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Role of Vitamin D in Periodontal Health and Disease

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ABSTRACT

Vitamin D is a secosteroid hormone essential for bone mineralization, calcium metabolism, and immunoregulation. Increasing evidence suggests a strong association between vitamin D deficiency and periodontal disease severity. Vitamin D exerts anti-inflammatory, antimicrobial, and host-modulatory effects that influence periodontal tissue homeostasis. This narrative review summarizes the biological basis linking vitamin D to periodontal health, explores clinical and genetic evidence for its role in periodontitis, and discusses implications for prevention and therapy.

INTRODUCTION

Vitamin D deficiency is a global health concern affecting nearly one billion people worldwide. Beyond its classical role in skeletal metabolism, vitamin D has emerged as a key regulator of immune and epithelial functions. Its deficiency has been implicated in systemic conditions such as diabetes, rheumatoid arthritis, cardiovascular diseases, and oral diseases including periodontitis. Periodontitis is a chronic inflammatory disorder initiated by microbial biofilms, leading to the destruction of tooth-supporting structures and alveolar bone.

The disease shares inflammatory pathways with systemic conditions, and nutritional factors—particularly vitamin D—modulate its progression. Observational and experimental data demonstrate that low serum 25-hydroxyvitamin D [25(OH)D_3] correlates with greater clinical attachment loss, alveolar bone resorption, and poorer treatment outcomes. This review outlines the biological pathways through which vitamin D acts on periodontal tissues and evaluates clinical evidence linking vitamin D status with periodontal health.

Vitamin D Metabolism and Mechanisms of Action

Vitamin D exists in two primary forms: D_3 (cholecalciferol), synthesized in the skin via UV-B exposure, and D_2 (ergocalciferol), obtained from diet and supplements. Both are hydroxylated in the liver to 25(OH)D_3, then in the kidneys and other tissues—via 1α-hydroxylase (CYP27B1)—to the active hormone 1,25$(OH)_2D_3$ (calcitriol). Calcitriol binds to the vitamin D receptor (VDR), a nuclear receptor expressed in epithelial cells, osteoblasts, and immune cells, modulating transcription of over 200 genes involved in mineral homeostasis, immunity, and inflammation.

1. Maintenance of Bone and Periodontal Tissue Integrity

Calcitriol maintains calcium-phosphate balance and bone mineralization by enhancing intestinal calcium absorption and regulating osteoblastic gene expression. Animal models demonstrate that deletion of the CYP27B1 gene increases pro-inflammatory cytokines (IL-1β, TNF-α) and matrix metalloproteinases (MMP-3, MMP-8), leading to accelerated alveolar bone loss. Conversely, exogenous calcitriol administration mitigates bone resorption and gingival inflammation in ligature-induced periodontitis. Through regulation of parathyroid hormone (PTH) and feedback on calcium metabolism, vitamin D deficiency results in compensatory hyperparathyroidism, osteoclastic bone resorption, and loss of alveolar bone density—key features of advanced periodontitis.

2. Epithelial and Immune Defense Mechanisms

Vitamin D strengthens the epithelial barrier of the gingiva and junctional epithelium—the first line of defense against bacterial invasion. Gingival fibroblasts and periodontal ligament cells locally express 25-hydroxylase and CYP27B1, enabling autocrine synthesis of calcitriol in response to microbial stimuli such as Porphyromonas gingivalis lipopolysaccharide.

Calcitriol enhances the expression of tight-junction proteins (E-cadherin, claudins) and attenuates TNF-α-induced MMP-9 activity via NF-κB inhibition, thereby maintaining epithelial integrity. This epithelial reinforcement limits bacterial penetration and modulates the local inflammatory milieu.

3. Antimicrobial Activity
Calcitriol induces potent antimicrobial peptides, particularly cathelicidin (LL-37) and β-defensins, via TLR-mediated pathways. LL-37 exhibits broad bactericidal action against Gram-positive and Gram-negative organisms, neutralizes endotoxins, and promotes wound healing. In vitro, calcitriol exposure increases LL-37 expression in human gingival epithelial cells up to 13-fold. Furthermore, vitamin D directly inhibits P. gingivalis virulence by suppressing fimbrial adhesins (fimA, hagA, hagB) and proteases (rgpA, rgpB, kgp), and enhances autophagic clearance of internalized pathogens—underscoring its role in innate immune defense.

4. Anti-Inflammatory and Host-Modulatory Effects
Calcitriol exerts broad immunomodulatory control by shifting cytokine profiles from a destructive Th1/Th17-dominant to a reparative Th2/Treg phenotype. It suppresses IL-1β, IL-6, TNF-α, and RANKL while promoting anti-inflammatory cytokines such as IL-10. Experimental periodontitis models show that calcitriol reduces NF-κB activation, downregulates RANKL expression, and limits osteoclastogenesis, thereby preserving alveolar bone.

Clinical studies corroborate these findings, reporting inverse correlations between serum $25(OH)D_3$ and circulating IL-6 or TNF-α levels in patients with periodontitis.

5. Genetic Determinants: Vitamin D Receptor (VDR) Polymorphisms
VDR polymorphisms influence individual susceptibility to periodontitis. Variants such as FokI, BsmI, and TaqI have been linked to greater attachment loss and bone resorption. The FokI (rs2228570) polymorphism alters VDR protein structure and transcriptional activity. Individuals with the FF genotype exhibit increased RANKL expression in gingival fibroblasts, enhancing osteoclastogenesis and periodontal breakdown. These genetic associations strengthen the biological plausibility of vitamin D's involvement in periodontal pathology.

6. Vitamin D Status and Serum Thresholds
Serum $25(OH)D_3$ is the most reliable biomarker of vitamin D status, reflecting both cutaneous synthesis and dietary intake. Although definitions vary, concentrations <20 ng/mL denote deficiency, while >30 ng/mL are considered sufficient. The Endocrine Society recommends maintaining levels above 30 ng/mL for optimal systemic and periodontal benefits.

7. Evidence Linking Vitamin D Deficiency to Periodontal Disease
Cross-sectional and case-control studies consistently demonstrate an inverse relationship between serum $25(OH)D_3$ and periodontal inflammation. Analyses of the NHANES III dataset revealed that individuals in the highest quintile of vitamin D had 20% lower odds of bleeding on probing compared with the lowest quintile. Similar trends were observed in the OsteoPerio study, where postmenopausal women with adequate vitamin D (>50 nmol/L) showed significantly reduced periodontal inflammation.

Vitamin D deficiency is particularly detrimental in vulnerable populations such as the elderly, postmenopausal women, and pregnant women, correlating with greater attachment loss and even adverse pregnancy outcomes like preterm birth.

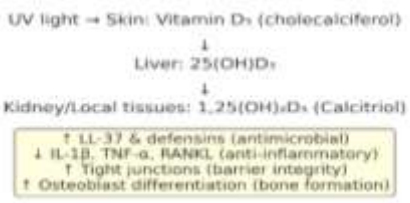

Figure 1. Biological roles of calcitriol in periodontal health

8. Longitudinal and Interventional Evidence
Long-term studies present mixed findings due to methodological heterogeneity. The Dental Longitudinal Study reported lower odds of severe periodontal disease among participants consuming ≥800 IU/day of vitamin D. Similarly, the Study of Health in Pomerania demonstrated an inverse association between serum vitamin D and tooth loss incidence. Conversely, the 5-year OsteoPerio follow-up found no significant relation between baseline vitamin D and periodontal progression, suggesting vitamin D may influence inflammation rather than long-term structural loss. Clinical trials evaluating supplementation have provided promising but modest results. Hiremath et al. observed dose-dependent reductions in gingival inflammation following daily vitamin D supplementation (500–2000 IU). Kraal et al. found that combined calcium

(500 mg) and vitamin D (700 IU) for 3 years reduced tooth loss by 60%. Randomized controlled trials assessing adjunctive vitamin D during non-surgical therapy showed slight improvements in probing depth reduction, though evidence remains inconclusive. Importantly, baseline sufficiency rather than supplementation during therapy appears critical for optimal healing and post-surgical outcomes.

DISCUSSION

The collective evidence underscores vitamin D's multifaceted role in maintaining periodontal health. Its effects span skeletal preservation, epithelial defense, microbial regulation, and immune modulation. However, heterogeneity among study designs—especially in defining vitamin D sufficiency, case definitions of periodontitis, and outcome measures—limits direct comparison. Most positive associations emerge from cross-sectional studies, while longitudinal and interventional data remain inconclusive. The potential confounding influence of calcium intake, sunlight exposure, and genetic polymorphisms further complicates interpretation. Nevertheless, maintaining physiological vitamin D levels appears beneficial for gingival inflammation control and may enhance response to periodontal therapy.

CONCLUSION

Vitamin D acts as a critical modulator of periodontal homeostasis through anti-inflammatory, antimicrobial, and osteogenic mechanisms. Deficiency correlates with increased periodontal inflammation and alveolar bone loss, while adequate serum 25(OH)D_3 supports healing and stability. Although definitive causality awaits stronger longitudinal and interventional evidence, ensuring vitamin D sufficiency—through sunlight exposure, diet, or supplementation—should be regarded as an adjunctive measure in periodontal health maintenance.

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