BRIEF REVIEW

Effects of Vitamin D Supplementation on Muscle Strength in Athletes: A Systematic Review

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Abstract

Chiang, C-m, Ismaeel, A, Griffis, RB, and Weems, S. Effects of vitamin D supplementation on muscle strength in athletes: A systematic review. *J Strength Cond Res* 31(2): 566–574, 2017 –The purpose of this systematic review of the literature was to investigate the effects of vitamin D supplementation on muscle strength in athletes. A computerized literature search of 3 databases (PubMed, MEDLINE, and Scopus) was performed. Included in the review were randomized controlled trials (RCTs), published in English, which measured serum vitamin D concentrations and muscle strength in healthy, athletic participants aged 18–45 years. Quality was assessed using the PEDro scale. Five RCTs and 1 controlled trial were identified, and quality assessment showed 5 trials were of "excellent quality" and 1 was of "good quality." Trials lasted from 4 weeks to 6 months and dosages ranged from 600 to 5,000 International Units (IU) per day. Vitamin D2 was found to be ineffective at impacting muscle strength in both studies wherein it was administered. In contrast, vitamin D3 was shown to have a positive impact on muscle strength. In 2 studies, strength outcome measures were significantly improved after supplementation ($p \leq 0.05$). In the other 2 studies administering vitamin D3, there were trends for improved muscle strength. Specifically, improvements in strength ranged from 1.37 to 18.75%. Additional studies are needed to confirm these associations.

Key Words isometric strength, 25-hydroxyvitamin D, skeletal muscle, adolescents, deficiency, exercise

Introduction

Vitamin D, an essential fat-soluble vitamin, is unique because in addition to obtaining it from food sources, the body also synthesizes it from sunlight exposure (23). The 2 primary forms of vitamin D are D2, ergocalciferol and D3, cholecalciferol. Ergocalciferol can be obtained from plant sources, whereas cholecalciferol can be obtained from animal foods and sunlight exposure. In the skin, under solar ultraviolet B radiation, 7-dehydrocholesterol is photoconverted to previtamin D3, which is converted to vitamin D (31). Although the 2 major forms of vitamin D differ in the structure of their side chains, they do not differ in their general metabolism or functions in the body. Vitamin D metabolism converts 2 prohormones, D2 and D3, to the biologically active form, calcitriol (1,25-dihydroxyvitamin D or $1,25[OH]_2D$).

The serum concentration of the active steroid hormone's precursor, 25(OH)D (calcidiol), is the best indicator of vitamin D status because it has a long half-life of 15 days (33,41). In this review, concentrations of 25(OH)D will be expressed in nanogram per milliliter units. Generally, serum 25(OH)D levels are higher than predicted on the basis of vitamin D intake alone because of sunlight exposure. However, the amount of sun needed to meet vitamin D requirements varies; factors that influence its production include skin color, amount of time spent in the sun, weather conditions, latitude and altitude, season, time of day, use of sunscreen, and type of clothing (56). An intake of 1,000 IU of vitamin D per day can raise blood serum levels by approximately 5 $ng \cdot ml^{-1}$ (41). Although the exact levels of 25(OH)D necessary for good health are unknown, levels below 10 $ng \cdot ml^{-1}$ are associated with the most severe deficiency diseases, including rickets in infants and children, and osteomalacia in adults. A concentration above 15 $ng \cdot ml^{-1}$ is generally considered adequate for good health, but hypovitaminosis D, a vitamin D deficiency, is officially diagnosed at values ranging from <12 to <20 $ng \cdot ml^{-1}$ (25,30,37,45,55).

Vitamin D is an important hormone with a wide range of functions. Its primary role is to regulate calcium homeostasis in conjunction with parathyroid hormone, which is necessary for bone development and growth (19). Secondary biological actions of vitamin D metabolites include regulation of immune function and protein synthesis (14,62).

Vitamin D has been estimated to regulate the expression of over 1,000 different genes in the human body, which is 5% of the entire protein-encoding genome (29). A deficiency in vitamin D has been shown to impair muscle action and lead to sarcopenia as well as decreased muscle strength

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(21,22,35,59,60). Additionally, in patients with a clinical vitamin D deficiency and muscle myopathy, symptoms of muscle weakness, muscle fiber atrophy, and reduction in motor unit potentials were reversed when the serum 25 (OH)D levels were normalized back to 20 $ng \cdot ml^{-1}$ or greater (35,47,50).

Vitamin D functions through vitamin D receptors (VDRs), and expression of VDRs has been reported in skeletal muscle (6). The VDRs with vitamin D bound can heterodimerize with a retinoid receptor and enter a cell nucleus, recognize a sequence in the DNA called the vitamin D response element, and determine gene activation or repression (39). Although it is not known whether vitamin D impacts muscle function directly or indirectly, several mechanisms underlying this action have been postulated.

Through the receptors in muscle fibers, vitamin D may control serum calcium concentrations, directly impacting muscle contraction (24). In cultured myoblasts, vitamin D was shown to influence muscle cell calcium uptake (8,46). At the nongenomic level, vitamin D has been shown to activate second messenger pathways that transmit a signal to the cytoplasm, influencing calcium transport and regulating intracellular calcium (13).

Vitamin D is also known to regulate the activation of mitogen-activated protein kinase (MAPK) signaling pathways in muscle, which impacts cell functions including proliferation, gene expression, differentiation, and mitosis (10,53). When activated, these MAPKs regulate cell processes through phosphorylation of other kinases, proteins, and transcription factors. Specifically, vitamin D has been shown to activate the extracellular signal-regulated kinase pathway to stimulate muscle cell proliferation and growth (9,11). An in vivo study in rats suggested that vitamin D may reduce exercise-induced apoptosis in muscle through activation of MAPKs (48). There also might be a direct action on muscles to induce expression of specific genes such as the Ca^{2+}ATPase enzyme, which could result in the division of myoblasts or the differentiation of myotubes (7). This division might explain the observation that vitamin D leads to an increase in type II muscle fiber size (5). Finally, the presence of VDRs in cardiac muscle and vascular tissue, in addition to skeletal muscle, supports the notion that vitamin D may impact the cardiovascular system's ability to transport

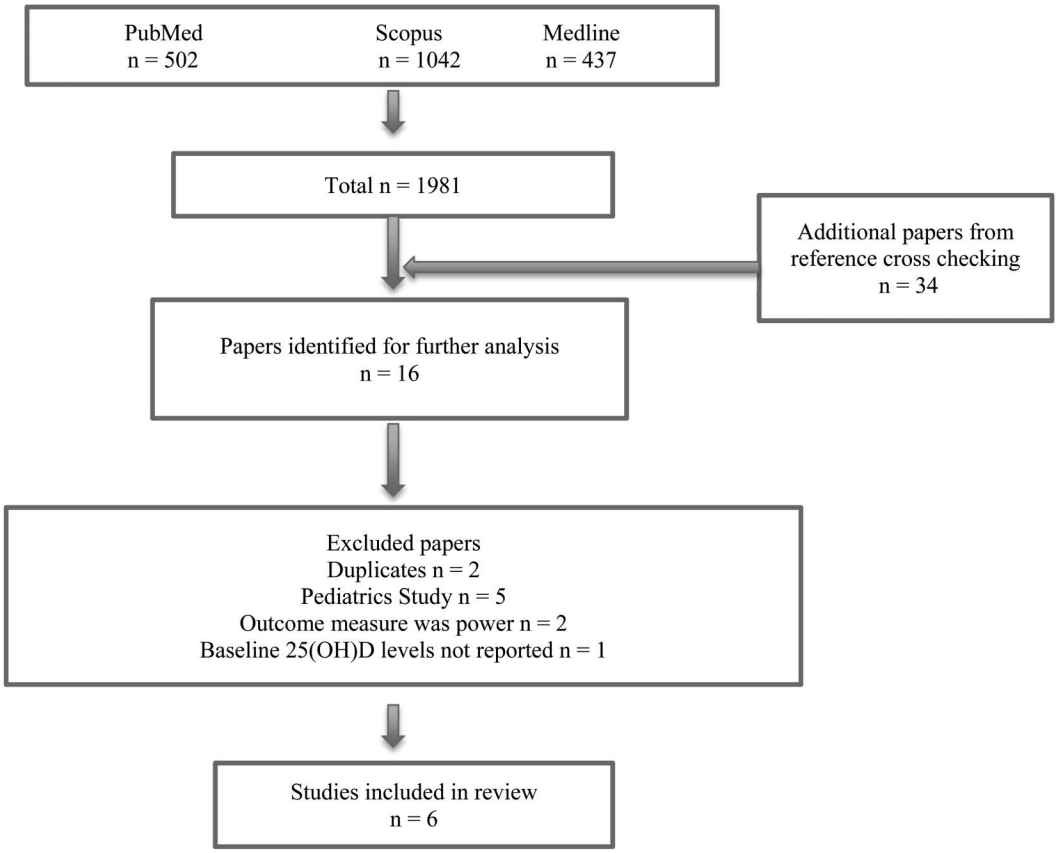

Figure 1. Flow diagram of the search strategy and results.

Table 1. Quality score for eligible studies.*

	PEDro scale items											Total
Author (ref)	1†	2	3	4	5	6	7	8	9	10	11	
Barker et al. (4)	+	+	+	+	+	+	+	+	+	+	+	11
Close et al. (16)	+	+	+	+	+	+	+		+	+	+	10
Close et al. (15)	+	+	+	+	+	+	+		+	+	+	10
Shanely et al. 2014	+	+	+	+	+	+	+	+	+	+	+	11
Nieman et al. (42)	+	+	+	+	+	+	+	+	+	+	+	11
Wyon et al. 2014	+			+				+	+	+	+	6
Total	6	5	5	6	5	5	5	4	6	6	6	Median 10.5

*PEDro Physiotherapy Evidence Database, + the item was clearly satisfied. The PEDro scale is based on the Delphi list developed at the Department of Epidemiology, University of Maastricht. Only criterions 2–11 are scored giving a total of 10: 1 eligibility criteria, 2 randomization, 3 concealed allocation, 4 groups similar at baseline, 5 blinding subjects, 6 blinding therapists, 7 blinding assessors, 8 measures obtained for >85%, 9 intention to treat, 10 between-group statistical comparison, and 11 point measures of variability.
†Column 1 not used in the calculation of the scores.

oxygenated blood and the skeletal muscle's ability to use oxygen (49). This idea is supported by the findings that treating severe 25(OH)D deficiency resulted in improved cardiac muscle function and improved mitochondrial oxidative capacity in skeletal muscle (43,52).

Most of the studies on vitamin D supplementation were limited to diseased populations and elderly adults because of their high risk of vitamin D deficiencies. However, according to recent studies, younger individuals, including many athletes, are also prone to fall into this deficiency category.

Table 2. Study characteristics.*

Study	Sample size (*n*)	Subject description	Subject age (y)	Subject height (cm)	Subject body mass (kg)	Length of intervention	Type of vitamin D (dosage)
Barker et al. (4)	28	Active adult males	30.5 (5.5)	178.0 (6.5)	86.4 (15.5)	35 d	D3 (4,000 IU·d^{-1})
Close et al. (16)	10	English male professional soccer players	18.0 (5.0)	181.0 (5.0)	72.0 (9.0)	8 wk	D3 (5,000 IU·d^{-1})
Close et al. (15)	30	Male club-level athletes	21.33 (1.3)	182 (6.0)	79.0 (9.67)	12 wk	D3 (20,000 IU or 40,000 IU·wk^{-1})
Shanely et al. 2014	33	Male high-school athletes	16.25 (0.26)	176 (2.0)	74.95 (4.28)	6 wk	D2 (600 IU·d^{-1})
Nieman et al. (42)	28	NASCAR pit crew athletes	38.8 (1.9)	NR	NR	6 wk	D2 (3,800 IU·d^{-1})
Wyon et al. 2014	24	Male and female elite ballet dancers	Men: 28.82 (5.13); women: 27.62 (3.77)	Men: 177.0 (7.0); women: 164.0 (3.0)	Men: 65.93 (9.27); women: 49.65 (6.19)	4 mo	D3 (2,000 IU·d^{-1})

*All data are mean (*SD*); NASCAR = the National Association for Stock Car Auto Racing; NR = not reported.

Table 3. 25(OH)D concentrations.*

Study	Baseline 25 (OH)D concentrations ($ng \cdot ml^{-1}$)	Postsupplementation 25(OH)D concentrations ($ng \cdot ml^{-1}$)	Δ25(OH)D	*p* for Δ25(OH)D in supplement group
Barker et al. (4)	30.8 (1.6)	Supplement: 47.9 (7.6); control: 29.1 (7.9)	Supplement: 17.1; control: −1.7	<0.05†
Close et al. (16)	16.43 (10.81)	Supplement: 41.27 (10.0); control: 29.65 (9.62)	Supplement: 24.84; control: 13.22	0.0028†
Close et al. (15)	20.43 (9.62)	Supplement: 20,000 IU: 34.05 (4.01); 40,000 IU: 36.46 (9.62); control: 16.43 (8.81)	Supplement: 20,000 IU: 13.62 40,000 IU: 16.03; control: −4.0	<0.05† (for both 20,000 IU and 40,000 IU)
Shanely et al. 2014	20.7 (1.1)	NR	NR	0.004†
Nieman et al. (42)	38.8 (1.91)	Supplement: 37.4 (1.9); control: 38.6 (1.8)	Supplement: −1.4; control: −0.2	0.127
Wyon et al. 2014	14.4 (4.85)	Not measured	N/A	N/A

*All data are mean (SD); NR = not reported; N/A = not applicable; Δ25(OH)D = postsupplementation concentrations-baseline concentrations.
†Indicates significant increase ($p \leq 0.05$).

Over 77% of the general population may be considered vitamin D deficient, and 1 study showed that up to 80% of athletes may have insufficient levels of vitamin D (18,44). Athletes may also need higher amounts of vitamin D because of higher calcium demands and their typically lean body masses, because adipose tissue is the major storage site for vitamin D (44). In athletes, a vitamin D deficiency decreases skeletal muscle function, recovery time from training, both force and power production, and testosterone production (17).

In this review, the discussion of vitamin D will primarily aim to identify the effect of vitamin D supplementation on an athlete's muscular strength. The definition of an athlete includes any individual who is regularly active, ranging from the fitness enthusiast to the competitive professional. Strength refers to the ability to generate maximal force, where speed of movement is irrelevant (20).

Methods

Experimental Approach to the Problem

This review of the literature was performed by searching 3 computerized databases for peer-reviewed articles published in English since 2010. The most recent systematic review of the literature concerning vitamin D and muscle strength was published in 2015, and this review searched studies with publication dates after 2000. The research articles were searched for and selected from electronic databases, including MEDLINE (EBSCO), Scopus, and PubMed. These databases were searched using the terms vitamin D OR vitamin D supplementation OR vitamin D2 OR vitamin D3 OR 1-alpha hydroxyvitamin D3 OR 1-alpha hydroxycalciferol OR 1,25-dihydroxyvitamin D3 OR 1,25 dihydroxycholecalciferol OR 25 hydroxycholecalciferol OR 25-hydroxyvitamin D OR calcitriol OR ergocalciferol OR cholecalciferol or calcifediol or alfa-calcidol or calcidiol or calciferol AND supplementation OR supplement AND muscle OR muscle function OR muscle strength OR force and power performance OR athletic performance.

Subjects

Inclusion criteria for studies were those that used a placebo-controlled approach where at least 1 arm of the experimental design included vitamin D supplementation of any form or any dose. Studies were also required to have incorporated some measure of muscle strength as a primary outcome measure on adult-aged athletic participants (18–45 years). Finally, included studies were those that provided baseline values of vitamin D (25[OH]D) levels. Exclusion was based on studies involving participants outside the chosen age range and those that incorporated cell or nonhuman animal models. Studies that measured any muscular function parameters other than strength were excluded as well. Finally, studies that incorporated any population considered to be nonhealthy were also excluded.

The initial search produced 502 citations on PubMed, 437 on MEDLINE, and 1,042 on Scopus. After screening for article type, text availability, publication date, language, and age of subjects, 16 articles underwent full-text review, after which 10 articles were excluded (Figure 1). Included articles report studies conducted with randomized and placebo-controlled trials. Vitamin D supplementation interventions in the studies were conducted with different dosages.

Table 4. Strength outcome measures.*†

Study	Strength outcome measure	Baseline strength	Postsupplementation strength	Δ strength (% change)	*p* for Δ strength
Barker et al. (4)	Recovery (%) in single-leg peak isometric LP force after damaging event	N/A	Supplement: 18%; control: 10%	N/A	<0.05‡
Close et al. (16)	1RM BP, 1 1RM BS	BP: 81.88 (14) kg; BS: 146.25 (12.2) kg	Supplement: BP: 88.38 (14) kg; BS: 155.25 (12.2) kg; control: BP: 84.38 (14) kg; BS: 149.25 (12.2) kg	Supplement: BP: 6.5 kg (7.94%); BS: 9.0 kg (6.15%); control; BP: 2.5 kg (3.05%); BS: 3.0 kg (2.05%)	BP: 0.065; BS: 0.094
Close et al. (15)	1RM BP, 1RM LP	BP: 86.67 (17.33) kg; LP: 195.33 (44.33) kg	Supplement: 20,000 IU: BP: 92.0 (15.0) kg; LP: 198.0 (28.0) kg; 40,000 IU: BP: 90.0 (20.0) kg; LP: 198.0 (63.0) kg; control: BP: 79.0 (18.0) kg; LP: 181.0 (43.0) kg	Supplement: 20,000 IU: BP: 5.33 kg (6.15%); LP: 2.67 kg (1.37%); 40,000 IU: BP: 3.33 kg (3.84%); LP: 2.67 kg (1.37%); control: BP: −7.67 kg (−8.85%); LP: −14.33 kg (−7.34%)	BP: 0.17; LP: 0.18
Shanely et al. 2014	LBD	5.14 (0.224) kg/kg§	Supplement: 5.01 (0.151) kg/kg§; control: 5.33 (0.217) kg/kg§	Supplement: −0.13 (−2.53%); control: 0.19 (3.70%)	0.466
Nieman et al. (42)	LBD, HGD, and BWBP	LBD: 188.5 (6.25) kg; HGD: 48.8 (2.05) kg; BWBP: 16.55 (1.5) reps	Supplement: LBD: 200 (5.7) kg; HGD: 53.7 (2.1) kg; BWBP: 17.3 (1.4) reps; control: LBD: 218 (8.6) kg; HGD: 48.3 (2.4) kg; BWBP: 16.5 (1.5) reps	Supplement: LBD: 11.5 kg (6.10%); HGD: 4.9 kg (10.04%); BWBP: 0.75 reps (4.53%); control: LBD: 29.5 kg (15.65%); HGD: −0.5 kg (−1.02%); BWBP: −0.05 reps (−0.30%)	LBD: 0.133; HGD: 0.208; BWBP: 0.083
Wyon et al. 2014	Quadriceps contraction	Supplement: 692.1 (363.7) N; control: 565.1 (253.1) N	Supplement: 821.9 (365.9) N; control: 566.9 (224.8) N	Supplement: 129.8 N (18.75%); control: 1.8 N (0.32%)	0.030‡

*LP = leg press; 1RM = 1 repetition maximum; BP = bench press; BS = back squat; LBD = leg-back dynamometer; HGD = hand grip dynamometer; N/A = not applicable; BWBP = body weight bench press; Δ strength, postsupplementation strength-baseline strength.
†*p*-value represents the condition (control vs. vitamin D) × time interaction effects.
‡Indicates significant difference ($p \leq 0.05$).
§kg/kg = deadlift strength normalized to body mass and reported as kg deadlift/kg body mass.

The primary aim was to identify randomized controlled trials (RCTs) of athletes in which a vitamin D supplementation intervention or noninterventional control group was used. The secondary aim was to find all studies in which athletes' muscular function outcome of strength was tested.

The PEDro checklist was used to assess the quality of the included studies (40). The PEDro scores are assigned of a total of 11: A study receiving a score between 9 and 11 is regarded as excellent, from 6 to 8 is good, between 4 and 5 is fair, and any study graded less than 4 is considered a study of poor quality (40). Five of the 6 studies were considered "excellent," and 1 study was considered "good." Refer to Table 1 for the quality scoring.

Results

Four of the studies (4,15,16,61) administered cholecalciferol (vitamin D3); the other 2 studies (42,51) administered ergocalciferol (vitamin D2). The length of the studies ranged from 6 weeks to 4 months. The supplement dosage also varied from study to study, ranging from 400 to 8,500 IU per day. Table 2 outlines the characteristics of each study.

All 6 studies reported subjects' baseline vitamin D serum levels (Table 3). The mean concentration of serum 25(OH)D was calculated to be 23.59 $ng \cdot ml^{-1}$ for all participants across all studies before supplementation. There seems to be no consensus among researchers regarding the level of 25(OH)D required for optimal health. For example, the Endocrine Society defines a vitamin D deficiency as a 25(OH)D serum level below 20 $ng \cdot ml^{-1}$ and an insufficiency as 21–29 $ng \cdot ml^{-1}$ (29). However, the Institute of Medicine considers a 25(OH)D concentration of 12–20 $ng \cdot ml^{-1}$ to be inadequate, a concentration of 20 $ng \cdot ml^{-1}$ or above to be adequate, and a concentration higher than 50 $ng \cdot ml^{-1}$ to be linked to potential adverse effects. The Institute of Medicine additionally sets the upper limit (UL) at 4,000 IU per day. However, the Endocrine Society sets the UL at 10,000 IU for adults (29).

The protocols used to measure muscular strength were also inconsistent in the studies included in this review. A 1 repetition maximum (1RM), the maximum amount of weight that can be lifted once for a specific exercise, is considered the gold standard for evaluating strength (38). Isokinetic dynamometry is often preferred because the dynamometer only allows movement to occur at a predetermined velocity, and the only variable condition is force. Additionally, there are normative data for a wide range of persons, including athletes, and values can be compared with these norms. However, although the velocity of movement is uncontrolled with free weights and machines, these methods are often preferred because they are more applicable to actual athletic movement. In contrast, isokinetic assessment bears little resemblance to the accelerative/decelerative motion implicit in limb movement during resistance training and sporting performance (38).

In the studies included in this review, 1 (16) used 1RM bench press (BP) and 1RM back squat (BS) testing using free weights according to the standard protocol (3). Another study (15) used the same protocol for 1RM BP testing, but added a 1RM leg press (LP) test using an isokinetic dynamometer as well. Shanely et al. (51) and Nieman et al. (42) assessed leg-back deadlift strength with a standard isometric leg-back dynamometer (LBD). In addition, Nieman et al. used a hand grip dynamometer (HGD) to measure maximum grip strength, where subjects assumed a slightly bent forward position with their dominant hand hanging down and forward while gripping the HGD maximally for 2–3 seconds. Subjects also bench pressed a weighted bar equal to their body weight as many times as possible to a metronome set at 60 $b \cdot min^{-1}$, or 30 lifts per minute, until fatigued. Wyon et al. (61) used a 5-second isometric quadriceps contraction (QC) as a measure of the muscle strength of the dominant leg, where the signal was processed by a data acquisition system. Finally, instead of measuring muscle strength specifically, Barker et al. (4) tested the influence of vitamin D on strength recovery after intense exercise that induces persistent deficit in peak isometric force. A randomly selected leg performed an exercise protocol consisting of 10 sets of 10 repetitive eccentric-concentric jumps on a custom horizontal plyo-press at 75% of body mass to induce muscle damage. Then single-leg strength testing was performed using a 3-second peak isometric QC. Table 4 outlines the strength outcome measurements of each study.

Discussion

The purpose of this review was to evaluate the effects of vitamin D supplementation on muscle strength in healthy athletes aged 18–45 years. Two studies (4,61) reported significant improvements in the muscular strength outcome measure with vitamin D3 supplementation. The study by Wyon et al. (61) was conducted in professional male and female elite ballet dancers. Before supplementation, the mean serum concentration of 25(OH)D of 14.4 $ng \cdot ml^{-1}$ was classified as insufficient according to the Endocrine Society's guidelines. Over the course of 4 months, 2,000 IU of vitamin D3 was administered to the experimental group daily. Isometric QC strength increased in the intervention group by 18.75%, which was a significant increase compared with controls ($p = 0.030$). In contrast, the control group displayed a very negligible (0.32%) increase in QC strength from baseline to the end of the study. A notable strength of the study was that confounding variables such as current nutritional intake were controlled for, and any athletes who were taking a multivitamin, a vitamin D, or fish oil supplement, tanned regularly, or had just returned from a vacation in a sunlight-enriched climate were excluded. A major weakness of the study, however, was that vitamin D levels were not measured at the end of the study. Additionally, the study by Barker et al. (4) also reported a significant, positive effect of vitamin D3 supplementation on muscle strength. At baseline, about half of the subjects displayed

insufficient or deficient vitamin D levels according to the Endocrine Society's guidelines (<29 ng·ml^{-1}), and the mean concentration of 25(OH)D was 30.8 ng·ml^{-1}. After supplementation, the experimental group displayed optimal levels of vitamin D (>29 ng·ml^{-1}), with mean 25(OH)D concentrations rising to 47.9 ng·ml^{-1}. The significant improvement in vitamin D status ($p \leq 0.05$) in the experimental group was accompanied by a concomitant enhancement in isometric LP force recovery after intense exercise. Specifically, the supplementation group showed an 8% higher recovery in strength compared with the control group ($p \leq 0.05$) after the damaging event. Interestingly, vitamin D supplementation also significantly attenuated the increase in alanine transaminase and aspartate transaminase, biomarkers representative of muscle damage after exercise, in comparison with the control group ($p \leq 0.05$).

In the other 2 studies in which vitamin D3 was administered, subjects in the supplementation groups also exhibited improvements in muscle strength, albeit not significant at the 5% level (15,16). The first study (16) was conducted in professional soccer players, and the subsequent one in club-level athletes (15). It is important to note that the first study is limited by the small sample size ($n = 10$). The baseline 25(OH)D concentrations were 16.43 and 20.43 ng·ml^{-1} in each of the studies, respectively. These values are representative of poor vitamin D status, and are categorized as deficient and insufficient according to the Endocrine Society's guidelines. In both studies, vitamin D supplementation led to significant improvements in vitamin D status ($p < 0.003$ and $p \leq 0.05$, respectively), with subjects' mean 25(OH)D concentrations increasing to above 34 ng·ml^{-1}. In addition, there were trends for enhanced strength in the supplementation groups of both studies compared with controls. In the first study (16), for example, 1RM BP increased by an average of 6.5 kg ($p = 0.065$ vs. controls) and 1RM BS increased by an average of 9.0 kg ($p = 0.094$ vs. controls) in the experimental group. In the other study (15), 1RM BP and 1RM LP were not as significantly increased after supplementation, but interestingly, the control groups actually showed an 8.85% decrease in BP strength and a 7.34% decrease in LP strength. Notably, in addition to measuring muscle strength, Close et al. (16) assessed 10-meter sprint times and vertical jump height. For these musculoskeletal performance measures, the supplementation group displayed significant improvements compared with the placebo group ($p = 0.008$ for both measures).

In the 2 studies wherein vitamin D2 was administered, supplementation had no effects on muscle strength (42,51). Muscle strength was not different between supplementation and placebo groups for any of the outcome measures tested. Both studies reported significant increases in 25(OH)D2 and significant decreases in 25(OH)D3, which resulted in either no significant changes in total 25(OH)D levels or very small increases in this concentration. This result is consistent with evidence that the entry of vitamin D2 into the total body pool of vitamin D dilutes the relative amount of vitamin D3. Stephensen et al. (54) similarly showed vitamin D2 supplementation had no effect on vitamin D status; whereas levels of 25(OH)D2 were increased, concentrations of 25(OH)D3 were decreased proportionally. This result is further evidence that vitamins D2 and D3 perhaps should not be regarded as equivalent despite being traditionally recognized as interchangeable. For example, vitamin D inactivation enzymes such as CYP3A4 may break down D2 at a significantly faster rate than vitamin D3, limiting vitamin D2's action in cells where it is expressed (36). Several experts now suggest that vitamin D2 is in fact much less effective than vitamin D3 in humans (2,28,34,57).

The failure to identify significant improvements in muscle strength in some of the studies where total serum vitamin D levels were increased may be due to the achieved endpoint concentrations still not being optimal for muscle function, despite being sufficient based on the Endocrine Society's guidelines (≥30 ng·ml^{-1}). It has been proposed that although these values have been shown to improve physiological function and disease prevention, the response curves of different tissues to hormones are not the same (27). Specifically, although these concentrations may be sufficient for a response in other tissues, a higher serum total 25(OH)D concentration is most likely necessary in skeletal muscle. Cannell et al. (12) suggest that peak athletic performance may occur when 25(OH)D levels approach the levels that are reached with natural, full-body summer sun exposure, or at least 50 ng·ml^{-1}. Researchers often argue that human physiology is fine-tuned to vitamin levels that prevailed during human evolution. For vitamin D, these levels would coincide with a 25(OH)D concentration between 40 and 80 ng·ml^{-1} (32,34,58). Therefore, a problem associated with the effect of vitamin D on muscle strength is the lack of consensus on the optimal serum ranges. The discrepancy may be further evidence of the triage theory of micronutrients (1). This theory suggests that some functions of micronutrients are restricted during shortages, and thus functions required for short-term survival take precedence over less essential functions. Given the plethora of roles vitamin D plays in the body, muscle strength may be limited for more important functions of the vitamin to be performed when serum levels are inadequate. This possibility is further supported by the finding that cholecalciferol does not begin to be stored in fat and muscle tissue for future use until 25(OH)D levels reach 50 ng·ml^{-1} (26,32). At lower concentrations, the body diverts most or all of the ingested or sun-derived vitamin D to immediate metabolic needs, which represents a state of chronic substrate starvation. Therefore, although some reference values are set at the levels associated with clinical symptoms, these values may still be lower than optimal despite the lack of any overt symptoms or pathology.

In one study in this review where baseline 25(OH)D concentrations were extremely low (14.4 ng·ml^{-1}), supplementation did result in a significant improvement in the muscle strength of elite ballet dancers (61). Additionally, in the 2 studies in which 25(OH)D concentrations reached levels

above 40 ng·ml^{-1}, there were also great improvements in musculoskeletal performance after supplementation (4,15).

One issue with the studies included in the review is that only 1 (61) recruited both men and women. In that particular study, significant improvements of up to 31% in QC strength were reported. Because women tend to have lower levels of 25(OH)D (41), future studies should include female athletes in assessments of vitamin D supplementation effects on muscle strength. In addition, there were great variations in outcome measurements used in the studies included in this review. This situation might affect the reliability and accuracy of the results.

Finally, it is important to note that the number of studies was limited, which suggests the importance for additional research. These studies should be designed as RCTs, establish an optimal dosing regimen, control for sex differences, consider the effects in larger athletic populations, monitor toxicity symptoms, use standard measurements for assessing vitamin D status, and use standard measurements for measuring muscle strength.

Practical Applications

Vitamin D2 seems to be ineffective at impacting muscle strength in athletes. In contrast, vitamin D3 supplementation may lead to improvements on muscle function parameters. In all the studies wherein vitamin D3 was administered, there were either trends for improved muscle strength or significant improvements in the outcome measure. Specifically, in one study, supplementation resulted in a ~19% increase in QC strength. Even in the studies not reporting significant increases, improvements in muscle strength were as high as ~8%, with mean 1RM bench presses and BS increasing by an average of 6.5 and 9.0 kg, respectively. Because of the limited amount of research assessing the impact of vitamin D supplementation on muscle strength in athletes, further studies are necessary to confirm these associations.

Coaches, sports dietitians, trainers, and medical staff should monitor athletes' serum levels of vitamin D throughout the year, especially during winter months. Specifically, it seems that serum concentrations of 25(OH)D should be raised over 40 ng·ml^{-1} for optimal muscular performance. Routine testing for adequacy should be incorporated into sports teams' programs. If an athlete displays a 25(OH)D concentration below 20 ng·ml^{-1}, he or she should be put on a sufficient dosage of at least 2,000 IU per day of vitamin D3 for 8–12 weeks or until levels are normalized.

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