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Environmental Exposures and Cardiovascular Disease: A Challenge for Health and Development in Low- and Middle-Income Countries

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Summary

Environmental exposures in low- and middle-income countries lie at the intersection of increased economic development and the rising public health burden of cardiovascular disease. Increasing evidence suggests an association of exposure to ambient air pollution, household air pollution from biomass fuel, lead, arsenic, and cadmium with multiple cardiovascular disease outcomes including hypertension, coronary heart disease, stroke, and cardiovascular mortality. While populations in low- and middle-income countries are disproportionately exposed to environmental pollution, the bulk of evidence that links these exposures to cardiovascular disease is derived from populations in high-income countries. More research is needed to further characterize the extent of environmental exposures and develop targeted interventions towards reducing cardiovascular disease in at-risk populations in low- and middle-income countries.

Keywords

environmental health; air pollution; household air pollution; heavy metals; lead; arsenic; cadmium; cardiovascular disease; global health

Introduction

In the wake of large-scale economic development in low- and middle-income countries (LMIC), environmental pollution has been a challenge that has spurred tension within countries and across regions[1]. The use of fossil fuel combustion to increase access to electricity and transportation for millions of people has simultaneously modernized a multitude of rural and urban communities while locally polluting the air and globally increasing air temperatures[2–4]. Extractive industries, such as mining, have fueled the

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economies of many middle-income countries, lifting large swaths of the population out of poverty while contaminating water with heavy metals[5]. The conflict over environmental pollution is so intense in some regions that large-scale demonstrations and even violence have erupted, thus threatening national and regional security[5]. While many have argued that poverty reduction and economic growth justify the subsequent damage to the environment, the health consequences of environmental pollution, particularly for the populations residing in LMIC must also be taken into account[6,7].

Exposure to environmental pollution is associated with multiple adverse health outcomes in children and adults. While environmental pollution often evokes concerns for neurological development, cancer and pulmonary disease, cardiovascular disease must be considered as well[8]. Cardiovascular disease is the top cause of mortality worldwide, and has been identified as a target for large-scale, multisectoral intervention at the population level[9,10]. Taking into account the necessary integration of public and private sector activities to reduce the population burden of cardiovascular disease, the substantial impact of environmental exposures on the burden of cardiovascular disease at the population-level must be acknowledged and addressed[11–13]. Understanding the impact of environmental exposures on cardiovascular disease has the potential to yield greater insight into the full human cost of economic development[14].

This review will discuss the extent of the exposure, mechanisms of disease pathogenesis and the impact on cardiovascular disease for the following 5 environmental exposures: air pollution, household air pollution, lead, arsenic, and cadmium (Figure 1). While the selected environmental exposures described in this review do not represent an exhaustive list of every exposure with an observed association with cardiovascular disease, these pollutants represent the most widely studied exposures. While the focus of this review is to discuss the impact of these exposures on cardiovascular disease in LMIC, data from studies of high-income countries will be incorporated as needed to better illustrate the full impact of these exposures on cardiovascular disease risk factors and outcomes (Table 1).

Ambient Air Pollution

Fossil fuels power economic development in LIMC, fueling the expansion of industry, homes, and transportation. However, fossil fuel combustion releases a heterogeneous mixture of gases and particles, all of which are components of ambient air pollution. Particulate matter is defined as particles suspended in the air of varying chemical composition and can be separated by particle size: coarse particulate matter less than 10 micrometers in diameter (PM_{10}), fine particulate matter less than 2.5 micrometers in diameter ($PM_{2.5}$), and ultrafine particulate matter less than 0.1 micrometers in diameter ($PM_{<0.1}$). The gaseous products of fossil fuel combustion include: carbon monoxide (CO), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), nitrogen oxides (NOx), and ozone (O_3). PM are heterogeneous in chemical composition and can contain different metallic and nonmetallic compounds from different sources and may exert differential health effects. Ambient air pollution is the most robustly studied environmental exposure that has been linked to cardiovascular disease.

Extent of the Exposure

Exposure to ambient air pollution in urban and periurban communities in LMIC is often much higher than what is observed in the large metropolitan areas of high-income countries. According to the 2016 Urban Air Quality Database 98% of urban centers with more than 100,000 inhabitants in LMIC are annually exposed to $PM_{2.5}$ levels greater than 10 μg/m^3 and PM_{10} greater than 20 μg/m^3, these levels are guidelines set forth by WHO[15, 198]. Lack of robust regulation of the sources of air pollution likely contribute to disproportionate air pollution exposure in LMIC[208]. While this database did not identify the sources of PM, common sources include diesel exhaust, industrial smokestack emissions, and biomass combustion.

For example, all of the Latin American major metropolitan areas with 2013 air quality data exceed the World Health Organization standards for $PM_{2.5}$ and PM_{10}, with Bogotá, Colombia and Lima, Peru leading the cities with highest annual mean $PM_{2.5}$ concentration at 35.1 μg/m^3 and 31.5 μg/m^3, respectively[2,3]. However, air pollution in China's capital Beijing largely exceeds cities in Latin America with an annual mean $PM_{2.5}$ concentration over 80 μg/m^3 in 2015[16]. Additionally, while much of the air pollution exposure in sub-Saharan Africa results from household air pollution from biomass fuel combustion and data on ambient air pollution exposure in the region are few, it is estimated the 32% of all West Africans are exposed to $PM_{2.5}$ levels that exceed the World Health Organization limit[16]. Considerable heterogeneity of air pollution exposure can exist within large metropolitan areas as well, often disproportionately affecting low-income communities[17]. Looking to the future, the effect of temperature on $PM_{2.5}$ concentration raises concern that the impact of air pollution exposure on health might continue to increase in the wake of climate change, differentially affecting LMIC with warm climates[4,18].

Mechanisms of Disease

Ambient air pollution affects cardiovascular health largely due to systemic inflammation from the incorporation of fine particulate matter into the pulmonary interstitium[19–21]. Additionally, ultrafine particulate matter and the gaseous components of air pollution have the potential to directly enter the bloodstream[22,23]. In the presence of air pollutants, multiple biochemical effects have been observed including: increased oxidative stress through increased production of reactive oxygen species; increased inflammatory biomarkers including IL-6 and CRP; increased pro-thrombotic factors including D-Dimer, platelet activation, increased fibrinogen, thrombin generation and impaired fibrinolysis; increased expression of adhesive molecules on monocytes and leukocytes; and impaired endothelial function, including NO-mediated vasodilation[20,24–33]. The acute physiological response to exposure to ambient air pollution includes increased plasma viscosity, reduced heart rate variability, impaired vasoreactivity, vasoconstriction, increased blood pressure, and increased insulin resistance[26,30,31,34–46].

Impact on Cardiovascular Disease

Chronic exposure to ambient air pollution has been associated with risk factors for cardiovascular disease in multiple cohorts. The association between chronic air pollution exposure and elevated blood pressure has been extensively studied, including data from

multiethnic cohorts in several countries[37,47–49]. Additionally, some evidence has emerged supporting an association between air pollution exposure with elevated fasting glucose and Type 2 Diabetes Mellitus[47,50,51]. Yet, the data have not been entirely consistent and additional studies on the factors that increase vulnerability to the blood pressure effects of air pollution exposure are needed, including a greater understanding of the specific air pollutants that account for the observed cardiometabolic effects[52,53]. The majority of the studies of ambient air pollution and cardiovascular risk factors were conducted in high-income countries, with very few studies conducted in LMIC[41].

Exposure to ambient air pollution is associated with multiple measures of subclinical cardiovascular disease. Ambient air pollution has been associated with measures of subclinical atherosclerosis including carotid-intimal thickness and aortic atherosclerotic plaques[54–58]. There is evidence that air pollution exposure is also associated with the progression of coronary calcium[59]. Additionally, air pollution exposure has also been associated with adverse cardiac remodeling, including right and left ventricular hypertrophy[55,60,61]. While most of these studies were conducted in high-income countries, several small studies in LMIC have recently emerged including a study of occupational air pollution exposure and cardiac structure and function in Iran[62].

Beyond subclinical cardiovascular disease, large studies have demonstrated a strong association between ambient air pollution exposure and adverse cardiovascular outcomes. Acute ambient air pollution exposure has been associated with angina, stroke, acute myocardial infarction, heart failure hospitalization, arrhythmias, cardiac arrest, heart failure hospitalization and cardiovascular mortality[48,63–81]. Data that are specific to LMIC are largely conducted in upper middle-income countries including China and in Latin America[48,82–89]. Of note, almost no studies of air pollution and cardiovascular disease in sub-Saharan Africa have been published. The discrepancy between the relatively high exposure to ambient air pollution in LMIC and the lack of data specific to LIMC suggests that the public health impact is potentially underestimated.

Household Air Pollution from Biomass Fuel Use

While economic development in LMIC has improved access to electricity, natural gas and liquefied petroleum gas, many communities depend on biomass fuels for daily needs[90]. Biomass fuels include wood, charcoal, dung and crop residue, which are burned in indoor and outdoor stoves for cooking and heating. Similar to fossil fuel combustion, biomass fuels produce gases and particulate matter that are suspended in air, including carbon monoxide and fine particulate matter ($PM_{2.5}$). Exposure to the components of biomass fuel combustion has been studied in several contexts in relation to cardiovascular disease risk factors and outcomes.

The Extent of the Exposure

Household air pollution from biomass fuel use affects 3 billion people worldwide, including 6.5 million Americans[90, 199]. While biomass fuel use can be found on every continent, it is more prevalent in resource-poor settings, disproportionately affecting low-income individuals in HIC and LMIC[199]. In many cultures women are more likely to perform

household cooking, and thus are more highly exposed to smoke from biomass fuel use along with small children in the home. The geographic distribution of biomass fuel use can vary by region due to social, cultural, economic, and climate differences. For example, in the Andean region of South America, daily biomass fuel use is primarily confined to rural communities[91]. In contrast, a large study in peri-urban Malawi found that 70.9% of the 6,445 households surveyed use wood and/or charcoal for cooking[92]. Furthermore, older age and low education were associated with the use of wood for cooking. Understanding and addressing the social and cultural factors that contribute to biomass fuel use is critical and has implications for the implementation of improved cook-stove interventions.

Mechanisms of Disease

The biochemical and physiologic response to the air pollutants released from biomass fuel combustion has not been as extensively studied as air pollution from fossil fuel combustion. While both forms of combustion release fine particulate matter, the chemical composition of the particulate matter vary according to fuel source, and some studies suggest that the chemical composition and diameter of particulate matter has differential impact on cardiovascular disease outcomes[69,93–95]. Coarse particulate matter is often found in ocean spray, dust, and construction byproducts. Acute exposure to wood smoke has been showed to cause arterial stiffness and decreased heart rate variability[96]. Additionally, observational studies conducted in women in villages in eastern India observed increased pro-inflammatory cytokines, higher serum c-reactive protein, and higher reactive oxygen species generation in the women exposed to biomass fuel smoke[97]. Another study of women in rural India observed an increase in systolic blood pressure during cooking times during which there was also an increase in exposure to the air pollutant black carbon, a major component of soot[98]. Additional research on the acute biochemical and physiologic response to household air pollution from biomass fuel combustion is needed to better understand how this exposure differs from ambient air pollution.

Impact on Cardiovascular Disease

Exposure to biomass fuel smoke has been associated with cardiovascular risk factors in multiple observational studies. The most common cardiovascular risk factor associated with biomass fuel use is elevated blood pressure. Multiple cohort studies in China, Peru, Guatemala and Nicaragua have identified an association between exposure to biomass fuel smoke and elevated blood pressure[99–104]. Replacement of traditional cookstoves with cleaner burning cookstoves was associated with lower blood pressure[101]. In addition to observing differences in blood pressure, 2 large studies in China and Peru also observed an increased prevalence of hypertension in daily biomass fuel users[100,103].

Exposure to biomass fuel smoke has also been associated with subclinical cardiovascular disease in several small studies. In Guatemala, biomass fuel use was associated with changes in the ST segment of the electrocardiogram in women prior to participating in an improved cookstove trial[105]. These changes improved after the cookstove intervention, suggesting an improvement in myocardial ischemia. Additionally, a cross sectional study of 266 individuals in Puno, Peru found that chronic exposure to biomass fuel smoke was associated with increased carotid intima-media thickness and a higher prevalence of carotid

atherosclerotic plaques[106]. However, contrary to what was previously hypothesized, in a sample from the same Peruvian cohort there was no association between biomass fuel use with elevated NT pro-BNP or right ventricular systolic pressure by echocardiography[107]. A small echocardiography study in a single hospital in Turkey observed that biomass fuel users had increased right ventricular systolic pressure and decreased left and right ventricular myocardial indices, indicating decreased biventricular systolic function[108]. However, the relationship between biomass fuel smoke exposure and cardiac structure and function is currently undergoing further examination in population-based cohorts.

There have been conflicting results in studies of the association of household air pollution from biomass fuel use with outcomes, such as coronary heart disease and cardiovascular mortality. While the Global Burden of Disease Study estimated the global impact of household air pollution due to biomass fuel use based on the observed relationship between ambient air pollution exposure and cardiovascular events, very few studies have examined cardiovascular outcomes in biomass fuel users. Emerging data suggest an association between biomass fuel use and coronary heart disease[109]. In a study of participants living in the Brazilian Amazon, elderly individuals with increased exposure to biomass fuel smoke had increased cardiovascular mortality when compared to age-matched controls[110]. However, large cohorts in Iran and Bangladesh have failed to demonstrate an association between chronic biomass fuel use and cardiovascular mortality[111,112]. Additional studies that prospectively study cardiovascular outcomes in biomass fuel users compared to nonusers are needed to better quantify the impact of household air pollution on cardiovascular disease.

Lead

The acute and chronic neurological effects of lead exposure have been widely described in both high-income countries and LMIC[113]. However, less public attention has been paid to the cardiovascular impact of chronic lead exposure and the contribution of heavy metal exposure on the burden of cardiovascular disease in LMIC. Globally, it is estimated the lead exposure ranks #26 as a risk factor for disability-adjusted life-years lost, yet in sub-regions of Latin America and Southern Africa this ranking rises to #20[90]. Lead exposure in LMIC deserves close examination as a modifiable risk factor for cardiovascular disease and a potential target for intervention at the population level.

Extent of the Exposure

Globally, an estimated 26 million people are at risk for lead toxicity, resulting in a loss of 9 million disability-adjusted life-years[114]. While lead exposure exists in high-income countries and LMIC alike from lead pipes and paint, in general, the prevalence of lead exposure has not decreased in LMIC to the degree that has been observed in many high-income countries[115,116]. Tobacco use is a common mode of lead exposure in HIC and LMIC however, there are multiple sources of lead exposure that are specific to the industries and cultures of LMIC[117]. While leaded petroleum was banned from high-income countries many decades ago, its use in LMIC continues in Yemen, Algeria, and Iraq, polluting the air and soil[114,118, 200]. Additionally, occupational exposures in battery manufacturing and recycling

factories have been well described, particularly in Kenya and several South Asian countries[119,120, 203]. Mining operations in Peru, Tanzania, Nigeria and Zambia have been associated with lead exposure not only for the workers at the mine, but also for the local communities located near the mines[121–123]. Toxic waste from other industrial sources, is also known to contaminate water and soil with lead[113]. Fishing and hunting with lead tools fashioned from industrial sources are associated with chronic lead exposure in Peruvian Amazon River Basin communities[204]. Moreover, the artisanal use of lead in pottery has also be a source of lead exposure in Latin America and Africa[114,115,124,125], and leaded paints are still being sold and used in some LMIC, as noted in a recent study in Cambodia[126]. Independent of the source of the lead contamination, children are often the most vulnerable population exposed to lead, with often unmeasured detriment to the present and future neurological and cardiovascular health[113,122,125,127–129].

Mechanisms of Disease

By promoting the generation of reactive oxygen species, lead increases oxidative stress in cardiovascular tissues and endothelial cells[130]. The increase in oxidative stress in the setting of lead exposure is also associated with decreased nitric oxide (NO) availability. Decreased NO availability in turn has been shown to cause sodium retention, vasoconstriction, and increased adrenergic tone[130]. Additionally, NFκB activation due to increased oxidative stress in the setting of lead exposure leads to the oxidation of LDL, increases the expression of adhesive molecules on monocytes and increases foam cell formation[130]. These processes in addition to platelet activation and vascular remodeling are the basis by which lead associated cardiovascular disease occurs[130].

Impact of Cardiovascular Disease

Hypertension is the cardiovascular risk factor most greatly associated with lead exposure. Multiple studies in the United States in addition to several studies in LMIC have demonstrated a convincing association between even low levels of lead exposure and increased blood pressure, gestational hypertension incidence, and hypertension prevalence[119,131–133]. Moreover, some evidence suggests the lead exposure is also associated in with decreased heart rate variability[131]. However, emerging evidence suggests that lead exposure is also associated with other cardiometabolic derangements including increased fasting glucose, decreased HDL, increased total cholesterol, and increased prevalence of the metabolic syndrome[124,134,135]. Several of these studies of cardiometabolic impairment in the setting of lead exposure were conducted in LMIC settings, including multiple settings in West Africa and the Americas, thus highlighting the potential role of environmental exposures on non-communicable disease risk in LMIC.

Lead exposure is associated with subclinical cardiovascular disease and cardiovascular outcomes. Increased carotid intimal medial thickness has been observed in association with increased serum lead levels in a Turkish population with concomitant renal disease[136]. Lead exposure has also been associated with reduced heart rate variability and abnormalities of cardiac structure and function, including increased left ventricular hypertrophy and decreased ejection fraction[131]. Clinical atherosclerotic disease has been observed in association with lead exposure including stroke, peripheral arterial disease, and coronary

heart disease[131,137]. Increased exposure to lead has also been associated with increased cardiovascular mortality in several studies of the United States population[131,138,139]. Despite the considerable exposure to lead in LMIC, there are limited published data on lead exposure and cardiovascular disease outcomes in LMIC populations.

Arsenic

Arsenic is a naturally occurring metalloid and a contaminant of drinking water, soil, and food. In contrast to acute arsenic poisoning, chronic arsenic exposure can be more difficult to identify, but ultimately is association with multiple adverse health outcomes, including CVD[114].

Extent of the exposure

Chronic arsenic exposure has been described in countries of all income levels and most commonly occurs from drinking wells contaminated with arsenic naturally present in the soil[114]. While arsenic contamination of wells within the US have been well documented, particularly in Native American reservations, arsenic contamination in LMIC countries including Bangladesh, India, Taiwan, and Turkey has also been well-documented[140–144]. However, as safe drinking water becomes more readily available within high-income countries, arsenic contamination from well water disproportionately affects low-income communities, such as Bangladesh where an estimated 20 million inhabitants consume arsenic contaminated water[142,144, 205]. Arsenic contamination of food such as rice represents a particularly important exposure risk factor for inhabitants of LMIC and constituents of global trade partners[205–207].

Mechanisms of disease

Arsenic typically enters the body through the gastrointestinal tract and is metabolized in the liver where it undergoes methylation, yielding toxic intermediates[145]. Arsenic exposure is associated with increased inflammatory markers, including IL-6 and IL-8, and matrix metalloproteinase- 2 and -9[146,147]. In animal models, arsenic exposure leads to myocardial fibrosis, which is proposed to be the mechanism by which QT prolongation in electrocardiogram occurs in response to arsenic toxicity[148]. Additionally, endothelial dysfunction associated with arsenic exposure has also been observed. In Bangladesh, gene by environment interaction in relation to increases in blood pressure from arsenic exposure has been well described, demonstrating variable cardiotoxicity due to variable methylation of arsenic[149–151]. In a separate Bangladesh study, folate supplementation promoted urinary excretion of arsenic and may attenuate arsenic toxicity[201].

Impact on Cardiovascular Disease

Chronic arsenic exposure has been associated with cardiovascular risk factors. While elevated blood pressure and hypertension in response to arsenic exposure has been observed in multiple LIMC settings including India, Bangladesh, Mexico and China, this observation has not been consistent[152–158]. Additionally, Type 2 Diabetes Mellitus, elevated triglycerides, and elevated total cholesterol have also been observed in association with arsenic exposure[142,156,159].

Chronic arsenic exposure is associated with subclinical cardiovascular disease, including increased carotid intimal-medial thickness, which has been observed in several studies in LMIC including Mexico and Bangladesh[156,160,161]. Moreover, left ventricular ejection fraction is reduced in children chronically exposed to arsenic in Mexico[157]. In terms of clinical cardiovascular disease, arsenic exposure is associated with peripheral arterial disease, cardiomyopathy, coronary heart disease, acute myocardial infarction, stroke, stroke mortality, and cardiovascular mortality[162–171]. Not only does arsenic exposure increase the risk of acquired heart disease, but also is associated with increased risk of congenital heart disease[172]. As more evidence is generated regarding the full spectrum of cardiovascular disease associated with chronic arsenic exposure, the potential cost of arsenic contamination in LMIC is being appreciated.

Cadmium

Cadmium does receive the same degree of attention from the lay public as lead or arsenic, however the public health burden in relation exposure to cadmium remains significant[114]. An estimated 5 million people are exposed chronically to cadmium, which has implications for cardiovascular disease risk at the population level in many LMIC[114].

Extent of the Exposure

Similar to lead, cadmium exposure commonly occurs from tobacco smoking, an exposure that has been well described in high-income countries and LMIC[132,173]. Additionally, cadmium from mining, smelting, refining and industrial waste can also pollute air, water, and soil leading to the contamination of foods including leafy vegetables, fish, and shellfish[114,173]. Cadmium is also used in the production of plastics, fertilizers, and batteries[114,174]. Communities in LMIC, particularly low-income communities, may be chronically exposed to cadmium, an exposure that is only recently made apparent as heavy metal monitoring is implemented in communities, as illustrated by studies from Ghana and Uganda[175,176]. In fact, cadmium exposure is likely to increase in the coming decades in part due to electronic waste disposal, as seen in Nigeria,[202]. Beyond contamination of the environment, serum levels of cadmium from individuals living in LIMC can be several orders of magnitude greater than what observed in high-income countries[177].

Mechanisms of Disease

Cadmium increases oxidative stress through the increased production and decreased metabolism of reactive oxygen species[178]. Moreover, cadmium has been shown to impair endothelial function[179]. Cadmium also has been associated with increased serum levels of galetin-3, a biomarker for myocardial fibrosis, in a population in Turkey[180]. Through these multiple mechanisms cadmium exposure is thought to cause cardiovascular disease.

Impact on Cardiovascular Disease

Similar to other environmental exposures, cadmium exposure is associated with elevated blood pressure and hypertension[181–184]. While much of the evidence was generated in high-income countries, a number of studies have been conducted in LIMC, including Thailand, China and Pakistan[185–187]. Of note, there are several studies that did not find an association

between measured cadmium exposure and hypertension, suggesting that additional data on the genetic and environmental risk factors for cadmium-related hypertension is needed[188,189]. Cadmium has also been associated with cardiometabolic derangement including Type 2 Diabetes, as noted in a study from China[187]. Additionally, increased carotid intimal-medial thickness and carotid plaques also have been associated with cadmium exposure[136].

The evidence regarding the association between cadmium exposure and cardiovascular disease outcomes overwhelmingly comes from high-income countries. Cadmium exposure is associated with diseases of atherosclerosis including peripheral arterial disease, stroke, ischemic heart disease, and acute coronary syndromes[182,190–193]. Cadmium exposure has also been associated with incident heart failure, although it unclear what percentage of heart failure cases are ischemic versus nonischemic in etiology[182,192,194]. The largest studies of cadmium and cardiovascular disease are from US NHANES data and the Strong Heart Study of US Native Americans. In these cohorts, cadmium exposure was associated with cardiovascular mortality, thus highlighting the likely unmeasured mortality burden that cadmium exposure potentially has in LMIC[195–197].

Summary

Environmental exposures in LIMC lie at the intersection of increased economic development and the rising public health burden of cardiovascular disease. Increasing evidence suggests an association of exposure to ambient air pollution, household air pollution from biomass fuel, lead, arsenic, and cadmium with multiple cardiovascular disease outcomes including hypertension, coronary heart disease, stroke, and cardiovascular mortality. While populations in LMIC are disproportionately exposed to environmental pollution, the bulk of evidence that links these exposures to cardiovascular disease is derived from populations in high-income countries. Low-income regions of high-income countries are at high risk of exposure. In order to better understand the extent to which environmental exposures contribute to the rising epidemic of cardiovascular disease in LMIC and develop interventions to reduce cardiovascular disease risk at the population level, additional research is needed.

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Key Points

- Environmental exposures, including air pollution and heavy metal and metalloid contamination, are more prevalent in low- and middle-income countries.
- Exposure to air pollution in the form of ambient air pollution and household air pollution from biomass fuel use is associated with hypertension, acute myocardial infarction, heart failure, arrhythmia, sudden cardiac death and cardiovascular mortality.
- Lead, arsenic and cadmium exposure is associated with hypertension, coronary heart disease and cardiovascular mortality.
- There is increasing epidemiological evidence of an association of environmental exposures with cardiovascular risk factors and cardiovascular disease, yet most of the research has been conducted in high-income countries.

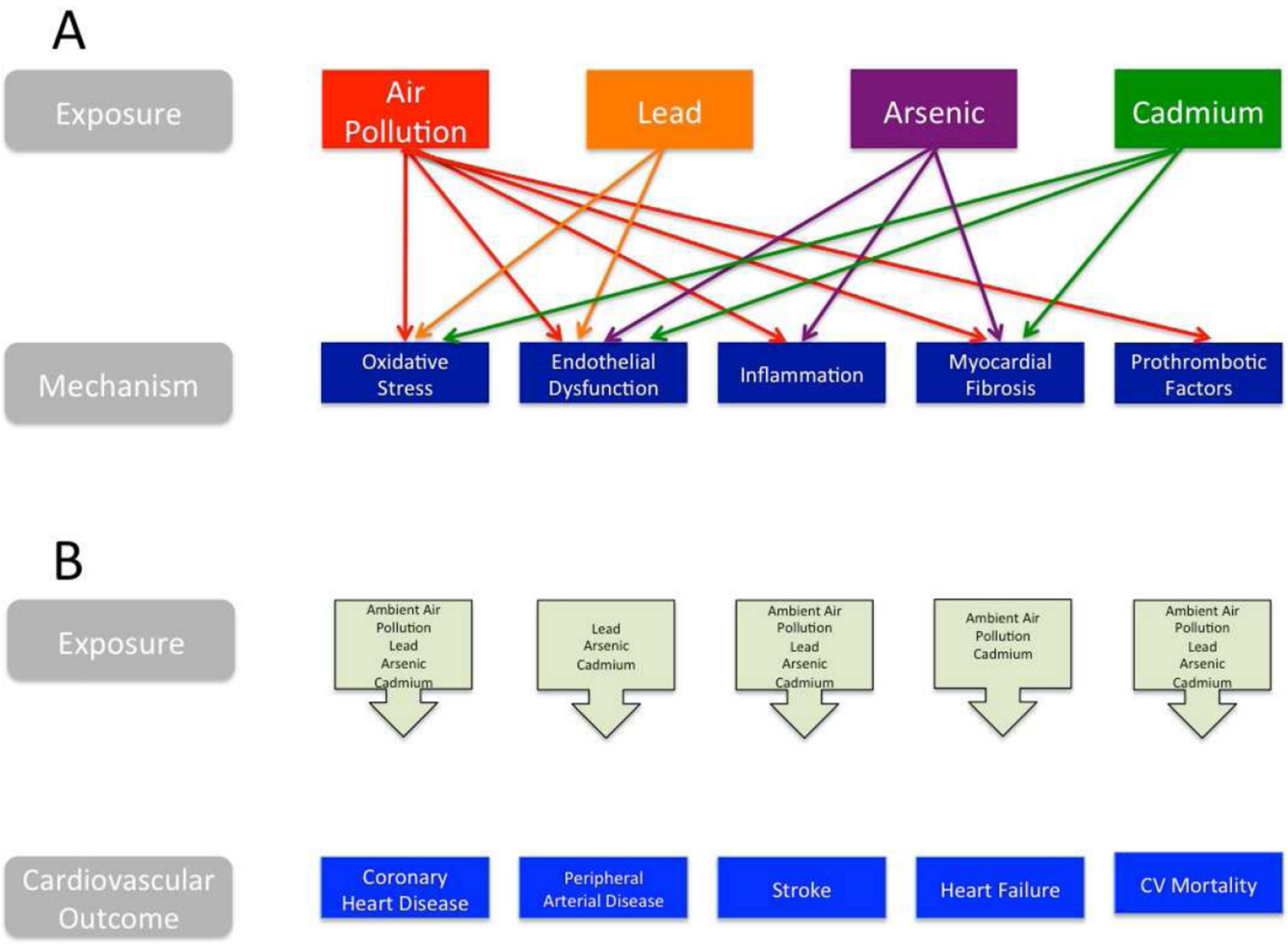

Figure 1.

Summary of the Association Between Environmental Exposures, Pathophysiologic Mechanisms and Cardiovascular Disease. Panel A illustrates the multiple mechanisms by which selected environmental exposures cause cardiovascular injury. Panel B shows the multiple cardiovascular outcomes that are associated with environmental exposures.

Table 1

The Origin of the Peer-Reviewed Literature on the Association Between Selected Environmental Exposures and Cardiovascular Disease by World Bank Country Income-Level

	Hypertension	Subclinical Atherosclerosis	Cardiac Structure and Function	Coronary Heart Disease	Heart Failure	Cardiovascular Hospitalization	Arrhythmia	Stroke	Cardiovascular Mortality
Ambient Air Pollution									
Biomass Fuel Air Pollution									
Lead									
Arsenic									
Cadmium									

Data derived from low- and middle-income countries

Data derived from high-income countries

Data derived from both low- and middle-income countries and high-income countries

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