Original Article

Coffee consumption and coronary heart disease risk using the Framingham risk score

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Background and Objectives: Although concerns regarding the influence of coffee consumption on human health have accompanied the massive increase in coffee consumption, the effects of coffee intake on the risk for coronary heart disease (CHD) remain controversial. Therefore, we evaluated the association between coffee consumption and CHD risk as estimated using the Framingham risk model in Korean adults. **Methods and Study Design:** This cross-sectional study involved 3,987 participants aged 30-74 years who participated in the fifth Korea National Health and Nutrition Examination Survey conducted in 2010. The frequency of coffee consumption was self-reported and classified into 4 categories (non-drinker, 1, 2, and ≥3 cups/day). The 10-year risk for CHD was determined from the Framingham risk score. **Results:** Across the levels of coffee consumption, there were significant differences in the frequency of smoking among men and advanced age, low high-density lipoprotein cholesterol level, diabetes, and smoking among women. In the multiple logistic regression analyses, the adjusted odds ratios (95% CI) for ≥20% 10-year CHD risk was 0.211 (0.060-0.745) for women who consumed ≥3 cups of coffee per day compared with women who consumed <1 cup per day. For women, a significant dose-response inverse association between the level of coffee consumption and 10-year CHD risk persisted even after adjusting for multiple confounding factors. For the men, however, there was no significant association between coffee consumption and 10-year CHD risk. **Conclusions:** Coffee consumption is associated with a lower risk for CHD in Korean women.

Key Words: coffee, coronary heart disease, risk, KNHANES, Framingham risk score

INTRODUCTION

Coffee is one of the most commonly consumed beverages in the world. According to nationally representative data for South Korea, approximately 90% of Korean adults drink coffee, and 65% drink coffee daily.[1] In Korea, the influence of coffee consumption on health is of enormous interest because of the high consumption of this beverage by the Korean population.

Coffee is a complex chemical mixture that reportedly contains more than a thousand different compounds, some of which have been linked to both positive and negative effects on human health.[2] The results of some studies suggest that coffee consumption may help reduce the risk for several chronic diseases, including diabetes,[3,4] Parkinson's disease,[5] and liver disease.[6,7] However, the effect of coffee consumption on coronary heart disease (CHD) risk is still debatable. Some studies have reported a positive association between coffee consumption and risk for CHD,[8-10] whereas others have suggested that there is no association or even an inverse association.[11,12] Although coffee consumption has been extensively studied in relation to CHD risk, the results of these studies have been inconclusive.[2,13,14]

CHD is one of the leading causes of mortality in Korea. The mortality rate attributable to CHD has continuously decreased in some developed countries,[15,16] but the mortality rate attributable to CHD has consistently increased in Korea.[17] The influence of coffee consumption on CHD risk in the Korean population could have considerable public health and clinical implications, as coffee consumption in Korea continues to increase at a rapid rate. However, most studies on the link between coffee consumption and CHD risk have been conducted in Western countries, and only a few studies have been undertaken in Asian countries including Korea. Therefore, we conducted a study to evaluate the association between coffee consumption and CHD risk as estimated using the Framingham risk score in Korean adults.

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METHODS

Data source and study participants

The present study is based on data acquired from the fifth Korea National Health and Nutrition Examination Survey conducted in 2010 (KNHANES V-1). The KNHANES has been conducted periodically since 1998 to assess the health and nutritional status of the civilian non-institutionalized population of Korea. The KNHANES V was a cross-sectional and nationally representative survey conducted by the Korea Centers for Disease Control and Prevention (KCDC) from 2010 to 2012. The survey was performed using a rolling sampling design, and the target population comprised non-institutionalized Korean civilians aged ≥1 year. The sampling frame was constructed from 2009 resident registration data for non-apartment residents and the 2008 bank apartment database for apartment residents. A stratified, multistage probability sampling design was used for the selection of household units. In the KNHANES V, there were 201,677 primary sampling units produced by proportional allocation, each containing approximately 60-80 households. For KNHANES V-1, 192 sampling frames were randomly selected from primary sampling units, and approximately 20 households were selected from each sampling frame using a systematic sampling method. Ultimately, 10,938 individuals from 3,840 households were selected, and 8,958 participated in the surveys (overall participation rate: 81.9%). Of the 5,416 participants aged 30-74, those with previous diagnoses of stroke or ischemic heart disease (n=220) and women who were pregnant (n=27) were excluded. The individuals with incomplete data for coffee consumption or test results were also excluded (n=1,182). A total of 3,987 participants were included in the final statistical analysis. All participants in this survey signed an informed consent form, and the KNHANES was reviewed and approved by the KCDC institutional review board (2010-02CON-21-C).

Study measurements

The survey consists of a health interview and a health examination. The survey collected data via face-to-face interviews inside the households and via direct standardized physical examinations conducted in specially equipped mobile examination centers. The health survey was conducted in the following sequence: intake, receipt of informed consent, blood pressure (BP) measurement, anthropometric measurement, blood sampling, and completion of the questionnaire. A standardized questionnaire was prepared to collect data for age, sex, socioeconomic characteristics, medical history and current drug use, family history of CHD, smoking status, alcohol consumption, and other lifestyle risk factors.

Menopause was defined as amenorrhea for 12 months following the final menstrual period.[18] In our study, a postmenopausal woman was defined as a woman with natural menopause whose current age was ≥1 year than her age at the time of her final menstrual period or a woman who underwent bilateral oophorectomy. Household equivalent income was calculated by summing the monthly income of all household members and dividing this sum by the square root of the household size. The participants were divided into 5 quintile groups by household equivalent income, and the equivalent income level (monthly household income/square root of family size) was assorted into 3 groups (lowest quintile, low; highest quintile, high; others, middle group).

The participants were classified as current smokers if they smoked currently and non-smokers if they had never smoked or had smoked previously but had quit. High-risk alcohol consumption was defined as ≥7 drinks per day for men and ≥5 drinks per day for women, at least twice per week. Regular walking was defined as walking at least 5 times per week for ≥30 min per session. Sleep time was self-reported and assessed using the question, "How many hours do you usually sleep every day?" The answer was collapsed to form categories of ≤6 h, 7-8 h, and ≥9 h. Mental health was assessed according to the presence of stress and depressive mood. To evaluate these factors, respondents were asked whether they perceived themselves to be experiencing more stress than they normally did, and whether they had been sad or hopeless enough to disrupt their daily lives for more than 2 consecutive weeks within the past year. A family history of CHD was defined as having at least one parent or sibling diagnosed with CHD.

BP was measured 3 times at 5-min intervals using a standard mercury sphygmomanometer (Baumanometer®, WA Baum Co., Inc., Copiague, NY, USA). The average of the second and third measurements was used as the final BP. Anthropometric data, including height, body weight, and waist circumference, were measured according to standardized guidelines. Body mass index was calculated by dividing the body weight by the height squared (kg/m^2). General obesity was defined as a body mass index ≥25 kg/m^2. Abdominal obesity was defined on the basis of ethnic-specific waist circumference cut-off values (≥90 cm for men and ≥85 cm for women).[19] Fasting plasma glucose, total cholesterol (T-C), and high-density lipoprotein cholesterol (HDL-C) were measured after a fasting period of at least 8 h using an autoanalyzer (Hitachi Automatic Analyzer 7600®, Hitachi, Tokyo, Japan).

Similar to the U.S. NHANES, KNHANES also collects detailed dietary information on all foods and beverages consumed in the previous 24 h (24-h recall) through in-person interviews conducted by trained dietary staff. The participants were asked how many cups of coffee they drank during a day, week, or month. On the questionnaire, the frequency of coffee consumption was classified into the following categories: rarely, 6-11 cups/year, 1 cup/month, 2-3 cups/month, 1 cup/week, 2-3 cups/week, 4-6 cups/week, 1 cup/day, 2 cups/day, and ≥3 cups/day. The frequency of coffee consumption was divided into 4 groups (non-drinker, 1 cup/day, 2 cups/day, and ≥3 cups/day) because of the small differences between the individual participants and the excessively small samples. Similar questions were asked about tea and soda. Tea and soda consumption were grouped into two categories (non-drinker and daily drinker) for data analyses to avoid categories with small numbers.

To determine the 10-year risk for CHD, we used the Framingham risk model. Using the Framingham algorithm outlined by Wilson et al,[20] a global risk score was calculated on the basis of the categorical values for age, sex, T-C, HDL-C, BP, smoking, and diabetes. Diabetes

was defined as a fasting plasma glucose level of ≥126 mg/dL or current treatment with insulin or oral hypoglycemic agents.

Statistical analysis

All estimates were calculated according to sample weights, which were evaluated by taking into consideration the sampling rate, response rate, and age and sex proportions of the reference population. The analysis was adjusted for the complex sample design of the survey. The continuous variables were tested for normality using graphical tools and the Kolmogorov–Smirnov test. Continuous data are presented as means and standard errors (SE), and categorical data are presented as frequencies and SE or 95% confidence intervals (CIs), as appropriate. The comparisons between 10-year CHD risk groups and between coffee consumption categories were performed using the Student t-test for continuous data and the chi-square test for categorical data. Logistic regression analyses were used to analyze the relationship between high 10-year CHD risk and coffee consumption. All analyses were divided by sex. All tests were two-sided, and p values <0.05 were considered statistically significant. The statistical analyses were performed using the SPSS statistical package (SPSS Inc., Chicago, IL, USA).

RESULTS

The mean age of the participants was 48.0 years (SE, 0.4 years), and 53.3% (n=2,367) of the sample comprised women. The estimated prevalence of high 10-year CHD risk (≥20%) was 10.3% (SE, 0.8%) in men and 2.0% (SE, 0.4%) in women. Regular coffee drinkers (≥1 cups/day) accounted for 75.2% (SE, 1.4%) of the men and 69.3% (SE, 1.2%) of the women. The general characteristics of the study population are presented in Tables 1 and 2. The prevalence of high 10-year CHD risk was lower in regular coffee drinkers, and this difference was significant for the women (p=0.006), but not the men. The difference be-

Table 1. General characteristics of the study population according to coffee consumption (n=3,987)

	Men			Women		
	Daily drinker (n=1,209)	Non-drinker (n=411)	*p*-value	Daily drinker (n=1,578)	Non-drinker (n=789)	*p*-value
Age (y)	46.7±0.5	48.0±0.7	0.101	47.4±0.4	49.4±0.6	0.005
Systolic blood pressure (mmHg)	119±0.5	119±0.9	0.836	116±0.6	116±0.8	0.402
Diastolic blood pressure (mmHg)	77.9±0.4	77.8±0.6	0.917	73.5±0.3	72.6±0.4	0.052
Fasting plasma glucose (mg/dL)†	100±0.8	103±2.4	0.282	94.3±0.5	97.0±0.9	0.010
Total cholesterol (mg/dL)†	193±1.3	193±3.0	0.744	191±1.1	189±1.5	0.228
HDL cholesterol (mg/dL)	45.8±0.4	45.9±0.7	0.889	51.6±0.3	50.2±0.4	0.019
Total calorie intake (kcal/d)†	2550±32	2375±55	0.007	1771±23	1724±28	0.179
Menopause	--	--	NA	35.8 (1.7)	46.9 (2.2)	<0.001
Marital status: single	5.6 (0.8)	5.7 (1.6)	0.964	12.8 (1.1)	16.8 (1.7)	0.039
Education level: <12 y	23.5 (2.0)	26.3 (2.7)	0.353	33.6 (1.9)	45.4 (2.2)	<0.001
Economic inactivity or unemployment	11.1 (1.1)	17.4 (2.2)	0.007	41.0 (1.5)	57.5 (2.3)	<0.001
Equivalent income level‡			0.006			0.023
Low	14.1 (1.3)	21.9 (2.7)		17.3 (1.5)	22.5 (1.9)	
Middle	65.8 (1.6)	58.9(3.2)		61.7 (1.8)	60.1 (2.3)	
High	20.1 (1.4)	19.2 (2.4)		21.0 (1.5)	17.4 (1.7)	
Current smoking	51.9 (1.7)	35.2(3.0)	<0.001	5.5 (0.9)	3.4 (0.8)	0.088
High-risk alcohol consumption	22.5 (1.5)	28.0 (2.9)	0.085	3.7 (0.7)	3.8 (0.8)	0.907
Regular walking activity	36.8 (1.6)	38.7(3.0)	0.585	39.0 (1.8)	38.9 (2.0)	0.977
Sleep time (h/d)			0.253			<0.001
≤6	41.0 (2.0)	38.0(3.1)		37.4 (1.5)	41.7 (2.1)	
7-8	53.1 (2.0)	53.5(3.0)		58.2 (1.5)	49.9 (2.1)	
≥9	5.8 (0.8)	8.5 (1.9)		4.5 (0.7)	8.4 (1.1)	
Stress	25.5 (1.6)	24.0 (2.5)	0.604	29.8 (1.3)	27.3 (2.0)	0.287
Depressive mood	8.7 (1.0)	5.6 (1.2)	0.075	14.3 (1.0)	16.0 (1.6)	0.347
Antihypertensive drug use	14.1 (1.1)	15.1 (1.9)	0.641	13.4 (1.1)	19.5 (1.9)	0.003
Hypoglycemic agent use	5.9 (0.7)	6.9 (1.5)	0.518	3.2 (0.5)	7.4 (1.2)	<0.001
Antidyslipidemic drug use	3.8 (0.6)	2.9 (0.9)	0.431	4.1 (0.5)	7.5 (1.2)	0.003
Family history of CHD	7.9 (1.1)	6.3 (1.8)	0.464	8.1 (1.0)	7.7 (1.3)	0.800
BMI ≥25 kg/m^2	39.6 (1.7)	37.4(3.3)	0.571	27.9 (1.2)	29.8 (1.8)	0.380
WC (≥90 cm for men, ≥85 cm for women)	26.3 (1.7)	25.9 (2.6)	0.894	22.5 (1.4)	24.7 (1.8)	0.304
Daily tea consumption	16.3 (1.3)	10.6 (2.0)	0.038	15.1 (1.1)	4.8 (1.0)	<0.001
Daily soda consumption	1.8 (0.4)	0.4 (0.4)	0.135	0.5 (0.2)	0.6 (0.3)	0.884
10-year CHD risk						
≥10%	34.5 (1.7)	31.4(3.0)	0.356	12.2 (1.0)	17.8 (1.5)	0.001
≥20%	8.8 (0.8)	10.4 (1.6)	0.340	1.3 (0.3)	2.7 (0.5)	0.006

BMI: body mass index; CHD: coronary heart diseases; HDL: high-density lipoprotein; NA: not applicable; WC: waist circumference.
Data are expressed as the estimated mean ± standard error or estimated percentage (standard error), as appropriate. *p*-values are those for the Student *t*-test for means or chi-square test for proportions.
†Values are the estimated means, but log-transformed values were used for comparisons.
‡Monthly household income/square root of family size.

Table 2. General characteristics of the study population with and without 10-year coronary heart disease risk (n=3,987)

	Men			Women		
	Without 10-year CHD risk (n=1,381)	With 10-year CHD risk (n=239)	*p*-value	Without 10-year CHD risk (n=2,308)	With 10-year CHD risk (n=59)	*p*-value
Age (y)	46.2±0.5	61.9±0.8	<0.001	47.8±0.3	64.5±1.0	<0.001
Systolic blood pressure (mmHg)	118±0.4	135±1.5	<0.001	116±0.5	142±2.4	<0.001
Diastolic blood pressure (mmHg)	77.5±0.4	82.2±1.2	<0.001	73.2±0.3	80.3±1.5	<0.001
Fasting plasma glucose (mg/dL)†	98.4±0.7	125±5.8	<0.001	94.5±0.5	127±4.2	<0.001
Total cholesterol (mg/dL)†	191±1.3	207±3.3	<0.001	190±1.0	208±6.2	0.003
HDL cholesterol (mg/dL)	46.4±0.4	41.0±0.6	<0.001	51.3±0.3	38.4±1.1	<0.001
Total calorie intake (kcal/day)†	2548±31	2237±61	<0.001	1755±21	1576±74	0.072
Menopause	--	--	NA	38.6 (1.4)	91.0 (5.4)	<0.001
Marital status: single	5.6 (0.8)	8.4 (2.2)	0.182	13.6 (0.9)	38.1 (8.3)	<0.001
Education level: <12 y	22.6 (1.7)	54.1 (4.5)	<0.001	36.9 (1.6)	94.9 (2.6)	<0.001
Economic inactivity or unemployment	11.3 (1.0)	30.2 (3.9)	<0.001	46.0 (1.4)	49.1(9.3)	0.736
Equivalent income level‡			<0.001			<0.001
Low	14.6 (1.3)	32.0 (3.8)		18.7 (1.3)	53.6 (6.8)	
Middle	64.8 (1.7)	54.4 (4.2)		61.2 (1.6)	36.2 (5.7)	
High	20.6 (1.4)	13.6 (2.7)		20.1 (1.3)	10.2 (5.0)	
Current smoking	46.0 (1.6)	58.5 (3.8)	0.002	4.9 (0.7)	3.3 (2.0)	0.504
High-risk alcohol consumption	24.5 (1.4)	13.6 (2.8)	0.003	4.1 (0.6)	0 (0)	0.267
Regular walking activity	37.3 (1.5)	40.5 (4.1)	0.436	38.7 (1.4)	44.1 (7.5)	0.462
Sleep time (h/d)			0.512			0.140
≤6	41.2 (1.9)	36.4 (4.1)		39.2 (1.3)	48.0 (6.7)	
7-8	52.3 (1.8)	56.0 (4.2)		54.8 (1.3)	41.9 (6.3)	
≥9	6.5 (1.0)	7.6 (2.0)		5.9 (0.7)	10.1 (4.8)	
Stress	24.9 (1.5)	21.6 (4.0)	0.458	29.5 (1.1)	27.4 (6.3)	0.739
Depressive mood	8.2 (1.0)	8.4 (2.1)	0.938	15.0 (0.9)	16.3 (5.8)	0.826
Antihypertensive drug use	13.2 (1.1)	32.6 (4.6)	<0.001	14.7 (1.0)	70.4 (7.8)	<0.001
Hypoglycemic agent use	4.3 (0.6)	24.1 (3.1)	<0.001	3.7 (0.5)	60.0 (7.3)	<0.001
Antidyslipidemic drug use	3.6 (0.7)	6.2 (2.1)	0.136	5.1 (0.6)	22.4 (6.0)	<0.001
Family history of CHD	7.8 (1.1)	7.0 (2.3)	0.763	8.0 (0.8)	3.8 (2.3)	0.206
BMI ≥25 kg/m^2	38.3 (1.6)	42.9 (4.6)	0.335	28.0 (1.0)	63.8 (6.1)	<0.001
WC (≥90 cm for men, ≥85 cm for women)	25.6 (1.6)	33.7 (4.4)	0.073	22.6 (1.1)	65.3 (8.1)	<0.001
Daily tea consumption	15.4 (1.1)	13.0 (2.8)	0.445	11.7 (0.8)	11.4 (4.7)	0.953
Daily soda consumption	1.7 (0.4)	0.5 (0.5)	0.187	0.6 (0.2)	0 (0)	0.697
Daily coffee consumption	76.0 (1.5)	72.4 (3.6)	0.340	69.6 (1.2)	51.4 (7.5)	0.006
1 cup/d	19.1 (1.1)	20.7 (3.2)		27.4 (1.2)	31.2 (5.8)	
2 cups/d	23.3 (1.3)	25.9 (3.6)		25.9 (1.1)	16.0 (5.3)	
≥3 cups/d	33.6 (1.7)	25.8 (3.9)		16.3 (0.9)	4.2 (2.3)	

BMI: body mass index; CHD: coronary heart diseases; HDL: high-density lipoprotein; NA: not applicable; WC: waist circumference.
Data are expressed as the estimated mean ± standard error or estimated percentage (standard error), as appropriate. *p*-values are those for the Student *t*-test for means or chi-square test for proportions.
†Values presented are the estimated means, but log-transformed values were used for comparisons.
‡Monthly household income/square root of family size

tween the levels of coffee consumption and 10-year CHD risk was significant for the women (p=0.004; not shown in the table).

The frequencies of the factors used in the Framingham risk model for each level of coffee consumption by sex are presented in Table 3. In this study population, age (≥40 years for men or ≥45 years for women), T-C (≥200 mg/dL), HDL-C (<45 mg/dL for men or <50 mg/dL for women), BP (≥130/85 mmHg for men or ≥140/90 mmHg for women), diabetes, and smoking were identified as risk factors in 62.9% (n=2,701), 39.7% (n=1,579), 50.1% (n=2,018), 22.0% (n=889), 8.4% (n=361), and 26.3% (n=770) of the participants, respectively. There were significant differences in terms of the level of coffee consumption according to current smoking status in men and according to all parameters except high T-C and high BP among women. For both men and women, the proportion of smokers was highest in the group that consumed ≥3 cups of coffee per day (all p<0.001). The prevalence of diabetes and the proportions of men aged ≥40 years and women aged ≥45 years were lowest in the group that consumed ≥3 cups of coffee per day; the differences between the levels of coffee consumption were significant among the women (p=0.001 and p<0.001, respectively), but not among the men.

The odds ratios (ORs) for ≥20% 10-year CHD risk were calculated using logistic regression analysis (Table 4). Marital status, education level, economic inactivity or unemployment, equivalent income level, high-risk alcohol consumption (for men), menopause (for women), antihypertensive drug use, antidyslipidemic drug use, general obesity, abdominal obesity, daily tea consumption,

Table 3. Prevalence of components used in the Framingham risk model for the level of coffee consumption by sex

Parameters by sex	Coffee consumption				*p*-value
	Non-drinker	1 cup/day	2 cups/day	≥3 cups/day	
Men (n=1,620)	(n=411)	(n=326)	(n=392)	(n=491)	
Age ≥40 years	72.1 (65.6-77.9)	69.0 (61.1-76.0)	68.7 (62.7-74.1)	66.9 (60.4-72.9)	0.649
T-C ≥200 mg/dL	39.7 (33.7-46.0)	41.2 (34.7-47.9)	44.6 (38.6-50.7)	40.5 (35.6-45.6)	0.668
HDL-C <45 mg/dL	51.1 (44.0-58.2)	47.2 (40.6-53.8)	54.8 (48.8-60.7)	52.9 (47.5-58.3)	0.416
BP ≥130/85 mmHg	33.2 (28.2-38.6)	34.5 (28.4-41.2)	35.6 (30.3-41.3)	28.4 (24.1-33.2)	0.183
Diabetes	11.7 (8.4-16.1)	10.6 (7.4-14.8)	11.8 (8.4-16.4)	7.7 (5.5-10.8)	0.243
Current smoking	35.2 (29.5-41.3)	42.6 (36.7-48.7)	42.0 (36.4-47.9)	64.4 (59.4-69.2)	<0.001
Women (n=2,367)	(n=789)	(n=667)	(n=582)	(n=329)	
Age ≥45 years	59.6 (54.8-64.1)	64.4 (59.8-68.9)	50.4 (44.4-56.4)	48.7 (41.7-55.7)	<0.001
T-C ≥200 mg/dL	37.4 (33.3-41.6)	39.7 (35.5-44.0)	36.5 (32.2-41.1)	39.0 (32.1-46.3)	0.772
HDL-C <50 mg/dL	51.5 (47.8-55.2)	50.7 (45.9-55.4)	49.4 (44.2-54.5)	36.4 (30.2-43.2)	0.001
BP ≥140/90 mmHg	10.9 (8.9-13.4)	15.1 (12.1-18.6)	10.4 (8.0-13.5)	9.2 (5.9-14.2)	0.057
Diabetes	9.2 (7.0-12.0)	7.0 (5.0-9.7)	5.6 (3.6-8.6)	2.0 (0.9-4.2)	0.001
Current smoking	3.4 (2.1-5.5)	2.4 (1.4-4.3)	3.8 (2.2-6.4)	13.6 (9.5-19.3)	<0.001

BP: blood pressure; HDL-C: high-density lipoprotein cholesterol; T-C: total cholesterol.
Data are expressed as the estimated percentage (95% confidence interval), and *p*-values are those for the chi-square test for proportions.

Table 4. Multiple logistic regression analysis of the 10-year coronary heart disease risk calculated using the Framingham model and the level of coffee consumption

	Non-drinker	1 cup/day	2 cups/day	≥3 cups/day	*p* for trend
Men, ≥20% 10-year CHD risk					
Crude OR	1 (reference)	0.945 (0.585-1.53)	0.971 (0.609-1.55)	0.670 (0.416-1.08)	0.105
Adjusted OR	1 (reference)	1.00 (0.576-1.75)	1.14 (0.655-1.99)	0.895 (0.520-1.54)	0.788
Women, ≥20% 10-year CHD risk					
Crude OR	1 (reference)	0.712 (0.394-1.29)	0.387 (0.164-0.911)	0.162 (0.054-0.486)	<0.001
Adjusted OR	1 (reference)	0.682 (0.359-1.30)	0.567 (0.230-1.40)	0.211 (0.060-0.745)	0.012

CHD: coronary heart disease; OR: odds ratio.
Data are expressed as odds ratio (95% confidence interval).
Crude OR: not adjusted.
Adjusted OR: adjusted for marital status, education level, economic inactivity or unemployment, equivalent income level, high-risk alcohol consumption (for men), menopause (for women), antihypertensive drug use, antidyslipidemic drug use, general obesity (body mass index ≥25 kg/m^2), abdominal obesity (waist circumference: ≥90 cm for men and ≥85 cm for women), daily tea consumption, and total calorie intake.

total calorie intake, and coffee consumption were included as independent variables in this model. Compared with women who consumed <1 cup of coffee per day, the crude ORs for ≥20% 10-year CHD risk were 0.712 (95% CI, 0.394-1.29), 0.387 (95% CI, 0.164-0.911), and 0.162 (95% CI, 0.054-0.486) for women who consumed 1, 2, and ≥3 cups per day, respectively (*p* for trend, <0.001). Using <1 cup of coffee per day as the reference category, significantly lower OR for ≥20% 10-year CHD risk (0.211 [95% CI, 0.060-0.745]) was observed for women who consumed ≥3 cups of coffee per day after adjusting for multiple confounding factors. For women, the dose-response inverse association between coffee consumption and ≥20% 10-year CHD risk remained significant after adjusting for multiple confounding factors (*p* for trend, 0.012). In the same models, there were no significant associations between coffee consumption and 10-year CHD risk in men.

DISCUSSION

The purpose of this study was to evaluate the association between coffee consumption and CHD risk as estimated using the Framingham risk model. Although the effect of coffee consumption on CHD was reported in 1963,[21] whether high coffee consumption is associated with CHD risk remains controversial. In this study of a Korean population, women who consumed ≥3 cups of coffee daily had a lower risk for CHD compared with women who consumed <1 cup of coffee daily. There was a dose-response relationship between coffee consumption and CHD risk as estimated using the Framingham risk score; the risk appeared to be higher among women who consumed <1 cup of coffee daily. An inverse association between coffee consumption and CHD risk was observed in women but not in men. The association was not modified by socioeconomic factors, antihypertensive drug use, antidyslipidemic drug use, general and abdominal obesity, or tea consumption.

Epidemiological studies examining the association between coffee consumption and CHD risk have reported controversial findings. Some case-control and prospective studies have demonstrated an increased CHD risk associated with coffee intake. Sofi et al have reported that the combined OR from 13 case-control studies showed significant associations between coffee consumption and CHD for the group with the highest intake (>4 cups per day; OR, 1.83; 95% CI, 1.49-2.24) and for the group with the second-highest intake (3-4 cups per day; OR 1.33;

95% CI, 1.04-1.71), compared with those who did not consume coffee.[22] In contrast, recent meta-analyses have reported that overall coffee consumption was not significantly associated with CHD risk in prospective cohort studies.[12,22] Furthermore, Wu et al showed that habitual moderate coffee drinking (1-4 cups per day) was associated with a lower risk for CHD (relative risk, 0.87; 95% CI, 0.80-0.86 in men and 0.82; 95% CI, 0.73-0.92 in women).[12] In addition, a recent study suggested that asymptomatic Korean adults with moderate coffee consumption (1-5 cups per day) have a lower prevalence of coronary artery calcium, according to cardiac computed tomography imaging.[23] Some epidemiological studies have also suggested a U-shaped or J-shaped relationship between coffee consumption and CHD risk.[24-26] This discrepancy has often been explained by assuming that coffee may have acute detrimental effects on the risk for CHD or that some studies could be more likely to include bias and confounding.[27]

Coffee contains several biologically active substances that may have either beneficial or harmful effects on CHD risk. Caffeine is the best-characterized pharmacologically active substance in coffee. Acute intake of coffee containing caffeine can raise BP, heart minute volumes, cardiac index, and arterial stiffness, as well as activate the sympathetic nervous system in non-habitual coffee drinkers.[28] A crossover study conducted in Costa Rica claimed that coffee intake could act as a trigger for coronary events in people with infrequent coffee consumption.[29] However, a larger cohort study has demonstrated that habitual intake of caffeinated beverages provided protection against the risk for heart disease-related mortality among elderly participants.[30] Shechter et al have also reported that acute caffeine ingestion significantly improved endothelial function and was associated with lower levels of plasma markers of inflammation.[31] The results of several studies suggest that caffeine and chlorogenic acid contribute to the homocysteine-raising effect of coffee.[32,33] Cafestol and kahweol are diterpenoid alcohols identified in the lipid fraction of coffee grounds that could increase the plasma concentration of cholesterol.[2] A meta-analysis examining the effect of coffee consumption on serum cholesterol concentrations has reported that the dose-dependent increase in serum total and low-density lipoprotein cholesterol concentrations was greater for unfiltered than filtered coffee.[34] The diterpenescafestol and kahweol, which are largely trapped by the use of paper filters in coffee preparation, appeared to be responsible for the increase in plasma cholesterol levels.[35] The conflicting results of the studies on coffee and CHD risk may be partially due to the difference in cholesterol-raising effects between brewing methods. Our study, however, used secondary data, and there were no data available regarding the type of coffee consumed.

On the other hand, coffee is abundant in antioxidants, such as chlorogenic acid, flavonoids, melanoidins, furans, pyrroles, and maltol, which reduce the risk for endothelial dysfunction and the expression of inflammatory molecules.[35] Some studies have shown that coffee has various antioxidant properties, such as reducing low-density lipoprotein oxidation susceptibility and increasing the plasma concentration of glutathione.[36,37] In addition, coffee contains several micronutrients, such as magnesium, potassium, niacin, trigonelline, and quinides, which may improve insulin sensitivity, decrease inflammation marker levels, and reduce diabetes risk.[4,38-40] As a result, coffee consumption has some beneficial effects on inflammation and endothelial function, which may offset the detrimental effects of coffee and consequently reduce CHD risk.

In the present study, although coffee consumption was significantly associated with lower CHD risk in women, there was no such association in men. Some other studies have also reported a relationship between coffee consumption and lower risk for CHD in women but not in men.[12,41,42] However, little is known about the effect of sex on the relationship between coffee and CHD risk. The explanation for this association, especially among women, is not clear. This sex difference may be due to unmeasured confounding or the somewhat smaller sample size of male participants. In this study, there were more elderly participants, smokers, and high-risk alcohol consumers among the men than among the women. Smoking, in particular, was strongly associated with coffee consumption both in men and in women, but the prevalence of smoking was much higher among men than among women. This finding suggests that the difference in lifestyle factors between men and women may partially account for the observed sex difference in the association between coffee consumption and CHD risk. In other words, coffee consumption might be associated with unhealthy lifestyle habits, such as smoking, and the beneficial effect of coffee consumption on CHD risk in men could be attenuated by the detrimental effects of unhealthy behaviors. Further studies are needed to investigate the sex difference and the mechanism underlying the association between coffee consumption and CHD risk.

The present study has some limitations. First, this study was cross-sectional in nature, making it impossible to establish whether a cause-effect relationship exists between the level of coffee consumption and CHD. Second, only the frequency of coffee intake was evaluated in this study. The standard questionnaires did not assess the type of coffee consumed, such as boiled, filtered, or instant, and it was difficult to assess brew strength. In addition, we did not assess the effect of caffeine on the risk for CHD because the necessary information was missing from the secondary data that we analyzed. Finally, the CHD risk estimated using the Framingham risk score does not include an actual CHD event. Furthermore, the Framingham risk model may over- or underestimate risk in Koreans with significantly different genetic profiles or social or environmental backgrounds. In this study, however, the distribution of the Framingham risk scores was used to stratify CHD risk and not to calculate the absolute risk for CHD. Despite these limitations, the advantage of our study is that it represents the first effort towards confirming the sex-specific relationships between coffee consumption and CHD risk according to the Framingham risk model in a relatively large number of Korean participants. The survey was conducted recently in a nationwide, population-based, representative sample of Koreans, and all of the analyses in this study were based on sample weights and adjusted for the complex sample design of

the survey. Thus, these results can be generalized to the entire Korean adult population. There is little information about coffee intake as a predictor of CHD risk in the Korean population.

In conclusion, the present study does not support the hypothesis that coffee consumption increases CHD risk as estimated with the Framingham risk score. Moreover, coffee consumption was associated with a lower risk for CHD, with the presence of a dose-dependent inverse relationship between coffee intake and CHD risk, particularly among Korean women. The results of this study suggest the association between coffee consumption and lower risk for CHD, as well as the risk for diabetes, Parkinson's disease, and liver disease, as reported in earlier studies. Our present study, however, does not resolve the controversy as to whether coffee consumption improves or adversely affects the risk for CHD. The debate still persists regarding whether heavy coffee intake increases or reduces CHD risk. More research is needed to confirm these findings and to clarify the roles of sex, ethnicity, and other lifestyle factors such as smoking in the relationship between coffee consumption and risk for CHD.

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AUTHOR DISCLOSURES

The authors declare no conflict of interest.

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