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THE IMPACT OF STRESS ON THE DEVELOPMENT OF CARDIOVASCULAR DISEASES

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ABSTRACT

Background: Cardiovascular disease (CVD) remains the leading cause of death worldwide despite advances in the identification and treatment of classic risk factors. Accumulating evidence suggests that psychosocial stress may play a significant role in the development and progression of CVD through biological and behavioral mechanisms.

Objective: The aim of the study was to summarize the current knowledge on the relationship between psychosocial stress and cardiovascular diseases, with particular emphasis on the pathophysiological mechanisms underlying this relationship and the possibilities of stress reduction in the prevention and treatment of CVD.

Methods: A narrative literature review was conducted using PubMed, Nature Reviews, and Google Scholar. Scientific publications published primarily between 2014 and 2025 were analyzed, including systematic reviews, meta-analyses, cohort studies, clinical trials, experimental studies, and epidemiological reports. Papers on the biological and psychological mechanisms linking psychosocial stress with cardiovascular diseases and interventions aimed at reducing stress were included.

Results: Psychosocial stress should be viewed as a multidimensional risk factor for cardiovascular disease, potentially contributing to both its development and progression. Incorporating stress assessment and implementing effective stress reduction strategies may be a valuable complement to current CVD prevention and treatment strategies. Further research involving large patient populations is needed to determine the role of stress-reducing interventions in routine cardiac care.

KEYWORDS

Psychosocial Stress, Cardiovascular Diseases, Stress Management Interventions

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1. Introduction

Psychosocial stress is most simply defined as a state of imbalance between environmental demands and an individual's adaptive capabilities, leading to psychological and physiological responses. According to Hans Selye, who first coined this term, stress was the body's nonspecific response to any demand placed upon it. Contemporary definitions, however, emphasize the importance of individual assessment of stressful situations and their impact on a person's mental and somatic functioning [1-2]. Psychosocial stress and its negative impact on the development and course of disease have been the subject of intensive scientific research for many decades. Researchers are particularly interested in the relationship between stress and cardiovascular diseases (CVD). CVD encompasses a diverse group of pathologies affecting the heart and blood vessels, such as coronary heart disease, stroke, peripheral vascular disease, rheumatic heart disease, and pulmonary embolism [3].

Despite the identification and potential elimination of many major risk factors, such as hypercholesterolemia, diabetes, obesity, hypertension, smoking, and poor diet, cardiovascular disease remains the leading cause of death worldwide, accounting for approximately 32% of all deaths [4-6]. The Global Burden of Diseases, Injuries, and Risk Factors Study (GBD), which analyzed the burden of cardiovascular disease in 204 countries, found that CVD was responsible for approximately 19.2 million deaths in 2023, while the number of people living with these diseases was approximately 626 million. At the same time, the number of CVD cases has more than doubled since 1990 [7-8]. Due to the enormous public health burden of cardiovascular disease, there is a search for new, often unusual risk factors whose identification could help reduce morbidity and mortality. Among these, psychological stress is receiving increasing attention. Current research indicates that both acute and chronic stress can influence the development and progression of CVD through sympathetic nervous system activation, hormonal disruptions, chronic inflammation, endothelial dysfunction, and adverse metabolic changes [9]. This article presents the current state of knowledge regarding the impact of psychosocial stress on the development of cardiovascular disease and discusses the potential mechanisms responsible for this relationship and methods for reducing it.

2. Methodology

This paper is a narrative review of the literature on the impact of psychosocial stress on the development, course, and prognosis of cardiovascular disease. The analysis was based on scientific publications published primarily between 2014 and 2025, supplemented by classic works that form the basis of the contemporary understanding of stress and its biological consequences.

A literature search was conducted using the PubMed, Nature Reviews and Google Scholar databases. The following keywords and their combinations were used in the search: "stress," "psychosocial stress," "chronic stress," "cardiovascular disease," "coronary artery disease," "myocardial infarction," "stroke," "heart rate variability," "autonomic nervous system," "hypothalamic-pituitary-adrenal axis," "endothelial dysfunction," "inflammation," "mental stress-induced myocardial ischemia," "stress management interventions," and "cardiovascular risk factors."

Review articles, meta-analyses, systematic reviews, cohort studies, clinical trials, and experimental studies examining the biological and psychological mechanisms linking stress to cardiovascular disease were included in the analysis. Epidemiological reports and studies from international health organizations regarding the global burden of cardiovascular disease were also included.

3. Results

3.1. Physiological mechanisms of stress in cardiovascular diseases

Large population-based studies have shown that adults exposed to chronic stress at work or in their personal lives have a 1.1 to 1.6-fold increased risk of developing coronary heart disease and stroke [10]. These findings are supported by a review of over 600,000 men and women participating in 27 cohort studies conducted in Europe, the United States, and Japan. Kivimäki and Kawachi demonstrated that occupational stressors, such as job strain and long work hours, are associated with a moderate increase in the risk of coronary heart disease and stroke. The increased risk in exposed individuals ranged from 10 to 40% compared to unexposed individuals, ruling out the association between age, gender, and socioeconomic status [11]. Despite the established association between stress and CVD, its significance in the general population appears to be lower than that of classic risk factors such as hypertension, smoking, or hypercholesterolemia [5]. At the same time, available data indicate that the impact of stress on the primary development of CVD is particularly pronounced when exposed early in life. Unlike stress experienced in childhood, stress in adulthood more often serves as a trigger for cardiovascular events in individuals initially at increased cardiovascular risk, rather than directly initiating the disease process [10, 12-14].

Although the number of studies confirming the association between stress and the occurrence of cardiovascular disease is constantly growing, the pathophysiological mechanisms underlying this relationship have not yet been fully confirmed. Current knowledge indicates that stress affects the cardiovascular system both directly, through activation of the sympathetic nervous system, neurohormonal disruptions, and the induction of chronic inflammation, and indirectly, influencing health behaviors and lifestyle. The most important behavioral consequences of chronic stress include poor eating habits, reduced physical activity, poor sleep quality, and an increased tendency to use stimulants. Below, we describe the most important elements of the body's stress response and discuss the current evidence regarding the pathophysiological mechanisms by which stress may contribute to both the development of atherosclerosis and the triggering of acute cardiovascular events.

3.2. Sympathetic nervous system activation

The sympathetic nervous system is the first element of the body's response to a stressor, becoming activated almost immediately after exposure to the stressor. It is responsible for initiating the "fight or flight" response, enabling the body to quickly adapt to threatening situations. Besides spontaneous activation, sympathetic nervous system activity also follows a circadian rhythm, peaking in the morning and gradually decreasing throughout the day. This phenomenon is consistent with the observed circadian rhythm in myocardial infarctions [15]. Sympathetic activation leads to increased secretion of catecholamines, primarily adrenaline and noradrenaline, which, by stimulating α - and β -adrenergic receptors, exert a positive chronotropic and inotropic effect and enhance vasoconstriction. This leads to an acceleration of heart rate, increased force of contraction, and increased blood pressure [10, 15-17]. Sustained activation of the

sympathetic nervous system can lead to chronic elevations in blood pressure, contributing to the development of hypertension, which is one of the most important modifiable risk factors for cardiovascular disease.

Increased sympathetic activity is accompanied by a simultaneous decrease in parasympathetic activity, particularly the vagus nerve. One of the most commonly used indicators of autonomic nervous system balance is heart rate variability (HRV), which reflects the body's ability to adapt to changing environmental conditions. Chronic stress leads to decreased HRV, which indicates reduced flexibility of autonomic regulation and a limited ability of the body to regenerate and maintain homeostasis [15,18-19]. The clinical importance of HRV has been confirmed in numerous studies, demonstrating its association with both the incidence of cardiovascular disease and cardiovascular mortality [20-21]. Reduced HRV is associated with an increased risk of heart rhythm disturbances, disease progression, and poor prognosis, particularly in patients with established CVD [26]. Additional support is provided by the results of a meta-analysis assessing the effectiveness of stress management interventions (SMI), which demonstrated a small but statistically significant improvement in HRV parameters in patients with cardiovascular disease [15]. Changes in the functioning of the autonomic nervous system also play a significant role in the pathogenesis of arrhythmias triggered by psychological stress and negative emotional states. Ventricular arrhythmias are of particular clinical importance, significantly increasing the risk of sudden cardiac death [15, 22]. Psychological stress has been shown to influence the occurrence of T-wave alternans, defined as alternating changes in the amplitude or morphology of the T wave in the electrocardiographic recording from one cardiac cycle to the next. This reflects the instability of the ventricular repolarization process and is considered a marker of an increased risk of life-threatening ventricular arrhythmias. The importance of this mechanism is particularly important in patients with existing heart disease and those with implantable cardiac devices, such as implantable cardioverter-defibrillators (ICDs), in whom stress may increase susceptibility to serious cardiac arrhythmias [22].

Taken together, these observations indicate that autonomic imbalance induced by chronic stress may be one of the important mechanisms linking psychosocial stress with an increased risk of developing and progressing cardiovascular disease.

3.3. Hypothalamic-pituitary-adrenal axis

Activation of the hypothalamic–pituitary–adrenal axis (HPA) is the next stage of the body's response to stress. It develops with a certain delay compared to the rapid activation of the sympathetic nervous system, usually within minutes of an acute stress stimulus, and occurs with the participation of adrenal cortex hormones.

Cortisol is the main glucocorticosteroid of the adrenal cortex. It plays a key role in coordinating the body's response to stress. However, its effects extend beyond the regulation of adaptive processes and also encompass a range of metabolic and hemostatic effects. Chronic exposure to stress disrupts the regulation of the HPA axis, resulting in persistent elevations in blood cortisol concentrations. Elevated cortisol levels have been shown to lead to increased blood pressure and glucose levels, as well as to enhance platelet activation, fibrinogen levels, blood viscosity, and the activity of coagulation factors [10]. These changes promote the development of a prothrombotic state, which may increase the risk of thromboembolic events, including acute coronary syndromes resulting from the occlusion of a coronary artery. The importance of chronic exposure to glucocorticoids in the development of cardiovascular disease is confirmed by observations in patients with Cushing's syndrome. This condition is characterized by long-term exposure to excessive glucocorticoid concentrations and is associated with an up to five-fold higher incidence of cardiovascular disease compared to the general population [3, 23].

Despite the important regulatory role of glucocorticoids in suppressing the stress response through a negative feedback mechanism within the HPA axis, their chronically elevated levels also cause numerous adverse metabolic consequences. The most important of these include weight gain, the development of hypertension, and insulin resistance, which are recognized risk factors for cardiovascular disease [13]. Consequently, long-term activation of the HPA axis may be one of the key mechanisms linking chronic psychosocial stress with an increased risk of developing and progressing cardiovascular diseases.

3.4. Endothelial dysfunction and inflammation

Endothelial cells play a fundamental role in maintaining vascular homeostasis, demonstrating the ability to rapidly recognize both endogenous and exogenous stress stimuli and adapt their function to changing environmental conditions. Disruption of these mechanisms leads to the development of endothelial dysfunction.

The primary mechanism of dysfunction involves an imbalance between vasodilatory and vasoconstrictive factors. Stress promotes increased secretion of potent vasoconstrictive factors, such as endothelin-1 and angiotensin II, and also reduces the bioavailability of nitric oxide (NO) [24-25]. Consequently,

this leads to impaired regulation of vascular tone, increased thrombotic processes, and the activation of the inflammatory response [13,15]. Increasing evidence indicates that chronic psychosocial stress may contribute to the development of endothelial dysfunction both directly and indirectly. Indirect mechanisms include activation of the sympathetic nervous system, increased production of reactive oxygen species leading to oxidative stress, and increased chronic inflammation. The importance of these mechanisms is supported by human studies, which have demonstrated that acute psychological stress can lead to transient endothelial dysfunction in both the coronary and peripheral circulation. Furthermore, stress increased the reactivity of vascular smooth muscle cells, which can result in episodes of angina pectoris and myocardial ischemia [26].

The close interaction between the sympathetic nervous system and the immune system also plays a crucial role in this process. Norepinephrine released in response to stress activates immune cells and initiates a cascade of inflammatory processes, while the hypothalamic-pituitary-adrenal axis modulates the immune response via glucocorticoids. Both chronic and acute stress, through activation of β -adrenergic receptors, can stimulate the production of inflammatory cells, enhance leukopoiesis, and increase the secretion of proinflammatory cytokines [10,12,24,27]. These cytokines play a significant role in the development and progression of atherosclerosis, contributing to increased plaque instability and the risk of plaque rupture [13]. Experimental studies also provide evidence for the link between stress and the severity of inflammatory processes. In animal models, chronic exposure to stress has been shown to lead to increased inflammation within the arterial wall. One study observed the development of premature atherosclerotic lesions characterized by increased inflammatory cell infiltration in mice subjected to long-term exposure to dominant individuals [27-28].

3.5. Accumulation of classic risk factors

The impact of stress on the development of cardiovascular disease is not limited to direct neurohormonal, hemodynamic, and inflammatory mechanisms. Growing evidence indicates that chronic psychosocial stress may also indirectly increase cardiovascular risk by promoting the occurrence and accumulation of classic risk factors for cardiovascular disease. Consequently, stress should be viewed as a significant element of a multifactorial pathogenetic process leading to the development of metabolic disorders, adverse changes in health behaviors, and acceleration of atherosclerotic processes [10,13].

The relationship between chronic stress and metabolic syndrome is of particular interest. Metabolic syndrome is defined as the coexistence of at least three of the following disorders: central obesity, hypertension, hyperglycemia, hypertriglyceridemia, and low HDL cholesterol. The Whitehall II study of British civil servants demonstrated a dose-response relationship between chronic occupational stress combined with low social support and the risk of developing metabolic syndrome. This risk increased with the frequency of stress reporting during subsequent follow-up periods. Importantly, metabolic syndrome accounted for approximately 15% of the association between occupational stress and the occurrence of coronary heart disease. In the same cohort, stress was also found to be associated with larger waist circumference, higher body mass index, and a more unfavorable lipid profile [11]. Despite these observations, the relationship between stress and obesity has not been unequivocally confirmed. A meta-analysis of cohort studies encompassing over 60,000 participants found no significant association between occupational stress and weight gain. Similar results were obtained in an analysis encompassing approximately 150,000 individuals from countries with varying socioeconomic levels, which found only a small association between chronic stress and obesity overall, with no evidence of an independent effect of stress on the development of abdominal obesity with a predominance of visceral fat [11]. These results suggest that the effect of stress on body weight and other anthropometric parameters is not clear-cut and may be influenced by a number of additional environmental, genetic, and behavioral factors.

At the same time, numerous studies indicate that chronic stress promotes the occurrence of unfavorable health behaviors, which indirectly increase the risk of metabolic disorders and cardiovascular disease. Individuals exposed to long-term stress are more likely to develop unhealthy eating habits, preferring high-calorie, processed foods. Furthermore, they exhibit lower levels of physical activity and are less likely to adhere to therapeutic recommendations [10,26]. They are also more likely to smoke tobacco and consume alcohol in amounts considered harmful to health [10,13]. These behaviors may contribute to the development of obesity, dyslipidemia, insulin resistance, and type 2 diabetes, which are recognized cardiovascular risk factors. As a result, individual risk factors mutually reinforce each other, significantly increasing the likelihood of cardiovascular events.

The accumulated data therefore indicate that stress should be viewed not only as a potential, independent risk factor for cardiovascular disease, but also as a factor contributing to the accumulation and exacerbation of classic cardiovascular risk factors. The multifaceted impact of stress on metabolic processes, health behaviors, and neurohormonal regulation may partially explain the observed increase in CVD morbidity and mortality in individuals chronically exposed to psychosocial stress.

3.6. Prevention and stress reduction strategies

The foundation of contemporary secondary prevention of cardiovascular disease is a comprehensive approach, encompassing pharmacotherapy and lifestyle modification. The use of statins, acetylsalicylic acid, beta-blockers, and antihypertensive medications tailored to the individual patient's needs, combined with adherence to healthy lifestyle recommendations such as the Mediterranean diet, at least 150 minutes of moderate-intensity physical activity per week, and smoking cessation, forms the basis of long-term care for patients with diagnosed cardiovascular disease or at high risk of developing it [29-30]. Similarly, the beneficial impact of cardiac rehabilitation on the prognosis of patients following acute myocardial infarction, percutaneous coronary intervention (PCI), and cardiac surgery has been documented. Participation in cardiac rehabilitation programs is associated with a faster return to function, improved quality of life, and a reduced risk of further cardiovascular events [31]. Unlike the aforementioned interventions, the effectiveness of which has been confirmed in numerous clinical trials, there is still a lack of clearly standardized methods of treatment aimed at reducing psychosocial stress. This can also be difficult to achieve in clinical practice. Kimivaki and Steptoe point out that stress management interventions (SMI) are time-consuming and may require changing long-established habits, and adherence to recommendations can be poor even with treatments as simple as taking a pill once a day to lower blood pressure or cholesterol [10].

Kavitha M. Chinnaiyan highlights the concept of psychological resilience, a low level of which is primarily responsible for the link between emotional states and stress and CVD. Resilience is defined as the ability to cope with stress, life challenges, and the ability to quickly return to balance after crises. The author suggests building resilience as one of the methods for primary and secondary prevention of cardiovascular disease. In her article, she highlights the need to distinguish two action strategies: population-based and targeted. While the former, targeted at the general population, would depend more on state action on behalf of citizens, such as reducing working hours, promoting occupational health and providing appropriate equipment, an individual strategy would require assessing the stress level of cardiac patients and selecting a method from among psychotherapy, meditation, and tai-chi [5].

Meditation has the most research on the effectiveness of stress reduction. It has been proven to lower blood pressure, likely reducing sympathetic activity, and thus increasing HVR [5]. Mindfulness-based stress reduction, combining breathing techniques with meditation, has demonstrated benefits in the general population and was proposed by the authors of the Mindful Heart Study report as a complementary intervention [49]. Another randomized, single-blind, controlled trial in a group of 60 cardiac patients described the beneficial effect of mindfulness-based stress reduction on normalizing blood pressure and perceived stress. The beneficial effects of stress-reduction interventions were also confirmed in the ENHANCED study, which assessed the effectiveness of stress management training as an adjunct to standard cardiac rehabilitation. The stress management program included health education, group support, and elements of cognitive-behavioral therapy. Patients participating in both cardiac rehabilitation and stress management training achieved greater psychological and physiological benefits than those receiving standard rehabilitation alone. Importantly, a time-to-event analysis showed that participants randomly assigned to the additional stress-reduction intervention had a lower incidence of adverse cardiovascular events (18%) compared to patients receiving cardiac rehabilitation alone [32].

Some of the basic principles of the interventions also appear to have a beneficial effect on stress reduction. The positive impact of regular physical activity on overall health and well-being is well documented. Growing evidence also indicates that both individual and cumulative sessions of physical activity can produce significant stress-reduction effects. In addition, some studies suggest that high levels of cardiorespiratory fitness (CRF) may beneficially modulate the response to chronic stress [33]. One randomized controlled trial examined the physiological response to psychosocial stress in healthy men ($n = 96$). The study provided experimental evidence that regular endurance activity may increase the body's resistance to psychosocial stress. The authors demonstrated that a 12-week training program led to a weakened response of both the HPA axis and the autonomic nervous system to stress [33-34]. In light of the available evidence, regular physical activity should be viewed as a multifaceted intervention, encompassing both the modification of traditional cardiovascular risk factors and the reduction of the adverse consequences of chronic psychosocial stress.

4. Discussion

The presented data indicate that psychosocial stress is an important factor influencing the development and course of cardiovascular diseases. Although its importance in the general population is lower than that of classic risk factors such as hypertension, smoking or hypercholesterolemia, chronic exposure to stress is associated with an increased risk of coronary heart disease and stroke [10, 11]. The accumulation of stressors and their long-term impact on the body may be of particular importance.

Available research indicates that the impact of stress on the cardiovascular system is multifactorial. Chronic activation of the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis play a key role, leading to hemodynamic disorders, endothelial dysfunction, increased inflammatory processes and prothrombotic changes [10, 12, 13]. At the same time, stress promotes unfavorable health behaviors, such as limiting physical activity, improper diet, smoking or excessive alcohol consumption. Another important observed phenomenon is the impact of stress on the autonomic regulation of heart function. Decreased HVR and increased susceptibility to arrhythmias may partially explain the association between chronic stress and a higher incidence of cardiovascular events, especially in patients with pre-existing heart disease [18–22].

The results of intervention studies suggest that activities aimed at reducing stress, including stress coping training, mindfulness techniques and regular physical activity, may have a beneficial effect on the level of stress and selected prognostic indicators in patients with cardiovascular diseases [5,18,32–34]. However, further research is necessary to determine the place of these interventions in standard cardiological management. Further multi-center studies involving large patient populations are necessary to more precisely determine the place of the described and other interventions of modern psychology and psychiatry in the management of patients with CVD.

In conclusion, psychosocial stress should be viewed as a multidimensional risk factor that may contribute to the development and progression of cardiovascular diseases. Taking into account stress assessment and its effective reduction may be a valuable addition to contemporary cardiovascular prevention strategies.

5. Conclusions

Psychosocial stress is a significant factor in the development and progression of CVD. Its effects include both direct biological mechanisms, such as activation of the sympathetic nervous system, the hypothalamic-pituitary-adrenal axis, endothelial dysfunction, and chronic inflammation, and indirect effects through the exacerbation of classic cardiovascular risk factors. Available data suggest that interventions aimed at reducing stress may be a valuable adjunct to standard CVD prevention and treatment. However, further research in large populations is needed to determine the place of stress assessment and treatment in routine cardiac care.

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