Nutrición Hospitalaria



Trabajo Original

Epidemiología y dietética

Fat intake and the risk of coronary heart disease among Jordanians

La ingesta de grasas y el riesgo de enfermedad coronaria de los jordanos

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Abstract

Introduction: dietary fat has been reported as one of the significant risk factors in the development of cardiovascular diseases (CVD).

Objective: this study aimed at assessing the possible association between fat intake and CVD.

Methods: the present case-control study was conducted in the center of coronary angiography. Three-hundred and ninety nine patients who referred for elective coronary angiography with clinical suspicion of coronary artery disease were enrolled. Dietary data were collected from each patient using an interview-based food frequency questionnaire.

Results: the findings of the present study revealed no significant differences between cases and controls regarding the intake of all types of fat either before or after energy adjustment. For both cases and controls the percentage of fat intake from total energy and the intakes of polyun-saturated and monounsaturated fats, cholesterol, omega-6 and omega-3 were within the recommended amounts. The intake of all fat types (except trans-fat) was not associated with the risk of developing CVD. Trans-fat intake in the second and third quartile increased the risk of CVD by OR 1.86 (95 % CI: 1.03-3.34) and 2.01 (95 % CI: 1.12-3.60), respectively.

Conclusions: while trans-fats may be significantly associated with the development of CVD in the first two quartiles, no association has been detected with other fat types.

Resumen

Introducción: se ha establecido que la grasa en la dieta es uno de los factores de riesgo significativos en el desarrollo de enfermedades cardiovasculares (ECV).

Objetivo: este estudio tuvo como objetivo evaluar la posible asociación entre la ingesta de grasa y la ECV.

Métodos: el presente estudio de casos y controles se realizó en el centro de la angiografía coronaria. Se inscribieron 399 pacientes que fueron remitidos para una angiografía coronaria electiva con sospecha clínica de enfermedad coronaria. Los datos dietéticos se obtuvieron de cada paciente mediante un cuestionario de frecuencia de alimentos basado en entrevistas.

Palabras clave:

Keywords:

Saturated fat. Trans-

fats. Cholesterol.

Monounsaturated

fats. Polyunsaturated fats and CVD.

Grasa saturada. Grasas trans. Colesterol. Grasas monoinsaturadas. Grasas poliinsaturadas y CVD. **Resultados:** los hallazgos del presente estudio no revelaron diferencias significativas entre los casos y los controles con respecto a la ingesta de todos los tipos de grasa, ya sea antes o después del ajuste de energía. Para ambos casos y controles, el porcentaje de ingesta de grasas de la energía total y las ingestas de grasas poliinsaturadas y monoinsaturadas, colesterol, omega-6 y omega-3 se encuentran dentro de las cantidades recomendadas. La ingesta de todos los tipos de grasa (excepto las grasas trans) no se asoció con el riesgo de desarrollar ECV. La ingesta de grasas trans en el segundo y tercer cuartil aumentó el riesgo de ECV en OR 1,86 (IC 95 %: 1,03-3,34) y 2,01 (IC 95 %: 1,12-3,60), respectivamente. **Conclusiones:** si bien las grasas trans pueden estar asociadas significativamente con el desarrollo de ECV en los dos primeros cuartiles, no se ha detectado asociación con otros tipos de grasa.

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INTRODUCTION

Cardiovascular disease (CVD) is the first cause of death and disability worldwide (1). Unhealthy diet is a leading risk factor for CVD where several studies indicated that excessive consumption of saturated fatty acids (SFA) increases the low-density lipoprotein (LDL) cholesterol, which may enhance the risk of developing CVD (1-3). Eckel et al. (2014) reported strong evidence that reducing SFA intake to 5-6 % of calories can be one important lifestyle modification for the management of CVD, mainly by lowering LDL cholesterol (4). However, several studies that evaluated the association of fat intake with CVD are controversial (5-8). Even though, for several decades, dietary guidelines have focused on the restriction of dietary cholesterol was not significantly associated with any coronary artery disease or ischemic stroke (7,8).

Epidemiological studies which examined the effect of monounsaturated fatty acids (MUFA) on CVD have shown mixed results. The Prevención con Dieta Mediterránea (PREDIMED) study reported that diets higher in MUFA reduced CVD events as compared with lower fat diets and, as a result, lowered the incidence of coronary heart disease (CHD) risk (9). Moreover, the Mediterranean dietary pattern, rich in MUFA, was recognized for its beneficial effects on CHD risk reduction in which a strong negative association was observed among followers of this dietary pattern and CHD risk (10). On the contrary, two metaanalyses of cohort studies found no significant association between MUFA and CHD events or death (11,12). Several studies investigated the effect of polyunsaturated fatty acids (PUFA) and the risk of CVD and the obtained results were conflicting (13-15). A meta-analysis of randomized controlled trials showed that the increase in PUFA intake actually reduced the risk of CHD death (14,16). However, some prospective cohort studies have shown that PUFA increased the risk of cardiovascular outcomes (13) or were not associated with risk (15).

Consumption of trans-unsaturated fatty acids was associated with a 34 % increase in all-cause mortality, 28 % increased risk of CHD mortality, and 21 % increase in the risk of CHD (17). However, no associations were observed for ruminant trans-fat with CHD (17).

This study aimed to explore the association between fat intake and the CVD risk in Jordan using a case-control design where a limited number of studies concerning risk factors for CVD has been published from the Middle East Countries. The concluded findings of this study would be of great importance for enhancing the public recognition about fat intake as a risk factor to develop CVDs. Those results could be used as a guidance to direct policy makers to initiate targeted nutritional and lifestyle strategies to prevent CVD events and alleviate their consequences. Also, those results could be used to establish more specified dietary guidelines regarding the intake of trans-fats and saturated fats for Jordanians.

SUBJECTS AND METHODS

PARTICIPANTS AND STUDY SETTING

A case-control study was conducted to assess the association between fat intake and CVD risk among Jordanians. Participants

of the present study were enrolled conveniently from the catheterization section of the Cardiology Department of Prince Hamzah Hospital, a referral hospital in the capital Amman, between January and December 2015. A total of 399 participants who underwent coronary angiography were included. The cases and controls were age and gender matched with 1:1 ratio. Participants with kidney disease, liver disease or gastrointestinal diseases were excluded. All participants were requested to sign a written consent form to participate in the study. The study protocol was designed according to the ethical guidelines of the 1975 Declaration of Helsinki, and the study was approved by the Institutional Review Board Ethics Committee at Prince Hamzah Hospital. One day before undergoing coronary angiography, all data were collected from patients upon filling a standardized questionnaire by trained dietitians to record socio-demographic factors, previous health issues (hypertension, diabetes mellitus, dyslipidaemia), smoking status, and family history of CVDs information.

CORONARY ANGIOGRAPHY

Seldinger technique was used to insert a catheter by trained cardiologists into the radial artery, and the tip was advanced to the aortic sinus cusp. To visualize the arterial tree, X-ray images of the transient radio-contrast distribution within the coronary arteries were carried out. The degree of obstruction was estimated as percentage of the arterial lumen by comparing the area of narrowing to an adjacent normal artery. Consistent with prior studies, CAD was defined as \geq 20 % stenosis of one or more coronary arteries (18,19). Participants with no stenosis (0 %) were enrolled as controls.

NUTRIENTS INTAKE ASSESSMENT

A validated Arabic quantitative food frequency questionnaire (FFQ) was used to assess the dietary intake pattern (20). The information dealing with dietary history of participants was investigated in the FFQ questions. During face-to-face interviews, the participants were asked to record how frequently, on average, they had consumed one standard serving of specific food items in nine categories (< 1/month, 2-3/month, 1-2/week, 3-4/week, 5-6/week, 1/day, 2-3/day, 4-5/day, or 6/day) during the past year. Food lists in the modified FFQ questions were classified based on types of food: 21 items of fruits and juices; 21 items of vegetables; eight items of cereals; nine items of milk and dairy products; four items of beans; 16 items of meat such as red meat (lamb and beef), chicken, fish, cold meat, and others; four items of soups and sauces; five items of drinks; nine items of snacks and sweets; and 14 items of herbs and spices. Food models and standard measuring tools were used for better estimation of portion size. Dietary analysis software (ESHA Food Processor SQL version 10.1.1; ESHA, Salem, OR, USA) was used to analyze dietary intakes with additional data on foods consumed in Jordan. After entering the amounts which were consumed daily from the raw fats, foods containing fats, fried foods and other foods to the ESHA program, total amounts of different fats were added and calculated. Recipes for Jordanian foods were entered and the total intake from these recipes was calculated. Energy (kcal), energy from fat (kcal), energy from saturated fat (kcal), energy from trans fatty acids (kcal), % of fat, fat (g), SFA (g), MUFA (g), PUFA (g), trans fat (g), cholesterol (mg), omega-3 (g), omega-6 (g), omega-3:omega-6, oleic (18:1) (g), linoleic (18:2) (g), lonolenic (18:3) (g), eicosen (20:1) (g), arachidon (20:4) (g), eicosapentaenoic acid (EPA) (20:5) (g), and docosapentaenoic acid (DPA) (22:5) (g) intake was assessed from the whole food items which are included in the used FFQ.

7-DAY PHYSICAL ACTIVITY RECALL (PAR)

A 7-day PAR validated questionnaire, which is an organized questionnaire, was used to calculate a participant's recall of time spent participating in exercise over a seven-day period (21). This questionnaire helps to divide individual physical activity levels into three categories. Participants were asked to respond to a PAR question based on the way they used to behave prior undergoing coronary angiography.

ANTHROPOMETRIC MEASUREMENTS

All anthropometric measurements were carried out by a trained dietitian. Body weight was measured to the nearest 0.1 kg, with minimal clothing and without shoes, using a calibrated scale (Seca[®], Hamburg, Germany). Height and waist circumference were measured to the nearest 1 cm with participants in standing position without shoes using a calibrated portable measuring rod. Body mass index (BMI) was calculated as weight (kg) divided by height square (m²).

STATISTICAL ANALYSIS

SPSS version 20.0 software (SPSS Inc., Chicago, IL, USA) was used to perform the statistical analysis. The significance level was set at $p \le 0.05$. Mean \pm standard error of mean (SEM) and percentages were used for descriptive statistics. To evaluate the differences between cases and controls in continuous variables, t-tests were used, and Chi-squared was used to detect the differences among categorical variables. Potential confounders (age, gender, BMI, smoking, physical activity, total energy intake, occupation, education level, marital status and family history) were chosen based on reported risk factors for CVDs. The quartiles were calculated using the cut-off points at 25, 50 and 75 % of total nutrients intake. The first quartile was determined if the intake was below 25 %, while the second one was determined if the intake was between 25-50 %. The third quartile was between 50-75 % and the fourth was above 75 %. Multinomial logistic regression model and linear logistic regression model were used to calculate odd ratios (OR) and its 95 % confidence interval (Cl) and p-for-trend for trend, respectively. The energy adjustment was performed using the residual method of Willett in which residuals were computed from a regression analysis (22).

RESULTS

Briefly, 239 males and 160 females participated in this study. The study participants' characteristics are shown in table I and have been as mentioned elsewhere (23). The main characteristics of study subjects categorized by gender are summarized in table I. The cases had higher mean fasting blood glucose levels compared to controls. Moreover, cases showed higher blood triglyceride levels compared to controls. In addition, there were differences in physical activity measured as MET (min/week). Overall, the cases were less active compared to controls, and reported more previous health problems than controls, in both men and women.

Table II reveals that no significant difference was detected between cases and controls in all types of fat intake either before or after energy adjustment. Also, the percentage of fat intake from total energy was within the recommended level. However, the amount of saturated fat (around 30 g; 9.0 %) was close to the amount of MUFA (around 33 g; 10.5 %) which is not consistent with a healthy diet. On the other hand, the intake of PUFA (19 g; 6.0 %) was lower than both saturated and MUFA. The consumption of cholesterol was below the recommended amount (intake: 255 mg *vs* recommended: 300 mg). Oleic fatty acid was the prominent type of fat among the cases and controls. Additionally, the intake of omega-6 and omega-3 among cases and controls was in agreement with the recommended amounts.

Table III shows the crude and adjusted ORs and their 95 % Cl for CVD by fat types quartiles. OR and their 95 % Cl for fat types were adjusted for age, gender, BMI, smoking, and physical activity. The intake of all fat types (except trans-fat) was not associated with the risk of developing CVD. Trans-fat intake in the second and third quartile increased the risk of CVD by adjusted for about 1.86 (95 % Cl: 1.03-3.34) and 2.01 odds (95 % Cl: 1.12-3.60), respectively. Similar results of the association between trans-fat and CVD obtained was obtained for the crude OR.

DISCUSSION

This study aimed at evaluating the association between the intakes of different fat types and the development of CVD among Jordanians. Due to the discrepancy in the findings of multiple studies, the research is still unclear to judge if there is really a positive association between SFA and CVD, as traditionally speculated. The main findings of this study did not support the results of many other studies which stated that total dietary fats, saturated fats and cholesterol were positively associated with the risk of developing CHD (12,24,25). However, our findings came in agreement with several other studies (17,25,26). De Souza et al. (2015) reported null associations between saturated fat intake

	Male (n = 239)	Female	(n = 160)	
Variable (mean \pm SEM)	Cases (n = 132)	Controls (n = 107)			
Ago (4)		48.4 ± 1.0	Cases (n = 73)	Controls (n = 87)	
Age (y)	48.1 ± 0.5		61.1 ± 1.2	54.1 ± 1.0	
BMI (kg/m ²)	29.9 ± 0.69	29.9 ± 0.56	31.9 ± 0.67	31.9 ± 0.50	
Physical activity (MET/min)	11,289.5 ± 765.4	13,258.9 ± 752.7	7,539.9 ± 493.1	10,312.8 ± 682.8	
Systolic blood pressure (mmHg)	130.8 ± 2.4	128.4 ± 2.3	142.9 ± 26.3	140.6 ± 23.9	
Diastolic blood pressure (mmHg)	75.7 ± 0.98	78.8 ± 1.6	81.2 ± 1.7	80.5 ± 2.5	
LDL (mg/dl)	109.8 ± 10.2	108.7 ± 9.5	110.0 ± 11.1	131.1 ± 17.2	
HDL (mg/dl)	38.8 ± 2.78	39.5 ± 1.4	47.2 ± 3.4	48.40 ± 2.09	
Triglycerides (mg/dl)	288.3 ± 45.8	163.1 ± 16.1	260.4 ± 47.9	176.3 ± 14.4	
Cholesterol (mg/dl)	208.4 ± 16.8	186.4 ± 7.9	189.3 ± 9.7	215.0 ± 8.9	
Fasting blood glucose (mmol/l)	8.7 ± 0.48	6.8 ± 0.31	10.5 ± 0.75	7.7 ± 0.42	
Variable n (%)					
		Marital status			
Married	128 (97.0)	103 (96.3)	57 (78.1)	77 (88.5)	
Single	4 (3.0)	3 (2.8)	2 (2.7)	1 (1.1)	
Divorced	0 (0.0)	1 (0.9)	1 (1.4)	2 (2.3)	
Widowed	0 (0.0)	0 (0.0)	13 (17.8)	7 (8.0)	
		Education level		1	
Illiterate	7 (5.3)	4 (3.7)	23 (31.5)	10 (11.5)	
Primary education	54 (41.2)	47 (43.9)	35 (47.9)	36 (41.4)	
Secondary education	41 (31.3)	30 (28.0)	12 (16.4)	23 (26.4)	
Diploma	15 (11.5)	11 (10.3)	3 (4.1)	16 (18.4)	
Bachelor	10 (7.6)	14 (13.1)	0 (0.0)	2 (2.3)	
Postgraduate	4 (3.1)	1 (0.9)	0 (0)	0 (0)	
Tostgraddato	+ (0.1)	BMI	0 (0)	0 (0)	
Underweight	1 (0.8)	0 (0.0)			
Normal	25 (18.9)	23 (21.5)	8 (11.0)	7 (8.0)	
Overweight	61 (46.2)	32 (29.9)	23 (31.5)	18 (20.7)	
	. ,				
Obese	45 (34.1)	52 (48.6)	42 (57.5)	62 (71.3)	
	-	cal activity categories		4 (1 4)	
Inactive	7 (5.3)	3 (2.8)	10 (13.7)	1 (1.1)	
Minimally active	36 (27.3)	14 (13.1)	22 (30.1)	14 (16.1)	
Health enhancing physical activity	89 (67.4)	90 (84.1)	41 (56.2)	72 (82.8)	
		Smoking			
Yes	93 (70.5)	54 (50.5)	6 (8.2)	16 (18.4)	
No	23 (17.4)	33 (30.8)	31 (42.5)	36 (41.4)	
Previous	11 (8.3)	9 (8.4)	4 (5.5)	1 (1.1)	
Passive	5 (3.8)	11 (10.3)	32 (43.8)	34 (39.1)	
		Health problem			
Yes	91 (68.9)	60 (56.1)	69 (94.5)	71 (81.6)	
No	41 (31.1)	47 (43.9)	4 (5.5)	16 (18.4)	
	Far	nily history of CAD			
Yes	52 (39.4)	28 (26.2)	31 (42.5)	42 (48.3)	
No	80 (60.6)	79 (73.8)	42 (57.5)	45 (51.7)	

Table I. General characteristics of study participants based on gender

*Significant difference was set at p < 0.05. SEM: standard error of mean; BMI: body mass index; MET: metabolic equivalent-minutes; LDL: low-density lipoprotein; HDL: high-density lipoprotein; CAD: coronary artery disease.

of energy intake							
Nutrients	Cases	Controls		Cases	Controls		
	(n = 205) (n = 194)		p-value	(n = 205)	(n = 194)	p-value	
	Crude Mean ± SEM			Adjusted for energy			
		-	0.040	Mean ± SEM			
Energy (kcal)	2,795.4 ± 68.1	2,914.1 ± 74.6	0.240	-	-	-	
Energy from fat (kcal)	933.9 ± 26.2	964.2 ± 29.0	0.438	-	-	-	
% of fat	33.2 ±0.49	33.0 ± 0.49	0.719	-	-	-	
% Saturated fat	9.2 ± 0.19	9.1 ± 0.20	0.853				
% Monounsaturated fat	10.4 ± 0.23	10.6 ± 0.24	0.540				
% Polyunsaturated fat	6.2 ± 0.14	6.1 ± 0.14	0.585				
% Trans fat	0.17 ± 0.04	0.17 ± 0.04	0.970				
Fat (g)	104.2 ± 2.9	107.6 ± 3.2	0.438	104.2 ± 1.4	107.6 ± 1.5	0.110	
Saturated fat (g)	28.9 ± 0.94	30.5± 1.2	0.302	28.9 ± 0.56	30.5 ± 0.67	0.078	
Monounsaturated fat (g)	32.5 ± 1.1	34.0 ± 1.1	0.316	30.5 ± 0.67	34.1 ± 7.4	0.878	
Polyunsaturated fat (g)	19.2 ± 0.58	19.4 ± 0.63	0.738	19.1 ± 0.41	19.4 ± 0.47	0.648	
Trans-fat (g)	0.55 ± 0.12	0.57 ± 0.14	0.897	0.55 ± 0.12	0.57 ± 0.14	0.897	
Cholesterol (mg)	263.9 ± 13.2	252.7 ± 13.4	0.553	263.9 ± 11.4	252.7 ± 10.2	0.469	
Omega-3 (g)	1.1 ± 0.03	1.1 ± 0.04	0.319	1.1 ± 0.03	1.1 ± 0.04	0.319	
Omega-6 (g)	16.6 ± 0.53	16.7 ± 0.56	0.879	$16.6 \pm 0.40.$	16.7 ± 0.44	0.845	
Omega-3/Omega-6	0.07 ± 0.00	0.07 ± 0.00	0.448	0.07 ±0.002	0.07 ± 0.002	0.448	
Oleic (18:1) (g)	30.4 ± 1.0	31.8 ± 1.0	0.318	30.4 ± 0.70	31.8 ± 0.72	0.150	
Linoleic (18:2) (g)	16.5 ± 0.53	16.6 ± 0.56	0.864	16.5 ± 0.40	16.6 ± 0.44	0.826	
Linolenic (18:3) (g)	0.98 ± 0.03	1.0 ± 0.03	0.287	0.98 ± 0.03	1.0 ± 0.03	0.287	
Eicosen (20:1) (g)	0.16 ± 0.01	0.17 ± 0.01	0.714	0.16 ±0.01	0.17 ±0.01	0.714	
Arachidon (20:4) (g)	0.14 ± 0.01	0.13 ± 0.01	0.202	0.14 ± 0.01	0.13 ± 0.01	0.202	
Eicosapentaenoic acid (20:5) (g)	0.02 ±0.00	0.02 ± 0.00	0.828	0.02 ± 0.002	0.02 ± 0.002	0.828	
Docosapentaenoic acid (22:5) (g)	0.01 ± 0.001	0.01 ± 0.001	0.604	0.01 ± 0.001	0.01 ± 0.001	0.604	

 Table II. Fat and type of intake of cases and controls before and after the adjustment of energy intake

and all-cause mortality (relative risk 0.99, 95 % CI: 0.91 to 1.09), CVD mortality (0.97, 0.84 to 1.12), total CHD (1.06, 0.95 to 1.17), ischemic stroke (1.02, 0.90 to 1.15), and type 2 diabetes (0.95, 0.88 to 1.03) (17). Siri-Tarino et al. (2010) illustrated that the intake of saturated fat was not associated with an increased risk of CHD, stroke, or CVD; the pooled relative risk estimates that compared extreme quantiles of saturated fat intake were 1.07 (95 % Cl: 0.96, 1.19; p = 0.22) for CHD, 0.81 (95 % Cl: 0.62, 1.05; p = 0.11) for stroke, and 1.00 (95 % CI: 0.89, 1.11; p = 0.95) for CVD (25). Additionally, Harcombe et al. (2016) revealed that none of the studies included in their meta-analysis found a significant relationship between CHD deaths and total dietary fat intake (26). Factors such as food matrix, source of saturated fat, and fatty acid chain length may influence the health effects of saturated fats and therefore, might explain the contradiction in the current evidence regarding their association with CVD (27,28).

Although the 2015-2020 Dietary Guidelines for Americans recommend substituting both MUFA and PUFA for saturated fats, the consistency in the current evidence is lacking (27,28). Higher intakes of PUFA were found to be significantly associated with a lower risk of CHD comparing the highest and lowest quintile for PUFAs 0.80, (0.73 to 0.88; p-trend < 0.0001) (29). On contrary, Chowdury et al. (2014) reported no relationship between dietary PUFA and coronary disease, with a risk ratio 0.98 (CI: 0.90 to 1.06) in eight cohort studies containing 206,376 participants with 8,155 events (12). The proportions among daily intake of different types of fat might provide a partial explanation for the inconsistent evidence concerning the association of unsaturated fats with CVD (30,31); the optimal balance among daily intake of different fatty acids can effectively improve the health while the incorrect ratio may increase disease risk (30,31). Here, no significant association was detected between the intake of PUFA, MUFA, omega-6 or omega-3 and CVD risk among cases and controls.

Nutrients	Q1*	Q2	Q3	Q4
		Fat (g)		
Cases number	57	56	45	47
Controls number	42	44	55	53
[†] Adjusted OR (95 % Cl)	1	0.89 (0.50-1.60)	0.57 (0.32-1.03)	0.80 (0.44-1.45)
Crude OR (95 % Cl)	1	0.94 (0.54-1.64)	0.60 (0.34-1.06)	0.65 (0.37-1.14)
p-for-trend	0.358		•	
		Saturated fat (g)	
Cases number	54	57	48	46
Controls number	45	43	53	53
[†] Adjusted OR (95 % Cl)	1	1.07 (0.60-1.93)	0.77 (0.43-1.37)	0.90 (0.49-1.64)
Crude OR (95 % Cl)	1	1.10 (0.63-1.93)	0.75 (0.43-1.32)	0.72 (0.41-1.27)
p-for-trend	0.516			
		Monounsaturated	fat (g)	
Cases number	51	56	51	47
Controls number	48	45	49	52
[†] Adjusted OR (95 % Cl)	1	1.08 (0.60-1.94)	0.85 (0.47-1.54)	0.78 (0.43-1.43)
Crude OR (95 % Cl)	1	1.17 (0.67-2.04)	0.98 (0.56-1.71)	0.85 (0.49-1.49)
p-for-trend	0.736			
		Polyunsaturated fa	at (g)	
Cases number	51	56	44	54
Controls number	49	44	55	46
⁺ Adjusted OR (95 % Cl)	1	1.31 (0.73-2.33)	0.91 (0.51-1.64)	1.19 (0.67-2.13)
Crude OR (95 % CI)	1	1.22 (0.70-2.13)	0.77 (0.44-1.34)	1.13 (0.65-1.97)
p-for-trend	0.361			
		Trans-fat (g)		
Cases number	45	54	57	49
Controls number	61	42	42	49
[†] Adjusted OR (95 % Cl)	1	1.86 (1.03-3.34)	2.01 (1.12-3.60)	1.38 (0.77-2.48)
Crude OR (95 % CI)	1	1.74 (1.00-3.04)	1.84 (1.06-3.20)	1.36 (0.78-2.36)
p-for-trend	0.311			
		Cholesterol (m	g)	
Cases number	50	57	47	51
Controls number	49	43	54	48
⁺ Adjusted OR (95 % Cl)	1	1.50 (0.83-2.69)	0.90 (0.50-1.62)	1.02 (0.56-1.86)
Crude OR (95 % CI)	1	1.30 (0.74-2.27)	0.85 (0.49-1.49)	1.04 (0.60-1.82)
p-for-trend	0.545			

Table III. The OR (95 % CI) for nutrient intake among Jordanian participants

*Reference quartiles. †Adjusted for age, gender, BMI, smoking, physical activity, total energy intake, education level and family history.

Two main reasons might have contributed to the null findings; firstly, our study findings revealed that the intake of PUFA (around 19 g/day; 6.0 %), MUFA (around 32 g/day; 10.5 %), omega-6 fatty acid (16 g/day) and omega-3 fatty acid (1.1 g/day) was similar in cases and controls. Secondly, all of these types of fat were consumed in approximately the recommended doses (32).

Trans-fat intake was found to be significantly associated with CVD among Jordanians, which is consistent with many studies (12,33). Trans-fats from foods may adversely affect the risk of

coronary disease by raising LDL cholesterol levels and lowering high-density lipoprotein (HDL) cholesterol levels (34), increasing Lp(a) lipoprotein levels (34), raising triglyceride levels (34), and interfering with essential-fatty acid metabolism (35). Trans-fats were associated with all-cause mortality, total CHD, and CHD mortality, probably because of higher levels of intake of industrial trans-fats than of ruminant trans-fats (17). Industrial and ruminant trans-fats consist of the same positional trans isomers, but in different proportions. The isomer profile depends on conditions of hydrogenation, such as catalysts used and temperature of hydrogenation for industrial trans-fats and rumen pH, and the composition of oils in the diet for ruminant trans-fatty acids (36). Chowdhury et al. (2014) revealed in their meta-analysis that the intakes of SFA, MUFA, alpha-linoleic acid, long-chain omega-3 or omega-6 fatty acids were not associated with coronary disease (12). However, they found that trans-fats increased the incidence of coronary disease (RR 1.16, 95 % CI: 1.06 to 1.27) (12). Li et al. (2015) studied 84,628 women (Nurses' Health Study, 1980 to 2010) and 42,908 men (Health Professionals Follow-up Study, 1986 to 2010) who were free of diabetes, cardiovascular disease, and cancer at baseline, and found that trans-fat intake was significantly associated with an increased risk of CHD (HR: 1.20, 95 % CI: 1.09 to 1.32; p-trend = 0.002) (29). It has been estimated that the consumption of about 5 g of trans-fat per day is associated with 25 % increase in the risk of CHD (29). Although the association between CVD and trans-fat appears to be causal, no randomized controlled trial with hard endpoints has been reported (37).

Regarding cholesterol findings, our results showed no significant association between the intakes of cholesterol and CVD risk, with insignificant difference in the mean intake of cholesterol for cases compared to controls. McNamara (2000) demonstrated in his review that the analysis of the available epidemiological and clinical data indicated that, for the general population, dietary cholesterol makes no significant contribution to atherosclerosis and risk of cardiovascular disease (38). A recent study of Rhee et al. (2017) performed on 30,068 participants (mean age 40.8 years; 84.5 % men) in a health screening program in Korea documented that dietary cholesterol intake did not show any association with LDL level or with risk for coronary artery calcification in apparently healthy Korean adults (39). The Scientific Report of the 2015 Dietary Guidelines Advisory Committee (DGAC) in the United States concluded that "cholesterol is not a nutrient of concern for overconsumption", suggesting that there no longer be a recommended upper limit for dietary cholesterol intake (40). This conclusion came after decades of the recommendation of 300 mg/d as the upper limit for dietary cholesterol. Despite eliminating the upper limit from the Dietary Guidelines, individuals should eat as little dietary cholesterol as possible as part of their healthy eating pattern to hinder CVD risk. Therefore, the lack of association between the dietary cholesterol and CVD in the current study could be due to the considerable low daily consumption of cholesterol (< 300 mg/ day) for both cases and controls.

The main strength points of this study are the use of a validated Arabic FFQ that was modified to reflect the food consumption pattern in Arab countries, especially Jordan, as well as the use of food models and measuring tools to estimate portion sizes. There are limitations in this study; for example, the one year dietary recall period may not be an accurate amount of time in which to conclude that an association exists between fat intake and CVD development. Nevertheless, we believe that the recall period of one year is very likely reflective of the previous years. Thus, the association between fat dietary intake and CVD may have been developing for several years. In conclusion, no association has been found between cholesterol, saturated fats, PUFA and MUFA and the risk of CVD. On the contrary, a significant association has been detected between trans-fats and the development of CVD. This may be attributed to the fact that the intake of most of these fat types (except transfats) are within the recommended percentages and amounts.

ETHICS APPROVAL

The study was approved by the IRB at Prince Hamza Hospital, and all participants gave written consent to participate in the study.

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REFERENCES

- Forouzanfar MH, Afshin A, Alexander LT, et al. Global, regional, and national comparative risk assessment of 79 behavioral, environmental and occupational, and metabolic risks or clusters of risks, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet 2016;388:1659-724.
- Feigin VL, Roth GA, Naghavi M, et al. Global burden of stroke and risk factors in 188 countries, during 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet Neurol 2016;15:913-24.
- Mensink R. Effects of saturated fatty acids on serum lipids and lipoproteins: a systematic review and regression analysis. World Health Organization: Geneva, Switzerland; 2016.
- Eckel RH, Jakicic JM, Ard JD, et al. 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2014;63:2960-84.
- Berger S, Raman G, Vishwanathan R, et al. Dietary cholesterol and cardiovascular disease: a systematic review and meta-analysis. Am J Clin Nutr 2015;102(2):276-94.
- McGee DL, Reed DM, Yano K, et al. Ten-year incidence of coronary heart disease in the Honolulu Heart Program: relationship to nutrient intake. Am J Epidemiol 1984;119:667-76.
- Posner BM, Cobb JL, Belanger AJ, et al. Dietary lipid predictors of coronary heart disease in men: the Framingham Study. Arch Intern Med 1991;151:1181-207.
- Nettleton JA, Brouwer IA, Geleijnse JM, et al. Saturated fat consumption and risk of coronary heart disease and ischemic stroke: a science update. Ann Nutr Metab 2017;70:26-33. DOI: 10.1159/000455681
- Estruch R, Ros E, Salas-Salvado J, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. N Engl J Med 2013;368:1279-90.
- Rosato V, Temple NJ, La Vecchia C, et al. Mediterranean diet and cardiovascular disease: a systematic review and meta-analysis of observational studies. Eur J Nutr 2019;58(1):173-91. DOI: 10.1007/s00394-017-1582-0
- Skeaff CM, Miller J. Dietary fat and coronary heart disease: summary of evidence from prospective cohort and randomised controlled trials. Ann Nutr Metab 2009;55:173-201.
- Chowdhury R, Warnakula S, Kunutsor S, et al. Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. Ann Intern Med 2014;160:398-406.
- Praagman J, Beulens JW, Alssema M, et al. The association between dietary saturated fatty acids and ischemic heart disease depends on the type and source of fatty acid in the European Prospective Investigation into Cancer and Nutrition-Netherlands cohort. Am J Clin Nutr 2016;103:356-65.

- Virtanen JK, Mursu J, Tuomainen T, et al. Dietary fatty acids and risk of coronary heart disease in men: the Kuopio Ischemic Heart Disease Risk Factor Study. Arterioscler Thromb Vasc Biol 2014;34:2679-87.
- Pietinen P, Ascherio A, Korhonen P, et al. Intake of fatty acids and risk of coronary heart disease in a cohort of Finnish men. The alpha-tocopherol, beta-carotene cancer prevention study. Am J Epidemiol 1997;145:876-87.
- Ramsden CE, Hibbeln JR, Majchrzak SF, et al. n-6 fatty acid-specific and mixed polyunsaturated dietary interventions have different effects on CHD risk: a meta-analysis of randomised controlled trials. Br J Nutr 2010;104:1586-600.
- De Souza RJ, Mente A, Maroleanu A, et al. Intake of saturated and trans unsaturated fatty acids and risk of all-cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. BMJ 2015;11(351):h3978.
- Bradley SM, Maddox TM, Štanislawski MA, et al. Normal coronary rates for elective angiography in the Veterans Affairs Healthcare System: insights from the VA CART program (veterans affairs clinical assessment reporting and tracking). J Am Coll Cardiol 2014;63(5):417-26.
- Maddox TM, Stanislawski MA, Grunwald GK, et al. Nonobstructive coronary artery disease and risk of myocardial infarction. JAMA 2014;312(17):1754-63
- Tayyem RF, Abu-Mweis SS, Bani-Hani K, et al. Validation of a food frequency questionnaire to assess macronutrient and micronutrient intake among Jordanians. J Acad Nutr Diet 2014;114(7):1046-52.
- Sallis JF, Haskell WL, Wood PD, et al. Physical activity assessment methodology in the Five-City Project. Am J Epidemiol 1985;121(1):91-106.
- Willett W. Nutritional Epidemiology. 3rd edition. New York, NY: Oxford University Press: 2013.
- Al-Shudifat A, Azab M, Agraib LM, et al. Is the intake of antioxidant associated with the risk of coronary heart disease? A Jordanian case-control study. Top Clin Nutr 2019;34(4):259-68.
- Guasch-Ferré M, Babio N, Martínez-González MA, et al. Dietary fat intake and risk of cardiovascular disease and all-cause mortality in a population at high risk of cardiovascular disease. Am J Clin Nutr 2015;102(6):1563-73.
- Siri-Tarino PW, Sun Q, Hu FB, et al. Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. Am J Clin Nutr 2010;91(3):535-46.
- Harcombe Z, Baker JS, Davies B. Evidence from prospective cohort studies did not support the introduction of dietary fat guidelines in 1977 and 1983: a systematic review. Br J Sports Med 2017;51(24):1737-42. DOI: 10.1136/ bjsports-2016-096409

- Hannon BA, Thompson SV, An R, et al. Clinical outcomes of dietary replacement of saturated fatty acids with unsaturated fat sources in adults with overweight and obesity: a systematic review and meta-analysis of randomized control trials. Ann Nutr Metab 2017;71(1-2):107-17.
- Michelle AB, Kristina SP, Penny MK. Saturated fatty acids and cardiovascular disease: replacements for saturated fat to reduce cardiovascular risk. Healthcare (Basel) 2017;5(2):29.
- Li Y, Hruby A, Bernstein AM, et al. Saturated fats compared with unsaturated fats and sources of carbohydrates in relation to risk of coronary heart disease: a prospective cohort study. J Am Coll Cardiol 2015;66(14):1538-48.
- Hammad S, Pu S, Jones PJ. Current evidence supporting the link between dietary fatty acids and cardiovascular disease. Lipids 2016;51(5):507-17.
- Regulska-Ilow B, Ilow R, Kawicka A, et al. Evaluation of fatty acids daily intake and diets atherogenicity of dietetics students of Wroclaw Medical University. Rocz Panstw Zakl Hig 2013;64(3):183-90.
- Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (2002/2005). Available from: www.nap.edu
- Oomen CM, Ocke MC, Feskens EJ, et al. Association between trans fatty acid intake and 10-year risk of coronary heart disease in the Zutphen Elderly Study: a prospective population-based study. Lancet 2001;357(9258):746-51.
- 34. Katan MB, Zock PL. Trans fatty acids and their effects on lipoproteins in humans. Annu Rev Nutr 1995;15:473-93.
- Kinsella JE, Bruckner G, Mai J, et al. Metabolism of trans fatty acids with emphasis on the effects of trans, trans-octadecadienoate on lipid composition, essential fatty acid, and prostaglandins: an overview. Am J Clin Nutr 1981;34:2307-18.
- Hulshof KF, van Erp-Baart MA, Anttolainen M, et al. Intake of fatty acids in Western Europe with emphasis on trans fatty acids: the TRANSFAIR Study. Eur J Clin Nutr 1999;53:143-57.
- Brouwer IA, Wanders AJ, Katan MB. Trans fatty acids and cardiovascular health: research completed? Eur J Clin Nutr 2013;67(5):5411-7.
- McNamara DJ. Dietary cholesterol and atherosclerosis. Biochim Biophys Acta 2000;1529(1-3):310-20.
- Rhee EJ, Ryu S, Lee JY, et al. The association between dietary cholesterol intake and subclinical atherosclerosis in Korean adults: The Kangbuk Samsung Health Study. J Clin Lipidol 2017;11(2):432-41.
- U. S. Department of Agriculture, U. S. Department of Health and Human Services. Scientific Report of the 2015 Dietary Guidelines Advisory Committee. Washington, DC: U. S. Government Printing Office; 2016.