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Dietary inflammatory index is associated with increased risk for prostate cancer among Vietnamese men

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ABSTRACT

Objective: Inflammatory potential of diet, as measured by the dietary inflammatory index (DII), has consistently been associated with an increased risk for prostate cancer (PCa). However, data has largely been reported in populations with more proinflammatory dietary patterns, whereas there is high diversity in dietary pattern worldwide. The aim of this study was to assess the association between DII scores and the risk for PCa in Vietnam.

Methods: A case-control study of 652 participants (244 incident PCa patients, 64–75 y of age, and 408 controls, frequency matched for age) was conducted in Ho Chi Minh City, Vietnam, from 2013 to 2015. Habitual diet was ascertained using a validated food frequency questionnaire (FFQ), whereas other factors, including demographic and lifestyle characteristics, were assessed via face-to-face interviews. The daily intake of pro- and anti-inflammatory nutrients for each participant was calculated from the FFQ and used to estimate individuals' energy-adjusted DII (E-DII) scores. Multivariate-adjusted odds ratios (OR) and 95% confidence intervals (CIs) were estimated using unconditional logistic regression models.

Results: Comparing the middle and highest versus lowest tertile of DII scores, there was an increased risk for overall PCa. The OR and associated 95% CI was 2.63 (1.61–4.37) and 3.35 (2.06–5.53), respectively (P_{trend} < 0.01). Similar results were found for low-moderate and high-grade PCa. The respective ORs (95% CI) were 3.34 (1.66–7.13) and 5.29 (2.69–11.18), P_{trend} < 0.001, and 2.51 (1.40–4.63) and 2.57 (1.43–4.73), P_{trend} 0.006.

Conclusion: A proinflammatory diet was associated with increased risk for PCa among Vietnamese men.

Introduction

Prostate cancer (PCa) is a major public health problem owing to its high incidence. In 2012, ~1.1 million incident cases of PCa and 307 000 deaths were reported globally. In men, PCa is the second most common cancer and the fifth leading cause of cancer death [1]. Despite lower PCa incidence in Southeast Asian populations than in Western populations [1,2], an upward trend has been seen in recent decades [2,3]. Although age, race (e.g., African American), and history of PCa in first-degree relatives are well-recognized risk factors for PCa [2], attention has been drawn to the role of chronic inflammation in PCa etiology [4]. It has been suggested that proinflammatory cytokines such as C-reactive protein [5], interleukin (IL)-6 [6], and leukocyte count [7] increase the risk for PCa. Meanwhile, anti-inflammatory markers, including adiponectin [6] and IL-10 [7], have been beneficially associated with PCa risk. Therefore, factors that influence inflammatory potential may contribute to the prevention of PCa.

Evidence suggests that diet may influence chronic inflammation through dietary bioactive compounds with anti- or proinflammatory activities [8–10]. The intake of highly proinflammatory dietary nutrients such as simple carbohydrates and saturated fat is strongly positively correlated with inflammatory cytokines [8,11]. Assessing

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dietary inflammation, therefore, may provide further insight into the relation between low-grade inflammation and risk for PCa.

The dietary inflammatory index (DII) is a literature-derived scoring system that estimates the inflammatory potential of an individual's diet [8]. Although accumulating evidence indicates a positive association between dietary inflammatory potential and the risk for PCa [12–15], the available evidence is largely found in populations with more proinflammatory dietary patterns [12,14–17]; for instance, populations that consume diets high in fat and meat [18] also tend to have high incidence of PCa [1]. Given considerable variations in both dietary patterns [19] and PCa incidence across regions [1], the relation between DII and PCa risk may vary by factors that influence disease severity and tumor grade. Asia has undergone a rapid nutrition transition [20] and has experienced an increase in PCa incidence [2]; however, to our knowledge, there have been no studies on the association between DII and PCa risk apart from two case-control trials in Iran [15,21]. Our previous report of carotenoid intake in relation to PCa in Vietnamese men [22] prompted us to conduct the present study to investigate the association between DII scores and the risk for PCa among Vietnamese men.

Methods

Selection of cases and controls

This case-control study was conducted in Ho Chi Minh City, the largest municipality in Vietnam. Between January 2013 and April 2015, we attempted to identify men 64 to 75 y of age diagnosed with PCa. The diagnosis was made within 1 wk before their interview. Of the 272 eligible patients, we interviewed 253 who consented to participate in the study. In addition, 9 patients were excluded either owing to missing or implausible information, resulting in 244 patients with PCa included for statistical analyses.

We selected eligible controls from communities and hospitals representing the source population from which the cases arose. For community controls, we recruited men who reported having no previous or current malignancy or other conditions that might be associated with changing dietary behavior. Hospital controls attended the same hospitals owing to minor health problems (e.g., minor injuries) and also had no previous or current malignancy or other condition that might be associated with changing dietary behavior. Controls were frequency matched to the cases by 5-y age groups. Of 700 eligible controls contacted, 429 men agreed to take part in the study; however, 62 were excluded because they refused to provide a blood sample for prostate-specific antigen (PSA) testing, they had >4 ng/mL serum PSA, or they had malignant or severe chronic diseases. As a result, 367 eligible men were recruited and interviewed. For hospital controls, we approached 120 eligible patients who attended the same hospitals as the cases for health check or for treatment of minor health issues and enrolled 83 men who consented to be interviewed. We further excluded 28 community controls and 14 hospital controls because of implausible or missing information. Thus, 652 subjects (244 cases and 408 controls [339 community-based and 69 hospital-based]) remained in the present study.

Interviewing

Structured face-to-face interviews were conducted to collect information on demographic characteristics and diet and other lifestyle behaviors. The duration of each interview was approximately 40 min. The participant's next of kin were encouraged to take part in the interview to improve response quality. To reduce response bias, interviewers and participants were blinded regarding the study hypotheses. The study was approved by the Human Research Ethics Committee of Curtin University, and all participants provided written informed consent before participating in the study.

Dietary assessment

Dietary habits were assessed with a semiquantitative food frequency questionnaire (FFQ) adapted from a previous, validated food frequency questionnaire [23] that was used in previous studies with Vietnamese adults [22,24]. This dietary questionnaire assessed 89 food and beverage items (e.g., tea, coffee, and other beverages such as canned beverages or fruit juice; fruits; vegetables; soy foods; meats, seafood; cereals; eggs; and dairy products) and was administered by a trained interviewer. The questions referred to the daily or weekly consumption of various foodstuff or groups of foodstuffs during the 3 y before the interview. Estimation of the food amounts consumed and portion size was based on a picture booklet provided to participants. Owing to limitations in nutrient data availability in the Vietnamese Food Composition Table (VFCT) [25], daily intakes of nutrients for each participant were calculated using the US Department of Agriculture nutrient database [26,27]. Intakes of macronutrients, total energy (kcal/d), and food or beverage items were estimated using the current Vietnamese Food Composition Table [25].

Other variables

Demographic and lifestyle factors including age, marital status, education level, smoking, and lifelong physical activity exposure [28] were measured, in addition to medical history (e.g., histologic examination of prostate gland and PSA level), height, and weight. The World Health Organization's STEPwise protocol was used to elicit information about smoking and alcohol drinking [29].

Dietary inflammatory index

The daily intakes of pro- and anti-inflammatory nutrients were used to estimate individuals' DII scores, using a scoring system reported in detail elsewhere [8], and described briefly here. In short, dietary data for each study participant were first linked to a regionally representative global database that provided an estimate of means and SDs for each of the food parameters considered (i.e., foods, nutrients, and other food components). The "standard mean" was subtracted from the actual food parameter value and divided by its SD. This *z*-score was then converted to a proportion (to minimize the effect of outliers or right-skewing, a common occurrence with dietary data). This value was then converted to a centered proportion by doubling and subtracting 1. Each centered proportion was then multiplied by the respective inflammatory effect score of the food parameters (derived from a literature review and scoring of 1943 "qualified" articles) to obtain the individual's food parameter–specific DII score. All of the food parameter–specific DII scores were then summed to create the overall DII score for each participant in the study. A higher DII score indicated the greater proinflammatory potential of the diet. For this study, we computed energy-adjusted DII (E-DII) scores, which were expressed per 1000 kcal of intake. The same procedure used to compute DII scores was used, and the global comparison database consisted of energy-adjusted intake values (also per 1000 kcal).

Statistical analysis

Participants' demographic and lifestyle characteristics were compared between case and control groups using two-sample *t* tests for continuous variables, and χ^2 tests for categorical variables. Distributions of various food groups also were examined across tertiles of E-DII scores.

Because the cases and controls were not individually matched, unconditional logistic regression analyses were performed to ascertain the strength of association between E-DII scores and PCa risk. E-DII scores were transformed into a categorical variable based on the distribution of E-DII scores in controls [30]. The lowest (most anti-inflammatory) E-DII tertile was treated as the reference category. Crude and adjusted odds ratios (OR) and their corresponding 95% confidence intervals (CIs) were calculated. In addition, tests for linear trend were performed on continuous variables using the median values of the tertile categories of E-DII in the logistic regression models.

The following variables were included in the logistic regression models as potential confounding factors: education level (primary, high, and tertiary), marital status (married, never married, or separated), smoking habit (never, former, current), history of PCa in a first-degree relatives (yes, no), lifelong physical activity (never, past active, regular), age (year, continuous), body mass index (BMI; kg/m^2, continuous), ethanol consumption (g/d, continuous), and total energy intake (kcal/d, continuous). They were adjusted because their possible effects on the association between E-DII and PCa risk as reported in the literature [31]. Subgroup analyses were conducted for low-medium (Gleason score ≤7) and high-grade (Gleason score 8–10) PCa [32]. R Statistics software version 3.3.3 [33] was employed to perform all statistical analyses, and a two-sided $P < 0.05$ was considered statistically significant.

Results

The sample consisted of 408 controls and 244 cases. The mean age of participants was 68.3 y (SD 6.4). Compared with controls, those with PCa smoked more cigarettes, consumed more alcohol, and had higher mean E-DII scores. They were married at a younger age, had fewer children, and had a lower energy intake before diagnosis. The two groups also were different in terms of educational level and lifetime physical activity, with the cases being less educated and less active than the controls. First-degree family history of PCa was reported by seven cases only (Table 1).

Table 1
Sample characteristics

Characteristics	Cases (n = 244)	Controls (n = 408)	*P*-value*
Age (y), mean (SD)	68.7 (7.3)	68 (5.8)	0.16
Body mass index (kg/m^2), mean (SD)	22 (3)	21.9 (3.3)	0.91
Ethanol (g/d), mean (SD)	22.6 (47.5)	14.9 (28.4)	0.02
Total energy (kcal/d), mean (SD)	1731.6 (664.9)	2097 (836)	<0.001
E-DII, mean (SD)	0.79 (1.39)	0.20 (1.88)	<0.001
Number of children, n (%)			
≤3	102 (41.8)	219 (53.7)	<0.001
4–6	93 (38.1)	152 (37.3)	
≥7	49 (20.1)	37 (9.1)	
Education level, n (%)			
Primary school	65 (26.6)	72 (17.6)	0.02
High school	134 (54.9)	261 (64)	
Tertiary education	45 (18.4)	75 (18.4)	
Marital status, n (%)			
Married	233 (95.5)	375 (91.9)	0.11
Never married or separated	11 (4.5)	33 (8.1)	
Smoking habit, n (%)			
Never	58 (23.8)	111 (27.2)	0.18
Former	121 (49.6)	172 (42.2)	
Current	65 (26.6)	125 (30.6)	
Prostate cancer in the first-degree relatives (yes), n (%)	7 (2.9)	0 (0)	0.002
Lifelong physical activity, n (%)			
Never	200 (82)	192 (47.1)	
Past active	25 (10.2)	102 (25)	<0.001
Regular	19 (7.8)	114 (27.9)	

E-DII; energy-adjusted dietary inflammatory index.
*P-value from t test or χ^2 test.

Table 2 depicts the distribution of study population characteristics across tertiles of E-DII scores. Compared with the lowest tertile, men in the highest tertile reported higher mean ethanol intakes (24.7 [SD 52.4] versus 14.4 [SD 21.6] g/d), but lower mean energy intakes (1964 [SD 880] versus 2128 [SD 656] kcal) and were less educated (25.8% versus 16.2% with just a primary school education).

Table 3 presents the distribution of the intake of major foods across tertiles of E-DII scores. Compared with men in the first tertile (E-DII <−0.59), men in the third tertile (E-DII >1) had much lower consumption of all vegetables, fruits, fish, and eggs, but higher consumption of alcohol.

The results of logistic regression analyses showed that higher E-DII scores were significantly associated with increased risk for PCa (Table 4). Specifically, the adjusted OR for overall PCa was 1.25 (95% CI, 1.12–1.40) for continuous E-DII scores. When E-DII scores were categorized into tertiles, the adjusted OR for overall PCa among the second and third tertile was 2.63 (95% CI, 1.61–4.37) and 3.35 (95% CI, 2.06–5.53), respectively ($P_{trend} < 0.001$). Similar positive associations were observed between E-DII and low-moderate PCa (Gleason score 4–7) and high-grade (Gleason score 8–10) PCa ($P_{trend} < 0.01$).

Discussion

The present case-control study found that higher E-DII scores were positively associated with the risk for PCa. The observed association was dose responsive and independent of factors commonly associated with PCa, including age, family history of PCa, BMI, and lifetime physical activity. The results also showed that men with higher E-DII scores consumed fewer vegetables and fruits and lower amounts of fish and eggs, but drank more alcohol. The results of the present study support the hypothesis that PCa risk increases with higher intakes of proinflammatory foods.

The present findings concur with previous case-control studies form Iran [15], Italy [14], Jamaica [17], and Canada [12] and a cohort study in France [16], which showed that higher DII scores were associated with increases of 1.3 to 3.5 times the risk for PCa. In a recent meta-analysis, the pooled relative risk for PCa was 1.52 times higher for elevated DII scores (95% CI, 1.17–1.86) [34]. Although DII was not significantly associated with overall risk for PCa among Mexican men, it seemed to increase the risk for high-grade PCa (Gleason score ≥8) among men in the highest tertile of E-DII (adjusted OR, 1.46; 95% CI, 0.88–2.42) [13]. The present study demonstrated a positive association between E-DII and both low-moderate and high-grade PCa. The difference between the Mexican case-control study and the present one may be ascribed to the high

Table 2
Sample characteristics by tertiles of dietary inflammatory index

	First tertile (<−0.59)	Second tertile (−0.59 to 1)	Third tertile (≥1)	*P*-value*
Age (y), mean (SD)	68.4 (5.9)	68.3 (6.8)	68.1 (6.4)	0.89
Body mass index (kg/m^2), mean (SD)	22.1 (3.3)	22.1 (2.9)	21.7 (3.3)	0.20
Ethanol (g/d), mean (SD)	14.4 (21.6)	12.6 (20.5)	24.7 (52.4)	0.001
Total energy (kcal/d), mean (SD)	2128 (656)	1829 (773)	1964 (880)	0.001
Number of children, n (%)				
≤3	87 (50.3)	120 (52.9)	114 (45.2)	0.41
4–6	67 (38.7)	79 (34.8)	99 (39.3)	
≥7	19 (11)	28 (12.3)	39 (15.5)	
Education level, n (%)				
Primary school	28 (16.2)	44 (19.4)	65 (25.8)	0.005
High school	107 (61.8)	131 (57.7)	157 (62.3)	
Tertiary education	38 (22)	52 (22.9)	30 (11.9)	
Marital status, n (%)				
Married	160 (92.5)	212 (93.4)	236 (93.7)	0.89
Never married or separated	13 (7.5)	15 (6.6)	16 (6.3)	
Smoking habit, n (%)				
Never	42 (24.3)	63 (27.8)	64 (25.4)	0.72
Former	81 (46.8)	104 (45.8)	108 (42.9)	
Current	50 (28.9)	60 (26.4)	80 (31.7)	
Prostate cancer in the first-degree relatives (yes), n (%)	3 (1.7)	2 (0.9)	2 (0.8)	0.61
Lifelong physical activity, n (%)				
Never	94 (54.3)	140 (61.7)	158 (62.7)	0.36
Past active	40 (23.1)	45 (19.8)	42 (16.7)	
Regular	39 (22.5)	42 (18.5)	52 (20.6)	

*P-values were obtained from analysis of variance or χ^2 test.

Table 3
Distribution of food group across tertiles of E-DII

Food groups	DII tertile (servings/d)			*P*-value*
	First tertile (<−0.59)	Second tertile (−0.59 to 1)	Third tertile (≥1)	
All vegetables, mean (SD)	4.1 (2.3)	2.4 (1.3)	1.7 (1)	<0.001
Fruits, mean (SD)	2.5 (1.7)	1.7 (1.4)	1.3 (1.3)	<0.001
Red meat, mean (SD)	0.1 (0.3)	0.1 (0.1)	0.1 (0.2)	0.31
Pork, mean (SD)	1.9 (1.7)	1.6 (2)	1.8 (2.4)	0.32
Poultry, mean (SD)	0.4 (0.5)	0.3 (0.5)	0.4 (1.2)	0.18
Fish, mean (SD)	0.6 (0.7)	0.5 (0.6)	0.4 (0.3)	0.001
Eggs, mean (SD)	0.5 (0.8)	0.3 (0.3)	0.3 (0.4)	<0.001
Coffee, mean (SD)	0.9 (1.1)	1.1 (1.3)	1.0 (1.2)	0.54
Ethanol, mean (SD)	1.3 (1.9)	1.1 (1.8)	2.2 (4.5)	0.001

E-DII, energy-adjusted dietary inflammatory index.
*P values were obtained from analysis of variance.

Table 4
Crude and adjusted odds ratios and associated 95% confidence intervals of prostate cancer risk according to tertiles of energy-adjusted dietary inflammatory index (E-DII)

E-DII			Continuous		First tertile (<-0.59)		Second tertile (-0.59 to 1.0)			Third tertile (≥1.0)		
PCa	Ca	Co	OR*	95%CI	Ca/Co	OR*	Ca/Co	OR*	95%CI	Ca/Co	OR*	95%CI
All	244	408	1.25	1.12–1.4	37/136	1.0	91/136	2.63	1.61–4.37	116/136	3.35	2.06–5.53
											P_{trend} <0.001	
Gleason 4–7†	110	408	1.3	1.13–1.5	12/136	1.0	41/136	3.34	1.66–7.13	57/136	5.29	2.69–11.18
											P_{trend} <0.001	
Gleason 8–10‡	134	408	1.2	1.05–1.37	25/136	1.0	50/136	2.51	1.40–4.63	59/136	2.57	1.43–4.73
											P_{trend} = 0.006	

Ca, cases; Co, controls; PCa, prostate cancer.
*Adjusted for age (year), body mass index (kg/m^2), ethanol consumption (g/day), number of children (≤3, 4–6, and ≥7), education level (primary, high, and tertiary), marital status (married, never married, or separated), smoking habit (never, former, current), PCa in first-degree relatives (yes, no), life-long physical activity (never, past active, regular).
†Low-moderate grade PCa.
‡High-grade PCa.

variation in prevalence of proinflammation diet across populations, in which the Mexican diet appeared to be more proinflammatory and less variable than the Vietnamese diet (mean E-DII of 0.52, SD 1.53, versus 0.20, SD 1.88). Taken together, findings from this study and the literature provide strong support for the role of chronic inflammation in the etiology of PCa and suggest that reducing consumption of proinflammatory foods such as those that are high in fat [35] may lower risk for PCa.

A proinflammatory diet, characterized by high DII scores, has been found to differ across countries [8]. To the best of our knowledge, this is the first study to investigate the relationship between the DII and PCa risk in Southeast Asia. In the present study, the mean E-DII among male controls was 0.20 (SD 1.88), which was relatively low compared with that observed in other populations such as Iranian men (mean E-DII 0.93, SD 1.40) [15] or Mexican men (mean E-DII 0.52, SD 1.53) [13]. In Vietnamese men, vegetable and fruit intake were the major contributors to reducing DII scores, whereas red meat was the predominant factor increasing the DII in Jamaican men [17]. In Vietnamese men, alcohol intake was strongly correlated with DII scores, perhaps because individuals who are less health conscious have high DII scores and high ethanol intake. Higher intakes of ethanol and red meat may play an etiologic role in the development and progression of PCa [36,37]. Generally, less proinflammatory diets among Vietnamese men may be a favorable factor that contributes to a low prevalence of PCa in Vietnam [38].

The association between proinflammatory diets and PCa risk may be explained by their effects on chronic systemic inflammation. Consumption of proinflammatory foods such as meat and butter can increase the levels of inflammatory molecules such as high-sensitivity C-reactive protein, E-selectin, IL-6, tumor necrosis factor-α, and soluble vascular cell adhesion molecules [39,40]. Regular consumption of proinflammatory diets will lead to chronic inflammation, which may initiate PCa through various mechanisms such as by increasing insulin resistance and oxidative stress [41,42] and influencing insulin-like growth factor (IGF)-1 axis. Because PCa is an androgen–sensitive cancer, activation of androgen receptor induces proliferation of prostate cells [43]. It has been demonstrated that insulin resistance, together with IGF signaling, can activate androgen receptor and therefore promote the development of PCa [44]. Chronic inflammation may cause prostate carcinogenesis via inflammatory cells and signaling pathways [45,46]. The initiation and progression of PCa also may be triggered by the expression of inflammatory molecules (e.g., cytokines and IL-6) and their encoded genes [45].

Some study limitations deserve mention when interpreting the results of the present study. First, there may have been selection biases, which could not be avoided because participants were voluntary and not randomly selected from the population. Also, information bias may be another concern; however, its effect may be dampened because all participants were unaware of the study hypothesis. Unlike in other parts of the world, the role of diet and inflammation in the development of PCa has not been widely broadcast in Vietnam, which is consistent with what is known about cancer prevention in ASEAN countries in general [47]. Recall bias may occur if cases recalled their history of dietary habits differently from the controls. To minimize the bias and to improve the accuracy of information obtained, we employed the same well-trained interviewers to conduct direct interviews of both case and control groups using an identical protocol. Information about dietary habits of the study participants was also sought from the

participant's next of kin. Second, we did not measure inflammatory molecules in blood to corroborate results from dietary intake; however, the DII has been construct validated against inflammatory biomarkers in different populations and under varying conditions [48–55]. Although all selected controls had a PSA level ≤4 ng/mL, misclassification of their case–control status is possible, although unlikely given the low incidence of PCa in Vietnam [56]. Lack of data on the stage of PCa and a limited sample size make it difficult to discern the potential heterogeneity across subgroups. However, histopathologic grade is widely thought to be more important than stage in PCa; hence, it was reasonable to use the Gleason score to establish the virulence of the disease [57,58]. Finally, nutrient values were estimated using the US Department of Agriculture's nutrient database [26,27], which may affect the level of inflammatory potential of habitual diet among the study participants. However, the main objective of this study was to assess the association between DII and the risk for prostate cancer, rather than to evaluate the inflammatory capacity of the Vietnamese diet.

Conclusion

This case-control study showed a positive, dose–response association between E-DII scores and PCa risk among Vietnamese men. The results support the hypothesis that a proinflammatory diet increases the risk for PCa.

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