

Diet patterns and the risk of renal cell carcinoma

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Abstract

Objectives: Our objective was to identify food intake patterns that might be associated with the risk of renal cell carcinoma.

Design: A total of 461 cases (210 females, 251 males) were age frequency matched to population controls. Diet factors were created using factor analysis of 69 food items from a food-frequency questionnaire. These factors were modelled using logistic regression to identify those associated with renal cell carcinoma.

Setting: We investigated the role of diet in the aetiology of renal cell carcinoma using a population-based case-control study conducted in Ontario between 1995 and 1996.

Subjects: Cases were Ontario residents 20 to 74 years of age identified through review of pathology reports in the Ontario Cancer Registry.

Results: A 'dessert' diet factor was positively associated with disease for both sexes (odds ratio estimate (OR) for males = 3.7, 95% confidence interval (CI) 2.0–6.9; OR for females = 1.4, 95% CI 0.8–2.2, for the highest vs. lowest quartile). In males, a 'beef' diet factor was identified and was associated with an increased risk of renal cell carcinoma. Furthermore, a 'juices' diet factor also showed an association with increased risk in males (OR = 1.8, 95% CI 1.0–3.1). For females, a positive association was observed between renal cell carcinoma and an 'unhealthy' diet factor (OR = 1.4, 95% CI 0.8–2.4).

Conclusions: Our findings confirmed that high-fat and high-protein diets might be risk factors for renal cell carcinoma. The data also suggest an increased risk associated with juice intake, a finding not previously reported.

Keywords
Renal cell carcinoma
Factor analysis
Case-control study
Diet patterns
Canada
Risk factors
Male
Female

The role of diet in the aetiology of renal cell carcinoma is unresolved. The most consistent results show increased risks associated with the consumption of foods such as meat, eggs and dairy products and reduced risk for fruits and vegetables^{1–7}. Studies have shown a higher risk associated with protein and fat^{8,9} and potential decreased risks associated with vitamin C and vitamin E^{8–13}. These studies have assessed diet as specific foods or nutrients. Yet individuals consume combinations of nutrients or foods at a time, and therefore are likely to experience effects that are a consequence of the interactions among these dietary items. Disease associations with diet may not be captured entirely by examining individual constituents.

The objective of this study was to identify dietary patterns associated with renal cell carcinoma, by reducing a large number of food items to a more parsimonious number of factors based on the correlations among foods.

Subjects and methods

Data came from the Ontario portion of the Enhanced Cancer Surveillance (ECS) of the Laboratory Centre for

Disease Control at Health Canada, a population-based multi-cancer case-control study¹⁴. Analyses were restricted to Ontario data because the dietary component of the questionnaire was different from that in other provinces. Cases were Ontario residents 20 to 74 years of age, with a histologically confirmed cancer diagnosed between January 1995 and December 1996, identified through review of pathology reports in the Ontario Cancer Registry¹⁵.

Controls were 20 to 74 years of age, Ontario residents, identified through the population-based assessment rolls of the Ontario Ministry of Finance, and selected to yield a control sample with a sex and 5-year age distribution similar to all cases combined.

Physicians were asked for consent to contact cases, to confirm the cancer diagnosis and to provide the patient's vital status, address, telephone number and next-of-kin (if necessary). Cases and controls were mailed an explanatory letter and a self-administered questionnaire. Follow-up to non-respondents included postcard, letter and telephone. Questionnaires were reviewed for comprehensibility and completeness, and subjects were telephoned to supply missing information or clarification.

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In total, 842 kidney cancer cases were identified (462 males and 380 females). Of those, 652 were mailed questionnaires while the remainder were not eligible by age ($n = 51$), deceased ($n = 31$), physician refusal/late consent ($n = 60$) or unknown physician/subject address ($n = 48$). Five hundred and thirty-five kidney cancer cases returned questionnaires (82.1%). Of these, nine did not meet diagnostic criteria and 16 were proxy respondents, and were excluded.

Questionnaires were sent to 2941 potential controls (1406 males, 1535 females); 1929 (901 males, 1028 females) were returned (64.1% response males, 67.0% females). Of these, 1588 were within the overall age range for renal cell carcinoma cases (787 males, 801 females). From these, male and female controls were frequency matched to the cases based on 5-year age groups, 1:1 for males and 2:1 for females.

Frequency of intake of 69 food items was entered as one of nine categories ranging from 'never or less than 1 per month' to '6 or more times per day' in the diet questionnaire using Canadian nutrient data¹⁶. The median frequencies of food use reported on a monthly or daily basis were converted to a period of weekly food use. For many items, where more than daily intake was rare, the data were collapsed to a maximum of ≥ 7 times per week.

To avoid extensive omission of cases and controls due to missing values, responses were imputed for subjects missing $\leq 5\%$ of food frequency variables. Imputed values were randomly selected from the intake frequencies provided by the subset of cases and controls with complete diet information within each case/control, sex, age and smoking stratum. Approximately 90% of cases and controls had $\leq 5\%$ of diet variables missing. The percentage of cases and controls with imputed diet variables did not differ. Analyses were based on 461 cases (210 females, 251 males) and 672 controls (422 females, 250 males).

Factor analysis¹⁷ was used to identify dietary factors, or combinations of foods, consumed in the study sample. Male and female controls were used to generate two separate sets of diet patterns. A correlation matrix of the original 69 dietary variables was created, and maximum likelihood factor analysis followed by varimax rotation was used to create a parsimonious set of factors. Only factors with eigenvalues ≥ 1.5 were selected. Dietary factors consisted of those items in each factor that correlated with the factor with factor loading of ≤ -0.20 or ≥ 0.30 . Factor scores for each dietary factor were calculated for cases and controls by multiplying the standardised scoring coefficient of each food included in the factor by the value reported in the food-frequency questionnaire, and summing across all foods in the factor. Subjects were assigned scores to indicate the degree to which their diet adhered to each of the factors. Labels were assigned to each factor based on an approximate description of the food items that were most highly represented.

Sex-specific unconditional logistic regression methods were used¹⁸. Odds ratios (ORs) and 95% confidence intervals (CIs) were estimated using EGRET¹⁹. Stepwise regression analysis was used to determine which diet patterns would be included in the final logistic regression model. Factors with a P -value ≤ 0.20 were included in the stepwise model. Evaluation of the effects of the dietary patterns was carried out while fitting the final logistic regression model. Factors that were significantly associated with an increased risk of renal cell carcinoma were included (P -value ≤ 0.05). Likelihood ratio statistics were used to assess the contribution of the individual variables in a model and were calculated based on the reduction in residual deviance after the addition of the variable to a prior fitted model. Potential confounding variables were age, smoking status (never/former/current) and body mass index (BMI), and were included in the modelling.

Results

Tables 1 and 2 present dietary factors from the factor analysis of the female and male controls, respectively. For females, diet variables converged into eight factors that explained 15.5% of the total variance. Thirteen dietary factors explaining 24% of the variance were generated for males. Of these, the first eight explained most of this (17.5%).

Although the numerical values of the factor loading were different for males and females, Factor I for both was highly correlated with many fruits and vegetables. Factor II consisted of 'dessert' items. Similarly, a 'meat' diet factor was identified for both sexes (Factor IV in females, Factor VIII in males). Another dietary factor consisting of meat was identified in males in Factor VII, in which beef was the only food variable represented.

In males, a 'fruit juice' dietary factor was represented in Factor V: frozen, powdered and fresh juices. In females, Factor VII included fruit juices that were consumed often, while the intake of liquor and wine was infrequent. Factor VIII in females was positively associated with white bread but inversely associated with broccoli, skimmed milk, 1% milk and dark bread, which might be termed an 'unhealthy diet'.

Tables 3 and 4 present the distribution of age, previously identified risk factors and the diet patterns for females and males, respectively. Variables that were associated with increased risk in females included BMI and smoking status. The highest quartile for BMI was associated with a 2.4-fold increased risk (95% CI 1.5–3.8). Current and former smokers were at somewhat increased risk, although this relationship was significant only among former smokers (OR = 1.7, 95% CI 1.1–2.5). While increasing pack-years were associated with increased risk, all confidence intervals included unity.

For females, Factor I ('fruits and vegetables') was associated with reduced renal cell carcinoma risk, with the

Table 1 Dietary food patterns identified for female controls (*n* = 422)

Food/beverage	Factor loading							
	Factor I Fruits/vegetables	Factor II Dessert	Factor III Miscellaneous +	Factor IV Meat	Factor V Miscellaneous –	Factor VI High protein	Factor VII Beverage	Factor VIII Unhealthy
Coffee					0.32			
Powdered drinks								
Bottled water			0.43					
1% milk								–0.24
Skimmed milk								–0.23
Fresh citrus juice							0.36	
Other fresh juice								
Tomato/vegetable juice					0.41			
Frozen juice/drink							0.46	
Beer								
Wine							–0.20	–0.20
Liquor							–0.20	
Apples/pears	0.39				–0.23			
Oranges	0.37							
Bananas	0.32						0.45	
Cantaloupe			0.42					
Other fruit – fresh, canned	0.44							
Tomatoes	0.37		0.34					
Carrots	0.54							
Broccoli	0.39							–0.21
Cabbage*	0.41							
Spinach or other greens	0.49							
Yellow squash			0.37					
Other vegetables	0.59							
Soups with vegetables								
Potatoes	0.39		–0.20		0.32			
French fries		0.37						
Tofu or soybean						0.56		
Baked beans or lentils								
Bran or granola cereals	0.35							
Cooked cereals								
White bread								0.37
Dark bread								–0.28
Rice					–0.40	0.60		
Macaroni, spaghetti			0.33					
Chicken or turkey						0.32		
Beef/pork/lamb – main dish								
Beef/pork/lamb – mixed dish								
Hamburger				0.48				
Hot dogs				0.64				
Luncheon meats				0.46				
Smoked meat, corned beef								
Bacon								
Sausage								
Liver		0.47						
Fish – fresh, frozen, canned	0.30							
Fish – smoked, salted, dried			0.43					
Eggs						0.36		
Cheese other than cottage								
Cake		0.51						
Cookies		0.44						
Doughnuts		0.61						
Pies		0.37						
Ice cream		0.46						
Chocolate		0.55						
Potato chips		0.30		0.35				
Peanut butter								–0.27
Nuts								
Butter on bread					0.32			
Margarine on bread			–0.20					
Mayonnaise/salad dressing					0.40			
Percentage of variance	3.13	2.45	1.85	1.78	1.70	1.67	1.64	1.28

Food items with factor loadings ≤ -0.20 and ≥ 0.30 were included.

*Cabbage includes cauliflower and Brussels sprouts.

Table 3 Age-adjusted ORs and 95% CIs for renal cell carcinoma and risk factors for age-matched females

	Cases, <i>n</i> (%)	Controls, <i>n</i> (%)	OR (95% CI)
Age group (years)			
35–39	10 (4.8)	19 (4.5)	
40–44	17 (8.1)	34 (8.1)	
45–49	25 (11.9)	48 (11.4)	
50–54	31 (14.8)	63 (14.9)	
55–59	25 (11.9)	51 (12.1)	
60–64	36 (17.1)	79 (18.7)	
65–69	32 (15.2)	62 (14.7)	
70–74	34 (16.2)	66 (15.6)	
BMI* (kg m⁻²)			
≤ 22.10	36 (17.1)	108 (25.6)	1.0
22.11–24.61	39 (18.6)	107 (25.4)	1.1 (0.7–1.9)
24.62–27.93	53 (25.2)	100 (23.7)	1.6 (1.0–2.7)
> 27.93	81 (38.6)	104 (24.6)	2.4 (1.5–3.8)
Education			
< 11 years post secondary	92 (43.8)	151 (35.8)	1.0
≥ 12 years post secondary	39 (18.6)	86 (20.4)	0.7 (0.5–1.2)
< 4 years college	61 (29.0)	137 (32.5)	0.7 (0.5–1.1)
≥ 4 years college	18 (8.6)	48 (11.4)	0.6 (0.3–1.1)
Smoking status			
Never	99 (47.1)	239 (56.6)	1.0
Former	61 (29.0)	88 (20.9)	1.7 (1.1–2.5)
Current	50 (23.8)	94 (22.3)	1.3 (0.8–1.9)
Pack-years†			
Never smokers	99 (47.1)	239 (56.6)	1.0
Former QI	26 (12.4)	40 (9.5)	1.6 (0.9–2.7)
Former QII	16 (7.6)	16 (3.8)	2.4 (1.2–5.0)
Former QIII	11 (5.2)	20 (4.7)	1.3 (0.6–2.9)
Former QIV	8 (3.8)	12 (2.8)	1.6 (0.6–4.1)
Never smokers	99 (47.1)	239 (56.6)	1.0
Current QI	2 (1.0)	5 (1.2)	1.0 (0.2–5.0)
Current QII	10 (4.8)	30 (7.1)	0.8 (0.4–1.7)
Current QIII	18 (8.6)	25 (5.9)	1.7 (0.9–3.3)
Current QIV	20 (9.5)	34 (8.1)	1.4 (0.8–2.6)
Smoking cessation (former smokers only)			
≥ 10 years	40 (65.6)	60 (68.2)	1.0
< 10 years	21 (34.4)	28 (31.8)	1.1 (0.6–2.3)
Smoke age (former and current smokers)			
< 20 years of age	31 (27.9)	65 (35.7)	1.0
≥ 20 years of age	79 (71.2)	118 (64.8)	1.4 (0.8–2.3)
Diet factor			
Factor I – Fruits and vegetables			
QI	62 (29.5)	105 (24.9)	1.0
QII	54 (25.7)	106 (25.1)	0.9 (0.5–1.3)
QIII	62 (29.5)	106 (25.1)	1.0 (0.6–1.5)
QIV	32 (15.2)	105 (24.9)	0.5 (0.3–0.8)
Factor II – Desserts			
QI	47 (22.4)	105 (24.9)	1.0
QII	33 (15.7)	106 (25.1)	0.7 (0.4–1.2)
QIII	62 (29.5)	105 (24.9)	1.3 (0.8–2.1)
QIV	68 (32.4)	106 (25.1)	1.4 (0.9–2.3)
Factor III – Miscellaneous+			
QI	66 (31.4)	105 (24.9)	1.0
QII	58 (27.6)	106 (25.1)	0.9 (0.5–1.4)
QIII	41 (19.5)	105 (24.9)	0.6 (0.4–1.0)
QIV	45 (21.4)	106 (25.1)	0.7 (0.4–1.1)
Factor IV – Meat			
QI	40 (19.0)	105 (24.9)	1.0
QII	53 (25.2)	106 (25.1)	1.3 (0.8–2.2)
QIII	49 (23.3)	106 (25.1)	1.2 (0.7–2.0)
QIV	68 (32.4)	105 (24.9)	1.7 (1.1–2.8)
Factor V – Miscellaneous –			
QI	33 (15.7)	106 (25.1)	1.0
QII	51 (24.3)	105 (24.9)	1.6 (0.9–2.6)
QIII	67 (31.9)	105 (24.9)	2.1 (1.2–3.4)
QIV	59 (28.1)	106 (25.1)	1.8 (1.1–3.0)
Factor VI – High protein			
QI	39 (18.6)	106 (25.1)	1.0
QII	63 (30.0)	105 (24.9)	1.6 (1.0–2.7)
QIII	55 (26.2)	106 (25.1)	1.4 (0.9–2.3)
QIV	53 (25.2)	105 (24.9)	1.4 (0.8–2.3)

Table 3. *Continued*

	Cases, n (%)	Controls, n (%)	OR (95% CI)
Factor VII – Beverages			
QI	48 (22.9)	106 (25.1)	1.0
QII	55 (26.2)	105 (24.9)	1.2 (0.7–1.9)
QIII	61 (29.0)	106 (25.1)	1.3 (0.8–2.0)
QIV	46 (21.9)	105 (24.9)	1.0 (0.6–1.6)
Factor VIII – Unhealthy			
QI	40 (19.0)	106 (25.1)	1.0
QII	46 (21.9)	105 (24.9)	1.2 (0.7–1.9)
QIII	68 (32.4)	106 (25.1)	1.7 (1.1–2.7)
QIV	56 (26.7)	105 (24.9)	1.4 (0.9–2.3)

* BMI – body mass index.

† Quartiles of pack-years determined using former and current smokers combined: QI < 5.1; 5.1 ≤ QII < 17.3; 17.3 ≤ QIII < 31.1; QIV ≥ 31.1.

highest consumption category associated with an OR of 0.5 (95% CI 0.3–0.8). A reduced risk was also associated with the highest intake levels of Factor III, the ‘miscellaneous+’ factor (OR = 0.7, 95% CI 0.4–1.1). Elevated risks were observed for diet factor IV (‘meat’) and diet factor V (‘miscellaneous –’).

For males (Table 4), high BMI was associated with a significant increase in risk (OR = 3.5, 95% CI 2.1–6.0). Reduced odds ratio estimates were observed for the most educated (OR = 0.7, 95% CI 0.4–1.1). Former and current smokers in the highest quartiles had a non-significant increased risk relative to non-smokers (current smokers OR = 1.3, 95% CI 0.8–2.1; former smokers OR = 1.6, 95% CI 1.0–2.5). The time since smoking cessation for former smokers did not appear to affect the risk of renal cell carcinoma. Smokers starting to smoke after the age of 20 were at an elevated risk relative to smokers who started at an early age; this risk estimate, however, was not significant (OR = 1.3, 95% CI 0.8–2.2).

A reduction in risk was observed among the highest quartile of Factor I (‘fruits and vegetables’) relative to the lowest (OR = 0.6, 95% CI 0.4–1.0). Most notable was the significant increased risk associated with the highest intake level of Factor II (‘desserts’) (OR = 3.9, 95% CI 2.2–6.9). Increased odds ratio estimates were also observed for Factors III (‘meats’) and VII (‘beef’).

Table 5 displays the final logistic regression models. For both males and females, a ‘desserts’ diet pattern was associated with an increased risk of renal cell carcinoma. Further, among males, an increased risk associated with the consumption of a ‘beef’ diet (Factor VII) was observed. The ‘juices’ pattern (Factor V) was associated with an increased risk of renal cell carcinoma for males (OR = 1.7, 95% CI 1.0–3.1). For females, a positive association between renal cell carcinoma and the ‘unhealthy’ factor (Factor VIII) was observed (OR = 1.4, 95% CI 0.8–2.4).

Discussion

Disease risk cannot be determined by the presence or absence of any single food or nutrient. Rather it is the

selection of foods in certain amounts and combinations that is more likely to play a role in carcinogenesis. The majority of epidemiological studies proposing diet as a risk factor for renal cell carcinoma have examined the association of cancer risk with the intake of single nutrients, foods or food groups. Failing to control for the correlations between foods, these studies may not have adequately considered the metabolic consequences of food items consumed concurrently or the inverse relationships of food intake.

In an attempt to control for the correlations among foods, factor analysis was used to derive dietary patterns as risk factors. The results of these analyses confirm those of previous studies in which beef, high-fat and high-protein diets are associated with renal cell carcinoma. Most of the evidence linking diet with renal cell carcinoma suggests that high protein consumption, particularly from meat, eggs or milk, is positively associated with the disease^{1,2,4,6,7,12,13,20,21}. Recently, a study ascertained that the population-attributable risk associated with protein intake was 19% for any intake above the lowest quartile²². Our results add evidence that this type of diet may be considered an important risk factor for renal cell carcinoma.

Despite the potential association with fat, few studies have examined the risk of renal cell carcinoma associated with dessert items. Maclure and Willett¹ found elevated but non-significant risks associated with moderate consumption of ice cream, pie, cake, doughnuts and cookies. Another study reported no association between a dessert food group and renal cell carcinoma¹³. Since fats have been associated with renal cell carcinoma^{6,8,9,12,23}, a diet rich in desserts may be associated with risk.

Our study did not consider energy adjustment of food intake. As obesity is an established risk factor of renal cell carcinoma, it is possible that overeating is a primary cause of the disease. In this situation, nutrients contributing to calories (proteins, fats, carbohydrates and alcohol) might be considered as the primary exposures that lead to increased calorie intake, which in turn cause disease²⁴. Therefore, adjustment for caloric intake would likely result

Table 4 Age-adjusted ORs and 95% CIs for renal cell carcinoma and previously identified risk factors for age-matched males

	Cases, <i>n</i> (%)	Controls, <i>n</i> (%)	OR (95% CI)
Age group (years)			
25–29	1 (0.4)	1 (0.4)	
30–34	2 (0.8)	2 (0.8)	
35–39	4 (1.6)	4 (1.6)	
40–44	18 (7.2)	18 (7.2)	
45–49	23 (9.2)	24 (9.6)	
50–54	31 (12.4)	30 (12.0)	
55–59	43 (17.1)	44 (17.6)	
60–64	41 (16.3)	41 (16.4)	
65–69	47 (18.7)	44 (17.6)	
70–74	41 (16.3)	42 (16.8)	
BMI* (kg m⁻²)			
≤ 23.77	32 (12.7)	62 (24.8)	1.0
23.78–25.71	35 (13.9)	63 (25.2)	1.1 (0.6–1.9)
25.72–28.08	68 (27.1)	62 (24.8)	2.1 (1.2–3.7)
> 28.08	115 (45.8)	63 (25.2)	3.5 (2.1–6.0)
Education			
< 11 years post secondary	92 (36.7)	83 (33.2)	1.0
≥ 12 years post secondary	51 (20.3)	44 (17.6)	1.0 (0.6–1.7)
< 4 years college	76 (30.3)	79 (31.6)	0.9 (0.6–1.3)
≥ 4 years college	32 (12.7)	44 (17.6)	0.7 (0.4–1.1)
Smoking status			
Never	56 (22.3)	74 (29.6)	1.0
Former	129 (51.4)	108 (43.2)	1.6 (1.0–2.5)
Current	66 (26.3)	68 (27.2)	1.3 (0.8–2.1)
Pack-years†			
Never smokers	56 (22.3)	74 (29.6)	1.0
Former QI	43 (17.1)	33 (13.2)	1.7 (1.0–3.1)
Former QII	32 (12.7)	30 (12.0)	1.4 (0.8–2.6)
Former QIII	24 (9.6)	25 (10.0)	1.3 (0.7–2.5)
Former QIV	27 (10.8)	18 (7.2)	2.1 (1.0–4.1)
Never smokers	56 (22.3)	74 (29.6)	1.0
Current QI	7 (2.8)	9 (3.6)	1.0 (0.4–2.9)
Current QII	8 (3.2)	14 (5.6)	0.7 (0.3–1.9)
Current QIII	24 (9.6)	21 (8.4)	1.5 (0.8–3.0)
Current QIV	26 (10.4)	24 (9.6)	1.5 (0.8–2.8)
Smoking cessation (former smokers only)			
≥ 10 years	96 (74.4)	78 (72.2)	1.0
< 10 years	33 (25.6)	30 (27.8)	0.9 (0.5–1.5)
Smoke age (former and current smokers)			
< 20 years of age	34 (17.4)	38 (21.6)	1.0
≥ 20 years of age	161 (82.6)	136 (77.3)	1.3 (0.8–2.2)
Diet factor			
Factor I – Fruits and vegetables			
QI	77 (30.6)	63 (25.2)	1.0
QII	58 (23.1)	62 (24.8)	0.8 (0.5–1.2)
QIII	69 (27.5)	62 (24.8)	0.9 (0.6–1.5)
QIV	47 (18.7)	63 (25.2)	0.6 (0.4–1.0)
Factor II – Desserts			
QI	24 (9.6)	63 (25.2)	1.0
QII	57 (22.7)	62 (24.8)	2.4 (1.3–4.4)
QIII	79 (31.5)	63 (25.2)	3.3 (1.9–5.9)
QIV	91 (36.2)	62 (24.8)	3.9 (2.2–6.9)
Factor III – Meat			
QI	39 (15.5)	62 (24.8)	1.0
QII	67 (26.7)	63 (25.2)	1.7 (1.0–2.9)
QIII	77 (30.7)	62 (24.8)	2.0 (1.2–3.3)
QIV	68 (27.1)	63 (25.2)	1.7 (1.0–2.9)
Factor IV – Rice, tofu			
QI	63 (25.1)	62 (24.8)	1.0
QII	62 (24.7)	63 (25.2)	1.0 (0.6–1.6)
QIII	79 (31.5)	63 (25.2)	1.2 (0.8–2.0)
QIV	47 (18.7)	62 (24.8)	0.7 (0.4–1.3)
Factor V – Juices			
QI	43 (17.1)	62 (24.8)	1.0
QII	40 (15.9)	63 (25.2)	0.9 (0.5–1.6)
QIII	95 (37.8)	62 (24.8)	2.2 (1.3–3.7)
QIV	73 (29.1)	63 (25.2)	1.7 (1.0–2.9)

Table 4. Continued

	Cases, n (%)	Controls, n (%)	OR (95% CI)
Factor VI – Fruit, dark bread			
QI	51 (20.3)	62 (24.8)	1.0
QII	80 (31.9)	63 (25.2)	1.5 (0.9–2.5)
QIII	71 (28.3)	63 (25.2)	1.4 (0.8–2.3)
QIV	49 (19.5)	62 (24.8)	1.0 (0.6–1.6)
Factor VII – Beef			
QI	49 (19.5)	62 (24.8)	1.0
QII	51 (20.3)	63 (25.2)	1.0 (0.6–1.7)
QIII	91 (36.3)	62 (24.8)	1.9 (1.1–3.0)
QIV	60 (23.9)	63 (25.2)	1.2 (0.7–2.0)
Factor VIII – Pies, cake‡			
QI	47 (18.7)	62 (24.8)	1.0
QII	64 (24.3)	63 (25.2)	1.3 (0.8–2.2)
QIII	66 (26.3)	63 (25.2)	1.4 (0.8–2.3)
QIV	74 (29.5)	62 (24.8)	1.6 (0.9–2.6)
Factor IX – No fast food			
QI	47 (18.7)	62 (24.8)	1.0
QII	68 (27.1)	63 (25.2)	1.4 (0.9–2.3)
QIII	68 (27.1)	63 (25.2)	1.4 (0.9–2.4)
QIV	68 (27.1)	62 (24.8)	1.5 (0.9–2.5)
Factor X – Miscellaneous			
QI	51 (20.3)	62 (24.8)	1.0
QII	70 (27.9)	63 (25.2)	1.4 (0.8–2.2)
QIII	74 (29.5)	62 (24.8)	1.5 (0.9–2.4)
QIV	56 (22.3)	63 (25.2)	1.1 (0.6–1.8)
Factor XI – Fruit			
QI	51 (20.3)	63 (25.2)	1.0
QII	68 (27.1)	62 (24.8)	1.4 (0.8–2.2)
QIII	69 (27.5)	63 (25.2)	1.4 (0.8–2.2)
QIV	63 (25.1)	62 (24.8)	1.3 (0.8–2.1)
Factor XII – Butter			
QI	73 (29.1)	63 (25.2)	1.0
QII	57 (22.7)	62 (24.8)	0.8 (0.5–1.3)
QIII	35 (13.9)	62 (24.8)	0.5 (0.3–0.8)
QIV	86 (34.3)	63 (25.2)	1.2 (0.7–1.9)
Factor XIII – Bottled water			
QI	42 (16.7)	63 (25.2)	1.0
QII	66 (26.3)	62 (24.8)	1.6 (1.0–2.7)
QIII	67 (26.7)	63 (25.2)	1.6 (1.0–2.7)
QIV	76 (30.3)	62 (24.8)	1.9 (1.1–3.1)

* BMI – body mass index.

† Quartiles of pack-years determined using former and current smokers combined: QI < 9.3; 9.3 ≤ QII < 22.2; 22.2 ≤ QIII < 34.7; QIV ≥ 34.7.

‡ Significant test for trend ($P \leq 0.05$).

in the over-adjustment of a variable in the causal pathway²⁴. Similarly adjusting for BMI may have also resulted in an over-adjustment in the model. However, the estimates in the final logistic regression models did not change when BMI was not included in the model (data not shown). As BMI is a known risk factor of renal cell carcinoma, we chose to include it in our final models.

Although fruits in general tend to be associated with decreased risk, the vegetables also found to have this effect include Cruciferous and orange/green vegetables^{3,5–7,10–12,23}. A dietary pattern consisting mostly of fruits and vegetables was identified in both males and females in our data, and was inversely associated with disease, although not in the adjusted analysis. Furthermore, an increase in risk was associated with an ‘unhealthy’ diet in females, in which items such as broccoli and low-fat milk were infrequently consumed. In this factor, it was the absence of healthy foods that was associated with increased risk.

Despite reduced risk associated with ‘fruits and vegetables’ intake, we found an increased risk associated with a high intake of fruit juices in males. Maclure and Willett examined fruit juices as a risk factor but observed a non-significant decreased risk¹. Wolk *et al.*¹⁰ reported a decrease in risk with increasing intake levels of citrus fruit, while two studies did not observe an association between citrus fruits and renal cell carcinoma, except in non-smokers^{12,13}.

Our data are consistent with an increased risk of fluid intake, *per se*. While Kreiger *et al.*⁴ did not report significantly increased risk associated with beverage intake, fluid intake has been shown to be positively associated with the risk of bladder cancer in some^{25–27} but not all^{28–30} studies.

One limitation of factor analysis is its subjectivity³¹. There are no accepted criteria for (1) the number of variables, (2) the number of factors, (3) the values of factor loadings to determine variables that contribute most to a

Table 5 Adjusted* ORs and 95% CIs for renal cell carcinoma risk and dietary patterns in final logistic regression model by sex

Dietary factor	OR (95% CI)
<i>Females</i>	
Factor II – Desserts	
QI	1.0
QII	0.6 (0.3–1.0)
QIII	1.2 (0.7–2.0)
QIV	1.4 (0.8–2.2)
Factor VIII – Unhealthy	
QI	1.0
QII	1.1 (0.6–1.8)
QIII	2.1 (1.3–3.4)
QIV	1.4 (0.8–2.4)
<i>Males</i>	
Factor II – Desserts	
QI	1.0
QII	2.1 (1.1–4.0)
QIII	3.3 (1.8–6.1)
QIV	3.7 (2.0–6.8)
Factor V – Juices	
QI	1.0
QII	0.8 (0.5–1.5)
QIII	2.2 (1.3–3.8)
QIV	1.7 (1.0–3.1)
Factor VII – Beef	
QI	1.0
QII	0.9 (0.5–1.6)
QIII	1.8 (1.0–3.1)
QIV	1.0 (0.6–1.7)

* Adjusted for age, smoking status and body mass index.

factor, (4) the method of rotation and (5) the naming of factors³¹. Furthermore, the generalisability of the results has not been fully explored. Dietary factors are driven by the study instrument used to measure diet intake and the sample from which they are derived. Our dietary factors were based on 69 food items that were presented in the food-frequency questionnaire; we are limited in generalising from these factors to the entire dietary intakes of our subjects. It is possible that the dietary factors explaining the most variation in dietary choices could be replicated in other populations, whereas factors accounting for less of the variability may not contribute to the dietary analysis of other populations³⁰.

Our dietary factors accounted only for a maximum of 15 to 20% of the variance, similar to that observed in previous studies^{30,32}. To explain greater variance, additional dietary factors would be needed although these added factors might be more difficult to interpret, representing smaller amounts of variance and the increasingly heterogeneous 'patterns'.

Although the recall of diet is greatly influenced by current diet, studies have indicated that a more reliable estimate of the past diet is attained if subjects are questioned directly about their past diet rather than their current^{24,33}. The questionnaire asked directly about dietary intake two years prior to the date of interview. Although diet tends to have some consistency over time, our diet information may not reflect subjects' past

diet allowing for appropriate exposure and latency periods²⁴. Similarly, our study may be subject to recall bias as cases and controls may have differed in their recall of past diet, with the possibility that cases or controls consistently under- or overreported their dietary intakes.

Factor analysis used for dietary assessment in nutritional epidemiology is advantageous because it identifies diet factors as they exist in a population. When consumed concurrently, correlated dietary factors may have interactive effects that could have positive or negative effects on health. Therefore, by representing diets as food factors, the multidimensional aspect of diet may be captured. Furthermore, patterns also consider the non-use of foods, as negatively correlated food items will appear within diet patterns.

Results from this study support an association between a diet characterised by high fat and protein foods and increased risk of renal cell carcinoma. An unexpected association between a fruit juice pattern and the risk of renal cell carcinoma was also observed among males, despite a reduced risk associated with a 'fruits and vegetables' dietary pattern. Further investigation is warranted to examine if fluid intake rather than intake of the specific constituents of fruit juices may be responsible for this finding.

Factor analysis has been used to generate diet factors in hope of understanding the aetiology of renal cell carcinoma in greater depth. By investigating dietary patterns and their associations with cancer risk, the biological mechanism between diet and cancer remains less clear. However, diet patterns identified for disease prevention may be important in making dietary recommendations for disease prevention.

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