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Citation

Rosner, S. A., A. Akesson, M. J. Stampfer, and A. Wolk. 2006. "Coffee Consumption and Risk of Myocardial Infarction among Older Swedish Women." American Journal of Epidemiology 165 (3): 288–93. https://doi.org/10.1093/aje/kwk013.

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American Journal of Epidemiology
Vol. 165, No. 3
DOI: 10.1093/aje/kwk013
Advance Access publication November 16, 2006
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Original Contribution

Coffee Consumption and Risk of Myocardial Infarction among Older Swedish Women

Sarah A. Rosner[1], Agneta Åkesson[2], Meir J. Stampfer[1,2], and Alicja Wolk[2]

[1] Department of Epidemiology, Harvard School of Public Health, Boston, MA.
[2] Division of Nutritional Epidemiology, Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden.

Received for publication May 12, 2006; accepted for publication June 28, 2006.

Numerous studies have examined the association between coffee consumption and risk of myocardial infarction (MI), but results have been inconsistent. Case-control studies generally suggest a harmful effect of coffee drinking, whereas cohort studies have mostly shown no association. Recent studies found that coffee may lower the risk of diabetes, a major coronary risk factor. The authors prospectively examined the effect of coffee consumption on MI risk in 32,650 older Swedish women, aged 40–74 years, participating in the Swedish Mammography Cohort; 459 cases of MI developed during 165,896 person-years of follow-up from 1997 to 2002. After adjustment for age, coronary heart disease risk factors, and dietary variables, the relative risk of MI associated with drinking ≥5 cups/week versus 0–4 cups/week was 0.68 (95% confidence interval (CI): 0.43, 1.07). The authors observed a nonsignificant trend toward lower risk with higher consumption levels. Compared with that for 0–4 cups/week, the relative risks of MI were 0.84 (95% CI: 0.51, 1.38) for 5–7 cups/week, 0.65 (95% CI: 0.41, 1.03) for 2–3 cups/day, 0.64 (95% CI: 0.39, 1.04) for 4–5 cups/day, and 0.65 (95% CI: 0.37, 1.12) for ≥6 cups/day (p-trend = 0.07). Contrary to previous case-control studies, the authors concluded that coffee consumption does not increase MI risk. Coffee consumption of ≥5 cups/week was nonsignificantly inversely associated with MI risk among older Swedish women.

coffee; myocardial infarction; Sweden; women

Abbreviations: CHD, coronary heart disease; CI, confidence interval; MI, myocardial infarction.

The effect of coffee consumption on coronary heart disease (CHD) risk has been frequently studied, but the results have been inconsistent. Most case-control studies have shown a positive association between coffee consumption and risk of CHD (1–6), whereas the majority of cohort studies have found no effect (7–12). However, more recent cohort studies have shown both increased risk (11, 13) and decreased risk (10, 14) associated with coffee drinking. It is plausible that coffee drinking may influence CHD risk because coffee has antioxidant properties and improves insulin sensitivity (15–19).

We investigated the effect of coffee consumption on the risk of myocardial infarction (MI) in women by using a prospective cohort design based on the Swedish Mammography Cohort. To our knowledge, only two previous cohorts, in US populations, have studied women specifically (9, 12, 14), and none has assessed an all-female cohort in a European population. Since both the type and the method of coffee preparation vary by country and may affect CHD risk differently, results from the US cohort may not be applicable to European women (4). Because coffee is frequently consumed in Sweden, this cohort provides a wider range of coffee intake than in the previous study of women. Additionally, since decaffeinated coffee is virtually nonexistent in Sweden, we can study solely the effects of regular coffee. Our null hypothesis was that coffee consumption was not associated with the risk of MI among older Swedish women.

Correspondence to Sarah A. Rosner, Harvard School of Public Health, Department of Epidemiology, Kresge Room 911, 677 Huntington Avenue, Boston, MA 02115 (e-mail: srosner@hsph.harvard.edu).

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MATERIALS AND METHODS

Study population

The Swedish Mammography Cohort has been described previously (20). Briefly, this population-based cohort was established between 1987 and 1990, when all women residing in Uppsala and Vastmanland counties between the ages of 40 and 74 years were invited to participate in a mammography screening program. The invitation included a six-page questionnaire that sought information on diet, parity, age at first birth, self-reported height and weight, and educational level; 74 percent responded. In 1997, an expanded follow-up questionnaire (70 percent response) was sent that requested information on diet; multivitamin/supplement use; aspirin use; cigarette smoking; hormone replacement therapy use; history of diabetes, hypertension, and hypercholesterolemia; family history of CHD; and physical activity. A physical activity score was derived from the exercise and lifestyle questions, as previously described (21). Since smoking status data were not elicited on the 1987 questionnaire and smoking is an important confounder of the coffee-CHD association, we considered the return date of the follow-up questionnaire, September 1997, as the baseline.

Of the 38,984 women who responded to the 1997 questionnaire, we excluded 6,334 at baseline who had a history of MI, ischemic heart disease, stroke, or cancer; an implausible answer to any of the open-ended diet questions (any value $\geq$4 standard deviations above the mean); an implausible caloric intake ($<$500 kcal/day, $>$3,500 kcal/day), missing data on coffee consumption; or an outcome event during the first month of follow-up. The study was approved by the ethics committee at the Karolinska Institutet in Stockholm and the Uppsala University Hospital.

Assessment of diet and CHD endpoints

Diet was assessed by using a 96-item, self-administered food frequency questionnaire. The women were asked how often, on average, they consumed each type of food or beverage. Eight prespecified response categories ranged from 0/month to $\geq$3/day. Open-ended questions were used for commonly consumed items, for example, coffee, tea, milk, and bread, where the participant was asked to report the number of times per day or per week she consumed the item. Nutrient intakes were computed by multiplying the consumption frequency of each food by the nutrient content of age-specific ($<$53, 53–65, $>$65 years) portion sizes, using composition values from the Swedish National Food Administration Database (22). The food frequency questionnaire was validated by using a subsample of 129 randomly selected women from the cohort (23). Spearman correlation coefficients were calculated between the food frequency questionnaire and an average of four 1-week diet records. For coffee, the correlation coefficient was 0.61.

CHD outcomes (fatal and nonfatal MI) were assessed by linkage with the Swedish Hospital Discharge Register and the Swedish Causes of Death Registry. These registries are considered more than 99 percent complete (24, 25). The diagnostic criteria, set up by The Swedish National Board of Health and Welfare (26), were, in brief, as follows: 1) specified changes in blood levels of troponin occurring $\geq$2 times, in addition to either specified symptoms or specified electrocardiography changes such as a new pathologic Q wave or pathologic ST segment elevation or inversion; 2) specified symptoms and ST segment elevation with no further possibility of characterization; or 3) autopsy findings showing myocardial necrosis or coronary thrombosis at an age compatible with the time of disease onset. A thorough validation of the registries for the years 1987 and 1995 revealed high sensitivity (94 percent) and a high positive predictive value (86 percent) for MI in comparison to other countries (27). Dates of death were ascertained through the Swedish Death Registry at the Central Statistics Bureau in Sweden.

Statistical analysis

The participants started accruing person-time from September 15, 1997, until the date of diagnosis of MI, stroke, or cancer; the date of death; or December 31, 2002, whichever occurred first. Participants who developed cancer during the follow-up period were censored at their date of diagnosis. Cox proportional hazards models were constructed by using calendar time as the time scale to estimate age-adjusted and multivariable-adjusted rate ratios with 95 percent confidence intervals. Coffee was entered into the model by using four indicator variables for the five categories (0–4 cups/week (referent), 5–7 cups/week, 2–3 cups/day, 4–5 cups/day, $\geq$6 cups/day). These categories were chosen to be similar to those in previous studies of coffee and CHD (9, 12). Because there were so few nondrinkers (0 cups/day), we could not compare drinkers with nondrinkers by using a dichotomous variable. Instead, we constructed a dichotomous variable that compared drinkers of $\geq$5 cups/week with very light drinkers (0–4 cups/week). Additionally, an ordinal score variable representing the five coffee categories was entered into the model to conduct a test for trend.

The following variables were included in multivariable models, using missing-data indicators when necessary: age (years); smoking status (never/former smoker; 1–9, 10–19, $\geq$20 cigarettes/day; current but unknown number of cigarettes/day); total activity score (quintiles); alcohol consumption (0, 1–4.9, 5–9.9, $\geq$10 g/day); self-reported hypercholesterolemia (yes/no); self-reported hypertension (yes/no); body mass index ($<$18.5, 18.5–24.9, 25–29.9, $\geq$30 kg/m^2); family history of MI before age 60 years (yes/no); postmenopausal hormone replacement therapy use (ever/never); multivitamin use (yes/no); vitamin E supplement use (yes/no); educational level (primary school, high school, university); quartiles of fiber, folate, saturated fat, monounsaturated fat, and polyunsaturated fat; tea (0–1 cups/day, $\geq$2 cups/day); and sugar in tea or coffee (0–2 times/day, $\geq$3 times/day). All nutrient variables were adjusted for total energy intake by using the residual method (28).

Because coffee consumption is inversely associated with the development of type 2 diabetes (16–19), we conducted subgroup analysis stratifying by both diabetes status and body mass index. All analyses were conducted by using SAS v.8.2 software (SAS Institute, Inc., Cary, North Carolina).

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TABLE 1. Characteristics of the study population ($n = 32,650$) at baseline according to categories of coffee consumption, Swedish Mammography Cohort, 1997–2002*

Variable	Coffee consumption (cups)					p value†
	0–4/week ($n = 1,312$)	5–7/week ($n = 3,748$)	2–3/day ($n = 16,230$)	4–5/day ($n = 8,528$)	≥6/day ($n = 2,832$)	
Age, years	60.4 (9.1)	62.6 (9.3)	61.9 (9.2)	60.5 (8.8)	59.2 (8.3)	<0.0001
Body mass index‡	25.0 (4.4)	25.0 (4.0)	24.9 (3.8)	25.0 (3.9)	25.2 (4.0)	0.0007
Total activity score, MET§-hours/day	42.0 (4.9)	42.2 (4.8)	42.4 (4.7)	42.9 (4.8)	43.3 (5.1)	<0.0001
Current smoking (no. (%))	108 (8.5)	354 (9.8)	2,050 (13.0)	1,903 (23.1)	1,002 (37.0)	<0.0001
Hypertension (no. (%))	273 (20.8)	846 (22.6)	3,373 (20.8)	1,547 (18.1)	475 (16.8)	<0.0001
Hypercholesterolemia (no. (%))	102 (7.8)	310 (8.3)	1,236 (7.6)	656 (7.7)	215 (7.6)	0.75
Diabetes (no. (%))	48 (3.7)	146 (3.9)	581 (3.6)	272 (3.2)	92 (3.3)	0.28
Ever HRT§ use (no. (%))	705 (54.5)	2,048 (55.3)	8,274 (51.6)	4,118 (48.7)	1,285 (45.9)	<0.0001
Aspirin use (no. (%))	575 (49.0)	1,617 (50.1)	6,824 (48.9)	3,669 (49.7)	1,273 (51.5)	0.13
Family history of MI§ before age 60 years (no. (%))	189 (17.7)	488 (16.4)	2,139 (16.6)	1,143 (16.7)	425 (18.5)	0.20
Educational level (no. (%))						<0.0001
Primary school	510 (39.3)	1,731 (46.7)	8,054 (50.1)	4,516 (53.4)	1,533 (54.7)	
High school	405 (31.2)	1,157 (31.2)	4,814 (29.9)	2,485 (29.4)	848 (30.3)	
University	383 (29.5)	823 (22.2)	3,210 (20.0)	1,456 (17.2)	420 (15.0)	
Total energy, kcal/day	1,594 (465)	1,558 (471)	1,643 (453)	1,724 (470)	1,797 (517)	<0.0001
Folate, μg/day	333 (116)	325 (105)	311 (96)	297 (86)	288 (92)	<0.0001
Fiber, g/day	22.7 (6.3)	22.4 (5.5)	22.2 (5.3)	21.9 (5.3)	21.2 (5.6)	<0.0001
Saturated fat, g/day	25.7 (6.5)	26.0 (6.1)	26.3 (6.0)	26.6 (6.2)	27.4 (6.8)	<0.0001
Monounsaturated fat, g/day	18.8 (3.6)	19.0 (3.5)	19.1 (3.4)	19.3 (3.5)	19.5 (3.6)	<0.0001
Polyunsaturated fat, g/day	8.3 (2.2)	8.3 (2.1)	8.3 (2.0)	8.3 (2.1)	8.4 (2.1)	0.16
Alcohol, g/day	3.6 (5.1)	4.1 (4.8)	3.9 (4.6)	3.8 (4.5)	3.5 (4.7)	<0.0001
Tea, cups/day	1.8 (1.7)	1.3 (1.2)	1.0 (1.0)	0.8 (1.0)	0.7 (1.2)	<0.0001
Sugar in tea or coffee, times/day	1.2 (1.8)	1.3 (1.7)	1.4 (1.8)	1.8 (2.5)	2.5 (3.8)	<0.0001
Multivitamin use (no. (%))	361 (27.5)	1,001 (26.7)	3,940 (24.3)	1,910 (22.4)	585 (20.7)	<0.0001
Vitamin C supplement use (no. (%))	241 (18.4)	614 (16.4)	2,426 (15.0)	1,225 (14.4)	382 (13.5)	<0.0001
Vitamin E supplement use (no. (%))	106 (8.1)	272 (7.3)	976 (6.0)	423 (5.0)	146 (5.2)	<0.0001

* Unless otherwise specified, all values are given as mean (standard deviation).
† p value for chi-square test for categorical variables, analysis of variance for continuous variables.
‡ Weight (kg)/height $(m)^2$.
§ MET, metabolic equivalent; HRT, hormone replacement therapy; MI, myocardial infarction.

RESULTS

During 165,896 person-years of follow-up, 459 cases of MI (391 nonfatal and 68 fatal) occurred among the 32,650 women for whom coffee consumption data were available. The average follow-up time was 5.1 years. Baseline characteristics of the study population are presented in table 1. Compared with subjects who drank 0–4 cups/week, heavy-coffee consumers were more likely to be current smokers, more likely to be never users of hormone replacement therapy, less likely to take multivitamins, and less educated (table 1).

On the basis of Cox proportional hazards modeling, the relative risks of MI comparing ≥5 cups/week with 0–4 cups/week were 0.80 (95 percent confidence interval (CI): 0.52, 1.24; $p = 0.32$) in the age-adjusted model, 0.72 (95 percent CI: 0.47, 1.12; $p = 0.15$) in the age- and smoking-adjusted model, and 0.68 (95 percent CI: 0.43, 1.07; $p = 0.10$) in the full multivariate-adjusted model. The relative risks from the Cox proportional hazards model for the association between risk of MI and categories of coffee consumption are presented in table 2. In the age-adjusted analysis, the relative risks comparing drinkers of 5–7 cups/week, 2–3 cups/day, or 4–5 cups/day with drinkers of 0–4 cups/week, were less than 1, although the confidence intervals included the null value. However, drinkers of ≥6 cups/day had a risk equivalent to that for drinkers of 0–4 cups/week. Since heavy-coffee drinkers were disproportionately more likely to be smokers, the apparent U-shaped risk curve in the age-adjusted model reflected the effect of confounding by smoking. In the age- and smoking-adjusted model, relative risks decreased in the top two categories of coffee drinking, with further decreases

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TABLE 2. Cox proportional hazards model results for the association between coffee consumption and myocardial infarction, Swedish Mammography Cohort, 1997–2002

Coffee consumption (cups)	Cases	Person-years	Age adjusted*			Age and smoking adjusted†			Multivariate adjusted‡		
			RR§	95% CI§	*p* value	RR	95% CI	*p* value	RR	95% CI	*p* value
0–4/week	21	6,637	1.00 (Ref§)			1.00 (Ref)			1.00 (Ref)		
5–7/week	63	18,963	0.86	0.53, 1.42	0.56	0.83	0.51, 1.37	0.47	0.84	0.51, 1.38	0.48
2–3/day	222	82,330	0.74	0.48, 1.16	0.20	0.69	0.44, 1.08	0.11	0.65	0.41, 1.03	0.07
4–5/day	113	43,475	0.83	0.52, 1.32	0.43	0.71	0.45, 1.14	0.15	0.64	0.39, 1.04	0.07
≥6/day	40	14,491	1.02	0.60, 1.72	0.96	0.77	0.45, 1.31	0.37	0.65	0.37, 1.12	0.12
											p-trend = 0.07

* Age (years).

† Age (years), smoking status (nonsmoker; 1–9, 10–19, ≥20 cigarettes/day; current but unknown no. of cigarettes/day).

‡ Age (years), smoking status (nonsmoker; 1–9, 10–19, ≥20 cigarettes/day; current but unknown no. of cigarettes/day), total activity score (quintiles), alcohol consumption (0, 1–4.9, 5–9.9, ≥10 g/day), diabetes (yes/no), hypercholesterolemia (yes/no), hypertension (yes/no), body mass index (weight (kg/height (m)^2): <18.5, 18.5–24.9, 25–29.9, ≥30), family history of myocardial infarction before age 60 years (yes/no), hormone replacement therapy use (ever/never), multivitamin use (yes/no), vitamin E supplement use (yes/no), educational level (primary school, high school, university), tea (≥2 cups/day vs. <2 cups/day), sugar in tea or coffee (≥3 vs. <3 times/day), and quartiles of energy-adjusted folate, fiber, saturated fat, monounsaturated fat, and polyunsaturated fat.

§ RR, relative risk; CI, confidence interval; Ref, referent.

in the full multivariate model (table 2). When the multivariate analysis was restricted to nonfatal MI cases ($n = 391$), the relative risks of MI were 1.00 (95 percent CI: 0.56, 1.78) for 5–7 cups/week, 0.75 (95 percent CI: 0.44, 1.29) for 2–3 cups/day, 0.78 (95 percent CI: 0.45, 1.37) for 4–5 cups/day, and 0.74 (95 percent CI: 0.40, 1.40) for ≥6 cups/day (p-trend = 0.21).

The relative risk of MI seemed to become closer to the null in the highest category of coffee consumption (≥6 cups/day) in the age- and smoking-adjusted model. To address potential residual confounding by smoking, we conducted a further analysis restricted to nonsmokers (320 cases; 133,014 person-years of follow-up). In the full multivariate model restricted to nonsmokers, the relative risks of MI were 0.90 (95 percent CI: 0.52, 1.56) for 5–7 cups/week, 0.62 (95 percent CI: 0.37, 1.03) for 2–3 cups/day, 0.58 (95 percent CI: 0.34, 1.01) for 4–5 cups/day, and 0.80 (95 percent CI: 0.42, 1.52) for ≥6 cups/day compared with 0–4 cups/week (p-trend = 0.10). These results are similar to those in the full cohort.

We assessed a further Cox proportional hazards model stratified by diabetes status. For nondiabetics (400 MI cases; 160,472 person-years), the results were very similar to those found for the full cohort. The relative risks of MI for nondiabetics were 0.71 (95 percent CI: 0.42, 1.20) for 5–7 cups/week, 0.57 (95 percent CI: 0.35, 0.91) for 2–3 cups/day, 0.57 (95 percent CI: 0.35, 0.94) for 4–5 cups/day, and 0.55 (95 percent CI: 0.31, 0.98) for ≥6 cups/day compared with 0–4 cups/week. The small number of diabetics (59 MI cases; 5,424 person-years) in the cohort, with only a single diabetic case in the referent group of 0–4 cups/week, precluded calculation of meaningful relative risks for the diabetic subgroup.

In models stratified by body mass index, for those of normal weight (body mass index = 18.5–24.9 kg/m^2; 225 cases), the relative risks were 0.88 (95 percent CI: 0.41, 1.89) for 5–7 cups/week, 0.77 (95 percent CI: 0.38, 1.56) for 2–3 cups/day, 0.79 (95 percent CI: 0.38, 1.63) for 4–5 cups/day, and 0.56 (95 percent CI: 0.24, 1.34) for ≥6 cups/day. For those whose body mass index was ≥25 kg/m^2 (210 cases), the relative risks were 0.68 (95 percent CI: 0.34, 1.37) for 5–7 cups/week, 0.49 (95 percent CI: 0.26, 0.94) for 2–3 cups/day, 0.50 (95 percent CI: 0.25, 0.98) for 4–5 cups/day, and 0.64 (95 percent CI: 0.30, 1.37) for ≥6 cups/day.

DISCUSSION

In this study, we found that women who drank ≥5 cups/week had approximately a 30 percent lower risk of MI compared with women who drank only 0–4 cups/week, although this result was not statistically significant. Overall, we observed a suggestion of a threshold association, such that increasing coffee consumption, up to 2–3 cups/day, was associated with a decreasing risk of MI, but there was no further decrease with greater intake.

Coffee has been frequently studied as a possible risk factor for MI, but findings have been inconsistent. Most case-control studies have reported an increased risk of MI associated with coffee drinking. In 1993, Greenland (29) published a meta-analysis that found a summary relative risk of 1.42 (95 percent CI: 1.30, 1.55) for 5 cups/day versus none for eight case-control studies. A second meta-analysis by Kawachi et al. (7) found a summary relative risk of 1.63 (95 percent CI: 1.50, 1.78) comparing 5 cups/day with none based on eight case-control studies. Since these meta-analyses were published, more recent case-control studies have continued to report an increased risk associated with coffee drinking (1, 3, 6, 30). However, the results from three case-control studies have suggested an inverse association between coffee and risk of CHD. No significant association for coffee drinking (relative risk = 0.84, 95 percent CI: 0.49, 1.42) was found in a US population (31). In the only known Swedish case-control study, compared with women drinking about 1–2 cups per day,

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those drinking about 3–6 cups per day had a nonsignificantly decreased risk of CHD (relative risk = 0.74, 95 percent CI: 0.54, 1.02) (4). Consumption of more than about 6 cups/day was associated with a nonsignificant increased risk (relative risk = 1.43, 95 percent CI: 0.81, 2.54). This J-shaped relation between coffee consumption and risk of CHD was also observed in a Greek case-control study in which "moderate" intake (about 1–2 cups/day) was associated with a decreased risk (relative risk = 0.69, 95 percent CI: 0.50, 0.86), whereas "heavy" (about 2–4 cups/day) or "very heavy" (>6 cups/day) coffee intake increased risk of CHD (5).

Although most case-control studies have suggested that coffee consumption is harmful, cohort studies typically have not, suggesting possible selection or recall bias in the retrospective studies. In a meta-analysis of 15 cohort studies, the overall relative risk was 1.05 (95 percent CI: 0.99, 1.12) when consumption of 5 cups/day was compared with none (7). A second meta-analysis of 11 cohorts found a summary relative risk of 1.01 (95 percent CI: 0.90, 1.12) for 4–6 cups/day versus none (8). A third meta-analysis found that the summary relative risk for cohort studies varied by publication date (29). The summary relative risk for the five cohort studies published before 1981 was 0.92, while the summary relative risk for nine cohort studies published after 1986 was 1.27 (29). It was hypothesized that this difference could be due to publication bias, misclassification of coffee consumption, or residual confounding. Since these meta-analyses were published, in the early 1990s, more recent cohort studies have continued to find no relation between coffee consumption and risk of MI (9–12). Neither the Nurses' Health Study, an all-female cohort, nor the Health Professionals Follow-up Study, an all-male cohort, found any association with coffee consumption of up to 6 cups/day (9, 12). The Iowa Women's Health Study, a cohort of postmenopausal women, found an inverse association between moderate coffee consumption (1–3 cups/day) and risk of cardiovascular mortality but no association for heavy consumption (≥6 cups/day) (14).

Contrary to the majority of previous cohort and case-control studies, we found a nonsignificant inverse association between coffee drinking and risk of MI for up to ≥6 cups/day. Several explanations are possible for the discrepancy between our results and those of other studies, particularly the cohort studies. First, it is possible that there are differences in the type or method of preparation of coffee in the Swedish population. Scandinavian-style boiled coffee contains up to 100 times more kahweol and up to 80 times more cafestol than filtered coffee (32). These two hypercholesterolemic lipophilic diterpenes are retained in the filter (33). The vast majority of the women in our cohort likely consumed filtered coffee; a previous study revealed that 6.6 percent of Swedish women consumed boiled coffee, 5.7 percent consumed both filtered and boiled coffee, and the remainder drank filtered coffee (4). Our study findings were consistent with the results from the only other known study conducted in Sweden, although that study used a case-control design (4). Second, our duration of follow-up of 5 years is considerably shorter than that for the other cohorts, which tended to have at least 10 years of follow-up. It is possible that short-term effects of coffee consumption are associated with decreased risk but that there is no effect over longer periods of time. It is also possible that with increasing follow-up time, there may be more misclassification of coffee consumption. Finally, the possibility that our results are due to unmeasured confounding or random chance cannot be ruled out.

There are several plausible biologic mechanisms through which coffee may reduce risk of MI. Coffee contains phenolic compounds, which are known antioxidants and may reduce oxidative stress (15). Additionally, coffee has been shown to improve insulin sensitivity and may protect against type 2 diabetes (15–19). In our study, the most strongly suggestive findings for a reduction in risk of MI were for those women without diabetes and with a higher body mass index.

Our study has several strengths. First, we used a prospective cohort design that is not subject to the problems of recall and selection bias in case-control studies. Second, we had a large sample size of nearly 33,000 women with a wide range of coffee intake and complete follow-up for CHD events through linkage with the national health-care system. Finally, we had a fairly homogeneous cohort, which allows for high internal validity.

A major limitation of our study is that we were able to use only a single measurement of coffee consumption. However, the validity of the food frequency questionnaire for coffee was demonstrated (23). Several of the covariates in our analysis, for example, hypercholesterolemia and hypertension status, were self-reported and thus could result in incomplete control of confounding because of misclassification. Additionally, the near absence of nondrinkers in the cohort made it impossible to assess the effect of any coffee consumption versus none. Our referent group of 0–4 cups/week included only 4 percent of the total person-time in our study. However, on the basis of the baseline characteristics presented in table 1, the referent group appears to be comparable to, or even somewhat healthier than, the other coffee consumption groups.

In conclusion, we found that coffee consumption of ≥5 cups/week was nonsignificantly inversely associated with MI among older Swedish women. Although there was some suggestion of a decreased risk of MI, the data are best interpreted as strong evidence ruling out any material increase in risk.

ACKNOWLEDGMENTS

Conflict of interest: none declared.

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