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Association between dietary inflammatory index and phase angle in university employees: a cross-sectional study

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Phase angle (PhA), measured by bioelectrical impedance analysis, indicates cellular health, integrity, and function. As inflammation can damage cells, phase angle may be useful in detecting inflammatory status early. The relationship between dietary inflammatory index (DII) and PhA has not been studied yet. Therefore, we aimed to examine this association in Iranian adults. This cross-sectional study included 206 university employees. Dietary intakes were assessed by using a validated 86-item food frequency questionnaire (FFQ). Anthropometric indices and blood pressure were measured. A short form of the validated International Physical Activity Questionnaire (IPAQ) was used for evaluating physical activity. The PhA was measured by the Body Composition Analyzer Mc780 MA device. The mean age of participants was 43.50 ± 8.82 years and the range of DII score was − 4.66 to 0 among them. The highest tertile of DII compared to the lowest tertile, showed greater weight, WC, HC, basal metabolic rate (BMR), and diastolic blood pressure. We found no significant association between DII and PhA (crude model: OR: 0.68; 95% CI 0.34, 1.33, fully-adjusted model: OR: 0.65; 95% CI 0.26, 1.64). Also, after BMI stratification this association remained (fully-adjusted: normal weight: OR: 0.61; 95% CI 0.11, 3.27; Overweight and obese: OR: 0.57; 95% CI 0.16, 1.98). Having a higher DII score was not associated with a lower PhA. Further well-controlled prospective studies are warranted.

Keywords Dietary inflammatory index, Phase angle, Adult, University employees

Bioelectrical Impedance Analysis (BIA) is an indirect method to evaluate body composition, through the correlation between impedance and body water content. The relationship between resistance (R) and reactance (Xc) vectors given by BIA makes the phase angle (PhA)[1]. Compared to other methods, this is a fast, safe, cheap, and non-invasive method that is used to determine the body composition and nutritional status of patients and healthy people[2]. In fact, PhA shows the electrical integrity of body membranes and reflects various cellular indicators and hydration status[3]. Low PhA indicates a decrease in the integrity of the cell membrane, poor muscle function, and strength[2,4]. In general, age, sex, and body mass index (BMI) are the most important determinants of PhA in healthy individuals[5,6]. This index is capable of assessing inflammatory status and has recently been validated as a screening tool to predict inflammatory processes associated with malnutrition[7] and chronic conditions including cancer[8], sarcopenia[9], kidney and heart diseases[10].

Dietary Inflammatory Index (DII) is a valid measurement to classify individuals' diets according to their inflammatory potential that was first used in 2009 and updated in 2014[11]. Chronic inflammation is related to a wide range of diseases and diet plays an important role in modulating inflammation[12]. Recently, the adoption of Western dietary habits in Asian countries, along with the rising consumption of sugar, sodium, saturated fats, and trans fats, has contributed to the growing prevalence of inflammatory diseases among them[11,13]. Therefore, DII can be used as a tool to measure the inflammatory potential of diet in different populations to predict levels of inflammatory markers such as C-reactive protein (CRP)[14] and Interleukin 6 (IL-6) or anti-inflammatory

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biomarkers such as Interleukin 10 (IL-10)[11,15]. Several recent studies have examined the correlation between DII and various inflammatory diseases[16–19].

According to the results of a prospective study conducted on hemodialysis patients, PhA may be reliable in the detection of changes in inflammatory parameters over time[10]. Furthermore, PhA can inversely be associated with IL-6, TNF-α and CRP in elderly women[20] and it was shown that obese women in the lower tertile of PhA had higher fat mass, glucose level, IL-6, and leptin[21]. Detopoulou et al. showed a dietary pattern rich in potatoes and animal proteins was a significant determinant of PhA among male patients with non-small-cell lung cancer[22]. Another study indicated that PhA may be a practical and efficient measure in the clinical follow-up of IBD patients, as it is associated with bilirubin levels and antioxidant enzymes[23]. Also, a cross-sectional study in Brazilian individuals revealed higher dietary quality and muscle mass decreased the odds for low phase angle[24]. However, no studies have been conducted to determine the relationship between PhA, and DII in Asian countries, especially Iran. Therefore, the purpose of the present study was the investigate the correlation between PhA and DII among university employees.

Methods and material

Study design and population

We applied the standard formula to calculate the sample size, considering the type I error (α) of 0.05 and type II error (β) of 0.20 and r = 0.27[25]. Due to the possibility of dropouts (10%), the number of 125 people was considered, and for the possibility of comparison between the two sexes, the final sample size was determined to be 250 people. Although the sample size was calculated to be 250, this single-center cross-sectional study, included 258 adult participants (18–60 years old) who were selected by an easy non-random sampling method. The participants were healthy adults who worked on the campus of Isfahan University of Medical Sciences, Isfahan, Iran in 2022 They had no history of adherence to a specific diet (very low-calorie, ketogenic, vegetarian, and athlete diets) or use of special medicine therapy (glucocorticoids and regular use of non-steroidal anti-inflammatory drugs (NSAIDs)). Pregnant or lactating women, and people with a history of metabolic diseases such as diabetes, liver or kidney diseases, and hypertension were excluded from the study. Also, total energy intake of < 800 or > 4200 kcal/day was considered as exclusion criteria. All eligible participants were informed about the purpose and method of the study and written informed consent was obtained from them. The study included a visit for anthropometric measures, PhA determinations, and a semi-quantitative food frequency questionnaire (FFQ) to evaluate the possible relationship between PhA and DII. Demographic data and medical history of participants were collected through a questionnaire. The unwillingness of participants to cooperate was considered in different stages of the project. The study was approved by the Ethical Committee of Isfahan University of Medical Sciences (IR.ARI.MUI.REC.1402.021).

Assessment of dietary intakes and physical activity

The food intake of the participants was evaluated using a validated semi-quantitative 86-item food frequency questionnaire (FFQ)[26]. The participants were asked to specify the frequency of their consumption of each item according to the food servings in the last year. Depending on the type of food consumption, the frequency of consumption daily, weekly, or monthly was asked, and it was completed by a trained nutritionist. Final portion sizes were converted to grams per day using household measures. Then, dietary intakes were analyzed using NUTRITIONIST 4 (First Data Bank, San Bruno, CA) software to calculate total energy and nutrient intakes. Participants' physical activity was assessed by using a short form of the validated International Physical Activity Questionnaire (IPAQ)[27].

Calculation of the dietary inflammatory index

We used a method described by Shivappa et al. for scoring DII. They found that 45 specific nutrients and foods affect the concentrations of some inflammatory or anti-inflammatory biomarkers, such as IL-6, interleukin-1β (IL-1β), tumor necrosis factor-α (TNF-α), CRP, IL-10, and interleukin-4 (IL-4)[11]. The dietary intakes in our study were adjusted for energy using the residual method[28]. The DII score was calculated for 28 food items including total energy, carbohydrate, fat, protein, total dietary fiber, cholesterol, mono-unsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), saturated fats (SFAs), n-3 fatty acids, n-6 fatty acids, thiamin, riboflavin, niacin, pyridoxine, folic acid, cobalamin, vitamin A, C, D, E, β-carotene, zinc, iron, selenium, magnesium, caffeine, tea. A z-score for all 28 food items was calculated by subtracting the "global mean intake of the dietary parameter" from the "amount of food items" consumed by each subject and dividing this value by the "global standard deviation". Finally, all of the DII scores computed for individual food items were summed to create an "overall DII score" for each subject. A higher DII score demonstrated the more inflammatory potential of the diet.

Anthropometric measurements and phase angle

Anthropometric indices including weight, height, waist circumference (WC) and hip circumference (HP) were measured. Body weight (kg) was measured at 100 g precision by a Seca scale while subjects wore light clothing and no shoes. Height was measured at 0.5 cm precision with non-stretchable tape in a standing position. Then, BMI was calculated as weight in kg divided by the square of height in m. WC and HC were taken using a non-stretchable tape at 0.1 cm precision. Body composition, including fat mass, fat-free mass, muscle mass, bone mass, visceral fat and PhA were measured by the Body Composition Analyzer Mc 780 MA device.

Statistical analysis

The quantitative data were reported as mean ± SD/SE and qualitative data as frequency (percentage). Based on the participants' scores, they were categorized into tertiles of DII. One-way analysis of variance (ANOVA) and

chi-square tests were used to compare quantitative and categorical variables between tertiles of DII. By using analysis of covariance (ANCOVA), age-, sex- and energy-adjusted dietary macro and micronutrient intakes of participants were compared across tertiles of DII. The Kolmogorov–Smirnov test was applied to show the normality of variables. Pearson's coefficient was used to show a linear correlation between DII and PhA scores. Multivariable logistic regression was applied to identify the association between DII with PhA status. We used the median of PhA as a cut point for categorizing participants (healthy ≥ 5.6). The odds ratio (OR) and 95% confidence interval (CI) for PhA status were calculated in crude and adjusted models. In the first model, we adjusted for age, sex, and energy intake. In the second model, further adjustments were made for education level, history of drug and supplement use. In the third model, more adjustments for BMI were made. In the last model, physical activity was also adjusted. The first tertile of DII in all models was considered as the reference category. The overall trend of OR across increasing DII tertile was examined by considering the tertile of DII as a continuous variable. Stratified analysis was done to obtain OR for PhA status in different categories of BMI (normal weight vs. overweight & obese individuals). SPSS software version 23 (IBM, Chicago, IL) was used for analyses. P-values < 0.05 (two-sided) were considered as statistically significant.

Results

In total, 19 out of 258 participants were excluded from the analysis due to not completing the FFQ questionnaire. During the analysis, 32 participants with energy intake > 4200 kcal/day and 1 person with < 800 kcal/day were removed. This study included 206 adults with a mean age of 43.50 ± 8.82 years. The range of DII score was − 4.66 to 0 in participants. General characteristics, body composition, and anthropometric measurements of study participants across tertiles of dietary inflammatory index are displayed in Table 1. Adults in the highest tertile of DII compared to those in the lowest tertile had higher weight, WC, HC, Basal Metabolic Rate (BMR), and diastolic blood pressure (P < 0.05). There were no significant differences in other general characteristics, body composition, and anthropometric measurements among tertiles of DII.

Table 2 shows the dietary intakes of participants across tertiles of DII. Participants in the higher tertile of DII, in comparison to the lower tertile, had greater intake of energy, niacin, and iron (P < 0.05). While the

	Tertiles of DII			
	T1 (n = 68)	T2 (n = 69)	T3 (n = 69)	P-value[2]
Range	< − 3.59	− 3.59 to − 1.59	> − 1.59	
Sex, n (%)				
Male	17 (24.6)	24 (34.8)	20 (29)	0.423
Female	52 (75.4)	45 (65.2)	49 (71.0)	
Age (year)	42.78 ± 9.86	44.20 ± 7.36	43.58 ± 9.24	0.643
Biological age (year)	43.45 ± 13.25	46.97 ± 10.94	45.03 ± 11.79	0.233
Weight (kg)	66.96 ± 10.59	72.82 ± 12.76	70.46 ± 14.17	0.025
BMI (kg/m^2)	25.37 ± 3.33	26.62 ± 3.80	26.58 ± 4.80	0.126
WC (cm)	89.17 ± 10.15	94.27 ± 11.27	92.76 ± 12.45	0.028
HC (cm)	101.12 ± 7.48	105.12 ± 6.59	104.54 ± 8.01	0.004
Phase angle (°)	5.55 ± 0.63	5.62 ± 0.81	5.60 ± 0.74	0.838
Phase angle status				
< 5.6	40 (58.8)	36 (52.2)	34 (49.3)	0.518
≥ 5.6	28 (41.2)	33 (47.8)	35 (50.7)	
BMR (kcal)	1364.97 ± 257.18	1498.79 ± 299.69	1457.24 ± 287.53	0.019
Fat mass (kg)	20.72 ± 6.26	22.70 ± 5.93	22.39 ± 6.95	0.154
Fat free mass (Kg)	46.30 ± 8.02	50.12 ± 10.65	48.45 ± 10.26	0.072
Muscle mass (kg)	43.93 ± 7.65	47.59 ± 10.16	46.05 ± 9.75	0.070
Bone mass (kg)	2.65 ± 2.53	2.53 ± 0.50	2.74 ± 2.52	0.832
Visceral fat	6.52 ± 3.25	7.59 ± 3.05	7.37 ± 3.64	0.144
History of supplement use (%)	33 (50.8)	30 (45.5)	28 (41.2)	0.539
Physical activity level				
Low	17 (24.6)	16 (23.2)	19 (27.5)	0.846
Moderate	52 (75.4)	52 (75.4)	49 (71.0)	
Intense	0 (0)	1 (1.4)	1 (1.4)	
Systolic blood pressure (mmHg)	110.84 ± 9.58	115.79 ± 11.82	113.95 ± 11.59	0.096
Diastolic blood pressure (mmHg)	72.84 ± 7.69	76.95 ± 7.49	75.23 ± 6.80	0.027

Table 1. General characteristics, body composition and anthropometric measurements of study participants across tertiles of dietary inflammatory index (DII). *BMI* body mass index, *BMR* basal metabolic rate, *HC* hip circumference, *WC* waist circumference. [1]Values presented as Mean ± SD or Number (Percent). [2]P-value for one-way analysis of variance (ANOVA) and χ^2 test for quantitative and categorical variables, respectively.

	Tertiles of DII			
	T1 (n = 68)	T2 (n = 69)	T3 (n = 69)	P-value[2]
Range	< − 3.59	− 3.59 to − 1.59	> − 1.59	
Energy, kcal	2358.19 ± 86.31	2509.97 ± 86.23	2673.74 ± 87.44	0.039
Protein, g	82.46 ± 2.43	83.34 ± 2.40	78.20 ± 2.46	0.287
Carbohydrate, g	376.81 ± 6.84	368.95 ± 6.77	382.61 ± 6.93	0.369
Total fat, g	85.79 ± 2.89	86.02 ± 2.86	80.30 ± 2.93	0.296
Total fiber, g	30.11 ± 0.64	22.91 ± 0.63	18.15 ± 0.65	≤ 0.001
Cholesterol, mg	229.73 ± 15.21	252.76 ± 15.06	212.85 ± 15.41	0.179
SFA, g	19.82 ± 0.71	20.54 ± 0.70	19.39 ± 0.72	0.522
MUFA, g	25.63 ± 1.40	24.96 ± 1.39	22.09 ± 1.42	0.178
PUFA, g	26.42 ± 1.47	27.81 ± 1.45	27.09 ± 1.49	0.799
Total omega 3, g	0.71 ± 0.05	0.50 ± 0.05	0.38 ± 0.05	≤ 0.001
Vitamin C, mg	319.04 ± 10.92	204.61 ± 10.82	144.78 ± 11.07	≤ 0.001
Vitamin D mcg	1.13 ± 0.17	1.27 ± 0.17	0.86 ± 0.18	0.256
Vitamin A, RAE	1632.60 ± 88.49	990.98 ± 87.67	677.03 ± 89.71	≤ 0.001
β-carotene mcg	980.32 ± 77.31	452.04 ± 76.60	287.35 ± 78.37	≤ 0.001
Thiamin, mg	2.08 ± 0.05	2.11 ± 0.05	2.12 ± 0.05	0.84
Riboflavin, mg	2.17 ± 0.06	1.92 ± 0.06	1.59 ± 0.06	≤ 0.001
Niacin, mg	21.97 ± 0.64	24.14 ± 0.63	24.40 ± 0.65	0.017
Pyridoxine, mg	2.31 ± 0.05	1.91 ± 0.05	1.50 ± 0.05	≤ 0.001
Vitamin E, mg	4.79 ± 0.27	3.51 ± 0.27	3.19 ± 0.27	≤ 0.001
Folate, mcg	482.98 ± 17.03	361.18 ± 16.87	259.08 ± 17.27	≤ 0.001
Cobalamin, mcg	3.36 ± 0.21	3.24 ± 0.21	2.82 ± 0.21	0.175
Iron, mg	19.30 ± 0.72	21.14 ± 0.71	22.64 ± 0.73	0.006
Magnesium, mg	452.69 ± 12.12	347.52 ± 12.01	256.23 ± 12.29	≤ 0.001
Zinc, mg	10.71 ± 0.35	9.78 ± 0.35	8.11 ± 0.36	≤ 0.001
Selenium, mcg	105.68 ± 6.31	97.99 ± 6.26	84.99 ± 6.40	0.073
Sodium, mg	1360.78 ± 55.74	1256.09 ± 55.22	1199.86 ± 56.50	0.127
Potassium, mg	5298.01 ± 119.09	4095.48 ± 117.99	2972.85 ± 120.72	≤ 0.001
Calcium, mg	1174.35 ± 42.14	973.58 ± 41.75	847.03 ± 42.72	≤ 0.001
Phosphorus, mg	1477.67 ± 39.67	1284.22 ± 39.31	1100.45 ± 40.22	≤ 0.001
Black tea g	0.80 ± 0.05	0.67 ± 0.05	0.58 ± 0.05	0.030
Coffee, g	0.19 ± 0.03	0.23 ± 0.03	0.16 ± 0.03	0.405

Table 2. Dietary intakes of study participants across tertiles of dietary inflammatory index (DII). *MUFA* monounsaturated fatty acid, *PUFA* polyunsaturated fatty acid, *SFA* saturated fatty acid. [1]Values are Mean ± SE. Energy intake was adjusted for age and gender; all other values were adjusted for age, gender and energy intake. [2]P-value obtained from ANCOVA test for adjustment.

highest vs. lowest tertile of DII was associated with lower intake of total fiber, omega 3, vitamin C, vitamin A, β-carotene, riboflavin, pyridoxine, vitamin E, folate, magnesium, zinc, potassium, calcium, phosphorus, and black tea ($P < 0.05$).

The correlation between PhA and DII is presented in Table 3. There was no significant correlation between them ($r = -0.027$, $P = 0.705$).

Crude and multivariate adjusted odds ratio and 95% CI for PhA status across tertiles of DII without and with stratified by BMI categories are illustrated in Tables 4 and 5, respectively. In none of the crude and adjusted models, there was an association between PhA status and tertiles of DII in the top category of DII compared with the bottom level (crude model: OR: 0.68; 95% CI 0.34, 1.33, fully-adjusted model: OR: 0.65; 95% CI 0.26,

	Phase angle	
	r	p
Dietary inflammatory index (DII)	− 0.027	0.705

Table 3. Correlation between phase angle and dietary inflammatory index (DII). Obtained by the Pearson correlation coefficient.

	Tertiles of DII			
	T1 (n = 68)	T2 (n = 69)	T3 (n = 69)	P-trend
Phase angle status				
Cases (n)	40	36	34	
Crude	1 (Ref.)	0.76 (0.38, 1.50)	0.68 (0.34, 1.33)	0.264
Model 1	1 (Ref.)	1.01 (0.44, 2.32)	0.60 (0.26, 1.36)	0.224
Model 2	1 (Ref.)	0.89 (0.37, 2.14)	0.58 (0.24, 1.39)	0.222
Model 3	1 (Ref.)	1.00 (0.40, 2.49)	0.65 (0.26, 1.63)	0.366
Model 4	1 (Ref.)	0.99 (0.40, 2.48)	0.65 (0.26, 1.64)	0.371

Table 4. Multivariate odds ratio (OR) and 95% confidence interval (CI) for phase angle status across tertiles of DII. [1]All values are odds ratios and 95% confidence intervals. Model 1: Adjusted for age, sex, and energy intake. Model 2: Additionally, adjusted for education level, history of drug and supplement use. Model 3: Additionally, adjusted for BMI. Model 4: Additionally, adjusted for physical activity. [2]Obtained by the use of tertiles of DII as an ordinal variable in the model.

	Tertiles of DII			
	T1 (n = 68)	T2 (n = 69)	T3 (n = 69)	P-trend
Phase angle status				
Normal weight (participants/cases)	34/24	26/18	29/21	
Crude	1 (Ref.)	0.93 (0.30, 2.85)	1.09 (0.36, 3.28)	0.881
Model 1	1 (Ref.)	1.01 (0.23, 4.46)	0.69 (0.16, 2.95)	0.632
Model 2	1 (Ref.)	0.87 (0.16, 4.56)	0.67 (0.13, 3.31)	0.622
Model 3	1 (Ref.)	0.84 (0.16, 4.38)	0.61 (0.11, 3.27)	0.574
Overweight and obese (participants/cases)	33/16	43/18	40/13	
Crude	1 (Ref.)	0.76 (0.30, 1.90)	0.51 (0.19, 1.32)	0.165
Model 1	1 (Ref.)	0.96 (0.33, 2.80)	0.48 (0.16, 1.45)	0.191
Model 2	1 (Ref.)	1.11 (0.32, 3.84)	0.62 (0.18, 2.12)	0.411
Model 3	1 (Ref.)	1.01 (0.28, 3.58)	0.57 (0.16, 1.98)	0.347

Table 5. Multivariate odds ratio (OR) and 95% confidence interval (CI) for phase angle status across tertiles of DII, stratified by BMI categories. [1]All values are odds ratios and 95% confidence intervals. Model 1: Adjusted for age, sex, and energy intake. Model 2: Additionally, adjusted for education level, history of drug and supplement use. Model 3: Additionally, adjusted for physical activity. [2]Obtained by the use of tertiles of DII as an ordinal variable in the model.

1.64). This association remained after stratification based on BMI (fully-adjusted model: normal weight: OR: 0.61; 95% CI 0.11, 3.27; Overweight and obese: OR: 0.57; 95% CI 0.16, 1.98).

Discussion

In the present study, we found no significant association between DII and PhA in university employees, even after adjusting confounders. The range of DII among our participants was zero, and it may be a reason for finding no significant association. To our knowledge, this study is the first investigation of the association between DII and PhA in Iranian adults. However, the DII reflects a comprehensive view of the inflammatory components in the diet and has been used as an indicator for recognizing the association between diet and the risk of chronic diseases. A review study showed that lower PhA is correlated with higher CRP, TNF-α, IL-6, and IL-10 in general and aging populations, and patients with chronic diseases[29]. Another systematic review demonstrated that PhA is inversely associated with inflammatory markers in cardiovascular diseases (CVDs)[30]. Barrea et al. in a cross-sectional study on adults with Prader–Willi syndrome reported that PhA is a valid biomarker of low-grade systemic inflammation in them[31]. Lee et al. conducted a retrospective observation study on 221 patients who were admitted to ICU after abdominal surgery. They suggested that PhA can be used as a predictor of infection as it shows a significant association with inflammatory markers[32]. Another cross-sectional study revealed that the lowest values of PhA are significantly associated with the severity of obesity and 25(OH)D deficiency[33]. Severo et al. illustrated that PhA may be a practical measure in clinical follow-up of IBD patients, being associated with bilirubin levels and antioxidant enzymes[23]. All findings of the aforementioned studies are inconsistent with our study.

In our study, it was observed that higher DII is related to an increment of weight, WC, HC, BMR and diastolic blood pressure. Xie et al. showed greater pro-inflammatory dietary potential (higher DII) is associated with biological aging among 35,575 adult participants[34]. There was a positive correlation between DII and

hypertriglyceridemic waist circumference phenotype among women with overweight and obesity in a cross-sectional study. Therefore, DII may increase WC and triglyceride levels in them[35]. However, another study found no association between the DII and obesity indices (body weight, BMI, body fat, WC, and visceral fat)[36]. Neufcourt et al. in a cohort study reported DII is associated with higher systolic blood pressure values at baseline and with higher systolic and diastolic blood pressure values after 13-year follow-up[37].

We revealed that increased DII is associated with a higher intake of energy, niacin, and iron and a lower intake of some macro and micronutrients such as fiber, omega 3, and many antioxidants. Schoolchildren with a more inflammatory diet consume less carbohydrates and fiber and have a lower quality diet, according to Mora-Urda et al. study[38]. Another observational study demonstrated average energy, simple sugars, proportion of energy from fats, SFA, PUFA, and cholesterol intakes are significantly higher in greater tertile of DII. Conversely, the proportion of energy from protein, carbohydrates, and MUFA is significantly lower after increasing DII[39]. Also, there was an inverse association between DII scores and healthy eating index scores in the National Health and Nutrition Examination Survey on adults[40].

Although we could show no relation between DII scores and PhA, some possible mechanisms have been proposed for it. Inflammation and oxidative stress are associated with cellular damage and chronic disease through immune cell mobilization and inflammatory cytokine production[41]. These injuries can cause changes in cellular structures by oxidation of lipids, protein, and deoxyribonucleic acid, which prompts apoptosis. The PhA may reflect inflammation, as it measures cellular integrity and health. It is associated with body cell mass and hydration[29]. Lower PhA scores are associated with a lower body cell mass and imbalances in cellular water. This happens in disease states and might be because the extracellular fluid is more oxidative than the intracellular space[42].

The present study has some strengths. It was the first study that assessed the association between DII and PhA. Besides, we adjusted the main confounders to find an independent relationship and the study population was homogenous. Nevertheless, it had some limitations. The cross-sectional design cannot examine causality; so, conducting prospective studies is necessary. Although we adjusted our results for several confounders, other factors may also affect it. Moreover, recall bias and other potential reporting biases may have affected the results. In the present study, the PhA was measured once for each person, which is suggested to be measured 2–3 times and reported mean of it in future studies. However, we did it for three participants and observed no difference.

Conclusion

Our findings indicated that diet-induced inflammation is not associated with a lower PhA score. Because of the existing limitations, further prospective studies are warranted for precise investigation.

Data availability

The datasets generated and/or analysed during the current study are not publicly available as per the rules and regulations of the Isfahan University of Medical Science but are available upon reasonable request from the corresponding author.

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Author contributions

Mahsa Rezazadegan, Mahsa Shirani, Fatemeh Samadinian, Mojtaba Akbari and Fatemeh Shirani contributed to the conception, design, data collection, data interpretation, manuscript drafting and approval of the final version of the manuscript, and agreed on all aspects of the work.

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Competing interests

The authors declare no competing interests.

Ethics declarations

The study procedure was performed according to the declaration of Helsinki and the STROBE checklist. All participants provided informed written consent and the study protocol was approved by the Ethics Committee of Isfahan University of Medical Sciences (IR.ARI.MUI.REC.1402.021).

Additional information

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