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VITAMIN D AND ATHLETIC PERFORMANCE  
A CRITICAL ASSESSMENT FOR COACHES AND ATHLETES

By

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B.S., California Lutheran University, Thousand Oaks, CA, 2009

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Vitamin D and Athletic Performance:  
A Critical Assessment for Coaches and Athletes

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In addition to the well-established role of vitamin D in bone health, there is evidence to suggest that vitamin D may also be associated with the prevention of a wide array of non-skeletal, autoimmune, and chronic diseases. In addition, correlational studies suggest that maintaining a sufficient vitamin D status may positively influence athletic performance indicators, namely strength, power, speed, cardiorespiratory fitness, reaction time, coordination, and body composition. However, there is a lack of consensus among the Institute of Medicine and various vitamin D researchers in defining ideal vitamin D concentrations for both health and performance related benefits. Although this controversy has led to conflicting evidence regarding the extent of vitamin D deficiency among the general population, the few studies examining the vitamin D status of athletes exclusively suggest that a large percentage of athletes may be insufficient or deficient in vitamin D. These deficiencies seem to be most common in the winter and spring regardless of geographic location or whether the athlete competes indoors or outdoors. Although direct interventional studies measuring vitamin D concentrations and athletic performance indicators are needed to confirm the association between vitamin D and athletic performance, this paper will demonstrate the potential mechanisms underlying the potential association as well as summarize the current vitamin D recommendations that may enhance health and performance in athletes.

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## Table of Contents

Chapter One: The Background of Vitamin D and its Importance .....	1
Vitamin D Synthesis and Sources .....	1
Vitamin D Metabolism.....	1
Measuring Vitamin D Status .....	2
Vitamin D Deficiency in the General Population .....	3
The Role of Vitamin D in Bone Health.....	4
The Role of Vitamin D in Chronic Disease Prevention, Immunity and Inflammation.....	4
The Role of Vitamin D in Skeletal Muscle Tissue and Function.....	5
Vitamin D Status among Athletes.....	5
Historical Perspective of the Potential Impact of Vitamin D Status on Performance and Overall Health in Athletes.....	6
Purpose .....	7
Chapter Two: Review of Literature .....	8
Vitamin D Requirements for Athletes.....	8
Vitamin D Intake of Athletes .....	9
Factors that May Affect Vitamin D Status in Athletes .....	10
Geographic location & season.....	11
Altitude.....	12
Race.....	12
Body composition.....	13
Indoor sports .....	14
Sunscreen.....	16
Early or late in the day training.....	16
Restrictive eating .....	17
Vitamin D and the Health of Athletes.....	17
The Association between Vitamin D and Athletic Performance Variables .....	19
Strength/power/speed .....	20
Cardiorespiratory fitness.....	24
Reaction time and coordination.....	28

Body composition.....	30
Chapter Three: Conclusions and Recommendations .....	35
Conclusions .....	35
Recommendations .....	36
Appendix A: A Summary of Vitamin D Recommendations for Athletes .....	41
References .....	42

## **Chapter One: The Background of Vitamin D and its Importance**

### **Vitamin D Synthesis and Sources**

Vitamin D is a fat-soluble vitamin produced in the skin by the action of ultraviolet-B irradiance (wavelength 290-315 nm) on 7-dehydrocholesterol in the deeper epidermis layers of the skin, converting it to pre-vitamin D<sub>3</sub>, which then quickly undergoes a heat-induced isomerization to form vitamin D<sub>3</sub> (Lehmann & Meurer, 2010). Vitamin D<sub>3</sub> is also contained in dietary sources, yet in quantities thought to be insufficient to achieve optimal serum vitamin D concentrations (Whiting, Langlois, Vatanparast, & Greene-Finestone, 2011). While the richest natural sources of dietary vitamin D<sub>3</sub> are found in wild-caught fatty fish, foods such as ready-to-eat breakfast cereals and milk are fortified with vitamin D<sub>3</sub>. Vitamin D<sub>3</sub> can also be taken orally through supplemental forms that generally come from ultraviolet-B irradiated sheep's wool lanolin or fish oil (NIH Office of Dietary Supplements, 2011). Fortified foods and supplements often contain vitamin D<sub>2</sub> as well, which is derived from yeast and mushrooms (NIH Office of Dietary Supplements, 2011). Although vitamin D<sub>2</sub> is metabolized the same as vitamin D<sub>3</sub> in the body, vitamin D<sub>3</sub> is considered a more favorable source of vitamin D, because it has a stronger affinity for vitamin D-binding protein and is two-thirds more effective in raising and maintaining adequate serum vitamin D concentrations (Armas, Hollis, & Heaney, 2004; Houghton, & Vieth, 2006).

### **Vitamin D Metabolism**

Regardless whether vitamin D<sub>3</sub> or vitamin D<sub>2</sub> is synthesized in the skin or consumed through dietary or supplemental sources, it requires a two-phase metabolism in the body to be

converted to its active form (Lehmann & Meurer, 2010). Vitamin D<sub>3</sub> and D<sub>2</sub> are first hydroxylated in the liver and converted its intermediate form—25 hydroxyvitamin D [25(OH)D]. The kidney and other organs then further hydroxylate 25(OH)D to its physiologically active form—1,25-dihydroxyvitamin D [1,25(OH)<sub>2</sub>D](Lehmann & Meurer, 2010). In its active form, vitamin D binds to vitamin D binding protein and is able to activate vitamin D receptors located in various tissues and organs in the body (Lehmann & Meurer, 2010).

### **Measuring Vitamin D Status**

Serum concentrations of the intermediate form of vitamin D [(25(OH)D] are used as the clinical measure of vitamin D status, because it is thought to provide a more meaningful measurement than its physiologically active form [1,25(OH)<sub>2</sub>D]. For instance, the half-life of 25(OH)D is ~ 3 weeks, whereas 1, 25(OH)<sub>2</sub>D has a half-life ~ 4 hours. This longer half-life of 25(OH)D is able to provide a more accurate indication of vitamin D stores in the body (Zerwekh, 2008). In addition, when an individual is deficient in vitamin D, there is a subsequent increase in serum parathyroid hormone secretion, which signals the kidneys to increase the production of 1, 25(OH)<sub>2</sub>D. Therefore, measuring serum 1, 25(OH)<sub>2</sub>D may be misleading, because concentrations may appear to be normal or even above-normal, although the individual may in fact be deficient (Chapuy et al., 2007; Souberbielle, 2003; Vieth, Ladak,& Walfish, 2003). For this reason, I will be referring to the intermediate form of vitamin D, 25(OH)D, when referencing vitamin D measured in research throughout the rest of this manuscript (Table 1).

**Table 1. Forms of Vitamin D**

<b>Form</b>	<b>Abbreviation</b>	<b>Description</b>
Previtamin D3	-	An intermediate in the production of vitamin D3; converted from 7-dehydrocholesterol in response to ultraviolet-B irradiance (wavelength 290-315 nm)
Vitamin D3	-	The primary form of vitamin D; converted from previtamin D3 following a heat-induced isomerization; dietary and supplemental sources are animal-derived
Vitamin D2	-	Dietary and supplemental sources are plant-derived
25-hydroxyvitamin D	25(OH)D	The intermediate form of vitamin D; hydroxylated in the liver; used as the clinical measure of vitamin D status due to its relatively long serum half-life
1,25-dihydroxyvitamin D	1,25(OH) <sub>2</sub> D	The physiologically active form of vitamin D; hydroxylated in the kidney and other organs; has a short serum-half life; binds to vitamin D binding protein and activates vitamin D receptors

### **Vitamin D Deficiency in the General Population**

There is conflicting evidence regarding the prevalence of vitamin D deficiency and insufficiency within the general population, which may be attributed to the lack of consensus among the Institute of Medicine and various vitamin D researchers in defining adequate vitamin D concentrations (Table 2). For instance, vitamin D researchers estimate that 50% of people worldwide are vitamin D deficient (serum 25(OH)D concentrations < 20 ng/mL) or insufficient (serum 25(OH)D concentrations = 21-29 ng/mL) (Holick, 2007). Contrarily, the Centers for Disease Control and Prevention estimate that only 8% of the U.S. population is at risk of vitamin D deficiency (serum 25(OH)D concentrations < 12 ng/mL), which demonstrates that vitamin D deficiency may not be as widespread as many believe it to be, and that there are plenty of people with sufficient vitamin D concentrations, despite their predisposition to various risk factors (Looker et al., 2011). Nevertheless, studies examining vitamin D status across different populations have shown that vitamin D deficiencies can extend across age groups and ethnicities

regardless of lifestyle or geographic location, including otherwise healthy individuals (Binkley et al., 2007; Gordon, DePeter, Feldman, Grace, & Emans, 2004; Looker et al., 2011; Levis et al., 2005; Yetley, 2008).

**Table 2. Serum 25(OH)D Sufficiency, Insufficiency, and Deficiency Classifications**

<b>Source</b>	<b>Sufficiency</b>	<b>Insufficiency</b>	<b>Deficiency</b>
The Institute of Medicine (IOM) & Centers for Disease Control and Prevention (CDC) Classifications	20-50 ng/mL	12-19 ng/mL	< 12 ng/mL
Non-IOM and CDC Classifications*	30-60 ng/mL	21-29 ng/mL	< 20 ng/mL

\* Bischoff-Ferrari et al., 2006; Cannell et al., 2006; Dawson-Hughes et al., 2005; Heaney, 2005; Hollis, 2005; Lips, 2004; Vieth, 2006

### **The Role of Vitamin D in Bone Health**

The effect of vitamin D on calcium and phosphorus metabolism is perhaps the most widely known function of vitamin D. By increasing intestinal calcium absorption to maintain adequate serum calcium concentrations, vitamin D is assumed to be essential for optimal bone health and preventing bone disorders such as rickets, osteoporosis and stress-related fractures (Holick, 2004).

### **The Role of Vitamin D in Chronic Disease Prevention, Immunity and Inflammation**

Additional health-related benefits of vitamin D have been elucidated in the past few decades with the identification of vitamin D receptors in nearly every tissue and cell in the human body including the intestine, bone, brain, heart, and immune cells (Holick, 2004). These discoveries have led to emerging evidence that vitamin D may be associated with the prevention of non-skeletal, autoimmune and chronic diseases such as cardiovascular disease, hypertension,

diabetes, various forms of cancer, rheumatoid arthritis and inflammatory bowel disease, and may also influence immune function and inflammatory responses (Zitterman, 2003).

### **The Role of Vitamin D in Skeletal Muscle Tissue and Function**

While vitamin D deficiency has long been associated with muscle pain and weakness, the mechanisms for this relationship have not yet been determined (Hamilton, 2010). However, there have been two vitamin D receptors identified in muscle tissue—one acting as a nuclear receptor and the other located at the cell membrane. This suggests that muscle responds to the physiologically active form of vitamin D and may help to explain the effects of vitamin D on skeletal muscle function (Birge et al., 2001; Simpson, Thomas, & Arnold, 1985).

### **Vitamin D Status among Athletes**

There have only been a handful of correlational studies that have focused on the vitamin D status among athletes exclusively. Although there is not enough evidence to suggest that athletes are more or less susceptible to a compromised vitamin D status than the general population, each of the studies that has examined vitamin D concentrations among athletes has reported high percentages of athletes to be either vitamin D deficient, insufficient, or borderline sufficient (according to definitions of vitamin D deficiency adopted by both the Institute of Medicine and vitamin D researchers), regardless of geographic location or whether the athlete competes indoors or outdoors (Hamilton et. al, 2010; Halliday, Peterson, & Thomas, 2011; Lehtonen-Veromaa et al, 1999; Lovell, 2008; Maimoun et al., 2006; Shindle et al., 2011; Willis, Broughton, & Larson-Meyer , 2009). As will be discussed in greater detail later in this manuscript, the only two factors to seemingly predict vitamin D deficiencies among athletes

have been skin pigmentation and the season of the year (Halliday et al., 2011; Lehtonen-Veromaa et al., 1999; Shindle et al., 2011).

### **Historical Perspective of the Potential Impact of Vitamin D Status on Performance and Overall Health in Athletes**

Given the role of vitamin D in bone health and its association with chronic disease reduction, immunity, inflammation and muscle function, vitamin D has the potential to influence physical and athletic performance (Cannell, 2009). In fact, the athletic performance enhancing effects of vitamin D have been assumed by the former Soviet Union and its satellite states for decades. In the early 20<sup>th</sup> century, Russian and German researchers used sunlamps to stimulate vitamin D production in their elite athletes. The increases in athletic performance and reductions in chronic pain seen in the irradiated athletes ignited controversy to whether vitamin D supplementation could constitute as “athletic unfairness” or “doping” (Cannell, 2009). Since then, additional studies have investigated the effects of a compromised vitamin D status on various health outcomes that may affect athletes, including bone and stress fractures, a compromised immune system, muscle injuries, and musculoskeletal pain, as well as athletic performance indicators, namely strength, power, speed, cardiorespiratory fitness, reaction time, coordination and body composition (Al Faraj & Al Mutairi, 2003; Al Mheid et al., 2009; Ardestani et al., 2007; Arunabh, Pollack, Yeh, & Aloia, 2003; Caan, 2007; Dhesi et al., 2004; Erikssen & Rodahl, 1979; Halliday et al., 2011; Kremer, Campbell, Reinhardt, & Gilsanz, 2009; Kristal-Boneh, Froom, Harari, Malik, & Ribak, 2000; Lappe et al., 2008; Mowry, Costello, & Heelan, 2009; Peterson et al., 2011; Plotnikoff & Quigley, 2003; Rosenblum, Castro, Moore, & Kaplan, 2008; Ruohola et al., 2008; Sato, Iwamoto, Kanoko, & Satoh, 2005; Shahar et al., 2010;

Shindle et al., 2011; Sibley, Turner, & Earthman, 2009; Stockton, Mengersen Paratz, Kandiah, & Bennell; 2011; Svedenhag & Sjodin, 1985; Ward et al., Willis et al., 2009).

## **Purpose**

Athletes are always searching for a competitive edge, and evidence has shown the potential of vitamin D to make a difference in athletic performance (Applegate & Grivetti, 1997). Athletes must be provided with appropriate recommendations for attaining adequate and safe vitamin D concentrations. Therefore, the purpose of this paper is threefold: to bring awareness of the effects of vitamin D status on the ability to train and perform, to examine the potential mechanisms that may explain the association between vitamin D and athletic performance, and to summarize current vitamin D recommendations that might enhance health and performance in athletes.

## Chapter Two: Review of Literature

### Vitamin D Requirements for Athletes

Despite the high energy demands of training for sport, there is no evidence to suggest that athletes require more vitamin D than the general population (American College of Sports Medicine, American Dietetic Association, & Dietitians of Canada, 2009). Athletes, thereby, are encouraged to follow the recommended dietary allowances (RDA) established by the Institute of Medicine for all healthy adults (American College of Sports Medicine et al., 2009; Whiting & Barabash, 2006). Currently, the Institute of Medicine recommends 600 International Units (IUs) daily for all people ages 1-70. The Institute of Medicine believes that these recommendations are adequate to attain a vitamin D concentration of  $> 20$  ng/mL—the minimal level needed for physical performance benefits in addition to a range of health outcomes (The Institute of Medicine, 2010).

The Institute of Medicine also has set tolerable upper intake level of vitamin D to caution that vitamin D may be harmful if taken in excess amounts. They conclude that once intakes of vitamin D surpass 4,000 IUs per day for people ages  $> 9$ , the risk for kidney and tissue damage also increases (The Institute of Medicine, 2010).

A component of the scientific community of vitamin D researchers disagrees with the values established by the Institute of Medicine. Based on published, randomized clinical trials, prospective and cross-sectional studies, and dose-response relations studies, they argue that a vitamin D concentration of 20 ng/mL is insufficient to meet the skeletal and physiological demands of vitamin D within the body and to prevent adverse health outcomes (Bischoff-Ferrari, Giovannucci, Willett, Dietrich, & Dawson-Hughes, 2006, Cannell, 2006; Dawson-Hughes et al.,

2005; Heaney, Davies, Chen, Holick, & Barger-Lux, 2003; Heaney, 2005; Hollis, 2005; Lips, 2004; Vieth, 2006; Vieth et al., 2007). While there is no agreement on an optimal serum 25(OH)D concentration, these studies have suggested that a desirable concentration may lie between ~30-60 ng/mL (Bischoff-Ferrari et al., 2006; Cannell et al., 2006; Dawson-Hughes et al., 2005; Heaney, 2005; Hollis, 2005; Lips, 2004; Vieth, 2006). To attain these concentrations, researchers have suggested that the RDA for vitamin D be increased from 600 IU/day to 800-2,600 IU/day and possibly as high as 5,000 IU/day (Bischoff-Ferrari et al., 2006; Cannell, 2006; Dawson-Hughes et al., 2005; Heaney, 2005; Hollis, 2005; Vieth, 2006). In addition, researchers also argue that the tolerable upper intake level is set so low that it prevents people from improving their vitamin D status. Several support that a safe, upper intake level for adult vitamin D consumption should be revised to 10,000 IU/day (Table 3) (Hathcock, Shao, Vieth, Heaney, 2007; Heaney, 2005; Vieth et al., 2007).

**Table 3. Dietary Vitamin D Needed to Reach Serum 25(OH)D Sufficiency**

<b>Source</b>	<b>Recommended Dietary Allowance (IU/day)</b>	<b>Upper Level Intake (IU/day)</b>
The Institute of Medicine Recommendation	600 IU*	4,000 IU
Non-IOM Recommendations**	800-5,000 IU	10,000 IU

\*For people ages 1-70 years

\*\*Bischoff-Ferrari et al., 2006; Cannell, 2006; Dawson-Hughes et al., 2005; Heaney, 2005; Hollis, 2005; Vieth, 2006

### **Vitamin D Intake of Athletes**

Studies that have assessed vitamin D dietary intake in athletes by methods of 3-4 day food recalls and food frequency questionnaires have all shown that athletes consume far less vitamin

D than the current Institute of Medicine recommendation of 600 IU/day, not to mention the 800-5,000 IU/day that vitamin D researchers and experts recommend (Bergen-Coco & Short, 1992; Bescós & Rodríguez, 2011; Clark, Reed, Crous, & Armstrong, 2003; Halliday et al., 2011; Lehtonen-Veromaa et al., 1999; Rankinen et al., 1998; Ziegler, Nelson, Barratt-Fornell, Fiveash, & Drewnowski, 2001). It is important to note that both 3-4 day food recalls and food frequency questionnaires are self-reported dietary assessments, and are thus subject to inaccuracies in total dietary intake (Thompson & Byers, 1994). In addition, when these studies were conducted, The Nutrient Data Laboratory of the US Department of Agriculture, which is used to quantify vitamin D intake, did not contain accurate values for the vitamin D2 and D3 content in many foods (Holden & Lemar, 2008). Nevertheless, results from these studies suggest that athletes should not rely on dietary sources alone to attain an adequate vitamin D status.

### **Factors that May Affect Vitamin D Status in Athletes**

There are many factors that may impair the cutaneous production of pre-vitamin D3 in athletes in response to ultraviolet-B radiation from the sun (Cannell et al., 2009, Larson-Meyer & Willis, 2010; Willis et al., 2008). Environmental factors include geographic location and season, altitude, early-or late-day training and training indoors. Behavioral factors include sunscreen and restrictive eating. Racial and anthropometric risk factors include skin pigmentation and body composition. As outlined below, athletes who meet any of these risk factors are at an increased risk of developing and sustaining vitamin D insufficiency or deficiency which, subsequently, may undermine athletic potential (Cannell et al., 2009; Larson-Meyer & Willis, 2010; Webb, 2006; Willis et al., 2008).

**Geographic location & season.** During the fall and winter seasons, there is an inverse correlation between latitude and the intensity of ultraviolet-B rays (Kimlin, Olds, & Moore, 2007). As a result, cutaneous pre-vitamin D<sub>3</sub> synthesis may be significantly reduced or non-existent at latitudes > ~35° North or South during the months of approximately November-February (Kimlin 2008; Kimlin, et al., 2007; Webb, Kline, Holick, 1988). Therefore, it is speculated that people who live in these geographic locations may have a poor vitamin D status in the winter months if they do not get adequate ultraviolet-B radiation exposure in the sunnier months (Kimlin 2008; Kimlin, Olds, & Moore, 2007; Webb, Kline, Holick, 1988).

Studies examining the vitamin D status of athletes, however, have not all shown the expected relationship between latitude and vitamin D concentrations. For instance, the lowest vitamin D concentrations ever documented in athletes were in those training in the Middle East (25.4°N; (~11 ng/mL) during an unspecified season, while the highest vitamin D concentrations were observed in Laramie, Wyoming (41.3°N; 49.0 ± 16.6 ng/mL) during the fall, and during unspecified seasons in both Baton Rouge, Louisiana (30.5°N; 38.7± 17.2 ng/mL) and Montpellier, France (43.6 °N; 32 ng/mL) (Halliday et al., 2011; Hamilton et al., 2010; Maimoun et al., 2006, Willis et al., 2009). Similarly, a recent meta-analysis of cross-sectional studies examining the effect of latitude on vitamin D concentrations in the general population reported that latitude did not seemingly predict vitamin D status (Hagenau et al., 2009). Other factors influencing vitamin D status, including clothing, time spent outdoors, vitamin D dietary intake, and various other genetic factors, may explain the lack of association between latitude on vitamin D status noted in these studies (Hagenau et al., 2009).

On the other hand, studies in athletes have consistently shown that vitamin D status is highly dependent upon the season. Halliday et al. (2011) found that in Laramie, Wyoming

(41.3°N), vitamin D concentrations reached their lowest point in the winter compared to the fall and spring. Similarly, Lehtonen-Veromaa et al. (1999) found that vitamin D concentrations averaged  $13.6 \pm 5.6$  ng/mL in the winter and increased to  $25.2 \pm 6.0$  ng/mL in the summer.

**Altitude.** Since the intensity of ultraviolet-B radiation increases at higher elevations, altitude is another factor that may influence cutaneous production of pre-vitamin D<sub>3</sub> in athletes (D. Rigel, E. Rigel & A. Rigel, 1999). There have been no known studies that have directly compared vitamin D concentrations to various solar altitudes. One study, however, compared the effects of altitude on ambient ultraviolet-B radiation in New York (40°N; 60 feet above sea level) and Vail, Colorado (39°N; 8,500 feet above sea level). The study found that despite the two cities being at a similar latitude, ultraviolet-B radiation readings in New York were approximately 60% less than those in Vail, Colorado. The study further suggested that there is an 8-10% increase in ultraviolet-B intensity per 1,000 feet of elevation gain (Rigel et al., 1999). The effect of altitude on cutaneous production of pre-vitamin D<sub>3</sub> may help to explain why among some of the highest documented vitamin D concentrations in athletes were in high-altitude city of Laramie, Wyoming (7,201 feet above sea level), regardless of the season (Halliday et al., 2011).

**Race.** Dark-skinned athletes, who have greater levels of melanin in their skin, are more likely to have lower concentrations of vitamin D than those with light-colored skin (Cannell et al., 2009, Larson-Meyer 2010, Webb, 2006; Willis et al., 2008). Melanin blocks the absorption of ultraviolet-B radiation, thus impairing the amount of pre-vitamin D<sub>3</sub> capable of being synthesized from sun exposure (Webb, 2006). Demographic studies conducted in the general

population show that the prevalence of vitamin D insufficiency is greatest among African-Americans compared with other ethnic groups in North America (Ginde, Liu, & Carmargo Jr., 2009). In the only known study to compare vitamin D status among athletes of different races, Shindle et al., (2011) examined vitamin D concentrations of 31 white and 58 black professional football players from a single NFL in the spring and found that mean vitamin D concentrations were significantly lower in black players compared to white players (20.4 ng/mL vs. 30.3 ng/mL;  $P < 0.001$ ).

**Body composition.** Athletes with either high or low body fat may also face an increased risk of a poor vitamin D status (Larson-Meyer & Willis, 2010; Willis et al., 2008). Studies in non-athletes have shown that lower vitamin D concentrations are more common in obese people than in those with a normal weight, and that these low vitamin D concentrations are inversely associated with a high percentage of body fat (Snijder, 2005; Wortsman, Matsuoka, Chen, Lu, & Holick, 2000). The mechanisms for these observations are not yet clear. However, it is speculated that in normal-weight individuals, pre-vitamin D<sub>3</sub> is stored in subcutaneous fat and released during times of reduced ultraviolet-B radiation exposure, such as the winter months (Holick, 2007). This process is thought to be impaired in individuals with excess body fat—pre-vitamin D<sub>3</sub> become trapped deep within their greater amounts of subcutaneous fat which, consequently, impairs the conversion of vitamin D<sub>3</sub> to 25(OH)D (Blum, 2008; Wortsman et al., 2000). Although it is also not clear whether individuals with low body fat face an increased risk of a poor vitamin D status, it is thought these individuals have a decreased ability to store pre-vitamin D in subcutaneous tissues, which may reduce the total production of 25(OH)D as well. (Hall, Sparks & Aris, 2010; Larson-Meyer & Willis, 2010; Willis et al., 2008)

Halliday et al. (2011) is the only known study to have compared vitamin D status to body composition in athletes participating in various sports. Although vitamin D concentrations were measured throughout the entire academic year, body composition was only measured once, in the springtime, using dual energy x-ray absorptiometry (DEXA). Assuming that body fat did not vary over the course of academic year, the study found a tendency for vitamin D concentrations to correlate negatively with body fat percentages in both the fall ( $r = -0.13$ ;  $P = .07$ ) and spring ( $r = -0.22$ ;  $P = 0.10$ ) in the 21-NCAA Division I collegiate athletes who were examined. No specific body fat percentages were given in the article, so it is unclear whether there was a critically high or low body fat percentage that significantly reduced vitamin D concentrations. In addition, it is not clear whether vitamin D deficiency affects body fat, or if body fat is a result of vitamin D deficiency.

**Indoor sports.** Athletes who train and compete indoors on the basis of their sport may not be exposed to the sun as often or as long as outdoor athletes, which may increase their risk of a compromised vitamin D status (Cannell et al., 2009; Larson-Meyer & Willis, 2010; Willis et al., 2008). Halliday et al. (2011) found that athletes participating in indoor sports such as wrestling, basketball and swimming had significantly lower vitamin D concentrations in the fall than athletes participating in outdoor sports, including football, soccer, cross-country or track and field and cheerleading ( $53.1 \pm 17.4$  vs.  $39.3 \pm 8.9$  ng/mL,  $P = 0.013$ ). The results of this study are in agreement with two other studies that examined vitamin D status among indoor athletes and reported a high percentage of vitamin D insufficient and deficient athletes. One of these studies was conducted in in the Germany in the early 1990s. The original article is written in German and is not accessible through scholarly databases in the United States. According to a

review by Cannell et al., (2009), German researchers examined 85 male and female competitive gymnasts (age 8-27) in the former East Germany and found that 77% these athletes had vitamin D concentrations < 35 ng/mL, and 37% had concentrations < 10 ng/mL. More recently, Lovell (2008) examined the vitamin D status of 18 female gymnasts (age 10-17) in Australia just two months after the end of the Australian summer season and found that 83% of the females had vitamin D concentrations < 30 ng/mL and 33% had levels < 20 ng/mL. This suggests indoor athletes may be at higher risk for insufficient vitamin D.

Inadequate vitamin D concentrations have been reported in outdoor athletes as well (Hamilton, Grantham, Racinais, & Chalabi, 2010; Willis et al., 2009). Hamilton et al., (2010) evaluated vitamin D concentrations in 93 Middle Eastern sportsmen (age 13-45), 79% of who trained outdoors. The study found that 100% of the athletes had a suboptimal vitamin D status—91% of athletes had vitamin D concentrations < 20 ng/mL, and 58% had concentrations < 10 ng/mL. In another study examining vitamin D status in outdoor athletes, Willis (2009) examined the vitamin D concentrations of 19 healthy male and female distance runners (age 19-45) in Louisiana who were on a standardized exercise and diet regimen. Blood samples taken after 36-hours after the last exercise bout revealed that 42% of the athletes had vitamin D concentrations < 32 ng/mL, which the researchers classified as borderline sufficient. In a third study examining vitamin D status in outdoor athletes, Maimoun et al. (2006) evaluated vitamin D concentrations of 7 competitive male cyclists age 20-39 living around Montpellier, France who had been training for an average of  $7 \pm 1.2$  years for ~16 hours/week. Vitamin D concentrations averaged ~32 ng/mL when measured on two different days—a concentration that most vitamin D researchers would classify as borderline sufficient. Taken together, these studies show that

participation in an outdoor sport does not entirely ensure that an athlete has an adequate vitamin D status.

**Sunscreen.** Sunscreen is designed to reduce or completely eliminate ultraviolet-B radiation from reaching the skin (Webb, 2006). In fact, Matsuoka, Ide, Wortsman, MacLaughlin, Holick, (1987) measured the effects of sunscreen on cutaneous production of vitamin D<sub>3</sub> and found that mean vitamin D concentrations did not increase after a single exposure to 1 MED of artificial light in participants who properly applied a sunscreen with a sun protection factor of 8, whereas unprotected participants increased vitamin D concentrations from  $1.5 \pm 1.0$  to  $25.6 \pm 6.7$  ng/mL. The researchers also found that chronic application of a sunscreen resulted in vitamin D deficiency. After participants chronically applied sunscreen with a sun protection factor of 8, vitamin D concentrations were  $16.1 \pm 1.3$  ng/mL compared to  $36.6 \pm 2.5$  ng/mL in the unprotected participants. This suggests that even athletes who train and compete outdoors and receive abundant sun exposure year-round may face a compromised vitamin D status if they regularly apply sunscreen greater or equal to a sun protection factor of 8.

**Early or late in the day training.** In the United States, ultraviolet-B radiation is the strongest midday--generally between the hours of 10 a.m. and 4 p.m. (United States Environmental Protection Agency). Even if atmospheric conditions are ideal, outdoor athletes who don't train during this time period may not be receiving adequate sunlight to achieve optimal vitamin D levels (Halliday et al., 2011; Holick, 1995; Larson-Meyer & Willis, 2010).

**Restrictive eating.** Restrictive eating behaviors practiced by athletes in sports that encourage a low body weight for better performance or appearance face a greater risk of vitamin D deficiency (i.e. distance runners, dancers, gymnasts, wrestlers, etc.). This is because the low caloric diets often demonstrated by these athletes are likely to lead to an insufficient dietary vitamin D3 intake (Bergen-Cico & Short, 1992; Lehtonen-Veromaa et al., 1999; Rankinen et al., 1998; Ziegler, Nelson, Barratt-Fornell, Fiveash, & Drewnowski, 2001). Likewise, vegetarian athletes who do not consume vitamin D3 from animal products and/or dairy may also be at risk of vitamin D deficiency (Craig, 2010). The major sources of dietary vitamin D3 for these athletes are fortified foods, which do not contain as much vitamin D3 per gram as natural sources (Chan, Jaceldo-Siegl, & Fraser, 2009). Vegans may have the most trouble attaining an adequate vitamin D status. Because both natural and supplemental vitamin D3 sources are derived from animals, strict vegans only consume foods fortified with vitamin D2, which may be less effective in raising and maintaining adequate vitamin D concentrations (Armas et al., 2004; Davey et al., 2002; Houghton & Vieth, 2006). In addition, the high-fiber content of vegetarian diets may increase vitamin D excretion and thus decrease absorption (Batchelor & Compston, 1983). At this time, however, there is no evidence to suggest that vegetarians and vegans are more susceptible to a compromised vitamin D status than meat-eaters.

### **Vitamin D and the Health of Athletes**

An adequate vitamin D status has been shown to be an essential component in reducing the incidence of stress-related bone fractures in active individuals. In one study that followed 800 male military recruits over 90-days, Ruohola et al., (2006) found that low average vitamin D concentrations predicted the incidence of stress-related fractures in the recruits. In fact, recruits

with vitamin D levels below the median (~30 ng/mL) were 3.6 times more likely to experience a stress-related fracture than recruits who were above the median. Another study showed that supplementing with 800 IU of vitamin D and 2,000 mg calcium per day for an 8-week period decreased the incidence of stress-related fractures by 21% among 3,700 female naval recruits when compared with a control group (8.6% vs. 6.8%;  $P = 0.02$ ) (Lappe et al., 2008). The results of these two studies highlight the importance maintaining an adequate vitamin D status during training for bone health.

In addition, vitamin D may also be an important component in immune function and inflammatory responses in athletes. Halliday et al., (2011) reported that low vitamin D concentrations in collegiate athletes in the spring was positively correlated with frequency of illness, which included upper respiratory infection, the common cold, influenza, and gastroenteritis. Another study in 19 collegiate athlete endurance runners also found that the inflammatory marker tumor necrosis factor significantly increased in the runners when vitamin D concentrations were  $< 32$  ng/mL ( $P = 0.001$ ) (Willis, et al., 2009). These findings suggest that athletes who maintain an adequate vitamin D status may be protected from illness and infection and experience lesser amounts of systematic inflammation that result from sports training (Halliday et al., 2011; Willis et al., 2009).

Low vitamin D concentrations have also been associated with musculoskeletal pain in otherwise healthy adults. For instance, Plotnikoff & Quigley (2003) assessed 150 male and female primary care outpatients (aged 10-65 years) with persistent, nonspecific musculoskeletal pain and reported that 93% were vitamin D deficient (serum 25(OH)D concentrations  $\leq 20$  ng/mL) and 28% were severely deficient (serum 25(OH)D concentrations  $\leq 8$  ng/mL). Similarly, in a study of 360 Saudi Arabians who had been attending spinal and internal medicine clinics

with unexplained low back pain for more than 6 months, Al Faraj & Al Mutairi (2003), found that 83% were either mildly vitamin D deficient (serum 25(OH)D concentrations = 15-22.4 ng/mL), moderately deficient (serum 25(OH)D concentrations = 10-14.9 ng/mL) or severely deficient (serum 25(OH)D concentrations = < 10 ng/mL). After an oral dosing of 5,000-10,000 U/day of Vitamin D3 for three months, 100% of the patients normalized their vitamin D concentrations, and 95% reported disappearance of low back pain.

Only one known study has investigated the association between vitamin D status and muscle injuries in athletes. Shindle et al., (2011) examined vitamin D concentrations of 16 professional football players who had lost practice or game due to a muscle injury the previous season. It was reported that the 16 players who had sustained a previous muscle injury had an average vitamin D concentration of 19.9 ng/mL, compared to the 24.7 ng/mL seen in the non-injured athletes.

Although all of the described studies were correlational, together they suggest that maintaining an adequate vitamin D status may reduce the incidence of illness and infections in athletes and be protective against injuries that occur during training or competition.

### **The Association between Vitamin D and Athletic Performance Variables**

Given the relation of vitamin D to bone health, immunity, and inflammation, there is the potential of vitamin D to influence various athletic performance variables, namely strength, power, speed, cardiorespiratory fitness, reaction time, coordination, and body composition. The following sections will review each of these variables and describe its association with vitamin D from both an athletic performance and potential mechanistic standpoint.

**Strength/power/speed.** There has only been one known study to investigate the relationship between vitamin D status and strength and power tests directly related to athletic performance. Ward et al. (2009) measured baseline vitamin D concentrations in 99 post-menarchal females' age ( $13.5 \pm 0.58$  years). These concentrations were compared to scores from a jumping mechanography test that measured muscle power, velocity and jump height. After controlling for BMI and weight, the study found a significant association between vitamin D concentrations and jump velocity ( $P = 0.002$ ), jump height ( $P = 0.005$ ), power ( $P = 0.003$ ) and force ( $P = 0.05$ ). The researchers did not adjust for physical activity, nor did they control for menstrual phase during training. However, the results of this study suggest that vitamin D status may influence muscle power and force in athletes.

Studies investigating the association between vitamin D and sprint speed are scarce as well. As reported by Cannell et al., (2009) in their review, Russian researchers found that irradiation with a sunlamp improved 100-meter sprint times in four students when compared to non-irradiated controls, both groups were engaged in identical training programs. However, the methodology of this 1938 study is unclear. The original article is written in Russian and is not accessible through scholarly databases in the United States. Without knowing more detail about the methodology of the study, including the length of sunlamp use, exposure time, and the age and gender of the participants, it makes it difficult to draw definite conclusions regarding effectiveness of vitamin D on increasing sprint speed

Despite the lack of studies that show an association between vitamin D and strength, power and/or speed, mechanistic studies certainly support a positive relationship. Two studies conducted in rats reported that vitamin D has a positive influence on increasing the number and size of type II muscle fibers (Birge & Haddad, 1975; Wassner, Li, Sperduto & Norman, 1983),

which are critical for anaerobic performance in strength-power athletes and sprinters (Kraemer, Fleck, & Evans, 1996). Birge & Haddad (1975) administered an oral dose of vitamin D3 (400 IU) to vitamin D deficient rats. The researchers reported increased muscle ATP concentrations and protein synthesis in the rats, suggesting that vitamin D may have a direct effect on skeletal muscle metabolism and growth. Wassner et al., (1983) later measured growth and muscle composition in vitamin D deficient rats before and after supplementation with vitamin D3. This study found that rats given a single dose of vitamin D3 (400 IU) or a chronic dose (75-80 IU 2x/week). Both treatment groups saw increases in muscle mass, weight gain, and muscle anabolism and a decrease in the rate of myofibrillar protein degradation.

Studies evaluating the effect of vitamin D supplementation on muscle strength in human adults are less clear. Stockton et al., (2011), in their meta-analysis of 52 studies examining the effects of vitamin D supplementation on muscle strength, concluded that vitamin D supplementation had no significant effect on grip strength (SMD -0.02, 95%CI -0.15,0.11) or proximal lower limb strength (SMD 0.1, 95%CI -0.01,0.22) in adults with vitamin D concentrations > 10 ng/mL. However, one study included in the meta-analysis showed that vitamin D2 supplementation in individuals who were vitamin D deficient (serum 25(OH)D concentrations < 10 ng/mL) saw increases in hip muscle strength (Sato et al., 2005). This particular study followed 96 vitamin D deficient elderly women for two years post-stroke. Patients were randomly assigned to a treatment group who received 1,000 IU vitamin D2 daily or a control group receiving placebo. Along with increases in hip muscle strength, this study also reported a decreased number of falls and increases in the relative number and size of type II fibers among individuals in the vitamin D supplementation treatment group (Sato et al., 2005).

A muscle biopsy study examining vitamin D supplementation on muscle strength that was not included in the meta-analysis also presents evidence of a positive association between vitamin D supplementation and muscle fiber growth. Sorenson et al., (1979) treated eleven osteoporotic patients with 1 alpha-hydroxycholecalciferol (a synthetic vitamin D3 analogue) and calcium for 3-6 months. Muscle biopsies taken before and after the treatment revealed that there was an increase in the relative number and cross sectional area of type IIa fibers. This study also found that total muscular ATP and creatine phosphate stores were low before the treatment, and increased to normal levels after the treatment period.

The identification of vitamin D receptors in skeletal muscle suggest that skeletal muscle is a target organ for vitamin D, and it may also explain the association between vitamin D and strength, power, and speed performance (Birge et al., 2001; Simpson et al., 1985). These vitamin D receptors contain various genotype polymorphisms that have previously been associated with bone health, immunity responses, and various chronic diseases (Uitterlinden, Fang, Van Meurs, Pols, & Van Leeuwen, 2004). The FokI and BsmI polymorphisms, in particular, have also been associated with increased muscle strength.

The FokI polymorphism involves the transition from thymine (T) to cytosine (C) on the translation site of the vitamin D receptor gene (Ceglia, 2009). The C variant of the FokI polymorphism results in a shorter vitamin D receptor than those with the T variant, and this shorter vitamin D receptor is associated with higher vitamin D receptor activity. Therefore, it is thought that individuals who are homozygous for the C variant may have greater muscle strength (Ceglia, 2009). However, this has been contradicted in two different studies examining vitamin D receptors and muscle strength. Roth, Zmuda, Cauley, Shea, & Ferrell (2004) found that the C variant of the FokI polymorphism was associated with less quadriceps strength in healthy elderly

men ( $33.0 \pm 1.3$  vs.  $40.6 \pm 2.2$  kg;  $P = 0.004$ ). Similarly, Hopkinson et al. (2008) reported that patients with chronic obstructive pulmonary disease and healthy control subjects who were homozygous for the C allele of the FokI polymorphism had less quadriceps strength compared to those with the T allele ( $36.5 \pm 12.2$  vs.  $41.2 \pm 14.0$  kg ;  $P = 0.01$ ). Neither of the two studies controlled for vitamin D concentrations. While these studies suggest that the FokI polymorphism influences muscle strength, it is not clear why the variant with higher vitamin D receptor activity is not associated with this effect (Roth et al., 2004).

The BsmI polymorphism of the vitamin D receptor gene may also be associated with higher vitamin D receptor activity and, thereby, influence muscle strength. Guesens et al., (1997) investigated the association between specific allelic variations of the BsmI polymorphism muscle strength in 501 non-obese women (age > 70 years). After controlling for confounding factors, including age, calcium intake, and bone mineral density, the study reported that the BsmI polymorphism bb allele was associated with 23% greater quadriceps strength ( $P < 0.01$ ) and 7% greater grip strength ( $P < 0.05$ ) compared to women with the BsmI polymorphism BB allele. There were no differences in vitamin D concentrations between the two alleles. Contrarily, Grundberg et al., (2004), in a study of 175 healthy women (age 20-39 years), later reported that women with the BsmI BB allele had greater hamstring strength compared to women with the BsmI bb allele BB ( $62.1 \pm 8.7$  vs.  $55.5 \pm 10.2$  Nm;  $P = 0.03$ ). This study did not control for vitamin D concentrations among the participants. While these two studies suggest that the BsmI polymorphism may influence muscle strength, the specific allelic variant responsible for this effect is unclear.

Other genomic and non-genomic actions of vitamin D in skeletal muscle may also influence strength and power performance. For one, studies conducted in chicks have shown

vitamin D to regulate muscle calcium uptake and direct intracellular calcium into the muscle cells (Massheimer, Fernandez, Boland, & de Boland, 1992; Vasquez, de Boland, & Boland, 1997). The regulation of calcium metabolism by vitamin D indicates that vitamin D has a potential modulating role in muscle contractibility (Bartoszewska, Kamboj, & Patel, 2010). In addition, it has been shown in rats that vitamin D<sub>3</sub> activates the p38 mitogen-activated protein pathway (Pardo, Boland, & de Boland, 2006). In humans, the p38 mitogen-activated protein kinase pathway is thought to be one of the main intracellular signaling pathways affecting the development of muscle protein synthesis (Keren, Tamir, & Bengal, 2006). Lastly, there is also a vitamin D receptor in the insulin-like growth factor binding protein 3 (IGFBP-3) promoter region (Peng, Malloy, & Feldman, 2004). It is believed that IGFBP-3 binds insulin-like growth factor 1 (IGF-1) and may influence numerous roles of IGF-1 in muscle cells, including myogenic cell proliferation and differentiation and skeletal muscle hypertrophy (Hamilton, 2010). In a study investigating the growth of children with vitamin-D deficient rickets, Soliman et al., (2008) showed that vitamin D may influence bone growth through its relationship with IGF-1. Six-months after receiving one intramuscular injection of vitamin D<sub>3</sub> mega-dose (300000 IU), IGF-1 concentrations significantly increased ( $26.6 \pm 12.8$  ng/mL before treatment vs.  $52.2 \pm 18.9$  ng/mL after treatment;  $P < 0.05$ ), and the growth rate of the children was correlated with the IGF-1 and vitamin D concentrations ( $r = 0.325$  and  $r = 0.314$ ;  $P < 0.01$ ). Together, these findings suggest that maintaining an adequate vitamin D status may improve strength, power, and speed performance by facilitating the genomic and non-genomic actions of vitamin D in skeletal muscle.

**Cardiorespiratory fitness.** Several studies have shown vitamin D concentrations to be positively associated with cardiorespiratory fitness. Al Mheid et al., (2009) examined vitamin D

concentrations in 163 healthy white males and females (age  $44 \pm 13$  years, BMI  $22 \pm 3$  kg/m<sup>2</sup>). VO<sub>2</sub> max and anaerobic threshold were measured through a cardiopulmonary exercise test. The study concluded that individuals with vitamin D concentrations > 30 ng/mL had a higher VO<sub>2</sub> max ( $r = 0.35$ ,  $P < 0.01$ ) and a higher anaerobic threshold ( $r = 0.37$ ,  $P < 0.01$ ). Similarly, Mowry et al., (2009) compared vitamin D concentrations and VO<sub>2</sub> max among 59 overweight females (age  $19.86 \pm 2.13$ , BMI  $36.16 \pm 8.19$  kg/m<sup>2</sup>). The study reported a significant association between VO<sub>2</sub> max and vitamin D concentrations ( $r = 0.36$ ;  $P < 0.05$ ). Most recently, Ardestani et al., (2011) examined vitamin D concentrations in relation to VO<sub>2</sub> max in 200 healthy adults (age  $40 \pm 14.4$  years, BMI  $26 \pm 5.1$  kg/m<sup>2</sup>). This study found that vitamin D concentrations were also significantly associated with VO<sub>2</sub> max ( $r = 0.29$ ,  $P < 0.001$ ). This study also compared VO<sub>2</sub> max levels among individuals who were vitamin D sufficient (serum 25(OH)D concentrations > 30 ng/mL), insufficient (serum 25(OH)D concentrations = 20-30 ng/mL), and deficient (serum 25(OH)D concentrations  $\leq 20$  ng/mL). After adjusting for age, gender, body mass index, and time spent exercising, the researchers reported higher VO<sub>2</sub> max values among those who were vitamin D sufficient (36.2 ml/kg/min) than those who were insufficient (33.5 ml/kg/min) or deficient (29.3 ml/kg/min). Although these studies are correlative, they suggest that athletes have better cardiorespiratory endurance when they are able to maintain an adequate vitamin D status.

There have also been studies that have examined the relationship between vitamin D and cardiorespiratory fitness in both athletes and non-athletes by tracking seasonal changes in cardiorespiratory fitness. In one study, Erikssen & Rodahl (1979) examined seasonal variation in fitness among 1,835 sedentary, but healthy Norwegian men (age 40-59) using a near maximal bicycle test. The test was performed during three separate periods (period 1 = January – April, Period 2 = May – August, Period 3 = September – December). The study found that maximum

work performance ability peaked late in August and started to decline in September. Aerobic capacity, measured by heart rates at the end of a fixed workload, was significantly higher during the months of May – August than during the two other periods ( $P < 0.001$ ). The researchers did not measure vitamin D concentrations in between the three periods leaving one to speculate as to why these results were seasonal. In another study of non-athletes, Kristal-Boneh et al., (2000) compared pairs of 24-hour heart rate variability data measured once in the summer and once in the winter of 120 healthy men in Israel. Even when adjusting for age, serum level of cholesterol, systolic blood pressure and body mass index, heart-rate variability was significantly lower in the winter, suggesting that cardiorespiratory fitness may be higher in the sunnier summer season than during the winter. Svedenhag & Sjodin (1985) measured the seasonal variation in physiological characteristics of ten members of the Swedish national track and field team who trained year-around. The study included five middle distance (age 21 years) and 5 long distance runners (age 23 years). The athletes completed a maximum treadmill test on 4 separate occasions over a period of one year (Test 1 = January; Test 2 = May; Test 3 = summer period; Test 4 = the following January). Although it is not clear whether the researchers controlled for time spent exercising in addition to the treadmill test,  $VO_2$  max was significantly higher during the summer than in the winter (74.2 vs. 77.4 ml/kg/min;  $P < 0.01$ ).

These three studies all show cardiorespiratory fitness peaking in the summer months. Since vitamin D concentrations have also been shown tend to peak at this same time, the studies point to vitamin D and its capability of influencing cardiorespiratory fitness (Hyppönen & Power, 2007). It is important to note, however, that not all of these studies controlled for time spent exercising, and since people tend to report exercising more in the summer months when there is warmer temperatures and more hours of daylight than the winter months, it is not clear

which is responsible for the improvements in cardiorespiratory fitness—the sunnier, summer weather or increased time spent exercising (Ardestani et al., 2011; Carson, V., & Spence, 2010; Pivarnik, Reeves, & Rafferty, 2003). Also, vitamin D concentrations were not measured in any of these studies. Rather, it was just assumed that concentrations were higher in the summer months.

There have been other studies conducted in Germany that have linked vitamin D to improved cardiorespiratory fitness by using ultraviolet irradiation on its participants. These studies are written in German and are not accessible through scholarly databases in the United States. However, Cannell et al. (2009), in their review, interpret the literature and provide a brief description of these studies. One of these intervention studies in particular is the only known study to have examined the effects of vitamin D supplementation on physical performance. The non-irradiated control group, whose cardiorespiratory fitness was 56% less than that of the irradiated treatment group, was given 250,000 IU of vitamin D as a single dose. One month later, their cardiorespiratory fitness reached that of the irradiated group. Although it is not clear whether the researcher measured vitamin D concentrations in the participants, the results suggest that vitamin D supplementation may, indeed, positively influence cardiorespiratory fitness (Cannell, 2009).

There are several mechanisms thought to be responsible for the association between vitamin D and cardiorespiratory fitness. For one, vascular endothelial cells contain both vitamin D receptors and 1- $\alpha$ -hydroxylase—the enzyme needed to activate these receptors to its hormonal form (Mheid et al., 2011). It has been shown that individuals with low vitamin D concentrations have abnormalities in several different indices of vascular function, namely brachial artery flow mediated dilation and reactive hyperemia (Mheid et al., 2011). Secondly, the renin-angiotensin-

aldosterone system is inappropriately activated in individuals with low vitamin D concentrations, which increases susceptibility to pulmonary hypertension and may result in left ventricle hypertrophy (Li et al., 2002; Mheid et al, 2011; Mitsuhashi, Morris Jr., & Ives, 1991). Based on this correlative data, it is speculated that the negative effects that a compromised vitamin D status has on both vascular health and cardiac hypertrophy may decrease VO<sub>2</sub> max (Ardestani et al., 2011).

In addition, since vitamin D<sub>3</sub> has been shown to activate the p38 mitogen-activated protein kinase pathway in rats, it is possible that adequate vitamin D concentrations may help influence some of the adaptations that occur in skeletal muscle in response to aerobic exercise, namely increases in mitochondrial mass, muscle oxidative capacity and sensitivity of muscle glucose uptake to insulin (Aronson et al., 1997; Pardo, Boland, & de Boland, 2006). However, this is mere speculation and has not been shown in research.

**Reaction time and coordination.** Reaction time—defined as the total elapsed time from an external stimulus (start gun, ball, opponent, etc.) to the initiation of the reaction movement—is a critical component of optimal athletic performance in many sports (Clark, Lucett, & National Academy of Sports Medicine, 2009).

There have only been two studies—both in non-athletes—that have examined the relationship between vitamin D concentrations and reaction time, both of which have shown there to be a positive association. One study to examine the relationship between vitamin D and reaction time was done in Germany in the 1950s. Because the study is written in German and not accessible through scholarly databases in the United States, the exact methodology of this study is unclear. Only one known vitamin D researcher has knowingly looked at and interpreted this

German study. He reported that the study included children and an unknown number of adults during October and November. Reaction time was measured by time needed to recognize a light signal and turn it off. After controlling for practice effects, the researchers administered nine-full body UV treatments (excluding the neck and face) over three weeks to the treatment group while the control group received placebo radiation. The study found that reaction time improved by 25% in children and 20% in adults in the treatment group. In the control group, reaction times declined from baseline tests. In the treatment group, the improvements were greatest at the end of the 3-week study period then returned to baseline 3-weeks later. The study also noted that faster reaction times were seen in the sunnier months (Cannell et al., 2009).

In the second study to examine the effects of vitamin D supplementation on neuromuscular function, 139 ambulatory older adults (age  $\geq 65$  years) with a vitamin D concentrations  $\leq 12$  ng/mL were randomized to receive a single intramuscular injection of 60,000 IU of vitamin D3 or a placebo. Choice reaction time was measured using an automated computer program that determined how quickly an individual responded to a four-choice item. As anticipated, the intervention group receiving the intramuscular injection of vitamin D3 saw a significant increase in vitamin D concentrations compared to the placebo group (+ 6.9 vs. + 2.7 ng/mL;  $P < 0.001$ ) 6-months after the injection. After controlling for practice effects, researchers found that the treatment group also saw a significant reduction in reaction time compared to the placebo group (-0.41 vs. + 0.06 seconds;  $P = 0.01$ ) (Dhesi et al., 2006).

Together, these studies suggest that supplementing with vitamin D may improve reaction time in some individuals. While there is not nearly enough evidence to verify this claim, the large amounts of mechanistic evidence linking vitamin D to brain development and function may support its merit. For one, every cell in the brain contains vitamin D receptors as well as  $1,\alpha$ -

hydroxylase, which is the enzyme needed to activate these receptors (McCann & Ames, 2008). These vitamin D receptors have also been identified in the nuclei of different cell types in the central nervous system, which largely dictates the speed of cognitive processing (Haywood & Getchell, 2009; McCann & Ames, 2008). Moreover, by altering the expression of multiple genes and proteins in the brain, vitamin D is thought to influence brain development (Harms, Burne, Eyles, & McGrath, 2011; McCann & Ames, 2008). In fact, studies conducted in rats restricted for UV light and dietary vitamin D have indicated that vitamin D deficiency during prenatal and early life leads to the dysregulation of several signaling pathways, resulting in abnormal brain development and disease (Almeras et al., 2007; Eyles et al., 2007). In addition, vitamin D target gene products are involved in many cellular brain functions, all of which influence brain signaling pathways (McCann & Ames, 2008). The role of vitamin D in brain development and function suggest that athletes with an adequate vitamin D status may improve athletic performance by improving nervous system function (Harms et. al, 2011; McCann & Ames, 2008).

**Body composition.** Regardless of the type of sport or specific position played, having a particular body fat to muscle mass ratio is a critical component to the success of athletes. Collectively, these two factors may affect an athlete's speed, endurance, power, strength and agility (American College of Sports Medicine, American Dietetic Association, & Dietitians of Canada, 2009). Thus, it is meaningful to explore the effects of vitamin D status on body composition.

Several studies have shown that insufficient vitamin D concentrations are associated with an unfavorable body composition in otherwise healthy individuals. For instance, one study

examined the relationship between vitamin D concentrations and anthropometric measures using both dual energy x-ray absorptiometry (DEXA) and computed tomography (CT) in 90 healthy females (aged 16-22) and reported that women with sufficient vitamin D concentrations ( $\geq 30$  ng/mL) had a significantly lower body weight ( $63.9 \pm 11.9$  vs.  $71.3 \pm 20.0$  kg;  $P = 0.046$ ) and body mass index ( $23.7 \pm 4.6$  vs.  $27.1 \pm 7.1$  kg/m<sup>2</sup>;  $P = 0.014$ ) than women with insufficient vitamin D concentrations ( $\leq 29$  ng/mL). In addition, the women with sufficient vitamin D concentrations had significantly less subcutaneous fat ( $203 \pm 98.9$  vs.  $288.1 \pm 174.0$  cm<sup>2</sup>;  $P = 0.029$ ) and visceral fat ( $24.74 \pm 33.8$  vs.  $44.81 \pm 46.83$  cm<sup>2</sup>;  $P = 0.009$ ) (Kremer et al., 2009). Arunabh et al., (2003) reported similar results in a study that examined the correlation between vitamin D concentrations and body composition in 410 healthy women (age 20-80 years). After adjusting for race, season, age and dietary vitamin D intake, this study concluded that vitamin D concentrations were inversely correlated with percent body fat ( $r = -0.13$ ;  $P = 0.013$ ). In the only known study to compare vitamin D status to body composition in athletes, Halliday et al. (2011) reported that vitamin D concentrations were not significantly associated with body weight or body mass index. However, there was a tendency for vitamin D concentrations to correlate negatively with body fat percentages in the fall and spring. The authors suggested that the lack of a stronger correlation could have been attributed to the narrow range of body fat percentages in the population of athletes.

In addition, studies in less-healthy individuals have shown that higher vitamin D concentrations are independently associated with successful weight-loss and fat loss. In a 2-year study of 322 adults, for example, Shahar et al. (2010) found that higher tertiles of serum vitamin D concentrations were related to greater diet-induced weight loss. For example, a median serum vitamin D concentration tertile of 14.5 ng/mL was associated with a loss of 3.1 kg, whereas a

median vitamin D concentration tertile of 30.2 ng/mL was associated with a loss of 5.3 kg. Similarly, Sibley et al., (2009) measured vitamin D concentrations in 38 overweight men and women before beginning an 11-week restricted calorie diet. This study found that higher pre-diet vitamin D concentrations were associated with greater overall weight loss—for every increase of 1 ng/mL of vitamin D, the participants lost 0.196 kg more at the conclusion of the study. In addition, higher pre-diet vitamin D concentrations were associated with a greater loss of subcutaneous abdominal fat. Supplementing with 100 IU of vitamin D and 250 mg of calcium/day for 16-weeks has also been associated with a decrease in absolute body weight ( $-2.5 \pm 3.3$  kg) and a significant reduction in visceral adipose tissue ( $-12.9 \pm 21.8$  cm<sup>2</sup>;  $P = 0.007$ ) in 171 overweight and obese men and women. However, these participants were on diets ~500 kcals less per day than their calorie maintenance levels. Therefore, the participants were likely losing weight and body fat due to their decreased caloric intake—not because of their supplementation with vitamin D and calcium.

On the other hand, it is also possible that vitamin D may help individuals to maintain weight. Caan et al., (2007) showed that postmenopausal women supplementing with 400 IU/day of vitamin D and 1,000 mg of calcium for an average of 7 years lost an average of 0.13 kg more than women who had consumed a placebo throughout this same time period. The women supplementing with vitamin D and calcium were also less likely to gain weight, and they were more likely to maintain their baseline weight within  $\pm 1$  kg.

Altogether, these studies suggest that adiposity should be taken into account when assessing vitamin D requirements in athletes and that attaining an adequate vitamin D status may assist athletes in achieving their ideal body composition based on the type of sport and specific position played. In order to clarify these claims, however, there will need to be more studies that

examine the relationship between vitamin D concentrations and body composition in athletes exclusively.

There are several possible explanations to why vitamin D may influence a favorable body composition in athletes. For one, vitamin D is stored in adipose tissue and contains allelic variations of the vitamin D receptor gene that have been shown to be associated with body type (Grunberg et al., 2004; Keen et al., 1997; Suarez, Zeghoun, Rossignol, Walrant, & Garabedian, 1997). Previous studies assessing this association have drawn similar conclusions, stating that individuals with the BsmI polymorphism BB allele have higher body weight and fat mass than those with the BsmI polymorphism bb allele (Grunberg et al., 2004; Keen et al., 1997; Suarez, Zeghoun, Rossignol, Walrant, & Garabedian, 1997). Also, it is thought that genotypes may be influenced by the environment in which people are exposed to early in life, and that this may continue to influence body type characteristics all the way through adulthood (Keen et al., 1997). Although the physiological mechanism for the association between vitamin D receptor genotype and body composition is unknown, the influence that vitamin D has on adipocyte differentiation and metabolism may serve as a possible explanation (Grunberg et al., 2004; Kamei et al., 1993).

Vitamin D may also influence body type through its relation with leptin—an adipocyte hormone that regulates energy intake and expenditure (Enriori, Evans, Sinnayah, Cowley, 2006; Zhang, Chen, Heiman, & Dimarchi, 2005). High amounts of adiposity impair the leptin signals that regulate energy homeostasis, so overweight and obese individuals tend to have relatively high leptin concentrations (Enriori et al., 2006; Zhang et al., 2005). Menendez et al., (2001) is the only known study to suggest an association between vitamin D and leptin. It found that that when retinoic acid and vitamin D3 was administered to human adipose tissue culture, there was a decrease in leptin synthesis. Although significant evidence of the effect of vitamin D on leptin is

scarce at this point, the results from Menendez et al., (2011) suggest that athletes with adequate vitamin D concentrations may be able to maintain a more favorable body composition than those with insufficient vitamin D concentrations by promoting energy homeostasis.

Vitamin D may also influence adiposity through its regulation of serum parathyroid hormone levels. For instance, when an individual is deficient in vitamin D, there is a compensatory increase in serum parathyroid hormone levels (Souberbielle et al., 2003). There is evidence that excessive secretion of parathyroid hormone stimulates calcium influx into the adipocyte cells. This process increases the production of fatty acid synthase, which, consequently, stimulates lipogenesis, inhibits lipolysis and increases triglyceride stores (Zemel, Shi, Greer, Dirienzo, & Zemel, P., 2000; Zemel, 2002). This suggests that the regulation of the parathyroid hormone by vitamin D may be the primary mechanism responsible for the association between vitamin D status and insulin resistance—a condition that inevitably results in weight gain and obesity (Teegarden & Donkin, 2009). These propositions suggest that suppressing parathyroid hormone levels by attaining an adequate vitamin D status may reduce adiposity in non-athletes and athletes alike and help them to achieve their ideal body composition.

## **Chapter Three: Conclusions and Recommendations**

### **Conclusions**

Several studies have suggested a positive association between maintaining an adequate vitamin D status and factors that may positively influence athletic performance, namely strength, power, speed, cardiorespiratory fitness, reaction time and coordination, and body composition. In addition, there is evidence to suggest that vitamin D is related to reductions in stress fractures, common illnesses, muscle injuries and chronic pain—all of which may also affect athletic training and performance. Perhaps the most convincing explanation for these associations is the recognition of vitamin D receptors in nearly every tissue and cell in the human body—all of which may play a crucial role in athletic performance. The evolving understanding of the genomic and non-genomic roles of vitamin D receptor gene polymorphisms also provide notable support to the claim that maintaining an adequate vitamin D status will result in improved athletic performance. At this point, however, it cannot be proven for certain that vitamin D enhances athletic performance. Most of the studies that have examined the effect of vitamin D on various athletic performance indicators have been correlational, allowing for the possibility that other lifestyle, dietary and genetic factors may also be playing a role in elevating vitamin D concentrations and, subsequently, improving athletic performance. Direct interventional studies measuring vitamin D concentrations and athletic performance indicators are needed to substantiate this claim

## **Recommendations**

There are several gaps in the literature that must be addressed in order to both verify the relation between vitamin D and athletic performance and make appropriate vitamin D supplementation recommendations. First, studies must compare vitamin D concentrations among both recreational and elite athletes in multiple sports in order to better predict which types of athletes are most susceptible to vitamin D deficiencies or insufficiencies, and to determine whether athletes require more vitamin D than the general population.

Also, there are no known studies that have tracked athletic performance indicators, namely speed, strength, power, cardiorespiratory fitness, reaction time, and body composition, throughout a vitamin D supplementation protocol. Randomized, double-blind, placebo controlled, and dose-response studies in athletes are needed to determine whether supplementing with vitamin D directly influences these athletic performance indicators. These studies should also examine the vitamin D concentration at which athletes experience the greatest improvements in athletic performance indicators, and determine whether this concentration is applicable to all athletes, or if optimal vitamin D concentrations for athletic performance-related benefits should be assessed on an individual basis.

In addition, the effects of periodizing vitamin D dosage between single high-dose and low-dose supplementation periods on both athletic performance indicators and the health of athletes have not been investigated. A controlled study that periodizes vitamin D supplementation throughout the training year may help to determine whether a single high-dose supplementation period could benefit athletes during times of increased training or competition.

Despite the limited research examining the relation between vitamin D and athletic performance, studies suggest that a large percentage of athletes may be insufficient or deficient

in vitamin D. These deficiencies seem to be most apparent in the winter and spring regardless of geographic location or whether the athlete competes indoors or outdoors. These findings suggest that all athletes should attempt to attain a sufficient vitamin D status through sunlight, artificial ultraviolet B light and/or supplementation, especially in the winter and spring months. Dark-skinned athletes and athletes with a high percentage of body fat seemingly face the greatest risk of vitamin D deficiencies and should attempt to increase their vitamin D intake accordingly. It is important to note that ideal vitamin D concentrations for both performance and health related benefits are unknown. Despite this, a prudent recommendation would be for athletes to attain adequate (>20 ng/ml) vitamin D concentrations.

For athletes choosing to increase vitamin D concentrations through sunlight, it is thought that 3-8 minutes of ¼ body surface area exposure to summer-noon day sun will produce vitamin D<sub>3</sub> concentrations equivalent to 400 IU of orally supplemented vitamin D in most light-skinned individuals (Terushkin, 2010). There is evidence that following this ultraviolet-B exposure guideline ~3 times a week may help to maintain vitamin D concentrations (> 20 ng/mL) year-round (Webb et al., 2010). Athletes with darker skin may take 6 times as long to obtain the same amount of previtamin D<sub>3</sub> (Clemens, Adams, Henderson, & Holick, 1982). In addition, overweight individuals and those who wear sunscreen may also require extra ultraviolet-B exposure time to achieve a similar response (Webb, 2006; Wortsman et al., 2000). There are no known cases of vitamin D toxicity from prolonged sun exposure, which can be attributed to various protective mechanisms of the skin preventing the body from synthesizing too much vitamin D (Hathcock, Shao, Vieth, & Heaney et al., 2007; Webb, DeCosta & Holick, 1989).

Frequent tanning bed use has also been shown to be effective in increasing vitamin D concentrations (Holick, Chen, Lu, & Sauter, 2007; Tangpricha et al., 2004). While there are no

established guidelines for treating vitamin D deficiency with tanning bed use, it has been shown in a primarily vitamin D deficient population (serum 25(OH)D  $\leq$  20ng/mL) that previtamin D<sub>3</sub> production increased at a linear rate of 1% per minute spent in a tanning bed, and that whole body exposure to tanning bed radiation three times a week for an equivalent of 0.75 MED increased vitamin D concentrations by 50% above baseline concentrations in one week, and 150% above baseline concentrations after five weeks (Holick et al., 2007). In addition, it has been shown that individuals who used a tanning bed once a week during the winter season were able to maintain vitamin D concentrations  $>$  44 ng/mL, which is 90% higher than control subjects who had not used a tanning bed during this same time period (Tangpricha et al., 2004).

Given the health risks associated with overexposure to ultraviolet-B radiation, these recommendations should be taken with vigilance. Athletes choosing to increase vitamin D concentrations through both sunlight and/or artificial ultraviolet-B radiation must contemplate whether the potential benefits of attaining an adequate vitamin D status outweigh the risk of skin cancer that may accompany ultraviolet-B exposure (Kennedy, Bajdik, Willemze, De Gruijl, & Bouwes Bavinck, 2003).

For athletes who tend to avoid the sun and/or do not like the idea of artificial ultraviolet-B exposure, oral supplementation with vitamin D<sub>3</sub> may be a safe and effective alternative as long as the appropriate doses are taken (Autier & Gandini, 2007). Oral vitamin D<sub>3</sub> supplements are available in nonprescription and prescription pills containing 400, 1,000, 2,000, 5,000, 10,000, and 50,000 IUs (Cannell & Hollis, 2008). The amount of vitamin D needed to attain an adequate vitamin D status will vary from athlete to athlete depending on numerous factors, including baseline concentrations of vitamin D, geographic location, season, body composition, and lifestyle habits. It is estimated that for individuals with a baseline vitamin D concentration  $\geq$  28

ng/mL, the average increase in vitamin D is 0.28 ng/mL for every daily vitamin D3 dose of 40 IU (Heaney, Davies, Chen, Holick, & Barger-Lux, 2003). Perhaps the most accurate way to determine how much supplemental vitamin D3 an athlete needs is to have their vitamin D concentrations measured at different seasons of the year. The athlete may then experiment orally supplementing with the Institute of Medicine's recommendation of 600 IUs/day to see if they are able maintain adequate levels throughout the year. If this dose is not effective, the athlete may consider consuming the vitamin D3 supplementation recommendations of several in the field, which varies from 800-5,000 IU/day. When doing so, the athlete must decide what they consider to be an adequate vitamin D concentration —the > 20 ng/mL established by the Institute of Medicine, or the 30-60 ng/mL established by others in the field. If an athlete is not able to get their vitamin D concentrations measured, they must base their dietary and supplemental dosages in regards to where they live, the season, their skin color, body composition and lifestyle habits. Despite the 4,000 IU/day tolerable upper limit established by the Institute of Medicine, and a suggested 10,000 IU/day by others, numerous studies have indicated that continuous short-term supplementation with doses between 10,000 and 50,000 IU/day did not result in vitamin D toxicity (Hathcock et al., 2007; Heaney et al., 2003; Heaney, 2005; Malabanan, Veronikis, & Holick, 1998; The Institute of Medicine, 2010; Vieth et al., 2007). However, although these high-dosages were effective in raising vitamin D concentrations acutely, long-term health consequences and vitamin D toxicity have not been sufficiently investigated under these conditions, which suggests that the risk-benefit of high-dose supplementation may not be more effective than continuous low-dose supplementation. Therefore, a continuous low-dose supplementation protocol may be the safest and most effective choice until the Institute of

Medicine and others can reach a consensus on both adequate vitamin D concentrations and a recommended supplemental dose of vitamin D.

## Appendix A: A Summary of Vitamin D Recommendations for Athletes

- All athletes should attempt to attain vitamin D concentrations >20 ng/ml through sunlight, artificial ultraviolet-B light, or supplements
- Sunlight and artificial ultraviolet-B light
  - 3-8 minutes of casual exposure to summer-noon day sun ~3x/week may help to maintain vitamin D concentrations > 20 ng/mL year-round
    - Athletes with darker skin may take 6x as long to obtain the same amount of previtamin D3
    - Overweight individuals and those who wear sunscreen may also require extra ultraviolet-B exposure time to achieve a similar response
  - Frequent tanning bed use has also been shown to be effective in increasing vitamin D concentrations
- Vitamin D supplementation
  - Encourage athletes get their vitamin D concentrations measured at different seasons of the year
  - Have the athletes supplement with 600 IUs/day to see if they are able maintain vitamin D concentrations >20 ng/ml throughout the year
  - If this dose is not effective, the athlete may consider consuming the vitamin D3 supplementation recommendations of several researchers in the field, which varies from 800-5,000 IU/day
  - If an athlete is not able to get their vitamin D concentrations measured, they must base their dietary and supplemental dosages in regards to where they live, the season, their skin color, body composition and lifestyle habits

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