RESEARCH ARTICLE

Editorial Process: Submission:07/21/2017 Acceptance:01/05/2018

Association between Dietary Inflammatory Index and Prostate Cancer in Shiraz Province of Iran

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Abstract

Background: Dietary components like food items and nutrients have been implicated to play a role in modulating inflammation and the development of prostate cancer. Studies examining this association have not been extensively explored in Middle Eastern Countries. **Material and Methods:** We examined the association between the dietary inflammatory index (DIITM) and prostate cancer in the Shiraz province of Iran. A total of 60 incident cases and 60 controls attending the same hospital as the cases were recruited. The energy adjusted DII (E-DIITM) was computed based on dietary intake assessed using a validated food frequency questionnaire (FFQ). Logistic regression was used to estimate odds ratios. **Results:** Men with higher E-DII (>0.96) were at higher risk of prostate cancer (OR = 2.55; 95% CI =1.04–6.23) compared to men with E-DII ≤0.96. **Conclusion:** These data suggest a pro-inflammatory diet, as indicated by increasing DII score, may be a risk factor for prostate cancer in Iranian men.

Keywords: Dietary inflammatory index- diet- inflammation- prostate cancer- case- control - Iran

Asian Pac J Cancer Prev, **19 (2)**, 415-420

Introduction

Prostate cancer is one of the most common cancers among men (Jemal et al., 2011) and the major cause of cancer mortality among males, irrespective of race (Xie and He, 2012). Based on the latest reports, prostate cancer is the third most common cancer among Iranian men and the first one among men in Shiraz ("Iranian Annual Cancer Registration Report", 2011). Current investigation also showed the incidence rate of prostate cancer is rising in Iranian population (Pakzad et al., 2016).

Considerable evidence is accumulating on the role of chronic inflammation in prostate cancer (Kopp et al., 2013; Cross et al., 2005; Nakai and Nonomura, 2013). Chronic inflammation involves continuous recruitment of pro-inflammatory cytokines (associated with increased blood flow to the injured tissue, due to histamine released by damaged mast cells) (Keibel et al., 2009). Consistent with this chronic inflammation hypothesis, innate immunity and inflammation play a modest role in the development of prostate cancer (Kazma et al., 2012) and in the Melbourne Collaborative Cohort Study higher levels of IL-6, a pro-inflammatory cytokine, was seen among malignant prostate cancer cases compared to those with benign disease (Tindall et al., 2012). Additionally, higher levels of inflammatory markers at baseline have been shown to be positively associated with prostate cancer risk (Guo et al., 2013; Toriola et al., 2013). Research into the role of diet in inflammation and prostate cancer suggests that diet represents a complicated set of exposures that often interact, and whose cumulative effect modifies both inflammatory responses and health outcomes. The Dietary Inflammatory Index (DIITM), a tool developed by researchers at the University of South Carolina's Cancer Prevention and Control Program, can be used in diverse populations in order to predict levels of inflammatory markers and inflammation-related health outcomes (Shivappa et al., 2014; Wirth et al., 2014a). The DII is a product of a process involving careful review and scoring of the scientific literature on diet and inflammation, and obtaining data sets from around the world to which individuals' dietary intakes could be compared. The DII is the only dietary index that has been developed based on a comprehensive literature review of the effects of food parameters on biological outcomes; i.e., to determine the inflammatory potential of individuals' diets (Shivappa et al., 2014). The parameters include various nutrients, whole food item and flavonoids (Wirth et al., 2014). The DII has been validated with various inflammatory markers, including C-reactive protein (Shivappa et al., 2014; Hebert et al., 2014), IL-6 (Turner-McGrievy et al., 2015; Wood et al., 2015), and tumor necrosis factor-alpha (Tabung et al., 2015), has been positively associated with risk of prostate cancer in Italy and Jamaica (Shivappa et al., 2015; Shivappa et al., 2015). Thus far, the DII has been found to be associated with inflammatory cytokines including

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CRP, interleukin-6 and homocysteine (Shivappa et al 2013; Wirth et al., 2014b; Wood et al 2014; Tabung et al., 2015), the glucose intolerance component of metabolic syndrome (Wirth et al., 2014b), increased odds of asthma and reduced FEV1 in an Australian population (Wood et al., 2014), shiftwork (Wirth et al., 2014), colorectal cancer (Tabung et al., 2015; Wirth et al., 2015) and pancreatic cancer (Shivappa et al., 2014).

The purpose of this study is to examine the association between the DII and prostate cancer in this case-control study of Iranian men. Our working hypothesis is that higher DII scores (indicating pro-inflammatory diet) increases risk of prostate cancer.

Materials and Methods

Participants

From April to September 2015, 125 patients (62 cases and 63 hospital based controls) were participated in a case-control study in Shiraz, Iran. Both cases and controls were selected from two main hospitals in Shiraz that are referral centers for urological disorders. Demographic and dietary intakes were collected by face-to-face interview and anthropometric indices also were measured.

Cases were newly diagnosed prostate cancer patients (maximum 1 month after diagnosis), who did not have any history of using dietary regimens to treat chronic diseases, diabetes or cancers of other sites. At the same time, controls were selected randomly from patients who visited the same hospitals due to non-neoplastic, non-diabetes conditions. They were admitted to hospital due to eye (n=21), ENT (ear, nose, throat) (n=20), kidney (n=8), nerve (n=5) and gastrointestinal (n=9) problems. As with cases, controls also were required not to be following any dietary regimens for chronic diseases. Cases and controls were matched for body mass index (<19, 19-25, 25-30, >30kg/m2) and age (5-year groups). Total energy intake of <800 or >4,200 kcal/day or poor response to food frequency questionnaire (FFQ) (do not respond to >70 items) were considered as exclusion criteria (Esmaillzadeh and Azadbakht., 2008).

Demographic and Anthropometric assessment

Lifestyle and demographic information were gathered using a questionnaire included questions on smoking (smokers/non-smokers), ethnicity (Fars/Non Fars), job (Employment/Unemployment), education (Illiterate and primary/ Diploma and academic), physical activity (less or never/moderate/high), and aspirin use (Yes/No). Weight was measured by a digital scale in light clothing to the nearest 0.1 kg (Glamor BS-801, Hitachi, China), and height was recorded using a non-stretchable tape measure without shoes to the nearest 0.1 cm. BMI was calculated as weight (kg)/height(m)^2.

Dietary intake assessment

Dietary intake was assessed using a valid and reliable semi-quantitative FFQ that represents the usual intakes of individuals over the past year (Nematy et al., 2013). Briefly, this questionnaire consists of 160 food items, determined based on common average portion sizes within the Iranian population. In order to determine the frequency of consumption of each food item, participants responded to nine categories: "never or less than once a month", "1 to 3 times a month", "once a week", "2 to 4 times a week", "5 to 6 times a week", "once a day", "2 to 3 times a day", "4 to 5 times a day", and "6 times or more a day", and for classifying portions, three sizes: small (half of the defined average use or less), medium (equal to the defined average use), and large (one half of the defined average use or more), were considered.

The FFQ was interviewer-administered. FFQ-derived dietary data were used to calculate DII scores for all participants. A total of 25 food parameters were available from the FFQ and therefore could be used to calculate DII (energy, carbohydrate, protein, total fat, fiber, cholesterol, saturated fat, mono-unsaturated fat, poly unsaturated fat, niacin, thiamin, riboflavin, vitamin B12, vitamin B6, iron, magnesium, selenium, zinc, vitamin A, vitamin C, vitamin D, vitamin E, folic acid, beta carotene and caffeine.).

Data from all FFQs were analyzed using specialized software (Borland Delphi Professional, version 7.0), and results were output to a raw data file. Using Visual Basic 2008 (VB 9.0), these data were analyzed and delivered as a SPSS file which included food items (in gram weights) and daily intakes of energy, macronutrients, fiber, and some micronutrients (vitamin A, vitamin E, folate, and potassium) were assessed using Nutritionist software 4.

The DII is based on literature published through 2010 linking diet to inflammation. Individuals' intakes of food parameters on which the DII is based are then compared to a world standard database. A complete description of the DII is available elsewhere (Shivappa et al., 2014). A description of validation work, including both dietary recalls and a structured questionnaire similar to an FFQ, also is available (Shivappa et al., 2013). Briefly, to calculate DII for the participants of this study, the dietary data were first linked to the regionally representative world database that provided a robust estimate of a mean and standard deviation for each parameter (Shivappa et al., 2014). These then become the multipliers to express an individual's exposure relative to the "standard global mean" as a z-score. This is achieved by subtracting the "standard global mean" from the amount reported and dividing this value by the standard deviation. To minimize the effect of "right skewing" (a common occurrence with dietary data), this value is then converted to a centered percentile score. The centered percentile score for each food parameter for each individual was then multiplied by the respective food parameter effect score, which is derived from the literature review, in order to obtain a food parameter-specific DII score for an individual. All of the food parameter-specific DII scores are then summed to create the overall DII score for every participant in the study (Shivappa et al., 2014). Energy adjusted DII (E-DII) was derived using energy density approach by calculated DII per 1000 kcal.

The E-DII was analyzed both as a continuous variable and as a dichotomous variable, categorized based on the median value of the DII (0.96). DII (as dichotomous) was examined across the following characteristics: age, education, ethnicity, physical activity level, body

mass index (BMI), smoking, use of aspirin and food groups using Student t-test or χ^2 test for continuous and categorical variables, respectively. BMI was calculated from measured weight and height. We also examined the distribution of various food groups across DII categories separately for cases and controls. Odds ratios and 95% confidence intervals (OR; 95% CI) were estimated using logistic regression models, adjusting only for age in the crude model and then fitting a model with additional adjustment for ethnicity, body mass index (BMI), education, physical activity, smoking status, and use of aspirin. The covariates were chosen a priori as they were shown to be risk factors for prostate cancer. Statistical tests were performed using SAS® 9.3 (SAS Institute Inc., Cary, NC); all p values were based on two-sided tests.

The study was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Each participant signed a written informed consent form.

Results

Two cases and three controls were excluded from the study due to poor response to FFQ. So data of 60 cases and 60 controls were included in final analysis. A summary of the characteristics of prostate cancer cases and controls is presented in Table 1. Cases were older and were less physically active than controls. Cases also had significantly higher E-DII scores compared to controls (1.55±1.16 vs 0.93±1.4, p-value=0.008). Participant characteristics by E-DII categories are provided in Table 2. There were few differences in sociodemographic and health behavior characteristics by E-DII categories. Men with DII >0.96 had high BMI and were more likely to be employed. Among controls, men with E-DII >0.96 had lower consumption of apples, parsley, walnut and raw dates; and consumed more carbonated drinks, sugar, hamburger, French fries, rice, and artificial juice compared

Table 1. Characteristics of Prostate Cancer Cases and Controls, Iranian Prostate Case-Control Study

Characteristics	Cases N =60	Controls N =60	P-value
Age, (years): mean ± sd	66.0±9.7	61.4±9.4	0.01
BMI, kg/m^2: mean ± sd	24.8±3.64	25.8±3.5	0.12
E-DII, mean ± sd	1.55±1.16	0.93±1.4	0.0008
Categorical variables			
Ethnicity (%)			0.66
Fars	48 (80.0)	46 (76.7)	
Non Fars	12 (20.0)	14 (23.3)	
Education (%)			0.09
Illiterate and primary	41 (68.3)	32 (53.3)	
Diploma and academic	19 (31.7)	28 (46.7)	
Job (%)			0.58
Employed	34 (56.7)	37 (61.7)	
Unemployed	26 (43.3)	23 (38.3)	
Aspirin use (%)			0.26
Yes	10 (16.7)	15 (25.0)	
No	50 (83.3)	45 (75.0)	
Smoking (%)			0.67
Non-smoker	46 (76.7)	44 (73.3)	
Current smoker	14 (23.3)	16 (26.7)	
Physical activity (%)			0.02
Less or never	23 (38.3)	12 (20.0)	
Moderate	25 (41.7)	24 (40.0)	
High	12 (20.0)	24 (40.0)	

Table 2. Participant Characteristics by Level of Dietary Inflammatory Index (DII), Iranian Prostate Case- Control Study

Continuous variables (mean ± SD)	DII≤0.96	DII>0.96	P-Value [a,b]
Age, (years): mean ± sd	61.1±8.8	61.7±10.2	0.82
BMI, kg/m^2: mean ± sd	25.4±3.1	26.3±3.1	0.35
Categorical variables:			
Ethnicity (%)			0.22
Far	21 (70.0)	25 (83.3)	
Non Fars	9 (30.0)	5 (16.7)	0.67
Education (%)			0.6
Illiterate and primary	15 (50.0)	17 (56.7)	
Diploma and academic	15 (50.0)	13 (43.3)	0.83
Job (%)			0.06
Employed	15 (50.0)	22 (73.3)	
Unemployed	15 (50.0)	8 (26.7)	
Aspirin use (%)			0.76
Yes	8 (26.7)	7 (23.3)	
No	22 (73.3)	23 (76.7)	
Smoking (%)			1
Nonsmoker	22 (73.3)	22 (73.3)	
Current smoker	8 (26.7)	8 (26.7)	
Physical activity (%)			0.78
Less or never	5 (16.7)	7 (23.3)	
Moderate	13 (43.3)	11 (36.7)	
High	12 (40.0)	12 (40.0)	

[a] Student t-test was used for continuous variables; [b] Chi-square test was used for categorical variables.

Table 3. Distribution of Intake of Specific Categories of Food Across E-DII, Iranian Prostate Case- Control Study

Food groups g/ week	Controls (N=60)		
	DII≤0.96 (N=30)	DII>0.96 (N=30)	P-value
(mean ± SD) a			
Apple	169.4 ±81.3	118.4 ±74.6	0.01
Carbonated drinks	7.0 ±12.6	30.2 ±37.5	0.002
Sugar	10.4 ±10.3	13.4 ±11.3	0.29
Hamburger	1.6 ±6.2	8.2 ±16.3	0.04
French fries	13.6 ±22.4	21.1 ±25.6	0.23
Rice	231.7 ±79.2	279.1 ±73.6	0.02
Parsley	7.3 ±5.1	4.6 ±2.6	0.01
Walnut	7.5 ±5.8	3.7 ±4.2	0.005
Artificial juice	4.9 ±9.7	14.9 ±26.4	0.06
Raw dates	16.1±19.3	7.2±7.2	0.02

[a] Student t-test was used

Table 4. Odds Ratios and Confidence Intervals for the Association between E-DII and Prostate Cancer, Iranian Prostate Case- Control Study

	Energy Adjusted-Dietary Inflammatory Index OR (95% CI)		P-Value	E-DII (Continuous) OR (95% CI)	P-Value
DII	DII≤0.96	DII>0.96			
Cases / controls	15/30	45/30		60/60	
Age-adjusted	1 (ref.)	2.77 (1.26, 6.10)	0.01	1.42 (1.05, 1.92)	0.02
Multivariate-adjusted [a]	1 (ref.)	2.60 (1.05, 6.41)	0.04	1.29 (0.91, 1.81)	0.15

[a], Adjusted for age; ethnicity; body mass index (BMI); education, physical activity, smoking status, and use of aspirin.

to men with E-DII≤0.96 (Table 3).

Odds ratios (OR) and 95% confidence intervals (CI) for the risk of prostate cancer according to cut-points of E-DII are shown in Table 4. Results obtained from modeling E-DII as a continuous variable in relation to risk of prostate cancer showed a positive association after adjustment for age (OR=1.42; 95% CI=1.05-1.92) and in the multivariable analyses (OR=1.29; 95% CI=0.91-1.81). When analysis was carried out with DII expressed as a dichotomous variable, and adjusting for age, men with E-DII score > 0.96 were at 2.8 times higher odds of having prostate cancer compared to men with E-DII ≤0.96 (ORDII (> 0.96/≤0.96) =2.77; 95% CI=1.03-4.30). After multivariable adjustment, men with E-DII >0.96 were at 2.6 times higher risk of having prostate cancer compared to men with E-DII ≤0.96 (ORDII (> 0.96/≤0.96) =2.60; CI=1.05-6.41).

Discussion

Using data from a case-control study on diet and prostate cancer conducted in Iran, we observed that consuming a more pro-inflammatory diet, as reflected in higher DII scores, was associated with increased risk of prostate cancer. We also observed that cases who reported consuming a more pro-inflammatory diet (DII>0.96) consumed fewer fruits and vegetables and consumed more carbonated drinks, sugar, and French fries. This result supports the hypothesis that men with a pro-inflammatory diet are at higher risk of developing prostate cancer (Kazma et al., 2012). Previously, in case-control studies in Italy and Jamaica, we found that increasing DII was associated with increased risk of prostate cancer (Shivappa et al., 2015; Shivappa et al., 2015). However, no association was observed in a Mexican case-control study (Vazquez-Salas et al., 2016). In a French cohort study, DII were positively associated with prostate cancer risk [quartile (Q) 4 compared with Q1, HR: 2.08; 95% CI: 1.06, 4.09] (Graffouillere et al., 2016). Higher DII scores also have been shown to be associated with decreased prostate cancer survival in an Italian study (HR highest vs. lowest DII tertile: 4.01; 95% CI: 1.25-12.86) (Zucchetto et al., 2016). Application of the dietary inflammatory index (DII) in various populations addresses the causal criterion of "consistency." Even though we have previously published five manuscripts on DII and prostate cancer, the population in this study is very different from those other populations. These results show that the DII as a tool can be applied to a variety of populations, using any competent dietary assessment tool including different types of FFQ. Previously, studies have been conducted to examine various dietary patterns and indices and their association with prostate cancer in men (Jackson et al., 2013; Bosire et al., 2013). In an Iranian case-control study, analyses were conducted looking at fruits and vegetable consumption and prostate cancer risk and it was observed that fruits and vegetable intake were inversely associated with prostate cancer risk (Askari et al., 2014b) and in another study an increased risk of prostate cancer was observed with the higher Western dietary pattern scores, which is characterized by high consumption of pro-inflammatory food items such as solid fat and sweets (Askari et al., 2014a). Previous studies also have shown higher consumption of fruits and cruciferous vegetables to be associated with reduced prostate cancer risk (Giovannucci et al., 2003; Schuurman et al., 1998). In a cohort study conducted among retired professionals inverse associations were observed between the Healthy Eating Index-2005 (HEI-2005), Alternate Healthy Eating Index-2010 (AHEI-2010), and prostate cancer risk (Bosire et al., 2013). Typically, people with higher scores within each of these patterns demonstrate a style of eating which most individuals would recognize as "nutritious;" i.e., more of anti-inflammatory food and less of pro-inflammatory food. For example, someone eating in a manner consistent with the high HEI-2005 score would consume a diet high in fruit and vegetables, and fish; and it would be low in red meat and sugars.

There are several mechanisms that are affected by increasing systemic inflammation; thus, a pro-inflammatory diet has an indirect effect in increasing insulin resistance (Esmaillzadeh et al., 2007; Festa et al., 2000) and oxidative stress (Vykhovanets et al., 2011) and this could be one of the mechanisms for the observed association. Consumption of food items such as meat and butter have been shown to increase levels of high-sensitivity C-reactive protein, E-selectin and soluble vascular cell adhesion molecule-that, in turn, increase systemic inflammation (Esmaillzadeh et al., 2007) which then is responsible for increasing insulin resistance (Festa et al., 2000). The influence of diet on cancer is difficult to measure precisely, and challenges in dietary exposure assessment are greatest in case-control studies. A normal human diet consists of both pro-inflammatory and anti-inflammatory food parameters. Hence, the DII, which takes in to account the full spectrum of inflammation-modulating food components, may more accurately reflect the relationship between diet and cancer risk than would individual nutrients.

Some of the strengths of our study are that it is one of the few studies that have looked at the association between diet as a whole and prostate cancer in Iran. Even with a

relatively small sample size we have observed significant results, which indicate the importance of consuming anti-inflammatory diet in protecting against prostate cancer. We used incident cases interviewed before they were made aware of their disease status; and in this manner avoided recall and interviewer bias. This approach strengthened the validity of the results.

Our study has several limitations. First, although we used a validated food-frequency questionnaire (FFQ) for assessing the dietary intake, measurement errors that might distort or obscure associations were inevitable. Even though diet in mid-life may be more important than the diet later in life, the long time passed from the patients' mid-life restricts our ability to evaluate that time period. In this study we measured diet during the past year to avoid the measurement errors and assumed that the low probability of changing diet in adulthood would work in our favor (Mikkila et al., 2007). However the use of current diet as a surrogate for past diet provides almost the similar information in some instances (willett, 1998). No data were available on inflammatory markers in this study; hence, the DII could not be validated with inflammation in this case-control study. We could not adjust our risk estimates for potentially confounding effects of family history of prostate cancer and access to health care because information about these exposures was not gathered at baseline (thus resulting in the potential for residual confounding). Family history of prostate cancer in particular has been consistently shown to be a risk factor for prostate cancer and including this variable as a covariate may have attenuated the association. Small sample size also is another limitation that might produce unstable risk estimates with wide confidence intervals. Notwithstanding the design limitations of case-control studies in general, we believe that our findings of a positive association between DII with prostate cancer are plausible and could be related to immune and hormonal factors (Vykhovanets et al., 2011; Pandey and Gupta, 2009; Kaaks and Lukanova, 2001).

The logical next step would be to use DII scores to predict incidence of other cancers and serum level of inflammatory markers in Iran and to look at other outcomes that are related to diet and inflammation such as cardiovascular diseases. The results from the current study are restricted to men, so using DII in studies with women would help to discern the generalizability of DII across genders, though clearly for outcomes other than prostate cancer.

In conclusion, the results of this study indicate that a pro-inflammatory diet, as indicated by increasing DII score, may be a risk factor for prostate cancer in Iranian men. Future studies are needed to gain insight into the relationship between DII and the risk of prostate cancer aggressiveness; this would deepen understanding about the role of diet in determining extent and virulence of prostate cancer.

Conflict of interest

Dr. James R. Hébert owns controlling interest in Connecting Health Innovations LLC (CHI), a company planning to license the right to his invention of the dietary inflammatory index (DII) from the University of South Carolina in order to develop computer and smart phone applications for patient counselling and dietary intervention in clinical settings. Dr Nitin Shivappa is an employee of CHI.

Acknowledgements

Drs. Shivappa and Hébert were supported by grant number R44DK103377 from the United States National Institute of Diabetes and Digestive and Kidney Diseases.

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