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REVIEW
Air pollution and environmental science

Climate change and cardiovascular risk factors management: emerging challenges and strategies for prevention and adaptation

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Abstract

Climate change represents an escalating global health crisis that profoundly influences the risk factors for cardiovascular disease (CVD). Human-driven alterations in climate—including rising ambient temperatures, more frequent and severe heatwaves, air pollution, and extreme weather events—directly and indirectly exacerbate hypertension, diabetes, hyperlipidaemia, and physical inactivity. Exposure to high temperatures and pollution promotes vascular dysfunction, inflammation, and oxidative stress, leading to worsened blood pressure control, dysglycaemia, and disrupted lipid metabolism. Extreme weather events, floods, and wildfires trigger acute spikes in cardiovascular events through dehydration, myocardial ischaemia, and arrhythmias, while also disrupting healthcare delivery and medication adherence. Moreover, climate-driven changes in food systems and nutritional quality exacerbate unhealthy dietary behaviours, further amplifying cardiometabolic risk. Vulnerable populations—including older adults, racial and ethnic minorities, and those of lower socioeconomic status—bear a disproportionate burden of these effects. Mitigating the cardiovascular consequences of climate change requires integrated approaches that incorporate climate-sensitive risk stratification, targeted education of patients and clinicians, and adaptive health system responses. Primary care physicians play a central role in delivering anticipatory guidance and equitable care to at-risk individuals. This review synthesizes evidence linking climate change with CVD risk profiles. It outlines clinical and public health strategies to strengthen climate resilience in cardiovascular medicine.

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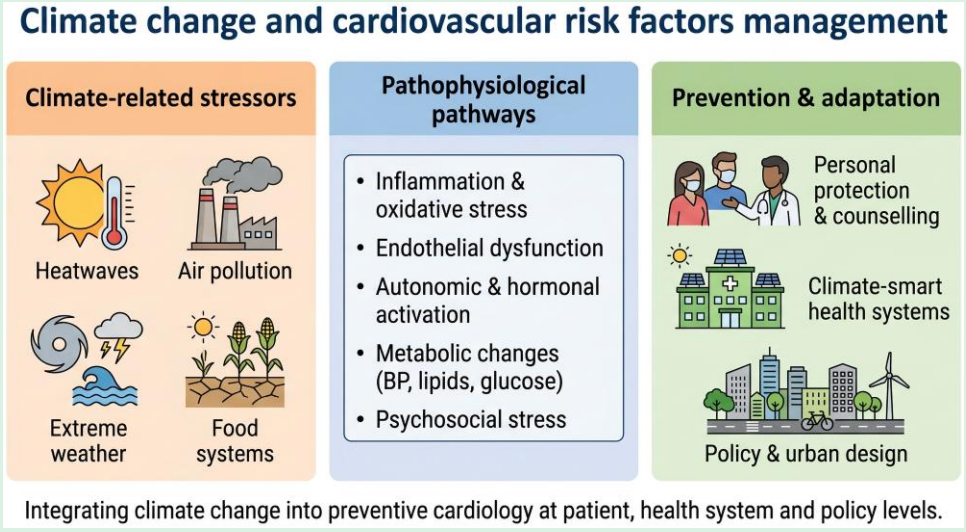
Graphical Abstract



Keywords Climate changes • Cardiovascular prevention • Primary care

Key messages

- Clinicians should incorporate environmental exposures (heat, air quality, noise, and climate-related stress) into cardiovascular risk assessment.
- Heatwaves and poor air quality require proactive adjustments in the management of hypertension, diabetes, and heart failure.
- Physical inactivity driven by environmental constraints as a major amplifier of CV risk.
- Women, children and older adults face unique physiological and social vulnerabilities that must be addressed with targeted strategies.
- Climate-resilient cardiovascular care requires coordinated clinical, community, and policy actions.

Introduction

Climate change, the long-term alteration of global or regional climate patterns, is primarily driven by human activity and is increasingly recognized as a worsening global crisis with profound implications for human health and well-being.[1] It threatens fundamental determinants of health, such as clean air, safe water, food security, and shelter, potentially reversing decades of public health progress. Among these threats, air pollution and rising temperatures pose particularly significant risks for individuals with, or at elevated risk of, cardiovascular disease (CVD).[1–3]

Human-generated greenhouse gases accumulate in the atmosphere, driving global warming and posing hazards such as more frequent and severe heatwaves and altered precipitation pattern.[4,5] The Intergovernmental Panel on Climate Change (IPCC) identifies climate change as a pressing public health challenge of the 21st century. Within this context, understanding its implicit on cardiovascular health is essential for developing targeted preventive strategies and adaptive responses to mitigate its adverse effects. Extreme heat is already taking a toll on public health, with adults over 65 experiencing an average of 13.8 heatwave days per person in 2023—the highest on record. Since the 1990s, heat-related deaths in this age group have risen by approximately 85%.[5] Abnormal environmental conditions like extreme temperatures, air pollution, and noise contribute to over 18% of CVD-related deaths in Europe.[6] These data underscore the urgency of integrating environmental determinants into cardiovascular prevention and management frameworks.

Climate change not only increases the incidence of cardiovascular events but also complicates the management of underlying risk factors, particularly in vulnerable groups.[7,8] Children, women and older adults are disproportionately affected due to sex-specific hormonal changes, age-related endothelial dysfunction, and reduced thermoregulatory capacity, all of which heighten susceptibility to heat stress, air pollution, and climate-related cardiometabolic risk.

Managing cardiovascular risk factors in the context of a changing climate requires innovate, climate-informed considerations. Environmental exposures and lifestyle disruptions associated with climate change exacerbate traditional risk factors such as hypertension, diabetes, and obesity. Climate-induced disruptions to healthcare access, medication adherence, and the rising burden of air pollution and allergens further complicate cardiovascular risk management.

In this review, we examine how climate change affects major cardiovascular risk factors, synthesize current evidence on these interactions, and propose practical strategies for clinical and public health management. We emphasize the crucial need to integrate climate change considerations into cardiovascular risk management frameworks to mitigate worsening health outcomes. We advocate for the development and implementation of adaptive care strategies that address extreme weather

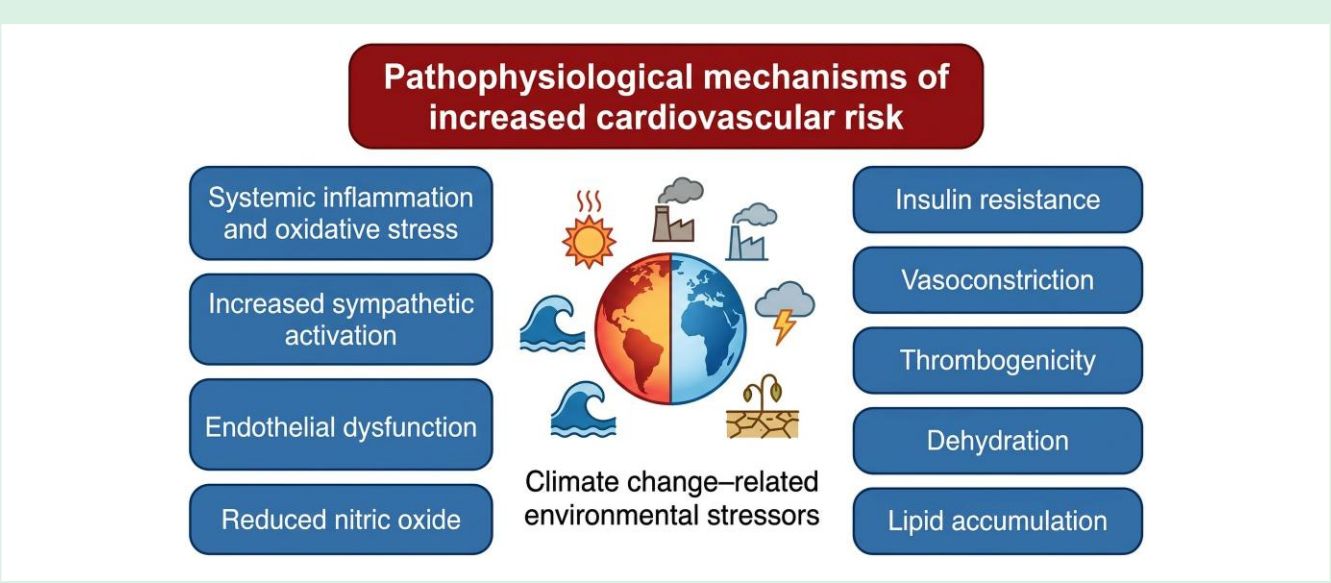


Figure 1 Pathophysiological mechanisms linking climate change to cardiovascular morbidity.

events, environmental pollution, and the social determinants of health exacerbated by climate change.

Methods

This document developed by a multidisciplinary panel of cardiologists, primary care physicians, exercise medicine specialists, internal medicine physicians, public health experts, and researchers. The writing group was organized into topic-specific subgroups, each responsible for drafting sections based on their clinical and scientific expertise.

A non-systematic narrative review of the literature was performed to identify key evidence on climate change and cardiovascular risk factors. Sources included PubMed/MEDLINE, major international reports (IPCC, WHO), key position papers and statements from cardiovascular societies that were judged by the writing group to be most relevant and up-to-date. Additional references were identified through citation tracking of seminal articles and the clinical and research experience of the multidisciplinary author team. Given the scope of this consensus, no formal search strategy, inclusion/exclusion criteria, or risk-of-bias assessment was applied.

The preliminary drafts produced by each subgroup underwent iterative rounds of revision and collective discussion. Consensus on the content was reached when ≥80% of the writing group agreed on the wording and interpretation. All authors reviewed and approved the final manuscript.

Climate change and cardiovascular health: Multifaceted direct and indirect impacts

Climate change presents a complex array of direct and indirect impacts on cardiovascular health (*Figure 1*), damaging health infrastructure and hindering access to healthcare services. Healthcare utilization patterns change during escalating weather phenomena, increasing demand for emergency services often due to significant disruptions in routine health care facilities and financial constraints.[9] Extreme weather events such as heatwaves, floods, storms, and droughts are increasing in frequency and severity. These contribute to increased cardiovascular morbidity and higher mortality rates, provoking heat stress, dehydration, myocardial infarction, stroke, and exacerbations of chronic cardiac conditions.[1–3] Adverse environmental exposures, especially air pollution, are significantly contributing to increased cardiovascular risk. More than three million annual deaths from ischaemic heart disease and stroke are linked to air pollution alone.[10,11] Air pollution contains a mix of particulate matter (PM) and gaseous pollutants like ozone, nitrogen dioxide, carbon monoxide, and sulphur dioxide. Fine particles such as PM2.5 are now well established as significant cardiovascular risk factors.[11–14] Interactions between pollution and weather extremes, especially high temperatures, further amplify these risks by enhancing the health effects of pollutants.[15,16]

Climate change also drives the emergence of new infectious disease threats with cardiovascular implications. Shifts in temperature and precipitation expand the habitats of vectors like mosquitoes and ticks, spreading illnesses such as malaria and Lyme disease, and increasing waterborne infections, all of which can exacerbate cardiovascular morbidity and mortality through systemic inflammation and stress.[17] Some regions may paradoxically experience extremely low temperatures, with one meta-analysis showing that 82% of studies quoting an association between cold exposure and increased cardiovascular mortality. These changes persisted even after accounting for influenza infection and seasonality.[7]

Tropic storms, hurricanes, floods, and mudslides have also been linked to increased CVD mortality and morbidity, ranging from. This often outlasted the extreme weather event, suggesting the long-term ramifications of these conditions.[7] Simultaneously, climate change contributes to rising psychosocial stress and mental health challenges. More than half of adults now report climate-related anxiety, depression, post-traumatic stress disorder, and sleep disturbances, all of which are established CVD.[18–20] This inadvertently leads to higher rates of obesity, diabetes, and micronutrient deficiencies. Reduced crop yields, impaired nutrient content, and water scarcity drive transitions toward less nutritious and more processed diets, which in turn increase the incidence of obesity, diabetes, and micronutrient deficiencies.

Traditional Mediterranean agriculture and dietary patterns offer resilience, but adaptation of food safety and nutritional policies is urgently required to address these ongoing risks.[21] Additionally, damage to marine ecosystems threatens fish populations integral

to heart-healthy diets. Tackling the multifaceted cardiovascular health impacts of climate change demands integrated approaches that incorporate environmental health, infectious disease management, mental health support, and food system adaptation. These disruptions jeopardize both food security and the nutritional quality essential to the Mediterranean diet, potentially undermining its cardiovascular health benefits.

Pathophysiology of climate change effects on cardiovascular function

Heat stress and cardiovascular function

Exposure to elevated ambient temperatures triggers a series of coordinated physiological responses designed to dissipate heat and maintain normothermia.[22] These include cutaneous vasodilation, which increases skin blood flow to facilitate convective and radiative heat loss; sweating, which facilitates evaporative cooling; and behavioural adjustments such as reduced activity, shade seeking shade, and increased hydration.[22,23]

Acute heat stress significantly strains the cardiovascular system by elevating heart rate, blood flow, and cardiac output, which leads to a higher cardiovascular workload. Climate chambers studies show that for each 1°C rise in core temperature, heart rate increases by approximately 26 beats per minute for each, cardiac output rises by an average of 1.0 [0.4, 1.6] L/min, and systolic pressure changes by −8 [−15, 0] and 2 [−2, 7] mmHg per 1°C temperature increase.[24] These adaptive mechanisms redistribute warm blood to the skin surface and promote evaporative heat loss through sweating. However, when thermoregulatory capacity is exceeded, severe hyperthermia (core temperature > 40.5°C), can lead to heat stroke, characterized by central nervous system dysfunction ranging from confusion to coma.

Classic heat stroke primarily affects older adults and individuals with chronic illnesses that impair their ability to dissipate heat. In contrast, exertional heat stroke typically occurs in younger individuals, such as athletes and outdoor workers, whose increased heat production from physical activity overwhelms their body's cooling mechanisms. Both forms triggers vasodilation, increased cardiac output, and fluid loss, leading to electrolyte imbalance. Extreme heat and humidity worsen thermal stress by reversing heat transfer. Prolonged heat exposure induces cytokine-driven inflammation, coagulation activation, and electrolyte loss, thereby increasing the risks of thrombosis, destabilization of atherosclerotic plaques, and arrhythmias.[25,26] Coupled with endothelial injury and impaired vasodilation from dehydration and oxidative stress, these mechanisms heighten cardiovascular risk both during and after heatwaves. Cardiovascular dysfunction, especially in the elderly and those with heart disease, raises heat stroke risk, with common cardiac issues including tachycardia, arrhythmias, ischaemia, heart failure, and sudden death. Some cardiac medications increase the risk of heat stroke.[26] Treatment focuses on rapid cooling below 39°C and supportive care. Prevention includes staying hydrated, wearing proper clothing, and avoiding heat exposure during heat waves. In environmental stress contexts relevant to climate change, sympathetic nervous system (SNS) activation is a frequent pathway coupling exposures to cardiovascular burden. During heat stress, core and skin temperatures rise, and circulatory demands shift blood to the cutaneous vasculature; to defend arterial pressure, baroreflex-mediated SNS outflow increases muscle sympathetic nerve activity (MSNA). Experimental human studies confirm that significant increases in MSNA occur during passive whole-body heating. Reflex tachycardia is commonly observed, while stroke volume (SV) may initially be maintained or even elevated due to enhanced contractility and augmented preload. However, at prolonged or extreme heat exposures—especially with dehydration—SV often declines as central volume becomes reduced.

Air pollution synergistic toxicity

Major air pollutants include nitrogen dioxide (NO_2), ozone (O_3), and sulphur dioxide (SO_2). Small particulate matter (PM2.5) with an aerodynamic diameter of <2.5 μm is particularly hazardous.[11] Exposure to PM air pollution induces adverse cardiovascular effects through diverse and complex cellular pathways. Oxidative stress and inflammation are key hallmarks of PM exposure. This oxidative stress depletes the body's antioxidant defenses, disrupts cellular homeostasis, and causes damage to lipids and DNA in circulation.[11] Another critical consequence in the cardiovascular system is the reduction of nitric oxide bioavailability—a key molecule that commonly promotes vasodilation, limits smooth muscle proliferation, inhibits platelet aggregation, and modulates inflammatory cells.[27]

Climate change impact on Major cardiovascular risk factors

Hypertension

There is growing evidence highlights in how non-traditional environmental factors interact with underlying physiological conditions that impact blood pressure regulation. Notably, epigenome-wide studies confirmed a link between environmental exposure and epigenetic changes promoting a rise in blood pressure (BP).[27]

High ambient temperatures and heat waves are strongly associated with increased CVD morbidity and mortality.[2,4] Most recent evidence indicate that global warming presents new risks for hypertension. Heat exposure and humidity redirect blood flow to the skin, increase heart rate, and cause fluid loss, leading to reduced plasma volume, increased blood viscosity, and a prothrombotic state. Seasonal differences in BP show higher values in colder months and lower day-time systolic BP in hot weather.[28,29] However, higher night-time BP and non-dipping patterns have been observed during hotter days and in summer.[30,31] Older people and those with pre-existing CVD are at higher risk due to impaired vascular responses to higher temperatures.[32] Heatwaves are linked to increasing BP in vulnerable populations, including those with hypertension, diabetes, and obesity. Mitigation strategies at the individual and population levels need to be considered, but their effectiveness has not been established.

The environmental 'exposome' contributes significantly to the global burden and disparities in hypertension, through shared biological axes, primarily neuroendocrine activation, systemic inflammation, and endothelial dysfunction. Some environmental factors may affect BP regulation or even trigger hypertension directly, while others may indirectly lead to hypertension. For example, urban areas with limited green space may promote sedentary behaviour, contributing to hypertension.

Air pollution has been consistently linked to hypertension. A meta-analysis showed that each 10 $\mu g/m^3$ additional exposure to PM2.5 increased systolic BP by 0.63 mmHg and diastolic BP by 0.31 mmHg, with a similar association for smaller and bigger PM.[33] Portable air cleaners removing PM2.5 decreased systolic BP by 3.9 mm Hg per 20.9 $\mu g/m^3$ decrease in PM2.5 (0.19 mmHg per 1 $\mu g/m^3$ reduction in PM2.5).[34] The global population-level increase in BP, estimated as the magnitude of BP elevation above the World Health Organization-recommended goal threshold of 5 $\mu g/m^3$, was 2.4/1.2 mmHg with notable regional variations.[35] This may at least partly explain the excess in hypertension in Black, Asian, and Hispanic or Latino communities in the United States who are exposed to higher PM2.5 levels.[35] The presence of metals and chemicals in food and groundwater, resulting from manufacturing technologies and inadequate waste disposal, further contributes to hypertension. The strongest association between manufactured chemicals and hypertension has been demonstrated for halogenated hydrocarbons, perfluoroalkyl substances, and

plastic-related chemicals, including phthalates, such as perfluorononanoic acid, perfluorooctanoic acid, and perfluorooctane sulfonic acid. Metals such as lead, cadmium, and mercury are strongly related to hypertension, often in a dose-dependent manner.[36]

Chronic exposure to environmental noise, mainly from transport and occupational sources, is another modifiable contributor. A 10 dBA increase is associated with a 1.2 mmHg rise in systolic BP and 1.1 mmHg in diastolic BP.[37] Although modest individually, these increases translate into significant population-level hypertension burden. Road traffic noise levels >65 dBA have been demonstrated in the UK Biobank population.[38] A meta-analysis of studies of road traffic noise found a 1.07 odds ratio (95% confidence interval 1.02–1.12) increase in hypertension prevalence per 10 dB noise increase.[39] Living near large airports in France showed a 1.36 hazard ratio for hypertension.[40] There was a 14% higher odds of hypertension per 10 dB night-time aircraft noise in the Hypertension and Exposure to Noise near Airports (HYENA) study.[41] Furthermore, residential noise increased as associated with the presence of apparent treatment-resistant hypertension,[37] long-term exposure to smog and fine particulate matter is associated not only with a higher incidence of myocardial infarction and stroke but also with increased cardiovascular mortality, as demonstrated in large population-based studies.[42]

Furthermore, in population-based cohorts, higher levels of outdoor artificial light-at-night have been associated with a greater prevalence of hypertension and higher incidences of obesity, hypercholesterolaemia, and haemorrhagic stroke.[43]

The mechanism linking noise to hypertension is likely driven by activation of the sympathetic nervous system, leading to chronically raised stress hormones. Cortisol and adrenaline trigger a cascade of inflammatory pathways leading to endothelial dysfunction. While there are no adequate trials providing evidence that noise reduction lowers BP, reduced environmental noise during COVID-19 lockdowns appeared to lower BP. Occupational exposure to chronic noise exceeding 60 dBA has led to 3.6-fold higher odds of hypertension.[44]

In women, hypertension cannot be interpreted solely through traditional clinical risk factors, as the female exposome, influenced by social disadvantage, chronic stress, air pollution, and unequal access to healthy lifestyle resources, interacts with hormonal transitions and endothelial vulnerability, accelerating inflammatory pathways and magnifying lifetime cardiovascular risk.[45]

Mitigation environmentally driven hypertension requires shifting from a reactive pharmacological approach to proactive prevention through robust environmental policy, improved urban planning, occupational health protections, and integration of ecological risk assessment into clinical practice.

Physical inactivity due to environmental and behavioural constraints

Air pollution exerts a bimodal effect on the health benefits of physical activity (PA). In a cohort of 469 972 Korean young adults, for those living in low-pollution areas, the relationship between the protective effect and the amount of PA is direct and linear.[46] Conversely, in highly polluted areas, a trade-off emerged between the benefits of PA and the adverse effects of excessive exposure to air pollution, indicating that environmental context critically modifies the health outcomes of PA. Beyond its direct physiological impact, air pollution indirectly affects PA engagement. In both children and older adults, accelerometer-based studies have demonstrated that increasing pollution levels correlate with reduced outdoor activity and greater sedentary time. This behavioural adaptation to environmental conditions disproportionately affects vulnerable individuals, further amplifying health inequalities.

In densely populated urban areas, exposure to multiple environmental stressors, such as heavy vehicular and aircraft noise, light and air pollution, synergistically contribute to physical inactivity. These combined exposures increase the risk not only of ischaemic heart disease but also of heart failure and arrhythmias. Predictors of PA are influenced by both individual and contextual factors. In a cohort of over 99 000 Brazilian adolescents, PA was associated with being male, older, and having a longer commute to school.[47] Other factors include the availability of green spaces, which can also reduce heat, neighbourhood crime levels, the socioeconomic status of families, social isolation, and the type of school attended (smaller schools achieved a higher level of PA, likely due to a greater availability of open spaces).[47] Built and environmental factors can either promote or hinder PA. Facilitators include accessible exercise and recreation facilities, low-traffic sidewalks or streets, walkability, and a pleasant natural environment. Conversely, the lack of sports spaces, high traffic volumes, unsafe roads, and poor street lighting act as significant barriers. Importantly, social and familial factors, such as parental education, the quality of the parent-child relationships, and good school-family partnerships, combined with a good level of education at school, can also encourage adolescents to engage in adequate PA.[48]

To optimize health outcomes, individuals should be encouraged to engage in PA in environments characterized by low pollution, green spaces, and minimal noise, while avoiding extreme temperatures and high-pollution periods of the day. On a systemic level, healthcare professionals must also contribute to reducing the sector's environmental footprint, including increased use of green energy in healthcare facilities, implementing telemedicine to reduce travel-related emissions, adopting reusable and compostable materials, and minimizing the carbon footprint associated with supplies and pharmaceuticals. Finally, they must support advocacy initiatives to promote policies and endorse candidates that address the issues of air pollution and climate change.

Dyslipidaemia

Climate change, through rising temperatures and increased air pollution, is associated with worsening lipid profiles and a higher risk of hyperlipidaemia, contributing to an increased burden of CVD—especially in vulnerable patient groups.[11]

A meta-analysis examining LDL and HDL levels during cold and warm periods showed increased LDL levels during colder months (probably due to diet changes or blood volume) and conflicting results for HDL and triglycerides[49] but long term exposure to high-temperature seems to increase significantly LDL levels.[50] Global warming reduces the levels of beneficial omega-3 fatty acids in bivalves, such as mussels and oysters—particularly in temperate regions—which could diminish the cardiovascular and metabolic health benefits of these foods for humans. Heat stress and temperature changes modulate the expression of key lipid metabolism proteins, including increasing lipoprotein lipase, fatty acid synthase, acetyl-CoA carboxylase, and heat shock proteins; it also affects central regulators such as PPARγ and C/EBPα, promotes sphingolipid metabolism (raising ceramides and sphingomyelins), and can lead to greater lipid accumulation and triglyceride synthesis in tissues, with effects that vary across species and environmental contexts.[51]

In parallel, heat exposure increases circulating free fatty acid concentrations by stimulating lipolysis.[52] This systemic fat mobilization, combined with altered lipid-handling enzyme activity, can alter lipid profiles, and may, in some cases, contribute to elevated plasma triglyceride and cholesterol levels. Environmental pollutants, such as dichlorodiphenyltrichloroethane and its metabolite dichlordiphenylethylene, are associated with increased obesity and disturbed lipid composition by impairing the mass and function of brown adipose tissue.[52] Animal studies have shown that combined exposure to air pollutants and heat profoundly disrupts lipid metabolism across multiple organs, leading to impaired hepatic cholesterol clearance,

increased lipolysis in white adipose tissue, and phenotypic 'whitening' of brown adipose tissue.[46,50]

Diabetes mellitus

Climate change, elevated temperatures, and heatwaves can impair insulin sensitivity, alter glucose regulation, and exacerbate chronic low-grade inflammation, all contributing to the development and worsening of diabetes. Prolonged heat exposure causes dehydration and electrolyte imbalances, interfering with insulin action and hepatic gluconeogenesis, thereby promoting insulin resistance. In addition, climate-related stress and disrupted food security may lead to unhealthy diets and reduced physical activity, further impairing glucose control.[53]

Evidence suggests that increased ambient temperatures are associated with higher incidence rates of both type 1 and type 2 diabetes and gestational diabetes during hot seasons.[52] Dehydration and vasodilation during heat stress can inhibit cellular insulin signalling, increasing the risk of hyperglycaemia and diabetes-related complications.[53] Cold temperatures can also provoke metabolic stress and impact glucose regulation, although the primary concern remains the effects of heat on insulin resistance.[53] Exposure to extreme temperatures, increases the risk of adverse diabetes-related events, higher rates of hospitalizations, emergency department visits, and diabetes-related mortality.[52]

Exposure to extreme temperatures, both heat and cold, substantially increases the risk of adverse diabetes-related events, leading to higher rates of hospitalizations, emergency department visits, and diabetes-related mortality.[54] Extreme temperatures and weather events—such as heatwaves, floods, and wildfires—increase the risk of complications and acute medical events like hypoglycaemia, dehydration, and cardiovascular issues in people with diabetes. These events can disrupt access to essential medical supplies, including insulin and other medications, and interfere with healthcare delivery by causing disruptions to transportation or infrastructure. Air pollution and rising ambient temperatures exacerbate glycaemic control by promoting inflammation and oxidative stress, while disruptions to the food system may compromise dietary management. Patients with diabetes are particularly vulnerable due to impaired thermoregulation and comorbid conditions, and some medications may amplify sensitivity to heat.

In women with type 2 diabetes, environmental and social exposures function as cumulative risk amplifiers, worsening inflammatory and metabolic profiles, resulting in pollution-induced vascular injury, ultimately translating into earlier onset and faster progression of CVD compared to other populations.[55,56]

Tobacco and substance use changes in climate-affected contexts

Tobacco and substance use behaviours are influenced by climate-affected contexts, often leading to changes in consumption patterns.[57] Climate-related stressors such as extreme weather events, displacement, economic hardship, and psychological distress can increase vulnerability to both the initiation and continuation of tobacco and substance as maladaptive coping responses. These conditions may exacerbate existing dependencies and hinder cessation efforts. Additionally, climate-induced disruptions to social support and healthcare infrastructure can reduce access to cessation support and treatment for substance use disorders, thereby amplifying adverse public health outcomes. Recognizing and addressing these interactions is crucial for designing effective interventions in climate-affected regions. Young people are particularly at risk, given their heightened susceptible to mental health and substance-use disorders and their prolonged exposure to the escalating impacts of climate change.[58]

Climate- and pollution-related cardiovascular vulnerability in distinct populations

Women: sex-specific pathways and social determinants

Women may experience a disproportionate cardiovascular burden from climate-related environmental stressors, driven by physiological, hormonal, and social vulnerability factors.[8] Women experience climate-related cardiovascular risk through a unique combination of biological, nutritional, psychosocial, and socio-cultural pathways, which collectively amplify lifetime exposure to environmental stressors.[58] Climate change disrupts multiple determinants of diet quality and cardiometabolic health, and women are disproportionately affected due to their physiological characteristics, caregiving roles, and heightened exposure to food-related responsibilities.[59] Social and cultural roles increase women's exposure to dietary instability. Women are often primarily responsible for food procurement, preparation, and household nutrition, all domains disrupted by climate-related food shortages, price spikes, and reduced availability of fresh produce. These disruptions force shifts towards cheaper, calorie-dense, nutrient-poor foods, contributing to obesity, metabolic syndrome, and hypertension, especially among disadvantaged women. Psychosocial stressors linked to food insecurity and environmental instability further contribute to unhealthy eating patterns, emotional eating, and stress-related cardiometabolic dysregulation. Finally, although sustainable dietary patterns such as the Mediterranean diet are highly protective for women, climate change threatens their viability by compromising agricultural resilience and nutritional value. Warmer temperatures, water scarcity, and altered ecosystems reduce availability of fruits, vegetables, and fish, core components of the Mediterranean pattern, posing a direct risk to women's cardiovascular prevention efforts. Air pollution and climate-driven disruptions to the food system also adversely affect metabolic pathways in women, promoting dyslipidaemia, visceral adiposity, and inflammatory activation that accelerate atherosclerotic progression.[60]

Older adults: age-related vulnerabilities

Older adults represent another population at markedly increased vulnerability. Ageing is associated with diminished thermoregulatory capacity, impaired cutaneous vasodilation, blunted sweating responses, and reduced thirst perception, all of which elevate the risk of dehydration, heat exhaustion, and heat stroke during periods of extreme heat.[61] Older adults also face indirect vulnerabilities: multimorbidity, polypharmacy, social isolation, reduced mobility, and dependence on medical devices or temperature-sensitive medications. Extreme heat and poor air quality can affect medication safety and efficacy, increasing risks of electrolyte disturbances, hypotension, and arrhythmias, particularly among those on diuretics, RAAS inhibitors, or beta-blockers. Climate-related disruptions to healthcare access, such as power outages, transportation barriers, and medication supply interruptions, further compromise chronic disease management in older adults.[60] Overall, both women and elderly individuals require targeted preventive strategies, including climate-sensitive risk assessment, patient education on heat and air-quality safety, medication monitoring during extreme weather, and strengthened social and healthcare support systems. However, vulnerability to climate- and air-pollution-related cardiovascular effects is heterogeneous across the life course. Although many studies report increased susceptibility among women and older adults, other data show that younger adults, adolescents and children can experience similar or even greater risks.[62,63] Emerging evidence also suggests that early-life and cumulative exposures may program long-term cardiometabolic vulnerability. Accordingly, no single demographic group can be regarded as uniformly 'most vulnerable'; rather, risk reflects interacting biological, behavioural, and social factors operating at different ages.[64]

Children and adolescents as climate-sensitive populations for early cardiovascular risk

Children and adolescents represent an often overlooked but highly vulnerable population in the context of climate change and environmental stressors, with potential long-term cardiovascular implications. Early life exposure to ambient air pollution—including fine $PM_{2·5}$, nitrogen oxides, traffic-related pollutants—has been associated with adverse changes in cardiometabolic markers, such as elevated body mass index, insulin resistance, and hypertension during adolescence or early adulthood.

Moreover, in very young children (e.g. preschool age), traffic-related air pollution has been linked to upregulation of circulating endothelial progenitor cells possibly reflecting an early vascular response to endothelial injury, a plausible early signal of vascular stress long before overt disease.[65]

In addition, children exhibit unique physiological susceptibilities: their thermoregulatory mechanisms, vascular and metabolic systems, and lung–heart interactions are still in development, making them less resilient to environmental stressors compared with adults. Early disruptions, during critical windows of growth, may program lifelong increased cardiovascular risk. Finally, environmental and social determinants often disproportionally affect youth, amplifying vulnerability.

Children are physiologically less able to regulate body temperature, and evidence shows that heat waves significantly increase paediatric morbidity.[8] Although cardiovascular outcomes are rarely reported directly, these heat-related disturbances occur during critical developmental windows and may contribute to early autonomic and vascular strain, supporting the classification of children as a climate-sensitive at-risk group.

Preventive cardiology in the age of climate change

Patients' awareness and health professionals' awareness

Understanding the level of awareness among both patients and healthcare professionals regarding the cardiovascular impacts of climate change is critical for effective CVD prevention and management. Strengthening knowledge of how environmental stressors affect cardiovascular function is essential for mitigating associated risks effectively. Improved awareness and education are foundational to building adaptive clinical care and public health strategies in the context of ongoing environmental change.

Despite increasing evidence, patient awareness of climate-related cardiovascular risks remains inconsistent. Many individuals do not recognize the impact of heatwaves or air pollution on their heart health, which can delay seeking timely care or adopting preventive measures.[66] Patient education on avoiding exposure during high-risk periods, recognizing symptoms, and managing chronic conditions is essential for reducing adverse outcomes.[67] For example, clinicians should advise patients with diabetes to have a diabetes emergency kit with at least a 14-day supply of medications, blood glucose monitoring supplies, and extra batteries.[67] Important documents, such as medication lists, care plans, and emergency contacts, should be included and kept in a waterproof container. Education should also include guidance on recognizing signs of high and low blood sugar, adjusting insulin during periods of disruptions, and alternative insulin storage during power outages.[67]

Among healthcare professionals, awareness of climate change as a cardiovascular risk factor is improving but is not yet universal. Increasing numbers of cardiologists are acknowledging the role of environmental factors in exacerbating CVD and advocating for the integration of climate considerations into clinical practice.[61] However, barriers such as limited clinical guidance, inadequate training, and lack of formal frameworks in 'climate cardiology', impede widespread implementation. Physicians have a responsibility to inform patients, incorporate environmental risk assessment, and engage in advocacy for health-protective policies.[65]

Medication adaptations

In the context of climate change, medication management necessitates careful consideration of altered drug safety, efficacy, and storage profiles resulting from environmental stressors, including heatwaves and extreme weather events (*Table 1*). Pharmaceuticals commonly prescribed for chronic conditions like hypertension, diabetes, and hyperlipidaemia may demonstrate variable stability under such conditions, impacting therapeutic outcomes.[67]

Heat exposure can alter pharmacokinetic and pharmacodynamic properties, affecting drug absorption, distribution, metabolism, and elimination. For instance, diuretics and angiotensin-converting enzyme (ACE) inhibitors—commonly used antihypertensive medications—can increase the risk of dehydration and electrolyte imbalances under heat stress, necessitating dose adjustments or close monitoring to prevent adverse events. Similarly, insulin and specific oral hypoglycaemic agents are susceptible to thermal degradation, which reduces their potency and necessitates stringent cold-chain maintenance and patient education regarding storage and handling. Lipid-lowering therapies may also be indirectly affected though climate-induced dietary changes that alter lipid metabolism and drug responsiveness.[68]

Additionally, the integrity of pharmaceuticals is compromised by elevated temperatures and humidity during storage, as well as transport disruptions caused by extreme weather. This necessitates enhanced supply chain resilience, including temperature-controlled logistics and real-time monitoring to preserve drug efficacy.[67]

Clinicians should integrate climate considerations into therapeutic decision-making, incorporating dynamic medication reviews, patient-specific risk assessments, and anticipatory guidance on medication management during extreme weather events. Recent evidence indicates that individuals with stage 1 hypertension, irrespective of medication use, may maintain thermal balance compared to those without hypertension, even under extreme hot and humid or hot and dry conditions[69]

Medication management in patients with hypertension during heat waves should be individualized and flexible, based on continuous monitoring and clinical presentation. Self-monitoring of BP is essential in cases of clinically significant hypotension, cautious dose reduction of antihypertensives is warranted, especially targeting diuretics.[69] However, complete discontinuation of antihypertensive therapy is generally discouraged due to the risk of rebound hypertensive crises during abnormal heat and the subsequent increase in BP once cooler conditions return.[67] Patients receiving diuretics require individualized daily monitoring of fluid intake and body weight to identify hypovolaemia or electrolyte imbalances quickly. Certain antihypertensive medications affect thermoregulatory processes primarily through their vascular actions, which can attenuate the typical increase in skin blood flow necessary for efficient heat dissipation, potentially leading to elevated core body temperatures. This effect is particularly evident with non-selective beta-adrenergic blockers, such as propranolol, which decrease skin temperature without significantly altering sweating responses. Experimental evidence during exercise demonstrates that acute administration of non-selective beta-blockers can increase oesophageal temperature by reducing cutaneous vasodilation and sweat production, thereby increasing heat storage.[60] Overall, medication adaptation should be individualized, integrating patient-specific factors, comorbidities, and environmental conditions rather than applying uniform protocols.[70] In parallel, sustainable prescribing practices, such as selecting medications with

Table 1 Algorithm for cardiovascular medication adjustments in climate change contexts

Assess environmental risk
Monitor for heatwaves, poor air quality, and related warnings
Provide alerts to patients with hypertension, diabetes, or heart failure regarding upcoming environmental risks
Identify at-risk patients
Elderly, those with multiple comorbidities
Patients on medications sensitive to environmental changes (diuretics, ACE inhibitors, beta-blockers, insulin/GLP-1s).
Patient self-monitoring
Instruct on daily monitoring of heatwaves, poor air quality, and related warnings
Encourage regular monitoring of blood glucose levels in individuals with diabetes.
Medication review and adjustment
For heat stress or dehydration risk:
• Evaluate need to reduce or temporarily withhold diuretics or high-dose ACE inhibitors.
• In arterial hypertension, do not discontinue all antihypertensives; prioritize stable BP.
• In diabetes, adjust insulin or oral hypoglycaemic regimens (risk of hypo/hyperglycaemia increases in extreme heat).
Symptom-triggered action
If patient develops hypotension, dizziness, acute kidney injury, electrolyte imbalance, or heat-related symptoms:
• reduce or suspend diuretic/RAAS inhibitor and monitor closely.
• consider reduced doses of diuretics or extended intervals if dehydration or hypotension develops.
In hypoglycaemia:
• lower insulin or sulfonylurea dose, increase glucose monitoring.
• avoid abrupt cessation without physician input unless severe adverse effects noted
Consider reduced doses or extended intervals if dehydration or hypotension develops.
Avoid abrupt cessation without physician input unless severe adverse effects noted.
Consider reduced doses or extended intervals if dehydration or hypotension develops.
Ensure medications are properly stored (insulin, some oral agents can degrade in heat).
Supportive measures
Ensure adequate hydration, cooling strategies, dietary adjustments.
Educate on medication safety in altered climate conditions.
Equipment to reduce contact with air pollution (i.e. face masks, air purifiers, electrostatic precipitators, etc.).
Follow-up
Arrange regular remote assessments or in-person visits during extended periods of extreme weather.
Use telemedicine where possible to maintain medication supervision and adjustment.

lower environmental impact and promoting responsible disposal, align clinical care with broader climate mitigation goals.

Notably, people taking certain cardio-vascular and other commonly prescribed drugs may be at particular risk during periods of extreme heat. Agents such as diuretics, ACE inhibitors/ARBs and mineralocorticoid receptor antagonists can promote volume depletion, electrolyte imbalance and hypotension, which in turn may precipitate syncope, arrhythmias or worsening heart failure. Beta-blockers and some calcium-channel blockers can blunt the compensatory increase in heart rate and vasodilation that supports heat loss, while anticholinergic medications and several psychotropic drugs reduce sweating and thirst, further impairing thermoregulation. In patients receiving SGLT2 inhibitors, RAAS blockers or high-dose diuretics, the combination of drug-induced volume loss and heat stress may heighten the risk of acute kidney injury and cardiovascular complications. Accordingly, during heatwaves, safe prescribing should include targeted medication review and, where appropriate, dose adjustment, anticipatory advice on hydration and cooling, more intensive monitoring of blood pressure, body weight and symptoms, and clear instructions.

Adaptation strategies: clinical, community, and policy levels

Embedding climate-sensitive risk assessment into routine clinical practice is urgently needed. This involves incorporating environmental factors, such as heat and air quality alerts, socioeconomic vulnerability indices, and psychosocial stressors into cardiovascular risk stratification.[71] The 2021 ESC Guidelines on CVD prevention in clinical practice identify air pollution, for the first time, as an important cardiovascular risk factor and advise high-risk individuals to minimize prolonged exposure to elevated pollution levels.[72]

On the other hand, primary care physicians and nurses are uniquely placed to identify patients at heightened vulnerability, including older adults, those with multimorbidity, or residents of socioeconomically deprived areas, and to deliver tailored interventions. Registry-based identification and real-time surveillance are increasingly being used to target outreach during heatwaves, aiming to reduce avoidable hospitalizations and mortality.[70] In addition, mitigation strategies—such as hydration and cooling, air quality control, and noise abatement—alongside close clinical monitoring and pharmacologic modulation may help manage sympathetic burden and reduce cardiovascular risk.

Risk communication is equally central. Climate change represents a diffuse and often abstract threat, yet its translation into actionable advice can save lives. Clear messages addressing hydration, medication adjustment during heat stress, and recognition of early warning signs of myocardial infarction or stroke are crucial. The 2003 European heatwave demonstrated severe mortality burden associated with inadequate preparedness; subsequent implementation of heat-health warning systems and targeted outreach has become core elements of public-health response in Europe.[73]

Today, digital technologies, including SMS alerts, teleconsultations, and community information platforms, offer scalable means of strengthening the dissemination of preventive advice, though their success depends on cultural appropriateness and equitable access.[74]

Adaptation, however, cannot be confined to the clinical setting. Building resilient health systems requires investment in energy-efficient, heat-adapted infrastructure, robust pharmaceutical supply chains, and integrated information systems that link environmental monitoring with health surveillance. Financing models that incentivize prevention, workforce training in climate-informed care, and cross-sectoral partnerships with public health authorities and local government are essential components of systemic

adaptation. Without these measures, the burden of climate-related CVD will continue to fall disproportionately on the most vulnerable.

Equity must remain the guiding principle. Socioeconomically deprived communities and those in low- and middle-income countries, face both greater exposure to environmental hazards and reduced adaptive capacity. Primary care, through its accessibility, continuity, and community orientation, provides a key platform to address these disparities. Equity-focused strategies—such as social prescribing for safe physical activity (PA), community gardens to enhance food security, and locally tailored health communication—connect cardiovascular prevention with broader societal adaptation.

Ultimately, integrating climate resilience into primary care is not only a clinical necessity but a moral imperative. Health systems that fail to adapt risk exacerbating global inequities and amplifying the cardiovascular toll of climate change. In contrast, health systems that place primary care, effective risk communication, and systemic adaptation at their core will be better prepared to confront the defining health challenge of this century.

The American Heart Association statement also emphasizes that individuals can take specific, evidence-informed actions to reduce their personal exposure to particulate matter and thereby lower cardiovascular risk.[74] Recommended measures include staying indoors or limiting outdoor activity during high-pollution episodes, using high-efficiency indoor air filtration when feasible, and avoiding proximity to major traffic or other local pollution sources when exercising or commuting (*Figure 2*).[73] The document also stresses that these personal strategies are meant to complement, not replace, population-level policies, and that choices should be tailored to each person's risk profile, local air-quality conditions, and practical constraints.

Alongside personal protective actions, meaningful reduction of exposome-related cardiovascular risk also requires coordinated structural efforts. These include stricter enforcement of air-quality and noise standards, acceleration of the transition away from fossil fuels, and urban-planning policies that promote clean transport, green spaces, and heat-resilient housing and public spaces. Health systems should also be prepared for climate-related extreme conditions by implementing early warning systems for heat and air pollution and ensuring continuity of cardiovascular care.

Finally, climate- and pollution-related cardiovascular risks are often higher in low- and middle-income countries because exposure levels and population vulnerability are greater, while access to preventive cardiology and emergency care is more limited. Adaptation therefore needs to rely on practical, low-cost measures, such as cleaner household energy, basic heat-health action plans, reliable supply of essential cardiovascular medications, and integration of climate- and pollution-related risk counselling into primary care and community-based prevention.

The pivotal role of primary care physicians in managing cardiovascular risk amidst climate change

Primary care physicians (PCPs) are on the frontline of managing the cardiovascular consequences of climate change, yet a clear gap exists between recognizing the problem and feeling adequately equipped to address it with patients. Primary care providers are in a unique position to identify vulnerable patients, provide anticipatory guidance, and manage cardiovascular risk factors in the context of a changing climate. Recent studies reveal a concerning paradox: while most PCPs acknowledge the relevance of climate change to their practice, a significant portion feel ill-equipped to address it. A 2023 survey found that while two-thirds of physicians believe climate change is relevant to primary care, less than one-third thought they should take an active role in discussing it with patients.[75] Similarly, a 2024 national survey of frontline clinic staff found that only 21.3% reported knowing 'a good deal' or 'a lot' about the health effects of climate change, whereas nearly a quarter (24.8%) admitted to knowing 'nothing at all' or 'hardly anything'.[74] This lack of knowledge is a significant barrier to effective patient care, with 61.2% of providers citing it as a reason for not discussing climate-related health risks with patients.[76] PCPs may leverage their trusted position to inform patients about heart risks associated with climate change, including heat safety, air quality, and the advantages of heart-healthy, climate-friendly foods like the Mediterranean diet. However, only 36.2% of providers currently discuss these risks with their patients, often citing limited time or knowledge as barriers.[77] Clinicians should evaluate each patient's vulnerability to climate-related cardiovascular risk based on individual, community, and health system factors. This includes considering a patient's occupational exposures, housing conditions (e.g. lack of air conditioning), and underlying comorbidities (*Table 2*).

Beyond individual patient care, PCPs can also play a vital role in building climate-resilient healthcare systems. This includes advocating for the decarbonization of the healthcare sector, developing contingency plans for extreme weather events, and participating in community-level adaptation and mitigation efforts.[77] A 2024 survey found that over half (54.4%) of frontline clinics have plans to address risks during extreme weather, but significant needs remain, particularly for emergency power and real-time information.[76]

Emerging evidence suggests that targeted education and incentive-based programmes can successfully embed climate-health considerations into primary care. A 2024 intervention study demonstrated that focused education and incentives increased physicians understanding and perceived relevance of climate-health issues, even amid persistent challenges such as a lack of training and time constraints.[72] This suggests that targeted educational interventions can be effective. Encouragingly, more than half of clinicians expressed a desire to learn more about how to help their patients prepare for extreme weather and mitigate cardiovascular risk.[78]

Gaps in knowledge and future perspectives

Despite growing evidence linking climate change to cardiovascular risk, substantial knowledge gaps remain. Most data derive from observational studies, with a lack of prospective cohorts and interventional trials evaluating climate-sensitive prevention strategies, medication adjustments, or targeted counselling. Robust evidence is currently lacking and studies evaluating mitigation strategies such as heatwave-adapted antihypertensive management, tailored physical activity advice during high-pollution episodes, and digital early-warning tools would be highly valuable to guide future practice., or digital early-warning interventions are urgently needed.

A minority of studies report no association between climate exposures and cardiovascular outcomes, highlighting the need for geographically diverse, context-specific research to clarify where and in whom climate factors have measurable effects. Evidence is also limited on how climate change disrupts continuity of care, medication stability, emergency responses, and community-based cardiovascular services; scalable, resilient models using telemedicine, remote monitoring, and robust supply chains should be tested. Global disparities in exposure and adaptive capacity call for international comparative research focused on low- and middle-income countries and for interdisciplinary collaboration across cardiology, primary care, environmental science, digital health, epidemiology, and policy to build climate-resilient cardiovascular prevention frameworks.

Most importantly, political action is essential to reduce cardiovascular risk from air pollution, particularly fine particulate matter (PM2.5). Policymakers should prioritize stricter air-quality standards, because current European Union limit values for annual PM2.5 (25 $\mu g/m^3$)

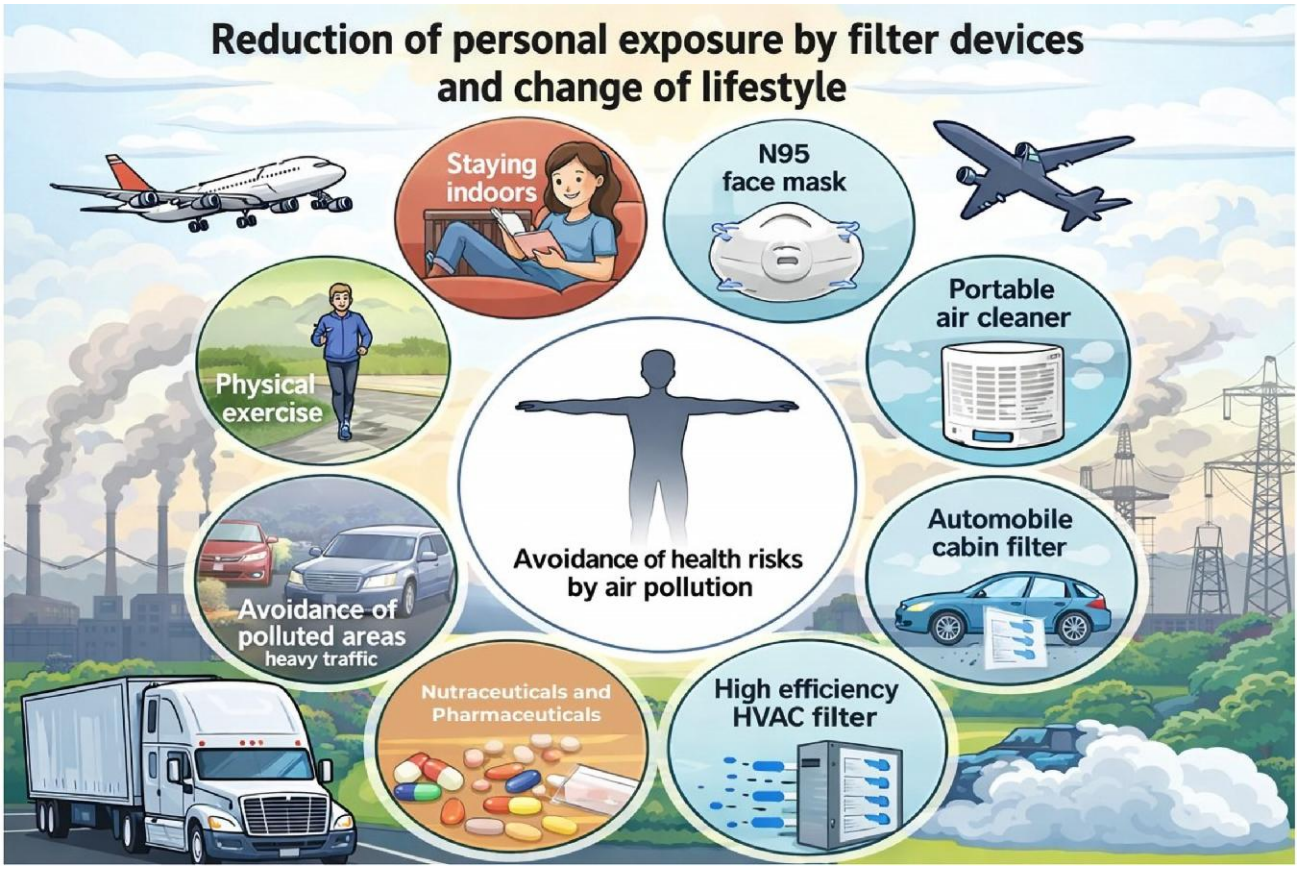


Figure 2 Personal-level actions to mitigate climate change and support cardiovascular health.

Table 2 Evaluation of patients' vulnerability to climate-related cardiovascular risk

Risk domain	Clinical relevance	Key questions for PCPs
Individual susceptibility	Pre-existing CVD, respiratory conditions, diabetes, and renal disease increase vulnerability.	Does the patient have underlying conditions that increase their risk?
Environmental exposure	Outdoor workers, residents of urban heat islands, and those living near high-traffic areas face greater exposure.	What are the patient's occupational and residential exposures?
Social and economic factors	Low-income individuals and those with limited access to resources are disproportionately affected.	Does the patient have access to air conditioning, transportation, and other resources?
Community resilience	The capacity of the local health system and infrastructure to respond to extreme weather events is critical.	What are the local climate hazards and the community's preparedness level?

are substantially higher than the World Health Organization's recommended guideline of 5 μg/m^3, leaving populations exposed to avoidable risk. Stronger regulation of industrial emissions, transport, and residential combustion, alongside urban planning that promotes clean transport and green spaces, can markedly lower PM2.5 levels and prevent pollution-attributable cardiovascular events. Finally, phasing out fossil fuels is a powerful cardiovascular prevention strategy, as it would sharply cut both greenhouse gas emissions and harmful air pollution. Recent evidence indicates that a global fossil-fuel phaseout could avert about 5.1 million premature deaths from air pollution each year, highlighting the enormous health co-benefits of ambitious climate policy for CVD prevention.[79]

Conclusions

The climate crisis represents a significant and multifaceted threat to cardiovascular health worldwide, amplifying existing risk factors while introducing new challenges through its impact on the environment, social determinants, and healthcare systems.

Addressing these complex interactions demands innovative, climate-informed approaches to cardiovascular prevention and management that emphasize equity and resilience.

Comprehensive education on climate changes and its health implications, starting early in school curricula and extending across the lifespan, is fundamental for building climate literacy and fostering adaptive behaviours.

Primary care physicians hold a unique and critical role in leading these efforts; however, significant gaps in knowledge and confidence must be urgently addressed through targeted education and access to resources. Effective prevention of climate- and pollution-related CVD will ultimately depend not only on individual and clinical actions but also on ambitious system-level and policy interventions that improve air quality, decarbonize and climate-proof health systems, and promote heart-healthy environments, especially for vulnerable populations. Integrating climate resilience into cardiovascular care is both a clinical necessity and a moral imperative, essential to safeguarding population health facing of the one of the greatest global challenges of our time.

Author contributions

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