

Hormonal Changes During Acute Stress Response

Zainab Tawfeeq Al-Joubour¹, Iqbal AbdAlaziz Huzam AlRufaei², Irfan Abdulzahra Al- Khazaali³,
Mohammed Abdulrazzaq Assi⁴, Ali A. Al-fahham⁵

¹ Department of Pharmacology and Toxicology, Faculty of Pharmacy, University of Kufa, Najaf, Iraq,

² Biology Department, College of Science, University of Basrah, Iraq,

³ Department of Adult Nursing, College of Nursing, University of Al-Qadisiyah,

⁴ Department of Anesthesia Techniques, College of Health and Medical Techniques/ Baghdad, Middle Technical University, Iraq,

⁵ Faculty of nursing, University of Kufa, Iraq,

ABSTRACT: In today's environment, people constantly find themselves confronted with a wide range of upsetting situations. It is possible for stress to cause changes in the levels of a wide variety of hormones that are found in the serum, including glucocorticoids, catecholamines, growth hormone, and thyroid hormones. The "fight or flight" response requires certain adjustments to be made in order to ensure the individual's safety. There is a possibility that certain stress-related responses can lead to endocrine illnesses such as Graves' disease, gonadal dysfunction, psychosexual dwarfism, and obesity. Furthermore, stress has the potential to alter the clinical status of a wide variety of preexisting endocrine illnesses, such as contributing to the onset of adrenal crisis and thyroid storm. Significant consequences of stress include changes in behavior and systemic alterations, as well as changes in cellular mechanisms, hormonal , and brain stimulation. These changes can be observed at all levels, including cellular mechanisms, hormonal changes, and brain stimulation. An entirely new field of research has lately revealed the impacts of stress on human behavior, social cognition, and health. This is in spite of the fact that the influence of stress on health has been the subject of substantial research for a considerable amount of time.

INTRODUCTION

Stress is commonly referred to as "any circumstance that has the potential to disrupt the equilibrium between a living organism and its environment." Work burnout, test stress, psychological and social stress, and bodily stress as a result of operations, traumas, infections, and a variety of medical ailments are only some of the stressful events that we encounter on a daily basis. Our lives are characterized by a multitude of stressful conditions. Within the scope of this review, we will present a comprehensive outline of the hormonal changes that take place during the acute stress response and the influence that these changes have on the endocrine system. (Ranabir & Reetu, 2011).

TYPES OF STRESS

Stress can be classified into two broad types based on duration of the response:

1. Acute stress: This is the temporary tension that is frequently the consequence of immediate stressors or difficult circumstances. Temporary physiological modifications, including elevated heart rate and adrenaline release, are the result of the body's fight-or-flight response process.

2. Chronic stress: The occurrence of this phenomena occurs when the stressor continues to exist for a considerable amount of time. Chronic stress can have a cumulative effect on both the body and the mind, which can increase the risk of developing health problems such as anxiety, depression, and cardiovascular disease. This can be a consequence of prolonged exposure to chronic stress. (Godoy et al., 2018).

In the physiology of the stress reaction, there are two components: a delayed response that is mediated by the HPA axis, and a rapid response that is mediated by the SAM axis. Both of these responses are responsible for the stress response. (Chu et al., 2024).

SYMPATHETIC-ADRENO-MEDULLAR SYSTEM

The sympathetic-adreno-medullar system (SAM) is responsible for initiating a quick stress response, which leads to an increase in the release of adrenaline and noradrenaline from the adrenal medulla into the systemic blood, as well as an increase in the release of noradrenaline from the peripheral sympathetic nerves. This causes an increase in the amount of noradrenaline that is present in

Hormonal Changes During Acute Stress Response

the brain while the process is taking place. (Joëls & Baram, 2009). When epinephrine and norepinephrine are released into the body, they connect with α - and β -adrenergic receptors in the central nervous system. Additionally, they interact with the membranes of smooth muscle cells and various organs throughout the body. Upon this connection, a cascade is established, in which these neurotransmitters bind to specific G-protein receptors on cell membranes, thereby establishing an intracellular cyclic adenosine monophosphate (cAMP) signaling pathway. This cascade is responsible for the transmission of signals between cells. This pathway is responsible for the initiation of rapid cellular responses. The activation of these receptors causes smooth muscle cells and cardiac muscle cells to contract, which contributes to vasoconstriction, increased blood pressure, heart rate, cardiac output, skeletal muscle blood flow, increased sodium retention, elevated glucose levels through glycogenolysis and gluconeogenesis, lipolysis, increased oxygen consumption, and thermogenesis. These effects are all caused by the activation of these receptors. In addition, this physiological response causes bronchioles to enlarge, inhibits the motility of the digestive tract, and produces cutaneous vasoconstriction. Additionally, the activation of the sympathetic-adrenal-medullary (SAM) system causes behavioral changes, such as analgesia, increased arousal, alertness, vigilance, cognition, and focused attention. These changes are brought about by the combination of these three systems. (Chu et al., 2024).

HYPOTHALAMIC-PITUITARY-ADRENAL SYSTEM

It is the stimulation of the HPA axis that is responsible for the delayed reaction. This stimulation causes the flow of corticotropin-releasing hormone (CRH) from the hypothalamus into the bloodstream. It is the hypothalamus that is responsible for the secretion of the corticotropin-releasing hormone (CRH), which binds to two receptors, specifically CRH-R1 and CRH-R2. A number of different regions of the mammalian brain include CRH-R1, which is the primary receptor that is accountable for the release of adrenocorticotrophic hormone (ACTH) from the anterior pituitary gland during times of stress. This receptor is found in a number of different regions of the brain. On the other hand, CRH-R2 is primarily located in peripheral tissues, which include the skeletal muscles, the gastrointestinal system, the heart, and the subcortical regions of the brain. On top of that, CRH-binding protein, also known as CRH-BP, exhibits a higher affinity for binding to CRH in contrast to receptors. The placenta, liver, brain, and pituitary gland are only few of the organs that contain CRH-BP. Many other tissues also contain it. (Ketchesin et al., 2017).

Through its attachment to around forty percent to sixty percent of CRH in the brain, CRH-BP has been shown to play a significant role in the regulation of the presence of CRH, as revealed by research. Negative feedback is produced as a result of the steady rise in the creation of CRH-BP that occurs as a result of exposure to stress. This mechanism serves to reduce the interaction between CRH and CRH-R1 receptors, which in turn provides a negative feedback loop. One way to determine the total amount of cortisol present in the body is to measure the serum cortisol level. A little less than ten percent of cortisol is bound to albumin, while around eighty percent is bound to cortisol-binding globulin. The form of cortisol that is physiologically active is the unbound form that is still present. Once this has occurred, the CRH that has been created stimulates the anterior pituitary gland to release ACTH into the bloodstream. Glucocorticoid hormones, such as cortisol, are secreted into the bloodstream by the adrenal cortex as a result of the stimulation that is provided by ACTH. (Chu et al., 2024).

ACUTE STRESS

The acute stress reaction often manifests within a minimum of three days. It involves the activation of many brain regions that are important for regulating physical balance and mental processes, as well as adjusting behavior. This process entails the stimulation of limbic regions, such as the amygdala or the hippocampus, while concurrently inhibiting the functioning of the prefrontal cortex. (Datta and Arnsten, 2019). The second impact is governed by increased levels of catecholamines, which are noticeable even in mild, unmanageable stress situations and become more significant after extended exposure to stress. This leads to reduced synaptic connection in the prefrontal cortex (PFC) at the cellular level. The decrease in prefrontal cortex (PFC) function, commonly known as the "prefrontal brake," is linked to deficiencies in working memory. As a result, there is a change in the way the brain regulates the body's response to stress. This change involves a shift in activity from the prefrontal cortex to the amygdala, which intensifies the emotional and physiological reactions to stress. (Arnsten, 2009). Instances of acute stress encompass situations such as experiencing tension related to public speaking. (Al-fahham, 2019), restraint stress (Al-jawad and Al-Fahham, 2020), exam stress (Vedhara et al., 2000).

Blood flow to major muscles is directed, heart rate is elevated, heart muscle contractions are enhanced, and heart vessels are expanded by short-term stress. On the other hand, persistent stress induces persistent activation of the sympathetic nervous system and HPA axis, leading to elevated levels of stress hormones such as cortisol and epinephrine. (Hall et al., 2004). These stress hormones foster oxidative stress, endothelial dysfunction, and inflammation, thus fostering atherosclerosis development and impairing vascular function. Additionally, stress-induced changes in lipid metabolism worsen dyslipidemia, further increasing cardiovascular risk. (Yaribeygi et al., 2017).

Hormonal Changes During Acute Stress Response

HORMONAL CHANGES DURING ACUTE STRESS

Glucocorticoids, catecholamines, growth hormone, and prolactin are among the hormones that are secreted at a higher rate in response to stress, which results in fluctuations in hormone levels. The mobilization of energy sources and the individual's adaptation to their current situation are facilitated by these hormonal responses. (Ranabir & Reetu, 2011).

CORTISOL

One of the most important neuroendocrine responses to stress is the activation of the pituitary-adrenal axis, which is necessary for the continuation of daily living. Following the production of corticotrophin-releasing factor (CRF) by the hypothalamus, the pituitary gland is stimulated to release adrenocorticotropin (ACTH), 8-lipotropin, and 3-endorphin. This stimulus is responsible for the release of these hormones. Humans are susceptible to experiencing an increase in the levels of these hormones in their plasma that can range anywhere from two to five times higher during times of stress. A number of neurotransmitters, including norepinephrine, serotonin, and acetylcholine, play important roles in the process of boosting the production of corticotropin-releasing factor (CRF). The paraventricular nucleus of the hypothalamus is responsible for coordinating the complex stress response. (Chu et al., 2024).

In the minutes following the onset of stress, the adrenal cortex is responsible for the secretion of cortisol, which results in an increase in the levels of cortisol within the brain. When cortisol is able to get through the blood-brain barrier, it sets off a chain reaction of complicated effects in the brain and the rest of the body. These effects are brought about by both delayed genomic and quick non-genomic mechanisms. (de Kloet, 2014).

In the context of the cerebral level, the influence of glucocorticoids on the process of memory consolidation is an example of the interrelationship that exists between the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic-adrenal-medullary (SAM) system. By activating the glucocorticoid receptor (GR) in a manner that is dependent on the dosage, glucocorticoids contribute to the activation of the process of memory consolidation. The presence of adrenergic activity in the amygdala is another factor that might have an effect on this activation. (de Quervain et al., 2017).

The alarm reaction stage is characterized by the initial physiological manifestations that manifest themselves in response to acute stress, which is then followed by the fight-or-flight response. Immediately following the initial shock, the body initiates the process of self-repair by lowering the levels of the stress hormone cortisol and reestablishing normal physiological responses, such as the heart rate and blood pressure. While the body is in the process of healing, it maintains vigilance until the stressor is completely eliminated. The body, on the other hand, will modify in order to deal with increased levels of stress if the stress continues for an extended period of time. (Hall et al., 2004).

The surgical procedure induces physiological stress, leading to an increase in cortisol levels, which is positively associated with the severity of the surgery. According to the criteria of the POSSUM scale, cortisol levels in patients who have undergone major surgeries often return to their normal levels during the first 1 to 5 days after the operation. There is no relationship between the intensity of pain experienced after cardiac surgery and the levels of cortisol in the body. It is worth mentioning that the use of opiate pain relief after surgery does not have any impact on the cortisol stress response in patients who are undergoing light, moderate, or severe procedures. Individuals with hypoadrenalism requiring cortisol replacement during surgery face major implications due to the fluctuating amounts of cortisol secretion in response to surgical stress. (van Gulik et al., 2016).

Al-jawad and Al-Fahham (2020) found that restraint stress significantly increases cortisol hormone in rats, but the results did not appear until the twentieth day.

CATECHOLAMINES

The activation of the pituitary-adrenal axis causes the secretion of catecholamines to be stimulated, which results in an increase in cardiac output, an improvement in blood circulation to skeletal muscles, the retention of sodium, a reduction in movement in the intestines, a constriction of blood vessels in the skin, an increase in glucose levels, a dilation of the bronchioles, and an increase in behavioral arousal. According to the findings of a previous study, the adrenosympathetic system is activated more frequently in response to high levels of occupational stress. (Yaribeygi et al., 2017).

During periods of stress, catecholamines like epinephrine and norepinephrine have a significant impact on the gastrointestinal system. These hormones interact with adrenergic receptors present throughout the gastrointestinal tract, affecting multiple physiological functions. By stimulating α -adrenergic receptors in the smooth muscle of the intestines, they lead to delayed gastric emptying and decreased intestinal motility. Activation of α -adrenergic receptors also induces vasoconstriction in the gastrointestinal vasculature, reducing blood flow to the gut and consequently inhibiting gastrointestinal secretions and nutrient absorption (Ranabir & Reetu, 2011).

During periods of stress, the body adjusts the distribution of blood volume in order to protect the brain's blood supply. This process is influenced by the amounts of norepinephrine and epinephrine in the plasma, which play a crucial role. The sympathetic nervous system is activated in reaction to many physiological dangers, such as low blood sugar, severe bleeding, intense exertion beyond

Hormonal Changes During Acute Stress Response

the point where oxygen is sufficient, and suffocation. Moreover, epinephrine is associated with behaviors such as proactive avoidance, hostility, and anxiety. (van Gulik et al., 2016).

An experience of a stressful scenario, whether it is driven by environmental elements or psychological stressors, is the beginning of a chain of events that involves stress hormones that induce physiological abnormalities. This chain of events is launched by the experience of a stressful circumstance. An acute stress reaction, often known as the fight-or-flight response, is triggered when the sympathetic nervous system is activated. This response gives humans the ability to either confront the threat or run away from the circumstance. The adrenal medulla is responsible for the release of adrenaline and noradrenaline, which causes the sympathetic nervous system to become activated throughout the body. This activation is extensive and affects nearly all parts of the sympathetic nervous system. This extensive activation causes physiological changes, such as an increase in arterial pressure, an increase in blood flow to active muscles, a decrease in blood flow to organs that are not essential for rapid movement, an acceleration in blood coagulation rates, an increase in cellular metabolism rates throughout the body, an increase in muscle strength, an increase in mental alertness, an increase in blood glucose levels, and an increase in glycolysis in the liver and muscles. These effects, when taken as a whole, motivate individuals to engage in activities that are more physically demanding than they would otherwise do. When the perceived threat is no longer present, the body returns to the state it was in before the threat occurred. (Yaribeygi et al., 2017).

VASOPRESSIN

Vasopressin and corticotropin-releasing hormone (CRH) are both released in a fast manner by the paraventricular nucleus of the hypothalamus when the hypothalamus is subjected to extreme stress. Vasopressin has the ability to improve the release of adrenocorticotropic hormone (ACTH) from the pituitary gland when it binds to the V1b receptor. This results in an increase in the intensity of the effects that CRH possesses. Vasopressin is often secreted at a higher rate than corticotrophic hormone (CRH) in situations where the hypothalamus is exposed to extended stress and displays heightened sensitivity to corticotrophs. (Ranabir & Reetu, 2011). Jeong et al. (2020) The levels of salivary vasopressin in dogs are investigated in this study after they have been subjected to a brief period of acute stress, namely thirty minutes of exposure to the sounds of a vacuum cleaner. According to the findings, it appears that.

THYROID HORMONES

During times of stress, thyroid function usually decreases. Both the levels of T3 and T4 decrease as a consequence of stress. Glucocorticoids in the central nervous system hinder the release of thyroid-stimulating hormone (TSH), leading to a decrease in its secretion. This effect is caused by stress. Initial evidence demonstrated rapid changes in brain T3 levels in response to acute stress. (Friedman et al., 1999). Fischer et al. (2019) Researchers have examined whether being exposed to psychosocial stress triggers an instantaneous activation of the hypothalamic-pituitary-thyroid (HPT) axis. It was discovered that there was a notable increase in TSH levels in response to the TSST. The highest point was noted 20 minutes after the start of the stressor, followed by a gradual decrease.

GROWTH HORMONE

The level of growth hormone (GH) is elevated during acute physical duress. Increases in the level may range from two to tenfold. As a result of its insulin-antagonistic effect, GH may increase metabolic activity. On the other hand, GH responses are seldom observed in psychological duress. (Ranabir & Reetu, 2011). Rather, there is a GH secretory defect that is accompanied by protracted psychosocial stress. Jezova et al. (2007) The study examined the GH responses to several stressful scenarios, including aerobic exercise, hypoglycemia, and hyperthermia. These situations were applied in two consecutive sessions with a time gap of 80-150 minutes. Following the initial exercise session, there was an increase in the production of growth hormone at all time intervals. The hormone levels remained elevated even after 80 minutes, when the second exercise began. No increases in growth hormone levels beyond the levels before exercising were observed in response to the second exercise test.

PROLACTIN

During instances of stress, the level of prolactin might vary, either increasing or decreasing according on the unique regulatory factors in the local environment. Vasopressin and peptide histidine isoleucine are potentially involved in the regulation of prolactin release during periods of stress. Nevertheless, the precise evolutionary significance of these alterations in prolactin levels remains ambiguous. Fluctuations of this nature have the potential to affect the immune system or specific components involved in maintaining internal equilibrium, often known as homeostasis. (Ranabir & Reetu, 2011). However, Lennartsson et al., (2011) In addition to investigating the possibility of differences between the sexes, the purpose of this study was to investigate the effect that acute psychosocial stress has on the levels of prolactin that are found in the blood of healthy men and women simultaneously. The results of the study demonstrated that an increase in prolactin levels is, in fact, a response to the presence of psychological stress. On the other hand, it is essential to point out that the degree of this reaction varied significantly from person to person when compared

Hormonal Changes During Acute Stress Response

to the whole population. It has been observed that there is no distinction between the pattern of prolactin response between males and females. On the other hand, there were indicators that suggested that women might have a higher degree of growth in comparison to men.

INSULIN

The level of insulin may decrease during periods of tension. This, in conjunction with an increase in its antagonistic hormones, may contribute to hyperglycemia induced by stress. (Mifsud et al., 2018). However, Romeo et al. (2007) I conducted an investigation into restraint stress in male rodents and discovered that insulin and thyroid hormone levels were not influenced by acute stress at either the age or time of day.

TESTOSTERONE

Zueger et al. (2013) The purpose of this study was to evaluate the differences in cortisol and testosterone levels that occurred among the same individuals throughout different types of stress and for different amounts of time. In addition, they explored the influence that baseline levels have on the reactions of hormones to stressed situations. According to what they found, there were significant increases in cortisol and testosterone levels during the situation of acute tension as well as the field exercise. The baseline testosterone levels were found to have a negative correlation with the acute cortisol response during the field exercise; however, this correlation was not observed during the acute stress phase of the experimental study.

Guo et al. (2018) In this study, both male and female rats were subjected to gonadectomy, which results in a reduction in the levels of sex hormones (estrogens and androgens). The researchers were interested in determining how the gonadectomy affected the acute and chronic stress responses of the rats. In this study, variations in plasma and hypothalamic sex hormones, as well as chemicals related to stress in the hypothalamus, are investigated. The research shows that decreased levels of sex hormones in the periphery lead to specific changes in the stress response systems of both the peripheral and central nervous systems in male and female rats. According to these findings, it is essential to select appropriate models for the research of various forms of mood disorders, taking into consideration the differences that have been discovered between the sexes.

On the other hand, Al-amery and Al-Fahham (2020) It was discovered that the application of restraint stress in rats led to a considerable fall in testosterone hormone levels. However, this effect was not observed until the twentieth rat.

The regular menstrual cycle is disrupted as a result of stress because it causes a drop in the quantities of gonadotropins and gonadal steroid hormones that are carried through the bloodstream. Stress that is experienced for an extended period of time might result in a complete impairment of reproductive functioning. There is a decrease in the stimulation of the pituitary gland by gonadotropin-releasing hormone (GnRH), which is most likely due to an increase in the production of corticotropin-releasing hormone (CRH) that is produced naturally by the body. (Al-fahham, A. and Al-Nowain, 2016).

CONCLUSIONS

Stress response is triggered by neuro-hormonal stimulation including catecholamines and cortisol. Some hormones such as GH and testosterone change in response to stress consequences.

REFERENCES

- 1) Alaa K. M. Al-amerya, Ali A. Al-Fahham (2020) . The Synergistic Effect of Ascorbic Acid and Selenium on Serum Testosterone in Restraint Stressed Rats . *Indian journal of forensic medicine and toxicology*,14(4) : 991-996.
- 2) Al-fahham, A. and Al-Nowainy, H. Q. (2016) The role of FSH, LH, and Prolactin Hormones in Female Infertility . 6(5) :110-118.
- 3) Al-Fahham, A.A. (2019) Effect of low dose vitamin C on public speaking stress during group presentation. *IOP Conf. Series: Journal of Physics: Conf. Series* 1294, doi:10.1088/1742-6596/1294/6/062054.
- 4) Chu, B., Marwaha, K., Sanvictores, T., Awosika, A. O., & Ayers, D. (2024). *Physiology, Stress Reaction*. In StatPearls. StatPearls Publishing.
- 5) Fischer, S., Strahler, J., Markert, C., Skoluda, N., Doerr, J. M., Kappert, M., & Nater, U. M. (2019). Effects of acute psychosocial stress on the hypothalamic-pituitary-thyroid (HPT) axis in healthy women. *Psychoneuroendocrinology*, 110, 104438. <https://doi.org/10.1016/j.psyneuen.2019.104438>
- 6) Friedman, Y., Bacchus, R., Raymond, R., Joffe, R. T., & Nobrega, J. N. (1999). Acute stress increases thyroid hormone levels in rat brain. *Biological psychiatry*, 45(2), 234–237. [https://doi.org/10.1016/s0006-3223\(98\)00054-7](https://doi.org/10.1016/s0006-3223(98)00054-7)
- 7) Godoy, L. D., Rossignoli, M. T., Delfino-Pereira, P., Garcia-Cairasco, N., & de Lima Umeoka, E. H. (2018). A Comprehensive Overview on Stress Neurobiology: Basic Concepts and Clinical Implications. *Frontiers in behavioral neuroscience*, 12, 127. <https://doi.org/10.3389/fnbeh.2018.00127>

Hormonal Changes During Acute Stress Response

- 8) Guo, L., Chen, Y. X., Hu, Y. T., Wu, X. Y., He, Y., Wu, J. L., Huang, M. L., Mason, M., & Bao, A. M. (2018). Sex hormones affect acute and chronic stress responses in sexually dimorphic patterns: Consequences for depression models. *Psychoneuroendocrinology*, 95, 34–42. <https://doi.org/10.1016/j.psyneuen.2018.05.016>
- 9) Hall, M., Vasko, R., Buysse, D., Ombao, H., Chen, Q., Cashmere, J. D., Kupfer, D., & Thayer, J. F. (2004). Acute stress affects heart rate variability during sleep. *Psychosomatic medicine*, 66(1), 56–62. <https://doi.org/10.1097/01.psy.0000106884.58744.09>
- 10) Jeong, Y. K., Oh, Y. I., Song, K. H., & Seo, K. W. (2020). Evaluation of salivary vasopressin as an acute stress biomarker in healthy dogs with stress due to noise and environmental challenges. *BMC veterinary research*, 16(1), 331. <https://doi.org/10.1186/s12917-020-02555-5>
- 11) Jezova, D., Radikova, Z., & Vigas, M. (2007). Growth hormone response to different consecutive stress stimuli in healthy men: is there any difference?. *Stress (Amsterdam, Netherlands)*, 10(2), 205–211. <https://doi.org/10.1080/10253890701292168>
- 12) Joëls, M., & Baram, T. Z. (2009). The neuro-symphony of stress. *Nature reviews. Neuroscience*, 10(6), 459–466. <https://doi.org/10.1038/nrn2632>
- 13) Ketchesin, K. D., Stinnett, G. S., & Seasholtz, A. F. (2017). Corticotropin-releasing hormone-binding protein and stress: from invertebrates to humans. *Stress (Amsterdam, Netherlands)*, 20(5), 449–464. <https://doi.org/10.1080/10253890.2017.1322575>
- 14) Lennartsson, A. K., & Jonsdottir, I. H. (2011). Prolactin in response to acute psychosocial stress in healthy men and women. *Psychoneuroendocrinology*, 36(10), 1530–1539. <https://doi.org/10.1016/j.psyneuen.2011.04.007>
- 15) Mifsud, S., Schembri, E. L., & Gruppetta, M. (2018). Stress-induced hyperglycaemia. *British journal of hospital medicine (London, England : 2005)*, 79(11), 634–639. <https://doi.org/10.12968/hmed.2018.79.11.634>
- 16) Ranabir, S., & Reetu, K. (2011). Stress and hormones. *Indian journal of endocrinology and metabolism*, 15(1), 18–22. <https://doi.org/10.4103/2230-8210.77573>
- 17) Romeo, R. D., Karatsoreos, I. N., Ali, F. S., & McEwen, B. S. (2007). The effects of acute stress and pubertal development on metabolic hormones in the rat. *Stress (Amsterdam, Netherlands)*, 10(1), 101–106. <https://doi.org/10.1080/10253890701204270>
- 18) van Gulik, L., Ahlers, S., van Dijk, M., Bruins, P., Meima, M. E., de Rijke, Y. B., Biemond-Moeniralam, H. S., Tibboel, D., & Knibbe, C. A. (2016). Procedural pain does not raise plasma levels of cortisol or catecholamines in adult intensive care patients after cardiac surgery. *Anaesthesia and intensive care*, 44(1), 52–56. <https://doi.org/10.1177/0310057X1604400109>
- 19) Vedhara, K., Hyde, J., Gilchrist, I. D., Tytherleigh, M., & Plummer, S. (2000). Acute stress, memory, attention and cortisol. *Psychoneuroendocrinology*, 25(6), 535–549. [https://doi.org/10.1016/s0306-4530\(00\)00008-1](https://doi.org/10.1016/s0306-4530(00)00008-1)
- 20) von Dawans, B., Strojny, J., & Domes, G. (2021). The effects of acute stress and stress hormones on social cognition and behavior: Current state of research and future directions. *Neuroscience and biobehavioral reviews*, 121, 75–88. <https://doi.org/10.1016/j.neubiorev.2020.11.026>
- 21) Yaribeygi, H., Panahi, Y., Sahraei, H., Johnston, T. P., & Sahebkar, A. (2017). The impact of stress on body function: A review. *EXCLI journal*, 16, 1057–1072. <https://doi.org/10.17179/excli2017-480>
- 22) Zahraa Abdel Al-Jawad, Ali A. Al-Fahham (2020) . The Effect of Ascorbic Acid and Selenium intake on serum Cortisol in rats Under Restraint Stress . *Indian journal of forensic medicine and toxicology*, 14(4) : 2454-2459 .
- 23) Zueger, R., Annen, H., & Ehlert, U. (2023). Testosterone and cortisol responses to acute and prolonged stress during officer training school. *Stress (Amsterdam, Netherlands)*, 26(1), 2199886. <https://doi.org/10.1080/10253890.2023.2199886>