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Vitamin D Deficiency, Adiposity, and Cardiometabolic Risk in Urban Schoolchildren

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Abstract

Objective—To determine the relationship between serum vitamin D levels and cardiometabolic risk factors independent of adiposity in urban schoolchildren.

Study design—We assessed the relationships among serum 25-hydroxyvitamin D [25(OH)D], adiposity measured by body mass index (BMI) z-score (BMIz), and 6 cardiometabolic risk factors (total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, triglycerides, interleukin-6, and C-reactive protein [CRP]) in a cross-sectional sample of 263 racially and ethnically diverse schoolchildren from the Boston area during late winter. Multivariate regression analyses adjusting for sociodemographic characteristics and BMIz examined associations of 25(OH)D and cardiometabolic risk factors.

Results—Overall, 74.6% of the children were vitamin D deficient [25(OH)D <50 nmol/L; mean, 41.8 ± 13.7 nmol/L]; 45% were overweight or obese (20%and 25%, respectively; BMIz = 0.75 ± 1.1). The 25(OH)D level was not associated with BMIz, but was positively associated with the cardiometabolic risk factor CRP (β = 0.03; $P < .05$). BMIz was associated with elevated triglycerides (β = 0.13), CRP (β = 0.58), and interleukin-6 (β= 0.14) and low high-density lipoprotein cholesterol (β = −0.09; all $P < .01$).

Conclusions—Vitamin D deficiency is highly prevalent during the late winter months in urban schoolchildren living in the northeastern United States. This widespread deficiency may contribute to the lack of associations between 25(OH)D and both BMIz and cardiometabolic risk factors. The association between 25(OH)D and CRP warrants further study.

Obesity in both children and adults has been tightly linked to the development of cardiovascular disease.[1,2] Risk factors in the development of cardiovascular disease and diabetes commonly observed in overweight children and adolescents include low high-density lipoprotein cholesterol (HDL-C), elevated low-density lipoprotein cholesterol (LDL-C) and triglycerides, high blood pressure, and impaired glucose tolerance.[3] Other cardiometabolic risk factors related to inflammatory pathways include C-reactive protein (CRP) and the proinflammatory cytokine interleukin (IL)-6, both of which are elevated in prepubertal obese children.[4,5]

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Obese individuals are more likely than normal-weight individuals to be vitamin D deficient.[6,7] Recent studies have indicated that vitamin D deficiency also may be an important independent risk factor for the development of cardiovascular disease. In more than 18 000 adult men participating in the Health Professionals Follow-up Study, those with a serum 25-hydroxyvitamin D [25(OH)D] level <37.4 nmol/L (15 ng/mL) were 2.4 times more likely to have a myocardial infarction than men with a 25(OH)D level >75 nmol/L (30 ng/mL).[8] In the same study, lower 25(OH)D level was associated with higher triglycerides and lower HDL-C.[8] Given that cardiovascular risk factors tend to track from childhood into adulthood,[9] these findings have potential clinical significance for pediatric care, given that an estimated 6 million US children are vitamin D deficient [25(OH)D <50 nmol/L (20 ng/mL)].[10,11] Recent studies in adolescents have identified inverse associations of 25(OH)D with serum glucose and triglycerides and direct associations with HDL-C, independent of weight status.[7] Given that HDL-C, triglycerides, and fasting glucose are established risk factors for diabetes and cardiovascular disease,[12] these findings suggest potential long-term consequences of 25(OH)D deficiency in childhood.

Vitamin D deficiency rates are known to be higher in persons who reside in northern latitudes, have darker skin pigmentation, and consume few foods rich in vitamin D.[6,11,13] These risk factors for vitamin D deficiency, along with a high prevalence of overweight/obesity, are likely exacerbated for urban schoolchildren living in the northeastern United States. Few previous studies have examined the relationships among vitamin D deficiency, adiposity, and cardiometabolic risk in these populations. Thus, the present study had 3 objectives: (1) to determine the prevalence of serum vitamin D deficiency in a racially diverse, urban population of schoolchildren living in the northeastern United States during the late winter months; (2) to determine the association between adiposity and 25(OH)D in this same population; and (3) to examine the relationship between 25(OH)D and cardiometabolic risk factors independent of adiposity in these children.

Methods

Students in grades 4–8 (age range, 9–14 years) were recruited from Somerville, Massachusetts, an urban school district north of Boston, during the late winter (January-March) of 2009. We chose to study this population because of its documented high prevalence of overweight and obesity. In 2009, 49% of the grade 4–8 students (n = 1636) in Somerville were overweight or obese (defined as a body mass index [BMI] >85th percentile).[14] The 2009 demographics of the student population of the school district were as follows: 52.1% male, 60% nonwhite, 66% in free/reduced-cost school lunch program, and 49.5% whose first language is not English. In 2008, the median household income in Somerville was $58 466.[15] Recruitment was done in the 7 elementary schools in the school district through school assemblies, school newsletters, and fliers sent home with the children. Recruitment materials outlined the purpose of the study and indicated that the children's blood lipid screening results would be provided to parents. No consented child was excluded from participation unless he or she consumed a food or beverage other than water after 10 PM the night before the blood draw. Each student was given a gift card to a large local retailer for participating. Consent forms and study information materials were available in English, Portuguese, Haitian-Creole, and Spanish, the major languages spoken in the community. All parents/guardians of participating children provided written informed consent, and all children gave written assent. The study protocol was approved by Tufts University's Institutional Review Board.

Participants arrived between 7 and 8 AM, before the start of the school day, after a 12-hour overnight fast. One blood sample was collected from each participant. Before each blood draw, the participant was asked about the occurrence of any recent illnesses and any severe

stress or intense exercise in the previous 48 hours, along with the use of any medications. Study procedures included height and weight measurement, phlebotomy, and a brief questionnaire to assess pubertal status and for any recent illness.

Measures

Sociodemographics—Birth date and race/ethnicity were reported by parents during the informed consent procedure. Parents were asked to choose one of the following: white/Caucasian, black/African-American, Mexican/Mexican-American, other Hispanic/Latino, Asian/Asian-American/Asian-Indian, Native American/American Indian, multiracial/multiethnic, or other. Age in months was calculated as test date minus birth date and converted to age in years. Lunch program status was extracted from the school administration record system and served as an indirect measure of family socioeconomic status, coded as a binary variable, free or reduced-cost (<185% of the poverty level) or paid lunch program.[15]

Anthropometrics—Each participant's standing height was measured in triplicate to the nearest 1/8 inch with a wall-mounted stadiometer (216 AccuHite; Seca, Snoqualmie, Washington). Weight also was measured in triplicate to the nearest 0.25 pound with a digital scale (Bella Model 840; Seca, Hanover, Maryland), with the child wearing light indoor clothing without shoes. BMI was calculated by dividing the child's average body weight by the square of their average height measurement. The BMI was subsequently converted to a *z*-score as recommended by the Centers for Disease Control and Prevention.[16] BMI *z*-score (BMIz) was used in all analyses.

Pubertal Status—Pubertal status was assessed by asking the female participants if they had reached menarche (yes/no) and male participants if their voice had changed (yes/no).[17] Affirmative responses were considered a marker for late puberty.

Serum 25(OH)D—Serum 25(OH)D level was measured by a competitive binding radioimmunoassay using controls provided by the manufacturer (DiaSorin, Stillwater, Minnesota). The intra-assay and interassay coefficients of variation (CV) were 8.6%–11.7% and 8.2%–11.0%, respectively. Vitamin D status was classified according to American Academy of Pediatrics (AAP)[18] and Institute of Medicine (IOM)[19] criteria as deficient, 25(OH)D <50 nmol/L (20 ng/mL), or not deficient, 25(OH)D ≥50 nmol/L.

Blood Lipids—Lipid levels were measured enzymatically on a Hitachi 917 analyzer (Roche Diagnostics; Indianapolis, Indiana) using reagents and calibrators from Roche Diagnostics (Indianapolis, Indiana) in a laboratory certified by the Centers for Disease Control and Prevention/National Heart, Lung, and Blood Institute's Lipid Standardization Program. As specified by the American Heart Association and the AAP,[20] abnormal blood lipid values are total cholesterol ≥170 mg/dL, triglycerides ≥110 mg/dL, HDL-C ≤40 mg/dL, and LDL-C ≥110 mg/dL.

Inflammatory Biomarkers

Serum IL-6 was measured by a quantitative enzyme-linked immunosorbent assay (Quantikine High-Sensitivity Human IL-6; R&D Systems, Minneapolis, Minnesota). The minimal detectable level of IL-6 was 0.039 pg/mL (normal range, 0.45–9.96 pg/mL), with an intra-assay CV of 6.9%–7.8% and an inter-assay CV of 6.5%–9.6%. Serum CRP was measured via a latex-enhanced turbidimetric immunoassay (Immulite 1000 High-Sensitivity CRP; Diagnostic Products, Los Angeles, California). The minimal detected CRP level was 0.10 mg/L, with intra-assay and interassay CVs of 4.2%–6.4% and 4.9%–10.0%, respectively.

Statistical Analyses

The χ^2 test was used to compare the distributions of categorical variables between the vitamin D–deficient and –nondeficient groups. Race/ethnicity categories were aggregated to white/Caucasian, black, Hispanic/Latino, Asian, and multiracial/other. Exploratory data analysis revealed that 25(OH)D, triglycerides, HDL-C, IL-6, and CRP were right-skewed; thus, the nonparametric Mann-Whitney *U* test was used to compare the distributions of continuous variables between the 2 groups, and these measures were natural logarithm-transformed before analyses. For the 53 subjects with an undetectable CRP level, a constant of 0.05 was added to all cases before the logarithmic transformation to retain them in the analysis.

Linear regressions were performed to determine the association between BMIz and 25(OH)D. First, in unadjusted analyses, 25(OH)D was regressed on each covariate separately. Then a multivariable regression was performed to evaluate the adjusted association between BMIz and 25(OH)D.

Because the cardiometabolic risk factors measured in this study are correlated and often co-vary, we used multivariate ANOVA to establish associations between 25(OH)D and each of the array of cardiometabolic risk factors (total cholesterol, LDL-C, HDL-C, triglycerides, IL-6, and CRP). The initial model included all explanatory variables [25(OH)D, BMIz, $BMIz^2$, age, sex, race/ethnicity, pubertal status, interaction terms between sex and puberty, and free/reduced-cost lunch eligibility] and all 2-way interaction terms that involved 25(OH)D, our main explanatory variable of interest. $BMIz^2$ was introduced to the model because the associations between outcomes and BMI *z*-score appear to be nonlinear and curved upward.[21] Because the test statistic (Wilks λ) of the multivariate analysis of variance was statistically significant, indicating that at least 1 outcome could be significantly explained by the set of explanatory variables, we performed further analyses to identify which cardiometabolic outcomes were associated with 25(OH)D. We applied a series of multiple regression models for each of the cardiometabolic risk factors. These models were refined based on 3 criteria: (1) Nonsignificant interaction terms ($P > .05$) that involved 25(OH)D were removed; (2) the interaction term between sex and puberty was removed if its *P* value was >.10; and (3) the quadratic term for BMIz was removed if its *P* value was >.10 or it demonstrated colinearity with the untransformed BMIz, rendering both BMI variables nonsignificant.

Because of the large number of subjects with a CRP level <0.10 mg/L, we performed additional sensitivity analysis by dividing the original CRP values into quartiles to test the robustness of our initial findings. Unless specified otherwise, statistical significance was based on an α value of 0.05. All statistical analyses were performed using SAS 9.2 (SAS Institute, Cary, North Carolina).

Results

Table I presents characteristics of the sample (n = 263; 11.7 ± 1.5 years) for both the total sample and the sample stratified by vitamin D status (deficient and nondeficient). The majority of children qualified for free or reduced-cost lunch (70.5%), and after white/Caucasian, the largest racial/ethnic group was Hispanic (30.5%). Nearly 45% of the children were overweight or obese. Some 75% of the children were vitamin D deficient [25(OH)D <50 nmol/L], and only 9 children had a 25(OH)D concentration ≥75 nmol/L (30 ng/mL), the optimal circulating concentration recommended by some researchers.[8,22] Children with vitamin D deficiency were more likely to be from racial/ethnic minorities, and when free and reduced-cost lunch were combined into one group, these children also were more likely to be vitamin D deficient (both $P < .05$).

With regard to the set of cardiometabolic risk factors, 45.1% of the children had 1 or more lipid levels falling outside the recommended range (Table II), distributed as follows: total cholesterol, 22.2%; triglycerides, 12.7%; HDL-C, 20.7%; LDL-C, 15.6%. CRP levels were highly skewed (median, 0.2 mg/L; range, 0.1–17.6 mg/L), with 78% of the children having elevated levels above detectable limits. Individual cardiometabolic risk factors did not differ between the deficient and nondeficient vitamin D status groups.

Relationship between BMIz and 25(OH)D

Table III shows the results of the regression analyses examining correlates of 25(OH)D. BMIz was not associated with 25(OH)D in either simple or multiple linear regression models. Mean 25(OH)D level was 9% lower in girls than in boys, and white/Caucasian children had the highest 25(OH)D levels—nearly 20% higher than any of the other racial/ethnic groups examined.

Relationship between 25(OH)D and Cardiometabolic Risk

The results of our multivariate regression analysis indicate a significant association between all our predictors as a group and cardiometabolic risk factors (Wilks $\lambda = 0.361$; $F = 4.48$; $P < .0001$). 25(OH)D was not associated with cardiometabolic risk factors at $\alpha = 0.05$ (Wilks $\lambda = 0.952$; $P = .069$), and BMIz was associated with overall cardiometabolic risk (Wilks $\lambda = 0.569$; $P < .0001$). The results of the multiple linear regression analysis investigating the associations among 25(OH)D, BMIz, and other covariates and the individual cardiometabolic risk factors are presented in Table IV. The interaction between sex and puberty was associated with HDL-C, triglycerides, and CRP ($P < .05$). $BMIz^2$ was associated with both triglycerides and CRP ($P < .05$). BMIz was negatively associated with HDL and positively associated with triglycerides, CRP, and IL-6. Based on the model, an increase in BMIz from 0.5 to 1.0 (corresponding to the approximate 70th and 85th percentiles, respectively) would be associated with a 5% decrease in median HDL, a 12% increase in median triglycerides, a 53% increase in median CRP, and a 7% increase in median IL-6.

After controlling for covariates, 25(OH)D and CRP were positively associated ($P < .05$), and 25(OH)D was marginally inversely associated with total cholesterol and LDL-C ($P < .10$). Age, sex, race/ethnicity, and pubertal status did not differ between subjects with undetectable CRP and those with detectable CRP; however, BMIz and 25(OH)D were higher in those with detectable CRP (data not shown; $P < .05$). The direction, magnitude, and P value of the coefficients were similar. 25(OH)D was not related to any of the other individual cardiometabolic risk factors.

Discussion

This study demonstrates that vitamin D deficiency, defined as 25(OH)D <50 nmol/L, is extremely common in this ethnically and racially diverse group of economically disadvantaged urban schoolchildren living in the northeastern United States studied during late winter. Our study population, although a convenience sample of fourth to eighth grade students from a single school district, was strongly representative of the demographics of the entire district, aside fromour recruitment of a slightly higher population of students eligible for free or reduced-cost lunch (70% vs. 66%). Vitamin D levels were lower in females and in non-white/Caucasian racial/ethnic groups, consistent with the findings of earlier studies,[23] and also in the children eligible for free/reduced price lunch. Other studies in the Boston area that measured vitamin D status throughout the year found that 12%of healthy infants and toddlers [24] and 42% of healthy adolescents were vitamin D deficient. [25] Our estimates, which were based on data collected from subjects in the community during late winter, are

more extreme than previous estimates from studies conducted in the Boston area and also than results from nationally representative surveys. Roughly 20% of US children have a 25(OH)D level below the recommended 50 nmol/L (compared with the 76% in our study), and more than 67% have a level <75 nmol/L (compared with our 97%), including 80% of Hispanic children and 92% of non-Hispanic black children (compared with 97.5% and 94.4%, respectively, in our population).[10]

We found no significant association between BMIz and 25(OH)D, in contrast with previous studies that found an inverse relationship between fat mass and 25(OH)D,[6,7] which has been attributed to sequestration of vitamin D within adipose tissue.[26] Furthermore, independent of adiposity, children in our study who did not have recommended lipid levels or elevated inflammatory biomarkers were not more likely to be vitamin D deficient. The low levels of 25(OH)D (median, 39.8 nmol/L) might have kept us from observing any differences in cardiometabolic risk between the deficient (≤50 nmol/L) and nondeficient (>50 nmol/L) groups. Had our sample included more children with a higher range of vitamin D sufficiency including levels ≥75 nmol/L, we might have observed an inverse association with cardiometabolic risk in the vitamin D-sufficient group.

Paradoxically, we observed a positive association between 25(OH)D and CRP, but no relationship between 25(OH)D and serum IL-6, which stimulates the release of CRP from the liver. The finding of a positive relationship between CRP and 25(OH)D was unexpected, and the mechanism of this association remains unclear. Although we asked about recent illness and bouts of strenuous exercise, it is possible that we were not able to completely control for other variables that might have affected these inflammatory biomarkers. As discussed earlier, the high rate of vitamin D deficiency also might have limited our ability to discern associations. Few previous studies have examined inflammatory biomarkers in relationship to 25(OH)D in pediatric populations. Low 25(OH)D was not associated with elevated CRP in children and adolescents examined in National Health and Nutrition Examination Survey 2001–2004,[23] and vitamin D deficiency was not associated with either blood lipids or CRP in a small sample of obese African-American female adolescents. [27] Some investigators have speculated that inflammation may mediate the relationship between low 25(OH) D and fasting glucose[7]; however, direct links to cardiometabolic risk in a pediatric population have not been elucidated and warrant future study.

The optimal healthy blood concentration of 25(OH)D in children is currently a matter of debate in the scientific community. The AAP and the IOM recommend an 25(OH)D level of at least 50 nmol/L.[18,19] However, studies in adults suggest an 25(OH)D level of at least 75 nmol/L to decrease the risk of heart disease.[8,22] Given our focus, we examined this threshold, and found that few children in our sample were vitamin D sufficient by this definition.

Several major factors have likely contributed to our findings. All children were evaluated during late winter, when 25(OH)D is likely at its lowest level, given that effective sunlight exposure, the primary determinant of vitamin D status, [22] emits insufficient UV-B rays to produce vitamin D cutaneously in northern latitudes (42°N) between November and March.[28] In addition, the high prevalence of overweight and obesity in the children studied (nearly 50%) also might have contributed to the low vitamin D levels measured. Finally, vitamin D intake through diet may be low in our study population; we do not have dietary data available for our sample, a limitation of this work. National data indicate that only 53% of boys and 47% girls aged 9–13 years consume an adequate intake of vitamin D,[29] and vitamin D intake is lower in non-Hispanic black children in the United States,[30] presumably for both physiological (lactose intolerance) and cultural reasons. Differences in dietary intake were not found between non-Hispanic white children and Mexican-American

children in the foregoing study[30]; however, the Hispanic/Latino participants were primarily from Central and South America and may have different racial composition and dietary patterns that contribute to a lower 25(OH)D status.

In this study, we cannot address the call to set vitamin D sufficiency at 25(OH)D concentrations ≥75 nmol/L to prevent health risks, because of the small number of children with concentrations at or above these levels. Based on a recent IOM report,[19] a serum 25(OH)D concentration of 50 nmol/L is adequate for skeletal health, which is currently the only established functional outcome indicator for vitamin D sufficiency. Thus, longitudinal studies are needed to establish whether a serum 25(OH)D concentration >75 nmol/L in children offers additional health benefits.

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Glossary

25(OH)D	25-Hydroxyvitamin D
AAP	American Academy of Pediatrics
BMI	Body mass index
BMIz	Body mass index z-score
CRP	C-reactive protein
CV	Coefficients of variation
HDL-C	High-density lipoprotein cholesterol
IL	Interleukin
IOM	Institute of Medicine
LDL-C	Low-density lipoprotein cholesterol

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Table I

Demographic data by vitamin Dstatus in urban schoolchildren

	Total (n = 263)		Deficient (n = 194)		Nondeficient (n = 69)		*P*
	n	%	n	%	n	%	value
Sex							
Female	136	51.7	101	74.3	35	25.7	.85
Male	127	48.3	93	73.2	34	26.8	
Ethnicity (n = 260) *							
White	95	36.5	59	62.1	36	37.9	.02
Black	18	6.9	15	83.3	3	16.7	
Hispanic/Latino	78	30.0	60	76.9	18	23.1	
Asian	22	8.5	18	81.8	4	18.2	
Multiracial/other	47	18.1	40	85.1	7	14.9	
Free/reduced-cost lunch (n = 258)							
Not eligible	76	29.6	49	64.5	27	35.5	.10
Reduced-cost lunch	46	17.9	35	76.1	11	23.9	
Free lunch	135	52.5	105	77.8	30	22.2	
Pubertal							
Yes	86	32.7	67	71.8	19	28.2	.29
No	177	67.3	127	77.9	50	22.1	
Weight category							
Underweight (<5th percentile)	7	2.7	5	71.4	2	28.6	.44
Normal weight (5th–84.9th percentile)	139	52.9	97	69.8	42	30.2	
Overweight (85th–95th percentile)	52	19.8	40	76.9	12	23.1	
Obese (>95th percentile)	65	24.7	52	80.0	13	20.0	

* $P < .05$ between deficient and nondeficient.

Table II

Serum 25(OH)D level and cardiometabolic risk factors by vitamin D status in urban schoolchildren

	Total (n = 263)		Deficient (n = 194)		Nondeficient (n = 69)		*P*
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**value**
25(OH)D, nmol/L *	41.9	13.7	35.2	7.5	60.2	10.2	<.001
Lipids, mg/dL							
HDL-C	50.6	12.6	50.3	12.6	51.4	12.8	.68
LDL-C	91.0	21.5	91.6	21.1	89.2	22.9	.48
Triglycerides	79.4	45.8	79.7	49.1	78.5	35.2	.44
Total cholesterol	154.0	23.7	154.2	23.6	153.5	24.3	.88
Inflammatory biomarkers							
IL-6, pg/mL	1.7	2.2	1.67	2.2	1.78	2.4	.93
CRP, mg/L	1.3	3.0	1.29	3.1	1.39	2.7	.43

* *P* <.05 between deficient and nondeficient.

Table III

Associations between BMIz and other covariates on 25(OH)D in urban schoolchildren

	Unadjusted		Adjusted	
Variables	**β**	**SE**	**β**	**SE**
Intercept	-	-	3.155	0.16**
BMIz	−0.012	0.02	−0.013	0.02
Age (year)	−0.036	0.01¶	−0.027	0.02‡
Male	0.083	0.04§	0.085	0.04§
Ethnicity				
White	0.210*	0.04**	†	-
Black	−0.110*	0.08	−0.240	0.08¶
Hispanic/Latino	−0.099*	0.04§	−0.186	0.05**
Asian	−0.047*	0.07	−0.177	0.08§
Multiracial/other	−0.133*	0.05§	−0.217	0.06**
Eligible for free/reduced-cost school lunch	−0.121	0.04¶	0.005	0.05
Entered puberty	−0.079	0.04‡	0.013	0.05

Outcome: natural log of 25(OH)D level. Unadjusted: simple linear regression with each variable as the only predictor. Adjusted: multiple linear regression that includes all of the predictors.

*Compared with all other races.

†Referent.

‡$P<.10$.

§$P<.05$.

¶$P<.01$.

**$P<.001$.

Table IV

Associations between 25(OH)D and cardiometabolic risk factors adjusting for BMIz in urban schoolchildren

	Cholesterol		LDL-C		HDL-C		Triglycerides		CRP		IL-6	
	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE
Intercept	5.29	0.09§	122.9	12.65§	4.00	0.13§	4.18	0.23§	−0.98	0.81	0.11	0.47
Vitamin D level	−0.003	0.002*	−0.49	0.25*	0.002	0.003	−0.004	0.005	0.03	0.02†	0.001	0.01
BMIz	0.006	0.008	1.59	1.23	−0.09	0.01§	0.13	0.03§	0.58	0.09§	0.14	0.05‡
Entered puberty	−0.05	0.03†	−6.04	3.54*	0.10	0.05†	−0.20	0.08†	−0.26	0.28	0.06	0.13
Sex × puberty					−0.15	0.06†	0.19	0.10*	0.66	0.35*		
$BMIz^2$							0.06	0.02§	0.18	0.05§		
Model *P* value	<.0001		.0005		<.0001		<.0001		<.0001		.0336	

*$P<.10$.

†$P<.05$.

‡$P<.01$.

§$P<.001$.