AMERICAN ACADEMIC PUBLISHER

INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

ISSN NUMBER: 2692 - 5206 Volume 6. No 05. MAY ,2026

VITAMIN D DEFICIENCY CAUSES, EFFECTS, AND TREATMENT

ROHAN SINGH
medical student, Samarkand state medical university, Uzbekistan
SOHAIL RAJ
medical student, Samarkand state medical university, Uzbekistan
NELIX
medical student, Samarkand state medical university, Uzbekistan
DUKHIMA
medical student, Samarkand state medical university , Uzbekistan
https://doi.org/10.5281/zenodo.20032901

Introduction

Vitamin D, often referred to as the "sunshine vitamin," is a fat-soluble secosteroid that plays a critical role in numerous physiological processes. Unlike most vitamins, the body is capable of synthesizing vitamin D endogenously through sun exposure. Nevertheless, deficiency remains one of the most prevalent nutritional disorders in the modern world.

Causes of Vitamin D Deficiency

Insufficient Sun Exposure

The skin synthesizes vitamin D3 (cholecalciferol) upon exposure to ultraviolet B (UVB) radiation (290–315 nm). Factors that reduce effective sun exposure include:

- Geographic latitude above 35°N or below 35°S, where UVB intensity is insufficient for synthesis during winter months
- Excessive use of sunscreen (SPF 30 blocks approximately 95–98% of UVB radiation)
- Indoor lifestyles, particularly in urban environments and developed nations
- Dark skin pigmentation — higher melanin content requires 3–5 times more sun exposure to synthesize equivalent vitamin D
- Institutional living, such as nursing homes or hospitals

Dietary Insufficiency

Very few foods naturally contain significant amounts of vitamin D. The primary dietary sources include fatty fish (salmon, mackerel, sardines), egg yolks, and UV-exposed mushrooms. Fortified foods — such as dairy products and cereals — partially compensate for dietary gaps in some countries, but global fortification policies are inconsistent.

Malabsorption Syndromes

Since vitamin D is a fat-soluble vitamin, conditions that impair fat absorption significantly reduce its uptake from the gastrointestinal tract. These include Crohn's disease, celiac disease, cystic fibrosis, and following bariatric surgery. Patients with these conditions represent a high-risk group requiring aggressive monitoring and supplementation.

Chronic Kidney and Liver Disease

Vitamin D metabolism requires hydroxylation steps in both the liver (25-hydroxylation) and kidneys (1α-hydroxylation). Chronic liver disease impairs the first step, while chronic kidney disease (CKD) impairs the final conversion to active calcitriol. Patients with CKD stage 3 and above frequently require supplementation with activated vitamin D analogs.

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 6. No 05. MAY ,2026

Medications

Several commonly used medications accelerate the catabolism of vitamin D, including anticonvulsants (phenytoin, carbamazepine), glucocorticoids, antiretrovirals, and certain antifungals. Long-term users of these medications should be regularly screened for vitamin D deficiency.

Obesity

Adipose tissue sequesters fat-soluble vitamins, including vitamin D. This volumetric dilution reduces bioavailability, meaning that obese individuals may require 2–3 times higher doses of supplementation to achieve adequate serum levels compared to normal-weight individuals.

Effects of Vitamin D Deficiency

Skeletal and Musculoskeletal Effects

The classical manifestations of vitamin D deficiency on the skeleton remain among its most welldocumented consequences:

• Rickets: A disease of impaired skeletal mineralization in children, characterized by bowed legs, delayed fontanelle closure, and widened growth plates

• Osteomalacia: The adult equivalent — soft bones resulting from defective mineralization — presenting with diffuse bone pain, proximal muscle weakness, and fractures

• Osteoporosis: Chronic deficiency contributes to low bone mineral density and increased fracture risk, particularly in postmenopausal women and older adults

Immune System Dysfunction

Vitamin D plays a pivotal immunomodulatory role. VDRs are expressed on T cells, B cells, and macrophages. Deficiency is associated with increased susceptibility to infections — most notably respiratory infections including tuberculosis and influenza — as well as dysregulation of autoimmune responses. Emerging data suggest associations with multiple sclerosis, rheumatoid arthritis, type 1 diabetes, and inflammatory bowel disease.

Cardiovascular Disease

Observational studies have consistently demonstrated an inverse relationship between serum 25(OH)D levels and cardiovascular risk. Mechanistic pathways include vitamin D's role in regulating the renin-angiotensin-aldosterone system, endothelial function, and vascular smooth muscle tone. Deficiency is associated with hypertension, heart failure, and myocardial infarction.

Mental Health and Neurological Effects

Brain tissue expresses VDRs, and vitamin D influences neurotransmitter synthesis including serotonin and dopamine. Deficiency has been linked to depression, seasonal affective disorder (SAD), cognitive decline in older adults, and an increased risk of schizophrenia. Prenatal vitamin D deficiency has been associated with neurodevelopmental disorders in children.

Diagnosis

Vitamin D status is assessed by measuring serum 25-hydroxyvitamin D [25(OH)D], the major circulating form and the best clinical indicator of overall vitamin D status. The following classifications are widely used:

Serum 25(OH)D Level	Classification	Clinical Significance
< 12 ng/mL (< 30 nmol/L)	Severe Deficiency	Rickets, osteomalacia risk
12–20 ng/mL (30–50 nmol/L)	Deficiency	Bone and muscle disease risk

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 6. No 05. MAY ,2026

20–30 ng/mL (50–75 nmol/L)	Insufficiency	Suboptimal for most functions
30–60 ng/mL (75–150 nmol/L)	Sufficiency	Optimal physiological range
> 100 ng/mL (> 250 nmol/L)	Potential Toxicity	Hypercalcemia risk

Treatment and Prevention

Supplementation

Oral vitamin D3 (cholecalciferol) is the preferred form of supplementation due to its greater efficacy in raising serum 25(OH)D levels compared to vitamin D2 (ergocalciferol). Recommended daily doses vary by guideline body and patient population:

- Healthy adults (18–70 years): 600–1500 IU/day (Endocrine Society: up to 2000 IU/day)
- Adults > 70 years: 800–2000 IU/day
- At-risk individuals (obese, malabsorption, limited sun exposure): 2000–4000 IU/day
- Therapeutic repletion in deficiency: 50,000 IU weekly for 8–12 weeks, then maintenance

Sun Exposure

Sensible sun exposure (10–30 minutes of midday sun on arms and legs, several times weekly) can maintain adequate vitamin D levels in fair-skinned individuals. However, risks of UV-induced skin damage and skin cancer require balancing sun exposure recommendations individually, particularly for those with personal or family histories of skin malignancy.

Dietary Approaches

While diet alone rarely achieves sufficiency in the absence of supplementation, increasing consumption of vitamin D–rich foods is beneficial. Recommended dietary sources include fatty fish, UV-treated mushrooms, egg yolks, and fortified dairy or plant-based milk alternatives.

Special Populations

Certain groups require heightened vigilance:

- Pregnant and lactating women: Deficiency during pregnancy is linked to preeclampsia, gestational diabetes, preterm birth, and neonatal rickets. Supplementation of 1500–2000 IU/day is recommended
- Infants and children: Exclusively breastfed infants should receive 400 IU/day from birth, as breast milk is a poor vitamin D source
- Elderly individuals: Reduced cutaneous synthesis, decreased mobility, and impaired renal activation collectively elevate risk
- Patients with chronic disease: Those with CKD, inflammatory bowel disease, or on corticosteroids require individualized management

Conclusion

Vitamin D deficiency is a pervasive and underrecognized public health challenge with ramifications that extend far beyond classical bone disease. Its causes are multifactorial — encompassing lifestyle, geography, diet, and comorbid conditions — and its effects touch virtually every organ system. Early identification through targeted screening of at-risk populations, combined with appropriate supplementation and dietary optimization, offers a safe, cost-effective intervention with substantial potential to reduce global disease burden.

AMERICAN ACADEMIC PUBLISHER

INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

ISSN NUMBER: 2692 - 5206 Volume 6. No 05. MAY ,2026

References:

1. Holick, M.F. (2007). Vitamin D deficiency. New England Journal of Medicine, 357(3), 266–281.
2. https://doi.org/10.1056/NEJMra070553
3. Holick, M.F., Binkley, N.C., Bischoff-Ferrari, H.A., et al. (2011). Evaluation, treatment, and prevention of vitamin D deficiency: An Endocrine Society clinical practice guideline. Journal of Clinical Endocrinology & Metabolism, 96(7), 1911–1930.
4. Bishop, N.J., Fewtrell, M.S., & Maclennan, A.H. (2019). Rickets prevention and management. Archives of Disease in Childhood, 104(7), 620–629.
5. Willett, W.C., & Ludwig, D.S. (2020). Milk and health. New England Journal of Medicine, 382, 644–654.
6. Garland, C.F., Gorham, E.D., Mohr, S.B., & Garland, F.C. (2009). Vitamin D for cancer prevention: Global perspective. Annals of Epidemiology, 19(7), 468–483.
7. Bouillon, R., Marcocci, C., Carmeliet, G., et al. (2019). Skeletal and Extraskeletal Actions of Vitamin D: Current Evidence and Outstanding Questions. Endocrine Reviews, 40(4), 1109–1151.
8. Płudowski, P., Holick, M.F., Grant, W.B., et al. (2018). Vitamin D supplementation guidelines. Journal of Steroid Biochemistry and Molecular Biology, 175, 125–135.
9. Roth, D.E., Abrams, S.A., Aloia, J., et al. (2018). Global prevalence and disease burden of vitamin D deficiency: a roadmap for action in low- and middle-income countries. Annals of the New York Academy of Sciences, 1430(1)