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#### Research Article

# Physiological Effects of Acute and Chronic Stress in Human Body

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#### **Abstract**

Do you forget things? Have you ever felt certain uneasiness in the body or you are tossing in your bed or /you feel uneasy while sleeping? These all could be the symptoms of stress.

Stress might be helpful for short period of time. It has been proven that stress can improve productivity for short span of time but for long duration it can lead to severe illness. It affects our physiology from brain to heart it starts altering their functions.

Stress is anything that challenges our state of homeostasis. Stress has both positive and negative impact in our body. It is the respond of our body to stimulus. It is acute – short period stress and chronic – long period stress. It has been observed that positive stress helps in motivation ,increases work efficiency by releasing hormones such as Cortisol, prolactin etc. which trigger our hypothalamic-pituitary-adrenocortical (HPA) axis, resulting increasing heart rate, breathing rate which might not harmful for short duration but for long period – chronic stress it can alter bodily system and give rise to many diseases. It is essential to understand the need of stress management in our daily life.

**Keywords:** Physiological, Stress, Chronic Stress, Cortisol, Cardiovascular.

# **Background**

The term "stress" had none of its contemporary connotations before the 1920s. It is a form of the Middle English *destresse*, derived via Old French from the Latin *stringere*, "to draw tight".<sup>[1]</sup> The word had long been in use in physics to refer to the internal distribution of a force exerted on a material body, resulting in strain. In the 1920s and '30s, biological and psychological circles occasionally used the term to refer to a mental strain or to a harmful environmental agent that could cause illness.

Walter Cannon used it in 1926 to refer to external factors that disrupted what he called homeostasis.<sup>2</sup> But "stress as an explanation of lived experience is absent from both lay and

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expert life narratives before the 1930s". The awareness of stress was increased after World War II when it was noticed that many routine life events like examinations, growing up increases stress and leads to dysfunction. The word stress was used by Hans Selye for the first time in 1936 to explain the effects of threats that disturbs homeostasis. Hans Selye also defined stress as a set of non-specific responses collectively called as "General Adaptation Syndrome". Hans Selye's theory of non-specificity was criticized by Mason Goldstein defined stress as "stress is a condition in which expectations, whether genetically programmed, established by prior learning or deduced from circumstances, do not match the current or anticipated perceptions of the internal or external environment and this discrepancy between what is observed or sensed and what is expected or programmed elicits patterned, compensatory responses". Walter B. Cannon explained the role of adrenal?

l glands and sympathetic nervous system in maintenance of body equilibrium after exposure to threat. Later the word allostasis was introduced to stress research, which is the process of adaptation to different stressors. When there is any defect in the process of adaptation, it leads to "allostatic load or overload".<sup>10</sup>

Physiological stress represents a wide range of physical responses that occur as a direct effect of a stressor causing an upset in the homeostasis of the body. Upon immediate disruption of either psychological or physical equilibrium the body responds by stimulating the nervous, endocrine, and immune systems. The reaction of these systems causes a number of physical changes that have both short- and long-term effects on the body.

## Introduction

Stress can be loosely defined as anything that challenges our state of homeostasis.

Stress can be classified as

- **Acute stress**: Acute stress is a very short-term type of stress that can either be positive or more distressing; this is the type of stress we most often encounter in day-to-day life.
- Chronic stress: Chronic stress is stress that seems never-ending and inescapable, like the stress of a bad marriage or an extremely taxing job; chronic stress can also stem from traumatic experiences and childhood trauma.
- **Episodic acute stress**: Episodic acute stress is acute stress that seems to run rampant and be a way of life, creating a life of ongoing distress.
- <u>Eustress</u>/ Positive Stress: Eustress is fun and exciting. It's known as a positive type of stress that can keep you energized. It's associated with surges of adrenaline, such as when you are skiing or racing to meet a deadline. Positive stress can also refer to the times you respond well to a challenge that you experience from a stressor. Positive stress is defined by the effects it produces. These stressors allow us to live outside our comfort zones.<sup>11</sup>

Physiological effects of Acute and Chronic stress

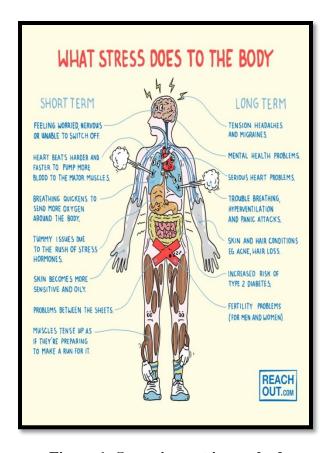


Figure 1: Stress impact in our body

Stress, either severe, acute stress or chronic low-grade stress may induce abnormalities in three principal regulatory systems in the body: serotonin systems, catecholamine systems, and the hypothalamic-pituitary-adrenocortical (HPA) axis The abnormalities in this systems is associated with aggressive behaviour.<sup>12</sup>

Stress originates in the brain, but has impact on all body systems including nervous, cardiovascular, respiratory, musculoskeletal, immune, gastrointestinal, endocrine, reproductive systems, epigenetic changes.

Reactions to stress are associated with enhanced secretion of a number of hormones such as cortisol, glucocorticoids, catecholamines, growth hormone and prolactin, secretion increases in respond to stress which effects the increase mobilization of energy sources and adapt the individual to its new circumstance.

## **Stress and Nervous System**

The nervous system consists of autonomic and somatic nervous systems. The autonomic nervous system has a direct role in physical response to stress and is divided into the sympathetic nervous system (SNS), and the parasympathetic nervous system (PNS). When the body is stressed, the SNS

contributes to what is known as the "fight or flight" response. The body shifts its energy resources toward fighting off a life threat, or fleeing from an enemy.

The Adrenal glands release hormones called *adrenalin (epinephrine)* and *cortisol* in respond to SNS signal. These hormones, together with direct actions of autonomic nerves, causing heart beat faster, respiration rate increases, blood vessels in the arms and legs dilate, digestive process change and glucose levels (sugar energy) in the bloodstream increase to tackle with the emergency.

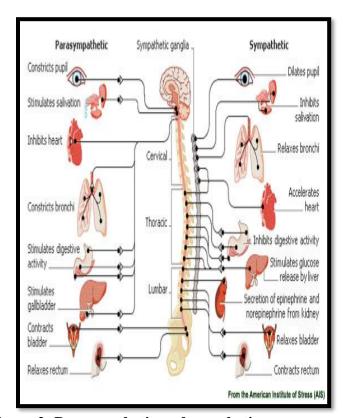


Figure 2: Parasymphetic and symphetic nervous system

The response of SNS is sudden in order to prepare body for emergency situation or acute stress—short term stressors. Once the critical situation is over, the body usually returns to the pre-emergency, unstressed state. PNS facilitates body in recovery, which generally has opposing effects to the SNS. But PNS over-activity can also contribute to stress reactions, for example by promoting bronchoconstriction (e.g., in asthma) or exaggerated vasodilation and compromised blood circulation.

Both the SNS and the PNS have powerful interactions with the immune system, which can also modulate stress reactions. The central nervous system is particularly important in triggering stress responses, as it regulates the autonomic nervous system and plays a central role in interpreting contexts as potentially threatening.

Experiencing stressor over a long period of time called chronic stress, results in a long –term drain on the body, which causes wear and tear due to triggering physical reaction by automatic nervous

system. Continuous activation of nervous system causes problem in physical body which is less problematic than that of what chronic stress does to nervous system.<sup>13</sup>

## **Stress and Cardiovascular System**

Cardiovascular diseases due to stress include hypertension, myocardial ischaemia, myocardial infarction, ventricular arrhythmia, and sudden cardiac death.<sup>14</sup>



Figure 3: Impact of chronic stress in heart.

Stress causes hazardous effects on cardiovascular system through activation of sympathetic nervous system and vagal withdrawal. Short term stress—Acute stress such as argument with your friends/family, deadline pressure, job interviews etc. causes an increase in heart rate and stronger contractions of the heart muscle due to the stress hormones - adrenaline, noradrenaline, and cortisol.

The body returns to its normal state as the acute stress passes. Chronic stress, or a constant stress experienced for long period of time, can contribute to long-term problems for heart and blood vessels. The consistent and ongoing increase in heart rate, and the elevated levels of stress hormones and of blood pressure, can increase the risk for hypertension, heart attack, or stroke.

Repeated acute stress and persistent chronic stress may also contribute to inflammation in the circulatory system, particularly in the coronary arteries, and this is one pathway that is thought to tie stress to heart attack. It also appears that how a person responds to stress can affect cholesterol levels.

The risk for heart disease associated with stress appears to differ for women, depending on whether the woman is premenopausal or postmenopausal. Levels of estrogen in premenopausal women appears to help blood vessels respond better during stress, thereby helping their bodies to better handle stress and protecting them against heart disease. Postmenopausal women lose this level of protection due to loss of estrogen, therefore putting them at greater risk for the effects of stress on heart disease. <sup>15</sup>

Perceived mental stress was associated with increased mortality from stroke for women and with CHD for men and women.<sup>16</sup>

# Stress and Musculoskeletal System

Figure 4: Stress and musculoskeletal disease

In order to protect an individual from injury and pain, reflex phenomenon muscle tension increases. However chronic exposure to stress causes prolonged increase in muscle tension and promotes musculo- skeletal disorders. The muscles of shoulders, neck and jaw, accessory muscles of respiration and diaphragm are commonly affected due to stress. 18

# Stress and Immune System

Activation of HPA axis during stress modulates both innate and adaptive immunity through increase in secretion of glucocorticoids. Cortisol modulates number and function of leucocytes, decreases production of cytokines, inhibits pro-inflammatory pathways in target tissues. Hence, chronic activation of HPA axis increases susceptibility to infections and tumors. Activation of autonomic system during stress has direct suppressive action on immune cells/organs. Glucocorticoids and catecholamines modulate balance between T helper-1 versus T helper-2 responses. <sup>18,19</sup>

#### Stress and Blood

The changes in parameter of blood cell are induced by Stress. <sup>20</sup> It was studied that, stress induced release of cortisol decreases the eosinophils by increasing their sequestration in spleen and also decreases the number of basophils whereas stress increases number of neutrophils, platelets and red blood cells. <sup>21</sup>

# **Stress and Gastro Intestinal System**

Stress by inhibiting dorsal vagal complex and increasing colonic motor activity suppresses gastric emptying through stimulation of sacral parasympathetic system. Stress weakens gastro intestinal barrier also increases gastrointestinal permeability and contributes to inflammatory bowel disease. Chronic stress decreases pain threshold for visceral sensations in patients with gastro intestinal disorders. On oral health, Stress has negative impact as there is a decrease in the salivary flow rates, total protein concentration was observed in stressful conditions. Further, stress contributes

to increase in the salivary cortisol and pH.<sup>27</sup>

#### **Stress and Metabolism**

Stress increases catabolism of glucose, lipids and proteins and simultaneously inhibits anabolic actions of growth hormone, insulin and sex steroids. This action of stress may be beneficial for short term basis to cope up with stressor. However, chronic activation of stress axis leads to metabolic syndrome through activation of HPA, LC/ NE sympathetic adrenal system and changes in health behavior. HPA axis activation increases gluconeogenesis in liver, increases plasma glucose levels, increases lipolysis, increases protein breakdown at skeletal muscles, bone and skin, which leads to visceral obesity, insulin resistance, atherosclerosis, decreases osteoblastic activity, muscle and bone mass. Further, chronic stress causes changes in physical activity like following sedentary life style, increased sleeping hours, changes in diet habits, which further worsen regulation of metabolic functions and increases the incidence of metabolic syndrome.<sup>28-31</sup>

# **Stress and Endocrine System**

Stimulation of pituitary-adrenal axis in stress increases secretion of corticotrophin releasing hormone (CRH) which increases secretion of adrenocorticotropin (ACTH), 8-lipotropin and 3-endorphin.

Increase in vasopressin secretion during stress potentiates the effect of CRH. Stress decreases levels of thyroid hormones through inhibition of thyroid-stimulating hormone secretion. Increase in the growth hormone secretion was reported followed by physical stress. GH responses for psychological stress were not well reported. Prolactin levels may increase or decrease during stress depending on local regulatory mechanisms. However, the role of altered prolactin levels is still unclear. Decrease in insulin secretion was observed in stress which may contribute for stress induced increase in blood glucose levels. 32-38

# **Stress and Reproductive System**

Though stress was reported to have negative impact on reproductive system, but the underlying mechanism was not clear. CRH and glucocorticoids exerts inhibitory effect on gonadotropin-releasing hormone (GnRH) neurons, pituitary gonadotrophs and gonads directly and indirectly. As a result, the production of steroid hormones will be decreased at gonads that lead to amenorrhea of stress in females and decreased libido and hypo-fertility in males.<sup>39,40</sup>

# **Stress and Epigenetic Changes**



Figure 5: Stress impact in DNA

Researchers found that chronic exposure to a stress hormone causes modifications to DNA in the brains of mice, prompting changes in gene expression. The new finding provides clues into how chronic stress might affect human behavior.

In the September 2010 issue of Endocrinology, the researchers reported that mice given corticosterone appeared more anxious during a maze test. Chronic exposure to corticosterone altered the expression of 3 HPA axis genes, including higher levels of Fkbp5 in the hippocampus, hypothalamus and blood. The protein that Fkbp5 codes for is part of a molecular complex that interacts with the glucocorticoid receptor. In addition, the researchers found decreased Fkbp5 methylation levels in the mice exposed to corticosterone.

Genetic variations in *Fkbp5* have been associated with posttraumatic stress disorder and mood disorders, which are characterized by abnormal glucocorticoid regulation. These results suggest that methylation of *Fkbp5* may play a role in mediating the effects of glucocorticoids on behavior.

"This gets at the mechanism through which we think epigenetics is important," says Potash. Epigenetic marks added to DNA through life experience may prepare an animal for future events, he explains. "If you think of the stress system as preparing you for fight or flight, you might imagine that these epigenetic changes might prepare you to fight harder or flee faster the next time you encounter something stressful."

With modern stressors, such as work deadlines, we can't fight or flee, and chronic stress may instead lead to depression or other mood disorders. Understanding the mechanism by which chronic stress leads to these conditions might help us find new ways to prevent or treat them in the future. This research suggests that epigenetic changes could play a role in the process. However, it's important to note that the connection is still speculative. Future studies will be needed to better understand the effects of chronic stress.<sup>41</sup>

#### Conclusion

Stress has both positive and negative impact in human body. Positive stress is beneficial for us as it helps to motivate and boosts our performance whereas chronic stress, over a long period of time can increases risk of cardiovascular, gastro, epigenetic variations etc. It is advisable to reduce stress in our daily life exercises, meditation, dance or pursuing one's hobby can be done.

# Acknowledgement: None

#### References

- 1. Chrousos GP. The concepts of stress and stress system disorders: overview of physical and behavioral homeostasis. *J Am Med Assn.* 1992;267(9):1244–1252.
- 2. Selye H. *The stress of life*. New York: McGraw-Hill; 1978.
- 3. Mason JW. A re-evaluation of the concept of non-specificity in stress theory. *J Psychiatr Res.* 1971;8(3):323–333.
- 4. Goldstein DS, Kopin IJ. Evolution of concepts of stress. *Stress*. 2007;10(2):109–120.
- 5. McEwen BS. Stress, adaptation, and disease. Allostasis and allostatic load. *Ann NY Acad Sci.* 1998;840:33–44.)
- 6. McEwen BS. Stress, adaptation, and disease. Allostasis and allostatic load. *Ann NY Acad Sci.* 1998;840:33–44.
- 7. Miller LH, Smith AD, Rothstein L. *The stress solution: an action plan to manage the stress in your life.* New York: Pocket Books; 1994.
- 8. McEwen BS. Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *Eur J Pharmacol*. 2008;58(2-3):174–185.
- 9. Tsigos C, Chrousos GP. Hypothalamic-pituitary-adrenal axis, neuroendocrine factors and stress. *J Psychosom Res.* 2002;53(4):865–871.
- 10. Vale S. Psychosocial stress and cardiovascular diseases. *Postgrad Med J.* 2005;81(957):429–435.
- 11. https://www.mhanational.org/conditions/stress
- 12. https://www.nami.org/About-NAMI/NAMI-News/2013/Stress-Boosts-Good-and-Bad-Habits-Study-Finds
- 13. <a href="https://psychcentral.com/blog/is-stress-good-for-you">https://psychcentral.com/blog/is-stress-good-for-you</a>
- 14. Hoffenberg R. Aetiology of hyperthyroidism-II. *BMJ*. 1974;3:508–10. [PMC free article]
- 15. Itoh N, Obata K, Yanaihara N, Okamoto H. Human preprovasoactive intestinal polypeptide contains a novel PHI-27-like peptide, PHM-27. *Nature*. 1983;304:547–9.
- 16. Sonino N, Girelli ME, Boscaro M, Fallo F, Busnardo B, Fava GA. Life events in the pathogenesis of Graves' disease: A controlled study. *Acta Endocrinol* (*Copenh*) 1993;28:293–6.
- 17. Kung AW. Life events, daily stresses and coping in patients with Graves' disease. Clin

- Endocrinol (Oxf) 1995;42:303-8.
- 18. Radosavljevi'c VR, Jankovic SM, Marinkovic JM. Stressful life events in the pathogenesis of Graves' disease. *Eur J Endocrinol*. 1996;134:699–701.
- 19. Yoshiuchi K, Kumano H, Nomura S, Yoshimura H, Ito K, Kanaji Y, et al. Stressful life events and smoking were associated with Graves' disease in women, but not in men. *Psychosom Med.* 1998;60:182–5.
- 20. Cielo CA, Christmann MK, Ribeiro VV, et al. Musculoskeletal stress syndrome, extrinsic laryngeal muscles and body posture: theoretical considerations. *Rev CEFAC*. 2014;16:1639–1649.
- 21. Qureshi F, Alam J, Khan MA, et al. Effect of examination stress on blood cell parameters of students in a Pakistani medical college. *J Ayub Med Coll Abbottabad*. 2002;14(1):20–22.
- 22. Grippo AJ, Johnson AK. Stress, depression and cardiovascular dysregulation: a review of neurobiological mechanisms and the integration of research from preclinical disease models. *Stress*. 2009;12(1):1–21.
- 23. Tamashiro KL, Sakai RR, Shively CA, et al. Chronic stress, metabolism, and metabolic syndrome. *Stress Amst Neth*. 2011;14(5):468–474.
- 24. Herman JP, Figueiredo H, Mueller NK, et al. Central mechanisms of stress integration: hierarchical circuitry controlling hypothalamo- pituitary-adrenocortical responsiveness. *Front Neuroendocrinol*. 2003;24(3):151–180.
- 25. Aguilera G, Subburaju S, Young S, et al. The parvocellular vasopressinergic system and responsiveness of the hypothalamic pituitary adrenal axis during chronic stress. Prog Brain Res. 2008;170:29–39.
- 26. Ranabir S, Reetu K. Stress and hormones. *Indian J Endocrinol Metab*. 2011;15(1):18–22.
- 27. Helmreich DL, Tylee D. Thyroid hormone regulation by stress and behavioral differences in adult male rats. *Horm Behav*. 2011;60(3):284–291.
- 28. Skuse D, Albanese A, Stanhope R, et al. A new stress-related syndrome of growth failure and hyperphagia in children, associated with reversibility of growth-hormone insufficiency. *Lancet*. 1996;348(9024):353–358.
- 29. Itoh N, Obata K, Yanaihara N, et al. Human preprovasoactive intestinal polypeptide contains a novel PHI-27-like peptide, PHM-27. *Nature*. 1983;304(5926):547–549.
- 30. Halter JB, Beard JC, Porte D. Islet function and stress hyperglycemia: plasma glucose and epinephrine interaction. *Am J Physiol*. 1984;247(1 Pt 1):E47–E52.
- 31. Traslavina GA, Franci CR. The CRH-R<sub>1</sub> receptor mediates luteinizing hormone, prolactin, corticosterone and progesterone secretion induced by restraint stress in estrogen-primed rats. *Brain Res.* 2011;1421:11–19.
- 32. Whirledge S, Cidlowski JA. Glucocorticoids, stress, and fertility. *Minerva Endocrinol*. 2010;35(2):109–125.
  - 33. Grippo AJ, Johnson AK. Stress, depression and cardiovascular dysregulation: a review of neurobiological mechanisms and the integration of research from preclinical disease

- models. Stress. 2009;12(1):1–21.
- 34. Tamashiro KL, Sakai RR, Shively CA, et al. Chronic stress, metabolism, and metabolic syndrome. *Stress Amst Neth*. 2011;14(5):468–474.
- 35. Herman JP, Figueiredo H, Mueller NK, et al. Central mechanisms of stress integration: hierarchical circuitry controlling hypothalamo- pituitary-adrenocortical responsiveness. *Front Neuroendocrinol*. 2003;24(3):151–180.
- 36. Aguilera G, Subburaju S, Young S, et al. The parvocellular vasopressinergic system and responsiveness of the hypothalamic pituitary adrenal axis during chronic stress. Prog Brain Res. 2008;170:29–39.
- 37. Walton, Kenneth G.; Levitsky, Debra K. (2003). "Effects of the Transcendental Meditation program on neuroendocrine abnormalities associated with aggression and crime". Journal of Offender Rehabilitation. 36 (1–4): 67–87. doi:10.1300/J076v36n01\_04. S2CID 144374302.
- 38. Black PH. Central nervous system-immune system interactions: Psychoneuroendocrinology of stress and its immune consequences. *Antimicrob Agents Chemother*. 1994;38:1–6. [PMC free article] [PubMed] [Google Scholar]
- 39. McGrady AV. Effects of psychological stress on male reproduction: a review. *Arch Androl.* 1984;131:1–10.
- 40. Palti Z. Psychogenic male infertility. *Psychosom Med.* 1969;31:326–330.
- 41. https://www.nih.gov/news-events/nih-research-matters/stress-hormone-causes-epigenetic-changes#:~:text=Researchers%20found%20that%20chronic%20exposure,prompting%20changes%20in%20gene%20expression