DOI: https://doi.org/10.17816/KMJ623568

Neurochemical markers of coping intelligence

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ABSTRACT

Coping intelligence is associated with an individual's ability to overcome stressful situations, maintaining health potential and increasing the potential for personal development. This study is a systematic review of biochemical and neuronal markers of different levels of coping intelligence, which determine different lines of human development in stressful situations. 45 publications selected from the Nature and RSCI electronic databases were analyzed, the results were summarized in three sections: (1) genetic and epigenetic correlates of individual differences in coping intelligence; (2) neurochemical systems of coping intelligence (glucocorticoids, interleukins, brain-derived neurotrophic factor, monoamines); (3) manifestations of stable and regressive lines of development of the subject in stressful situations. Molecular genetic determinants of coping intelligence were systematized according to the following systems: serotonergic, dopaminergic, noradrenergic, etc. The interaction of neurochemical systems (catecholamines, glucocorticoids, interleukins, brain-derived neurotrophic factor, monoamines) reflects the peculiarities of the stress reaction in humans and determines the development line of the subject in stressful situations. Genetic predisposition, unfavorable epigenetic factors and chronic stress increase the risk of developing stress-related diseases (regressive line of development). A stable stress-coping system is associated with a balance of mineralocorticoid and glucocorticoid receptors, pro-inflammatory and anti-inflammatory cytokines, an optimal ratio of cortisol and dehydroepiandrosterone sulfate, a sufficient level of brain-derived neurotrophic factor, and a healthy microbiota (stable line). A review of the literature indicated the need to analyze neurochemical systems (monoamines, opioid receptors, acetylcholine, microbiota) that determine a high level of coping intelligence (a progressive line of human development in stressful situations). The study of neurochemical markers of coping intelligence should be accompanied by personality analysis (mental representations of stress, coping strategies) to provide personalized medical care and preserve a person's health potential.

Keywords: patient's personality; stress-related diseases; stress resistance; personalized medicine.

To cite this article:
Kuvaeva IO, Volkova EV. Neurochemical markers of coping intelligence. *Kazan Medical Journal.* 2024;105(2):260–271. DOI: https://doi.org/10.17816/KMJ623568

Received: 07.12.2023 **Accepted:** 25.01.2024 **Published:** 01.04.2024

DOI: https://doi.org/10.17816/KMJ623568
УДК 616-052

Нейрохимические маркёры совладающего интеллекта

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АННОТАЦИЯ

Совладающий интеллект связан со способностью личности преодолевать стрессовые ситуации, сохраняя потенциал здоровья и преумножая потенциал развития личности. Данное исследование представляет собой систематический обзор биохимических и нейрональных маркёров разных уровней совладающего интеллекта, обусловливающих разные линии развития человека в стрессовых ситуациях. Проанализировано 45 публикаций, отобранных из электронных баз Nature и РИНЦ, результаты обобщены в три раздела: (1) генетические и эпигенетические корреляты индивидуальных различий совладающего интеллекта; (2) нейрохимические системы совладающего интеллекта (глюкокортикоиды, интерлейкины, нейротрофический фактор головного мозга, моноамины); (3) проявления стабильной и регрессивной линий развития субъекта в стрессовых ситуациях. Молекулярно-генетические детерминанты совладающего интеллекта систематизированы по системам: серотонинергическая, дофаминергическая, норадренергическая и др. Взаимодействие нейрохимических систем (катехоламины, глюкокортикоиды, интерлейкины, нейротрофический фактор головного мозга, моноамины) отражает особенности протекания стрессовой реакции у человека и определяет линию развития субъекта в стрессовых ситуациях. Генетическая предрасположенность, неблагоприятные эпигенетические факторы и хронический стресс повышают риск развития стресс-сопряжённых заболеваний (регрессивная линия развития). Устойчивая стресс-совладающая система сопряжена с балансом минералокортикоидных и глюкокортикоидных рецепторов, провоспалительных и противовоспалительных цитокинов, оптимальным соотношением кортизола и дегидроэпиандростерона сульфата, достаточным уровнем нейротрофического фактора головного мозга, здоровой микробиотой (стабильная линия). Обзор литературы указал на необходимость анализа нейрохимических систем (моноамины, опиоидные рецепторы, ацетилхолин, микробиота), обусловливающих высокий уровень совладающего интеллекта (прогрессивная линия развития человека в стрессовых ситуациях). Изучение нейрохимических маркёров совладающего интеллекта должно сопровождаться анализом личности (ментальные репрезентации стресса, стратегии совладания) для оказания персонализированной медицинской помощи и сохранения потенциала здоровья человека.

Ключевые слова: личность больного; стресс-сопряжённые заболевания; стрессоустойчивость; персонализированная медицина.

Как цитировать:
Куваева И.О., Волкова Е.В. Нейрохимические маркёры совладающего интеллекта. *Казанский медицинский журнал.* 2024;105(2):260–271. DOI: https://doi.org/10.17816/KMJ623568

Рукопись получена: 07.12.2023 **Рукопись одобрена:** 25.01.2024 **Опубликована:** 01.04.2024

Stressful situations commonly occur in our lives. According to R. Sapolsky, a prominent neuroendocrinologist and stress specialist, humans frequently activate a physiological system that is intended to respond to acute physical emergencies [1]. Prolonged or excessive stress can overload an organism's regulatory capabilities and adaptive resources, and the resulting general adaptation syndrome becomes a pathogenic factor [2] and contributes to the development of stress-related diseases.

Stress is defined as a state of threat to the homeostatic equilibrium caused by various internal or external stressors, whether real or perceived [3]. To maintain optimal homeostasis within physiological limits, the organism has developed a complex stress-adaptive system that enables self-regulation, adaptation, and energy redistribution in response to situational demands and needs [4].

During stress, cognitive programs shift from complex associative operations to quickly develop effective strategies. Monoamines, cytokines, glutamate, γ-aminobutyric acid, and other central mediators play key roles in providing normal stress responses [4]. The degree and duration of the activation of stress-realizing and -limiting systems vary depending on the quality, strength, and duration of exposure to the stressor [5].

Physiological and biochemical reactions to challenging life situations are common. However, psychological reactions vary after the activation of defense mechanisms during threat-level assessment [6].

According to A.V. Waldman, the organism's subsequent complex reaction is qualified as emotional and stressful when a signal is evaluated as negative and rejected (aversive), and it is impossible to get rid of it or when unprepared stereotyped mechanisms of "shielding avoidance" are activated [2]. Attitude, i.e., perception and evaluation of this effect as harmful, is determined not so much by innate, biologically fixed mechanisms but by the whole complex of stable properties of an individual, formed during the development and activity of this person in a certain social environment [2].

In psychology, the term "ability" refers to a set of stable human properties. Coping intelligence (CI) is a type of ability that explains individual differences in psychological and physiological reactions to a threat. However, E. Libina interpreted CI solely in terms of effective or ineffective strategies and psychological defenses. According to A.V. Waldman, the ability to effectively manage stressful situations without compromising one's health should be studied comprehensively, and CI should be viewed as a biopsychosocial phenomenon.

This study aimed to identify neurochemical markers that indicate CI levels. The latest genetic, biochemistry, immunology, and medicine data will be used to examine the CI phenomenon in a new way. Collected data will help identify biochemical correlates of individual differences in CI and an organism's development in stressful situations.

The correlation between an organism's developmental trajectory in stressful situations and its vulnerability to difficulties and high risk of stress-related diseases is regressive. Conversely, stability in stressful situations is associated with a low risk of stress-related diseases, whereas a progressive trajectory is linked to the preservation of health potential and increase of personal development potential in unstable living conditions [7].

The study of human developmental lineages during stress requires an examination of the molecular genetic basis of CI, role of epigenetic factors, biochemistry of acute stress, neurochemical markers of stress tolerance, and stress-related diseases. This passage explores the cause-and-effect relationship of the formation of different CI levels (low, medium, and high) and their effect on human development (regressive, stable, and progressive) [7]. The data presented suggest that health and productivity are markers of CI.

This study included review articles and empirical studies published from 2006 to 2023. The publication search devoted to the presentations of the biological level of CI was conducted in the database of the electronic journal *Nature* (nature.com) and the Russian information and analytical portal in the field of science RSCI[1] (elibrary.ru).

Criteria for the inclusion of articles:

1. Biochemical changes in the body in response to acute and chronic stressors
2. Biochemistry of stress-related diseases
3. Biomarkers of stress resistance
4. Biomaterials such as blood, saliva, hair, and microbiota

This review presented various neurochemical markers of CI, including cortisol, interleukins (ILs), brain-derived neurotrophic factor (BDNF), catechol-O-methyltransferases (COMTs), and monoamines. The study analyzed and summarized 23 articles in English and 20 articles in Russian. The findings are presented in three sections: genetic and epigenetic predictors of CI levels, neurochemical markers of acute and chronic stresses, and neurochemistry of stress resistance and stress-related diseases.

The publication search included criteria and keywords.

– Outcome: Biological CI levels (keywords included stress, coping, and cortisol; stress, coping, and BDNF; stress, coping, and IL-6; stress, coping, and IL-4; stress, coping, and IL-10; stress, coping, and COMT; stress, coping and CDH1; stress, coping and HTR3; stress, coping and TNF-α; stress, coping and TLR9; stress, coping and ADRA2A; stress, coping and OPRM1).

– Participants: humans, rodents, and fish. Since experiments on humans leading to health deterioration are inadmissible, laboratory and experimental data on the biochemistry of acute and chronic stresses in animals were analyzed.

[1] RSCI is the Russian Science Citation Index.

– Subjects: health or resilience, disease or irritable bowel syndrome, cardiovascular disease or oncology, posttraumatic stress disorder (PTSD), anxiety disorder, depression, microbiota, acute stress, and chronic stress.

The search strategy for both review and empirical studies differed significantly between foreign and Russian databases. The PRISMA strategy, traditionally used in review preparation, was used on the electronic resource *Nature* (https://www.nature.com/). The selected articles mainly contained data on the biochemistry of stress-related diseases (10 articles) and stress resistance (5 articles).

The use of the PRISMA strategy is not possible in the RSCI publication database (elibrary.ru) because of differences in capabilities [12]. Finding relevant publications is challenging because highly rated journals in genetics, biochemistry, psychophysiology, and medicine cannot be filtered. A keyword search in the RSCI system produced a list of 3106 publications, each of which was evaluated for relevance based on titles and abstracts. Only 16 reviews in the Russian language focused on the biochemistry of stress and coping, which is a limited number.

Moreover, 41 empirical studies were analyzed to confirm the existence of CI biomarkers and expand upon the data presented in the review articles, providing further clarification and disclosure of the review papers. Data on cortisol (1211 publications) and IL (1537 publications) studies are more frequently presented in the Russian language than in studies of other stress response markers.

GENETIC AND EPIGENETIC CORRELATES OF INDIVIDUAL DIFFERENCES IN CI

Genetics is the study of the regularities of heredity and variability, which refers to the ability of living organisms to preserve and transmit the features of their structure and development to the next generation and ensure the diversity of traits within a species. Stress can affect gene expression by altering deoxyribonucleic acid methylation, which contributes to the accumulation of "epigenetic memory" regarding stressful events [13]. Gene variants involved in regulating the hypothalamic–pituitary–adrenal axis (HPAA) can directly or indirectly affect the activity of this system [14], which determines human CI. Because of natural selection, a range of genetically determined sensitivities to environmental signals and corresponding responses are preserved [15].

Studies [6, 14, 16–21] have indicated that many of the described genes/polymorphisms are associated with human predisposition to stress-related diseases in the regressive lineage. However, no studies have examined stable and progressive lines of human development in stressful situations.

Based on the work of [6], molecular genetic determinants of CI can be described by systems.

– The serotoninergic system modulates the HPAA during acute and chronic stress.

– Polymorphisms in dopaminergic system genes are associated with anxiety–depressive disorders.

– Adrenergic receptor genes are associated with motivation, anxiety, depression, and suicidal tendencies and development of irritability and hostility.

– Hyperfunction of the noradrenergic system causes arousal and increased search activity.

– The arginine–vasopressin system is involved in HPAA activation, learning processes, and regulation of social behavior.

– Oxytocin system gene polymorphisms can act as a protective factor in stressful situations, promoting positive social interaction.

Data on the genetic prerequisites for the formation of different personality types depending on the variations in the *COMT* gene, which correlates with high stress resistance [22], are noteworthy. There are two personality types: the "anxious person" type that is characterized by increased sensitivity to pain and an advantage in memory and attention tasks and the "fighter" type that is characterized by the ability to tolerate severe pain and cope with anxiety. A study [22] revealed that increased dopamine release can enhance performance in individuals classified as fighters, whereas those classified as anxious may experience a decrease in performance.

Early-life difficulties can lead to neurochemical changes in the body. For example, maternal separation experienced by young primates and rats at an early age can contribute to an increase in the levels of proinflammatory cytokines IL-1β and IL-6 in adulthood [23]. Conversely, good maternal care in the absence of a stressor can ensure low basal corticosterone levels in rodent cubs, whereas threatening situations can lead to high corticosterone levels. Rats raised under conditions of poor maternal care exhibit a deficiency of glucocorticoid receptors in the hippocampus, stably high corticosterone levels, and impaired shutdown of the stress response. Chronic stress in these rats is associated with inflammation and an initial decrease in BDNF [24]. Early-life stress increased susceptibility in adult female mice [25].

Children from families with low socioeconomic status exhibit higher levels of cortisol and inflammation. High cortisol levels in children aged 6–8 years are associated with higher body mass index in girls and somatic complaints in boys [26]. In addition, children who grow up in orphanages have a larger amygdala, which may serve as a biomarker for the complexity of emotional regulation when coping with stressful situations [27].

The analysis of reviews and empirical publications has clarified some genetic and epigenetic correlates of the individual differences in CI. Basic processes in human development are genetically programmed; however, gene expression in stressful situations is influenced by both socio-environmental factors and human experience [7].

DOI: https://doi.org/10.17816/KMJ623568

Table 1. The cortisol as a "switch" between vulnerability and resilience
Таблица 1. Кортизол как «переключатель» между уязвимостью и жизнестойкостью

Criteria	Mineralocorticoid receptors	Glucocorticoid receptors
Purpose	Low-cost coping	Containment of primary stress reactions, and cognitive control over emotional reactivity
Affinity (cortisol binding strength)	High	Low
Cortisol loading	Resting state	Circadian rhythm and post-stress condition
Long-term memory	Retrieval of coping strategies for similar stressful events	Retaining new experiences to cope with future stressful events
Coping	Search, proactive risk assessment, and familiar patterns	Cognitive control and contextualization
Mode of resource use	Energy-saving	Expensive

CONJUGATION OF NEUROCHEMICAL MARKERS AND MANIFESTATIONS OF CI

Glucocorticoids: Glucocorticoids are believed to have protective biological effects by mobilizing the organism to respond adequately to stressors. During acute stress, cortisol levels increase in humans [28], animals [18, 29], and juvenile dorado [30]. Cortisol plays a vital role in the stress response and acts on the feedback principle to protect the stress-responsive system from overload. Compared with animals with passive behavior, animals with active responses to stressors exhibit a lower glucocorticoid response [18].

Prolonged exposure to a stressor can both increase and decrease HPAA activity, which can lead to ambiguous changes in cortisol levels [28]. Chronically low or high levels of glucocorticoids suggest suboptimal adaptation, while moderate or controlled increases indicate good physical and mental health [31].

To exert its protective effect, cortisol activates mineralocorticoid and glucocorticoid receptors. Increased circulating cortisol concentration after a stressful event enables the "shutdown" of the stress response mediated by glucocorticoid receptors and the restoration of homeostasis [32].

Table 1 summarizes the results of previous publications [14, 32], suggesting the important role of cortisol in maintaining circadian rhythms, coping with stress, and promoting health. Impaired switching of mineralocorticoid and glucocorticoid receptors can alter HPAA activity, leading to chronic stress and increased vulnerability to stress-related diseases.

The buffered form (dehydroepiandrosterone sulfate [DHEA-S]) of the endogenous steroid dehydroepiandrosterone has pronounced antiglucocorticoid activity [28]. A decrease in the *cortisol/DHEA-S* index leads to an increase in the severity of depressive illness [28]. A higher *cortisol/DHEA-S* index and decreased circulating DHEA levels are characteristic of people experiencing emotional stress at work [33].

Interleukins: The body's immune system responds to exposures to both acute and chronic stresses. The development of acute stress through neurocytokine mechanisms is associated with the implementation of syntoxic and catatoxic adaptation programs. The syntoxic program of adaptation, which is activated by acetylcholine and is associated with cytokines IL-2 and IL-12, focuses on reproduction and is characterized by high levels of DHEA and fertile factors. To promote the survival of the organism, the catatoxic program is activated by extreme and psychosocial stimuli. This is achieved by triggering the stress response, which involves the release of catecholamines, IL-1, IL-4, IL-6, IL-10, adrenocorticotropic hormone, and cortisol [34]. The relationship among stress, inflammation, and coping strategies was discussed in detail in a review [35] that covered both the general mechanisms and specific manifestations of this relationship in patients with rheumatologic disorders.

Circulating immune cells support hippocampal neurogenesis, spatial memory, BDNF expression, and stress tolerance [36]. The immune system retains the memory of the stressor and offers protection against similar stressors in the future [37].

The *brain–gut–microbiota* axis regulates the immune system, mood, and behavior [38, 39]. A comparison between the microbiota of patients with PTSD and healthy subjects exposed to a single stressor showed a decrease in the abundance of the *Actinobacteria, Lentishaerae*, and *Verrucomicrobia* genera in patients with PTSD [38]. The activation of HPAA is associated with a local increase in the amount of proinflammatory cytokines IL-1β, IL-18, and tumor necrosis factor (TNF)-α in the microbiota [39].

BDNF affects choline, serotonin, and dopaminergic neurons and is involved in neuronal growth, synapse formation, and modification. At the organismal level, BDNF regulates body weight and energy homeostasis [40]. According to a previous study, serum BDNF levels of <300 pg/mg after a brain injury increased the risk of depression in the long term, whereas levels of >600 pg/mL indicated a sufficient potential for cognitive recovery [41].

According to Linz et al. [42], serum BDNF levels increase during the trier social stress test (TSST) and return

to baseline levels after the test. The results of the analysis of the interaction between cortisol and BDNF expression during the TSST suggest an antagonistic relationship between the two indicators. Higher BDNF levels after the test were found to be associated with a faster recovery of cortisol levels, whereas the magnitude of the cortisol response to the TSST correlated with a faster recovery of BDNF after the test [42].

Glucocorticoids affect the neurotrophin system [40, 43, 44]. The duration of the stressor determines the change in BDNF expression (43). Stress negatively affects BDNF levels: high cortisol levels reduce BDNF expression, and chronic glucocorticoid concentration impairs neurogenesis, suppresses cell proliferation, and weakens negative feedback between the hippocampus and the HPAA [40]. The repression of the *BDNF* gene, which occurs because of prolonged exposure to proinflammatory cytokines and glucocorticoids, can lead to brain atrophy and development of mental disorders in susceptible individuals [44].

Neuroplasticity helps promote neuronal development and enhance the adaptive capacity of the organism [45]. However, decreased neuroplasticity is associated with HPAA dysfunction and development of various neurological pathologies [46].

Monoamines: Monoaminergic systems are significant in the integrative functions of the body, determining the organism's adaptive abilities [47]. Studies have shown that acute stress in humans, animals, and fish is associated with high levels of serotonin, noradrenaline, and dopamine [18, 30].

STABLE DEVELOPMENT OF AN ORGANISM IN STRESSFUL SITUATIONS

Analysis of the literature indicates the presence of *rhythms, cycles, and synchronization of the systems* at the biological level of the stress-survival system [18]. Cortisol synthesis has a pulsating characteristic and follows daily rhythms. The concentration of cortisol in the blood is the highest during wakefulness and significantly decreases during sleep. The amount of cortisol is at its maximum upon awakening and decreases to the basal level within an hour [14]. BDNF synthesis follows circadian and seasonal rhythms, with higher levels observed in spring and summer and lower levels in fall and winter. Full sleep and rest can correct BDNF levels in humans [41]. Disturbances to the circadian rhythm can be a stress factor that increases the allostatic load in both adult humans [45] and rodents [48].

The stress-coping system is a cycle between responding to stressors and recovering resources. Adrenaline and noradrenaline are released, rapidly mobilizing the body for "fight or flight" responses. Adrenaline also triggers insulin release, which enhances glucose uptake in active organs. Consequently, adrenaline levels in the blood decreased.

If the body does not actively respond and high adrenaline levels remain in the blood, the HPAA is triggered. The release of glucocorticoids promotes prolonged mobilization of the body and maintains high energy levels while suppressing the immune and reproductive systems and slowing the body's development. The stress response is terminated by activated glucocorticoid receptors, which reduce HPAA activity through negative biofeedback. To prevent a stress state, high levels of endogenous opioids are produced [49].

The excessive production of reactive oxygen species is the final stage of the damaging effect of stressors. Antioxidant systems are then activated to prevent this effect [50]. The HPAA, autonomic nervous system, and immune system interact to regulate the hormonal and inflammatory response to stressors [37]. The maintenance of organismal resistance is achieved through the optimal ratio of neuropeptide Y and corticotropin-releasing hormone and the balance of glucocorticoid and mineralocorticoid receptors [18].

REGRESSIVE LINE OF AN ORGANISM'S DEVELOPMENT IN STRESSFUL SITUATIONS

Chronic and repetitive stressors and prolonged anticipation of stress exposure can prolong the action of glucocorticoids, impair HPAA regulation, and cause depletion of resources [28]. Stress caused by living in a crowded city and a lack of personal space can lead to immune system dysfunction, chronic inflammation, and development of various diseases, including autoimmune, allergic, cardiovascular diseases and cancer [51].

The cardiovascular system is a universal indicator of an organism's adaptation and adaptive reactions. Excessive secretion of mineralocorticoids and hyperaldosteronism correlate with increased blood pressure and development of myocardial necrosis [52]. Cortisol levels were significantly higher in patients with hypertension and patients with panic disorders than in controls [33].

Irritable bowel syndrome (IBS) is linked to impaired immune function, changes in microbiota composition, and increased levels of serotonin and BDNF in the plasma [53]. During the exacerbation stage, patients with IBS have significantly lower levels of serotonin (151.91 ± 12.46 ng/mL) and higher levels of serum cortisol (656.96 ± 25.86 nmol/L) compared to healthy controls [54]. IBS is associated with mental disorders such as neuroticism, anxiety, and depression [55].

An analysis of biochemical studies on major depressive disorder revealed high levels of cortisol [26, 28], IL-1β, IL-6, and TNF-α [37]. In addition, low BDNF levels [56] and maximal activation of the HPAA, corticotropin-releasing hormone, and noradrenergic system [2] were observed. Persistent HPAA hyperactivation is associated with a high probability of Alzheimer's disease progression [57]. Persistent HPAA hyperactivation is associated with a high risk

of Alzheimer's disease progression [57]. Chronically high levels of catecholamines, corticotropin-releasing hormone, and inflammatory markers and reduced cortisol levels were found to correlate with PTSD [58].

PROGRESSIVE LINE OF AN ORGANISM'S DEVELOPMENT DURING STRESSFUL SITUATIONS

Publications found through keyword searches in *Nature* and RSCI databases did not meet our scientific criteria and did not provide sufficient information on the signs of progressive development in a stressful situation. Our future research will focus on analyzing other neurochemical systems, such as monoamines and opioid receptors that affect the energetic and emotional aspects of human behavior in challenging life situations, including those associated with various stresses, such as physical, intellectual, and emotional stress.

The maintenance of intellectual processes in CI realization is likely caused by the neurochemical systems of acetylcholine and serotonin. The biochemistry of the emotional aspects of CI should be sought in the area of opioid receptors (κ, δ, and μ) and microbiota that influence emotional dispositions, emotional state, and human behavior.

CONCLUSIONS

This study was conducted to search for neurochemical markers indicative of different CI levels. The identification of CI biomarkers allows us to describe some aspects of the regressive and stable line of human development in stressful situations.

Genetic predisposition refers to an organism's sensitivity to certain stressful influences. Exposure to stressful situations increases the risk of developing stress-related diseases, which can be linked to a propensity for regressive lineage. During early childhood and current life, epigenetic factors can also establish certain trajectories of physiological and behavioral reactions in relation to future events.

Poor maternal care in infancy or a highly stressful life in adulthood can activate an unfavorable scenario that alters the expression of some genes. However, a low CI, which leads to unproductive resolution of difficult life situations and poor health (such as suboptimal cortisol levels, unbalanced cytokine profile, and unhealthy microbiota), is not predetermined by "bad genetics" or unfavorable living conditions.

Conscious management of nutrition, physical activity, and rest and the maintenance of adequate sleep allows for the restoration of resources and the correction of the organism's biochemical status. In stressful situations, stable human development requires a balanced stress-coping system, including a balance of mineralocorticoid and glucocorticoid receptors, an optimal ratio of DHEA-S and cortisol, and a healthy composition of microbiota. In addition, a multidimensional mental model of the stressful situation, considering both subjective and extra-subjective resources, must be constructed.

ADDITIONAL INFORMATION

Contribution of the authors. E.V.V. — conceptualization, editing, approval of the manuscript for publication; I.O.K. — review of publications on the topic of the article, processing of literary data, writing the text of the manuscript.

Funding. The study was supported by the Russian Science Foundation grant No. 23-18-00293, https://rscf.ru/project/23-18-00293/.

Conflict of interest. The authors declare that there is no conflict of interest in the presented article.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. Е.В.В. — концептуализация, редактирование, утверждение рукописи для публикации; И.О.К. — обзор публикаций по теме статьи, обработка литературных данных, написание текста рукописи.

Источник финансирования. Исследование выполнено при поддержке гранта Российского научного фонда №23-18-00293, https://rscf.ru/project/23-18-00293/.

Конфликт интересов. Авторы заявляют об отсутствии конфликта интересов по представленной статье.

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