Habitual and meal-specific carbohydrate quality index and their relation to metabolic syndrome in a sample of Iranian adults

Maryam Majdi
Tehran University of Medical Sciences School of Medicine https://orcid.org/0000-0002-9945-7785
Hossein Imani
Tehran University of Medical Sciences
Elham Bazshahi
Tehran University of Medical Sciences
Fatemeh Hosseini
Tehran University of Medical Sciences
Kurosh Djafarian
Tehran University of Medical Sciences
Azadeh Lesani
Tehran University of Medical Sciences
Zahra Akbarzade
Tehran University of Medical Sciences
Sakineh Shab-Bidar (s_shabbidar@tums.ac.ir)
Tehran University of Medical Sciences

Research

Keywords: carbohydrate quality index (CQI), metabolic syndrome, meal-specific, cross-sectional study

Posted Date: March 30th, 2021

DOI: https://doi.org/10.21203/rs.3.rs-323180/v1

Abstract

Objective: Most studies on diet quality have focused on the habitual and overall intake of foods without considering intakes at specific eating occasions**.** The aim of this study was to assess the relationship between habitual and meal-specific carbohydrate quality index (CQI) and metabolic syndrome (MetS) in Iranian adults.

Methods: In this cross-sectional study, data from 850 participants were analyzed. Dietary information was obtained from a 3-day non-consecutive 24 hours recall. CQI was calculated from three criteria: dietary fiber, glycemic index, and solid carbohydrate/available carbohydrate ratio. The association between CQI and MetS was assessed by logistic regression.

Results: The prevalence of MetS in the lowest and highest tertile of CQI were 30.1 and 33.7, respectively (P=0.6). We identified, the trend of elevated FBG with CQI in dinner meal was statistically significant. In habitual diet and all three meals, we failed to find any significant association between tertiles of CQI and MetS either before or after adjustment for covariates**.**

Conclusion: The results of this study showed that CQI was not associated with MetS and its components. Further investigations into the mechanisms underlying the role of carbohydrate quality in developing metabolic disorders are warranted.

Introduction

Metabolic syndrome (MetS) represents an interrelated metabolic disorder characterized by central obesity, deviant glucose hemostasis, lipid disorders including elevated triglyceride levels, and low high-density lipoprotein cholesterol, and high blood pressure[1]. The prevalence of the MetS among adults in the US was estimated between 34.3% and 38.5%[2], and in some European countries, at least 25%[3]. Recent published data from Iran show that the prevalence of MetS is 21.1%[4]. Based on the evidence, a combination of genetic and environmental factors has been shown to play a role in developing this syndrome[5, 6]. Among environmental factors, meal-timing, meal frequency, and dietary quality may play an important role in cardiometabolic risk factor management[7]. Meal-timing can affect circadian rhythm and cardiometabolic risk factors[8–10]. Epidemiological studies indicated that eating meals at the wrong time of the day increases the risk of obesity, type2 diabetes, and cardiovascular disease (CVD)[11, 12]. Limited studies have been performed on meal frequency (number of daily eating occasions) and cardiometabolic risk factors that had different results. One study showed that meal frequency was inversely related to high triglycerides (TG), high blood pressure (BP), and obesity[13]. Another study conducted on individuals with type1 diabetes found that skipping breakfast was associated with higher blood glucose concentration and lower odds of good glycemic control. In this study, a median of 6 daily meals was reported[14]. Besides, individuals who take three meals/day than individuals who received one meal/day, lower systolic and diastolic blood pressure, lower cholesterol level, and higher TG concentration have been reported[15].

In recent decades, the effort to prevent MetS has focused on reducing risk factors and promoting healthy behaviors, especially healthy eating habits[16]. In this regard, studies and evidence have shown that improving the quality of dietary carbohydrates, rather than modification of their quantity, may have a greater impact on modulating cardiac-metabolic risk factors[17]. Most of the available evidence has examined separately the various components of the quality of carbohydrates received, such as fiber intake, glycemic index, and glycemic load[18, 19]. Previous studies have explored the association between carbohydrate intake and MetS and have found positive[20–22], inconsistent[23], and null[24] effects. But since a single component cannot be appropriate criteria for evaluating the quality of carbohydrate revived, therefor border criteria that can accommodate several single components are used as the carbohydrate quality index (CQI), which is a convenient indicator of the quality of the carbohydrate intake. The CQI was defined by summing up the following criteria: glycemic index (GI), dietary fiber intake, whole grains to total grains ratio, and solid carbohydrates to available carbohydrates[25]. Since the use of whole grains in the Iranian diet is limited, in this study, its calculation has been abandoned. Few studies have investigated the association between CQI and pathological conditions. A cross-sectional study in Ghana indicated that a reverse association between CQI and abdominal obesity[26]. As well as the inverse association between COI and general obesity/overweight has been demonstrated in one prospective study[25]. Researchers examined the relationship between dietary patterns based on macronutrient intake and blood factors such as lipid profile and fasting blood glucose (FBG), but there has not been much attention paid to meals[27–29]. In addition, a dietary recommendation based on meals can be an effective intervention in changing inappropriate habitual intake[30].

Since carbohydrates as a major nutrient provide an important part of the energy requires of the adult population, it is assumed that carbohydrate intake may play a more prominent role in public health. Hence the purpose of this study is to determine the association between CQI of meal-specific dietary pattern and MetS and its components among Tehranian adults

Materials And Methods

2.1. Data and Study Participants

This cross-sectional study was done within 25 Health houses in the Tehran Metropolis. 850 adult participants aged between 18-65 years included. This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects were approved by the ethical standards of the Tehran University of Medical Sciences (ethic number: IR.TUMS.MEDICINE.REC.1399.797), which approved the protocol and informed consent form. All participants signed a written informed consent prior to the start of the study.

Adults with a previous history of any major illness such as myocardial infarction (MI), diabetes, cancer, renal disease, cardiovascular diseases (CVD) and who were not desired to contribute to the study were excluded from the study. Additionally, those who were experiencing any special diet or diet therapy were also excluded from the study. This study included healthy adults of both genders and living in the study region and willing to participate in this study.

2.2. Dietary assessment, meal timing, and CQI calculation

Dietary data were gathered by using repeated but non-consecutive (Monday to Sunday) 24-hour dietary recall method. The 24-hour meal was structured and included breakfast. The first 24-hour recall is obtained through interviews, the other two recalls are recorded by telephone during two repeated non-random days during the study, and information was recorded. Food and food groups were extracted through these questionnaires. Meals usually include breakfast, lunch, dinner, and snacks. If two or more meals were reported within 59 minutes, they were considered as one meal. Otherwise, mealtimes were coded with more energy content as a meal or as snacks. In some cases, lunch was not served, but dinner and supper were both reported (some people refer to the common concept of a conventional lunch in the United States, a half-day meal, dinner). If dinner and supper were more than an hour apart, and dinner had less energy than supper, dinner would be coded for lunch and supper for dinner. If more than one dinner was reported, the second dinner could not be recorded as lunch unless the first dinner has already been recorded as lunch to maintain the timing of the meal[31]

Finally, the total value of nutrients in all meals and snacks was calculated daily. CQI was calculated based on the energy-adjusted amount of total carbohydrate intake values calculated using the residual method[32]. CQI was defined by summing up the following four criteria: 1) ratio of solid carbohydrates to avaiable carbohydrates, 2) dietary fiber intake (g/day), 3) GI, and 4) ratio of whole grains to total grains (whole grains, refined grains, and their products). Subjects were categorized into quintiles and take a value (ranging from 1 to 5) for each quintile according to each of these four criteria; however, the scoring of GI was reversed; thus, those in the fifth quintile received one point, and those in the first quintile received five points. Finally, an overall CQI was computed by adding together all values of the four criteria (ranging from 4 to 20). It was also ranked into quintiles[25]. But Given that the Iranians are using whole grains in the diet is limited, this component is not calculated in this calculation, so the final score ranges from 3 to 15.

GI values were obtained from international tables[33], the glycemic index of Iranian foods[34], and literature reviews. Glucose was used as the reference (GI for glucose= 100). The mean of the GI values was assigned if more than one eligible GI value was available for a specific food item. The carbohydrate content of each food was determined using standard portion sizes from the United States Department of Agriculture food composition databases [35]. All nuts and vegetables except starchy roots, considered as very low GI (ranged from 10 to 20). Solid carbohydrates were obtained by subtracting the amount of liquid carbohydrate (summing up sweetened beverages and fruit juice) from total carbohydrate intake.

2.3. Anthropometric assessment and biochemical tests

Weight was measured using a Seca weighing scale (Seca and Co. KG; 22 089 Hamburg, Germany; Model: 874 1321009; designed in Germany; made in China) with light clothing (without a coat and raincoat). A wall stadiometer board was used for the height of participants without shoes with a sensitivity of 0.1 cm height measurements. BMI was calculated as weight (in kilograms) divided by height (in meters squared). Normal weight was defined as BMI ≤24.9 kg/m2, and the presence of overweight and obesity as BMI=25–29.99 kg/m2 and ≥30 kg/m2, respectively[36]. Waist circumference (WC) was measured according to the guiding protocol of WHO, at the midpoint between the lower border of the rib cage and the iliac crest, using a non-stretchable fiberglass measuring tape[37] and classified according to the International Diabetes Federation criteria[1]. The waist- to- height ratio (WHtR) was defined as the WC divided by the measured height. WHtR ≥0.5 was adopted for overweight and abdominal obesity for the purpose of uniformity regarding age differences[38]. Blood pressure is measured by a digital barometer (BC 08, Beurer, Germany) after at least 10-15 minutes of rest and sitting. Blood pressure was measured twice for each person, and the average blood pressure was reported for each person. Of all participants, 10 ml of fasting blood was taken between 7-10 a.m. in the acid-washed test tubes without anticoagulant until after room temperature maintenance (RT Temperature (RT)) for 30 minutes. Minute blood clots and centrifuge at 1500 g for 20 minutes. The serums are poured into micro-clean tubes and stored in the 80 CC freezer until the test. Measurement of serum sugar and fat was performed using the enzymatic method, based on colorimetry, using commercial kits (Pars Azmoun, Iran) with the automatic machine (Selecta E, Vitalab, Netherland). The tests were performed on people on the same day as the blood draw.

2.4. Sociodemographic and lifestyle variables

General information such as age, marital status, smoking status, living situation (alone or with someone), and disease status by asking participants with a general information questionnaire registered. International Physical Activity Questionnaire (IPAQ) is used to examine people's physical activity[39], which records three intensity levels of activity based on the metabolic equivalents (METs). METs were classified as low (< 600 MET-minutes/week), moderate (600–3,000 MET-minutes/week), and vigorous (> 3,000 MET-minutes/week).

2.5. Metabolic syndrome

MetS and its components were defined using the following criterion(1). Individuals who have at least three or more of the following disorders were classified as having MetS: high waist circumference (≥88 for women and ≥102 for men); elevated triglyceride levels (≥150 mg/dl); low HDL-C levels (≤50 mg/dl for women and ≤40 mg/dl for men); high blood pressures (systolic blood pressure ≥ 130 mmHg and diastolic blood pressure ≥ 85 mmHg) or use of anti-hypertensive medication; and high fasting glucose levels (≥ 100 mg/dl) or use of hypoglycemic medication.

2.6. Statistical analysis

Energy-adjusted dietary CQI was used to classify participants into tertiles. According to the type of variables, the comparison of quantitative mean variables between the tertiles of subject characteristics and anthropometric measurement was performed using ANOVA (one-way variance analysis) and comparison of qualitative variables distribution between the tertiles with Chi-2 square test. Logistic regression was performed to investigate the relationship between CQI as an independent variable and MetS and its components as a dependent variable in an unadjusted and multivariable adjusted model. In this regard, age, sex, energy intake, physical activity, marital status, smoking status, educated status, underlying disease, and BMI were included as covariates in the modified regression model. All statistical analyses were done using IBM Statistical Package for Social Sciences (V.22; SPSS Inc.), and p<0.05 was considered statistically significant.

Results

The mean age of study participants with MetS was 46.1 ± 10, and the mean BMI with MetS was 29.2 ± 4.71. The prevalence of MetS among participants in the lowest and highest tertiles of CQI were 30.1 and 33.7, respectively (P = 0.6). The mean CQI in participants with MetS was 9.15 ± 2.83 **(Supplementary Table 1).**

30, 24, and 5 participants were removed from breakfast, lunch, and dinner, respectively, due to the lack of enough information for the analysis and lack of cooperation in their dietary intake reports. As a result, 820, 826, and 845 participants remained in the study for final analysis at breakfast, lunch, and dinner meals.

General characteristics of study participants according to carbohydrate quality score based on habitual diet and meal is indicated in Table 1. Within lunch meal, those in the top tertiles of CQI were less likely to be a current smoker (p = 0.05). In habitual diet and all three meals, the distribution of participants in terms of other general characteristics across tertiles of CQI was not significantly different.

Table 1
General characteristic of study participants according to tertiles (T) of Carbohydrate Quality Index (CQI)

	CQI (breakfast)				**CQI (lunch)**				**CQI (dinner)**				**CQI (habitual)**			
	T1 (3–7)	**T2 (7–11)**	**T3 (11–15)**	**P**	**T1 (3–7)**	**T2 (7–11)**	**T3 (11–15)**	**P**	**T1 (3–7)**	**T2 (7–11)**	**T3 (11–15)**	**P**	**T1 (3–7)**	**T2 (7–11)**	**T3 (11–15)**	**P**
Participant	273	274	273		275	276	275		281	282	282		276	277	276	
Sex																
Male%	30	40	30	0.1	37.7	33.3	29	0.3	32.9	32.3	34.8	0.9	27.8	31.9	40.3	0.1
Female%	33.9	31.9	34.2	0.1	32.3	33.7	34	0.3	33.3	33.6	33	0.9	34.5	33.7	31.8	0.1
Age	42.7 ± 11	42.2 ± 10	42 ± 11.6	0.7	42.3 ± 11	42.7 ± 10.8	42 ± 11	0.7	41.5 ± 10.7	43.3 ± 10.9	42.1 ± 11	0.1	41.8 ± 10.7	42.5 ± 10.6	42.4 ± 10.4	0.7
Educated	33.3	33.8	32.9	0.6	33.7	32.5	33.8	0.03	33.4	33.2	33.4	0.9	33.6	33.6	32.8	0.7
Marital status (married %)	32.6	34.9	32.5	0.2	33.2	34.1	32.7	0.8	34.2	33.1	32.7	0.4	32.9	33.8	33.2	0.7
Smoking (smoker %)	23.3	37.2	39.5	0.3	50	30.6	19.4	0.05*	32.6	34.9	32.6	0.9	34.1	36.6	29.3	0.8
Underlying disease (yes %) ‡	35.8	31.3	32.8	0.3	31	34.8	34.2	0.4	31.7	34.8	33.4	0.6	32.5	34.3	33.1	0.8
Activity score																
Low	34.1	32.5	33.4	0.9	34.2	32.4	31.6	0.3	32.1	34.3	33.6	0.5	34.6	33.9	31.6	0.3
Moderate	32.5	34.7	32.8	0.9	32.4	32.4	35.3	0.3	34.8	32.6	32.6	0.5	33.9	32.9	33.2	0.3
High	28.8	34.2	37	0.9	31.6	30.3	38.2	0.3	35.1	32.5	32.5	0.5	24.3	32.4	43.2	0.3
BMI	27.6 ± 7.31	27.2 ± 4.63	27.1 ± 4.63	0.5	27.4 ± 6.84	27 ± 5.27	27.4 ± 5.27	0.6	27.1 ± 4.18	27.7 ± 7.04	27.1 ± 5.3	0.2	27 ± 4.42	27.3 ± 4.52	27.3 ± 4.59	0.6
Weight (kg)	72.4 ± 14.6	72.3 ± 13.6	71.8 ± 13.5	0.8	72.5 ± 13.8	71.6 ± 13.9	72.1 ± 13.9	0.7	71.8 ± 11.6	72.5 ± 14.7	72 ± 15.1	0.8	71.6 ± 13	72.71 ± 4.5	72.8 ± 13.5	0.5
Height (cm)	162.4 ± 9.88	163 ± 8.84	162.5 ± 8.38	0.7	162.9 ± 9.51	162.6 ± 8.13	162.3 ± 9.46	0.7	162.8 ± 8.76	162.1 ± 9.34	162.9 ± 8.89	0.4	162.2 ± 9.13	162.8 ± 8.5	162.9 ± 8.9	0.5
WC (cm)	89.4 ± 12.3	89 ± 12.9	89.1 ± 12.1	0.9	89.5 ± 12.1	88.6 ± 11.4	89.1 ± 12.7	0.6	88.8 ± 11.1	89.2 ± 12.1	89.3 ± 13	0.8	88 ± 11.6	90 ± 12	89.6 ± 11.5	0.09

Values are means ± standard deviations (SD) or percentages

† Chi-square test used for categorical variables, one-way ANOVA for continuous variables

* *P* < 0.05

‡ Underlying disease: Including diabetes, hypertension, dyslipidemia, cardiovascular disease, stroke, cancer, respiratory disease and osteoporosis

Abbreviation: *BMI* body mass index; *WC* waist circumference

Kg kilogram; *cm* centimeter

In breakfast meal, participants in the top tertiles of CQI had lower TG and SBP compared with subjects in the lowest. Thus, we found a significant association between tertiles in participants for TG (P = 0.04). In addition, no significant statistical differences were found in other terms of laboratory characteristics across tertiles of CQI in all three meals (Table 2).

Table 2
Laboratory results of study participants according to tertiles (T) of Carbohydrate Quality Index (CQI)

	CQI (breakfast)				**CQI (lunch)**				**CQI (dinner)**				**CQI (habitual)**			
	T1 (3–7)	**T2 (7–11)**	**T3 (11–15)**	**P**	**T1 (3–7)**	**T2 (7–11)**	**T3 (11–15)**	**P**	**T1 (3–7)**	**T2 (7–11)**	**T3 (11–15)**	**P**	**T1 (3–7)**	**T2 (7–11)**	**T3 (11–15)**	**P**
Participants	273	274	273		275	276	275		281	282	282		276	277	276	
FBG (mg/dl)	108.4 ± 36.8	108.4 ± 36.6	105.6 ± 28	0.5	108.1 ± 34.6	106.7 ± 27.8	108.5 ± 39.7	0.8	104.1 ± 28	109.7 ± 35.6	109.5 ± 37.9	0.08	104.8 ± 28.2	110.4 ± 44.7	108.5 ± 33.1	0.1
TG (mg/dl)	152.8 ± 83.2	139.3 ± 73.1	138.5 ± 70.7	0.04*	145.2 ± 72.9	151.6 ± 87.6	139.9 ± 76.9	0.2	137.1 ± 75.1	148.7 ± 86	149.1 ± 75.7	0.1	141.7 ± 77	145.2 ± 78.8	146.7 ± 76.3	0.7
TC (mg/dl)	193.6 ± 41.9	196.4 ± 46.7	196.1 ± 45.4	0.7	196.4 ± 45.9	195.5 ± 45.1	195.4 ± 44.4	0.9	192.2 ± 41.6	195.9 ± 47.4	199.3 ± 45	0.1	193.3 ± 44.3	198.3 ± 48.9	196.8 ± 41.3	0.4
HDL-C (mg/dl)	49.2 ± 10.5	50.4 ± 9.98	49.9 ± 9.99	0.3	49.8 ± 10.7	50 ± 9.9	49.4 ± 9.86	0.7	49.9 ± 9.75	50.1 ± 10.7	49.6 ± 10.1	0.8	50.5 ± 10.2	50.2 ± 10.7	49 ± 9.68	0.1
SBP (mmHg)	118.4 ± 18	114.2 ± 22.4	116.6 ± 19.8	0.05*	115.9 ± 20.3	117.1 ± 20.6	116.2 ± 20.2	0.7	115.1 ± 23.2	117.3 ± 20.1	116.8 ± 17	0.3	116.5 ± 14.7	119 ± 14.6	118.4 ± 15.7	0.1
DBP (mmHg)	78.8 ± 13.6	77.8 ± 13.5	78 ± 12.2	0.6	78.2 ± 12.8	78.8 ± 13.5	77.4 ± 13.3	0.4	77.7 ± 14.5	79 ± 13.3	77.6 ± 11.4	0.3	78.9 ± 8.88	78.6 ± 10.3	78.7 ± 9.75	0.9

Data are presented as mean ± standard deviation (SD).

†One-way ANOVA test used for assessment variables.

* All values were adjusted for energy intake.

* P < 0.05

Abbreviations: *FBG* fasting blood glucose; *TG* triglyceride; *HDL-C* high density lipoprotein-cholesterol; *SBP* systolic blood pressure; *DBP* diastolic blood pressure

mg milligram; *dl* deciliter

The selected dietary intake of study participants across tertiles of CQI is shown in **Supplementary Table 2 and** Table 3. In habitual diet, we observed a significant association between tertiles in participants for linolenic acid, total sugar, glycemic index (P < 0.001 for all), and total fiber (P = 0.002). Within breakfast meal, dietary intake of total sugar and glycemic index were significantly different across tertiles of CQI (P < 0.001 for all). In lunch meal, dietary intakes of total fiber, total sugar, and glycemic index were significantly different across tertiles of CQI among participants (p = 0.001 for all). Moreover, the total energy intake of participants was significant across tertiles of CQI (P = 0.005). Within the dinner meal, participants in the top of tertiles of CQI had a higher intake of carbohydrate and a lower intake of SFA. Moreover, dietary intakes of total fiber, total sugar, and glycemic index were significantly different across tertiles of CQI (p = 0.001 for all).

Table 3
Odds ratio (OR) and 95% CI for metabolic syndrome and components among the study participants according to tertiles (T) of Carbohydrate Quality Index (CQI) in habitual diet

	CQI				
	T1 **(3–7)**	**T2** **(7–11)**	**T3** **(11–15)**	**P-value**	**P-trend**
Participant	276	277	276		
Mets†					
Crude	1.00	1.13 (0.79–1.63)	1.18(0.82–1.69)	0.6	0.3
Model 1	1.00	1.11 (0.76–1.63)	1.21 (0.83–1.76)	0.5	0.3
Model 2	1.00	1.11 (0.75–1.64)	1.25 (0.85–1.85)	0.5	0.2
Model 3	1.00	1.08 (0.72–1.61)	1.24 (0.83–1.86)	0.5	0.3
Abdominal obesity					
Crude	1.00	1.3 (0.93–1.83)	1.06 (0.76–1.49)	0.2	0.7
Model 1	1.00	1.32 (0.92–1.89)	1.15 (0.8–1.66)	0.3	0.4
Model 2	1.00	1.32 (0.92–1.91)	1.14 (0.79–1.65)	0.3	0.4
Model 3	1.00	1.31 (0.84–2.05)	1.11 (0.71–1.74)	0.4	0.6
Elevated BP‡					
Crude	1.00	1.24 (0.76–2.03)	1.36 (0.84–2.21)	0.4	0.2
Model 1	1.00	1.21 (0.72–2.02)	1.34 (0.81–2.21)	0.5	0.2
Model 2	1.00	1.16 (0.68–1.97)	1.25 (0.74–2.11)	0.6	0.4
Model 3	1.00	1.13 (0.66–1.93)	1.25 (0.73–2.13)	0.7	0.4
Elevated FBG					
Crude	1.00	1.34 (0.96–1.88)	1.4 (1-1.97)	0.09	**0.04***
Model 1	1.00	1.37 (0.97–1.94)	1.44 (1.02–2.03)	0.07	**0.03***
Model 2	1.00	1.36 (0.96–1.93)	1.46 (1.03–2.07)	0.07	**0.03***
Model 3	1.00	1.36 (0.95–1.93)	1.45 (1.02–2.06)	0.08	**0.03***
Low HDL-C					
Crude	1.00	0.96 (0.69–1.34)	1 (0.72–2.4)	0.9	0.9
Model 1	1.00	1 (0.71–1.41)	1.06 (0.75–1.49)	0.9	0.7
Model 2	1.00	0.98 (0.69–1.39)	1.08 (0.76–1.54)	0.8	0.7
Model 3	1.00	0.98 (0.69–1.39)	1.08 (0.76–1.53)	0.8	0.7
Elevated TG					
Crude	1.00	1.1 (0.78–1.56)	1.2 (0.85–1.7)	0.5	0.2
Model 1	1.00	1.07 (0.75–1.53)	1.14 (0.8–1.63)	0.7	0.4

Binary logistic regression test used for assessment variables.

Crude: unadjusted model

Model 1: adjusted for age, gender and energy intake.

Model 2: additionally, adjusted for marital status, physical activity, education status, smoking, and metabolic disorder

Model 3: further adjustment was made for BMI.

†Defined as the presence of ≥ 3 of the following components: abdominal obesity (waist circumference > 88 for women and > 102 for men); elevated blood pressure (BP ≥ 130/85 mmHg); elevated fasting blood glucose (FBG ≥ 100 mg/dl); low high density lipoprotein-cholesterol (HDL-C < 50 for women and < 40 for men); elevated triglyceride (TG ≥ 150 mg/dl)

‡Elevated blood pressure (systolic ≥ 130 and diastolic ≥ 85)

Abbreviations: *Mets* metabolic syndrome; *BP* blood pressure; *FBG* fasting blood glucose; *HDL-C*: high density lipoprotein cholesterol; *TG* triglyceride

	CQI				
Model 2	1.00	1.08 (0.75–1.54)	1.19 (0.83–1.71)	0.6	0.3
Model 3	1.00	1.07 (0.74–1.53)	1.18 (0.82–1.69)	0.6	0.3
Binary logistic regression test used for assessment variables.					
Crude: unadjusted model					
Model 1: adjusted for age, gender and energy intake.					
Model 2: additionally, adjusted for marital status, physical activity, education status, smoking, and metabolic disorder					
Model 3: further adjustment was made for BMI.					
†Defined as the presence of ≥ 3 of the following components: abdominal obesity (waist circumference > 88 for women and > 102 for men); elevated blood pressure (BP ≥ 130/85 mmHg); elevated fasting blood glucose (FBG ≥ 100 mg/dl); low high density lipoprotein-cholesterol (HDL-C < 50 for women and < 40 for men); elevated triglyceride (TG ≥ 150 mg/dl)					
‡Elevated blood pressure (systolic ≥ 130 and diastolic ≥ 85)					
Abbreviations: *Mets* metabolic syndrome; *BP* blood pressure; *FBG* fasting blood glucose; *HDL-C*: high density lipoprotein cholesterol; *TG* triglyceride					

Multivariate- adjusted odds ratio for MetS and its component across tertiles of habitual and meal-specific CQI is indicated in Table 3 **and** Table 4. In habitual diet and also in all three meals, we failed to find any significant association between tertiles of CQI and MetS either before or after adjustment for covariates. Within lunch meal, after adjustment for potential cofounding, CQI being close to a significant level associated with elevated BP (p-value = 0.05). Actually, participants in the highest tertiles had less elevated blood pressure compared to lower tertiles. In addition, no overall significant association was observed between CQI and other components of MetS either before and after adjustment for covariates in habitual diet and all three meals. It should be noted that the trend of elevated FBG across tertiles of CQI was marginally significant in habitual diet (OR: 1.45; 95% CI: 1.02–2.06 for trend p = 0.03) and in dinner meal (OR: 1.39; 95% CI: 0.98–1.96 for trend p = 0.05).

Table 4

Odds ratio (OR) and 95% CI for metabolic syndrome and components among the study participants according to tertiles (T) of Carbohydrate Quality Index (CQI)

	CQI (breakfast)					CQI (lunch)					CQI (dinner)				
	T1 (3–7)	T2 (7–11)	T3 (11–15)	P-value	P-trend	T1 (3–7)	T2 (7–11)	T3 (11–15)	P-value	P-trend	T1 (3–7)	T2 (7–11)	T3 (11–15)	P-value	P-trend
Participant	273	274	273			275	276	275			281	282	282		
Mets†															
Crude	1.00	0.7 (0.48-1)	0.77 (0.53–1.11)	0.1	0.1	1.00	0.97 (0.67–1.39)	1.01 (0.7–1.46)	0.9	0.9	1.00	1.04 (0.72–1.5)	1.31 (0.92–1.88)	0.2	0.1
Model 1	1.00	0.71 (0.49–1.04)	0.78 (0.53–1.13)	0.1	0.1	1.00	0.96 (0.66–1.39)	1.02 (0.7–1.49)	0.9	0.8	1.00	0.96 (0.66–1.4)	1.29 (0.89–1.86)	0.2	0.1
Model 2	1.00	0.72 (0.49–1.06)	0.79 (0.53–1.16)	0.2	0.2	1.00	0.96 (0.65–1.41)	1.03 (0.69–1.51)	0.9	0.8	1.00	0.93 (0.63–1.37)	1.32 (0.9–1.92)	0.1	0.1
Model 3	1.00	0.74 (0.5–1.11)	0.8 (0.54–1.2)	0.3	0.2	1.00	1.01 (0.67–1.5)	1.01 (0.67–1.51)	0.9	0.8	1.00	0.88 (0.59–1.32)	1.32 (0.89–1.94)	0.1	0.1
Abdominal obesity															
Crude	1.00	0.83 (0.59–1.17)	0.88 (0.63–1.24)	0.5	0.4	1.00	1.07 (0.77–1.51)	1.25 (0.89–1.76)	0.4	0.1	1.00	0.92 (0.66–1.29)	0.99 (0.71–1.38)	0.8	0.9
Model 1	1.00	0.85 (0.6–1.21)	0.9 (0.63–1.27)	0.6	0.5	1.00	1.04 (0.73–1.48)	1.3 (0.92–1.85)	0.2	0.1	1.00	0.84 (0.59–1.18)	0.96 (0.68–1.35)	0.5	0.8
Model 2	1.00	0.87 (0.61–1.24)	0.95 (0.67–1.37)	0.7	0.8	1.00	0.99 (0.69–1.41)	1.24 (0.86–1.77)	0.3	0.2	1.00	0.85 (0.59–1.2)	0.99 (0.7–1.4)	0.5	0.9
Model 3	1.00	0.87 (0.57–1.31)	0.96 (0.63–1.45)	0.7	0.8	1.00	1.05 (0.69–1.59)	1.29 (0.85–1.97)	0.4	0.2	1.00	0.79 (0.53–1.19)	1.03 (0.68–1.54)	0.4	0.9
Elevated BP‡															
Crude	1.00	0.82 (0.5–1.34)	0.97 (0.6–1.56)	0.7	0.9	1.00	1.48 (0.92–2.39)	0.97 (0.58–1.63)	0.1	0.9	1.00	1.41 (0.87–2.28)	1.06 (0.64–1.75)	0.2	0.5
Model 1	1.00	0.88 (0.53–1.47)	1 (0.6–1.64)	0.8	0.9	1.00	1.56 (0.95–2.57)	0.96 (0.56–1.64)	0.1	0.9	1.00	1.27 (0.77–2.08)	1 (0.59–1.69)	0.5	0.9

Binary logistic regression test used for assessment variables.

Crude: unadjusted model

Model 1: adjusted for age, gender and energy intake.

Model 2: additionally, adjusted for marital status, physical activity, education status, smoking, and metabolic disorder

Model 3: further adjustment was made for BMI.

†Defined as the presence of ≥ 3 of the following components: abdominal obesity (waist circumference > 88 for women and > 102 for men); elevated blood pressure (BP ≥ 130/85 mmHg); elevated fasting blood glucose (FBG ≥ 100 mg/dl); low high density lipoprotein-cholesterol (HDL-C < 50 for women and < 40 for men); elevated triglyceride (TG ≥ 150 mg/dl)

‡Elevated blood pressure (systolic ≥ 130 and diastolic ≥ 85)

Abbreviations: *Mets* metabolic syndrome; *BP* blood pressure; *FBG* fasting blood glucose; *HDL-C*: high density lipoprotein cholesterol; *TG* triglyceride

	CQI (breakfast)					**CQI (lunch)**					**CQI (dinner)**				
Model 2	1.00	0.92 (0.54–1.56)	0.98 (0.58–1.65)	0.9	0.9	1.00	1.49 (0.87–2.55)	0.93 (0.52–1.66)	0.1	0.9	1.00	1.27 (0.76–2.12)	0.99 (0.58–1.7)	0.5	0.9
Model 3	1.00	0.97 (0.56–1.66)	1.03 (0.6–1.75)	0.9	0.8	1.00	1.73 (1.01–2.95)	0.97 (0.55–1.71)	0.05*	0.9	1.00	1.2 (0.71–2.02)	0.96 (0.56–1.65)	0.6	0.8
Elevated FBG															
Crude	1.00	0.81 (0.58–1.13)	0.77 (0.55–1.09)	0.2	0.1	1.00	0.94 (0.67–1.33)	0.85 (0.6–1.21)	0.6	0.3	1.00	1.28 (0.92–1.79)	1.39 (1-1.95)	0.1	**0.04***
Model 1	1.00	0.82 (0.58–1.16)	0.79 (0.55–1.11)	0.3	0.1	1.00	0.94 (0.66–1.33)	0.85 (0.6–1.21)	0.6	0.3	1.00	1.21 (0.86–1.7)	1.38 (0.98–1.94)	0.1	**0.06**
Model 2	1.00	0.83 (0.58–1.18)	0.83 (0.58–1.18)	0.4	0.3	1.00	0.95 (0.67–1.35)	0.86 (0.6–1.23)	0.7	0.4	1.00	1.21 (0.86–1.7)	1.39 (0.98–1.95)	0.1	**0.05***
Model 3	1.00	0.84 (0.59–1.19)	0.84 (0.59–1.96)	0.5	0.3	1.00	0.98 (0.68–1.39)	085 (0.6–1.23)	0.6	0.4	1.00	1.19 (0.84–1.68)	1.39 (0.98–1.96)	0.1	**0.05***
Low HDL-C															
Crude	1.00	0.71 (0.5–0.99)	0.9 (0.64–1.26)	0.1	0.5	1.00	0.7 (0.5–0.98)	0.9 (0.64–1.27)	0.1	0.5	1.00	0.86 (0.61–1.2)	0.97 (0.7–1.36)	0.6	0.9
Model 1	1.00	0.7 (0.5–0.99)	0.9 (0.64–1.26)	0.1	0.5	1.00	0.72 (0.51–1.01)	0.89 (0.63–1.25)	0.1	0.5	1.00	0.87 (0.62–1.21)	0.98 (0.7–1.37)	0.6	0.9
Model 2	1.00	0.7 (0.49–0.99)	0.87 (0.62–1.23)	0.1	0.4	1.00	0.67 (0.48–0.96)	0.87 (0.61–1.23)	0.08	0.4	1.00	0.86 (0.61–1.21)	1.01 (0.72–1.41)	0.5	0.9
Model 3	1.00	0.7 (0.49–0.99)	0.88 (0.62–1.24)	0.1	0.4	1.00	0.68 (0.48–0.96)	0.87 (0.61–1.23)	0.09	0.4	1.00	0.85 (0.61–1.2)	1.01 (0.72–1.41)	0.5	0.9
Elevated TG															
Crude	1.00	0.83 (0.58–1.17)	0.81 (0.57–1.15)	0.4	0.2	1.00	0.94 (0.67–1.33)	0.85 (0.6–1.21)	0.6	0.3	1.00	1.12 (0.79–1.58)	1.3 (0.92–1.84)	0.3	0.1
Model 1	1.00	0.84 (0.59–1.19)	0.82 (0.58–1.17)	0.5	0.2	1.00	0.94 (0.66–1.33)	0.85 (0.6–1.21)	0.6	0.3	1.00	1.08 (0.76–1.53)	1.29 (0.91–1.82)	0.3	0.1

Binary logistic regression test used for assessment variables.

Crude: unadjusted model

Model 1: adjusted for age, gender and energy intake.

Model 2: additionally, adjusted for marital status, physical activity, education status, smoking, and metabolic disorder

Model 3: further adjustment was made for BMI.

†Defined as the presence of ≥ 3 of the following components: abdominal obesity (waist circumference > 88 for women and > 102 for men); elevated blood pressure (BP ≥ 130/85 mmHg); elevated fasting blood glucose (FBG ≥ 100 mg/dl); low high density lipoprotein-cholesterol (HDL-C < 50 for women and < 40 for men); elevated triglyceride (TG ≥ 150 mg/dl)

‡Elevated blood pressure (systolic ≥ 130 and diastolic ≥ 85)

Abbreviations: *Mets* metabolic syndrome; *BP* blood pressure; *FBG* fasting blood glucose; *HDL-C*: high density lipoprotein cholesterol; *TG* triglyceride

	CQI (breakfast)					CQI (lunch)					CQI (dinner)				
Model 2	1.00	0.88 (0.62–1.26)	0.85 (0.59–1.22)	0.6	0.3	1.00	0.95 (0.67–1.35)	0.86 (0.6–1.23)	0.7	0.4	1.00	1.06 (0.74–1.51)	1.28 (0.9–1.81)	0.3	0.1
Model 3	1.00	0.89 (0.62–1.28)	0.86 (0.6–1.24)	0.7	0.4	1.00	0.98 (0.68–1.39)	0.85 (0.6–1.23)	0.6	0.4	1.00	1.04 (0.73–1.48)	1.28 (0.9–1.81)	0.3	0.1

Binary logistic regression test used for assessment variables.

Crude: unadjusted model

Model 1: adjusted for age, gender and energy intake.

Model 2: additionally, adjusted for marital status, physical activity, education status, smoking, and metabolic disorder

Model 3: further adjustment was made for BMI.

†Defined as the presence of ≥ 3 of the following components: abdominal obesity (waist circumference > 88 for women and > 102 for men); elevated blood pressure (BP ≥ 130/85 mmHg); elevated fasting blood glucose (FBG ≥ 100 mg/dl); low high density lipoprotein-cholesterol (HDL-C < 50 for women and < 40 for men); elevated triglyceride (TG ≥ 150 mg/dl)

‡Elevated blood pressure (systolic ≥ 130 and diastolic ≥ 85)

Abbreviations: *Mets* metabolic syndrome; *BP* blood pressure; *FBG* fasting blood glucose; *HDL-C*: high density lipoprotein cholesterol; *TG* triglyceride

Discussion

The present study examined the relationship between Habitual and meal-specific CQI and odds of MetS and its components in a sample of Iranian adults. Our findings showed a non-significant association between CQI with MetS either before and after adjustment for potential confounders in all three meals and habitual diet. Also, we found no significant link between MetS components after adjustment for covariates. However, there was an increasing trend for elevated FBG in dinner meal and habitual diet.

Carbohydrates are a heterogeneous class of nutrients, and the consumption of refined carbohydrates for enhancing the quality of received carbohydrates from a public health perspective has been suggested(40). In this direction, the available studies on adults indicate that total carbohydrate intake or dietary carbohydrate proportion is not associated with the risk of obesity(41, 42). According to a report from a population with a balanced diet and lower carbohydrate intake, carbohydrate quality is more important factor to determine diet quality compared with the quality of fat(43) as far as, it has been proposed that a reduction in fat intake was compensated with an increased intake of refined starches and sugars(44). There is compelling evidence that carbohydrate quality has important effects on the progression and treatment of CVD, the MetS, T2D, and obesity(45). Aspects of carbohydrate quality that may be important in these components include dietary fiber, whole-grain, GI, and GL, and in particular, the intake of sugar-sweetened drinks. However, their properties are often highly inter-related, and it may be difficult to implicate one over another in any particular condition(45). Therefore, in this context, this seems to be more important in Iranian populations because of higher intake of carbohydrate(46).

To our knowledge, there has been no observational study examining the relationship between integrated carbohydrate quality in meals and metabolic disorders. Dietary approaches derive from meal timing are a hopeful plan for the modulation of circadian rhythms and clock-controlled metabolic functions in humans. Besides, studies have proposed that certain time windows are more suitable for the consumption of carbohydrate-rich or fat-rich food to maintain metabolic health. In a cross-over trial, Kessler and Pivovarova-Ramich(47) investigated that consumption of high carb meals in the evening has an unfavorable influence on blood glucose level and glycemic control in individuals with impaired glucose metabolism. In agreement with this finding, other studies in humans suggested that a carbohydrate-rich diet at the beginning of the day could be safe against the development of diabetes and MetS(48, 49). In the modern lifestyle, the main meal is dinner (50). Western main meals end with a sweet dessert. In addition, drinking beverages during the main meal was considered to be a part of modern lifestyle, as well as consuming special foods for breakfast that differ largely from the foods eaten at other meals (51). On the other hand, skipping breakfast is very common in modern societies. In study of eight young men, in the first condition, participants ate three main meals (breakfast, lunch, and dinner), while in the other condition, the same amount of energy was consumed at lunch and dinner times only. They found that skipping breakfast increased the average blood glucose concentration during the afternoon and sleep, subsequently resulting in an overall increased 24-hour average blood glucose concentration (52). Another study assessed the glucose metabolism of healthy adults in two conditions of breakfast skipping and dinner skipping. They showed, breakfast skipping resulted in higher glucose concentrations and insulin resistance after lunch (53). In line with our findings in habitual diets, a cross-sectional study conducted on Korean adults observed no significant associations between CQI and T2DM or MetS, although the quality of carbohydrate consumed is associated with the risk of obesity and high blood pressure(54). Also, in another study, there was no association between GI and GL and MetS(55).

Contrary to our findings, a cross-sectional study from Ghana demonstrates that the diet with high CQI levels inversely related to general and abdominal obesity(56). In another study identified that consuming refined foods with high carbohydrate content was a positive association with a higher risk of abdominal obesity in Ghanaian University students(57). A cohort study from Spain in university graduates reported an inverse association between dietary CQI and general obesity(58). The result of a systematic review and meta-analysis study that were focusing on the association between carbohydrate quality and NCDs incidence and metabolic biomarkers showed that daily consumption of dietary fiber was associated with a reduced risk of health-related consequence(59).

These findings are supported by cohort studies, which report a reduced risk of coronary heart disease incidence and mortality and incidence of diabetes. However glycemic index of a meal can also be affected by its protein and fat content, in that dietary fat slows carbohydrate absorption from the gut, although this effect is probably minimal within the context of a mixed meal[60–62].

High carbohydrate diets, which are common in developing nations, especially Asian countries, contain a high content of refined sources (such as white rice and white bread), which are low in fiber. These types of diets usually reflect poor food quality and mainly have high GI content, which can lead to negative metabolic consequences[63–65]. According to the National Food Consumption Survey, most of the calorie intake in Iranian people, which is about more than 60%, obtained from carbohydrates. In other words, the dietary intake of carbohydrates among Iranians is 450 g per day (rural areas: 413 g/d and urban areas: 518 g/d)[66]. Due to these data and consumption of whole grains in the Iranian diet is limited, so the quality of carbohydrates in this study is lower than other populations, which included whole grains.

For this reason, we may not have been to see an exact relationship between CQI with MetS and its components. The content of dietary fiber and vitamin-minerals in whole-grains is higher than refined carbohydrates. The protective effects of these nutrients against the risk of chronic diseases are well-known[67, 68]. Due to their physical structure and dietary fiber content, whole grains are categorized as low GI foods. As mentioned earlier, most of the dietary carbohydrate intake in Iran is in the form of refined grains, which usually contain a higher value than the GI and GL[34]. The use of whole grains is a useful way to increase the amount of fiber in the diet and reduce the risk of non-communicable disease (NCDs). In addition, fruits and vegetables are important factors in fiber intake in the diet. According to the above, our calculated CQI has low quality, but due to the consumption of medium to high fiber content in this study, it can be concluded that the intake of fruits and vegetables and solid carbohydrates in the diet of individuals are higher. These substances contain fructose. Fructose is found in large amounts in sucrose (table sugar), high fructose corn sweetener, natural fruit juices, and fruits. Fructose also led to a significant elevation in fasting glucose, insulin levels and decreased insulin sensitivity[45]. As our results, in this study showed a significant increase trend in FBG. Although there were no statistically significant, the correlations between CQI and MetS and its components were positive. Based on the substantial role of carbohydrates in Iranian diets and their low-quality diets, it seems that focusing on the improve carbohydrate quality would be a practical and advantageous strategy to make better food choices among Iranians[46].

In prospective studies, the consumption of liquid carbohydrates was associated with weight gain, whereas there was an inverse association between consumption of solid carbohydrates and high weight gain[69–72]. Examination of the systematic evidence presented for the effect of long-term intervention with low GI and GL on fasting insulin level and proinflammatory markers showed that it could be effective in preventing obesity-related disease[73].

High intakes of dietary fiber and whole grains are more clearly associated with good health outcomes than measures of GI or GL. Although the glycemic index provides a measure of the glycemic potential of the carbohydrate content of foods, some low glycemic index foods might have other attributes that are not health-promoting. Foods with added fructose or sucrose, as well as mixed foods high in SFA and carbohydrate (i.e., confectionery products), may have a low GI[74]. findings from a dose-response meta-analysis showed that diets identified by low dietary fiber contribute to diverse NCDs and that administration of quantitative recommendations for dietary fiber intake will be beneficial. While consumption in the range of 25–29 g/day is sufficient, dose-responses data showed that amounts greater than 30 g/day have more benefits[59].

Given the effect of low GI in the development of obesity, there is evidence that low GI diets increase satiety by reducing voluntary food intake, thus reducing total energy intake, and it can be effective for body-weight maintenance and may prevent obesity[75–77]. In contrast, the intake of a high GI diet causes increases in hunger and subsequently leads to the increased intake of food, thus potentially affecting energy balance and body composition[78]. Fiber-containing foods should be chewed before passing through the stomach and into the small bowel, where they affect satiety, glucose and insulin responses, and lipid absorption. Whole foods that require chewing and retain much of their structure in the gut are more likely to cause a feeling of satiety, which in turn leads to weight loss and modulation of carbohydrate and lipid metabolism. In the large bowel, fiber is almost completely broken down by the resident microflora under a set of anaerobic reactions known as fermentation. The gut microbiota plays many important roles in human health[79].

The present study has important strengths. To the best of the authors' knowledge, this study was the first to investigate CQI in meals and its association with MetS and its components. We had a sufficient sample size in this study that was done within 25 Health houses in the Tehran Metropolis. Despite these strengths, the study has some limitations. First, this study had a cross-sectional design, and the findings do not establish causality between CQI and MetS; therefore, the results ought to be interpreted with caution. Second, using the questionnaire retrospectively may reduce information recall. There was also under-reporting and over-reporting of food items received. Third, even though the data were controlled for some potential confounders, the effects of eating behavior, menopausal status, and residual confounding cannot be discounted.

Conclusions

In conclusion, in the present study, CQI was not associated with MetS. Further investigations into the mechanisms underlying the role of carbohydrate quality in the development of metabolic disorders are warranted. Additionally to the quantity and quality of the food we eat, time to eat and the frequency of eating is also an important aspect of healthy eating habits. Optimization of mealtimes has significant benefits for the prevention of chronic diseases and great promise for lifestyle interventions in the near future.

Declarations

Ethical approval and consent to participate

This study was approved by the Tehran University of Medical Sciences (TUMS) (Ethics no. IR.TUMS.MEDICINE.REC.1399.797).

Consent for publication

This study was conducted according to the guidelines laid down in the Declaration of Helsinki all procedures involving research study participants were approved by the Tehran University of Medical Sciences. Written informed consent was obtained from all subjects/patients.

Availability of data and materials

The data that support the findings of this study are available from the corresponding author, **Sakineh Shab-Bidar**, upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

Funding Source

This research received no specific grant from any funding agency, commercial or not-for-profit sectors**.**

Authors Contributions

Sakineh Shab-Bidar, **Hossein Imani**, and **Kurosh Djafarian** considered and developed the idea for the paper and revised the manuscript; **Maryam Majdi**, **Fatemeh Hosseini**, and **Azadeh Lesani** contributed to the analysis and, **Maryam Majdi**, **Elham Bazshahi**, and **Zahra Akbarzade** wrote the drafts. All authors read and approved the final manuscript.

Acknowledgments

We thank all participants of the study for their cooperation. We also thank **Dr**.**Sakineh Shab-Bidar**, **Dr**.**Hossein Imani**, **Dr**.**Kurosh Djafarian** and **Azadeh Lesani** for their scientific consultations.

Authors Information

Not applicable.

References

1. Alberti K, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, et al. Harmonizing the metabolic syndrome: a joint interim statement of the international diabetes federation task force on epidemiology and prevention; national heart, lung, and blood institute; American heart association; world heart federation; international atherosclerosis society; and international association for the study of obesity. Circulation. 2009;120(16):1640-5. DOI: 10.1161/CIRCULATIONAHA.109.192644
2. Ford ES, Li C, Zhao G. Prevalence and correlates of metabolic syndrome based on a harmonious definition among adults in the US. Journal of diabetes. 2010;2(3):180-93. DOI: 10.1111/j.1753-0407.2010.00078.x.
3. Martínez MA, Puig JG, Mora M, Aragón R, O'Dogherty P, Antón JL, et al. Metabolic syndrome: prevalence, associated factors, and C-reactive protein: the MADRIC (MADrid RIesgo Cardiovascular) Study. Metabolism. 2008;57(9):1232-40. DOI: 10.1016/j.metabol.2008.04.017.
4. Mazloomzadeh S, Khazaghi ZR, Mousavinasab N. The prevalence of metabolic syndrome in Iran: a systematic review and meta-analysis. Iranian journal of public health. 2018;47(4):473.
5. Hollenberg N. Genetic versus environmental etiology of the metabolic syndrome among male and female twins. Current hypertension reports. 2002;4(3):178-. DOI: 10.1007/s11906-002-0003-8
6. Yosaee S, Khodadost M, Esteghamati A, Speakman JR, Shidfar F, Nazari MN, et al. Metabolic syndrome patients have lower levels of adropin when compared with healthy overweight/obese and lean subjects. American journal of men's health. 2017;11(2):426-34.
7. St-Onge MP, Ard J, Baskin ML, Chiuve SE, Johnson HM, Kris-Etherton P, et al. Meal Timing and Frequency: Implications for Cardiovascular Disease Prevention: A Scientific Statement From the American Heart Association. Circulation. 2017;135(9):e96-e121. DOI: 10.1161/CIR.0000000000000476.
8. Froy O. Metabolism and circadian rhythms--implications for obesity. Endocrine reviews. 2010;31(1):1-24. DOI: 10.1210/er.2009-0014
9. Wang JB, Patterson RE, Ang A, Emond JA, Shetty N, Arab L. Timing of energy intake during the day is associated with the risk of obesity in adults. Journal of human nutrition and dietetics : the official journal of the British Dietetic Association. 2014;27 Suppl 2:255-62. DOI: 10.1111/jhn.12141
10. Maugeri A, Kunzova S, Medina-Inojosa JR, Agodi A, Barchitta M, Homolka M, et al. Association between eating time interval and frequency with ideal cardiovascular health: Results from a random sample Czech urban population. Nutrition, metabolism, and cardiovascular diseases : NMCD. 2018;28(8):847-55. DOI: 10.1016/j.numecd.2018.04.002
11. Antunes LC, Levandovski R, Dantas G, Caumo W, Hidalgo MP. Obesity and shift work: chronobiological aspects. Nutrition research reviews. 2010;23(1):155-68. DOI: 10.1017/S0954422410000016
12. Jiang P, Turek FW. Timing of meals: when is as critical as what and how much. American journal of physiology Endocrinology and metabolism. 2017;312(5):E369-e80. DOI: 10.1152/ajpendo.00295.2016
13. Ha K, Song Y. Associations of Meal Timing and Frequency with Obesity and Metabolic Syndrome among Korean Adults. Nutrients. 2019;11(10). DOI: 10.3390/nu11102437

14. Ahola AJ, Mutter S, Forsblom C, Harjutsalo V, Groop PH. Meal timing, meal frequency, and breakfast skipping in adult individuals with type 1 diabetes - associations with glycaemic control. Scientific reports. 2019;9(1):20063. DOI: 10.1038/s41598-019-56541-5
15. Stote KS, Baer DJ, Spears K, Paul DR, Harris GK, Rumpler WV, et al. A controlled trial of reduced meal frequency without caloric restriction in healthy, normal-weight, middle-aged adults. The American journal of clinical nutrition. 2007;85(4):981-8. DOI: 10.1093/ajcn/85.4.981
16. Pérez-Martínez P, Mikhailidis DP, Athyros VG, Bullo M, Couture P, Covas MI, et al. Lifestyle recommendations for the prevention and management of metabolic syndrome: an international panel recommendation. Nutrition reviews. 2017;75(5):307-26. DOI: 10.1093/nutrit/nux014
17. Martínez-González MA, Fernandez-Lazaro CI, Toledo E, Díaz-López A, Corella D, Goday A, et al. Carbohydrate quality changes and concurrent changes in cardiovascular risk factors: a longitudinal analysis in the PREDIMED-Plus randomized trial. The American Journal of Clinical Nutrition. 2020. DOI: 10.1093/ajcn/nqz298
18. Juanola-Falgarona M, Salas-Salvadó J, Buil-Cosiales P, Corella D, Estruch R, Ros E, et al. Dietary glycemic index and glycemic load are positively associated with risk of developing metabolic syndrome in middle-aged and elderly adults. Journal of the American Geriatrics Society. 2015;63(10):1991-2000. DOI: 10.1111/jgs.13668.
19. Zhang J-Y, Jiang Y-T, Liu Y-S, Chang Q, Zhao Y-H, Wu Q-J. The association between glycemic index, glycemic load, and metabolic syndrome: a systematic review and dose–response meta-analysis of observational studies. European journal of nutrition. 2019:1-13. DOI: 10.1007/s00394-019-02124-z
20. Kwon Y-J, Lee H-S, Lee J-W. Association of carbohydrate and fat intake with metabolic syndrome. Clinical nutrition. 2018;37(2):746-51. DOI: 10.1016/j.clnu.2017.06.022
21. Ahn J, Kim N-S, Lee B-K, Park S. Carbohydrate intake exhibited a positive association with the risk of metabolic syndrome in both semi-quantitative food frequency questionnaires and 24-hour recall in women. Journal of Korean medical science. 2017;32(9):1474-83. DOI: 10.3346/jkms.2017.32.9.1474
22. Ha K, Kim K, Chun OK, Joung H, Song Y. Differential association of dietary carbohydrate intake with metabolic syndrome in the US and Korean adults: data from the 2007–2012 NHANES and KNHANES. European journal of clinical nutrition. 2018;72(6):848-60.
23. DOI: 10.1038/s41430-017-0031-8
24. Kim K, Yun SH, Choi BY, Kim MK. Cross-sectional relationship between dietary carbohydrate, glycaemic index, glycaemic load and risk of the metabolic syndrome in a Korean population. British journal of nutrition. 2008;100(3):576-84. DOI: 10.1017/S0007114508904372
25. Bruscato NM, da Costa Vieira JL, do Nascimento NMR, Canto MEP, Stobbe JC, Gottlieb MG, et al. Dietary intake is not associated to the metabolic syndrome in elderly women. North American journal of medical sciences. 2010;2(4):182. DOI: 10.4297/najms.2010.2182
26. Santiago S, Zazpe I, Bes-Rastrollo M, Sánchez-Tainta A, Sayón-Orea C, de la Fuente-Arrillaga C, et al. Carbohydrate quality, weight change and incident obesity in a Mediterranean cohort: the SUN Project. European journal of clinical nutrition. 2015;69(3):297-302. DOI: 10.1038/ejcn.2014.187
27. Suara SB, Siassi F, Saaka M, Foroshani AR, Sotoudeh G. Association between Carbohydrate Quality Index and general and abdominal obesity in women: a cross-sectional study from Ghana. BMJ open. 2019;9(12). DOI: 10.1136/bmjopen-2019-033038
28. Tay J, Luscombe-Marsh ND, Thompson CH, Noakes M, Buckley JD, Wittert GA, et al. A very low-carbohydrate, low–saturated fat diet for type 2 diabetes management: a randomized trial. Diabetes Care. 2014;37(11):2909-18. DOI: 10.2337/dc14-0845
29. Zinn C, McPhee J, Harris N, Williden M, Prendergast K, Schofield G. A 12-week low-carbohydrate, high-fat diet improves metabolic health outcomes over a control diet in a randomised controlled trial with overweight defence force personnel. Applied Physiology, Nutrition, and Metabolism. 2017;42(11):1158-64. DOI: 10.1139/apnm-2017-0260
30. Stentz FB, Brewer A, Wan J, Garber C, Daniels B, Sands C, et al. Remission of pre-diabetes to normal glucose tolerance in obese adults with high protein versus high carbohydrate diet: randomized control trial. BMJ Open Diabetes Research and Care. 2016;4(1). DOI: 10.1136/bmjdrc-2016-000258
31. Myhre JB, Løken EB, Wandel M, Andersen LF. Meal types as sources for intakes of fruits, vegetables, fish and whole grains among Norwegian adults. Public Health Nutrition. 2015;18(11):2011-21.
32. Huang TTK, Roberts SB, Howarth NC, McCrory MA. Effect of screening out implausible energy intake reports on relationships between diet and BMI. Obesity research. 2005;13(7):1205-17. DOI: 10.1038/oby.2005.143
33. Willett W, Stampfer MJ. Total energy intake: implications for epidemiologic analyses. American journal of epidemiology. 1986;124(1):17-27. DOI: 10.1093/oxfordjournals.aje.a114366
34. Atkinson FS, Foster-Powell K, Brand-Miller JC. International tables of glycemic index and glycemic load values: 2008. Diabetes care. 2008;31(12):2281-3. DOI: 10.2337/dc08-1239
35. Taleban F, Esmaeili MJNN, publication FTRI. Glycemic index of Iranian foods. 1999.
36. US Department of Agriculture ARS. FoodData Central. USDA Washington DC; 2019.
37. Organization WH. Obesity: preventing and managing the global epidemic: World Health Organization; 2000.
38. Consultation W. Waist circumference and waist-hip ratio. Report of a WHO Expert Consultation Geneva: World Health Organization. 2008;2008:8-11.
39. Ashwell M, Hsieh SD. Six reasons why the waist-to-height ratio is a rapid and effective global indicator for health risks of obesity and how its use could simplify the international public health message on obesity. International journal of food sciences and nutrition. 2005;56(5):303-7. DOI: 10.1080/09637480500195066
40. Craig C, Marshall A, Sjostrom M, Bauman A, Lee P, Macfarlane D, et al. International Physical Activity Questionnaire-Short Form. 2017.
41. Roberts CK, Liu SJJotAoN, Dietetics. Carbohydrate intake and obesity: an association that needs “refining”. 2009;109(7):1163-4.
42. Iqbal SI, Helge JW, Heitmann BLJO. Do energy density and dietary fiber influence subsequent 5-year weight changes in adult men and women? 2006;14(1):106-14. DOI: 10.1038/oby.2006.13

43. Ludwig DS, Pereira MA, Kroenke CH, Hilner JE, Van Horn L, Slattery ML, et al. Dietary fiber, weight gain, and cardiovascular disease risk factors in young adults. 1999;282(16):1539-46. DOI: 10.1001/jama.282.16.1539
44. Zazpe I, Sanchez-Tainta A, Santiago S, de la Fuente-Arrillaga C, Bes-Rastrollo M, Martínez JA, et al. Association between dietary carbohydrate intake quality and micronutrient intake adequacy in a Mediterranean cohort: the SUN (Seguimiento Universidad de Navarra) Project. 2014;111(11):2000-9. DOI: 10.1017/S0007114513004364
45. Yancy WS, Wang C-C, Maciejewski MLJPhn. Trends in energy and macronutrient intakes by weight status over four decades. 2014;17(2):256-65. DOI: 10.1017/S1368980012005423
46. Slyper AHJJoPE, Metabolism. The influence of carbohydrate quality on cardiovascular disease, the metabolic syndrome, type 2 diabetes, and obesity–an overview. 2013;26(7-8):617-29. DOI: 10.1515/jpem-2012-0419
47. Azadbakht L, Mohammadifard N, Akhavanzanjani M, Taheri M, Golshahi J, Haghighatdoost FJIJoFS, et al. The association between dietary glycemic index, glycemic load and diet quality indices in Iranian adults: results from Isfahan Healthy Heart Program. 2016;67(2):161-9. DOI: 10.3109/09637486.2015.1134443
48. Kessler K, Pivovarova-Ramich O. Meal Timing, Aging, and Metabolic Health. International journal of molecular sciences. 2019;20(8). DOI: 10.3390/ijms20081911
49. Almoosawi S, Prynne CJ, Hardy R, Stephen AM. Time-of-day and nutrient composition of eating occasions: prospective association with the metabolic syndrome in the 1946 British birth cohort. International journal of obesity (2005). 2013;37(5):725-31. DOI: 10.1038/ijo.2012.103
50. Almoosawi S, Prynne CJ, Hardy R, Stephen AM. Diurnal eating rhythms: association with long-term development of diabetes in the 1946 British birth cohort. Nutrition, metabolism, and cardiovascular diseases : NMCD. 2013;23(10):1025-30. DOI: 10.1016/j.numecd.2013.01.003
51. Story M, Kaphingst KM, Robinson-O'Brien R, Glanz K. Creating healthy food and eating environments: policy and environmental approaches. Annual review of public health. 2008;29:253-72. DOI: 10.1146/annurev.publhealth.29.020907.090926
52. Sproesser G, Ruby MB, Arbit N, Akotia CS, Alvarenga MdS, Bhangaokar R, et al. Understanding traditional and modern eating: the TEP10 framework. BMC Public Health. 2019;19(1):1606.
53. Kobayashi F, Ogata H, Omi N, Nagasaka S, Yamaguchi S, Hibi M, et al. Effect of breakfast skipping on diurnal variation of energy metabolism and blood glucose. Obesity research & clinical practice. 2014;8(3):e249-e57. DOI: 10.1016/j.orcp.2013.01.001
54. Nas A, Mirza N, Hägele F, Kahlhöfer J, Keller J, Rising R, et al. Impact of breakfast skipping compared with dinner skipping on regulation of energy balance and metabolic risk. 2017;105(6):1351-61. DOI: 10.3945/ajcn.116.151332
55. Kim DY, Kim S, Lim HJJoHN, Dietetics. Association between dietary carbohydrate quality and the prevalence of obesity and hypertension. 2018;31(5):587-96. DOI: 10.1111/jhn.12559
56. de Mello Fontanelli M, Sales CH, Carioca AAF, Marchioni DM, Fisberg RMJEjon. The relationship between carbohydrate quality and the prevalence of metabolic syndrome: challenges of glycemic index and glycemic load. 2018;57(3):1197-205. DOI: 10.1007/s00394-017-1402-6
57. Suara SB, Siassi F, Saaka M, Foroshani AR, Sotoudeh GJBo. Association between Carbohydrate Quality Index and general and abdominal obesity in women: a cross-sectional study from Ghana. 2019;9(12). DOI: 10.1136/bmjopen-2019-033038
58. Mogre V, Nyaba R, Aleyira S, Sam NBJS. Demographic, dietary and physical activity predictors of general and abdominal obesity among university students: a cross-sectional study. 2015;4(1):226. DOI: 10.1186/s40064-015-0999-2
59. Santiago S, Zazpe I, Bes-Rastrollo M, Sánchez-Tainta A, Sayón-Orea C, de la Fuente-Arrillaga C, et al. Carbohydrate quality, weight change and incident obesity in a Mediterranean cohort: the SUN Project. 2015;69(3):297-302. DOI: 10.1038/ejcn.2014.187
60. Reynolds A, Mann J, Cummings J, Winter N, Mete E, Te Morenga L. Carbohydrate quality and human health: a series of systematic reviews and meta-analyses. Lancet (London, England). 2019;393(10170):434-45. DOI: 10.1016/S0140-6736(18)31809-9
61. Liu S, Willett WC, Manson JE, Hu FB, Rosner B, Colditz GJTAjocn. Relation between changes in intakes of dietary fiber and grain products and changes in weight and development of obesity among middle-aged women. 2003;78(5):920-7. DOI: 10.1093/ajcn/78.5.920
62. Vega-López S, Ausman LM, Griffith JL, Lichtenstein AHJDc. Interindividual variability and intra-individual reproducibility of glycemic index values for commercial white bread. 2007;30(6):1412-7. DOI: 10.2337/dc06-1598
63. Owen B, Wolever TMJNr. Effect of fat on glycaemic responses in normal subjects: a dose-response study. 2003;23(10):1341-7.
64. Dehghan M, Mente A, Zhang X, Swaminathan S, Li W, Mohan V, et al. Associations of fats and carbohydrate intake with cardiovascular disease and mortality in 18 countries from five continents (PURE): a prospective cohort study. 2017;390(10107):2050-62. DOI: 10.1016/S0140-6736(17)32252-3
65. Nakamura Y, Okuda N, Okamura T, Kadota A, Miyagawa N, Hayakawa T, et al. Low-carbohydrate diets and cardiovascular and total mortality in Japanese: a 29-year follow-up of NIPPON DATA80. 2014;112(6):916-24. DOI: 10.1017/S0007114514001627
66. Augustin LS, Kendall CW, Jenkins DJ, Willett WC, Astrup A, Barclay AW, et al. Glycemic index, glycemic load and glycemic response: an International Scientific Consensus Summit from the International Carbohydrate Quality Consortium (ICQC). 2015;25(9):795-815. DOI: 10.1016/j.numecd.2015.05.005
67. Mohammadi Nasrabadi F, Omidvar N, Khoshfetrat M, Kolahdooz FJE-eMHJ. Household food insecurity in the Islamic Republic of Iran: a systematic review and meta-analysis. 2014;20(11):698-706.
68. Esmaillzadeh A, Azadbakht LJTJon. Legume consumption is inversely associated with serum concentrations of adhesion molecules and inflammatory biomarkers among Iranian women. 2012;142(2):334-9. DOI: 10.3945/jn.111.146167
69. Okarter N, Liu RHJCrifs, nutrition. Health benefits of whole grain phytochemicals. 2010;50(3):193-208. DOI: 10.1080/10408390802248734
70. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FBJNEJoM. Changes in diet and lifestyle and long-term weight gain in women and men. 2011;364(25):2392-404. DOI: 10.1056/NEJMoa1014296

71. Fogelholm M, Anderssen S, Gunnarsdottir I, Lahti-Koski MJF, research n. Dietary macronutrients and food consumption as determinants of long-term weight change in adult populations: a systematic literature review. 2012;56(1):19103. DOI: 10.3402/fnr.v56i0.19103
72. Hutfless S, Gudzune KA, Maruthur N, Wilson RF, Bleich SN, Lau BD, et al. Strategies to prevent weight gain in adults: a systematic review. 2013;45(6):e41-e51.
73. Du H, van der A DL, Boshuizen HC, Forouhi NG, Wareham NJ, Halkjær J, et al. Dietary fiber and subsequent changes in body weight and waist circumference in European men and women. 2010;91(2):329-36. DOI: 10.3945/ajcn.2009.28191
74. Schwingshackl L, Hoffmann GJN, Metabolism, Diseases C. Long-term effects of low glycemic index/load vs. high glycemic index/load diets on parameters of obesity and obesity-associated risks: a systematic review and meta-analysis. 2013;23(8):699-706. DOI: 10.1016/j.numecd.2013.04.008
75. Atkinson FS, Foster-Powell K, Brand-Miller JCJDc. International tables of glycemic index and glycemic load values: 2008. 2008;31(12):2281-3. doi: 10.2337/dc08-1239
76. Arumugam V, Lee J-S, Nowak JK, Pohle RJ, Nyrop JE, Leddy JJ, et al. A high-glycemic meal pattern elicited increased subjective appetite sensations in overweight and obese women. 2008;50(2-3):215-22.
77. Esfahani A, Wong JM, Mirrahimi A, Srichaikul K, Jenkins DJ, Kendall CWJJotACoN. The glycemic index: physiological significance. 2009;28(sup4):439S-45S.
78. Burton-Freeman B, Keim NJIJoO. Glycemic index, cholecystokinin, satiety and disinhibition: is there an unappreciated paradox for overweight women? 2008;32(11):1647-54. DOI: 10.1038/ijo.2008.159
79. Goss AM, Goree LL, Ellis AC, Chandler-Laney PC, Casazza K, Lockhart ME, et al. Effects of diet macronutrient composition on body composition and fat distribution during weight maintenance and weight loss. 2013;21(6):1139-42. DOI: 10.1002/oby.20191
80. Stephen AM, Cummings JHJN. Mechanism of action of dietary fibre in the human colon. 1980;284(5753):283-4. DOI: 10.1038/284283a0

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Additionalfile1STROBE.docx
- SupplementaryFiles.docx