UDC: 612.111.6:612.397.13:636.52/.58-092

THE DEVELOPMENT OF HEMOGLOBIN SYNTHESIS AND IRON METABOLISM BIOCHEMICAL PROCESSES AS A RESULT OF NUTRITION WITH WHEY POWDER AND CHITOSAN

Rakhmonov Farhod Kholbayevich
Assistant, Zarmed University, Samarkand, Uzbekistan.
E-mail: ***farxod1313jon@gmail.com***
Mukumova Shakhzoda Ilkhomjonovna
Student, Zarmed University, Samarkand, Uzbekistan.
E-mail: ***sahzodamukumova@gmail.com***

https://doi.org/10.5281/zenodo.18582132

Annotation. *The present paper examines the biochemical mechanisms related to hemoglobin formation and iron metabolism under the influence of dietary supplementation with whey powder and chitosan. Scientific evidence is analyzed to assess how these bioactive components improve heme synthesis, enhance iron absorption, support ferritin-based storage, regulate oxidative stress and increase erythropoietic activity. Lactoferrin, cysteine and glycine, present in whey, are shown to contribute to intestinal iron uptake, enterocyte transport and stabilization of erythrocyte membranes by promoting antioxidant defense. The findings demonstrate that the combination of whey powder and chitosan may serve as an effective dietary intervention for preventing iron-deficiency anemia and activating the hematopoietic system.*

Keywords: *hemoglobin synthesis, iron metabolism, whey powder, chitosan, erythropoiesis, lactoferrin, ferritin, iron absorption, antioxidant defense, enterocyte transport, bioavailability, red blood cells, anemia prevention, heme formation, intestinal permeability.*

Introduction. Hematopoiesis governs the continuous production and functional renewal of erythrocytes, a vital process necessary for sustaining oxygen transport, cellular redox balance and aerobic energy production [17, 18]. Hemoglobin, the central oxygen-carrying protein, consists of a heme core in which iron is the key metallic element [1, 18]. When dietary iron intake is insufficient or its absorption is impaired, erythropoiesis declines, causing anemia and reduced physiological resilience [21, 22]. It is important to stress that consumption of iron-rich food does not directly equate to efficient iron uptake.

Absorption depends on the biochemical status of the intestinal mucosa, the presence of reducing agents, inflammatory tone, microbiota composition and the efficiency of enterocyte-mediated transport [8, 9]. Whey powder possesses high biological value, containing essential amino acids such as cysteine, glycine, valine and isoleucine, which are necessary substrates for heme synthesis [3, 6]. Among whey-derived proteins, lactoferrin is particularly important because it binds iron, prevents its conversion to toxic free radicals, provides safe intracellular transport and contributes to immune defense [2, 10, 13].

Meanwhile, chitosan, a natural biopolymer, improves gut epithelium permeability, suppresses inflammation and strengthens iron bioavailability, which makes it especially important when intestinal iron absorption is physiologically limited [4, 20].

Methodology. The study employed a theoretical and bibliographic research design. Literature was retrieved from PubMed, Scopus, Google Scholar, e-Library and CyberLeninka databases. The conceptual base included the classical physiological-biochemical models of iron homeostasis described by Hallberg, Anderson and Murray [1, 5, 8], as well as the experimental

findings of Rakhmonov F.-X. who studied the physiological and biochemical impacts of chitosan and whey powder on broiler chickens [14, 15, 16].

Main body. Heme biosynthesis begins within mitochondria where glycine condenses with succinyl-CoA, a process dependent on pyridoxal phosphate (vitamin B6), to form δ-aminolevulinic acid [1, 18]. Subsequent enzymatic stages lead to protoporphyrin IX, into which ferrous iron (Fe^{2+}) is inserted to complete the heme molecule [6, 18]. For this incorporation to occur efficiently, iron must be available in reduced form. Reduction of ferric iron (Fe^{3+}) into ferrous form is facilitated by glutathione – the synthesis of which depends primarily on cysteine availability [7]. Upon entry into the intestine, dietary iron undergoes several steps: the enzyme Dcytb mediates redox conversion; the DMT1 channel transfers iron across enterocyte membranes; and transferrin functions as the extracellular iron carrier to hematopoietic tissues [9, 12]. Lactoferrin acts synergistically, preventing oxidative stress-induced damage and stabilizing ferritin, enabling safe intracellular iron storage [2, 10, 11]. Chitosan, due to its cationic structure, improves intestinal barrier integrity, modulates cytokine activity, accelerates nutrient diffusion and increases the efficiency of iron transport from the lumen into systemic circulation [4, 20]. Experimental data by Rakhmonov F.-X. indicate that combined supplementation of chitosan and whey powder in broiler chicks increased hemoglobin values by 12–19 percent and total iron-binding capacity by 25–31 percent [14, 15, 16].

Analysis. Examination of research findings allows the following synthesized interpretations. Whey powder increases true bioavailability of iron by up to 42 percent, particularly due to improved reducing conditions and absorption efficiency [3, 19]. Lactoferrin supports the stabilization of ferritin levels and prevents iron-induced free radical formation [2, 11, 13]. Chitosan contributes to restoration of gut membrane integrity, enhances iron transport and supports systemic hematopoietic activity [4, 20]. Post-Soviet clinical works describe erythropoiesis metabolism as a central diagnostic indicator of nutritional health, confirming that nutritional substrates may serve as modifiable physiological factors influencing red blood cell synthesis [17, 21].

Conclusion. Whey powder and chitosan influence iron metabolism at molecular and systemic levels. Whey provides substrates and biochemical cofactors required for heme synthesis, while chitosan optimizes gastrointestinal absorption conditions. When administered in combination, these substances reduce the risk of anemia, increase hematopoietic efficiency and support red blood cell membrane stability.

References.

1. Murray R. Biochemistry. New York: McGraw-Hill, 2018.
2. Ochoa T. J. Lactoferrin and iron metabolism. Clin. Rev., 2015, 18(2): 58–67.
3. Brown C. Biologic activity of whey peptides. Nutr. Rev., 2021, 79(8): 62–75.
4. Wang S., Liu F. Chitosan and mineral uptake. Food Chem., 2019, 220: 154–162.
5. Anderson G. J. Iron homeostasis mechanisms. J. Biochem., 2014, 98(5): 320–329.
6. Smith K. Amino acid metabolism and erythropoiesis. J. Med. Sci., 2017, 25(4): 120–138.
7. Layne E. Glutathione biosynthesis pathways. Mol. Cell. Sci., 2016, 11: 77–89.
8. Hallberg L. Iron absorption in humans. Scand. J. Clin. Lab. Invest., 2012, 46(3): 234–247.
9. Kim S. Enteral iron uptake physiology. Asian Med. J., 2019, 14(1): 42–53.

10. Pan V. Lactoferrin transport in intestinal epithelium. Int. J. Med. Biol., 2018, 77(2): 200–204.
11. Stevens K. Ferritin modulation in erythropoiesis. Clin. Hematol., 2016, 28(3): 140–149.
12. Kravchenko A. V. Transferrin and its role in iron metabolism. Biochemistry and Medicine, 2017, 14(4): 50–57.
13. Petrova N. N. Lactoferrin in iron deficiency anemia therapy. Med. Journal RF, 2019, 9(1): 42–49.
14. Holbayevich R. F. Chitosan and physiological indicators of broiler chicks. Open Access Repository, 2023, 4(3): 1389–1395.
15. Rakhmonov F., Eshimov D. The effect of chitosan and whey powder on weight of broiler chickens. BIO Web of Conf., 2024, 95: 01025.
16. Holbayevich R. F., Dusmurod E. Physio-biochemical indicators of broiler chicks fed whey. Academia Repository, 2024, 5(2): 184–187.
17. Zaytseva I. I. Erythropoiesis metabolism. Kazan Med. J., 2018, 99(6): 11–22.
18. Belova M. A. Biochemistry of erythrocytes. St. Petersburg: MedLit, 2015.
19. Turner J. Whey protein effects on metabolism. Sports Sci. Rev., 2022, 33(4): 88–107.
20. Ivanchenko A. Chitosan and GI absorption. Biol. Khim., 2020, 66(7): 90–97.
21. Hamilton R. Nutrition and hematology. Hematol. Today, 2015, 12(2): 15–31.
22. Fedorov O. P. Iron deficiency and dietary substrates. Ter. Arkhiv, 2020, 92(1): 55–61.