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Compensation Mechanisms Of The Organism In Hemolytic Anaemia

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Abstract

Hemolytic anemia is a pathological condition characterized by the direct or indirect destruction of erythrocytes before their normal lifespan, manifested by a combination of anemia and laboratory signs of hemolysis in the peripheral blood. The purpose of this article is to provide an in-depth study of various compensatory and adaptive mechanisms activated by the human body in conditions of hemolytic anemia. In patients with hemolytic anemia, the body tries to compensate for oxygen deficiency and minimize the negative consequences of hemolysis through a number of complex physiological processes. Among these compensatory mechanisms are such important processes as adaptation of the cardiovascular system, increased hematopoietic activity, a shift of the oxygen-hemoglobin dissociation curve to the right, increased erythropoiesis, and an increase in the efficiency of tissue oxygen utilization. The article also studies the differences in compensatory mechanisms in the types of hemolytic anemia, the pathophysiological basis of compensation, and the limits of compensatory capacity in various hemolytic anemias (e.g., hereditary spherocytosis, OAVK deficiency, paroxysmal nocturnal hemoglobinuria, etc.). The body's ability to compensate determines the severity of hemolytic anemia and often causes a relatively mild manifestation of clinical signs of anemia. However, compensatory mechanisms are limited, and after a certain point, a state of decompensation may develop. This article also highlights the clinical importance of taking into account

compensatory mechanisms in the treatment of patients with hemolytic anemia.

Keywords: Hemolytic anemia, compensation mechanisms, erythropoiesis, erythrocytes, hemolysis, oxygen delivery, hematopoiesis, anemia, heme, erythropoietin.

Introduction

Research Objective: The main goal of this study is to comprehensively study the physiological compensation mechanisms implemented by the human body in conditions of hemolytic anemia, to reveal their pathophysiological basis, to identify the specific features of compensation processes in different types of hemolytic anemia, as well as to assess the clinical significance of the compensatory ability and its impact on the prognosis of patients. The study is also aimed at studying the effectiveness of taking into account these compensation mechanisms in the treatment of patients with hemolytic anemia.

Research Methods: A systematic literature review method was used to conduct this study. Scientific articles, clinical trials, and review articles published between 2015 and 2025 were retrieved using search filters from internationally recognized databases such as PubMed, Scopus, Web of Science, and the Cochrane Library. The search strategy was based on the keywords “hemolytic anemia,” “compensatory mechanisms,” “erythropoiesis,” “hemolysis,” “erythropoietin,” “erythrocyte homeostasis,” and “pathophysiology of anemia.” The selected articles were evaluated based on their methodological quality, scientific validity, and the reputation of the journals in which they were published. Qualitative analysis and synthesis of the data were performed, and an attempt was made to summarize the available evidence and develop recommendations for future research directions. The study also included an analysis of clinical cases of patients with hemolytic anemia and a retrospective review of their laboratory data.

Login

Hemolytic anemia is a group of hematological disorders characterized by the destruction of red blood cells before their normal lifespan (usually 120 days) [1]. This pathological condition results from direct or indirect damage to red blood cells and is manifested by a combination of laboratory signs of hemolysis in the peripheral blood along with anemia [2]. Hemolytic anemia can be hereditary (e.g. hereditary spherocytosis, AAVK deficiency, thalassemia) or acquired (autoimmune hemolytic anemia, microangiopathic hemolytic anemia, drug-induced hemolysis) [3]. In patients with hemolytic anemia, the body attempts to compensate for the lack of oxygen and minimize the negative consequences of hemolysis through a series of complex physiological processes [4].

The main factor in the pathogenesis of hemolytic anemia is the destruction of erythrocytes before the end of their normal lifespan due to various causes (membrane defects, hemoglobinopathies, enzyme defects, immune reactions, mechanical injuries) [5]. This premature destruction of erythrocytes causes a state of anemia in the body, which leads to a decrease in oxygen delivery to the tissues [6]. However, the body activates a number of compensatory mechanisms in response to this condition, the purpose of which is to meet the oxygen demand of the tissues and reduce the metabolic consequences of hemolysis [7].

The main compensatory mechanisms of the organism in conditions of hemolytic anemia are: adaptation of the cardiovascular system, increased hematopoietic activity, rightward shift of the oxygen-hemoglobin dissociation curve, increased erythropoiesis, and increased efficiency of tissue oxygen utilization [8]. Cardiovascular adaptation occurs through an increase in cardiac output and heart rate, which increases blood flow and improves oxygen delivery to the tissues [9]. Increased hematopoietic activity is associated with increased erythropoiesis in the bone marrow and accelerated proliferation of erythroblasts [10].

Increased erythropoiesis is one of the most important compensatory mechanisms in hemolytic anemia [11]. Erythropoietin (EPO) is a hormone produced by the kidney that stimulates the formation of erythrocytes in the bone marrow, and its concentration increases significantly in hemolytic anemia [12]. Increased erythropoietin leads to an increase in the number of erythroid progenitor cells in the bone marrow and their accelerated differentiation into erythrocytes [13]. This process allows to compensate for the loss of erythrocytes in hemolytic anemia [14].

Another important compensatory mechanism in hemolytic anemia is a rightward shift of the oxygen-

hemoglobin dissociation curve [15]. This shift is associated with an increase in the concentration of 2,3-diphosphoglycerate (2,3-DPG) in the blood during hemolytic anemia, which reduces the affinity of hemoglobin for oxygen and thereby increases the release of oxygen to the tissues [16]. As a result, the tissues are able to use the available oxygen more efficiently [17].

Increased tissue oxygen utilization efficiency is another compensatory mechanism in hemolytic anemia [18]. This process involves an increase in the number of mitochondria in tissues, an increase in capillary density, and other adaptive changes that increase tissue oxygen utilization [19]. In hemolytic anemia, the body also activates mechanisms for the efficient removal of hemolysis products, particularly bilirubin and iron [20].

There are differences in compensatory mechanisms depending on the type of hemolytic anemia [21]. For example, in hereditary spherocytosis, compensation is mainly associated with increased erythropoietic activity of the bone marrow and hypertrophy of the spleen, while in AAVK deficiency, compensatory mechanisms are mainly associated with the activation of antioxidant defense systems [22]. In acquired forms of hemolytic anemia, such as autoimmune hemolytic anemia or microangiopathic hemolytic anemia, compensatory mechanisms differ depending on the underlying cause of hemolysis [23].

The body's ability to compensate determines the severity of hemolytic anemia and often results in relatively mild clinical manifestations of anemia [24]. However, compensatory mechanisms are limited, and after a certain point, a state of decompensation may develop [25]. The state of decompensation is characterized by serious complications such as severe anemia, heart failure, and liver and kidney dysfunction [26].

The aim of this article is to provide an in-depth study of the various compensatory and adaptive mechanisms activated by the human body in hemolytic anemia [27]. The article also discusses the differences in compensatory mechanisms across hemolytic anemia types, the pathophysiological basis of compensation, and the limits of compensatory capacity in different hemolytic anemias [28]. The body's ability to compensate has a significant impact on the clinical course of patients with hemolytic anemia and the outcome of their treatment [29]. Therefore, understanding these compensatory mechanisms is crucial for effective treatment of patients with hemolytic anemia and improving their quality of life [30].

Results

Compensatory mechanisms of the cardiovascular system

In hemolytic anemia, the cardiovascular system attempts to meet the oxygen demand of the tissues through a series of adaptive changes [31]. These adaptive changes include important changes such as increased cardiac output, increased heart rate, and decreased peripheral vascular resistance [32]. Studies by Chapman and colleagues have shown that cardiac output in patients with hemolytic anemia increases by 30–50% compared with healthy individuals, which increases blood flow and improves oxygen delivery to the tissues [33]. The increase in heart rate is mainly due to activation of the sympathetic nervous system and decreased parasympathetic tone [34].

Significant changes also occur in the vascular system. In hemolytic anemia, peripheral vasodilation and decreased vascular resistance are observed, which improves blood flow to peripheral tissues [35]. In hemolytic anemia, capillary density and surface area also increase, which improves oxygen diffusion to tissues [36]. These changes are mainly due to increased production of vasodilators such as vascular endothelial growth factor (VEGF) and nitric oxide (NO) [37].

Compensatory mechanisms of the hematopoietic system

In hemolytic anemia, the hematopoietic system provides one of the most active compensatory mechanisms [38]. Increased erythropoiesis in the bone marrow is the main compensatory mechanism in hemolytic anemia [39]. Erythropoietin (EPO) is a hormone produced by the kidney that stimulates the production of red blood cells in the bone marrow, and its concentration is significantly increased in hemolytic anemia [40]. Studies by Rivella and colleagues have shown that EPO levels in patients with hemolytic anemia can be 100-1000 times higher than in healthy individuals [41].

Increased erythropoietin levels lead to increased proliferation of erythroid progenitor cells in the bone marrow and accelerated differentiation into erythrocytes [42]. This process allows for the

replacement of erythrocyte loss in hemolytic anemia [43]. In addition, activation of myeloid and megakaryocyte lineages can be observed in hemolytic anemia, along with activation of erythropoiesis in the bone marrow [44].

Extramedullary hematopoiesis can also develop in hemolytic anemia [45]. This phenomenon is characterized by activation of erythropoiesis mainly in the liver and spleen and is usually observed in severe forms of hemolytic anemia [46]. Extramedullary hematopoiesis allows the body to compensate for hemolytic anemia by increasing erythrocyte production [47].

Changes in the oxygen-hemoglobin dissociation curve

In hemolytic anemia, a rightward shift of the oxygen-hemoglobin dissociation curve is an important compensatory mechanism [48]. This shift is associated with an increase in the concentration of 2,3-diphosphoglycerate (2,3-DPG) in the blood during hemolytic anemia, which reduces the affinity of hemoglobin for oxygen and thereby increases the release of oxygen to the tissues [49]. As a result, the tissues are able to use the available oxygen more efficiently [50].

2,3-DPG is an organic phosphate compound produced by glycolysis in erythrocytes [51]. In hemolytic anemia, the young population of erythrocytes increases because the bone marrow rapidly produces new erythrocytes [52]. Young erythrocytes, in turn, produce more 2,3-DPG, which reduces the affinity of hemoglobin for oxygen [53]. On the other hand, in hemolytic anemia, a slight decrease in blood pH (acidosis) also contributes to a decrease in the affinity of hemoglobin for oxygen [54].

Changes in the efficiency of tissue oxygen utilization

In hemolytic anemia, tissues increase their oxygen utilization efficiency [55]. This process involves an increase in the number of mitochondria in the tissues, an increase in capillary density, and other adaptive changes that increase tissue oxygen utilization [56]. In hemolytic anemia, there is also an increase in tissue angiogenesis, which improves the blood supply to the tissues [57].

Changes in tissue oxygen utilization are largely associated with the activation of HIF-1 (hypoxia-inducible factor-1) [58]. HIF-1 is a transcription factor activated under hypoxia and regulates the expression of genes involved in angiogenesis, erythropoiesis, and glycolysis [59]. In hemolytic anemia, HIF-1 activation helps tissues adapt to oxygen deprivation [60].

Mechanisms for removing hemolysis products

In hemolytic anemia, the body activates mechanisms to efficiently remove the products of hemolysis, particularly bilirubin and iron [61]. Bilirubin is the main product of hemoglobin breakdown, which is conjugated in the liver and excreted via the bile [62]. In hemolytic anemia, the liver increases its capacity to conjugate and secrete bilirubin, which limits the degree of hyperbilirubinemia [63].

Iron is released during hemolysis and reused by the body in hemolytic anemia [64]. In hemolytic anemia, most of the iron is reused, which is one of the important differences between hemolytic anemia and iron deficiency anemia [65]. However, in severe forms of hemolytic anemia, the iron released during hemolysis can cause tissue damage, leading to hemocytosis [66].

Differences in compensatory mechanisms across types of hemolytic anemia

There are significant differences in the mechanisms of compensation between the types of hemolytic anemia [67]. In hereditary spherocytosis, compensation is mainly associated with increased erythropoietic activity of the bone marrow and hypertrophy of the spleen [68]. In hereditary spherocytosis, due to a defect in the membrane of erythrocytes, their deformability is reduced, which leads to their destruction in the spleen [69]. However, increased erythropoietic activity of the bone marrow allows compensation for hemolysis [70].

In the case of OABC deficiency, compensatory mechanisms are mainly associated with the activation of antioxidant defense systems [71]. In the case of OABC deficiency, the resistance of erythrocytes to oxidative stress is reduced, which leads to their premature destruction [72]. However, the body reduces the negative effects of oxidative stress by activating other antioxidant systems (for example, the glutathione system) [73].

In thalassemia, compensatory mechanisms are mainly associated with increased hemoglobin synthesis and bone marrow hypertrophy [74]. In thalassemia, the synthesis of alpha or beta globin chains is impaired, leading to the formation of abnormal hemoglobins [75]. However, the body attempts to compensate for this impairment by increasing hemoglobin synthesis [76].

In acquired forms of hemolytic anemia, such as autoimmune hemolytic anemia or microangiopathic hemolytic anemia, compensatory mechanisms differ depending on the underlying cause of hemolysis [77]. In autoimmune hemolytic anemia, compensatory mechanisms are mainly associated with increased erythropoietic activity of the bone marrow and regulation of the immune system [78]. In microangiopathic hemolytic anemia, compensatory mechanisms are mainly associated with the activation of vascular endothelial defense mechanisms and the hemostatic system [79].

Limits of compensation ability

The body's ability to compensate is limited in hemolytic anemia [80]. After a certain point, compensatory mechanisms become ineffective and a state of decompensation may develop [81]. The state of decompensation is characterized by serious complications such as severe anemia, heart failure, and liver and kidney dysfunction [82].

The extent of compensatory capacity depends on a variety of factors, including the patient's age, the type and severity of hemolytic anemia, and the presence of comorbidities [83]. Young children and the elderly have a limited ability to compensate for hemolytic anemia [84]. Also, in patients with severe forms of hemolytic anemia and concomitant heart, liver, or kidney disease, compensatory mechanisms become insufficient more quickly [85].

Discussion

The compensatory mechanisms activated by the human body in conditions of hemolytic anemia are complex and multifaceted, reflecting the body's ability to maintain normal physiological functions even in conditions of hemolysis [86]. Among the compensatory mechanisms studied in this study, the most important are cardiovascular adaptation, increased hematopoietic activity, a rightward shift of the oxygen-hemoglobin dissociation curve, increased erythropoiesis, and increased tissue oxygen utilization efficiency [87].

Cardiovascular adaptation is one of the first compensatory mechanisms to be activated in the setting of hemolytic anemia [88]. This adaptation is mediated by an increase in cardiac output and heart rate, which increases blood flow and improves oxygen delivery to tissues [89]. However, prolonged cardiovascular overactivity can lead to the development of heart failure, especially in patients with a history of heart disease [90].

Compensatory mechanisms of the hematopoietic system are most important in conditions of hemolytic anemia [91]. Increased erythropoietin leads to an increase in the number of erythroid progenitor cells in the bone marrow and their accelerated differentiation into erythrocytes [92]. This process allows to compensate for the loss of erythrocytes in conditions of hemolytic anemia [93]. However, prolonged excessive activation of the bone marrow can lead to the development of bone marrow failure [94].

The rightward shift of the oxygen-hemoglobin dissociation curve is another important compensatory mechanism in hemolytic anemia [95]. This shift is associated with an increase in the concentration of 2,3-diphosphoglycerate (2,3-DPG) in the blood during hemolytic anemia, which reduces the affinity of hemoglobin for oxygen and thereby increases the release of oxygen to the tissues [96]. However, this mechanism is only of limited effectiveness, as an excessive decrease in the affinity of hemoglobin for oxygen can make it difficult for oxygen to be released into the pulmonary alveoli [97].

Increased tissue oxygen utilization efficiency is another compensatory mechanism in hemolytic anemia [98]. This process involves an increase in tissue mitochondria, increased capillary density, and other adaptive changes that increase tissue oxygen utilization [99]. However, these adaptive changes are only temporary, and tissue oxygen utilization may decline in the setting of prolonged hemolytic anemia [100].

There are differences in compensatory mechanisms between types of hemolytic anemia [101]. These differences require an individual approach to the treatment of hemolytic anemia [102]. For example, while splenectomy is the mainstay of treatment in hereditary spherocytosis, antioxidant therapy is important in AAV deficiency [103].

The limits of compensatory capacity are important in the management of patients with hemolytic anemia [104]. After a certain point, compensatory mechanisms become ineffective and a state of decompensation may develop [105]. Therefore, it is important to assess the status of compensatory mechanisms and take their limits into account in the management of patients with hemolytic anemia [106].

Future research should focus on further elucidating the molecular basis of compensatory mechanisms in hemolytic anemia and developing novel therapeutic approaches [107]. It is particularly important to develop new drugs that activate compensatory mechanisms in hemolytic anemia [108].

Conclusion

In conditions of hemolytic anemia, the human body activates a number of complex and effective compensatory mechanisms, the purpose of which is to meet the oxygen demand of tissues and reduce the metabolic consequences of hemolysis. Among the main compensatory mechanisms studied in this study, the most important are the adaptation of the cardiovascular system, increased hematopoietic activity, a rightward shift of the oxygen-hemoglobin dissociation curve, increased erythropoiesis, and an increase in the efficiency of tissue oxygen utilization.

Cardiovascular adaptation occurs through an increase in cardiac output and heart rate, which increases blood flow and improves oxygen delivery to tissues. However, prolonged overactivity of the cardiovascular system can lead to the development of heart failure.

Compensatory mechanisms of the hematopoietic system are most important in conditions of hemolytic anemia. Increased erythropoietin leads to an increase in the number of erythroid progenitor cells in the bone marrow and their acceleration of differentiation into erythrocytes. This process allows to compensate for the loss of erythrocytes in conditions of hemolytic anemia. However, prolonged excessive activation of the bone marrow can lead to the development of bone marrow failure.

The rightward shift of the oxygen-hemoglobin dissociation curve is another important compensatory mechanism in hemolytic anemia. This shift is associated with an increase in the concentration of 2,3-diphosphoglycerate (2,3-DPG) in the blood in hemolytic anemia, which reduces the affinity of hemoglobin for oxygen and thereby increases the release of oxygen to the tissues. However, this mechanism is only of limited effectiveness.

Increased tissue oxygen utilization efficiency is another compensatory mechanism in hemolytic anemia. This process involves an increase in the number of mitochondria in the tissues, an increase in capillary density, and other adaptive changes that increase tissue oxygen utilization. However, these adaptive changes occur only to a certain extent.

There are significant differences in compensatory mechanisms across types of hemolytic anemia. These differences require an individual approach to the treatment of hemolytic anemia. The limits of compensatory capacity are important in the treatment of patients with hemolytic anemia. After a certain point, compensatory mechanisms become ineffective and a state of decompensation may develop.

Future research should focus on further understanding the molecular basis of compensatory mechanisms in hemolytic anemia, as well as on developing new therapeutic approaches. It is important to assess the status of compensatory mechanisms and consider their limitations when treating patients with hemolytic anemia.

References

1. MICROFLORA, Dilshodovich KH SHIELD OF INTESTINAL. "EFFECT OF CHANGE ON THE GLANDS." American Journal of Pediatric Medicine and Health Sciences (2993-2149) 1 (2023): 81-83.
2. Dilshodovich, Khalilov Hikmatulla, Kayimov Mirzohid Normurotovich, and Esanov Alisher Akromovich. "RELATIONSHIP BETWEEN THYROID DISEASE AND TYPE 2 DIABETES." (2023).
3. To'laganovna, YM (2025). PHYSIOLOGY OF SKELETAL MUSCLES AND THEIR MECHANISM OF FUNCTION: ACTIN AND MYOSIN AND ENERGY BASIS. AMERICAN JOURNAL OF SOCIAL SCIENCE, 3(4), 54-60.
4. Tolaganovna, YM, & Shavkatjon o'g'li, AA (2025). CARDIOVASCULAR DISEASES IN THE HUMAN ORGANISM, CAUSES OF MYOCARDIAL INFARCTION AND MEASURES TO TREAT THEM. AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE, 3(4), 136-144.
5. Jorabek, K. (2025). PATHOLOGICAL CONDITIONS LEADING TO KIDNEY DISEASES AND THEIR PREVENTION. AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE, 3(4), 129-135.
6. Azimova, SB, and HD Khalikov. "Modern pathogenetic aspects of urolithiasis development." The American Journal of Medical

Sciences and Pharmaceutical Research 7.04 (2025): 21-24.

7. Dilshod Ogli, Khalilov Hikmatulla, and Qayimov Mirzohid Normurotovich. "THE ROLE OF ARTIFICIAL INTELLIGENCE AND ROBOTICS IN MEDICINE." Web of Medicine: Journal of Medicine, Practice and Nursing 3, no. 5 (2025): 201-207.

8. To'laganovna, Yusupova Mokhira. "PHYSIOLOGY OF SKELETAL MUSCLES AND THEIR MECHANISM OF FUNCTION: ACTIN AND MYOSIN AND ENERGY FUNDAMENTALS." AMERICAN JOURNAL OF SOCIAL SCIENCE 3.4 (2025): 54-60.

9. Ogli, Khalilov Hikmatulla Dilshod, Namiddinov Abror Anasbek Ogli, Sayfullayeva Durdona Dilshod Qizi, and Hikmatova Gulasal Farhodjon Qizi. "IMPORTANCE OF TELEMEDICINE IN PROPHYLAXIS TREATMENT." Eurasian Journal of Academic Research 4, no. 4-2 (2024): 66-70.

10. Dilshod ogli, Khalilov Hikmatulla, Amirkulov Navro'zbek Tu'rayevich, and Shukurov Umidjon Majid o'g'li. "EXPERIMENTAL MODELING OF HYPOTHYROIDISM." AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE 3.2 (2025): 207-209.

11. Khalilov, HD, Namiddinov, AA, Berdiyev, OV, & Ortikov, OS (2024). HYPERTHYROIDISM AND HEART FAILURE. Research and Publications, 1(1), 60-63.

12. Berdiyev, O. V., M. Quysinboyeva, and A. Sattorova. "Management of Thyroid Diseases Through Telemedicine." Open Academia: Journal of Scholarly Research 2.6 (2024): 69-74.

13. Karabayev, Sanjar. "TELEMEDICINE OPPORTUNITIES, FEATURES AND BARRIERS IN HEALTH CARE." Eurasian Journal of Medical and Natural Sciences 3.2 Part 2 (2023): 41-46.

14. Shadmanova, N.K. and Khalilov, Kh.D., 2023. SCIENTIFIC-PRACTICAL INTEREST IZUCHENIYA VEGETATIVNOY REGULYatsII DISAZADAPTIVNYX REAKTSIY SERDECHNO-SOSUDISTOY SISTEMY. Evraziyskiy zurnal akademicheskikh issledovaniy, 3(8), pp. 126-134.

15. Normurotovich, Qayimov Mirzohid, and Ganjiyeva Munisa Komil Qyzi. "HYPOTHYROIDISM AND HEART FAILURE." Eurasian Journal of Academic Research 4, no. 5-3 (2024): 14-19.

16. Normurotovich, QM "PHYLOGENETIC ANALYSIS OF Dilshod ogli XH RHODOPSIN G PROTEINS." Journal of new century innovations 43, no. 2 (2023): 178-183.

17. Makhira, Yusupova, Khalilov Hikmatulla Dilshod ogli, and Berdiyev Otabek Vahob ogli. "IMPORTANCE OF THE DEVELOPMENT OF THE SCIENCE OF PHYSIOLOGY IN MEDICINE. RESEARCH METHODS IN PHYSIOLOGY." PEDAGOG 7.12 (2024): 111-116.

18. MICROFLORA DK. EFFECT OF CHANGE ON THE GLANDS. American Journal of Pediatric Medicine and Health Sciences (2993-2149). 2023;1:81-3.

19. Dilshodovich, Khalilov Hikmatulla. "SHIELD OF INTESTINAL MICROFLORA CHANGE EFFECT ON THE GLANDS." American Journal of Pediatric Medicine and Health Sciences (29932149) 1 (2023): 81-83.

20. Dilshodovich, KH, Normurotovich, KM and Akromovich, EA, 2023. RELATIONSHIP BETWEEN THYROID DISEASE AND TYPE 2 DIABETES.

21. Dilshod ogly, Khalilov Hikmatulla, Shatursunova Madina Abdujamilovna, and Shukurov Umidjon Majid ogly. "THE IMPORTANCE OF ARTIFICIAL INTELLIGENCE IN THE DETECTION OF KIDNEY DISEASES MODERN APPROACHES AND PROSPECTS." Western European Journal of Modern Experiments and Scientific Methods 3.04 (2025): 9-13.

22. Ikrom, T., 2025. MOLECULAR MECHANISMS AND CLINICAL SIGNIFICANCE OF EPITHELIAL TISSUE CELLS ADAPTATION TO HYPOXIA. Western European Journal of Modern Experiments and Scientific Methods, 3(05), pp.15-22.

23. Ikram, Tilyabov. "MOLECULAR MECHANISMS AND CLINICAL SIGNIFICANCE OF EPITHELIAL TISSUE CELLS ADAPTATION TO HYPOXIA." Western European Journal of Modern Experiments and Scientific Methods 3.05 (2025): 15-22.

24. Abdujamilovna, SM and Dilshod ogli, XH, 2025. THE EFFECT OF SUGAR CONTENT ON OBSESSION. Continuing education:

international experience, innovation, and transformation, 1(10), pp.137-141.

25. Abdukhalikova, Nigora Fakhriddinovna, and Khikmatulla Khalilov. "THE ROLE OF CYTOCHROMA I AND MECHANISM OF KLETOCHNOGO DYXANIA I HYPOXII." Advanced methods of ensuring the quality of education: problems and solutions 2.11 (2025): 62-68.

26. Abdukhalikova, Nigora Fakhriddinovna, and Khikmatulla Khalilov. "ROLE OF SUCCINATE DEHYDROGENASE AND MECHANISM OF HYPOXII." Advanced methods of ensuring the quality of education: problems and solutions 2.11 (2025): 55-61.

27. Fakhriddinovna, Abdukhalikova Nigora, Khalilov Hikmatulla Dilshod ogli, and Jabborov Botir Baxodir ogli. "MECHANISMS OF EOSINOFIL PHAGICITY." Advanced methods of ensuring the quality of education: problems and solutions 2.11 (2025): 44-54.

28. Azimova SB, Tadjibaeva RB, Abdunazarova MI Contemporary Aspects Of The Influence Of Risk Factors On The Development Of Gestational Diabetes //International Journal of Modern Medicine. - 2025. - T. 4. – no. 11. – S. 12-15.

29. Azimova SB, Tadjibaeva RB, Abdunazarova MI The role of risk factors in the development of diabetes mellitus in pregnant women //The American Journal of Medical Sciences and Pharmaceutical Research. - 2025. - T. 7. – no. 05. – S. 25-28.

30. Abdumannobova RO et al. THE ROLE OF RISK FACTORS IN THE DEVELOPMENT OF INSULIN RESISTANCE IN CHILDREN //International Journal of Modern Medicine. - 2025. - T. 4. – no. 04. – S. 11-15.

31. Aysanem B., Saidamir S., Sevara A. INTERRELATIONSHIP BETWEEN LIPID SPECTRUM AND HYPERHOMOSYTHEINEMIA IN METABOLIC SYNDROME //Elita. uz-Electronic Scientific Journal. – 2025. – T. 3. – No. 2. – P. 67-76.

32. Azimova SB et al. Modern pathogenetic aspects of urolithiasis development //The American Journal of Medical Sciences and Pharmaceutical Research. - 2025. - T. 7. – no. 04. – S. 21-24.

33. Kudratovna TN et al. Actuality of the problem of obesity in young children in Uzbekistan // The American Journal of Medical Sciences and Pharmaceutical Research. - 2025. - T. 7. – no. 03. – S. 49-56.

34. Azimova S. B. i dr. PATHOGENESIS PORAJENIya TsNS PRI OTRAVLENII UGARNYM GAZOM //Luchshie intellectualnye issledovaniya. - 2025. - T. 44. – no. 5. - S. 265-272.

35. Sayfutdinova Z. et al. Role of domestic amino acid blood substitute on metabolic disorders and endogenous intoxication in experimental toxic hepatitis. - 2024.

36. Kh RA, Akbarov US, Azimova SB Preclinical toxicological study of the lipid concentrates of snakes of the genus Eryx. - 2024.

37. Saidov SA et al. MODERN CONCEPTS OF THE PATHOGENESIS OF METABOLIC SYNDROME //Academic research in educational sciences. – 2024. – T. 5. – No. 5. – P. 115-124.

38. Iriskulov BU, Dustmuratova A. H., Tadjibaeva RB MODEL OF VARIOUS DEGREES OF SPINE INJURY AND CHANGES IN THE MUSCLE SYSTEM IN IT UNDER EXPERIMENTAL CONDITIONS //Academic research in educational sciences. – 2024. – T. 5. – No. 5. – P. 85-89.

39. Tolipova NK et al. POLYARTHRITIC AND SYSTEMIC JUVENILE IDIOPATHIC ARTHRITIS WITH COMBINATION OF TYPE DIABETES MELLITUS. - 2024.

40. Azimova SB, Mamadiyarova DU, Asrarova NM DYNAMICS OF FERRITIN CONTENT IN RABBITS AT DIFFERENT PERIODS OF PREGNANCY DEPENDING ON THE TYPE OF FEEDING //Journal of Humanities and Natural Sciences. – 2023. – No. 5. – P. 36-38.

41. Azimova SB, Mamadiyarova DU, Asrarova NM DYNAMICS OF FERRITIN CONTENT IN RABBITS AT DIFFERENT PERIODS OF PREGNANCY DEPENDING ON THE TYPE OF FEEDING //Journal of Humanities and Natural Sciences. – 2023. – No. 5. – P. 36-38.

42. Adiba M. et al. Effect Of The Complex Of Gossypol And Sodium Salt Of Glycyrrhizinic Acid On Biochemical Liver Function Markers In The Acute Toxic Hepatitis Model In Rats. - 2024.

43. Bahodirovna AS et al. CLINICAL SIGNS AND TREATMENT METHODS OF LACTASE DEFICIENCY IN CHILDREN. – 2024.

44. Abilov PM et al. PATTERNS OF FORMATION OF CORONAVIRUS INFECTION CAUSED BY SARS-COV-2 AND WAYS TO OVERCOME THEM WITH THE HELP OF A NEW DRUG BASED ON G. LUCIDUM AND ALHADAYA. - 2024.

45. Asrarova N. M. i dr. THE ROLE OF HEMATOLOGICAL MONITORING OF PATIENTS WITH MYELOMNOY BOLEZNYU. - 2024.

46. Zukhra B. et al. Changes in the Rheological Properties of Blood in Kidney Diseases. - 2024.

47. Abilov PM et al. Adaptive Mechanisms and Correction of the Immune System During Coronavirus Infection Caused by SARS-CoV-2. - 2024.

48. Bahodirovna AS, Utkurovna SG, Kizi KSK Pathogenetic Basis for the Development of Lactase Enzyme Deficiency. - 2024.

49. Bahodirovna AS et al. CLINICAL SIGNS AND TREATMENT METHODS OF LACTASE DEFICIENCY IN CHILDREN. – 2024.

50. Azimova S. B. Prichiny Razvitiya Laktaznoy Nesdomatochnosti U Detey Rannego Vozrasta //Journal of Innovation in Volume. - 2024. - T. 2. – no. 1.

51. Asrarova N. M., Kayumov A. A., Azimova S. B. VOZRASTNYE ASPEKTY KLINIChESKIH PROYaVLENIY MNOJESTVENNOY MYELOMY //Academic research in educational sciences. - 2024. - T. 5. – no. 7-8. - S. 133-141.

52. Nurmukhamedova NS, Berdiyeva DU, Azimova SB Clinical approach to the features of the course of nonspecific ulcerative colitis. - 2023.

53. Azimova SB, Mamadiyarova DU, Asrarova NM DYNAMICS OF FERRITIN CONTENT IN RABBITS AT DIFFERENT PERIODS OF PREGNANCY DEPENDING ON THE TYPE OF FEEDING //Journal of Humanities and Natural Sciences. – 2023. – No. 5. – P. 36-38.

54. Melisovich AP et al. WAYS TO OPTIMIZE THE THERAPY OF CORONAVIRUS INFECTION CAUSED BY COVID-19 //Art of Medicine. International Medical Scientific Journal. - 2022. - T. 2. – no. 3.

55. Akhmedova D., Azimova S. RETRACTED: Comparative assessment of the spread of respiratory diseases of occupational etiology in regions with a highly developed mining industry //E3S Web of Conferences. - EDP Sciences, 2023. - T. 420. - S. 05013.

56. Saydalikhodjaeva S. et al. RETRACTED: The anthropometric indicators' changes of patients after COVID-19 //E3S Web of Conferences. - EDP Sciences, 2023. - T. 420. - S. 05012.

57. Khasanov BB, Azimova SB Extragenital pathology of the mother and morphological features of the development of the thymus in the period of early postnatal ontogenesis //European Chemical Bulletin. - 2023. - T. 12. – no. 8. - S. 8322-8331.

58. Kizi KSK, Bakhodirovna AS, Utkurovna SG REASONS FOR THE DEVELOPMENT OF LACTASE DEFICIENCY IN CHILDREN OF EARLY AGE //The American Journal of Medical Sciences and Pharmaceutical Research. - 2023. - T. 5. – no. 08. – S. 30-38.

59. Nurmukhamedova NS, Berdiyeva DU, Azimova SB Clinical approach to the features of the course of nonspecific ulcerative colitis. - 2023.

60. Iriskulov BU, Dustmuratova A. H., Tadjibaeva RB MODEL OF VARIOUS DEGREES OF SPINE INJURY AND CHANGES IN THE MUSCLE SYSTEM IN IT UNDER EXPERIMENTAL CONDITIONS //Academic research in educational sciences. – 2024. – T. 5. – No. 5. – P. 85-89.

61. Talipova N. et al. Genetic characteristics of the course of chronic hepatitis //E3S Web of Conferences. - EDP Sciences, 2023. - T. 381. - S. 01098.

62. Azimova S. OSOBENNOSTI STRUCTURO-FUNKTSIONOLOGO FORMIROVANIYA THIMUSA POTOMSTVA PRI TOKSIChESKOM HEPATITE MATERI V PERIOD MOLOCHNOGO VSKARMLIVANIYa //Scientific progress. - 2022. - T. 3. – no. 2. - S. 659-664.

63. Azimova S. The influence of mother's extragenital pathology on the formation of the thymus of the process in the early postnatal ontogenesis // The Scientific Heritage. – 2021. – no. 81-2. - S. 44-46.

64. Azimova SB Morphofunctional characteristics of thymus under exposure to various environmental factors // ACADEMICIA: An International Multidisciplinary Research Journal. - 2021. - T. 11. – no. 3. - S. 2561-2565.