American Journal of Epidemiology
Vol. 149, No. 2
Printed in U.S.A.

A BRIEF ORIGINAL CONTRIBUTION

Coffee and Tea Intake and the Risk of Myocardial Infarction

Howard D. Sesso,[1,2] J. Michael Gaziano,[1,3,4] Julie E. Buring,[1,2,5] and Charles H. Hennekens[1,2,5]

The authors investigated the association of caffeinated coffee, decaffeinated coffee, and tea with myocardial infarction in a study of 340 cases and age-, sex-, and community-matched controls. The odds ratio for drinking ≥4 cups/day of caffeinated coffee versus drinking ≤1 cup/week was 0.84 (95% confidence interval (CI) 0.49–1.42) after adjustment for coronary risk factors (1 cup = 237 ml). The odds ratio for drinking >1 cup/day of decaffeinated coffee versus nondrinkers was 1.25 (95% CI 0.76–2.04). For tea, the odds ratio for drinking ≥1 cup/day versus nondrinkers was 0.56 (95% CI 0.35–0.90). In these data, only tea was associated with a lower risk of myocardial infarction. *Am J Epidemiol* 1999;149:162–7.

caffeine; case-control studies; coffee; flavones; myocardial infarction; tea

Recent meta-analyses suggest no association between caffeinated coffee and myocardial infarction after adequate control of cigarettes, but they could not rule out a possible increased risk of myocardial infarction for consumption greater than 5 cups per day (1 cup = 237 ml) (1, 2). Evidence of an association between decaffeinated coffee and myocardial infarction remains sparse and conflicting (3–10). Whereas earlier studies on tea consumption suggested no association (11–16), recent evidence suggests a reduction in the risk of myocardial infarction due to its flavonoid content (17–22). Regular black tea is a major source of flavonoids (20, 21), a group of compounds in plant foods with antioxidant effects (23, 24) that may help to retard atherosclerosis (25, 26). Using a case-control study of patients with first myocardial infarction and neighborhood controls of the same age and sex, we investigated the effects of each beverage on the risk of myocardial infarction.

Received for publication December 29, 1997, and accepted for publication June 8, 1998.

Abbreviations: CI, confidence interval; OR, odds ratio.

[1] Division of Preventive Medicine, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA.
[2] Department of Epidemiology, Harvard School of Public Health, Boston, MA.
[3] Cardiovascular Division, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA.
[4] Massachusetts Veterans Epidemiology Research and Information Center, Veterans Affairs Medical Center, Brockton/West Roxbury, MA.
[5] Department of Ambulatory Care and Prevention, Harvard Medical School, Boston, MA.
Reprint requests to Dr. Howard D. Sesso, Brigham and Women's Hospital, 900 Commonwealth Avenue East, Boston, MA 02215.

MATERIALS AND METHODS

Subjects and methods of the Boston Area Health Study have been described previously (27). Briefly, subjects included white men and women aged less than 76 years, with no prior history of myocardial infarction or angina pectoris. Cases of myocardial infarction had no symptoms more than 24 hours before presentation at six different Boston area hospitals and were confirmed by evidence of a rise in creatine kinase. Cases had home interviews approximately 10 weeks after hospital discharge. For each eligible and willing case, we selected a control matched on age (within 5 years), sex, and area of residence. Of the eligible subjects contacted, 84 percent of cases discharged alive and 60 percent of controls were enrolled, yielding 340 case-control pairs in the study.

Extensive information was obtained on coronary risk factors related specifically to the time before the myocardial infarction for the cases and before the interview for the controls. A 116-item semiquantitative food frequency questionnaire assessed average dietary intake over the past year; for cases, average intake represented the year preceding their myocardial infarction. We defined four approximately equal categories of beverage consumption based upon the distribution among the controls: for caffeinated coffee, ≤1 cup/week, 2–7 cups/week, >1–3 cups/day, and ≥4 cups/day; for decaffeinated coffee, none, 1–3 cups/month, 1–7 cups/week, and >1 cup/day; and for tea, none, 1–3 cups/month, 1–6 cups/week, and ≥1 cup/day. Lipid levels from fasting venous blood samples were determined using Lipid Research Clinics methods (28–30). The statistical analyses with lipids are restricted to subjects with full lipid profiles.

Risk factor comparisons between cases and controls were conducted using t tests for continuous variables and χ^2 tests for categorical variables. All t tests for continuous variables were age and sex adjusted. We compared subjects according to beverage preference, defined as drinking ≥1 cup/day of one beverage but <1 cup/day of the other beverages. Matched-pair and unmatched odds ratios for myocardial infarction were virtually identical for all logistic regression analyses, allowing us to present unmatched analyses (31). Odds ratios and 95 percent confidence intervals were calculated using the lowest level of consumption as the reference category. Trend tests were calculated using categorical variables. Crude models were adjusted for age and sex, and multivariate models were adjusted for all medical and behavioral risk factors for myocardial infarction. Finally, lipids were individually entered into the multivariate model.

RESULTS

As expected, the prevalence and levels of major known coronary risk factors and lipids among cases differed from those among controls ($p < 0.05$) in table 1. We then compared subjects in the highest versus lowest levels of intake for each beverage (data not shown). The heaviest caffeinated coffee drinkers tended to be younger and male, to smoke cigarettes, and to have a type A personality (all $p < 0.05$). Drinking ≥2 cups of decaffeinated coffee daily was associated with higher rates of treatment for high blood pressure and diabetes (both $p < 0.05$). Finally, the heaviest tea drinkers were older and smoked less (both $p < 0.05$), and they had more favorable high density lipoprotein cholesterol levels (1.04 vs. 0.98 mmol/liter (40.3 vs. 37.9 mg/dl), $p = 0.059$) despite lower alcohol consumption.

Few subjects exclusively drank one single beverage. Therefore, in table 2, we compared subjects with a strong preference for drinking each beverage. Subjects preferring caffeinated coffee tended to be younger men who were heavier smokers and drinkers. Preferred decaffeinated coffee drinkers had smoking rates similar to those of caffeinated coffee drinkers but higher rates of treatment for high blood pressure and diabetes. Finally, preferred tea drinkers were older, smoked less, and were less physically active, with higher rates of treatment for high blood pressure compared with caffeinated coffee drinkers.

A total of 70.2 percent of cases and 71.5 percent of controls drank at least 1 cup of caffeinated coffee per day. We found no association between caffeinated coffee intake and the risk of myocardial infarction (table 3). Results were not materially altered by the addition of individual lipids into the model. In a comparison of the highest recorded category of caffeinated coffee intake (≥6 cups/day; $n = 47$) with the reference group (≤1 cup/week; $n = 148$), an apparent crude association (odds ratio (OR) = 1.88, 95 percent confidence interval (CI) 0.94–3.78) was attenuated after multivariate adjustment (OR = 1.38, 95 percent CI 0.63–3.00), mainly because of confounding by smoking. Decaffeinated coffee consumption was considerably less prevalent than was caffeinated coffee, with 27.5

TABLE 1. Characteristics of cases and controls in the Boston Area Health Study, 1982–1983*

	Age (years)	Male sex (%)	History of treatment for high blood pressure (%)	History of diabetes (%)	Family history of myocardial infarction (%)	Type A personality (%)	Cigarette smoking (%)			Taking aspirin daily (%)	Body mass index (kg/m²)
							Never	Former	Current		
Cases (n = 340)	57.7 (9.6)§	78.2	34.7	13.5	22.1	58.8	25.4	31.6	43.1	7.7	25.9 (3.9)
Controls (n = 340)	57.7 (9.7)	78.2	25.1	7.6	15.3	49.4	31.6	40.7	27.7	9.4	25.6 (4 0)
p value	0.95	1.00	0.006	0.013	0 024	0.014		0.001¶		0.42	0.40

	Physical activity index (kcal/week)	Total caloric intake (kcal/day)	Calories from saturated fat (%)	Alcohol intake (g/day)	Total cholesterol (mmol/liter)†	LDL cholesterol (mmol/liter)†	HDL cholesterol (mmol/liter)†	Triglycerides (mmol/liter)‡	LDL/HDL ratio
Cases (n = 340)	3,090 (2,970)	2,482 (927)	13.1 (3.4)	15.1 (26.5)	5.58 (1.13)	3.56 (0.99)	0.91 (0.25)	4.46 (2.95)	4.08 (1.27)
Controls (n = 340)	3,542 (3,009)	2,354 (773)	12.2 (3.0)	19.6 (27.6)	5.48 (1.09)	3.41 (0.92)	1.13 (0.31)	3.49 (2.74)	3.22 (1.25)
p value	0.049	0.050	<0.001	0.031	0.25	0.055	<0.001	<0.001	<0.001

* Unmatched analyses performed. The statistical analyses for lipids (total cholesterol, low density lipoprotein (LDL) cholesterol, high density lipoprotein (HDL) cholesterol, triglycerides, and LDL/HDL) are restricted to those subjects with full lipoprotein profiles (303 cases and 297 controls). All lipid values are age and sex adjusted.

† To convert to mg/dl, multiply by 38.67.

‡ To convert to mg/dl, multiply by 88.57.

§ Numbers in parentheses, standard deviation.

¶ χ^2 test for all three smoking categories.

TABLE 2. Risk factor distribution for myocardial infarction among beverage preferers, Boston Area Health Study, 1982–1983*

	Age (years)	Male sex (%)	History of treatment for high blood pressure (%)	History of diabetes (%)	Family history of myocardial infarction (%)	Type A personality (%)	Cigarette smoking (%)			Take aspirin daily (%)	Body mass index (kg/m²)†
							Never	Former	Current		
Coffee drinkers											
Caffeinated (n = 279)	56.7 (9.4)¶	83.5	23.7	10.8	18.3	55.2	22.6	35.1	42.3	7.6	25.3 (3.9)
Decaffeinated (n = 58)	58.9 (9.1)	65.5	36 2	17.2	36.2	51.7	35.1	21.1	43.9	10.3	25.9 (3.9)
Tea drinkers (n = 40)	60.6 (9.7)	62.5	30.8	2.5	7.5	50.0	37.5	45.0	17.5	7.5	25.0 (3.8)

	Physical activity index (kcal/week)†	Total caloric intake (kcal/day)†	Calories from saturated fat (%)†	Alcohol intake (g/day)†	Total cholesterol (mmol/liter)†,‡	LDL cholesterol (mmol/liter)†,‡	HDL cholesterol (mmol/liter)†,‡	Triglycerides (mmol/liter)†,§	LDL/HDL ratio†
Coffee drinkers									
Caffeinated (n = 279)	3,444 (2,850)	2,452 (844)	12 5 (3.0)	20.4 (28.2)	5.62 (1.02)	3.54 (0.94)	1.03 (0.30)	4.11 (3.31)	3.69 (1.38)
Decaffeinated (n = 58)	3,619 (3,111)	2,425 (697)	12.1 (3.2)	16.4 (26.2)	5.68 (1.29)	3.54 (1.10)	0.94 (0.27)	4.14 (2.14)	3.89 (1.38)
Tea drinkers (n = 40)	2,783 (1,912)	2,419 (888)	12.2 (3.0)	15.2 (29.9)	5.40 (0.96)	3 45 (0.86)	1.02 (0.26)	3.63 (1.93)	3.54 (0.96)

* Subjects drinking ≥1 cup (≥237 ml)/day of one beverage while drinking <1 cup (<237 ml)/day of other beverages. The statistical analyses for lipids (total cholesterol, low density lipoprotein (LDL) cholesterol, high density lipoprotein (HDL) cholesterol, triglycerides, and LDL/HDL) are restricted to those subjects with full lipoprotein profiles (caffeinated coffee, n = 248; decaffeinated coffee, n = 55; tea, n = 34).
† Age and sex adjusted.
‡ To convert to mg/dl, multiply by 38.67.
§ To convert to mg/dl, multiply by 88.57.
¶ Numbers in parentheses, standard deviation.

TABLE 3. Odds ratios and 95% confidence intervals for myocardial infarction by level of caffeinated coffee intake, Boston Area Health Study, 1982–1983

	Cases/controls (no.)	Levels of caffeinated coffee intake				p value (trend)
		≤1 cup/week* (n = 148)	2–7 cups/week (n = 171)	>1–3 cups/day (n = 214)	≥4 cups/day (n = 146)	
Full models						
Age, sex	339/340	1.0	0.76 (0.49–1.18)	0.99 (0.65–1.51)	1.11 (0.69–1.77)	0.45
Age, sex + coronary risk factors†	336/337	1.0	0.78 (0.48–1.26)	0.91 (0.58–1.45)	0.84 (0.49–1.42)	0.69
Lipid-limited models‡						
Age, sex + coronary risk factors†	299/295	1.0	0.83 (0.50–1.39)	0.98 (0.60–1.61)	0.93 (0.53–1.62)	0.97
Age, sex + coronary risk factors† + LDL/HDL ratio§	299/295	1.0	0.86 (0.50–1.47)	0.95 (0.57–1.60)	0.88 (0.49–1.58)	0.78

* One cup = 237 ml.
† Adjusted for age (10-year categories), sex, smoking status (never, former, <1 pack/day, 1–2 packs/day, >2 packs/day), history of medication for high blood pressure, type A personality, family history of myocardial infarction, diabetes, daily aspirin use, body mass index, log of physical activity index, percentage of calories from saturated fat, total caloric intake, and alcohol intake.
‡ Analysis limited to those subjects with full lipoprotein profiles.
§ LDL/HDL ratio, ratio of high density lipoprotein to low density lipoprotein cholesterol.

percent of cases and 23.2 percent of controls drinking ≥1 cup/day. We found no evidence for an association between increasing decaffeinated coffee consumption and the risk of myocardial infarction (table 4). Adjustment for coronary risk factors or individual lipids did not alter the results.

For tea, 24.9 percent of cases and 32.0 percent of controls drank ≥1 cup/day. Tea drinkers of ≥1 cup/day (table 5) had a significantly lower risk of myocardial infarction compared with tea nondrinkers (OR = 0.55, 95 percent CI 0.36–0.85), independent of coronary risk factors and lipids. Subjects drinking tea in the two middle levels of consumption had nonsignificant reductions in the risk of myocardial infarction of lower magnitude but consistent with a significant linear trend across levels of tea intake (p = 0.012).

DISCUSSION

In this study, neither caffeinated nor decaffeinated coffee was associated with the risk of myocardial infarction. For tea consumption, we found an inverse

TABLE 4. Odds ratios and 95% confidence intervals for myocardial infarction by level of decaffeinated coffee intake, Boston Area Health Study, 1982–1983

	Cases/controls (no.)	Levels of decaffeinated coffee intake				p value (trend)
		None (*n* = 325)	1–3 cups/month* (*n* = 109)	1–7 cups/week (*n* = 143)	>1 cup/day (*n* = 101)	
Full models						
Age, sex	338/340	1.0	0.94 (0.61–1.45)	0.96 (0.65–1.43)	1.34 (0.85–2.10)	0.38
Age, sex + coronary risk factors†	335/337	1.0	1.05 (0.66–1.70)	1.09 (0.71–1.69)	1.25 (0.76–2.04)	0.39
Lipid-limited models‡						
Age, sex + coronary risk factors†	298/295	1.0	0.94 (0.56–1.58)	1.07 (0.67–1.70)	1.32 (0.79–2.22)	0.34
Age, sex + coronary risk factors† + LDL/HDL ratio§	298/295	1.0	1.08 (0.62–1.85)	1.11 (0.68–1.81)	1.23 (0.71–2.13)	0.45

* One cup = 237 ml.

† Adjusted for age (10-year categories), sex, smoking status (never, former, <1 pack/day, 1–2 packs/day, >2 packs/day), history of medication for high blood pressure, type A personality, family history of myocardial infarction, diabetes, daily aspirin use, body mass index, log of physical activity index, percentage of calories from saturated fat, total caloric intake, and alcohol intake.

‡ Analysis limited to those subjects with full lipoprotein profiles.

§ LDL/HDL ratio, ratio of high density lipoprotein to low density lipoprotein cholesterol.

TABLE 5. Odds ratios and 95% confidence intervals for nonfatal myocardial infarction by level of tea intake, Boston Area Health Study, 1982–1983

	Cases/controls (no.)	Levels of tea intake				p value (trend)
		None (*n* = 155)	1–3 cups/month* (*n* = 179)	1–6 cups/week (*n* = 150)	≥1 cup/day (*n* = 192)	
Full models						
Age, sex	338/338	1.0	0.72 (0.46–1.11)	0.72 (0.46–1.13)	0.55 (0.36–0.85)	0.012
Age, sex + coronary risk factors†	335/335	1.0	0.70 (0.44–1.13)	0.77 (0.47–1.27)	0.56 (0.35–0.90)	0.032
Lipid-limited models‡						
Age, sex + coronary risk factors†	298/294	1.0	0.72 (0.44–1.20)	0.74 (0.44–1.25)	0.49 (0.29–0.83)	0.012
Age, sex + coronary risk factors† + LDL/HDL ratio§	298/294	1.0	0.73 (0.43–1.25)	0.69 (0.40–1.20)	0.55 (0.32–0.94)	0.033

* One cup = 237 ml.

† Adjusted for age (10-year categories), sex, smoking status (never, former, <1 pack/day, 1–2 packs/day, >2 packs/day), history of medication for high blood pressure, type A personality, family history of myocardial infarction, diabetes, daily aspirin use, body mass index, log of physical activity index, percentage of calories from saturated fat, total caloric intake, and alcohol intake.

‡ Analysis limited to those subjects with full lipoprotein profiles.

§ LDL/HDL ratio, ratio of high density lipoprotein to low density lipoprotein cholesterol.

association with the risk of myocardial infarction with a significant reduction among those drinking ≥1 cup/day, independent of lipid and nonlipid coronary risk factors.

Our finding of no association for caffeinated coffee consumption with the risk of myocardial infarction is consistent with some (3, 6, 9, 32–36) but not all (14, 37–41) cohort studies. On the other hand, most (4, 5, 7, 8, 11–13, 33) but not all (42, 43) case-control studies have yielded positive associations, leading to speculation that any effect of caffeinated coffee on the risk of myocardial infarction may be acute (1). Heavy caffeinated coffee drinkers have clustered coronary risk factors, making them more prone to myocardial infarction (44). We found these subjects to be heavy smokers, which strongly attenuated our results. Because caffeinated coffee drinkers consume more alcohol, the inverse association between alcohol and myocardial infarction (45) may offset any positive association between caffeinated coffee and myocardial infarction risk.

Some case-control (4, 10) and cohort (6, 8) studies suggest a lower threshold of decaffeinated coffee intake for myocardial infarction risk compared with caffeinated coffee. Other studies have found no such evidence (3, 5, 7, 9). Although we found no association between decaffeinated coffee and myocardial infarction, subjects may switch to decaffeinated coffee as a healthy alternative, as reflected in their higher rates of hypertension and diabetes.

Early case-control (11–14) studies largely found no association between tea consumption and coronary heart disease. However, a true inverse association may have been masked because of the low prevalence

of heavy tea consumption or because of potential recall, information, or selection biases. Some recent cohort studies have demonstrated an inverse association between tea and coronary heart disease death (17–20).

We offer two possible explanations for the inverse association between tea and the reduced risk of myocardial infarction. First, flavonoids in black tea may reduce myocardial infarction by inhibiting low density lipoprotein cholesterol oxidation (22), reducing platelet aggregation (46), or reducing ischemic damage (47). Increased tea consumption has been associated with little or no improvement in lipid profiles (15, 17, 47–51). We found only modest, beneficial differences in the lipid profiles of subjects preferring tea. Recent European prospective studies, where black tea has a greater contribution to total flavonoid intake, have suggested an inverse association between flavonoids and coronary heart disease mortality (18–20). Only men with preexisting coronary heart disease had an inverse association in an American cohort study (22). Alternatively, higher tea consumption may be a surrogate for a healthier lifestyle. Our data suggest that tea drinkers differ from nondrinkers in terms of health behaviors and medical conditions. Nevertheless, adjustment for neither coronary risk factors nor individual lipids changed the risk estimates.

Several important limitations of this study should be considered. First, despite varying response rates of 60–90 percent for controls among the neighborhoods of the six hospitals, we found no difference between the overall or neighborhood-specific risk estimates. Though our results may only apply to nonfatal myocardial infarction, this aspect of case selection enabled us to ascertain extensive information on coronary risk factors. Only smoking appreciably confounded our results, suggesting that residual confounding by unmeasured factors or in the smoking variable should be modest at best. Finally, misclassification of beverage intake may have occurred. Neither subjects nor interviewers were aware of any specific study hypothesis, so we expect reported usual intakes over the past year to be without recall bias or seasonality. The inability to distinguish coffee brewing methods, tea type, or tea content may also introduce misclassification. However, in this study of older men and women during the early 1980s, we expect consumption to be limited to filtered coffee and caffeinated black tea.

In summary, only tea was inversely associated with the risk of myocardial infarction. More data from prospective cohort studies will help to distinguish whether tea drinking has a true biologic effect or serves as a surrogate for a risk profile promoting lower myocardial infarction risk.

ACKNOWLEDGMENTS

The Boston Area Health Study was supported by research grants HL-24423 and HL-21006 from the National Heart, Lung, and Blood Institute. Howard D. Sesso is supported by institutional training grant HL-07575 from the National Heart, Lung, and Blood Institute.

The authors thank the six Boston area hospitals that participated in this study: Emerson Hospital (Dr. Marvin H. Kendrick), Framingham Union Hospital (Dr. Marvin Adner and Dr. Gerald Evans), Leonard Morse Hospital (Dr. L. Frederick Kaplan), Mount Auburn Hospital (Dr. Leonard Zir), Newton-Wellesley Hospital (Dr. James Sidd), and Waltham-Weston Hospital (Dr. Solomon Gabbay).

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