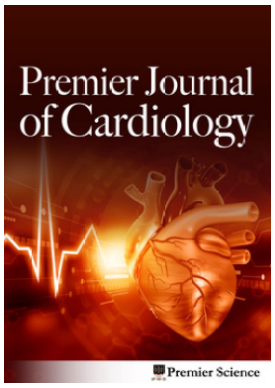

The Influence of Environmental Factors on Cardiovascular Disease Risk: A Comprehensive Review

Ambreen Ilyas, Khadija Batool and Hina Javed

ABSTRACT

Cardiovascular disease is a leading risk factor globally and has been proven to be fatal, with nearly 18 million deaths worldwide each year, accounting for one-third of the morbidity and mortality from all other causes. Cardiovascular disease (CVD) accounts for the highest proportion of non-communicable diseases worldwide. Alongside genetic factors, environmental factors are likely to be linked to cardiovascular disorders. To reduce the risk of CVD, environmental determinants for CVD are enormously studied in developed countries. This comprehensive review will integrate the prevalence, incidence, etiology, and pathology of these environmental factors with an emphasis on CVD. A consideration of how several factors of the environment, that is, natural, social, and personal domains, affect CVD and could cause a more acceptable assessment of CVD risk is required to help in the betterment of new control measurements to decrease the high weightage of cardiovascular diseases globally. Alleviative measures to lessen the effects of environmental factors will also be discussed in this review study. The review was conducted following PRISMA 2020 guidelines. Comprehensive searches were performed across databases including PubMed, Scopus, and Web of Science for articles published in English between 2000 and 2023. Search terms included "environmental stressors" AND "cardiovascular diseases." Two independent reviewers screened studies autonomously, and inclusion criteria encompassed peer-reviewed articles reporting environmental impacts on CVD outcomes. Data extraction focused on environmental factors and their association with CVD and suggested mitigation strategies. The evidence of CVD is strongly linked to aspects of the environment such as air contaminants, weather extremes, wildfires, sunlight, and noise pollution. Besides the conventional causes of CVD such as hypertension, elevated blood sugar, obesity, hypercholesterolemia, tobacco smoke, and inherited susceptibility, there are a lot of shreds of evidence showing the relatedness of CVD with physiochemical factors of the environment. Changes in geographical locations, lifestyle changes, and shifts in social adaptations can alter the severity of CVD possibility even without heredity factors. It is important to note here that these environmental stressors are a major cause, and they increase with the intensification of urbanization. Understanding the multifactorial impact of environmental determinants on CVD is critical for developing targeted interventions. This review underscores the need for integrating environmental considerations into global health strategies to reduce the burden of cardiovascular diseases.

Keywords: Cardiovascular disease, Environmental factors, Air pollution, Circadian rhythm, Vitamin D deficiency

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Introduction

Cardiovascular diseases, being a part of non-communicable diseases, are a leading cause of mortality globally with an accelerating ratio remarkably in the low- and middle-income countries (Table 1). Their associated morbidities also affect the socio-economic status of various populations.[1] Despite several advances in treatment and prevention, CVD is a persistent cause of the highest number of deaths each year. Deaths from heart diseases are rising significantly globally. In the United States, where the occurrence of the prevalence of CVDs was declining from its peak in 1960, since 1990,[2] its declining ratio has slowed down and 40% of the population will be affected by some type of CVD risk by 2030.[3]

The perception behind the prevalence of CVD is that CVD is caused by a set of severe disorders, arising due to a nexus of the interconnectedness of heredity susceptibility and environmental stressors that may lead to the gradual worsening of the structure and function of the blood circulatory system and organs. It is a common perception that although CVD is a result of chromosomal abnormalities with some unusual types of heart disorders, major cardiac abnormalities are a result of the linking of some different genes and ecological variants. The connectedness of genetic makeup and environmental factors is not fully understood so far; however, it is considered that lifestyle choices and genes contribute to a collective character in prevalence of the CVD arousal. This thought may be a result of multiple factors based on several researches describing a certain extent CVD is controlled by adopting fine life habits. Likewise, facts from the previous study[4] show that 82% of CVD danger can be minimized by adopting a sound lifestyle.

The alterable nature of CVD is more clearly understood by the data from different research depicting that only environmental alterations can cause cardiovascular risk in a community when genetic factors are absent. This is most effectively described by data from a study in China, showing that age-standardized

Table 1 | Distribution of communicable and non-communicable diseases worldwide

Types of Diseases	Percentages
Non-communicable diseases	52%
Communicable diseases	34%
Injuries and trauma	14%

mortality rates by CVD accelerated by 27% for men and 50% for women in Beijing, due to environmental alterations that occurred from 1984 to 1999.[5]

The death rate from coronary heart disease (CHD) in England has decreased by 45% in women and 62% in men in the past years. Approximately half of this decrease was characterized by low levels of environmental risk factors.[6]

Additional evidence about the changing nature of CVD is strengthened by the facts from migrants' study, which showed that migration to a new location could consequentially alter the CVD risk. A significant rise in the ratios of CHD mortality rates in the male population of Japan was observed in 1960 and 1970; who migrated from Japan to the United States.[7] Likewise, people coming from India and living in the United Kingdom[8] have more chances of CVD danger as compared to their peers residing in India. These facts support the concept of the epidemiology of CVD risk factors due to environmental variations even if genetic changes have not occurred.

CVD has a complex etiology, exhibiting multiple factors, including environmental factors, that is, natural, social, and personal domains of the environment.

Objectives

This review explicitly seeks to:

1. Summarize the evidence on the prevalence, incidence, and etiology of CVD concerning environmental factors.
2. Explore the interaction between genetic predisposition and environmental stressors in influencing CVD risk.
3. Assess the impact of environmental changes, including geographic location and urbanization, on CVD outcomes.
4. Identify potential strategies to mitigate the adverse effects of environmental determinants on CVD (Figure 1).

Methods

This systematic review was conducted following the PRISMA 2020 guidelines.[9] The review has been registered with the Research Registry (UIN: Reviewregistry-1934). The study analyzed peer-reviewed articles published in English between 2019 and 2023. A comprehensive search was conducted in databases including PubMed, Scopus, and Web of Science using the keywords: "environmental stressors" AND "cardiovascular diseases." Inclusion criteria were studies focusing on environmental factors influencing cardiovascular diseases, while exclusion criteria included non-peer-reviewed articles and studies not related to human subjects. The study complies with AMSTAR 2 guidelines,[10] achieving high compliance.

Eligibility Criteria (5):
Included peer-reviewed studies (2000–2023) in English on environmental factors influencing CVD in humans. Excluded non-peer-reviewed articles, unrelated articles, and animal studies. Studies grouped by environmental factors (natural, social, personal) affecting CVD.

Information Sources (6):
Databases searched: PubMed, Google Scholar, Scopus, Web of Science, and reference lists. Search last updated on 23-12-2024.

Search Strategy (7):
Combined controlled vocabulary and keywords like "environmental stressors" AND "cardiovascular diseases," refined with Boolean operators and filters for articles from 2000 to 2023.

Selection Process (8):
Two independent reviewers screened titles/abstracts; full-text eligibility assessed. Disagreements resolved by discussion or third reviewer.

Data Collection Process (9):
Two independent reviewers extracted data using standardized forms. Authors communicated for unclear data if needed.

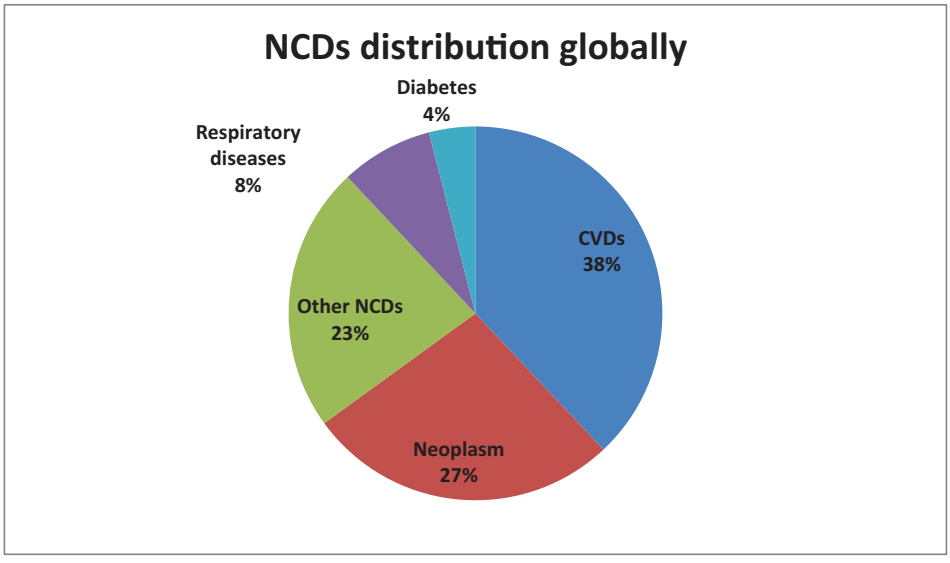

Fig 1 | Percentage distribution of NCDs worldwide

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Data Items (10):
10a. Outcomes: CVD risk and environmental impacts.
10b. Other variables: demographics, location, funding, and assumptions for missing data noted.

Study Risk-of-Bias Assessment (11):
Two independent reviewers; conflicts resolved by consensus.

Effect Measures (12):
Risk ratios, odds ratios, mean differences used.

Synthesis Methods (13):
13a. Grouped studies by environmental factors and outcomes.
13b. Prepared data by handling missing outcomes.
13c. Results displayed using figures and tables.

Reporting Bias Assessment (14):
Assessed using previous studies.

Certainty Assessment (15):
Previously published articles used, considering risk of bias, consistency, directness, precision, and publication bias.

CVD and the Human Environment

It is considered that CVD is controllable, and if it is gradually affected by environmental factors, then we have to understand how it works. How do different environmental forces affect the CVD to accelerate it or to lessen its underlying effects? Which factor of the environment affects the CVD and its causes mostly? What are its underlying risk factors and how this threat is passed on? Why do the environments affect CVD and its risk factors? To understand this, we have to consider the chemistry of the environment completely. All animals live in natural, abrupt, and open environments while human creates their own artificial micro-environments. They live in different geographical environments and they have evolved during the journey of their advancement. Therefore, to configure and evaluate the complexity of the environment, it is essential to integrate the natural, social, and personal realms of the human environment, collectively making up the human environment. The most common factor of the environment is its natural environment, including the circadian cycle, seasons, geographical changes due to natural phenomena, and evolution. In addition, due to increased acculturation, social environments are created in human environments. These social domains have become the basic areas of human activities. These environments are influenced by the pathogenic effects of human-created activities and pollute the environment. Moreover, in these social and natural environments, humans, with their own thinking abilities, make personal ecosystems that they may pollute by their own activities. Being close to a ductile environment, the personal domain plays a playful role in the health of people. Nonetheless, as reviewed here, all; personal, social, and natural realms (Figure 2) individually or collectively affect the CVD and its risk factors.

Natural Environment

Nature is the primary realm of the natural environment. Humans have changed throughout time by adjusting to the natural environment, along with all other living things. With the increasing effects of the social environment, the natural environment also poses a remarkable effect on the health of the people. The high threat of CVD could be due to the mismatch status between old human genetic makeup and recent human environments. This difference could be due to

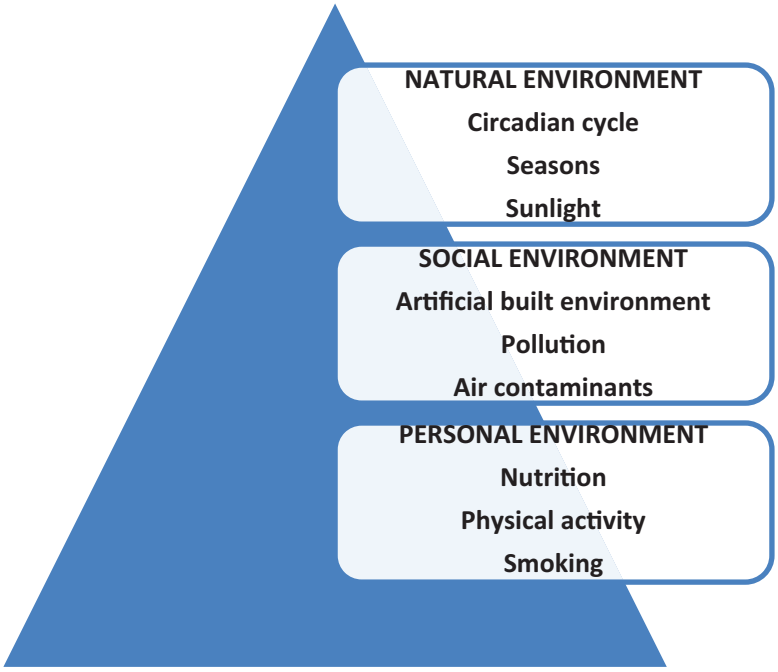

Fig 2 | Domains of the environment affecting the risk of CVD

the reason that environmental changes have outpaced genetic changes. Ancient alleles adjusted to old environments have maladjusted to current environments, thus, imparting disease risk. Principal attributes of the natural domain that may be associated with CVD are described as follows:

Circadian Rhythm

The circadian rhythm is the basic and constant factor of the natural environment. This circadian cycle regulates cardiovascular health and functioning; the rate of blood flow, that is, the blood flow rate, is slowest at night and while sleeping and increases just before getting up. Circadian hints also change the cardiovascular genetic makeup and the frequency of cardiovascular proteins,[11,12] likewise the production of hormones that change the cardiovascular function that is, angiotensin II, growth hormones, rennin, and some peptides.[13] By this strict regulation, it is considered that the occurrence of CVD events varies with the day. The occurrence of random CVD episodes is visualized to be connected with the circadian clock mechanism, but not to the pressure of getting up, as according to studies, when in a novel topographic location, the events of CVD in a traveler peak for some days, concerning the pattern that relates to their original time zone.[14]

Besides the increasing levels of CVD risk, the circadian cycle also affects CVD intensity. Cardiovascular strokes that occur at midnight are more severe,[15] and angiography or stenting done at night are less successful.[16] In fact, the usual disturbance in the sleep-wake cycle can cause obesity, diabetes mellitus,[17,18] risk of CVD, high blood pressure,[19] and hormonal disturbances. Even short-term circadian cycles can increase blood pressure, inflammations,[20] and blood glucose and insulin levels.[21]

Though it is unclear why the circadian cycle regulates CVD risk factors, it is documented that some basic cardiovascular benefits can be obtained by stabilizing the diurnal rhythms and, thereby, treating sleep orders, controlling blood pressure, and resetting hormonal profiles.[22] Collectively, the studies on the circadian cycle emphasize the fact that both human body shape and disease vulnerability are connected to the natural domain of the environment and its primeval patterns.

Seasons

Variations in seasons can also take part in the making of natural environments. In many parts of the world, changes in seasons can largely contribute to variations in humidity, temperature, and the duration of the day. A regulation in season can alter sunlight exposure, feeding behavior, and physical activity, and alterations in the anatomical activity and digestion mechanisms can affect overall human health and CVD function. In different geographical locations, the number of low-density lipoproteins (LDL) and high-density lipoproteins (HDL) and sugar levels in the blood moderately rise in winter and fall in summer and most individuals on their lipid-lowering treatment get access to their earmarked LDL levels in summer[23] than in cold weather, indicating that lipoproteins in human plasma can be altered by the change in seasons.[24]

Seasonal differences in CVD-causing risks are linked to changes in cardiac deaths; those occur basically more in cold than in summer. Cold outdoor temperature is itself an important factor that raises vascular aversion and hypertension, leading to a rise in oxygen requirement.[25,26] Anyhow, low temperatures are not the only factor causing the mortality as it is documented that mortality cases are even higher in the areas where the seasonal temperature does not fluctuate at larger degrees, for example, Los Angeles.[27] Although considering the role of temperature and seasonal changes in causing CVD-risk mortality is not fully understood and there is a lot more to study and research, recent data supports the fact that seasonal changes and temperature fluctuation induce a strong impact on CVD threat arousal and deaths.

Low temperatures are more often connected to long-term cardiac episodes, meanwhile, highly warm climates impart equal contribution. Heat waves of high intensity in various parts of the globe are linked to CVD corporality, especially in weak people including older people who are not strong enough to adopt rapid changes in heat.[28] The temperature of planet earth is much greater than it used to be in the previous 100,000 years. This fluctuation in temperature seems to influence the seasonal and climatic conditions globally, and this change directly affects the crop production as well as the socio-economic conditions that directly or indirectly affect the CVD and its underlying risk factors, specifically among resource-lacking poor and vulnerable populations.

Sunlight

It is particularly documented that low wavelengths of sunrays accessing the earth in cold could accelerate the CVD sensitivity. Could sunshine actually influence CVD status? Some research shows that large exposures to sunrays in early life decrease the CVD risk by 0.6 to 2.1 years.[29,30] Staying outdoors preferably in sunlight is oppositely related to CVD mortality.[31] Remarkably, further studies showed that the CVD-causing deaths due to insufficiency of sunrays were equal to that of the use of tobacco. In a recent analysis of 200 districts of the United Kingdom, it was observed that the time of sunshine was inversely linked to the rate of CVD mortality.[32]

Exposure to sunlight could affect CVD and its underlying risk factors due to many aspects, but the well-documented understanding links to vitamin D, which can only be manufactured in the occurrence of sunrays. The proficiency of this technique is however connected to the quantity of packets of photons that are embedded in the inner surface of the epidermis which directly depends on the melanin presence in the skin.[33] Hence, people with brown skin require high quantities of photons to manufacture an equal quantity of vitamin D as compared to the individuals with lighter skin. This is because UVB radiations are crucial

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for the synthesis of vitamin D reaching the earth from the sun. Therefore, the efficacy of the synthesis of vitamin D relies upon the amount of UVB accessing the earth. When the sunshine is lowest in the sky that is, during cold, sunsets, or sunrises, the amount of UVB reaching the earth becomes more scattered due to low intensity as compared to the time when the sun is straightly at its peak (during summer or at noon). As a result, the production of vitamin D is influenced by the time of the day and weather. Consequently, larger vitamin D insufficiency is observed in autumn and winter than in spring and hot weather.[34]

Climatic alterations in the quantity of vitamin D are linked to geographical and climatic changes in blood flow. Blood flow is higher in winter, causing hypertension.[35,36] When UVB rays reaching the earth's surface are low and become lower in winters when UVB levels are higher on sunny days. Besides the blood flow, vitamin D also fluctuates in other cardiac activities. All cardiac tissues exhibit receptors (Figure 3) for vitamin D, affecting the makeup of approximately 200 genes. Collectively, 3% of genes of total human genomic makeup are altered by vitamin D.[37]

Even though the links between the sunrays and vitamin D are well studied, it is still feasible that levels of vitamin D in the human body indicate common well-being and there could be more unknown aspects that link the sunrays to cardiovascular well-being.

Social Environment

Like all living beings, humans create small communities. The settlement into micro-environments can benefit the community in many ways: it creates a network of social interaction, cooperation, and support. Humans make small environments to help align with the severity of the natural environment, the environmental stressors of the natural world, from the danger of natural predators and pests. Man-made domains of the environment make other issues like overpopulation, noise pollution, and air contamination, attributes of the environment that ultimately limit the quality of health of individuals. In addition, the need to build small environments by humans necessitate the requirement of development of a complex social stratum to maintain the division of labor. As discussed below, extensive studies documented the influence of socially built environment and pollution on CVD and cardiovascular health risks.

Artificial Built Environment

By changing the local environment, and by crafting the artificial built environment, non-natural biological systems can either increase or decrease the ratio of disease. It could protect the environment by creating sanitized, climate-protected spaces, but it also promotes diseases by creating unfavorable conditions. Socio-environmental conditions can contribute to CVD mortality, and the risk is associated with the attributes of different geographical conditions of the areas, social unity, and accusation.[38]

However, approximately 40-plus published articles report that adopting socially abandoned areas higher the CVD risk.[39] The real risk factors underlying this are still unknown. In unfavorable neighborhoods, the accessibility and price of different varieties of food, the handiness of cigarettes, the settlement of parks and playgrounds, and other contrasts in the built environment are all contributors to CVD risk cases. Moreover, other sources of facilitation including

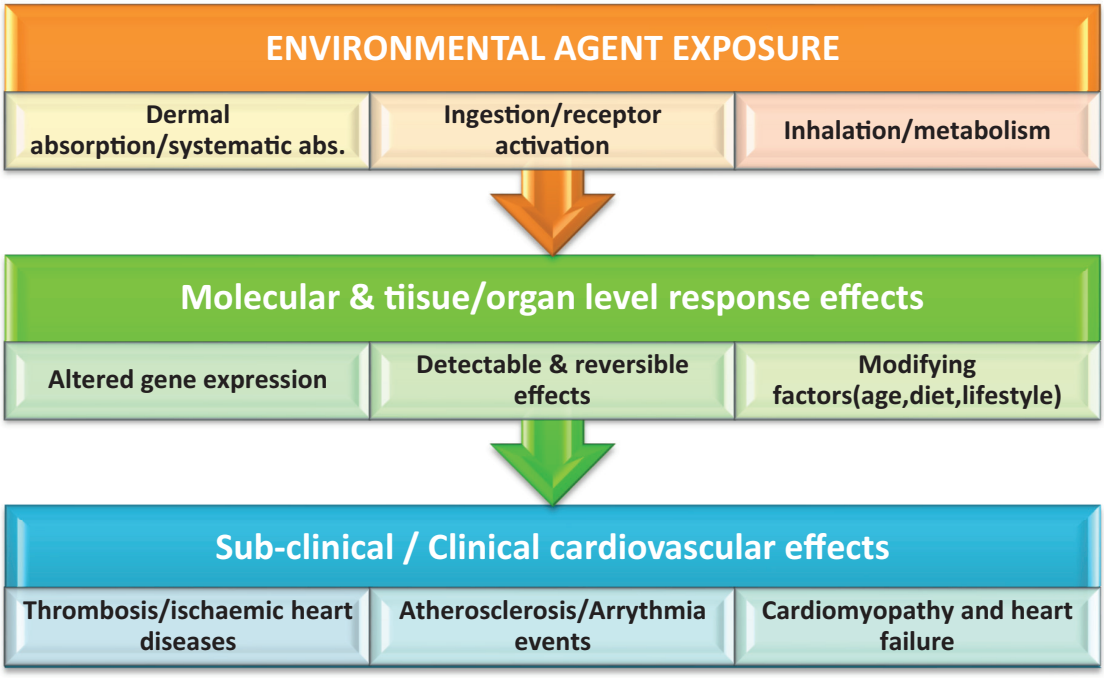

Fig 3 | Characteristics of the impacts of environmental influencers in cardiovascular disease

transportation, health care services, and social interactions might also be crucial.

The most obvious influence of the built environment may be observed in obesity. Obesity and adolescence are major concerns regarding CVD; on the other hand, children with normal weight rarely have cardio-metabolic health-related issues.[40] Environmental factors are worth considering in this regard. Besides the genetic factors involved in CVD, parental exposure to environmental contaminants may affect the growth factors of children and the development of CVD and their risk factors. A systematic review of almost more than 60 research studies depicts that features of the built environment are directly related to obesity,[41] particularly in the older populations.[42] Highly documented pieces of evidence were studied for good-quality diet, best places to visit, recreational parks, and highly sanitized health care units. Each of these attributes is positively correlated with body mass index. Most calculations of obesity may also be linked to the large frequency of junk food restaurants.[43] Every day, each hour spent in a car can increase the risk of obesity by 6%. Each kilometer walk per day can decrease the risk of obesity by 55%. These impacts of obesity conciliate on the built environment of CVD threats; almost 50% of the indirect connection between neighborhood guidance and blood flow may be evaluated by contrasts in measures of the body-mass-to-weight ratio.[44] Aspects linked to the other 50% of the connectedness are not known at the time. Collectively, CVD risks are likely to cluster in unfavorable territories due to several means of susceptibility, which may link to the attributes of the built environment, anxiety, tensions, insufficiency of the space to exercise, and exposure to multiple environmental contaminants.

Pollution

The current air is flushed with man-made toxic compounds and contaminants. By a rough estimate, more than 30,000 pollutants are currently in implication. Out of these, almost 5,500 contaminants are produced in 100 tons each year.[45] Almost all the basic rivers and other freshwater resources are highly contaminated with all types of pollutants that is, heavy metals, synthetic chemicals, and pesticides. Although the levels of air pollutants have decreased up to a remarkable level in developed countries than during its peak in the 1950s to 1960s, in underdeveloped countries, they still remain up. A high amount of air contamination is made up of a complex combination of aerosol particulates and gases. Particulate air contamination consists of particulate matter consisting of coarse particles and fine particles when differentiated according to size. Aerosol particulate matter released in the air is comprised of soot, minerals, pollens, and spores whereas organic compounds make the secondary aerosols (Table 2).

The World Health Organization (WHO) calculated that contamination in the air may be associated with about 7 million preterm deaths per year globally. In its health influences, air contaminants directly affect high blood pressure, tobacco smoking, and physical sedentary.[46] Prone to air pollutants is prevalent and unadjustable. In domains of the globe, about 95% of the total humans live in urban areas where the threshold quantities of air contamination exceed the air quality index (AQI) of WHO limits.[47] The effect of air contamination on cardiac health may be dependent upon individual sensitivity. Individuals with CVD, high blood sugar, or people who use tobacco could be more sensitive to the harmful impacts of pollution.

Besides the individual sensitivity factors, sensitivity to open-air contamination is also mediated by different environmental aspects including noise pollution, tobacco smoke, or other air contaminations.

Air Contaminants

Exposure to air contaminants may accelerate the rate of danger of heart disease.[48–50] In a similar way, prone to air contaminants has long-lasting effects on heart structure and function, that is, thickening of heart walls from early childhood.[51–55]

A review of a recent study illustrated that open-air particulate matter reaches the circulation system through the liver.[56] These floating particles then become a part of the Kupffer units of liver cells and these contaminated Kupffer cells then secrete the cytokines that cause the inflammation and production of perisinusoidal cells.

Another visual of the impacts of the environmental factors is the production of a collection of diseases that may lead to CVD and diabetes in adulthood, which can be caused by these factors, including air contaminants, premature birth, and low birth weight,[57–58] which as a result can be linked to the danger of chronic diseases such as CVD in later life.

A large number of recent studies enforce the role of lack of vitamin D and vitamin B12 on CVD and diabetes, likewise its other risk factors like insulin resistance, high blood pressure, and obesity. By keeping in mind the reciprocal link of the AQI to a derivative of vitamin D in expected women, babies,[59] and young

Table 2 | Potential CVD health effects of addressing neighborhood environment

Level of Impact	Study Goals	Potential Outcomes
Population	Integrate the health effects of social policies	Reduced structural inequities
Health system	Evaluate neighborhood measures into health systems	Risk mediation at points of care
Community	Manipulate the built environment	Asset-based initiatives to enhance cardiovascular health
Individuals	Explicate multilevel drivers including upstream and downstream pathways	Healthful behavioral changes

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children,[60] it can be proposed that one of the criteria of the dangerous effects of contaminated air impacts on the origins of CVD may be cured by treating vitamin D deficiency.

All the above-cited points indicate that being prone to air pollutants for a very long time may cause the increased risk of development of CVD and its derivatives.

Indirect tobacco smoke may cause serious health effects for different age classes, particularly for children. Exposure to cigarettes and other tobacco-containing pipes exerts adverse effects on people's health besides its foremost effect on pregnant ladies. One of the major deadly effects of tobacco smoke is that it is instrumental in CVD's acceleration.[61–69] Numerous methods are connected in this regard. Indirect smoking is linked to swelling and hardening of the arteries.[70–73] These findings may help to aid in the treatment of CVD to protect children from all forms of heart and circulatory diseases.

According to an assessment, climate change accounts for more than 88% of CVD, and its underlying risk factors mostly affect youngsters aged less than 5 years.[74] Various contraptions are studied for the health effects of climate change. These effects may be direct or indirect; through environmental (heat or cold) stressors or through effects on the natural ecosystem. Exposure of pregnant ladies to severe heat may make them to be prone to toxemia of pregnancy, premature birth with low birth weight.[75] This is due to the fact that fetal age and weight of the newborn affect CVD and its risk factors later in life.

Depending upon climate changes, it is considered that greenhouse gases may contribute to increased temperatures, which as a result may promote extreme environmental phenomena such as drought and storms, and particularly affect children and pregnant ladies. Climate change can affect the pollutant concentrations globally and affect their intensity as well, which can harm the health of almost all age groups.[76–78] The World Health Organization integrated the cause-specific death cases exhibited by the environment per year in the whole world, depicting CVD as the foremost leading cause of mortality (Table 3).

Personal Environment

The personal domain of the environment is crafted by an individual's life choices and needs. The most conclusive theory of upgrading the character of the personal environment results from research, documenting the straightforward link between lifestyle habits and CVD risk. The impact of the environment starts with the beginning episodes of cardiovascular disease growth. Various pieces of research indicate that the ambient environment depicts the CVD vulnerability in adult life. Preterm birth, which is a sign of unhealthy uterine ambience, shows an inverse relation with CHD. These diseases, specifically hypertension and blood sugar levels, previously considered to happen only in later life, now occur at a young age as well. It is now assumed that the present generation of children may exhibit short life span than the children of the past due to higher occurrence of blood sugars and cardiac issues.

Nutrition

Personal diet preferences are significant factors in CVD harm. However, the diet selection is not random; it is described in parts by traditional and economic status. Methods of making food are different to each particular region and exhibit a constant uniqueness that transfers to next communities. Nutrition choices are confined by the social domain, availability, and distribution states of the diet. Hence, contrasts in dieting behavior and differentiation of coronary cardiac diseases are somehow linked to traditionally inherited or economically confined nutrition patterns.

Even though diet is commonly considered to play the role of a primary variant of health, its influences on CVD are still not fully understood. Several studies have reported the remarkable CVD impacts on eating habits. The relevant researches reflect that mere regulations in diet can change the CVD and its harmful effects. In the previous study, it was evident that shifting from saturated fat to unsaturated fat could lessen the effects of CVD by 45%.[79] It clearly indicates that diet intake can alter the CVD risk. This concept was strengthened by the data from saturated fats. In a quantitative study integration of 4 types of research, saturated fats may be linked to a 2% rise in energy expenditure and were seen to be linked to a 23% gradation in CVD occurrence. A diet enriched with trans fatty acids can increase the CVD risk by 3% and cause sudden death.[80] Whereas, the population who eats fresh fruits, vegetables, and well-judged food may halve the risk of CVD than those who take processed foods.

Eating habits influence all the cardiovascular risk causes. Nutrition full of saturated fats and high salt intake can increase cholesterol levels and the risk of hypertension. Trans fat increases the LDL concentration but decreases HDL, thus promoting atherogenesis.

Table 3 | Calculations from the world health organization depicting cause-specific mortality cases governed by the environment per year globally

Rank	Cause	Total Estimated Numbers
1st	Cardiovascular diseases	4.8 million
2nd	Cancer	1.7 million
3rd	Unintentional injuries	1.69 million
4th	Severe respiratory issues	1.4 million
5th	Diarrheal diseases	846,000

The cell- and tissue-level mechanisms by which eating habits can influence CVD and its risk causes are still not fully studied.

Dietary habits are gained from childhood. In addition to providing healthy dietary requirements, dietary routine is essential for tracking and understanding CVD and its underlying mechanisms from early life to adulthood. The most vital dietary habits that make the development and growth of CVD are related to high fatty acid food, high-calorie intake, and saturated fatty acids. Following the dietary guidelines and nutrition rich in vitamins and minerals is essential to supplement the required criteria.[81–88]

Physical Activity

Workout habits are a significant feature of healthy living. Due to various causes, physical inactivity in humans of the current era has led to increased risks of CVD. It accelerates the risk of severe heart issues by 45%, high blood pressure by 30%, stroke by 60%, and diabetes by 50%, and it is significant to add here that 13% of all preterm births in the United States are because of physical inactivity.[89] On the other hand, routine exercise lessens the CVD risk.[90] These effects are dose-specific; moderate activity decreases the risk by 26% of CVD whereas a high intensity of physical activity decreases the risk of CVD by 42%. Moderate- to high-intensity physical activities increase the rate of life span by 1.3 to 3.7 years and physically active persons remain CVD-free for 1 to 3 years as compared to their other counterparts. Several mechanics highlight the significance of exercise. Hard physical activity can accelerate the oxygen supply to heart tissues and result in the better working of heart-muscle contractions and relaxations. Moreover, exercise increases HDL (good cholesterol) levels while lessening LDL concentrations, while inactivity can cause hypertension, stroke, and insulin resistance.

The advantages of regular physical workout routines are very well studied and documented for all age groups. Attaining an active lifestyle by reducing sedentary chores remarkably by reducing screen time and increasing physical activities can provide good CVD health. Additionally, controlling the excessively increasing body weight can prevent and cure CVD and its underlying risk factors.[91–97]

A profound consideration of the impacts of workout habits is needed to consider novel techniques and the betterment of cardiac health and sportsmen activities. It also prevents and controls the environmental causes that affect workout habits. Stress and anxiety are examples of such supportive factors for physical inactivity. People who undergo depression are highly inactive, because the relationship between depression and inactivity is highly correlated, and such individuals undergo CVD risks very often.[98] Consequently, an active lifestyle is not just a choice but a social determinant of cardiac health.

Smoking

Smoking is one of the utmost dangerous choices an individual can make among all other personal choices. Smokers can die 13 to 14 years before nonsmokers. In the United States, the use of tobacco is responsible for 443,000 preterm mortalities annually.[99] Besides, 16% American population carries on smoking each day, and nearly 2,100 young children turn out to be smokers every day. Globally, about 20% of people use tobacco, about 5 trillion cigarettes are made annually, and almost 15 billion cigarettes are sold per day. In developed countries, almost 40% to 50% of people have cut off smoking in the 1960s, and globally 1 billion people have carried out smoking.[100]

Even though tobacco smoke accelerates the danger of lung cancer and other respiration-related issues, almost 50% of premature deaths related to smoking are a cause of CVD and its risk factors. Smoking has also become a big cause of jerky heartbeat, stroke, and sudden cardiac failure.[101] The large susceptibility of smokers to cardiovascular risks highlights the high vulnerability of cardiac tissues to contaminants. Research on air contaminants, indirect smoke pollution, and tobacco smoke has persistently explained cardiac tissue rupture at levels and duration of exposure much shorter than those linked to any respiratory problem. According to research, WHO estimated that CVD is the fundamental reason for mortality due to airborne contaminants, and the amount of CVD mortalities surpass those factorized by other respiratory issues and cancer.

The use of tobacco, that is, cigarettes, pipes, or any other forms, can be the cause of CVD. Youngsters most commonly adopt the habit of smoking and carry it up to old age. Likewise, in all other age groups, the use of tobacco has the most severe effects on the health of people, leading to its contribution to heart-related abnormalities and disorders.[102]

Even though the actual mechanism for the CVD risk by smoking is not yet studied enough, it is clear that it affects cardiovascular health independently of other aspects. Quantitative integration of 54 various studies emphasized that smoking increases LDL concentration and decreases HDL, while changes in the concentration of lipids remain less than 1/10 of the increased CVD threats for smokers. Likewise, although smoking strongly affects blood pressure, smokers usually exhibit lower blood pressure.[103] The regulation of insulin is also uncertain because of the effects of smoking. Several studies explain that insulin resistance rises by smoking while some other studies show no clear link between smoking and diabetes mellitus.[101] Nonetheless, it is a common consideration that the smoke produced by burning tobacco contains toxic organic compounds, that contribute to the toxic impacts of smoking. Based on this consideration, novel techniques have been formulated that produce nicotine in the form of a spray of some organic molecules. Some researchers thought that these devices, such as e-cigarettes, exhibit lower toxicity than tobacco-burning cigarettes.[104] Due to this consideration, the implication of e-cigarettes is becoming popular. In America, about 9 million individuals used e-cigarettes in 2014. Nevertheless, the safety status of e-cigarettes is not known.[104] Regardless of the impacts of e-cigarettes and how these devices may

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affect cardiac health hereafter, the widespread use of e-cigarettes is a fundamental matter of study of how the toxic factors and controlling policies affect CVD risk and influence overall cardiac health.

Next-Generation Environmental Technologies and Innovative Mitigations

Cutting-edge techniques with the use of AI can mitigate the environmental datasets to anticipate CVD risks. With the use of AI models, one can predict the link between real-time data to predict the highly polluted air zones and their effects on CVD exposures. Likewise, urban planning strategies involving the abundance of green areas to help reduce pollution should be taken into account. The factors involving individual behavior and social conditions are linked to environmental factors and may be communicated through coherent policies. All the data discussed above highlights the link between environmental contaminants and CVD. This emphasizes the role of better AQI and an active campaign to educate the communities for better public health. In low-resource setups, cost-effective measures, such as tree planting by the public and public health education, can provide a fruitful outcome at a low cost.

The findings of this study are globally relevant but must be adjusted to each specific domain. In urban domains with high pollution index, strategies based on pollution control may be preferred, while countryside areas can get easy access to clean water. Mitigations may also account for cultural alterations. Educating the people through campaigns should be in their native languages to obtain maximum output.

Results

Study Selection (16a)

500 records identified; 50 duplicates removed; 450 screened; 300 assessed for eligibility; 103 included.

Study Exclusions (16b)

Excluded studies: Study A (insufficient sample size), Study B (did not assess primary outcome), Study C (methodological flaws).

Study Characteristics (17)

Key details: Study design, population (age, health), intervention (treatment or exposure), outcomes, sample size.

Risk of Bias (18)

Assessed using Cochrane risk-of-bias tool; categorized as low, high, or unclear risk for each study.

Results of Individual Studies (19)

Summary statistics and effect estimates (e.g., mean difference, odds ratio) with 95% CI in tables and plots.

Results of Syntheses

Study Characteristics for Synthesis (20a)

Brief summary of contributing studies' designs, sample sizes, risk of bias, key findings.

Heterogeneity Analysis (20b)

Subgroup analyses and meta-regression revealed age as a potential moderating factor.

Sensitivity Analyses (20c)

Robustness tested by excluding high-risk studies; results consistent despite missing data.

Reporting Biases

Risk of Bias Due to Missing Results (21)

No significant publication bias detected.

Certainty of Evidence

Assessment of Certainty (22)

Moderate certainty for most outcomes using the GRADE approach.

Discussion

Interpretation (23a)

Intervention significantly improves outcomes, consistent with previous studies, but further high-quality studies are needed.

Limitations of Evidence (23b)

Limitations: small sample sizes, high risk of bias, lack of standardized outcomes.

Limitations of Review Process (23c)

Missed studies due to language/access restrictions, heterogeneity challenges.

Implications (23d)

Intervention is effective; further research needed; cautious policy implications.

Other Information

Amendments (24)

No amendments made during the review.

Sources of Financial or Non-financial Support (25)

Financial Support: Self-funded.

Non-financial Support: Provided access to proprietary data by the University of the Punjab, Lahore, Pakistan.

Competing Interests (26)

No Competing Interests: "The authors declare no competing interests."

Availability of Data, Code, and Other Materials (27)

Data Collection Forms: Available at Google Scholar, PubMed, NIH repository

Perspective

In conclusion, the documentation reported here strongly evidences the hypothesis that CVD is an environmental abnormality. This issue finds its roots in living in unfavorable environments and experiencing it, in parts by occurring in a form of asynchrony with the beats of the natural domain of the environment and a contradiction between our ancestors' genome and our current environmental domains. This consideration gives a novel consideration on CVD control and prevention by which we may be able to control the disease by checking the environmental factors. A coherent environmental state of CVD also highlights the significance of its control. As mentioned earlier,

CVD is avoidable, and since the 1960s, its stopping measures have been the actual cause of the decline in CVD mortality. Its preventive measures may become more adaptive if these are not expensive enough to bear among underdeveloped countries. Anyhow, if the major part of CVD is accredited to the environmental domains, large extensive benefits, in contrast, the issue could be acknowledged by the combined steps, that is, reforming urban areas, decreasing contamination, a better approach to a nutritious diet, besides earmarking personal choices and lifestyle habits only. Besides the principal character of the environment, extraordinary endeavors should be accessed to gain personally the effects of natural, social, and personal realms of the environment and to consider the mechanism by which these realms mitigate the impact of other realms and how this connection influences CVD overall. Here is the provocation for the next researchers to painstakingly unravel the threads of environmental factors and also mitigate the influence of different elements of the environment into an overarching stereotype. Such a replica may integrate how the effect of the natural domain of the environment may influence the social and personal environments and how these effects may limit or regulate the natural environment. More additional research studies are still to be done in this regard, but even a gradational advance in our study on the environmental basis of CVD may assist in implementing more precise preventive measures, which could remarkably slow down the unpromising increase and outspreading of CVD in modern communities.

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