PAPER

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Association between the composite dietary antioxidant index and chronic kidney disease: evidence from NHANES 2011–2018†

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Objectives: There is growing evidence that antioxidant-rich diets protect against chronic kidney disease (CKD). However, the relationship between the Composite Dietary Antioxidant Index (CDAI), an important measure of an antioxidant diet, and CKD has received little attention. Therefore, here we investigated the relationship between the CDAI and CKD through a cross-sectional analysis of the National Health and Nutrition Examination Survey (NHANES) 2011–2018 data. *Methods*: The CDAI was calculated based on the intake of six dietary antioxidants. A survey-based multivariate linear regression analysis was performed to analyze the independent relationship between the CDAI and CKD. Weighted multivariate regression and subgroup analyses were conducted to explore the relationship between the CDAI and CKD. *Results*: A total of 6874 NHANES participants represented 181.9 million non-institutionalized US residents (mean age, 46.43 ± 0.38 years; 49.87% female; 40.62% non-Hispanic white; 20.24% non-Hispanic black; and 13.94% Mexican American). The weighted linear regression model with full adjustment for confounding variables was −0.0155 (−0.0417, 0.0107) for Q2 (*P* for trend <0.0001), −0.0052 (−0.0346, 0.0242) for Q3 (*P* for trend <0.0001), and −0.0305 (−0.0491, −0.0120) for Q4 (*P* for trend = 0.0094) upon comparison with the lowest quartile of the CDAI. None of the interactions in any subgroup analysis were statistically significant except for individuals with a history of diabetes or the aged population (≥60 years) (*P* for interaction <0.05). *Conclusions*: The CDAI was positively associated with a lower prevalence of CKD in adults in the United States. Further large-scale prospective studies are required to analyze the role of the CDAI in CKD.

1. Introduction

Kidney failure is a major health concern worldwide. Chronic kidney disease (CKD) is a chronic disease characterized by proteinuria; normal or reduced estimated glomerular filtration (eGFR) rates; and progressive glomerular, tubular and interstitial damage. CKD, which affects 15–20% of adults globally, is associated with a very high risk of premature death due to cardiovascular disease, the most common cause of death in CKD.[1,2] Therefore, clinicians should pay attention to this condition.

The Composite Dietary Antioxidant Index (CDAI) is a composite estimate of an individual's overall pro- and antioxidant exposure status.[3] The CDAI is an individual antioxidant index based on a combination of dietary antioxidants (manganese,

selenium, zinc and vitamins A, C, and E).[4] Previous studies reported that a high CDAI is associated with a reduced risk of various types of cancer and diabetes and positively correlated with the plasma levels of S-Klotho, an anti-aging indicator.[4–8] A high CDAI is associated with a decreased risk of all-cause and cardiovascular mortality.[5] Oxidative stress is correlated with renal damage; however, the relationship between the inflammatory indicator CDAI and CKD remains unclear.[9–11]

Therefore, this study aimed to explore the association between the CDAI and CKD among the participants of the US National Health and Nutrition Examination Survey (NHANES). We hypothesized that an elevated CDAI would be associated with a lower risk of CKD.

2. Materials and methods

2.1 Study population

The NHANES is a series of cross-sectional surveys representing the non-institutionalized civilian population of the United States (https://www.cdc.gov/nchs/nhanes/). The NHANES

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includes demographic, socioeconomic, dietary, and health-related questionnaire data collected through face-to-face interviews, physical and physiological examinations, and extensive laboratory tests. A detailed description of the NHANES was reported previously.[12,13] A total of 39 156 participants completed the survey in four NHANES cycles (NHANES 2011–2012, 2013–2014, 2015–2016, and 2017–2018 cycles). Among them, we excluded 15 331 participants under 18 years of age, 1548 for whom a urinary albumin/creatinine ratio (UACR) was lacking, 15 331 participants for whom a CDAI was lacking, 1021 participants for whom eGFR was lacking, 10 499 participants for whom weight values were unavailable, 836 for whom alcohol status was unavailable, 773 for whom a poverty income ratio (ratio of family income to poverty threshold [PIR]) was unavailable, 79 for whom body mass index (BMI) information was lacking, 221 for whom waist circumference (WC) information was lacking, 4 for whom the education status was lacking, 114 for whom the smoking status was unavailable, 77 without a history of diabetes mellitus (DM), 118 participants for whom low-density lipoprotein cholesterol (LDL-C) information was lacking and 4 participants for whom serum uric acid (UA) information was lacking. Therefore, a total of 6874 participants was included in this study (Fig. 1).

2.2 Data collection

2.2.1 Exposure variable. Diet affects the CDAI. In the NHANES, dietary intake data were obtained *via* a 24-hour dietary recall interview conducted by the mobile inspection center. The NHANES computer-assisted dietary interview system collected and recorded the types and amounts of food and beverages (including all water) consumed in the 24 hours prior to the interviews. The University of Texas Food Intake Analysis System and the United States Department of Agriculture Survey Nutrient Database were used to assess the dietary nutrient intake. The nutritional estimates did not include any nutrients obtained from dietary supplements or medications.

The development of the CDAI was described and verified in a previous report.[3] The intake of antioxidants, micronutrients, and total energy was calculated using the US Department of Agriculture's Dietary Research Food and Nutrition Database.[14] According to the questionnaire survey, we determined each participant's intake of dietary supplements in the past month, including dose, frequency and number of doses.[15] To estimate the CDAI, we standardized each of the same six dietary vitamins and minerals by subtracting the global average and divid-
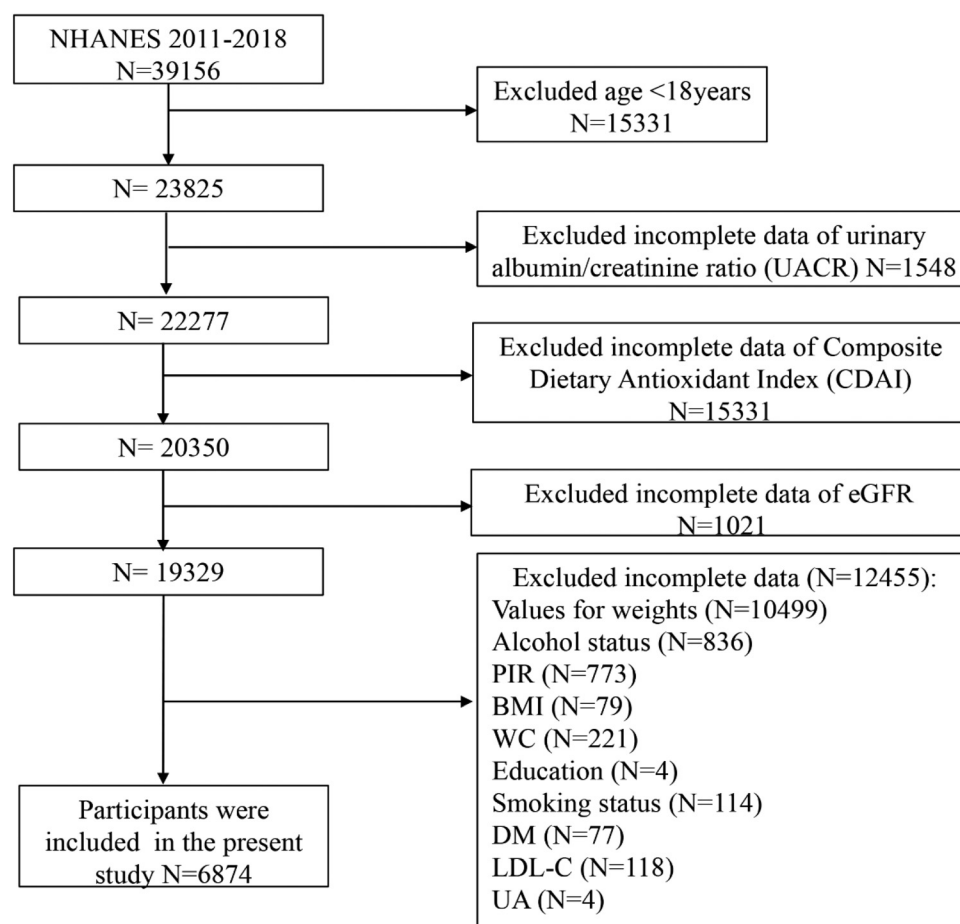

Fig. 1 A flowchart showing the selection of study participants.

ing by the global standard deviation. We then calculated the CDAI by adding the standard intake of these vitamins and minerals as follows.

$$CDAI = \sum_{i=1}^{n=6} \frac{Individual\ Intake - Mean}{SD}$$

2.2.2 Outcome variable. The urine albumin/creatinine ratio was used to compute the UACR. The eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration algorithm. CKD was identified at a UACR $\geq$ 30 mg g^{-1} and/or an eGFR < 60 mL per min per 1.73 m^2.[16]

2.2.3 Baseline data collection. Information about covariates was obtained using baseline questionnaires. These questionnaires included questions about age, gender, race/ethnicity, education level, marital status, smoking status, PIR, BMI, WC, and self-reported baseline medical history such as DM and hypertension. BMI was computed by measuring the height and weight. Blood glucose (Glu), serum creatinine (Cr), UA, total cholesterol (TC), triglycerides (TG), alanine aminotransferase (ALT), and aspartate aminotransferase (AST) were included in the biochemical profile.

We divided race into non-Hispanic white, non-Hispanic black, Mexican-American, and other groups. Educational background was divided into less than high school (less than 9th grade or 9–11th grade [including 12th grade with no diploma]), high school or equivalent, and more than high school (some college or associate's degree or college graduate or above). The marital status was classified as never married, divorced/widowed/separated, or married/living with a partner. The smoking status was categorized as never/former/now smoker. The diagnostic criteria for alcohol consumption and status were: current heavy drinking ($\geq$4 drinks per day for men, $\geq$3 drinks per day for women, or binge drinking $\geq$5 days per month), current moderate drinking ($\geq$3 drinks per day for men, $\geq$2 drinks per day for women, or binge drinking $\geq$2 days per month), or current light drinking (not meeting the above criteria), never (had <12 drinks in lifetime), former (had $\geq$12 dinks in 1 year and did not drink last year, or did not drink last year but drank $\geq$12 drinks in lifetime).

Hypertension was defined as average systolic blood pressure (SBP) $\geq$ 140 mmHg and/or average diastolic blood pressure (DBP) $\geq$ 90 mmHg; or self-reported diagnosis of hypertension and intake of antihypertensive medications.[17,18] DM was defined as (1) doctor diagnosed diabetes; (2) glycohemoglobin >6.5%; (3) fasting glucose $\geq$7.0 mmol L^{-1}; (4) random blood glucose $\geq$11.1 mmol L^{-1}; (5) two-hour oral glucose tolerance test blood glucose $\geq$11.1 mmol L^{-1}; and (6) use of diabetes medication or insulin.[19] The full measurement technique for these variables is available at **https://www.cdc.gov/nchs/nhanes/**.

2.3 Statistical analyses

Continuous variables are expressed as weighted means ± standard deviation and were compared using weighted linear regression analysis. Categorical variables are described using unweighted frequencies (weighted percentages) and were compared using the chi-squared test. These means and frequencies can be generalized to the US adult population. A multivariate linear regression model was used to study the correlation between the CDAI and CKD. The CDAI is generally converted into categorical variables according to quartiles, and the P values of the trend are calculated. Three models are used in this study. Model 1 was a crude model not adjusted for potential confounding factors. Model 2 was adjusted for age, gender, race and education level. Model 3 was further adjusted for alcohol consumption, smoking status, BMI, WC, PIR, SBP, Glu, UA, TG, eGFR, UACR, hypertension and DM. The association of the CDAI with CKD was further analyzed stratified by gender (male/female), age (<60/$\geq$60 years), smoking status (never/former/now smoker), hypertension (yes/no), and DM (yes/no/preDM). The BMI was categorized as <25.0, 25.0–30.0 and $\geq$30.0 kg m^{-2}, corresponding to normal weight, overweight and obese, respectively. PIR was categorized as <1.3, 1.3–1.8, and >1.8. These stratification factors were also considered pre-specified potential effect modifiers. An interaction term was also added to test for heterogeneity in the associations between subgroups. All analyses were performed using R software version 4.2.2 (**https://www.R-project.org**; R Foundation, Austria). Appropriate examination weights were used to represent the complex survey design. Statistical significance was set at P < 0.05.

3. Results

3.1 Participant characteristics

A total of 6874 participants (mean age, 46.43 ± 0.38 years; 3428 male, 3446 female) were included. Table 1 shows the weighted baseline characteristics of the study participants. BMI, WC, Glu, Cr, TG, UACR, UA and SBP were lower in the non-CKD *versus* the CKD group (P < 0.05). Education level and PIR were higher in the non-CKD group. In terms of the smoking status, 29.1% of patients with CKD had a history of smoking or were current smokers. Moderate-drinkers were more often in non-CKD than CKD participants. In contrast, no significant trends were observed for race, ALT, AST, TC, high-density lipoprotein cholesterol (HDL-C) and LDL-C (P > 0.05).

3.2 Association between the CDAI and CKD

Table 2 displays the relationships between the CDAI and diverse variables, such as age, gender, race, education, PIR, alcohol status, smoking status, BMI, WC, SBP, DBP, history of DM and hypertension, and some biochemical indicators. An increase in the CDAI serves as a protective factor against CKD. Table 3 presents the above relationships assessed using multivariate analyses. In this study, three models were constructed to examine the relationship between the CDAI and CKD. Model 1, no covariate adjustment; Model 2 adjustment for age, gender, race and education level; Model 3, Model 2 plus adjustment for the smoking status, alcohol status, BMI, WC, PIR, SBP, Glu, UA, TG, eGFR, UACR, hypertension and DM as

Table 1 General characteristics of participants (N = 6874) stratified by CKD or non-CKD in the NHANES 2011–2018 data

Characteristic	Overall	Non-CKD (N = 5777)	CKD (N = 1097)	P value
Age [mean (SD)]	46.43 (0.38)	44.58 (0.40)	59.06 (0.84)	<0.0001
Gender (%)				<0.001
Male	3428 (49.87)	2897 (88.91)	531 (11.09)	
Female	3446 (50.13)	2880 (85.58)	566 (14.42)	
Race/ethnicity (%)				0.07
Non-Hispanic white	2792 (40.62)	2306 (87.15)	486 (12.85)	
Non-Hispanic black	1391 (20.24)	1145 (84.90)	246 (15.10)	
Mexican American	958 (13.94)	814 (88.04)	144 (11.96)	
Other race	1733 (25.21)	1512 (88.94)	221 (11.06)	
Education (%)				<0.0001
Less than high school	1290 (19.31)	1017 (81.32)	273 (18.68)	
High school or equivalent	1463 (21.9)	1173 (83.59)	290 (16.41)	
More than high school	3926 (58.78)	3402 (89.39)	524 (10.61)	
PIR [mean (SD)]	2.99 (0.06)	3.04 (0.06)	2.68 (0.09)	<0.0001
Alcohol status (%)				<0.0001
Never	986 (14.34)	810 (83.52)	176 (16.48)	
Former	873 (12.7)	657 (77.84)	216 (22.16)	
Mild	2493 (36.27)	2097 (87.59)	396 (12.41)	
Moderate	1131 (16.45)	993 (90.08)	138 (9.92)	
Heavy	1391 (20.24)	1220 (90.55)	171 (9.45)	
Smoking status (%)				<0.0001
Never	3975 (57.83)	3427 (88.86)	548 (11.14)	
Former	1595 (23.2)	1247 (82.69)	348 (17.31)	
Now	1304 (18.97)	1103 (88.21)	201 (11.79)	
BMI [mean (SD)]	29.15 (0.15)	28.96 (0.15)	30.43 (0.38)	<0.001
WC[mean (SD)]	99.59 (0.37)	98.91 (0.38)	104.28 (0.89)	<0.0001
SBP [mean (SD)]	121.41 (0.31)	119.99 (0.32)	131.09 (0.69)	<0.0001
DBP [mean (SD)]	70.15 (0.25)	70.35 (0.27)	68.79 (0.51)	0.01
Hypertension (%)				<0.0001
No	4083 (59.4)	3757 (93.34)	326 (6.66)	
Yes	2791 (40.6)	2020 (76.86)	771 (23.14)	
DM (%)				<0.0001
No	4299 (62.54)	3893 (92.15)	406 (7.85)	
preDM	1206 (17.54)	987 (84.70)	219 (15.30)	
DM	1369 (19.92)	897 (68.08)	472 (31.92)	
Glu	5.91 (0.03)	5.79 (0.03)	6.72 (0.09)	<0.0001
Alt	24.81 (0.24)	24.90 (0.26)	24.20 (0.78)	0.41
Ast	24.71 (0.24)	24.51 (0.23)	26.12 (0.96)	0.11
Cr	76.84 (0.36)	74.39 (0.30)	93.58 (2.28)	<0.0001
UA	325.05 (1.48)	320.11 (1.47)	358.74 (3.19)	<0.0001
TG	1.26 (0.02)	1.24 (0.02)	1.39 (0.04)	<0.001
TC	4.89 (0.02)	4.89 (0.02)	4.90 (0.04)	0.78
HDL-C	1.41 (0.01)	1.41 (0.01)	1.43 (0.02)	0.32
LDL-C	2.91 (0.02)	2.92 (0.02)	2.84 (0.04)	0.06
eGFR	96.16 (0.47)	98.91 (0.45)	77.37 (1.33)	<0.0001
UACR	27.96 (2.49)	7.74 (0.09)	166.06 (18.76)	<0.0001
CDAI	0.69 (0.07)	0.78 (0.08)	0.06 (0.12)	<0.0001

Data are presented as frequencies (percentages) or mean (SD). CDAI, composite dietary antioxidant index; PIR, poverty income ratio; BMI, the body-mass index is determined as follows: the weight in kilograms (kgs)/height in square meters (m^2); WC, circumference waist; SBP, systolic blood pressure; DBP, diastolic blood pressure; DM, diabetes mellitus; preDM, prediabetes; Glu, blood glucose; ALT, alanine aminotransferase; AST, aspartate aminotransferase; Cr, serum creatinine; UA, serum uric acid; TG, triglycerides; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; eGFR, estimated glomerular filtration; UACR, urinary albumin/creatinine ratio; and CKD, chronic kidney disease.

covariates. Participants with a higher CDAI had a lower risk of developing CKD. This association was significant both in Model 1 [odds ratio (OR), −0.0048; 95% confidence interval [CI], (−0.0065, −0.0030); $P < 0.0001$], Model 2 [OR, −0.0037; 95% CI, (−0.0054, −0.0019); $P < 0.001$] and Model 3 [OR, −0.0017; 95% CI, (−0.0033, −0.0001), $P = 0.0399$]. For sensitivity analysis, we converted the CDAI from a continuous variable to a quadratic categorical variable for trend testing and obtained consistent findings (Table 3).

3.3 Subgroup analysis

In the subgroup analysis (Fig. 2), this positive association remained significant in patients older than or equal to 60 years, with a PIR greater than or equal to 1.8 and a BMI between 25 and 30 and in those with a history of hypertension and DM. After stratified gender, age, PIR, BMI, and the history of Hypertension and DM, the interaction test were all observed between the CDAI and CKD (P for interaction <0.05).

Table 2 Univariate analysis of the patients with CKD and non-CKD participants

Characteristic	B	95% CI	P value
Age [mean (SD)]	0.01	(0.00, 0.01)	<0.0001
Gender (%)			
Male	Reference	Reference	Reference
Female	0.03	(0.02, 0.05)	<0.001
Race/ethnicity (%)			
Non-Hispanic white	Reference	Reference	Reference
Non-Hispanic black	0.02	(0.00, 0.05)	0.08
Mexican American	0.01	(−0.04, 0.02)	0.51
Other race	0.02	(−0.04, 0.01)	0.12
Education (%)			
Less than high school	Reference	Reference	Reference
High school or equivalent	0.02	(−0.06, 0.01)	0.22
More than high school	0.08	(−0.11, −0.05)	<0.0001
PIR [mean (SD)]	0.01	(−0.02, −0.01)	<0.0001
Alcohol status (%)			
Never	Reference	Reference	Reference
Former	0.06	(0.01, 0.10)	0.01
Mild	0.04	(−0.08, 0.00)	0.04
Moderate	0.07	(−0.11, −0.03)	0.002
Heavy	0.07	(−0.11, −0.03)	<0.001
Smoking status (%)			
Never	Reference	Reference	Reference
Former	0.06	(0.04, 0.09)	<0.0001
Now	0.01	(−0.02, 0.03)	0.64
BMI [mean (SD)]	0	(0.00, 0.01)	<0.001
WC [mean (SD)]	0	(0.00, 0.00)	<0.0001
SBP [mean (SD)]	0	(0.00, 0.01)	<0.0001
DBP [mean (SD)]	0	(0.00, 0.00)	0.004
Hypertension (%)			
No	Reference	Reference	Reference
Yes	0.16	(0.14, 0.19)	<0.0001
DM (%)			
No	Reference	Reference	Reference
preDM	0.07	(0.05, 0.10)	<0.0001
DM	0.24	(0.21, 0.27)	<0.0001
Glu	0.04	(0.03, 0.05)	<0.0001
ALT	0	(0.00, 0.00)	0.42
AST	0	(0.00, 0.00)	0.13
Cr	0	(0.00, 0.00)	<0.0001
UA	0	(0.00, 0.00)	<0.0001
TG	0.03	(0.02, 0.05)	<0.001
TC	0	(−0.01, 0.01)	0.78
HDL-C	0.01	(−0.01, 0.04)	0.31
LDL-C	−0.01	(−0.02, 0.00)	0.06
eGFR	−0.01	(−0.01, 0.00)	<0.0001
UACR	0	(0.00, 0.00)	<0.0001
CDAI	0	(−0.01, 0.00)	<0.0001

Data are presented as frequencies (percentages) or mean (SD); OR, odds ratio; 95% CI, 95% confidence interval; CDAI, composite dietary antioxidant index; PIR, poverty income ratio; BMI, body-mass index is determined as follows: the weight in kilograms (kgs)/height in square meters (m^2); WC, circumference waist; SBP, systolic blood pressure; DBP, diastolic blood pressure; DM, diabetes mellitus; preDM, prediabetes; Glu, blood glucose; ALT, alanine aminotransferase; AST, aspartate aminotransferase; Cr, serum creatinine; UA, serum uric acid; TG, triglycerides; TC, total cholesterol; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; eGFR, estimated glomerular filtration; UACR, urinary albumin/creatinine ratio; and CKD, chronic kidney disease.

Table 3 Association of the composite dietary antioxidant index and CKD

	OR (95% CI), P value		
	Model 1	Model 2	Model 3
Continuous			
	−0.0048 (−0.0065, −0.0030)	−0.0037 (−0.0054, −0.0019)	−0.0017 (−0.0033, −0.0001)
	<0.0001	<0.001	0.0399
Categories			
Q1	Reference	Reference	Reference
Q2	−0.0257 (−0.0533, 0.0018)	−0.0315 (−0.0599, −0.0031)	−0.0635 (−0.0841, −0.0430)
Q3	−0.0293 (−0.0555, −0.0031)	−0.027 (−0.0562, 0.0022)	−0.0522 (−0.0730, −0.0314)
Q4	−0.0155 (−0.0417, 0.0107)	−0.0052 (−0.0346, 0.0242)	−0.0305 (−0.0491, −0.0120)
P for trend	<0.0001	<0.0001	0.0094

Model 1: no covariates were adjusted. Model 2: age, gender, race and education were adjusted. Model 3: age, gender, race, education, alcohol status, smoking status; OR, odds ratio; 95% CI, 95% confidence interval; BMI, body-mass index is determined as follows: the weight in kilograms (kgs)/height in square meters (m^2); WC, circumference waist; PIR, poverty income ratio; SBP, systolic blood pressure; Glu, blood glucose; UA, serum uric acid; TG, triglycerides; eGFR, estimated glomerular filtration; UACR, urinary albumin/creatinine ratio; and hypertension and DM (diabetes mellitus) were adjusted.

4. Discussion

In this study, we examined the relationship between the CDAI and the risk of developing CKD. After the adjustment for multiple covariates in the adult population based on the NHANES data, we found a negative association between the CDAI and CKD, suggesting that the CDAI protects against the development of CKD. There was a clear trend toward a lower CKD risk with a higher CDAI.

Oxidative stress is an imbalance between the production of antioxidants and pro-oxidants, which subsequently damages the tissues and organs. The accumulation of reactive oxygen species (ROS) can lead to the oxidation of DNA, proteins, carbohydrates, and lipids; apoptosis; and organ dysfunction.[20] As an external factor, diet regulates the plasma redox status and protects against ROS and reactive nitrogen species. To maintain a steady biological redox state, antioxidants may scavenge oxidants, thus preventing oxidative stress.[21] The exogenous intake of antioxidants prevents inflammation, atherosclerosis, insulin resistance, and oxidative stress in CKD and dialysis patients.[22–24]

Several clinical studies have examined the association between specific antioxidant micronutrients and CKD; however, the findings have been inconsistent. Some studies have suggested a negative correlation between the intake of vitamins A, C, and E, carotenoids, selenium, zinc and CKD,[25–28] whereas others showed no significant relationship.[29,30] Previous clinical studies have focused primarily on the effects of individual nutrients on CKD. However, considering the natural combination of nutrients in food, assessing overall dietary antioxidant intake can provide a more comprehensive understanding. The CDAI is a measure of total antioxidant levels in the diet and has been widely used in many studies. Previous studies demonstrated that high CDAI
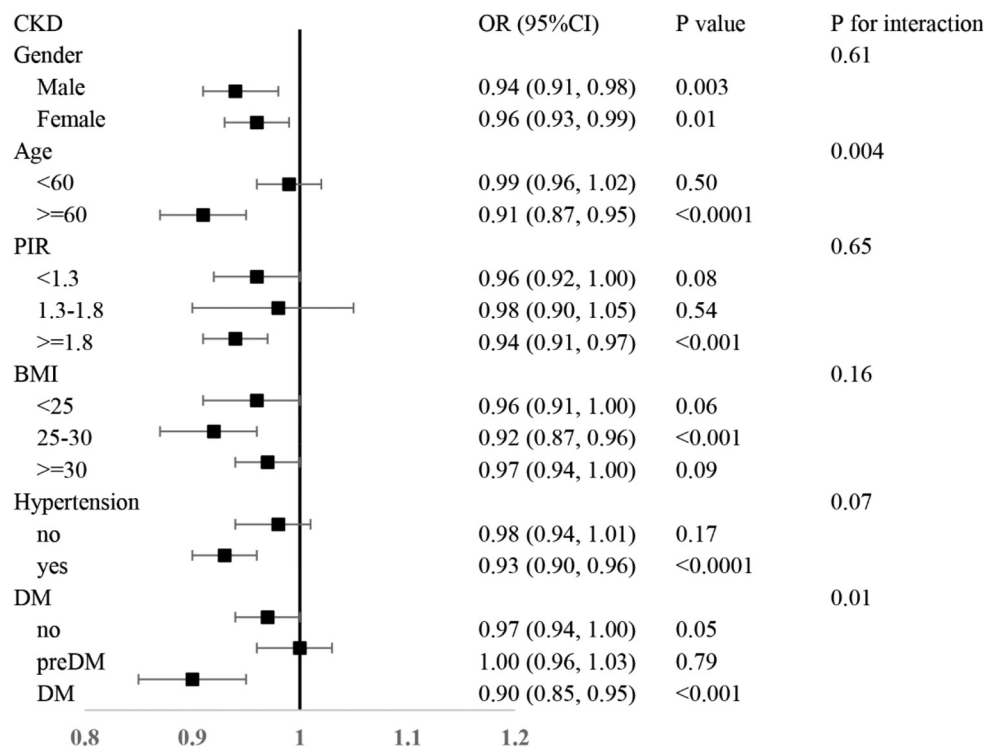
Fig. 2 Subgroup analysis for the association between the CDAI and CKD.

levels reduce the levels of inflammatory factors and lower the risk of various diseases, such as lung cancer, non-alcoholic fatty liver disease, and DM.[3,8,31–33] However, existing evidence of the association between higher dietary antioxidant intake and CKD is limited. This study aimed to address this research gap and provide evidence that adequate antioxidant intake may reduce the incidence of CKD.

In accordance with the Strengthening the Reporting of Observational Studies in Epidemiology statement, we conducted a subgroup analysis to enhance the utilization of data to reveal the underlying truth. In the subgroup analysis, we observed a significant interaction between dietary antioxidant indicators and the predefined risk factors for CKD. The CDAI showed a strong negative correlation with elderly patients and patients with DM complications. These results are consistent with those of previous studies.[6,8] Although the specific reasons for this finding are unclear, it may be because these individuals have higher levels of oxidative stress, and exogenous antioxidant intake appears to be more protective in those with higher innate or acquired ROS-levels.[34] Our results suggest that people at high risk of CKD may benefit more from overall dietary antioxidant intake.

There are several limitations to this study. First, due to its retrospective design, it was unable to construct or confirm any causal inferences. Second, despite the adjustment for potential confounding factors, residual confounding factors may still exist, that may affect the relationship between the CDAI and CKD. Third, as the population of this study was American and did not include special populations such as minors, we were

unable to analyze special populations or other ethnicities because of the limited sample size. Further studies are required to determine whether the benefits of dietary antioxidants can be extended to other populations.

In summary, this cross-sectional study based on four cycles (2011–2018) of the NHANES detected a negative correlation between the CDAI and CKD in American adults after adjustment for potential confounding factors. This study provides a new way to explore the factors affecting dietary interventions to reduce the incidence of CKD. In the future, more randomized controlled trials or cohort studies are urgently needed to confirm this finding and provide more accurate and effective prevention and treatment options for CKD.

Abbreviations

ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
BMI	Body mass index
CDAI	Composite dietary antioxidant index
CKD	Chronic kidney disease
Cr	Serum creatinine
DM	Diabetes
DBP	Diastolic blood pressure
eGFR	Estimated glomerular filtration
Glu	Blood glucose
HDL-C	High density lipoprotein cholesterol
LDL-C	Low density lipoprotein cholesterol

NHANES	National Health and Nutrition Examination Survey
OR	Odds ratio
PIR	Poverty income ratio
ROS	Reactive oxygen species
SBP	Systolic blood pressure
TC	Total cholesterol
TG	Triglycerides
UA	Uric acid
UACR	Urinary albumin/creatinine ratio
WC	Waist circumference
95% CI	95% confidence interval

Author contributions

Min Wang and Qiu-Lin Fan designed the research. Min Wang and Zhao-hui Huang performed the research. Min Wang, Yong-hong Zhu and Ping He analyzed the data. Min Wang wrote the paper. All authors contributed to the article and approved the final manuscript.

Data availability

The original contribution presented in this study is included in the article. The dataset was based on the NHANES, which is publicly available and could be found below: https://www.cdc.gov/nchs/nhanes/.

Conflicts of interest

There are no conflicts to declare.

Acknowledgements

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