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Effects of Vitamin D on Orthodontic Tooth Movement and Treatment Stability: A Literature Review

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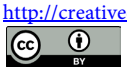

Abstract

Objective: Vitamin D, a key regulator of phosphocalcic metabolism, plays a central role in bone remodeling—a fundamental process underlying orthodontic tooth movement and post-treatment stability. Its specific influence on orthodontic outcomes, and in particular on relapse prevention, remains insufficiently characterized. This review systematizes the recent evidence on the role of vitamin D in orthodontics, focusing on its biological mechanisms and its potential to enhance treatment stability. **Materials and Methods:** A literature search was conducted in PubMed, ScienceDirect, Springer, and Google Scholar for studies published between 2013 and 2024. Selection followed the PRISMA recommendations, with an initial screening of titles and abstracts followed by full-text analysis. Relevant data were then extracted and critically appraised. **Results:** Of 3792 references initially identified, 2223 were retained after removal of duplicates and date restriction. After full-text reading, 27 articles were included: 6 meta-analyses and systematic reviews, 2 narrative reviews, 4 randomized clinical trials, 2 prospective studies, 1 *in vitro* study, 10 *in vivo* studies, and 2 longitudinal studies. **Discussion:** The body of evidence converges on a decisive role of vitamin D in bone remodeling, through modulation of the RANKL/OPG ratio and of the osteoclast/osteoblast balance. At physiological doses, it accelerates tooth movement and promotes higher-quality bone regeneration, reducing the risk of relapse and supporting periodontal fiber reorganization. Inappropriate dosing may, however, produce paradoxical effects, notably excessive bone densification that hinders tooth mobility. Methodological heterogeneity and small sample sizes limit the strength of current conclusions. **Conclusion:** Vitamin D emerges as a promising therapeutic adjunct in orthodontics, with notable benefits for bone remodeling and post-treatment stability. An individualized approach accounting for each patient's vitamin D status remains essential, as do large-scale clinical trials to establish reliable recommendations.

Subject Areas

Dentistry

Keywords

Vitamin D, Calcitriol, Orthodontic Tooth Movement, Orthodontic Stability, Relapse, Bone Remodeling, RANKL/OPG

1. Introduction

Vitamin D is a steroid hormone essential to phosphocalcic balance and bone health. Although it can be synthesized in the skin under the effect of UVB radiation, a widespread deficiency is observed in many regions, including Morocco, where nearly 85% of men and 77% of women over 50 years of age show vitamin insufficiency [1]. This situation, paradoxical in a country with abundant sunshine, is explained by cultural, dietary, and behavioral factors [2]. Genetic variations affecting key genes, such as CYP27B1, which is involved in the conversion of vitamin D to its active form, may exacerbate this deficit, particularly in populations where consanguinity is common [3].

These often-underestimated deficiencies have important systemic implications and also consequences for specific disciplines such as orthodontics, where they may influence the biological response to treatment and post-therapeutic stability. In orthodontics, post-treatment stability is an essential yet difficult goal to achieve. Relapse—defined as the return of teeth toward their initial position after removal of active appliances—remains a major problem despite retention: some studies report that it affects 30% to 50% of patients [4]. This phenomenon is linked to the slow reorganization of periodontal fibers, soft-tissue pressure, and individual biological variations [5].

The mechanisms of orthodontic stability are directly governed by bone remodeling, in which resorption and formation must be finely balanced. In this context, vitamin D plays a crucial role by regulating osteoblastic and osteoclastic activity through the RANK/RANKL/OPG pathway, a system in which the balance between RANKL—which promotes osteoclastic activity—and osteoprotegerin (OPG)—its regulator—governs remodeling [6]. Available data on its specific influence in orthodontics nonetheless remain limited: some experimental studies show that optimal levels improve the bone response and limit relapse, whereas others find no significant effect, highlighting methodological limitations and interindividual variability [3] [6].

Objective: This work aims to assess the impact of vitamin D on the stability of orthodontic outcomes, focusing on its role in bone regeneration, periodontal reorganization, and relapse prevention. Secondary objectives are to study its effect on the speed and efficiency of orthodontic tooth movement (OTM), to analyze the bone-remodeling mechanisms it modulates, to appraise the relation-

ship between vitamin D status and stability, and to examine the effects of supplementation, with a view to identifying ways to integrate it into clinical protocols.

2. Materials and Methods

2.1. Search Strategy

This review, based on an electronic literature search, follows the PRISMA recommendations for handling references. The databases queried were PubMed (via MEDLINE), Springer, ScienceDirect, and Google Scholar, selected for their coverage and accessibility. Three concepts structured the search: 1) vitamin D, 2) orthodontic tooth movement, and 3) orthodontic treatment stability.

Keywords associated with each concept were combined using Boolean operators according to two equations: (*Vitamin D OR Calciferol OR Calcitriol*) *AND* (*Deficiency OR Administration*) *AND* (*Orthodontic Treatment OR Tooth Movement OR Orthodontic Tooth Movement*); and (*Vitamin D OR Calciferol OR Calcitriol*) *AND* (*Deficiency OR Administration*) *AND* (*Orthodontic Stability OR Orthodontic Relapse*). Reference management and duplicate removal were performed using Zotero.

2.2. Selection Criteria

Inclusion Criteria: articles published between 2013 and 2024 addressing the effect of vitamin D on OTM and/or orthodontic stability; systematic reviews and meta-analyses, clinical trials, retrospective and prospective studies, literature reviews, and *in vitro* and *in vivo* (animal) experimental studies; publications in English or French.

Exclusion Criteria: sponsored articles or those with conflicts of interest, case series, editorials and expert opinions, viewpoints, duplicates, and publications prior to 2013.

3. Results

The initial search identified 3792 references from Springer, PubMed, ScienceDirect, and Google Scholar. After removing duplicates and applying the date restriction (2013-2024), 2223 references were retained. Screening of titles and abstracts yielded a preselection of 47 articles; after full-text review and application of the exclusion criteria, 27 articles were definitively included (Table 1).

Table 1. Distribution of the 27 included articles by study type.

Study Type	Number of Articles
Meta-analyses and systematic reviews	6
Narrative reviews	2
Randomized clinical trials	4
Prospective clinical studies	2

Continued

In vitro studies	1
In vivo (animal) studies	10
Longitudinal studies	2
Total	27

4. Discussion

4.1. Vitamin D: Metabolism and Deficiency

Vitamin D, a fat-soluble prohormone, derives from the cutaneous synthesis of 7-dehydrocholesterol into cholecalciferol (D3) under UVB exposure, and from dietary intake or supplements. Its bioavailability depends on age, skin pigmentation, and photoprotection [7]-[10]. It then undergoes a double hydroxylation: hepatic, producing 25-hydroxyvitamin D (calcifediol), the reference circulating form of vitamin D status owing to its 2- to 3-week half-life [11]; then renal, under the action of 1α-hydroxylase, producing 1,25-dihydroxyvitamin D (calcitriol), the biologically active form, regulated by calcemia, phosphatemia, and PTH [7]. Calcitriol acts by binding to the vitamin D receptor (VDR), expressed in numerous tissues, and exerts effects that are not only mineral but also immunomodulatory and anti-inflammatory [9] [12].

A serum 25(OH)D level between 30 and 60 ng/mL is considered optimal; a value below 20 ng/mL defines deficiency, and a value of 21 - 29 ng/mL insufficiency [8] [13]. The Moroccan Society of Rheumatology recommends levels ≥ 30 ng/mL and, given a prevalence exceeding 85% among adult women, advocates strategies that combine screening of at-risk populations, targeted supplementation, and food fortification [1] [14]. In severe deficiency (<10 ng/mL), a loading phase (e.g., 50,000 IU/week for 6 to 8 weeks) precedes a daily maintenance dose of 1000 to 2000 IU [8] [13].

4.2. Vitamin D and Bone Metabolism

Bone metabolism continuously balances resorption of old bone and formation of new bone, ensuring skeletal strength and phosphocalcic homeostasis [12]. Experimental studies show that vitamin D deficiency disrupts this balance: Khalaf and Almudhi (2022) report, in the rat, a decreased RANKL/OPG ratio and reduced osteoclastic activity [15], while Gong *et al.* (2018) observe inhibition of osteoblastic formation and periodontal degeneration in the deficient mouse [16]. Conversely, calcitriol supply restores bone formation, particularly in the maxillary region [17], and supplementation increases bone mineral density (BMD) and bone volume while reducing porosity [6] [12] [18]. Several studies emphasize that vitamin D can also stimulate the controlled resorption required for tooth movement [19] [20], and that it can counterbalance the inhibitory effect of bisphosphonates on remodeling [21] (**Table 2**).

Table 2. Effects of vitamin D on bone remodeling according to the included studies.

Study	Type	Population	Observed Effect
Khalaf & Almudhi (2022)	Exp. (rats)	Induced deficiency	↓ RANKL/OPG ratio; ↓ osteoclastic activity
Narmada *et al.* (2019)	Exp. (rats)	Pregnant, supplemented	↑ RANKL; ↑ osteoclast number
Fügl *et al.* (2015)	Exp. (rats)	Vitamin D deficiency	↑ bone formation with local calcitriol
Gong *et al.* (2018)	Exp. (mice)	Vitamin D deficiency	↓ osteoblasts; periodontal degeneration
Gratton (2022)	Exp. (rats)	Systemic administration	↑ BMD and bone volume; ↓ porosity
Kazemian *et al.* (2023)	Meta-analysis	Osteoporotic patients	↑ BMD; improved bone formation
Sundar *et al.* (2023)	Syst. review	Bone regeneration (adjuvant)	Better bone formation and osseointegration

Biphasic Effect: The action of vitamin D depends closely on its concentration. At physiological levels, moderate doses reduce the RANKL/OPG ratio and support balanced remodeling [22]; in initially deficient subjects (<50 nmol/L), moderate supplementation (400 - 800 IU/day) slightly improves BMD, notably at the femoral neck [23]. Conversely, high doses (>1000 IU/day) may induce hypercalcemia without additional benefit, and supraphysiological local concentrations increase mineralization to the point of slowing tooth movement [23] [24]. A targeted and individualized approach, therefore, appears necessary to maximize benefits while avoiding deleterious effects.

4.3. Vitamin D and Orthodontic Tooth Movement

Orthodontic tooth movement relies on remodeling of the alveolar bone: bone formation on the tension side and resorption on the compression side, under the control of the RANKL/OPG pathway [25]-[29]. Most studies report an accelerating effect of vitamin D. Al-Attar and Abid (2022) report a 23.5% reduction in the alignment time of the mandibular incisors, associated with a decrease in initial pain [5]; Varughese *et al.* (2019) an increase in canine distalization speed (0.38 → 0.49 mm/week) [30]; Nishitha *et al.* an acceleration of 33.6%, comparable to prostaglandin E1 [31]. Farzanegan *et al.* (2023) observe acceleration in the second month after restoration of serum levels and regulation of the RANKL/OPG ratio [32]. Meta-analyses confirm this trend, with standardized mean differences (SMD) of 1.63 ($p < 0.05$) in Al-Attar *et al.* (2021) [33] and 1.43 ($p = 0.002$) in Tini *et al.* (2024) [34].

Synergistic effects are described with prostaglandin E2, displacement increasing from 0.231 mm (control) to 0.702 mm in combination [20], along with a "rescue" effect on remodeling under alendronate, vitamin D restoring an accelerated OTM of 21% [21]. One paradoxical result is worth noting: Shetty *et al.* (2015) observe a

slowdown (1.14 vs 1.86 mm/week) attributed to excessive bone densification under local administration [24], underscoring the importance of precise dosing (**Table 3**).

Table 3. Summary of the effects of vitamin D on orthodontic tooth movement (OTM). RCT: randomized clinical trial; PGE2: prostaglandin E2; PRP: platelet-rich plasma; SMD: standardized mean difference.

Study	Type/Route	Main Result
Al-Attar & Abid (2022)	RCT—systemic	↓ 23.5% in mandibular incisor alignment time
Varughese *et al.* (2019)	RCT—local (intraligamentary)	Canine distalization: 0.38 → 0.49 mm/week
Nishitha *et al.* (2017)	Clinical—local	↑ 33.6% in displacement speed
Navya *et al.* (2022)	RCT—local	0.42 mm/week (vs 0.32 control; 0.46 PRP)
Farzanegan *et al.* (2023)	Clinical—oral	Acceleration at month 2 (RANKL/OPG regulation)
Shetty *et al.* (2015)	Clinical—local	Slowdown: 1.14 vs 1.86 mm/week (densification)
Gratton *et al.* (2022)	Exp. (rats)—syst./local	Systemic: ↑ bone strength, OTM slowed
Moradinejad *et al.* (2024)	Exp. (rats)—systemic	+21% OTM alone; restores OTM under alendronate
Seifi *et al.* (2013)	Exp. (rats)—systemic	0.231 → 0.702 mm with PGE2 (synergy)
Al-Attar *et al.* (2021)	Meta-analysis	Increased OTM—SMD 1.63 ($p < 0.05$)
Tini *et al.* (2024)	Meta-analysis	Increased OTM—SMD 1.43 ($p = 0.002$)

4.4. Vitamin D and Orthodontic Treatment Stability

Post-treatment stability depends on complete bone regeneration and on the reorganization of the collagen fibers of the periodontal ligament (PDL), a process that can extend over several months and justifies appropriate retention [5] [12] [35] [36]. Before it is complete, residual forces tend to draw the tooth back toward its initial position; periodontal, occlusal, muscular, and anatomical factors, together with craniofacial growth, contribute to relapse [36]-[38].

Three complementary studies by Khamees *et al.* (2023) illuminate the role of vitamin D in stability. In a relapse model (0.5 N force for 14 days, 7-day retention, 7-day relapse), the deficient group (VDD) showed increased resorption, a decreased osteoblast count, and impaired regeneration, with more relapse; the supplemented group (VDS) showed greater bone deposition and reduced relapse [39]. At the root level, the cementum deposition/resorption ratio fell from 46% (control) to 31.6% in the deficiency group and rose to 68.4% under supplementation, reflecting improved cemental remodeling (**Table 4**). Finally, the PDL appeared irregular and fragmented in deficiency, hence increased mobility, but well-defined and close to normal under supplementation, confirming the action of vitamin D on hard tissues as well as soft tissues [40]-[42].

Table 4. Cementum deposition/resorption ratio according to vitamin D status (after Khamees *et al.*, 2023). CR: cemental resorption; CD: cemental deposition.

Group	n	Resorption (CR)	Deposition (CD)	CD/CR (%)
Control	10	15	6.1	46%
Deficient (VDD)	10	63	20	31.6%
Supplemented (VDS)	10	10.1	6.6	68.4%

These observations are consistent with earlier work showing that local 1,25-dihydroxyvitamin D balances resorption and deposition and stabilizes teeth after movement [43] [44], and that vitamin D stimulates osteoblastic proliferation and matrix mineralization [45]. Gratton (2022) confirms the superiority of the systemic route: gavage of 2000 IU/kg/day for 47 days improves BMD and bone volume (BV/TV) and reduces porosity, with a significant decrease in late relapse, whereas local injection has no notable effect on these parameters [12]. Taken together, the evidence supports integrating screening of vitamin D status and, where appropriate, prior systemic supplementation, particularly in patients at risk of deficiency or with a history of relapse.

4.5. Clinical Implications

Vitamin D, administered systemically or locally, constitutes a promising approach to optimizing orthodontic treatment: acceleration of OTM, reduction of pain, and support of post-treatment stability. Systemic correction of an established deficiency before the active phase, followed by maintenance of optimal serum levels during retention, appears to be the most rational lever, provided that dosing is individualized to avoid the paradoxical effects of overdosing. In a Moroccan context marked by a high prevalence of hypovitaminosis D, targeted screening of orthodontic patients could represent a simple and cost-effective measure.

4.6. Limitations

Several limitations restrict the scope of these conclusions. Studies directly targeting post-treatment stability remain few, with most data concerning bone remodeling and OTM. A large proportion of the work relies on animal models, whose transposability to humans is uncertain. The heterogeneity of protocols—force magnitude and direction, retention duration, biomarkers (RANKL/OPG, BMD), route and dosage of supplementation—together with the diversity of populations, complicates comparisons. Finally, the absence of standardized supplementation protocols hampers the development of precise recommendations. Rigorous clinical trials, in diverse human cohorts and with longitudinal follow-up, are needed.

5. Conclusion

Vitamin D appears to be a key factor in bone remodeling, orthodontic tooth movement, and post-treatment stability. By modulating the RANKL/OPG ratio and the

osteoblast/osteoclast balance, it promotes efficient tooth movement, high-quality bone regeneration, and reorganization of periodontal fibers, thereby reducing the risk of relapse—provided that dosing is appropriate, since an excess may instead densify the bone and limit tooth mobility. A promising therapeutic adjunct, it warrants an individualized approach based on the patient's vitamin D status. Further standardized, large-scale research remains indispensable to confirm these observations and establish reliable clinical recommendations.

Author Contributions

Afaf Moussaid: Literature search, data analysis, and writing—original draft. **Hajar Bouzid:** Conceptualization, supervision, writing—review & editing, and corresponding author. **Sanaa Alami:** Methodology and validation. **Amal El Aouame:** Supervision and critical revision of intellectual content.

Conflicts of Interest

The authors declare no conflicts of interest.

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