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Stephanie A. Navarro Silvera  
*Montclair State University, silveras@montclair.edu*

Meera Jain  
*University of Toronto*

Geoffrey R. Howe  
*Columbia University - Mailman School of Public Health*

Anthony B. Miller  
*University of Toronto*

Thomas E. Rohan  
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# Intake of Coffee and Tea and Risk of Ovarian Cancer: A Prospective Cohort Study

Stephanie A. N. Silvera, Meera Jain, Geoffrey R. Howe, Anthony B. Miller,  
and Thomas E. Rohan

**Abstract:** *There is some evidence from case-control studies that coffee consumption might be positively associated with ovarian cancer risk, whereas the epidemiologic evidence regarding tea consumption and ovarian cancer is inconsistent. To date, there have been few prospective studies of these associations. Therefore, we examined ovarian cancer risk in association with both coffee and tea intake in a prospective cohort study of 49,613 Canadian women enrolled in the National Breast Screening Study (NBSS) who completed a self-administered food frequency questionnaire between 1980 and 1985. Linkages to national mortality and cancer databases yielded data on deaths and cancer incidence, with follow-up ending between 1998 and 2000. Data from the food frequency questionnaire were used to estimate daily intake of coffee and tea. Cox proportional hazards models were used to estimate hazard ratios (HR) and 95% confidence intervals (CI) for the association between categories of coffee and tea intake and ovarian cancer risk. During a mean 16.4 years of follow-up, we observed 264 incident ovarian cancer cases. Tea intake was not associated with ovarian cancer risk in our study population. In contrast, a borderline positive association was observed among women who drank >4 cups coffee/day compared to women who did not drink coffee (HR = 1.62, 95% CI = 0.95–2.75,  $P_{\text{trend}} = 0.06$ ). Given the pervasive use of these beverages, the associations between coffee and tea consumption and ovarian cancer risk warrant investigation in further prospective studies.*

## Introduction

Coffee and tea are widely consumed throughout the world. Hence it is of interest to know if they have any effects on health. Many outcomes have been examined in relation to coffee and tea consumption, including hypertension, coro-

nary heart disease, and various cancers (1–3). In relation to cancer, some of the volatile components of coffee, including caffeine, may be carcinogenic to humans (4), whereas tea has tumor-inhibitory effects in several organs (including skin, oral cavity, lung, esophagus, stomach, small intestine, pancreas, and mammary gland) (5).

Ovarian cancer is the leading cause of fatality among gynecological malignancies. However, relatively little is known about its etiology (6). There is some biological support for the possibility that coffee and tea consumption might influence ovarian cancer risk. While caffeine may enhance or mimic the effect of gonadotropins, which are thought to increase the risk of ovarian cancer (4), tea and some of its components have been shown to be inversely associated with circulating estrogen levels (7,8), which are thought to increase proliferation and malignant transformation of ovarian epithelial cells (9).

To date, it appears that the relationship between coffee intake and ovarian cancer risk has been examined in ten case-control studies (4,10–18) and three prospective cohort studies (10–12). In two of those prospective investigations no association was observed (10,11) while the other one (12) found a non-significant increased risk. Most case-control studies of coffee intake and ovarian cancer risk have reported positive associations, although the increases in risk have generally been small (13). The results of case-control studies of tea intake and ovarian cancer risk have been inconsistent (4,11,14,23–26) and the two prospective cohort studies to date have reported a decrease in risk associated with relatively high tea consumption (14,15). The results of previous studies are summarized in Table 1.

Given the paucity of prospective data, we examined the relationship between coffee and tea intake and ovarian cancer risk in a large prospective cohort study of Canadian women.

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Stephanie A.N. Silvera and Thomas E. Rohan are affiliated with Department of Epidemiology and Population Health, Albert Einstein College of Medicine, 1300 Morris Park Avenue, Room 1301, NY 10461. Meera Jain and Anthony B. Miller are affiliated with Department of Public Health Sciences, University of Toronto, Toronto, Canada. Geoffrey R. Howe is affiliated with Department of Epidemiology, Mailman School of Public Health, Columbia University, New York, NY.

**Table 1.** Summary of Previous Epidemiologic Studies of Tea and Alcohol Consumption and Ovarian Cancer Risk

Reference	Study Design	Number of Cases	Number of Controls/ Non-Cases	Comparison	Risk estimate (95% CI)
Tea Consumption					
Byers et al., 1983 (39)	Case-control	274	1034	≥ 3 C/day vs. none	0.84 (ns)
Miller et al., 1987 (40)	Case-control	290	1056	≥ 5 C/day vs. none	0.5 (0.2–1.0)
LaVecchia et al., 1992 (24)	Case-control	742	6417	≥ 1 C/day vs. none	1.2 (1.0–1.4)
Kuper et al., 2000 (4)	Case-control	563	523	≥ weekly vs. never	1.06 (0.83–1.36)
Tavani et al., 2001 (22)	Case-control	1031	2411	≥ 1C/month vs. none	0.9 (0.75–1.08)
Zhang et al., 2002 (29)	Case-control	254	601	≥ 1 C/day vs. none	0.39 (0.27–0.57)
Jordan et al., 2004 (23)	Case-control	696	786	≥ 4 C/day vs. never	1.10 (0.76–1.61)
Zheng et al., 1996 (14)	Prospective Cohort	107	Cohort of 35,369	Weekly vs. none	0.53 (0.49–1.84)
Larsson et al., 2005 (10)	Prospective Cohort	301	Cohort of 66,651	≥ 2 C/day vs. <1 C/day	0.54 (0.31–0.91)
Coffee Consumption					
Trichopoulos et al., 1981 (41)	Case-control	92	105	≥ 3C/day vs. none	2.2
Hartge et al., 1982 (42)	Case-control	158	187	≥ 4C/day vs. none	1.4 (0.6–3.0)
Byers et al., 1983 (39)	Case-control	274	1034	≥ 3C/day vs. none	0.97
Cramer et al., 1984 (43)	Case-control	215	215	≥ 5C/day vs. none	1.5
LaVecchia et al., 1984 (44)	Case-control	247	494	≥ 4C/day vs. none	2.2 (1.2–1.4)
Tzonou et al., 1984 (45)	Case-control	150	250	≥ 3.5C/day vs. none	1.5
Miller et al., 1987 (40)	Case-control	290	580	≥ 5C/day vs. none	1.1 (0.6–2.0)
Whittemore et al., 1988 (46)	Case-control	188	539	≥ 4C/day vs. none	2.07 (0.97–4.38)
Polychronopoulou et al., 1993 (47)	Case-control	189	200	≥ 2C/day vs. none	1.09 (0.52–2.27)
Kuper et al., 2000 (4)	Case-control	549	516	≥ 4C/day vs. none	1.88 (1.14–3.09)
Snowdon & Philips, 1984 (11)	Prospective Cohort	50	Cohort of 16,190	≥ 1C/day vs. < 1	1.2 (0.6–2.5)
Stensvold et al., 1994 (12)	Prospective Cohort	93	Cohort of 21,238	≥ 7C/day vs. ≤ 2	2.0
Larsson et al., 2005 (10)	Prospective Cohort	301	Cohort of 66,651	≥ 4C/day vs. none	1.02 (0.62–1.69)

## Methods

### Study Population

The design of the study has been described in detail elsewhere (16). Briefly, 89,835 women aged 40–59 yr were recruited into the Canadian National Breast Screening Study (NBSS) between 1980 and 1985 from the general Canadian population by various means, including personal invitation by letter, group mailings to employees of large institutions and to members of professional associations, advertisements in newspapers, and public service announcements on radio and television (17).

### Questionnaires

At recruitment into the cohort, participants completed self-administered questionnaires that sought information on demographic characteristics, lifestyle factors, menstrual and reproductive history, and use of oral contraceptives and replacement estrogens. Women who reported having regular menstrual periods within the past 12 mo were classified as premenopausal. Women whose menstrual periods ceased at least 12 mo before enrollment into the study were considered to be postmenopausal (18).

Starting in 1982 (that is, after some participants had completed their scheduled visits to the screening centers), a self-administered food frequency questionnaire (FFQ) was distributed to all new attendees at all screening centers and to

women returning to the screening centers for re-screening (19). The FFQ sought information on usual portion size and frequency of consumption of 86 food items, and included photographs of various portion sizes to assist respondents with quantifying intake. As part of the FFQ women were queried about usual frequency and amount of coffee and tea consumed. A comparison between the self-administered questionnaire and a full interviewer-administered questionnaire, which has been subjected to both validity and reliability testing (19) and used in a number of epidemiologic studies (20), revealed that the two methods gave estimates of intake of the major macro-nutrients which were moderately to strongly correlated with each other (reported correlation coefficients ranged from 0.47 to 0.72) (19). A total of 49,613 dietary questionnaires were returned and available for analysis.

### Ascertainment of Incident Ovarian Cancer Cases and Deaths

Incident ovarian cancer cases and deaths amongst cohort members were ascertained respectively by means of computerized record linkages to the Canadian Cancer Database and to the National Mortality Database, both of which are maintained by Statistics Canada. The linkages to the databases yielded data on cancer incidence and mortality to December 31, 2000, for women in Ontario, December 31, 1998, for women in Quebec, and December 31, 1999, for women in other provinces.

**Table 2.** Baseline Characteristics of the Study Population by Outcome

	Cases of incident ovarian cancer ( <i>n</i> = 264)	Non-cases ( <i>n</i> = 48,508)
Mean age (yr)	49.7 (5.7) <sup>‡</sup>	48.6 (5.6)
Mean coffee intake (cups/day)	2.3 (2.0)	2.1 (2.0)
Mean tea intake (cups/day)	1.4 (1.6)	1.4 (1.7)
Mean education (yr)	13.2 (3.0)	12.8 (3.0)
Mean body mass index (kg/m <sup>2</sup> )	24.5 (4.2)	24.8 (4.6)
Parity (% parous)	78.0	85.2
Mean age at first live birth (yr) <sup>¥</sup>	24.6 (4.6)	24.3 (4.7)
Mean age at menarche (yr)	12.8 (1.5)	12.8 (2.0)
Menopausal status (% postmenopausal)	46.8	42.8
Smoking history (% ever)	52.6	48.5
Any alcohol intake (%)	74.6	73.9
Any vigorous physical activity (%)	75.7	71.0

<sup>‡</sup> Numbers in parentheses represent the standard deviation.

<sup>¥</sup> Among parous women only.

### Statistical Analysis

Of the 49,613 women for whom dietary data were available, we excluded women with extreme energy intake values (at least three standard deviations above or below the mean value for log<sub>e</sub> caloric intake) (*n* = 502); women with prevalent ovarian cancer at baseline (*n* = 20); and women who reported at baseline that they had had a bilateral oophorectomy (*n* = 315). These exclusions left 48,776 women available for analysis, among whom there were 264 incident cases of ovarian cancer. Study participants were considered to be at risk from their date of enrollment until the date of diagnosis of ovarian cancer, the date of termination of follow-up (the date to which cancer incidence data were available for women in the corresponding province), or the date of death, whichever occurred earliest.

Cox proportional hazards models (using age as the time scale) were used to estimate hazard ratios (HR) and 95% confidence intervals (CI) for the association between coffee and tea intake (separately) and ovarian cancer risk. For these analyses, coffee and tea intake were categorized to create 4-level variables (none, and three non-zero levels). Multivariate models included the variables listed in the footnotes to Table 2. To test for trends in risk with increasing levels of the exposures of interest, we assigned the coffee and tea intake categories their ordinal number and then fitted the assigned value of each risk factor as a continuous variable in the risk models. We then evaluated the statistical significance of the corresponding coefficient using the Wald test (21). Use of the lifestest procedure in SAS<sup>®</sup> showed that the proportional hazards assumption was met in this dataset. All analyses were performed using SAS version 9 (SAS Institute, Cary, NC).

### Results

The average duration of follow-up for cohort members was 16.4 yr, corresponding to a total of 801,414 person-years of follow-up. The mean (SD) age at diagnosis for the

cases was 59.4 (± 7.2) years. For the cohort as a whole, the mean (± SD) coffee intake was 2.1 cups/day (± 2.0) and the mean tea intake was 1.4 cups/day (± 1.7). Compared with non-cases, ovarian cancer cases were less likely to be parous at baseline, and more likely to have ever smoked and to have participated in any vigorous physical activity (Table 2). No appreciable difference was observed in mean age at baseline, body mass index, age at menarche, alcohol intake, or age at first birth by case status at the end of follow-up.

Table 3 shows that in both age- and multivariate-adjusted models, ovarian cancer risk varied little in association with tea intake. In contrast, women who drank ≥4 cups of

**Table 3.** Adjusted Hazard Ratios<sup>§</sup> and 95% Confidence Intervals (CI) for the Association Between Daily Coffee and Tea Intake and Risk of Ovarian Cancer

Intake (cups/day)	Cases/ Person Years	Age-adjusted		Multivariate	
		HR	95% CI	HR	95% CI
<b>Coffee</b>					
None	34/126,119	1.00	Referent	1.00	Referent
>0 – <=1	110/362,997	1.14	0.77–1.68	1.18	0.76–1.83
2–3	79/217,426	1.36	0.91–2.05	1.36	0.86–2.15
≥ 4	41/94,853	1.66	1.04–2.64	1.62	0.95–2.75
P <sub>trend</sub>			0.02		0.06
<b>Tea</b>					
None	61/179,331	1.00	Referent	1.00	Referent
>0 – <=1	143/449,340	0.90	0.66–1.22	0.86	0.63–1.18
2–3	38/121,890	0.86	0.57–1.29	0.81	0.53–1.23
≥ 4	22/50,834	1.18	0.72–1.93	1.07	0.64–1.79
P <sub>trend</sub>			0.90		0.77

<sup>§</sup> Multivariate models included age (time to event variable), smoking history (never, former, current), pack-years of smoking (never plus 4 non-zero categories), alcohol intake (any vs. none), education (>12 yr vs. <=12 yr), BMI in kg/m<sup>2</sup> (<25, 25–29, >30), parity (parous vs. nulliparous), participation in vigorous physical activity (any vs. none), menopausal status, oral contraceptive use (zero plus 3 non-zero categories), energy intake (continuous), lactose intake (continuous), study center, and randomization group.

coffee/day had a 62% increase in ovarian cancer risk compared to women who did not drink coffee (HR = 1.62, 95% CI = 0.95–2.75,  $P_{\text{trend}} = 0.06$ ) in the multivariate model. Mutual adjustment for both coffee and tea in a multivariate model did not materially alter the hazard ratios for each beverage (data not shown).

The results for each of the analyses presented above were similar after exclusion of case subjects diagnosed within 1 yr of recruitment ( $n = 10$ ).

## Discussion

In the present cohort study, we observed a positive association between coffee intake and ovarian cancer risk over a 16-yr follow-up period. In contrast, there was no association between tea consumption and risk.

Our findings for coffee consumption are similar to those of the majority of previous case-control studies (4,10–18) and to those of one (12) of the three (10–12) previous prospective studies, the other two of which showed no association (10,11) (Table 3). It is important to note that the caffeine levels in coffee can range from 70–150 mg/cup due to differences in brewing method and species of coffee used (13), which may account, in part, for some of the conflicting findings in the literature. With respect to tea consumption, our null finding is in accord with those of three previous case-control studies (4,22,23). However, the epidemiologic evidence concerning the relationship between tea consumption and ovarian cancer risk is inconsistent, given that one case-control study (24) reported a weak positive association and there was some evidence of an inverse association between tea and ovarian cancer risk in three (14,24,25) case-control studies and two prospective cohort studies (14,15).

Although the potential carcinogenicity of caffeine has been the subject of much investigation, its role in cancer development is unresolved (13). There is some evidence that caffeine may affect DNA structure and function by inhibition of poly(ADP-ribosylation) reactions, which are important in postreplication repair of damaged DNA (4), and that it may potentiate DNA damage via inhibition of DNA repair during the S and G2 phases of the cell cycle (25). In addition to caffeine, coffee contains volatile substances (heterocyclic rings containing oxygen, sulfur or nitrogen including aldehydes) which may be carcinogenic to humans (26). In contrast, coffee also contains lignans (27) and has been shown to increase plasma enterolactone concentrations (27) and serum equol concentrations (28). There is some evidence that phytoestrogens (e.g., isoflavonoids and lignans) with estrogen-like activities may be associated with a reduced risk of hormone-dependent neoplasms (28) and that individuals with high serum equol concentrations may be at lower risk for hormone dependant neoplasms such as breast cancer (27). The potentially divergent effects of caffeine and the phytoestrogen effect of coffee may explain, in part, the conflicting literature. These possible mechanisms of action are speculative and further research is needed to investigate the potential role of caffeine in carcinogenesis.

In contrast to coffee, there is evidence from animal studies that tea has anti-carcinogenic effects (3,29). In animal experiments, tea polyphenols, the most likely anti-carcinogenic agents in tea (3), have been shown to be absorbed from the digestive tract and to reach various organs, including the ovary, via the circulation (30). The concentration of antioxidative polyphenol compounds in tea varies according to the type of tea (e.g., green, black, oolong) (3), with green tea having greater total phenols and higher antioxidant capacity than black tea (31). Therefore, differences in findings from epidemiologic studies may be due to differences in the types of tea consumed in different populations (29). In the 1980s (when data on tea consumption were collected for this study), black tea was the predominant form of tea consumed in North America, accounting for approximately 88% of all the tea consumed (32). Currently black tea accounts for approximately 76–80% of all tea consumed in North America (33,34) whereas green tea accounts for only 9% of all tea consumed in the United States, up from 4% in 2001 (35). In contrast, a survey in Japan has shown that 65% of the population reported drinking green tea >5 times per week and 85% report drinking green tea at least once per week (35). These differences likely account for the discrepancy between our findings and those from the cohort study of Zheng et al. (14). Larsson et al. (15), however, examined black tea consumption in a cohort study in Sweden and found a statistically significant reduced risk of ovarian cancer (Table 1). In addition, there is evidence to suggest that the method of preparation (hot vs. cold/iced) and the amount of dry tea used may influence the anticancer effects of tea by altering the concentration of polyphenols (36,37). Differences in the method of preparation may account, in part, for the conflicting results in the literature. Thus, future studies of tea consumption and cancer risk should measure not only for the amount of intake but also the type of tea consumed and the methods of preparation.

Our data are limited in that the number of ovarian cancer cases may have been insufficient to allow detection of small effects. In addition, inaccurate recall of coffee and tea consumption may have biased our estimates of association towards the null (38). This limitation applies to previous (prospective) studies as well. It is also possible that the use of a one-time assessment of coffee and tea intake may not have been representative of the consumption habits of the study participants over the course of follow-up. If individuals in the cohort changed their consumption patterns over time, this may have resulted in misclassification of exposure. Finally, although we adjusted our estimates for a wide range of potentially confounding variables, residual confounding by dietary and other factors cannot be excluded.

In conclusion, the results of our study suggest that while tea intake was not associated with ovarian cancer risk, relatively high coffee intake may be associated with an increase in risk. Given the pervasive use of these beverages and biologic support for possible carcinogenic (for coffee) or anti-carcinogenic (for tea) effects, the associations between coffee and tea consumption and ovarian cancer risk warrant investigation in further prospective studies, particularly those

in which type of beverage and methods of preparation are assessed.

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