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Original article

Association Between Nutrients Intake and Coronary Heart Disease Among Adults in Saudi Arabia: A Case-Control Study

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Abstract. *Background and Objective***:** Coronary heart disease (CHD) is a leading cause of mortality worldwide. Increasing evidence links CHD to the dietary intake among different populations. This paper aims to examine the association between nutrients intake and CHD in Saudi Arabia. *Methods and Study Design***:** A case-control study was conducted in western Saudi Arabia. An interview-administered questionnaire was performed to collect data on socio-demographic characteristics and family histories of the disease. All participants completed three consecutive daily food records. Fasting blood samples were collected to measure glucose and total cholesterol. Body weight, height, and blood pressure measurements were also recorded. *Results:* A total of 85 patients were included in this study. Case participants had significantly higher intakes of monounsaturated fatty acids and polyunsaturated fatty acids but lower intakes of total energy, saturated fatty acids, and *trans* fatty acids than did controls. In addition, case participants consumed significantly fewer carbohydrates and less calcium, sodium, and zinc than did controls. The excess intake of total energy, *trans* fatty acid, and sodium associated with an increased risk of CHD. Also, deficient intake of polyunsaturated fatty acids, protein, zinc, vitamin A, vitamin C, and alpha-linolenic acid associated with an increased risk of CHD among participants. *Conclusions:* The current study's findings provide appropriate nutritional solutions to prevent and control the incidence of CHD in Saudi Arabia. Further studies with larger sample size are essential to confirm these findings.

Key Words: coronary heart disease, dietary intake, nutrients, fatty acids, omega 3, Saudi Arabia.

Introduction

Cardiovascular diseases (CVDs) are a group of diseases that affect the heart and blood vessels. One main type of CVD is coronary heart disease (CHD), which results from atherosclerosis (1). Other CVDs include several diseases, such as rheumatic heart disease and heart failure (2, 3). CVD is a serious public health issue in Saudi Arabia (4, 5). The World Health Organization (WHO) estimates that CVD results in 17 million deaths globally each year (4). The global death rate of CVD is predicted to increase by 2030, and CVD will remain the leading cause of death (6). Over the last four decades, Gulf Cooperation Council (GCC) countries, including Saudi Arabia, have undergone important economic development that has led to the majority of the population adopting a modern lifestyle; thus, the incidence of CHD and associated risk factors have increased among the Gulf population (7). The prevalence of CHD in the Saudi population was reported to be 5.5% based on a national representative survey (8). Statistics collected in Saudi Arabia over the past 40 years indicate a large increase in deaths from CVD-related conditions, including CHD and stroke (9). The country's Ministry of Health reported CVD as the second most common cause of death in

2018, with 6,372 deaths (14.2% of the total number of deaths 44,783) (10).

Diet is one key modifiable risk factor for CHD (11-14). Studies show that the risk of CHD can be reduced through a diet rich in fruits, vegetables, whole grains (as the main source of carbohydrates), and dietary fat mainly from nonhydrogenated unsaturated fats, along with sufficient omega-3 fatty acids (15). The key protective components in whole grains, fruit, and vegetables are fibres, vitamins, and minerals that have a functional role in reducing blood pressure, oxidative stress, and inflammation and improving the lipoprotein profile and insulin sensitivity (16-18). On the other hand, diets high in saturated fats and refined and processed carbohydrates are associated with an elevated risk of CVD due to higher levels of blood glucose, total cholesterol, and low-density lipoprotein (LDL) cholesterol (15, 19). In addition, a diet with a variety of nutrients can provide synergistic and antagonistic effects for optimal health (20). For example, clinical and epidemiological studies indicate that omega-3 fatty acids significantly contribute to the prevention of CHD (21-23). Previous interventional studies have shown that a diet rich in omega-3 fatty acids, particularly alpha linolenic acid (ALA), reduces cardiac disease. Therefore, the ratio of saturated fatty acids (SFA) to polyunsaturated fatty acids (PUFA) is essential to determining diet healthfulness (21-23).

The majority of scientific evidence regarding nutritional risk factors and CHD is mainly derived from the developed world. In Saudi Arabia, research investigating the relationship between dietary intake and CHD risk is limited (17, 24). Research that gathers and compares relevant information about the health effects of dietary intake will aid in the implementation of more efficient preventive measures to minimize the public's risk of CHD in Saudi Arabia (24-26). Considering the potential for the increased prevalence of CHD and associated risk factors in the Saudi population, a detailed investigation of the effect of nutritional risk factors on increased CHD risks within the country is necessary. To our knowledge, this is the first study examining the association between the intake of fatty acids and CHD in Saudi Arabia. Thus, the current study aims to assess the dietary intake of total energy, macronutrients, and other selected nutrients among the population of Saudi Arabia and to examine the association between nutrient intake and the risk of CHD in Saudi Arabia.

Materials and Methods

Study design and participants

The study design, participants, and data collection have been detailed elsewhere (27). Briefly, the study participants were recruited from three hospitals in the Western region of Saudi Arabia: the King Abdullah Medical City and Tunsi private hospitals in Makkah city, and the King Abdul Aziz University Hospital in Jeddah city. Data collection occurred between May and October 2015. The study sample comprised 325 participants; however, only 85 participants filled in the three-day food record. Hence, 85 participants were included in the current study: 40 cases (participants with CHD) and 45 controls (participants without CHD). Participants with CHD were patients previously diagnosed with clinical artery disease, myocardial infarction, or chronic stable angina or patients who had their first acute event at King Abdullah Medical City. Participants without CHD came from outpatient clinics, including family medicine clinics, nose and throat clinics in Tunsi private hospital, and ophthalmology clinics in King Abdulaziz University Hospital. They were matched by age and gender with cases, and they had no history of CVD. Written informed consent forms were signed by eligible subjects before participating in the study. The study protocol conformed to ethical guidelines of the 1975 Declaration of Helsinki, and the study was approved by the Griffith University Human Research Ethics Committee (GU Ref No: MED/59/14/HREC), the Research Ethics Committee at King Abdulaziz University Hospital (Reference No ll8-15), and the Institutional Review Board at King Abdullah Medical City (IRB No: 15-194).

Data collection

An interview-administered questionnaire was undertaken for all study participants. Data regarding socio-demographics, family CVD history, and

behavioural risk factors were collected during the interview. In addition, blood pressure and anthropometric measurements were completed for all participants using standard equipment. Blood samples were collected to assess fasting plasma glucose, total cholesterol, and the serum levels of 25 hydroxy vitamin D for participants.

Nutrients intake assessment

Three-day food records were used to collect dietary intake data from study participants. Participants recorded food consumption on three consecutive days, two weekdays, and one weekend day. During the three-day period, subjects were asked to always carry the food diary with them and to record food and beverage consumption at the time of consumption. Participants were asked to provide as much detail as possible, specifying the types and brands of foods as well as the cooking and preparation methods. Furthermore, participants were asked to estimate and record all food and drink consumed using household measurements (e.g. cups, plates, table, and spoons). These records were validated during the interview by the researcher using a photographic food atlas (28). Food intake was converted to nutrient intake and averaged for the three days using WinDiets (Robert Gordon's University Aberdeen). McCance and Widdowson food tables were used for Western foods (29). For Saudi foods not in the available food tables, Saudi recipes were consulted to estimate the amount of each food whilst considering the portions suggested by the recipe. The percentages of energy from protein, carbohydrates, total fat, SFA, *trans* fatty acid, monounsaturated fatty acids (MUFA), and PUFA were calculated for each day. Dietary recommendations for the Saudi population are yet to be established, so the UK Dietary Reference Intake (DRI) level was used (30) to compare the intake of food energy and nutrients with recommendations (31).

Statistical analysis

All data were analysed using SPSS version 26 computer software (SPSS Inc, Chicago, IL, USA). Data were represented as means ± standard deviation (SD). A *t*-test was used to evaluate differences between cases and controls in continuous variables. Multivariate logistic regression analyses were conducted to examine the association between nutrients intake (including total energy, fat, protein, carbohydrates, fibre, calcium, iron, magnesium, sodium, potassium, selenium, zinc, vitamin A, and vitamin C) and the risk of CHD. In addition, the association between the intake of fatty acids (including SFA, MUFA, PUFA [n-3 PUFAs (ALA) and n-6 PUFAs (LA)] and *trans* fatty acids) and the risk of CHD was also examined via multivariate logistic regression analyses. The regression analysis was performed using three models with adjustments for potential confounders. The second and third models of the regression analysis included the following confounding variables: age, gender, education level, employment, marital status, family history of CVD, smoking, exercise, BMI, fasting plasma glucose, total cholesterol, and vitamin D. The findings of a multivariate logistic regression were stated as odds ratios (OR) with 95% confidence intervals (CI). A $p<0.05$ value was considered significant.

Results

Table 1 presents the general characteristics of 40 cases and 45 controls. CHD cases were more likely to be married ($p<0.005$) and to be cigarette smokers ($p<0.001$) than were control participants. However, controls were more highly educated ($p< 0.001$) and more often employed ($p<0.001$) than were participants in CHD group.

The comparison of nutrients intake among case and control groups and the nutrients recommendations appear in Table 2. Marked differences exist in macronutrient and micronutrient compositions between the case and control participants. Controls reported higher averages of total energy intake than did cases ($p<0.001$). Energy provided by SFA was significantly higher in controls (14.6%) than in cases (12.4%) ($p<0.001$). Likewise, energy provided by *trans* fatty acids was higher in controls (1.5%) than in cases (1.2%) ($p<0.05$). However, the percentages of energy provided by MUFA and PUFA were significantly higher in cases than in controls: 12.1% for MUFA and

Table 1. General characteristics of study participants

Variables	Control (*n*=45) *n*(%)	CHD (*n*=40) *n*(%)	*p*-value
Age (years)			
<49	9 (20)	4 (10)	0.201
≥49	36 (80)	36 (90)	
Gender			
Men	15 (33)	16 (40)	0.524
Women	30 (67)	24 (60)	
Marital status			
Single	13 (30)	1 (2)	0.005
Married	22 (48)	26 (65)	
Divorced/Widowed	10 (22)	13 (33)	
Education			
Up to primary levels	5 (11)	22 (55)	<0.001
High School and bachelor or diploma degree	40 (89)	18 (45)	
Employment			
Employed (Full time, Part time, self-employed)	34 (76)	11 (28)	<0.001
Unemployed (Student, Retired, Housewife)	11 (24)	29 (72)	
Family income (SR†/monthly)			
<5000	13 (29)	16 (40)	0.029
5000-15000	31 (69)	18 (45)	
15000-25000	1 (2)	6 (15)	
Smoke cigarettes			
Current <20 cigarettes/day	3 (7)	9 (22)	<0.001
Previous smoker	1 (2)	10 (25)	
Non-smoker	41 (91)	21 (53)	
Water pipe smoker			
Yes	6 (13)	1 (2)	0.293
No	39 (87)	39 (98)	
Exercise			
Never and rarely	21 (47)	14 (35)	0.301
1–2 times/week	10 (22)	7 (18)	
More than 3–4 times/week	14 (31)	19 (47)	
Family history of CVD			
Yes	27 (60)	22 (55)	0.641
No	18 (40)	18 (45)	

p-value based on X^2-test; †Saudi Riyal (1SR = 0.37 AUD).

6.9% from PUFA in cases (p<0.001) and 9.1% from MUFA and 3.3% from PUFA in controls (p<0.001). The percentage of energy provided by carbohydrates was higher in controls (54%) than in cases (p<0.05). Moreover, the mean intake of protein and carbohydrates was significantly higher in controls than in cases: 89.2 g/protein/day and 306.3 g/carbohydrates/day (p<0.015) and 79.2 g/protein/day and 250.7 g/

Table 2. Comparison of nutrients intake among case and control groups and nutrients recommendations.

Nutrients	Control (*n*=45) Mean ± SD	CHD (*n*=40) Mean ± SD	DRV†		*p*-value
			Males	Females	
Energy **(kcal/d)**	2270±232.7	1971±301.26	2550	1940 1900‡	<0.001
Total fat **(g/d)**	85.05±17.24	78.46±20.84	33	33 0.135	0.114
% of daily energy	35.6	33.6			
SFA **(g/d)**	37.12±10.15	27.36±10.68	10	10 0.007	<0.001
% of daily energy	14.6	12.4			
MUFA **(g/d)**	22.9±7.51	26.6±8.13	12	12 <0.001	0.032
% of daily energy	9.1	12.1			
PUFA **(g/d)**	8.5±2.84	15.3±5.59	6	6 <0.001	<0.001
% of daily energy	3.3	6.9			
trans fatty acid **(g/d)**	3.8±2.15	2.6±1.53	2	2 0.050	0.003
% of daily energy	1.5	1.2			
Protein **(g/d)**	89.2±18.12	79.2±18.76	55.5 53.3‡	45 46.5‡ 0.651	0.015
% of daily energy	15.7	16.1			
Carbohydrates**(g/d)**	306.3±44	250.7±49.31	50	50 0.050	<0.001
% of daily energy	54	51			
Fibre **(g/d)**	6.6±2.97	5.4±2.54	12	12	0.393
Calcium **(mg/d)**	835.5±213.1	703.2±240.8	700	700	0.009
Iron **(mg/d)**	15.6±5.41	17.4±10.1	8.7	14.8 8.7‡	0.005
Magnesium **(mg/d)**	271.4±59.72	259.1±118.7	300	270	0.539
Sodium **(g/d)**	4.2±1.1	3.3±1.4	1.60	1.60	0.005
Potassium **(g/d)**	3.2±1.4	2.9±1.9	3.50	3.50	0.201
Selenium **(µg/d)**	48.4±16.10	45.1±28.28	75	60	0.502
Zinc **(mg/d)**	10.3±2.43	8.03±2.43	9.5	7.0	<0.001
Vitamin A **(µg/d)**	535.5±227.20	980.1±119.82	700	600	0.006
Vitamin C **(mg/d)**	57.54±38.66	72.6±53.3	40	40	0.135
Vitamin E **(mg/d)**	4.61±2.22	5.82±2.19	15	15	0.014

SFA indicates saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids. DRV, dietary reference values for food energy and nutrients †19-50 y, ‡50+ y.[27] *p*-value based on *t* test.

carbohydrates/day ($p<0.001$), respectively. No significant difference appeared in the total fat intake between cases and controls (Table 2).

Regarding micronutrients intake, control participants had significantly higher dietary intakes of calcium ($p<0.009$), sodium ($p<0.05$), and zinc ($p<0.001$) than did cases. On the other hand, cases had higher intakes of iron ($p<0.005$), vitamin A ($p<0.006$), and vitamin E ($p<0.014$) compared to control participants. There was no significant difference in the intake of fibre, magnesium, potassium, selenium, and vitamin C between the two groups (Table 2).

Findings from comparing the intake of omega-3 and omega-6 fatty acids among study participants and the fatty acids recommendations are shown in Table 3. The intake of total omega-3 fatty acid by

Table 3. Comparison of the intake of types of omega 3 and 6 fatty acids among case and control groups and nutrients recommendations.

Nutrients	Control (*n*=45) Mean ± SD	CHD (*n*=40) Mean ± SD	AI		*p*-value
			Males	Females	
Total n-3 PUFAs (g/d)	0.37±0.22	1.87±0.15			<0.001
% of daily energy†	0.15±0.02	0.61±0.53	0.2	0.2	<0.001
ALA (g/d)‡	0.36±0.12	1.28±0.21	1.6	1.1	<0.001
EPA (g/d)‡	0.004±0.0001	0.13±0.003	0.45	0.45	0.016
DHA (g/d)‡	0.001±0.0001	0.14±0.13			0.004
Total n-6 PUFAs (g/d)	2.89±0.01	3.77±0.29			0.138
% of daily energy†	1.14± 0.77	1.68±0.40	1	1	0.030
LA (g/d)‡	2.87±0.12	3.66±0.20	17	12	0.169
AA (g/d)	0.023±0.010	0.11±0.10	N/A	N/A	0.001

n-3, omega 3; ALA, alpha-linolenic acid (18:3 n-3); EPA, eicosapentaenoic acid (20:5 n-3); DHA, docosahexaenoic acid (22:6 n-3); n-6, omega 6; LA, Linoleic acid (18:2 n-6); AA, arachidonic acid (20:4 n-6). †Reference Nutrient intake 19-50 y.[27] ‡Dietary Reference Intake.[28] N/A, not applicable. AI, adequate intake. *p*-value based on *t* test.

CHD participants was over twice the amount compared to controls ($p<0.05$). The mean intake of total omega-3 fatty acids was higher for case subjects (1.87 g/day) than for controls (0.37 g/day), mainly in the form of ALA (1.28 g/day for the cases and 0.36 g/day for the control; $p<0.001$). The intake of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) was also higher in cases than in controls: 0.13 g/EPA/day and 0.14 g/DHA/day ($p<0.016$) and 0.004 g/EPA/day and 0.001 g/DHA/day ($p<0.004$), respectively. On the other hand, no significant difference appeared in the mean intake of total omega-6 fatty acid between the two groups, except for the form of arachidonic acid (AA), was significantly higher in cases than in controls: 0.11 g/day and 0.023 g/day ($p<0.001$), respectively.

Multivariate logistic regression analyses findings for case and control groups are shown in Table 4. After adjusting for age, gender, education level, employment, marital status, family history of CVD, smoking,

Table 4. The association between nutrients intake and the risk of CHD among study participants.

Nutrients†	Crude OR[1] (95% CI)	Adjusted OR[2] (95% CI)	Adjusted OR[3] (95% CI)
Energy (kcal/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Excess	1.6 (1.31,19.16)**	1.95 (1.13,14.2)**	2.12 (0.99,13.09)**
Total fat (%)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Excess	0.55 (0.24, 1.36)	0.38 (0.28, 4.60)	0.50 (0.31, 5.58)
SFA (%)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Excess	1.19 (0.84, 6.01)**	1.58 (1.11, 21.3)**	2.31 (1.47, 6.91)**
MUFA (%)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	0.89 (0.14, 0.98)	0.43 (0.16, 2.46)	0.49 (0.08, 4.24)

Nutrients†	**Crude OR[1] (95% CI)**	**Adjusted OR[2] (95% CI)**	**Adjusted OR[3] (95% CI)**
PUFA (%)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	2.60 (0.25, 6.219)**	2.77 (1.64, 6.79)**	2.78 (2.08, 4.94)**
trans fatty acid (%)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Excess	1.18 (0.85, 6.56)**	1.81 (1.24, 7.44)**	2.71 (1.38, 9.53)**
Protein (g/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	1.29 (0.81, 2.92)**	2.64 (0.84, 34.54)**	3.16 (0.37, 9.51)**
Carbohydrates (g/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Excess	0.69 (0.59, 2.71)	0.91 (0.48, 4.73)	0.55 (0.23, 3.45)
Fibre (g/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	0.31 (0.29, 1.76)	1.34 (0.16, 2.67)**	0.68 (0.31, 1.11)
Calcium (mg/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	1.23 (1.12, 7.75)**	0.96 (0.81, 5.88)	0.23 (0.16, 4.61)
Iron (mg/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	0.13 (0.11, 2.49)	0.41 (0.35, 3.43)	0.99 (0.39, 3.24)
Magnesium (mg/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	0.23 (0.12, 3.11)	0.21 (0.11, 2.45)	0.82 (0.52, 1.07)
Sodium (g/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Excess	2.23 (1.09, 7.95)**	3.44 (1.13, 11.06)**	4.18 (2.12, 15.69)**
Potassium (g/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	0.94 (0.87, 7.48)	0.56 (0.40, 7.65)	0.98 (0.27, 26.14)
Selenium (μg/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	0.29 (0.28, 1.92)	0.50 (0.40, 1.12)	0.76 (0.64, 3.41)
Zinc (mg/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	2.12 (2.02, 27.86)**	3.31 (2.18, 13.37)**	3.85 (2.40, 19.38)**
Vitamin A (μg/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	0.71 (0.21, 1.15)	0.74 (0.13, 1.69)	2.35 (0.31, 5.63)**

Table 4. (continued)

Nutrients†	Crude OR[1] (95% CI)	Adjusted OR[2] (95% CI)	Adjusted OR[3] (95% CI)
Vitamin C (mg/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	0.31 (0.25, 1.93)	0.78 (0.11, 1.81)	2.04 (0.19, 8.88)**
ALA (g/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	3.88 (0.16, 13.4)**	3.42 (0.397, 7.21)**	3.35 (0.817, 4.35)**
LA (g/d)			
Adequate	1.00 (referent)	1.00 (referent)	1.00 (referent)
Deficiency	0.41 (0.15, 3.29)	0.18 (0.11, 4.27)	0.91 (0.33, 3.41)

†Comparison with UK Dietary Reference Values for food energy and nutrients (DRV)[27,28] [1]Multivariate Logistic Regression model with no adjustment; [2]Multivariate Logistic Regression model after adjustment for age, gender, education, employment, marital status, and family history of CVD; [3]The final model after additional adjustment for smoking, exercise, BMI, fasting blood glucose, total cholesterol, vitamin D** $p < 0.05$. ALA, alpha-linolenic acid, LA, Linoleic acid

exercise, BMI, fasting plasma glucose, total cholesterol, and vitamin D, an excess intake of total energy (OR: 2.12, 95% CI: 0.99–13.09), SFA (OR: 2.31, 95% CI:1.47–6.91), *trans* fatty acid (OR: 2.71, 95% CI:1.38–9.53), and sodium (OR: 4.18, 95% CI:2.12–15.69) was significantly associated with an increased risk of CHD ($p<0.05$). On the other hand, after adjusting for potential confounders, the deficient intake of PUFA (OR: 2.78, 95% CI: 2.08–4.94), protein (OR: 3.16, 95% CI: 0.37–9.51), zinc (OR: 3.85, 95% CI: 2.40–19.38), vitamin A (OR: 2.35, 95% CI: 0.31–5.63), vitamin C (OR: 2.04, 95% CI: 0.19–8.88), and ALA (OR: 3.35, 95% CI: 0.81–4.35) was significantly associated with an increased risk of CHD ($p<0.05$). No significant association appeared between the risk for CHD and other nutrients, including total fat, MUFA, carbohydrates, fibre, calcium, iron, magnesium, potassium, selenium, and linoleic acid (LA).

Discussion

CHD is undisputedly the main cause of death globally; in fact, CHD is the number one cause of death in the USA and the UK (4). In Saudi Arabia, CHD ranks as the second-most significant cause of death (10). With changes in lifestyle, exposure to CHD risk factors, such as adopting diets high in SFAs and low in dietary fibre and increasing rates of obesity, hypertension, diabetes, hyperlipidaemia, smoking, and inactivity, has become more frequent (32). The current study examined the association between nutrients intake and CHD in Saudi Arabia. The findings indicated that participants without CHD had higher values of total energy intake and energy provided by SFA, *trans* fatty acids, and carbohydrates. Participants with CHD, however, had higher values of energy provided by MUFA and PUFA. The differences in consumption of total energy and energy provided by various forms of fats are potentially related to the increased awareness of case participants about how adjusting their diet affects their heart health.

An association between SFA and the risk of CHD was observed in this study. Several studies showed a positive correlation between dietary SFA intake and CHD risk, along with plasma cholesterol concentrations (33, 34). According to Jakobsen *et al.*, women with a 5% higher SFA level of energy were 36% more susceptible to higher CHD risks (35). The current study's results also indicated that subjects with excess *trans* fatty acids (% of daily energy from *trans* fatty acids >2%) were approximately 3 times more likely to suffer from CHD than subjects with adequate *trans* fatty acids (% of daily energy from trans fatty acids ≤ 2%). Similar findings appeared in Sun *et al.*'s study, which reported that subjects in the highest quartile of erythrocyte *trans* fatty acid composition were three-fold more likely to have a risk of CHD

than subjects in the lowest quartile (36). Skeaff *et al*. reported that dietary *trans* fatty acid intake increased the risk of mortality and incidence of CHD by 1.32 (1.08–1.61, p=0.006) and 1.25 (1.07–1.46, p=0.007), respectively (37). Similarly, a meta-analysis of cohort studies demonstrated that increased intake (2.5–6.3% total energy) of *trans* fatty acids substantially increased the risk of death by CVD by greater than 30% compared to low (<1–2.4% of total energy) *trans* fatty acid intake (38). Scientific research has demonstrated that *trans* fatty acids are positively related with lipoprotein (39), inflammation, and plasma triacylglycerol levels (40) and are strongly associated with CVD (36, 40).

Notably, the daily intake of total omega-3 fatty acids was increased greater than two-fold among CHD participants compared to controls in the current study. CHD participants had high intakes of total omega-3 fatty acids (1.87 g/day) and ALA (1.28 g/day). Control subjects exhibited lower mean daily intakes of ALA and LA compared with the amount recommended in the Institute of Medicines Food Nutrition Board's Dietary Reference Intake Report (ALA: 1.6 g/day for men and 1.1 g/day for women; LA: 17 g/day for men and 12 g/day for women 19 to 50 years of age) (41). Adjusting a diet to include omega-3 fatty acid sources can reduce morbidity and mortality rates by 30–50% (37, 42). Some evidence has shown inconsistent results regarding the effect of omega-3 fatty acid consumption on cardiovascular health. For instance, a review stated that omega-3 fatty acids did not exhibit strong effects on reducing blood lipids, fibrinolysis, or plasminogen activator inhibitor-1 (37). However, many studies have reported the opposite (43-45). Furthermore, American and European heart associations recommend that individuals obtain sufficient intake of long-chain PUFA (1 g/day of EPA and DHA) to prevent CHD (46). Indeed, the current study revealed that subjects with a deficiency of ALA fatty acids (<1.6 g/day for men and <1.1 g/day for women) were 3 times more likely to suffer from CHD compared with subjects with adequate ALA fatty acids (≥1.6 g/day for men and ≥1.1 g/day for women). Several studies have indicated that CHD is inversely associated with ALA intake (44, 45). Moreover, primary and secondary prevention studies have shown that consuming 2–3 g/day ALA can minimize the risk of CHD (44). A large case-control study of 3638 men and women in Costa Rica similarly found a strong inverse association between ALA status (measured via adipose tissue concentrations) and corresponding intakes of 1.8 g/day with nonfatal myocardial infarction (MI) (47). Among Dutch men (n = 20,069; aged 20–65 y), individuals who consumed >1 g/day of ALA had a 35–50% lower risk of stroke after an 8- to 13-y follow-up period (48). Moreover, epidemiological studies demonstrated that omega-6 fatty acid intake has an important effect on plasma LDL, total cholesterol, and high-density lipoprotein cholesterol levels (HDL) (49). To the researcher's knowledge, the current study is the first to examine the relationship between the intake of fatty acids and CHD in Saudi Arabia.

Although the causes of CVD commonly overlap, numerous experimental and clinical studies have contributed similar data showing that deficiencies in vitamins, minerals, and trace elements contribute significantly to CVDs and CVD symptoms (50). In the present study, the case group exhibited significantly reduced intake of calcium, sodium, and zinc and increased intake of iron, vitamin A, and vitamin E than did the control group. In this study, the mean estimation of calcium intake was 703.2 mg/day for cases and 835.5 mg/day for controls, and these levels are within the UK Department of Health's recommendations (30). A recent study by Khamis *et al*. found no significant association between dietary calcium intake and CHD mortality rate (51). In contrast, two studies found that calcium intake was accompanied by a decreased risk of CHD (52-53), and another study found that increased calcium intake resulted in an increased risk of CHD (54). Both groups in the current study exhibited high sodium intake (4.2 g/day for the control group and 3.3 g/day for the case group) that was greater than twice the Department of Health's current recommendation of 1.6 g/day (50). Additionally, a positive association between dietary sodium intake and increased risk for CHD was reported among the study sample. Moreover, Musaigar stated that populations of the Arabian Gulf region are more likely to consume more sodium because high amounts of salt exist in almost all traditional Gulf dishes, fast foods, and canned foods (55). Studies report a correlation between high sodium intake and high blood pressure, strokes, heart attacks,

and heart failure (56, 57). Zinc, an essential micronutrient, was constructively associated with risk factors for CVD (58, 59). Previous studies demonstrated similar results regarding associations between zinc deficiency and increased odds of CHD among participants (58, 60). Milton *et al*. reported an increased risk of CHD with an increase in the dietary intake of zinc (61). Several studies reported inverse associations of vitamin A (62) and vitamin C with CHD based on their antioxidant capabilities (63).

The current study has numerous strengths. First, dietary macro- and micronutrients and fatty acid intake were assessed to understand the whole diet, not only certain foods or nutrients. Data were collected via a three-day food record, showing deficient and excess nutrient intakes. This method is most efficient to assess the health of a large population because nutrient intake, both adequate and excessive, exerts considerable effects, especially among certain populations (64). Furthermore, a subject's memory is not required to complete a three-day dietary diary (65). The study also has limitations. Using household measures to obtain dietary data is less accurate than collecting data via a weighed food diary, but this method was essential for obtaining people's cooperation in recording every detail of the dietary information. Additionally, subjects must maintain their dietary habits during this period. However, this method is less costly and does not disrupt individuals' daily activities compared to other methods, such as a weighed food diary. The case-control design does not determine a causal relationship; it only focuses on the correlation between exposure and outcome. In addition, case and control participants were recruited from different hospitals, possibly introducing selection bias. Furthermore, the sample size for the current study is relatively small; however, this is the first study examining the relationship between the consumption of fatty acids and CHD in Saudi Arabia, which is the main strength of the study.

Conclusion

The current study found that participants without CHD had higher values of total energy intake and energy provided by SFA, *trans* fatty acids, and carbohydrates. Participants with CHD had higher values of energy provided by MUFA and by PUFA. In addition, controls exhibited increased intake of protein, carbohydrates, calcium, sodium, and zinc. Participants with CHD exhibited increased intake of iron, vitamin A, vitamin E, total omega-3 fatty acids, ALA, DHA, EPA, and AA. The results indicated that an excess intake of total energy, *trans* fatty acids, and sodium was associated with an increased risk of CHD. Moreover, deficient intakes of PUFA, protein, zinc, vitamin A, vitamin C, and ALA were associated with an increased risk of CHD among study participants. These findings provide more nutritional solutions suitable for preventing and controlling the incidence of CHD in Saudi Arabia. Further studies with larger sample sizes are essential to confirm these findings.

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