



Review of literature: Effect of stress on brain cells in adults

Rand Abdulateef Abdullah *

Department of Anatomy, College of Medicine, University of Mosul, Mosul, Iraq

GSC Biological and Pharmaceutical Sciences, 2025, 31(02), 065-067

Publication history: Received on 14 March 2025; revised on 03 May 2025; accepted on 05 May 2025

Article DOI: <https://doi.org/10.30574/gscbps.2025.31.2.0169>

Abstract

The literature review explores how stress influences adult brain cells, beginning with an explanation that stress is a natural, often beneficial reaction (known as eustress) that can enhance focus and performance. However, when stress becomes chronic or excessive, it leads to detrimental physical, emotional, and behavioral effects, including impaired immunity and an increased risk of various health disorders. The review details how the brain orchestrates the stress response through the HPA axis and autonomic nervous system, triggering the release of hormones such as cortisol. Key brain regions like the hippocampus—which is essential for memory and regulation of the stress response—are particularly vulnerable; prolonged exposure to stress hormones can lead to hippocampal atrophy and disrupted neuronal function. Additionally, the review touches on conditions like PTSD, where abnormal cortisol responses and increased neuroinflammation further compromise brain cell function. The overall aim is to understand how stress exposure affects the microscopic structure of brain cells in adults.

Keyword: Stress; Brain Cells; Neuron Degeneration; Hippocampus Atrophy

1. Introduction

Stress is defined as the natural reaction of the body to demands or challenges occur. It can result in many different physical, emotional and behavioral responses(1). Stress is a normal part of life, to some extent, it is a necessary part as it has positive aspects. It can be a motivation like that for students helping them to achieve their targets and named as eustress which is the positive cognitive response to stress that is healthy comes from exciting events and positive challenges. So, it can be beneficial inspiring and encouraging personal growth improving the concentration to meet challenges (2).

The body is designed to experience stress and react to it but the problem with stress is when it became chronic and when there is excess exposure to stress. Mostly people are unaware of stress only they become aware when warning signs appear and start to suffer from serious body health problems (3).

According to the American Psychological Association (APA) the most five common sources of stress are Money, Work, Family responsibilities, and Health concerns (4). In many cases, the pathophysiological complications of disease occur from stress like those who work or live in stressful environments, have a higher risk of many disorders (5).

Stress also effects immunity as it stimulates the immune system's ability to fight off antigens is reduced. Individuals under stress are more vulnerable to infections. corticosteroid is considered as stress hormone which can reduce the normal action of immunity and it may end with cardiac and vascular problems (6).

Aim of this review study is to find out the effect of exposure to stress on the histological structure of the brain cells in adults.

* Corresponding author: Rand Abdulateef Abdullah

2. Mechanisms of stress in the brain

The organ responsible for response and adaptation to stressing factors is the brain as it is the only organ that determine the threatens, emotional compressions and it maintains the physiological and social responses that may be harmful or defensive (7).

The activation of the autonomic nervous system and hypothalamo-pituitary-adrenal (HPA) axis is the key of the stress response, the hypothalamus and brain stem, are crucial in autonomic and neuroendocrine responses to stressors. In addition, the “fight-or-flight” response is the standard path of predicting the behavioral and physiological responses to a threat from a dangerous situation. These specific regions in the brain are considered as targets of stress and stress hormones release and the impact of the stress experiences will affect how they respond in combination with genetic factors (8).

In 1960s researchers found that there are receptors for adrenal steroids in the hippocampal formation (9), which is the site for spatial, episodic, and contextual memory formation (10). This has led to a wide variety of researches about the functional significances of adrenal steroids action over the life span.

Landfield and Sapolsky found that adrenal hormones can lead to gradual atrophy of hippocampus which plays an important role in stopping the HPA stress response and damage or atrophy of the hippocampus impairs the shut off and leads to a more prolonged HPA response to psychological stressors (11). This led to the “glucocorticoid cascade hypothesis” of stress and aging (12). It is found that excessive secretion of cortisol during a yearly exam over a 5-years period is associated with decrease hippocampal volume and performance (13).

The glucocorticoids act on their receptors in hippocampus and other brain regions through different factors including corticosteroid binding globulin (CBG), multiple drug resistance P-glycoprotein (MDRpG), and metabolism by 11-hydroxysteroid dehydrogenase type 1 (11 HSD-1). The corticosteroid binding globulin in the blood binds natural glucocorticoids such as corticosterone, cortisol, and their 11-dehydro-metabolites, but not the synthetic glucocorticoid dexamethasone; only unbound steroid is able to enter the brain (14).

Post-traumatic stress disorder (PTSD) is a special illness which occurs after suffering disturbing occasion, such as military war and car accidents. This condition is associated to changes in the stress-response systems which occurs in the brain cells, that regulates the body response. Research found that cortisol levels in people with PTSD can appear normal everyday but become low when exposed to stressful situations. This abnormal response contributes to the symptoms of PTSD. Additionally, PTSD is associated with increased activity in certain brain systems, leading to increased levels of norepinephrine (15).

Chronic exposure to stress will induce the immune system to secrete more pro-inflammatory cytokines which cross the blood brain barrier inducing inflammation in the brain cells and disturbing neuronal cell function. Neuroinflammation has been linked to the development of depression, neurodegenerative changes, mental health disorders, and cognitive deterioration (16).

3. Conclusion

Chronic stress hormones can lead to hippocampal atrophy and disrupted neuronal function through the stress response by the HPA axis and autonomic nervous system stimulation which trigger the release of hormones such as cortisol.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

References

- [1] Larzelere MM, Jones GN. Stress and health. *Prim Care*. 2008; 35: 839-856.
- [2] Rasheed N. Prolonged Stress Leads to Serious Health Problems: Preventive Approaches. *Int J Health Sci (Qassim)*. 2016;10(1):V-VI. PMID: 27004066; PMCID: PMC4791152.

- [3] Ahmed SM, Lemkau JP, Hershberger PJ (2011) Psychosocial Influences on Health, In: Rakel RE, Textbook of Family Medicine, 8th ed, Philadelphia :Saunders Elsevier.
- [4] Cooper C L, Palmer S (2000) Conquer your stress. Institute of Personnel and Development
- [5] Butler G, Hope T (2007) Manage your mind. The Mental Fitness Guide. Second Edition.
- [6] Stein M, Miller AH. Stress, the immune system, and health and illness. In: Handbook of stress: Theoretical and clinical aspects, 2nd ed. New York, NY, US: Free Press; 1993. p. 127–41.
- [7] McEwen BS. Protective and Damaging Effects of Stress Mediators. New England J. Med. 1998; 338:171–179. doi: 10.1056/NEJM199801153380307.
- [8] Roozendaal B, Griffith QK, Buranday J, De Quervain DJ, McGaugh JL. The hippocampus mediates glucocorticoid-induced impairment of spatial memory retrieval: dependence on the basolateral amygdala. Proc Natl Acad Sci U S A. 2003; 100 (3):1328-33. doi: 10.1073/pnas.0337480100.
- [9] McEwen BS, Weiss JM, Schwartz LS. Selective retention of corticosterone by limbic structures in rat brain. Nature. 1968; 30;220(5170):911-2. doi: 10.1038/220911a0.
- [10] Squire LR. The hippocampus and the neuropsychology of memory. Neurobiology of the Hippocampus. 1983:491-511.
- [11] Sapolsky RM, Krey LC, McEwen BS. The neuroendocrinology of stress and aging: the glucocorticoid cascade hypothesis. Endocr Rev. 1986;7(3):284-301. doi: 10.1210/edrv-7-3-284.
- [12] Herman JP, Cullinan WE. Neurocircuitry of stress: central control of the hypothalamo-pituitary-adrenocortical axis. Trends Neurosci. 1997 ;20(2):78-84. doi: 10.1016/s0166-2236(96)10069-2.
- [13] Lupien SJ, de Leon M, de Santi S, Convit A, Tarshish C, Nair NP, Thakur M, McEwen BS, Hauger RL, Meaney MJ. Cortisol levels during human aging predict hippocampal atrophy and memory deficits. Nat Neurosci. 1998;1(1):69-73. doi: 10.1038/271.
- [14] Hill AR, Spencer-Segal JL. Glucocorticoids and the Brain after Critical Illness. Endocrinology. 2021 ;162(3): bqaa242. doi: 10.1210/edocr/bqaa242.
- [15] Miao, XR., Chen, QB., Wei, K. et al. posttraumatic stress disorder: from diagnosis to prevention. Military Med Res. 2018; 5: 32. doi: 10.1186/s40779-018-0179-0.
- [16] Ressler KJ, Smoller JW. Impact of Stress on the Brain: Pathology, Treatment and Prevention. Neuropsychopharmacology. 2016; 41(1):1-2. doi: 10.1038/npp.2015.306