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Vitamin D and human health – A multisystem action based on a literature review

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ABSTRACT

Vitamin D helps keep calcium and phosphate levels stable and supports healthy bone formation. It also affects many other systems in the body. Vitamin D receptors and the enzymes that process this vitamin are found in many types of cells, which shows how important it is. Research suggests that low levels of vitamin D increase the risk of osteoporosis, fractures, respiratory infections, cardiovascular problems, neurodegenerative diseases and autoimmune disorders. Supplementation is especially helpful for people with clearly reduced serum levels. Vitamin D influences the skeletal, immune and nervous systems, and maintaining its optimal level may lower the risk of several chronic conditions and improve some metabolic markers. More high-quality studies are still needed to determine the best supplementation dose and to identify who benefits from vitamin D the most.

Keywords: vitamin D, immune system, cardiovascular system, nervous system and autoimmune diseases.

1. INTRODUCTION

Vitamin D's best-known role is to regulate calcium and phosphate balance. This process involves maintaining a balance between intestinal absorption, renal reabsorption and the storage of these minerals in bone tissue. Consequently, vitamin D plays a crucial role in ensuring proper bone mineralization. It keeps the skeleton strong and prevents problems with bone mineralization, such as osteomalacia and rickets. However, it is now clear that bone mineral density is not the only target of vitamin D's biological action. Vitamin D also participates in many other processes, including the regulation of immune responses, cardiovascular and nervous system functions. The vitamin D also influences the modulation of inflammatory and proliferative pathways (Charoenngam & Holick, 2020).

Molecular and immunological research has shown that many immune cells express the vitamin D receptor (VDR). These include T and B lymphocytes, monocytes, macrophages and dendritic cells. They also produce enzymes involved in local vitamin D metabolism, such as 1 α -hydroxylase. This indicates that immune

system cells can also activate vitamin D. It allows it to affect other cells in a paracrine or autocrine manner. Vitamin D acts locally and helps regulate the immune system's response. This indicates its vital role in the immune system, helping to regulate the body's response to pathogens and other harmful agents. Vitamin D receptors are found in many tissues, so the vitamin D response is not limited to the immune system.

Vitamin D affects many tissues, including endothelial cells, vascular smooth muscle cells and neurons. It also plays a role in the metabolism of skeletal muscles and adipose tissue. By acting in many systems, vitamin D helps maintain the proper functioning of the body. Low levels increase the risk of chronic diseases. Information collected in epidemiological and clinical studies indicates that low serum concentrations of Calcitriol increase the risk of several disorders. These include autoimmune and rheumatologic diseases, cardiovascular conditions, respiratory infections and neurological or psychiatric abnormalities (Latic & Erben, 2020; Martineau et al., 2017).

Because of its broad range of actions, vitamin D is considered as a hormone that influences numerous organs and systems throughout the body. This review shows what we know about vitamin D and its role in the human body. The focus is on its impact on the skeletal, immune, cardiovascular and nervous systems. The review also discusses how vitamin D may influence autoimmune diseases and disorders of carbohydrate metabolism.

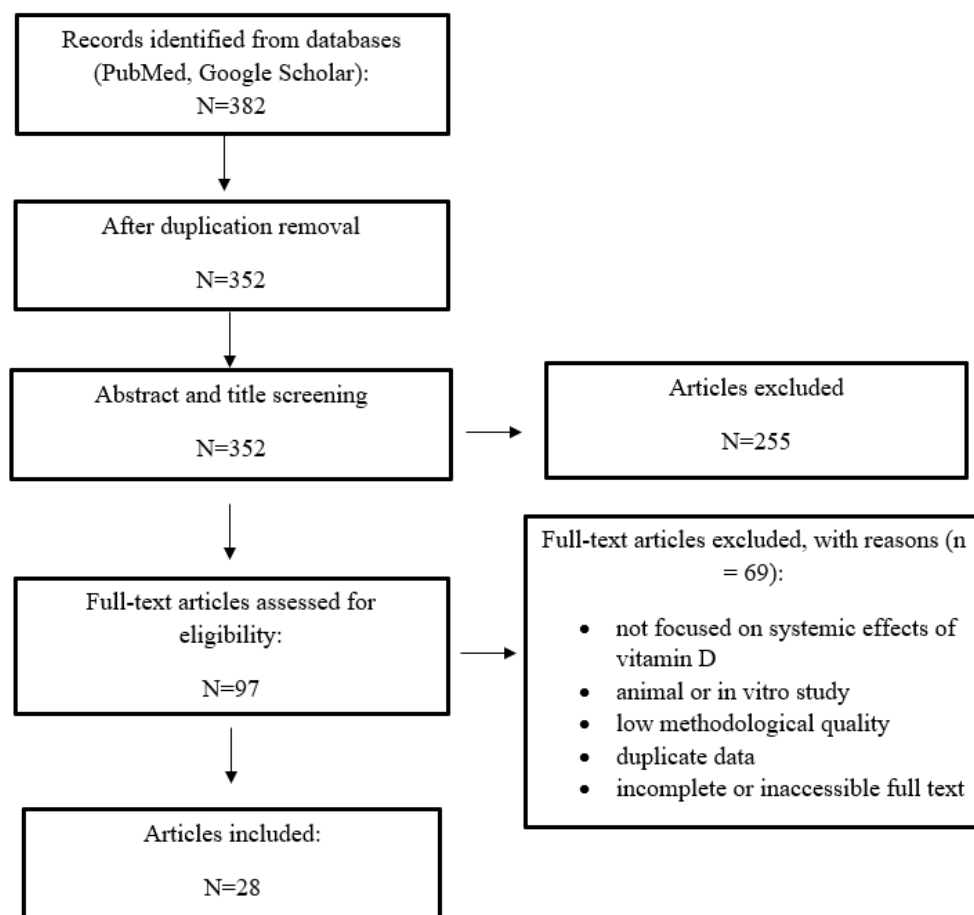


Figure 1. PRISMA flow diagram of the study selection process

2. REVIEW METHODS

We used the PRISMA guidelines to make the review clear and easy to follow. We searched PubMed and Google Scholar. We focused on studies that described the systemic effects of vitamin D in humans. The search took place from March to September 2025. We used the following key terms in different combinations: vitamin D, immune system, cardiovascular system, nervous system and autoimmune diseases. Figure 1 (PRISMA flow diagram) shows the selection process.

3. RESULTS & DISCUSSION

Vitamin D: Key Regulator of Bones and Muscles

Vitamin D regulates calcium and phosphate levels. It plays fundamental role in the proper function of the musculoskeletal system. It is essential for both normal skeletal development during the growth period and for maintaining appropriate skeletal integrity and stability in adulthood. Calcitriol - the active form of vitamin D acts as a steroid hormone that binds to the vitamin D receptor (VDR). This interaction controls the expression of genes involved in calcium and phosphate transport and metabolism. As a result, calcitriol increases the intestinal absorption of these minerals, supports bone matrix mineralization and helps regulate bone formation and remodeling. This mechanism is crucial during growth and remains essential for maintaining the balance between bone formation and resorption in adults.

When children lack vitamin D, calcium, or phosphate, their body balance is disturbed. This affects bone mineralization and can slow normal skeletal growth. Low calcium levels (hypocalcemia) may develop. This triggers the parathyroid glands to release more hormone. As a result, secondary hyperparathyroidism occurs and calcium is absorbed from the bones. If this condition persists, blood phosphate levels fall and the growth cartilage fails to harden properly. As a result, bone mineralization is disturbed, leading to skeletal deformities typical of rickets (Glorieux & Pettifor, 2014).

Rickets is a classic outcome of long-term vitamin D deficiency during growth. However, it still occurs today. It is most noticeable among individuals with limited sunlight exposure or breastfed infants who lack proper supplementation. First of all, the primary cause of vitamin D deficiency is insufficient exposure to UVB rays. This results in insufficient production of the active form of vitamin D in the skin and its deficiency. The time required for effective synthesis varies among ethnic groups because skin pigmentation affects the amount of UV radiation absorbed. Other factors, such as cold climates, clothing that covers most of the body, extensive burns and gastrointestinal disorders that impair absorption, also lower vitamin D levels. Premature babies and babies of mothers with low vitamin D often lack this vitamin. Breastfed infants without supplements also have a higher risk. Obesity adds risk because fat traps vitamin D.

Chronic kidney disease also lowers its activation. For these reasons, doctors recommend vitamin D supplements for pregnant women, infants and children. The dose depends on age, weight and season. In adults, the risk factors remain similar, but vitamin D deficiency leads to different clinical consequences. Decreased serum concentrations of 25(OH)D result in disturbances of bone metabolism. It weakens the trabecular architecture and consequently an increased risk of fractures, exceptionally in the spine and femoral neck. The largest groups at risk include older adults and postmenopausal women (Wimalawansa, 2024).

In women, lower estrogen levels after menopause accelerate bone loss. Clinical evidence shows that vitamin D with calcium lowers fracture risk in osteoporosis. A meta-analysis by Bischoff-Ferrari et al. found that taking 700–800 IU of vitamin D daily reduced hip fracture risk by 26% and other fractures by 23%. People with low vitamin D levels saw the most significant benefit (Sanders et al., 2010; Bischoff-Ferrari et al., 2005).

In summary, vitamin D supplementation provides the greatest benefits to people who have a confirmed deficiency. Maintaining adequate levels of vitamin D is crucial for maintaining a healthy musculoskeletal system. Optimal vitamin D concentrations in the blood reduce the risk of bone loss and fractures. They also have a beneficial effect on osteoporosis.

Vitamin D in Immune Defense and Respiratory Health

Vitamin D supports normal immune function, which can influence the risk of acute respiratory infections (ARI). Monocytes, macrophages and T and B lymphocytes — contain the vitamin D receptor (VDR) and the enzymes needed for its activation. Calcitriol, boosts innate immunity. It is essential to make antimicrobial peptides like cathelicidin (LL-37) and defensins. These peptides remove pathogens from the respiratory lining. Vitamin D also influences the adaptive functions of the immune system. It reduces levels of proinflammatory cytokines and limits Th1 function. Vitamin D also affects Treg cells, which control inflammation and protect cells. Research indicates that vitamin D supplementation may reduce the risk of respiratory infections (ARIs) and other infections. A meta-analysis by Martineau et al., (2017), which examined approximately 11,000 participants across 25 trials, found a 12% reduction in ARI risk among individuals who supplemented with vitamin D. The effect was most pronounced among individuals with severe deficiency. Another extensive review by Jolliffe et al., (2021), which included over 48,000 participants, confirmed that regular daily or weekly doses—not occasional high ones—reduced the risk of ARI, especially in those with low vitamin D levels (Pilz et al., 2012).

In summary, vitamin D shows broad range of effects. It stimulates antimicrobial peptides and regulates cytokines and the Th/Treg balance. Vitamin D helps to maintain mucosal and systemic immunity. This suggests that supplementing may reduce infection risk in those with deficiency.

Vitamin D: Implications for Cardiovascular Function

After examining the scientific research on vitamin D, we can conclude that it also fundamentally affects the cardiovascular system. Epithelial cells, smooth muscle cells and cardiac muscle cells all contain vitamin D receptors (VDRs). This allows vitamin D to act directly on these tissues (Andrukova et al., 2014). Calcitriol helps control blood pressure. It lowers Renin–Angiotensin–Aldosterone System activity, reduces inflammation and oxidative stress and supports endothelial function (Haider et al., 2023).

People with low vitamin D are more likely to develop heart and blood vessel problems. They have higher risks of high blood pressure, atherosclerosis, heart attack and stroke (Rejnmark et al., 2017). Reviews of several clinical trials also suggest that vitamin D deficiency, mainly below (<20 ng/mL) is related with a higher risk of these events. It is still unclear whether low vitamin D levels cause these disorders or only reflect them (Manson et al., 2019).

In contrast, data from randomized controlled trials (RCTs) are inconclusive. Most extensive trials, including VITAL and ViDA, did not demonstrate a significant reduction in cardiovascular events following vitamin D supplementation in general populations. Many analyses show that vitamin D supplements may help people with severe deficiency. They may also benefit obese individuals and patients taking cardiovascular drugs, especially statins. Interest in a personalized approach to vitamin D supplementation in the prevention of cardiovascular disease is still growing. Checking vitamin D levels, risk factors and health conditions can help target treatment. It shows who will benefit most. Several experimental studies have shown that vitamin D may influence cardiovascular health. Observational findings point in the same direction. Evidence is still not strong enough to recommend vitamin D supplementation for primary prevention of cardiovascular disease. Further studies are needed, to determine these issues. Especially those focused on high-risk groups and on how individuals differ in their response to supplementation (Scragg et al., 2017; Barbarawi et al., 2019).

Vitamin D: Effects on Neural Development and Function

The nervous system depends on vitamin D for proper function. Many brain cells have vitamin D receptors (VDR) and the enzyme 1 α -hydroxylase. These cells include neurons, astrocytes, oligodendrocytes and microglia. They can convert vitamin D into its active form, 1,25(OH)₂D. The active form then works locally in the brain (Sangha et al., 2023).

Vitamin D supports many brain functions. It helps oligodendrocytes grow and mature. It also increases neurotrophin production. Vitamin D reduces oxidative stress and limits neurodegeneration and inflammation. In addition, vitamin D may help maintain the integrity of the blood–brain barrier (Cui & Eyles, 2022).

Among the described mechanisms, its relationship with the pathogenesis of multiple sclerosis (MS) has been particularly well described. This makes MS a helpful model for illustrating the neuromodulatory and neuroprotective roles of vitamin D (Shirazi et al., 2015). MS affects the nervous system through several mechanisms. It causes damage to oligodendrocytes, which leads to axonal demyelination. Although remyelination may occur—particularly in early stages of the disease—it progressively declines over time. Oligodendrocyte regeneration depends on neural stem cells (NSCs) and oligodendrocyte progenitor cells (OPCs). These cells like mature oligodendrocytes, express vitamin D receptors (VDR).

Vitamin D receptor- retinoid X receptor signaling helps oligodendrocyte progenitor cells mature into oligodendrocytes. The active form of vitamin D makes neural stem cells grow and guides them toward the oligodendrocyte lineage. It also supports oligodendrocyte progenitor cells (OPCs) growth. Studies in cell cultures and animal models show that vitamin D leads to more (OPCs), more oligodendrocytes and higher levels of myelin proteins like MBP and PLP. This suggests less myelin damage and better myelin repair (Gomez-Pinedo et al., 2020). Beyond its role in the oligodendroglial lineage, vitamin D also affects key mechanisms in multiple sclerosis (MS). It modulates the immune system by inhibiting Th1 and Th17 activity and promoting Treg differentiation. Vitamin D reduces dendritic cell maturation and antigen presentation and alters B-cell function. It strengthens the blood–brain barrier by blocking VDR–NF- κ B and lowering Matrix Metalloproteinase 9(MMP-9) activity. At the same time, it preserves tight junction proteins such as Zonula Occludens-1 (ZO-1), claudin-5 and occludin. Vitamin D also limits microglial and astrocytic activation, promoting anti-inflammatory cell states. In addition, it may increase neurotrophic factors, including Brain-Derived Neurotrophic Factor (BDNF), Glial cell line-Derived Neurotrophic Factor (GDNF), Neurotrophin-3 (NT-3) and Neurotrophin-4 (NT-4), as well as other neuroprotective molecules.

It can help remyelination. It causes increased Neural Stem Cells (NSC) proliferation and Oligodendrocyte Precursor Cells (OPC) differentiation. It also raises myelin proteins MBP and PLP (Bscheider & Butcher, 2016; Won et al., 2015).

In summary, mechanistic and observational data suggest an essential role of vitamin D in MS.

However, most randomized trials in patients with relapsing–remitting MS (RRMS) have shown inconclusive or negative results. Further well-designed RCTs are needed. It should consider the timing of intervention (especially early stages), dosage, baseline 25(OH)D levels, disease phenotype and safety (Littlejohns et al., 2014). At present, preventing vitamin D deficiency in accordance with population-based guidelines is justified. The role of targeted supplementation as a disease-modifying treatment in MS, however, remains uncertain.

Low serum levels of Vitamin D can cause many brain disorders. These include Alzheimer’s disease, Parkinson’s disease, stroke and multiple sclerosis (Melo et al., 2022). Inadequate vitamin D levels may have an additional role in pathogenesis of these conditions. Unfortunately; clinical trials have not clearly shown that vitamin D supplementation improves outcomes or prevents these diseases. In summary maintaining vitamin D levels at a recommended levels is still advised (Xiong et al., 2024).

Vitamin D: A Key Role in Autoimmune Disorders

Vitamin D plays an essential role in the immune system. Many cells have (VDR) receptors. These include T and B lymphocytes, macrophages, neutrophils and dendritic cells. Vitamin D can modulate the immune system. Its active form reduces pro-inflammatory activity of Th1 and Th17 lymphocytes. It also stimulates regulatory T cells (Treg) and Th2 cells. As a result, the secretion of anti-inflammatory cytokines, such as IL-10, increases (Latic & Erben, 2020). Due to these broad effects on the immune system, researchers naturally began exploring the connection between vitamin D and autoimmune diseases.

As mentioned earlier for multiple sclerosis, low vitamin D levels have been associated with several autoimmune conditions. These include rheumatoid arthritis (RA) and other rheumatologic diseases, systemic lupus erythematosus (SLE), systemic sclerosis, type 1 diabetes and autoimmune thyroid disorders (Ao et al., 2021).

In a large study called VITAL, published in 2022, 25,000 people took 2,000 units of vitamin D for over five years. These people had a 22% lower risk of developing autoimmune diseases compared to the placebo group. However, the most substantial effect occurred in the first two years of follow-up. These results indicate that there may be some prevention of autoimmune diseases in people who supplement vitamin D. However, further research is needed to definitively determine this and indicate the appropriate dose for supplementation.

Vitamin D: Effects on Glucose Metabolism

Vitamin D is essential for managing carbohydrates and insulin. Its receptors (VDR) are in the pancreas, muscles and fat tissue. This lets vitamin D directly affect insulin release and how tissues respond to it. Studies show that vitamin D supports pancreatic β -cells and helps tissues take up glucose. When vitamin D levels are low, insulin release tends to fall and muscle and fat cells become less responsive to circulating insulin. Vitamin D may also regulate genes involved in glucose and lipid metabolism, which could explain some of its wider metabolic effects. Overall, these findings underscore the significance of vitamin D in maintaining energy balance and promoting metabolic health.

Clinical data indicate that individuals with higher vitamin D levels tend to have better blood glucose control. They also seem less likely to develop insulin resistance. The effect is more potent in people with low vitamin D or a higher risk of metabolic disease. Supplementation trials show mixed results and apparent impact is not always clear. Even so, several studies report better glucose metabolism and lower oxidative stress. This supports a possible role for vitamin D in preventing metabolic disorders (Tang et al., 2018; Cojic et al., 2021). The main functions and effects of vitamin D are summarized in Table 1 for clarity.

Table 1. Overview of Vitamin D Functions

System/Area	Main effects/mechanisms	Research conclusions
Musculoskeletal system	Regulation of calcium–phosphate balance; effects on bone density and muscle function.	Low vitamin D levels increase fracture risk (Bischoff-Ferrari et al., 2005; Sanders et al., 2010).
Immune system	Immunomodulation; influence	Vitamin D plays a key role in autoimmune

	on inflammatory responses.	disease mechanisms (Charoenngam & Holick, 2020; Hahn et al., 2022).
Cardiovascular system	Endothelial function regulation; influence on inflammation and blood pressure.	Evidence is mixed regarding cardiovascular benefits (Pilz et al., 2012; Barbarawi et al., 2019; Haider et al., 2023).
Nervous system	Neuroprotective and regenerative effects.	Vitamin D may support neuronal health (Cui & Eyles, 2022; Won et al., 2015; Shirazi et al., 2015).
Glucose metabolism	Regulation of insulin sensitivity and carbohydrate metabolism.	Possible role in preventing type 2 diabetes and prediabetic states (Tang et al., 2018; Cojic et al., 2021).
Supplementation	Various doses and supplementation strategies.	Supplementation beneficial mainly in deficient individuals (Manson et al., 2019; Martineau et al., 2017; Jolliffe et al., 2021).

4. CONCLUSION

Vitamin D has many functions beyond its traditional role in calcium and phosphate balance. It affects the musculoskeletal, immune, cardiovascular and nervous systems. People with low serum levels have higher risk of bone fracture and injuries. It has been documented in both experimental and observational studies. However, results from some interventional trials are still uncertain. It also plays a crucial role in the pathogenesis and prevention of autoimmune diseases, which may have a significant impact on the future research. It also affects glucose metabolism and insulin sensitivity. It is suggesting a possible role in preventing type 2 diabetes and prediabetic states. Vitamin D supplements can lower the risk of diseases, especially in people with low levels. We still need more research to define the best dose and duration of supplementation. Identifying groups that benefit the most could help create more individualized prevention and treatment approaches.

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All authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

All authors contributed substantially to the work.

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Data and materials availability

All data associated with this study will be available based on reasonable request to the Corresponding Author.

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