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CONTEXT-DEPENDENT EFFECTS OF VITAMIN D SUPPLEMENTATION ON BONE MINERAL DENSITY AND FRACTURE RISK: A NARRATIVE REVIEW ACROSS CLINICAL AND ATHLETIC POPULATIONS

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ABSTRACT

Background: Vitamin D plays a well-established role in calcium absorption and bone metabolism, yet its effectiveness as a supplementation strategy has been increasingly questioned, with fractures representing the most serious complication of deficiency-related bone loss. These questions are particularly relevant in athletic populations, where vitamin D insufficiency is widespread and has been linked to bone stress injuries despite high levels of physical activity.

Objective: This narrative review synthesises evidence from both clinical and athletic populations to identify when and for whom vitamin D supplementation is most likely to confer measurable skeletal benefit.

Results: In athletic populations, vitamin D deficiency is widespread yet does not consistently impair bone mineral density, most likely because mechanical loading compensates for its effects. In genuinely deficient athletes, targeted supplementation may help reduce stress fracture risk, though randomised trial evidence remains limited. In clinical populations, vitamin D alone does not reliably reduce fracture risk or improve bone mineral density in unselected individuals. More consistent benefits emerge when calcium is combined with vitamin D, particularly in postmenopausal women, institutionalised elderly, and patients on pharmacological osteoporosis treatment, which provides useful comparative context for understanding the conditions under which supplementation is most likely to benefit athletes and active individuals.

Conclusions: The skeletal effects of vitamin D are highly context-dependent, influenced by baseline deficiency, calcium intake, physical activity, and population characteristics. Routine universal supplementation is not justified. A targeted approach based on confirmed deficiency and individual clinical context is recommended across both populations.

KEYWORDS

Vitamin D, Bone Mineral Density, Fracture Risk, Athletic Populations, Calcium

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1. Introduction

Osteoporosis is a global, metabolic bone disease associated with the aging of the immune and skeletal systems [1], [2]. It is a chronic disease most frequently observed in older populations [1], [3]. It is characterized as a condition resulting in skeletal fragility due to deterioration of bone microstructure, bone density and bone strength [2], [3]. Osteoporosis is frequently linked to increased risk of fracture, with a predisposition to having recurrent fractures if left untreated [2]. The consequences of such accidents can range from disability (for example day to day movement restrictions caused by vertebrae fractures) to mortality, as illustrated by osteoporotic hip fracture, which carries approximately a one in five chance of death within the first year [1], [2]. The seriousness of possible fracture complications highlights the urgent need in their prevention.

Vitamin D plays a crucial role in bone remodelling. It regulates calcium–phosphate balance primarily by promoting intestinal calcium absorption, which supports bone mineralisation and structural integrity [3], [4]. Adequate serum vitamin D concentrations suppress parathyroid hormone (PTH) secretion. Low vitamin D levels cause bone loss because of increasing bone turnover through PTH stimulation, which is not followed by promotion of bone growth [3]. Vitamin D deficiency is highly prevalent among older adults and postmenopausal women, driven by reduced sunlight exposure, age-related decline in cutaneous synthesis, hormonal changes, and inadequate dietary intake [5], [6]. Nevertheless, even though vitamin D seems to be a crucial factor determining bone health and endurance, evidence dating to at least 2016 has highlighted inconsistencies in the clinical impact of supplementation on bone density and fracture rates [7]. In addition to inconsistent evidence regarding vitamin D supplementation, real-world management of osteoporosis remains suboptimal, with only moderate adherence to guideline-recommended pharmacotherapy in primary care settings [2], [8].

Earlier evidence suggested a positive link between calcium and vitamin D supplementation on managing osteoporotic fracture risk [9]. However, recent large meta-analyses and randomized trials have questioned the effectiveness of vitamin D supplementation in reducing fracture risk [10], [11], [12]. Evidence suggests there is no significant link of vitamin D or calcium supplementation on fracture risk or fall reduction overall [10]. This absence of effect extends further to healthy midlife and older adults who were not previously diagnosed with osteoporosis or vitamin D deficiency, as well as community-dwelling older populations [11], [12]. Beyond traditional high-risk populations, interest has expanded to athletes, in whom vitamin D insufficiency is prevalent and has been linked to stress fracture risk, although the effectiveness of supplementation in improving bone outcomes remains uncertain [13].

While the clinical and athletic literature on vitamin D and bone health have largely developed simultaneously, direct comparison of supplementation outcomes across these populations remains limited. This narrative review synthesises evidence from both domains to evaluate whether the context-dependence of vitamin D's skeletal effects observed in clinical populations is similarly reflected in athletic cohorts, and to identify the conditions under which supplementation is most likely to result in measurable benefit. The findings are of particular relevance to sports medicine practitioners and athletic support staff seeking evidence-based guidance on vitamin D monitoring and supplementation in competitive and recreational athletes.

2. Methods

Data search for the review was conducted using major databases such as PubMed, Scopus, Google Scholar. Search terms included combinations of 'vitamin D', 'vitamin D deficiency', 'calcium', 'bone mineral density', 'osteoporosis', 'fracture risk', 'calcium supplementation', 'athletes', and 'athletic populations'. Language exclusion criterion was applied, only including for consideration papers available in English or Spanish. An age inclusion criterion was applied qualifying only papers published between 2006-2026. Special attention was paid to papers published within the last 2 years, as they provided valuable and new insight into the researched topic.

3. Results

Understanding the clinical evidence on vitamin D and bone health in osteoporotic populations provides essential context for interpreting findings in athletic cohorts, where bone stress, deficiency prevalence, and supplementation outcomes present a distinct but related challenge.

While vitamin D deficiency is consistently associated with accelerated bone loss in clinical populations, studies have shown that active vitamin D metabolites stimulate osteoclast development and therefore in high doses may increase fracture and fall risk [7].

3.1. Vitamin D alone

Large meta-analyses provide the strongest evidence against vitamin D monotherapy. A 2018 systematic review and meta-analysis of 81 randomised controlled trials found no clinically meaningful effect of vitamin D supplementation on fracture or fall prevention, or bone mineral density, with no difference observed between higher and lower doses [14]. An accompanying editorial argued that these results call into question the widespread use of vitamin D supplements for bone and muscle health [10].

The VITAL trial reinforces this conclusion. The randomised placebo-controlled study of 25,871 generally healthy midlife and older adults, found that vitamin D supplementation did not result in a significantly lower risk of fractures compared with placebo. Participants were not selected based on vitamin D deficiency, low bone mass, or osteoporosis [11]. A different meta-analysis of 33 randomised trials in community-dwelling older adults found no association between supplementation with calcium, vitamin D, or both, and reduced fracture risk [12]. Similarly, a 2019 systematic review and meta-analysis found that neither intermittent nor daily dosing with standard doses of vitamin D alone was associated with reduced fracture risk, simultaneously noting potential benefit of vitamin D and calcium supplementation on reducing the fracture risk [15].

Importantly, this finding is also supported by studies in populations with high deficiency prevalence. A community-based study of 1,723 individuals in Saudi Arabia, a population with well-documented high rates of vitamin D deficiency, found no correlation between vitamin D levels and bone mineral density across any age group or sex [16]. This further suggests that deficiency alone may not reliably predict impaired bone density and that correction may not automatically restore it.

Taken together, these findings suggest that vitamin D supplementation in unselected populations confers little skeletal benefit. However, evidence from studies using combined supplementation protocols presents a more nuanced picture.

3.2. Combined Vitamin D and Calcium

Combined supplementation produces more consistent results than vitamin D alone, particularly in postmenopausal women and institutionalised populations. A systematic review and meta-analysis of randomised controlled trials found that combined calcium and vitamin D supplementation had a favourable effect on bone mineral density and could prevent hip fracture in postmenopausal women [17]. A meta-analysis of 19 randomised trials confirmed that calcium combined with vitamin D significantly reduced fracture incidence and enhanced BMD, however, was also linked with no difference to mortality rate and as a potential source of gastrointestinal problems with increasing doses [18].

Benefits are especially pronounced in high-risk institutional settings. A systematic review of pharmacological management in nursing home populations, a population in whom deficiency and dietary inadequacy are reliably severe, found that 1,200 mg calcium combined with 800 IU vitamin D reduced fracture risk and improved bone mineral density in the elderly residents [19].

More recent evidence supports these findings. A 2026 study of 50 healthy individuals with asymptomatic hypovitaminosis D found that one year of cholecalciferol and calcium supplementation produced a significant rise in serum vitamin D levels. It was further associated with increased BMD, however, no impact on trabecular bone score was observed [20]. A 2025 study in Indian osteoporotic patients similarly reported significant improvements in BMD and reduced fracture risk following combined supplementation [21]. A one-year calcium and vitamin D intervention was associated with increased BMD and improvements in physical performance, with greater gains observed in patients under 70 years of age [22]. Older patients showed reduced bone loss compared to the non-supplemented group.

Adherence is a consistent moderator of outcomes. Evidence indicates that patients who adhere to supplementation recommendations show significantly greater fracture risk reduction [9]. The relative contribution of calcium versus vitamin D in producing these outcomes remains debated.

These findings suggest that the clinical context in which combined supplementation is applied, such as baseline deficiency, age, institutionalisation, and adherence substantially determines its efficacy. The following section examines these modifying factors in greater detail, with particular focus on populations in whom context-dependent effects are most clinically relevant.

3.3. Context-dependent modifiers and clinical management

3.3.1. Menopausal transition.

The menopausal transition accelerates bone loss through hormonal shifts that alter calcium metabolism. A 2026 study found significant associations between serum calcium levels and lumbar spine BMD, with greater BMD decline in individuals with high FSH levels, suggesting that monitoring serum calcium and FSH during the menopausal transition may be important for bone protection [23]. Vitamin D deficiency in this population is both prevalent and undertreated as shown by a study of 314 women over the age of 40 with osteoporosis [24]. These findings take on additional significance when considered alongside evidence that peak bone mass is achieved in young adulthood, and that approximately half of women over 50 will experience an osteoporotic fracture. A 2026 study of 260 healthy women aged 18–30 found that plant-based dietary patterns were not independently associated with bone density once diet quality, physical activity, and body composition were accounted for, suggesting that vitamin D status rather than dietary pattern may be the more meaningful determinant of bone mass development in this critical life stage [25].

3.3.2. Baseline deficiency state and dietary intake.

Whether supplementation is effective appears to depend largely on a patient's baseline vitamin D and calcium levels. A study from 2016 showed that among patients with osteoporotic fracture, approximately 80% had calcium and vitamin D intake below recommended levels. Dietary calcium intake was positively associated with BMD, while low calcium intake showed no relationship with family history of osteoporosis, suggesting it reflects habitual behaviour rather than genetic predisposition [26]. Similarly, data from a study conducted in France showed that at least half of postmenopausal osteoporotic women were consuming less vitamin D than recommended, indicating that a large proportion of this population may benefit from supplementation simply by meeting basic intake requirements [27].

3.3.3. Pharmacotherapy context.

Vitamin D sufficiency appears to be a prerequisite for the optimal functioning of pharmacological osteoporosis treatments. In bisphosphonate-treated patients experiencing BMD decline, vitamin D insufficiency was identified as the most frequent cause [28]. Correction of insufficiency in these patients led to a significant increase in BMD. For patients receiving denosumab, co-administration of vitamin D and calcium prevented the post-treatment decline in serum calcium caused by denosumab monotherapy [29]. It additionally inhibited bone resorption to a greater extent, and increased BMD, particularly at the hip. A 2025 systematic review found that in postmenopausal women undergoing pharmacological treatment for osteoporosis, vitamin D supplementation was associated with reduced gastrointestinal adverse events and lower mortality, while calcium supplementation showed no significant association with any of the measured outcomes [30]. These findings suggest that vitamin D monotherapy or combined vitamin D and calcium may be superior to calcium supplementation alone in this context.

3.3.4. Adherence and real-world practice.

Even where evidence supports supplementation, real-world adherence limits its impact. A 2026 study examining osteoporosis management in primary care found that prescribing practices were only moderately aligned with guidelines, with doctors more likely to correctly identify who needed treatment than to prescribe the most appropriate first-line drug [8]. Current (2025) clinical guidance has moved away from universal supplementation, instead calling for treatment plans tailored to each patient's individual fracture risk, with calcium and vitamin D recommended as supportive measures within a broader care strategy rather than as treatments [1].

3.4. Athletic populations

3.4.1. Prevalence of deficiency.

Vitamin D insufficiency is common in athletic populations, with prevalence varying by sport, season, and geography. In one collegiate cohort of female dancers and cheerleaders, 74% of tested athletes had insufficient serum vitamin D levels, with both groups showing mean concentrations below recommended thresholds despite engaging in over 20 hours of physical activity per week [31]. Athletes with limited sun exposure, indoor training environments, and high training loads are particularly at risk, and seasonal variation in both diet and sunlight exposure contributes to fluctuations in serum vitamin D levels [13], [32].

3.4.2. Effect of supplementation.

Vitamin D insufficiency has been linked to elevated stress fracture risk in athletes, with a serum vitamin D value below 75.8 nmol/L identified as a clinically relevant threshold, alongside other contributing factors such as female sex, prior inactivity, menstrual disturbances, and reduced bone thickness [33]. Stress fractures affect around 20% of all athletic competitors, and some data suggest that daily supplementation with 800 IU vitamin D and 2,000 mg calcium may reduce their prevalence, with intake recommendations for athletes reaching up to 2,000 IU per day [33]. However, when put to the test in a randomised controlled trial, weekly 4,000 IU vitamin D over 12 weeks produced no significant improvement in bone mass or performance in collegiate basketball players compared to placebo, with the authors suggesting that the training stimulus itself likely outweighed any supplementation effect [34]. A study of collegiate acrobatics and tumbling athletes pointed in the same direction. Despite low vitamin D levels, BMD was high and no link between vitamin D status, BMD, and injury was found, again pointing to physical loading as a likely compensatory factor [35]. Overall, a recent review of vitamin D in sport concludes that supplementation benefits on bone remain inconsistent and that routine monitoring with individualised supplementation is preferred over blind administration of high-dose protocols [36].

4. Discussion

Table 1. Comparison of vitamin D supplementation effects on skeletal outcomes across clinical and athletic populations

Outcome	Clinical populations	Athletic populations	Comparison
Mechanistic basis	Strong role in calcium absorption and PTH regulation [3], [7]	Same mechanism applies but may be masked by training load [13], [32]	Biological plausibility does not guarantee clinical effect
Baseline deficiency	Deficiency prevalent but does not reliably predict reduced BMD [14], [16]	Highly prevalent; seasonal variation and indoor training contribute [13], [31]	Deficiency is contributory but not sufficient to predict poor bone outcomes
BMD (bone mineral density)	Vitamin D alone shows minimal or no improvement [11], [14]	No consistent association, mechanical loading may dominate [31], [34]	Vitamin D is not a primary driver of BMD in either group
Fracture risk	No reduction with vitamin D alone, modest benefit with calcium combination [12], [15]	Stress fracture risk associated with deficiency in observational studies [33], [35]	Effects appear context-dependent rather than universal
Combined effect of vitamin D and Ca	Modest benefit in selected high-risk groups [17], [18]	Targeted supplementation in deficient athletes may reduce stress fracture risk, randomised trial evidence remains inconclusive [33], [34]	Benefits mainly in deficient or high-risk individuals

Taken together, the available evidence reveals a consistent gap between the well-established biological role of vitamin D and its variable effects when used as a supplement. Vitamin D is central to calcium absorption, phosphate balance, and PTH regulation. Yet despite this solid mechanistic foundation, supplementation alone does not reliably lead to meaningful improvements in bone mineral density, fracture prevention, or other skeletal outcomes.

Looking at clinical and athletic populations side by side, a similar pattern emerges in both groups. Deficiency is common, the underlying biology is the same, yet universal supplementation does not consistently improve bone health in either context. The difference lies in what is driving bone problems in each population. In older adults and postmenopausal women, bone loss is primarily hormonal and metabolic in origin, meaning that correcting a vitamin D deficit alone is not enough without additional pharmacological intervention. In athletes, high mechanical loading appears to offset the negative effects of deficiency on BMD, as shown in the acrobatics cohort which maintained high BMD despite low vitamin D levels and the basketball trial where the training stimulus outweighed any supplementation benefit [34], [35]. Where athletes do seem to benefit is in stress fracture prevention when genuinely deficient, suggesting that vitamin D becomes most clinically relevant in this population under conditions of acute skeletal stress [33].

5. Limitations

The studies included in this review vary in population characteristics, supplementation doses, and follow-up duration, which reflects the broader heterogeneity of the literature in this area. Evidence concerning athletic populations remains more limited compared to clinical populations, consistent with the relative novelty of this research area. Further randomised trials in athletic cohorts would help consolidate future conclusions. Adherence variability across the included trials may also have influenced some of the reported outcomes, and future studies incorporating standardised compliance monitoring would provide a clearer picture of real-world supplementation effects.

6. Clinical Implications

The evidence points toward targeted rather than universal supplementation in both populations. In clinical practice, combined calcium and vitamin D is most justified in people with confirmed deficiency, postmenopausal women, residents of care facilities, and patients on pharmacological osteoporosis treatment, where adequate vitamin D is a known prerequisite for optimal drug efficacy [28], [29], [30]. For athletes, the priority should be routine monitoring of serum vitamin D with individualised supplementation, particularly in those at high risk of stress fractures, rather than applying blanket high-dose protocols without prior testing [33], [36]. Supplementing healthy, non-deficient individuals in either population is not supported by the available evidence. More broadly, these findings suggest a need to move away from viewing vitamin D as a standalone intervention for bone health, and toward treating it as one supportive element within a wider, more individualised approach to skeletal care.

7. Conclusions

The evidence supports a model in which vitamin D functions as a contributory but not sufficient factor in establishing bone health, with **effects that vary across clinical and athletic populations**. Its clinical effectiveness is highly context-dependent, influenced by calcium intake, baseline deficiency, physical activity levels, and population characteristics. Therefore, vitamin D should not be considered an independent therapeutic agent for fracture prevention but rather an adjunctive component of a broader nutritional and lifestyle-based strategy. Future research should focus on identifying specific subgroups that may benefit from targeted supplementation and clarifying the interaction between vitamin D status, mechanical loading, and calcium metabolism in skeletal health.

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