

Interaction between major dietary patterns and cardiorespiratory fitness on metabolic syndrome in Iranian adults: a cross-sectional study

Hossein Shahinfar

Tehran University of Medical Sciences <https://orcid.org/0000-0002-4499-4102>

Yahya Jalilpiran

Tehran University of Medical Sciences

Mahtab Ghanbari

Tehran University of Medical Sciences

Nastaran Payandeh

Tehran University of Medical Sciences

Mahshid Shahavandi

Tehran University of Medical Sciences

Nadia Babaei

Tehran University of Medical Sciences

Kurosh Djafarian

Tehran University of Medical Sciences

Cain C.C. Clark

Coventry University Faculty of Health and Life Sciences

Sakineh Shab-Bidar (✉ s_shabbidar@tums.ac.ir)

Tehran University of Medical Sciences (TUMS)

Research

Keywords: Dietary pattern, Cardiorespiratory fitness, Metabolic syndrome, VO2max

Posted Date: September 1st, 2020

DOI: <https://doi.org/10.21203/rs.3.rs-65490/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License. [Read Full License](#)

Version of Record: A version of this preprint was published at Nutrition Journal on April 13th, 2021. See the published version at <https://doi.org/10.1186/s12937-021-00695-4>.

Abstract

Background

Several researches have been conducted on the associations between diet and cardiorespiratory fitness (CRF) and major cardiovascular risk factors. However, there is no report about the interaction between major dietary patterns and CRF on metabolic syndrome (MetS) and its components. To investigate the combined association of major dietary patterns and CRF on MetS and its components.

Methods

This cross-sectional study was conducted in 270 adults living in Tehran, Iran. Dietary intake was evaluated using a validated food frequency questionnaire (FFQ). CRF was assessed using a graded exercise treadmill test. Socio-economic status, anthropometric measures, biochemical parameters, and blood pressure were evaluated according to standard methods. Major dietary patterns were identified by factor analysis.

Results

Three major identified dietary patterns were (healthy, mixed, and western dietary patterns). Adherence to mixed pattern showed a significant decrease for blood glucose ($P < 0.01$). There was a significant increase in triglycerides level among tertiles of western pattern ($P = 0.03$). However, there was no significant difference for MetS and its components between tertiles of CRF. Also we found that there was no interaction of any of dietary patterns (healthy ($P = 0.69$), mixed ($P = 0.80$), and western pattern ($P = 0.60$)) and CRF on odds of MetS.

Conclusions

we showed that the combination of dietary patterns and CRF is not associated with odds of MetS among Iranian adults. More studies are needed to clarify these associations.

Introduction

Metabolic syndrome (Mets) is a cluster of conditions that occur together, including excess fat accumulation around the waist, impaired metabolism of glucose due to insulin resistance, dyslipidemia such as increased blood triglycerides (TG), decreased high-density lipoprotein cholesterol (HDL-C) [1, 2]. The Mets also increases the risk for development of cardiovascular disease (CVD), type 2 diabetes, and hypertension [3, 4]. According to world health organization (WHO) reports CVDs are the number 1 cause of death globally, taking an estimated 17.9 million lives each year [5]. Cardiorespiratory fitness (CRF) is also identified as a key predictor of premature death and cardiovascular disease [6–8]. CRF is defined as the maximum cardiovascular capacity and respiratory systems to supply oxygen to the skeletal muscles during exercise [4]. As a result, it enables the person to maintain physical activity for longer. This ability relates to the three respiratory, cardiovascular, and muscular systems that work together to increase respiratory capacity but the cardiovascular system is more important than other systems [9–11]. Lifestyle changes are an important step in preventing the complications of MetS and the progression of CVD. There are several studies on the association between individual's food intake such as fruit, vegetable and legume intake or dietary fiber with cardiovascular risk factors among Iranian adults other population [12, 13]. Some researchers have also studied the association of processed foods or animal proteins with the risk of cardiovascular disease [14, 15]. Then, the current approach of nutritional epidemiology is to research the effects of dietary patterns instead of food groups on different disease [16, 17]. The results of recent studies on Iranian population showed that the Western dietary pattern causes higher levels of Lp-PLA2 in individuals. Which is directly related to vascular inflammation and cardiovascular disease [17]. Also, a prospective study in Mashhad showed that a balanced diet has no significant relationship with the risk of cardiovascular disease, but the Western diet significantly increases this risk [18]. Despite related studies, limited data are available on the study of CVD-related dietary patterns in the Middle East [19]. However, the data are contradictory in this regard. Numerous studies have been performed, showed that following the West or unhealthy dietary pattern was no significant relationship with mortality from CVD [20, 21]. So, previous studies have suggested the importance of poor cardiorespiratory fitness in adulthood as a risk factor for developing cardiovascular disease in middle age [22]. Because of cardiorespiratory fitness is a powerful predictor of CVD mortality [23], we have examined the combined association of dietary patterns and CRF on MetS among Iranian population.

Methods

Study design

270 adults contain 118 men and 152 women were participated in this cross-sectional study. Participants were recruited exploiting advertisement. Subjects were chosen by convenience sampling. The research criteria included age range of 18–45, participant who apparently healthy, having desire to take part in study, subjects who reside in Tehran. We excluded those who have extreme values of dietary intake (less than 800 kcal/d or more than 4200 kcal/d, respectively), suffering from kidney, liver and lung diseases and other conditions affecting the body composition status or infectious and

active inflammatory diseases, pregnancy, lactation, routine supplement or drug use, such as weight loss, hormonal, sedative drugs, thermogenic supplements like caffeine and green tea, conjugated linoleic acid (CLA) etc. This study was conducted according to the guidelines laid down in the Declaration of Helsinki. All necessary explanations about the project were given to the participants. All procedures were in accord with the ethical standards of the Tehran University of Medical Sciences (Ethic Number: IR.TUMS.VCR.REC.1396.4085), who approved the protocol and informed consent form. All participants signed a written informed consent prior to the start of the study.

Assessment of other variables

Participants completed a questionnaire designed to assess the participants' demographic including age, gender (male/female), marital status (single/married), smoking (non-smoker/former smoker/current smoker), diabetes (yes/no), cardiovascular disease (yes/no) and menopause status (yes/no). Physical activity was assessed using the international physical activity questionnaire (IPAQ)[24]. Subjects were grouped into three categories including very low (< 600 MET-minute/week), low (600–3000 MET-minute/week), moderate and high (> 3000 MET-minute/week) calculated based on Metabolic Equivalents (METs)[25].

Dietary intakes

The dietary intake of participants was assessed by a valid and reliable semi-quantitative Food Frequency Questionnaire (FFQ), which contained 168 food items [26]. FFQ was administered by trained dietitians via face-to-face interviews, asking participants to report their frequency of consumption of each food item, during the past year on a daily, weekly or monthly basis. To convert the portion sizes of the consumed foods to grams, household measures were used. Mean energy and nutrient intakes from the FFQs were calculated using a modified version of NUTRITIONIST IV software for Iranian foods (version 7.0; N-Squared Computing, Salem, OR, USA).

Anthropometric measures

Body weight was determined using a standard body weight scale (seca 707; Seca GmbH & Co. KG., Hamburg, Germany). Height was measured with a tape measure mounted on the wall. Patient's height was measured without shoes by a Stadiometer (Seca, Germany). We measured waist circumference (WC) based on the middle of bottom ribs and pelvic bones after a normal exhale using an inelastic tape. To calculate waist-hip ratio (WHR), WC in centimeter divided by hip circumference in centimeter. Body mass index (BMI) calculated as weight in kilogram divided by height in meters squared. Body composition was measured using body composition analyzer (InBody 720, Biospace, Tokyo, Japan). For this analysis, all patients were asked to follow these conditions before measurement: no food ingestion for at least 4 hours, minimal intake of 2 L of water the day before, no physical activity for at least 8 h, no coffee or alcoholic beverage consumption during at least 12 hours, and no diuretic use for at least 24 hours. Patients were required to empty their bladder immediately before the body composition test [27]. To assess blood pressure, first we asked participants to sit for 10 min. Blood pressure was then measured using a standard mercury sphygmomanometer, twice with a 5 min interval, while participants were sitting. The mean of the two measurements was recorded as the participant's blood pressure.

Laboratory investigations

All participants donated 10 ml of blood between the hours of 7–10 am, in a fasted status. Following this, blood samples were collected in acid-washed test tubes without anticoagulant. Then, after being stored at room temperature for 30 minutes and clot formation, blood samples were centrifuged at 1500 g for 20 minutes. Serums were stored in – 80 ° C until future testing. Fasting blood sugar (FBS) was assayed by the enzymatic (glucose oxidase) colorimetric method using a commercial kit (Pars Azmun, Tehran, Iran). Serum total cholesterol (TC) and high-density lipoprotein cholesterol (HDL-C) were measured using a cholesterol oxidase phenol aminoantipyrine method, and triglyceride (TG) was measured using a glycerol-3 phosphate oxidase phenol aminoantipyrine enzymatic method. Serum low-density lipoprotein cholesterol (LDL-C) was calculated using the Fried Ewald formula.

Definition of terms

MetS was defined according to the National Cholesterol Education Program (NCEP) Adult Treatment Panel-III (ATP III) classification as three or more of WC > 102 cm in males and WC > 88 cm in females, fasting plasma glucose \geq 110 mg/dl in both gender, or a known diagnosis diabetes, fasting serum triglyceride \geq 150 mg/dl in both gender, fasting high-density lipoprotein (HDL) cholesterol < 40 mg/dl in males and HDL < 50 mg/dl in females, or blood pressure \geq 130/85 mmHg in both gender[28].

Cardiorespiratory fitness testing

To assess CRF, the maximum rate of oxygen consumed (VO₂ max) by the treadmill and the respiratory gas analyzer (Cortex Metabolizer 3B) was measured. Accordingly, the participants warmed up for 5 minutes on the treadmill at a speed of 5 km / h, and then the Bruce test was used to determine the VO₂max [29]. After completing the Bruce test, the participants walked at a speed of 4 km / h in order to cool down for 3 minutes and perform 10 to 5 minutes of stretching. The conditions for the end of the test were: the patient's heart rate reaches more than 90% of the maximum heart rate, the ratio of respiratory rate of up to 1.1 and having the plateau rate of oxygen intake, despite the increase in exercise intensity.

Dietary patterns

Foods and beverages from the FFQ were categorized into 17 food groups based on the similarity of nutrients. Factor loadings for each of the 25 food groups was estimated using principle component analysis (PCA) method. Orthogonal transformation was used to keep identified factors uncorrelated and to improve the interpretation. Eigenvalues, the scree plot test, and interpretability were evaluated to retain factors (> 1.5) for further analysis. An absolute factor loading \geq 0.3 was used to define a subset of at least 6 food groups in each factor. The identified factors were labeled based on our

interpretation of the data and based on previous studies that found similar dietary patterns in adults [30–32]. Factor scores for each pattern was obtained by summing intakes of food groups weighted by their factor loadings[33]. Each participant received a factor score for each identified pattern. Participants were categorized based on tertiles of dietary pattern scores.

Statistical analysis

All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS version 25; SPSS Inc.). We considered $p < 0.05$ as significance level. Participants were divided based on the tertiles of major dietary patterns and CRF. To compare general characteristics among tertiles, we used one-way ANOVA and chi-square tests for quantitative and qualitative variables, respectively. Multivariate adjusted means test was performed to evaluate the association between major dietary patterns, CRF and MetS components and each its components after adjusting for potential confounders. Two-way ANOVA was used to investigate the combined association of major dietary patterns and CRF on MetS components. Multivariate adjusted odds ratios test was done for indicating the interaction between CRF and dietary pattern with MetS.

Results

General characteristics of study participants by tertiles of CRF are shown in Table 1. A total of 270 participants (117 men and 153 women) were included in this study. Mean age of participants was 36.71 ± 13.18 . The mean BMI was 25.61 ± 4.67 for them. There were no significant differences in mean weight and having history of CVD and smoking across tertiles CRF. In the third tertile of CRF the mean height (174.01 ± 7.93) and FFM (56.38 ± 11.49) were higher ($p < 0.001$). The number of males ($p < 0.001$) and single ($p < 0.001$) participants were higher in the third compared to the first tertile. With increasing the tertiles of CRF the number of women with menopause ($p < 0.001$) and diabetes ($p = 0.01$) history and the mean of age ($p < 0.001$), FM ($p < 0.001$), WC ($p < 0.001$), BMI ($p < 0.001$), and WHR ($p < 0.001$) significantly reduced. Moreover, participants in the highest tertile of CRF were more active ($p\text{-value} < 0.001$) and more smoker ($p\text{-value} = 0.33$).

Table 1
General characteristics of study participants by tertiles of CRF

	All = 270	Tertiles of CRF			P _{value}
	Mean ± SD or%	T1	T2	T3	
n	270	90	88	92	
Height(cm)	168 ± 9.96	163 ± 9.57	166 ± 49	174 ± 7.93	< 0.001
Age(year)	36.7 ± 13.18	42.8 ± 13.78	35.2 ± 12.36	32.1 ± 11.02	< 0.001
FFM(kg)	50.1 ± 12.65	46.6 ± 11.40	47.2 ± 12.68	56.3 ± 11.49	< 0.001
FM(kg)	22.4 ± 9.39	27.9 ± 10.23	23.2 ± 7.27	16.3 ± 6.34	< 0.001
Weight (kg)	72.7 ± 16.06	74.5 ± 17.82	71.0 ± 16.34	72.6 ± 13.82	0.34
WC (cm)	89.6 ± 12.57	93.4 ± 13.48	89.6 ± 12.74	85.9 ± 10.30	< 0.001
WHR	0.90 ± 0.06	0.92 ± 0.05	0.91 ± 0.06	0.87 ± 0.06	< 0.001
BMI (kg/m ²)	25.6 ± 4.67	27.6 ± 5.36	25.3 ± 4.12	23.8 ± 3.62	< 0.001
Sex, male, n(%)	117(43.5%)	16(5.9%)	32(11.9%)	69(25.7)	< 0.001
Marital status ,n(%)					< 0.001
Single	115(43.3)	23(8.6)	36(13.5)	57(21.3)	
Married	152(56.7)	66(24.6)	51(19.0)	35(13.0)	
Smoking, n(%)					0.33
Non-smoker	233(86.9)	83(31.0)	75(28.0)	75(28.0)	
Former and current smoker	35(13.1)	6(2.20)	12(4.50)	17(6.30)	
Physical activity ,n(%)					< 0.001
Low	103(38.4)	43(16.0)	39(14.6)	21(7.80)	
Medium	110(41.0)	37(13.8)	34(12.7)	39(14.6)	
high	55(20.5)	9(3.40)	14(5.20)	32(11.9)	
Diabetes, yes, n(%)	9(3.40)	7(2.60)	2(0.70)	0(0.0)	0.01
CVD, yes, n(%)	5(1.90)	3(1.10)	1(0.40)	1(0.40)	0.43
Menopause status, yes,	36(13.5)	34(12.7)	2(0.70)	0(0.0)	< 0.001
n (%)					
P value less than 0.05 was considered significant.					
Values are based on average ± standard deviation or reported number (percentage).					
One-way anova for quantitative data and Chi-2 test for qualitative data have been used.					
BMI:Body Mass Index; WC: Waist Circumference; FFM: fat free mass; FM: fat mass; WHR: waist to hip ratio; CRF: cardiorespiratory fitness					

General characteristics of participants across the tertiles of three major dietary patterns are indicated in Table 2. Participants in the highest tertile of Healthy dietary pattern (HDP) had highest BMI (p-value = 0.01), WC (p-value = 0.01), FFM (p-value < 0.01), height (p-value = 0.04), weight (p-value < 0.01), and age (p-value < 0.001). The number of males (p = 0.03) and women with menopause status (p-value = 0.03) increased significantly by increasing the tertiles of this pattern. Adherence to mixed dietary pattern showed a significant increase for FM(p-value = 0.02), WC(p-value = 0.04), WHR(p-value < 0.01), and menopause status(p-value = 0.02). Participants in the highest tertile of Western dietary pattern (WDP) had highest FFM(p-value = 0.03), BMI (p-value < 0.001), and less menopause status(p-value < 0.001) (Table 2).

Table 2
General characteristics of participants across the tertiles of three major dietary patterns

Characteristics	Healthy pattern				Mixed pattern				Western pattern			
	T1	T2	T3	P	T1	T2	T3	p	T1	T2	T3	p
n	89	90	89		89	90	89		89	90	89	
Height(cm)	166 ± 10.0	168 ± 9.04	169 ± 10.6	0.04	169 ± 11.0	167 ± 9.43	167 ± 9.35	0.13	165 ± 10.3	168 ± 9.70	171 ± 9.04	< 0.001
Age(year)	33.4 ± 12.1	35.4 ± 12.7	40.9 ± 13.1	< 0.001	32.7 ± 11.6	36.6 ± 12.5	40.3 ± 14.0	< 0.001	41.7 ± 14.4	34.0 ± 11.6	34.1 ± 11.7	< 0.001
FFM(kg)	47.6 ± 12.4	50.1 ± 11.6	52.7 ± 13.4	< 0.01	51.3 ± 15.0	49.3 ± 11.4	49.8 ± 11.2	0.44	48.5 ± 12.3	49.3 ± 12.2	52.6 ± 13.1	0.03
FM(kg)	21.6 ± 8.12	22.3 ± 9.57	23.3 ± 10.4	0.22	20.8 ± 8.17	22.4 ± 10.2	24.1 ± 9.56	0.02	22.9 ± 8.97	22.4 ± 10.5	21.9 ± 8.70	0.50
Weight (kg)	69.2 ± 15.8	72.5 ± 16.4	76.4 ± 15.3	< 0.01	72.6 ± 16.5	71.6 ± 16.0	73.9 ± 15.8	0.57	71.4 ± 14.0	71.7 ± 17.8	75.1 ± 16.1	0.12
WC (cm)	87.3 ± 12.2	89.4 ± 12.9	92.0 ± 12.3	0.01	88.0 ± 12.1	88.9 ± 12.5	91.8 ± 12.9	0.04	88.9 ± 10.9	88.7 ± 14.2	91.1 ± 12.3	0.24
WHR	0.89 ± 0.06	0.90 ± 0.06	0.91 ± 0.06	0.08	0.89 ± 0.06	0.90 ± 0.06	0.91 ± 0.07	< 0.01	0.89 ± 0.05	0.90 ± 0.07	0.91 ± 0.06	0.05
BMI (kg/m ²)	24.8 ± 4.38	25.4 ± 4.68	26.5 ± 4.85	0.01	25.0 ± 4.12	25.5 ± 4.98	26.2 ± 4.87	0.08	26.0 ± 25.2	25.2 ± 5.35	25.5 ± 4.29	0.38
Sex, male, n(%)	30 (11.2)	40 (14.9)	47 (17.5)	0.03	42 (15.7)	33 (12.3)	42 (15.7)	0.26	42 (10.4)	33 (13.1)	42 (20.1)	< 0.001
Marital status ,n(%)				0.40				0.27				0.14
Single	51 (19.0)	39 (14.6)	36 (13.4)		46 (17.2)	38 (14.2)	32 (11.9)		32 (11.9)	42 (15.7)	42 (15.7)	
Married	38 (14.2)	51 (19.0)	53 (19.8)		43 (16.0)	52 (19.4)	57 (21.3)		57 (21.3)	48 (17.9)	48 (17.9)	
Smoking, n(%)				0.31				0.13				0.24
Non-smoker	81 (30.2)	78 (29.1)	73 (27.2)		77 (28.7)	81 (30.2)	74 (27.6)		77 (28.7)	84 (31.3)	71 (26.5)	
Former and current smoker	8 (3.2)	12 (4.8)	16 (6.4)		12 (4.5)	9 (3.4)	15 (5.6)		12 (4.5)	6 (2.3)	18 (6.7)	
Physical activity ,n(%)				0.10				0.19				0.92
Low	40 (14.9)	35 (13.1)	27 (10.1)		33 (12.3)	40 (14.9)	29 (10.8)		31 (11.6)	35 (13.1)	36 (13.4)	
Medium	36 (13.4)	39 (14.6)	36 (13.4)		33 (12.3)	38 (14.2)	40 (14.9)		40 (14.9)	37 (13.8)	34 (12.7)	
high	13 (4.9)	16 (6.0)	26 (9.7)		23 (8.6)	12 (4.5)	20 (7.5)		18 (6.7)	18 (6.7)	19 (7.1)	
Diabetes, yes, n(%)	3 (1.1)	2 (0.7)	4 (1.5)	0.70	1 (0.4)	3 (1.1)	5 (1.9)	0.25	5 (1.9)	2 (0.7)	2 (0.7)	0.35
CVD, yes, n(%)	2 (0.7)	2 (0.7)	2 (0.7)	0.90	0 (0.0)	3 (1.1)	3 (1.1)	0.22	2 (0.7)	1 (0.4)	3 (1.1)	0.60
Menopause status, yes, n (%)	11 (4.1)	10 (3.7)	15 (5.6)	0.03	6 (2.2)	12 (4.5)	18 (6.7)	0.02	21 (7.9)	10 (3.7)	5 (1.9)	< 0.001
P value less than 0.05 was considered significant.												
Values are based on average ± standard deviation or reported number (percentage).												
One-way ANOVA for quantitative data and Chi-2 test for qualitative data have been used.												
BMI: body mass index; CVD: cardiovascular disease; WC: waist circumference; WHR: waist to hip ratio; FFM: fat free mass; FM: fat mass;												

Table 3 shows food groups and their loading factors for three major identified dietary patterns. Positive loading demonstrated strong associations between the food groups and dietary patterns, while negative loading demonstrated negative associations. Healthy dietary pattern was characterized by high positive loading for the consumption of legumes, vegetables, poultry, fruits and fruits juices, nuts, fish, egg, low fat dairy product, olive and

olive oil. The mixed dietary pattern showed positive loading for the consumption of non-refined cereals, vegetables, vegetable oils, mayonnaise, high fat dairy product, and pickles. The western dietary pattern was represented mainly by positive loading for refined cereals, red or processed meat, soft drinks, sweets and desserts, tea and coffee, salty snacks, and french fries. (Table 3).

Table 3
Food groups and their loading factors for three identified dietary patterns.

Group details		Dietary Patterns		
		Healthy pattern	Mixed pattern	Western pattern
Refined cereals	Lavash bread, baguette bread, rice, pasta, others			0.456
Non refined cereals	Dark breads (e.g., <i>barbari</i> , <i>sangak</i> , <i>taftun</i>), bran breads, others		0.468	
Legumes	Lentils, split pea, beans, chick pea, fava bean, soy, others	0.623		
Red or processed meat	Beef and veal, lamb, minced meat, sausage, deli meat, hamburger			0.401
Vegetables	Cauliflower, carrot, tomato and its products, spinach, lettuce, cucumber, eggplant, onion, greens, green bean, green pea, squash, mushroom, pepper, corn, garlic, turnip, others	0.538	0.467	
Vegetable oils	Vegetable oils (except for olive oils)		0.428	
Poultry	Chicken	0.727		
Organ meats	Heart, kidney, liver, tongue, brain, offal, rennet			
Soft drinks	Soft drinks			0.699
Sweets and desserts	Cookies, cakes, biscuits, muffins, pies, chocolates, honey, jam, sugar cubes, sugar, candies, sweet tahini, others			0.469
Salt	Salt			
Mayonnaise	Mayonnaise		0.767	
Tea and coffee	Tea and coffee			0.313
Salty snacks	Corn puffs, crackers, potato chips, others			0.547
High fat dairy product	High-fat milk, high-fat yogurt, cream cheese, cream, dairy fat, ice cream, others		0.543	
French fries	French fries			0.621
Potatoes	Potatoes			
Fruits and fruits juices	Melon, watermelon, honeydew melon, plums, prunes, apples, cherries, sour cherries, peaches, nectarine, pear, fig, date, grapes, kiwi, pomegranate, strawberry, banana, persimmon, berry, pineapple, oranges, dried fruits, all juices, others	0.582		
Nuts	Almonds, peanut, walnut, pistachio, hazelnut, seeds, others	0.485		
Fish	All fish types	0.581		
Pickles	Pickles, sauerkraut		0.718	
Egg	Eggs	0.648		
Low fat dairy product	Low-fat milk, skim milk, low-fat yogurt, cheese, <i>Kashk</i> , yogurt drink, others	0.451		
Hydrogenated fats	Hydrogenated vegetable oils, solid fats (animal origin), animal butter, margarine			
Olive and olive oil	Olive and olive oil	0.453		
Factor loadings < 0.30 for all three patterns were excluded.				

Multivariate adjusted means for FBS, TG, WC, HDL, SBP and DBP across tertiles of major dietary patterns are indicated in Table 4. Adherence to HDP showed a significant increase for WC ($p = 0.04$) and SBP ($p\text{-value} < 0.01$). Adjustment for confounding factors including age, sex, education, physical activity, smoking, income, total energy and, BMI attenuated the significance of the results. Adherence to MDP showed a significant increase for WC ($p = 0.04$) and DBP ($p\text{-value} = 0.04$). Adjustment for confounding factors attenuated these results. However, after adjustment for confounding factors, participants in highest tertile of this pattern had lowest FBS (< 0.001). The results showed that, participants in the highest tertile of WDP had highest TG ($p = 0.03$) and WC ($p = 0.05$) after adjustment for potential confounders. A significant decrease was showed for WC across the tertiles of CRF ($p\text{-value} < 0.001$). Results remained significant after adjustment for confounding factors ($p\text{-value} < 0.01$). Also Participants in the highest tertile of CRF had highest SBP ($p\text{-value} = 0.04$). This finding remained significant after adjustment for confounding factors, too ($p\text{-value} < 0.001$).

Table 4
Multivariate adjusted means for FBS, TG, WC, HDL, SBP, and DBP across tertiles of major dietary patterns and CRF

Tertiles of major dietary patterns						
Healthy Dietary Pattern						
	T1	T2	T3	P*	P _{Trend}	P§
n	90	88	92			
FBS (mg/dl)	98.5 ± 12.0	96.7 ± 10.5	99.9 ± 28.6	0.53	0.63	0.72
TG (mg/dl)	117 ± 65.5	132 ± 80.5	109 ± 59.8	0.08	0.46	0.10
WC (cm)	87.3 ± 12.2	89.4 ± 12.9	92.0 ± 12.3	0.04	0.01	0.86
HDL (mg/dl)	49.1 ± 10.5	50.8 ± 11.7	189 ± 40.2	0.47	0.98	0.50
SBP (mmHg)	106 ± 26.9	110 ± 10.8	116 ± 14.5	0.01	< 0.001	0.15
DBP (mmHg)	69.9 ± 11.7	69.7 ± 10.4	72.2 ± 9.75	0.22	0.15	0.72
Mixed Dietary Pattern						
	T1	T2	T3	P	P _{Trend}	P _{ANCOVA}
FBS (mg/dl)	101 ± 28.6	97.98 ± 10.29	96.2 ± 11.5	0.23	0.09	0.01
TG (mg/dl)	116 ± 69.7	124.90 ± 72.44	117 ± 67.1	0.70	0.92	0.81
WC (cm)	88.0 ± 12.1	88.94 ± 12.59	91.8 ± 12.9	0.11	0.04	0.24
HDL (mg/dl)	49.5 ± 9.81	50.54 ± 11.97	49.1 ± 10.2	0.67	0.82	0.55
SBP (mmHg)	109 ± 18.6	110.75 ± 20.74	114 ± 17.8	0.22	0.09	0.94
DBP (mmHg)	69.7 ± 8.31	69.23 ± 12.38	72.9 ± 10.7	0.04	0.04	0.63
Western Dietary Pattern						
	T1	T2	T3	P	P _{Trend}	P _{ANCOVA}
FBS (mg/dl)	97.9 ± 8.81	97.2 ± 12.6	100 ± 28.8	0.56	0.46	0.80
TG (mg/dl)	108 ± 54.90	129 ± 75.1	122 ± 75.9	0.10	0.17	0.03
WC (cm)	88.9 ± 10.96	88.7 ± 14.2	91.1 ± 12.3	0.35	0.24	0.053
HDL (mg/dl)	49.5 ± 9.84	50.3 ± 11.5	49.3 ± 10.7	0.80	0.52	0.86
SBP (mmHg)	114 ± 18.04	110 ± 19.7	109 ± 19.5	0.24	0.13	0.25
DBP (mmHg)	70.0 ± 13.11	70.2 ± 8.81	71.5 ± 9.80	0.59	0.35	0.17
Cardiorespiratory fitness (ml/kg/min)						
	T1	T2	T3	P	P _{Trend}	P _{ANCOVA}
FBS (mg/dl)	97.5 ± 9.76	100 ± 27.5	97.5 ± 14.9	0.58	0.98	0.59
TG (mg/dl)	123 ± 60.7	125 ± 80.2	110 ± 66.4	0.27	0.20	0.61
WC (cm)	93.4 ± 13.4	89.6 ± 12.7	85.9 ± 10.3	< 0.001	< 0.001	0.01
HDL (mg/dl)	49.8 ± 9.16	49.5 ± 12.0	49.9 ± 10.7	0.95	0.93	0.96
SBP (mmHg)	108 ± 27.9	111 ± 13.76	114 ± 11.1	0.12	0.04	< 0.001
DBP (mmHg)	71.1 ± 12.2	70.2 ± 11.7	70.3 ± 7.69	0.81	0.59	0.62

Values are presented as mean ± SD.

* obtained using one-way ANOVA test.

§ obtained using analysis of covariance (ANCOVA) test (adjusted for age, sex, marital status, physical activity, smoking, total energy, diabetes, cardiovascular disease history, menopause status, and body mass index).

Abbreviations: FBS: Fasting Blood Sugar; CRF: cardiorespiratory fitness; TG: Triglyceride; HDL: High Density Lipoprotein; SBP: Systolic Blood Pressure; DBP: Diastolic Blood; WC = Waist Circumference

Table 5 shows multivariate adjusted odds ratios and 95% confidence intervals for metabolic syndrome and its components across tertiles of CRF. Multiple logistic regression models showed that CRF had significantly negative association (highest vs lowest tertile) with abdominal obesity in the crude model (OR = 0.09, 95% CI (0.04–0.20)) and model 1 (OR = 0.12, 95% CI (0.04–0.32)). Our results also showed that being in the third compared to the first category of CRF was inversely associated with metabolic syndrome (OR = 0.43, 95% CI (0.20–0.94)) and low HDL-C levels (OR = 0.44, 95% CI (0.24–0.82)) in crude model.

Table 5
Multivariate adjusted odds ratios and 95% confidence intervals for metabolic syndrome and its components across tertiles of CRF

	Tertiles of CRF			P for trend
	T1	T2	T3	
Metabolic Syndrome				
Crude	1.00	0.64(0.31,1.32)	0.43(0.20,0.94)	0.10
Model 1*	1.00	0.81(0.35,1.86)	0.66(0.23,1.84)	0.73
Model 2 §	1.00	1.16(0.50,2.71)	1.22(0.41,3.65)	0.91
Hypertriglyceridemia				
Crude	1.00	0.85(0.43,1.70)	0.80(0.40,1.60)	0.82
Model 1	1.00	0.85(0.40,1.83)	0.83(0.34,2.05)	0.90
Model 2	1.00	0.92(0.42,2.01)	0.97(0.37,2.54)	0.97
Hypertension				
Crude	1.00	1.02(0.28,3.66)	0.37(0.07,2.00)	0.45
Model 1	1.00	1.01(0.21,4.91)	0.35(0.04,2.91)	0.53
Model 2	1.00	1.33(0.22,8.04)	0.66(0.06,7.23)	0.75
Hyperglycemia				
Crude	1.00	0.76(0.41,1.42)	0.87(0.48,1.60)	0.70
Model 1	1.00	1.02(0.50,2.06)	1.43(0.62,3.29)	0.62
Model 2	1.00	1.09(0.53,2.27)	1.68(0.68,4.11)	0.46
Low HDL-C				
Crude	1.00	1.11(0.61,2.01)	0.44(0.24,0.82)	0.01
Model 1	1.00	1.54(0.77,3.06)	1.04(0.45,2.39)	0.36
Model 2	1.00	1.59(0.79,3.20)	1.11(0.46,2.68)	0.35
Abdominal obesity				
Crude	1.00	0.38(0.20,0.70)	0.09(0.04,0.20)	< 0.001
Model 1	1.00	0.40(0.20,0.79)	0.12(0.04,0.32)	< 0.001
Model 2	1.00	0.67(0.23,1.93)	0.30(0.06,1.56)	0.36
Data are presented as OR (95%CI)				
*Adjusted for age and sex, smoking, physical activity, socioeconomic status				
§ Adjusted for age, sex, smoking, physical activity, socioeconomic status, body mass index and energy.				
HDL-C: high density lipoprotein-cholesterol; CRF: cardiorespiratory fitness				

Multivariate adjusted odds ratios and 95% confidence intervals for metabolic syndrome and its components across tertiles of identified dietary patterns are summarized in Table 6. Accordingly, the results showed that greater adherence to the HDP (third vs first tertile) was significantly associated with lower odds of low HDL-C levels (OR = 0.54, 95% CI (0.29– 0.99)) in crude model. However, after adjustment for potential confounders was attenuated. Also, our results indicated positive association (highest vs lowest tertile) between MDP and metabolic syndrome in crude model (OR = 2.57, 95% CI (1.10– 6.02)) and model 1 (OR = 2.66, 95% CI (1.06–6.62)). This association changed to non-significant results after additional

adjustment for body mass index and energy intakes. Moreover, we did not find any significant association between the WDP and odds of metabolic syndrome and its components in crude and adjusted models.

Table 6

Multivariate adjusted odds ratios and 95% confidence intervals for metabolic syndrome and its components across tertiles of dietary patterns

	Healthy pattern				Mixed pattern				Western pattern			
	T1	T2	T3	P	T1	T2	T3	p	T1	T2	T3	p
Metabolic Syndrome												
Crude	1.00	1.30 (0.62,2.71)	0.92 (0.42,2.00)	0.62	1.00	2.87 (1.24,6.66)	2.57 (1.10,6.02)	0.03	1.00	1.30 (0.62,2.71)	0.92 (0.42,2.00)	0.62
Model 1*	1.00	1.31 (0.61,2.81)	0.95 (0.41,2.17)	0.67	1.00	2.88 (1.17,7.04)	2.66 (1.06,6.62)	0.05	1.00	1.42 (0.63,3.20)	1.25 (0.53,2.94)	0.68
Model 2 §	1.00	1.24 (0.56,2.75)	0.68 (0.27,1.71)	0.40	1.00	3.06 (1.21,7.77)	2.68 (0.92,7.78)	0.05	1.00	1.38 (0.60,3.18)	1.02 (0.39,2.63)	0.67
Hypertriglyceridemia												
Crude	1.00	2.22 (1.09,4.54)	1.43 (0.67,3.01)	0.08	1.00	1.54 (0.76,3.11)	1.39 (0.68,2.84)	0.46	1.00	1.72 (0.85,3.45)	1.22 (0.59,2.53)	0.29
Model 1	1.00	2.02 (0.89,4.19)	1.23 (0.56,2.69)	0.12	1.00	1.79 (0.84,3.78)	1.59 (0.73,3.44)	0.29	1.00	2.06 (0.95,4.46)	1.49 (0.66,3.34)	0.19
Model 2	1.00	1.96 (0.94,4.08)	1.04 (0.45,2.39)	0.10	1.00	1.78 (0.84,3.79)	1.49 (0.64,3.49)	0.31	1.00	2.03 (0.94,4.41)	1.38 (0.58,3.27)	0.18
Hypertension												
Crude	1.00	0.31 (0.06,1.60)	0.65 (0.17,2.39)	0.37	1.00	1.33 (0.29,6.13)	1.70 (0.39,7.36)	0.77	1.00	0.73 (0.15,3.37)	1.26 (0.32,4.87)	0.76
Model 1	1.00	0.25 (0.04,1.38)	0.33 (0.08,1.41)	0.17	1.00	1.23 (0.25,6.09)	1.05 (0.21,5.08)	0.96	1.00	1.32 (0.23,7.40)	2.32 (0.49,10.9)	0.53
Model 2	1.00	0.18 (0.03,1.17)	0.28 (0.05,1.41)	0.13	1.00	1.62 (0.29,8.82)	2.73 (0.37,19.9)	0.61	1.00	1.24 (0.20,7.54)	3.88 (0.60,24.9)	0.27
Hyperglycemia												
Crude	1.00	0.49 (0.26,0.92)	0.59 (0.32,1.08)	0.07	1.00	1.66 (0.90,3.07)	1.01 (0.53,1.88)	0.16	1.00	0.89 (0.48,1.63)	0.78 (0.42,1.44)	0.73
Model 1	1.00	0.47 (0.25,0.90)	0.58 (0.29,1.13)	0.06	1.00	1.42 (0.74,2.70)	0.85 (0.43,1.67)	0.27	1.00	0.94 (0.48,1.82)	0.91 (0.46,1.82)	0.96
Model 2	1.00	0.46 (0.24,0.89)	0.54 (0.26,1.12)	0.05	1.00	1.41 (0.74,2.70)	0.81 (0.38,1.74)	0.27	1.00	0.94 (0.48,1.83)	0.95 (0.45,1.98)	0.98
Low HDL-C												
Crude	1.00	0.78 (0.43,1.41)	0.54 (0.29,0.99)	0.13	1.00	1.48 (0.81,2.69)	1.01 (0.54,1.83)	0.32	1.00	1.23 (0.68,2.24)	1.04 (0.57,1.91)	0.76
Model 1	1.00	0.83 (0.44,1.56)	0.65 (0.33,1.29)	0.47	1.00	1.37 (0.71,2.64)	1.11 (0.56,2.18)	0.61	1.00	1.41 (0.72,2.79)	1.70 (0.84,3.44)	0.32
Model 2	1.00	0.80 (0.42,1.52)	0.57 (0.27,1.19)	0.33	1.00	1.36 (0.70,2.62)	1.03 (0.48,2.19)	0.59	1.00	1.42 (0.72,2.81)	1.77 (0.82,3.83)	0.32
Abdominal obesity												
Crude	1.00	1.28 (0.67,2.42)	1.58 (0.84,2.97)	0.36	1.00	1.42 (0.75,2.69)	1.67 (0.88,3.15)	0.27	1.00	0.84 (0.45,1.57)	0.81 (0.43,1.52)	0.79
Model 1	1.00	1.35 (0.68,2.69)	1.79 (0.87,3.66)	0.28	1.00	1.16 (0.58,2.33)	1.41 (0.70,2.86)	0.61	1.00	1.09 (0.53,2.22)	1.33 (0.64,2.75)	0.72
Model 2	1.00	1.23 (0.41,3.74)	2.19 (0.58,8.21)	0.48	1.00	1.62 (0.53,4.95)	1.29 (0.35,4.64)	0.69	1.00	2.49 (0.71,8.67)	1.78 (0.49,6.39)	0.35
Data are OR (95%CI)												
*Adjusted for age and sex, smoking, physical activity, socioeconomic status												
§ Adjusted for age, sex, smoking, physical activity, socioeconomic status, body mass index, and energy intake.												
HDL-C: high density lipoprotein-cholesterol												

Multivariate adjusted odds ratios and 95% confidence intervals for interaction between CRF and identified dietary patterns with metabolic syndrome are indicated in Table 7. The results showed that there was not significant interaction between CRF and HDP ($p = 0.69$), MDP ($p = 0.80$), and WDP ($p = 0.60$) with odds of metabolic syndrome after adjustment for potential confounders.

Table 7
Multivariate adjusted odds ratios and 95% confidence intervals for interaction between CRF and dietary pattern with metabolic syndrome

CRF	Tertiles of healthy dietary pattern			P _{value} for interaction
	T1	T2	T3	
	90	88	92	
T1	1.00	1.00	1.00	0.69
T2	1.00	0.85(0.14,5.06)	1.85(0.29,11.70)	
T3	1.00	0.57(0.09,3.53)	0.43(0.05,3.22)	
CRF	Tertiles of mixed dietary pattern			P _{value}
	T1	T2	T3	
T1	1.00	1.00	1.00	0.80
T2	1.00	1.54(0.19,12.07)	1.32(0.16,10.48)	
T3	1.00	1.36(0.17,10.91)	0.47(0.05,4.28)	
CRF	Tertiles of western dietary pattern			P _{value}
	T1	T2	T3	
T1	1.00	1.00	1.00	0.60
T2	1.00	0.58(0.09,3.54)	1.60(0.25,10.21)	
T3	1.00	1.36(0.21,8.72)	0.76(0.09,6.23)	
Data are presented as OR (95%CI)				
CRF:cardiorespiratory fitness				

Multivariate adjusted means for interaction between CRF and dietary patterns with metabolic syndrome components are shown in Table 8. Adherence to WDP showed a significant decrease for HDL across tertiles of CRF (p -value = 0.03). Also, adherence to HDP indicated a significant increase for SBP across tertiles of CRF ($p = 0.03$). Moreover, adherence to MDP showed a significant decrease for WC (p -value = 0.05) and DBP (p -value < 0.01) across tertiles of CRF (Table 8).

Table 8
Multivariate adjusted means for interaction between CRF and dietary patterns with metabolic syndrome components

FBS (mg/dl)	CRF (ml/kg/min)			
Tertiles of healthy pattern	T1	T2	T3	P _{value}
	90	88	92	
T1	98.0 ± 11.8	100 ± 14.0	96.4 ± 9.63	0.45
T2	97.6 ± 7.25	94.6 ± 8.09	98.1 ± 14.5	
T3	96.8 ± 9.58	106 ± 48.1	97.6 ± 18.1	
FBS (mg/dl)	CRF (ml/kg/min)			
Tertiles of mixed pattern	T1	T2	T3	P _{value}
T1	97.5 ± 13.4	104.5 ± 42.0	99.3 ± 16.8	0.86
T2	98.3 ± 9.85	97.8 ± 10.3	97.7 ± 11.0	
T3	96.6 ± 7.41	96.6 ± 10.7	95.1 ± 15.9	
FBS (mg/dl)	CRF (ml/kg/min)			
Tertiles of western pattern	T1	T2	T3	P _{value}
T1	98.7 ± 7.94	97.9 ± 8.05	96.7 ± 10.6	0.22
T2	97.1 ± 9.72	95.5 ± 13.0	99.1 ± 14.7	
T3	96.0 ± 12.8	106 ± 44.0	96.8 ± 17.8	
TG (mg/dl)	CRF (ml/kg/min)			
Tertiles of healthy pattern	T1	T2	T3	P _{value}
T1	110 ± 47.2	127 ± 80.6	114 ± 70.8	0.66
T2	138 ± 65.7	133 ± 90.5	125 ± 83.3	
T3	126 ± 70.7	115 ± 66.2	94.1 ± 41.4	
TG (mg/dl)	CRF (ml/kg/min)			
Tertiles of mixed pattern	T1	T2	T3	P _{value}
T1	129 ± 87.9	109 ± 64.0	117 ± 64.0	0.27
T2	118 ± 46.0	146 ± 86.4	110 ± 79.1	
T3	125 ± 56.4	126 ± 90.8	101 ± 56.4	
TG (mg/dl)	CRF (ml/kg/min)			
Tertiles of western pattern	T1	T2	T3	P _{value}
T1	109 ± 45.6	121 ± 63.5	93.9 ± 57.3	0.84
T2	135 ± 63.4	136.6 ± 91.4	115 ± 65.3	
T3	133 ± 77.5	119 ± 81.0	117 ± 73.1	
HDL (mg/dl)	CRF (ml/kg/min)			
Tertiles of healthy pattern	T1	T2	T3	P _{value}
T1	49.5 ± 8.56	47.1 ± 11.0	50.9 ± 12.6	0.39
T2	51.5 ± 10.0	49.7 ± 13.4	51.5 ± 11.4	
T3	48.3 ± 9.38	51.7 ± 11.5	48.0 ± 8.66	
HDL (mg/dl)	CRF (ml/kg/min)			
Tertiles of mixed pattern	T1	T2	T3	P _{value}
T1	50.3 ± 9.26	49.1 ± 10.0	49.4 ± 10.1	0.65

FBS (mg/dl)	CRF (ml/kg/min)			
T2	51.6 ± 9.55	48.7 ± 13.9	51.1 ± 12.5	
T3	47.7 ± 8.80	50.8 ± 12.7	49.5 ± 9.99	
HDL (mg/dl)	CRF (ml/kg/min)			
Tertiles of western pattern	T1	T2	T3	P _{value}
T1	51.9 ± 9.73	47.6 ± 11.2	48.0 ± 8.19	0.03
T2	47.8 ± 7.80	49.0 ± 13.2	54.2 ± 11.8	
T3	48.6 ± 9.76	51.3 ± 11.6	48.0 ± 10.8	
WC (cm)	CRF (ml/kg/min)			
Tertiles of healthy pattern	T1	T2	T3	P _{value}
T1	90.3 ± 13.8	86.0 ± 11.0	84.1 ± 10.4	0.78
T2	94.5 ± 13.6	88.9 ± 12.9	85.5 ± 11.2	
T3	96.3 ± 12.9	94.5 ± 13.4	87.3 ± 9.47	
WC (cm)	CRF (ml/kg/min)			
Tertiles of mixed pattern	T1	T2	T3	P _{value}
T1	92.0 ± 15.2	88.7 ± 12.3	85.1 ± 9.35	0.05
T2	89.2 ± 13.8	88.7 ± 13.6	88.7 ± 10.0	
T3	98.0 ± 11.2	91.9 ± 12.6	84.1 ± 11.3	
WC (cm)	CRF (ml/kg/min)			
Tertiles of western pattern	T1	T2	T3	P _{value}
T1	91.8 ± 11.7	86.4 ± 10.7	87.0 ± 9.26	0.32
T2	91.6 ± 15.6	90.7 ± 15.0	83.5 ± 10.4	
T3	98.3 ± 13.2	90.9 ± 11.6	86.9 ± 10.8	
SBP (mmHg)	CRF (ml/kg/min)			
Tertiles of healthy pattern	T1	T2	T3	P _{value}
T1	99.0 ± 39.0	108 ± 11.9	115 ± 13.4	0.03
T2	113.0 ± 13.4	108 ± 9.23	111 ± 9.71	
T3	115.7 ± 16.9	119 ± 17.1	115 ± 10.4	
SBP (mmHg)	CRF (ml/kg/min)			
Tertiles of mixed pattern	T1	T2	T3	P _{value}
T1	99.3 ± 32.6	111 ± 9.88	113 ± 11.6	0.12
T2	108 ± 28.7	107 ± 15.6	117 ± 11.3	
T3	113 ± 24.0	117 ± 14.8	112 ± 10.1	
SBP (mmHg)	CRF (ml/kg/min)			
Tertiles of western pattern	T1	T2	T3	P _{value}
T1	114 ± 24.2	110 ± 11.7	117 ± 10.9	0.26
T2	106 ± 29.7	112 ± 14.9	111 ± 10.3	
T3	100 ± 31.2	111 ± 14.2	114 ± 11.3	
DBP (mmHg)	CRF (ml/kg/min)			
Tertiles of healthy pattern	T1	T2	T3	P _{value}

FBS (mg/dl)	CRF (ml/kg/min)			
T1	69.8 ± 14.1	69.8 ± 10.9	69.7 ± 9.06	0.78
T2	70.2 ± 10.1	68.5 ± 13.3	70.5 ± 6.84	
T3	74.0 ± 11.6	72.7 ± 10.4	70.5 ± 7.59	
DBP (mmHg)	CRF (ml/kg/min)			
Tertiles of mixed pattern	T1	T2	T3	P _{value}
T1	69.0 ± 9.13	69.4 ± 9.60	70.5 ± 6.46	0.01
T2	68.0 ± 13.6	66.8 ± 14.5	73.1 ± 6.43	
T3	75.4 ± 11.5	75.5 ± 9.09	67.4 ± 9.21	
DBP (mmHg)	CRF (ml/kg/min)			
Tertiles of western pattern	T1	T2	T3	P _{value}
T1	70.9 ± 15.1	66.0 ± 14.7	72.5 ± 6.56	0.13
T2	71.5 ± 9.16	71.1 ± 9.75	67.9 ± 6.98	
T3	71.2 ± 10.8	72.6 ± 10.5	70.6 ± 8.62	
P value less than 0.05 was considered significant.				
Data are presented as mean ± standard deviation				
P _{value} derived from two-way analysis of variances				
CRF: cardiorespiratory fitness; FBS, Fasting Blood Sugar; TG, Triglyceride; HDL, High Density Lipoprotein; SBP: Systolic Blood Pressure; DBP: Diastolic Blood; WC = Waist Circumference				
mg/dl: milligram per deciliter; cm ² : centimeter ² mmHg: millimetre of mercury; ml/kg/min: milliliter per kilogram per minute				

Discussion

In this cross-sectional study we identified three major dietary patterns including healthy, mixed, and western patterns. None of these patterns were associated with odds of metabolic syndrome or its components. In addition, we found that there was no interaction of any of dietary patterns (healthy, mixed and western pattern) and CRF on odds of MetS. However, Adherence to WDP showed a significant decrease for HDL across tertiles of CRF. Adherence to HDP indicated a significant increase for SBP across tertiles of CRF and adherence to MDP showed a significant decrease for WC and DBP across tertiles of CRF.

To date, several epidemiological studies have been performed on the impact of dietary pattern on MetS. The results of previous studies showed that HDP (rich in vegetables, fruits, and whole grains) was inversely related to metabolic syndrome and its component [34–36]. Evidence from a cross-sectional study of 4984 women aged 30–79 years indicated that HDP (high in green-yellow vegetables, healthy-protein foods, seaweeds, and bonefish) is associated with a decreased risk for MetS [37]. In another study by Esmailzadeh et al., in 486 Tehrani female teachers the HDP (high in fruits, poultry and vegetables) was associated with less prevalence of the MetS [38]. Williams et al., in a cross sectional study of 802 UK-population aged 40–65 years reported that a HDP (high in fruits, raw and salad vegetables, fish and low in fried foods) is associated with decreased central obesity and fasting glucose concentration [39]. In addition, a previous prospective study of 15972 white and black men and women 45 to 64 years of age revealed adherence to prudent dietary pattern that described by intakes of cruciferous and carotenoid vegetables, fruit, fish, and poultry, was not correlated with an increase in the prevalence of MetS [40]. One of the reasons for the decrease in the prevalence of metabolic syndrome with more adherence to a HDP can be ascribed to its healthy ingredients, such as vegetables, fruits, whole grains, fiber, high quality protein, monounsaturated fatty acids, magnesium, phytochemicals and phytoestrogen [41–45]. In the current study, it seems that a HDP (high in legumes, vegetables, poultry, fruits and fruits juices, nuts, fish, egg, low fat dairy product, olive and olive oil) is neither associated with a reduction in the prevalence of metabolic syndrome nor its components. In the present study, other factors such as weight control in people who have a HDP may affect the desired effects. For example, in our study, participants in the highest tertile of HDP had highest BMI, weight and WC. In addition, participants in the highest tertile of HDP are older than those in the lowest tertile. The prevalence of metabolic syndrome increases with age, so this may be influential. Generally, although the HDP identified in our study is rich in foods that have the potential to reduce the risk of metabolic syndrome but it cannot be propounded exclusively a health-promoting diet. This pattern is more a reflection of the effort to choose healthy foods.

WDP that is determined by excessive consumption of fat, sweets, and refined grains showed inconsistent results with metabolic syndrome and its components [46–48]. In research by Cho et al., the WDP (high in fast foods, animal fat-rich foods, fried foods, grilled meat and seafoods, and sweet foods) indicated no association with MetS [49]. However, WDP in 486 Tehrani female teachers in the study of Esmailzadeh et al ., was related with

greater risks of MetS [31]. Adherence to a WDP that determined by high consumption of refined grains, processed meat, fried foods, and red meat, was correlated with a 18% higher risk of occurrence MetS [50]. One of the reasons we can rely on it to justify the positive relationship between the WDP and the metabolic syndrome is the high amounts of saturated fats [51], red meat [52, 53] and refined grains [54] in this pattern. In our study, Dietary consumption of a WDP (including refined cereals, red or processed meat, soft drinks, sweets and desserts, tea and coffee, salty snacks, and french fries) was not correlated with an increased risk of metabolic syndrome and its components. This maybe because people who follow the WDP also consume great amounts of food from healthy dietary pattern. Finally, it can be concluded that beneficial foods and nutrients in a HDP can reduce the negative effects of foods in the WDP.

In relation to the MDP, which has a combination of healthy (non-refined cereals, vegetables, vegetable oils) and unhealthy (mayonnaise, high fat dairy product) foods in present study is almost identical to the pattern described in Esmailzadeh and colleagues study as the traditional food pattern [31]. It has been suggested that healthy combinations of this pattern can have a protective effect on the syndrome, while unhealthy foods can have a negative effect on the prevalence of the syndrome. As well as, the synergistic effect of other food compounds on HDP may be a factor in reducing the risk of metabolic syndrome [31]. The result of our analysis showed, Dietary consumption of a MDP (high consumption of non-refined cereals, vegetable oils, mayonnaise, high fat dairy product, and pickles) was not associated with odds of metabolic syndrome or its components. Anthropometric characteristics and menopause status may influence the relationship between the MDP and the metabolic syndrome. In this pattern subjects in the highest tertile of MDP had highest FM, WC, WHR, and menopause status. Several studies have reported an opposite association between metabolic syndrome and the amount of body fat [55–57] and menopausal status [58, 59].

Our results indicated that having a higher score on the CRF compared with a lower score was associated with decreased odds of MetS, however, were not statistically significant. Also, higher CRF levels were associated with lower measures of abdominal obesity. Several studies have investigated the associations of CRF and MetS. Shaibi et al., indicated that CRF was not associated with any individual risk factor of the metabolic syndrome [60]. In addition, in a population-based cohort on 605 middle-aged men and women found no independent association between CRF and the development of MetS [61]. In contrast, Ekelund et al., reported significant reverse association between CRF and with indicators of insulin resistance, hyperglycemia, hyperlipidemia, and clustered metabolic risk in children [62]. Others studies have shown that CRF is linked to insulin sensitivity as a risk factor for MetS [63, 64]. Hassinen et al., in 1226 men and women aged 57–78 years, reported that higher levels of CRF protect against MetS [65]. Increased muscle insulin sensitivity, lipoprotein lipase activity in active musculoskeletal, transport of lipids and lipoproteins from the peripheral blood circulation and tissues to the liver and reduction abdominal obesity [66] are mechanisms by which MetS may improve with fitness [67, 68]. The differences between the results of the association between CRF and MetS in different studies may be related to different sample sizes, group characteristics, and differences in study methods. In addition, genetic, age, body composition and most importantly, physical activity determines CRF [69]. In this study, subjects who have top levels of CRF have more physical activity, have greater FFM, lower BMI, WC and WHR than those who have lower level of CRF. Indeed, CRF moderates the link between obesity and metabolic status [70]; considering that the risk of metabolic disorders, including MetS, increases with obesity.

All of these articles separately evaluated the association between dietary pattern and cardiorespiratory fitness on metabolic syndrome. Food selection are closely linked to lifestyle [71]. Recently, it has been reported that following more healthy dietary patterns is related with a better selection of foods, increase physical activity and thus reducing the incidence of obesity [72, 73]. In this regard, some studies have evaluated that the association between CRF and dietary patterns [74, 75]. For example; in a cross-sectional study in 279 students aged 14–18 years, the result indicated CRF was associated with a healthier dietary pattern include higher intake of fruit, vegetables, whole grain and lower intake of sweets, chocolates, fries and sugar-sweetened drinks [74]. In another study, in 2632 participants aged 38–50 years, meat dietary pattern was negatively correlated with CRF [76]. Accordingly, we considered the combined effect of these two related factors on metabolic syndrome. However, we did not find significant interaction between dietary patterns and CRF on MetS.

In this study, follow to HDP showed a significant increase for systolic blood pressure across tertiles of CRF. Interestingly, some people develop high blood pressure notwithstanding an active physical lifestyle or a healthy diet, while some sedentary people with unhealthy dietary patterns may have optimal blood pressure. A number of studies have shown that higher cardiorespiratory fitness and physical activity are associated with lower blood pressure [77, 78]. However some studies did not find a connection [79] and in some cases it is even accompanied by a scant increase [80]. The two chief factors that can lead to these differences are the level of primary phenotype and familial aggregation [69]. In general, information on the contribution of genetic diversity to the antihypertensive effect associated with cardiorespiratory fitness is low. In this study, more adherence to the mixed dietary pattern was associated with an increase in waist circumference and blood pressure. But in people who have a mixed dietary pattern, waist circumference and diastolic blood pressure decreased with increasing physical activity and cardiorespiratory fitness. Physical activity decreased the effect of genetic factors like FTO genotype to cause high BMI and WC [81]. Eventually high cardiorespiratory fitness overcame the harmful effects of a mixed dietary pattern on WC and DBP. In relation to the interaction of western dietary pattern and cardiovascular fitness with the components of MetS it is notable that, despite have reported that HDL-C is highly responsive in exercise and have ascertained that exercise and high level of cardiorespiratory fitness increases HDL-cholesterol [82] adherence to WDP showed a significant decrease for HDL across tertiles of CRF. many studies the western dietary pattern was inversely associated with HDL-C [83, 84]. One mechanism clarifying the association between the WDP and lower HDL-C could be higher intake of refined carbohydrates among participants who follow the WDP [84]. Finally, western dietary pattern dominates the detrimental effects of a cardiorespiratory fitness on HDL-C.

Our study had some strengths. According to our knowledge, that interaction between major dietary patterns and cardiorespiratory fitness on metabolic syndrome is reported for the first time in our study. However, it should be mentioned that present study had some limitations owing to the cross-sectional design prevent any causal conclusion between diet, CRF and metabolic syndrome being distinguished. Also Residual disruptors that are unmeasurable and possible in observational studies could affect the study.

Conclusion

We showed that the combination of dietary patterns and CRF is not associated with risk of MetS among Iranian adults. More studies should be conducted to clarify the association.

Abbreviations

HDP: Healthy Dietary Pattern; MDP: Mixed Dietary Pattern; WDP: Western Dietary Pattern; CRF: Cardiorespiratory Fitness; FBS: Fasting Blood Sugar; TC: Total Cholesterol; TG: Triglyceride; HDL-C: High Density Lipoprotein; LDL-C: Low Density Lipoprotein; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; FFM: Fat Free Mass; FM: Fat Mass; WC: Waist Circumference; CVD: cardiovascular disease; WHR: waist to hip ratio; BMI: Body Mass Index

Declarations

Acknowledgments

Authors thank all those who participated in this study.

Authors contributions

SSb contributed to conception/design of the research; MGH, MSH and NP contributed to acquisition, analysis, or interpretation of the data; HSH and CC drafted the manuscript; KD and SSb critically revised the manuscript, and SS-b agrees to be fully accountable for ensuring the integrity and accuracy of the work. All authors read and approved the final manuscript.

Funding

None.

Availability of data and materials

The datasets generated or analyzed during the current study are not publicly available but are available from the corresponding author on reasonable request.

Ethics approval and consent to participate

This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving research study participants were approved by the ethics committee of Tehran University of Medical Sciences (TUMS.VCR.REC.1396.4085). Written informed consent was obtained from all subjects/patients.

Consent for publication

Participants were provided a study overview and verbal consent was attained.

Competing interests

The authors declare that they have no competing interests.

References

1. Grundy SM. Metabolic syndrome update. *Trends Cardiovasc Med.* 2016;26(4):364 – 73. doi:10.1016/j.tcm.2015.10.004.
2. Mirmiran P, Ziadlou M, Karimi S, Hosseini-Esfahani F. The association of dietary patterns and adherence to WHO healthy diet with metabolic syndrome in children and adolescents: Tehran lipid and glucose study. *BMC Public Health.* 2019;19(1):1457. doi:10.1186/s12889-019-7779-9.
3. Monnerie S, Comte B, Ziegler D, Morais JA, Pujos-Guillot E, Gaudreau P. Metabolomic and Lipidomic Signatures of Metabolic Syndrome and its Physiological Components in Adults: A Systematic Review. *Sci Rep.* 2020;10(1):669. doi:10.1038/s41598-019-56909-7.PMC6971076.
4. Myers J, Kokkinos P, Nyelin E. Physical Activity, Cardiorespiratory Fitness, and the Metabolic Syndrome. *Nutrients.* 2019;11(7).doi:10.3390/nu11071652.PMC6683051.
5. WHO. cardiovascular disease. 2019.

6. Kaminsky LA, Arena R, Ellingsen Ø, Harber MP, Myers J, Ozemek C, et al. Cardiorespiratory fitness and cardiovascular disease - The past, present, and future. *Prog Cardiovasc Dis.* 2019;62(2):86–93. doi:10.1016/j.pcad.2019.01.002.
7. Vancampfort D, Mugisha J, Rosenbaum S, Firth J, De Hert M, Probst M, et al. Cardiorespiratory fitness levels and moderators in people with HIV: A systematic review and meta-analysis. *Prev Med.* 2016;93:106 – 14. doi:10.1016/j.ypmed.2016.10.001.
8. Vancampfort D, Ward PB, Stubbs B. Physical fitness levels and moderators in people with epilepsy: A systematic review and meta-analysis. *Epilepsy Behav.* 2019;99:106448. doi:10.1016/j.yebeh.2019.106448.
9. Blair SN, Kampert JB, Kohl HW 3rd, Barlow CE, Macera CA, Paffenbarger RS Jr, et al. Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. *Jama.* 1996;276(3):205–10.
10. Sandbakk SB, Nauman J, Lavie CJ, Wisloff U, Stensvold D. Combined Association of Cardiorespiratory Fitness and Body Fatness With Cardiometabolic Risk Factors in Older Norwegian Adults: The Generation 100 Study. *Mayo Clin Proc Innov Qual Outcomes.* 2017;1(1):67–77. doi:10.1016/j.mayocpiqo.2017.05.001.PMC6135019.
11. Wei M, Kampert JB, Barlow CE, Nichaman MZ, Gibbons LW, Paffenbarger RS Jr, et al. Relationship between low cardiorespiratory fitness and mortality in normal-weight, overweight, and obese men. *Jama.* 1999;282(16):1547–53. doi:10.1001/jama.282.16.1547.
12. Miller V, Mente A, Dehghan M, Rangarajan S, Zhang X, Swaminathan S, et al. Fruit, vegetable, and legume intake, and cardiovascular disease and deaths in 18 countries (PURE): a prospective cohort study. *Lancet.* 2017;390(10107):2037–49. doi:10.1016/s0140-6736(17)32253-5.
13. Soliman GA. Dietary Fiber, Atherosclerosis, and Cardiovascular Disease. *Nutrients.* 2019;11(5).doi:10.3390/nu11051155.PMC6566984.
14. 10.1136/bmj.l1451.PMC6538975
 Srour B, Fezeu LK, Kesse-Guyot E, Allès B, Méjean C, Andrianasolo RM, et al. Ultra-processed food intake and risk of cardiovascular disease: prospective cohort study (NutriNet-Santé). *Bmj.* 2019;365:l1451.doi: 10.1136/bmj.l1451.PMC6538975 and declare no support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; no other relationships or activities that could appear to have influenced the submitted work.
15. Richter CK, Skulas-Ray AC, Champagne CM, Kris-Etherton PM. Plant protein and animal proteins: do they differentially affect cardiovascular disease risk? *Adv Nutr.* 2015;6(6):712 – 28. doi:10.3945/an.115.009654.PMC4642426.
16. Archundia Herrera MC, Subhan FB, Chan CB. Dietary Patterns and Cardiovascular Disease Risk in People with Type 2 Diabetes. *Curr Obes Rep.* 2017;6(4):405 – 13. doi:10.1007/s13679-017-0284-5.
17. Seyedi SHS, Mottaghi A, Mirmiran P, Hedayati M, Azizi F. The relationship between dietary patterns and lipoprotein-associated phospholipase A2 levels in adults with cardiovascular risk factors: Tehran Lipid and Glucose Study. *J Res Med Sci.* 2020;25:3. doi:10.4103/jrms.JRMS_256_19.PMC7003539.
18. Asadi Z, Shafiee M, Sadabadi F, Heidari-Bakavoli A, Moohebbati M, Khorrami MS, et al. Association of dietary patterns and risk of cardiovascular disease events in the MASHAD cohort study. *J Hum Nutr Diet.* 2019;32(6):789–801. doi:10.1111/jhn.12669.
19. Menotti A, Alberti-Fidanza A, Fidanza F, Lanti M, Fruttini D. Factor analysis in the identification of dietary patterns and their predictive role in morbid and fatal events. *Public Health Nutr.* 2012;15(7):1232–9. doi:10.1017/s1368980011003235.
20. Nettleton JA, Polak JF, Tracy R, Burke GL, Jacobs DR. Jr. Dietary patterns and incident cardiovascular disease in the Multi-Ethnic Study of Atherosclerosis. *Am J Clin Nutr.* 2009;90(3):647 – 54. doi:10.3945/ajcn.2009.27597.PMC2728647.
21. Osler M, Heitmann BL, Gerdes LU, Jørgensen LM, Schroll M. Dietary patterns and mortality in Danish men and women: a prospective observational study. *Br J Nutr.* 2001;85(2):219 – 25. doi:10.1079/bjn2000240.
22. Carnethon MR, Gidding SS, Nehgme R, Sidney S, Jacobs DR Jr, Liu K. Cardiorespiratory fitness in young adulthood and the development of cardiovascular disease risk factors. *Jama.* 2003;290(23):3092 – 100. doi:10.1001/jama.290.23.3092.
23. Swainson MG, Ingle L, Carroll S. Cardiorespiratory fitness as a predictor of short-term and lifetime estimated cardiovascular disease risk. *Scand J Med Sci Sports.* 2019;29(9):1402–13. doi:10.1111/sms.13468.
24. Moghaddam MB, Aghdam FB, Jafarabadi MA, Allahverdipour H, Nikookheslat SD, Safarpour S. The Iranian Version of International Physical Activity Questionnaire (IPAQ) in Iran: content and construct validity, factor structure, internal consistency and stability. *World applied sciences journal.* 2012;18(8):1073–80.
25. Wareham NJ, Jakes RW, Rennie KL, Schuit J, Mitchell J, Hennings S, et al. Validity and repeatability of a simple index derived from the short physical activity questionnaire used in the European Prospective Investigation into Cancer and Nutrition (EPIC) study. *Public Health Nutr.* 2003;6(4):407–13. doi:10.1079/phn2002439.
26. Mirmiran P, Esfahani FH, Mehrabi Y, Hedayati M, Azizi F. Reliability and relative validity of an FFQ for nutrients in the Tehran lipid and glucose study. *Public Health Nutr.* 2010;13(5):654 – 62. doi:10.1017/s1368980009991698.
27. Korth O, Bosy-Westphal A, Zschoche P, Gluer CC, Heller M, Muller MJ. Influence of methods used in body composition analysis on the prediction of resting energy expenditure. *Eur J Clin Nutr.* 2007;61(5):582–9. doi:10.1038/sj.ejcn.1602556.
28. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP). Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). *Jama.* 2001;285(19):2486–97. doi:10.1001/jama.285.19.2486.

29. Bruce RA, Kusumi F, Hosmer D. Maximal oxygen intake and nomographic assessment of functional aerobic impairment in cardiovascular disease. *Am Heart J.* 1973;85(4):546 – 62. doi:10.1016/0002-8703(73)90502-4.
30. Hu FB, Rimm E, Smith-Warner SA, Feskanich D, Stampfer MJ, Ascherio A, et al. Reproducibility and validity of dietary patterns assessed with a food-frequency questionnaire. *Am J Clin Nutr.* 1999;69(2):243–9. doi:10.1093/ajcn/69.2.243.
31. Esmailzadeh A, Kimiagar M, Mehrabi Y, Azadbakht L, Hu FB, Willett WC. Dietary patterns, insulin resistance, and prevalence of the metabolic syndrome in women. *Am J Clin Nutr.* 2007;85(3):910–8. doi:10.1093/ajcn/85.3.910.
32. Esmailzadeh A, Azadbakht L. Major dietary patterns in relation to general obesity and central adiposity among Iranian women. *J Nutr.* 2008;138(2):358 – 63. doi:10.1093/jn/138.2.358.
33. Kim J-O, Mueller CW. *Factor analysis: Statistical methods and practical issues*: sage; 1978.
34. Esposito K, Ciotola M, Giugliano D. Mediterranean diet and the metabolic syndrome. *Mol Nutr Food Res.* 2007;51(10):1268–74. doi:10.1002/mnfr.200600297.
35. Sonnenberg L, Pencina M, Kimokoti R, Quatromoni P, Nam BH, D'Agostino R, et al. Dietary patterns and the metabolic syndrome in obese and non-obese Framingham women. *Obes Res.* 2005;13(1):153–62. doi:10.1038/oby.2005.20.
36. Wirfält E, Hedblad B, Gullberg B, Mattisson I, Andrén C, Rosander U, et al. Food patterns and components of the metabolic syndrome in men and women: a cross-sectional study within the Malmö Diet and Cancer cohort. *Am J Epidemiol.* 2001;154(12):1150–9. doi:10.1093/aje/154.12.1150.
37. Cho YA, Kim J, Cho ER, Shin A. Dietary patterns and the prevalence of metabolic syndrome in Korean women. *Nutrition Metabolism Cardiovascular Diseases.* 2011;21(11):893–900.
38. Esmailzadeh A, Kimiagar M, Mehrabi Y, Azadbakht L, Hu FB, Willett WC. Dietary patterns, insulin resistance, and prevalence of the metabolic syndrome in women. *Am J Clin Nutr.* 2007;85(3):910–8.
39. Williams DE, Prevost AT, Whichelow MJ, Cox BD, Day NE, Wareham NJ. A cross-sectional study of dietary patterns with glucose intolerance and other features of the metabolic syndrome. *Br J Nutr.* 2000;83(3):257–66.
40. Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome. *Circulation.* 2008;117(6):754–61.
41. Esmailzadeh A, Kimiagar M, Mehrabi Y, Azadbakht L, Hu FB, Willett WC. Fruit and vegetable intakes, C-reactive protein, and the metabolic syndrome. *Am J Clin Nutr.* 2006;84(6):1489–97.
42. Esmailzadeh A, Mirmiran P, Azizi F. Whole-grain consumption and the metabolic syndrome: a favorable association in Tehranian adults. *Eur J Clin Nutr.* 2005;59(3):353–62.
43. He K, Liu K, Daviglius ML, Morris SJ, Loria CM, Van Horn L, et al. Magnesium intake and incidence of metabolic syndrome among young adults. *Circulation.* 2006;113(13):1675–82.
44. Minich DM, Bland JS. Dietary management of the metabolic syndrome beyond macronutrients. *Nutrition reviews.* 2008;66(8):429–44.
45. Slavin JL, Martini MC, Jacobs DR Jr, Marquart L. Plausible mechanisms for the protectiveness of whole grains. *The American journal of clinical nutrition.* 1999;70(3):459 s-63 s.
46. Sonnenberg L, Pencina M, Kimokoti R, Quatromoni P, Nam BH, D'agostino R, et al. Dietary patterns and the metabolic syndrome in obese and non-obese Framingham women. *Obesity research.* 2005;13(1):153–62.
47. Muzio F, Mondazzi L, Harris WS, Sommariva D, Branchi A. Effects of moderate variations in the macronutrient content of the diet on cardiovascular disease risk factors in obese patients with the metabolic syndrome. *Am J Clin Nutr.* 2007;86(4):946–51.
48. Baxter AJ, Coyne T, McClintock C. Dietary patterns and metabolic syndrome—a review of epidemiologic evidence. *Asia Pacific journal of clinical nutrition.* 2006;15(2).
49. Cho YA, Kim J, Cho ER, Shin A. Dietary patterns and the prevalence of metabolic syndrome in Korean women. *Nutr Metab Cardiovasc Dis.* 2011;21(11):893–900. doi:10.1016/j.numecd.2010.02.018.
50. Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome: the Atherosclerosis Risk in Communities study. *Circulation.* 2008;117(6):754 – 61. doi:10.1161/circulationaha.107.716159.
51. Riccardi G, Giacco R, Rivellese AA. Dietary fat, insulin sensitivity and the metabolic syndrome. *Clin Nutr.* 2004;23(4):447 – 56. doi:10.1016/j.clnu.2004.02.006.
52. Tappel A. Heme of consumed red meat can act as a catalyst of oxidative damage and could initiate colon, breast and prostate cancers, heart disease and other diseases. *Med Hypotheses.* 2007;68(3):562–4. doi:10.1016/j.mehy.2006.08.025.
53. Song Y, Manson JE, Buring JE, Liu S. A prospective study of red meat consumption and type 2 diabetes in middle-aged and elderly women: the women's health study. *Diabetes Care.* 2004;27(9):2108–15. doi:10.2337/diacare.27.9.2108.
54. Song S, Lee JE, Song WO, Paik HY, Song Y. Carbohydrate intake and refined-grain consumption are associated with metabolic syndrome in the Korean adult population. *J Acad Nutr Diet.* 2014;114(1):54–62. doi:10.1016/j.jand.2013.08.025.
55. Ramírez-Vélez R, Correa-Bautista JE. Percentage of Body Fat and Fat Mass Index as a Screening Tool for Metabolic Syndrome Prediction in Colombian University Students. *Nutrients.* 2017;9(9).doi:10.3390/nu9091009.
56. Kim K, Park SM. Association of muscle mass and fat mass with insulin resistance and the prevalence of metabolic syndrome in Korean adults: a cross-sectional study. *Sci Rep.* 2018;8(1):2703. doi:10.1038/s41598-018-21168-5.Pmc5807388.

57. Knowles KM, Paiva LL, Sanchez SE, Revilla L, Lopez T, Yasuda MB, et al. Waist Circumference, Body Mass Index, and Other Measures of Adiposity in Predicting Cardiovascular Disease Risk Factors among Peruvian Adults. *Int J Hypertens*. 2011;2011:931402.doi. 10.4061/2011/931402.Pmc3034939.
58. Koh JH, Lee MY, Nam SM, Sung JK, Jung PM, Noh JK, et al. Relationship between menopausal status and metabolic syndrome components in Korean women. *Korean Diabetes Journal*. 2008;32(3):243–51.
59. Jouyandeh Z, Nayezbadeh F, Qorbani M, Asadi M. Metabolic syndrome and menopause. *J Diabetes Metab Disord*. 2013;12(1):1. doi:10.1186/2251-6581-12-1.Pmc3598172.
60. Shaibi GQ, Cruz ML, Ball GD, Weigensberg MJ, Kobaissi HA, Salem GJ, et al. Cardiovascular fitness and the metabolic syndrome in overweight latino youths. *Med Sci Sports Exerc*. 2005;37(6):922–8.
61. Ekelund U, Brage S, Franks PW, Hennings S, Emms S, Wareham NJ. Physical activity energy expenditure predicts progression toward the metabolic syndrome independently of aerobic fitness in middle-aged healthy Caucasians: the Medical Research Council Ely Study. *Diabetes Care*. 2005;28(5):1195 – 200. doi:10.2337/diacare.28.5.1195.
62. Ekelund U, Anderssen SA, Froberg K, Sardinha LB, Andersen LB, Brage S. Independent associations of physical activity and cardiorespiratory fitness with metabolic risk factors in children: the European youth heart study. *Diabetologia*. 2007;50(9):1832–40. doi:10.1007/s00125-007-0762-5.
63. Imperatore G, Cheng YJ, Williams DE, Fulton J, Gregg EW. Physical activity, cardiovascular fitness, and insulin sensitivity among U.S. adolescents: the National Health and Nutrition Examination Survey, 1999–2002. *Diabetes Care*. 2006;29(7):1567–72. doi:10.2337/dc06-0426.
64. Ball GD, Shaibi GQ, Cruz ML, Watkins MP, Weigensberg MJ, Goran MI. Insulin sensitivity, cardiorespiratory fitness, and physical activity in overweight Hispanic youth. *Obes Res*. 2004;12(1):77–85. doi:10.1038/oby.2004.11.
65. Hassinen M, Lakka TA, Savonen K, Litmanen H, Kiviahio L, Laaksonen DE, et al. Cardiorespiratory fitness as a feature of metabolic syndrome in older men and women: the Dose-Responses to Exercise Training study (DR's EXTRA). *Diabetes Care*. 2008;31(6):1242–7. doi:10.2337/dc07-2298.
66. Wedell-Neergaard AS, Krogh-Madsen R, Petersen GL, Hansen ÅM, Pedersen BK, Lund R, et al. Cardiorespiratory fitness and the metabolic syndrome: Roles of inflammation and abdominal obesity. *PLoS One*. 2018;13(3):e0194991. doi:10.1371/journal.pone.0194991.
67. Wei M, Gibbons LW, Mitchell TL, Kampert JB, Lee CD, Blair SN. The association between cardiorespiratory fitness and impaired fasting glucose and type 2 diabetes mellitus in men. *Ann Intern Med*. 1999;130(2):89–96. doi:10.7326/0003-4819-130-2-199901190-00002.
68. Stefanick M, Wood P, editors. Physical activity, lipid and lipoprotein metabolism, and lipid transport. Physical activity, fitness and health: International proceedings and consensus statement; 1994: Human Kinetics, Champaign, IL.
69. 10.1097/00005768-200106001-00013
Bouchard C, Rankinen T. Individual differences in response to regular physical activity. *Med Sci Sports Exerc*. 2001;33(6 Suppl):S446-51; discussion S52-3.doi: 10.1097/00005768-200106001-00013.
70. Katzmarzyk PT, Church TS, Janssen I, Ross R, Blair SN. Metabolic syndrome, obesity, and mortality: impact of cardiorespiratory fitness. *Diabetes Care*. 2005;28(2):391–7. doi:10.2337/diacare.28.2.391.
71. Patterson RE, Haines PS, Popkin BM. Health lifestyle patterns of US adults. *Preventive medicine*. 1994;23(4):453–60.
72. Granic A, Jagger C, Davies K, Adamson A, Kirkwood T, Hill TR, et al. Effect of Dietary Patterns on Muscle Strength and Physical Performance in the Very Old: Findings from the Newcastle 85 + Study. *PLoS One*. 2016;11(3):e0149699.doi. 10.1371/journal.pone.0149699.
73. Xu B, Houston DK, Locher JL, Ellison KJ, Gropper S, Buys DR, et al. Higher Healthy Eating Index-2005 scores are associated with better physical performance. *J Gerontol A Biol Sci Med Sci*. 2012;67(1):93 – 9. doi:10.1093/gerona/glr159.
74. Howe AS, Skidmore PM, Parnell WR, Wong JE, Lubransky AC, Black KE. Cardiorespiratory fitness is positively associated with a healthy dietary pattern in New Zealand adolescents. *Public Health Nutr*. 2016;19(7):1279–87. doi:10.1017/s1368980015002566.
75. López-Gil JF, Brazo-Sayavera J, García-Hermoso A, Yuste Lucas JL. Adherence to Mediterranean Diet Related with Physical Fitness and Physical Activity in Schoolchildren Aged 6–13. *Nutrients*. 2020;12(2):567.
76. Shikany JM, Jacobs DR Jr, Lewis CE, Steffen LM, Sternfeld B, Carnethon MR, et al. Associations between food groups, dietary patterns, and cardiorespiratory fitness in the Coronary Artery Risk Development in Young Adults study. *Am J Clin Nutr*. 2013;98(6):1402–9. doi:10.3945/ajcn.113.058826.Pmc3831533.
77. Boreham CA, Ferreira I, Twisk JW, Gallagher AM, Savage MJ, Murray LJ. Cardiorespiratory fitness, physical activity, and arterial stiffness: the Northern Ireland Young Hearts Project. *Hypertension*. 2004;44(5):721–6. doi:10.1161/01.HYP.0000144293.40699.9a.
78. Barlow CE, LaMonte MJ, Fitzgerald SJ, Kampert JB, Perrin JL, Blair SN. Cardiorespiratory fitness is an independent predictor of hypertension incidence among initially normotensive healthy women. *Am J Epidemiol*. 2006;163(2):142 – 50. doi:10.1093/aje/kwj019.
79. Lin CY, Chen PC, Kuo HK, Lin LY, Lin JW, Hwang JJ. Effects of obesity, physical activity, and cardiorespiratory fitness on blood pressure, inflammation, and insulin resistance in the National Health and Nutrition Survey 1999–2002. *Nutr Metab Cardiovasc Dis*. 2010;20(10):713-9. doi:10.1016/j.numecd.2009.06.005.
80. Bouchard C, Rankinen T. Individual differences in response to regular physical activity. *Medicine and science in sports and exercise*. 2001;33(6).

81. Mustelin L, Silventoinen K, Pietiläinen K, Rissanen A, Kaprio J. Physical activity reduces the influence of genetic effects on BMI and waist circumference: a study in young adult twins. *Int J Obes (Lond)*. 2009;33(1):29–36. doi:10.1038/ijo.2008.258.
82. Parto P, Lavie CJ, Swift D, Sui X. The role of cardiorespiratory fitness on plasma lipid levels. *Expert Rev Cardiovasc Ther*. 2015;13(11):1177–83. doi:10.1586/14779072.2015.1092384.
83. Drake I, Sonestedt E, Ericson U, Wallström P, Orho-Melander M. A Western dietary pattern is prospectively associated with cardio-metabolic traits and incidence of the metabolic syndrome. *Br J Nutr*. 2018;119(10):1168–76. doi:10.1017/s000711451800079x.
84. Peñalvo JL, Oliva B, Sotos-Prieto M, Uzhova I, Moreno-Franco B, León-Latre M, et al. Greater adherence to a Mediterranean dietary pattern is associated with improved plasma lipid profile: the Aragon Health Workers Study cohort. *Rev Esp Cardiol (Engl Ed)*. 2015;68(4):290-7. doi:10.1016/j.rec.2014.09.019.