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RESEARCH ARTICLE

# The Effects Of Vitamin D3 Supplementation On Muscle Physical Performance And Vitamin Status In A Physically Active Population: A Randomized, Placebo-Controlled Blind Intervention Study

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ABSTRACT

Objectives: This study aimed to determine if an eight-week supplementation of 50,000 IU of VitD3 will reverse deficiency in a physically active population.

Patients or Other Participants: A total of 29 physically active collegiate subjects (age =23.21±1.52 yrs, weight =70.66±14.23 kg, height=171.18 ± 9.03 cm) volunteered to participate in the study.

Intervention(s): Before completing pre-testing performance measures and beginning supplementation, venous blood samples to assess baseline serum 25(OH)D levels were collected from the cubital vein.

Main Outcome Measures: Serum 25(OH)D levels and muscle performance metrics (bench press, vertical jump, and 40m sprint) were analyzed to determine differences between pre-test and post-test data of both the control and treatment groups, respectively.

Results: A repeated measures ANOVA showed a significant increase in serum 25(OH)D levels after supplementation ($p < .001$) with a more prominent increase in the treatment group. These analyses indicate that there was no significant difference between the two treatment groups (VitD3 vs. placebo) at pre-treatment measurement, and post-treatment serum 25(OH)D status increased at a greater magnitude for Vitamin D (VitD) compared to the placebo group, who showed a relatively small change in serum 25(OH)D levels. Prior to D3 supplementation 19 of 29 total subjects (66%) had serum 25(OH)D concentrations considered to be deficient (<32 ng/ml).

Conclusions: Total serum 25(OH)D concentration significantly increased and eliminated pathological deficiency in the supplementation group after eight weeks of supplementation with 50,000 IU of VitD3. Muscle performance metrics increased over the treatment period but were not significantly increased between treatment and control.

**Keywords:** Athletic Health, Blood, Muscle Strength, Nutritional Deficiency.

## INTRODUCTION

Many factors affect athletic performance including training, genetics, nutrition, and the environment. Combining these factors can also influence serum 25(OH)D vitamin D (VitD) levels available for use in the body. Vitamin D is a fat-soluble prohormone primarily obtained from ultraviolet radiation, and gained from a small number of food sources.[1–3] In the last twenty years, there has been increasing evidence to show the numerous roles VitD plays throughout the body.[3] While VitD aids in disease prevention and muscle function, a high incidence of VitD deficiency is reported in over 60% of children and 75% of adults in the United States.[1,4] Additionally, recent research has focused on VitD deficiency and supplementation in elderly and young populations.[5,6] Low serum 25(OH)D is connected to many health-related issues including low bone density, cancer, heart disease, and increased risk for infection.[7] The discovery of muscle weakness, generalized pain, and poor muscle tone in children led to research on the supplementation of VitD to reverse these abnormalities.[8] Further studies examined the effects of supplementation on reaction time, balance, and neuromuscular function in older adults, and have found significant improvements in these measurements.[5,6,9]

While extensive research has investigated the effects of VitD on neuromuscular function in older adults. There is limited research on supplementation of VitD to help improve physical performance in athletes.[5,6,10] It has been suggested that serum 25(OH)D may play a role in muscle recovery, but this information is limited in humans thus far.[11] Low serum 25(OH)D concentrations have been related to lower muscle strength and protein metabolism.[12] In Europe, research has determined that many professional or elite athletes across various sports are VitD deficient.[14-16] In the United States, the prevalence of VitD deficiency was examined in over 200 collegiate athletes and found that nearly 70% did not meet adequate levels.[17] Research has shown an increased prevalence of VitD deficiency is present in the elderly and young population and athletic populations.[13,16,17]

To date, little evidence is available on the effects of supplementation on athletes or physically active individuals to determine if $VitD_3$ can improve physical performance, muscle function, and return athletes to a normal serum 25(OH)D level. Supplemented athletes with varying levels of serum 25(OH)D levels and lengths of time found improved serum levels.[17,18] Vitamin D is a fat-soluble vitamin present in foods such as fish, liver, beef or chicken eggs, which contain significant levels of naturally occurring VitD. Vitamin D is found in fortified food sources, including milk, fruit juices, bread and cereals. Interestingly, most humans ingest little natural VitD. A recent systematic review and meta-analysis on randomized controlled studies suggested that despite achieving elevation in serum 25(OH)D levels concentration via supplementation, physical performance in athletes did not improve.[17] The research on the prescribed dose of $VitD_3$ and duration of supplementation improvements in physical performance is unclear. One study on ballet dancers found that isometric strength and vertical jump improved with $VitD_3$ supplementation.[18] However, one investigation found no improvement in physical performance for athletes supplemented with $VitD_3$.[19] These inconsistencies show the lack of evidence on effective $VitD_3$ treatment to improve physical performance in a physically active population. Therefore, this study determines if an eight-week supplementation of 50,000 IU of $VitD_3$ will reverse vitamin deficiency in a young, physically active population. While high dose $VitD_3$ influences increasing serum 25(OH)D, little evidence is available on dosage regime and ability to assess if $VitD_3$ supplementation will increase a physically active person's musculoskeletal function and physical performance ability in a 40-meter (m) sprint, one repetition max (1RM) bench press, and vertical jump.

## METHODS

This study was a double-blinded, randomized controlled trial of two treatment groups, $VitD_3$ (n=14) and control (n=15). Participants were primarily recruited from the university's intercollegiate, intramural or club sport teams. The participant's serum 25(OH)D levels, 40m sprint, vertical jump height, and 1RM bench press were measured pre-supplementation and then again after eight weeks of supplementation.

Twenty-nine physically active collegiate subjects (age $=23.21\pm1.52$ yrs, weight $=70.66\pm14.23$ kg, height$=171.18\pm9.03$ cm) volunteered to participate in the study. All subjects included in the study were physically active for at least six months prior to the study and currently taking part in moderate-intensity exercise for 30 minutes, five

days per week or vigorous activity for 20 minutes, three days per week as determined via activity log. The study was conducted according to the Declaration of Helsinki and the university institutional review board. All subjects were fully familiarized with experimental procedures within this study due to the regular weekly meetings implemented as part of the treatment and monitoring system. Exclusion criteria included a history of musculoskeletal injury, cardiovascular problems, history of diabetes, thyroid dysfunction, or excessive weight loss, currently taking fish oil, a multi-vitamin or $VitD_3$ supplement, which compromise the completion of the study protocol. Subjects who met inclusion criteria contacted the investigator, signed up for a time to participate in the study, and provided contact information. Subjects were randomly assigned to one of two groups (independent of gender) by drawing a number out of an envelope. The participants in this study lived in a warm climate with comfortable temperatures throughout the study period. Ultimately, subjects reported increased time to sun exposure consistently throughout the sample group.

On the first day of the study, subjects arrived at the testing site between 8-10 am and informed of procedures and expectations of the study. Subjects reported to the laboratory wearing exercise clothing and a short-sleeved shirt that exposed their cubital vein for blood testing. In addition, subjects refrained from having caffeine and any dietary/performance enhancer on the days of performance testing. Each subject completed a university institutional review board-approved informed consent form agreeing to participate in the study, a demographic information sheet, a medical history questionnaire, and a VitD consumption questionnaire adopted from Halliday et al[20] before participating. After completing the paperwork, venous blood samples to assess serum 25(OH)D levels were collected from the cubital vein within a two-hour time frame prior to the pre-test. After completing the initial blood testing, subjects were notified to return for pre-testing of performance measures and begin supplementation. Non-fasting venous blood samples were taken from the cubital vein. A state certified phlebotomist drew 8ml of blood in Corvac monoject blood collection tubes (Mansfield, MA). Centrifuging the sample of 25 µl for ten minutes at 3,000 RPM separated the serum 25(OH)D and the serum 25(OH)D stored at -18° Celsius until analysis time. After collecting a venous blood sample, serum 25(OH)D was analyzed using automated chemiluminescence immunoassay technology (Liaison 25-OH VitD total Assay; Diasorin Inc, Saluggia, Vercelli Italy). The test does not differentiate between the serum 25(OH)D metabolites, with sensitivity for serum 25(OH)D set at 7 nmol/L. The intra- and inter-assay coefficients of variation were 4% and 6%, respectively.

Subjects were randomly assigned to the $VitD_3$ treatment group or the control group by drawing a number out of an envelope. The supplementation group took 50,000 IU of $VitD_3$ (BioTech Pharmacia Inc. Fayetteville, AR) once a weeks. The control group took one methylcellulose supplement (Letco Med., Decatur, AL) per week. All supplement and control capsules were similar in size, shape, and color. The subjects maintained their dietary habits or workouts during the eight-week treatment period. The treatment group received 50,000 IU of $VitD_3$, distributed by the principal investigator, one time per week for eight weeks. The control group received identical capsules of methylcellulose distributed by the principal investigator. Subjects performed a five-minute warm-up and were allowed to perform five minutes of stretching of their choice. Baseline measurements of height, weight, age, activity logs, upper body strength (bench press), lower body power (vertical jump) and sprint test were collected for physical performance measures. There was computer-generated randomization of testing for each subject, and the subjects received five minutes of rest between each activity. During weeks two through eight of the investigation, subjects met with the investigator to receive the weekly supplementation. The day after consuming their final supplement ($VitD_3$ or placebo), subjects returned for post-test measures following the same pre-test physical performance testing procedure. Following performance testing, venous blood samples were collected. Serum 25(OH)D levels were collected within a two-hour time frame from the post-test exactly as administered in the pre-test, including the 8-10:00 am time period.

Testing the subject's estimated 1RM of the bench press assessed upper body muscular strength. The subject performed warm-up sets using approximately 50-70% of their estimated 1RM based on previous weightlifting experiences. Subjects estimated their 1RM, and that weight was placed on the bar. The subject laid supine on the bench, had their feet on the ground approximately

shoulder-width apart, have their shoulders and back touching the bench, and placed their hands about shoulder-width apart on the bar. The researcher provided supervision and spotting. The subject performed multiple repetitions until they could no longer perform any more lifts with proper form, then determined using a multiple 1RM conversion chart established by the National Strength and Conditioning Association (NSCA).[21] If the subject completed more than ten repetitions of the weight, they were given a four-minute break before the weight was increased and performed the lift again until fatigue.

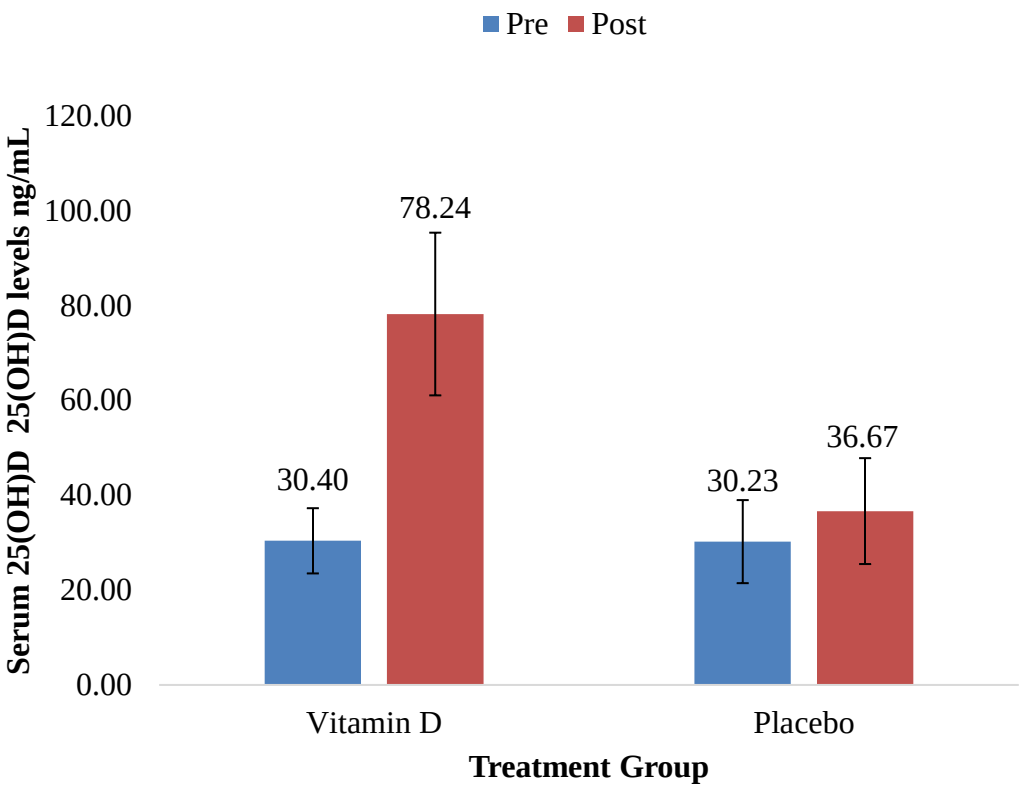
Vertical jump height was measured using a jump and reach method following the standards established by NSCA.[21] The subject was instructed to rub chalk on the tips of the fingers of one hand. The subject stood flat-footed and reached as high as possible for baseline measurement on the Vertec Jump Tester (JumpUSA Corp., Sunnyvale, CA). The Vertec comprises swivel vanes at half-inch increments, and the subject displaces the highest possible vane. The subject then jumped and touched the Vertec at the highest point they could maximally reach. The distance between the top point of the standing touch and the jumping touch was measured. Subjects took 30-second rest between each jump, and the best of three attempts was recorded. The subject ran maximally for 40m on an indoor track, and the time was measured using Bower electronic timing system (Brower, Draper, UT). The automated timing system starts when the subject crosses the starting line and stopped when they cross the finish line. Only one attempt was performed to prevent fatigue, influencing maximal effort and creating an extraneous variable.

Data analysis was performed using the Statistical Package for Social Sciences (v.23, SPSS, Inc., Chicago, IL). Serum 25(OH)D levels (ng/mL) and performance metrics (1RM bench press, vertical jump, and 40m sprint) were analyzed using repeated-measures ANOVAs to determine differences between pre-test and post-test data of both the control and treatment group, respectively. Partial eta squared effect sizes provide a more robust context to the magnitude of change compared to Cohen's D magnitude of the effect.[22]

## RESULTS

Vitamin D Status. Total serum 25(OH)D concentration significantly increased after eight weeks of supplementation with 50,000 IU of $VitD_3$ ($p < 0.001$). Prior to supplementation, 19 of the 29 total subjects (66%) had serum 25(OH)D concentrations considered to be deficient (< 32 ng/mL).[5] The mean serum 25(OH)D level prior to supplementation was $30.1 \pm 7.7$ ng/mL. After $VitD_3$ supplementation, seven out of twenty-nine total subjects were VitD deficient. The mean serum 25(OH)D level prior to treatment was 30.40 ng/mL in the treatment group. Of the subjects receiving $VitD_3$ supplementation, 93% were below optimal 50 ng/mL levels, and 64% were VitD deficient. After eight weeks of 50,000 IU of $VitD_3$, 100% of the participants had reached optimal levels, and the mean serum 25(OH)D concentration was 78.24 ng/mL. In the control group, 10/15 (66%) of the subjects were VitD deficient pre-supplementation, and after eight weeks of control treatment, 47% of the subjects were VitD deficient (Table 1).

Table 1. Mean Serum 25(OH)D Vitamin D Levels




Note: Bars represent standard deviation values; numbers represent group

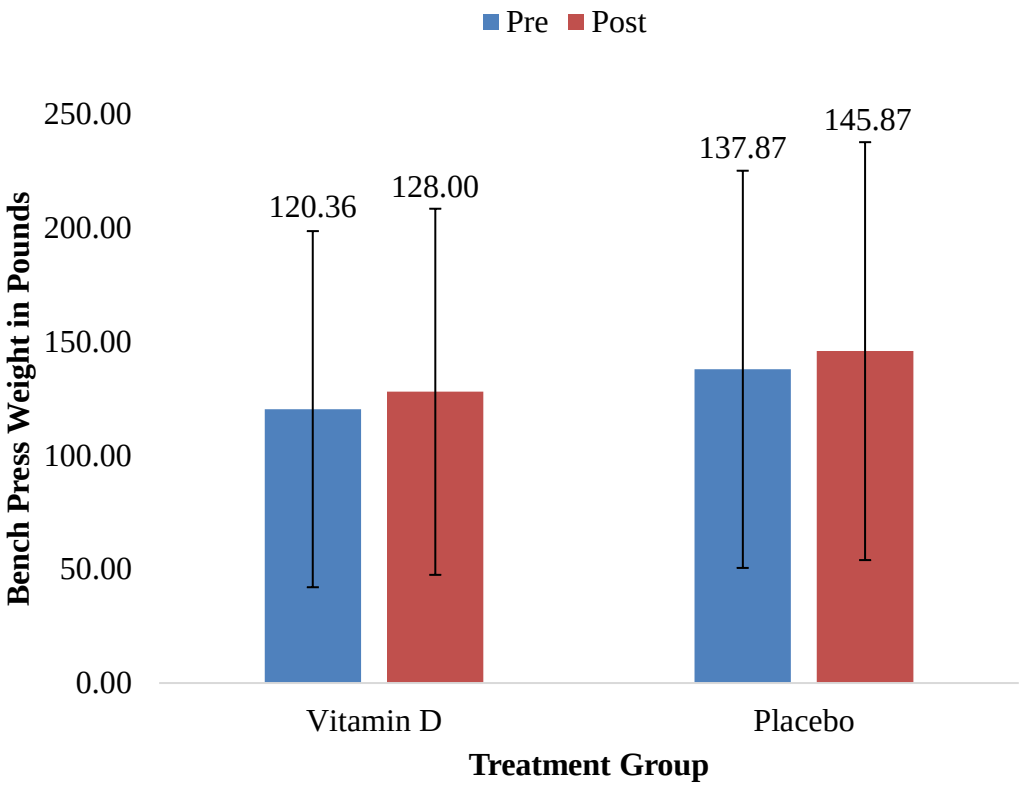
A 2 (between groups) X 2 (pre-and post-treatment serum 25(OH)D levels) repeated measures ANOVA showed a significant increase in serum 25(OH)D levels after supplementation, $F(1, 27) = 196.91$, $p < .001$, *partial* $\eta^2 = 0.88$. Cohen[22] suggests that a small eta-squared value is 0.0099, medium = 0.0588, and large = 0.1379. The eta-squared value is quite large by his conventions of the small, medium, and large. Although the control group increased slightly, $M\Delta = 6.45$ ng/mL±7.11 ($M\%\Delta = 162.48 \pm 49.58$), as expected, there was a larger increase in the treatment group, $M\Delta = 47.84$ ng/mL±13.07 ($M\%\Delta = 23.75 \pm 33.67$). As shown in Table 1, there was a significant treatment group X time interaction, $F(1, 27) = 114.49$, $p < 0.001$, *partial* $\eta^2 = 0.81$. Taken together, these analyses indicate that there was no significant difference between the two treatment groups ($VitD_3$ vs. placebo) in serum 25(OH)D status at pre-treatment measurement, and post-treatment serum 25(OH)D status increased at a greater magnitude for those who received $VitD_3$ compared to the placebo group, who showed a relatively small change in VitD levels.

Performance Metrics. Three separate 2 (treatment group) X 2 (pre- and post-treatment) repeated measures ANOVAs were run on1RM bench press weight, vertical jump in inches, and 40-meter dash time in seconds. Results indicated there were no significant differences between the control group and the $VitD_3$ treatment group in any of the three models, $Fs < 0.32$, $ps > 0.58$, and there were no significant interactions between group and pre-post treatment values, $Fs < 0.60$, $ps > 0.45$. However, from pre- to post-treatment assessments there were significant improvements in bench press, $F(1, 27) = 27.03$, $p < 0.001$, *partial* $\eta^2 = 0.47$, and sprint time, $F(1, 27) = 10.37$, $p = 0.003$, *partial* $\eta^2$

= 0.28, but only a marginally significant change in vertical jump height, vertical jump, $F(1, 27) = 3.77$, $p = 0.06$, *partial* $\eta^2 = 0.12$.

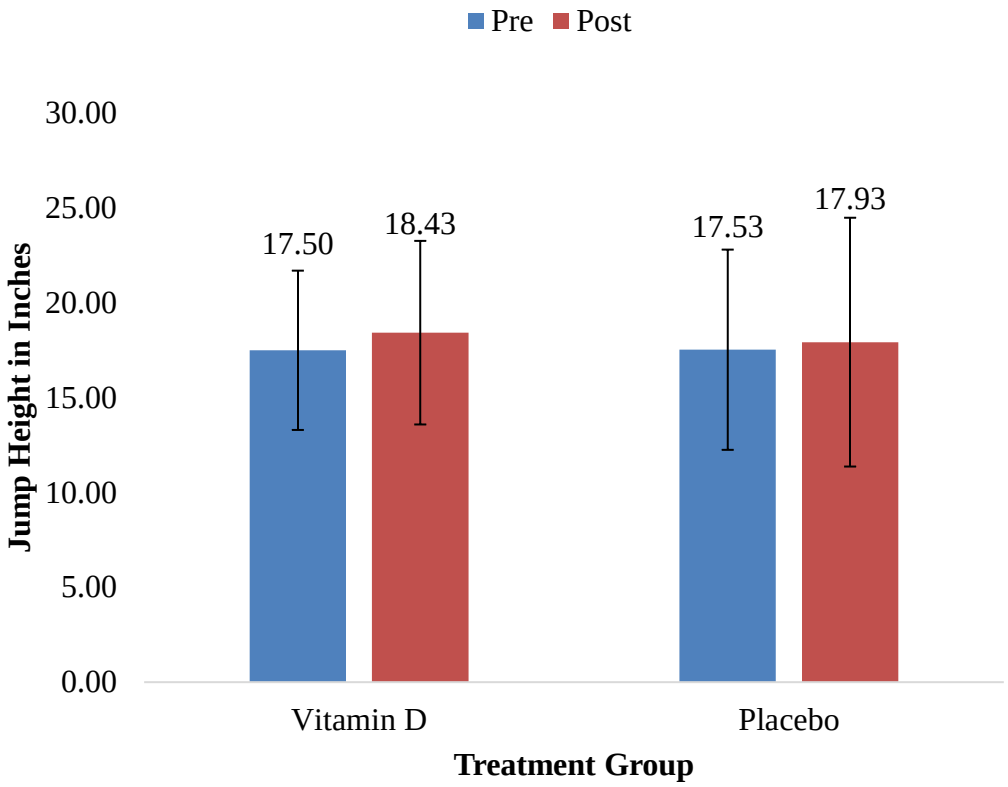
Mean values from the performance tests are shown in Figures 2, 3, and 4. In the treatment group, there was a 6.35% (7.39%±5.76) increase in the bench press, a 5.31% (5.17%±9.53) increase in vertical jump height, and a 2.98% (2.31%±5.86) decrease in sprint time. In the control group, there was a 5.81% (6.86%±8.35) increase in the bench press, a 2.26% (1.02%±10.58) increase in vertical jump height, and a 3.87% (3.55%±4.53) decrease in sprint time (Figure 1, 2, 3, respectively).

Figure 1. Mean 1RM Bench Press



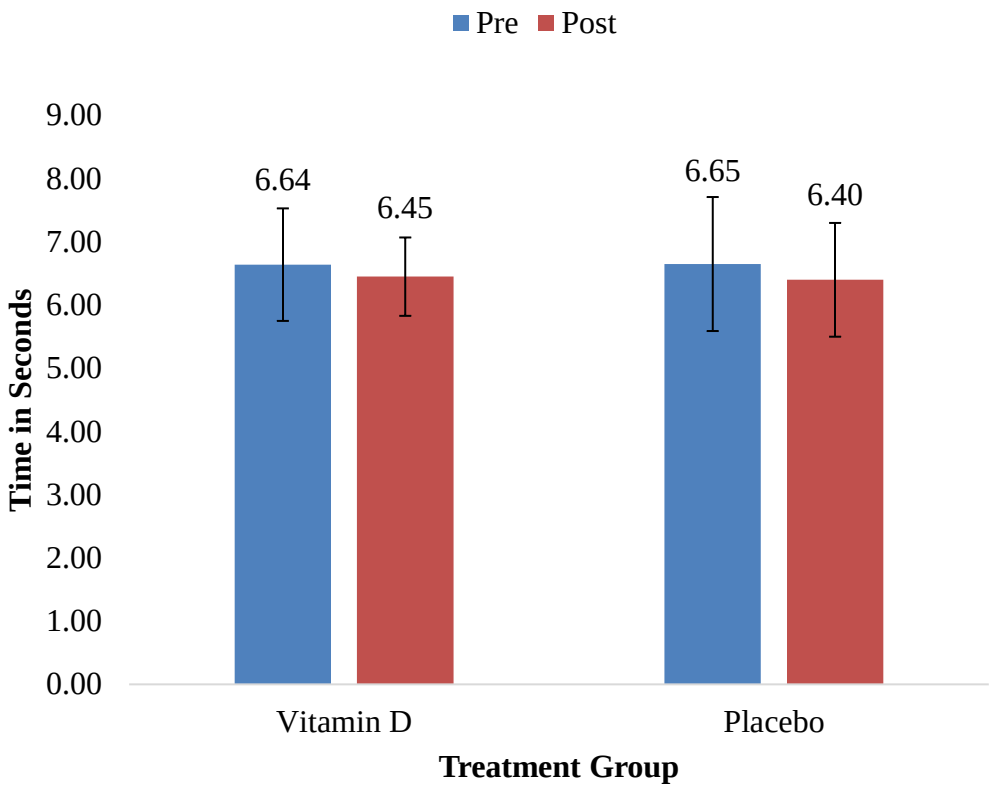

Note: Bars represent standard deviation values; numbers represent group average

Figure 2. Mean Vertical Jump




Note: Bars represent standard deviation values; numbers represent group averages

Figure 3. Mean 40m Sprint




Note: Bars represent standard deviation values; numbers represent group averages

DISCUSSION

This study aimed to examine the effects of 50,000 IU of once-weekly $VitD_3$ on increasing serum 25(OH)D levels and physical performance measures in young, physically active adults. We found that 50,000 IU of $VitD_3$ for eight weeks could bring all subjects to optimal serum 25(OH)D levels (50 ng/ml). However, in this study, VitD3 supplementation had no significant effect on increasing the physical performance measures of vertical jump height, 40m sprint, or 1RM bench press.

Empirical evidence has established VitD deficiency is common in that general and athletic populations.[6,8,14,16,17] In this study, we found that 66% of the physically active subjects were VitD deficient (<32 ng/ml), and 86% were below the optimal level (50ng/ml), further supporting the high incidence of VitD deficiency within a college-age population.[4,20,23] Outside of the United States, similar results have been found in club-level athletes, where 57% of subjects were VitD deficient at baseline.[15] For example, a study conducted by Close et al. found that 62% of the athletes and 73% of the controls were VitD deficient.[2] While the percentage of subjects who were VitD deficient was comparable in most studies; these two studies had a much larger percentage of deficient subjects.[13] Clinical studies indicate examples of ethnicity having a predominance of VitD deficiency

compared to other cultures.[2] For example, one hundred percent of college-aged Asian women and 100% of elite ballet dancers are VitD deficient.[13] The reported mean serum 25(OH)D level at baseline in this study was 30.1±7.7 ng/ml. While the previous investigation found that in healthy Asian females, the mean serum 25(OH)D level was 9.3±3.37 ng/ml and in 215 Norwegian adults between the ages of 18-50, the mean serum 25(OH)D level was 26 nmol/ L (10.4 ng/ml).[24]

Many previous investigations have reported mean values smaller than values found in this study; there were two studies whose mean was larger. Halliday et al[20] found a serum 25(OH)D mean value of 40.1±14.9 ng/ml in college-level athletes, while Division I college athletes reported a mean value of 41.9±14.6 ng/ml. Both studies drew samples in the spring or summer months when athletes spend more time outside in the sun, increasing their UV VitD absorption.[17,20] The more significant numbers reported from our study may also be accounted for under this same reasoning. The participants in this study lived in a warmer climate and reported increased time to sun exposure throughout the sample group. While a lack of sun exposure may account for a large portion of VitD deficiency, nutritional sources should not be ignored. Many American diets lack food significant sources of VitD, particularly dairy products. Most dietary VitD is consumed through dairy cows' milk and fatty fish, which are not a standard part of most American diets over the past 15 years.[8] However, many dairy and cereal products have been fortified with $VitD_3$ to meet dietary needs.[1] It should be considered a concern that the only fortified foods are dairy products leaving those lactose intolerant at an increased risk for VitD deficiency.

Vitamin $D_3$ supplementation is becoming more evident as recent research discovers a growing number of people are VitD deficient, because of the lack of dietary sources and increased use of sunscreen to combat the ill effect of excessive sun exposure. However, there is much concern that current supplementation standards are not adequate.[7,25] The recommended daily dosage of VitD is debatable in determining an optimal serum 25(OH)D concentration, which varies among different organizations and researchers. For example, the National Institute Health (NIH)[26] recommends that VitD levels are adequate when greater than 50 nmol/l (20 ng/ml), whereas the Endocrine Society[27] states that 30-100 ng/ml is sufficient. While the adequate levels of sufficiency vary, toxicity occurs at serum 25(OH)D levels greater than 150 ng/ml.[25]

The 50,000 IU of $VitD_3$ supplemented for eight weeks in this study is higher than most other research investigations in a physically active population. At the end of the eight-week supplementation period, no subjects in the present study were VitD deficient, and all had reached optimal levels greater than 50ng/ml (78.2+17.2). In a similar study, club-level athletes were supplemented with either 20,000 IU or 40,000 IU per week for 12 weeks. At the end of the VitD3 supplementation period, all the subjects were no longer classified as deficient. Only those subjects supplemented with 40,000 IU reached optimal levels.[15] Another study supplemented professional athletes and healthy adults with 5,000 IU once daily for eight weeks, with 60% of the total subjects reaching optimal levels.[2] Overall, research indicates that low dosage VitD supplementation may return subjects to levels that are no longer considered deficient. Higher levels of supplementation may be needed to bring subjects to optimal levels.

This study looked at the effects of $VitD_3$ supplementation on young, physically active adults. No significant improvements were found in 40m sprints, vertical jump height, or 1RM bench press after eight weeks of supplementation with 50,000 IU $VitD_3$ weekly. Previous investigations found improvements in physical performance in isometric measures and shorter (10m) sprint distances.[2] Consistent with the present investigation, Fairbairn et al[28] saw little difference in performance outcomes after 12 weeks of 50,000 IU in professional rugby players. Not only is research on the dosage of VitD insufficient, but the effects of VitD on physical performance measures are controversial. In an elderly population, research has established that $VitD_3$ supplementation can improve neuromuscular function, increase muscle strength, and reduce the risk of falls.[5,6] Muscle biopsies in older, VitD deficient adults have indicated a decreased number of type II muscle fibers and plays a vital role in muscle degeneration allowing for VitD to have a more significant effect on an elderly population than a healthy, young population.[29-30] Therefore, research should look at the effects of VitD supplementation on physical performance in athletes, aging and healthy adults.

This study's significant difference in physical performance may be due to investigative limitations. Practical effects sizes may help show that improvements of 0.19 secs (Vit D group) versus 0.23 secs (placebo group) may have fundamental importance in athletic performance. The difference between winning and losing (1st to 4th) the 100m final at the world champions this year was <0.07 secs (IAAF.org). A limited number of subjects indicated that future research should use a larger sample size to determine the effects of $VitD_3$ supplementation on physical performance. Current dietary intakes were not assessed in the current study for foods such as fatty fish, egg yolks and other fortified foods, which may have influenced current VitD status. Further, a standardized dietary regime and exercise program may have ruled out factors that may have influenced changes in performance or changes in VitD status throughout the eight weeks.

## CONCLUSIONS

In summary, our data questions whether $VitD_3$ supplementation for serum 25(OH)D deficient will adequately affect athletes or physically active individuals' ability to improve physical performance and return/or within normal limits for their serum 25(OH)D levels. Prior to VitD3 supplementation, 19 of the 29 subjects had serum 25(OH)D concentrations considered to be deficient at less than 32 ng/ml (30.1±7.7 ng/ml). After supplementation of $VitD_3$, seven out of 29 total subjects were VitD deficient. The mean serum 25(OH)D level prior to treatment was 30.4 ng/ml (+6.9) in the treatment group. In future research, healthcare practitioners working with athletes who are presented with a serum 25(OH)D deficient athlete must factor in diet, including lactose intolerance and UVB exposure, to supplement that athlete. However, while many individuals supplement with lower doses on the market, a weekly dosage of 50,000 IU of $VitD_3$ supplementation has been shown to return a deficiency of VitD serum 25(OH)D concentrations within normal limits. Any other recommendation for practice should be verified in a visit to their medical provider to have their vitamin levels checked prior to supplementing of any kind.

## PRACTICAL APPLICATION

- In the general world population, over 60% of children and 75% of adults are VitD deficient.
- $VitD_3$ supplementation for serum 25(OH)D deficient will adequately affect a physically active individual's ability to improve physical performance and return/or them within normal limits for their serum 25(OH)D levels.
- Weekly dosage of 50,000 IU of $VitD_3$ supplementation will return a deficiency of VitD serum 25(OH)D concentrations within normal limits.

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Declarations of Interest: None

**REFERENCES**

1 Constantini NW, Arieli R, Chodick G, et al. High prevalence of vitamin d insufficiency in athletes and dancers. *Clin J Sport Med* 2010; 20(5):368–371. Doi: 10.1097/JSM.0b013e3181f207f2.

2 Close GL, Russell J, Cobley JN, et al. Assessment of vitamin d concentration in non-supplemented professional athletes and healthy adults during the winter months in the UK: Implications for skeletal muscle function. *J Sports Sci* 2013; 31(4):344–353. Doi: 10.1080/02640414.2012.733822.

3 Moran DS, McClung JP, Kohen T, et al. Vitamin d and physical performance. *Sport Med* 2013:601–611. Doi: 10.1007/s40279-013-0036-y.

4 Forney LA, Earnest CP, Henagan TM, et al. Vitamin d status, body composition, and fitness measures in college-aged students. *J Strength Cond Res* 2014; 28(3):814–824. Doi: 10.1519/JSC.0b013e3182a35ed0.

5 Bischoff-Ferrari HA, Dietrich T, Orav EJ, et al. Higher 25-hydroxyvitamin d concentrations are associated with better lower-extremity function in both active and inactive persons aged ≥60 y. *Am J Clin Nutr* 2004; 80(3):752–758. Doi: 80/3/752 [pii].

6 Pfeifer M, Begerow B, Minne HW, et al. Vitamin d status, trunk muscle strength, body sway, falls, and fractures among 237 postmenopausal women with osteoporosis. *Exp Clin Endocrinol Diabetes* 2001; 109(2):87–92. Doi: 10.1055/s-2001-14831.

7 Chatterjee S. Vitamin D, Optimal health and athletic performance: A review study. *Int J Nutr Food Sci* 2014; 3(6):526. Doi: 10.11648/j.ijnfs.20140306.16.

8 Bartoszewska M, Kamboj M, Patel DR. Vitamin d, muscle function, and exercise performance. *Pediatr Clin North Am* 2010:849–861. Doi: 10.1016/j.pcl.2010.03.008.

9 Minshull C, Biant LC, Ralston SH, et al. A systematic review of the role of vitamin d on neuromuscular remodelling following exercise and injury. *Calcif Tissue Int* 2016:426–437. Doi: 10.1007/s00223-015-0099-x.

10 Chiang C, Ismaeel A, Griffis RB, et al. Effects of vitamin d supplementation on muscle strength in athletes a systematic review. *J Strength Cond Res* 2016:1. Doi: 10.1519/JSC.0000000000001518.

11 Heaton LE, Davis JK, Rawson ES, et al. Selected in-season nutritional strategies to enhance recovery for team sport athletes: a practical overview. *Sport Med* 2017:2201–2218. Doi: 10.1007/s40279-017-0759-2.

12 Backx E, Van Der Avoort C, Tieland M, et al. Seasonal variation in vitamin d status in elite athletes: A longitudinal study. *Int J Sport Nutr Exerc Metab* 2017; 27(1):6–10. Doi: 10.1123/ijsnem.2016-0177.

13 Wolman R, Wyon MA, Koutedakis Y, et al. Vitamin d status in professional ballet dancers: Winter vs. summer. *J Sci Med Sport* 2013; 16(5):388–391. Doi: 10.1016/j.jsams.2012.12.010.

14 Morton JP, Iqbal Z, Drust B, et al. Seasonal variation in vitamin d status in professional soccer players of the english premier league. *Appl Physiol Nutr Metab* 2012; 37(4):798–802. Doi: 10.1139/h2012-037.

15 Close GL, Leckey J, Patterson M, et al. The effects of vitamin d(3) supplementation on serum total 25[OH]D concentration and physical performance: a randomised dose-response study. *Br J Sports Med* 2013; 47(11):692–696. Doi: 10.1136/bjsports-2012-091735.

16 Magee PJ, Pourshahidi LK, Wallace JMW, et al. Vitamin d status and supplementation in elite Irish athletes. *Int J Sport Nutr Exerc Metab* 2013; 23(5):441–448. Doi: 10.1123/ijsnem.23.5.441.

17 Villacis D, Yi A, Jahn R, et al. Prevalence of abnormal vitamin d levels among division i ncaa athletes. *Sports Health* 2014; 6(4):340–347. Doi: 10.1177/1941738114524517.

18 Lewis RM, Redzic M, Thomas DT. The effects of season-long vitamin d supplementation on collegiate swimmers and divers. *Int J Sport Nutr Exerc Metab* 2013; 23(5):431–440. Doi: 10.1123/ijsnem.23.5.431.

19 Farrokhyar F, Sivakumar G, Savage K, et al. Effects of vitamin d supplementation on serum 25-hydroxyvitamin d concentrations and physical performance in athletes: a systematic review and meta-analysis of randomized controlled trials. *Sport Med* 2017:2323–2339. Doi: 10.1007/s40279-017-0749-4.

20 Halliday TM, Peterson NJ, Thomas JJ, et al.

Vitamin d status relative to diet, lifestyle, injury, and illness in college athletes. *Med Sci Sports Exerc* 2011; 43(2):335–343. Doi: 10.1249/MSS.0b013e3181eb9d4d.

21 Baechle T, Earle R, Wathen D. Essentials of Strength and Conditioning. *Hum Kinet Publ* 2000:395–425. Doi: 10.1080/14763140208522788.

22 Cohen J. Statistical power analysis for the behavioral sciences. *Stat Power Anal Behav Sci* 1988:567. Doi: 10.1234/12345678.

23 Willis KS, Smith DT, Broughton KS, et al. Vitamin d status and biomarkers of inflammation in runners. *Open Access J Sport Med* 2012; 3:35–42. Doi: 10.2147/OAJSM.S31022.

24 Knutsen K V., Madar AA, Lagerløv P, et al. Does vitamin d improve muscle strength in adults? a randomized, double-blind, placebo-controlled trial among ethnic minorities in Norway. *J Clin Endocrinol Metab* 2014; 99(1):194–202. Doi: 10.1210/jc.2013-2647.

25 Larson-Meyer DE, Willis KS. Vitamin d and athletes. *Curr Sports Med Rep* 2010:220–226. Doi: 10.1249/JSR.0b013e3181e7dd45.

26 Ross AC, Manson JE, Abrams SA, et al. The 2011 report on dietary reference intakes for calcium and vitamin d from the Institute of Medicine: What clinicians need to know. *J Clin Endocrinol Metab* 2011; 96(1):53–58. Doi: 10.1210/jc.2010-2704.

27 The Endocrine Society. Evaluation, treatment, and prevention of vitamin d deficiency. *J Clin Endocrinol Metab* 2011; 96(7):1911–1930. Doi: 10.1210/jc.2011-0385.

28 Fairbairn KA, Ceelen IJM, Skeaff CM, et al. Vitamin d3 supplementation does not improve sprint performance in professional rugby players: a randomized, placebo-controlled, double-blind intervention study. *Int J Sport Nutr Exerc Metab* 2018; 28(1):1–9. Doi: 10.1123/ijsnem.2017-0157.

29 Edouard P, Serra J-M, Lafleur M, et al. Prevalence of vitamin d deficiency in high-level athletics athletes. *Ann Phys Rehabil Med* 2016; 59S:e22. Doi: 10.1016/j.rehab.2016.07.053.

30 Grimaldi AS, Parker BA, Capizzi JA, et al. 25(OH) Vitamin d is associated with greater muscle strength in healthy men and women. *Med Sci Sports Exerc* 2013; 45(1):157–162. Doi: 10.1249/MSS.0b013e31826c9a78.