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Associations Between 25-Hydroxyvitamin D and Weight Gain in Elderly Women

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Abstract

Background: 25-Hydroxyvitamin D [25(OH)D] levels are lower in obese individuals. Determining whether low vitamin D status can predispose weight gain requires a longitudinal study.
Methods: From a community-based multicenter U.S. prospective cohort of 9704 (Study of Osteoporotic Fractures [SOF]), 4659 women aged ≥65 with baseline 25(OH)D measurement were followed for 4.5 years. They were weighed at baseline and follow-up visits, and a subset ($n=1054$) had 25(OH)D levels remeasured at follow-up.
Results: Women with 25(OH)D levels ≥30 ng/mL had lower baseline weight (141.6 pounds) compared to women with 25(OH)D levels <30 ng/mL (148.6 pounds) ($p<0.001$). Overall, 25(OH)D status was not associated with weight change over 4.5 years, although there was a significant interaction between 25(OH)D status and weight change category (loss, gain, stable) ($p<0.0001$). In women who gained ≥5% weight, those with baseline 25(OH)D levels ≥30 ng/mL gained 16.4 pounds (12.2% of baseline weight) over 4.5 years compared to 18.5 pounds (13.9% of baseline weight) in women with levels <30 ng/mL ($p=0.04$). In women who lost ≥5% weight or remained stable (<5% weight change), there was no association between 25(OH)D status at baseline and weight change. Among women who gained weight and had 25(OH)D measured at both visits, having sustained or developing 25(OH)D levels ≥30 ng/mL was associated with less weight gain between visits (14.81 vs. 16.34 pounds, $p=0.04$).
Conclusions: Higher 25(OH)D levels are associated with lower weight gains, suggesting low vitamin D status may predispose to fat accumulation.

Introduction

Increased body fat and obesity have been associated with lower circulating 25-hydroxyvitamin D [25(OH)D] levels.[1–10] Although this association has been attributed to increased storage of 25(OH)D in fat tissue or potential lifestyle differences between obese and nonobese populations,[11,12] evidence from epidemiologic,[1–10] weight loss,[13–17] and *in vitro*/animal studies[18–24] suggests that 25(OH)D might be involved in weight regulation. Vitamin D receptors (VDR) are present on human adipocytes.[22] *In vitro*, 25(OH)D appears to influence lipogenesis, lipolysis,[18,19] adipogenesis,[21–24] and adipocyte gene transcription.[22] In weight loss trials, patients with higher 25(OH)D levels experience more weight and fat loss than patients with lower levels.[16,25]

Vitamin D is a unique vitamin in that the primary natural source is not food but synthesis in skin after sun exposure.[26] From a teleologic standpoint, decreased sun exposure in fall and winter could be a trigger, via 25(OH)D signaling, to increase fat storage for the coming cold weather and diminished food supply. As modern societies move indoors, we propose that decreased sunlight exposure leads to chronic 25(OH)D insufficiency and subsequent weight gain year round (not just seasonally). We sought to evaluate the relationship of 25(OH)D status to weight change over time in older women. Older adults are an ideal population for such a study, as they both have a high prevalence of 25(OH)D deficiency[27] and experience dynamic weight changes. Although aging is generally associated with weight loss, a significant proportion of older adults actually gain weight.[28]

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In this study, we sought to determine if 25(OH)D levels among 4659 community-dwelling ambulatory women aged ≥65 are associated with weight change over about 4 years. In a smaller cohort of women who had 25(OH)D levels determined at both visits ($n=1054$), we examined whether change in 25(OH)D level is associated with change in weight. We hypothesized that higher 25(OH)D levels would be associated with less weight gain over time.

Materials and Methods

Study sample

In 1986–1988, the Study of Osteoporotic Fractures (SOF) recruited 9704 community-dwelling women, aged ≥65 (>99% non-Hispanic white) in four U.S. regions: Baltimore County, Maryland; Minneapolis, Minnesota; Portland, Oregon; and the Monongahela Valley near Pittsburgh, Pennsylvania.[29] Women unable to walk without assistance and those with bilateral hip replacements were excluded. All women provided written consent, and SOF was approved by each site's Institutional Review Board.

All surviving participants were invited to attend a year 6 examination between August 1992 and July 1994. A total of 6818 women completed the year 6 clinic or home visit, and 6256 had measurement of the 25(OH)D level.[30] Of these, 4659 women (74%) had weight measurements at both the year 6 and the year 10 examinations (average of 4.5 years later); these women are the subject of the baseline 25(OH)D analyses in this report (Fig. 1). In addition, a subcohort of women who participated in the year 10 examination were randomly selected to have 25(OH)D levels retested. The 1054 women with weights and 25(OH)D levels at both the year 6 and year 10 visits comprise the analytic cohort for the longitudinal 25(OH)D analyses.

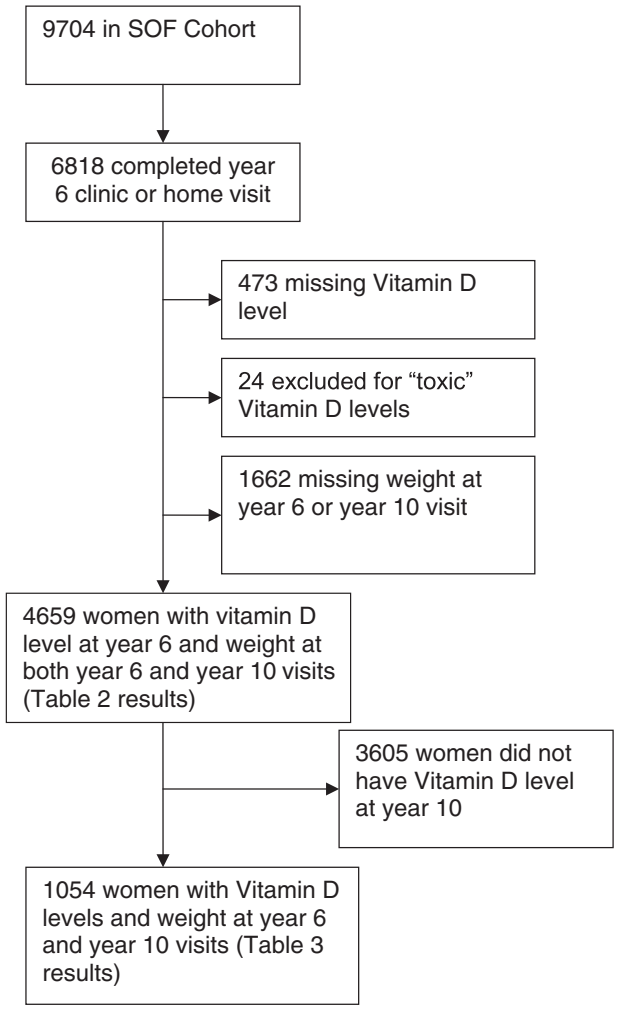

FIG. 1. Flow of participants. SOF, Study of Osteoporotic Fractures.

Weight

During the home or clinic visits, body weight was measured in light clothing with a standard balance beam or digital scale. At clinic visits, height was measured with a wall-mounted Harpenden stadiometer (Holtain, England). These measures were then used to compute body mass index (BMI).[29]

Other characteristics

Age, education, smoking, medical history, health behaviors (attempting to lose weight, walking for exercise), independent activities of daily living (IADL), and self-reported health were determined by questionnaire and interview at baseline, and current medications, including supplements, were recorded.

Measurement of 25(OH)D

Fasting morning blood was collected, and serum was prepared immediately after phlebotomy and then was stored at −70°C. The samples were batch analyzed by clinic visit. All of the year 6 visit sera were run at the same time. The year 10 visit serum samples were also run as a batch, but at a different time than year 6 visit. Measures for $25(OH)D_2$ (derived from ergocalciferol) and $25(OH)D_3$ (derived from cholecalciferol) were performed at the Mayo Clinic using liquid chromatography tandem mass spectroscopy (LC-MS/MS) as previously described (ThermoFisher Scientific, Franklin, MA; Applied Biosystems-MDS Sciex, Foster City, CA).[31] Deuterated stable isotope (D_3-25-hydroxyvitamin D) was added to a 0.2-mL serum sample as internal standard; $25(OH)D_2$, $25(OH)D_3$, and the internal standard were extracted using acetonitrile precipitation. The extracts were then further purified online and analyzed by LC-MS/MS using multiple reaction monitoring. Duplicate pooled serum controls were included in every other assay run. $25(OH)D_2$ and $25(OH)D_3$ were quantified and summed for total 25(OH)D. The minimum detectable limit for $25(OH)D_2$ was 4 ng/mL and for $25(OH)D_3$ was 2 ng/mL. For $25(OH)D_2$, the interassay coefficient of variation (CV) ranged from 4.7% to 6.2%, and the intraassay CV ranged from 3.3% to 4.4%. For $25(OH)D_3$, the interassay CV ranged from 5.0% to 6.8%, and the intraassay CV ranged from 2.4% to 4.7%.

Statistical analyses

Baseline characteristics were compared using chi-square for categorical variables and analysis of variance (ANOVA) *F*-test for continuous variables. General linear models least squares means were used to compare weight at year 6 and weight change between year 6 and year 10 visits among those with different 25(OH)D levels. Consistent with a predefined

analysis plan, we divided the population into those with 25(OH)D levels <30 ng/mL or ≥30 ng/mL. This cutoff was chosen because parathyroid hormone (PTH) levels have been shown to plateau and calcium absorption has been found to be most efficient at 25(OH)D levels above 30 ng/mL.[32] Previous 25(OH)D studies in this[30] and other populations[27] have also used this 30 ng/mL cutoff. Because of debate in the field about the most appropriate cutoff for 25(OH)D insufficiency, we also examined a 25(OH)D cutoff of 20 ng/mL[33] and quartiles of 25(OH)D.

We characterized participants into categories based on their weight change between the year 6 and year 10 visits. Those who experienced <5% change in body weight between the two visits were considered to have stable weight. Those who gained ≥5% of their body weight were considered to have gained weight; those who lost ≥5% of their body weight were considered to have lost weight.[34] Possible interactions, including weight change category, baseline BMI, and age, were tested. Because the interaction between 25(OH)D and weight change category was significant, analyses were stratified by weight change category. Results are presented as adjusted means and p values for absolute change in weight (in pounds) and for percentage change in weight. Models were adjusted for baseline weight, age, season, and years of follow-up and then for multiple confounders.

In the women who had 25(OH)D levels determined at both year 6 and year 10 visits, we categorized their change in 25(OH)D levels between visits into the following two categories: (1) remained ≥30 ng/mL at both visits or changed from <30 to ≥30 ng/mL and (2) remained <30 ng/mL at both visits or changed from ≥30 to <30 ng/mL. Using general linear models least squares means, we compared change in weight among women according to their change in 25(OH)D levels. Models were adjusted for baseline weight, age, season, and years of follow-up and then for multiple confounders.

Age, season, and baseline weight and years of follow-up (for longitudinal models) were forced into all final models. We developed specific multivariable models for each weight change strata[35,36] by evaluating several potential confounding factors known to influence hydroxyvitamin D and weight, including demographic (age, race/ethnicity, education), lifestyle (smoking, trying to lose weight), medication/supplement use (estrogen, calcium, hydroxyvitamin D, thiazides), and health status (self-reported health, IADL impairments, walks for exercise, diabetes). Each of these covariates was added individually to the base models. All variables that altered the association between 25(OH)D and weight (>10% difference in adjusted compared to unadjusted weight change or p value <0.10) were further evaluated in general linear models using manual backwards stepwise selection, where age, baseline weight, years of follow-up, and season were forced. Statistically significant variables remained in final models ($p<0.05$).

Statistical analyses were completed using SAS version 9.2 (SAS, Inc., Cary, NC). We considered $p<0.05$ to be significant.

Results

Baseline characteristics

The majority (60.2%) of women remained weight stable (defined as <5% weight change, average of 0.3% weight loss overall) between the year 6 and year 10 visits (Table 1). Close to one quarter of women (27.5%) lost ≥5% of their baseline weight (average loss of 10.4%, or 15.7 pounds) between the two visits, and 12.3% gained ≥5% of their baseline weight (average gain of 9.7%, or 13.6 pounds). Women who lost weight were older (77 years vs. 76 years in weight stable and 75 years in weight gain categories) but did not differ by race and education compared to women who were weight stable or gained weight. Baseline weight and BMI were highest in the weight loss group and lowest in the weight gain group. Slightly more than one third of women in the weight loss group decreased to a lower BMI category; 40% of those in the weight gain group increased into a higher BMI category. Only about a quarter of the women losing weight (28.5%) were actually trying to lose weight; more of the women whose weight was stable or increasing (32.2% and 36.1%, respectively) were trying to lose weight.

Among women not trying to lose weight, the number of women with cancer (20%) was the same in the weight stable (21%) and weight gain (20%) groups ($p=0.93$, data not shown). Women who lost or gained weight had lower self-reported health status than those whose weight was stable, although >80% of participants in each weight category reported good/excellent health. Further, 82% of the women who lost weight when they were not trying to reported good/excellent health; in comparison, 85% of those who maintained weight and 81% of those who gained weight reported good/excellent health ($p=0.04$, data not shown). These data suggest that women who lost weight did not have more cancer or other comorbidities.

Association between baseline hydroxyvitamin D sufficiency and weight change between visits

Women with 25(OH)D levels ≥30 ng/mL had lower baseline weight (141.6 pounds) compared to women with 25(OH)D levels <30 ng/mL (148.6 pounds)($p<0.001$). Overall, women with 25(OH)D levels ≥30 ng/mL had similar changes in weight between visits 6 and 10 (−3.3 pounds) compared to women with 25(OH)D levels <30 ng/mL (−2.8 pounds)($p=0.24$). There was also no difference in 25(OH)D level or the percentage of women with 25(OH)D levels ≥30 ng/mL in the weight loss, stable, or gain populations (Table 1).

As there was a significant interaction between weight change category (loss, stable, gain) and 25(OH)D status ($p<0.0001$), we compared change in weight between the year 6 and year 10 visits according to year 6 25(OH)D status within each weight change category. In the group of women who gained at least 5% of their weight, there was an association between 25(OH)D level and amount of weight gain (Table 2). Women whose 25(OH)D level was ≥30 ng/mL at baseline gained 16.4 pounds (12.2% of baseline weight) between visits (average 4.5 years later) compared to 18.5 pounds (13.9% of baseline weight) in women whose 25(OH)D level was <30 ng/mL ($p=0.04$). In women who were losing weight or whose weight was stable, there was no association between 25(OH)D level and change in weight. We repeated the analysis using a 25(OH)D cutoff of 20 ng/mL and 25(OH)D quartiles, and there was no association noted between 25(OH)D status and change in weight. There was no significant interaction between 25(OH)D status and baseline BMI category (underweight/normal, overweight, obese) or age (data not shown).

Table 1. Baseline Characteristics by Weight Change Category

	Weight loss n=*1281 (27.5%)*	*Weight stable* n=*2807 (60.2%)*	*Weight gain* n=*571 (12.3%)*	p *value*
Hydroxyvitamin D status				
Total 25(OH)D at baseline, mean (SD)	22.6 (9.1)	23.2 (8.8)	23.1 (8.9)	0.13
25(OH)D < 30 ng/mL, *n* (%)	1008 (78.7)	2193 (78.1)	436 (76.4)	0.53
Weight characteristics				
Weight, lbs, mean (SD)	149.5 (28.7)	146.7 (26.4)	143.5 (27.6)	< 0.0001
BMI category, *n* (%)				< 0.0001
< 18.5	17 (1.4)	35 (1.3)	16 (2.9)	
< 24.9	433 (34.5)	1147 (41.4)	251 (44.7)	
25–29.9	481 (38.3)	1049 (37.8)	196 (34.9)	
30+	325 (25.9)	541 (19.5)	99 (17.6)	
Weight change, % baseline–>follow-up, mean (SD)[a]	−10.4 (5.0)	−2.7 (2.7)	9.7 (8.4)	< 0.0001
Trying to lose weight, *n* (%)	364 (28.5)	899 (32.2)	206 (36.1)	0.003
BMI category change, *n* (%)				< 0.0001
Decrease to lower BMI category	428 (35.0)	81 (3.0)	-	
No change in BMI category	795 (65.0)	2425 (88.6)	337 (60.8)	
Increase to higher BMI category	-	231 (8.4)	217 (39.2)	
Height, cm, mean (SD)	158.0 (6.2)	158.9 (5.9)	158.9 (5.7)	< 0.0001
Demographics				
Age, years, mean (SD)	77.0 (4.8)	75.6 (4.1)	75.4 (4.0)	< 0.0001
Race, white, *n* (%)	1278 (99.8)	2797 (99.6)	568 (99.5)	0.60
Education, *n* (%)				
< 12 years	262 (20.5)	505 (18.0)	99 (17.3)	0.12
12 years	524 (40.9)	1123 (40.0)	246 (43.1)	
> 12 years	495 (38.6)	1179 (42.0)	226 (39.6)	
Health history				
Self-reported health, good/excellent, *n* (%)	1050 (82.0)	2392 (85.2)	465 (81.4)	0.01
Smoking status, current, *n* (%)	78 (6.1)	125 (4.5)	36 (6.3)	0.04
Diabetes, *n* (%)	92 (7.2)	109 (3.9)	27 (4.7)	< 0.0001
Cancer, *n* (%)	252 (19.7)	589 (21.0)	119 (20.8)	0.62
Total hip BMD, mean (SD)	0.74 (0.14)	0.75 (0.13)	0.74 (0.13)	0.01
Medications				
Calcium, current use, *n* (%)	582 (45.4)	1285 (45.8)	257 (45.0)	0.94
Estrogen, current use, *n* (%)	215 (16.8)	566 (20.2)	107 (18.7)	0.04
Thiazide, current use, *n* (%)	282 (22.0)	512 (18.2)	106 (18.6)	0.02
Hydroxyvitamin D, current use, *n* (%)	199 (15.5)	431 (15.4)	75 (13.1)	0.36
Functional status				
IADL impairments ≥1, *n* (%)	449 (35.1)	674 (24.1)	155 (27.2)	< 0.0001
Walks for exercise, *n* (%)	564 (44.1)	1598 (57.1)	299 (52.6)	< 0.0001
Walking speed, m/sec, mean (SD)	0.94 (0.22)	1.00 (0.20)	0.98 (0.22)	< 0.0001

Weight change over an average of 4.5 years.
[a]Overall population had a mean weight loss of 1.8% (−2.9 lbs).
BMD, bone mineral density; BMI, body mass index; IADL, independent activities of daily living; SD, standard deviation.

Association between longitudinal change in vitamin D status and change in weight

We examined whether change in 25(OH)D status was associated with change in weight among women who either lost or gained at least 5% of their body weight between year 6 and year 10 (Table 3). Among weight gainers, women with sustained or developed 25(OH)D levels ≥30 ng/mL had less weight gain between year 6 and year 10 visits compared to women with 25(OH)D levels <30 ng/mL (16.3 vs. 14.8 pounds; p = 0.04). In women losing weight, change in 25(OH)D status was not significantly associated with weight change, although there was borderline less weight loss in the women who had persistent or developed 25(OH)D levels of ≥30 ng/mL (−14.4 vs −16.1 pounds, $p = 0.06$).

Discussion

In a population of older women, although baseline weight was higher in women with lower 25(OH)D, we found no overall association between 25(OH)D and change in weight. However, there was a significant interaction between 25(OH)D status and weight change category (gain, loss, stable) over 4.5 years. In other words, the association between 25(OH)D status with weight change was significantly different based on type of weight change (gain, loss, stable). In women gaining weight, 25(OH)D levels ≥30 ng/mL at baseline were associated with less weight gain over 4.5 years compared to those with lower levels. Moreover, in a subset whose 25(OH)D level remained ≥30 ng/mL or whose 25(OH)D level increased from <30 to ≥30 ng/mL over that 4.5 years, there was less weight gain during that same time

Table 2. Association Between Hydroxyvitamin D Status and Weight Change, by Weight Change Category

	n *(%)*	*Weight change, lbs Mean (SE)*	*Weight change, % Mean (SE)*	p *value*[a]
Interaction between hydroxyvitamin D status and weight change category				<0.0001
Loss[b]				
25(OH)D ≥30 ng/mL	273 (21.3)	−15.60 (0.48)	−10.4 (0.3)	0.79
25(OH)D <30 ng/mL	1008 (78.7)	−15.61 (0.25)	−10.3 (0.2)	
Stable[c]				
25(OH)D ≥30 ng/mL	614 (21.9)	0.005 (0.23)	0.02 (0.2)	0.64
25(OH)D <30 ng/mL	2193 (78.1)	−0.059 (0.18)	−0.03 (0.1)	
Gain[d]				
25(OH)D ≥30 ng/mL	135 (23.6)	16.45 (1.17)	12.2 (1.0)	0.04
25(OH)D <30 ng/mL	436 (76.4)	18.47 (0.87)	13.9 (0.7)	

[a]*p* value is for comparison of percent weight change.
[b]Adjusted for baseline weight, age, season, follow-up years, clinic and walking speed.
[c]Adjusted for baseline weight, age, season, follow-up years, and smoking status.
[d]Adjusted for baseline weight, age, season, follow-up years, clinic and smoking status.
SE, standard error.

period. There was no statistically significant associations between 25(OH)D and weight change in those who remained weight stable (<5% weight change) or lost ≥5% of their weight.

Our results extend previous cross-sectional data showing that low 25(OH)D levels are associated with increased body fat and obesity.[1–10] Some have proposed that increased body fat is associated with lower 25(OH)D levels because 25(OH)D accumulates in fat or because of lifestyle differences between obese and nonobese populations.[11,12] However, our longitudinal data in older women gaining weight are consistent with the hypothesis that low 25(OH)D levels could actually predispose to fat accumulation. Hydroxyvitamin D supplementation (along with calcium and whey protein) has been shown to reduce fat mass in animals.[37] *In vitro*, 25(OH)D directly regulates differentiation and gene transcription in adipocytes via the VDR and nuclear VDR (nVDR).[20–22] 1,25 Dihydroxyvitamin D_3 (the vitamin's active form) can induce adipocyte cell death,[38] thus decreasing fat mass. With 25(OH)D insufficiency comes increased PTH secretion, and PTH increases lipogenesis and decreases lipolysis.[18,19]

Several weight loss trials have found that those with higher 25(OH)D levels had more fat and weight loss and improved fat oxidation and total energy expenditure.[16,25] Our population was quite different from a typical weight loss trial, however, in that SOF participants had an average age of 76 years and most were not trying to lose weight. Nevertheless, we also found 25(OH)D was associated with positive effects on body composition (less weight gain among those who gained). Two European trials of 25(OH)D supplementation in overweight and obese adults did not find that 25(OH)D was associated with weight loss over 12 months[39,40]; however, 12 months may not be a sufficiently long time over which to measure 25(OH)D's effects on body weight. Randomized clinical trials have also examined the effect of calcium combined with 25(OH)D on weight change, with mixed findings.[41–43] Postmenopausal women (aged 50–79) in the Women's Health Initiative (WHI) Calcium with Vitamin D (CaD)

Table 3. Longitudinal Association over a Mean of 4.5 Years Between Hydroxyvitamin D Status Change and Weight Change, by Weight Change Category

	n *(%)*	*Weight change, lbs Mean (SE)*	*Weight change, % Mean (SE)*	p *value*[a]
Loss[b]				
Persistent 25(OH)D ≥30 ng/mL or 25(OH)D increase to ≥30 ng/mL by follow-up	85 (32.4)	−14.36 (0.77)	−9.6 (0.5)	0.06
Persistent 25(OH)D <30 ng/mL or 25(OH)D decrease to <30 ng/mL by follow-up	177 (67.6)	−16.15 (0.57)	−10.7 (0.4)	
Gain[c]				
Persistent 25(OH)D ≥30 ng/mL or 25(OH)D increase to ≥30 ng/mL by follow-up	42 (30.2)	14.81 (1.31)	10.6 (0.9)	0.04
Persistent 25(OH)D <30 ng/mL or 25(OH)D decrease to <30 ng/mL by follow-up	97 (69.8)	16.34 (1.08)	11.9 (0.8)	

[a]*p* value is for comparison of percent weight change.
[b]Adjusted for baseline weight, age, season, follow-up years, and functional status.
[c]Adjusted for baseline weight, age, season, follow-up years, history of diabetes, and smoking status.

trial randomized to 1000 mg calcium and 400 IU 25(OH)D supplementation gained 0.3 pounds less annually than those given placebo over 7 years.[43] However, the effects of calcium and 25(OH)D could not be disentangled in the analyses.

Vitamin D level was associated with an average 2 pounds less weight gain over 4.5 years, which may not be clinically important for an individual. However, this statistically significant difference in weight gain, although small, supports our hypothesis that a population decrease in sun exposure, with resultant 25(OH)D deficiency, could lead to increased fat storage constantly rather than just seasonally, causing an increased population prevalence of overweight and obesity. In a recent study, children with low 25(OH)D levels had a 0.1 kg/m^2 greater increase in BMI annually.[44]

In women who lost ≥5% of their weight, the group with the poorest health status, 25(OH)D was not clearly associated with weight change. However, those who had a 25(OH)D level that remained <30 ng/mL or declined to <30 ng/mL had borderline more weight loss than those with a 25(OH)D level ≥30 ng/mL by the end of follow-up. Although we adjusted for several health status measures, this association could be due to unmeasured confounding factors (such as a lower nutritional status in those who are sicker and losing weight) rather than a true association between low 25(OH)D levels and weight loss.

Our study has important strengths. It was set within a large community-based prospective study of older women, with rigorous quality control of weight measurements and covariate measurements. Retention of survivors was excellent, with well over 95% completion of follow-up information, including women who became too frail to attend subsequent visits in person. We were able to adjust for several important covariates, including baseline weight and season of 25(OH)D measurement.

Our study also has several limitations. It was conducted in postmenopausal white women aged ≥65 years and may not be generalizable to men, other ethnic groups, and younger women. The women in the study were also exceptionally healthy, and the findings may not apply to those with poorer health. However, it provides important information for a group with a high prevalence of 25(OH)D deficiency—older white women. We did not measure PTH, which has been shown to be associated with both 25(OH)D levels and adiposity. We had follow-up 25(OH)D levels in a subset of women, so our ability to determine if change in 25(OH)D level was related to change in weight was more limited.

Vitamin D insufficiency is rampant in the United States, and it increases with aging.[27,45,46] As our cohort similarly had a high prevalence (78%) of 25(OH)D <30 ng/mL, our ability to compare to women with normal 25(OH)D levels was limited, and it is possible we underestimated the effects of 25(OH)D on weight change. Future studies to further evaluate the impact of 25(OH)D status on long-term weight change in other populations, such as younger women and men and those with comorbidities, are important next steps.

Conclusions

Vitamin D deficiency is becoming a worldwide problem,[47] and filling the knowledge gaps in 25(OH)D research is critical. How 25(OH)D relates to body composition and obesity is an important area of research, especially given the growing threat obesity poses to public health. Our data support the need for improved understanding of how 25(OH)D is associated with body weight in older women, especially given that 25(OH)D repletion is easy, well-tolerated, and inexpensive.[48–50]

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Disclosure Statement

E.S.L. receives funding from Amgen pharmaceuticals, which does not represent a competing financial interest. M.H. served on an advisory board for and owns stock in Theralogix LLC, a company that markets nutritional supplements (including vitamin D). Compensation for 2011 was less than $10,000. There are no competing financial interests for the rest of the authors.

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