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Association of meal-specific protein intake and cardiometabolic risk factors: a cross-sectional study

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Abstract

We aimed to investigate the association of main meals' specific protein intake with cardiometabolic risk factors, including general and abdominal obesity, serum lipid profile, and blood pressure (BP). This cross-sectional study was conducted on 850 subjects aged 20–59 years. Dietary intakes were assessed by completing three 24-h recalls, and the protein intake of each meal was extracted. Anthropometric measures, lipid profile, fasting blood sugar and BP were measured. Multivariate logistic regression controlling for age, physical activity, sex, marital status, smoking status, BMI and energy intake was applied to obtain OR and CI. The mean age was 42 years, and the mean BMI of the participants was 27·2. The mean protein intake for breakfast, lunch and dinner was 12·5, 22·2 and 18·7 g/d, respectively. After adjustment for confounders, higher protein intake was not associated with any of the cardiometabolic risk factors, including LDL-cholesterol, HDL-cholesterol, total cholesterol (TC), TAG, body weight, BP and fasting plasma glucose, in any of the three main meals consumed within a day. Adherence to a higher protein intake at each meal was not associated with cardiometabolic risk factors in Iranian adults. Further prospective studies are needed to justify our findings.

Key words: **Protein: Meal: Cardiometabolism risk factors: Macronutrients**

CVD are the main cause of death all over the world, and 17·9 million people died from CVD in 2019[1]. It is expected that CVD would be the cause of more than 23 million (about 30·5 %) deaths by 2030 in the world[2]. More than 80 % of CVD deaths are due to heart attacks and strokes, and one-third of these deaths occur in people under 70 years old[3]. CVD resulted in 46 % of all deaths and 20–23 % of the disease burden in Iran[2]. CVD is a multicausal disorder that starts with cardiovascular risk factors and continues via progressive vascular disease to target organ damage, end-organ failure and death[4]. Modifiable risk factors of CVD such as elevated blood pressure (BP), abnormal lipid profile, diabetes mellitus, central obesity, tobacco use, stress, low consumption of fruits and vegetables, and physical inactivity are the main factors contributing to cardiovascular morbidity and mortality[5]. These risk factors can finally lead to elevated BP, elevated blood glucose, abnormal lipid profile, and overweight or obesity[3].

One of the most important therapeutic interventions is lifestyle modification. To reduce abdominal obesity, it is suggested to focus on physical activity and weight management[6]. Several dietary modifications can be taken to reduce the risk of CVD including dietary adjustments, for example reducing cholesterol intake to 100–120 mg/d, reducing caloric intake by 500–1000 calories per d to produce a weight loss of 0·5–1 kg per week, and recommending adherence to a Mediterranean diet guideline and the dietary approaches to stop hypertension (DASH) diet[7–10].

Some previous studies have shown a strong positive association between the protein content of the diet and mortality from CVD in different countries. A cohort study in Japanese populations suggested no association between total protein and the risk of stroke[11]. Also, a population-based cross-sectional study showed that higher dietary protein intake is independently associated with enhanced HDL-cholesterol levels, waist circumference and diastolic BP[12]. Based on the previous literature, the lowest and highest percentiles of protein intake ranges from 5·9 to 22·6 g at breakfast, 14·0 to 34·6 g at lunch and 24·3 to 46·8 g at dinner in adults[13].

Abbreviations: BP, blood pressure; TC, total cholesterol.

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There are many studies investigating the effects of dietary protein intake concerning cardiovascular risk factors, but data on the relation between meal-specific dietary protein and cardiovascular risk factors are scarce. Nutritional advice based on meals might be easier and more useful for people to understand and follow. Identifying and analysing meals allow us to understand how different combinations of foods and beverages at eating events could influence the overall diet quality and health outcomes[14,15]. Therefore, we aimed to examine the association between meal-specific protein intake and cardiovascular risk factors in a sample of Iranians.

Methods

Study population

This cross-sectional study was conducted on 850 healthy men and women, aged 20–59 years. Participants were recruited by multistage cluster random sampling method from health centres in five geographical areas (northern, southern, eastern, western and central) of Tehran, the capital city of Iran. Different health centres were chosen from each area, and then participants were selected based on inclusion criteria. The sample size of 546 was determined using this formula: $n = (pqz^2)/E^2$ considering where n = sample size; z^2 = square of the Accepted Article confidence level in standard error units (1·96); p = the estimate of the proportion of normal; q = 1-p, or the estimated proportion of obese people; and E^2 = the square of the maximum allowance for error between the true proportion and the sample proportion (0·04). The final sample size of 850 was selected for participation due to under- and over-reporting of total energy intake or other causes of exclusion. Subjects with CVD, cancer, diabetes and recent alternation in diet due to medical reasons were excluded. An informed consent form was signed by all participants before participation. The study was administered in accordance with the ethical committee of the Tehran University of Medical Sciences (Ethics Number: IR.TUMS.VCR.REC.1397.157).

Assessment of dietary protein intake

Dietary protein intake was obtained by utilising three 24-h recalls on each subject on non-consecutive days (two work days and one weekend). The first 24-h recall was collected by face-to-face interview; the other two were recorded using the telephone, and the meals and food groups were extracted. The 24-h recall was based on a standard five-step method designed by the US Department of Agriculture for use in national diet monitoring and administered by an experienced dietitian. The size of reported items was converted to grams per d by applying standard published guidelines, so we obtained the grams of items for each participant. Protein and nutrient intake were calculated using the Nutritionist IV programme.

Meal's definition

Based on previous studies, breakfast was defined as an eating occasion when a large amount of food or energy was consumed between 05.00 and 11.00, lunch was defined as an eating occasion when a large amount of food or energy was consumed between 11.00 and 16.00, and diner was defined as the main meal when a large amount of food or energy was consumed between 16.00 and 23.00[16].

Anthropometric assessment and blood pressure

Body weight, height, waist and hip circumference were measured according to standard guidelines. We used a stadiometer with a sensitivity of 0·1 cm (Seca to measure the height of patients, unshod, and a digital scale (808 Seca) with an accuracy of 0·1 kg was used to measure the weight with light clothing, and also obesity was determined by using BMI and waist circumference. BMI was calculated by dividing the weight in kilograms by the square of height in metres (kg/m^2), and < 18·5 was defined as low weight, 18·5–24·9 normal weight, 25–29·9 overweight, and ≥ 30 obese. Waist and hip circumference were measured using a tape measure, concerning standard protocol, and the waist-to-hip ratio was assessed for every individual, which, according to the NCEP ATP III is defined as follows: > 102 cm (> 40 in) for men or > 88 cm (> 35 in) for women. BP was assessed by a digital barometer (BC 08, Beurer) after 10 min of seated rest. Reported BP is the average of two BP measurements.

Laboratory investigations

Early in the morning, after 12 h of fasting, blood subjects were taken from the clients, and the fasting blood glucose, total cholesterol (TC), TAG, and HDL-cholesterol were measured shortly afterwards. Blood samples were measured by standard methods at the Nutrition and Biochemistry Laboratory of the School of Nutritional Sciences and Dietetics at Tehran University of Medical Sciences. Fasting blood glucose was assayed by the enzymatic (glucose oxidase) colorimetric method using commercial kit (Pars Azmoon). TC and HDL-cholesterol were measured using a cholesterol oxidase phenol amino antipyrine method, and TAG was measured using a glycerol-3 phosphate oxidase phenol amino antipyrine enzymatic method. All these tests were done by commercial kits (all from Pars Azmoon, Iran) using an auto-analyzer system (Selectra E, Vitalab).

Cardiometabolic risk factors

Waist circumference over 90 cm is considered abdominal obesity[17], BMI > 30 kg/m^2 as general obesity, BP ≥ 130/≥ 85 mmHg as hypertension, TAG level ≥ 150 mg/dl as hypertriacylglycerolaemia, HDL-cholesterol level less than 40 mg/dl (men) or 50 mg/dl (women) as low HDL-cholesterol, TC level ≥ 200 mg/dl as hypercholesterolemia, LDL-cholesterol level ≥ 130 mg/dl as elevated LDL-cholesterol, and fasting blood glucose ≥110 mg/dl as hyperglycaemia[6,18].

Physical activity

Physical activity level was estimated by using the International Physical Activity Questionnaire (IPAQ) consisting of physical activity related to occupation, housekeeping, transportation, sports and leisure, recreation, and sitting activities[19]. Patients were asked to recall all their moderate and intense activities in the past week, along with the time taken to complete them. This

information was used to determine MET h/d, and according to this, and subjects were divided into low, moderate, and high groups.

Covariates

A demographic questionnaire consisting of included age (continuous variable), sex (male or female), CVD (yes or no), diabetes (yes or no), smoking status (current, former or never smoking) and marital status (single or married) was used to gather and record general information of the subjects.

Statistical analysis

Statistical analysis of data was performed using SPSS Statistics software and reported as mean ± standard deviation or percentage. Participants were categorised based on tertiles of meal-specific protein intake. Regarding the type of variables, the means of quantitative variables were compared between the tertiles using one-way ANOVA and comparison of the distribution of qualitative variables between the tertiles using the chi-square test. Multivariate-adjusted logistic regression was performed to examine the relationship between protein intake, as an independent variable with obesity, and metabolic syndrome as a dependent variable controlled for age, physical activity, sex, marital status, smoking status, fat, carbohydrate percent and energy intake. P-values less than 0·05 was defined, a priori, to indicate statistically significant.

Results

The characteristics of participants by tertiles of dietary protein intake are shown in Table 1. There were no significant statistical differences in mean age across tertiles of breakfast ($P = 0·08$), lunch ($P = 0·16$) and dinner protein intake ($P = 0·54$). Distribution of education, sex, marital status, smoking and physical activity score were not significantly different across tertiles of meal-specific protein intake. Mean energy intake was significantly different between the tertiles of breakfast protein intake ($P < 0·001$). Participants in the first tertile of lunch-specific protein intake had a significantly higher percentage of energy from carbohydrates ($P < 0·001$) and a lower percentage of energy from protein ($P < 0·001$) and fats ($P = 0·01$). Furthermore, mean meal energy was significantly higher in the highest tertile of the lunch protein intake ($P < 0·001$). Those in the first tertile of dinner-specific protein intake had a significantly higher percentage of meal energy from carbohydrates ($P < 0·001$) and a lower percentage of energy from protein ($P < 0·001$).

Multivariate-adjusted means of anthropometric measures and biochemical variables across tertiles of meal-specific protein intake are provided in Table 2. After controlling for age, sex, education, marriage, smoking, physical activity, total energy intake and BMI (except for weight and BMI), we found that people at the top tertile of protein intake at lunch had lower levels of TC ($P = 0·02$) and LDL-cholesterol ($P = 0·04$) compared with those at the first tertile.

Multivariate-adjusted OR for cardiometabolic risk factors across tertiles of the protein intake for each main meal are presented in Table 3. Having the highest consumption of protein compared with the lowest protein intake was not significantly associated with the odds of abdominal obesity in breakfast (OR 0·83, 95 % CI 0·48, 1·53, $P = 0·54$) and lunch (OR 0·95, 95 % CI 0·57, 1·59, $P = 0·79$), and dinner (OR 1·09, 95 % CI 0·62, 1·89, $P = 0·80$) after adjusting for potential confounders. Also, having a higher intake of protein compared with the lowest intake did not change the BP level in breakfast (OR 0·81, 95 % CI 0·49, 1·86, $P = 0·42$), lunch (OR 1·67, 95 % CI 0·95, 2·93, $P = 0·08$) and dinner (OR 0·91, 95 % CI 0·50, 1·63, $P = 0·74$). Having a higher intake of protein in comparison with the lower tertile of intake was not significantly associated with plasma glucose after adjustment for breakfast (OR 1·21, 95 % CI 0·77, 1·90, $P = 0·41$), lunch (OR 0·97, 95 % CI 0·60, 1·59, $P = 0·93$) and dinner (OR 0·94, 95 % CI 0·58, 1·53, $P = 0·80$). In addition, having the highest consumption of protein compared with the lowest intake was not associated with a significant change in odds of general obesity in breakfast (OR 0·79, 95 % CI 0·48, 1·29, $P = 0·36$), lunch (OR 1·17, 95 % CI 0·68, 2·03, $P = 0·58$) and dinner (OR 1·25, 95 % CI 0·77, 2·02, $P = 0·39$) after adjusting for potential confounders. Finally, the levels of HDL-cholesterol, LDL-cholesterol and TC did not change significantly based on the protein consumption in any of the three main meals.

Discussion

Our findings revealed that having the highest consumption of protein compared with the lowest intake at any main meal level was not associated with a significant change in any of the obesity-related disorders, after adjusting for potential confounders. Also, higher intake compared with the lower intake of meal-specific protein had no association with the odds of dyslipidemia and high blood glucose.

Our study did not reveal any association between general and abdominal obesity with meal-specific protein intake; only after adjusting for age, sex, education, marriage and smoking; there was a decrease in abdominal obesity in a group who consumed the highest protein at breakfast compared with the lowest protein intake. In accordance with the result of our study, Mott *et al.* by using the prospective data from the National Growth and Health Study (NGHS) and the Framingham Offspring Study (FOS), indicated that girls who consumed more morning protein had the highest total protein intake. Furthermore, it showed total protein is associated with lower levels of adiposity and higher levels of skeletal muscle mass during later adolescence. These findings may lead us to the conclusion that maybe the beneficial effects were due to the morning protein intake[20]. Another cross-sectional study was conducted among Iranians, and three major dietary patterns at the lunch meal were identified: 'Bread, grains, and fat' and 'Western' and 'Potato and eggs'. Participants at the top tertile of the lunch-specific 'Bread, grains, and fat' intake had a higher waist-to-hip ratio compared with those in the lowest tertile. There was not any other significant association[21]. Another cross-sectional study on a total of 840 Iranian adults with an age range of 20–65 years showed a significant association between a lunch-specific animal-based low-carbohydrate diet, collected via 24-h dietary recall, and general obesity after essential adjustments[22]. Aoyama *et al.* compared the effect

Table 1. Characteristics of participants by tertiles (T) of dietary protein intake*

	Breakfast										Lunch										Dinner									
	T1			T2			T3				T1			T2			T3				T1			T2			T3			
	%	Mean	SD	%	Mean	SD	%	Mean	SD	*P*	%	Mean	SD	%	Mean	SD	%	Mean	SD	*P*	%	Mean	SD	%	Mean	SD	%	Mean	SD	*P*
Participants	281			281			282				281			282			281				280			280			280			
Protein intake (g)		4·76	2·86		11·8	1·66		21·1	7·67	< 0·001		8·62	4·96		20·7	3·26		37·2	13·2	< 0·001		6·47	4·21		17·0	2·84		32·6	10·2	< 0·001
Age (years)		42·3	10·5		41·9	10·9		43·1	10·2	0·08		41·8	10·3		43·1	10·4		41·5	10·9	0·16		42·7	10·6		42·2	10·4		41·8	10·7	0·54
Meal energy (kcal)		227	155		435	173		630	232	< 0·001		525	259		523	253		528	258	0·98		258	179		503	195		733	304	< 0·001
Daily energy (kcal)		1600	393		1670	365		1758	374	< 0·001		1628	431		1710	357		1695	354	0·03		1562	373		1717	380		1746	372	< 0·001
Carbohydrate (% of energy)	55·8			61·9			39·3			0·63	56·9			49·2			43·4			< 0·001	64·8			54·6			50·3			< 0·001
Fat (% of energy)	39·0			26·9			45·8			0·48	31·2			34·1			34·2			0·01	32·2			29·1			29·6			0·32
Protein (% of energy)	5·2			11·2			14·9			0·30	11·9			16·7			22·4			< 0·001	13·0			16·3			20·1			< 0·001
Sex										0·42										< 0·001										0·13
Male %	33·3			29·3			37·4				24·5			30·6			44·9				27·6			32·4			40·0			
Female%	33·3			34·1			32·6				35·2			34·0			30·8				34·5			33·5			31·9			
Education %										0·77										0·25										0·70
Illiterate	37·5			26·8			35·7				26·8			41·1			32·1				37·5			28·6			33·9			
Sub-diploma	32·3			30·8			36·8				31·7			33·2			35·1				35·6			34·7			29·7			
Diploma	33·6			33·6			32·9				33·1			37·2			29·7				30·3			35·5			34·1			
Educated	32·9			35·9			31·2				35·8			28·3			35·8				33·9			31·2			34·9			
Marriage %										0·79										0·60										0·56
Single	31·0			38·9			30·1				29·6			30·4			40·0				27·2			32·5			40·4			
Married	33·6			32·9			33·5				34·0			34·2			31·8				34·6			33·4			32·0			
Divorced	33·3			25·0			41·7				25·0			25·0			50·0				33·3			41·7			25·0			
Widowed	34·2			26·3			39·5				34·2			31·6			34·2				28·9			31·6			39·5			
Smoking %										0·87										0·12										0·27
Not smoking	32·9			33·7			33·4				33·3			34·3			32·4				33·7			32·5			33·8			
Quit smoking	40·0			35·0			25·0				40·0			15·0			45·0				25·0			55·0			20·0			
Current smoking	39·3			28·6			32·1				28·6			21·4			50·0				29·6			40·7			29·6			
Activity score %										0·67										0·76										0·92
Low	34·2			33·1			32·7				32·3			33·0			34·8				33·6			33·6			32·9			
Moderate	31·4			35·1			33·5				34·1			35·0			31·0				32·7			32·4			34·9			
High	36·0			26·7			37·3				36·0			29·3			34·7				34·7			36·0			29·3			

*All values are means ± standard deviation.
P-values result from ANOVA for quantitative variables and χ^2 test for qualitative variables.

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Table 2. Multivariate-adjusted means of anthropometric measures and biochemical indicators across tertiles (T) of meal-specific protein intake*

	Breakfast pro intake							Lunch pro intake							Dinner pro intake						
	T1		T2		T3			T1		T2		T3			T1		T2		T3		
	Mean	SE	Mean	SE	Mean	SE	*P*	Mean	SE	Mean	SE	Mean	SE	*P*	Mean	SE	Mean	SE	Mean	SE	*P*
Weight (kg)	73·3	0·79	72·1	0·79	71·6	0·79	0·31	73·4	0·79	72·3	0·78	71·3	0·79	0·18	72·6	0·80	72·6	0·79	71·9	0·79	0·74
BMI (kg/m^2)	27·7	0·26	27·0	0·26	27·0	0·26	0·11	27·5	0·26	27·3	0·26	26·8	0·26	0·18	27·1	0·26	27·4	0·26	27·2	0·26	0·70
Waist circumference (cm)	89·1	0·49	89·1	0·49	89·2	0·49	0·97	88·8	0·49	89·1	0·49	89·4	0·49	0·65	89·3	0·49	88·9	0·48	89·0	0·49	0·81
Waist-to-height ratio	0·87	0·01	0·85	0·01	0·86	0·01	0·06	0·85	0·01	0·86	0·01	0·87	0·01	0·06	0·86	0·01	0·86	0·01	0·86	0·01	0·89
Fasting plasma glucose (mg/dl)	105	1·11	104	1·11	105	1·11	0·76	106	1·23	105	1·23	105	1·23	0·58	104	1·24	106	1·22	105	1·23	0·41
TAG (mg/dl)	148	4·21	143	4·18	143	4·21	0·66	148	4·19	140	4·18	146	4·19	0·41	143	4·24	143	4·18	147	4·20	0·72
Total cholesterol (mg/dl)	200	2·58	194	2·57	192	2·58	0·09	201	2·57	193	2·56	191	2·57	0·02	198	2·61	195	2·58	193	2·59	0·42
HDL-cholesterol(mg/dl)	49·4	0·57	49·7	0·57	50·3	0·57	0·54	50·0	0·57	50·0	0·57	49·3	0·57	0·56	50·0	0·58	49·1	0·57	50·3	0·57	0·28
LDL-cholesterol (mg/dl)	121	2·25	116	2·24	115	2·25	0·12	122	2·24	116	2·24	114	2·24	0·04	120	2·27	118	2·25	114	2·26	0·25

*All values are means ± SE.
P ANCOVA: adjusted for sex, age, education, marriage, smoking, activity score, total energy intake and BMI (except for weight and BMI)

of feeding a high-protein meal at breakfast or dinner in mice. It revealed that the skeletal muscle index and grip strength were higher in subjects who habitually consumed a high-protein breakfast than in those who had a high-protein dinner. They found higher expression levels of myogenic factors (Igf1, Myog and Myf5) and the autophagy marker (LC3B-II levels) in the overloaded muscles of mice fed a high-protein diet in the morning phase. The second part of this study was done on healthy older women divided into two groups consisting of breakfast protein group and dinner protein group with no significant difference regardless of their distribution of protein intake. The results showed that older women who ingested high protein at breakfast had a higher skeletal muscle index and grip strength than those with a high-protein meal at dinner[23]. The reason for this difference in the existing literature and our results might be since none of the discussed studies exclusively assessed the relation between meal-based protein intake and obesity-related outcomes.

In our finding, there was no relationship between consuming more protein in meals and less possibility of altered lipid profile. A cross-sectional study in Iran showed a significant association between a lunch-specific low-carbohydrate diet with lower HDL-cholesterol levels and a lunch-specific vegetable-based low-carbohydrate diet with elevated TAG concentration after confounders adjustment. There was not any other significant association[22]. Azizi *et al.* found that adults with the highest Food Quality Score and lowest meal frequency had higher levels of HDL-cholesterol. In addition, the joint association of meal frequency and snack frequency with diet quality showed a higher chance of having MetS. It offered that an increment in meal frequency could conceal the good effects of the diet quality. In this study, breakfast, lunch and dinner time did not associate with the prevalence of MetS[24]. Meng *et al.* assessed the relationship between dietary animal and plant protein intake and cardiometabolic risk factors. It showed that more consumption of animal protein might increase serum concentrations of TC, LDL-cholesterol, non-HDL-cholesterol, and uric acid, while a high intake of plant protein is associated with lower non-HDL-cholesterol and uric acid concentrations and LDL-cholesterol:HDL-cholesterol ratio. Furthermore, plant protein intake was positively associated with HDL-cholesterol and HbA1c concentrations. In total, high-protein intake is associated with a high level of TC, HDL-cholesterol and uric acid concentration[25]. In contrast to our finding, Analysis of the National Health and Nutrition Examination Survey (NHANES) 2013–2016 data presented that consuming protein at breakfast was positively associated with HDL-cholesterol[13]. A cross-over study by Davis *et al.* investigated the effect of different amounts of protein at two different times of eating on blood glucose concentrations and insulin. Healthy adults who consumed a high-protein meal at night had a lower peak glucose concentration compared with the standard test meal at night. Peak glucose concentrations were not significantly different between the same test meal types consumed at different times of the day[26]. These findings could be due to higher insulin secretion and improved insulin sensitivity which is affected by protein intake[26,27]. Leidy *et al.* has suggested that diets containing protein in the range of 1·2–1·6 g protein/kg/d and potentially including at least 25–30 g

Table 3. OR and 95 % CI for overweight, obesity and cardio metabolic risk factors among the tertiles (T) of the meal-specific protein intake

	Breakfast						Lunch						Dinner					
		T2		T3				T2		T3				T2		T3		
	T1	OR	95 % CI	OR	95 % CI	*P*	T1	OR	95 % CI	OR	95 % CI	*P*	T1	OR	95 % CI	OR	95 % CI	*P*
Abdominal obesity																		
Crude	1	0·66	0·47, 0·93	0·75	0·54, 1·05	0·10	1	1·25	0·90, 1·75	0·94	0·67, 1·32	0·73	1	1·17	0·84, 1·64	1·00	0·71, 1·40	0·99
Model 1	1	0·69	0·48, 0·98	0·69	0·49, 0·98	0·04	1	1·15	0·81, 1·63	0·89	0·62, 1·27	0·52	1	1·22	0·86, 1·73	1·02	0·71, 1·45	0·92
Model 2	1	0·79	0·50, 1·26	0·83	0·48, 1·45	0·54	1	1·30	0·87, 1·95	0·95	0·57, 1·59	0·79	1	1·29	0·81, 2·06	1·09	0·62, 1·89	0·80
Elevated blood pressure																		
Crude	1	0·93	0·65, 1·34	0·86	0·60, 1·25	0·44	1	1·28	0·88, 1·85	1·37	0·95, 1·99	0·09	1	1·04	0·69, 1·57	0·94	0·62, 1·41	0·75
Model 1	1	1·01	0·68, 1·48	0·79	0·53, 1·17	0·24	1	1·17	0·79, 1·73	1·39	0·94, 2·06	0·10	1	1·10	0·71, 1·68	0·98	0·63, 1·51	0·91
Model 2	1	1·07	0·69, 1·65	0·81	0·49, 1·36	0·42	1	1·42	0·91, 2·23	1·67	0·95, 2·93	0·08	1	1·00	0·62, 1·63	0·91	0·50, 1·63	0·74
Elevated TAG																		
Crude	1	0·81	0·57, 1·14	0·75	0·53, 1·06	0·11	1	0·79	0·56, 1·11	1·00	0·71, 1·40	0·99	1	1·19	0·84, 1·68	1·12	0·79, 1·58	0·54
Model 1	1	0·84	0·59, 1·19	0·72	0·51, 1·03	0·07	1	0·75	0·53, 1·07	0·95	0·67, 1·34	0·76	1	1·19	0·84, 1·69	1·10	0·77, 1·56	0·60
Model 2	1	0·94	0·64, 1·40	0·80	0·50, 1·29	0·36	1	0·75	0·50, 1·12	0·92	0·56, 1·52	0·81	1	1·18	0·79, 1·76	1·06	0·66, 1·70	0·83
Elevated fasting plasma glucose																		
Crude	1	0·89	0·64, 1·24	1·04	0·74, 1·44	0·83	1	0·92	0·66, 1·29	0·93	0·67, 1·30	0·67	1	1·29	0·93, 1·80	0·89	0·64, 1·24	0·50
Model 1	1	0·93	0·66, 1·31	1·01	0·72, 1·41	0·97	1	0·87	0·62, 1·22	0·95	0·68, 1·34	0·78	1	1·34	0·95, 1·88	0·93	0·66, 1·30	0·67
Model 2	1	1·09	0·74, 1·59	1·21	0·77, 1·90	0·41	1	0·95	0·64, 1·41	0·97	0·60, 1·59	0·93	1	1·29	0·87, 1·90	0·94	0·58, 1·53	0·80
Low HDL-cholesterol																		
Crude	1	0·88	0·63, 1·22	0·85	0·61, 1·18	0·33	1	0·86	0·62, 1·20	1·24	0·89, 1·73	0·20	1	1·14	0·82, 1·59	1·04	0·75, 1·46	0·80
Model 1	1	0·90	0·64, 1·26	0·83	0·59, 1·16	0·28	1	0·91	0·65, 1·27	1·35	0·96, 1·90	0·09	1	1·14	0·81, 1·60	1·09	0·78, 1·53	0·60
Model 2	1	0·86	0·59, 1·25	0·72	0·45, 1·13	0·15	1	0·82	0·55, 1·21	1·20	0·72, 1·99	0·44	1	1·12	0·77, 1·63	1·09	0·70, 1·70	0·70
General obesity																		
Crude	1	0·72	0·50, 1·02	0·85	0·60, 1·22	0·39	1	0·99	0·70, 1·40	0·89	0·63, 1·27	0·53	1	1·37	0·97, 1·95	1·15	0·81, 1·62	0·42
Model 1	1	0·75	0·52, 1·08	0·81	0·56, 1·16	0·25	1	0·93	0·65, 1·33	0·90	0·63, 1·30	0·58	1	1·44	1·01, 2·08	1·22	0·85, 1·74	0·27
Model 2	1	0·74	0·49, 1·11	0·79	0·48, 1·29	0·36	1	1·19	0·78, 1·81	1·17	0·68, 2·03	0·58	1	1·44	0·96, 2·17	1·25	0·77, 2·02	0·39
Elevated LDL-cholesterol																		
Crude	1	0·66	0·46, 0·94	0·70	0·49, 0·99	0·04	1	0·80	0·56, 1·13	0·62	0·43, 0·89	0·01	1	0·91	0·64, 1·29	0·58	0·40, 0·84	< 0·001
Model 1	1	0·65	0·45, 0·93	0·67	0·47, 0·97	0·03	1	0·77	0·54, 1·09	0·63	0·44, 0·92	0·01	1	0·93	0·65, 1·33	0·60	0·41, 0·86	< 0·001
Model 2	1	0·79	0·52, 1·19	0·90	0·55, 1·48	0·68	1	0·87	0·57, 1·34	0·75	0·42, 1·34	0·33	1	1·01	0·67, 1·52	0·64	0·38, 1·07	0·09
Elevated cholesterol																		
Crude	1	0·68	0·49, 0·56	0·80	0·57, 1·12	0·19	1	0·62	0·44, 0·86	0·62	0·44, 0·87	< 0·001	1	0·93	0·67, 1·30	0·68	0·49, 0·95	0·03
Model 1	1	0·67	0·47, 0·95	0·77	0·54, 1·08	0·13	1	0·58	0·41, 0·82	0·63	0·45, 0·89	< 0·001	1	0·96	0·68, 1·35	0·70	0·49, 0·99	0·04
Model 2	1	0·84	0·57, 1·23	1·04	0·65, 1·65	0·87	1	0·61	0·40, 0·92	0·60	0·35, 1·03	0·07	1	1·11	0·75, 1·65	0·82	0·51, 1·34	0·43

Model 1: age, sex, education, marriage, smoking; model 2: model 1 + physical activity, total energy intake, carbohydrate and fat percentage, and BMI (except for general obesity).
Abdominal obesity was defined as waist circumference ≥ 90 cm.

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protein/meal provide improvements in body weight management, cardiometabolic risk factors and appetite[28].

We did not find any association between meal-specific protein intake and hypertension. Similar to our results, Bergia *et al.* in a randomised, double-blind, cross-over acute feeding study on thirty-one pre-hypertensive participants concluded that consuming a high-protein, a low-fat meal does not influence BP[29]. In contrast to our null finding, protein consumption at breakfast was inversely associated with systolic and diastolic BP, based on a cross-sectional study by Berryman et al, conducted on 10 112 adults above 19 years of age, located in NHANES 2013–2016 data[13]. The overall meat intake, one of the richest sources of daily protein, is very low in the Iranian society due to mainly financial reasons. This contributes heavily to the low overall intake of protein at lunch and dinner in our study sample which may interpret the lack of significant association in our results. Since total meat is rarely consumed at breakfast, the amount of protein consumed at breakfast is very similar between our data and previous literature.

The strengths of the current observational study include the relatively large representative sample size, being the first study that evaluates the meal-specific protein intake, the inclusion of a large number of covariates and assessing the main cardiometabolic risk factor. There are some limitations in the current study containing cross-sectional design and any remaining residual confounding like chronotype and sleeping time. We also did not investigate the different sources of protein. Furthermore, we used experienced nutritionists for anthropometric measurement but still not just for anthropometrics but also for laboratory measurement, and there might be measurement errors. Although we adjusted for the age, the large range of ages in our sample data might impact the results of our study since different age groups have different dietary needs for protein and the protein intake in ages over 50 years might have a significant influence. Although due to the high percentage of the participants being between 35 and 45 years of age, it was quite inefficient to categorise based on the age groups in our study. The combination of these limitations highlights the necessity for more investigation into different populations.

Conclusions

In conclusion, we did not find any association between meal protein and cardiometabolic risk factors at meal levels. Further prospective studies are needed to confirm our findings.

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There are no conflicts of interest.

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