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Association of Dietary and Lifestyle Inflammation Score with General and Central Obesity in Iranian Adults

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ABSTRACT

Background: Previous studies have suggested a positive association between a more pro-inflammatory diet and lifestyle- factors with obesity, separately.
Objective: Determine the relationship between the dietary and lifestyle inflammatory-score (DLIS) and general and abdominal obesity in adults.
Methods: We included 834 adults aged 18 to 59 years (69% female, mean age: 44.7 ± 10.7 years). Using a 168-item semi-quantitative food frequency questionnaire, we collected dietary-intakes. The DLIS was calculated by using the dietary inflammatory score, calculated by data from 18 components of the diet, and two components of the lifestyle including physical-activity and cigarette smoking. The odds ratio (OR) and 95% CI of the general and abdominal adiposity across quartiles of the DLIS were calculated by logistic-regression analysis after controlling for age, sex, marital status, occupation, education status, and energy intake.
Results: The DLIS ranged between -3.00 and 2.32 (man ± SD: -0.31 ± 0.99). The ORs of central obesity, as assessed by waist-to-hip ratio (≥0.8 for women and ≥1 for men) across quartiles of the DLIS were as follows: 1.91 (95%CI: 1.21, 3.02), 1.62 (95%CI: 1.03, 2.56), and 1.25 (95%CI: 0.79, 1.97) for the second, third, and fourth quartiles, respectively. The results for weight-to-height ratio (>0.5) were 2.26 (95%CI: 1.24, 4.11), 1.08 (95%CI: 0.63, 1.86), and 1.38 (95%CI: 0.79, 2.39) for the second, third, and fourth quartiles, respectively. Higher DLIS was not associated with increased waist-circumference and general-obesity as assessed by body mass index.
Conclusion: Having a diet and lifestyle with more pro-inflammatory properties may be associated with abdominal-adiposity.
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Introduction

Obesity, a chronic condition, is a consequence of the accumulation of fat stores in the body resulting from a positive energy balance. Body mass index (BMI) is the most accepted tool for defining obesity and accordingly, BMI values of ≥30 were defined as obesity (1). Today, about one-third of the world's population in developing and developed countries suffer from obesity (2). The prevalence of obesity among Iranian adults is reported to be 21.7 % (3). Obesity is

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a major risk factor for chronic diseases such as cardiovascular disease, cancer and diabetes (4) and as a results is responsible for a large part of health care costs (5).

The adiposity is a major driver of low-grade chronic-inflammation (6). The association between adiposity and non-communicable chronic disease risk is mediated, in part, by low-grade systemic inflammation (7). Obesity and low-grade chronic inflammation- a bridge between obesity and other chronic diseases- are a complex multifactorial phenomenon that are influenced by genetics, lifestyle, environmental and social factors. Of those, diet and lifestyle patterns play a major role in driving obesity and inflammation (8, 9). Low physical-activity and sedentary-lifestyle is a major risk-factors of adiposity (10) and inflammation (11). In addition, detrimental effects of cigarette smoking on inflammatory status has been established (12).

The associations of dietary habits with obesity and inflammation have been investigated in several observational studies. Current evidence suggests that higher intake of some food groups such as fruit, nuts, legumes, whole grains, and red meat (13); or higher adherence to healthy dietary patterns (14) may be associated with the risk of adiposity. There is also a strong link between dietary habits and low-grade inflammation (15, 16).

In recent years, dietary inflammatory index (DII) (17) and empirically diet inflammatory pattern (EDIP) (18) have been developed to evaluate the inflammatory properties of the diet. To reflect the joint contribution of diet and lifestyle to inflammation, a new dietary and lifestyle inflammation score (DLIS) have been developed (19) to account for important inflammation related exposures including dietary habits, physical activity, cigarette-smoking, obesity, and alcohol-drinking.

It is recently hypothesized that there might be a bidirectional link between adiposity and low grade systemic inflammation. Several observational studies have suggested a positive association between a more pro-inflammatory diet, as assessed by DII, and adiposity (20-22). To expand their perspective and evaluate the potential association between the new inflammation-score with adiposity, we performed a population-based cross-sectional study to investigate the association of the DLIS with likelihood of general and abdominal-adiposity in Iranian adult populations.

Materials and Methods

Ethical approval

Prior to entering the study, written information regarding the background and procedures of the study was presented to participants and then, written informed consent was obtained from each participant. This study was performed in accordance with the Declaration of Helsinki and approved by the ethical committee of the Tehran University of Medical Sciences (Ethic Number: IR.TUMS.VCR.REC.1397.157), with the support of the Tehran University of Medical Sciences (Grant number: 40186).

Participants

We recruited 850 adults who attended the health care centers in five districts of Tehran, Iran from 2018 to 2019. Eligibility criteria were as follows: healthy adults, aged between 18 to 59 years, living in Tehran, who had the willingness to take part in the study.

The following formula was used for sample size calculation: n = (pqz2)/E2. Considering the total prevalence of 65% for overweight and obesity (23), an error coefficient of d=0.04 and at α level of 0.05, the sample size of 546 people was obtained. With a design effect of 1.5 and to compensate for the potential exclusion of participants due to under- and over-reporting of total energy intake, or attrition due to other reasons, the

final sample size of 850 participants was selected for inclusion. Initially, eight health centers were randomly selected from each district (North, West, East, South, and center) justifiably spread all over Tehran in Iran. Then, the total number of participants (850) was divided by the total number of health centers (40) to obtain the number of participants per health center. Finally, 834 individuals were included in the analyses, after excluding participants who had at least one incomplete variable.

Demographic factors

At first visit in each health center and by using pre-specified data extraction forms, trained interviewers obtained information about age, sex (male, female), education (illiterate, under diploma, diploma, educated), marriage status (single, married, divorced), smoking status (never smoker, former smoker, light smoker, heavy smoker), and occupation (employee, housekeeper, retired, unemployed).

Physical activity

By using the validated International Physical Activity Questionnaire (IPAQ), we assessed physical-activity. Data were expressed as a metabolic equivalent minute per week (MET-minute /week) (24) and accordingly, participants were categorized into three groups including very low (<600 MET-minute/week), low (600-3000 MET-minute/week), and moderate and high physical activity (>3000 MET-minute/week).

Anthropometric and blood pressure assessments

The weight, height, waist (WC), and hip circumference (HP) of the study participants were measured by trained dietitians. Subjects were weighted with slight clothing, by an adult's digital scale (808Seca, Germany) that its sensitivity was 0.1 kg. The subject's height was metered using a wall stadiometer with 0.1 cm precision, barefoot with relaxing shoulders (Seca, Germany). By using a flexible anthropometric tape, WC was measured by using an elastic anthropometric tape in the middle between the iliac crest and the lower rib margin. The HC was measured by the unstretched tape in the correct horizontal position at the extreme level from the sidelong facet over light clothing, without any force to body surface and with an accuracy of 0.1 cm. The BMI was calculated as weight in kg divided by height in meters squared. Waist-to-height ratio (WHtR) was gained by dividing the WC in centimeters by height in centimeters. Waist-to-hip ratio (WHR) was determined by dividing the WC in centimeter by HP in centimeter.

Definition of general and abdominal obesity

In this study, we considered BMI ≥30 kg/m^2 as the general obesity (according to WHO definition) (25). Abdominal obesity was defined as follows: WHtR ≥ 0.5 (26, 27), WHR ≥ 0.8 for women and WHR ≥ 1 for men (28), and WC ≥102 cm for men and ≥88cm for women (25).

Dietary assessment and calculating the dietary and lifestyle inflammation score (DLIS)

By using a semi-quantitative food frequency questionnaire, we obtained the average food intake in the past year for each participant (29). This 168-item questionnaire enclosed a list of groceries with a standard portion size for each food item. The frequency of consumption of each food (daily, weekly, monthly, and annual) was determined for each subject. By using household measures, portion sizes were then converted to grams per day. The codes were then allocated to the records and questionnaire data was entered into Nutritionist IV software based on the Iranian foods-modified US Department of Agriculture food composition database (30), and then food intake was gained based on the quantity of food consumed.

We used the method introduced by Byrd et al. to calculate the DLIS for each participant (19). The score includes dietary inflammation score (DIS) and lifestyle inflammation score (LIS). The DIS includes

19 dietary components including leafy greens and cruciferous vegetables, tomatoes, apples and berries, deep yellow or orange vegetables and fruit, other fruits and real fruit juices, other vegetables, legumes, fish, poultry, red and organ meats, processed meats, added sugars, high-fat dairy, low-fat dairy, coffee and tea, nuts and other fats, refined grains and starchy vegetables and supplement score. Of these, the supplement Score was removed due to lack of relevant information, and the DIS score was then calculated with the remaining 18 components. To calculate the weight of each component in the DIS, each dietary component was treated as a continuous variable (g/d) and then was standardized by sex, to a mean of 0 and SD of 1.0. Next, each dietary component was scored based on the strength of its association with an inflammation biomarker score in the REGARDS case-cohort study. Multivariable linear regression was used to estimate the maximum likelihood estimates for the βcoefficients, which represent the average change in the inflammation biomarker score per 1 SD increase in a dietary component. Each dietary component intake was multiplied by the weight (βcoefficient) and then summed to calculate the DIS.

The LIS included 4 components: smoking status, physical activity, alcohol intake, and obesity. Smoking status was divided into two groups: "current" or "former/never." Physical activity was divided into two groups: "high or moderate" and "low or no physical activity". Obesity was removed because it was part of the outcome. Alcohol intake was eliminated due to Iranians' religious beliefs. For the LIS, dummy variables were created for physical activity and smoking status, and then multivariable linear regression was applied to estimate the βcoefficients to represent the average change in the inflammation biomarker score per having a certain lifestyle behavior relative to its referent category. To calculate the DLIS, the DIS and LIS for each participant were summed, with the higher DLIS indicating a more pro-inflammatory diet and lifestyle and low DLIS indicating a more anti-inflammatory diet and lifestyle.

Statistical methods

By using the Statistical Package for the Social Sciences (SPSS version 16; SPSS Inc), statistical analyses were done. $P< 0.05$ was considered as a statistically significant level. The DLIS was presented as quartile and then characteristics of the study participants were presented accordingly. Quantitative variables were reported as mean and standard deviation (SD) and qualitative variables as frequency (percent). Chi-square test was applied to compare the frequency of qualitative variables across the DLIS quartiles and Analysis of Variance (ANOVA) was used to compare the means of quantitative variables. Values of anthropometric measures across the DLIS quartiles were adjusted for age, sex, marriage status, occupation, and education using an analysis of covariance (ANCOVA). Odds Ratios (OR) and 95% confidence intervals (CI) of general and abdominal adiposity across quartiles of the DLIS were determined through binary logistic regression in three target models: a crude model which was unadjusted, model 1 that controlled for sex, age, and energy intake, and model 2 that adjusted further for marriage history, and occupation and education status.

Results

Of the initial 850 participants enrolled in the study, 16 participants were removed due to insufficient data for one of the variables, which yielded 834 participants (68.7% female) for the present study. The mean ± SD of age and BMI of participants were 44.7 ± 10.7 years and 27.9 ± 5.6 kg/m^2, respectively. The range of DLIS was -3.00 to 2.32 (mean ± SD: -0.31±0.90). The prevalence of general obesity (BMI≥30) was 28.2%. Prevalence of central obesity based

on WHtR, WHR, and WC were 81.1%, 42.2%, and 48.6%, respectively.

Table 1. Sociodemographic characteristics of Iranian adults according to quartile of the dietary and lifestyle inflammation score (n =834).

Characteristics	**Q1 (n=207) (most anti-inflammatory) -3.00 to -0.88**	**Q2 (n=208) -0.87 to -0.27**	**Q3 (n=211) -0.26 to 0.31**	**Q4 (n=208) (most pro-inflammatory) 0.32 to 2.32**	**P-value***
Age (years)	45.2±10.8	44.2±10.8	44.9±10.7	44.4±10.6	0.61
Sex (%male)	37.2%	27.9%	32.7%	27.4%	0.10
Marriage status (%married)	79.2%	84.6%	83.4%	76.0%	0.09
Smoking status (%Current)	2.4%	5.8%	4.3%	7.8%	0.03
Occupation (%)					0.64
Employee	24.2%	28.4%	23.7%	26.9%	
Housekeeper	58.9%	50.5%	57.8%	57.7%	
Retired	14.5%	17.3%	16.1%	11.5%	
Unemployed	2.4%	3.8%	2.4%	3.8%	
Physical activity (%)					0.78
No physical activity	63.8%	63.5%	64.5%	62.5%	
Moderate or heavy	36.2%	36.7%	35.5%	37.5%	
Education (%university graduated)	38.2%	32.7%	29.9%	36.1%	0.29

Abbreviation: Q, quartile.
The Values are presented as mean±SD for continuous variables and percent for categorical variables.
**ANOVA test was used for quantitative data and chi-square test for qualitative data. P<0.05 is significant.*

Sociodemographic characteristics of the study participants across quartiles of the DLIS are presented in Table 1. The proportion of current smokers increased in the top quartile of the DLIS compared with the lowest quartile. There were no significant differences across quartiles of the DLIS in terms of other factors.

Anthropometric measures of the study participants across quartiles of the DLIS are shown in Table 2. Values of anthropometric measures w*ere compared* by *ANCOVA test* controlling for age, sex, marriage status, occupation, and education status**.** Anthropometric measures did not diff*er significantly along with the increase in the DLIS.*

Dietary intake of participants according to quartiles *of th***e** *DLIS* are indicated in Table 3. The intake of added sugar, processed meats, refined grains, and starchy vegetables increased significantly along with the increase in the DLIS. In contrast, intakes of monounsaturated fats, leafy greens and cruciferous vegetable, tomatoes, apples and berries, deep yellow or orange vegetables and fruit, other fruits and real fruit juices, other vegetables, legumes, fish, poultry, red and organ meats, high-fat dairy, low-fat dairy, coffee and tea, nuts and other fats decreased proportionally across quartiles of the DLIS. No other significant difference was observed in terms of *daily food consumption* among quartiles of the DLIS.

The association of the DLIS with general and abdominal adiposity are presented in Table 4. The ORs of central obesity, as assessed by WHR (≥0.8 for women and ≥1 for men)

across quartiles of the DLIS in the fully adjusted model were 1.91 (95%CI: 1.21, 3.02), 1.62 (95%CI: 1.03, 2.56), and 1.25 (95%CI: 0.80, 1.97) for the second, third, and fourth quartiles, respectively (P for trend = 0.52). The results for WHtR (>0.5) were 2.26 (95%CI: 1.24, 4.11), 1.08 (95%CI: 0.63, 1.86), and 1.38 (95%CI: 0.79, 2.39) for the second, third, and fourth quartiles, respectively (P for trend = 0.77). Higher DLIS was not associated with general adiposity (as assessed by BMI) and increased WC, neither in crude nor in the adjusted model.

Table 2. Anthropometric measures of Iranian adults according to quartile of the dietary and lifestyle inflammation score (n =834).

Characteristics*	Q1 (n=207) (most anti-inflammatory) -3.00 to -0.88	Q2 (n=208) -0.87 to -0.27	Q3 (n=211) -0.26 to 0.31	Q4 (n=208) (most pro-inflammatory) 0.32 to 2.32	P-value**
Weight (kg)	75.2±14.0	71.8±12.8	73.7±13.6	73.3±13.9	0.222
Height (cm)	163±8.76	162±9.22	162±8.82	162±8.79	0.519
WC (cm)	91.8±12.2	93.0±13.3	92.2±12.4	91.2±11.8	0.397
WHtR (cm)	0.562±0.080	0.575±0.09	0.567±0.080	0.564±0.079	0.221
WHR	0.880±0.085	0.890±0.087	0.891±0.084	0.887±0.187	0.401
BMI (kg/m2)	28.0±4.83	27.6±7.23	27.7±4.50	28.01.7±5.47	0.748

Abbreviations: BMI, body mass index; Q, quartile; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio.
**The Values are presented as mean±SD.*
*** Obtained from ANCOVA test controlling for* age, sex, marriage status, occupation and education. P<0.05 is significant.

Table 3. Daily food consumption according to quartile of the dietary and lifestyle inflammation score for the participants (n =834).

Daily consumption DIS	Q1(N=207) (most anti -inflammatory) -3.00 to -0.88	Q2(N=208) -0.87 to -0.27	Q3(N=211) -0.26 to 0.31	Q4(N=208) (most pro-inflammatory 0.32 to 2.32	P-value*
Energy (kcal/d)	2630±1312	2651±3501	2450±949	2537±1117	0.43
Carbohydrate (g/d)	383±191	414±880	364±148	377±181	0.63
Protein (gr/d)	89.8±41.6	86.0±68.3	85.9±38.0	83.8±38.5	0.23
Total fat (g/d)	88.1±64.9	79.8±54.2	78.7±40.1	82.4±42.7	0.25
Fiber (g/d)	19.0±11.2	36.7±177	18.4±10.2	19.6±12.1	0.54
PUFA (g/d)	18.0±16.4	16.4±11.4	16.6±10.1	17.5±12.5	0.76
MUFA (g/d	30.3±48.8	24.0±21.0	23.8±13.8	25.0±14.0	0.06
Leafy greens & cruciferous vegetables (g/d)	38.4±29.3	32.8±27.1	27.2±21.8	25.4±24.9	<0.001
Tomatoes (g/d)	42.3±49.4	27.6±28.3	21.8±24.3	15.0±14.2	<0.001
Apples and berries (g/d)	70.4±83.5	31.5±77.2	29.7±79.5	14.3±20.4	<0.001
Deep yellow or orange vegetables and fruit (g/d)	95.1±96.5	38.8±39.3	32.5±41.2	23.5±32.8	<0.001
Other fruits and real fruit juices (g/d)	407±364	251 ±260	230±441	153±160	<0.001
Other vegetables (g/d)	52.3±73.6	24.6±42.3	17.8±29.5	17.9±22.3	<0.001
Legumes (g/d)	74.0±117	63.5±78.9	50.8±44.5	46.8±35.3	<0.001
Fish (g/d)	1.37±9.23	0.34±2.00	0.26±1.30	0.23±1.07	0.01
Poultry (g/d)	48.8±84.9	48.9±96.2	20.9±55.9	15.3±50.4	<0.001
Red and organ meats (g/d)	312±341	274±464	234±293	204±193	<0.001
Processed meats (g/d)	19.6±18.0	19.1±16.2	22.0±34.6	30.9±28.1	<0.001
Added sugars (g/d)	652±655	588±342	630±463	936±1298	<0.001
High-fat dairy (g/d)	341±283	258±367	218±244	208±334	<0.001
Low-fat dairy (g/d)	22.1±28.1	18.8±28.9	13.6±15.3	15.6±23.6	0.001
Coffee and tea (g/d)	8.09±24.2	6.30±15.3	3.75±8.70	4.38±20.1	0.01
Nuts (g/d)	29.9±36.7	15.5±17.2	16.2±37.1	8.90±13.0	<0.001
Other fats (g/d)	32.8±46.9	16.6±23.5	15.7±23.1	17.3±31.6	<0.001
Refined grains and starchy vegetables (g/d)	486+382	445+271	468+253	596+358	<0.001

Data are expressed as mean ± SD.
Abbreviations: Q, quartile.
** Obtained from ANCOVA test controlling for* age, sex, marriage status, occupation and education. P<0.05 is significant.

Table 4. Association of the dietary and lifestyle inflammation score with general and abdominal adiposity and in Iranian adults (n=834).

Variable	Q1 (N=207) (most anti -inflammatory) -3.00 to -0.88	Q2 (N=208) -0.87 to -0.27	Q3 (N=211) -0.26 to 0.31	Q4 (N=208) (most pro -inflammatory 0.32 to 2.32	P trend *
Increased WC (≥88 cm for women and ≥102 cm for men)					
Crude	1.0	1.29 (0.88-1.90)	1.19 (0.81-1.74)	1.03 (0.70-1.51)	0.99
Model 1	1.0	1.32 (0.86-2. 04)	1.21 (0.74-1.87)	0.96 (0.62-1.49)	0.76
Model 2	1.0	1.34 (0.86-2.09)	1.19 (0.77-1.84)	0.98 (0.63-1.51)	0.78
Increased WHR (≥0.8 for women and ≥1 for men)					
Crude	1.0	1.84 (1.24-2.73)	1.56 (1.05-2.32)	1.35 (0.90-2.01)	0.26
Model 1	1.0	1.93 (1.23-3.02)	1.66 (1.06-2.61)	1.24 (0.79-1.95)	0.54
Model 2	1.0	1.91 (1.21-3.02)	1.62 (1.03-2.56)	1.25 (0.79-1.97)	0.52
Increased WHtR (≥0.5)					
Crude	1.0	1.68 (1.00-2.83)	1.02 (0.63-1.64)	1.13 (0.00-1.84)	0.93
Model 1	1.0	2.18 (1.23-3.88)	1.12 (0.66-1.89)	1.30 (0.76-2.23)	0.85
Model 2	1.0	2.26 (1.24-4.11)	1.08 (0.63-1.86)	1.38 (0.79-2.39)	0.77
General obesity (BMI≥30 kg/m^2)					
Crude	1.0	0.80 (0.52-1.23)	0.95 (0.62-1.44)	0.86 (0.56-1.32)	0.68
Model 1	1.0	0.79 (0.51-1.24)	0.95 (0.62-1.47)	0.85 (0.55-1.32)	0.68
Model 2	1.0	0.79 (0.50-1.25)	0.93 (0.60-1.44)	0.86 (0.55-1.33)	0.67

Abbreviations: BMI, body mass index; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio**.**
Model 1 adjusted for sex, age , and energy intake.
Model 2 adjusted further for marriage status, occupation and education*.*
Obtained from binary logistic analysis. P<0.05 is significant.

Discussion

In this cross-sectional study of adults in Tehran, no significant association was found between adherence to a pro-inflammatory diet and lifestyle with general-obesity. However, a significant positive association was observed between a more pro-inflammatory diet and lifestyle with abdominal obesity, as assessed by WHR, either in the crude or in the maximally adjusted model. There was also a significant association between the DLIS and increased WHtR, even after adjusting for potential confounder variables.

Existing evidence suggests that adiposity (31), especially central adiposity (32), is a strong driver of low-grade systemic inflammation. Abdominal deposition of visceral fat may play an underlying role in these situations, because visceral adipose tissue secretes many bioactive substances, including pro- and anti-inflammatory proteins (33). Adipose tissue is a metabolically active endocrine organ that has a high percentage of adipocytes and macrophages. These cells produce inflammatory cytokines such as C-reactive protein (CRP) (34).

However, it is recently suggested that there might be a bidirectional relationship between adiposity and inflammation (35), which may lead to a vicious cycle of positive feedback (36). Recent cross-sectional investigations have suggested a positive association between the inflammatory potential of the diet and the prevalence of general and abdominal-adiposity (20, 37-40). There is evidence that oxidative stress can induce an inflammatory response via activation of transcription factors (41) and the production

of cytokines, like TNF-α(42). Semicarbazide-sensitive amine oxidase is an antioxidant enzyme found in adipocytes and is highly activated in obese subjects (43). Higher activity of this antioxidant enzyme, resulting from oxidative stress, can induce adipose tissue glucose uptake (43) and adipocyte differentiation (44). Also, low-grade inflammation can cause insulin resistance by transcriptional mechanisms (45). It is purposed that insulin resistance and hyperinsulinemia can trigger obesogenic pathways in adipose tissue (46, 47).

Previous research has examined the association of single components of the DLIS with general and abdominal adiposity. A cross-sectional study has shown that a Western dietary pattern, characterized by high consumption of refined grains, red and processed meat, and soft drinks, independent of energy intake, was associated with a higher prevalence of general and abdominal-obesity (48). A cross-sectional study on a similar population in Tehran indicated that higher adherence to a Western-style dietary pattern including high energy-adjusted intake of refined grains, red and processed meat, and high-fat dairy products, had a strong positive association with general and abdominal obesity (49). A meta-analysis of cross-sectional studies has also indicated that higher red and processed meat consumption may increase the risk of general and abdominal obesity (50).

These findings were supported by the findings of recent investigations that reported a positive association between inflammatory properties of the diet and obesity (20, 37, 38, 40). These findings suggest that the inflammatory properties of the diet are one of the biological pathways that may mediate the association of dietary habits and adiposity.

About smoking, the results of previous studies indicated that current smokers were at higher risk for general (51-53) and abdominal obesity (54-57). However, there is inconsistent evidence (58). The association between cigarette smoking and adiposity may be explained by the fact that smoking increases secretion of cortisol, a stress hormone (59) and insulin resistance (60, 61), as well as by the anti-estrogenic effect of smoking (62). The present findings suggest that the association between smoking and adiposity may be mediated by inflammatory pathways. There is also evidence that supports the anti-inflammatory effects of physical-activity and moderate exercise (63, 64). It is indicated that higher sedentary time was associated with a higher level of inflammation markers (65).

In the present study, we found that adopting a more pro-inflammatory diet and lifestyle was associated with a higher odds of central adiposity. It is proposed that central obesity is a better predictor of increased inflammatory biomarkers in plasma than is general-obesity (66). Since the BMI may underestimate the actual amount of obesity and body fat stores (67); therefore, how fat is distributed in the body and central deposition of fat may be more important than general-obesity (68, 69).

Strengths of this study are: All measurements were accurate because the collection of the information was done by expert dietitians, by using valid and reliable questionnaires and cut-offs; relatively large sample size; and using a new index that considers important inflammation-related lifestyle components to estimate the joint inflammatory properties of the diet and lifestyle.

However, our study has some limitations that must be considered in interpreting the results. Because the study design was cross-sectional, the temporal sequence and causal relationship could not be determined.

Besides, in this study, 18 dietary parameters out of 19 parameters were available for calculating the DIS, and one parameter, which was the supplementary score, was missed. Also from four lifestyle items to calculate the LIS, two factors were not

considered. Alcohol consumption was removed due to the religious beliefs of the Iranian people regarding the prohibition of alcohol consumption. Another one was BMI, which was not included because it was part of the study outcome. The error of measurement and wrong classification of participants cannot be separated from the study. Despite adjusting several distortions in this study, the potential effects of the remaining confounders were uncontrollable.

Conclusions

In conclusion, the present study indicated that higher adherence to a more pro-inflammatory diet and lifestyle, reflected by a higher DLIS, was associated with a higher likelihood of central adiposity. However, no association was found between the DLIS and general-obesity. Further studies with the prospective design are needed to confirm the present findings.

References

1. Identification EPot, Overweight To, Adults Oi, Heart N, Lung, Institute B, et al. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report: National Institutes of Health, National Heart, Lung, and Blood Institute; 1998.
2. Chooi YC, Ding C, Magkos F. The epidemiology of obesity. Metabolism. 2019;92:6-10.
3. Sarokhani D, Sarokhani M, Dehkordi AH, Gheshlagh RG, Fakhri M. Prevalence of obesity and overweight in Iranian students: a systematic review and meta-analysis. Journal of Pediatric Endocrinology and Metabolism. 2020;1(ahead-of-print).
4. Visscher TL, Seidell JC. The public health impact of obesity. Annual review of public health. 2001;22(1):355-75.
5. Tremmel M, Gerdtham U-G, Nilsson PM, Saha S. Economic burden of obesity: a systematic literature review. International journal of environmental research and public health. 2017;14(4):435.
6. Karczewski J, Śledzińska E, Baturo A, Jończyk I, Maleszko A, Samborski P, et al. Obesity and inflammation. European cytokine network. 2018;29(3):83-94.
7. Furman D, Campisi J, Verdin E, Carrera-Bastos P, Targ S, Franceschi C, et al. Chronic inflammation in the etiology of disease across the life span. Nature medicine. 2019;25(12):1822-32.
8. Minihane AM, Vinoy S, Russell WR, Baka A, Roche HM, Tuohy KM, et al. Low-grade inflammation, diet composition and health: current research evidence and its translation. British Journal of Nutrition. 2015;114(7):999-1012.
9. Beavers KM, Nicklas BJ. Effects of lifestyle interventions on inflammatory markers in the metabolic syndrome. Frontiers in bioscience (Scholar edition). 2011;3:168.
10. Wiklund P. The role of physical activity and exercise in obesity and weight management: Time for critical appraisal. Journal of Sport and Health Science. 2016;5(2):151-4.
11. Burini RC, Anderson E, Durstine JL, Carson JA. Inflammation, physical activity, and chronic disease: An evolutionary perspective. Sports Medicine and Health Science. 2020.
12. Lee J, Taneja V, Vassallo R. Cigarette smoking and inflammation: cellular and molecular mechanisms. Journal of dental research. 2012;91(2):142-9.
13. Schlesinger S, Neuenschwander M, Schwedhelm C, Hoffmann G, Bechthold A, Boeing H, et al. Food groups and risk of overweight, obesity, and weight gain: a systematic review and dose-response meta-analysis of prospective studies. Advances in Nutrition. 2019;10(2):205-18.
14. Rezagholizadeh F, Djafarian K, Khosravi S, Shab-Bidar S. A posteriori healthy dietary patterns may decrease the risk of central obesity: findings from a systematic review and meta-analysis. Nutrition Research. 2017;41:1-13.
15. Schwingshackl L, Hoffmann G. Mediterranean dietary pattern, inflammation and endothelial function: a systematic review and meta-analysis of intervention trials. Nutrition, Metabolism and Cardiovascular Diseases. 2014;24(9):929-39.
16. Barbaresko J, Koch M, Schulze MB, Nöthlings U. Dietary pattern analysis and biomarkers of low-grade inflammation: a systematic literature review. Nutrition reviews. 2013;71(8):511-27.
17. Shivappa N, Steck SE, Hurley TG, Hussey JR, Hébert JR. Designing and developing a literature-derived, population-based dietary inflammatory index. Public health nutrition. 2014;17(8):1689-96.
18. Tabung FK, Smith-Warner SA, Chavarro JE, Wu K, Fuchs CS, Hu FB, et al. Development and validation of an empirical dietary inflammatory index. The Journal of nutrition. 2016;146(8):1560-70.
19. Byrd DA, Judd SE, Flanders WD, Hartman TJ, Fedirko V, Bostick RM. Development and validation of novel dietary and lifestyle inflammation scores. The Journal of Nutrition. 2019;149(12):2206-18.

20. Ruiz-Canela M, Zazpe I, Shivappa N, Hébert JR, Sánchez-Tainta A, Corella D, et al. Dietary inflammatory index and anthropometric measures of obesity in a population sample at high cardiovascular risk from the PREDIMED (PREvencion con DIeta MEDiterranea) trial. British journal of nutrition. 2015;113(6):984-95.
21. Mirmajidi S, Izadi A, Saghafi-Asl M, Vahid F, Karamzad N, Amiri P, et al. Inflammatory potential of diet: association with chemerin, omentin, lipopolysaccharide-binding protein, and insulin resistance in the apparently healthy obese. Journal of the American College of Nutrition. 2019;38(4):302-10.
22. Shivappa N, Steck SE, Hurley TG, Hussey JR, Ma Y, Ockene IS, et al. A population-based dietary inflammatory index predicts levels of C-reactive protein in the Seasonal Variation of Blood Cholesterol Study (SEASONS). Public health nutrition. 2014;17(8):1825-33.
23. Kiadaliri AA, Jafari M, Mahdavi M-RV, Faghihzadeh S, Kalantari N, Asadi-Lari M. The prevalence of adulthood overweight and obesity in Tehran: findings from Urban HEART-2 study. Medical journal of the Islamic Republic of Iran. 2015;29:178.
24. Ainsworth BE, Haskell WL, Herrmann SD, Meckes N, Bassett Jr DR, Tudor-Locke C, et al. 2011 Compendium of Physical Activities: a second update of codes and MET values. Medicine & science in sports & exercise. 2011;43(8):1575-81.
25. Organization WH. Obesity: preventing and managing the global epidemic: World Health Organization; 2000.
26. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 0· 5 could be a suitable global boundary value. Nutrition research reviews. 2010;23(2):247-69.
27. Ashwell M, Hsieh SD. Six reasons why the waist-to-height ratio is a rapid and effective global indicator for health risks of obesity and how its use could simplify the international public health message on obesity. International journal of food sciences and nutrition. 2005;56(5):303-7.
28. WhO O. Preventing and managing the global epidemic. Report of a WHO consultation on obesity Geneva: WHO. 1997:17-40.
29. 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 nutrition. 2010;13(5):654-62.
30. Haytowitz D, Lemar L, Pehrsson P, Exler J, Patterson K, Thomas R, et al. USDA national nutrient Lung Cellular and Molecular Physiology. 2004;286(2):L344-L53. database for standard reference, release 24. US Department of Agriculture: Washington, DC, USA. 2011.
31. Das U. Is obesity an inflammatory condition? Nutrition. 2001;17(11-12):953-66.
32. Berg AH, Scherer PE. Adipose tissue, inflammation, and cardiovascular disease. Circulation research. 2005;96(9):939-49.
33. Matsuzawa Y. Therapy insight: adipocytokines in metabolic syndrome and related cardiovascular disease. Nature clinical practice Cardiovascular medicine. 2006;3(1):35-42.
34. Lago F, Dieguez C, Gómez-Reino J, Gualillo O. The emerging role of adipokines as mediators of inflammation and immune responses. Cytokine & growth factor reviews. 2007;18(3-4):313-25.
35. Moreno-Aliaga M, Campión J, Milagro F, Berjón A, Martínez J. Adiposity and proinflammatory state: the chicken or the egg. Adipocytes. 2005;1:1-16.
36. Gregor MF, Hotamisligil GS. Inflammatory mechanisms in obesity. Annual review of immunology. 2011;29:415-45.
37. San KMM, Fahmida U, Wijaksono F, Lin H, Zaw KK, Htet MK. Chronic low grade inflammation measured by dietary inflammatory index and its association with obesity among school teachers in Yangon, Myanmar. Asia Pacific Journal of Clinical Nutrition. 2018;27(1):92.
38. Shakeri Z, Mirmiran P, Khalili-Moghadam S, Hosseini-Esfahani F, Ataie-Jafari A, Azizi F. Empirical dietary inflammatory pattern and risk of metabolic syndrome and its components: Tehran Lipid and Glucose Study. Diabetology & metabolic syndrome. 2019;11(1):16.
39. Oliveira TMS, Bressan J, Pimenta AM, Martínez-González M-Á, Shivappa N, Hébert JR, et al. Dietary inflammatory index and prevalence of overweight and obesity in Brazilian graduates from the Cohort of Universities of Minas Gerais (CUME project). Nutrition. 2020;71:110635.
40. Ramallal R, Toledo E, Martínez JA, Shivappa N, Hébert JR, Martínez-González MA, et al. Inflammatory potential of diet, weight gain, and incidence of overweight/obesity: The SUN cohort. Obesity. 2017;25(6):997-1005.
41. Rahman I, MacNee W. Role of transcription factors in inflammatory lung diseases. Thorax. 1998;53(7):601-12.
42. Brown D, Donaldson K, Borm P, Schins R, Dehnhardt M, Gilmour P, et al. Calcium and ROS-mediated activation of transcription factors and TNF-α cytokine gene expression in macrophages exposed to ultrafine particles. American Journal of Physiology-
43. Yu PH, Wang M, Fan H, Deng Y, Gubisne-Haberle D. Involvement of SSAO-mediated

deamination in adipose glucose transport and weight gain in obese diabetic KKAy mice. American Journal of Physiology-Endocrinology and Metabolism. 2004;286(4):E634-E41.
44. MERCIER N, MOLDES M, EL HADRI K, FÈVE B. Semicarbazide-sensitive amine oxidase activation promotes adipose conversion of 3T3-L1 cells. Biochemical Journal. 2001;358(2):335-42.
45. Shoelson SE, Lee J, Goldfine AB. Inflammation and insulin resistance. The Journal of clinical investigation. 2006;116(7):1793-801.
46. Noakes TD. So what comes first: the obesity or the insulin resistance? And which is more important? : Clinical Chemistry; 2018.
47. Erion KA, Corkey BE. Hyperinsulinemia: a cause of obesity? Current obesity reports. 2017;6(2):178-86.
48. Paradis A-M, Godin G, Pérusse L, Vohl M-C. Associations between dietary patterns and obesity phenotypes. International journal of obesity. 2009;33(12):1419-26.
49. Esmaillzadeh A, Azadbakht L. Major dietary patterns in relation to general obesity and central adiposity among Iranian women. The Journal of nutrition. 2008;138(2):358-63.
50. Rouhani M, Salehi-Abargouei A, Surkan P, Azadbakht L. Is there a relationship between red or processed meat intake and obesity? A systematic review and meta-analysis of observational studies. Obesity Reviews. 2014;15(9):740-8.
51. Dare S, Mackay DF, Pell JP. Relationship between smoking and obesity: a cross-sectional study of 499,504 middle-aged adults in the UK general population. PloS one. 2015;10(4):e0123579.
52. Dvorak RD, Del Gaizo AL, Engdahl RM, Eliason CJ. Tobacco use and body mass index: mediated effects through physical inactivity. Journal of health psychology. 2009;14(7):919-23.
53. Schoenberg NE, Huang B, Seshadri S, Tucker TC. Trends in cigarette smoking and obesity in Appalachian Kentucky. Southern medical journal. 2015;108(3):170-7.
54. Kim Y, Jeong SM, Yoo B, Oh B, Kang H-C. Associations of smoking with overall obesity, and central obesity: a cross-sectional study from the Korea National Health and Nutrition Examination Survey (2010-2013). Epidemiology and health. 2016;38.
55. Chiolero A, Faeh D, Paccaud F, Cornuz J. Consequences of smoking for body weight, body fat distribution, and insulin resistance. The American journal of clinical nutrition. 2008;87(4):801-9.
56. Clair C, Chiolero A, Faeh D, Cornuz J, Marques-Vidal P, Paccaud F, et al. Dose-dependent positive association between cigarette smoking, abdominal obesity and body fat: cross-sectional data from a population-based survey. BMC public health. 2011;11(1):23.
57. Canoy D, Wareham N, Luben R, Welch A, Bingham S, Day N, et al. Cigarette Smoking and Fat Distribution in 21, 828 British Men and Women: A Population-based Study. Obesity research. 2005;13(8):1466-75.
58. Gasperin LdOF, Neuberger M, Tichy A, Moshammer H. Cross-sectional association between cigarette smoking and abdominal obesity among Austrian bank employees. BMJ open. 2014;4(7).
59. Direk N, Newson RS, Hofman A, Kirschbaum C, Tiemeier H. Short and long-term effects of smoking on cortisol in older adults. International Journal of Psychophysiology. 2011;80(2):157-60.
60. Facchini FS, Hollenbeck CB, Jeppesen J, Chen Y-DI, Reaven G. Insulin resistance and cigarette smoking. The Lancet. 1992;339(8802):1128-30.
61. Mouhamed DH, Ezzaher A, Neffati F, Douki W, Gaha L, Najjar M, editors. Effect of cigarette smoking on insulin resistance risk. Annales de Cardiologie et d'Angéiologie; 2016: Elsevier.
62. Tankó LB, Christiansen C. An update on the antiestrogenic effect of smoking: a literature review with implications for researchers and practitioners. Menopause. 2004;11(1):104-9.
63. Ertek S, Cicero A. Impact of physical activity on inflammation: effects on cardiovascular disease risk and other inflammatory conditions. Archives of medical science: AMS. 2012;8(5):794.
64. Beavers KM, Brinkley TE, Nicklas BJ. Effect of exercise training on chronic inflammation. Clinica chimica acta. 2010;411(11-12):785-93.
65. Falconer C, Cooper A, Walhin J, Thompson D, Page A, Peters T, et al. Sedentary time and markers of inflammation in people with newly diagnosed type 2 diabetes. Nutrition, Metabolism and Cardiovascular Diseases. 2014;24(9):956-62.
66. Hermsdorff HHM, Zulet MÁ, Puchau B, Martínez JA. Central adiposity rather than total adiposity measurements are specifically involved in the inflammatory status from healthy young adults. Inflammation. 2011;34(3):161-70.
67. Shah NR, Braverman ER. Measuring adiposity in patients: the utility of body mass index (BMI), percent body fat, and leptin. PloS one. 2012;7(4):e33308.
68. Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. Obesity reviews. 2012;13(3):275-86.
69. Cornier M-A, Despres J-P, Davis N, Grossniklaus DA, Klein S, Lamarche B, et al. Assessing adiposity: a scientific statement from the American Heart Association. Circulation. 2011;124(18):1996-2019.