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Coffee and tea consumption in relation to prostate cancer prognosis

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Abstract

Background—Bioactive compounds found in coffee and tea may delay the progression of prostate cancer.

Methods—We investigated associations of pre-diagnostic coffee and tea consumption with risk of prostate cancer recurrence/progression. Study participants were men diagnosed with prostate cancer in 2002–2005 in King County, Washington, USA. We assessed the usual pattern of coffee and tea consumption two years before diagnosis date. Prostate cancer outcome events were identified using a detailed follow-up survey. Multivariable Cox proportional hazards regression models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs).

Results—The analysis of coffee intake in relation to prostate cancer recurrence/progression included 630 patients with a median follow-up of 6.4 years, during which 140 prostate cancer recurrence/progression events were recorded. Approximately 61% of patients consumed at least one cup of coffee per day. Coffee consumption was associated with a reduced risk of prostate cancer recurrence/progression; the adjusted HR for 4 cups/day versus 1 cup/week was 0.41 (95% CI: 0.20, 0.81; P for trend = 0.01). Approximately 14% of patients consumed one or more cups of tea per day, and tea consumption was unrelated to prostate cancer recurrence/progression.

Conclusion—Results indicate that pre-diagnostic coffee consumption is associated with a lower risk of prostate cancer recurrence/progression. This finding will require replication in larger studies.

Introduction

A recent study showed evidence of an inverse association between coffee consumption and the progression of prostate cancer (1). That prospective study evaluated the risk of lethal prostate cancer, which was defined as metastatic disease or prostate cancer-specific mortality. The authors showed a substantially lower risk for men consuming six or more cups of coffee per day. The inverse association was attributed to several potentially chemopreventive compounds in coffee, including cafestol, kahweol, chlorogenic acid, and caffeic acid (2–6). Very few other studies have comprehensively investigated this relationship (1).

Besides coffee, there has been considerable interest in the potential anti-carcinogenic effect of tea against prostate cancer. Potential beneficial compounds in tea are monomeric polyphenols such as catechins and flavonols (7, 8). Several lines of experimental evidence suggested that tea polyphenols delay the development and progression of prostate cancer (9–11). As far as we know, the association of tea consumption with prostate cancer outcomes has not been investigated.

In the present analysis, we investigated pre-diagnostic coffee and tea consumption in relation to prostate cancer outcomes. The study was conducted among a population-based cohort of prostate cancer patients from King County, Washington, USA, who were followed prospectively for more than five years for the development of adverse prostate cancer outcomes.

Materials and Methods

Study participants and data collection

The study population consisted of prostate cancer patients ($n = 1,001$) from a population-based, case-control study of prostate cancer. These men were aged 35 to 74 years at diagnosis, which occurred from January 1, 2002, through December 31, 2005 (12). Study participants 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 as well as primary therapy for prostate cancer. 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

Usual dietary intake was derived from a validated food frequency questionnaire (FFQ) (13), that queried dietary intake two years prior to prostate cancer diagnosis date. With regard to tea and coffee consumption, frequency of intake was assessed by using nine standard response 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 participants (90%). Men who had missing data on consumption of coffee ($n = 3$) or tea ($n = 5$) were excluded from the respective analyses. The median time between prostate cancer diagnosis and completion of the FFQ was 8.3 months. Participants 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 prostate cancer screening history.

Prostate cancer outcomes

Prostate cancer recurrence/progression events were ascertained using a follow-up survey in 2011 that included questions on a physician's diagnosis of prostate cancer recurrence/progression, use of secondary therapies, PSA test results, or a positive biopsy, bone scan, CT, or MRI showing evidence of recurrent/progressing prostate cancer. All patients who were alive at the time of the follow-up survey, consented to future contact, and were diagnosed with non-metastatic disease were included in the analysis of prostate cancer recurrence/progression ($n = 900$). Of these men, 661 completed the follow-up survey (73%). Response to the follow-up survey was unrelated to clinical parameters including Gleason grade, disease stage, and PSA level at diagnosis. Prostate cancer recurrence/progression defined on the basis of follow-up PSA tests varied by primary treatment: a post-treatment PSA value of 0.2 ng/mL or greater in men who underwent radical prostatectomy; nadir PSA

level + 2 ng/mL (Phoenix criteria) (15), for men treated with radiation therapy; or a rising PSA in men treated with primary androgen deprivation therapy (ADT). We coded a patient as being on active surveillance if they did not undergo any form of primary therapy within one year after diagnosis date. If the patient was treated later (12+ months post diagnosis date), but we did not find evidence from a repeat biopsy showing Gleason score upgrading or a physician's statement that the patient had progressed based on PSA testing, we did not consider them as having a progression event. The date of recurrence/progression was determined for patients who died from prostate cancer prior to the follow-up survey using multiple imputations ($n = 23$) (16), and for one case from medical records. The censoring date for non-events was the date that the follow-up questionnaire was returned and the date of last follow-up for recurrence/progression events was July 24, 2011 (the date the last questionnaire was returned). Data on prostate cancer recurrence/progression were available for 685 participants, and 630 and 629 of these men provided data on coffee and tea consumption, respectively.

Cause-specific deaths were ascertained from the SEER registry, which links quarterly with the Washington State vital statistics database and annually with the National Death Index. The completeness of survival follow-up through linkage with these registries is estimated to be close to 100%. Underlying cause of death obtained from these registries was verified by a review of death certificates, with over 99% agreement. The date of last follow-up for survival was July 1, 2012.

Statistical analyses

Coffee and tea consumption were tabulated according to a number of selected patient characteristics. Cox proportional hazards regression was used to obtain hazard ratios (HRs) and corresponding 95% confidence intervals (CIs) for the associations between coffee and tea consumption and risk of prostate cancer recurrence/progression. P values for linear trends were calculated by using median values within consumption categories. We modeled five categories of coffee consumption (1/week; 2–6/week; 1/day; 2–3/day; 4/day) and three categories of tea consumption (1/week; 2–6/week; 1/day) with the lowest category (1/week) as the referent group. Age-adjusted models and multivariable models adjusted for age at diagnosis (years), Gleason score (2–6, 7(3+4), 7(4+3), 8–10), stage at diagnosis (local, regional), diagnostic PSA level (<10, 10–19, ≥20), primary treatment approach (radical prostatectomy, radiation with or without androgen deprivation therapy (ADT), ADT only, no active treatment (active surveillance), other treatment), 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 diagnosis date (none, digital rectal examination only, PSA testing) were completed. The following factors were additionally assessed as potential confounders: education, lifetime alcohol consumption, recent exercise frequency, body mass index (BMI), regular aspirin use, regular non-aspirin NSAID use, history of diabetes mellitus, and consumption of tea (when analyzing coffee) or coffee (when analyzing tea). None of these was included in the final models, as they had little effect on the risk estimates or precision. The proportional hazards assumption was tested using the scaled Schoenfeld residuals (17). All tests were two-sided, with a P-value of less than 0.05 considered to be statistically significant. Analyses were performed using STATA software (release 12, STATA Corporation, College Station, TX).

Results

The analysis of prostate cancer recurrence/progression and coffee consumption included 630 patients. Table 1 shows the baseline characteristics of patients according to coffee consumption categories. Approximately 61% of patients consumed at least one cup of coffee

per day and 12% of patients were in the highest category of coffee consumption (≥ 4 cups/day). Coffee consumption was higher in Caucasians, men who were (former, current) smokers, and in those reporting alcohol consumption (all $P < 0.01$). Coffee consumption was lower in men who exercised frequently (≥ 4 times/week; $P = 0.02$) and in those reporting tea consumption (≥ 1 cup/day; $P < 0.01$). All other characteristics were unrelated to coffee consumption (Table 1). The distribution of coffee consumption was not different compared to the entire patient cohort ($n = 894$) (data not shown).

The analysis of prostate cancer recurrence/progression and tea consumption included 629 patients. Table 2 shows the baseline characteristics of patients according to tea consumption categories. Approximately 14% of patients consumed one or more cups of tea per day. Tea consumption was higher in men who reported having a college or graduate degree ($P < 0.01$) and lower in men who were (former, current) smokers ($P = 0.03$) and in those reporting coffee consumption ($P < 0.01$). All other selected characteristics were unrelated to tea consumption (Table 2). The distribution of tea consumption was not different compared to the entire patient cohort ($n = 892$) (data not shown).

For the analysis of coffee consumption, there were 140 prostate cancer recurrence/progression events (median: 6.4 years of follow-up; interquartile range (IQR): 5.2–7.6 years). These 140 events included 25 fatal prostate cancer cases, 10 cases who developed metastases, 77 cases who received secondary treatment for recurrent/progressing prostate cancer, and 28 cases with a rising PSA level (i.e. biochemical only). In the fully-adjusted model, coffee consumption was associated with a reduced risk of prostate cancer recurrence/progression with an HR of 0.41 (95% CI: 0.20, 0.81) for ≥ 4 cups/day versus < 1 cup/week (P for trend = 0.01) (Table 3). In an additional analysis we excluded those men for whom the only evidence of recurrence/progression was a rise in PSA ($n = 28$); the adjusted HR for ≥ 4 cups/day versus < 1 cup/week was 0.44 (95% CI: 0.21, 0.93; P for trend = 0.02) (data not shown).

For the analysis of tea consumption, there were 141 prostate cancer recurrence/progression events (Table 3). These events included 25 fatal prostate cancer cases, 11 cases who developed metastases, 77 cases who received secondary treatment for prostate cancer, and 28 cases with a rising PSA level (i.e. biochemical only). Tea consumption was not associated with prostate cancer recurrence/progression; the adjusted HR for ≥ 1 cup/day versus < 1 cup/week was 1.08 (95% CI: 0.65, 1.78; P for trend = 0.64). The results were unchanged after excluding 28 men for whom the only evidence of recurrence/progression was a rise in PSA (data not shown).

We also evaluated daily coffee consumption in relation to prostate cancer-specific mortality in all 894 patients with data on coffee intake. During a median follow-up time of 8.5 years (IQR: 7.3–9.5), 125 deaths were identified, including 38 prostate cancer-specific deaths. Daily coffee consumption (≥ 1 cup/day versus < 1 cup/day) was not associated with prostate cancer-specific mortality (adjusted HR = 0.77; 95% CI: 0.41, 1.46) or other-cause mortality (adjusted HR = 0.69; 95% CI: 0.45, 1.06). The point estimate for prostate cancer-specific mortality was similar when Cox regression was used instead of competing risk regression. We did not investigate the association of tea consumption with cause-specific mortality because there were too few events in the category of ≥ 1 cup of tea per day.

Discussion

In this US population-based cohort of prostate cancer survivors, pre-diagnostic coffee consumption was associated with a reduced risk of prostate cancer recurrence/progression.

Patients in the highest compared to the lowest category of coffee consumption (4 cups/day versus 1 cup/week) had a 59 percent reduction in risk. No association was observed for tea.

Our study included detailed data on prostate cancer recurrence and progression. A composite definition of recurrence/progression was developed that included patients who died of prostate cancer, developed metastasis, received secondary treatment, or had a rising PSA. Because pretreatment PSA levels may be a poor predictor of prostate cancer specific-survival (18), we repeated the analysis after excluding events defined as a rise in PSA only, and found that the association between coffee and a reduced risk of prostate cancer recurrence/progression remained statistically significant. The inverse association is consistent with the results from a recent prospective analysis completed in the Health Professionals Follow-up Study cohort (1). The authors evaluated both incident prostate cancer (n = 5,035 cases) and lethal prostate cancer (defined as prostate cancer-specific death or metastatic disease; n = 642 cases). The study showed that men in the highest compared to lowest category of coffee consumption (6 cups/day versus non-consumers) had a modestly decreased risk of developing overall prostate cancer (adjusted HR = 0.82; 95% CI: 0.68, 0.98) and the association was much more pronounced for men who subsequently developed metastatic/lethal prostate cancer (adjusted HR = 0.40; 95% CI: 0.22, 0.75). The authors additionally found an inverse association with advanced prostate cancer at diagnosis or follow-up (defined as stage T3b, T4, N1, or M1 at diagnosis or metastatic/lethal prostate cancer) and a non-significant decrease in risk of high-grade prostate cancer (Gleason 8–10) at diagnosis. The few additional studies that have investigated the association of coffee consumption with the risk of high stage or high grade prostate cancer showed mixed results (19–21), and further studies of this issue are needed. In our patient cohort, however, we found no evidence that coffee consumption was related to clinical parameters such as disease stage, Gleason grade, or PSA level at diagnosis.

In an additional analysis we evaluated daily coffee consumption (1 cup/day versus <1 cup/day) in relation to prostate cancer-specific mortality and observed a non-significant 23 percent lower risk. This analysis included only 38 events and thus had insufficient power to investigate the potential coffee- prostate cancer-specific mortality relationship. The limited number of events after a follow-up of 8.5 years is expected given the typically long time from prostate cancer progression to a fatal outcome (22). Our study also showed that coffee consumption was associated with a 31 percent lower risk of death from other causes. An inverse association between coffee drinking and mortality is supported by the results from a recent large-scale, US-based, prospective study by Freedman et al. (23). This study found that coffee consumption was associated with a significantly decreased risk of overall mortality and death due to several causes. The potential association between coffee consumption and prostate cancer-specific mortality was not examined in the Freedman et al. study (23). Additional large prospective studies of the relationship between coffee and prostate cancer outcomes, including recurrence/progression and prostate cancer-specific mortality, are urgently needed to confirm whether coffee intake is beneficial for secondary prevention.

Several phytochemicals in coffee may be responsible for the associations observed in our study. There is evidence from laboratory studies that caffeine delays metastasis (6), and possible anti-cancer effects of caffeine include anti-proliferative and pro-apoptotic activities (24, 25). Total caffeine intake has been associated with a reduced risk of several cancer types, including basal cell carcinoma (26), glioma (27), and ovarian cancer (28). Wilson et al. however, showed that the inverse association with lethal prostate cancer was similar for regular and decaffeinated coffee, suggesting that the association may be due to non-caffeine components of coffee (1). Other potentially bioactive compounds in coffee are the diterpenes cafestol and kahweol, and biological properties of these compounds may be

related to the induction of phase II enzymes, which may inhibit carcinogenesis (29). Coffee is also a major source of chlorogenic acid; several biological activities have been associated with the consumption of these phenolic compounds, including anti-inflammatory and antioxidant effects and modulation of glucose metabolism (3, 30). There is also evidence that caffeic acid and chlorogenic acid in coffee can inhibit DNA methylation, a biochemical process involved in the development and progression of many cancer subtypes (31).

There has also been considerable interest in the possible chemopreventative effects of tea on prostate cancer. A number of *in vivo* and *in vitro* laboratory studies showed that tea extracts inhibit prostate cancer growth (8–11). There has been one small proof-of-principle clinical trial that showed that green tea catechins may delay the progression of prostate intra-epithelial neoplasia to prostate cancer (32). This interesting finding, however, has not been reproduced and needs further corroboration (9). To our knowledge, our study is the first to investigate the potential association between tea consumption and prostate cancer outcomes, and we found no evidence that tea drinking alters progression/recurrence-free survival. It is important to note, however, that few patients in our cohort were regular tea drinkers and the highest category of tea consumption was defined as one or more cups per day. The association of tea drinking with prostate cancer recurrence or progression should be investigated in future studies that have access to populations with higher levels of tea consumption.

Our study has several strengths, including its population-based prospective design and extended patient follow-up for assessing both prostate cancer recurrence/progression and cause-specific mortality. We also had detailed information on lifestyle, medical history, and clinical and pathological parameters and were, therefore, able to adjust for potential confounding. The study has some limitations as well. Repeated exposure measurements during the follow-up period were not available. It may be that changes in the consumption of tea or coffee after diagnosis resulted in risk attenuation of our estimates. Although we had sufficient power to study associations between coffee consumption and prostate cancer recurrence/progression, we had limited power to assess the risk of prostate cancer-specific mortality. The sample size also did not allow us to study associations among men with a specific primary treatment (e.g., radical prostatectomy, radiation therapy). We cannot rule out the possibility of survivor bias. We, however, investigated coffee and tea intakes in men who were not included in the recurrence/progression analysis and found no evidence that intake patterns were different. An additional limitation is the lack of data on whether men consumed caffeinated or decaffeinated coffee. We also had no data on how coffee was prepared (espresso, boiled, filtered), and the bioactive chemicals of coffee may differ according to the method of preparation (4).

In conclusion, our study demonstrates that prostate cancer patients who consumed four or more cups of coffee per day, before prostate cancer diagnosis, had a substantial increase in prostate cancer recurrence/progression-free survival. The association needs to be confirmed in future prostate cancer studies that include larger cohorts with a wide range of coffee consumption levels and extended follow-up for assessment of prostate cancer outcomes.

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References

1. 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]
2. Arab L. Epidemiologic evidence on coffee and cancer. *Nutr Cancer.* 2010; 62:271–83. [PubMed: 20358464]
3. 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]
4. Higdon JV, Frei B. Coffee and health: a review of recent human research. *Crit Rev Food Sci Nutr.* 2006; 46:101–23. [PubMed: 16507475]
5. Nkondjock A. Coffee consumption and the risk of cancer: an overview. *Cancer Lett.* 2009; 277:121–5. [PubMed: 18834663]
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. 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]
8. 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]
9. 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]
10. 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]
11. Johnson JJ, Bailey HH, Mukhtar H. Green tea polyphenols for prostate cancer chemoprevention: a translational perspective. *Phytomedicine.* 2010; 17:3–13. [PubMed: 19959000]
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. Roach M 3rd, Hanks G, Thames H Jr, et al. Defining biochemical failure following radiotherapy with or without hormonal therapy in men with clinically localized prostate cancer: recommendations of the RTOG-ASTRO Phoenix Consensus Conference. *Int J Radiat Oncol Biol Phys.* 2006; 65:965–74. [PubMed: 16798415]
16. Wright JL, Kwon EM, Lin DW, et al. CYP17 polymorphisms and prostate cancer outcomes. *Prostate.* 2010; 70:1094–101. [PubMed: 20503394]
17. Grambsch PM, Therneau TM. Proportional hazards tests and diagnostics based on weighted residuals. *Biometrika.* 1994; 81:515–26.
18. Denham JW, Steigler A, Wilcox C, et al. Why are pretreatment prostate-specific antigen levels and biochemical recurrence poor predictors of prostate cancer survival? *Cancer.* 2009; 115:4477–87. [PubMed: 19691097]
19. Arab L, Su LJ, Steck SE, et al. Coffee consumption and prostate cancer aggressiveness among African and Caucasian Americans in a population-based study. *Nutr Cancer.* 2012; 64:637–42. [PubMed: 22564042]
20. Geybels MS, Neuhouwer ML, Stanford JL. Associations of tea and coffee consumption with prostate cancer risk. *Cancer causes & control : CCC.* 2012 (accepted).
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. Klotz L. Active surveillance: the canadian experience with an “inclusive approach”. *J Natl Cancer Inst Monogr*. 2012; 2012:234–41. [PubMed: 23271779]
23. Freedman ND, Park Y, Abnet CC, Hollenbeck AR, Sinha R. Association of coffee drinking with total and cause-specific mortality. *N Engl J Med*. 2012; 366:1891–904. [PubMed: 22591295]
24. Hashimoto T, He Z, Ma WY, et al. Caffeine inhibits cell proliferation by G0/G1 phase arrest in JB6 cells. *Cancer Res*. 2004; 64:3344–9. [PubMed: 15126379]
25. He Z, Ma WY, Hashimoto T, Bode AM, Yang CS, Dong Z. Induction of apoptosis by caffeine is mediated by the p53, Bax, and caspase 3 pathways. *Cancer Res*. 2003; 63:4396–401. [PubMed: 12907610]
26. Song F, Qureshi AA, Han J. Increased caffeine intake is associated with reduced risk of basal cell carcinoma of the skin. *Cancer Res*. 2012; 72:3282–9. [PubMed: 22752299]
27. Holick CN, Smith SG, Giovannucci E, Michaud DS. Coffee, tea, caffeine intake, and risk of adult glioma in three prospective cohort studies. *Cancer Epidemiol Biomarkers Prev*. 2010; 19:39–47. [PubMed: 20056621]
28. Tworoger SS, Gertig DM, Gates MA, Hecht JL, Hankinson SE. Caffeine, alcohol, smoking, and the risk of incident epithelial ovarian cancer. *Cancer*. 2008; 112:1169–77. [PubMed: 18213613]
29. Huber WW, Rossmanith W, Grusch M, et al. Effects of coffee and its chemopreventive components kahweol and cafestol on cytochrome P450 and sulfotransferase in rat liver. *Food Chem Toxicol*. 2008; 46:1230–8. [PubMed: 17983700]
30. Tunncliffe JM, Shearer J. Coffee, glucose homeostasis, and insulin resistance: physiological mechanisms and mediators. *Appl Physiol Nutr Metab*. 2008; 33:1290–300. [PubMed: 19088791]
31. Lee WJ, Zhu BT. Inhibition of DNA methylation by caffeic acid and chlorogenic acid, two common catechol-containing coffee polyphenols. *Carcinogenesis*. 2006; 27:269–77. [PubMed: 16081510]
32. 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]

Selected characteristics of study participants with data on prostate cancer recurrence/progression according to categories of coffee consumption, King County, Washington, 2002–2005

Table 1

Variables	Coffee consumption categories (cups/unit of time)									
	1/week		2–6/week		1/day		2–3/day		4/day	
	n = 167	Mean (SD)	n = 79	Mean (SD)	n = 113	Mean (SD)	n = 198	Mean (SD)	n = 73	Mean (SD)
Age, y		61.9 (7.9)		60.8 (7.7)		61.2 (7.9)		62.9 (7.1)		60.9 (8.4)
Caucasian	80.8		91.1		90.3		97.0		97.3	
First-degree family history of prostate cancer	26.3		17.7		24.8		22.2		20.5	
College or graduate degree	66.5		63.3		62.8		66.2		49.3	
Never smoker	67.7		49.4		45.1		33.8		21.9	
High (15 drinks/week) alcohol consumption	10.8		8.9		27.4		14.1		31.5	
High (4 times/week) recent exercise frequency	36.5		30.4		27.4		23.7		17.8	
Body mass index, kg/m ²		27.1 (4.4)		27.3 (4.3)		26.6 (3.8)		27.4 (3.7)		27.3 (3.5)
Aspirin use ^a	49.7		51.9		50.4		52.0		50.7	
Non-aspirin NSAID use ^a	21.6		21.5		23.0		23.7		24.7	
Diabetes mellitus	8.4		7.6		8.8		5.6		12.3	
History of PSA testing ^b	75.4		74.7		70.8		77.3		69.9	
Prostate cancer screening as a result of symptoms	22.2		21.5		26.6		22.7		16.4	
1 cup of tea per day	23.4		12.7		14.3		10.2		4.2	
Gleason score										
2–6	51.5		48.7		47.8		54.5		57.5	
7 (3+4)	33.5		28.2		37.2		27.8		28.8	
7 (4+3)	6.0		11.5		6.2		9.6		2.7	
8–10	9.0		11.5		8.8		8.1		11.0	
Stage of cancer										
Local	80.2		84.8		82.3		80.3		83.6	
Regional	19.8		15.2		17.7		19.7		16.4	
PSA value at diagnosis		6.4 (4.9–9.0) ^c		5.6 (4.3–7.7) ^c		5.4 (4.5–8.3) ^c		5.1 (4.7–9.6) ^c		5.3 (4.2–8.0) ^c

Variables	Coffee consumption categories (cups/unit of time)									
	1/week		2-6/week		1/day		2-3/day		4/day	
	n = 167		n = 79		n = 113		n = 198		n = 73	
	%	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%	Mean (SD)
Primary treatment for prostate cancer										
Radical prostatectomy	49.1		50.6		52.2		52.5		63.0	
Radiation with or without ADT	37.7		40.5		36.3		34.8		21.9	
ADT only	3.0		2.5		2.7		1.0		1.4	
Active surveillance	9.6		6.3		8.8		11.1		12.3	
Other	0.6		0.0		0.0		0.5		1.4	

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

^aUse at least once a week for 3 months or longer

^bPSA testing within the 5-year period before prostate cancer diagnosis

^cMedian (p25–p75)

Table 2

Selected characteristics of participants with data on prostate cancer recurrence/progression according to categories of tea consumption, King County, Washington, 2002–2005

Variables	Tea consumption categories (cups/unit of time)					
	1/week		2–6/week		1/day	
	n = 446	n = 95	n = 88			
Age, y	Mean (SD)	Mean (SD)	Mean (SD)	%	%	Mean (SD)
	61.8 (7.9)	62.0 (6.4)	61.7 (7.8)			
Caucasian	91.5	87.4	89.8			
First-degree family history of prostate cancer	22.0	29.5	22.7			
College or graduate degree	60.8	62.1	77.3			
Never smoker	43.0	46.3	58.0			
High (15 drinks/week) alcohol consumption	18.6	15.8	11.4			
High (4 times/week) recent exercise frequency	26.9	30.5	29.5			
Body mass index, kg/m ²	27.2 (3.8)	28.0 (4.6)	25.9 (3.9)			
Aspirin use ^a	50.2	52.6	52.3			
Non-aspirin NSAID use ^a	21.3	24.5	28.4			
Diabetes mellitus	7.8	8.4	5.7			
History of PSA testing ^b	74.2	73.7	75.0			
Prostate cancer screening as a result of symptoms	22.7	16.8	27.3			
1 cup of coffee per day	66.0	51.6	44.3			
Gleason score						
2–6	49.7	53.7	63.6			
7 (3+4)	33.3	26.3	23.9			
7 (4+3)	8.1	10.5	1.1			
8–10	9.0	9.5	11.4			
Stage of cancer						
Local	80.7	78.9	87.5			
Regional	19.3	21.1	12.5			
PSA value at diagnosis	6.1 (4.6–9.2) ^c	5.8 (4.3–7.3) ^c	5.8 (4.5–7.8) ^c			

Variables	Tea consumption categories (cups/unit of time)					
	1/week		2–6/week		1/day	
	n = 446	n = 88	n = 95	n = 88	n = 95	n = 88
	%	Mean (SD)	%	Mean (SD)	%	Mean (SD)
Primary treatment for prostate cancer						
Radical prostatectomy	54.9		51.6		43.2	
Radiation with or without ADT	34.5		36.8		34.1	
ADT only	1.6		1.1		5.7	
Active surveillance	8.5		10.5		15.9	
Other	0.4		0.0		1.1	

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

^aUse at least once a week for 3 months or longer
^bPSA testing within the 5-year period before prostate cancer diagnosis
^cMedian (p25–p75)

Table 3

Hazard ratios (HR) and 95% confidence intervals (CI) for prostate cancer recurrence/progression according to categories of coffee and tea consumption, King County, Washington, 2002–2011

		Prostate cancer recurrence/progression				
	Person-years	No. events	HR ^a	95% CI ^d	HR ^b	95% CI ^d
Coffee consumption						
1 cup/week	1,005	47	1.00		1.00	
2–6 cups/week	469	18	0.84	(0.49, 1.44)	0.78	(0.45, 1.37)
1 cup/day	704	17	0.52	(0.30, 0.90)	0.48	(0.27, 0.84)
2–3 cups/day	1,187	46	0.82	(0.55, 1.23)	0.75	(0.49, 1.16)
4 cups/day	477	12	0.54	(0.29, 1.02)	0.43	(0.22, 0.85)
P trend			0.07		0.02	
Tea consumption						
1 cup/week	2,718	98	1.00		1.00	
2–6 cups/week	566	24	1.18	(0.75, 1.84)	1.18	(0.75, 1.85)
1 cup/day	544	19	0.98	(0.60, 1.60)	1.04	(0.63, 1.70)
P trend			0.87		0.70	

^a Age-adjusted model (years)

^b Multivariable model adjusted for age at diagnosis (years), race (Caucasian, African-American), first-degree family history of prostate cancer (no, yes), smoking status (never, former, current), and history of prostate cancer screening (none, digital rectal exam only, PSA testing)

^c Multivariable model adjusted for age at diagnosis (years), Gleason score (2–6, 7 (3+4), 7 (4+3), 8–10), stage at diagnosis (local, regional), diagnostic PSA level (<10, 10–19.9, ≥20), primary treatment approach (radical prostatectomy, radiation with or without androgen deprivation therapy (ADT), ADT only, no active treatment (active surveillance), other treatment), race (Caucasian, African-American), first-degree family history of prostate cancer (no, yes), smoking status (never, former, current), and history of prostate cancer screening (none, digital rectal exam only, PSA testing)