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Effects of climate change on cardiovascular health: a focus on temperature extremes

Efeitos das mudanças climáticas na saúde cardiovascular: um enfoque nos extremos de temperatura

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ABSTRACT

Introduction: Ambient temperature impacts cardiovascular health through multifaceted biological mechanisms, causing imbalances in thermal homeostasis, which impair the cardiovascular system. Temperature extremes lead to vascular changes, autonomic nervous system responses, arrhythmias, inflammation, and oxidative stress.

Objective: Analyze the existing scientific literature in order to synthesize the main information about the influence of climate change on cardiovascular events.

Method: Searches were conducted in the PubMed, ScienceDirect, Web of Science, and Scielo databases. The descriptors used were: "cardiovascular diseases, climate change, and body temperature regulation". The inclusion criteria comprised review articles published from 2005 onwards, in Portuguese and English. A total of 24 articles were selected.

Result: Cold causes vasoconstriction, tachycardia, and increased blood pressure, resulting in myocardial ischemia, especially in patients with coronary plaques. Cold exposure increases angiotensin-II, leading to vasoconstriction and hypertension. This mechanism overloads the myocardium and increases oxygen demand. Heat induces vasodilation and sweating, which increases cardiovascular demand due to elevated heart rate. Resulting dehydration alters hemoconcentration and electrolyte balance, triggering acute cardiovascular events.

Conclusion: Extreme cold and heat generate thermal stress, which induces systemic inflammation, contributing to the progression of cardiovascular diseases. Understanding the biological mechanisms that mediate the adverse effects of cold and heat on the cardiovascular system is essential for developing effective intervention strategies.

KEYWORDS: Cardiovascular diseases. Climate change. Body temperature regulation.

Central message

Climate change has significant impacts on cardiovascular health, especially during temperature extremes, especially impacting vulnerable populations such as the elderly and people with comorbidities. This study sheds light on how hot and cold flashes interfere with the proper functioning of the cardiovascular system and what the consequences of these changes are.

Perspective

Studies on climate change and cardiovascular disease indicate that exposure to temperature extremes is correlated with a significant increase in cardiovascular events such as stroke, heart failure, arrhythmias, and thrombus formation. Understanding these changes is of paramount necessity to predict and mitigate possible adverse effects triggered

RESUMO

Introdução: A temperatura ambiental impacta a saúde cardiovascular por mecanismos biológicos multifacetados, causando desequilíbrios na homeostase térmica, o que prejudica o sistema cardiovascular. Os extremos de temperatura geram alterações vasculares, respostas do sistema nervoso autônomo, arritmias, inflamação e estresse oxidativo.

Objetivo: Analisar as principais publicações sobre a influência das mudanças climáticas nos eventos cardiovasculares.

Método: Busca nas bases de dados PubMed, ScienceDirect, Web of Science e Scielo. Foram utilizados os descritores: "cardiovascular diseases, climate change e body temperature regulation". Os critérios de inclusão compreenderam artigos de revisão publicados a partir de 2005, em português e inglês. Foram selecionados 24 artigos.

Resultado: O frio provoca vasoconstrição, taquicardia e aumento da pressão arterial, resultando em isquemia miocárdica, especialmente em pacientes com placas coronárias. Exposição a ele aumenta angiotensina-II, levando à vasoconstrição e hipertensão arterial. Esse mecanismo sobrecarrega o miocárdio e aumenta a demanda de oxigênio. O calor induz vasodilatação e sudorese, o que aumenta a demanda cardiovascular pela elevação da frequência cardíaca. A desidratação decorrente altera a hemoconcentração e o balanço eletrolítico, desencadeando eventos cardiovasculares agudos.

Conclusão: Frio e calor extremos geram estresse térmico, que induzem inflamação sistêmica, contribuindo para progressão de doenças cardiovasculares. A compreensão dos mecanismos biológicos que mediam os efeitos adversos do frio e do calor sobre o sistema cardiovascular é essencial para o desenvolvimento de estratégias de intervenção eficazes.

PALAVRAS-CHAVE: Doenças cardiovasculares. Mudança climática. Regulação da temperatura corporal.

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INTRODUCTION

Cardiovascular diseases are the leading cause of death worldwide1 and have become major evils of this century in Brazil and in most developed countries. In addition, coronary heart disease is the leading cause of mortality in the world, according to the World Health Organization (WHO)[1], being the second in Brazil (12.4%), followed by stroke (9.1%).[2] In addition, in Brazil, according to data from the Ministry of Health, the circulatory system was responsible for 31.46% of deaths, with the largest cause being higher than the sum of the following 2 (neoplasms and external causes).[3] In addition, evidence indicates that for every 1°C increase in ambient temperature, there is a 2.1% increase in cardiovascular mortality and a 0.5% increase in cardiovascular morbidity. These associations are especially worrisome in tropical regions, where the risks are higher.[4]

Cardiovascular diseases are usually silent, with a slow evolution, with a long asymptomatic period, but they can be identified, treated and controlled before any complication or the outbreak of a serious event.[5]

The relationship between climate change and cardiovascular health is complex and multifaceted, impacting the human body in several ways, and can occur through numerous biological mechanisms.[6] It is known that they are usually associated with thermoregulatory efforts caused by exposure to extreme cold and heat and involve vascular changes, autonomic nervous system responses, arrhythmias, inflammation, and oxidative stress.[7]

The objective of this review was to analyze the existing scientific evidence on the influence of climate change on cardiovascular events.

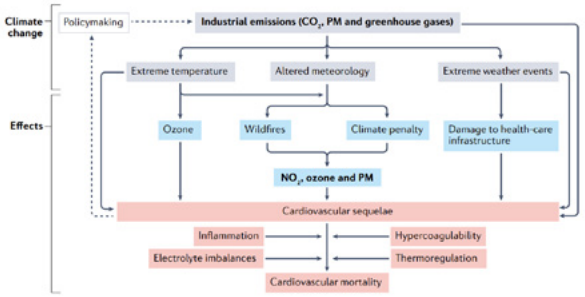
METHOD

This is a narrative review carried out in the PubMed, ScienceDirect, Web of Science and Scielo databases. The search used the following keywords: "cardiovascular diseases, climate change, body temperature regulation". The inclusion criteria included review articles from 2005 onwards, published in Portuguese and English, available in full text. Those who avoided the theme were excluded. In total, 24 articles were selected.

DISCUSSION

Mechanisms by which climate change affects the cardiovascular system include increased oxidative stress, inflammation, and hemodynamic changes (Figure). Exposure to extreme temperatures can disrupt thermal homeostasis, leading to dehydration and increased blood viscosity, which promotes thrombus formation.[8]

The fundamental mechanisms that contribute to the development of cardiovascular diseases in extreme temperature conditions are associated with several regulatory pathways. These include the sympathetic reaction to temperature, activation of the renin-angiotensin system (RAS) in response to cold, dehydration caused by both cold and heat, changes caused by extreme temperatures, and the systemic inflammatory response resulting from heat stroke.[10]



Source: Romanello et al.[9]

FIGURE — The effect of climate change on the development of cardiovascular diseases

In terms of direct impact, systemic inflammation caused by heat stress can increase the levels of several inflammatory markers, contributing to the progression of atherosclerosis. The most commonly elevated markers include C-reactive protein (CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and cell adhesion molecules such as intercellular adhesion molecule-1 (ICAM-1) and vascular adhesion molecule-1 (VCAM-1). C-reactive protein (CRP) is a marker of inflammation that is widely used in clinical practice. High levels of it are associated with a higher risk of cardiovascular events, such as myocardial infarction and stroke. IL-6 — a pro-inflammatory cytokine — is also often elevated in response to exposure to air pollutants and heat stress, promoting vascular inflammation and endothelial dysfunction. TNF-α is another key cytokine in systemic inflammation, contributing to endothelial cell apoptosis and atherosclerotic plaque formation. In addition, cell adhesion molecules such as ICAM-1 and VCAM-1 facilitate the adhesion of leukocytes to blood vessel walls, promoting inflammation and the progression of atherosclerosis.[11]

Exposure to cold causes vasoconstriction, tachycardia and increased blood pressure. These effects can lead to myocardial ischemia, especially in patients with coronary plaques, which can thicken and rupture. Changes in hemoconcentration (blood concentration of coagulation factors, platelets, cholesterol, fibrinogen, and erythrocytes) due to polyuria contribute to this process. In addition, during the colder seasons, the level of cholesterol in the blood is usually higher. These mechanisms can lead to increased vascular resistance and blood pressure, coronary spasms, the formation and rupture of plaques, and even thrombosis, which can cause cardiovascular events.[12]

Other mechanisms related to activations of the sympathetic system and the renin-angiostesine system have been associated with increased blood pressure and cardiovascular events, such as myocardial infarction. Research has shown increased sympathetic activation and angiotensin-II levels after exposure to cold. Mechanisms related to the growth of atherosclerotic plaque, in addition to increased instability and the possibility of plaque rupture during

cold, have been pointed out.[13]

The body, aiming to generate heat in the coldest periods, intensifies muscle activity, thus causing overload on the heart.[14] In addition to the vasoconstriction caused by lower air temperatures, there are also physiological factors that change with low temperatures. The second would be the greater activation of clot-forming factors, causing a greater occurrence of ischemic diseases, such as infarction.[15]

Studies indicate that exposure to cold air can significantly increase plasma angiotensin-II levels.[16,17] At low temperatures, the release of norepinephrine and the activation of the renin-angiotensin system can cause systemic vasoconstriction and arterial hypertension. This increases myocardial work and oxygen demand, increasing the risk of cardiovascular and cerebrovascular events. Interestingly, epinephrine levels do not appear to be affected by lower temperatures, suggesting that the increase in norepinephrine may be due to the release of sympathetic nerve endings rather than secretion from the adrenal medulla.[18]

Exposure to heat, on the other hand, causes vasodilation and sweating. These mechanisms aim to reduce the increase in core temperature, but imposing acute demands on the cardiovascular system. In this chain of events, blood accumulates in the cutaneous region, reducing the central blood volume, in the chest and abdomen region. Due to this reduction, cardiac output is improved through increased heart rate. This may be one of the reasons why heart failure patients respond poorly to heat stress. Heat induces sweating and increased blood volume in the skin regions, which cause dehydration, increased blood viscosity, hypotension, and increase cardiac overload, which are also related to cardiovascular problems. Exposure to heat also causes changes in hemoconcentration, related to dehydration caused by sweating. Also in relation to hydration, changes in electrolyte and acid-base balance can be considered as a trigger for acute cardiovascular events.[19]

In addition, exposure to extreme heat can lead to dehydration and increased blood viscosity, promoting the formation of thrombi and, consequently, acute cardiovascular events. On the other hand, intense cold waves also contribute to cardiovascular mortality, as they can cause constriction of blood vessels and increased blood pressure, exacerbating the risk of heart attacks and strokes.[20] Therefore, dehydration contributes to cardiovascular risk not only through volume depletion, but also through hemoconcentration and increased blood viscosity. Hyperthermia can cause electrolyte abnormalities, increasing the risk of cardiac arrhythmias.[21,22] Hypothermia, on the other hand, can induce bradycardia and trigger potentially fatal arrhythmias. Changes in the electrocardiogram are seen in individuals with low body temperatures.[23] Heat can cause changes in heat-sensitive proteins, activating an inflammatory cascade that contributes to multi-organ failure.[23,24] Heat stroke is a severe heat-related condition that leads to multiple organ dysfunction and encephalopathy, mainly affecting the elderly, chronically ill patients, or patients with cardiovascular disease.[23,25]

CONCLUSION

Temperature variations have a significant impact on cardiovascular health, especially among vulnerable populations, such as the elderly and people with preexisting comorbidities. After observing the pathophysiological mechanisms of cold and heat on metabolism, it was defined that extreme cold is associated with an increase in the incidence of myocardial infarction, arrhythmias and stroke, and on the other hand, extreme heat can cause electrolyte disturbances and thermal overload, leading to a higher incidence of heart failure, arrhythmias and also stroke. In this sense, it is essential to understand these biological mechanisms for the development of effective intervention strategies, and it is crucial to implement preventive measures, such as monitoring cardiovascular health during hot and cold waves, especially in risk groups. By prioritizing these actions, it will be possible to mitigate the adverse effects of climate change on the cardiovascular health of the population.

Authors' contributions

Evelyn Kloster Mazur: Conceptualization
Homero Grein Neto: Formal analysis
Sofia Sant' Anna Pofahl Biscaro: Research
Methodology: Bruno Henrique Strapasson Moraes
Isabela Bonk Palma: Project Administration
Luiz Eduardo Latorre Guimarães: Writing (original draft)
All authors: Writing (proofreading and editing)

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