# Nutrición Hospitalaria



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10.20960/nh.02761

#### **1OR 2761 EPIDEMIOLOGÍA**

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6La ingesta de grasas y el riesgo de enfermedad coronaria del corazón de 7los jordanos

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17**Received:** 27/06/2019

18Accepted: 12/01/2020

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*Availability of data and material: The datasets generated and/or analyzed during the current study are not publicly available because secondary analysis is now being conducted, but they are available from the corresponding author on reasonable request.* 

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#### 28ABSTRACT

29**Introduction:** dietary fat has been reported as one of the significant risk 30factors in the development of cardiovascular diseases (CVD).

31**Objective:** this study aimed at assessing the possible association 32between fat intake and CVD.

33**Methods:** the present case-control study was conducted in the center of 34coronary angiography. Three-hundred and ninety nine patients who

35referred for elective coronary angiography with clinical suspicion of 36coronary artery disease were enrolled. Dietary data were collected from 37each patient using an interview-based food frequency questionnaire.

38**Results:** the findings of the present study revealed no significant 39differences between cases and controls regarding the intake of all types of 40fat either before or after energy adjustment. For both cases and controls 41the percentage of fat intake from total energy and the intakes of 42polyunsaturated and monounsaturated fats, cholesterol, omega-6 and 43omega-3 were within the recommended amounts. The intake of all fat 44types (except trans-fat) was not associated with the risk of developing 45CVD. Trans-fat intake in the second and third quartile increased the risk of 46CVD by OR 1.86 (95% CI: 1.03-3.34) and 2.01 (95% CI: 1.12-3.60), 47respectively.

48**Conclusions:** while trans-fats may be significantly associated with the 49development of CVD in the first two quartiles, no association has been 50detected with other fat types.

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52**Keywords:** Saturated fat. Trans-fats. Cholesterol. Monounsaturated fats. 53Polyunsaturated fats and CVD.

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#### 55**RESUMEN**

56**Introducción:** se ha establecido que la grasa en la dieta es uno de los 57factores de riesgo significativos en el desarrollo de enfermedades 58cardiovasculares (ECV).

59**Objetivo:** este estudio tuvo como objetivo evaluar la posible asociación 60entre la ingesta de grasa y la ECV.

61**Métodos:** el presente estudio de casos y controles se realizó en el centro 62de la angiografía coronaria. Se inscribieron 399 pacientes que fueron 63remitidos para una angiografía coronaria electiva con sospecha clínica de 64enfermedad coronaria. Los datos dietéticos se obtuvieron de cada 65paciente mediante un cuestionario de frecuencia de alimentos basado en 66entrevistas.

67**Resultados:** los hallazgos del presente estudio no revelaron diferencias 68significativas entre los casos y los controles con respecto a la ingesta de 69todos los tipos de grasa, ya sea antes o después del ajuste de energía. 70Para ambos casos y controles, el porcentaje de ingesta de grasas de la 71energía total y las ingestas de grasas poliinsaturadas y monoinsaturadas, 72colesterol, omega-6 y omega-3 se encuentran dentro de las cantidades 73recomendadas. La ingesta de todos los tipos de grasa (excepto las grasas 74trans) no se asoció con el riesgo de desarrollar ECV. La ingesta de grasas 75trans en el segundo y tercer cuartil aumentó el riesgo de ECV en OR 1,86 76(IC 95%: 1,03-3,34) y 2,01 (IC 95%: 1,12-3,60), respectivamente.

77**Conclusiones:** si bien las grasas trans pueden estar asociadas 78significativamente con el desarrollo de ECV en los dos primeros cuartiles, 79no se ha detectado asociación con otros tipos de grasa.

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81**Palabras clave:** Grasa saturada. Grasas trans. Colesterol. Grasas 82monoinsaturadas. Grasas poliinsaturadas y CVD.

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#### 84INTRODUCTION

85Cardiovascular disease (CVD) is the first cause of death and disability 86worldwide (1). Unhealthy diet is a leading risk factor for CVD where 87several studies indicated that excessive consumption of saturated fatty 88acids (SFA) increases the low-density lipoprotein (LDL) cholesterol, which 89may enhance the risk of developing CVD (1-3). Eckel et al. (2014) reported 90strong evidence that reducing SFA intake to 5-6% of calories can be one 91important lifestyle modification for the management of CVD, mainly by 92lowering LDL cholesterol (4). However, several studies that evaluated the 93association of fat intake with CVD are controversial (5-8). Even though, for 94several decades, dietary guidelines have focused on the restriction of 95dietary cholesterol for heart health (5,6), numerous studies revealed that 96dietary cholesterol was not significantly associated with any coronary 97artery disease or ischemic stroke (7,8).

98Epidemiological studies which examined the effect of monounsaturated 99fatty acids (MUFA) on CVD have shown mixed results. The Prevención con 100Dieta Mediterránea (PREDIMED) study reported that diets higher in MUFA 101reduced CVD events as compared with lower fat diets and, as a result, 102lowered the incidence of coronary heart disease (CHD) risk (9). Moreover, 103the Mediterranean dietary pattern, rich in MUFA, was recognized for its 104beneficial effects on CHD risk reduction in which a strong negative 105association was observed among followers of this dietary pattern and CHD 106risk (10). On the contrary, two meta-analyses of cohort studies found no 107significant association between MUFA and CHD events or death (11,12). 108Several studies investigated the effect of polyunsaturated fatty acids 109(PUFA) and the risk of CVD and the obtained results were conflicting (13-11015). A meta-analysis of randomized controlled trials showed that the 111increase in PUFA intake actually reduced the risk of CHD death (14,16). 112However, some prospective cohort studies have shown that PUFA 113increased the risk of cardiovascular outcomes (13) or were not associated 114with risk (15).

115Consumption of trans-unsaturated fatty acids was associated with a 34% 116increase in all-cause mortality, 28% increased risk of CHD mortality, and 11721% increase in the risk of CHD (17). However, no associations were 118observed for ruminant trans-fat with CHD (17).

119This study aimed to explore the association between fat intake and the 120CVD risk in Jordan using a case-control design where a limited number of 121studies concerning risk factors for CVD has been published from the 122Middle East Countries. The concluded findings of this study would be of 123great importance for enhancing the public recognition about fat intake as 124a risk factor to develop CVDs. Those results could be used as a guidance 125to direct policy makers to initiate targeted nutritional and lifestyle 126strategies to prevent CVD events and alleviate their consequences. Also, 127those results could be used to establish more specified dietary guidelines 128regarding the intake of trans-fats and saturated fats for Jordanians.

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#### 130SUBJECTS AND METHODS

131Participants and study setting

132A case-control study was conducted to assess the association between fat 133intake and CVD risk among Jordanians. Participants of the present study 134were enrolled conveniently from the catheterization section of the 135Cardiology Department of Prince Hamzah Hospital, a referral hospital in 136the capital Amman, between January and December 2015. A total of 399 137 participants who underwent coronary angiography were included. The 138cases and controls were age and gender matched with 1:1 ratio. 139Participants with kidney disease, liver disease or gastrointestinal diseases 140were excluded. All participants were requested to sign a written consent 141 form to participate in the study. The study protocol was designed 142according to the ethical guidelines of the 1975 Declaration of Helsinki, and 143the study was approved by the Institutional Review Board Ethics 144Committee at Prince Hamzah Hospital. One day before undergoing 145coronary angiography, all data were collected from patients upon filling a 146standardized questionnaire by trained dietitians to record socio-147demographic factors, previous health issues (hypertension, diabetes 148mellitus, dyslipidaemia), smoking status, and family history of CVDs 149information.

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#### 151**Coronary angiography**

152Seldinger technique was used to insert a catheter by trained cardiologists 153into the radial artery, and the tip was advanced to the aortic sinus 154cusp. To visualize the arterial tree, X-ray images of the transient radio-155contrast distribution within the coronary arteries were carried out. The 156degree of obstruction was estimated as percentage of the arterial lumen 157by comparing the area of narrowing to an adjacent normal artery. 158Consistent with prior studies, CAD was defined as  $\geq$  20% stenosis of one 159or more coronary arteries (18,19). Participants with no stenosis (0%) were 160enrolled as controls.

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#### 162Nutrients intake assessment

163A validated Arabic quantitative food frequency questionnaire (FFQ) was 164used to assess the dietary intake pattern (20). The information dealing

165 with dietary history of participants was investigated in the FFQ questions. 166During face-to-face interviews, the participants were asked to record how 167 frequently, on average, they had consumed one standard serving of 168specific food items in nine categories (< 1/month, 2-3/month, 1-2/week, 3-1694/week, 5-6/week, 1/day, 2-3/day, 4-5/day, or 6/day) during the past year. 170Food lists in the modified FFQ questions were classified based on types of 171food: 21 items of fruits and juices; 21 items of vegetables; eight items of 172cereals; nine items of milk and dairy products; four items of beans; 16 173items of meat such as red meat (lamb and beef), chicken, fish, cold meat, 174and others; four items of soups and sauces; five items of drinks; nine 175items of snacks and sweets; and 14 items of herbs and spices. Food 176models and standard measuring tools were used for better estimation of 177portion size. Dietary analysis software (ESHA Food Processor SQL version 17810.1.1; ESHA, Salem, OR, USA) was used to analyze dietary intakes with 179additional data on foods consumed in Jordan. After entering the amounts 180which were consumed daily from the raw fats, foods containing fats, fried 181foods and other foods to the ESHA program, total amounts of different fats 182were added and calculated. Recipes for Jordanian foods were entered and 183the total intake from these recipes was calculated. Energy (kcal), energy 184 from fat (kcal), energy from saturated fat (kcal), energy from trans fatty 185acids (kcal), % of fat, fat (g), SFA (g), MUFA (g), PUFA (g), trans fat (g), 186cholesterol (mg), omega-3 (g), omega-6 (g), omega-3:omega-6, oleic 187(18:1) (g), linoleic (18:2) (g), lonolenic (18:3) (g), eicosen (20:1) (g), 188arachidon (20:4) (g), eicosapentaenoic acid (EPA) (20:5) (g), and 189docosapentaenoic acid (DPA) (22:5) (g) intake was assessed from the 190whole food items which are included in the used FFQ.

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#### 1927-day physical activity recall (PAR)

193A 7-day PAR validated questionnaire, which is an organized questionnaire, 194was used to calculate a participant's recall of time spent participating in 195exercise over a seven-day period (21). This questionnaire helps to divide 196individual physical activity levels into three categories. Participants were

197asked to respond to a PAR question based on the way they used to behave 198prior undergoing coronary angiography.

#### 199

### 200**Anthropometric measurements**

201All anthropometric measurements were carried out by a trained dietitian. 202Body weight was measured to the nearest 0.1 kg, with minimal clothing 203 and without shoes, using a calibrated scale (Seca<sup>®</sup>, Hamburg, Germany). 204Height and waist circumference were measured to the nearest 1 cm with 205participants in standing position without shoes using a calibrated portable 206measuring rod. Body mass index (BMI) was calculated as weight (kg) 207divided by height square (m<sup>2</sup>).

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#### 209**Statistical analysis**

210SPSS version 20.0 software (SPSS Inc., Chicago, IL, USA) was used to 211perform the statistical analysis. The significance level was set at  $p \le 0.05$ . 212Mean  $\pm$  standard error of mean (SEM) and percentages were used for 213 descriptive statistics. To evaluate the differences between cases and 214controls in continuous variables, t-tests were used, and Chi-squared was 215used to detect the differences among categorical variables. Potential 216confounders (age, gender, BMI, smoking, physical activity, total energy 217 intake, occupation, education level, marital status and family history) were 218chosen based on reported risk factors for CVDs. The quartiles were 219calculated using the cut-off points at 25, 50 and 75% of total nutrients 220intake. The first quartile was determined if the intake was below 25%, 221 while the second one was determined if the intake was between 25-50%. 222The third guartile was between 50-75% and the fourth was above 75%. 223Multinomial logistic regression model and linear logistic regression model 224were used to calculate odd ratios (OR) and its 95% confidence interval (CI) 225and p-for-trend for trend, respectively. The energy adjustment was 226performed using the residual method of Willett in which residuals were 227computed from a regression analysis (22).

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229**RESULTS** 

230Briefly, 239 males and 160 females participated in this study. The study 231participants' characteristics are shown in table 1 and have been as 232mentioned elsewhere (23). The main characteristics of study subjects 233categorized by gender are summarized in table 1. The cases had higher 234mean fasting blood glucose levels compared to controls. Moreover, cases 235showed higher blood triglyceride levels compared to controls. In addition, 236there were differences in physical activity measured as MET (min/week). 237Overall, the cases were less active compared to controls, and reported 238more previous health problems than controls, in both men and women.

239Table 2 reveals that no significant difference was detected between cases 240and controls in all types of fat intake either before or after energy 241adjustment. Also, the percentage of fat intake from total energy was 242within the recommended level. However, the amount of saturated fat 243(around 30 g; 9.0%) was close to the amount of MUFA (around 33 g; 24410.5%) which is not consistent with a healthy diet. On the other hand, the 245intake of PUFA (19 g; 6.0%) was lower than both saturated and MUFA. The 246consumption of cholesterol was below the recommended amount (intake: 247255 mg *vs* recommended: 300 mg). Oleic fatty acid was the prominent 248type of fat among the cases and controls. Additionally, the intake of 2490mega-6 and omega-3 among cases and controls was in agreement with 250the recommended amounts.

251Table 3 shows the crude and adjusted ORs and their 95% CI for CVD by fat 252types quartiles. OR and their 95% CI for fat types were adjusted for age, 253gender, BMI, smoking, and physical activity. The intake of all fat types 254(except trans-fat) was not associated with the risk of developing CVD. 255Trans-fat intake in the second and third quartile increased the risk of CVD 256by adjusted for about 1.86 (95% CI: 1.03-3.34) and 2.01 odds (95% CI: 2571.12-3.60), respectively. Similar results of the association between trans-258fat and CVD obtained was obtained for the crude OR.

## 260**DISCUSSION**

261This study aimed at evaluating the association between the intakes of 262different fat types and the development of CVD among Jordanians. Due to

263the discrepancy in the findings of multiple studies, the research is still 264 unclear to judge if there is really a positive association between SFA and 265CVD, as traditionally speculated. The main findings of this study did not 266support the results of many other studies which stated that total dietary 267 fats, saturated fats and cholesterol were positively associated with the risk 268of developing CHD (12,24,25). However, our findings came in agreement 269 with several other studies (17,25,26). De Souza et al. (2015) reported null 270 associations between saturated fat intake and all-cause mortality (relative 271risk 0.99, 95% CI: 0.91 to 1.09), CVD mortality (0.97, 0.84 to 1.12), total 272CHD (1.06, 0.95 to 1.17), ischemic stroke (1.02, 0.90 to 1.15), and type 2 273 diabetes (0.95, 0.88 to 1.03) (17). Siri-Tarino et al. (2010) illustrated that 274the intake of saturated fat was not associated with an increased risk of 275CHD, stroke, or CVD; the pooled relative risk estimates that compared 276 extreme quantiles of saturated fat intake were 1.07 (95% CI: 0.96, 1.19; p 277 = 0.22) for CHD, 0.81 (95% CI: 0.62, 1.05; p = 0.11) for stroke, and 1.00 278(95% CI: 0.89, 1.11; p = 0.95) for CVD (25). Additionally, Harcombe et al. 279(2016) revealed that none of the studies included in their meta-analysis 280 found a significant relationship between CHD deaths and total dietary fat 281intake (26). Factors such as food matrix, source of saturated fat, and fatty 282acid chain length may influence the health effects of saturated fats and 283therefore, might explain the contradiction in the current evidence 284 regarding their association with CVD (27,28).

285Although the 2015-2020 Dietary Guidelines for Americans recommend 286substituting both MUFA and PUFA for saturated fats, the consistency in the 287current evidence is lacking (27,28). Higher intakes of PUFA were found to 288be significantly associated with a lower risk of CHD comparing the highest 289and lowest quintile for PUFAs 0.80, (0.73 to 0.88; p-trend < 0.0001) (29). 290On contrary, Chowdury et al. (2014) reported no relationship between 291dietary PUFA and coronary disease, with a risk ratio 0.98 (CI: 0.90 to 1.06) 292in eight cohort studies containing 206,376 participants with 8,155 events 293(12). The proportions among daily intake of different types of fat might 294provide a partial explanation for the inconsistent evidence concerning the 295association of unsaturated fats with CVD (30,31); the optimal balance 296among daily intake of different fatty acids can effectively improve the 297health while the incorrect ratio may increase disease risk (30,31). Here, no 298significant association was detected between the intake of PUFA, MUFA, 299omega-6 or omega-3 and CVD risk among cases and controls. Two main 300reasons might have contributed to the null findings; firstly, our study 301findings revealed that the intake of PUFA (around 19 g/day; 6.0%), MUFA 302(around 32 g/day; 10.5%), omega-6 fatty acid (16 g/day) and omega-3 303fatty acid (1.1 g/day) was similar in cases and controls. Secondly, all of 304these types of fat were consumed in approximately the recommended 305doses (32).

306Trans-fat intake was found to be significantly associated with CVD among 307Jordanians, which is consistent with many studies (12,33). Trans-fats from 308 foods may adversely affect the risk of coronary disease by raising LDL 309cholesterol levels and lowering high-density lipoprotein (HDL) cholesterol 310levels (34), increasing Lp(a) lipoprotein levels (34), raising triglyceride 311 levels (34), and interfering with essential-fatty acid metabolism (35). 312Trans-fats were associated with all-cause mortality, total CHD, and CHD 313mortality, probably because of higher levels of intake of industrial trans-314 fats than of ruminant trans-fats (17). Industrial and ruminant trans-fats 315consist of the same positional trans isomers, but in different proportions. 316The isomer profile depends on conditions of hydrogenation, such as 317catalysts used and temperature of hydrogenation for industrial trans-fats 318and rumen pH, and the composition of oils in the diet for ruminant trans-319 fatty acids (36). Chowdhury et al. (2014) revealed in their meta-analysis 320that the intakes of SFA, MUFA, alpha-linoleic acid, long-chain omega-3 or 3210mega-6 fatty acids were not associated with coronary disease (12). 322However, they found that trans-fats increased the incidence of coronary 323disease (RR 1.16, 95% CI: 1.06 to 1.27) (12). Li et al. (2015) studied 32484,628 women (Nurses' Health Study, 1980 to 2010) and 42,908 men 325(Health Professionals Follow-up Study, 1986 to 2010) who were free of 326diabetes, cardiovascular disease, and cancer at baseline, and found that 327trans-fat intake was significantly associated with an increased risk of CHD 328(HR: 1.20, 95% CI: 1.09 to 1.32; p-trend = 0.002) (29). It has been

329estimated that the consumption of about 5 g of trans-fat per day is 330associated with 25% increase in the risk of CHD (29). Although the 331association between CVD and trans-fat appears to be causal, no 332randomized controlled trial with hard endpoints has been reported (37).

333Regarding cholesterol findings, our results showed no significant 334association between the intakes of cholesterol and CVD risk, with 335insignificant difference in the mean intake of cholesterol for cases 336compared to controls. McNamara (2000) demonstrated in his review that 337the analysis of the available epidemiological and clinical data indicated 338that, for the general population, dietary cholesterol makes no significant 339contribution to atherosclerosis and risk of cardiovascular disease (38). A 340recent study of Rhee et al. (2017) performed on 30,068 participants 341(mean age 40.8 years; 84.5% men) in a health screening program in Korea 342documented that dietary cholesterol intake did not show any association 343 with LDL level or with risk for coronary artery calcification in apparently 344healthy Korean adults (39). The Scientific Report of the 2015 Dietary 345Guidelines Advisory Committee (DGAC) in the United States concluded 346that "cholesterol is not a nutrient of concern for overconsumption", 347suggesting that there no longer be a recommended upper limit for dietary 348cholesterol intake (40). This conclusion came after decades of the 349 recommendation of 300 mg/d as the upper limit for dietary cholesterol. 350Despite eliminating the upper limit from the Dietary Guidelines, 351 individuals should eat as little dietary cholesterol as possible as part of 352their healthy eating pattern to hinder CVD risk. Therefore, the lack of 353 association between the dietary cholesterol and CVD in the current study 354could be due to the considerable low daily consumption of cholesterol (< 355300 mg/day) for both cases and controls.

356The main strength points of this study are the use of a validated Arabic 357FFQ that was modified to reflect the food consumption pattern in Arab 358countries, especially Jordan, as well as the use of food models and 359measuring tools to estimate portion sizes. There are limitations in this 360study; for example, the one year dietary recall period may not be an 361accurate amount of time in which to conclude that an association exists 362between fat intake and CVD development. Nevertheless, we believe that 363the recall period of one year is very likely reflective of the previous years. 364Thus, the association between fat dietary intake and CVD may have been 365developing for several years.

366In conclusion, no association has been found between cholesterol, 367saturated fats, PUFA and MUFA and the risk of CVD. On the contrary, a 368significant association has been detected between trans-fats and the 369development of CVD. This may be attributed to the fact that the intake of 370most of these fat types (except trans-fats) are within the recommended 371percentages and amounts.

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#### **373ETHICS APPROVAL**

374The study was approved by the IRB at Prince Hamza Hospital, and all 375participants gave written consent to participate in the study.

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#### 377**ACKNOWLEDGEMENT**

378We thank the patients who participated in the study, and the hospital 379management for facilitating the data collection and all other aspects of 380this research.

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# **Table 1. General characteristics of study participants based on** 538**gender**

	Male (n = 2	39)	Female ( $n = 160$ )		
Variable (mean ± SEM)	Cases	Controls	Cases	Controls	
	(n = 132)	(n = 107)	(n = 73)	(n = 87)	
Age (y)	48.1 ± 0.5	48.4 ± 1.0	61.1 ± 1.2	54.1 ± 1.0	
BMI (kg/m²)	29.9 ± 0.69	29.9 ± 0.56	31.9 ± 0.67	31.9 ± 0.50	
Physical activity	11,289.5	13,258.9 ±	7,539.9 ±	10,312.8 ±	
(MET/min)	± 765.4 752.7 493.		493.1	682.8	
Systolic blood pressure	130.8 ±	120 1 + 2 2	142.9 ±	$140.6 \pm$	
(mmHg)	2.4	120.4 ± 2.5	26.3	23.9	
Diastolic blood pressure	75.7 ±	70 0 ± 1 6	81.2 ±	005 + 25	
(mmHg)	0.98	70.0 ± 1.0	1.7	00.J ± 2.J	
	109.8 ±	$109.7 \pm 0.5$	$110.0 \pm$	131.1 ±	
	10.2	100.7 ± 9.5	11.1	17.2	
	38.8 ±	20 5 ± 1 4	47.2 ±	48.40 ±	
	2.78	$59.5 \pm 1.4$	3.4	2.09	
Triglycerides (mg/dl)	288.3 ±	$163.1 \pm 16.1$	260.4 ±	176.3 ±	

	45.8		47.9	14.4			
	208.4 ±	100 1 7 0	189.3 ±				
Cholesterol (mg/dl)	16.8	$186.4 \pm 7.9$	97	$215.0 \pm 8.9$			
Fasting blood glucose	10.0		105+				
	$8.7 \pm 0.48$ 6.8 ± 0.31		10.5 ±	7.7 ± 0.42			
(mmol/l)			0.75				
Variable n (%)							
Marital status	<b>i</b>	ì	i				
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							
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)			
BMI							
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)			
Physical activity categori	es		•				
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							
	89 (67.4)	90 (84.1)	41 (56.2)	72 (82.8)			
Smoking	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	<b>.</b>		1				
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)			
Family 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)			
540							

541Significant difference was set at p < 0.05. SEM: standard error of mean; BMI:\*</td>542body mass index; MET: metabolic equivalent-minutes; LDL: low-density lipoprotein;543.HDL: high-density lipoprotein; CAD: coronary artery disease

Table 2. Fat and type of intake of cases and controls before and a	fter the ad
intake	

Intance						
	Controls (n =	Cases (n =		Controls (n =	Cases (n =	
	194)	205)	p-	194)	205)	
p-value	Adjusted for en	ergy	value	Crude		
	Mean + SEM			Mean + SFM		
_		_	0.240	7/6 + 2.01/1	+ 2 705 /	
-	-	-	0.240	74.0 ± 2,914.1	<u> </u>	
					68.1	
-	-	-	0.438	29.0 ± 964.2	± 933.9	
					26.2	
-	-	-	0.719	0.49 ± 33.0	±0.49 33.2	
			0.853	9.1 ± 0.20	9.2 ± 0.19	
			0.540	$10.6 \pm 0.24$	$10.4 \pm 0.23$	
		/	0.585	$6.1 \pm 0.14$	$6.2 \pm 0.14$	
			0.970	0.17 ± 0.04	$0.17 \pm 0.04$	
0.110	$1.5 \pm 107.6$	$1.4 \pm 104.2$	0.438	3.2 ± 107.6	2.9 ± 104.2	
0.078	0.67 ± 30.5	0.56 ± 28.9	0.302	1.2 30.5±	0.94 ± 28.9	
0.878	7.4 ± 34.1	0.67 ± 30.5	0.316	$1.1 \pm 34.0$	$1.1 \pm 32.5$	
0.648	0.47 ± 19.4	$0.41 \pm 19.1$	0.738	0.63 ± 19.4	0.58 ± 19.2	
0.897	0.14 ± 0.57	$0.12 \pm 0.55$	0.897	0.14 ± 0.57	$0.12 \pm 0.55$	
	10.2 ± 252.7	± 263.9			± 263.9	
0.469		11 /	0.553	$13.4 \pm 252.7$	12.2	
0.310	0.01 + 1.1	11.4	0 310	0.01 + 1.1	13.2	
0.319	$0.04 \pm 1.1$	$0.05 \pm 1.1$	0.319	$0.04 \pm 1.1$	$0.03 \pm 1.1$	
0.045	$0.44 \pm 10.7$	+0.002.0.07	0.079	$0.30 \pm 10.7$	$0.33 \pm 10.0$	
0.440	$0.002 \pm 0.07$ 0.72 + 31.8	10.0020.07	0.440	$1.0 \pm 31.8$	$1.0 \pm 30.4$	
0.130	$0.72 \pm 51.0$	$0.70 \pm 30.4$	0.510	$1.0 \pm 51.0$	$1.0 \pm 30.4$	
0.020	$0.44 \pm 10.0$	$0.40 \pm 10.3$	0.004	$0.30 \pm 10.0$	$0.03 \pm 0.08$	
0.207	$+0.03 \pm 1.0$	$+0.03 \pm 0.90$	0.207	$0.05 \pm 1.0$	$0.03 \pm 0.90$	
0.714	-0.01 + 0.17	10.01 + 0.10	0.714	$0.01 \pm 0.17$	$0.01 \pm 0.10$	
0.202	$0.01 \pm 0.15$	$0.01 \pm 0.14$	0.202	$0.01 \pm 0.15$	$0.01 \pm 0.14$	
0.828	0.002 + 0.02	$\pm 0.02$	0.828	0.00 + 0.02	+0.000.02	
		0.002				
0.604	0 001 + 0 01	$\pm 0.01$	0.604		$\pm 0.01$	
0.004	$0.001 \pm 0.01$	0.001	0.004	$0.001 \pm 0.01$	0.001	

Table 3. The OR (95% CI) for nutrient intake among Jordanian					
participants					
Q4	Q3	Q2	*Q1	Nutrients	
Fat (g)					
47	45	56	57	Cases number	
53	55	44	42	Controls number	
(0.44-1.45) 0.80	0.32-) 0.57	0.50-) 0.89	1	Adjusted OR <sup>†</sup>	

	(1.03	(1.60		(95% CI)		
0.37-) 0.65	0.34-) 0.60	0.54-) 0.94	1	Crude OR (95%		
(1.14	(1.06	(1.64		CI)		
0.358				p-for-trend		
Saturated fat (g)						
46	48	57	54	Cases number		
53	53	43	45	Controls number		
	0.43-) 0.77	0.60-) 1.07		Adjusted OR <sup>+</sup>		
(0.49-1.64) 0.90	(1.37	(1.93		(95% CI)		
0.41-) 0.72	0.43-) 0.75	0.63-) 1.10	1	Crude OR (95%		
(1.27	(1.32	(1.93	L	CI)		
0.516	1		$\sim$	p-for-trend		
Monounsaturateo	l fat (g)		/			
47	51	56	51	Cases number		
52	49	45	48	Controls number		
	0.47-) 0.85	0.60-) 1.08	-	Adjusted OR <sup>+</sup>		
(0.43-1.43) 0.78	(1 54	(1.94	1	(95% CI)		
0.49-) 0.85	0.56-) 0.98	0.67-) 1.17	12.7	Crude OR (95%		
(1 49	(1 71	(2.04	1	CI)		
0 736		(2.04		n-for-trend		
Polyunsaturated i	fat (a)		/			
54	44	56	51	Cases number		
46	55	44	49	Controls number		
10	0.51-) 0.91	0.73-) 1.31		Adjusted OR <sup>†</sup>		
(0.67-2.13) 1.19	(1.64	(2.22	1			
0.65 \ 1.13	(1.04)			(95% CI) Crude OR (05%		
0.05-) 1.15	0.44-) 0.77	0.70-) 1.22	1	CIULE OR (95%		
(1.97	(1.34	(2.13		CI)		
0.361				p-for-trend		
Trans-fat (g)			45			
49	57	54	45	Cases number		
49	42	42	61			
(0.77-2.48) 1.38	1.12-) 2.01	1.05-) 1.80	1	Adjusted OK		
	(3.60	(3.34		(95% CI)		
0.78-) 1.36	1.06-) 1.84	1.00-) 1.74	1	Crude OR (95%		
(2.36	(3.20	(3.04	L	CI)		
0.311 p-for-trend						
Cholesterol (mg)						
51	47	57	50	Cases number		
48	54	43	49	Controls number		
(0 56-1 86) 1 02	0.50-) 0.90	0.83-) 1.50	1	Adjusted OK '		
	(1.62	(2.69	-	(95% CI)		

	0.60-) 1.04	0.49-) 0.85	0.74-) 1.30	1	Crude OR (95%
	(1.82	(1.49	(2.27	T	CI)
	0.545	•			p-for-trend
54	5				

546\*Reference quartiles. <sup>†</sup>Adjusted for age, gender, BMI, smoking, physical 547activity, total energy intake, education level and family history.