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Regulation of oxidative stress and lipid peroxidation induced by epinephrine: The corrective role of L-Glutamic acid

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Abstract. Oxidative stress is related to the development of metabolic and chronic diseases. Mitigation and prevention of the oxidative stress influence remain one of the most pressing issues in biology and medicine. The objective of the research was to examine and compare the role of the glutamic acid, both individually and in combination with pyridoxine, in mitigating the oxidative stress effects elicited by epinephrine. Biochemical methods (determination of the activity of antioxidant enzymes, alanine and aspartate aminotransferases, lipid peroxidation products) and statistical methods were used in the research. The findings indicate that the additional use of L-glutamic acid, both individually and in combination with pyridoxine, allows the body to reach control values or approach them to a greater extent than in groups of animals that did not receive these substances. In particular, such data were found for the following indicators: restored glutathione, lipid hydroperoxides (third experimental group), glutathione peroxidase, thiobarbituric acid reactive substances (second and third experimental groups), superoxide dismutase (spleen, liver, brain), catalase (liver, brain). In contrast, in the first experimental group, which only experienced stress, the activity of superoxide dismutase (spleen, brain, and liver) and catalase (brain, liver, and lungs) decreased compared to the control and the second and third experimental groups. When modelling epinephrine-induced oxidative stress, L-glutamic acid, both individually and in combination with pyridoxine, demonstrated a mitigating effect on the oxidant-antioxidant imbalance, which is a key factor in the level of oxidative stress. The research has shown the potential application of L-glutamic acid for mitigating and protecting the body during states accompanied by oxidative stress

Keywords: activity; biochemical reactions; damage; antioxidant enzymes; rats

Introduction

Oxidative stress, its impact on the human and animal body, and the search for substances with antioxidant properties are among the priority areas of biology and medicine. In particular, the modern lifestyle significantly contributes to the onset of oxidative stress. According to T.R. Kıran *et al.* [1], oxidative stress is an imbalance between the production of free radicals on one hand and antioxidant protection on the other. Stress is one of the factors that lead to damage to organs and systems and the development of diseases. H. Qi *et al.* [2] have investigated that prolonged exposure to oxidative stress can cause structural defects in deoxyribonucleic acid (DNA), as well as functional changes in certain enzymes and cellular structures, consequently leading to cell death.

Amino acids have different antioxidant activities. L-glutamic acid (L-Glu) can protect the body due to its many properties, including antioxidant properties. The L-glutamic amino acid is the main metabolic centre in many organisms [3, 4]. Besides its role in synthesis of protein, it is involved in a variety of processes. L-Glu is a precursor for other amino acids, including L-aspartate, L-alanine, L-proline and L-ornithine. And most importantly, this amino acid, together with L-cysteine and L-glycine, is the synthesis precursor of reduce glutathione (GSH). GSH maintains

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redox homeostasis in the cell and protects against oxidative damage. J. Kumar *et al.* [5] have hypothesized that L-Glu may enhance the antioxidant status and affect the concentration of neurotransmitters. The positive effect of glutamic acid, both alone and in combination with other substances, has also been established in the treatment of nutritional deficiencies [6, 7]. L.A. Ponomarenko *et al.* [8] found that the use of drug based on glutamic acid in the basic therapy of patients with gastroenterological pathology has increased the level of reduced glutathione and normalized lipoperoxidation processes. The results obtained by P.H. Tsai *et al.* [9] revealed that glutamine consumption reduces the expression of genes associated with oxidative stress, and increases antioxidant potential in diabetic rats. D.H. Tran *et al.* [10] propose the existence of a cystine/glutamate antiporter system, wherein intracellular glutamate is expelled to facilitate the uptake of cystine. These researchers also demonstrated that with an increase in oxygen concentration, the uptake of L-glutamic acid by endothelial cells increases. As noted by K. Stach *et al.* [11], pyridoxine (Pyr) or vitamin B6 is a highly significant compound for cellular metabolism. M. Parra *et al.* [12] indicate that Pyr is an extremely important cofactor for numerous biochemical reactions occurring in the cell. Summarizing the above, oxidative stress contributes to the development of diseases and mitigating its consequences remains one of the most urgent problems. Thereby, the aim of the research was to investigate the markers of the antioxidant system and lipid peroxidation under conditions of oxidative stress caused by epinephrine.

Materials and Methods

The studies were conducted using 40 adult Wistar rats (180-200 g) which were kept on the standard diet of the vivarium of the Institute of Animal Biology. The research was conducted in 2020. The rats were kept at the temperature of 22 ± 2°C, the humidity of 50 ± 5% and a 12-h light/12-h dark cycle. Rats (10 animals per group) were divided into 4 groups: three experimental (EG) (Exp.1, Exp.2, Exp.3) and Control (CG). The experiment duration was 24 hours. Three groups of rats (first, second, and third) were administered epinephrine intraperitoneally (2 mg/kg). Then, the rats of the second group were injected with the L-Glu (750 mg/kg), the third experimental group was injected with L-Glu (750 mg/kg) and Pyr (0.430 mg/kg). Rats of the control group – the appropriate amount of saline. Blood and tissues were collected after decapitation of animals under thiopental anaesthesia. The blood samples were centrifuged at 3000×g for 15 min; the tissue samples were homogenized, then centrifuged at 15000×g for 15 min.

Glutathine peroxidase activity (GPx, EC 1.11.1.9) was controlled by the glutathione restoration rate in the nicotinamide adenine dinucleotide phosphate (NADPH) presence [13]. The activity of glutathione reductase (GR, EC 1.6.4.2) was determined by the method of catalysis of NADPH-dependent reduction of the oxidised glutathione (GSSG) and reported as μmol NADPH/min/mg protein [13]. The activity of superoxide dismutase (SOD, EC 1.15.1.1) was defined by the level of inhibition of the rate of nitroblue tetrazolium-reduction. The catalase activity (CAT, EC 1.11.1.6) was defined by formation of a stable complex of molybdenum salts and hydrogen peroxide [13]. The level of reduced GSH was quantified by reactions between the SH groups of GSH and 5,'5'-dithio-bis (2-nitrobenzoic acid) [13]. The content of lipid hydroperoxide (LOOH) was counted as the difference between the control and the experimental values and the content of products reacting from thiobarbituric acid (TBARS) based on the interaction between thiobarbituric acid and malonic aldehyde and represented as nmol of TBARS/mL [13]. The concentration of *aspartate aminotransferase (ASAT)* and alanine *aminotransferase (ALAT)* were investigated in the blood plasma by using a biochemical analyser "Humalyzer 2000" (Germany). Experimental data were processed by methods of variation statistics using OriginPro 8 software. To determine differences between sample means, the Student's t-test was used. Differences with a P value of less than 5% ($P < 0.05$) were considered significant. Research conducted as per the principles of the "European Convention for the Protection of Vertebrate Animals used for Experimental and Other Scientific Purposes" [14] and the Law of Ukraine No. 3447-IV "On Protection of Animals from Cruelty" [15].

Results

Glutathione has a fundamental role against oxidative stress and oxidative damage [16]. The increase in the content of reduced glutathione in the red blood cells (RBC) of rats of the 1st and 2nd groups in comparison with CG was found (Fig. 1(A)). It is worth mentioning the content of the above tripeptide in the L-Glu/Pyr group did not undergo significant changes. GR activity was significantly reduced in animals of all EG (Fig. 1(B)).

The GPx activity in RBC was significantly higher in animals of the first EG in relation to the CG (Fig. 2(A)). Instead, GPx activity did not change in the animals of the 2nd EG and 3rd EG that received additional the above-mentioned substances. The activity of the studied enzyme in animals of the second and third groups was probably lower than in animals of the first EG. No changes in catalase activity were observed in any of the EG of animals (Fig. 2(B)). The decrease in GH may be the lack of reduced NADPH coenzymes formed in the pentose phosphate cycle. It is difficult to explain the prolonged activation of GPx in the animals of the first EG, which was almost 5 times higher compared to the second and third EG that received additional L-Glu and Pyr. Moreover, catalase activity did not change in any of the EG of animals compared to the control. Such changes can be interpreted as mobilisation of the body to overcome the effects of oxidative stress. It can be assumed that this occurred after a previous decrease in this indicator.

The key regulatory systems of the organism include the antioxidant protection system, which regulates the level of free radicals and peroxides formed in biochemical reactions involving reactive oxygen species. The antioxidant

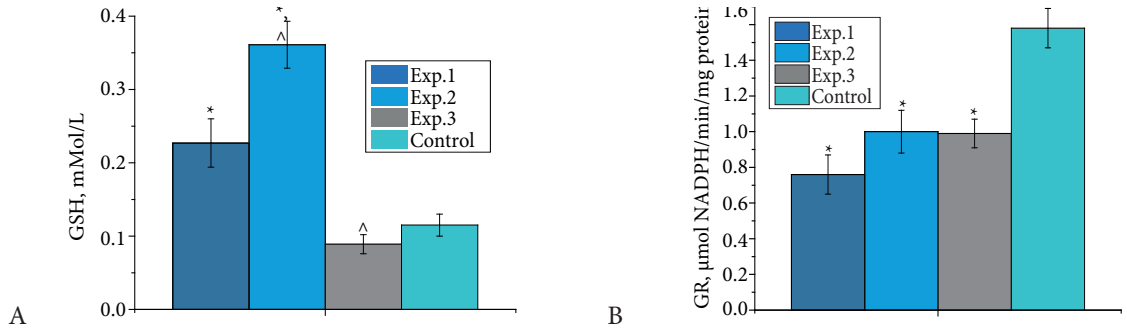

Figure 1. Influence of L-Glu and L-Glu/Pyr on GSH content and GR activity in rat RBCs
Notes: A — GSH content in rat red blood cells under the influence of L-Glu and L-Glu/Pyr. B – GR activity in rat red blood cells under the influence of L-Glu and L-Glu/Pyr. * – differs significantly from the CG ($P < 0.05$). ^ – differs significantly from the 1st EG ($P < 0.05$)
Source: compiled by the author

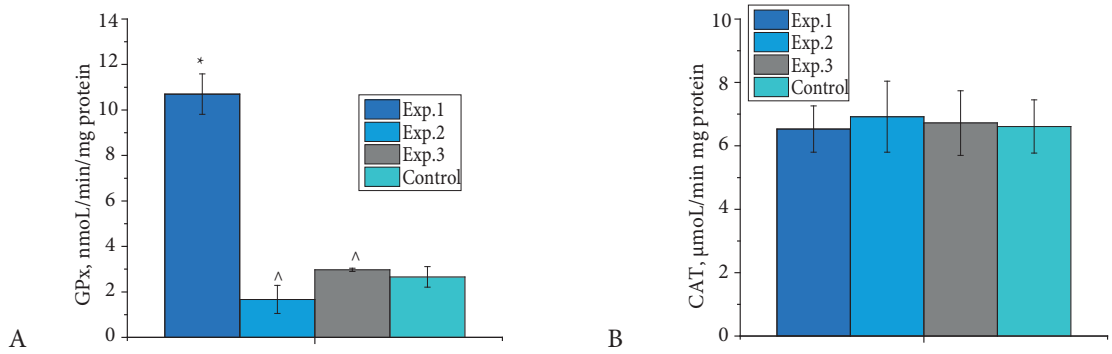

Figure 2. Influence of L-Glu and L-Glu/Pyr on GPx and CAT activity in rat RBCs
Notes: A — GPx activity in rat red blood cells under the influence of L-Glu and L-Glu/Pyr. B – CAT activity in rat red blood cells under the influence of L-Glu and L-Glu/Pyr. * – differs significantly from the CG ($P < 0.05$). ^ – differs significantly from the 1st EG ($P < 0.05$)
Source: compiled by the author

defence system prevents the development of uncontrolled reactions, in particular, lipid peroxidation reactions. Intensification of free radical processes is a universal mechanism of cell damage. This research has shown an intensification of lipid peroxidation in the first and second EG exposed to stress. It is worth noting the increase in the LOOH content in the first and second groups by 65.5% and 43.6%, respectively, and the content of TBARS in 2 times in animals of the first group of animals compared to the control (Fig. 3(A, B)).

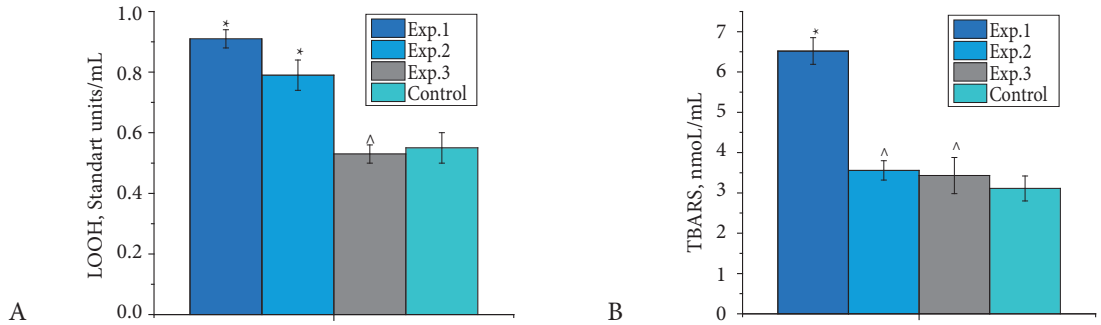

Figure 3. LOOH and TBARS content in rat blood plasma under the influence of L-Glu and L-Glu/Pyr
Notes: A — LOOH content in rat blood plasma under the influence of L-Glu and L-Glu/Pyr. B – TBARS content in rat blood plasma under the influence of L-Glu and L-Glu/Pyr. * – differs significantly from the CG ($P < 0.05$). ^ – differs significantly from the 1st EG ($P < 0.05$)
Source: compiled by the author

The antioxidant defence system in body tissues prevents the development of lipid peroxidation reactions. SOD provides dismutation of the superoxide radical, which is a precursor of the hydroxide radical. The results obtained indicate that superoxide dismutase activity in kidney tissue was higher in animals of the first and second EG compared to control (Fig. 4(A)). This can be explained by the activation of antioxidant enzymes in response to stress. It should be noted that the increase in SOD activity was most pronounced in animals of the first and second EG compared to the CG. Superoxide dismutase activity in spleen tissues was significantly reduced in animals of the first EG by 31.6% compared to the CG. This confirms the data on the antioxidant and membrane-stabilising effects of L-Glu. Lower superoxide dismutase activity was observed in the brain and liver tissues of rats of the first EG injected with epinephrine without the above amino acids compared to the CG. The SOD activity in lung and myocardial of all EG was at the level of the CG. CAT activity in brain and liver tissues was significantly lower by 60.5% and 38.8%, respectively, in animals of the first EG that received epinephrine (Fig. 4(B)). In the animals of the 2nd EG and 3rd EG, which received glutamic acid and glutamic acid in combination with Pyr, CAT activity was at the control level. The amount of catalase in a cell is sufficient to prevent a small amount of H_2O_2 from causing potential toxicity. When analysing catalase activity in lung tissue, it should be noted that this indicator decreased in animals of the first and second EG by 19% and 16.4%, respectively, compared to the CG of rats.

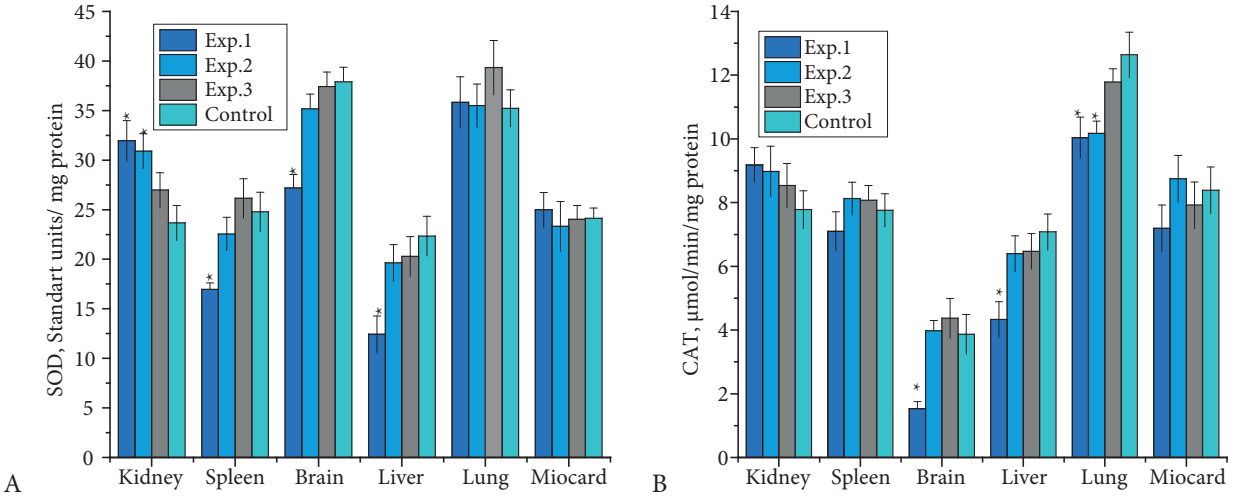

Figure 4. SOD and CAT activity in various rat tissues under the influence of L-Glu and L-Glu/Pyr
Notes: A — SOD activity in rat tissues under the influence of L-Glu and L-Glu/Pyr. B – CAT activity in rat tissues under the influence of L-Glu and L-Glu/Pyr. * – differs significantly from the CG ($P < 0.05$), ^ – differs significantly from the 1st EG ($P < 0.05$)
Source: compiled by the author

The protein level metabolism in the body is indicated by the intensity of reamination processes, which are characterized by the activity of two aminotransferases. Due to the increased biosynthesis of proteins in the organism of rats, the activity of transamination reactions increases. ALAT catalyses the reaction between L-alanine and 2-oxoglutarate, which converts them to L-glutamate and pyruvic acid salt. As can be seen (Table 1), the activity of ALAT in blood plasma did not significant changes. ALAT activity was higher in the first EG of rats, but these data were not significant.

Table 1. Aminotransferase activity in rat blood plasma under the influence of L-Glu and L-Glu/Pyr

Groups	ASAT (U/mL)	ALAT (U/mL)
EG 1	1.57± 0.12*	0.31± 0.03
EG 2	1.03± 0.01	0.29± 0.02
EG 3	0.96± 0.01	0.23± 0.03
Control	0.96± 0.02	0.26± 0.02

Notes: * – differs significantly from the CG ($P < 0.05$)
Source: compiled by the author

As for ASAT (Table 1), which catalyses the reaction between L-aspartate and 2-oxoglutarate, as a result of which they are converted to L-glutamate and oxaloacetate, a significantly higher activity of this aminotransferase was found in animals of the first EG by 1.6 times compared to the control. Elevated levels of ASAT under stress are

considered a sign of mitochondrial stimulation and a marker of tricarboxylic acid cycle activity. The results summing, the additional use of L-glutamic acid, both alone and in combination with pyridoxine, had a positive effect on the body of animals. This was established based on the analysis of most of the studied parameters, the values of which were close to the CG of animals.

Discussion

The effect of the test substances on free radical processes in the blood of rats were investigated. A correlation between oxidative stress indicators and the enzymes' activity was found. The decrease of the GR activity and increase in GPx activity in animals of the first EG exposed to experimental stress may be due to compensatory reactions occurring in the organism in response to the effects of experimental stress. The data obtained by W. He *et al.* [17] also indicate that Glu reduces oxidative stress through direct antioxidant effects and significantly changes the activity of investigated enzymes. SOD and CAT activity in animal tissues also underwent changes under stress, in particular, a reduced content of SOD (spleen, brain, and liver) and CAT (brain, liver, and lungs) of the first EG was found compared to the CG. The SOD activity decrease in first EG of rats is probably due to the depletion of the antioxidant capacity and formation of free radical oxidation products. This is probably due to the adaptive response of superoxide dismutase as a substrate-dependent enzyme to stress. It is worth pointing out that SOD is involved in the scavenging of superoxide generated by electron transport chain complexes. It is worth noting that superoxide dismutase activity in myocardial and lung tissue of all EG was at the level of the CG. This suggests that under conditions of oxidative stress, the antioxidant system is mobilised.

The animals which were injected with L-Glu and Glu/Pyr, respectively, differed favourably. The investigated antioxidant enzymes' activity in the above-mentioned groups were more similar to the data in the CG of rats. Z. Liu *et al.* [18] suggest that there are complex interactions between reactive oxygen species and different types of antioxidants to restore redox balance. This may be associated with the fact that, among other things, the membrane-stabilizing and antioxidant properties of the amino acid which was used in these studies. Pyr is the coenzyme in the conversion of homocysteine to cysteine, which supports GSH biosynthesis. Pyr is a powerful antioxidant, which stores are quickly depleted under stress. Deficiency of Pyr leads to alterations in the many aminoacids metabolism, including glutamate. L-Glu plays the key role in the metabolic processes of many organisms, including nitrogen uptake, amino acid biosynthesis and cofactor production. It can be assumed that this may be due to the fact that glutamic acid is a synthesis precursor of reduced glutathione. G. Lian *et al.* [19] results indicate that glutamine catabolism leads to de novo GSH synthesis. A number of authors also point to the ability of the glutamic acid to reduce the effects of oxidative stress in both plants and animals by regulating the level of antioxidant enzymes [20-22]. J. Fardus *et al.* [20] assume that L-Glu pretreatment mitigated oxidative damage due to keeping ionic homeostasis and raising the activity of antioxidant enzymes (ascorbate peroxidase and catalase).

Other researchers have found that feeding of glutamine can effectively improve immune status by increasing the antioxidant capacity of the experimental animals [21]. The findings are consistent with a number of researchers who have concluded that L-Glu has antioxidant properties [23-25]. The antioxidant properties of glutamic acid are primarily related to the fact that this amino acid is a precursor of numerous biologically active substances, such as reduce glutathine, poly-glutamine phosphate cofactors, pyrimidine and purine nucleotides, some amino acids, in particular, alanine, aspartate, proline, arginine. This is also with the results of this work. In particular, S. Mahdavifard *et al.* [24] established that the use of the aforementioned amino acid led to the normalisation in the level of glutathione in the animals of the EG compared to the control. H. Zhang *et al.* [25] point to the glutamic acid ability to inhibit lipid peroxidation and increase antioxidant capacity. The above-mentioned authors suggest and assume that Glu reduces oxidative stress through direct antioxidant action and increased antioxidant enzyme activity. Based on their own research, authors K. Grucza *et al.* [26] also consider that an increase in GSH levels in the body can be achieved with the help of glutamic acid supplements. The use of the aforementioned amino acid led to the normalisation by the amount of GSH in the animals of the third EG compared to the CG of investigated animals. In the modelling of oxidative stress induced by epinephrine, there is a mitigating effect of glutamic acid, both alone and in combination with pyridoxine on the oxidant-antioxidant imbalance, which is the main factor in the degree of oxidative stress. L-glutamic acid in combination with pyridoxine and L-glutamic acid individually reduce oxidative stress by intensification of the antioxidant enzymes activity and inhibiting lipid oxidation. Moreover, L-Glu/Pyr has a more significant effect than L-Glu. The aforementioned amino acid, by restoring the oxidant-antioxidant balance, participating in protein metabolism, had a positive effect on the body of rats under stress.

Conclusions

When studying and comparing the role of L-glutamic acid individually and in conjunction with Pyr in mitigating the effects of the epinephrine-induced oxidative stress, a change in oxidative stress markers was detected. The results obtained indicate that the supplementary use of L-Glu and L-Glu/Pyr allows the organism to achieve the control level values or approach them to a greater extent than in animals that did not receive the above substances. In particular, such data were found for the following indicators: GSH, LOOH (third EG), GPx, TBARS (second and third EG), SOD (spleen, liver, brain), CAT (liver, brain).

In contrast, the activity of SOD and CAT in the studied tissues also changed under the influence of stress. In particular, in animals of the first EG, a decrease in the content

of SOD (spleen, brain, and liver) and CAT (brain, liver, and lungs) were found compared to the control and the second and third EG. In animals treated with L-Glu and L-Glu/Pyr, no changes in these parameters were found compared to the CG. It was found that the superoxide dismutase activity in the lung and myocardial tissues was at the level of the CG in all study groups. These studies have shown the possibility of using L-Glu to mitigate and defend the body in conditions accompanied by oxidative stress. Further scientific studies are recommended to extensively investigate the possible relationships among various antioxidant markers under the influence of oxidative stress and the role of amino acids in these processes. These investigations will provide more insights into the interaction of the body's antioxidant systems and determine how amino acids affect these systems under stress conditions. This can be of significant importance in the development of new methods for the prevention and treatment of diseases associated with oxidative stress.

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None.

Conflict of Interest

The author declares no conflict of interest.

References

[1] Kıran TR, Otlu O, Karabulut AB. Oxidative stress and antioxidants in health and disease. J Lab Med. 2023;47(1):1–11. DOI: 10.1515/labmed-2022-0108
[2] Qi JH, Dong FX. The relevant targets of anti-oxidative stress: A review. J Drug Target. 2021;29(7):677–86. DOI: 10.1080/1061186X.2020.1870987
[3] Liu W, Zhou Y, Xu X. Study on vapour – liquid equilibrium and microscopic properties of DL-proline, L-glutamic acid, and L-serine aqueous solutions. Front Chem. 2022;10:1005291. DOI:10.3389/fchem.2022.1005291
[4] Salyha NO. L-glutamic acid effect on changes in biochemical parameters of rats intoxicated by carbon tetrachloride. Biol. Tvarin. 2021;23(1):18–22. DOI: 10.15407/animbiol23.01.018
[5] Kumar J, Liddle EB, Fernandes CC, Palaniyappan L, Hall EL, Robson SE, et al. Glutathione and glutamate in schizophrenia: A 7T MRS study. Mol Psychiatry. 2020;25:873–82. DOI: 10.1038/s41380-018-0104-7
[6] Kushta AA, Perminov DO, Melnyk AV, Voloshchuk NI, Taran IV. Study of the effect of arginine glutamate on cognitive processes in food-deprived rats. Rep Vinnytsia Natl Med Univ. 2023;27(2):220–25. DOI: 10.31393/reports-vnmedical-2023-27(2)-07
[7] Vergeles O, Zaytsev O, Holub R. Prospects for the development and use of preparations based on sodium salt of glutamic and succinic acid for agricultural animals. Graal Sci. 2021;(8):138–44. DOI: 10.36074/grail-of-science.24.09.2021.27
[8] Ponomarenko LA, Lykholat OA, Ponomarenko OA. Changes of indicators of oxidative stress in patients with acid-dependent diseases in treatment. Med Clin Chem. 2018;(3):84–89. DOI: 10.61751/mcch.2410-681X.2018.v0.i3.9570
[9] Tsai PH, Liu JJ, Yeh CL, Chiu WC, Yeh SL. Effects of glutamine supplementation on oxidative stress-related gene expression and antioxidant properties in rats with streptozotocin-induced type 2 diabetes. Br J Nutr. 2012;107(8):1112–8. DOI: 10.1017/S0007114511004168
[10] Tran DH, Hung HM, Beckers P, Desmet N, Lamrani M, Massie A, Hermans E, et al. Structural investigation of human cystine/glutamate antiporter system xc- (Sxc-) using homology modeling and molecular dynamics. Front Mol Biosci. 2022;9:1064199. DOI: 10.3389/fmolb.2022.1064199
[11] Stach K, Stach W, Augoff K. Vitamin B6 in health and disease. Nutrients. 2021;13(9):3229. DOI: 10.3390/nu13093229
[12] Parra M, Stahl S, Hellmann H. Vitamin B_6 and its role in cell metabolism and physiology. Cells. 2018;7(7):84. DOI: 10.3390/cells7070084
[13] Salyha NO. Activity of the glutathione system of antioxidant defense in rats under the action of L-glutamic acid. Ukr Biochem J. 2013;85(4):40–47. DOI: 10.15407/ubj85.04.040
[14] European Convention for the Protection of Vertebrate Animals used for Experimental and Other Scientific Purposes. No. 123. Strasbourg; 18.III.1986. [Internet]. [cited 2023 Sep 07]. Available from: https://rm.coe.int/168007a67b
[15] Law of Ukraine No. 3447-IV "On Protection of Animals from Cruelty" (21.02.2006). Available from: https://zakon.rada.gov.ua/laws/show/3447-15#Text
[16] Kumar P, Osahon O, Vides DB, Hanania N, Minard CG, Sekhar RV. Severe glutathione deficiency, oxidative stress and oxidant damage in adults hospitalized with COVID-19: Implications for glynac (glycine and n-acetylcysteine) supplementation. Antioxidants (Basel). 2021;11(1):50. DOI: 10.3390/antiox11010050
[17] He W, Furukawa K, Toyomizu M, Nochi T, Bailey CA, Wu G. Interorgan metabolism, nutritional impacts, and safety of dietary L-glutamate and L-glutamine in poultry. Adv Exp Med Biol. 2021;1332:107–28. DOI: 10.1007/978-3-030-74180-8_7
[18] Liu Z, Ren Z, Zhang J, Chuang C-C, Kandaswamy E, Zhou T, Zuo L. Role of ROS and nutritional antioxidants in human diseases. Front Physiol. 2018;9:477. DOI: 10.3389/fphys.2018.00477
[19] Lian G, Gnanaprakasam JR, Wang T, Wu R, Chen X, Liu L, et al. Glutathione de novo synthesis but not recycling process coordinates with glutamine catabolism to control redox homeostasis and directs murine T cell differentiation. Elife. 2018;7:e36158. DOI: 10.7554/eLife.36158

[20] Fardus J, Hossain MS, Fujita M. Modulation of the antioxidant defense system by exogenous L-glutamic acid application enhances salt tolerance in lentil (*Lens culinaris* Medik.). Biomolecules. 2021;11(4):587. DOI: 10.3390/biom11040587
[21] Wang D, Du Y, Wang S, You Z, Liu Y. Effects of sodium humate and glutamine combined supplementation on growth performance, diarrhea incidence, blood parameters, and intestinal microflora of weaned calves. Anim Sci J. 2021;92(1):e13584 DOI: 10.1111/asj.13584
[22] Wang Q, Han S, Zhu Y, Wang G, Chen D. Poly-γ-glutamic acid coating polymeric nanoparticles enhance renal drug distribution and cellular uptake for diabetic nephropathy therapy. J Drug Target. 2023;31(1):89–99. DOI: 10.1080/1061186X.2022.2106488
[23] Tabassum S, Ahmad S, Madiha S, Shahzad S, Batool Z, Sadir S, Haider S. Free L-glutamate-induced modulation in oxidative and neurochemical profile contributes to enhancement in locomotor and memory performance in male rats. Sci Rep. 2020;10:11206. DOI: 10.1038/s41598-020-68041-y
[24] Mahdavifard S, Sekhavatmand N. Glutamine is a superior protector against lead-induced hepatotoxicity in rats via antioxidant, anti-inflammatory, and chelating properties. Biol Trace Elem Res. 2022;200(11):4726–32. DOI: 10.1007/s12011-021-03046-w
[25] Zhang H, Wang Z, Li Z, Wang K, Kong B, Chen Q. L-glycine and L-glutamic acid protect Pediococcus pentosaceus R1 against oxidative damage induced by hydrogen peroxide. Food Microbiol. 2022;101:103897. DOI: 10.1016/j.fm.2021.103897
[26] Grucza K, Chołbiński P, Kwiatkowska D, Szutowski M. Effects of supplementation with glutathione and its precursors on athlete performance. Biomed J Sci Tech Res. 2019;12(4):9434–41. DOI: 10.26717/BJSTR.2019.12.002293

Регуляція оксидативного стресу та пероксидного окиснення ліпідів, індукованих адреналіном: коригуюча роль L-глутамінової кислоти

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Анотація. Окислювальний стрес асоціюється з розвитком метаболічних та хронічних захворювань. Пом'якшення та запобігання наслідків оксидативного стресу залишається однією з найактуальніших проблем біології та медицини. Метою дослідження було вивчити та порівняти роль L-глутамінової кислоти як окремо, так і в комбінації з піридоксином у пом'якшенні наслідків оксидативного стресу, спричиненого епінефрином. У роботі використовували біохімічні методи (визначення активності антиоксидантних ензимів, аланін- та аспартатамінотрансфераз, вмісту продуктів пероксидного окиснення ліпідів) та статистичні методи. Отримані результати свідчать, що додаткове застосування L-глутамінової кислоти, як окремо, так і у комплексі з піридоксином дозволяє організму вийти на рівень контрольних значень або наблизитися до них більшою мірою, ніж у групах тварин, які не отримували вищезазначені речовини. Зокрема, такі дані були виявлені щодо наступних показників: відновлений глутатіон, гідропероксиди ліпідів (третя дослідна група), глутатіонпероксидаза, продукти реакції з тіобарбітуровою кислотою (друга і третя дослідні групи), супероксиддисмутаза (селезінка, печінка, мозок), каталаза (печінка, мозок). На відміну від цього, у першій дослідній групі, яка зазнавала лише дії стресу, активність супероксиддисмутази (селезінка, мозок і печінка) та каталази (мозок, печінка і легені) знижувалася порівняно з контролем та другою і третьою дослідними групами. При моделюванні оксидативного стресу, індукованого епінефрином, відзначається пом'якшувальний вплив L-глутамінової кислоти як окремо, так і в комбінації з піридоксином на оксидантно-антиоксидантний дисбаланс, що є основним чинником рівня оксидативного стресу. Дослідження показали можливість застосування L-глутамінової кислоти, з метою пом'якшення та захисту організму при станах, що супроводжуються оксидативним стресом

Ключові слова: активність; біохімічні реакції; пошкодження; антиоксидантні ензими; щури