Original article

IRSTI: 76.29.51 https://doi.org/10.70264/jbr.v1.3.2025.3

EFFECTS OF CHRONIC STRESS ON ANTIOXIDANT DEFENSE SYSTEM AND LIVER FUNCTION IN MALE ALBINO RATS

Owolabi Dorcas Olasimbo[1,*], Thomas Emmanuel Olusegun[2], Fawumi Daniel Oluwole[3]

[1]*Department of Biology, University of Ilesa, Ilesa, Osun State, Nigeria.*

[2]*Department of Pure and Applied Zoology, Federal University of Agriculture, Abeokuta, Ogun State, Nigeria.*

[3]*Department of Biological Sciences, Olusegun Agagu University of Science and Technology, Okitipupa, Ondo State, Nigeria.*

*Corresponding Author: Owolabi D.O., dorcas_owolabi@unilesa.edu.ng.

ABSTRACT

Stress is becoming prevalent in our world today, its effects on physiological processes is a major concern. This research investigates the effects of chronic stress on the antioxidant defense system and liver function in male albino rats. Eighteen male albino rats were used for the study. They were divided into three groups: control group, group exposed to force swimming, and group subjected to social isolation. Following a four-week stress protocol, the rats' liver enzymes and antioxidant levels were determined using spectrophotometric assays. It was revealed from the findings of this work that rats exposed to swimming stress showed significant increases in liver enzymes, including alanine aminotransferase (ALT) levels (54.46 ±0.46 U/L) compared to controls (26.79 ±1.20 U/L), and aspartate aminotransferase (AST) levels (71.30±1.30 U/L) compared to controls (22.17 ±0.17 U/L). Additionally, antioxidant enzymes like superoxide dismutase (SOD) increased to 197.14 ±2.14 units with swimming stress compared to controls (65.71±0.70 units), and catalase activity rose to 48.29±0.29 kU compared to controls (29.78±0.08 kU). The findings suggest that chronic stress can lead to liver damage and oxidative stress, with swimming stress having a greater impact than isolation stress. These results have implications for understanding the impact of chronic stress on liver function and overall health.

Keywords: *chronic stress, antioxidant, liver, oxidative, responses.*

Received: June 17, 2025 / *Accepted*: September 20, 2025 / *Published*: September 22, 2025

Citation: Owolabi D.O., Thomas E.O., Fawumi D.O.(2025). Effects of chronic stress on antioxidant defense system and liver function in male albino rats. Journal of Biological Research, 1(3), 21-27. https://doi.org/10.70264/jbr.v1.3.2025.3.

1 INTRODUCTION

Stress can be described as any situation that disrupts the balance between an organism and its environment, triggering physiological and psychological responses [1]. Stress impacts multiple system of the body, including musculoskeletal, respiratory, cardiovascular, endocrine, gastrointestinal, nervous and reproductive.system. This comprehensive effect highlights the far-reaching consequences of stress on overall health and well-being [2]. While our bodies can manage short-term stress, prolonged or chronic stress can have severe and lasting impacts on physical and mental health.

Chronic stress exposure is linked to increased oxidative stress, inflammation, and potential organ damage [3]. Chronic stress can lead to various health issues, including cardiovascular disease, impaired immune function, gastrointestinal problems, metabolic disorders, anxiety, depression, mood disorders, and cognitive impairment [4]. Additionally, chronic stress induces oxidative stress, prompting the activation of antioxidant enzymes such as superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase to protect against cellular damage [5]. Recent studies have demonstrated the impact of chronic stress on enzymatic and oxidative activity. For example, a study by Corona-Pérez et al. [6], found that chronic restraint stress increased oxidative stress and inflammation in the liver, leading to increased levels of liver enzymes like ALT and AST. Similarly, a study by Stojiljković et al. [7], found that chronic stress induced oxidative stress and altered antioxidant enzyme activity in the brain and liver of rats.

Various animal models have been used to study the effects of chronic stress on enzymatic and oxidative activity. For example, studies have used rat models of chronic restraint stress [6], forced swim stress [7], and social isolation stress [8] to investigate the impact of chronic stress on antioxidant defenses and liver function.

Specific enzymes that serve as biochemical markers of stress responses include several key antioxidant and liver enzymes. Superoxide dismutase (SOD) is a crucial antioxidant enzyme that catalyzes the conversion of superoxide anions into hydrogen peroxide [5]. Glutathione peroxidase (GPx) reduces hydrogen peroxide to water and lipid hydroperoxides to their corresponding alcohols [9]. Catalase further breaks down hydrogen peroxide into water and oxygen, thereby preventing oxidative damage [5]. In addition, liver enzymes such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are commonly released into the bloodstream in response to liver damage, serving as important indicators of hepatic stress [10].

Understanding how stress affects antioxidant defenses and liver enzyme activity can inform strategies for the prevention and management of stress-related diseases, ultimately contributing to improved health outcomes. Therefore, this study investigates the impact of chronic stress on antioxidant defense mechanisms and liver function in male albino rats.

2 MATERIALS AND METHODS

2.1 Study Area. The experiment was conducted in the Biological Science laboratory at Olusegun Agagu University of Science and Technology, Okitipupa. Okitipupa is situated between latitude 6°33′ N and longitude 4°43′ E, characterized by savannah vegetation and a climate marked by intense rainfall from April to October and daily temperatures ranging from 23°C to 37°C. The study took place from June to August 2023.

2.2 Experimental Animals. Eighteen male albino rats (*Rattus norvegicus albinus*) weighing 100-150 grams, were sourced from a reputable supplier for this study. The rats were acclimatized in two weeks. This was done to facilitate a better adaptation of these animals to their new environment. They were weighed with an electronic digital weighing balance (SF-400) and were individually housed in standard laboratory cages under controlled conditions (temperature: 22°C ± 2°C, humidity: 50-60%, 12-hour light-dark cycle) at the Biological Sciences Laboratory, Olusegun Agagu University of Science and Technology (OAUSTECH), Okitipupa, following university approval. The rats were fed with rat chow and given adequate water throughout acclimatization and afterward.

2.3 Experimental Design. Rats were randomly assigned into three groups of six members based on the stress protocol,

GROUP A: CONTROL: No stress protocol

GROUP B: Rats exposed to Forced Swim Test

GROUP C: Rats exposed to Social Isolation

The stress groups underwent a four-week chronic stress protocol, whereas the control group remained under standard, stress-free conditions.

2.3.1 Stress Induction Protocols.

2.3.1.1 Forced Swim Test. This test was conducted by placing rats in a water tank with specific dimensions (30-40 cm diameter, 50-60 cm height) filled with water at 25°C ± 1°C to a depth of 30-40 cm, and forcing them to swim for a specified duration, typically 10 minutes daily, while observing and scoring their behavior, particularly immobility time, with careful consideration of rat handling, environmental factors (e.g. light, temperature), and test duration and frequency [11].

2.3.1.2 Social Isolation. Rats were isolated in individual cages for 12 hours each day, during the dark phase to prevent physical interaction with other rats, while maintaining standard laboratory conditions with controlled temperature, humidity, and lighting. Cages were cleaned regularly as routine monitoring of the rats' behaviour, appetite, and physical condition were observed [12].

2.3.2 Stressor Randomization. Stressors were randomly applied at varying times of day to avoid habituation and prevent rats from adapting to a predictable stress pattern. The stress groups underwent a four-week chronic stress protocol, designed to simulate prolonged stress exposure. Stressors were randomly assigned to each rat using a randomization schedule generated prior to the start of the experiment. Stressors were applied at varying times of day, including morning (8:00 AM - 12:00 PM), afternoon (12:00 PM - 4:00 PM), and evening (4:00 PM - 8:00 PM).

The stressors used included forced swimming, social isolation, and restraint stress, applied in a randomized manner to prevent predictability.

The Four-Week Protocol was as follows:

Stressors were applied 3-4 times per week, with each stressor lasting 10-60 minutes.

Week 1: Forced swimming (10 minutes, 3 times), social isolation (12 hours, 2 times);

Week 2: Restraint stress (30 minutes, 3 times), forced swimming (10 minutes, 2 times);

Week 3: Social isolation (24 hours, 1 time), restraint stress (45 minutes, 2 times);

Week 4: Forced swimming (20 minutes, 2 times), social isolation (12 hours, 2 times);

The duration and intensity of stressors were adjusted over time to maintain chronic stress exposure. Experimental procedures were consistent across all rats and stressors to minimize variability.

2.4 Determinationof Liver Enzymes. Twenty-eight days following the stress protocol, the rats were administered anesthesia using a combination of ketamine (5-10 mg) and xylazine (0.625-1.25 mg) via intraperitoneal injection, sourced from local veterinary supplier (Ceva Animal Health, Nigeria), and subsequently sacrificed for further examination and analysis. Blood samples were collected from each group (3 rats/group) through ocular puncture into an anticoagulant tube (EDTA bottle) to prevent coagulation. Spectrophotometric analysis was performed to determine: ALT and AST levels [13] and ALP levels [14].

2.5 Determination of Antioxidant Enzymes. Antioxidant enzymes were determined using spectrophotometric assays:

2.5.1 SOD Acrivity. SOD activity was measured by inhibiting pyrogallol autoxidation [15], one unit of SOD activity is typically defined as the amount of enzyme required to inhibit 50% of pyrogallol auto-oxidation. Tissue homogenates of the rats' liver were prepared in a phosphate buffer. The homogenate was added to a reaction mixture containing pyrogallol and the change in absorbance were measured at 420 nm using a spectrophotometer.

2.5.2 CAT Activity. CAT activity is measured by monitoring H_2O_2 decomposition [16], one unit of CAT activity is typically defined as the amount of enzyme required to decompose 1 μmol of H_2O_2 per minute. Tissue homogenates of the rats' liver were prepared in a suitable buffer. The homogenate was added to a reaction mixture containing H_2O_2 and the decrease in absorbance at 240 nm were measured using a spectrophotometer.

2.5.3 GPx Activity. GPx activity is measured by monitoring NADPH oxidation in the presence of glutathione reductase, which is coupled to the reduction of oxidized glutathione (GSSG) formed by GPx activity [17]. One unit of GPx activity is typically defined as the amount of enzyme required to oxidize 1 nmol of NADPH per minute. Tissue homogenates of the rat's liver were prepared in a suitable buffer. The homogenate was added to a reaction mixture containing glutathione, glutathione reductase, and NADPH. The decrease in absorbance was measured at 340 nm due to NADPH oxidation using a spectrophotometer.

2.5.4 GSH Activity. Glutathione (GSH) levels were measured using DTNB (5,5'-dithiobis-(2-nitrobenzoic acid)) [18]. Tissue homogenates or cell lysates were prepared. DTNB was added to the sample, and the reaction was initiated. DTNB reacts with thiol groups (-SH) of GSH, forming a yellow-colored compound (TNB) that was measured spectrophotometrically at 412nm.

2.6 Statistical Analysis. Data were analyzed using Microsoft Excel 2019. Descriptive statistics (means ± standard deviations) were calculated, and inferential statistics (t-tests and ANOVA) were used to compare control and stress groups, with $p < 0.05$ considered statistically significant.

3. RESULTS

3.1 Liver enzymes

Variations observed in the activities of liver enzymes (ALT, ALP and AST) in stressed rats are shown in Table 1.

Table 1 – Effects of Stress on Liver Enzymes in Male Albino Rats (^{abc}Mean (±Standard deviation) in the same column having similar superscript are not significantly different ($p > 0.05$).

	ALT U/L	AST U/L	ALP mm/min
Control Male	26.79±1.20^{b}	22.17±0.17^{c}	145.65±1.98^{b}
Swimming Male	54.46±0.46^{a}	71.30±1.30^{a}	153.07±0.97^{a}
Isolated Male	20.76±0.06^{c}	60.84±1.17^{b}	130.00±3.00^{c}

The levels of these liver enzymes were significantly ($p < 0.05$) higher in the swimming male rats. On the other hand, activity of AST was significantly lowest in the control male rats while activities of ALT and ALP were significantly lowest in the isolated male rats.

3.2 Antioxidant Enzymes.

There was no significant difference ($p > 0.05$) recorded in the activity of glutathione peroxidase recorded in the control male rats and stressed male rats (Table 2).

On the other hand, reduced glutathione, cellulase and superoxide dismutase were significantly lowest in the control male rats than those of the stressed male rats. However, these (reduced glutathione, cellulase and superoxide dismutase) were highest in the swimming male rats.

3.3 Relationship between liver enzymes and antioxidant enzymes.

Very strong significantly ($p < 0.01$) positive correlation was recorded between ALT and activities of catalase ($r = 0.917$) and Superoxide dismutase ($r = 0.969$) (Table 3).

Similarly, the correlation between AST and reduced glutathione, catalase and superoxide dismutase were positively strong and significant. Also, the correlation between ALP and superoxide dismutase was positive and significant ($r = 0.682$, $p < 0.05$).

4 DISCUSSION

The body respond to stress by activating the hypothalamic-pituitary-adrenal (HPA) axis, releasing cortisol and adrenaline, which can impact liver function by increasing gluconeogenesis and altering liver enzyme activity, potentially leading to liver damage [19]. Additionally, chronic stress can deplete antioxidants like glutathione and impair antioxidant enzyme activity, increasing oxidative stress and contributing to cellular damage and disease [20].

The liver plays a vital role in maintaining overall health and function. Liver enzymes ALT, AST, and ALP play crucial roles in liver function, amino acid metabolism, and bone mineralization, with elevated levels potentially indicating liver

Table 2 – Effects of Stress on Antioxidant Enzymes in Male Albino Rats (^{abc}Mean (±Standard deviation) in the same column having similar superscript are not significantly different ($p > 0.05$)

	GPx	GSH	Catalase	SOD
Control Male	242.50±1.80^{a}	12.00±1.00^{b}	29.78±0.08^{c}	65.71±0.70^{c}
Swimming Male	243.71±3.01^{a}	16.50±0.50^{a}	48.29±0.29^{a}	197.14±2.14^{a}
Isolated Male	241.37±1.37^{a}	15.75±0.05^{a}	34.34±0.34^{b}	77.14±1.14^{b}

Table 3 – Correlation between liver enzymes and antioxidant enzymes in stressed rats (**Correlation significant at $p < 0.01$; *Correlation significant at $p < 0.05$

	ALT	AST	ALP	GPx	GSH	Catalase	SOD
ALT	1						
AST	0.529	1					
ALP	0.832**	0.002	1				
GPx	0.463	0.096	0.515	1			
GSH	0.483	0.963**	-0.056	0.192	1		
Catalase	0.917**	0.822**	0.563	0.344	0.767*	1	
SOD	0.969**	0.720*	0.682*	0.411	0.668*	0.987**	1

damage, heart attack, muscle injury, or bone disorders [21]. Stress can exacerbate liver conditions as natural killer cells in the liver become overactive, targeting and damaging liver cells [22].

Antioxidant enzymes provide crucial defense against free radicals. The key enzymes are superoxide dismutate (SOD) which neutralizes superoxide radicals, Catalase breaks down hydrogen peroxide and glutathione peroxidase that converts hydroperoxides to harmless molecules (water/alcohol and oxygen). These enzymes serve as the first line of defense, protecting cells from oxidative damage [23].

In this study, changes in liver enzymes and antioxidant enzymes suggest that stress, particularly swimming stress, can Induce liver damage or dysfunction (elevated ALT and AST) and can also activate antioxidant defense mechanisms (increased SOD, catalase, and GSH).

Stress exposure, particularly swimming stress, altered enzymatic activities in male rats. Liver enzymes (ALT and AST) increased significantly, suggesting potential liver damage. Antioxidant enzymes (SOD, catalase, and GSH) also increased, indicating activation of defense mechanisms. Swimming stress had a more pronounced effect than isolation stress, highlighting distinct physiological adaptations to different stressors. The differences between swimming and isolation stress responses may indicate distinct stress-induced physiological adaptations. These findings suggest that swimming and isolation stress may have different effects on liver function.

Other studies have reported similar results that chronic stress induced liver damage and increased liver enzymes in rats according to Jiang et al. [24]. Also, Akhtar et al. [25] observed that forced swimming stress increased oxidative stress and liver damage in mice. Chronic stress altered antioxidant enzyme activity and increased liver damage in rats as reported by Stojiljković et al. [7]. Antioxidant enzymes activity has been reported to increase in response to stress. Geddie et al. [26] stated that SOD and catalase activity increased in response to chronic stress in rats while the experiment conducted by de Haan et al. [27], revealed that GPx activity increased in response to oxidative stress in mice.

5 CONCLUSION

This work has in a way shed light on the consequences of stress on the physiology of animals. The results can show the impact on health and can thus guide stakeholders in effective stress management strategies. It concludes that chronic stress, especially those generated from forced swimming, can cause liver damage and trigger antioxidant defenses in rats. The findings emphasize the importance of considering the type and duration of stress in understanding its impact on health.

AUTHOR CONTRIBUTIONS

Conceptualization, O.D.O.; methodology, O.D.O. and T.E.O.; validation, O.D.O. and F.D.O.; formal analysis, O.D.O. and T.E.O.; investigation, O.D.O.; resources, O.D.O.; data curation, O.D.O. and F.D.O.; writing-original draft preparation, O.D.O.; writing-review and editing, O.D.O.; visualization, O.D.O. and T.E.O.; project administration, O.D.O.; funding acquisition, O.D.O. All authors have read and agreed to the publication of the final version of the manuscript.

FUNDING

N/A

CONFLICT OF INTEREST

There are no conflicts of interest to declare.

LITERATURE

1. Ranabir S., Reetu K. Stress and hormones // Indian Journal of Endocrinology and Metabolism. – 2011. – Vol. 15(3). – P. 193-199. https://doi.org/10.4103/2230-8210.77573

2. Publication manual of the American Psychological Association (7th ed.), 2020. Available online: https://jems.davnepal.edu.np/storage/files/shares/Online%20Resources/American%20Psychological%20Association%20-%20Publication%20Manual%20of%20the%20American%20Psychological%20Association,%20Seventh%20Edition%20(2020)%20[with%20PDF%20bookmarks]-American%20Psychological%20Association%20(2020).pdf (accessed on 10 June 2025)

3. Kabat-Zinn J. Mindfulness-based interventions in context: Past, present, and future. Clinical Psychology // Science and Practice. – 2003. – Vol. 10(2). – P. 144-156. https://doi.org/10.1093/clipsy.bpg016

4. Mayer E.A., Ryu, H.J., Bhatt, R.R. The neurobiology of irritable bowel syndrome // Molecular psychiatry. – 2023. – Vol. 28(4). – P. 1451–1465. https://doi.org/10.1038/s41380-023-01972-w

5. Sies H. Oxidative stress: Concept and some practical aspects // Antioxidants. – 2020. – Vol. 9(9). – P. 852. https://doi.org/10.3390/antiox9090852

6. Corona-Pérez A., Díaz-Muñoz M., Cuevas-Romero E., Luna-Moreno D., Valente-Godínez H., Vázquez-Martínez O., Martínez-Gómez M., Rodríguez-Antolín J., Nicolás-Toledo L. Interactive effects of chronic stress and a high-sucrose diet on nonalcoholic fatty liver in young adult male rats // Stress (Amsterdam, Netherlands). – 2017. – Vol. 20(6). – P. 608–617. https://doi.org/10.1080/10253890.2017.1381840

7. Stojiljković V., Todorović A., Kasapović J., Pejić S., Pajović S.B. Antioxidant enzyme activity in rat hippocampus after chronic and acute stress exposure // Annals of the New York Academy of Sciences. – 2005. – Vol. 1048. – P. 373–376. https://doi.org/10.1196/annals.1342.042

8. Mumtaz F., Khan M.I., Zubair M., Dehpour A.R. Neurobiology and consequences of social isolation stress in animal model – A comprehensive review // Biomedicine and Pharmacotherapy. – 2018. – Vol. 105. – P. 1205-1222. https://doi.org/10.1016/j.biopha.2018.05.086

9. Birben E., Sahiner U.M., Sackesen C., Erzurum S., Kalayci O. Oxidative stress and antioxidant defense // World Allergy Organization Journal. – 2012. – Vol. 5(1). – P. 9-19. https://doi.org/10.1097/WOX.0b013e3182439613

10. Vere C.C., Streba C.T., Streba L.M., Ionescu A.G., Sima F. Psychosocial stress and liver disease status // World journal of gastroenterology. – 2009. – Vol. 15(24). – P. 2980–2986. https://doi.org/10.3748/wjg.15.2980

11. Porsolt R.D., Bertin A., Jalfre M. Behavioral despair in mice: a primary screening test for antidepressants // Archives Internationales de Pharmacodynamie et de Therapie. – 1977. – Vol. 229(2). – P. 327-336.

12. Fone K.C., Porkess M.V. Behavioral and neurochemical effects of post-weaning social isolation in rodents – Relevance to developmental neuropsychiatric disorders // Neuroscience & Biobehavior Recviews. – 2008. – Vol. 32(6). – P. 1087-1102. https://doi.org/10.1016/j.neubiorev.2008.03.003

13. Reitman S., Frankel S. A colorimetric method for the determination of serum glutamic oxalacetic and glutamic pyruvic transaminases // American Journal of Clinical Pathology. – 1957. – Vol. 28(1). – P. 56-63. https://doi.org/10.1093/ajcp/28.1.56

14. Bessey O.A., Lowry O.H., Brock M.J. A method for the rapid determination of alkaline phosphatase with five cubic millimeters of serum // Journal of biological chemistry. – 1946. – Vol. 164(1). – P. 321-329.

15. Marklund S., Marklund G. Involvement of the superoxide anion radical in the autoxidation of pyrogallol and a convenient assay for superoxide dismutase // European Journal of Biochemistry. – 1974. – Vol. 47(3). – P. 469-474. https://doi.org/10.1111/j.1432-1033.1974.tb03714.x

16. Aebi H. Catalase in vitro // Methods in Enzymology. – 1984. – Vol. 105. – P. 121-126. https://doi.org/10.1016/s0076-6879(84)05016-3

17. Paglia D.E., Valentine W.N. Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase // Journal of Lanoratory and Clinical Medicine. – 1967. – Vol. 70(1). – P. 158-169.

18. Ellman G.L. Tissue sulfhydryl groups // Archives of Journal of Biochemistry and Biophysics. – 1959. – Vol. 82(1). – P. 70-77. https://doi.org/10.1016/0003-9861(59)90090-6

19. Sapolsky R.M., Romero L.M., Munck A.U. How Do Glucocorticoids Influence Stress Responses? Integrating Permissive, Suppressive, Stimulatory, and Preparative Actions // Endocrine Reviews. – 2000. – Vol. 21(1). – P. 55-89. https://doi.org/10.1210/edrv.21.1.0389

20. Cohen S., Janicki-Deverts D., Miller G.E. Psychological Stress and Disease // JAMA. – 2012. – Vol. 298(14). – P. 1685-1687. https://doi.org/10.1001/jama.298.14.1685

21. Giboney P.T. Mildly elevated liver transaminase levels in the asymptomatic patient // American Family Physician. – 2005. – P. 71(6). – P. 1105-1110.

22. Tian Z., Chen Y., Gao B. Natural killer cells in liver disease // Hepatology (Baltimore, Md.). – 2013. – Vol. 57(4). – P. 1654–1662. https://doi.org/10.1002/hep.26115

23. Ighodaro O.M., Akinloye O.A. First line defence antioxidants-superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx): Their fundamental role in the entire antioxidant defence grid // Alexandria Journal of Medicine. – 2018. – Vol. 54(4). – P. 287-293. https://doi.org/10.1016/j.ajme.2017.09.001

24. Jiang S., Guo T., Guo S., Gao J., Ni Y., Ma W., Zhao R. Chronic Variable Stress Induces Hepatic Fe(II) Deposition by Up-Regulating ZIP14 Expression via miR-181 Family Pathway in Rats // Biology. – 2021. – Vol. 10(7). – P. 653. https://doi.org/10.3390/biology10070653

25. Akhtar M., Pillai K.K., Vohora D. Effect of thioperamide on modified forced swimming test-induced oxidative stress in mice // Basic & clinical pharmacology & toxicology. – 2005. – Vol. 97(4). – P. 218–221. https://doi.org/10.1111/j.1742-7843.2005.pto_140.x

26. Geddie H., Cairns M., Smith L., van Wyk M., Beselaar L., Truter N., Rautenbach F., Marnewick J.L., Joseph D.E., Essop M.F. The impact of chronic stress on intracellular redox balance: A systems level analysis // Physiological reports. – 2023. – Vol. 11(7). – P. e15640. https://doi.org/10.14814/phy2.15640

27. de Haan J.B., Bladier C., Griffiths P., Kelner M., O'Shea R.D., Cheung N.S., Bronson R.T., Silvestro M.J., Wild S., Zheng S.S., Beart P.M., Hertzog P.J., Kola I. Mice with a homozygous null mutation for the most abundant glutathione peroxidase, Gpx1, show increased susceptibility to the oxidative stress-inducing agents paraquat and hydrogen peroxide // The Journal of biological chemistry. – 1998. – Vol. 273(35). – P. 22528–22536. https://doi.org/10.1074/jbc.273.35.22528

REFERENCES

1. Ranabir S., Reetu K. Stress and hormones // Indian Journal of Endocrinology and Metabolism. – 2011. – Vol. 15(3). – P. 193-199. https://doi.org/10.4103/2230-8210.77573

2. Publication manual of the American Psychological Association (7th ed.), 2020. Available online: https://jems.davnepal.edu.np/storage/files/shares/Online%20Resources/American%20Psychological%20Association%20-%20Publication%20Manual%20of%20the%20American%20Psychological%20Association,%20Seventh%20Edition%20(2020)%20[with%20PDF%20bookmarks]-American%20Psychological%20Association%20(2020).pdf (accessed on 10 June 2025)

3. Kabat-Zinn J. Mindfulness-based interventions in context: Past, present, and future. Clinical Psychology // Science and Practice. – 2003. – Vol. 10(2). – P. 144-156. https://doi.org/10.1093/clipsy.bpg016

4. Mayer E.A., Ryu, H.J., Bhatt, R.R. The neurobiology of irritable bowel syndrome // Molecular psychiatry. – 2023. – Vol. 28(4). – P. 1451–1465. https://doi.org/10.1038/s41380-023-01972-w

5. Sies H. Oxidative stress: Concept and some practical aspects // Antioxidants. – 2020. – Vol. 9(9). – P. 852. https://doi.org/10.3390/antiox9090852

6. Corona-Pérez A., Díaz-Muñoz M., Cuevas-Romero E., Luna-Moreno D., Valente-Godínez H., Vázquez-Martínez O., Martínez-Gómez M., Rodríguez-Antolín J., Nicolás-Toledo L. Interactive effects of chronic stress and a high-sucrose diet

on nonalcoholic fatty liver in young adult male rats // Stress (Amsterdam, Netherlands). – 2017. – Vol. 20(6). – P. 608–617. https://doi.org/10.1080/10253890.2017.1381840

7. Stojiljković V., Todorović A., Kasapović J., Pejić S., Pajović S.B. Antioxidant enzyme activity in rat hippocampus after chronic and acute stress exposure // Annals of the New York Academy of Sciences. – 2005. – Vol. 1048. – P. 373–376. https://doi.org/10.1196/annals.1342.042

8. Mumtaz F., Khan M.I., Zubair M., Dehpour A.R. Neurobiology and consequences of social isolation stress in animal model – A comprehensive review // Biomedicine and Pharmacotherapy. – 2018. – Vol. 105. – P. 1205-1222. https://doi.org/10.1016/j.biopha.2018.05.086

9. Birben E., Sahiner U.M., Sackesen C., Erzurum S., Kalayci O. Oxidative stress and antioxidant defense // World Allergy Organization Journal. – 2012. – Vol. 5(1). – P. 9-19. https://doi.org/10.1097/WOX.0b013e3182439613

10. Vere C.C., Streba C.T., Streba L.M., Ionescu A.G., Sima F. Psychosocial stress and liver disease status // World journal of gastroenterology. – 2009. – Vol. 15(24). – P. 2980–2986. https://doi.org/10.3748/wjg.15.2980

11. Porsolt R.D., Bertin A., Jalfre M. Behavioral despair in mice: a primary screening test for antidepressants // Archives Internationales de Pharmacodynamie et de Therapie. – 1977. – Vol. 229(2). – P. 327-336.

12. Fone K.C., Porkess M.V. Behavioral and neurochemical effects of post-weaning social isolation in rodents – Relevance to developmental neuropsychiatric disorders // Neuroscience & Biobehavior Recviews. – 2008. – Vol. 32(6). – P. 1087-1102. https://doi.org/10.1016/j.neubiorev.2008.03.003

13. Reitman S., Frankel S. A colorimetric method for the determination of serum glutamic oxalacetic and glutamic pyruvic transaminases // American Journal of Clinical Pathology. – 1957. – Vol. 28(1). – P. 56-63. https://doi.org/10.1093/ajcp/28.1.56

14. Bessey O.A., Lowry O.H., Brock M.J. A method for the rapid determination of alkaline phosphatase with five cubic millimeters of serum // Journal of biological chemistry. – 1946. – Vol. 164(1). – P. 321-329.

15. Marklund S., Marklund G. Involvement of the superoxide anion radical in the autoxidation of pyrogallol and a convenient assay for superoxide dismutase // European Journal of Biochemistry. – 1974. – Vol. 47(3). – P. 469-474. https://doi.org/10.1111/j.1432-1033.1974.tb03714.x

16. Aebi H. Catalase in vitro // Methods in Enzymology. – 1984. – Vol. 105. – P. 121-126. https://doi.org/10.1016/s0076-6879(84)05016-3

17. Paglia D.E., Valentine W.N. Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase // Journal of Lanoratory and Clinical Medicine. – 1967. – Vol. 70(1). – P. 158-169.

18. Ellman G.L. Tissue sulfhydryl groups // Archives of Journal of Biochemistry and Biophysics. – 1959. – Vol. 82(1). – P. 70-77. https://doi.org/10.1016/0003-9861(59)90090-6

19. Sapolsky R.M., Romero L.M., Munck A.U. How Do Glucocorticoids Influence Stress Responses? Integrating Permissive, Suppressive, Stimulatory, and Preparative Actions // Endocrine Reviews. – 2000. – Vol. 21(1). – P. 55-89. https://doi.org/10.1210/edrv.21.1.0389

20. Cohen S., Janicki-Deverts D., Miller G.E. Psychological Stress and Disease // JAMA. – 2012. – Vol. 298(14). – P. 1685-1687. https://doi.org/10.1001/jama.298.14.1685

21. Giboney P.T. Mildly elevated liver transaminase levels in the asymptomatic patient // American Family Physician. – 2005. – P. 71(6). – P. 1105-1110.

22. Tian Z., Chen Y., Gao B. Natural killer cells in liver disease // Hepatology (Baltimore, Md.). – 2013. – Vol. 57(4). – P. 1654–1662. https://doi.org/10.1002/hep.26115

23. Ighodaro O.M., Akinloye O.A. First line defence antioxidants-superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx): Their fundamental role in the entire antioxidant defence grid // Alexandria Journal of Medicine. – 2018. – Vol. 54(4). – P. 287-293. https://doi.org/10.1016/j.ajme.2017.09.001

24. Jiang S., Guo T., Guo S., Gao J., Ni Y., Ma W., Zhao R. Chronic Variable Stress Induces Hepatic Fe(II) Deposition by Up-Regulating ZIP14 Expression via miR-181 Family Pathway in Rats // Biology. – 2021. – Vol. 10(7). – P. 653. https://doi.org/10.3390/biology10070653

25. Akhtar M., Pillai K.K., Vohora D. Effect of thioperamide on modified forced swimming test-induced oxidative stress in mice // Basic & clinical pharmacology & toxicology. – 2005. – Vol. 97(4). – P. 218–221. https://doi.org/10.1111/j.1742-7843.2005.pto_140.x

26. Geddie H., Cairns M., Smith L., van Wyk M., Beselaar L., Truter N., Rautenbach F., Marnewick J.L., Joseph D.E., Essop M.F. The impact of chronic stress on intracellular redox balance: A systems level analysis // Physiological reports. – 2023. – Vol. 11(7). – P. e15640. https://doi.org/10.14814/phy2.15640

27. de Haan J.B., Bladier C., Griffiths P., Kelner M., O’Shea R.D., Cheung N.S., Bronson R.T., Silvestro M.J., Wild S., Zheng S.S., Beart P.M., Hertzog P.J., Kola I. Mice with a homozygous null mutation for the most abundant glutathione peroxidase, Gpx1, show increased susceptibility to the oxidative stress-inducing agents paraquat and hydrogen peroxide // The Journal of biological chemistry. – 1998. – Vol. 273(35). – P. 22528–22536. https://doi.org/10.1074/jbc.273.35.22528

СОЗЫЛМАЛЫ СТРЕССТІҢ ЕР АЛЬБИНОС ЕГЕУҚҰЙРЫҚТАРЫНДАҒЫ АНТИОКСИДАНТТЫҚ ҚОРҒАНЫС ЖҮЙЕСІНЕ ЖӘНЕ БАУЫР ФУНКЦИЯСЫНА ӘСЕРІ

Оволаби Доркас Оласимбо[1, *], Томас Эммануэль Олусегун[2], Фавуми Даниэл Олуволе[3]

[1]*Биология бөлімі, Илеса университеті, Илеса, Осун штаты, Нигерия.*
[2]*Таза және қолданбалы зоология кафедрасы, Федералдық ауыл шаруашылығы университеті, Абеокута, Огун штаты, Нигерия.*
[3]*Биология ғылымдары бөлімі, Олусегун Агагу ғылым және технология университеті, Окитипупа, Ондо штаты, Нигерия.*

*Тілші автор: Оволаби Д.О., dorcas_owolabi@unilesa.edu.ng.

АҢДАТПА

Қазіргі уақытта біздің әлемде стресс кең таралған, оның физиологиялық процестерге әсері басты алаңдаушылық тудырады. Бұл зерттеу созылмалы стресстің антиоксиданттық қорғаныс жүйесіне және еркек альбинос егеуқұйрықтардағы бауыр қызметіне әсерін зерттейді. Зерттеуге он сегіз еркек альбинос егеуқұйрықтары пайдаланылды. Олар үш топқа бөлінді: бақылау тобы, күштеп жүзу әсеріне ұшыраған топ және әлеуметтік оқшаулануға ұшыраған топ. Төрт апталық стресс протоколынан кейін егеуқұйрықтардың бауыр ферменттері мен антиоксидант деңгейлері спектрофотометриялық талдаулар арқылы анықталды. Осы жұмыстың нәтижелері бойынша жүзу стрессіне ұшыраған егеуқұйрықтарда бауыр ферменттерінің, соның ішінде аланинаминотрансфераза (ALT) деңгейлері (54,46 ±0,46 U/L) бақылаумен (26,79 ±1,20 U/L) және аспартатаминотрансфераза (AST ± 3,0 U/L) бақылау деңгейлерімен (71,1 U/L) салыстырғанда айтарлықтай жоғарылағаны анықталды (22,17 ±0,17 U/L). Сонымен қатар, супероксид дисмутаза (SOD) сияқты антиоксиданттық ферменттер бақылаумен (65,71±0,70 kU) салыстырғанда жүзу стрессімен 197,14 ± 2,14 kU бірлікке дейін өсті, ал каталаза белсенділігі бақылаумен (29,78 ± 0,08 kU) салыстырғанда 48,29 ± 0,29 kU дейін өсті. Нәтижелер созылмалы стресс бауырдың зақымдалуына және тотығу стресіне әкелуі мүмкін екенін көрсетеді, ал жүзу стрессі оқшаулау стрессіне қарағанда көбірек әсер етеді. Бұл нәтижелер созылмалы стресстің бауыр функциясына және жалпы денсаулыққа әсерін түсінуге көмектеседі.

Түйін сөздер: *созылмалы стресс, антиоксидант, бауыр, тотығу, жауаптар.*

ВЛИЯНИЕ ХРОНИЧЕСКОГО СТРЕССА НА АНТИОКСИДАНТНУЮ СИСТЕМУ ЗАЩИТЫ И ФУНКЦИЮ ПЕЧЕНИ У САМЦОВ БЕЛЫХ КРЫС

Оволаби Доркас Оласимбо[1,*], Томас Эммануэль Олусегун[2], Фавуми Даниэль Олуволе[3]

[1] *Кафедра биологии, Университет Илеса, Илеса, штат Осун, Нигерия.*
[2] *Кафедра чистой и прикладной зоологии, Федеральный университет сельского хозяйства, Абеокута, штат Огун, Нигерия.*
[3] *Кафедра биологических наук, Университет науки и технологий Олусегуна Агагу, Окитипупа, штат Ондо, Нигерия.*

*Автор-корреспондент: Оволаби Д.О., dorcas_owolabi@unilesa.edu.ng.

АННОТАЦИЯ

Стресс становится все более распространенным явлением в современном мире, его влияние на физиологические процессы вызывает серьезную озабоченность. Данное исследование посвящено изучению влияния хронического стресса на антиоксидантную систему защиты и функцию печени у самцов белых крыс. В исследовании приняли участие восемнадцать белых крыс-самцов. Они были разделены на три группы: контрольную, группу, подвергавшуюся принудительному плаванию, и группу, подвергавшуюся социальной изоляции. После четырехнедельного стрессового протокола у крыс определялись уровни печеночных ферментов и антиоксидантов с помощью спектрофотометрических анализов. Результаты этой работы показали, что у крыс, подвергшихся стрессу, связанному с плаванием, наблюдалось значительное повышение уровня печеночных ферментов, включая уровни аланинаминотрансферазы (АЛТ) (54,46 ± 0,46 ед/л) по сравнению с контрольной группой (26,79 ± 1,20 ед/л) и уровни аспартатаминотрансферазы (АСТ) (71,30 ± 1,30 ед/л) по сравнению с контрольной группой (22,17 ± 0,17 ед/л). Кроме того, активность антиоксидантных ферментов, таких как супероксиддисмутаза (СОД), увеличилась до 197,14 ± 2,14 единиц при стрессе, связанном с плаванием, по сравнению с контрольной группой (65,71 ± 0,70 единиц), а активность каталазы увеличилась до 48,29 ± 0,29 кЕд по сравнению с контрольной группой (29,78 ± 0,08 кЕд). Полученные данные свидетельствуют о том, что хронический стресс может приводить к повреждению печени и развитию окислительного стресса, причем стресс, связанный с плаванием, оказывает более сильное влияние, чем стресс, связанный с изоляцией. Эти результаты имеют важное значение для понимания влияния хронического стресса на функцию печени и общее состояние здоровья.

Ключевые слова: *хронический стресс, антиоксидант, печень, окислительные реакции.*