Coffee, tea, and alcohol intake in relation to risk of type 2 diabetes in African American women[1–4]

Deborah A Boggs, Lynn Rosenberg, Edward A Ruiz-Narvaez, and Julie R Palmer

ABSTRACT

Background: Numerous studies have reported inverse associations of coffee, tea, and alcohol intake with risk of type 2 diabetes, but none has reported results separately among African American women.

Objective: We prospectively examined the relation of coffee, tea, and alcohol consumption to diabetes risk in African American women.

Design: The study included 46,906 Black Women's Health Study participants aged 30–69 y at baseline in 1995. Dietary intake was assessed in 1995 and 2001 by using a validated food-frequency questionnaire. During 12 y of follow-up, there were 3671 incident cases of type 2 diabetes. Relative risks (RRs) and 95% CIs were estimated by using Cox proportional hazards models adjusted for diabetes risk factors.

Results: Multivariable RRs for intakes of 0–1, 1, 2–3, and ≥4 cups of caffeinated coffee/d relative to no coffee intake were 0.94 (95% CI: 0.86, 1.04), 0.90 (95% CI: 0.81, 1.01), 0.82 (95% CI: 0.72, 0.93), and 0.83 (95% CI: 0.69, 1.01), respectively (*P* for trend = 0.003). Multivariable RRs for intakes of 1–3, 4–6, 7–13, and ≥14 alcoholic drinks/wk relative to never consumption were 0.90 (95% CI: 0.82, 1.00), 0.68 (95% CI: 0.57, 0.81), 0.78 (95% CI: 0.63, 0.96), and 0.72 (95% CI: 0.53, 0.98), respectively (*P* for trend < 0.0001). Intakes of decaffeinated coffee and tea were not associated with risk of diabetes.

Conclusion: Our results suggest that African American women who drink moderate amounts of caffeinated coffee or alcohol have a reduced risk of type 2 diabetes. *Am J Clin Nutr* 2010;92:960–6.

INTRODUCTION

The prevalence of type 2 diabetes among US adults is projected to rise from 9% in 2001 to 14% in 2031 (1). African American adults are disproportionately affected, with a prevalence about twice that of whites (2). Obesity is the most important risk factor for type 2 diabetes, and several dietary factors, including cereal fiber and glycemic load, have been shown to influence diabetes risk independently of body mass index (BMI) (3–5).

Numerous epidemiologic studies have observed a reduction in type 2 diabetes risk with higher levels of caffeinated coffee consumption (6, 7). Many studies have also found that decaffeinated coffee is inversely associated with diabetes risk (7). Results concerning tea consumption have been less consistent (7). The mechanism for the protective effect of coffee on diabetes risk is unclear. Coffee constituents other than caffeine, including chlorogenic acid and lignans, play a role in glucose homeostasis (8, 9). Tea is also a major source of lignans and other polyphenols that may increase insulin sensitivity (10).

The association between alcohol intake and type 2 diabetes risk has been examined in many cohort studies, and a U-shaped relation, in which moderate consumption was associated with the lowest diabetes risk and heavy consumption with an increase in risk, was shown in each of 3 meta-analyses (11–13). A possible mechanism may be through increased insulin sensitivity (14–16).

None of the previous studies of coffee, tea, and alcohol consumption in relation to type 2 diabetes has reported separately on African American women. The prevalence of type 2 diabetes among African American women is >2 times than that among white women (11.4% compared with 5.0%) (2). Caffeinated coffee intake tends to be lower in African American women than in white women (17), and alcohol intake also tends to be lower (18). We prospectively investigated the association between intakes of caffeinated and decaffeinated coffee, tea, and alcohol and risk of type 2 diabetes in a large cohort of African American women.

SUBJECTS AND METHODS

Study population

The Black Women's Health Study (BWHS) is an ongoing, prospective follow-up study of black women in the United States. The study was established in 1995 when women from across the United States were enrolled through postal questionnaires (19). The baseline questionnaire collected information on demographic characteristics, lifestyle factors, and medical history. Participants also completed a self-administered food-frequency questionnaire (FFQ) at baseline. A total of 59,000 women aged

[1] From the Slone Epidemiology Center at Boston University, Boston, MA.

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[3] Supported by the NIDDK (DK068738) and the NCI (CA58420).

[4] Address requests for reprints and correspondence to JR Palmer, Slone Epidemiology Center at Boston University, 1010 Commonwealth Avenue, Boston, MA 02215. E-mail: jpalmer@bu.edu.

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21–69 y whose addresses were judged to be valid have been followed through mailed questionnaires every 2 y. Follow-up questionnaires update exposure information and identify incident disease. Follow-up has averaged >80% of the baseline cohort in the 6 questionnaire cycles.

The present analyses are based on follow-up from 1995 through 2007, with follow-up beginning at age 30 y to exclude possible cases of type 1 diabetes. We excluded women who at baseline reported a history of diabetes (n = 2913), gestational diabetes (n = 644), myocardial infarction (n = 483), stroke (n = 380), or cancer (n = 1216); were pregnant at baseline (n = 959); had missing data on weight or height at baseline (n = 649); left >10 dietary questions blank (n = 1560); had missing or implausible (<500 or >3800 kcal) energy intake values (n = 3082); or did not complete any follow-up questionnaires (n = 235). After these exclusions, 46,906 women remained in the present analysis.

Case definition

Incident cases of diabetes were ascertained through self-report on biennial follow-up questionnaires between 1995 and 2007. We assessed the accuracy of self-reported diabetes among a sample of 227 participants whose physicians provided data from their medical records. The diagnosis of type 2 diabetes was confirmed for 218 (96%) of the women. Of the remaining 9, 3 did not have diabetes, 2 had type 1 diabetes, 2 had gestational diabetes, 1 had steroid-induced diabetes, and 1 was classified as having metabolic syndrome. Women who reported a diagnosis of diabetes before age 30 y were excluded so that the case group would be unlikely to include any cases of type 1 diabetes.

Dietary assessment

We assessed usual diet at baseline in 1995 with a 68-item modified version of the National Cancer Institute (NCI)–Block FFQ and in 2001 with an 85-item version (20). The 9 frequency responses for beverage items, including caffeinated coffee, decaffeinated coffee, and tea, ranged from never or <1 serving/mo to ≥6 servings/d. We did not have information on the type of coffee consumed (eg, filtered or instant). The question on tea intake specified both hot and iced tea but did not differentiate between black and green tea. In 1995, we asked participants to specify a small, medium, or large portion size, and small and large servings were weighted as 0.5 and 1.5 times a medium serving size, respectively. In 2001, a supersize portion, equivalent to ≥2 times the size of medium, was added. Nutrients were calculated by using the NCI's DIETSYS software (21) for the 1995 FFQ and by using the NCI's Diet*Calc software (22) for the 2001 FFQ. The 1995 FFQ was validated among 408 participants by using a 3-d dietary record and ≤3 telephone 24-h recalls (23). Energy-adjusted and deattenuated Pearson's correlation coefficients for the FFQ compared with dietary records and recalls ranged from 0.5 to 0.8 for total fat, saturated fat, protein, carbohydrate, fiber, calcium, vitamin C, folate, and β-carotene.

Information on alcohol intake was obtained at baseline and was updated on biennial follow-up questionnaires. In 1995, participants were asked if they ever drank alcoholic beverages "at least once a week for at least a year," with response categories of "yes, I drink currently," "yes, but I no longer drink," and "no." Current drinkers were asked to report the average frequency of beer, wine, and liquor consumption during the previous year, with 5 frequency responses for each type of alcohol ranging from <1 drink/wk to ≥21 drinks/wk (one drink equivalent to ≈12 g alcohol). Total alcohol intake was calculated by summing over the 3 types of alcoholic beverages. On follow-up questionnaires, participants were asked about the frequency of total alcohol consumption during the previous year, with 8 response categories ranging from none to ≥28 drinks/wk

Assessment of nondietary exposures

Information on height and current weight was obtained at baseline in 1995. Current weight was updated every 2 y by follow-up questionnaire. BMI was calculated as weight in kilograms divided by squared height in meters. In a validation study of 115 participants conducted at Howard University Cancer Center, self-reported height and weight were highly correlated with measured values (r = 0.93 and r = 0.97, respectively) (24). Information on education was ascertained on the 1995 and 2003 questionnaires. First-degree family history of diabetes was asked on the 1995 and 1999 questionnaires. Data on vigorous activity and smoking status were obtained at baseline and have been updated on biennial follow-up questionnaires.

Statistical analysis

Person-years of follow-up were calculated from baseline on 1 March 1995 to the diagnosis of diabetes, death, loss to follow-up, or end of follow-up on 1 March 2007, whichever occurred first. Cox proportional hazards models, jointly stratified by age in 1-y intervals and by questionnaire cycle, were used to estimate relative risks (RRs) and 95% CIs for risk of diabetes in relation to intakes of coffee, tea, and alcohol. Consumption of coffee and tea at baseline was assessed in relation to diabetes incidence between 1995 and 2001, and coffee and tea consumption in 2001 was assessed in relation to diabetes incidence between 2001 and 2007. Alcohol intake was updated every 2 y at the start of each questionnaire cycle.

Multivariable models were adjusted for total energy intake (quintiles), education (≤12, 13–15, ≥16 y), family history of diabetes, vigorous activity (none, <1 h/wk, 1–2 h/wk, 3–4 h/wk, ≥5 h/wk), smoking status (never; past; current, <15 cigarettes/d; current, ≥15 cigarettes/d), glycemic index (quintiles), cereal fiber (quintiles), sugar-sweetened soft drinks (<1/mo, 1–7/mo, 2–6/wk, 1/d, ≥2/d), BMI (in kg/m^2; <23, 23–24, 25–29, 30–34, 35–39, 40–44, ≥45), history of hypertension, and history of high cholesterol. Intakes of caffeinated coffee, decaffeinated coffee, tea, and alcohol were mutually adjusted for each other in the multivariable models. Covariates that changed over time (eg, vigorous activity, smoking status, and BMI) were treated as time-dependent variables in the analysis. Tests for trend were conducted by using the median of each category modeled as a continuous variable.

We assessed whether the associations between coffee, tea, and alcohol and diabetes risk were modified by age, BMI, or smoking status. Tests for interaction were performed by using a likelihood ratio test that compared models with and without interaction terms. All statistical analyses were performed by using SAS version 9.1 (SAS Institute Inc, Cary, NC).

RESULTS

Baseline characteristics of study participants according to consumption of caffeinated coffee and alcohol are presented in **Table 1**. Caffeinated coffee and alcohol intake were positively associated with each other, and both were strongly associated with older age and cigarette smoking. Higher alcohol intake was associated with lower level of education. Decaffeinated coffee intake was strongly associated with older age, whereas tea intake was not as strongly associated with age (*see* Supplemental Table 1 under "Supplemental data" in the online issue). Frequent tea consumption was associated with higher level of education and lower levels of smoking. In a comparison of characteristics of women in the highest categories of decaffeinated coffee intake with those in the highest categories of caffeinated coffee intake, it appeared that women with high decaffeinated coffee consumption were more likely to have a history of hypertension or high cholesterol, more likely to exercise, and less likely to smoke than women with high caffeinated coffee consumption.

During 439,048 person-years of follow-up, we identified 3671 cases of diabetes. Among women in our cohort, 38% drank no coffee, 35% drank caffeinated coffee exclusively, 13% drank decaffeinated coffee exclusively, and 14% drank both types of coffee. Higher levels of caffeinated coffee consumption were associated with a significant decrease in risk of diabetes (**Table 2**). Multivariable RRs were 0.94 (95% CI: 0.86, 1.04), 0.90 (95% CI: 0.81, 1.01), 0.82 (95% CI: 0.72, 0.93), and 0.83 (95% CI: 0.69, 1.01) for consumption of 0–1, 1, 2–3, and $\geq$4 cups of coffee/d, respectively, relative to no intake of either caffeinated or decaffeinated coffee (P for trend = 0.003). Decaffeinated coffee consumption was not significantly associated with diabetes risk; the multivariable RR for $\geq$4 cups of decaffeinated coffee/d compared with no intake of caffeinated or decaffeinated coffee was 1.10 (95% CI: 0.81, 1.49). Results for both caffeinated and decaffeinated coffee were similar when we excluded women with a history of hypertension at baseline from the analysis; relative to no intake of caffeinated or decaffeinated coffee, RRs were 0.79 (95% CI: 0.68, 0.92) for $\geq$2 cups of caffeinated coffee/d and 0.96 (95% CI: 0.75, 1.24) for $\geq$2 cups of decaffeinated coffee/d.

Seventy-four percent of participants drank tea at least once a month. Tea intake was also not significantly associated with risk of diabetes, with a multivariable RR of 1.14 (95% CI: 0.92, 1.42) for $\geq$4 cups of tea/d relative to no tea intake.

In the BWHS, 28% of participants were current alcohol drinkers, and only 5% drank $\geq$7 drinks/wk. Higher alcohol intake was associated with a significant reduction in diabetes risk (Table 2). Relative to women who rarely drank alcohol, multivariable RRs were 0.90 (95% CI: 0.82, 1.00), 0.68 (95% CI: 0.57, 0.81), 0.78 (95% CI: 0.63, 0.96), and 0.72 (95% CI: 0.53, 0.98) for intakes of 1–3, 4–6, 7–13, and $\geq$14 drinks/wk, respectively (P for trend $<$ 0.0001). There was a nonsignificant increase in risk of diabetes among former drinkers (RR: 1.07; 95% CI: 0.99, 1.16).

The inverse associations between caffeinated coffee consumption and risk of diabetes were present across most strata of age, BMI, and smoking (**Table 3**). In the few strata for which a statistically significant trend was not observed, the RRs for the highest consumption level relative to the lowest were $<$1.0.

For decaffeinated coffee, stratum-specific results were less consistent (*see* Supplemental Table 2 under "Supplemental data" in the online issue). Decaffeinated coffee intake was inversely associated with diabetes risk only among never smokers, with a multivariable RR for $\geq$2 cups of decaffeinated coffee/d relative to that for no coffee intake of 0.73 (95% CI: 0.53, 1.01). However, the corresponding RRs among former smokers and current smokers were 1.19 (95% CI: 0.91, 1.57) and 1.30 (95% CI: 0.88–1.93). A test for interaction (P = 0.04) indicated a significant interaction of smoking and decaffeinated coffee consumption on risk of diabetes. Finally, for tea consumption, there was no evidence of an association with diabetes risk, overall or within strata of age, BMI, and smoking.

DISCUSSION

In this large prospective study of African American women, higher intakes of caffeinated coffee and alcohol were each associated with a decrease in risk of type 2 diabetes over 12 y of follow-up. There was no evidence of an association between consumption of decaffeinated coffee or tea and risk of diabetes overall.

Our findings for caffeinated coffee consumption in relation to diabetes risk are consistent with meta-analyses of numerous cohort studies (6, 7). A meta-analysis of 18 cohort studies reported a summary RR of 0.76 (95% CI: 0.69, 0.82) for 3–4 cups/d relative to none or $\leq$2 cups/d (7). Frequency of coffee consumption was lower in our cohort than that reported in studies comprised mostly of white women. We observed an 18% reduction in diabetes risk for consumption of $\geq$2 cups of caffeinated coffee/d relative to no coffee intake. There was not a further reduction in risk for $\geq$4 cups/d. Our study is the first to report on coffee and diabetes risk among African American women separately. The Atherosclerosis Risk in Communities Study, a cohort that includes an appreciable number of African American adults, observed an overall inverse association between higher caffeinated coffee intake and self-reported diabetes, but results were not presented according to race (25).

We observed no evidence of an association between decaffeinated coffee and diabetes risk overall, but there was a nonsignificant inverse association among women who had never smoked cigarettes. Most studies that examined decaffeinated coffee separately have observed an inverse association with risk of diabetes (26–30), and a meta-analysis of 6 cohort studies reported a summary RR of 0.64 (95% CI: 0.54, 0.77) for consumption of 3–4 cups/d compared with none (7). In one study, decaffeinated coffee intake was inversely associated with diabetes risk only among participants aged $\leq$60 y (27). Both caffeinated and decaffeinated coffee were inversely associated with C-peptide concentrations in the Nurses' Health Study, suggesting that both increase insulin sensitivity (31). Given the increasing epidemiologic evidence that coffee constituents other than caffeine are responsible for the association with diabetes risk, it is unclear why we observed an association for caffeinated but not decaffeinated coffee. Women with high decaffeinated coffee intake appeared to have more underlying cardiovascular disease (ie, history of hypertension or high cholesterol) and healthier habits (ie, less smoking and more physical activity) than did women with high caffeinated coffee intake. Previous studies included a higher proportion of participants with low baseline risk of diabetes (ie, nonobese persons) than did our cohort of African American women, and an association would be more apparent in a lower-risk population. An alternative explanation for the difference in

TABLE 1
Baseline characteristics according to intakes of caffeinated coffee and alcohol in the Black Women's Health Study, 1995[1]

	Caffeinated coffee intake					Alcohol intake					
Characteristic	<1 cup/mo	1 cup/mo to <1 cup/d	1 cup/d	≥2 cups/d	*P* for trend[2]	Never	Former	1–3 drinks/wk	4–6 drinks/wk	≥7 drinks/wk	*P* for trend[2,3]
No. of participants	24,645	11,619	4945	5697		28,425	6134	6202	3198	2664	
Age (y)	37.0 ± 10.5[4]	37.6 ± 9.9	41.1 ± 9.8	42.5 ± 9.6	<0.0001	37.2 ± 10.2	40.8 ± 10.6	38.7 ± 10.0	39.2 ± 10.1	41.0 ± 10.2	<0.0001
BMI (kg/m^2)	27.6 ± 6.5	27.7 ± 6.4	27.7 ± 6.3	27.7 ± 6.1	0.21	27.5 ± 6.5	28.6 ± 6.8	27.4 ± 6.0	27.1 ± 5.9	27.6 ± 6.2	0.001
Education ≤ 12 y (%)	17.5	16.9	16.2	16.1	0.003	15.1	21.9	16.6	20.7	24.1	<0.0001
Family history of diabetes (%)	25.3	25.9	26.6	26.0	0.14	25.8	26.6	24.8	26.0	25.7	0.41
History of hypertension (%)	20.9	20.1	20.0	19.2	<0.0001	19.3	23.2	19.8	20.2	23.9	<0.0001
History of high cholesterol (%)	18.0	17.5	17.3	17.4	0.07	17.7	19.4	17.6	16.8	16.3	0.11
Vigorous activity ≥5 h/wk (%)	14.2	13.6	13.3	13.7	0.04	13.5	13.1	14.1	15.4	16.1	<0.0001
Current smoker (%)	10.8	16.6	21.5	31.5	<0.0001	9.9	18.9	22.7	28.6	40.8	<0.0001
Energy (kcal/d)	1561 ± 689	1543 ± 679	1664 ± 690	1788 ± 706	<0.0001	1545 ± 681	1684 ± 715	1598 ± 677	1672 ± 699	1802 ± 729	<0.0001
Glycemic index	49.6 ± 6.8	49.5 ± 6.6	49.6 ± 6.1	51.0 ± 6.3	<0.0001	50.0 ± 6.6	49.8 ± 6.8	49.4 ± 6.5	49.0 ± 6.4	47.1 ± 6.9	<0.0001
Cereal fiber (g)	3.7 ± 2.4	3.6 ± 2.2	3.5 ± 2.2	3.4 ± 2.1	<0.0001	3.8 ± 2.4	3.7 ± 2.3	3.5 ± 2.1	3.2 ± 1.9	2.7 ± 1.8	<0.0001
Total fat (g)	44.0 ± 10.1	44.5 ± 9.4	43.8 ± 9.2	43.5 ± 9.3	0.65	44.3 ± 9.8	43.9 ± 9.9	44.0 ± 9.3	44.3 ± 9.3	42.0 ± 9.4	<0.0001
Saturated fat (g)	14.1 ± 4.0	14.4 ± 3.6	14.2 ± 3.6	14.4 ± 3.6	<0.0001	14.3 ± 3.8	14.2 ± 3.8	14.3 ± 3.7	14.4 ± 3.6	13.7 ± 3.6	<0.0001
Soft drinks (cans/wk)	3.7 ± 7.4	3.4 ± 6.2	4.0 ± 6.6	4.3 ± 7.6	<0.0001	3.7 ± 7.1	3.8 ± 7.1	3.5 ± 6.6	3.8 ± 6.8	4.4 ± 7.9	0.02
Caffeinated coffee (cups/wk)	—	—	—	—	—	3.6 ± 7.9	4.9 ± 10.3	6.0 ± 10.3	6.2 ± 10.0	6.8 ± 11.1	<0.0001
Decaffeinated coffee (cups/wk)	1.7 ± 5.3	1.5 ± 4.4	1.0 ± 3.8	1.1 ± 5.5	<0.0001	1.2 ± 4.3	1.7 ± 5.9	1.7 ± 5.7	1.6 ± 5.5	1.7 ± 6.4	<0.0001
Tea (cups/wk)	4.6 ± 8.6	3.7 ± 6.7	3.6 ± 6.3	5.2 ± 9.7	0.0002	4.2 ± 7.9	4.4 ± 8.7	4.4 ± 8.2	4.3 ± 8.0	4.2 ± 8.5	0.21
Alcohol (drinks/wk)	1.1 ± 3.6	1.6 ± 4.3	1.8 ± 4.3	2.4 ± 5.0	<0.0001	—	—	—	—	—	—

[1] With the exception of age, means and percentages were standardized to the age distribution of the cohort at baseline.
[2] *P* values were derived from age-adjusted tests for linear trend across categories of intake.
[3] Test for trend excluded former drinkers.
[4] Mean ± SD (all such values).

TABLE 2
Intakes of coffee, tea, and alcohol in relation to risk of diabetes in the Black Women's Health Study, 1995–2007[1]

	Cases	Person-years	RR (95% CI)[2]	RR (95% CI)[3]
Caffeinated coffee				
No coffee	1278	167,264	1.00 (Ref)	1.00 (Ref)
1 cup/mo to <1 cup/d	903	112,148	0.97 (0.89, 1.06)	0.94 (0.86, 1.04)
1 cup/d	444	51,261	0.92 (0.82, 1.02)	0.90 (0.81, 1.01)
2–3 cups/d	311	38,817	0.82 (0.72, 0.93)	0.82 (0.72, 0.93)
≥4 cups/d	119	12,704	0.88 (0.72, 1.06)	0.83 (0.69, 1.01)
P for trend			0.009	0.003
Decaffeinated coffee				
No coffee	1278	167,264	1.00 (Ref)	1.00 (Ref)
1 cup/mo to <1 cup/d	735	84,704	0.96 (0.87, 1.05)	1.04 (0.93, 1.16)
1 cup/d	206	18,772	1.03 (0.89, 1.20)	1.07 (0.92, 1.25)
2–3 cups/d	109	10,097	0.95 (0.78, 1.16)	1.01 (0.83, 1.24)
≥4 cups/d	46	3363	1.09 (0.81, 1.47)	1.10 (0.81, 1.49)
P for trend			0.65	0.58
Tea				
None to <1 cup/mo	945	112,638	1.00 (Ref)	1.00 (Ref)
1 cup/mo to <1 cup/d	2063	252,073	1.03 (0.95, 1.11)	1.04 (0.96, 1.13)
1 cup/d	334	40,816	0.95 (0.84, 1.08)	0.99 (0.87, 1.12)
2–3 cups/d	234	24,284	1.10 (0.95, 1.27)	1.12 (0.97, 1.30)
≥4 cups/d	95	9236	1.06 (0.86, 1.31)	1.14 (0.92, 1.42)
P for trend			0.49	0.17
Alcohol				
Never	1669	208,356	1.00 (Ref)	1.00 (Ref)
Former	1159	104,764	1.22 (1.13, 1.31)	1.07 (0.99, 1.16)
1–3 drinks/wk	552	77,309	0.84 (0.76, 0.93)	0.90 (0.82, 1.00)
4–6 drinks/wk	132	25,071	0.60 (0.51, 0.72)	0.68 (0.57, 0.81)
7–13 drinks/wk	97	14,885	0.70 (0.57, 0.86)	0.78 (0.63, 0.96)
≥14 drinks/wk	43	6565	0.71 (0.52, 0.96)	0.72 (0.53, 0.98)
P for trend[4]			<0.0001	<0.0001

[1] RR, relative risk (estimated by using Cox proportional hazards models); Ref, referent.
[2] Adjusted for age, questionnaire cycle, and energy intake.
[3] Adjusted for age, questionnaire cycle, energy intake, education, family history of diabetes, vigorous activity, smoking, glycemic index, cereal fiber, sugar-sweetened soft drinks, BMI, history of hypertension, and history of high cholesterol. Caffeinated coffee, decaffeinated coffee, tea, and alcohol were included in the same model.
[4] Test for trend excluded former drinkers.

our findings for decaffeinated coffee from results in studies of whites may have to do with different coffee drinking patterns in African Americans and whites; there is evidence that African Americans are more likely to drink decaffeinated coffee exclusively rather than drink both types of coffee (32).

Several potential mechanisms have been hypothesized for coffee's effect on diabetes risk. Coffee is a major source of chlorogenic acid and lignans, antioxidants that may also have beneficial effects on insulin sensitivity and glucose metabolism (8, 9). Indeed, 2 studies have shown an association between coffee consumption and increased insulin sensitivity (33, 34). In a cross-sectional study, after adjustment for diabetes risk factors, coffee consumption was associated with significantly lower fasting plasma glucose concentrations (35). However, a recent intervention trial showed favorable effects of coffee on inflammatory markers and lipids but not on glucose metabolism (36). Thus, mechanisms other than glucose metabolism and insulin resistance may play a role in how coffee drinking reduces diabetes risk.

There was no evidence of an association between tea consumption and risk of diabetes in our study. Studies of tea intake in relation to diabetes risk have been less consistent than for coffee. A meta-analysis of 7 cohort studies reported a significant 18% decrease in risk of diabetes for 3–4 cups of tea/d compared with none (7). Studies of Asian populations, with a relatively high prevalence of tea consumption, have also produced inconsistent results (37–39). A Japanese study observed an inverse association between green tea and diabetes risk but no association with black tea (37), whereas another Japanese study reported no association for either green or black tea (39). The Singapore Chinese Health Study found a modest inverse association between black tea and risk of diabetes but no association with green tea (38). We did not ask about the type of tea consumed, but black tea comprises the majority of tea consumption in Western populations.

We found an inverse association between moderate alcohol consumption (≥4 drinks/wk) and risk of diabetes risk, consistent with findings from 3 meta-analyses (11–13). In the most recent meta-analysis, which included 20 cohort studies, the summary RR among women was 0.60 (95% CI: 0.52, 0.69) for 24 g/d (≈2 drinks/d) compared with lifetime abstainers. The risk reduction remained until ≈50 g/d (≈4 drinks/d), but risk increased at higher amounts of drinking (13). One study that included African Americans observed an inverse association between alcohol intake and risk of diabetes among women, but the RRs for >2 drinks/d were based on very few women (40). In our cohort, there was a significant 32% reduction in diabetes risk among women who consumed 4–6 drinks/wk, with no greater reduction among drinkers of ≥7 drinks/wk. We were unable to assess the effects of heavy alcohol consumption because only 5% of women in our

TABLE 3
Intakes of caffeinated coffee and alcohol in relation to risk of diabetes, according to age, BMI, and smoking status in the Black Women's Health Study, 1995–2007[1]

	Caffeinated coffee intake						Alcohol intake					
	No coffee	1 cup/mo to <1 cup/d	1 cup/d	≥2 cups/d	*P* for trend	*P* for interaction	Never	Former	1–3 drinks/wk	≥4 drinks/wk	*P* for trend[2]	*P* for interaction
Age												
<45 y												
Cases	537	305	117	105			558	350	191	90		
RR	1.00	0.89	0.88	0.81	0.05		1.00	1.16	0.95	0.79	0.04	
(95% CI)	(Ref)	(0.76, 1.05)	(0.71, 1.08)	(0.65, 1.01)			(Ref)	(1.01, 1.34)	(0.80, 1.12)	(0.63, 1.00)		
≥45 y						0.98						0.26
Cases	741	598	327	325			1,111	809	361	182		
RR	1.00	0.98	0.92	0.84	0.005		1.00	1.03	0.87	0.67	<0.0001	
(95% CI)	(Ref)	(0.87, 1.10)	(0.81, 1.06)	(0.73, 0.96)			(Ref)	(0.93, 1.13)	(0.77, 0.99)	(0.57, 0.79)		
BMI												
<25 kg/m^2												
Cases	85	70	30	28			108	72	41	27		
RR	1.00	1.12	0.93	0.80	0.32		1.00	1.21	0.85	0.71	0.13	
(95% CI)	(Ref)	(0.78, 1.62)	(0.60, 1.44)	(0.50, 1.27)			(Ref)	(0.88, 1.67)	(0.58, 1.23)	(0.45, 1.12)		
25–29 kg/m^2												
Cases	326	210	121	111			431	282	160	83		
RR	1.00	0.85	0.88	0.77	0.02	0.44	1.00	1.12	0.94	0.71	0.008	0.61
(95% CI)	(Ref)	(0.70, 1.03)	(0.70, 1.09)	(0.62, 0.97)			(Ref)	(0.95, 1.31)	(0.77, 1.13)	(0.56, 0.91)		
≥30 kg/m^2												
Cases	867	623	293	291			1,130	805	351	162		
RR	1.00	0.96	0.91	0.85	0.02		1.00	1.05	0.90	0.72	<0.0001	
(95% CI)	(Ref)	(0.86, 1.08)	(0.79, 1.04)	(0.74, 0.98)			(Ref)	(0.95, 1.15)	(0.79, 1.02)	(0.61, 0.86)		
Smoking												
Never												
Cases	834	504	201	139			1,147	494	242	97		
RR	1.00	0.97	0.88	0.75	0.001		1.00	1.17	0.91	0.90	0.19	
(95% CI)	(Ref)	(0.86, 1.10)	(0.75, 1.04)	(0.62, 0.90)			(Ref)	(1.05, 1.31)	(0.79, 1.05)	(0.73, 1.11)		
Former												
Cases	294	251	139	146			347	437	180	81		
RR	1.00	0.91	0.85	0.84	0.07	0.64	1.00	0.87	0.83	0.58	<0.0001	0.005
(95% CI)	(Ref)	(0.76, 1.10)	(0.69, 1.05)	(0.69, 1.03)			(Ref)	(0.75, 1.00)	(0.69, 1.00)	(0.45, 0.74)		
Current												
Cases	148	148	104	145			175	227	129	94		
RR	1.00	0.86	0.97	0.89	0.58		1.00	1.15	0.89	0.66	0.001	
(95% CI)	(Ref)	(0.67, 1.10)	(0.75, 1.26)	(0.70, 1.13)			(Ref)	(0.94, 1.41)	(0.71, 1.13)	(0.51, 0.85)		

[1] RR, relative risk; Ref, referent. RRs were estimated by using Cox proportional hazards models, adjusted for age, energy intake, education, family history of diabetes, vigorous activity, smoking, glycemic index, cereal fiber, sugar-sweetened soft drinks, BMI, history of hypertension, and history of high cholesterol. Caffeinated coffee, decaffeinated coffee, tea, and alcohol were included in the same model.

[2] Test for trend excluding former drinkers.

cohort consumed ≥1 drink/d. Interestingly, in our study there was a nonsignificant 7% increase in diabetes risk among former drinkers, some of whom may have quit due to health reasons (41).

There is evidence that moderate consumption of alcohol increases insulin sensitivity (14–16). In a randomized trial in 109 participants with type 2 diabetes, moderate alcohol consumption of 1 drink/d significantly lowered fasting plasma glucose concentrations (42). In a large cross-sectional study of participants with type 1 or type 2 diabetes, increasing alcohol consumption was significantly associated with lower glycated hemoglobin concentrations, suggesting that alcohol has a beneficial effect on glycemic control (43).

Strengths of our study include its large size, prospective design, high rate and length of follow-up, and information on type 2 diabetes risk factors and other potential confounders. The assessment of type 2 diabetes relied on self-reports of physician diagnosis. Our validation study indicated that specificity of self-report was high. The prevalence of undiagnosed diabetes is estimated to be 3.8% among African American women (2). Nondifferential misclassification of outcome status would have led to an underestimation of the true magnitude of the associations with coffee, tea, and alcohol intake. Misclassification of long-term dietary intake would likely be random and would have attenuated true associations, but intakes of coffee and alcohol have been shown to be relatively well reported (44, 45). It is possible that unknown lifestyle factors may have influenced our findings; however, we were able to control for several established diabetes risk factors, and they did not appreciably affect our results. Participants in the BWHS are from all regions of the United States, and 97% have completed high school or a higher level of education. Among all African American women of the same ages, 83% have at least a high school education (46). Thus, our results are generalizable to most African American women, with the exception of the least educated.

In conclusion, our results within a cohort of African American women support the findings that moderate intakes of caffeinated coffee and alcohol reduce the risk of type 2 diabetes.

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