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# Preventive Measures in Cardiology: Behavioral and Environmental Determinants of Cardiovascular Disease

Hung Quoc To
Toquochung2010@yahoo.com

## ABSTRACT

Cardiovascular disease (CVD) remains the leading cause of global mortality. As of 2017, more than 17 million deaths were attributed to CVD, with over 360 million disability-adjusted life years lost, making CVD the highest-burden disease worldwide. As this burden continues to rise, it is essential to understand how behavioral and environmental conditions predispose individuals to CVD within socioeconomic and environmental contexts. In this review, the term "lifestyle factors" refers to health behaviors influenced by socioeconomic and environmental conditions. Using a health equity lens, this review examines how behavioral and environmental factors are linked to structural disparities, contributing to unequal distributions of cardiovascular risk.

Key risk factors of CVD include hypertension, diabetes, obesity**,** poor diet, physical inactivity, tobacco use, and alcohol use. Prevention strategies focus on health behavior modifications that may address multiple risk factors simultaneously. Sustained physical activity (PA) has been shown to lower cardiovascular mortality. Likewise, consumption of whole grains, fruits, vegetables, proteins, and omega-3 fatty acids may decrease cardiovascular mortality. However, the ability to adopt protective behaviors and avoid detrimental exposures varies across populations due to distinct neighborhood conditions, environmental conditions, and resource availability. Therefore, cardiovascular risk is often observed in structurally disadvantaged settings with increased exposure and limited access to protective conditions.

CVD is a multifactorial disease characterized by interactions among behavioral, biological, and environmental factors. Prevention strategies that integrate individual behaviors with social and environmental conditions may substantially alleviate the global burden of CVD and reduce cardiovascular inequities.

***Keywords:*** Cardiovascular disease (CVD), population health, behavioral and environmental determinants, chronic exposure, health equity, physiological stress accumulation

## INTRODUCTION

Chest pain, fatigue, and shortness of breath are symptoms of cardiovascular disease. Despite medical advances, the global burden of CVD continues to escalate, with 20.5 million deaths recorded in 2021.[1,2,3] Heart disease refers to multiple conditions, such as coronary artery disease (CAD), cardiomyopathy, heart failure (HF), and rheumatic heart disease.[4] These conditions occur alongside an unequal distribution of modifiable risk factors. Behavioral determinants such as PA, diet, sleep duration, and tobacco use do not occur independently. They are influenced by and constrained within both socioeconomic and environmental contexts. These factors vary across social position, including race, socioeconomic status, gender, age, and housing stability. They influence an individual's pollution exposure, access to safe activity spaces, cumulative stress burden, and preventive care, which in turn correlate with cardiovascular outcomes.[5] CAD, characterized by the accumulation of plaque in the coronary arteries, illustrates how these conditions may culminate in adverse outcomes.[6] CAD mortality is associated with a lack of physical activity, with physically inactive individuals experiencing significantly higher risks of premature CVD and hypertension.[7] This pattern differs based on neighborhood safety, access to recreational spaces, as well as transportation infrastructure.

Sleep disturbances and chronic psychosocial stress occur at greater rates among individuals experiencing housing instability or economic insecurity than among those who do not experience these conditions. In this review, behavioral risk factors are socially structured and rooted in differences in social, economic, and environmental conditions. Physical activity, diet, sleep, substance use, and stress responses are shaped by neighborhood environments, working conditions, labor organization, food systems, environmental exposures, and access to resources. These influences are distributed inequitably across groups, reflecting long-standing socioeconomic and racial inequities that correspond to consistent differences in cardiovascular risk. When these behaviors are positioned solely as matters of personal responsibility, the structural forces that limit opportunities for cardiovascular health may contribute to disparities that would be obscured. Positioning these factors within a public health and health equity framework, this review incorporates the concept of allostatic load, describing the cumulative physiological "wear and tear" on the body following chronic stress. This concept describes how ongoing external pressures exert cumulative biological effects over time. Repeated stress responses contribute to inflammation, elevated blood pressure, metabolic changes, and disrupted sleep duration, which may elevate cardiovascular risk over time.[8] By bringing behavioral and environmental risk factors together under an equity lens, these findings may strengthen CVD prevention efforts. Table 1 shows how individual exposures fit into broader structural and systemic settings.

**Table 1. Behavioral and Environmental Cardiovascular Risk Domains, Affected Populations, and Policy-Relevant Structural Drivers**

| Domain of Risk Factors | Primary Exposures or Behaviors for Risks | Most Affected Populations by Risk Factors | Structural Components that Influence Risk Factors | Strategies for Policy and Systems Change |
|---|---|---|---|---|
| Ambient Air Pollution | Long-term exposure to fine particulate matter (PM2.5), traffic emissions, and industrial pollutants | Low-income communities, communities of color, and residents near highways or industrial zones | Residential segregation, zoning decisions, transportation infrastructure, and weak emissions enforcement | Air quality standards, emissions regulation, land-use planning, and environmental justice enforcement |
| Contaminated Drinking Water | Arsenic, lead, and agricultural runoff | Rural populations, Indigenous communities, low-income households, and areas with aging infrastructure | Underinvestment in water systems, regulatory gaps, and reliance on groundwater | Water infrastructure investment, regulations |
| Diet and High-fat, High-Sugar, and High-Salt (HFSS) Foods | High intake of ultra-processed, high-fat, high-sugar, high-salt foods | Low-income households, racial and ethnic minority communities, and food-insecure populations | Food deserts, targeted marketing, pricing, and subsidy structures | Regulation of food marketing, zoning of food outlets, nutrition assistance programs, and pricing policies |
| Physical Activity | Sedentary behavior, limited access to safe activity spaces | Low-income communities, older adults, women, and people with disabilities | Built environment design, neighborhood safety, transportation access, and work schedules | Walkable urban design, public transit investment, park development, community safety initiatives |
| Sleep Duration and Insomnia | Short sleep duration, chronic insomnia | Shift workers, individuals with housing instability, and racial and ethnic minority populations | Labor practices, housing conditions, noise exposure, and chronic stress | Work-hour protections, housing stability policies, and neighborhood noise regulation |
| Psychosocial Stress | Chronic stress exposure, sustained neuroendocrine activation | Communities facing discrimination, economically insecure populations | Racism, economic insecurity, unsafe working conditions, and housing instability | Labor protections, anti-discrimination policy, and community safety investments |

| | | | | |
|---|---|---|---|---|
| Alcohol Consumption | Heavy or harmful alcohol use | Individuals with lower educational attainment, communities exposed to intensive marketing | Alcohol pricing, availability, targeted advertising, and stress exposure | Taxation, advertising restrictions, outlet density regulation, and access to treatment |
| Tobacco Use | Cigarette smoking, secondhand smoke exposure | Low-income populations, service-sector workers, and communities with high retailer density | Retailer density, marketing practices, and limited smoke-free protections | Comprehensive smoke-free laws, taxation, retailer density regulation, and cessation access |

Table 1 (produced by the author) summarizes behavioral and environmental factors that influence cardiovascular risk, showing how individual exposures fit within broader structural and systemic contexts. This framework indicates that cardiovascular health is connected to social environments and highlights policy/system-level changes that may help reduce health inequities.

## METHODS

This literature review examines the behavioral and environmental determinants of CVD, with a focus on population trends and health equity. The primary literature search was conducted between January and August 2025. Additional literature searches were conducted during manuscript revisions in 2026 to incorporate new and more relevant evidence. Google Scholar and PubMed were searched using Boolean operators to identify relevant peer-reviewed literature published between 2015 and 2026. The search query combined terms related to cardiovascular outcomes with behavioral and environmental exposures, including: "cardiovascular disease" OR "heart disease" OR "CVD" AND "physical activity" OR "diet" OR "stress" OR "smoking" OR "alcohol" AND "environmental exposures" OR "air pollution" OR "water contamination" OR "neighborhood conditions" OR "food environment". Additional searches linked the terms: "mortality," "incidence," "hypertension," and "coronary artery disease." The selected search terms were chosen to find studies focused on the many modifiable behavioral and ecological factors that are major targets for CVD prevention. The terms "lifestyle" and "CVD prevention" were used to find studies of health-related behaviors, ranging from physical activity, diet, sleep, tobacco use, and alcohol consumption. The term "environmental risk factors" was used to identify studies on structural exposures, such as air pollution, water quality, neighborhood conditions, and food systems. Collectively, these terms align with the theoretical focus of this review by identifying pathways through which repeated exposures and constrained opportunities compound cumulative physiological strain, in relation to the concept of allostatic load.

A subsequent full-text review was performed to determine if studies met the established inclusion criteria. A qualitative synthesis of research evidence was conducted to provide a cohesive narrative,

with attention to biological mechanisms and variations in exposure and risk across groups. Studies were included if they were peer-reviewed, examined CVD or cardiovascular risk outcomes, and evaluated at least one behavioral, environmental, or structural exposure within the scope of this review. While studies were excluded if they did not contain cardiovascular-related outcomes, did not address the behavioral or environmental topics evaluated in this review, or were not in the field of CVD prevention.

Titles and abstracts were thoroughly assessed for relevance to the review objectives, along with a full-text analysis of potential studies to determine whether they met the inclusion criteria. Due to the narrative scope of this review, no formal quantitative synthesis of the evidence or standardized risk-of-bias evaluation was conducted. However, several aspects of the study design, including key analytical limitations (i.e., many of the studies in the literature were observational and may have been subject to confounding), were considered when interpreting the results of each study included in this review. Additionally, the peer-reviewed literature is supplemented by information from public health agency reports and international organizations that provide population data and policy and regulatory frameworks, to support the article when evidence was limited in the current body of scholarly research. These sources support contextual and policy background and cannot be used to establish causation between an exposure and an outcome. They are differentiated from empirical research studies throughout the review. Although no formal evidence-grading framework was utilized, the credibility of the findings was evaluated based on study design, sample sizes, methodological limitations, and alignment of findings among the broader literature. The review focuses on key domains (sedentary behavior and physical activity (PA), high-fat, high-sugar, and high-salt (HFSS) and ultra-processed foods, air pollution and CW, sleep duration and insomnia, psychosocial stress and stress hormones, and alcohol and tobacco use) given their potential for modification in CVD prevention and their connection to social and environmental conditions. Hypertension and dyslipidemia (cholesterol) were not treated as primary domains because they function as intermediate clinical risk factors that may occur along with multiple upstream behavioral and structural exposures. Discrimination functions as a major structural stressor that may contribute to varying levels of cardiovascular health.

## DISCUSSION

### *Ambient Air Pollution and Cardiovascular Risk*

Air pollution represents a foundational exposure that contributes to cumulative physiological strain through repeated vascular and inflammatory stress.[9] Ambient air pollution (AAP) is a major risk for CVD, as it is one of the most consequential environmental contributors to cardiovascular morbidity and mortality.[10] Multiple air pollutants contribute to cardiovascular morbidity, particularly particulate matter (PM), a mixture of small airborne particles including dust, dirt, soot, and smoke. Because exposure sources vary, individuals may be exposed to a wide range of pollutants, notably PM2.5 (fine

particles with a diameter of 2.5 microns or less).[11] Exposure to air pollution varies based on where people live, work, and travel. AAP comprises PM and gaseous pollutants (ozone, nitrogen oxides, carbon monoxide, and sulfur dioxide), as well as toxic compounds such as benzene, polycyclic aromatic hydrocarbons, and acrolein.[12] The effect of PM on the heart depends on particle size, measured by the aerodynamic diameter (AD) of the particle.[10] Fine particulate matter (PM 2.5) poses cardiovascular risk due to its ability to penetrate the alveolar membranes[10] and enter the body's circulatory system, where it may trigger inflammation and oxidative stress that amplify cardiovascular mortality. Over time, chronic exposure may culminate in acute cardiovascular events, such as heart attacks and arrhythmias, or premature death.[13] AAP levels remain persistently elevated worldwide.[14] This determinant may also influence behavioral risk factors, as individuals living near major roadways or industrial areas may be discouraged from outdoor physical activity.[15] In 2019, 99% of the world's population lived in areas that did not meet the World Health Organization (WHO) air quality guidelines.[16] In the United States, almost 79.5 million U.S. residents lived within 500 meters of major roadways in 2020, with pollution concentrations highest within 0-300 meters from the point of release.[17] Globally, around 2.4 billion people are exposed to levels of AAP, with 7 million at risk of premature mortalities each year.[16] Table 2 presents the estimated premature mortality associated with various outdoor air pollution sources in different geographic regions worldwide.

**Table 2. Estimated Contributions of Major Outdoor Air Pollution Sources to Premature Mortality Globally and in the United States.**

| Pollution Source Category | Share of Global Premature Mortality (%) | Share of U.S. Premature Mortality (%) | Regions With Highest Contribution | Dominant Pollutant Constituents |
|---|---|---|---|---|
| Residential Energy Use | 31 | 6 | China, India, Indonesia, Vietnam | Carbon dioxide, carbon monoxide, volatile organic compounds, nitrogen oxides, sulfur dioxide, mercury, fine particulate matter |
| Agricultural Activities | 20 | 29 | Europe, Russia, Japan, eastern United States | Ammonia-derived inorganic fine particulate matter, sulfates, nitrates |
| Power Generation | 14 | 31 | United States, Russia, Korea, Turkey | Sulfate- and nitrate-containing fine particulate matter, mercury |
| Industrial Sources | 7 | 6 | Japan, Germany, China | Sulfate-containing fine particulate matter, volatile organic compounds, and hydrocarbons |

| | | | | |
|---|---|---|---|---|
| Biomass Burning | 5 | 5 | Canada, Africa, South America, Australia, Southeast Asia | Fine particulate matter, nitrogen oxides, carbon monoxide, sulfur dioxide, lead, mercury |
| Land Transportation | 5 | 21 | United States, Germany, Russia, Japan | Ultrafine particles, fine particulate matter, nitrogen oxides, volatile organic compounds, ozone |
| Natural Sources | 18 | 2 | Africa, Middle East | Coarse particulate matter, airborne dust |

Table 2 illustrates significant regional variations for the primary sources of outdoor air pollution that add to premature deaths in both global and U.S. settings. The estimates presented show the relative contribution of the predominant sources of pollution and demonstrate the geographic variability in how populations are exposed to pollutants. The data were compiled by the author and synthesized from Bhatnagar et al.[17]

Evidence linking AAP exposure to cardiovascular morbidity and mortality is substantial, though many of the population-level findings are observational. Thus, there is a possibility that the associations found might be affected by the presence of residual confounding due to such factors as socioeconomic status, occupational and environmental exposure, and preexisting health state. Exposure to AAP is ubiquitous and varies by neighborhood and region. Communities with lower socioeconomic status and communities of color experience disproportionately higher exposure in relation to historical zoning practices, residential segregation, proximity to industrial facilities, and transportation infrastructure.[18] During periods of poor air quality, individuals may lower their exposure by staying indoors, sealing windows, and wearing masks. Ultimately, these findings support population-level and policy-based interventions, including emissions regulations, land-use planning, and enforcement of air quality standards, to advance environmental justice.[19] While individuals may take preventive measures to limit their exposure, lowering cardiovascular risk from air pollution also depends on addressing the conditions that impact exposure throughout multiple communities.

### *Contaminated Water and Cardiovascular Risk*

Chronic exposure to contaminated drinking water could account to long-term physiological strain, increasing cardiovascular vulnerability over the lifespan.[20] Arsenic (As) and lead (Pb) are among the most commonly detected contaminants in drinking water today.[21] Regular consumption of these toxins could be linked to CVD risk and harmful structural heart changes, including left ventricular hypertrophy, defined as thickening of the heart's main pumping chamber (left ventricle).[22] These changes impose ongoing stress on the body, heightening susceptibility to CVD. A 2024 study found that over 2 billion people worldwide lack access to clean drinking water,[23] with 1 in 4 people exposed

to contaminated water (CW).[24] Water contamination may occur when pesticides and fertilizers applied to agricultural lands infiltrate groundwater through rainfall or snowmelt.[25] Approximately one million deaths are attributed to unsafe drinking water, lack of sanitation, and poor hygiene.[23] Access to safe drinking water remains unequal across regions, with the greatest burdens falling on low and middle-income countries, particularly in sub-Saharan Africa, where poor infrastructure, limited regulatory oversight, and climate-related stressors increase reliance on contaminated groundwater sources.[26] Similar patterns of unequal exposure are seen in the United States. The Strong Heart Study, which evaluated CVD risk factors among American Indian adults, found that they were frequently exposed to arsenic through drinking water in areas where groundwater has been contaminated.[22] However, the relationship between contaminated water and cardiovascular risk appears to be different among populations due to the type and concentration of contaminants, exposure duration, and regional water quality. Hence, findings from studies of specific populations, such as the Strong Heart Study, are unlikely to fully represent the cardiovascular effects of contaminated water in other communities. Addressing inequities in exposure to contaminated water requires investment in water infrastructure, regulatory enforcement, and environmental governance, especially in neglected regions, while acknowledging that individual mitigation, such as water quality testing, may also reduce exposure at the household level.[27]

### *Physical Activity and Cardiovascular Risk*

Constraints on physical activity (PA) operate as upstream conditions that interact with chronic stress and accumulated physiological burden.[28] PA is essential for cardiovascular well-being, yet opportunities to be physically active depend on social and environmental conditions. Regular PA offers substantial cardiovascular benefits, as moderate or vigorous activity can strengthen the heart muscle,[29] enabling the heart to pump blood more effectively throughout the body.[29] The Centers for Disease Control and Prevention (CDC) recommends that adults get 150 minutes of physical activity per week, which is equivalent to 30 minutes a day, five days a week.[30] Despite these established guidelines, only 65% of adults worldwide meet recommended activity levels, while 35% fall short.[31] Meanwhile, increased screen time has exacerbated this trend by encouraging sedentary lifestyles and obesity.[32] However, the ability to remain physically active is not merely based on individual effort. It varies based on an individual's environmental conditions, including access to parks and recreational facilities, work schedules, or income. Several studies have found that parks in low-income neighborhoods are used less than parks in higher-income neighborhoods, even though low-income groups have more leisure time.[33] Consequently, low-income communities, certain racial and ethnic minority groups, women, older adults, and individuals with disabilities are often unable to engage in routine exercise despite similar or greater health needs.[33] Limited access to physical activities may contribute to a sedentary state, defined by prolonged sitting or inactivity,[34] doubling the risk of developing or dying from CVD (most prominently CAD), compared to physically active people.[35] Because most studies rely on self-reported PA, it remains difficult to determine how PA patterns and underlying environmental and social conditions influence cardiovascular risk. Other factors, including income, pre-existing health conditions, occupation, and neighborhood amenities,

could also affect both an individual's ability to engage in PA and their cardiovascular health. Overall, evidence suggests that promoting PA at the population level depends on environments and policies that make active living safer and more attainable, while individual engagement may also minimize CVD risk. Improvements in public transit access, neighborhood safety initiatives, and land-use policies could enable better opportunities for PA at the population level.[36]

### *Sleep Duration and Cardiovascular Disease Risk*

Insufficient sleep can result from impediments linked to work schedules, housing conditions, and chronic stress, all of which are associated with cardiovascular vulnerability over time.[37] Optimal sleep duration is necessary for maintaining cardiovascular health. Sleep specialists recommend that adults get 7-9 hours of sleep each night.[38] Transitions to daylight saving time have been linked to short-term changes in myocardial infarction incidence.[39] Research has shown that during the first 3-4 days following the springtime shift in March, myocardial infarctions increase by 24% globally.[39]

Short sleep duration corresponds with multiple adverse health outcomes, including diabetes, hypertension, obesity, and CVD.[40] Contemporary work schedules, technology use, and daily demands have been linked to delayed sleep timing and insufficient sleep.[41] Sleep is therefore recognized as a social determinant of health, as shift work, racial discrimination, housing instability, and exposure to chronic psychosocial stress may impede opportunities for adequate sleep,[42] affecting specific communities. In fact, nearly 1 in 3 adults do not meet the recommended seven hours per night.[39] Persistent sleep deficiency alters cardiovascular function, straining the cardiovascular system over time.

### *Insomnia and Cardiovascular Risk*

Insomnia is the most prevalent sleep disorder[43] and is characterized by difficulty initiating or maintaining sleep.[43] Short-term insomnia may occur in response to elevated levels of stress or changes in the environment, typically lasting up to a few days or weeks.[44] The overall prevalence of insomnia is approximately 22%, with higher rates observed among women than men.[45] A substantial body of evidence links insomnia to hypertension, CAD, and HF.[46] The Nord-Trondelag Health Study followed more than 52,000 adults over 11 years to assess symptoms of insomnia. Depending on the specific insomnia symptom, participants exhibited a 27%-45% higher risk of acute MI.[46] One study reported that individuals with insomnia had a 45% higher risk of CVD,[47] and were more likely to have increased stress hormones. Importantly, the relationship between insomnia and CVD is also linked to factors such as age, stress level, lifestyle, and existing health conditions, which means that insomnia does not fully account for a CVD outcome.

Variation in sleep duration and insomnia prevalence is linked to inequities in cardiovascular risk.

Black and Hispanic adults in the U.S. who have lower SES levels experience higher rates of short sleep and insomnia; these patterns correlate with their work schedules, housing conditions, and chronic psychosocial stress. The same factors associated with higher rates of cardiovascular morbidity show how social and environmental conditions can influence sleep behaviors.[42] These trends suggest that policy interventions (i.e., work-hour protections, housing stability measures, and neighborhood noise regulations) may improve cardiovascular well-being, alongside individual-level practices,[42] such as reducing evening screen time, moderating meal sizes, and maintaining a cool, dark environment. Recommended sleep hours vary among life stages.[48] Table 3 presents the recommended hours of sleep for individuals at different life stages.

**Table 3. Age-Specific Recommended Sleep Duration Across the Life Course**

| Life Stage | Typical Recommended Daily Duration |
|---|---|
| Infancy (4–12 months) | Approximately 12–16 hours |
| Early childhood (1–5 years) | Approximately 10–14 hours |
| Middle childhood (6–12 years) | Approximately 9–12 hours |
| Adolescence (13–18 years) | Approximately 8–10 hours |
| Adulthood (≥18 years) | Approximately 7–8 hours |

Table 3 outlines the recommended sleep duration among different life stages. The ranges for each age group are based on national recommendations. The data were compiled by the author and synthesized from published national sleep guidelines.[48]

***Psychosocial Stress and Neuroendocrine Pathways in Cardiovascular Disease***

Psychological stress is an important pathway connecting structural circumstances with cumulative physiological strain, making it a key component of the allostatic load framework.[49] Psychological stress activates neuroendocrine pathways that directly alter cardiovascular function. When a threat is perceived, the hypothalamus initiates the body's alert system.[50] This signaling stimulates the adrenal glands to release stress hormones, particularly adrenaline and cortisol,[50] which increase heart rate and blood pressure.[51] Excessive release of these hormones can adversely affect the heart. Chronic stress exposure frequently follows broader social and economic inequalities. This is evident in communities facing systemic disadvantage, where economic hardship is associated with intensified stress-related biological wear and tear and greater vulnerability to CVD.[52]

Adrenaline enhances mental alertness and memory during acute stress.[53] However, prolonged

exposure has been implicated in endothelial dysfunction and myocardial injury. Excess sympathetic activation is also linked to plaque instability and acute coronary events.[54] Experimental evidence indicates that adrenaline is involved in plaque rupture, followed by the formation of a thrombus that could completely block blood flow and cause a heart attack.[53] Cortisol also influences the cardiovascular system, impacting glucose metabolism, lipid profiles, and blood pressure.[55] Disruption of sleep and insomnia may amplify these hormonal effects; a recent meta-analysis hypothesized that patients with insomnia have elevated cortisol levels during the day and night, particularly in the evening.[56]

Sustained hormonal activation could exacerbate cardiovascular dysfunction through increased levels of blood sugar, cholesterol, blood pressure, and triglycerides.[53,57] Chronic activation of hormonal pathways driven by cortisol and adrenaline, is not a universal experience. These problems are tied to environmental inequities: economic insecurity, housing instability, discrimination, and other systemic limitations associated with biological wear and tear. Research has found that chronic psychosocial stressors like racial discrimination are correlated with impaired hypothalamic (pituitary) adrenal axis and altered cortisol secretion.[58] Due to differences in individual stress responses, chronic stress and CVD risks remains difficult to determine because these outcomes also depend on socioeconomic conditions, lifestyle, and existing health conditions. Moreover, discrimination is a persistent stressor correlated with cumulative biological strain through repeated neuroendocrine activation, a process vital to the concept of allostatic load.[58] Stress is a pervasive factor that affects nearly all individuals. Although it is experienced at the individual level, many dominant stressors stemming from economic insecurity, unsafe working conditions, and racial discrimination often originate from structural and environmental settings that exceed personal control. Individual-level strategies, particularly regular physical activity, have been associated with lower stress levels and improved cardiovascular health.[59] Similar benefits are seen with social support networks (family, a close friend, or religious organizations). However, preventive measures alone do not address the structural conditions that generate chronic stress exposure. Successful stress reduction at the population level requires policies and interventions that address structural sources of stress, such as labor protections, community safety initiatives, and housing stability.[60] Integrating individual stress management with socioenvironmental change is crucial to reducing stress and improving cardiovascular health equity.

### *Dietary Factors and Cardiovascular Disease Risk*

Dietary habits are closely related to food systems and economic access to food, which interact over prolonged periods to create chronic metabolic and inflammatory strain. Diet is a major driver of cardiovascular risk at the population and individual level.[61] Projections estimated that between 2025 and 2050, there will be a 90% increase in CVD prevalence, with an expected 20.5 million CVD deaths in 2025 and 35.6 million CVD deaths in 2050.[62] Frequent intake of ultra-processed and high-fat, high-sugar, and high-salt (HFSS) foods is tied to premature CVD,[63] as these products contain high levels of sodium, saturated and trans fats, and additives such as artificial preservatives, flavor

enhancers, and colorants.[63] Many low-income neighborhoods have fewer affordable grocery options, more fast-food outlets, and greater marketing of HFSS products.[64] High densities of fast-food outlets and the targeted advertising of unhealthy products in these areas are associated with greater dietary risks and health inequities, as well as systemic inflammation, insulin resistance, and metabolic syndrome.[63] When sustained over time, these dietary exposures act as a chronic physiological stressor, raising cumulative metabolic and inflammatory burden (allostatic load) that increases cardiovascular risk.[65]

### *High-Fat, High-Sugar, and High-Sodium Dietary Exposures*

HFSS foods are products high in saturated and trans fats, added sugars, and sodium, commonly found in processed foods and sweetened beverages.[66] Overconsumption of HFSS foods is a risk factor for hypertension and deleterious cholesterol levels.[67] Diets high in trans and saturated fat may raise the risk of heart disease by amplifying low-density lipoprotein (LDL) cholesterol.[68] Therefore, elevated LDL cholesterol is responsible for atherosclerosis, stroke, CAD, and hypertension.[69] Sweetened beverages such as sodas, energy drinks, and sports drinks are by far the largest sources of added sugar in the American diet,[70] with high intake corresponding with insulin resistance and inflammation, thereby increasing cardiometabolic risk.[71] High sodium intake elevates the risk of hypertension, a major risk factor for stroke, heart disease, and impaired renal function.[72,73] Food insecurity and lack of access to affordable groceries are more prevalent in low-income and disadvantaged communities.[74] A review of nutrition and health disparities found that among low-income households eligible for SNAP, Black and multiracial households were more likely than White households to experience food insecurity, suggesting structural barriers to accessing benefits.[74] These disparities highlight that food insecurity is associated to social and environmental conditions, through neighborhood food availability, targeted marketing of dense foods, and socioeconomic constraints, as distinct from individual choice alone. Diet varies widely among individuals, and other factors such as income, culture, eating patterns, and pre-existing health conditions may affect cardiovascular outcome. Although individual dietary choices support cardiovascular health, efforts to reduce cardiovascular risk through nutrition may combine individual-level guidance with population-level strategies (strengthening nutrition assistance programs, regulating unhealthy food marketing, and expanding affordable heart-healthy food options) that improve food environments and decrease structural limitations to healthy eating,[75] primarily in communities experiencing persistent food insecurity.

### *Alcohol Use and Cardiovascular Outcomes*

Alcohol consumption is widespread and often normalized through cultural, economic, and commercial influences; however, intake exceeding the recommended amount poses serious cardiovascular outcomes.[76] Excessive alcohol intake is linked to higher blood fats (triglyceride) levels. Increased LDL triglyceride levels are associated with atherosclerotic cardiovascular disease, including myocardial infarction and ischemic stroke.[77] Other outcomes from overconsumption of

alcohol include hypertension, cardiomyopathy, cardiac arrhythmias, or death.[77] When sustained over years, alcohol exposure interacts with psychosocial and structural stressors in connection with cumulative cardiovascular strain, a characteristic of allostatic load.[78] Studies report that cardiovascular harm in relation to alcohol use varies with social and environmental conditions spanning from chronic stress, educational attainment, to intensive alcohol marketing. Educational inequities are evident among women with less than a high school education, who experience nearly three times the risk of alcohol-related mortality compared to women holding a bachelor's degree or higher (HR = 2.74). Similar patterns are observed in men, emphasizing that harm from alcohol consumption is concentrated among individuals with lower educational attainment.[79] Observational studies reported that light to moderate drinkers are at a lower risk for CVD, while heavy, excessive drinkers are at the highest risk.[80]

Since those findings came from observational studies, the apparent lower risk in light/moderate drinkers does not establish that alcohol itself is protective. Therefore, other possible factors could include diet, socioeconomic status, existing health conditions, and other lifestyle behaviors. Public health guidance suggests behavioral strategies such as setting limits, monitoring intake, and addressing triggers.[81] At the same time, population-level strategies, including taxation, advertising restrictions, improved access to care, and harm reduction, are necessary to resolve these inequities.[82] Alcohol-related cardiovascular harm accumulates alongside biological stress and unequal social conditions; understanding the importance of population-level policies may ease the CVD burden.

***Tobacco Use and Cardiovascular Outcomes***

Inhaling the toxic mix of chemicals in a cigarette interferes with normal physiological processes. Tobacco toxicants are absorbed into the bloodstream, where they impair cardiovascular function and damage the vascular endothelium.[83] Smoking is a leading risk factor for CVD, responsible for over 8 million deaths worldwide annually.[84] Smoking raises triglycerides, lowers HDL cholesterol, promotes atherosclerotic plaque formation, damages endothelial cells, and is a potential factor in the thickening and narrowing of blood vessels.[85] Atherosclerotic plaque formations restrict blood flow through arteries and other blood vessels, reducing blood flow to vital organs such as the heart and brain. These processes may result in thrombus formation, with other outcomes such as myocardial infarction, stroke, or death.[86] Regular exposure to tobacco smoke elevates inflammatory and vascular stress, while cumulative biological strain is associated with cardiovascular vulnerability. The cardiovascular effects of smoking emerge through prolonged biological stress and may be correlated with disparities in policy protection consistent with an allostatic load framework.[87] Smoking cessation interventions include behavioral counseling and pharmacologic therapies such as varenicline and bupropion.[85] These steps may mitigate cardiovascular risk at the individual level, while population-level reductions in tobacco harm could be achieved through tobacco taxation and restrictions on advertising. Tobacco-related cardiovascular harm is disproportionately experienced, with higher burdens concentrated in communities facing economic disadvantages and insufficient access to cessation resources. Workers in low-wage and service-sector occupations are significantly less likely to be

covered by comprehensive smokefree laws, primarily in states with local preemption of tobacco control policies. Furthermore, preemption limits local governments' ability to regulate tobacco retailer density, which is often highest in economically disadvantaged regions.[89] These disparities point to the importance of population-level interventions, through comprehensive smoke-free laws, regulations on retailer density, and equitable access to cessation resources, to decrease exposure and mitigate cardiovascular inequities.

## CONCLUSION

### *Limitations*

This review has several methodological limitations. As a narrative literature review, the study was designed to bring together evidence from several behavioral and environmental factors. Although studies were selected based on the inclusion and exclusion criteria described in the Methods, a standardized risk-of-bias assessment was not conducted. Publication bias may also affect the evidence available for this review, since studies finding significant associations are more likely to appear in published literature than studies reporting little or no association. The studies also differ in their populations, locations, methods of measuring exposure, and cardiovascular outcomes. Because of these differences, findings from one study or population may not always apply to another. Another limitation is that much of the evidence discussed in this review comes from observational studies. These studies can identify relationships between exposures and cardiovascular outcomes, but they cannot always determine whether one directly caused the other. Other conditions, including socioeconomic factors, health conditions, and environmental exposures, may also affect these relationships. For these reasons, these constraints may be considered when interpreting the findings.

### *Implications for Cardiovascular Disease and Health Equity*

Cardiovascular disease is a significant threat to global health, responsible for the deaths of millions annually. Although advances have been made in clinical care to enhance treatment options and improve the survival rates for those with CVD, effective strategies to reduce the burden of this disease include both early preventive measures and environmental and public policy-based interventions. Behavioral factors such as diet, exercise, smoking cessation, and stress management play a key role in cardiovascular prevention. However, they are influenced by the contexts and conditions in which people live and work, including housing insecurity, neighborhood pollution, targeted advertising, and structural obstacles to care, which may influence the distribution of these risk factors across populations. Preventing CVD at a population level requires upstream, equity-driven approaches. Implementation of equitable policies involves ensuring fair access to clean air and water, safe spaces for PA, healthy and affordable food, and culturally competent health services. Public health program initiatives must also prioritize communities that have been marginalized and continue to bear a disproportionate burden of cardiovascular risk. Highlighting the significance of equity-focused interventions and individual strategies is fundamental to reducing cardiovascular disparities and improving population health in a more just and lasting way. Future

research studies on this topic would be enhanced by collecting and using exposure data regarding behavioral and environmental exposures with unified frameworks, particularly allostatic load, to quantitatively examine the relationship of cumulative structural conditions and cardiovascular disease risk among diverse populations.

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