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Composite dietary antioxidant index and obesity among U.S. adults in NHANES 2007–2018

Zhaoxiang Wang^{1,3}, Qianqian Wang^{1,3}, Fengyan Tang¹ & Shao Zhong^{1,2}✉

Antioxidant-rich diets serve as protective factors in preventing obesity. The composite dietary antioxidant index (CDAI) represents a novel, comprehensive metric for assessing the antioxidant capacity of diets. Our objective is to investigate the relationship between the CDAI and obesity prevalence among adults in the United States. Dietary and anthropometric information about adults aged 20 years and older were obtained from the National Health and Nutrition Examination Survey (NHANES) 2007–2018. The CDAI was derived from six dietary antioxidants. Obesity is defined as a body mass index (BMI, kg/m^2) $\geq 30 \text{ kg}/\text{m}^2$, and abdominal obesity as a waist circumference (WC, cm) $\geq 102 \text{ cm}$ for men and $\geq 88 \text{ cm}$ for women. The relationship between CDAI and obesity, including abdominal obesity, was analyzed using logistic regression and subgroup analyses. A total of 25,553 participants were analyzed. With higher tertiles of the CDAI, both obesity (41.28% vs. 38.62% vs. 35.09%, $P < 0.001$) and abdominal obesity (63.75% vs. 59.54% vs. 52.09%, $P < 0.001$) prevalence notably declined. Adjusting for multiple confounders, the CDAI was found to be independently linked to obesity (OR = 0.980, 95%CI = 0.971–0.989, $P < 0.001$) and abdominal obesity (OR = 0.972, 95%CI = 0.963–0.982, $P < 0.001$) risks. Subgroup analyses revealed a stronger relationship between CDAI and obesity in non-hypertensive individuals and a more significant association with abdominal obesity in women and those without hypertension. Our findings reveal a negative relationship between CDAI levels and both general and abdominal obesity. Additional extensive research is necessary to investigate CDAI's contribution to obesity.

Keywords NHANES, Obesity, Abdominal obesity, CDAI, Antioxidant

Obesity and its global prevalence are increasingly becoming a significant worldwide risk¹. Extensive evidence to date shows that excess body fat is associated with increased markers of oxidative stress². Oxidative stress plays a crucial role in the onset and progression of obesity and related diseases³. Under physiological conditions, antioxidants delicately control reactive oxygen species levels, sourced both internally and externally⁴. Insufficient antioxidant consumption can lead to an excess of reactive oxygen species, causing oxidative stress and subsequently exacerbating the inflammatory state associated with obesity^{3,5,6}. The CDAI stands as a robust and credible nutritional instrument designed to evaluate the comprehensive antioxidant profile of one's diet. This index aggregates the effects of six key dietary antioxidants: vitamins A, C, and E, alongside carotenoids, selenium, and zinc, offering a succinct measure of dietary antioxidant capacity^{7–9}. Prior research has elucidated an inverse correlation between CDAI levels and the risk of various diseases, such as hypertension, diabetes, cancer, osteoporosis, and depression^{10–14}. However, whether the CDAI can effectively identify high-risk populations for obesity remains uncertain. Furthermore, as a novel dietary index, it is yet to be determined if it could assess the dietary antioxidant capacity of obese individuals.

Utilizing data from the NHANES database, this study aims to meticulously explore the potential link between CDAI and both general and abdominal obesity risk through a detailed cross-sectional analysis.

Materials and methods

Data source

This study utilized data from the NHANES, an extensive survey executed by the National Center for Health Statistics within the Centers for Disease Control and Prevention. NHANES implemented a meticulously designed, randomized, stratified, multi-stage survey approach to achieve a representative sample of the national

¹Department of Endocrinology, Affiliated Kunshan Hospital of Jiangsu University, Kunshan 215300, Jiangsu, China.

²Department of Clinical Nutrition, Affiliated Kunshan Hospital of Jiangsu University, Kunshan 215300, Jiangsu, China. ³Zhaoxiang Wang and Qianqian Wang contributed equally to this work. ✉email: drzhong@163.com

population. Participants were subjected to comprehensive physical examinations, alongside health and nutrition questionnaires, and laboratory evaluations^{15,16}. The study protocol of NHANES was sanctioned by the Ethics Review Board of the National Center for Health Statistics (<https://www.cdc.gov/nchs/nhanes/irba98.htm>). All methods were performed in accordance with the Declaration of Helsinki. The participants provided their written informed consent to participate in this study. In-depth methodology and data from this investigation are further available at <https://www.cdc.gov/nchs/nhanes/>. This study compiled data from the NHANES cycles spanning 2007–2008, 2009–2010, 2011–2012, 2013–2014, 2015–2016, and 2017–2018, including 59,842 participants. We excluded those who were under 20 years old, pregnant, or did not provide complete dietary questionnaires or anthropometric data (including BMI and WC), resulting in a total of 25,553 eligible participants.

Exposure and outcome definitions

In the NHANES database, the intake of food and nutrients for each participant was documented through a 24-hour dietary recall interview. The initial recall was carried out face-to-face, followed by a second recall conducted over telephone 3 to 10 days later. The CDAI, representing each participant's total dietary antioxidant intake, was calculated using the food frequency questionnaire (FFQ) data^{7,8}. Based on the questionnaire interview, we determined the antioxidant components, including vitamins A, C, E, carotenoids, zinc, and selenium. Dietary antioxidant carotenoids were determined by adding the intake of α -carotene, β -carotene, β -cryptoxanthin, lutein, zeaxanthin, and lycopene¹⁷. The normalization of these six antioxidants was conducted by subtracting the mean and dividing by the standard deviation. Following this, the CDAI was calculated based on the sum of these normalized values. The detailed calculation formula is as follows: $CDAI := \sum_{i=1}^6 \left(\frac{\text{Individual Intake} - \text{Mean}}{\text{standard deviation}} \right)$. On other hand, obesity is defined as a $BMI \geq 30 \text{ kg/m}^2$, and particularly abdominal obesity as a $WC \geq 102 \text{ cm}$ for men and $\geq 88 \text{ cm}$ for women.

Covariate definitions

Demographic information such as age (years), gender (female/male), and race (Mexican American/Non-Hispanic Black/Non-Hispanic White/Other Hispanic/Other Race), along with a range of potential covariates including annual household income (below or above \$20,000), education level (above high school or not), physical activity (moderate or not), smoking status (smoker/non-smoker), hypertension (yes/no), diabetes (yes/no), cardiovascular disease (yes/no), glycohemoglobin (HbA1c) levels, systolic and diastolic blood pressure (SBP and DBP, in mmHg), and total energy intake were gathered. Smokers were categorized as either current or past smokers. Self-reported cases of diabetes, hypertension, and cardiovascular disease were also recorded. Cardiovascular disease was identified through self-reports of heart attack, stroke, congestive heart failure, coronary artery disease, or angina. Detailed methods for measuring all variables are available in the NHANES database.

Statistical analysis

The statistical analysis followed the guidelines of the Centers for Disease Control and Prevention, employing a complex multistage cluster survey design and incorporating weights from six cycles. Continuous variables were described as mean \pm standard errors (SE), and categorical variables as percentages (SE). Differences in continuous and categorical variables between groups were assessed using the weighted Student's t-test and chi-squared test, respectively. The relationship between CDAI (continuous/quartile) and obesity, abdominal obesity, BMI, and WC was examined through logistic and linear regression models. Three models were applied: Model 1 without adjustments; Model 2 adjusted for age, gender, and race; and Model 3 adjusted for age, gender, race, annual household income, education level, physical activity, smokers, hypertension, diabetes, cardiovascular disease, HbA1c, SBP, DBP, and total energy intake. Subgroup analyses were conducted based on age (<60/ \geq 60 years), gender (female/male), race (white/non-white), hypertension (yes/no), diabetes (yes/no), and cardiovascular disease (yes/no). Additionally, the relationship between CDAI and obesity, abdominal obesity, BMI, and WC was further explored based on the Restricted Cubic Spline (RCS) analysis. All statistical analyses were carried out using the Empower software (<http://www.empowerstats.com>) and the R software (<http://www.R-project.org>), with a two-side P value <0.05 considered statistically significant.

Results

Baseline characteristics of study population

This study ultimately encompassed 25,553 participants who met the inclusion criteria (Table 1). The mean age was 48.07 years, with males making up 48.08% of the participants. For lifestyles and risk factors associated with obesity, the prevalence rates were observed as follows: 13.18% among individuals with an annual household income below \$20,000; 62.59% in those with an education level above high school; 42.32% for those engaging in moderate physical activity; 44.06% among smokers; 32.26% for individuals with hypertension; 9.78% for those diagnosed with diabetes; and 8.38% for individuals with cardiovascular disease. The population's average BMI was 29.11 kg/m^2 , WC was 99.85 cm , HbA1c was 5.63%, SBP was 121.79 mmHg , DBP was 70.55 mmHg , total energy was 2092.82 kcal, carotenoids was 9774.35 mcg, vitamin A was 648.88 mg, vitamin E was 9.50 mg, vitamin C was 81.31 mg, selenium was 113.84 mcg, zinc was 11.43 mg, and CDAI was 0.91. Among participants, 38.11% were classified as obesity, and 58.05% exhibited abdominal obesity.

Clinical features of the participants according to the tertiles of CDAI

Participants were divided into three groups according to their CDAI levels (Table 2). As CDAI levels increased from tertile 1 to tertile 3, there was a significant decrease in age, the proportion of female, annual household income below \$20,000, smokers, and those with hypertension, diabetes, cardiovascular disease, BMI, WC, HbA1c, and SBP ($P < 0.05$). Conversely, education level above high school, moderate physical activity, DBP,

Baseline characteristics	Overall (N = 25,553)
Age (years)	48.07 ± 0.24
Male gender, % (SE)	48.08 (0.34)
Race, % (SE)	
Mexican American	7.96 (0.71)
Non-Hispanic Black	10.91 (0.72)
Non-Hispanic White	68.35 (1.38)
Other Hispanic	5.58 (0.48)
Other Races	7.20 (0.37)
Annual household income (below \$20,000), % (SE)	13.18 (0.52)
Education level (above high school), % (SE)	62.59 (0.93)
Moderate physical activity, % (SE)	42.32 (0.63)
Smokers, % (SE)	44.06 (0.65)
Hypertension, % (SE)	32.26 (0.56)
Diabetes, % (SE)	9.78 (0.62)
Cardiovascular disease, % (SE)	8.38 (0.25)
BMI (kg/m ²)	29.11 ± 0.09
WC (cm)	99.85 ± 0.23
HbA1c (%)	5.63 ± 0.01
SBP (mmHg)	121.79 ± 0.22
DBP (mmHg)	70.55 ± 0.22
Total energy (kcal)	2092.82 ± 8.48
Carotenoids (mcg)	9774.35 ± 143.26
Vitamin A (mg)	648.88 ± 7.28
Vitamin E (mg)	9.50 ± 0.10
Vitamin C (mg)	81.31 ± 0.94
Selenium (mcg)	113.84 ± 0.52
Zinc (mg)	11.43 ± 0.07
CDAI	0.91 ± 0.06
Obesity, % (SE)	38.11 (0.60)
Abdominal obesity, % (SE)	58.05 (0.68)

Table 1. The baseline characteristics of study population, weighted. BMI, body mass index; WC, waist circumference; HbA1c, glycohemoglobin; SBP, systolic blood pressure; DBP, diastolic blood pressure; CDAI, composite dietary antioxidant index.

total energy, carotenoids, vitamin A, vitamin E, vitamin C, selenium, and zinc escalated, along with significant race distribution observed ($P < 0.001$). Notably, the prevalence of general obesity (41.28% vs. 38.62 vs. 35.09%, $P < 0.001$) and abdominal obesity (63.75% vs. 59.54 vs. 52.09%, $P < 0.001$) decreased markedly. Additionally, compared to the general population, individuals with overall obesity and abdominal obesity exhibit lower levels of CDAI ($P < 0.001$) (**Attachment 1**).

Associations between CDAI and general obesity, abdominal obesity, BMI, and WC

Our research indicates a significant inverse relationship between CDAI and the risk of obesity. This negative association persists across various models: Model 1 ($OR = 0.971$, 95%CI = 0.965–0.977, $P < 0.001$), Model 2 ($OR = 0.983$, 95%CI = 0.977–0.990, $P < 0.001$), and Model 3 ($OR = 0.980$, 95%CI = 0.971–0.989, $P < 0.001$). Specifically, individuals in the highest tertile of CDAI were found to have a 14.0% reduced risk of obesity in the fully adjusted model, compared to those in the lowest tertile ($OR = 0.860$, 95%CI = 0.785–0.942, $P = 0.001$) (Table 3). Moreover, a significant inverse correlation between CDAI and abdominal obesity was identified after adjusting for potential confounders ($OR = 0.972$, 95%CI = 0.963–0.982, $P < 0.001$). When analyzing CDAI into tertiles, it was evident that individuals in higher CDAI tertile presented a lower prevalence of abdominal obesity than those in the lowest tertile ($OR = 0.818$, 95%CI = 0.745–0.898, $P < 0.001$) (Table 3). Furthermore, linear regression analyses, with BMI and WC as dependent variables, also revealed a significant negative relationship between CDAI and both BMI ($\beta = -0.057$, 95%CI = -0.083–0.031, $P < 0.001$) and WC ($\beta = -0.185$, 95%CI = -0.246–0.124, $P < 0.001$) (Table 4). Due to inappropriate dietary habits commonly followed by obese individuals, we further assessed whether general and abdominal obesity populations, compared to normal populations, have a higher risk of exhibiting low CDAI levels (Table 5). The results indicate that individuals with general and abdominal obesity indeed have a higher risk of low CDAI levels ($P < 0.001$).

	Tertile 1 (-8.32, -1.58)	Tertile 2 (-1.58,1.48)	Tertile 3 (1.48, 44.56)	P value
Age (years)	48.40 \pm 0.29	48.38 \pm 0.32	47.52 \pm 0.32	0.011
Male gender, % (SE)	32.66 (0.51)	45.42 (0.65)	62.90 (0.67)	< 0.001
Race, % (SE)				< 0.001
Mexican American	8.31 (0.86)	7.77 (0.68)	7.85 (0.73)	
Non-Hispanic Black	14.45 (1.04)	10.00 (0.71)	8.94 (0.61)	
Non-Hispanic White	63.95 (1.75)	69.37 (1.46)	70.90 (1.28)	
Other Hispanic	6.45 (0.65)	5.39 (0.49)	5.06 (0.43)	
Other Races	6.84 (0.43)	7.47 (0.46)	7.25 (0.45)	
Annual household income (below \$20,000), % (SE)	18.39 (0.70)	11.76 (0.60)	10.38 (0.48)	< 0.001
Education level (above high school), % (SE)	50.91 (1.09)	64.38 (1.00)	70.22 (0.98)	< 0.001
Moderate physical activity, % (SE)	39.36 (0.74)	42.08 (0.84)	44.91 (1.05)	< 0.001
Smokers, % (SE)	47.46 (0.95)	43.16 (0.86)	42.20 (0.84)	< 0.001
Hypertension, % (SE)	34.84 (0.72)	32.24 (0.79)	30.22 (0.87)	< 0.001
Diabetes, % (SE)	11.22 (0.47)	10.05 (0.42)	8.38 (0.40)	< 0.001
Cardiovascular disease, % (SE)	9.89 (0.46)	8.50 (0.33)	7.06 (0.32)	< 0.001
BMI (kg/m ²)	29.46 \pm 0.11	29.21 \pm 0.12	28.75 \pm 0.12	< 0.001
WC (cm)	100.15 \pm 0.26	100.12 \pm 0.32	99.28 \pm 0.30	< 0.001
HbA1c (%)	5.67 \pm 0.01	5.64 \pm 0.01	5.60 \pm 0.01	0.005
SBP (mmHg)	122.69 \pm 0.32	121.75 \pm 0.29	121.12 \pm 0.25	< 0.001
DBP (mmHg)	69.78 \pm 0.28	70.73 \pm 0.24	71.00 \pm 0.23	< 0.001
Total energy (kcal)	1484.10 \pm 8.28	2025.45 \pm 10.32	2642.22 \pm 13.85	< 0.001
Carotenoids (mcg)	4004.56 \pm 53.02	7992.33 \pm 82.20	16058.44 \pm 262.56	< 0.001
Vitamin A (mg)	320.19 \pm 3.51	560.80 \pm 3.79	994.21 \pm 14.23	< 0.001
Vitamin E (mg)	4.83 \pm 0.03	8.06 \pm 0.06	14.58 \pm 0.16	< 0.001
Vitamin C (mg)	39.41 \pm 0.57	70.48 \pm 0.84	124.96 \pm 1.59	< 0.001
Selenium (mcg)	74.55 \pm 0.37	108.71 \pm 0.50	150.05 \pm 1.00	< 0.001
Zinc (mg)	7.01 \pm 0.04	10.62 \pm 0.06	15.74 \pm 0.12	< 0.001
Obesity, % (SE)	41.28 (0.80)	38.62 (0.82)	35.09 (0.81)	< 0.001
Abdominal obesity, % (SE)	63.75 (0.71)	59.54 (0.92)	52.09 (1.04)	< 0.001

Table 2. The baseline characteristics based on CDAI tertiles, weighted.

Subgroup analyses

Subgroup analysis was conducted to assess the consistency of the CDAI-obesity and CDAI-abdominal obesity associations across various demographics (Figs. 1 and 2). The results show that age, gender, race, diabetes, and cardiovascular disease presence do not significantly modify the CDAI-obesity relationship (*P* for interaction > 0.05). However, a significant interaction between hypertension and the CDAI-obesity link was observed, with a stronger correlation in non-hypertensive individuals (*P* for interaction < 0.05). On other hand, these factors do not significantly influence the CDAI-abdominal obesity association (*P* for interaction < 0.05), except for a notable interaction between gender, hypertension, and the CDAI-abdominal obesity relationship, indicating a stronger correlation in non-hypertensive females compared to hypertensive males (*P* for interaction < 0.05). Lastly, the RCS results revealed a negative relationship between the CDAI and the risk of obesity and abdominal obesity, with no significant threshold effect detected (*P* for nonlinear = 0.466 and 0.403) (Fig. 3).

Discussion

Our population-based study examined the association of CDAI with obesity and abdominal obesity. The present study found that obesity was prevalent in approximately 38.11% among U.S. adults, while abdominal obesity was found in 58.05% of the adult population. This result is broadly consistent with previous estimates of the prevalence of obesity and abdominal obesity in the US population¹⁸. We observed an inverse relationship between CDAI and both general and abdominal obesity, suggesting that CDAI might function as a protective factor against the inflammatory conditions associated with obesity.

Oxidative stress plays a key role in obesity's pathophysiology by affecting mitochondrial function, altering inflammation mediators linked to adipocyte size and number, driving lipogenesis, encouraging preadipocyte differentiation into mature cells, and influencing hypothalamic neurons that regulate appetite and energy balance³. Dietary intake of vitamins A, C, and E, alongside carotenoids, selenium, and zinc, play crucial roles in modulating oxidative stress and inflammation. Vitamin A, alongside carotenoids, is pivotal in maintaining immune function, while also modulating adipogenesis, the process by which preadipocytes mature into adipocytes^{19,20}. Vitamin C, beyond its well-documented role in immune enhancement, contributes to the

	OR (95%CI), P value		
	Non-adjusted model 1	Adjusted model 2	Adjusted model 3
Obesity			
Continuous			
CDAI	0.971 (0.965, 0.977), < 0.001	0.983 (0.977, 0.990), < 0.001	0.980 (0.971, 0.989), < 0.001
Categories			
Tertile 1	Reference	Reference	Reference
Tertile 2	0.917 (0.862, 0.974), 0.005	0.984 (0.925, 1.048), 0.622	0.971 (0.900, 1.047), 0.442
Tertile 3	0.763 (0.718, 0.812), < 0.001	0.866 (0.812, 0.924), < 0.001	0.860 (0.785, 0.942), 0.001
P for trend	< 0.001	< 0.001	< 0.001
Abdominal obesity			
Continuous			
CDAI	0.945 (0.939, 0.951), < 0.001	0.977 (0.970, 0.983), < 0.001	0.972 (0.963, 0.982), < 0.001
Categories			
Tertile 1	Reference	Reference	Reference
Tertile 2	0.846 (0.795, 0.900), < 0.001	0.995 (0.931, 1.064), 0.891	0.983 (0.907, 1.064), 0.667
Tertile 3	0.591 (0.556, 0.629), < 0.001	0.828 (0.774, 0.885), < 0.001	0.818 (0.745, 0.898), < 0.001
P for trend	< 0.001	< 0.001	< 0.001

Table 3. The relationship between CDAI and obesity, abdominal obesity. OR: odds ratio. 95% CI: 95% confidence interval. Adjusted model 2: age, gender, and race were adjusted. Adjusted model 3: additionally adjusted for annual household income, education level, moderate physical activity, smokers, hypertension, diabetes, cardiovascular disease, HbA1c, SBP, DBP, total energy.

	β (95%CI), P value		
	Non-adjusted model 1	Adjusted model 2	Adjusted model 3
BMI			
Continuous			
CDAI	-0.095 (-0.114, -0.075), < 0.001	-0.054 (-0.074, -0.034), < 0.001	-0.057 (-0.083, -0.031), < 0.001
Categories			
Tertile 1	Reference	Reference	Reference
Tertile 2	-0.250 (-0.456, -0.044), 0.017	0.004 (-0.200, 0.208), 0.969	0.013 (-0.209, 0.236), 0.908
Tertile 3	-0.867 (-1.072, -0.661), < 0.001	-0.417 (-0.626, -0.209), < 0.001	-0.367 (-0.632, -0.102), 0.007
P for trend	< 0.001	< 0.001	< 0.001
WC			
Continuous			
CDAI	-0.123 (-0.170, -0.077), < 0.001	-0.162 (-0.208, -0.115), < 0.001	-0.185 (-0.246, -0.124), < 0.001
Categories			
Tertile 1	Reference	Reference	Reference
Tertile 2	0.030 (-0.464, 0.525), 0.904	-0.062 (-0.542, 0.417), 0.799	-0.075 (-0.597, 0.447), 0.778
Tertile 3	-0.840 (-1.334, -0.345), < 0.001	-1.236 (-1.728, -0.745), < 0.001	-1.135 (-1.756, -0.514), < 0.001
P for trend	< 0.001	< 0.001	< 0.001

Table 4. The relationship between CDAI and BMI, WC. 95% CI: 95% confidence interval. Adjusted model 2: age, gender, and race were adjusted. Adjusted model 3: additionally adjusted for annual household income, education level, moderate physical activity, smokers, hypertension, diabetes, cardiovascular disease, HbA1c, SBP, DBP, total energy.

synthesis of collagen and the lipid metabolism²¹. Vitamin E, recognized for its antioxidant capacity, is involved in preventing lipid peroxidation within cell membranes, thereby protecting cells from oxidative damage²². Selenium and zinc are trace elements essential for the proper functioning of various antioxidant enzymes. Selenium's role in the activity of glutathione peroxidase, an enzyme that reduces peroxides, highlights its significance in mitigating oxidative damage²³. Zinc, on the other hand, enhances the activity of antioxidant proteins and enzymes, including glutathione and catalase²⁴. Thus, it can be definitively stated that dietary antioxidants, through their bioactive compounds, generate a synergistic action that mitigates oxidative stress and delivers

	β (95%CI), P value		
	Non-adjusted model 1	Adjusted model 2	Adjusted model 3
CDAI			
BMI < 30 kg/m ²	Reference	Reference	Reference
BMI ≥ 30 kg/m ²	-0.513 (-0.621, -0.404), < 0.001	-0.282 (-0.388, -0.175), < 0.001	-0.193 (-0.287, -0.099), < 0.001
CDAI (female)			
WC < 88 cm	Reference	Reference	Reference
WC ≥ 88 cm	-0.603 (-0.739, -0.467), < 0.001	-0.491 (-0.633, -0.348), < 0.001	-0.357 (-0.482, -0.231), < 0.001
CDAI (male)			
WC < 102 cm	Reference	Reference	Reference
WC ≥ 102 cm	-0.538 (-0.705, -0.371), < 0.001	-0.401 (-0.573, -0.230), < 0.001	-0.253 (-0.403, -0.104), < 0.001

Table 5. The relationship between BMI, WC category and the risk of low CDAI levels. 95% CI: 95% confidence interval. Adjusted model 2: age, (gender), and race were adjusted. Adjusted model 3: additionally adjusted for annual household income, education level, moderate physical activity, smokers, hypertension, diabetes, cardiovascular disease, HbA1c, SBP, DBP, total energy.

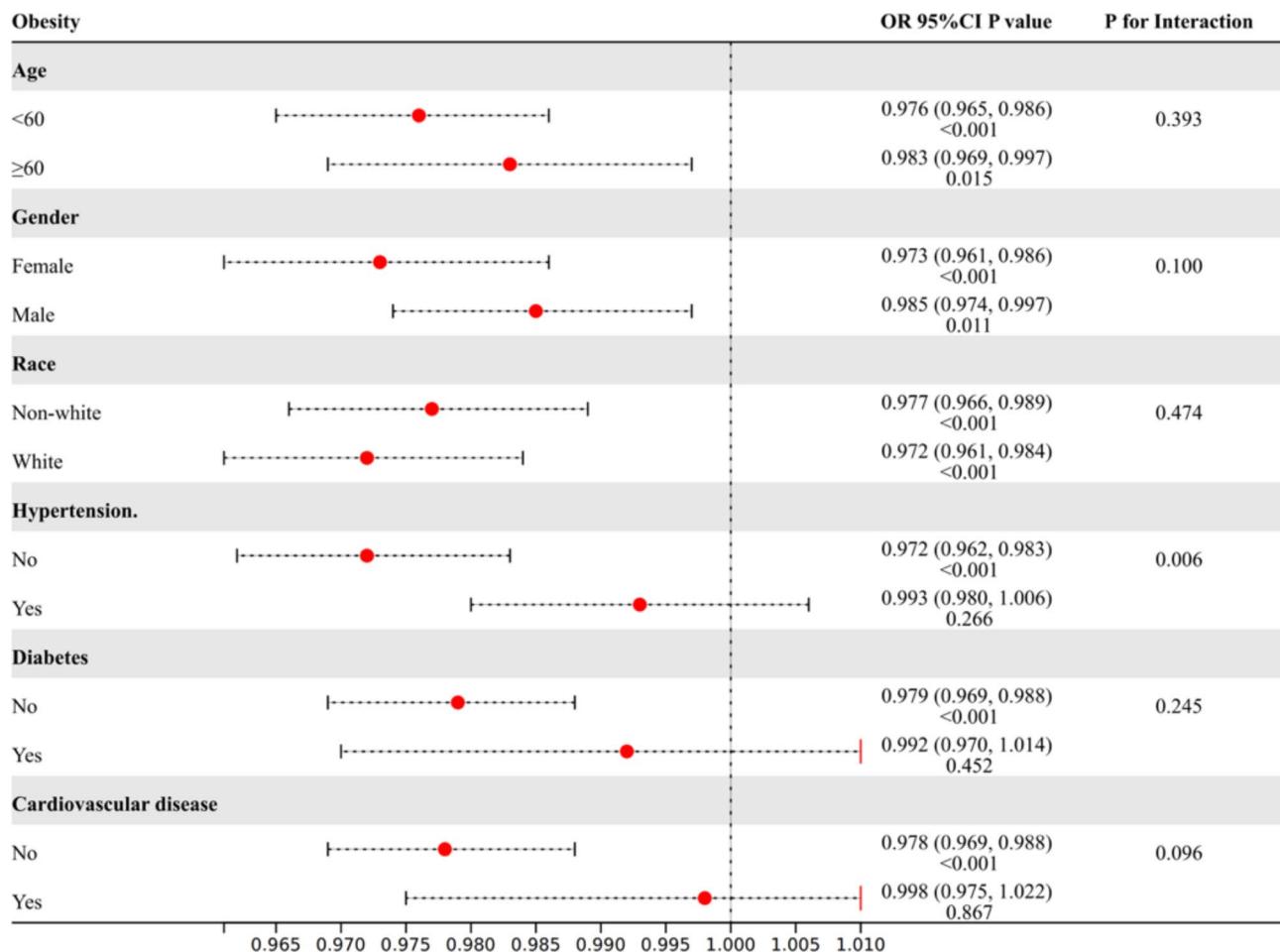


Fig. 1. Subgroup analyses between CDAI and obesity.

antioxidant advantages, consequently diminishing the risk of obesity^{11,25}. The CDAI, serving as an innovative antioxidant index, effectively encapsulates the cumulative impact of these dietary antioxidants, evaluating the genuine antioxidant efficacy from a clinical standpoint. Nevertheless, it is imperative to note that the precise molecular mechanisms remain elusive, necessitating further investigation.

This study leveraged a sample representative of the ethnic diversity found within the US adult population, however it's important to acknowledge its limitations. The cross-sectional approach restricts our capacity to establish causality between CDAI and the risk of obesity, further longitudinal studies and clinical trials are

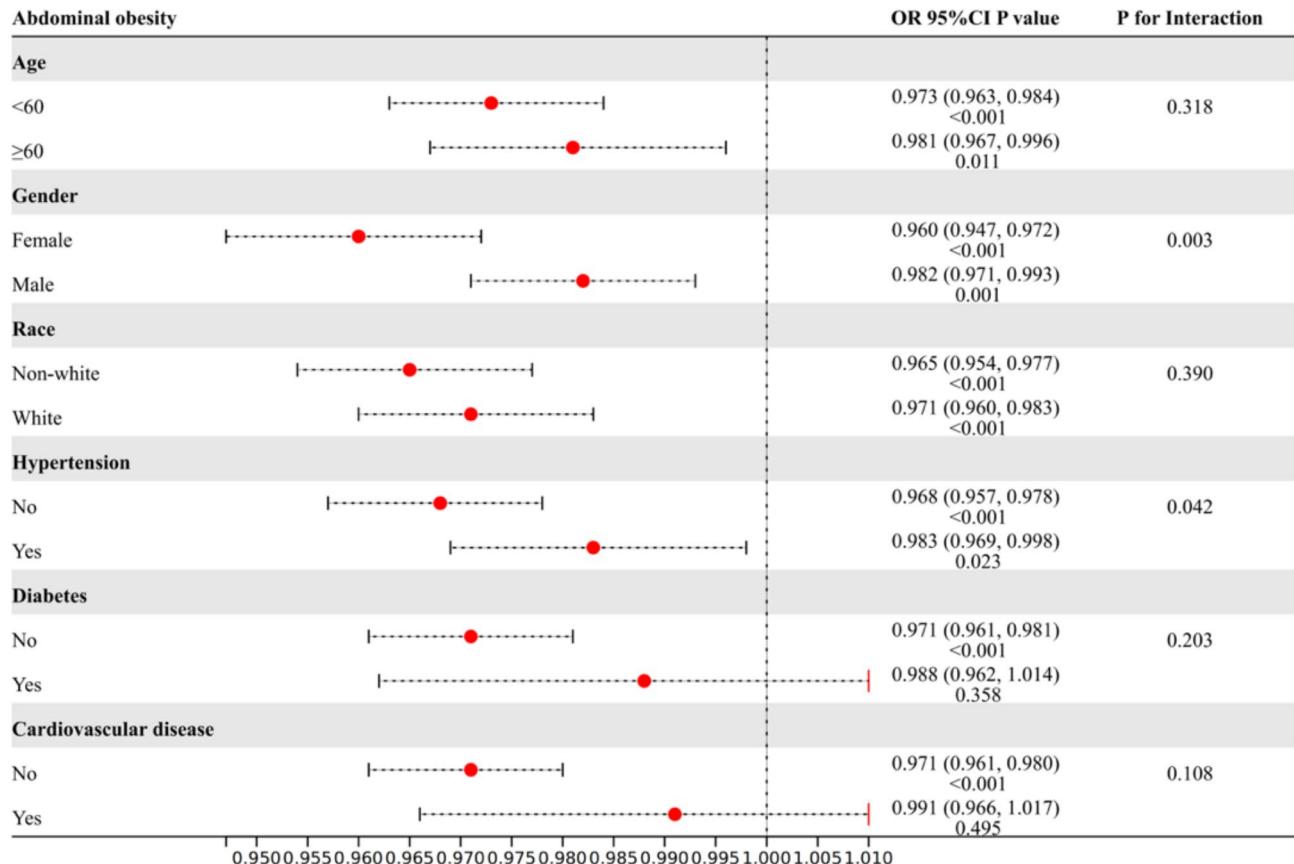


Fig. 2. Subgroup analyses between CDAI and abdominal obesity.

indispensable. Moreover, the exclusion of potential confounding factors such as metabolic syndrome and nonalcoholic fatty liver disease may have impacted our results. Additionally, although our study indicates a negative correlation between CDAI and obesity prevalence, future research is needed to further define the threshold for evaluating sufficient antioxidant properties in diets. Finally, considering this study's focus on the US adult population, extending its conclusion to other demographic groups requires more in-depth analysis.

Conclusion

An inverse relationship was observed between CDAI and both general and abdominal obesity. Maintaining high CDAI levels might act as a protective factor against obesity-related inflammatory conditions. However, further studies are required to validate our findings.

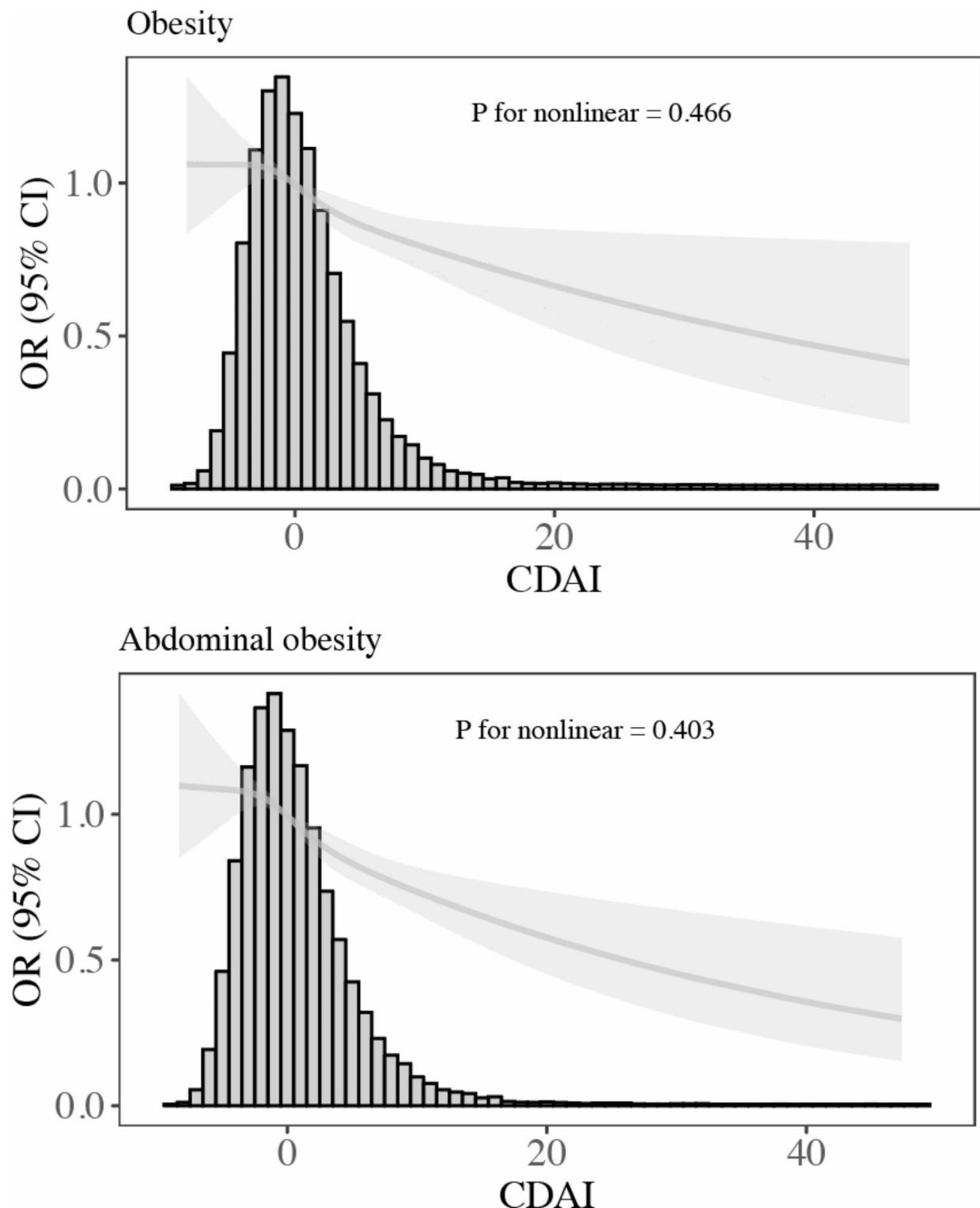


Fig. 3. The results of RCS analysis. (Adjusted for age, gender, race, annual household income, education level, moderate physical activity, smokers, hypertension, diabetes, cardiovascular disease, HbA1c, SBP, DBP, and total energy).

Data availability

The datasets generated and/or analyzed during the current study are available in the NHANES database (<https://www.cdc.gov/nchs/nhanes/>).

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Author contributions

Z.W. and Q.W. wrote the main manuscript text. F.T. prepared figures and tables. S.Z. reviewed the manuscript.

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Competing interests

The authors declare no competing interests.

Ethical approval

This study involving human participants were reviewed and approved by the Ethics Review Board of the National Center for Health Statistics (<https://www.cdc.gov/nchs/nhanes/irba98.htm>). All methods were performed in accordance with the Declaration of Helsinki.

Informed consent

The patients/participants provided their written informed consent to participate in this study.

Additional information

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Correspondence and requests for materials should be addressed to S.Z.

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