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Association between the composite dietary antioxidant index and erectile dysfunction in US men: a cross-sectional study

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Abstract

Background Erectile dysfunction (ED) is a common issue among adult males. The Composite Dietary Antioxidant Index (CDAI) reflects anti-inflammatory levels and has been linked to various diseases, but its relationship with ED is unclear.

Materials and methods This cross-sectional study utilised comprehensive data on clinical factors from the 2001–2004 National Health and Nutrition Examination Survey (NHANES). To investigate the link between variables and ED, we used multivariate regression analysis, univariate analysis, and subgroup analysis. The linear relationship between CDAI and ED was investigated by dose-response curve analysis. For sensitivity analysis, propensity score matching (PSM) was utilised to exclude the influence of potential confounders. Finally, we investigated the association between CDAI and ED using threshold effects analysis.

Results We included in our research a total of 2896 persons with data on CDAI from NHANES 2001–2004. Among these, 2,098 participants were thought to be free of ED, whereas 798 participants had ED. We found that compared to the ED group, men in the non-ED group had higher levels of CDAI ($p < 0.0001$ before PSM and $p = 0.0145$ after PSM). Additionally, after adjusting for covariates, it was found that an elevated CDAI was associated with a reduced incidence of ED [OR = 0.65($p = 0.001$) before PSM and OR = 0.62($p = 0.002$) after PSM]. Subgroup analysis indicated stronger associations in high-risk groups, and dose-response curves confirmed a linear negative correlation between CDAI and ED.

Conclusions This study revealed a negative linear relationship between CDAI and the incidence of ED. The CDAI can be used as an indicator for assessing ED risk and for ED prevention.

Keywords NHANES, Composite dietary antioxidant index (CDAI), Erectile dysfunction (ED), Propensity score matching (PSM), Exogenous antioxidant intake

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Introduction

For the past few years, an increasing number of men worldwide have been plagued by erectile dysfunction problems [1]. The inability to obtain or sustain an erection long enough for satisfying sexual activity for longer than three months is referred to as this condition [2]. Patients and their partners may have severe problems with their quality of life because of ED. For American men over 40, the prevalence of ED surpasses 40% [3]. By 2025, over 322 million men worldwide may experience erectile dysfunction, according to this report. As a result, the patients, their spouses, and the social health system bear an increasingly heavy weight.

A social psychological disorder with physiological foundations, ED has a complicated aetiology [4]. ED is linked to a wide range of risk factors, including age, obesity, smoking, diabetes, cardiovascular disease, hypertension, status of activity, and hypogonadism [5–8]. Reduced endothelial cell production of NO has detrimental effects on somatic smooth muscle, which in turn causes a reduction in blood flow and a decrease in smooth muscle relaxation [9]. According to recent studies, ED prevalence positively correlates with inflammatory indices [10, 11]. Inflammation influences endothelial cells and has a role in the pathophysiology of metabolic syndromes, all linked to ED [12, 13]. The primary line of treatment for ED patients to improve their sexual function and general health is thought to be lifestyle change [14]. Diet as a part of the lifestyle, it has become increasingly popular to prevent ED using modifiable factors like nutrition to lower inflammation and safeguard endothelial function [15].

Based on a variety of dietary vitamins and minerals having antioxidant characteristics, such as carotenoids, the minerals zinc, and selenium, and vitamins E, C, and A, the composite dietary antioxidant index (CDAI) is established. The dietary total antioxidant capacity (TAC) of a person is evaluated using this summary score [16, 17]. According to recent publications, CDAI is connected to biomarkers of inflammation [18, 19].

However, the relationship between CDAI and ED remains unclear. This cross-sectional study uses data from the National Health and Nutrition Examination Survey (NHANES) 2001–2004 to examine any potential correlation between the prevalence of ED and CDAI. We also investigated the possible mechanisms and hope that these results will serve as a basis for exogenous antioxidant diets for ED prevention.

Materials and methods

Source of data

The NHANES is a major program conducted by the National Centre for Health Statistics (NCHS). Data on the general health of the US population that is nationally representative has been gathered. It makes use of cross-sectional surveys, which since 1999. It collects data from about 5000 people yearly by sophisticated, multistage, stratified probability sampling technique [20]. Furthermore, it has a two-year survey cycle. Information was gathered regarding the sociodemographic status, diet, laboratory test indicators, physical examination parameters, and questionnaire data. Every participant submitted informed consent, and the NCHS Research Ethics Committee gave their approval to every procedure [21]. The website has comprehensive details about the program (https://www.cdc.gov/nchs/nhanes).

Study population

In this study, two cycles of NHANES data (2001–2004) were analysed. From 2001 to 2004, 21,161 participants in total took part in the survey. Firstly, we excluded females(n=10860) and males under 20 years of age(n=5374). The following were the subsequent exclusion criteria: (1) information about dietary(n=973) is unknown; (2) interviewees who hadn't finished the ED survey (n=838) ;(3) data about body mass index (n=61) is unknown; (4) education status (n=1) is unknown; (5) data about PIR (Poverty income ratio) (n=169) is unknown; (6) information about hypertension status (n=14) is unknown; (7) information about moderate activities (n=1) is unknown; and (8) smoking status (n=1) is unknown. Finally, a total of 2896 participants were included in the study after screening procedures, including 798 men with ED and the remaining without ED. The steps for screening are displayed in Fig. 1.

CDAI Measurement

The current component of the NHANES nutritional assessment is the 24-hour dietary recall interview with participants. Dietary recall interviews are conducted in person by trained dietary interviewers fluent in Spanish and English. Information on participants' food intake was collected by the NHANES through discontinuous two-day, 24-hour dietary recall interviews. The first interview was conducted at a mobile examination center (MEC) [22]. A standard set of measurement guidelines was available in the dietary interview room at each mobile examination center. Three to ten days later, the second dietary recall interview was conducted by telephone.

To minimize limitations, we used data from a single visit in 2001 ~ 2002, when there were no two measurements. In 2003 ~ 2004, when two measurements were available, we used the average of the two measurements to improve measurement precision. We used a changed version created by former scholars to compute CDAI for each participant [23, 24]. The six dietary antioxidants that constitute CDAI are zinc, selenium, carotenoids, and vitamins A, C, and E. After subtracting the mean, we standardized each micronutrient by dividing the

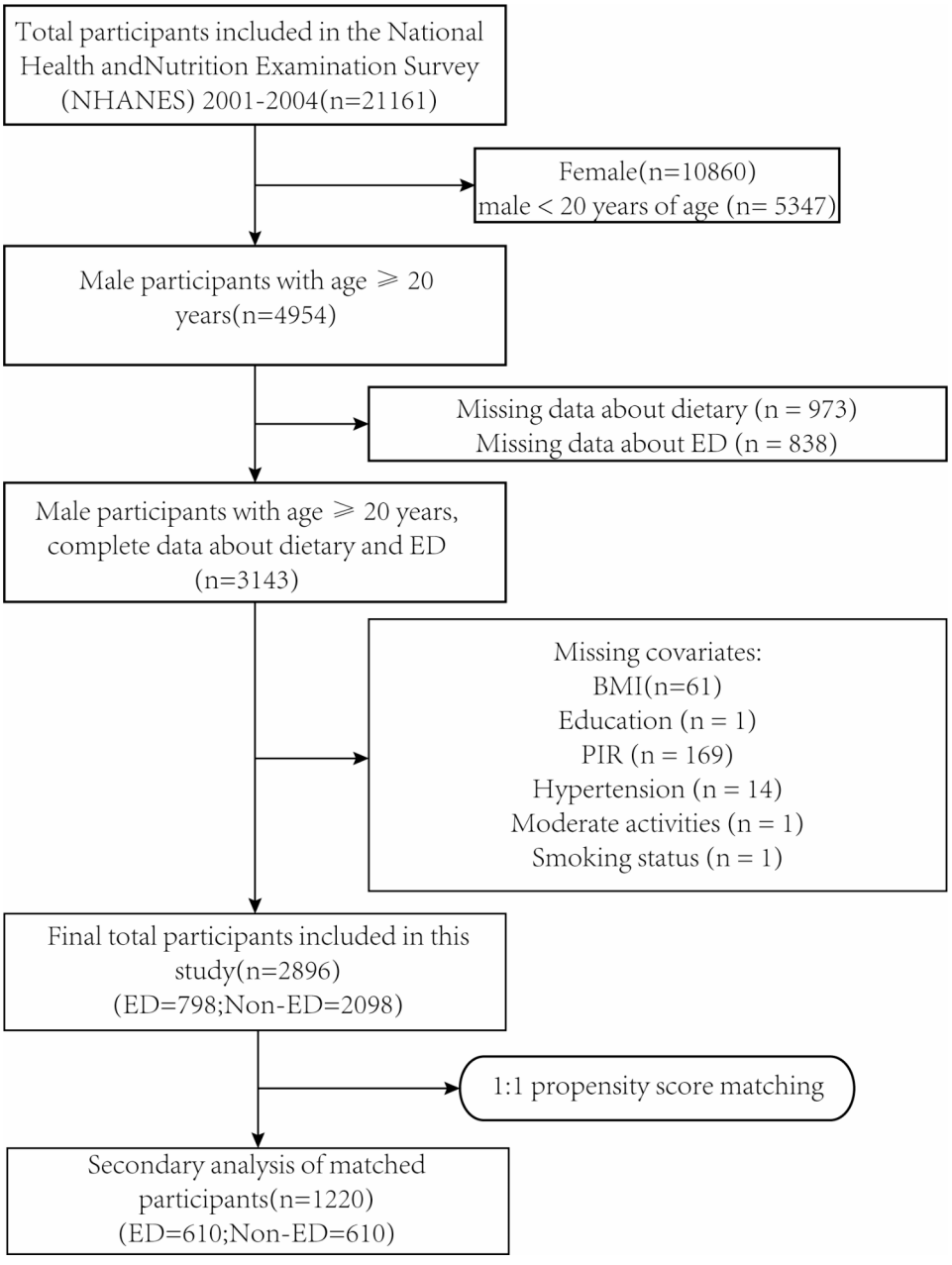

Fig. 1 Flowchart for screening research participants

outcomes by the standard deviation of the six dietary vitamins and minerals. We then summed the calculated standardized fractions of the micronutrients to calculate the CDAI. The calculation formula is as follows:

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

Measurement of ED

To evaluate ED, a question about "Ability to maintain an erection" (KIQ400) was answered by participants. It has been demonstrated that this question correctly identifies men who have an ED clinical diagnosis. Four options were presented to the participants, "almost always or always able", "usually able", "sometimes able" and "never able". Those who reported being "almost always(/always) able" and "usually able" in this study were excluded from consideration of having ED. Participants who responded with the last two answers were considered to be suffering from ED.

Covariate measurement

This study chose the following possible confounding variables that might influence ED outcomes according

to previous publications [25–31]. These factors included information on personal circumstances, medical history, physical examination results, and demographics. We included race, age, education, PIR, smoking status, hypertension, diabetes, depression status [32, 33], testosterone levels and activity status as categorical factors. Then, we divided the participants into two groups at the age of 50 years. The first group was younger than 50 years old, while the second group was older than or equal to 50 years old. We divided education status into three categories: below high school diploma, high school diploma, and above high school diploma. We categorized PIR < 2 as low PIR and PIR ≥ 2 as high PIR. Patients with hypertension were defined according to the following rules: participants who answered "yes" to the questionnaire "Have you been told you have hypertension?", participants who were taking antihypertensive medication, and participants whose average of three times measurement of systolic blood pressure (BP) was ≥140mmHg, and whose average of three times measurement of diastolic blood pressure (BP) was ≥90mmHg. When participants answered "yes" to the question about having diabetes or if they were using insulin, they were judged to have diabetes. Meanwhile, diabetes was taken into consideration by their fasting blood glucose (≥126 mg/dl) and glycosylated haemoglobin (≥6.5%).

Having engaged in at least ten minutes of vigorous or moderate activity during the past thirty days was considered to be a moderate or vigorous activity status. Participants were identified as smokers by answering "yes" to the question about smoking status (SMQ020). "Have you had a period of two weeks or longer when you felt sad, depressed, or empty in the past 12 months?" was the question used to evaluate the depression of each participant. Testosterone values < 3 ng/mL were defined as low testosterone levels and ≥3 ng/mL were defined as normal levels.

Statistical analysis

NHANES sample weights were used in the data analysis process to ensure the national representation of the study population. After categorising participants according to ED, a baseline characterisation was conducted. Categorical variables were expressed as weighted percentages (%) and compared using a chi-square test. Continuous variables were compared using weighted linear regression and expressed as mean (±standard deviation). Univariate Analysis was used to explore whether there were significant trends and differences. Then, in order to investigate the relationships between CDAI and ED using multivariate regression analysis, the study created four adjustment models. As an initial model, model 1 didn't modify any covariate variables. In model 2, race and age were added. In model 3, education, PIR, BMI, diabetes, hypertension, cigarette smoking, moderate activity, and vigorous activity were included based on model 2. Model 4 further incorporated testosterone and depression status based on Model 3. Furthermore, CDAI were split into 4 groups (Grouping of quartiles, Q1-4), with Q1 as the baseline value. At the same time, we performed a trend test. Then, using subgroup analysis, the stratified association between CDAI and ED was examined. Also, in order to evaluate the interplay of relationships between subgroups, interaction tests were included.

The restricted cubic splines (RCS) and smooth curve fitting were used to characterize the dose-response connection between CDAI and ED. The study investigated if there is a nonlinear association between CDAI and ED using the RCS, setting the node to 3 and using the median CDAI as the reference value. For smooth curve fitting under the completely modified model, a generalized additive model (GAM) was created to validate the dose-response relationship.

The "MatchIt" program from R Software was used to execute PSM (propensity score matching) by 1:1. To ensure that the covariate distributions of the ED and without ED groups were comparable, propensity score matching was carried out with the following factors: race, age, education, PIR, BMI, diabetes, hypertension, moderate activity, vigorous activity, and cigarette smoking [34]. To confirm the validity of the findings, the sample population was examined once more following PSM.

Empowerstats Software (Version 4.2) and R (version 4.4.0) were used for all statistical analyses. The two-sided p-value of less than 0.05 was deemed statistically significant.

Result

Population characteristics

Based on the screening criteria shown in Fig. 1, a total of 2896 eligible participants from NHANES 2001–2004 were selected. Of those, 2098 did not have ED, whereas 798 did. Table 1 displays the weighted estimates for the study population's baseline characteristics. The majority of study participants with erectile dysfunction were found to be over 50 years old. Moreover, participants with ED had lower levels of education, lower PIR, higher rates of smoking, higher BMI, less vigorous or moderate exercise status, and a higher probability of having a history of diabetes and hypertension than those without ED.

The influences of potential confounding factors related to ED were balanced by propensity score matching (PSM) analysis. The 1:1 PSM analysis was carried out in this study (Fig. 2). After PSM, the study included 1220 people in total, 610 of whom had ED and the remaining participants did not (Fig. 1). Table S1 showed the study population's weighted fundamental features after PSM. To a certain degree, covariate differences between the

Table 1 Weighted basic characteristics of screened participants(*N* = 2896) before PSM

Characteristics	Total	Erectile dysfunction		*P*-value
		No	Yes	
Age, years (%)				< 0.0001
<50	62.33 (58.98 ,65.57)	73.24 (70.04 ,76.21)	15.43 (11.38 ,20.59)	
≥50	37.67 (34.43 ,41.02)	26.76 (23.79 ,29.96)	84.57 (79.41 ,88.62)	
Race (%)				0.0023
Mexican American	7.68 (5.45 ,10.74)	8.11 (5.87 ,11.10)	5.87 (3.37 ,10.04)	
Other Hispanic	3.42 (2.19 ,5.30)	3.17 (2.10 ,4.74)	4.52 (2.26 ,8.84)	
Non-Hispanic white	75.27 (70.71 ,79.32)	74.12 (69.44 ,78.30)	80.20 (74.60 ,84.81)	
Non-Hispanic black	9.71 (7.56 ,12.38)	10.24 (7.92 ,13.15)	7.41 (5.33 ,10.19)	
Other	3.92 (2.93 ,5.22)	4.36 (3.16 ,6.01)	2.00 (1.03 ,3.88)	
Education (%)				< 0.0001
Less than high school	14.71 (13.03 ,16.58)	11.86 (10.37 ,13.52)	27.01 (22.60 ,31.93)	
High school diploma	25.45 (22.96 ,28.12)	26.01 (23.35 ,28.85)	23.06 (19.04 ,27.62)	
More than high school	59.83 (56.75 ,62.84)	62.14 (59.23 ,64.96)	49.93 (45.21 ,54.65)	
PIR (%)				0.0272
<2	26.08 (23.78 ,28.52)	25.12 (22.69 ,27.72)	30.22 (25.78 ,35.08)	
≥2	73.92 (71.48 ,76.22)	74.88 (72.28 ,77.31)	69.78 (64.92 ,74.22)	
Smoking (%)				< 0.0001
Yes	56.54 (53.11 ,59.90)	53.13 (49.53 ,56.70)	71.20 (66.05 ,75.86)	
No	43.46 (40.10 ,46.89)	46.87 (43.30 ,50.47)	28.80 (24.14 ,33.95)	
BMI (kg/m^2) (%)				< 0.0001
<25	29.16 (26.59 ,31.87)	30.80 (27.74 ,34.03)	22.14 (18.78 ,25.91)	
25-29.99	40.69 (38.22 ,43.20)	41.26 (38.60 ,43.97)	38.21 (34.24 ,42.35)	
≥30	30.15 (27.54 ,32.90)	27.94 (25.04 ,31.04)	39.65 (34.65 ,44.87)	
Hypertension (%)				< 0.0001
No	66.09 (62.39 ,69.60)	71.36 (67.43 ,74.99)	43.40 (38.42 ,48.52)	
Yes	33.91 (30.40 ,37.61)	28.64 (25.01 ,32.57)	56.60 (51.48 ,61.58)	
Diabetes (%)				< 0.0001
No	89.81 (88.44 ,91.04)	93.96 (92.55 ,95.12)	71.96 (67.46 ,76.06)	
Yes	10.19 (8.96 ,11.56)	6.04 (4.88 ,7.45)	28.04 (23.94 ,32.54)	
Vigorous activity status (%)				< 0.0001
Yes	40.55 (37.47 ,43.70)	45.62 (42.25 ,49.03)	18.72 (15.01 ,23.10)	
No	59.45 (56.30 ,62.53)	54.38 (50.97 ,57.75)	81.28 (76.90 ,84.99)	
Moderate activity status (%)				0.0001
Yes	59.38 (56.50 ,62.19)	61.23 (57.98 ,64.37)	51.42 (47.03 ,55.79)	
No	40.62 (37.81 ,43.50)	38.77 (35.63 ,42.02)	48.58 (44.21 ,52.97)	
Testosterone (%)				0.001
Low	2.02 (1.43 ,2.84)	1.53 (1.05 ,2.22)	4.14 (2.49 ,6.81)	
Normal	12.08 (10.23 ,14.22)	12.64 (10.57 ,15.06)	9.66 (6.77 ,13.61)	
Unknown	85.90 (83.71 ,87.84)	85.83 (83.36 ,87.98)	86.20 (82.67 ,89.11)	
Depression (%)				< 0.0001
Yes	3.16 (2.31 ,4.30)	3.83 (2.82 ,5.18)	0.26 (0.06 ,1.09)	
No	14.93 (12.67 ,17.50)	17.85 (15.19 ,20.85)	2.36 (1.30 ,4.24)	
Unknown	81.92 (79.17 ,84.37)	78.32 (75.22 ,81.13)	97.38 (95.31 ,98.55)	
CDAI (mean)	2.30 (0.32)	2.55 (0.35)	1.23 (0.41)	< 0.0001

Data are shown as means (SD) or percentages (95%CI)

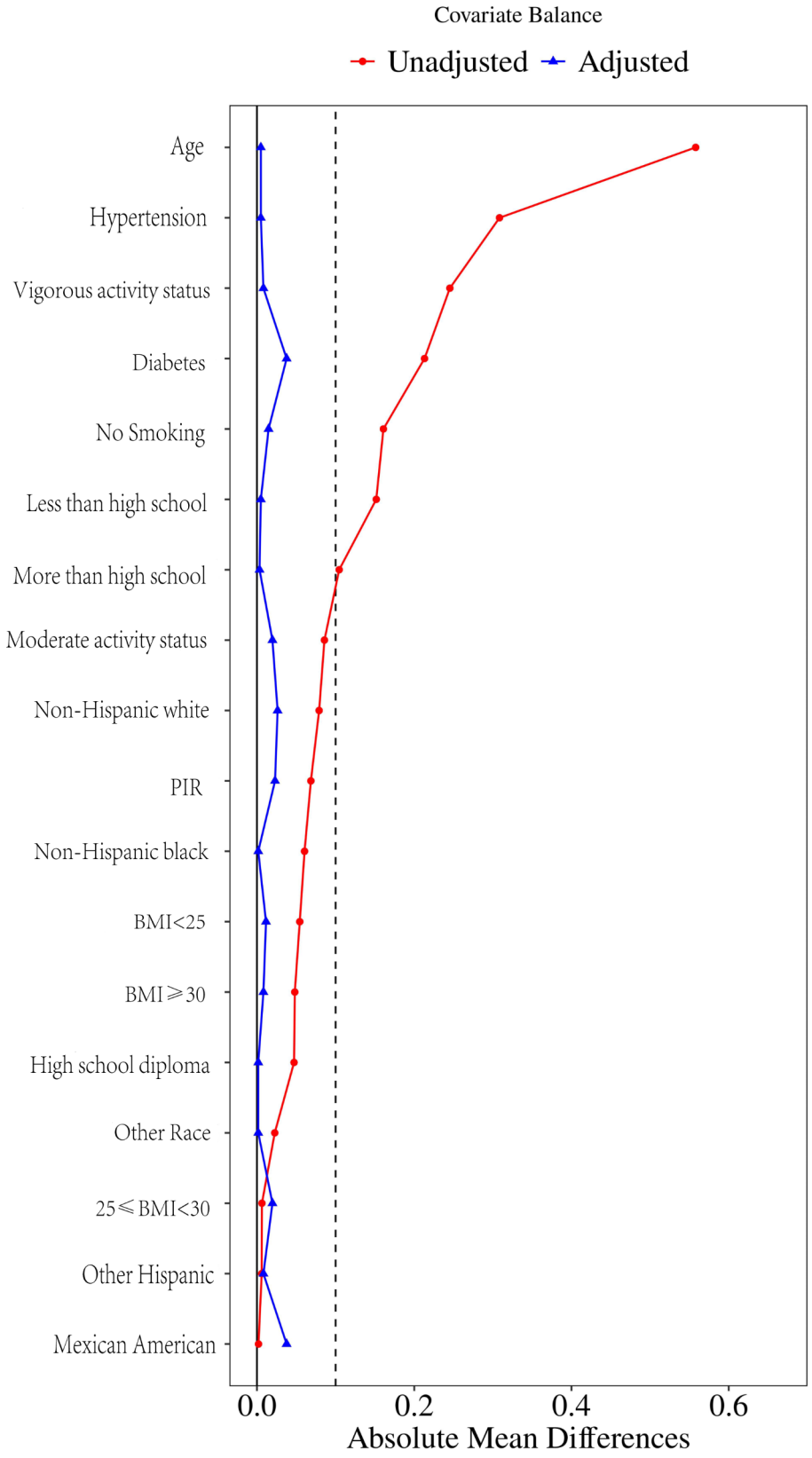

Fig. 2 PSM analysis was performed by the 1:1-based minimum adjacency method

two groups were controlled. After PSM, it was seen that only education and CDAI showed a significant correlation with ED ($p=0.0107$ and $p=0.0145$).

Univariate analysis of ED

The outcomes of the univariate analysis are displayed in Table 2. We concluded that before PSM, ED was positively associated with age≥50 years old, BMI≥25, hypertension, diabetes, without vigorous activity, and without moderate activity. In addition, ED was negatively associated with high school education level and above, non-Hispanic blacks or other races, PIR≥2, without smoking, normal testosterone levels, and CDAI value. After PSM, erectile dysfunction was not linked to

Table 2 Univariate regression analysis prior to and following PSM

Variables	Prior to PSM		Following PSM	
	OR (95%CI)	*P*	OR (95%CI)	*P*
Age (years)				
<50	1.00 (Reference)		1.00 (Reference)	
≥50	14.73 (11.76 ~ 18.47)	**< 0.001**	0.97 (0.72 ~ 1.30)	0.818
Race				
Mexican American	1.00 (Reference)		1.00 (Reference)	
Other Hispanic	1.22 (0.75 ~ 1.97)	0.422	1.52 (0.79 ~ 2.91)	0.210
Non-Hispanic white	1.16 (0.94 ~ 1.43)	0.157	1.25 (0.94 ~ 1.67)	0.123
Non-Hispanic black	0.69 (0.52 ~ 0.91)	**0.009**	1.22 (0.81 ~ 1.82)	0.342
Other	0.40 (0.21 ~ 0.76)	**0.005**	1.31 (0.56 ~ 3.08)	0.537
Education				
Less than high school	1.00 (Reference)		1.00 (Reference)	
High school diploma	0.48 (0.38 ~ 0.60)	**< 0.001**	1.02 (0.75 ~ 1.39)	0.884
More than high school	0.47 (0.39 ~ 0.57)	**< 0.001**	1.02 (0.79 ~ 1.33)	0.863
PIR				
<2	1.00 (Reference)		1.00 (Reference)	
≥2	0.75 (0.63 ~ 0.88)	**< 0.001**	0.90 (0.72 ~ 1.14)	0.402
Smoking				
Yes	1.00 (Reference)		1.00 (Reference)	
No	0.50 (0.42 ~ 0.60)	**< 0.001**	1.07 (0.84 ~ 1.36)	0.583
BMI (kg/m^2)				
<25	1.00 (Reference)		1.00 (Reference)	
25-29.99	1.24 (1.01 ~ 1.51)	**0.040**	0.91 (0.69 ~ 1.21)	0.525
≥30	1.43 (1.15 ~ 1.77)	**0.001**	0.98 (0.72 ~ 1.33)	0.884
Hypertension				
No	1.00 (Reference)		1.00 (Reference)	
Yes	3.66 (3.09 ~ 4.34)	**< 0.001**	0.98 (0.78 ~ 1.23)	0.863
Diabetes				
No	1.00 (Reference)		1.00 (Reference)	
Yes	5.05 (4.03 ~ 6.31)	**< 0.001**	1.27 (0.96 ~ 1.68)	0.101
Vigorous activity status				
Yes	1.00 (Reference)		1.00 (Reference)	
No	3.39 (2.77 ~ 4.15)	**< 0.001**	0.95 (0.73 ~ 1.25)	0.728
Moderate activity status				
Yes	1.00 (Reference)		1.00 (Reference)	
No	1.41 (1.20 ~ 1.66)	**< 0.001**	0.92 (0.74 ~ 1.16)	0.491
Testosterone				
Low	1.00 (Reference)		1.00 (Reference)	
Normal	0.29 (0.17 ~ 0.49)	**< 0.001**	0.75 (0.38 ~ 1.49)	0.415
Depression				
Yes	1.00 (Reference)		1.00 (Reference)	
No	1.07 (0.36 ~ 3.21)	0.899	0.83 (0.16 ~ 4.15)	0.815
CDAI	0.92 (0.90 ~ 0.94)	**< 0.001**	0.97 (0.94 ~ 0.99)	**0.031**

Abbreviations: PIR, Poverty income ratio; BMI, body mass index; PSM, propensity score matching; CDAI, Composite Dietary Antioxidant Index; OR, odds ratio; CI, confidence interval

covariates including age, education, race, income, hypertension, diabetes, smoking, exercise level, etc. (Table 2). Only CDAI showed a negative correlation with ED (OR = 0.97, $P = 0.031$).

The relationships between CDAI and ED

By quaternity, CDAI was transformed (Q1-4; Q1 serving as a point of comparison) and analysed. Figure 3 illustrates how the three correction models relate to CDAI and ED both before and following PSM. ED prevalence decreased progressively as CDAI rose under every correction model regardless of before or after PSM [In Model 3, Q4 is compared to Q1 with the OR = 0.60(0.45, 0.81) and 0.53 (0.37, 0.75) before and after PSM, the *p*-values were both less than 0.001, and the *P* for trend is less than 0.001 and equal to 0.001, respectively].

In addition, we added covariates to Model 3: testosterone levels and depression status (both of which had many missing values) as Model 4. Regression analyses before and after PSM were performed by trisecting the population's CDAI (Table S2). Model 1 used a basic model without any covariate adjustments. Model 2 had adjustments for race and age. Based on Model 2, additional variables were introduced In Model 3, including education, PIR, BMI, diabetes, hypertension, cigarette smoking, and moderate activity/vigorous activity. Model 4 was adapted much further for testosterone and depression based on Model 3. It is still evident that when CDAI rises, the incidence of ED progressively declines, both before and after PSM.

Subgroup analysis between CDAI and ED

Table 3 displays the stratification of the study by race, age, education, PIR, BMI, diabetes, hypertension, smoking or not, moderate activity, and vigorous activity. In individuals who were 50 years of age or older, had completed higher than high school education, were non-Hispanic black or Mexican American, had low PIR, had a BMI of more than 30, had diabetes, engaged in vigorous activity, and did not participate in moderate activity, CDAI showed a strong negative correlation with ED. Furthermore, whether smoking or not, participants' CDAI had a significant negative association with the occurrence of ED. ($P < 0.05$).

After PSM, the research also demonstrated that ED and CDAI had the previously indicated characteristics (Table 3). Moreover, vigorous activity has been shown to have a significant interaction with CDAI. (P for interaction = 0.012).

Connectivity between CDAI and ED in terms of dose-response

Figure 4 displays the RCS result of the dose-response relationship. CDAI and the prevalence of ED did not show a nonlinear relationship under the fully adjusted model, both before and after PSM (Before PSM, *p* for nonlinearity was 0.293, while after PSM, *p* for nonlinearity was 0.147). The reference values before and after PSM were 1.067 and 0.515, respectively, for CDAI at OR = 1. This data could be used as a reference to help men avoid developing ED.

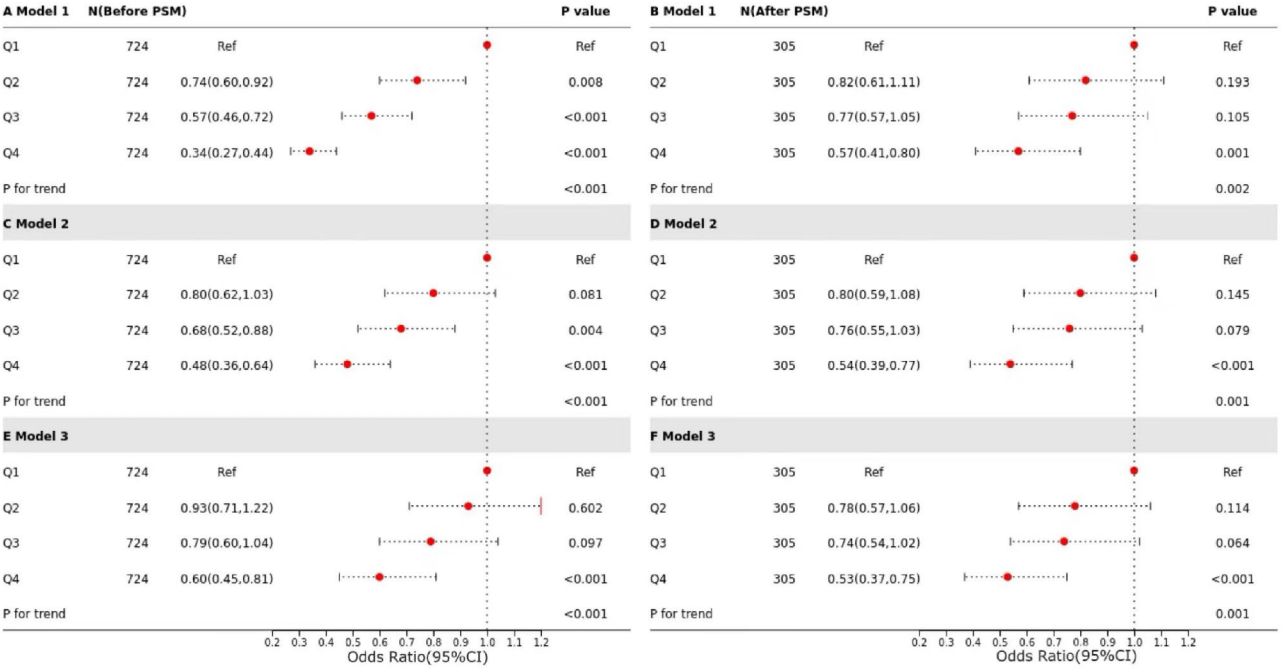

Fig. 3 The relationship between ED prevalence and CDAI before and after PSM. It is denoted by the letters "A", "C", and "E", before PSM. Meanwhile, it is denoted by the letters "B", "D", and "F", after PSM

Table 3 Stratified relationships between CDAI and ED prevalence in U.S. male adults

Subgroup	Prior to PSM			Following PSM		
	OR (95%CI)	*P*	*P* for interaction	OR (95%CI)	*P*	*P* for interaction
Age(y)			0.988			0.549
< 50	0.77(0.51,1.17)	0.229		0.83 (0.46,1.50)	0.534	
≥ 50	0.70(0.56,0.89)	**0.003**		0.71 (0.55,0.91)	**0.007**	
Race			0.128			0.088
Mexican American	0.58 (0.38,0.91)	**0.017**		0.50 (0.29,0.87)	**0.014**	
Other Hispanic	1.24 (0.30,5.22)	0.767		2.12 (0.31,14.50)	0.444	
Non-Hispanic white	0.82 (0.63,1.07)	0.144		0.81 (0.60,1.08)	0.155	
Non-Hispanic black	0.42 (0.22,0.78)	**0.006**		0.37 (0.17,0.78)	**0.009**	
Other	1.28 (0.26,6.19)	0.763		26.22 (0.35,1937.19)	0.137	
Education			0.532			0.649
Less than high school	0.78 (0.54,1.13)	0.183		0.67 (0.43,1.05)	0.082	
High school diploma	0.60 (0.39,0.92)	**0.018**		0.77 (0.47,1.25)	0.290	
More than high school	0.72 (0.53,0.98)	**0.036**		0.66 (0.47,0.93)	**0.019**	
PIR			0.396			0.415
< 2	0.61 (0.44,0.86)	**0.005**		0.59 (0.39,0.89)	**0.012**	
≥ 2	0.78 (0.60,1.01)	0.057		0.80 (0.60,1.06)	0.122	
Smoking			0.850			0.811
Yes	0.73 (0.57,0.93)	**0.012**		0.75 (0.56,0.99)	**0.041**	
No	0.66 (0.46,0.95)	**0.025**		0.64 (0.42,0.97)	**0.038**	
BMI(kg/m2)			0.431			0.443
< 25	0.63 (0.42,0.96)	**0.033**		0.95 (0.58,1.54)	0.822	
25-29.99	0.79 (0.59,1.08)	0.137		0.69 (0.49,0.98)	**0.038**	
≥ 30	0.69 (0.47,0.99)	**0.046**		0.61 (0.41,0.93)	**0.022**	
Hypertension			0.752			0.822
No	0.72 (0.54,0.95)	**0.023**		0.72 (0.51,1.02)	0.067	
Yes	0.74 (0.55,0.98)	**0.038**		0.72 (0.53,1.00)	0.050	
Diabetes			0.419			0.091
No	0.77 (0.61,0.96)	**0.023**		0.80 (0.62,1.04)	0.094	
Yes	0.56 (0.35,0.88)	**0.013**		0.46 (0.27,0.79)	**0.005**	
Vigorous activity status			0.053			**0.012**
Yes	0.54 (0.35,0.83)	**0.005**		0.42 (0.25,0.71)	**0.001**	
No	0.80 (0.63,1.01)	0.057		0.85 (0.65,1.10)	0.221	
Moderate activity status			0.177			0.227
Yes	0.89 (0.66,1.19)	0.429		0.90 (0.64,1.27)	0.535	
No	0.61 (0.46,0.81)	**< 0.001**		0.62 (0.45,0.85)	**0.003**	

In this model, all covariates have been adjusted without adjusting the stratified variable itself

Moreover, smooth curve fitting, GAM, and a model of threshold effects derived from the RCS data were used to investigate the linear relationship between CDAI and the prevalence of ED. As CDAI rose, the prevalence of ED declined (Fig. 5). Furthermore, Table 4 illustrates the threshold impact between CDAI and the occurrence of ED. The inflexion point (k) was 5.04 both before and after PSM in the Complete Adjustment Model [Log-likelihood ratios were 0.101 (before PSM) and 0.041(after PSM)]. The linear model provided a more comprehensive explanation of the link between CDAI and ED [OR=0.97, *P*=0.0055 (before PSM); OR=0.97, *P*=0.0183(After PSM)]. Furthermore, we can conclude that with a CDAI of less than 5.04, the incidence of ED decreased by 7% for each unit increase in CDAI (OR=0.93, p=0.0023, Log-likelihood ratio=0.041).

Discussion

This is the first paper to investigate the relationship between CDAI and ED using a sizable sample size. Our cross-sectional investigation of the population in the NHANES sample, which was nationally representative, yielded some noteworthy findings. We concluded that a higher CDAI is associated with a lower prevalence of ED, independent of the adjustment model. In addition, an association between CDAI and ED prevalence that is linearly negative was supported by smooth curve fitting, RCS and analysis of the threshold effect. Thus, ED

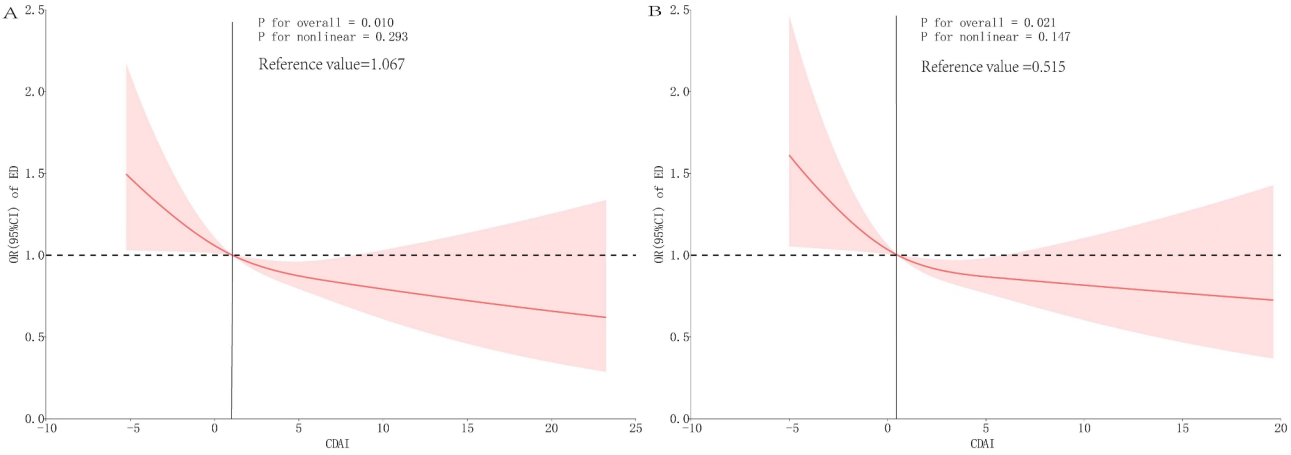

Fig. 4 CDAI dose-response analysis and ED prevalence prior to and following PSM(RCS). Notes: A: whole modified model, before PSM; B: whole modified model, after PSM. Terminologies: PSM, propensity score matching; ED, erectile dysfunction; RCS, restricted cubic splines

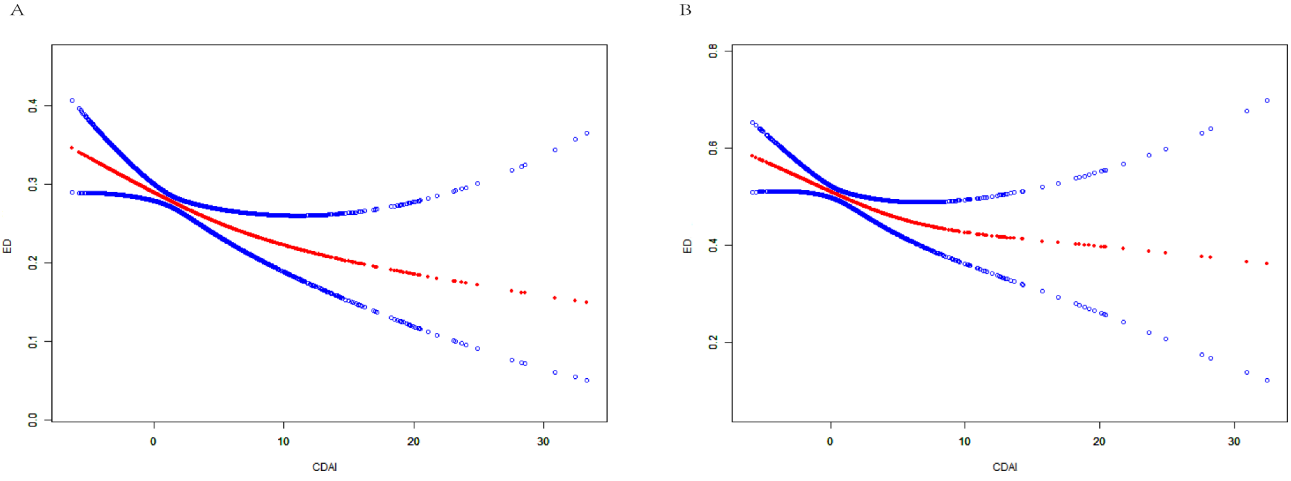

Fig. 5 Association between the risks of ED and CDAI Levels with Smooth curve fitting. Notes: A, B: whole modified model (before, after PSM). Terminologies: PSM, propensity score matching; CDAI, Composite Dietary Antioxidant Index; ED, erectile dysfunction

Table 4 Relationship between CDAI and prevalence of ED (analysis of the threshold effect)

Outcome	OR (95% CI), *P*	
	Prior to PSM	**Following PSM**
Model one		
Linear impact	0.97 (0.94, 0.99) **0.0055**	0.97 (0.94, 0.99) **0.0183**
Model two		
Inflection point (K)	5.04	5.04
CDAI < K	0.94 (0.90, 0.98) **0.0026**	0.93 (0.89, 0.97) **0.0023**
CDAI > K	1.00 (0.95, 1.05) 0.9858	1.01 (0.96, 1.07) 0.6463
Log likelihood ratio	0.101	**0.041**

This model included adjustments for all covariates

incidence may be decreased by maintaining a specific level of CDAI.

The cavernous tissues of the penis, as well as endocrine, vascular, and neurological tissues, are all involved in the intricate physiological process of erection [25]. Patients may experience ED if there are abnormalities in any one or more of these factors. Modifiable risk factors for ED include metabolic syndrome, smoking status, obesity, and not exercising enough, among others [35, 36]. Remarkably, proinflammatory states are strongly associated with all of these characteristics. The development and course of ED are significantly influenced by oxidative damage and inflammation. Oxidative stress and inflammation levels were greater in the ED group, according to the prospective studies [11, 37]. The peroxynitrite, hydroxyl groups, and highly activated singlet oxygen are produced when Reactive Oxygen Species (ROS) interact with one another target protein lipids and DNA, impairing transport, cell signalling, energy metabolism, and other processes [38]. According to the findings of earlier research, oxidative stress—which is typified by an excessive generation of ROS—plays a significant part in the aetiology of ED [39].

In response to sexual excitement, vascular endothelial cells release endothelial factors, such as nitric oxide

(NO). The erectile tissue's arterial smooth muscle is relaxed by these elements. Meanwhile, they prevent its venous return and enhance blood flow to the penis. An erection eventually results from blood becoming confined inside the cavernosa and a large increase in cavity pressure [7]. The integrity of the endothelium is crucial to a penile erection, but it is damaged and weakened by oxidative strain and chronic inflammation [40, 41]. There are several pathways by which oxidative stress affects endothelial function: (1) Oxidative stress produces free radicals that damage endothelial cells and affect their function. (2) Endothelial dysfunction impairs associated blood vessel contraction and dilatation as well as the synthesis and release of physiological NO [42]. (3) Oxidative stress promotes the release of inflammatory mediators, leading to activation and damage of endothelial cells, further compromising their integrity. (4) Oxidative stress can increase the permeability of endothelial cells, leading to vascular leakage and affecting nutrient and waste removal from tissues. (5) Chronic oxidative stress is associated with atherosclerosis, which impairs endothelial function.

Antioxidant therapy therefore has the potential to be a treatment for ED. Certain medications, such as vardenafil, tadalafil, and sildenafil, among others, can sustain penile erection by lowering Malondialdehyde (MDA) levels through the stimulation of Superoxide Dismutase (SOD) and catalase activity [43].

Chronic inflammation, a significant contributing factor to many metabolic illnesses, can be brought on by poor dietary practices [44]. A pro-inflammatory diet can exacerbate the body's inflammatory response by elevating oxidative stress and autoimmune disorders. Reactive oxygen species (ROS) are produced as a result of pro-inflammatory behaviours, and ROS can result in endothelial dysfunction and chronic vascular inflammation [45]. According to earlier research, consuming an antioxidant-rich diet can dramatically lower the death rate among Americans in general [46–48]. Increasing dietary antioxidant consumption also appears to protect against a host of other illnesses, such as depression, osteoporosis, cancer, and HPV infection [49]. Diet affects plasma redox state as an external factor and offers defence against reactive nitrogen species and ROS. Dietary antioxidants may scavenge oxidants to maintain a stable cellular redox state and avert oxidative stress [50].

The effects of certain nutrients on ED have been the main focus of earlier clinical research. For example, lycopene negatively correlates with the incidence of ED [28]. However, evaluating total dietary antioxidant consumption can offer a more thorough insight, given the inherent combinations of nutrients included in food [51]. Based on characteristics that are resistant to inflammation and represent a person's profile of antioxidants, CDAI was established and used in much research widely. Nevertheless, there is currently little proof linking CDAI to ED. This study fills this research gap and demonstrates that the level of dietary antioxidant intake is negatively associated with the incidence of ED.

We performed a subgroup analysis in compliance with the statement Strengthening the Reporting of Observational Studies in Epidemiology in order to improve the use of data to uncover the fundamental truth [52]. In subgroup analysis, we detected a strong negative connection between CDAI and ED among the specified risk factors. Additionally, a significant interaction between CDAI and vigorous activity status was found. The precise causes of this discovery are unknown, however, these people may have higher oxidative stress levels because exogenous antioxidant intake seems to be more protective for people with higher innate or acquired ROS levels [53]. Our findings imply that a high level of CDAI may be more beneficial to those at high risk for ED. A single-linear model might account for the linear negative connection between CDAI and ED prevalence that was also revealed by threshold effect, RCS, and GAM analyses, as well as results from smoothed curve fitting. In addition, the relationship between rising CDAI and decreasing ED incidence was more pronounced at CDAI values less than the K value of 5.04(OR = 0.93, $p = 0.0023$).

ED is often considered an early warning sign of cardiovascular problems due to common risk factors such as poor vascular health. Negative correlations suggest that higher dietary antioxidant intake (higher CDAI) is associated with a lower risk of erectile dysfunction. Antioxidants help reduce oxidative stress, which has been linked to both cardiovascular health and erectile dysfunction. Public health programs can promote diets rich in antioxidants such as fruits, vegetables, nuts, and whole grains. By encouraging healthier dietary patterns, especially in populations at high risk for both CVD and ED, public health programs can improve chronic disease prevention and management more broadly. Increasing dietary intake of antioxidants to reduce the prevalence of ED can have a positive impact on quality of life and mental health. Policymakers may consider incorporating dietary recommendations to reduce ED risk into broader public health nutritional guidelines, especially in areas with a high prevalence of cardiovascular disease and poor dietary practices. In areas where antioxidant-rich foods are not part of the traditional diet, public health professionals could develop targeted interventions to encourage people to eat more of these foods in a way that respects local preferences. These would make health policies more targeted and effective, with lasting benefits for individual and group health.

Our research, however, has several shortcomings. We were unable to draw an explicit causal conclusion from

the survey because it was cross-sectional in nature. We believe that future research on this topic (utilizing other databases or surveys conducted by the authors themselves) could take the form of a longitudinal study, refining the findings by tracking ED in the population over time (annually, every three years, etc.). In addition, our study used patient self-report questions from the NHANES database to assess ED, which has potential limitations. For example, (1) Subjectivity: respondents may not accurately understand and describe their symptoms, leading to information bias. (2) Social expectations: respondents may conceal their true symptoms due to social pressure.

In addition, although the correlation between CDAI and ED in US men was examined using a nationally representative database, the global general population could not be included in this association. However, the associations we identified between antioxidant diets and ED are based on biological and behavioural mechanisms that are likely to exist in different populations. For example, an antioxidant diet effectively reduces oxidative stress to ensure endothelial functional integrity, which effectively reduces the risk of ED. This may suggest that similar patterns can be observed in other populations. We recognize that dietary habits, healthcare delivery, and other lifestyle factors vary considerably across regions. Therefore, future studies are necessary to explore how these differences may affect the relationship between antioxidant diets and ED incidence, especially in populations with different nutritional statuses and healthcare systems. Future studies could replicate our study in countries with different diets, such as Asia or Europe.

Conclusions

This research attempts to increase knowledge about the role that diet plays in the onset of ED. The results show that ED prevalence and CDAI have a linearly negative connection. Furthermore, the effect of dietary intake of exogenous antioxidants is more pronounced in people at high risk of ED. Therefore, for ED risk assessment and prevention, the CDAI might be utilized as an important indicator.

Abbreviations

CDAI	Composite dietary antioxidant index
ED	Erectile dysfunction
PSM	Propensity score matching
BMI	Body mass index
PIR	Poverty income ratio
OR	Odds ratio
CI	Confidence interval
Ref	Reference

Supplementary Information

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Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

Supplementary Material 4

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Author contributions

Conception and design: XFJ. Data collection: XFJ and LS. Data analysis and interpretation: XFJ, LS, HXL and YL. Manuscript writing: XFJ and LS. Final approval of manuscript: All authors.

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Data availability

The data used and analyzed during the present study are available from NHANES (https://www.cdc.gov/nchs/nhanes/). Data collated by the authors are provided in the supplementary information document.

Declarations

Competing interests

The authors declare no competing interests.

Ethics approval and consent to participate

NHANES was approved by National Center for Health Statistics Research Ethic Review Board. All subjects signed the informed consent during the recruitment period.

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