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ORIGINAL ARTICLE

Low vitamin D status is associated with reduced muscle mass and impaired physical performance in frail elderly people

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BACKGROUND/OBJECTIVES: Serum 25-hydroxyvitamin D (25(OH)D) status has been associated with muscle mass, strength and physical performance in healthy elderly people. Yet, in pre-frail and frail elderly people this association has not been studied. The objective of this study was to explore the association between vitamin D intake and serum 25(OH)D status with muscle mass, strength and physical performance in a pre-frail and frail elderly population.

SUBJECTS/METHODS: This cross-sectional study included 127 pre-frail and frail elderly people in The Netherlands. Whole body and appendicular lean mass (ALM) (dual energy X-ray absorptiometry), leg strength (one repetition maximum), handgrip strength and physical performance (short physical performance battery) were measured, and blood samples were collected for the assessment of serum 25(OH)D status (liquid chromatography-tandem mass spectrometry). In addition, habitual dietary intake (3-day food records) and physical activity data (accelerometers) were collected.

RESULTS: In total, 53% of the participants had a serum 25(OH)D level below 50 nmol/l. After adjustment for confounding factors, 25(OH)D status was associated with ALM ($\beta = 0.012$, $P = 0.05$) and with physical performance ($\beta = 0.020$, $P < 0.05$). Vitamin D intake was associated with physical performance ($\beta = 0.18$, $P < 0.05$) but not with ALM ($P > 0.05$).

CONCLUSION: In this frail elderly population, 25(OH)D status is low and suggests a modest association with reduced ALM and impaired physical performance. In addition, vitamin D intake tended to be associated with impaired physical performance. Our findings highlight the need for well-designed intervention trials to assess the impact of vitamin D supplementation on 25(OH)D status, muscle mass and physical performance in pre-frail and frail elderly people.

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INTRODUCTION

Frailty is a geriatric syndrome of decreased reserves and resistance to stressors, which increases the risk for falls, disability, morbidity and institutionalization.[1,2] An important and fundamental component of frailty is sarcopenia.[3] Sarcopenia is characterized by a progressive loss of skeletal muscle mass and physical performance.[4]

The cause of this loss in muscle mass and performance is multifactorial and might include vitamin D deficiency.[5,6] The Institute of Medicine currently considers a serum 25-hydroxyvitamin D (25(OH)D) level below 50 nmol/l as being deficient.[7] The estimated prevalence of vitamin D deficiency among healthy elderly people is between 45 and 57%,[8–10] and among compromised geriatric patients vitamin D deficiency is even more pronounced.[11,12] An inadequate 25(OH)D status has been associated with poor muscle mass and impaired physical performance in community-dwelling elderly people.[5,6,13–15] Although significant associations between inadequate 25(OH)D status and reduced muscle mass, strength and physical performance have been well-established in a healthy elderly population,[5,6,13–15] there are few data available on such associations in more compromised, frail elderly subpopulations. Studying the association between 25(OH)D level and muscle mass, strength and physical performance in a frail elderly population is important, as an inadequate 25(OH)D status might be more pronounced within this population together with decreased muscle mass, impaired physical performance[16] and their risk for falls and fractures.[1,2] A low 25(OH)D status among frail elderly may predispose to the development of muscle mass loss and impairments in strength and physical performance resulting in more frequent falls and fractures.[17–20] Therefore, in the present study, we examined the association between 25(OH)D level and vitamin D intake with muscle mass, strength and physical performance in a pre-frail and frail elderly population.

MATERIALS AND METHODS

Study sample

Community-dwelling elderly participants, >65 years, were recruited between December 2009 and September 2010. A detailed description is provided elsewhere.[21–22] In short, subjects were screened for pre-frailty and frailty using the Fried criteria.[2] These criteria were: (1) unintentional weight loss, (2) weakness, (3) self-reported exhaustion, (4) slow walking speed and (5) low physical activity. Pre-frailty was classified when one or two criteria were present, and frailty was defined when three or more criteria were present. Furthermore, subjects who were diagnosed with any form of cancer, chronic obstructive pulmonary disease, diabetes type 1 and 2 ($\geqslant$7 mmol/l)[23] or renal insufficiency (epidermal growth factor receptor $<$60 ml/min/1.73 m^2)[24] were excluded. None of the subjects had

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participated in a resistance-type exercise program over the past 2 years. The baseline data set of 127 pre-frail and frail elderly subjects was used for the current analysis. The Wageningen University Medical Ethical Committee approved the study, and subjects gave their written informed consent.

Blood sampling and analysis

After an overnight fast, blood samples were collected in ethylenediaminetetraacetic acid-containing tubes and in serum tubes. The ethylenediaminetetraacetic acid-containing tubes were centrifuged at 1000 *g* at 4 °C for 10 min, and serum tubes were centrifuged 90 min after the blood collection at 1000 *g* at 20 °C for 15 min. Aliquots of plasma and serum were snap frozen in liquid nitrogen and stored at − 80 °C. The plasma samples were used to determine the subjects' glucose and insulin concentrations and the serum samples for creatinine and 25(OH)D concentrations. Plasma glucose concentrations were measured with a COBAS FARA analyzer (Uni Kit III; Roche, Basel, Switzerland). Insulin was measured by radioimmunoassay (Insulin RIA Kit; LINCO Research Inc, St Charles, MO, USA). Serum creatinine was measured using Roche Modular System P (Roche Diagnostics GmbH, Mannheim, Germany). Serum 25(OH)D levels were measured using isotope dilution-online solid phase extraction liquid chromatography-tandem mass spectrometry (ID-XLC-MS/MS) by the Endocrine Laboratory of the VU University Medical Center, the Netherlands. 25(OH)D was released from its binding protein(s) and a deuterated internal standard (IS: 25(OH)D3-d6) was added. Samples were extracted and analyzed using XLC-MS/MS (a Symbiosis online SPE system; Spark Holland, Emmen, The Netherlands) coupled to a Quattro Premier XE tandem mass spectrometer (Waters Corp, Milford, MA, USA). The intra-assay and inter-assay coefficient of variance for this analysis were $<6\%$ and $<8\%$, respectively, when using three concentrations between 25 and 180 nmol/l, and the Limit of Quantification was 4.0 nmol/l.

Dietary intake

Dietary intake data, including vitamin D intake, were obtained by means of 3-day food records. The 3 days of recording were randomly assigned to ensure that all days of the week, including weekend, were equally represented. Vitamin D supplement use was not assessed. Dietary data were coded (type of food, time of intake and estimated portion size) and calculated using a food calculation system (BAS nutrition software 2004, Arnhem, The Netherlands), in which the Dutch food composition database of 2006 was included.

Body composition, maximum strength and physical performance

Assessment of body composition, maximum strength and physical performance were performed within 4 weeks following blood sampling. Lean body mass, appendicular lean mass (ALM), leg lean mass and bone

Table 1. Subject characteristics according to their 25(OH)D status

Variable	*Total sample, n = 127*	*25(OH)D < 50 nmol/l, n = 67*	*25(OH)D ⩾ 50 nmol/l, n = 60*	P-*value*
25(OH)D (nmol/l) (median ± lower/upper quartile)	47 (33–73)	35 (24–40)	73.5 (65–89.5)	0.00
Vitamin D intake (μg/day)	4.6 ± 3.0	4.2 ± 3.2	5.0 ± 2.8	0.17
Age (years)	79.0 ± 7.8	80.8 ± 7.5	77.0 ± 7.6	0.01
Women (%)	61	55	67	0.19
Weight (kg)[a]	76.2 ± 13.8	77 ± 14	75 ± 14	0.38
Height (m)	1.66 ± 0.09	1.66 ± 0.09	1.66 ± 0.09	0.86
BMI (kg/m^2)[a]	27.5 ± 4.3	27.8 ± 4.3	27.2 ± 4.4	0.55
Alcohol (%)				
None	28	30	25	0.67
< 1 Consumption/day	32	34	31	
⩾ 1 Consumption/day	40	36	44	
Education level (%)				
Low	5.5	6.0	5.0	0.89
Middle	59.6	61.2	58.3	
High	34.6	32.8	36.7	
Smoking (%)[b]	6.5	6.2	6.9	0.87
Total lean mass (kg)[c]	46.3 ± 9.3	46.8 ± 9.9	45.7 ± 8.5	0.50
Appendicular lean mass (kg)[c]	19.5 ± 4.3	19.7 ± 4.7	19.3 ± 3.9	0.32
Lean leg mass (kg)[c]	14.7 ± 3.2	14.8 ± 3.5	14.5 ± 2.8	0.23
Fat mass (kg)[c]	26.2 ± 9.0	26.5 ± 8.8	25.9 ± 9.2	0.69
Bone mineral content (kg)[c]	2.5 ± 0.6	2.6 ± 0.7	2.5 ± 0.6	0.64
1-RM leg press (kg)[d]	120 ± 34	120 ± 35	120 ± 34	0.99
1-RM leg extension (kg)[e]	57 ± 19	57 ± 19	58 ± 19	0.85
Handgrip strength (kg)[a]	26.1 ± 9.2	26.0 ± 8.6	26.2 ± 10.1	0.95
SPPB (points)[b]	8.2 ± 2.8	7.1 ± 2.9	9.2 ± 2.3	0.00
Balance (points)[b]	3.1 + 1.1	2.8 ± 1.3	3.5 ± 0.8	0.01
Gait speed (sec)[b]	5.6 ± 2.7	6.3 ± 3.2	4.8 ± 1.7	0.01
Chair rise (sec)[f]	15.1 ± 6.2	16.6 ± 6.0	13.6 ± 6.0	0.02
Physical activity (counts/min)[g]	139.5 ± 94.1	99.6 ± 66.7	181 ± 101	0.00
Energy intake (MJ/day)[a]	8.2 ± 2.2	8.0 ± 2.2	8.4 ± 2.1	0.36
Calcium intake (mg)	1032 ± 395	990 ± 347	1078 ± 440	0.21
Protein intake (g/day)[a]	76.4 ± 21.6	75.8 ± 20.6	77.0 ± 22.9	0.75
Glucose (mmol/l)[h]	5.3 ± 0.5	5.3 ± 0.5	5.2 ± 0.4	0.57
Insulin (mU/l)[h]	18.5 ± 7.0	18.6 ± 6.4	18.3 ± 7.6	0.85
Creatinine (mmol/l)	72.8 ± 14.8	75.0 ± 14.4	70.4 ± 13.8	0.07
Season (%)				
Summer	48	52	43	0.32
Fall	52	48	57	

Abbreviations: BMI, body mass index; SPPB, short physical performance battery; 1-RM, one repetition maximum; 25(OH)D, 25-hydroxyvitamin D. Values are expressed as a mean ± s.d., median with upper and lower quartile or percentage. Superscript indicate missing values. [a]1 missing value. [b]4 missing values. [c]10 missing values. [d]16 missing values. [e]18 missing values. [f]28 missing values. [g]21 missing values. [h]17 missing values.

mineral content were measured with dual energy X-ray absorptiometry scan (Lunar Prodigy Advance; GE Health Care, Madison, WI, USA). Maximum strength was assessed by one repetition maximum strength tests on leg press and leg-extension machines (Technogym, Rotterdam, the Netherlands). During a familiarization session, the proper lifting technique was demonstrated and practiced, after which maximum strength was estimated. In a second session, $\geqslant$1 week after the first strength estimation, the subjects' one repetition maximum strength was determined.[25] Handgrip strength was measured using a hydraulic hand dynamometer (Jamar, Jackson, MI, USA). Three consecutive measures of handgrip strength (kg) exerted by both hands were recorded to the nearest 0.5 kg with subjects sitting in an upward position with the arm in a 90° angle. The maximum strength effort was reported.

Physical performance was assessed with the short physical performance battery (SPPB) that comprises three components, that is, standing balance, gait speed and chair stands.[26] Scores of 1–4 were on the basis of categories of performance in the balance tests, on the time necessary to complete the walk and on the time needed to perform the chair rise test. When subjects were unable to perform a test, a score of 0 was allocated. A summary of SPPB score between 0 and 12 was obtained through summation of the scores obtained in the three individual tests.

Potential confounders

The following potential confounders were included in the statistical analysis: age, gender, height, body weight, alcohol (none, <1 consumption/day, >1 consumption/day), habitual physical activity, season of data collection, education (low, medium and high), serum creatinine, smoking, energy and protein intake. Height was measured with a wall-mounted stadiometer and body weight with a calibrated digital scale (ED-6-T; Berkel, Rotterdam, The Netherlands). Habitual physical activity data were quantified using a tri-axial accelerometer (ActiGraph GTX3, 2009, Pensacola, FL, USA) worn on the hip for 1 week. Change of acceleration per second and epochs of 60 s were used. After 7 days, data were uploaded for analysis and analyzed using the MAH/UFFE analyzer, version 1.9.0.3 (MRC Epidemiology Unit, Cambridge, UK). Data files that did not meet 10 h of monitoring per day on at least 5 days as well as files that included periods of >100 min without activity were excluded from the analysis. Calcium intake and appendicular lean body mass (strength and physical performance outcomes) were not included in the model, because this did not change the β substantially.

Statistics

Characteristics of the study population were reported as the mean ± s.d., as percentage or as medians (25–75 percentile). Participants were grouped according to published 25(OH)D status cut points: <50 and $\geqslant$50 nmol/l.[6, 27] χ^2 tests for categorical variables and independent sample *t*-tests for continuous variables were performed to compare participants with 25(OH)D levels below and above 50 nmol/l. The multiple linear regression analysis was used to investigate the association of 25(OH)D status and vitamin D intake with the outcome variables, adjusted for age, gender, height, body weight, alcohol, physical activity, education, smoking, creatinine (model 2) and energy and protein intake (model 3). Serum creatinine was not included as a confounder in any of the models investigating the association of vitamin D intake with the dependent variables. The statistical analysis was carried out using SPSS version 19 (SPSS, Chicago, IL, USA). A *P*-value $\leqslant$0.05 was considered as statistically significant.

RESULTS

General characteristics of the study population are presented in Table 1. In total, 53% of the elderly in this study were vitamin D deficient, as reflected by the number of persons with serum 25(OH)D levels below 50 nmol/l. In addition, 94% had a vitamin D intake below the estimated average requirement of 10 μg/day,[7] with an average vitamin D intake of 4.6 ± 3.0 μg/day. Participants with sufficient 25(OH)D levels were more likely to be younger ($P = 0.01$) and more physically active ($P < 0.001$) compared with those with insufficient levels. Furthermore, crude data, as presented in Table 1, suggest better physical performance with

Table 2. Association of 25(OH)D status and vitamin D intake with body composition

Variable	*25(OH)D status*				*Vitamin D intake*			
	β	*95% CI*	P-*value*	*n*	*β*	*95% CI*	P-*value*	*n*
Total lean mass (kg)								
Model 1	0.002	−0.061–0.065	0.958	117	0.265	−0.287–0.818	0.343	117
Model 2	0.015	−0.010–0.039	0.234	96	0.028	−0.176–0.232	0.783	96
Model 3	0.018	−0.006–0.041	0.139	95	−0.096	−0.319–0.127	0.394	95
Appendicular lean mass (kg)								
Model 1	0.007	−0.022–0.036	0.631	117	0.114	−0.143–0.371	0.382	117
Model 2	0.011	−0.002–0.024	0.093	96	−0.007	−0.116–0.102	0.899	96
Model 3	0.012	0.000–0.025	0.050	95	−0.037	−0.157–0.084	0.546	95
Leg lean mass (kg)								
Model 1	0.005	−0.016–0.027	0.624	117	0.088	−0.101–0.277	0.359	117
Model 2	0.008	−0.002–0.019	0.124	96	−0.005	−0.093–0.083	0.910	96
Model 3	0.009	−0.001–0.019	0.079	95	−0.014	−0.112–0.083	0.773	95
Fat mass (kg)								
Model 1	−0.048	−0.108–0.013	0.124	117	0.210	−0.329–0.749	0.442	117
Model 2	−0.016	−0.039–0.008	0.198	96	−0.128	−0.325–0.070	0.202	96
Model 3	−0.019	−0.041–0.004	0.103	95	−0.025	−0.240–0.191	0.820	95
Fat percentage (%)								
Model 1	−0.047	−0.106–0.013	0.126	117	0.069	−0.462–0.600	0.798	117
Model 2	−0.023	−0.058–0.013	0.202	96	−0.115	−0.410–0.181	0.443	96
Model 3	−0.027	−0.061–0.007	0.115	95	0.024	−0.298–0.346	0.884	95

Abbreviations: 25(OH)D, 25-hydroxyvitamin D; CI, confidence interval. Model 1: unadjusted model. Model 2: adjusted for age, gender, height, body weight, alcohol, physical activity, education, smoking and creatinine. Model 3: additionally adjusted for energy and protein intake. Creatinine was not included as a confounder in any of the models investigating the association of vitamin D intake with the dependent variables.

higher serum 25(OH)D levels. Participants with 25(OH)D levels ⩾50 nmol/l scored two points higher on the SPPB when compared with participants with levels of 25(OH)D <50 nmol/l ($P<0.001$). Analyzing the different components of the SPPB showed a similar trend. Gait speed and chair rise were performed significantly faster and also balance scores were higher among those with sufficient 25(OH)D levels, 4.8 vs 6.3 s ($P=0.01$), 13.6 vs 16.6 s ($P=0.02$) and 3.5 vs 2.8 points on a four point scale ($P=0.01$), respectively. Vitamin D intake did not correlate with serum 25(OH)D levels, $r=0.09$ ($P=0.30$).

Associations between serum 25(OH)D status and vitamin D intake with measures of body composition are shown in Table 2. Serum 25(OH)D status appeared to be positively associated with ALM, $\beta=0.012$ ($P=0.05$) and showed a tendency for a positive association with leg lean mass, $\beta=0.009$ ($P=0.08$). There was no association between vitamin D intake and measures of body composition.

Table 3 presents the associations of 25(OH)D status and vitamin D intakes with measures of maximum strength and physical performance. After full adjustment, significant associations were observed for 25(OH)D status and vitamin D intake with SPPB ($\beta=0.020$ ($P=0.035$) and $\beta=0.180$ ($P=0.038$), respectively).

DISCUSSION

In total, 53% of the frail elderly had 25(OH)D status below <50 nmol/l. This low 25(OH)D status was modestly associated with reduced ALM and impaired physical performance. Moreover, a tendency toward an inverse association between vitamin D intake and impaired physical performance was observed.

The present study is the first cross-sectional study investigating the association between 25(OH)D status and muscle mass in a pre-frail and frail elderly population. To allow the inclusion of an adequate sample of frail elderly subjects, 1420 elderly people were approached, 398 were screened and finally 127 participants met the frailty criteria described by Fried *et al*.[2] These criteria have been reported to be highly predictive for falls, hospitalization, disability and mortality.[2] In agreement, the selected subjects were characterized by low baseline physical performance, strength and poor handgrip strength (Table 1). Moreover, this study revealed a high prevalence of elderly people with an insufficient 25(OH)D level. As our blood sampling took place during summer and fall, this reported prevalence may be an underestimation of the 25(OH)D status for winter and early spring. During the winter and early spring, 25(OH)D levels have been reported to be substantially lower due to low sunlight exposure,[28] suggesting an

Table 3. Association of 25(OH)D status and vitamin D intake with physical performance

Variable	*25(OH)D status*				*Vitamin D intake*			
	β	*95% CI*	P-*value*	*n*	*β*	*95% CI*	P-*value*	*n*
1-RM leg press (kg)								
Model 1	0.040	−0.193–0.274	0.732	111	2.497	0.426–4.567	0.019	111
Model 2	0.141	−0.079–0.361	0.205	95	1.463	−0.358–3.284	0.114	95
Model 3	0.141	−0.076–0.357	0.200	94	1.646	−0.383–3.676	0.110	94
1-RM leg extension (kg)								
Model 1	0.055	−0.074–0.185	0.397	109	0.945	−0.235–2.126	0.115	109
Model 2	0.062	−0.049–0.174	0.268	93	0.203	−0.746–1.153	0.671	93
Model 3	0.063	−0.050–0.176	0.269	92	0.235	−0.842–1.312	0.665	92
Handgrip strength (kg)								
Model 1	0.004	−0.057–0.065	0.902	126	0.533	0.002–1.065	0.049	126
Model 2	−0.016	0.068–0.035	0.527	103	0.008	−0.411–0.427	0.970	103
Model 3	−0.013	−0.064–0.039	0.620	102	−0.061	−0.529–0.408	0.797	102
SPPB (points)								
Model 1	0.033	0.016–0.051	0.000	123	0.160	−0.004–0.324	0.056	113
Model 2	0.022	0.003–0.040	0.020	102	0.064	−0.093–0.220	0.442	102
Model 3	0.020	0.001–0.038	0.035	101	0.180	0.010–0.350	0.038	101
Gait speed (m/s)								
Model 1	−0.028	−0.045–−0.011	0.001	123	−0.147	−0.302–0.007	0.062	123
Model 2	−0.016	−0.033–0.001	0.059	102	−0.054	−0.196–0.088	0.448	102
Model 3	−0.014	−0.031–0.003	0.095	101	−0.160	−0.313 –−0.007	0.041	101
Chair rise (s)								
Model 1	−0.068	−0.115–0.022	0.004	99	0.102	−0.289–0.493	0.607	99
Model 2	−0.048	−0.106–0.010	0.103	82	0.211	−0.215–0.638	0.326	82
Model 3	−0.043	−0.100–0.015	0.145	81	−0.021	−0.505–0464	0.993	81
Balance score (points)								
Model 1	0.010	0.003–0.017	0.005	123	0.035	−0.029–0.099	0.278	123
Model 2	0.004	−0.003–0.012	0.268	102	0.011	−0.056–0.078	0.749	102
Model 3	0.004	−0.004–0.012	0.311	101	0.042	−0.033–0.117	0.267	101

Abbreviations: CI, confidence interval; SPPB, short physical performance battery; 1-RM, one repetition maximum; 25(OH)D, 25-hydroxyvitamin D. Model 1: unadjusted model. Model 2: adjusted for age, gender, height, body weight, alcohol, physical activity, education, smoking and creatinine. Model 3: additionally adjusted for energy and protein intake. Creatinine was not included as a confounder in any of the models investigating the association of vitamin D intake with the dependent variables.

even higher prevalence of frail elderly people with inadequate 25(OH)D levels.

In our study, a significant association between 25(OH)D status and ALM in a frail elderly population was found. This association of 25(OH)D status and ALM is supported by some,[5,6] but not all epidemiological studies.[29,30] Mechanistically, it is suggested that the vitamin D receptor in the skeletal muscle tissue has an important role in the balance of muscle protein turnover.[31] The activation of the vitamin D receptor might stimulate skeletal muscle protein synthesis[31,32] and might prevent type 2 muscle fiber atrophy.[33] However, most work has been done *in vitro* and more research is needed in humans to understand the underlying mechanisms that support our findings on ALM.

Our results indicated that 25(OH)D status and vitamin D intake are positively associated with physical performance measures in frail elderly people. We found that participants with 25(OH)D levels $\geqslant 50$ nmol/l scored two points higher on the SPPB when compared with participants with levels of 25(OH)D < 50 nmol/l ($P < 0.001$). In accordance, the SPPB score significantly improved with 0.02 points per 1 nmol/l increase in 25(OH)D and 0.18 points per 1 μg increase in vitamin D intake, indicating a clinically relevant improvement.[34] These findings are in line with the majority,[5,15,35–40] but not with all observational studies.[29,41] Our findings are in line with randomized, placebo-controlled trials, showing an improvement of physical performance after vitamin D supplementation in community-dwelling elderly people.[14,42,43] This improvement of physical performance might be attributed to the role of 125-dihydroxyvitamin D (125(OH)2D), the active form of 25(OH)D, in muscle. It has been suggested that 125(OH)2D regulates muscle calcium concentrations by modulating the activity of calcium pumps in sarcoplasmic reticulum and sarcolemma.[13] Alterations in intracellular calcium concentrations regulate the contraction and relaxation of muscle, which may impact physical performance. The latter underpins the importance of an adequate vitamin D intake and 25(OH)D status to improve or maintain physical performance.

A possible limitation might be the appearance of reverse causation owing to the cross-sectional design of the study. It may be that participants with the highest physical activity level and physical performance score are the ones that are the most likely to go outside and consequently have a higher 25(OH)D status, and not *vice versa*. However, a causal relationship seems plausible because of biological mechanisms[31] and evidence obtained from randomized, placebo-controlled trials[33,43,44] that confirm the causality of 25(OH)D status and physical performance.

Despite the association of 25(OH)D status with ALM, we found no significant association between vitamin D intake and ALM. The latter finding might be explained by the lack of correlation between vitamin D intake and 25(OH)D status. In our study, vitamin D intake was 4.6 ± 3.0 μg/day which is in line with our expectations, as food fortification in The Netherlands is not broadly practiced and vitamin D rich products are often not part of the daily diet. The lack of association between vitamin D intake and 25(OH)D status might be attributed by that low and narrow range of vitamin D intake. Despite the lack of correlation between vitamin D intake and 25(OH)D status in our study, a recent meta-regression analysis did show a significant association between vitamin D intake and 25(OH)D status.[45] Moreover, ample evidence presented an increase in 25(OH)D status after vitamin D supplementation,[12,46–48] suggesting that vitamin D supplementation represents an effective strategy to improve 25(OH)D status. Especially among frail elderly people, there is a greater need to take vitamin D supplements, because endogenous vitamin D synthesis decreases with age. This decreased vitamin D synthesis might be explained by a low outdoor habitual physical activity[49] and thus a low sunlight exposure as well as the reduced capacity to synthesize vitamin D in the skin.[50] More well-designed interventions studies are warranted to investigate the impact of vitamin D supplementation on 25(OH)D status and its impact on muscle mass and physical performance in a frail elderly population.

In conclusion, 25(OH)D was modestly associated with reduced ALM and impaired physical performance. In addition, vitamin D intake was modestly associated with physical performance.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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