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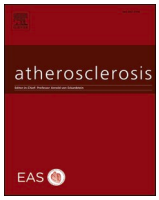

Low and high air temperature and cardiovascular risk

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ABSTRACT

Temperature extremes are one facet of global warming caused by climate change. They have a broad impact on population health globally. Due to specific individual- and area-level factors, some subgroups of the population are at particular risk.

Observational data has demonstrated that the association between temperature and mortality and cardiovascular mortality is U- or J-shaped. This means that beyond an optimal temperature, both low and high temperatures increase cardiovascular risk. In addition, there is emerging epidemiological data showing that climate change-related temperature fluctuations may be particularly challenging for cardiovascular health.

Biological plausibility for these observations comes from the effect of cold, heat, and temperature fluctuations on risk factors for cardiovascular disease. Shared mechanisms of heat and cold adaptation include sympathetic activation, changes in vascular tone, increased cardiac strain, and inflammatory and prothrombotic stimuli. The confluence of these mechanisms can result in demand ischemia and/or atherosclerotic plaque rupture.

In conclusion, public health and individual-level measures should be taken to protect susceptible populations, such as patients with risk factors and/or pre-existing cardiovascular disease, from the adverse effects of non-optimal temperatures.

This review aims to provide an overview of the association between temperature extremes and cardiovascular disease through the lens of pathophysiology and observational data. It also highlights some specific meteorological aspects, gives insight to the interplay of air temperature and air pollution, touches upon social dimensions of climate change, and tries to give a brief outlook into what to expect from the future.

1. Introduction

Our relationship with the weather is shaped by two facts. First, weather is always present and cannot be turned off. Second, it is constantly changing. In other words, changing weather stimuli affect everyone. This requires constant physiological adaptation. The autonomic nervous system manages automatic responses, regulation, and compensatory mechanisms in circulation, metabolism, and thermoregulation. While healthy individuals can generally adapt even to extremes, adaptation can be challenging for subgroups of the population with pre-existing conditions. In addition to these individual factors, contextual factors also play a role that can mitigate or exacerbate this association, such as geographical region, land use, social determinants, and other environmental factors such as air pollution [1]. This totality of lifelong changes in human physiology and pathophysiology induced by exposure to environmental stressors is best described by the exposome

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concept. It aims to encompass the variety and breadth of exposures, such as chemical and physical factors (e.g. synthetic chemicals, air pollution, noise, ultraviolet radiation, and air temperature), lifestyle and behavioral factors (e.g. dietary elements, smoking habits, physical activity, and cosmetics), as well as psychosocial stressors, along with their associated biological responses [2,3].

There are many diverse effects of climate change, including extreme weather events like intense and heavy rainfall, floods, storms, droughts, and an increased risk of forest fires [4]. These extreme weather events are often related to the steady and slow increase in temperature known as global warming caused by climate change. According to the Intergovernmental Panel on Climate Change [5] (IPCC), the global mean air temperature will continue to rise until at least the middle of the century under all emission scenarios considered. Global warming of 2 °C is thus likely to be exceeded within the 21st century. However, higher mean annual air temperatures are not the main reason that climate change is one of the most dangerous crises of our time. As visualized in Fig. 2, global warming causes a shift of the complete temperature distribution. For example, when a mid-20th century temperature distribution is compared to a more recent distribution, the frequency of "normal" temperature days decreased, whereas the number of hot days (and especially extremely hot days) increases markedly. On the other hand, the number of very cold days decreases, although cold days are, of course, still occurring. This can easily be verified when looking at real data, for example from Germany. The German Weather Service has shown that the number of hot days (with maximum air temperatures $\geq$30 °C) per year increased between 1951 and 2023 by 8.8 days, whereas the number of days with temperatures below freezing (maximum air temperatures $<$0 °C) per year decreased simultaneously by 14.4 days [6]. In addition to increasing ambient air temperature extremes, temperature variability (unexpected and rapid temperature changes) has also increased [7,8]. Tropical regions, home to many low- and middle-income countries, are experiencing a more pronounced increase in temperature variability than other parts of the world. This makes the impact and burden of climate change unequally distributed globally and may contribute to increased disparities [9].

While genetic or acquired personal risk factors play an important role in the gradual development of cardiovascular diseases (CVD), environmental stressors can trigger acute manifestations [10–12]. Both extreme cold and extreme heat are associated with increased cardiovascular risk [13,14]. The "Global Burden of Diseases, Injuries, and Risk Factors Study 2019″ estimated that 11.7 million healthy life years were lost worldwide due to high temperatures [15]. Moreover, non-optimal temperatures were ranked tenth among all risk factors and second among environmental risk factors for deaths in 2021 worldwide [16]. Cold is an important risk factor for cardiovascular events – a fact that cardiologists have known for decades. An example of this can be found in Augsburg, Germany, where one study demonstrated that temperature effects on myocardial infarctions (MIs) changed between 1987 and 2014. Between 1987 and 2000, MIs were primarily caused by cold exposure, but heat emerged as a significant additional risk factor between 2001 and 2014, especially in individuals with diabetes or dyslipidemia [17]. Biological plausibility for this observation comes from the known effects of cold and heat on autonomic function, as well as inflammatory and thrombotic pathways that are involved in the sequelae of acute CVD events [18]. Specifically, a central regulatory mechanism that underlies the association between temperature extremes and CVD risk may be increased activation of the sympathoadrenergic and hypothalamic-pituitary-adrenal axes [18]. The neuroendocrine correlates of this stress response include an increase in the stress hormones noradrenaline, epinephrine, and cortisol, as well as the promotion of chronic inflammatory systemic and vascular processes and increased thrombogenicity [18]. These in conjunction can lead to demand ischemia, and favor the sequelae of atherosclerotic plaque destabilization, plaque rupture, thrombosis, acute ischemia, and acute coronary syndrome [19].

Temperature fluctuations, heatwaves, and cold spells all play an important role in the development, progression, and acute clinical manifestation of CVD. This review aims to summarize the current knowledge of known and still relevant cold effects while also discussing specific aspects of heat effects which have not been covered in depth by more recent reviews of heat effects [20,21]. Moreover, a specific focus is put on the potential pathophysiological mechanisms leading from temperature exposure to (severe) health outcomes (Fig. 1).

2. Relevant meteorological parameters

Temperature metrics (including mean, minimum, and maximum temperatures) are fundamental to assessing exposure levels in both short-term and long-term studies [13,22,23]. Temperature variability metrics capture fluctuations that may also contribute to physiological stress [24]. These metrics quantify the increases in extreme temperature events and temperature variability caused by climate change which amplify the impact of temperature on health outcomes [4].

Heatwaves and cold spells are when temperatures go beyond a certain threshold or percentile for a number of consecutive days, with definitions varying inconsistently by region and study design [25–27]. In one example from Liu et al. [25], a low-intensity heatwave is defined as temperatures between the 90th and 93rd percentiles compared to a reference time period, medium intensity between the 94th and 96th percentiles, and high intensity at or above the 97th percentile (Table 1). As the literature on the health effects of cold spells is limited, we opted in our Table for a more general definition than for heatwaves (Table 1).

3. Cold effects on CVD morbidity and mortality

3.1. Ischemic heart disease

Ischemic heart disease (IHD) represents the predominant contributor to global mortality attributed to CVD. Numerous studies have established associations between short-term exposure to low temperatures and MI, with evidence from diverse geographical regions, including the U.S., Sweden, Germany, China, Japan, and Australia [22,28–47]. A global study within the Multi-Country Multi-City Collaborative Network (MCC), which assembled a database of daily counts of cardiovascular deaths from 567 cities across 27 countries, demonstrated that extreme cold (1st percentile of temperature) was associated with a significantly higher risk of mortality from IHD compared to the minimum mortality temperature (MMT) [28]. Extreme cold exposure was associated with a pooled relative risk (RR) of 1.33 (95 % confidence interval [CI]: 1.26–1.41) for mortality due to IHD [28]. This study underscored the substantial impact of cold on CVD mortality, with consistent findings across multiple causes of death, including stroke and heart failure (HF). However, the specific role of extreme events, such as cold spells, in exacerbating MI risk is less frequently studied. A recent national study in Sweden simultaneously explored the effects of lower temperatures and cold spells, defined as periods of at least two consecutive days with a daily mean temperature below the 10th percentile of the temperature distribution for each municipality, on MI during the cold season (October to March) [22]. Instead of absolute temperature values, the study used percentiles of daily temperatures experienced by individuals in the same municipality as exposure indicators to account for geographic adaptation. It found that a 1-unit decrease in percentile temperature was associated with an increased risk of total MI, non-ST-elevation MI (NSTEMI), and ST-elevation MI (STEMI) at a lag of 2–6 days, with odds ratios (ORs) of 1.099 (95 % CI: 1.057–1.142), 1.110 (95 % CI: 1.060–1.164), and 1.076 (95 % CI: 1.004–1.153), respectively. Furthermore, short-term exposures to air temperature and cold spells were independently associated with increased risks of MI after mutual adjustment. However, findings are not entirely consistent across studies. A study in Helsinki, Finland found that decreased air temperature during the cold season was linked to an increased risk of MI hospitalization,

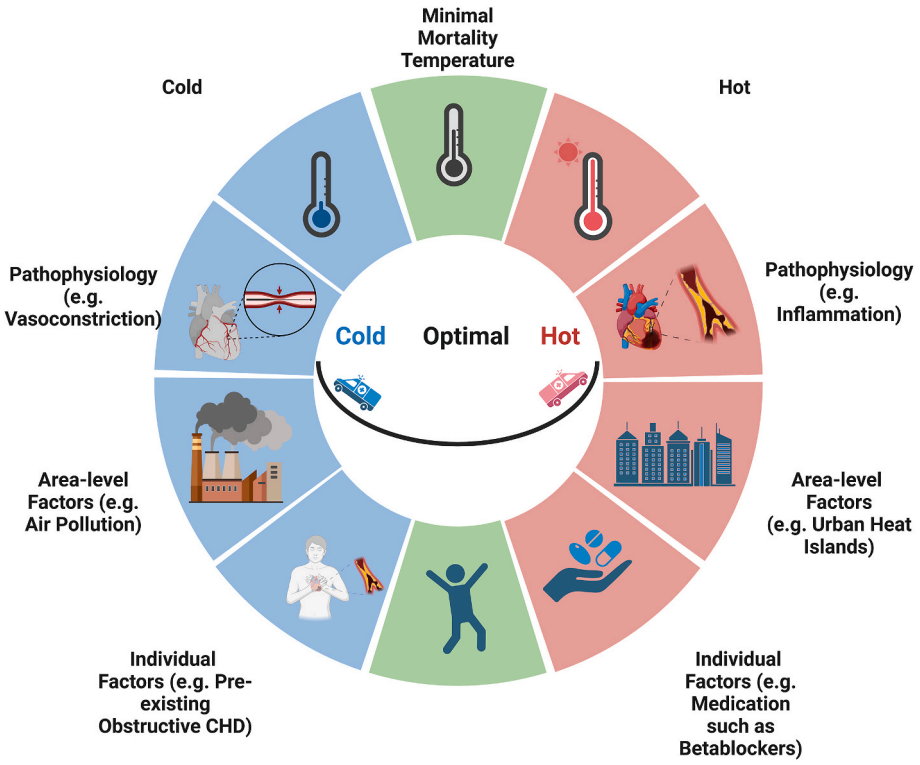

Fig. 1. Graphical abstract – Cardiovascular risk of low and high air temperature.

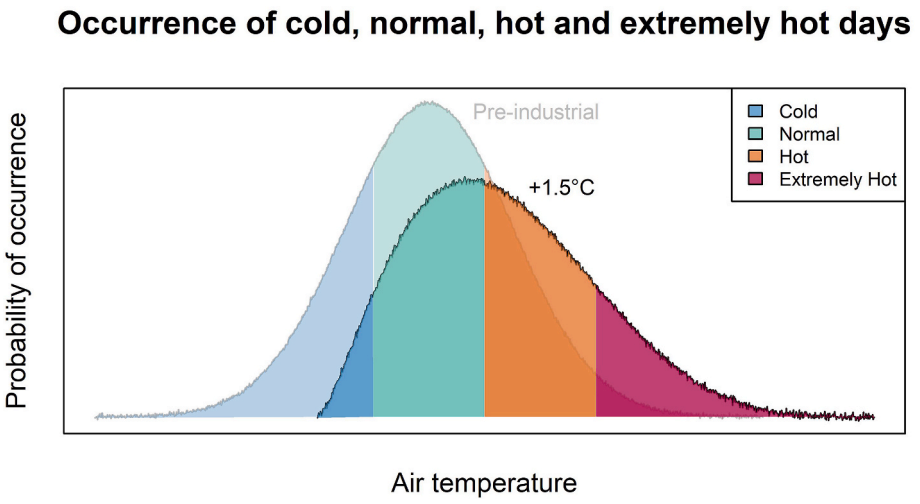

Fig. 2. Temperature distribution shift by climate change.
(Adapted from Ref. [120])

while cold spells showed no significant effect [48]. Despite these observations regarding short-term effects, the long-term impacts of low temperatures on IHD morbidity and mortality remain largely unexplored, highlighting a notable gap in the literature. A study focusing on fee-for-service Medicare beneficiaries aged 65 years and older in the U.S. found that decreases in cold-season average temperatures (from January through March and October through December) were associated with an increased rate of MI hospital admissions, showing a rate difference of 5.01 admissions per 100,000 person-years (95 % CI: 2.18–7.67) [49].

3.2. Heart failure

The relationship between low temperatures and HF morbidity and mortality remains relatively underexplored compared to other cardiovascular conditions. Emerging evidence suggests a significant impact of cold exposure on HF-related health outcomes. A large study conducted by the MCC reported that exposure to extreme cold (1st percentile of temperature compared to the MMT) was associated with a pooled RR of 1.37 (95 % CI: 1.28–1.47) for HF mortality [28]. Similarly, a nationwide study from Spain found that low temperature (1st percentile of temperature vs. MMT) increased the risk of HF mortality with a RR of 1.54 (95 % empirical CI [eCI]: 1.33 to 1.78) [50]. Research from Hong Kong, China, and Quebec, Canada reported that lower daily temperatures were significantly associated with increased mortality from HF [51,52]. Additionally, a nationwide study in Japan indicated a heightened risk of HF hospitalization during low temperatures (5th percentile vs. MMT),

Table 1
Relevant air temperature metrics in epidemiological studies.

Temperature Metrics	Definition
Average or mean temperature [°C]	Average ambient temperature [°C] during a specific time period, e.g., daily or yearly average.
Maximum temperature [°C]	The measured peak value for ambient temperature [°C] during a specific time period.
Minimum temperature [°C]	The measured minimum value for ambient temperature [°C] during a specific time period.
Apparent temperature [°C]	Accounts for ambient temperature, relative humidity [%], vapor pressure, and wind speed [m/s].
"Wet-bulb Globe" temperature (WBGT) [°C]	Accounts for ambient temperature, relative humidity [%], solar radiation (kW/m^2), and wind speed [m/s].
Heat index [°C]	Accounts for ambient temperature and relative humidity [%].
Universal Thermal Climate Index (UTCI) [°C]	Accounts for ambient temperature, relative humidity [%], wind speed [m/s], and mean radiant temperature (function that includes temperature and cloud coverage).
Physiological Equivalent Temperature (PET) [°C]	Accounts for ambient temperature, latitude/longitude, relative humidity [%], wind speed [m/s], cloud coverage, and individual attributes (e.g., size, weight, age, sex, clothing, physical activity).
Hot night excess temperatures	Between sunset and sunrise: sum of hourly positive deviations from a specific threshold temperature
Temperature Variability	
Diurnal temperature range (DTR) [°C]	Difference between the maximum [°C] and minimum [°C] temperatures of a day.
Temperature change between consecutive days [°C]	Difference in daily average temperatures [°C] between two consecutive days.
Intraday temperature variability	Weighted average standard deviation of the minimum and maximum temperature on one day.
Interday temperature variability	Weighted standard deviation of daily average temperatures over a specified number of days.
8-day temperature variability [°C]	Difference between the daily average temperature and the mean daily average temperature of the preceding seven days.
Heatwaves	
Low intensity heatwave	Daily average temperature/minimum/maximum/apparent temperature [°C] exceeds the temperature threshold of ≥90–93rd percentiles of the annual average for 0–4 consecutive days.
Medium intensity heatwave	Daily average temperature/minimum/maximum/apparent temperature [°C] exceeds the temperature threshold of ≥94–96th percentiles for 0–3 consecutive days.
High intensity heatwave	Daily average temperature/minimum/maximum/apparent temperature [°C] exceeds the temperature threshold of ≥97th percentiles for 0–2 consecutive days.
Heat Wave Duration Index (HWDI)	≥5 consecutive days on which the daily maximum temperature exceeds the average maximum temperature of a reference period by 5 °C or more.
Coldwaves	
Cold Wave Duration Index (CWDI)	≥5 consecutive days on which the daily minimum temperature falls below the average minimum temperature of a reference period by 5 °C or more.
Cold spell	Drop of the daily average temperature/minimum/maximum/apparent temperature [°C] below a certain threshold (e.g. 5th or 3rd percentile compared to a reference period) for a specified number of consecutive days (at least 2 days, up to 5 days)

reporting a RR of 1.571 (95 % CI: 1.487–1.660), which was notably higher than the risk for IHD (RR: 1.119, 95 % CI: 1.040–1.204) and stroke (RR: 1.107, 95 % CI: 1.062–1.155) within the same dataset [53]. While these studies illustrate the effects of low temperatures on HF, it's important to note that these investigations, including the MCC study, did not encompass all countries and populations. This limitation underscores the need for more comprehensive research efforts to confirm the association between cold exposure and HF globally and across diverse demographic groups.

3.3. Stroke

Previous research has demonstrated a significant association between low temperatures and an increased risk of stroke. A recent study, including data on 3.4 million ischemic stroke deaths and 2.5 million hemorrhagic stroke deaths across 522 cities in 25 countries, highlighted that extreme cold contributed to excess deaths [54]. Specifically, for every 1000 ischemic stroke deaths, extreme cold days (1st percentiles of each city's distribution of temperatures vs. MMT) were responsible for 9.1 excess deaths (95 % eCI: 8.6–9.4), and for every 1000 hemorrhagic stroke deaths, extreme cold days were associated with 11.2 excess deaths (95 % eCI: 10.9–11.4). A recent meta-analysis that pooled results from 20 studies across Asia, Europe, North America, South America, and Australia, further supports these findings, indicating that low ambient temperatures increase the risk of stroke morbidity by 33 % (RR: 1.33; 95 % CI: 1.17–1.51) and stroke mortality by 18 % (RR: 1.18; 95 % CI: 1.06–1.31) [24]. These results align with a previous meta-analysis conducted in 2015, which included data from nine countries across Asia to Oceania, in which each 1 °C decrease in temperature was associated with an increase in morbidity and mortality of major adverse cerebrovascular events by 1.2 % (95 % CI: 0.8–1.6) and 1.2 % (95 % CI: 0.9–1.5), respectively [55]. Additionally, the study suggested that individuals with a body mass index <25 kg/m^2 or with hyperglycemia might be more susceptible to the effects of cold. Contrasting these findings, a meta-analysis conducted in 2016 found no significant association between ambient temperature and ischemic stroke occurrence (RR: 0.98, 95 % CI: 0.55–1.02) [56].

3.4. Arrhythmia

The diversity of arrhythmia subtypes and the variability in endpoints make understanding the link between low temperature and cardiac arrhythmias particularly challenging. A global study by the MCC found a significant association between extreme cold exposure (1st percentile temperatures compared to MMT) and an increased risk of arrhythmia-related mortality, with a pooled RR of 1.19 (95 % CI: 1.07–1.33) [28]. In Seoul, South Korea, a study found that each 1 °C decrease in mean temperature was linked to a 1.06 % increase in the risk of cardiac arrhythmias [57]. Similarly, research conducted in London, United Kingdom, observed a 1.2 % rise in the risk of ventricular arrhythmias for every 1 °C decrease in ambient temperature [58]. The risk was particularly pronounced at very low temperatures, with an 11.2 % increase in defibrillator shock events for each 1 °C decline below 2 °C, and individuals over the age of 65 being especially vulnerable to temperature-induced ventricular arrhythmias. Atrial fibrillation (AF), the most common sustained cardiac arrhythmia, has also been linked to cold exposure. A national study across 322 cities in China indicated an association between decreasing temperatures and increased AF onset risk, with a RR of 1.25 (95 % CI: 1.08–1.45) for episode onset at extremely low temperatures (−9.3 °C) over a 0–7 day lag period, compared to a reference temperature of 31.5 °C [59]. The risk was highest at a 3-day lag. A prospective study in Boston, Massachusetts of 200 patients with dual-chamber implantable cardioverter-defibrillators found that lower temperatures in the 48 h preceding the study were associated with an increased risk of new-onset AF among 49 patients [60]. Another study reported a significant negative linear correlation ($R = -0.60$; $p = 0.001$) between daily temperatures and the number of AF episodes [61]. However, findings are not entirely consistent. A nationwide cohort study in Israel found no statistically significant association between low winter temperatures and hospitalizations for AF (cumulative OR at lag 0–7: 1.004, 95 % CI: 0.860–1.230) [62]. Seasonal variations in AF episodes have also been documented, with higher rates

during winter and lower rates in summer, as highlighted in a systematic review [63]. The inconsistencies observed within these limited evidence suggest a need for further investigation to clarify the nuanced relationship between low temperature and arrhythmias across different populations and conditions.

4. Physiological and pathological response to cold exposure

The clinical symptoms that can occur due to cold exposure in individuals living with coronary artery disease (known or yet unknown) are depicted in Fig. 3.

4.1. Demand ischemia and type 2 myocardial infarction

As shown in Fig. 4, exposure to cold activates the sympathetic nervous system as well as the renin-angiotensin system which results in vasoconstriction. Vasoconstriction of peripheral arteries leads to increased cardiac afterload due to increased systemic blood pressure. This leads to increased cardiac oxygen demand, increased cardiac strain and can result in a mismatch between cardiac oxygen supply and demand. As a result, symptoms of angina in individuals living with pre-existing coronary artery disease may occur [14,64]. Furthermore, vasoconstriction of the coronary arteries per se can exacerbate the mismatch between cardiac oxygen supply and demand and aggravate symptoms of angina. Further exacerbating cardiac strain is the pooling of blood from the periphery to the body center as muscle tone increases and skin blood flow decreases due to peripheral vasoconstriction. This mechanism, which is the physiologic response to cold to counteract heat loss, results in higher cardiac preload. As a result of increased cardiac preload, stroke volume must increase, which, combined with higher cardiac afterload due to peripheral resistance due to vasoconstriction exacerbates cardiac strain [14]. In sum, these mechanisms may result in demand ischemia and type 2 MI.

Fig. 3. Frank Netter's illustration of angina pectoris due to cold exposure. (Source: [121])

4.2. Thrombotic and inflammatory stimuli and type 1 myocardial infarction

A decrease in core body temperature can promote cholesterol crystal deposition in atherosclerotic plaques, which may increase the risk of plaque rupture and type 1 MI in vulnerable individuals [64]. Furthermore, increased muscle tone, including in the smooth muscle cells of the bladder and the associated increase in urine voiding, may lead to dehydration and the sequelae of hemoconcentration and hyperviscosity, a mechanism that may favor a prothrombotic milieu [14,64]. In sum, cholesterol crystallization, hemoconcentration and hyperviscosity, in particular combined with the sympathetic activation and elevated blood pressure levels can lead to the sequelae of atherosclerotic plaque destabilization and type 1 MI [64].

5. Heat effects on CVD morbidity and mortality

While significant and consistent adverse associations have been found between low temperatures and cardiovascular health, the associations between heat and cardiovascular outcomes have so far been smaller and more inconsistent.

Numerous reviews and studies have identified a correlation between rising ambient temperatures, heat waves, and high temperatures with increased cardiovascular mortality and morbidity. The Global Burden of Disease study [65] reported a rise in cardiovascular-related disability-adjusted life years, years of life lost, and mortality caused by high temperatures since 1990. Supporting this, Kazi et al. [66] found that 86 % (87 out of 101) of the studies they reviewed indicated that exposure to extreme heat was associated with higher cardiovascular mortality.

5.1. Strokes

The link between heat and ischemic strokes has been well studied. Alahmad et al. [67] found that extreme heat (temperature in the 99th percentile) was associated with a higher risk of ischemic stroke mortality, reporting a RR of 1.13 (95 % CI: 1.06–1.20). Reviews from Zafeiratou et al. [23], Wen et al. [24], Song et al. [68], and Liu et al. [25] corroborated this finding, reporting increased instances of ischemic strokes with exposure to higher ambient temperatures and extreme heat. In contrast, a review by Dahn et al. [69] indicated a decreased risk of ischemic stroke with higher temperatures.

5.2. Myocardial infarction

Research investigating the association between MI and heat exposure has also shown an increased risk of MI. Song et al. [68] reported that hospitalizations for MI increased by 0.6 %–12 % for every 1 °C rise in temperature during heat exposure. Findings from Phung et al. [70], Wang et al. [56] and Cicci et al. [71] supported this result. Additionally, Sun et al. [44] reported that MI mortality increased (RR: 1.639; 95 % CI: 1.087–2.470) during heat waves.

5.3. Other cardiovascular outcomes

Several studies have reported associations between heat and other cardiovascular outcomes, including HF, hypertension, hypotension, and dysrhythmia. Cicci et al. [71] documented an increase in hospitalizations for HF, dysrhythmia, and hypotension during heat waves. While Song et al. [68] observed an increase in hospitalizations for HF due to heat exposure, they found no significant link between heat exposure and dysrhythmia.

Previous studies have highlighted several limitations that future

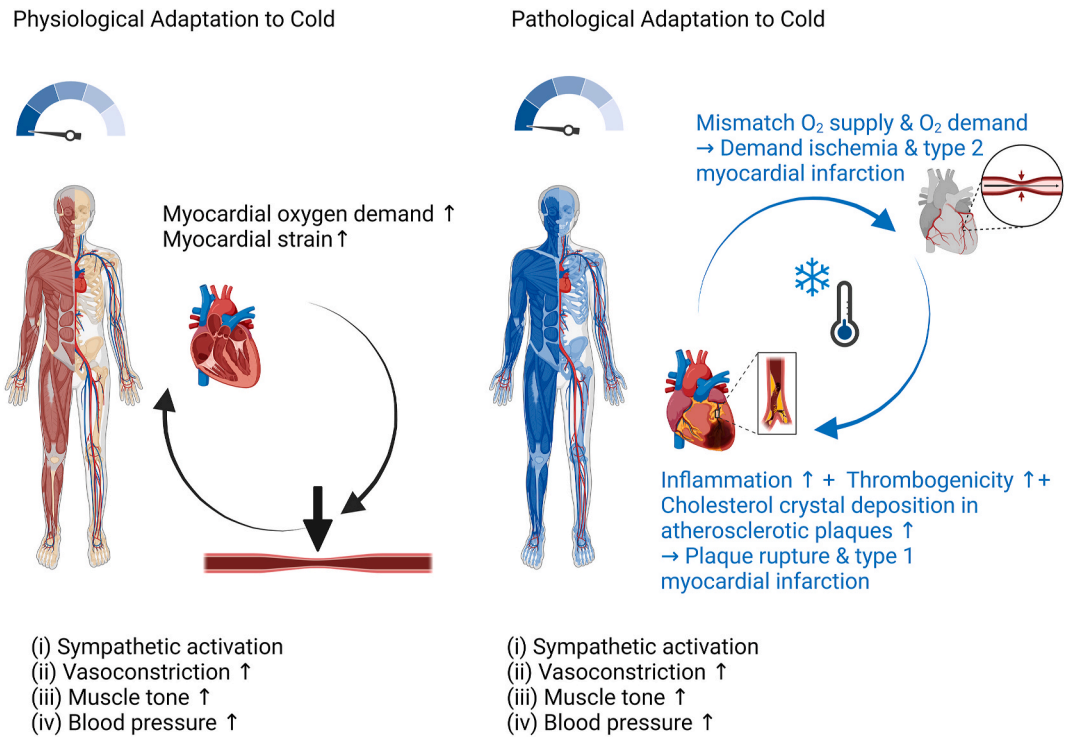

Fig. 4. Cold exposure: Physiological and pathological adaptive responses. (Created with BioRender.com)

research needs to address. First, few studies have examined the relationship between heat and cardiovascular outcomes other than stroke, MI, and general cardiovascular mortality and morbidity. Second, existing literature shows significant heterogeneity in the definitions of heat waves and high temperatures; therefore, more precise definitions should be utilized, considering climatic and regional differences.

6. Physiological and pathological response to heat exposure

As shown in Fig. 5, two physiological adaptation mechanisms occur during heat exposure to reduce core body temperature: (i) peripheral vasodilatation to increase blood flow to the skin to facilitate heat dissipation and (ii) increased sweat secretion to cool the skin by evaporation [20,64]. Due to pre-existing cardiovascular conditions and/or

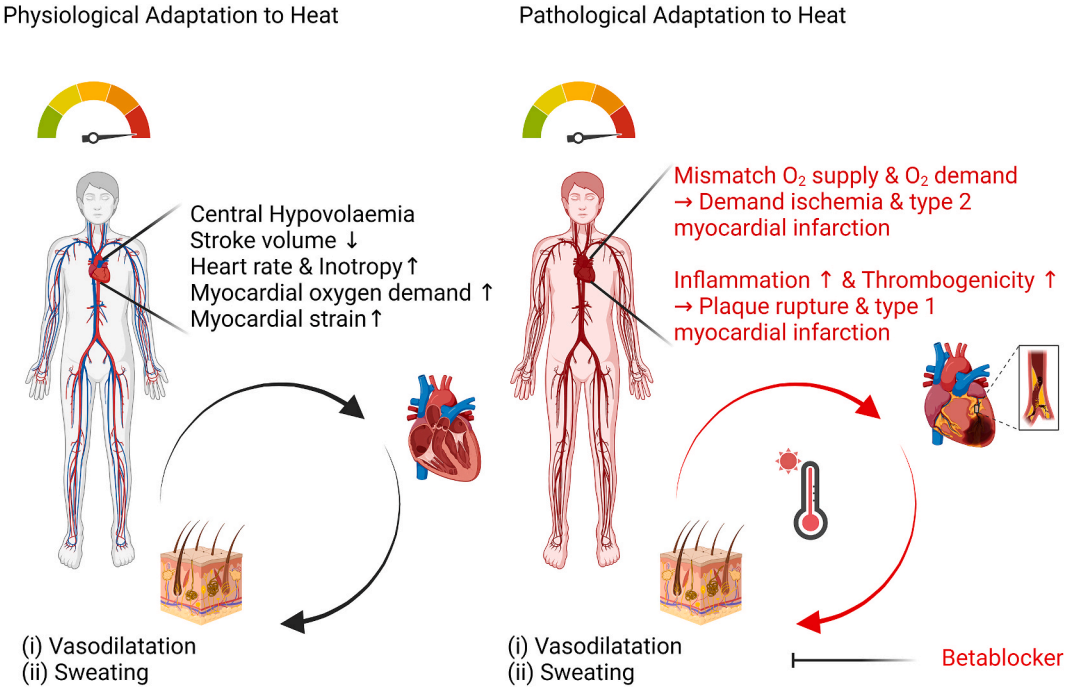

Fig. 5. Heat exposure: Physiological and pathological adaptive responses. (Adapted from Ref. [122], created with BioRender.com)

pre-existing pharmacotherapy, these adaptation mechanisms can be disrupted at several levels. In susceptible individuals living with pre-existing cardiovascular conditions, this can lead to the vicious cycle described in (i), (ii), and (iii) which may result in demand ischemia or atherosclerotic plaque rupture, causing MI and/or stroke [20,64].

(i) Increased blood flow to the skin due to heat-induced peripheral vasodilatation results in redistribution of blood volume to the periphery. This leads to a reduction in cardiac filling pressures, with a consequent reduction in cardiac stroke volume. To compensate for reduced stroke volume, both the heart rate and the inotropy (the force with which the heart contracts) increase, resulting in increased cardiac strain due to higher oxygen demand. In the presence of pre-existing heart disease, particularly HF, this can lead to a mismatch between cardiac oxygen supply and demand. If left untreated, this discordance between oxygen supply and demand can lead to myocardial ischemia, type 2 MI and cardiogenic shock [20,64].

(ii) The second major compensatory mechanism to deal with heat is the secretion of sweat to cool the skin by evaporation. If the resulting fluid loss is not compensated for, it will lead to a reduction in intravascular blood volume. As a result, the stroke volume of the heart decreases, causing a compensatory increase in heart rate to ensure cardiac output to supply blood to critical organs (cardiac output = stroke volume x heart rate). This further exacerbates the vicious circle described in (i). In addition, sweating can promote exsiccosis. The associated reduced intravascular volume can in turn lead to (a) renal damage due to reduced prerenal-induced renal failure and (b) reduced blood flow to the splanchnic area with the possible sequelae of endotoxemia, septic shock with multiple organ failure, and death [20,64].

(iii) Other biologically plausible pathophysiological mechanisms that could underlie the association between heat exposure and increased cardiovascular risk, such as type 1 MI, include heat-associated local and systemic inflammatory processes (e.g., in the context of the endotoxemia described in (ii)) as well as increased hemoviscosity with consecutive promotion of a prothrombotic milieu [72,73]. It is noteworthy that the effects of heat exposure on inflammation and hemostasis in older people with and without pre-existing CVD are not well investigated, raising the question of the transferability of data from younger patient populations [74].

7. Factors of individual susceptibility and area-level vulnerability

The impact of extreme temperatures on cardiovascular health varies across different sub-populations, with risks being modified by both individual- and area-level factors (Fig. 6). Studies have consistently reported greater susceptibility of older individuals due to their reduced thermoregulatory capacity and a higher prevalence of comorbidities [75–77]. The effects of sex on susceptibility to temperature-related cardiovascular issues appear to be more variable. While e.g. studies by Cleland et al. [78], Cicci et al. [71], and Chen et al. [79] found that females were more vulnerable to cardiac hospitalization or mortality during high-temperature exposure, the study by Lian et al. indicated an increased risk for males compared to females. A very recent systematic literature review and meta-analysis [80] showed slightly higher female vulnerability to increasing temperatures for CVD mortality, but not CVD morbidity. However, the effects of heat waves were inconclusive regarding sex-specific impacts. Pre-existing conditions, including cardiopulmonary diseases, diabetes, and mental health disorders, may impair thermoregulatory response and behavioral adaptation to extreme temperatures, increasing the temperature-related CVD risk [20,81]. Additionally, many commonly prescribed medications for CVD, such as beta-blockers and diuretics, can influence physiological responses to heat stress and exacerbate cardiovascular risks during extreme temperature events [77].

Race/ethnicity and socioeconomic status (Textbox 1) may also influence individual susceptibility to extreme temperatures. In general, racial/ethnic minorities and people with lower socioeconomic status are at higher CVD risk during heat and cold exposure [82,83]. This heightened susceptibility could be attributable to limited access to healthcare, a higher prevalence of CVD risk factors, and reduced capacity to adapt to extreme temperatures using resources such as air conditioning or heating [76,84]. Additionally, outdoor manual workers who experience prolonged exposure and physical exertion, particularly refugees and immigrants, represent another population vulnerable to heat stress [84–86].

Geographical variations in temperature-related risk of CVD have been linked to climate zones, socioeconomic characteristics, urbanization, and built environment. For instance, a meta-analysis of 266 studies found stronger associations between higher temperatures and CVD mortality in individuals living in tropical climates or in low- and middle-income countries, compared to other climate zones or high-income countries [25]. Studies from Europe showed greater heat vulnerability

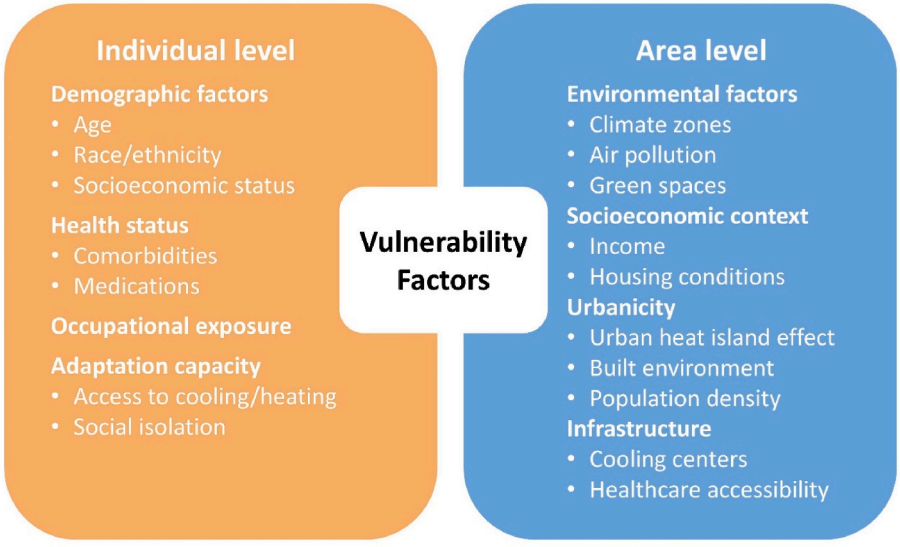

Fig. 6. Individual susceptibility and area-level vulnerability factors in temperature-related health effects.

Textbox 1
Social dimensions in heat exposure

As exposure to extreme heat becomes more common with the progression of global climate change, so too do heat-related health impacts and deaths. However, heat-related morbidity and mortality are not experienced equally across populations [109]. The available evidence increasingly demonstrates that socially disadvantaged individuals and communities are disproportionately experiencing chronic and acute health outcomes and deaths caused by heat. Systematic reviews and meta-analyses have found that women and people with low education were more vulnerable to heat-related mortality [110], and that cities with lower gross domestic product [111,112] and higher economic inequality (measured using the Gini index) [112] have higher heat-related mortality.

One factor contributing to these inequalities is unequal exposure to heat stress. A systematic review of social inequalities in heat stress exposure [113] found evidence of these disparities from all global regions. This review of 123 studies showed that in general, people who are more socially disadvantaged are more likely to be exposed to both subjectively reported and objectively measured heat stress. Income and education are the most studied factors in relation to inequalities in heat exposure. For example, a global study of 25 large cities found that lower-income neighborhoods experienced higher heat exposure [114], and another study of 700,000 households across 52 low- and middle-income countries found that, in countries with warmer climates, households with lower incomes tended to experience higher temperatures [115].

Inequalities in exposure occur across many other social dimensions [113]. Racial and ethnic minorities and immigrants tend to be more exposed to heat stress and to live in hotter neighborhoods. People who are unemployed, outdoor and agricultural workers, and people engaged in manual or low-paying jobs tend to be more exposed at work than people in higher paying "white collar" jobs. Regarding age-related inequalities, contrary to what is found in studies of heat-related health disparities, the review found that children, adolescents, and young adults tend to be more exposed to heat stress than older people.

Inequalities in heat exposure are complex [113]. Social inequalities in heat stress exposure are context-specific: in one setting may be very different than in another, even within the same country. These inequalities are also dynamic, meaning that they may also differ by setting (home versus workplace or school) or depending on the time of day (during a commute or at night while sleeping). More than one social factor will influence whether someone is more (or less) exposed to heat, making inequalities in heat stress exposure multi-dimensional. It's also important to use an intersectional lens when considering inequalities in heat exposure, because multiple social factors in combination may act to affect someone's exposure to heat differently than each of those factors individually (for example: being a woman, being young, being a minority, and having a low-income have different effects on heat exposure than the effect of the combination of these four characteristics).

in areas with high degrees of urbanization, elevated air pollution, and limited green spaces [1,87]. Urban heat island effects (Textbox 2, Fig. 7), particularly in densely populated cities with low greenness, can intensify exposure to extreme temperatures. Additionally, local infrastructure, such as access to healthcare and cooling centers, is crucial for enhancing community resilience to temperature-related CVD risks. Implementing the right adaptation policies could therefore make quite a difference for our future CVD prognosis within the context of climate change (Textbox 3).

The entirety of all these factors is reflected by the exposome concept, which comprises two components – the general external environment and the specific external environment. The first component consists not only of chemical and physical factors, such as air pollution and air temperature, but also of socioeconomic and mental health determinants, including the social environment and capital, exposure to viral and bacterial pathogens, and psychosocial stress [2]. They usually cannot be easily modified by the individual. The second component is defined by lifestyle or behavioral factors, including smoking habits, alcohol consumption, unhealthy diet, physical inactivity and use of consumer products [2]. These factors can more easily be modified by the individual than e.g. controlling outside temperature. With this exposome approach a multi-exposure health effect is considered reflecting better the situation in real life. It also shows key levers for disease prevention and population resilience.

8. Interaction of heat exposure with pharmacotherapy

Heat and pharmaceuticals interact via two mechanisms; (i) heat can alter the onset and/or duration of drug action and/or (ii) interfere with the body's own physiological adaptation mechanisms as a result of heat exposure.

(i) Heat affects the onset and/or duration of drug action via several regulatory mechanisms: (a) heat can lead to relative drug overdosage of renally excreted drugs due to heat-induced exsiccosis with prerenal-induced impaired renal function, (b) heat can lead to a faster onset of action of subcutaneously administered drugs such as insulin with the risk of life-threatening hypoglycemia due to peripheral vasodilation with increased blood flow to the cutis and subcutis, and (c) heat can increase the effect of antihypertensive or anti-anginal drugs through natural compensatory

Textbox 2
What is an Urban Heat Island?

The Urban Heat Island (UHI) effect (Fig. 7) describes the observed phenomenon where urban areas experience higher air temperatures compared to their rural counterparts [116]. This disparity arises primarily from anthropogenic activities, high-density infrastructure, a severe lack of greenness and water bodies, as well as low air flow. Materials like concrete and asphalt absorb, store, and re-radiate heat more effectively than natural surfaces, while the lack of greenness and large water bodies limits the cooling effects of shading and evapotranspiration (sum of all processes by which water moves from the land surface to the atmosphere) [117]. Waste heat, for example produced by air conditioners during hot days, further strengthens the effect. UHI is particularly pronounced at nighttime [118]. The daytime air temperature range (maximum minus minimum air temperature) gets smaller in highly urbanized spots because minimum nighttime air temperature remains significantly high. The larger the urban area, the more intense the UHI effect tends to be.

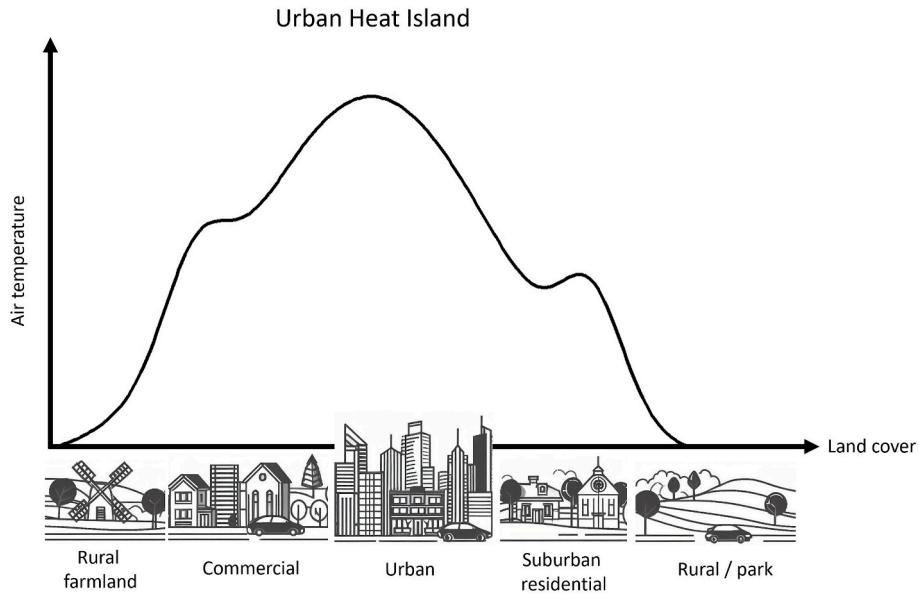

Fig. 7. Schema of an urban heat island.
(Adapted from Ref. [123])

Textbox 3
Future projections

For Europe, climate change-related mortality has been estimated for different favorable and non-favorable future climate and population change scenarios of cold susceptibility and heat adaptation by integrating existing location- and age-specific exposure-response functions for cold and heat effects [119]. In various emission and population scenarios, it has been estimated that the total burden over the course of the century until 2100 will be between 700,000 and two million deaths in Europe alone. The increase in heat-related deaths exceeds the slight decrease in cold-related deaths in all scenarios considered, so the net effect consistently shows an increase in mortality. Furthermore, climate change interacts with demographic trends such as population ageing, multimorbidity and polypharmacy, which can exacerbate heat-related risks even more [20].

mechanisms such as heat-induced vasodilation, which may lead to hypotension in patients with hypertension or HF [88,89].

(ii) Beta-blockers, as shown in Fig. 5, reduce the potential for vasodilation of peripheral vessels and may therefore impair heat dissipation by convection. They are also associated with an increased sweat secretion threshold and thus a reduced ability to dissipate heat [88]. Thus, beta-blockers interact with the described basic physiological adaptation mechanisms to heat exposure. This lends biological plausibility to the observation of an increased rate of heat-related non-fatal MI rate in the general population in the area of Augsburg under concomitant medication with beta-blockers [90].

9. Interactive effects of air temperature and air pollution

Air pollution is the primary environmental risk factor contributing to global cardiovascular morbidity and mortality [91]. This is largely attributed to cardiovascular conditions triggered or aggravated by fine particulate matter ($PM_{2.5}$), such as IHD, MI, HF, stroke, hypertension, and diabetes [92].

Climate change and air quality are linked in many ways [93]. For example, black carbon, one of the components of particulate matter (PM) and the result of incomplete combustion of fuels and biomass, absorbs solar and infrared rays in the atmosphere and thus has a warming effect. On the other hand, in the course of climate change, changes in the atmospheric processes for the transport and mixing of air masses are observed, which can influence pollution [93,94]. Higher air temperatures, in combination with more intense solar radiation, also promote the formation of ozone, but particulate matter pollution can also increase due to the formation of so-called secondary aerosols. Furthermore, climate change influences the frequency and intensity of natural forest or vegetation fires, which can further increase PM concentrations.

These interactions between climate and air pollution have led to an increasing number of studies being conducted over the past decade on the potential interactive effects on cardiovascular health [95,96]. There are shared pathomechanisms as to the effects of (extreme) temperatures and air pollutants on regulatory processes in the body.

Most of the studies published have shown that high temperatures or heat increases the effects of PM and ozone on cardiovascular mortality [95]. In contrast, there are only a limited number of studies investigating whether air pollution modifies the effects of heat. A study from the MCC in 482 cities in 24 countries showed that the effect of heat on mortality from CVD and respiratory diseases was exacerbated by air pollution, including PM and ozone [97]. More specifically, cardiovascular mortality increased by 1.6 % (95 % CI: 1.5 %–1.6 %), 5.1 % (95 % CI: 5.1 %–5.2 %), and 8.7 % (95 % CI: 8.7 %–8.8 %) at low, medium, and high ozone levels, respectively.

A recent U.S. study also showed that the risk of cardiovascular mortality in California increased by 7.6 % (95 % CI: 4.1 %–11.3 %) on days with extremely high temperatures only in the period 2014–2019 and by 4.5 % (95 % CI: 1.1 %–8.0 %) on days with extremely high $PM_{2.5}$

[98] concentrations only. On days with both extreme heat and high $PM_{2.5}$ concentrations, the risk of cardiovascular mortality increased by 29.9 % (95 % CI: 3.3 %–63.3 %), which was higher than the sum of the individual effects of heat and $PM_{2.5}$ alone.

Few studies have investigated combined effects or effect modifications of temperature and air pollutants on hospitalizations or other morbidity endpoints, with inconsistent results. For example, studies from the U.S., China, and Spain showed that high PM concentrations increased the effects of heat on cardiovascular and metabolic hospital admissions or that high temperatures influenced the effects of PM [50,99,100]. However, there are also studies that showed no combined effects or effect modifications or only found effects for cold and air pollutants [101,102]. Other studies also found evidence of interactive effects of high temperatures and $PM_{2.5}$ or ozone on heart rate and heart rate variability [103,104], as well as of low temperatures and $PM_{2.5}$ on blood pressure and markers of endothelial function [105,106].

The potential interactive effects of chronic exposure to air temperature and PM or ozone [107,108] have not been well investigated. Given the changing climate, however, it is important to understand the long-term effects of air temperature on health and their interaction with chronic air pollution exposures [96].

10. Conclusions

This review provides an updated synthesis of the effects of non-optimal temperatures on cardiovascular health, which are especially relevant for patients with atherosclerosis. While all individuals are susceptible to the impacts of weather and climate, certain population groups exhibit more pronounced reactions, such as those with underlying risk factors like dyslipidemia and diabetes, or those living with pre-existing cardiovascular conditions. The diverse composition of these groups gives rise to a wide spectrum of needs and demands on the health system, underscoring the multifaceted nature of climate-related health impacts. These impacts extend beyond individual concerns, significantly affecting the health system itself, its actors, and, consequently, broader organizational and economic structures.

Exposure to both low and high temperatures increase the risk of acute manifestation of CVD. Biological mechanisms underlying the association between cold and heat exposure and increased risk for acute cardiovascular events include neuroendocrine stress responses that affect vascular tone and blood pressure as well as inflammatory and thrombotic stimuli. Understanding the impact of non-optimal temperatures on the cardiovascular system and the interaction of heat and cold with cardiovascular medications is, therefore, essential. This knowledge is crucial for optimizing clinical care pathways and for providing information on preventive behavioral measures for patients.

The implementation of temperature-related health protection measures within the framework of adaptation plans in clinics and healthcare facilities and their execution by medical professionals, particularly cardiologists, care homes, and pharmacists is imperative to safeguard susceptible populations (such as patients with risk factors and/or pre-existing CVD) from the adverse effects of non-optimal temperatures. To protect affected population groups and individuals, the available evidence must be considered and integrated into respective health communication strategies, campaigns, and materials as a core element of these action plans. It is crucial to tailor communication strategies for all target audiences, including the selection of communication channels, the language used, and the creation specific messages and recommended behaviors. Furthermore, it is imperative to disseminate the findings and make them accessible to all of the various actors involved in cold- or heat-health-action planning across different countries, levels and contexts. This ensures integration with ongoing processes, enhancing the effectiveness of these initiatives.

Researchers must strengthen their connections and collaborations with all relevant decision-makers and actors in the domain of temperature extremes and health. These actors include public health authorities responsible for developing and implementing measures to safeguard population health from the consequences of heat and cold and relevant service providers from the medical and social sectors. Action is needed on the issue of non-optimal temperature and CVD.

CRediT authorship contribution statement

Wenli Ni: Conceptualization, Writing – original draft, Writing – review & editing. **Ashtyn T. Areal:** Conceptualization, Writing – original draft, Writing – review & editing. **Katharina Lechner:** Visualization, Writing – original draft, Writing – review & editing. **Susanne Breitner:** Conceptualization, Writing – original draft, Writing – review & editing. **Siqi Zhang:** Visualization, Writing – original draft, Writing – review & editing. **Margarethe Woeckel:** Visualization, Writing – original draft, Writing – review & editing. **S Claire Slesinski:** Writing – original draft, Writing – review & editing. **Nikolaos Nikolaou:** Visualization, Writing – original draft, Writing – review & editing. **Marco Dallavalle:** Visualization, Writing – original draft, Writing – review & editing. **Tamara Schikowski:** Conceptualization, Writing – original draft, Writing – review & editing. **Alexandra Schneider:** Conceptualization, Supervision, Writing – original draft, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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