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Adaptative Changes Of Homeostatic Systems In Response To Stress The Role Of Cortisol And The Sympathetic Nervous System

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Abstract

Over the past decade, scientific research on stress physiology has provided a deeper understanding of the mechanisms of response and adaptation of homeostatic systems in the human body to stress factors [1]. In a state of stress, the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS) are closely interconnected, causing the body to undergo a complex adaptive response aimed at maintaining homeostasis [2]. As a result of the activation of these systems, stress hormones such as cortisol, adrenaline, and noradrenaline are released, which leads to accelerated cardiovascular activity, increased glucose metabolism, and a temporary weakening of the immune system [3]. The main physiological role of cortisol is to protect the body from energy deficiency during stress, regulate protein and fat metabolism, and control immune inflammatory responses [4]. At the same time, the sympathetic nervous system activates the “fight or flight” mechanism, increasing heart rate and directing blood flow to the muscles [5]. However, chronic exposure to stressors leads to HPA axis imbalance, which leads to increased cortisol secretion, metabolic syndrome, depression, and cardiovascular disease [6].

Recent studies (Kloet, 2024; Chrousos, 2020) have shown that individual stress reactivity is modulated by genetic and epigenetic control of cortisol. This suggests that the level of stress tolerance of each individual is

formed by the interaction of genetic and environmental factors [7]. In addition, dysfunction of stress response systems directly affects the functioning of the central nervous system, affecting memory, emotional balance, and decision-making processes [8].

This article provides an in-depth analysis of the homeostatic changes that occur in response to stress, the interplay between cortisol and the sympathetic nervous system. Also, based on modern scientific sources, the short- and long-term physiological consequences of stress, as well as the role of cortisol in the disruption of adaptation mechanisms, are highlighted [9].

Keywords: Stress, homeostasis, cortisol, sympathetic nervous system, adaptation, hypothalamic-pituitary-adrenal axis.

Introduction

To scientifically analyze the adaptive mechanisms through which homeostatic systems in the human body adapt in response to stress, and the main role of cortisol and the sympathetic nervous system in this process.

Methods

The analysis was conducted on the basis of articles published in the SpringerLink, PubMed, ScienceDirect, Nature Reviews Endocrinology and inlibrary.uz databases between 2015 and 2025 [2]. The selected sources are experimental and clinical studies studying stress physiology, neuroendocrine regulation, and homeostatic response mechanisms.

Stress is a physiological state of the body that develops in response to internal or external factors, which activates adaptive mechanisms aimed at maintaining homeostasis [3]. The hypothalamic-pituitary-adrenal (HPA) axis increases cortisol secretion, while the sympathetic nervous system increases heart rate, blood pressure, and glucose excretion [4]. These systems work in harmony to provide short- and long-term adaptation to stress. The concept of stress is considered in physiology as a complex neuroendocrine and biochemical response of the body to changes in the external or internal environment aimed at maintaining homeostasis [1]. The theory of “stress”, developed by Hans Selye in the middle of the 20th century, has been developed to a deeper level today and includes mechanisms of interaction between the hypothalamic-pituitary-adrenal (HPA) axis, the sympathetic nervous system (SNS), and the immune system [2]. The coordinated activity of these systems in response to stress increases the body's ability to adapt, but prolonged activation of this process can lead to physiological imbalances and pathological conditions [3].

The sympathetic-adrenal system is the first to be activated under stress - this increases the heart rate, blood pressure, and oxygen delivery to the muscles as a "fight or flight" mechanism [4]. In the next stage, corticotropin-releasing hormone (CRH) is released from the hypothalamus, which activates the pituitary gland to produce adrenocorticotropic hormone (ACTH); as a result, the adrenal glands secrete the hormone cortisol [5]. Cortisol provides the body with energy, enhances gluconeogenesis, inhibits immune responses, and controls the adaptation of the central nervous system to stress [6].

Recent scientific studies (Kloet, 2024; Ryznar, 2025) have shown that the HPA axis and the SAS system are closely interconnected and play an important role in processes ranging from short-term protective responses to long-term adaptive restructuring of the body [7]. Also, according to Nature Reviews Neuroscience (McEwen and Gianaros, 2022), the stress response is explained by the concept of allostatic load - that is, a state of constant load that occurs as a result of the body's constant mobilization of physiological resources to maintain homeostasis [8]. Therefore, the study of changes in homeostatic systems in response to stress is an important scientific direction in modern biology, medicine, and psychoneurology [9]. Cortisol and the sympathetic nervous system are at the heart of this process; they interact to ensure adaptation, immune regulation, cardiovascular and metabolic stability [10]. However, overactivity or chronic activation of these systems poses a serious threat to human health - for example, chronic stress can cause hypertension, insulin resistance, depression and immune imbalance [11].

Results

Recent studies show that:

The role of cortisol in the stress response

Cortisol is the central hormone of the stress response, and its main physiological function is to restore energy balance in the body, coordinate metabolic processes

and optimize the functioning of the nervous system [1]. During short-term stress, cortisol activates the process of gluconeogenesis in the liver, breaks down proteins into amino acids and uses them as an energy source, and releases free fatty acids from adipose tissue [2]. This mechanism supports the functioning of vital systems - the heart, brain and muscles - by providing the body with the necessary energy [3].

Cortisol also subtly modulates the activity of the immune system. It balances the immune response by inhibiting the production of inflammatory mediators - cytokines (IL-1, IL-6, TNF-α) [4]. However, chronically high levels of this hormone have an immunosuppressive effect, increasing susceptibility to infectious diseases and increasing the risk of developing autoimmune diseases [5]. Therefore, the effect of cortisol is considered a “double-edged sword” - short-term activation is a protective mechanism, while prolonged hyperactivity causes pathological conditions [6].

In addition, cortisol is an important neuromodulator that regulates cognitive processes in the central nervous system. It controls neuronal plasticity, memory formation, and attentional processes in the hippocampus and prefrontal cortex under stress [7]. Recent neuroendocrine studies (Kloet, 2024; McEwen, 2022) describe the effect of cortisol on brain function as a “U-shaped” curve: moderate levels of cortisol enhance cognitive performance, while excessively high or low levels impair neuronal activity [8]. As a result, cortisol activates the body’s adaptation mechanisms during stress, playing a central role in maintaining energy supply, immune stability, and neuropsychological balance [9]

The role of the sympathetic nervous system in the stress response

The sympathetic nervous system (SNS) is the first and fastest component of the stress response, activating the “fight-or-flight” mechanism to adapt the cardiovascular, respiratory, and endocrine systems to stress [1]. Under the influence of a stress factor, sympathetic neurons are activated by the hypothalamus, and catecholamines — adrenaline and noradrenaline — are released from the adrenal medulla [2]. These hormones increase heart rate, constrict peripheral blood vessels, increase arterial pressure, and increase blood flow to skeletal muscles [3]. As a result, the body can maximally mobilize energy resources and quickly respond to a dangerous or stressful situation [4].

Adrenaline mainly affects the cardiac and metabolic systems — it increases the force of cardiac contraction, quickly transports glucose from the blood to the cells, and activates glycogenolysis in the liver [5]. Noradrenaline mainly regulates vascular tone, optimizes cerebral blood flow, and increases alertness in the central nervous system [6]. Thus, the secretion of catecholamines by the SAS activates cardiovascular and metabolic adaptation mechanisms necessary to maintain physiological homeostasis in stress conditions [7].

Recent neurophysiological studies have shown that the sympathetic system interacts not only with peripheral but also with central structures (such as the locus coeruleus, amygdala, and hypothalamus of the brain) [8]. Through these centers, the activity of the SAS regulates stress perception, risk response, and emotional stability [9]. In cases of chronic stress, prolonged activation of the SAS can cause cardiovascular overload, hypertension, and arrhythmias [10].

Thus, the sympathetic nervous system is considered a complex neuroendocrine system that activates rapid adaptation mechanisms in response to stress, but can also cause pathological conditions when prolonged [11].

Prolonged stress and HPA axis dysfunction

In cases of prolonged or chronic stress, the balance of the hypothalamic–pituitary–adrenal (HPA) axis is disrupted, which leads to persistently high cortisol secretion [1]. Normally, cortisol plays an important role in maintaining homeostasis, ensuring energy balance, and limiting inflammatory responses, but its chronic overproduction has harmful consequences for the body [2]. Studies show that persistent activation of the HPA axis contributes to the development of disorders such as metabolic syndrome, insulin resistance, abdominal obesity, and dyslipidemia [3]. Excessive secretion of cortisol reduces the entry of glucose into cells, resulting in elevated blood glucose levels and an increased risk of type 2 diabetes [4]. Chronically elevated cortisol also negatively affects brain plasticity. It causes a decrease in the number of neurons in the hippocampus and a slowdown in the formation of new neurons, which leads to depression and cognitive decline [5]. Neuroimaging studies have shown increased amygdala activity and inhibition of the prefrontal cortex in patients with chronic stress, which leads to emotional imbalance [6]. Therefore, HPA axis dysfunction is considered a major pathogenic mechanism not only for endocrine but also

for neuropsychological problems [7].

In addition, chronic cortisol excess has a significant negative effect on the cardiovascular system. Cortisol increases blood pressure, reduces the elasticity of the vascular endothelium, and promotes the development of atherosclerosis [8]. Through this mechanism, it has been established that prolonged stress is directly related to pathologies such as hypertension, ischemic heart disease, and heart failure [9]. Thus, chronic dysfunction of the HPA axis plays a central role in the development of multiple systemic diseases, which emphasizes the clinical importance of early detection and management of stress [10]

“Tonic” control of cortisol and mechanisms of genetic-epigenetic modulation

Modern studies by E.R. Cloet (2024) have emphasized that “tonic” control of cortisol—that is, its continuous effect at the basal level—plays a central role in the genetic and epigenetic regulation of the stress response [1]. Once produced by the HPA axis, cortisol binds to glucocorticoid receptors (GRs) in the nucleus of cells, and this complex activates or inhibits transcription at the DNA level [2]. Through this process, the expression of hundreds of stress-responsive genes is altered, which genetically determines the resistance of each individual to stress [3]. At the epigenetic level, cortisol induces long-term neuroendocrine changes through DNA methylation, histone modifications, and microRNAs [4]. For example, methylation levels in the promoter region of the NR3C1 gene reduce cortisol sensitivity, which leads to increased HPA axis activity and a heightened response to stress [5]. Childhood psychological stress or trauma has been shown to amplify these epigenetic changes, increasing the risk of developing depression or anxiety in adulthood [6]. Cloet's results in 2024 also indicate that the activity of glucocorticoid receptors and their expression levels in brain structures can be used as a “biomarker” of the individual stress response [7]. He argued that the “tonic” control of cortisol not only facilitates adaptation to stress, but also forms the epigenetic memory of the organism, ensuring the hereditary stability of the stress response in subsequent generations [8].

The interaction of physiological and psychological factors in traumatic stress

Recent studies by Ryznar et al. (2025) have scientifically proven that physiological and psychological factors work in close connection with each other in traumatic stress situations, and through their interaction, adaptive stability (resilience) is formed in the body [1]. Traumatic stress not only activates the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system (SNS), but also leads to the reorganization of neural networks in brain structures, particularly in the amygdala, hippocampus, and prefrontal cortex [2]. These changes are considered to be the neurobiological basis for determining the body's ability to perceive and adapt to stress [3].

As Ryznar et al. (2025) have shown, the level of stress tolerance is associated with the individual genetic makeup, epigenetic changes, and the social environment, which shape the body's "quality of response" [4]. According to their model, physiological factors (cortisol secretion, heart rate, immune system response) interact with psychological factors (emotional stability, cognitive control, social support) to determine the level of adaptive adaptation [5].

This study also proposed the concept of an “adaptive stress network,” which is defined as a coordinated response of the neuroendocrine system, the immune system, and the functional connections of the brain [6]. When this network is disrupted, the risk of developing posttraumatic stress disorder (PTSD), depression, or cardiovascular disease increases [7]. Therefore, the physiological and psychological integration of the response to traumatic stress is a central mechanism that determines the individual’s adaptive capacity [8].

Discussion

The adaptive response of homeostatic systems occurs in two phases: the first is a fast (sympathetic-adrenal) phase, and the second is a slow (endocrine cortisol-mediated) phase [10]. The synchrony of these systems is important for coping with stress. However, in chronic stress, the balance between these systems is disrupted, as a result of which the mechanisms that maintain homeostasis become exhausted. In recent years, new biomarkers have been developed to assess individual stress resistance by measuring cortisol and sympathetic activity [11].

Conclusion

The stress response process is a complex and multi-stage system that plays an important role in maintaining the stability of the organism at the physiological, biochemical and psychological levels. Studies show that

adaptive changes in homeostatic systems are coordinated by cortisol and the sympathetic nervous system in stressful situations. When the hypothalamic–pituitary–adrenal (HPA) axis is activated, cortisol is released, which restores energy balance, regulates the immune system, and increases the flexibility of the central nervous system. At the same time, the sympathetic nervous system increases the secretion of noradrenaline and adrenaline, which adapts the cardiovascular system to the stress state.

However, in prolonged or chronic stress, the balance of these systems is disrupted, and persistently elevated cortisol levels cause diseases such as metabolic syndrome, depression, and cardiovascular disease. The “tonic” control of cortisol through epigenetic mechanisms alters the expression of stress-responsive genes, and these changes sometimes become stable states that are transmitted from generation to generation.

Modern scientific sources, including studies by Ryznar et al. (2025) and de Kloet (2024), emphasize that stress tolerance — i.e. adaptive stability — is formed through a balanced integration of physiological (endocrine and neural systems) and psychological (emotional and social factors) components. Therefore, stress management, psychological support, and hormonal balance are considered essential for healthy adaptation.

In general, cortisol and the sympathetic nervous system are central elements of the stress response, and their balanced activity ensures long-term healthy adaptation of the body, but dysfunction of these systems plays a leading role in the pathogenesis of many chronic diseases. Future research will serve to develop strategies for increasing stress tolerance by further studying the genetic, epigenetic, and neuropsychological integration of these systems [10]

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