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IMPACT OF STRESS ON EMOTIONAL HEALTH AND COGNITIVE FUNCTION

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Background. Emotional stress can have both a positive effect, which is aimed at adaptation, and a negative one, which affects the higher integrative functions of the brain, and also leads to the development of numerous diseases. In this regard, the problem of establishing the influence of stress factors on the emotional state and cognitive function becomes relevant, which creates the prerequisites for a detailed analysis of the scientific data.

Aim: to investigate the impact of chronic stress on emotional health and to determine the impact of stressful factors on human cognitive functions.

Material and methods. The review included 63 articles, which have been selected using the following keywords: «chronic stress», «cortisol», «cognitive functions», «emotions», «memory», in the databases of scientific medical data PubMed, Scopus and Web of Science. An analysis of the existing research results on the impact of stress on emotional health and cognitive functions was carried out.

Results. Stress causes a multiple effect on the human nervous system, leading to structural changes in different parts of the brain such as atrophy and reduction of brain volume and mass with long-term consequences for the nervous system resulting in impaired cognitive abilities and memory. Alteration of neuronal plasticity, caused by chronic stress, due to dendrite atrophy and decreased spinal density may underlie the depressive disorders. Additionally, chronic inflammation, which also results from prolonged stress, can develop depression and disturb cognitive functions. The hippocampus contains the high density of glucocorticoids receptors, thus increased basal concentration of cortisol may result in functional and structural changes in the hippocampus with atrophy and impaired neurogenesis. Chronic stress can affect cognitive function both acutely and chronically. The acute effect is caused by beta-adrenergic effects, while the chronic effect is caused by long-term changes in gene expression mediated by steroid hormones.

Conclusion. Chronic stress with an increased basal concentration of glucocorticoids affects the hippocampus leading to impaired memory, cognition, and thinking, also increases risk of depression, anxiety disorders.

Key words: chronic stress, cortisol, cognitive functions, emotions, memory.

Background. Stress is a disease of the modern lifestyle. A moderate and controlled eustress has a positive effect on physical and mental health [1], while uncontrollable severe stress – distress – usually results in significant damage of individual's health and increases a risk of cardiovascular diseases [2, 3], mental disorders such as depression and anxiety [4, 5, 6], autoimmune diseases [7], tumors [8].

Studies show that stress causes a multiple negative effect on the human nervous system, resulting in structural changes in some areas of the brain [9]. In conditions of chronic stress, atrophy of brain and reduction of brain volume and mass

[10] as well as structural changes in the brain with long-term consequences for the nervous system [11] and impaired cognitive abilities and memory [12] were found out.

The acute affection of cognitive functions by stress-factors was proved to be associated with catecholamines influence on the central nervous system, whereas chronic alterations were more probably caused by glucocorticoids [13]. It was demonstrated that an impact of chronic stress on memory and learning is related to the hippocampus [14, 15,16].

Over the past few decades, the role of limbic system, particularly the hippocampus, and

prefrontal cortex in adaptation to stress have been actively investigated and discussed. The findings proved the involvement of mentioned brain structures in development of cognitive dysfunction in stress-conditions [17, 18, 19].

Thus, the large number of investigations devoted to chronic stress influence on mental and physical health resulted in the modern concept of stress-associated development of illness. However, the mechanisms by which the stress chemicals can cause depression are largely undefined.

Aim: to investigate the impact of chronic stress on emotional health and to determine the impact of stressful factors on human cognitive functions.

MATERIAL AND METHODS

The review included 63 articles, which have been selected using the following keywords: «chronic stress», «cortisol», «cognitive functions», «emotions», «memory», in the databases of scientific medical data PubMed, Scopus and Web of Science. An analysis of the existing research results on the impact of stress on emotional health and cognitive functions was carried out.

RESULTS AND DISCUSSION

Stress is a negative life experience followed by physiological, cognitive, emotional and behavioral remodeling aimed at changing the event or adapting to its consequences. Any situation or event that requires adaptation can be experienced as stress [20]. Thus, stress is not defined by negative events only, but also may be driven by positive ones (for example, marriage, school attendance, etc.). If stress lasts a long time or recurs frequently, it becomes chronic [21], that causes a variety of dysfunctions [22]. Currently, it has been stated that stress is one of the causes for most of diseases [23].

In science, there is an idea that emotional stress, as a psycho-emotional state of the subject, is characterized by a complex of psychophysiological, autonomic and hormonal manifestations. Symptoms of emotional stress can be both physical and psychological. Physical symptoms include: heart palpitations or chest pain,

general body aches, headaches, teeth grinding or clenching of the jaws, shortness of breath, dizziness, feeling tired, anxiety, depression, weight loss or gain, gastrointestinal problems, drowsiness or insomnia. Psychological symptoms include: feeling overwhelmed, increased emotionality, trouble remembering, trouble making decisions, impaired concentration, alcohol or drug use [5].

The effectiveness and velocity of adaptation to stressful factors in high extend depend upon the person's capability to anticipate and control a particular stressor as well as upon the rate of functions normalization once equilibrium is restored.

Thus, the stress response, characterized by neuroendocrine and behavioral indices, shows whether a given stressor is manageable or, on the contrary, cannot be managed and, therefore, becomes harmful to the body. This means that not all stimuli that trigger strong neuroendocrine responses are true stressors, but only those that exceed a person's ability to change and adapt. The stress response is formed due to the activation of pituitary-adrenal mechanisms, including the activation of ACTH and adrenocorticoid function of the adrenal glands [24].

With emotional stress, selective disorders of various physiological functions can occur: cardiovascular, gastrointestinal and others. On the background of relative stability of some physiological functions, disorders of others may occur. Thus, emotional stress is a systemic multi-level reaction of the body to a conflict situation. Currently, there is a lot of scientific data on the effects of chronic stress on brain structures [25]. Chronic stress has been shown to be associated with changes in certain areas of the brain. For example, animal studies have described stress-related effects in the prefrontal cortex and limbic system, characterized by a decrease in the volume of some structures and an alteration in neuronal plasticity due to dendrite atrophy and decreased spinal density [26]. These morphological changes are similar to those found in the brains of depressed patients examined posthumously, suggesting that they may underlie the depressive disorders that are often associated with chronic stress in humans.

Thus, Blix E. and colleagues observed atrophy of the basal ganglia and a significant decrease in gray matter in certain areas of the prefrontal cortex in subjects suffering from long-term occupational stress [27].

It should be noted that the mechanisms by which stress chemicals can cause depression are largely undefined, but active research is underway. Thus, Ota K.T. and colleagues (2014) identified a molecular mechanism triggered by chronic stress that promotes neuronal atrophy in prefrontal cortex and limbic system commonly seen in depressed patients, regardless of the cause of the depression [28]. Authors reported the depression of genes which regulate protein synthesis-dependent synaptic plasticity in neurons of prefrontal cortex in chronic stress conditions in experimental rats.

Stress-induced cytokines have also been found to be involved in the development of chronic depression. Chronic inflammation resulting from prolonged stress can contribute in depression development and cognitive functions violation. It should be noted that the biochemical mechanisms underlying cytokine-induced depression are not well defined, but may include alterations in serotonin and glutamatergic transmission [29].

There are multiple data revealed that physical exercises can support brain health by regulating the production of neurotrophic factors – neurotransmitters, which can lead to improved cognitive abilities, reduced risk of neurodegenerative diseases, and mitigation of depression [30].

In stressful situations the pituitary gland and adrenal cortex secrete hormones which are critical for capability to withstand the stress-conditions and yet play an important role in behavior, memory, and cognition [31]. The hippocampus is the most important link between different brain structures in converting short-term memory into long-term memory. It was found out that the hippocampus contains the highest density of glucocorticosteroid receptors [17]. The results of Mak'yun B.S. research have shown that stress can introduce functional and structural changes in the hippocampus that lead to atrophy and impaired neurogenesis [32]. In addition, chronic stress causes an increase in plasma cortisol concentration that may result

in the destruction of the dendrites and synaptic endings of neurons [33].

It has been found that high concentrations of glucocorticosteroids in plasma for a long time can cause atrophy of the hippocampus, which leads to memory impairment [34]. In addition, people with Cushing's syndrome (increased secretion of glucocorticosteroids) or people receiving high doses of exogenous synthetic anti-inflammatory drugs have hippocampal atrophy and associated memory impairment [35]. Studies have shown that even routine therapeutic doses of glucocorticosteroids and dexamethasone can cause memory problems [36].

Two factors are involved in the memory process during times of stress: norepinephrine, which creates the emotional aspects of memories in the basolateral area of the amygdala, and corticosteroids. There is a reciprocal balance between these two hormones to the creation of a response in the memory process. An important condition for adaptation to emotional stress is the body's ability to restore normal levels of norepinephrine in the hypothalamus, increase its content in the midbrain, and also increase the level of dopamine in the hypothalamus, midbrain and medulla oblongata. [37].

It should be noted that stress does not always have a negative effect on memory, in some situations stress can sharpen memory [38]. For example, in Vedhara K. research, it was described that when taking a written exam, participants can improve their memory for a short time, which is associated with a decrease in the concentration of cortisol in saliva [39]. Other studies have shown that stress, before starting to study, can also increase memory or decreases it [40, 41].

Chronic stress can affect cognitive function both acutely and chronically. The acute effect is caused by beta-adrenergic effects, while the chronic effect is caused by long-term changes in gene expression mediated by steroid hormones [42].

CONCLUSION

Review of scientific data allowed to conclude that chronic stress activates the autonomic nervous system, the central neurotransmitter link, the

neuropeptide link, as well as the hypothalamic-pituitary system, which leads to the increase in the basal concentration of glucocorticoids. High concentrations of glucocorticoids can affect the hippocampus, the main link in the brain responsible for memory, cognition, and learning. In chronic stress, the state of depression and anxiety may be associated with neural degeneration and lowering of protein synthesis-dependent synaptic plasticity in prefrontal cortex and limbic system. Coping techniques are important for both mental and physical health (proper stimulation of peripheral tissues, such as skeletal muscle, can alleviate symptoms of stress), constructive problem solving in crisis situations, relaxation techniques, and adaptation to unexpected life changes. Since stressful situations are often impossible to avoid completely, you need to look at them as a source of new experiences, new opportunities and perspectives.

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REFERENCE

1. Hostinar CE, Gunnar MR. Social Support Can Buffer against Stress and Shape Brain Activity. *AJOB Neurosci*. 2015 Jul 1;6(3):34-42. DOI: 10.1080/21507740.2015.1047054.
2. Aggarwal NT, Clark CJ, Beck TL, Mendes de Leon CF, DeCarli C, Evans DA, Everson Rose SA. Perceived stress is associated with subclinical cerebrovascular disease in older adults. *Am J Geriatr Psychiatry*. 2014 Jan;22(1):53-62. DOI: 10.1016/j.jagp.2012.06.001.
3. Tsutsumi A, Kayaba K, Kario K, Ishikawa S. Prospective study on occupational stress and risk of stroke. *Arch Intern Med*. 2009 Jan 12;169(1):56-61. DOI: 10.1001/archinternmed.2008.503.
4. Catabay CJ, Stockman JK, Campbell JC, Tsuyuki K. Perceived stress and mental health: The mediating roles of social support and resilience among black women exposed to sexual violence. *J Affect Disord*. 2019 Dec 1;259:143-149. DOI: 10.1016/j.jad.2019.08.037.
5. Koolhaas JM, Bartolomucci A, Buwalda B, de Boer SF, Flügge G, Korte SM, Meerlo P, Murison R, Olivier B, Palanza P, Richter-Levin G, Sgoifo A, Steimer T, Stiedl O, van Dijk G, Wöhr M, Fuchs E. Stress revisited: a critical evaluation of the stress concept. *Neurosci Biobehav Rev*. 2011 Apr;35(5):1291-301. DOI: 10.1016/j.neubiorev.2011.02.003.
6. Dantzer R, O'Connor JC, Freund GG, Johnson RW, Kelley KW. From inflammation to sickness and depression: when the immune system subjugates the brain. *Nat Rev Neurosci*. 2008 Jan;9(1):46-56. DOI: 10.1038/nrn2297.
7. Glaser R, Kiecolt-Glaser JK. Stress-induced immune dysfunction: implications for health. *Nat Rev Immunol*. 2005 Mar;5(3):243-51. DOI: 10.1038/nri1571.
8. Reiche EM, Nunes SO, Morimoto HK. Stress, depression, the immune system, and cancer. *Lancet Oncol*. 2004 Oct;5(10):617-25. DOI: 10.1016/S1470-2045(04)01597-9.
9. Lupien SJ, McEwen BS, Gunnar MR, Heim C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nat Rev Neurosci*. 2009 Jun;10(6):434-45. DOI: 10.1038/nrn2639.
10. Yaribeygi H, Panahi Y, Sahraei H, Johnston TP, Sahebkar A. The impact of stress on body function: A review. *EXCLI J*. 2017 Jul 21;16:1057-1072. DOI: 10.17179/excli2017-480.
11. Reznikov LR, Grillo CA, Piroli GG, Pasumarthi RK, Reagan LP, Fadel J. Acute stress-mediated increases in extracellular glutamate levels in the rat amygdala: differential effects of antidepressant treatment. *Eur J Neurosci*. 2007 May;25(10):3109-14. DOI: 10.1111/j.1460-9568.2007.05560.x.
12. Lupien SJ, McEwen BS, Gunnar MR, Heim C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nat Rev Neurosci*. 2009 Jun;10(6):434-45. DOI: 10.1038/nrn2639.
13. McEwen BS, Sapolsky RM. Stress and cognitive function. *Curr Opin Neurobiol*. 1995 Apr;5(2):205-16. DOI: 10.1016/0959-4388(95)80028-x.
14. Borcel E, Pérez-Alvarez L, Herrero AI, Brionne T, Varea E, Berezin V, Bock E, Sandi C, Venero C. Chronic stress in adulthood followed by

- intermittent stress impairs spatial memory and the survival of newborn hippocampal cells in aging animals: prevention by FGL, a peptide mimetic of neural cell adhesion molecule. *Behav Pharmacol.* 2008 Feb;19(1):41-9. DOI: 10.1097/FBP.0b013e3282f3fca9.
15. Sandi C, Davies HA, Cordero MI, Rodriguez JJ, Popov VI, Stewart MG. Rapid reversal of stress induced loss of synapses in CA3 of rat hippocampus following water maze training. *Eur J Neurosci.* 2003 Jun;17(11):2447-56. DOI: 10.1046/j.1460-9568.2003.02675.x.
 16. Joëls M, Fernandez G, Roozendaal B. Stress and emotional memory: a matter of timing. *Trends Cogn Sci.* 2011 Jun;15(6):280-8. DOI: 10.1016/j.tics.2011.04.004.
 17. de Kloet ER, Oitzl MS, Joëls M. Stress and cognition: are corticosteroids good or bad guys? *Trends Neurosci.* 1999 Oct;22(10):422-6. DOI: 10.1016/s0166-2236(99)01438-1.
 18. Lupien SJ, Lepage M. Stress, memory, and the hippocampus: can't live with it, can't live without it. *Behav Brain Res.* 2001 Dec 14;127(1-2):137-58. DOI: 10.1016/s0166-4328(01)00361-8.
 19. McEwen BS, Bowles NP, Gray JD, Hill MN, Hunter RG, Karatsoreos IN, Nasca C. Mechanisms of stress in the brain. *Nat Neurosci.* 2015 Oct;18(10):1353-63. DOI: 10.1038/nn.4086.
 20. Dhabhar FS. Effects of stress on immune function: the good, the bad, and the beautiful. *Immunol Res.* 2014 May;58(2-3):193-210. DOI: 10.1007/s12026-014-8517-0. PMID: 24798553.
 21. Silverman MN, Sternberg EM. Glucocorticoid regulation of inflammation and its functional correlates: from HPA axis to glucocorticoid receptor dysfunction. *Ann N Y Acad Sci.* 2012 Jul;1261:55-63. DOI: 10.1111/j.1749-6632.2012.06633.x.
 22. Yaribeygi H, Panahi Y, Sahraei H, Johnston TP, Sahebkar A. The impact of stress on body function: A review. *EXCLI J.* 2017 Jul 21;16:1057-1072. DOI: 10.17179/excli2017-480.
 23. Gawlik KS, Melnyk BM, Tan A. Associations Between Stress and Cardiovascular Disease Risk Factors Among Million Hearts Priority Populations. *Am J Health Promot.* 2019 Sep;33(7):1063-1066. DOI: 10.1177/0890117119847619.
 24. Black PH. Stress and the inflammatory response: a review of neurogenic inflammation. *Brain Behav Immun.* 2002 Dec;16(6):622-53. DOI: 10.1016/s0889-1591(02)00021-1.
 25. Mariotti A. The effects of chronic stress on health: new insights into the molecular mechanisms of brain-body communication. *Future Sci OA.* 2015 Nov 1;1(3):FSO23. DOI: 10.4155/fso.15.21.
 26. Lucassen PJ, Pruessner J, Sousa N, Almeida OF, Van Dam AM, Rajkowska G, Swaab DF, Czeh B. Neuropathology of stress. *Acta Neuropathol.* 2014 Jan;127(1):109-35. DOI: 10.1007/s00401-013-1223-5.
 27. Blix E, Perski A, Berglund H, Savic I. Long-term occupational stress is associated with regional reductions in brain tissue volumes. *PLoS One.* 2013 Jun 11;8(6):e64065. DOI: 10.1371/journal.pone.0064065.
 28. Ota KT, Liu RJ, Voleti B, Maldonado-Aviles JG, Duric V, Iwata M, Duthiel S, Duman C, Boikess S, Lewis DA, Stockmeier CA, DiLeone RJ, Rex C, Aghajanian GK, Duman RS. REDD1 is essential for stress-induced synaptic loss and depressive behavior. *Nat Med.* 2014 May;20(5):531-5. DOI: 10.1038/nm.3513.
 29. Dantzer R, O'Connor JC, Freund GG, Johnson RW, Kelley KW. From inflammation to sickness and depression: when the immune system subjugates the brain. *Nat Rev Neurosci.* 2008 Jan;9(1):46-56. DOI: 10.1038/nrn2297.
 30. Cotman CW, Berchtold NC, Christie LA. Exercise builds brain health: key roles of growth factor cascades and inflammation. *Trends Neurosci.* 2007 Sep;30(9):464-72. DOI: 10.1016/j.tics.2007.06.011.
 31. de Kloet ER. Stress in the brain. *Eur J Pharmacol.* 2000 Sep 29;405(1-3):187-98. DOI: 10.1016/s0014-2999(00)00552-5.
 32. McEwen BS. Stress and hippocampal plasticity. *Annu Rev Neurosci.* 1999;22:105-22. DOI: 10.1146/annurev.neuro.22.1.105.
 33. Sapolsky RM, Uno H, Rebert CS, Finch CE. Hippocampal damage associated with prolonged glucocorticoid exposure in primates. *J Neurosci.* 1990 Sep;10(9):2897-902. DOI: 10.1523/JNEUROSCI.10-09-02897.1990.

34. Issa AM, Rowe W, Gauthier S, Meaney MJ. Hypothalamic-pituitary-adrenal activity in aged, cognitively impaired and cognitively unimpaired rats. *J Neurosci*. 1990 Oct;10(10):3247-54. DOI: 10.1523/JNEUROSCI.10-10-03247.1990. PID: 2170594; PMCID: PMC6570181.
35. Ling MH, Perry PJ, Tsuang MT. Side effects of corticosteroid therapy. *Psychiatric aspects. Arch Gen Psychiatry*. 1981 Apr;38(4):471-7. DOI: 10.1001/archpsyc.1981.01780290105011.
36. Kirschbaum C, Wolf OT, May M, Wippich W, Hellhammer DH. Stress- and treatment-induced elevations of cortisol levels associated with impaired declarative memory in healthy adults. *Life Sci*. 1996;58(17):1475-83. DOI: 10.1016/0024-3205(96)00118-x.
37. Joëls M, Fernandez G, Roozendaal B. Stress and emotional memory: a matter of timing. *Trends Cogn Sci*. 2011 Jun;15(6):280-8. DOI: 10.1016/j.tics.2011.04.004.
38. Schwabe L, Wolf OT, Oitzl MS. Memory formation under stress: quantity and quality. *Neurosci Biobehav Rev*. 2010 Mar;34(4):584-91. DOI: 10.1016/j.neubiorev.2009.11.015.
39. Vedhara K, Hyde J, Gilchrist ID, Tytherleigh M, Plummer S. Acute stress, memory, attention and cortisol. *Psychoneuroendocrinology*. 2000 Aug;25(6):535-49. DOI: 10.1016/S0306-4530(00)00008-1. PMID: 10840167.
40. Schwabe L, Bohringer A, Chatterjee M, Schachinger H. Effects of pre-learning stress on memory for neutral, positive and negative words: Different roles of cortisol and autonomic arousal. *Neurobiol Learn Mem*. 2008 Jul;90(1):44-53. DOI: 10.1016/j.nlm.2008.02.002.
41. Diamond DM, Campbell AM, Park CR, Woodson JC, Conrad CD, Bachstetter AD, Mervis RF. Influence of predator stress on the consolidation versus retrieval of long-term spatial memory and hippocampal spinogenesis. *Hippocampus*. 2006;16(7):571-6. DOI: 10.1002/hipo.20188.
42. Diamond DM, Campbell AM, Park CR, Woodson JC, Conrad CD, Bachstetter AD, Mervis RF. Influence of predator stress on the consolidation versus retrieval of long-term spatial memory and hippocampal spinogenesis. *Hippocampus*. 2006;16(7):571-6. DOI: 10.1002/hipo.20188.

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ВПЛИВ СТРЕСУ НА ЕМОЦІЙНЕ ЗДОРОВ'Я ТА КОГНІТИВНІ ФУНКЦІЇ

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Актуальність. Емоційний стрес може мати як позитивний вплив, який спрямований на адаптацію, так і негативний, який впливає на вищі інтегративні функції головного мозку, а також призводить до розвитку численних захворювань. У зв'язку з цим є актуальним встановлення впливу стресових факторів на емоційний стан та когнітивні функції, що створює передумови для детального аналізу існуючих наукових даних.

Ціль: дослідити вплив хронічного стресу на емоційне здоров'я та визначити вплив стресових чинників на когнітивні функції людини.

Матеріали та методи. Проведено огляд 63 статті, які були відібрані за такими ключовими словами: «хронічний стрес», «кортизол», «когнітивні функції», «емоції», «пам'ять», у базах наукових медичних даних PubMed, Scopus та Web of Science. Проведено аналіз існуючих результатів досліджень про вплив стресу на емоційне здоров'я та когнітивні функції.

Результати. Стрес спричиняє численний вплив на нервову систему, що призводить до структурних і функціональних змін у різних ділянках мозку, таких як атрофія, зменшення об'єму та маси мозку з довгостроковими наслідками для нервової системи, в результаті чого відбувається порушення когнітивних здібностей та пам'яті. В основі депресивних розладів може лежати зміна пластичності нейронів, викликана хронічним стресом. Крім того, хронічне запалення, яке також може бути результатом тривалого стресу, може бути чинником розвитку депресії та порушувати когнітивні функції. Гіпокамп містить високу щільність глюкокортикоїдних рецепторів, тому підвищена базальна концентрація кортизолу може призводити до функціональних і структурних змін гіпокампу з атрофією і порушенням нейрогенезу. Хронічний стрес може впливати на когнітивні функції як гостро, так і хронічно. Гострий ефект викликається адренергічними ефектами, тоді як хронічний ефект викликаний тривалими змінами експресії генів, опосередкованими стероїдними гормонами.

Висновок. Хронічний стрес з підвищеною базальною концентрацією глюкокортикоїдів впливає на структуру та функціональну активність гіпокампу, приводячи до порушення пам'яті, когнітивних функцій і мислення, також підвищує ризик розвитку депресії та тривожних станів.

Ключові слова: хронічний стрес, кортизол, когнітивні функції, емоції, пам'ять.