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Research Article

Vitamin D and Cardiovascular Health

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Abstract: Vitamin D has emerged as a potential modulator of cardiovascular health. Epidemiological studies link vitamin D insufficiency with higher risks of hypertension and cardiovascular disease (CVD) events. Receptors of Vitamin D are expressed in cardiac and vascular tissues and severe deficiency of vitamin D is reported worldwide. However, does vitamin D play a causal protective role in cardiovascular health remains under debate, given inconsistent trial results. This review details the physiological role of Vitamin D in cardiovascular health which includes its role in the renin-angiotensin-aldosterone system (RAAS), vascular endothelial health, inflammation and oxidative stress, vascular calcification, disease risk in deficiencies and supplementation. In summary, vitamin D shows biological plausibility and modest signals of benefit for cardiovascular health, but robust evidence for routine supplementation to prevent heart disease or improve vascular outcomes is still lacking.

Keywords: Vitamin D; cardiovascular disease; inflammation; renin-angiotensin-aldosterone system; endothelial dysfunction

INTRODUCTION

Vitamin D serves several critical physiological roles in the human body, influencing various systems and functions (1). Vitamin D aids in the absorption of calcium and phosphorus from the intestines into the bloodstream, which helps maintain sufficient mineral levels in the body (2). This vitamin is essential for the proper growth of bone, remodelling, and mineralization (3). A deficiency of vitamin D in the body can cause bones to become thin, brittle, or misshapen. This deficiency may lead to conditions such as osteomalacia in adults and rickets in children (3). Vitamin D collaborates with parathyroid hormone (PTH) to control the levels of calcium in the blood (4).

Sufficient levels of vitamin D promote muscle contraction and relaxation, which are crucial for balance, coordination, and preventing falls, particularly in older adults (5). Vitamin D exhibits immunomodulatory effects, impacting both innate and adaptive immunity responses. It aids in controlling the production of different immune cells, cytokines, and antimicrobial peptides that are essential for defending against infections and managing inflammatory responses (6). Receptors of Vitamin D are found in various cells and tissues in the body, highlighting its importance in cellular growth, proliferation, and differentiation (7). Vitamin D could help regulate cell cycles and support tissue integrity (7). Research is ongoing regarding vitamin D's impact on cardiovascular health. Lack of vitamin D may be associated with a heightened risk of hypertension, heart disease, and stroke (8–10). Receptors of Vitamin D are present in cardiovascular tissues, and vitamin D may influence vascular function and blood pressure regulation (11,12). Vitamin D receptors are also found in brain regions that regulate mood (13). Insufficient vitamin D levels are associated with an elevated risk of depression and Seasonal Affective Disorder (SAD) (13). It is believed that vitamin D may affect neurotransmitter function and neuronal activity associated with mood (14). When necessary, sufficient vitamin D levels through exposure to sunlight, proper diet and supplements are vital for health and wellness (15).

This review discusses one of the above pathophysiology - the influence of vitamin D on heart health and to check if vitamin D is crucial for a healthy heart.

Vitamin D and Blood Pressure Regulation

Vitamin D contributes to maintaining blood pressure balance, primarily by influencing the renin-angiotensin-aldosterone system (RAAS). Animal studies show that mice without the vitamin D receptor exhibit hyperactive RAAS and develop hypertension, indicating that vitamin D typically suppresses renin release (16). Epidemiological research links low vitamin D levels to higher blood pressure. Vitamin D deficiency, typically defined as 25(OH)D levels below 30 ng/mL, serves as a separate risk factor for hypertension and is linked to increased cardiovascular mortality rates (16). In humans, blood pressure is typically lower in the summer, when vitamin D levels reach their highest, and higher in winter. This aligns with the notion that sufficient vitamin D may have a slight effect in reducing blood pressure (16).

Clinical trials have produced inconsistent outcomes. Numerous randomized trials focused solely on supplementation of vitamin D have failed to demonstrate notable decreases in blood pressure or RAAS activity among hypertensive patients (17,18). Studies indicate that while vitamin D may improve blood pressure management when used alongside traditional antihypertensive treatments, its effectiveness as a standalone approach

seems minimal (16). Overall, a lack of vitamin D might increase the risk of hypertension, but correcting it does not consistently yield significant improvements in blood pressure in clinical settings trials.

Effects on Endothelial Function and Arterial Stiffness

Vitamin D plays a crucial role in vascular endothelial function. Low levels of vitamin D are associated with endothelial dysfunction, which is marked by decreased nitric oxide availability and heightened arterial stiffness (19). This research, which included over 500 adults, revealed that lower levels of 25(OH)D correlate with diminished flow-mediated dilation and higher pulse-wave velocity, indicating stiffer arteries. Those with vitamin D insufficiency exhibited poorer vascular function compared to those with adequate levels (19).

Vitamin D enhances the endothelial nitric oxide synthase (eNOS) function, aiding vasodilation and potentially lowering vascular smooth muscle contractility. It also plays a role in limiting excessive vasoconstrictors and protecting endothelial integrity. Small intervention studies indicate that replenishing vitamin D can enhance endothelial-dependent vasodilation. For instance, hypertensive patients who received vitamin D supplementation exhibited improved flow-mediated dilation (FMD), with a significant increase compared to the placebo group (16). Specific trials observed reductions in circulating markers of endothelial activation, such as intracellular adhesion molecule-1 (ICAM-1), after vitamin D treatment (20).

Arterial stiffness is a cardiovascular metric that is affected by vitamin D levels. Research shows that individuals with low vitamin D levels experience increased arterial stiffness, such as elevated pulse wave velocity (19). Despite this, randomized controlled trials (RCTs) have produced mixed results concerning the effect of vitamin D supplementation on arterial health stiffness. An extensive evaluation and meta-analysis from 2016, which included 13 trials with approximately 800 participants, determined that vitamin D supplementation resulted in negligible reductions in pulse-wave velocity and augmentation index compared to placebo (21). The combined results indicated no statistically significant enhancement in arterial stiffness measures, implying an average lack of benefit (21). The variation in trials, such as dosing, baseline vitamin D levels, and populations, could clarify the inconsistent results (21). In brief, sufficient vitamin D is linked to improved endothelial function and more compliant arteries. However, supplementation trials have not consistently demonstrated enhancements in arterial stiffness, possibly only in deficient individuals or specific case subgroups.

Vitamin D Deficiency and Cardiovascular Disease Risk

An increasing amount of evidence connects vitamin D deficiency to a higher risk of cardiovascular diseases (CVD), including coronary artery disease, stroke, and heart failure. Low levels of serum 25(OH)D are commonly seen in CVD patients and are believed to play a role in disease development through various mechanisms such as hypertension, diabetes, and atherosclerosis. Many observational studies have demonstrated a negative correlation between 25(OH)D levels and cardiovascular health events. A meta-analysis of 19 prospective studies, involving around 66,000 individuals and over 6,000 cases of cardiovascular disease (CVD), found that individuals in the lowest vitamin D category faced a 52% greater risk of experiencing total CVD events in comparison to individuals with the highest levels of vitamin D (22). Likewise, insufficient levels of vitamin D were linked to increased risks of coronary heart disease (RR ~1.38) and stroke (RR ~1.64) in this analysis (22). A separate dose-dependent meta-analysis involving over 180,000 participants revealed a 10% reduction in cardiovascular disease (CVD) event risk for each 10 ng/mL rise in 25(OH)D levels (23). The research revealed that people with elevated vitamin D levels experienced reduced rates of cardiovascular disease (CVD) events and mortality. Specifically, for every increase of 10 ng/mL in 25(OH)D, the relative risk of experiencing CVD events was 0.90 (95% CI 0.86–0.94), while the risk of CVD-related death was 0.88 (23). The data indicate a graded inverse relationship between levels of vitamin D and cardiovascular health risk. Additionally, a deficiency of vitamin D is associated with intermediary conditions such as atherosclerosis. Those who are deficient often exhibit increased vascular calcification and endothelial dysfunction, which are early indicators of atherosclerosis (19).

Certain research indicates that a lack of vitamin D is connected with a greater risk of hypertension, metabolic syndrome, and diabetes, all of which are risk factors for cardiometabolic issues. (16,22). It's essential to recognize that although Vitamin D deficiency is associated with a greater risk of cardiovascular disease (CVD), this link does not create a causal relationship; Insufficient vitamin D levels may suggest diminished health. However, the persistent associations and reasonable biological explanations enhance the argument that insufficient Vitamin D contributes to cardiovascular disease development.

Impact on Inflammation and Oxidative Stress

Vitamin D may affect cardiovascular health through the regulation of inflammation and oxidative stress. It possesses anti-inflammatory characteristics; a deficiency in vitamin D is linked to increased levels of proinflammatory cytokines and markers. For example, individuals lacking sufficient vitamin D show elevated amounts of tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and high-sensitivity C-reactive protein (CRP) (24). Chronic, low-grade inflammation significantly contributes to atherosclerosis and plaque instability, highlighting the importance of vitamin D's immunomodulatory function. Sufficient vitamin D levels can reduce inflammatory signaling by inhibiting NF-κB activation in immune cells and fostering an anti-inflammatory response milieu.

Vitamin D functions as an antioxidant within the vascular system. When exposed to vitamin D, endothelial cells experience less oxidative stress. Particularly, 1,25-dihydroxy vitamin D inhibits the NADPH oxidase enzyme responsible for producing reactive oxygen species (ROS) (25). Vitamin D reduces superoxide production by inhibiting NADPH oxidase activity and enhancing

antioxidant defenses such as superoxide dismutase (25). Maintaining redox balance safeguards nitric oxide (NO) from oxidative breakdown, supporting endothelial function. In cases of vitamin D deficiency, an abundance of reactive oxygen species (ROS) can diminish NO levels and hinder vasodilation, leading to endothelial dysfunction and vascular harm (25).

Clinical studies confirm these mechanistic insights. In a controlled trial, hypertensive patients receiving vitamin D supplementation showed notable decreases in oxidized LDL (a marker of oxidative stress) and ICAM-1 (an inflammatory adhesion molecule) compared to their baseline levels, while also enhancing endothelial function (FMD) (16). Other studies have also observed reductions in CRP and inflammatory cytokines associated with vitamin D, particularly among patients with both vitamin D deficiency and cardiometabolic conditions. Through its impact on inflammation and oxidative stress, vitamin D may assist in stabilizing atherosclerotic plaques and enhancing overall vascular health. Nonetheless, some studies do not demonstrate significant changes in circulating inflammatory markers due to supplementation, suggesting that the impact could be greater in individuals with marked vitamin D deficiency or underlying inflammatory conditions. Overall, vitamin D sufficiency seems to provide protective benefits to the vasculature, partly by reducing pro-inflammatory pathways and oxidative stress within the cardiovascular system (24,25).

Vitamin D and Vascular Calcification

The connection between vitamin D and vascular calcification is intricate and often referred to as a "double-edged sword." Adequate vitamin D can shield against vascular calcification by aiding normal calcium metabolism and decreasing inflammation. Conversely, both significant deficiency and excess vitamin D can trigger harmful calcification in blood vessels. Studies reveal a biphasic effect: inadequate or elevated vitamin D levels may lead to harmful vascular calcification (26)

Vitamin D deficiency can lead to secondary hyperparathyroidism and inflammation, which may encourage calcification of arteries. Research using experimental models indicates that animals lacking vitamin D experience increased arterial calcification, along with elevated levels of TNF-α and osteogenic factors in their blood vessel walls (26). A deficiency can disrupt calcium and phosphate balance (resulting in low calcium, high phosphate, and elevated PTH), leading to a pro-calcific state within the blood vessels. (26).

In mice with inadequate vitamin D, the increase of bone-related proteins in arteries results in calcification resembling bone formation within the vessel media (26). These results suggest that vitamin D plays a role in preventing calcification, while a deficiency removes this regulation, particularly in the context of inflammation.

On the other hand, excessive vitamin D (hypervitaminosis D) is recognized as a key element in vascular calcification. Toxic levels of this vitamin lead to hypercalcemia and hyperphosphatemia, which may trigger calcium-phosphate accumulation in arterial walls. Studies in animal models demonstrate that elevated amounts of vitamin D result in accelerated calcification and stiffening of arteries (26).
In the past, instances of vitamin D overdose in humans—whether from industrial accidents or excessive supplementation—have resulted in extensive arterial calcification and atherosclerosis (27). Vitamin D toxicity, typically indicated by 25(OH)D levels exceeding 150 ng/mL, increases calcium and phosphate levels in the blood. Upon imaging, patients with this condition often show calcifications in blood vessels, heart valves, and kidneys (28).

Therefore, maintaining vitamin D levels in the ideal range is essential. Adequate vitamin D seems to help prevent vascular calcification, in part by influencing inflammation and controlling the differentiation of vascular smooth muscle cells (26). Excessive vitamin D or long-term deficiency can lead to an imbalance favoring calcification. This highlights the necessity of steering clear of both extremes. While addressing deficiency of vitamin D in patients, such as those with chronic kidney disease or atherosclerosis, might reduce calcification, excessive supplementation that results in hypercalcemia could instead speed up calcific vascular disease. Current evidence suggests a nuanced perspective: vitamin D protects against vascular calcification at normal levels, but may be harmful in excessively high doses (26).

Effects of Vitamin D Supplementation on Cardiovascular Outcomes

Due to the significant observational connections between low vitamin D levels and cardiovascular disease (CVD), Many studies have examined if vitamin D supplementation can improve cardiovascular health outcomes. Nevertheless, randomized controlled trials have mostly failed to demonstrate substantial cardiovascular benefits from vitamin D on its own. For instance, the VITAL trial (n=25,871) administered 2,000 IU/day of vitamin D3 to middle-aged and older adults, showing no meaningful reduction in myocardial infarction, stroke, or cardiovascular death rates compared to placebo (18).

Likewise, numerous large-scale RCTs involving generally healthy or at-risk populations have also found null results for primary cardiovascular endpoints. In a 2019 meta-analysis that combined data from 21 RCTs with more than 83,000 participants, it was determined that supplementation of vitamin D neither reduced major adverse cardiovascular events (MACE) nor significantly influenced the rates of heart attacks, strokes, or cardiovascular disease (CVD) deaths (18). The relative risk of MACE associated with vitamin D was roughly 1.00 (indicating no effect), and no significant benefit on all-cause mortality was observed either (18). These results indicate that merely supplementing vitamin D in populations with adequate nutrition does not effectively prevent heart disease events.
Nonetheless, there are subtleties. Specific trials focusing on patients with vitamin D deficiency or particular subgroups have demonstrated modest advantages. For example, small studies involving hypertensive or heart failure patients with insufficient levels of vitamin D have suggested enhancements in cardiac function, decreased blood

pressure, or reduced inflammation when vitamin D was administered as supplementary therapy (16). In Australia, the D-Health Trial involving 21,315 older adults investigated the effects of a once-a-month high dose of vitamin D (60,000 IU) over 5 years. Although the main results did not achieve statistical significance, a trend suggested a decrease in significant cardiovascular incidents in individuals taking vitamin D (hazard ratio ~0.91, p=0.06), indicating approximately 5.8 fewer events per 1,000 individuals (29). Interestingly, participants taking cardiovascular medications seemed to experience a more significant risk reduction (HR ~0.84), although this subgroup finding was exploratory (29). This suggests that some high-risk individuals may gain marginal benefits from supplementation, or that vitamin D could work in conjunction with other nutrient therapies.

In summary, systematic reviews and meta-analyses suggest that supplementation of vitamin D does not considerably reduce the risk of heart attacks, strokes, or mortality in the general population (18). Despite significant observational links, the absence of impact in trials implies that low vitamin D could indicate poor health rather than serve as a direct cause. Additionally, simply correcting the levels of vitamin D may not be enough to influence the intricacies of cardiovascular disease. The advantages of supplementation may primarily appear in individuals with significant deficiencies or particular health conditions. Future and ongoing studies looking at patients with deficiencies or adding vitamin D to other treatments will help elucidate its potential role in preventing cardiovascular issues. Currently, vitamin D supplements are recommended to address and deter deficiencies for better overall health, but they should not be relied upon as a specific supplement for heart protection due to variable results in trials (18).

Recommended Vitamin D Levels for Cardiovascular Health and Risks of Excess

Although there is no widespread agreement on the perfect vitamin D level for cardiovascular health, it is generally advised to maintain adequate vitamin D levels. Clinical guidelines typically identify deficiency of vitamin D as serum 25(OH)D levels less than 20 ng/mL (50 nmol/L), while insufficiency is around 20 to 30 ng/mL. Numerous experts suggest aiming for at least 30 ng/mL (75 nmol/L) to maximize health benefits, including possible cardiovascular advantages beyond just bone health (16). Observational data supports this threshold, indicating that cardiovascular risk rises when 25(OH)D levels drop below approximately 20–30 ng/mL (26). For instance, one meta-analysis observed a mostly linear reduction in cardiovascular disease risk as vitamin D levels increased from 20 to 60 nmol/L (24 ng/mL) (26). Therefore, maintaining 25(OH)D levels in the mid-normal range (around 30–50 ng/mL) is a sensible objective for overall health and cardiovascular well-being health.

It is crucial to avoid excessively high vitamin D levels, since substantial doses can be harmful. The commonly referenced upper intake limit for adults is about 4,000 IU daily, although higher short-term doses may be employed for therapeutic purposes deficiency). Serum 25(OH)D levels exceeding 100 ng/mL (250 nmol/L) are typically excessive, and levels above 150 ng/mL (375 nmol/L) may be harmful (28). Excessive vitamin D can result in hypercalcemia, or elevated calcium levels in the bloodstream. This condition may lead to symptoms such as nausea, kidney stones, confusion, and harmful calcification in soft tissues. Notably, severe vitamin D surplus can trigger calcium accumulation in blood vessels and cardiac tissues, which unexpectedly heightens cardiovascular risk (28).

Vitamin D toxicity in clinical settings is infrequent and usually arises from megadose supplementation, such as during industrial accidents or dosing mistakes. This highlights the caution that “more is not always better." After reaching adequate levels of 25(OH)D (approximately 30–50 ng/mL), exceeding those levels offers no validated cardiovascular benefits, and might even pose risks.

In conclusion, addressing vitamin D deficiency and ensuring sufficient levels are wise for cardiovascular health, since low levels are linked to hypertension, atherosclerosis, and negative cardiac effects. The advised serum 25(OH)D levels typically fall in the sufficient range of 20–30 ng/mL to promote overall wellness (16).

Physicians typically target a 30–50 ng/mL range for at-risk patients. Vitamin D supplementation is safe when correctly used, but staying within recommended dosing limits is essential to prevent hypercalcemia. By maintaining sufficient levels and preventing deficiency, it may be possible to reduce certain cardiovascular risk factors while steering clear of the dangers associated with excessive intake. Recent clinical trials and meta-analyses support a balanced strategy: restoring low vitamin D levels to normal is advantageous, but excessive doses beyond physiological requirements offer no additional cardiovascular benefits and could be harmful (18,28).

CONCLUSION

These comprehensive and high-quality studies suggest that Vitamin D supplementation has little effect on primary cardiovascular outcomes in the general population. This reinforces the need to prioritize vitamin D for addressing deficiencies and promoting overall health instead of viewing it primarily as a means of cardiovascular protection agent.

Abbreviations:

CRP - C-reactive protein
CVD - cardiovascular diseases
eNOS - endothelial nitric oxide synthase
ICAM-1 - intracellular adhesion molecule-1
IL-6 - interleukin-6
RAAS - renin-angiotensin-aldosterone system
ROS - reactive oxygen species
TNF-α - tumor necrosis factor-alpha

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