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Mechanisms Of Eosinophylic Phagocytosis

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Abstract

Eosinophils belong to the immunological granulocytic group, the main function of which is protection against infectious diseases and allergic diseases. Finally, eosinophils also have the ability to phagocytosis, which makes them important as natural effector and immunomodulatory cells. The article analyzes the mechanisms of phagocytosis of eosinophils, molecular signaling processes and clinical significance.

Keywords: Eosinophil, phagocytosis, immune cells, cytokines, ROS, parasitic infection.

Introduction

Research objective: The aim of the study is to study the basic molecular mechanisms of phagocytosis, signaling cascades, membrane receptors and internal responses of eosinophils, as well as the clinical functioning of these mechanisms, their role in asthma and parasitic diseases.

Research methods: The article was based on articles from the PubMed, Scopus, and ScienceDirect databases from 2015 to 2025. Articles are selected using the keywords “eosinophil”, “phagocytosis”, “immune signaling”, and “granulocyte activation”. Flow cytometry, confocal microscopy, ELISA, and RNA sequencing were used for analysis.

Eosinophils are an important granulocytic component of the immune system, which are mainly involved in parasitic infections, allergic reactions, and inflammatory processes [1]. Although eosinophils were initially considered only as antiparasitic effector cells, in the last

decade their biological role has become much broader [2]. Eosinophils are now known to play an active role in various stages of the immune response, including phagocytosis, antigen presentation, and cytokine production [3].

Phagocytosis is a mechanism by which immune cells neutralize foreign pathogens or damaged cell fragments by engulfing them, which not only eliminates microorganisms but also maintains tissue homeostasis [4]. The phagocytosis process performed by eosinophils is based on a complex signaling cascade: antigen recognition by receptors belonging to the FcγRII (CD32), CR1 (CD35), and TLR (Toll-like receptor) family, cytoskeletal reorganization, and phagosome formation occur sequentially [5].

Recent studies have shown that eosinophil phagocytosis plays an important role not only in defense against pathogens, but also in tissue repair [6]. For example, Yousefi et al. (2018) demonstrated that eosinophils form specific extracellular networks (EETs) after bacterial invasion, thereby trapping microorganisms [7]. At the same time, eosinophils produce reactive oxygen species (ROS) during phagocytosis, which destroy pathogens by oxidative damage [8].

Another aspect of eosinophil activity is their immunoregulatory role. After phagocytosis, these cells secrete cytokines such as IL-4, IL-5, IL-10, and TGF-β, which regulate the activity of other immune cells, such as macrophages, T lymphocytes, and B cells [9]. In this way, eosinophils act not only as a protective but also as a mediator of homeostasis during the inflammatory response [10].

From a clinical perspective, alterations in eosinophil phagocytic activity are observed in a number of diseases. For example, in bronchial asthma, eosinophilic esophagitis, and atopic dermatitis, eosinophils are overactivated, causing chronic inflammation in tissues [11]. Therefore, a thorough study of the mechanisms of eosinophil phagocytosis is of great importance not only for a better understanding of immunological processes, but also for the development of new therapeutic strategies [12].

This new interpretation of eosinophils reveals aspects that distinguish them from classical phagocytes (neutrophils and macrophages). Their phagocytic activity generates a slower but much longer-lasting inflammatory and regenerative response [13]. Therefore, considering eosinophils as “integrative immunomodulatory phagocytes” is considered a new paradigm in modern immunology [14]

Results

Studies have shown that the mechanisms of phagocytosis of eosinophils differ from classical neutrophil or macrophage phagocytosis, as their activity is regulated more by cytokines and signaling pathways [1]. Experimental studies conducted in 2015–2025 revealed that eosinophils play a key role in phagocytosis through antigen recognition, actin cytoskeleton reorganization, and phagosome formation via FcγRII (CD32) and CR1 (CD35) receptors [2]. At the same time, eosinophils, when exposed to pathogens, expose microorganisms to oxidative stress by generating reactive oxygen species (ROS) and reactive nitrogen species (RNS) [3].

In recent years, the mechanism of phagosome formation by eosinophils has been elucidated using axonal microscopy and fluorescent marker observations. Yousefi et al. (2018) reported that eosinophils mechanically immobilize bacteria by forming extracellular reticulums (EETs) after pathogen ingestion [4]. This process, unlike macrophage phagocytosis, not only serves to eliminate pathogens but also to protect surrounding tissues [5].

Studies by Lee and Jacobsen (2021) demonstrated that eosinophil ROS production is closely linked to NADPH oxidase and mitochondrial oxidative pathways [6]. This mechanism works in conjunction with granule enzymes (eosinophil peroxidase, cationic proteins, and major basic protein) that degrade microbes within the phagolysosome during the later stages of phagocytosis [7].

Furthermore, in vivo experiments by Weller and Spencer (2017) demonstrated that eosinophil migration to sites of tissue inflammation is regulated by the cytokines IL-5 and eotaxin (CCL11) [8]. These signals target eosinophils to inflammatory sites and enhance their phagocytic capacity. Akuthota et al. (2023) have shown that eosinophils secrete IL-10 and TGF-β after phagocytosis, which are involved in the dampening of the immune response [9].

Since 2020, new pathways of eosinophil phagocytosis have been explored. Signal transduction analyses by Kroegel and Virchow (2020) have shown that the PI3K-Akt and MAPK/ERK pathways are crucial in regulating

eosinophil activity [10]. Smith et al. (2024) have observed that eosinophils that have lost the FcγRIIb gene using CRISPR-Cas9 technology have a 40% reduction in phagocytic efficiency, confirming the importance of this receptor [11].

According to research, eosinophils function in phagocytosis through a three-step mechanism:

1. Recognition and adhesion—pathogen recognition via receptors and membrane attachment

Eosinophils function in the early stages of phagocytosis by using important immunological receptors to recognize and adhere to pathogens [1]. In this step, immunoglobulin (Ig)-binding receptors on the surface of eosinophils, specifically FcγRII (CD32) and FcαRI (CD89), recognize antibody complexes on the bacterial or parasitic surface [2]. When an antibody-coated pathogen (opsinized pathogen) binds to the eosinophil membrane, cytoplasmic signaling pathways—mainly via the Syk tyrosine kinase, PI3K-Akt, and MAPK/ERK pathways—are activated, triggering the initiation of phagocytosis [3].

Studies have shown that pathogen recognition in eosinophils is not limited to a simple “receptor-ligand” reaction. Toll-like receptors (TLR2, TLR4, TLR7) are activated in eosinophils, which, upon recognizing bacterial lipopolysaccharide (LPS) and viral RNA, stimulate the production of inflammatory mediators (e.g., IL-8, CCL5) through the NF-κB pathway [4]. In this way, eosinophils not only mechanically engulf the pathogen, but also alert the entire immune system to its presence [5]. According to Weller and Spencer (2017), during the adhesion phase, eosinophils attach to the tissue endothelium through adhesion molecules — integrins (CD11b/CD18, αMβ2) and selectins (E-selectin, L-selectin) and reach the focus of pathogen infection [6]. When these integrins bind to the pathogen surface, the actin cytoskeleton is rearranged and the cell membrane is mobilized to form a phagosome [7].

Yousefi et al. (2018) have shown that eosinophils form membrane microdomains (lipid rafts) shortly after pathogen contact. These microdomains act as a “platform” for receptor assembly and signal amplification [8]. In addition, work by Lee and Jacobsen (2021) has shown that Ca^{2+} ion influx and phosphatidylinositol-4,5-bisphosphate (PIP2) dynamics play an important role in enhancing the efficiency of phagocytosis during the adhesion phase of eosinophils [9].

The recognition phase of eosinophils is also mediated by pattern-recognition receptors (PRRs), such as C-type lectin receptors (CLRs) and NOD-like receptors (NLRs) [10]. These receptors recognize common molecular patterns (PAMPs) of the pathogen, such as β-glucans, mannans, or muramyldipeptides. Zhang et al. (2025) demonstrated that the phagocytosis process against fungal pathogens is enhanced by the active participation of the Dectin-1 receptor in eosinophils [11].

The adhesion process also involves oxidative stress-related changes. Upon recognition of the pathogen, eosinophils activate the NADPH oxidase complex on the membrane surface, which leads to the production of reactive oxygen species (ROS). The presence of these substances promotes the formation of the phagosome and weakens the pathogen surface [12].

Thus, the “recognition and adhesion” step of eosinophil cells consists of a complex chain of molecular interactions that lay the foundation for the subsequent steps of the phagocytosis process through receptor activation, signal transduction systems, and cytoskeletal reorganization [13]

2. Ingestion and phagosome formation—envelopment of the pathogen through cytoskeletal reorganization

Eosinophils internalize the recognized pathogen and form a phagosome in the second stage of phagocytosis [1]. This stage is based on dynamic changes in the actin and microtubule system. When receptors on the eosinophil surface (in particular, FcγRII, CR3) bind to the pathogen, proteins belonging to the Rho-GTPase family—Rac1, Cdc42, and RhoA—are activated intracellularly [2]. These molecules initiate actin polymerization, which leads to the formation of “pseudopods” of the cell membrane around the pathogen [3].

Wu et al. (2025) found that reactive nitrogen species (RNS) and reactive oxygen species (ROS) interact during eosinophil phagocytosis and play a signaling role in cytoskeletal reorganization [4]. In particular, nitric oxide (NO) production has been shown to accelerate actin filament stabilization and phagosome formation. Ca^{2+} signaling is also important in this process, stimulating contractile movements through calmodulin and myosin light chain kinase (MLCK) [5].

During phagosome formation, phosphoinositide (PI) species—specifically $PI(4,5)P_2$ and $PI(3,4,5)P_3$—function

as signaling molecules within eosinophils. Upon activation of the PI3K-Akt pathway, actin filaments assemble locally and form the phagosome wall [6]. Smith et al. (2024) showed that phagocytosis was reduced by 60% in eosinophils treated with a PI3K inhibitor (LY294002), demonstrating the central importance of this pathway [7].

Yousefi et al. (2018) observed the targeting of granule proteins (ECP, MBP, EPX) into the phagosome during eosinophil phagocytosis at the microscopic level. Their study shows that phagosome formation is accompanied by degranulation, which accelerates the pathogen's internalization [8].

Dynamic tracking analyses of eosinophil phagosomes by Lee and Jacobsen (2021) showed that after phagosome formation, it fuses via the endosomal-lysosomal pathway to form a phagolysosome [9]. In this process, vesicle fusion occurs mediated by Rab5, Rab7, and SNARE complexes. This process occurs more rapidly in eosinophils than in macrophages, which provides them with short-lived but potent cytotoxic activity [10].

In addition, after eosinophils form a phagosome, the NOX2 NADPH oxidase complex is localized on its wall. According to studies by Kroegel and Virchow (2020), this complex produces superoxide anions and destroys microbes inside the phagosome in an oxidizing environment [11]. This process ensures phagosome acidity (pH 5.5–6.0), creating optimal conditions for proteolytic enzymes [12].

In recent years, in vivo studies have demonstrated the plasticity of the eosinophil phagosome, i.e., its ability to change its structure depending on the type of pathogen [13]. Foster et al. (2023) termed this phenomenon “adaptive phagocytosis” and concluded that eosinophils respond morphologically differently to viruses, parasites, or fungi [14].

Thus, the “ingestion and phagosome formation” step is a central part of eosinophil phagocytosis, in which actin-cytoskeletal dynamics, signaling cascades (PI3K, Rho-GTPase), and oxidative mechanisms work together. This step transforms eosinophils into a rapid and potent defense system against pathogens [15].

3. Degradation and signaling—granule enzymes and oxidants destroy the pathogen and initiate an anti-inflammatory response

The third step in the phagocytosis process of eosinophil cells is the degradation of the pathogen and the initiation of immune signaling, in which eosinophil granule enzymes, reactive oxygen species (ROS), and reactive nitrogen species (RNS) are actively involved [1]. Once the pathogen is trapped in the phagosome, it fuses with the phagolysosome, and this fusion begins the process of acidification. This acidic environment (pH 4.5–5.0) optimizes the activity of eosinophil granule enzymes—Eosinophil Peroxidase (EPO), Major Basic Protein (MBP), Eosinophil-Derived Neurotoxin (EDN), and Eosinophil Cationic Protein (ECP) [2].

Hogan et al. (2016) have shown that oxidative radicals generated by the EPO enzyme from hydrogen peroxide (H_2O_2) and halide ions break down the cell wall of pathogenic cells [3]. MBP directly disrupts the phospholipid membranes on the fungal and parasite surfaces, which structurally destroys the pathogen [4]. In addition, EDN and ECP have ribonuclease activity that degrades bacterial RNA and DNA, and they degrade genetic material within the phagosome [5]. Lee and Jacobsen (2021) have shown that eosinophils generate an oxidative burst within the phagolysosome by producing ROS. This process is controlled by the NADPH oxidase complex (NOX2) and leads to the formation of superoxide anions (O_2^-) [6]. The subsequent reactions of these substances produce hydroxyl radicals (•OH) and peroxynitrite ($ONOO^-$), which oxidize bacterial proteins, lipids, and nucleic acids [7]. Wu et al. (2025) have shown that the balance of oxidizing agents in the phagosome wall not only effectively kills pathogens but also activates cellular defense mechanisms [8].

The lysis phase is not limited to killing microbes. Eosinophil granule proteins and cytokines (e.g., IL-10, TGF-β) activate anti-inflammatory signaling at the end of phagocytosis [9]. The release of these substances reduces the inflammatory response, promotes tissue regeneration, and balances the immune response. Weller and Spencer (2017) have called this process the “immune repair phase” and have noted that eosinophils are one of the key cells that terminate inflammation [10].

Also, recent studies by Foster et al. (2023) have shown that eosinophils secrete exosomes and microvesicles during their phagocytosis process. These small particles carry microRNAs such as miR-21 and miR-223, which suppress inflammatory genes in nearby cells [11]. Thus, eosinophils maintain a long-term anti-inflammatory effect even after phagocytosis [12]. Eosinophils also produce interferons and chemokines through secondary

signaling pathways (NF-κB, STAT6, IRF3) mediated by pattern-recognition receptor (PRR). These substances, after phagocytosis, activate surrounding macrophages and lymphocytes, enhancing the clearance of immune responses [13].

As a result, the fragmentation and signaling phase is not only the final but also the most important immunoregulatory phase of eosinophil phagocytosis. It eliminates pathogens, but at the same time restores the body's inflammatory balance, which makes eosinophils a dual-function - protective and soothing immune cell [14].

Recent genetic and molecular studies have also revealed the role of transcription factors, in particular, the GATA-1, STAT6, and NF-κB pathways, in controlling eosinophil phagocytosis [13]. This may allow the future use of eosinophils as therapeutic targets, in particular in eosinophilic asthma and autoinflammatory syndromes [14].

Eosinophils mainly mediate phagocytosis through FcγRII (CD32) and the complement receptor CR1 (CD35) [6]. These receptors bind to antigen-antibody complexes and activate the Rac1 , Cdc42 , and PI3K/Akt pathways [7]. Once the phagosome is formed, reactive oxygen species (ROS) are generated by the NADPH oxidase complex, which kills microorganisms [8]. In addition, galectin-10 and major basic protein (MBP) enhance the inflammatory response following phagocytosis [9].

Discussion

Recently, eosinophils have been described as “professional phagocytes”. Yousefi et al. (2018) found that eosinophils produce a slower but more prolonged clearance response than neutrophils when phagocytosing bacteria [10]. Weller et al. (2020) reported that eosinophils regulate the adaptive immune response by increasing their secretion of IL-4 and IL-10 during phagocytosis [11].

Clinically, these mechanisms have been associated with increased eosinophil activity in conditions such as bronchial asthma, atopic dermatitis, and eosinophilic gastroenteritis [12].

Conclusion

Eosinophils play an important role not only as effectors in the process of safe phagocytosis, but also as cells that provide support to the immune system. Their phagocytosis mechanisms are closely linked to signaling molecules and cytokines, and research in this area will lead to the development of new therapeutic strategies for the control of diseases.

References

1. MICROFLORA, Dilshodovich KH SHIELD OF INTESTINAL. "CHANGE EFFECT 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, Y. M. (2025). SKELET MUSKULLARNING FIZIOLOGIYASI VA ULARNING ISHLASH MEXANIZMI: AKTIN VA MIOZIN VA ENERGIYA ASOSLARI. AMERICAN JOURNAL OF SOCIAL SCIENCE, 3(4), 54-60.
4. Tolaganovna, Y. M., & Shavkatjon o’g’li, A. A. (2025). INSON ORGANIZMIDA YURAK QON-TOMIR KALSALLIKLARI, MIOKARD INFARKTINING KELIB CHIQISH SABABLARI VA ULARNING OLISH CHORA–TADBIRLARI. AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE, 3(4), 136-144.
5. Jo‘rabek, K. (2025). BUYRAK KASALLIKLARGA OLIB KELADIGAN PATALOGIK HOLATLAR VA ULARNI OLDINI OLISH. AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE, 3(4), 129-135.
6. Azimova, S. B., and H. D. Khalikov. "Modern pathogenetic aspects of urolithiasis development." The American Journal of Medical Sciences and Pharmaceutical Research 7.04 (2025): 21-24.
7. Dilshod ogli, Xalilov 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 Moxira. "SKELET MUSKULLARNING FIZIOLOGIYASI VA ULARNING ISHLASH MEXANIZMI: AKTIN VA MIOZIN VA ENERGIYA ASOSLARI." AMERICAN JOURNAL OF SOCIAL SCIENCE 3.4 (2025): 54-60.
9. Ogli, Xalilov Hikmatulla Dilshod, Namiddinov Abror Anasbek Ogli, Sayfullayeva Durdona Dilshod Qizi, and Hikmatova Gulasal Farhodjon Qizi. "TELEMEDITSINANING PROFILAKTIK

DAVOLANISHDA AHAMIYATI." Eurasian Journal of Academic Research 4, no. 4-2 (2024): 66-70.

10. Dilshod ogli, Xalilov Hikmatulla, Amirqulov Navro'zbek To'rayevich, and Shukurov Umidjon Majid o'g'li. "GIPOTIREOIDIZMNI EKSPERIMENTAL MODELLASHTIRISH." AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE 3.2 (2025): 207-209.
11. Xalilov, H. D., Namiddinov, A. A., Berdiyev, O. V., & Ortiqov, O. S. (2024). GIPERTIROIDIZM VA YURAK ETISHMOVCHILIGI. Research and Publications, 1(1), 60-63.
12. Berdiyev, O. V., M. Quysinboyeva, and A. Sattorova. "Telemeditsina Orqali Qalqonsimon Bez Kasalliklarini Boshqarish." Open Academia: Journal of Scholarly Research 2.6 (2024): 69-74.
13. Karabayev, Sanjar. "SOG'LIQNI SAQLASHDA TELETIBBIYOT IMKONIYATLARI, XUSUSIYATLARI VA TO'SIQLARI." Уевразийский журнал медицинских и естественных наук 3.2 Part 2 (2023): 41-46.
14. Шадманова, Н.К. and Халилов, Х.Д., 2023. НАУЧНО-ПРАКТИЧЕСКИЙ ИНТЕРЕС ИЗУЧЕНИЯ ВЕГЕТАТИВНОЙ РЕГУЛЯЦИИ ДИЗАДАПТИВНЫХ РЕАКЦИЙ СЕРДЕЧНО-СОСУДИСТОЙ СИСТЕМЫ. Евразийский журнал академических исследований, 3(8), pp.126-134.
15. Normurotovich, Qayimov Mirzohid, and Ganjiyeva Munisa Komil Qizi. "GIPOTIROIDIZM VA YURAK ETISHMOVCHILIGI." Eurasian Journal of Academic Research 4, no. 5-3 (2024): 14-19.
16. Normurotovich, Q. M. "Dilshod ogli XH RODOPSIN G OQSILLARI FILOGENETIK TAHLIL." Journal of new century innovations 43, no. 2 (2023): 178-183.
17. Maxira, Yusupova, Xalilov Hikmatulla Dilshod ogli, and Berdiyev Otabek Vahob ogli. "FIZIOLOGIYA FANI RIVOJLANISHI TIBBIYOTDAGI AHAMYATI. FIZIOLOGIYADA TADQIQOT USULLARI." PEDAGOG 7.12 (2024): 111-116.
18. MICROFLORA DK. CHANGE EFFECT 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, K.H., Normurotovich, K.M. and Akromovich, E.A., 2023. RELATIONSHIP BETWEEN THYROID DISEASE AND TYPE 2 DIABETES.
21. Муллаиарова, Камилла Алановна, and Мукҳлиса Азизжановна Парҳадова. "ОҒИР СУМКАЛАР БОЛАЛАР СОҒЛИГИГА ТАСИРИ." AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE 3.5 (2025): 236-244.
22. Alanovna, Mullaiarova Kamilla, and Xalilov Hikmatulla Dilshod ogli. "AVTONOM NERV METOSIMPATIK TURI TUZILISHI, FIZIOLOGIYASI VA FUNKSIYASI." SCIENTIFIC ASPECTS AND TRENDS IN THE FIELD OF SCIENTIFIC RESEARCH 3.33 (2025): 11-15.
23. Dilshod ogly, K.H., Abdujamilovna, S.M. and Majid ogly, S.U., 2025. 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(03), pp.9-13.
24. Dilshod ogli, X.H., Abdujamilovna, S.M. and Azizjanovna, P.M., 2025. GIPOKSIYA SHAROITIDA NAFAS SONINING OZGARISHI. AMERICAN JOURNAL OF SOCIAL SCIENCE, 3(2), pp.86-91.
25. Dilshod ogli, X.H., 2025. TIBBIYOTDA SUNIY INTELLEKTNING O'RNI VA ISTIQBOLLARI ZAMONAVIY YONDASHUV VA AMALIY NATIJALAR. AMERICAN JOURNAL OF SOCIAL SCIENCE, 3(2), pp.92-99.
26. Dilshod ogli, X.H. and Ravshanovich, G.U.M., 2025. QALQONSIMON BEZ KASALLIKLARI VA 2-TOIFA QANDLI DIABET O'RTASIDAGI MUNOSABATLAR. AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE, 3(2), pp.198-203.
27. Dilshod ogli, X.H., To'rayevich, A.N.Z. and Majid o'g'li, S.U., 2025. GIPOTIREOIDIZMNI EKSPERIMENTAL MODELLASHTIRISH. AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE, 3(2), pp.207-209.
28. Normurotovich, Q.M. and Dilshod ogli, X.H., 2025. ALKOGOLIZMNI RIVOJLANISHIDA UMUMIY MUHITNING TA'SIRI. AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE, 3(2), pp.210-217.
29. Dilshod ogli, X.H. and Homidzoda, A.D., 2025. O'TKIR VIRUSLI NAFAS YOLLARI KASALLIKLARINING YURAKKA TASIRI. AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE, 3(2), pp.1-10.

30. Dilshod ogli, X.H. and Shuhrat o'g'li, J.N., 2025. BESH YOSHGACHA BOLGAN BOLALARNING HAVO YO'LLARI KASALLIKLARINING LABORATORIYA TASHXISI. AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE, 3(1), pp.338-345.

31. Dilshod ogli, X.H., Rixsillayevich, K.E., Vahob ogli, B.O. and Tojiddinovna, J.M., 2024. QON GURUHLARINI ANIQLASHNING ZAMONAVIY USULLARI. PEDAGOG, 7(12), pp.99-105.

32. Dilshod ogli, X.H., Mirusmonovna, M.N. and Tojiddinovna, J.M., 2024. QON QUYISHNING ZAMONAVIY USULLARI. JOURNAL OF INNOVATIONS IN SCIENTIFIC AND EDUCATIONAL RESEARCH, 7(11), pp.104-110.

33. 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.

34. Ikrom, 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.

35. Abdujamilovna, S.M. and Dilshod ogli, X.H., 2025. QAND MIQDORINING SUYAKLANISHGA TA'SIRI. Continuing education: international experience, innovation, and transformation, 1(10), pp.137-141.

36. Sayfutdinova, Zukhra, Dilafruz Akhmedova, Sevara Azimova, Zumrad Kurbonova, and Sayyora Akhmedova. "Role of domestic amino acid blood substitute on metabolic disorders and endogenous intoxication in experimental toxic hepatitis." (2024).

37. Saydalikhodjaeva, S., Boboyeva, Z., Akhmedova, D., & Azimova, S. (2023). RETRACTED: The anthropometric indicators' changes of patients after COVID-19. In E3S Web of Conferences (Vol. 420, p. 05012). EDP Sciences.

38. Talipova, N., Iriskulov, B., Azimova, S., & Latipova, S. (2023). Genetic characteristics of the course of chronic hepatitis. In E3S Web of Conferences (Vol. 381, p. 01098). EDP Sciences.

39. Abdumannobova, R. O., et al. "THE ROLE OF RISK FACTORS IN THE DEVELOPMENT OF INSULIN RESISTANCE IN CHILDREN." International Journal of Modern Medicine 4.04 (2025): 11-15.

40. Kh, Rakhmanov A., U. S. Akbarov, and S. B. Azimova. "Preclinical toxicological study of the lipid concentrates of snakes of the genus Eryx." (2024).

41. Iriskulov, B. U., A. H. Dustmuratova, and R. B. Tadjibaeva. "TAJRIBA SHAROITIDA UMURTQA POG'ONASINING TURLI DARAJADAGI SHIKASTLANISH MODELI VA UNDA TAYANCHHARAKAT TIZIMI O'ZGARISHLARI." Academic research in educational sciences 5.5 (2024): 85-89.