Vitamin D deficiency and anthropometric indicators of adiposity in school-age children: a prospective study[1–3]

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ABSTRACT

Background: Cross-sectional studies have indicated that vitamin D serostatus is inversely associated with adiposity. It is unknown whether vitamin D deficiency is a risk factor for the development of adiposity in children.

Objective: We investigated the associations between vitamin D serostatus and changes in body mass index (BMI; in kg/m^2), skinfold-thickness ratio (subscapular-to-triceps), waist circumference, and height in a longitudinal study in children from Bogota, Colombia.

Design: We quantified plasma 25-hydroxyvitamin D [25(OH)D] concentrations in baseline samples of a randomly selected group of 479 schoolchildren aged 5–12 y and classified vitamin D status as deficient [25(OH)D concentrations $<$50 nmol/L], insufficient [25(OH)D concentrations $\geq$50 and $<$75 nmol/L], or sufficient [25(OH)D concentrations $\geq$75 nmol/L]. We measured anthropometric variables annually for a median of 30 mo. We estimated the average change in each anthropometric indicator according to baseline vitamin D status by using multivariate mixed linear regression models.

Results: Vitamin D–deficient children had an adjusted 0.1/y greater change in BMI than did vitamin D–sufficient children (P for trend = 0.05). Similarly, vitamin D–deficient children had a 0.03/y (95% CI: 0.01, 0.05/y) greater change in subscapular-to-triceps skinfold-thickness ratio and a 0.8 cm/y (95% CI: 0.1, 1.6 cm/y) greater change in waist circumference than did vitamin D–sufficient children. Vitamin D deficiency was related to slower linear growth in girls ($-$0.6 cm/y, P = 0.04) but not in boys (0.3 cm/y, P = 0.34); however, an interaction with sex was not statistically significant.

Conclusion: Vitamin D serostatus was inversely associated with the development of adiposity in school-age children. *Am J Clin Nutr* 2010;92:1446–51.

INTRODUCTION

Many regions worldwide are undergoing a rapid nutrition transition through which obesity-related chronic conditions account for an increasing percentage of the disease burden (1). The rapid increase in the rates of obesity in school-age children (2) is particularly concerning because childhood obesity is a risk factor for obesity (3) and related risk factors for cardiometabolic disease (4) later in life. It is crucial to identify modifiable risk factors that are involved in the early development of adiposity to guide future prevention and treatment efforts.

Vitamin D insufficiency is highly prevalent in the world; it is estimated that $\geq$1 billion people have 25-hydroxyvitamin D [25(OH)D] concentrations consistent with insufficiency ($\leq$75 nmol/L) (5). Even children who live in subtropical climates are at risk of vitamin D deficiency according to recent studies in Brazil (6) and Costa Rica (7).

Inadequate vitamin D status could be a risk factor for childhood obesity. Vitamin D affects lypolysis (8, 9) and adipogenesis (10, 11) in human adipocytes through its role in regulating intracellular calcium concentrations. Cross-sectional studies indicated that plasma 25(OH)D concentrations are inversely associated with body mass index (BMI; in kg/m^2) (12–14) and waist circumference (15, 16) in children. However, the interpretation of these associations is limited because vitamin D can be sequestered out of the blood and into the larger adipose tissue mass of obese subjects because of its hydrophobic properties (17). The cross-sectional nature of previous studies precludes the making of an inference regarding the directionality of the association between vitamin D and adiposity.

We conducted a prospective study to evaluate the associations between vitamin D serostatus assessed in subjects at enrollment and changes in indicators of adiposity, including BMI, subscapular-to-triceps skinfold-thickness ratio, and waist circumference, over 3 y of follow-up in a representative sample of low- and middle-income school-age children from Bogota, Colombia. In addition, we assessed the association between vitamin D serostatus and linear growth.

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SUBJECTS AND METHODS

Study population and field procedures

In February 2006, we recruited 3202 children aged 5–12 y from public schools in Bogota, Colombia as part of an observational longitudinal study in nutrition and health. Details on recruitment procedures and study design were previously published (18). In summary, we used a cluster random-sampling strategy in which clusters were defined as classes of all public primary schools in the city by the end of 2005. Because the public school system enrolled 57% of all primary school children in the city, and that 89% of them came from low- and middle-income socioeconomic backgrounds (19), the study population was representative of low- and middle-income families who lived in Bogota.

At the time of enrollment, we distributed a self-administered questionnaire to parents through which we collected information on sociodemographic characteristics including age, parity, education level, and household socioeconomic status. The response rate for the survey was 81%. During the following weeks, trained research assistants visited the schools to obtain anthropometric measurements and fasting blood samples from the children. Height was measured without shoes to the nearest 1 mm with a wall-mounted portable Seca 202 stadiometer (Seca, Hanover, MD), weight was measured in light clothing to the nearest 0.1 kg on Tanita HS301 solar-powered electronic scales (Tanita, Arlington Heights, IL), skinfold thicknesses were measured to the nearest 0.5 mm with SlimGuide Skinfold Calipers (Creative Health Products Inc, Plymouth, MI), and waist circumference was measured to the nearest 1 mm with a nonextensible measuring tape at the level of the umbilicus according to standard protocols (20). Follow-up anthropometric measurements were obtained in June and November 2006 and once yearly thereafter by visiting the schools or homes of the children when they were absent from school on the day of assessment.

The parents or primary caregivers of all children provided written informed consent before enrollment in the study. The study protocol was approved by the Ethics Committee of the National University of Colombia Medical School; the Institutional Review Board at the Harvard School of Public Health approved the use of data from the study.

Laboratory methods

Blood samples were collected by venipuncture in 2816 (88%) children at baseline. On the day of collection, the samples were protected from sunlight, ice packed, and transported to the National Institutes of Health (Bogota, Colombia) where plasma was separated from an EDTA-coated aliquot and cryopreserved at −70°C until transportation to the Harvard School of Public Health. We randomly selected 479 samples for quantification of 25(OH)D, which is a valid biomarker of vitamin D status (17), at the Clinical and Epidemiologic Research Laboratory of the Children's Hospital Boston (Boston, MA). Plasma 25(OH)D concentrations were quantified by an enzyme immunoassay (Immunodiagnostic Systems Inc, Fountain Hills, AZ) that uses a competitive binding technique. The sensitivity of the assay was 5 nmol 25(OH)D/L. The intraclass CV was 5.3% at 39 nmol 25(OH)D/L, 5.6% at 67.1 nmol 25(OH)D/L, and 6.7% at 165 nmol 25(OH)D/L. The interclass CV was 4.6% at 40.3 nmol 25(OH)D/L, 6.4% at 72.0 nmol 25(OH)D/L, and 8.7% at 132 nmol 25(OH)D/L. All samples were analyzed in duplicate.

Data analyses

We categorized vitamin D status, which was the main exposure of interest, as deficient [25(OH)D concentrations <50 nmol/L], insufficient [25(OH)D concentrations ≥ 50 and <75 nmol/L], or sufficient [25(OH)D concentrations ≥ 75 nmol/L] (21).

The primary outcomes were changes in BMI as an indicator of overall adiposity (22), subcapular-to-triceps skinfold-thickness ratio as an indicator of truncal adiposity (23), and waist circumference as an indicator of central adiposity (24). We also examined associations with changes in height.

We compared the distribution of 25(OH)D concentrations by categories of child and maternal characteristics to determine whether those characteristics could possibly confound the associations between vitamin D serostatus and the outcomes of interest. Children were classified according to their BMI as thin, healthy, or overweight or obese by using the International Obesity Task Force recommendations (25, 26). Maternal BMI was calculated from measured height and weight in 26.1% of the mothers and from self-reported data otherwise. Maternal weight status was classified according to BMI categories as underweight (<18.5), adequate (18.5–24.9), overweight (25.0–29.9), or obese (≥ 30.0) (27). Comparisons were made with the use of Wilcoxon's rank-sum test. For ordinal characteristics, a test of trend was obtained from linear regression models with robust estimates of variance (28).

We examined the associations between baseline vitamin D serostatus and the anthropometric outcomes. We compared the mean changes in BMI, skinfold-thickness ratio, waist circumference, and height during follow-up between vitamin D categories with the use of mixed effects models (PROC MIXED, Statistical Analyses System software, version 9.1; SAS Institute, Cary, NC) (29). In each model, the anthropometric measure was the outcome, whereas predictors included indicator variables for vitamin D serostatus categories, age in decimal years, and vitamin D serostatus category × age interactions. These mixed models included random effects for the intercept and slope; we specified an unstructured variance-covariance matrix for these random effects (30). These methods do not require an even number of observations or that measurements be collected at exactly the same time in all subjects; thus, all measurements available for every child were included in the analyses. For the waist-circumference model, random effects for the slope were not included because measurements were only obtained in the second and third years of follow-up; the change in waist circumference represented the change between the 2 measurements taken during follow-up. Models were adjusted for sex, menarcheal status in girls, baseline age, and baseline BMI-for-age z scores (31) as a means to decrease variability in vitamin D status because of adiposity at recruitment. Models for the skinfold-thickness ratio and height were also adjusted for baseline skinfold-thickness ratio and height-for-age z score (31), respectively. Because there is some evidence that vitamin D may interact with estrogen (32), sex-specific estimates were obtained by analyzing boys and girls separately. Models in girls were adjusted for menarcheal status at baseline to control for the potential variability in sexual-maturation stage. Other variables

that were related to overweight in this population (33) or to 25 (OH)D concentrations in univariate analyses including maternal characteristics, socioeconomic-status indicators, and time spent playing outdoors were entered into the models. However, none of these variables were significantly related to the outcomes or changed the estimates of association between vitamin D status and anthropometric change. All models used empirical estimates of the variance (28). Tests for trend were estimated by introducing a continuous variable into the models that represented ordinal categories of vitamin D. Effect modification by sex was assessed with the use of the likelihood ratio test in each model.

We conducted supplemental analyses in which participants who were thin at baseline were excluded to rule out potential associations because of catch-up growth of malnourished children. We also examined the effect of excluding children who were obese at baseline. In addition, we conducted analyses restricted to young children who were unlikely to have entered puberty at the time of vitamin D assessment (<9 y of age for girls and <10 y of age for boys) to assess potential confounding by sexual-maturation status. Results from these analyses were similar to those including all children; thus, only the latter are presented.

All analyses were carried out with the use of the Statistical Analyses System software (version 9.1; SAS Institute Inc).

RESULTS

At baseline, the mean (±SD) age of children was 8.9 ± 1.6 y; 52% of the children were girls. The prevalence of overweight was 11%, and the mean BMI *z* score was 0.1 ± 0.9 in girls and 0.2 ± 1.1 in boys. These children did not differ from the rest of participants in the cohort with regards to baseline sociodemographic or anthropometric characteristics or duration of follow-up. Mean vitamin D concentrations were 73.2 ± 19.8 nmol/L; 10.2% of children were vitamin D-deficient, and another 46.4% of children were insufficient. Plasma 25(OH)D concentrations of the children were significantly and inversely associated with female sex, age, and weight status and with maternal parity and BMI (**Table 1**).

The mean (±SD) follow-up time was 29.0 ± 5.1 mo during which each child contributed a mean of 4.4 ± 0.7 measurements (1.9 ± 0.3 measurements for waist circumference).

Vitamin D status was inversely associated with a change in BMI during follow-up (**Table 2**). Compared with vitamin D–sufficient children, the BMI change was 0.1/y greater in insufficient and deficient children (*P* for trend = 0.05). The association did not significantly vary by sex (*P* for interaction = 0.96). Vitamin D deficiency was also associated with a mean 0.03 larger annual change in subscapular-to-triceps skinfold ratio (*P* = 0.003; *P* test for trend = 0.01) (Table 2). In addition, vitamin D deficiency was associated with an average greater change in waist circumference of 0.8 cm/y (*P* = 0.03; *P* test for trend = 0.05) (Table 2). There was not a significant interaction between vitamin D status and sex in relation to skinfold-thickness ratios (*P* for interaction = 0.63) or waist circumference (*P* for interaction = 0.74).

Vitamin D status was not associated with linear growth in all children (**Table 3**); however analyses stratified by sex suggested an association between vitamin D deficiency and slower growth in girls (adjusted difference = −0.6 cm/y; 95% CI = −1.1, 0.0 cm/y;

TABLE 1
Plasma 25-hydroxyvitamin D [25(OH)D] concentrations in school-age children from Bogota, Colombia, according to sociodemographic and anthropometric characteristics

Characteristic	n[1]	25(OH)D concentration[2]	P[3]
		nmol/L	
Overall	479	73.2 ± 19.8	—
Child characteristics			
Child sex			<0.01
F	250	70.8 ± 18.3	—
M	229	75.9 ± 21.0	—
Age			<0.0001
5–6 y	73	80.0 ± 24.5	—
7–8 y	158	75.1 ± 20.2	—
9–10 y	207	70.3 ± 16.7	—
11–12 y	41	68.2 ± 19.7	—
Born in Bogota			0.55
Yes	397	72.9 ± 20.2	—
No	48	73.9 ± 19.0	—
Height-for-age *z* score[4]			0.72
Less than −2.0	44	68.7 ± 18.3	—
−2.0 to −1.1	157	75.9 ± 19.5	—
−1.0 to 0.9	259	71.8 ± 18.5	—
≥1.0	19	80.4 ± 34.9	—
Weight status[5]			0.03
Thin	44	74.1 ± 23.9	—
Adequate	378	74.1 ± 19.7	—
Overweight or obese	54	66.1 ± 15.9	—
Time spent playing outside			0.93
≤2.0 h/wk	123	73.5 ± 22.7	—
2.1–5.0 h/wk	67	69.4 ± 16.9	—
5.1–8.0 h/wk	68	73.3 ± 20.1	—
>8.0 h/wk	116	73.1 ± 18.3	—
Maternal characteristics			
Education			0.97
Primary or less (≤5 y)	128	73.8 ± 20.6	—
Incomplete secondary (6–10 y)	106	71.4 ± 17.5	—
Complete secondary or university (≥11 y)	210	73.6 ± 20.9	—
Parity			0.03
1	40	74.1 ± 19.0	—
2	168	75.8 ± 22.7	—
3	134	71.7 ± 17.8	—
4	57	72.5 ± 17.5	—
≥5	38	67.6 ± 16.3	—
BMI			0.02
<18.5 kg/m^2	16	77.0 ± 22.1	—
18.5–24.9 kg/m^2	243	74.5 ± 22.0	—
25.0–29.9 kg/m^2	117	71.5 ± 16.7	—
≥30.0 kg/m^2	36	67.3 ± 13.9	—
Household socioeconomic stratum[6]			0.24
1 (lowest)	34	72.3 ± 17.0	—
2	178	71.6 ± 20.1	—
3 or 4	235	74.3 ± 20.3	—

[1] Totals may be <479 because of missing values.
[2] Values are means ± SDs.
[3] From Wilcoxon's rank-sum test for child's sex and place of birth. For all other predictors (ordinal), *P* was for a test of linear trend when a variable that represented the ordinal categories of the predictor was introduced into a linear regression model as continuous (Wald test).
[4] According to World Health Organization 2007 child-growth reference (31).
[5] According to International Obesity Task Force criteria (25, 26)
[6] According to the city's classification of public service fees of neighborhoods.

TABLE 2
Change in adiposity indicators in school-age children from Bogota, Colombia, according to vitamin D status[1]

	Vitamin D status			
	Sufficient (n = 208)	Insufficient (n = 222)	Deficient (n = 49)	P for trend[2]
Baseline BMI (kg/m^2)[3]	16.3 ± 1.9	16.9 ± 2.1	17.2 ± 2.3	—
Change in BMI (kg/m^2 per y)[4]	0.5 ± 0.0	0.6 ± 0.0	0.6 ± 0.1	—
Unadjusted difference (95% CI)	Reference	0.1 (0.0, 0.2)	0.1 (−0.1, 0.3)	0.10
Adjusted difference (95% CI)	Reference	0.1 (0.0, 0.2)	0.1 (0.0, 0.3)	0.05
Baseline skinfold-thickness ratio[3]	0.71 ± 0.17	0.71 ± 0.19	0.72 ± 0.17	—
Change in skinfold-thickness ratio (/y)[4]	0.01 ± 0.00	0.02 ± 0.00	0.04 ± 0.01	—
Unadjusted difference (95% CI)	Reference	0.01 (0.00, 0.02)	0.03 (0.01, 0.05)	0.004
Adjusted difference (95% CI)	Reference	0.01 (−0.01, 0.02)	0.03 (0.01, 0.05)	0.01
Change in waist circumference (cm/y)[4]	2.8 ± 0.2	2.9 ± 0.2	3.3 ± 0.4	—
Unadjusted difference (95% CI)	Reference	0.1 (−0.4, 0.7)	0.5 (−0.4, 1.4)	0.30
Adjusted difference (95% CI)	Reference	0.1 (−0.4, 0.6)	0.8 (0.1, 1.6)	0.05

[1] Classifications: deficient [<50 nmol 25(OH)D/L], insufficient [≥50 and <75 nmol 25(OH)D/L], and sufficient [≥75 nmol 25(OH)D/L]. Estimated mean (±SE) values and unadjusted differences are from mixed-effects linear regression models with the adiposity measure as the outcome and predictors that included indicator variables for vitamin D categories, time (age in decimal years), and vitamin D × age interaction terms. Random effects included subject-specific intercepts and slopes. An unstructured variance-covariance matrix was specified for subject-specific intercepts and trends. Adjusted models also included baseline age and BMI-for-age z score, sex, menarcheal status in girls, and a sex × age interaction term as predictors. The adjusted model for skinfold-thickness ratio also controlled for baseline skinfold-thickness ratio.

[2] For a test of linear trend when a variable that represented the ordinal categories of vitamin D status was introduced into a linear regression model as a continuous predictor (Wald test).

[3] Values are means ± SDs.

[4] Values are means ± SEs.

P = 0.04) but not in boys (adjusted difference = 0.3 cm/y; 95% CI = −0.3, 0.8 cm/y; P = 0.34); the interaction was not significant (P for interaction = 0.18).

DISCUSSION

We examined the associations between vitamin D serostatus and changes in anthropometric indicators of total adiposity and fat distribution, as well as height, in a longitudinal study of school-age children from Bogota, Colombia. After baseline adiposity and other potential confounders were controlled for, a lower vitamin D serostatus was associated with greater increases in BMI and indexes of central adiposity.

Although cross-sectional studies reported inverse associations between vitamin D serostatus and BMI in children (12–14), it was not possible to conclude that vitamin D increased the risk of overweight because of reverse-causation bias given that vitamin D can be sequestered in adipose tissue (17). Only randomized trials or longitudinal studies that were adjusted for baseline adiposity could overcome the potential for reverse causation. Our results suggested that inadequate vitamin D status may prospectively lead to increased adiposity during childhood. It is unlikely that these findings reflect the catch-up growth of malnourished children because the exclusion of children who were thin at baseline did not alter results. In addition, the mean baseline BMI z score was above zero, and the BMI trends by

TABLE 3
Height in school-age children from Bogota, Colombia, according to vitamin D status[1]

	Vitamin D status			
	Sufficient	Insufficient	Deficient	P for trend[2]
Total (n)	208	222	49	—
Baseline height (cm)[3]	125.7 ± 10.3	128.7 ± 10.0	131.0 ± 11.0	—
Change in height (cm/y)[4]	5.8 ± 0.1	6.2 ± 0.1	5.6 ± 0.2	—
Unadjusted difference (95% CI)	Reference	0.4 (0.1, 0.7)	−0.3 (−0.8, 0.2)	0.69
Adjusted difference (95% CI)	Reference	0.3 (0.1, 0.5)	−0.2 (−0.6, 0.2)	0.78

[1] Classifications: deficient [<50 nmol 25(OH)D/L], insufficient [≥50 and <75 nmol 25(OH)D/L], and sufficient [≥75 nmol 25(OH)D/L]. Estimated mean (±SE) values are from mixed-effects linear regression models with height as the outcome and predictors that included indicator variables for vitamin D categories, time (age in decimal years), and vitamin D × age interaction terms. Random effects included subject-specific intercepts and slopes. An unstructured variance-covariance matrix was specified for subject-specific intercepts and trends. Adjusted models also included baseline age, baseline BMI and height-for-age z scores, sex, a sex × age interaction term, and menarcheal status in girls as predictors.

[2] For a test of linear trend when a variable that represented the ordinal categories of vitamin D status was introduced into a linear regression model as a continuous predictor (Wald test).

[3] Values are means ± SDs.

[4] Values are means ± SEs.

age in this population were very close to the reference median of the World Health Organization (31); thus, greater BMI changes in vitamin D–deficient children likely represent unhealthy weight gains.

There is limited and inconsistent evidence of the association between vitamin D and adiposity from prospective studies. In a calcium-intervention trial in 69 pubertal children, a higher baseline vitamin D status was significantly associated with less weight gain over 24 mo in univariate analyses (34). The Women's Health Initiative, which is a large trial that assigned women to receive either 1000 mg Ca plus 400 IU vitamin D/d or a placebo reported that women who received the regimen of calcium and vitamin D had a small significant lower gain in BMI and waist circumference over 7 y of follow-up (35); in that study, it was impossible to separate the effects of calcium and vitamin D. In contrast, a trial of overweight adults showed that supplementation with either 20,000 or 40,000 IU cholecalciferol/wk and 500 mg Ca/d did not lead to significantly greater weight loss than the calcium-only control group (36); however, none of the groups experienced significant weight changes over the 1-y study period. It is possible that vitamin D may limit weight gain but does not affect long-term weight loss in individuals who are already overweight.

The mechanisms by which vitamin D may influence adiposity are unknown, and possible explanations are still speculative. In vitro experiments suggested that vitamin D may prospectively influence the risk of obesity by modulating the catabolic (8, 9) and anabolic (10, 11) activity of adipocytes. Studies have shown that intracellular calcium concentrations modulate lipolytic activity in isolated human adipocytes (8, 9), which raises the possibility that vitamin D could influence body weight and energy expenditure through calcium regulation. In vitro studies have also shown that vitamin D can inhibit the expression of a key adipogenesis regulator, peroxisome proliferator-activated receptor-gamma (10, 11).

In our longitudinal study, we showed that vitamin D status was negatively associated with changes in waist circumference and subscapular-to-triceps skinfold-thickness ratio after adjustment for baseline adiposity. These findings are particularly worrisome because central adiposity is strongly related to all components of metabolic syndrome in children, including hypertension (24, 37) and insulin resistance (37). Furthermore, children who accumulate central body fat may be at greater risk of central adiposity and its associated morbidities later in life (38).

Our results suggested that vitamin D status may have been positively related to linear growth in girls, although an interaction with sex was not significant. Previous studies reported a clear association between vitamin D–deficiency rickets and linear-growth retardation (39), but the relation between vitamin D and linear growth is less clear in children who do not have clinical manifestations of deficiency. Some (40, 41), but not all (42), cross sectional studies of apparently healthy adolescent girls and young women reported significant, positive associations between serum 25(OH)D concentrations and height. These studies were limited by potential reverse causation and confounding by age and other characteristics. A calcium intervention trial in 69 children showed that baseline vitamin D status was significantly and positively associated with height gain in healthy pubertal children in unadjusted analyses (34). Our results also suggest that vitamin D status may be positively associated with linear growth in the absence of clinical rickets in girls.

Incidentally, we observed a high prevalence of vitamin D insufficiency in this population, which is representative of low- and middle-income children from Bogota, Colombia. The concentrations of vitamin D insufficiency in our population were similar to those reported for adolescents from Sao Paulo, Brazil (6), but twice as high as those reported for children from Costa Rica (7). Although Bogota is located at a subtropical latitude of 4°34′N, the children there may be at particularly high risk of vitamin D insufficiency because the mountainous topography results in a cool climate that may lead to less ultraviolet-B skin exposure. The recent increase in air pollution could also limit ultraviolet-B skin exposure (43). The potential for vitamin D deficiency in this population is compounded by the fact that dairy products are not systematically fortified with vitamin D in Colombia.

Our study had several strengths. We collected blood samples from a large and representative sample of children in a setting where the increasing prevalence of child overweight is becoming a serious public-health problem. Our prospective design and use of repeated anthropometric measures enhanced our ability to explore the temporal relation between vitamin D and anthropometric measures and to account for potential reverse causation. One potential limitation of the study is that we assumed that baseline vitamin D serostatus was representative of the cumulative exposure during follow-up. Nevertheless, studies in which 25(OH)D has been measured repeatedly over long periods suggest that the within-subject correlation is high and that a single baseline measurement could be a valid indicator of long-term exposure (44). Outdoor physical activity could be a confounder of the association between vitamin D status and weight change. However, we did not find that adjustment for the time spent playing outdoors changed the estimates of association in our study; in addition, prospective studies suggested that the association between physical activity and BMI change in children is not strong (45). Another limitation is that we lacked detailed measurements of sexual maturation status (such as Tanner staging), which might be a potential confounder; however, adjustment for menarcheal status at baseline did not change the associations observed in girls.

In conclusion, vitamin D serostatus was inversely associated with the development of adiposity in school-age children. Randomized intervention studies are needed to ascertain the effect of improving vitamin D status in children on the risk of obesity and other risk factors for chronic disease.

The authors' responsibilities were as follows—DG-D, AB, MM-P, CM, JEA, and EV: contributed to the study design; AB, MMP, CM, JEA, and EV: contributed to the data collection; DG-D, MH, WCW, and EV: participated in statistical data analyses. DG-D: conducted data analyses, interpreted results, and wrote the initial draft of the manuscript; and DG-D, AB, MH, WCW, and EV: participated in the data interpretation and helped to write the final draft of the manuscript. None of the authors declared a conflict of interest.

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