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Evidence-Based Medicine REVIEW

Cold exposure and acute chest pain-related life-threatening cardiovascular diseases: A scientific statement

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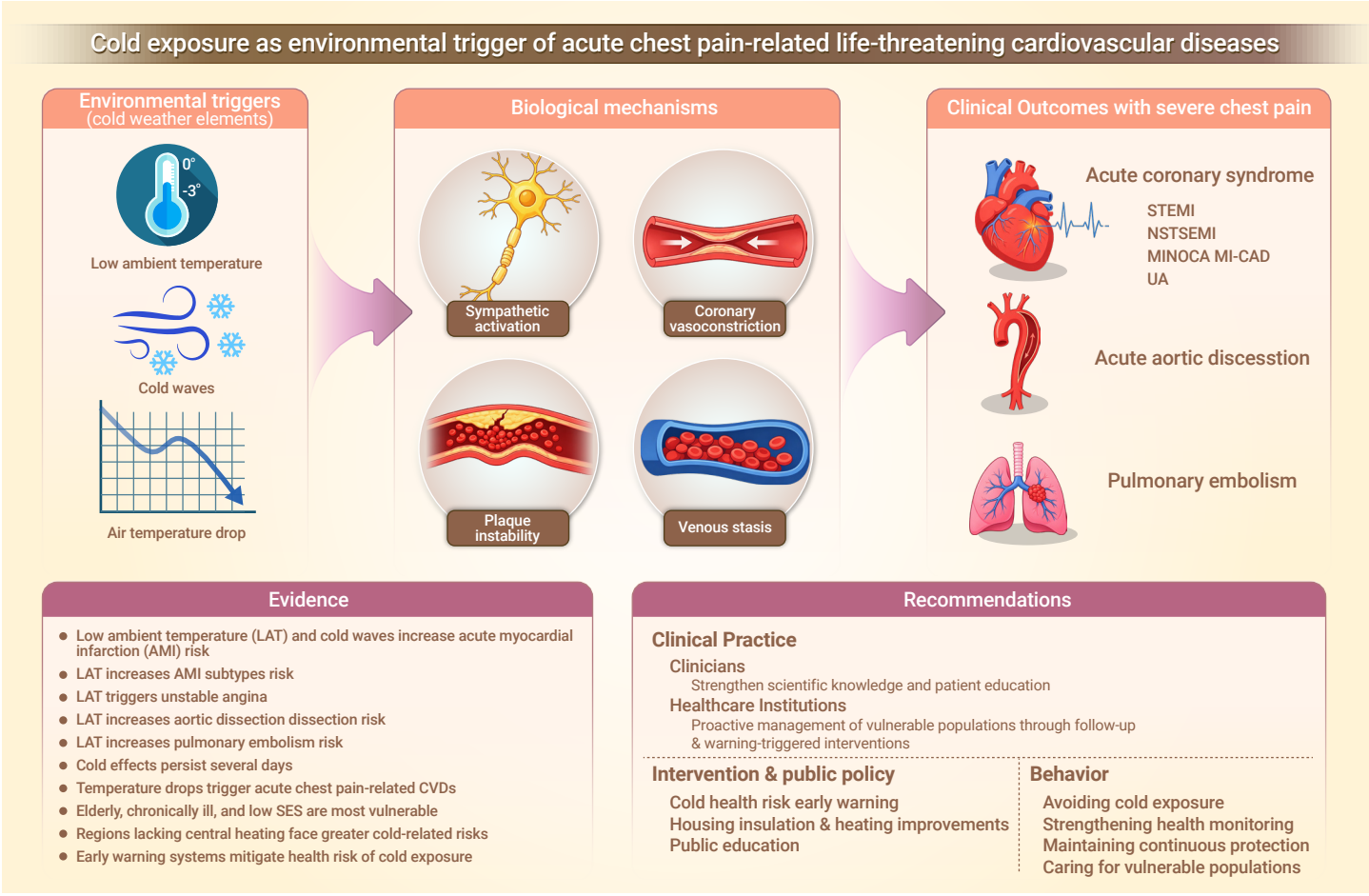
GRAPHICAL ABSTRACT



PUBLIC SUMMARY

- Cold exposure is a major but preventable trigger of acute life-threatening cardiovascular events.
- Multidisciplinary evidence links cold exposure to coronary events, aortic dissection, and pulmonary embolism.
- Older adults, chronically ill patients, and low-SES communities are especially vulnerable to cold exposure.
- Integrated clinical, policy, and behavioral measures can reduce cold-related cardiovascular risks.

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Climate change has amplified the variability and intensity of cold weather, contributing to a growing health burden. Cold exposure serves as a significant, yet preventable, environmental trigger for acute chest pain–related life-threatening cardiovascular diseases (CVDs), such as acute coronary syndrome, acute aortic dissection, and pulmonary embolism. This scientific statement synthesizes multidisciplinary evidence from meteorology, environmental epidemiology, basic science, and clinical research to offer an updated evaluation of the impact of cold exposure on these acute chest pain-related life-threatening CVDs. The evidence consistently demonstrates that cold weather significantly increases the incidence of such events, often with delayed effects lasting several days to weeks. Vulnerable groups, including the elderly, individuals with chronic conditions, and those of lower socioeconomic status, are particularly at risk. Data also suggest that interventions, including central heating, integrated health warning systems, and appropriate personal protective measures, can effectively mitigate the associated risks. Based on this evidence, the statement provides expert consensus recommendations across clinical, policy, and behavioral domains. Strengthening prevention and response to cold-related cardiovascular risks is essential for building climate-resilient health systems and mitigating the health impacts of climate change.

INTRODUCTION

The Global Burden of Disease Study (GBD 2023) has demonstrated that cold exposure is a significant environmental risk factor for both the incidence and mortality of cardiovascular diseases (CVDs). In 2023, approximately 1.348 million deaths globally were attributed to cold exposure, with over 475,122 of these due to ischemic heart disease, a primary acute chest pain–related life-threatening CVD.[1] China bears the largest burden, with an estimated 326,404 cold-related CVD deaths during the same period.[1] Research on the cardiovascular effects of cold exposure has shifted from overall CVD to specific disease subtypes. Recent studies highlight the strong association between cold exposure and multiple acute chest pain–related life-threatening CVDs, which are characterized by sudden onset, rapid progression, high mortality, and limited treatment windows. These include acute coronary syndrome (ACS), acute aortic dissection (AAD), and pulmonary embolism (PE).[2,3] Of these, ACS is the most prevalent[4] and accounts for approximately 20% of all CVD-related deaths.[5] The incidence and mortality burden of these conditions in China continue to rise. The crude mortality rate for acute myocardial infarction (AMI) increased significantly from 43.92 per 100,000 in 2006 to 138.94 per 100,000 in 2020.[6] Similarly, the incidence of PE rose from 1.2 per 100,000 in 2007 to 7.1 per 100,000 in 2016.[7] Given the well-established risks associated with cold exposure and the growing epidemiological burdens, acute chest pain–related life-threatening CVDs warrant prioritized surveillance and prevention in cold environments.

In recent years, climate change has exacerbated cold-related health risks by increasing air temperature variability and intensifying cold weather events, including more sudden and severe cold waves.[8,9] Despite the overall trend of global warming, climate change has resulted in more frequent and extreme cold events, posing substantial challenges for the prevention and management of acute chest pain–related life-threatening CVDs. As the risks of cold exposure grow in the context of climate change, the global community has recognized the need to integrate health considerations into climate governance. At the 2023 United Nations Climate Change Conference (COP28), the *Declaration on Climate and Health* emphasized that "human health should be placed at the heart of climate action." Similarly, the World Health Organization (WHO)'s *Fourteenth General Programme of Work (2025–2028)* identifies climate change as its primary strategic priority. Concurrently, the World Heart Federation has included "placing cardiovascular health at the core of health and climate policy" in its 2030 agenda.

These global commitments underscore the central role of health in contemporary climate governance. In this context, the impact of cold exposure on acute chest pain–related life-threatening CVDs poses a significant challenge to both public health and clinical practice, highlighting the need for coordinated, evidence-based actions across disciplines and sectors. This statement calls for a shift in CVD prevention and control—from a disease-centered approach to a health-centered model—and aims to support the development of climate-resilient cardiovascular health systems in the context of global climate change.

OBJECTIVES AND METHODS

To address this growing public health concern, this scientific statement synthesizes multidisciplinary evidence from meteorology, environmental epidemiology, basic medical science, and clinical practice to provide an updated assessment of the associations between cold exposure and acute chest pain–related life-threatening CVDs.

The writing group was composed of experts from a broad range of disciplines, including cardiovascular medicine, environmental epidemiology, statistics, atmospheric sciences, and public policy. Relevant literature published up to October 2025 was identified through searches of Web of Science, PubMed, China National Knowledge Infrastructure, and Wanfang Data. Key search terms included "ambient air temperature," "low air temperature," or "cold," combined with terms such as "chest pain," "cardiovascular disease," "acute coronary syndrome," "acute aortic dissection," "pulmonary embolism," "ST-segment elevation myocardial infarction," "non-ST-segment elevation myocardial infarction," and "unstable angina." Corresponding Chinese keywords were used when searching Chinese-language databases. Additional studies were sourced from reference lists of retrieved publications and through the expert knowledge of the writing group members. All evidence statements, conclusions, and recommendations represent the consensus of the writing group and were developed through formal discussions.

The statement first reviews the classification of cold exposure and the underlying biological mechanisms identified in animal and human studies. It then summarizes key epidemiological evidence on mortality, morbidity, and surrogate outcomes. Finally, consensus perspectives and recommendations are presented to inform clinical practice, public policy, and behavioral strategies.

COLD EXPOSURE ASSESSMENT

To systematically evaluate the impact of cold exposure on acute chest pain–related life-threatening CVDs, three commonly used indicators of cold exposure were summarized based on the existing literature (Table 1).

Low ambient air temperature (LAT) typically refers to outdoor air temperatures that fall below the threshold of human thermal comfort. In epidemiological studies, LAT is often defined using the minimum risk temperature (MMT)—the temperature at which mortality or morbidity risk is lowest.[1,10] This threshold varies significantly by region. For instance, a global analysis found that the MMT in tropical regions generally corresponds to the 60th percentile of local air temperature distributions, whereas in temperate regions, it typically falls within the 80th to 90th percentiles.[10,11] Therefore, temperatures below the region-specific percentile threshold are classified as LAT.

A cold wave refers to an extreme low-temperature event during the cold season, characterized by air temperatures dropping below a defined threshold and persisting for several consecutive days. Although no universal definition exists, most epidemiological studies use two criteria: (1) temperature threshold, defined either by an absolute criterion (e.g., 4°C lower than the historical mean for the same period) or by a relative criterion (e.g., below the 2.5th, 5th, or 7.5th percentile of the local daily mean air temperature during the study period); and (2) duration, where air temperatures below the threshold persist for several consecutive days.[11-15] For example, a nationwide study in China defined a cold wave as a period during which the daily mean temperature fell below the 7.5th percentile of the local air temperature distribution for at least two consecutive days.[12]

Air temperature drop refers to a short-term decrease in air temperature during the cold season.[11] Recent studies suggested that the extreme day-to-day temperature changes in low and mid-latitudes are likely to be intensified

Table 1. Exposure indicators for cold weather.

Exposure Indicator	Assessment Method
Low Ambient Air Temperature	Air temperature during the cold season
Cold Wave*	An extreme low air temperature event during the cold season, defined by air temperatures below a defined threshold and persisting for several consecutive days
Air Temperature Drop	A decrease in air temperature within a short period

* Also referred to as cold spell in the literature; cold wave is used in this statement.

under future global warming scenarios.[16] In epidemiological research, air temperature drop is often treated as a continuous exposure indicator, quantified by the magnitude of air temperature decrease within a specified time window (e.g., between two consecutive days or within a seven-day period). For example, a nationwide study defined an air temperature drop as a decrease in daily mean temperature between two consecutive days.[17]

BIOLOGICAL MECHANISMS LINKING COLD EXPOSURE TO ACUTE CHEST PAIN-RELATED LIFE-THREATENING CVDS

Cold exposure may increase the risk of acute chest pain–related CVDs through several interconnected pathophysiological pathways.

Sympathetic activation and hemodynamic stress

One important mechanism involves activation of the sympathetic nervous system during cold exposure. Cold weather stimulates sympathetic activity, resulting in increased heart rate and blood pressure.[18,19] Peripheral vasoconstriction further elevates systemic vascular resistance and diastolic pressure, thereby increasing myocardial oxygen demand and cardiac workload.[18,19] Cold-induced increases in afterload and microcirculatory dysfunction may shorten diastole, reduce subendocardial perfusion, and increase extravascular compression, thereby exacerbating myocardial ischemia.[20] Moreover, these hemodynamic responses may contribute to acute aortic dissection. Increased heart rate and blood pressure elevate aortic wall stress, while peripheral vasoconstriction augments afterload, predisposing susceptible individuals to dissection events.[21-23]

Coronary vasoconstriction and oxygen supply–demand imbalance

Cold exposure may induce coronary vasoconstriction and coronary artery spasm, thereby reducing coronary blood flow and impairing myocardial perfusion. Activation of cold receptors in the skin or airways increases catecholamine release, which may trigger spasms in both the epicardial coronary arteries and the coronary microcirculation, potentially contributing to ischemic events such as myocardial infarction with non-obstructive coronary arteries (MINOCA).[24,25] In individuals with coronary artery stenosis, reduced myocardial oxygen supply and increased cardiac workload may cause oxygen supply-demand imbalance, leading to myocardial ischemia and angina.[26,27]

Plaque instability

Cold exposure may also promote instability of atherosclerotic plaques. Exposure to low temperatures has been associated with elevated low-density lipoprotein cholesterol (LDL-C) levels, plaque enlargement, and the formation of larger necrotic cores.[28] These changes can weaken the fibrous cap and promote plaque rupture, a key initiating event in ACSs, including AMI and non-ST-segment elevation myocardial infarction (NSTEMI).[28] Sustained elevations in heart rate and blood pressure during thermoregulatory responses may increase hemodynamic stress on vulnerable plaques and further contribute to plaque instability.[29-34]

Prothrombotic and inflammatory mechanisms

Cold exposure promotes a prothrombotic state through multiple pathways. Low temperatures enhance platelet aggregation, upregulate coagulation factors, impair vascular endothelial function, and trigger inflammatory responses.[35,36] Cold exposure may also induce diuresis, plasma volume reduction, and hemoconcentration, which increase blood viscosity and favor coronary thrombosis. The combined effects of vasoconstriction, tachycardia, elevated blood pressure, and increased blood viscosity may further increase the risk of occlusive cardiovascular events.[37] Peripheral vasoconstriction may reduce blood flow velocity and contribute to venous stasis, while reduced physical activity during cold weather may further aggravate it, potentially favoring thrombogenesis and increasing the risk of deep vein thrombosis and subsequent PE.[35,38-40]

RESEARCH EVIDENCE ON COLD EXPOSURE ASSOCIATED ACUTE CHEST PAIN-RELATED LIFE-THREATENING CVDS

Through a systematic literature review and evidence assessment, ten lines of evidence on the impact of cold weather exposure on acute chest pain–related life-threatening CVDs were summarized (Table 2). These evidence statements were categorized into three primary domains: disease risk, vulnerable populations, and protective measures.

Evidence quality was assessed using the internationally recognized Grading of Recommendations Assessment, Development, and Evaluation (GRADE) system,[41] which classifies evidence as high, moderate, low, or very low. High-quality evidence indicates a high level of confidence in the estimated effect, whereas moderate-quality evidence suggests that further research may influence the estimate. Low-quality evidence reflects substantial uncertainty, and very low-quality evidence warrants cautious interpretation of the findings.

Evidence on disease risk

(1) Low ambient air temperature and cold waves are associated with increased incidence and mortality of acute myocardial infarction (High-quality evidence). Extensive global research has consistently demonstrated a strong association between cold exposure and an increased incidence of acute AMI. As early as the 1930s, clinical observations and epidemiological studies suggested that low ambient air temperatures could act as a significant trigger for coronary events.[42,43] Subsequent systematic studies across multiple countries have consistently confirmed that cold exposure substantially elevates both the incidence and mortality of AMI.[44,45] A 1999 multicenter study by the WHO found that for every 10°C decrease in ambient air temperature, the risk of AMI increased by approximately 13%.[46] More recent large-scale epidemiological studies have supported this finding, identifying low ambient air temperature as a major risk factor for AMI.[47-49] Furthermore, cold waves have been linked to increased AMI risk. A case-crossover study involving over 400,000 AMI cases across 232 Chinese cities from 2015 to 2021 showed that during cold waves (defined as periods when the daily mean air temperature fell below the 7.5th percentile for at least two consecutive days), AMI incidence increased by 4.24% compared to non-cold wave periods.[12] Numerous studies have consistently demonstrated that both the incidence and mortality of AMI are significantly higher during cold waves than in non-cold wave periods.[50,51]

(2) Low ambient air temperature is associated with increased incidence of AMI subtypes (High-quality evidence). Previous research has demonstrated that low ambient air temperature significantly increases the incidence of various AMI subtypes. An analysis of over one million cases from the China Chest Pain Centers (2015–2021) revealed that low ambient air temperature notably elevated the risk of both ST-segment elevation myocardial infarction (STEMI) and NSTEMI. Compared to the minimum-risk temperature, extremely low air temperature (−14.2°C) raised the incidence of STEMI and NSTEMI by 32% and 36%, respectively, with no significant difference between the two.[50] Another study using the same dataset showed that, compared to the minimum-risk temperature, extreme low air temperature (1st

Table 2. Evidence on the impacts of cold exposure on acute chest pain–related life-threatening cardiovascular diseases.

No.	Evidence Description	Evidence Level
Disease Risk		
1	Low ambient air temperature and cold waves are associated with increased incidence and mortality of acute myocardial infarction	High
2	Low ambient air temperature is associated with increased incidence of acute myocardial infarction subtypes	High
3	Low ambient air temperature is associated with increased incidence of unstable angina	Low
4	Low ambient air temperature is associated with increased incidence of acute aortic dissection	High
5	Low ambient air temperature is associated with increased incidence of pulmonary embolism	Moderate
6	The impacts of low ambient air temperature and cold wave on acute chest pain–related life-threatening CVDs can persist for several days	Moderate
7	Ambient air temperature drop in cold season is associated with increased incidence of acute chest pain–related life-threatening CVDs	High
Vulnerable Populations		
8	Elderly individuals, patients with chronic diseases, and populations with low socioeconomic status are more vulnerable to acute chest pain–related life-threatening CVDs under cold exposure	Moderate
Protective Measures		
9	Regions with insufficient central heating coverage may experience higher risks of acute chest pain–related life-threatening CVDs associated with cold exposure	Moderate
10	Early warning systems and protective measures can mitigate the impacts of cold weather on acute chest pain–related life-threatening CVDs	Moderate

percentile) increased the risks of MINOCA and myocardial infarction with obstructive coronary artery disease (MI-CAD) by 58% and 32%, respectively,[52] highlighting cold exposure as a potent trigger for both subtypes.

(3) Low ambient air temperature is associated with increased incidence of unstable angina (Low-quality evidence). The association between low ambient air temperature and unstable angina (UA) remains inconclusive. However, several epidemiological studies have reported a notable winter peak in UA-related hospital visits and mortality,[53,54] suggesting potential adverse effects of cold exposure. Notably, most existing studies have not distinguished between stable angina (SA) and UA. For example, a meta-analysis of over 300,000 angina cases found that each 10°C decrease in air temperature increased the overall risk of angina by 8%, without differentiating UA.[55] Thus, current evidence on the link between cold exposure and UA remains limited.

(4) Low ambient air temperature is associated with increased incidence of acute aortic dissection (High-quality evidence). Accumulating evidence indicates that low ambient air temperature significantly increases the risk of AAD, acting as a key environmental determinant of disease onset. A nationwide study involving more than 40,000 patients from China Chest Pain Centers (2015–2020) reported that each 10°C decrease in air temperature was associated with a 27% increase in AAD incidence.[17] At extremely low air temperatures (−10°C), the risk rose by 184% compared to optimal temperatures (28°C).[56] Similarly, a Japanese study of over 90,000 patients from hospitals between 2012 and 2020 found a 128% higher AAD incidence at −10°C compared to 20°C.[57] Both studies confirmed that cold exposure elevates the risk of both type A and type B AAD.[17,57]

(5) Low ambient air temperature is associated with increased incidence of pulmonary embolism (Moderate-quality evidence). Low ambient air temperature increases the risk of PE. An analysis of nearly 20,000 PE patients from China Chest Pain Centers (2015–2020) revealed that each 10°C decrease in air temperature was associated with a 10% higher PE risk, with the effect persisting for up to 72 hours after exposure.[58] Similarly, a nationwide study in Italy, including over 220,000 PE hospitalizations (2006–2015), found a 10.7% increase in risk per 10°C decrease in air temperature.[59] However, the wide confidence intervals in both studies necessitate cautious interpretation. Overall, epidemiological evidence linking cold exposure to PE remains limited, and causal pathways require further investigation.

(6) The effects of low ambient air temperature and cold wave on acute chest pain–related life-threatening CVDs can persist for several days (Moderate-quality evidence). The adverse effects of low ambient air temperature and cold waves on acute chest pain–related life-threatening CVDs may persist for several days following exposure. Data from more than one million cases reported by China Chest Pain Centers showed that AMI incidence remained significantly elevated for up to three weeks after cold exposure.[50] Subtype-specific analyses revealed that the impact of low ambient air temperature on MINOCA and MI-CAD manifested one day post-exposure, peaked at 4–5 days, and lasted for 7–9 days.[52] A case-crossover study of over 400,000 AMI cases across 232 Chinese cities from 2015 to 2021 further found that cold waves (defined as daily mean air temperatures below the 7.5th percentile for at least two consecutive days) increased AMI risk starting the day after exposure, with the effect persisting for 4–5 days.[12] In contrast, data from China Chest Pain Centers indicate a shorter lag effect for AAD and PE, with risk peaks occurring on the day of exposure and the following day.[56,58]

(7) Ambient air temperature drop in cold season is associated with increased incidence of acute chest pain–related life-threatening CVDs (High-quality evidence). Ambient air temperature drops have consistently been associated with significant increases in the incidence of acute chest pain–related life-threatening CVDs, particularly AMI and AAD. As early as the mid-20th century, clinical observations, such as those by Petersen, noted a rise in coronary thrombosis following abrupt temperature decreases.[43] The first systematic evidence came from Teng et al., who analyzed 1,386 AMI cases in Dallas hospitals (1946–1951) and found a marked increase in incidence during polar cold-air outbreaks.[60] More recent multicenter studies have reaffirmed the strong association between air temperature drops and elevated risks of both AMI[61] and AAD.[17,56] For instance, an analysis of over 40,000 AAD cases from 2015 to 2020 in China found that each 10°C decrease in the daily air temperature difference (mean air temperature of the current day minus that of the previous day) was associated with a 26% increase in incidence risk.[17]

Evidence on vulnerable populations

(8) Elderly individuals, patients with chronic diseases, and populations with low socioeconomic status are more vulnerable to acute chest pain–related life-threatening CVDs under cold exposure (Moderate-quality evidence). Elderly individuals, patients with chronic conditions (such as diabetes, hyperlipidemia, and hypertension), and populations with low socioeconomic status (SES) are particularly vulnerable to acute chest pain–related life-threatening CVDs during cold exposure. Observational studies across multiple countries have consistently shown that low ambient air temperatures and cold waves significantly increase the incidence and mortality of AMI and its subtypes among older adults.[50,52] For instance, a study from the

United Kingdom found that individuals aged 75–84 experienced a significantly higher AMI risk during cold waves (2003–2006) compared to younger individuals,[48] while in Australia, AMI-related hospital admissions (2013–2015) among the elderly rose substantially within four hours of cold wave onset.[62] Similarly, a large-scale Japanese study involving nearly 100,000 patients between 2012 and 2020 reported a notable increase in AAD incidence among individuals aged over 75 during cold waves.[57] Data from China Chest Pain Centers indicate that adults aged 65 and above between 2015 and 2020 had approximately twice the risk of PE compared with younger adults.[58]

Patients with chronic conditions, such as diabetes,[62,63] hyperlipidemia,[62,64] and hypertension,[65] are also more vulnerable to cold-induced acute chest pain–related life-threatening CVDs, likely due to reduced physiological adaptability to environmental stressors. When traditional cardiovascular risk factors are well-managed, environmental triggers, such as cold exposure, may become the predominant precipitating factor. Additionally, populations with low SES face heightened risks due to poor housing insulation, limited access to heating, and lower health awareness compared to higher SES groups.[66-68]

Evidence on protective measures

(9) Regions with insufficient central heating coverage may experience higher risks of acute chest pain–related life-threatening CVDs associated with cold exposure (Moderate-quality evidence). Populations in areas with limited central heating, such as southern China, experience higher risks of acute chest pain–related life-threatening CVDs during cold exposure. Data from China Chest Pain Centers suggest that under low-temperature conditions, the incidence of AMI and its subtypes,[52] as well as AAD and PE, is significantly higher among residents in southern regions compared to those in northern regions with central heating. Specifically, for every 10°C decrease in ambient air temperature, the risk of PE increases by 15% in southern residents but only by 7% in northern residents from 2015 to 2020.[58] Even within northern regions, the risk of acute chest pain–related life-threatening CVDs during the non-heating winter period is substantially higher than during the heating period [50], highlighting the protective role of central heating.

In regions without central heating, adequate personal cold protection measures, such as maintaining indoor warmth and wearing layered clothing, can mitigate the impact of cold exposure on acute chest pain–related life-threatening CVDs. The Eurowinter study published in The Lancet demonstrated that Finnish residents, who maintained higher indoor temperatures and adopted additional protective measures, had significantly lower cold-related health risks than Athens residents, who lacked similar measures.[69,70]

(10) Early warning systems and protective measures can mitigate the impacts of cold weather on acute chest pain–related life-threatening CVDs (Moderate-quality evidence). Timely cold weather warnings and protective interventions, such as household heating support, have been shown to significantly reduce cold-induced cardiovascular risks.[71-74] Countries like the United Kingdom, Canada, and the United States have established cold weather health warning systems that facilitate early public action[75-77] For example, the UK's Cold Weather Plan, introduced in 2011, uses a five-level warning and action framework. Its implementation has been associated with significant reductions in cold-related cardiorespiratory mortality among individuals younger than 65 years between 2007 and 2015.[78] In China, significant progress has been made in cold risk warnings for acute chest pain–related life-threatening CVDs. The "ACS Cold Risk Forecast and Early Warning System" developed by the China Chest Pain Centers in collaboration with the National Institute of Environmental Health, Chinese Center for Disease Control and Prevention, is the country's first disease-specific cold risk alert system. This system uses a four-tier (green, yellow, orange, and red) health risk framework and has been implemented nationwide through the China Chest Pain Center network, providing patients with personalized risk assessments and tailored protective recommendations.

CONCLUSIONS AND RECOMMENDATIONS

Based on the synthesized evidence, this scientific statement concludes that cold weather significantly increases the incidence of acute chest pain–related life-threatening CVDs, often with delayed effect. Elderly individuals, people with chronic diseases, and populations with low SES are particularly vulnerable. However, interventions such as central heating, health warning systems, and appropriate personal protective measures can effectively mitigate the adverse effects of cold exposure on these conditions.

Clinical recommendations for risk mitigation

Clinicians: Strengthen scientific knowledge and patient education. While clinicians recognize the role of climate factors in disease prevention and management, their understanding and application of this knowledge in clinical practice remain limited.[79,80] Evidence-based protective practices, such as maintaining adequate indoor air temperatures, enhancing local body insulation, and avoiding outdoor exposure or vigorous activities during early morning hours, can effectively reduce health risks associated with cold exposure.[73,81,82]

Therefore, clinicians should develop a systematic and up-to-date understanding of the cardiovascular risks associated with cold exposure. In routine practice, meteorological risk factors should be incorporated into medical history taking and risk assessments, with particular attention to cold waves and air temperature drops. Clinicians should educate patients on evidence-based protective strategies, such as recognizing early warning signs of cold-related illness, ensuring appropriate thermal protection, and modifying behavior during cold waves, through personalized counseling and digital or community-based health communication channels. Additionally, climate-health knowledge and response competencies should be integrated into continuing medical education programs and residency training to improve clinicians' climate-health literacy.

Healthcare institutions: Proactive management of vulnerable populations through follow-up and warning-triggered interventions. Proactive follow-up and risk management by healthcare institutions have been shown to improve health outcomes among elderly and chronically ill patients,[83,84] thereby helping reduce cold-related cardiovascular events. Community nurses, who maintain frequent and trusted contact with residents, play a pivotal role in health education and behavioral support during cold seasons.[85,86] Additionally, general hospitals can enhance primary care capacity through optimized resource allocation, strengthened collaboration, and data sharing,[87] while large-scale clinical data offers valuable insights for climate-health research and risk prediction.[88]

Healthcare institutions should monitor cold-weather health risk forecasts and implement automated response protocols during cold waves or ambient temperature drop alerts. Medical resources should be strategically prepositioned, and staff training in risk identification, health education, and emergency response should be strengthened. Primary healthcare facilities should maintain registries of high-risk populations, conduct risk stratification, and implement targeted follow-up. During warning periods, general hospitals should optimize cardiovascular emergency pathways and activate regional collaborative networks—such as medical consortia, specialty alliances, or internet hospitals—to share real-time information, provide remote consultations, and ensure efficient two-way referral systems. Simultaneously, hospitals should leverage clinical data to support research and develop predictive models for cold-related CVD events.

Interventions and public policy

Cold health risk early warning. Developing effective early warning systems is crucial for reducing health risks associated with meteorological events. Although studies specifically addressing cold-weather alerts are limited,[78,89] broader climate-health adaptation research shows that early warning systems can significantly reduce adverse health outcomes.[90] Multisectoral collaboration is recognized as a fundamental element of climate-related health governance,[91,92] and warnings with actionable guidance greatly enhance public adherence to protective behaviors.[93]

Therefore, government authorities should lead the development of a multi-tiered early warning system for acute chest pain–related life-threatening CVDs during cold weather. This system should facilitate data sharing and joint analysis among meteorological, healthcare, and public health sectors, thereby establishing a prevention framework centered on early warning, rapid response, and coordinated action. Graded alerts should clearly define risk levels, targeted populations, and recommended actions, and be disseminated through various accessible communication channels that provide

timely information with clear behavioral guidance.

Housing insulation and heating improvements. Improving residential insulation and heating systems is an effective strategy for mitigating cold-related health risks. Randomized controlled trials have shown that maintaining adequate indoor temperatures significantly reduces the incidence and mortality of cold-related diseases.[73,94] Regional studies further demonstrate that central heating provides significant protective effects, whereas populations in areas with mild winters and inadequate heating, such as southern China, face prolonged exposure and higher CVD risk.[50,52,58,69,95]

Regions with centralized heating should ensure stable and continuous operation while advancing energy-efficient retrofitting of older housing, including improvements in wall and window insulation. Areas without centralized heating should implement temperature-responsive heating strategies to maintain comfortable indoor environments through on-demand heating and adaptive control systems.

Public education. Systematic climate and health education can effectively increase public awareness and promote protective behaviors, thereby reducing cold-related health risks.[96,97] Randomized trials have shown that older adults receiving health education interventions report better perceived health and greater adoption of protective behaviors.[96] Additionally, the design of communication strategies and message framing plays a significant role in improving intervention effectiveness.[97,98]

Thus, a multisector communication network involving public health authorities, meteorological services, healthcare systems, and community organizations should be established. Both traditional and digital media should be utilized to reach diverse audiences, with tailored interventions for high-risk groups. Given the substantial burden of CVDs already observed among adolescents and young adults,[99] public education should also adopt a life-course approach and promote early awareness of modifiable cardiovascular risks. Continuous health education should be implemented in key community settings, such as hospitals, community centers, schools, and nursing homes, especially in regions with mild winters but limited heating and low public awareness.

Behavioral strategies

Avoiding cold exposure. Appropriate personal protective measures—such as maintaining adequate indoor air temperatures, reinforcing insulation of key body areas, and avoiding early-morning outdoor exposure—can significantly mitigate the adverse health effects of cold exposure.[73,81,82]

The public should closely monitor meteorological and public health warnings during cold weather, take preventive measures in advance, and limit unnecessary outdoor activities. Vulnerable individuals should limit outdoor exposure, particularly during early morning or nighttime hours. When outdoors, individuals should wear layered clothing, with particular attention to protecting the head, neck, mouth, nose, and extremities. Indoors air temperature should be maintained at appropriate levels, and precautions should be taken to prevent sudden temperature drops during activities such as bathing or after returning from outdoor exposure.

Strengthening health monitoring. Continuous or home-based monitoring may help reduce the risk of adverse outcomes among older adults and patients with chronic diseases.[100,101] Therefore, high-risk individuals are encouraged to monitor key physiological indicators, such as blood pressure and heart rate, record measurements daily, and seek medical attention promptly if abnormal readings or chest discomfort occur.

Maintaining continuous protection. Cold exposure has prolonged effects, with elevated CVD risks persisting for one to three weeks after exposure.[12,48,50,52] Protective measures should be maintained during and after cold periods, focusing on continued avoidance of cold exposure and consistent health monitoring.

Caring for vulnerable populations. Elderly individuals, those with chronic diseases, and population with lower SES are at heightened risk for acute chest pain–related life-threatening CVDs during cold weather.[52,62-68] These groups often exhibit a pattern characterized by high risk but low risk perception and limited protective actions,[102] while strong family support has been shown to enhance risk awareness and adherence to protective behaviors.[103,104]

Therefore, a family-centered health support system should be considered. Family members should actively recognize health risks, communicate cold-protection knowledge, remind elderly or chronically ill relatives to monitor vital signs and take medications, and provide emotional support and assistance with medical visits. Continuous family engagement can strengthen self-management and support sustained protective behaviors.

REFERENCES

1. Institute for Health Metrics and Evaluation (IHME) (2023). Global Burden of Disease Study 2021 (GBD 2021).
2. Gulati M., Levy P. D., Mukherjee D., et al. (2021). 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR guideline for the evaluation and diagnosis of chest pain: A report of the American college of cardiology/American heart association joint committee on clinical practice guidelines. *J. Am. College Cardiol.* **78**:e187–e285. DOI:10.1016/j.jacc.2021.07.053
3. Harskamp R. E., Laeven S. C., Himmelreich J. C. L., et al. (2019). Chest pain in general practice: A systematic review of prediction rules. *BMJ Open* **9**:e027081. DOI:10.1136/bmjopen-2018-027081
4. Eisen A., Giugliano R. P. and Braunwald E. (2016). Updates on acute coronary syndrome: A review. *JAMA Cardiol.* **1**:718–730. DOI:10.1001/jamacardio.2016.2049
5. Timmis A., Kazakiewicz D., Townsend N., et al. (2023). Global epidemiology of acute coronary syndromes. *Nat. Rev. Cardiol.* **20**:778–788. DOI:10.1038/s41569-023-00884-0
6. Liu K. and Qian D. (2024). Trends in mortality from acute myocardial infarction among urban and rural residents aged 15 – 79 years in China from 2006 to 2020: An age-period-cohort analysis (In Chinese). *Chinese Journal of Public Health* **40**:681–685. DOI:10.11847/zgggws1143413
7. Zhang Z., Lei J., Shao X., et al. (2019). Trends in hospitalization and in-hospital mortality from VTE, 2007 to 2016, in China. *Chest* **155**:342–353. DOI:10.1016/j.chest.2018.10.040
8. Cohen J., Agel L., Barlow M., et al. (2021). Linking Arctic variability and change with extreme winter weather in the United States. *Science* **373**:1116–1121. DOI:10.1126/science.abi9167
9. Zhang R., Screen J. A. and Zhang R. (2022). Arctic and Pacific Ocean conditions were favorable for cold extremes over Eurasia and North America during winter 2020/21. *Bull. Amer. Meteor. Soc.* **103**:E2285–E2301. DOI:10.1175/BAMS-D-21-0264.1
10. Gasparrini A., Guo Y., Hashizume M., et al. (2015). Mortality risk attributable to high and low ambient temperature: A multicountry observational study. *Lancet* **386**:369–375. DOI:10.1016/S0140-6736(14)62114-0
11. World Meteorological Organization. (2023). Guidelines on the Definition and Characterization of Extreme Weather and Climate Events. https://library.wmo.int/viewer/58396/download?file=1310_Guidelines_on_DEWCE_en.pdf&type=pdf&navigator=1
12. Jiang Y., Yi S., Gao C., et al. (2023). Cold spells and the onset of acute myocardial infarction: A nationwide case-crossover study in 323 Chinese cities. *Environ. Health Perspect.* **131**:087016. DOI:10.1289/EHP11841
13. Ryti Niilo R. I., Guo Y. and Jaakkola Jouni J. K. (2016). Global association of cold spells and adverse health effects: A systematic review and meta-analysis. *Environ. Health Perspect.* **124**:12–22. DOI:10.1289/ehp.1408104
14. Ni W., Areal A. T., Lechner K., et al. (2025). Low and high air temperature and cardiovascular risk. *Atherosclerosis* **406**:119238. DOI:10.1016/j.atherosclerosis.2025.119238
15. Lei J., Chen R., Yin P., et al. (2022). Association between cold spells and mortality risk and burden: a nationwide study in China. *Environ. Health Perspect.* **130**:027006. DOI:10.1289/EHP9284
16. Liu Q., Fu C., Xu Z., et al. (2025). Global warming intensifies extreme day-to-day temperature changes in mid–low latitudes. *Nat. Clim. Chang.* **16**:69–76. DOI:10.1038/s41558-025-02486-9
17. Zhang Q., Peng L., Hu J., et al. (2022). Low temperature and temperature decline increase acute aortic dissection risk and burden: A nationwide case crossover analysis at hourly level among 40,270 patients. *Lancet Reg. Health West. Pac.* **28**:100562. DOI:10.1016/j.lanwpc.2022.100562
18. Greaney J. L., Kenney W. L. and Alexander L. M. (2017). Neurovascular mechanisms underlying augmented cold-induced reflex cutaneous vasoconstriction in human hypertension. *J. Physiol.* **595**:1687–1698. DOI:10.1113/jp273487
19. Greaney J. L., Kenney W. L. and Alexander L. M. (2017). Sympathetic function during whole body cooling is altered in hypertensive adults. *J. Appl. Physiol. (1985)* **123**:1617-1624. DOI:10.1152/japplphysiol.00613.2017
20. Williams R. P., Asrress K. N., Lumley M., et al. (2018). Deleterious effects of cold air inhalation on coronary physiological indices in patients with obstructive coronary artery disease. *J. Am. Heart Assoc.* **7**:e008837. DOI:10.1161/JAHA.118.008837
21. Huynh N., Thordsen S., Thomas T., et al. (2019). Clinical and pathologic findings of aortic dissection at autopsy: Review of 336 cases over nearly 6 decades. *Am. Heart J.* **209**:108–115. DOI:10.1016/j.ahj.2018.11.006
22. Edwards D. G., Gauthier A. L., Hayman M. A., et al. (2006). Acute effects of cold exposure on central aortic wave reflection. *J. Appl. Physiol.* **100**:1210–1214. DOI:10.

1152/japplphysiol.01154.2005
23. Manfredini R., Boari B., Gallerani M., et al. (2004). Chronobiology of rupture and dissection of aortic aneurysms. *J. Vasc. Surg.* **40**:382–388. DOI:10.1016/j.jvs.2004.04.019
24. Da Costa A., Isaaz K., Faure E., et al. (2001). Clinical characteristics, aetiological factors and long-term prognosis of myocardial infarction with an absolutely normal coronary angiogram; a 3-year follow-up study of 91 patients. *Eur. Heart J.* **22**:1459–1465. DOI:10.1053/euhj.2000.2553
25. Carder M., McNamee R., Beverland I., et al. (2005). The lagged effect of cold temperature and wind chill on cardiorespiratory mortality in Scotland. *Occup. Environ. Med.* **62**:702–710. DOI:10.1136/oem.2004.016394
26. Neill W. A., Duncan D. A., Kloster F., et al. (1974). Response of coronary circulation to cutaneous cold. *Am. J. Med.* **56**:471–476. DOI:10.1016/0002-9343(74)90478-1
27. Hattenhaur M. and Neill W. A. (1975). The effect of cold air inhalation on again pectoris and myocardial oxygen supply. *Circulation* **51**:1053–1058. DOI:10.1161/01.CIR.51.6.1053
28. Dong M., Yang X., Lim S., et al. (2013). Cold exposure promotes atherosclerotic plaque growth and instability via UCP1-dependent lipolysis. *Cell Metab.* **18**:118–129. DOI:10.1016/j.cmet.2013.06.003
29. Shea D. J., Ockene I. S. and Greene H. L. (1981). Acute myocardial infarction provoked by a cold pressor test. *Chest* **80**:649–651. DOI:10.1378/chest.80.5.649
30. Campisi R., Czernin J., Schöder H., et al. (1998). Effects of long-term smoking on myocardial blood flow, coronary vasomotion, and vasodilator capacity. *Circulation* **98**:119–125. DOI:10.1161/01.cir.98.2.119
31. Zeiher A. M., Drexler H., Wollschlaeger H., et al. (1989). Coronary vasomotion in response to sympathetic stimulation in humans: Importance of the functional integrity of the endothelium. *J. Am. Coll. Cardiol.* **14**:1181–1190. DOI:10.1016/0735-1097(89)90414-2
32. Fairbairn T. A., Motwani M., Mather A. N., et al. (2014). Cardiac MR imaging to measure myocardial blood flow response to the cold pressor test in healthy smokers and nonsmokers. *Radiology* **270**:82–90. DOI:10.1148/radiol.13122345
33. Muller M. D., Gao Z., Drew R. C., et al. (2011). Effect of cold air inhalation and isometric exercise on coronary blood flow and myocardial function in humans. *J. Appl. Physiol.* **111**:1694–1702. DOI:10.1152/japplphysiol.00909.2011
34. Saeki K., Obayashi K., Iwamoto J., et al. (2014). The relationship between indoor, outdoor and ambient temperatures and morning BP surges from inter-seasonally repeated measurements. *J. Hum. Hypertens.* **28**:482–488. DOI:10.1038/jhh.2014.4
35. Keatinge W. R., Coleshaw S. R., Cotter F., et al. (1984). Increases in platelet and red cell counts, blood viscosity, and arterial pressure during mild surface cooling: Factors in mortality from coronary and cerebral thrombosis in winter. *BMJ* **289**:1405–1408. DOI:10.1136/bmj.289.6456.1405
36. Zucker M. B. and Borrelli J. (1954). Reversible alterations in platelet morphology produced by anticoagulants and by cold. *Blood* **9**:602–608. DOI:10.1182/blood.V9.6.602.602
37. Saw J., Humphries K., Aymong E., et al. (2017). Spontaneous coronary artery dissection: Clinical outcomes and risk of recurrence. *J. Am. Coll. Cardiol.* **70**:1148–1158. DOI:10.1016/j.jacc.2017.06.053
38. Woodhouse P. R., Khaw K., Plummer M., et al. (1994). Seasonal variations of plasma fibrinogen and factor VII activity in the elderly: Winter infections and death from cardiovascular disease. *Lancet* **343**:435–439. DOI:10.1016/S0140-6736(94)92689-1
39. Kattlove H. E. and Alexander B. (1971). The effect of cold on platelets. *I. Cold-induced platelet aggregation. Blood* **38**:39–48. DOI:10.1182/blood.V38.1.39.39
40. Gallerani M., Boari B., Smolensky M. H., et al. (2007). Seasonal variation in occurrence of pulmonary embolism: Analysis of the database of the Emilia-Romagna region, Italy. *Chronobiol. Int.* **24**:143–160. DOI:10.1080/07420520601139755
41. Grade Working Group. (2004). Grading quality of evidence and strength of recommendations. *BMJ* **328**:1490. DOI:10.1136/bmj.328.7454.1490
42. Bean W. B. and Mills C. A. (1938). Coronary occlusion, heart failure, and environmental temperatures. *Am. Heart J.* **16**:701–713. DOI:10.1016/S0002-8703(38)90952-4
43. Petersen W. F. (1947). Organic variability in heart disease. *Postgrad. Med.* **1**:36–43. DOI:10.1080/00325481.1947.11691627
44. Anderson T. W. and Le Riche W. H. (1970). Cold weather and myocardial infarction. *Lancet* **1**:291–296. DOI:10.1016/s0140-6736(70)90651-3
45. Mannino J. A. and Washburn R. A. (1989). Environmental temperature and mortality from acute myocardial infarction. *Int. J. Biometeorol.* **33**:32–35. DOI:10.1007/BF01045895
46. Danet S., Richard F., Montaye M. I., et al. (1999). Unhealthy effects of atmospheric temperature and pressure on the occurrence of myocardial infarction and coronary deaths: A 10-year survey: The Lille-World Health Organization MONICA project (Monitoring trends and determinants in cardiovascular disease). *Circulation* **100**:e1–e7. DOI:10.1161/01.CIR.100.1.e
47. Wolf K., Schneider A., Breitner S., et al. (2009). Air temperature and the occurrence of myocardial infarction in Augsburg, Germany. *Circulation* **120**:735–742. DOI:10.1161/CIRCULATIONAHA.108.815860
48. Bhaskaran K., Hajat S., Haines A., et al. (2010). Short term effects of temperature on risk of myocardial infarction in England and Wales: Time series regression analysis of the Myocardial Ischaemia National Audit Project (MINAP) registry. *BMJ* **341**:c3823. DOI:10.1136/bmj.c3823
49. Mohammad M. A., Koul S., Rylance R., et al. (2018). Association of weather with day-to-day incidence of myocardial infarction: A SWEDEHEART nationwide observational study. *JAMA Cardiol.* **3**:1081–1089. DOI:10.1001/jamacardio.2018.3466
50. Jiang Y., Hu J., Peng L., et al. (2022). Non-optimum temperature increases risk and burden of acute myocardial infarction onset: A nationwide case-crossover study at hourly level in 324 Chinese cities. *EClinicalMedicine* **50**. DOI:10.1016/j.eclinm.2022.101501
51. Chen K., Breitner S., Wolf K., et al. (2019). Temporal variations in the triggering of myocardial infarction by air temperature in Augsburg, Germany, 1987-2014. *Eur. Heart J.* **40**:1600–1608. DOI:10.1093/eurheartj/ehz116
52. Huang J., He Q., Jiang Y., et al. (2024). Low ambient temperature and incident myocardial infarction with or without obstructive coronary arteries: a Chinese nationwide study. *Eur. Heart J.* **46**:ehae711. DOI:10.1093/eurheartj/ehae711
53. Khan R. C. and Halder D. (2014). Effect of seasonal variation on hospital admission due to cardiovascular disease - findings from an observational study in a divisional hospital in Bangladesh. *BMC Cardiovasc. Disord.* **14**:76. DOI:10.1186/1471-2261-14-76
54. Hodzic E., Perla S., Iglica A., et al. (2018). Seasonal incidence of acute coronary syndrome and its features. *Mater. Sociomed.* **30**:10. DOI:10.5455/msm.2018.30.10-14
55. Bunker A., Wildenhain J., Vandenbergh A., et al. (2016). Effects of air temperature on climate-sensitive mortality and morbidity outcomes in the elderly; a systematic review and meta-analysis of epidemiological evidence. *EBioMedicine* **6**:258–268. DOI:10.1016/j.ebiom.2016.02.034
56. Chen J., Gao Y., Jiang Y., et al. (2022). Low ambient temperature and temperature drop between neighbouring days and acute aortic dissection: A case-crossover study. *Eur. Heart J.* **43**:228–235. DOI:10.1093/eurheartj/ehab803
57. Kato K., Nishino T., Otsuka T., et al. (2024). Nationwide analysis of the relationship between low ambient temperature and acute aortic dissection-related hospitalizations. *Eur. J. Prev. Cardiol.* **32**:zwae278. DOI:10.1093/eurjpc/zwae278
58. Xue X., Hu J., Peng L., et al. (2022). Low ambient temperature might trigger the symptom onset of pulmonary embolism: A nationwide case-crossover study at hourly level in China. *Sci. Total Environ.* **853**:158524. DOI:10.1016/j.scitotenv.2022.158524
59. Di Blasi C., Renzi M., Michelozzi P., et al. (2022). Association between air temperature, air pollution and hospital admissions for pulmonary embolism and venous thrombosis in Italy. *Eur. J. Int. Med.* **96**:74–80. DOI:10.1016/j.ejim.2021.09.019
60. Teng H. C. and Heyer H. E. (1955). The relationship between sudden changes in weather and the occurrence of acute myocardial infarction. *Am. Heart J.* **49**:9–20. DOI:10.1016/0002-8703(55)90049-1
61. Zhang N., Cao P., Zhao L., et al. (2023). Effect of temperature fluctuations in cold seasons on acute myocardial infarction hospitalisations in northeast China: A retrospective observational cohort study. *BMJ Open* **13**:e073528. DOI:10.1136/bmjopen-2023-073528
62. Cheng J., Su H., Xu Z., et al. (2021). Extreme temperature exposure and acute myocardial infarction: Elevated risk within hours. *Environ. Res.* **202**:111691. DOI:10.1016/j.envres.2021.111691
63. Lam H. C. Y., Chan J. C. N., Luk A. O. Y., et al. (2018). Short-term association between ambient temperature and acute myocardial infarction hospitalizations for diabetes mellitus patients: A time series study. *PLOS Med.* **15**:e1002612. DOI:10.1371/journal.pmed.1002612
64. Vassalle C., Grifoni D., Gozzini B., et al. (2024). Environmental temperature, other climatic variables, and cardiometabolic profile in acute myocardial infarction. *J. Clin. Med.* **13**:2098. DOI:10.3390/jcm13072098
65. Tseng C., Chen D., Chang S., et al. (2023). Ambient temperature effect on acute myocardial infarction by risk factors: daily data from 2000 to 2017, Taiwan. *JACC: Asia* **3**:228–238. DOI:10.1016/j.jacasi.2022.12.002
66. Dawson L. P., Andrew E., Nehme Z., et al. (2022). Temperature-related chest pain presentations and future projections with climate change. *Sci. Total Environ.* **848**:157716. DOI:10.1016/j.scitotenv.2022.157716
67. Cheng J., Bambrick H., Tong S., et al. (2020). Winter temperature and myocardial infarction in Brisbane, Australia: Spatial and temporal analyses. *Sci. Total Environ.* **715**:136860. DOI:10.1016/j.scitotenv.2020.136860
68. Yang M., He Q., Wu K., et al. (2025). Urban–rural disparities in the short-term effects of cold and heat on myocardial infarction mortality in Anhui Province, China. *Am. J. Epidemiol.* **194**:1933–1940. DOI:10.1093/aje/kwae108
69. The Eurowinter Group. (1997). Cold exposure and winter mortality from ischaemic heart disease, cerebrovascular disease, respiratory disease, and all causes in warm and cold regions of Europe. *Lancet* **349**:1341-1346.
70. Barnett A. G., Dobson A. J., McElduff P., et al. (2005). Cold periods and coronary events: an analysis of populations worldwide. *J. Epidemiol. Community Health* **59**:551–557. DOI:10.1136/jech.2004.028514
71. Chiu C. H., Vagi S. J., Wolkin A. F., et al. (2014). Evaluation of the National Weather Service extreme cold warning experiment in North Dakota. *Weather Clim. Soc.* **6**:22–31. DOI:10.1175/WCAS-D-13-00023.1
72. Chalabi Z., Hajat S., Wilkinson P., et al. (2016). Evaluation of the cold weather plan for England: Modelling of cost-effectiveness. *Public Health* **137**:13–19. DOI:10.1016/j.

puhe.2015.11.001
73. Saeki K., Obayashi K., Iwamoto J., et al. (2013). Influence of room heating on ambulatory blood pressure in winter: A randomised controlled study. *J. Epidemiol. Community Health* **67**:484–490. DOI:10.1136/jech-2012-201883
74. Milner J. and Wilkinson P. (2017). Commentary: Effects of home energy efficiency and heating interventions on cold-related health. *Epidemiology* **28**:86–89. DOI:10.1097/EDE.0000000000000570
75. Yan B., Chebana F., Masselot P., et al. (2020). A cold-health watch and warning system, applied to the province of Quebec (Canada). *Sci. Total Environ.* **741**:140188. DOI:10.1016/j.scitotenv.2020.140188
76. Quijal-Zamorano M., Petrova D., Martínez-Solanas È., et al. (2024). Forecast skill assessment of an operational continental heat-cold-health forecasting system: New avenues for health early warning systems. *Sci. Adv.* **10**:eado5286. DOI:10.1126/sciadv.ado5286
77. Masato G., Bone A., Charlton-Perez A., et al. (2015). Improving the health forecasting alert system for cold weather and heat-waves in England: A proof-of-concept using temperature-mortality relationships. *PLoS One* **10**:e0137804. DOI:10.1371/journal.pone.0137804
78. Murage P., Hajat S. and Bone A. (2018). Variation in cold-related mortality in England since the introduction of the cold weather plan: Which areas have the greatest unmet needs. *Int. J. Environ. Res. Public Health* **15**:2588. DOI:10.3390/ijerph15112588
79. Ryan E. C., Dubrow R. and Sherman J. D. (2020). Medical, nursing, and physician assistant student knowledge and attitudes toward climate change, pollution, and resource conservation in health care. *BMC Med. Educ.* **20**:200. DOI:10.1186/s12909-020-02099-0
80. Hampshire K., Ndovu A., Bhambhvani H., et al. (2021). Perspectives on climate change in medical school curricula—A survey of U. *S. medical students. J. Clim. Chang. Health* **4**:100033. DOI:10.1016/j.joclim.2021.100033
81. Barnett A. G., Lucas M., Platts D., et al. (2013). The benefits of thermal clothing during winter in patients with heart failure: A pilot randomised controlled trial. *BMJ Open* **3**:e002799. DOI:10.1136/bmjopen-2013-002799
82. Ryti N. R. I., Korpelainen A., Seppänen O., et al. (2020). Paradoxical home temperatures during cold weather: A proof-of-concept study. *Int. J. Biometeorol.* **64**:2065–2076. DOI:10.1007/s00484-020-01998-7
83. Vohra A. S., Chua R. F. M., Besser S. A., et al. (2020). Community health workers reduce rehospitalizations and emergency department visits for low-socioeconomic urban patients with heart failure. *Crit. Pathw. Cardiol.* **19**:139–145. DOI:10.1097/hpc.0000000000000220
84. Shi W., Wu L., Li X., et al. (2024). Community-embedded follow-up management intervention for geriatric primary care: A mixed-methods study of an integrated health services model. *BMC Health Serv. Res.* **24**:298. DOI:10.1186/s12913-024-10804-8
85. Haines A., Kimani-Murage E. W. and Gopfert A. (2024). Strengthening primary health care in a changing climate. *Lancet* **404**:1620–1622. DOI:10.1016/S0140-6736(24)02193-7
86. Reis da Silva T. H. (2024). The impact of cold weather on older people and the vital role of community nurses. *Br. J. Community Nurs.* **30**:28–34. DOI:10.12968/bjcn.2024.0003
87. Freijser L., Annear P., Tenneti N., et al. (2023). The role of hospitals in strengthening primary health care in the Western Pacific. *Lancet Reg. Health West. Pac.* **33**:100698. DOI:10.1016/j.lanwpc.2023.100698
88. Wang Y., Deng R. and Geng X. (2025). Exploring the integration of medical and preventive chronic disease health management in the context of big data. *Front. Public Health* **13**:1547392. DOI:10.3389/fpubh.2025.1547392
89. Benmarhnia T., Zhao X., Wang J., et al. (2019). Evaluating the potential public health impacts of the Toronto cold weather program. *Environ. Int.* **127**:381–386. DOI:10.1016/j.envint.2019.03.042
90. Toloo G., FitzGerald G., Aitken P., et al. (2013). Evaluating the effectiveness of heat warning systems: Systematic review of epidemiological evidence. *Int. J. Public Health* **58**:667–681. DOI:10.1007/s00038-013-0465-2
91. Bowen K. J. and Ebi K. L. (2015). Governing the health risks of climate change: Towards multi-sector responses. *Curr. Opin. Environ. Sustain.* **12**:80–85. DOI:10.1016/j.cosust.2014.12.001
92. Yang Z., Huang W., Xu R., et al. (2026). More than warming: Climate change as an emerging health crisis. *Innov. Med.* **4**:100206. DOI:10.59717/j.xinn-med.2026.100206
93. Heidenreich A. and Thieken A. H. (2024). Individual heat adaptation: Analyzing risk communication, warnings, heat risk perception, and protective behavior in three German cities. *Risk Anal.* **44**:1788–1808. DOI:10.1111/risa.14278
94. Lazo Green K., Tan M. M. C., Johnson E. E., et al. (2024). Interventions for cold homes: A rapid review of the health impacts. *Eur. J. Public Health* **34**:682–695. DOI:10.1093/eurpub/ckae058
95. Fan J., Xiao Y., Feng Y., et al. (2023). A systematic review and meta-analysis of cold exposure and cardiovascular disease outcomes. *Front. Cardiovasc. Med.* **10**:1084611. DOI:10.3389/fcvm.2023.1084611
96. Nitschke M., Krackowizer A., Hansen A. L., et al. (2017). Heat health messages: A randomized controlled trial of a preventative messages tool in the older population of South Australia. *Int. J. Environ. Res. Public Health* **14**:992. DOI:10.3390/ijerph14090992
97. Renn O. (2020). Risk communication: Insights and requirements for designing successful communication programs on health and environmental hazards. In Handbook of Risk and Crisis Communication, (Routledge), pp. 80-98.
98. Maibach E. W., Nisbet M., Baldwin P., et al. (2010). Reframing climate change as a public health issue: An exploratory study of public reactions. *BMC Public Health* **10**:299. DOI:10.1186/1471-2458-10-299
99. Zhang B., Luo W., Cai Y., et al. (2024). Global burden of adolescent and young adult cardiovascular diseases and risk factors: Results from Global Burden of Disease Study 2019. *Innov. Med.* **2**:100063. DOI:10.59717/j.xinn-med.2024.100063
100. Cappuccio F. P., Kerry S. M., Forbes L., et al. (2004). Blood pressure control by home monitoring: Meta-analysis of randomised trials. *BMJ* **329**:145. DOI:10.1136/bmj.38121.684410.AE
101. Koehler F., Koehler K., Prescher S., et al. (2020). Mortality and morbidity 1 year after stopping a remote patient management intervention: Extended follow-up results from the telemedical interventional management in patients with heart failure II (TIM-HF2) randomised trial. *Lancet Digital Health* **2**:e16–e24. DOI:10.1016/S2589-7500(19)30195-5
102. Ban J., Lan L., Yang C., et al. (2017). Public perception of extreme cold weather-related health risk in a cold area of Northeast China. *Disaster Med. Public Health Prep.* **11**:417–421. DOI:10.1017/dmp.2016.176
103. Luo Z., Li K., Chen A., et al. (2024). The influence of family health on self-efficacy in patients with chronic diseases: The mediating role of perceived social support and the moderating role of health literacy. *BMC Public Health* **24**:3398. DOI:10.1186/s12889-024-20906-x
104. Siegler E. L., Lama S. D., Knight M. G., et al. (2015). Community-based supports and services for older adults: A primer for clinicians. *J. Geriatr.* **2015**:678625. DOI:10.1155/2015/678625

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AUTHOR CONTRIBUTIONS

Tiantian Li, Xiaofeng Xu, and Junbo Ge contributed to the design of the statement. Meilin Yan, Qinghua Sun, Jing Yang, and Tiantian Li conceived the statement, participated in its design and coordination among co-authors, and helped draft the manuscript. All authors contributed to and approved the final manuscript.

DECLARATION OF INTERESTS

The authors declare no competing interests.

DATA AND CODE AVAILABILITY

No new data were created or analyzed in this study.