



Original Article

Dietary Antioxidant Index (DAI) and Its Association with Ischemic Heart Disease: Evidence from a Case-Control Study

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Article info

Article History:

Received: March 13, 2025

Revised: November 7, 2025

Accepted: July 2, 2026

published: September 27, 2026

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Abstract

Introduction: Ischemic heart disease (IHD) is a chronic condition associated with various cardiovascular complications. Oxidative stress was reported to play a prominent role in the development of IHD. The Dietary Antioxidant Index (DAI) measures dietary antioxidant capacity, yet its association with IHD odds remains unclear. This study aimed to investigate the link between IHD odds and DAI in Iranian adults.

Methods: This case-control study included 443 cases with stable IHD and 443 controls without IHD. Dietary intake was assessed using a validated semi-quantitative food frequency questionnaire (FFQ) and Nutritionist IV software was applied to assess the intake of different types of dietary antioxidants. DAI was then calculated using Wright's method. The multivariable logistic regression was used for statistical analysis and the adjustment of the confounders.

Results: Patients with IHD had lower intakes of several micronutrients, including potassium, sodium, selenium, vitamin A, vitamin E, vitamin C, vitamin K, vitamin B12, lycopene, and choline. The cases had significantly lower total DAI scores compared to the controls. A significant inverse association was observed between IHD and total DAI score (OR=0.948, 95%CI: 0.912-0.985, $P=0.006$) and vitamin C (OR=0.803, 95%CI: 0.672-0.959, $P=0.016$). These associations remained significant after adjustment for energy, age, gender, marital status, job, physical activity, BMI, smoking, and alcohol consumption.

Conclusion: Our findings suggest a significant inverse relationship between IHD and dietary antioxidant index may be associated with lower odds of IHD. Dietary antioxidant assessment may be beneficial as part of lifestyle counseling in high-odds patients.

Keywords: Ischemic heart disease, Dietary antioxidant intake, DAI, Diet, IHD

Introduction

Ischemic heart disease (IHD) is a chronic inflammatory condition and the leading cause of mortality worldwide, responsible for approximately one-third of global

deaths ^{1,2}. The economic burden of IHD is substantial, contributing significantly to healthcare costs associated with cardiovascular diseases. IHD can lead to various cardiovascular events, such as stroke, heart failure, and



myocardial infarction.³ The age-standardized prevalence of IHD in Iran was 5143.08 per 100,000 people which was 7.5% higher than that in 1990⁴. Several risk factors including hypertension, hyperlipidemia, hyperglycemia, and obesity are strongly associated with IHD.⁵⁻⁷ Additionally, some unhealthy lifestyle factors, such as smoking, poor diet, and physical inactivity, can accelerate the progress of atherosclerosis.

Atherosclerosis, as the main precursor to IHD, is a progressive and chronic condition characterized by the accumulation of fatty deposits, cholesterol, cellular debris, and calcium within arterial walls, leading to plaque formation and restricted blood flow. Subsequently, the affected arteries become less flexible and have reduced capacity for blood flow, eventually leading to IHD.⁸ In addition, if atherosclerosis in the coronary arteries develops to the point where a plaque ruptures or a blood clot forms at the site of the plaque, it can thoroughly block blood flow to a portion of the heart, causing a heart attack.⁹ Dietary patterns may have a substantial effect on the overall burden of IHD. Numerous epidemiological studies have consistently demonstrated that diets higher in saturated fatty acids, trans fatty acids, refined sugars and processed food are associated with an increased odds of IHD, while diets rich in fruits, vegetables and whole grains provides a protective effect.^{10,11}

Oxidative stress and inflammation play a prominent role in the development of atherosclerosis and IHD.¹² Dietary antioxidants are protective components against oxidative stress and inflammation and may decrease the progress of atherosclerosis. The Dietary Antioxidant Index (DAI) is a composite measure designed to quantify dietary antioxidant intake, including vitamins, zinc, selenium, and carotenoids.^{13,14} Researchers are recently employing DAI to investigate the association between the antioxidant capacity of diets and chronic diseases such as cardiovascular diseases, cancer, and diabetes, among others.¹⁵⁻¹⁷ This contributes to a better comprehension of the overall potential role of diet in disease prevention and management. Furthermore, DAI can guide dietary recommendations to enhance antioxidant capacity and improve overall health.¹⁸

DAI has been linked to various diseases, including cancer, depression, osteoporosis, and papillomavirus infection.¹⁹⁻²² However, the connection between DAI and IHD remains underexplored. Despite growing evidence on the role of oxidative stress in the pathogenesis of IHD, few studies have utilized composite dietary antioxidant measures such as the DAI in this context. Most previous research has focused on individual antioxidants, without accounting for their synergistic dietary effects. Therefore, our research aimed to investigate the relationship between DAI and IHD odds, addressing a critical gap in the literature and offering insights into potential dietary strategies for cardiovascular disease prevention. This study is among the first to assess the association between the DAI and IHD in an Iranian population, which applies DAI as a practical and culturally adaptable tool for evaluating antioxidant intake in cardiovascular risk assessment.

Materials and Methods

This was a case-control study conducted on 886 patients with stable IHD and control participants without IHD. The case group (n=443) was selected consecutively from patients referred to Shahid Rajaei Hospital and Tehran Heart Center with a confirmed diagnosis of stable IHD, based on clinical evaluations by attending physicians. Diagnoses were based on clinical evaluation by a cardiologist and confirmed using standard diagnostic tools, including electrocardiography (ECG), echocardiography, exercise stress testing, and/or coronary angiography, in accordance with the ESC guidelines for chronic coronary syndromes. Diagnoses were confirmed and recorded in the patients' medical records prior to recruitment.

The control group (n=443) included hospital staff and individuals attending the hospital for conditions unrelated to cardiovascular disease. Controls were screened for absence of cardiovascular disease based on medical history. Both written and oral explanations were provided to each individual about the purpose and method of conducting the study. The inclusion criteria for cases were adults aged 40-80 years, written consent to participate, and diagnosed with stable IHD. Controls were matched for age (± 5 years) and sex. Exclusion for both groups included a history of major chronic diseases including mental disorders, cancer, and chronic diseases (e.g., renal failure, liver disease), extreme energy intake (< 800 or > 4200 kcal/day), taking antioxidant supplements, or/and incomplete questionnaire responses.

General information, medical history of patients, data related to anthropometric indices, physical activity, and food consumption were collected by trained interviewers. General information such as age, gender, medical history, current health status, drug or supplement usage, cigarette smoking or tobacco, and alcohol and physical activity were self-reported. Weight was measured using Seca digital scale with an accuracy of 0.1 kg with minimal clothing. Height was measured in a standing position without shoes and with a tape measure with an accuracy of 1 cm. After that, BMI was calculated by dividing weight (kg) by the square of height (meters).

Dietary intake was evaluated using a semi-quantitative food frequency questionnaire (FFQ) with 168 items.²³ Antioxidant intake was analyzed with Nutritionist IV software, and the Dietary Antioxidant Index (DAI) was calculated following Wright's method. The DAI score was standardized by comparing individual nutrient intakes (vitamins A, C, E, selenium, zinc, and manganese) to global averages of our own study population and dividing by their standard deviations, as reported in published literature.²⁴ DAI was calculated as both crude and energy-adjusted forms and applied as continuous variables. The International Valid Physical Activity Questionnaire (IPAQ) – short form was used to assess physical activity and was expressed as MET- min/week.²⁵

Statistical Analysis

Missing data were minimal (less than 5% for all variables) and were handled using multiple imputation or mean

substitution, depending on the nature and extent of the missingness. We assessed the normality of continuous variables using the Shapiro–Wilk test. Additionally, Q–Q plots were visually inspected to confirm the distribution of the data. Data analysis was performed using SPSS software version 25 and $P < 0.05$ was considered as significant in all analyses. Descriptive statistics, including, mean and standard deviation were used to describe quantitative data, while number and percentage were used to describe qualitative data. To analyze the association between IHD and DAI and to adjust the confounding variables, multivariable logistic regression modeling was used, where the existence of the disease was entered as a dependent variable, and the antioxidant index of the diet was entered as an independent variable. Different models of logistic regression were as Crude (Model 1), adjusted for age and gender (Model 2), further adjusted for marital status and job (Model 3), and further adjustment for physical activity, BMI, smoking, and using alcohol (Model 4). Potential interactions between key variables were tested using interaction terms in the regression models.

Results

Table 1 presents the demographic and clinical characteristics of the study participants. The prevalence of hypertension ($P < 0.001$), diabetes ($P < 0.001$), and higher systolic and diastolic blood pressure ($P = 0.002$ and $P = 0.016$, respectively) were significantly greater in the

IHD group compared to controls. Smoking and alcohol consumption were more frequent in the case group, though these differences were not statistically significant. There were no significant differences in age, sex, height, weight, BMI, or physical activity levels (MET) between the case and control groups.

Table 2 compares dietary intake between the groups. Patients with IHD exhibited notably lower intake levels of several key micronutrients, including potassium, sodium, selenium, vitamin A, vitamin E, vitamin C, vitamin K, vitamin B12, lycopene, and choline, compared to the control group.

Additionally, patients with IHD had lower total DAI scores (-0.35 ± 4.45 vs. 0.34 ± 5.11 , $P = 0.031$), as well as lower DAI scores for vitamins A (-0.09 ± 0.90 vs. 0.08 ± 1.07 , $P = 0.008$), C (-0.11 ± 0.92 vs. 0.11 ± 1.05 , $P = 0.001$), E (-0.06 ± 0.95 vs. 0.06 ± 1.03 , $P = 0.043$), and selenium (-0.07 ± 0.93 vs. 0.07 ± 1.05 , $P = 0.035$). Notably, all differences in DAIs remained significant after adjusting for calorie intake, except for vitamin E (Table 3).

The association between the DAI and IHD odds was investigated through a series of logistic regression models (Table 4). In the crude model (Model 1), a significant inverse association was observed between total DAI score and the odds of IHD (OR = 0.948, 95%CI: 0.914–0.984, $P = 0.005$). This association remained significant after adjusting for age and gender in Model 2 (OR = 0.952, 95%CI: 0.917–0.988, $P = 0.009$), and further after adjusting

Table 1. General Characteristics of Participants in Relation to Ischemic Heart Disease (IHD) and the Dietary Antioxidant Index (DAI)

Variables		Controls (n=443)	Cases (n=443)	P
Age		54.67 ± 8.78	55.59 ± 8.13	0.105
gender	Males	204 (45%)	212 (47.8%)	0.420
	Female	249(55.0%)	231 (52.14%)	0.420
Marital	Unmarried	2 (0.4%)	3 (0.7%)	0.443
	Married	412 (90.9%)	392 (90.1%)	0.433
	Widowed	33 (7.3%)	38(8.7%)	0.433
	Divorced	6 (1.3%)	2 (0.5%)	0.433
Height		161.027 ± 9.05	161.483 ± 9.34	0.462
Weight		72.64 ± 12.93	74.38 ± 13.58	0.051
BMI		28.061 ± 4.75	28.54 ± 4.75	0.135
SBP		116.64 ± 15.77	120.18 ± 17.32	0.002
DBP		72.92 ± 9.59	74.52 ± 9.96	0.016
MET (MET- min/week)		38.00 ± 8.44	37.53 ± 7.83	0.391
Smoke Cigarette	No	87 (19.6%)	108 (24.9%)	0.062
	Yes	357 (80.4%)	326 (75.1%)	0.062
Use Alcohol	No	399 (89.9%)	396 (91.2%)	0.492
	Yes	45(10.1%)	38 (8.8%)	0.492
Has Diabetes	No	371 (83.4%)	309 (69.7%)	<0.001
	Yes	74 (16.6%)	134 (30.3%)	<0.001
Has Hypertension	No	322 (72.4%)	227 (51.2%)	<0.001
	Yes	123 (27.6%)	216 (48.8)	<0.001

Data are presented as mean ± SD for continuous variables and n (%) for categorical variables. Independent samples t-test was used to compare continuous variables between cases and controls, and the chi-square test was used to compare categorical variables. Fisher's exact test was used when the expected cell count was insufficient. BMI, body mass index; IHD, ischemic heart disease; MET, metabolic equivalent of task; SBP, systolic blood pressure; DBP, diastolic blood pressure. A P value < 0.05 was considered statistically significant.

Table 2. Dietary Intake of Participants Concerning IHD and DAI

Variables	Controls (n=443) (Mean±SD)	Cases (n=443) (Mean±SD)	P
Protein (g/d)	76.36±28.51	74.90±25.22	0.421
Total lipid (fat) (g)	65.26±28.80	62.02±25.29	0.076
Carbohydrate (g)	394.54±137.04	396.96±135.19	0.792
Energy (kcal)	2415.91±814.51	2396.41±764.57	0.714
Starch (g)	11.47±15.03	10.19±10.58	0.143
Sucrose (g)	45.52±30.26	43.53±32.08	0.345
Glucose (dextrose) (g)	29.36±17.59	27.15±15.50	0.048
Fructose (g)	34.17±19.76	31.60±17.87	0.043
Lactose (g)	4.31±4.93	4.52±5.46	0.547
Maltose (g)	0.49±0.66	0.45±0.43	0.305
Alcohol (g)	0.034±0.074	0.027±0.07	0.167
Caffeine (mg)	185.26±119.40	198.60±140.36	0.130
Sugar total (g)	142.40±67.81	133.43±62.78	0.042
Galactose (g)	0.210±0.218	0.185±0.178	0.070
Fiber total (g)	28.00±11.71	27.11±10.50	0.236
Calcium (mg)	897.77±374.76	892.94±328.55	0.839
Iron (mg)	13.09±5.10	12.83±4.47	0.409
Magnesium (mg)	344.86±124.12	334.39±109.86	0.185
Phosphorus (mg)	1182.96±451.40	1155.34±388.03	0.330
Potassium (mg)	3842.83±1524.57	3626.15±1269.19	0.022
Sodium (mg)	4821.69±2583.03	4322.35±1920.05	0.001
Zinc (mg)	9.911±3.80	9.605±3.26	0.200
Copper (mg)	1.82±0.72	1.76±.059	0.147
Fluoride (µg)	3460.59±2217.78	3706.40±2607.49	0.133
Manganese (mg)	5.47±1.94	5.63±2.05	0.263
Selenium (µg)	53.58±29.40	49.64±25.97	0.035
Vitamin A (IU)	9223.37±6784.10	8792.40±6316.63	0.029
Retinol (µg)	330.84±271.05	298.41±209.13	0.047
Vitamin A (mcg)	765.22±478.75	686.02±402.67	0.008
Beta Carotene (µg)	4629.12±3415.45	4118.66±3151.01	0.021
Alpha Carotene (µg)	815.30±1009.53	745.82±1018.68	0.310
Vitamin E (alpha-tocopherol) (mg)	7.42±3.65	6.94±3.37	0.043
Vitamin D (IU)	42.09±30.86	38.76±25.62	0.082
Cryptoxanthin beta (µg)	370.55±354.12	329.45±305.57	0.065
Lycopene (µg)	14234.50±8426.72	12991.41±7281.56	0.019
Lutein + Zeaxanthin (µg)	2441.00±1690.05	2319.30±1423.24	0.248
Vitamin C (total ascorbic acid) (mg)	163.65±101.90	141.40±88.74	0.001
Thiamin (mg)	1.55±0.596	1.56±0.598	0.786
Riboflavin (mg)	1.73±0.69	1.67±0.60	0.202
Niacin (mg)	17.80±7.10	17.20±6.22	0.186
Pantothenic Acid (mg)	5.88±2.14	5.67±1.88	0.117
Vitamin B6 (mg)	10.03±6.03	9.77±5.47	0.500
Folate total (µg)	380.35±153.52	368.57±140.79	0.236
Vitamin B12 (µg)	6.15±4.36	5.62±3.34	0.040
Choline total (mg)	259.73±108.03	245.37±98.67	0.040
Vitamin K (µg)	180.77±137.65	164.20±100.03	0.041
Cholesterol (mg)	252.11±126.46	238.04±114.66	0.084
Fatty Acid total trans (g)	0.25±0.30	0.20±0.22	0.017
Saturated fatty acids (g)	25.81±13.15	24.17±11.18	0.047

Table 2. Continued.

Variables	Controls (n=443) (Mean±SD)	Cases (n=443) (Mean±SD)	P
Fatty acids total monounsaturated (MUFA) (g)	20.47±9.44	19.44±8.58	0.092
Fatty acids total polyunsaturated (PUFA) (g)	11.64±5.09	11.27±4.87	0.271
n3 total (g)	0.034±0.027	0.031±0.020	0.040
Docosahexaenoic acid (DHA) (g)	0.014±0.019	0.011±0.012	0.011
Eicosapentaenoic acid (EPA) (g)	0.45±0.30	0.46±0.37	0.704
Alpha Linolenic acid (ALA) (g)	7.22±4.63	7.10±4.21	0.688
N-6 total (g)	4.65±3.49	4.62±3.44	0.903

Data are presented as mean±SD. Independent samples t-test was used to compare dietary intake variables between cases and controls. IHD, ischemic heart disease; DAI, Dietary Antioxidant Index. A *P* value<0.05 was considered statistically significant.

Table 3. Comparison of DAI and Energy-Adjusted DAI between IHD Patients and Controls

Variables	Controls (n=443) (Mean±SD)	Cases (n=443) (Mean±SD)	P
Dietary Antioxidant Index (DAI)			
DAI Vitamin A	0.08±1.07	-0.09±0.90	0.008
DAI Vitamin C	0.11±1.05	-0.11±0.92	0.001
DAI Vitamin E	0.06±1.03	-0.06±0.95	0.043
DAI Selenium	0.07±1.05	-0.07±0.93	0.035
DAI Manganese	-0.03±0.97	0.039±1.03	0.263
DAI Zinc	0.04±1.07	-0.04±0.91	0.200
Total DAI Score	0.34±5.11	-0.35±4.45	0.031
Energy-Adjusted DAI Scores			
DAI Vitamin A	0.09±1.01	-0.10±0.97	0.003
DAI Vitamin E	0.06±0.98	-0.06±1.03	0.072
DAI Vitamin C	0.11±1.05	-0.11±0.93	0.001
DAI Selenium	0.07±1.00	-0.07±0.98	0.024
DAI Manganese	-0.055±0.99	0.046±1.00	0.132
DAI Zinc	0.044±1.05	-0.05±0.95	0.163
Total DAI Score	0.33±3.75	-0.35±3.58	0.006

Data are presented as mean±SD. Independent samples t-test was used to compare DAI scores between cases and controls. DAI, Dietary Antioxidant Index; IHD, ischemic heart disease. A *P* value<0.05 was considered statistically significant.

Table 4. Association between IHD and Adjusted DAI across Different Models

Variables	Model 1		Model 2		Model 3		Model 4	
	OR (CI95%)	P	OR (CI95%)	P	OR (CI95%)	P	OR (CI95%)	P
DAI Total	0.948 (0.914-0.984)	0.005	0.952(0.917-0.988)	0.009	0.951(0.917-0.987)	0.009	0.948(0.912-0.985)	0.006
DAI Vitamin A	0.915(0.771-1.087)	0.312	0.916(0.771-1.088)	0.317	0.919(0.773-1.092)	0.336	0.919(0.772-1.094)	0.341
DAI Vitamin E	1.068(0.900-1.266)	0.452	1.080(0.909-1.284)	0.382	1.083(0.910-1.287)	0.370	1.069(0.897-1.273)	0.456
DAI Vitamin C	0.811(0.680-0.967)	0.019	0.813(0.682-0.969)	0.021	0.809(0.678-0.965)	0.018	0.803(0.672-0.959)	0.016
DAI Selenium	0.866(0.707-1.061)	0.166	0.874(0.710-1.076)	0.206	0.873(0.709-1.075)	0.201	0.871(0.706-1.073)	0.194
DAI Manganese	1.083(0.946-1.241)	0.248	1.087(0.949-1.247)	0.229	1.083(0.945-1.243)	0.252	1.066(0.928-1.225)	0.367
DAI Zinc	1.048(0.865-1.271)	0.629	1.042(0.860-1.263)	0.675	1.042(0.859-1.263)	0.677	1.051(0.865-1.276)	0.619

Multivariable logistic regression analysis was used to estimate odds ratios (ORs) and 95% confidence intervals (CIs) for the association between DAI scores and IHD. Model 1, crude model; Model 2, adjusted for age and gender; Model 3, additionally adjusted for marital status and job status; Model 4, additionally adjusted for physical activity, BMI, smoking, and alcohol consumption. DAI, Dietary Antioxidant Index; IHD, ischemic heart disease; OR, odds ratio; CI, confidence interval; BMI, body mass index. A *P* value<0.05 was considered statistically significant.

for marital status and job status in Model 3 (OR=0.951, 95%CI: 0.917-0.987, *P*=0.009). The inverse association persisted even after additional adjustments for physical activity, BMI, smoking, and alcohol consumption in Model 4 (OR=0.948, 95%CI: 0.912-0.985, *P*=0.006). These findings indicate that higher DAI scores are consistently associated with a reduced odds of IHD, independent of several confounding factors. The odds ratio of 0.948 for

total DAI in the fully adjusted model suggests that for each one-unit increase in the DAI, the odds of having IHD decrease by approximately 5.2%. Regarding individual antioxidant components of DAI (Table 5), a significant inverse association was specifically identified between vitamin C intake and IHD odds (OR=0.803, 95%CI: 0.672-0.959, *P*=0.016) in the fully adjusted model. The odds ratio of 0.803 for vitamin C suggests that for each unit increase

Table 5. Association between IHD and DAI of nutrients

Variables	Model 1		Model 2		Model 3		Model 4	
	OR (CI95%)	P	OR (CI95%)	P	OR (CI95%)	P	OR (CI95%)	P
DAI Total	0.971 (0.944-0.998)	0.037	0.967(0.938-0.997)	0.032	0.968(0.939-0.998)	0.036	0.966(0.937-0.997)	0.033
DAI Vitamin A	0.945 (0.757-1.180)	0.618	0.947 (0.757–1.185)	0.636	0.954(0.762-1.193)	0.678	0.958(0.763-1.202)	0.709
DAI Vitamin C	0.747(0.603-0.926)	0.008	0.748(0.604-0.927)	0.008	0.743(0.600-0.921)	0.007	0.737(0.594-0.915)	0.006
DAI Vitamin E	1.078(0.858-1.354)	0.519	1.096(0.870-1.381)	0.436	1.100(0.873-1.386)	0.419	1.084(0.858-1.369)	0.500
DAI Selenium	0.824(0.634-1.072)	0.149	0.835(0.640-1.088)	0.182	0.832(0.638-1.085)	0.174	0.823(0.629-1.076)	0.154
DAI Manganese	1.227(1.026-1.468)	0.025	1.221(1.021-1.461)	0.182	1.219(1.018-1.459)	0.031	1.197(0.998-1.435)	0.052
DAI Zinc	1.078(0.796-1.461)	0.626	1.048(0.767-1.432)	0.770	1.052(0.769-1.440)	0.749	1.086(0.791-1.491)	0.609

Multivariable logistic regression analysis was used to estimate odds ratios (ORs) and 95% confidence intervals (CIs) for the association between individual DAI nutrient components and IHD. Model 1, crude model; Model 2, adjusted for age and gender; Model 3, additionally adjusted for marital status and job status; Model 4, additionally adjusted for physical activity, BMI, smoking, and alcohol consumption. DAI, Dietary Antioxidant Index; IHD, ischemic heart disease; OR, odds ratio; CI, confidence interval; BMI, body mass index. A *P* value<0.05 was considered statistically significant.

in vitamin C, the odds of IHD decrease by approximately 20%. While other individual antioxidants (e.g., Vitamin A, Vitamin E, and Zinc) were also analyzed, their associations with IHD did not reach statistical significance in the fully adjusted models. The associations were also confirmed to remain significant in the energy-adjusted form of DAI.

Discussion

This study revealed that Patients with IHD exhibited lower dietary antioxidant intake compared to the controls, and crucially, highlighting a significant inverse association between dietary antioxidants scores and the odds of IHD. These findings, which remained robust after adjusting for multiple confounders, indicate that higher DAI scores correlate with a reduced odds of IHD, suggesting a potential association rather than a causal protective effect against IHD.

A sufficient intake of dietary antioxidants may play a role in preventing or postponing the development of oxidative stress-related disorders in individuals with cardiovascular diseases. For instance, vitamin C, which is a potent water-soluble antioxidant, reduces LDL oxidation and vitamin E protects cell membrane from oxidative damage.²⁶ The review highlighted the role of vitamin E, vitamin C, flavonoids, and carotenoids as the most potent dietary antioxidants for CVD prevention.²⁷ Also, a systematic review and meta-analysis by Ju et al. demonstrated that selenium supplementation led to increased levels of glutathione peroxidase and a reduction in serum C-reactive protein, indicating a potential positive impact on inflammation and oxidative stress in patients with coronary heart disease (CHD).²⁸ Furthermore, a systematic review and dose-response meta-analysis conducted by Anue et al. revealed that higher dietary intake and/or blood concentrations of antioxidants derived from fruits and vegetables, such as α-tocopherol, carotenoids, and vitamin C, were associated with a reduced odds of mortality from CVD.²⁹ These results advocate for CVD prevention strategies that prioritize increased consumption of fruits and vegetables over the use of antioxidant supplements.²⁹

While many previous studies have focused on the effects of antioxidant supplements, our findings highlight the importance of dietary antioxidants obtained

naturally through food sources.³⁰ Whole foods contain a complex matrix of nutrients and bioactive compounds that may act synergistically to reduce oxidative stress and inflammation.³¹ Promoting the consumption of antioxidant-rich foods such as fruits, vegetables, nuts, and whole grains may contribute to dietary strategies for cardiovascular odds reduction, although causal inference cannot be established. Public health strategies should therefore prioritize improving dietary quality rather than relying solely on supplementation.³²

The novelty of this study lies in its investigation of the association between the DAI and IHD odds within a large Iranian population, offering a unique perspective that integrates the effects of multiple dietary components. By utilizing the DAI, our research contributes to the existing literature by providing a more nuanced understanding of how overall dietary antioxidant capacity, derived from real-world food consumption patterns, relates to IHD, moving beyond the scope of single nutrient analysis or supplementation trials. However, this study has some limitations. First, its case-control design inherently limits the ability to establish causality. Second, reliance on a self-reported Food Frequency Questionnaire (FFQ) for dietary data introduces potential for recall bias. Third, the study population was confined to a specific region, which may limit the generalizability of these findings to more diverse global populations. The use of hospital-based controls may introduce selection bias. Patients with IHD may have modified their diet following diagnosis. Lastly, while the study’s sample size is substantial, the focus on a specific demographic (ages 40-80) in designated hospitals may restrict the applicability of the findings to younger populations or those outside the selected healthcare settings.

Conclusion

This study demonstrates that higher dietary antioxidant capacity, as measured by the DAI, is significantly associated with a reduced odds of IHD. These findings highlight the importance of dietary patterns rich in antioxidant as a protective strategy against cardiovascular disease. These findings suggest that assessing dietary antioxidant intake using the DAI may help identify individuals at higher

odds for IHD. Incorporating antioxidant-rich foods into dietary recommendations could support cardiovascular prevention strategies. Clinicians may consider dietary antioxidant assessment as part of lifestyle counseling in high-risk patients. Future research should focus on prospective cohort studies, include diverse populations, and incorporate biomarker assessments to deepen our understanding of the relationship between dietary antioxidant and IHD prevention.

Acknowledgments

We thank all the participants in this study for their good cooperation. This paper was taken from the approved research project of Shahid Beheshti University of Medical Sciences, Tehran, Iran. Artificial intelligence (AI)-based tools were used solely for language refinement. All scientific content, data analysis, interpretation of results, and final manuscript revisions were performed and verified by the authors.

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Competing Interests

The authors declare that they have no competing interests.

Ethical Approval

The ethics committee of Shahid Beheshti University of Medical Sciences, Tehran, Iran approved the study (Ethics Code: IR.SBMU.NNFTRI.REC.1400.030). All study participants signed a written informed consent.

Funding

Funding for this study was provided by Shahid Beheshti University of Medical Sciences, Tehran, Iran (Code 22450).

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