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Association Between the Composite Dietary Antioxidant Index and Anemia: A Cross-Sectional Study

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Abstract

As an important indicator of an antioxidant-rich diet, the composite dietary antioxidant index (CDAI) has been associated with various disease conditions. However, its associations with anemia remains poorly understood. Our study aimed to investigate the relationship of the CDAI with anemia in the general adults. A total of 9958 participants were enrolled from the National Health and Nutrition Examination Surveys (NHANES) 2011-2014. The CDAI was calculated from the intake of the six dietary antioxidants: vitamins A, C and E, carotenoid, selenium, and zinc. Multivariable logistic regressions were employed to explore the associations between the CDAI and anemia. Additionally, the restricted cubic spline (RCS) was performed to explore the non-linearity between the CDAI and anemia. Subgroup analysis was performed to confirm the association of the CDAI with anemia. Mediation analysis was used to explore the role played by albumin in the association between the CDAI and anemia. In the multivariable logistic regression model, the odds ratio (95% confidence interval, OR, 95%CI) of the CDAI per 1 unit increase for anemia was 0.97 (0.95-0.98; $P = 0.001$). Compared to the lowest quartile, the highest quartile of the CDAI was associated with a lower risk of anemia (0.73 [0.59-0.90]; $P = 0.004$). The CDAI component-specific analysis revealed that the odds ratios (95%CI) were 0.87 (0.76, 0.98; $P = 0.035$) for vitamin A, 0.98 (0.98, 0.99; $P = 0.034$) for vitamins E, and 0.98 (0.96, 0.99; P=0.003) for Zinc, respectively. Restricted cubic spline suggested that the relationship between the CDAI and anemia was linear. Further, subgroup analysis indicated a stable negative association between the CDAI and anemia (all P for interaction >0.05). Mediation analyses showed that the association of CDAI with anemia was mediated by the albumin, accounting for 42.92% ($P < 0.001$) of the total effects. Our study highlighted a negative linear association between the CDAI and anemia in the general adults. Intake of the six dietary antioxidants composing the CDAI could help to prevent the occurrence of anemia.

Keywords: Composite dietary antioxidant index; Anemia; Mediation analysis.

Abbreviations

BMI = body mass index; TC = total cholesterol; CHF = congestive heart failure; CHD = coronary heart disease; CDAI = composite dietary antioxidant index; OR = odds ratio; CI = confidence interval; Ref = reference.

Introduction

According to the Global Burden of Disease Study 2019, approximately 1.8 billion people worldwide were inflicted with anemia [1]. Anemia can cause severe hypoxia of different organs, and patients may present symptoms such as

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cognitive decline, depression, cardiovascular complications, indigestion, dizziness, and exertional dyspnea [2]. When reactive oxygen species production was imbalanced with antioxidant defenses, oxidative stress can result in damage to cell death and functional impairment [3]. Clinical studies have demonstrated that anemia patients have been in hyper oxidative state [4]. Intake of antioxidants can mitigate the oxidative stress by boosting plasma levels of antioxidants, reducing the risk of anemia [5].

The CDAI is a composite estimate of a person's overall affinity for antioxidant exposure status, which is a summary score of the six dietary antioxidants, including vitamins A, C and E, carotenoid, selenium, and zinc [6]. Previous studies revealed that the higher CDAI is associated with a reduced risk of a variety of entities [7-10]. However, the relationship between the CDAI and anemia is not yet to be reported. Therefore, We hypothesized that elevated CDAI levels could prevent anemia, using data from the 2011-2014 National Health and Nutrition Examination Survey (NHANES) database.

Methods

Study population

The current study included participants from the National Health and Nutrition Examination Survey (NHANES 2011-2014). Individuals with missing dietary or hemoglobin data were excluded. We further excluded individuals younger than 18 years old and pregnant women. Finally, adults with complete information were included in our study. The survey was approved by the Ethical Review Board of National Center for Health Statistics and all participants provided written informed consent. This study used publicly available datasets from the NHANES database. The data can be found at https://www.cdc.gov/nchs/nhanes/index.htm.

Calculation of the CDAI

Each participants' food and nutrient intake in the NHANES was collected from the dietary interview component, which was called “what we eat in America.” The data analysis was conducted by the Food Surveys Research Group of the US Department of Agriculture. The participants responded to two recall surveys: the first time was in the Mobile Examination Center, while the second was on the telephone. We used the first recall data to avoid memory drift caused by too long a time. Based on the interview, we determined the intake of dietary supplements, including dosage, frequency, and duration of consumption [11]. The CDAI was based on the dietary intake of the six antioxidants: zinc, selenium, carotenoids, vitamins A, C, and E. Six antioxidants were standardized by subtracted the mean and divided by standard deviation. The dietary antioxidant intake in this study was calculated based on pure absolute intake and did not include antioxidants obtained from supplements, medications, or other additional sources. Then the CDAI was based on the sum of these standardized consumptions. The formula for the CDAI is as follows:

$$CDAI = \sum_{i=1}^{6} X_i - u_i/S_i.$$

Where, X_i is the daily intake of antioxidants, u_i is the mean of antioxidants, and S_i is the standard deviation of u_i (7).

Measurement of hemoglobin and Diagnosis of Anemia

According to the World Health Organization diagnostic criteria, the hemoglobin level (g/dL) for diagnosing anemia at sea level is <12 for women and <13 for men, respectively. Beckman Coulter HMX instrument in the Mobile Examination Centre produces a full blood count on a blood sample and provides blood cell distribution for all attendees.

Study variables

To control the influence of potential confounding factors, we selected the following covariates: age, sex, race, education levels, smoking status, drinking status, examination information on body mass index (BMI), total cholesterol (TC), serum iron, folate as well as self-reported diseases like diabetes mellitus and hypertension, congestive heart failure, and myocardial infarction. We divided race into non-Hispanic white, non-Hispanic black, Mexican-American, and other groups. The education level was divided into three groups: below high school, high school, and above high school. Smoking status and drinking status were both categorized as never, former, and current. ‘Never smoking’ referred to someone who has not smoked over 100 cigarettes during their lifetime and does not smoke at present. ‘Former smoking’ was someone who has smoked over 100 cigarettes during their lifetime, but has not smoked within the past 28 days. ‘Current smoking’ was defined as a subject who has smoked over 100 cigarettes during their lifetime and has smoked at least once within the past 28 days. Never, former, and current drinking corresponded to the following situations, respectively, participants who drunk less than 12 drinks in their lifetime, individuals who drunk more than 12 drinks during their lifetime, but no drinks in the past year, and subjects who drunk in the past year. BMI was computed utilizing the formula weight (kg) divided by the square of height in meters.

Statistical analysis

Participants were divided into two groups based on whether or not they had anemia. Continuous variables were presented as means ± standard deviation while categorical variables were presented as counts (percentages). Student’s t test was used to analyze continuous variables and outcomes,

and Chi-Square test was used for categorical variables. Multiple imputation for 25 times was performed for missing values. Logistic regression models was used to explore the relationship between the CDAI and anemia. Model 1 did not adjust for covariates, model 2 adjusted for age, sex, and race, and model 3 further adjusted for age, sex, race, education, total cholesterol, serum iron, BMI, folate, smoking, drinking, hypertension, congestive heart failure, diabetes mellitus, and myocardial infarction. The restricted cubic splines were performed to explore the nonlinearity between the CDAI and anemia. Multivariable linear regression models were employed to investigate the association between CDAI and the serum albumin. Mediator analysis was also used to explore whether the serum albumin mediated the association between the CDAI and anemia. Subgroup/sensitivity analysis was further performed to confirm the association of the CDAI with anemia stratified by sex, smoking status, drinking status, educational level, age, BMI, total cholesterol, folate, hypertension, and diabetes. These stratification factors were also considered pre-specified potential effect modifiers. An interaction term was added to test for heterogeneity in the associations between subgroups. All the analyses were performed using STATA version 17.0 and R software version 4.2.0. Statistical significance was defined as a two-tailed *P* value <0.05.

Results

Baseline Characteristics

This study involved a total of 9958 participants (Figure.1). The baseline features of participants were described in detail in Table 1. Patients with anemia had lower CDAI scores. In addition, patients with anemia were older and female, had higher BMI and had lower albumin.

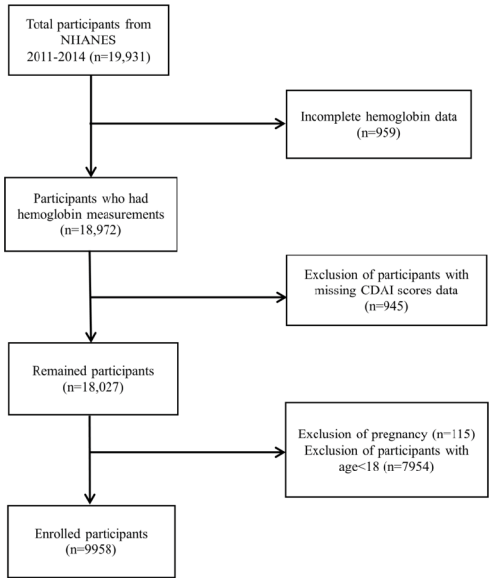

Figure 1: The flow chart of participant selection

Table 1: Characteristics of the study population based on the presence of anemia status.

Variables	Overall (n = 9958)	Non-Anemia (n = 8888)	Anemia (n = 1070)	*P*
Age, years	47.23 ± 18.37	46.48 ± 18.10	53.41 ± 19.49	<0.001
Male, n (%)	4907(49.28)	4532 (50.99)	375(35.05)	<0.001
Race, n (%)				<0.001
Mexican American	1203(12.08)	1094(12.31)	109(10.19)	
Non-Hispanic White	4107(41.24)	3845(43.26)	262(24.49)	
Non-Hispanic Black	2265(22.75)	1784(20.07)	481(44.95)	
Others	2383(23.93)	2165(24.36)	218(20.37)	
Education level, n (%)				<0.001
Less than high school	2018(21.44)	1744(20.76)	274(27.00)	
High school	2064(21.93)	1848(22.01)	216(21.28)	
Above high school	5331(56.63)	4806(57.23)	525(51.72)	
Marital status, n (%)				<0.001
Having a partner	5460(58.00)	4936(58.76)	524(51.63)	
Others	3955(42.00)	3464(41.24)	491(48.37)	
Smoking status, n (%)				<0.001
Never	5550(57.27)	4482(56.44)	668(64.17)	
Former	2209(22.97)	1958(22.64)	251(24.11)	
Current	1932(19.94)	1810(20.92)	122(11.72)	
Drinking status, n (%)				<0.001
Never	1476(15.67)	1270(15.11)	206(20.38)	
Former	1551(16.47)	1315(15.64)	236(23.34)	
Current	6390(67.86)	5821(69.25)	569(56.28)	
BMI, kg/m2	28.88 ± 7.08	28.79 ± 7.01	29.65 ± 7.64	<0.001
Energy intake, kcal/day	2135.67 ± 1022.22	2166.97 ± 1033.22	1875.72 ±884.37	<0.001
Albumin, g/L	42.78 ± 3.31	43.06 ± 3.19	40.45 ± 3.39	<0.001
Serum iron, umol/L	15.11 ± 6.41	15.66 ± 6.24	10.47 ± 5.88	<0.001
TC, mmol/L	4.90 ± 1.07	4.94 ± 1.07	4.56 ± 1.05	<0.001
Hemoglobin, g/dL	13.97 ± 1.52	14.29 ± 1.23	11.34 ± 1.06	<0.001
Folate, nmol/L	2.14 ± 13.76	2.03 ± 12.85	3.03 ± 19.76	0.027
CDAI	0.22 ± 4.13	0.31 ± 4.18	-0.59 ± 3.56	<0.001
Hypertension, n (%)	1,343(13.49)	1108(12.47)	235(21.96)	<0.001
Diabetes, n (%)	1742(17.50)	1430(16.10)	312(29.16)	<0.001
CHF, n (%)	304(3.23)	218(2.60)	86(8.5)	<0.001
CHD, n (%)	364(3.88)	290(3.46)	74(7.31)	<0.001

Continuous variables were presented as means ± standard deviation while categorical variables were presented as counts (percentages). Student's t test was used to analyze continuous variables and outcomes, and Chi-Square test was used for categorical variables. BMI=body mass index; TC=total cholesterol; CHF=congestive heart failure; CHD=coronary heart disease; and CDAI=composite dietary antioxidant index.

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Table 2: Association of the CDAI with anemia on a continuous or a categorical scale.

Model	Model 1		Model 2		Model 3	
	OR (95%CI)	*P*	OR (95%CI)	*P*	OR (95%CI)	*P*
Continuous						
CDAI	0.94(0.92,0.95)	<0.001	0.97(0.95,0.98)	<0.001	0.97(0.95,0.98)	0.001
Categories						
Q1	Ref	-	Ref	-	Ref	-
Q2	0.81 (0.68,0.95)	0.013	0.91(0.76,1.08)	0.261	0.89(0.74,1.07)	0.221
Q3	0.69 (0.58,0.82)	<0.01	0.85(0.71,1.02)	0.089	0.87(0.72,1.06)	0.161
Q4	0.54 (0.45,0.65)	<0.01	0.74(0.61,0.89)	0.002	0.74(0.60,0.91)	0.004
P for trend	<0.001		0.002		0.005	

Model 1 was adjusted for none; Model 2 was adjusted for age, sex and race; Model 3 was adjusted for age, sex, race, education, total cholesterol, Serum iron, BMI, folate, smoking, drinking, hypertension, congestive heart failure, myocardial infarction, diabetes mellitus. OR=odds ratio; CI=confidence interval; Ref=reference; BMI=body mass index; and CDAI=composite dietary antioxidant index.

Continuous variables were presented as means ± standard deviation while categorical variables were presented as counts (percentages). Student's t test was used to analyze continuous variables and outcomes, and Chi-Square test was used for categorical variables. BMI=body mass index; TC=total cholesterol; CHF=congestive heart failure; CHD=coronary heart disease; and CDAI=composite dietary antioxidant index.

Association between the CDAI and anemia

In this study, three models were constructed to examine the relationship between the CDAI and anemia (Table 2). In the multivariable analysis, the odds ratio (OR) and 95% confidence interval (CI) was 0.97 (0.95–0.98; $P = 0.001$) per 1 unit increase in the CDAI for the risk of anemia. On the categorical scale, as compared to the lowest CDAI quartile (Q1), the multivariable analysis revealed that the ORs for the risk of anemia were 0.89 [0.74-1.07] for Q2 ($P = 0.221$), 0.87 (0.72-1.06) for Q3 ($P = 0.161$), and 0.74 (0.60-0.91) for Q4 ($P = 0.004$). This association was strengthened with the incremental quartiles of the CDAI (P for trend=0.005).

Model 1 was adjusted for none; Model 2 was adjusted for age, sex and race; Model 3 was adjusted for age, sex, race, education, total cholesterol, Serum iron, BMI, folate, smoking, drinking, hypertension, congestive heart failure, myocardial infarction, diabetes mellitus. OR=odds ratio; CI=confidence interval; Ref=reference; BMI=body mass index; and CDAI=composite dietary antioxidant index.

Association between the components of the CDAI and anemia

We added a sensitive analysis on the association of the six components of the CDAI with anemia. As shown in Table 3, after adjusting for all variables, the association of the CDAI with anemia was driven by vitamins A (OR [95% CI]: 0.86 [0.76, 0.98]; $P = 0.020$), vitamins E (OR [95% CI]: 0.98 [0.98, 0.99]; $P = 0.020$), selenium(OR [95% CI]: 0.98 [0.97, 0.99]; $P = 0.006$) and Zinc (OR [95% CI]: 0.98 [0.96, 0.99]; $P = 0.004$). Further, restricted cubic spline suggested that the relationship between the CDAI and anemia was linear (P for nonlinearity = 0.154; Figure. 2A). At the same CDAI level, female have a higher risk of anemia than male (Figure. 2B).

Table 3: Association of six components of composite dietary antioxidant index and anemia.

	Model 1		Model 2		Model 3	
Components	OR (95%CI)	*P*	OR (95%CI)	*P*	OR (95%CI)	*P*
Vitamins A	0.81(0.72,0.93)	0.002	0.88(0.78,0.99)	0.037	0.86(0.76,0.98)	0.02
Vitamins C	1.00(0.99,1.01)	0.164	0.99(0.99,1.00)	0.135	1.00(0.99,1.01)	0.174
Vitamins E	0.98(0.97,0.99)	<0.001	0.98(0.97,0.99)	0.01	0.98(0.98,0.99)	0.02
Selenium	0.96(0.95,0.97)	<0.001	0.98(0.96,0.99)	0.001	0.98(0.97,0.99)	0.006
Carotenoid	0.99(0.98,0.99)	0.006	0.99(0.98,1.00)	0.045	1.00(0.99,1.02)	0.062
Zinc	0.96(0.94,0.97)	<0.001	0.98(0.97,0.99)	0.002	0.98(0.96,0.99)	0.004

Model 1 was adjusted for none; Model 2 was adjusted for age, sex and race; Model 3 was adjusted for age, sex, race, education, total cholesterol, Serum iron, BMI, folate, smoking, drinking, hypertension, congestive heart failure, myocardial infarction, diabetes mellitus. OR=odds ratio; CI=confidence interval; and BMI=body mass index.

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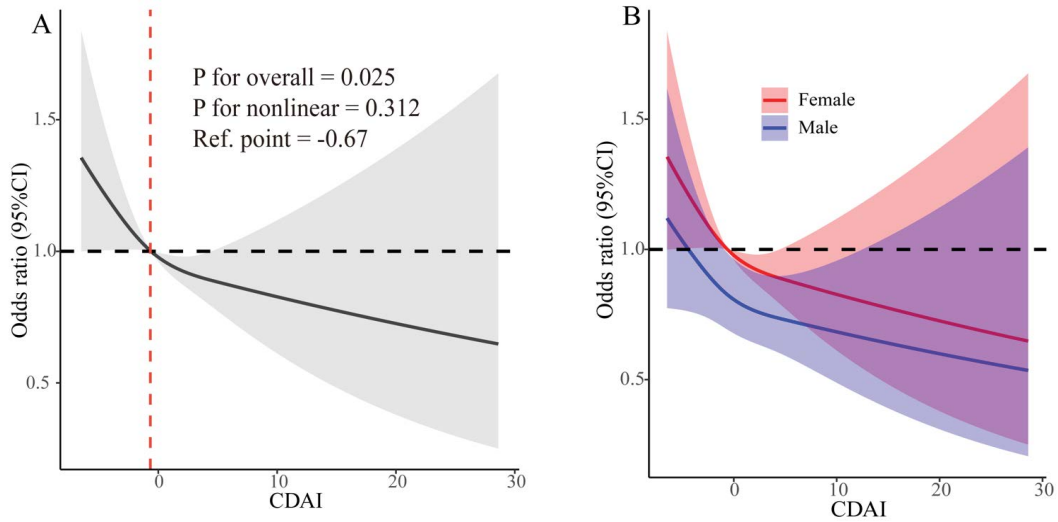

Figure 2: The dose-response relationship between the CDAI and anemia in total (A) and in female (red line and shadow) or male (purple line and shadow) (B). Models were adjusted for age, sex, race, education, total cholesterol, Serum iron, BMI, folate, smoking, drinking, hypertension, congestive heart failure, myocardial infarction, and diabetes mellitus. BMI=body mass index; CDAI=composite dietary antioxidant index.

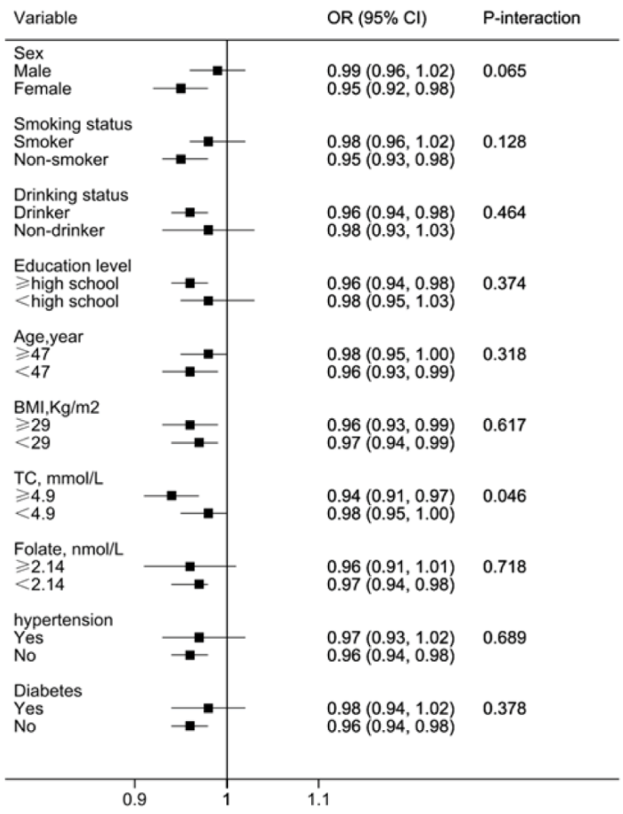

Figure 3: Subgroup analysis for the association between the CDAI and anemia. Models were adjusted for age, sex, race, education, total cholesterol, Serum iron, BMI, folate, smoking, drinking, hypertension, congestive heart failure, myocardial infarction, diabetes mellitus. The age, BMI, TC, folate were categorized into two subgroups by the median value. BMI=body mass index; TC=total cholesterol; and CDAI=composite dietary antioxidant index.

Subgroup analysis

To explore the interactive effects on the association, we conducted subgroup analysis through a series of strata variables and found that the association between the CDAI and anemia was consistent as shown in Figure. 3. The results indicated a stable negative association between the CDAI and anemia and the relationship showed no statistically significant differences in all groups (all *P* for interaction >0.05).

Roles of albumin played in the association between the CDAI and anemia

Table 4 showed the association between the CDAI and the albumin according to the linear regression. The results showed that higher CDAI were related to higher levels of albumin. Furthermore, we examined the mediating effects of albumin in the association between the CDAI and anemia as shown in Figure. 4, and the mediating effects of the albumin accounted for 42.91% of the total effects.

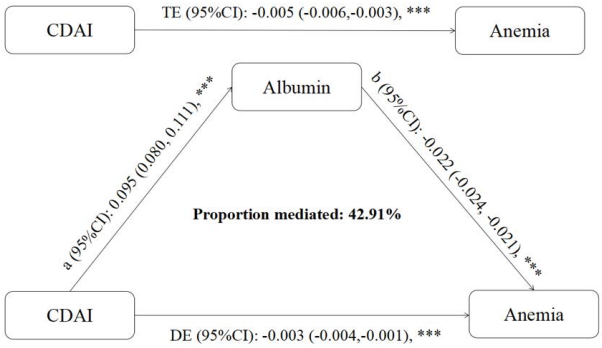

Figure 4: The association between CDAI and anemia mediated by albumin. TE=total effect; a*b=indirect effect; DE=direct effect. *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

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Table 4: Associations of CDAI with albumin.

	Model 1		Model 2		Model 3	
CDAI	β(95%CI)	*P*	β(95%CI)	*P*	β(95%CI)	*P*
Q1	Ref	-	Ref	-	Ref	-
Q2	0.30(0.11,0.48)	0.002	0.08(-0.09,0.25)	0.369	0.004(-0.16,0.16)	0.963
Q3	0.66(0.47,0.84)	<0.001	0.25(0.08,0.42)	0.005	0.160(0.001,0.326)	0.048
Q4	1.08(0.90,1.27)	<0.001	0.36(0.19,0.54)	<0.001	0.190(0.03,0.36)	0.023
P for trend	<0.001		<0.001		0.002	

Model 1 was adjusted for none; Model 2 was adjusted for age, sex and race; Model 3 was adjusted for age, sex, race, education, total cholesterol, Serum iron, BMI, folate, smoking, drinking, hypertension, congestive heart failure, myocardial infarction, diabetes mellitus. BMI=body mass index; OR=odds ratio; CI=confidence interval; and Ref=reference.

Discussion

The key findings in the current study revealed that the CDAI was negatively associated with anemia (OR (95% CI, 0.97(0.95-0.98)), mainly driven by vitamins A, vitamins E, Selenium, and Zinc. This negative correlation was linear in a dose-response manner; the serum albumin could mediate this association, accounting for 42.91% ($P < 0.001$) of the total effects, as confirmed by the mediator analysis. According to our findings, there was a negative correlation between CDAI and anemia. The CDAI is a composite score to reflect individuals' antioxidant status. The CDAI may theoretically hold greater significance than individual antioxidants, as it served as a key metric for dietary antioxidant capacity. Unlike single-dietary-antioxidant indicators, the CDAI better captured and reflected synergistic interactions among antioxidants, thereby facilitating a more comprehensive assessment of overall dietary quality [12]. Furthermore, oxidative stress in the body arises from the combined effects of multiple free radicals and oxidizing agents. Single-dietary-antioxidant indicators cannot fully reflect an organism's overall capacity to cope with oxidative stress. By integrating the intake of various dietary antioxidants, the CDAI provided a more accurate assessment of an individual's total dietary antioxidant potential and their ability to counteract cumulative oxidative load [13]. Accumulating evidence supports oxidative stress as a significant risk factor for anemia. The inhibition of oxidative stress could maintain redox homeostasis, thereby alleviating anemia [14]. Vitamin A promoted iron absorption and metabolism, thus playing a role in preventing anemia [15]. The current study showed that vitamin A supplementation reduced the risk of anemia by 14% (OR= 0.86, 95%CI: 0.76 to 0.98, $P = 0.020$), lower than the reported risk reduction by 26% (OR = 0.74, 95%CI: 0.66 to 0.82, $P< 0.001$) by a meta-analysis. The studied participants could explain this difference: the meta-analysis included children and pregnant women, who were more likely to be influenced by vitamin A and the current study, however, included only adults, who were less likely influenced by vitamin A [16]. A positive relationship between the supplementation of vitamin E and elevated hemoglobin levels was revealed in mildly anemic adults (β = 0.983) [17], supporting our findings that vitamin E can reduce the risk of anemia. Vitamin E was considered to be an important lipophilic antioxidant in biological membranes, which can eliminate free radicals and act as a chain-breaking antioxidant. Severe vitamin E deficiency can lead to impaired immune response and free radical-induced hemolytic anemia [18]. Another study showed that anemia in patients with eating disorder was substantially correlated with zinc deficiency [19], which in turn supported that zinc could reduce the risk of anemia in the current study. What zinc supplementation can promote erythropoiesis confirmed in anemic rats may be a plausible explanation [20-22]. Zinc steady-state imbalance can cause a series of adverse reactions, such as increased oxidative stress, erythropoietin resistance and atherosclerosis [23]. The current study reported a significant, albeit slight, association of selenium with decreased risk of anemia (OR (95%CI), 0.98(0.98-0.99)), similar to previous reports (OR=0.97, 95%CI: 0.96-0.98, $P < 0.001$) [24]. The binding of selenium to selenoproteins prevented peroxidation and oxidative cell damage [25]. Previous studies have shown that selenium deficiency leads to macrocytic anemia, which was significantly improved by selenium supplementation [26]. However, these studies have focused exclusively on specific antioxidants and have not examined the potential synergistic effects between various antioxidant nutrients. The CDAI had a comprehensive effect on an individual's pro-antioxidant status and highlights the advantages of a comprehensive evaluation of antioxidant exposure.

Our findings indicated that after adjusting for multiple confounding factors, the association between vitamin C and carotenoids and anemia remained statistically insignificant. Possible explanations was that heterogeneity within the study population, including various types of anemia such as iron-deficiency and chronic disease-related anemia, may have diluted the effects of these components. Nevertheless, the significant negative correlation with CDAI overall suggested

that vitamin C and carotenoids may contribute to anemia prevention through synergistic interactions with vitamins A, E, selenium, and zinc. As part of our investigation, we further explored the potential pathways that linked the CDAI to anemia. We found that the albumin mediated the relationship between the CDAI and anemia. Albumin, a major plasma protein synthesized by the liver, was influenced by dietary intake, particularly of proteins and micronutrients, and was commonly used as a biomarker of nutritional status. The CDAI, which reflected the intake of six dietary antioxidants (vitamins A, C, E, carotenoids, selenium, and zinc), may improve nutritional status by supporting metabolic processes, reducing oxidative stress, and enhancing protein synthesis, including that of albumin. Higher albumin levels were associated with reduced anemia risk, potentially due to the biochemical structure of albumin that made it a good indicator of oxidative stress in the circulatory system. As an extracellular antioxidant and major transport protein, albumin inhibited free radicals (oxidative stress) and alleviated anemia in patients through its antioxidant properties [27]. Thus, albumin may mediate the CDAI-anemia relationship by acting as both a nutritional marker and a functional contributor to redox homeostasis. A protective relationship between the albumin and anemia was found in our study, which was in agreement with the findings of a case-control study [28] and of a study of 80 years old and centenarians that a higher serum albumin concentration could reduce the risk of anemia [29].

There are several limitations to this study. First, due to its cross-sectional design, our findings should be considered as hypothesis-generating and were unable to make any causal inferences. Second, despite adjustment for many confounders, possible unmeasured confounders cannot be completely excluded. Third, the CDAI construction, which assessed dietary antioxidant capacity, considering only essential nutrients and carotenoids, may have overlooked other beneficial nutrients.

Conclusion

Our study highlighted a negative linear association between the CDAI and anemia in the general adults. Intake of the six dietary antioxidants composing the CDAI could help to prevent the occurrence of anemia.

Acknowledgments

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Conflicts of Interest

The authors declare no conflict of interest.

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