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Associations of tea and coffee consumption with prostate cancer risk

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Abstract

Purpose: Tea and coffee contain bioactive compounds and both beverages have recently been associated with a reduced risk of prostate cancer (PCa).

Methods: We studied associations of tea and coffee consumption with PCa risk in a population-based case-control study from King County, Washington, US. Prostate cancer cases were diagnosed in 2002-2005 and matched to controls by five-year age groups. Logistic regression was used to generate odds ratios (ORs) and 95% confidence intervals (CIs).

Results: Among controls, 19% and 58% consumed at least one cup per day of tea and coffee, respectively. The analysis of tea included 892 cases and 863 controls and tea consumption was associated with a reduced overall PCa risk with an adjusted OR of 0.63 (95% CI: 0.45, 0.90; P for trend = 0.02) for men in the highest compared to lowest category of tea intake (≥2 cups/day versus ≤1 cup/week). Risk estimates did not vary substantially by Gleason grade or disease stage. Coffee consumption was not associated with risk of overall PCa or PCa in subgroups defined by tumor grade or stage.

Conclusions: Our results contribute further evidence that tea consumption may be a modifiable exposure that reduces PCa risk.

Keywords

prostate cancer; tea; coffee; risk; stage; Gleason grade

Background

Tea and coffee contain biologically active compounds that may have anti-cancer effects. While the biological properties of tea are mostly attributed to its polyphenol content (1, 2), coffee is a complex mixture of many potentially chemopreventive agents such as caffeine, cafestol, kahweal, polyphenols, chlorogenic acid, and caffeic acid (3-6).

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Prostate cancer (PCa) is the most frequent cancer among men in developed countries (7). Compared with other common cancers such as breast and lung cancer, however, very few modifiable PCa risk or protective factors have been identified. Both tea and coffee consumption have recently been associated with a decreased risk of PCa in one large-scale study each (8, 9). The study of tea, which was conducted in the Netherlands, showed that tea consumption was inversely associated with the risk of advanced stage PCa, but not overall or non-advanced PCa (8). The study of coffee, which was conducted among US Health professionals, showed an association with a modest decrease in risk of overall PCa and a non-significant association with a decreased risk of high grade PCa (9). The study additionally showed that coffee consumption was associated with a substantial decrease in risk of lethal PCa (defined as PCa-specific death or metastatic disease) (9). A number of other studies of tea and coffee consumption in relation to PCa risk have been conducted and the results of those studies have been inconclusive (10, 11). Most of those prior studies, however, had some limitations related to sample size, lack of a wide range of consumption, and no data on clinical features of PCa such as tumor stage and grade.

To further investigate this, we performed a population-based case-control study of PCa and both tea and coffee consumption among men from King County, Washington, US. We investigated overall PCa risk and PCa risk in subgroups based on disease stage and grade.

Materials and Methods

Study population

Study participants were Caucasian and African-American men, aged 35 to 74 years, enrolled in a population-based case-control study conducted in King County, Washington, US (12). Incident cases (n = 1,001) were diagnosed with histologically confirmed adenocarcinoma of the prostate from January 1, 2002, through December 31, 2005, and were identified via the Seattle-Puget Sound Surveillance, Epidemiology, and End Results (SEER) Program cancer registry. This registry provided information on Gleason score, tumor stage, and serum prostate-specific antigen (PSA) level at diagnosis. Control subjects (n = 942) were identified via random digit dialing, frequency matched to cases by five-year age groups, and recruited evenly throughout the ascertainment period for cases. The study was approved by the institutional review board of the Fred Hutchinson Cancer Research Center, and written informed consent was obtained from all participants.

Exposure assessment

Dietary intake of study participants was derived from a 120-item food frequency questionnaire (FFQ) (13), that concentrated on intake two years prior to the reference date (date of diagnosis for cases and an assigned date for controls that approximated the distribution of the diagnosis dates of cases). With regard to tea and coffee consumption, frequency of intake was assessed by using nine categories that ranged from never or less than once a month to six or more times per day. Although the type of tea was not specified, it is assumed that men in this US-based study rarely drink tea other than black tea (14). The FFQ was completed by 897 cases (90%) and 865 controls (92%). An additional five cases and two controls had missing data on tea consumption and were excluded from those analyses (892 cases, 863 controls). An additional three cases and five controls had missing data on coffee consumption and were excluded from those analyses (894 cases, 860 controls). Subjects also completed a structured, in-person interview, administered by a trained male interviewer, about demographic and lifestyle information, family history of cancer, medical history, medication use, and PCa screening history prior to the reference date.

Statistical analyses

Tea and coffee consumption was tabulated according to a number of selected characteristics. Unconditional logistic regression was used to calculate odds ratios (ORs) and corresponding 95% confidence intervals (CIs) for the associations between tea and coffee consumption and overall PCa risk and PCa risk in subgroups defined by Gleason grade and disease stage. We modeled four categories of tea consumption (≤1/week; 2-6/week; 1/day; ≥2/day) and five categories of coffee consumption (≤1/week; 2-6/week; 1/day; 2-3/day; ≥4/day) with the lowest category (≤1/week) as the referent group. We completed age-adjusted (five-year age groups) models and multivariable models that were additionally adjusted for race (Caucasian, African-American), first-degree family history of prostate cancer (no, yes), smoking status (never, former, current), and prostate cancer screening within the five-year period prior to the reference date (none, digital rectal examination only, PSA testing). P values for linear trends were calculated by using median values within consumption categories. We also modeled tea and coffee consumption as a continuous variable (one cup per week increment). The following factors were additionally assessed as potential confounders: education, body mass index (BMI), lifetime alcohol consumption, recent exercise frequency, aspirin use, non-aspirin NSAID use, diabetes mellitus, energy-adjusted total fat intake, and consumption of tea (when studying coffee) or coffee (when studying tea). None of these was included in the final models, as they had little effect on the effect estimates or precision. Effect modification of the association between tea and coffee consumption and PCa by reference age (median age of controls as cut-off value; <62, ≥62 years) was tested by using cross-product terms in the regression model. The Wald statistic was used to test for interaction. Risk differences by race and family history of PCa were not examined due to limited sample sizes. All P values were two-sided and a P value less than 0.05 was considered to be statistically significant. Analyses were performed using STATA software (release 11, STATA Corporation, College Station, TX).

Results

For the analysis of tea there were 892 PCa cases and 863 controls and for the analysis of coffee there were 894 cases and 860 controls. Prostate cancer cases compared to controls were more likely to be African-American (P = 0.03), have a family history of PCa in a first-degree relative (P <0.01), and to have had PSA testing prior to the reference date (P <0.01) (data not in tables). Among control subjects, 19% consumed at least one cup of tea per day and 11% were in the highest category of tea consumption (≥2 cups/day). Tea consumption was associated with older age, less (current and former) cigarette smoking, and lower coffee consumption (all P <0.01) (Table 1). Among controls, 58% consumed at least one cup of coffee per day and 12% were in the highest category of coffee consumption (≥4 cups/day). Coffee consumption was higher in Caucasians and men who ever smoked and coffee consumption was lower in those reporting tea consumption (all P <0.01) (Table 1).

Tea consumption was associated with a decreased risk of overall PCa with an adjusted OR of 0.63 (95% CI: 0.45, 0.90; P for trend = 0.02) for men in the highest compared to lowest category of consumption (≥2 cups/day versus ≤1 cup/week) (Table 2). The adjusted OR for an increment of one cup of tea per week was 0.98 (95% CI: 0.96, 0.99). Risk estimates for men in the highest compared to lowest category of tea intake did not vary substantially by Gleason grade or stage of PCa. The association of tea consumption and overall PCa risk was similar when the analysis was restricted to men who received PSA testing within the five-year period before reference date (adjusted OR for an increment of one cup per week of 0.97; 95% CI: 0.95, 0.99) or men who were never smokers (adjusted OR for an increment of one cup per week of 0.98; 95% CI: 0.96, 1.00) (data not in tables).

Coffee consumption was not associated with risk of overall PCa; the adjusted OR for the highest versus lowest category of intake (≥4 cups/day versus ≤1 cup/week) was 1.16 (95% CI: 0.82, 1.63; P for trend = 0.32) (Table 3). No associations between coffee consumption and a decreased PCa risk were observed in subgroups based on Gleason grade and disease stage. We, however, observed a statistically significant association with an increased risk of Gleason 7(4+3)-10 PCa for men consuming 2-6 cups/week versus ≤1cup/week (adjusted OR = 1.72; 95% CI: 1.00, 2.97). Coffee consumption was not associated with overall PCa risk when the analysis was restricted to men with a positive PSA screening history or men who were never smokers (data not in tables).

The association between tea and coffee consumption and overall PCa risk was not modified by reference age (data not in tables). We observed a slightly stronger association between tea consumption and overall PCa risk in the older age group (≥62 years), but there was no significant interaction (P for interaction >0.05) (data not in tables).

Discussion

In this population-based case-control study, tea consumption was associated with a reduced risk of PCa. No associations were observed for coffee. The ORs were similar after multivariable adjustment, which implies that there was no substantial confounding by any of the additionally included covariables.

Our finding of an inverse association between tea consumption and PCa risk is supported by the results from a recent large prospective study from the Netherlands Cohort Study (8). Similar to that study, men in our US-based population were assumed to rarely drink any other tea than black tea (14). The study from the Netherlands was conducted among 58,279 men and during 17.3 years of follow-up, 3,362 prostate cancers were identified, including 1,164 advanced (stage III/IV) cancers. The authors showed that men in the highest compared to lowest category of tea consumption (≥5 versus ≤1 cups/day) had a decreased risk of stage III/IV PCa (hazard ratio (HR) = 0.75; 95% CI: 0.59, 0.97) and stage IV PCa (HR = 0.67; 95% CI: 0.50, 0.91). In contrast to our study, no associations were observed between tea consumption and risk of overall and non-advanced PCa. Although we had low case numbers for the analysis by grade and stage, particularly for the highest intakes, we found suggestive evidence that associations with tea consumption were more pronounced for higher grade and more advanced stage PCa. One difference between our study and the Netherlands study is the much higher frequency of tea consumption in the Netherlands, where the highest and lowest categories of tea consumption were ≥5 and ≤1 cups per day, respectively. In our US-based case-control study, only 19% of control subjects consumed one or more cups of tea per day. The actual tea intake in both populations, however, may be not that different because it is estimated that the standard cup size in the US is approximately two times larger than in the Netherlands (8oz versus 125 mL or ≈ 4oz) (15).

The inverse association of tea consumption with PCa risk that we observed was consistent with one previous case-control study from Canada (16). The study included 617 cases and 637 controls and showed that men who consumed two cups or more per day compared to non-drinkers had an OR of overall PCa of 0.70 (95% CI: 0.50, 0.99). An inverse association of black tea consumption and overall PCa risk was, however, not found in a meta-analysis of 11 studies (three prospective cohort studies, two retrospective cohort studies, and six case-control studies) including a total of 3,367 PCa cases (11). In subgroup analyses, the authors did show that the risk reduction was stronger among prospective cohort studies (OR = 0.83; 95% CI: 0.63, 1.08) than case-control studies (11). There have been two small-scale, prospective studies of black tea consumption and PCa risk, not included in the meta-analysis described above, that showed a positive association (17, 18). The contradictory evidence

from observational studies of black tea and PCa may be attributed to the differences in tea consumption pattern and drinking habits across the various study populations. These differences may be related to brewing method (e.g. infusion time), the brand of black tea, and possibly leaf size and tea bag porosity, which affects the concentration of bioactive tea compounds (19). Most observational studies, including ours, had no data on these variables. Also, interpretation of many studies may be hampered by the possibility that recall and selection biases or insufficient control of confounders influenced the results. Furthermore, tea consumption in some studies was probably too low to find a meaningful association.

About 78% of all tea produced worldwide is black tea, which is the most commonly consumed type of tea in the Western world (14). Green tea, which is popular in Japan and parts of China, accounts for about 20% of worldwide tea production (14). Both green and black tea are produced from the leaves of the plant *Camellia sinensis*, and these tea types have some chemical differences due to a different production process (14). In the processing of green tea, fresh tea leaves are heat-treated immediately after harvest, resulting in minimal oxidation of the naturally occurring polyphenols. In the processing of black tea, tea leaves are dried and crushed upon harvesting to encourage oxidation, which converts part of the endogenous tea polyphenols (primarily catechins) to theaflavins and thearubigens. Although the anti-cancer mechanisms may be different for black and green tea, potential chemopreventive effects of tea are mostly attributed to monomeric polyphenols such as catechins and flavonols, which are abundantly present in both tea types (1, 2, 20). A recent meta-analysis of PCa and consumption of green tea (seven studies) showed an association with a decreased PCa risk (11). The pooled estimate, however, only reached statistical significance for case-control studies (three studies), and not for cohort studies (four studies).

A recent report from the Health Professionals Follow-up Study by Wilson et al. showed that coffee reduced the incidence of PCa (9). That study was conducted among 47,911 US health professionals and included 5,035 prostate cancers, including 896 advanced cancers (642 metastatic or fatal cancers plus 254 additional extraprostatic cancers). The data showed that men in the highest compared to lowest category of coffee intake (≥6 cups/day versus non-consumers) had a modest decreased risk of overall PCa (HR = 0.82; 95% CI: 0.68, 0.98) and the association was more pronounced for advanced (HR = 0.47; 95% CI: 0.28, 0.77) and lethal PCa (defined as PCa-specific death or metastatic disease; HR = 0.40; 95% CI: 0.22, 0.75). Our study did not confirm the association between coffee consumption and a reduced overall PCa risk. Metastatic and fatal prostate cancers in the Wilson et al. study were identified after additional follow-up of incident prostate cancers. Our analysis was focused on PCa risk and not outcomes, and PCa progression or PCa-specific mortality were therefore not an endpoint in our study. In both our study and the Wilson et al. study, risk of PCa was assessed in subgroups on the basis of Gleason grade. Wilson et al. showed that men in the highest compared to lowest category of coffee intake had a non-significant decreased risk of Gleason 8-10 PCa (HR for ≥6 cups/day versus non-consumers = 0.53; 95% CI: 0.27, 1.02). This was not confirmed by our study where we found no evidence of a reduced risk of high grade (Gleason 7(4+3)-10) PCa (OR for ≥4 cups/day versus ≤1 cup/week = 1.04; 95% CI: 0.55, 1.96). Interestingly, we found an increased risk of high grade PCa for men consuming 2-6 cups of coffee per week versus ≤1 cup/week (OR = 1.72; 95% CI: 1.00, 1.72). This latter finding was unexpected and requires further study. Another recent small-scale prospective study, among 6,016 Scottish men, showed that coffee consumption was not associated with overall PCa risk, but the investigators found a reduced risk of Gleason 8-10 PCa for men consuming 3 or more cups per day compared to non-consumers (HR = 0.45; 95% CI: 0.23, 0.90) (21). The association, however, was not significant after multivariable adjustment and it is important to note that the study included only 38 incident Gleason 8-10 prostate cancers. There has been a meta-analysis of coffee consumption and overall PCa risk (eight case-control studies and four cohort studies) and this study does not support an association

with a reduced risk (10). In subgroup analyses, there was an association with an increased PCa risk in case-control studies but not in cohort studies. The meta-analysis did not evaluate subgroups based on grade, stage, or PCa outcomes.

The potential chemopreventive effects of tea on PCa have received considerable attention and animal studies and studies using cell lines showed that extracts from green and black tea reduce PCa growth (20, 22, 23). Most of those experimental studies used polyphenolic fractions isolated from green tea or purified epigallocatechin-3-gallate (20), which is a catechin that is found almost exclusively in tea (24, 25). Tea polyphenols are mainly absorbed from the small intestine and are then subject to phase II metabolism, which likely affects their bioavailability (20). A human preprostatectomy trial, however, showed that black and green tea polyphenols are bioavailable in the prostate (26). There has been one small proof-of-principle clinical trial investigating the potential chemopreventive effects of green tea catechins on PCa that deserves particular attention (27). That study included 60 men with high grade prostate intra-epithelial neoplasia, which is thought to be a precursor of PCa (28), and men in the treatment group (n = 30) were given three (200 mg) green tea catechins capsules per day. After 1 year of follow-up, only one man in the treatment group was diagnosed with PCa compared to nine in the placebo group. Potential anticancer activities of polyphenols may be related to their antioxidant activities, effects on enzyme activities, anti-inflammatory effects, and inhibition of angiogenesis (29-31).

Strengths of our study include the population-based approach, detailed data on tumor grade and stage, and relatively wide range of tea and coffee intakes. Due to the observational nature of our study, bias due to confounding cannot be ruled out. One potentially important confounder of the association between tea consumption and PCa risk is PCa screening. In an attempt to address this possibility, we included PCa screening history in the multivariable analysis and found that the inverse association remained statistically significant. We additionally showed that when the analysis was restricted to men who received PSA testing prior to the reference date, tea consumption continued to be associated with a lower risk of overall PCa. Besides that, the high frequency if PCa screening in our US-based population makes it difficult to compare results for overall PCa with studies in populations were PCa screening is less common such as Western Europe. Another limitation is the possibility that non-participants of our study had a different prevalence of tea and coffee consumption. Although we had no data on non-participants, the prevalence of tea and coffee consumption in our control group is similar to that in other large US-based study populations (9, 15, 32). Recall bias is possible but this study was conducted prior to publications suggesting a preventive effect of tea on PCa.

In conclusion, this US population-based study showed that tea consumption is associated with a reduced PCa risk. These results are supported by prior observational and experimental studies and provide further evidence that consumption of tea may be a modifiable exposure that can reduce PCa risk.

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References

1. Higdon JV, Frei B. Tea catechins and polyphenols: health effects, metabolism, and antioxidant functions. Crit Rev Food Sci Nutr. 2003; 43:89–143. [PubMed: 12587987]

2. Yang CS, Wang H, Li GX, Yang Z, Guan F, Jin H. Cancer prevention by tea: Evidence from laboratory studies. Pharmacol Res. 2011; 64:113–22. [PubMed: 21397027]
3. Arab L. Epidemiologic evidence on coffee and cancer. Nutr Cancer. 2010; 62:271–83. [PubMed: 20358464]
4. Farah A, Monteiro M, Donangelo CM, Lafay S. Chlorogenic acids from green coffee extract are highly bioavailable in humans. J Nutr. 2008; 138:2309–15. [PubMed: 19022950]
5. Higdon JV, Frei B. Coffee and health: a review of recent human research. Crit Rev Food Sci Nutr. 2006; 46:101–23. [PubMed: 16507475]
6. Yang H, Rouse J, Lukes L, et al. Caffeine suppresses metastasis in a transgenic mouse model: a prototype molecule for prophylaxis of metastasis. Clin Exp Metastasis. 2004; 21:719–35. [PubMed: 16035617]
7. Jemal A, Bray F, Center MM, Ferlay J, Ward E, Forman D. Global cancer statistics. CA Cancer J Clin. 2011; 61:69–90. [PubMed: 21296855]
8. Geybels MS, Verhage BA, Arts IC, van Schooten FJ, Bausch RA, van den Brandt PA. Dietary flavonoid intake, black tea consumption, and risk of overall and advanced stage prostate cancer. Am J Epidemiol. 2012 Accepted for publication.
9. Wilson KM, Kasperzyk JL, Rider JR, et al. Coffee consumption and prostate cancer risk and progression in the Health Professionals Follow-up Study. J Natl Cancer Inst. 2011; 103:876–84. [PubMed: 21586702]
10. Park CH, Myung SK, Kim TY, Seo HG, Jeon YJ, Kim Y. Coffee consumption and risk of prostate cancer: a meta-analysis of epidemiological studies. BJU Int. 2010; 106:762–9. [PubMed: 20590551]
11. Zheng J, Yang B, Huang T, Yu Y, Yang J, Li D. Green tea and black tea consumption and prostate cancer risk: an exploratory meta-analysis of observational studies. Nutr Cancer. 2011; 63:663–72. [PubMed: 21667398]
12. Agalliu I, Salinas CA, Hansten PD, Ostrander EA, Stanford JL. Statin use and risk of prostate cancer: results from a population-based epidemiologic study. Am J Epidemiol. 2008; 168:250–60. [PubMed: 18556686]
13. Patterson RE, Kristal AR, Tinker LF, Carter RA, Bolton MP, Agurs-Collins T. Measurement characteristics of the Women's Health Initiative food frequency questionnaire. Ann Epidemiol. 1999; 9:178–87. [PubMed: 10192650]
14. Yuan JM, Sun C, Butler LM. Tea and cancer prevention: epidemiological studies. Pharmacol Res. 2011; 64:123–35. [PubMed: 21419224]
15. Genkinger JM, Li R, Spiegelman D, et al. Coffee, tea, and sugar-sweetened carbonated soft drink intake and pancreatic cancer risk: a pooled analysis of 14 cohort studies. Cancer Epidemiol Biomarkers Prev. 2012; 21:305–18. [PubMed: 22194529]
16. Jain MG, Hislop GT, Howe GR, Burch JD, Ghadirian P. Alcohol and other beverage use and prostate cancer risk among Canadian men. Int J Cancer. 1998; 78:707–11. [PubMed: 9833763]
17. Shafique K, McLoone P, Qureshi K, Leung H, Hart C, Morrison DS. Tea consumption and the risk of overall and grade specific prostate cancer: a large prospective cohort study of Scottish men. Nutr Cancer. 2012; 64:790–7. [PubMed: 22697604]
18. Montague JA, Butler LM, Wu AH, et al. Green and black tea intake in relation to prostate cancer risk among Singapore Chinese. Cancer Causes Control. 2012; 23:1635–41. [PubMed: 22864870]
19. Kyle JA, Morrice PC, McNeill G, Duthie GG. Effects of infusion time and addition of milk on content and absorption of polyphenols from black tea. J Agric Food Chem. 2007; 55:4889–94. [PubMed: 17489604]
20. Henning SM, Wang P, Heber D. Chemopreventive effects of tea in prostate cancer: green tea versus black tea. Mol Nutr Food Res. 2011; 55:905–20. [PubMed: 21538852]
21. Shafique K, McLoone P, Qureshi K, Leung H, Hart C, Morrison DS. Coffee consumption and prostate cancer risk: further evidence for inverse relationship. Nutr J. 2012; 11:42. [PubMed: 22695052]
22. Connors SK, Chornokur G, Kumar NB. New insights into the mechanisms of green tea catechins in the chemoprevention of prostate cancer. Nutr Cancer. 2012; 64:4–22. [PubMed: 22098273]

23. Johnson JJ, Bailey HH, Mukhtar H. Green tea polyphenols for prostate cancer chemoprevention: a translational perspective. Phytomedicine. 2010; 17:3–13. [PubMed: 19959000]
24. Arts IC, van De Putte B, Hollman PC. Catechin contents of foods commonly consumed in The Netherlands. 2. Tea, wine, fruit juices, and chocolate milk. J Agric Food Chem. 2000; 48:1752–7. [PubMed: 10820090]
25. Arts IC, van de Putte B, Hollman PC. Catechin contents of foods commonly consumed in The Netherlands. 1. Fruits, vegetables, staple foods, and processed foods. J Agric Food Chem. 2000; 48:1746–51. [PubMed: 10820089]
26. Henning SM, Aronson W, Niu Y, et al. Tea polyphenols and theaflavins are present in prostate tissue of humans and mice after green and black tea consumption. J Nutr. 2006; 136:1839–43. [PubMed: 16772446]
27. Bettuzzi S, Brausi M, Rizzi F, Castagnetti G, Peracchia G, Corti A. Chemoprevention of human prostate cancer by oral administration of green tea catechins in volunteers with high-grade prostate intraepithelial neoplasia: a preliminary report from a one-year proof-of-principle study. Cancer Res. 2006; 66:1234–40. [PubMed: 16424063]
28. Montironi R, Mazzucchelli R, Lopez-Beltran A, Scarpelli M, Cheng L. Prostatic intraepithelial neoplasia: its morphological and molecular diagnosis and clinical significance. BJU Int. 2011; 108:1394–401. [PubMed: 21883826]
29. Garcia-Lafuente A, Guillamon E, Villares A, Rostagno MA, Martinez JA. Flavonoids as anti-inflammatory agents: implications in cancer and cardiovascular disease. Inflamm Res. 2009; 58:537–52. [PubMed: 19381780]
30. Moon YJ, Wang X, Morris ME. Dietary flavonoids: effects on xenobiotic and carcinogen metabolism. Toxicol In Vitro. 2006; 20:187–210. [PubMed: 16289744]
31. Yang CS, Lambert JD, Sang S. Antioxidative and anti-carcinogenic activities of tea polyphenols. Arch Toxicol. 2009; 83:11–21. [PubMed: 19002670]
32. Sinha R, Cross AJ, Daniel CR, et al. Caffeinated and decaffeinated coffee and tea intakes and risk of colorectal cancer in a large prospective study. Am J Clin Nutr. 2012; 96:374–81. [PubMed: 22695871]

Table 1

Selected characteristics of population-based controls according to categories of tea and coffee consumption, King County, Washington, 2002-2005

	Categories of consumption (cups/unit of time)				
Tea	**≤1/week**	**2-6/week**	**1/day**	**≥2/day**	
Controls, No.	594	109	69	91	
Mean age, y (SD)	61.2 (8.4)	59.3 (7.1)	63.8 (6.9)	63.5 (7.3)	
Caucasian, %	90.7	85.3	92.8	92.3	
First-degree family history of prostate cancer, %	12.3	11.9	7.2	9.9	
College or graduate degree, %	56.4	62.4	65.2	65.9	
Mean BMI, kg/m^2 (SD)	27.9 (4.6)	27.5 (4.1)	26.5 (4.8)	27.1 (4.8)	
Never smoker, %	44.8	40.4	56.5	57.1	
Aspirin use*, %	51.3	53.2	53.6	53.8	
Non-aspirin NSAID use*, %	20.9	23.9	13.0	16.5	
Diabetes mellitus, %	10.4	11.9	10.1	9.9	
History of PSA testing†, %	58.2	52.3	60.9	71.4	
≥2 cups of coffee per day, %	46.5	35.5	32.4	24.2	
Coffee	≤1/week	2-6/week	1/day	2-3/day	≥4/day
Controls, No.	257	100	146	253	104
Mean age, y (SD)	60.3 (8.5)	60.9 (7.8)	62.1 (8.5)	62.0 (7.8)	61.4 (7.2)
Caucasian, %	86.8	81.0	90.4	95.3	96.2
First-degree family history of prostate cancer, %	11.7	10.0	11.0	13.0	10.6
College or graduate degree, %	60.3	56.0	61.0	62.5	46.2
Mean BMI, kg/m^2 (SD)	27.6 (5.2)	28.2 (4.6)	27.2 (4.1)	27.6 (4.1)	27.8 (4.5)
Never smoker, %	62.3	56.0	50.0	36.4	17.3
Aspirin use*, %	47.9	51.0	52.1	53.0	60.6
Non-aspirin NSAID use*, %	21.0	17.0	17.8	21.3	21.4
Diabetes mellitus, %	9.7	10.0	9.6	12.6	9.6
History of PSA testing†, %	55.3	51.0	67.1	61.7	57.7
≥1 cup of tea per day, %	28.9	8.1	22.6	15.8	3.8

Abbreviations: SD, standard deviation; NSAID, non-steroidal anti-inflammatory drug; PSA, prostate-specific antigen.

*Use at least once a week for 3 months or longer.

†PSA testing within the 5-year period before reference date.

Table 2

Odds ratio (OR) with 95% confidence interval (CI) of prostate cancer according to categories of tea consumption, King County, Washington, 2002-2005

	Categories of tea consumption (cups/unit of time)									
	≤1/week	2-6/week		1/day		≥2/day		P trend	Cup/week increment	
	OR	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)		OR	(95% CI)
Controls, No.	594	109		69		91				
All prostate cancers, No.	634	135		61		62				
Age-adjusted OR (95% CI)	1.00	1.17	(0.89, 1.54)	0.82	(0.57, 1.18)	0.63	(0.45, 0.89)	0.02	0.98	(0.96, 0.99)
Multivariable-adjusted OR (95% CI)*	1.00	1.13	(0.86, 1.50)	0.83	(0.58, 1.20)	0.63	(0.45, 0.90)	0.02	0.98	(0.96, 0.99)
Gleason 2-7(3+4) cancers, No.	521	105		51		53				
Age-adjusted OR (95% CI)	1.00	1.09	(0.82, 1.47)	0.85	(0.58, 1.24)	0.67	(0.47, 0.95)	0.04	0.98	(0.96, 1.00)
Multivariable-adjusted OR (95% CI)*	1.00	1.05	(0.78, 1.42)	0.86	(0.58, 1.26)	0.65	(0.45, 0.94)	0.03	0.98	(0.96, 0.99)
Gleason 7(4+3)-10 cancers, No.	116	30		10		9				
Age-adjusted OR (95% CI)	1.00	1.53	(0.97, 2.41)	0.67	(0.33, 1.35)	0.47	(0.23, 0.96)	0.07	0.98	(0.95, 1.01)
Multivariable-adjusted OR (95% CI)*	1.00	1.51	(0.96, 2.39)	0.69	(0.34, 1.40)	0.51	(0.25, 1.04)	0.11	0.98	(0.95, 1.01)
Local stage cancers, No.	510	107		49		55				
Age-adjusted OR (95% CI)	1.00	1.17	(0.87, 1.57)	0.81	(0.55, 1.19)	0.69	(0.48, 0.99)	0.06	0.98	(0.97, 1.00)
Multivariable-adjusted OR (95% CI)*	1.00	1.13	(0.84, 1.52)	0.83	(0.56, 1.23)	0.68	(0.47, 0.97)	0.06	0.98	(0.96, 1.00)
Regional/distant stage cancers, No.	124	28		12		7				
Age-adjusted OR (95% CI)	1.00	1.17	(0.74, 1.86)	0.91	(0.47, 1.73)	0.39	(0.18, 0.87)	0.06	0.96	(0.93, 1.00)
Multivariable-adjusted OR (95% CI)*	1.00	1.17	(0.74, 1.87)	0.94	(0.49, 1.80)	0.40	(0.18, 0.90)	0.08	0.96	(0.93, 1.00)

*Multivariable models were adjusted for age, race, first-degree family history of prostate cancer, smoking status, and history of prostate cancer screening.

Table 3

Odds ratio (OR) with 95% confidence interval (CI) of prostate cancer according to categories of coffee consumption, King County, Washington, 2002-2005

	Categories of coffee consumption (cups/unit of time)											
	≤1/week	2-6/week		1/day		2-3/day		≥4/day		P trend	Cup/week increment	
	OR	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)		OR	(95% CI
Controls, No.	257	100		146		253		104				
All prostate cancers, No.	246	113		154		273		108				
Age-adjusted OR (95% CI)	1.00	1.18	(0.86, 1.63)	1.10	(0.82, 1.46)	1.12	(0.88, 1.43)	1.08	(0.79, 1.49)	0.50	1.00	(0.99, 1.01)
Multivariable-adjusted OR (95% CI)*	1.00	1.22	(0.88, 1.69)	1.13	(0.84, 1.51)	1.16	(0.90, 1.50)	1.16	(0.82, 1.63)	0.32	1.00	(0.99, 1.01)
Gleason 2-7(3+4) cancers, No.	207	87		124		222		91				
Age-adjusted OR (95% CI)	1.00	1.08	(0.77, 1.52)	1.06	(0.78, 1.43)	1.10	(0.85, 1.42)	1.09	(0.78, 1.52)	0.51	1.00	(0.99, 1.01)
Multivariable-adjusted OR (95% CI)*	1.00	1.13	(0.80, 1.59)	1.09	(0.80, 1.49)	1.15	(0.88, 1.51)	1.19	(0.83, 1.71)	0.27	1.00	(0.99, 1.01)
Gleason 7(4+3)-10 cancers, No.	39	28		30		51		18				
Age-adjusted OR (95% CI)	1.00	1.79	(1.04, 3.08)	1.28	(0.76, 2.15)	1.26	(0.80, 1.98)	1.11	(0.61, 2.04)	0.75	1.00	(0.98, 1.01)
Multivariable-adjusted OR (95% CI)*	1.00	1.72	(1.00, 2.97)	1.30	(0.77, 2.19)	1.25	(0.78, 1.99)	1.04	(0.55, 1.96)	0.81	1.00	(0.98, 1.01)
Local stage cancers, No.	200	95		123		222		85				
Age-adjusted OR (95% CI)	1.00	1.22	(0.87, 1.71)	1.07	(0.79, 1.45)	1.11	(0.85, 1.43)	1.05	(0.74, 1.47)	0.70	1.00	(0.99, 1.01)
Multivariable-adjusted OR (95% CI)*	1.00	1.25	(0.89, 1.76)	1.07	(0.79, 1.47)	1.13	(0.86, 1.49)	1.12	(0.78, 1.61)	0.50	1.00	(0.99, 1.01)
Regional/distant stage cancers, No.	46	18		31		51		23				
Age-adjusted OR (95% CI)	1.00	1.02	(0.56, 1.85)	1.24	(0.75, 2.06)	1.20	(0.77, 1.86)	1.30	(0.75, 2.26)	0.27	1.01	(0.99, 1.02)
Multivariable-adjusted OR (95% CI)*	1.00	1.01	(0.55, 1.83)	1.27	(0.77, 2.11)	1.23	(0.78, 1.93)	1.33	(0.74, 2.38)	0.24	1.01	(0.99, 1.02)

*Multivariable models were adjusted for age, race, first-degree family history of prostate cancer, smoking status, and history of prostate cancer screening.