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Relationship Between the Risk of Coronary Heart Disease and Nutritional Status of Adult

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ABSTRACT

Cardiovascular diseases (CVD) have the highest prevalence among non-communicable diseases so prevention from CVD is very important. The aim of this study was to determine the risk of cardiovascular events in adults over the next 10 years with the Framingham Risk Score (FRS) and to evaluate the relationship between this risk and the nutritional patterns of individuals. This study was conducted with a total of 238 individuals (78 males, 160 females) aged between 30-64 years. Energy, micro and macronutrients intake levels of the participants according to FRS and gender were determined by taking a daily food consumption record with a 24-hour retrospective reminder method. Participants' ten-year coronary disease risk was assessed with FRS which was classified as low risk (<10%), moderate risk (10-20%), and high risk (>20%). While 44.9% of the men had moderate/high coronary disease risk, all of women had low risk. Energy, many macronutrients and micronutrients intake levels of men with low risk of coronary heart diseases (CHD) were higher than women. FRS values of all individuals participating in the research were related to energy, carbohydrate (g, %), protein (g), total fat (%), mono-unsaturated fatty acid/MUFA (g), poly-unsaturated fatty acid/PUFA (%), riboflavin, sodium, and zinc. Of these parameters, total fat and PUFA were negatively correlated, while the others were positively correlated. The parameter that most affected the FRS value was carbohydrate (g). These results clearly demonstrate the relationship between nutrition and CVD risk. Therefore, identifying individuals with medium/high FRS and taking nutritional initiatives are important in reducing the CVD prevalence and health costs.

INTRODUCTION

Chronic diseases that are rapidly increasing in prevalence are among the leading causes of mortality and morbidity. It has been reported that 40 million of 56 million deaths that occurred worldwide in 2015 were due to non-communicable diseases (NCDs), and 48% of these deaths occurred in low or middle-income countries. NCDs such as heart disease, stroke, cancer, chronic respiratory diseases, and diabetes are among the leading causes of mortality (WHO, 2021).

Unhealthy dietary habits, which are among the modifiable risk factors of cardiovascular diseases with high prevalence (CDC, 2019), affect many other risk factors that may affect cardiometabolic health such as heart disease and stroke and create significant economic burdens (Micha et al., 2017). Saturated fatty acid (SFA) and carbohydrate contents are high, fiber content is low nutrition; increases intravascular plaque regression, cardiovascular diseases (CVD) morbidity, and mortality. Consumption of high SFA increases LDL-C levels while decreasing HDL-C levels and thus adversely affects heart health (Heart Foundation, 2019). The fiber content of the diet is also an effective risk factor for CVD. It has been reported that dietary fiber plays an effective role in the protection of cardiometabolic health as it decreases high blood glucose and cholesterol levels, and shows anti-inflammatory and antihypertensive properties (Gunashekar et al., 2017). It is possible to reduce the risk of CVD and prevent its development by interventions for nutrition and other cardiovascular risk factors (Buttar et al., 2005). The aim of this study was to determine the risk of cardiovascular events in adults over the next 10 years with the Framingham Risk Score (FRS) and to evaluate the relationship between this risk and the nutritional patterns of individuals.

MATERIAL AND METHODS

Sample Size and Selection

This study was conducted in accordance with the Helsinki Declaration principles with a total of 238 individuals [78 males (32.8%) and 160 females (67.2%)] with a mean age of 48.5±9.80 years who applied to a Family Health Center. Individuals with systemic disorders such as CVD, SVD, complications of diabetes, or severe hepatic, renal or hematological diseases were not included in the study. The sample size of the study was calculated by using one-way variance analysis with 0.05 significance level and 0.80 power. Ethics Committee Approval was obtained in order to conduct the research from Ankara University Clinical Research Ethics Committee.

Data Collection and Evaluation

The food consumption status of the participants was determined by taking a daily food consumption record with a 24-hour dietary recall (24HR). Food and Food Photography Catalog was used for the accurate evaluation of the amount of food consumed (Rakıcıoğlu et al., 2009). Computer-Aided Nutrition Program (BeBis), Nutrition Information System was used to calculate the energy and nutrient intake obtained from the amount of food consumed. The data obtained were evaluated according to Dietary Reference Intakes (DRI). In this evaluation, energy and nutrient intake of individuals were classified as <67.0% "insufficient", 67.0%-133.0% "sufficient", >133.0% "over consumption" according to ±33.0% cut-off point. (National Research Council, 1986).

Evaluation of Risk of Coronary Heart Disease

FRS was used to determine the ten-year coronary disease risk of the participants. 10-year coronary event risks of individuals were calculated with the total score calculated according to values and categories (NIH, 2013). FRS was classified as low risk (<10%), moderate risk (10-20%), and high risk (>20%) (Kannel et al., 1976).

Statistical Evaluation of Data

The data were analyzed with the SPSS statistical package program. Descriptive statistics are shown as mean ± standard deviation (SD) for variables with normal distribution, and median and interquartile range (IQR) values for variables with the non-normal distribution. The relationship between two categorical variables was evaluated by the Chi-Square test. In the study, if normal distribution assumptions are provided independent t-test was used to determine whether there is a statistically significant difference between the

Table 1. Mean, SD, median and IQR values of energy and nutrient intake of individuals according to FRS classification (%)

	<10%					10-20%£	>20%£		
	Male (n:43)	Female (n:160)	Total (n:203)			Male (n:28)	Male (n:7)		
Energy and nutrients	$\bar{x}$±SD Median (IQR)	$\bar{x}$±SD Median (IQR)	$\bar{x}$±SD Median (IQR)	t/z	p	$\bar{x}$±SD Median (IQR)	$\bar{x}$±SD Median (IQR)	χ_e^2/F_e χ_t^2/F_t	p_e p_t
Energy (kcal)	2075.5±719.22	1589.8±471.09	1692.7±567.30	4.194	0.000[b*]	1967.1±569.34	1917.6±399.7	0.338 3.307	0.714[β] 0.038[β*]
Carbohydrate (g)	251.5±104.0	193.6±59.91	205.8±75.07	3.498	0.001[b*]	220.5 (171.4)	210.5 (43.4)	0.047 8.207	0.977[α] 0.017*[α]
Carbohydrate (%)	49.7±10.59	50.4±9.61	50.3±9.81	-0.404	0.686[b]	52.3±9.63	51.2±9.51	0.572 0.579	0.567[β] 0.561[β]
Protein (g)	61.7 (28.7)	48.1 (23.8)	50.9 (24.7)	-3.442	0.001[a*]	59.4 (27.3)	60.7 (45.9)	0.704 5.086	0.703[α] 0.079[α]
Protein (%)	12.0 (4.0)	13.0 (4.0)	13.0 (4.0)	-0.116	0.907[a]	13.0 (5.0)	13.0 (7.0)	0.659 0.648	0.719[α] 0.723[α]
Total fat (g)	80.8 (44.2)	59.7 (35.8)	66.4 (38.6)	-3.674	0.000[a*]	70.9 (42.5)	60.1 (44.5)	1.129 1.894	0.569[α] 0.388[α]
Total fat (%)	36.7±8.62	36.0±9.01	36.2±8.91	0.417	0.677[b]	34.6±9.77	34.0±8.52	0.601 0.564	0.551[β] 0.570[β]
SFA (%)	8.8 (5.3)	10.0 (4.6)	9.8 (4.7)	-0.848	0.396[a]	9.1 (5.0)	8.0 (7.7)	0.056 0.249	0.972[α] 0.883[α]
MUFA (%)	13.0±4.77	12.3±4.13	12.5±4.27	0.938	0.349[b]	11.6±2.82	12.1±2.87	1.037 0.542	0.360[β] 0.583[β]
PUFA (%)	11.9±5.20	11.3±5.47	11.4±5.41	0.699	0.486[b]	8.7 (7.7)	8.6 (8.0)	1.984 1.335	0.371[α] 0.513[α]
Omega-3 (g)	1.3 (1.1)	1.0 (0.8)	1.0 (1.0)	-2.439	0.015[a*]	0.9 (0.6)	0.9 (0.7)	4.117 0.359	0.128[α] 0.836[α]
Omega-6 (g)	23.0 (26.0)	17.0 (13.9)	18.1 (15.4)	-2.822	0.005[a*]	18.1 (22.8)	14.6 (15.7)	1.450 0.401	0.484[α] 0.818[α]
Cholesterol (mg)	209.5 (174.9)	144.0 (193.5)	152.9 (195.8)	-1.810	0.070[a]	123.8 (153.1)	118.0 (320.5)	1.680 0.114	0.432[α] 0.944[α]
Fiber (g)	25.2±12.56	22.5±8.78	23.0±9.73	1.354	0.182[b]	24.2±10.92	25.3±7.52	0.070 0.321	0.932[β] 0.726[β]

Note: £ Since there were no female individuals in this group, the table was not included. χ_e^2, F_e and p_e were used to evaluate men individuals; χ_t^2, F_t and p_t were used to evaluate all individuals in different risk groups. [a] Mann Whitney-U test; [b] Independent-t test [α] Kruskal Wallis test; [β] One Way ANOVA test was used. *p<0.05. SFA: saturated fatty acid, MUFA: mono-unsaturated fatty acid, PUFA: poly-unsaturated fatty acid.

qualitative variables, if not provided Mann-Whitney U test was used. The difference between the quantitative variables with normal distribution was determined by Pearson Correlation, and variables with non-normal distribution were determined by the Spearman Correlation Coefficient. Statistically significant difference situation between the categories of quantitative variables with ≥3 categories was evaluated with One Way ANOVA test if normal distribution assumptions were provided; if not Kruskal Wallis test was used. In all statistical tests, the confidence interval was accepted as 95.0% and it was evaluated at p<0.05 significance level.

RESULTS

Daily Energy and Nutrient Intakes of Individuals

Energy, carbohydrate, protein, total fat, omega-3, and omega-6 fatty acid intake levels of men with low risk of CHD were higher than women (p<0.05). When all the participants were compared according to FRS classification, there was a difference in energy and carbohydrate intake (p<0.05). As a result of the statistical evaluations, it was found that this difference was caused by the difference between low and medium-risk groups (Table 1).

Table 2. Mean, SD, median and IQR values of micronutrient intake according to FRS classification (%)

	<10%					10-20%[£]	>20%[£]		
	Male (n:43)	Female (n:160)	Total (n:203)			Male (n:28)	Male (n:7)		
Micronutrients	$\bar{x}$±SD Median (IQR)	$\bar{x}$±SD Median (IQR)	$\bar{x}$±SD Median (IQR)	t/z	p	$\bar{x}$±SD Median (IQR)	$\bar{x}$±SD Median (IQR)	χ_e^2/F_e χ_t^2/F_t	p_e p_t
Vitamin A (mcg)	683.9 (898.7)	628.7 (511.0)	632.4 (538.5)	-1.044	0.297[a]	585.3 (405.4)	506.8 (800.8)	1.686 1.080	0.430[α] 0.583[α]
Thiamine (mg)	0.7 (0.3)	0.6 (0.3)	0.6 (0.3)	-1.606	0.108[a]	0.7±0.28	0.8±0.23	0.135 3.001	0.874[β] 0.223[α]
Riboflavin (mg)	0.9 (0.8)	0.9 (0.4)	0.9 (0.4)	-2.418	0.016[a]*	1.0 (0.3)	1.0 (0.7)	0.055 4.092	0.055[α] 0.129[α]
Niacin (mg)	18.0 (10.8)	14.4 (7.8)	15.3 (9.0)	-3.401	0.001[a]*	18.0 (8.4)	17.7 (11.6)	0.400 4.034	0.819[α] 0.133[α]
Pyridoxine (mg)	1.1 (0.6)	1.0 (0.5)	1.0 (0.5)	-1.513	0.130[a]	1.1 (0.6)	1.2 (0.6)	1.468 2.277	0.480[α] 0.320[α]
Vitamin C (mg)	76.3 (84.7)	80.3 (85.4)	78.3 (85.5)	-0.626	0.531[a]	72.1 (68.7)	86.3 (82.3)	0.802 0.509	0.670[α] 0.775[α]
Vitamin E (mg)	23.6 (14.9)	16.1 (13.9)	17.9 (15.3)	-3.499	0.000[a]*	17.6 (21.6)	20.3 (8.2)	1.842 0.799	0.398[α] 0.671[α]
Sodium (mg)	4145.2 (2123.0)	3215.8 (1759.8)	3338.6 (1907.4)	-3.573	0.000[a]*	3949.3 (2045.8)	3576.5 (1818.6)	1.605 6.923	0.448[α] 0.031[a]*
Potassium (mg)	1940.8 (1147.9)	1960.4 (1064.6)	1955.0 (1131.9)	-0.420	0.675[a]	1810.6 (887.1)	2280.9 (748.9)	1.003 1.053	0.606[α] 0.591[α]
Calcium (mg)	584.4 (381.5)	530.5 (295.5)	539.9 (310.4)	-1.190	0.234[a]	563.1 (363.1)	561.9 (376.9)	0.064 0.837	0.968[α] 0.658[α]
Phosphorus (mg)	974.4 (627.1)	863.4 (428.6)	880.1 (438.0)	-2.348	0.019[a]*	919.2 (455.4)	1232.9 (402.6)	1.436 3.293	0.488[α] 0.193[α]
Magnesium (mg)	218.8 (147.3)	199.0 (117.4)	203.5 (119.6)	-1.357	0.175[a]	205.4 (116.9)	233.2 (177.5)	1.724 1.288	0.422[α] 0.525[α]
Zinc (mg)	8.8 (5.6)	6.8 (3.8)	7.2 (4.0)	-3.782	0.000[a]*	9.1±3.89	10.1±4.12	0.380 5.205	0.685[β] 0.074[α]

Note: [£] Since there were no female individuals in this group, the table was not included. χ_e^2, F_e and p_e were used to evaluate men individuals; χ_t^2, F_t and p_t were used to evaluate all individuals in different risk groups. [a] Mann Whitney-U test; [b] Independent-t test; [α] Kruskal Wallis test; [β]One Way ANOVA test was used. *p<0.05.

Vitamin E, riboflavin, niacin, sodium, phosphorus, and zinc intake of men with low risk of CHD were higher than female subjects (p<0.05). When all individuals included in the study were evaluated according to CHD risk status, there was a difference in the amount of sodium intake (p<0.05). As a result of statistical evaluations, it was found that this difference was caused by the difference between intake amounts of individuals with low and medium risk (Table 2).

According to the FRS classification and DRI of individuals participating in the research, their levels of meeting their daily energy and nutrients needs are given in Table 3. There was a statistically significant difference between the levels of fiber, thiamine, riboflavin, niacin, vitamin C, vitamin E, sodium, and calcium in men and women at low risk of CHD; omega-3 fatty acid, thiamine, riboflavin, niacin, and vitamin C requirements in all risk groups evaluated in terms of CHD (p<0.05). Since the meeting levels of phosphorus and potassium requirements are high by all individuals and therefore no analysis can be made (Table 3).

According to Table 4, FRS values of men at low risk for CHD are associated with omega-3 fatty acid consumption; FRS values of women are correlated with carbohydrate (%), total fat (%), SFA (%), PUFA (g, %) and omega-6 fatty acid consumption; FRS values of all individuals at low risk are related to energy, carbohydrate (g) and SFA (%) consumption (p<0.05). FRS values of men at medium risk for CHD have a significant correlation with total fat (g) (p<0.05). It was found that FRS values of all individuals participating in the research were related to energy, carbohydrate (g, %), protein (g), total fat (%), MUFA (g), and PUFA (%) (p<0.05) (Table 4).

It was found that FRS values of all individuals at low risk for CHD were associated with riboflavin and sodium; FRS values of individuals at medium risk were associated with niacin, pyridoxine, vitamin C, potassium, and magnesium. FRS values of all individuals participating in the study were found to be related to riboflavin, sodium, and zinc (p<0.05) (Table 5).

Table 3. Levels of individuals meeting their daily energy and nutrient requirements according to FRS classification (%) and DRI

Energy and nutrients	<%10				%10-20[£]	>%20[£]		
	Male (n:43)	Female (n:160)	Total (n:203)	χ^2 p	Male (n:28)	Male (n:7)	χ_e^2 χ_t^2	p_e[b] p_t[b]
	%	%	%		%	%		
Energy (kcal)								
Insufficient	18.6	29.4	27.1	4.952	7.2	-	5.477	0.215
Over consumption	18.6	4.4	7.4	0.084[a]	21.4	-	2.618	0.586
Carbohydrate (%)								
Insufficient	30.2	30.6	30.5	0.073	21.4	42.8	3.584	0.441
Over consumption	4.7	3.8	4.0	0.964[a]	10.7	14.4	6.001	0.157
Protein (%)								
Insufficient	23.3	19.4	20.2	1.909	28.6	14.4	2.132	0.885
Over consumption	2.3	0.6	1.0	0.344[b]	-	-	2.630	0.711
Total fat (%)								
Insufficient	2.3	3.8	3.4	0.207	7.1	-	3.560	0.463
Over consumption	55.8	55.0	55.2	0.902[a]	42.9	28.6	4.530	0.286
SFA (%)								
Insufficient	23.3	18.1	19.2	2.446	25.0	28.6	1.331	0.872
Over consumption	37.2	50.6	47.8	0.294[a]	46.4	42.8	1.259	0.898
MUFA (%)								
Insufficient	48.8	46.9	47.3	0.888	57.2	42.8	3.980	0.415
Over consumption	27.9	23.1	24.1	0.641[a]	10.7	14.4	3.327	0.504
PUFA (%)								
Insufficient	16.3	21.2	20.2	0.633	32.1	28.6	3.962	0.410
Over consumption	62.8	56.9	58.1	0.729[a]	42.9	42.8	3.821	0.402
Omega-3 (g)								
Insufficient	27.9	29.4	29.1	0.210	57.1	71.4	8.370	0.062
Over consumption	23.3	25.6	25.1	0.900[a]	14.3	-	12.128	0.010*
Omega-6 (g)								
Insufficient	16.3	20.0	19.2	0.302	21.4	-	5.872	0.183
Over consumption	58.1	55.6	56.2	0.860[a]	42.9	28.6	7.753	0.076
Fiber (g)								
Insufficient	55.8	25.6	32.0	14.240	46.4	42.8	3.516	0.450
Over consumption	11.6	17.5	16.3	0.001[a]*	3.6	-	5.046	0.252
Vitamin A (mcg)								
Insufficient	44.2	36.3	37.9	1.602	53.6	57.1	3.467	0.489
Over consumption	25.6	23.1	23.7	0.449[a]	10.7	28.6	5.093	0.259
Thiamine (mg)								
Insufficient	100.0	31.9	46.3	30.853	100.0	100.0	-[d]	-[d]
Over consumption	-	7.5	5.9	0.000[b]*	-	-	18.026	0.001*
Riboflavin (mg)								
Insufficient	100.0	31.9	46.3	63.262	100.0	100.0	-[d]	-[d]
Over consumption	-	7.5	5.9	0.000[a]*	-	-	39.195	0.000*
Niacin (mg)								
Insufficient	9.3	-	2.0	92.450	3.6	14.3	2.428	0.640
Over consumption	39.5	100.0	87.2	0.000[b]*	32.1	28.6	49.625	0.000*
Pyridoxine (mcg)								
Insufficient	23.3	38.8	35.5	3.651	42.8	14.3	4.724	0.298
Over consumption	11.6	10.6	10.8	0.161[a]	3.6	-	3.384	0.463

Table 3. (continued)

Energy and nutrients	<%10				%10-20£	>%20£		
	Male (n:43)	Female (n:160)	Total (n:203)	χ^2 p	Male (n:28)	Male (n:7)	χ_e^2 χ_t^2	p_e[b] p_t[b]
	%	%	%		%	%		
Vitamin C (mg)								
Insufficient	11.6	28.1	24.6	18.358	3.6	-	2.069	0.696
Over consumption	76.8	40.0	47.8	0.000[a]*	85.7	100.0	19.564	0.000*
Vitamin E (mg)								
Insufficient	39.5	63.8	58.6	12.413	60.7	42.9	4.817	0.280
				0.002[a]*			5.010	0.251
Over consumption	14.0	3.1	5.4		14.3	-		
Sodium (mg)								
Insufficient	-	-	-	5.404	-	-	3.166	0.298
				0.020[c]*			0.435	0.733
Over consumption	100.0	93.1	94.6		92.9	100.0		
Calcium (mg)								
Insufficient	67.4	87.5	83.2	11.816	67.9	71.4	1.631	0.903
				0.002[b]*			6.447	0.178
Over consumption	4.7	-	1.0		-	-		
Magnesium (mg)								
Insufficient	69.8	56.9	59.6	3.336	85.7	71.4	3.084	0.520
				0.189[a]			7.284	0.088
Over consumption	9.3	7.5	7.9		3.6	-		
Zinc (mg)								
Insufficient	30.2	26.3	27.1	0.300	32.1	14.3	1.733	0.807
				0.861[a]			1.599	0.816
Over consumption	14.0	13.7	13.8		7.2	14.3		

Note: £ Since there were no female individuals in this group, the table was not included. χ_e^2 and p_e were used to evaluate men individuals, χ_t^2 and p_t were used to evaluate all individuals in different risk groups. [a] Pearson chi-square test; [b] Fisher exact chi-square test; Likelihood chi-square test was used. *p<0.05. [d] The evaluation could not be done because there was not enough number of individual.

Linear regression of parameters with significant correlation in Table 5 is given Table 6. While the FRS values of the participants were statistically significantly explained by the level of energy (4.3%), carbohydrate (g) (6.4%), protein (1.8%), riboflavin (2.2%) and zinc (2.8%) intake, the highest level of explanation among these values belonged to carbohydrates (p<0.05). It was also determined that one gram increase in carbohydrate intake will cause an increase of 0.252 in the FRS value (%) (Table 6).

DISCUSSION

Coronary Heart Disease Risk

The FRS values of men and women participating in the study were 9.7±6.87% and 1.8±2.10%, respectively (p<0.05). While 55.1% of male individuals are low; 35.9% have moderate and 9.0% high CVD risk, all women have low CVD risk. In the study of Meseri et al. (2014) with 10878 individuals, 67.4% of the participants have low, 23.2% are medium and 9.4% are high CVD risk. In the study conducted by Tekkeşin et al. (2011) it has been determined that 57.9% and 46.7% of men and women have low, 32.7% and 48.7% have medium; 9.4% and 4.6% have high CVD risk respectively. According to these results, the rate of individuals with medium risk is at a considerable level. This indicates that improving risk factors is an important requirement for the prevention of CVD.

Daily Energy and Macronutrient Intake

The increasing burden of CVD has increased the need for effective strategies to prevent disease development and health disease. Besides being one of the most important behavioral factors affecting health, nutrition explains approximately one-third of global

Table 4. Correlation between FRS values (%), energy and nutrient uptake of individuals

Energy and nutrients	<10%			10-20%£	>20%£		
	Male (n:43)	Female (n:160)	Total (n:203)	Male (n:28)	Male (n:7)		
	r	r	r	r	r	r_e	r_t
	p	p^{β}	p	p^{β}	p^{β}	p_e^{β}	p_t^{β}
Energy (kcal)	0.246	-0.014	0.214	-0.310	-0.214	0.007	0.223
	0.111$^{\alpha}$	0.856	0.002$^{\alpha}$*	0.108	0.645	0.951	0.001*
Carbohydrate (g)	0.270	0.118	0.259	-0.297	-0.321	0.069	0.270
	0.080$^{\beta}$	0.138	0.000$^{\alpha}$*	0.125	0.482	0.546	0.000*
Carbohydrate (%)	0.048	0.229	0.097	-0.007	-0.179	0.082	0.156
	0.762$^{\alpha}$	0.004*	0.169$^{\alpha}$	0.971	0.702	0.475	0.016*
Protein (g)	0.229	-0.023	0.114	-0.254	0.000	0.011	0.169
	0.139$^{\beta}$	0.775	0.105$^{\beta}$	0.192	1.000	0.923	0.009*
Protein (%)	-0.117	-0.006	-0.032	0.040	0.054	0.008	-0.013
	0.456$^{\beta}$	0.942	0.655$^{\beta}$	0.841	0.908	0.943	0.839
Total fat (g)	0.187	-0.127	0.050	-0.383	0.107	-0.091	0.086
	0.229$^{\beta}$	0.109	0.480$^{\beta}$	0.044*	0.819	0.427	0.188
Total fat (%)	-0.040	-0.239	-0.118	-0.029	0.143	-0.117	-0.162
	0.800$^{\alpha}$	0.002*	0.094$^{\alpha}$	0.883	0.760	0.308	0.013*
SFA (g)	0.265	-0.105	0.034	-0.171	-0.286	0.034	0.083
	0.086$^{\beta}$	0.186	0.630$^{\beta}$	0.385	0.535	0.766	0.201
SFA (%)	0.025	-0.156	-0.138	0.076	-0.179	0.020	-0.119
	0.876$^{\beta}$	0.049*	0.049$^{\beta}$*	0.702	0.702	0.859	0.067
MUFA (g)	0.069	-0.058	0.099	-0.364	-0.179	-0.140	0.139
	0.661$^{\beta}$	0.466	0.160$^{\beta}$	0.057	0.702	0.222	0.032*
MUFA (%)	-0.087	-0.047	-0.040	0.039	0.143	-0.082	-0.051
	0.581$^{\beta}$	0.558	0.566$^{\alpha}$	0.844	0.760	0.474	0.434
PUFA (g)	0.020	-0.177	-0.037	-0.284	0.500	-0.153	-0.014
	0.901$^{\alpha}$	0.025*	0.602$^{\beta}$	0.144	0.253	0.181	0.826
PUFA (%)	-0.194	-0.221	-0.129	-0.191	0.607	-0.206	-0.166
	0.214$^{\alpha}$	0.005*	0.066$^{\alpha}$	0.329	0.148	0.070	0.010*
Omega-3 (g)	0.385	-0.082	0.071	-0.294	-0.342	-0.120	0.032
	0.011$^{\beta}$*	0.302	0.313$^{\beta}$	0.129	0.452	0.297	0.624
Omega-6 (g)	-0.031	-0.180	-0.050	-0.275	0.571	-0.156	-0.023
	0.842$^{\alpha}$	0.023*	0.475$^{\beta}$	0.156	0.180	0.172	0.728
Cholesterol (mg)	0.022	0.014	0.068	-0.257	-0.071	-0.143	0.034
	0.887$^{\beta}$	0.865	0.332$^{\beta}$	0.187	0.879	0.212	0.602
Fiber (g)	0.178	-0.037	0.055	-0.280	-0.071	0.044	0.043
	0.253$^{\beta}$	0.641	0.435$^{\alpha}$	0.149	0.879	0.703	0.505

Note: £ Since there were no female individuals in this group, the table was not included. r_e and p_e were used to evaluate men individuals, r_t and p_t were used to evaluate all individuals in different risk groups. $^{\alpha}$ Pearson correlation; $^{\beta}$ Spearman correlation test was used. *p<0.05.

Table 5. Correlation between FRS values (%) and micronutrient uptake of individuals

Micronutrients	<10%			10-20%£	>20%£		
	Male (n:43)	Female (n:160)	Total (n:203)	Male (n:28)	Male (n:7)		
	r	r	r	r	r	r_e	r_t
	p	p^{β}	p^{β}	p^{β}	p^{β}	$p_e{}^{\beta}$	$p_t{}^{\beta}$
Vitamin A (mcg)	0.076	0.010	0.033	-0.348	-0.571	-0.114	-0.011
	0.627β	0.904	0.642	0.070	0.180	0.322	0.865
Thiamine (mg)	0.160	0.049	0.083	-0.303	-0.393	0.041	0.117
	0.307α	0.542	0.239	0.117	0.383	0.723	0.071
Riboflavin (mg)	0.144	0.082	0.142	-0.229	-0.643	0.028	0.178
	0.356β	0.305	0.043*	0.241	0.119	0.811	0.006*
Niacin (mg)	0.116	-0.053	0.067	-0.414	0.571	-0.056	0.125
	0.460β	0.503	0.346	0.028*	0.180	0.623	0.055
Pyridoxine (mg)	0.067	-0.042	0.002	-0.463	0.000	-0.017	0.037
	0.670β	0.599	0.978	0.013*	1.000	0.882	0.566
Vitamin C (mg)	-0.069	0.065	0.011	-0.391	0.000	-0.002	0.013
	0.658β	0.411	0.881	0.039*	1.000	0.987	0.847
Vitamin E (mg)	-0.040	-0.146	-0.008	-0.270	0.750	-0.175	0.025
	0.798β	0.065	0.908	0.164	0.052	0.125	0.696
Sodium (mg)	0.102	0.089	0.182	-0.294	0.036	-0.061	0.220
	0.514β	0.261	0.009*	0.129	0.939	0.597	0.001*
Potassium (mg)	0.132	0.027	0.034	-0.520	-0.214	0.002	0.037
	0.399β	0.738	0.633	0.005*	0.645	0.988	0.568
Calcium (mg)	0.226	0.012	0.082	-0.211	-0.643	0.027	0.093
	0.146β	0.883	0.245	0.282	0.119	0.812	0.154
Phosphorus (mg)	0.200	-0.048	0.057	-0.364	-0.107	0.014	0.095
	0.199β	0.546	0.422	0.057	0.819	0.903	0.146
Magnesium (mg)	0.156	-0.075	-0.012	-0.444	0.214	-0.054	-0.014
	0.318β	0.346	0.868	0.018*	0.645	0.639	0.834
Zinc (mg)	0.123	-0.034	0.098	-0.228	0.071	-0.028	0.158
	0.430α	0.699	0.164	0.243	0.879	0.809	0.015*

Note: £ Since there were no female individuals in this group, the table was not included. r_e and p_e were used to evaluate men individuals, r_t and p_t were used to evaluate all individuals in different risk groups. α Pearson correlation; β Spearman correlation test was used. *p<0.05.

CVD mortality and is considered as a primary target in the prevention and treatment of CVD. With the first dietary recommendations published by the American Heart Association Nutrition Committee in 1957, a nutrient-based approach began to form (Ravera et al., 2016).

In a study, the amount of total fat and SFA that individuals received with diet were found to be statistically significantly related to systolic blood pressure, which is among the risk factors of CVD; intake of total fat, SFA, MUFA, PUFA, cholesterol were found to be related to diastolic blood pressure (Mazidi et al., 2017). In a study conducted by Xu et al. (2006) it has been observed that the risk of CVD mortality increases with the increase in the consumption of total fat, SFA and MUFA. In a study conducted by Hu et al. (1999) it was found that the risk of CVD increases with increasing the content of the SFA of diet, and this risk decreases with the increase of MUFA and PUFA intake. In the study conducted by Hariri et al. (2017) it was

concluded that the rate of PUFA/SFA was statistically significantly higher in individuals with high risk compared to individuals with low and medium risk according to FRS. In the study carried out by Sohn et al. (2012) it was found that individuals with FRS>20% had lower PUFA intake than those with FRS<10%. There was no significant difference between individuals with high and low FRS levels in MUFA, SFA, and total fat intake. In this study, it was found that a positive correlation between the FRS value and intake of energy and MUFA (g) of all individuals in different risk groups in terms of CHD risk, and a negative correlation between the total fat (%) and PUFA (%) intakes ($p<0.05$) (Table 4).

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Various studies have shown that high glycemic index and glycemic load increase the risk of cardiovascular events (Beulens et al., 2007; Sieri et al., 2010; Mursu et al., 2011) as well as increased risk of CVD with increased carbohydrate consumption ($p<0.05$) (Sieri et al., 2010). As a result of the study conducted by Bazzanno et al. (2014) it was found that weight loss, HDL-C level increase, and total fat loss were significantly higher, but the ratio of TC/HDL-C was lower in individuals who followed a low-carbohydrate diet. When all individuals participating in this study are compared according to the risk of CHD, there is a difference in carbohydrate (g) intake ($p<0.05$). At the same time, both the amount of carbohydrate intake (g) and the percentage of the carbohydrate taken in total energy were found to have a positive correlation with the FRS value of all the individuals participating in the study. Also, it was determined that the nutrient that most affected (6.4%) the FRS values of the participants was carbohydrate (g) ($p<0.05$). A study conducted by Dehghan et al. (2017) found that a high intake of carbohydrates in the diet significantly increased total mortality and decreased the intake of fat, SFA, MUFA, and PUFA. It was observed that the total fat, saturated fat, and unsaturated fat content of the diet had no significant effect on CVD mortality and MI risk. In a study by Virtanen et al. (2014) it was determined that the risk of CHD is reduced when the content of SFA and PUFA of diet is replaced with carbohydrate. In a study conducted by Similä et al. (2013) it was observed that when the foods with high GI content in the diet were replaced with low GI foods, the risk of CVD was not decreased, and when replaced with SFA, this risk decreased and increased when replaced with MUFA. As a result of the study, it was stated that the carbohydrate and fat composition of the diet should be evaluated together for the risk of CVD.

In a study in which a diet, high in SFA, sugar, salt content, and low in vegetable and fruit content was accepted as a low dietary quality indicator, it was observed that the risk of CVD, CHD, and stroke increases as the diet quality of individuals decreases ($p<0.05$). This increase has been reported to be more obvious in overweight individuals (Adriouch et al., 2017). In the study conducted by Hariri et al. (2017), it was stated that for each increase in the consumption of fruit portions, a 0.14% reduction in FRS would be achieved. As a result of the study of Setayeshgar et al. (2015) it was found that the rate of FRS>10% was high

Table 6. Linear regression of patients' FRS value (%) and energy, macronutrients, micronutrients

	B	%95 (CI)	β	R^2	p
Energy (kcal)	0.002	0.001-0.003	0.207	0.043	0.001*
Carbohydrate (g)	0.018	0.009-0.027	0.252	0.064	0.000*
Carbohydrate (%)	0.052	-0.022-0.126	0.090	0.008	0.167
Protein (g)	0.024	0.001-0.047	0.134	0.018	0.039*
Total fat (%)	-0.068	-0.149-0.012	-0.108	0.012	0.097
MUFA (g)	0.046	-0.023-0.116	0.085	0.007	0.189
PUFA (%)	-0.098	-0.230-0.033	-0.095	0.009	0.142
Riboflavin (mg)	1.949	0.266-3.631	0.147	0.022	0.023*
Sodium (mg)	0.000	0.000-0.001	0.124	0.015	0.057
Zinc (mg)	0.252	0.061-0.451	0.166	0.028	0.010*

Note: *$p<0.05$

in individuals with abdominal obesity and low vegetable and fruit consumption and high potato consumption. In the study of Bhupathiraju & Tucker (2011), it was stated that inflammation and FRS value decreased with increasing variety compared to the number of vegetables and fruits consumed. In the study of Lairon et al. (2005) it was determined that weight, waist-to-hip ratio, blood pressure, apolipoprotein B, apolipoprotein B/A1, cholesterol, and homocysteine levels, which increase the risk of CVD, decrease as the intake fiber and insoluble fiber intake of individuals increases ($p<0.05$). As a common result of these studies, it has been stated that in societies with high CVD risk, the intake of fiber provided with fruit and vegetable consumption will be an important and effective method in reducing the risk of CVD. In this study, when the energy and nutrient intake levels of individuals were compared, there was a statistically significant correlation in the level of fiber intake between the males and females with FRS<10%, there was no statistically significant correlation between FRS value and fiber consumption.

In this context, the NCEP-ATP III guideline recommends therapeutic lifestyle changes to reduce the risk of CVD. Nutrition rich in unsaturated fatty acids is associated with low TG, high HDL-C levels. Besides, the reduction of SFA and cholesterol intake lowers the level of LDL-C. It is stated that the diet pattern with these features can reduce the disease prevalence with the positive changes in risk factors of FRS that help to estimate the risk of CVD (NIH, 2001).

Daily Micronutrient Intake

Vitamins have positive effects on CVD morbidity and mortality by preventing oxidative stress that causes the development of atherosclerosis (Núñez-Córdoba & Martínez-González, 2011). In the study, a significant difference was observed between thiamine, riboflavin, niacin, and vitamin C intake levels of individuals with different CHD risk (Table 3). At the same time, it has been found that the FRS value has a positive correlation with riboflavin in individuals at low risk of CHD; and has a negative correlation with niacin, pyridoxine, and vitamin C in individuals at moderate risk ($p<0.05$). However, when all individuals in different risk groups are evaluated, there is a positive correlation between FRS value and riboflavin intake ($p<0.05$).

In the study of Sohn et al. (2012), no significant difference was found between the vitamin intakes of individuals with different CHD risks. However, when evaluated according to the Food Quality Index, a significant difference was found between thiamine, niacin, A, B6, and C vitamins. In a study by Horigan et al. (2010), a statistically significant decrease was observed in both SBP and DBP levels of individuals in the group who received 1.6 mg riboflavin daily for 16

weeks compared to the placebo group. As a result of the study, it was stated that riboflavin may be effective in the prevention and treatment of hypertension and may play a role in reducing health expenditures. In the meta-analysis study conducted by Lavigne & Karas (2013), it was found that in addition to diet, niacin supplementation was associated with a decrease in CVD events and major CHD. In the study of Cangemi et al. (2013), vitamin E level was accepted as an independent risk factor for cardiovascular events and it was stated that the risk of CVD increased statistically in individuals with vitamin E level < 4.2 μmol/mmol. In a study by Weber et al. (1996), individuals with increased monocyte adhesion causing atherosclerosis were given daily vitamin C supplements (2 g/day). It was found that this supplement significantly reduced the adhesion level in smokers compared to non-smokers. These results show that adequate intake of all vitamins, especially vitamins with antioxidant properties, and therefore especially increasing the consumption of vegetables and fruits have an important role in reducing the risk of CVD.

Minerals play an important role in the regulation of cardiovascular functions. Mineral level imbalances are a potential risk factor that can lead to the development of CVDs (Mohammadifard et al., 2019). According to the results of this research, the FRS value in individuals with a moderate risk of CVD; has a negative correlation with potassium and magnesium ($p<0.05$). In the study conducted by Lai et al. (2015), the rate of cardiovascular mortality was found statistically significantly higher in individuals with low-normal serum potassium levels compared to individuals with normal potassium levels. In parallel with the results of this study, in a meta-analysis study, it was stated that the risk of stroke and heart failure decreases as a result of increased dietary magnesium intake (Fang et al., 2016). In the study by Kieboom et al. (2016), it was determined that low serum magnesium level increased carotid intima-media thickness, heart rate, CHD, and sudden heart death risks. Sohn et al. (2012), found no significant difference between the mineral intakes of individuals with different CHD risks. In this study, when all individuals in different risk groups are evaluated, among the sodium intake amount; there is a significant difference between the sodium and calcium intake levels of male and female individuals with low CHD risk ($p<0.05$). Besides, when individuals with low CHD risk and all individuals in different risk groups were evaluated, a positive correlation was found between FRS value and sodium ($p<0.05$). Many studies have found that sodium consumption increases the risk of CVD, which supports the results of this study (Aburto et al., 2013; Poggio et al., 2015). Many studies have found that sodium consumption increases the risk of CVD, this data supports the results of this research (Aburto et al., 2013; He et al., 2013; Poggio et al., 2015). Therefore, it is thought that the diet that will ensure the sodium intake to be at the recommended level will be effective in preventing increased CVD prevalence, morbidity and mortality.

CONCLUSION

As a result, CVD is a health problem that can be prevented by a healthy diet and lifestyle change, or that can be improved by medical treatment, nutritional therapy, and lifestyle changes after it occurs. Türkiye as well as in the world, the prevalence of CVD, rates of CVD mortality, and morbidity are increasing day by day. The primary target for preventing the increasing prevalence of CVD is to identify individuals with a high risk of disease and to make individual-specific changes in these individuals regarding risk factors and lifestyle. The measures to be taken will not only increase the quality of life of individuals but also reduce the use of resources. In the study, it was determined that there was a risk of CVD in both genders, more pronounced in male individuals. In this context, limiting salt consumption, reducing consumption of SFA, providing dietitian employment in primary health care centers, providing individuals with adequate and balanced nutrition education starting from an early age, conducting periodic risk screening, raising awareness of individuals about CVD risks, creating healthy living environments, establishing nutritional policies for preventive measures at the country level and ensuring their continuity; among the effective measures that can be taken to prevent CVD and to decrease the prevalence of CVD. Besides, broadcasting that encourages healthy nutrition and exercise and informs about CVD risk

factors via the media will help raise awareness about the disease.

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Compliance with Ethical Standards

Authors' Contributions

SG: Manuscript design, Field study, Statistical analyses, Writing.

AK: Manuscript design, Draft checking.

Both authors read and approved the final manuscript.

Conflict of Interest

The authors declare that there is no conflict of interest.

Ethical Approval

The studies have been approved by the appropriate institutional and/or national research ethics committee and have been performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

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