A CROSS-SECTION ANALYSIS OF VITAMIN D STATUS ON MUSCLE STRENGTH AND POWER IN NCAA ATHLETES

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Major Field: HEALTH AND HUMAN PERFORMANCE

Abstract:

Purpose: The purpose of this study was to assess the prevalence of vitamin D inadequacy in collegiate athletes and to determine the influence of serum vitamin D (25-OH D) on muscle strength and power in this population.
Methods: Demographic and anthropometric data were collected on collegiate athletes (n=103) from three separate NCAA athletic programs. Dietary vitamin D and calcium intake, and sun exposure were assessed using validated questionnaires. Serum 25-OH D was evaluated and a series of physical performance measures that are established indicators of muscular strength and power were completed. The performance measures included the Vertical Jump Test, Shuttle Run Test, Triple Hop for Distance Test and the 1 Repetition Maximum (1 RM) Squat Test. Categorical data were evaluated using Chi Square and Pearson Correlations were performed to examine the relationship between serum vitamin D and performance measures. Comparisons between groups were accomplished using ANCOVA with lean body mass as a covariate.
Results: Using serum 25-OH D concentrations of 30 ng/mL and 20 ng/mL as the cutoff points for insufficient and deficient status, 68% of the study participants were considered vitamin D adequate, while 22.7% were insufficient and 8.9% were deficient. Analyses using ANCOVA revealed a lower ($p < 0.05$) 1 RM Squat Test when adjusting for lean body mass with vitamin D deficiency. However, no other performance measures were significantly different.
Discussion: These findings suggest that the majority of this population of collegiate athletes were vitamin D sufficient. Athletes with adequate vitamin D status may have an ergogenic advantage in terms of the 1 RM Squat compared to those who are vitamin D inadequate. Further research is needed to determine the potential implications of these findings on athletic performance and studies are warranted to determine if vitamin D supplementation can improve performance outcomes.
Key Words: VITAMIN D, 25-OH D, MUSCLE STRENGTH, MUSCLE POWER, ATHLETES

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CHAPTER I

INTRODUCTION

Current estimates indicate that approximately 1 billion people worldwide suffer from suboptimal serum vitamin D levels (i.e., vitamin D insufficiency or deficiency)[1]. Inadequate vitamin D status, defined as serum 25-hydroxyvitamin D (25-OH D) < 30 ng/mL, is known to alter calcium homeostasis and have detrimental effects on bone health. The increasing incidence of vitamin D insufficiency and deficiency has become a growing concern among many allied healthcare professionals in the U.S., especially as the scientific literature has indicated that vitamin D is also associated with a variety of chronic diseases (e.g., diabetes mellitus, certain cancers, and depression) and impaired physical function[2-4].

In addition to influencing chronic disease and physical capacity, vitamin D status can, in itself, be affected by factors such as the age, ethnicity, exposure to the sun, dietary intake and body composition of an individual[2]. It is clear that as an individual ages, the ability to synthesize cutaneous vitamin D decreases, leading to a greater prevalence of vitamin D inadequacy among older adults[5]. Ethnicity also contributes to vitamin D deficiency[6]. For example, in a sample of Caucasian 9-11 year olds, 48% were vitamin D inadequate, while 57% of healthy Caucasian older adults were reported to be inadequate[3,5]. Among a sample of healthy older African American adults, the prevalence of vitamin D inadequacy was as high as 84%[3].

The average adult synthesizes most of their daily vitamin D requirement subcutaneously from exposure to ultraviolet rays[4]. However, exposure to sun is limited for some populations due to seasonal changes in the sun's zenith angle or latitude of residence, leaving the diet to serve as an alternative source of vitamin D[6]. According to the Institute of Medicine's 2010 recommendations, the daily recommended intake of vitamin D for people ages 9-70 years is 600 International Units (IU)/day[7]. Vitamin D, naturally found in foods such as oily fish and shiitake mushrooms, is also fortified into foods such as dairy products and certain grains in the United States[8,9]. Although most adults meet their daily macronutrient needs[10], many individuals have inadequate intake of several micronutrients due to unhealthy food choices[10,11]. Among college-aged students in particular, study habits, social interaction, cost, convenience, and belief all influence dietary intake[10-13]. The limited dietary sources of vitamin D (i.e. natural and fortified) are often expensive and may be cost prohibitive for many students. Additionally, vitamin D can be sequestered within the adipose tissue[14] and so body composition and distribution of body fat can play a role in the vitamin D status of an individual[14]. Therefore, individuals with excessive subcutaneous adiposity are at greater risk of having inadequate serum levels of vitamin D. These factors may contribute to the increase in vitamin D insufficiency observed in the young adult population.

In addition to vitamin D's role in bone health and disease prevention, accumulating scientific evidence demonstrates a strong relationship between serum 25-OH D and muscle strength and muscle power[1,8,15-18]. Populations studied vary based on factors that influence vitamin D status such as age, ethnicity, latitude of residence and occupation[17,19-26]. Studies have shown older adult, young adult and adolescent populations experienced significant improvements in lower extremity muscle function when serum vitamin D status was sufficient (i.e., > 30 ng/mL)[1,13,19-22]. In a cross section of older adults, the positive correlation between vitamin D status and muscle strength was demonstrated by a decrease in the incidence of falls as well as an increase in grip strength in those whose vitamin D status improved from inadequate to adequate[25]. Importantly, the greatest strength gains were seen in individuals who progressed from deficient to sufficient, but muscle strength and power gains

continued to be positively correlated even for those individuals who were considered vitamin D adequate[25,26]. In a cross-sectional study of children and adolescents, muscular strength and muscular power, as indicated by vertical jump or squat jump score, were positively correlated with vitamin D status[17]. Also, young adults with suboptimal serum concentrations of vitamin D (< 30ng/mL), who were recovering from an anterior cruciate ligament (ACL) reconstruction, were slower to obtain pre-surgical strength than young adults with the same surgical procedure who were vitamin D sufficient[27].

A major role of vitamin D in maintaining muscle function during physical activity is regulating the availability of the micronutrients, calcium and phosphorus [26,28]. Vitamin D aids in the absorption and transport of calcium and phosphorus to the muscles[28]. The mechanisms by which vitamin D improves muscle function have been associated with: 1) effective influx and uptake of calcium, 2) increased phosphate metabolism, and 3) enhanced muscle cell proliferation and differentiation[28-32]. The effect of 1α,25-OH_2 D, the biologically active form of vitamin D, on calcium is attributed primarily to the interaction with the plasma membrane vitamin D receptor (mVDR)[28,32]. This interaction is non-genomic, (i.e., outside the nucleus), and involves the rapid mobilization of calcium from the sarcoplasmic reticulum, stored calcium within the cytoplasm and extracellular calcium that is regulated by the voltage dependent calcium channels[16,28,32]. Vitamin D mediated phosphate metabolism is also non-genomic with vitamin D regulating the absorption and availability of free phosphate and adenosine tri-phosphate (ATP) utilized in muscle contractions[28]. In contrast, vitamin D also has genomic effects on muscle cell proliferation and differentiation that are initiated by the interaction of vitamin D and the VDR which in turn elicits a cascade of events that lead to myogenesis[28].

Despite continued efforts to understand the mechanism by which vitamin D status affects muscle function, and the supporting evidence that vitamin D is positively correlated with muscular strength and muscular power in certain populations, there is limited data examining the relationship between muscle function and vitamin D status for the purposes of improving muscular strength and muscular power in the collegiate athlete[28,33,34]. Previously, athletes, ranging from junior high to elite

competitors, have been shown to have a high prevalence of vitamin D insufficiency[33-39]. Lovell[38] reported that 100% of the female elite gymnasts they studied had serum concentrations of vitamin D in the suboptimal range, while Lehtonon-Veromaa and colleagues[36] reported that 68% of adolescent runners and gymnasts were vitamin D insufficient. In a recent meta-analysis examining the prevalence of vitamin D insufficiency, the authors found consistency in the research that few collegiate athletes met the dietary intake of 600 IU per day[34]. Willis, et al[37], estimated that elite athletes only consume ~330 IU of vitamin D per day, rendering them vitamin D insufficient if sunlight exposure is limited. However, the question remains as to the extent to which an athlete's serum vitamin D is related to muscular strength and muscular power, which are measures of athletic performance.

Formal Statement of the Problem

Studies have shown a positive correlation between serum 25-OH D and muscular strength and muscular power in non-athletic populations. Moreover, with improved 25-OH D status, muscle strength and power were increased in these populations. However, few studies have examined the correlation of vitamin D status to muscle strength and muscle power in collegiate athletes.

Purpose of the Study

The purpose of this study is to assess the prevalence of vitamin D inadequacy in collegiate athletes and to determine the influence of vitamin D status on measures of muscular strength and muscular power in highly trained NCAA collegiate athletes.

Significance of the Study

The aim of this research is to determine the relationship of vitamin D status on muscle strength and muscle power by evaluating performance measures related to lower body strength and power. Athletes often search for ergogenic aids that can provide them with a competitive edge[40]. By better understanding the relationship between vitamin D status and muscle strength and muscle power in the collegiate athletic population, investigators will be able to determine if vitamin D supplementation studies are warranted.

Null Hypotheses

The primary null hypotheses tested are:

1) There will be no participants who are vitamin D deficient/insufficient in cross-section of collegiate athletes.
2) Collegiate athletes with sufficient vitamin D status will not have higher Vertical Jump Test scores than those who are insufficient or deficient.
3) Collegiate athletes with sufficient vitamin D status will not have lower Shuttle Run Test times than those who are insufficient or deficient.
4) Collegiate athletes with sufficient vitamin D status will not have longer Triple Hop for Distance Test scores than those who are insufficient or deficient.
5) Collegiate athletes with sufficient vitamin D status will not have higher 1 Repetition Maximum (1 RM) Squat scores than those who are insufficient or deficient.
6) Collegiate athletes with sufficient vitamin D status will not have greater muscle power output scores based on the Vertical Jump Test than those who are insufficient or deficient.
7) Collegiate athletes with sufficient vitamin D status will not have greater muscle force output scores based on the Vertical Jump Test than those who are insufficient or deficient.

The ancillary null hypotheses tested were:

1) There will be no difference in vitamin D status based on subject's vitamin D intake in collegiate athletes.
2) There will be no difference in vitamin D status based on subjects sport.
3) There will be no difference in vitamin D status based on subject's gender in collegiate athletes.
4) There will be no difference in vitamin D status based on subject's ethnicity in collegiate athletes.
5) There will be no difference in vitamin D status based on subject's body composition (% fat) in collegiate athletes.

Specific Aims

The specific aims of this study are:

1) To determine the incidence of vitamin D deficiency/insufficiency in a cross section of NCAA collegiate athletes.
2) To explore the relationship between vitamin D status and muscular strength and muscular power by assessing specific functional performance measures, including Vertical Jump Test, Triple Hop for Distance Test, Shuttle Run Test, and 1 Repetition Maximum Squat.
3) To explore the relationship between vitamin D status and muscular strength and muscular power by assessing specific calculated performance measures including power and force.
4) To explore differences in vitamin D status based on vitamin D intake, sport, gender, ethnicity and body composition in NCAA collegiate athletes.

Definition of Terms

1) Vitamin D Status

   Deficient- serum 25-OH D concentration at < 20 ng/mL.

   Insufficient- serum 25-OH D concentration 20- 30 ng/mL.

   Sufficient- serum 25-OH D concentration at > 30 ng/mL.

   Intoxication- serum 25-OH D concentration at ≥ 150 ng/mL; or complaints of signs and symptoms of fatigue, hypercalciuria and hypercalcemia.

2) International Units (IU)- a unit of measure based on the available biological activity; 1 IU vitamin D is equal to 0.025 µg.
3) Muscle Power- the ability to exert a rapid force (power = force*distance/time)[41].
4) Muscle Strength- amount of force a muscle can produce in a single maximal effort (force= power*time/distance)[42].
5) Incremental tests for peak power- increases in speed or workload until the point of exhaustion[41].
6) Iso-inertial tests- a single movement performed explosively against body mass. The measure of distance of height is the measure of power, using the mean of several trials[41].

Assumptions

The assumptions of this study are:

1) Subjects performed at maximal effort for each of the performance test.
2) Subjects responded to the Food Frequency Questionnaire (FFQ) to the best of their ability.
3) Subjects responded to the Calcium Questionnaire (CQ) to the best of their ability.
4) Subjects responded to the Sun Exposure Questionnaire (SEQ) to the best of their ability.
5) Subjects responded to the Health History Questionnaire (HHQ) to the best of their ability.
6) Researchers conducted performance measures accurately.

Limitations

The limitations of the study are:

1) Researchers did not control for vitamin D status of participants.
2) Data collection took place at several times of the year based on availability of subjects and may have biased vitamin D status.
3) Participants may not have responded accurately to questionnaires.
4) Participants may not have performed at their optimal level.

Study Design

This project utilized a cross-sectional study design to evaluate of the relationship between current vitamin D status and athletic performance measures of a subpopulation of NCAA collegiate athletes at The University of Tulsa (TU), Oklahoma State University (OSU) and Southern Nazarene University (SNU). Data such as health history, exogenous vitamin D (i.e., vitamin D from the diet), calcium intake, sun exposure, anthropometric and performance measures were collected to assess the effect vitamin D status has on the collegiate athlete.

CHAPTER II

REVIEW OF LITERATURE

Historical Perspectives of Vitamin D

It is generally accepted that the need for vitamin D arose through evolution[43]. According to researchers in the field[4,43,44], the sea was rich in minerals and provided the early vertebrates with adequate amounts of calcium for essential function. However, as these vertebrates began to migrate onto land, calcium became more sparse in the natural environment so these land dwelling creatures had to develop a new way to meet their daily need[43]. While it is still unknown how the relationship between vitamin D and sunlight evolved[44], it is evident that vitamin D is essential for calcium homeostasis to maintain metabolic function[43]. It was not until society began to industrialize and experience less exposure to sunlight that the physiological requirement for vitamin D became apparent[45].

The Industrial Revolution was a time of change and advancement in Europe and the United States[46]. The push to move toward faster production of goods created jobs that existed inside factories and large warehouses[44]. The resulting decrease in sunlight exposure contributed to an epidemic of vitamin D insufficiency that went unrecognized until the mid-20th century[25,46-50]. As a result of the lack of sunlight, and subsequently the rise of vitamin D insufficiency, rickets became one of the most debilitating diseases in children during this time[51]. The search for a cure for rickets stimulated the exploration of the health benefits of vitamin D[52].

From 1822, when the relationship between sunlight exposure and rickets was first discovered, until 1918 when exposure to sunlamps and administration of cod liver oil was shown to cure rickets, the functions of vitamin D were largely ignored by the public and for the most part, the scientific community[43,46]. It was not until 1921, when researchers exposed several foods to irradiation as a cure for rickets, that vitamin D was considered a link to the disease[43,44,46]. Later, in 1926, Hess and colleagues, through animal studies and the irradiation of butter fat, discovered that cholesterol contained an impurity that proved to have antirachitic properties[53]. However, it was not until 1928, that vitamin D_1 was identified[44]. In the mid-1930s the sources of vitamin D were recognized which resulted in the renaming of vitamin D_1 as vitamin D_2 (i.e., plant sources) and vitamin D_3 (i.e., animal sources)[44,54,55].

In the 1930s, an agreement was made between researchers in England, Germany and the United States, to isolate the cholesterol impurity and determine the sources of vitamin D[53]. Previously discovered in the late 1920s, this impurity had peak ultraviolet absorption between the ultraviolet wavelengths (UV) of 270-320 nanometers (nm)[53]. Windaus and Hess (1931) began their investigation on steroid compounds that had peak UV absorption within this range, leading to the detection and isolation of ergosteriol or vitamin D_2[53]. However, this team of researchers knew that sunlight was the best way to achieve antirachitic doses of vitamin D, and ergosterol was not an animal source[56]. In 1935, Windaus and colleagues, isolated the cholesterol impurity, naming it 7-dehydrocholesterol (7-DHC) but it was not until 1937 that 7-DHC was identified as the precursor to vitamin D_3[53].

Today there has been a resurgence of rickets within society[51]. Less exposure to sunlight and greater vitamin D deficiency has become increasingly recognized by the allied healthcare and medical community as a major public health concern[44,57,58]; however, there is little congruity among medical professionals and researchers as to what constitutes deficient, insufficient, and sufficient concentrations of vitamin D as it relates to the overall health of an individual[59].

Defining Vitamin D Status

The definition of vitamin D sufficiency, insufficiency, deficiency and intoxication has been an area of much debate[47,60]. Serum 25-OH D has been used as the basis for determining vitamin D status[42], but there have been discrepancies in what physiological markers should be examined in determining serum 25-OH D, and therefore the definition of adequate concentrations[4]. Levis[61] and Holick[4], two well-known researchers in the field of vitamin D, have defined vitamin D deficiency as < 20 ng/mL, insufficiency at 21 ng/mL to 29 ng/mL, and sufficiency as ≥ 30 ng/mL, which are based on individual differences in parathyroid hormone (PTH) regulation. The American Medical Association (AMA) has defined deficiency as ≤ 10 ng/mL and insufficiency at 11 ng/mL to 24 ng/mL[48]. In individuals with adequate 25-OH D, generally defined as ≥30 ng/mL, there is no disruption of optimal blood calcium concentrations by dietary calcium absorption or elevation in parathyroid hormone (PTH) regulation[44]. Vitamin D intoxication are not likely to occur until serum concentrations of 25-OH D are >150 ng/mL[4,61,62].

More recently there has been an interest by clinicians in determining optimal vitamin D status on an individual basis, using maximal calcium absorption and serum PTH levels as criteria[2,44,59,62]. Vitamin D insufficiency has a negative effect on calcium homeostasis, specifically leading to clinical secondary hyperparathyroidism, suboptimal calcium absorption, high bone turnover, and if the metabolic state persists, reduced bone mineral density (BMD)[63]. In contrast, vitamin D deficiency leads to clinical secondary hyperparathyroidism, malabsorption of calcium and osteomalacia[44,64], while also exacerbating a number of health conditions, such as diabetes mellitus or osteoarthritis, which is linked to joint aches, muscle pain and overall weakness[33,61,63,65].

Prevalence of Vitamin D Deficiency and Insufficiency

An accumulating body of evidence has demonstrated that vitamin D deficiency and insufficiency is a major public health concern worldwide. The prevalence of vitamin D

insufficiency has been studied extensively on 5 of the 7 continents: Australia, Asia, Europe, Africa, and North America[66]. Australia has the highest occurrence of vitamin D insufficiency (i.e., 80% of the population), while in Asia 60%-70% of the population has inadequate serum 25-OH D[66]. Europe and Africa's populations are both 50%-55% deficient in vitamin D while North America reported that 30%-40% of the population suffered from inadequate vitamin D status[66]. Some have characterized this state of vitamin D insufficiency a pandemic of all ages, including young adults[21,45,67,68], older adults[3,4,44,69], and youth[49,68,70]. Those populations at greatest risk of developing vitamin D insufficiency are individuals with limited sunlight exposure and low dietary intake of vitamin D[67] and older adults in long-term care facilities[44].

In young children and infants, rickets remains a public health problem in most developing countries[71,72] as well as developed countries[51]. Factors that likely contribute to the high incidence of rickets are overcrowding, purduh (or complete covering of skin in Muslim women), a lack of access to sunlight and lack of vitamin D fortification of foods[51,71,72]. Schools in Australia require students to wear additional clothing during their lunch period, such as hats, long sleeves and pants to reduce the amount of sunlight exposure[38]. While decreasing the probability of skin cancers in the future, these practices are likely to increase future long-term fracture risk due to lower peak bone mineral density (BMD) at crucial periods of maturation[38].

Teenagers are another population at risk for vitamin D deficiency[48]. Wagoner and Greer[73], demonstrated a significant increase in PTH and a decrease in 25-OH D occurred as children matured into adolescence. In the U.S., 17% of teenagers in the south that were tested in the winter months and 8% of the teenagers in the north that were tested in the summer months were vitamin D deficient[48]. Just as vitamin D concentrations tended to decrease from childhood into adolescence, the decline continues into adulthood[4]

Several populations of older adults have been the subject of vitamin D research which examined the prevalence of inadequate vitamin D[74,75]. A study, conducted by Holick[4] and colleagues, determined that 50% of postmenopausal women taking osteoporosis medication were

vitamin D deficient. Furthermore, MacDonald, et al[74], found that 40% - 100% of community dwelling older adults were vitamin D deficient. In Europe, free living older adults (n=834) from 16 towns in 11 countries were studied[76]. Thirty-six percent of males and 47% of females experienced vitamin D concentrations below 12 ng/mL[75,76]. In the United States, Meunier, et al[75], found that vitamin D deficiency ranged from 3% to 28% in institutionalized older adults, but Gloth, et al[77], reported as high as 54% of older adults living in long term care facilities were vitamin D deficient. The differences in prevalence of vitamin D deficiency among institutionalized older adults reported in literature can likely be attributed to differences in dietary intake[75].

Signs and Symptoms of Vitamin D Deficiency, Insufficiency and Intoxication

Commonly recognizable signs and symptoms of vitamin D insufficiency include bone frailty or fracture, resulting from an increase osteoclast activity and a decrease in BMD[64]. Chapuy, et al[78], discovered that older ambulatory women experienced a 43% decrease in hip fracture and a 32% reduction in non-vertebral fracture when they were supplemented with calcium and vitamin D, verse those who were not supplemented. However, recent recommendations by the U.S. Preventative Services Task Force have brought the use of calcium and vitamin D supplementation (< 1000 mg calcium and < 400 IU vitamin D_3) as a primary fracture prevention strategy into question[79]. Similarly, Fuleihan, et al[70], studied school aged girls, who had deficient serum concentrations of 25-OH D (6 ng/mL to 22 ng/mL). The investigators discovered a significant correlation between BMD at the spine, femoral neck and radius and vitamin D status[70]. Other signs and symptoms of inadequate vitamin D include chronic kidney disease, generalized bone pain, myalgia, generalized weakness and hypocalcemia[46,80,81]. Ladhani, et al[82], showed that children (n = 17), with radiographic markings of rickets, experienced many of these signs and symptoms. In a study by Glerup, et al[33], veiled Arab women complained of unexplained muscle pain (88%) and deep bone pain (36%) prior to vitamin D supplementation.

However, most of the women reported a decrease in muscle and bone pain after 1 to 1.5 months of 800 IU daily vitamin D supplementation, and the muscle and bone pain continued to decrease at 3 to 6 months post supplementation[33].

Vitamin D is a fat soluble vitamin which means the potential for intoxication exist with mega doses (i.e., > 50,000 IU vitamin D weekly)[4,47]. Intoxication occurs when blood calcium homeostasis has been reached, but serum 25-OH D concentrations continue to increase in response to cutaneous synthesis or dietary intake[8]. With the accumulation of excess vitamin D, 24-hydroxylase is unable to catabolize the metabolites of vitamin D (25-OH D or 1α,25-OH_2 D) at a rapid enough rate[47]. Clinical signs of vitamin D intoxication include: an increase in urine calcium concentration, decrease renal function, and calcification of soft tissues (i.e., kidney, blood vessels, heart and lungs)[4,47]. Hypersensitivity to vitamin D may be a concern in some individuals, such as those with chronic granulomatous, due to macrophage production of 1α, 25-OH_2 D which leads to hypercalciuria and hypercalcemia[4,62]. These persons should not avoid vitamin D consumption or the sunlight, but should be aware of their vitamin D status and maintain their status at the lower end of the normal range (i.e. 30 ng/mL)[62].

Optimal serum concentrations of 25-OH D may differ by individual based on various parameters, such as ethnicity, age and sunlight exposure[66]. The need to agree upon definitions of deficient, insufficient, sufficient, and intoxication is essential in order to better identify individuals at risk for sub-adequate serum concentrations of vitamin D based on genetic or lifestyle factors[81,83].

Factors That Influence Vitamin D Status

Serum 25-OH D can be influenced by a number of factors[4]. These factors range from ethnicity, age and determinants of sunlight exposure, such as latitude and season of year[4,66].

Ethnicity

Although vitamin D insufficiency is prevalent among most nationalities, skin pigmentation plays a major role in determining serum vitamin D concentrations[6,60]. Melanin in the skin serves as a natural inhibitor of cutaneous vitamin D synthesis[84]. Melanin competes with 7-DHC for UV rays to produce skin color[85]. Chen, et al[86] studied 4 human skin types to determine the effect melanin has on vitamin D synthesis. The investigators found that subjects with darker skin pigmentation required longer exposure to UV rays in order to reach and maintain serum 25-OH D concentrations than fair pigmented subjects[86]. Similar the same sun exposure requirements were found by several researchers when examining different ethnic groups[8,45,87].

Research suggest that ethnicity may have an effect on vitamin D status[60]. Typically, non-Hispanic/white populations have the highest serum 25-OH D and African, Caribbean, or African American have the lowest serum concentrations of 25-OH D when exposed to the sun for equitable lengths of time[6,26,57,60]. Mithal, et al[88], found that in the U.S., Caucasian Americans were less likely to have serum 25-OH D below the recommended range compared to populations of African American or Hispanic American origin. The researchers also stated that immigrants to Australia from the Middle East and Asia had serum 25-OH D concentrations less than native Australians and European Australians[88]. Bischoff-Ferrari, et al[60], demonstrated that BMD was also highly correlated with culture and serum concentrations of vitamin D. The research team showed that in young adults (i.e., 20 – 49 years of age), BMD in vitamin D sufficient white individuals was 4.1% higher than vitamin D deficient African Americans[60]. By comparison, the BMD for vitamin D sufficient Mexican Americans and African Americans was 1.8% and 2.5 % higher than their respective vitamin D deficient counterparts[60]. These studies suggest that ethnicity is a determining factor in the vitamin D status of an individual as indicated by serum 25-OH D as well as BMD.

Age

Age is another factor that influences the serum concentration of 25-OH D[69]. As people age the thickness of the skin begins to decrease, altering the its ability to initiate the synthesis of vitamin D in response to UV rays[85,89]. By the 7th decade of life, most individuals will have experienced a 75% decrease in 7-DHC compared to young adults[62,65]. Therefore, vitamin D deficiency is a growing concern not only for senior citizens living in long-term care facilities, but also for older adults who are community-dwelling members of society[69].

In addition to the decrease in cutaneous synthesis of vitamin D with increasing age an inverse correlation has been demonstrated between the prevalence of nuclear vitamin D receptors (nVDR) and age[86]. The decrease in nVDRs in older adults was reported in a study performed *in situ*, of young and older adults undergoing back surgery[90]. The young adult back patients had a higher number of nuclear VDRs in the muscle cell, as well as a higher serum 25-OH D concentration, compared to the older adults[90]. In a follow up to that study, Bischoff-Ferrari, et al[91], determined that as individuals matured from adolescents into adulthood, a smaller quantity of VDRs were expressed in muscle cells. This pattern continued to decrease as they aged. These findings have lead researchers to postulate that genomic effects (i.e., muscle cell proliferation and differentiation) of vitamin D also decrease with age[86]. Fewer nuclear VDR and less 7-DHC in older adults may explain why even with adequate sunlight exposure, healthy older individuals often have suboptimal vitamin D status[67].

Sunlight Exposure

Exposure to the sun is the greatest source of vitamin D for humans[25,56]. In today's society, the average person wears clothing that covers all but 5% of their body; defined as the face, hands and arms[8,14,48,89] . In people with fair, light-skinned pigmentation, exposing 5% of the body to sunlight for 5 minutes 2 to 3 times per week during the summer can produce up to 400 IU of vitamin D daily[8,14,89]. Levis[61] and colleagues reported that people who lived in sun rich

environments maintained serum 25-OH D status between 40 ng/mL and 65 ng/mL. In addition to skin pigmentation[48,68] and sun exposure[92], there are several other factors that inhibit the sun's effectiveness to promote vitamin D synthesis in the skin, including latitude[8,46,61], season of the year[49,93], adipose tissue[14,50,68].

Latitude is a major determinant of endogenous vitamin D synthesis and therefore, 25-OH D serum concentrations[46,61]. For vitamin D to be synthesized in the skin, the sun must emit wavelengths of 270-320 nanometers (nm)[43,57,94]. The UV wavelength may be altered by factors such as time of day, atmospheric interference (e.g., pollution and cloud cover), and the distance between the Sun and Earth's surface[67,85]. When considering these complexities, UV waves available to reach human skin for vitamin D production are limited[57]. At extreme latitudes (35° N or 35° S), very few UV rays are at the appropriate wavelength for vitamin D synthesis to occur year round[57]. Boston, which is located at 42°N, has sunlight that is unable to sustain cutaneous vitamin D synthesis between November and February. North of Boston (i.e., 310 miles) in Edmonton, Canada, non-vitamin D producing sunlight is emitted between October and March[64]. In France, two general urban adult populations were tested in the north (51° N) and in the south along the Mediterranean Coast at 43°N[92]. The populations tested in the northern communities were 31% insufficient while 7% of the people in the south were insufficient[92]. In sunny areas of the world such as Milan, the Mediterranean and the Middle East, compromised vitamin D status may be attributed to heightened awareness of skin cancer, which leads to greater use of sunscreen or avoidance of the sun[61].

Healthy young adults, older adults and children have all shown fluctuations in serum concentration of vitamin D by season of the year[49,93]. During the winter months, the angle at which UV rays strike the Earth's surface change[37]. Due to the alteration of the zenith angle, the UV rays are not able to reach the critical wavelengths needed to promote cutaneous vitamin D production[37,57]. This is evident in Northern Italy (45°N), where 57% of older adult women were deficient during the months of December through May and 17% were deficient June through

November[64]. Of children studied in the winter months in coastal northern Spain (43°N), 80% had serum 25-OH D concentrations lower than 20 ng/mL, and in the summer the average concentration increased to 29 ng/mL[49]. In Europe, plasma vitamin D varied greatly based on region, but in all countries subjects had higher concentrations of 25-OH D in the summer compared to the winter[93]. In the U.S., Florida's community-dwelling older adults demonstrated similar results as the previous two studies, with subjects having greater serum concentrations of vitamin D in the summer[61].

The wavelengths of the sun are not the only natural inhibitor of endogenous vitamin D production[14]. Subcutaneous adipose distribution has also been shown to affect serum 25-OH D in adolescents[14] and adults[50]. Vitamin D and 7-DHC can be sequestered in adipose tissue which decreases the bioavailability of active vitamin D[25,50]. Studies by Zamboni, et al[25], and Snijder, et al[50], reported that the more subcutaneous adipose tissue an individual had, the lower the vitamin D status. Gilsanz, et al[21], found that higher concentrations of intramuscular adipose tissue correlated with a greater degree of vitamin D insufficiency. When compared to women with sufficient vitamin D status, women that were vitamin D insufficient had 24% greater intramuscular adipose deposits[21]. At this time, additional research is needed to determine the extent in which adiposity affects vitamin D status to inform dietary recommendations for vitamin D intake[21].

Dietary Recommendations for Vitamin D

While sunlight and irradiated foods are sources of vitamin D, the IOM reviewed and established the Dietary Reference Intakes (DRI) for vitamin D[7]. In November 2010, an IOM panel was convened to review the Recommended Daily Intake (RDA) for vitamin D and calcium, which had been set in 1997, to determine if the DRI needed to be revised based on more recent data[7]. The committee found that changes to the vitamin D RDA were prudent. The concluded there was unsatisfactory evidence to support claims of any additional health benefits aside from

those benefits associated with skeletal health and that further research was needed[7]. The current IOM recommendations are shown by age group in Table 1.

Table 1. Dietary Recommendations of the Institute of Medicine for Vitamin D [7]

Age Groups	EAR (IU)	RDA (IU)	UL (IU)
Birth to 6 months	N/A	N/A	1,000
6 to 12 months	N/A	N/A	1,500
1 to 3 years	400	600	2,500
4 to 8 years	400	600	3,000
9 to 70 years	400	600	4,000
70 years and older	400	800	4,000

EAR= Estimated Average Requirement; RDA=Recommended Dietary Allowance; UL= Upper Limit

The IOM recommendations were made on the basis that most of the American and Canadian public are receiving minimal sun exposure and experiencing normal blood calcium and low to normal fasting serum phosphorus[7]. The committee also found that most individuals have serum 25-OH D concentrations above 20 ng/mL, which is needed to maintain bone health; however, the same population was consuming less than the Estimated Average Requirement (EAR)[7]. These findings suggested that the sun exposure requirement was being met, limiting the amount of vitamin D needed through dietary and supplementation sources[7].

Following the 2010 IOM recommendations, several researchers expressed that the recommendations were too low to meet the daily requirement of the general populations. Dong, et al[95], reported that after 16 weeks of supplementation at 2,000 IU per day, African American youth increased an average of 34 ng/mL. Heaney and Holick released a commentary stating that the new recommendations disregarded all research on the non-skeletal benefits of vitamin D and that the science does not support that bone health can be maintained at a serum 25-OH D concentration of 20 ng/mL, as the IOM had claimed[96]. In a 2012 commentary by Heaney[59] the issue of defining vitamin D status in response to the IOM report was addressed. He stated that

current research had treated vitamin D intake as a medication and adopted the Evidence Based Medicine model. He argued that instead vitamin D should be treated as a nutrient, using an Evidenced Based Nutrition model to conduct research[59]. Heaney[59] also identified several key factors that make this type of research difficult to consider in the traditional medical model, such as: 1) subject specific dose response curves, 2) ethical dilemma in placing a deficient control group on low dose supplementation, and 3) the feasibility of a zero-intake control group. He concluded that until a solution for these issues is found, it will prove difficult to accurately define insufficiency, deficiency and sufficiency[59]. In order to conduct evidence based nutrition research, it is important to have good understanding of the food sources of vitamin D[96].

Food Sources of Vitamin D

Very few foods naturally contain vitamin D[9,47,94]. It is most commonly found in fish oils, cod liver oil, fatty fish (salmon, mackerel, sardines) and eggs[94,97] (Table 2). Due to the limited number of natural sources of vitamin D and the rickets epidemic of the Industrial Revolution, the United States, United Kingdom and Canada began to fortify foods such as dairy and grains[45,61]. In theory, fortification of vitamin D subsidized what was lacking in the diet, however, there has been no consensus among experts in the field as to the amount of vitamin D supplementation and fortification that was optimal[47]. Today, fortified dairy sources include milk

Table 2. Dietary Sources of Vitamin D[94]		
Food	**Serving Size**	**Vitamin D (IU)**
Cod Liver Oil	1 Tbsp	1,360
Wild Salmon	3.5 oz	981
Sun Dried Shiitaike Mushrooms	1 oz	400 - 500
Canned Sardines	3.5 oz	270
Farmed Salmon	3.5 oz	249
Tuna Ahi	3.5 oz	164
Milk, Fortified	8 oz	100
Orange Juice, Fortified	8 oz	100
Cod	3.5 oz	80
Yogurt, Fortified	4 - 6 oz	8 - 80
Margerine, Fortified	1 Tbsp	60
Kraft 2% Milk Singles (American)	1 slice	40
Cereal, Fortified	3/4 - 1 cup	40
Egg Yolk	1	18

and yogurt, while non-dairy sources include orange juice, breads and cereals[45,56]. Approximately 98% of all fluid milk is fortified with 100 IU for every 8 ounce serving of milk[56,94]. In a study examining the correlation between dietary intake of fortified foods and serum 25-OH D concentrations, healthy adolescent subjects displayed an increase in serum 25-OH D with greater consumption of milk and cereals and decreased serum concentrations with juice and soft drink consumption[68]. However, with the increasing incidence of reported lactose intolerance, fortified dairy foods may be avoided, leading to an inconsistency of vitamin D dietary intake[48]. Therefore, over-the-counter multivitamins and single vitamin supplements can provide an alternative source of vitamin D for these individuals[47,68]

Supplementation of Vitamin D

Due to the limited food sources and the many inhibitors to cutaneous synthesis of vitamin D, supplementation has become a popular means of attaining adequate vitamin D status[49,52,92]. However, knowledge of an individual's current serum 25-OH D and supplementation practices are essential to ensure sufficient vitamin D status is maintained[43].

The current RDA for adults (i.e., 9-70 years of age) is 600 IU per day[7,47,67]. Based on previous recommendations, Chel, et al[52], showed that 200 IU per day was enough to maintain current serum 25-OH D in older adults, but would not increase serum vitamin D. However, Chapuy, et al[92], reported that while 200 IU maintained current concentrations of serum 25-OH D, it should be considered inadequate for young adults due to a decrease in the time spent outdoors, poor dietary habits and general maturation. Docio, et al[49], showed that adults living in South Florida, 97% were vitamin D deficient while supplementing greater than 200 IU per day. It has been estimated that for every 100 IU of vitamin D consumed, plasma concentration status increases 1 ng/mL[43]. While supplementation has been shown to be effective in increasing serum 25-OH D concentration, caution should be taken when supplementing vitamin D to avoid intoxication[47].

Intoxication from supplementation has most often been reported in individuals who consumed large doses of vitamin D for short periods of time[52]. The tolerable upper limit (UL) is the amount where there is no potential risk for harm[9]. To ensure safety, an individual assessment of 25-OH D is required[47,56,60]. Table 3 compares the Institute of Medicine and reviewed literature recommendations for adequate vitamin D supplementation to maintain a sufficient serum 25-OH D concentrations in individuals who are at increased risk for inadequacy.

Table 3. Comparison of the IOM and Reviewed Literature Recommendations for Vitamin D Supplementation of Persons 9-70 Years of Age

	Deficiency (osteomalacia)	Insufficiency (osteoporosis)	Sufficiency	Desirable (Optimal Ca Absorption)
Institute of Medicine Recommendation	0 IU	200-400 IU	200-600 IU	Not Stated
Proposed Expert Opinion[45]	200 IU	400-600 IU	1,000-4,000IU	4,000-10,000 IU

Inhibitors of Vitamin D Bioavailability

The examination of the pharmacokinetics of vitamin D has been difficult for 2 reasons: 1) vitamin D is stored in tissues that are located throughout the body (i.e., adipose and muscle); and 2) humans are never fully deprived of vitamin D[98]. Animal studies have provided the opportunity to study the pharmacokinetics of vitamin D *in vivo* by controlling these conditions[98,99]. Lawson, et al[99], exposed rats to artificial UV rays and measured 25-OH D at various time points post exposure. They found that adipose stores contain the largest pool of vitamin D[99]. However, only 5% was recoverable to be hydrolyzed to 1α,25-OH_2 D and, 95% of cutaneous vitamin D failed to be activated by hydroxylation and was lost through urinary excretion[99]. Furthermore, only 25% of the vitamin D consumed through diet or supplementation was hydrolyzed to 25-OH D in the liver[98], and the remaining 75% was catabolized by 24-hydroxylase in the kidney and excreted[98,100].

Another factor in the bioavailability of vitamin D was the pharmacological half-life of serum 25-OH D, which ranged from 10 to 21 days[98]. In contrast, the half-life of vitamin D stored within subcutaneous fat or muscle cell increases to 30-60 days[43,46]. A study conducted by Preece and colleagues[101] compared the baseline and final serum 25-OH D of submariners after a 2 month tour with little access to vitamin D fortified foods and no vitamin D supplementation. They discovered that the serum concentration of the submariners decreased 50% during the 2 month time period, supporting the idea that vitamin D status declines in absence of sunlight, and the

body must rely on stored vitamin D[98,101]. This study supports the biological half-life and endorses that in most individuals, inadequate levels of 25-OH D may be observed in as little as 2 months, depending on the individuals vitamin D status when adequate dietary intake of vitamin D ceased[63,102].

Metabolism of Vitamin D

The two primary sources of vitamin D include cutaneous synthesis and dietary intake. Once vitamin D enters the circulation, the metabolism is somewhat complex and involves several hydroxylation and isomerization reactions as it progresses from previtamin D to a biologically active hormone[43].

Cutaneous Synthesis of Vitamin D

Cutaneous synthesis of vitamin D, whether by natural sunlight or artificial sun lamps, begins though photochemical isomerization of 7-DHC located in the phospholipid bilayer of the of the epidermis[43,56,87]. Holick, et al[103], demonstrated that 65% of 7-DHC is found in the epidermis of the skin, while the remaining 35% was located in the dermis. 7-DHC contains a chromophore that absorbs the UV (280-320 nm), initiating isomerization from 7-DHC to pre-vitamin D[43]. Havinga[104] revealed that the isomerization from pre-vitamin D to vitamin D was not affected by acids, bases, or catalyst, but was a temperature dependent isomerization (i.e., ambient air temperature at 77°F) within the epidermal layers of the skin. After Vitamin D's temperature dependent isomerization, vitamin D_3 is translocated into the dermal layer of the skin[43,45,87], followed by subsequent diffusion into the blood[94]. Approximately 60% of cutaneous vitamin D binds to vitamin D binding protein (DBP) to be taken to the liver for hydroxylation to 25-OH D or delivered to the muscle or adipose tissue to be stored[94]. Prolonged over exposure of cutaneous vitamin D to sunlight, can result in the reversible formation of luminsterol and tachysterol, or can result in the irreversible formation of toxisterol[43,87] (Figure 1). When sun exposure is not available, the body must rely on diet to maintain adequate vitamin D serum concentrations[8].

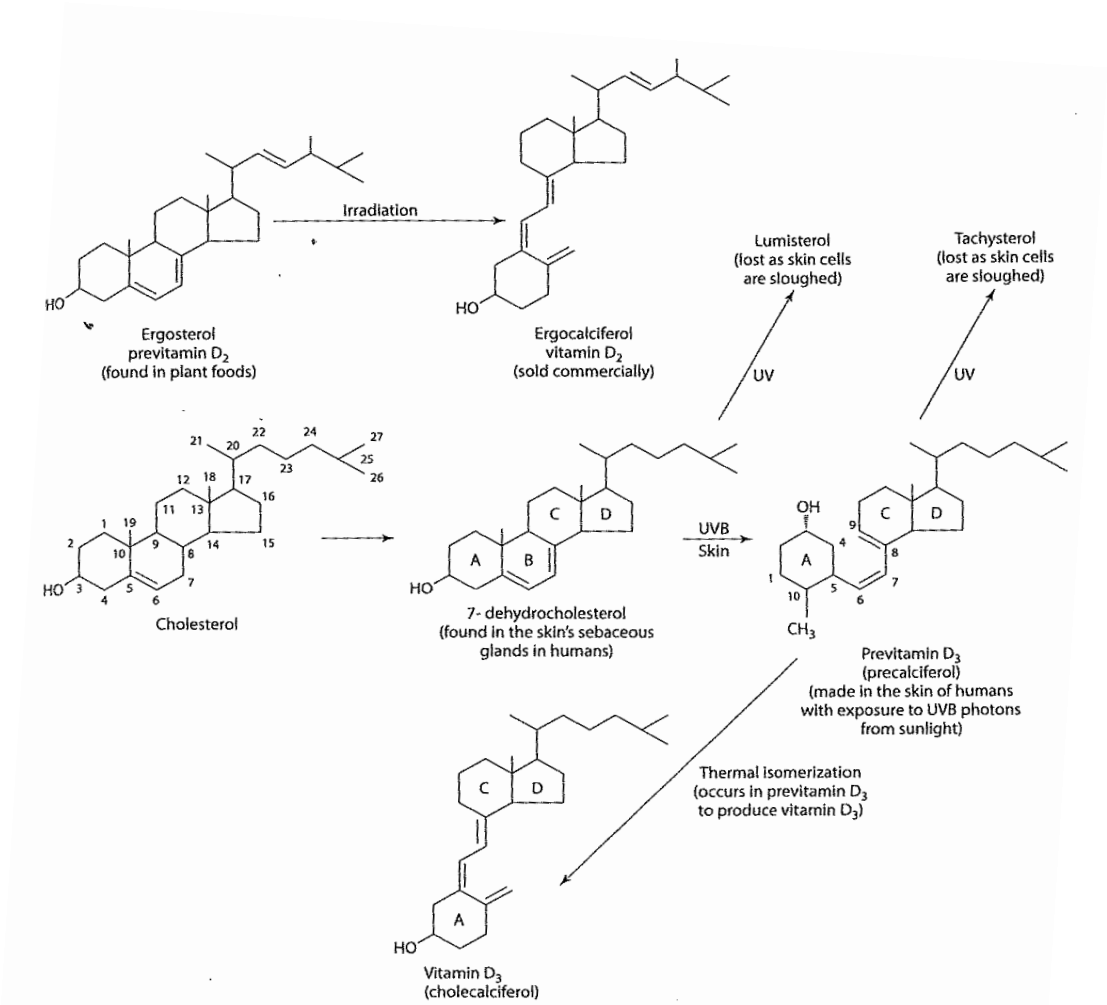

Figure 1: Cutaneous Synthesis of Vitamin D[45]

Absorption of Dietary Vitamin D

Metabolism of dietary vitamin D is crucial for the absorption and transport of the fat soluble vitamin[105,106]. Large, particles containing fat soluble vitamins are collected at the enterocyte, located in the wall of the small intestine, where a carbohydrate is added to form a chylomicron[106]. The chylomicron, consisting of triglycerides, cholesterol, fat soluble vitamins (~ 40-60% of dietary vitamin D) and phospholipids, is transported to the cell membrane and exocytosed into the lymphatic system where it is diffused into the circulatory system[106]. Chylomicrons are transported through the serum, where they undergo lipolysis by lipoprotein lipase. Chylomicrons release free fatty acids and diglycerols into cells, leaving only a

chylomicron remnant[105]. During the breakdown of the chylomicron by lipases, vitamin D may be released by the chylomicron to a DBP to travel to muscle tissue or adipose tissue for storage[105]. Some chylomicron remnants may transport vitamin D to the liver for hydroxylation to 25-OH D[105].

There are many known factors that can interfere with vitamin D absorption, including certain hormones and disease[107]. However, the literature is conflicting in describing the extent and effect each element has on vitamin D and its metabolites[47]. Hormones such as PTH, estrogen, and testosterone can increase vitamin D absorption in the gut while growth hormone and thyroid hormone are known to decrease the absorption of vitamin D[107]. Bouillon[108] described various diseases that can also have a negative effect on vitamin D status, such as renal disease or liver diseases. Liver failure can lead to vitamin D malabsorption; while liver disease and kidney disease lead to a decrease in hydroxylation of vitamin D metabolites[108]. Regardless of the source of vitamin D, to become biologically active it must undergo a series of hydroxylation reactions[43].

Hydroxylation of Vitamin D to Biologically Active Forms

Vitamin D, bound by DBP and transported through the circulatory system to the liver, is enzymatically hydroxylated by 25-hydroxylase within the endoplasmic reticulum or 27-hydroxylase within the mitochondria of hepatocytes[109]. In a meta-analysis, Holick, et al[43] showed that the hydroxylation from pre-vitamin D to 25-OH D is not controlled by vitamin D or calcium status[43], nor does it favor endogenous or exogenous vitamin D sources[109]. Rather it is controlled by release of PTH from the parathyroid gland[110], which stimulates the release of 25-OH D from the liver into the circulatory system, bound by DBP[94].

The DBP transports 25-OH D to the kidney to be further activated by 25-hydroxyvitamin D1α-hydroxylase[43,94], resulting in 1α,25-OH_2 D[9,43,56]. This hydroxylation reaction is slower than the reaction catalyzed by 25-hydroxylase[43,47,64]. The process is regulated by PTH in response to a change in serum calcium[43,47,64]. Takeyama and Kitanaka[111] used knockout mice to demonstrate

that the 25-hydroxyvitamin D1α-hydroxylase reaction does not occur in any other tissues, but the kidney and, most notably in the proximal tubule[111]. Once 1α,25-OH_2 D leaves the kidney, DBP again transports the metabolite to target tissues throughout the body for interaction with either the membrane-bound VDR (mVDR) or genomic VDR (nVDR)[43,65]. If serum 25-OH D concentrations for an individual are sufficient, then 25-OH D is catabolized by 25-hydroxyvitmain D24-hydroxylase in the distal renal tubule[43,112]. This hydroxylation allows 1α,24,25-OH_3 D to enter the C24 Oxidation Pathway, converting the vitamin D metabolites from hydrophobic to hydrophilic units and excreted through the urine[43,112] .

Vitamin D Receptor

The biologically active form of vitamin D, 1α,25-OH_2 D functions as a hormone. With the aid of transport proteins, it moves through the cell membrane to the membrane bound vitamin D receptor (mVDR), or travels through the cytosol to the nuclear vitamin D receptor (nVDR)[28,74,113,114]. Through a series of protein interactions, the VDR binds with the nuclear membrane[43], where it forms a heterodimer with retinoid-x receptor (RXR) resulting in a transcription of vitamin D response elements (VDRE)[43,74,113].

VDR possesses the ability to produce genomic or non-genomic effects[28,113,115]. Genomic effects are mediated by the nVDR, while non-genomic effects are mediated by the mVDR[28,43]. Genomic actions are generated through a slower pathway and are activated by gene transcriptions, which have been shown to generate responses in as many as 30 identified target tissues[28,113]. Non-genomic effects are described as rapid responses and cannot typically be explained by the activation of slower genomic gene transcriptions[28,113]. These genomic and non-genomic events that are mediated by vitamin D result in 1α,25-OH D functioning in a variety of physiological processes, including muscle function[43].

Muscle Function and Vitamin D

Vitamin D is essential for maintaining muscular calcium homeostasis[28,116] and neuromuscular function[28,61]. Muscle fibers contain VDRs that, when activated by their ligand 1α,25-OH_2, functions as a steroid hormone and contributes both genomic and non-genomic effects [116,117]. Genomic effects include increased synthesis of various cytoskeletal proteins that: 1) regulate calcium movement within the cell (e.g., calmodulin and calbindin D-9K); 2) induce hypertrophy (insulin-like growth factor) and; 3) control remodeling of the muscle cell surface (e.g., tyrosine kinase)[28,65,118]. Genomic effects of 1α,25-OH_2 D also include increased phosphate metabolism and the regulation of muscle cell proliferation and differentiation due to mechanisms unrelated to calcium[28]. In vitamin D deficient or insufficient individuals, these functions may be seriously compromised before classic signs of bone loss or severe deficiency appear clinically[33,61,116]. For instance, a decline in muscle strength has been associated with serum 25-OH D concentrations at or below 30 ng/mL in the older adult population[65]. However, as serum concentration of 25-OH D improve, lower extremity functions improve as well[33,67]. Currently, three working theories have proposed that vitamin D status improves muscle function by: 1) a 1α,25-OH_2 D mediated increase in the rate of the excitation-contraction mechanism by enhancing calcium re-uptake from the sarcoplasm to the sarcoplasmic reticulum 2) the VDR in striated muscle cells target 1α,25-OH_2 D, increasing phosphate transport, and 3) increasing cell proliferation and differentiation through genomic effects[28,31].

Vitamin D's Role in Calcium Re-Uptake from the Sarcoplasm

To improve muscle contraction, cellular calcium homeostasis must be maintained[119]. Intracellular calcium concentrations can be altered by voltage-dependent (non-genomic) and ligand binding (genomic) mechanisms[28,45]. Vitamin D dependent calcium binding proteins, calmodulin and calbindin D-9K, located on the cell membranes and endoplasmic reticulum membrane, bind to the calcium ions and stimulate conformational changes and alterations in cell

function[28,119]. Upon binding to these calcium binding proteins, protein kinase C (PKC) is activated which in turn stimulates an influx of extracellular calcium from the store operated calcium channels (SOCC)[28].

1α,25-OH_2 D is essential for intracellular calcium movement during a muscle contraction[120]. In the resting state, muscle fiber intracellular calcium is located in the terminal cisternae of the sarcoplasmic reticulum[121]. These cisterna have a series of calcium voltage-gates calcium channels, calcium induces-calcium release channels and an ATP dependent Ca-ATPase pump[121]. As the action potential travels down the T-tubules, the sodium/calcium channels and calcium voltage-gated channels open, allowing the calcium to enter the cell[45,122]. This increase in intracellular calcium causes the calcium induced-calcium release channels of the sarcoplasmic reticulum membrane to release large amounts of calcium into the sarcoplasm[45,121]. Animal models of vitamin D deficiency have shown that when the excitation-contraction cycle is inefficient calcium turnover in the muscle is poor[31]. The proposed mechanism by which this phenomenon occurs is a result of increased relaxation time or the availability of free calcium ions[31]. In vitamin D deficient animals, the release of troponin C from actin is delayed[33,65,123] and time to relaxation of the fibrils is prolonged[33,65]. This increase in relaxation time resulted in less tension of the muscle fibers, and overtime, decreased strength during a contraction[123]. Rodman, et al[124], demonstrated that peak tension and recovery to a resting state was delayed in vitamin D deficient rats and this response was not related to serum calcium or phosphate but could be reversed with the administration of 1α,25-OH_2 D. By exposing vitamin D depleted skeletal muscle to 1α,25-OH_2 D, *in vitro*, calcium uptake significantly increased[30,31,117,118], especially when vitamin D was administered in short intervals[117,118]. Vitamin D has been shown to increase the function of sarco(endo)plasmic reticulum calcium adenosine triphosphate-ase (SERCA)[29,33,65], an enzyme that transfers calcium from the sarcoplasm to the sarcoplasmic reticulum during muscle relaxation[125].

Another mechanism by which vitamin D influences the excitation-contraction cycle is by the number of free calcium ions available in the sarcoplasmic reticulum[123]. Calcium influx in vitamin D deficient animals was shown to be partly regulated by G-protein[126]. However in vitamin D sufficient muscle, calcium influx was regulated by protein kinase C and protein kinase A, which can activate the voltage-gated calcium channels and, increase calmodulin binding for intracellular calcium movement[35,120,126]. Vitamin D activation by this non-genomic mechanism[118] stimulates the transport of calcium which produces a greater excitation-contraction cycle[31].

Vitamin D Role in Phosphorus Regulation

The most readily available energy system that is most often associated with power production is the phosphocreatine system (ATP-CP)[125,127,128]. 1α,25-OH_2 D and 25-OH D increase phosphate uptake in the muscle cells[126], suggesting that vitamin D may play a role in ATP synthesis in the ATP-CP system[29,33,65,129]. Inorganic phosphate is considered the rate limiting factor for ATP synthesis[129]. The sodium-phosphate co-transporter enzyme that is responsible for phosphate reabsorption from the renal tubules is also present in the membranes of myocytes[126]. In vitamin D depleted animals, the ATP-CP system failed quickly, but supplementation of vitamin D prolonged the use of ATP-CP metabolism[31] making the amount of inorganic phosphate in the muscle cell, the first indication that proper vitamin D concentration had been reached[129].

More recently, research has focused on ATP-dependent calcium uptake by the sarcoplasmic reticulum, as well as normal phosphorus fluctuation across the sarcolemma[28,32]. While the correlation between the 1α,25-OH_2 D and ATP-dependent calcium uptake is not clear, some investigators have proposed a mechanism involving the activation of FGF-23[28], which increases the accumulation of phosphorus within the cell[28,32]. However, further investigation into the mechanism of the increase of phosphorus by FGF-23 is warranted[32].

Vitamin D's Role in Muscle Cell Proliferation and Differentiation

Not only does vitamin D play an important role in calcium homeostasis in the sarcoplasmic reticulum and in phosphorus metabolism, but it is also important for myoblast proliferation which aids in hypertrophy of the muscle[29,33,65,126,129]. The binding of 1α,25-OH_2 D to the VDR leads to the dephosphorylation of c-Src, a tyrosine kinase protein[28]. The dephosphorylation of c-Src stimulates the phosphorylation of several families of the mitogen activated protein kinase (MAPK)[28,32], resulting in a cascade of transcription factors regulating cell growth and differentiation[28].

Muscle myopathies, as well as atrophy, are common in vitamin D deficient individuals[126]. Sorensen[130] found that supplementation of 400 IU 1α,25-OH_2 D and 500 mg calcium for 3 to 6 months increased Type II muscle fiber area. Moreover, Sato[131] found that supplementation of 1,000 IU vitamin D alone for 2 years increased type II muscle diameter and percentage within the muscle composition. Research conducted by Ceglia and Sorensen confirmed these findings[130,132]. At this point it is not clear which of the proposed mechanisms or combination of mechanisms explains the relationship between vitamin D status and muscle function[28]. Further research is needed to determine how vitamin D serum concentrations has a potential to impact muscle strength and power[32].

Vitamin D and Muscle Strength and Muscle Power

Muscle strength and power are essential for activities of daily living[65]. Adequate vitamin D status has been associated with increased gains in muscle function in a variety of age groups and populations ranging from the adolescent to the older adult[65]. In contrast, low serum concentrations of 25-OH D are associated with compromised muscle function and increased disability[25].

Muscle Strength and Muscle Power in the General Population

Adolescents to older adults have been studied to understand the relationship between vitamin D status and muscle function[18,69]. Visser, et al[69], reported that older adults with < 25 ng/mL serum 25-OH D were at a greater risk for sarcopenia based on grip strength and muscle mass compared to a control group of vitamin D sufficient older adults. Crocombe and Mughal[133] evaluated 8 case studies over a 5 year period, and concluded that older adult subjects were unable to stand unassisted from a squat position or ascend stairs due to myopathy. In post-menarchal adolescent girls who were suffering from muscle weakness, poor vitamin D status was associated with a decrease in muscle strength, power and endurance[18]. Girls with low vitamin D serum concentrations (11.6 ng/mL) had lower vertical jump height, velocity of jump, level of fitness and generated force production[18]. Despite the low levels of serum 25-OH D, none of the girls exhibited clinical signs and symptoms of vitamin D insufficiency[18]. The measures of muscle performance used in that study have also examined muscle fiber type[65].

In the general population, muscles that have the greatest effect on muscle strength and power are mainly composed of Type II muscle fibers (i.e., fast twitch muscle fibers)[65]. Pfeifer, et al[65], demonstrated that the relationship of vitamin D serum concentration to muscle strength depended on the type of muscle fiber that was targeted. Latham, et al, showed that in frail older adults, biopsies of Type I muscle fibers (i.e., slow twitch fibers) revealed no change in activities of daily living (ADLs) with supplementation of a single dose of 300,000 IU of vitamin D[134]. Other studies found that type II muscle fibers were associated with power and have a significant effect on muscular function[18,25,26,116,134]. It has been proposed that the sarcoplasmic reticulum of type II muscle fibers is more developed than Type I muscle fibers, allowing Type II to transport calcium in and out of the sarcoplasmic reticulum at an increased rate[127]. Type II muscle fibers without exposure to adequate vitamin D, as shown through animal models, exchange calcium similar to Type I muscle fibers[65]. These studies provide evidence that supplementation of vitamin D may result in higher serum 25-OH D concentrations and affect muscle strength and power[116].

The Effect of Vitamin D Supplementation on Muscle Strength and Muscle Power

Supplementation of vitamin D not only has varying effects based on muscle fiber type, but is also specific to muscles based on anatomical location[65]. Lower serum concentrations of 25-OH D in subjects resulted in a greater loss of appendicular muscle mass[69]. Lower extremity muscle function increased the most when subjects' serum concentrations were in the range of 35 ng/mL - 40 ng/mL[26]. The most pronounced effects were observed in the muscles responsible for lower extremity power, such as the quadriceps and gastrosoleus complex[18,33,60,134,135].

In the older population, muscle strength and muscle power of the lower extremity was assessed by the number of falls over a period of time or by balance[4,26,35,52,69,134]. Studies showed that in older adults supplemented with vitamin D (800 IU), the number of falls decreased, leading the researchers to conclude the vitamin D specifically improves lower extremity musculoskeletal function[116]. Saadi, et al[63], tested vitamin D status in older women and found that low levels of 25-OH D were associated with a decrease in walking ability and increase in falls. Two other studies reported a decrease in upper and lower extremity strength associated with low levels of 25-OH D[25,69]. Much research has demonstrated that in both independent and dependent-living senior citizens vitamin D status can affect muscle strength and power, however, little research has been done to determine the effect of vitamin D status on muscle strength and power in athletes[18,70].

Vitamin D Status and the Relation to Athletic Performance

Power can be defined as force multiplied by the shortening velocity of the muscle[136]. In gross anatomy, force is most manipulated by movement requiring a high force output such as a 1 repetition maximum (1 RM), and shortening velocity is influenced by speed movement such as jumping[136]. This phenomenon, known as the force-velocity relationship, states that as the velocity of the shortening muscle increases, less actin and myosin cross bridges attach, decreasing the total force produced[137]. The opposite holds true for a muscle which is shortening at a slower velocity

(i.e., more cross bridges attach, increasing force production)[137]. However, power can also be influenced by both metabolic and ionic changes that occur within the cell[136].

Vitamin D's contribution to athletic performance can also be found in performance outcomes of muscle power and strength[39]. Several studies from Germany and Russia in the early to mid-20th century, examined the effects of pre-competition sun lamp treatments on elite athletes[37,39]. In 1927, Germany tested the effectiveness of sun lamps on swimmers before their competition and found that swimmers had decreased split times[39]. Results of this study were conveyed to the German Olympic Committee and the practice of using sun lamps prior to competition was prohibited; termed as "doping" for performance enhancement[39]. Similarly, in 1930, Russia tested the effect of sun lamps on 100 meter sprinters noting an improvement in speed as high as 7% over their baseline speed[39,138]. In the mid-1940s, Germany and the United States continued research focused on vitamin D status and performance as it related to endurance[136,139]. Both countries, using non-athletes, reported an increase in cardiovascular endurance on the bike ergometer[136,138]. Germany continued with vitamin D research and , in the 1950s began to supplement children with high doses of vitamin D to determine if supplementation was as effective as sun lamps[140,141]. The first of these studies put sun lamps in a classroom of school children to see what improvements were made in athletic performance[140]. In a subsequent study, this research team supplemented a separate classroom of students with 250,000 IU of vitamin D[141]. They concluded that 3 months post-supplementation both classrooms of school children had comparable levels of improved athletic performance[141]. During this same time period, Germany again began using sun lamps on their elite athletes to improve performance[142]. The last known study to correlate ultraviolet irradiation through the use of sun lamps to improve performance was in the United States in the 1960s[143]. A single dose of irradiation was given to the collegiate non-athlete women, which resulted in an increase in strength, speed and endurance[143-145]. These researchers concluded that exposure to irradiation can

improve muscle performance in young adults; however, this research did not incorporate any investigation into other lifestyle factors that may have contributed to this phenomenon[143].

Due to the collegiate lifestyle and dietary habits, most college athletes do not meet with RDI of vitamin D[11,146]. Athletes who train and compete indoors year round or use sun block while practicing outdoors may never achieve adequate levels of vitamin D[34,37,39]. Several studies have conducted cross sectional analysis of vitamin D serum concentrations in a variety of athletes[36,37,39]. For instance, Rankinen, et al[147], concluded that Finnish male ski jumpers had inadequate vitamin D status due to low dietary intake of all nutrients, a consequence of the sport (Table 4). A year later in 1999, Lehtonon-Veromaa, et al[36], found that Finnish gymnasts and runners when compared to controls, were just as likely to be vitamin D insufficient, with 11.3% of those studied having serum 25-OH D below 30 ng/mL (Table 4). The research team also found that vitamin D intake was low, even though they were meeting the RDA of most other micronutrients. Lovell[38] completed a similar study on elite adolescent gymnasts. He discovered that the athletes averaged 22 ng/mL serum 25-OH D (range of 11 ng/mL to 33 ng/mL), with over half of the study participants showing signs of stress fracture and unexplained muscle pain[38] (Table 4). Clark, et al[148] and Halliday, et al[146], both studied NCAA Division I athletes and both groups concluded that vitamin D intake was inadequate. However, Halliday, et al[146], found that vitamin D status or serum 25-OH D of NCAA Division I collegiate athletes was sufficient throughout the year regardless of intake. Clark, et al[148], did not assess vitamin D status against intake as part of their analysis (Table 4).

Table 4. Vitamin D Intake of Highly Trained Athletes

Study	Participants	Assess Index	Vitamin D Intake (IU)
Rankinen, et al[147] 1998	21 Finnish Elite Male Ski Jumpers, age 16-22	4 day food recall	28-172 IU
Lehtonon-Veromaa, et al[36] 1999	66 competitive runners, 65 competitive gymnast, 60 non athletic controls Age 9-15	FFQ 4 day food recall	FFQ: 88-256 IU 4 day food Recall: 56-176 IU
Clark, et al[148] 2003	13 female NCAA Div. 1 soccer player, age 19	3 day food recall	Pre-season:90-102 IU Post-season:0-204 IU
Lovell, et al 2008	18 Australian Elite Gymnast, age 10-17	FFQ	Not Reported
Halliday, et al[146] 2011	41 Div. I collegiate athletes	FFQ	Fall: 403-81 IU Winter: 488-76 IU Spring: 375-33 IU

Low exogenous vitamin D, a contributing factor to low vitamin D status, may be linked to diminished athletic performance[37]. Two studies reported that vitamin D supplementation may improve the performance[34,146]. These effects were observed only in athletes who were deficient or insufficient[34]. These studies imply that vitamin D supplementation has the potential to improve muscle power and strength in athletes with low vitamin D concentrations.

The prevalence of vitamin D inadequacy among the collegiate athletic population is likely to be high, due to lifestyle factors and unhealthy food choices[36]. Evidence from human and animal studies has demonstrated that there is a positive association between vitamin D concentration and muscle strength and power. This association may be due to: 1) calcium regulation in the sarcolemma, 2) phosphorus regulation, or 3) muscle cell proliferation[28]. Supplementation of vitamin D has the potential to improve vitamin D status, and could potentially lead to gains in strength and power. Should vitamin D status be found to highly

correlate with muscle strength and power performance measures, additional research will be needed to examine the extent to which supplementation can improve athletic performance in highly trained athletes[39].

Conclusion

Current literature suggest that in non-athletic populations there is a correlation between vitamin D status and muscular strength and power[39]. Among the athletic population, vitamin D serum concentrations may also be low due to: 1) little sun exposure, 2) the latitude of where they practice or play, 3) ethnicity, and 4) dietary intake. As such, the sun is an unreliable source for athletes to meet their daily vitamin D requirement, and dietary sources may be limited[9,12,37]. To date, there is very little data on the vitamin D status of collegiate athletes and only one study on post-menarchal adolescent girls that focused on the correlation of vitamin D status to performance measures. It is important to determine if collegiate athletes have adequate concentrations of vitamin D, what factors contribute to a lack of serum 25-OH D, and whether vitamin D status affects performance as measured by muscle strength and power.

CHAPTER III

METHODOLOGY

Study Design

The purpose of this study was to: 1) determine the prevalence of vitamin D deficiency/insufficiency in a cross section of NCAA collegiate athletes and 2) evaluate the relationship between vitamin D status and muscle strength and muscle power. This purpose was accomplished by using a cross-sectional study design to evaluate the current vitamin D status of collegiate athletes and indicators of muscle strength and muscle power.

Subjects

Participants were male and female NCAA collegiate athletes (n=103) representing NCAA Divisions IA, IAA and II. They were recruited from The University of Tulsa (TU), Oklahoma State University (OSU) and Southern Nazarene University (SNU) over a 2 year period of time. Participants were recruited though the respective athletic training staff, strength and conditioning staff and coaches. All participants were 18 years of age or older and provided written consent for participation. Inclusion criteria were clearance by a physical exam administered by the respected medical staff. Exclusion criteria included any current injury (acute or chronic) that prevented the subject from completing the performance measures, the consumption of any sport supplement that may alter the physiological effects

of strength and/or power production, and the inability to provide a viable blood specimen. Sport supplements that were considered to interfere with vitamin D status included creatine, whey protein, and supplements containing additional branch-chain amino acids.

General Procedures

Flyers describing the research study were sent to athletic training staffs, strength and conditioning staffs and coaches prior to meeting with the respective team representative. The team representative then contacted the Primary Investigator (PI) to discuss the research protocol and to determine when the team could participate in the research study.

At each school, the PI met with the teams to provide an explanation of the study and allow time for questions about the protocol. Subjects were allowed to take a folder containing the Health History Questionnaire (HHQ), Food Frequency Questionnaire (FFQ), Calcium Questionnaire (CQ) and Sun Exposure Questionnaire (SEQ) home with them and return them the following day. Additional questionnaires were available on site the day of testing if needed. It should be noted that participants were asked to include the time spent in a tanning bed as a part of the SEQ inquiring about sun or UV exposure. On the day of testing, the PI reviewed the testing protocol with each investigator at designated stations. Subjects underwent anthropometric measures of height, body weight, body composition, and girth measurements. Participants then proceeded to the respective testing areas to complete the performance measures of Vertical Jump Test, Shuttle Run Test, Triple Hop for Distance Test and a 1 Repetition Maximum (1 RM) Squat Test. Next, the subjects went to a designated area where a trained, licensed phlebotomist collected approximately 10 cc of venous blood from the non-dominant arm for analysis of vitamin D (i.e., 25-OH D). The samples were kept on ice and then centrifuged at 3,000 rpm. The serum was separated into 4 x 1,000 mL aliquots, and stored at -80°C.

Demographic and Anthropometric Data

Demographic data were collected and included ethnicity, gender, general health and injury history, and information about the average number of hours of sun exposure for the week prior to testing. Anthropometric data collection incorporated height, weight, skin tone analysis, body composition, and circumference measures. Height and weight were determined using a Decto Standing Physicians Scale (Decto, Webb City, MO).

Seven site skin-fold measurements were performed on each participant. Anatomical sites of measurement included: abdomen (one inch adjacent to umbilicus), midaxilla (four inches lateral to the xiphoid process), subscapula (midway between the vertebral border and inferior angle), triceps (midway between the acromium and olecranon process), suprailium (crest of ilium), thigh (midway between the anterior inferior iliac spine and superior patellar pole), and calf (medial aspect at maximum circumference of gastrocnemius) using the Jackson-Pollack equation to determine body composition[149]. Body circumference (i.e., girth) measures were assessed using a Medco Sports Medicine Measuring Tape (Medco, Tonawanda, NY) at the waist, hip, upper arm, mid-thigh and calf. The girth measurements were taken at the cross sectional site with the greatest girth.

Food Frequency Questionnaire

The FFQ was a 12 point questionnaire that assessed vitamin D supplement usage and dietary intake of foods that are vitamin D rich[150]. This questionnaire has been validated by United States Food standards. In short, subjects were asked to recall how many servings of each item identified in the questionnaire they had consumed in the previous month. The IUs per item was adjusted to take into account the number of servings consumed by the participants to determine a monthly intake. To determine daily vitamin D consumption, the monthly was intake was divided by 31. Permission to use the FFQ and the key to determine the IU available in each item was kindly provided by J. Dahl, who was involved in designing the questionnaire.

Serum Vitamin D Status

Non-fasting serum samples were used for the evaluation of 25-OH D. The vitamin D metabolite 25-OH D was assessed using a commercially available assay (Diasorin, Stillwater, MN). The assays were performed in collaboration with the Cooper Institute (Dallas, TX), where the guidelines set by the manufacture were followed.

Performance Measures

To assess muscular power and strength, 4 different performance measures were used including the incremental test for peak power (i.e., shuttle run), 2 iso-inertial tests (i.e., vertical jump test and the 1 legged triple hop for distance)[151], and a 1 RM squat that assessed maximal strength.

The Vertical Jump test was evaluated using a combination of the Tendo Weightlifting Analyzer V-207 (Sorinex, Irmo, SC) and contact mat. The TENDO unit cord was attached to a nylon buckle and fastened around the athletes waist with the unit placed flat on the floor behind the subject. This placement allowed for valid readings to be obtained without interfering with jump technique. The contact mat was placed under the athlete's feet to record the time from when the athlete's feet left the mat to the point when they returned. Subjects performed a series of 3 maximal jumps with their hands on their hips to prevent momentum from the upper body. A 30 second rest interval was allowed between each jump. The average of the 3 recordings was used for maximal jump force. Tendo Weightlifting Analyzer Output provided vertical height (d), power (P), and time in the air (t). Force was calculated using the formula F=P*t/d.

The One Legged Triple Hop for Distance test was used to assess lower extremity power output. A single measuring tape was fixed to the floor up to 6 meters. The subject stood on their dominant leg, which was defined as the "kicking leg". With the verbal prompt of "1, 2, 3, Go" the athlete performed 3 maximal hops on the dominant leg[149,151]. The subject performed 3 maximal trials with an average taken for record.

The Shuttle Run test has been shown to have high reliability in relation to the functional power and endurance[152]. Subjects began at the baseline of the court, sprinted to a specified distance, rapidly decelerated to a stop, turned 180 degrees, and sprinted to the baseline where they decelerated again, turned 180 degrees and sprinted a new distance. The series of sprints were 3 consecutive tests with a 10 second rest between each run. The start and stop time measured for power.

The 1 RM squat is a maximal effort test and was performed on Nautilus Xplode Plate Loaded Squat Rack (Nautilus, Vancouver, WA). 1 RM was assessed by placing 120, 150 or 200 pounds (varied by sport and gender) on the bar and asking the subject to "lift as many times possible". The 1 RM was determined when the subject could no longer complete full knee extension without assistance.

Statistical Analysis

Due to the limited number of participants, sports were combined for the analysis by sport creating an other group for both the men (i.e., cheer and track) and women (i.e., rowing, tennis and track). Descriptive statistics were calculated using SPSS version 21.0 (IBM, Armonk, NY) for all outcome variables including means, standard deviations, medians, minima and maxima. Outliers were identified as being > 2 standard deviations from the mean and removed from analysis. Data were analyzed using chi square frequencies, bivariate correlation, analysis of variance (ANOVA) and analysis of covariance (ANCOVA). Analysis of demographic groups (gender, ethnicity, sun exposure, body mass index (BMI), body composition and sport) utilized a cross tabulation Chi Square to determine the frequency of occurrence within vitamin D categories (i.e., deficient, insufficient and adequate). Chi Square cross tabulation was also used to determine the frequency distribution of subjects by performance measure normative value groups within each of the vitamin D categories. Significant differences between normative values within vitamin D status were calculated using a standardized z-score by SPSS. Within Body Mass Index

and Body Composition (% fat), the overweight and obese categories were collapsed to increase cell sizes for analysis. ANOVA was also used to analyze mean vitamin D concentrations for anthropometric measures, body composition based on normative values[153], and dietary intake.

The relationship between vitamin D concentration and indices of muscular strength and muscular power were explored using bivariate correlation analysis. The scores of performance measures in relation to the vitamin D concentration were graphed as a scatter plot with Microsoft Excel. One-way analyses of variance were used to compare the mean vitamin D concentration within the demographic characteristics of gender, ethnicity, BMI, body composition and sport. Where appropriate pairwise comparisons were evaluated using a Tukey HSD post hoc analysis. An ANCOVA was conducted to determine the effect of vitamin D status on calculated muscle strength and power controlling for lean mass, using a Sidak post hoc analysis. Lean mass was chosen as the covariate due to the high colinearity lean mass shared with vitamin D status. The significance level for all analyses was set at $p < 0.05$.

CHAPTER IV

RESULTS

Demographic Characteristics of the Study Population

NCAA athletes (n = 113) were consented in this study, of which 103 (91.1%) completed and were eligible for analysis. The reason participants (n= 10) failed to complete the study was the inability to provide an adequate blood sample. Athletes from three different universities, representing 12 NCAA collegiate sports participated in the research study. Of the population, 66.0% competed in NCAA Division II athletics while 18.8% competed in NCAA Division IA and the remaining 14.8% competed in NCAA Division IAA (**Table 5**). The majority of the subjects were in their sophomore, junior and senior years, while 16.5% represented the freshman class and < 1% represented 5^{th} year seniors (**Table 5**). Of the 12 teams that were represented in the study, 8 were women's teams and 4 were men's teams (**Table 5**).

Sixty-eight females and 35 males participated in the study (**Table 6**). Body mass index (BMI) was calculated from measured height and weight; while percent fat was determined using skin fold measurements. Based on the recommended BMI (i.e., healthy = 18.5-24.9), the average BMI of the study population (25.7 ± 5.2) was classified as overweight (**Table 6**). The recommended range for percent body fat for healthy females, age 20-40 years, is 21%-33%, and for healthy males 20-40 years, 8%-19% body fat. Based on the mean BMI and percent fat, females were within the "healthy" ranges (**Table 6**). In contrast, the mean for the males was

overweight based on BMI and were 8.7% over the healthy range in percent fat mass classifying them as overweight (**Table 6**).

Table 5. Study Population Characteristics by NCAA Division, Academic Classification and Sport

Characteristic	Overall n (%)
NCAA Division	
NCAA Division IA	19 (18.8)
NCAA Division IAA	16 (14.8)
NCAA Division II	68 (66.0)
Academic Classification	
Freshman	17 (16.5)
Sophomore	30 (29.1)
Junior	28 (27.2)
Senior	27 (26.2)
5th Year	1 (1.00)
Sport	
Women's Basketball	13 (12.6)
Volleyball	11 (10.7)
Women's Cheer	11 (10.7)
Softball	13 (12.6)
Women's Soccer	10 (9.7)
Baseball	13 (12.6)
Football	12 (11.7)
Women's Other (Rowing, Track, Tennis)	10 (9.7)
Men's Other (Cheer and Track)	10 (9.7)

Data is represented as number of subjects (percentage of the total study population).

Evaluation of dietary intake of vitamin D and calcium was based on a self-report FFQ and a calcium questionnaire. The average intake for vitamin D was 187.7 ± 226.5 IU, which is 31.1% of the RDA or 600 IU of vitamin D per day (**Table 6**). The vitamin D intake ranged from 0-1100 IU per day, with some participants not reporting any dietary vitamin D intake over the past month in their FFQ. Females consumed 50.3 IU more vitamin D per day of than males (**Table 6**). The comparison of dietary vitamin D intake means across vitamin D deficient, insufficient and sufficient status resulted in no significant differences (*data not shown*). In contrast to the low vitamin D intake relative to the RDA, daily calcium intake exceeded the RDA of 1.0 g per day by 230% (**Table 6**). There was no difference in dietary intake of calcium between males and females (**Table 6**). For all but 3 participants, the primary source of calcium intake was

from food sources and not calcium supplements (*data not shown*). Sun exposure was reported for the week previous to testing in a range of 0-2 with 0 representing < 5 minutes of sun exposure, 1 representing 5 - 30 minutes of sun exposure and 2 representing > 30 minutes sun exposure. Males and females received approximately the same amount of sun exposure with an overall average of 1.53 ± 0.48 **(Table 6)**. Even with low vitamin D intake, participates were getting adequate sun exposure with 93.1% reporting at least 5 - 30 minutes per day in the sun the week prior to data collection. Only 6.9% reported less than 5 minutes per day in the sun (*data not shown*).

Table 6. Age, Anthropometric, Vitamin D and Calcium Intake of NCAA Collegiate Athletes

Characteristic	Overall	Males	Females
n	103	35	68
Age (yrs)	20.6 ± 1.9	20.7 ± 1.7	20.6 ± 1.9
Anthropometrics			
Height (cm)	171.7 ± 10.1	179.3 ± 6.7	167.8 ± 9.4
Weight (kg)	76.3 ± 16.9	89.1 ± 14.0	69.7 ± 14.3
Body Mass Index	25.7 ± 5.2	27.7 ± 4.2	24.7 ± 5.4
Body Composition			
Fat Mass (%)	23.1 ± 6.7	20.4 ± 7.0	24.4 ± 6.1
Lean Mass (kg)	17.9 ± 5.3	21.7 ± 4.4	15.9 ± 4.6
Dietary Intake			
Vitamin D Intake (IU)*	187.7 ± 226.5	154.4 ± 209.8	204.7 ± 234.3
Calcium Intake (g)**	2.3 ± 1.8	2.3 ± 1.8	2.2 ± 1.8
Sun Exposure	1.53 ± 0.48	1.67 ± 0.42	1.46 ± 0.50

Data presented as mean ± SD. *RDA for vitamin D= 600 IU per day[7] **RDA for calcium = 1.0 g per day[7]

Comparison of Serum 25-OH D Concentration by Gender, Ethnicity, BMI, Percent Body Fat and Sport

Subjects were tested over a two year period of time in either late spring, late summer, or early fall. Time of year had no statistically significant effect ($p = 0.78$) on vitamin D status (*data not shown*). The incidence of vitamin D deficiency, insufficiency and sufficiency was 8.9%, 22.7%, and 68.3% of the population, respectively. The distribution frequency was evaluated using a chi square test and the differences in means were determined using an ANOVA. The range of serum 25-OH D concentrations of the study population was 30-70 ng/mL for those in the

sufficient groups, and 21-29 ng/mL for those in the insufficient group and 11-19 ng/mL for those in the deficient group (**Figure 2**). As expected, when the mean serum vitamin D concentrations were compared, participants in the deficient and insufficient category had significantly lower serum 25-OH D concentrations ($p < 0.01$) compared to participants in the sufficient category (**Figure 2**). Athletes in the insufficient category had higher serum 25-OH D ($p = 0.01$) than those within the deficient category (**Figure 2**).

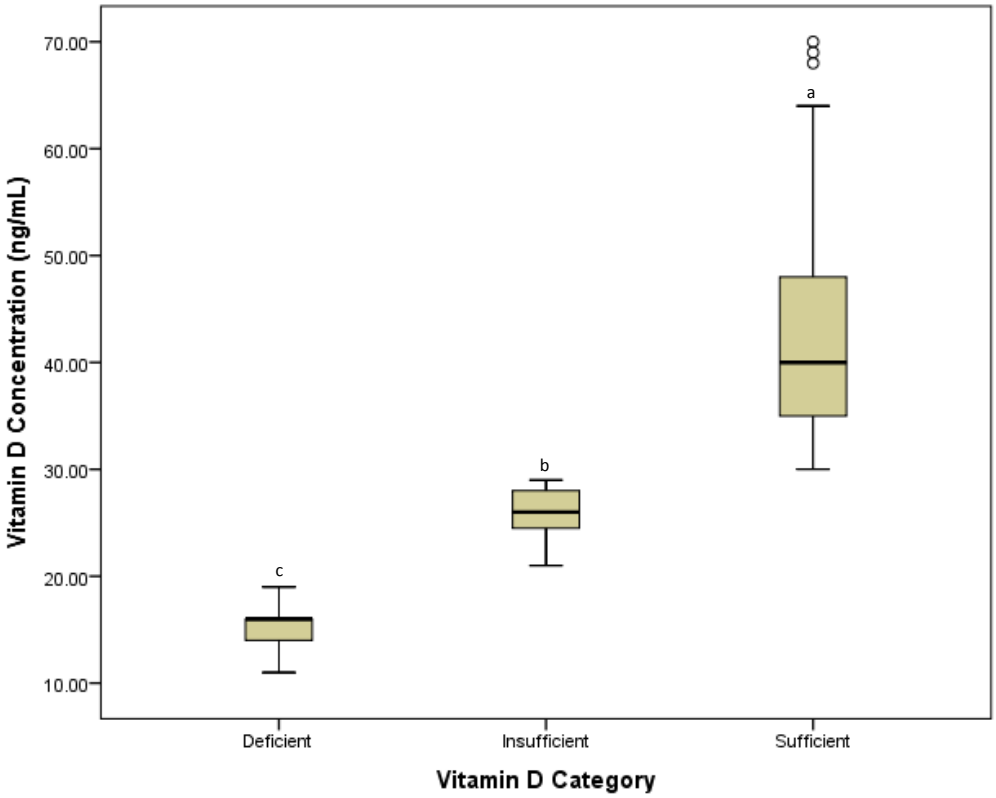

Figure 2. Study participants' serum 25-OH D distributed by vitamin D status (Deficient <20 ng/mL; Insufficient 20-29 ng/mL; sufficient ≥ 30 ng/mL). Bars show the distribution of participants within each category with the dark line within the bar indicating the mean. Bars that do not share the same superscript letter are statistically different from each other ($p < 0.05$).

To examine the differences in serum 25-OH D by gender, ethnicity, BMI, percent body fat and sport, one-way ANOVA were conducted. Females had higher serum 25-OH D ($p = 0.03$) compared to males (**Table 7**). African American and the other ethnic groups had significantly lower serum 25-OH D compared to Caucasians ($p < 0.001$) (**Table 7**). The other group included

study participants that were Hispanic, Latin and Asian Pacific. Vitamin D status was altered relative to BMI and body composition. Based on BMI, overweight/obese participants had lower (p = 0.01) serum 25-OH D compared to participants with a BMI in the normal range (**Table 7**). The comparison of body composition (i.e., % fat) and serum 25-OH D reflects similar results. Participants with % fat in the overweight/obese categories had lower serum 25-OH D concentration (p = 0.01) compared to those with percent body fat classified as normal or underfat (**Table 7**). In comparing serum 25-OH D concentrations across sports, football players had a lower (p< 0.05) 25-OH D than volleyball players, but all other pairwise comparisons revealed no significant differences between sports (**Table 7**).

Table 7. Comparison of 25-OH D Concentrations by Gender, Ethnicity, BMI, Percent Body Fat and Sport of NCAA Athletes

Characteristic	n	Vitamin D Concentration (ng/mL) Mean ± SD	*p*-value
Gender			0.03
Female	66	38.4 ± 12.5[a]	
Male	35	32.6 ± 12.4[b]	
Ethnicity			0.00
Caucasian	76	39.4 ± 11.6[a]	
African American	12	25.1 ± 10.8[b]	
Other	13	28.9 ± 12.2[b]	
Body Mass Index			0.01
Normal	51	39.5 ± 12.1[a]	
Overweight/Obese	50	33.0 ± 12.7[b]	
Body Composition (% Fat)			0.01
Underfat	23	37.7 ± 12.9[a]	
Normal	58	38.4 ± 12.8[a]	
Overweight/Obese	20	29.0 ± 10.1[b]	
Sport			0.04
Women's Basketball	11	32.4 ± 8.6[b,c]	
Volleyball	11	44.0 ± 10.3[a,b]	
Women's Cheer	11	42.2 ± 15.2[b,c]	
Softball	13	39.4 ± 13.9[b,c]	
Women's Soccer	10	35.3 ± 7.8[b,c]	
Baseball	13	31.6 ± 6.9[b,c]	
Football	12	27.8 ± 11.3[c]	
Women's Other	10	36.4 ± 15.7[b,c]	
Men's Other	10	39.7 ± 16.7[b,c]	

Comparison of mean 25-OH D concentrations using ANOVA. Data presented as mean ± SD
For a given characteristic, means that do not share the same superscript letter are statistically different from each other
Level of significance set at $p < 0.05$

Distributions of Study Participants by Vitamin D Status

The frequency distributions of the study population within a vitamin D status category (i.e., deficient, insufficient, and sufficient) was evaluated according to gender, ethnicity, BMI, body composition, and sport (**Table 8**). There was no statistically significant difference in the distribution of study participants by gender in the deficient, insufficient or sufficient categories. The frequency distribution of athletes across ethnic groups was significantly different ($p < 0.001$) by vitamin D status (**Table 8**). Caucasian participants were more likely to be sufficient or insufficient; compared to African American or other ethnicities (**Table 8**). Interestingly, there were no differences in distribution of participants by vitamin D status based on sun exposure (**Table 8**). BMI category distribution by vitamin D status did not reach the level of statistical significance. There was a trend ($p = 0.06$) toward more participants with a normal BMI in the sufficient category (**Table 8**). There were also no statistically significant differences in the distribution of study participants across body composition and sport categories by vitamin D category that were observed (**Table 8**).

Table 8. Distribution of Study Participants by Vitamin D Status by Gender, Ethnicity, Sun Exposure, Body Mass Index, Body Composition and Sport

Characteristic	Vitamin D Status				
	Totals n (%)	Deficient n (%)	Insufficient n (%)	Sufficient n (%)	*p*-Value
Gender					0.21
Female	66 (65.3)	5 (55.6)	12 (52.2)	49 (71.0)	
Male	35 (34.6)	4 (44.0)	11 (47.8)	20 (29.0)	
Ethnicity					0.00
Caucasian	76 (75.2)	1 (11.1)	14 (60.9)[a]	61 (88.4)[a]	
African American	12 (11.8)	5 (55.6)	4 (17.4)[b]	3 (4.3)[b]	
Other	13 (12.9)	3 (33.3)	5 (21.7)[b]	5 (7.2)[b]	
Body Mass Index					0.06
Normal	51 (50.4)	2 (22.2)	9 (39.1)	40 (58.0)	
Overweight/Obese	50 (49.5)	7 (77.8)	14 (60.9)	29 (42.0)	
Body Composition (%Fat)					0.62
Underfat	23 (22.7)	1 (11.1)	5 (21.7)	17 (24.6)	
Normal	59 (58.4)	5 (55.6)	12 (52.2)	41 (59.4)	
Overweight/Obese	20 (19.8)	3 (33.3)	6 (26.1)	11 (15.9)	
Sport					0.29
Women's Basketball	11 (10.8)	1 (11.1)	3 (13.0)	7 (10.1)	
Volleyball	11 (10.8)	0 (0.0)	1 (4.3)	10 (14.5)	
Women's Cheer	11 (10.8)	1 (11.1)	1 (4.3)	9 (13.0)	
Softball	13 (12.8)	2 (22.2)	4 (4.3)	10 (14.5)	
Women's Soccer	10 (9.9)	0 (0.0)	3 (13.0)	7 (10.0)	
Baseball	13 (12.9)	0 (0.0)	5 (21.7)	8 (11.6)	
Football	12 (11.9)	3 (33.3)	5 (21.7)	4 (5.8)	
Women's Other	10 (9.9)	1 (11.1)	3 (13.0)	6 (8.7)	
Men's Other	10 (9.9)	1 (11.1)	1 (4.3)	8 (11.6)	

Data is represented as the number of participants (percentage by vitamin D status category).
A cross tabs chi square analysis was done to provide frequencies by category.
For a given characteristic, column proportions (z-test) with different superscript letters indicate significantly different proportions within a vitamin D status category

Evaluation of Performance Measures Based on Vitamin D Concentration

The relationships between serum 25-OH D concentration and performance measures associated with muscle strength and power were investigated first using Pearson product-moment correlation coefficient. Three measures of functional muscle power (i.e., height, speed and distance) were assessed based on the results of the Vertical Jump Test, Shuttle Run Test, and the Triple Hop for Distance Test, respectively. Although there was no significant correlation between vertical jump height and serum 25-OH D concentration (**Figure 3a**), an inverse relationship ($p < 0.05$) was observed between 25-OH D concentrations and the average power calculated from the

Vertical Jump Test (**Figure 3b**). Likewise, there were no significant correlations between serum 25-OH D and the Shuttle Run Test time (**Figure 3c**) and the Triple Hop for Distance Test (**Figure 3d**).

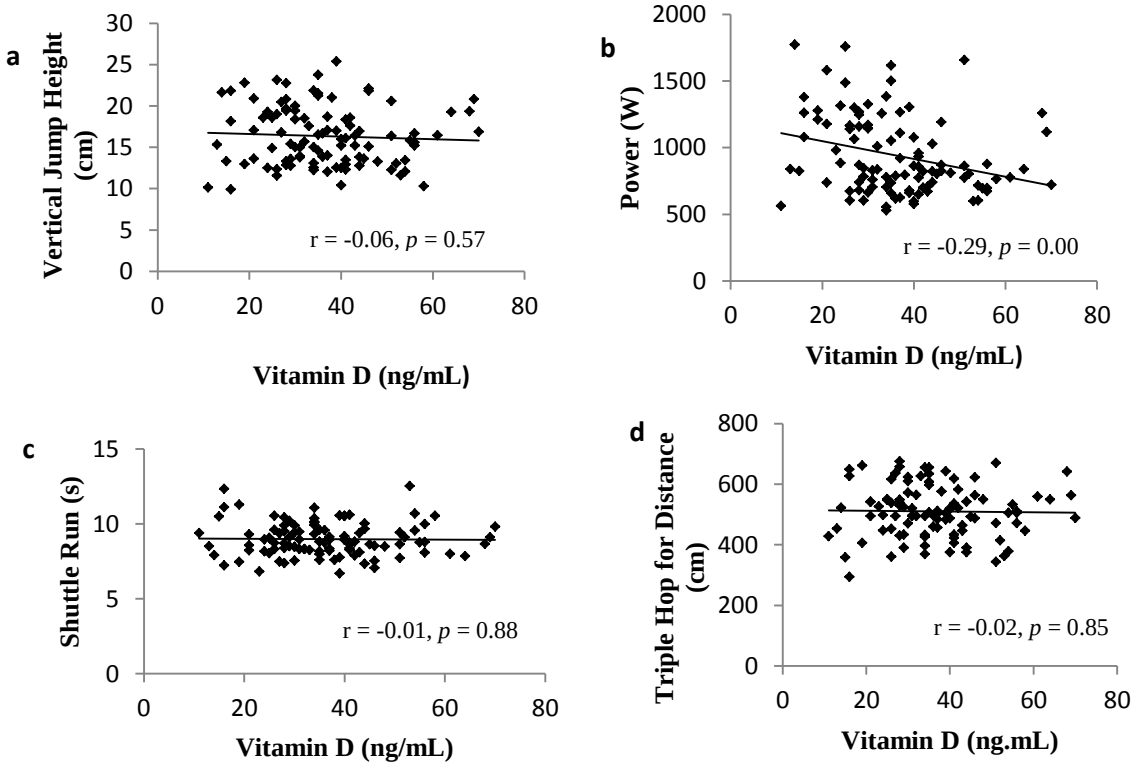

Figure 3- Correlations between 25-OH D concentrations and a) Vertical Jump height; b) average power calculated the Vertical Jump Test; c) Shuttle Run time; d) Triple Hop for Distance

Functional muscle strength was assessed by a 1 RM Squat and force generated was calculated from the Vertical Jump Test. No correlation was observed between 25-OH D concentrations and functional muscle strength based on the 1 RM Squat (**Figure 4a**), but there was an inverse relationship ($p < 0.05$) observed between 25-OH D concentrations and calculated force (**Figure 4b**). Correlations between performance measures and 25-OH D status were also evaluated based on gender and each sport separately. There were no changes in the correlations between performance measures and vitamin D concentrations observed when separated by gender and sport (*data not shown*), compared to correlations for the total sample.

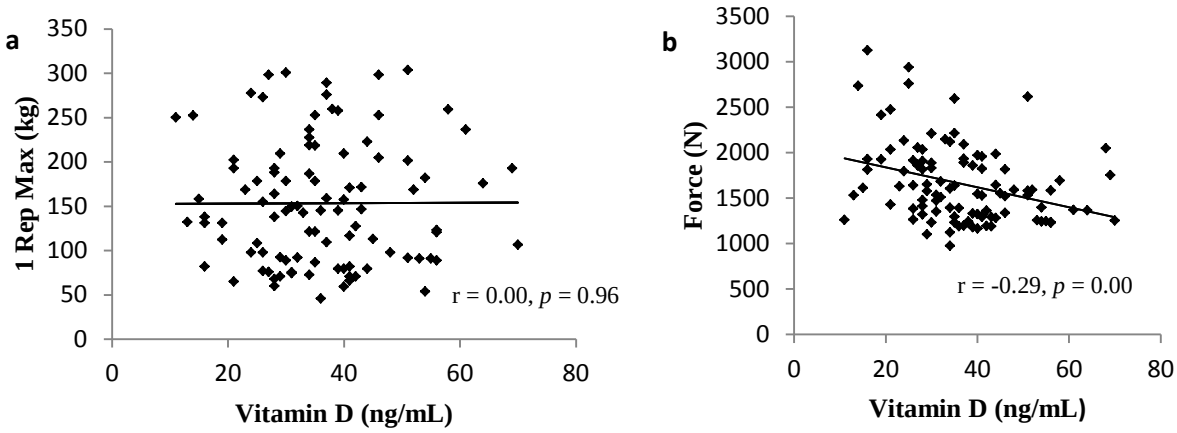

Figure 4- Correlations between 25-OH D concentrations and a) 1 RM Squat and; b) force calculated from the Vertical Jump Test

Distribution of Athletes' Performance Measures Based on Vitamin D Status

The frequency distribution of participants' performance measures within each vitamin D category was explored using chi square analyses. Normative values for performance measures of a collegiate athletic population established by the American College of Sport Medicine (ACSM) were used. These analyses did not include calculated force or the Triple Hop for Distance Test due to lack of established norms. There was no difference in the distribution of the Vertical Jump Test scores ($p = 0.50$) within the deficient, insufficient and sufficient vitamin D categories, It should be noted that 96% of the participants scored in the average category (**Table 9**). Participants' scores for the Shuttle Run Test did result in differences ($p = 0.02$) in distribution of performance levels within vitamin D status (**Table 9**). Participants with either insufficient or sufficient vitamin D status were more likely to have an excellent performance measure for the Shuttle Run Test compared to those who scored in the average or poor categories. In contrast, there was no difference in the distribution of Shuttle Run Test scores in those who were vitamin D deficient. In terms of the 1 RM Squat scores, there were no differences in the distribution of study participants by vitamin D status, however, there is a trend ($p = 0.09$) of participates who

performed in the excellent category to have sufficient vitamin D status (**Table 9**). The calculated power measure from the Vertical Jump Test resulted in no significant differences in frequency distributions within vitamin D status (**Table 9**).

Table 9. Frequency of Study Participants' Functional Performance Indicators

Functional Performance Measure	Vitamin D Status				
	Total	Deficient	Insufficient	Sufficient	*p* value
Vertical Jump (cm)					0.50
Excellent	4 (4.0)	1 (11.1)	1 (4.3)	2 (2.9)	
Average	96 (96.0)	8 (88.9)	22 (95.7)	66 (97.1)	
Poor	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	
Shuttle Run (s)					0.02
Excellent	90 (89.1)	5 (55.6)	22 (95.7)[a]	63 (91.3)[a]	
Average	5 (5.0)	2 (22.2)	0 (0.0)[b]	3 (4.3)[b]	
Poor	6 (5.9)	3 (22.2)	1 (4.3)[b]	3 (4.3)[b]	
1 RM Squat (kg)					0.09
Excellent	94 (93.1)	7 (77.8)	21 (91.3)	66 (95.7)	
Average	6 (5.9)	2 (22.2)	1 (4.3)	3 (4.3)	
Poor	1 (1.0)	0 (0.0)	1 (4.3)	0 (0.0)	
Calculated Performance Measure					
Power (W) (Calculated from Vertical Jump Test)					0.54
Excellent	53 (53.0)	7 (77.8)	13 (56.5)	33 (48.5)	
Average	30 (30.0)	1 (11.1)	7 (30.4)	22 (32.4)	
Poor	17 (17.0)	1 (11.1)	3 (13.0)	13 (19.1)	

Data is represented as number of subjects (percentage of vitamin D status).
A chi square analysis was done to provide frequencies by category.
For a given characteristic, column proportions (z-test) different superscript letters indicate significantly different proportions.

Evaluation of Performance Measures of the Subject Population Based on Vitamin D Status

Comparisons of the mean functional and calculated performance measures based on vitamin D status were accomplished using an ANOVA analysis (**Table 10**). There were no significant differences in performance measures by vitamin D status for the Vertical Jump Test, Shuttle Run Test, Triple Hop for Distance Test, and 1 RM Squat. However, there were significant differences in calculated performance measures by vitamin D status with participants demonstrating lower performance output as vitamin D status improved (**Table 10**). Participants in

the sufficient category had a lower power output ($p = 0.01$) compared to participants in the insufficient and deficient categories. Furthermore, those participants in the insufficient category had lower power output ($p < 0.001$) compared to participants in the deficient category (**Table 10**). Participants with sufficient vitamin D produced less force calculated from the Vertical Jump Test compared to those with insufficient and deficient status; and participants who were insufficient produced less force than those who were deficient (**Table 10**). In general, these findings suggest that improved vitamin D status resulted in less capacity for force production than participants with compromised vitamin D status (**Table 10**).

Table 10. Comparison of Performance Measure Means by Vitamin D Status

Functional Performance Measure		Vitamin D Status			
	n	Deficient	Insufficient	Sufficient	*p* value
Vertical Jump (cm)	100	16.2 ± 5.1	16.9 ± 3.6	16.2 ±3.4	0.67
Shuttle Run (s)	101	9.5 ± 1.9	8.8 ± 1.0	9.0 ± 1.1	0.30
Triple Hop for Distance (cm)	101	489.0 ± 133.3	520.7 ± 80.0	509.3 ± 81.1	0.64
1 RM Squat (kg)	101	154.0 ± 58.8	150.0 ± 72.6	176.8 ± 150.5	0.96
Calculated Performance Measure					
Power (1 x 10^3 W)*	100	1.13 ± 0.36[a]	1.05± 0.32[b]	0.88 ± 0.26[c]	0.01
Force (1 x 10^3 N)**	100	2.04 ± 0.61[a]	1.81 ± 0.46[b]	1.54 ± 0.40[c]	0.00

ANOVA data presented as mean ± SD
*Power calculated from Vertical Jump Test; **Force calculated from Vertical Jump Test.
For a given characteristic, means that do not share the same superscript letter are statistically different from each other.
Level of significant at $p < 0.05$

Because lean body mass could potentially confound the evaluation of vitamin D status on muscle strength and power performance measures, an ANCOVA was performed controlling for lean body mass. In the Vertical Jump Test and Triple Hop for Distance Test, controlling for lean body mass resulted in no differences in the mean scores by vitamin D status (**Table 11**). The Shuttle Run Test time tended ($p = 0.07$) to decrease as vitamin D status improved when adjusting for lean body mass (**Table 11**). Moreover, the mean performance for the 1 RM Squat was statistically different across vitamin D status ($p = 0.03$) when controlling for lean muscle mass

(**Table 11**). Post hoc analyses revealed that the amount of weight that could be lifted in the 1 RM Squat was greater ($p = 0.05$) in the participants who had sufficient vitamin D compared to those who were deficient (**Table 11**). The maximum squat lifted was 48.0% less in the vitamin D deficient group compared to participants in the vitamin D sufficient groups (Table 11). Although the comparison of the 1 RM Squat was 33.0% less for participants in the insufficient group compared to the sufficient groups, differences between these two groups did not reach the level of statistical significance ($p = 0.65$). Interestingly, no significant differences were observed by vitamin D status on calculated muscle power and force (**Table 11**). These data suggest that lean muscle mass accounted for the negative effects of vitamin D status that were observed on the calculated performance measures when compared across vitamin D status.

Table 11. Comparison of Performance Measure Means by Vitamin D Status Adjusted for Muscle Mass

Functional Performance Measure		Vitamin D Status			
	n	Deficient	Insufficient	Sufficient	*p* value
Vertical Jump (cm)	100	14.8 ± 1.2	16.3 ± 0.7	16.5 ±0.4	0.38
Shuttle Run (s)	101	9.8 ± 0.4	9.0 ± 0.2	8.9 ± 0.1	0.07
Triple Hop for Distance (cm)	101	460.5 ± 28.3	509.3 ± 17.3	516.9 ± 10.0	0.19
1 RM Squat (kg)	101	100.0 ± 41.3[a]	128.0 ± 25.3[a,b]	191.2 ± 14.7[b]	0.03
Calculated Performance Measure					
Power (1 x 10^3 W)*	100	0.91 ± 0.06	0.95± 0.4	0.94 ± 0.02	0.83
Force (1 x 10^3 N)**	100	1.77 ± 0.12	1.70 ± 0.73	1.60 ± 0.42	0.29

ANCOVA data presented as mean ± SD with muscle mass as the covariate followed by Sidak post hoc test for pairwise comparisons.

*Power calculated from Vertical Jump Test; **Force calculated from Vertical Jump Test.

For a given characteristic, means that do not share the same superscript letter are statistically different from each other Level of significant at $p < 0.05$.

CHAPTER V

DISCUSSION OF RESULTS

This study was designed to determine the prevalence of inadequate vitamin D status in NCAA athletes and to explore the relationship between vitamin D status and muscular strength and muscular power. The study population was recruited from three different universities within the state of Oklahoma and represented three different divisions of NCAA competition. The findings of this study revealed that the majority of this group of athletes (i.e., 68.3%) had serum 25-OH D levels that would be considered sufficient and only 31.7% were considered vitamin D insufficient or deficient. The prevalence of inadequate vitamin D status observed in this study is was not as high as has been reported in the general population; however, a number of these studies have focused on vitamin D status in older adults or adolescents[1,4,49,66,68,69].

Data from studies examining the prevalence of vitamin D inadequacy in collegiate and elite athletes have shown that vitamin D status is highly variable in this population. In NCAA collegiate athletes at the University of Wyoming, only 2.4% failed to maintain serum 25-OH D > 30 ng/mL throughout the academic year, regardless of sport[146]. Maimoun and colleagues[154] showed that elite cyclists had an average of 33 ng/mL serum 25-OH D and reported that 44% of the athletes had inadequate serum 25-OH D. Lehtonon-Veromaa and others[36] observed that approximately 11.3% of elite Finnish gymnasts (i.e., 9-15 years of age) were vitamin D

inadequate. The 31.7% of the athletes in the present study that had serum 25-OH D below recommended levels demonstrate that compromised vitamin D status is a health concern in this population.

The high proportion of athletes in the study with sufficient vitamin D could not be explained by vitamin D intake. Based on the results of the FFQ, study participants had a mean vitamin D intake of 31.1% (i.e., 187.7 IU) of the 600 IU per day RDA. Other studies that have examined vitamin D intake among collegiate athletes have also reported that the RDA was not being met[147,148]. For example, Rankinen and colleagues[147] concluded that elite Finnish ski jumpers had vitamin D intakes of 28-172 IU per day. Similarly, Clark, et al[148] reported that NCAA Division I soccer players had vitamin D intakes ranging from 0-204 IU per day. In the current study, vitamin D intake ranged from 0-1,100 IU per day, with a mean intake of 187.7 ± 226.5 IU per day. These studies in conjunction with the results of the current study indicate that collegiate athletes are not meeting the vitamin D dietary requirement.

Even with relatively low dietary intake of vitamin D, the majority of the participants in the current study had sufficient vitamin D status. This observation might be explained by sun exposure. Optimal subcutaneous synthesis of vitamin D (i.e., pre-vitamin D ceases to be converted to vitamin D) can be achieved with an average of 20 minutes unprotected exposure to UV rays (i.e., without sunscreen or other topical agents)[84]. The RDA for dietary vitamin D intake assumes that an individual is not meeting the sun exposure requirements, therefore the IOM made the dietary recommendation for vitamin D intake based on minimal sun exposure[7]. Approximately 53% of participants in this study reported average daily sun exposure of 5-30 minutes the week prior to testing, with 39.6% of those getting > 30 minutes per day. Only 6.9% had sun exposure < 5 minutes per day. These findings are consistent with the study by Halliday, et al[146] who reported that participants were able to maintain sufficient vitamin D status by meeting the sun exposure recommendations (i.e., 31 minutes) even with low dietary vitamin D

intake. Taken together these studies suggest that a large portion of the athletes studied were meeting their vitamin D requirement over the course of the year through sunlight exposure.

In addition to dietary vitamin D intake and sunlight exposure, there are other factors that may influence an athletes' vitamin D status including ethnicity. In the present study, differences in serum 25-OH D were observed across ethnic groups. Caucasians had higher serum 25-OH D concentrations (i.e., sufficient) compared to African American and Other ethnic groups who were mostly deficient/insufficient. These findings were in agreement with Shea, et al[3], who reported a serum 25-OH D in African American subjects of 21 ng/mL, while their Caucasian counterparts had serum 25-OH D of approximately 29 ng/mL throughout the year. Shindle, et al[155] compared the vitamin D status of football players in the National Football League (NFL) across ethnic groups. They found that 25-OH D concentrations were significantly lower in African American players (20.4 ng/mL) compared to Caucasian players (30.0 ng/mL)[155]. This phenomenon may be due to the increase of melanin in the skin of ethnic groups with darker skin tone[156]. It should be noted that the current study sample was predominantly Caucasian (75.2%), with lighter skin tones, which may have contributed to the high serum 25-OH D.

Body composition (i.e., percent fat) is also recognized as a factor that can influence serum 25-OH D. Individuals with high percent fat (i.e., males > 25%, females > 33%) or low percent fat (i.e., males < 8%, females < 18%) have been shown to be at increased risk for serum 25-OH D levels below 30 ng/mL[14,21,153]. Lenders and colleagues[14] concluded that in a population of obese adolescents, body fat was inversely related to 25-OH D. In the current study, participants whose percent body fat was categorized as normal had higher vitamin D concentrations compared to those who were overweight or obese. Those athletes in the normal range had a mean serum 25-OH D of 38.4 ± 12.1ng/mL and those in the overweight/obese had a mean serum 25-OH D of 29.0 ± 10.1 ng/mL. Lagunova, et al[157], reported that serum vitamin D was inversely correlated with morbid obesity based on BMI > 40. However, because athletes have higher percent lean mass, the authors suggested that fat mass may be a better predictor of 25-OH D than BMI[157].

Additionally, Bartoszewska and colleagues[32] demonstrated that vitamin D is stored in muscle tissue. It has been suggested that athletes who have higher lean muscle mass also have higher serum 25-OH D, but further research is needed to understand the relationship between lean muscle mass and vitamin D[157].

In addition to studying the prevalence of inadequate vitamin D status among athletes, this study was also designed to determine the extent to which vitamin D status influenced muscle strength and power. When the relationship between serum 25-OH D and functional performance measures (i.e., Vertical Jump Test, Shuttle Run Test, Triple Hop for Distance Test and the 1 RM Squat) was explored, no significant correlations were observed. Weak, but statistically significant, inverse correlations existed between serum 25-OH D and the calculated performance measures of power and force. When comparing mean performance scores across vitamin D status, athletes with sufficient vitamin D did not have improved scores compared to those with inadequate status. Furthermore, athletes with deficient vitamin D status had significantly higher calculated performance outputs for power and force than those who were sufficient. In order to determine if lean muscle mass was confounding these results, an ANCOVA was utilized to control for lean body mass. The negative effects of vitamin D sufficiency that were previously observed on calculated power and force were no longer evident when controlling for lean body mass. Athletes who were vitamin D sufficient tended to have faster Shuttle Run times than those who were vitamin D deficient, although this difference did not reach the level of statistical significance ($p = 0.07$). Furthermore, athletes who were vitamin D sufficient were able to lift more weight (kg) while performing a 1 RM squat than those who were vitamin D deficient. The only other study to evaluate the correlation between vitamin D and muscle power and strength utilizing some of the same measures used in the current study was conducted in a population of adolescent females who were not identified as athletes[18]. The researchers investigated the relationship between vitamin D status and the Vertical Jump Test and found that those with higher serum 25-OH D had improved performance for jump velocity, jump height, power and

force[18]. Moreover, Barker and colleagues[27], concluded that there was a positive correlation between serum 25-OH D concentrations and post-surgical quadriceps muscle strength[27] in young adults. Studies with older adults have also shown improved muscle strength and power by testing the Timed Up and Go Test and Eight Foot Walk Test[23,26]. Bischoff-Ferrari and others[60] reported that older adults with serum 25-OH D > 30 ng/mL were able to stand more quickly and walk farther than those who had serum 25-OH D < 30 ng/mL. Saadi and colleagues[63] also demonstrated that older adults with sufficient vitamin D status were able to walk farther and noted a significant decrease in falls. These studies support that muscle performance is associated with higher levels of 25-OH D concentration.

In the current study, the Shuttle Run Test represented a functional measure of power and the 1 RM Squat represented a functional measure of force. Shuttle Run Test times were approximately 1 second faster in those that were vitamin D sufficient compared to those who were deficient, even though statistically this resulted in only a trend toward faster times. Participants who were vitamin D sufficient had an approximately 2-fold higher squat compared to those who were vitamin D deficient. Although no significant difference in the 1 RM Squat was observed between athletes who were vitamin D sufficient and those who were insufficient (p = 0.67), the vitamin D sufficient athletes had scores that were 1.5-fold higher than the athletes who were insufficient. It is well established that muscle power and strength are predictors of athletic performance and highly correlated with an athlete's ability to perform at their highest level[151,158-160]. The performance measure data from this study suggest that better vitamin D status likely contributes to greater performance in both the Shuttle Run Test and 1 RM Squat and may translate to potential for a higher level of sport performance.

In conclusion, the current study of collegiate athletes demonstrated that greater than 30% of the population was vitamin D inadequate indicating that one-third of the population may benefit from vitamin D supplementation. Furthermore, results of the current research indicate that athletes with higher vitamin D status had higher functional performance, specifically with the

Shuttle Run Test and 1 RM Squat, compared to athletes with deficient vitamin D status. These results reveal that athletes who are vitamin D deficient, or approximately 9% of the population, may experience compromised muscle strength and power. More research is needed to determine the factors that contribute to vitamin D deficiency and insufficiency among college athletes and to examine the extent to which vitamin D supplementation can improve serum 25-OH D and athletic performance in this population.

CHAPTER VI

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS FOR FUTURE RESEARCH

Summary

A high prevalence of vitamin D inadequacy has been documented among the general population[4] and higher 25-OH D concentrations have been associated with greater muscle strength and power in older adult populations[26]. Research focused on serum 25-OH D and muscle strength and power in collegiate athletes has been limited. In the current study, NCAA Division IA, IAA and II athletes (n = 103) performed a battery of functional performance tests, completed a series of questionnaires related to factors known to affect vitamin D status and had 25-OH D serum concentrations and anthropometric measures assessed. Analyses of the data revealed the majority (i.e., 68%) of the study participants were vitamin D sufficient (i.e., > 30 ng/mL) and 31.6% has inadequate vitamin D status. Factors influencing vitamin D status included ethnicity, body mass index, percent fat and sport. There were no significant correlations between vitamin D concentration and the Vertical Jump Test, Shuttle Run Test, Triple Hop for Distance Test and 1 RM Squat scores. A weak inverse relationship existed between serum 25-OH D and calculated power and calculated force. However, when controlling for lean body mass, athletes who were vitamin D sufficient had a greater 1 RM Squat score compared to athletes that were vitamin D deficient. Furthermore, Shuttle Run Test times tended to be faster in athletes whose vitamin D status was sufficient compared to athletes who had vitamin D insufficient and deficient status

These findings indicate the prevalence of inadequate vitamin D status among this sample of collegiate athletes was lower than what has been reported in the general population, but was similar with studies conducted with elite and collegiate athletes[36,146]. The effect of vitamin D concentration on muscle strength is evident in the 1 RM Squat and concurs with a previous study conducted with post-menarchal females[18].

Conclusions

The primary null hypotheses tested were:

1) There will be no participants who are vitamin D deficient/insufficient in cross-section of collegiate athletes.

   Reject the null hypothesis. The distribution of study participants across vitamin D categories revealed that while most study participants were vitamin D sufficient (68.3%), 22.7% of the athletes were insufficient and 8.9% were deficient. Based on these findings the null hypothesis was rejected.

2) Collegiate athletes with sufficient vitamin D status will not have higher Vertical Jump Test scores than those who are insufficient or deficient.

   Fail to reject the null hypothesis. Comparison of mean Vertical Jump Test scores among athletes that were deficient, insufficient or sufficient revealed that there was no statistically significant difference between vitamin D status and Vertical Jump height. When controlling for lean body mass, the comparison of mean Vertical Jump Test scores remained insignificant. Therefore, the null hypothesis was not rejected.

3) Collegiate athletes with sufficient vitamin D status will not have faster Shuttle Run Test times than those who are insufficient or deficient.

   Fail to reject the null hypothesis. Comparison of mean Shuttle Run Test times among athletes that were deficient, insufficient or sufficient revealed that there was no statistically significant difference between vitamin D status Shuttle Run times. When controlling for lean body mass, the comparison of mean Shuttle Run Test scores approached the statistical significance. However, based on these findings the null hypothesis was not rejected.

4) Collegiate athletes with sufficient vitamin D status will not have longer Triple Hop for Distance Test scores than those who are insufficient or deficient.

   Fail to reject the null hypothesis. Comparison of mean Triple Hop for Distance scores among athletes that were deficient, insufficient or sufficient revealed that there was no statistically significant difference in Triple Hop scores by vitamin D status. When controlling for lean body mass, the comparison of mean Triple Hop for Distance Test scores remained insignificance. Based on these findings, the null hypothesis is not rejected.

5) Collegiate athletes with sufficient vitamin D status will not have higher 1 Repetition Maximum (1 RM) Squat scores than those who are insufficient or deficient.

   Reject the null hypothesis. Comparison of mean 1 RM Squat scores among athletes that were deficient, insufficient or sufficient revealed that there was no statistically significant difference in 1 RM Squat score across vitamin D status groups. When controlling for lean body mass, the comparison of mean 1 RM Squat scores became significant with participants in the deficient vitamin D status significantly lower than

those in the vitamin D sufficient status. Based on these findings the null hypothesis was rejected.

6) Collegiate athletes with sufficient vitamin D status will not have greater muscle power output scores based on the Vertical Jump Test than those who are insufficient or deficient.

   Fail to reject the null hypothesis. Comparison of mean calculated power scored based on the Vertical Jump Test among athletes that were deficient, insufficient or sufficient revealed that there was a statistically significant difference power output among participants with deficient vitamin D status compared to participants that were vitamin D sufficient. However, when controlling for lean body mass, no significant difference in calculated power scores was observed on vitamin D status. Therefore, the null hypothesis was not rejected.

7) Collegiate athletes with sufficient vitamin D status will not have greater muscle force output scores based on the Vertical Jump Test than those who are insufficient or deficient.

   Fail to reject the null hypothesis. Comparison of mean calculated force scored based on the Vertical Jump Test among athletes that were deficient, insufficient or sufficient revealed that participants who were vitamin D deficient had significantly higher force outputs then those who were vitamin D sufficient. However, when controlling for lean body mass, the comparison of mean calculated force based on vitamin D status was not statistically significant. Based on these findings the null hypothesis was not rejected.

The ancillary null hypotheses tested were:

1) There will be no difference in vitamin D status based on subject's vitamin D intake in collegiate athletes.

   Fail to reject the null hypothesis. There was no significant difference in vitamin D intake across vitamin D status categories. Based on these findings the null hypothesis was not rejected.

2) There will be no difference in vitamin D status based on the subjects sport.

   Fail to reject the null hypothesis. There was no statistically significant difference in the frequency of athletes by sport across vitamin D status categories. Based on these findings the null hypothesis is rejected.

3) There will be no difference in vitamin D status based on subject's gender in collegiate athletes.

   Fail to reject the null hypothesis. There was no statistically significant difference in the frequency of athletes by gender across vitamin D status categories. Based on these findings the null hypothesis is not rejected.

4) There will be no difference in vitamin D status based on subject's ethnicity in collegiate athletes.

   Reject the null hypothesis. There was a statistically significant difference in the frequency of athletes by ethnicity across vitamin D status categories. Based on these findings the null hypothesis is rejected.

5) There will be no difference in vitamin D status based on subject's body composition (% fat) in collegiate athletes.

   Fail to reject the null hypothesis. There was no statistically significant difference in the frequency of athletes by body composition (% fat) across vitamin D status categories. Based on these findings the null hypothesis is not rejected.

Recommendations for Future Research

In this study, approximately one-third of the athletes in the study population had inadequate vitamin D concentrations. It is important to emphasize that the primary source of serum 25-OH D was not dietary intake which suggests that these findings cannot be extrapolated to other sports or other regions where sun exposure may vary. Future research is first needed with a larger sample size to clearly establish if inadequate vitamin D status is an issue among collegiate athletes in Oklahoma. To determine the prevalence of vitamin D inadequacy in other populations of athletes, future studies need to be designed giving consideration to season of the year and latitude which can significantly affect sun exposure. If inadequate vitamin D status is established as a significant health issue among collegiate athletes, research will be needed to determine the contributing factors (e.g., sun exposure, latitude of residence, and vitamin D intake) so that health care professionals can utilize the information to identify individuals who should be monitored and might benefit from vitamin D supplementation.

The results of this study also showed that athletes who had inadequate vitamin D status demonstrated lower performance outputs for the 1 RM Squat and a trend toward slower times in the Shuttle Run Test with decreased serum 25-OH D. However, no other muscle power and strength performance measures reached the level of statistical significance. Although there were 103 participants in the current study, the population can be considered heterogeneous due to the inclusion of both males and females and athletes from 12 different sports teams. This approach resulted in having only 10-13 athletes from each sport or rather small cell sizes. Future studies

investigating the relationship between vitamin D status and muscle power and strength should consider studying a larger and more homogenous study population.

Based on the findings of this study, additional research is needed to determine if supplementing athletes who have inadequate vitamin D status would allow them to become vitamin D sufficient and improve performance outcomes. Using an experimental research design, participants should be recruited and screened based on vitamin D status and ethnicity to achieve equal sample sizes. The significance of this study would identify athletes who could benefit from vitamin D supplementation as an ergogenic aid and provide allied health care professionals with another tool to enhance rehabilitation of athletes after injury.

Lastly, studies are needed to compare if vitamin D supplementation equally affects athletes who participate in a sport that specializes in power (e.g., power cling) and strength (e.g., 1 RM Squat). Using an experimental research design, participants would be recruited based on specialization and supplemented with vitamin D based on status. The significance of this research would aid the athletic community in further identifying athletes who may achieve gain in muscle strength and power resulting from improved vitamin D status.

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APPENDICES

Oklahoma State University Institutional Review Board

Date:	Thursday, April 19, 2012
IRB Application No	ED1274
Proposal Title:	A Cross-Sectional Study Examining Vitamin D Status in Relation to Muscular Strength and Muscle Power in Collegiate Athletes
Reviewed and Processed as:	Expedited

Status Recommended by Reviewer(s): Approved Protocol Expires: 4/18/2013

Principal Investigator(s):

Rachel Hildebrand
180 Colvin Center
Stillwater, OK 74078

Brenda Smith
420 HES
Stillwater, OK 74078

The IRB application referenced above has been approved. It is the judgment of the reviewers that the rights and welfare of individuals who may be asked to participate in this study will be respected, and that the research will be conducted in a manner consistent with the IRB requirements as outlined in section 45 CFR 46.

☒ The final versions of any printed recruitment, consent and assent documents bearing the IRB approval stamp are attached to this letter. These are the versions that must be used during the study.

As Principal Investigator, it is your responsibility to do the following:

1. Conduct this study exactly as it has been approved. Any modifications to the research protocol must be submitted with the appropriate signatures for IRB approval.
2. Submit a request for continuation if the study extends beyond the approval period of one calendar year. This continuation must receive IRB review and approval before the research can continue.
3. Report any adverse events to the IRB Chair promptly. Adverse events are those which are unanticipated and impact the subjects during the course of this research; and
4. Notify the IRB office in writing when your research project is complete.

Please note that approved protocols are subject to monitoring by the IRB and that the IRB office has the authority to inspect research records associated with this protocol at any time. If you have questions about the IRB procedures or need any assistance from the Board, please contact Beth McTernan in 219 Cordell North (phone: 405-744-5700, beth.mcternan@okstate.edu).

Sincerely,

Shelia Kennison, Chair
Institutional Review Board

Oklahoma State University Institutional Review Board

Date: Tuesday, September 11, 2012 Protocol Expires: 9/10/2013

IRB Application No: ED1083

Proposal Title: A Cross-Sectional Study Examining Vitamin D Status in Relation to Muscular Strength and Muscle Power in Division I Collegiate Athletes

Reviewed and Processed as: **Modification/Continuation**

Status Recommended by **Approved**

Principal Investigator(s):

Rachel Hildebrand	Brenda Smith	Steven Edwards
180 Colvin Center	420 HES	325U Willard
Stillwater, OK 74078	Stillwater, OK 74078	Stillwater, OK 74078

Approvals are valid for one calendar year, after which time a request for continuation must be submitted. Any modifications to the research project approved by the IRB must be submitted for approval with the advisor's signature. The IRB office MUST be notified in writing when a project is complete. Approved projects are subject to monitoring by the IRB.

☒ The final versions of any printed recruitment, consent and assent documents bearing the IRB approval stamp are attached to this letter. These are the versions that must be used during the study.

Comment:
The modification request to increase sample population by 58 and expand to include data collection at Southern Nazarene University is approved.

Signature : Shelia M. Kennison

Shelia Kennison, Chair, Institutional Review Board

Tuesday, September 11, 2012
Date

THE UNIVERSITY of TULSA

Office of Research and Sponsored Programs

MEMORANDUM

TO: Nadine Brink, Sports Medicine

FROM: Dawnett Watkins
Human Protections Administrator

DATE: October 25, 2010

SUBJECT: IRB Approval – Protocol No. 11-24

The University of Tulsa Institutional Review Board has reviewed your proposal, "A Cross-Sectional Study Examining Vitamin D Status in Relation to Muscular Strength and Muscle Power in Division I Collegiate Athletes," under the University's expedited review procedures. The Board found that this research would not constitute a risk to participants beyond those of normal, everyday life. Therefore, the Board has approved the use of human subjects in this research. A stamped approved copy indicating the approval and expiration dates of your informed consent and recruitment materials, if appropriate, is attached to this approval.

This approval is under Expedited Category 2. for research on the collection of blood samples by finger stick, heel stick, ear stick, or venipuncture as follows: (a) from healthy, nonpregnant adults who weigh at least 110 pounds. For these subjects, the amounts drawn may not exceed 550 ml in an 8 week period and collection may not occur more frequently than 2 times per week; or (b) from other adults and children, considering the age, weight, and health of the subjects, the collection procedure, the amount of blood to be collected, and the frequency with which it will be collected. For these subjects, the amount drawn may not exceed the lesser of 50 ml or 3 ml per kg in an 8 week period and collection may not occur more frequently than 2 times per week.

This approval is for a period of twelve months from October 6, 2010, provided that the research procedures are not changed from those described in your application and attachments. Should you wish to deviate from the described subject procedures, you must notify me and obtain prior approval from the Board for the changes.

Please note that the IRB will forward an annual review reminder notice to you by email, as a courtesy. Nevertheless, **it is your responsibility as principal investigator to remember the renewal date of your protocol's review.** Please submit your continuation application at least two weeks prior to the renewal date, to insure the IRB has sufficient time to complete the review.

At the end of the research, you must submit a short report describing your use of human subjects in the research and the results obtained. If the research is to continue beyond October 5, 2011, the Board has determined that a progress report must be submitted no later than September 6, 2011 with a request for re-approval. A copy of the form that may be used can be found at our website: http://www.utulsa.edu/research/ under Human Subjects Compliance.

If you leave The University of Tulsa (TU) and are no longer a student or employee, you cannot continue to reference TU on any documents (including the informed consent form) or conduct the study under the auspices of TU.

If you have any questions concerning your approval or changes to your protocol, please contact me at 631-3310 or via e-mail at dawnett-watkins@utulsa.edu. Additional information concerning the requirements for the protection of human subjects may be found at the Office of Human Research Protection website - http://hhs.gov/ohrp/.

/dw

cc: Elana Newman, Chair, IRB

attachments: Informed Consent

THE UNIVERSITY of TULSA

Office of Research and Sponsored Programs

MEMORANDUM

TO: Nadine Brink, Sports Medicine

FROM: Dawnett Watkins
Human Protections Administrator

DATE: February 25, 2011

SUBJECT: IRB Revision Approval: Protocol No. 11-24

The University of Tulsa Institutional Review Board has reviewed your requested revision to 1) do blood draws in TU's Athletic Training Facility rather than TU's Alexander Health Center, 2) remove "residence for summer months of 2010" from demographic data, 3) change how data is collected on the Vertical Jump Test and 4) use graduate students in data collection to the subject protocol entitled "A Cross-Sectional Study Examining Vitamin D Status in Relation to Muscular Strength and Muscle Power in Division I Collegiate Athletes." The Board found that this revision would not constitute a risk to participants beyond those of normal, everyday life. Therefore, the Board has approved the revised protocol. A stamped approved copy indicating the approval and expiration dates of your informed consent and recruitment materials, if appropriate, is attached to this approval.

If you have any questions concerning your approval, or changes to your protocol, you may contact me at 631-3310 or via e-mail at dawnett-watkins@utulsa.edu. Additional information concerning the requirements for the protection of human subjects may be found at the Office of Human Research Protection website - http://hhs.gov/ohrp/.

/dw

cc: Paula Cadogan, Chair, IRB

enclosures:

Southern Nazarene University

CHARACTER | CULTURE | CHRIST

Institutional Review Board

September 7, 2012

Rachel Hildebrand
SNU – Kinesiology
Campus

RE: Research Submission #12-08-02

Dear Rachel::

The Institutional Review Board (IRB) reviewed your research request submitted August 14, 2012, The IRB at Southern Nazarene University **has approved** your project as presented. Any changes made to this project must again be presented to the IRB for approval prior to performing research.

Please note that the IRB must be notified in writing once the research is complete. You may contact the IRB at (405) 491-6323 with any questions or visit our web site at www.snu.edu. Best wishes for success with your research project.

Sincerely,

Dorothy A Stasser

Dorothy Stasser
IRB Chair

Institutional Review Board
6729 N.W. 39th Expressway
Bethany, OK 73008
Beaver Science Building - 433
405.491.6323 - kadams@snu.edu

Release of Vitamin D Test Results

I, _____, give my permission for Rachel Hildebrand, ATC/LAT and Dr. Brenda Smith to release my vitamin D status to Dave Polanski, ATC/LAT, Head Athletic Trainer of The University of Tulsa Athletics.

_____ _____

Student-Athlete Signature Date

_____ _____

Witness Signature Date

Food Frequency Questionnaire

Hello, and thank you for participating in our study. Please complete all 11 pages of this form honestly and with as much accurately as possible. Your responses are important to us. Thanks again, and have a great day!

Participant's Code: _____

Today's Date: _____

For the following section, please indicate how many **servings** of each food or drink you consume **per month (31 days)**. You may report a whole number or use fractions or decimals. The size of a serving is listed beside each food or drink. Please pay close attention to the food description and the serving size listed for each item. If you had no servings of that food or drink, please leave the box empty- do not write a zero.

A calculator will be available if needed.

If you consistently consume a certain food or drink on a weekly basis, it may help to think of the number of times per week you consume that item and multiply that number by 4.

For example, if you have 2 cups of milk every day, this would count as 62 servings of milk/month.

Size comparisons are listed below:

1 cup = tennis ball
1/4 cup = golf ball
3 oz = deck of cards

Note:

1 Tbs. (tablespoon) = 3 teaspoons

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Supplements:		
MULTIVITAMINS:		
One-A-Day		
Women's Health	1 tablet	
Men's Health	1 tablet	
Weight Smart	1 tablet	
All Day Energy	1 tablet	
Active	1 tablet	
Nature Made		
For Him 50+	1 tablet	
For Her 50+	1 tablet	
For Her	1 tablet	
Multi-Complete	1 tablet	
Centrum		
Performance	1 tablet	
Cardio	1 tablet	
Silver	1 tablet	
Sundown		
Daily Multivitamin	1 tablet	
Complete Womens	1 tablet	
Others		
Disney Gummies	2 gummies	
Flinstone Gummies	2 gummies	
Flinstone Chewable	1 tablet	
Publix Complete Animal Shapes	1 tablet	
Publix Century Advantage Multivitamin	1 tablet	
Viactiv Multivitamin Flavor Glides	1 tablet	
STAND ALONE D:		
Sundown Naturals	1 soft gel	
NatureMade D	1 tablet	
NatureMade Cod Liver Oil	1 soft gel	
CALCIUM/ VITAMIN D:		
Publix Oyster Shell Calcium and D	1 tablet	
PublixCalcium Soft Chews	1 piece	
Publix Calcium Citrate	2 caplets	
Publix Calcium 500 w/ D	1 caplet	
Publix Natural High Potency Calcium and D	3 caplets	
Viactiv Calcium Chews (caramel and chocolate)	1 chew	
Citrical Calcium Citrate	2 tablets	
OsCal	1 caplet	
OsCal Chewable 500 w/ D	1 piece	
Caltrate 600-D	1 tablet	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Cheese:		
Kraft		
Singles-Slices (2% American)	1 slice	
Singles-Slices American with Calci-3	1 slice	
Singles-Slices White American with Calci-3	1 slice	
Borden		
Singles Triple Calcium Slices- American, With American	1 slice	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Cottage Cheese:		
Breakstone's		
4% Milkfat Large Curd	1/2 cup	
Fat Free Small Curd	1/2 cup	
Cottage Doubles (strawberry or pineapple)	1 container	
LiveActive Digestive Health (Lowfat Plain, with Pineapple, or with Mixed Berries)	1 container	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
MILK:		
Publix Brand		
Fat Free	1 cup	
Low Fat (1% milkfat)	1 cup	
Reduced Fat (2% milkfat)	1 cup	
Whole Milk	1 cup	
Whole Chocolate Milk	1 cup	
Low Fat Chocolate Milk	1 cup	
Greenwise Whole or Reduced Fat Milk	1 cup	
Greenwise Soy- Plain, Chocolate, or Vanilla varieties	1 cup	
Gustafon Farms		
Whole, Skim, 2% Reduced Fat, Chocolate varieties	1 cup	
Smart Balance		
Fat Free, or Lowfat (1%) varieties	1 cup	
Lactose Free	1 cup	
1% Chocolate Milk	1 cup	
Nesquick		
Chocolate Milk, Reduced Fat Chocolate and Strawberry Milk, and Very Vanilla Reduced Fat Milk varie	1 cup	
Strawberry Shake, Chocolate Shake	1 cup	
Extra Calcium Fat Free Chocolate Milk	1 cup	
Lactaid		
Lactose Free (Reduced Fat, Lowfat, Fat Free, Whole, or Lowfat Chocolate varieties)	1 cup	
Meyenberg		
Goat Milk or Lowfat Goat Milk	1 cup	
Silk		
Soymilk-Organic Plain or Light Plain varieties	1 cup	
Soymilk-Vanilla or Light Vanilla varieties	1 cup	
Soymilk-Chocolate or Light Chocolate varieties	1 cup	
Soymilk- Bone Health with Extra Calcium	1 cup	
Soymilk- Omega-3 DHA	1 cup	
Soymilk-Plus Fiber	1 cup	
Soymilk-Vanilla Fortified for Kids	1 cup	
Soymilk Lactose Free Chai	1 cup	
Horizon		
Organic-Whole, Reduced Fat(2%), Lowfat(1%), Fat Free, Chocolate Lowfat, and DHA Omega-3 varie	1 cup	
Organic Valley		
Organic- 2%, 1%, Whole, Fat Free, and Chocolate varieties	1 cup	
Kefir		
Cultured and Lowfat Cultered Milk Smoothies	1 cup	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Eggs:		
Whole Egg (generic)	**1 egg**	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Egg Substitutes:		
Egg Beaters		
Original	1/4 cup	
Cheese and Chive	1/4 cup	
Southwestern/Garden Vegetable flavors	1/4 cup	
Publix		
Eggstirs	1/4 cup	
Hannaford by Sweetbay		
Egg Mates	1/4 cup	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Orange Juice		
Fortified Orange Juice		
Tropicana		
With Calcium/Vitamin D (Lots of Pulp or No Pulp)	8 oz (1 cup)	
Healthy Kids No Pulp	8 oz (1 cup)	
Florida's Natural		
Home Squeezed Style with Calcium and Vitamin D	8 oz (1 cup)	
Minute Maid		
16 Vitamins & Minerals Enhanced Juice	8 oz (1 cup)	
With Calcium and D	8 oz (1 cup)	
Hannaford by Sweetbay		
Plus Calcium and Vitamin D	8 oz (1 cup)	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Yogurt		
Dannon		
DanActive Immunity- Strawberry, Blueberry, Vanilla, and Mixed Berry flavors	1 bottle	
Activia by Dannon- Vanilla, Blueberry, Prune, Strawberry, Cherry, Peach, or Mixed Berry flavors	1 container	
Activia Light- All Flavors	1 container	
Light&Fit Smoothies Carb Control- Strawberry Banana Cream flavor	1 bottle	
Light&Fit Smoothies-Strawberry Banana, Peach Passion, Mixed Berry, or Strawberry flavors	1 bottle	
Light&Fit Lowfat Yogurt- All flavors	1 container	
Light&Fit Family Packs-Strawberry, Banana, Blackberry, or Vanilla flavors	1 container	
Light&Fit Carb Control Yogurt	1 container	
Light&Fit Plus Yogurt	1 container	
Danimals Smoothies-All Flavors	1 bottle	
Danimals Extreme Smoothies	1 bottle	
Frusion Smoothies- Pina Colada, Banana Berry flavors	1 bottle	
Light&Fit 32oz Tubs-Vanilla flavor	1 cup	
Yoplait		
Fiber One Nonfat Yogurt- Strawberry, Peach, Key Lime Pie flavors	1 container	
YoPlus Digestive Health- Peach, Strawberry, Cherry, Raspberry flavors	1 container	
Yoplait Kids 25% Less Sugar-Vanilla, Banana, Peach, Strawberry Banana flavors	1 container	
Trix Yogurt	1 container	
Go-Gurt Portable Lowfat Yogurt	1 tube	
Fizzix-FizzyYogurt Snack	1 tube	
Thick and Creamy Lowfat Yogurt	1 container	
Thick and Creamy Light (all flavors)	1 container	
Yoplait Light (all flavors)	1 container	
Yoplait Original	1 container	
Whips- Light and Fluffy	1 container	
Whips-Chocolate Mousse Style flavors	1 container	
Light Yogurt Smoothie (strawberry banana)	1 container	
Yogurt Smoothie (Strawberry)	1 container	
StoneyField Farm Organic Yogurt		
Lowfat varieties (Blueberry, Peach, Strawberry, Vanilla)	1 container	
Fat Free (all flavors)	1 container	
YoCalcium (wild berry)	1 container	
YoKids (Raspberry, Strawberry Vanilla)	1 container	
Publix Brand		
No Sugar Added Varieites	1 container	
Weight Watchers		
Light Yogurts (all flavors)	1 container	
Breyers		
YoCrunch	1 container	
YoCrunch Light	1 container	
YoCrunch Naturals	1 container	
Light Yogurt-Boosts Immunity (strawberry, blueberry, peach)	1 container	
Hannaford By Sweetbay		
Lowfat Yogurt Smoothies	1 container	
Nonfat Yogurt Smoothies	1 container	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Cereals		
General Mills		
Golden Grahams	3/4 cup	
Wheaties	3/4 cup	
Chex (wheat, strawberry, chocolate, honey nut, bran varieties)	3/4 cup	
Chex (rice and corn varieties)	1 cup	
Cheerios (honey nut, frosted, oat cluster, berry burst, apple cinnamon varieties)	3/4 cup	
Cheerios (original or multigrain varieties)	1 cup	
Total (whole grain or honey cluster varieties)	3/4 cup	
Total (Raisin Bran variety)	1 cup	
Lucky Charms (regular or chocolate)	1 cup	
Reese's Puffs	3/4 cup	
Kix	3/4 cup	
Cinnamon Toast Crunch	3/4 cup	
Cocoa Puffs	3/4 cup	
Cookie Crisp	3/4 cup	
Trix	3/4 cup	
Curves Cereal	3/4 cup	
Basic 4 Cereal	1 cup	
Small Boxes (Lucky Charms, Golden Grahams, Frosted Cheerios, Cocoa Puffs, Cinnamon Toast Crunch, Trix, Honey Nut Cheerios)	1 box	
Small Boxes (Cheerios)	1 box	
Quaker		
King Vitamin	1-1/2 cup	
Kashi		
Vive	1-1/4 cup	
Kellogs		
Raisin Bran	1 cup	
Raisin Bran Crunch	1 cup	
Cocoa Krispies	3/4 cup	
Rice Krispies	1-1/4 cup	
Froot Loops	1 cup	
Froot Loops Reduced Sugar	1-1/4 cup	
Honey Smacks	3/4 cup	
Crispix	1 cup	
All-Bran Wheat Flakes	3/4 cup	
All-Bran Original	1/2 cup	
All-Bran Yogurt Bites	1-1/4 cup	
All-Bran Strawberry Medley	1 cup	
All-Bran Extra Fiber	1/2 cup	
All-Bran Bran Buds	1/3 cup	
Corn Flakes	1 cup	
Frosted Mini-Wheats	24 biscuits	
Unfrosted Mini-Wheats	30 biscuits	
Mueslix	2/3 cup	
Cracklin' Oat Bran	3/4 cup	
Smart Start (Toasted Oat, Maple and Brown Sugar varieties)	1-1/4 cup	
Smart Start (Cinnamon Raisin, and Antioxidant varieties)	1 cup	
Special K (Fruit & Yogurt, Chocolatey Delight, Cinnamon Pecan, and Vanilla Almond varieties)	3/4 cup	
Corn Pops	1 cup	

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month?**
...Cereals:		
Kellogs		
Frosted Flakes	3/4 cup	
Apple Jacks	1 cup	
Grab'N Go Froot Loops	1 pack	
Grab'N Go Pops	1 pack	
Mini Box Frosted Flakes	1 box	
Mini Box Raisin Bran	1 box	
Mini Box Rice Krispies	1 box	
Mini Box Cheerios	1 box	
Mini Box Honey Nut Cheerios	1 box	
Mini Box Apple Jacks	1 box	
Mini Box Cocoa Krispies	1 box	
Wild Animal Crunch	3/4 cup	
Publix Brand		
Toasted Oats	1 cup	
Toasted Oats-Apple Cinnamon	3/4 cup	
Toasted Oats-Triple Berry	1 cup	
Raisin Bran	1 cup	
Crunchy Granola Raisin Bran	1 cup	
Magic Stars Frosted Oat Cereal	3/4 cup	
Crispy Rice Toasted Rice Cereal	1-1/4 cup	
Crispy Corn and Rice	1 cup	
Bran Flakes Enriched Wheat Cereal	3/4 cup	
Fruit Rings	1 cup	
Crunchy Rice and Wheat with Strawberries	1 cup	
Crunchy Rice and Wheat with Strawberries	1 cup	
Almonds and Oats Sweetened Multi-Grain Cereal	3/4 cup	
Honey and Oats Sweetened Multigrain Cereal	3/4 cup	
Frosted Flakes	3/4 cup	
Nutty Nuggets Wheat and Barley Cereal	1/2 cup	
Apple Dapples	1 cup	
Cocoa Crisp	3/4 cup	
Publix Greenwise Organic Cereals		
Honey Nut Toasted Oats	1 cup	
Honey Crunch and Oats	3/4 cup	
Raisin Bran	1 cup	
Toasted Oatmeal Flakes	3/4 cup	
Post	3/4 cup	
Honey Bunches of Oats (Honey Roasted, with Strawberries, Peaches, Almonds, Chocolate Clusters,or Cinnamon Clusters varieties)	3/4 cup	
Honey Bunches of Oats with Vanilla Clusters	1 cup	
Grape Nuts	1/2 cup	
Trail Mix Crunch	1/2 cup	
Live Active (Digestive Health)	1 cup	
Post Selects (Great Grains, Maple Pecan Crunch, Cranberry Almond varieties)	3/4 cup	
Post Selects (Blueberry Morning, Banana Nut Crunch varieties)	1 cup	
Honey Combs	1-1/2 cup	

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
...Cereals:		
...Post		
Cocoa Pebbles or Fruity Pebbles	3/4 cup	
Raisin Bran	3/4 cup	
Golden Crisp	1 cup	
South Beach Living		
Vanilla Almond Crunch	1 cup	
SweetBay Hannaford Cereals		
Crunchy Granola Raisin Bran	1 cup	
Berry Toasted Oats	1 cup	
Apple and Cinnamon Tasteeos	3/4 cup	
Toasted Oats	1 cup	
Honey Nut Tasteeos	3/4 cup	
Raisin Granola	1/2 cup	
Crispy Rice	1-1/4 cup	
Toasted Corn Cereal	1 cup	
Oats and More (with Almonds, with Strawberries, or with Honey)	3/4 cup	
Fruity Krisp	3/4 cup	
Cinnin-Mini Crunch	3/4 cup	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Pasta:		
Spaghetti-O's, w/ meatballs	1 cup	
Spaghetti-O's, w/ sliced franks	1 cup	
Spaghetti-O's, original	1 cup	
Spaghetti-O's, ravioli-o's	1 cup	
Spaghetti-O's, plus Calcium	1 cup	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Starbucks:		
Latte		
Latte	Tall (12oz)	
Latte	Grande (16oz)	
Latte	Venti (20oz)	
Latte, Nonfat	Tall (12oz)	
Latte, Nonfat	Grande (16oz)	
Latte, Nonfat	Venti (20oz)	
Latte, Soy	Tall (12oz)	
Latte, Soy	Grande (16oz)	
Latte, Soy	Venti (20oz)	
Cappucinno		
Cappucinno	Tall (12oz)	
Cappucinno	Grande (16oz)	
Cappucinno	Venti (20oz)	
Cappucinno, Nonfat	Tall (12oz)	
Cappucinno, Nonfat	Grande (16oz)	
Cappucinno, Nonfat	Venti (20oz)	
Cappucinno, Soy	Tall (12oz)	
Cappucinno, Soy	Grande (16oz)	
Cappucinno, Soy	Venti (20oz)	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Fish:		
Herring/Trout, cooked	75 g (about 2.66 oz)	
Salmon-Atlantic, cooked	75 g (about 2.66 oz)	
Salmon-Chinook, Coho, Humpback, Sockeye	75 g (about 2.66 oz)	
Sardines, Atlantic canned	75 g (about 2.66 oz)	
Sardines, Pacific canned	75 g (about 2.66 oz)	
Tuna, canned-Light or White	75 g (about 2.66 oz)	
Tuna, Yellowfin (Albacore, Ahi)	75 g (about 2.66 oz)	
Tuna, Skipjack cooked	75 g (about 2.66 oz)	
Tuna, Bluefin cooked	75 g (about 2.66 oz)	
Mackeral, cooked	75 g (about 2.66 oz)	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Margarine:		
SmartBalance		
Buttery Spread	1 Tbs	
Light Buttery Spread	1 Tbs	
Low Sodium Buttery Spread Whipped	1 Tbs	
Fleischmann's		
Light Margarine	1 Tbs	
Original Margarine	1 Tbs	
Unsalted Margarine	1 Tbs	
Promise Activ		
Buttery Spread	1 Tbs	
Country Crock		
Country Crock Spread plus Vitamins and Minerals	1 Tbs	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Mushrooms:		
Mushrooms, generic (white or portobello)	5 whole mushrooms	
Dole Mega-D sliced portobellos	1 mushroom	
ALL OTHERS...		

Type of food or drink	1 serving	How many **servings** of this food or drink do you have **per month**?
Liver:		
Liver, beef cooked	3.5 oz	
ALL OTHERS...		

Thank you for participating.

Name: _____ Date: _____

Patient number: _____

Directions: In the yellow (shaded) boxes below, write in the number of servings of each of the following foods you eat in a typical week.

Food or Beverage	Reference serving	Number servings per week
"Total"® brand dry cereals (not other brands)	1 cup	
Instant breakfast drinks, shakes, diet shakes, liquid supplements	12 fl oz	
Milk, any kind, including on cereal, in beverages, etc	1 cup	
Yogurt (not frozen)	1 cup	
Calcium-fortified orange juice	1 cup	
Latte, cappucino, frappucino, etc	12 fl oz	
Meal replacement or energy bars	1 med	
Cheese: Swiss, cheddar, provolone, American, others (including on sandwiches and burgers)	1 oz/1 slice	
Sardines or salmon with bones	3 ounces	
Pizza with cheese	1 slice	
Lasagna, etc with cheese	1 cup	
Macaroni and cheese	1 cup	
Taco, burritos, etc, with cheese	1 each	
Soup made with milk	1 cup	
Breakfast bars	1 medium	
Tofu, firm, processed with calcium sulfate	½ cup	
Broccoli, collards, turnip greens, kale, bok choy	½ cup	
Beans: kidney, navy, black, baked, etc	1 cup	
Ice cream, frozen yogurt	½ cup	
Cottage cheese	¾ cup	
Pudding, made with milk	½ cup	
Pancakes, waffles, French toast	2 each	
Other dry cereals (not including *Total®*)	1 cup	
Almonds	¼ cup	
Other calcium-fortified drinks and juices	1 cup	

Have you taken any of the following in the past month?

Vitamin/mineral supplements	Yes ☐	No ☐
Calcium supplements or pills	Yes ☐	No ☐
Tums ®, Rolaids ®, etc.	Yes ☐	No ☐

If yes, complete the following:

Name of product # 1: _____
- Calcium (mg) per dose:
- Average number doses taken per week:
- *Average calcium (mg/day)*

Name of product # 2 _____
- Calcium (mg) per dose:
- Average number doses taken per week:
- *Average calcium (mg/day)*

Name of product # 3: _____
- Calcium (mg) per dose:
- Average number doses taken per week:
- *Average calcium (mg/day)*

For office use only:

Number of servings ____ x 1000 = ☐

Number of servings ____ x 400 = ☐

Number of servings ____ x 300 = ☐

Number of servings ____ x 200 = ☐

Number of servings ____ x 100 = ☐

Subtotal from diet ☐ mg/wk

Divide by 7 to get daily average / 7

Subtotal from diet ☐ mg/day

Miscellaneous from diet [add 200] + 200 mg/day

Daily calcium intake from food ☐ mg/day

Daily calcium intake from suppls + ☐ mg/day

TOTAL DAILY CALCIUM INTAKE ☐ **mg/day**

Short Calcium Questionnaire (version SCQ 2002)
Nutrition Department, NIH Clinical Center
National Institutes of Health, Bethesda, MD 20892-1078 USA

Subject ID Number _____

Sun Exposure Questionnaire

Please circle the number that best describes the time spent outdoors and the amount of skin exposed during that time for the past week.

	Time Outdoors			Amount of Skin Exposed			
	< 5 minutes	5-30 minutes	> 30 minutes	Hands and Face	Hands, Face, and Arms	Hands, Face, and Legs	Bathing Suit
Monday	0	1	2	1	2	3	4
Tuesday	0	1	2	1	2	3	4
Wednesday	0	1	2	1	2	3	4
Thursday	0	1	2	1	2	3	4
Friday	0	1	2	1	2	3	4
Saturday	0	1	2	1	2	3	4
Sunday	0	1	2	1	2	3	4

Ambient UV Exposure

What is your skin type?

Type 1: Fair skinned; always burn never tan
Type 2: Burn easily; hardly tan
Type 3: Sometimes burn; gradually tan
Type 4: Rarely burn; always tan
Type 5: Medium to dark skinned; seldom burn and always tan
Type 6: blue-black skin; never burn and tan darkly

Questionnaire from Hanwell H, Vieth R, Cole D, et al. Sun exposure questionnaire predicts circulating 25-hydroxyvitamin D concentrations in Caucasian hospital workers in Southern Italy. *Journal of Steroid Biochemistry and Molecular Biology*. 2010;In press.

VITA

Rachel Ann Hildebrand

Candidate for the Degree of

Doctor of Philosophy

Thesis: A CROSS-SECTION ANALYSIS OF VITAMIN D STATUS ON MUSCLE STRENGTH AND POWER IN NCAA ATHLETES

Major Field: Health and Human Performance

Biographical:

Education:

Completed the requirements for the Doctor of Philosophy in Health, Leisure and Human Performance at Oklahoma State University, Stillwater, Oklahoma in August, 2013.

Completed the requirements for the Master of Education in Sport Administration at Xavier University, Cincinnati, Ohio in 2009.

Completed the requirements for the Bachelor of Science in Athletic Training at The University of Tulsa, Tulsa, Oklahoma in 2006.

Experience:

2012-	Southern Nazarene University, Bethany, Oklahoma Program Director, Athletic Training Education Program
2010	University of Central Oklahoma, Adjunct Faculty
2009-2010	Oklahoma State University, Graduate Assistant, Certified Athletic Trainer (ATC)
Spring 2009	Xavier University, Athletic Training Assistant, ATC
2006-2009	Xavier University, Athletic Training Assistant, ATC
Summer 2005	RehabWorks Internship, NASA/ Kennedy Space Center, Florida
2002-2006	The University of Tulsa, Student Athletic Trainer

Professional Memberships:

August 2009	Athletic Training License, State of Oklahoma License Number: 569
November 2008	American Red Cross Professional Rescuer Instructor
April 2006	National Athletic Trainers Association BOC Certification Number: 060602201