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THE ROLE OF VITAMIN D IN MUSCLE PERFORMANCE AND INJURY PREVENTION IN ATHLETES

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ABSTRACT

Vitamin D, a fat-soluble secosteroid, is traditionally associated with the regulation of calcium and phosphate metabolism; however, its role also extends to muscle function and immunity. The active form of this vitamin, 1,25-dihydroxyvitamin D, acts through receptors in muscle tissue (VDR), stimulating protein synthesis and regulating intracellular calcium balance. In this way, it supports proper muscle strength and structure. Vitamin D deficiency disrupts these mechanisms—leading to atrophy of type II muscle fibers, impaired mitochondrial function, and reduced muscle strength and bone density. The result is an increased susceptibility to injury, slower regeneration, and a prolonged recovery period, particularly in athletes. Studies confirm that vitamin D deficiency is common even among physically active individuals, especially those who train indoors or have limited sun exposure. Regular vitamin D supplementation effectively raises serum 25(OH)D levels, reduces the risk of stress fractures (by more than 75% in some groups), and improves functional outcomes, especially when combined with appropriate training. However, in individuals with adequate vitamin D levels, additional supplementation does not provide further performance benefits. Regular monitoring of 25(OH)D concentration and an individualized approach to supplementation—taking into account risk factors such as skin type, geographic latitude, and training environment—are essential. While some methodological limitations are present in the scientific literature, the available evidence suggests that maintaining adequate vitamin D levels plays a key role in preventing injuries and supporting both bone and muscle health among physically active individuals.

KEYWORDS

Vitamin D, Athletes, Muscle Function, Injury Prevention, Supplementation, 25(OH)D, Bone Health

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1. Introduction

Vitamin D is a fat-soluble secosteroid hormone classically known for its role in calcium-phosphate homeostasis and bone health. [1] In recent years, there has been a growing appreciation for the broader physiological importance of vitamin D. Vitamin D plays a crucial role in immune function, protein synthesis, muscle function, cardiovascular function, inflammatory response, cell growth, and the regulation of the musculoskeletal system. [2], [3], [4], [5]

Concerning the musculoskeletal system, vitamin D in the body acts by stimulating the absorption of calcium and phosphorus in the intestine, stimulating the mobilization of calcium in the bone, and increasing the reabsorption of calcium in the distal tubules. Bone resorption, modeling, and remodeling are also considered to be vitamin D-dependent processes. Vitamin D must be metabolized to 25-hydroxyvitamin D3 by the liver and then to 1, 25-dihydroxyvitamin D3 by the kidneys. 1, 25-dihydroxyvitamin D3 binds to a specific receptor in the intestinal nucleus to stimulate calcium transport. 1, 25-Dihydroxyvitamin D3, acting through its receptor, causes the transcription of particular genes that encode proteins responsible for transporting calcium and phosphorus. Only one protein, a calcium-binding protein, has been identified as being vitamin D-dependent. [6], [7], [8] Vitamin D also indirectly influences other processes occurring due to the presence of calcium, including neurotransmission, blood clotting, and secretion, by increasing absorption and reducing loss from the body.

A breakthrough in the assessment of the effects of vitamin D on the body was the discovery in 1979 of the presence of VDR receptors (vitamin D receptors) in tissues and organs not involved in the calcium-phosphate metabolism. The discovery of vitamin D receptors (VDR)

in muscle fibers has prompted research into how vitamin D status might influence athletic performance and injury risk. The Vitamin D receptor (VDR) has been positively associated with skeletal muscle mass, function and regeneration. Vitamin D activates many metabolic processes in muscle tissue. Calcitriol is a

calcitropic hormone, so it can only affect those cells that have specific receptors for it. [9] It therefore acts via two types of receptors: nuclear - VDR (through gene activation) and membrane, the role of which is less known. Among the genes activated by the vitamin D-linked VDR receptor are bone proteins, as well as proteins responsible for intracellular calcium transport and phospholipid metabolism. By stimulating protein synthesis, the number of type II muscle cells increases, and thus, muscle speed and strength increase. The result of the cooperation of activated proteins is muscle contraction. Calcitriol plays an additional role in muscle cell proliferation, differentiation, and inhibition of apoptosis. [10] Vitamin D deficiency causes serious health complications in the form of reduced mobility and weakening of the antigravity muscles. [11]. In adults, in some cases of severe vitamin D deficiency (<10 ng/ml), generalized muscle weakness, especially proximal, of the upper and lower limbs, has been described. Even mild vitamin D deficiency (<20 ng/ml) reduces skeletal muscle strength, and its supplementation improves functional efficiency, reduces the risk of falls, and accelerates recovery from fractures in people undergoing rehabilitation. Vitamin D supplementation supporting regular physical activity is even more effective. [10] In clinical studies, VDR expression is elevated and associated with recovery from muscle damage and is acutely increased (1–3 h) following resistance exercise [12], suggesting an association with muscle growth and maintenance. The study showed a positive role of enhanced VDR expression on skeletal muscle, showing that gain of function of VDR autonomously stimulates hypertrophy associated with increased AKT/mTOR anabolic signalling, muscle protein synthesis, ribosomal biogenesis and satellite cell activation. VDR expression and Vitamin D processing enzymes are significantly correlated with RET-induced hypertrophy in humans. Based on these data, VDR expression may be a robust marker of the hypertrophic response to resistance exercise. [13] VDR polymorphisms in humans are associated with muscle strength and falls. [14] Ablation of the VDR in mice has been shown to lead to impaired motor coordination, shorter stride length, and abnormal swimming. [15], [16]

Vitamin D deficiency is not uncommon among athletes, both amateur and professional, despite careful nutrition and training. The prevalence of deficiency depends on many factors. However, existing published data emphasize that the main cause of vitamin D deficiency in athletes is residence in areas above 40° north latitude, especially in the winter months, when clothing covering a large part of the body surface is usually worn and training takes place indoors. [17], [18], [19], [20] This is partly because the human body synthesizes this vitamin after exposing the skin to sunlight containing ultraviolet radiation. Nevertheless, a high incidence of vitamin D deficiency has been described in young and adult professional athletes living both in low (e.g., Russia, Ireland) and high (Spain, Qatar, Australia) sun exposure regions. [21], [22], [23], [24] Even athletes who regularly exercise outdoors for extended periods of time during the summer months may still have insufficient 25(OH)D concentration, which may partly be attributed to exercise-induced stress. [25], [26], [27], [28] The estimated prevalence of vitamin D deficiency in athletes, depending on the study, is around 50%, similar to the general population worldwide. [29] Low 25-hydroxyvitamin D [25(OH)D] levels in athletes have been linked to muscle weakness, decreased endurance, and increased injury incidence in some studies. [1], [30], [31]

This report reviews current evidence (primarily from the last 5 years) on the role of vitamin D in physical performance and the prevention of muscle injuries in athletes. We summarize recent peer-reviewed studies and clinical trials, discuss physiological mechanisms, and highlight clinical implications for sports medicine practice, including screening and supplementation guidelines.

2. Methodology

To prepare this review, a comprehensive search of scientific literature was conducted, focusing on publications from the years 2011–2024. Databases such as PubMed, SPORTDiscus, and Web of Science were systematically searched using combinations of the following keywords:”vitamin D, "“athlete, "“muscle, "“performance, "“strength, " and”injury." Priority was given to randomized controlled trials (RCTs), systematic reviews, meta-analyses, and authoritative clinical guidelines in the field of sports medicine. Studies included in the analysis were those evaluating vitamin D status or supplementation in relation to muscle strength, physical performance, recovery, or the incidence of musculoskeletal injuries among physically active individuals and athletes. All information was derived from peer-reviewed sources. No new clinical trials were conducted within the scope of this review.

3. Results

Physiology and Mechanisms of Vitamin D Action in Muscles

Vitamin D exerts multidirectional effects on skeletal muscle physiology through both genomic and non-genomic pathways. Following its conversion into the active form, 1, 25-dihydroxyvitamin D (calcitriol), it binds to the vitamin D receptor (VDR) in muscle cells, modulating the transcription of genes involved in myocyte proliferation and differentiation. [32] Notably, vitamin D stimulates the expression of genes promoting muscle growth, particularly in fast-twitch type II fibers, and inhibits their atrophy. [33] Histopathological studies of muscle biopsies from individuals with severe vitamin D deficiency have demonstrated type II fiber atrophy accompanied by fatty infiltration; these changes improved upon normalization of vitamin D levels. [34] The genomic actions of vitamin D enhance muscle protein synthesis and mitochondrial function, whereas its non-genomic mechanisms influence calcium homeostasis within muscle fibers. [35] For instance, binding of vitamin D to membrane-associated VDR increases calcium uptake into the sarcoplasmic reticulum and promotes actin-myosin cross-bridge formation, leading to immediate enhancement of muscle contraction. [36] The above mechanisms indicate that maintaining optimal vitamin D concentrations is essential for proper muscle function. Deficiency is a well-documented cause of myopathy and muscle weakness; in osteomalacia, patients often report diffuse muscle pain and weakness that resolve following vitamin D treatment. [37] Adequate vitamin D levels help preserve muscle fiber integrity, support neuromuscular functions, and may accelerate regenerative processes due to their anti-inflammatory properties and stimulatory effects on muscle cell renewal. [38]

Vitamin D Status in Athlete Populations

Beyond its theoretical importance, many athletes exhibit suboptimal vitamin D levels. Cross-sectional studies have shown that 40–70% of athletes have insufficient or deficient vitamin D status (depending on the definition and the population studied). [39] Athletes who train indoors, those at higher latitudes, or during winter months generally present lower 25(OH)D concentrations than athletes practicing outdoor sports in sunny climates. [40] One study reported that, during winter, the mean 25(OH)D level among professional football players was approximately 16 ng/mL, and over 90% fell below the threshold considered sufficient. [41]

Risk factors for vitamin D deficiency in athletes include limited sun exposure (training early morning, late evening, or in indoor facilities), darker skin pigmentation, increased adiposity (as vitamin D is sequestered in adipose tissue), and inadequate dietary intake. Moreover, high training loads may alter vitamin D metabolism or increase its requirement. [42] Table 1 summarizes recent studies on vitamin D levels in athletes and associated outcomes. It is noteworthy that many athletes do not meet the vitamin D intake recommendations established for the general population. [43]

As discussed later in this review, the high prevalence of vitamin D insufficiency has rendered its monitoring and supplementation an important consideration in sports nutrition and sports medicine.

Vitamin D and Musculoskeletal Injury Risk

Numerous studies indicate that an adequate serum 25(OH)D concentration is a significant modulator of musculoskeletal injury risk. Available evidence suggests that vitamin D deficiency may adversely affect both bone strength—thereby increasing susceptibility to stress fractures—and muscle function, which predisposes to strains and muscle fiber tears. The underlying mechanisms include calcitriol's role in maintaining normal bone mineral density and myocyte function. Moreover, insufficient vitamin D intake can impair regenerative processes and prolong healing of damaged tissues, reflecting its involvement in the modulation of the inflammatory response. For these reasons, maintaining optimal levels of vitamin D metabolites is considered an important component of preventing both bony injuries and soft-tissue damage in physically active individuals.

The relationship between serum 25(OH)D concentration and prevention of skeletal muscle injuries remains incompletely understood. Some observational studies suggest that athletes with lower vitamin D levels may be more prone to acute muscle damage. [44] Biologically, deficiency of 1, 25-dihydroxyvitamin D is associated with reduced muscle strength and increased vulnerability of muscle fibers to high-intensity exercise–induced injury. For example, a study of NFL players reported a higher incidence of muscle strains and deep-tissue injuries in those with vitamin D deficiency (<20 ng/mL) compared to athletes with sufficient levels; however, this was not a randomized analysis. [45]

Despite these observations, the data are not uniformly consistent. A case–control study of 56 professional footballers in Turkey found no significant difference in mean 25(OH)D concentrations between

those who experienced a muscle strain and those who did not—both groups averaged around 16 ng/mL. [46] Additionally, no association was observed between vitamin D metabolite levels and recovery time following muscle damage. [47] Similarly, a 2021 systematic review by Jakobsen et al., which included nine studies examining the link between vitamin D and overall musculoskeletal injury incidence, found that only two studies demonstrated a statistically significant association between low vitamin D levels and increased injury rates, while the remaining studies did not confirm such a relationship. [48] The authors emphasize that, at present, there is no consistent evidence that vitamin D deficiency directly increases the risk of soft-tissue injuries or overuse injuries in athletes. It should be noted that any potential effect of vitamin D on muscle injury susceptibility may be subtle or confounded by other factors, such as training load or baseline muscle strength. In summary, although maintaining adequate 25(OH)D concentrations may help reduce injury risk by preventing muscle weakness, current knowledge does not allow a definitive conclusion that supplementation or correction of deficiency alone significantly lowers muscle injury rates in athletes.

In contrast to muscle injuries, the relationship between 25(OH)D status and stress-fracture risk is much better documented. Vitamin D plays a critical role in bone mineralization, and its deficiency leads to decreased bone mineral density and accumulation of micro-damage, thereby promoting stress fracture development. Numerous reports indicate that athletes with persistently low 25(OH)D levels are at greater risk for stress fractures. Jakobsen et al.'s literature review showed that in seven studies, 25(OH)D concentrations below 30 ng/mL were consistently associated with elevated fracture risk. [50] Meta-analytic results further confirm that inadequate vitamin D increases the likelihood of these injuries. [51] Importantly, interventional trials demonstrate a causal relationship: correcting deficiencies reduces stress-fracture incidence. In a prospective study of collegiate athletes, Williams et al. found that cholecalciferol supplementation (commonly 4, 000 IU daily during winter) decreased stress-fracture rates from 7.5% in prior seasons to 1.6% during the supplementation period—an over 75% reduction. [52] These significant improvements suggest that maintaining optimal vitamin D status—especially in combination with calcium—enhances bone strength and helps reduce the risk of stress fractures. Comparable results have been observed in military recruits, where daily supplementation with 800 IU of vitamin D and calcium led to a lower incidence of fractures compared to placebo. [53] Collectively, existing evidence clearly indicates that maintaining appropriate 25(OH)D levels is an essential element in preventing stress fractures in athletes and may also support overall musculoskeletal integrity.

Table 1. Key studies on vitamin D, sports performance, and injury risk

Study (Author, Year)	Population/ Research Project	Key Concepts
Han et al., 2019 (Systematic review and meta-analysis) [54]	5 randomized controlled trials (RCTs) of vitamin D_3 supplementation in 163 athletes (multiple sports); 4–12 week interventions	Vitamin D supplementation increased 25(OH)D levels to sufficient values but did not significantly improve muscle strength (e.g., 1-RM) compared with placebo. It can be concluded that vitamin D does not have a general ergogenic effect on strength in athletes. [54]
Sist i in., 2023 (RCTs meta-analysis) [55]	11 RCTs including 436 athletes; evaluation of the effect of vitamin D on strength (1-RM tests) and power (vertical jump)	No strong evidence for improved maximal strength or power with vitamin D supplementation in athletes. Small, statistically insignificant benefits were observed only in those with baseline deficiency; no effect if 25(OH)D levels ≥ 75 nmol/l (~30 ng/ml). Practical implication: correcting deficiency is important, but excess vitamin D does not improve performance in athletes with adequate levels. [55]

Jakobsen et al., 2021 (Systematic review and meta-analysis).[56]	16 studies (observational and interventional) on vitamin D status and sports injuries (stress fractures and other musculoskeletal injuries)	Vitamin D levels <30 ng/ml were associated with an increased risk of stress fractures; a meta-analysis showed an increased risk of stress fractures with vitamin D deficiency. For other musculoskeletal injuries (strains, overuse injuries), the evidence was inconsistent, with only 2 of 9 studies showing an association with low vitamin D levels. Conclusion: Vitamin D deficiency is a clear risk factor for stress fractures, but the data for muscle/tendon injuries are equivocal. [56]
Yoon et al., 2021 (Narrative review) [57]	A review of vitamin D in athletes (including prevalence of deficiency, impact on performance, injuries, and supplementation recommendations)	It highlights the high incidence of vitamin D deficiency in athletes and the negative impact of low levels on muscle strength, power, and endurance, as well as the increased incidence of stress fractures and possibly acute muscle injuries. It recommends maintaining 25(OH)D levels >32 ng/ml (ideally >40 ng/ml) in athletes and routine monitoring. It suggests daily supplementation of 2, 000–6, 000 IU in individuals with low levels. [57]
Williams et al., 2020 (Prospective cohort with intervention) [58]	High-risk collegiate athletes (e.g., track and field, cross-country, N=118) followed in fall/winter; vitamin D deficient individuals were given D_3 supplements; comparison with historical control group	Athletes with vitamin D deficiency were given approximately 4, 000 IU of vitamin D_3 daily. The incidence of stress fractures decreased from 7.51% to 1.65% after supplementation (p=0.009). There was a significant reduction in the number of stress fractures in the supplemented group compared to earlier seasons. This suggests that supplementing vitamin D deficiency in athletes at risk significantly reduces the incidence of bone injuries. No adverse effects were observed. [58]

Abbreviations: 25(OH)D – 25-hydroxyvitamin D (circulating form of vitamin D) RCT – randomized controlled trial

4. Clinical Implications for Sports Medicine

Recommendations for 25(OH)D Screening in Athletes

Current research highlights several important clinical implications for vitamin D management in sports medicine. Foremost among these is the recommendation for routine screening. Numerous studies have documented a high prevalence of vitamin D insufficiency or deficiency among various athletic populations, emphasizing that such deficiencies may negatively impact both strength and endurance, while also increasing the risk of injury and infection. The etiology of low vitamin D status is multifactorial, including factors such as race, decreased cutaneous synthesis due to limited sunlight exposure, and inadequate dietary intake. As a result, several authors advocate for the routine assessment of vitamin D status in athletes, particularly through measurement of serum 25-hydroxyvitamin D [25(OH)D] concentrations. This assessment should be prioritized in athletes belonging to risk groups, if not universally implemented [66]. Specific risk factors for deficiency include younger age (under 18 years), low body fat percentage, and darker skin pigmentation. Additional high-risk groups include athletes who predominantly train indoors, individuals residing at latitudes above 35° N/S, those wearing full-body clothing (such as swimmers in full suits or Muslim sportswomen), and athletes with a history of stress fractures or recurrent illnesses. In these populations, screening aims to identify individuals

with deficient (<20 ng/mL) or insufficient (20–30 ng/mL) vitamin D levels, who are most likely to benefit from targeted intervention [67]. Serum 25(OH)D levels are typically assessed via blood testing, optimally conducted in late winter, when concentrations are expected to be at their lowest [66]. An alternative approach involves biannual measurement—once in early spring and again in late summer—to account for seasonal variation. Some experts also recommend adapting screening schedules to align with training intensities and competition calendars, providing individualized monitoring for athletes across different disciplines [67]. Vitamin D status is influenced by seasonal sunlight exposure, with levels typically declining from August through December, as well as by the training environment—indoor athletes generally display lower concentrations than their outdoor counterparts. Dietary habits and variability further affect serum levels, underscoring the need for regular monitoring. For most athletes, annual or biennial assessment of vitamin D may suffice to ensure optimal 25(OH)D concentrations are maintained [66].

Target 25(OH)D Values for Athletes

An essential consideration in sports medicine is the determination of optimal serum 25(OH)D concentrations for athletes. Evidence indicates that a serum 25(OH)D level of 30 ng/mL marks the threshold distinguishing sufficiency from deficiency, with normal reference ranges typically defined as 30–50 ng/mL (75–125 nmol/L) or, for building physiological reserves, 40–60 ng/mL (100–150 nmol/L). Concentrations between 20–30 ng/mL (50–75 nmol/L) are classified as insufficient, and values below 20 ng/mL (<50 nmol/L) denote deficiency [66]. For athletic populations, it has been established that the target serum 25(OH)D should reside within the mid-sufficient range. Maintaining a minimum of >30 ng/mL (75 nmol/L) is recommended, with an optimal goal of 40–50 ng/mL to ensure an adequate buffer above the seasonal winter nadir. Organizations such as the American College of Sports Medicine and the Academy of Nutrition and Dietetics of Canada emphasize that serum concentrations of 32–50 ng/mL (ideally above 40 ng/mL) are associated with improved training adaptation and physiological function in athletes [66]. Conversely, maintaining serum vitamin D at levels significantly above 50–60 ng/mL has not been associated with additional health benefits, underscoring the importance of achieving sufficiency without exceeding recommended upper limits [68]. Another critical aspect involves strategies for correcting and maintaining vitamin D status. There are three primary approaches: increased natural sunlight exposure, artificial UVB radiation, and oral supplementation—the latter being the most commonly utilized in clinical practice. While each method presents both potential benefits and risks, current evidence supports that the positive outcomes of intervention strongly outweigh any adverse effects and are far preferable to leaving deficiency untreated [67]. When athletes are identified as deficient or insufficient in vitamin D, supplementation becomes the first-line intervention. Clinical data indicate that targeted supplementation effectively raises serum 25(OH)D concentrations, particularly in those engaged in endurance training. Although both vitamin D_2 and D_3 preparations are capable of increasing serum levels, vitamin D_3 (cholecalciferol) is generally preferred due to its superior efficacy and bioavailability, a result of its greater affinity for vitamin D–binding proteins compared to either vitamin D_2 or ergocalciferol (a combination of D_3 and D_2) [66, 67]. Standard recommendations for athletes typically range from 2, 000 to 6, 000 IU of vitamin D daily, tailored to individual needs based on baseline status and risk factors. Emerging research suggests that, in select cases, dosages exceeding 10, 000 IU may be required to achieve sufficiency, though the long-term safety and clinical outcomes of such high-dose regimens warrant further investigation [66]. A personalized approach to vitamin D supplementation strategy is recommended, tailored to each athlete's individual needs. Nevertheless, one should aim to increase and maintain serum concentrations > 30 ng/mL. To this end, the Clinical Practice Guidelines of the Endocrine Society suggest a dose of at least 1, 500–2, 000 IU/day, taking into account factors affecting blood levels such as baseline 25(OH)D concentration, BMI, age, and serum albumin concentration. The following equation may be used to estimate the required dose:

Dose = [(8.52 – Desired change in serum 25(OH)D (ng/mL)) + (0.074 × Age) – (0.20 × BMI) + (1.74 × Serum albumin concentration) – (0.62 × Baseline serum 25(OH)D (ng/mL))] / (–0.002)

Current evidence supports that regular, continuous low-dose vitamin D supplementation—administered daily or weekly—is both more effective and safer than intermittent high-dose injections. This preference is based on findings from clinical studies, such as a trial in rugby players where supplementation with 70, 000 IU per week, compared to 35, 000 IU per week over three months, resulted in greater conversion of vitamin D into its inactive metabolite, 24, 25$(OH)_2$D. As a result, there was a reduction in the concentration and bioavailability of the physiologically active form of vitamin D. Notably, elevated levels of the inactive

metabolite persisted even after discontinuation of supplementation, leading to a marked decrease in serum 25(OH)D and 1, 25(OH)$_2$D concentrations. In cases of moderate deficiency, evidence from a randomized controlled trial involving 20 healthy men with serum 25(OH)D below 20 ng/mL showed that supplementation with 4, 000 IU daily for six weeks significantly increased serum 25(OH)D levels, which in turn activated repair and regenerative processes in skeletal muscle. Similarly, research involving taekwondo athletes with vitamin D deficiency found that daily supplementation with 5, 000 IU for four weeks during winter produced a substantial rise in serum 25(OH)D concentrations. Despite the demonstrated efficacy of supplementation in correcting deficiencies, preventive strategies aimed at maintaining adequate levels are preferable. Regular screening is therefore essential, with assessments ideally conducted at the end of summer—when vitamin D stores are at their peak—and at the end of winter, when levels typically reach their lowest point [69].

Vitamin D supplementation is considered safe when administered within the recommended dosage range. Maintaining serum 25(OH)D concentrations between 30–60 ng/mL is associated with optimal immune, muscular, and skeletal function in athletes, while remaining within established safety margins. However, because vitamin D is fat-soluble, it can accumulate in body tissues if consumed in excessive amounts, a scenario sometimes observed among athletes who use large or prolonged doses [69]. Although the risk is low, excessive or disproportionate intake—particularly mega-doses—may lead to complications such as hyperphosphatemia, hypercalcemia, and hypercalciuria, which in turn can result in organ dysfunction. Over-supplementation can disturb the calcium-phosphate balance and impair organ systems, even in individuals with a history of vitamin D deficiency. For this reason, it is essential to practice caution and conduct regular blood testing to monitor vitamin D status and ensure that concentrations remain within the safe and effective range [67]. It is also important to note that co-nutrients like magnesium and vitamin K can enhance the positive effects of vitamin D on bone and muscle health. Therefore, combining vitamin D supplementation with other fat-soluble vitamins may offer additional benefits [69]. In conclusion, sports health professionals should take a proactive approach to assessing and managing vitamin D status in athletes. Vitamin D supplementation is a straightforward and cost-effective strategy for addressing a common nutritional deficiency, thereby supporting musculoskeletal health and potentially reducing the incidence of bone injuries. Ensuring that vitamin D deficiency is not an unrecognized limiting factor for an athlete's performance, strength, or recovery is of practical importance. Nevertheless, there is no evidence that achieving serum concentrations above the recommended range provides further benefit; thus, resources should instead be allocated to evidence-based interventions that directly enhance athletic performance, such as training optimization, recovery strategies, nutrition, and sport-specific skill development.

5. Recommendations

Vitamin D plays a pivotal role in maintaining the proper function of both the musculoskeletal and immune systems, as well as supporting tissue regeneration. Among athletes, adequate vitamin D status is particularly important, as it influences physical performance, facilitates recovery, and contributes to the prevention of both injuries and infections. Vitamin D$_3$ (cholecalciferol) is the preferred form for supplementation, given its greater efficacy in increasing serum 25(OH)D levels compared to vitamin D$_2$ (ergocalciferol). This superiority is attributable to vitamin D$_3$'s higher bioavailability and its stronger binding affinity for the vitamin D–binding protein, which translates into a longer biological half-life and more robust physiological effects [70]. The vitamin D requirements of athletes are not uniform and are shaped by factors such as exercise intensity, duration and type of training, body composition, and environmental conditions—including the amount of UVB exposure. Particular attention should be paid to so-called"deficiency windows, " which are periods during the training season when vitamin D levels may decline sharply. For this reason, decisions regarding initiation or adjustment of supplementation should be informed by serum 25(OH)D measurements taken at various stages throughout the training cycle [71]. To support optimal musculoskeletal health, the recommended minimum serum 25(OH)D concentration is 30 ng/mL (75 nmol/L), while the ideal target range is 40–50 ng/mL (100–125 nmol/L). Maintaining vitamin D within these parameters is associated with enhanced muscle function, more efficient recovery, and a reduced risk of musculoskeletal injuries [72]. Annual monitoring of serum 25(OH)D is recommended, especially for athletes considered at high risk of deficiency—such as those training primarily indoors, individuals with darker skin pigmentation, those residing at higher latitudes, or athletes with limited sun exposure. Late winter and early spring are the optimal times for such assessments, as serum vitamin D concentrations are typically at their lowest during these periods [73]. When deficiency is detected, tailored supplementation should be implemented. For mild deficiency (20–30 ng/mL), a daily dose of 2, 000–3, 000 IU is recommended. In cases of moderate to severe deficiency (<20

ng/mL), daily supplementation may need to be increased to 4, 000–5, 000 IU. After correcting a substantial deficit, high-dose regimens—such as 50, 000 IU once weekly for eight weeks—can be used, followed by maintenance therapy. Serum 25(OH)D should be reassessed after 8–12 weeks to determine the effectiveness of the intervention and guide further treatment adjustments. Although adverse effects from vitamin D supplementation are rare, excessive intake can result in complications, most commonly hypercalcemia. Prolonged supplementation with doses exceeding 10, 000 IU per day should be avoided unless there is a clear medical indication [74]. In athletes whose serum 25(OH)D levels are already sufficient or elevated, additional supplementation confers no added benefit and may be harmful. If levels rise above 50–60 ng/mL, dose reduction is advised to mitigate the risk of hypercalcemia and other adverse outcomes. In summary, vitamin D supplementation in athletes should be personalized and based on monitoring serum 25(OH)D levels. Properly managed intervention preserves bone and muscle health, reduces injury risk, supports recovery, and may beneficially affect overall performance. Key elements include choosing the appropriate vitamin form, correct dosing, avoiding excess, and regular biochemical monitoring.

6. Conclusions and Summary

Vitamin D's pleiotropic actions on the musculoskeletal system and beyond underscore its centrality to athlete health. On the molecular level, the bioactive form, 1, 25-dihydroxyvitamin D (calcitriol), binds to nuclear VDRs to upregulate genes involved in calcium and phosphorus transport, protein synthesis, and muscle fiber differentiation, while its non-genomic interactions at the membrane facilitate rapid calcium flux essential for actin–myosin cross-bridge formation. Deficiency disrupts this tightly regulated axis: inadequate 25(OH)D impairs type II fiber integrity, diminishes mitochondrial function, skews inflammatory balance, and compromises bone remodeling, collectively manifesting as proximal muscle weakness, delayed tissue repair, and markedly heightened stress-fracture risk.

Clinical evidence affirms that correcting vitamin D insufficiency (< 30 ng/mL) through tailored cholecalciferol regimens (e.g., 2, 000–6, 000 IU daily or 50, 000 IU weekly for repletion) not only restores serum levels to the mid-sufficient range (40–50 ng/mL) but also translates into measurable reductions in injury rates—stress fractures decline by over 75% in supplemented cohorts—and improvements in functional outcomes, particularly when combined with resistance training. Conversely, supra-physiological dosing yields diminishing returns and poses a low but real risk of hypercalcemia and hyperphosphatemia. Thus, a precision-based framework, incorporating individual factors such as baseline 25(OH)D, BMI, skin pigmentation, latitude, seasonal UVB exposure, and training load, is paramount to maximize benefits while mitigating risks.

7. Limitations

This review is subject to several important limitations that temper the strength of its conclusions. First, the studies we examined vary widely in design, from small, short-term randomized trials to retrospective cohort analyses and narrative reviews, and they encompass athletes of differing levels, ages, sexes, ethnicities and sports. These differences, together with inconsistent thresholds for defining vitamin D sufficiency (ranging from 20 to 40 ng/mL), make it difficult to generalize findings across athletic populations. Intervention protocols also differ markedly—daily versus bolus dosing, vitamin D_2 versus D_3, and supplementation periods from a few weeks to a year—while outcome measures range from maximal strength tests to jump height and disparate injury definitions. Such variability hinders identification of optimal dosing regimens and complicates cross-study synthesis. Moreover, many of the associations between low 25(OH)D levels and injury risk derive from observational or cross-sectional studies, which cannot establish causality and are prone to confounding by factors such as training load, diet, sun exposure and genetic differences in vitamin D metabolism. Even the randomized trials tend to have relatively short follow-up (typically under three months), limiting insight into the long-term effects of sustained vitamin D repletion on muscle adaptation, performance and injury prevention. Seasonality, latitude, clothing and indoor versus outdoor training further confound endogenous vitamin D synthesis, yet are inconsistently controlled for across studies. Data on the underlying biological pathways—particularly human in vivo investigations into VDR signaling—remain scarce, and genetic polymorphism studies have generally been underpowered to explain interindividual variability in response to supplementation. Finally, by focusing on peer-reviewed English-language publications, this review may be affected by publication and language bias, potentially overrepresenting positive findings. Together, these limitations underscore the need for large, well-controlled, long-term trials with standardized definitions and outcome measures to clarify vitamin D's true impact on athletic health and performance.

8. Future Directions for Research

Despite these advances, significant gaps persist. Most supplementation trials are short-term (8–12 weeks) and heterogeneous in population, dosing protocols, and outcome measures, impeding meta-analytic clarity on ergogenic effects beyond musculoskeletal health. Well-powered, long-duration RCTs with standardized definitions of deficiency and sufficiency—and uniform performance metrics—are needed to determine whether maintaining higher 25(OH)D thresholds confers incremental gains in power, endurance, or neuromuscular coordination among athletes with baseline adequacy. Moreover, interindividual variability driven by VDR gene polymorphisms and differential expression of vitamin D–metabolizing enzymes warrants exploration through combined genomic and proteomic approaches in human in vivo models. Finally, the interplay between vitamin D and co-nutrients such as magnesium and vitamin K, as well as its potential immunomodulatory role in reducing exercise-induced inflammation and infection susceptibility, represents a promising frontier to optimize holistic athlete performance and recovery.

9.Discussion:

Aggregate findings from studies on vitamin D's impact in athletes indicate beneficial effects on athletic performance, physical fitness, and a reduction in injury and fracture risk. Available scientific evidence demonstrates that vitamin D supplementation can contribute to increases in muscle strength and mass. Conversely, deficiency correlates significantly with declines in athletic performance, stemming in part from bone demineralization and reduced muscle force. Additionally, inadequate vitamin D status is associated with impaired pulmonary function, which directly translates into diminished exercise capacity and work efficiency in athletes. [59][60] Experts concur that optimal serum vitamin D concentrations for athletes should range between 30 and 60 ng/mL. This interval is considered both safe and conducive to proper immune, muscular, and musculoskeletal system function. Consequently, regular monitoring of vitamin D levels in athletes—particularly in those at high risk for deficiency—is recommended. [61][62] It remains indisputable that the advantages of supplementation—such as improved exercise capacity and reduced muscle soreness—primarily pertain to individuals with vitamin D deficiency. In athletes with sufficient vitamin D status, additional supplementation does not demonstrate a significant effect on enhancing performance. The literature suggests that 25(OH)D concentrations of 40–50 ng/mL represent a threshold beyond which no further muscular benefits are observed. Attention should also be paid to the risk of toxicity from excessive supplementation, which may occur at levels exceeding 150 ng/mL and pose potential health hazards. [63][64] Numerous studies report a potentially favorable impact of vitamin D supplementation on athletic performance. However, interpreting these results requires caution due to considerable methodological limitations. Many trials feature relatively short supplementation periods (typically 8–12 weeks), which may be insufficient to reveal long-term physiological effects. [65] Moreover, study populations are often heterogeneous—encompassing both recreationally active individuals and elite athletes—without adequately accounting for differences in age, training status, or baseline serum 25(OH)D. Such variability greatly complicates the formulation of clear, generalizable conclusions regarding the efficacy of vitamin D supplementation for improving physical performance.

Disclosure

Author's Contributions

Conceptualization, Katarzyna Rumianek-Fidziukiewicz, and Marta Ewelina Lis;
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Software, check: not applicable;
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