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Macronutrient intake and physical activity levels in individuals with and without metabolic syndrome: An observational study in an urban population

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Short Communication

Abstract

BACKGROUND: We aimed to compare dietary macronutrient intake and physical activity level (PAL) between community-based samples of Iranian adults with metabolic syndrome (MetS+) and without metabolic syndrome (MetS-).

METHODS: This cross-sectional study was conducted among 3800 men and women aged 35-65 years. The International Diabetes Federation (IDF) criteria were used to define MetS. A 24-hour recall was used to evaluate dietary intake. The James and Schofield human energy requirements equations were used to calculate PAL and questions were categorized into time spent on activities during work (including housework), during non-work time, and in bed.

RESULTS: The mean ± standard deviation (SD)age of the MetS+ and MetS- subjects was, respectively, 48.8 ± 7.8 years (521 men and 1178 women) and 47.6 ± 7.5 years (714 men and 1222 women) (P = 0.930). The mean energy intake was higher in the MetS+ men compared with MetS- men (1977.4 ± 26.6 vs. 1812.7 ± 21.7 Kcal; P < 0.001). Crude and energy-adjusted intake from total fat was lower in MetS+ women compared with MetS- women (both P ≤ 0.010). PALs were lower in MetS+ compared with MetS- participants (P < 0.001). After adjusting for confounders, no significant association was observed between the intake of individual macronutrients and MetS. In contrast, PAL was inversely associated with the incidence of MetS [OR = 0.34 (95% CI: 0.17-0.57); P < 0.001].

CONCLUSION: In the current study, there was an inverse relationship between PAL and the risk of MetS, but no association between individual dietary macronutrients intake and the incidence of MetS.

Keywords: Nutritional Assessment, Basal Metabolic Rate, Physical Activity

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Introduction

Metabolic syndrome (MetS) as defined by the International Diabetes Federation (IDF) criteria is characterized by waist circumference ≥ 80 and ≥ 94 in women and men, respectively, and 2 or more of the following criteria: increased blood pressure, impaired glucose tolerance, hypertriglyceridemia,

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and low concentrations of serum high-density lipoprotein (HDL) cholesterol.[1] MetS is an important risk factor for mortality and morbidity associated with several chronic conditions including cardiovascular disease (CVD), diabetes mellitus (DM), and some cancers.[2-6] In American adults, the prevalence of MetS has been reported as 34%.[7] The number of Iranian women with MetS (55%) is higher compared to men (30.1%).[8]

Several factors are known to contribute to the etiology of MetS.

Several studies have showed that lifestyle factors such as an inappropriate diet and physical activity level (PAL) have a key role in the development of MetS.[2,9] An energy restricted diet including a suitable distribution of macronutrients coupled with moderate PAL is associated with an improvement in several risk factors of MetS and it has been shown that it delays the beginning of complications.[10]

A macronutrient diet evidently has an important effect on obesity via affecting several metabolic processes, appetite, and thermogenesis.[11] Additionally, macronutrient oxidation and total energy expenditure in obese subjects can be affected by changes in the content of the diet.[11]

Some studies have reported that a high carbohydrate intake is associated with low levels of serum high-density lipoprotein cholesterol (HDL-C) and increased plasma triacylglycerol concentration.[12,13] Moreover, Kim et al. suggested that both the quantity and quality of carbohydrate intake have a positive relationship with the risk of MetS in women, although this relationship was dependent on body mass index (BMI).[14] Additionally, there are several studies that support a relationship between a high-fat diet (HFD) and the presence of obesity; this may be due to hyperphagia,[15,16] or a reduction in sensitivity to satiety hormones (e.g., cholecystokinin).[17] A HFD is also more likely to result in a positive energy balance.[18] HFD-related postprandial insulin resistance (IR) is importantly mediated by impairment of parasympathetic-dependent insulin action, which is associated with adiposity.[19] A sedentary lifestyle with a low PAL also plays a vital role in the constellation of risk factors associated with MetS.[20,21] Adequate leisure-time physical activity is important in preventing MetS phenotypes.[20] Exercise reduces abdominal adiposity, improves insulin action and HDL-C levels, and reduces the risk of type 2 diabetes even without weight reduction.[22,23]

Despite the fact that the association between several aspects of diet and components of MetS (obesity, high blood pressure, dyslipidemia, and glucose intolerance) has been investigated, few observational studies that have evaluated the association of macronutrient intake and physical activity levels with the presence of MetS. Furthermore, the results of the limited data that have been published are not conclusive and cannot be extrapolated to the Iranian population. Therefore, the aim of this study was to assess the relationship between macronutrient intake and physical activity levels, and the presence of MetS in order to provide evidences for lifestyle modification as an important factor related to MetS.

Materials and Methods

A population of 3800 subjects aged 35-65 years was selected from Mashhad Stroke and Heart Atherosclerotic Disorders (MASHAD: 2010-2020) Study, Mashhad, Iran, using stratified-cluster method.[24-27] The following subjects were excluded: pregnant women, individuals with CVD, diabetes mellitus, or other metabolic disease, and those taking dietary supplements. The definition of MetS was based on the definition of the IDF.[1] Each participant provided a written informed consent form, and the study was approved by the ethics committee of Mashhad University of Medical Sciences, Iran.

Anthropometric parameters (weight, height, and waist circumference) were measured using standard protocols on which detailed information is presented in previous studies.[24-27] Blood pressure (BP) was measured twice 30 minutes apart in the seated position in a participant at rest for at least 15 minutes using a standard mercury sphygmomanometer calibrated according to the Iranian Institute of Standards and Industrial Research. BMI was calculated as weight (kg) divided by height squared (m^2).

More information on measurements is provided elsewhere.[24-27] A full fasting lipid profile, comprising total cholesterol, triglycerides, HDL-C, and low-density lipoprotein cholesterol (LDL-C), was determined for each participant. Serum lipid and fasting blood sugar (FBS) concentrations were measured using enzymatic methods.

A 24-hour recall questionnaire was used to collect information on food and drink consumed over the last 24 hours.[2] To assess the nutritional intake of the subjects, Dietplan6 software (Forestfield Software Ltd., UK) was used to analyze macronutrient and micronutrient intake. The total energy intake and the values of crude and energy-

adjusted intake of all macronutrients were reported in this study. Macronutrients were considered as a percentage of total caloric intake.

PAL was evaluated by a physical activity questionnaire. PAL was calculated as the total energy expenditure (TEE) as a ratio of the basal metabolic rate (BMR) over a 24-hour period.[2] The questions on physical activity were divided into time spent on activities during work, during non-work time, and in bed.[28] Physical activity level at work was scored using an ascending scale of intensity.[29] The sum of all scores constitutes the overall PAL. The participants were divided into quartiles within total PAL.

SPSS software (version 16.0, SPSS Inc., Chicago, IL, USA) was used to analyze the data. The histogram and Kolmogorov-Smirnov test were applied to check the normal distribution of continuous variables. Categorical variables were compared using chi-square test. Continuous data with normal distribution were presented as mean and standard deviation (SD) or median and interquartile range in the case of skewed distribution, and categorical data were presented as frequency or percentage (%). Nutrient intake adjustment for total energy intake was done via residual method.[30] Energy-adjusted nutrient intakes were obtained as the residuals from the regression model in which absolute nutrient intake and total energy intake were considered as the dependent and independent variable, respectively.[30] Qualitative variables were compared using chi-square test. The normally distributed data (data were presented as mean ± SD) of the participants with and without MetS were compared using Student's t-test. For variables with non-normal distribution (data was presented as the median and interquartile range) Mann-Whitney test was applied.

Logistic regression was applied to calculate the odds ratios (ORs) and 95% confidence intervals (CIs) for MetS (lowest quintile considered as that reported by Freire et al.[30]). Models were corrected for sex, age, smoking, PAL, total energy, BMI, and past medical history. Trend analysis was performed by assigning the median intake for each nutrient to individuals, then, considering it as a continuous variable in logistic regression.[30] A two-sided $P \leq 0.050$ was used to characterize significant results.

Results

Of the 3800 (aged 35 to 65 years) participants in the final analytic sample, 1699 (44%) had MetS. The prevalence of MetS was 42% (n = 521) in men and 49% (n = 1178) in women (P = 0.001). MetS+ participants were older than MetS- participants, but age differences reached significance only in women (48.7 vs. 46.9 years; P < 0.001). Systolic BP (SBP), diastolic BP (DBP), waist girth, blood glucose, total cholesterol and triglycerides, uric acid, and C-reactive protein (CRP) (all P < 0.001) were significantly higher and HDL cholesterol was significantly lower (P < 0.001) in both men and women with MetS compared with those without MetS. Furthermore, MetS+ men had higher BMI than their MetS- counterparts (128.9 vs. 118.8 kg/m^2; P < 0.001), while the MetS+ women group had higher LDL cholesterol (126.4 vs. 123.5 mg/dl; P = 0.040) and higher prevalence of smokers (21% vs. 14%; P < 0.001) compared with the MetS- women group (Table 1). Significant gender × MetS interactions were apparent for smoking, and HDL and LDL cholesterol.

The mean values of total energy intake and crude intake of all macronutrients were higher in men with MetS than in those without MetS (all P < 0.001). However, there were no differences in energy-adjusted intake of macronutrients between the two groups (P > 0.050). Surprisingly, the crude intake of total fat (P < 0.010), saturated fatty acid (P < 0.050), and monounsaturated fatty acid (P < 0.010) was higher in women without MetS. Energy-adjusted intake of total fat was significantly different between MetS+ and MetS- subgroups in women (P = 0.010). Gender × MetS interactions were significant for crude intake of total fat, and crude and total energy-adjusted intake of poly-unsaturated fatty acid.

Once correction (sex, age, PAL, smoking, past medical history, energy intake, and BMI) was carried out, it appears that the likelihood of having MetS was no longer significant for the intake of macronutrients (P > 0.050 for all) (Table 2). The ORs of MetS decreased across quartiles of PAL with the top quartile of PAL being associated with an OR of 0.34 (95% CI: 0.17-0.57) relative to the lowest quartile (Table 3).

Discussion

PALs were lower in the subjects with MetS compared subjects without MetS. However, adjusted total macronutrient intake had no significant association with the presence of MetS.

MetS+ subjects had higher levels of serum high sensitivity C-reactive protein (hs-CRP) and uric acid concentration.

Table 1. Demographic, cardiometabolic factors, and daily intake characteristics of subjects with and without metabolic syndrome

Characteristic	Total			Men			Women			Gender* MetS interaction P
	MetS+ (n = 1699)	MetS− (n = 1936)	P	MetS+ (n = 521)	MetS− (n = 714)	P	MetS+ (n = 1178)	MetS− (n = 1222)	P	
Education level			0.750			0.625			0.382	0.732
< 1 year (%)	299 (17.60)	292 (15.08)		40 (7.0)	49 (6.8)		259 (21.9)	243 (19.9)		
1-8 years (%)	822 (48.30)	897 (46.30)		218 (41.8)	288 (40.3)		604 (51.4)	609 (49.9)		
> 8 years (%)	578 (34.00)	747 (38.50)		263 (50.4)	377 (52.9)		315 (26.7)	370 (30.2)		
Current cigarette smoking			0.440			0.396			< 0.001	< 0.050
Yes (%)	389 (22.80)	387 (20.00)		139 (28.0)	209 (24.0)		250 (21.0)	178 (14.0)		
Physical activity level (%)			< 0.001			< 0.010			< 0.010	0.192
Sedentary (%)	1319 (77.63)	1118 (57.70)		415 (80)	416 (58.2)		904 (76.0)	702 (58.0)		
Low activity (%)	289 (17.01)	561 (28.97)		73 (14.0)	164 (23.2)		216 (18.0)	397 (33.0)		
Active (%)	68 (4.00)	196 (10.10)		21 (4.1)	88 (12.4)		47 (3.0)	108 (9.0)		
Very active (%)	20 (1.10)	59 (3.00)		10 (1.9)	44 (6.2)		10 (0.8)	15 (1.2)		
Age	48.79 ± 7.93	47.66 ± 7.65	< 0.001	49.10 ± 7.6	48.90 ± 8.00	0.694	48.70 ± 8.06	46.90 ± 7.30	< 0.001	0.939
Weight (kg)	75.97 ± 12.35	68.40 ± 12.35	< 0.001	83.40 ± 11.0	72.70 ± 30.01	< 0.001	72.60 ± 11.40	66.41 ± 11.90	0.901	0.321
Waist circumference (cm)	100.72 ± 9.78	91.06 ± 12.10	< 0.001	101.80 ± 6.9	89.50 ± 10.80	< 0.001	100.20 ± 10.70	91.90 ± 12.60	< 0.001	0.462
Body mass index (kg/m2)	29.77 ± 4.13	26.70 ± 4.40	< 0.001	28.80 ± 3.3	25.50 ± 3.20	< 0.001	30.10 ± 4.30	27.52 ± 4.66	0.811	0.553
Systolic blood pressure (mm Hg)	128.98 ± 19.43	116.13 ± 15.25	< 0.001	128.90 ± 17.1	118.80 ± 15.50	< 0.001	128.90 ± 20.30	114.50 ± 14.80	< 0.001	0.762
Diastolic blood pressure (mm Hg)	83.95 ± 11.71	76.23 ± 10.21	< 0.001	85.20 ± 10.5	78.40 ± 10.20	< 0.001	83.30 ± 12.10	74.90 ± 9.90	< 0.001	0.144
Blood glucose (mg/dl)	88.10 ± 25.10	80.08 ± 14.95	< 0.001	87.92 ± 22.7	81.13 ± 15.27	< 0.001	88.17 ± 23.90	79.40± 4.70	< 0.001	0.545
Cholesterol (mg/dl)	198.57 ± 38.77	188.38 ± 37.50	< 0.001	190.60 ± 37.8	184.50 ± 37.90	0.005	200.60 ± 38.70	190.60 ± 37.30	< 0.001	0.366
High-density lipoprotein cholesterol (mg/dl)	37.96 ± 7.56	44.77 ± 10.24	< 0.001	34.96 ± 6.4	40.39 ± 8.66	< 0.001	40.66 ± 7.33	47.30 ± 10.20	< 0.001	< 0.050
Low-density lipoprotein cholesterol (mg/dl)	123.93 ± 34.29	122.10 ± 32.09	0.110	118.40 ± 32.7	119.70 ± 31.80	0.514	126.39 ± 34.70	123.50 ± 32.10	0.042	< 0.050
Triglyceride (mg/dl)	185.42 ± 92.67	112.52 ± 58.45	< 0.001	179.0 (IQR 144-236)	109.0 (IQR 79-150)	< 0.001	166.0 (IQR 119-210)	97.0 (IQR 73-125)	< 0.001	0.474
Uric acid (mg/dl)	4.92 ± 2.17	4.42 ± 1.25	< 0.001	5.62 ± 1.3	5.17 ± 1.20	< 0.001	4.61 ± 2.30	3.9±1.02	< 0.001	0.072
High sensitive C-reactive protein (mg/l)	1.8 (IQR 1.12-3.47)	1.3 (IQR 0.83-2.52)	0.110	1.4 (IQR 1.01-2.7)	1.3 (IQR 0.8-2.3)	< 0.001	2.1 (IQR 1.2-3.8)	1.3 (IQR 0.9-2.6)	< 0.001	< 0.050
Energy (kcal)	1651.65 ± 586.67	1644.06 ± 560.53	0.590	1977.40 ± 26.6	1812.70 ± 21.70	< 0.001	1465.30± 13.40	1473.00 ± 13.80	0.632	0.122
Protein										
Crude intake (g)	60.41 ± 24.74	59.51 ± 24.70	0.270	71.40 ± 1.1	65.90 ± 0.90	< 0.001	55.40 ± 0.65	55.70 ± 0.70	0.802	0.469
Total energy adjusted (g)	59.72 ± 15.44	59.06 ± 16.16	0.210	59.90 ± 0.8	59.90 ± 0.60	0.994	59.60 ± 0.40	58.50 ± 0.40	0.703	0.338

Table 1. Demographic, cardiometabolic factors, and daily intake characteristics of subjects with and without metabolic syndrome (continue)

Characteristic	Total			Men			Women			Gender* MetS interaction P
	MetS+ (n = 1699)	MetS− (n = 1936)	P	MetS+ (n = 521)	MetS− (n = 714)	P	MetS+ (n = 1178)	MetS− (n = 1222)	P	
Total energy (%)	14.79 ± 3.76	14.58 ± 3.93	0.110	13.60 ± 0.1	13.7 ± 0.1	0.740	13.80 ± 0.10	13.50 ± 0.10	0.021	0.229
Carbohydrate										
Crude intake (g)	232.31 ± 96.36	230.25 ± 94.53	0.510	282.07 ± 4.6	258.80 ± 3.70	< 0.001	210.10 ± 2.41	213.60 ± 2.50	0.312	0.322
Total energy adjusted (g)	230.65 ± 39.75	229.73 ± 41.40	0.490	231.30 ± 2.1	232.90 ± 1.60	0.544	230.30 ± 1.03	228.00 ± 1.10	0.213	0.265
Total energy (%)	55.93 ± 9.83	55.57 ± 10.23	0.270	55.30 ± 0.4	55.30 ± 0.30	0.768	54.20 ± 0.20	53.50 ± 0.20	0.604	0.140
Total fat										
Crude intake (g)	59.99±25.07	60.42 ± 24.58	0.600	70.50 ± 1.1	64.30 ± 0.90	< 0.001	55.20 ± 0.60	58.03 ± 0.60	0.004	< 0.050
Total energy adjusted (g)	59.06±15.28	59.74 ± 16.14	0.190	58.60 ± 0.7	58.04 ± 0.60	0.118	59.20 ± 0.40	60.60 ± 0.40	0.012	0.124
Total energy (%)	32.85±8.57	33.39 ± 8.96	0.060	31.10 ± 0.3	31.00 ± 0.30	0.108	32.00 ± 0.20	33.00 ± 0.20	0.009	0.262
Saturated fatty acid										
Crude intake (g)	16.20±8.27	16.49 ± 8.00	0.270	18.90 ± 0.4	17.90 ± 0.30	0.039	14.90 ± 0.20	15.60 ± 0.20	0.041	0.233
Total energy adjusted (g)	16.62±6.42	16.98 ± 6.41	0.080	16.50 ± 0.3	16.90 ± 0.20	0.188	16.60 ± 0.10	16.90 ± 0.10	0.202	0.882
Monounsaturated fatty acid										
Crude intake (g)	16.30±7.39	16.29 ± 7.41	0.970	16.20 ± 0.3	15.90 ± 0.20	< 0.001	14.50 ± 0.20	14.70 ± 0.20	0.042	0.124
Total energy adjusted (g)	16.03±4.97	16.09 ± 5.46	0.720	16.24 ± 0.3	15.98 ± 0.20	0.114	15.90 ± 0.10	16.10 ± 0.10	0.165	0.951
Poly unsaturated fatty acid										
Crude intake (g)	20.72±10.88	20.64 ± 10.69	0.820	23.90 ± 0.5	21.20 ± 0.30	< 0.001	18.80 ± 0.30	19.30 ± 0.30	0.015	< 0.050
Total energy adjusted (g)	20.48±8.59	20.50 ± 8.81	0.960	20.10 ± 0.4	19.20 ± 0.30	0.074	20.60 ± 0.20	21.20 ± 0.20	0.085	< 0.050
Cholesterol										
Crude intake (g)	204.74 ± 179.76	207.09 ± 177.56	0.690	252.30 ± 9.9	236.40 ± 7.10	0.181	183.60 ± 4.30	189.80 ± 4.70	0.342	0.786
Total energy adjusted (g)	203.10 ± 169.06	206.19 ± 169.08	0.580	217.90 ± 9.5	218.60 ± 6.90	0.955	196.60 ± 4.10	198.70 ± 4.50	0.729	0.816

* P < 0.050 is significant. Values are expressed as mean ± SD for normally distribution data and median with interquartile range of non-normally distributed data. The Student's t-test is used for comparison of normally distributed data and Mann-Whitney test for comparison of non-normally distribution data between men with and without MetS and women with and without MetS.

MetS: Metabolic syndrome; MetS+: with metabolic syndrome; MetS−: without metabolic syndrome; IQR: Interquartile range; SD: Standard deviation

Macronutrient intakes are reported as a percentage of the total energy.

Table 2. Odds ratios of metabolic syndrome across quintiles of energy-adjusted macronutrients intake

Nutrients	Quintiles of intake					P
	Q1	Q2	Q3	Q4	Q5	
Protein						0.182
Median intake (g/d)	41.1	52.2	58.9	66.0	78.20	
Range of intake	< 47.7	47.8-55.7	55.7-62.1	62.1-70.5	> 70.5	
OR	1	0.79 (0.64-0.99)	0.96 (0.77-1.02)	0.92 (0.74-1.15)	0.85 (0.68-1.06)	
Carbohydrate						0.666
Median intake (g/d)	181.2	211.5	230.2	248.5	279.08	
Range of intake	< 199.7	199.8-221.7	221.8-238.8	238.9-260.4	> 260.5	
OR	1	0.78 (0.62-0.97)	1.05 (0.84-1.31)	0.97 (0.78-1.21)	0.94 (0.75-1.17)	
Fat						0.376
Median intake (g/d)	40.1	52.0	58.9	66.5	78.30	
Range of intake	< 47.0	47.0-55.5	55.5-62.8	62.8-71.5	> 71.5	
OR	1	1.24 (0.99-1.55)	1.10 (0.88-1.37)	1.04 (0.84-1.30)	0.97 (0.78-1.23)	
Saturated fatty acid						0.092
Median intake (g/d)	10.0	13.5	16.0	18. 9	24.70	
Range of intake	< 12.0	12.0-14.8	14.8-17.3	17.3-21.0	> 21.00	
OR	1	1.05 (0.84-1.31)	1.06 (0.85-1.32)	0.89 (0.71-1.11)	0.88 (0.70-1.09)	
Monounsaturated fatty acid						0.545
Median intake (g/d)	9.9	13.6	16.0	23.2	26.20	
Range of intake	< 12.2	12.2-14.9	14.9-17.1	17.1-45.9	> 45.90	
OR	1	1.21 (0.97-1.52)	1.20 (0.96-1.49)	1.22 (0.98-1.53)	1.07 (0.86-1.33)	
Polyunsaturated fatty acid						0.958
Median intake (g/d)	9.9	16.1	20.2	24.2	31.30	
Range of intake	< 13.3	13.3-18.4	18.4-22.0	22.0-27.0	> 27.00	
OR	1	1.10 (0.88-1.37)	0.98 (0.79-1.22)	1.06 (0.85-1.32)	1.02 (0.82-1.27)	
Niacin						0.889
Median intake (g/d)	57.1	113.7	158.5	232.8	391.20	
Range of intake	< 89.8	89.8-135.6	135.7-186.1	186.2-312.3	> 312.50	
OR	1	0.98 (0.79-1.23)	0.98 (0.78-1.22)	0.86 (0.69-1.07)	1.04 (0.84-1.30)	

Data are expressed as median intake, range of intake, and OR (95% CI) and are adjusted for sex, age, physical activity level, smoking, past medical history, energy intake, and BMI.
ORs: Odds ratios; CI: Confidence interval; BMI: Body mass index

Chen et al. found a significant positive relationship between serum concentration of hs-CRP and the occurrence of MetS.[31] Furthermore, Konishi et al. reviewed the association between hs-CRP and MetS in epidemiological studies and reported that serum hs–CRP, as a main component of MetS, is generally increased in obese, hypertensive, and diabetic subjects as well as in subjects with low HDL-cholesterol and high triglyceride.[32] Oda and Kawai showed that hs-CRP could be considered as an inflammatory component of MetS in Japanese individuals.[33]

In agreement with our results, several other studies found that serum uric acid was associated with MetS and its related risk factors.[34-42] Zhang et al. suggested that uric acid levels may be an independent risk marker for obesity, hypertension, and dyslipidemia, as the main components of MetS.[43] Another study showed an association between hyperuricemia and increased risk of MetS.[34]

Table 3. Odds ratios of metabolic syndrome across quartiles of physical activity level

Physical activity	Quartiles of PAL				P
	Q1	Q2	Q3	Q4	
PAL					< 0.001
Median	1.22	1.47	1.69	2.06	
Range	(0.00-1.40)	(1.40-1.59)	(1.60-1.89)	(1.90-36.95)	
OR	1	0.64 (0.50-0.81)	0.45 (0.29-0.66)	0.34 (0.17-0.67)	

Data are expressed as median intake, range of intake, and OR (95% CI) and are adjusted for sex, age, smoking, past medical history, energy intake, and BMI.
ORs: Odds ratios; CI: Confidence interval; BMI: Body mass index; PAL: Physical activity level

The results of a cross-sectional study among Korean Urban Rural Elderly (KURE) individuals suggested a positive relationship between increased serum concentration of uric acid and the presence of MetS.[36] In a longitudinal study, a high level of uric acid has been shown to be related to a higher risk of MetS in men after 10 years of follow-up.[44]

A significant inverse association was detected between physical activity and the presence of MetS. This is consistent with the findings of two previous studies that reported regular physical exercise reduces overall body adipose tissue in general, and abdominal adiposity in particular, in obese and overweight subjects.[45,46] Therefore, as a result of reduced adipose tissue, other components of MetS would be affected.[45] Guinhouya et al. reviewed cross-sectional, prospective, cohort, and intervention studies examining the effect of physical activity on MetS, its components, and IR.[46] They found that higher PAL was consistently associated with an improved metabolic profile and a reduced risk for MetS and IR, which was in agreement with the current study findings.[46] A significant association has been reported for the highest duration of time watching television or playing screen-related games, and sedentary time with MetS.[47]

The negative association between physical activity and the risk of MetS can be attributed to the response of the adipocytes cell surface receptors to various factors including secretion of catecholamines during exercise which in turn mobilize fat deposits,[2] and attenuate IR as the most important components of MetS. Physical activity has been reported to improve the markers of endothelial dysfunction in adolescents with MetS.[48] Furthermore, exercise can help to maintain a higher lean body mass (LBM) and a higher resting energy expenditure (REE) associated with reduced age-related changes including obesity caused by lowered REE and LBM or inflammation which has occurred mainly as a result of obesity.[2]

Adjusted total macronutrient intake had no significant association with the presence of MetS. Brunner et al.[49] and Dalle Grave et al.[50] have shown that dietary carbohydrate intake is unrelated to the risk of MetS. Likewise, Ludwig et al. reported that the dietary intake of fat, carbohydrate, and protein had conflicting associations with the risk factors of CVD.[51] In addition, a meta-analysis of prospective epidemiological cohort studies evaluating the association of saturated fat with CVD concluded that there is no significant evidence for the association between dietary saturated fat and an increased risk of CVD.[52] However, one study has previously shown that a high total carbohydrate intake may be associated with elevated concentrations of plasma lipids and fasting glucose levels, and may result in the MetS in some individuals.[53] Lopez et al. observed the adverse effects of saturated fatty acid (SFA) on the risk of CVD and glycemic control.[54] The differences between the current findings and the results of previous studies could be attributed to the differences in study design, definitions of MetS, sample size, population group, and the method of dietary assessment.

The lack of consistency of the result of the present study with other studies regarding the association between macronutrient intake and MetS might be the result of the greater impact of dietary intake in earlier phases of life.[52-55] According to the Barker hypothesis, maternal nutrition and fetal and infant growth have an important impact on the risk of CVD in adults.[56] Additionally, modification in the dietary intake of the participants by previous medical recommendation could be considered as another reason for the lack of significant differences of the adjusted macronutrient intake between subjects with and without MetS and lack of relationship between nutritional factors and MetS.

Overall, the cross-sectional design of the present study should be taken into account when interpreting the results. Since 24-hour dietary recall methodology, which is based on self-recall, was used in the present study, its inadequate precision especially among obese subjects or women should be considered as a limitation. Scagliusi et al. suggested that a greater BMI, social desirability score, and body dissatisfaction score and lower income are associated with systematic underreporting.[57] Therefore, underreporting of energy intake might be an obstacle in the investigation of obese subjects.[58]

In the present study, the association between nutrient intake and MetS was investigated among a large sample of adults, which was a representative sample. Therefore, the results of the present study could reflect the general population of Iran. Additionally, we controlled for a number of confounding factors including age, sex, smoking, family history of chronic diseases, as well as total energy intake. However, there are some potential limitations, which need to be considered. The first limitation was the cross-sectional design of the study that does not allow the determination of cause-effect relationships. Second, assessing energy and nutrient

intake in this study was based on a single 24-hour dietary recall. In spite of adjusting several potential confounding factors, recall bias and unresponsive bias potentially affected the result of risk factor analysis. Moreover, it may have been better if 3 days of 24-hour dietary recall were analyzed.

Conclusion

An inverse relationship was found between PAL and the risk of MetS, but adjusted dietary macronutrients intake had no significant association with the presence of MetS. Serum concentrations of hs-CRP and uric acid were significantly higher in subjects with MetS.

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Conflict of Interests

Authors have no conflict of interests.

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