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RESEARCH PAPER

Association of dietary patterns and risk of cardiovascular disease events in the MASHAD cohort study

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Abstract

Background: Cardiovascular disease (CVD) is the principal cause of mortality and disability in Iranian adults. We aimed to evaluate the relationship between dietary patterns and CVD incidence in a large sample of adults in northeastern Iran.
Methods: The present study comprised a prospective study of 5706 CVD-free men and women aged 35–65 years who participated in a cohort study. All of the participants were followed up for a 6-year period. Dietary patterns were derived from a 65-item validated food frequency questionnaire and the factor analysis method was used to determine dietary patterns.
Results: We identified two major dietary patterns: (i) a Balanced dietary pattern (a high intake of green leafy vegetables, other vegetables, fruits, dairy products, red meats, poultry, seafoods, legumes and nuts, as well as a low intake of sugar) and (ii) a Western dietary pattern (a high intake of sugar, tea, egg, snacks, fast foods, potato, carbonated beverages, pickled foods, organs meat and butter) by factor analysis. The hazard ratio (HR) and 95% confidence intervals (CIs) of total CVD in the highest versus lowest tertiles of the Balanced pattern were 1.29 (95% CI = 0.67–2.47; $P = 0.44$). The HR and 95% CIs of CVD in the highest versus lowest tertiles of Western pattern were 2.21 (95% CI = 1.08–4.45; $P = 0.03$).
Conclusions: During the 6-year follow-up, we found that adherence to a Balanced dietary pattern was not significantly associated with CVD events. However, adherence to a Western dietary pattern was associated with a significantly increased risk of CVD events and its associated risk.

Introduction

Life expectancy has been increasing in the Iranian population as a result of the introduction of a successful health services system from 1980 onwards [(1)]. However, cardiovascular disease (CVD) remains the principal cause of mortality, leading to approximately 50% of all deaths per annum and approximately 50% of the deaths as a result of chronic disease in Iran [(2)]. Moreover, the need for hospitalisation and outpatient treatment of CVD

patients has an impact on public health expenditure [3]. In recent decades, the Iranian diet has changed considerably as a result of urbanisation, social change and instability of economic status. Because of this transition, a larger number of Iranian adults consume lower amounts of several micronutrients [4]. It is now clear that a low dietary intake of fruits, vegetables and whole grains in Iranian diet are dietary risk factors that may contribute to the development of CVD [1].

Several studies have examined the relationship between dietary patterns and the prevalence of CVD worldwide [5–12]. Dietary patterns are changing rapidly in developing countries, with a trend toward higher intakes of fat and red meat and lower intakes of carbohydrate and fibre [13]. Previous studies have indicated that a Western dietary pattern, characterised by a high dietary intake of meat, refined grains, high-fat snacks and dairy products, as well as reduced fish intake, increases the risk of CVD in Iranian population [14], whereas a healthy dietary pattern that includes a high intake of fruits, fruit juices, vegetables, liquid oils and nuts, as well as a low intake of refined grains, reduces the CVD risk [15]. However, only a few studies have assessed the association between dietary patterns and CVD events in limited areas of Iran (Tehran and Isfahan) [14–17]. Furthermore, the association of dietary patterns with CVD risk factors has not been evaluated previously. Therefore, we aimed to prospectively evaluate the relationship between major dietary patterns identified by factor analysis with CVD events, CVD risk factors and anthropometric variables in a large population-based sample of Iranian adults. We hypothesised that a healthy dietary pattern would decrease the risk of CVD, whereas an unhealthy dietary pattern would increase the risk among the study population.

Materials and methods

Study population

All subjects were derived from Mashhad stroke and heart atherosclerotic disorder (MASHAD) study, a cohort of 9704 free-living individuals aged 35–65 years from northeastern Iran [18]. The MASHAD study project started in 2010 with the aim of evaluating the relationship between CVD events and various risk factors. The sample size was determined based on the prevalence of diabetes as a risk factor of CVD. According to the Ministry of Health, the prevalence of diabetes was estimated to be 3% in the Mashhad population. The entire population of Mashhad was obtained using the 2006 National Iranian Census. Moreover, a 40% drop in the sample size was estimated, and the sample size of the MASHAD study project was estimated at 9704 individuals.

Three regions of Mashhad city (Mashhad Health Center numbers 1, 2 and 3) were chosen and each region was divided into nine locations centred upon Mashhad Healthcare Center divisions. Individuals aged 35–65 years and those without history of CVD (coronary artery disease, stroke and peripheral arterial disease) and other chronic diseases (cancer and chronic kidney disease) were included in the study [18]. We collected all necessary data during three follow-up periods. The first follow-up started in 2011 at which point 255 participants indicated that they had suffered a CVD event. The second follow-up started in 2014 during which 513 participants claimed to have a CVD event. Thus, there were a total of 768 subjects who claimed to have a CVD event, of whom 110 did not attend the third follow-up and were not included in the final analysis. Finally, 658 subjects attended the third follow-up from April 2015 to May 2016. Individuals who were lost to follow-up were excluded from the analyses (413 subjects were lost to the first follow-up, 629 subjects were lost to the second follow-up and 110 subjects were lost to the third follow-up). Participants who reported taking treatments for diabetes mellitus were excluded from the analysis (n = 138). We also excluded individuals with an estimated energy intake of <800 or >4200 kcal (n = 124) [19], as well as those who did not complete more than 10% of the food frequency questionnaire (FFQ) items (n = 2584). Thus, the final sample population included in the final analysis consisted of 5706 subjects (2343 men and 3363 women) (Fig. 1).

Ethical approval

The study protocol, informed consent form and other study-related documents were reviewed and approved by the Human Research Ethics Committee of Mashhad University of Medical Sciences (MUMS). All participants provided their written and informed consent.

Diagnosis of cardiovascular diseases

The presence of CVD (outcome) among subjects was confirmed at the third follow-up by a typical history of myocardial infarction or angina pectoris together with electrocardiographic evidence of a definite Q wave using the Minnesota Code [20,21], physical examination and a detailed medical history that was taken by a cardiologist. Suspicious cases were also examined by echocardiography, stress echocardiography, radioisotope, angiography, computed tomography angiography and an exercise tolerance test at a complementary medical examination. A definitive diagnosis was made based on a consensus decision of a panel of experts. A diagnosis of CVD was made in 235

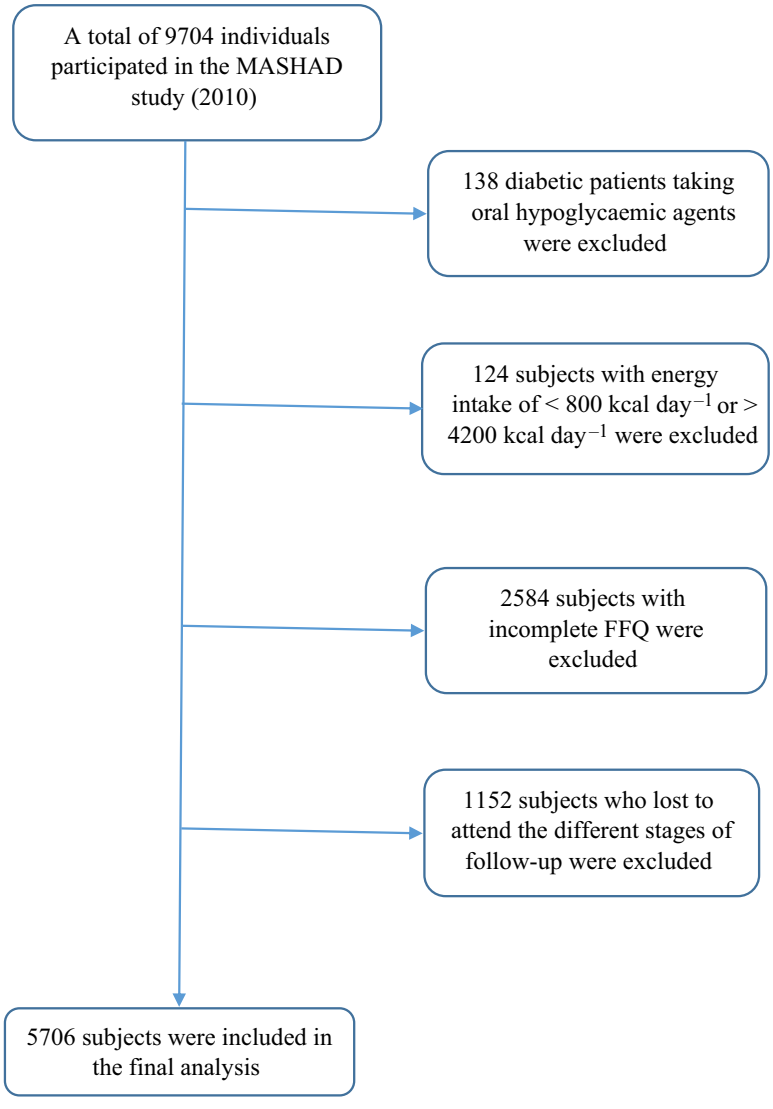

Figure 1 Participant flow diagram. FFQ, food frequency questionniare; MASHHAD, Mashhad stroke and heart atherosclerotic disorder

patients, including 120 subjects with unstable angina, 75 subjects with stable angina and 40 subjects with myocardial infarction. Healthy, nonsymptomatic individuals (n = 5471) were considered as the healthy control group. The Framingham cardiovascular examination questionnaire was completed by cardiologists for all participants [22].

Anthropometric assessments

Height (cm), weight (kg), body mass index (BMI) (kg m^{-2}), waist circumference (WC) (cm), hip circumference (HC) (cm), waist–hip ratio (WHR) and mid-arm circumference (MAC) (cm) were measured in all subjects. Height, WC, HC and MAC were measured to the nearest millimeter with a tape measure. Weight was measured to the nearest 0.1 kg with electronic scales.

Laboratory evaluation

Blood samples of all subjects were collected after a 12-h overnight fast. Fasting blood glucose (FBG), total cholesterol (TC), Low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C) and triglycerides (TG) were measured enzymatically using an automated analyser.

Dietary intake assessment

We used a previously validated semi-quantitative FFQ at baseline [23]. The agreement of the current FFQ and 24-h recall was assessed by the intra-class correlation coefficient, which reports the validity of the FFQ. Values of less than 0.21 represent poor agreement, 0.21–0.40 represents

fair agreement, 0.41–0.60 represents moderate agreement, 0.61–0.80 represents substantial agreement and >0.80 represents almost perfect agreement [24]. The data indicated a perfect validity of honey; a substantial validity of food items including beverages and sugars; a moderate validity of fruits, grains, leafy vegetables and pickles; a fair validity for snacks, dairy products and animal protein; and a poor validity for fast foods, other vegetables, legumes and nuts. After completing the FFQ twice with a definite interval, the correlation between FFQ1 and FFQ2 was assessed by Pearson's correlation coefficient, which indicated the reproducibility of the FFQ [23]. We found a high reproducibility of some food items including grains, dairy products, fast foods, leafy vegetables, beverages, honey and other vegetables; moderate reproducibility of snacks, fruits, sugar, pickles and animal protein; and weak reproducibility for legumes and nuts [23].

The FFQ included data on the consumption of 65 food items. For each food item, the questionnaire consisted of five frequency categories (frequency of use per day, week, month, rarely and never) and serving size. Serving sizes are defined based on household scales (e.g. a glass of milk), natural portions (e.g. one banana) or standard weight and volume measures of the servings, which was completed by skilled nutritionists via face-to-face interview. For our analysis, the reported frequency for each food item was converted into daily intake. Total energy intake was computed by summing the energy intakes from all foods.

Assessment of dietary patterns

The procedure for deriving dietary patterns (exposure) using food consumption data involved first classifying the 65 food items into 22 predefined food groups (g day^{-1}) as predictors, based on the similarity of food items. We performed factor analysis (principal components) to derive dietary patterns based on the 22 foods or food groups [25]. The factors were orthogonally rotated (varimax rotation) to keep them uncorrelated, as well as to improve interpretation. When determining the number of factors to retain, components with an eigenvalue >1, the scree test and the interpretability of the factors were considered [26]. Therefore, a three-component dietary pattern was obtained in the current analysis. The first component demonstrates the lower consumption of one dietary pattern, whereas the last component represents the higher consumption. The first component of each dietary pattern was chosen as the reference group because it was the most interpretable factor among the study population.

After adding the consumption of the food groups weighted by their factor loadings, the factor scores were estimated for each dietary pattern. Finally, each participant received a score for each of the two determined dietary patterns. The percentage variance explained by each factor was not used because this criterion depends greatly on the total number of variables included in the analyses [27]. The first tertile of each dietary pattern was chosen as the reference category.

Assessment of physical activity level

The James and Schofield human energy requirements equations were performed to assess the physical activity level [28]. Questions were divided into time spent on activities during work (including housework), during the non-work time, and in bed (resting in bed and sleep).

Assessment of other variables

Information on participants' demographic characteristics (e.g. age, sex and education), medical history and lifestyle factors including tobacco use, cardiovascular risk-related questionnaire, and anxiety and depression tests were collected by health care professionals and a nurse interview. The Beck's Depression Inventory II and the Beck's Anxiety Inventory were used to assess depression and anxiety symptoms among participants [29,30]. These questionnaires contain 21 items and each answer is scored on a zero (lack of depression/anxiety symptoms) to three (severe depression/anxiety symptoms) scale. Thus, the total score of the questionnaire ranges from 0 to 63. The validity of these questionnaires has been recently assessed in Iranian population [31,32]. Blood pressure (BP) was measured using the left arm, with individuals in a seated position and at rest for 15 min, using a mercury sphygmomanometer. This was repeated twice in exactly the same manner. If discrepant, with the readings differing by more than 15 mmHg in diastolic or more than 25 mmHg in systolic blood pressure, we took a third measurement and averaged the two closest readings [33,34].

Statistical analysis

Participants were followed from the onset of the study (2010) until the occurrence of cardiovascular disease or until 19 April 2016. Descriptive analyses of the main variables of interest [including age, weight, BMI, WC, WHR, MAC, systolic blood pressure (SBP) and diastolic blood pressure (DBP), lipid profile, FBG] across tertiles of each dietary pattern score were performed. Analysis of variance (ANOVA) and Kruskal–Wallis tests were used to evaluate mean differences of normal and non-normal distributed data across tertiles of each dietary pattern, respectively. The chi-squared test was used to determine differences in the distribution of qualitative variables across tertiles of each dietary pattern. Leven's test for equality of variances

was used to compare tertiles of each dietary pattern. Sex-adjusted means and proportions for baseline characteristics across each dietary pattern tertile were calculated using analysis of variance and logistic regression, respectively. The age variable was categorised into two groups (35–48 years and ≥48 years) according to the association between age groups and a disease occurrence by the latent Dirichlet allocation, *K*-means and hierarchical clustering algorithms [35].

The person-years of the follow-up were calculated based on the time interval from the baseline to the date of the CVD event, or the date of follow-up visit, whichever came first. Means and proportions of selected CVD risk factors were calculated based on tertiles of each dietary pattern score. We used the lowest tertile as the reference category for analysis of the hazard ratios (HRs) and their 95% confidence intervals (CIs) for the other tertiles. We first adjusted for age categories (34–48 years and ≥49 years). Second model was constructed to additionally adjust for potential confounders including sex, baseline body mass index (m kg^2), smoking status (never, past, and current), education level (low, moderate and high), marital status (married, single, divorced and widow), total energy intake, diabetes mellitus, hypertension, and dyslipidaemia, which were significantly different across tertiles of identified dietary patterns. Statistical analysis was performed using SPSS, version 16.0 (SPSS, Chicago, IL, USA). $P < 0.05$ was considered statistically significant.

Results

In the present study, we included 5706 participants from the MASHAD cohort study among whom the diagnosis of CVD was made in 235 patients by expert cardiologists. However, 74 CVD patients with an incomplete FFQ were excluded from the final analysis. Thus, 161 CVD patients were included in the study and Healthy, nonsymptomatic individuals ($n = 5545$) were considered as the healthy control group.

During the 6-year period between the baseline and the final follow-up assessment, 2.82% ($n = 161$) of the participants experienced CVD including unstable angina ($n = 82$), stable angina ($n = 48$) and myocardial infarction ($n = 31$). Basic characteristics of the participants are presented in Table 1. Approximately 60% of the study population was female. The mean (SD) BMI was 27.94 (4.68) kg m^{-2}. Approximately 24% of participants had hypertension and 20.2% were current smoker. We also found a high prevalence of dyslipidaemia and obesity within this population: approximately 85% and 30%, respectively.

Food consumption data for the 22 predefined food groups were entered into the factor analysis procedure. The Scree plot of eigenvalues indicated two major patterns. Factor-loading matrixes for the two major factors are listed in Table 2. The larger the loading factor of a food item or group, the greater the contribution of that food item or group to a specific factor. The 'Balanced' dietary pattern was characterised by high positive loadings for the consumption of green leafy vegetables, other vegetables, fruits, dairy products, red meats, poultry, seafoods, legumes and nuts, as well as a negative loading factor for sugar consumption. The second pattern, which we labelled the 'Western' dietary pattern, showed positive

Table 1 Characteristics of the study participants

Variables	Total ($n = 5706$)
Demographic and lifestyle	
Gender, male, % (*n*)	41.1 (2343)
Age (years)	48.25 (8.12)
Marriage status, % (*n*)	
Single/divorced/widow	6.3 (360)
Married	93.7 (5346)
Education (year)	8.17 (4.87)
Hypertension, % (*n*)	23.8 (1359)
Diabetes, % (*n*)	8.1 (462)
Dyslipidaemia, % (*n*)	85.20 (4862)
Obesity, % (*n*)	30.20 (1720)
Smoking status, % (*n*)	
Nonsmoker	69.9 (3990)
Ex-smoker	9.9 (565)
Current smoker	20.2 (1151)
Physical activity level	1.60 (0.29)
Physical measurements	
BMI (kg m^{-2})	27.94 (4.68)
Waist circumference (cm)	94.68 (12.12)
Hip circumference (cm)	103.62 (9.40)
MAC (cm)	30.49 (4.05)
Systolic blood pressure (mmHg)	121.99 (18.65)
Diastolic blood pressure (mmHg)	79.41 (10.98)
Lipid and metabolic markers	
Fasting plasma glucose (mg dL^{-1})	91.20 (35.29)
Cholesterol (mg dL^{-1})	191.84 (38.18)
Triglycerides (mg dL^{-1})	120 (85–171)
LDL-C (mg dL^{-1})	115.79 (35.07)
HDL-C (mg dL^{-1})	43.09 (9.95)
Psychological factors	
Anxiety score	10.59 (9.79)
Depression score	12.69 (9.49)

Data are presented as the mean (SD) or median (interquartile range) for continuous variables and as numbers and percentages for categorical variables. Hypertension was defined as systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg. Diabetes was defined as fasting blood glucose ≥126. Dyslipidaemia was defined as total cholesterol ≥200, or triglycerides ≥150, or low-density lipoprotein cholesterol (LDL-C) ≥130, or high-density lipoprotein cholesterol (HDL-C) <40 (for men) and HDL-C <50 (for women). Obesity was defined as body mass index (BMI) ≥ 30.
MAC, mid-upper arm circumference.

loadings for the consumption of sugar, tea, egg, snacks, fast foods, potato, carbonated beverages, pickled foods, organs meat and butter.

We calculated the mean values or percentages of selected CVD risk factors based on two identified dietary patterns. Those in the highest tertile of Balanced dietary pattern tended to be significantly older, taller and more educated. They had lower WC, HC, WHR and MAC and a higher weight, BMI and TG than those in the first tertile. Moreover, smoking habits were lower in the highest tertile versus lowest tertile of Balanced dietary pattern (Table 3). On the other hand, those in the highest tertile of Western dietary pattern tended to be significantly less educated, older and were less likely to be ex-smokers or current smokers than the two other tertiles. Subjects in the third tertile of Western dietary pattern had significantly higher BMI, WHR, SBP, DBP, FBG, TC and TG. Conversely, height and MAC were significantly lower among subjects in the third tertile. As expected, the percentages of patients with obesity, diabetes, hypertension and dyslipidaemia were significantly higher in the third tertile of Western dietary pattern (Table 3).

Table 2 Factor-loading matrix for major dietary patterns identified using food consumption data obtained from the food frequency questionnaire

Food items	Balanced dietary pattern	Western dietary pattern
Other vegetables	0.64	–
Green leafy vegetables	0.60	–
Fruits	0.52	–
Dairy products	0.48	–
Red meats	0.39	–
Poultry	0.34	–
Seafoods	0.28	–
Legumes	0.23	–
Nuts	0.23	–
Coffee	–	–
Sugar	–0.20	0.59
Tea	–	0.48
Egg	–	0.47
Snacks	–	0.42
Fast foods	–	0.40
Potato	–	0.38
Carbonated beverages	–	0.35
Pickled foods	–	0.29
Organs meat	–	0.28
Butter	–	0.27
Refined grains	–	–
Whole grains	–	–

Foods or food groups with factor loadings <0.20 for both factors were excluded.

The Balanced dietary pattern was associated with a reduced age-adjusted HRs (95% CI) of stable angina (HR = 0.32; 95% CI = 0.11–0.93; $P = 0.04$). However, this dietary pattern showed no significant association with multivariable adjusted HRs (95% CI) of myocardial infarction, stable angina, unstable angina and total CVD (Table 4). The Western dietary pattern showed a significant positive association with crude and multivariable adjusted HRs (95% CI) of total CVD. The crude HRs (95% CI) of total CVD in the highest compared to the lowest tertile of Western dietary pattern was 2.18 (95% CI = 1.16–4.08; $P = 0.01$). The multivariable HRs (95% CI) of total CVD in the highest versus lowest tertile of Western dietary pattern was 2.21 (95% CI = 1.08–4.45; $P = 0.03$) (Table 5).

Discussion

In this cohort study, two major dietary patterns were identified: a 'Balanced' dietary pattern and a 'Western' dietary pattern. The Balanced dietary pattern, characterised by high intakes of green leafy vegetables, other vegetables, fruits, dairy products, red meats, poultry, seafood, legumes and nuts, was not significantly associated with the risk of CVD incidence. The Western dietary pattern, characterised by high intakes of sugar, tea, egg, snacks, fast foods, potato, carbonated beverages, pickled foods, organs meat and butter, was significantly associated with a higher incidence of CVD. Both patterns were significantly associated with CVD risk factors at baseline, including BMI, WC, HC, WHR, SBP, DBP, FBG, TC, TG and HDL-C.

The relationship between dietary patterns and CVD events has been previously investigated in different parts of the world [5,7,8,36]. In the present study, the Balanced dietary pattern, which was considered as a healthy dietary pattern, showed no significant association with CVD incidence. The Balanced dietary pattern presented in the present study was similar to the prudent dietary pattern described in the Nurses' Health Study and Health Professionals Studies [7,37,38], except for consuming protein from all food sources, especially red meat. This difference might explain the observed beneficial effects of food groups of the prudent dietary patterns (including fruits, vegetables, poultry and seafood) on CVD risk. By contrast, it is possible that the positive effects of vegetables and seafood consumption attenuate the harmful effects of red meat and red meat products [39]. Several studies have reported that adherence to prudent dietary pattern was associated with significant decrease in CVD incidence [7,39,40]. The results of a review and meta-analysis study have indicated that a prudent/healthy dietary pattern was inversely associated with CVD mortality [41]. Lack of an

Table 3 Cardiovascular disease risk factors of participants according to tertiles of Balanced and Western dietary patterns

	Balanced dietary pattern			
	T1	T2	T3	*P*
Age (year)*	47.98 (0.17)	48.51 (0.17)	48.93 (0.26)	0.005
Education level, % (*n*)				
Low (trade school)	67 (1562)	45.1 (1084)	33.8 (327)	<0.001
Moderate (high school)	27.5 (641)	40.6 (974)	46.4 (449)	
High (university)	5.6 (130)	14.3 (343)	19.8 (191)	
Smoking status, % (*n*)				
Nonsmoker	65.4 (1527)	72.6 (1745)	74.3 (718)	<0.001
Ex-smoker	10.7 (249)	9.7 (232)	8.7 (84)	
Current smoker	23.9 (558)	17.8 (427)	17.1 (165)	
Physical activity level*	1.59 (0.01)	1.57 (0.005)	1.58 (0.01)	0.12
Weight (kg)*	70.86 (0.26)	73.49 (0.26)	73.16 (0.40)	<0.001
Height (cm)*	161.30 (0.001)	162.10 (0.001)	162.10 (0.002)	<0.001
BMI (kg m^{-2})*	27.31 (0.09)	28.01 (0.09)	27.90 (0.15)	<0.001
Obesity, % (*n*)	28.70 (670)	31.80 (764)	29.60 (286)	0.065
WC (cm)*	94.47 (0.25)	95.06 (0.25)	92.97 (0.39)	<0.001
HC (cm)*	103.14 (0.19)	103.51 (0.19)	102.67 (0.30)	0.05
WHR*	0.92 (0.002)	0.92 (0.002)	0.90 (0.003)	<0.001
MAC (cm)*	30.35 (0.09)	30.62 (0.08)	30.24 (0.13)	0.02
Diabetes, % (*n*)	7.8 (182)	7.7 (185)	9.8 (95)	0.10
Hypertension, % (*n*)	23.6 (550)	24.2 (582)	23.5 (227)	0.84
Dyslipidaemia, % (*n*)	84.30 (1967)	86.10 (2071)	85.20 (824)	0.205
SBP (mmHg)*	122.23 (0.39)	121.98 (0.38)	121.90 (0.61)	0.87
DBP (mmHg)*	79.28 (0.23)	79.68 (0.23)	79.80 (0.36)	0.34
FBG (mg dL^{-1})*	91.02 (0.75)	90.44 (0.73)	92.69 (1.15)	0.25
Cholesterol (mg dL^{-1})*	190.02 (0.80)	191.72 (0.78)	192.41 (1.24)	0.17
TG (mg dL^{-1})*	118 (83–167)	122 (87–173)	122 (86–177)	0.01
LDL-C (mg dL^{-1})*	115.89 (0.74)	115.24 (0.72)	114.47 (1.14)	0.57
HDL-C (mg dL^{-1})*	42.81 (0.20)	42.47 (0.20)	42.69 (0.31)	0.48
	Western dietary pattern			
	T1	T2	T3	*P*
Age (year)*	46.49 (0.31)	47.09 (0.16)	50.31 (0.17)	<0.001
Education level, % (*n*)				
Low (trade school)	44.8 (317)	50.8 (1253)	55.5 (1403)	<0.001
Moderate (high school)	43.7 (309)	37 (913)	33.3 (842)	
High (university)	11.5 (81)	12.1 (299)	11.2 (284)	
Smoking status, % (*n*)				
Nonsmoker	53 (375)	67.8 (1673)	76.8 (1942)	<0.001
Ex-smoker	14.6 (103)	9.8 (242)	8.7 (220)	
Current smoker	32.4 (229)	22.4 (553)	14.5 (368)	
Physical activity level*	1.58 (0.01)	1.57 (0.005)	1.60 (0.006)	0.72
Weight (kg)*	72.68 (0.48)	72.40 (0.25)	72.43 (0.26)	0.87
Height (cm)*	162.70 (0.002)	162.20 (0.001)	160.90 (0.001)	<0.001
BMI (kg m^{-2})*	27.54 (0.17)	27.55 (0.09)	27.99 (0.10)	0.002
Obesity, % (*n*)	23.50 (166)	28.40 (700)	33.80 (854)	<0.001
WC (cm)*	94.16 (0.47)	94.43 (0.24)	94.72 (0.26)	0.52
HC (cm)*	103.56 (0.35)	103.37 (0.18)	103.04 (0.19)	0.31
WHR*	0.91 (0.003)	0.91 (0.002)	0.92 (0.002)	0.004
MAC (cm)*	30.74 (0.16)	30.52 (0.08)	30.31 (0.09)	0.03
Diabetes, % (*n*)	4.1 (29)	6.4 (157)	10.9 (276)	<0.001
Hypertension, % (*n*)	20.5 (145)	20.9 (516)	27.6 (698)	<0.001
Dyslipidemia, % (*n*)	79.50 (562)	84.80 (2093)	87.20 (2207)	<0.001

Table 3 Continued

	Western dietary pattern			
	T1	T2	T3	*P*
SBP (mmHg)*	119.76 (0.71)	120.43 (0.37)	124.14 (0.37)	<0.001
DBP (mmHg)*	78.57 (0.42)	78. 89 (0.22)	80.16 (0.22)	<0.001
FBG (mg dL^{-1})*	86.11 (1.36)	88.57 (0.71)	95.28 (0.75)	<0.001
Cholesterol (mg dL^{-1})*	187.89 (1.47)	190.15 (0.77)	192.70 (0.81)	0.01
TG (mg dL^{-1})*	116 (77–165)	118 (85–167)	124 (87–176)	0.001
LDL-C (mg dL^{-1})*	112.34 (1.35)	115.91 (0.71)	115.42 (0.74)	0.06
HDL-C (mg dL^{-1})*	43.29 (0.37)	42.52 (0.19)	42.54 (0.20)	0.16

*Adjusted for sex.

Data are presented as the mean (SD) or median (interquartile range). An independent sample *t*-test and the Kruskal–Wallis test were used where appropriate.

BMI, body mass index; DBP, diastolic blood pressure; FBG, fasting blood glucose; HC, hip circumference; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; MAC, mid- upper arm circumference; SBP, systolic blood pressure; TG, triglyceride; WC, waist circumference; WHR, waist-to-hip ratio.

Table 4 Hazard ratio (95% confidence intervals) of cardiovascular events according to tertiles of Balanced dietary pattern

	Tertiles of Balanced dietary pattern			
	T1 (low)	T2	T3 (high)	*P*
Person years	19 592	20 436	8236	
Myocardial infarction				
Number of cases	13	11	7	
Crude HR	1	0.82 (0.37–1.83)	1.30 (0.52–3.27)	0.57
Age-adjusted HR	1	0.79 (0.35–1.77)	1.24 (0.49–3.11)	0.647
Multivariable HR*	1	0.71 (0.30–1.66)	1.09 (0.38–3.12)	0.874
Stable angina				
Number of cases	28	15	5	
Crude HR	1	0.52 (0.28–0.97)	0.43 (0.17–1.12)	0.08
Age-adjusted HR	1	0.50 (0.26–0.93)	0.41 (0.16–1.05)	0.064
Multivariable HR*	1	0.54 (0.28–1.06)	0.43 (0.15–1.23)	0.115
Unstable angina				
Number of cases	34	29	19	
Crude HR	1	0.83 (0.49–1.42)	1.08 (0.56–2.07)	0.82
Age-adjusted HR	1	0.79 (0.48–1.30)	1.20 (0.68–2.13)	0.526
Multivariable HR*	1	0.79 (0.47–1.33)	1.18 (0.61–2.27)	0.617
Total CVD				
Number of cases	75	55	31	
Crude HR	1	0.82 (0.50–1.35)	1.35 (0.77–2.36)	0.30
Age-adjusted HR	1	0.68 (0.48–0.96)	0.91 (0.60–1.39)	0.671
Multivariable HR*	1	0.69 (0.47–0.99)	0.90 (0.56–1.46)	0.674

*The hazard ratio (HR) was adjusted for age (35–48 years and ≥49 years), sex, body mass index, smoking status (nonsmoker, ex-smoker and current smoker), education level (low, moderate, high), marital status (married, single, divorced, widow), total energy intake, diabetes mellitus, hypertension and dyslipidaemia.

CI, confidence interval; CVD, cardiovascular disease.

association between Balanced dietary pattern, as a healthy pattern, and CVD events in the present study might be a result of the high consumption of red meat. Our results are comparable to those of British Regional Heart Study that demonstrated no significant association between 'prudent' dietary pattern and cardiovascular events or mortality in older adults. The prudent dietary pattern described by Atkins *et al.* [42] was characterised by a high consumption of poultry, fish, vegetables, legumes, fruits, pasta and rice and wholemeal bread, which is relatively similar to the 'Balanced' dietary pattern in the present study.

Table 5 Hazard ratio (95% confidence intervals) of cardiovascular events according to tertiles of Western dietary pattern

	Tertiles of Western dietary pattern			
	T1 (low)	T2	T3 (high)	*P*
Person years	6051	20 848	21 365	
Myocardial infarction				
Number of cases	2	14	15	
Crude HR	1	2.01 (0.46–8.85)	2.11 (0.48–9.22)	0.32
Age-adjusted HR	1	1.97 (0.45–8.67)	1.70 (0.39–7.46)	0.482
Multivariable HR*	1	2.21 (0.47–10.35)	1.88 (0.35–10.01)	0.457
Stable angina				
Number of cases	3	18	27	
Crude HR	1	1.73 (0.51–5.87)	2.54 (0.77–8.37)	0.13
Age-adjusted HR	1	1.69 (0.50–5.75)	2.04 (0.62–6.73)	0.244
Multivariable HR*	1	1.75 (0.49–6.26)	2.08 (0.53–8.06)	0.291
Unstable angina				
Number of cases	6	33	43	
Crude HR	1	1.58 (0.66–3.78)	2.02 (0.86–4.75)	0.11
Age-adjusted HR	1	1.50 (0.63–3.59)	1.59 (0.67–3.74)	0.290
Multivariable HR*	1	1.90 (0.76–4.74)	2.26 (0.84–6.07)	0.106
Total CVD				
Number of cases	11	65	85	
Crude HR	1	1.70 (0.90–3.22)	2.18 (1.16–4.08)	0.01
Age-adjusted HR	1	1.64 (0.86–3.11)	1.73 (0.92–3.25)	0.088
Multivariable HR[10]	1	1.91 (0.98–3.73)	2.13 (1.04–4.38)	0.04

*The hazard ratio (HR) was adjusted for age (35–48 years and ≥49 years), sex, body mass index, smoking status (nonsmoker, ex-smoker and current smoker), education level (low, moderate, high), marital status (married, single, divorced, widow), total energy intake, diabetes mellitus, hypertension, and dyslipidaemia.

CI, confidence interval; CVD, cardiovascular disease.

Although recent studies have shown that a Western dietary pattern is significantly associated with an increased risk of CVD [7,14,39,40], several previous studies have found no significant association between Western dietary pattern and cardiovascular events [12,41,43]. Previous studies evaluating the associations between major dietary patterns and CVD risk in Iran found similar categories of dietary patterns. Mirmiran *et al.* [14] found two major dietary patterns in Tehranian adults and labelled the patterns as Western and Traditional patterns. Mirmiran *et al.* [14] found that a higher Western dietary pattern score was associated with increased risk of CVD events [14]. In another study conducted by Mohammadifard *et al.* [14], four major dietary patterns were obtained that were different from the patterns obtained in the present study. Likewise, Mohammadifard *et al.* [14] also observed no significant association between Western dietary pattern and CVD events. A Western dietary pattern usually leads to obesity and an excessive accumulation of white adipose tissue that increases systemic inflammation and inflammatory mediators such as C-reactive protein. This might be a potential mechanism by which the Western dietary pattern increases the risk of CVD [44,45]. Oikonomou *et al.* [46] reported that there is a relationship between Western dietary pattern, as an unhealthy dietary pattern, and severity of coronary artery lesions in adults with stable coronary artery disease. Furthermore, we have previously found an association between adherence to the Western dietary pattern and risk of metabolic syndrome [47], a major risk factor for cardiovascular morbidity and mortality [48].

In the present study, a Balanced dietary pattern was associated with lower WC, HC, WHR, MAC and smoking. Moreover, the Western dietary pattern was significantly associated with a higher BMI, WHR, SBP, DBP, TG, TC and FBG concentrations. These findings are in accordance with previous studies that report an inverse association between a prudent/healthy dietary pattern and BMI, WC and WHR [49–51]. It has also been observed that adherence to a prudent/healthy dietary pattern was associated with lower smoking habits [52]. Sun *et al.* [53] found three major dietary patterns and labelled them as healthy, Balanced and Western patterns. In agreement with our findings, Sun *et al.* [53] indicated that adherence to Western pattern is associated with higher BMI, WC, systolic and diastolic blood pressures, and TG concentration compared to healthy and Balanced dietary patterns. In another study, the Western dietary pattern was

associated with higher serum TC and HDL-C concentrations in women and higher LDL-C concentration in both sexes, although the lowest Western pattern score was associated with higher systolic and diastolic blood pressures compared with the highest score [54]. Berg *et al.* [50] reported that a high consumption of starch, sugar, saturated fatty acids and trans-fatty acids and low intakes of omega-3, natural antioxidants and fibre from fruits and vegetables, which are indicative of a Western dietary pattern, are associated with metabolic syndrome components (including high blood pressure and WC, as well as high TC, TG and LDL-C concentrations).

Adherence to a Western dietary pattern was associated with a higher obesity rate and consequently a higher CVD risk in the present analysis. Obesity and being overweight are the most important causes of mortality and disability as a result of CVD. Obesity is associated with an increase in the blood pressure, blood lipid profiles and blood glucose levels, and also changes in body weight are associated with a change in these risk factors. The results of a cohort study in five south American countries showed that obesity has a weak, moderate and strong correlation with myocardial infarction, stroke and high blood pressure, respectively [55]. Obesity has a major impact on CVD incidence such as heart failure, coronary artery disease, sudden cardiac death and atrial fibrillation and is also associated with reduced survival. Despite these unfavourable effects, several studies have demonstrated the paradox of obesity and the results of these studies suggest that individuals who are overweight and obese, and who also experienced cardiovascular diseases such as high blood pressure, heart failure, coronary artery disease and peripheral vascular disease, have a better prognosis compared to those who are not obese and overweight [56,57]. One of the possible mechanisms reported in these studies was that adipose tissue expresses a soluble receptor for tumor necrosis factor-α that could play a protective role against tumor necrosis factor-α and neutralise its effects. Also, overweight and obese individuals have a higher arterial pressure and can tolerate higher doses of CVD medications. However, more studies are needed in this area and targeted weight loss with respect to preventing and treating CVD is supported based on the complete information of recent studies [58].

Although we did not examine the role of epigenetics on the association of diet with CVD risk, several studies have focused on this. The effect of diet as one of the CVD risk factors, on the atherogenesis process could be partly attributed to epigenetic changes, which have not been studied sufficiently in this area. Recent investigations described several mechanisms for the role of epigenetics in this association such as the methylation of several genes correlated with atherosclerosis, which can also be caused by inflammation [59]. It was also reported that unfavourable metabolic or mental health phenotypes could develop among individuals whose mothers exposed to adverse situations such as famine during pregnancy [60]. An increased weight of birth is one of the possible causes of higher ischaemic heart disease mortality in this group of adults [61]. The methylation patterns in epigenetic alterations vary in coronary heart disease patients compared to healthy individuals. Therefore, we can hypothesise that these patterns would increase the risk of CVD. However, further investigations are required to determine whether epigenetic changes can lead to CVD over time and also whether they can be inherited [59].

Strengths and limitations

The main strength of the present study was the cohort design, which was effective in indicating causality. Individuals with diabetes mellitus were excluded from the analysis because this group are at high risk for CVD events. Moreover, three models of HRs were reported and the last one of these was adjusted for possible confounding factors. The present study was conducted in a large population-based sample of Iranian. Furthermore, most of CVD risk factors have been reported in relation to identified dietary patterns. Finally, the HR and 95% CIs were adjusted for a wide range of confounding factors and possible CVD risk factors that indicate a robust association of CVD events with dietary patterns.

The present study was conducted in a representative sample of the Iranian population aged 35–65 years and may not be generalisable to other populations or other age groups. Other limitations of the present study included the subjective decision made at different levels of factors interpretation because factor analysis is an exploratory method. The dietary patterns of all participants were obtained by completing the FFQ one time at the baseline of the study. The accuracy of dietary assessment could be improved if the FFQ could be completed more than once with definite intervals because it is possible that the dietary intake of participants has been changed during this period, which can influence the results of the study. Moreover, we did not examine the effect of epigenetics, genes and family history of CVD on the association of dietary patterns with CVD risk because inheritance plays an important role in the development of CVD based on previous evidence [62].

Conclusions

The results of the present study have demonstrated two major dietary patterns among patients with CVD events including Balanced and Western dietary patterns. The

Balanced dietary pattern showed no significant association with CVD incidence, whereas the Western dietary pattern was associated with increased CVD events among Iranian adults. We also found independent associations between dietary patterns and anthropometric measurements, blood pressure, serum lipids and other cardiometabolic biomarkers, further supporting the protective effects of Balanced pattern and unfavorable effects of Western pattern on CVD risk factors.

Transparency declaration

The lead author affirms that this manuscript is an honest, accurate and transparent account of the study being reported. The reporting of this work is compliant with STROBE guidelines. The lead author affirms that no important aspects of the study have been omitted and that any discrepancies from the study as planned have been explained.

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Conflict of interests, source of funding and authorship

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ZA wrote the first draft of the paper. MGM, AHB, MM and MS contributed to the conception and design of the research. MS, FS, MSK, SD, SH, TH and FM collected the data. MT and HE contributed to acquisition and analysis of the data. GAF critically revised the manuscript. All authors agree to be fully accountable for ensuring the integrity and accuracy of the work, and read and approved the final manuscript submitted for publication.

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