MINI REVIEW

# Cold weather exposures of cardiovascular disease patient's health

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**ABSTRACT**

Cold exposure, both acute and chronic, has an impact on cardiovascular responses, which can be influenced by underlying cardiovascular illness. Furthermore, exercising in a cold atmosphere raises cardiovascular strain even more, but the consequences on people with heart disease are unknown. In those with moderate hypertension, controlled trials using whole-body or local cold exposure show a similar or increased increase in cardiac workload, but worsened cutaneous vasoconstriction. A cold presser test with high sympathetic stimulation increases cardiac strain in people with Coronary Artery Disease (CAD), but not significantly different from those with less severe disease or those who are healthy. Cold exposure, on the other hand, lowers myocardial oxygen supply in CAD patients, potentially leading to ischemia. In people with CAD, exercising in the cold increases cardiac workload more than exercising in the heat. When performed in a temperature-neutral environment reduced myocardial perfusion, on the other hand, can lead to ischemia, angina, and poor performance. In the cold, having heart failure also affects submaximal and maximum performance. Antinational therapy lowers blood pressure in the cold, but it has little effect on the degree of cold-related cardiovascular reactions in hypertension. Similarly, taking blood pressure medication enhances exercise performance in the cold in both people with CAD and people with heart failure.

**Key Words:** *Heart Failure; Cardiovascular Disease; Hypertension; Cardiac Workload; Low Temperature; Coronary Artery Disease*

## INTRODUCTION

A higher incidence of cardiovascular illness and mortality has been recorded globally during the winter season, or in connection with extended episodes of extremely low temperatures (cold spells) [5,6]. The negative health effects of colds are frequently linked to cardiovascular reasons. Heart failure (HF), atrial fibrillation, ventricular arrhythmias, angina pectoris, Acute Myocardial Infarctions (AMI), and Sudden Cardiac Deaths (SCD) are all associated with a higher number of cardiac symptoms (angina, arrhythmias, or dyspnea) and health events such as hypertensive crisis, deep venous thrombosis, pulmonary embolism, aortic ruptures/dissection, Cold-related mortality outnumbers the adverse effects of heat in both global and national research. In contrast to popular belief, the majority of poor health effects are not associated with cold extremes even at milder non-optimal levels, temperature-related mortality occurs [1]. In healthy people, either an acute drop in temperature or its seasonal repercussions raise cardiovascular strain through physiological reactions aimed at maintaining thermal balance. These symptoms may be exacerbated in those who have cardiovascular disorders that affect the brain system, heart, and circulatory system. Cold exposure and activity both increase cardiovascular stress. Cardiovascular responses appear to be more potentiated during exercise in the cold compared to a warm environment, according to studies of healthy people. Cardiovascular disease may exacerbate these responses, but this has not been thoroughly investigated. The increased cardiovascular strain that comes with exercising in the cold may lead to negative health outcomes in people who already have heart disease. Sudden or intense exercise, such as working outdoors or participating in winter sports, for example, increases the risk of AMI. There has been little previous controlled study on the acute effects of cold alone and in combination with exercise in people with cardiovascular problems [2]. The current review reviews the available information from controlled trials looking at the effects of cold and exercise alone and in combination on cardiovascular responses in people with cardiovascular disease. The research considered in this review include controlled studies that include patients with Hypertension (HTN), Coronary Artery Disease (CAD), and Heart Failure (HF), include some sort of exercise (dynamic, isometric) [3], and use various types of cold exposure (whole-body, local). The results of investigations using the Cold Presser Test (CPT) are also compared to the results of studies using other cold stimuli that induce thermoregulatory responses.

Finally, the goal is to illustrate probable links to epidemiological research that show increased cardiovascular morbidity and mortality during the winter months. Cold air with or without wind, immersion in water, or contact with cold objects are examples of the types of exposure that humans are exposed to [4]. The type and intensity of the exposure, the physical activity used, and the protection provided by garment insulation are the most important elements determining body heat loss and cardiovascular responses in the cold. A variety of individual characteristics, including age, hydration and nutrition status, anthropometry, body composition, past adaption, fitness, health status, and medication use, all play a role in acute responses. Different types of cooling occur depending on the type of exposure, including whole-body cooling of skin (cold chamber experiments, water-perfusion suits), deeper body areas, or regional cooling of the facial region (e.g. external application of cold to the front head), hands (cold pressure test), or respiratory tract (e.g. inhalation of cold air while exercising in the cold). As a result, several cardiovascular reactions occur, which are briefly described below for healthy people [5].

## DISCUSSION

Despite a wealth of evidence confirming a link between low ambient temperature and negative cardiovascular health outcomes, particularly among people with cardiovascular disease, there are just a few controlled studies that evaluate the probable pathophysiological responses. For cardiovascular stimulation, many of them have employed the cold pressure test. Whole-body (with or without facial exposure) skin cooling, local cooling of skin or face cold water immersion in conjunction with sauna cardiovascular responses were compared to healthy controls in the majority of the trials [6], but not all of them. In many studies assessing the influence of medicine on cold-related cardiovascular responses, just one patient group was given different treatments. The bulk of the cardiovascular disease patients analyzed were middle-aged (mean age about 50 years), while a few studies looked at early disease development and included younger (20 years to 35 years old) subjects. Thing or brief strong whole-body cold exposures were all mentioned in the study (cry stimulation) [7].

Hypertension affects people all around the world, with 24% of males and 20% of women having high blood pressure. Changes in renal (e.g. excessive salt intake), neuronal (e.g. sympathetic over activity), vascular (endothelial dysfunction, vascular remodeling), and hormonal (activation of the Renin-angiotensin-aldosterone system, RAAS) function are the main pathophysiologic mechanisms. Hypertension can cause both minor end-organ damage and major events such as stroke, heart attack, kidney failure, and dementia. Both an immediate cold-induced rise in blood pres-

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-sure and a prolonged rise in blood pressure over the cold season may exacerbate hypertension deregulation and raise the risk of cold-related cardiovascular events. Hypertension, due to decreased autonomic cardiovascular control and typical sympathetic over activity, could result in increased cardiovascular reactions in response to acute cold exposure. Hypertension is linked to increased arterial stiffness and endothelial dysfunction, both of which contribute to the basic vascular tone [8], as well as the cold-induced pressure response. Furthermore, increased central vascular stiffness and pressure response may be a result of ageing in combination with hypertension. They found that hypertension patients had a higher increase in blood pressure (BP) and muscle sympathetic nerve activity (MSNA) than normotensive controls. They also looked into Bar Reflex Sensitivity (BRS), which is a major short-term control mechanism for blood pressure that might be disrupted in hypertension.

Their findings linked functional buffering of cooling-induced increases in BP to a cold-related rise in BRS in hypertensive, but not normotensive, participants. When hypertension participants were compared to normotensive subjects, the results revealed an exacerbated increase in skin sympathetic nervous activity (SSNA) and peripheral cutaneous vasoconstrictor response to skin cooling. The observed increased vasoconstriction was not caused by variations in cutaneous adrenergic sensitivity [9]. As a result, the authors hypothesized that this occurs as a result of increased cutaneous sympathetic output and a larger dependence on non-adrenergic neurotransmitters. The trials stated above were conducted with whole-body cooling of broad skin areas (water perfusion suit), but not the head. In contrast, analyses using whole-body cooling, which includes facial cooling and greater skin areas shielded with winter clothes, found that hypertension and normotensive patients had similar cardiovascular responses. This type of cold exposure causes autonomic co-activation, as seen by increased blood pressure, heart rate variability, and decreased HR in both untreated and treated hypertension participants when compared to normotensive subjects. Furthermore, hypertension had no effect on the cold-induced rise in augmentation index (A surrogate marker of aortic stiffness) or decrease in sub-endocardia viability ratio (a measure of myocardial oxygen supply/demand ratio). In contrast to prior findings, BRS rose in untreated hypertensive and normotensive participants in comparable amounts, implying that cardio protective mechanisms are intact in the early stages of the disease [10].

In conclusion, when compared to controls, hypertension causes an aggravated or similar increase in cardiovascular strain after whole-body cold exposure. In essential hypertension, local cooling appears to up regulate pathways related to endothelial dysfunction of the microcirculation. Variations in reactions may be due to changes in study populations (e.g., illness progression, medication use) and protocols, as well as whether or not cooling of the head is involved. The increased cardiac workload seen in the cold is mostly due to an increase in sympathetic activity and its impact on vasoconstriction and elevated blood pressure. Hypertensive people may have a higher risk of cardiovascular events due to their higher baseline blood pressure and cold sensitivity. Cold exposure to the face, for example, temporarily raised SBP above 200 mmHg in untreated moderately hypertensive patients. Antihypertensive drugs lower blood pressure but have no effect on how responsive the body is too cold during whole-body chilling or the cold pressure test. In the cold, proper protection may help to minimize the pressure reaction [11].

## CONCLUSION

In healthy people, the effects of cold exposure and exercise on cardiovascular responses are well recognized. The effects of cardiovascular illness on these reactions are less well understood. This knowledge aids in deciphering the pathophysiological reactions that may lead to a climate's globally proven increased cardiovascular morbidity and mortality. With the current information, more research into the types, intensity, and duration of cold exposure that mirror circumstances that people would encounter in their daily lives is needed. Furthermore, because regular exercise is beneficial to everyone's health, its importance for people with chronic diseases cannot be overstated. Health-improving exercise, according to undisputed research, is an effective method of secondary prevention, since it slows or stops the progression of cardiovascular illnesses and lowers the risk of death negative health consequences However, because both cold exposure and exercise raise circulatory strain, data on their combined effects is required. Currently, there is little research on aerobic performance in the cold, especially among people with cardiovascular illnesses. Congestive heart failure symptoms in the cold, for example, may hinder people from exercising regularly. Information on how hypertension affects cardiovascular responses during cold exercise is exceptionally rare. Another area of research would be to see how isometric exercise affects cardiovascular responses in the cold in those with cardiovascular problems. This would explain why, for people with cardiovascular illness, such as an ischemic heart condition, scenarios involving low temperatures and sudden or intensive exercise (such as snow removal) may pose a health risk. Individuals would benefit from the information produced if they exercised outside on a regular basis all year. It would also be useful for health care professionals when advising on proper cold-weather protection.

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