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# The Sunshine-Vitamin Deficit in the Athlete's Mouth: Vitamin D Levels May Influence the Effectiveness of Orthodontic Tooth Movement, Risk of Root Resorption and Retention

***A thorough examination connecting low vitamin D levels in sports nutrition with the changes in alveolar bone - is serum 25(OH)D an important, easily modified marker relevant to both sports medicine and orthodontics?***

## Authors

Natalia Smyczyńska, ORCID https://orcid.org/0009-0008-8213-3531 E-mail: smyczynskanatalia@gmail.com SZPZLO Warszawa Praga-Północ, Warsaw, Poland

Mikołaj Kubik, ORCID https://orcid.org/0009-0002-5875-4713 E-mail: mikolaj.kubik24@gmail.com SZPZLO Warszawa Praga-Północ, Warsaw, Poland

Marta Krzyżanowska, ORCID https://orcid.org/0009-0001-2932-0337 E-mail: krzyzanowska6@gmail.com Non-Public Healthcare Facility Mix-Med Arima, Radom, Poland

Izabela Truchel, ORCID https://orcid.org/0009-0001-6485-2435 E-mail: truchel.iza@gmail.com Corten Dental, Warsaw, Poland

Aleksandra Barszcz, ORCID https://orcid.org/0009-0000-7391-9825 E-mail: olabarszcz2000@gmail.com Lux Med Stomatologia Herbu Oksza 26, 02-495 Warszawa

Julia Szymańska, ORCID https://orcid.org/0009-0005-2847-0105 E-mail: julia_szymanska00@interia.pl Medical Center of The Medical University of Warsaw, Warsaw, Poland

Julia Kamut, ORCID https://orcid.org/0009-0005-8630-1035 E-mail: juliakamut@gmail.com Medical Center of The Medical University of Warsaw, Warsaw, Poland

Karolina Kot-Ceglarek, ORCID https://orcid.org/0009-0000-3891-3559 E-mail: kot.karolina5a@gmail.com Centrum Stomatologiczne DEMED, Wołomin, Poland

## Abstract

Approximately 56% of athletes suffer from low vitamin D levels due to a combination of reasons, including indoor training, high latitude, lack of UV-B radiation in winter, and energy deficit in sports. Furthermore, active 1,25-dihydroxyvitamin D directly modulates the RANKL/OPG system that is required for alveolar remodeling in OTM [6,8,9].

The purpose of this work is the combination of the information about vitamin D deficiency in athletes with existing data from human and preclinical studies concerning the influence of vitamin D on OTM and investigation of whether the levels of 25(OH)D in serum are the common factor affecting orthodontic outcomes in active people.

For the analysis of data, a systematic narrative review using the databases PubMed, Scopus, Web of Science, Embase and Cochrane has been carried out.

According to small RCTs on human subjects, vitamin D may promote OTM (SMD≈1.43, very low certainty, large CI; [1]) although there is no data for EARR. Studies on animals support the hypothesis of tension side consolidation [15]. While vitamin D deficiency in athletes is highly prevalent but treatable, there is currently a complete absence of direct clinical data-zero published studies worldwide-evaluating OTM, EARR, or retention specifically within vitamin D-deficient athletic cohorts, leaving the clinical impact of secondary hyperparathyroidism entirely unquantified.

Serum 25(OH)D is a relatively simple and inexpensive variable which unites sports nutrition and orthodontics. There should be more targeted research on specific groups of athletes to prove the hypothesis about possible deficiency as a variable.

## Keywords

vitamin D; 25-hydroxyvitamin D; orthodontic tooth movement; RANKL/OPG; external apical root resorption; sports nutrition; Relative Energy Deficiency in Sport; alveolar bone remodeling

## 1. Introduction

In the world of competitive sports, low levels of essential nutrients are increasingly recognized as a common and impactful issue, with vitamin D deficiency being one of the most notable examples. A comprehensive review involving 2,313 athletes revealed that around 56% showed insufficient vitamin D levels, especially in certain situations: training indoors, living in higher latitudes, and during the low-ultraviolet-B months of winter and spring [2]. These external

factors are made worse by physiological ones. A prolonged state of low energy availability-initially identified as the Female Athlete Triad and later expanded into the Relative Energy Deficiency in Sport (REDs) framework-impacts bone development and disrupts the endocrine environment crucial for maintaining bone health [3,4,5]. This leads to a clear deficiency profile: athletes training indoors in high latitudes during winter or those with restricted energy intake are often found to have low levels of serum 25-hydroxyvitamin D [25(OH)D], which can usually be corrected [6,7]. This issue revolves mainly around sports nutrition and overall bone health, and it is on this basis that this review is presented to those interested in physical culture.

Vitamin D does much more than just help with calcium absorption. Through its action on the vitamin D receptor (VDR) in osteoblasts, precursors of osteoclasts, and fibroblasts in the periodontal ligament, 1,25-dihydroxyvitamin D plays a role in regulating the balance between receptor activator of nuclear factor-κB ligand (RANKL) and osteoprotegerin (OPG)- the mechanism that connects bone resorption and formation [6,8]. The RANKL/OPG pathway also controls how alveolar bone is remodeled, allowing teeth to shift through it [9]. This reveals a seldom-explored link: a systemic nutritional state that has mainly been examined for its impact on stress-fracture risk and athletic performance is the same signaling element that a specific clinical field uses to facilitate tooth movement.

Orthodontic tooth movement (OTM) presents a unique opportunity- a measurable bioassay for observing alveolar remodeling in living humans over time. If the level of vitamin D in the body truly influences bone turnover, then the periodontium subjected to orthodontic pressure is one of the few areas where this effect should be evident in clinical results. Given the high bone turnover rate in the periodontium during OTM, this model provides an ideal platform to observe vitamin D's systemic effects on local bone remodeling. Additionally, the oral health of elite athletes has been documented as a significant concern that affects performance, making it valid to view the athlete's mouth as an important area of physiological study rather than just an afterthought. Importantly, because 25(OH)D testing is relatively inexpensive and routinely performed in athletic cohorts to monitor bone health and performance, any secondary orthodontic benefits would be captured at virtually no additional cost.

Thus, two well-established bodies of research exist side-by-side without integration. Sports medicine has thoroughly detailed the issue of vitamin D deficiency among athletes but has not considered its implications for dental health. Conversely, orthodontics has largely viewed vitamin D as a local agent used to speed up tooth movement- administered or supplemented-without investigating which patients actually suffer from a deficiency. This review aims to connect these two fields. The focus isn't solely on the speed of tooth movement, but rather on the deficiency phenotype driven by sports against a comprehensive set of outcomes: the rate of OTM, external apical root resorption (EARR)- a notable, complex iatrogenic risk associated with treatment [11]-and the issues of retention and relapse after treatment [12]. This shift in focus is intentional: vitamin D is reframed from being "a local agent to speed tooth movement" to "the overall nutritional state of active patients as a factor influencing treatment outcomes," clearly linked to the literature on REDs and bone stress injuries. The underlying aim is corrective rather than performance-enhancing-the key question is whether bringing deficient athletes up to normal levels can restore proper remodeling, not whether exceeding normal dosing results in faster tooth movement.

That ambition requires a rare level of honesty regarding what the evidence still does not support, and there are four clear warnings to consider. First, the biology behind causal OTM relies mainly on animal studies and mostly local-injection pharmacology [13,14,15]; the available human data only come from small trials on supplementation rather than phenotyping deficiencies [1,16]. Second, the data on athletes only includes systemic serum measurements:

no research has looked at alveolar turnover or OTM in athletes with deficiencies, making the systemic-to-local-alveolar transition an assumption. Third, the strong link between low 25(OH)D and fractures primarily comes from military groups [17], while solid evidence from athletes remains limited [18]; thus, using these findings to suggest an athlete phenotype involves a distinct extrapolation from military to athlete contexts. Fourth, the actual impact of any effect is truly uncertain: chronic deficiency accompanied by secondary hyperparathyroidism might enhance movement but also harm bone quality—contradicting the simple idea that "deficiency equals slow" [19]. No definitive direction is claimed here.

Following this, three research questions arise.

RQ1. How common and severe is vitamin D inadequacy among different athlete subgroups (indoor vs outdoor, latitude, season, REDs/low energy availability), and how effectively is it addressed through supplementation?

RQ2. How does vitamin D status (deficiency vs repletion) influence OTM rate, external apical root resorption, and bone consolidation/retention on the tension side in both human and preclinical studies?

RQ3. Is it possible for serum 25(OH)D to serve as an affordable, standalone pre-treatment variable that can forecast and influence orthodontic results in patients who are physically active?

## 2. Methodology

### 2.1 Design and rationale

The review aims to connect two separate areas of study that have progressed independently - the study of vitamin D status among athletes and active individuals, and the impact of vitamin D on orthodontic tooth movement (OTM). Each area was not originally designed to address the common question we are exploring here, and there are no primary studies that examine orthodontic outcomes in athletes lacking sufficient vitamin D.

### 2.2 Information sources and search

The pertinent literature was located through organized searches of PubMed, Scopus, Web of Science, Embase, and the Cochrane Library, along with manual searches of the reference lists of key syntheses on each arm [1, 2, 7, 11, 16, 17, 20]. Search terms were integrated across three categories - vitamin D / 25-hydroxyvitamin D status; orthodontic tooth movement, alveolar bone remodeling, and root resorption; and athlete, physically active population, and relative energy deficiency in sport.

### 2.3 Eligibility criteria

The exposure group included athletes and physically active individuals whose vitamin D levels were assessed [2,7], along with specific cohorts related to bone outcomes, notably military personnel [17] and collegiate athletes [18]. In the outcome group, human patients receiving orthodontic treatment for tooth movement (OTM) were considered, alongside preclinical OTM models in animals, which were included solely for mechanism analysis [13,14,15].

Vitamin D levels (whether deficient or sufficient) and/or vitamin D treatments were examined, which included both systemic oral supplements and local/periodontal injections. The distinction between local and systemic methods was monitored as a key factor.

The study compared placebo groups or those without supplements, supplemented versus placebo groups, and replete versus deficient categories, as well as untreated quadrants in split-mouth and animal studies.

The outcomes were pre-defined as three key areas: (I) the rate/efficiency of OTM; (II) external apical root resorption (EARR); and (III) bone consolidation on the tension side, including retention/relapse after treatment, in addition to secondary markers of bone turnover and RANKL:OPG levels [1,11,15,16].

Study designs included human randomized controlled trials (RCTs) and systematic reviews/meta-analyses that addressed efficacy; systematic reviews/meta-analyses and cohorts concerning athletes contributed to the exposure group; while mechanistic animal studies were included only for the mechanism section, specifically kept separate from clinical efficacy conclusions.

### 2.4 Risk-of-bias and quality appraisal

The evidence included was assessed using appropriate tools based on study design: the Cochrane risk-of-bias tool for randomized trials, RoB 2 [23], for human RCTs; ROBINS-I [24] for non-randomized human studies, including the prevalence and cohort data in the exposure group; and SYRCLE's risk-of-bias tool [25] for preclinical animal research.

### 2.5 AI.

AI was utilized for two specific purposes in this research. Text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies. Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. AI tools were used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process.

## 3. Results

The link between vitamin D and orthodontics is supported by a well-understood chain of endocrine events. Vitamin D from the skin and diet is converted in the liver to 25-hydroxyvitamin D [25(OH)D], which reflects nutritional levels in the bloodstream. Following this, it is further processed in the kidneys to create the active form known as 1,25-dihydroxyvitamin D [$1,25(OH)_2D$] [6]. This active form works by interacting with the nuclear vitamin D receptor (VDR), a transcription factor activated by binding with the retinoid X receptor. Together, they attach to vitamin D response elements, effectively altering gene expression in various skeletal and non-skeletal cells [8]. The primary endocrine function of this system involves regulating calcium levels: $1,25(OH)_2D$ enhances calcium absorption in the intestines and, in conjunction with parathyroid hormone (PTH), controls how the kidneys manage calcium and ensure it is available for bone formation [6]. When levels of 25(OH)D drop, it leads to less substrate for the kidney to convert to $1,25(OH)_2D$, resulting in reduced

calcium absorption. This causes a risk of low calcium levels, which is primarily compensated for by an increase in PTH [6]. Since VDR is present in osteoblasts, osteocytes, osteoclast precursors, and periodontal ligament (PDL) fibroblasts, $1,25(OH)_2D$ also has specific local effects on these cells, which are involved in bone remodeling. This connects dietary vitamin D levels to the local processes of remodeling [8]. It is this dual role, functioning both as a calcium regulator and as a modulator for bone cells, that positions serum 25(OH)D as a key factor in localized processes like orthodontic tooth movement.

Orthodontic tooth movement (OTM) involves a precise cycle of bone removal and formation at the tissue level, which is influenced by the mechanical forces applied to the periodontal ligament (PDL). The main molecular regulator in this process is the RANKL/osteoprotegerin (OPG) system. RANKL, which is produced by osteoblasts, PDL fibroblasts, and osteocytes, interacts with RANK receptors on osteoclast precursors, promoting their activation and differentiation. In contrast, OPG functions as a decoy receptor that captures RANKL, thereby inhibiting the formation of osteoclasts. Consequently, the ratio of RANKL to OPG determines the local interaction between osteoclasts and osteoblasts. During OTM, this ratio varies across the tooth that is under pressure: on the compressed side, RANKL levels increase, leading to bone resorption ahead of the root's movement, while on the stretched side, OPG and osteoblastic activity are more prominent, facilitating new matrix formation. Importantly, $1,25(OH)_2D$ serves as a key transcriptional regulator of RANKL and OPG in osteoblastic and stromal cells, linking vitamin D levels to the process that governs alveolar remodeling.

These two physiological processes create a consistent, though not yet verified, causal connection (Figure 1). The exposure profile driven by sports - characterized by indoor training, high latitudes, limited winter ultraviolet-B, and low energy availability - leads to decreased levels of circulating 25(OH)D. As a result, there is less substrate for the kidneys to produce $1,25(OH)_2D$ [6]. This reduction in ligand availability is anticipated to diminish VDR-mediated gene expression in osteoblasts, PDL fibroblasts, and osteoclast precursors. Such changes can disrupt the RANKL:OPG ratio and affect the calcium supply necessary for mineralization [8,9]. The expected downstream effects align with the triad of orthodontic outcomes. On the side experiencing pressure, altered VDR-dependent RANKL signaling could disrupt the formation of osteoclasts and affect tooth movement rates. On the tension side, insufficient support from $1,25(OH)_2D$ for osteoblastic mineralization would result in weaker, less mineralized bone fill, possibly increasing the risk of relapse after treatment. Additionally, since vitamin D influences the inflammatory-resorptive environment in which remodeling happens, changes in signaling may also impact the activity of cells involved in external apical root resorption (EARR), a well-known and complex risk factor in OTM, the details of which are still not fully understood [11]. A change in the local balance of RANKL:OPG, or in the cytokine environment it shapes, presents a plausible mechanism through which vitamin D levels might either increase or decrease the resorptive pressure on the root surface, although it is unclear what the actual impact would be [9,11]. This is the mechanism referenced in the literature on human synthesis, while clearly stating that inconsistent study designs make definitive conclusions difficult [16].

An alternative pathway: secondary hyperparathyroidism and ambiguity in direction.

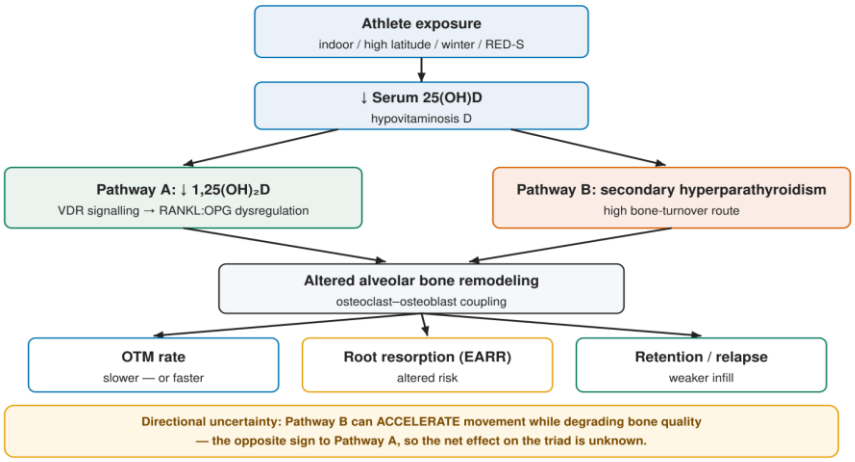
A simple interpretation of the statement-"deficiency slows and destabilises remodeling"-is not the only possibility, or even the most probable physiological outcome, and this review highlights that uncertainty without providing a resolution. Long-term vitamin D deficiency reduces serum calcium levels and leads to an increase in PTH, known as secondary hyperparathyroidism, which is well established in relation to bone loss and fractures [19]. When PTH levels remain high, it creates a remodeling condition characterized by increased activity: it boosts osteoblast-produced RANKL and overall osteoclastic activity, which mobilizes

calcium from the bones, compromising their quality [19]. In the context of orthodontically stressed periodontium, this alternative process implies the opposite effect on the rate of tooth movement-deficiency might accelerate the process by enhancing osteoclast activity-while also degrading the mineral quality of newly formed bone, which could hinder tension-side consolidation and increase the risk of root resorption. Thus, the two mechanisms exert conflicting influences on the rate of orthodontic tooth movement (OTM), even if they both point towards reduced bone quality. Consequently, the overall effect of vitamin D deficiency on OTM is truly unclear: it could result in slower movement accompanied by poor infill or faster movement with lower quality where the three aspects diverge (rate increases, retention decreases). No existing data can definitively clarify these processes, and any claim based on a single outcome would misinterpret the biological reality. This unresolved divergence remains a critical analytical issue throughout the rest of the review.

The specific evidence relating to the effect on orthodontic tooth movement (OTM)primarily comes from animal studies and focuses on local delivery methods. The original studies showing that 1,25(OH)$_2$D accelerates OTM are based on animal models, specifically cats [13] and rats, where local periodontal injections were compared directly with prostaglandin E$_2$ [14]. Similarly, enhancements in bone formation and tooth stability on the tension side following movement also stem from local injections in rats [15]. It's important to note that the physiological responses and local dosing in animals do not directly correlate with systemic nutritional deficiencies in humans, making the transition from animal studies to human implications quite significant. The data driving the conclusions around the athlete phenotype relies on systemic serum 25(OH)D levels, while the suggested effect pertains to local alveolar turnover, which has not been assessed in deficient athletes. Consequently, the assumption that systemic measures translate to local alveolar changes has yet to be verified in the specific target group. In summary, while the pathway from vitamin D receptor (VDR) biology through the RANKL:OPG axis [8,9] to orthodontic outcomes is biologically logical and has support at each step, constructing a clinical prediction for athletes with deficiencies depends on assumptions-namely, from animal studies to humans and from systemic to local effects-that the current evidence does not sufficiently support.

**Figure 1. Vitamin D and the orthodontic outcome triad: a plausible chain of uncertain direction**

How the athlete deficiency phenotype could reach the alveolus — and why the sign of the effect is genuinely unknown




**Translation gaps (core limitations):**

- Animal → human: causal OTM biology rests on local-injection models in cats (Collins 1988) and rats (Kale 2004; Kawakami 2004).
- Systemic → local alveolar: no study measures alveolar turnover/OTM in deficient athletes.
- Local dosing ≠ systemic status; military → athlete: hard bone-outcome data are military (Dao 2015), narrowed by an athlete cohort (Millward 2020).

*OTM = orthodontic tooth movement; RED-S = relative energy deficiency in sport.*

Figure 1. Vitamin D and the orthodontic outcome triad: a plausible chain of uncertain direction.

The athlete deficiency phenotype (indoor training, high latitude, winter, low energy availability/REDs) lowers serum 25(OH)D, which can act through two competing routes: Pathway A (reduced $1,25(OH)_2D$ → VDR/RANKL:OPG dysregulation) and Pathway B (secondary hyperparathyroidism, a high-turnover route). Because the two pathways push in opposite directions on tooth-movement rate, the sign of any net effect on the outcome triad (OTM rate, external apical root resorption, retention/relapse) is genuinely uncertain. Core translation gaps (animal→human; systemic→local-alveolar; local dosing ≠ systemic status; military→athlete, narrowed by [18]) are shown.

The presence of vitamin-D inadequacy is a significant issue in athletic groups, not just an occasional observation. The most comprehensive analysis combined data from 23 studies with 2,313 athletes, showing a 56% prevalence rate of vitamin-D inadequacy (95% CI 44-67%). This was determined using a serum level of 25(OH)D below 32 ng/mL (below 80 nmol/L) [2]. It is particularly striking that over half of the athletes studied did not meet this standard, especially since they are typically perceived as young, fit, healthy individuals who spend time outdoors. The wide confidence interval reflects substantial variability among the groups studied, indicating significant differences rather than errors in measurement. This variability provides valuable insight, highlighting the environmental and behavioral factors distinguishing well-supplied athletes from those lacking sufficient vitamin D. The selected threshold is crucial; the 32 ng/mL (80 nmol/L) benchmark indicates the line between adequacy and severe deficiency, illustrating how many athletes do not meet the sufficiency standard instead of telling us how many are severely deficient. Despite this, the magnitude of the issue is significant, and the commonality of this exposure, as opposed to a rare metabolic issue, emphasizes the need for action based on these findings.

This synthesis identified those gradients as reliable risk factors, which shape the deficiency phenotype that is the focus of this review [2]. The season showed a significant influence: athletes evaluated during winter or spring were substantially more prone to inadequacy than their counterparts evaluated in sunnier months (risk ratio 1.85, 95% CI 1.27-2.70), highlighting the direct impact of seasonal ultraviolet-B (UV-B) availability on skin vitamin-D production. The training environment also played an independent role: athletes who trained indoors had a higher risk compared to those who trained outdoors (risk ratio 1.19, 95% CI 1.09-1.30), indicating a small but statistically significant effect. Furthermore, prevalence differed clearly by the type of sport - mixed indoor/outdoor sports showed the highest risk of inadequacy (risk ratio 2.54, 95% CI 1.03-6.26), though this was based on a small subgroup reflected in a broad interval - and by location, which aligns with decreased solar angle in higher latitudes and differing sun-exposure behaviors in various disciplines, although the signal related to latitude was less clear and weaker. Altogether, these risk factors paint a recognizable profile of an at-risk athlete - typically one who trains indoors, competes in winter, and is located at high latitudes - who faces compromised vitamin-D levels due not to health issues, but to regular training conditions.

The factors influencing this phenotype stem from both meteorological and behavioral aspects, but an equally significant factor is physiological: insufficient energy availability. When regular energy consumption does not satisfy the combined needs of exercise and resting metabolism, it can disrupt endocrine and skeletal functions, affecting micronutrient levels and bone remodeling. The International Olympic Committee's 2023 consensus on Relative Energy Deficiency in Sport formally defines this syndrome and its far-reaching impacts on bone health [5], expanding upon the earlier framework of the Female Athlete Triad [3], which first linked disrupted energy availability to bone impairment. The underlying mechanism is quantitative:

there is a dose-response connection between lower energy availability and bone turnover, leading to decreased bone formation and increased resorption as energy availability decreases [4]. Consequently, insufficient energy availability worsens the effects of seasonal training and indoor workouts, resulting in an athlete who not only synthesizes less vitamin D but is also poised for negative bone changes. It is crucial to note that these three factors do not act independently; they often appear together. For instance, an indoor athlete training intensely during winter in a high-latitude area faces significant energy demands that hinder bone formation, resulting in risk factors clustering around specific individuals rather than spreading evenly throughout the population.

When a deficiency is widespread, the good news is that it can be easily fixed. A systematic review and meta-analysis of randomized controlled trials showed that taking oral supplements can consistently increase serum 25(OH)D levels in athletes [7]. Therefore, using this pharmacological approach is both efficient and low-cost. However, the clinical benefits are not as clear-cut: the same analysis revealed that the impact of supplementation on physical performance was varied and generally uncertain [7]. This distinction-where a biomarker can be changed reliably but the actual benefits are unclear-is crucial to express clearly. It warns against considering repletion as a performance-enhancing strategy while still allowing for the reasonable viewpoint that recognizing a common, easily measured, and correctable condition is worthwhile.

The most compelling connection between low 25(OH)D levels and stress fractures actually comes from military personnel rather than athletes. A meta-analysis of these groups showed that serum 25(OH)D levels were lower in individuals diagnosed with stress fractures, revealing a pooled mean difference of around -2.26 ng/mL (95% CI -3.89 to -0.63) [17]. While the effect is real, it is modest and observational, importantly stemming from soldiers whose training regimes, exposure patterns, and selection processes differ significantly from those of competitive athletes. Hence, using military data to suggest an athlete deficiency phenotype is more of an interpretation than a solid finding, and this distinction should be made clear in discussions. However, evidence from the athletic space is beginning to emerge: a cohort of collegiate athletes has shown a link between lower serum vitamin D levels and stress fractures [18], which aligns with military results. Nonetheless, the data from athletes [18] does not carry the same weight or design as that from military studies [17], and the existing literature regarding athlete bone outcomes is still limited and less definitive. The best interpretation is that the new collegiate findings seem to lessen the gap between military and athletic data but do not fully bridge it, making the extrapolation more plausible yet still not absolute.

Ultimately, the epidemiology discussed is crucial for a review focused on the mouth, as athletes' systemic issues affect their oral health as well. A comprehensive review concerning the oral health of elite athletes revealed a significant prevalence of oral diseases among top performers, linking these issues to their overall performance and well-being [10]. This highlights that athletes are a viable and often overlooked group in need of oral health care. The vitamin-D deficiency profile outlined here is widespread, follows specific patterns, stems from low energy availability and REDs, can be easily addressed, and is only indirectly related to bone fragility. This scenario sets the stage for the orthodontic topics explored in the next section.

Table 1. Signals of vitamin D insufficiency and bone outcomes in groups that are physically active.

| Source | Population | Measure | Estimate (95% CI) | Note |
|---|---|---|---|---|
| [2] | Athletes (23 studies; 2,313 athletes) | Vitamin D inadequacy prevalence, 25(OH)D <32 ng/mL (<80 nmol/L) | 56% (44-67%) | Wide CI reflects between-cohort heterogeneity; inadequacy (sufficiency shortfall), not frank deficiency |
| [2] | Athletes assessed in winter/spring | Risk ratio for inadequacy vs sunnier months | RR 1.85 (1.27–2.70) | Seasonal UV-B fingerprint on cutaneous synthesis |
| [2] | Indoor vs outdoor athletes | Risk ratio for inadequacy | RR 1.19 (1.09-1.30) | Modest but statistically robust; narrow interval |
| [2] | Mixed indoor/outdoor sport | Risk ratio for inadequacy | RR 2.54 (1.03-6.26) | Highest subgroup risk; wide interval (small subgroup) |
| [17] (MILITARY) | Military personnel with stress fracture | Serum 25(OH)D mean difference, cases vs controls at diagnosis | MD -2.26 ng/mL (-3.89 to -0.63) | Military, not athletes - signposted military→athlete extrapolation; modest, observational |
| [18] (ATHLETE) | Collegiate athletes | Association of lower serum 25(OH)D with stress fractures | Descriptive; direction concordant with [17] (no pooled estimate) | Genuine athlete-derived signal; narrows but does not close the military→athlete gap |

This review presents a three-part clinical question. Instead of simply exploring whether Vitamin D accelerates tooth movement, it examines how it influences the overall scope of orthodontic treatment: the speed of tooth migration, the potential for external apical root resorption (EARR) caused by this movement, and the behavior of retention and relapse in corrected teeth. These three aspects are analyzed as distinct layers of evidence (Figure 3), with a careful separation between human and animal studies, as their implications differ significantly. A key point that underlines this section is introduced right away: all human studies focus on vitamin D use-either through oral supplements or local injections—within generally healthy or nutritionally diverse participants, whereas this paper specifically addresses athletes with baseline deficiencies. No study involving humans or animals has evaluated the rate of OTM, EARR, or retention in

athletes who are deficient in vitamin D. Thus, the following triad can only be informed through extrapolation from evidence that was not originally designed to address it.

(I) OTM rate - while it is the most compelling, it remains a weak link.

The most comprehensive human synthesis available is [1], which is a systematic review and meta-analysis covering six randomized controlled trials involving humans. By combining the results of these trials, it was found that vitamin D is linked to quicker orthodontic tooth movement, showing a standardised mean difference (SMD) of 1.43 (95% CI 0.691 to 2.169) in favor of the vitamin D group compared to the control. This figure appears significant, making it the most cited statistic in the dialogue surrounding vitamin D and orthodontic tooth movement. However, it should be interpreted with caution. The wide confidence interval, the small participant numbers in individual trials, and varying protocols - such as different methods of delivery, including systemic oral doses and localized injections, along with discrepancies in appliance systems, dosages, and follow-up durations - all contribute to its ambiguity. Concerns regarding risk of bias further complicate this imprecision. Consequently, according to a GRADE evaluation, the certainty of evidence for this rate is considered low: while the direction of the effect seems generally consistent, its strength and applicability to everyday practice cannot be confidently asserted. Presenting the point estimate of 1.43 as a reliable factor for acceleration would be an exaggeration; the truthful assessment is that vitamin D, whether supplemented or injected locally, could potentially enhance movement, albeit based on low-certainty evidence.

Another limitation of the pooled estimate of 1.43, which is easy to overlook, lies in its combination of systemic and local delivery methods into one figure. By averaging oral supplements with peri-dental injections, it assumes a biological equivalence that isn't supported, which means the observed effect cannot be accurately credited to either a systemic mechanism or a local pharmacological one- the key distinction around which this review's argument about deficiencies is based.

The scoping review conducted by [20] provides a comprehensive overview of vitamin research related to OTM, showing a clear trend that indicates a focus on administration-to-accelerate methods instead of addressing deficiency correction. Meanwhile, [16], which evaluates the same human evidence, takes a much more cautious stance than what a meta-analytic point estimate might lead one to believe. This caution arises from the fact that the primary studies show significant variations in both design and reporting, making it difficult to confidently claim efficacy. Mechanistic support comes from findings in animals: both [13] with cats and [14] with rats found that when 1,25-dihydroxyvitamin D is locally injected, it accelerates tooth movement, with [14] noting effects on the rate that are fairly similar to those of prostaglandin E2. Although these studies suggest biological plausibility for a pro-remodelling effect, they highlight the first gap in translating these findings to humans. They focus on animal trials that utilize localized pharmacological delivery to the periodontium and do not reflect the systemic nutritional status of human patients, particularly those who are chronically deficient. Thus, local dosing effects observed in animals cannot be used as evidence that low systemic levels of 25(OH)D hinder movement, as these results do not confirm that relationship.

(II) EARR - genuinely inconclusive

The current evidence for external apical root resorption does not allow for any strong conclusions. In [1], the combined effect on root resorption resulted in a standard mean difference (SMD) of -0.51 (95% CI -3.051 to 2.031). Although the negative point estimate suggests a potentially protective effect, the confidence interval covers zero extensively,

indicating both possible significant harm and protective effects. This result is not significant and is derived from a small number of varied studies, where the broad confidence interval itself indicates the findings: vitamin D could worsen, leave unchanged, or benefit the roots. The GRADE rating for the EARR segment falls to very low, reflecting serious uncertainty, inconsistency, and indirectness affecting the entire area of research. It would be misleading to claim vitamin D offers protection for the roots based on this information.

The systematic review by [16], which covers OTM, EARR, and bone biomarkers collectively, arrives at a similar cautious conclusion through a different analysis: it identifies the evidence for EARR as inconsistent and refrains from making a definite conclusion. The necessary background is provided by [11], which underscores that root resorption is a prevalent and multifactorial result of orthodontic forces, meaning any potential influencing factor must contend with a noisy, high baseline outcome. The situation is further complicated by measurement issues: [27] indicates that how resorption is detected varies significantly depending on the imaging technique used, noting that cone-beam computed tomography surpasses panoramic radiography. Thus, pooled EARR estimates from studies employing different measurement tools will inherently carry extra variability. In essence, the signals being measured are weak, the tools for obtaining these measurements are inconsistent, and the confidence interval suggests the issue is still unresolved.

(III) Retention and relapse - having promising mechanisms but lacking clinical testing.

The third limb is the slimmest. The only supporting evidence that exists is from animal studies and mechanistic data.[15] indicated that injecting 1,25-dihydroxyvitamin D directly into the site increased bone formation, which helped stabilize teeth after they were experimentally moved in rats, serving as a preclinical model for the retention phase.This narrative aligns well with the biological idea of tension-side consolidation, but it is based solely on a rodent study involving local injections. No human orthodontic research has provided evidence of retention or relapse related to vitamin D post-treatment. In light of the comprehensive evaluation of retention methods by [12], which highlights both the significance of achieving stable long-term outcomes and the generally low quality of evidence surrounding retention, the issue of vitamin D's role in retention remains largely unexplored in humans. The level of confidence in this limb is quite low, largely mechanistic in nature, suggesting that it should be recognized as a notable gap in research rather than an established clinical fact.

When considering all factors, the conclusion is imbalanced. Evidence, which has low certainty, suggests that supplemental or locally administered vitamin D may speed up the rate of orthodontic tooth movement, but its impact on external apical root resorption (EARR) has not been demonstrated, and the time frame is significantly irrelevant.The retention advantages are currently only backed by animal studies. [16] pointed out that the varied study designs hinder drawing dependable conclusions, which serves as the highest realistic assessment for the entire field.Emphasizing a key constraint: all human data results from giving vitamin D to either restore levels in patients or those not selected for deficiency, and no research has specifically focused on vitamin-D-deficient athletes, the focus of this review. Therefore, it is essential to clearly differentiate between animal and human studies, as well as local versus systemic vitamin D status, rather than merging these categories quietly.

The directional warning mentioned before emphasizes a crucial aspect. If chronic deficiency primarily leads to secondary hyperparathyroidism and a high-turnover bone condition, then an

athlete with a deficiency may, in theory, exhibit quicker movements that aren't as high in quality - resulting in a split in the triad where speed increases while retention and foundational integrity decline. No existing dataset can confirm this situation, highlighting the importance of measuring the entire triad instead of focusing solely on rate in future athlete studies.This approach of connecting the triad with phenotype also sets this synthesis apart from catalog reviews like [21], which merely identify vitamin D as one of many pharmacological influences, lacking either a deficiency phenotype or an athletic perspective.

Table 2. Vitamin D and the orthodontic outcome triad. The effect estimates represent the meta-analytic figures reported by [1], included with their original confidence intervals; GRADE certainty is used for the evidence obtained.

| Outcome | Best evidence | Effect (95% CI) | Direction | Certainty (GRADE) |
|---|---|---|---|---|
| OTM rate | [1] (SR/MA, 6 human RCTs); animal rate support: [13], [14] | SMD 1.43 (0.691-2.169) | Favours faster OTM; administration only (systemic + local pooled), not deficiency correction | Low |
| EARR | [1] (SR/MA); [16] (cautious) | SMD -0.51 (-3.051 to 2.031) | Non-significant; CI spans harm and protection — do not claim root protection | Very low |
| Retention / stabilization | [15] (rat, local injection); no human data | (no human estimate) | Animal-only tension-side consolidation signal; untested in humans | Very low |

Orthodontic therapy primarily unfolds over time. Typically, treatments involving fixed appliances last between eighteen to thirty months, and each extra month spent in these devices results in more time in them, increased appointment frequency, and a longer duration of the daily challenges that such treatment brings. For patients who are active, this situation presents a significant challenge. The presence of appliances can hinder the proper fit of customized mouthguards and increase the likelihood of soft-tissue injuries during contact sports. Additionally, they may temporarily impact speech, create discomfort during physical activity, and change the facial appearance, which can be important for competitors as well as for fulfilling sponsorship and media responsibilities associated with high-level sports. These factors likely influence how consistently athletes follow treatment - including wearing elastics, using retainers, and attending adjustments - all of which affect how long treatment lasts and the stability of the results. In this context, any common and easily modified factor that could help normalize the pace and effectiveness of alveolar remodeling deserves attention, as it directly influences the critical factor of treatment duration, which impacts the athlete’s psychosocial experience. The literature on athlete oral health already recognizes that dental issues can affect

performance rather than being solely cosmetic [10], highlighting the importance of viewing the length of orthodontic treatment as a concern within sports medicine as well as dentistry.

The economic reasoning aligns with the same principles and is better expressed as a rationale instead of a detailed financial statement, as no study has evaluated this approach in terms of cost. When needed, a serum 25(OH)D test, followed by oral cholecalciferol supplementation, represents one of the most affordable and lowest-risk treatments available in any clinical setting [28]; this supplementation consistently increases circulating 25(OH)D levels in athletes [7]. In contrast, the opposing costs-such as prolonged use of appliances, additional chairside appointments, and addressing negative outcomes like external apical root resorption or post-retention relapse-are significant in terms of the time required from clinicians, the burden on patients, and the necessary re-treatment for relapses. Root resorption poses a real, multifactorial risk associated with tooth movement [11], and keeping track of it and managing it involves its own financial implications. Thus, there is a structural imbalance: a low-cost, one-time assessment compared to ongoing, recurring costs. If addressing a common and easily identifiable deficiency even slightly enhanced the efficiency or stability of remodeling, screening active patients would likely hold positive value. This condition is intentional-the orthodontic advantages are not yet proven, as the human factor is limited to a small, varied rate effect and an inconclusive resorption assessment [1]-so the argument relies on the low cost and biological soundness of the approach rather than any confirmed savings.

The framing of equity and opportunity cost clarifies the argument. Many athletes experience vitamin D deficiency, particularly among specific groups such as those who train indoors, those in high-latitude areas, athletes who train in winter, and those with low energy availability [2,3,5]. It is crucial that many individuals in these groups are either already monitored or should be monitored for 25(OH)D levels because low levels are linked to stress fractures in military personnel [17] and at least one group of collegiate athletes [18]. In sports medicine, replenishing vitamin D is a common practice. Thus, the additional cost of using the existing test for an orthodontic purpose is minimal-one biomarker could benefit two fields (showing shared-biomarker efficiency as noted in H1). On the other hand, choosing not to investigate amounts to spending nothing while missing out on easily accessible information.

This leads to a conclusion with little regret. Finding and treating vitamin D deficiency is warranted for an athlete's health, regardless of orthodontic considerations: it addresses a recognized and common deficiency [2] with an intervention known to be safe and effective for improving levels [7,28], and it may provide benefits for bone strength[17,18]. Therefore, the reasoning for orthodontics is supplementary, not essential. The focus should always be on correcting the deficiency to normalize remodeling instead of aiming for supra-physiological doses to hasten tooth movement. The aim is the screening and repletion of patients who are deficient rather than enhancing those who already have adequate levels. Economic and psychosocial arguments do not replace the need for efficacy evidence; they merely indicate that the risk of taking action is low and justified on its own, while the potential benefits for orthodontics, although unproven, would be gained at nearly no extra cost.

Table 3. Evidence map: outcome triad × evidence tier. Cells list the studies supplying each type of evidence for each triad outcome; the corresponding GRADE certainty (Table 2) is low for OTM rate and very low for EARR and retention. The athlete-deficiency column is empty for all three outcomes - the core evidence gap this review identifies.

| Outcome | Animal (local injection) | Human SR/RCT (supplementation) | Athlete-deficiency cohort |
|---|---|---|---|
| OTM rate | [13]; [14] | [1]; [20] | none |
| EARR | (none) | [1]; [16]; [11] (baseline context) | none |
| Retention / relapse | [15] | none | none |

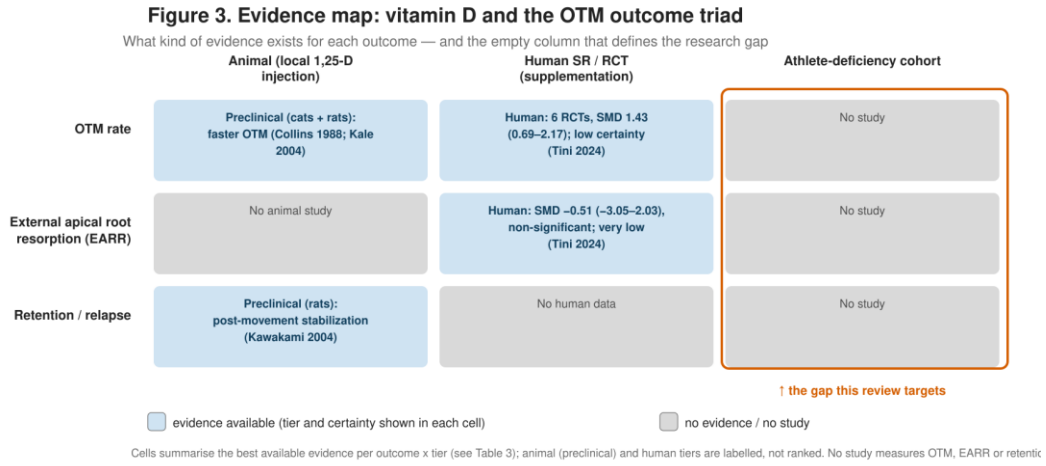



Figure 3. Evidence map: vitamin D and the orthodontic outcome triad. Best available evidence for each outcome (rows: OTM rate; external apical root resorption; retention/relapse) across three evidence tiers (columns: animal local-injection; human supplementation SR/RCT; athlete-deficiency cohort). Cells are shaded by whether evidence is available or absent; the tier (animal/preclinical vs human) and the certainty are stated within each cell. The athlete-deficiency column is empty for all three outcomes - the gap this review targets. Content corresponds to Table 3.

## 4. Discussion

This review brings together two areas of literature that have not previously been considered alongside each other: the epidemiology of vitamin D deficiency in physically active individuals and the biological role of vitamin D in orthodontic tooth movement (OTM). This connection is intentional, yet it remains speculative throughout. There has been no research assessing the rate of OTM, external apical root resorption (EARR), or post-treatment retention in athletes with low vitamin D levels; the information regarding bone-related outcomes influencing the athlete's profile is derived partly from military groups [17] and is only somewhat clarified by data from actual athletes [18].Additionally, research involving human OTM mostly explores vitamin D administration in primarily unscreened patients [1,16], rather than addressing the correction of existing deficiencies. Therefore, the unique contribution of this paper lies not in providing a definitive effect estimate but rather in proposing a testable new perspective - considering serum

25(OH)D as a shared, easily adjustable nutritional status for active patients, treating it as an outcome covariate in the orthodontic clinic instead of merely a quickening adjunct. This perspective is articulated through three specific, testable hypotheses, each accompanied by a clear testing method and a condition under which it would be dismissed. In line with the ongoing mechanistic uncertainty (the pathway involving chronic deficiency and secondary hyperparathyroidism that might increase rather than reduce turnover), the potential direction of any effects remains open-ended, and no hypothesis is presented as a confirmed finding among athletes.

4.1 H1 - "The shared-biomarker hypothesis"

Claim. Serum 25(OH)D represents a shared variable that influences two clinically different outcomes: the risk of bone-stress injury in active individuals and the effectiveness of alveolar remodeling during OTM. If this holds true, a single, affordable measurement could provide valuable insights for both sports medicine and orthodontics, allowing both fields to legitimately share the use of this biomarker.

The evidence supporting the two limbs is valid but uneven and originates from distinct groups. On the side of bone stress injury, a lack of vitamin D is frequently seen among athletes and varies by location [2]. This deficiency can be effectively treated with supplements [7]. The link between low levels of 25(OH)D and stress fractures is moderate and moderate and based on observations, becoming most evident in military members [17] and somewhat confirmed in a true collegiate athlete group [18]. Regarding orthodontic remodeling, vitamin D influences the RANKL/OPG pathway responsible for the coupling of osteoclasts and osteoblasts on both pressure and tension sides [9]. Reports involving humans suggest a possible directional impact on the rate of orthodontic tooth movement, though the effects on external apical root resorption remain unclear [1,16]. The central idea is that both aspects reflect the same fundamental ability for organized bone remodeling under mechanical or metabolic stress. If vitamin D status partially shapes that ability, it stands to reason that one measure might provide relevant information for the other. However, an essential point is that there is currently no dataset linking these two aspects within the same individuals, meaning the shared-axis assertion is now more of an inference drawn from two separate bodies of evidence rather than a correlation within individuals. Additionally, the strength of the two limbs varies; the remodeling aspect is based on mechanistic, small randomized controlled trials, while the injury aspect relies on observational studies and different populations.

Falsification/Test: A forward-looking study of physically active individuals where initial levels of 25(OH)D are compared regarding the occurrence of bone stress injuries and alveolar remodeling indicators/orthodontic movements, designed to focus on the weaker of the two relationships. Hypothesis 1 is disproven if baseline 25(OH)D forecasts one outcome yet remains statistically irrelevant to the other, suggesting no shared foundation exists.

It is important to note that the bone-stress-injury aspect still heavily depends on military data [17]; reference [18] refines but does not fully dismiss the extrapolation from military to athlete, as training and exposure patterns differ, and a common biomarker cannot be assumed until both aspects are evaluated together in athletes.

4.2 H2 - "The deficiency-as-covariate hypothesis"

Claim. The condition of untreated athlete hypovitaminosis D results in slower, less stable, and more resorption-prone OTM, which serves as an unrecognized confounding factor in studies evaluating orthodontic outcomes among active patients. Consequently, this deficiency phenotype - characterized by indoor training, high latitudes, winter sessions, and exposure to

REDs [2,5] - is subtly incorporated into orthodontic groups, leading to biased outcome comparisons.

Evidence supporting this claim is largely indirect. The triad of outcomes has some backing from both mechanistic and human-synthesis perspectives: a slight signal for OTM rate [1], unclear evidence of EARR [1,16] against a baseline of EARR being a genuine, multifocal OTM risk [11], and a common, correctable deficiency phenotype [2]. However, all human studies on OTM assess administration in either non-selected or fully sufficient patients, which means H2's fundamental assumption - that existing baseline deficiency negatively impacts outcomes - has never been directly validated. The logic of confounding underlines H2's practical implications: if deficient athletes are disproportionately found in the slower, less stable, or more resorption-prone segments of orthodontic cohorts, then research that overlooks 25(OH)D will incorrectly attribute variations to devices, force systems, or biological factors, while a manageable nutritional issue remains unconsidered. Therefore, treating 25(OH)D as a predefined covariate rather than merely an afterthought represents the minimum adjustment that the hypothesis necessitates.

Falsification/Test: An orthodontic cohort investigated prospectively, sorted by baseline 25(OH)D levels (deficient versus sufficient), with key endpoints of OTM rate, EARR measured by CBCT [27], and relapse after retention [12].In this scenario, 25(OH)D should be included as a predefined covariate, alongside adjustments for REDs and training intensity [5]; if adjusted baseline 25(OH)D shows no correlation with any endpoint in the triad, then H2 is disproven.

A necessary caution regarding direction clarifies the prediction of "slower. " Chronic lack can lead to secondary hyperparathyroidism and a high-turnover condition [6,19], which may actually speed up movement while compromising bone quality - a reversal of the idea that "deficiency = slow" along the rate axis, although the prediction of instability/resorption may still be valid concerning quality and retention. Thus, it is essential for the test to assess the entire triad instead of just the rate, as the endpoints could diverge in their directions.

### 4.3 H3 - "The repletion-normalization (not acceleration) hypothesis"

Assertion. Fixing deficiencies brings back a balanced RANKL/OPG-driven coupling - improving efficiency, root protection, and infill on the tension side [9,15] - while excessive doses do not provide additional benefits and could even have negative effects. The expected dose–response follows a plateau or ceiling at sufficiency rather than a continuous increase; this is about replenishing a deficit, not using drugs to enhance the periodontium, which differentiates this review from acceleration-focused studies [20].

The reasoning is based on the physiology of replenishment and comparisons of doses, rather than any trials focused on correcting deficiencies. Guidelines outline sufficiency goals and caution against excessive dosing [28]; supplementation tends to reliably increase 25(OH)D levels in athletes but shows varied effects on performance [7]; research in animals indicates that local 1,25-D speeds up rates similarly to PGE2 [14] and helps with stabilization after movement [15]; human studies have been conducted with diverse doses and delivery methods [1], which is why it is crucial to establish a dose-finding approach before making any claims about efficacy. Mechanically, the expectation of a plateau makes sense: when VDR-mediated signaling reaches saturation at physiological sufficiency, extrasubstrate should not increase

balanced RANKL/OPG coupling proportionately, and too much may disrupt calcium management or the resorptive environment instead of enhancing it [8]. Clinically, this leads to a clear rejection of the "supplement-to-accelerate" perspective: the aim is to normalize to sufficiency, not to maximize; thus, a favorable result for H3 indicates a ceiling, not a slope.

Falsification/Test: In a three-part RCT involving active patients with deficiencies-comparing no supplement,physiological repletion to adequate levels, and supra-physiological dosing- the goal is to investigate whether there is a plateau or ceiling in the dose-response relationship regarding OTM rate, EARR, and the filling of tension-side bone.Hypothesis H3 would be proven false if a consistent dose-response continues into the supra-physiological range (suggesting "more leads to faster"), or if repletion shows no benefit compared to ongoing deficiency.

### 4.4 Reconciling the directional-uncertainty tension

All three hypotheses hinge on a single unresolved mechanistic fork that needs to be openly acknowledged instead of glossed over. The simple expectation is that a deficiency hinders and makes remodeling unstable. However, an alternative belief is that long-term deficiency, through secondary hyperparathyroidism, creates a high-turnover situation [6,19] that might speed up tooth movement but result in lower-quality, under-mineralised bone- leading to quicker movement at the expense of diminished retention and possibly altered EARR. These reflect conflicting signs on the rate axis, but they could align regarding the quality axis. Currently, there is no dataset capable of resolving this distinction, which is precisely why the proposed tests evaluate the complete outcome triad and allow H2's endpoints to diverge. Regardless of the outcome on the rate axis, deficiency remains a likely influence on the quality of remodeling, and measuring 25(OH)D should be the covariate retained.

### 4.5 Research agenda

The most significant and informative output is the study that is missing from all these literatures: focused groups of athletes that address the gap in the athlete-deficiency section of Table 3. The key priorities include (i) OTM trials that are baseline-deficiency stratified, capturing the complete triad - rate, CBCT-measured EARR [27], and retention/relapse [12]; (ii) standardized 25(OH)D assays along with clear sufficiency thresholds so that effects can be compared across different studies [2,28]; (iii) assessment of alveolar turnover markers in athletes who are deficient to directly examine the systemic→local transition [9,16]; (iv) identifying the appropriate dosage before making efficacy claims [1,7]; and (v) pre-registered collaborative data gathering from sports medicine and orthodontics concerning REDs and training load adjustments [5], including the characterization of energy availability as it relates to its dose-dependent effects on bone turnover [4], ensuring that one biomarker is relevant for both areas. Regarding RQ3 - whether serum 25(OH)D can serve as a singular, affordable pre-treatment variable that predicts and influences orthodontic success in active patients - this question is addressed not by current findings but through the proposed screening initiative and agenda: it represents a hypothesis waiting to be explored, rather than a confirmed result.

### 4.6 Practice-facing screening message

Until more data is available, there is a cautious, reasonable approach worth considering. For physically active orthodontic patients - particularly those training indoors in high-latitude locations during winter or athletes at risk for REDs [2,5] - measuring and correcting 25(OH)D

could be included as part of the standard pre-treatment evaluation.This measurement is cost-effective, the correction process is reliable and carries low risk [7], the biomarker is frequently tracked for reasons related to bone health and athletic performance, and oral health in athletes is recognized as a relevant factor for performance [10]. This is suggested as a logical and hypothesis-aligned practice based on its own health benefits, rather than as a mandated orthodontic requirement: currently, there are no direct OTM data for athletes, and the value of screening lies in addressing a prevalent deficiency rather than guaranteeing quicker orthodontic results.

## 5. Limitations

This review's findings are impacted by a series of translation gaps that we highlight rather than downplay, and readers should consider this when evaluating any causal statements.

Dependence on animals for human data. The causal relationship between OTM and vitamin D relies heavily on pharmacological studies that involve local injections in animals like cats and rats [13,14,15]. The internal validity of these study designs may vary when assessed with tools specific to those animals [25]. In humans, the evidence comes mainly from small randomized controlled trials on supplementation [1], rather than on identifying deficiency characteristics, making it difficult to confidently apply these findings to clinical predictions.

Jumping from systemic to local alveolar effects. Data from athletes consist of systemic serum 25(OH)D measurements [2]. However, no research has looked at alveolar bone turnover or OTM in athletes with deficiencies.Thus, the transition from circulating vitamin D levels to the pressures and tensions in the orthodontically loaded periodontium is based on inference, not direct observation.

Extrapolating military data to athletes. The strongest evidence linking low 25(OH)D levels to stress fractures comes from military groups, where effects are often modest and sometimes not statistically significant [17]. While a more targeted collegiate-athlete group helps narrow the gap [18], it does not eliminate it due to differing training loads and exposure patterns; thus, the athlete-injury aspect of hypothesis H1 remains an extrapolation.

Local dosing does not equal systemic status. In human OTM trials, researchers administer vitamin D either through supplementation or local delivery to generally replete or non-selected patients [1]. These trials do not address existing baseline deficiency. Consequently, the claim that deficiency acts as a covariate spans two bodies of evidence and has never been analyzed within a single athletic group.

Scant, varied evidence with broad intervals. Human OTM samples are small and differ in their protocols and dosing, resulting in wide confidence intervals - the EARR estimate covers a range well beyond null findings - leading to low to very low certainty ratings when formally assessed [26]. It's essential that we do not make broad assumptions about the EARR effect [16]. We intentionally refrain from recalibrating these estimates into a singular figure.

No data on athlete OTM exists. The main issue is clear: no research presents information regarding OTM rate, EARR, or retention in athletes lacking vitamin D. The evidence for athlete deficiency is entirely absent, and every statement concerning RQ2/RQ3 is limited by this gap.

Uncertainty in direction. Chronic deficiency accompanied by secondary hyperparathyroidism might speed up movement while harming bone quality [6,19], creating genuine ambiguity regarding the effect's direction, leading us to state that it does not lean toward any one side.

Narrative, non-registered design. This work presents a critical narrative synthesis organized methodically without being a registered systematic review, lacking a counted screening base, and no new meta-analysis was executed [22].Variability in assay cut-offs and the potential for unpublished negative findings further restrict the evidence gathered.

## 6. Conclusion

The synthesis reveals that serum 25(OH)D serves as a biologically reasonable, low-cost factor connecting sports-nutrition vitamin D deficiency to the triad of orthodontic outcomes: movement rate, external apical root resorption, and retention/relapse. However, this relationship is inferred rather than directly demonstrated in athletes. The most solid finding suggests a slight indication that vitamin D supplementation or its local delivery could enhance OTM rate [1]; however, the EARR impact remains uncertain [16], and support for the retention aspect exists only in animal studies[15]. Importantly, no research has evaluated any of these outcomes in athletes who are vitamin D deficient. The direction of any potential effect remains truly ambiguous, as chronic deficiency with secondary hyperparathyroidism could lead to quicker movement but at the cost of bone quality [6,19], and acknowledging this is not a way to hedge but a crucial finding.

The review's main contribution lies in its reframing approach rather than merely estimating effects. It shifts the perspective on vitamin D, moving it from a local adjunct for acceleration to one that is integrated into the overall nutritional status of an active patient, serving as a covariate for treatment outcomes. This change is connected to the literature on REDs and bone stress injuries [2,5,17], making it possible to test this new framing through three hypotheses that can be proven false, involving specific groups of athletes. The lack of evidence regarding athlete deficiency is not a flaw in the synthesis but a crucial finding, highlighting the potential for sports medicine and orthodontics to collaborate on a common biomarker with minimal cost.

For readers of Quality in Sport, the practical takeaway aligns with the existing evidence. Addressing a common deficiency that is easy to detect is a low-risk action, already supported by independent research on bone and overall health [7]. The potential benefits for orthodontic outcomes, however, still need confirmation from dedicated athlete studies. In summary, track what is inexpensive and shared, and while waiting for better information - evaluate it before making any orthodontic adjustments to an athlete's teeth.

## Declarations

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