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Consumption of deep-fried foods and risk of prostate cancer^{a,b}

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Abstract

Background—Evidence suggests that high-heat cooking methods may increase the risk of prostate cancer (PCa). The addition of oil/fat, as in deep-frying, may be of particular concern, and has not specifically been investigated in relation to PCa. Potential mechanisms include the formation of potentially carcinogenic agents such as aldehydes, acrolein, heterocyclic amines, polycyclic aromatic hydrocarbons, and acrylamide.

Methods—We estimated odds ratios (OR) and 95% confidence intervals (CI) for the association between tertiles of intake of deep-fried foods from a food frequency questionnaire (French fries, fried chicken, fried fish, doughnuts and snack chips) and PCa risk, adjusted for potential confounders, among 1,549 cases and 1,492 controls. We additionally examined associations with more aggressive PCa (defined as regional/distant stage, elevated Gleason score or prostate specific antigen level).

Results—Compared with <1/week, there was a positive association with PCa risk for intake 1/week of French fries (OR=1.37; 95% CI, 1.11–1.69), fried chicken (OR=1.30; 95% CI, 1.04–1.62), fried fish (OR=1.32; 95% CI, 1.05–1.66), and doughnuts (OR=1.35; 95% CI, 1.11–1.66). There was no association for snack chips (OR=1.08; 95% CI, 0.89–1.32). Most of the estimates were slightly stronger for more aggressive disease (OR=1.41; 95% CI, 1.04–1.92 for fried fish).

Conclusion—Regular consumption of select deep-fried foods is associated with increased PCa risk. Whether this risk is specific to deep-fried foods, or whether it represents risk associated with regular intake of foods exposed to high heat and/or other aspects of the Western lifestyle, such as fast food consumption, remains to be determined.

Keywords

deep-fried foods; high-heat cooking; fast food; prostate cancer

Introduction

Environmental or lifestyle factors are estimated to account for most prostate cancer (PCa) risk, with about 58% of risk attributable to these factors [1]. Incidence of PCa is substantially different between various ethnic and geographic populations, and immigrants to countries with a high PCa incidence rate, such as the USA, experience a sharp increase in incidence within just one generation [2, 3]. For example, PCa is relatively rare in most of Asia, yet Chinese and Japanese men experience a substantial increase in incidence after

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migration to the USA [3, 4]. Customary diets in regions of the world with low PCa incidence rates, such as Asia, are different with respect to multiple factors from usual diets consumed in Western countries such as the USA, and may represent an important environmental trigger for the development of PCa.

Factors that have been proposed to characterize a Western-style diet, such as excess dietary fat and consumption of red meat, have been investigated in relation to PCa risk [5, 6]. Results from meta-analyses suggested a small positive association between total dietary fat consumption and PCa risk [5], and animal studies indicated an association between high fat intake and increased tumor growth [7, 8]. It has been suggested that excess dietary fat may impact the insulin-like growth factor (IGF) system and/or alter hormone metabolism [8, 9], which may influence PCa risk. However, strong evidence of a meaningful association between dietary fat and PCa in humans is lacking [5]. Although several case-control and cohort studies, including our own [10], have observed an increased risk of PCa associated with consumption of red meat [6, 11], it was concluded in a recent meta-analysis of prospective studies that evidence did not support an independent positive association between red meat intake and overall risk of PCa [6]. However, cooking methods were not assessed in that analysis. Positive associations with high-heat cooking methods and/or well-done meat have been observed in several studies [12–15], although a few studies did not find such an association [16, 17]. A recent population-based case control study reported a positive association between PCa risk and meats cooked at high temperatures, particularly pan-fried red meat and poultry [18].

We propose that the consumption of foods cooked in fat/oil that has been exposed to very high heat, as opposed to consumption of gently-heated or raw fats/oils, may be a key factor in any risk associated with fat consumption. Deep frying calls for high temperatures of approximately 350–370°F, usually using polyunsaturated seed/vegetable oils such as canola, corn and soybean oil, which are easily oxidized [19] and generate potentially carcinogenic/mutagenic compounds such as aldehydes [20] and acrolein [21]. Heterocyclic amines (HCAs) and polycyclic aromatic hydrocarbons (PAH) are known carcinogens, and are formed when muscle meats are cooked at high temperatures [22–24]. The carcinogen, acrylamide, is formed when carbohydrate-rich foods are exposed to high-temperature cooking such as deep frying [25].

We investigated whether the consumption of deep-fried foods as measured by food frequency questionnaires (FFQs) from two large population-based case-control studies is associated with risk of developing PCa. We additionally investigated associations with more aggressive disease features.

Materials and Methods

Study Population and Interview Data

We used data from two prior population-based case-control studies, as previously described [26, 27]. Study participants were Caucasian and African American residents of King County, Washington. Cases from the first study were 40–64 year old men diagnosed with histologically confirmed prostate cancer between 1993 and 1996. Cases from the second study were 35–74 year old men diagnosed with histologically confirmed prostate cancer between 2002 and 2005. For both studies, cases were identified from the metropolitan Seattle-Puget Sound population-based tumor registry that is operated as part of the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program. The case participation proportions were 82% and 75% for the first and second study, respectively.

Controls were participants without a self-reported physician's diagnosis of PCa, and were recruited by random-digit telephone dialing. In both studies, household census information was obtained, and men within the study age range and without a prior diagnosis of PCa and who agreed to receive information, were contacted. Controls were recruited evenly throughout the ascertainment period for cases, and were matched to cases by 5-year age groups in both studies. The overall response proportions for control subjects were 75% and 63% for the first and second study respectively.

As previously described [26, 27], we interviewed study participants using a structured questionnaire eliciting demographic characteristics, medical and lifestyle history, and PCa screening history. Body mass index (BMI) was calculated as $\text{weight(kg)/height(m}^2\text{)}$ and was determined from self-reported height and weight one year prior to the reference date (date of diagnosis for cases and a randomly assigned date for controls that approximated the distribution of cases' diagnosis dates).

After the interview, participants were given a FFQ and asked to complete it at home and return it by mail. The FFQ assessed usual dietary intake during the 3–5 years before the reference date. The FFQ was divided into three sections: adjustment questions, food items, and summary questions. The adjustment items queried types of foods and preparation techniques, and were used to alter calculations of the nutrient content of specific food items. The food items section consisted of 99 items and queried usual frequency of specific foods or food groups (from “never or less than once a month” to “2+ times/day”) and portion size (small, medium or large, compared to the stated medium portion size). Summary questions at the end of the FFQ consisted of questions on usual intake of fruits and vegetables and use of fat/oil in cooking. The first study included the summary question “How often did you use butter, margarine, oil or other fat in cooking?” and the second study asked “How often did you eat foods that were cooked in fat (pan-fried, sautéed, or deep-fried?) Count all fat such as margarine, butter, oil or lard.” The nutrient database used to analyze the FFQ was derived from the Nutrition Data Systems for Research (NDS-R, University of Minnesota Nutrition Coordinating Center), utilizing the USDA Nutrient Database for Standard Reference as well as information from food manufacturers. We conducted analyses on a total of 1,549 cases and 1,492 controls with FFQ data.

We focused on five deep-fried food items queried in the two FFQs: French fries, fried chicken, fried fish, doughnuts and snack chips. Some of these items were described slightly differently in the two FFQs. The first study listed “French fries, fried potatoes,” “fried chicken,” “fried fish, fish sandwich, fried shellfish,” “doughnuts, cakes, pastries,” and “snack chips (potato chips, corn chips, cheese crackers, tortilla chips).” In the second study, we identified “French fries, fried potatoes and hash browns,” “fried chicken, including nuggets and tenders,” “fried fish, fish sandwich and fried shellfish,” “doughnuts, pies and pastries,” and “regular potato chips, tortilla chips, corn chips and puffs.” All of these categories were assumed to be comprised primarily of deep-fried foods. There were some exceptions, such as listings for “cheese crackers” in the first study, and “hash browns” in the second study; however the contribution of these single items within its general category is likely to be small. On the other hand, the category comprising doughnuts likely included a substantial proportion of non-deep-fried foods. The contribution of doughnuts was estimated at 50% of its category for both the first and second study, based on national food consumption data [28].

The Seattle–Puget Sound SEER cancer registry provided information on stage and Gleason score of prostate cancer at diagnosis for cases. The studies were approved by the Fred Hutchinson Cancer Research Center's Institutional Review Board. Written informed consent was obtained from all study participants prior to participation.

Data Analysis

We grouped consumption of deep-fried foods into tertiles based on straightforward time measures (e.g. weeks, months) that provided a relatively similar distribution among controls of frequency of intake across the different foods: (1) < 1/month, (2) 1–3 times/month, and (3) 1/week. We estimated the odds ratio (OR) and 95% confidence intervals (CI) by fitting unconditional adjusted logistic regression models to examine the association between risk of PCa and tertiles of consumption for each deep-fried food, with the lowest category (< 1/month) as the referent group. We repeated analyses after excluding 67 cases and 70 controls with extremely low or high calorie intakes (<800 kcal/day; > 5000 kcal/day).

We assessed the following factors as potential confounders: race, family history of prostate cancer in a first-degree relative (yes/no), BMI (categorical), screening history by prostate specific antigen (PSA) and/or digital rectal examination (DRE) testing in the previous 5 years (categorical), education (categorical), total energy (in kilocalories), and total fat consumption (as percentage of total energy). None of these covariates changed the age-adjusted estimates by more than 10%; however, for completeness, we also reported estimates adjusted for age, race, family history, BMI, PSA and DRE screening history, and education. We additionally assessed whether any of these covariates were effect modifiers by including a product term with deep-fried foods in adjusted models, and using a log-likelihood ratio test to compare logistic models with and without the product terms.

We characterized tumors by a composite measure of aggressiveness and conducted polytomous logistic regression with respect to consumption of deep-fried foods, adjusted for age, race, family history, BMI, PSA and DRE screening history, and education. Tumors were classified as “less aggressive” if they met the following criteria: local stage, Gleason score 2–6 or 7 (3 + 4), and diagnostic serum PSA <20 ng/mL. “More aggressive” cancer was defined by regional/distant stage, Gleason score 7 (4 + 3) or 8–10, or diagnostic serum PSA ≥ 20 ng/mL. Clinical stage and biopsy Gleason score were used for men who did not undergo radical prostatectomy as primary therapy. For patients treated with surgery, pathological stage and Gleason score were used to define aggressiveness.

In secondary analyses, we examined whether tertiles of fat/oil used in cooking were associated with PCa risk. Although these questions did not ask about deep-frying as a separate category, these analyses were conducted to provide additional information on the overall risk of cooking food using fat/oil, and possibly provide insight into the specificity of deep frying as a risk factor for PCa. We categorized by: (1) < 1/week (reference), (2) 1–4 times/week, and (3) ≥ 5 times/week. All tests were two-sided and statistical significance was set at $p < 0.05$. We used Stata statistical software (version 11.0, STATA Corporation, College Station, TX) for all analyses.

Results

There were 1,549 cases and 1,492 controls in the present study. Cases and controls were similar in age, race, education level, and BMI (Table 1). Cases were more likely than controls to have a first-degree family member with PCa, and were more likely to have undergone PSA/DRE testing within the five years prior to their reference date. Most PCa patients had localized disease and low/moderate Gleason score.

Compared with the reference (less than once per month), consumption of French fries, fried chicken, fried fish and doughnuts at least once per week was associated with increased PCa risk, adjusted for age, race, family history of prostate cancer, body mass index, PSA/DRE tests in previous 5 years, and education. (Table 2). Compared with the reference, intake 1/week of French fries was associated with a 37% increased risk of PCa (95% CI, 1.11–1.69);

fried chicken with a 30% increased risk (95% CI, 1.04–1.62); fried fish with a 32% increased risk (95% CI, 1.05–1.66); and doughnuts with a 35% increased risk of PCa (95% CI, 1.11–1.66). There was no association for the highest tertile of snack chips compared with the lowest tertile (OR=1.08; 95% CI, 0.89–1.32). Adjustment for age alone did not materially change the estimates (Table 2). We did not observe effect modification of the estimates by any of these covariates. Results did not change materially after exclusion of men with extreme calorie intake (results not shown).

There was a trend toward higher risk of PCa across tertiles for French fries ($p=0.004$) and doughnuts ($p=0.01$), but not for the other selected deep-fried foods (Table 2). With the exception of French fries and doughnuts, we observed no increased risk of PCa for consumption of fried chicken, fried fish and snack chips at an intake level of 1–3 times/month compared with <1/month.

Of the 1,549 cases in the present study, 1,015 participants were classified as having less aggressive PCa and 534 with having more aggressive PCa. Adjusted association estimates were slightly stronger for more aggressive PCa than less aggressive disease in a polytomous model; however the heterogeneity between groups was not significant (Table 3). Compared with the reference (<1/month), intake 1/week of French fries was associated with a 41% increased risk of more aggressive PCa (95% CI, 1.05–1.87); fried chicken with a 30% increased risk (95% CI, 0.97–1.75); fried fish with a 41% increased risk (95% CI, 1.04–1.92); doughnuts with a 38% increased risk of more aggressive PCa (95% CI, 1.06–1.80); and snack chips with a 14% increased risk of more aggressive PCa (95% CI, 0.87–1.31).

There were 1,542 cases and 1,486 controls with data on the frequency of cooking food in fat/oil. We observed a weak association between fat/oil used in cooking and risk of PCa: compared with the lowest tertile (<1/week), those in the second tertile (1–4/week) had a 14% increased risk of PCa (95% CI, 0.95–1.37), and those in the highest tertile (≥5/week) of intake had an 11% increased risk of PCa (95% CI, 0.95–1.37) in models adjusted for age. With further adjustment for race, family history of prostate cancer, BMI, PSA/DRE tests in previous 5 years, and education, those in the second tertile had a 26% increased risk of PCa (95% CI, 1.04–1.52), and those in the highest tertile had a 28% increased risk of PCa (95% CI, 1.03–1.60) compared with the lowest tertile.

Discussion

We observed positive associations between PCa risk and intake 1/week of French fries, fried chicken, fried fish and doughnuts compared with intake < 1/week, with increased risk of PCa for these deep-fried foods ranging between 27% and 35%. Although we observed an association between PCa risk and intake 1–3 times/month for French fries and doughnuts, the associations between PCa risk and select deep-fried foods appeared to be primarily confined to the highest tertile of consumption (≥1/week), suggesting that it is regular consumption of these deep-fried foods that confers particular risk for development of PCa. The associations observed for intake 1/week of French fries, fried fish and doughnuts additionally appeared to be slightly stronger for more aggressive PCa compared with less aggressive PCa, with increased risk of aggressive PCa ranging between 38% and 41% for the highest compared with the lowest tertile of intake of these deep-fried foods.

We did not observe an association between intake of snack chips and risk of PCa. The reason for this is unclear. Snack items that are consumed between meals are frequently forgotten or underreported, reducing complete exposure identification. Baked chips, crackers and items such as “puffs,” which are not deep-fried, may also have been included in this category to a larger extent than originally assumed, which may have biased results

closer to the null. There may additionally be characteristics that differ between men who regularly consume snack chips compared with men who regularly consume deep-fried foods such as fried chicken and fish; however analyses of variables such as total fat intake did not reveal any meaningful differences (results not shown). We observed a weak association between fat/oil used in cooking and risk of PCa. However, this exposure included lower-heat cooking methods, such as sautéing. In addition, oil/fat added toward the end of the cooking process for flavor may also have been included in this category; thus further reducing our ability to isolate deep-frying as an exposure.

In a population-based study of 1096 controls, 717 localized and 1140 advanced cases, Joshi et al. recently observed an increased risk of advanced PCa associated with the highest versus the lowest quartile of consumption of pan-fried meat (OR=1.4; 95% CI, 1.0–1.8) adjusted for age, BMI, total calorie intake, and family history of PCa [18]. Using the same cohort of men, they also reported increased relative risks of advanced PCa associated with high intake of white fish cooked with high-temperature methods (pan-frying, oven-broiling and grilling) [29]. Pan-frying is similar to deep-frying as it involves use of high heat and oil; however, generally the amount of oil used in pan-frying is only just enough to coat the pan, and temperatures may not be as high as those used for deep-frying.

An association between well-done meat and risk of PCa has been observed in several studies. High-temperature cooking methods are generally used to produce well-done meat, and longer lengths of cooking time allow for larger amounts of potential mutagens, such as heterocyclic amines, to accumulate. In a population-based case-control study, Norrish et al. reported an increased risk of PCa for well-done beef (RR=1.68; 95% CI, 1.02–2.77) [12]. In a US prospective cohort study, Cross et al. observed that consumption of more than 10g per day of very well done meat compared with no intake of well-done meat was associated with increased risk of PCa (RR=1.69; 95% CI, 1.19–2.40) [13]. In a US prospective cohort study, Koutros et al. reported an increased risk of PCa associated with the highest tertile of well-done meat (RR=1.26; 95% CI, 1.02–1.54), and greater risk for advanced disease (RR=1.97; 95% CI, 1.26–3.08) [14]. None of the aforementioned studies observed an association between total meat intake and risk of PCa. In another prospective cohort study, Sinha et al. observed an increased risk of PCa associated with barbecued/grilled meat [15]. However, an association between pan-fried meat and risk of PCa was not observed in these prospective studies. In addition, no associations between well done meat and PCa risk were observed in a multiethnic nested case-control study [16] and a European prospective cohort study [17].

To the best of our knowledge, the intake of deep-fried food has not been specifically studied with respect to PCa; however, increased risks of other types of cancers have been reported in association with consumption of deep-fried foods. In a population-based case-control study among Chinese women in Shanghai, there was an increased risk of breast cancer associated with high intake of deep-fried red meat (OR=1.92; 95% CI, 1.30–2.83) and deep-fried fish (OR=1.52; 95% CI, 1.05–2.22) [30]. Intake of deep-fried food has also been associated with risk of pancreatic [31] and lung [32] cancers. Consumption of fried foods in general has been associated with oral/pharyngeal [33], esophageal [33], and laryngeal [34] cancers.

Foods cooked with high heat contain high levels of advanced glycation endproducts (AGEs) [35]. Food-derived AGEs have been associated with increased oxidative stress and pro-inflammatory effects [36, 37], and a low-AGE diet (obtained through avoidance of high heat cooking methods) has been shown to reduce markers of oxidative stress and inflammation [38, 39]. Deep-fried foods are among the very highest AGE-content foods. For example, a chicken breast deep-fried for 20 minutes has more than nine times the amount of AGEs as a chicken breast boiled for an hour [35]. Chronic inflammation may play a key role in prostate carcinogenesis [40], and studies with prostate cancer cell lines suggest that interactions

between AGEs and the receptor for AGE (RAGE) may be a driver in prostate cancer progression [41, 42].

The carcinogen, acrylamide, is formed in large amounts during deep-frying of common foods, in particular carbohydrate-rich foods such as potatoes [25]. The association between acrylamide and PCa risk has been studied in two case-control and four prospective cohort studies (see Lipworth et al. for a review [43]). None of these studies found an association between acrylamide and PCa risk. However, food items such as coffee are one of the biggest contributors to acrylamide intake [44]; thus acrylamide cannot be used as a proxy for deep-fried food consumption. In addition, we did not find an association between PCa risk and snack chips, which are likely to contain high amounts of acrylamide. Taken together, this evidence suggests that the increased risk of PCa that we observed for intake of French fries and doughnuts is likely not due to acrylamide (or acrylamide alone).

Other potential mechanisms for the carcinogenicity of deep-fried foods are heterocyclic amines (HCAs) and polycyclic aromatic hydrocarbons (PAHs), which are formed when muscle meats, including chicken and fish, are cooked at high temperatures [23, 24, 45]. However, the increased risk of PCa that we observed in association with French fries and doughnuts was similar to that observed for chicken and fish, suggesting a role for agents other than HCAs and PAHs.

Characteristics of oil/fat exposed to high temperatures may be a primary risk factor. Deep-frying changes the chemical structure of oils through oxidation and hydrogenation, decreasing unsaturated fats and increasing trans fatty acids [19]. The process of deep-frying generates mutagenic compounds such as aldehydes, which remain in the oil after frying, are incorporated into fried food, and metabolized in the gut [46, 47]. Toxic compounds are increased with re-use of oil and increased length of frying time. Deep-fried foods such as French fries, fried chicken and fried fish are common food choices at restaurants, especially fast food restaurants, and these types of foods are likely to be primarily consumed outside of the home. It is possible that the association between regular intake of these foods and PCa risk that we observed may be a marker of high fast food consumption in general. In addition to high-heat-exposed fats, fast food is generally high in refined carbohydrate, sugars, salt, as well as flavorings and additives such as sodium phosphate and sodium nitrite/nitrate. Fast food consumption has increased dramatically over the past several decades, accounting for an increasing proportion of energy intake [48, 49]. The number of fast food restaurants has increased substantially, particularly in urban Black neighborhoods [50], a demographic correlated with particularly high PCa incidence and mortality [51]. It is notable that we did not observe an association between intake of snack chips and PCa risk. This result may be consistent with the possibility that a habitual fast food diet, not individual deep-fried foods, is the underlying risk factor.

In an analysis of 68 national chain fast food restaurants, researchers reported that 69% of these restaurants used corn oil to prepare French fries, followed by canola, soybean, cottonseed, sunflower and palm oil [52]. These oils are very high in omega-6 fatty acids, which have been shown to induce proliferation of human prostate tumor cells [53]. In rodent experiments, omega-6-rich diets resulted in rapid prostate tumor growth and progression [54, 55]. Results in human populations have been mixed, with some studies reporting an increased risk of PCa associated with omega-6 intake [56], and others failing to find such an association [57]. We did not observe an association between omega-6 fatty acids and PCa risk in our data (results not shown). However the possibility remains that intake of omega-6 oils exposed to high heat may be of particular concern, a factor that we were not able to isolate from our data.

The association that we observed between select deep-fried foods and PCa risk may be a dietary marker for other dietary choices, such as lack of fruit and vegetable consumption. We previously reported that high consumption of vegetables, particularly cruciferous vegetables, was associated with a reduced relative risk of PCa [58]. A similar reduction in risk estimates for certain vegetables has been confirmed in several, although not all, additional studies (see Hori et al. for a review [59]). To explore this possibility, we additionally adjusted primary analyses for daily vegetable consumption to determine whether there was an association between deep-fried foods and PCa risk over and above any potential correlation with vegetable intake. Additional adjustment for this variable did not change the estimates: for example, the age-adjusted association between PCa risk and intake 1/week of French fries (OR=1.31; 95% CI, 1.07–1.60) was almost identical after additional adjustment for daily vegetable intake (OR=1.30; 95% CI, 1.07–1.59). However, the possibility remains that consumption of deep-fried foods is linked to other unhealthy behaviors that are associated with PCa, and that we were unable to measure or measure adequately.

The present study has several other limitations. Recall bias is a possibility; however, we have no reason to believe that this misclassification would differ between cases and controls; thus any error from this source is most likely to bias results toward the null. Although we wanted to investigate deep-fried foods specifically, some of the categories contained foods cooked by other methods (such as “hash browns” within the French fries category). However, exposure classification from this source is likely to be minor. The category most affected by this was the doughnuts category, which also comprised foods such as pastries and pies. We used national food consumption data to estimate the likely proportion of doughnuts in this category. In addition, any potential bias from this type of exposure misclassification is expected to be towards the null.

Conclusions

In this population-based case-control study, we observed a moderate association between PCa risk and regular consumption (at least once per week) of deep-fried foods, including French fries, fried chicken, fried fish, and doughnuts. We noted slightly stronger associations with more aggressive disease, suggesting that regular intake of deep-fried foods may contribute to progression of PCa. The observed associations with PCa risk may be particular to the deep-fried foods identified, but may also represent an association with regular consumption of foods exposed to very high heat, or possibly regular consumption of food from chain and fast food restaurants. This study adds to the body of evidence suggesting that foods subjected to high heat and/or other methods of cooking/processing common in the Western diet may represent a potentially modifiable risk factor for PCa.

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Table 1

Distributions for selected characteristics among prostate cancer cases and controls, King County, Washington, 1993–1996 and 2002–2005

Characteristic	Cases (n=1,549)		Controls (n=1,492)	
	N	%	N	%
Age (years)				
< 50	110	7.1	121	8.1
50–59	610	39.4	618	41.4
60–69	654	42.2	587	39.3
70–74	175	11.3	166	11.1
Race				
Caucasian	1,402	90.5	1,398	93.7
African American	147	9.5	94	6.3
Family history of prostate cancer				
No	1,231	79.5	1,328	89.0
Yes	318	20.5	164	11.0
Education level				
High school or less	306	19.8	277	18.6
Some college	394	25.4	361	24.2
Undergraduate degree	426	27.5	417	28.0
Graduate degree	423	27.3	437	29.3
Body mass index, kg/m ² ¹				
< 25.0	493	31.8	456	30.6
25.0 – 29.9	753	48.6	708	47.5
30+	303	19.6	328	22.0
PSA/DRE test status ²				
Never tested	167	10.8	216	14.5
DRE only	285	18.4	553	37.1
PSA	1,097	70.8	723	48.5
Stage of disease				
Local	1,205	77.8		
Regional	304	19.6		
Distant	40	2.6		
Gleason score				
Low (2–4)	88	5.7		
Moderate [5–7 (3+4)]	1,203	77.7		
High [7 (4+3), 8–10]	254	16.4		
Missing	4	0.3		

¹Body mass index = weight in kilograms/(height in meters)².

²Tests performed in the 5 years before reference date.

Odds ratios of prostate cancer associated with deep-fried foods, King County, Washington, 1993–1996 and 2002–2005

Table 2

Food item	Cases (n=1,549)		Controls (n=1,492)		Age-adjusted model			Fully adjusted model ^I		
	n	%	n	%	OR	(95% CI)	P (trend)	OR	(95% CI)	P (trend)
French fries										
< 1 per month	285	(18.4)	319	(21.4)	1.00	(referent)	0.01	1.00	(referent)	0.004
1–3 times per month	676	(43.6)	658	(44.1)	1.17	(0.96–1.42)		1.20	(0.98–1.47)	
1 per week	578	(37.3)	510	(34.2)	1.31	(1.07–1.60)		1.37	(1.11–1.69)	
missing	10	(0.7)	5	(0.3)						
Fried chicken										
< 1 per month	575	(37.1)	565	(37.9)	1.00	(referent)	0.08	1.00	(referent)	0.05
1–3 times per month	658	(42.5)	679	(45.5)	0.96	(0.82–1.12)		1.00	(0.85–1.18)	
1 per week	305	(19.7)	238	(16.0)	1.27	(1.03–1.56)		1.30	(1.04–1.62)	
missing	11	(0.7)	10	(0.7)						
Fried fish										
< 1 per month	621	(40.1)	604	(40.5)	1.00	(referent)	0.12	1.00	(referent)	0.07
1–3 times per month	661	(42.7)	678	(45.4)	0.96	(0.82–1.11)		0.97	(0.83–1.14)	
1 per week	257	(16.6)	197	(13.2)	1.27	(1.02–1.58)		1.32	(1.05–1.66)	
missing	10	(0.7)	13	(0.9)						
Doughnuts										
< 1 per month	654	(42.2)	675	(45.2)	1.00	(referent)	0.004	1.00	(referent)	0.01
1–3 times per month	541	(34.9)	536	(35.9)	1.06	(0.90–1.24)		1.08	(0.91–1.27)	
1 per week	350	(22.6)	273	(18.3)	1.35	(1.11–1.64)		1.35	(1.11–1.66)	
missing	4	(0.3)	8	(0.5)						
Snack chips										
< 1 per month	360	(23.2)	328	(22.0)	1.00	(referent)	0.61	1.00	(referent)	0.31
1–3 times per month	581	(37.5)	601	(40.3)	0.90	(0.74–1.09)		0.92	(0.76–1.12)	
1 per week	600	(38.7)	553	(37.1)	1.02	(0.85–1.24)		1.08	(0.89–1.32)	
missing	8	(0.5)	10	(0.7)						

^I Adjusted for age, race, family history of prostate cancer, body mass index, PSA/DRE tests in previous 5 years, and education.

Table 3

Odds ratios[/] of prostate cancer associated with deep-fried foods, stratified by tumor aggressiveness, King County, Washington, 1993–1996 and 2002–2005.

Food item	Less aggressive (n=1,015)			More aggressive (n=534)		
	OR	(95% CI)	P (trend)	OR	(95% CI)	P (trend)
French fries						
< 1 per month	1.00	(referent)	0.02	1.00	(referent)	0.01
1–3 times per month	1.21	(0.97–1.43)		1.16	(0.88–1.54)	
1 per week	1.35	(1.06–1.71)		1.41	(1.05–1.87)	0.81
Fried chicken						
< 1 per month	1.00	(referent)	0.10	1.00	(referent)	0.12
1–3 times per month	0.99	(0.82–1.19)		1.02	(0.81–1.28)	
1 per week	1.29	(1.00–1.65)		1.30	(0.97–1.75)	0.94
Fried fish						
< 1 per month	1.00	(referent)	0.29	1.00	(referent)	0.03
1–3 times per month	0.89	(0.74–1.07)		1.14	(0.92–1.43)	
1 per week	1.27	(0.99–1.64)		1.41	(1.04–1.92)	0.52
Doughnuts						
< 1 per month	1.00	(referent)	0.01	1.00	(referent)	0.03
1–3 times per month	1.10	(0.91–1.33)		1.03	(0.82–1.30)	
1 per week	1.33	(1.06–1.67)		1.38	(1.06–1.80)	0.83
Snack chips						
< 1 per month	1.00	(referent)	0.48	1.00	(referent)	0.29
1–3 times per month	0.89	(0.71–1.10)		1.00	(0.76–1.31)	
1 per week	1.05	(0.84–1.32)		1.14	(0.87–1.31)	0.59

[/] Adjusted for age, race, family history of prostate cancer, body mass index, PSA/DRE tests in previous 5 years, and education.