# The Effects of Vitamin D Deficiency in Athletes



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Vitamin D acts to maintain calcium and phosphate homeostasis within the body. It is now estimated that 1 billion people world-wide are vitamin D deficient. This problem is particularly important to athletes of all ages, as vitamin D plays a significant role in bone health, immune function, and physical performance. In the deficient state, the athlete may be at an increased risk for potential problems such as stress fractures, respiratory infections, and muscle injuries. The purpose of this article is to examine vitamin D deficiency and review its relationship to the athlete.

Keywords: vitamin D deficiency; athletic performance; nutritional physiology

Vitamin D deficiency is a common but underrecognized problem within the global population. Although there is no consensus on optimal serum levels of 25-hydroxyvitamin D<sub>3</sub> (25(OH)D3), vitamin D deficiency is defined by most experts as a total 25-hydroxyvitamin D level of less than 20 ng/mL. Vitamin D insufficiency is defined as a level of 20 to 31 ng/mL, and a level of 32 ng/mL or greater is indicative of sufficient levels. Vitamin D intoxication is observed when serum levels of 25-hydroxyvitamin D are greater than 150 ng/mL. <sup>10,11</sup> The effects of vitamin D deficiency and insufficiency involve skeletal health regarding bone and muscle function; deficiency can also relate to chronic illnesses such as cancer, infectious diseases, autoimmune diseases, and cardiovascular diseases. <sup>11</sup>

It is estimated that 1 billion people, including the elderly, young adults, and children, are vitamin D deficient or insufficient. In a recent study performed at an urban hospital in Boston, it was estimated that 42% of the adolescent patients examined had vitamin D deficiency. In additional study in Israel found that 48% of the young athletes and dancers examined were vitamin D insufficient while another 25% were vitamin D deficient. A recent retrospective study found that 43% of preoperative orthopaedic surgery patients were vitamin D insufficient, and of these 40% had deficient levels. The specialty-specific data revealed that 52.3% of the sports service patients, which included patients undergoing anterior cruciate ligament reconstruction and/or meniscal repair or

meniscectomy, were vitamin D insufficient, and one-third of these patients had deficient levels.

This high prevalence of vitamin D deficiency also extends to the elite athlete population. A recent study from the Hospital for Special Surgery in New York examined the vitamin D levels from 89 players on a single National Football League team and found that 30% of the players were deficient while 51% had insufficient levels (Shindle, unpublished data, 2011). The players with muscle injuries were found to have significantly lower vitamin D levels than were uninjured players. A similar study examined 18 elite Australian gymnasts and found that 15 had insufficient levels, and 6 of these athletes had deficient levels. 14

The causes of vitamin D deficiency can be multifactorial and can often relate to reduced skin synthesis and/or absorption of dietary vitamin D. There are several well-known acquired and heritable disorders of vitamin D metabolism that can also lead to deficiency. The main cause of vitamin D deficiency in the athletic population is the direct result of decreased ultraviolet B (UVB) radiation absorption into the skin; therefore there is a concomitant decrease in the cutaneous synthesis of vitamin D. This especially affects indoor athletes and athletes who live and train in northern latitudes; the lack of UVB absorption has a similar effect on dark-skinned athletes with increased skin pigmentation.

This article provides an overview of the physiology of vitamin D and its role in musculoskeletal health. In particular, we will focus on the effects of vitamin D deficiency as it relates to the athlete in terms of bone and muscle function as well as the athlete's physical performance.

### VITAMIN D PHYSIOLOGY

The main function of vitamin D is to maintain calcium and phosphate homeostasis within the body by targeting organs such as the intestine, kidney, parathyroid glands, bone, and skeletal muscle. Sources of dietary vitamin D

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include foods such as fatty fish, eggs, and dairy products. The dietary intake and intestinal absorption of vitamin D is a minor part of the total vitamin D requirement.

The major source of vitamin D is provided through the interaction of the skin with UVB light. In the skin, 7-dehydrocholesterol is converted into previtamin  $D_3$  by UVB radiation (wavelength 290-315 nm). The previtamin  $D_3$  is slowly isomerized into vitamin  $D_3$  (cholecalciferol) and then bound by vitamin D binding protein, and transferred to the liver with its metabolites.

Once in the liver, the cholecalciferol is hydroxylated to 25-hydroxyvitamin D (25(OH)D3; calcifediol), which is the major circulating metabolite in the body. The 25(OH)D3 is then further metabolized in the kidney by 25-hydroxyvitamin D-1\(\alpha\)- hydroxylase (CYP27B1) to its biologically active form, 1,25-dihydroxyvitamin D (1,25(OH)D3; calcitriol). \(^{11}\) This hydroxylation is controlled by calcium, phosphate, and parathyroid hormone levels. \(^{11}\) Vitamin D production is influenced by age, season, geographic location, and skin pigmentation. \(^{5}\) These are important considerations for the athlete; the training environment may affect vitamin D production, specifically indoor training, winter sports, and people who live in northern latitudes. \(^{5}\)

## VITAMIN D AND MUSCLE FUNCTION

Much of the initial work examining the relationship between vitamin D and muscle function was done by Birge and Haddad.2 They found that calcifediol regulates the intracellular accumulation of phosphate within the muscle cells, and this helps to maintain muscle function and metabolism. On a molecular level, vitamin D has its effect on muscle via several known pathways. The active metabolite of vitamin D, 1,25(OH)D3, binds to the muscle nuclear hormone receptor, vitamin D receptor (VDR). This pathway promotes gene transcription, leading to increased cell protein synthesis and growth.  $^{5}\,\mathrm{A\,VDR}$  knock out mouse model demonstrated a phenotype with abnormal muscle fiber development and maturation. As a result, the rodents were found to have poor motor and balance function, as indicated by poor swimming ability. 15 Furthermore, work by Bischoff-Ferrari and colleagues3 have shown that vitamin D receptor expression within muscle tissue decreases with age, which may provide a partial explanation as to why athletic performance declines with age. All of these findings suggest that vitamin D is directly related to muscle strength, mass, and function. Such factors are crucial to the performance of an athlete.<sup>5,6</sup>

Vitamin D also exerts its effects through various cellular signaling cascades, one of which is the mitogen-activated protein kinase (MAPK) signaling pathway. Once activated, this pathway initiates myogenesis, cell proliferation, and differentiation in muscle tissue. Studies in vitamin D-deficient rats have shown that supplementation increases protein synthesis and muscle mass; there was also a decrease in the rate of exercise-induced muscle cell apoptosis.<sup>5,6</sup>

Muscle biopsy specimens from adults with low vitamin D levels have revealed atrophic changes predominantly of the type II muscle fibers. There was enlargement of the interfibrillar spaces, fatty infiltration, fibrosis, and

increased glycogen granules.<sup>1,6</sup> These changes appear to be reversible; several studies have shown an increase in type II fiber composition after supplementation with vitamin D and calcium in older adults.<sup>1</sup>

## VITAMIN D AND BONE

The role of vitamin D in skeletal bone mineralization and calcium homeostasis is well known. In its active form, 1,25(OH)D3 is an important factor in activating intestinal calcium absorption and raising serum calcium concentrations. Many studies have identified a direct relationship between serum vitamin D levels and bone mineral density in adults of all races.

When serum vitamin D levels fall below 30 ng/mL, parathyroid hormone levels are increased, which triggers an increase in osteoclastic activity in bone. <sup>11,16</sup> Additionally, 1,25(OH)D3 directly stimulates osteoblasts to produce receptor activator nuclear factor-kB (RANKL), which in turn enhances osteoclastogenesis and mobilization of calcium from the bone. <sup>11</sup> This can potentially affect athletes who are at an increased risk for stress fractures based on activity or sport levels. Work by Lappe and colleagues <sup>13</sup> has shown that calcium and vitamin D supplementation significantly decreased the incidence of stress fractures among female military recruits.

# ATHLETIC PERFORMANCE AND VITAMIN D

A link between vitamin D and athletic performance has been known for many years. Seasonal variability of athletic performance within the northern hemisphere was reported as early as the mid 1950s, with peak performance occurring during the late summer months when sun exposure was maximal and then declining to a low point in the winter. These findings have been supported by further studies that have shown consistently improved athletic performance in summer, with a gradual decline and a nadir in winter months despite consistent training throughout the year. There appears to be a direct correlation between the seasonal variation of athletic performance and the seasonal fluctuations in serum vitamin D levels. 1,12

Furthermore, UVB radiation exposure has long been thought to improve athletic performance. Russian researchers as far back as 1938 found improvements in 100-m dash times in subjects who had UV radiation as compared with nonirradiated controls.<sup>5</sup> Another study from Germany in 1952 showed UV irradiation improved cardiovascular fitness on a bicycle ergometer in schoolchildren. When the control, nonirradiated children were given vitamin D supplementation, their performance approached that of the irradiated group.<sup>5</sup>

More recent research by Ward and colleagues<sup>19</sup> found a direct relationship between serum 25(OH)D3 levels and muscle power, force, velocity, and jump height as determined by jump mechanography in girls aged 12 to 14 years. A similar study by Foo et al<sup>8</sup> noted an increased hand grip strength in 301 teenage girls with normal vitamin D levels compared with those who were deficient.

# TREATMENT OF VITAMIN D DEFICIENCY

The treatment of an athlete should begin with an appropriate assessment of the vitamin D level. The best measure is the serum concentration of the inactive 25(OH)D3 level; this represents the vitamin D level from dietary intake, sunlight exposure, and adipose stores. Recent studies have recommended a normal range between 30 and 50 ng/mL; serum concentrations below this value stimulate the production of parathyroid hormone. Additionally, optimal calcium absorption requires at least 30 ng/mL of vitamin D in healthy adults. 16,17

The latest United States recommended dietary allowance (RDA) for vitamin D is 600 IU per day for children (over the age of 1 year), adolescents, and adults up to 70 years of age. This represents the recommended dietary intake to achieve serum concentration of 25(OH)D3 between 30 and 50 ng/mL. However, dietary intake may vary between individuals based on diet and intestinal absorption, and serum concentrations can be significantly affected by sunlight exposure. As the potential for vitamin D toxicity is extremely rare, supplementation can be prescribed aggressively, especially in people with deficient levels.

Vitamin D2 or D3 can be used for oral supplementation (D2 being approximately 30% as effective as D3 in achieving desired serum levels), and both can often be found in over-the-counter formulations as high as 10,000-IU tablets. For the deficient patient, treatment regimens include 50,000-IU capsules of vitamin D2 (prescription strength) every week for 8 weeks, followed by 50,000 IU of vitamin D2 every 2 to 4 weeks for maintenance based on a repeat serum concentration at 8 weeks. Alternative regimens include daily intake of 1000 IU of vitamin D3 or 3000 IU of vitamin D2 for 8 weeks, followed by a repeat serum 25(OH)D3 level and adjustment of dosing accordingly.11

# CONCLUSION

Numerous studies have identified vitamin D deficiency in the adolescent and adult athlete populations. 1,5,6,11,16,17 This is especially a problem for athletes who have limited sun exposure because of geography and limited seasonal UVB exposure. It can also be a problem for athletes who use excessive sunscreen and patients with dark skin pigmentation. Current evidence suggests that the treatment of athletes who are vitamin D deficient may help to improve their athletic performance. The magnitude of this improvement has not been determined, and the specific benefits of supplementation for athletes who have insufficient levels have also not been determined. There is currently no level 1, randomized controlled trial that examines the ideal vitamin D levels for peak athletic performance. Additionally, there are no data to suggest that vitamin D supplementation (supraphysiologic) is a source of performance enhancement for sports activities. In fact, a recent report from the Institute of Medicine suggests daily intake of higher levels of vitamin D may be linked to other health problems such as kidney and tissue

damage. 18 Further study is clearly necessary to identify the role of vitamin D in athletic performance.

Despite the lack of level 1 evidence, the sports medicine physician should be aware of the effects of vitamin D deficiency on athletic performance and overall musculoskeletal health, including such potential problems as stress fractures and muscular injuries. Based on the current evidence, we recommend assessing total serum 25(OH)D3 levels in high-risk athletes and treating those who show deficient or insufficient levels.

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