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A PROHORMONE OF OUR ORGANISM

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Abstract
Vitamin D possesses both vitamin and hormone properties, with its primary function being the regulation of calcium-phosphorus metabolism and the maintenance of bone strength. Recent studies demonstrate its significant role in the immune system, endocrine and cardiovascular processes, as well as in the regulation of glucose metabolism. Vitamin D deficiency is recognized as a global public health issue, mainly caused by limited sun exposure, unbalanced diet, and chronic diseases. Deficiency leads to impaired bone mineralization and increases the risk of autoimmune, cardiovascular, and metabolic disorders. The article presents information on the functions of vitamin D, its sources, risk factors for deficiency, and preventive measures.
Keywords: vitamin D; calcium-phosphorus metabolism; bone health; immune system; prevention.

Introduction. For a long time, vitamin D was considered mainly in the context of maintaining mineral metabolism, bone health, and the prevention of rickets. It was often referred to as the "sunshine vitamin," emphasizing the key role of ultraviolet radiation in its synthesis. Currently, vitamin D is regarded not only as a vitamin but also as a prohormone capable of regulating a wide range of biological processes, including immune, endocrine, metabolic, and neuroprotective mechanisms [1]. Vitamin D belongs to the group of fat-soluble vitamins and includes several compounds, among which ergocalciferol (D_2) and cholecalciferol (D_3) are of the greatest importance. Vitamin D enters the body through two pathways: alimentary intake (with food and dietary supplements) and endogenous synthesis in the skin under the influence of ultraviolet B (UV-B) radiation, which distinguishes it from most other vitamins. In recent years, vitamin D deficiency has been recognized as one of the most significant global public health problems. Its insufficiency is associated not only with the development of rickets in children and osteoporosis in adults but also with an increased risk of cardiovascular, endocrine, and autoimmune diseases.

Metabolism of Vitamin D. The vitamin D group includes six sterols (vitamins D_1, D_2, D_3, D_4, D_5, and D_6). Two of them play a key role in the human body: vitamin D_2 (ergocalciferol) and vitamin D_3 (cholecalciferol) [2]. The metabolic pathways are common for both forms and include 25-hydroxylation to calcidiol (25(OH)D) by hepatic enzymes CYP2R1 and CYP27A1, followed by 1α-hydroxylation to the active metabolite 1,25-dihydroxyvitamin D (calcitriol, $1,25(OH)_2D$), catalyzed by the cytochrome P450–associated enzyme CYP27B1, which is present primarily in the kidneys but also in extra-renal tissues [3,4]. Vitamin D_2 is synthesized in plant cells from ergosterol. Its main dietary sources include fish, milk, bread, and mushrooms. After ingestion, vitamin D_2 is absorbed in the small intestine in the presence of bile, incorporated into chylomicrons, and transported via the lymphatic system into the venous circulation, subsequently undergoing metabolic steps similar to those of cholecalciferol. Adequate fat intake is required for normal absorption. Disorders of bile secretion significantly

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impair intestinal absorption of vitamin D [2]. Vitamin D_3 is synthesized in the Malpighian and basal layers of the epidermis from 7-dehydrocholesterol as a result of a non-enzymatic photolysis reaction induced by ultraviolet radiation with a wavelength of 290–315 nm. The activity of this process is directly proportional to radiation intensity and inversely proportional to skin pigmentation. In the epidermis, cholecalciferol binds to vitamin D–binding protein; approximately 70% enters the liver, while the remaining portion is deposited in adipose tissue, forming a vitamin D reserve [5]. After entering the bloodstream, both vitamin D_2 and D_3 are transported to the liver, where cytochrome P450 enzymes hydroxylate them to 25(OH)D. Subsequently, in the kidneys, CYP27B1 converts 25(OH)D into the biologically active form, $1,25(OH)_2D_3$ [6]. Unlike other metabolites, vitamin D_3 is the most biologically active form and binds to vitamin D receptors (VDRs), which are present in almost all organs and tissues. It has been shown that a single erythemal dose of sun exposure increases blood vitamin D_3 levels to the same extent as an oral intake of 10,000 IU of vitamin D_3 [5]. However, hypervitaminosis D does not occur with prolonged sun exposure due to regulatory mechanisms that limit excessive vitamin D entry into the bloodstream and promote its conversion into inactive compounds. With aging, the concentration of 7-dehydrocholesterol in the epidermis decreases, leading to a more than fourfold reduction in vitamin D_3 synthesis after the age of 65 years [7,8].

Biological Role of Vitamin D. Vitamin D occupies a unique position among biologically active compounds, as it combines the properties of a vitamin and a hormone-like substance exerting multifaceted effects on physiological processes.

1. Regulation of Calcium–Phosphorus Metabolism.

The active form of vitamin D, calcitriol [$1,25(OH)_2D$], enhances intestinal absorption of calcium and phosphorus, promotes their renal reabsorption, and regulates osteoblast and osteoclast activity, thereby ensuring normal bone growth and mineralization.

2. Support of the Musculoskeletal System

Adequate vitamin D levels prevent rickets in children and osteomalacia in adults, reduce the risk of osteoporosis and related fractures, and contribute to maintaining normal muscle tone.

3. Immunomodulatory Effects

Vitamin D supports immune function by enhancing both innate and adaptive immunity. It increases the expression of antimicrobial peptides such as cathelicidin and defensins through binding of 1,25-dihydroxyvitamin D to VDRs [9].

4. Effects on the Cardiovascular System

Vitamin D participates in blood pressure regulation via the renin–angiotensin–aldosterone system. The presence of VDRs in the endothelium and myocardium indicates its cardioprotective role, including improvement of endothelial function and reduction of atherosclerotic risk. Low vitamin D levels are associated with an increased risk of arterial hypertension, atherosclerosis, and heart failure [10].

5. Regulation of Endocrine and Metabolic Processes

Vitamin D is involved in carbohydrate and lipid metabolism. Its deficiency is associated with insulin resistance, type 2 diabetes mellitus, and metabolic syndrome. It also plays a role in parathyroid gland function and insulin secretion by pancreatic β-cells [11].

6. Role in Cancer Prevention

By regulating cell proliferation and differentiation, vitamin D can inhibit malignant cell growth, suppress angiogenesis, and reduce metastatic potential. It may lower the risk of colorectal, breast, and prostate cancers [12].

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7. Nervous System Function

Vitamin D contributes to nervous system health through its anti-inflammatory effects, promotion of neurotrophic factor synthesis, and positive influence on cognitive function. It reduces the risk of depressive disorders and neurodegenerative diseases [13].

Sources of Vitamin D.

The primary source of vitamin D is endogenous synthesis in the skin under UV-B radiation. Its efficiency depends on latitude, season, age, skin pigmentation, and lifestyle factors [14]. Exogenous sources include fatty fish (salmon, herring, mackerel), cod liver oil, egg yolk, and dairy products [15]. Pharmaceutical forms include vitamin D supplements and fortified foods containing ergocalciferol (D_2) or cholecalciferol (D_3) [16].

Vitamin D Deficiency: Current Data

Epidemiological studies indicate that vitamin D deficiency is widespread worldwide, regardless of geographic region. According to international criteria, serum 25(OH)D levels of 30–80 ng/mL are considered normal; 20–30 ng/mL indicate insufficiency; 10–20 ng/mL deficiency; and <10 ng/mL severe deficiency [17].

Conclusion

Vitamin D is one of the most important biologically active compounds, combining vitamin and hormone properties. Its role extends beyond calcium–phosphorus metabolism to include regulation of immune, endocrine, nervous, and cardiovascular systems. Vitamin D deficiency has systemic manifestations and is associated with an increased risk of chronic diseases. Therefore, timely diagnosis, prevention, and correction of vitamin D insufficiency are essential public health measures. Effective strategies include balanced nutrition, food fortification, adequate sun exposure, and targeted supplementation in high-risk groups. The development of comprehensive monitoring and correction programs may significantly reduce disease burden and improve population quality of life.

Risk Groups for Vitamin D Deficiency

According to WHO data, approximately 1 billion people worldwide suffer from vitamin D insufficiency or deficiency [26]. This condition is prevalent in both temperate climate countries and regions with high solar activity. Vitamin D deficiency remains a pressing public health issue and is observed across various population groups. The most vulnerable include:

1. Exclusively breastfed infants. This is attributed to the low vitamin D content in breast milk, particularly in cases of limited insolation (sun exposure) or dark skin pigmentation. The American Academy of Pediatrics recommends a prophylactic intake of 400 IU per day.
2. Young children (1–6 years old). A study conducted in China demonstrated that in the 3–6 age group, the risk of deficiency increases 11.5 times compared to infants (0–1 year), especially during the winter-spring period.
3. The elderly and sedentary patients (e.g., in nursing homes). Aging reduces the skin's capacity to synthesize vitamin D. For residents of elderly care facilities, the risk of deficiency is 2–3 times higher, particularly among immigrants from the Middle East.
4. Individuals with insufficient sun exposure. This group includes residents of northern latitudes, people who wear concealing clothing for cultural reasons, and office workers.
5. People with dark skin pigmentation. Elevated melanin levels reduce the skin's capacity to synthesize vitamin D. This is particularly significant for individuals of African, Caribbean, and South Asian descent.
6. Pregnant and lactating women. Increased physiological requirements, limited synthesis, and the risk of low intake make these groups especially vulnerable.

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7. Individuals with chronic diseases and malabsorption. This includes patients with liver, kidney, and gastrointestinal (GI) disorders (such as celiac disease and inflammatory bowel disease), which impair vitamin D metabolism.
8. Individuals with obesity. Excess adipose tissue sequesters fat-soluble vitamin D, thereby reducing its bioavailability.
9. Vegans and strict vegetarians. Limited dietary intake of vitamin D makes supplementation particularly important.
10. Regional specificities. For instance, in South Asian countries (Pakistan, India, Bangladesh, Nepal, Sri Lanka), vitamin D deficiency is prevalent in 48–73% of the adult population, highlighting the global nature of this problem [18].

Risk groups for vitamin D deficiency [19].

Musculoskeletal Disorders: Rickets, osteomalacia, osteoporosis, bone pain, bone deformities, fractures, and aseptic osteonecrosis.
Calcium and Phosphorus Metabolic Disorders: Hypo- and hypercalcemia, hypo- and hyperphosphatemia, calciuria, phosphaturia, hypo- and hyperphosphatasia.
Use of Medications: Corticosteroid therapy, ketoconazole, antiretroviral, and antiepileptic therapy.
Digestive Disorders: Digestive disorders, malabsorption, cystic fibrosis, and chronic inflammatory bowel disease (IBD).
Liver Diseases: Hepatic failure (liver failure), cholestasis, and fatty liver disease.
Kidney Diseases: Renal failure (kidney failure), post-transplant status, and nephrocalcinosis.
Endocrine Disorders: Hyper- and hypoparathyroidism, hyper- and hypothyroidism, type 1 diabetes mellitus, growth hormone deficiency, and polyglandular syndromes.
Developmental Disorders: Short stature, tall stature, obesity, and cachexia; delayed physical and psychomotor development.
Nervous System Diseases: Cerebral palsy, chronic immobilization, autism, multiple sclerosis, epilepsy, seizures of unknown etiology, myopathy, and muscular dystrophy.
Allergies: Asthma and atopic dermatitis.
Autoimmune Diseases: Collagen vascular diseases (connective tissue diseases), rheumatoid arthritis, type 1 diabetes mellitus, and Hashimoto's thyroiditis.
Immune System Disorders: Recurrent respiratory tract infections, asthma, and recurrent or chronic inflammatory conditions.
Neoplasms (Oncology): Leukemias, lymphoid neoplasms (neoplasms of the lymphatic system), tumors, and post-oncological treatment status.
Cardiovascular Diseases: Hypertension (arterial hypertension) and ischemic heart disease (coronary artery disease).

Metabolic Disorders: Type 2 diabetes, lipid metabolism disorders (dyslipidemia), obesity, and metabolic syndrome.

Causes of Vitamin D Deficiency in the Body

All causes of vitamin D deficiency in the body can be linked to several types of disturbances [20, 21].

Type I – Impaired Vitamin D Intake/Supply:

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• Insufficient insolation (UVB rays 280–315 nm) and reduced vitamin D synthesis in the skin (use of sunscreens, UV-protective creams, skin covered by clothing, living in high latitudes, especially during winter months, high levels of atmospheric pollution, dense cloud cover);
• Dietary insufficiency of vitamin D-containing foods (ocean fish being the most crucial dietary factor affecting serum 25(OH)D concentration, to a lesser extent – animal fat and meat) [20];
• Absence of vitamin D supplementation.
Type II – Impaired Absorption and Utilization of Vitamin D:
• Gastrointestinal disorders (celiac disease, Crohn's disease, cystic fibrosis);
• Low-fat diets;
• Skin type (the natural skin tone determines its sensitivity to sunlight and the rate of vitamin D synthesis/absorption);
• Impaired liver or kidney function (Hepatic or renal dysfunction);
• Overweight and obesity;
• Magnesium deficiency in the body;
• Age (by the age of 70, the body's capacity to synthesize vitamin D decreases by nearly 70%);
• Intake of certain medications.
Type III – Impaired Vitamin D Metabolism:
• Reduced synthesis of 25(OH)D in liver diseases;
Type III – Impaired Vitamin D Metabolism (continued):
• Reduced production of 1,25(OH)2D3 (in kidney diseases – Chronic Kidney Disease (CKD), hypoparathyroidism, sex hormone deficiency, under the influence of glucocorticosteroids and antiepileptic drugs);
• Decreased activity of vitamin D receptors (VDRs) in tissues during vitamin D deficiency;
• Increased excretion of 25(OH)D and vitamin D-binding protein (DBP).
Type IV – Development of Resistance to 1α,25(OH)2D3 (primarily age-related (> 65 years) and caused by a reduction in the number of VDRs in target tissues, especially in the intestine, kidneys, and skeletal muscles) [22].
Clinical Manifestations of Vitamin D Deficiency
1. Musculoskeletal Manifestations:
• In children: Rickets (impaired bone mineralization); delayed growth and physical development; skeletal deformities (bowing of legs, chest, skull); delayed tooth eruption, enamel defects.
• In adults: Osteomalacia (bone softening, bone and muscle pain); osteoporosis, increased risk of fractures; chronic muscle weakness, difficulties with standing and walking.
2. Neurological and Muscular Manifestations:
• In adults: Muscle cramps and paresthesia; increased fatigue, and reduced work capacity.
• In children: Delayed motor development (delayed sitting and walking).
3. Immune System Disorders: Increased susceptibility to infections (ARVI/Acute Respiratory Viral Infections, bronchitis, pneumonia); predisposition to chronic inflammatory diseases; increased risk of autoimmune diseases (Type 1 diabetes, multiple sclerosis).
4. Cardiovascular Manifestations: Increased arterial blood pressure (hypertension); predisposition to atherosclerosis; impaired myocardial function.
5. Endocrine and Metabolic Disorders: Reduced tissue sensitivity to insulin (risk of Type 2 diabetes); calcium and phosphorus metabolic disorders, secondary hyperparathyroidism; obesity and metabolic syndrome.

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6. In Pregnant Women and Neonates:
- In women: Increased risk of preeclampsia, gestational diabetes, and preterm birth.
- In neonates: Hypocalcemia, seizures, and low birth weight.

Prevention of Vitamin D Deficiency

Prevention of vitamin D deficiency involves a comprehensive approach: dietary adjustment, lifestyle optimization, and, if necessary, pharmacological supplementation.

1. Dietary Intake

Regular consumption of foods rich in vitamin D is recommended, including fatty fish species (salmon, mackerel, herring), cod liver, egg yolks, dairy products, and foods artificially fortified with vitamin D (such as milk, bread, and yogurt).

2. Ultraviolet Radiation Exposure

Regular outdoor activity during daylight hours promotes the endogenous synthesis of vitamin D. For individuals with a fair skin phototype, 15–20 minutes of sun exposure several times a week with the face and hands exposed is sufficient.

3. Lifestyle

Maintaining a healthy body weight, engaging in regular physical activity, and monitoring the functional status of organs involved in vitamin D metabolism (liver, kidneys, and intestines).

4. Medical Prophylaxis

According to current guidelines, children over one year of age are recommended a daily intake of at least 600 IU of vitamin D, while for adults, the recommended dose is approximately 800 IU. Individuals in high-risk groups (the elderly, pregnant women, patients with chronic liver and kidney diseases, and those with limited sun exposure) may require higher dosages, adjusted on an individual basis [23]. The daily requirement of vitamin D depends on an individual's age. Current recommended levels are as follows [26]

Daily dose of vitamin D by age.

Age Group	Daily dose, IU	Maximum permitted quantity (thousand), IU
Children up to 6 months	400	1
Toddlers 7 months to 1 year	400	1,5
Children 1-8 years old	600	2,5 - 3
Teenagers 9-17 years old	600	4
Adults 18-70 years old	600	4
Seniors over 71 years old	800	4
Pregnancy and breastfeeding	800	4

5. Screening and individualized approach.

If there is a family history of osteoporosis, hypovitaminosis D, or serum 25(OH)D levels, a specialist consultation is recommended to select the optimal preventive or therapeutic strategy. [24]

Conclusion

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References.

1. Bouillon R., Marcocci C., Carmeliet G., Bikle D., White J.H., Dawson-Hughes B., Lips P., Munns C.F., Lazaretti-Castro M., Giustina A., et al. Skeletal and Extraskeletal Actions of Vitamin D: Current Evidence and Outstanding Questions. Endocr. Rev. 2019; 40:1109–1151. doi: 10.1210/er.2018-00126.
2. Громова О. А. Торшин И.Ю., Пронин А. В. Особенности фармакологии водорастворимой формы витамина D на основе мицелл.
3. Пигарова Е. А., Мазурина Н.В., Трошина Е. А. Витамин D в профилактике костных и метаболических нарушений // Consilium Medicum. - 2019. - Т. 21. - №4. - С. 84-90. https://doi.org/10.26442/20751753.2019.4.190342
4. Saponaro F, Saba A, Zucchi R. An Update on Vitamin D Metabolism. Int J Mol Sci. 2020;21(18):6573.
5. Holick M.F. Vitamin D deficiency // N Engl J Med. 2007; 357:266-81.
6. Hossein-nezhad A., Holick M.F. Vitamin D for health: a global perspective // Mayo Clin Proc. — 2013.
7. Риггз Б.Л., Мелтон Л.Дж. Остеопороз. Этиология, диагностика, лечение. Пер с англ. — М. — СПб: Издательство БИНОМ: «Невский диалект», 2000. — 560 с.
8. Шварц Г. Я. Витамин D и D-гормон. — М.: Анахарсис, 2005. — 152 с.
9. Гомбарт А.Ф., Боррегаард Н., Кеффлер Х. Ген антимикробного пептида (CAMP) кателицидина человека является прямой мишенью рецептора витамина D и сильно активируется в миелоидных клетках 1,25-дигидроксивитамином D3. FASEB J. 2005; 19:1067–1077.
10. Вималаванса С.Д. Витамин D и сердечно-сосудистые заболевания: причинно-следственная связь. J. Steroid Biochem. Mol. Biol. 2018; 175:29–43.
11. Argano C, Mirarchi L, Amodeo S, et al. The role of Vitamin D and its molecular bases in insulin resistance, diabetes, metabolic syndrome, and cardiovascular disease: state of the art. Int. J. Mol. Sci. 2023;24(20):15485.
12. Лаппе Дж., Гарланд К., Горхэм Э. Добавки витамина D и риск развития рака. ДЖАМА. 2017; 318:299–300.
13. Ши М.К., Баргер К., Доусон-Хьюз Б., Леурганс С.Е., Фу Х., Джеймс Б.Д., Холланд Т.М., Агарвал., Ван Дж., Матушек Г., и др. Формы витамина D в мозге, снижение когнитивных функций и невропатология у пожилых людей, проживающих в сообществе. Деменция Альцгеймера. 2023; 19:2389–2396.
14. Holick M. F. Vitamin D deficiency. N Engl J Med. 2020.
15. Pilz S, Trummer C, Pandis M, et al. Vitamin D: current guidelines and future outlook. Nutrients. 2018;10(5):546.

Western European Journal of Medicine and Medical Science
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https://westerneuropeanstudies.com/index.php/3
ISSN (E): 2942-1918 Open Access| Peer Reviewed

16. Bouillon R, Marcocci C, Carmeliet G, et al. Skeletal and extra-skeletal actions of vitamin D: Current evidence and outstanding questions. Endocr Rev. 2022;43(2):110–190.
17. Захарова. И.Н., Мальцев С. В., Боровик Т.Э. Недостаточность витамина D у детей раннего возраста в России (результаты многоцентрового исследования — зима 2013–2014 гг.) // Педиатрия им. Г. Н. Сперанского. — 2014. — Т. 93, № 2. — С. 75–80.
18. Шестерикова В. В., Иванова И. Е., Стержанова Н.В. Современный взгляд на витамин D //Лечение и профилактика. 2014. С.57-61.
19. Orwoll E., Nielson C.M., Marshall L.M. et al. Vitamin D deficiency in older men // J Clin Endocrinol Metab. — 2009. — Vol. 94. — P. 1214–1222.
20. Мальцев С. В., Архипова Н. Н., Шакирова Э.М. Витамин D, кальций и фосфаты у здоровых детей и при патологии. — Казань, 2012. — 120 с.
21. Schwartz J.B., Lai J., Lizaola B., Kane L., Markova S., Weyland P., Terrault N.A., Stotland N., Bikle D. A Comparison of Measured and Calculated Free 25(OH) Vitamin D Levels in Clinical Populations // The Journal of Clinical Endocrinology & Metabolism. — 2014, May 1. —Vol. 99 (5). — P. 1631-1637.
22. Oleg Tsuprykov, Xin Chen, Carl-Friedrich Hocher et al. Why should we measure free 25(OH) vitamin D? // The Journal of Steroid Biochemistry and Molecular Biology. — 2018, June. — Vol. 180. —P. 87–104.
23. Bouillon R., Manousaki D., Rosen C. et al. The health effects of vitamin D supplementation: evidence from human studies. Nat Rev Endocrinol. 2022; 18:96–110.
24. World Health Organization. Vitamin D deficiency: global status report. Geneva: WHO; 2020.
25. Pigarova E.A., Dzeranova L.K., Yatsenko D.A. Absorption and metabolism of vitamin D in health and in gastrointestinal tract diseases. Obesity and metabolism. 2022;19(1):123-133.
26. https://www.vympel-medcenter.ru/about/articles/defitsit_vitamina_d/