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Review Article

Pollution, climate extremes, and psychosocial stress: emerging environmental risks for cardiovascular disease: a review

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ABSTRACT

Environmental stressors like pollution, extreme temperatures, and psychosocial stress are increasingly recognized as significant but underappreciated contributors to cardiovascular disease (CVD), including cardiomyopathy. These exposures disrupt cardiovascular homeostasis through oxidative, inflammatory, and metabolic pathways, yet their integrated mechanisms remain poorly addressed in clinical risk frameworks. We aim to bridge this gap. We searched PubMed, Embase and Scopus for studies linking pollution, extreme climate changes and psychosocial stress with cardiomyopathy or other CVD outcomes using preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines. A total of 17 epidemiologic and experimental studies reporting CVD outcomes and mechanistic data were included. Air pollution (PM2.5 and PM10) was significantly associated with increased systolic and diastolic blood pressure with 0.2-0.6% higher all-cause mortality, and 0.3-0.7% cardiovascular mortality. These outcomes were mediated by reactive oxygen species (ROS) generation, endothelial dysfunction, and systemic inflammation. Noise and light pollution disrupted circadian rhythms, elevating cortisol, IL-6, and TNF-α levels. Heat exposure triggered autonomic imbalance and endothelial injury via heat shock protein activation, increasing arrhythmia risk. Shared molecular mechanisms included eNOS uncoupling, NADPH oxidase–mediated ROS production, and Ly6C^{+} monocyte activation, promoting atherogenesis and plaque instability. Environmental stressors are emerging, modifiable, and underrecognized contributors to cardiovascular morbidity and mortality. Despite varied triggers, these exposures converge on common mechanistic pathways that fuel myocardial dysfunction. Our findings underscore a need for preventive action. Targeted preventive strategies including air quality regulation, heat stress mitigation, urban greening, and psychosocial support programs are urgently needed. Future research should further delineate these pathways and guide the integration of environmental risk into cardiovascular disease prevention and policy.

Keywords: Environmental stressors, Chronic exposure, Cardiovascular disease

INTRODUCTION

Cardiovascular disease continues to be the foremost cause of global mortality, with traditional risk models focusing primarily on individual factors such as genetics, lifestyle behaviors, and metabolic conditions. However, emerging evidence highlights the significant role of environmental stressors including air pollution, extreme temperatures, and psychosocial stress as under-recognized contributors to cardiovascular pathology. These environmental factors present unique challenges as they exert population-wide effects that transcend geographic boundaries and socioeconomic status.

Pathophysiological mechanisms involve complex interactions between oxidative stress, inflammatory cascades, and endothelial dysfunction, ultimately promoting the development and progression of cardiomyopathy. Recent epidemiological studies demonstrate clear associations between environmental exposures and adverse cardiovascular outcomes, with particulate matter (PM2.5) increasing hypertension risk, heat stress elevating arrhythmia susceptibility, and chronic psychosocial stress accelerating atherosclerotic processes.

Despite these established connections, current clinical risk assessment frameworks and preventive strategies often fail to incorporate environmental determinants. This review systematically examines the biological pathways linking environmental stressors to cardiomyopathy, analyzes their population-level health impacts, and evaluates evidence-based interventions to mitigate these risks.

By integrating findings from environmental science, cardiovascular physiology, and public health research, we aim to bridge critical knowledge gaps and advocate for the inclusion of environmental risk factors in contemporary cardiovascular prevention paradigms.

METHODS

Literature review strategy

An extensive literature search was done using databases such as PubMed, Scopus and Google Scholar. The review was conducted using the PRISMA guidelines.[1] After considerable deliberation 17 articles were selected from PubMed, Scopus, and Google Scholar (Figure 1).

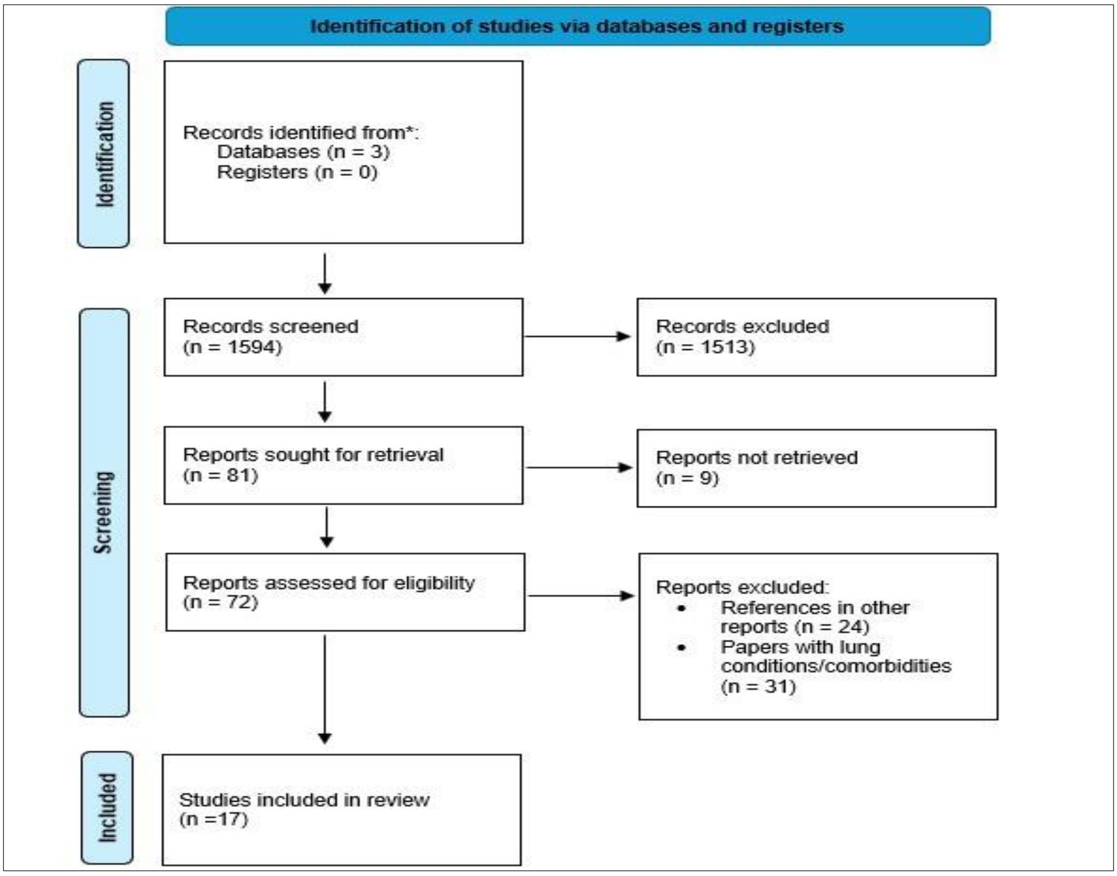

Figure 1: PRISMA chart.

The search plan comprised of using appropriate keywords along with the Boolean operators like "AND" as well as "OR" depending on the nature of the search. The primary keywords used were (heart OR cardiomyopathy) AND (heat OR stress OR environmental OR pollution OR climate).

Inclusion criterion

Articles included in the systematic review met all the following criteria: articles matched with the above keywords, from the listed databases; both prospective and retrospective cohort studies, as well as randomized and non-randomized controlled trials, were screened; case studies with human adult participants (≥18 years old) excluding individuals with pre-existing cardiovascular comorbidities and established risk factors; and articles in English language.

Exclusion criterion

Studies that didn't match the inclusion criteria or had any of the following: pay to access articles or articles outside the above-mentioned databases, and articles not written in English.

DISCUSSION

Cardiovascular disease (CVD) remains the leading cause of global mortality, with growing recognition that environmental stressors represent significant, yet often overlooked, contributors to disease pathogenesis. This systematic review synthesizes current evidence demonstrating that diverse environmental exposures, including pollution, noise, artificial light at night, temperature extremes, and psychosocial stress promote cardiovascular dysfunction through interconnected biological pathways. Three principal mechanisms emerge as central to environmental cardiovascular toxicity: oxidative damage through reactive oxygen species (ROS) generation, chronic low-grade inflammation mediated by cytokine release, and endothelial impairment via nitric oxide signaling disruption. These pathways collectively create a maladaptive physiological state that accelerates myocardial remodeling, reduces cardiac efficiency, and increases susceptibility to acute cardiovascular events. Notably, the cardiovascular effects of environmental stressors persist even after accounting for traditional risk factors such as hypertension, diabetes, and dyslipidemia, suggesting these exposures constitute independent determinants of cardiovascular health that warrant specific clinical and public health attention.

The cumulative impact of chronic environmental exposure presents particular concern for cardiovascular morbidity and mortality. Unlike acute exposures where physiological compensation may occur, prolonged contact with environmental stressors triggers cellular damage through multiple irreversible processes, including telomere shortening, mitochondrial dysfunction, and epigenetic modifications. This explains the strong dose-dependent relationships observed in longitudinal studies, such as the association between long-term PM2.5 exposure and left ventricular hypertrophy incidence.[2,3] Furthermore, emerging evidence suggests that combined exposures such as concurrent heat stress and air pollution may interact synergistically, following an exponential rather than additive risk model. For instance, during heat waves, the cardiovascular mortality risk associated with PM2.5 exposure increases disproportionately, highlighting the need to consider environmental exposures as interconnected rather than isolated risk factors.

Among specific environmental stressors, air pollution has been most extensively studied and demonstrates particularly robust cardiovascular effects. Fine particulate matter (PM2.5) exerts multisystem toxicity, with each 10 $\mu g/m^3$ increment associated with a 0.63 mmHg rise in systolic blood pressure (95% CI 0.42-0.84), a 0.7% increase in cardiovascular mortality, and a 12% higher risk of coronary events.[4,5] These effects occur through a well-characterized pathophysiological sequence: alveolar deposition of pollutants triggers local inflammation, leading to systemic cytokine release that induces endothelial nitric oxide synthase (eNOS) uncoupling. This critical molecular alteration reduces nitric oxide bioavailability by 40-60% while simultaneously increasing superoxide production, creating a pro-oxidant state that promotes vasoconstriction, platelet activation, and vascular inflammation. The clinical consequences manifest across the cardiovascular continuum, from subclinical endothelial dysfunction to acute myocardial infarction, with vulnerability observed among individuals with pre-existing cardiovascular conditions.

Temperature extremes represent another major environmental determinant of cardiovascular health, demonstrating bidirectional effects through distinct yet complementary pathways.[6] Heat stress imposes significant hemodynamic challenges, increasing blood viscosity by 15-20% through dehydration while simultaneously triggering heat shock protein-mediated inflammation (evidenced by 20-25% increases in C-reactive protein). These physiological changes, combined with autonomic imbalance (characterized by heart rate increases exceeding 20 bpm per °C above threshold temperatures), heighten the risk of arrhythmias, hypotension, and cardiovascular collapse.[7] Conversely, cold exposure induces dramatic peripheral vasoconstriction, elevating systemic vascular resistance by 30-40% and substantially increasing myocardial oxygen demand. Epidemiological studies reveal clear seasonal mortality patterns, with vulnerable populations particularly older adults, and those with coronary artery disease experiencing 3-5-fold higher event rates during temperature extremes.[8,9] The sympathetic hyperactivation characteristic of cold stress, marked by elevated catecholamine release, may provoke coronary vasospasm in susceptible individuals, contributing to the well-documented winter peak in acute myocardial infarctions.

Noise pollution and artificial light at night represent more recently recognized environmental cardiovascular risk factors that operate through neurohormonal activation and circadian disruption, respectively. Chronic noise exposure (>55 dB) maintains persistent activation of the hypothalamic-pituitary-adrenal (HPA) axis, sustaining cortisol levels 25-30% above baseline and promoting a proinflammatory vascular phenotype.[10] The resulting endothelial dysfunction, evidenced by 30% reductions in flow-mediated dilation in high-noise environments, accelerates atherogenesis and increases hypertension prevalence by 8-12 mmHg in affected populations. Similarly, artificial light at night suppresses melatonin secretion by 40-60% in urban populations, disrupting circadian regulation of autonomic tone and promoting metabolic dysfunction. The resultant increase in sympathetic activity, combined with sleep fragmentation and low-grade systemic inflammation, creates a maladaptive state that elevates cardiovascular risk across multiple domains.[11]

Psychosocial stress completes the spectrum of environmental cardiovascular risk factors, with chronic stress exposure inducing sustained HPA axis overactivation and amygdala-driven sympathetic stimulation.[12] The resulting endocrine dysregulation characterized by cortisol levels 40-50% above normal promotes insulin resistance (evidenced by HOMA-IR increases of 1.5-2.0), abdominal adiposity, and endothelial dysfunction. Simultaneously, persistent sympathetic overdrive increases arrhythmia susceptibility 2-3-fold while promoting left ventricular remodeling through chronic β-adrenergic stimulation. These pathways explain the well-established associations between psychosocial stress and incident cardiovascular events, particularly in high-stress occupational environments (Figure 2).

At the molecular level, environmental stressors converge on three fundamental pathological processes: oxidative stress, chronic inflammation, and endothelial dysfunction (Figure 3).[13] NADPH oxidase activation increases ROS production 3-5-fold, damaging cellular components while simultaneously quenching nitric oxide to form peroxynitritea potent oxidant that further exacerbates vascular injury.[14] Concurrent cytokine release (particularly IL-6 and TNF-α) creates a self-perpetuating inflammatory cascade, recruiting $Ly6C^+$ monocytes that differentiate into proinflammatory macrophages within atherosclerotic plaques. These cellular events occur alongside dramatic upregulation of adhesion molecules (ICAM-1 and VCAM-1 expression increases of 60-80%), promoting leukocyte infiltration and plaque instability.[15] The resultant prothrombotic shift, characterized by 40-50% higher tissue factor expression and impaired fibrinolysis, creates a vulnerable vascular environment predisposed to acute ischemic events.

Effective mitigation of environmental cardiovascular risk requires a multi-level approach addressing individual behaviors, community infrastructure, and policy interventions. Personal protective strategies demonstrate significant efficacy, with N95 mask use during high-pollution events reducing PM2.5 exposure by 60-80% and indoor HEPA filtration decreasing particulate concentrations by 45-50%.[16] Antioxidant supplementation, particularly vitamin E (400 IU/day), can attenuate oxidative stress markers by 25-30%, while personalized exposure monitoring through wearable technology enables targeted avoidance behaviors. Community-level interventions show comparable promise: urban green spaces (achieving 30% tree cover) lower ambient PM2.5 by 10-12%, while noise reduction initiatives and heat shelter networks reduce associated mortality by 15-20%.[17] Policy solutions remain essential for population-level protection, including stricter air quality standards (PM2.5 $\leq 8\ \mu g/m^3$ annual mean), climate-resilient urban planning, and targeted zoning regulations to protect vulnerable communities.

Health equity considerations must inform all intervention strategies, as socioeconomic disparities dramatically influence environmental exposure burdens and health outcomes. Marginalized populations experience 3-4-fold higher pollution exposures due to 30-40% greater PM2.5 concentrations in low-income neighborhoods, coupled with 50-60% reduced access to protective resources like air conditioning or quality healthcare (Figure 4).

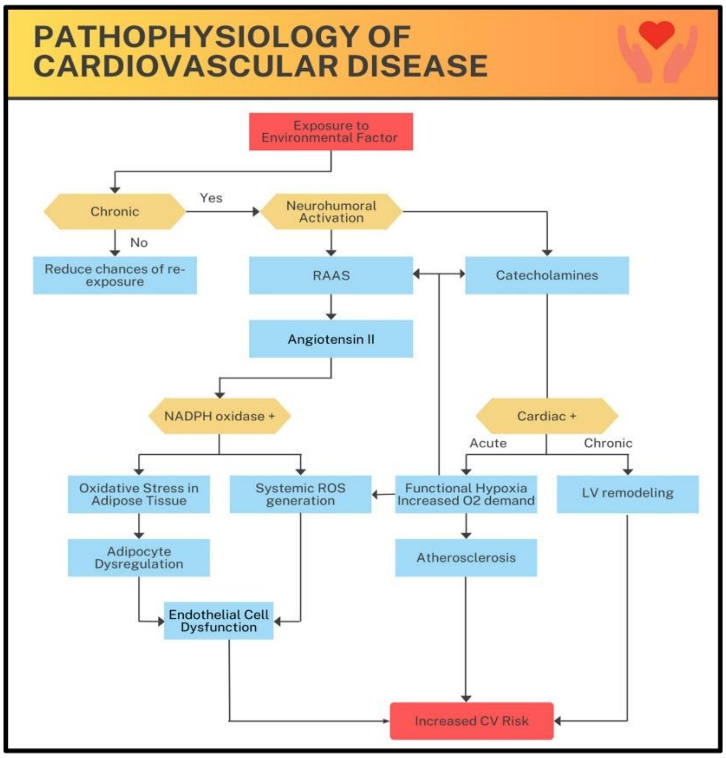

Figure 2: Pathophysiology.

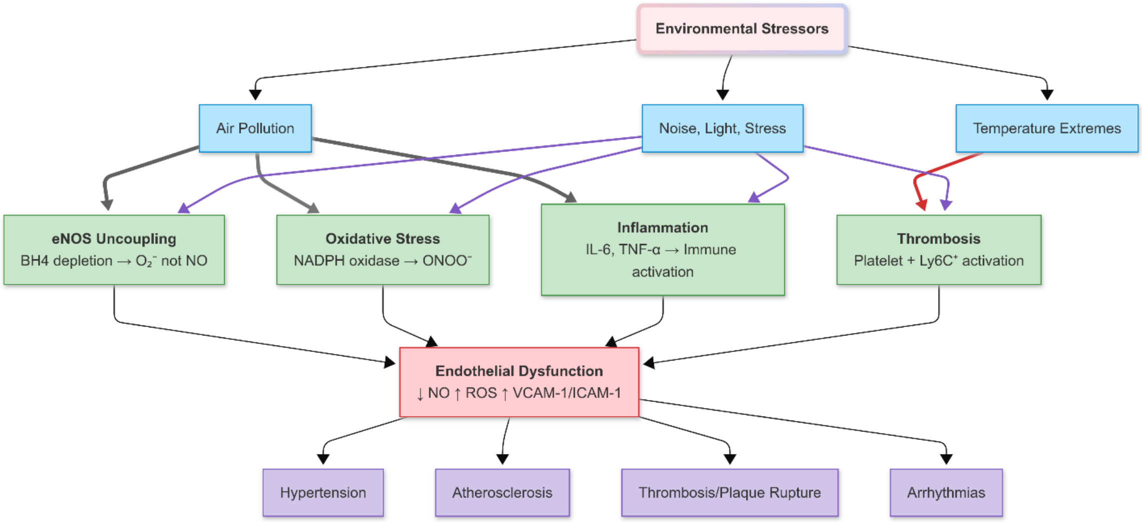

Figure 3: Molecular mechanisms.

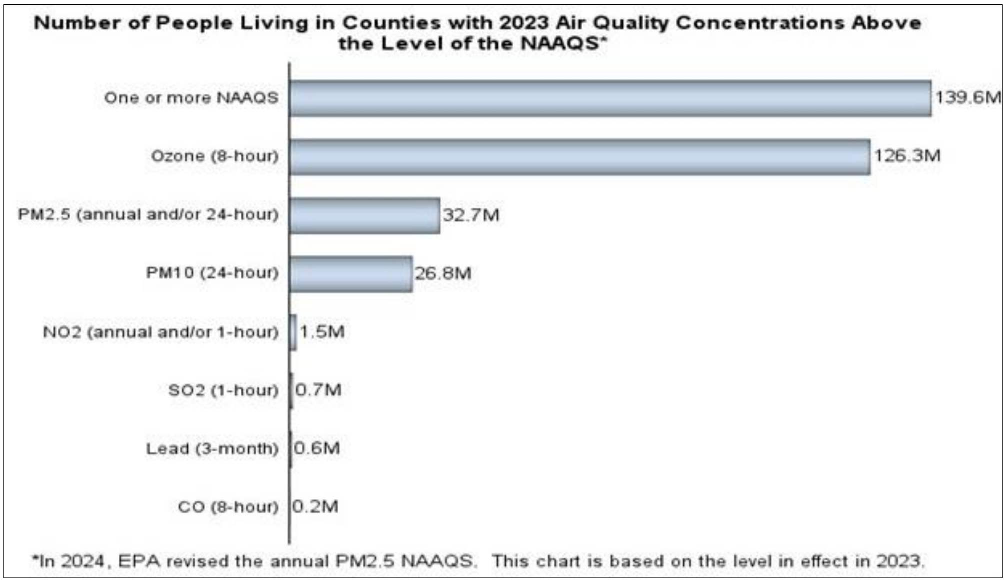

Figure 4: NAAQ indices.

These disparities manifest clinically during environmental crises, with disadvantaged communities showing 25-30% higher cardiovascular hospitalization rates during heat waves and wildfire smoke events. Recent analyses estimate that environmental factors account for 18-22% of the cardiovascular risk disparity between high- and low-income populations, underscoring the urgent need for targeted interventions.

Future research should prioritize development of environmental stress biomarkers, quantification of exposure interaction effects, and rigorous evaluation of intervention cost-effectiveness. Emerging techniques in exposomics and geospatial analysis offer promising tools for personalized risk assessment, while advances in biomonitoring may enable real-time evaluation of environmental cardiovascular toxicity. Concurrently, implementation of science must guide translation of evidence into policy, ensuring effective deployment of preventive strategies across diverse communities.

This comprehensive analysis establishes environmental stressors as major, modifiable determinants of cardiovascular health that operate through well-defined biological pathways.[18,19] The consistent demonstration of dose-response relationships, plausible mechanisms, and intervention efficacy meets established criteria for causal inference, suggesting environmental factors should be formally incorporated into cardiovascular risk assessment frameworks.[20] Clinicians can immediately begin addressing these risks through targeted screening questions about patient exposures and personalized prevention recommendations, while policymakers must prioritize environmental health measures as essential components of cardiovascular disease prevention. As climate change and urbanization intensify environmental stressors, proactive integration of environmental medicine into cardiovascular practice will be critical to mitigate the growing global burden of environment-related heart disease.

CONCLUSION

Environmental stressors significantly contribute to cardiovascular disease through oxidative stress, inflammation, and endothelial dysfunction. These exposures including air pollution, extreme temperatures, noise, and psychosocial stress act as independent risk factors, disproportionately affecting vulnerable populations. Effective mitigation requires a multi-level approach combining personal protective measures, urban planning strategies, and policy interventions. Clinicians should incorporate environmental risk assessments into cardiovascular care, while policymakers must strengthen regulations to reduce harmful exposures. Addressing these environmental determinants through collaborative efforts between healthcare, public health, and urban design sectors is crucial for reducing the global burden of cardiovascular disease. Future research should focus on exposure biomarkers and targeted prevention strategies to protect high-risk communities. Proactive measures integrating environmental health into cardiovascular prevention can significantly improve population health outcomes.

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REFERENCES

1. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;372:n71.
2. Bhatnagar A. Environmental determinants of cardiovascular disease. Circ Res. 2017;121:162-80.
3. Yang AM, Lo K, Zheng TZ, Yang JL, Bai YN, Feng YQ, et al. Environmental heavy metals and cardiovascular diseases: Status and future direction. Chronic Dis Transl Med. 2020;6(4):251-9.
4. Sagheer U, Al-Kindi S, Abohashem S, Phillips CT, Rana JS, Bhatnagar A, et al. Environmental Pollution and Cardiovascular Disease: Part 1 of 2: Air Pollution. JACC Adv. 2023;3(2):100805.
5. Basith S, Manavalan B, Shin TH, Park CB, Lee WS, Kim J, et al. The Impact of Fine Particulate Matter 2.5 on the Cardiovascular System: A Review of the Invisible Killer. Nanomaterials (Basel). 2022;12(15):2656.
6. Centers for Disease Control and Prevention. Clinical overview of heat and cardiovascular disease. CDC Heat and Health. 2024. Available at: https://www.cdc.gov/heat-health/hcp/clinical-overview/heat-and-people-with-cardiovascular-disease.html. Accessed on 24 May 2025.
7. Chen WT, Lin CH, Hsieh MH, Huang CY, Yeh JS. Stress-induced cardiomyopathy caused by heat stroke. Ann Emerg Med. 2012;60(1):63-6.
8. Desai Y, Khraishah H, Alahmad B. Heat and the heart. N Engl J Med. 2023;389:1-3.
9. American College of Cardioly. Cold weather exposure linked to increased risk of heart attacks. 2024. Available at: https://www.acc.org/About-ACC/Press-Releases/2024/09/02/10/31/Cold-Weather-Exposure. Accessed on 24 May 2025.
10. Kreibig SD. Emotion, motivation, and cardiovascular response. Wright RA, Gendolla GHE, Editors. How motivation affects cardiovascular response: Mechanisms and applications. American Psychological Association. 2012;93-117.
11. Münzel T, Hahad O, Sørensen M, Lelieveld J, Duerr GD, Nieuwenhuijsen M, et al. Environmental risk factors and cardiovascular diseases: a comprehensive expert review. Cardiovasc Res. 2022;118(14):2880-902.
12. Cairns M, Marais E, Joseph D, Essop MF. The Role of Chronic Stress in the Pathogenesis of Ischemic Heart Disease in Women. Compr Physiol. 2025;15(1):e70000.
13. Pendyala S, Natarajan V. Redox regulation of Nox proteins. Respir Physiol Neurobiol. 2010;174:265-71.
14. Fernandez-Marcos PJ, Nóbrega-Pereira S. NADPH: new oxygen for the ROS theory of aging. Aging (Albany NY). 2016;8:956-7.
15. Swirski FK, Libby P, Aikawa E, Alcaide P, Luscinskas FW, Weissleder R, et al. Ly-6Chi monocytes dominate hypercholesterolemia-associated monocytosis and give rise to macrophages in atheromata. J Clin Invest. 2007;117(1):195-205.
16. Patel V, Kantipudi N, Jones G, Upton A, Kamath MV. Air Pollution and Cardiovascular Disease: A Review. Crit Rev Biomed Eng. 2016;44(5):327-46.
17. Münzel T, Hahad O, Sørensen M, Lelieveld J, Duerr GD, Nieuwenhuijsen M, et al. Environmental risk factors and cardiovascular diseases: a comprehensive expert review. Cardiovasc Res. 2022;118(14):2880-902.
18. Alman BL, Pfister G, Hao H, Stowell J, Hu X, Liu Y, et al. The association of wildfire smoke with respiratory and cardiovascular emergency department visits in Colorado in 2012: a case crossover study. Environ Health. 2016;15(1):64.
19. DeFlorio-Barker S, Crooks J, Reyes J, Rappold AG. Cardiopulmonary Effects of Fine Particulate Matter Exposure among Older Adults, during Wildfire and Non-Wildfire Periods, in the United States 2008-2010. Environ Health Perspect. 2019;127(3):37006.
20. Chen H, Samet JM, Bromberg PA, Tong H. Cardiovascular health impacts of wildfire smoke exposure. Part Fibre Toxicol. 2021;18:2.

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