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Dietary inflammatory index and odds of coronary artery disease in a case-control study from Jordan

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ABSTRACT

Objective: The aim of this study was to examine the association between inflammatory potential diet as determined by dietary inflammatory index (DII) scores and coronary artery disease (CAD) in a population-based, case-control study of middle-aged Jordanian adults.

Methods: In the present study, 388 patients who were referred for elective coronary angiography at Prince Hamza Hospital, Amman, were enrolled. Of these, 198 were confirmed CAD cases and 190 were CAD-free control participants. DII scores were computed from dietary intake assessed by a food frequency questionnaire. Logistic regression models were used to estimate odds ratios (ORs) and 95% confidence intervals (CIs).

Results: CAD cases had significantly higher DII scores, higher body mass index (BMI), higher prevalence of diabetes, lower educational attainment, and lower physical activity than the CAD-free controls. A statistically significant higher risk for CAD was observed in those with DII scores in the highest tertile than in those in the lowest ($OR_{tertile3vs1}$, 2.10; 95% CI, 1.18–3.66), after adjusting for cardiovascular risk factors. A positive association was found between higher DII and CAD risk when DII score was used as a continuous variable ($OR_{continuous}$, 1.13 per unit increase in DII corresponding to ~11% of its range in the current study; 95% CI, 1.00–1.32).

Conclusions: The present findings, obtained in this ordanian population, add to the growing literature indicating that a proinflammatory diet is associated with higher risk for developing CAD.

Introduction

Cardiovascular disease (CVD) is the major cause of morbidity and mortality in different countries, accounting for 17.92 million deaths worldwide in 2015 [1]. In Jordan, it was estimated that coronary artery disease (CAD) was responsible for 27.5% or 7100 deaths in 2012 [2]. Cardiovascular disease is a condition that describes a range of disorders that affect the heart and blood vessels, such as hypertension, atherosclerosis, coronary heart disease, myocardial infarction, arrhythmias, heart failure, stroke, and CAD [3]. The probability of developing many forms of CAD can be increased with unhealthy lifestyles such as poor diet, physical inactivity, overweight and obesity, excessive stress, alcohol consumption, and tobacco use [4–6].

Chronic inflammation is a known risk factor in the development of CAD and related mortality [7]. Overnutrition and physical inactivity result in hypersecretion of inflammatory cytokines [8]. Current evidence indicates that inflammation plays a crucial role in the pathogenesis of CAD, including the initiation and development of atheroma, plaque destabilization and rupture, and postangioplasty and

AEAS, MA, and RFT conceived, designed, and supervised the study. AEAS and MA oversaw patient recruitment and were responsible for data curation. NS and LMA analyzed and interpreted the data. LMA and SA drafted the manuscript. JRH reviewed and provided inputs on the manuscript. All authors critically reviewed the manuscript and approved the final version. JRH owns controlling interest in Connecting Health Innovations LLC (CHI), a company planning to license the right to his invention of the dietary inflammatory index from the University of South Carolina in order to develop computer and smartphone applications for patient counseling and dietary intervention in clinical settings. NS is an employee of CHI. The authors have no conflicts of interest to declare.

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recurrent stenosis [9]. Previous studies have indicated that several potential inflammatory biomarkers such as C-reactive protein (CRP), interleukin (IL)-1, IL-6, IL-8, and IL-1β are potent inflammatory mediators in developing CAD and may correlate with severity of the disease [9–11].

The dietary inflammatory index (DII) is a literature review–based score that classifies individuals' diets according to their inflammatory potential on a continuum from maximal proinflammatory to maximal anti-inflammatory. The DII was first developed by researchers at the University of South Carolina [12] and updated by Shivappa et al. in 2014 [13]. The development of the DII involved scoring 1943 peer-reviewed articles between 1950 and 2010. The articles looked at the association between various dietary components and inflammation. A higher DII score indicates a more proinflammatory diet, whereas a lower DII score represents a more anti-inflammatory diet. The DII can be calculated from various dietary assessment tools, including food frequency questionnaires (FFQs) [14,15]. The DII has been validated with several inflammatory markers in various populations [13,14,16–18], including Middle Eastern populations [19,20]. Numerous studies have evaluated the association between the DII and the pathogenesis of various chronic inflammation–related health outcomes such as CAD incidence and mortality [18,21,22], lung function [16], bone health [23], some types of cancers [24–26], and cognitive disorders [15,27]. In a recent meta-analysis from 14 studies that looked at the association between DII and CAD risk and mortality, individuals in the highest versus the lowest (reference) DII category showed an overall 36% increased risk for CAD incidence and mortality, with moderate evidence of heterogeneity (relative risk [RR], 1.36; 95% confidence interval [CI], 1.19–1.57; $I^2 = 69\%$; $P < 0.001$) [28]. However, to our knowledge, no study has focused specifically on the association between the DII and CAD, and no study has been conducted to explore the DII–CAD association in a Middle Eastern population whose dietary characteristics are different from those of other populations studied. The purpose of the present study was to investigate the association between the inflammatory potential of diet, as measured by the DII score, and CAD in a case-control study in Jordan.

Methods

Study setting and participants

The study was carried out at Prince Hamzah Teaching Hospital, a referral hospital in the capital, Amman. Participants were recruited from the catheterization section of the Cardiology Department between January and December 2015. The study protocol conformed 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. Patients with clinical suspicion of CAD (stable angina, ischemic heart disease, chest pain, and positive cardiac stress test), male or female, >25 y of age who underwent elective coronary angiography, were consecutively enrolled in the study. Patients with the following diseases were excluded: acute coronary syndrome, kidney disease, liver disease, or gastrointestinal disease. Women who were pregnant or lactating were also excluded. All patients provided written informed consent to participate in the study.

Data collection

Patients were admitted the day before undergoing coronary angiography. Trained research assistants recorded data on sociodemographics (e.g., age, marital status, household income, occupation, and education level), health status, diet, previous health issues (hypertension, diabetes mellitus, dyslipidemia), smoking status, and family history of CAD using interview-based standardized questionnaires. The physical activity level was assessed using a 7-d validated physical activity recall questionnaire that focuses on a participant's recall of the usual time spent doing physical activity over a 7-d period [29]. All anthropometric measurements were carried out by a trained dietitian. Body weight was measured to the nearest 0.1 kg, with the participants wearing minimal clothing and no 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.

Dietary intake

Dietary data were collected from 400 participants who met the inclusion exclusion criteria. A validated Arabic-language quantitative FFQ aimed at measuring diet in Jordanians was adapted from the Diet History Questionnaire of the US National Cancer Institute [30]. Intake was reported for the year before diagnosis for cases and the year before interview for controls. A period of 1-y before the diagnosis date was selected to reflect seasonal variation in some food types. In face-to-face interviews, a qualified dietitian asked participants if they had eaten a specific food in the previous year. An affirmative answer resulted in determining the average rate of consumption during the past year for each food for which they had consuming at least one standard serving size in these time-related categories: <1/mo, 2–3/mo, 1–2/wk, 3–4/wk, 5–6/ wk, 1/d, 2–3/d, 4–5/d, or 6/d. If a participant's dietary pattern did not include a food type, then related questions were skipped. The modified FFQ questions contained 21 items of fruits and juices; 21 items of vegetables; 8 items of cereals; 9 items of milk and dairy products; 4 items of beans; 16 items of meat such as red meat (lamb and beef), chicken, fish, cold meat, and others; 4 items of soups and sauces; 5 items of drinks; 9 items of snacks and sweets; and 14 items of herbs and spices [30]. Food models and standard measuring tools were used to help participants estimate portion size. Data from 12 participants were excluded owing to daily caloric intake >5000 or <500 kcal. Dietary analysis software (ESHA Food Processor SQL, version 10.1.1; ESHA, Salem, OR, USA) was used to analyze the dietary intakes. It should be noted that not all of the food items consumed in Jordan are listed in the ESHA database; therefore, for these missing foods we calculated the food value from local food composition tables and these were entered into the ESHA database as new items [30,31].

The dietary inflammatory index

Development and validation of the DII has been described in detail elsewhere [12,13]. The search was conducted to discern the relationship between six

Table 1
Characteristics of patients in a Jordanian coronary artery disease case-control study, January to December 2015 (N = 388)

Variables	Control (n = 190)	Cases (n = 198)	*P*-value*,†
Age (y)	50.9 ± 10.1	52.8 ± 10.3	0.56
Dietary inflammatory index	−1.5 ± 1.5	−1.1 ± 1.8	**0.025**
Sex			
Male	104 (54.7)	126 (63.6)	0.07
Female	86 (45.3)	72 (36.4)	
BMI (kg/m^2)	30.7 ± 5.2	31 ± 7.2	0.69
Waist circumferences (cm)	105.9 ± 14.9	108.2 ± 17.8	0.21
Fasting blood glucose (mmol/L)	7.2 ± 3.4	9.1 ± 5.2	**0.001**
Family history of CAD			
Yes	69 (36.3)	79 (39.9)	0.47
No	121 (63.7)	119 (60.1)	
Hypertension	84 (44.2)	94 (47.5)	0.52
Dyslipidemia	9 (4.7)	10 (5.1)	0.59
Diabetes	42 (22.1)	84 (42.4)	**0.002**
Education			
Illiterate	13 (6.8)	28 (14.3)	**0.04**
Primary education	80 (42.1)	87 (44.4)	
Secondary education	54 (28.4)	51 (26)	
Diploma	27 (14.2)	16 (8.2)	
Bachelors	15 (7.9)	10 (5.1)	
Masters & Ph.D.	1 (0.5)	4 (2)	
Physical activity (METs/wk)			
Inactive	5 (2.6)	17 (8.6)	**0.001**
Minimally active	27 (14.2)	54 (27.3)	
HEPA active‡	158 (83.2)	127 (64.1)	
Smoking			
Yes	69 (36.3)	93 (47)	0.11
No	67 (35.3)	54 (27.3)	
Previous	10 (5.3)	14 (7.1)	
Passive	44 (23.2)	37 (18.7)	

HEPA, health-enhancing physical activity; METs, metabolic equivalents.
Values are mean ± standard deviation or n (%).
*Student's *t* test was used for continuous variables.
†χ^2 test was used for categorical variables.
‡The HEPA category "active" included any participant who performed vigorous-intensity activity on ≥3 d/wk, accumulated ≥1500 MET-min/wk, or who performed any combination of walking, moderate-intensity or vigorous-intensity activities ≥5 d achieving a minimum of ≥3000 MET-min/wk. Significant p-values set in bold.

inflammatory biomarkers and various micronutrients, macronutrients, and whole-food items (termed *food parameters*). These food parameters, which include several nutrients, foods, and bioactive compounds were not chosen a priori; rather they were identified prospectively when the search was done as part of the effort to identify the dietary components that have an effect on inflammation.

Each individual's dietary intake was standardized to a world database, which contained the means and standard deviations (SDs) of intake for the food parameters from 11 populations around the world [13]. The original DII consists of 45 components. However, many studies that use an FFQ for dietary assessment cannot account for all of the components. In the present study, the available components that were used for calculating DII included the following nutrients: alcohol, vitamin B_{12}, vitamin B_6, β-carotene, carbohydrates, cholesterol, energy, fat, fiber, folic acid, iron, magnesium, monounsaturated fatty acids (MUFAs), niacin, ω-3, ω-6, protein, polyunsaturated fatty acids (PUFAs), riboflavin, saturated fat, selenium, thiamin, trans-fat, vitamins A, C, D, and E, and zinc. The FFQ also provided information on onions and tea and the following bioactive compounds: flavan-3-ol, flavones, flavonols, and isoflavones. The world mean value for each food parameter was subtracted from the reported intake value for each and then divided by the world SD to create a *z*-score. The next step converted the *z*-scores to proportions using the PROBNORM function in SAS (SAS Institute, Cary, NC, USA). These values were then centered by doubling the value and subtracting 1. This value was then multiplied by the inflammatory effect score for each food parameter. These were then summarized across all food parameters to derive the overall DII score. Higher DII scores indicate a more proinflammatory diet, whereas lower scores indicate a more anti-inflammatory diet. Finally, it should be noted that DII scores were calculated per 1000 calories/d consumed, which required using an energy-adjusted world referent database.

Statistical analysis

Differences in baseline variables between patients with and without CAD were estimated using χ^2 tests for categorical variables and student's *t* tests for continuous variables. We analyzed the DII as a continuous variable and categorized into tertiles, with cutpoints derived from data obtained in the controls. Odds ratios (ORs) and 95% CIs were estimated using logistic regression models, adjusting only for age first and then fitting a model with additional adjustment for sex, education, physical activity, body mass index (BMI), total energy, hypertension, dyslipidemia, diabetes, smoking, and family history of CAD. All tests were two-sided and the significance level was set at $P < 0.05$. Data were analyzed using SPSS for Windows version 23 (SPSS Inc., Chicago, IL, USA).

Results

General characteristics of study population

In this study, DII scores ranged from −3.40 (most anti-inflammatory score) to +3.89 (most proinflammatory score). Table 1 presents the general sociodemographic characteristics and lifestyle variables of the 198 cases and 190 controls. There were some significant differences between cases and controls in terms of sociodemographic factors and lifestyle habits. Cases had significantly higher DII scores and fasting blood glucose, higher prevalence of diabetes, lower educational level, and lower physical activity than the control group. The difference between the two groups was not statistically significant for the following variables: age, sex, BMI, waist circumferences, family history of CAD, or smoking status.

Table 2 shows the general sociodemographic characteristics and lifestyle variables of cases according to DII tertiles. There were no statistical differences in sociodemographic factors and lifestyle habits across DII tertiles. General characteristics of the controls according to DII tertiles are presented in Table 3. Participants in the top tertile of DII with more proinflammatory diet were significantly younger than participants in lowest tertile ($P = 0.01$). BMI and waist circumference were lower in the highest tertile than in the lowest tertile ($P = 0.003$ and $P = 0.002$, respectively). Participants in the

Table 2
Participant characteristics by tertile of dietary inflammatory index among cases, Jordanian coronary artery disease case-control study, January to December 2015 (N = 198)

Variables	Tertile 1 (≤−1.30)	Tertile 2 (−1.30 to 0.29)	Tertile 3 (>0.29)	*P*-value*,†
Age (y)	53.4 ± 10.4	54.1 ± 11.3	51.2 ± 9.1	0.22
Sex				
Male	27 (60)	48 (60.8)	51 (68.9)	0.49
Female	18 (40)	31 (39.2)	23 (31.1)	
BMI (kg/m^2)	30.9 ± 5.8	31.1 ± 6.8	30.9 ± 8.4	0.98
Waist circumferences (cm)	110.6 ± 20.4	108.8 ± 17	106.1 ± 16.9	0.42
Fasting blood glucose (mmol/L)	9.52 ± 5.5	9 ± 4.9	9.03 ± 5.52	0.86
Family history of CAD				
Yes	19 (42.2)	31 (39.2)	29 (39.2)	0.94
No	26 (57.8)	48 (60.8)	45 (60.8)	
Hypertension	32 (53.3)	35 (51.5)	27 (38.6)	0.18
Dyslipidemia	4 (6.7)	3 (4.4)	3 (4.3)	0.79
Diabetes	30 (50.0)	32 (47.1)	22 (31.4)	0.07
Education				
Illiterate	10 (22.2)	9 (11.5)	9 (12.3)	0.42
Primary education	21 (46.7)	35 (44.9)	31 (42.5)	
Secondary education	8 (17.8)	20 (25.6)	23 (31.5)	
Diploma	2 (4.4)	6 (7.7)	8 (11)	
Bachelors	3 (6.7)	5 (6.4)	2 (2.7)	
Masters & Ph.D.	1 (2.2)	3 (3.8)	0 (0)	
Physical activity (METs/wk)				
Inactive	5 (11.1)	9 (11.4)	3 (4.1)	0.32
Minimally active	9 (20)	21 (26.6)	24 (32.4)	
HEPA active‡	31 (68.9)	49 (62)	47 (63.5)	
Smoking				
Yes	20 (44.4)	31 (39.2)	42 (56.8)	0.12
No	14 (31.1)	27 (34.2)	13 (17.6)	
Previous	2 (4.4)	4 (5.1)	8 (10.8)	
Passive	9 (20)	17 (21.5)	11 (14.9)	

HEPA, health-enhancing physical activity; METs, metabolic equivalents.
Values are mean ± SD or n (%).
*Student's *t* test was used for continuous variables.
†χ^2 test was used for categorical variables.
‡The HEPA category "active" included any participant who performed vigorous-intensity activity on ≥3 d/wk, accumulated ≥1500 MET-min/wk, or who performed any combination of walking, moderate-intensity or vigorous-intensity activities ≥5 d achieving a minimum of ≥3000 MET-min/wk.

Table 3
Participant characteristics by tertile of dietary inflammatory index among controls, Jordanian coronary artery disease case-control study, January to December 2015 (N = 190)

Variables	Tertile 1 (≤−1.30)	Tertile 2 (−1.30 to 0.29)	Tertile 3 (>0.29)	*P*-value*,†
Age (y)	53.6 ± 9	51.1 ± 10	47.9 ± 10.6	**0.01**
Sex				
Male	28 (44.4)	37 (58.7)	39 (60.9)	0.13
Female	35 (55.6)	26 (41.3)	25 (39.1)	
BMI (kg/m^2)	32.5 ± 5	30.1 ± 5.4	29.6 ± 4.8	**0.003**
Waist circumferences (cm)	110.85 ± 14.2	106.4 ± 14.6	100.2 ± 14.3	**0.002**
Fasting blood glucose (mmol/L)	7.5 ± 3.6	7.8 ± 3.5	6.5 ± 2.9	0.09
Family history of CAD				
Yes	22 (34.9)	24 (38.1)	23 (35.9)	0.93
No	41 (65.1)	39 (61.9)	41 (64.1)	
Hypertension	39 (57.4)	22 (36.7)	23 (37.1)	**0.02**
Dyslipidemia	4 (5.9)	2 (3.3)	3 (4.8)	0.68
Diabetes	29 (42.6)	13 (21.7)	10 (16.1)	**0.002**
Education				
Illiterate	4 (6.3)	3 (4.8)	6 (9.4)	0.20
Primary education	28 (44.4)	26 (41.3)	26 (40.6)	
Secondary education	12 (19)	25 (39.7)	17 (26.6)	
Diploma	11 (17.5)	5 (7.9)	11 (17.2)	
Bachelors	8 (12.7)	4 (6.3)	3 (4.7)	
Masters & Ph.D.	0 (0)	0 (0)	1 (1.6)	
Physical activity (METs/wk)				
Inactive	1 (1.6)	3 (4.8)	1 (1.6)	0.73
Minimally active	9 (14.3)	10 (15.9)	8 (12.5)	
HEPA active‡	53 (84.1)	50 (79.4)	55 (85.9)	
Smoking				
Yes	15 (23.8)	26 (41.3)	28 (43.8)	**0.006**
No	29 (46.0)	14 (22.2)	24 (37.5)	
Previous	1(1.6)	7 (11.1)	2 (3.1)	
Passive	18 (28.6)	16 (25.4)	10 (15.6)	

HEPA, health-enhancing physical activity; METs, metabolic equivalents.
Values are mean ± SD or n (%).
*Student's *t* test was used for continuous variables.
†χ^2 test was used for categorical variables.
‡The HEPA category "active" included any participant who performed vigorous-intensity activity on ≥3 d/wk, accumulated ≥1500 MET-min/wk, or who performed any combination of walking, moderate-intensity or vigorous-intensity activities ≥5 d achieving a minimum of ≥3000 MET-min/wk.

highest DII tertile were less likely to be diabetic ($P = 0.002$) and hypertensive ($P = 0.02$) and were more likely to smoke ($P = 0.006$).

Food group distribution among DII tertiles

Table 4 shows the distribution of 32 food groups (g/wk) across DII tertiles with percentage difference between highest and lowest tertiles described in cases. The food groups that showed the greatest reduction (≥50%) from tertile 1 to tertile 3 were whole grains (92%), bulgur (63%), vegetables (58%), fruits (54%), and soup (53%); and the food groups that showed greatest increase (≥20%) were regular soft drink (201%), fried potato (135%), snacks (102%), processed meat (100%), fast foods (53%), breakfast cereals (44%), popcorn (31%), refined grains (28%), and artificial fruit juices (20%). Food groups that differed little (≤5%) across DII tertiles included tea, pickles, and egg.

Table 5 presents the corresponding data for the distribution of the 32 food groups (g/wk) among controls across DII tertiles with percentage differences between the highest and lowest tertiles described. The food groups that showed the greatest reduction (50%) from tertile 1 to tertile 3 were whole grains (95%), breakfast cereal (70%), diet soft drinks (57%), vegetables (54%), nuts and seeds (54%), dried fruit (53%), and fruits (52%); and the food groups that showed the greatest increase (≥20%) were processed meat (201%), sweets (191%), regular soft drinks (179%), artificial fruit juices (101%), snacks (100%), fried potatoes (98%), refined grains (68%), eggs (60%), dairy (47%), fast foods (46%), coffee (34%), and meat (27%). Food groups that differed little (≤5%) across DII tertiles included tea and vegetable oil.

The association between DII and CAD risk

The ORs and 95% CIs for CAD are shown in Table 6. Results obtained from modeling DII scores as a continuous variable in relation to the odds of developing CAD suggested a positive association after adjusting for age (OR, 1.10; 95% CI, 0.94–1.25). Similar results were obtained in the multivariable analyses model, with an OR of 1.13 (95% CI, 1.00–1.32) for 1-unit increase in DII score (i.e., ~11% of the range of DII). In the age-adjusted models for DII tertiles, participants in the highest DII tertile had an OR of 1.77 (95% CI, 1.05–2.97) compared with participants in the lowest tertile. Results obtained after multivariable adjustment showed that participants in the top tertile of DII were more likely to have a CAD (110% higher likelihood) than participants in the lowest tertile ($OR_{\text{tertile 3vs1}}$, 2.10; 95% CI, 1.18–3.66; P_{trend}, 0.02).

Discussion

In this ordanian case-control study, a 110% excess CAD risk was found among people who consumed the most proinflammatory diets (i.e., in the top tertile of DII scores) versus those consuming the most anti-inflammatory diet (i.e., in the lowest tertile of DII scores). Furthermore, we observed that people with lower DII scores (tertile 1) consumed higher amounts of whole grains, vegetables, and fruits; which contain many anti-inflammatory dietary components. By contrast, people with the highest DII scores (tertile 3) consumed more soft drinks, processed meat, and fried potatoes, which contain proinflammatory components. The reason for observing lower DIIs among participants with diabetes or hypertension could be that

Table 4
Distribution of food groups across tertiles of DII* among cases, Jordanian cardiovascular disease case-control study, 2010 to 2012

Food group (g/wk)	Direction increase with DII†	Difference (T3–T1)/T1 (%)	T1	T2	T3
Whole grains	–	−92	42.5	10.9	3.5
Bulgur	–	−63	15.8	10.4	5.8
Vegetables	–	−58	405.4	292.1	170.2
Fruits	–	−54	514.6	511.1	238.8
Soup	–	−53	267.3	171.5	124.5
Dried Fruits	–	−43	14.6	14.5	8.3
Diet soft drink	–	−41	19.8	20.4	11.6
Stuffed vegetables	–	−40	25.7	20.8	15.4
Legumes	–	−35	197.9	162.8	128.8
Fresh juices	–	−25	73.9	100.0	55.1
Vegetable oil	–	−25	12.1	10.6	9.1
Fish	–	−25	20.3	20.9	15.3
Sweets	–	−22	13.3	11.2	10.3
Starchy vegetables	–	−18	34.5	48.1	28.2
Nuts and seeds	–	−17	13.4	15.9	11.1
Olive oil and olive pickles	–	−14	16.7	17.9	14.4
Tea	0	−2	547.4	577.0	534.3
Pickles	0	−1	30.9	30.6	30.6
Eggs	0	0	24.1	27.9	24.1
Coffee	+	6	140.4	171.6	148.9
Poultry	+	9	33.4	40.7	36.3
Dairy	+	13	230.1	269.0	259.7
Meat	+	16	34.1	38.9	39.5
Artificial fruit juices	+	20	63.8	71.0	76.4
Refined grains	+	28	223.0	235.0	284.8
Popcorn	+	31	0.8	1.0	1.1
Breakfast cereals	+	44	0.1	0.0	0.2
Fast food	+	53	24.2	25.4	37.2
Processed meat	+	100	6.0	3.8	12.0
Snacks	+	102	36.9	72.7	74.5
Fried potatoes	+	135	14.0	27.3	32.8
Regular soft drink	+	210	131.5	240.7	407.2

*The foods listed are meant to provide a sense of how dietary intake varies across DII tertiles; although some listed foods are indeed among the 37 parameters that are used in computation of DII in the Iowa Women's Health Study (IWHS), these are not meant to be a list of contributors to the DII.
†+ indicates increase across tertiles; – indicates decrease across tertiles; 0 indicates no or <5% change.

individuals who were diagnosed with these chronic diseases may have begun consuming healthier diets with lower inflammatory potential. As for thin individuals or younger people having high DII, those who are thin may consume less of everything, including anti-inflammatory components, and hence the score may be high. Younger people in Jordan may be influenced by Westernization, which may result in them consuming more proinflammatory foods rich in components such as saturated fat and low in anti-inflammatory components like fruits and vegetables.

The results of the present study provided additional evidence for the effect of a proinflammatory diet on increasing the risk for developing CAD. The findings agree with previous reports that found that DII scores were positively associated with the risk for CAD in United States [32,33], Australian [34], and European [18,24,35] populations. A meta-analysis using data from 14 eligible studies exploring the association between the DII and risk for CAD and CAD mortality revealed a 36% increase in the risk for CAD incidence in individuals in the highest versus the lowest (reference) DII category, with moderate evidence of heterogeneity (RR, 1.36; 95% CI, 1.19–1.57; $I^2 = 69\%$; $P < 0.001$) [28]. In addition, the study showed an 8% increase in the risk for CAD and mortality for each 1-point increase in the DII score when DII was analyzed as a continuous variable [28]. Another systematic review and meta-analysis study conducted by Namazi et al., in 2018 [36], found that there was a suggestion of a positive relationship between the DII and the risk for incidence for CAD, with a pooled RR of 1.35 (95% CI, 1.13–1.60; $I^2 = 28.6\%$; $P = 0.21$) [36].

In an Australian prospective cohort study, the likelihood of experiencing a CAD event doubled in men with a proinflammatory diet over the study period (OR, 2.00; 95% CI, 1.03–3.96) [37]. Results from the prospective Spanish PREDIMED (Prevention with Mediterranean Diet) study indicated that the risk for CAD increased by 73% when comparing DII quartile 4 to quartile 1 ($HR_{quartile\ 4vs.1}$, 1.73; 95% CI, 1.15–2.60) [21]. Results from a case-control study conducted in northern Sweden showed that the risk for myocardial infarction increased by 57% ($OR_{quartile\ 4vs1}$, 1.57; 95% CI, 1.21–2.02; $P = 0.02$) in male participants with the most proinflammatory DII scores [18]. Moreover, in an Italian case-control study, the proinflammatory diet, indicated by higher DII scores, was determined to increase the likelihood of acute myocardial infarction by 14% per DII point (OR, 1.14; 95% CI, 1.05–1.24); a 1-unit increase in DII score corresponds to ~9% of the range of DII. When expressed in quartiles, the results are very consistent with what has been seen in the other studies, with a 60% increase across the extreme quartiles ($OR_{quartile\ 4vs1}$, 1.60; 95% CI, 1.06, 2.41; P_{trend}, 0.02) [35].

The association between diet and the risk for CAD has been investigated previously in this Jordanian case-control study [38] and in other places around the world [39–42]. In Jordan, a "healthy dietary pattern" (rich in olive oil, fruits, vegetables, legumes, whole grains, and fish and low in meat) and a "high-fiber pattern" (mainly composed of legumes and bulgur) have been found to be inversely associated with the odds of CHD [38]. Dietary patterns associated with consuming large amounts of whole vegetables and fruits and limiting intake of red and processed meat, refined grains, and sugar-sweetened foods and beverages provide plentiful nutrients, such as anthocyanins and flavanones [43,44], folate [45,46], vitamin B_6 [45,46], vitamin D [47,48], and fiber [56], which have been found to be inversely associated with CAD (especially acute myocardial infarction). Adherence to a healthy dietary pattern has been found to reduce the risk for CHD by 20% to 33%, whereas Western-

Table 5
Distribution of food groups across tertiles of DII* among controls in Jordanian cardiovascular disease case-control study, 2010 to 2012

Food group (g/wk)	Direction increase with DII†	Difference (T3–T1)/T1 (%)	T1	T2	T3
Whole grains	–	−95	49.6	12.1	2.4
Breakfast cereals	–	−70	0.4	0.1	0.1
Diet soft drinks	–	−57	23.1	33.4	10
Vegetables	–	−54	433.6	312.1	198.5
Nuts and seeds	–	−54	15.5	14.7	7.1
Dried fruits	–	−53	21.1	17.9	10
Fruits	–	−52	638.8	548.2	305.5
Pickles	–	−43	56.2	30.4	31.8
Fresh juices	–	−40	96	49	57.6
Sauce	–	−40	22.1	21.8	13.3
Fish	–	−39	23.3	19.7	14.3
Starchy vegetables	–	−35	59.9	38.9	39.1
Stuffed vegetables	–	−24	24.6	23.7	18.6
Bulgur	–	−23	12.6	11.1	9.7
Poultry	–	−23	39.8	36.3	30.8
Popcorn	–	−13	1.3	1.5	1.2
Legumes	–	−12	163.9	158.9	143.5
Olive oil and olive pickles	–	−12	19.6	17.5	17.2
Vegetable oil	–	−4	10.5	10.1	10.1
Tea	0	0	569.5	557.1	568.3
Meat	+	27	27.2	34.7	34.7
Coffee	+	34	125.7	145.2	167.8
Fast foods	+	46	26.5	36.6	38.9
Dairy	+	47	220.4	237.9	324.4
Eggs	+	60	18.2	30.8	29.1
Refined grains	+	68	176.7	261.7	297
Fried potatoes	+	98	21.5	32.1	42.8
Snacks	+	100	48.8	82.2	97.5
Artificial fruit juices	+	101	37.1	83.7	74.7
Regular soft drinks	+	179	147.4	238.1	410.7
Sweets	+	191	6.8	12.7	19.7
Processed meat	+	201	2.7	6.5	8.2

*The foods listed are meant to provide a sense of how dietary intake varies across DII tertiles; although some listed foods are indeed among the 37 parameters that are used in computation of DII in the Iowa Women's Health Study (IWHS), these are not meant to be a list of contributors to the DII.
†+ indicates increase across tertiles; − indicates decrease across tertiles; 0 indicates no or <5% change.

type patterns increased the CHD risk by ≤45%, especially in US studies and in individuals >50 y of age of age [39,49,50]. Furthermore, increased adherence to the Mediterranean diet was found to be associated with a lower risk for incident of CHD and stroke in women [51]. It is known that blood lipids and lipoproteins, systemic inflammatory or oxidative stress factors like high-sensitivity CRP and oxidized low-density lipoprotein cholesterol, and carotid atherosclerosis increase risk, whereas improving endothelial health and blood pressure decrease risk [52–55].

Mechanisms of action associated with high DII scores may operate by inducing the migration of inflammatory cells (such circulating monocytes and platelets) into vascular tissue; and these, in turn, mediate the adhesion of leukocytes to the vascular endothelium by IL-6 and other proinflammatory cytokines (IL-1 and tumor necrosis factor-α) [9,56,57].

To our knowledge, this is the first case-control study conducted in a ordanian, or any other Middle Eastern, population to investigate the association between the inflammatory potential of diet (as expressed using DII scores) and CAD. The dietary choices and socioeconomic characteristics of Middle Eastern populations are culturally different from the better-studied Western populations, and the results in this population reinforce the value of the DII as a tool for relating inflammatory potential of diet to CAD in a wide variety of populations.

Despite its strengths, a potential limitation of this study was its small sample size, which reduced the precision of the OR estimates, resulting in wider CIs than what would be observed in a larger sample size. However, recruitment strategies help to ensure that the sample is representative of the population. FFQs have been shown to be subject to reporting biases, especially those associated with underreporting [58–60]. Even after we excluded individuals with extreme energy intakes, this remained as a potential problem. However, the FFQs were administered by highly trained, professional staff, which may have helped to reduce response biases. However, we did not measure, and therefore could not account for, sources of possible reporting error, including from response sets such as such as social approval or social desirability [58–62]. Still, there may be less motivation to misreport in this population owing

Table 6
OR (95% CIs) for the association between DII coronary artery in a Jordanian case-control study, January to December 2015 (N = 388)

DII	Energy adjusted-DII (tertiles), OR (95% CI)				Energy adjusted-DII (continuous)*
	Tertile 1 (≤−1.30)	Tertile 2 (−1.30 to 0.29)	Tertile 3 (>0.29)	P_{trend}†	OR (95% CI)
Age-adjusted	1 (ref.)	**1.80 (1.08–2.99)**	**1.77 (1.05–2.97)**	**0.03**	1.10 (0.94–1.25)
Multivariate-adjusted‡	1 (ref.)	**2.00 (1.15–3.44)**	**2.10 (1.18–3.66)**	**0.02**	**1.13 (1.01–1.32)**

*1-unit increase equals to ~11% range of DII in this study (−3.40 to +3.89).
†P_{trend} derived using the median approach.
‡Adjusted for age, sex, education, physical activity, total energy, body mass index, hypertension, dyslipidemia, diabetes, smoking, family history of CAD.

to the fact that diet-related hypotheses are not as widely broadcast as in the West. In addition, we did not obtain quantitative information on the use of dietary supplements because it is not common or widespread in ordan; therefore, we did not consider their use in this study. Another limitation could be the non-availability of data on inflammatory markers to validate the DII in this study; however the DII has been validated with inflammatory markers in the Middle Eastern population in several studies [19,20,63]. Finally, dietary data collected over the past year may not be sufficient to determine an association with a disease state that may take many years, or even decades, to develop. However, this study provided data to support conducting long-term, prospective studies involving nutritional intakes and ongoing outcome assessment. In this case-control, population-based study, a more proinflammatory diet as estimated by the DII was associated with an increased risk for CAD. These findings indicated a detrimental role of inflammation as a link between diet and cardiovascular risk as well as the usefulness of the DII for estimating the inflammatory effects of diet.

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