

The Impact Of Climate Change On Cardiovascular Health: A Review Of Pathophysiological Mechanisms, Thermal Extremes, And Social Vulnerability

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Abstract

Background: Climate change represents an escalating threat to global public health, with 2024 and 2025 recording historic temperature peaks.

Methods: This is a narrative/integrative literature review based on scientific statements and meta-analyses published between 2020 and 2025. This review analyses the impact of heat extremes on cardiovascular morbidity and mortality, focusing on vulnerable populations such as the elderly and those with pre-existing cardiovascular diseases (CVD).

Results and Conclusion: Thermal stress triggers a cascade of physiological responses, including systemic inflammation (elevated IL-6), dehydration, and a state of hypercoagulability that predisposes individuals to ischemic events and arrhythmias. Furthermore, the interaction between extreme heat and air pollution potentiates cardiac risks. Urban adaptation strategies and public health policies are urgent to mitigate a projected increase of up to 233% in heat-related cardiovascular deaths by 2036.

Keywords: climate change; cardiovascular health; extreme heat; social vulnerability

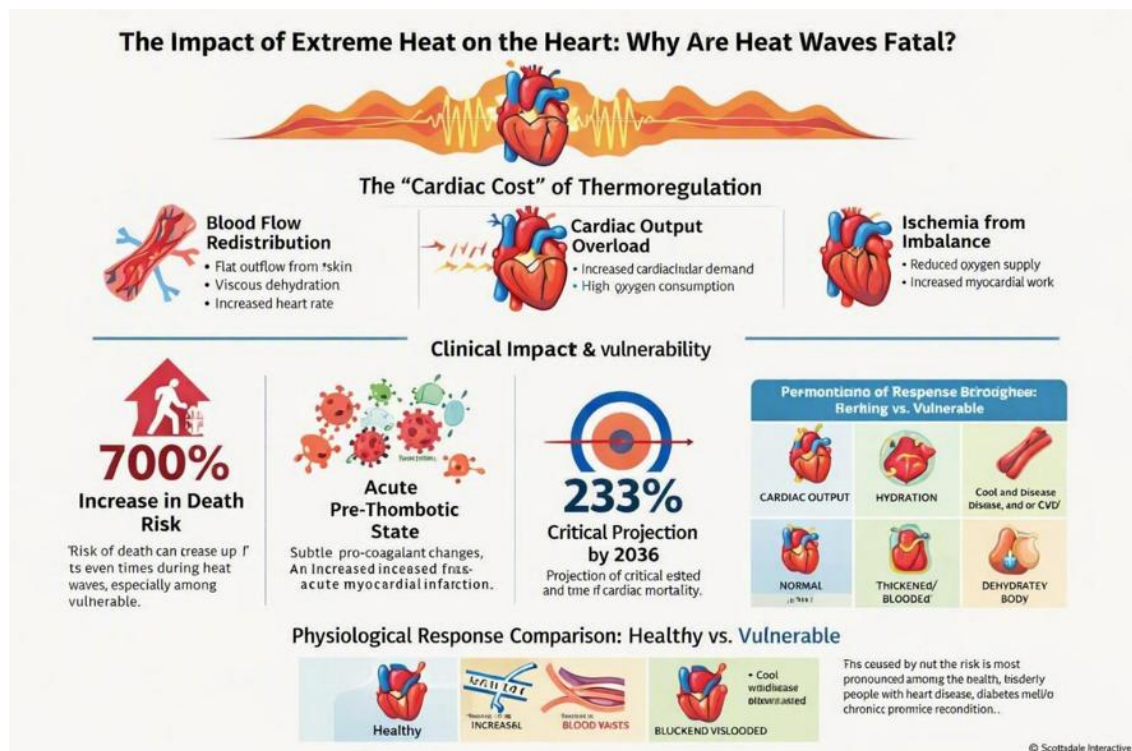
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I. Introduction

Global warming, driven by anthropogenic activities, has intensified the frequency and severity of heatwaves and extreme weather events. Epidemiological data demonstrate that, during thermal extremes, patients with cardiovascular disease (CVD) exhibit a mortality risk up to 700% higher than the general population. The introduction must address the concept of the exposome, which defines the totality of environmental exposures across a lifetime and how they interact with biological and social factors to determine cardiovascular health. The objective of this article is to review the mechanisms explaining why the heart is the organ most affected by thermal stress.

The sustained increase in global temperatures has transcended environmental alerts to solidify itself as a critical challenge for public health and clinical practice. The year 2024 established historic temperature records, and according to data from the Copernicus Climate Change Service, it is virtually certain that 2025 will surpass these milestones. In this scenario, cardiovascular physiology serves as the fundamental mechanistic link between epidemiology and bedside intervention. Understanding how thermal stress destabilizes homeostasis is not merely an academic exercise, but a strategic necessity to mitigate mortality in vulnerable populations, where the heart is frequently the first organ to fail under the burden of thermoregulatory demand. The infographic below summarizes the great impact of extreme heat and the cardiac cost of thermoregulation



Infographic: The impact of extreme heat on the heart: the "cardiac cost" of thermoregulation (AI-generated by the authors - NotebookLM).

II. Methodology

This is a narrative/integrative literature review based on scientific statements and meta-analyses published between 2020 and 2025. Primary sources include documents from the European Society of Cardiology (ESC) and the American Heart Association (AHA). The methodology describes the search for evidence regarding:

- Mechanisms of human thermoregulation.
- Epidemiological impact of historic heatwaves (e.g., Chicago 1995 and Europe 2022).
- Vulnerability factors (age, sex, race, and socioeconomic status).

III. Theoretical Framework

Epidemiological Context and Clinical Definitions

For risk assessment, we adopted the definitions established by the ESC Scientific Statement 2025:

- **Heat Extremes:** Temperatures exceeding the 95th percentile of the local seasonal average.
- **Heatwaves:** Periods of abnormally hot weather lasting at least 2 to 3 consecutive days.
- **Hyperthermia:** Elevation of core body temperature (up to ~39.5°C) with relative preservation of thermoregulatory defense mechanisms.
- **Heatstroke:** A catastrophic failure of thermoregulation (core temperature >40.5°C) accompanied by central nervous system dysfunction.

Epidemiologically, patients with CVD demonstrate a disproportionate vulnerability, experiencing up to a 700% increase in mortality risk during extreme thermal events. Literature shows that each 1°C increase above the local mortality threshold correlates with specific increases in relative risk (RR):

- **Stroke:** 3.8% increase in risk.
- **Acute Coronary Syndrome (ACS):** 3.5% increase in risk.
- **Cardiac Arrest (Overall):** 1.6% increase in risk.
- **Out-of-Hospital Cardiac Arrest:** 2.1% increase in risk.

Biometeorological Perspective ("So What?"):

The discrepancy observed between actual mortality and hospital admissions is alarming. The fact that the risk is significantly higher for out-of-hospital cardiac arrests indicates that many victims succumb before accessing emergency medical services. Consequently, hospital statistics severely underestimate the true burden of heatwaves, masking the urgent need for secondary prevention protocols and remote monitoring of chronic patients during summer months.

Thermoregulation and Hemodynamic Response to Heat

The dissipation of metabolic and environmental heat demands an integrated response of cutaneous vasodilation and sweating. Strategically, the body promotes a massive redistribution of blood flow from the core to the periphery to maximize convective and radiative heat loss. However, this cutaneous vasodilation drastically reduces total peripheral resistance.

To maintain mean arterial pressure (MAP), the cardiovascular system must increase cardiac output (CO). Notably, in an upright posture, stroke volume (SV) is, at best, barely maintained or frequently reduced due to peripheral venous pooling. Therefore, the increase in CO depends almost exclusively on marked escalations in heart rate (HR) and myocardial contractility. **Table 1** summarizes the physiological response to heat across different age groups and clinical profiles.

Table 1: Physiological Response to Heat: Healthy vs. Vulnerable

Physiological Parameter	Young & Healthy Individuals	Elderly (>60 years) & CVD Patients
Cutaneous Vasodilation	Rapid, high-magnitude response.	Blunted; reduced blood flow redistribution.
Sweat Rate	High; efficient evaporative cooling.	Reduced; increased core heat accumulation.
Stroke Volume	Maintained via compensatory mechanisms.	Frequently reduced; lower functional reserve.
Heart Rate	Proportional increase relative to demand.	Chronotropic limitation (age or medication).

The "Cardiac Cost" of Thermoregulation:

This hemodynamic effort imposes a severe "cardiac cost." In a previously compromised or aging heart, sustaining a high cardiac output against low peripheral resistance acts as an ischemic trigger. Ventricular failure occurs when the myocardium can no longer compensate for the drop in MAP, shifting the body's cooling attempt into cardiogenic shock or lethal arrhythmias.

Pathophysiology of Primary and Platelet Hemostasis

Hyperthermia acts as a direct modulator of platelet function, inducing a state of hyperaggregability even before the full clinical onset of heatstroke. *In vitro* and *in vivo* data reveal that exposure to temperatures up to 43°C irreversibly alters the platelet cytoskeleton; even after cooling, platelets retain an increased capacity for aggregation.

Heat Shock Proteins (HSPs) are the key molecular mediators of this response. Specifically, the HSP-20, HSP-40, HSP-70, and HSP-90 subfamilies regulate the stability and activation of platelet integrins under thermal stress. Evidence that aspirin prevents thermal hyperaggregability suggests that heat activates cyclooxygenase-dependent or integrin-signaling pathways that can be pharmacologically blunted.

Impact of Chronic Overexpression ("So What?"):

While HSPs are protective during isolated acute events, their chronic overexpression resulting from frequent heatwaves can accelerate atherogenesis. They act as autoantigens, triggering pro-inflammatory and autoimmune responses that compromise vascular integrity. Thus, thermal stress should not be viewed merely as an acute threat, but as a longitudinal accelerator of atherosclerotic cardiovascular disease.

Coagulation, Inflammation, and Endothelial Dysfunction

The progression from moderate hyperthermia to systemic cellular toxicity is marked by an inflammatory cascade in which Interleukin-6 (IL-6) serves as the central biomarker. Elevated IL-6 levels correlate directly with clinical severity and mortality, playing a key role in the upregulation of Tissue Factor (TF).

"Heat-Induced Coagulopathy" stems from excessive activation of the extrinsic coagulation pathway, leading to uncontrolled thrombin generation and fibrin deposition. Unchecked, this condition rapidly progresses to Disseminated Intravascular Coagulation (DIC) alongside the depletion of natural anticoagulants (Antithrombin, Protein C). The microvascular environment is further aggravated by the release of reactive oxygen species (ROS) and the formation of Neutrophil Extracellular Traps (NETs), rendering the endothelium highly pro-thrombotic.

The Role of Hemoconcentration:

As few as six hours of exposure to intense heat are sufficient to induce hemoconcentration and increased plasma viscosity. In patients with pre-existing microvascular impairment, this increase in viscosity—combined with endothelial activation—precipitates arterial and venous thrombotic events, even in the absence of critical coronary stenosis.

Myocardial Ischemia: Supply-Demand Mismatch

Heat-induced ischemia is essentially a metabolic supply-demand mismatch. In individuals with stable coronary artery disease (CAD), coronary flow reserve is severely restricted. Under heat stress, increased cardiac workload requires a proportionally higher blood supply that the myocardium cannot receive due to impaired coronary vasomotion.

Clinical evidence demonstrates detectable ischemia following brief exposures (such as Finnish saunas) or passive heating that raises core body temperature by merely 0.5°C. At the cellular level, thermal stress drives mitochondrial dysfunction and cardiomyocyte lipotoxicity, directly compromising myocardial energy efficiency.

Type 2 Myocardial Infarction and Arrhythmias:

Unlike classic Type 1 myocardial infarction caused by atherothrombotic plaque rupture, heat stress frequently precipitates Type 2 Myocardial Infarction, where ischemia results purely from a supply-demand mismatch in the setting of systemic stress. Clinicians must recognize that fatal arrhythmias and myocardial injury can occur during heatwaves without patients exhibiting the classic neurological symptoms of heatstroke.

Dehydration, Electrolytes, and Pharmacological Interference

Hypohydration is the primary amplifier of cardiovascular stress. A fluid loss exceeding 2% of body mass is sufficient to impair cutaneous vasodilation and reduce plasma volume, escalating core temperature and cardiac strain.

Electrolyte imbalances (Sodium, Potassium, and Magnesium) alter transmembrane action potentials, lowering the threshold for ventricular fibrillation and other lethal arrhythmias. Pharmacological management of CVD patients requires heightened vigilance:

- **Loop Diuretics:** Exacerbate volume depletion and electrolyte loss.
- **Beta-Blockers:** Although recent evidence suggests they do not significantly impair sweating in healthy populations, their chronotropic blunting limits heart rate acceleration—the main physiological mechanism for maintaining elevated cardiac output in extreme heat—thereby increasing the risk of syncope and hemodynamic collapse.

Plaque Rupture and the Sympathetic Nervous System:

Dehydration and thermal stress vigorously activate the Sympathetic Nervous System (SNS). This surge in adrenergic drive increases arterial wall shear stress, which can trigger the rupture of vulnerable atherosclerotic plaques, transforming a demand-driven stress (Type 2) into an acute thrombotic event (Type 1).

IV. Discussion

The discussion is structured around key foundational pillars:

1. **Pathophysiological Mechanisms:** Heat induces extreme cutaneous vasodilation to dissipate thermal energy, which markedly decreases peripheral resistance and demands a compensatory surge in cardiac output and heart rate. In diseased hearts, this exertion drives myocardial ischemia via oxygen supply-demand mismatch.
2. **Inflammation and Thrombosis:** Thermal exposure elevates key systemic markers such as IL-6, promotes platelet hyperaggregation, and causes endothelial dysfunction. Extreme temperatures can activate the extrinsic coagulation cascade, fostering a pro-thrombotic milieu.
3. **The Nocturnal Risk:** Elevated minimum nighttime temperatures prevent the human body from achieving nocturnal thermal recovery. This sustained stress is strongly linked to an increased risk of ischemic stroke, particularly among the elderly.
4. **Vulnerability and Environmental Injustice:** Risk distribution is profoundly unequal. Substandard housing, lack of urban canopy ("urban heat island" effect), and restricted access to cooling infrastructure (air conditioning) disproportionately exacerbate mortality rates in low-income communities.
5. **A Paradigm Shift:** Projections indicate that up to 42 thermal events comparable to the 2014 benchmarks will occur annually by the end of the century. Identifying thermal thresholds (such as 19°C and 25°C) translates empirical data into actionable clinical intelligence: clinicians must integrate biometeorological forecasts into their secondary prevention strategies. During heatwaves, differential diagnoses for ACS and arrhythmias in older adults should prioritize environmental triggers, prompting immediate adjustments to fluid intake,

electrolyte monitoring, and potential dosage modifications of cardiovascular medications to protect this growing, vulnerable population.

V. Conclusion

The cardiovascular burden imposed by climate change is preventable, yet it demands long-term strategy and proactive prevention plans. It is paramount that cardiologists integrate climate risk into clinical guidelines and patient counseling, particularly regarding the titration of medications like diuretics and beta-blockers during heat extremes. Ultimately, an interdisciplinary framework linking physiologists, bedside clinicians, and climate scientists is imperative to shield vulnerable populations.

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