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Original article

Nutritional factors and metabolic variables in relation to the risk of coronary heart disease: A case control study in Armenian adults

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ABSTRACT

Introduction: Dietary factors can affect the coronary heart disease (CHD). Results of previous studies on the association between the diet and CHD are not consistent in different countries. There were no data on this association in Armenia.
Objective: Aims of this case-control study were to evaluate the association between nutritional factors and CHD among Armenians in Yerevan.
Methods: During 2010 and 2011, we randomly selected 320 CHD patients with a diagnosis of CHD less than 6 months and 320 subjects without CHD (≥30 years old) from the hospitals and polyclinics in Yerevan. Dietary intakes with 135 food items over the previous 12 months were evaluated using a semi-quantitative food frequency questionnaire.
Results: After adjusting for some CHD risk factors higher intakes of polyunsaturated fatty acids (PUFA) and monounsaturated fatty acids (MUFA) were associated with a reduced risk of CHD, while this association was not witnessed for saturated fatty acids (SFA). In addition, findings indicated an inverse relation between vitamins (E, B6 and B12, folic acid) and fiber with CHD. In this population, smoking, hypertension, and metabolic syndrome (MetS) were significantly more common among patients with CHD.
Conclusion: The intake of vitamins E, B6 and B12, folic acid, PUFA, MUFA and fiber appeared to be predictors of CHD, independently of other risk factors.

1. Introduction

Dietary intakes are recognized to play prominent roles in the prevention and treatment of coronary heart disease (CHD) [1]. Awareness of dietary changes as a strategy is the most useful for both primary and secondary prevention of coronary risks [2]. The study of the relationship between dietary factors and CHD has been focused largely for almost half a century [3]. In prospective cohort studies dietary risk factors such as high saturated fatty acids (SFAs) [4–6], trans-fatty acid [7], low in marine omega-3 fatty acids, fiber, legume [8], vegetable and fruit have been well-established [3,9]. In addition, during the past decade, in numerous studies dietary factors have been associated with CHD risks such as the blood pressure [10,11], waist-to-hip ratio [11], dyslipidemia [12], and metabolic risk factor [13,14]. Despite decades of interesting researches, the association between dietary fat, particularly, its quantity and quality, and the risk of CHD is a subject of debate. In line with the same argument, prospective cohort studies have investigated the association between the CHD incidence and intake of total dietary fat with discrepant findings [4,15].

Evidence that the dietary intake of B vitamins is a protective risk factor for cardiovascular disease (CVD) remains limited. The Japan Public Health Center-based Prospective Study indicated that CHD was inversely associated with the dietary intake of folic acid, vitamin B6 and vitamin B12 after adjustment for the age and sex [16].

Epidemiological studies have not been entirely consistent with regard to the relationship between the intake of antioxidant vitamins and CVD [17]. Also, the preventive effect of antioxidant supplementation on cardiovascular events in humans is unproven [16,18,19]. However; few studies have examined the potential effect of the mixture of these vitamins [16,20]. The combination of multiple dietary factors is more powerful than a single factor [21].

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Recently, in a reviewed study from large cohort studies in Japan, investigators have reported that adequate control of total energy with restriction of SFAs from animal foods, increased intake of *n*-3 polyunsaturated fatty acids (PUFAs) including fish, soybean products, fruit and vegetable together with the low salt intake are responsible for promoting CHD prevention [2]. To the best of our knowledge, no previous study has looked at the association between dietary intake of nutrients and metabolic variable with CHD in the Armenia Adults. The objective of this study was to investigate nutritional factors and the metabolic variable in relation to the risk of CHD in this population.

2. Materials and methods

2.1. Participants

This observational case-control study was conducted from March 2010 to February 2011 in the Yerevan State Medical University (YSMU) hospitals and polyclinics, Patients aged ≥ 30 years as the case group ($n = 320$) with established CHD identified by cardiologists and for control ($n = 320$) were individuals aged ≥ 30 years without CHD who attended for check-up in hospitals and polyclinics in Yerevan. Subjects with previous history of myocardial infraction (MI), admission for angiography, heart surgery or angioplasty for CHD, pregnant women, and patients with history of systemic diseases according to the medical records were excluded. The study protocol was approved by the Bioethics Committee of the YSMU after M.Heratsi.

2.2. Data collection

In this study, an informed consent was obtained from all study subjects. Research assistants collected data on family history of diabetes, heart diseases, hypertension, socioeconomic status, lifestyle factors (including smoking habits, physical activity, and alcohol drinking) and the dietary intake for each subject. Next, weight was measured by using digital scales while subjects were wearing light clothing and no shoes. Height was measured with a tape measure while the subjects were in a standing position (without shoes) and the shoulders were in a normal position. Measurements were recorded to the nearest 100 g and 0.1 cm for weight and height respectively. Waist circumference was measured while subjects were standing with a soft tape midway between the lowest rib and the iliac and it was not wrapped too tight or too loose. Then systolic and diastolic blood pressure was measured twice with a standard mercury sphygmomanometer after the participants sat for 15 min; the means of the two measurements were considered to be the participant's blood pressure at the time of health check-up.

2.3. Assessment of the dietary intake

Information on the usual intakes of foods and dishes over the previous year was obtained using a semi-quantitative food frequency questionnaire (FFQ). Nutritionists and public health specialists assisted in determining constructing a list of foods which ultimately consisted of approximately 135 foods and beverage items with a standard serving size that was commonly consumed by Armenians. Before the FFQ was implemented in the study, it was adapted to Armenian conditions and was field-tested on 50 individuals. Subjects were asked to select their frequency of consumption and amount of each food item during the previous 12 months by using household measures. For each subject, a mean intake according to grams per day of each food was calculated. Then, total energy and nutrients based on daily averages in both groups were calculated by Food Processor Software, Ver. 12. It is worth mention that vitamin and mineral supplements were not included in computing nutrient intakes.

2.4. Statistical analysis

The data collected through the questionnaire, clinical examinations, laboratory findings, and dietary intakes were entered into the database. All data were statistically analyzed using the SPSS, version 15. The qualitative data were compared between cases and controls by using the Chi Square test. Comparisons between the two continuous variables were made using an independent sample *t*-test. The relation between intakes of nutrients and the CHD risk was calculated by the univarite and multiple logistic regression, with further control for potential risk factors including hypertension, metabolic syndrome (MetS), the family history of CHD, physical activity status, smoking habits, waist circumference, alcohol consumption, and education status of the participants. All tests were 2-tailed and $P < 0.05$ was considered significant.

3. Results

The mean and percentage values of various cofactors among the cases and controls are shown in Table 1. The mean ± SD of age were 57.22 ± 10.9 and 55.5 ± 11.7 years in case and control groups respectively. Although patients in the case group were slightly older, the statistical analysis did not reveal any significant differences in age between groups. In our study, the percentage of current smokers, current alcohol consumers, the family history of CHD and MetS, using National Cholesterol Education Program-Third Adult Treatment Panel [NCEP-ATP (III)] definition, modified by American Heart Association/National Heart, Lung, and Blood Institute (AHA/NHLBI) [22], was significantly higher. Also, the cases had significantly higher means of waist girth (cm) than did

Table 1
Demography and clinical characteristics of participants with CHD and controls.

Variable	Cases N = 320)	Controls (N = 320)	
Age (y) Mean (SD)	57.22 (10.9)	55.50 (11.7)	P > 0.05
Male Sex N (%)	162 (50.6)	141 (44.1)	P > 0.05
Waist Girth (cm) Mean (SD)	102.19 (13.2)	98.53 (13.55)	P < 0.001
BMI (Kg/m2) Mean (SD)	29.52 (5.50)	29.38 (5.51)	P > 0.05
Obese N (%)	137 (42.8)	133 (41.6)	P > 0.05
Current Smokers N (%)	128 (40)	67 (20.9)	P < 0.0001
Current Alcohol Consumers N (%)	175 (54.7)	106 (33.1)	P < 0.0001
Family History of CHD N (%)	45 (14.1)	10 (3.1)	P < 0.0001
MetS N (%)	255 (79.6)	222 (69.4)	P < 0.005
Hypertension N (%)	250 (78.1)	206 (64.4)	P < 0.0001

Table 2
Total daily calorie, fat and vitamins intake in patients with CHD and controls.

Nutrients (daily intake)	Cases mean (±SD)	Controls mean (±SD)	P value
Total energy (kcal)	1515.9 (269)	1574 (305.1)	P = 0.010
Carbohydrate (g)	150.6 (36.4)	150.5 (29.8)	P = 0.977
(% of total energy)	39.7 (9.6)	38.2 (7.5)	
Protein (g)	72.8 (19.4)	76.8 (25)	P = 0.026
(% of total energy)	19.2 (5.1)	19.5 (6.3)	
Total fat (g)	69.1 (11.4)	73.9 (16.3)	P = 0.000
(% of total energy)	41. (6.7)	42.2 (9.3)	
PUFA (g)	13.1 (4.5)	15.1 (5.6)	P = 0.000
(% of total energy)	7.79 (2.7)	8.65 (3.2)	
MUFA (g)	21.7 (5.1)	22.9 (6.4)	P = 0.013
(% of total energy)	12.9 (3)	13.1 (3.7)	P = 0.500
SFA(g)	30.1 (8)	30.7 (8.8)	P = 0.329
(% of total energy)	17.9 (4.7)	17.6 (5)	
Cholesterol (g)	358.8 (89.6)	343.8 (100.1)	P = 0.047
Dietary Fiber (g)	13.7 (2.8)	14.6 (3.6)	P = 0.000
Folic acid without supplements (mcg)	197.2 (40.6)	204.9 (49.2)	P = 0.031
Vitamin E without supplements (mg)	9.9 (2.8)	11.7 (5.1)	P = 0.000
Vitamin A without supplements (RE)	1252 (204)	1178 (193)	P = 0.000
Vitamin C without supplements (mg)	61.8 (18)	60.2 (16)	P = 0.256
Vitamin B6 (mg)	1.3 (0.24)	1.4 (0.37)	P = 0.000
Vitamin B12 (mcg)	5.5 (2.1)	5.8 (2.8)	P = 0.120

the control subjects while there was not a significant difference between obesity and the body mass index (BMI) in both groups.

The means of daily calorie, fat, and vitamins intakes among the cases and controls are presented in Table 2. In this study, the cases had significantly lower total calorie, protein, fat, PUFA, monounsaturated fatty acids (MUFAs), fiber, vitamins E, B6, and folic acid intakes. The mean daily intakes of carbohydrate, SFAs, vitamin C, and vitamin B12 did not differ significantly between the both groups. Although the mean energy intake was lower, the cholesterol intake was higher in the patients with CHD than that in the controls (358.8 g/d vs. 343.8 g/d; $p < 0.05$). We reported a significant difference based on the mean percentages of energy from PUFAs in cases and controls (7.79% vs. 8.65%, respectively; $p < 0.001$). While there was not a significant difference in the percent of energy from SFAs and MUFAs between both groups either those who had CHD or those who did not.

The odds ratio (OR) and 95% CI of the highest and lowest quartile of the nutrient intake associated with CHD are reported in Table 3. In the univariate logistic regression analysis, results showed that dietary intakes of total fat, PUFA, fiber, and vitamins E were inversely associated with the risk of CHD. The OR (95% CI) the in highest vs. lowest quartile of total fat intake was 0.51(0.33–0.80) for CHD; for the highest vs. lowest quartile of PUFA intake was 0.22 (0.14–0.35). Also, for the highest vs. lowest quartile of fiber, and vitamins E intakes were 0.50(0.32–0.78) and 0.21(0.13–0.35), respectively. In contrast, participants in the highest quartile of cholesterol and vitamin A had the OR of 2.55 (95% CI: 1.62, 3.99); 6.05 (95% CI: 3.71, 9.87), respectively. There were no statistically significant interactions for CHD between quartiles of dietary SFAs, MUFAs, and vitamin C intakes.

Further control was made for hypertension, MetS, family history of CHD, physical activity status, smoking habits, waist circumference, alcohol consumption, and education status to investigate the relationship between some nutritional factors and the CHD. In the multiple logistic regressions, the output indicated that the PUFAs, MUFAs, fiber, vitamins E, B6, B12, and folic acid were significant predictors for CHD. There was not a significant relationship between cholesterol and vitamin C with CHD (Table 4). The odds ratios were estimated after taking into account the effect of hypertension, MetS, and family history of CHD, physical activity status, smoking habits, waist circumference, alcohol consumption, and education status of the participants.

4. Discussion

To our knowledge, this is the first study to assess the association between nutritional factors and metabolic variables with CHD in Armenia. In this case-control study, the prevalence of MetS and high blood pressure (HBP) was high in both groups, the specific sample of initially selected population who referred to the hospitals with HBP and MetS components.

The odds ratio indicated that the risk of CHD decreased by about 11% for each additional gram per day of fiber (Table 4). 30.2% of the cases and 19.9% of the controls were in the lowest quartile of fiber consumption (OR: 0.5; 95% CI: 0.32, 0.78) (Table 3). Consistently, a study has shown that dietary fiber intake was inversely associated with several CVD risk factors [11].

In the present study, it was observed that both cases and controls had a high intake of cholesterol (358.8 g/d and 343.8 g/d, respectively) (Table 2). Although the intake of cholesterol after adjusting for other risk factors, was not associated with CHD (Table 4; $p > 0.05$), the odds ratio of subjects with cholesterol consumption in the highest quartile, compared to the lowest, was 2.55 (95% CI: 1.62, 3.99) (Table 3). We also found that OR: 1.91 (95% CI: 1.33, 2.75) based on the cholesterol $\geq$ 300 mg/day 75% and 60.6% of the cases and controls had a cholesterol intake more than 300 mg/d (data not shown). A case-control study, in Indonesia, has reported a consistent OR: 4.7 (95% CI: 2.3, 9.7) based on a cholesterol intake in the highest quartile in comparison to the lowest [23].

We observed an inverse association between the PUFAs and MUFAs with CHD. Multiple logistic regressions indicated that for

Table 3
Distribution of cases and controls in the highest and lowest quartiles, the odds ratio (95% confidence intervals) according to their nutrients intakes.

Variable	Cases N (%)	Controls N (%)	Odds ratio	95% CI
Fat (g/day)				
1 st quartile (<61.6 g/d)	84 (25.9)	79 (24.4)	0.51	0.33–0.80
4th quartile (>79.5 g/d)	57 (17.6)	104 (32.1)		
PUFA (g/day)				
1 st quartile (<11.5 g/d)	106 (32.4)	61 (18.7	0.22	0.14–0.35
4th quartile (>15.6 g/d)	45 (13.8)	115 (35.2)		
MUFA (g/day)				
1 st quartile (<18.4 g/d)	83 (25.8)	78 (24.2)	0.7	0.45–1.08
4th quartile (>24.9 g/d)	69 (21.4)	92 (28.6)		
SFA (g/day)				
1 st quartile (<24.9 g/d)	77 (24)	84 (26.2)	0.84	0.54–1.31
4th quartile (>34.1 g/d)	70 (21.8)	90 (28)		
Cholesterol (mg/day)				
1 st quartile (<294 mg/d)	56 (17.3)	106 (32.7)	2.55	1.62–3.99
4th quartile (>409 mg/d)	93 (28.7)	69 (21.3)		
Fiber (g/day)				
1 st quartile (<11.8 g/d)	97 (30.2)	64 (19.9)	0.5	0.32–0.78
4th quartile (>15.8 g/d)	69 (21.5)	91 (28.3)		
Vitamin E (g/day)				
1 st quartile (<9.28 mcg/d)	96 (29.8)	64 (19.9)	0.21	0.13–0.35
4th quartile (>11.8 g/d)	40 (12.4)	122 (37.9)		
Vitamin A (RE/day)				
1 st quartile (<1092 RE/d)	37 (11.5)	124 (38.6)	6.05	3.71–9.87
4th quartile (>1361 RE/d)	103 (32.1)	57 (17.8)		
Vitamin C (mcg/day)				
1 st quartile (<49.6 mcg/d)	37 (11.5)	124 (38.6)	0.87	0.56–1.36
4th quartile (>71 mcg/d)	103 (32.1)	57 (17.8)		

every g PUFAs and MUFAs consumption, the risk may decrease by about 11% and 5%, respectively. Also the individuals in the highest quartile of consumption of PUFAs and MUFAs had about 78% and 30% lower CHD when compared to those in the lowest quartile respectively. This result is consistent with the finding, of previous prospective cohort studies [4,24–26]. In contrast, in the Spanish cohort study, with a relatively high intake of fish, no association was found between EPA, DHA and total omega-3 fatty acid intake and risk of CHD [27]. Likewise, Spain is a country with a high intake of fish [26] and a low incidence of CHD, which may explain, at least in part, the lack of association between total omega-3 fatty acid, EPA or DHA intakes and the risk of CHD in this study.

There was no association between the SFAs intake and incidence of CHD in our study. Although it was reported in a study that only long chains SFAs were associated with a small increase in the risk [24], the result was supported by other studies [4,23,28].

Table 4
Adjusted odds ratio of CHD calculated from multiple logistic regression models for the assessed of the effect of dietary factors on coronary risk.

Dietary factors	OR	95% CI		Wald	P-Value
		Lower	Upper		
Calorie	0.999	0.998	1.000	10.37	0.001
Protein	0.988	0.98	0.996	8.29	0.004
Total Fat	0.940	0.961	0.986	17.20	0.000
PUFA (g/day)	0.911	0.868	0.955	14.65	0.000
MUFA	0.955	0.927	0.984	8.851	0.003
Cholesterol (mg/day)	1.001	1.000	1.003	2.585	0.108
Fiber (g/day)	0.895	0.846	0.946	15.238	0.000
Vitamin E (mcg/day)	0.883	0.834	0.936	17.838	0.000
Folic acid (mcg/day)	0.995	0.991	0.999	6.262	0.012
Vitamin B6 (mg/day)	0.344	0.190	0.623	12.404	0.000
Vitamin B12 (mg/day)	0.927	0.860	0.999	3.898	0.048
Vitamin C (mg/day)	1.002	0.992	1.012	0.107	0.744

However, although the result showed a significant difference in the dietary fat intake between the case and control groups, a high total fat intake compared to the recommendations made by AHA among cases and controls was reported, 41.06% and 42.25% of total calories, respectively. The high fat dairy products, butter and local foods including Bozbash, Plav and Dolma, and fried foods were the major food source of the total fat intake in this population for both groups. The higher intake of total fat, SFAs, lower intake of MUFAs and PUFAs suggests a less healthy eating pattern among the Armenian population that may contribute to higher CHD.

Multiple logistic regressions indicated that for every mcg consumption of vitamin E, the CHD risk may decrease by about 14%. The individuals in the highest quartile of consumption of vitamin E had about 79% lower CHD when compared to those in the lowest quartile. In this study, unexpectedly, the benefit of other antioxidant vitamins including vitamins A and C for the CHD was not confirmed.

The vitamin A intake revealed that it had a positive association with CHD. In Saudi population, there was a similar association between dietary vitamin A and the coronary risk [17]. In an earlier study, the use of antioxidant supplements of vitamin E and beta-carotene did not have effects on the major coronary risk [18,19]. A significant inverse association with CHD was observed for folic acid and vitamin B6 while the significant inverse association with CHD was marginal for vitamin B12. In 2008, Ishihara et al. similarly reported that CHD was associated with folic acid, vitamins B6, and B12 [16,29]. An earlier study has shown that individuals in the highest fifth of the folic acid intake had a 55% reduction of coronary

events as compared to those in the lowest fifth. A high dietary intake of vitamin B6 had no significant association whereas the risk of CHD was significantly reduced in those who had a high B12 intake [20].

In a case control study, Mean plasma level of folate in patients with CAD was lower than control subjects [30]. In another study, Mean serum levels of Vitamins B12 and folate were lower in cases of coronary atherosclerosis than controls [31].

Serum vitamin B12 deficiency and hyperhomocysteinemia are related with cardiovascular risk factors in patients with CHD [32]. However, in a systematic review, results identified a valid association of a few nutrients including folic acid, vitamins E and C, fiber, and trans-fatty acids with CHD [3].

The current study had several limitations. First, because we used an FFQ to assess dietary intakes, the misclassification was a major concern. Furthermore, calories are frequently underreported when dietary data are collected by FFQ. The caloric intake may also get underreported due to social desirability, particularly by overweight people [33,34]. Second, it is possible that individuals who had a low intake of vitamins and fiber were at higher risk of CHD due to other unhealthy habits and behaviors. The likelihood of this was reduced, however, by the multivariable adjustment for a number of potential confounding variables including hypertension, MetS, family history of CHD, physical activity status, smoking habits, waist circumference, alcohol consumption, and education status. The final potential limitation was the design of the study as we had to assess MetS and its components including abdominal obesity, elevated blood glucose, dyslipidemia, and high blood pressure in case and control groups, affecting on food patterns. In order to eliminate selection bias, subjects with previous history of MI, admission for angiography, heart surgery or angioplasty for CHD, pregnant women, and patients with history of systemic diseases were excluded from the study. These exclusions may have reduced the influence of such conditions subjects' behavioral changes, especially food habits.

5. Conclusion

Results of this case-control study showed that intakes of vitamins E, B6, B12, folic acid, and fiber are inversely and independently associated with the CHD.Thus, this association suggests that diets rich in these nutrients prevent the risk of CHD. Prospective cohort studies related to dietary habits and their nutrients in Armenian population are recommended.

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