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Coffee Consumption and Risk of Heart Failure in Men: an Analysis from the Cohort of Swedish Men

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Abstract

Background—A previous study found that consuming 5 or more cups of coffee per day was associated with increased incidence of heart failure (HF). We sought to evaluate this association in a larger population.

Methods—We measured coffee consumption using food-frequency questionnaires among 37,315 men without history of myocardial infarction, diabetes, or HF. They were followed for HF hospitalization or mortality from January 1, 1998 until December 31, 2006 using record linkage to the Swedish inpatient and cause-of-death registries. Cox proportional hazards models adjusted for age, dietary, and demographic factors were used to calculate incidence rate ratios (RR) and 95% confidence intervals (CI).

Results—Over 9 years of follow-up, 784 men experienced a HF event. Compared to men who drank ≤ 1 cup of coffee per day (unadjusted rate 29.9 HF events/10,000 person-years), RR were 0.87 (95% CI 0.69–1.11, unadjusted rate 29.2/10,000 person-years,) for 2 cups/day, 0.89 (95% CI 0.70–1.14, unadjusted rate 25.1/10,000 person-years) for 3 cups/day, 0.89 (95% CI 0.69–1.15, unadjusted rate 25.0/10,000 person-years) for 4 cups/day, and 0.89 (95% CI 0.69–1.15, unadjusted rate 18.1/10,000 person-years) for ≥ 5 cups/day (p for trend in RR = 0.61).

Conclusions—This study did not support the hypothesis that high coffee consumption is associated with increased rates of HF hospitalization or mortality.

Heart failure (HF) is the leading cause of hospitalization in people over 65 years of age in the United States. ¹ As the average age of the population increases, and people survive longer after myocardial infarction (MI), the incidence of HF will undoubtedly increase. ² However, despite improved treatment regimens, mortality from HF still remains high with approximately one quarter of patients dying within one year and half within five years of diagnosis. ^{3, 4} Thus, lifestyle approaches to prevent the occurrence of HF have the potential to reduce the burden of this life-threatening syndrome.

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Based on one prior study,⁵ a recent statement by the American Heart Association on the prevention of HF noted that high coffee consumption may increase the risk of HF, but its independent effect remains to be confirmed.⁶ Although coffee consumption is associated with hypertension,^{7–11} its effects on coronary heart disease are less clear.^{12–18} Early reports raised concerns about adverse cardiovascular effects of coffee consumption, but a recent study reported that high coffee consumption may be associated with a decreased risk of MI.¹⁸ In addition coffee consumption has been associated with lower risk of type 2 diabetes mellitus.^{19–21} Increases in blood pressure and decreases in the risk of type 2 diabetes mellitus and coronary heart disease would be expected to have opposing effects, suggesting that coffee consumption could decrease or increase the risk of HF.

Given the uncertainty of the effect of coffee consumption on HF incidence, we sought to investigate its effects and either confirm or refute the findings of the previously published study suggesting that coffee consumption is a risk factor for the occurrence of HF.

METHODS

Participants were drawn from the Cohort of Swedish Men, a prospective cohort of 48,850 men aged 45–79 years old who resided in Västmanland or Örebro counties in Sweden.²² Men residing in those counties received a questionnaire in 1997 requesting information on demographic, lifestyle, and behavioral factors, including dietary intake. The dietary information was collected using a 96-item, validated, food frequency questionnaire.²³

We excluded subjects with an incorrect or incomplete national registration number, as well as those who returned blank questionnaires, who reported improbable energy intake defined as more than three standard deviations from the mean value of the natural logarithm-transformed energy intake, or who had a previous history of cancer ($n = 3,507$), those with a prior history of HF ($n = 743$) and those with missing data for coffee consumption ($n = 2,304$). In the primary analysis we additionally excluded men with a history of MI or diabetes at baseline ($n = 4,981$), because these men were likely to have received dietary counseling which could affect their diet and their reporting of diet, leaving 37,315 participants available for this analysis. The study was approved by the Regional Ethical Review Board at the Karolinska Institute.

Participants were asked to report average intake of coffee over the last year in cups/day or cups/week. In a validation study of the food-frequency questionnaire among women from central Sweden, the Spearman correlation coefficient between the food-frequency questionnaire and an average of four one-week diet records was 0.63 for coffee.²⁴ Following the previous report, coffee consumption was classified as < 5 cups/day and ≥ 5 cups per day.⁵ To further explore the association, we classified coffee consumption as ≤ 1 cup/day, 2 cups/day, 3 cups/day, 4 cups/day and ≥ 5 cups/day.

Study participants were followed from January 1, 1998 to the date of first admission for HF, death from HF or other causes, or the end of the study follow-up period (December 31, 2006), whichever came first. The primary endpoint was defined as a composite of hospitalization for HF as a primary diagnosis (as determined through the Swedish Inpatient Registry records) or mortality from HF as the primary cause (as determined by the Swedish National Death Register records) identified by the ICD-9 code 428 and the ICD-10 codes I50 or I11.0. A secondary outcome included hospitalizations and deaths with HF listed as either a primary or secondary diagnosis. The Swedish inpatient register was validated in a previous study, and 95% of the patients with these ICD codes as the primary diagnosis were confirmed to have a diagnosis of HF by medical record review, as defined by European Society of Cardiology criteria.²⁵ HF was confirmed in 82% of patients with primary or secondary diagnosis of HF.²⁶ In the case of multiple admissions for HF, only the first admission was used.

We graphed age-adjusted survival probabilities using Kaplan-Meier plots. Cox proportional hazards models were used to estimate incidence rate ratios (RR) and their corresponding 95% confidence intervals (CI). In all models, age was accounted for by allowing the baseline incidence rate of HF to vary by age. We adjusted for body mass index (BMI, kg/m²), total activity score (MET-hour/day), smoking (current, past, or never), history of high cholesterol (yes or no), family history of MI before age 60 (yes or no), education level (less than high school, high school, or university), marital status (married, single, divorced, or widowed), aspirin use (yes or no), alcohol (g/day), tea (serving/day), energy-adjusted fat intake (saturated, monounsaturated and polyunsaturated), and energy-adjusted daily sodium intake. Covariates were chosen based on their relationships to coffee consumption and HF incidence in our data and in the literature. In the primary analysis, we did not adjust for self-reported hypertension. Coffee consumption can increase blood pressure, an established risk factor for HF; adjusting for hypertension could result in an underestimate of the potential harm associated with coffee consumption. We chose to use linear terms for all continuous covariates based on an algorithm that compared linear terms to fractional polynomials of the form $X^p + X^q$ where p and q are chosen from the set -2, -1, -0.5, natural logarithm, 0.5, 1, 2, 3 based on the best fit to the data;²⁷ the fractional polynomials did not significantly improve the model fit. The variables included in the models were not highly correlated with each other (Pearson correlation < 0.45 for all variable pairs). We used indicator variables when there was missing data for covariates. Assuming that the rate of HF hospitalization in this population will be similar to national rate for Swedish men in 2000, 23.7 cases per 10,000 person-years,²⁸ and setting the significance level at 0.05, we estimated that we would have 75% power to detect a RR of 1.25 and 86% power to detect a RR of 1.30 comparing men who consumed ≥ 5 cups of coffee/day to those who consumed < 5 cups/day. A test for linear trend across categories of coffee consumption was performed by entering the value for the median intake in each category and then modeling coffee consumption as a continuous variable. We examined whether including coffee consumption added information to the model by conducting a 4 degree of freedom likelihood ratio test. We tested whether the association between coffee consumption and HF varied by body mass index (< 25 kg/m² vs ≥ 25 kg/m²), cigarette smoking (current smoker vs never or past smoker), and alcohol intake (≥ 10 g/day vs < 10 g/day).

In a sensitivity analysis, we excluded subjects who developed HF during the first two years of follow-up because of the possibility that symptoms of unrecognized HF at baseline may affect coffee consumption. In additional sensitivity analyses, we did not adjust for tea consumption, excluded men who did not consume any coffee (n = 408) from the lowest exposure group because these individuals may represent a sicker subgroup of the population, and controlled for self-reported hypertension at baseline and incident MI during follow-up, potential mediators of the association between coffee consumption and HF. To address the concern that men who consumed the most coffee may be more likely to die from causes other than HF, obscuring an association between coffee and HF, we examined the association between coffee consumption and all-cause mortality. We also examined the association between coffee consumption and HF events among men with diabetes or history of MI at baseline. We tested for violation of the proportional hazards assumption by entering the product of coffee consumption and the natural logarithm of time into the model; we did not find evidence for deviation from proportionality. All analyses were conducted using SAS 9.1 (Cary, NC). We considered 2-sided p-values < 0.05 to be statistically significant.

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RESULTS

Over 9 years of follow-up of 37,315 men without known HF, diabetes, or MI at baseline, 690 men were hospitalized for HF as a primary diagnosis and 94 men died of HF as the primary cause, corresponding to a rate of 24.5 cases per 10,000 person-years. Compared to men who drank ≤ 1 cup of coffee per day, men who drank the most coffee were, on average, younger, more likely to have a less than high school education, and more likely to be a current smoker (Table 1). They also consumed less tea.

Survival free of HF is shown in the Figure. Men who consumed 5 or more cups did not have significantly increased rates of HF in age-adjusted models (RR = 1.08, 95% CI 0.90–1.28) or multivariable-adjusted models (RR = 0.99, 95% CI 0.82–1.18) compared to men who consumed less than 5 cups of coffee per day. We did not find evidence for an association when coffee consumption was considered as a multiple level exposure (Table 2). Overall, coffee consumption was not a significant predictor of HF events (likelihood ratio test $p = 0.86$). We did not find evidence for variation of the association between coffee consumption and HF by overweight status, current smoking, or alcohol intake. Excluding the first 2 years of follow-up and leaving tea consumption out of the multivariable-adjusted models did not materially change the results. The associations were not altered when men who did not drink any coffee were excluded from the analysis and when we controlled for self-reported hypertension at baseline or incident MI during follow-up.

There were 1,678 men who had events with HF as a primary or secondary diagnosis. Compared to men who drank ≤ 1 cup/day, the RR of events with HF as a primary or secondary diagnosis were 0.82 (0.70–0.97) for men who drank 2 cups/day, 0.83 (95% CI 0.70–0.98) for men who drank 3 cups/day, 0.88 (95% CI 0.74–1.05) for men who drank 4 cups/day, and 0.92 (95% CI 0.77–1.09) for men who drank ≥ 5 cups/day (p for linear trend = 0.81). Coffee consumption was not associated with an increase in the rate of all-cause mortality (compared ≤ 1 cup/day, RR for 2 cups/day = 0.86, 95% CI 0.77–0.96, RR for 3 cups/day = 0.83, 95% CI 0.74–0.92, RR for 4 cups/day = 0.84, 0.74–0.94, RR for ≥ 5 cups/day = 0.87, 95% CI 0.78–0.98, p for linear trend = 0.17).

Among the 4,981 men with a history of diabetes or MI at baseline, there were 438 incident HF events. We found no association between coffee consumption and HF in these men (multivariable-adjusted RR comparing ≥ 5 cups of coffee per day versus ≤ 1 cup per day = 0.96, 95% CI 0.69–1.33, p for linear trend = 0.95).

DISCUSSION

In this population, coffee consumption was not significantly associated with incidence of HF; however, we could not rule out a small increase in rate of HF events. This is in contrast to the study by Wilhelmsen and colleagues who found an odds ratio for HF of 1.17 (95% CI 1.05–1.30) comparing men who consumed 5 or more cups of coffee per day to those who consumed less than 5 cups of coffee per day.⁵ One major difference between the studies is the use of logistic regression in the previous study which does not account for the variable length of follow-up because of loss to follow-up or death from other causes.

While coffee has been shown to be associated with a small increase in the risk of hypertension,^{7–11} it has also been shown to be inversely associated with type 2 diabetes.^{21, 29} Results of studies that have looked at the relationship between coffee and cardiovascular disease events have been mixed.^{13, 17, 18, 30–32} In meta-analyses, case-control studies of the association between heavy coffee consumption and cardiovascular disease have shown positive associations, but cohort studies have shown no association overall.^{13, 17, 23, 24} Because of the potential for differential recall of coffee consumption between healthy controls and patients

with a recent cardiovascular event in case-control studies, the prospective cohort studies may be more reliable. A recent prospective cohort study has suggested that regular coffee consumption may have a protective effect on all-cause mortality and mortality from cardiovascular disease in particular.³³

Coffee is a complex mixture that contains a number of compounds that could affect cardiovascular health including caffeine, polyphenols, and diterpenes.³⁴ Caffeine inhibits adenosine receptors, and thus prevents adenosine's negative inotropic effect³⁵. Caffeine also facilitates the release of norepinephrine from sympathetic nerve endings, and thus causes an increase in sympathetic activity³⁶. In addition to increasing the sensitivity of the myofilaments to calcium³⁷, the level of intracellular calcium is increased by caffeine by two mechanisms, depending on dose: at low doses it causes the release of calcium from the sarcoplasmic reticulum, and at high doses it inhibits calcium reuptake into the sarcoplasmic reticulum; both results in high calcium levels and consequent inotropic effects³⁸. The polyphenols in coffee may have beneficial antioxidant effects, and the diterpenes in unfiltered coffee can increase LDL cholesterol.^{34, 39}

There are several important limitations of our study. Information regarding exposure to coffee and important covariates was obtained by self-report and is subject to misclassification. We did not have information on medications. We did not have information on the type of HF these patients were diagnosed with; it is possible that coffee has a differential effect on HF with impaired or preserved systolic function, or HF of an ischemic versus nonischemic etiology. This cohort was drawn from the Swedish population. In Sweden, almost all coffee is caffeinated.²² The results of this study may therefore not be relevant to decaffeinated coffee drinkers. The questionnaires did not differentiate between caffeinated and noncaffeinated sodas so we were unable to measure total caffeine intake. In addition, it is possible the results may have limited generalizability to other population groups or countries with different coffee consumption habits.

In Sweden, all hospitalizations are recorded in the Swedish Inpatient Registry and deaths are recorded in the Swedish National Death Register. Events such as hospitalizations for HF and deaths from HF can be examined by using record linkage to these datasets, which allows for a nearly complete assessment of outcomes, though HF that did not result in hospitalization was not ascertained. The rate of events with HF listed as a primary diagnosis in this population, 24.5 HF events per 10,000 person-years, was similar to the overall rate of primary HF hospitalization in Sweden, 23.7 per 10,000 men per year in 2000.²⁸ Other strengths of this study include the large number of study men enrolled and the prospective nature of the study design.

In summary, this study did not support the hypothesis that high coffee consumption is associated with increased rates of HF hospitalization or mortality; however, we can not rule out a small adverse effect of coffee. The results suggest that caution should be taken before declaring coffee consumption to be a risk factor for incident HF or advising those at risk of HF to reduce coffee consumption.

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figure.

Survival free of heart failure by coffee consumption, adjusted for age.

Table 1
Baseline characteristics* of 37,315 men in the Cohort of Swedish Men by coffee consumption

	Coffee consumption, cups/day				
	≤ 1	2	3	4	≥ 5
N	4,262	7,751	8,499	6,582	10,221
Age, yr	60.4 (9.6)	61.1 (9.7)	59.6 (9.5)	59.4 (9.5)	56.9 (8.7)
Body mass index (kg/m ²)	25.6 (3.4)	25.6 (3.2)	25.6 (3.1)	25.6 (3.0)	25.8 (3.2)
Physical activity (MET-hour/day)	41.6 (4.4)	41.7 (4.2)	41.7 (4.2)	41.9 (4.3)	42.4 (4.7)
Education (%)					
Less than high school	63.7	66.2	66.5	69.6	71.6
High School	15.3	15.2	14.4	13.9	14.0
University	21.0	18.6	19.1	16.4	14.3
Marital status (%)					
Single	8.1	6.2	5.7	6.3	6.7
Married	79.3	82.9	85.2	84.7	83.4
Divorced	8.0	6.5	6.0	6.4	7.4
Widowed	4.6	4.4	3.2	2.6	2.5
Cigarette smoking (%)					
Current	16.4	18.5	21.7	24.3	36.7
Past	36.9	38.1	39.0	40.5	37.7
Never	46.7	43.4	39.3	35.3	25.6
High cholesterol (%)	11.4	11.6	12.2	11.1	12.7
Family history of MI at < 60 years (%)	15.6	14.5	16.1	15.6	17.5
Aspirin use (%)	32.6	32.8	32.2	31.7	32.3
Alcohol, g/day	10.7 (10.9)	10.8 (10.3)	10.9 (9.8)	10.6 (9.8)	10.5 (10.4)
Tea, servings/day	1.2 (1.6)	0.7 (1.1)	0.5 (0.9)	0.4 (0.9)	0.3 (0.8)
Saturated fat, g/day [†]	33.3 (7.9)	33.5 (7.4)	33.7 (7.4)	34.0 (7.5)	34.5 (8.1)
Monounsaturated fat, g/day [†]	25.1 (4.6)	25.3 (4.1)	25.4 (4.1)	25.5 (4.1)	25.7 (4.3)
Polysaturated fat, g/day [†]	10.0 (2.3)	9.9 (2.1)	10.0 (2.0)	9.9 (2.0)	9.9 (2.0)
Sodium, mg/day [†]	3233 (501)	3215 (438)	3219 (429)	3195 (414)	3210 (429)

MI = myocardial infarction
* Mean (standard deviation) or percent
† Adjusted for energy using the residuals method

Table 2
Coffee consumption and incidence of heart failure hospitalization or mortality

	Coffee consumption (cups/day)					P for trend
	≤ 1	2	3	4	≥ 5	
Cases	108	192	183	141	160	
Person-years	36,162	65,806	72,844	56,455	88,590	
Unadjusted rate (cases/10,000 person- years)	29.9	29.2	25.1	25.0	18.1	
RR (95% CI) *	1 (Reference)	0.88 (0.69–1.11)	0.91 (0.72–1.16)	0.92 (0.71–1.18)	0.99 (0.77–1.26)	0.67
RR (95% CI) †	1 (Reference)	0.87 (0.69–1.11)	0.89 (0.70–1.14)	0.89 (0.69–1.15)	0.89 (0.69–1.15)	0.61

RR = incidence rate ratio; CI = confidence interval

* Cox proportional hazards model accounting for age.

† Additionally adjusted for body mass index (BMI, kg/m²), total activity score (MET-hour/day), smoking (current, past, or never), history of high cholesterol (yes or no), family history of MI before age 60 (yes or no), education level (less than high school, high school, or university), marital status (married, single, divorced, or widowed), aspirin use (yes or no), alcohol (g/day), tea (serving/day), energy-adjusted fat intake (saturated, monounsaturated and polyunsaturated), and energy-adjusted daily sodium intake.