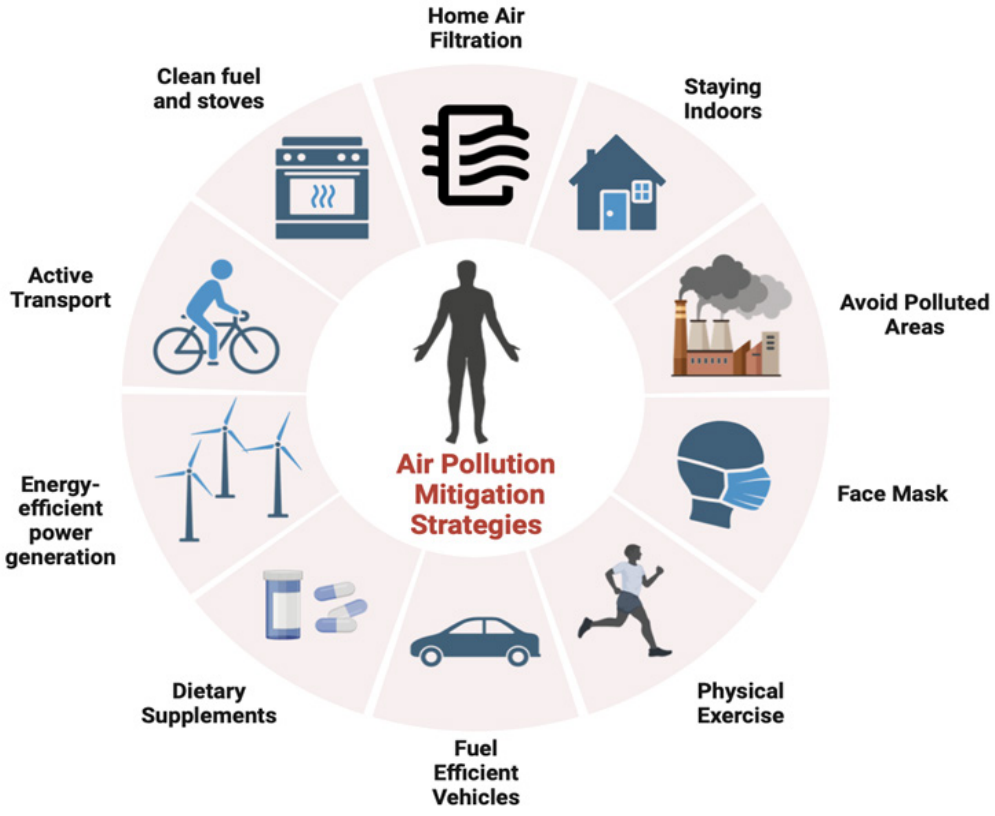

Figure 4: Various air pollution mitigation strategies that work towards reducing cardiovascular diseases.

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allow physicians to predict the risk of STEMI up to two days before the onset of infarction [49]. In another study conducted between 1996 and 2013 in Ontario Canada, cold days were found to increase the number of CHD hospitalizations by 9% (95% CI: 1%-22%) compared to hospitalizations on days with optimal temperatures [50].

Stroke

The increased risk of stroke has also been associated with changes in temperature, especially during cold days. In a 2023 case-crossover study in Lithuania, there was a significant positive relationship between cold spells and ischemic stroke. It was reported that each additional cold day in the week prior to the onset of stroke, increased the risk of ischemic stroke by 3% (95% CI: 1.00%–1.07%) [51]. Furthermore, during the summer, each cold day increased the risk of ischemic stroke by 8% (95% CI 1.00–1.16) [51]. No significant associations were identified between cold temperatures and hemorrhagic stroke.

Pathophysiology

Sympathetic Nervous System

Exposure to low environmental temperatures triggers the release of catecholamines, such as epinephrine and norepinephrine, within the body. As a result, the activation of these neurochemicals stimulates the process of thermogenesis, which is the body's natural physiological response to maintain homeostasis. Additionally, these cold temperatures initiate a downstream signaling cascade that leads to peripheral vasoconstriction. This indirectly increases the pulse and heart rate within the body which also increases the myocardial work, but decreases the subendocardial variability ratio (SEVR), an index that measures the myocardial oxygen supply [12]. Furthermore, cold temperatures elevate both the erythrocyte count and the concentration of plasma fibrinogen accompanied by a diuretic effect [52, 53]. The activation of the sympathetic nervous system due to cold temperatures also increases the activity of the renin-angiotensin system. This system decreases NO production by suppressing NO synthase activity which results in cold-induced hypertension [54].

Water Pollution

Background

Water pollution is a prevalent public health issue that impacts the health of millions of individuals across the globe. According to WHO, more than two billion people live in countries with contaminated water sources, which is expected to become exacerbated in the coming decade due to population growth and climate change [55]. Inorganic arsenic is a highly toxic substance that is commonly found in contaminated water. It is also a confirmed carcinogen, and it has been associated with CVD and diabetes. Approximately 140 million people in 70 countries have been consuming water containing arsenic at levels well above the WHO standard health guidelines of 10 µg/L [56]. The most common sources of arsenic exposure are drinking water, food prepared with contaminated water, and crops irrigated with contaminated water.

Epidemiology

Cardiovascular Mortality

Arsenic exposure has been linked to cardiovascular mortality across multiple studies. In a study conducted in Spain, drinking water from 1700 cities covering about 25 million people was examined and the standardized mortality ratio (SMR) was analyzed. It was reported that compared to the overall Spanish population, the SMR for CVD increased in cities with drinking water that contained arsenic concentrations greater than 10 µg/L [57]. Furthermore, compared to cities with arsenic concentrations less than 1 µg/L, the cardiovascular mortality rate increased by 2.2%. Another study from Bangladesh investigated the effect of varying arsenic concentration levels on cardiovascular mortality. They found that cardiovascular mortality was 214.3 per 100,000 person years when arsenic concentrations were less than 12 µg/L. In drinking water with arsenic concentrations greater than 12 µg/L, the mortality rate increased to 271.1 per 100,000 person years [58]. Therefore, it can be concluded that there is a significant association between elevated levels of arsenic concentrations in drinking water and cardiovascular mortality.

Coronary Heart Disease

A study from 2019 investigated the impact of Σas (total sum of inorganic and methylated arsenic concentrations) in young adults that were free from any cardiovascular risk factors on left ventricular (LV) size and function. They found that increased arsenic exposure was strongly associated with both measures of LV size (such as posterior wall thickness, intraventricular septum, and mass index) and LV function (such as stroke volume and ejection fraction) [59]. Additional assessments revealed that these relationships were stronger in individuals with pre-hypertension or hypertension. Moreover, elevated concentrations of arsenic increase the risk of developing coronary heart disease and peripheral artery disease [60].

Stroke

The relationship between arsenic exposure and ischemic stroke hospitalizations was explored in a study performed in Michigan. In this report, arsenic was strongly associated with an increased risk of stroke hospital admissions (relative risk of 1.03; 95% CI: 1.01-1.05 per µg/L increase in arsenic levels) [61]. From this analysis, it can be concluded that there is a high risk of stroke associated with even low levels of arsenic exposure in drinking water.

Pathophysiology

Vascular Inflammation

The digestion of arsenic from drinking water triggers the expression of pro-inflammatory cytokines such as monocyte chemoattractant protein-1(MCP-1), interleukin-6 (IL-6), and tumor necrosis factor α [62]. These cytokines are typically produced within vascular smooth cells and are increasingly expressed in atherosclerotic lesions that are formed from arsenic consumption [63]. Ultimately, the inflammatory response activated by arsenic exposure plays an integral role in atherosclerotic plaque formation.

Public Health Response

Mitigating the effects of arsenic exposure from drinking water is a difficult task. Given the fact that there is no effective treatment for arsenic poisoning, the primary goal should be to reduce the concentration and duration of exposure [64]. Some short-term measures that can be executed are to utilize rainwater for consumption instead of groundwater, operate in sanitary environments free from contaminants, and change cooking habits. Additionally, long-term measures include involving the government and local communities to evaluate the current water supply and identify ways to improve it from a public health perspective [64].

Soil Pollution

Background

Healthy soil is essential for the survival of human life. A healthy solid foundation is necessary to grow food, plant crops, and sustain population growth. Approximately 95% of the food we consume comes from soil [65]. In addition to food, soil slows climate change, fosters diverse ecosystems, and can prevent floods by storing water. Thus, soil pollution can have adverse effects on human life. Heavy metals, such as cadmium and lead, pesticides, and nano-plastic particles have all been shown to cause soil contamination and cardiovascular disease [66].

Epidemiology

Cardiovascular Mortality

In a large population-based cohort study from 2014, researchers explored the relationship between cadmium exposure with cardiovascular mortality. Urine cadmium concentration was utilized as a measure of cadmium exposure. It was reported that the hazard ratio for cardiovascular mortality was 1.43(95% CI: 1.21-1.70); thus, leading to the conclusion that urine cadmium is strongly associated with increased cardiovascular mortality [67]. Another study noted elevated blood lead levels were associated with higher cardiovascular-related deaths as indicated by a hazard ratio of 1.70(95% CI: 1.30–2.22) [68].

Cardiovascular Disease

Both stroke and coronary heart disease were shown to have strong associations with urinary cadmium concentrations as well. The hazard ratios were 1.33(95% CI: 1.05-1.68) and 1.87(95% CI: 1.22-2.86), respectively [67]. In a cross-sectional study from China, it was documented that elevated blood lead levels were associated with higher odds of carotid artery plaques (odds ratio of 1.53, 95% CI: 1.29–1.82) and cardiovascular disease (odds ratio of 1.44, 95% CI: 1.17–1.76) [66].

Pathophysiology

As mentioned previously, oxidative stress and vascular inflammation are two common signaling pathways that are initiated by pollution. Similarly, both cadmium and lead cause endothelial dysfunction, vascular damage, and atherosclerosis through oxidative processes. Figure 1 highlights how each organ system is affected by soil pollution [66].

Conclusion

According to the European Society of Cardiology, about nine million people die each year due to pollution. Approximately 60% of pollution-related death and disease worldwide is due to cardiovascular disease [69]. Evidently, environmental factors such as pollution have detrimental effects on cardiovascular health, and it has become a global public health problem. Ultimately, both short-term and long-term actions need to be taken to help mitigate the current crisis.

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Competing Interests

All the authors have read the manuscript and declare no conflict of interest. No writing assistance was utilized in the production of this manuscript.

Consent for Publication

All the authors have read the manuscript and consented for publication.

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