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The association between vitamin E intake and remnant cholesterol, total cholesterol, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol in US adults: a cross-sectional study

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Abstract

Background Blood lipid profiles are associated with various nutritional elements and dietary factors. This study aimed to explore the association between total dietary vitamin E intake and remnant cholesterol (RC), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) using data from the National Health and Nutrition Examination Survey (NHANES).

Methods A cross-sectional analysis was conducted using NHANES 2007–2018 data. A total of 8,639 eligible participants (45.58% men and 54.42% women) with an average age of 46.12 ± 16.65 years were included in this study. Weighted multivariate linear regression and subgroup analyses were used to examine the association between vitamin E intake and RC, TC, HDL-C, and LDL-C. Smooth curve fitting was used to explore potential non-linear associations.

Results After adjusting for other covariates, multivariate linear regression analysis showed that higher vitamin E intake was negatively associated with plasma RC (β = -0.22, 95% CI: -0.27, -0.16), TC (β = -0.33, 95% CI: -0.51, -0.16), LDL-C (β = -0.25, 95% [confidence interval] CI: -0.40, -0.10) and positively associated with HDL-C (β = 0.13, 95% CI: 0.07, 0.20) in US adults. Subgroup analysis indicated that age may influence the association between vitamin E intake and RC. At the same time, gender may also affect the association between vitamin E intake and HDL-C.

Conclusion Higher vitamin E intake was negatively associated with plasma RC, TC, LDL-C and positively associated with HDL-C.

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Introduction

Atherosclerotic cardiovascular disease (ASCVD), the world's main cause of mortality and a significant economic burden, is largely caused by dyslipidemia [1–3]. Previous studies showed that dyslipidemia is connected with various other diseases, including osteoporosis, hyperuricemia, and chronic kidney disease [4–6]. Remnant cholesterol (RC) comprises triglyceride-rich lipoproteins, including chylomicron remnants, very low-density lipoprotein cholesterol (VLDL-C), and intermediate-density lipoprotein cholesterol (IDL-C) [7]. The calculation of RC involves deducting total cholesterol (TC) from high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C) [3, 8]. It serves as a lipoprotein marker with a crucial role in predicting the risk of ASCVD and ischemic stroke [3, 9]. A cohort study of 87,192 participants aged 20 to 69 years in Copenhagen demonstrated that individuals with RC levels of ≥1.0 mmol/L (22% of the population) exhibited multivariable-adjusted mortality hazard ratios (HR) of 2.2 (95% [confidence interval] CI: 1.3–3.5) for cardiovascular disease, compared with individuals with levels under 0.5 mmol/L [10]. These findings suggest that identifying the factors influencing RC and regulating its levels is crucial for promoting human health.

Vitamin E was discovered more than a century ago by Katharine Scott Bishop [11]. Vitamin E is a vital micronutrient from nutritional sources and supplements [12]. In both humans and animals, alpha-tocopherol represents the primary form of vitamin E [13]. It is proven to have a variety of biological properties, such as neutralizing free radicals by functioning as an antioxidant [14].

Research has explored the potential relationship between vitamin E supplementation and blood lipid levels [15, 16]. However, this relationship has not been fully elucidated, possibly due to limitations in experimental design and sample size. Controlling for confounding factors and increasing the study sample size is necessary to clarify the exact association. Therefore, a cross-sectional analysis was performed to investigate the association between total vitamin E intake and RC, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and TC utilizing data from the National Health and Nutrition Examination Survey (NHANES).

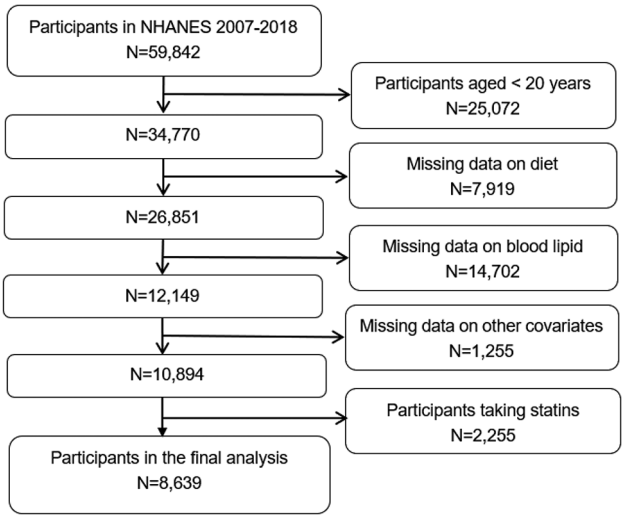

Fig. 1 Inclusion and exclusion criteria for study participants

Materials and methods

Data sources

This research extracted data spanning six NHANES cycles: 2007–2008, 2009–2010, 2011–2012, 2013–2014, 2015–2016 and 2017–2018. Participation was contingent upon written informed consent, with all study protocols subjected to the scrutiny and approval of the ethics committee.

From the 2007 to 2018 cohorts, 59,842 participants were initially considered. A total of 25,072 non-adult participants (under 20 years of age) were excluded. Additional exclusions were made for those lacking total nutrient intake data (7,919 individuals) and those missing information on blood lipids (14,702 individuals). Further, 1,255 participants with incomplete data on other relevant covariates including gender, age, body mass index (BMI), race, education level, the ratio of family income to poverty (PIR), uric acid, aspartate aminotransferase (AST), alanine aminotransferase (ALT), glycohemoglobin (HbA1c), smoking status, diabetes, stroke, and presence of cancer or malignancy were also excluded. Finally, participants who had taken one or more statins (including atorvastatin, fluvastatin, pitavastatin, lovastatin, simvastatin, rosuvastatin, and pravastatin) within the preceding 30 days were excluded (2,255 individuals). After applying these criteria, the study comprised 8,639 participants deemed eligible for analysis. Sampling weights (WTDR2D) were employed during the data analysis, and a weighted analysis was conducted. (Fig. 1)

Vitamin E intake

Total vitamin E intake (mg) was obtained from the NHANES nutritional data. The dietary interviews obtained detailed information on dietary intake from NHANES participants. It used statistic on participants' daily food and drink intake, including water, and estimated the nutrients, energy, and other food components consumed. The interviews were conducted in two distinct sessions. The first set of data was obtained in the examination center, and the other set was obtained three to ten days later. To minimize the impact of statistical bias and error, the data on dietary total vitamin E intake

included in this analysis were derived from the mean of the data from the two sessions.

Lipid-related indicators

The blood lipid measurements were made after a minimum of 8.5 h of fasting. Serum HDL-C levels (mg/dL) was measured with an immunoassay, while TC (mg/dL) were quantified through direct measurement using an enzymatic assay. The method to measure triglycerides was an endpoint reaction. TC, HDL-C, and TG (mg/dL) values that were directly measured were used to compute serum LDL-C (mg/dL) levels. The Friedewald equation was employed for the calculation of LDL-C [17]. LDL-C and HDL-C were subtracted from TC to determine the serum RC values [18].

Covariates

The following covariates were added to adjust the model based on previous studies. Categorical variables: gender, education level (under high school, high school or equivalent, college graduate or above), ethnicity/race (non-Hispanic White, non-Hispanic Black, other Hispanic, Mexican American, other race), smoking status (yes, no), presence of cancer/malignancy (yes, no), diabetes (yes, borderline, no), and stroke (yes, no). Continuous variables: age (years), BMI (kg/m^2), PIR, energy (kcal), protein (gm), AST (U/L), ALT (U/L), uric acid (mg/dL), and HbA1c (%). Please refer to the NHANES website for detailed data on the covariates.

Statistical analysis

Weighted percentages were used to represent the categoric variables, and either the mean or the quartile with standard deviations was used to describe the continuous variables. The association between total dietary vitamin E intake and RC, LDL-C, HDL-C, and TC was evaluated in the current study using weighted multivariate linear regression analysis. In the multivariate linear regression analysis, three models were created: model 1 (non-adjusted), model 2 (adjusted for age, race, and gender), and model 3 (adjusted for all covariates). Based on age, BMI, and race/ethnicity, subgroup analysis were carried out. Threshold effect analysis investigated the association and inflection point between total dietary vitamin E intake and RC, LDL-C, HDL-C, and TC. R version 4.3.2 and EmpowerStats version 2.0 were used for the analyses.

Results

Baseline characteristics

This study encompassed 8,639 qualified participants, 45.58% males, and 54.42% females, with an average age of 46.12±16.65 years. This cohort was established after excluding individuals due to incomplete data sets. The quartiles of the paticipants' total dietary vitamin E intake were used to stratify them into four different groups (Quartile 1: <4.75 mg, Quartile 2: 4.75–6.93 mg, Quartile 3: 6.93–9.94 mg, Quartile 4: >9.94 mg). Notable statistical disparities were observed across these quartiles concerning demographic and health-related variables, including gender, BMI, ethnicity/race, educational attainment, poverty income ratio (PIR), energy, protein, ALT, HbA1c, smoking status, stroke, TC, TG, LDL-C, HDL-C, and RC, all showing significant differences. Conversely, no significant variation was detected in age, AST, uric acid, incidence of diabetes, and presence of cancer among the groups, indicating a selective influence of vitamin E consumption on specific health metrics. (Table 1)

Association between total vitamin E intake and TC, HDL-C, LDL-C, and RC

Total dietary vitamin E intake was used as a continuous variable and, following quartile translation, as a categorical variable in a multivariate linear regression analysis involving RC, TC, HDL-C, and LDL-C. In model 1, which did not adjust for variables, it was observed that total vitamin E intake exhibited an inverse association with RC, TC, and LDL-C, and positively associated with HDL-C. Upon adjustment for gender, age, and ethnicity/race as covariates, total vitamin E intake remained inversely associated with RC, TC, LDL-C, and positively associated with HDL-C. The results of model 3, which was adjusted for all covariates, indicated that a higher total intake of vitamin E was associated with a lower RC (β = -0.22, 95% CI: -0.27, -0.16), TC (β = -0.33, 95% CI: -0.51, -0.16), LDL-C (β = -0.25, 95% CI: -0.40, -0.10) and a higher HDL-C (β=0.13, 95% CI: 0.07, 0.20). Compared to individuals in the lowest quartile of total vitamin E intake, those in the highest quartile had reduced mean levels of RC, TC, and LDL-C. Those in the highest quartile of total vitamin E intake had greater HDL-C levels than those in the first quartile, with a statistically difference. (Table 2) Further, applying smooth curve fitting techniques revealed a clear non-linear relationship between total vitamin E intake and RC, LDL-C, HDL-C, and TC levels. (Fig. 2) Furthermore, the threshold effect of total vitamin E intake was investigated. A notable inverse correlation was identified between total vitamin E intake and RC before and after the inflection point. However, the effect observed before the inflection point (11.18 mg) was more pronounced than that observed after the inflection point. The findings indicated that when total vitamin E intake was below the inflection point (11.19 mg), there was a notable decline in TC with the increase in vitamin E intake. Once the inflection point was exceeded, the effect ceased to be significant. Similar results were noted in the threshold effect analysis between vitamin E intake and HDL-C. Conversely, when the consumption of vitamin E was before the inflection point (2.52 mg), the observed

Table 1 Baseline characteristics of participants from 2007 to 2018

Characteristics	Dietary vitamin E intake (mg)				*P*-value
	Q1 (< 4.75) *N* = 2,155	Q2 (4.75–6.93) *N* = 2,163	Q3 (6.93–9.94) *N* = 2,159	Q4 (> 9.94) *N* = 2,162	
Age (years)	44.49 ± 17.17	44.26 ± 16.66	43.93 ± 15.61	44.48 ± 14.96	0.616
Gender					***< 0.001***
Male	705 (32.74%)	919 (42.48%)	1034 (47.91%)	1231 (56.94%)	
Female	1450 (67.26%)	1244 (57.52%)	1125 (52.09%)	931 (43.06%)	
BMI (kg/m^2)	29.17 ± 6.98	28.94 ± 6.99	28.78 ± 6.73	28.37 ± 7.02	***0.001***
WC (cm)	98.28 ± 16.43	98.08 ± 16.62	98.47 ± 16.32	97.45 ± 16.58	0.172
Race					***< 0.001***
Mexican American	190 (8.82%)	224 (10.35%)	199 (9.21%)	176 (8.13%)	
Other Hispanic	163 (7.56%)	128 (5.93%)	106 (4.90%)	128 (5.94%)	
Non-Hispanic White	1325 (61.49%)	1391 (64.31%)	1444 (66.89%)	1524 (70.46%)	
Non-Hispanic Black	292 (13.54%)	242 (11.20%)	224 (10.39%)	170 (7.87%)	
Other Race	185 (8.59%)	178 (8.21%)	186 (8.62%)	164 (7.60%)	
Education Level (%)					***< 0.001***
Under high school	492 (22.83%)	342 (15.79%)	274 (12.67%)	181 (8.38%)	
High school or equivalent	575 (26.68%)	498 (23.04%)	452 (20.94%)	409 (18.92%)	
College graduate or above	1088 (50.49%)	1323 (61.17%)	1433 (66.39%)	1572 (72.70%)	
PIR	2.41 ± 1.60	2.80 ± 1.64	3.04 ± 1.64	3.24 ± 1.64	***< 0.001***
Energy (kcal)	1420.91 ± 523.10	1906.30 ± 535.61	2242.71 ± 620.21	2695.08 ± 898.01	***< 0.001***
Protein (gm)	56.56 ± 23.55	74.68 ± 24.96	87.63 ± 28.07	105.30 ± 39.43	***< 0.001***
Uric acid (mg/dL)	5.38 ± 1.41	5.43 ± 1.39	5.42 ± 1.35	5.43 ± 1.31	0.592
ALT (U/L)	23.54 ± 17.40	24.47 ± 18.78	25.45 ± 19.72	25.97 ± 17.71	***< 0.001***
AST (U/L)	24.73 ± 18.51	24.68 ± 19.78	24.97 ± 16.88	25.71 ± 16.35	0.173
HbA1c (%)	5.52 ± 0.78	5.53 ± 0.77	5.51 ± 0.73	5.46 ± 0.77	***0.006***
Smoking Status					***< 0.001***
Yes	1010 (46.87%)	960 (44.36%)	921 (42.64%)	810 (37.45%)	
No	1145 (53.13%)	1203 (55.64%)	1238 (57.36%)	1352 (62.55%)	
Diabetes					0.096
Yes	115 (5.34%)	119 (5.51%)	114 (5.26%)	84 (3.91%)	
No	2005 (93.03%)	2016 (93.22%)	2009 (93.05%)	2037 (94.21%)	
Borderline	35 (1.63%)	28 (1.27%)	36 (1.69%)	41 (1.88%)	
Stroke					***< 0.001***
Yes	70 (3.26%)	48 (2.23%)	38 (1.75%)	19 (0.89%)	
No	2085 (96.74%)	2115 (97.77%)	2121 (98.25%)	2143 (99.11%)	
Cancer					0.790
Yes	157 (7.30%)	174 (8.05%)	160 (7.39%)	161 (7.43%)	
No	1998 (92.70%)	1999 (91.95%)	1999 (92.61%)	2001 (92.57%)	
TC (mg/dL)	198.27 ± 42.13	197.35 ± 39.75	195.73 ± 39.21	193.47 ± 39.65	***< 0.001***
HDL-C (mg/dL)	54.43 ± 16.44	54.63 ± 15.54	54.08 ± 15.35	55.67 ± 16.18	***0.004***
LDL-C (mg/dL)	119.75 ± 35.97	119.50 ± 34.87	118.49 ± 34.09	116.68 ± 34.27	***0.012***
TG (mg/dL)	120.49 ± 66.08	116.11 ± 64.71	115.77 ± 68.71	105.59 ± 61.28	***< 0.001***
RC (mg/dL)	24.09 ± 13.22	23.21 ± 12.94	23.16 ± 13.75	21.12 ± 12.28	***< 0.001***

Values were represented as mean ± SD or number (%). P-value < 0.05 was considered statistically significant

association between vitamin E and LDL-C was not statistically significant. However, after exceeding the inflection point, the effect became statistically substantial. (Table 3)

Subgroup analysis

Subgroup analysis were carried out based on age, BMI, and race/ethnicity. The results demonstrated a positive association between total vitamin E intake and HDL-C levels in women but not men. This suggested that gender may be a crucial factor influencing the connection between vitamin E intake and HDL-C levels. Additionally, those over 50 showed a negative connection between vitamin E intake and RC. (Table 4)

Table 2 Association between total dietary vitamin E intake and TC, HDL-C, LDL-C, and RC by multivariate linear regression analysis

	Model 1	Model 2	Model 3
	β (95%CI), *P*-value	**β (95%CI), *P*-value**	**β (95%CI), *P*-value**
RC (continuous)	-0.20 (-0.25, -0.15), ***< 0.01***	-0.27 (-0.32, -0.22), ***< 0.01***	-0.22 (-0.27, -0.16), ***< 0.01***
Quartile 1	Ref.	Ref.	Ref.
Quartile 2	-0.87 (-1.70, -0.05), ***0.04***	-1.33 (-2.14, -0.53), ***< 0.01***	-1.39 (-2.17, -0.61), ***< 0.01***
Quartile 3	-0.93 (-1.74, -0.12), ***0.02***	-1.56 (-2.35, -0.77), ***< 0.01***	-1.61 (-2.43, -0.80), ***< 0.01***
Quartile 4	-2.97 (-3.76, -2.18), ***< 0.01***	-4.07 (-4.85, -3.29), ***< 0.01***	-3.78 (-4.69, -2.88), ***< 0.01***
TC (continuous)	-0.37 (-0.52, -0.21), ***< 0.01***	-0.33 (-0.48, -0.19), ***< 0.01***	-0.33 (-0.51, -0.16), ***< 0.01***
Quartile 1	Ref.	Ref.	Ref.
Quartile 2	-0.92 (-3.45, 1.62), 0.48	-0.48 (-2.89, 1.93), 0.70	-0.89 (-3.34, 1.55), 0.48
Quartile 3	-2.54 (-5.03, -0.06), ***0.04***	-1.69 (-4.06, 0.69), 0.16	-2.40 (-4.96, 0.16), 0.07
Quartile 4	-4.80 (-7.23, -2.37), ***< 0.01***	-4.12 (-6.46, -1.77), ***< 0.01***	-4.60 (-7.42, -1.78), ***< 0.01***
HDL (continuous)	0.13 (0.07, 0.19), ***< 0.01***	0.30 (0.24, 0.35) ***< 0.01***	0.13 (0.07, 0.20), ***< 0.01***
Quartile 1	Ref.	Ref.	Ref.
Quartile 2	0.20 (-0.80, 1.21), 0.69	1.37 (0.44, 2.31), ***< 0.01***	0.61 (-0.28, 1.49), 0.18
Quartile 3	-0.36 (-1.34, 0.63), 0.48	1.40 (0.48, 2.32) ***< 0.01***	0.12 (-0.80, 1.04), 0.80
Quartile 4	1.24 (0.28, 2.20), ***0.01***	3.97 (3.06, 4.88), ***< 0.01***	1.77 (0.75, 2.79), ***< 0.01***
LDL (continuous)	-0.29 (-0.42, -0.16), ***< 0.01***	-0.36 (-0.49, -0.23), ***< 0.01***	-0.25 (-0.40, -0.10), ***< 0.01***
Quartile 1	Ref.	Ref.	Ref.
Quartile 2	-0.25 (-2.44, 1.95), 0.83	-0.52 (-2.64, 1.61), 0.63	-0.11 (-2.26, 2.04), 0.92
Quartile 3	-1.26 (-3.41, 0.90), 0.25	-1.53 (-3.63, 0.57), 0.15	-0.91(-3.16, 1.34), 0.43
Quartile 4	-3.07 (-5.18, -0.96), ***< 0.01***	-4.02 (-6.09, -1.95), ***< 0.01***	-2.58 (-5.06, -0.10), ***0.04***

Model 1: None covariates were adjusted; Model 2: Gender, race and age were adjusted; Model 3: Gender, race, age, education level, PIR, BMI, energy, protein, uric acid, ALT, AST, HbA1c, smoking status, stroke, diabetes and presence of cancer/malignancy were adjusted

Discussion

This study analyzed the association between total vitamin E intake and blood lipid indicators, including RC, LDL-C, HDL-C, and TC, based on NHANES data. Higher vitamin E intake was found to be positively correlated with HDL-C and adversely correlated with plasma RC, TC, and LDL-C in Americans aged 20 years and older. These results demonstrate the possible role played by vitamin E in modulating blood lipid levels.

Current research in clinical and epidemiological settings has emphasized the function of RC in the development and prognosis of various diseases. The highest quartile (quartile 4) of RC showed HRs of 1.11 (95% CI: 1.08–1.13) for Alzheimer's disease, 1.11 (95% CI: 1.09–1.13) for all-cause dementia, and 1.15 (95% CI: 1.09–1.21) for vascular dementia, according to a cohort study carried out in Asia [19]. A nationwide cohort study of 3,403,414 participants in China found that individuals with RC≥27.7 mg/dL exhibited higher HRs for all-cause mortality than those with RC<17.9 mg/dL [20]. Numerous studies utilizing NHANES data have also demonstrated a causal relationship between RC and other diseases, including osteoporosis, hyperuricemia, and kidney stones [5, 21, 22].

Maintaining a reasonable level of RC is of great significance. However, few large-scale studies have investigated the underlying factors influencing RC levels. The results of this study indicated that a higher intake of vitamin E was associated with lower plasma RC levels. A decrease in TC levels and a rise in HDL-C levels may be partly to blame this impact. These results suggest a potential approach for regulating RC levels.

HDL is the smallest circulating lipoprotein, comprising proteins and lipids, and is present in nearly all cells [23]. It is responsible for transporting cholesterol, facilitating intercellular communication, and inactivating harmful substances [24]. The risk of coronary heart disease (CHD) is inversely correlated with HDL-C levels, according to several studies [25–27]. The progression of hyperglycemia or atherosclerosis has been reduced or even reversed through the exogenous application or transgenic overexpression of apolipoprotein A-I [28, 29]. Drugs elevating serum HDL-C levels include niacin and fibrates [30].

Conversely, LDL is a significant risk factor for cardiovascular disease due to its causal relationship with ASCVD [31, 32]. It is estimated that approximately 7.3% of global deaths annually can be attributed to high LDL-C levels, representing a substantial health burden [33]. Statins are the primary therapeutic intervention for reducing LDL-C levels in clinical practice [34]. A meta-analysis revealed a 21% relative risk decrease in cardiovascular events associated with statin-assisted LDL-C lowering of 1 mmol/L [35].

According to a meta-analysis, LDL-C and TC levels in the serum were found to decrease in response to vitamin E administration, regardless of whether it was given in isolation or in conjunction with omega-3 or magnesium. Nevertheless, no notable impact was discerned

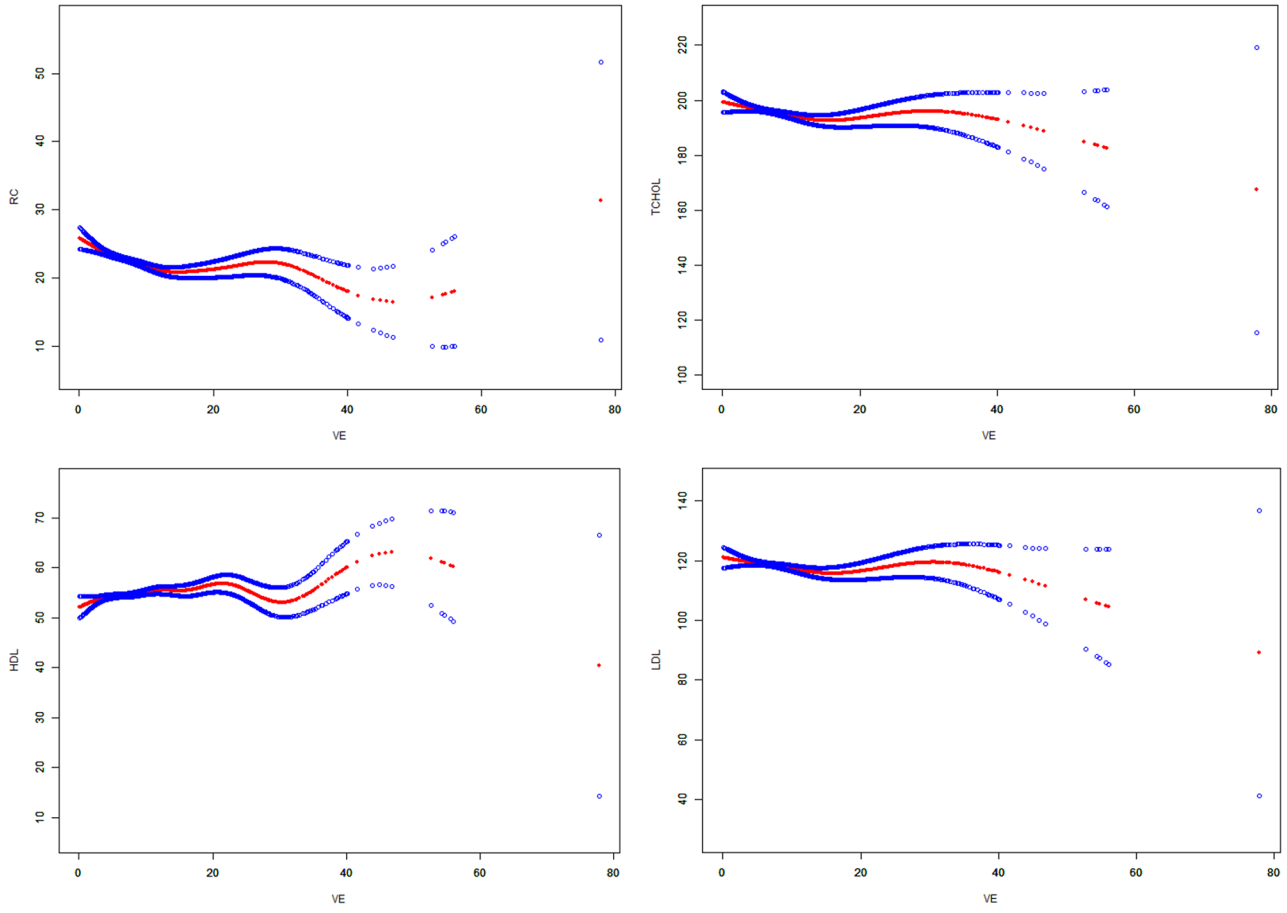

Fig. 2 Non-linear relationship between total dietary vitamin E intake and RC, TC, HDL-C and LDL-C. The smooth curve fit between the variables is shown by the red line. The 95% confidence interval from the fit is represented by the blue bands. All covariates were adjusted in the analysis

Table 3 Threshold effect analysis of relationship of dietary vitamin E intake and RC, TC, HDL-C and LDL-C

Variables	Outcomes	β (95% CI)	*P*-value
Vitamin E intake < 11.18 mg	RC	-0.48 (-0.60, -0.37)	***< 0.001***
Vitamin E intake > 11.18 mg	RC	-0.08 (-0.15, 0)	***0.049***
Difference in effects		0.41 (0.25, 0.56)	***< 0.001***
Vitamin E intake < 11.19 mg	TC	-0.68 (-1.04, -0.32)	***< 0.001***
Vitamin E intake > 11.19 mg	TC	-0.15 (-0.39, 0.09)	0.210
Difference in effects		0.52 (0.04, 1.01)	***0.033***
Vitamin E intake < 10.77 mg	HDL-C	0.26 (0.13, 0.40)	***< 0.001***
Vitamin E intake > 10.77 mg	HDL-C	0.07 (-0.01, 0.16)	0.087
Difference in effects		-0.19 (-0.37, -0.01)	***0.037***
Vitamin E intake < 2.52 mg	LDL-C	-3.80 (-8.06, 0.46)	0.081
Vitamin E intake > 2.52 mg	LDL-C	-0.24 (-0.39, -0.09)	***0.002***
Difference in effects		3.56 (-0.71, 7.82)	0.103

Gender, ethnicity/race, age, education level, PIR, BMI, energy, protein, uric acid, ALT, AST, HbA1c, smoking status, stroke, diabetes and presence of cancer/malignancy were adjusted in the threshold effect analysis

on HDL-C levels [36]. Serum LDL-C and HDL-C levels did not change statistically significantly in 37 subjects who took 200 or 400 mg of vitamin E daily for 50 days in a clinical investigation [37]. A comparable result was observed in a randomized, double-blind study involving 78 participants, in which the administration of 728 mg α-tocopherol per day demonstrated no notable impact on HDL-C levels [38]. These findings were not entirely consistent with the results of this study, and further research is required to elucidate the association between vitamin E intake and blood lipid levels.

Substantial evidence suggests that oxidatively modified LDL plays a role in the development of atherosclerosis [39, 40]. Previous studies have indicated that vitamin E functions as a fat-soluble antioxidant, increasing the resistance of HDL and LDL to oxidation by scavenging free radicals [37, 41]. The haptoglobin 2–2 genotype has been linked to an elevated risk of developing cardiovascular disease in individuals with diabetes, and alpha-tocopherol has been shown to improve HDL function in haptoglobin 2–2 carriers [42, 43]. These results point to a possible effect of vitamin E on cardiovascular wellness. However, the correlation between vitamin E and HDL-C observed in this study was exclusive to female subjects, with no such association evident in males.

Table 4 Subgroup analysis for the association between vitamin E intake and RC, TC, HDL-C and LDL-C by gender, age, race and BMI

Total vitamin E intake (mg)	RC β (95%CI) *P*-value	TC β (95%CI) *P*-value	HDL-C β (95%CI) *P*-value	LDL-C β (95%CI) *P*-value
PGender				
Man	-0.20 (-0.27, -0.13) < 0.01	-0.45 (-0.67, -0.22) 0.03	0.01 (-0.08, 0.09) 0.92	-0.25 (-0.44, -0.05) 0.01
Woman	-0.24 (-0.32, -0.17) < 0.01	-0.18 (-0.44, 0.08) 0.19	0.32 (0.22, 0.41) < 0.01	-0.24 (-0.47, -0.02) 0.03
P for interaction	0.381	0.121	***< 0.001***	0.974
Age (years)				
< 50	-0.17 (-0.24, -0.11) < 0.01	-0.27 (-0.49, -0.05) 0.02	0.11 (0.03, 0.19) 0.01	-0.17 (-0.37, 0.02) 0.08
≥ 50	-0.27 (-0.35, -0.19) < 0.01	-0.37 (-0.65, -0.09) 0.01	0.17 (0.07, 0.27) < 0.01	-0.35 (-0.59, -0.10) 0.01
P for interaction	***0.039***	0.571	0.321	0.271
Race				
Mexican American	-0.03 (-0.23, 0.17) 0.75	0.07 (-0.57, 0.70) 0.84	0.08 (-0.15, 0.31) 0.48	0.05 (-0.51, 0.61) 0.85
Other Hispanic	-0.09 (-0.34, 0.17) 0.51	-0.28 (-1.07, 0.52) 0.50	0.03 (-0.26, 0.32) 0.85	-0.19 (-0.90, 0.52) 0.60
Non-Hispanic White	-0.23 (-0.30, -0.17) < 0.01	-0.29 (-0.49, -0.09) 0.01	0.16 (0.08, 0.23) < 0.01	-0.23 (-0.41, -0.06) 0.01
Non-Hispanic Black	-0.21 (-0.41, -0.01) 0.04	-0.63 (-1.27, 0.01) 0.06	0.16 (-0.07, 0.39) 0.17	-0.57 (-1.14, -0.01) 0.04
Other Race	-0.29 (-0.51, -0.08) 0.01	-0.57 (-1.23, 0.09) 0.09	0.14 (-0.10, 0.37) 0.27	-0.46 (-1.05, 0.12) 0.12
P for interaction	0.289	0.562	0.899	0.562
BMI (kg/m^2				
< 25	-0.17 (-0.25, -0.08) < 0.01	-0.17 (-0.44, 0.10) 0.21	0.11 (0.01, 0.20) 0.03	-0.18 (-0.41, 0.06) 0.14
25–30	-0.27 (-0.37, -0.17) < 0.01	-0.37 (-0.68, -0.06) 0.02	0.16 (0.04, 0.27) 0.01	-0.23 (-0.50, 0.04) 0.10
≥ 30	-0.18 (-0.29, -0.08) < 0.01	-0.43 (-0.76, -0.10) 0.01	0.07 (-0.04, 0.19) 0.22	-0.30 (-0.59, -0.01) 0.04
P for interaction	0.243	0.433	0.604	0.797

The following factors were adjusted: education level, PIR, energy, protein, uric acid, ALT, AST, HbA1c, smoking status, stroke, diabetes, and presence of cancer or malign

Study strengths and limitations

First, participants were drawn from NHANES, which employs a multistage, stratified, random sampling method, representing the overall US population. Large sample size enhances the reliability of the results. Furthermore, three models were employed to account for potential confounding factors, and subgroup analyses were conducted based on gender, age, BMI, and ethnicity/race. Threshold effect analysis identified many significant inflection points in the analysis.

This study has several limitations. Firstly, cross-sectional study results are limited to examining the association between vitamin E intake and blood lipid levels; they are unable to establish a causality. The total vitamin E intake data were based on participants' intake over just two days, which may need to take into account their enduring vitamin E condition. Additionally, the survey method for assessing total E intake is subject to recall bias. The analysis did not consider potential factors influencing blood lipid levels, such as other nutrient intakes and genetic background. More data are also needed on serum vitamin E and the components of RC, including IDL-C and VLDL-C, to ensure a more accurate analysis.

Moreover, the precise mechanisms through which dietary vitamin E influences RC, TC, HDL-C, and LDL-C serum levels remain unclear. The results might not apply to other nations and regions because every participant was an American. Therefore, further research is needed to validate these results in more extensive and diverse populations.

Conclusion

This study demonstrated that an elevated vitamin E intake was associated with an increase in serum HDL-C and a reduction in serum RC, TC, and LDL-C. These findings highlighted the function of nutritional factors in regulating blood lipid and cardiovascular health. They might also provide fresh perspectives on how to manage lipids in everyday life.

Abbreviations

RC	Remnant cholesterol
TC	Total cholesterol
HDL-C	High-density lipoprotein cholesterol
LDL-C	Low-density lipoprotein cholesterol
NHANES	National Health and Nutrition Examination Survey
ASCVD	Atherosclerotic cardiovascular disease
HR	Hazard ratio
CI	Confidence interval
IDL-C	Intermediate-density lipoprotein cholesterol
VLDL-C	Very low-density lipoprotein cholesterol
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
PIR	Family income to poverty
BMI	Body mass index
CHD	Coronary heart disease

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Author contributions

Y.W. contributed to the of the work and writing of the Manuscript. H.L., Z.H.Z. and F.W. contributed to the acquisition and analysisof data. J.L. and Z.Z.Z.

contributed to Production of graphs and tables. H.X. contributed to the conception of the work and Revision of the manuscript. All authors reviewed the manuscript.

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Data availability
The data are available from the NHANES website (https://www.cdc.gov/nchs/nhanes/nhanes).

Declarations

Ethics approval and consent to participate
The data used in this study are available online that have passed ethical review.

Consent for publication
All the authors approved to publication.

Competing interests
The authors declare no competing interests.

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