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Correlation between Vitamin E and Body Mass Index in US Adolescents Aged 10–19 Years: A Cross-Sectional Study

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Abstract: *The relationship between serum vitamin E (sVE) levels and body mass index (BMI) in the adolescent population has not been thoroughly investigated. This cross-sectional study aimed to explore this association using data from the National Health and Nutrition Examination Survey (NHANES) for adolescents aged 10–19 years. The analysis included 9,428 participants from NHANES cycles conducted between 1999 and 2006. sVE was treated as the primary exposure, while BMI served as the outcome measure. The relationship was evaluated using multivariable linear regression models, controlling for potential confounders such as age, sex, race/ethnicity, and socioeconomic status. Stratified analyses by sex and non-linear curve fitting methods were also performed. Results indicated a significant inverse association between sVE and BMI (β = -2.53, 95% CI: -2.92, -2.14), which was consistent across both males and females (p < 0.0001).Further analysis revealed distinct patterns: a linear trend was observed in females, whereas males exhibited an L-shaped relationship, highlighting potential gender-based variations. These findings suggest that higher sVE levels are associated with lower BMI in US adolescents, with notable differences in the nature of this relationship between sexes.*

Keywords: *Vitamin E, Body Mass Index, Adolescents, Nhanes, Obesity*

1. Introduction

Adolescent obesity rates have gradually increased over the past few decades [1], and rising global obesity prevalence is associated with an increased risk of chronic diseases. Body Mass Index (BMI) is a widely used measure to assess weight and height and is commonly used to classify weight status [2]. Vitamin E is a fat-soluble antioxidant found in large quantities in vegetable oils, nuts, seeds, and other dietary sources and plays a key role in reducing oxidative damage at the cellular level[3]. Given its antioxidant properties, vitamin E may influence body weight and metabolic parameters by regulating oxidative stress. However, the relationship between vitamin E and body mass index remains inconsistent across studies.

Some investigations report no significant impact of vitamin E supplementation on body weight or BMI. For instance, a meta-analysis encompassing 24 randomized controlled trials found no substantial effects of vitamin E supplementation on body weight, BMI, or waist circumference ($P > 0.05$) [4]. However, subgroup analyses revealed a potential association between vitamin E supplementation and increased BMI in individuals with normal BMI (18.5–24.9) ($P = 0.047$)[5]. In contrast, other studies have identified an inverse correlation between vitamin E levels and BMI. Research has shown that obese children exhibit significantly lower serum vitamin E concentrations compared to their normal-weight peers, suggesting a negative association with BMI[6]. Conversely, some evidence indicates elevated serum vitamin E levels in obese children, particularly those with abdominal obesity [7]. Additionally, vitamin E supplementation has been reported to significantly affect BMI in specific subgroups, such as postmenopausal women and individuals with diabetes [8]. These discrepancies suggest that the relationship between vitamin E and BMI may vary based on population characteristics, study design, and intervention protocols. Notably, limited research has explored this association in adolescent populations, underscoring the need for further investigation.

Therefore, we further propose to investigate the relationship between vitamin E and body mass index

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in the US adolescent population.

2. Methods

2.1 Study Population

This study analyzed data from the National Health and Nutrition Examination Survey (NHANES), a nationally representative cross-sectional survey capturing demographic, medical, and laboratory data. The study followed STROBE guidelines [9].

Data from four NHANES cycles (1999–2006) were included, selected for serum vitamin E availability. Adolescents aged 10–19years (n=9436) with complete serum vitamin E and BMI data were eligible. Exclusions comprised missing serum vitamin E (n=1044), levels <300 (n=6) or >2500 (n=2), and missing BMI (n=506), yielding a final sample of 9428 participants. The selection process is detailed in Figure 1.

NHANES protocols were approved by the NCHS Institutional Ethical Review Board. Written informed consent was obtained from participants aged ≥18, while parental/guardian consent was secured for those under 18[10].

2.2 Variables

Serum vitamin E, the independent variable, was measured via high-performance liquid chromatography with photodiode array detection. BMI, the dependent variable, was calculated as weight (kg)/height (m^2), measured by trained technicians using standardized protocols.

Covariates, selected based on literature, included age, sex, race/ethnicity (Mexican American, Other Hispanic, Non-Hispanic White, Non-Hispanic Black, Other/Multi-Racial), and education level. Additional covariates included laboratory measures (e.g., total cholesterol, glycohemoglobin, triglycerides) and anthropometric data. Blood pressure was averaged from three measurements. Further details on protocols are available at: http://www.cdc.gov/nchs/nhanes/.

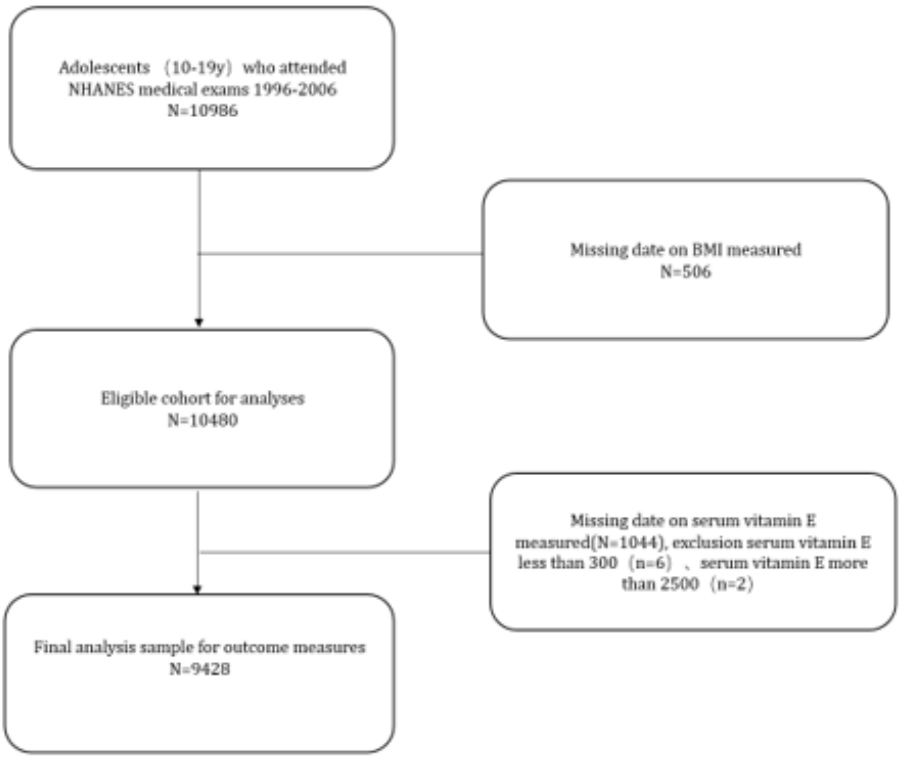

Figure 1 Flow chart of study population.

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2.3 Statistical analysis

Analyses were performed using R (version 6.0; R Foundation) and EmpowerStats (http://www.empowerstats.com), with significance set at $p < 0.05$. NHANES sample weights were applied to all calculations. Continuous variables were expressed as weighted means ±SD, and categorical variables as weighted frequencies (percentages). Serum vitamin E (sVE) levels were categorized into tertiles.

Weighted multiple linear regression models evaluated the sVE-BMI relationship, adjusting for confounders. Three models were used: Model 1 (unadjusted), Model 2 (adjusted for age, sex, race/ethnicity, education, SBP, DBP), and Model 3 (further adjusted for total cholesterol, triglycerides, glycohemoglobin). Model design was based on prior studies[11].

Smoothed curve fitting assessed linear/nonlinear relationships. Interaction and stratified analyses examined sex-specific effects of sVE on BMI, incorporating interaction terms and sex stratification in regression models.

Table 1 Baseline characteristics and BMI according to the tertiles of Vitamin E (n = 9428).

	Vitamin E (ug/dL)			
Parameters	Tertile1	Tertile2	Tertile3	P-value
	322.00-690.90	691.00-828.90	829.00-2460.50	
	N=3142	N=3137	N=3149	
Demographics				
age (years)				<0.001
10-14	1866 (59.39%)	1782 (56.81%)	1662 (52.78%)	
15-19	1276 (40.61%)	1355 (43.19%)	1487 (47.22%)	
Sex				<0.001
Male	1684 (53.60%)	1591 (50.72%)	1475 (46.84%)	
Female	1458 (46.40%)	1546 (49.28%)	1674 (53.16%)	
Ethnicity				
Black	1101 (35.04%)	1068 (34.05%)	851 (27.02%)	<0.001
Mexican	1089 (34.66%)	1074 (34.24%)	1096 (34.80%)	0.886
Other-hispanic	117 (3.72%)	110 (3.51%)	128 (4.06%)	0.503
Other	114 (3.63%)	135 (4.30%)	130 (4.13%)	0.368
Education				<0.001
Low	2801 (89.20%)	2723 (86.83%)	2612 (83.00%)	
Middle	174 (5.54%)	214 (6.82%)	281 (8.93%)	
High	165 (5.25%)	199 (6.35%)	254 (8.07%)	
mean systolic(mmHg)	108.68	108.23	108.30	0.5330
	(107.96 ,109.39)	(107.55 ,108.92)	(107.60 ,108.99)	
mean diastolic(mmHg)	59.87	60.01	60.90	0.0534
	(58.77 ,60.96)	(59.27 ,60.75)	(60.14 ,61.65)	
Total cholesterol	139.78	159.94	185.13	<0.0001
(mg/dL)	(138.47 ,141.10)	(158.65 ,161.24)	(183.21 ,187.04)	
Glycohemoglobin (%)				<0.001
Low	688 (21.90%)	586 (18.68%)	594 (18.86%)	

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Middle	1203 (38.29%)	1153 (36.75%)	1195 (37.95%)	
High	915 (29.12%)	984 (31.37%)	882 (28.01%)	
No record	336 (10.69%)	414 (13.20%)	478 (15.18%)	
Triglycerides (mg/dL)				<0.001
Low	1327 (42.23%)	898 (28.63%)	454 (14.42%)	
Middle	954 (30.36%)	997 (31.78%)	790 (25.09%)	
High	522 (16.61%)	825 (26.30%)	1415 (44.93%)	
No record	339 (10.79%)	417 (13.29%)	490 (15.56%)	
Body Mass Index	23.11	22.43	22.74	0.0004
(kg/m**2)	(22.84 ,23.38)	(22.13 ,22.72)	(22.43 ,23.04)	

The study included 9428 participants. Continuous variables are presented as weighted means (95% CI), with p-values derived from weighted one-way ANOVA. Categorical variables are expressed as weighted percentages, with p-values calculated using weighted chi-square tests. Missing data for covariates were as follows: total cholesterol (0.4%, n =36), glycohemoglobin (13.0%, n=1228), triglycerides (13.2%, n=1246), mean systolic blood pressure (3.0%, n=287), and mean diastolic blood pressure (3.0%,n=287).

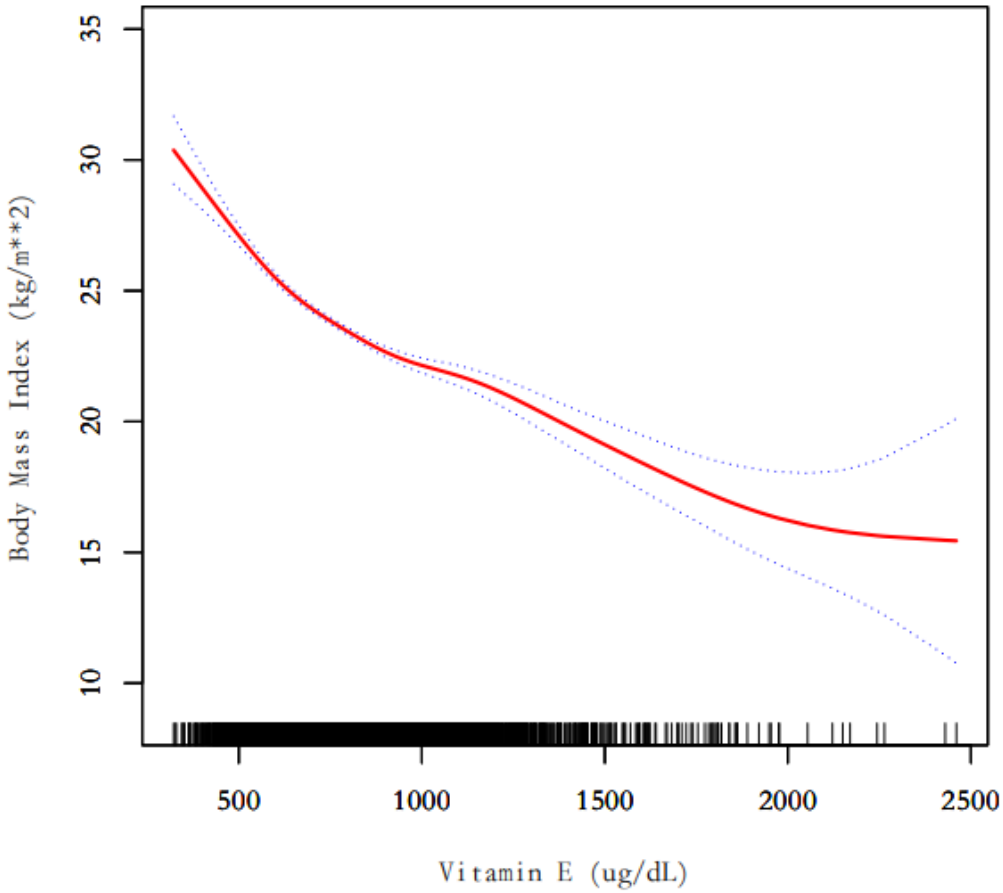

Figure 2 The graph depicts the association between serum vitamin E concentrations and BMI. The 95% CI is shown as a shaded region bounded by two blue dashed lines. Individual data points, representing serum vitamin E levels, are linked to form a continuous trend. The analysis was controlled for age, sex, ethnicity, education, triglycerides, cholesterol, glycohemoglobin, and blood pressure (SBP, DBP).

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Table 2 Association of serum vitamin E with body mass index in different models among all participants.

	Events(%)	Model 1 β (95% CI) P value	Model 2β (95% CI) P value	Model 3 β (95% CI) P value
Per 1 increment	786.24 ± 197.93	0.00 (-0.00, 0.00) 0.7174	0.00 (-0.00, 0.00) 0.6719	-0.01 (-0.01, -0.01) <0.0001
tertile				
T1(322.00-690.90)	3142 (33.33%)	0	0	0.
T2(691.00-828.90)	3137 (33.27%)	-0.68 (-1.01, -0.36) 0.0001	-0.56 (-0.88, -0.24) 0.0014	-1.42 (-1.75, -1.10) <0.0001
T3(691.00-828.90)	3149 (33.40%)	-0.37 (-0.72, -0.02) 0.0406	-0.22 (-0.56, 0.12) 0.2046	-2.53 (-2.92, -2.14) <0.0001
P for trend		0.0600	0.2490	<0.0001

Model 1: Unadjusted.

Model 2: Adjusted for age, sex, race/ethnicity, education, SBP, and DBP.

Model 3: Further adjusted for triglycerides, total cholesterol, and glycohemoglobin.

Table 3 β values for BMI by sVE across models stratified by sex.

	Model 1β (95% CI) P value	Model 2β (95% CI) P value	Model 3β (95% CI) P value
Male			
Per 1 increment	0.00 (0.00, 0.00) 0.0307	0.00 (0.00, 0.00) 0.0002	-0.01 (-0.01, -0.01) <0.0001
T1(322.00-690.90)	0.	0	0
T2(691.00-828.90)	-0.47 (-0.88, -0.05) 0.0308	-0.02 (-0.37, 0.33) 0.9105	-1.00 (-1.36, -0.64) <0.0001
T3(691.00-828.90)	0.05 (-0.44, 0.53) 0.8532	0.52 (0.16, 0.89) 0.0050	-2.03 (-2.47, -1.60) <0.0001
P for trend	0.8456	0.0063	<0.0001
Female			
Per 1 increment	-0.00 (-0.00, -0.00) 0.0499	-0.00 (-0.00, -0.00) 0.0026	-0.01 (-0.01, -0.01) <0.0001
T1(322.00-690.90)	0	0	0
T2(691.00-828.90)	-0.98 (-1.57, -0.39) 0.0018	-0.93 (-1.33, -0.54) <0.0001	-1.66 (-2.06, -1.26) <0.0001
T3(691.00-828.90)	-0.89 (-1.42, -0.36) 0.0018	-0.99 (-1.39, -0.60) <0.0001	-2.88 (-3.35, -2.40) <0.0001
P for trend	0.0033	<0.0001	<0.0001
P value for interaction	0.0401	<0.0001	0.0082

Model 1: Unadjusted.

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Model 2: Adjusted for age, sex, race/ethnicity, education, SBP, and DBP.

Model 3: Further adjusted for triglycerides, total cholesterol, and glycohemoglobin.

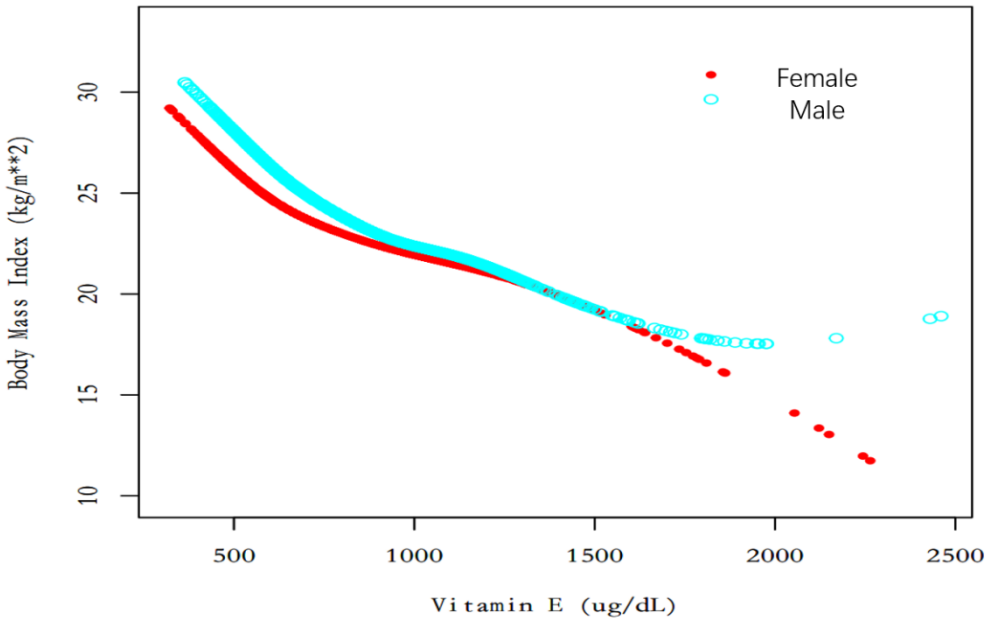

Figure 3 Smooth curve fitting plots revealed nonlinear associations between serum vitamin E and BMI, stratified by sex.

3. Results.

3.1 Participant Characteristics

This study included 9,428 participants (4,750 males and 4,678 females). Table 1 outlines their demographic and clinical characteristics stratified by baseline vitamin E tertiles. Except for SBP, Other Hispanic, and Mexican ethnic groups, all variables differed significantly across tertiles ($p < 0.01$). Participants in the highest tertile (T3) were more likely to be female, younger, non-Black, and have lower education levels, higher total cholesterol, and higher triglycerides compared to the lowest tertile (T1) ($p < 0.01$).

3.2 Association Between sVE and BMI

Multivariate linear regression (Table 2) revealed a negative association between serum vitamin E (sVE) and BMI in the fully adjusted model ($\beta = -0.01$, 95% CI: -0.01, -0.01). Figure 2 further illustrates this inverse correlation. When sVE was categorized, T2 and T3 showed lower BMI compared to T1 across all models (T2: Model 1 $\beta = -0.68$, 95% CI: -1.01, -0.36; Model 2$\beta = -0.56$, 95% CI: -0.88, -0.24; Model 3 $\beta = -1.42$, 95% CI: -1.75, -1.10; T3: Model 1 $\beta = -0.37$, 95% CI: -0.72, -0.02; Model 2 $\beta = -0.22$, 95% CI: -0.56, 0.12; Model 3 $\beta = -2.53$, 95% CI: -2.92, -2.14). The trend test for stratified variables was significant only after full adjustment ($p < 0.0001$).

3.3 Subgroup Analyses

Subgroup analyses (Table 3) highlighted sex-specific associations. In males, Model 1 ($\beta = 0.05$, 95% CI: -0.44, 0.53) and Model 2 ($\beta = 0.52$, 95% CI: 0.16, 0.89) showed positive associations, while Model 3 demonstrated a strong negative association ($\beta = -2.03$, 95% CI: -2.47, -1.60). In females, significant negative associations were observed across all models (Model 1: $\beta = -0.89$, 95% CI: -1.42, -0.36, $p = 0.0018$; Model 2: $\beta = -0.99$, 95% CI: -1.39, -0.60, $p < 0.0001$; Model 3: $\beta = -2.88$, 95% CI: -3.35, -2.40, $p < 0.0001$). Trend tests were significant in all models (Model 1: $p = 0.0033$; Model 2: $p < 0.0001$; Model 3: $p < 0.0001$).As shown in Figure 3, the results can be observed and analyzed more clearly.

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3.4 Interaction Analysis

Significant interactions between sex and vitamin E levels were observed in all models (Model 1: $p = 0.0401$; Model 2: $p < 0.0001$; Model 3: $p = 0.0082$), indicating sex-specific differences in the association between vitamin E and BMI.

4. Discussion

The objective of this study was to examine the relationship between sVE and BMI in adolescents, using data from the national health and nutrition survey (NHANES). Our results show a significant negative correlation between sVE and BMI. Adolescents with a higher BMI have low levels of sVE. This negative correlation remains constant after adjustment for potential conquerors. A stratified analysis showed a negative correlation between sVE and female BMI. This study and a recent study of 4,014 adolescents found a negative correlation between evs levels and changes in bmi among girls and not boys[12]. Specifically, increasing sVE standard deviation is associated with a 92% reduction in the risk of obesity among women[13]. But our study, based on the fit of smooth curves, found non-linear (l-shaped) relationships observed in male adolescents and significant linear correlations in female adolescents. And there is a potential interaction between sVE and gender. This difference can be attributed to gender-specific hormonal effects, since previous studies have linked hormonal factors to the prevalence of obesity in female adolescents [14]. However, randomised controlled trials (RCTS) are needed to confirm these results.

In addition, VE consumption is inversely associated with the risk of many cancers, cardiovascular disease, Parkinson's disease, depression, metabolic syndrome and fractures[15]. Vitamine E has anti-inflammatory effects on inflammatory diseases and associated comorbidities, such as obesity[16].

Adolescent obesity is associated with comorbidities such as metabolic disorders, cardiovascular diseases, and type 2 diabetes[17]. As a potent lipophilic antioxidant, vitamin E may regulate metabolism and influence BMI through multiple mechanisms. It mitigates oxidative stress, which is elevated in obesity and contributes to metabolic dysfunction[18]. Vitamin E metabolites also exhibit hormone-like activities, modulating gene expression and cellular signaling pathways involved in lipid metabolism and energy homeostasis. For example, long-chain vitamin E metabolites inhibit 5-lipoxygenase at nanomolar concentrations, reducing inflammation commonly associated with obesity[19]. Dietary factors further complicate the relationship between vitamin E and BMI. High-fat diets, prevalent in obese individuals, increase vitamin E requirements, yet imbalanced diets often lead to inadequate intake and deficiency. Altered intestinal absorption in obese populations may also reduce vitamin E bioavailability[20]. These findings suggest that vitamin E supplementation could improve metabolic health and reduce body weight, though further research is required to validate this hypothesis.

To the best of our knowledge, this study represents the first investigation into the association between vitamin E and adolescent obesity. Leveraging a large sample size, we conducted extensive subgroup analyses and utilized smooth curve fitting to examine the relationship between serum vitamin E (sVE) and BMI, revealing a consistent negative correlation. However, the findings are limited to adolescents aged 10–19 years and may not be generalizable to other age groups. Several limitations should be acknowledged. First, the cross-sectional design precludes the establishment of causal relationships. Overweight or obese individuals may consume diets high in fat and low in vitamins, potentially introducing confounding effects and reverse causality. Second, although we employed advanced methods to address missing data, some variables had incomplete information, which may introduce bias. Third, despite adjusting for numerous confounders, residual confounding from unmeasured factors cannot be ruled out. Fourth, while BMI is a widely used metric, other obesity-related measures, such as waist circumference and waist-to-hip ratio, were not included in the analysis. Fifth, dietary vitamin E intake, which may influence obesity, was not assessed, as our analysis focused solely on sVE levels. Finally, the study relied on NHANES data from 1999–2006, and more recent data on vitamin E and BMI remain unexplored.

sVE is an easily measurable and cost-effective biomarker. This study highlights a significant negative correlation between sVE and adolescent obesity, suggesting its potential as a tool for early obesity identification and weight management. Further research is needed to elucidate the causal mechanisms underlying this relationship and to explore the therapeutic potential of vitamin E supplementation in

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obesity prevention and treatment.

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Author Contributions

JJZ conducted the literature review, designed the study, collected and analyzed data, interpreted results, and drafted the manuscript. JZJ and JYH conceptualized the study, coordinated data collection, and performed data analysis. CX, JHH and YZC provided critical revisions. All authors reviewed and approved the final manuscript.

Data Availability

The data supporting this study are publicly accessible via NHANES: https: //www. cdc. gov/nchs/nhanes/index.htm.

Ethical Approval

This study adhered to ethical standards in accordance with institutional, national, and Declaration of Helsinki guidelines. Informed consent was obtained from all participants for both participation and publication.

Competing Interests

The authors declare no competing interests.

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