Should We Be Concerned About the Vitamin D Status of Athletes?

Kentz S. Willis, Nikki J. Peterson, and D. Enette Larson-Meyer

A surprisingly high prevalence of vitamin D insufficiency and deficiency has recently been reported worldwide. Although very little is known about vitamin D status among athletes, a few studies suggest that poor vitamin D status is also a problem in athletic populations. It is well recognized that vitamin D is necessary for optimal bone health, but emerging evidence is finding that vitamin D deficiency increases the risk of autoimmune diseases and nonskeletal chronic diseases and can also have a profound effect on human immunity, inflammation, and muscle function (in the elderly). Thus, it is likely that compromised vitamin D status can affect an athlete’s overall health and ability to train (i.e., by affecting bone health, innate immunity, and exercise-related immunity and inflammation). Although further research in this area is needed, it is important that sports nutritionists assess vitamin D (as well as calcium) intake and make appropriate recommendations that will help athletes achieve adequate vitamin D status: serum 25(OH)D of at least 75 or 80 nmol/L. These recommendations can include regular safe sun exposure (twice a week between the hours of 10 a.m. and 3 p.m. on the arms and legs for 5–30 min, depending on season, latitude, and skin pigmentation) or dietary supplementation with 1,000–2,000 IU vitamin D$_3$ per day. Although this is significantly higher than what is currently considered the adequate intake, recent research demonstrates these levels to be safe and possibly necessary to maintain adequate 25(OH)D concentrations.

**Keywords**: exercise, training, health, exercise performance, nutrition, physical performance

Vitamin D deficiency is currently considered an epidemic for all age groups in the United States (Calvo & Whiting, 2003; Chen et al., 2007; Hanley & Davison, 2005; Holick, 2003; Nesby-O’Dell et al., 2002) and worldwide (Glerup et al., 2000; Isaia et al., 2003; Lips, 2007; Zittermann, 2003). It is widely accepted that vitamin D is necessary for proper bone formation (Holick, 2006c), but emerging evidence is also finding that vitamin D deficiency increases the risk of nonskeletal chronic diseases including cardiovascular disease, diabetes, hypertension, and certain types of cancer (Chiu, Chu, Go, & Saad, 2004; Garland et al., 2006; Holick, 2003, 2004),

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as well as autoimmune disorders such as multiple sclerosis, rheumatoid arthritis, systemic lupus erythematosus (Kamen et al., 2006), and inflammatory bowel disease (Cantorna, Munnsick, Bemiss, & Mahon, 2000). In addition, several recent studies have found that vitamin D status has a profound effect on human immunity (Cannell et al., 2006) and helps control inflammation (Cannell et al.; Moore, Piazza, McCartney, & Lynch, 2005). There is also some thought that low vitamin D status might be linked to back pain and suboptimal athletic performance (Cannell, 2006). Despite these wide-ranging health benefits, very little is known about vitamin D status among athletes and fitness enthusiasts. Considering the potential for significant effects on an athlete’s overall health and ability to train (i.e., by affecting bone health, innate immune response, and exercise-related immunity and inflammation), this is an area that warrants further research and demands the attention of the athletic community. This article reviews what is known about the vitamin D status of athletes and whether vitamin D deficiency or insufficiency is likely to affect the short- and long-term health and performance of athletes. Current implications for sports dietitians and areas of future research are also discussed.

**Vitamin D Synthesis and Sources**

Vitamin D is a unique nutrient in that physiological sufficiency can be met entirely through endogenous synthesis (Holick, 2006a; Hollis, 2005). When the skin is exposed to ultraviolet B (UVB) radiation (wavelength range of 290–315 nm), the 7-dehydrocholesterol in the plasma membranes of the epidermis and dermis is converted to precholecalciferol (previtamin D$_3$). This previtamin D$_3$ is quickly converted to the more thermodynamically stable vitamin D$_3$ (cholecalciferol) and moves from the plasma membrane to the dermal capillary bed, pulled into circulation by vitamin-D-binding protein. Once synthesized, vitamin D$_3$ undergoes obligate hydroxylations, first in the liver to 25-hydroxyvitamin D, or 25(OH)D, and then in the proximal kidney tubules (as well as other extrarenal tissues) to create the biologically active form, 1,25-dihydroxyvitamin D, or 1,25(OH)$_2$D. Because sunlight is necessary to activate this process, any factor that limits the amount or quality of sun exposure can compromise vitamin D status. Such factors include the following (adapted from Webb, 2006; Whiting & Calvo, 2005b):

- Aging (impairs synthesis capacity)
- Skin pigmentation (melanin absorbs UVB photons)
- Regular sunscreen use (SPF ≥15)
- Clothing
- Cloud cover, atmospheric pollution
- Time of day
- Wintertime latitude >35° N or S

This includes the winter season in northern or southern locales distant from the equator (latitude >~35° N or S; Kimlin, Olds, & Moore, 2007) because the solar zenith angle during these months is large and UVB photons are absorbed by the atmosphere and therefore cannot stimulate endogenous synthesis (Kimlin et al.; Webb, 2006).
Vitamin D can also be obtained in the diet from limited natural and fortified sources that include fatty fish, fortified milk, margarine, and ready-to-eat cereal (Table 1). Dietary vitamin D includes vitamin D$_3$ (cholecalciferol) and vitamin D$_2$ (ergocalciferol), which is derived from UVB radiation of the plant and yeast sterol ergosterol. After absorption, dietary vitamin D is transported to the liver and subsequently hydroxylated as previously described. Once it reaches this point, dietary vitamin D$_3$ is metabolized essentially the same as endogenously synthesized vitamin D$_3$. Vitamin D$_2$, however, appears to be only about 20–40% as effective as D$_3$ at increasing and maintaining endogenous 25(OH)D concentrations (Armas, Hollis, & Heaney, 2004; Houghton & Vieth, 2006; Trang et al., 1998).

**Assessment of Vitamin D Status**

The biologically active form of vitamin D, 1,25(OH)$_2$D, is a result of hydroxylation on the 25th carbon by the kidneys. The active 1,25(OH)$_2$D and parathyroid hormone (PTH) function to maintain intracellular and extracellular calcium concentrations within a physiologically acceptable range (Holick, 2005; Lips, 2006). Inadequate vitamin D concentrations result in decreased intestinal absorption of calcium. This creates a small decrease in serum calcium concentrations that stimulates PTH secretion. The increase in PTH mobilizes calcium and phosphorus from bone to restore serum calcium concentrations to normal. The latter effect is a result of vitamin-D-induced expression of calbindin (an intestinal calcium-binding protein).

**Table 1 Dietary Sources of Vitamin D**

<table>
<thead>
<tr>
<th>Food</th>
<th>Serving size</th>
<th>Vitamin D (IU)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cod-liver oil</td>
<td>1 Tbsp</td>
<td>1,360</td>
</tr>
<tr>
<td>Wild salmon</td>
<td>3.5 oz</td>
<td>981</td>
</tr>
<tr>
<td>Sun-dried shiitake mushrooms</td>
<td>1 oz</td>
<td>400–500</td>
</tr>
<tr>
<td>Canned sardines</td>
<td>3.5 oz</td>
<td>270</td>
</tr>
<tr>
<td>Farmed salmon</td>
<td>3.5 oz</td>
<td>249</td>
</tr>
<tr>
<td>Tuna ahi-YT</td>
<td>3.5 oz</td>
<td>164</td>
</tr>
<tr>
<td>Milk, fortified</td>
<td>8 oz</td>
<td>100</td>
</tr>
<tr>
<td>Orange juice, fortified</td>
<td>8 oz</td>
<td>100</td>
</tr>
<tr>
<td>Cod</td>
<td>3.5 oz</td>
<td>80</td>
</tr>
<tr>
<td>Yogurt, fortified</td>
<td>4–6 oz</td>
<td>8–80</td>
</tr>
<tr>
<td>Margarine, fortified</td>
<td>1 Tbsp</td>
<td>60</td>
</tr>
<tr>
<td>Kraft 2% milk singles (American)</td>
<td>1 slice (21 g)</td>
<td>40</td>
</tr>
<tr>
<td>Cereal, fortified (10% DV)</td>
<td>3/4–1 cup</td>
<td>40</td>
</tr>
<tr>
<td>Egg yolk</td>
<td>1</td>
<td>18</td>
</tr>
</tbody>
</table>

*b* Chen et al., 2007.  
*c* Holick, 2005.  
*d* Food-label values.  
*e* Murphy et al. (2001) found that only 47.7% of 648 samples of milk collected over a 4-year period in New York State were within the acceptable range for vitamin D (400–600 IU/quart). Most milk samples that were out of compliance were underfortified. Chen, Shao, Heath, and Holick (1993) found that 49% of 173 milk samples collected in the United States and Canada contained less than 80% of the recommended vitamin D content.
and an epithelial calcium-channel protein (Holick, 2005), which increases the efficiency of calcium and phosphorus absorption by active transport across the intestinal mucosa.

The inactive but major circulating form of the vitamin, 25(OH)D, is used as the clinical measure of vitamin D status because it provides a more stable and meaningful measure of vitamin D status than does the biologically active form (Holick, 2005; Lips, 2004) for several reasons. First, as stated earlier, compromised vitamin D status will trigger an increase in PTH. The increased PTH concentration signals the kidney to increase production of 1,25(OH)₂D. This serves to maintain normal, or even slightly elevated, 1,25(OH)₂D concentrations when vitamin D status is in fact compromised. Second, 1,25(OH)₂D has a relatively short half-life (<4 hr; Holick, 1990), and its concentrations are ~1,000 times lower than its precursor, 25(OH)D. With a reported half-life of 2–8 weeks (Holick, 2004; Vieth, 1999), 25(OH)D concentrations are much more stable than 1,25(OH)₂D. Finally, 25(OH)D is also used as a substrate for most extrarenal tissues, which have the capacity to synthesize 1,25(OH)₂D for their own use but depend on adequate serum concentration of 25(OH)D (Whiting & Calvo, 2005b).

Prevalence of Vitamin D Deficiency in the General Population

A surprisingly high prevalence of vitamin D insufficiency and deficiency (Calvo & Whiting, 2003), along with the reemergence of pediatric rickets (Weisberg, Scanlon, Li, & Cogswell, 2004), has recently been reported in the United States and Canada and is also thought to be an issue worldwide (Calvo, Whiting, & Barton, 2005; Glerup et al., 2000; Isaia et al., 2003; Lips, 2007; Zittermann, 2003). Although results from the National Health and Nutrition Examination Survey (NHANES III) suggest that Americans consume more than the recommended intake of vitamin D (group mean >280 IU/day; Calvo et al.), many studies indicate that serum 25(OH)D concentrations are often deficient. One study of 165 young adults age 18–29 years living in Boston found that of 36% were vitamin D deficient at the end of winter, with 25(OH)D concentrations ≤50 nmol/L (Tangpricha, Pearce, Chen, & Holick, 2002). Another study of otherwise healthy individuals (age 10–65 years) who had persistent nonspecific musculoskeletal-pain syndromes found that 93% had some degree of vitamin D deficiency (serum concentration ≤50 nmol/L) and 28% had severe deficiency—25(OH)D concentrations ≤20nmol/L (Plotnikoff & Quigley, 2003). In addition, a very recent study of 307 urban adolescents living in Massachusetts who were screened during their annual physical examinations found that 24% were deficient, 25(OH)D ≤37.5 nmol/L, and 4.6% were severely deficient, 25(OH)D ≤20 nmol/L (Gordon, DePeter, Feldman, Grace, & Emans, 2004). Given these and other recent findings, many experts feel that the recommended adequate intake and tolerable upper limit for vitamin D might be too low to maintain bone health and reduce chronic-disease risk (Bischoff-Ferrari, Giovannucci, Willett, Dietrich, & Dawson-Hughes, 2006; Cannell, 2006; Chen et al., 2007; Dawson-Hughes et al., 2005; Heaney, 2005; Vieth, 2006; Whiting & Calvo, 2005b). One current difficulty in assessing vitamin D status, however, is that exact thresholds for serum concentrations of 25(OH)D that define deficiency, insufficiency, and
adequacy have not been uniformly agreed on (Hollis, 2005; Lips, 2004; Whiting & Calvo, 2005a). In most cases serum concentrations of 25(OH)D of <20–25 nmol/L are defined as deficient (Heaney, 2004) or severely deficient (Vieth, 1999) because of their association with clinical osteomalacia among adults, whereas serum concentrations of 20–25 nmol/L and the lower end of the typical reference (37.5–50 nmol/L) are defined as insufficiency (Heaney, 2004; Snijder et al., 2005) or marginal deficiency (Vieth, 1999), but the terms are also used interchangeably (Tangpricha et al., 2002). Insufficiency, however, appears to be the currently favored term for the range of marginal deficiency and is the theoretical serum concentration that is not high enough to protect against certain chronic diseases (Whiting & Calvo, 2005a). As indicated in Table 2, circulating concentrations of 20–29 nmol/L will prevent rickets, but concentrations of at least 75–80 nmol/L are needed to support optimal health and disease prevention (Chen et al., 2007; Dawson-Hughes et al.; Heaney, 2005; Vieth, 2006; Whiting & Calvo, 2005b).

Although vitamin D insufficiency appears to be problematic in the general population, studies are also finding that individuals of certain ethnic and racial groups (Calvo & Whiting, 2003; Harris & Dawson-Hughes, 1998; Harris, Soteriades, Coolidge, Mudgal, & Dawson-Hughes, 2000; Looker, Dawson-Hughes, Calvo, Gunter, & Sahyoun, 2002; Nesby-O’Dell et al., 2002; Plotnikoff & Quigley, 2003) and those with specific dietary patterns (Calvo et al., 2005; Davey et al., 2003; Outila, Karkkainen, Seppanen, & Lamberg-Allardt, 2000; Whiting & Calvo, 2005b) might be at even greater risk. The prevalence of vitamin D insufficiency, for example, is found to be higher in Black men and women living in the United States and Canada (Calvo & Whiting; Harris & Dawson-Hughes; Harris et al., 2000; Nesby-O’Dell et al.) and in Canadian Aboriginal women (Weiler, Leslie, Krahn, Steiman, & Metge, 2007) than in similar-age light-skinned individuals. Although some of these differences might result from reduced vitamin D intake

### Table 2 Vitamin D Recommendations

<table>
<thead>
<tr>
<th>Source</th>
<th>Recommended serum 25(OH)D (nmol/L)a</th>
<th>Recommended intake (IU/day)b</th>
<th>Recommended tolerable upper limit (IU/day)b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bischoff-Ferrari et al., 2006</td>
<td>90–100</td>
<td>&gt;1,000</td>
<td>—</td>
</tr>
<tr>
<td>Cannell, 2006</td>
<td>125</td>
<td>Up to 5,000</td>
<td>—</td>
</tr>
<tr>
<td>Dawson-Hughes et al., 2005</td>
<td>70–80</td>
<td>800–1,000</td>
<td>—</td>
</tr>
<tr>
<td>Hathcock, Shao, Vieth, and Heaney, 2007</td>
<td>—</td>
<td>—</td>
<td>10,000</td>
</tr>
<tr>
<td>Heaney, 2005</td>
<td>80</td>
<td>2,600</td>
<td>10,000</td>
</tr>
<tr>
<td>Chen et al., 2007</td>
<td>75–150</td>
<td>800–1,000</td>
<td>—</td>
</tr>
<tr>
<td>Hollis, 2005</td>
<td>80</td>
<td>Possibly &gt;2,000</td>
<td>—</td>
</tr>
<tr>
<td>Lips, 2004</td>
<td>50</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Vieth, 2006</td>
<td>75</td>
<td>2,000</td>
<td>—</td>
</tr>
</tbody>
</table>

aTo convert these values to ng/mL, divide by ~2.5. bRecommendations are for vitamin D₃, cholecalciferol.
Vitamin D and Athletes

Vitamin D appears to be a largely ignored nutrient in sports nutrition. Very few investigations and reviews focusing on the micronutrient status of athletes and their potential performance implications have even included discussions of vitamin D. Of the studies that have assessed vitamin D intake (Table 3), it has been found that athletes in general report consuming far less than the current daily recommendation for vitamin D despite presumably having higher energy needs than the general population (which should also presumably result in higher energy and nutrient intakes). It is important to note, however, that the nutrient-composition databases used to quantify vitamin D intake (including the USDA national nutrient database) do not have current data on the vitamin D content of many foods (Calvo et al., 2005). A current and comprehensive database is necessary to properly evaluate dietary intake. Although research has demonstrated that African American men and women in the general population have a lower intake of vitamin D from milk and ready-to-eat cereals than do Whites, comparative data are not available for athletes (Calvo et al.).

Even less appears to be known about the prevalence of vitamin D insufficiency in athletes. In our review of the literature, we were able to locate only three published studies reporting the vitamin D status of athletic populations (Bannert, Starke, Mohnike, & Frohner, 1991; Lehtonen-Veromaa et al., 1999; Maimoun et al., 2006). In one study of young Finnish female athletes (runners and gymnasts) and nonathletes, age 9–15 years, nearly 68% were vitamin D insufficient, serum 25(OH)D <37.5 nmol/L, in the winter months, and 13% of those were deficient, serum 25(OH)D <10 nmol/L. The prevalence of vitamin D insufficiency and deficiency, however, was not different in the athletes than in the nonathletes. Although the investigators found that the mean 25(OH)D concentration in the group was
drastically improved at the end of the summer, they were not significantly affected by 3 months of supplementation of 400 IU of vitamin D (Lehtonen-Veromaa et al.). In another study of 85 athletes age 8–27 in intensive training in the former East Germany, 37% were vitamin D deficient, serum 25(OH)D <25 nmol/L. The decreased serum 25(OH)D concentrations, nevertheless, did not appear to correlate with the retarded bone maturation also noted in a large number of the children, particularly the gymnasts (Bannert et al.). In a third recently published study, 7 male competitive road cyclists living around Montpellier, France, and training an average of 16 hr/week had serum 25(OH)D concentrations indicative of adequate status (81 ± 16 nmol/L; M ± SD); however, the group mean was surprisingly (considering their outdoor training schedules) only borderline sufficient, and the number with insufficient status was not reported (Maimoun et al., 2006). In agreement with these studies, preliminary results from our laboratory found that 40% of a small group (N = 20) of distance runners age 19–45 residing in Baton Rouge, LA (latitude 30° N), were vitamin D insufficient, 25(OH)D <80 nmol/L, despite training outdoors and living at a latitude where endogenous vitamin D synthesis is possible throughout the year (Willis, Peterson, & Larson-Meyer, 2007). In this study, however, there were no significant relationships between vitamin D status and season tested, sex, weekly training, or vitamin supplementation.
Potential Impact of Vitamin D Status on Health and Performance

Because vitamin D plays an important role in bone health and the prevention of autoimmune and chronic diseases, it is also likely that this vitamin affects health and performance in athletes. Specifically, adequate vitamin D status might increase an athlete’s chance of maintaining good health during training by reducing risk of stress fractures and keeping the immune system and inflammatory responses in check. There is also some evidence that vitamin D status might affect athletic performance. The following section will review the potential impact of vitamin D on the health and performance of athletes.

Impact of Vitamin D Status on Bone Health in Athletes

The classic function of vitamin D is its influence on bone health through its regulation of calcium homeostasis. The first-discovered and most well-known symptoms of vitamin D deficiency are diseases such as rickets in children and osteomalacia and osteoporosis in adults. Although athletes are at a low risk for such diseases, a less severe deficiency can still negatively affect bone health. In fact, serum concentrations of 25(OH)D have been found to be related to bone-mineral density (Bischoff-Ferrari et al., 2006; Valimaki et al., 2004), with the greatest density observed at 25(OH)D concentrations nearing 100 nmol/L (Bischoff-Ferrari et al., 2006). These associations have been observed in younger (20–49 years), as well as older (>50 years), individuals.

Stress fractures are a common problem for many athletes and can represent a serious obstacle to training. Although reports vary, some studies of track and field athletes demonstrate cumulative annual incidences as high as 21% (Snyder, Koester, & Dunn, 2006). Studies of other college athletes report much lower incidences of 1–3%. Fortunately, some of these cases might be preventable. In one study, an increased risk of fracture in young males was significantly associated with decreased serum 25(OH)D concentrations (Ruohola et al., 2006). In another, stress fractures were associated with higher PTH concentrations (which are directly influenced by calcium intake and vitamin D status), even though 25(OH)D concentrations were not directly related (Valimaki et al., 2005). Furthermore, supplementation with vitamin D has been shown to decrease incidence of fractures in older populations (Bischoff-Ferrari et al., 2006) and is of great interest in athletic populations. In fact, preliminary data from a Creighton University study indicate a significant decrease in stress-fracture incidence among female naval recruits supplemented with vitamin D and calcium when compared with a control (Lappe, Cullen, Thompson, & Ahlf, 2007). From these findings it is evident that compromised vitamin D status can diminish bone health, even in otherwise healthy athletes. This compromised status might increase stress-fracture risk, an undesirable outcome for an athlete in training.

Impact of Vitamin D on Gene Expression and Cell Growth

Our understanding of the noncalcitropic functions of vitamin D has increased dramatically over the past 25 years. Although it has long been recognized that vitamin
D influences bone health by up-regulating the expression of genes that enhance calcium absorption and bone deposition, vitamin D also functions as a modulator of the expression of many other genes involved in cell growth and immunity. In this role the hormonally active form, \(1,25(\text{OH})_2\text{D}\), interacts with its nuclear vitamin D receptor, which is present in most tissues and cells in the human body—including intestine and bone, as well as brain, heart, immune cells (Stumpf, Sar, Reid, Tanaka, & DeLuca, 1979), and skeletal muscle (Bischoff-Ferrari et al., 2004; Simpson, Thomas, & Arnold, 1985). The vitamin D–vitamin D receptor complex subsequently bonds with the retinoic acid \(x\)-receptor, which is then recognized by specific gene sequences known as vitamin D response elements to regulate the expression of specific genes involved in cell growth, immune function, and protein synthesis. In immune cells, the vitamin D–vitamin D receptor complex appears to regulate T- and B-lymphocyte function, cytokine production, macrophage activation, and monocyte maturation (Adorini, 2002; Deluca & Cantorna, 2001). In skeletal muscle, the vitamin D–vitamin D receptor complex is hypothesized to regulate protein synthesis, although its specific action is not yet known (Costa, Blau, & Feldman, 1986; Simpson et al.). Higher concentrations of 25(OH)D in circulation are imperative to this function because 25(OH)D is used directly by the cell (which contains 1-\(\alpha\)-hydroxylase) to make \(1,25(\text{OH})_2\text{D}\). These specific functions of vitamin D represent a small, albeit vital, piece of its involvement in the innate immune response and possibly also physical performance.

**Impact of Vitamin D Status on Immunity**

In addition to its direct functions in immune-cell proliferation, vitamin D is also involved in another important aspect of the innate immune system. Recent research has uncovered a direct link between vitamin D status and the release of disease-fighting antimicrobial peptides (AMPs; Gombart, Borregaard, & Koeffler, 2005; Liu et al., 2006; Wang et al., 2004). AMPs can be secreted by many components of the innate immune system, including (but not limited to) neutrophils, natural killer cells, macrophages, monocytes, mast cells, and epithelial cells in the respiratory tract (Cannell, 2007; Gombart et al.). Generally these AMPs exert their effects by compromising the integrity of the cell membrane of invading pathogens (De Smet & Contreras, 2005). The release of AMPs such as cathelicidin (CAMP) is triggered when invading pathogens are recognized by toll-like receptors. Liu et al. demonstrated that stimulation of the toll-like receptors triggers a conversion of 25(OH)D\(_3\) to the active \(1,25(\text{OH})_2\text{D}\) and induces the expression of the vitamin D receptor that promotes the release of CAMP.

Considering that vitamin D is such a potent stimulator of human AMPs, vitamin D status can potentially have significant effects on innate immunity. In fact, Liu et al. (2006) discovered that African Americans with reduced 25(OH)D concentrations exhibit a decreased ability to synthesize CAMP. These findings might help explain the increased susceptibility to tuberculosis observed in populations that exhibit lower 25(OH)D concentrations (Sita-Lumsden, Lapthorn, Swaminathan, & Milburn, 2007). Furthermore, it has been demonstrated that a single dose of vitamin D\(_3\) (100,000 IU) can enhance innate immune response and restrict the growth of mycobacteria (Martineau et al., 2007). Cannell et al. (2006) also suggest that the ability of vitamin D to regulate the expression of AMPs in the epithelial cells of the respiratory tract could influence an individual’s susceptibility to the influenza virus.
In fact, preliminary data have suggested that 3 years of supplementation with vitamin \(D_3\) (800 IU for 2 years and 2,000 IU for 1 additional year) reduced self-reported incidence of influenza and the common cold and abolished the seasonality of these infections (Aloia & Li-Ng, 2007). In this manner, seasonal fluctuations in vitamin D status could, in part, explain the seasonality of viral respiratory infections.

These findings in a normal population might be directly applicable to athletes who are at similar or increased risk for infection and illness. Although not all studies are in agreement, some research suggests that prolonged intense training has a suppressive effect on innate immune function and increases risk of upper respiratory tract infection (Nieman, 2000; West, Pyne, Renshaw, & Cripps, 2006). A specific mechanism has yet to be elucidated but would likely involve multiple aspects of the immune system. Although there are currently no published studies addressing the effects of exercise on AMP function, suppressed CAMP activity resulting from decreased vitamin D status could certainly affect an athlete’s immune status. Further research is necessary to determine the effect (if any) that vitamin D supplementation might have on infection and illness in athletes in training.

**Impact of Vitamin D Status on Exercise-Related Inflammation**

Vitamin D might also work through the immune system to control inflammation, which results from the accumulation of fluid and immune cells in injured tissue. Vitamin D has been shown to increase the production of anti-inflammatory cytokines, transforming growth factor, and interleukin-4 (IL-4; Cantorna, Woodward, Hayes, & DeLuca, 1998) and reduce production of the proinflammatory cytokines interleukin-6 (IL-6), interferon–\(\gamma\), interleukin-2 (IL-2), and tumor necrosis factor (Lemire, 1992; Muller, Diamant, & Bendtzen, 1991; Rigby, Denome, & Fanger, 1987; Rigby, Noelle, Krause, & Fanger, 1985; Rigby, Stacy, & Fanger, 1984; Zhu, Mahon, Fröicu, & Cantorna, 2005). Although there is no evidence to date that directly links vitamin D insufficiency to sports-related inflammation, several of the proinflammatory cytokines, particularly IL-6, are elevated after a single bout of exercise (Ostrowski et al., 1998), and this unexplainably occurs to a greater degree in some well-trained athletes than in others (Edwards, Burns, Ring, & Carroll, 2006; Ostrowski et al.). The release of IL-6 also appears to be increased after intensified periods of training (Robson-Ansley, Blannin, & Gleeson, 2007). Elevated concentrations of the proinflammatory cytokines are hypothesized to be involved in the overreaching (or overtraining) syndrome associated with high-volume training (Smith, 2000). In this cascade of events, muscle or skeletal trauma induces release of the proinflammatory cytokine IL-6, which activates conversion of circulating monocytes to macrophages. Macrophages in turn produce large quantities of other proinflammatory cytokines including IL-1beta, tumor necrosis factor, and additional IL-6. These elevated circulating cytokines might then coordinate a whole-body (or systemic) inflammatory response by communicating with the brain to alter mood and behavior and with the liver to up-regulate gluconeogenesis and de novo synthesis of acute phase proteins, both of which are classic symptoms of overreaching. Although it is not completely known whether and how vitamin D influences the inflammatory cascade, studies in mice have found that vitamin D supplementation reduces the inflammatory cycle in inflammatory bowel disease and autoimmune encephalomyelitis (an animal model of multiple sclerosis; Cantorna et al., 2000) and could therefore also be involved in exercise-associated inflammation.
Based on our current understanding of the role that vitamin D plays in inflammation, it is possible that compromised vitamin D status increases the risk or severity of certain sports-related injuries, a possibility that was alluded to in a 1950s German report (Spellerberg, 1952). In that report, athletes training at the Sports College of Cologne experienced a significant reduction in chronic pain from sports injuries after an extensive 6-week program of irradiation (on both sides of their bodies for up to 10 min twice a week) with a “central sun lamp.” The report also stated that athletes experienced improved performance, but this was poorly defined. Several large-scale studies have also found that adequate vitamin D status protects against cartilage loss and disease progression in individuals with knee osteoarthritis (McAlindon et al., 1996) and reduces disease risk in those with rheumatoid arthritis (Merlino et al., 2004). In osteoarthritis, vitamin D most likely acts to prevent softening of the joint cartilage, whereas in rheumatoid arthritis it most likely acts to reduce immune response and inflammation. In a small experimental study in multiple sclerosis patients, 6 months of supplementation with 1,000 IU vitamin D was also found to suppress serum concentrations of the anti-inflammatory cytokine transforming growth factor but did not conclusively alter concentrations of the proinflammatory cytokines interferon–γ, IL–2, and tumor necrosis factor (Mahon, Gordon, Cruz, Cosman, & Cantorna, 2003).

Impact of Vitamin D Status on Performance

A recent review in the Vitamin D Newsletter presented evidence that vitamin D might benefit athletic performance. According to the article, numerous studies were performed in the early part of the 20th century, mostly by Russian and German scientists, that indirectly suggested that vitamin D status might affect athletic performance (Cannell, 2007). Specifically, most of the evidence suggests that irradiation with a sun lamp that provides artificial UVB radiation (and increased endogenous vitamin D synthesis) was associated with improved athletic performance and was perhaps a well-kept secret of former Soviet and German athletic powers (Cannell, 2007). To our knowledge, however, current studies have investigated the effect of vitamin D status on physical performance only in the elderly. These studies have found that poor vitamin D status is significantly associated with reduced muscle strength and poor physical function (Houston et al., 2007; Mowe, Haug, & Bohmer, 1999; Wicherts et al., 2007). Physical function was assessed by evaluating grip strength, stair-climbing ability, outdoor activity, and fall occurrence (Mowe et al.); a short physical-performance battery (Houston et al.); and specific walking, standing, and balance movements (Wicherts et al.). Some (Bischoff et al., 2003; Dhesi et al., 2004) but not all studies (Kenny, Biskup, Robbins, Marcella, & Burleson, 2003) have even demonstrated that vitamin D supplementation can improve muscle function and lessen the risk of falling in older adults. In these studies, a single high dose (600,000 IU ergocalciferol; Dhesi et al.) appeared to be more effective at improving muscle function at 6 months than regular low-dose supplementation (1,000 IU cholecalciferol plus 500 mg calcium/day; Kenny et al.). Furthermore, Bischoff et al. were able to demonstrate a significant decrease in falls after only 3 months of supplementation with vitamin D plus calcium (800 IU + 1,200 mg/day) when compared with those supplemented with calcium (1,200 mg/day) alone. Although the specific action of vitamin D is not known, Sato, Iwamoto, Kanoko, and Satoh (2005) found that improvements in strength with vitamin D supplementation...
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(1,000 IU/day for 2 years) were correlated with increases in the relative number and size of Type II muscle fibers in female stroke survivors. In addition, patients with vitamin D deficiency often complain of aching bones and muscle discomfort, which is thought to be caused by a mineralization defect that innervates pain fibers (Tangpricha et al., 2004; Whiting & Calvo, 2005a).

Vitamin D Requirements for Athletes

In the most recent revision of the dietary recommendations for Americans and Canadians, the adequate daily intakes for vitamin D are 200 IU for children and adults up to 50 years of age, 400 IU for adults age 51–70, and 600 IU for adults 71 years of age or older. Most vitamin D experts agree, however, that the recommended adequate intake falls short for optimizing health in today’s sun-avoiding societies (Glerup et al., 2000; Heaney, 2005; Hollis, 2005; Vieth, 2004; Whiting & Calvo, 2005b). Many are also advocating for a U.S. governmental reevaluation of the vitamin D requirements (Glerup et al.; Heaney, 2005; Holick, 2005; Hollis; Vieth et al., 2007) that would include establishing an estimated average requirement (Whiting & Calvo, 2005a) from emerging evidence linking poor vitamin D status to increased risk of chronic and autoimmune diseases. Although specific recommendations of many of these experts vary from 1,000 to 4,000 IU/day (Table 2), it is important to keep in mind that the oral requirement depends on both endogenous synthesis and vitamin D storage (Calvo et al., 2005). As an example, recent research has estimated that without adequate sun exposure, individuals of all ages require at least 600 and 1,000 IU (Bischoff-Ferrari et al., 2006; Boonen et al., 2006; Glerup et al.; Heaney, Davies, Chen, Holick, & Barger-Lux, 2003; Holick, 2006b; Larsen, Mosekilde, & Foldspang, 2004), much higher than the current recommended adequate intake.

Despite increased training, there is no evidence to suggest that the vitamin D requirement for athletes is different than for the general population. Athletes at risk of poor vitamin D status include those with low vitamin D intake or limited sun exposure. The latter includes athletes who only practice indoors or those who practice outdoors in the early mornings and late afternoons, as well as those who diligently apply sunscreen, wear athletic clothing with sun-protection factors (which is becoming increasingly available from many athletic clothing companies), or have dark-pigmented skin. In addition, athletes with high or low body fat might also be at increased risk for vitamin D insufficiency during the winter months (or other periods of reduced UVB exposure). In normal-weight individuals, vitamin D$_3$, synthesized in the skin (or consumed in the diet) is stored in subcutaneous fat and released as needed during the winter months (Chen et al., 2007; Glerup et al., 2000). There is evidence to suggest, however, that this process is ineffective in obese individuals with excess body fat because vitamin D might be sequestered deep in the subcutaneous fat, decreasing its availability for conversion into 25(OH)D (Wortsman, Matsuoka, Chen, Lu, & Holick, 2000). This is supported by the observations that 25(OH)D concentrations are inversely correlated with body-fat percentage (within the typical range of sedentary individuals; Snijder et al., 2005) and are commonly depressed with obesity (along with elevated PTH concentrations; Bell et al., 1985; Hypponen & Power, 2007; Snijder et al., 2005; Yanoff et al., 2006). Athletes with extremely low body fat, on the other hand, might also be at increased risk during
the winter months simply because of a decreased ability to store vitamin D in subcutaneous tissues, although this is currently purely speculation.

**Vitamin D Intoxication**

Although intoxication from excess intake or supplementation is extremely rare, it can be caused by ingesting (intentionally or unintentionally) extremely high supplemental doses. Doses of 150,000–2,604,000 IU per day for 2 years, for example, have been found to raise 25(OH)D concentrations to 1,217 nmol/L and induce both hypercalcemia and hyperphosphatemia (Koutkia, Chen, & Holick, 2001). Doses of 10,000 IU per day for up to 5 months do not cause toxicity (Vieth, 2004) and appear to be safe (Heaney et al., 2003). A recent report described a case of vitamin D intoxication that was caused by manufacturer error in which 188,640 IU of vitamin D had been added to a vitamin supplement (serving size = six capsules/day) instead of the intended 400 IU (Klontz & Acheson, 2007). In this case, the patient was admitted (after 2 months of supplementation) with severe hypercalcemia (>3.75 mmol/L) and elevated serum 25(OH)D (1,171 nmol/L) concentrations and presented with classic signs and symptoms of toxicity including fatigue, constipation, back pain, forgetfulness, nausea, and vomiting. Prolonged hypercalcemia is known to produce soft-tissue calcification, hypertension, and heart-rhythm abnormalities. Possible vitamin D intoxication issues are important to consider in the athletic population because many athletes, coaches, and trainers hold the philosophy that “if a little is good, more is better.” Vitamin D intoxication from solar exposure, on the other hand, does not lead to toxicity because excess previtamin D (that does not escape into the circulation) photoisomerizes to biologically inert photoproducts with prolonged UV exposure (Holick, 1981).

**Recommendations and Practical Applications**

Clearly, additional research is needed to determine whether vitamin D can influence training and performance in athletes. Given its role in bone health and recently recognized role in immunity, inflammation, and chronic-disease prevention it is important that sports nutritionists assess vitamin D, as well as calcium, intake and evaluate potential risk for vitamin D insufficiency (or deficiency). Direct assessment of vitamin D status via measurement of 25(OH)D concentrations might also be helpful, but the assay is costly and not readily available to athletes. Assessment of calcium intake is important because adequate calcium intake reduces vitamin D turnover through suppression of PTH (Lips, 2004).

It is not known exactly how much vitamin D sports nutritionists should recommend to their athletes and active clients. Individual researchers (Table 2) along with the Vitamin D Council (Cannell, 2006), however, are currently recommending between 800 and 2,000 IU/day. Because it is very difficult to regularly obtain that much vitamin D from food sources, sensible sun exposure or vitamin D supplementation is required. Holick (2007) recently suggested that exposing arms and legs to sunlight for 5–30 min (depending on season, latitude, and skin pigmentation) between the hours of 10 a.m. and 3 p.m. twice a week without sunscreen should be adequate to maintain vitamin D status (Chen et al., 2007). Sunscreen with an SPF of at least 15 should then be applied to prevent damaging
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effects of excessive sunlight exposure (Holick, 2005). During the winter months, however, vitamin D supplementation is required for athletes living at northern and southern latitudes distant from the equator (>35° N or S; Holick, 2005; Kimlin et al., 2007).

The recommendation to obtain vitamin D via a dietary supplement is favored by many experts because of the reported link between skin cancer and excessive sunlight exposure, especially sunlight that causes sunburn (Kennedy, Bajdik, Willemze, De Grujil, & Bouwes Bavinck, 2003; Wolpowitz & Gilchrest, 2006). To be most effective, supplements should contain vitamin D₃ (cholecalciferol) rather than vitamin D₂ (ergocalciferol) and should not contain vitamin A, which can interfere with the function of vitamin D (Trang et al., 1998). It is also a good idea that supplements be purchased from a reputable company. Keep in mind, however, that vitamin D₃ is not acceptable to vegans and many vegetarians because it is commercially synthesized from animal fat (Holick, 2005). Vitamin D₂, the plant form acceptable to vegetarians and the form used to fortify products targeted at vegetarians (including rice and soy milks), is much less effective than vitamin D₃ at increasing concentrations of 25(OH)D (Armas et al., 2004; Houghton & Vieth, 2006; Trang et al.). Vitamin D scientists are also questioning whether higher doses of vitamin D₂ offer the same overall benefits of vitamin D₃ in preventing bone fractures (and possible other chronic conditions) and whether high doses might lead to a higher risk of toxicity than vitamin D₃ (Houghton & Vieth). Because vitamin D supplements are commonly available in 1,000-IU doses, sports nutritionists might want to consider athletes’ dietary intake, frequency of sun exposure, skin color, body size, history of stress fractures, occurrence of illness or bone and joint injury, and recent serum 25(OH)D concentration, if available, when making recommendations. Indeed, the vitamin D content in a multivitamin (400 IU) is likely not sufficient.

Frequent use of tanning beds, which typically emit 2–6% UVB radiation, has also been suggested as an alternative to meeting vitamin D needs (Chen et al., 2007), but this recommendation meets with obvious controversy. Regular users of tanning beds have been found to have robust serum concentrations of 25(OH)D (approx 112 nmol/L) and higher bone density in the hip at the end of winter than nontanners, who had concentrations of approximately 45 nmol/L (Tangpricha et al., 2004). Exposure to a tanning bed for 30–50% of the typical time recommended with sunscreen on the face was found to be effective at maintaining vitamin D status in patients with fat malabsorption (Koutkia, Lu, Chen, & Holick, 2001).

Conclusions

Given the known role of vitamin D in bone health and its recently recognized role in chronic-disease prevention, immunity, and inflammation, it is imperative that sports nutritionists assess vitamin D status and make appropriate recommendations that will help athletes achieve adequate vitamin D status. Careful attention should be paid when assessing athletes with restrained eating patterns who also spend most of their time indoors (gymnasts, ballet dancers, figure skaters, wrestlers) because they might be at an even greater risk of insufficiency. Considering the relative inefficiency of vitamin D₂, strict vegetarian or vegan athletes who spend most of their time indoors might also be at increased risk. Although the recommendation to achieve adequate vitamin D status, 25(OH)D of at least 75 or 80 nmol/L, appears
to be important for improving overall health and reducing risk of autoimmune and chronic disease, athletes with a history of stress fracture, frequent illness, or bone and joint injury are also likely to have more short-term benefits. Research is needed, however, to determine whether insufficient vitamin D status increases an athlete’s risk of illness or injury and whether vitamin D supplementation can affect athletic training and improve overall health and performance.

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References


