

Tea and Coffee Consumption and Risk of Colon and Rectal Cancer in Middle-Aged Finnish Men

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Abstract: *The association between coffee and black tea consumption and the subsequent risk of colon and rectal cancer was investigated within a Finnish clinical trial cohort. One hundred eleven cases of colon cancer and 83 cases of rectal cancer were diagnosed over a median of 8.0 years of follow-up. Proportional hazards regression models were used to derive adjusted relative risks (RR) and 95% confidence intervals (CI) for the association between coffee and tea consumption and cancer incidence. After controlling for confounders, coffee was not significantly associated with colon or rectal cancer. A positive association was seen for increased consumption of tea drinking and colon cancer. Compared with persons who did not drink tea, those who consumed <1 cup/day had an RR of 1.40 (95% CI = 0.84–2.33) and those who consumed ≥1 cup/day had an RR of 2.09 (95% CI = 1.34–3.26, p for trend = 0.001). In contrast, tea consumption had little effect on rectal cancer incidence. This study does not support the hypothesis that coffee and tea protect against colorectal cancer risk. However, given the strength of the tea-colon cancer association and the significant gradient of risk we observed across level of intake, further epidemiologic research of this relationship in other populations seems warranted.*

Introduction

Coffee consumption in Finland is among the highest in the world (1). In a national report on health behavior, only 7% of a sample of Finnish men between the ages of 55 and 64 years reported that they did not drink coffee (2). The results of epidemiologic studies of coffee and large bowel cancer have not been consistent. Coffee drinking has been inversely (3–11) and positively (12–14) associated with colon or rectal cancer, and some studies have seen no relationship (15–18).

Until recently, tea has received substantially less attention than coffee as a potential risk factor for colorectal cancer. Tea drinkers are the minority in Finland: only 38% of the National Public Health Institute sample of older men reported that they drank tea (2). The reported results docu-

menting the relationship between tea and colorectal cancer are inconsistent and are complicated by evidence that green and black tea are chemically different and may not have similar associations with colon or other cancers. Green tea is produced from heated and dried tea leaves; black tea is produced from leaves by enzymatic oxidation, which also converts most of the flavonols and some other phenols to oxidized forms (19). A recent review by Kohlmeier and co-workers (20) concluded that the available evidence suggested a protective effect of green tea on the development of colon cancer. For black tea, these authors noted that some epidemiologic evidence indicated an increased risk of colon or rectal cancer. Some studies found no association, and several studies known to have collected information on tea consumption have yet to report their results.

We present here results for an analysis of the association between tea and coffee consumption and risk for colon and rectal cancers conducted within a trial-based cohort of Finnish, middle-aged, male smokers. In Finland, the majority of tea drinkers consume black tea; therefore, our results have implications primarily for black tea.

Subjects and Methods

Sample Population

The Alpha-Tocopherol Beta-Carotene Cancer Prevention Study (ATBC Study) was a large, randomized, double-blind, placebo-controlled prevention trial to determine whether daily supplementation with α -tocopherol, β -carotene, or both would reduce the incidence of lung or other cancers. The study was conducted in Finland between 1985 and 1993 as a joint project between the National Public Health Institute in Finland and the US National Cancer Institute. The overall design, rationale, and objectives of this study have been published (21). Briefly, 29,133 male smokers between the ages of 50 and 69 years were recruited from southwestern Finland between 1985 and 1988 and randomly assigned to one of four groups on the basis of a 2 × 2 factorial design.

Participants received 50 mg/day α -tocopherol (as *dl*- α -tocopheryl acetate), 20 mg/day β -carotene, α -tocopherol and β -carotene, or placebo. Follow-up continued for five to eight years during the trial, until death or 30 April 1993 (median follow-up 6.1 yr), and follow-up was continued after intervention. Men who were alcoholics, who had cirrhosis of the liver, severe angina with exertion, or chronic renal insufficiency, or who had been previously diagnosed with cancer were excluded from the study. Those taking supplements of vitamin E or A or β -carotene in excess of defined amounts or receiving anticoagulant therapy were also excluded.

Case Identification

For this analysis, cohort cases were defined as all incident cases of colon cancer (ICD-9 code 153; $n = 111$) and rectal cancer (ICD-9 code 154; $n = 83$) diagnosed between May 1985 and May 1995 and identified through the Finnish Cancer Registry (22). Medical records were reviewed at the central office by study physicians, including oncologists, to confirm diagnoses. Pathology and cytology specimens were reviewed, and a final histopathological diagnosis (World Health Organization classification) was assigned. For the few cases with more than one malignant colorectal tumor, the histology and diagnosis date for the earliest cancer diagnosed were used.

Data Collection

At baseline, study subjects completed a demographic and general medical history questionnaire and a food-frequency (use) questionnaire and provided a fasting blood sample. Of the total cohort, 27,111 men completed the dietary questionnaire. Dietary information was available for 106 colon and 79 rectal cancer cases. The ATBC Study food use questionnaire consisted of a modified diet history including portion size and frequency of consumption for 203 food items and 73 mixed dishes (23). Color pictures of foods and beverages, with examples of different portion sizes, assisted the participants to specify the portion size consumed. The coffee and tea questions in the diet history do not discriminate between caffeinated and decaffeinated beverages or between black and green tea; however, only black tea is pictured in the booklet. This instrument was intended to measure usual intake over the previous 12 months. Dietary intake was estimated through the use of food composition data available from the National Public Health Institute of Finland. The dietary instrument used to quantify measurement of tea and coffee consumption has been shown to be reproducible (intra-class correlation coefficients of 0.74 and 0.70, respectively) and valid (Pearson correlation coefficients of 0.66 and 0.76, respectively).

Statistical Analysis

Statistical analyses were performed using Statistical Analysis Systems software (23,24). Cox regression methods were used to estimate the association between tea and coffee

and incidence of colon and rectal cancers and between tea and coffee and colon cancer mortality (25). Our analysis used follow-up time as the underlying time metric and adjusted for age at randomization as a continuous variable. Biologic evidence (26) and previous reports from this population (27–29) and others (30,31) suggest that risk factors are not consistent between colon and rectal cancer. Thus, for analysis, models were developed for colon and rectal cancers as separate sites.

Tea and coffee were not highly correlated with total energy intake [Spearman $R = 0.05$ and 0.22 for logarithmically transformed continuous measures of tea and coffee with logarithmically transformed energy (kcal), respectively]; therefore, they were not energy adjusted for analysis. We confirmed the validity of this approach by testing logarithmically transformed continuous measures of tea and coffee, either energy adjusted or unadjusted, in multivariate models and concluded that the two results (energy adjusted vs. unadjusted) were not appreciably different from each other. For tea and coffee, three categories were constructed on the basis of the distribution of intake within the cohort, but with the categories defined relative to the 170-ml cups (tea) and 110-ml cups (coffee) pictured in the food use questionnaire. For tea, categories were nondrinkers, <1 cup/day, and ≥ 1 cup/day. For coffee, categories were 0–4 cups/day, 4–6 cups/day, and >6 cups/day. Other dietary variables of interest, including fat, protein, fiber, sugar, alcohol, calcium, folate, iron, fruit, vegetable, and red meat intake, were more highly correlated with energy intake and were logarithmically transformed to normalize their distributions and adjusted for total energy intake according to the residual method of Willett (32). Supplemental intake of vitamins and minerals of interest were added to dietary intake before transformation and energy adjustment. Dietary variables (other than tea and coffee) were entered into models as indicator variables defined by the second through fourth quartiles of intake among the cohort, with the lowest quartile as the referent group. To conduct a linear trend test across levels of coffee and tea consumption, variables were created using exposure scores based on the median values for each category of each beverage among the controls.

Variables included in the multivariate models were those that confounded the association between coffee or tea and colon or rectal cancer and those that were risk factors for cancer at either site. The association between tea and coffee consumption and colon and rectal cancers was evaluated within multivariate models that included age at randomization, intervention group, calcium intake, occupational physical activity, and body mass index (BMI) (kg/m^2). Serum cholesterol (mmol/l , logarithmically transformed) was also included in the rectal cancer models. Intervention group assignment was coded as three indicator variables for α -tocopherol, β -carotene, and α -tocopherol and β -carotene supplementation and with the placebo group as a reference. Occupational physical activity was coded as four indicator variables, with sedentary work as the referent category. Results are reported

as multivariate adjusted relative risks (RR) of colon or rectal cancer incidence with 95% confidence intervals (CI).

Effect modification was assessed by including factors and their cross-product terms in the model and through stratified analyses within low and high categories of factors (on the basis of median splits). For effect modification analyses, tea and coffee were coded as continuous variables, and the median value from each of the energy-adjusted quartiles was used as a continuous variable for other dietary factors. We checked the validity of the proportional hazards assumption by examining the cross-product term of follow-up time and the covariate of interest. There were no departures from proportional hazards assumption for any covariate included in the final models. The results were unchanged when persons with cancers diagnosed during the first two years of follow-up were eliminated from the analysis. Therefore, all persons with colon and rectal cancer diagnosed during the trial were included in the final models.

Results

One hundred eleven incident cases of colon cancer and 83 cases of rectal cancer were ascertained. Median follow-up

was 8.0 years. Overall, there were few differences between cancer cases and noncases (data not presented). Colon cancer cases were older, had a higher BMI, were less physically active at work, and tended to consume less calcium, less coffee, and more tea than noncases. Persons with rectal cancer were more likely to be older and to have a lower serum cholesterol level than noncases. Approximately 97% of this population drank coffee and 40% consumed tea. Of the study participants, 34% drank coffee and tea, 63% consumed only coffee, 2% consumed only tea, and only 1% did not drink coffee or tea. Coffee and tea intake were not highly correlated (Spearman $R = -0.29$ for logarithmically transformed continuous variables, $p = 0.001$). Because there were some notable differences between tea drinkers and nondrinkers in this population, selected baseline characteristics for participants (Table 1) are presented by tea drinking status (drinker vs. nondrinker). For the most part, tea drinking was associated with more healthful behaviors, including less smoking, lower serum cholesterol levels, a lower intake of red meat and fat, and a higher intake of fruits and vegetables. In contrast, calcium intake was lower and alcohol intake was higher among tea drinkers than among nondrinkers. In the four months before completion of the baseline medical his-

Table 1. Selected Baseline Characteristics for Tea Drinkers and Nondrinkers

Characteristic	Means \pm SD		P Value
	Nondrinkers (<i>n</i> = 17,364)	Tea drinkers (<i>n</i> = 9,744)	
Follow-up, yr	7.5 \pm 1.9	7.6 \pm 1.8	0.001
Age, yr	57.2 \pm 5.0	57.1 \pm 5.1	0.43
Body mass index, kg/m ²	26.3 \pm 3.8	26.2 \pm 3.7	0.23
Smoking, pack-yr	37.9 \pm 18.2	35.2 \pm 18.1	<0.001
Serum cholesterol, mmol/l	6.3 \pm 1.2	6.2 \pm 1.1	<0.001
Daily intake ^a			
Energy, kcal/day	2,788 \pm 788	2,862 \pm 783	<0.001
Fat, g/day	124 \pm 17	121 \pm 17	<0.001
Fiber, g/day	25.6 \pm 8.0	25.7 \pm 7.6	0.36
Alcohol, g/day	17.6 \pm 21.1	18.8 \pm 21.8	<0.001
Calcium, mg/day	1,424 \pm 426	1,366 \pm 699	<0.001
Fruits, g/day	84.3 \pm 78.9	96.6 \pm 78.7	<0.001
Vegetables, g/day	43.2 \pm 40.8	54.5 \pm 45.4	<0.001
Red meat, g/day	167 \pm 79.9	160 \pm 72.1	<0.001
Coffee, ml/day	674 \pm 344	489 \pm 305	<0.001
Physical activity (work), ^b %			0.001
Nonworker	43	40	
Sedentary	13	6	
Walker	17	21	
Walker/lifter	17	15	
Heavy labor	10	8	
Gastrointestinal symptoms, % past 4 mo			
Constipation	7	7	0.61
Diarrhea	8	11	0.001
Flatulence	25	31	0.001
Heartburn	26	31	0.001
Intestinal cramps	17	21	0.001
Nausea	5	5	0.11

a: Energy-adjusted means and standard deviations are reported for nutrients and foods.

b: Percentages may not add to 100 because of rounding.

tory questionnaire, tea drinkers were more likely to report experiencing gastrointestinal symptoms, including diarrhea, intestinal cramps, nausea, heartburn, and flatulence. Gastrointestinal symptoms did not appreciably alter the associations between tea, coffee, and colon or rectal cancer. Risk estimates for the association between symptoms and colon cancer ranged from 0.9 to 1.4 and for the association between symptoms and rectal cancer ranged from 0.7 to 1.9 and, in general, had wide CI and were not important predictors of cancer incidence at either site.

The median intake of coffee did not differ between cases of colon and rectal cancer and noncases (median 5.0 cups/day). The evaluation of the association between coffee and colon and rectal cancer is presented in Table 2. Overall, coffee drinking was not significantly associated with cancer at either site. Initially, we observed a nonsignificant protective association of coffee intake for colon cancer; however, in subsequent analyses, we found that tea was a significant confounder for the relationship between coffee and cancer risk and, when included in the multivariate models, adjusted the RR for coffee upward.

Table 2. RR of Colon and Rectal Cancer According to Coffee Intake^a

Coffee Intake, cups/day	Cases/Noncases	RR	95% CI	P for Trend
<i>Colon cancer^b</i>				
≤4	51/10,083	1.00		0.11
>4	31/8,804	0.73	0.47–1.16	
>6	24/8,115	0.69	0.42–1.13	
<i>Colon cancer^c</i>				
≤4	51/10,083	1.00		0.45
>4	31/8,804	0.83	0.52–1.32	
>6	24/8,115	0.84	0.50–1.40	
<i>Rectal cancer^d</i>				
≤4	33/10,099	1.00		0.44
>4	29/8,806	1.05	0.63–1.75	
>6	17/8,124	0.77	0.43–1.40	
<i>Rectal cancer^e</i>				
≤4	33/10,099	1.00		0.36
>4	29/8,806	1.02	0.61–1.71	
>6	17/8,124	0.74	0.40–1.36	

a: Abbreviations are as follows: RR, relative risk; CI, confidence interval.

b: Adjusted for age, intervention group, calcium, occupational physical activity, and body mass index (BMI).

c: Adjusted for age, intervention group, calcium, occupational physical activity, BMI, and tea.

d: Adjusted for age, intervention group, calcium, occupational physical activity, BMI, and serum cholesterol.

e: Adjusted for age, intervention group, calcium, occupational physical activity, BMI, serum cholesterol, and tea.

The distribution of intake of tea was profoundly and negatively skewed for cases and noncases, with median values <1 cup/day. The RR of colon and rectal cancer according to tea intake is shown in Table 3. There was a significant, positive association between tea consumption and risk for colon cancer, and there was a strong dose-response effect. Compared with persons who did not drink tea, those who consumed <1 cup/day had an RR of 1.40 (95% CI = 0.84–2.33) and moderate-to-heavy tea drinkers, those who consumed ≥1 cup/day, had an RR of 2.09 (95% CI = 1.34–3.26) for colon cancer (*p* for trend = 0.001). In contrast, for rectal cancer, there was little effect of tea consumption. The RR for the middle and upper categories of tea intake, compared with persons who did not consume tea, were 1.02 (95% CI = 0.57–1.82) and 0.87 (95% CI = 0.47–1.60), respectively (*p* for trend = 0.64).

There was no effect modification observed by intake level of fat, fiber, sugar, alcohol, iron, fruit, or red meat intake for any of the coffee or tea associations with cancer risk. There was no interaction between coffee and tea intake in any of the models. In addition, the effect of coffee or tea was not modified by the intervention, by location of the tumor in the colon (distal vs. proximal), or by smoking level.

Complete information was available for 46 colon cancer deaths in this population. Not surprisingly, in this subgroup analysis, coffee was not associated with death from colon cancer. Tea was positively and significantly associated with colon cancer mortality. The RR of tea and death for colon cancer, after adjustment for other covariates, was 1.42 (95% CI = 0.65–3.10) for the middle category and 2.38 (95% CI = 1.24–4.59, *p* for trend = 0.01) for the upper category compared with the lowest category of tea consumption. With only 23 rectal cancer deaths, calculated risk estimates were too unstable to draw strong inferences concerning an association between coffee and/or tea consumption and rectal cancer mortality.

Table 3. RR of Colon and Rectal Cancer According to Tea Intake

Tea Intake, cups/day	Cases/Noncases	RR	95% CI	P for Trend
<i>Colon cancer^a</i>				
0	52/17,312	1.00		0.001
<1	22/4,946	1.40	0.84–2.33	
≥1	32/4,744	2.09	1.34–3.26	
<i>Rectal cancer^b</i>				
0	50/17,314	1.00		0.64
<1	16/4,953	1.02	0.57–1.82	
≥1	13/4,762	0.87	0.47–1.60	

a: Adjusted for age, intervention group, calcium, occupational physical activity, and BMI.

b: Adjusted for age, intervention group, calcium, occupational physical activity, BMI, and serum cholesterol.

Discussion

In this trial-based prospective cohort, we found an inverse association between coffee intake and both colon and rectal cancer that was statistically nonsignificant after adjustment for other covariates. Furthermore, coffee consumption was not associated with colon cancer mortality. Surprisingly, we found that tea intake was associated with increased risk of cancer of the colon and with colon cancer mortality. Tea was not associated with rectal cancer incidence.

The data suggesting that coffee intake may be related to risk of large bowel cancer are not consistent, particularly among the prospective cohort studies with published results (4,13–15,33,34). Of the four studies based in US populations, one found a significant positive association between coffee and colon cancer incidence (13) and one conducted within the same prospective cohort showed a significant relationship between coffee consumption and fatal colon cancer (14). A large Norwegian cohort study found a positive but statistically nonsignificant association for colon cancer and a nonsignificant negative association for rectal cancer (34).

Several published case-control studies have evaluated the relationship between coffee consumption and risk for colon or rectal cancer. Most have shown a protective effect, with little consistency in the magnitude of the association. We found seven studies that reported a significantly decreased risk for colorectal cancer with increased coffee consumption, with odds ratios for the highest level of intake ranging from 0.38 to 0.78 (6–11,35). Only two case-control studies reported significantly increased risk for colorectal cancer with coffee consumption (12,36), and one found no association (17).

Two recent reviews have examined the relationship between coffee consumption and large bowel cancer risk (5,19). A working group from the International Agency for Research on Cancer concluded that the available cohort studies had generally been interpreted as showing no association, and the collective evidence for case-control studies was consistent with a protective effect. In a critical review of the literature, Rosenberg (5) concluded that the validity of some of the studies of coffee and colon and rectal cancer cannot be assessed, because authors provided few details on their methods, measures of consumption were not provided, the distribution of cases and controls according to consumption was not presented, and adjustments were not made for important confounders.

Our results for coffee intake and colon and rectal cancer incidence are in general agreement with the null findings of those of cohort studies conducted in Norway (34) and Sweden (4), where, like Finland, coffee is also consumed at high levels. Lowenfels (37) speculated that the inverse association of coffee with colon cancer risk might be evident only in populations that consume high-fat, low-fiber diets, putatively because of coffee's salutary effect on intestinal transit

time and bile acid excretion. The Finnish diet differs from the typical "Western" diet. For example, although the fat content of our study participants' diet was relatively high (Table 1), fiber intake was well above the mean intake for US men of 17.5 g/day (38). If Lowenfels' theory is true, this may at least partially explain the lack of association we observed for coffee and large bowel cancer.

Recent reviews of the epidemiologic literature have shown inconsistencies in the reported relationship between tea and most cancers, including colon and rectal cancers (19,20). Published studies have been conducted in a variety of populations that differed in patterns of tea consumption and in the type of tea consumed, as well as in other health-related factors.

Ecological evidence supports a potential harmful effect of tea consumption on colon cancer risk. For example, countries with traditionally high tea consumption (up to 4.4 kg/capita/yr), such as England, Ireland, and Australia, have colon cancer rates more than twice that seen in Finland, a country with relatively low tea intake (0.2 kg/capita/yr). In a study that surveyed tea consumption in 29 countries, intake was positively related to colon cancer in men and women (39).

Of the five published prospective cohort studies conducted in Europe and the United States (31,40–43), only one showed a significant inverse association. In a population of London men (42), tea intake was negatively associated with colon and rectal cancer and a marginally significant trend was seen for colon cancer. Unfortunately, in their analysis, the authors did not attempt to adjust for other colon cancer risk factors that have been associated with tea drinking (42). This study is also unique because of the relatively high amount of tea consumed by the referent group, which included persons who consumed up to 5 cups/day. No association was observed between tea consumption and risk of colon or rectal cancer in The Netherlands (40). In a cohort of Hawaiian Japanese men (30), a population at high risk for rectal cancer, tea intake was not correlated with colon cancer risk but was positively related to rectal cancer risk, with a significant trend. In Iowa women (43), the RR for colon and rectal cancer for the upper compared with the lowest quartile of tea intake were about 0.75, but were not significant, and no trends were observed. Phillips and Snowden (13), in their prospective study of Seventh-Day Adventists, did not present data on tea; however, they commented that they found no significant association between tea and colorectal cancer mortality risk.

We found five case-control studies that have examined the association between black tea consumption and large bowel cancer (6,7,11,31,44). Of these, two reported significant associations. In a Swedish study, Baron and co-workers (11) observed a protective association between tea consumption and rectal cancer, although no relationship was seen for tea and colon cancer. In an Italian study including 339 colon and 236 rectal cancer cases (6), a marginally significant positive association was seen between tea and colon cancer

and a significant positive association was seen between tea and rectal cancer. Unfortunately, the authors only report data that group participants as nondrinkers or drinkers of tea; therefore, a trend across level of intake could not be assessed.

There is experimental evidence to support a positive association for black tea with colon cancer. Several groups have demonstrated that black tea is mutagenic *in vitro* (45–47), and one study suggested that mutagenic substances found in tea are made available in the lower digestive tract through cleavage by gut enzymes (48). Black tea also contains tannin, a known carcinogen, in higher amounts than coffee (49) and forms hydrogen peroxide (50) and free radicals (51) *in vitro*. In mice, Bogovski and associates (52) found that black tea infusion shortened tumor appearance time. Phenols, found in tea, have been reported to have strong cancer-promoting actions in laboratory animals (53), and subcutaneous injections of tannin from black tea resulted in tumor formation (54). Although phenols have also been shown to trap nitrosating species *in vitro* and *in vivo*, which would suggest a protective effect, paradoxically, many foods with exceptionally high phenol content (tea gruel, betel nuts) have been associated with increased risk for upper digestive tract cancers (55,56).

Some issues should be considered when the results of this study are interpreted. First, the distribution and levels of intake of coffee and tea were very different from each other in this population. Coffee consumption was uniformly high (mean daily consumption 550 ml) and relatively homogeneous (interquartile range 420–770 ml). When we analyzed coffee intake as our primary exposure we were not able to use nondrinkers of coffee as the referent group, because very few cancer cases did not consume coffee. This may have made it more difficult to observe significant risk differences across levels of coffee intake. By contrast, tea consumption was uniformly low (mean daily consumption 73 ml) and the distribution of tea intake was negatively skewed; there were many nondrinkers of tea and fewer moderate-to-heavy tea drinkers. This may have been an advantage in our analysis, because, with many nondrinkers of tea, risk estimates for the referent group are quite stable. In addition, our observation that tea intake and colon cancer risk are significantly associated may have been due to our ability to better tease out group differences at the very low end of the dose-response curve represented by this population. On the other hand, the limited number of moderate-to-heavy tea drinkers may differ from the rest of the population in other areas related to diet and health. These behaviors or conditions may serve to confound an association between tea intake and cancer risk. Indeed, tea drinkers were more likely to report having several gastrointestinal conditions or symptoms at baseline; however, none of these were associated with colon cancer risk. In our analyses, we carefully controlled for all important available measured confounders; however, we cannot rule out the possibility that residual confounding contributed to our results. In these analyses, inclusion of gastrointestinal problems in the final

models did not appreciably alter the RR estimates for tea intake. Furthermore, although tea drinking and healthful behaviors were related, the disease was not diagnosed earlier in colon or rectal cancer cases who were tea drinkers (e.g., time from randomization to diagnosis was similar). The dietary information for this study, including tea consumption, was collected at baseline, well before cancers were diagnosed. The dietary instrument used to quantify measurement of tea and coffee consumption has been shown to be both valid and reproducible, minimizing the probability of misclassifying cases and controls as to the level of intake of these two beverages. Indeed, the significant association between tea and colon cancer observed in this study probably cannot be explained by misclassification, inasmuch as the impact of misclassification would likely attenuate the true relationship between tea and cancer. Lastly, the generalizability of these results may be somewhat limited because the study was restricted to older male smokers who participated in a clinical trial.

In summary, we observed an upward trend in risk of colon cancer with increasing level of tea consumption but found no association between coffee and colon cancer risk. For rectal cancer, we observed no significant association with tea or coffee intake. Given the strength of the tea-colon cancer association and the significant gradient of risk we observed across level of intake, further epidemiologic research in other populations, with careful consideration of confounding, should be encouraged. Future studies to examine the effect of tea earlier in the process of colonic neoplasia by analyzing the association between tea consumption and the incidence of colorectal adenomatous polyps may be warranted.

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