

Original Article



The Association between Dietary Antioxidant Quality Score and Cardiorespiratory Fitness in Iranian Adults: a Cross-Sectional Study

Hossein Shahinfar ,^{1,2} Mahshid Shahavandi ,¹ Aliyu Tijani Jibril ,¹ Kurosh Djafarian ,³ Cain C. T. Clark ,⁴ Sakineh Shab-Bidar ¹

OPEN ACCESS

Received: Apr 15, 2020
Revised: Jul 5, 2020
Accepted: Jul 7, 2020

Correspondence to Sakineh Shab-Bidar

Department of Community Nutrition, School of Nutritional Sciences and Dietetics, Tehran University of Medical Sciences, No 44, Hojjat-dost Alley, Naderi St., Keshavarz Blvd, Tehran 14167-53955, Iran.

E-mail: s_shabbidar@tums.ac.ir

Copyright © 2020. The Korean Society of Clinical Nutrition

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<https://creativecommons.org/licenses/by-nc/4.0/>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ORCID iDs

Hossein Shahinfar
<https://orcid.org/0000-0002-4499-4102>
Mahshid Shahavandi
<https://orcid.org/0000-0002-6198-5283>
Aliyu Tijani Jibril
<https://orcid.org/0000-0003-3930-9091>
Kurosh Djafarian
<https://orcid.org/0000-0002-9134-7178>
Cain C. T. Clark
<https://orcid.org/0000-0002-6610-4617>
Sakineh Shab-Bidar
<https://orcid.org/0000-0002-0167-7174>

Funding

This manuscript has been granted by Tehran University of Medical Sciences (grant No: 33887).

¹Department of Community Nutrition, School of Nutritional Sciences and Dietetics, Tehran University of Medical Sciences, Tehran 14167-53955, Iran

²Students' Scientific Research Center, Tehran University of Medical Sciences, Tehran 14167-53955, Iran

³Department of Clinical Nutrition, School of Nutritional Sciences and Dietetics, Tehran University of Medical Sciences, Tehran 14167-53955, Iran

⁴Centre for Sport, Exercise, and Life Sciences, Coventry University, Coventry CV1 5FB, UK

ABSTRACT

The association between dietary antioxidant quality score (DAQS) and cardiovascular risk factors such as low cardiovascular fitness (CRF) and elevated blood pressure (BP) has rarely been investigated. To investigate the association between DAQS, CRF, and BP. This cross-sectional study was conducted on 270 adult subjects living in Tehran, Iran. Dietary intake was evaluated using a validated food frequency questionnaire. The DAQS was calculated using antioxidant-nutrient intake. Socio-economic status, anthropometric measures, and BP were recorded by a trained interviewer, using standard methods. A significant increase was found in maximal oxygen uptake (p value = 0.01) across tertiles of DAQS. After adjusting for confounders, the association remained unchanged (p value = 0.02). Participants in the highest tertile of DAQS had higher systolic BP (SBP) (p value = 0.01) and diastolic BP (DBP) (p value = 0.03), although adjustment for confounding factors attenuated the results (p value = 0.3 for DBP and p value = 0.6 for SBP). Our results revealed that higher DAQS is associated with better CRF in Iranian adults. Further studies are needed to establish the veracity of our results.

Keywords: Antioxidants; Minerals; Cardiorespiratory fitness; Blood pressure

INTRODUCTION

Cardiovascular fitness (CRF) is an important marker of physiological condition and maybe defined as the ability of the circulatory, respiratory, and muscular systems to supply oxygen during sustained physical activity. CRF is usually expressed in metabolic equivalents (METs) or maximal oxygen uptake (VO_2 max) measured by exercise tests, using treadmill or cycle ergometers [1]. Low CRF and elevated blood pressure (BP) are considered to be risk factors and strong, independent, identifiers of cardiovascular diseases (CVDs) [2,3]. Numerous studies have reported on the effect of antioxidants on training adaptation in response to endurance training, and the results are equivocal. In addition, a number of studies on humans have shown no beneficial effect of vitamin C on endurance [4], or on aerobic and anaerobic capacity [5,6]. Training studies with vitamin E supplementation alone have also

Conflict of Interest

The authors declare that they have no competing interests.

shown no effect on endurance capacity [7], or indeed on cardiorespiratory efficiency and motor fitness [8]. In young competitive swimmers, no effect on VO_2 max during exercise in healthy individuals after supplementation with vitamin C and E has also been reported by Yfanti et al. [9]. However, in animal studies, vitamin C and E have been purported to elicit beneficial effects on endurance capacity in aging rats [10]. The synthesis of free radicals have been identified to influence BP [11]; whilst exogenous administration of antioxidants has been used in animal models and in humans with hypertension to counteract the hypertensive effect of reactive oxygen species (ROS), due to their potential role in improving vascular function and reducing BP [12]. While several studies have suggested an inverse association between dietary antioxidants and BP [13-22], published results from randomized controlled clinical trials do not support the hypothesis that vitamin E or β -carotene supplementation has a protective effect on BP [23,24]. Relatedly, diets consist of a variety of foods, with complex combinations of antioxidant nutrients. The most consistently used approach in determining the potential role of antioxidant dietary intake on health outcome has been based on the content and amount of individual antioxidant nutrients in the diet. The dietary antioxidant quality score (DAQS), which sums certain dietary antioxidants and assigns a score based on calculated quantity compared with the recommended daily intake (RDI) quantity, has been suggested as a sensitive and accurate method [25]. To our knowledge, there is no available evidence regarding the association between DAQS and cardiovascular risk factors such as low CRF and elevated BP. Thus, the purpose of this study was to assess the association of DAQS with cardiorespiratory fitness and BP among Iranian adults.

MATERIALS AND METHODS

Study design

This cross-sectional study was conducted on 270 adults (118 males and 152 females), aged between 18–45 years' old who lived in Tehran, Iran between February 2017 and December 2018. Participants were recruited using advertisement, distribution of flyers in the common area, and information sessions held at residential facilities. The participants were selected based on the following inclusion criteria: 1) apparently healthy people with the age range of 18–45 years, 2) no alcohol or drug abuse, 3) participants with special diets, such as weight loss or weight gain diets, pregnant and lactating women, receiving any special medication or supplements (slimming medicine, hormone, sedative, supplements containing thermogenic substances, such as caffeine and green tea, linoleic acid conjugate, etc.) were excluded from the study. This study was conducted according to the guidelines laid down in the Declaration of Helsinki. All procedures were followed in accordance with the ethical standards of the Tehran University of Medical Sciences (ethic number: IR.TUMS.VCR.REC.1396.4085), who approved all aspects of the study. All participants signed a written informed consent prior to the start of the study.

Exposures and outcomes

Information on lifestyle was collected via self-administered questionnaires, and included age (continuous variable), sex (male or female), CVD (yes or no), diabetes (yes or no), and smoking status (current, former or never smoking), marital status (single or married) and physical activity was assessed using a validated short form of the International Physical Activity Questionnaire [26]. Subjects were grouped into 3 categories including very low (< 600 MET-minute/week), low (600–3,000 MET-minute/week), moderate and high (> 3,000 MET-minute/week), calculated based on METs [27].

Anthropometric measures and body composition

Body weight was determined using a standard body weight scale (Seca 707; Seca GmbH & Co. KG., Hamburg, Germany). The participant's height was measured, unshod, using a stadiometer (Seca GmbH & Co. KG.). To measure waist-hip ratio, waist circumference (WC) in centimeters was divided by hip circumference in centimeters. We measured WC between the middle of bottom ribs and pelvic bones, after normal exhalation, using a non-stretch tape measure. Body mass index (BMI) was calculated as weight in kilograms, divided by height in meters squared. Body composition was measured using a body composition analyzer (InBody 720; Biospace, Seoul, Korea); where all participants were asked to follow these conditions before measurement: no food ingestion for at least 4 hours, minimal intake of 2 L of water the day before, no physical activity for at least 8 hours, no coffee or alcoholic beverage consumption during at least 12 hours, and no diuretic use for at least 24 hours, prior to assessment, respectively. Participants were required to urinate immediately before the body composition test [28].

Measurement of BP

To assess BP, first, we asked participants to sit for 10 minutes; BP was then measured using a standard mercury sphygmomanometer. The mean of the 2 measurements was recorded as the participant's BP.

Cardiorespiratory fitness testing

The VO_2 max was measured using a treadmill and respiratory gas analyzer (Cortex Metabolizer 3B). The subjects warmed up for 5 minutes on the treadmill at a speed of 5 km/hr, next the Bruce test was used to determine the VO_2 max, following standard procedures [29]. After completing the Bruce test, the subjects walked at a speed of 4 km/hr in order to cool down for 3 minutes and encouraged to perform 5-to-10 minutes of stretching. The conditions for test cessation were: the participant's heart rate reaches > 90% of the maximum heart rate, a respiratory exchange ratio over 1.1, and having a plateau in oxygen intake, despite increases in exercise intensity.

Dietary assessment

The dietary intake of participants was assessed using a valid and reliable semi-quantitative food frequency questionnaire (FFQ) [30], which contained 168 food items. FFQ was administered by trained dietitians, via face-to-face interviews, asking participants to report their frequency of consumption of each food item, during the past year on a daily, weekly, or monthly basis. These reports were then converted to daily intakes. The food items were analyzed for their energy content using Nutritionist IV software, modified for Iranian foods (version 7.0; N-Squared Computing, Salem, OR, USA).

Measurement of DAQS

DAQS was obtained from some vitamins and minerals that have antioxidant functions including selenium, zinc, vitamin A, vitamin C, and vitamin E [31]. To create a DAQS, we compared daily intake of nutrients to that of the RDI [32]. Each of the 5 antioxidant intakes was assessed and then we allocated a value of 0 or 1, separately, for every all components. According to Tur et al. [31] method when the intake was lower than 2/3 of the RDI, it was assigned a value of 0. Similarly, when the intake was higher than 2/3 of the RDI, it was assigned a value of 1. Thus, the total DAQS ranged from 0 (very poor quality) to 5 (high quality) [31]. The percentage of the RDI as well as the proportion of individuals with intakes below the RDI, 2/3 of the RDI, and 1/3 of the RDI were calculated. The proportion of

individuals with intakes below 2/3 of the RDI was the criterion used to estimate the risk of inadequate intake [33].

Statistical analysis

Participants were categorized based on tertiles of DAQS. Higher tertiles of DAQS demonstrate higher antioxidant intake compared to lower tertiles. General characteristics of study participants among tertiles of DAQS were tested using analysis of variance for continuous variables, and χ^2 for categorical variables. We used analysis of covariance (ANCOVA) to compare VO₂ max, heart rate, pulse pressure, and BP among tertiles of DAQS also all values were adjusted for age, sex, weight, height, smoking, physical activity, and energy intake. Multiple regression analysis was used to evaluate the association between CRF, antioxidant intakes, and DAQS score after adjustment for covariates, including age, sex, body weight and height, smoking, physical activity, and energy intake. All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS version 25; SPSS Inc., Chicago, IL, USA) We considered $p < 0.05$ to represent statistical significance.

RESULTS

The general characteristics of the participants by tertiles of DAQS are shown in **Table 1**. Among 270 participants, 43.7% were men and 56.3% were women. The mean age of participants was 36.77 ± 13.19 years with a mean BMI of 25.62 kg/m^2 . Compared with females in the lowest tertile, those in the highest tertile of the DAQS had significantly higher FM, weight, BMI, and WC (p value = 0.01 for all comparisons). Dietary intake of nutrients according to the tertiles of the DAQS are presented in **Table 2**. Adherence to DAQS showed a significant increase for intake of vitamin B₆ (p value = 0.02), riboflavin (p value < 0.001), folate (p value = 0.03), selenium (p value = 0.03), vitamin D (p value < 0.001), and calcium (p value < 0.001). Compared with participants in the lowest tertile, those in the highest tertile of the DAQS had significantly lower intakes of carbohydrates (p value < 0.01), thiamin (p value < 0.001) and higher intakes of magnesium (p value < 0.01), vitamin C (p value = 0.02), zinc, protein, vitamin A, and energy (p value < 0.001 for all). Daily intake of the nutrients in the study population are shown in **Table 3**.

Men had lower daily intake of Folate (p value = 0.04), vitamin D, vitamin E, and magnesium (p value < 0.001 for all) than Dietary Reference Intake (DRI). Women also had lower intake of folate, vitamin D, vitamin E, calcium, selenium, and magnesium (p value < 0.001 for all) than DRI. The association between CRF and antioxidant nutrients was examined using multiple regression analysis models adjusted by age, sex, weight, height, smoking, physical activity, and energy intake, and is presented in **Table 4**. Multiple linear regression models showed that DAQS had a significant positive association with VO₂ max in the crude model (p value = 0.02). after Adjustment for confounding factors including age, sex, weight, height, smoking, physical activity, and energy intake, the association remained significant (p value = 0.03). The multivariate adjusted means for CRF, heart rate, pulse pressure, SBP, and DBP according to tertiles of DAQS have shown in **Table 5**. A significant increase was found for VO₂ max (p value = 0.01) across tertiles of DAQS. After adjusting for potential confounders including age, sex, weight, height, physical activity, smoking, and energy intake, the association remained unchanged (phylogenetic ANCOVA = 0.02) (**Table 5**).

Table 1. General characteristics of study participants by T of DAQS

| Characteristics | Values | T of DAQS | | | | | | | | |
|---------------------------|-------------------|-------------------|-------------------|-------------------|-----------------|-------------------|-------------------|-------------------|-------|---------|
| | | Males (43.7%) | | | | p value | Females (56.3%) | | | p value |
| | | T1 (≤ 1) | T2 (1-2) | T3 (≥ 3) | T1 (≤ 1) | | T2 (1-2) | T3 (≥ 3) | | |
| No. | 270 | 10 | 41 | 67 | | 23 | 91 | 38 | | |
| Height (cm) | 168.16 \pm 9.96 | 176.40 \pm 2.63 | 177.28 \pm 7.91 | 175.88 \pm 7.51 | 0.631 | 160.78 \pm 6.90 | 161.83 \pm 6.59 | 162.97 \pm 5.42 | 0.744 | |
| Age (yr) | 36.72 \pm 13.15 | 35.50 \pm 12.53 | 36.05 \pm 12.72 | 39.71 \pm 13.35 | 0.304 | 33.48 \pm 13.97 | 35.51 \pm 12.55 | 37.66 \pm 14.56 | 0.478 | |
| FFM (kg) | 50.11 \pm 12.86 | 59.36 \pm 5.19 | 61.89 \pm 10.08 | 61.61 \pm 9.04 | 0.732 | 40.62 \pm 6.35 | 41.19 \pm 5.41 | 42.10 \pm 8.37 | 0.646 | |
| FM (kg) | 22.45 \pm 9.384 | 18.43 \pm 7.78 | 19.73 \pm 9.08 | 21.57 \pm 9.52 | 0.447 | 23.53 \pm 8.76 | 22.37 \pm 7.85 | 27.72 \pm 11.72 | 0.011 | |
| Weight (kg) | 72.76 \pm 16.02 | 77.79 \pm 11.48 | 81.64 \pm 15.96 | 83.05 \pm 14.16 | 0.555 | 64.16 \pm 13.04 | 63.58 \pm 11.23 | 70.90 \pm 16.14 | 0.013 | |
| WC (cm) | 89.61 \pm 12.53 | 90.96 \pm 10.40 | 92.54 \pm 12.53 | 95.27 \pm 12.57 | 0.391 | 85.11 \pm 11.82 | 84.63 \pm 10.36 | 91.15 \pm 13.61 | 0.012 | |
| WHR | 0.90 \pm 0.06 | 0.91 \pm 0.05 | 0.91 \pm 0.06 | 0.92 \pm 0.07 | 0.442 | 0.88 \pm 0.05 | 0.89 \pm 0.05 | 0.90 \pm 0.06 | 0.054 | |
| BMI (kg/m ²) | 25.62 \pm 4.66 | 25.03 \pm 3.92 | 25.88 \pm 4.06 | 26.85 \pm 4.11 | 0.274 | 24.87 \pm 4.95 | 24.20 \pm 4.20 | 27.01 \pm 6.27 | 0.012 | |
| Marital status (%) | | | | | 0.248 | | | | 0.183 | |
| Single | 46.8 | 5.0 | 17.8 | 22.8 | | 10.0 | 27.8 | 10.0 | | |
| Married | 53.2 | 3.4 | 16.9 | 33.9 | | 4.6 | 32.5 | 15.2 | | |
| Smoking (%) | | | | | 0.255 | | | | 0.235 | |
| Non-smoker | 86.6 | 8.5 | 29.7 | 35.6 | | 13.9 | 59.6 | 23.2 | | |
| Former and current smoker | 13.4 | 0.0 | 5.0 | 21.2 | | 0.7 | 0.7 | 2.0 | | |
| Physical activity (%) | | | | | 0.237 | | | | 0.211 | |
| Low | 38.3 | 3.4 | 11.9 | 16.1 | | 4.6 | 30.5 | 8.6 | | |
| Medium | 41.3 | 5.1 | 12.7 | 20.3 | | 8.6 | 23.2 | 11.9 | | |
| High | 20.4 | 0.0 | 10.2 | 20.3 | | 1.3 | 6.6 | 4.6 | | |
| Diabetes (%) | | | | | 0.701 | | | | 0.123 | |
| Yes | 3.3 | 0.0 | 0.8 | 2.5 | | 1.3 | 0.7 | 1.3 | | |
| No | 96.7 | 8.5 | 33.9 | 54.2 | | 13.2 | 59.6 | 23.8 | | |
| CVD (%) | | | | | 0.712 | | | | 0.326 | |
| Yes | 2.2 | 0.0 | 0.8 | 2.5 | | 0.7 | 0.7 | 0.0 | | |
| No | 97.8 | 8.5 | 33.9 | 54.2 | | 14.0 | 60.0 | 24.7 | | |

Values are based on mean \pm standard deviation or reported percentage. The p value less than 0.05 was considered significant. One-way analysis of variance for quantitative data and χ^2 test for qualitative data have been used.

Subjects in the first T of DAQS had DAQS score between (≤ 1); second T: between (1-2); third T: between (≥ 3).

T, tertiles; DAQS, dietary antioxidant quality score; FFM, fat free mass; FM, fat mass; WC, waist circumference; WHR, waist to hip ratio; BMI, body mass index; CVD, cardiovascular disease.

Table 2. Dietary intake of nutrients according to the T of the DAQS

| Variables | Values | T of DAQS | | | p value | p for trend |
|-------------------------------------|---------------------|--------------------|---------------------|---------------------|---------|-------------|
| | | T1 (≤ 1) | T2 (1-2) | T3 (≥ 3) | | |
| No. | 270 | 34 | 132 | 104 | | |
| Energy (1,000 kcal/day) | 2.39 \pm 0.96 | 1.44 \pm 0.36 | 2.03 \pm 0.55 | 3.11 \pm 10.02 | < 0.001 | < 0.001 |
| Carbohydrates (g/day/1,000 kcal) | 142.25 \pm 20.14 | 149.54 \pm 60.88 | 144.33 \pm 19.37 | 137.21 \pm 20.51 | 0.002 | 0.004 |
| Protein (g/day/1,000 kcal) | 38.23 \pm 8.82 | 35.74 \pm 7.10 | 35.61 \pm 6.82 | 42.04 \pm 10.05 | < 0.001 | < 0.001 |
| Total fat (g/day/1,000 kcal) | 33.21 \pm 8.35 | 31.32 \pm 7.56 | 33.44 \pm 7.62 | 33.47 \pm 7.62 | 0.411 | 0.225 |
| Thiamin (mg/day/1,000 kcal) | 0.78 \pm 0.16 | 0.84 \pm 0.12 | 0.79 \pm 0.16 | 0.75 \pm 0.15 | < 0.001 | < 0.001 |
| Riboflavin (mg/day/1,000 kcal) | 0.73 \pm 0.21 | 0.64 \pm 0.16 | 0.68 \pm 0.17 | 0.82 \pm 0.24 | < 0.001 | < 0.001 |
| Niacin (mg/day/1,000 kcal) | 9.37 \pm 1.82 | 9.49 \pm 1.65 | 9.11 \pm 1.70 | 9.67 \pm 1.98 | 0.065 | 0.640 |
| Vitamin B6 (mg/day/1,000 kcal) | 0.62 \pm 0.19 | 0.56 \pm 0.16 | 0.61 \pm 0.19 | 0.66 \pm 0.20 | 0.021 | 0.012 |
| Folate (IU/day/1,000 kcal) | 133.54 \pm 38.80 | 126.12 \pm 32.20 | 129.35 \pm 39.26 | 141.61 \pm 39.31 | 0.030 | 0.074 |
| Vitamin D (μ g/day/1,000 kcal) | 0.97 \pm 0.83 | 0.73 \pm 0.51 | 0.82 \pm 0.60 | 1.24 \pm 1.07 | < 0.001 | 0.003 |
| Vitamin E (mg/day/1,000 kcal) | 1.89 \pm 1.03 | 1.73 \pm 0.46 | 1.78 \pm 0.60 | 2.07 \pm 1.47 | 0.071 | 0.105 |
| Vitamin A (μ g/day/1,000 kcal) | 582.01 \pm 401.34 | 316.20 \pm 63.37 | 632.56 \pm 480.04 | 598.44 \pm 311.76 | < 0.001 | < 0.001 |
| Vitamin C (mg/day/1,000 kcal) | 60.05 \pm 28.30 | 49.62 \pm 18.90 | 64.01 \pm 30.62 | 58.1 \pm 26.73 | 0.026 | 0.131 |
| Zn (mg/day/1,000 kcal) | 4.09 \pm 1.03 | 3.91 \pm 0.85 | 3.71 \pm 0.87 | 4.63 \pm 1.05 | < 0.001 | < 0.001 |
| Se (μ g/day/1,000 kcal) | 0.02 \pm 0.01 | 0.02 \pm 0.01 | 0.01 \pm 0.01 | 0.02 \pm 0.01 | 0.032 | 0.074 |
| Fe (mg/day/1,000 kcal) | 9.21 \pm 3.16 | 10.14 \pm 3.73 | 9.10 \pm 2.91 | 9.07 \pm 3.28 | 0.210 | 0.095 |
| Ca (mg/day/1,000 kcal) | 433.56 \pm 152.32 | 376.13 \pm 76.88 | 409.92 \pm 125.78 | 481.76 \pm 184.41 | < 0.001 | 0.002 |
| Magnesium (mg/day/1,000 kcal) | 122.31 \pm 24.08 | 119.52 \pm 19.02 | 117.43 \pm 25.15 | 128.62 \pm 22.98 | 0.003 | 0.081 |

Values are based on mean \pm standard deviation. The p value less than 0.05 was considered significant. The p value obtained from 1-way analysis of variances test.

Subjects in the first T of DAQS had DAQS score between (≤ 1); second T: between (1-2); third T: between (≥ 3).

T, tertiles; DAQS, dietary antioxidant quality score; IU, international unit.

Table 3. Daily intake of the nutrients in the study population

| Variables | Males | | p value | Females | | p value |
|---------------------|---------------------|--------|---------|-------------------|--------|---------|
| | Mean ± SD | MD | | Mean ± SD | MD | |
| Thiamin (mg/day) | 2.10 ± 1.19 | 0.991 | < 0.001 | 1.64 ± 0.62 | 0.54 | < 0.001 |
| Riboflavin (mg/day) | 2.08 ± 1.26 | 0.78 | < 0.001 | 1.52 ± 0.65 | 0.42 | < 0.001 |
| Niacin (mg/day) | 27.34 ± 16.62 | 11.31 | < 0.001 | 19.10 ± 7.17 | 5.12 | < 0.001 |
| Vitamin B6 (mg/day) | 1.71 ± 1.08 | 0.411 | < 0.001 | 1.33 ± 0.59 | 0.03 | 0.512 |
| Folate (IU/day) | 357.55 ± 225.41 | -42.60 | 0.040 | 288.55 ± 124.67 | -111 | < 0.001 |
| Vitamin D (µg/day) | 2.81 ± 3.22 | -12.11 | < 0.001 | 2.00 ± 1.82 | -13.0 | < 0.001 |
| Vitamin E (mg/day) | 5.05 ± 2.93 | -9.94 | < 0.001 | 3.98 ± 3.28 | -11.0 | < 0.001 |
| Vitamin A (µg/day) | 1,566.45 ± 1,252.10 | 666.21 | < 0.001 | 1,235.41 ± 805.72 | 535.44 | < 0.001 |
| Vitamin C (mg/day) | 144.23 ± 81.71 | 54.20 | < 0.001 | 134.22 ± 75.40 | 59.73 | < 0.001 |
| Zn (mg/day) | 11.54 ± 6.78 | 0.59 | 0.345 | 8.49 ± 3.40 | 0.49 | 0.071 |
| Se (µg/day) | 0.05 ± 0.04 | 0.003 | 0.324 | 0.03 ± 0.02 | -0.01 | < 0.001 |
| Fe (mg/day) | 24.47 ± 12.76 | 16.43 | < 0.001 | 19.51 ± 9.33 | 1.58 | 0.040 |
| Ca (mg/day) | 1,176.11 ± 633.02 | 176.51 | 0.005 | 924.22 ± 440.67 | -75.6 | 0.032 |
| Magnesium (mg/day) | 322.52 ± 144.16 | -97.60 | < 0.001 | 262.52 ± 97.33 | -57.2 | < 0.001 |

The p value obtained from 1 sample t-test. The p value less than 0.05 was considered significant. SD, standard deviation; MD, mean difference; IU, international unit.

Table 4. Multiple regression analysis models exploring the association of nutrient intake with cardiorespiratory fitness

| Variables | β* ± SE | p value | t | 95% CI |
|----------------|-------------------|---------|-------|---------------|
| Vitamin C (mg) | | | | |
| Model 1 | -0.0062 ± 0.0064 | 0.332 | -0.96 | -0.01, 0.006 |
| Model 2 | 0.0031 ± 0.0055 | 0.574 | 0.56 | -0.007, 0.01 |
| Vitamin E (mg) | | | | |
| Model 1 | 0.1412 ± 0.1523 | 0.341 | 0.94 | -0.15, 0.43 |
| Model 2 | 0.0751 ± 0.1030 | 0.520 | 0.64 | -0.14, 0.28 |
| Vitamin A (µg) | | | | |
| Model 1 | 0.0001 ± 0.0001 | 0.415 | 0.80 | -0.001, 0.001 |
| Model 2 | 0.0001 ± 0.0001 | 0.616 | 0.50 | -0.001, 0.001 |
| Zn (mg) | | | | |
| Model 1 | 0.2540 ± 0.0801 | 0.003 | 2.97 | 0.08, 0.43 |
| Model 2 | 0.1410 ± 0.1022 | 0.141 | 1.47 | -0.05, 0.34 |
| Se (µg) | | | | |
| Model 1 | 45.5920 ± 13.6460 | 0.001 | 3.34 | 18.73, 72.45 |
| Model 2 | 5.0850 ± 9.9915 | 0.615 | 0.50 | -14.60, 24.77 |
| DAQS | | | | |
| Model 1 | 1.4745 ± 0.6262 | 0.022 | 2.36 | 0.24, 2.70 |
| Model 2 | 1.1933 ± 0.5751 | 0.031 | 2.07 | 0.05, 2.33 |

The p value less than 0.05 was considered significant.

Model 1, crude; Model 2, adjusted for age, sex, weight, height, smoking, physical activity and energy intake; SE, standard error; CI, confidence interval; DAQS, dietary antioxidant quality score.

*β coefficient obtained from linear regression.

Table 5. The multivariate adjusted means for cardiorespiratory fitness, heart rate, pulse pressure and blood pressure across T of DAQS

| Variables | Values | T of DAQS | | | P ₁ * | P ₂ † | P ₃ ‡ |
|---------------------------------|----------------|----------------|----------------|----------------|------------------|------------------|------------------|
| | | T1 (≤ 1) | T2 (1-2) | T3 (≥ 3) | | | |
| No. | 270 | 34 | 132 | 104 | | | |
| VO ₂ max (mL/kg/min) | 31.19 ± 7.73 | 28.68 ± 7.75 | 30.56 ± 7.01 | 32.81 ± 8.32 | 0.01 | < 0.01 | 0.02 |
| Heart rate (BPM) | 171.33 ± 20.04 | 169.60 ± 18.37 | 173.50 ± 20.86 | 169.06 ± 19.36 | 0.24 | 0.89 | 0.28 |
| Pulse pressure (mmHg) | 79.68 ± 10.96 | 80.10 ± 8.93 | 80.59 ± 10.54 | 78.47 ± 11.93 | 0.35 | 0.48 | 0.80 |
| SBP (mmHg) | 111.57 ± 19.13 | 109.59 ± 21.74 | 108.56 ± 21.64 | 115.96 ± 13.42 | 0.01 | 0.09 | 0.60 |
| DBP (mmHg) | 70.63 ± 10.68 | 70.68 ± 10.04 | 69.03 ± 11.94 | 72.62 ± 8.78 | 0.03 | 0.36 | 0.35 |

Values are based on mean ± standard deviation. The p value less than 0.05 was considered significant.

Subjects in the first T of DAQS had DAQS score between (≤ 1); second T: between (1-2); third T: between (≥ 3).

T, tertiles; DAQS, dietary antioxidant quality score; VO₂ max, maximal oxygen uptake; BPM, beats per minute; SBP, systolic blood pressure; DBP, diastolic blood pressure.

*Crude model; †The p for trend; ‡Obtained from analysis of covariance test adjusted by age, sex, weight, height, smoking, physical activity and energy intake.

DISCUSSION

In the present study, we sought to investigate the association of DAQS with CRF and BP among Iranian adults. A significant and positive correlation across tertiles of DAQS for VO_2 max was found. In addition, we observed that the total DAQS was significantly related to CRF. Our findings also showed a positive correlation between intakes of selenium and zinc and vitamin C with CRF. Despite its relative insufficiency in diet, zinc performs important roles in body metabolism regulation including energy utilization and work performance [34]. Numerous studies have examined the association between zinc intake and CRF [34-38], and while some studies have confirmed hypozincemia in athletes, there have been no deficiency symptoms generally reported [34,35,38-40]. Moreover, no data are available to indicate that zinc supplementation improves physical performance for athletes [39]. Zinc-containing enzymes are involved in many components of the metabolism. In addition, certain enzymes that contain zinc, such as carbonic anhydrase and lactate dehydrogenase, are involved in intermediary metabolism during activity; another enzyme, superoxide dismutase, protects against free radical damage [41]. Research involving zinc supplementation and exercise performance is extremely limited. However, our results are in line with the results of some other studies [34-37]. In Lukaski [34], the author suggested that zinc may play a major role in promoting strength and cardiorespiratory function in healthy people and athletes. The author also found, for men during exercise, low dietary zinc resulted in a significant decrease in zinc status and impaired cardiorespiratory function [35]. Wada and King [37] reported that men receiving 5.5 compared to 16.5 mg zinc had lower resting energy expenditure and lower respiratory exchange ratio. Moreover, lower overall and unique activity of the carbonic anhydrase isozyme in red blood cells was observed following a lower intake of zinc. The activity of the carbonic anhydrase isozyme, as well as cardiorespiratory function, reduces during severe intensity, and prolonged submaximal exercise, respectively [35,37]. The present study highlighted a relationship between dietary intake of selenium and CRF. Salehi and Moradi [42] reported that consuming a selenium supplement for one month improved cardiovascular function in active males. In contrast, no significant associations between CRF and dietary selenium intake were observed in other studies; for example, a study conducted by Williams [40] showed that selenium supplementation did not improve sporting performance among well-nourished athletes. Tessier et al., [43] also revealed that selenium supplementation, after endurance training, has no beneficial impact on the antioxidant capacity and physical performance. Potential mechanisms which may explain how plasma selenium is associated with CRF remain unclear; however, glutathione peroxidase, an antioxidant enzyme containing selenium, may act to prevent peroxidation of erythrocyte membrane and muscle cell substructures involved in oxygen metabolism [40].

Participants in our study had a lower intake of vitamin E and higher intake of vitamin C, and vitamin A than DRI. A study of Schneider et al. [44] showed that the higher antioxidant diet provided twice the DRI of vitamin E, 5 times the DRI of vitamin C, and twice the DRI of vitamin A, while the regular antioxidant diet provided the DRI of vitamin E, twice the DRI of vitamin C and the DRI of vitamin A. Our study highlighted a relationship between dietary intake of vitamin C and CRF. Most human disease is distinguished by enhanced ROS. Several beneficial adaptations were linked with the use of antioxidant vitamin C [45]. In line with our results, Gomez-Cabrera et al. [5] showed that high dosages of vitamin C affected adaptation to endurance exercise training in both an animal and a human model. In contrast, Roberts et al. [46] confirmed that no effects of vitamin C supplementation on male participants' adaptations to high-intensity running exercise. VO_2 max and endurance

performance improved equally in supplemented and placebo groups [46]. In our study, participants in the highest tertile of DAQS had lower heart rate and pulse pressure. Chen et al. [14] showed that antioxidant vitamins can be essential for the underlying cause and hypertension prevention, whilst Waśkiewicz et al., [47] suggested that the consumption of foods with a high content of antioxidants was associated with lower chances of hypertension in a Polish adult population. The strong association of BP with some oxidative stress-related parameters and suggest a possible role of oxidative stress in essential hypertension pathophysiology [12]. Rodrigo et al. [12] found a strong correlation between BP and some oxidative stress parameters and suggested a potential role of oxidative stress in essential hypertension pathophysiology. ROS exposure increases antioxidant activity of the enzymes, therefore, genes encoding these enzymes are coordinately controlled in their regulatory regions by the antioxidant responsive elements (ARE), a mechanism that occurs through the activation of the transcription factor NF-E2 associated factor 2 (Nrf2). Binding Nrf2 to these ARE sites results in up-regulation of downstream genes, which, in-turn, regulate the activity of antioxidant enzymes in order to compensate for the toxicity of ROS. In most hypertensive patients, this mechanism may be enabled for response to their ROS levels [48-50]. The non-significant association found in our study may have several possible explanations. One explanation is that there was insufficient variation in antioxidant intakes across tertiles of DAQS. Second, because of the temporal relationship between the measured exposure and the outcome, a relationship could remain undetected that did not cover the true latent period. Third, unmeasured variables exist which we did not control to affect the relationship between antioxidants and CRF may have impacted our results. Fourth, the differences observed in our study, as opposed to other studies, may be due to the cross-sectional design which prevents causal inferences to be made. Moreover, the small number of participants in our study may be another reason for non-significant results, although we had enough power to detect the diet-disease relationship. Despite the aforementioned limitations, this is the first study, to our knowledge, to have evaluated the relationship between the DAQS and cardiorespiratory fitness, and thus represents an important addition to the literature.

CONCLUSION

Our results highlighted that DAQS has a significant association with VO_2 max. Moreover, we demonstrated that there was a reduction in heart rate and pulse pressure across tertiles of DAQS, although this association was not significant. It is evident that more prospective studies are needed to affirm confirm the veracity of our results.

ACKNOWLEDGEMENTS

Authors thanks all those who participated in this study.

REFERENCES

1. Lee DC, Artero EG, Sui X, Blair SN. Mortality trends in the general population: the importance of cardiorespiratory fitness. *J Psychopharmacol* 2010;24:27-35.

[PUBMED](#) | [CROSSREF](#)

2. Lawes CMM, Rodgers A, Bennett DA, Parag V, Suh I, Ueshima H, MacMahon S; Asia Pacific Cohort Studies Collaboration. Blood pressure and cardiovascular disease in the Asia Pacific region. *J Hypertens* 2003;21:707-16.
[PUBMED](#) | [CROSSREF](#)
3. Wei M, Kampert JB, Barlow CE, Nichaman MZ, Gibbons LW, Paffenbarger RS Jr, Blair SN. Relationship between low cardiorespiratory fitness and mortality in normal-weight, overweight, and obese men. *JAMA* 1999;282:1547-53.
[PUBMED](#) | [CROSSREF](#)
4. Gey GO, Cooper KH, Bottenberg RA. Effect of ascorbic acid on endurance performance and athletic injury. *JAMA* 1970;211:105.
[PUBMED](#) | [CROSSREF](#)
5. Gomez-Cabrera MC, Domenech E, Romagnoli M, Arduini A, Borrás C, Pallardo FV, Sastre J, Viña J. Oral administration of vitamin C decreases muscle mitochondrial biogenesis and hampers training-induced adaptations in endurance performance. *Am J Clin Nutr* 2008;87:142-9.
[PUBMED](#) | [CROSSREF](#)
6. Keren G, Epstein Y. The effect of high dosage vitamin C intake on aerobic and anaerobic capacity. *J Sports Med Phys Fitness* 1980;20:145-8.
[PUBMED](#)
7. Lawrence JD, Bower RC, Riehl WP, Smith JL. Effects of alpha-tocopherol acetate on the swimming endurance of trained swimmers. *Am J Clin Nutr* 1975;28:205-8.
[PUBMED](#) | [CROSSREF](#)
8. Sharman IM, Down MG, Norgan NG. The effects of vitamin E on physiological function and athletic performance of trained swimmers. *J Sports Med Phys Fitness* 1976;16:215-25.
[PUBMED](#)
9. Yfanti C, Akerström T, Nielsen S, Nielsen AR, Mounier R, Mortensen OH, Lykkesfeldt J, Rose AJ, Fischer CP, Pedersen BK. Antioxidant supplementation does not alter endurance training adaptation. *Med Sci Sports Exerc* 2010;42:1388-95.
[PUBMED](#) | [CROSSREF](#)
10. Asha Devi S, Prathima S, Subramanyam MV. Dietary vitamin E and physical exercise: I. Altered endurance capacity and plasma lipid profile in ageing rats. *Exp Gerontol* 2003;38:285-90.
[PUBMED](#) | [CROSSREF](#)
11. Czernichow S, Blacher J, Hercberg S. Antioxidant vitamins and blood pressure. *Curr Hypertens Rep* 2004;6:27-30.
[PUBMED](#) | [CROSSREF](#)
12. Rodrigo R, Prat H, Passalacqua W, Araya J, Guichard C, Bächler JP. Relationship between oxidative stress and essential hypertension. *Hypertens Res* 2007;30:1159-67.
[PUBMED](#) | [CROSSREF](#)
13. Block G, Mangels AR, Norkus EP, Patterson BH, Levander OA, Taylor PR. Ascorbic acid status and subsequent diastolic and systolic blood pressure. *Hypertension* 2001;37:261-7.
[PUBMED](#) | [CROSSREF](#)
14. Chen J, He J, Hamm L, Batuman V, Whelton PK. Serum antioxidant vitamins and blood pressure in the United States population. *Hypertension* 2002;40:810-6.
[PUBMED](#) | [CROSSREF](#)
15. Diaz MN, Frei B, Vita JA, Keaney JF Jr. Antioxidants and atherosclerotic heart disease. *N Engl J Med* 1997;337:408-16.
[PUBMED](#) | [CROSSREF](#)
16. Duffy SJ, Gokce N, Holbrook M, Huang A, Frei B, Keaney JF Jr, Vita JA. Treatment of hypertension with ascorbic acid. *Lancet* 1999;354:2048-9.
[PUBMED](#) | [CROSSREF](#)
17. Fotherby MD, Williams JC, Forster LA, Craner P, Ferns GA. Effect of vitamin C on ambulatory blood pressure and plasma lipids in older persons. *J Hypertens* 2000;18:411-5.
[PUBMED](#) | [CROSSREF](#)
18. Ghosh SK, Ekpo EB, Shah IU, Girling AJ, Jenkins C, Sinclair AJ. A double-blind, placebo-controlled parallel trial of vitamin C treatment in elderly patients with hypertension. *Gerontology* 1994;40:268-72.
[PUBMED](#) | [CROSSREF](#)
19. Palumbo G, Avanzini F, Alli C, Roncaglioni MC, Ronchi E, Cristofari M, Capra A, Rossi S, Nosotti L, Costantini C, Cavalera C. Effects of vitamin E on clinic and ambulatory blood pressure in treated hypertensive patients. Collaborative Group of the Primary Prevention Project (PPP)--hypertension study. *Am J Hypertens* 2000;13:564-7.
[PUBMED](#) | [CROSSREF](#)

20. Rimm EB, Stampfer MJ, Ascherio A, Giovannucci E, Colditz GA, Willett WC. Vitamin E consumption and the risk of coronary heart disease in men. *N Engl J Med* 1993;328:1450-6.
[PUBMED](#) | [CROSSREF](#)
21. Stampfer MJ, Hennekens CH, Manson JE, Colditz GA, Rosner B, Willett WC. Vitamin E consumption and the risk of coronary disease in women. *N Engl J Med* 1993;328:1444-9.
[PUBMED](#) | [CROSSREF](#)
22. Tribble DL. AHA Science Advisory. Antioxidant consumption and risk of coronary heart disease: emphasis on vitamin C, vitamin E, and beta-carotene: a statement for healthcare professionals from the American Heart Association. *Circulation* 1999;99:591-5.
[PUBMED](#) | [CROSSREF](#)
23. Hennekens CH, Buring JE, Manson JE, Stampfer M, Rosner B, Cook NR, Belanger C, LaMotte F, Gaziano JM, Ridker PM, Willett W, Peto R. Lack of effect of long-term supplementation with beta carotene on the incidence of malignant neoplasms and cardiovascular disease. *N Engl J Med* 1996;334:1145-9.
[PUBMED](#) | [CROSSREF](#)
24. Heart Outcomes Prevention Evaluation Study Investigators, Yusuf S, Dagenais G, Pogue J, Bosch J, Sleight P. Vitamin E supplementation and cardiovascular events in high-risk patients. *N Engl J Med* 2000;342:154-60.
[PUBMED](#) | [CROSSREF](#)
25. Rivas A, Romero A, Mariscal-Arcas M, Monteagudo C, López G, Lorenzo ML, Ocaña-Peinado FM, Olea-Serrano F. Association between dietary antioxidant quality score (DAQs) and bone mineral density in Spanish women. *Nutr Hosp* 2012;27:1886-93.
[PUBMED](#)
26. Moghaddam MB, Aghdam FB, Jafarabadi MA, Allahverdi-pour H, Nikookheslat SD, Safarpour S. The Iranian Version of International Physical Activity Questionnaire (IPAQ) in Iran: content and construct validity, factor structure, internal consistency and stability. *World Appl Sci J* 2012;18:1073-80.
27. Wareham NJ, Jakes RW, Rennie KL, Schuit J, Mitchell J, Hennings S, Day NE. Validity and repeatability of a simple index derived from the short physical activity questionnaire used in the European Prospective Investigation into Cancer and Nutrition (EPIC) study. *Public Health Nutr* 2003;6:407-13.
[PUBMED](#) | [CROSSREF](#)
28. Korth O, Bosy-Westphal A, Zschoche P, Glüer CC, Heller M, Müller MJ. Influence of methods used in body composition analysis on the prediction of resting energy expenditure. *Eur J Clin Nutr* 2007;61:582-9.
[PUBMED](#) | [CROSSREF](#)
29. Bruce RA, Kusumi F, Hosmer D. Maximal oxygen intake and nomographic assessment of functional aerobic impairment in cardiovascular disease. *Am Heart J* 1973;85:546-62.
[PUBMED](#) | [CROSSREF](#)
30. Mirmiran P, Esfahani FH, Mehrabi Y, Hedayati M, Azizi F. Reliability and relative validity of an FFQ for nutrients in the Tehran lipid and glucose study. *Public Health Nutr* 2010;13:654-62.
[PUBMED](#) | [CROSSREF](#)
31. Tur JA, Serra-Majem L, Romaguera D, Pons A. Does the diet of the Balearic population, a Mediterranean type diet, still provide adequate antioxidant nutrient intakes? *Eur J Nutr* 2005;44:204-13.
[PUBMED](#) | [CROSSREF](#)
32. Shils ME, Shike M. *Modern nutrition in health and disease*. Philadelphia (PA): Lippincott Williams & Wilkins; 2006.
33. Aranceta J, Serra-Majem L, Pérez-Rodrigo C, Llopis J, Mataix J, Ribas L, Tojo R, Tur JA. Vitamins in Spanish food patterns: the eVe study. *Public Health Nutr* 2001;4:1317-23.
[PUBMED](#) | [CROSSREF](#)
34. Lukaski HC. Magnesium, zinc, and chromium nutrition and athletic performance. *Can J Appl Physiol* 2001;26 Suppl:S13-22.
[PUBMED](#) | [CROSSREF](#)
35. Lukaski HC. Low dietary zinc decreases erythrocyte carbonic anhydrase activities and impairs cardiorespiratory function in men during exercise. *Am J Clin Nutr* 2005;81:1045-51.
[PUBMED](#) | [CROSSREF](#)
36. Rahman MM, Davies RE, Deyoe CW, Reid BL, Couch JR. Role of zinc in the nutrition of growing pullets. *Poult Sci* 1961;40:195-200.
[CROSSREF](#)
37. Wada L, King JC. Effect of low zinc intakes on basal metabolic rate, thyroid hormones and protein utilization in adult men. *J Nutr* 1986;116:1045-53.
[PUBMED](#) | [CROSSREF](#)

38. Milton AH, Vashum KP, McEvoy M, Hussain S, McElduff P, Byles J, Attia J. Prospective study of dietary zinc intake and risk of cardiovascular disease in women. *Nutrients* 2018;10:38.
[PUBMED](#) | [CROSSREF](#)
39. Williams MH. *Nutrition for health, fitness and sport*. Boston (MA): WCB/McGraw-Hill; 1999.
40. Williams MH. Dietary supplements and sports performance: minerals. *J Int Soc Sports Nutr* 2005;2:43-9.
[PUBMED](#) | [CROSSREF](#)
41. Vallee BL, Falchuk KH. The biochemical basis of zinc physiology. *Physiol Rev* 1993;73:79-118.
[PUBMED](#) | [CROSSREF](#)
42. Salehi K, Moradi F. Effect of intake Selenium supplementation on lipid profile and cardiorespiratory function in rest situation and subsequent an exercise session in active men. *J Birjand Univ Med Sci* 2019;26:137-46.
[CROSSREF](#)
43. Tessier F, Margaritis I, Richard MJ, Moynot C, Marconnet P. Selenium and training effects on the glutathione system and aerobic performance. *Med Sci Sports Exerc* 1995;27:390-6.
[PUBMED](#) | [CROSSREF](#)
44. Schneider CD, Bock PM, Becker GF, Moreira JC, Bello-Klein A, Oliveira AR. Comparison of the effects of two antioxidant diets on oxidative stress markers in triathletes. *Biol Sport* 2018;35:181-9.
[PUBMED](#)
45. Jacob RA, Burri BJ. Oxidative damage and defense. *Am J Clin Nutr* 1996;63:985S-990S.
[PUBMED](#) | [CROSSREF](#)
46. Roberts LA, Beattie K, Close GL, Morton JP. Vitamin C consumption does not impair training-induced improvements in exercise performance. *Int J Sports Physiol Perform* 2011;6:58-69.
[PUBMED](#) | [CROSSREF](#)
47. Waśkiewicz A, Zujko ME, Szczeńniewska D, Tykarski A, Kwaśniewska M, Drygas W, Witkowska AM. Polyphenols and dietary antioxidant potential, and their relationship with arterial hypertension: a cross-sectional study of the adult population in Poland (WOBASZ II). *Adv Clin Exp Med* 2019;28:797-806.
[PUBMED](#) | [CROSSREF](#)
48. Bae I, Fan S, Meng Q, Rih JK, Kim HJ, Kang HJ, Xu J, Goldberg ID, Jaiswal AK, Rosen EM. BRCA1 induces antioxidant gene expression and resistance to oxidative stress. *Cancer Res* 2004;64:7893-909.
[PUBMED](#) | [CROSSREF](#)
49. Lee JM, Johnson JA. An important role of Nrf2-ARE pathway in the cellular defense mechanism. *J Biochem Mol Biol* 2004;37:139-43.
[PUBMED](#)
50. Talalay P, Dinkova-Kostova AT, Holtzclaw WD. Importance of phase 2 gene regulation in protection against electrophile and reactive oxygen toxicity and carcinogenesis. *Adv Enzyme Regul* 2003;43:121-34.
[PUBMED](#) | [CROSSREF](#)