



THE ROLE OF VITAMIN D IN CHRONIC HEART FAILURE

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Abstract

This article examines the role of vitamin D in chronic heart failure and its effect on the cardiovascular system. It has been established that vitamin D deficiency negatively affects the contractile activity of the heart muscle, the activity of the renin-angiotensin-aldosterone system, and inflammatory processes. As a result of the studies, it was shown that a sufficient level of vitamin D can improve heart function, slow down the development of the disease, and serve to improve the quality of life of patients.

Keywords: Chronic heart failure, vitamin D, cardiovascular system, myocardial function, renin-angiotensin-aldosterone system, hypovitaminosis D, inflammatory process, cardiac activity, prevention, metabolic disorders.

Chronic heart failure is one of the most pressing problems of the cardiovascular system and is characterized by high morbidity and mortality rates worldwide. This disease occurs as a result of a decrease in the heart's ability to adequately supply the body's tissues with blood. In recent years, the role of metabolic factors, including vitamins and trace elements, in the pathogenesis of chronic heart failure has been widely studied. Vitamin D is considered not only a regulator of calcium-phosphorus metabolism, but also a biologically active hormone that affects the activity of the heart muscle. It has been established that its deficiency increases the risk of developing arterial hypertension, ischemic heart disease, and chronic heart failure. Vitamin D affects the cardiovascular system through several mechanisms. One of the most important mechanisms is the regulation of the renin-angiotensin-



aldosterone system (RAAS). Scientific research shows that vitamin D deficiency leads to excessive activation of the renin-angiotensin-aldosterone system, which is one of the main links in the pathogenesis of heart failure. According to experimental data, vitamin D can suppress the expression of the renin gene. As a result, the level of angiotensin II decreases, arterial pressure increases, and the load on the heart decreases. Vitamin D deficiency is observed in most patients with chronic heart failure. According to the results of randomized clinical trials, the intake of vitamin D supplement in such patients led to a decrease in the level of aldosterone in blood plasma. This helps to slow down the processes of structural restructuring of the heart muscle. In addition, some meta-analyses have shown that vitamin D intake leads to an improvement in left ventricular function and a decrease in ventricular remodeling. It is assumed that this effect can be realized through a decrease in the activity of the system. Additionally, vitamin D has anti-inflammatory effects, reduces oxidative stress, and improves endothelial function. This leads to an increase in the contractile ability of the heart muscle and the general health of patients.

Chronic heart failure is a complex clinical syndrome characterized by the inability of the heart to pump sufficient blood to meet metabolic demands. Despite advances in pharmacological and device therapies, prognosis remains poor. Recently, attention has focused on modifiable metabolic and hormonal factors contributing to CHF progression, including vitamin D deficiency.

Vitamin D deficiency affects more than one billion individuals worldwide and is particularly common in elderly patients and those with chronic diseases, including heart failure.

Vitamin D Metabolism and Cardiovascular System

Vitamin D is obtained through skin synthesis following sunlight exposure and from dietary sources. It undergoes hepatic conversion to 25-hydroxyvitamin D and



renal activation to 1,25-dihydroxyvitamin D (calcitriol), the biologically active form.

Vitamin D receptors (VDRs) are expressed in multiple cardiovascular tissues, including:

- Cardiomyocytes
- Vascular smooth muscle cells
- Endothelial cells
- Immune cells

This widespread distribution suggests an important regulatory role beyond skeletal health.

Pathophysiological Links Between Vitamin D and Chronic Heart Failure

1. Regulation of the Renin–Angiotensin–Aldosterone System (RAAS)

Vitamin D suppresses renin gene expression. Deficiency leads to activation of the RAAS, resulting in:

- Increased vasoconstriction
- Sodium retention
- Ventricular hypertrophy
- Cardiac remodeling

These mechanisms contribute directly to heart failure progression.

2. Anti-Inflammatory Effects

Chronic heart failure is associated with systemic inflammation. Vitamin D modulates immune responses by reducing pro-inflammatory cytokines such as:



- Tumor necrosis factor- α (TNF- α)
- Interleukin-6 (IL-6)

Reduced inflammation may help prevent myocardial damage and fibrosis.

3. Effects on Myocardial Structure and Function

Vitamin D influences:

- Cardiomyocyte proliferation
- Calcium handling
- Myocardial contractility

Deficiency has been associated with left ventricular hypertrophy and impaired systolic function.

4. Endothelial Function and Vascular Health

Vitamin D improves endothelial function by enhancing nitric oxide production and reducing oxidative stress, thereby improving vascular compliance and peripheral circulation.

Vitamin D Deficiency in Patients with Chronic Heart Failure

Studies report vitamin D deficiency in up to 70–90% of CHF patients. Contributing factors include:

- Reduced sunlight exposure
- Advanced age
- Renal dysfunction
- Malnutrition
- Limited physical activity



Clinical Evidence on Vitamin D Supplementation

Clinical trials evaluating vitamin D supplementation in CHF patients have produced mixed results.

Some studies demonstrate:

- Improved inflammatory markers
- Enhanced left ventricular function
- Improved quality of life

However, large randomized controlled trials show inconsistent improvement in mortality or hospitalization outcomes. Differences in dosage, patient selection, baseline deficiency, and study duration may explain these discrepancies.

Current evidence suggests supplementation may be most beneficial in patients with confirmed deficiency rather than as universal therapy.

Conclusion

Vitamin D plays an important modulatory role in the pathophysiology of chronic heart failure through its effects on neurohormonal activation, inflammation, myocardial function, and vascular health. Although vitamin D deficiency is strongly associated with worse outcomes in CHF patients, evidence supporting routine supplementation for all patients remains inconclusive. Screening and targeted correction of deficiency may represent a promising supportive strategy in comprehensive heart failure management.

REFERENCES:

1. Braunwald E. Heart Disease: A Textbook of Cardiovascular Medicine. – 11th ed. – Philadelphia: Elsevier, 2019.



2. Холов, Х. А., Тешаев, О. Р., Бобошарипов, Ф. Г., Амонуллаев, А. Х., & Надирова, Ю. И. (2023). ОСТРЫЙ ПАНКРЕАТИТ-КАК НЕРЕШЕННАЯ ПРОБЛЕМА МЕДИЦИНЫ. *Академические исследования в современной науке*, 2(8), 192-206.

3. Бобошарипов, Ф. Г., Холов, Х. А., Тешаев, О. Р., & Надирова, Ю. И. (2023). ПОСТБАРИАТРИЧЕСКАЯ ГИПОГЛИКЕМИЯ И ГИПОТОНИЯ. *ОБРАЗОВАНИЕ НАУКА И ИННОВАЦИОННЫЕ ИДЕИ В МИРЕ*, 21(5), 105-113.

4. Надирова, Ю. И., Жаббаров, О. О., Бобошарипов, Ф. Г., Турсунова, Л. Д., & Мирзаева, Г. П. (2023). ОЦЕНКА ЭФФЕКТИВНОСТИ И ОПТИМИЗАЦИЯ ДЕЗАГГРЕГАНТНОЙ ТЕРАПИИ У БОЛЬНЫХ С ИБС.

5. Надирова, Ю. И., & Бобошарипов, Ф. Г. (2024). Клинико-диагностические аспекты раннего развития остеопороза при хронической сердечной недостаточности. In *International scientific-online conference*.

6. Bobosharipov, F. G., Ruxullayevich, T. O., Amonullayevich, X. X., & Isomovna, N. Y. (2024). GENETIC INFLUENCES FOR PEPTIC ULCER DISEASE ARE INDEPENDENT OF GENETIC FACTORS IMPORTANT FOR HP INFECTION.

7. Bobosharipov, F. G., Xolov, X. A., & Yu, N. (2024). ACUTE PANCREATITIS AFTER ELECTIVE LAPAROSCOPIC CHOLECYSTECTOMY: RETROSPECTIVE STUDY. In *Proceedings of Scientific Conference on Multidisciplinary Studies* (Vol. 3, No. 6, pp. 132-136).

8. Rakhmatov, A. M., & Zaripov, S. I. (2024). Gout and its association with gouty nephropathy: an analysis of 46 patients. *Современные подходы и новые исследования в современной науке*, 3(16), 100-102.



9. Jumanazarov, S., Jabborov, O., Qodirova, S., & Rahmatov, A. (2022). THE ROLE OF PODOCYTIC DYSFUNCTION IN THE PROGRESSION OF CHRONIC GLOMERULONEPHRITIS.

10. Rakhmatov, A. M., & Jabbarov, A. A. (2022). KodirovaSh. A., Jumanazarov SB, 140-141.

11. Султонов, П. И., Умарова, З. Ф., Жаббаров, О. О., Ходжанова, Ш. И., Кодирова, Ш. А., Жуманазаров, С. Б., & Рахматов, А. М. (2023). Антиагрегант Терапияни Сурункали Буйрак Касаллигида Буйрак Функционал Захирасига Таъсири.

12. Мирзаева, П. П., Жаббаров, О. О., Аликулов, И. Т., Бувамухамедова, Н. Х., & Рахматов, А. М. (2022). Особенности течения подагрического поражения почек у больных с ожирением.

13. Sulstonov, P. I., Umarova, Z. F., Jabbarov, O. O., Khodjanova, S. I., Jumanazarov, S. B., Rahmatov, A. M., & Rahimov, I. S. (2023). EFFECT OF ARTIAGREGANT THERAPY ON KIDNEY FUNCTIONAL RESOURCES IN CHRONIC DISEASE. *Theoretical aspects in the formation of pedagogical sciences*, 2(5), 137-138.

14. Po'latovna, R. G. (2025). USING INTERACTIVE METHODS IN TEACHING MEDICAL STUDENTS IN HIGHER EDUCATION. *FARS International Journal of Education, Social Science & Humanities.*, 13(11), 511-515.

15. Рахимова, Г. П. (2025, December). КАРДИОРЕНАЛЬНЫЙ СИНДРОМ: СОВРЕМЕННЫЕ ПРЕДСТАВЛЕНИЯ, МОЛЕКУЛЯРНО-ГЕНЕТИЧЕСКИЕ МЕХАНИЗМЫ И ВОЗМОЖНОСТИ ГЕНЕТИЧЕСКОЙ ДИАГНОСТИКИ. In *CONFERENCE ON GLOBAL RESEARCH PERSPECTIVES* (Vol. 1, No. 1, pp. 30-37).



16. Рахимова, Г. П. (2025). ХРОНИЧЕСКАЯ СЕРДЕЧНАЯ НЕДОСТАТОЧНОСТЬ: СОВРЕМЕННЫЕ ПРЕДСТАВЛЕНИЯ, ПАТОФИЗИОЛОГИЯ И ПОДХОДЫ К ЛЕЧЕНИЮ. *GLOBAL RESEARCH AND ACADEMIC INNOVATIONS*, 1(1), 160-165.

17. Жаббаров, О. О., Джуманиязова, З. Ф., & Рахимова, Г. П. (2022). Клинико-патогенетические аспекты кардиоренального синдрома.

18. Рахимова, Г. П. (2022). Особенности почечной гемодинамике при кардиоренального синдрома (Doctoral dissertation, Ташкент).

19. Po'latovna, R. G. (2025). SURUNKALI YURAK YETISHMOVCHILIGI: ZAMONAVIY TUSHUNCHALAR, PATOFIZIOLOGIYA VA DAVOLASH YONDASHUVLARI. *Ta'lim innovatsiyasi va integratsiyasi*, 58(3), 246-249.

20. Po'latovna, R. G. (2025). KARDIORENAL SINDROM: KLINIK, PATOGENETIK VA GENETIK JIHATLARNING PROGNOSTIK ANAMIYATI. *Modern education and development*, 39(2), 250-256.

21. Бобошарипов, Ф. Г., Холов, Х. А., Тешаев, О. Р., Алимов, С. У., & Надирова, Ю. И. (2023). КОМОРБИДНОЕ ТЕЧЕНИЕ ОСТРОГО ХОЛЕЦИСТИТА У БОЛЬНЫХ COVID-19. *Models and methods in modern science*, 2(4), 51-58.

22. Холов, Х. А., Тешаев, О. Р., Бобошарипов, Ф. Г., Амонуллаев, А. Х., & Надирова, Ю. И. (2023). ОСТРЫЙ ПАНКРЕАТИТ-КАК НЕРЕШЕННАЯ ПРОБЛЕМА МЕДИЦИНЫ. *Академические исследования в современной науке*, 2(8), 192-206.

23. Umarova, Z. F., Tursunova, L. D., Maksudova, M. X., Hodjanova, S. I., Mirzayeva, G. P., & Nadirova, Y. I. (2023). Diastolic dysfunction in patients with coronary artery disease late after coronary stenting (Doctoral dissertation).



In *International scientific-online conference International scientific-online conference*.

24. Бобошарипов, Ф. Г., Холов, Х. А., Тешаев, О. Р., & Надирова, Ю. И. (2023). ПОСТБАРИАТРИЧЕСКАЯ ГИПОГЛИКЕМИЯ И ГИПОТОНИЯ. *ОБРАЗОВАНИЕ НАУКА И ИННОВАЦИОННЫЕ ИДЕИ В МИРЕ*, 21(5), 105-113.

25. Надирова, Ю. И., Жаббаров, О. О., Бобошарипов, Ф. Г., Турсунова, Л. Д., & Мирзаева, Г. П. (2023). ОЦЕНКА ЭФФЕКТИВНОСТИ И ОПТИМИЗАЦИЯ ДЕЗАГГРЕГАНТНОЙ ТЕРАПИИ У БОЛЬНЫХ С ИБС.