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Laboratory Markers of Chronic and Acute Stress: Diagnostic Value and Clinical Implications (Part 1: Pathophysiology of Acute and Chronic Stress in the Context of Its Influence on Cardiovascular System)

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Abstract: *chronic stress significantly impacts human health by dysregulating the hypothalamic-pituitary-adrenal (HPA) axis and autonomic nervous system (ANS), leading to neuroendocrine, immunological and metabolic imbalances. Chronic hyperactivity of this system increases cardiac output, induces vasoconstriction, and raises blood pressure, gradually leading to vascular remodeling, myocardial hypertrophy and an elevated risk of cardiovascular events. To explore the diagnostic and pathophysiological significance of neuroendocrine biomarkers in the assessment of stress and its influence on the cardiovascular system. A comprehensive literature review of 76 literature sources in English was conducted using PubMed, Scopus, Web of Science and Google Scholar, focusing on the relationship between the hypothalamic-pituitary-adrenal (HPA) axis, the autonomic nervous system (ANS) and chronic stress; the search included peer-reviewed publications from 2020 to 2025 with keywords including "acute stress", "chronic stress", "HPA axis dysregulation", "autonomic nervous system", "chronic stress", "cortisol", "epinephrine", "norepinephrine", "dehydroepiandrosterone", "dopamine", "aldosterone", "tumor necrosis factor alpha", "interleukin-1", "interleukin-6", "C-reactive protein", "insulin-like growth factor-1", "cholesterol", "albumin", "glycosylated hemoglobin" and "cardiovascular system"; the inclusion criteria encompassed original research studies, systematic and narrative reviews, meta-analyses and clinical guidelines; non-peer-reviewed sources and publications not in English were generally excluded. Neuroendocrine biomarkers provide essential insights into the physiological burden of stress and the functioning of the HPA axis and ANS. Cortisol remains the most established biomarker for both acute and chronic stress, with hair cortisol offering unique advantages for long-term assessment. Catecholamines reflect acute sympathetic activation, but their diagnostic value in chronic stress is limited. Neuroendocrine markers enhances clinical decision-making and may support personalized strategies in stress-related diseases prevention and management. Early detection of elevated neuroendocrine markers associated with chronic stress may provide valuable insight into the mechanisms underlying increased cardiovascular risk and support more effective management of patients with cardiovascular disease.*

Keywords: Physiology; Neuroendocrinology; Hormones; Cardiovascular System; Cardiovascular Diseases; Stress.

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Introduction

Chronic stress is one of the key factors influencing human health, gradually depleting the body's adaptive capabilities and contributing to the development of numerous somatic diseases [1]. Physiological stress is the body's adaptive response to internal or external challenges, divided into acute and chronic stress. Acute stress is a short-term reaction that activates the sympathetic nervous system and hypothalamic-pituitary-adrenal (HPA) axis, temporarily increasing cortisol and heart rate (HR) before the body returns to homeostasis. In contrast, chronic stress persists over time, leading to sustained cortisol elevation, oxidative stress, and damage to physiological systems. While acute stress is generally protective, chronic stress contributes to aging, neurodegenerative diseases, and other health issues. Managing stress effectively is essential for preventing long-term health complications. Unlike acute stress, which triggers a short-term mobilization of physiological resources, chronic stress leads to prolonged alterations in the functioning of the neuroendocrine, immune and cardiovascular systems [2, 3]. Persistent exposure to stressors results in the exhaustion of adaptive mechanisms, hormonal imbalances, and dysregulation of inflammatory processes [3]. These changes create a foundation for the onset of severe conditions such as hypertension, ischemic heart disease, metabolic syndrome, depressive disorders, and even autoimmune diseases [3]. The pathogenesis of chronic stress is primarily driven by dysfunction of the HPA [1, 2, 3]. Continuous stimulation of this system leads to excessive cortisol production, which initially facilitates energy mobilization but eventually disrupts carbohydrate, lipid, and protein metabolism [2]. Chronically elevated cortisol levels suppress the hypothalamic negative feedback mechanism, further amplifying its secretion. Additionally, an imbalance between cortisol and dehydroepiandrosterone (DHEA) negatively impacts neuroplasticity, cognitive function, and emotional stability [4]. Beyond HPA axis activation, the sympathetic nervous system plays a crucial role in the pathogenesis of chronic stress by promoting the release of catecholamines-adrenaline, noradrenaline, and dopamine [5]. Chronic hyperactivity of this system increases cardiac output, induces vasoconstriction, and raises blood pressure, gradually leading to vascular remodeling, myocardial hypertrophy and an elevated risk of cardiovascular events [6]. Chronic stress, aging, and the development of neurodegenerative diseases are closely linked to persistently elevated cortisol levels in the blood [7]. Activation of HPA axis leads to the secretion of cortisol, the consequence of this is oxidative stress. Increased level of cortisol contributes to the release of reactive oxygen species (ROS) [8], which may damage DNA, RNA and proteins, thus, contributing to aging and development of age-associated diseases [9]. Oxidative stress, exacerbated by prolonged catecholamine elevation, contributes to vascular inflammation and the progression of atherosclerosis [10, 11]. Chronic stress also contributes to brain changes that may lead to anxiety and depression [12], impact reproductive system [13] and pathogenesis of autoimmune or inflammatory disorders [14, 15]. The diagnosis of chronic stress relies on a comprehensive analysis of neuroendocrine, immunological and metabolic markers. Key neuroendocrine markers include cortisol levels in various biological fluids (hair, saliva, serum, sweat, urine), DHEA concentrations and the cortisol/DHEA ratio, which reflects the balance between catabolic and anabolic processes [16]. A detailed analysis of laboratory markers of chronic stress allows for the assessment of physiological stress burden and the development of personalized strategies for preventing and managing stress-related disorders.

Materials and Methods

A comprehensive literature review of 76 literature sources in English was conducted using using PubMed, Scopus, Web of Science and Google Scholar to gather relevant articles for this manuscript. The search focused on the relationship between the HPA axis, the ANS and chronic stress. The keywords "acute stress", "chronic stress", "HPA axis dysregulation", "autonomic nervous system", "chronic stress", "cortisol", "epinephrine", "norepinephrine", "dehydroepiandrosterone", "dopamine", "aldosterone", "tumor necrosis factor alpha", "interleukin-1", "interleukin-6",

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"C-reactive protein", "insulin-like growth factor-1", "cholesterol", "albumin", "glycosylated hemoglobin" and "cardiovascular system" were utilized. The search was restricted to peer-reviewed articles published between 2020 and 2025. To ensure a solid theoretical foundation, selected earlier textbooks and publications were consulted for key physiological concepts and essential definitions in basic medical science. The inclusion criteria encompassed original research studies, systematic and narrative reviews, meta-analyses and clinical guidelines; non-peer-reviewed articles and publications in languages other than English were mainly excluded.

Review and discussion

Pathophysiology of Chronic Stress

The physiological effects of stress are primarily mediated by the HPA axis and ANS. While acute stress triggers adaptive mechanisms that enhance alertness and physical performance, chronic stress leads to maladaptive changes that contribute to disease development [1, 2, 3]. In response to an acute stressor, the sympathetic nervous system (SNS) is rapidly activated, leading to the "fight-or-flight" reaction [17]. The hypothalamus signals the adrenal medulla to release catecholamines (adrenaline and noradrenaline), which increase HR, blood pressure, and glucose availability to prepare the body for immediate action [18]. Simultaneously, the HPA axis is activated: the hypothalamus secretes corticotropin-releasing hormone (CRH); CRH stimulates the pituitary gland to release adrenocorticotropic hormone (ACTH); ACTH prompts the adrenal cortex to release cortisol, a glucocorticoid that enhances energy mobilization, suppresses non-essential functions (such as digestion and immune activity) and modulates inflammatory responses [19]. The schematic representation of the sites of synthesis and action of hormones and mediators involved in the process of response to stress is shown in Figure 1.

Once the stressor is removed, negative feedback mechanisms inhibit the HPA axis, and cortisol levels return to baseline, restoring homeostasis. This acute stress response is adaptive and protective, as it enhances survival in dangerous or demanding situations [21].

When stress becomes chronic, the persistent activation of the HPA axis and SNS leads to dysregulation of multiple physiological systems. Prolonged exposure to elevated cortisol levels disrupts normal metabolic, immune and neurological functions. Unlike acute stress, where homeostasis is quickly restored, chronic stress leads to a state of prolonged imbalance, predisposing the body to various diseases [22].

Chronic activation of the HPA axis results in sustained cortisol secretion, which disrupts its normal negative feedback loop. Over time, this can lead to HPA axis dysfunction, characterized by either hypercortisolism (excess cortisol) or hypocortisolism (adrenal exhaustion). These imbalances contribute to metabolic disorders, immune suppression, and neurodegenerative changes. Prolonged stress alters neurotransmitter levels, particularly dopamine, serotonin, and gamma-aminobutyric acid (GABA), leading to mood disturbances, anxiety, and depression. Additionally, chronic stress promotes sympathetic dominance in the ANS, reducing parasympathetic activity responsible for rest and recovery. This imbalance contributes to increased HR and blood pressure (risk of hypertension and cardiovascular disease), impaired digestion due to suppressed parasympathetic function and sleep disturbances caused by excess arousal [23].

Constant exposure to the increased cortisol levels can downregulate antioxidant enzymes including superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx) [24], sabotaging organism's defense against oxidative stress. The chronic stress promotes inflammatory response which generates reactive oxygen species (ROS), magnifying oxidative stress. On the cellular level, chronic stress can lead to mitochondrial dysfunction [25], increasing electron leakage in the electron transport chain and generating excess ROS. This damages mitochondrial DNA and proteins, impairing metabolic pathways. Each of the pathways creates a vicious cycle, where oxidative stress further induces stress-related damage, leading to long-term effects on the healthspan. While acute stress can temporarily suppress inflammation, chronic stress exerts a pro-inflammatory effect that gradually undermines overall health. Elevated levels of cortisol, the

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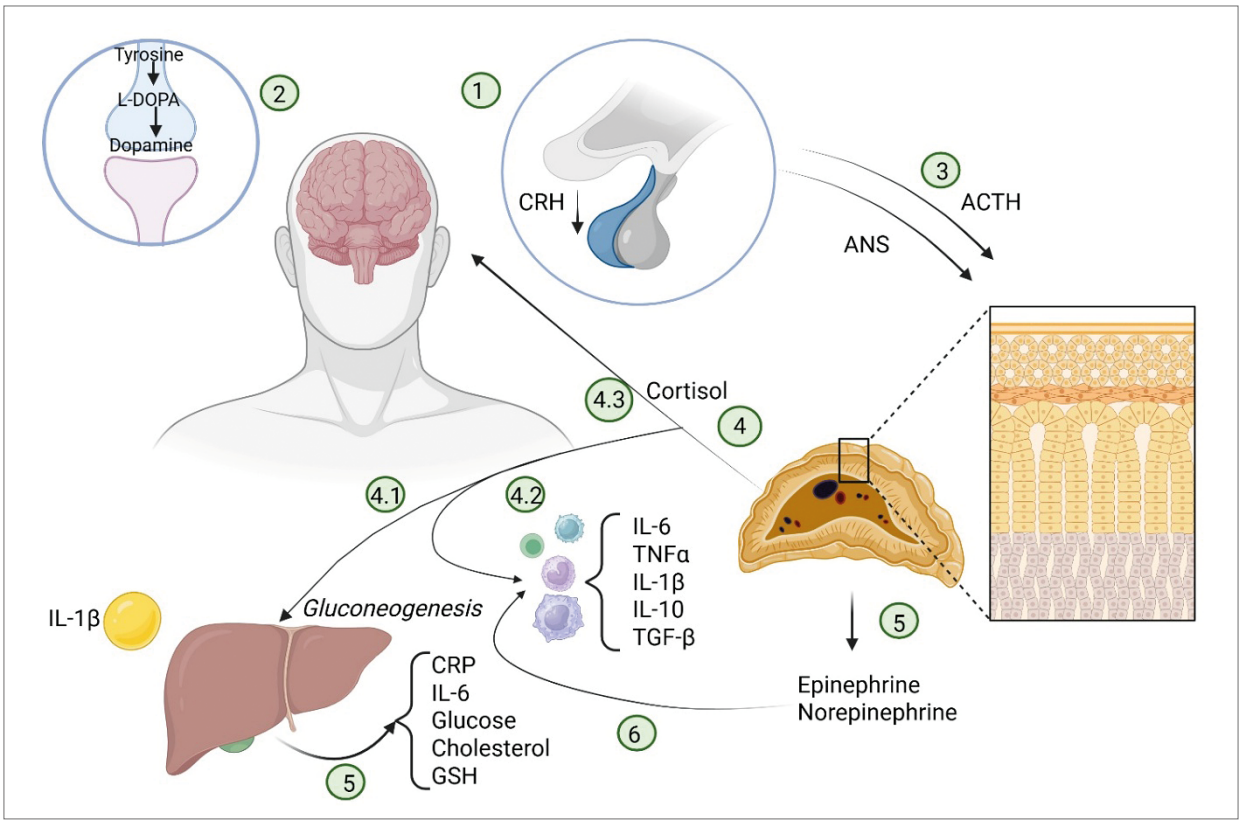

Figure 1. Interaction between the HPA axis and ANS stress response. The managed processes include: 1) Activation of the Hypothalamus and CRH Release. CRH stimulates the anterior pituitary to release ACTH (adrenocorticotropic hormone) into the bloodstream. 2) Dopaminergic pathways are activated, influencing motivation, mood, and behavioral responses under stress. 3) ACTH acts on the adrenal cortex, promoting the synthesis and release of glucocorticoids, primarily cortisol. The autonomic nervous system is activated and gives an influence on adrenal's medulla. 4) The adrenal cortex releases cortisol, the key hormone of the stress response. Cortisol: 4.1 Stimulates gluconeogenesis in the liver (increasing blood glucose levels), 4.2 Modulates the immune system (can have both pro- and anti-inflammatory effects), increases levels of CRP, IL-6, cholesterol, glucose and reduces antioxidant capacity, 4.3 Negative feedback loop: Cortisol exerts negative feedback on the hypothalamus and anterior pituitary to suppress further CRH and ACTH release. 5) The autonomic nervous system is triggering the adrenal medulla to release epinephrine and norepinephrine. 6) Stress and cortisol also affect immune function: cytokines like IL-6, TNF-α, IL-1β, IL-10 and TGF-β are released; a shift in balance between pro- and anti-inflammatory signals; immune cells influence liver function, promoting an acute-phase response.

body's primary stress hormone, disrupt immune regulation and lead to an overproduction of pro-inflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α). This state of persistent low-grade inflammation becomes a silent contributor to multiple chronic diseases. One of the most critically affected systems is the cardiovascular system. Chronic inflammation accelerates the development of atherosclerosis – the buildup of fatty plaques in arterial walls. These plaques reduce arterial elasticity and narrow the vessel lumen, which restricts blood flow and increases blood pressure. Over time, this leads to hypertension, coronary artery disease, heart attacks, and strokes. Stress-related hormonal imbalances (e.g., excessive cortisol and adrenaline) can also cause endothelial dysfunction, increased HR and elevated blood pressure – further stressing the cardiovascular system.

Given that cardiovascular diseases continue to be the leading cause of mortality worldwide, identifying and managing chronic stress is not just advisable is a public health priority. Regular monitoring of stress levels, blood pressure, and inflammatory markers, along with

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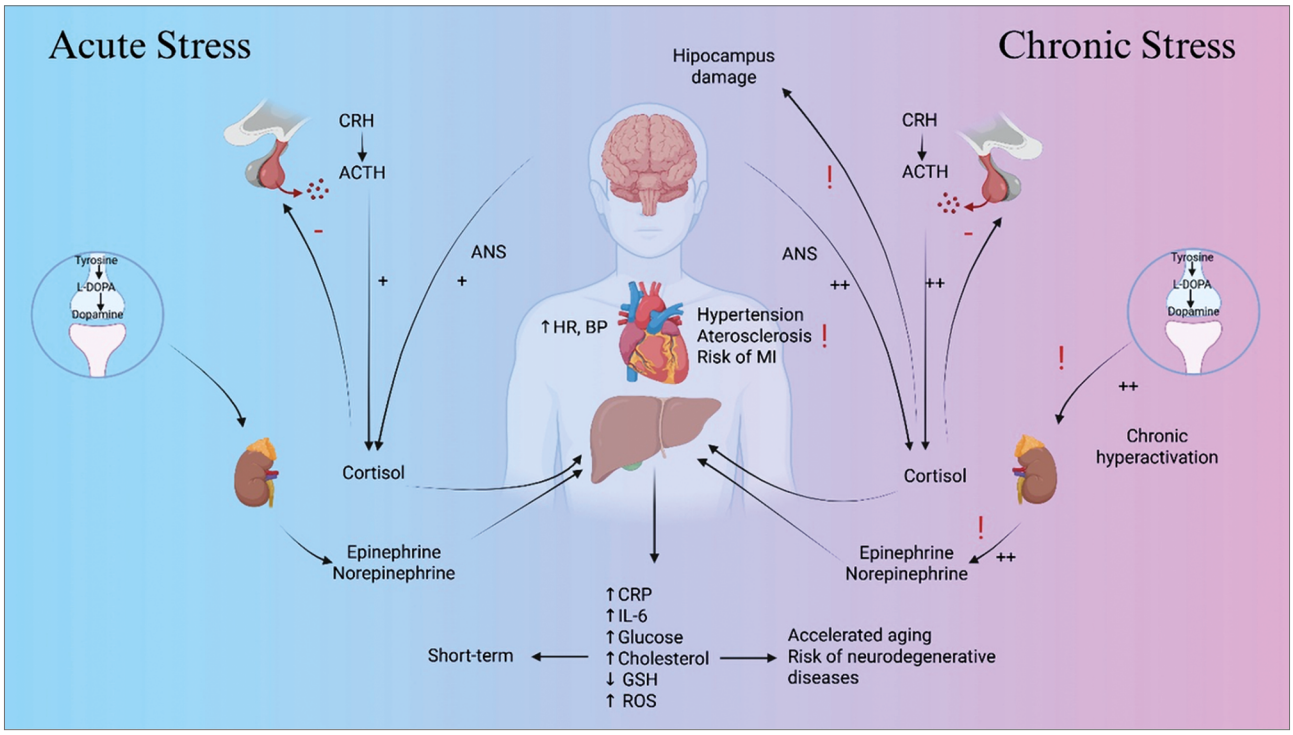

Figure 2. Comparison of the Mechanisms of Acute and Chronic Stress Development and Their Physiological Effects on the Human Body. 1) Acute stress: The hypothalamus is activated and begins to secrete CRH. CRH stimulates the anterior pituitary gland to release ACTH, which then acts on the adrenal cortex, triggering the production of cortisol. The ANS is activated, stimulating the adrenal medulla to release the catecholamines epinephrine and norepinephrine. It leads to a short-term increase in heart rate and blood pressure, as well as elevated levels of glucose, cholesterol, CRP and IL-6. The body's antioxidant capacity (reduced glutathione GSH) is also rises, although ROS are simultaneously produced. These changes help mobilize energy and resources for rapid adaptation and survival during acute stress exposure. 2) Chronic stress: the continuous activation of the hypothalamus results in persistent CRH secretion, which causes the pituitary gland to chronically release ACTH. This maintains elevated cortisol levels in the body and leads to sustained hyperactivation of the sympathetic nervous system, which continuously secretes epinephrine and norepinephrine. This chronic state leads to damage of the hippocampus, which is responsible for memory and negative feedback regulation of the HPA axis, due to the prolonged influence of cortisol. As a result, the body's ability to regulate the stress response becomes impaired. Chronic stress is associated with an increased risk of hypertension, atherosclerosis, myocardial infarction, and neurodegenerative diseases. Metabolically, it is characterized by persistently elevated levels of glucose, cholesterol, CRP, IL-6 and ROS, along with reduced GSH, reflecting oxidative stress and depletion of the body's defenses.

lifestyle interventions, such as physical activity, mindfulness practices, and psychosocial support, are crucial steps in preventing life-threatening complications.

While acute stress is a normal and adaptive response that helps the body cope with immediate challenges, chronic stress leads to widespread physiological dysregulation that contributes to numerous chronic diseases. The prolonged overactivation of the HPA axis and ANS results in systemic inflammation, neurotransmitter imbalances and long-term damage to multiple organ systems. Understanding these mechanisms highlights the importance of stress management strategies to reduce the risk of chronic stress-related health complications. The comparison of the mechanisms underlying the development of acute and chronic stress in the human body is presented in Figure 2.

Conclusions

The assessment of stress-related biomarkers provides a comprehensive understanding of stress pathophysiology. By integrating neuroendocrine, immunological, and metabolic markers, clinicians and researchers can develop targeted interventions to mitigate the adverse

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effects of chronic stress. Early detection of elevated neuroendocrine markers associated with chronic stress may provide valuable insight into the mechanisms underlying increased cardiovascular risk and support more effective management of patients with cardiovascular disease. Future studies should focus on refining biomarker panels and exploring individualized therapeutic approaches to stress management.

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This study did not receive external funding.

Conflict of interests

The authors declare no conflict of interest.

Consent to publication

All authors have read the manuscript and agreed to its publication.

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A – Work concept and design, B – Data collection and analysis, C – Responsibility for statistical analysis, D – Writing the article, E – Critical review, F – Final approval of the article

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Лабораторні маркери хронічного та гострого стресу: діагностична цінність та клінічні наслідки (Частина 1: Патофізіологія гострого та хронічного стресу в контексті його впливу на серцево-судинну систему)

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Анотація: *хронічний стрес суттєво впливає на здоров'я людини, спричиняючи дизрегуляцію гіпоталамо-гіпофізарно-надниркової (ГГН) осі та автономної нервової системи (АНС), що призводить до нейроендокринних, імунологічних і метаболічних порушень. Хронічна гіперактивність цієї системи збільшує серцевий викид, викликає вазоконстрикцію та підвищує артеріальний тиск, поступово сприяючи ремоделюванню судин, гіпертрофії міокарда та зростанню ризику серцево-судинних подій. Метою роботи було дослідити діагностичне та патофізіологічне значення нейроендокринних біомаркерів в оцінці стресу та його впливу на серцево-судинну систему. Було проведено огляд літератури, що охоплює 76 джерел англійською мовою, за допомогою баз даних PubMed, Scopus, Web of Science та Google Scholar, зосереджуючись на взаємозв'язку між гіпоталамо-гіпофізарно-надниркової*

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віссю, автономною нервовою системою та хронічним стресом; пошук включав рецензовані публікації за період з 2020 по 2025 роки за ключовими словами: «гострий стрес», «хронічний стрес», «дисрегуляція ГГН-осі», «автономна нервова система», «кортизол», «епінефрин», «норепінефрин», «дегідроепіандростерон», «дофамін», «альдостерон», «фактор некрозу пухлин-альфа», «інтерлейкін-1», «інтерлейкін-6», «С-реактивний білок», «інсуліноподібний фактор росту-1», «холестерин», «альбумін», «глікозильований гемоглобін» та «серцево-судинна система». Критерії включення охоплювали оригінальні наукові дослідження, систематичні та наративні огляди, метааналізи та клінічні настанови; нерецензовані джерела та публікації не англійською мовою загалом виключалися. Нейроендокринні біомаркери надають важливу інформацію щодо фізіологічного навантаження, спричиненого стресом, та функціонування ГГН-осі й АНС. Кортизол залишається найбільш усталеним біомаркером як гострого, так і хронічного стресу, при цьому кортизол волосся має унікальні переваги для довготривалої оцінки. Катехоламіни відображають гостру симпатичну активацію, однак їх діагностичне значення при хронічному стресі є обмеженим. Використання нейроендокринних маркерів посилює клінічне прийняття рішень та може сприяти персоналізованим стратегіям профілактики та менеджменту захворювань, пов'язаних зі стресом. Раннє виявлення підвищення нейроендокринних маркерів, асоційованих із хронічним стресом, може надати цінну інформацію щодо механізмів підвищеного серцево-судинного ризику та сприяти ефективнішому веденню пацієнтів із серцево-судинними захворюваннями.

Ключові слова. Стрес, фізіологія, нейроендокринологія, гормони, серцево-судинна система