Vitamin D and Athletes

D. Enette Larson-Meyer[1] and Kentz S. Willis[2]

[1]Departments of Family and Consumer Sciences (Human Nutrition), University of Wyoming, Laramie, WY;
[2]Cooperative Extension Service, Sheridan, WY

LARSON-MEYER, D.E. and K.S. WILLIS. Vitamin D and athletes. *Curr. Sports Med. Rep.*, Vol. 9, No. 4, pp. 220–226, 2010. *While it is well recognized that vitamin D is necessary for optimal bone health, emerging evidence is finding that adequate vitamin D intake reduces risk for conditions such as stress fracture, total body inflammation, infectious illness, and impaired muscle function. Studies in athletes have found that vitamin D status is variable and is dependent on outdoor training time (during peak sunlight), skin color, and geographic location. Although research has found that athletes generally do not meet the U.S. dietary reference intake for vitamin D, inadequate endogenous synthesis is the most probable reason for insufficient/deficient status. Given the recent findings, it is imperative that sports dietitians and physicians routinely assess vitamin D status and make recommendations to help athletes achieve a serum 25(OH)D concentration of ≥32 and preferably ≥40 ng·mL^{-1}. Further research is needed to determine the effect of vitamin D status on injury, training, and performance in athletes.*

INTRODUCTION

The previous decade has been marked with the discovery of many new physiological functions of vitamin D as well as the documentation of epidemic vitamin D deficiency and insufficiency among adults and children worldwide (11,24,26,30,37,42,50,55). While less is known about vitamin D status in athletes, emerging evidence is suggesting that vitamin D plays an important role in an athlete's health, training and performance. Specifically, it is now recognized that vitamin D is imperative for bone health, immune function, and inflammatory modulation (11,13,55), and it may even be necessary for optimal muscle function (7,19,29,50) and performance (10). In addition, vitamin D deficiency is linked to an increased risk for chronic and autoimmune disorders — including hypertension, cardiovascular disease, rheumatoid arthritis, osteoarthritis, inflammatory bowel disease, depression, and certain types of cancer (24,26,55) — many of which occur even in the trained athlete. This review will provide an update on the importance of this vitamin in athletes and provide tips for assessing and treating vitamin D deficiency and insufficiency.

Address for correspondence: D. Enette Larson-Meyer, Ph.D., R.D., FACSM, Department 3354, 1000 E University Avenue, University of Wyoming, Laramie, WY 82071 (E-mail: enette@uwyo.edu).

VITAMIN D SYNTHESIS AND SOURCES

Vitamin D is a unique nutrient in that physiological sufficiency can be met entirely through endogenous synthesis (23,26,55). When the skin is exposed to ultraviolet-B radiation (UVB, wavelength of 290–315 nm), 7-dehydrocholesterol — present in the plasma membrane of epidermal and dermal cells — is converted to previtamin D_3 (precholecalciferol). Previtamin D_3 then undergoes thermal isomerization to vitamin D_3 (cholecalciferol) over a period of 2 to 3 d (23). Vitamin D_3 then migrates into the dermal capillary bed and into circulation with the assistance of vitamin D binding protein (VDBP) and is subsequently hydroxylated in the liver by enzymes of the cytochrome P-450 system to 25(OH)D (24,26). Further hydroxylation in kidney tubules to the hormonally-active form, 1,25(OH)$_2$D, is driven by parathyroid hormone (PTH) when serum calcium and phosphate concentrations fall below the physiological range (24,26,55). In addition, many extra-renal cells (and tissues), including macrophages, brain, colon, breast, and others, have the enzymatic machinery (1-α-hydroxylase) to produce 1,25(OH)$_2$D locally (11,26,51). Cutaneous production of vitamin D, however, is variable and dependent on environmental and individual factors, including time of day, season, cloud cover, smog, latitude, skin pigmentation, age, and sunscreen use. In addition, vitamin D cannot be synthesized in winter months at latitudes greater than 35° to 37° degrees (11,26) because the zenith angle of the sun prevents sufficient UVB photons from reaching the earth's surface (26).

Vitamin D also can be obtained in the diet from limited natural and fortified sources including fatty fish, egg yolks, sun-dried mushrooms, and fortified milk, margarine, and

cereals (24). Dietary vitamin D includes both D_3 (animal form, cholecalciferol) and D_2 (ergocalciferol), which is derived from UVB exposure of precursor fungi and yeast ergosterols. Both D_3 and D_2 are absorbed into intestinal mucosal cells from micelles in association with lipid (and the aid of bile salts) where they are incorporated into chylomicrons and enter circulation via the lymphatic system (4,24). Both are readily absorbed (~50% bioavailable) except in individuals with malabsorption syndromes (4). Following absorption, dietary vitamin D is transported to the liver and metabolized similarly to endogenous vitamin D_3.

INDICES OF VITAMIN D STATUS

There is general agreement that 25(OH)D concentration is the best indicator of vitamin D status (11,24,25,27). Circulating concentration of $1,25(OH)_2D$ is dependent on PTH (and sensitive to serum calcium and phosphorus concentrations) and is not reflective of vitamin stores. In fact, serum $1,25(OH)_2D$ concentration may be above or within the normal clinical range when vitamin D stores are deficient (11,26,55). In addition, 25(OH)D has a longer half-life (2–3 wk) (55) than $1,25(OH)_2D$ (<4 h) (25) and is better reflective of intestinal calcium absorption (22,55). Finally, extra-renal tissues are dependent on adequate serum 25(OH)D (11,51) to synthesize (adequately) $1,25(OH)_2D$ for local cellular use (55).

Definitive thresholds for vitamin D status, however, are not clear. Most experts currently define vitamin D deficiency as a

25(OH)D concentration <20 ng·mL^{-1} (24–26), insufficiency as a concentration <30 (24–26) to <32 ng·mL^{-1} (27), and toxicity as a concentration >150 ng·mL^{-1} when coupled with an elevated serum calcium concentration (11,24,27). Cutoff for deficiency is the approximate concentration whereby PTH rises abruptly (25,27,55), whereas the cutoff for insufficiency is the approximate concentration in which calcium absorption is maximized (25). Optimal concentration thresholds are not yet established but are proposed to fall in the range of 40 to 70 ng·mL^{-1} (9), which is believed to be the concentration in which the human genome evolved (11,27).
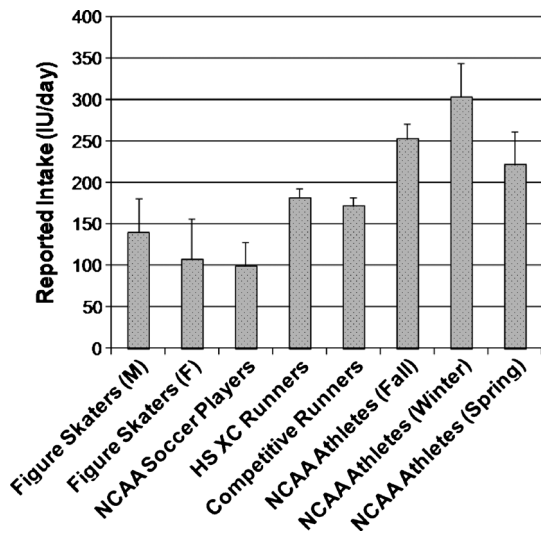
VITAMIN D INTAKE AND STATUS OF ATHLETES

Although numerous studies have documented a high prevalence of vitamin D insufficiency and deficiency in the general population worldwide, only a handful of studies have focused on athletes (Table 1). Overall, these studies have found that 25(OH)D concentration varies by population and generally is higher in summer compared with winter (18,34). Interestingly, the lowest status was found in athletes training in both Finland (60.4° N) (34) and the Middle East (24.4° N) (20). The best status was documented in college athletes training in a high-altitude region of the United States (2195 m; 41.3° N) (18). Even though dietary assessment studies have found that athletes generally do not meet the U.S. daily adequate intake (AI) for vitamin D of 200 IU (5,14,18,34,54) (Fig.), the most probable reason for poor vitamin D status is inadequate endogenous synthesis from

TABLE 1. Vitamin D status in athletes living in various geographic locations.

Reference	Type of Athlete	Location	Season	25(OH)D (ng·mL^{-1})	Vitamin D Status
Bannert et al. (3)	85 male and female competitive gymnasts (8–27 yr)	Magdeburg, East Germany (52.5° N)	NR	~20–24^e (0–80)	37% <10 ng·mL^{-1}
Hamilton et al. (20)	93 Middle Eastern sportsmen (21 yr average)	Doha, Qatar (25.4° N)	NR	11.0 (est) (<7–28)	91% <20 ng·mL^{-1} 58% <10 ng·mL^{-1}
Halliday et al. (18)	18 male and 23 female college athletes (18–24 yr)	Laramie, WY (41.3° N)	Fall	49.0 ± 16.6	12.2% <32 ng·mL^{-1} 2.4% <20 ng·mL^{-1}
			Spring	30.5 ± 9.4	63.6 <32 ng·mL^{-1} 3.0% >20 ng·mL^{-1}
			Winter	41.9 ± 14.6	20.0% <32 ng·mL^{-1} 4.0% <20 ng·mL^{-1}
Lehtonen-Veromaa et al. (34)	66 female gymnasts, 65 runners, 60 controls (9–15 yr)	Turku, Finland (60.4° N)	Winter	13.6 ± 5.6	68% <15 ng·mL^{-1} 13% <4 ng·mL^{-1}
			Summer	25.2 ± 6.0	11.6% <15 ng·mL^{-1}
Lovell (36)	18 female gymnasts (10–17 yr)	Canberra, Australia (35.3° S)	Fall (early May)	22.4 (3.6–33.7)	83% <30 ng·mL^{-1} 33% <20 ng·mL^{-1}
Maimoun et al. (38)	7 male competitive road cyclists (20–39 yr)	Montpellier, France (43.6° N)	Competitive season	32.5 ± 6.4	NR
Willis (52)	9 male and 10 female endurance runners (19–45 yr)	Baton Rouge, LA (30.5° N)	Random sample	38.7 ± 17.2 (15.6–81.0)	42% <32 ng·mL^{-1} 0.5% <20 ng·mL^{-1}

Data are presented as means ± SD. The range, if reported, is given in parenthesis. NR, not reported.

Figure. Vitamin D intake in athletes from a variety of sports (5,14,18, 34,54). M, male; F, female; NCAA, National Collegiate Athletic Association; XC, cross-country.

insufficient UVB exposure. Factors that may impair synthesis in athletes include skin pigmentation (20), early- or late-day training (20), indoor training (18), geographic location (Table 2) (34), and sunscreen use (39).

In addition to endogenous synthesis, intake of vitamin D-containing supplements, including a multivitamin, may result in higher but not necessarily optimal serum 25(OH)D concentration (34). In our study in college athletes, for example, we found that approximately 33% of athletes take a multivitamin at least $4 \cdot d \cdot wk^{-1}$ and that vitamin D status correlates with multivitamin intake in the winter (18). In contrast, 75% never take a larger supplemental dose (>1000 IU). Although most multivitamins contain 400 IU, some now contain higher vitamin D levels and may be an effective way to improve status in athletes.

BODY FAT AND INDICES OF VITAMIN D STATUS

Recent research suggests that excess adiposity may increase risk for low vitamin D status. In normal-weight individuals, a certain fraction of both D_3 and D_2 are stored in subcutaneous fat (53) and released during the winter (or other periods of reduced UVB exposure) (24). In individuals with excess body fat, however, more vitamin D appears to be sequestered deep in subcutaneous fat, decreasing its availability for conversion into 25(OH)D (53). In nonathletes, studies have found that 25(OH)D concentration is low in obese individuals (30,46) and inversely correlated with body fat percentage (46). Preliminary results from our studies show that 25(OH)D concentration tends to correlate negatively with body fat percentage within the body fat range of our college athletes (8%–29%), but this relation was not apparent in the winter when 25(OH)D concentration dropped precipitously (18). Studies in larger populations of athletes are necessary to confirm these findings and to address whether athletes with extremely low body fat may be at risk for deficiency in winter months because of decreased storage capacity.

IMPORTANCE OF VITAMIN D

Recent research has found that vitamin D functions as a modulator of up to 1000 genes involved in cellular growth, immune function and protein synthesis (10). In this role, cellular-derived $1,25(OH)_2D$ interacts with its nuclear vitamin D receptor (VDR), which is present in most tissues and cells — including intestine, bone, brain, heart, immune cells (24,26), and skeletal muscle (7,10,19,26). The vitamin D-VDR complex then binds with the retinoic acid x-receptor, which is recognized by specific vitamin D-response elements on the gene sequence, to regulate expression of specific genes (10,24,26). As reviewed earlier, higher serum 25(OH)D concentration increases the substrate for intracellular $1-\alpha$-hydroxylase, thereby promoting the adequate intracellular $1,25(OH)_2D$ concentration necessary for appropriate modulation of gene expression.

Bone Health

Vitamin D influences bone health by upregulating expression of genes that enhance intestinal calcium absorption, renal tubular reabsorption (in association with elevated PTH), and osteoclastic activity (24,26,55). A powerful example is found with fractional calcium absorption, which is approximately 10%–15% in the vitamin D-deficient state and greater than 30% when serum 25(OH)D concentration is at least 30 $ng \cdot mL^{-1}$. Serum 25(OH) D concentration also is found to correlate with bone mineral density and/or bone mineral content in the hip and lumbar spine in both younger and older women. Several recent studies have provided evidence that sufficient vitamin D status is important for bone health and prevention of bone injury in active individuals. One study in young Finnish military recruits found that stress fracture risk was significantly increased when serum 25(OH)D concentration fell below 30 $ng \cdot mL^{-1}$ (44). Another study in female naval recruits found that stress fracture incidence was significantly reduced (vs placebo) following an 8-wk supplementation with 800 IU of vitamin D plus 2000 mg calcium (33).

Skeletal Pain, Strength, and Performance

Musculoskeletal pain and weakness are recognized (but often forgotten) symptoms of vitamin D deficiency (11,24,26,55). A relatively recent study conducted at an inner-city clinic in Minnesota found that 93% of individuals with persistent nonspecific musculoskeletal pain had a 25(OH)D concentration <20 $ng \cdot mL^{-1}$ and 28% had a concentration <8 $ng \cdot mL^{-1}$ (42). Another study in South Asian women found that chronic pain was 3.5 times more common in women with a 25(OH)D concentration <10 $ng \cdot mL^{-1}$ (37). Of particular clinical interest, two recent studies found that vitamin D_3 supplementation improves muscle pain (1,15). In the largest of these open studies, 3 months of treatment with 5000–10,000 $IU \cdot d^{-1}$ resulted in clinically improved reductions in back pain in all patients with initially low vitamin D stores (1). Randomly assigned clinical trials or trials in chronically injured athletes, however, have yet to be conducted.

TABLE 2. Clinical assessment of vitamin D status.

Anthropometrics and Biological Factors

- Age
- Body weight and body mass index
- Body fat percentage
- Weight history

Biochemical (Laboratory) Data

- 25(OH)D concentration
- Other, particularly if 25(OH)D is low: parathyroid hormone, alkaline phosphatase, serum calcium, serum phosphorus, thyroid stimulating hormone

Clinical

History

- Stress and other bone fractures
- Bone pain
- Muscle pain, weakness, "heaviness in legs"
- Chronic injury
- Frequent illness
- Photosensitivity
- Skin cancer/melanoma
- Family history of skin cancer

Medications

- Anticonvulsants, corticosteroids, cimetidine, theophylline, orlistat, antituberculosis agents (may decrease vitamin D status)
- Thiazide diuretics, atorvastatin, other statins (may increase circulating vitamin D)
- Sulfonamides, phenothiazines, tetracyclines, psoralens increase photosensitivity; may signal sun avoidance

Physical exam

- Idiopathic musculoskeletal pain
- Muscle weakness (proximal limbs)
- Undue pain on sternal or tibial pressure
- Bowel function (steatorrhea)
- Skin pigmentation (or type)/hair color
- Contraindications to sunlight (albinism, porphyrias, xeroderma pigmentosum)

Dietary

- Vitamin D
- Vitamin D-containing supplements (including multivitamin)
- Calcium
- Phosphate (nutritional supplements and soda consumption; may elevate PTH) (55)
- Magnesium (commonly low in Western diet and important for muscle and bone health)
- Other nutrients: vitamins A, C, and K, omega-3/omega-6 fatty acids

Lifestyle and Environmental

- Training regimen
- Training environment in relation to sunlight exposure

(continued)

TABLE 2. (Continued).

- Training latitude, altitude, climate
- Sunscreen use (SPF of 15 lowers synthesis capacity by >98%) (26)
- Uniform or athletic clothing worn and SPF of clothing
- Leisure sun exposure (frequency, duration, and time of day)
- Tanning bed use (must emit UVB)
- Bathing habits after sun exposure (may rinse off skin cells containing newly synthesized vitamin D) (8)
- Belief system (concerning sunlight exposure)

PTH, parathyroid hormone; SPF, sun protection factor; UVB, ultraviolet-B.

Impaired muscle strength and weakness associated with low vitamin D status also is well documented, although mostly in older populations (7,29). A very recent study, however, found that vitamin D status was correlated with jump height, velocity, and power in young (12–14 yr) postmenarchal British girls (50). Because vitamin D status was suboptimal in a high percentage of the subjects — average 25(OH)D concentration was 11.6 ng·mL^{-1} — these results indicate that muscle performance is impaired by suboptimal vitamin D status (and not that performance is enhanced by supraoptimal status). The physiologic explanation for vitamin D's beneficial effect on muscle strength most likely is explained by its autocrine function, which promotes gene expression and muscle cell growth (19). In agreement, animal studies have found that vitamin D deficiency induces atrophy of fast-twitch muscle fibers, impairs sarcoplasmic calcium uptake, and prolongs time to peak contractile tension and relaxation (19). A recent study of hospitalized patients also found that vitamin D deficiency delayed functional rehabilitation (31), which may be applicable to injury rehabilitation in athletes. Further, studies have found that treatment with at least 1000 IU·d^{-1} of vitamin D normalizes muscle strength and improves muscle function in vitamin D-deficient patients in 1 to 2 months (55). Although controlled studies have not yet been conducted in athletes, evidence from Russian and German literature at the turn of the 20th century suggests that vitamin D has the potential to impact athletic performance (10). See reviews of Hamilton (19) and Cannell and colleagues (10) for additional information.

Inflammation and Immunity

Vitamin D recently has been recognized as an important regulator of inflammation and immunity. Specifically, vitamin D upregulates gene expression of broad spectrum antimicrobial peptides (AMP) — which are important regulators in innate immunity — and also downregulates expression of inflammatory cytokines such as tumor neurosis factor-alpha (TNF-alpha) and interleukin-6 (IL-6) (35,49). AMP is secreted by many components of the innate immune system including macrophages, monocytes, natural killer cells, and epithelial cells in the respiratory tract (17) and exerts its effect by compromising the integrity of the cell membrane of invading pathogens. The release of AMP — such as cathelicidin — is triggered when invading pathogens are recognized by toll-like receptors (TLR). Liu *et al.* (35) recently demonstrated

that TLR stimulation triggers conversion of 25(OH)D to 1,25(OH)$_2$D, and induces expression of the VDR, which in turn promotes the synthesis of cathelicidin. Vitamin D also is found to have an immunomodulatory effect on T and B lymphocytes (26,48,55) in the adaptive immune system.

While vitamin D's pivotal role in innate immunity has been recognized for only a few years, a recent review presented intriguing evidence that influenza epidemics and other winter-time infections may be related to seasonal deficiencies of AMP secondary to deficiencies of vitamin D (13). In support of this hypothesis, a post-hoc analysis of a randomized control trial found that vitamin D supplementation (800 IU·d^{-1} for 2 yr followed by 2000 IU·d^{-1} for an additional year) was associated with dramatically decreased reported incidence of colds and flu (2). In our study of college athletes, poor vitamin D status was associated with increased frequency of documented illness — including common cold, influenza, and gastroenteritis (18). The breakpoint for contracture of a single illness appeared to occur at approximately 38–40 ng·mL^{-1}, such that all athletes who maintained stores lower than the breakpoint in the winter and spring contracted one or more illnesses. Studies evaluating the effect of vitamin D deficiency on susceptibility to upper respiratory infections, particularly during intense training and competition, would be of interest.

Several proinflammatory cytokines, including IL-6 and TNF-alpha are elevated after exercise, and elevated concentrations of the proinflammatory cytokines have been implicated in overreaching (overtraining) syndrome (45). Interestingly, overreaching occurs to a greater degree in some well trained athletes compared with others (16,45). Although there currently is no evidence that directly links vitamin D insufficiency to sports-related inflammation, animal studies have found that vitamin D supplementation reduces the inflammatory cycle in inflammatory bowel disease and auto-immune encephalomyelitis (55). Further, preliminary results from our work in distance runners has found that TNF-alpha concentration is elevated when 25(OH)D concentration falls below 32 ng·mL^{-1} (52). In our group of college athletes (most of whom maintained sufficient status throughout the academic year), however, there was no link between vitamin D status and overuse or inflammatory injuries (52). Additional studies in a larger population of athletes prone to overtraining injuries are needed to further evaluate this potential relationship.

VITAMIN D REQUIREMENTS FOR ATHLETES

In the United States, the AI for vitamin D is currently 200 IU for children and adults up to 50 yr of age, 400 IU for adults aged 51–70 yr, and 600 IU for adults over 70 yr (51). These recommendations are currently being reevaluated by the Institute of Medicine (IOM). Many researchers have suggested that the recommended daily allowance (RDA) may need to be set upwards of 800–2200 IU (9,21,24,26) and possibly as high as 7000 IU in certain stages of the lifecycle including pregnancy and lactation (9). The tolerable upper limit (TUL), currently set at 2000 IU, also is in need of revision (21).

Establishing a realistic RDA for vitamin D will be an important step towards helping athletes achieve optimal vitamin D status and improved health outcomes. Currently many

health professionals — including sports dietitians and physicians — are reluctant to recommend maintenance supplementation at values higher than the AI and/or TUL. The establishment of a more appropriate RDA will serve to optimize health and perhaps athletic training and performance While we await the updated recommendations, it is important that the clinician recognize that requirements are highly individualized due to their dependence on diet, endogenous synthesis, and storage. Despite increased training, there currently is no evidence to suggest that the vitamin D requirement for athletes is different from the general population.

CLINICAL ASSESSMENT, EVALUATION, AND TREATMENT

Vitamin D screening has been recommended as a routine part of the annual physical exam (9,11). In athletes, ideally screening should be measured at least biannually in association with training season and probable peak (late summer/early fall) and nadir (late winter) 25(OH)D concentration. If routine screening of all athletes is not possible, those with a history of stress fracture, frequent illness, bone and joint injury, skeletal weakness or pain, or signs of overtraining syndrome should be screened. These athletes may experience dramatic changes in health and performance if poor status is detected and immediately corrected. Careful attention also should be given to athletes with restrained eating patterns who also spend the majority of time indoors (gymnasts, ballet dancers, figure skaters, and wrestlers) as they may be at a higher risk than most athletes for poor vitamin D status. Considering the potential for reduced bioavailability of vitamin D$_2$ (11) (sometimes called vegan vitamin D), evaluation of status in strict vegetarian or vegan athletes may be warranted.

Important steps for assessing vitamin D status are outlined in Table 2. While measurement of 25(OH)D concentration is the most important serum parameter, measurement of PTH, alkaline phosphatase and serum calcium and phosphorus concentrations may provide additional information when bone density is low, stress fracture (or reaction) is evident, and/or vitamin D and calcium intakes are severely restricted. PTH concentration typically increases as 25(OH)D concentration falls below 10–20 ng·mL^{-1} (25,27,55) and correlates independently with bone density (18) and stress fracture risk (44). The history should address bone health, idiopathic muscle pain and weakness (which can be described as "heaviness in the legs"), overtraining injury, and infectious illness (including a frequency of respiratory tract infections) in all athletes, not just those with apparent musculoskeletal injury. While the physical exam may be unremarkable, specific assessment of muscle weakness and bone pain may be useful, along with a bone density scan (preferably of the hip and lumbar spine). Documentation of current and recent medications also is important because the conversion of vitamin D$_3$ to 25(OH)D in the liver uses the cytochrome P-450 enzyme system, which is altered by many medications (9,11).

The dietary assessment should focus on estimating vitamin D intake, which can be obtained by evaluating consumption frequency of the limited natural and fortified-foods, as well as supplements. In many cases, dietary assessment also should include a more detailed assessment of calcium (because vitamin D

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deficiency drastically decreases intestinal calcium absorption), magnesium, and other nutrients important to muscle function and bone health (including caffeine and vitamins A, C, and K), particularly if bone fracture or low bone density is apparent. Suboptimal magnesium intake is common in the Western diet (unless the athlete consumes ample nuts, seeds, legumes, whole grains, and green leafy vegetables) and may influence both bone and muscle function (40,43). If inflammatory injury or overtraining syndrome is evident, assessment should focus additionally on intake of omega-3 relative to omega-6-rich oils and on carbohydrate and protein intake in relation to exercise training.

Following a detailed assessment, recommendations for achieving/maintaining optimal vitamin D status can be individualized to each athlete's current 25(OH)D concentration, clinical symptoms, diet, lifestyle habits, and belief system. The recommendation to obtain 5 min (in very fair-skinned people) to 30 min (in darker-skinned people) of sunlight exposure to arms, legs, and back several times a week at close to solar noon without sunscreen (11,24) usually leads to sufficient vitamin D synthesis, as does regular use of tanning beds that emit UVB radiation (47). For example, when fair-skinned individuals sunbathe in the summer, they produce 10,000–20,000 IU of vitamin D in less than 30 min (27). Research suggests, however, that athletes with limited sun exposure may require a daily supplement providing as little as 800 to as much as 7000 $IU \cdot d^{-1}$ to achieve optimal status (9,21,24,26). Higher doses are required for athletes with little sun exposure, darker skin, and higher adiposity. Athletes who live/train at locations >35° to 37° degrees latitude (8,11) also will need supplements during the winter, even if they maintain adequate stores during the nonwinter months (18). Neither regular consumption of a multivitamin or vitamin D-fortified foods is sufficient to maintain adequate status in the absence of adequate UVB exposure (9,11).

Clinical guidelines for supplemental vitamin D intake are not yet established. However, a commonly reported rule of thumb — established from clinical studies — is to increase supplemental vitamin D intake by 1000 IU over 3–4 months for every 10 $ng \cdot mL^{-1}$ elevation in 25(OH)D desired (9). For example, a normal-weight athlete with a serum 25(OH)D concentration of 10 $ng \cdot mL^{-1}$ would require 2000 IU daily to increase stores to 30 $ng \cdot mL^{-1}$ in 3–4 months. Because the kinetics are not linear, however, more vitamin D may be needed to improve status as 25(OH)D concentration approaches more optimal ranges [i.e., more than 2000 IU may be needed to increase 25(OH)D concentration from 30 to 50 $ng \cdot mL^{-1}$]. Furthermore, higher supplemental doses most likely will be required in overweight athletes.

Athletes with deficient status may benefit from short-term, high-dose "loading" regimens to replenish stores more rapidly, particularly if clinical symptoms are present. A maintenance dose of 1000–5000 $IU \cdot d^{-1}$ of D_2 or D_3 then will be required to maintain optimal stores. High-dose regimens, which currently include 50,000 $IU \cdot wk^{-1}$, preferably of D_3, for 8–16 wk or 10,000 $IU \cdot d^{-1}$ for several weeks (9,11,24,26) should be monitored by the athlete's team or personal physician. [See Holick for additional regiments (24)]. Cod liver oil is not recommended as a source of D_3 because it contains high amounts of vitamin A, which can antagonize the action of vitamin D (12). Further, the rare athlete with malabsorption syndrome may need to be treated using sunlight or artificial UVB exposure (32). While controversial, regular use of a tanning bed, which emits UVB radiation, is shown to maintain 25(OH)D concentration within the normal limit and resolve musculoskeletal pain and weakness associated with deficiency (32). Tanning bed use also is shown to help healthy individuals (47) and athletes maintain adequate vitamin D stores (18). Finally, athletes — who often believe more is better — should be cautioned that daily supplementation with >10,000 IU could lead to toxicity (21).

LABORATORY ANALYSIS

Unfortunately, assessment, diagnosis, and treatment of vitamin D deficiency/insufficiency currently is confounded by variability in serum 25(OH)D concentration between methodologies (6,25,28) and clinical laboratories (8). A description of the assays used to assess 25(OH)D is beyond the scope of this update; however, the clinician should be aware that while modest between-laboratory correlation is found, the mean bias ranges between 2.9 and 5.2 $ng \cdot mL^{-1}$ (6). Fortunately, the National Institute of Standards and Technology, in collaboration with the National Institutes of Health and the Office of Dietary Supplements, currently is developing standards to serve as in-house quality-control references for clinical laboratories (41). Until national standardization is developed, however, the clinician should be aware of the procedures used by the employed laboratory as well as the reliability and validity of their procedures. The typical intra- and interassay variations for commercial vitamin D assays range from 8% to 15% (25). Consideration also should be given as to whether the commercial assay measures both 25(OH)D_2 and -D_3 derivatives. Assays that only measure 25(OH)D_3 may underestimate status of athletes who consume a significant portion of foods or supplements containing D_2 (specifically vegetarian and vegan athletes). The reader is referred to Hollis and Horst (28), Holick (25), and Binkey and colleagues (6) for additional information.

CONCLUSION

Given the established role of vitamin D in bone health and the more recently recognized role in immunity, inflammation, and chronic disease prevention, it is imperative that sports nutritionists and physicians routinely assess vitamin D status and make appropriate recommendations. Recent research has provided evidence to suggest that maintaining adequate vitamin D status may reduce stress fracture risk, total body inflammation, common infectious illness, and impaired muscle function. However, research is needed to determine whether insufficient vitamin D status increases an athlete's risk for injury and whether vitamin D supplementation can affect athletic training and improve overall health and performance. Future studies should include both double-blind, randomly assigned trials of vitamin D supplementation and investigations of vitamin D status in relation to adiposity, skin color, and training regimens in large populations of athletes.

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