Vitamin D intake is inversely related to risk of developing metabolic syndrome in African American and white men and women over 20 y: the Coronary Artery Risk Development in Young Adults study[1–3]

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ABSTRACT

Background: Vitamin D intake may play a key role in the prevention of cardiovascular disease.

Objective: We evaluated associations of dietary and supplemental vitamin D intake with the 20-y incidence of metabolic syndrome.

Design: Data from 4727 black and white young men and women from the Coronary Artery Risk Development in Young Adults study were used to examine relations of dietary plus supplemental vitamin D intake with the incidence of metabolic syndrome (as defined by Adult Treatment Panel, third report, guidelines) and the prevalence of its components, including abdominal obesity, elevated blood pressure, and high glucose, low HDL, and high triglyceride concentrations.

Results: The intake of vitamin D from dietary and supplemental sources was inversely related to the 20-y cumulative prevalence of abdominal obesity ($P = 0.05$) and high glucose ($P = 0.02$) and low HDL ($P = 0.004$) concentrations after adjustment for age, sex, race, education, center, and energy intake. In comparison with the lowest intake quintile (quintile 1), HRs (95% CIs) of developing incident metabolic syndrome for quintiles 2–5 of vitamin D intake were 0.82 (0.67, 1.00), 0.84 (0.68, 1.03), 0.70 (0.56, 0.88), and 0.82 (95% CI: 0.65, 1.02), respectively (P-trend = 0.03) after adjustment for demographic and lifestyle factors.

Conclusions: In young adults, the dietary plus supplemental vitamin D intake was inversely related to the development of incident metabolic syndrome over 20 y of follow-up. These findings support the recommendations of the Dietary Guidelines for Americans to increase intakes of vitamin D–rich foods, such as milk and fish. *Am J Clin Nutr* 2012;96:24–9.

INTRODUCTION

Metabolic syndrome is a known precursor to cardiovascular events (1). Components of metabolic syndrome such as abdominal obesity and high blood pressure have shown an inverse relation with serum 25-hydroxyvitamin D and dietary vitamin D in several studies (2–7), whereas no relations with each have been reported in other studies (8, 9). Studies have also shown both inverse (2, 7, 10, 11) and no (7, 12) associations between high triglyceride, high blood glucose, low HDL, and serum 25-hydroxyvitamin D or dietary vitamin D concentrations. In a prospective study, baseline serum 25-hydroxyvitamin D was shown to be inversely related to metabolic syndrome after 10 y of follow-up (8); however, to our knowledge, there have been no published studies that investigated the relation between the dietary intake of vitamin D and incident metabolic syndrome over several years of follow-up.

Dietary and supplemental vitamin D intakes have been inversely associated with cardiovascular disease (CVD) risk (13–16), metabolic syndrome, and its components (7). For example, dietary vitamin D was inversely and significantly related to BMI in a cross-sectional study in both men and women (13). In another cross-sectional study, Liu et al observed that predominantly white middle-aged women in the highest category of total vitamin D and calcium intakes had the lowest prevalence of each of the components of metabolic syndrome, although the association of dietary vitamin D and calcium with high triglycerides was not significant (7). In a prospective study, women ages 30–55 y who consumed more calcium plus vitamin D had a lower risk of developing incident diabetes (14). However, this relation was attenuated after adjustment for calcium, magnesium, and other dietary factors.

Not all studies, however, have observed significant relations between dietary vitamin D intake and CVD risk factors or metabolic syndrome (7, 14). Differences in findings were potentially attributed to different study designs, most of which were cross-sectional designs, and diet data-collection instruments, study populations, most of which were middle-aged populations, white adults with higher risk of chronic disease, and the use of different cutoffs for dietary exposures (17). Relations between the dietary and supplemental intake of vitamin D and risk of the development of metabolic syndrome and prevalence of components were examined and

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compared after 20 y. On the basis of data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, we hypothesized that a greater intake of dietary and supplemental vitamin D would be associated with lower risk of the development of metabolic syndrome and its components in young African American and white men and women at baseline who were followed for 20 y.

SUBJECTS AND METHODS

Study participants

The CARDIA study is a multisite, population-based, longitudinal study that examines the evolution of coronary artery disease risk factors in young black and white adults. At baseline (1985–1986), 5115 black (52%) and white (48%) participants between ages 18–30 y enrolled in the study. The study population consisted of men (46%) and women (54%) of whom 40% had a high-school education or less. The recruitment of study participants occurred in one of the 4 clinical centers located in Birmingham, AL, Chicago, IL, Minneapolis, MN, and Oakland, CA. Reexaminations occurred at years 2, 5, 7, 10, 15, and 20 with retention rates of 91%, 86%, 81%, 79%, 74%, and 72%, respectively. Details of study participants and recruitment have been previously reported (18, 19). After the study protocol was approved by institutional review boards at all sites, all participants signed written informed consent forms for each examination. Data were excluded for participants who had prevalent metabolic syndrome ($\geq$3 components of metabolic syndrome at baseline; n = 110), women who were pregnant at the time of examination (n = 7), participants who did not complete baseline dietary intakes or had missing measures of metabolic syndrome components (n = 185), and subjects who consumed extremely low ($<$600 kcal for women and $<$800 kcal for men) or extremely high ($>$6000 kcal for women and $>$8000 kcal for men) energy intakes (n = 108).

Dietary assessment

The CARDIA diet history has been previously described (20). Briefly, the CARDIA diet history, which reflects the usual dietary intake in the past 30 d, was interviewer administered at baseline and year 7 to obtain the intake frequency and portion sizes of 100 food and beverage categories, including major food and beverage sources of vitamin D and vitamin and mineral supplements. Information obtained of vitamin and mineral supplements included brand names (if available), frequency, and quantity consumed. A validation study was conducted at baseline to test the accuracy of the diet-history questionnaire (21). Correlation coefficients for calcium intakes between the diet history and seven 24-h recalls ranged from 0.66 to 0.80 in black and white men and women (21). Although the correlation between the diet history and 24-h recall interviews for vitamin D intake was not reported, the major contributor to vitamin D and calcium intakes was milk. Therefore, it is likely that the correlation for vitamin D between diet instruments was similar to that of calcium.

Relation between dietary and serum vitamin D in a subsample of CARDIA participants

Serum 25-hydroxyvitamin D concentrations were measured in a sample of 402 study participants aged 25–36 y, including 109 black men, 114 white men, 95 black women, and 84 white women, enrolled at year 7 (June 1991 to May 1993) at the Oakland CARDIA field center, who were participating in a study of bone mineral homeostasis (22). After adjustment for age, race, and sex, a significant but weak correlation was observed between serum 25-hydroxyvitamin D and dietary vitamin D (r = 0.13, P = 0.016). Dietary and supplemental vitamin D has been a major determinant of plasma 25-hydroxyvitamin D concentrations and vitamin D biomarkers in other studies (23–25).

Clinical measurements

Before each CARDIA examination, participants were asked to fast for $\geq$8 h and refrain from smoking and heavy physical activity 2 h before the exam. Waist circumferences were laterally measured in centimeters halfway between the iliac crest and the lowest portion of the rib cage. Measurements began and ended between the umbilicus and xyphoid process of the sternum. Blood pressure was measured by using the right arm with a Hawksley random 0 sphygmomanometer (WA Baum Company). Participants were seated and blood pressure was measured after a 5-min rest 3 times with 1-min intervals in between. Values were averaged from the second and third measurements. Plasma, serum, and whole blood samples were drawn from the antecubital vein. HDL cholesterol was measured after dextran-magnesium precipitation (26), and LDL concentrations were calculated by using Friedewald's equation (27). Triglycerides were also enzymatically measured by using a blank corrected method.

As defined by Adult Treatment Panel, third report, guidelines (28), metabolic syndrome included 3 of the following 5 components: abdominal obesity ($>$102 cm for men; $>$88 cm for women), elevated blood pressure (systolic blood pressure $\geq$130 mm Hg or diastolic blood pressure $\geq$85 mm Hg or taking antihypertensive medication), low HDL cholesterol (HDL concentrations $<$40 mg/dL for men and $<$50 mg/dL for women), high triglyceride concentrations ($\geq$150 mg/dL), and high glucose concentrations [fasting glucose concentrations $\geq$110 mg/dL or diagnosed with diabetes (ie, having blood glucose concentrations $\geq$26 mg/dL or reported use of an oral hypoglycemic medication or insulin at examinations)].

Other measures

Demographics (age, sex, race, education, and field center) and behavioral characteristics (cigarette smoking and physical activity) were measured by using standard questionnaires. Questionnaire data of lifestyle physical activity were used as a proxy to determine the amount of exposure to sunlight and outdoor activity. Smoking behaviors of participants were determined as current smokers, former smokers, or nonsmokers.

Statistical methods

Data were analyzed with SAS (version 9.2; SAS Institute). Quintiles of vitamin D from dietary and supplemental sources reported at baseline are shown in **Table 1**. Means ($\pm$SEs) and frequencies of baseline characteristics were reported according to quintiles of baseline vitamin D intake that were adjusted for age, sex, race, education, field center, and energy intake.

To increase precision, vitamin D intakes from food and supplement sources at years 0 and 7 were averaged and quintiles of the average vitamin D intake were created. Generalized linear

TABLE 1
Selected characteristics across quintiles of daily vitamin D intake at baseline in white and African American young adults without metabolic syndrome (n = 4727)[1]

	Quintiles of daily diet plus supplement vitamin D intake at baseline					
	1	2	3	4	5	*P*-trend
Vitamin D (μg)	<2.94	2.94–5.13	5.14–8.30	8.31–13.5	>13.5	—
n	942	948	947	946	944	—
Demographic characteristics						
Age (y)	25.0 ± 0.12	25.0 ± 0.12	24.8 ± 0.12	24.8 ± 0.12	24.7 ± 0.12	0.05
Sex, M (%)	57.2 ± 1.5	55.7 ± 1.5	51.8 ± 1.4	54.1 ± 1.5	56.6 ± 1.5	0.59
Race, black (%)	62.4 ± 1.5	56.3 ± 1.5	50.5 ± 1.5	43.3 ± 1.5	37.5 ± 1.5	<0.001
Education, high school graduate (%)	60.9 ± 1.6	62.9 ± 1.5	62.3 ± .5	62.0 ± 1.5	60.1 ± 1.6	0.64
Lifestyle characteristics						
Smoking (%)	32.4 ± 1.5	30.4 ± 1.4	32.5 ± 1.4	27.2 ± 1.4	24.1 ± 1.5	<0.001
Physical activity score	401 ± 9.3	401 ± 8.9	414 ± 8.8	415 ± 8.9	461 ± 9.3	<0.001
Energy (kcal/d)	2052 ± 36.8	2544 ± 36.1	2820 ± 36.1	3097 ± 36.2	3679 ± 36.7	<0.001
Milk (servings/d)	0.43 ± 0.05	0.69 ± 0.05	1.27 ± 0.05	1.74 ± 0.05	3.45 ± 0.05	<0.001
Fish (servings/d)	0.76 ± 0.05	0.88 ± 0.04	0.93 ± 0.04	0.93 ± 0.04	1.24 ± 0.05	<0.001
Alcohol (servings/d)	0.22 ± 0.02	0.19 ± 0.02	0.21 ± 0.02	0.18 ± 0.02	0.13 ± 0.02	0.004
Clinical characteristics						
BMI (kg/m^2)	24.7 ± 0.2	24.3 ± 0.2	24.3 ± 0.2	24.0 ± 0.2	24.1 ± 0.2	0.005
Waist circumference (cm)	78.0 ± 0.3	77.1 ± 0.3	77.3 ± 0.3	76.5 ± 0.3	76.8 ± 0.3	0.009
SBP (mm Hg)	110.2 ± 0.3	110.1 ± 0.3	109.8 ± 0.3	109.9 ± 0.3	110.1 ± 0.3	0.67
DBP (mm Hg)	68.5 ± 0.3	68.3 ± 0.3	68.1 ± 0.3	68.5 ± 0.3	68.1 ± 0.3	0.56
HDL cholesterol (mg/dL)	52.9 ± 0.4	53.7 ± 0.4	53.2 ± 0.4	54.2 ± 0.4	53.4 ± 0.4	0.30
Triglycerides (mg/dL)	70.5 ± 1.4	72.2 ± 1.4	70.2 ± 1.4	68.9 ± 1.4	69.3 ± 1.4	0.24
Glucose (mg/dL)	81.9 ± 0.3	81.9 ± 0.3	81.3 ± 0.3	81.5 ± 0.3	81.4 ± 0.3	0.11

[1] Characteristics were adjusted for age, sex, race, education, center, and energy intake. DBP, diastolic blood pressure; SBP, systolic blood pressure.

regression models evaluated the relation of the cumulative 20-y prevalence of metabolic-syndrome components across quintiles of year 0 + 7 dietary and supplemental vitamin D intake.

The 20-y incidence of metabolic syndrome was defined as the first occurrence of metabolic syndrome at years 2, 5, 7, 10, 15, or 20, at which time the follow-up time was terminated for subjects with this event (yes or no). Because the actual time to diagnosis was unknown, the time to metabolic syndrome was interval censored by the schedule of follow-up visits. The follow-up time for those without metabolic syndrome ended at the last attended examination or at year 20. Cox proportional hazards regression analysis was used to evaluate associations of average year 0 + 7 dietary and supplemental vitamin D intake with the 20-y incidence of metabolic syndrome. HRs were computed for the second through fifth quintiles of vitamin D intake compared with the first quintile (lowest intake) as the referent group. Four

TABLE 2
Cumulative prevalence of metabolic syndrome components over 20 y across quintiles of daily dietary and supplemental vitamin D intake (n = 4727)[1]

	Quintiles of daily diet plus supplement intake of vitamin D					
	1	2	3	4	5	*P*-trend
Vitamin D (μg)	0.22–3.70	3.71–6.09	6.10–8.99	9.0–13.5	>13.5	—
n	945	946	945	945	946	—
Abdominal obesity[2]	39.8 ± 1.6[3]	35.8 ± 1.5	38.5 ± 1.5	33.1 ± 1.5	36.0 ± 1.6	0.05
EBP[4]	35.8 ± 1.6	32.9 ± 1.5	33.5 ± 1.5	33.4 ± 1.5	31.1 ± 1.6	0.09
High glucose[5]	13.1 ± 1.1	13.3 ± 1.1	13.1 ± 1.0	9.8 ± 1.1	10.3 ± 1.1	0.01
Low HDL[6]	55.3 ± 1.7	56.1 ± 1.6	56.3 ± 1.6	50.9 ± 1.6	49.6 ± 1.7	0.004
High triglyceride[7]	25.4 ± 1.5	24.9 ± 1.4	28.3 ± 1.4	23.4 ± 1.4	26.8 ± 1.5	0.81

[1] Adjusted for age, sex, race, education, center, and energy intake. Quintiles of daily vitamin D intake are averages of year 0 + 7 dietary and supplement data.
[2] Defined as >102 cm for men and >88 cm for women.
[3] Percentage ± SE (all such values).
[4] EBP, elevated blood pressure (defined as systolic blood pressure ≥130 mm Hg or diastolic blood pressure ≥85 mm Hg or taking antihypertensive medications).
[5] Defined as fasting glucose ≥110 mg/dL.
[6] Defined as <40 mg/dL for men and <50 mg/dL for women.
[7] Defined as fasting triglycerides ≥150 mg/dL.

TABLE 3
HRs of developing incident metabolic syndrome over 20 y of follow-up across quintiles of daily dietary and supplemental vitamin D intake (n = 4727)[1]

	Quintiles of daily diet plus supplement intake of vitamin D					
	1	2	3	4	5	P-trend
Vitamin D (μg)	0.22–3.70	3.71–6.09	6.10–8.99	9.0–13.5	>13.5	—
n	945	946	945	945	946	—
MS[2]	208	181	182	150	168	—
Model 1	1.0 (—)[3]	0.80 (0.66, 0.98)	0.82 (0.67, 1.01)	0.69 (0.55, 0.86)	0.79 (0.63, 0.98)	0.01
Model 2	1.0 (—)	0.82 (0.67, 1.00)	0.84 (0.68, 1.03)	0.70 (0.56, 0.88)	0.82 (0.65, 1.02)	0.03
Model 3	1.0 (—)	0.81 (0.66, 1.00)	0.83 (0.67, 1.02)	0.69 (0.54, 0.86)	0.77 (0.60, 1.00)	0.02

[1] Quintiles of daily vitamin D intake are averages of year 0 + 7 diet and supplement data. Model 1 was adjusted for age, race, sex, education, field center, and total energy. Model 2 was adjusted as for model 1 plus smoking, physical activity, and alcohol. Model 3 was adjusted as for model 2 plus dietary and supplemental calcium.
[2] MS, number of individuals who developed metabolic syndrome over 20 y of follow-up.
[3] HR; 95% CI in parentheses (all such values).

models were developed. Model 1 was adjusted for energy intake and demographic factors, including age, sex, race, education, and field center. Because dietary intake may be related to other behaviors, model 2 was adjusted for model 1 plus lifestyle factors, including smoking, physical activity, and alcohol intake. In model 3, we further adjusted for dietary and supplemental calcium intake. A linear trend across quintiles was tested with contrast statements by using orthogonal polynomial coefficients.

RESULTS

Baseline characteristics adjusted for age, sex, education, study center, and energy intake are presented across quintiles of dietary and supplemental vitamin D intake for participants without metabolic syndrome (Table 1). Black participants consumed less vitamin D from food and supplemental sources than did whites. The mean age, BMI, waist circumference, and frequency of smoking were lower with a greater vitamin D intake at baseline. Milk and fish consumption was greater across quintiles of vitamin D intake (P-trend < 0.001); however, alcohol intake was lower across vitamin D quintiles (P-trend = 0.004). CVD factors, including elevated blood pressure, low HDL, elevated blood triglycerides, and elevated blood glucose, were not related to the vitamin D intake in participants free of metabolic syndrome at baseline. In 4727 participants, 1064 subjects reported the consumption of vitamin and mineral dietary supplements at baseline. The proportion of dietary supplement users was greater across increasing vitamin D intake quintiles 1 through 5 [1.4% (quintile 1), 5.8% (quintile 2), 11.3% (quintile 3), 36.9% (quintile 4), and 57.2% (quintile 5)] ($P < 0.001$).

The prevalence of components of metabolic syndrome by year 20 according to vitamin D intake is shown in **Table 2**. The prevalence of most metabolic syndrome components, including abdominal obesity, high glucose, and low HDL cholesterol, was significantly lower across quintiles of vitamin D intake ($P \leq 0.05$) but not elevated blood pressure or high triglyceride concentrations.

Almost 19% of CARDIA participants (n = 889) developed the metabolic syndrome by year 20. As shown in **Table 3**, there was a significant inverse association between the dietary and supplemental vitamin D intake and risk of developing metabolic syndrome over 20 y after adjusting for demographics and lifestyle characteristics. Compared with individuals who consumed an average of 2.47 μg vitamin D in quintile 1, risk of developing metabolic syndrome was 18% lower in subjects who consumed an average of 20.58 μg vitamin D in quintile 5 (P-trend = 0.03). After additional adjustment for diet plus supplemental calcium, risk of metabolic syndrome remained stable. Adjustment for dietary fiber, saturated fat, and sugar-sweetened beverages did not change the relation (data not shown).

DISCUSSION

The intake of vitamin D from diet and supplement sources was inversely associated with risk of incident metabolic syndrome over 20 y after adjustment for demographic characteristics, lifestyle factors, and calcium intake from diet and supplement sources. There was no significant effect modification of race or sex on the relation between vitamin D intake and risk of metabolic syndrome. Individuals free of metabolic syndrome at baseline with higher vitamin D intakes consumed vitamin D supplements and a greater number of servings from milk and fish. Subjects who consumed higher amounts of total (dietary plus supplemental) vitamin D were younger, nonsmokers, and white. These subjects also had higher physical activity scores and lower BMI and waist circumferences. We also observed a lower prevalence of components for metabolic syndrome across increasing quintiles of total vitamin D intake.

In our study, dietary and supplemental vitamin D intake was generally beneficially related to components of metabolic syndrome. Our findings showed significant and inverse relations of waist and BMI levels across quintiles of vitamin D intake. Consistent with our results, an inverse relation was also observed between dietary vitamin D intake and waist circumference in a cross-sectional study that included 39,876 female health professionals ≥45 y of age from the Women's Health Study (7). In 2 cross-sectional studies of serum 25-hydroxyvitamin D, a greater vitamin D intake was significantly associated with a lower prevalence of abdominal obesity in 6810 white men from a British cohort and in 8421 noninstitutionalized US adults aged ≥20 y enrolled in NHANES III (9, 29).

Our findings showed a lower 20-y cumulative prevalence of low HDL cholesterol with a greater intake of total vitamin D. In cross-sectional studies, significant positive relations of serum

25-hydroxyvitamin D and dietary vitamin D with concentrations of HDL cholesterol were observed in women aged ≥45 y and NHANES adults >20 y (7, 30). In the study of middle-aged women, statistical models were not adjusted for sun exposure; however, a greater vitamin D intake was related to higher physical activity (7). We also observed no relation between blood pressure and triglycerides across increasing quintiles of vitamin D. In other studies, significant inverse relations were observed for triglycerides and hypertension with total vitamin D in samples of 45-y-old white men (9) and triglycerides in adults ≥20 y of age (29). Additional research on individual metabolic syndrome components, including lipid concentrations and vitamin D intakes, is warranted.

We observed an inverse relation between the total vitamin D intake and prevalence of high glucose concentrations in young adults that was consistent with results of other studies. In young adults aged 20–29 y and US adults enrolled in the NHANES III, higher serum 25-hydroxyvitamin D concentrations were beneficially related to the insulin-sensitivity index (11, 31) and glucose concentrations (29, 31), respectively. Subjects from the Ely prospective study showed a significant inverse relation between baseline serum 25-hydroxyvitamin D and incident high fasting glucose concentrations after 10 y of follow-up (8). The prospective Nurse's Health Study of 83,779 nurses aged 30–55 y showed decreased risk of type 2 diabetes with greater calcium and vitamin D intakes (14). The Framingham Study in 3418 older white participants showed decreased risk of incident type 2 diabetes with increased predicted plasma 25-hydroxyvitamin D over an average of 7 y (32).

The cross-sectional Women's Health Study and NHANES (2003–2004) showed inverse relations between vitamin D intakes and risk of having metabolic syndrome (7, 29, 30), which were consistent with findings from our prospective study. Our results also showed that, after adjustment for calcium, the significant association between vitamin D and metabolic syndrome was maintained, which suggested that a lower total vitamin D intake was an independent predictor of risk of developing metabolic syndrome over time. When adjusted for calcium intakes in NHANES (2003–2004) data, the association of vitamin D intakes with odds of having metabolic syndrome was maintained but attenuated in the Women's Health Study (7, 30). Results from NHANES III showed lower concentrations of serum hydroxyvitamin D in subjects with metabolic syndrome but higher serum 25-hydroxyvitamin D in subjects without metabolic syndrome (30). In the Framingham Offspring Study, increased serum 25-hydroxyvitamin D concentrations were associated with decreased risk of death from CVD in older white men and women (33). The development of metabolic syndrome is closely linked with CVD morbidity; thus, our findings support a potential strategy in the prevention of CVD.

Vitamin D intake may modulate risk of developing metabolic syndrome and its components through several potential mechanisms. Vitamin D is involved in the promotion of the influx of calcium in the regulation of insulin secretion and, therefore, the maintenance of glucose tolerance (34, 35). In addition, blood pressure may be regulated via sufficient 1,25-dihydroxyvitamin D production to suppress the renin synthesis in renal tubular cells (36). In addition, weight may be regulated with intakes of vitamin D–enriched dairy products. High-calcium diets are thought to inhibit fatty acid synthase and activate lipolysis (37), whereas some researchers proposed that a fall in circulating serum 25-hydroxyvitamin D increases the body weight set point (38). However, vitamin D mechanisms to prevent the development of the metabolic syndrome components of low HDL and high triglyceride concentrations remain unclear.

One limitation of the study was the assessment of self-reported dietary intake; however, the CARDIA diet history was validated against multiple 24-h recalls in a study of 128 participants aged 18–35 y (21). Although the correlation for dietary vitamin D was not reported in the validation study, the main source of both calcium and vitamin D intake was milk consumption, whereas fish and seafood intake was another top contributor of dietary vitamin D. The correlation of calcium between the 2 diet methods ranged from 0.66 to 0.80 for black and white men and women (21). In addition, sun exposure was not reported by CARDIA participants. However, geographic location has been used as a proxy measure for sunlight exposure; therefore field-center location were included as a confounding factor in statistical models (39). Furthermore, outdoor, indoor, and vigorous physical activity has been shown to correlate with higher serum and plasma 25-hydroxyvitamin D concentrations (40, 41). Plasma 25-hydroxyvitamin D was lower in US health professional white males who had lower physical activity scores (39). Our study used physical activity as a proxy for sun exposure.

Although we studied dietary vitamin D and not serum 25-hydroxyvitamin D, we showed a significant correlation between dietary plus supplemental vitamin D and serum 25-hydroxyvitamin D measured at year 7 in a subsample of 380 CARDIA participants (22). This study examined calcium metabolism and sunlight exposure in whites and African Americans; metabolic syndrome was not an outcome in this study. Our study was prospective in design with >20 y of follow-up, which allowed for the measurement of incident chronic conditions.

In conclusion, our findings show that total vitamin D consumption in young black and white men and women, including the intake of supplements, may lower risk of developing metabolic syndrome as these individuals transition to middle age. This research supports the recommendations of the Dietary Reference Intakes and 2010 Dietary Guidelines for Americans to consume vitamin D–fortified milk products, certain types of fish, egg yolks, and other foods that are fortified with vitamin D, including breakfast cereals, beverages, orange juice, and margarine. Our study findings contribute to the body of literature that showed a beneficial relation of serum or dietary vitamin D with chronic disease and suggest that vitamin D intake may be a potential strategy to prevent the development of adverse CVD risk factors.

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The authors' responsibilities were as follows—GJF and LMS: designed the research and wrote the first draft of the manuscript; LMS: supervised statistical analyses; XZ: performed statistical analyses; LMS: had primary responsibility for the final content of the manuscript and is the guarantor of the manuscript, having had full access to data in the study, and takes responsibility for the integrity of data and accuracy of the data analysis; and all authors: interpreted study results and reviewed and revised the manuscript. None of the authors had a conflict of interest.

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