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Association of dietary antioxidant index with body mass index in adolescents

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Abstract

Background: Dietary antioxidants may decrease body fat through reduction of oxidative stress. This study aimed to examine the association between dietary antioxidant index (DAI) and body mass index (BMI) in adolescent boys.

Methods: In this cross-sectional study, 593 adolescent boys aged 12–16 years were randomly selected and were divided into two groups of overweight and non-overweight individuals. Data on physical activity and anthropometric measurements were collected. Dietary intake was assessed using 168-item semi quantitative food frequency questionnaire and the DAI score was calculated to measure the antioxidant capacity of the diet.

Results: The overweight adolescents had higher intake of energy (2490.55 ± 632.49 vs. 2354.33 ± 632.64 kcal/d, $p = 0.01$), carbohydrate (290.21 ± 71.41 vs. 272.93 ± 79.22 g/d, $p = 0.01$), fat (111.51 ± 40.76 vs. 104.51 ± 35.56 g/d, $p = 0.04$), calcium (811.70 ± 283.70 vs. 741.06 ± 251.17 g/d, $p = 0.003$), and vitamin D (1.41 ± 1.17 vs. 1.18 ± 1.19 µg/d, $p = 0.031$) in comparison with normal weight adolescents. The DAI had an inverse association with BMI after adjustment for age and caloric intake (OR: 0.85, 95% CI: 0.76–0.96, $p = 0.009$). Additional adjustment for dietary intake of vitamin A, vitamin E, vitamin C, zinc, manganese, and selenium did not change the results.

Conclusion: The results of the study showed that following a diet rich in antioxidants may be effective in preventing obesity in adolescent boys. Further longitudinal studies are needed to confirm these finding and to determine the underlying mechanisms.

KEYWORDS
adolescents, boys, dietary antioxidant, obesity

1 | INTRODUCTION

Childhood and adolescence obesity has been increased in recent decades worldwide,[1] which may lead to higher risk of obesity in adulthood.[2] Obesity in adolescence is considered as a risk factor for a broad range of chronic diseases such as type 2 diabetes, cancer, metabolic syndrome, cardiovascular disease, and hypertension.[3] The prevalence of obesity in Iranian adolescents was about 16% in 2016.[4] The most common criterion used for obesity assessment is body mass index (BMI)[5] and higher BMI of adolescents was reported to be associated with cardiovascular and all-cause mortality after 40-year follow-up.[6] The development of obesity in children and adolescents is caused by a variety of factors such as physical inactivity and poor dietary habits.[7–9] Some studies indicated that male students have higher prevalence of obesity in Iran[10,11] and consume more fast foods and less antioxidants rich sources such as fruits and vegetables than female students.[12]

Furthermore, antioxidant properties of diet may be involved in the development of obesity.[13] A recent study found that the improvement of antioxidant defense system may cause higher protection against overweight and obesity.[14] Reactive oxygen species may play a direct role in adipogenesis and oxidative stress can modulate different factors involved in obesity including genetics, sleep, gut microbiome, insulin, ghrelin, inflammation, adipokines, leptin, stress, hypothalamic-pituitary-adrenal axis, and the hypothalamus.[15] Furthermore, it was reported that the level of dietary antioxidants such as vitamin C and vitamin E were in appropriate range only in young girls with normal weight.[16] Lower levels of total antioxidant capacity (TAC) was reported to be significantly associated with higher weight and obesity in childhood and adolescence.[17] Some studies reported that lower levels of selenium and vitamin C were associated with obesity through reducing adipose tissue fibrosis.[18–20] However, contradictory results were reported on the association of obesity and dietary antioxidants, especially in adolescents.[21–23] A recent meta-analysis concluded that vitamin E supplementation had no effects on waist circumference (WC) and BMI.[24] A systematic review and meta-analysis reported that higher TAC had a significant association with lower WC while had no significant association with BMI in adult population.[25]

Dietary antioxidant index (DAI) as a validated nutritional tool has been recently considered to evaluate the antioxidant capacity of diets and is calculated based on the intake of major dietary antioxidants including vitamin A, vitamin C, vitamin E, selenium, manganese, and zinc.[26] The relationship between the DAI and the risk of chronic diseases has been reported.[27] The DAI may accurately predict some serum antioxidant and anti-inflammatory levels and estimate the risk of health outcomes better than assessment of single antioxidants.[28] The present study aimed to examine the association between DAI and BMI in adolescent boys. The hypothesis of this study was that the level of dietary antioxidants is related to BMI in adolescent boys.

2 | METHODS

2.1 | Study population

This cross-sectional study was conducted on 593 adolescent boys aged 12–16 years which were selected from two schools by using simple random sampling. The participants were divided into two groups of overweight ($n = 214$) and normal weight ($n = 321$) boys based on the BMI Z-score. The boys having chronic heart disease, lung disease, kidney disease, acute or chronic inflammatory diseases, epilepsy and celiac diseases, besides taking antioxidant supplements including vitamin A, E, C, B, carotene, and zinc, the energy intake of higher than 5500 kcal/d and less than 800 kcal/d, and using weight loss diet programs were excluded.

2.2 | Anthropometric measurements

Weight and height were assessed by using a digital scale and non-elastic tape, respectively in a standing position with minimal clothing and without shoes. Obesity was defined based on World Health Organization (WHO) criteria, as BMI, $Z > +2$ in adolescent boys.[29]

2.3 | Assessment of dietary intake

Dietary intake was assessed using 168-item semi quantitative food frequency questionnaire (FFQ).[18] The frequency of food consumption over the past year was examined through a face-to-face interview. Household measures were taken into account for portion sizes and then were converted to grams. The food composition table (FCT) of the United States Department of Agriculture (USDA) was used to evaluate the amount of energy and nutrients. The Iranian FCT was considered for local foods that were not existed in the FCT. Data collected from FFQ were converted to grams of nutrients using Nutritionist IV software (First Databank Division, the Hearst Corporation, modified for Iranian foods).

2.4 | Dietary antioxidant index assessment

The DAI score was calculated using the Wright method to measure the antioxidant capacity of the diet.[19] First, the average global intake of each item of vitamin A, vitamin C, vitamin E, magnesium, selenium and zinc was deducted from the amount received by that item then, divided the result by the global standard deviation (SD). Finally, the standardized amount of six items was summed up to calculate DAI.[19,20]

$$DAI = \sum_{i=1}^{n=6} \cdot \frac{\text{individual intake} - \text{global mean}}{\text{global SD}}$$

2.5 | Physical activity measurements

Daily activities were assessed using international physical activity questionnaire (IPAQ).[21] In this questionnaire, participants were asked about the frequency and duration of walking, moderate, and heavy physical activities that lasted at least 10 min during the last 7 days. According to the IPAQ, times of walking, moderate and heavy activities per week were obtained by multiplying the days by the average duration of each activity. To calculate walking, moderate and heavy activity scores, the number of minutes per activity per week was multiplied by the specific metabolic rate task (MET) for that activity. The MET value for walking, moderate and heavy activity was defined as 3.3, 4, and 8, respectively. The total score of physical activity was obtained from the sum of walking, moderate and heavy activity scores.

3 | STATISTICAL ANALYSIS

Chi-square and independent t-test were used to compare qualitative and quantitative variables between the participants, respectively. The logistic and linear regression analysis methods were used to investigate the association between BMI and DAI. A stepwise (Forward) selection procedure was used for modeling and variables were selected based on significance and background knowledge as adjusted for age, physical activity and caloric intake (Model 1), and further adjustment for dietary intake of vitamin A, vitamin E, vitamin C, zinc, manganese, and Selenium (Model 2). Statistical analyses were performed using SPSS software version 20 (SPSS Inc.) and p-value <0.05 was considered as significant in all analyses.

4 | ETHICAL CONSIDERATIONS

At the beginning of the study, the objectives of the study were explained orally to all participants and written consent was obtained. The study protocol was approved by the ethics research committee of the Shahid Beheshti University of Medical Sciences, Tehran, Iran (Code: IR.SBMU.RETECH.REC.1397.1139).

5 | RESULTS

The Shapiro-Wilk test showed that the data was normally distributed. Table 1 shows the distribution of general characteristics and micronutrients intakes across the participants. The overweight adolescents had higher mean of BMI (26.40 ± 4.60 vs. 19.50 ± 2.47 kg/m^2, $p < 0.0001$) and higher intake of calorie (2490.55 ± 632.49 vs. 2354.33 ± 632.64 kcal/d, $p = 0.01$), carbohydrate (290.21 ± 71.41 vs. 272.93 ± 79.22 g/d, $p = 0.01$), and fat (111.51 ± 40.76 vs. 104.51 ± 35.56 g/d, $p = 0.04$) in comparison with adolescents with normal weight.

Table 2 shows the comparison of micronutrient intake between normal weight and obese adolescent boys. Overweight adolescents had higher intake of calcium (811.70 ± 283.70 vs. 741.06 ± 251.17 mg/d, $p = 0.003$) and vitamin D (1.41 ± 1.17 vs. 1.18 ± 1.19 µg/d, $p = 0.031$) compared with normal weight adolescents. Other micronutrients intake were not different between normal weight and obese adolescent boys ($p > 0.05$).

The distribution of characteristics across the participants with high or low DAI is presented in Table 3. Adolescents with DAI ≥ -0.61 score had higher consumption of total intake of energy (3029.76 ± 897.29 vs. 1847.52 ± 628.66 kcal/d, $p < 0.001$), protein (111.05 ± 30.44 vs. 68.28 ± 30.44 g/d, $p < 0.001$), carbohydrate (350.53 ± 103.77 vs. 215.47 ± 80.67 g/d, $p < 0.001$), total fat (135.80 ± 64.55 vs. 8.38 ± 37.98 g/d, $p < 0.001$), potassium (3288.65 ± 1212.16 vs. 1911.75 ± 806.11 mg/d, $p < 0.001$), vitamin A ($1343.87 \pm 4137/18$ vs. 287.97 ± 228.82 µg/d, $p = 0.007$), vitamin C (101.50 ± 99.52 vs. 46.42 ± 30.56 mg/d, $p < 0.001$), calcium (929.26 ± 442.49 vs. 639.95 ± 337.49 mg/d, $p < 0.001$), iron (20.21 ± 7.33 vs. 12.50 ± 3.88 mg/d, $p < 0.001$), vitamin E (44.81 ± 32.67 vs. 26.00 ± 17.40 mg/d, $p < 0.001$), thiamin (2.17 ± 0.75 vs. 1.38 ± 0.48 mg/d, $p < 0.001$), riboflavin (2.32 ± 1.61 vs. 1.34 ± 0.59 mg/d, $p < 0.001$), niacin (32.65 ± 15.13 vs. 20.17 ± 12.15 mg/d, $p < 0.001$), zinc (18.00 ± 7.81 vs. 9.19 ± 4.22 mg/d, $p < 0.001$), manganese (6.66 ± 3.02 vs. 3.17 ± 1.32 mg/d, $p < 0.001$), and selenium (167.94 vs. 77.97 ± 34.59 µg/d, $p < 0.001$) than adolescents with DAI < -0.61 score.

The odds ratio and confidence intervals for the association between DAI and BMI in two ways of categorical and continuous DAI are presented in Table 4. In the categorical type (based on the median), no association was found between DAI and BMI after controlling for confounders including age, total energy intake, vitamin A, vitamin E, calcium, zinc, manganese, and selenium (OR: 0.87, CI 95%: 0.39–1.93, $p = 0.742$). Whereas, continuous DAI had a significant inverse association with BMI after adjustment for age and caloric intake (OR: 0.85, CI 95%: 0.76–0.96, $p = 0.009$). The association remained significant after further adjustments for vitamin A, E, and C and zinc, manganese, and selenium (OR: 0.56, 95%, CI: 0.35–0.90, $p = 0.016$).

Linear regression found an inverse association between BMI and DAI ($B = 0.19$, $p = 0.009$) (Table 5). The result remained significant after additional adjustments for vitamin A, E, and C and zinc, manganese, and selenium ($B = 0.22$, $p = 0.004$).

6 | DISCUSSION

In the present study, obese adolescents had higher intake of energy, carbohydrate, calcium, and vitamin D in comparison with normal weight participants. Adolescents with a DAI score of ≥ -0.61 had higher intake of antioxidants, including vitamin A, C, E, zinc, and selenium. Also, the DAI was inversely associated with BMI in adolescent boys when it was considered as a continuous variable. There was a linear inverse association between BMI and DAI after adjustments for different confounding variables.

Few studies investigated the association between dietary antioxidants and BMI in adolescents.[22,23,30] Imboden et al. reported that

TABLE 1 Distribution of characteristics and dietary intakes across overweight and normal weight participants

	Mean ± SD		
	Overweight (*n* = 214)	Normal weight (*n* = 321)	*p*-value
Age (years)	13.95 ± 1.07	14.20 ± 1.37	0.03
Weight (kg)	82.50 ± 5.02	76.81 ± 7.54	0.04
Height (cm)	165.14 ± 5.28	165.08 ± 4.51	0.88
Body mass index (kg/m^2)	26.40 ± 4.60	19.50 ± 2.47	<0.0001
Energy (kcal/d)	2490.55 ± 632.49	2354.33 ± 632.64	0.01
Protein (g/d)	90.24 ± 24.95	89.04 ± 30.25	0.61
Carbohydrate (g/d)	290.21 ± 71.41	272.93 ± 79.22	0.01
Fat (g/d)	111.51 ± 40.76	104.51 ± 35.56	0.04
Physical activity (MET-minutes/week)	1005 ± 71.8	1218 ± 168.7	0.76

	Overweight (*n* = 214)	Normal weight (*n* = 321)	*p*-value
Sodium (mg/d)	2882.37 ± 5469.26	2390.53 ± 977.47	0.114
Potassium (mg/d)	2637.29 ± 756.59	2550.47 ± 883.22	0.241
Vitamin A (μg/d)	759.31 ± 1125.30	897.66 ± 2759.25	0.491
Vitamin C (mg/d)	72.71 (39.85)	75.59 ± 65.04	0.565
Calcium (mg/d)	811.70 ± 283.70	741.06 ± 251.17	0.003
Iron (mg/d)	16.56 ± 4.30	16.00 ± 4.97	0.182
Vitamin D (μg/d)	1.41 ± 1.17	1.18 ± 1.19	0.031
Vitamin E (mg/d)	36.31 ± 19.43	33.90 ± 15.82	0.118
Thiamin (mg/d)	1.80 ± 0.46	1.73 ± 0.51	0.095
Riboflavin (mg/d)	1.86 (0.78)	1.77 (0.95)	0.266
Niacin (mg/d)	26.74 ± 9.73	25.82 ± 9.98	0.292
Zinc (mg/d)	13.48 ± 4.27	13.73 ± 5.97	0.590
Manganese (mg/d)	322.93 (84.32)	316.13 ± 110.86	0.449
Selenium (μg/d)	122.05 ± 43.55	123.85 ± 64.60	0.723

TABLE 2 The comparison of macro- and micro-nutrients intake between normal weight and obese adolescent boys

there was no association between antioxidant intake and BMI in children. Also, overweight children had higher intake of antioxidant compared with normal weight and obese children.[31] However, the results of the present found no difference in antioxidant intake between normal weight and obese adolescents. This could be due to the age difference between the studies as well as the higher consumption of energy in overweight than normal weight children indicating the higher intake of antioxidants. A systematic review and meta-analysis observed that TAC was related to WC but not BMI among adults.[25] Whereas, another study reported that dietary TAC was inversely related with BMI in children and adolescents.[22] The contradictory results on the association between BMI and dietary antioxidants might be due to the difference of age groups in different studies and BMI is more likely to be associated with dietary antioxidant in children and adolescents.

Some studies indicated that adherence to antioxidant-rich diets as free radical scavengers may reduce BMI and oxidative stress.[32,33]

Individuals with obesity compared to individuals with normal weight experience imbalanced antioxidant defenses or reduced antioxidant values.[34,35]

A 10-week weight loss program intervention in children showed that changing in food selections and higher intake of fruits and vegetables may reduce BMI.[36] However, a possible cause of this association can be the presence of fiber in fruits and vegetables, which decrease appetite, and thus restrict energy intake and reduce weight and BMI.[37] Regarding to the antioxidant serum level, a survey examined the association between serum vitamin E levels and BMI in adolescents and found that there was a negative relationship between vitamin E and BMI in girls, but not boys.[38] Another study found that BMI could significantly affect vitamin C status in adolescent boys but not girls.[39] The negative association between DAI and BMI in adolescent boys can be explained by the hypothesis that obesity with increased adipose tissue contribute to generate reactive oxygen species (ROS) in oxidation reactions and subsequently antioxidant enzymes are

TABLE 3 Distribution of characteristics across groups of the DAI (*n* = 229)

	DAI < −0.61 (n = 268)	DAI ≥ −0.61 (n = 267)	p-value
Age (year)	13.87 ± 1.03	13.96 ± 1.00	0.47
Total energy intake (kcal/d)	1847.52 ± 628.66	3029.76 ± 897.29	<0.001
Protein (g/d)	68.28 ± 30.44	111.05 ± 40.26	<0.001
Carbohydrate (g/d)	215.47 ± 80.67	350.53 ± 103.77	<0.001
Total fat (g/d)	81.38 ± 37.98	135.80 ± 64.55	<0.001
Cholesterol intake (mg/d)	235.92 ± 199.91	373.94 ± 336.50	<0.001
Saturate fatty acid (g/d)	21.44 ± 11.27	34.67 ± 17.72	<0.001
Mono unsaturated fatty acid (g/d)	30.81 ± 15.67	51.87 ± 27.29	<0.001
Poly unsaturated fatty acid (g/d)	21.12 ± 12.69	37.52 ± 22.76	<0.001
Sodium (mg/d)	2457.92 ± 9226.87	2918.70 ± 1594.54	0.59
Potassium (mg/d)	1911.75 ± 806.11	3288.65 ± 1212.16	<0.001
Vitamin A (µg/d)	287.97 ± 228.82	1343.87 ± 4137.18	0.007
Vitamin C (mg/d)	46.42 ± 30.56	101.50 ± 99.52	<0.001
Calcium (mg/d)	639.95 ± 337.49	929.26 ± 442.49	<0.001
Iron (mg/d)	12.50 ± 3.88	20.21 ± 7.33	<0.001
Vitamin D (µg/d)	1.16 ± 1.48	1.48 ± 2.09	0.18
Vitamin E (mg/d)	26.00 ± 17.40	44.81 ± 32.67	<0.001
Thiamin (mg/d)	1.38 ± 0.48	2.17 ± 0.75	<0.001
Riboflavin (mg/d)	1.34 ± 0.59	2.32 ± 1.61	<0.001
Niacin (mg/d)	20.17 ± 12.15	32.65 ± 15.13	<0.001
Zinc (mg/d)	9.19 ± 4.22	18.00 ± 7.81	<0.001
Manganese (mg/d)	3.17 ± 1.32	6.66 ± 3.02	<0.001
Selenium (µg/d)	77.97 ± 34.53	167.94 ± 56.41	<0.001

Abbreviation: DAI, dietary antioxidant index.

TABLE 4 Odds ratios and confidence intervals for the association between BMI and DAI

	DAI (categorical)[a] OR (95% CI)			DAI (continuous)	
DAI	DAI < −0.61	DAI ≥ −0.61	p-value	OR (95% CI)	p-value
Model A	1 (ref.)	1.38 (0.70–2.72)	0.339	0.85 (0.76–0.96)	0.009
Model B	1 (ref.)	0.87 (0.39–1.93)	0.742	0.56 (0.35–0.90)	0.016

Note: Model A: adjusted for age, physical activity and total energy intake. Model B: Model A+ adjusted for vitamin A, E, and C and Zinc, Manganese, and Selenium.

Abbreviations: BMI, body mass index; CI, confidence interval; DAI, dietary antioxidant index; OR, odds ratio.

[a]Classified according to the median.

TABLE 5 Linear regression of the association between BMI and DAI

	B	p
Model 1	0.19	0.009
Model 2	0.22	0.004

Note: Model 1: adjusted for age, physical activity and total energy intake. Model 2: Model A+ adjusted for vitamin A, E, and C and Zinc, Manganese, and Selenium.

Abbreviations: BMI, body mass index; DAI, dietary antioxidant index.

decreased which can lead to several abnormalities such as atherosclerosis. Also, production of inflammatory cytokines from adipose tissue and other biomarkers of oxidative damage is higher in individuals with obesity compared to the others which can be related to BMI, the percentage of body fat, and low density lipoprotein (LDL) oxidation.[40] In this condition, antioxidant defense markers are lower because of the higher amounts of body fat and central obesity.[41,42]

The negative association between DAI and BMI in adolescent boys can be explained by the hypothesis that obesity with increased adipose

tissue contribute to generate ROS in oxidation reactions and subsequently antioxidant enzymes are decreased which can lead to several abnormalities such as atherosclerosis. Also, production of inflammatory cytokines from adipose tissue and other biomarkers of oxidative damage is higher in individuals with obesity compared to the others which can be related to BMI, the percentage of body fat, and LDL oxidation.[40] In this condition, antioxidant defense markers are lower because of the higher amounts of body fat and central obesity.[41,42]

The exact mechanisms of the effect of antioxidants on obesity are not yet clear. Antioxidants could have beneficial effects in weight loss.[43] The antioxidant sources may affect the BMI by modification of lipids and carbohydrates metabolism, regulation of appetite, and altering the secretion of adipocytokines.[44] Another study indicated that high-fat and high-carbohydrate diets caused a significant increment of oxidative stress and inflammation in individuals with obesity.[45] A possible mechanism of the inflammatory effects of high-fat and high-carbohydrate diets is that postprandial hypertriglyceridemia and hyperglycemia are associated with increased oxidative stress due to the production of nitrotyrosine in this situation.[46] Moreover, higher free radicals induce higher insulin resistance and higher levels of pro-inflammatory markers.[47] The anti-obesity effect of dietary antioxidants could be related to promotion of catabolism in adipose tissue, induction of apoptosis in mature adipocytes, and inhibition of intestinal fat absorption.[48,49] Another mechanism regarding prevention obesity by intake of antioxidant sources can be through regulation of brown adipose tissue metabolism and increase thermogenesis which can control body weight.[50] For instance, vitamin A triggers the transcription of the gene encoding uncoupling protein 1 that is vital to brown adipose tissue thermogenesis.[51] In addition, leptin is a hormone released by adipose tissue and plays a role in appetite regulation, can be decreased by consumption of antioxidant supplemented food so that the obesity propagation cycle could be inhibited.[52] Moreover, recent studies reported the mediatory role of transcriptional factors in the association between obesity and oxidative stress.[53–55] For example, nuclear erythroid factor 2-like 2 (NRF2), one of the most important transcription factors in the oxidative stress response, is now considered to play a crucial role in obesity.[56] NRF2 knock-out mice were reported to be partially protected from high-fat diet-induced obesity.[57] Nrf2 has been introduced as a new target for combating obesity.[58]

One of the strengths of this study was that it was conducted on adolescent boys who were less studied. However, this study had some limitations. First, it was a cross-sectional study that does not allow us to interpret the causal relationships. The study sample was limited to adolescent boys and the results could not be generalized to girls and other age groups. Also, assessing adolescent food intake with the FFQ questionnaire may not be accurate. However, the results of a meta-analysis indicated that FFQ is an accurate tool for collecting data and could be used for ranking adolescents in terms of energy and nutrient intakes.[59] More longitudinal studies are needed on the relationship between dietary antioxidant status and obesity in adolescents.

7 | CONCLUSION

In conclusion, there was an association between DAI and BMI in adolescent boys. Following antioxidant-rich diets during adolescence may help prevent obesity. To our knowledge, it was the first study to investigate the association between DAI and BMI in adolescent boys. Both categorical and continuous types of DAI were considered. Further longitudinal studies are warranted to determine if there is a relationship between different weight classes and DAI in both genders.

AUTHOR CONTRIBUTIONS

Bahareh Aminnejad, Saeid Doaei, Naeemeh Hasanpour Ardekanizadeh and Asal Ataei designed the study, and carried out the data collection. Bahareh Aminnejad, Maryam Gholamalizadeh, Farhad Vahid and Asal Ataei were involved in the design of the study, analysis of the data, and critically reviewed the manuscript. This study was conducted at the Shahid Beheshti University of Medical Sciences, Tehran, Iran.

CONFLICT OF INTEREST

The authors declare no competing interests.

CONSENT FOR PUBLICATION

Institutional consent forms were used in this study.

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