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Coffee and Tea Consumption Are Inversely Associated with Mortality in a Multiethnic Urban Population[1–3]

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Abstract

Coffee and tea are commonly consumed beverages. Inverse associations with mortality have been suggested for coffee and tea, but the relationships with cause-specific mortality are not well understood. We examined regular and decaffeinated coffee and tea in relation to mortality due to all causes, vascular, nonvascular, and cancer in the multi-ethnic, prospective, population-based Northern Manhattan Study. The study population included 2461 participants with diet data who were free of stroke, myocardial infarction, and cancer at baseline (mean age 68.30 ± 10.23 y, 36% men, 19% white, 23% black, 56% Hispanic). During a mean follow-up of 11 y, we examined the associations between coffee and tea consumption, assessed by food frequency questionnaire, and 863 deaths (342 vascular related and 444 nonvascular including 160 cancer deaths) using multivariable-adjusted Cox models. Coffee consumption was inversely associated with all-cause mortality [for each additional cup/d, HR = 0.93 (95% CI: 0.88, 0.99); $P = 0.02$]. Caffeinated coffee was inversely associated with all-cause mortality, driven by a strong protection among those who drank ≥4 cups/d. An inverse dose-response relationship between tea and all-cause mortality was suggested [for each additional cup/d, HR = 0.91 (95% CI: 0.84, 0.99); $P = 0.01$]. Coffee consumption ≥4/d was protective against nonvascular death [vs. <1/mo, HR = 0.57 (95% CI: 0.33, 0.97)] and tea consumption ≥2/d was protective against nonvascular death [HR = 0.63 (95% CI: 0.41, 0.95)] and cancer [HR = 0.33 (95% CI: 0.14, 0.80)]. There was a strong inverse association between coffee and vascular-related mortality among Hispanics only. Further study is needed, including investigation into the mechanisms and compounds in coffee and tea responsible for the inverse associations with mortality. The differential relationship between coffee and vascular death across race/ethnicity underscores the need for research in similar multi-ethnic cohorts including Hispanics. J. Nutr. 143: 1299–1308, 2013.

Introduction

In 2000, The National Coffee Association reported that 54% of adults in the US drink coffee daily (1). Based on the antioxidants per serving and the per capita consumption, coffee is the primary source of antioxidants in the U.S. diet (2). An average cup of brewed coffee has 396 mg of polyphenols, including ~100 mg of chlorogenic acid. Tea is the second most commonly consumed beverage in the world (3) and also contains many polyphenols, including catechins and theaflavins (4). However, coffee and tea are also important sources of caffeine, and studies have shown some negative health consequences of caffeine consumption, particularly in relation to cardiovascular disease (CVD)[6] risk, including raised blood pressure (5), arterial stiffness, circulating norepinephrine, and decreased endothelial-dependent vasodilation (6). These effects may only be acute and therefore not relevant for long-term cardiovascular health risk of coffee and tea consumption, but that remains unclear.

Elucidation of the health benefits of coffee and tea is important due to their popularity in the US and globally, yet the relationship between coffee and tea and mortality remains unclear. Several large studies have shown inverse associations between coffee consumption and all-cause mortality (7–11). However, the cause-specific mortality driving the associations is not well understood. High coffee consumption is associated with decreased mortality due to CVD (7,8,12–14), respiratory disease (7), stroke (7,8), diabetes (7), infections (7), and inflammatory diseases (14), but inconsistent results have been observed across studies (10,15–22). A potential J-shaped relationship for CVDs has also been suggested [e.g., heart failure (23)]. The relationship with cancer mortality is particularly unclear, with a few studies failing to show an association (7,8) or showing differences in the association across gender (9,10).

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[3] Supplemental Tables 1–4 are available from the "Online Supporting Material" link in the online posting of the article and from the same link in the online table of contents at http://jn.nutrition.org.
[6] Abbreviations used: CVD, cardiovascular disease; MI, myocardial infarction; NOMAS, Northern Manhattan Study.
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The relationship between tea consumption and all-cause and cause-specific mortality has been less well studied. Whereas some studies have suggested a protective effect of tea on all-cause mortality (24,25), specifically CVD mortality (13,24–27), the findings have been inconsistent and much of the research has been conducted in Japan. The evidence for a relationship between tea consumption and cancer mortality is lacking (25) and more research is needed.

This study's goal was to examine the relationship of coffee and tea with all-cause mortality, mortality due to vascular causes, nonvascular causes, and cancer specifically in the prospective population-based Northern Manhattan Study (NOMAS). This study adds to the literature by examining the relationship of both coffee and tea with cause-specific mortality in a racially/ethnically diverse population of whites, blacks, and Hispanics living in the same community. Hispanics represent the fastest growing U.S. minority group and little is known about the potential effects of coffee and tea on mortality in a predominantly Hispanic population, where the risk of stroke and other causes of death are elevated.

Materials and Methods

Study population. NOMAS is a prospective cohort study designed to determine the incidence and risk factors for vascular outcomes in a multi-ethnic urban population. Study details have been published (28).

Eligible participants had never been diagnosed with stroke, were >40 y old, and resided in Northern Manhattan for ≥3 mo in a household with a telephone. Participants were identified by random-digit dialing and recruited from the telephone sample to have an in-person interview and assessment. The enrollment response rate was 75% for a total of 3298 participants, with an average annual contact rate of 95%. Participants with a previous myocardial infarction (MI) (n = 201) or cancer diagnosis (n = 303) and those without information on coffee or tea consumption (n = 333) were excluded. The study was approved by the Columbia University and University of Miami institutional review boards. All participants provided informed consent.

Baseline evaluation (1993–2001). Data were collected through interviews with trained research assistants in English or Spanish. Physical and neurological examinations were conducted by study neurologists. Race-ethnicity was based on self-identification through questions modeled after the U.S. census and conforming to standard definitions outlined by Directive 15 (29). Standardized questions were adapted from the Behavioral Risk Factor Surveillance System by the CDC regarding hypertension, diabetes, smoking, and cardiac conditions (30). Measurement of blood pressure and fasting blood specimens for glucose and lipids and the definitions of hypertension, hypercholesterolemia, diabetes, moderate-heavy physical activity, and moderate alcohol use have been described (31,32).

Coffee and tea consumption. At baseline, participants were administered a modified Block National Cancer Institute FFQ by trained bilingual research assistants (33). The questionnaire contained questions regarding the average consumption of decaffeinated coffee, regular (caffeinated) coffee, and tea (hot or iced). For this study, instances of intake referred to the consumption of approximately one medium cup. The possible responses were: never or <1/mo, 1–3/mo, 1/wk, 2–4/wk, 5–6/wk, 1/d, 2–3/d, 4–5/d, and ≥6/d.

Due to small sample sizes for each individual consumption category, and to make our analyses comparable to other studies, all coffee (regular and decaffeinated combined) and regular coffee were categorized as: <1/mo (reference), 1/mo to 4/wk, 5–7/wk, 2–3/d, and ≥4/d. Due to the lower frequency of tea and decaffeinated coffee consumption in our population, the top 3 exposure categories for these variables were combined in categorical analyses as ≥2/d. The coffee and tea exposures were also examined as pseudo-continuous variables approximated as cups/day, assigning the middle value for each questionnaire response category.

The consumption frequency of other beverage variables that were assessed in a similar manner included regular soft drinks, diet soft drinks, orange and grapefruit juice, other fruit juices or fortified drinks, Tang or Start breakfast drinks, whole milk, 2% milk, and skim or 1% milk. The consumption frequency of these other beverages was summed to create a continuous variable representing other non-water beverage consumption per day. The FFQ also assessed the frequency of consumption of commonly used additives to coffee and tea, including cream or half-and-half, milk, and nondairy creamer. The frequency of consumption of these additives was assessed in a similar manner and included individually as continuous covariates to examine the effects of coffee and tea consumption independent of their creamers.

Annual follow-up and definition of outcomes. Participants were telephone screened annually to determine changes in vital status. Hospital surveillance of admission and discharge data, including ICD-9 codes, also provided morbidity and mortality data. Family members were reminded to notify us in the event of death.

The primary outcome was all-cause mortality. Secondary outcomes were death due to confirmed vascular causes (stroke, MI, heart failure, pulmonary embolus, cardiac arrhythmia), death due to nonvascular causes (accident, cancer, pneumonia, chronic obstructive pulmonary disease, other pulmonary disorders, and other nonvascular causes), and death due to cancer. Deaths were classified as vascular or nonvascular based on information obtained from the family and physicians and validated with medical records and death certificates. Follow-up procedures and outcome classifications were previously detailed (34,35).

Statistical analysis. We constructed Cox proportional hazards models to examine the associations between coffee (any and regular only) and tea consumption and mortality, and HRs and 95% CIs were calculated. Decaffeinated coffee alone was examined in secondary exploratory analyses. Person-time of follow-up was accrued from baseline to the end of follow-up (March, 2012), death, or loss to follow-up, whichever came first. We used the following sequence of models: *1*) adjusted for demographics (age, sex, race/ethnicity, education); *2*) demographics, behavioral risk factors [smoking (never, former, current), moderate-heavy physical activity, moderate alcohol consumption], daily diet (total kilocalories, grams protein, total fat, saturated fat, carbohydrates), and BMI; and *3*) demographics, behavioral risk factors, diet, BMI, previous cardiac disease, diabetes, hypertension, and hypercholesterolemia. In model 3, we mutually controlled for tea and coffee. We examined interactions of coffee and tea with age, sex, and race/ethnicity in relation to each of the mortality outcomes in model 3, and stratified analyses were conducted when effect modification was suggested (P-interaction < 0.05). We observed no interaction between coffee and tea in relation to any of the mortality outcomes. Because self-reported total daily kcal <500 or >4000 might indicate inaccurate reporting of diet, we conducted sensitivity analyses excluding these participants (n = 73). In an exploratory analysis, we also examined the combined effect of coffee and tea by adding the coffee and tea cups per day and examined this exposure as a continuous variable in relation to the mortality outcomes in model 3. The proportionality assumption of the hazards was first confirmed in the Cox models. This was done by examining interactions between the coffee and tea exposures of interest with the logarithm of follow-up time in relation to the mortality outcomes in model 3. No significant interactions were found.

We also ran a sensitivity analysis model 4 that included a variable representing the number of other non-water beverages (soda, juice, and milk) consumed per day and nondairy creamer, cream or half-and-half, and milk added to coffee and tea. In model 4, we controlled for smoking in continuous pack-years rather than as a categorical variable and alcohol as a continuous variable, representing the glasses of beer, wine, and liquor consumed per day, rather than as a dichotomous variable.

Lastly, we ran a sensitivity analysis simultaneously examining vascular and nonvascular death using competing risks regression rather than Cox models in model 3 and sensitivity analysis model 4.

In all analyses α = 0.05. All analyses were conducted using SAS version 9.3 (SAS Institute).

Results

Of the 3298 NOMAS participants, 2461 were included in this study. **Supplemental Table 1** shows the distribution of regular coffee, decaffeinated coffee, and tea consumption in NOMAS. **Table 1** shows the vascular risk factor profile of the study population overall and according to coffee consumption categories. During a mean follow-up of 11 y (range 0–18 y), 863 deaths accrued, 342 due to vascular causes (40 strokes, 48 MIs, 42 heart failure events, 8 pulmonary emboli, 165 cardiac arrhythmias, 36 other vascular causes) and 444 due to nonvascular causes (11 accidents, 160 cancer, 100 pneumonia or chronic obstructive pulmonary disease, 61 infections, 30 renal causes, 72 other nonvascular causes).

Table 2 shows the relationship between coffee and tea and all-cause mortality. A dose-response inverse relationship was suggested between coffee and all-cause mortality with a 7% reduced mortality for each additional cup per day in model 3 ($P < 0.05$). Regular caffeinated coffee in particular was inversely associated with all-cause mortality, driven by a strong protection among those who drank ≥4 cups/d. An inverse dose-response relationship between tea consumption and all-cause mortality was also suggested, with 9% decreased mortality for each increased cup per day ($P < 0.05$). In sensitivity model 4, the association for all coffee was slightly attenuated but was somewhat stronger for tea. Decaffeinated coffee consumption was relatively rare, limiting statistical power to examine it separately, but exploratory analyses suggested a possible inverse trend with all-cause mortality [model 3: decaffeinated cups per day, HR = 0.88 (95% CI: 0.79, 0.98)] (**Supplemental Table 2**). When coffee and tea were combined, an inverse association was found, with an 8% decreased mortality for each cup per day [model 3, (95% CI: 0.88, 0.97)].

For vascular death, the associations for coffee and tea were slightly attenuated and no significant associations were observed (**Table 3**), including when they were combined as a single variable [model 3 cups/d, HR = 0.95 (95% CI: 0.88, 1.02)]. We found a negative interaction between Hispanic ethnicity and all coffee and regular coffee in relation to vascular death (P-interaction < 0.05). There was no association between coffee consumption, any or regular only, and vascular-related mortality among whites or blacks. However, among Hispanics, there was

TABLE 1 Characteristics of the multiethnic, urban study population, overall and according to coffee consumption[1]

Covariates	Overall study population	Coffee consumption (regular or decaffeinated)				
		<1/mo	1/mo to 4/wk	5–7/wk	2–3/d	≥4/d
n	2461	420	349	965	569	136
Age, *y*	68.3 ± 10.2	68.4 ± 10.9	67.5 ± 10.2	68.7 ± 10.3	68.2 ± 9.73	67.9 ± 9.60
Male sex[2]	36	37	42	32	36	45
Race/ethnicity,[2] %						
Black	23	31	38	22	13	10
White	19	15	18	16	24	30
Hispanic	56	51	41	60	62	59
Education,[2] %						
≤8th grade	41	39	31	44	43	40
Some high school	14	14	16	15	13	13
Completed high school	18	16	23	18	17	19
Some college	11	12	13	10	10	14
College graduate or more	16	20	17	13	16	13
Smoking,[2] %						
Current	18	11	16	15	25	35
Former	34	37	32	35	34	29
Never	48	52	52	50	41	36
Moderate-heavy physical activity, %	8	10	9	7	10	10
Moderate alcohol use, %	33	30	33	32	37	36
BMI, kg/m^2	28.2 ± 5.7	28.3 ± 5.7	28.6 ± 6.1	28.1 ± 5.5	28.1 ± 5.7	28.5 ± 5.0
Hypertension, %	73	71	74	75	70	68
Diabetes, %	21	17	25	21	21	22
Hypercholesterolemia,[2] %	57	50	58	58	59	57
Previous cardiac disease, %	18	22	18	17	18	12
Total energy,[2] *kcal/d*	1570 ± 742	1470 ± 770	1540 ± 739	1520 ± 715	1700 ± 729	1790 ± 779
Protein,[2] *g/d*	62.1 ± 31.8	58.1 ± 34.2	61.0 ± 32.0	60.2 ± 30.1	66.3 ± 29.8	72.4 ± 37.9
Saturated fat,[2] *g/d*	20.5 ± 13.0	18.3 ± 13.3	19.5 ± 13.1	20.0 ± 12.1	22.6 ± 13.1	25.6 ± 15.4
Total fat,[2] *g/d*	61.2 ± 34.2	58.2 ± 34.7	58.9 ± 34.5	59.5 ± 32.0	66.8 ± 34.8	72.7 ± 39.4
Carbohydrates,[2] *g/d*	188 ± 90.0	178 ± 91.6	184 ± 93.6	182 ± 86.5	204 ± 90.3	205 ± 84.7
Tea consumption,[2] %						
<1/mo	37	32	23	41	38	50
1/mo to 4/wk	35	28	43	34	35	31
5–7/wk	21	29	21	20	19	11
≥2/d	8	11	13	5	8	9

[1] Values are means ± SDs for continuous variables and percent for categorical variables.
[2] $P < 0.05$ across categories of coffee consumption.

TABLE 2 Relationship between coffee and tea consumption and all deaths in a multiethnic, urban study population (n = 2461, 863 events)

	HRs (95% CIs)			
Beverage consumption	Model 1[1]	Model 2[2]	Model 3[3]	Model 4[4]
All coffee (decaf and regular)				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.91 (0.72, 1.15)	0.89 (0.70, 1.13)	0.85 (0.67, 1.09)	0.86 (0.67, 1.09)
5–7/wk	0.86 (0.71, 1.04)	0.80 (0.66, 0.97)	0.78 (0.64, 0.95)	0.81 (0.66, 0.98)
2–3/d	0.83 (0.67, 1.03)	0.74 (0.59, 0.92)	0.73 (0.58, 0.91)	0.74 (0.58, 0.95)
≥4/d	0.76 (0.54, 1.07)	0.61 (0.43, 0.87)	0.57 (0.39, 0.82)	0.66 (0.45, 0.97)
Continuous (cups/d)	0.97 (0.92, 1.02)	0.93 (0.88, 0.98)	0.93 (0.88, 0.99)	0.95 (0.89, 1.01)
Regular coffee				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.87 (0.69, 1.09)	0.80 (0.63, 1.01)	0.76 (0.60, 0.97)	0.77 (0.61, 0.98)
5–7/wk	0.96 (0.81, 1.13)	0.88 (0.74, 1.05)	0.90 (0.75, 1.07)	0.94 (0.78, 1.12)
2–3/d	0.93 (0.76, 1.14)	0.80 (0.65, 0.99)	0.84 (0.68, 1.04)	0.94 (0.75, 1.17)
≥4/d	0.81 (0.54, 1.19)	0.58 (0.39, 0.88)	0.54 (0.35, 0.83)	0.68 (0.44, 1.05)
Continuous (cups/d)	0.98 (0.93, 1.04)	0.93 (0.87, 0.99)	0.94 (0.88, 1.00)	0.98 (0.92, 1.04)
Tea				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.97 (0.82, 1.14)	1.01 (0.85, 1.20)	0.98 (0.83, 1.16)	1.07 (0.90, 1.27)
5–7/wk	0.84 (0.70, 1.02)	0.90 (0.74, 1.09)	0.85 (0.70, 1.04)	0.89 (0.73, 1.08)
≥2/d	0.82 (0.64, 1.06)	0.85 (0.65, 1.10)	0.77 (0.59, 1.01)	0.71 (0.54, 0.94)
Continuous (cups/d)	0.92 (0.85, 1.00)	0.93 (0.86, 1.01)	0.91 (0.84, 0.99)	0.87 (0.80, 0.95)

[1] Model 1: Adjusted for demographics (age, sex, race/ethnicity, education).
[2] Model 2: Adjusted for demographics (age, sex, race/ethnicity, education) and behavioral risk factors (smoking, moderate alcohol use, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), and BMI.
[3] Model 3: Adjusted for demographics (age, sex, race/ethnicity, education), behavioral risk factors (smoking, moderate alcohol use, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), BMI, and vascular risk factors (history of cardiac disease, diabetes, hypertension, hypercholesterolemia), and mutually adjusted for coffee and tea.
[4] Model 4: Adjusted for demographics (age, sex, race/ethnicity, education), behavioral risk factors (pack-years of smoking, alcohol consumed/day, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), BMI, vascular risk factors (history of cardiac disease, diabetes, hypertension, hypercholesterolemia), other non-water beverage consumption, milk in coffee/tea, cream in coffee/tea, and nondairy creamer in coffee/tea, and mutually adjusted for coffee and tea.

a protective association between both any coffee and regular coffee consumption and vascular death [any coffee HR = 0.78 (95% CI: 0.65, 0.94)] (**Table 4**).

There was a significant protective association for nonvascular death observed among those who consumed ≥4 cups/d of coffee [model 3, HR = 0.48 (95% CI: 0.29, 0.81)] (**Table 5**). The potential protective association for all coffee appeared to be driven by decaffeinated coffee. In an exploratory analysis of decaffeinated coffee assessed continuously, a strong inverse trend was observed [model 3, HR = 0.81 (95% CI: 0.68, 0.96)] (Supplemental Table 2). An inverse association was found for tea consumption, with a 13% reduction in nonvascular mortality with each additional tea cup per day in model 3 ($P < 0.05$), and this association was only strengthened in model 4. When coffee and tea were combined, a 9% decreased risk of nonvascular death was observed for each cup per day of coffee or tea [model 3, (95% CI: 0.85, 0.98)].

Although the strength of association between coffee consumption, assessed continuously, and cancer mortality was slightly more protective than that for all nonvascular death, the association was not significant (**Table 6**). The association for regular coffee was also not significant. However, the power to conduct these analyses was limited (**Supplemental Tables 3** and **4**) and the effect estimates were not inconsistent with a potential protective association of high coffee consumption (e.g., ≥4 cups/d) with cancer mortality. There was an inverse relationship between tea consumption and cancer mortality such that cancer mortality decreased by 26% for each additional cup per day in model 3 ($P = 0.02$) (Table 6). This association appeared to be driven by a potentially decreased cancer mortality among those in the highest category of tea consumption (≥2/d). A 14% reduced risk of death due to cancer was found for each cup per day of coffee or tea in the combined exposure analysis ($P < 0.05$).

For all analyses, the associations remained consistent when we excluded those with improbably low (<500) or high (>4000) reported daily kilocalorie consumption (not shown). We observed no significant interaction between coffee or tea consumption and the demographic variables in relation to overall mortality, nonvascular mortality, or cancer mortality.

Table 7 shows the relationship between all coffee, regular coffee, decaffeinated coffee, and tea consumption in relation to vascular death and nonvascular death in models 3 and 4 using competing risk regression. Overall, the conclusions from these analyses were consistent with the results of the corresponding Cox models, although the effect estimates were attenuated. In these analyses, neither coffee nor tea was associated with vascular death in the full cohort. However, consumption of ≥4 cups/d of coffee was associated with a decreased risk of nonvascular death, as was consumption of ≥2 cups/d of tea in model 4. In fact, a protective dose-response relationship between tea consumption and risk of nonvascular death remained apparent.

Discussion

The majority of adults sampled in NOMAS drank coffee, often daily, and almost two-thirds drank tea. Due to the popularity of

TABLE 3 Relationship between coffee and tea consumption and vascular death in a multiethnic, urban study population (n = 2461, 342 events)

	HRs (95% CIs)			
Beverage consumption	Model 1[1]	Model 2[2]	Model 3[3]	Model 4[4]
All coffee (decaf and regular)				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.90 (0.62, 1.30)	0.93 (0.64, 1.36)	0.90 (0.61, 1.32)	0.92 (0.63, 1.33)
5–7/wk	0.85 (0.63, 1.15)	0.82 (0.61, 1.11)	0.81 (0.59, 1.12)	0.81 (0.60, 1.11)
2–3/d	0.76 (0.54, 1.07)	0.72 (0.50, 1.02)	0.71 (0.49, 1.03)	0.70 (0.48, 1.03)
≥4/d	0.82 (0.48, 1.39)	0.71 (0.41, 1.24)	0.74 (0.42, 1.31)	0.80 (0.45, 1.41)
Continuous (cups/d)	0.97 (0.89, 1.05)	0.94 (0.86, 1.03)	0.95 (0.87, 1.05)	0.96 (0.87, 1.05)
Regular coffee				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.83 (0.58, 1.20)	0.82 (0.57, 1.19)	0.79 (0.54, 1.16)	0.87 (0.61, 1.26)
5–7/wk	0.91 (0.70, 1.19)	0.87 (0.66, 1.14)	0.90 (0.68, 1.19)	0.91 (0.69, 1.20)
2–3/d	0.75 (0.54, 1.05)	0.69 (0.49, 0.98)	0.76 (0.53, 1.09)	0.86 (0.61, 1.23)
≥4/d	0.87 (0.48, 1.59)	0.69 (0.36, 1.32)	0.70 (0.36, 1.38)	0.88 (0.46, 1.68)
Continuous (cups/d)	0.95 (0.86, 1.05)	0.92 (0.83, 1.01)	0.94 (0.85, 1.04)	0.97 (0.88, 1.08)
Tea				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.86 (0.66, 1.13)	0.92 (0.70, 1.21)	0.88 (0.67, 1.17)	0.97 (0.74, 1.27)
5–7/wk	0.89 (0.67, 1.20)	0.98 (0.72, 1.32)	0.93 (0.68, 1.26)	0.97 (0.72, 1.31)
≥2/d	0.80 (0.54, 1.19)	0.82 (0.55, 1.24)	0.75 (0.49, 1.14)	0.73 (0.48, 1.11)
Continuous (cups/d)	0.94 (0.84, 1.07)	0.95 (0.84, 1.07)	0.93 (0.82, 1.05)	0.90 (0.80, 1.02)

[1] Model 1: Adjusted for demographics (age, sex, race/ethnicity, education).
[2] Model 2: Adjusted for demographics (age, sex, race/ethnicity, education) and behavioral risk factors (smoking, moderate alcohol use, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), and BMI.
[3] Model 3: Adjusted for demographics (age, sex, race/ethnicity, education), behavioral risk factors (smoking, moderate alcohol use, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), BMI, and vascular risk factors (history of cardiac disease, diabetes, hypertension, hypercholesterolemia), and mutually adjusted for coffee and tea.
[4] Model 4: Adjusted for demographics (age, sex, race/ethnicity, education), behavioral risk factors (pack-years of smoking, alcohol consumed/day, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), BMI, vascular risk factors (history of cardiac disease, diabetes, hypertension, hypercholesterolemia), other non-water beverage consumption, milk in coffee/tea, cream in coffee/tea, and nondairy creamer in coffee/tea, and mutually adjusted for coffee and tea.

these beverages, a greater understanding of their health consequences is imperative. In this prospective cohort study, we observed a protective dose-response relationship between coffee and tea consumption and all-cause mortality.

For nonvascular death, an inverse association with coffee consumption was also suggested, driven by a protective effect observed among those in the highest exposure category (≥4/d). The inverse association between any coffee with nonvascular death was likely attributed to decaffeinated rather than caffeinated coffee, although further study in populations with greater consumption of decaffeinated coffee is needed to confirm these findings. Tea consumption also appeared to be potentially protective for nonvascular death, particularly cancer death. In fact, tea appeared to be more associated with nonvascular death and cancer than coffee. Despite effect estimates consistent with a potentially protective association between frequent coffee consumption (≥4 cups/d) and cancer mortality, no significant association was found in this power-limited analysis of cancer mortality.

Overall, our findings in a multi-ethnic, predominantly Hispanic cohort are consistent with other studies that have demonstrated inverse associations between coffee consumption and mortality (7–11). The results of several studies, including the Nurses' Health Study and Health Professionals Follow-up Study, have suggested that death due to CVD may be driving the association for coffee (7,8,12–14) rather than cancer or other nonvascular causes. However, our results did not support those conclusions, as those with the highest consumption of coffee in NOMAS had a decreased risk for nonvascular death and the effect estimates for coffee cups per day were similar for vascular and nonvascular death. Although studies have shown inverse associations between coffee consumption and morbidity and mortality due to overall

TABLE 4 Relationship between coffee and vascular death stratified by race/ethnicity in a multiethnic, urban study population (n = 2406, 335 events)[1]

	HRs (95% CIs)		
Coffee	Black (n = 571, 114 cases)	White (n = 466, 95 cases)	Hispanic (n = 1369, 126 cases)
All coffee (decaf and regular) continuous, cups/d	0.98 (0.83, 1.15)	1.11 (0.93, 1.32)	0.78 (0.65, 0.94)
Regular coffee continuous, cups/d	0.97 (0.80, 1.18)	1.11 (0.92, 1.34)	0.74 (0.61, 0.90)

[1] Model 3: Adjusted for demographics (age, sex, race/ethnicity, education), behavioral risk factors (smoking, moderate alcohol use, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), BMI, vascular risk factors (history of cardiac disease, diabetes, hypertension, hypercholesterolemia), and tea.

TABLE 5 Relationship between coffee and tea consumption and nonvascular death in a multiethnic, urban study population (n = 2461, 444 events)

	HRs (95% CIs)			
Beverage consumption	Model 1[1]	Model 2[2]	Model 3[3]	Model 4[4]
All coffee (decaf and regular)				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.93 (0.67, 1.29)	0.88 (0.63, 1.23)	0.83 (0.59, 1.17)	0.82 (0.58, 1.15)
5–7/wk	0.83 (0.64, 1.09)	0.76 (0.58, 1.00)	0.73 (0.55, 0.96)	0.78 (0.59, 1.03)
2–3/d	0.92 (0.69, 1.23)	0.79 (0.58, 1.06)	0.77 (0.57, 1.05)	0.81 (0.58, 1.13)
≥4/d	0.72 (0.45, 1.17)	0.55 (0.33, 0.90)	0.48 (0.29, 0.81)	0.57 (0.33, 0.97)
Continuous (cups/d)	0.98 (0.91, 1.05)	0.93 (0.86, 1.01)	0.93 (0.86, 1.00)	0.95 (0.87, 1.03)
Regular coffee				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.95 (0.69, 1.30)	0.83 (0.60, 1.16)	0.80 (0.57, 1.12)	0.74 (0.53, 1.05)
5–7/wk	1.02 (0.80, 1.29)	0.92 (0.72, 1.18)	0.93 (0.73, 1.19)	0.99 (0.77, 1.27)
2–3/d	1.09 (0.83, 1.44)	0.90 (0.68, 1.20)	0.93 (0.70, 1.24)	1.01 (0.75, 1.37)
≥4/d	0.87 (0.51, 1.50)	0.60 (0.35, 1.05)	0.56 (0.31, 0.99)	0.67 (0.37, 1.22)
Continuous (cups/d)	1.02 (0.94, 1.10)	0.95 (0.88, 1.04)	0.96 (0.88, 1.04)	1.00 (0.91, 1.08)
Tea				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	1.01 (0.80, 1.27)	1.05 (0.83, 1.32)	1.02 (0.81, 1.29)	1.00 (0.87, 1.39)
5–7/wk	0.86 (0.66, 1.13)	0.92 (0.70, 1.20)	0.86 (0.66, 1.13)	0.85 (0.65, 1.13)
≥2/d	0.78 (0.53, 1.13)	0.79 (0.54, 1.17)	0.72 (0.49, 1.07)	0.63 (0.41, 0.95)
Continuous (cups/d)	0.88 (0.76, 0.99)	0.88 (0.78, 1.00)	0.87 (0.76, 0.99)	0.82 (0.72, 0.93)

[1] Model 1: Adjusted for demographics (age, sex, race/ethnicity, education).
[2] Model 2: Adjusted for demographics (age, sex, race/ethnicity, education) and behavioral risk factors (smoking, moderate alcohol use, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), and BMI.
[3] Model 3: Adjusted for demographics (age, sex, race/ethnicity, education), behavioral risk factors (smoking, moderate alcohol use, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), BMI, vascular risk factors (history of cardiac disease, diabetes, hypertension, hypercholesterolemia), and mutually adjusted for coffee and tea.
[4] Model 4: Adjusted for demographics (age, sex, race/ethnicity, education), behavioral risk factors (pack-years of smoking, alcohol consumed/day, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), BMI, vascular risk factors (history of cardiac disease, diabetes, hypertension, hypercholesterolemia), other non-water beverage consumption, milk in coffee/tea, cream in coffee/tea, and nondairy creamer in coffee/tea, and mutually adjusted for coffee and tea.

CVD (7,8,12–14), an effect on stroke has also been suggested, yet evidence is limited and inconsistent (7,8,26,36–38).

To the best of our knowledge, our study is the first to show differences by race/ethnicity for the association between coffee and vascular mortality. We observed a strong inverse association between coffee and vascular-related mortality among Hispanics only, suggesting the need for further research in large, multiethnic cohorts including Hispanics. The Hispanic population in NOMAS was primarily of Caribbean descent. We previously showed differences in dietary patterns across race/ethnic groups in our cohort (32), and in the current study, we found that whites and Hispanics in NOMAS consumed coffee more frequently than blacks. However, our study was not designed to elucidate whether there may be relevant differences in the types of coffees consumed by Hispanics and non-Hispanics in our community-based cohort. Some of the effects of coffee are dependent on the genotypes of certain enzymes, such as the CYP1A2 enzyme that is the main metabolizer of caffeine (39,40). Both positive and negative associations of coffee are affected by the speed at which its constituents are metabolized and how long they remain in the body. It is possible that variability in genes involved in the metabolism of caffeine, antioxidants, and other components of coffee could be responsible for differences in associations across race/ethnic groups, but other future studies will have to investigate this possibility further. For example, there is some evidence to suggest that there are race/ethnic differences in CYP1A2 enzyme activity in liver microsomes (41).

Our findings of an inverse association between tea consumption and all-cause mortality are also consistent with the findings of a few previous studies, although the literature has been scant and inconclusive (24,25). In our study, the protective relationship between tea and overall mortality appeared to be driven by nonvascular causes, particularly cancer. However, the limited statistical power of our analyses of tea is important to note (Supplemental Tables 3 and 4) and no strong conclusions can be made regarding the relationship between tea consumption and vascular mortality. Other studies have shown tea to be protective against CVD mortality (13,24–27).

Evidence for a protective effect of coffee or tea on the risk of death from cancer is lacking, which may be attributed to the etiologic heterogeneity of cancer when all types are grouped together, yet our data suggested an inverse trend between tea consumption and cancer death. Unfortunately, we were not able to examine cancer types separately. A large Japanese study showed an inverse relationship between coffee and all-cause mortality in both men and women, whereas a weak inverse association with cancer mortality was observed only in women (9). In terms of cancer morbidity, the relationships for coffee or tea consumption are inconclusive. Protective associations have been suggested for particular cancer types in some studies, but the findings have been inconsistent (42). For example, in some, but not all, studies, coffee consumption was inversely associated with colon cancer incidence (43,44), aggressive prostate cancer (45), and bladder cancer (46). Although our findings were consistent with a possible

TABLE 6 Relationship between coffee and tea consumption and cancer death in a multiethnic, urban study population (n = 2461, 160 events)

	HRs (95% CIs)			
Beverage consumption	Model 1[1]	Model 2[2]	Model 3[3]	Model 4[4]
All coffee (decaf and regular)				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	1.17 (0.69, 1.98)	1.08 (0.63, 1.85)	1.12 (0.64, 1.94)	0.91 (0.52, 1.60)
5–7/wk	0.90 (0.57, 1.42)	0.78 (0.49, 1.24)	0.75 (0.46, 1.21)	0.78 (0.49, 1.26)
2–3/d	0.98 (0.59, 1.63)	0.77 (0.46, 1.28)	0.76 (0.45, 1.29)	0.92 (0.53, 1.59)
≥4/d	0.82 (0.37, 1.83)	0.57 (0.25, 1.28)	0.48 (0.20, 1.13)	0.53 (0.21, 1.34)
Continuous (cups/d)	0.99 (0.88, 1.11)	0.92 (0.81, 1.05)	0.90 (0.79, 1.03)	0.95 (0.83, 1.10)
Regular coffee				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.91 (0.54, 1.54)	0.78 (0.46, 1.33)	0.75 (0.43, 1.30)	0.61 (0.34, 1.08)
5–7/wk	0.92 (0.61, 1.39)	0.79 (0.52, 1.19)	0.78 (0.51, 1.18)	0.83 (0.55, 1.27)
2–3/d	1.07 (0.67, 1.68)	0.80 (0.50, 1.28)	0.77 (0.48, 1.26)	0.97 (0.59, 1.60)
≥4/d	0.79 (0.31, 2.01)	0.49 (0.19, 1.27)	0.46 (0.18, 1.20)	0.60 (0.23, 1.58)
Continuous (cups/d)	1.02 (0.89, 1.16)	0.93 (0.81, 1.06)	0.94 (0.85, 1.04)	0.99 (0.86, 1.15)
Tea				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.95 (0.65, 1.37)	1.00 (0.69, 1.46)	0.96 (0.66, 1.41)	1.20 (0.83, 1.74)
5–7/wk	0.73 (0.46, 1.15)	0.82 (0.52, 1.30)	0.76 (0.47, 1.21)	0.69 (0.42, 1.14)
≥2/d	0.44 (0.21, 0.93)	0.49 (0.23, 1.04)	0.39 (0.17, 0.87)	0.33 (0.14, 0.80)
Continuous (cups/d)	0.74 (0.59, 0.94)	0.78 (0.61, 0.98)	0.74 (0.58, 0.95)	0.69 (0.54, 0.88)

[1] Model 1: Adjusted for demographics (age, sex, race/ethnicity, education).
[2] Model 2: Adjusted for demographics (age, sex, race/ethnicity, education) and behavioral risk factors (smoking, moderate alcohol use, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), and BMI.
[3] Model 3: Adjusted for demographics (age, sex, race/ethnicity, education), behavioral risk factors (smoking, moderate alcohol use, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), BMI, and vascular risk factors (history of cardiac disease, diabetes, hypertension, hypercholesterolemia), and mutually adjusted for coffee and tea.
[4] Model 4: Adjusted for demographics (age, sex, race/ethnicity, education), behavioral risk factors (pack-years of smoking, alcohol consumed/day, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), BMI, vascular risk factors (history of cardiac disease, diabetes, hypertension, hypercholesterolemia), other non-water beverage consumption, milk in coffee/tea, cream in coffee/tea, and nondairy creamer in coffee/tea, and mutually adjusted for coffee and tea.

protection against cancer mortality among those who drank ≥2 cups/d of tea, more research is required before any strong conclusions can be made (25,47–51). Much of the research on tea consumption in relation to cancer and other diseases has been conducted in Asian countries, where tea is consumed in higher volumes, and findings may not be generalizable across populations.

Epidemiologic studies like ours are not able to identify the underlying mechanisms or compounds in coffee/tea that may be responsible for the protective associations with mortality. However, the high antioxidant content of these beverages is hypothesized to play a role (2). The chlorogenic acid in coffee may decrease blood pressure by increasing NO (52). The antioxidants in coffee, such as phenolic acid, have been shown to increase the resistance of LDL to oxidation (53,54). Coffee consumption is also associated with lower plasma concentrations of certain markers of inflammation and endothelial dysfunction (55) as well as uric acid (56–58). An inverse association between caffeine and coffee consumption and risk of diabetes, an important cause of mortality in the elderly, has been reported, likely due to the reduction in free radical generation from antioxidants derived from coffee consumption (59). In mice, both coffee and caffeine have been shown to improve glucose tolerance, insulin sensitivity, and hyperinsulinemia, findings that are at least partly a result of reduced inflammatory adipocytokine expression (60).

A role of caffeine consumption in mortality, particular vascular-related mortality, should also be considered, because coffee is a leading source of caffeine intake. As previously mentioned, caffeine has myriad health consequences, including raised blood pressure (5), arterial stiffness, circulating norepinephrine, and decreased endothelial-dependent vasodilation (6). Whereas acute consumption of caffeine can increase blood pressure, habitual use of caffeine does not have a linear association with incident hypertension over the long term (61). It may be that the antioxidant content or other beneficial components of coffee consumption simply outweigh a potential deleterious effect of caffeine. When we restricted our analysis to specifically examine regular coffee, the associations did not become appreciably stronger, and in fact when we performed exploratory analyses examining decaffeinated coffee only we saw that it was inversely associated with all-cause mortality and nonvascular-related mortality, with dose-response effect estimates slightly stronger than those for regular coffee. Previous studies with more power also showed protective effects for decaffeinated coffee in relation to overall mortality and CVD-related mortality (7,8). Therefore, the caffeine in regular coffee is most likely not responsible for the protective associations observed.

The examination of both coffee and tea, separately and combined, in relation to cause-specific mortality makes this study particularly innovative and the primary strength is the use of a racially/ethnically diverse population of adults living in the same community. This primarily Hispanic population represents one that has not been well studied in terms of the health consequences of coffee and tea consumption. However, the use of an older adult population from northern Manhattan with a

TABLE 7 Relationship between coffee and tea consumption and vascular and nonvascular death using competing risk regression in a multiethnic, urban study population (n = 2461)

	HRs (95% CIs)			
Beverage consumption	Vascular death: model 3[1]	Vascular death: model 4[2]	Nonvascular death: model 3[1]	Nonvascular death: model 4[2]
All coffee (decaf and regular)				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.96 (0.67, 1.44)	0.95 (0.65, 1.46)	0.89 (0.63, 1.32)	0.85 (0.60, 1.35)
5–7/wk	0.90 (0.66, 1.29)	0.91 (0.65, 1.33)	0.77 (0.59, 1.08)	0.80 (0.60, 1.15)
2–3/d	0.83 (0.58, 1.13)	0.84 (0.56, 1.16)	0.87 (0.64, 1.14)	0.88 (0.63, 1.17)
≥4/d	0.82 (0.48, 1.18)	0.89 (0.49, 1.33)	0.67 (0.42, 0.90)	0.66 (0.38, 0.92)
Continuous (cups/d)	0.97 (0.88, 1.09)	0.98 (0.89, 1.08)	0.97 (0.90, 1.08)	0.97 (0.89, 1.10)
Regular coffee				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.96 (0.67, 1.36)	1.00 (0.69, 1.45)	0.93 (0.66, 1.31)	0.86 (0.59, 1.24)
5–7/wk	0.98 (0.75, 1.40)	0.97 (0.73, 1.41)	1.01 (0.79, 1.43)	1.00 (0.77, 1.45)
2–3/d	0.93 (0.66, 1.22)	0.98 (0.68, 1.30)	1.05 (0.79, 1.35)	1.03 (0.76, 1.34)
≥4/d	0.82 (0.43, 1.16)	0.97 (0.50, 1.41)	0.84 (0.50, 1.11)	0.82 (0.45, 1.12)
Continuous (cups/d)	0.97 (0.88, 1.07)	0.99 (0.90, 1.10)	1.00 (0.93, 1.08)	1.01 (0.92, 1.10)
Decaffeinated coffee				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.84 (0.58, 1.22)	0.73 (0.49, 1.10)	0.97 (0.69, 1.37)	1.04 (0.73, 1.48)
5–7/wk	0.83 (0.58, 1.20)	0.84 (0.57, 1.27)	0.63 (0.44, 1.89)	0.67 (0.45, 0.93)
≥2/d	0.93 (0.57, 1.34)	0.91 (0.54, 1.34)	0.71 (0.44, 1.03)	0.70 (0.41, 1.03)
Continuous (cups/d)	0.96 (0.83, 1.12)	0.96 (0.82, 1.13)	0.84 (0.70, 1.01)	0.85 (0.70, 1.00)
Tea				
<1/mo	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1/mo to 4/wk	0.94 (0.72, 1.22)	0.96 (0.73, 1.27)	1.08 (0.86, 1.35)	1.08 (0.85, 1.37)
5–7/wk	0.98 (0.74, 1.28)	1.03 (0.77, 1.36)	0.95 (0.73, 1.19)	0.89 (0.67, 1.13)
≥2/d	0.90 (0.60, 1.19)	0.88 (0.57, 1.19)	0.78 (0.53, 1.02)	0.70 (0.45, 0.93)
Continuous (cups/d)	0.97 (0.86, 1.09)	0.95 (0.84, 1.08)	0.90 (0.80, 1.01)	0.85 (0.74, 0.97)

[1] Model 3: Adjusted for demographics (age, sex, race/ethnicity, education), behavioral risk factors (smoking, moderate alcohol use, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), BMI, and vascular risk factors (history of cardiac disease, diabetes, hypertension, hypercholesterolemia), and mutually adjusted for coffee and tea.

[2] Model 4: Adjusted for demographics (age, sex, race/ethnicity, education), behavioral risk factors (pack-years of smoking, alcohol consumed/day, moderate-heavy physical activity), diet (total daily energy, protein, carbohydrates, total fat, saturated fat), BMI, vascular risk factors (history of cardiac disease, diabetes, hypertension, hypercholesterolemia), other non-water beverage consumption, milk in coffee/tea, cream in coffee/tea, and nondairy creamer in coffee/tea, and mutually adjusted for coffee and tea.

unique race/ethnic composition limits the generalizability of our findings to other populations. Although we controlled for many potential confounders that are known risk factors for death, residual confounding by unmeasured and measured risk factors, including correlated dietary habits, is possible. Despite the use of a validated and reliable Block FFQ, there are some limitations to our coffee and tea exposure data. The analysis focuses on frequency of consumption and is not standardized for cup size. Though the majority of coffee and tea consumers reported that their normal dose was a "medium cup," this may be interpreted differently across individuals. Misclassification due to self-reported beverage consumption is possible, although the study is prospective and we excluded those with improbably low or high self-reported total daily energy consumption in sensitivity analyses with the goal of minimizing misclassification bias. Although the FFQ is designed to measure average consumption over the previous year, dietary information was collected at one time (baseline) and we lacked information on duration of coffee and tea consumption as well as changes during follow-up. Although most Americans drink filtered coffee, NOMAS did not distinguish in the questionnaire whether individuals were drinking boiled unfiltered coffee or filtered coffee. Unfiltered coffee has been associated with an increased risk for CVD due to the diterpene oils found in unfiltered coffee that raise serum cholesterol concentrations. Filtered coffee has <0.1 mg/100 mL of the diterpene oils and unfiltered coffee can have 0.2–18.0 mg/100 mL (2). An additional limitation is that the FFQ did not specify the types of tea consumed (black, green, etc.). Different types of teas have varying amounts and types of polyphenols and other constituents (4). Reverse causality is also possible, if individuals with mortality-related diseases reduce or eliminate their coffee consumption. However, our cohort was stroke-free at baseline and we excluded individuals with an MI or cancer diagnosis prior to baseline. Lastly, some misclassification of the cause of death cannot be ruled out. In practice, it is difficult to tease out causes of death and vascular causes are often ascribed due to lack of definitive information. Also, if someone has a history of cancer and dies, they may get classified as a cancer death, when the actual cause is something else.

In conclusion, in our multi-ethnic population-based cohort of adults living in northern Manhattan with >10 y of follow-up, we found that coffee and tea consumption were inversely associated with mortality. Further study is needed, including investigation into the potential underlying mechanisms and compounds in coffee and tea that may be responsible for this inverse association, before public health recommendations are warranted.

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H.G., T.R., C.B.W., M.S.V.E., and R.L.S. designed research; H.G., T.R., C.B.W., M.S.V.E., and R.L.S. conducted research; H.G. analyzed data and performed statistical analysis; and H.G. wrote the paper and had primary responsibility for final content. All authors read and approved the final manuscript.

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