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Increased inflammatory potential of diet is associated with increased odds of prostate cancer in Argentinian men

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Abstract

Purpose Various aspects of diet, including specific food items and nutrients, have been shown to modulate inflammation and have been implicated in the etiology of prostate cancer (PrCA). No study examining the role of diet-associated inflammation in PrCA has been conducted in Latin America.

Method We examined the association between the Dietary Inflammatory Index (DII®) and PrCA in a population-based case–control study in Córdoba, Argentina. A total of 153 incident cases of PrCA and 309 controls frequency matched on sex, age (±5 years), and place of residence were recruited from 2008 to 2015. The DII was developed to determine the inflammatory potential of individuals' diets and was computed from a validated food frequency questionnaire using nutrient data from diet only. Multi-level logistic regression models were fit to evaluate the association between DII scores and PrCA, adjusting for age, body mass index, energy intake, and occupational exposure as first-level covariates and family history of prostate cancer as the second-level variable. Odds ratios were estimated in all subject and stratified by BMI (< 30 vs. ≥ 30 kg/m^2).

Results Men in the most pro-inflammatory group (tertile 3) had 50% higher odds of having PrCA compared to men in the most anti-inflammatory group (tertile 1) ($OR_{tertile3\ vs.\ tertile1}$ 1.50; 95% CI 1.24–1.80). The odds of prostate cancer were higher in obese men ($n = 109$, $OR_{tertile3\ vs.\ tertile1}$ 1.81; 95% CI 1.45–2.27), while no association was found among non-obese men ($n = 375$, $OR_{tertile3\ vs.\ tertile1}$ 0.93; 95% CI 0.25–3.51).

Conclusions A pro-inflammatory diet, reflected by higher DII scores, was positively associated with PrCA occurrence. Based on these results and those from other studies, steps should be taken to promote a diet rich in anti-inflammatory foods, in order to reduce risk of PrCA and other chronic diseases. Future studies should explore this association in a prospective setting.

Keywords Dietary Inflammatory Index · Prostate cancer · Case–control · Argentina

Introduction

Prostate cancer (PrCA) is the second most commonly diagnosed non-skin cancer [1]. Data on PrCA incidence in Argentina are sparse; however, reports from GLOBOCAN, 2012 shows that, PrCA is the most frequently diagnosed cancer and it is the third most common cause of cancer death among Argentinean men [2]. Analyses indicating socioeconomic and geographical variation in cancer incidence rates in Córdoba (Argentina) have motivated the study of possible lifestyle and environmental factors involved in the development of PrCA, including diet [3]. Chronic inflammation, a persistent state of low-grade systemic inflammation in which tissue destruction and repair occur simultaneously [4, 5] and involving continuous recruitment of pro-inflammatory cytokines (associated with increased blood flow to

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the injured tissue due to histamine released by damaged mast cells) [6], has been shown to play a major role in the development of PrCA [7–9].

Consistent with the inflammation-PrCA hypothesis, several studies have shown chronic inflammation to be associated with PrCA [10]. Additionally, chronic inflammation, as measured by levels of inflammatory markers at baseline, has been shown to be positively associated with PrCA risk [11, 12]. Chronic inflammation increases insulin resistance, which leads to increased circulating levels of insulin that, in turn, have been associated with the development of PrCA by inhibiting apoptosis and stimulating cell proliferation [13].

There is growing evidence that specific dietary components influence both acute and chronic inflammation [14–17]. Although many studies have been conducted to discern the relationship between diet and PrCA, results are inconclusive [18–20]. Research into the role of diet in inflammation and PrCA suggests that diet represents a complicated set of exposures that often interact, and whose cumulative effect modifies both inflammatory responses and health outcomes [21]. The Dietary Inflammatory Index (DII®), 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 related health outcomes [22, 23]. A higher DII score indicates a pro-inflammatory dietary milieu rich in nutrients like saturated fat and total cholesterol; a lower DII score indicates that diet is more anti-inflammatory, rich in nutrients such as vitamins, minerals, and flavonoids [22].

Thus far, the DII has been found to be associated with inflammatory cytokines including C-reactive protein (CRP), interleukin-6, and homocysteine [23–27]. In the first validation study higher DII scores were associated with values of hs-CRP > 3 mg/l (OR 1.08; 95% CI 1.01, 1.16, $p = 0.035$) [23]. In another study, conducted in Iran, for every 1-unit increase in DII, there was a corresponding increase in interleukin-6 of 0.15 pg/ml, 95% CI (< 0.01, 0.28) [28]. In a cross-sectional study conducted in Belgium, multivariable analyses showed significant positive associations between the DII and the inflammatory markers IL-6 (> 1·6 pg/ml) (OR 1.19, 95% CI 1.04, 1.36) and homocysteine (> 15 µmol/l) (OR 1.56, 95% CI 1.25, 1.94) [29]. The DII also has been associated with the glucose intolerance component of metabolic syndrome [24], increased odds of asthma in an Australian population [25], shiftwork [30], cardiovascular disease [31], colorectal cancer [32–34], gastric cancer, [35] and pancreatic cancer [36]. Previous research among Cordoba, Argentinian men showed that those with a higher adherence to the traditional dietary pattern characterized by high consumption of fatty red meats, offal, processed meat, starchy vegetables, added sugars and sweets, and vegetable oils (OR 2.82, 95% CI 1.57–5.10) and a carbohydrate pattern (OR 2.14, 95% CI 1.47–3.13) showed increased odds for PrCA [37].

The DII-PrCA association has been examined in Italy, Jamaica, Iran, Mexico, France, and Canada [38–44]. However, this association has not been explored in a Latin American country where the dietary habits are very different from other regions [45–47] . The objective of this case–control study, conducted in Cordoba, Argentina, was to examine if increasing DII scores are associated with increased odds of prostate cancer. Our working hypothesis is that higher DII scores (indicating a pro-inflammatory diet) increases odds of developing PrCA in this Argentinian population.

Methods

Full details regarding the design of this case–control study have been published elsewhere [37]. In brief, this study was conducted within the framework of the Environmental Epidemiology of Cancer in Córdoba (EECC) project. It was conducted from January 2008 to December 2015 in Córdoba, the second most populated Argentinean province (3,067,000 inhabitants, per the 2010 census), located in the center of the country. Cases were men with incident, histologically confirmed PrCA (ICD-10th Edition, ICIE10:C61) with no previous diagnosis of cancer at other sites. Cases were identified in public and private health institutions and registered at the Córdoba Tumor Registry (CTR). Controls were selected based on geographical residence. Two controls per case, frequency matched on age (± 5 years), were chosen from blocks randomly selected from the same neighborhoods and time-period as cases, and were included in the study after verifying the absence of any neoplastic diseases. Cases and controls with diseases (e.g., diabetes, cardiovascular diseases, celiac disease, renal insufficiency) that may generate a long-term modification of dietary habits where excluded. A total of 153 men with PrCA aged 48–89 (median age 72) years and 309 controls aged 46–89 (median age 71) years were included (response rate 91% in cases and 89% in controls). Subjects interviewed were from rural (54%) and urban (46%) areas (including the most populated area, Córdoba City, with 1,300,000 inhabitants). This study was conducted per the guidelines laid down in the Declaration of Helsinki and its later amendments. In addition, specific national laws have been observed. All procedures involving human subjects were approved by the Ethical Committee of the Faculty of Medical Sciences, University of Córdoba. Written informed consent was obtained from all subjects.

Subject information

All participants were interviewed at home by centrally trained and routinely supervised nutritionists. A structured questionnaire was completed including information about sociodemographic characteristics, occupational history, smoking habits, alcohol consumption, self-reported anthropometric characteristics, physical activity, medical insurance, personal medical history, and family history of cancer. To assess dietary exposure, a validated Food Frequency Questionnaire (FFQ) of 127 items [48] was completed. Subjects were asked about their dietary intake over the 5 years prior to diagnosis (cases) or interview (controls). The FFQ was coupled with a validated photographical atlas based on standard portion sizes in Argentina [49]. The seasonal pattern of consumption of each vegetable or fruit also was taken into account by averaging across all days of the year foods reported to have been consumed in a particular season. Physical activity was measured by means of the International Physical Activity Questionnaire [50]. Frequency and duration of physical activity were then expressed as metabolic equivalent of tasks (METs). Frequency, duration and intensity of physical activity were then expressed as METs. Subsequently, METs were categorized into low (< 600 METs), moderate (600–1,500 METs), and high (> 1,500 METs) categories of physical activity intensity.

Dietary Inflammatory Index (DII®)

The development [22] and validation [23] of the DII has been explained elsewhere. Through evaluation of peer-reviewed literature published from 1950 up to 2010, the score is based on 1,943 articles that identified 45 individual nutrient, food, or flavonoid intake parameters in relation to these six established inflammatory biomarkers: IL-1b, IL-4, IL-6, IL-10, tumor necrosis factor-α (TNF-α) and CRP. Points were assigned to each of these parameters according to whether they increased (+ 1), decreased (− 1) or had no (0) effect on the six established inflammatory biomarkers. The score for each of the DII components was weighted according to the study designs and total number of research articles. Overall parameter-specific inflammatory effect scores were then calculated based on the ratio of the total weighted number of articles to the weighted pro- and anti-inflammatory articles for each parameter followed by subtracting the anti- from the pro-inflammatory fraction. Parameters which had a robust pool of literature, i.e., greater than the median number of 236 weighted articles, were assigned the full value of that score. Parameters with a number of weighted articles < 236 were adjusted according to the distance of their number from this median.

Actual dietary intake data from the FFQ were adjusted against a reference global daily mean and standard deviation intake for each parameter to obtain a *Z* score. These, in turn, were converted to proportions (i.e., with values from 0 to 1) to prevent problems with right skewing of the data (as often occurs with dietary data). They were then centered on zero by doubling and subtracting 1. The global intake database was based on consumption data from 11 countries in different parts of the world. The centered proportion for each intake parameter was multiplied by its respective parameter-specific inflammatory effect score. All of the DII component-specific DII scores were then summed to create the overall DII score for each participant in the study, $DII = b_1 \times n_1 + b_2 \times n^2 + ... + b_{22} \times n_{22}$, where b_i ($i = 1,..., 22$) refers to the literature-derived inflammatory effect scores for each of the evaluable DII components and n_i refers to the DII component-specific centered percentiles, which were computed from the FFQ-derived dietary data. DII density scores, which adjust for energy intake and use an energy-adjusted global comparison database, also were calculated; prior to standardization with the global intakes the parameters were each converted to a per 1,000 kcal consumption value and the parameter of energy intake was excluded. The steps involved in DII calculation are shown in Fig. 1.

For the current study, data on 22 of the 45 DII components could be derived from the FFQ and were thus used for calculating the DII scores. These include: pro-inflammatory components (carbohydrate, protein, fat, saturated fat, iron, cholesterol) and anti-inflammatory components (alcohol, fiber, mono-unsaturated fat, poly-unsaturated fat, omega-3, omega-6, niacin, thiamin, riboflavin, magnesium, zinc, vitamin A, vitamin C, vitamin E, garlic, and onions). Data on these components were derived from diet only.

Statistical analysis

A Chi square test was used to compare categorical variables. In order to evaluate associations between DII and PrCA, odds ratios and 95% confidence intervals (OR; 95% CI) were estimated using multi-level logistic regression (MLR) models (31) for the binary response (1 if a PrCA case, 0 if a control). On this basis, a hierarchical structure in the data was assumed. Subjects were included in a first level, in order to capture the inter-individual variability and to assess individual-level variable effects such as the DII score in relation to the outcome. These were then nested into a second level, the family history of cancer, defined according three categories, first- or second-degree relatives with PrCA, first- or second-degree relatives with any other cancer, or no family history of cancer. Family history of cancer was considered as an underlying or latent variable referring to the contextual (family) dimension. Although it depicts a random variable whose manifestations are hidden from us, its approximation could be represented with a self-reported scale, like the one

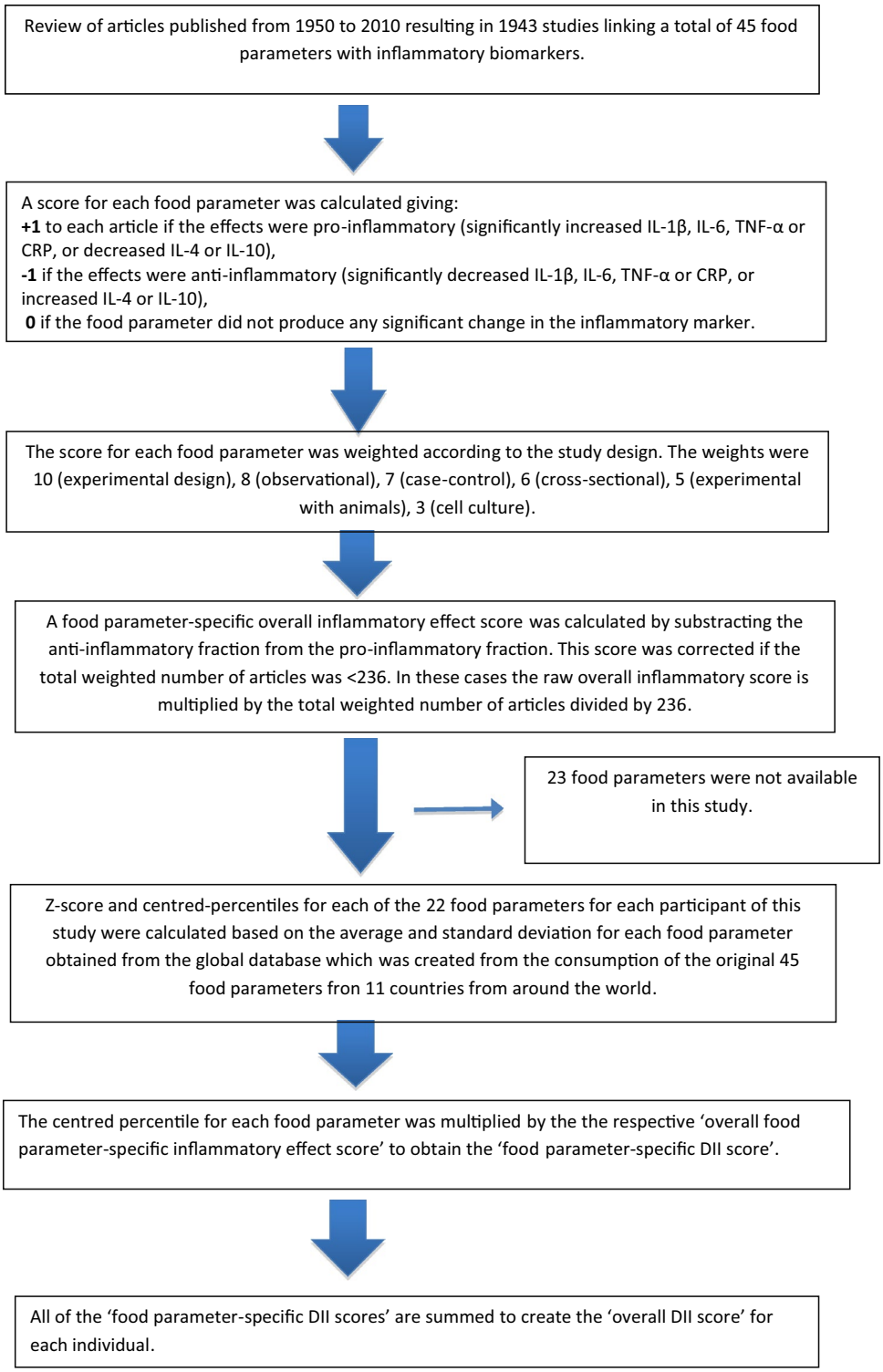

Fig. 1 Sequence of steps in creating the DII in the Argentinian prostate cancer case–control study

used in the questionnaire employed here. Including latent variables, typically referred to as random effects, in statistical models is a common way of taking unobserved heterogeneity into account. Then, we assumed fixed effects for all covariates (DII score, age, BMI, etc.) and a random variable to control for underlying clustering information coming from the family history of cancer. This model combined the classical logistic model with a variance component in order to capture variance coming from a latent variable.

DII score (as continuous variable or categorized into tertiles, based on controls cutpoints), age (as continuous variable), body mass index [BMI = weight(kg)/height(m)^2, as

continuous variable], energy intake (as continuous variable), and occupational exposure (two or more years of industrial exposure to chemical contaminants recognized by IARC as carcinogens—i.e., dyes, paints, textiles, plastics, rubber, leather, herbicides, automotive, chemical, coal) were included as first-level covariates. The risk measures were estimated in all subjects and stratified by BMI (< 30 vs. ≥ 30 kg/m^2) as well. SAS® 9.3 was used for DII calculation and Stata® 14.2 software was used for statistical analysis. $\alpha < 0.05$ was used as the criterion for assessing statistical significance.

Results

A summary of the characteristics of PrCA cases and controls is presented in Table 1. Cases and controls had a similar distribution of socioeconomic status, occupational exposure, smoking habits, physical activity, BMI, and energy intake. Compared to controls, cases had more frequently a lower educational level ($p = 0.049$) and a family history of PrCA ($p < 0.001$) (Table 1).

The mean value of the DII score was + 1.47 (SD 1.13) indicating a slightly pro-inflammatory diet overall in this study compared to the mean DII in the previous study in Argentina, which looked at the association between DII and colorectal cancer where the mean DII was + 1.24 (SD 1.26); [51] and scores ranged from − 1.94 (most anti-inflammatory score) to + 3.82 (most pro-inflammatory score). Participant characteristics by DII categories are provided in Table 2. Reported intake of energy, fat, red meat, and alcohol increased across tertiles of DII and the proportion of men with family history of PrCA is higher in tertiles II and III (Table 2).

Odds ratios (OR) and 95% confidence intervals (CI) for having been diagnosed with PrCA according to DII score are shown in Table 3. Increasing DII score (as a continuous variable) corresponding to 17% increase in DII in the current study showed significant positive associations with PrCA (OR 1.12; 95% CI 1.06–1.95) after adjusting for age, BMI, energy intake, and occupational exposure at the individual level, and fitting the family history of any cancer as the clustering variable. When the DII score was categorized into tertiles, the third tertile showed a significant effect, increasing odds of PrCA (OR 1.50; 95% CI 1.24–1.80) compared to the first tertile (Table 3).

Results obtained within strata of BMI (< 30 vs. ≥ 30 kg/m^2) are presented in Table 4. No association was found among men with BMI < 30 kg/m^2 ($OR_{continuous}$ 0.90; 95% CI 0.61–1.34 and $OR_{tertile3\ vs.\ tertile1}$ 0.93; 95% CI 0.25–3.51), while higher DII scores, were positively associated with PrCA occurrence in obese men (BMI ≥ 30 kg/m^2), with higher ORs than in all subjects without stratifying ($OR_{continuous}$ 1.22; 95% CI 1.08–1.38 and $OR_{tertile3\ vs.\ tertile1}$ 1.81 95% CI 1.45–2.26).

Table 1 Characteristics of cases and control subjects in the EECC case–control study of prostate cancer, Córdoba, Argentina (2008–2015)

	Cases ($n = 163$) Subjects (%)	Controls ($n = 324$) Subjects (%)
Age (years)		
≤ 50	1 (0.61)	4 (1.23)
51–60	14 (8.59)	32 (9.88)
61–70	49 (30.06)	112 (34.57)
> 70	99 (60.74)	176 (54.32)
Socioeconomic status		
Low	37 (22.70)	84 (25.93)
Middle	55 (33.74)	106 (32.72)
High	65 (39.88)	124 (38.27)
Unknown	6 (3.86)	10 (3.09)
Educational level*		
Low	36 (22.09)	51 (15.74)
Middle	75 (46.01)	149 (45.99)
High	46 (28.22)	111 (34.26)
Unkwon	6 (3.68)	13 (4.01)
Family history of PC*		
No	137 (84.05)	307 (94.75)
Yes	26 (15.95)	17 (5.25)
Unknown	–	–
Occupational exposure[a]		
No	107 (68.15)	227 (72.52)
Yes	50 (31.85)	86 (27.48)
Smoking habits		
No	55 (33.74)	101 (31.17)
Yes	108 (66.26)	223 (68.83)
Lifetime PA		
Low	70 (42.94)	128 (39.51)
Middle	37 (22.70)	88 (27.16)
High	56 (34.36)	108 (33.33)
BMI		
≤ 24.9	36 (22.09)	91 (28.09)
25–29.9	90 (55.21)	158 (48.77)
≥ 30	36 (22.09)	73 (22.53)
Unknown	1 (0.61)	2 (0.62)
Energy intake[b]		
Low	45 (27.61)	108 (33.33)
Middle	55 (33.74)	108 (33.33)
High	63 (38.65)	108 (33.33)

NSAID nonsteroidal anti-inflammatory drugs, *PC* prostate cancer, *BMI* body mass index

*$p < 0.05$ as level of significance

[a]Exposure to chemical contaminants for 2 years or longer

[b]Categories based on tertiles of intake in controls

Table 2 Characteristics of all subjects by tertiles of DII, in the EECC case–control study of prostate cancer, Córdoba, Argentina (2008–2015)

	Tertiles of DII		
	I (DII < 0.98) $n = 154$	II (DII 0.99–1.96) $n = 161$	III (DII > 1.96) $n = 172$
Age (years)[a]	71.89 (8.46)	69.85 (8.12)	69.31 (8.43)
Current BMI (kg/m^2)[a]	27.04 (3.13)	27.49 (3.85)	27.69 (3.75)
Usual BMI (kg/m^2)[a]	27.11 (3.46)	27.42 (3.91)	27.71 (4.04)
Smoker (%)	64.29	68.32	70.93
Family history of PC (%)	6.49	10.56	9.30
Occupational exposure (%)[b]	30.00	32.68	24.55
Vigorous or moderate PA (%)[c]	51.30	53.42	63.37
Energy intake (kcal)[a]	3,049.78 (987.89)	3,379.89 (1,021.50)	4,297.96 (1,532.72)
Fat intake (g)[a]	113.49 (56.75)	135.86 (57.92)	197.86 (95.99)
Red meat intake (g)[a]	176.85 (114.70)	224.77 (130.03)	325.78 (194.62)
Alcohol intake (g)[a]	21.76 (22.88)	23.53 (25.38)	29.53 (43.25)

DII Dietary Inflammatory Index, *BMI* body mass index, *PC* prostate cancer, *PA* physical activity

[a]Mean (standard deviation)

[b]Exposure to chemical contaminants for 2 years or longer

[c]Subjects who performed regular physical activity reaching at least 600 METs by min/week

Table 3 Associations between the DII and prostate cancer from multi-level logistic modeling in the EECC case–control study of colorectal cancer, Córdoba, Argentina (2008–2015)

	Subjects	Crude OR (95% CI)	Adjusted[a] OR (95% CI)
DII			
Continuous		1.11 (0.94–1.32)	1.12 (1.06–1.20)**
Tertile I	46/108	1 (ref)	1 (ref)
Tertile II	53/108	1.15 (0.71–1.85)	1.10 (0.61–1.96)
Tertile III	64/108	1.39 (0.87–2.21)	1.50 (1.24–1.80)**

OR odds ratio, *CI* confidence interval

**$p < 0.001$ as level of significance

[a]Age, usual BMI, energy intake and occupational exposure were included as covariates at first level, and family history of cancer at second level

Table 4 Associations between the DII and prostate cancer stratified by body mass index (< 30 vs. ≥ 30 kg/m^2) from multi-level logistic modeling in the EECC case–control study of colorectal cancer, Córdoba, Argentina (2008–2015)

	Subjects	Crude OR (95% CI)	Adjusted OR (95% CI)
DII			
BMI < 30 kg/m^2			
Continuous		0.92 (0.66–1.29)	0.90 (0.61–1.34)
Tertile I	12/28	1	1
Tertile II	11/31	0.83 (0.32–2.17)	0.73 (0.51–1.06)
Tertile III	13/32	0.95 (0.37–2.41)	0.93 (0.25–3.51)
BMI ≥ 30 kg/m^2			
Continuous		1.19 (0.97–1.45)	1.22 (1.08–1.38)*
Tertile I	34/80	1	1
Tertile II	42/77	1.28 (0.74–2.22)	1.25 (0.66–2.38)
Tertile III	51/76	1.57 (0.92–2.67)	1.81 (1.45–2.27)**

OR odds ratio, *CI* confidence interval, *BMI*, body mass index

*$p < 0.005$; **$p < 0.001$ as levels of significance

[a]Age, energy intake, body mass index and occupational exposure were included as covariates at first level, and family history of cancer at second level

Discussion

In this case–control study of Argentinian males, consuming a more pro-inflammatory diet, as reflected by higher DII scores, was associated with increased odds of PrCA. This effect was more pronounced among obese men. Hence, these results confirm our hypothesis and are consistent with findings from other studies. For example, in a large case–control study in Italy, participants in quartiles 3 and 4 relative to quartile 1 of the DII were at higher odds of having PrCA ($OR_{Quartile3\ vs.1}$ 1.32, 95% CI 1.03–1.69 and $OR_{Quartile4\ vs.\ 1}$ 1.33, 95% CI 1.01–1.76; $P_{trend} = 0.04$) [38]. In another case–control study in Jamaica, men in the highest quartile of the DII were at higher odds of having PrCA ($OR_{Quartile4\ vs.\ 1}$ 2.39; 95% CI 1.14–5.04) [39]. In an Iranian case–control study men with higher DII score (> 0.23) were at higher odds of having PrCA (OR 3.96; 95% CI 1.29–12.16) compared to men with lower DII scores (≤ 0.23) [40]. A positive association of similar magnitude was observed in a prospective study in France ($HR_{Quartile4\ vs.\ 1}$ 2.08, 95% CI 1.06–4.09) [42]. These results show that the DII can be applied to widely varying populations, using any competent dietary assessment tool, including different types of validated FFQs, to predict PrCA.

Furthermore, results obtained are very consistent across widely varying populations. We observed stronger association among obese men; this is along expected lines, because obesity itself is both a risk factor for PrCA [52] and is a pro-inflammatory condition [53]. However, it also is conceivable that the effect of obesity could countervail that of diet and it simply did not do so in this study.

Previous results from this same study found that two different patterns increased odds of having PrCA. The Traditional pattern (fatty red meats, offal, processed meat, starchy vegetables, added sugars and sweets, candies, fats, and vegetable oils) nearly tripled the odds (OR 2.82, 95% CI 1.57–5.10); and the carbohydrate pattern (sodas/juices and bakery products) more than doubled the odds (OR 2.14, 95% CI 1.47–3.13) [37].

Other studies conducted to examine the association between various dietary patterns and PrCA have shown increased risk with unhealthy patterns of intake [54, 55]. In a systematic review of the epidemiological studies on diet and risk of PrCA focusing on those carried out in South America, suggested that dairy products, red meat, processed meat, α-linolenic fatty acids, as well as dietary patterns characterized by higher intakes of red and processed meat, eggs, and grains may play a role in the development of PrCA [46].

One of the possible mechanisms responsible for the association observed in this study is the effect of a pro-inflammatory diet on systemic inflammation and insulin resistance [56, 57]. 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 [56], which then is responsible for increasing insulin resistance [57]. Increasing insulin resistance leads to increased circulating levels of insulin, which has been demonstrated to play a role in the development of PrCA by inhibiting apoptosis and stimulating cell proliferation [13].

A normal human diet consists of both pro-inflammatory and anti-inflammatory DII components. Hence, the DII, which takes into account the full spectrum of inflammation-influencing food components in all peer-reviewed publications through the year 2010, more accurately reflects the relationship of diet in relation to cancer risk than would individual nutrients.

In keeping with the intention of accounting for heterogeneity of responses coming from a second level of aggregation of data, we used a modeling approach that included the family history of cancer as a clustering variable. Family history of cancer was selected based on the known heritability of this disease derived from either genetic susceptibility [58] or exposure to common environmental factors [59], or both. In accordance, the results showed a dependence on the PrCA risk linked to this clustering, and the multilevel model improved the precision of the individual effect estimations. This approach confers a statistical and interpretative advantage as it proposes a theoretical construct for addressing diet–cancer relationship that takes into account an important way in which humans organize their work and residential environments [60].

The main strength of this study is that it is the first investigation regarding diet quality in relation to inflammatory potential and PrCA in Argentina, which has a high incidence of PrCA among men and unusual dietary patterns. These unique characteristics enabled us to explore the association between the inflammatory properties of diet, using the DII, in relation to incident PrCA. It is one of the few studies that has looked at the association between diet as a whole and PrCA. Even with a relatively small sample size we have observed significant results, which indicate the importance of consuming anti-inflammatory diet in protecting against PrCA.

As with any case–control study of diet and health outcomes, this investigation shares weaknesses of such designs. Most notably these include information bias related to knowledge of disease state and selection bias. To avoid potentially important bias due to confounders, similar distribution of age and place of residence in cases and controls was sought, and both groups were interviewed in the same period. Controls came from the same geographical "catchment area" as the cases and mostly the same interview setting (the home) were used for both groups, and there was almost complete participation (about 90% participation rate). All of these procedures contribute to minimizing both selection and information biases. Community controls obviate some of the problems of selection bias that may occur among hospital controls. With reference to information bias, a classification bias caused by "rumination" in cases regarding the possible causes of their disease must be considered.

The kind of biases that exist in this Argentinian population may be quite different that what would be expected in other studies of the DII in relation to PrCA conducted in Europe and North America. Indeed, we anticipate that the bias in recalling food intake by cases should be small, given the limited knowledge and attention paid in this population at the time of the study to the possible relationship between diet and cancer (including PrCA). Indeed, the sensitivity analysis performed previously [37] based on the possibility of the systematic errors mentioned, showed no major evidence of influence of bias. A further limitation of our study is that we could derive DII score from only 22 of the potential 45 food and nutrient items that theoretically can be used to compute this index. The DII components that were missing from this study were saffron, turmeric, thyme/oregano, ginger, rosemary, eugenol, beta-carotene, pepper, tea, anthocyanidins, flavan3ol, flavones, flavonols, flavonones, isoflavones, magnesium, vitamin-D, B1, B3, B12, folic acid, and trans-fat. However, other published studies also

derive DII scores from a sub-optimal number of items, and the ability to still detect significant associations suggests that this has only led to a potential underestimate of the association between DII and PrCA risk. Additionally, some of the missing DII components such as saffron, ginger and turmeric are consumed infrequently in this population; so, non-availability of these parameters may not have exerted a major impact. However, availability of information on DII components like vitamin B1, B3, B12 and magnesium, which are present in several of the food items consumed in Argentina, might have modified the results. Further to this issue of calculating DII from fewer DII components, we have previously demonstrated in the SEASONS study that DII scores calculated from 44 DII components using the 24-h recalls and DII scores calculated from 27 DII components using 7-day dietary recall resulted in virtually identical ORs, where CRP (> 3 mg/l) was the study outcome [22]. The components present in this study to calculate the DII are all nutrients, which are present in almost all of our previous DII and PrCA publications. The values of these components are what determines the DII score and this is what makes the DII different for different populations. For example, the mean DII among controls for a study in Canada [43], where DII was derived from almost similar number of DII components, was + 0.023 whereas the mean DII among controls in the current Argentinian study was − 0.35. So, from this we can discern that the diet in the Canadian study is slightly more pro-inflammatory than the diet among controls in the current Argentinian study. It must be emphasized that the list of 45 DII components was not specified *a priori*. A search was first done to see which dietary components have been studied the most in relation to the six inflammatory biomarkers. Thus, the list is the result of this search. This search was first done in 2007 and later in 2011 (reflecting the literature through 2010). An updated search may be done in the future to see if any other component can be added to this list or if the estimates of effect might have changed.

The influence of diet on cancer is difficult to measure precisely, and challenges in dietary exposure assessment are greatest in case–control studies. Although we used a validated FFQ for assessing dietary intake, measurement errors that might distort associations were inevitable. Even though diet in mid-life may be more important than the diet later in life, the long time that had passed from the patients' mid-life restricts our ability to evaluate that time-period. No data were available on inflammatory markers in this study; hence, the DII could not be validated with inflammation in this case–control study. Because most of the parameters used to calculate the DII are nutrients, non-availability of supplement data can be considered a limitation. In previous studies that have examined DII with and without supplements [61–63], improved prediction was seen based on the DII including supplements. This may be because dietary supplements tend to be anti-inflammatory; so, the overall mean DII tends to be lower, and the range of the DII scores is larger compared to DII with diet only [61–63]. So, because of the increased contrast, the most pro-inflammatory category of the DII with supplements appears to have a larger effect on health outcomes; the logical corollary is that the most anti-inflammatory category of the DII with supplements had a more protective association with health outcomes [61–63].

Small sample size is another limitation that might result in unstable risk estimates with wide confidence intervals. Epidemiological and statistical literature provide asymptotic formulas for the computation of case–control sample sizes required for odds ratios, whether unadjusted or adjusted for a confounder [64]. However, all these recommendations take into account only fixed effects of covariates, including the intercept. The limited number of parameters fit in the model and the constraint on the sources of variability (a variance component to quantify the intraclass correlation), constitute a reasonable effort to compensate for the small size of our study. It also should be kept in mind that the average DII score in this population was generally higher (more pro-inflammatory) than in other populations (i.e., around 1.5 vs. 0) and the range was narrower than we normally see (around 5 vs. 9). Finally, in this study there are no data on lycopene intake and PrCA screening, two variables that have been shown to be associated with PrCA. In Argentina, there are no population-level data on lycopene intake. Also, the prevalence of prostate cancer screening in Argentina is unknown. According to the results from the few available studies on the subject, the mean intake of lycopene ranges from 3,000 to 5,000 µg per day in adult men [65, 66]. Prostate cancer screening is not widely available in Argentina; there are no official screening programs for PrCA.

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 [13, 67, 68]. They also are consistent with prior investigations of the DII in relation to PrCA.

The logical next step would be to use DII scores to predict incidence of other cancers and serum levels of inflammatory markers in Argentina and other Latin American countries in prospective studies and examine other outcomes which are related to diet and inflammation like cardiovascular diseases.

In conclusion, this study implicates diet-associated inflammation in the etiology of PrCA, a finding that it consistent with both biological mechanisms of action and prior results implicating DII scores in PrCA. Findings are most pronounced in obese men. There is a need for other studies to be conducted in different populations and with prospective cohorts to more firmly establish cause and effect.

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Compliance with ethical standards

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 DII® from the University of South Carolina in order to develop computer and smart phone applications for patient counseling and dietary intervention in clinical settings. Dr Nitin Shivappa is an employee of CHI.

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