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Heart failure at the crossroads of climate change and environmental health: emerging risks, mechanistic insights, and future directions

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Abstract

Climate change is increasingly recognized as a major determinant of cardiovascular health, yet its impact on heart failure (HF) remains underexplored. Extreme temperatures, air pollution, and climate-related disasters represent critical stressors that can exacerbate HF incidence, hospitalizations, and mortality. In this scoped narrative review, we summarize and critically discuss evidence linking environmental exposures to HF outcomes, while distinguishing established epidemiological associations from mechanistic hypotheses and emerging areas of clinical translation. Pathophysiological mechanisms potentially involved include oxidative stress, systemic inflammation, hemodynamic instability, and autonomic imbalance, with disproportionate effects on vulnerable populations such as older adults, women, and socioeconomically disadvantaged groups. Recent evidence demonstrates that particulate matter, ground-level ozone, and wildfire smoke contribute to worsening HF outcomes, while extreme weather events disrupt healthcare delivery and continuity of care. This review summarizes the current evidence linking climate change and environmental exposures with HF, identifies key knowledge gaps, and highlights opportunities for prevention and adaptation strategies. A collaborative approach involving clinicians, researchers, public health systems, and policymakers is urgently needed to mitigate these risks and ensure equitable cardiovascular health in the era of climate change.

Key words: heart failure, climate change, air pollution, extreme heat, environmental health.

Introduction

Cardiovascular disease remains a leading cause of morbidity and mortality worldwide, accounting for more than 18 million deaths each year, while heart failure (HF) represents one of its most disabling and resource-intensive clinical manifestations [1]. While traditional risk factors such as hypertension, ischemic heart disease, and diabetes are well established, growing evidence suggests that climate change and environmental exposures are emerging and underappreciated drivers of cardiovascular disease. Extreme temperatures, ground-level ozone, wildfire smoke, and hurricanes have been consistently associated with increased cardiovascular morbidity and mortality [2,3]. Air pollution, particularly PM2.5 and nitrogen dioxide, has been strongly linked to both the incidence of HF and the exacerbation of existing disease [4]. Higher levels of PM10 and carbon monoxide have also been shown to significantly increase the risk of HF readmissions in patients with unstable angina, with disproportionate effects among women with fewer comorbidities [5]. The cardiovascular impact of climate change also extends through indirect pathways, including the disruption of healthcare systems during extreme weather events, the psychological burden of environmental crises, and the long-term effects of food insecurity [6]. These adverse outcomes are not distributed equally, with older adults, women, low-income communities, and populations in low- and middle-income countries bearing a disproportionate burden, thereby amplifying existing health inequities. In this review, we explore the intersection between HF and climate change, focusing on mechanisms, vulnerable populations, and strategies for adaptation. Our aim is to highlight HF as a sentinel condition for understanding how environmental stressors threaten cardiovascular health and to emphasize the urgent need for multidisciplinary action.

A structured literature search was performed in PubMed/MEDLINE, Scopus, and Web of Science from January 2000 to December 2024. Search terms were combined using Boolean operators and included “heart failure”, “climate change”, “extreme temperature”, “heatwave”, “cold exposure”, “air pollution”, “PM2.5”, “PM10”, “nitrogen dioxide”, “ozone”, “wildfire smoke”, “dust storm”, “environmental exposure”, and “cardiovascular outcomes”. We prioritized meta-analyses, systematic reviews, large cohort studies, time-series and case-crossover designs, and key mechanistic/translational studies when clinically relevant. Given the broad and interdisciplinary nature of the topic, study selection followed a structured narrative approach, prioritizing high-quality epidemiological, clinical, and mechanistic evidence. This framework was intended to integrate heterogeneous data across environmental health and HF research, while maintaining caution in causal interpretation where evidence remains indirect. Inclusion criteria were studies reporting HF incidence, hospitalization, decompensation, or mortality in relation to environmental exposures, or studies providing plausible mechanistic pathways linking climate-related stressors to HF instability. Exclusion

criteria included non-English articles, studies without cardiovascular outcomes, and reports lacking quantitative exposure assessment. Reference lists of key reviews and selected articles were also screened to identify additional relevant publications.

Climate-driven environmental exposures and heart failure

Extreme heat and cold

Exposure to extreme temperatures represents one of the most powerful environmental stressors affecting cardiovascular health, and HF appears to be among the most temperature-sensitive conditions. Large-scale epidemiological evidence demonstrates a clear U- or J-shaped association between ambient temperature and cardiovascular mortality, with excess risk during both periods of extreme heat and extreme cold [7]. Extreme temperatures impose acute physiological strain that may overwhelm thermoregulatory and cardiovascular compensatory mechanisms, particularly in patients with pre-existing HF whose reserve is limited [8].

At the global level, extreme heat and cold contribute significantly to cause-specific mortality from HF, with cold exposure showing the strongest impact. In a dataset including more than 3.6 million HF deaths across 27 countries, extreme hot days accounted for 2.6 excess HF deaths per 1000 HF deaths, whereas extreme cold days accounted for 12.8 excess HF deaths per 1000 HF deaths, underscoring the disproportionate burden of cold-related mortality [7]. These findings align with physiological evidence showing that heat and cold trigger opposing but equally destabilizing responses: heat leads to peripheral vasodilation, volume depletion, tachycardia, and increased metabolic demand, while cold induces vasoconstriction, increased afterload, hemoconcentration, and heightened sympathetic activation [8,9].

Heat-related cardiovascular risk is expected to rise as global temperatures increase, with extreme heatwaves becoming more frequent, more intense, and longer-lasting. Rising ambient temperatures increase cardiovascular strain through dehydration, hypotension, and autonomic imbalance, and these effects are amplified in individuals with chronic cardiovascular disease, multimorbidity, or polypharmacy [10]. The physiological load of heat may also interact with other climate-driven hazards—such as wildfires, air pollution, and water scarcity—further compounding cardiovascular vulnerability [11].

Conversely, the burden of cold-related HF events remains substantial. Cold exposure increases systemic vascular resistance, elevates blood pressure, and promotes a pro-thrombotic and inflammatory state, all of which can precipitate HF decompensation. Nationwide data from Spain confirm that cold temperatures exert one of the highest relative risks for HF hospitalization and mortality, with extreme cold associated with a relative risk of 1.537 for HF admissions and particularly pronounced effects among older adults and vulnerable groups [12]. These observations support the concept that temperature sensitivity reflects both direct

physiological stress and social or infrastructural factors, including access to heating, insulation, and socioeconomic resources [12,13].

Geographical variations further modify temperature-related HF risk. Regions unaccustomed to extreme climatic conditions—either heat or cold—experience disproportionate increases in HF events when such extremes occur. This reflects differences in population-level adaptation, climatic baseline, housing quality, urbanization, and availability of cooling or heating resources. Both heatwaves and cold spells therefore represent high-risk environmental exposures for HF patients, and their amplified frequency under current climate trajectories raises substantial public-health concerns [11,14].

Air pollution

Air pollution is a pervasive environmental threat and one of the most powerful non-traditional drivers of cardiovascular disease, with HF emerging as one of its most vulnerable clinical expressions [15]. Exposure to particulate matter—especially fine and ultrafine particles ($PM_{2.5}$, PM_{10})—as well as nitrogen dioxide (NO_2) and ozone (O_3), has consistently been associated with increased cardiovascular morbidity and mortality across multiple epidemiological and experimental models [16]. Even small daily increments in pollutant concentrations are followed within hours or days by measurable increases in HF hospitalizations, arrhythmic events, acute coronary syndromes, and cardiovascular deaths, highlighting a steep, short-lag response curve [17].

Long-term exposure carries an even greater burden, accelerating the development of HF, amplifying the risk of recurrent hospitalizations, and worsening outcomes in both preserved and reduced ejection fraction phenotypes [18]. Prolonged inhalation of $PM_{2.5}$ promotes subclinical and overt atherosclerosis, endothelial dysfunction, autonomic imbalance, oxidative stress, and systemic inflammation, creating a biological milieu that heightens cardiovascular vulnerability across the lifespan [19]. These chronic pathways explain why individuals living in highly polluted areas exhibit faster progression of structural heart disease and higher rates of HF-related mortality, even after adjusting for traditional risk factors [20].

Pollutant exposure also appears to destabilize ischemic heart disease. In patients with established coronary syndromes, transient increases in $PM_{2.5}$, PM_{10}, NO_2 or CO are associated with myocardial ischemia, recurrent angina, and higher rates of HF readmissions, further illustrating the continuum between air pollution, coronary vulnerability, and HF decompensation [5]. These effects are magnified in those with multimorbidity, older adults, socioeconomically disadvantaged populations, and communities living in proximity to major traffic corridors, underscoring profound environmental inequities [16].

Biological plausibility is strongly supported by mechanistic research. Inhaled particles trigger pulmonary oxidative stress and inflammation, with downstream systemic propagation affecting vascular tone, thrombotic balance, metabolic regulation, and autonomic stability [15]. Fine particles penetrate deeply into the respiratory tract, and ultrafine species may translocate into the circulation or central nervous system, disrupting vascular and myocardial integrity through sustained oxidative injury and inflammatory activation [18,21]. These processes can precipitate acute HF decompensation, particularly in vulnerable individuals exposed to concurrent triggers such as heat, infection, or physical strain [20,22].

Natural experiments during the COVID-19 lockdowns provided additional evidence: sharp reductions in $PM_{2.5}$ and NO_2 were paralleled by significant short-term declines in cardiovascular events, supporting the potential reversibility of some pollution-related cardiovascular risks [23].

Wildfires, smoke composition and emerging cardiovascular risks

Wildfires represent one of the most rapidly expanding environmental cardiology threats, magnified by rising temperatures, prolonged droughts, and widespread ecosystem aridification. Beyond their immediate destructive potential, these events generate complex aerosols enriched in $PM_{2.5}$, ultrafine particles, CO, NO_2, O_3, black carbon, aldehydes, PAHs, volatile organics, and secondary oxidants—an atmospheric mixture substantially more toxic and pro-inflammatory than typical urban pollution, largely due to the chemical heterogeneity of wildfire smoke [24,25]. As smoke plumes travel hundreds or thousands of kilometers, entire populations distant from burn areas experience acute spikes in exposure, often coinciding with extreme heat, low humidity, and stagnation—conditions that amplify cardiovascular stress [26,27].

Recent data provide compelling evidence of a multi-hazard scenario particularly dangerous for patients with heart failure. In Mediterranean climates, peaks in wildfire-$PM_{2.5}$, O_3, and CO during the hottest months correspond to measurable increases in circulatory and respiratory mortality, with risk rising sharply when thermal stress and pollutant accumulation co-occur [28]. This combined heat–pollution environment drives systemic inflammation, endothelial dysfunction, autonomic imbalance, blood pressure surges, and heightened myocardial oxygen demand—mechanisms well recognized in HF decompensation [26]. Controlled human exposure studies further show that even short, standardized inhalation of wood-smoke surrogates can acutely increase arterial stiffness, oxidative stress markers, and pro-inflammatory cytokines [25], reinforcing the plausibility of these mechanistic pathways. A major conceptual advance has come from the systematic review and meta-analysis on fuel types. Evidence now demonstrates that what burns fundamentally modifies cardiovascular

toxicity. Fires involving structures or peat—rich in metals, toxic organics, plastics, and chlorinated compounds—produce significantly higher risks for all cardiovascular ED visits (RR 1.08), dysrhythmia (RR 1.10), and hypertensive crises compared with fires involving only natural vegetation [29]. These results suggest that smoke chemical composition, more than smoke quantity alone, shapes cardiovascular hazard. Peat fires in particular generate persistent smoldering emissions with high concentrations of toxic combustion by-products and prolonged $PM_{2.5}$ release, consistent with the elevated PAH and aldehyde yields described in combustion analyses [30]. Additional epidemiological signals refine this risk profile. North American analyses show robust associations between wildfire-specific $PM_{2.5}$ and emergency visits for asthma, COPD, bronchitis, and mixed respiratory illness, with a less consistent but biologically plausible relationship for acute cardiovascular events [31]. The respiratory burden—through hypoxia, inflammation, infection susceptibility, and autonomic shifts—acts as an upstream trigger for HF destabilization even when direct cardiovascular associations appear modest in short-term models [24,32].

Importantly, wildfire smoke penetrates indoor environments far more efficiently than previously recognized. Real-world infiltration studies show that indoor $PM_{2.5}$ levels frequently reach 50–75% of outdoor concentrations during smoke episodes, making standard "stay indoors" advice insufficient unless combined with filtration [33]. A complementary but increasingly relevant threat is the rise of dust storms in arid regions. Climate-driven desertification, soil drying, and atmospheric instability have increased the frequency and intensity of dust events in several continents, including North America.

Dust storms elevate coarse $PM_{2.5-10}$, biological particles, metals, and pathogens, and are associated with meaningful same-day increases in ICU admissions, particularly for respiratory presentations—but also for overall critical care utilization [34]. While mechanistically distinct from wildfire aerosol toxicity, dust storms share convergent pathways that can destabilize vulnerable cardiac patients: oxidative stress, acute bronchoconstriction, systemic inflammation, and autonomic imbalance.

Pathophysiological pathways linking climate and environmental stressors to heart failure

The following pathways should be interpreted as biologically plausible and increasingly supported by epidemiological and translational evidence, although their relative contribution may vary according to exposure type, HF phenotype, comorbidity burden, and individual vulnerability.

Hemodynamic stress, autonomic imbalance, and volume/pressure overload

Acute heat exposure induces a stereotyped pattern of cardiovascular stress that is particularly hazardous in heart failure. A recent meta-analysis of more than 400 laboratory heat-exposure studies (6,858 participant-exposures) demonstrated that even moderate heat loads produce a median rise in core temperature of approximately 0.9°C, accompanied by an average heart rate increase of approximately 27 beats/min and consistent elevations in cardiac output and rate–pressure product, reflecting increased cardiac workload. These effects are more pronounced under encapsulated heating conditions (e.g., protective clothing), closely mirroring real-world occupational and urban heat exposure during heat waves [35]. In healthy individuals, thermoregulation depends on a substantial increase in skin blood flow and cardiac output. Cutaneous circulation may rise from 5–10% to as much as 50–70% of resting cardiac output, and total cardiac output can nearly double during whole-body heating [36,37]. This compensatory response, however, comes at the cost of tachycardia, increased myocardial oxygen demand, and a delicate balance between peripheral vasodilation and maintenance of arterial pressure [37]. Exercise-induced heat stress further amplifies this pattern. In young healthy men, intermittent work in the heat increased indices of myocardial work and reduced estimated coronary perfusion compared with normothermic exercise, despite unchanged aortic stiffness—suggesting a relative mismatch between oxygen supply and demand even in structurally normal hearts [38]. In chronic heart failure, this physiological "stress test" becomes intrinsically maladaptive. In a controlled whole-body heating protocol, patients with stable NYHA class II–III HF exhibited a comparable rise in core temperature and preserved sweating, yet their increase in cutaneous vascular conductance was reduced by approximately 50% compared with matched controls, and maximal vasodilator capacity was markedly blunted [39]. Subsequent investigations have confirmed that HF patients display a limited capacity to augment cardiac output, impaired cutaneous vasodilation, and exaggerated neurohormonal activation during heat stress—features that collectively narrow the margin between compensation and decompensation [37]. To preserve arterial pressure with a relatively fixed cardiac output, these patients must restrict skin perfusion and rely more heavily on tachycardia and sympathetic activation, at the expense of effective heat dissipation and coronary perfusion. Contemporary models therefore conceptualize classic heat stroke largely as a cardiovascular failure of thermoregulation: when an already compromised heart cannot sustain the combined demands of peripheral vasodilation, intravascular volume shifts, and increased metabolic load, circulatory collapse, multiorgan dysfunction, and death may ensue [40].

Lung-heart interactions: bronchoconstriction, hypoxia, infections, pulmonary congestion

Wildfire smoke represents a uniquely potent respiratory toxin, characterized by elevated concentrations of $PM_{2.5}$, reactive gases, polycyclic aromatics, and combustion by-products capable of overwhelming host airway defenses. Evidence indicates that wildfire-specific $PM_{2.5}$ produces greater respiratory morbidity than ambient urban pollution, with disproportionate increases in asthma, COPD exacerbations, and acute bronchitic episodes during major fire events [41,42]. Mechanistically, these particles induce neutrophilic airway inflammation, epithelial barrier disruption, oxidative injury, impaired mucociliary clearance, and macrophage dysfunction, thereby increasing susceptibility to infection and accelerating loss of ventilatory reserve [42]. Severe smoke inhalation may further cause chemical airway injury, surfactant dysfunction, atelectasis, and systemic hypoxia due to CO and cyanide exposure, rapidly precipitating respiratory failure [43]. These pulmonary insults have direct cardiopulmonary consequences: hypoxemia, bronchoconstriction, and inflammation elevate pulmonary vascular resistance, increase right ventricular afterload, and aggravate pre-existing congestion in HF patients, lowering the threshold for acute decompensation. Importantly, wildfire exposure rarely occurs in isolation. Population-level analyses from California demonstrate supradditive effects of concurrent extreme heat and wildfire-related $PM_{2.5}$, with approximately 8% of respiratory hospitalizations and 5–6% of cardiovascular and renal hospitalizations attributable to this interaction within short exposure windows [44]. This multi-hazard scenario is particularly concerning in heart failure, where heat-induced tachycardia and vasodilation intersect with smoke-induced hypoxia and inflammation, creating a highly unstable hemodynamic environment. More broadly, air pollution—including bushfire smoke—is consistently associated with increased cardiovascular morbidity and mortality across HF, ischemic heart disease, arrhythmias, and stroke [45]. Collectively, these data support a unifying concept: acute respiratory compromise—whether driven by smoke inhalation, hypoxemia, inflammation, or infection—acts as an upstream trigger capable of destabilizing cardiopulmonary homeostasis, particularly in HF phenotypes with limited pulmonary reserve or pre-existing congestion.

Systemic inflammation, thrombosis, endothelial dysfunction and microvascular injury

Exposure to fine and ultrafine particulate matter generates a systemic inflammatory response that begins within minutes at the pulmonary level and rapidly extends to the vascular compartment, creating a biological environment particularly hazardous for patients with heart failure. $PM_{2.5}$ and $PM_{0.1}$ penetrate deeply into the alveoli and may reach the circulation, transporting surface-bound metals, organic radicals, and reactive quinones that amplify

oxidative and nitrosative stress across the cardiovascular system [19,46]. This oxidative burden depletes antioxidant defenses, uncouples endothelial nitric oxide synthase, enhances superoxide formation, and promotes peroxynitrite generation—thereby impairing vasodilation, damaging endothelial structures, and accelerating vascular stiffness and microvascular rarefaction [47].

Systemic inflammation is central to this process. Both acute and chronic exposure to $PM_{2.5}$ increases circulating C-reactive protein, fibrinogen, IL-6, adhesion molecules, and multiple cytokines, reflecting an immediate pulmonary inflammatory reflex followed by secondary systemic propagation [19]. Activation of the NLRP3 inflammasome further amplifies caspase-1 activity, IL-1β release, and macrophage polarization toward a pro-inflammatory M1 phenotype, fostering a persistent proatherogenic and prothrombotic milieu. Ultrafine particles phagocytosed by macrophages may induce "frustrated phagocytosis," generating sustained oxidative bursts and perpetuating endothelial injury [48]. Endothelial dysfunction represents a critical convergence point between air pollution and cardiovascular risk. Both gaseous pollutants (NO_2, O_3, CO) and particulate fractions reduce nitric oxide bioavailability, disrupt endothelial barrier integrity, and enhance leukocyte adhesion and transendothelial migration [47]. A meta-analysis of controlled human exposure studies demonstrated that each 10 μg/m³ increase in short-term $PM_{2.5}$ exposure raises circulating ICAM-1 and VCAM-1 levels by 1.55% and 1.97%, respectively—biomarkers of endothelial activation and vascular inflammation [49].

These alterations facilitate monocyte recruitment, foam cell formation, and plaque destabilization, compromising microvascular integrity. Thrombotic pathways are similarly activated. $PM_{2.5}$ exposure enhances platelet activation, tissue factor expression, and procoagulant protein release, shifting the hemostatic balance toward thrombosis. Epidemiologic evidence consistently links pollution peaks to acute coronary syndromes, ischemic stroke, venous thromboembolism, and cardiovascular mortality, with stronger effects in older adults and individuals with pre-existing cardiovascular disease [50]. This prothrombotic phenotype arises from the interplay of endothelial dysfunction, oxidative stress, autonomic imbalance, and systemic inflammation. Microvascular injury is an additional, often underrecognized mechanism of pollution-related cardiovascular harm. Chronic $PM_{2.5}$ exposure promotes capillary rarefaction, impairs coronary microcirculatory reserve, and induces diffuse microvascular inflammation and remodeling—mechanisms particularly relevant to HFpEF and patients with multimorbidity [51]. Due to their small size and high surface reactivity, ultrafine particles can disrupt endothelial tight junctions and potentiate localized oxidative injury within the microcirculation [47].

Vulnerable phenotypes (HFpEF, right HF, CKD, elderly, etc.)

The impact of climate and environmental stressors on heart failure varies substantially across patient subgroups. Older adults are consistently among the most vulnerable.

In a nationwide HF cohort, long-term exposure to $PM_{2.5}$, PM_{10}, SO_2, and CO was associated with increased HF readmissions in patients aged 65 years ($PM_{2.5}$ HR 1.15; PM_{10} HR 1.12; SO_2 HR 1.81; CO HR 3.60), whereas no significant associations were observed in younger individuals [52]. Similarly, in India, acute decompensated HF admissions and mortality peaked during the coldest months, with strong inverse correlations between ambient temperature and HF events ($r = –0.78$ for admissions; $r = –0.65$ for mortality), particularly among elderly patients [53]. These findings likely reflect age-related reductions in autonomic, vascular, and thermoregulatory reserve.

Geographic and neighborhood context further modulate vulnerability. In China, pollution-related cardiovascular risk was observed predominantly in South China—where outdoor exposure is greater—while no clear associations emerged in North China, where indoor confinement and reduced ventilation may limit exposure [52]. At the neighborhood level, analyses of more than 6,800 HFpEF patients demonstrated that individuals residing in low-income, high-exposure urban environments exhibited higher BNP levels, greater left ventricular mass index, more frequent renal dysfunction, and significantly worse long-term survival. Air pollution, ozone, environmental noise, lead levels, and water contaminants clustered within these disadvantaged areas, reflecting the convergence of environmental and social risk [54]. Chronic kidney disease (CKD) further amplifies environmental susceptibility. Long-term $PM_{2.5}$ exposure has been associated with increased CKD incidence (HR approximately 1.13–1.14), and co-exposure to high $PM_{2.5}$ concentrations and elevated temperature demonstrated a synergistic effect (synergy index 3.18) [55].

Mechanistically, particulate matter accelerates glomerulosclerosis, tubular fibrosis, oxidative stress, renin–angiotensin–aldosterone system activation, and endothelial injury. In HF patients with concomitant CKD, reduced renal reserve magnifies the destabilizing effects of pollution peaks, dehydration, temperature extremes, and autonomic imbalance. HFpEF represents another highly environment-sensitive phenotype. Characterized by systemic inflammation, microvascular dysfunction, and multimorbidity—including obesity, diabetes, and CKD, all of which are worsened by air pollution and heat—HFpEF patients living in polluted or socioeconomically deprived neighborhoods demonstrate more severe congestion biomarkers, adverse remodeling, and poorer survival [54].

Environmental triggers such as heat waves, $PM_{2.5}$ and ozone spikes, noise exposure, and respiratory infections frequently precipitate decompensation in this subgroup. Patients with

right-sided HF or concomitant pulmonary disease, including COPD, are particularly susceptible to air pollutants, which promote bronchial inflammation, hypoxemia, and increased pulmonary vascular tone. Even modest elevations in pulmonary vascular resistance may overwhelm limited right ventricular reserve, leading to rapid clinical deterioration.

In patients with HF–COPD overlap, cold spells, smog events, and respiratory infections commonly precipitate exacerbations that secondarily trigger HF decompensation [56,57]. Finally, environmental stress interacts closely with social determinants of health. Elderly HF patients residing in poorly insulated housing, lacking air conditioning or adequate heating, or living in high-pollution, low-resource neighborhoods often have limited capacity to mitigate exposure during heat waves, pollution peaks, or cold spells. These structural constraints contribute to higher readmission rates and mortality, defining an increasingly relevant "environmentally vulnerable HF phenotype" [54,58].

Table 1 summarizes the principal environmental exposures, their mechanistic pathways, and their clinical consequences in HF, underscoring the multifactorial nature of climate- and pollution-related vulnerability.

Epidemiology of heart failure outcomes in a changing climate

A large spatiotemporal Bayesian analysis from Canada, encompassing more than 1.2 million patients and 922,132 HF episodes over a decade, showed that environmental variables can improve the prediction of weekly fluctuations in HF hospitalizations. Models incorporating climatic signals outperformed demographic-only approaches, achieving correlations exceeding 0.96 between predicted and observed events [59].

A pronounced seasonal cyclicity emerged, with HF admissions peaking during colder periods and declining consistently in warmer months, underscoring climate as a powerful modulator of population-level hemodynamic stability. Notably, environmental sensitivity was strongly age-dependent: individuals 70 years exhibited the greatest number of significant climatic predictors and the steepest exposure–response gradients, confirming the disproportionate vulnerability of older adults. Global mortality analyses further contextualize the magnitude of temperature-related risk. Non-optimal temperatures account for approximately 1.17 million cardiovascular deaths annually worldwide. In one of the largest multinational temperature–mortality assessments, extreme cold (1st percentile) was associated with a 37% increase in HF mortality, whereas extreme heat (99th percentile) increased mortality by 12% [7]. HF demonstrated steeper risk curves than ischemic heart disease or stroke, suggesting that volume-sensitive and autonomic-sensitive phenotypes are particularly susceptible to thermal stress. Cold exposure exhibited a prolonged "memory effect," with excess risk persisting for up to 14 days, whereas heat produced a more immediate but shorter-lived increase in events. Prolonged

heatwaves (5 days) disproportionately affected frail older adults, while cold spells exerted greater impact in temperate regions characterized by suboptimal housing insulation and limited heating infrastructure [60]. Air pollution constitutes an additional—and often concurrent—determinant of HF burden. A comprehensive meta-analysis demonstrated that long-term $PM_{2.5}$ exposure increases HF incidence by approximately 20% and HF mortality by 9% per 10 μg/m³ increment, with consistent associations observed for PM_{10} and NO_2 [61]. Short-term pollutant elevations exert even more acute effects: increases in $PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3 are associated with rises in HF emergency visits and hospitalizations within hours to days. Notably, HF admissions often increase more sharply during pollution peaks than other cardiovascular outcomes, suggesting impaired compensatory reserve and heightened cardiorespiratory strain in this population [4].

Pollution-related exacerbations appear particularly severe in older individuals and in patients with HFpEF, right ventricular dysfunction, or chronic kidney disease—phenotypes characterized by amplified susceptibility to oxidative stress, endothelial injury, and systemic inflammation. Importantly, environmental stressors rarely occur in isolation. Combined exposures—such as elevated temperature with high particulate burden, extreme cold with NO_2 accumulation, or wildfire events generating simultaneous heat and smoke—produce synergistic and disproportionate risk. This “climate penalty” has been observed across multiple settings. In California, days characterized by concurrent extreme heat and elevated $PM_{2.5}$ were associated with excess mortality exceeding 20%, surpassing the additive effects of each exposure alone [1]. European multicenter analyses similarly demonstrated that ozone-related cardiovascular mortality increased substantially during hot days, reflecting enhanced oxidative reactivity and deeper lower-airway pollutant penetration [62]. These findings indicate that HF patients experience compounded physiologic strain when climatic and pollution stressors coincide—a pattern likely to intensify under ongoing climate instability. Data from low- and middle-income countries (LMICs) highlight a critical global dimension. Despite bearing the highest pollution burdens and substantial temperature variability, LMICs remain underrepresented in the literature, with approximately 72% of published studies originating from China alone. Available evidence nevertheless demonstrates meaningful associations: short-term $PM_{2.5}$ exposure increases cardiovascular morbidity and mortality by approximately 0.6–0.7% per 10 μg/m³, while long-term exposure increases morbidity by 13% and mortality by 9% [63]. In urban India, winter months were associated with the highest rates of HF admissions and mortality, and maximum ambient temperature emerged as the strongest inverse predictor of acute decompensation [64]. Collectively, these data indicate that heart failure epidemiology is measurably shaped by climate-related environmental factors, including

ambient temperature, air pollution, and broader atmospheric dynamics. Older adults remain particularly susceptible, while regional adaptation, socioeconomic context, housing characteristics, and indoor–outdoor exposure patterns further modulate risk [11]. Emerging evidence suggests that environmental signals could contribute to short-term forecasting of HF events, supporting their potential future integration into risk surveillance and population-level preparedness strategies, although dedicated validation studies are needed before such approaches can be translated into routine clinical or organizational practice. Figure 1 synthesizes the interaction between climate change, environmental exposures, epidemiological patterns, and pathophysiological mechanisms relevant to heart failure.

Clinical perspectives and future directions for risk stratification in heart failure patients

Integrating climate and pollution variables into individualized heart failure risk prediction

Traditional HF risk stratification models rely predominantly on clinical and biochemical variables—age, renal function, natriuretic peptides, blood pressure, comorbidities, imaging parameters—and assume relative environmental stability. However, growing evidence indicates that environmental stressors function as dynamic modifiers of short-term HF risk [65-68].

Integrating climate and pollution variables into predictive frameworks could therefore improve temporal precision and contextual relevance, although this approach remains investigational and requires dedicated validation before routine clinical implementation.

Recent population-level analyses demonstrate near-continuous exposure–response relationships between ambient particulate matter and cardiovascular outcomes, even below current regulatory thresholds [69]. These findings support the inclusion of environmental exposure as a quantitative risk gradient rather than a binary variable. The exposome framework offers a structured approach to capturing cumulative and interacting environmental influences—including pollution burden, thermal variability, noise exposure, and socioeconomic context—alongside traditional clinical predictors [54,66]. In HF populations, geocoded environmental metrics have been associated with distinct clinical phenotypes and differential outcomes, particularly in socioeconomically disadvantaged neighborhoods [54].

Technological advances now permit near–real time exposure characterization. High-density sensor networks, satellite-derived particulate estimates, wildfire-smoke modeling, and IoT-enabled meteorological systems provide continuously updated environmental data streams that may be linked to clinical datasets [70]. These platforms create the possibility of dynamic risk modeling, in which short-term forecasts of temperature extremes or pollution peaks inform anticipatory monitoring strategies. Rather than static baseline scores, exposure-adjusted models could identify transient “high-risk windows” during which HF patients are more

susceptible to decompensation. Emerging multi-omics approaches further suggest that environmental susceptibility may be biologically stratifiable. Metabolomic, transcriptomic, and methylomic signatures associated with pollutant exposure may help identify patients with heightened inflammatory or oxidative responsiveness to environmental triggers [71,72].
Such markers, if validated, could complement traditional biomarkers and refine personalized risk assessment. Importantly, environmental variables should be conceptualized as risk modifiers that interact with established clinical substrates rather than as independent standalone predictors. Their integration into HF risk prediction models may improve short-term forecasting accuracy, enhance allocation of preventive resources, and support the development of context-aware cardiovascular care strategies in an era of increasing climatic variability [65-69].

Practical management in daily practice

Translating environmental evidence into day-to-day HF care requires an anticipatory, exposure-informed approach, while recognizing that most proposed interventions are supported by indirect evidence, expert opinion, or extrapolation from broader cardiovascular and environmental-health literature rather than dedicated HF trials.
Periods of extreme heat, cold spells, poor air quality, or wildfire smoke intrusion should be considered transient high-risk windows for HF destabilization, prompting reinforced counseling, closer monitoring, and attention to medication safety in vulnerable patients [69,73]. Patient-level recommendations focus on exposure reduction and early detection of decompensation. During high pollution or smoke days, patients should limit outdoor activities and avoid exertion, prioritize cleaner indoor environments, and follow air-quality alerts. Since a substantial proportion of exposure occurs indoors, portable HEPA air cleaners can meaningfully reduce indoor PM burden and represent one of the most practical mitigation tools during pollution peaks and wildfire episodes [33,74-77]. When outdoor exposure cannot be avoided during wildfire smoke events, the use of high-filtration respirators may reduce inhalational dose [78]. Environmental stressors interact with HF pharmacotherapy. Heat and dehydration may reduce tolerance to diuretics, vasodilators, and renin–angiotensin system inhibitors, while beta-blockers can limit chronotropic compensation during thermal stress. In selected high-risk patients (older adults, HFpEF, CKD), clinicians should consider temporary dose adjustments, reinforce hydration advice, and intensify monitoring of weight, blood pressure, renal function, and symptoms during extreme conditions [11,78-80]. Remote monitoring programs and telemedicine can support earlier recognition of clinical deterioration and reduce avoidable admissions during environmentally unstable periods [79]. Finally, clinicians should recognize that environmental crises cluster with sleep disruption,

psychological stress, and respiratory infections—common upstream triggers for HF decompensation. Maintaining a low threshold for evaluating sleep, mood, and respiratory symptoms during prolonged heat/pollution or wildfire periods is appropriate, particularly in frail and multimorbid patients [11].

Conclusions

Climate change and environmental degradation are emerging as major—yet still under-recognized—determinants of HF incidence, decompensation, and mortality. Extreme temperatures, air pollution, wildfire smoke, dust storms, and climate-amplified respiratory stressors converge through shared biological pathways—hemodynamic strain, autonomic imbalance, lung–heart interactions, systemic inflammation, and endothelial and microvascular injury—to destabilize already fragile cardiovascular systems. Epidemiological evidence consistently identifies HF as one of the most temperature-sensitive and pollution-sensitive cardiovascular phenotypes, with particularly elevated risks during cold spells, heatwaves, and peaks in fine particulate matter. Older adults, patients with HFpEF, right-sided HF, chronic kidney disease, multimorbidity, and those living in socioeconomically deprived, high-exposure environments bear a disproportionate burden. These insights carry important clinical and public-health implications, although several proposed strategies remain evidence-informed rather than trial-proven in HF-specific populations. Environmental exposures can no longer be considered merely as background context; they may usefully inform HF risk stratification, counseling, and follow-up. Exposure-informed self-management, individualized review of diuretics and vasodilators during thermal extremes, optimization of indoor air quality, and remote monitoring during high-risk periods represent promising strategies that warrant prospective evaluation.

Health systems must likewise anticipate climate-sensitive surges in HF events, ensure continuity of care during heatwaves, cold spells, and wildfires, and prioritize protection of the most vulnerable populations. Important research gaps remain. Robust data from low- and middle-income countries, deeper characterization of environmentally vulnerable HF phenotypes, mechanistic studies linking exposure-omics signatures to clinical trajectories, and pragmatic trials testing mitigation strategies—such as air filtration, tailored pharmacologic adaptation, and digital early-warning systems—are urgently needed. Integration of high-resolution environmental data into HF prediction models may enable proactive resource allocation and personalized alerts. Cardiology cannot remain isolated from broader mitigation efforts. Reducing air pollution and greenhouse gas emissions, promoting climate-resilient urban planning and housing, and embedding environmental justice into health policy are integral to HF prevention in the 21st century.

Recognizing HF as a sentinel condition of climate-related cardiovascular vulnerability offers a strategic opportunity: by aligning clinical care, public health, and environmental policy, we may curb the growing burden of climate-sensitive HF and advance more equitable cardiovascular health in a rapidly changing world.

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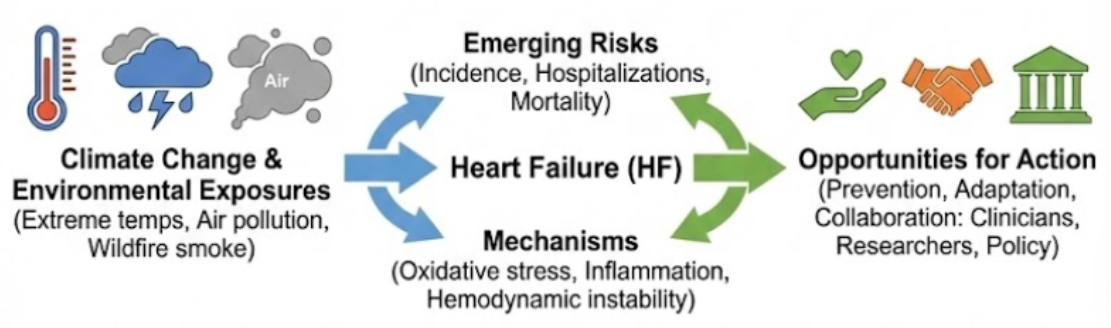

Figure 1. Climate change, environmental exposures, and heart failure at the crossroads.

Table 1. Environmental stressors, mechanisms, and clinical relevance in heart failure

Environmental stressor	Main mechanisms in HF	Clinical relevance	Key refs
Temperature extremes (heatwaves, cold spells)	Hemodynamic stress, autonomic imbalance, volume/pressure overload	Peaks in HF hospitalizations and mortality, especially in older adults	Alahmad 2023; Ni 2025; Achebak 2024
Ambient air pollution ($PM_{2.5}$, PM_{10}, NO_2, O_3, CO)	Pulmonary inflammation, oxidative stress, endothelial dysfunction, thrombosis	Higher HF incidence, readmissions, arrhythmias, CV death	Bhatnagar 2022; Montone 2023; Zhang 2023; Ward-Caviness 2023
Wildfire smoke and dust storms	Lung–heart interactions, hypoxia, bronchoconstriction, systemic inflammation	Short-term surges in cardiorespiratory ED visits and HF decompensation	Williams 2024; Duarte 2023; Stowell 2019; Rublee 2020
Climate-related system disruption (extreme events, heat + pollution)	Interrupted care, medication non-adherence, compounded physiologic stress	Excess HF events during multi-hazard episodes and disasters	Münzel 2024; Khraishah 2023; Bernstein 2021
Socioenvironmental vulnerability (low SES, poor housing, high-exposure neighborhoods)	Reduced adaptive capacity, cumulative exposome burden	Disproportionate HF morbidity and mortality in disadvantaged groups	Liem 2024; Gyaase 2025

HF, heart failure; CV, cardiovascular; PM, particulate matter;NO_2, nitrogen dioxide; O_3, ozone; CO, carbon monoxide; ED, emergency department; SES, socioeconomic status.