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ORIGINAL ARTICLE

Association between dietary vitamin E intake and chronic kidney disease events in US adults: a cross-sectional study from NHANES 2009–2016

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ABSTRACT

Background. The relationship between vitamin E supplementation and the prevalence of chronic kidney disease (CKD) is unclear. We discussed the relationship between vitamin E intake and CKD prevalence and further investigated the effect on different CKD risk strata.

Methods. We ultimately included 20 295 participants from the National Health and Nutrition Examination Survey (NHANES) database from 2009 to 2016. Multiple logistic regression and restricted cubic splines (RCS) were applied to explore the relationship between vitamin E intake and CKD prevalence and risk stratification. Subgroup analysis was applied to assess the stability of the association between vitamin E intake and CKD.

Results. In the CKD prevalence study, we found a negative association between high vitamin E intake and CKD prevalence through an adjusted multiple logistic regression model, the odds ratio (OR) was 0.86 [95% confidence interval (CI) 0.74–1.00; *P* for trend = .041] and RCS showed a nonlinear negative correlation (*P*-nonlinear = .0002, <.05). In the CKD risk stratification study, we found that in very high–risk patients, the OR was 0.51 (95% CI 0.32–0.84; *P* for trend = .006) and the RCS also showed a nonlinear negative correlation (*P*-nonlinear <.0001, <.05). Subgroup analysis demonstrated that the correlations were stable across populations (*P*-values >.01 for all interactions).

Conclusion. Dietary vitamin E intake was negatively associated with the prevalence of CKD in US adults. Increased vitamin E intake was a protective factor across CKD risk strata, and as vitamin E intake increased, there was a non-linear downward trend in the proportion progressing to very high–risk CKD.

LAY SUMMARY

We used data from the National Health and Nutrition Examination Survey (NHANES) from 2009 to 2016 to analyze dietary vitamin E intake and the prevalence of chronic kidney disease (CKD) among US adults (age ≥20 years). The results showed that the average daily intake of vitamin E among US adults was far below recommended levels and

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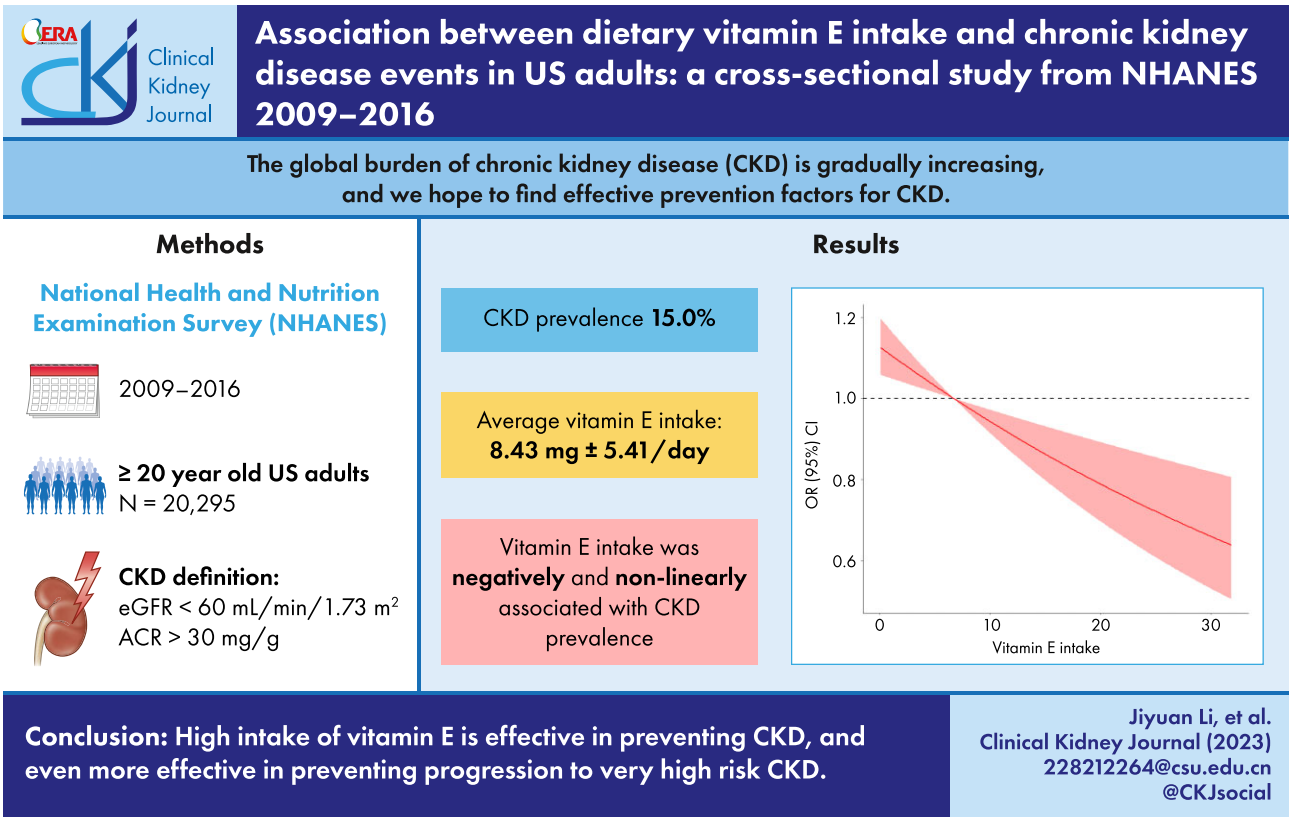
that high vitamin E intake was negatively associated with the prevalence of CKD, with this negative association being more pronounced in the very high–risk stratum of CKD patients. Therefore, increasing vitamin E intake is expected to be an important preventive measure for CKD.

GRAPHICAL ABSTRACT



Keywords: chronic kidney disease, cross-sectional study, National Health and Nutrition Examination Survey, prevalence, vitamin E

INTRODUCTION

The number of patients who have chronic kidney disease (CKD) is on the rise [1], with diabetes and hypertension causing the majority of cases [2, 3], affecting approximately 15%–20% of adults worldwide [4], and increasing the risk of adverse outcomes. CKD is the primary reason for catastrophic health expenditures (health expenditures exceeding 40% of household income) globally [5] and is predicted to become the fifth leading cause of death worldwide by 2040 [6]. Currently, there is a large gap in the treatment capacity for CKD around the world, and CKD patients in some areas are not receiving effective treatment such as dialysis and transplantation [7, 8]. Therefore, it is necessary to ascertain preventive measures to reduce the prevalence of CKD. Some surveys have suggested that dietary factors are related to the prevalence of CKD. The risk of developing CKD may be negatively associated with increased fiber and potassium intake and moderate alcohol consumption, but positively associated with increased salt intake in the diet [9–12].

Vitamin E is lipid-soluble and has an important function in preventing oxidative damage to cellular components [13, 14]. However, vitamin E can only be obtained from external sources and is not synthesized in the body, so dietary intake of vitamin E is particularly important. The pro-oxidative setting produced by kidney impairment may enhance the need for vitamin E to protect cells against lipid peroxidation [15]. Several cohort studies have demonstrated a negative or L-shaped association between vitamin E intake and the prevalence of CKD [12, 16], but one study has suggested a non-significant association [17]. In the past studies, large sample size studies on the relationship between vitamin E and CKD in the USA were limited, and the impact of differences in demographics and socio-economics and disease status on the association of vitamin E intake with CKD has not been adequately investigated. Therefore, we used National Health and Nutrition Examination Survey (NHANES) data from 2009 to 2016 to perform a large cross-sectional survey to explore the relationship between dietary vitamin E intake and CKD prevalence in Americans, and further explored the relationship between vitamin E intake and different CKD risk stratification.

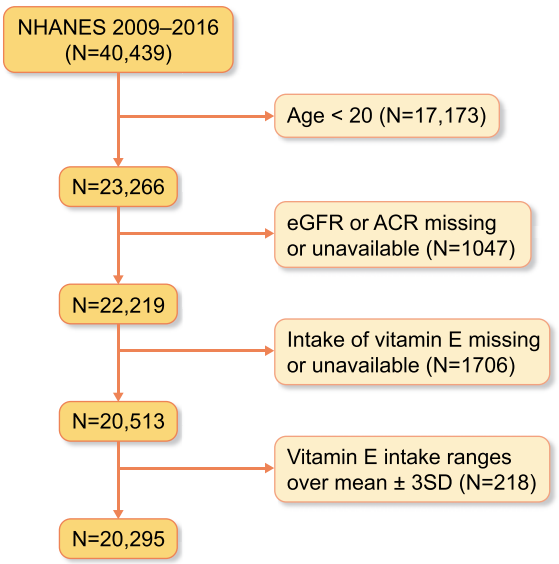


Figure 1: Flowchart of participant screening based on NHANES database from 2009 to 2016.

MATERIALS AND METHODS

Study design and population

NHANES is a population-based investigation that serves as a research project to collect health and nutrition data from the US household population [18]. We selected 23 266 subjects aged ≥20 years from NHANES data for the four survey cycles 2009–16. We excluded data for which estimates of glomerular filtration rate (eGFR) or albumin-to-creatinine ratio (ACR) were missing ($N = 1706$) and those lacking dietary vitamin E intake data ($N = 1706$). In addition, subjects with daily vitamin E intakes above the mean ± 3 SD were excluded ($N = 218$). Eventually, 20 295 subjects were entered into the final analysis (Fig. 1). For NHANES, the study protocol was authorized by the Centers for Disease Control's Institutional Review Board, and informed consent was signed by all participants included in the study.

Study definitions

We obtained information on participants' dietary vitamin E intake from the recall diet interview section of the NHANES database. The dietary recall interview was collected in person at a mobile examination center (MEC), and the first 24-h dietary vitamin E intake from that interview was defined as the independent variable in this study. We defined the dependent variable as the presence of CKD, which in the current study was mainly defined as a urinary ACR >30 mg/g or an eGFR <60 mL/min/1.73 m^2 [16, 19, 20]. Risk stratification of CKD patients was also performed according to CKD staging and albuminuria grading, which were classified as moderate risk, high risk and very high risk [21]. Based on available clinical knowledge and studies, we also included factors that were potentially associated with vitamin E intake and presence of CKD. Demographic characteristics included age (20–40 years, 40–60 years, >60 years); sex (female/male); race (Mexican American, non-Hispanic Black, non-Hispanic white, other races); marriage (married/cohabiting with partner, widowed/divorced/separated, never married); education (<high school, high school, >high school); and other potential correlates included body mass index (BMI) (≤25 kg/m^2, 25–30 kg/m^2 , >30 kg/m^2), high-density lipoprotein (HDL), total cholesterol (TC), smoking status (no/yes), diabetes (no/yes), alcohol status (no/yes), hypertension (no/yes), serum potassium and serum sodium. BMI was calculated as weight (kg) divided by height squared (m^2). The definition of hypertension is an average systolic blood pressure ≥130 mmHg or an average diastolic blood pressure ≥80 mmHg [22]. Participants with glucose levels ≥126 mg/dL fasting or 2-h level >200 mg/dL (measured by an oral glucose tolerance test) or treated with antidiabetic medication or diagnosed in self-report form were considered to have diabetes [23]. The definition of smoking was at least 100 cigarettes in a lifetime and drinking was defined as at least 12 drinks per year. The simplified Modification of Diet in Renal Disease equation was used for the calculation of eGFR [24]. We performed random forest interpolation for covariates with missing values [25].

Statistical analyses

NHANES uses a complex multi-stage probabilistic sampling system, and we also give appropriate weights to the data samples during the statistical and analytical process. Categorical variables are expressed as the number of people before weighting (weighted percentages), and continuous variables are expressed as the weighted mean ± standard error. Weighted analysis of variance and weighted chi-square tests were used to analyze continuous and categorical variables, respectively, so as to discuss between-group differences in vitamin E intake. Daily vitamin E intake was divided into three groups of Q1, Q2 and Q3 according to the tertile level. Logistic regression models were applied to test the relationship between vitamin E intake and CKD, using the lowest tertile class Q1 as a reference. Three correction models were developed: Model 1 did not adjust for relevant factors, and Model 2 adjusted for basic characteristics such as age, race and gender. Model 3 adjusted for all potential correlates included (age, sex, ethnicity, marital status, education level, BMI, HDL, TC, smoking status, alcohol status, diabetes, hypertension, serum potassium and serum sodium). Dose–response relationships between CKD prevalence and vitamin E intake were examined using restrictive cubic splines (RCS) curves. Subgroup analysis was stratified by age, sex, race, marital status, education, BMI, smoking status, hypertension, diabetes mellitus and alcohol status with the aim of discussing the stability of the association between CKD prevalence and vitamin E intake in each stratified subgroup. The interactions between the different covariates were discussed by likelihood ratio tests and the results of the subgroup analyses were visualized as forest plots. Patients with CKD are classified as moderate risk, high risk and very high risk according to the risk classification. Logistic regression models were also used to test the relationship between vitamin E intake and different CKD risk stratification, using the lowest tertile class Q1 as a reference. We used R4.21 version for all analyses in this study [19, 26, 27].

RESULTS

Baseline data characteristics

A total of 40 439 participants were enrolled in this study, and 20 295 participants were selected after inclusion of relevant criteria. A weighted population baseline table was made according to the daily dietary vitamin E intake grading (Q1 ≤5.060 mg/day, Q2 5.060–8.770 mg/day, Q3 ≥8.770 mg/day) (Table 1). Of these,

Table 1: Baseline information, weighted.

Characteristic	Overall	Vitamin E intake			*P*-value
		Q1	Q2	Q3	
N	20 295	6748	6778	6769	
Age, *n* (%)					<.001
20–40 years	7314 (38.1)	2273 (37.6)	2472 (37.7)	2569 (38.8)	
40–60 years	6848 (37.2)	2176 (34.5)	2250 (36.6)	2422 (39.7)	
>60 years	6133 (24.8)	2299 (27.9)	2056 (25.7)	1778 (21.5)	
Gender, *n* (%)					<.001
Male	10 468 (51.8)	3987 (60.8)	3580 (54.0)	2901 (42.8)	
Female	9827 (48.2)	2761 (39.2)	3198 (46.0)	3868 (57.2)	
Race, *n* (%)					<.001
Mexican America	3036 (8.7)	1018 (8.8)	1081 (9.6)	937 (7.8)	
Non-Hispanic Black	4266 (11.2)	1526 (13.3)	1397 (11.4)	1343 (9.5)	
Non-Hispanic white	8423 (66.3)	2581 (62.2)	2772 (65.4)	3070 (70.2)	
Other races	4570 (13.8)	1623 (15.7)	1528 (13.7)	1419 (12.5)	
Marriage, *n* (%)					<.001
Married/living with partner	12 000 (62.2)	3708 (57.3)	4101 (63.4)	4191 (65.0)	
Widowed/divorced/separated	4454 (18.6)	1778 (23.1)	1446 (18.3)	1230 (15.4)	
Never married	3841 (19.2)	1262 (19.6)	1231 (18.3)	1348 (19.7)	
Education, *n* (%)					<.001
<High school	4872 (16.0)	2104 (21.7)	1549 (15.3)	1219 (12.1)	
High school	4506 (21.4)	1589 (24.4)	1550 (22.2)	1367 (18.4)	
>High school	10 917 (62.6)	3055 (53.9)	3679 (62.5)	4183 (69.5)	
BMI, *n* (%)					<.001
≤25 kg/m^2	5876 (29.9)	1882 (28.3)	1956 (29.8)	2038 (31.3)	
25–30 kg/m^2	6633 (33.0)	2136 (31.0)	2275 (33.1)	2222 (34.4)	
>30 kg/m^2	7786 (37.1)	3730 (40.7)	2547 (37.1)	2509 (34.3)	
HDL, mg/dL	53.75 ± 16.68	53.12 ± 16.68	54.10 ± 16.58	53.94 ± 16.76	.083
TC, mg/dL	194.15 ± 41.25	194.75 ± 42.36	194.44 ± 41.49	193.42 ± 40.14	.332
Smoking status, *n* (%)					.005
No	11 369 (55.6)	3704 (53.1)	3863 (57.1)	3805 (56.2)	
Yes	8926 (44.4)	3044 (46.9)	2918 (42.9)	2964 (43.8)	
Diabetes, *n* (%)					<.001
No	16 499 (85.7)	5291 (83.2)	5540 (85.8)	5668 (87.4)	
Yes	3796 (14.3)	1457 (16.8)	1238 (14.2)	1101 (12.6)	
Alcohol status, *n* (%)					<.001
No	9211 (36.9)	3616 (44.9)	3040 (36.5)	2555 (30.9)	
Yes	11 084 (63.1)	3132 (55.1)	3738 (63.5)	4214 (69.1)	
Hypertension, *n* (%)					.003
No	9676 (51.6)	3001 (48.7)	3251 (52.4)	3424 (53.2)	
Yes	10 619 (48.4)	3747 (51.3)	3527 (47.6)	3345 (46.8)	
Serum potassium, mmol/L	3.97 ± 0.33	3.95 ± 0.34	3.97 ± 0.32	3.99 ± 0.32	<.001
Serum sodium, mmol/L	139.15 ± 2.17	139.14 ± 2.20	139.20 ± 2.18	139.11 ± 2.14	.233
eGFR, mL/min1.73 m^2	92.54 ± 25.33	91.65 ± 25.85	92.64 ± 26.99	93.15 ± 23.31	.022
ACR, mg/g	33.05 ± 242.85	42.72 ± 293.50	31.77 ± 237.32	26.59 ± 199.90	.002
CKD, *n* (%)					<.001
No	16 578 (85.0)	5321 (82.5)	5547 (84.5)	5710 (87.4)	
Yes	3717 (15.0)	1427 (17.5)	1231 (15.5)	1059 (12.6)	

Mean ± standard error for continuous variables, percentages (%) for categorical variables.
Q1, ≤5.060 mg/day; Q2, 5.060–8.770 mg/day; Q3, ≥ 8.770 mg/day).

48.2% were female, 51.8% were male, 8.7% were Mexican American, 11.2% were non-Hispanic Black, 66.3% were non-Hispanic white and 13.8% were other races. In addition, 66.2%, 18.6% and 19.2% of participants were married/living with a partner, widowed/divorced/separated and never married, respectively. In addition, 62.6% had education higher than high school, 21.4% had only high school education and 16.0% had education lower than high school. Furthermore, 14.3% had diabetes mellitus, 48.4% had hypertension, 44.4% smoked and 63.1% drank alcohol. The weighted overall prevalence of CKD in this study was 15.0%, and CKD prevalence was higher in participants in the low vitamin E intake tertile than in those in the high vitamin E intake tertile (Q1, 17.5%; Q2, 15.5%; Q3, 12.6%).

The relationship between vitamin E and CKD

To avoid the influence of extreme data on the statistical analysis, data >3 SD from the mean of vitamin E intake have been excluded before including the final data. We performed a multifactorial logistic regression analysis (Table 2), in which Model 1 was unadjusted for correlates and Model 2 was adjusted for basic characteristics such as age, race and gender. Model 3

Table 2: Findings from a multiple logistic regression analysis of the association between vitamin E intake and CKD, weighted.

	Actual population numbers	Weight percentage of population (%)	OR (95% CI)		
			Model 1	Model 2	Model 3
Q1	6748	29.4	Ref	Ref	Ref
Q2	6778	33.0	0.86 (0.76, 0.97)	0.90 (0.79, 1.03)	0.97 (0.85, 1.11)
Q3	6769	37.5	0.68 (0.59, 0.77)	0.78 (0.67, 0.90)	0.86 (0.74, 1.00)
P for trend			<.001	.001	.041

Q1, ≤ 5.060 mg/day; Q2, 5.060–8.770 mg/day; Q3, ≥ 8.770 mg/day.
Model 1: adjusted for no covariates.
Model 2: adjusted for basic characteristics such as age, ethnicity and gender.
Model 3: adjusted for all potential correlates.

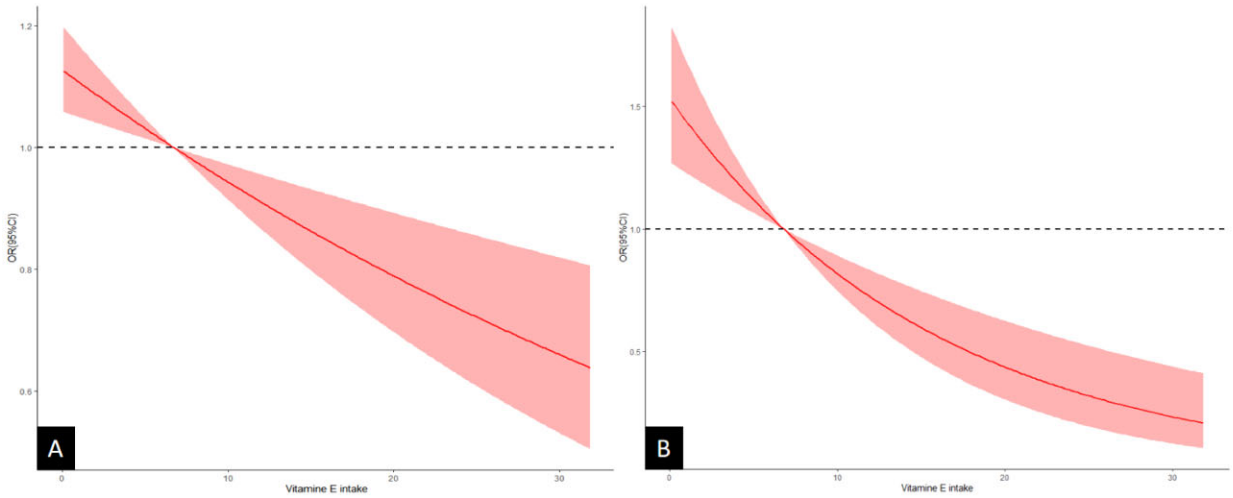


Figure 2: (**A**) RCS curves describing the dose–response relationship between vitamin E intake and CKD. (**B**) RCS curves describing the dose–response relationship between vitamin E intake and very high–risk stratification for CKD. The following covariates were adjusted for: age, sex, race, marital status, education level, BMI, HDL, TC, smoking status, alcohol status, diabetes mellitus, hypertension, serum sodium and serum potassium.

adjusted for all potential correlates included. In the analyses, the OR (referenced to Q1) were 0.86 (95% CI 0.76–0.97) and 0.68 (95% CI 0.59–0.77) in the 2nd tertile and 3rd tertile in Model 1, 0.90 (95% CI 0.79–1.03) and 0.78 (95% CI 0.67–0.90) in Model 2, and 0.97 (95% CI 0.85–1.11) and 0.86 (95% CI 0.74–1.00) in Model 3. The *P* for trend of the above models were <.001, 0.001 and 0.041, respectively. This means that the prevalence of CKD tends to decrease as vitamin E intake increases. In addition, we plotted the dose–response relationship between CKD events and vitamin E intake based on RCS and found a strong nonlinear negative correlation (*P*-nonlinear = .0002, <.05) (Fig. 2). Finally, we further performed risk stratification for CKD patients, using the logistic regression method described above. The results of the adjusted Model 3 showed that in moderate-risk CKD patients, the OR of high vitamin E intake Q3 was 0.92 (95% CI 0.78–1.08; *P* for trend = .267). In high-risk patients, the OR was 0.77 (95% CI 0.57–1.03; *P* for trend = .006). In very high–risk patients, the OR was 0.51 (95% CI 0.32–0.84; *P* for trend = .006) (Table 3), and RCS showed nonlinear (*P*-nonlinear <.0001, <.05) correlations (Fig. 2). In conclusion, there was a stable negative correlation between high intake of vitamin E and the prevalence of CKD, and in the multivariate adjusted model, high vitamin E intake reduced the risk of CKD by 8% for moderate risk, 23% for high risk and 49% for very high risk compared with low vitamin E intake.

Subgroup analysis

We performed subgroup analyses and drew forest plots to visualize the results (Fig. 3), and analyzed the interactions between CKD and the categorical variables in the study. The results demonstrated a stable negative association between high vitamin E intake and the incidence of CKD events (*P*-values >.01 for all interactions) in people with different demographic characteristics, lifestyle habits and disease status.

DISCUSSION

In the study, we investigated the relationship between the prevalence of CKD and dietary vitamin E intake in the US adult population. A total of 20 295 participants were enrolled in this study, with a mean daily dietary vitamin E intake of 8.43 mg ± 5.41/day, far below the recommended level (15 mg/day for individuals >14 years of age) [28]. The overall prevalence of CKD was 15.0%. In multivariate logistic regression analysis, higher vitamin E intake was found to be independently associated with lower prevalence of CKD after controlling for relevant covariates, and this association became stronger as the CKD risk classification increased. By plotting RCS curves, we found a negative correlation between vitamin E intake and the prevalence of CKD. Therefore, supplemental vitamin E intake appears to contribute significantly to the prevention of CKD.

Table 3: Findings of a multiple logistic regression analysis of the association between vitamin E intake and CKD risk stratification, weighted.

Stratification factors	Cases/non-cases	Q1	Q2	Q3	*P* for trend
CKD cases with moderate risk of progression	2552/16 578				
Model 1		Ref	0.92 (0.80, 1.07)	0.77 (0.66, 0.90)	<.001
Model 2		Ref	0.95 (0.82, 1.11)	0.86 (0.74, 1.00)	.049
Model 3		Ref	1.01 (0.86, 1.18)	0.92 (0.78, 1.08)	.267
CKD cases with high risk of progression	714/16 578				
Model 1		Ref	0.87 (0.66, 1.14)	0.57 (0.43, 0.75)	<.001
Model 2		Ref	0.93 (0.68, 1.26)	0.70 (0.52, 0.95)	.026
Model 3		Ref	1.01 (0.75, 1.37)	0.77 (0.57, 1.03)	.083
CKD cases with very high risk of progression	451/16 578				
Model 1		Ref	0.52 (0.40, 0.67)	0.32 (0.23, 0.47)	<.001
Model 2		Ref	0.58 (0.44, 0.76)	0.46 (0.31, 0.69)	<.001
Model 3		Ref	0.62 (0.44, 0.86)	0.51 (0.32, 0.84)	.006

Please refer to Table 2 for details.

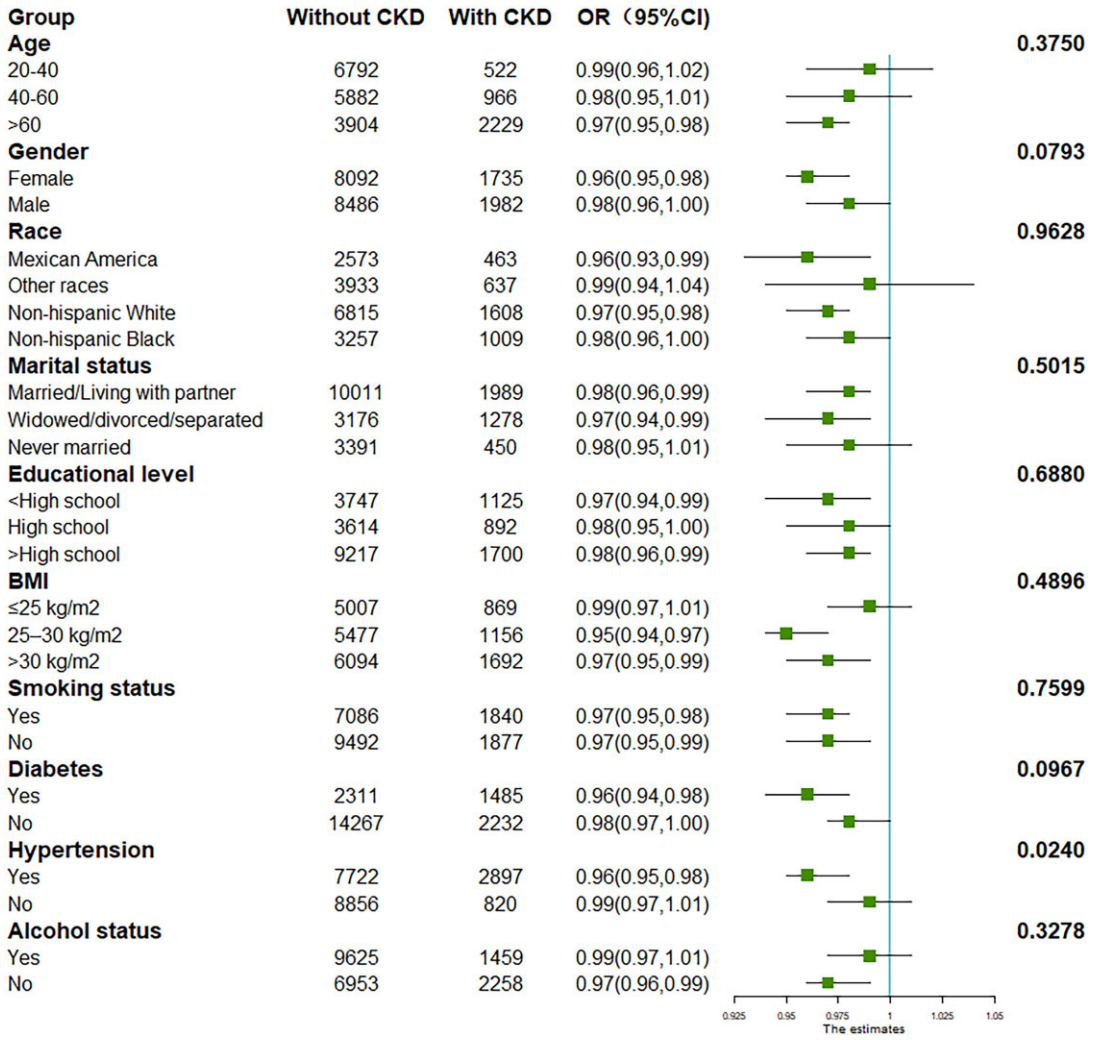


Figure 3: Forest plots for subgroup analysis. Subgroup analysis was stratified by age, gender, race, marital status, education level, BMI, smoking status, diabetes, hypertension and alcohol status.

Currently, the relationship between vitamin E and CKD prevalence remains unclear. In a prospective cohort study in Iran containing 1692 participants, a negative association between dietary vitamin E intake and the prevalence of CKD was suggested [12], while another study containing 8901 participants found no correlation between Korean individuals [17]. Meanwhile, a prospective study of 4038 participants from four US urban areas in young adults aged 18–30 years suggested an L-shaped association between dietary vitamin E intake and the prevalence of CKD [16]. These conflicting results may be due to differences in the

number of participants, age, dietary habits, economic conditions and ethnic background. For this reason we conducted a cross-sectional study with a large sample using the NHANES database stratified multi-stage probability sampling method and assigning weights to the sample data in order to explore the association between dietary vitamin E intake and the incidence of CKD in all ≥20-year-old adults in the USA, and the results showed a negative correlation.

The biological mechanism of this potential association is not yet clear. On one hand, as CKD progresses further, patients are at increased risk for oxidative damage and lipid abnormalities, which may impair the absorption and antioxidant effect of vitamin E [29, 30]. One review has shown that a relative lack of vitamin E was seen in CKD [15]. On the other hand, several studies have shown that vitamin E scavenges fatty hydrogen peroxide radicals through its H-atom donor properties and partially alleviates oxidative damage and hyperlipid peroxidation in CKD [15, 31, 32]. In addition, vitamin E can function as a lipophilic radical-trapping antioxidant to inhibit ferroptosis [33], which is currently considered one of the most important causes of nephron loss [34]. However, the effect of vitamin E as an antioxidant and nutritional material in controlling further progression and degenerative transformation of tissues in CKD still needs further investigation. Supplementation with more vitamin E in patients who receive HD may be useful to improve the vitamin E/lipid ratio to reduce oxidative damage in tissues [30], and a randomized controlled study shows that increasing vitamin E intake can reduce the risk of progression of diabetic nephropathy [35]. Recent studies suggest that hemodialysis with vitamin E–coated membranes may improve anemia by decreasing oxidative stress and inflammatory status [36–38]. Although vitamin E has been used to treat some patients with CKD, further research is necessary to ascertain whether increasing vitamin E intake can be an important preventive measure for CKD.

Some strengths of this study are worth mentioning. First, our data were obtained from the NHANES database and the reliability of the results was enhanced by the use of appropriate weights and adjustment for confounding factors during the statistical analysis. Second, this is the first study comprising a large national sample ($n = 20\ 295$) to assess the relationship between daily vitamin E intake and CKD prevalence in US adults. In addition, we further performed risk stratification of CKD cases and explored the effect of different vitamin E intakes on different CKD risk strata. However, limitations of the study exist. First, since this is a cross-sectional study, it can only show that there is an association between vitamin E intake and CKD, and no causal relationship can be established. Second, there may be selection bias, as many subjects were excluded from the study due to missing or unavailable data. Finally, we used dietary data from the first 24-h dietary recall interview which may be subject to recall bias and do not fully reflect each individual's vitamin E intake.

CONCLUSIONS

In conclusion, our investigators found that dietary vitamin E intake was negatively associated with the prevalence of CKD in US adults and that the average intake of vitamin E in US adults was far below the recommended intake. When risk stratification for CKD was performed, increased daily vitamin E intake was a protective factor across CKD risk strata. As daily vitamin E intake increased, there was a non-linear downward trend in the proportion progressing to very high–risk CKD. Therefore, higher intake of vitamin E is expected to be a primary prevention measure for CKD and to reduce the prevalence of CKD.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data underlying this article will be shared on reasonable request to the corresponding author.

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