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Vitamin D and Hypertension: Mechanistic Insights, Epidemiological Evidence, Clinical Trials and Future Directions

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Abstract

Background: Vitamin D, generally associated with bone health, may also potentially play a role in regulating cardiovascular function, as vitamin D receptors are ubiquitously expressed in the vascular, heart and kidney tissue. Hypertension is a significant contributor to cardiovascular diseases and mortality across the globe.

Evidence: In experimental studies, the role of vitamin D in blood pressure regulation via the inhibition of the renin-angiotensin-aldosterone system, improvement in endothelial function, attenuation of oxidative stress and inflammation and modulation of vascular remodeling has been demonstrated. In observational studies, there is an established inverse relationship between 25-hydroxyvitamin D (25(OH)D) levels in the serum and the risk of hypertension. However, most of the large vitamin D supplementation studies and randomized controlled trials have not, as a rule, demonstrated significant blood pressure reduction after vitamin D supplementation.

Clinical implications: Although there is no justification for the routine use of vitamin D supplements in the treatment of hypertension based on the available evidence, vitamin D deficiency correction may be associated with cardiovascular benefits, especially in high-risk groups such as those who are obese, have metabolic syndrome, chronic kidney disease, resistant hypertension or severe vitamin D deficiency.

Conclusion: Vitamin D is involved in several biological pathways that are important for blood pressure regulation, but definite clinical evidence for its use in treating hypertension does not exist. Although addressing vitamin D deficiency may contribute to lowering the risk of cardiovascular disease in some populations, there is no evidence that would support a widespread approach of supplementation to treat hypertension. Further well-designed clinical trials are needed to define its role in personalised cardiovascular care.

Keywords: vitamin D, hypertension, blood pressure, cardiovascular disease, endothelial dysfunction, renin-angiotensin-aldosterone

Introduction

Hypertension is a risk factor for heart disease, stroke, kidney disease and mortality. It is still one of the

leading causes of death and disease worldwide (World Health Organization , 2023). Despite advances in the management of hypertension, many patients are failing to achieve optimal blood pressure control and are remaining at elevated risk of cardiovascular disease (Mills et al., 2020). As a consequence, there continues to be a search for additional interventions to reduce cardiovascular risk.

Vitamin D and calcium homeostasis and bone health has been known for long time. However, vitamin D is also correlated with cardiovascular function. The presence of vitamin D receptors in vascular endothelial cells, vascular smooth muscle cells, cardiomyocytes, and renal tissues suggests that vitamin D may play a role in blood pressure regulation and cardiovascular health.

(Li et al., 2002; Andrukhova et al., 2014). There are some proposed biological mechanisms for this correlation. In experimental studies it has been shown that vitamin D can inhibit transcription of renin gene and change the renin-angiotensin-aldosterone system (RAAS). This resulted in decreased angiotensin II production and contributes to blood pressure regulation (Li et al., 2002; Tomaschitz et al., 2010).

Vitamin D can improve endothelial function by increasing nitric oxide availability and promotes vascular relaxation (Andrukhova et al., 2014; Vanhoutte et al., 2017). Vitamin D deficiency is also correlated with increased oxidative stress, vascular inflammation, and endothelial dysfunction. That leads to hypertension and cardiovascular damage. (Judd & Tangpricha, 2009;Pilz et al., 2016). Moreover, Vitamin D can inhibit vascular smooth muscle cells proliferation and adverse vascular remodeling. It helps maintain arterial compliance and reduce cardiovascular risk. (Talmor et al., 2008; Vanhoutte et al., 2017)

Additionally, there is also some epidemiologic data suggestive of possible involvement of vitamin D in BP regulation. The meta-analysis showed that low levels of 25-hydroxyvitamin D [25(OH)D] were associated with a higher risk of developing hypertension compared to sufficient vitamin D levels (Burgaz et al., 2011; Kunutsor et al., 2013). Additionally, prospective cohort studies have demonstrated that lower initial vitamin D levels were associated with an increased risk of future development of hypertension (Forman et al., 2007; Kunutsor et al., 2013). Although causality cannot be proven with observational studies, the consistency of the results from different populations has strengthened the role of vitamin D in cardiovascular regulation (Mirhosseini et al., 2018).

But results from randomized control trials were fairly ambiguous. While it is apparent from the laboratory studies and observational studies that vitamin D status may be beneficial for blood pressure, many of the intervention studies have shown a modest or no significant reduction in blood pressure with vitamin D supplementation (Beveridge et al., 2015;Pilz et al., 2015). There was no effect on blood pressure despite significant increase in the vitamin D levels in major clinical trials such as Vitamin D and OmegA-3 Trial (VITAL) and Women's Health Initiative (Manson et al., 2019;Margolis et al., 2008).

This discrepancy may be attributed to the baseline status of the vitamin D among the study participants, dose of the supplementation, duration of the treatment, characteristics of study population and their CV risk profiles. Vitamin D supplementation may be more beneficial in specific groups, e.g., severe vitamin D deficiency, obesity, metabolic syndrome or resistant hypertension (Mirhosseini et al., 2018).

Interestingly, recent findings have also suggested that there may be a complex relationship between vitamin D status and blood pressure regulation. In a recent systematic review, vitamin D supplementation was associated with modest reduction in systolic blood pressure and diastolic blood pressure in some populations. However, high variability in the studies and inconsistency of the clinical outcomes precluded any definitive conclusions regarding its antihypertensive effects (John et al., 2025).

Amer et al. (2025) further reported modest but statistically significant reductions in both systolic and diastolic blood pressure following vitamin D supplementation. Although the observed effects were small,

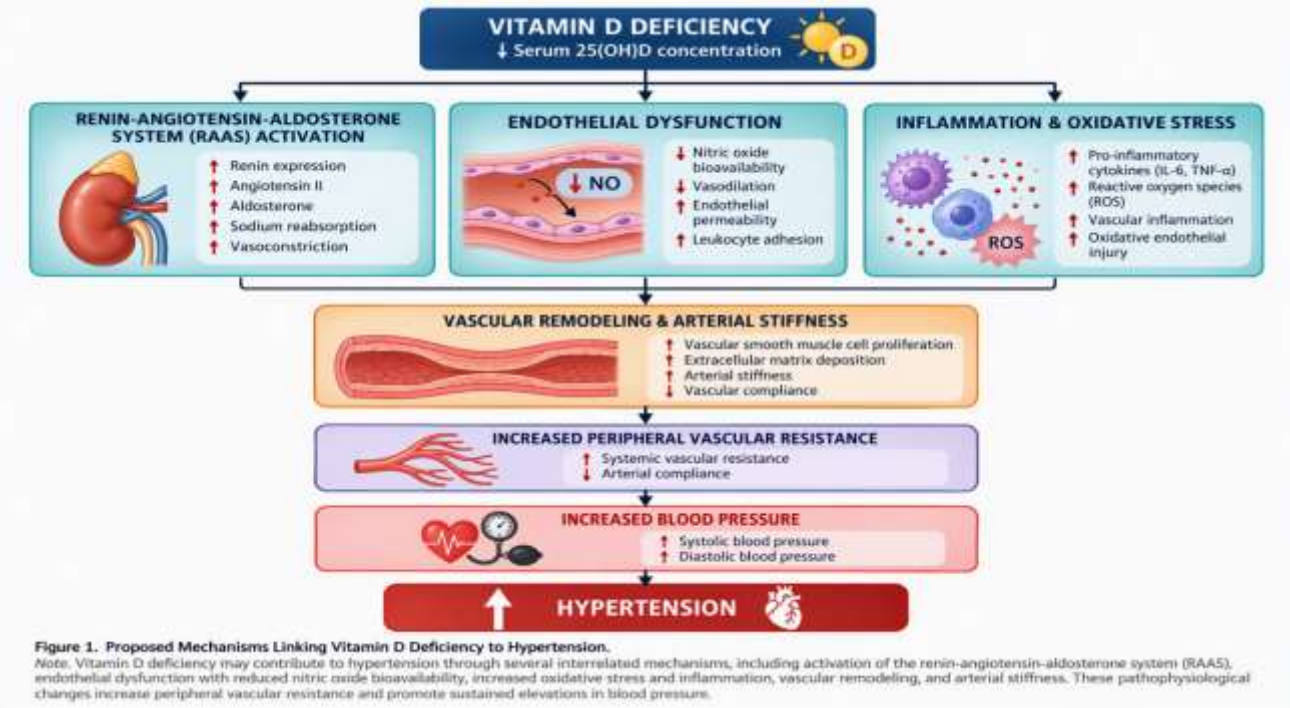
these findings suggest that vitamin D may serve as a complementary strategy for blood pressure management in selected populations rather than as a primary antihypertensive therapy. Nevertheless, substantial heterogeneity among studies highlights the need for cautious interpretation and further investigation to identify the patient groups most likely to benefit.

Since there is increasing interest in the effects of vitamin D beyond those on the skeleton and since the findings reported in observational studies, clinical trials, and recent meta-analyses have been inconsistent, it is appropriate to carry out a thorough assessment of the existing evidence. That is why this systematic review seeks to look at the impact of vitamin D supplementation on blood pressure in adults and to evaluate its possible role in the prevention and control of hypertension.

Literature Search Strategy

Systematic literature search was performed in PubMed, Scopus, and Google Scholar, guided by the PRISMA 2020 guidelines from January 2000 to June 2025. The search terms were "vitamin D", "25-hydroxyvitamin D", "hypertension", "blood pressure", "cardiovascular disease", "endothelial dysfunction", and "renin-angiotensin-aldosterone system". We limited the search results to English-language literature. Randomized controlled trials, observational studies, cohort studies, systematic reviews, and meta-analyses that assessed the relationship between vitamin D and blood pressure or risk of hypertension were included. Animal studies, editorials, conference proceedings, duplicate articles, and studies that lacked relevant outcomes were excluded. We screened the studies by title and abstract and checked full-text articles for eligibility. We also searched manually for references to find other relevant articles. We narrative summarized the included evidence to summarize the mechanistic, epidemiological, and clinical evidence on the relationship between vitamin D and hypertension.

Figure 1 ***Mechanistic Pathways Connecting Vitamin D and Hypertension***



Note. *Conceptual model developed from findings reported by Li et al. (2002), Andrukhova et al. (2014), and Vanhoutte et al. (2017), illustrating potential mechanisms linking vitamin D deficiency to hypertension.*

Mechanistic Pathways That Connect Vitamin D and Hypertension:

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

Experimental studies have shown that vitamin D acts as a regulator of the renin-angiotensin-aldosterone system by suppressing renin gene transcription and reducing the production of angiotensin II (Li et al., 2002; Tomaschitz et al., 2010).

2. Endothelial Function.

Vitamin D enhances endothelial-dependent vasodilation and vascular tone by increased oxide production and decreased arginase-2 activity (Andrukhova et al., 2014; Vanhoutte et al., 2017).

3. Inflammation and Oxidative Stress

Vitamin D deficiency is associated with oxidative stress, vascular inflammation and endothelial dysfunction, all of which increase blood pressure and damage vessels (Pilz et al., 2016; Judd & Tangpricha 2009).

4. Remodeling of vessels

Vitamin D may inhibit smooth muscle cell proliferation and prevent adverse remodeling, thus maintaining arterial compliance and reducing cardiac risk (Talmor et al., 2008; Vanhoutte et al., 2017).

Epidemiologic evidence

There is epidemiological evidence that a higher vitamin D status is associated with a lower risk of hypertension. Meta-analyses show that low 25(OH)D levels are associated with increased risk of developing high blood pressure compared to adequate vitamin D levels (Burgaz et al., 2011; Kunutsor et al., 2013).

Prospective cohort studies also show that low baseline vitamin D is predictive of future hypertension risk, reinforcing the idea that vitamin D may influence heart regulation (Forman et al.,2007; Kunutsor et al. 2013). While observational studies cannot prove cause, the consistent results across groups strengthen the hypothesis that vitamin D may influence heart health (Mirhosseini et al., 2018).

Clinical intervention trials

Laboratory and population studies have suggested an association between vitamin D status and blood pressure, but randomized controlled trials have generally reported inconsistent or modest effects of vitamin D supplementation on blood pressure outcomes (Beveridge et al., 2015; Pilz et al., 2015). In large-scale trials like the VITAL study and the Women's Health Initiative, vitamin D supplementation did not result in clinically meaningful blood pressure reductions in the overall study populations, despite raising circulating vitamin D concentrations (Manson et al., 2019; Margolis et al., 2008).

But there is some more recent evidence suggesting potential benefits may be confined to particular subgroups. Serra et al. (2024) conducted systematic review and meta-analysis and found a modest decrease in systolic blood pressure in people with hypertension and vitamin D deficiency, but effects on diastolic blood pressure were not consistent. A meta-analysis by (Amer et al., 2025) published in 2025 also showed small but statistically significant reductions in systolic and diastolic blood pressure with vitamin D supplementation. But the authors noted considerable heterogeneity between studies, including baseline vitamin D status, supplementation regimens, participant characteristics and study duration. Thus, the clinical significance and generalizability of these findings are still poorly defined.

The discrepancy between observational and interventional evidence may be accounted for by methodological and biological factors. In many supplementation trials, inclusion of participants without

vitamin D deficiency may have limited the opportunity to find treatment effects. Inconsistent findings may also be explained by differences in dose, duration of supplementation, baseline cardiovascular risk and co-interventions (Beveridge et al., 2015; Pilz et al., 2015). Current evidence does not support vitamin D supplementation as a global strategy for blood pressure reduction. Nevertheless, addressing vitamin D deficiency might be a rational part of holistic cardiovascular risk management in certain patients, though further robust trials are necessary to clarify which populations, if any, experience significant blood pressure benefits (Mirhosseini et al., 2018; Serra et al., 2024; Amer et al., 2025).

Table 1

Clinical Trials and Meta-Analyses Evaluating Vitamin D Supplementation and Blood Pressure Outcomes

Study	Sample Size	Intervention	Blood Pressure Outcome
Manson et al.,(2019)	25,871	Vitamin D_3 2,000 IU/day	No significant blood pressure benefit reported
Pilz et al. (2015)	200	Vitamin D_3 2,800 IU/day for 8 weeks	No significant reduction in 24-hour blood pressure
Women's Health Initiative (Margolis et al.,2008)	36,282	Vitamin D plus calcium	No significant effect on blood pressure or incident hypertension
Witham et al. (2009)	11 trials	Various vitamin D regimens	Limited evidence of clinically meaningful benefit

Note. Overall, vitamin D supplementation showed little to no significant effect on blood pressure across major clinical trials and reviews.

Clinical Translation: Precision Supplementation

Although many large randomized controlled trials have failed to find drops in blood pressure following vitamin D supplementation in general groups new evidence suggests some people gain more benefit. Vitamin D may be more effective in individuals with vitamin D deficiency, obesity, metabolic syndrome, chronic kidney disease or resistant hypertension, because vitamin D activates pathways such as the renin-angiotensin-aldosterone system, endothelial function and vascular inflammation.

The current evidence supports a precision approach to vitamin D supplementation to target those with deficiency or at higher cardiovascular risk, rather than universal supplementation.

In this way vitamin D use is aligned to medicine ideas and maximizes benefit while avoiding unnecessary vitamin D for those who already have enough.

Table 2

Comparison of Universal and Targeted Vitamin D Supplementation Strategies

Parameter	Universal Approach	Targeted Approach
Population	General population	Vitamin D-deficient or high-risk individuals
Blood Pressure Effect	Minimal or none	Modest reduction

Goal	General prevention	Correction of deficiency and vascular support
Evidence Base	Large randomized trials	Mechanistic and subgroup studies
Clinical Role	Limited	Adjunctive therapy

Note. Evidence from randomized trials and meta-analyses suggests minimal blood pressure benefits in the general population, with modest effects potentially observed in vitamin D-deficient or high-risk subgroups.

Discussion

The association between vitamin D status and hypertension has a biologically plausible foundation and is supported by a substantial body of mechanistic and epidemiological evidence. Experimental studies suggest that vitamin D influences several pathways involved in blood pressure regulation, including suppression of the renin-angiotensin-aldosterone system (RAAS), improvement of endothelial function, attenuation of oxidative stress and inflammation, and inhibition of adverse vascular remodeling (Li et al., 2002; Andrukhova et al., 2014; Pilz et al., 2016; Vanhoutte et al., 2017). Through these mechanisms, vitamin D may contribute to the maintenance of vascular homeostasis and cardiovascular health. These findings provide a biological rationale for the inverse association between circulating 25-hydroxyvitamin D [25(OH)D] concentrations and hypertension risk observed in numerous observational studies (Forman et al., 2007; Burgaz et al., 2011; Kunutsor et al., 2013).

Consistent with previous epidemiological investigations, observational evidence indicates that individuals with lower serum 25(OH)D concentrations are more likely to develop hypertension than those with adequate vitamin D status (Forman et al., 2007; Burgaz et al., 2011). Meta-analyses have further demonstrated an inverse relationship between vitamin D levels and incident hypertension across diverse populations (Kunutsor et al., 2013; Mirhosseini et al., 2018). Collectively, these findings suggest that vitamin D deficiency may represent a modifiable risk factor for elevated blood pressure. However, because observational studies cannot fully account for residual confounding or reverse causation, causal inferences should be made cautiously.

Despite promising mechanistic and observational findings, evidence from randomized controlled trials (RCTs) has been less conclusive. Large intervention studies including the Women's Health Initiative and the VITAL trial showed little or no significant reduction in blood pressure after vitamin D supplementation among the overall study populations (Margolis et al., 2008; Manson et al., 2019). Similarly, previous systematic reviews and meta-analyses have found vitamin D supplementation to have limited effects on blood pressure in unselected populations (Beveridge et al., 2015; Pilz et al., 2015). These results indicate that increasing vitamin D levels alone may not be enough to achieve clinically meaningful reductions in blood pressure in the general population.

Recent evidence, however, suggests that the effect of vitamin D supplementation may be dependent on patient characteristics at baseline. Serra et al. (2024) conducted a systematic review and meta-analysis, finding small reductions in systolic blood pressure in people with hypertension and vitamin D deficiency. The largest benefits were seen in certain subgroups, such as older adults and those who received higher cumulative doses of vitamin D. In line with this, Amer et al. (2025) found small but statistically significant reductions in both systolic and diastolic blood pressure following vitamin D supplementation, although there was substantial heterogeneity across the studies included. These findings suggest that the cardiovascular benefits of vitamin D may be more evident in those with vitamin D deficiency or high cardiometabolic risk than in generally representative populations.

This difference between observational and interventional findings may be due to several reasons. First, many of the RCTs included participants with adequate baseline vitamin D concentrations, which may have decreased the chances of detecting benefits of supplementation. Second, there is a considerable variation between studies in the vitamin D dosage, treatment duration, formulations, adherence and participant characteristics which makes direct comparisons difficult (Beveridge et al., 2015; Serra et al., 2024). Third, hypertension is a complex disorder involving genetic, environmental, metabolic, and lifestyle factors and this complexity may dilute the measurable contribution of vitamin D when studied in isolation. There is therefore no evidence to support universal supplementation with vitamin D as an antihypertensive intervention at this time. Correction of vitamin D deficiency might be one part of a holistic strategy to reduce cardiovascular risk, especially in those at high risk of deficiency or vascular dysfunction (Mirhosseini et al., 2018; Pilz et al., 2015).

Some limitations should be considered in interpreting the available evidence. The bulk of the current literature is observational and thus prone to residual confounding and selection bias. Publication bias cannot be excluded as well, and studies with significant associations are more likely to be published than studies with null findings. Furthermore, there has been great heterogeneity in supplementation regimens, characteristics of participants, baseline vitamin D status, and methods of outcome assessment in clinical trials. Many studies also had relatively short follow-up periods which may have limited their ability to detect long-term effects on blood pressure, vascular health or cardiovascular outcomes. These methodological limitations may partially explain the inconsistent findings reported in the literature.

Future Directions

Future studies should include adequately powered randomized controlled trials involving participants with confirmed vitamin D deficiency and standardized supplementation protocols. Longer follow-up periods are needed to evaluate the sustainability of blood pressure reductions and potential effects on cardiovascular outcomes. Particular attention should be given to high-risk populations, including individuals with obesity, metabolic syndrome, chronic kidney disease, and resistant hypertension, as these groups may derive greater benefit from vitamin D supplementation. Incorporating biomarkers of endothelial function, inflammation, oxidative stress, and renin-angiotensin-aldosterone system activity may further clarify the biological mechanisms linking vitamin D and hypertension. Additionally, precision medicine approaches and individual participant-level analyses could help identify patient subgroups most likely to respond to supplementation and support more personalized cardiovascular prevention strategies.

Conclusion

Vitamin D is involved in multiple physiological pathways related to blood pressure regulation, including modulation of the renin-angiotensin-aldosterone system, endothelial function, oxidative stress, inflammation, and vascular remodeling. Observational evidence consistently demonstrates an association between low serum vitamin D levels and an increased risk of hypertension. However, randomized controlled trials have generally shown limited or inconsistent blood pressure benefits from vitamin D supplementation in the overall population. Current evidence does not support routine vitamin D supplementation as a primary antihypertensive therapy. Nevertheless, correction of vitamin D deficiency may provide cardiovascular benefits in selected high-risk individuals and should be considered within a broader cardiovascular risk-reduction strategy. Further high-quality research is required to determine

which patient populations may benefit most and to define the role of vitamin D in personalized hypertension management.

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