REVIEW

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Vitamin D and muscle health: insights from recent studies

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Purpose of review
The purpose of this review is to critically evaluate the effects of vitamin D on muscle mass and physical/muscle function in middle-aged and older adults, based on recent human studies, including cross-sectional, observational, and intervention studies. Vitamin D, beyond its well established role in bone health, has shown potential in influencing muscle physiology, making it a nutrient of interest in the context of sarcopenia and related chronic conditions.

Recent findings
The review states how vitamin D affects muscle function, emphasizing its role in muscle cell proliferation, differentiation, and key signaling pathways. Additionally, the review of recent human studies revealed an inconsistent relationship between vitamin D and sarcopenia and related indices, with mixed results regarding muscle mass and strength. Variability in supplementation dose, duration, and baseline 25-hydroxyvitamin D levels may contribute to these inconsistencies.

Summary
While animal studies indicate vitamin D's effectiveness in muscle growth, cross-sectional, observational, and intervention studies do not show clear benefits of maintaining efficient vitamin D levels on muscle mass or function in humans. Although vitamin D impacts muscle health, it is insufficient alone, emphasizing the need for a multifaceted approach to sarcopenia prevention and management.

Keywords
muscle mass, muscle strength, physical function, sarcopenia, vitamin D

INTRODUCTION

Vitamin D, a crucial nutrient for maintaining bone health, has garnered significant attention for its potential impact on muscle mass and function. Given the rising interest in optimizing muscle health to combat sarcopenia and its associated chronic diseases (i.e. type 2 diabetes and cardiovascular diseases), an updated, evidence-based review is essential. This study aims to explore the latest findings on vitamin D status and its effects on skeletal muscle in middle-aged and older individuals. By synthesizing recent clinical trials and studies, we seek to provide a comprehensive understanding of vitamin D's role in muscle physiology, offering insights that could inform clinical practices and public health strategies to improve muscle health across populations.

Vitamin D metabolism

Vitamin D is a fat-soluble nutrient primarily obtained through sunlight exposure and to a lesser extent from dietary sources and supplements. In humans, vitamin D exists in two primary forms: vitamin D_2 (ergocalciferol), derived from plant sources, and vitamin D_3 (cholecalciferol), synthesized in the skin through sun exposure and also found in animal-based foods. Once ingested or produced, vitamin D_2/D_3 is metabolized in the liver to 25-hydroxyvitamin D (25(OH)D), the main circulating form, by cytochrome P450 oxidases,

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KEY POINTS

- Vitamin D affects muscle cell proliferation, differentiation, and calcium homeostasis, which are critical for muscle function.
- Cross-sectional, longitudinal and intervention studies present mixed findings regarding the correlation between vitamin D intake or serum 25(OH)D levels and muscle mass, strength, and physical performance.
- While vitamin D plays a role in muscle health, it is insufficient alone to prevent sarcopenia.

primarily CYP2R1. This compound is then further hydroxylated in the kidneys to its active form, 1,25-dihydroxyvitamin D ($1,25(OH)_2D$) by 25-hydroxylase (CYP27B1) [1].

Role of vitamin D in the modulation of skeletal muscle mass and function

$1,25(OH)_2D$ works with the vitamin D receptor (VDR) to induce both genomic and nongenomic actions in skeletal muscle. Genomically, $1,25(OH)_2D$ binds to VDR, which then forms a complex with the retinoid X receptor (RXR) and modulates gene expression by binding to vitamin D response elements (VDREs) in DNA. Nongenomically, $1,25(OH)_2D$ influences intracellular signaling pathways, including the activation of c-Src and MAPK pathways, and modulates calcium homeostasis, impacting muscle function and proteostasis. Vitamin D treatment in-vitro setting has been shown to influence key aspects of muscle cell behavior, including myoblast proliferation, differentiation, and myotube formation. Notably, high doses of $1,25(OH)_2D$ can inhibit myoblast proliferation but promote hypertrophy in differentiated myotubes. For example, recent study demonstrated that $1,25(OH)_2D_3$ significantly enhances myogenic differentiation in C2C12 muscle cells by increasing myogenin expression through a functional VDRE on the myogenin promoter. This effect of vitamin D also resulted in larger myotube diameters and greater expression of myosin heavy chain isoforms, indicating its critical role in muscle cell development [2].

In animal studies, the role of vitamin D in muscle function and regeneration has been extensively explored using various models. Whole-body and tissue-specific VDR knockout models exhibit significant reductions in grip strength and muscle fiber size, along with increased expression of atrophy-related genes, underscoring the importance of VDR signaling in maintaining muscle integrity. Additionally, in injury models, the administration of vitamin D postinjury enhances muscle regeneration by promoting cell proliferation, decreasing apoptosis, and improving overall muscle repair [3]. These findings collectively suggest that vitamin D plays a crucial role in preserving muscle health and enhancing recovery following muscle damage or disease. Aside from the morphological changes, vitamin D influences muscle function by modulating calcium transport in skeletal muscle through nongenomic responses. It facilitates rapid calcium and phosphate movement across cell membranes, essential for muscle contraction. VDR regulates calcium flow when activated, further influencing muscle contraction. These mechanisms highlight vitamin D's role in calcium handling and muscle contraction [4].

Review of human studies

In focusing on the relationship between vitamin D and muscle health, sarcopenia – which increases the risk of clinical outcomes such as disability, falls, and mortality – is an important issue. Therefore, we reviewed articles published in the last 18 months that examined the relationship between vitamin D nutritional status and/or serum 25-hydroxyvitamin D [25(OH)D] levels and sarcopenia and related indicators such as muscle mass, muscle strength, and physical performance, in independent middle-aged and older adults. A systematic search was conducted using Medline (PubMed), with a detailed search strategy provided in Supplementary information, http://links.lww.com/COCN/A26.

We excluded RCT studies in which vitamin D was supplemented with other nutrients except for calcium. The results of the review are presented in Tables 1 and 2.

The relationship of dietary vitamin D intake and sarcopenia indicators (cross-sectional studies)

The studies about the relationship between vitamin D intake and indicator of sarcopenia diagnosis are two studies, but the results are not consistent. Among 719 older adults aged 70 years in Sweden, vitamin D intake assessed by diet history method showed a significant positive correlation with limb skeletal muscle mass index by DXA method ($r = 0.219$, $P = 0.004$), but no significant relationship with walking speed or grip strength [5]. On the other hand, there was no significant difference in the association between vitamin D intake by one-day dietary record including supplement and the muscle mass by DXA method among 159 Brazilian older adults aged 80 years or older [6].

Table 1. The association between vitamin D intake or serum concentration and physical performance and sarcopenia in cross-sectional and longitudinal studies

	n	Age (years) mean (SD)	Sex (proportion)	Other demographic	skeletal muscle mass index	Muscle mass	Muscle strength (Hand grip strength)	Arm curl test	Chair stand test	Walking speed	Fast walking speed	Times up and go test (TUG)	6-min walk test	SPPB (score)	Sarcopenia	Bone mineral density	Falls
Intake of vitamin D																	
Borda MG *et al.* [5]	719	70.5 (0.3)	M & F(M/F: 311/408)		+		n.s.			n.s.					n.s.		
Fonte *et al.* [6]	159	87.0 (3.9)	M & F(M/F: 43/116)			n.s.										n.s.	
Serum 25-hydroxivitamin D [25(OH)D] concentration (cross-sectional study)																	
Krasniqi *et al.* [7]	135	67.6 (9.7)	M			n.s.	n.s.	+	+	+	+	n.s.	n.s.				
	162	63.3 (10.2)	F			n.s.	n.s.	n.s.	+	n.s.	n.s.	n.s.	n.s.				
Zhang *et al.* [14]	3025	56.3 (9.9)	F			+											
Yang *et al.* [8]	58	Age unshown ※60 years and over	M		+	n.s.	n.s.			−					−		
	74	Age unshown ※60 years and over	F		+	+	n.s.			n.s.							
Yamada *et al.* [9]	163	62.0 (53.0–73.0)†	M & F(M/F: 96/67)	Antiaging health checkup recipients	+	+	n.s.									n.s.	
Welford *et al.* [10]	102	60.5 (4.7)	F	Healthy postmenopausal women	n.s.	n.s.	n.s.										
Foroni *et al.* [11]	200	85.5 (83.0–90.0)†	M & F(M/F: 53/147)		n.s.	n.s.										−	n.s.
Dwimartutie *et al.* [12]	95	70.1 (5.1)	M & F(M/F: 31/64)				+										
Stolakis *et al.* [13]	90	72.6 (6.2)	M & F(M/F: 13/77)	Outpatients with falls			+			+				+			n.s.
Kim *et al.* [17] Sarcopenia+DM (only male)	2952	Age unshown ※65 years and over	M & F(M/F: 1271/1681)	Diabetes											−:		
Hsu *et al.* [18]	n=110	Age unshown ※50–80 years	M & F (M/F: 46/64)												−		
Silveira *et al.* [16]	171	79.4 (5.9)	M & F(M/F: 58/113)												−		
Fox *et al.* [15▪]	2576	54.3 (14.2)	M & F(M/F: 1149/1427)				+										
(Longitudinal study)																	
Li *et al.* [19]	1910	Age unshown ※50 years and over	M & F(M/F: 655/1255)												n.s.		

+, positive association; -, negative association; n.s., no association; †, the value is shown as median (25th percentile - 75th percentile); Sarcopenia+DM (only male).
SPPC, Short Physical Performance Battery.

Table 2. Baseline characteristics of the included studies: effects of Vitamin D supplementation on physical function, performance test, and activity indexes

References	Group	Mean age in years (no. of individuals)	BMI	Vitamin D added, ng/ml	Intervention duration	Baseline 25 (OH)D, ng/ml, mean (SD)	After intervention 25 (OH)D, ng/ml, mean(SD)	Outcomes	Main outcome in the intervention group
Chou *et al.* [20■]	Intervention	64.7 (6.3) [520]	28.1 (5.3)	50 μg/day	24 months	27.6 (8.8)	40.0 (9.0)	Grip strength, TUG, walking speed, standing balance, repeated chair stands, SPPB	No significant improvement in physical performance
	Control	65.1 (6.6) [534]	28.3 (5.4)	0 μg/day (placebo)		28.7 (9.3)	No significant change		
Houston *et al.* [22]	Intervention	73.7 (6.3) [66]	30.2 (4.3)	50 μg/day	12 months	19.4 (4.2)	28.6 (6.7)	Lower-extremity leg power, leg and grip strength, SPPB, health ABC physical performance battery, TUG, postural sway, gait velocity	No significant improvement in leg power, strength, or physical performance
	Control	73.1 (6.3) [70]	30.4 (4.6)	0 μg/day (placebo)		19.9 (4.9)	20.2 (5.0)		
Schrack *et al.* [24]	Intervention	77.2 (5.3) [293]	30.6 (5.8)	≥25 μg/day (25 μg/d, 50 μg/d, 100 μg/d)	24 months	22.0 (4.9)	No description	SPPB, physical activity outcomes (total activity counts per day, active minutes per day, and activity fragmentation)	No significant improvement in physical activity outcomes
	Control	77.2 (5.3) [278]	30.6 (6.4)	5 μg/day		22.0 (5.2)	No description		
Draxler *et al.* [23]	Intervention	VD daily: No description [27] VD monthly: No description [31]	VD daily: 28.3 (4.4)VD monthly: 26.8 (4.3)	VD daily: 20 μg/ dVD monthly: 1250 μg/m	17 weeks	VD daily: 23.9 (6.0)VD monthly: 23.7 (6.6)	VD daily: 28.0 (7.6)VD monthly: 32.9 (7.6)	Arm curl test, TUG, 6-min walk test, 30- second chair stand test, the handgrip strength	No significant improvement in physical performance
	Control	No description [27]	26.2 (4.9)	0 μg/day (placebo)		20.8 (4.8)	23.6 (8.3)		
Dawson-Hughes *et al.* [25]	Intervention	71 (5) [185]	26.9 (4.0)	17.5 μg/d	36 months	22.2 (7.7)	30.8 (7.5)	Muscle performance (timed walk, grip strength, and chair-rise), two balance tests, the one-leg stand and tandem stand	The dominant hand grip strength significantly decreased in the VD group compared with the placebo group [-4.4 (18.9) vs -1.0 (17.1), $P=0.024$].No significant improvement in other physical performance and balance measure
	Control	71 (5) [201]	26.8 (4.2)	0 μg/day (placebo)		21.3 (7.0)	22.7 (6.3)		
Haghighi *et al.* [21]	Intervention	VD: 57.4 (4.8) [11] VD+resistance training: 55.4 (3.8) [11]	VD: 29.9 (5.0) VD+resistance training: 28.6 (3.1)	1250 μg/2 wks (Equivalent to 75 μg/d)	12 weeks	VD: 22.59 (14.67) VD+resistance training: 32.85 (12.49)	VD: 28.39 (18.05) VD+resistance training: 44.63 (17.4)	Leg press, chest press, leg extension, leg curl, shoulder press exercises	No significant improvement in upper- and lower-extremities muscle strength
	Control	Placebo+resistance training: 55.4 (0.0) [11] Placebo: 55.8 (4.7) [11]	Placebo+resistance training: 29.8 (3.7)Placebo: 30.11 (5.3)	0 μg/day (placebo)		Placebo+resistance training: 32.73 (6.74)Placebo: 19.91 (9.43)	Placebo+resistance training: 19.94 (9.1)Placebo: 20.2 (14.02)		

25(OH)D, 25-hydroxyvitamin D; SPPB, Short Physical Performance Battery; TUG, Timed-Up and Go; VD, Vitamin D.

The relationship of serum 25(OH)D concentration and muscle mass and physical/muscle function (cross-sectional studies)

Nine studies examined the relationship between serum 25(OH)D concentration and muscle mass, strength or function. Four investigated the relationship between serum 25(OH)D concentration and both of skeletal muscle mass and muscle strength [7–10]. There were four studies that only included older people [8,11–13]. Skeletal muscle mass was assessed by BIA in three studies [7,8,14] and by DXA in three studies [9–11]. Seven, two and three studies assessed muscle strength (hand grip strength) [7–10,12,13,15▪] and walking speed [7,8,13], respectively. For all indicators, the results were not consistent by sex and age group. In a study measuring serum 25(OH)D concentration in four seasons (spring, summer, autumn and winter), the results differed from season to season [10]. Considering that serum 25(OH)D concentration is also affected by sunlight, the season in which the study was conducted may have influenced the results.

The relationship between 25(OH)D concentration and falls (cross-sectional studies)

There are two studies about the relationship between 25(OH)D concentration and falls [11,13]. Participants were asked about their history of falls in the past 12 months. While participants with falls had a significantly lower serum 25(OH)D concentration than those without falls among people aged 65 years and over (24.3 ± 8.9 vs. 30.6 ± 9.3 ng/ml, $P = 0.001$) [13], no association was observed in the study for participants aged 80 years and over [11].

The relationship between sarcopenia and serum 25(OH)D concentration (cross-sectional studies)

Four studies examined the relationship between sarcopenia and serum 25(OH)D concentration. The diagnosis of sarcopenia used was the European Working Group on Sarcopenia in Older People (EWGSOP2) criteria in two articles [8,16] and the Asian Sarcopenia Working Group 2019 (AWGS2019) criteria in two articles [17,18].

The participants in all studies had a relatively high range of serum 25(OH)D concentrations. However, only one study found no association between sarcopenia diagnosis and serum 25(OH)D concentration [17]. However, in the study, the group with both sarcopenia and type 2 diabetes had the lowest serum 25(OH)D concentrations compared to those with sarcopenia alone, diabetes alone, and neither condition. A study conducted in Taiwan [18] included participants with type 2 diabetes, categorized into nonsarcopenia, possible sarcopenia, and sarcopenia groups. The findings demonstrated a significant inverse association between diabetes diagnosis and serum 25(OH)D levels. Furthermore, participants with both sarcopenia and osteoporosis had the lowest levels of vitamin D, which was generally consistent with previous study [16]. Future studies on the associations between serum 25(OH)D concentrations and sarcopenia should consider the involvement of other metabolic diseases.

Longitudinal study

In a cohort study involving 1910 Chinese men and women aged 50 years and older, the association between changes in sarcopenia status over 4 years and serum 25(OH)D levels was investigated. Sarcopenia was diagnosed using AWGS 2019 criteria. Although the assay method for determining serum 25(OH)D concentrations was not specified, no significant association was found after adjusting for covariates [19].

Vitamin D supplementation (intervention studies)

In this section, we review randomized controlled trials (RCTs), all of which were double-blind, investigating the effects of continuous vitamin D supplementation on physical functions and performance tests (Table 2). Six RCTs were selected [20▪,21–25]. Three studies used vitamins alone, one included calcium, one involved resistance exercise, and one combined vitamin D with both calcium and resistance exercise. Vitamin D dosages ranged from 17.5 to 50 μg per day, with interventions lasting from 12 weeks to 3 years. Individuals were aged 50 years or older, and five out of six studies included both men and women.

Three studies investigated the effects of vitamin D supplementation on physical performance, function, and activity. Two studies examined the effect of only supplemental vitamin D_3 (cholecalciferol, 50 μg/day) on physical function and performance tests. Over a 2-year period in middle to older adults [20▪] and a 12-month period in older adults [22], both studies consistently reported no significant effects on physical function and performance tests. Another study [24] observed the effect of 50 μg/day vitamin D_3 supplementation over 24 months on physical activity during free-living conditions in individuals over 70 years old. The results showed no significant changes in overall physical activity

levels, suggesting that vitamin D supplementation alone does not influence daily physical activity patterns in older adults.

One study examined the effect of combining vitamin D and calcium on muscle strength and physical performance [25]. This study found no significant improvement in muscle performance measures such as walking speed, grip strength, and chair-rise time, or balance measures like the one-leg stand and tandem stand compared to placebo. In fact, despite the supplementation with vitamin D_3 and calcium, grip strength of the dominant hand decreased in the supplemented group compared to the placebo group. On the contrary, higher 3-year mean 25(OH)D levels were positively associated with improved one-leg stand time, suggesting a potential benefit for balance.

Another study investigated the combined effects of vitamin D supplementation and resistance training over 12 weeks [23]. The results indicated significant improvements in muscle strength and power with resistance training. However, vitamin D supplementation alone did not provide any additional benefit. This highlights the importance of physical exercise in improving muscle function and suggests that vitamin D alone is not sufficient.

Overall, recent intervention studies suggest that vitamin D supplementation does not significantly enhance physical performance, muscle function, or daily physical activity patterns in middle-aged to

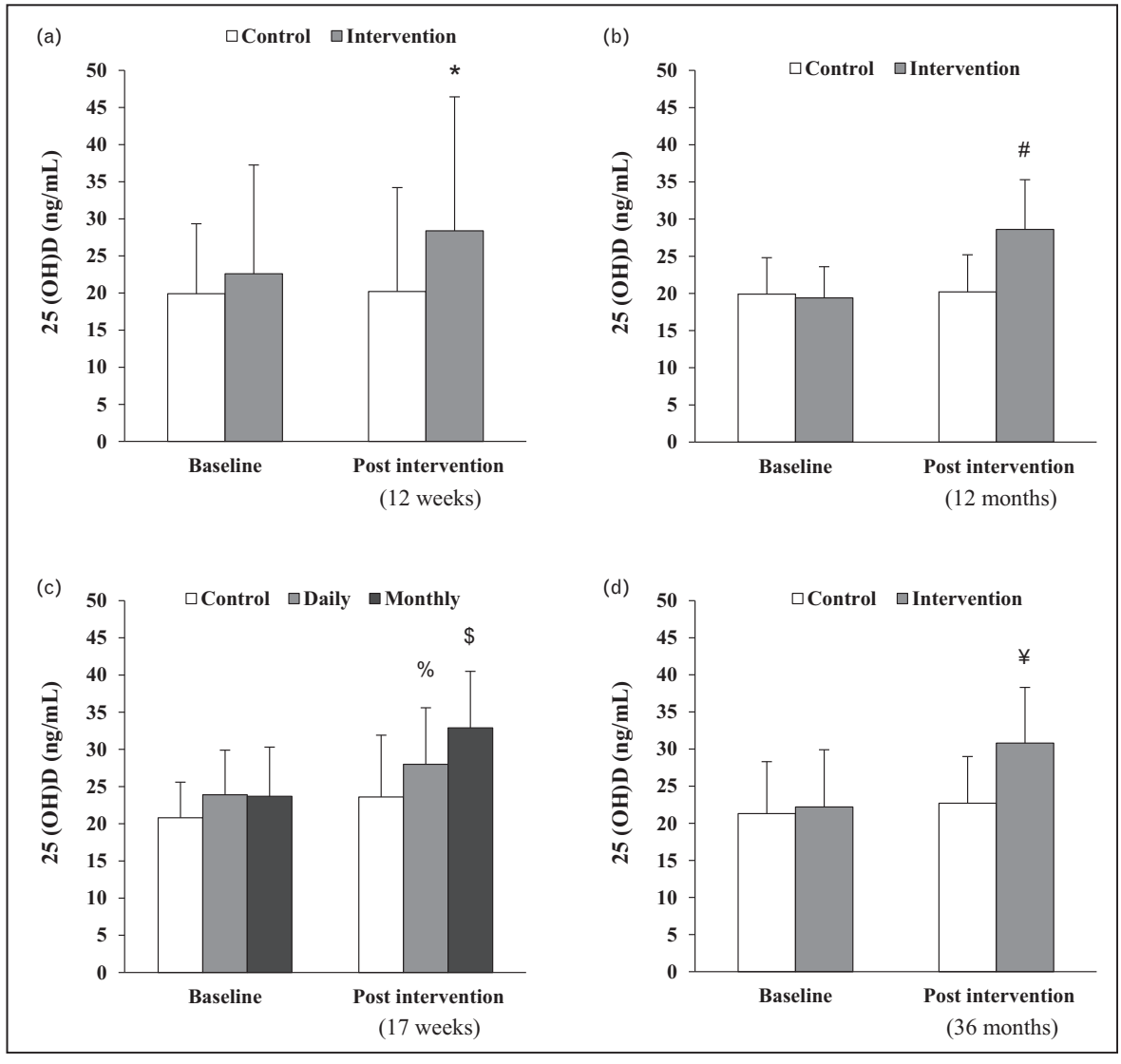

FIGURE 1. Changes in Serum 25-hydroxyvitamin D [25(OH)D] concentrations before and after Vitamin D supplementation across four randomized controlled trials (RCTs). Panels (a: Ref. [21], b: Ref. [22], c: Ref. [23], and d: Ref. [25]) correspond to different RCTs. * significantly different from baseline, # significant difference in change from pre to postintervention between groups ($P<0.0001$), % $P=0.01$ vs. baseline, $ $P<0.001$ vs. baseline, ¥ $P<0.001$ vs. control.

older adults regardless of the frequency, dose, or duration of the intervention. One contributing factor is that the study was conducted in populations with vitamin D sufficiency (25(OH)D level $\geq$ 20 ng/ml) (Fig. 1). A previous review indicated that the effectiveness of vitamin D intervention on clinical outcomes is poor in vitamin D sufficient individuals with baseline circulating 25(OH)D levels of at least 20 ng/ml [26].

In addition, a recent meta-analysis further complicates this picture, revealing that vitamin D supplementation might negatively affect muscle health, including increased time for timed up and go (TUG) tests, decreased maximal muscle strength during knee flexion, and a trend toward lower Short Physical Performance Battery (SPPB) scores [27]. These findings suggest that vitamin D alone may not provide clear benefits for muscle health and may even have adverse effects under certain conditions. Furthermore, inconsistent findings regarding vitamin D's effects on muscle mass and function may stem from variations in supplementation dose, duration, baseline 25(OH)D levels, and genetic factors. Polymorphisms in the *VDR* gene, such as BsmI and FokI, might influence individual responses to vitamin D, leading to varied outcomes [28]. However, it is too early to draw conclusions due to the small number of studies reviewed.

CONCLUSION

The relationship between vitamin D and muscle health in middle-aged and older adults is complex and not fully conclusive. Recent human studies have shown inconsistent results regarding the benefits of vitamin D status on muscle mass, strength, and function. These mixed outcomes may be due to differences in study design, ingested dosages, and participants' baseline 25(OH)D levels. While in-vitro and animal studies indicate that vitamin D plays a significant role in muscle physiology, these findings are not always reflected in human trials. It is clear that vitamin D alone is not sufficient to prevent sarcopenia, emphasizing the need for a multifaceted approach that includes resistance training and takes into account individual genetic differences. Future research should focus on unraveling the detailed interactions between vitamin D, muscle function, and other influencing factors to improve muscle health strategies for aging populations.

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Conflicts of interest

All authors have no conflict of interest to disclose.

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- ■ of special interest
- ■■ of outstanding interest

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