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Coffee and tea consumption in relation to risk of rheumatoid arthritis in the Women's Health Initiative Observational Cohort

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Abstract

Objectives—To evaluate whether tea or coffee consumption is associated with an increased risk of older-onset rheumatoid arthritis (RA) using the Women's Health Initiative Observational Study (WHI-OS).

Methods—WHI-OS is a longitudinal prospective cohort study conducted from 1993 to 1998. 76,853 women completed a self-administered questionnaire about their daily consumption of tea and coffee. 185 self-reported and validated incident cases of RA were observed after three years of observation. Multivariable Cox proportional hazard models were performed to assess the relationship between consumption habits and disease incidence. Trend tests were calculated using categorical variables modeled as a continuous variable without collapsing.

Results—There was no increase in the hazard ratio (HR) for incident RA in those participants who drank coffee compared to those who did not. The amount of coffee consumed and the method of preparation (caffeinated/decaffeinated; filtered/unfiltered) also did not alter the risk of incident RA. There was a positive association of incident RA and caffeinated tea consumption in the trend test (p=0.03). When assessing any caffeinated tea consumption versus no tea consumption, the HR for incident RA was 1.40 (CI 1.01–1.93, p=0.04).

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Conclusions—In a large prospective cohort of older women, there was no association between coffee consumption and incident RA. A small association between daily caffeinated, non-herbal tea consumption and incident RA was found.

INTRODUCTION

Multiple known risk factors for RA include female gender, smoking and having a family history of autoimmune disease. It is generally agreed upon that RA has a complex etiology involving interplay of variable environmental exposures and a multitude of genetic risk factors [1]. The body of knowledge on environmental triggers remains limited. While cigarette smoking is an established risk factor for several autoimmune diseases, including RA [2–4], other environmental triggers have been implicated with varying degrees of consistency [5, 6]. Despite ever increasing knowledge about the pathogenesis of RA, little remains known about specific risk factors for incident RA in older populations.

Many polyphenol containing teas (including green tea, black tea, and oolong tea) are considered potential antioxidants [7], thus possibly reducing the risk of RA; however, findings regarding tea consumption and the risk of RA have been inconsistent. Mikuls et al [8] found that subjects consuming more than 3 cups of tea per day were protected (RR 0.39, 95% CI 0.16–0.97). Kim et al found that polyphenolic compound from green tea significantly reduced adjuvant arthritis in rats [9]. Also, they had lower concentration of proinflammatory cytokine IL-17 and higher concentration of immunoregulatory cytokine IL-10. Several other studies however have suggested no relationship [10–13] between tea consumption and RA.

Several investigations have been undertaken to determine if coffee consumption is a possible risk determinant for RA [8, 10, 11, 14–16]. Heliovaara et al showed direct proportional increase in rheumatoid factor (RF) titer in relation to number of cups of coffee consumption. The significance of association however was lost when smoking was entered into the model [14]. Indeed, increased coffee consumption has been associated with cigarette smoking in both males and females [17]. To that end, most other studies investigating the association between coffee and incident RA account for this confounder, but with variable results. Two studies have shown an increased risk of RA associated with consumption of decaffeinated coffee, but no such association with caffeinated coffee intake [8, 10]. In contrast, two other studies [11, 16], including the largest prospective study to date [11], found no association between caffeinated coffee or decaffeinated coffee and the risk of RA. One variable missing from all of these studies has been any assessment on the method of coffee preparation.

If coffee consumption does potentially influence the risk of developing RA, it would be important to determine if the effect is related to by-products of coffee preparation. Diterpene cafestol, which is found in relatively high concentrations in unfiltered coffee brews (eg. espresso, boiled, percolated, and French press), is associated with high serum cholesterol levels [18, 19] which in turn have been shown to correlate with RF positivity [14] and risk of RA [20]. Additionally, solvents used to extract caffeine from coffee beans may increase the risk for RA. While solvent residues are normally reduced to trace levels by steaming the coffee beans, it is possible that chronic ingestion of small amounts could have a negative

biologic effect on humans. Many of these solvents have been implicated in a number of connective tissue diseases, including scleroderma, undifferentiated connective tissue disease, lupus, and RA [21]. The aim of this study is to examine the potential relationships between coffee and tea consumption and the risk of incident RA in a population of post-menopausal women.

MATERIALS AND METHODS

Study Population

The Women's Health Initiative Observational Study (WHI-OS) is a longitudinal cohort study of 93,676 postmenopausal women aged 50–79 years enrolled from 40 clinics throughout the US from 1993 to 1998. In this prospective study, the participants completed clinic visits at baseline, then were contacted yearly to update their health status, and completed another in-person clinic visit at year 3. Questionnaires collected data on medical history, medication use, and a variety of lifestyle behaviors.

Identification of RA

A previous study of the WHI determined that self-report was a nonspecific indicator of RA diagnosis compared with medical records review, but specificity was improved in those participants who were documented taking disease modifying anti rheumatic drugs (DMARDs); (specificity 95.4% for RA and 99.4% for SLE) [22]. Therefore, we classified prevalent cases based on self-reported RA and DMARD use (including prednisone) at baseline, and classified incident cases based on newly self-reported RA at year 1, 2, or 3 plus DMARD use at year 3. An analysis sample included baseline participants with complete data who were at risk of developing RA. We subsequently excluded prevalent cases (n=815; case classification described below) under the assumption they could not become new cases, as well as those with missing data on the food frequency questionnaire (FFQ), self-report of RA, or medication use at baseline or follow-up (n=16,823). A final sample size of 76,853 at risk participants was included in the study. The incident case group was 185 RA cases including 8 cases reporting both RA and SLE.

Exposure information

76,853 women completed a self-administered food frequency questionnaire (FFQ) at baseline providing information on daily consumption of tea and coffee. Questions related to tea consumption included the following: "Do you drink tea each day? (Do not include decaf or herbal tea.)" – Yes or no, and "How many cups of tea do you usually drink each day? (Do not include decaf or herbal tea.)" – Categorized to none, 1 cup, 2–3 cups, 4–5 cups, and 6 or more cups. Questions related to coffee use included the following: "Do you drink coffee each day" – yes or no; "How many cups of regular coffee (not decaf) do you usually drink each day?" – categorized to none, 1 cup, 2–3 cups, 4–5 cups, and 6 or more cups; "How many cups of decaf coffee do you usually drink each day?" – categorized similarly; and "How is the coffee usually made?" – categorized to drip, espresso, instant, boiled, percolated, and French press. For analysis, the methods of coffee preparation were divided into filtered (drip and instant) and unfiltered (espresso, boiled, percolated, and French press). For the purposes of analysis, the amount of daily beverage consumed (both tea and coffee)

was divided in categories of none, 1 cup, 2–3 cups, and 4 or more cups. Total caffeine intake was calculated by using FFQ-based intakes of soda (all types), coffee, and tea. Information on the caffeine content of other dietary sources, such as chocolate, or the caffeine content of medications was not available in the WHI. We divided the results into quintiles and categorized as <58 mg/day, 58–91 mg/day, 91–177 mg/day, 177–242 mg/day, and >242 mg/day.

Covariates

Covariate data were derived from the baseline questionnaire responses, including potential confounding risk factors that might be related to coffee and tea use and RA, i.e., age, race/ethnicity, education, pack-years of smoking, body mass index (BMI), marital status, and hormone therapy use.

Statistical analysis

Analyses were conducted using SAS, version 9.1. Following bivariate analyses of RA with covariate and coffee and tea consumption variables, multivariable analyses were limited to those with complete data on all variables. Cox regression was used to estimate hazard ratios (HRs) and 95% confidence intervals (95% CIs) in adjusted models. Trend test *p* values were obtained from models including the consumption variables as linear terms without collapsing categories, assuming a monotonic increase in effect across levels (statistical significance $p<0.05$).

RESULTS

Table 1 compares the baseline characteristics of the WHI women with incident RA and those in the study sample without RA. There were no significant differences in these baseline characteristics between the different groups except that women with RA had slightly higher BMI.

Coffee does not increase the risk of RA

In multivariate analyses, there was no observed increase in the hazard ratio for incident RA in those participants who drank coffee compared to those who did not (HR 1.09 (95% CI 0.77–1.54) p=0.63) (Table 2). Additionally, there was no statistically significant trend between the amount of coffee consumed and the risk of incident RA (p=0.16 for trend test). This was true regardless of whether the coffee was caffeinated or decaffeinated (Table 3). The method of coffee preparation (filtered versus unfiltered) did not show any significant trends (filtered p=0.08, unfiltered p=0.38 for trend test) (Table 3). There was also no observed associations between the calculated total caffeine intake and incident RA in this population (p=0.56 for trend test) (Table 3).

Tea consumption increases the risk for developing RA

Women with a history of non-herbal, non-decaffeinated tea consumption of any amount had an increased risk of developing RA during the first three years of observation in the WHI-OS (HR 1.40 (95%CI 1.01–1.93); p=0.04) (Table 2). Furthermore, there was a dose relationship observed with an increasing hazard ratio for increasing amounts of tea

consumed (p=0.03 for the trend test). Women who drank ≥4 cups of tea per day had a hazard ratio of 1.78 (95%CI 0.83–3.82) for the development of RA compared to those who drank none (Table 3).

DISCUSSION

Our analysis does not show any association between intake of coffee and RA in a population of post-menopausal women in the US. The lack of association between coffee consumption and the risk of RA is in contrast with some large epidemiologic studies [8, 10], but in agreement with others [11, 16]. Our analysis however goes further to suggest that regardless of the method of coffee preparation and amount of consumption, the risk remains unchanged.

Mikuls et al [8] found that subjects consuming 4 or more cups of decaffeinated coffee per day were at increased risk of RA (RR 2.58, 95% CI 1.63–4.06). Lee et al [13] published a meta-analysis and came with conclusion that total coffee intake is associated with RA but caffeinated coffee, decaffeinated coffee, or total caffeine intake is not associated. None of these studies however investigated the type preparation technique of the coffee by the study participants.

Based on our analysis, there is however a minimal increased risk of incident RA with caffeinated tea consumption in this group of post-menopausal woman. This observation appears to be in contrast to nearly all other studies, which have suggested no relationship [10–13] between tea consumption and RA. The mechanism by which non-herbal, non-decaffeinated tea could potentially confer a statistically significant, albeit minimal, increased risk for the development of RA is unclear, and is not explained by caffeine alone. Increased dietary intake of flavonoids, one of the components of tea, has been previously associated with an increased risk of RA in a large Finnish population [23], despite having known anti-inflammatory properties. However, as was noted in that study, there is no obvious mechanism for this observation, but one that merits further investigation.

Further clouding the interpretation of the WHI-OS data is the lack of clarity in defining the type of the tea consumed by this group of women. As per Tea Association of America, different types of tea consumed in America are as follows: Black Tea 84%, Green Tea 15%, and the small remaining amount were Oolong, White and Dark Tea. Since our observation indicated that increased tea consumption is associated with an increase in incident RA, but caffeine dose is not, it is possible that other chemical ingredients utilized in the growing or preparation of tea are responsible for this observation. Though all the tea types mentioned above are leaves of *Camellia sinensis*, they are processed differently, possibly leading to changes in chemical ingredients. Furthermore, brewing methods may create additional variability which we are unable to account for. Finally, a recent study has identified exposure to insecticides as a risk factor for RA in this same WHI-OS cohort [24], and thus we cannot rule out potential pesticide use in tea growing as a potential confounder.

While the questionnaire limited the quantification of tea consumption to that of non-herbal, non-decaffeinated preparations, other aspects such as types and preparations remain

unknown. Since many people believe that green tea is decaffeinated, it is unknown if the respondents in this study have correct knowledge whether the tea they are consuming is caffeinated, decaffeinated or herbal.

Additional limitations of this study include those inherit to a post-hoc analysis of an observational cohort which was not designed to look at arthritis. Methodology in the validation study which was utilized to identify incident RA cases had the potential to include some cases of unclassified or misclassified arthritis patients, as well as some cases where RA had merely been undiagnosed prior to study enrollment. Furthermore, many of the questionnaires used in the WHI-OS were non-validated instruments thus introducing a potential source of recall bias. That being said, this validation has been accepted as an acceptable, albeit not perfect, measure of RA in the WHI cohort.

The strengths of this study are large sample size, well standardized data, and validation of incident reported RA cases at the end of three-year period.

CONCLUSION

Our study showed that coffee consumption, irrespective of type or method of preparation, is not associated with RA in a post-menopausal population of women living in USA. The findings that non-herbal non-decaffeinated tea is associated with RA merits further studies. However, the risk is minimal. The findings could be confounded by other unknown environmental risk factors. It is not known if the study population was fully aware if the consumed tea was caffeinated or decaffeinated.

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Informed consent: All participants provided written informed consent. Institutional review board approval was obtained from each of the participating study centers and from the Fred Hutchinson Cancer Research Center, which currently serves as the IRB of record for the WHI. Federalwide Assurance number (FWA) is 00001920 and the registrations numbers for each of IRBs are:

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References

1. Gourley M, Miller FW. Mechanisms of Disease: environmental factors in the pathogenesis of rheumatic disease. Nat Clin Pract Rheumatol. 2007; 3:172–80. [PubMed: 17334340]
2. Costenbader KH, Karlson EW. Cigarette smoking and autoimmune disease: what can we learn from epidemiology? Lupus. 2006; 15:737–45. [PubMed: 17153844]

3. Harel-Meir M, Sherer Y, Shoenfeld Y. Tobacco smoking and autoimmune rheumatic diseases. Nat Clin Pract Rheumatol. 2007; 3:707–15. [PubMed: 18037930]
4. Klareskog L, Padyukov L, Alfredsson L. Smoking as a trigger for inflammatory rheumatic diseases. Curr Opin Rheumatol. 2007; 19:49–54. [PubMed: 17143096]
5. Alamanos Y, Drosos A. Epidemiology of adult rheumatoid arthritis. Autoimmun Rev. 2005; 4:130–6. [PubMed: 15823498]
6. Parks CG, Cooper GS. Occupational exposures and risk of systemic lupus erythematosus: a review of the evidence and exposure assessment methods in population-and clinic-based studies. Lupus. 2006; 15:728–36. [PubMed: 17153843]
7. Yang CS, Wang ZY. Tea and cancer. J Natl Cancer Inst. 1993; 85:1038–49. [PubMed: 8515490]
8. Mikuls TR, Cerhan JR, Criswell LA, Merlino L, Mudano AS, Burma M, et al. Coffee, tea, and caffeine consumption and risk of rheumatoid arthritis: Results from the Iowa Women's Health Study. Arthritis Rheum. 2002; 46:83–91. [PubMed: 11817612]
9. Kim HR, Rajaiah R, Wu Q-L, Satpute SR, Tan MT, Simon JE, et al. Green Tea Protects Rats against Autoimmune Arthritis by Modulating Disease-Related Immune Events. J Nutr. 2008; 138:2111–6. [PubMed: 18936206]
10. Formica MK, Palmer JR, Rosenberg L, McAlindon TE. Lifestyle factors associated with the development of rheumatoid arthritis (RA): results for the Black Women's Health Study (BWHS). Arthritis Rheum Suppl. 2001; 44:S376.
11. Karlson EW, Mandl LA, Aweh GN, Grodstein F. Coffee consumption and risk of rheumatoid arthritis. Arthritis Rheum. 2003; 48:3055–60. [PubMed: 14613266]
12. Pattison DJ, Symmons DPM, Lunt M, Welch A, Luben R, Bingham SA, et al. Dietary risk factors for the development of inflammatory polyarthritis: Evidence for a role of high level of red meat consumption. Arthritis Rheum. 2004; 50:3804–12. [PubMed: 15593211]
13. Lee YH, Bae S-C, Song GG. Coffee or tea consumption and the risk of rheumatoid arthritis: a meta-analysis. Clin Rheumatol. 2014; 33:1575–83. [PubMed: 24763752]
14. Heliövaara M, Aho K, Knekt P, Impivaara O, Reunanen A, Aromaa A. Coffee consumption, rheumatoid factor, and the risk of rheumatoid arthritis. Ann Rheum Dis. 2000; 59:631–5. [PubMed: 10913061]
15. Pedersen M, Jacobsen S, Garred P, Madsen HO, Klarlund M, Svejgaard A, et al. Strong combined gene–environment effects in anti–cyclic citrullinated peptide–positive rheumatoid arthritis: A nationwide case–control study in Denmark. Arthritis Rheum. 2007; 56:1446–53. [PubMed: 17469102]
16. Pedersen M, Stripp C, Klarlund M, Olsen SF, Tjønneland AM, Frisch M. Diet and risk of rheumatoid arthritis in a prospective cohort. J Rheumatol. 2005; 32:1249–52. [PubMed: 15996059]
17. Schreiber GB, Robins M, Maffeo CE, Masters MN, Bond AP, Morganstein D. Confounders contributing to the reported associations of coffee or caffeine with disease. Prev Med. 1988; 17:295–309. [PubMed: 3405986]
18. de Roos B, Katan MB. Possible mechanisms underlying the cholesterol-raising effect of the coffee diterpene cafestol. Curr Opin Lipidol. 1999; 10:41–6. [PubMed: 10095989]
19. Heckers H, Göbel U, Kleppel U. End of the coffee mystery: diterpene alcohols raise serum low-density lipoprotein cholesterol and triglyceride levels. J Intern Med. 1994; 235:192–3. [PubMed: 8308487]
20. Heliövaara M, Aho K, Knekt P, Reunamen A, Aromaa A. Serum Cholesterol And Risk Of Rheumatoid Arthritis In A Cohort Of 52 800 Men And Women. Br J Rheumatol. 1996; 35:255–7. [PubMed: 8620301]
21. Garabrant DH, Dumas C. Epidemiology of organic solvents and connective tissue disease. Arthritis Res. 2000; 2:5–15. [PubMed: 11094414]
22. Walitt BT, Constantinescu F, Katz JD, Weinstein A, Wang H, Hernandez RK, et al. Validation of self-report of rheumatoid arthritis and systemic lupus erythematosus: The Women's Health Initiative. J Rheumatol. 2008; 35:811–8. [PubMed: 18398940]
23. Knekt P, Kumpulainen J, Järvinen R, Rissanen H, Heliövaara M, Reunanen A, et al. Flavonoid intake and risk of chronic diseases. Am J Clin Nutr. 2002; 76:560–8. [PubMed: 12198000]

24. Parks CG, Walitt BT, Pettinger M, Chen JC, de Roos AJ, Hunt J, et al. Insecticide use and risk of rheumatoid arthritis and systemic lupus erythematosus in the Women's Health Initiative Observational Study. Arthritis Care Res (Hoboken). 2011; 63(2):184–94. [PubMed: 20740609]

Table 1

Baseline characteristics of incident RA cases in the WHI Observational Study

Characteristic	Non-RA cases N=76,668		RA cases N=185		
	N	%	N	%	p-value*
Age					0.20
50–59	24341	31.8	50	27.0	
60–69	34199	44.6	82	44.4	
70–79	18128	23.6	53	28.7	
Mean (SD)	63.6	(7.3)	64.6	(7.5)	0.05
Race/ethnicity					0.46
White	65191	85.0	154	83.2	
Black	5448	7.1	17	9.2	
Hispanic	2457	3.2	9	4.9	
American Indian	295	0.4	0	0.0	
Asian/Pacific Islander	2253	2.9	3	1.6	
Unknown	1024	1.3	2	1.1	
Body Mass Index					0.01
<25	31971	42.2	56	30.9	
25 – <30	25837	34.1	75	41.4	
>=30	17990	23.7	50	27.6	
Mean (SD)	27.1	(5.7)	27.7	(5.1)	0.10
Education					0.06
0–8 yrs	928	1.2	2	1.1	
Some high school	2291	3.0	10	5.4	
High school diploma/GED	12033	15.8	34	18.4	
School after high school	27474	36.1	75	40.5	
College degree or higher	33361	43.9	64	34.6	
Hormone therapy use					0.54

Characteristic	Non-RA cases N=76,668		RA cases N=185		p-value*
	N	%	N	%	
Never	30126	39.3	66	35.7	
Former	11317	14.8	27	14.6	
Current	35167	45.9	92	49.7	
Smoking status					0.60
Never	39110	51.6	89	48.0	
Former	32422	42.8	80	44.0	
Current	4277	5.6	13	7.1	
Alcohol use					0.90
Non-drinker	8141	10.7	20	11.0	
Past drinker	13544	17.8	37	20.3	
<1 drink per week	24337	31.9	54	29.7	
1-<7 drinks per week	20227	26.5	48	26.4	
7+ drinks per week	10039	13.2	23	12.6	
Marital status					0.87
Never married	3561	4.7	8	4.3	
Divorced	11551	15.1	26	14.1	
Widowed	12783	16.7	35	18.9	
Presently married	48451	63.5	116	62.7	

* based on a chi-square test of association for categorical variables and a t-test for continuous variables.

Table 2

Association of coffee and tea intake with RA

Nutrient	No. of Cases	Age adjusted HR	Age adjusted 95% CI	Multivariable adjusted* HR	Multivariable adjusted* 95% CI
Total coffee, cups/day					
None	49	1.00	Referent	1.00	Referent
Any	132	1.09	(0.78, 1.51)	1.09	(0.77, 1.54)
p value			0.62		0.63
Caffeinated coffee, cups/day					
None	79	1.00	Referent	1.00	Referent
Any	105	1.04	(0.77, 1.39)	1.04	(0.77, 1.41)
p value			0.81		0.80
Decaffeinated coffee, cups/day					
None	121	1.00	Referent	1.00	Referent
Any	60	1.03	(0.76, 1.41)	1.00	(0.72, 1.37)
p value			0.83		0.98
Unfiltered coffee†, cups/day					
None	167	1.00	Referent	1.00	Referent
Any	12	0.92	(0.51, 1.65)	0.76	(0.40, 1.45)
p value			0.77		0.41
Filtered coffee‡, cups/day					
None	61	1.00	Referent	1.00	Referent
Any	118	1.12	(0.82, 1.52)	1.18	(0.85, 1.64)
p value			0.48		0.32
Tea, cups/day					
None	125	1.00	Referent	1.00	Referent
Any	56	1.29	(0.94, 1.77)	1.40	(1.01, 1.93)
p value			0.11		0.04

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* Adjusted for age, race/ethnicity, marital status, smoking history, alcohol use, use of hormone therapy, education, and BMI

† Espresso + boiled + percolated + French press coffee preparation

‡ Drip + instant coffee preparation

Table 3

Association of the amount of coffee, tea and caffeine intake with RA

		Age adjusted		Multivariable adjusted*	
Nutrient	**Cases**	**HR**	**95% CI**	**HR**	**95% CI**
Total coffee, cups/day					
None	49	1.00	Referent	1.00	Referent
1	23	0.85	0.52, 1.39	0.89	0.53, 1.47
2–3	61	1.05	0.72, 1.53	1.08	0.73, 1.60
4 or more	48	1.32	0.89, 1.96	1.29	0.84, 1.98
p value for trend			0.10		0.16
Caffeinated coffee, cups/day					
None	79	1.00	Referent	1.00	Referent
1	28	0.88	0.57, 1.36	0.93	0.60, 1.44
2–3	54	1.02	0.72, 1.45	1.01	0.70, 1.45
4 or more	23	1.36	0.85, 2.16	1.37	0.84, 2.23
p value for trend			0.30		0.37
Decaffeinated coffee, cups/day					
None	121	1.00	Referent	1.00	Referent
1	22	0.80	0.51, 1.25	0.75	0.47, 1.21
2–3	28	1.14	0.76, 1.72	1.08	0.71, 1.66
4 or more	10	1.70	0.89, 3.24	1.76	0.92, 3.36
p value for trend			0.32		0.41
Unfiltered coffee†, cups/day					
None	167	1.00	Referent	1.00	Referent
1	2	0.70	0.17, 2.81	0.69	0.17, 2.80
2–3	5	0.78	0.32, 1.89	0.77	0.32, 1.89
3 or more	5	1.32	0.54, 3.21	0.80	0.32, 1.89
p value for trend			0.97		0.38
Filtered coffee‡, cups/day					
None	61	1.00	Referent	1.00	Referent
1	21	0.87	0.53, 1.44	0.95	0.57, 1.57

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		Age adjusted		Multivariable adjusted*	
Nutrient	**Cases**	**HR**	**95% CI**	**HR**	**95% CI**
2–3	56	1.11	0.77, 1.60	1.19	0.81, 1.74
3 or more	41	1.31	0.88, 1.95	1.38	0.91, 2.11
p value for trend			0.11		0.08
Tea, cups/day					
None	125	1.00	Referent	1.00	Referent
1	27	1.26	0.83, 1.91	1.33	0.87, 2.04
2–3	22	1.26	0.80, 1.98	1.38	0.87, 2.18
4 or more	7	1.59	0.74, 3.40	1.78	0.83, 3.82
p value for trend			0.10		0.03
Total Coffee and/or Tea, cups/day					
None	30	1.00	Referent	1.00	Referent
1	25	1.07	0.63, 1.83	1.19	0.68, 2.05
2–3	56	1.08	0.69, 1.68	1.19	0.74, 1.91
4 or more	72	1.44	0.94, 2.20	1.57	0.99, 2.49
p value for trend			0.02		0.01
Caffeine, mg/day					
≤57.53	23	1.00	Referent	1.00	Referent
57.53–90.56	38	1.64	0.98, 2.75	1.65	0.97, 2.82
90.56–177.65	40	1.74	1.04, 2.91	1.85	1.08, 3.15
177.65–242.40	32	1.41	0.83, 2.42	1.48	0.85, 2.60
≥242.96	40	1.79	1.07, 2.99	1.83	1.07, 3.15
p value for trend			0.51		0.56

*Adjusted for age, race/ethnicity, marital status, smoking history, alcohol use, use of hormone therapy, education, and BMI

†Espresso + boiled + percolated + French press coffee preparation

‡Drip + instant coffee preparation