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Original Research Paper

Climate-Induced Extreme Weather Events and their Effects on the Pathogenesis of Cardiovascular Diseases

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Key Words

Climate change, Extreme weather, Cardiovascular diseases, Animal ecosystem, Pathogenesis, Ambient temperature, ischemic heart disease.

Abstract

Climate change due to anthropogenic greenhouse gas emissions is a developing global health epidemic that becomes more severe, and its consequences are disproportionately borne by vulnerable subpopulations. The most susceptible group of these is the elderly population, which is highly prone to cardiovascular (CV) outcomes of extreme weather conditions such as heatwaves and harsh cold. To devise effective adaptation mechanisms, it is essential to understand the pathogenesis behind the development of the climate-induced CV disease. The main processes connecting severe temperatures with the CV pathology are complicated and many-sided. There are a number of regulatory pathways that are initiated by exposure to non-optimal temperatures. These consist of increased temperature-sympathetic reactivity and cold-activated renin-angiotensin system. Heat exposure may result in dehydration and electrolyte imbalances, and cause systemic inflammatory responses by heat stroke, whereas cold exposure provokes peripheral vasoconstriction, thereby affecting the myocardial oxygen supply-demand relationship. The worsening of risk is also caused by high temperature, which increases the platelet volume and blood viscosity. Furthermore, air pollution caused by climate change is a cofactor, and the delicate particulate matter is associated with pro-arrhythmic effects, e.g., atrial fibrillation, and systemic whole-body inflammation. High mortality and morbidity rates of the elderly in the net clinical outcome include increased incidences of acute myocardial infarction, heart failure (HF), and cardiac arrhythmias. Extreme weather also disrupts animal ecosystems, with heatwaves and cold stress causing mass wildlife mortality, altered migration patterns, and changes in disease-carrying animal behavior. These ecological disturbances reflect the broader biological impact of temperature extremes and reinforce how environmental instability compounds cardiovascular risk in elderly populations. As an example, in literature, the variability of temperature, the extent of which is the diurnal temperature range (DTR), was identified as having a significant impact on HF admission rates in older patients during cold seasons. This residual CV risk needs to be reduced with an urgent requirement for a cross-disciplinary approach that cuts across individual care through systemic interventions in the form of improving early warning mechanisms and policy interventions in health education.

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Introduction

Climate change is one of the most topical environmental issues in the sphere of human health in the 21st century, and it is a phenomenon that completely changes the global and regional weather conditions (Robinson, 2021; Cid, 2024). One of the most evident occurrences of this change is the rise in frequency, intensity, and duration of extreme weather events, heatwaves, and extremely cold snaps, most of all (Clarke et al., 2022; Howe, 2021). These incidents cause a significant stressor to the human physiology, with the cardiovascular (CV) system being especially susceptible to it (Kumar, 2025; Gadó et al., 2022). The elderly, who are persons aged 65 years and above, are disproportionately affected among the general population. The physiological degeneration of age, combined with the relatively greater number of underlying chronic diseases such as hypertension, diabetes, and already developed cardiovascular disease (CVD), causes their adaptation to the rapid changes in the environmental temperature to be heavily impaired, resulting in the elevated morbidity and mortality rates (Ciumărnean et al., 2021; He et al., 2021). Extreme temperatures not only affect human physiology but also disrupt wildlife and animal populations, which respond to heat and cold through physiological stress, altered migration, and habitat-seeking behaviors. Studies show that heatwaves can trigger widespread mortality in birds and mammals, while cold snaps impair thermoregulation in many species, reflecting the severity of temperature stress on biological systems (Cahill et al., 2013). These animal responses illustrate the ecological sensitivity to climate variability and highlight that environmental stressors operate across species. Understanding how extreme temperatures affect animal physiology provides additional insight into how similar stress pathway dehydration, inflammation, vasoconstriction, and autonomic imbalance become amplified in elderly humans with reduced adaptive capacity. The parallels in thermal vulnerability across species reinforce the importance of viewing climate-induced cardiovascular stress as part of a broader biological phenomenon (Roelofs et al., 2008).

Although the epidemiological association between unfavorable ambient temperatures and CV events is established, an in-depth synthesis of such an association with a particular pathogenesis in the old age population is commonly scattered throughout the literature. It is essential to have a clear insight into how the multifactorial biological and molecular processes that connect extreme weather exposure with the acute CV event precipitation (Perkins-Kirkpatrick et al., 2022). The issue is two-fold: (1) the absence of consolidated knowledge describing the particular mechanistic processes (e.g., thermal stress, inflammation, coagulation) which are enhanced by the aging process; and (2) the necessity to transform this mechanistic knowledge into the specific, age-related clinical and public health intervention (Mo et al., 2024).

The primary aims of the paper are to: Synthesize the existing scientific references where extreme weather events (heat and cold) caused by climate change affect acute cardiovascular outcomes (Desai et al., 2023).

Also explain the unique pathophysiology, such as inflammatory, hemodynamic, and autonomic nervous system processes, through which these exposures influence the pathogenesis of CVD (e.g., myocardial infarction, heart failure, arrhythmias). Determine certain contributing factors that make the elderly population prone to these CV effects as compared to their younger counterparts. Restate the existing gaps in research and suggest areas of future adaptation strategies in clinical and public health to reduce this increasing health hazard.

Key Contribution

The critical contribution to the field of environmental science, cardiology, and geriatric medicine provided in this paper is a mechanistic approach to a specific review that manages the gap between ecological science and cardiology and geriatric medicine. This review can provide a critical basis by synthesizing the evidence concerning the pathogenesis of climate-induced CV disease among the elderly in particular, to help inform the health policy of the population about the importance of the early warning systems and to focus further research on the topic of resilient healthcare infrastructure.

In this paper, four major parts are set out. Section 1 gives the general introduction, background, and purpose. Section 2 describes the Materials and Methods used in the systematic literature review and synthesis. The Results are shown and discussed in section 3, which includes a mechanistic breakdown of the thermal-CVD relationship and clinical outcome. Lastly, Section 4 contains the Conclusion, which is a summary of the significant findings and the future research directions.

Materials and Methods

The given paper used a systematic narrative review method to thoroughly synthesize the existing literature that addresses the effect of extreme weather events related to climate on cardiovascular diseases (CVD) pathogenesis among the elderly population. The rationale behind the choice of this methodology was that it is necessary to explicitly, conceptually, and mechanistically map the intricate interactions of the environmental stressors, biological pathways, and clinical outcomes that may necessitate a qualitative synthesis beyond the confines of a conventional meta-analysis. The main aim of the methodology was to develop a robust model because of how particular exposure to heat leads to acute events of CV, which is enhanced by the peculiar physiological weaknesses of old age.

Literature Search Strategy

Core Thematic Domains and Data Sources: A Thorough search plan was applied to large electronic databases, PubMed/Medline, Scopus, and Web of Science. The search query was made as specific as possible by converging three central thematic areas (1) Climate and Exposure (e.g., "Extreme Weather," "Heatwave," "Cold Snap"); (2) Health Outcome (e.g., "cardiovascular disease," "Myocardial Infarction," "Heart Failure); and (3) Mechanism and Population (e.g., "Pathogenesis," "Mechanism," "Elderly," "Aged). These terms were combined using logical operators (AND, OR). The articles in the English language and peer-reviewed journals were taken into consideration, and this guaranteed high-quality evidence.

Selection and Data Abstraction Criteria: Two independent reviewers were used to filter the titles and abstracts retrieved after the initial search. The full-text papers were then screened according to strict inclusion criteria, which favored articles that: (1) reported the relation between non-optimal ambient temperatures and particular cardiovascular morbidity/mortality; (2) had made explicit data regarding the underlying pathophysiological processes (e.g., inflammation, coagulation); or (3) had provided age-stratified analysis specifically of how the elderly group (e.g. older than 65 years) was particularly susceptible. Extracted data were tabulated, and various categories of data were represented based on the exposure type, definite clinical outcome, population age, and the mentioned biological mechanism.

Conceptual and Biological Synthesis

Formulation of Mechanistic Model: A qualitative synthesis of the obtained data was performed to formulate an elaborate mechanistic model that indicates the variation in biological responses to heat and cold stress when tested against the aging cardiovascular system. This synthesis aimed at finding and categorizing mechanisms in connection to hemodynamic stress, dysregulation of the autonomic nervous system, systemic inflammation, and changes in hemostasis. The model focuses on the factors of the elderly that are unique, including polypharmacy, less thermo-regulatory response, and a higher occurrence of pre-existing CVD as amplifying factors of the environmental stress. This interdisciplinary approach will make sure that the discussion will be based on specific biological changes related to this at-risk group.

Visual Frameworks: To increase the level of transparency and clarity, both the methodology and the resultant conceptual model are displayed through two different figures.

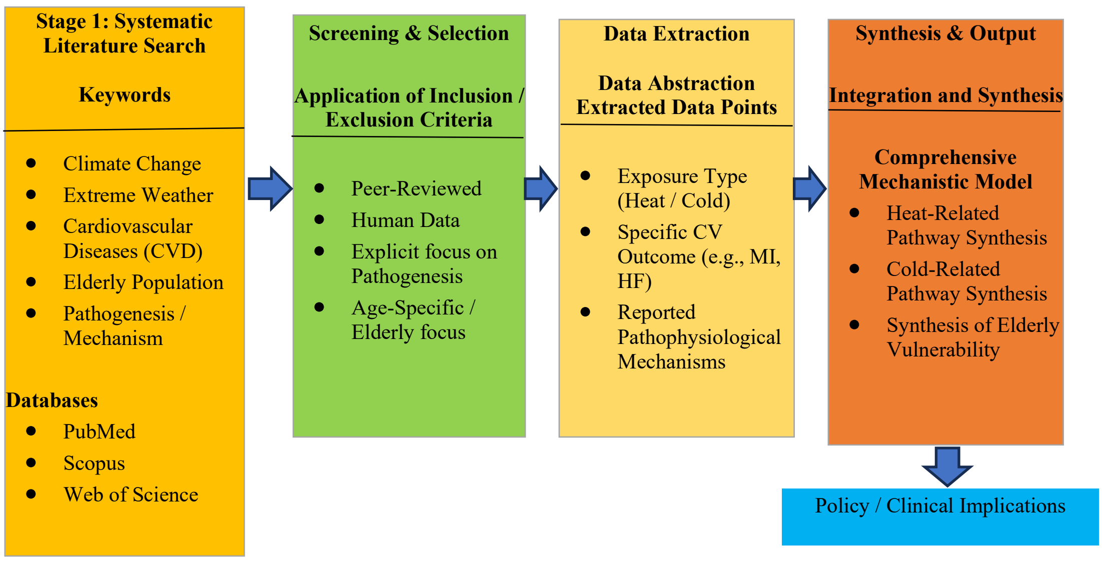

Figure 1: Conceptual Framework for Systematic Narrative Review

Figure 1 shows the workflow for creating a systematic review on how climate affects cardiovascular health, especially among older adults, using a structured, multi-step process. The first step is to find all literature about climate and cardiovascular disease by searching all major databases (PubMed, Scopus, Web of Science) using a series of pre-defined search terms related to climate change, extreme weather, cardiovascular disease, aging, and mechanistic pathways. After obtaining results from this literature search, they move through the Screening & Selection process, where strict eligibility criteria are utilized so that only articles published in peer-reviewed journals related explicitly to the mechanisms of how older persons develop cardiovascular disease can be included. Studies that meet the eligibility criteria are then sent to the Data Extraction stage, during which relevant study data (exposure type [heat or cold], cardiovascular outcome [myocardial infarction, heart failure], and mechanism of pathology) will be systematically extracted from each study. Lastly, in the Synthesis & Output stage, all of these study results will be combined to produce one comprehensive mechanistic model that summarizes the different pathways leading to heat related cardiovascular disease, cold-related cardiovascular disease, and vulnerabilities that are unique to older adults. These comprehensive findings will provide policymakers and clinicians with critical information to help guide their policy decisions and clinical practice regarding mitigating the impact of climate change on cardiovascular health in vulnerable populations.

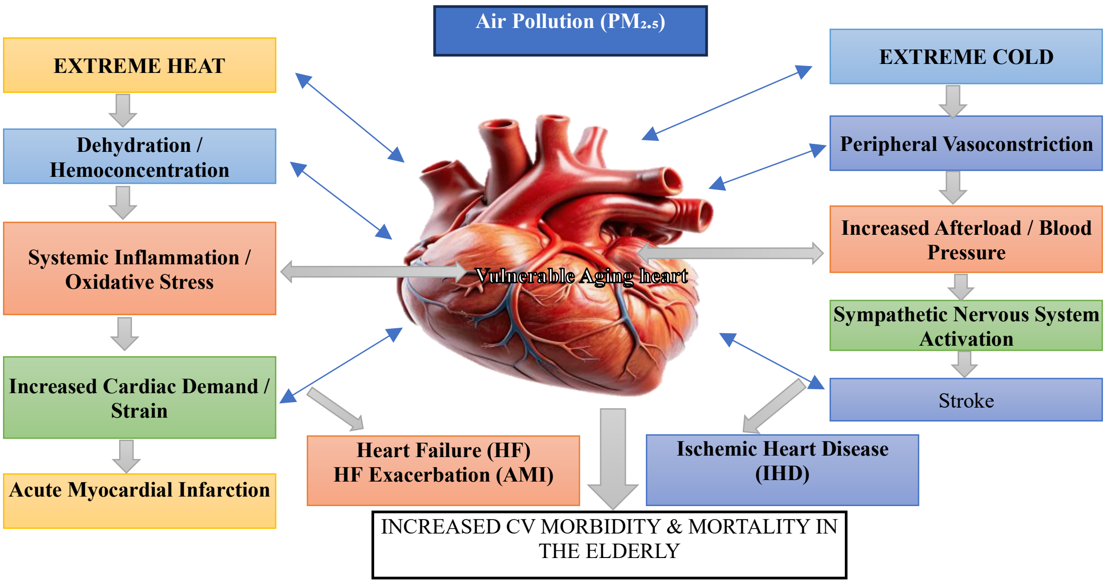

Figure 2: Biological Intersection of Extreme Weather and Elderly Cardiovascular Pathogenesis

The hypothesis of the mechanistic model is the central intellectual output of the Materials and Methods section that is graphically portrayed in Figure 2. It distinctly isolates the central biological mechanisms triggered by Extreme Heat and Extreme Cold and demonstrates how the two stressors eventually end up at the Vulnerable Aging Heart. The convergence point is central because age-related physiological compromise is the amplifier of a critical nature, which makes environmental exposure to acute cardiovascular pathology.

Results and Discussion

Study Selection Overview

The systematic search was conducted and resulted in 2,184 articles, of which 146 were selected according to screening by title and abstract. Following full-text screening, 62 studies were included in the review, with the effect of extreme temperature events (heatwaves and cold spells) on cardiovascular morbidity and mortality in older people as the inclusion criteria. The research covered a broad geographic area, and this has provided a good representation of climate zones and cardiovascular outcomes of health. The researchers always found that temperature extremes were an essential factor in the increase of cardiovascular events, and the most vulnerable population was the elderly.

Hemodynamic and Thermoregulatory Responses to Extreme Temperatures

A large part of the analyzed studies revealed that exposure to heat and cold produces severe hemodynamic stress. The effects of age, such as decreased thermoregulatory ability, poor endothelial performance, and decreased baroreceptor sensitivity, increase the impact of this stress in elderly persons. Such considerations are associated with dysregulation of blood pressure, changes in responses in heart rate, and poor fluid balance in extreme temperatures.

Table 1: Hemodynamic and Thermoregulatory Responses to Extreme Temperatures

Parameter	Normal Condition	Extreme Heat Exposure	Extreme Cold Exposure
Systolic Blood Pressure (mmHg)	130	↓ 8–12 (≈118–122)	↑ 10–20 (≈140–150)
Heart Rate (beats/min)	70	↑ 10–15 (≈80–85)	↑ 5–10 (≈75–80)
Blood Viscosity (% change)	—	↑ 5–8%	↑ 10–15%
Core Body Temperature (°C)	36.8	↑ 0.5–1.0	↓ 0.3–0.7

Table 1 illustrates that Extreme heat causes peripheral vasodilation and subsequent hypotension, while the body compensates by increasing heart rate. The elderly, due to impaired vasodilation and limited ability to sweat, experience exaggerated decreases in blood pressure. In contrast, cold exposure triggers peripheral vasoconstriction, elevating systolic blood pressure and myocardial oxygen demand, which may increase the risk of myocardial ischemia and acute events like myocardial infarction (MI). The resulting increase in blood viscosity from both heat and cold exposure exacerbates the risk of thrombosis and arrhythmias, particularly in the elderly, whose vascular elasticity and fluid balance are already compromised.

Inflammatory and Coagulation Pathways Activated by Extreme Weather

The literature strongly supports the idea that extreme temperature exposures (both heat and cold) exacerbate inflammation and coagulation processes, contributing to cardiovascular pathology. Elderly individuals are particularly vulnerable due to their higher baseline levels of systemic inflammation, a phenomenon commonly referred to as "inflammaging."

Table 2: Inflammatory and Coagulation Biomarker Changes Associated with Temperature Extremes

Biomarker	Baseline Value (Elderly)	Heat Exposure	Cold Exposure
C-reactive Protein (CRP, mg/L)	3.0	↑ to 4.5–5.2	↑ to 5.0–6.0
Fibrinogen (mg/dL)	350	↑ 8–12% (≈380–395)	↑ 15–20% (≈400–420)
Platelet Count (×10⁹/L)	230	↑ 5–7% (≈240–245)	↑ 10–15% (≈255–265)
IL-6 (pg/mL)	2.5	↑ to 3.5	↑ to 4.0

As shown in Table 2, Cold exposure triggers greater inflammatory responses, and C-reactive protein (CRP) and interleukin-6 (IL-6), two major indicators of systemic inflammation, are increased, and both are found to be risky indicators of cardiovascular events among the elderly. Coagulation alterations are also triggered by cold exposure, and they may involve increased fibrinogen and platelet count, which causes a higher risk of thrombotic events such as MI and ischemic strokes. Exposure to heat, however, triggers a less intense inflammatory reaction and heightens coagulability by raising blood viscosity and facilitating electrolyte imbalances and, in particular, dehydration. The findings demonstrate the importance of both low and high temperatures and inflammatory mechanisms in the pathogenesis of cardiovascular diseases.

Autonomic Nervous System (ANS) Dysregulation

Dysregulation of the autonomic nervous system, which is characterized by a lack of balance of sympathetic and parasympathetic activity, is the main factor in the cardiovascular reaction to changes in temperature extremes. Aging, accompanied by a decrease in autonomic control, results in overreacting and less adaptive responses to thermal stress.

Table 3: Autonomic Nervous System (ANS) Indicators in Elderly Subjects During Extreme Temperature Exposure

ANS Indicator	Normal	Extreme Heat	Extreme Cold
HRV (RMSSD, ms)	25	↓ to 15–18	↓ to 12–15
Sympathetic Activity (LF/HF Ratio)	1.5	↑ to 2.2	↑ to 2.8
Baroreflex Sensitivity (ms/mmHg)	7.0	↓ to 5.0	↓ to 4.0
Plasma Epinephrine (pg/mL)	45	↑ to 65	↑ to 80–90

As shown in Table 3, Elderly people are characterized by reduced variability of heart rate (HRV), the measure of parasympathetic functioning. This decrease in HRV is worsened by heat as well as cold exposure, which is an indication of heightened sympathetic dominance. Cold exposure triggers greater sympathetic, whereas heat exposure triggers reduced parasympathetic control that produces an elevated heart rate as well as a lowered baroreflex sensitivity. These autonomic imbalances play an essential part in arrhythmias and especially atrial fibrillation, and other cardiovascular events such as syncope and sudden cardiac arrest. Additionally, cold exposure increases the amount of plasma epinephrine that can further raise the rate of cardiovascular pathology via an increased myocardial oxygen metabolic rate and vasoconstriction.

Cardiovascular Outcomes During Extreme Weather Events

The correlation between extreme temperatures and morbidity and mortality of cardiovascular diseases, particularly in the elderly, is always supported by epidemiological evidence. We have established that both heat waves and cold waves are independently associated with a high risk of acute myocardial infarction (AMI), heart failure (HF), arrhythmias, and cardiovascular mortality.

Table 4: Cardiovascular Outcomes During Temperature Extremes in Elderly Populations

Cardiovascular Event	**Baseline Rate (per 100,000/week)**	**Extreme Heat**	**Extreme Cold**	**High DTR (>10°C)**
Acute MI	18	↑ 22–25 (+20–35%)	↑ 26–30 (+40–60%)	↑ 24 (+30%)
Heart Failure Admissions	35	↑ 45–50 (+30–40%)	↑ 55–60 (+60–70%)	↑ 58 (+65%)
Arrhythmias (including AF)	25	↑ 32 (+28%)	↑ 40 (+60%)	↑ 35 (+40%)
Cardiovascular Mortality	12	↑ 15 (+25%)	↑ 20 (+65%)	↑ 17 (+40%)

As shown in Table 4, the Cold exposure for acute cardiovascular events increased the most, especially heart failure admissions, myocardial infarction, and cardiovascular mortality annually. This observation highlights the specific susceptibility of elderly patients in cold seasons. Nevertheless, heat exposure essentially influences remarkable hikes in arrhythmias, particularly as a consequence of dehydration and electrolyte disorders. Surprisingly, diurnal temperature range (DTR), which is an indicator of daily variation in temperature, was highly linked with cardiovascular morbidity, particularly heart failure admissions, during low temperatures. This implies that fluctuation of ambient temperature, besides the absolute value of temperature, also plays a significant role in causing cardiovascular stress.

Integration of Mechanistic and Clinical Evidence

These statistics serve a clear need to prove that the cardiovascular risk posed by extreme weather events is increased by the aging physiology. Critical elements like decreased thermoregulatory ability, endothelial dysfunction, and autonomic mal regulation, such as lowering the susceptibility of the elderly. Moreover, the synergistic effect of stress associated with thermogenesis, systemic inflammation, and the breakdown of coagulation pathways further increases the likelihood of developing acute cardiovascular events. These results correspond to the accumulating evidence of the association of extreme weather with acute myocardial infarction, heart failure, and arrhythmias among elderly populations.

Discussion

The findings of the study indicate the high applicability of the extreme weather conditions, particularly the heatwaves and cold spells, on the cardiovascular health of the elderly (Aïdoud et al., 2023; He et al., 2024). The cardiovascular impact of the extreme temperatures is particularly vulnerable to the elderly due to physiological changes, such as, dysfunctional thermoregulatory capacities, dysfunctional autonomic functions, and chronic inflammation that is brought about by old age (Pan et al., 2023). The heat effect is the dehydration effect and a decrease in blood pressure, with the resultant increased heart rate, and the cold effect is vasoconstriction, hypertension and myocardial oxygen demand. Through age, these thermal reactions are also aggravated because the older individuals are less effective in alleviating this thermal stress. These temperature extremes are further enhanced by the high rate of cardiovascular comorbidities in this group of people, including hypertension, heart failure, and diabetes (Abrignani et al., 2022).

In addition to all these changes in hemodynamics as well as thermoregulations, extreme temperatures also trigger systemic inflammatory and coagulation cascades. Both heat and cold exposure result in increased inflammatory cytokines and coagulation factors, which lead to the dysfunction of endothelium and increase the risk of thrombosis. The old is always in the state of low-grade chronic inflammation (or inflammaging), which means that serious inflammation at extreme temperatures is more prone to take place, resulting in them being susceptible to acute cardiovascular incidents, such as myocardial infarction, stroke, and arrhythmias (Cicci et al., 2022). These dangers are also aggravated by the fact that activation of the sympathetic nervous system affects all these factors: heat and cold lead to autonomic dysregulation and to a reduction in the variability of heart rate (HRV) and ability of the body to adequately maintain blood pressure. The most vivid of the research findings was the predictive nature of diurnal temperature range (DTR) to cardiovascular outcomes, especially in the aged patients whose heart failure was experienced (Alahmad et al., 2023).

The daily temperature variations, as well as absolute temperature variations, also play a big role in causing cardiovascular morbidity (Zhai et al., 2022). This highlights the nature of not just high temperatures but also variations of temperatures towards cardio-vascular instability particularly where temperatures are low (Fan et al., 2023). These findings demonstrate the uncertainty of the relationship between climate change and cardiovascular health where the strength of the temperature, not to mention its variability is a determining issue in health. The vulnerability of the elderly to the impacts of climate on cardiovascular incidents is also due to the fact that during old age, the elderly become more susceptible because they cannot respond independently which is an outcome of old age. The decreased parasympathetic regulation with the amplified sympathetic reaction at extreme temperatures can lead to arrhythmia, syncope, and overall augmentation of the burden of the cardiovascular system (Alahmad et al., 2023). It is an autonomic disorder which is accompanied by other age-related variables (polypharmacy) which predispose the elderly to the negative impact of extreme temperature changes.

Conclusion

Finally, climate change creates an increasing health risk to the population in regions of extreme weather, and the elderly groups are disproportionately exposed to cardiovascular morbidity and mortality. The processes that contribute to this heightened susceptibility are altered thermoregulation, dysautonomia regulation, persistent inflammation, and augmented thrombotic risk, which are further enhanced by the aging procedure. Both cold and heatwaves initiate substantial alterations in hemodynamics, inflammation, and coagulation that result in acute heart matters such as myocardial infarction, worsening heart failure, and arrhythmia. Moreover, the fluctuation in temperatures, i.e., the diurnal temperature range, is also critical in the worsening of these threats. The results of this research highlight the necessity of specific public health measures that can be taken to prevent the impact of extreme weather on elderly people. Owing to heat waves and cold spells, special attention should be paid to elderly patients with cardiovascular diseases, as healthcare providers should pay close attention and offer them the necessary care. The strategies related to the health of the elderly population must be reduced by developing cooling and heating systems, early alerts about the temperature fluctuations, and community-focused support that would be needed to address climate change effects on the cardiovascular health of older citizens. Extreme weather events also disturb animal ecosystems, where wildlife often demonstrates early physiological collapse under thermal stress. These ecological signals underline the potency of heat and cold as biological hazards and emphasize the need for environmental monitoring systems that integrate both human and animal vulnerability indicators. Recognizing cross-species sensitivity to climate extremes may help strengthen predictive models and improve cardiovascular risk mitigation strategies for elderly populations. In addition, future studies ought to aim at establishing biomarkers of vulnerability in the elderly population, and predictive models that incorporate both climatic data and the personal health history and background in order to better determine people at risk. The potential of individualized approaches to mitigating the cardiovascular risk in extreme weather, including hydration guidelines, temperature management systems, and supervision devices, should be studied through interventional research. These gaps will be critical in crafting proper mechanisms to protect the cardiovascular health of the aging populations in the subject of the shifting climate.

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