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Coffee and tea intake and stroke events among West Africans: findings from the SIREN Study

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Abstract

Background Coffee and tea intake is rising in parts of Africa, but evidence on associations with stroke is limited. This study examined the relationship of coffee and tea intake with stroke among Africans recruited in the matched case-control Stroke Investigative Research and Educational Network (SIREN) study in West Africa.

Methods Using data of 7,368 case-control pairs (1:1 matched for age, sex and ethnicity) from the SIREN study from multiple sites in Nigeria and Ghana, we evaluated the adjusted odds ratio (aOR) and 95% confidence intervals (CIs) for stroke (verified based on cranial computed tomography or magnetic resonance imaging) by level of coffee or tea intake (in cups per day) in a multivariable adjusted conditional logistic regression models at a two-sided $P < 0.05$.

Results Overall, 2,263 (30.7%) and 3,201 (43.4%) of participants reported coffee or tea intake of ≥ one cup per day, respectively. In the multivariate-adjusted model, coffee intake showed a statistically insignificant, suggestive inverse association with stroke among those consuming 4–5 cups/day (aOR: 0.99; 95% CI: 0.35–2.79) and ≥ 6 cups/day (aOR: 0.71; 95% CI: 0.29–1.76), *P for trend* = 0.77 compared to non-coffee consumers. Similarly, higher tea intake was linked with statistically insignificant, suggestive lower odds of stroke; aOR: 0.90 (95% CI: 0.51–1.60) for 4–5 cups/day, and aOR: 0.81 (95% CI: 0.51–1.28) for ≥ 6 cups/day, *P for trend = 0.03*. The direction of associations was consistent, independent of stratification by stroke subtype, age group, and sex, for tea intake but not for coffee intake.

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Conclusion We observed a statistically insignificant but suggestively inverse association between coffee and tea intake and stroke, with generally lower intake levels in this sample of indigenous Africans. However, prospective studies would be necessary to clarify these associations.

Keywords Stroke, Coffee, Tea, Beverages, Africa, SIREN study

Introduction

Stroke is a severe disease that often results in disability, cognitive dysfunction, and death worldwide [1]. The burden and impact are steadily increasing among Africans [2–4], with an incident rate of 105 per 100,000 persons [5] and an age-adjusted incidence of 316 per 100,000 persons annually [4]. Distinct features like early onset, severity, and short-term mortality have marked the characterization of stroke among Africans [6, 7], necessitating the need to prioritize stroke care and understand the nuanced patterns to guide relevant and timely intervention. Previous studies in Africa have linked certain dietary patterns [8–10] to stroke, with limited evidence on the significance of beverage intake, including coffee and tea intake and stroke occurrence among Africans.

Coffee and tea are two of the most widely consumed beverages globally [11], and Africa is experiencing a rise in intake [12]. These beverages are rich dietary sources of polyphenolic compounds and have attracted considerable attention in recent years for their potential cardiovascular health benefits. They achieve this through multiple complex mechanisms, including lowering blood pressure and cholesterol levels, exerting antioxidant and anti-inflammatory effects, and enhancing vascular endothelial function and insulin sensitivity [13]. Despite the purported benefits, conflicting findings have emerged regarding the impact of coffee and tea on stroke events. For example, several studies have reported a protective relationship between coffee intake and stroke [13–15], but others have linked high coffee intake to stroke, with no association among moderate coffee consumers [13]. Similarly, a study reported linear protective trends for risk of stroke mortality, heart disease, diabetes, and cancer, with daily coffee intake [16], but another found a U-shaped association, with high doses significantly showing a reduced stroke risk [17].

It is essential to clarify the significance of coffee and tea intake to draw viable conclusions and inform public health advisories and interventions for primordial stroke prevention, especially among Africans, where coffee intake is still evolving. In addition, evaluating the association of coffee and tea intake with stroke among Africans will contribute to the body of literature by revealing findings from indigenous African populations, thereby extending the scope of evidence on the subject. Also, using a large sample size of well-phenotyped stroke case-control pairs from the widely known Stroke Investigation Research and Educational Network (SIREN) [9] study in Africa is a reputable resource likely to provide robust inference for gaining insight into the association of coffee and tea intake with stroke. Therefore, this study assessed the association of coffee and tea intake with stroke occurrence among indigenous Africans, using one of the most extensive datasets on stroke epidemiology on the African continent. Additionally, we hypothesized that the association is unlikely to vary by stroke subtype, age group, and sex.

Methods

Study dataset and sample size

This study utilized data from the SIREN study [9], which is a multicenter case-control study conducted across fifteen hospital sites in Nigeria and Ghana. The study protocol was approved by the University of Ibadan/University College Hospital Ethical Review Committee (Institutional Review Board No. UI/EC/13/0105). Briefly, the SIREN study was designed to recruit adults aged 18 and over to understand the stroke burden, causes, characteristics, and associated factors among West Africans. Details of the design [18] and preliminary findings [9] of the SIREN study have been documented elsewhere. Stroke cases were recruited from designated hospital sites after providing consent. Stroke cases were included in the study if they experienced a first-ever clinical stroke within 8 days of symptom onset and had neuroimaging confirmation by CT or MRI within 10 days of symptom onset. Participants were excluded if they were younger than 18 years; had a prior history of stroke; had extra-axial hemorrhage, subarachnoid hemorrhage, brain tumor, or brain abscess; were hospitalized for coronary heart disease; were unable to provide informed consent and had no eligible surrogate respondent; or were unable to communicate due to severe stroke, aphasia, or dementia without a valid surrogate (defined as a spouse or first-degree relative who had lived with the patient within the past year). Stroke-free controls were recruited with duly informed consent from nearby communities around hospital sites and verified for stroke-free status was confirmed with the eight-item Questionnaire for Verifying Stroke-Free Status (QVSFS) before recruitment [19].

Stroke ascertainment

Stroke cases were ascertained through clinical assessment and confirmed using standard protocols that incorporated brain imaging (CT or MRI), ECG, transthoracic echocardiography, and carotid Doppler ultrasound [9].

Ischemic stroke subtypes were clarified according to the Oxfordshire Community Stroke Project guidelines [20] and the Trial of ORG 10,172 in Acute Stroke Treatment guidelines [21]. Intracerebral haemorrhage was categorized into Structural, Medication-related, Amyloid angiopathy, Systemic or other diseases, Hypertension, and Undetermined causes [9, 22]. Controls consisted of consenting adults without a history of stroke, recruited mainly from communities near any of the fifteen hospital sites where cases were identified. A pre-validated 8-item questionnaire was used to confirm participants' stroke-free status [23]. Sample size determination for SIREN followed the previously published protocol, which targeted 80% power at a 0.05 significance level [18]. To ensure accurate comparisons and minimize confounding, stroke cases were matched with stroke-free controls at a 1:1 ratio on key demographic factors known to confound stroke epidemiology, including age (± 5 years), sex, and ethnicity, for a total of 7368 participants (3684 cases and 3684 controls) in this study. All data collection, including interviews, assessments, and sample collection, was performed by trained personnel in keeping with standard protocol.

Coffee and tea intake

Coffee and tea intake were self-reported, with participants indicating the number of cups of coffee or tea typically consumed per day. Tea intake includes Japanese green tea, black tea, and other types of tea. The level of coffee or tea intake were categorized as "no" where participants reported to have never consumed coffee or tea, "1 cup" for reporting one cup per day, "2–3 cups" for 2 to 3 cups per day, "4–5 cups" for up to 5 cups a day, and "6 cups and above" for consuming at least 6 cups per day as described earlier elsewhere [16].

Covariates

Sociodemographic characteristics

Sex was categorized as either male or female. Age was reported in years and classified as < 60 or ≥ 60 years. The level of education completed was self-reported, classified as "none" for those with no formal education, "primary education" for those who completed only primary school, and "secondary education or higher" for those who completed at least secondary school. Monthly income was self-reported and classified as "low" for participants earning less than US$100 per month and "high" for those earning US$100 or more per month.

Lifestyle, anthropometric, and clinical characteristics

Lifestyle characteristics, including smoking was dichotomized as "ever" (where participants were current smokers or used any form of tobacco product in their lifetime) or "never" (where participants have never used any form of tobacco product in a lifetime), and alcohol use was dichotomized as "ever" (where participants were current alcohol users or used any form of alcoholic beverage in a lifetime) or "never" (where participants have never used any form of alcoholic beverage in a lifetime). Physical inactivity was defined as the absence of participation in moderate-intensity activities (such as walking, cycling, or gardening) or vigorous-intensity activities (such as jogging, playing football, or vigorous swimming) for at least 4 h per week [24, 25]. Family history of cardiovascular risk or disease was defined as a self-reported history of any of the following conditions in the participant's family lineage up to second-degree relatives: hypertension, diabetes, dyslipidemia, stroke, cardiac disease, or obesity. Anthropometric measurements, including height, waist and hip circumference (in cm), and weight (in kg), were measured using standard scales and protocols. Waist-to-hip ratio (WHR) was estimated as the function of ratio of waist to hip circumference, and elevated risk of abdominal obesity was defined as a WHR ≥ 0.90 for men and ≥ 0.85 for women [26]. Body mass index (BMI) was estimated as a function of weight in kilograms divided by the square of height in metres, and overweight/obesity [26] was defined as BMI ≥ 25 kg/m^2. Participants were classified as hypertensives if the average systolic and diastolic blood pressure measurements from three readings taken at baseline in a resting state were ≥ 140/90 mmHg or a current history of hypertension by trained health personnel, or current use of antihypertensive drugs [27]. Diabetes mellitus (DM) was defined as one of the following conditions: a self-reported history of DM, current use of antidiabetic medications, a glycated haemoglobin (HbA1c) level ≥ 6.5%, or a fasting blood glucose (FBG) level ≥ 7.0 mmol/L. For stroke cases, FBG was measured after seven days of admission to account for potential stress-induced hyperglycemia [28].

Statistical analysis

Characteristics of participants by level of coffee or tea intake were summarised using counts and percentages for categorical variables and means (standard deviations) for continuous variables. The level of coffee or tea intake was fitted as an exposure variable in a stepwise conditional logistic regression model to estimate the odds ratios (OR) and 95% confidence intervals (CI) for stroke. The crude models were fitted without any covariates. The age-adjusted models were fitted, with age in years considered as a continuous variable only. The multivariable-adjusted models were fitted adjusting for age in years, highest education completed (none, primary school and above), monthly income (<$100, ≥$100), ever smoked (no, yes), ever used alcohol (no, yes), physical inactivity (no, yes), family history of CVD (no, yes), waist-to-hip ratio (< 0.90 for men or 0.85 for women, ≥ 0.90 for men

or 0.85 for women), hypertension (no, yes), and DM (no, yes). The linear association was modelled by treating coffee or tea intake as a continuous variable (cups/d), with no consumers assigned 0 cups/d to estimate P for trends. In addition, the OR and 95% CI were estimated using subgroup analyses by stroke subtype (Ischemic only or Intracerebral haemorrhage only), sex (male and female), and age group (< 60 and ≥ 60 years). Furthermore, a restricted cubic spline with 5 knots for coffee and tea intake was used to visualize the non-linear relationship between coffee and tea intake and stroke. Missingness in this data was handled using multivariate imputation by chained equations (MICE) in R software. Hypothesis testing was two-sided, with P values < 0.05 considered statistically significant, and all statistical analyses were carried out in R (version 4.4.1).

Results

Participant characteristics

Of the 3684 stroke cases, 68.3% (2516) were from Nigeria, 54.2% (1996) were male, and the mean age was 59.5 ± 14.0 (Table S1). Most of the participants, 61.0% (2247), had at least a secondary school education, 30.6% (1102) had ever consumed alcohol, 37.1% (1340) had a family history of CVD, 83.1% (2824) had an elevated waist to hip ratio, and 37.7% (1369) had DM compared to the controls ($P < 0.001$).

Coffee and tea intake by participant characteristics

In this study, 69.3% (5105) of participants reported no coffee intake, 22.7% (1,670) reported consuming up to 1 cup daily, and 0.7% (55) reported consuming ≥ 6 cups daily (Table 1). Regarding tea intake, 24.4% (1,799) consumed up to 1 cup daily, 14.5% (1071) consumed 2–3 cups daily, and 2.9% (215) consumed ≥ 6 cups daily. Among participants with stroke, 30.8% (1135) reported coffee intake, 43.4% (1599) reported tea intake, and 43.5% (1602) of controls reported tea intake, with no significant differences in coffee or tea intake between stroke cases and stroke-free controls. (Table S1). Participant characteristics by level of coffee intake (Table 1) revealed that the mean age of those consuming ≥ 6 cups/day of coffee was 56.7 ± 14.3 years, while it was 59.1 ± 14.1 years among non-consumers. The proportions of participants with at least secondary education, high income, and physical inactivity were comparable across levels of coffee intake. However, the prevalence of ever-smoked status, ever used alcohol status, and family history of CVD was generally higher among non-consumers compared to those who take ≥ 6 cups of coffee daily.

According to the level of tea intake (Table 1), most non-consumers are males (54.5%, $n = 2270$), and the age distribution does not differ by tea intake level. While the proportion of those with at least secondary school education was lower among those who take ≥ 6 cups of tea daily (47.4%, $n = 102$) compared to non-consumers (56.2%, $n = 2343$), the proportion of those who earn ≥$100 was higher among those who take ≥ 6 cups of tea daily (45.6%, $n = 98$) compared to non-consumers (43.3%, $n = 1806$), $P < 0.001$. Also, the prevalence of smoking was higher ($n = 24$, 11.4% vs. $n = 307$, 7.5%), but the prevalence of alcohol use ($n = 51$, 24.2% vs. $n = 1175$, 28.6%; $P = 0.002$) and family history of CVD ($n = 60$, 28.4% vs. $n = 1199$, 29.1%; $P < 0.001$) was lower among those who take ≥ 6 cups of tea daily compared to non-consumers. The prevalence of physical inactivity, abdominal obesity, hypertension, and DM was comparable across different levels of tea intake.

Coffee intake and stroke

Using non-consumers as a reference group, the OR (95% CI) for stroke was estimated by level of coffee or tea intake in stepwise conditional logistic regression models (Table 2). In the crude model, the unadjusted OR(95%CI) for stroke among coffee drinkers were; 1.02 (0.90, 1.14) for 1 cup/day, 1.01 (0.84, 1.21) for 2–3 cups/day, 1.18 (0.66, 2.11) for 4–5 cups/day, and 0.78 (0.46, 1.34) for ≥ 6 cups/day (P for trend 0.65) and age adjustment yielded comparable results. In the fully multivariable-adjusted models, coffee intake exhibited a non-significant but suggestive inverse association; 0.94 (0.78, 1.13) for 1 cup/day, 1.13 (0.84, 1.51) for 2–3 cups/day, 0.99 (0.35, 2.79) for 4–5 cups/day and 0.71 (0.29, 1.76) for ≥ 6 cups/day, though the overall trend remained non-significant (P for trend = 0.77), with the inconsistent pattern across intermediate intake levels suggest potential threshold effects or non-linear relationships. A similar trend was observed in stratified analyses by stroke subtype, sex, and age group, except among women and those aged < 60 years (Table 2).

Tea intake and stroke

The crude OR (95% CI) among tea drinkers, using non-consumers as the reference, was 1.03 (0.92, 1.15) for 1 cup/day, 0.98 (0.86, 1.12) for 2–3 cups/day, 0.87 (0.60, 1.26) for 4–5 cups/day, and 0.90 (0.68, 1.18) for ≥ 6 cups/day (P for trend 0.69). The OR (95% CI) estimate for the age-adjusted model was comparable to that from the crude model. The multivariate adjusted model demonstrated a more consistent evidence of protective linear trend by level of tea intake; 0.83 (0.70,0.98) for 1 cup/day, 0.81 (0.66,1.00) for 2–3 cups/day, 0.90 (0.51–1.60) for 4–5 cups/day, and 0.81 (0.51,1.28) for ≥ 6 cups/day; P for trend = 0.03, with an evidence of a rectilinear relationship (Table 3). The stratified analysis by stroke subtype, sex, and age group showed similar, statistically insignificant trends across levels of intake (Table 3). The non-linear

Table 1 Characteristics of participants by level of coffee and tea intake

	Coffee intake						Tea intake					*P* value
	No coffee(N=5105)	1 cup(N=1670)	2-3 cups(N=492)	4-5 cups(N=46)	≥ 6cups(N=55)	P value	No Tea(N=4167)	1 cup(N=1799)	2-3 cups(N=1071)	4-5 cups(N=116)	≥ 6cups(N=215)	
Country												
Ghana	2056 (40.3)	240 (14.4)	74 (15.0)	8 (17.4)	6 (10.9)	<0.001	1571 (37.7)	577 (32.1)	188 (17.6)	21 (18.1)	27 (12.6)	<0.001
Nigeria	3049 (59.7)	1430 (85.6)	418 (85.0)	38 (82.6)	49 (89.1)		2596 (62.3)	1222 (67.9)	883 (82.4)	95 (81.9)	188 (87.4)	
Sex												
Male	2709 (53.1)	954 (57.1)	269 (54.7)	34 (73.9)	26 (47.3)	0.002	2270 (54.5)	948 (52.7)	591 (55.2)	63 (54.3)	120 (55.8)	0.66
Female	2396 (46.9)	716 (42.9)	223 (45.3)	12 (26.1)	29 (52.7)		1897 (45.5)	851 (47.3)	480 (44.8)	53 (45.7)	95 (44.2)	
Age (years) mean ± SD	59.1±14.1	58.9±13.5	59.0±14.0	57.9±17.1	56.7±14.3	0.70	59.0±14.1	58.9±13.8	59.0±13.8	61.5±14.0	59.1±13.9	0.42
<60 years	2508 (49.1)	814 (48.7)	226 (45.9)	25 (54.3)	32 (58.2)	0.37	2062 (49.5)	877 (48.7)	518 (48.4)	51 (44.0)	97 (45.1)	0.55
≥60 years	2597 (50.9)	856 (51.3)	266 (54.1)	21 (45.7)	23 (41.8)		2105 (50.5)	922 (51.3)	553 (51.6)	65 (56.0)	118 (54.9)	
Formal Education												
None	1040 (20.4)	365 (21.9)	107 (21.7)	8 (17.4)	11 (20.0)	0.09	911 (21.9)	309 (17.2)	215 (20.1)	28 (24.1)	68 (31.7)	<0.001
Primary School	1158 (22.7)	344 (20.6)	124 (25.3)	4 (8.7)	13 (23.6)		913 (21.9)	419 (23.3)	247 (23.1)	19 (16.4)	45 (20.9)	
≥Secondary School	2907 (56.9)	961 (57.5)	261 (53.0)	34 (73.9)	31 (56.4)		2343 (56.2)	1071 (59.5)	609 (56.8)	69 (59.5)	102 (47.4)	
Monthly Income												
<$100	2797 (54.8)	825 (49.4)	280 (56.9)	17 (37.0)	31 (56.4)	<0.001	2361 (56.7)	873 (48.5)	546 (51.0)	53 (45.7)	117 (54.4)	<0.001
≥$100	2308 (45.2)	845 (50.6)	212 (43.1)	29 (63.0)	24 (43.6)		1806 (43.3)	926 (51.5)	525 (49.0)	63 (54.3)	98 (45.6)	
Lifestyle confounders												
Ever smoked (Yes)	390 (7.8)	157 (9.7)	34 (7.2)	3 (6.7)	3 (5.8)	0.12	307 (7.5)	156 (8.8)	90 (8.7)	10 (8.9)	24 (11.4)	0.14
Ever used Alcohol (Yes)	1537 (30.4)	449 (27.7)	123 (26.1)	13 (28.9)	10 (18.9)	0.04	1175 (28.6)	582 (32.8)	287 (27.5)	37 (32.7)	51 (24.2)	0.002
Physical Inactivity (Yes)	189 (3.9)	48 (3.1)	14 (3.0)	0 (0.0)	2 (3.9)	0.35	148 (3.7)	66 (3.8)	31 (3.1)	1 (0.9)	7 (3.4)	0.47
Family history of CVD (Yes)	1600 (31.6)	499 (30.4)	135 (28.4)	16 (35.6)	12 (22.6)	0.31	1199 (29.1)	610 (34.3)	353 (33.6)	40 (35.4)	60 (28.4)	<0.001
WHR, mean ± SD	0.93±0.1	0.93±0.1	0.94±0.1	0.95±0.1	0.94±0.1	<0.001	0.93±0.1	0.93±0.1	0.94±0.1	0.94±0.1	0.94±0.1	0.01
WHR ≥0.90 (men) and 0.85 (women)	3698 (75.8)	1187 (77.1)	358 (80.3)	31 (79.5)	39 (79.6)	0.23	3012 (76.3)	1292 (76.0)	771 (76.8)	89 (84.0)	149 (77.2)	0.45
BMI (kg/m^2), mean ± SD	26.4±5.6	26.4±5.6	26.3±5.5	26.4±4.7	25.1±4.6	0.59	26.4±5.6	26.3±5.3	26.5±5.7	26.7±6.1	26.3±5.1	0.83
BMI ≥ 25kg/m^2	2519 (60.5)	792 (61.0)	217 (59.1)	18 (52.9)	20 (46.5)	0.32	2057 (60.5)	846 (59.2)	505 (61.3)	61 (66.3)	97 (59.9)	0.63
Hypertension (Yes)	3929 (77.5)	1271 (77.4)	367 (76.8)	36 (80.0)	40 (75.5)	0.98	3207 (77.7)	1372 (76.9)	816 (77.6)	88 (77.2)	160 (75.8)	0.95
Diabetes mellitus (Yes)	1293 (25.5)	363 (22.1)	109 (22.7)	11 (24.4)	10 (18.9)	0.05	974 (23.6)	486 (27.2)	247 (23.5)	33 (29.2)	46 (21.8)	0.02
Stroke Status												
Control	2556 (50.1)	830 (49.7)	246 (50.7)	21 (45.7)	31 (56.4)	0.86	2082 (50.0)	887 (49.3)	540 (50.4)	62 (53.4)	113 (52.6)	0.82
Cases	2549 (49.9)	840 (50.3)	246 (50.0)	25 (54.3)	24 (43.6)		2085 (50.0)	912 (50.7)	531 (49.6)	54 (46.6)	102 (47.4)	

Table 2 Odds ratio and 95% confidence interval of the associations between Coffee intake and Stroke stratified by stroke subtypes, sex and age in a stepwise conditional logistic regression model

	All participant	No coffee	1 cup	2–3 cups	5–4 cups	>=6 cups	*P* for trend
All participants	3684	2549 (49.9)	840 (50.3)	246 (50.0)	25 (54.3)	24 (43.6)	
Crude odds (95% CI)		1	1.02 (0.90, 1.14)	1.01 (0.84, 1.21)	1.18 (0.66, 2.11)	0.78 (0.46, 1.34)	0.65
Age-adjusted odds ratio (95% CI)		1	1.05 (0.93, 1.18)	1.06 (0.87, 1.28)	1.16 (0.64, 2.13)	0.82 (0.47, 1.43)	0.34
Multivariate-adjusted odds ratio (95% CI)		1	0.94 (0.78, 1.13)	1.13 (0.84, 1.51)	0.99 (0.35, 2.79)	0.71 (0.29, 1.76)	0.77
Stroke Subtypes							
Ischemic Stroke	2573	1729 (49.7)	617 (50.5)	191 (50.9)	15 (48.4)	21 (48.8)	
Crude odds (95% CI)		1	1.04 (0.90, 1.19)	1.05 (0.85, 1.29)	0.95 (0.47, 1.92)	0.96 (0.53, 1.75)	0.61
Age-adjusted odds ratio (95% CI)		1	1.08 (0.94, 1.25)	1.08 (0.87, 1.35)	0.93 (0.45, 1.95)	0.99 (0.53, 1.85)	0.34
Multivariate-adjusted odds ratio (95% CI)		1	0.97 (0.77, 1.21)	1.22 (0.88, 1.70)	0.79 (0.20, 3.13)	0.92 (0.30, 2.81)	0.52
Haemorrhagic Stroke	1101	815 (50.4)	220 (49.7)	53 (46.5)	10 (66.7)	3 (25.0)	
Crude odds (95% CI)		1	0.96 (0.77, 1.20)	0.87 (0.59, 1.27)	1.92 (0.65, 5.64)	0.32 (0.09, 1.20)	0.97
Age-adjusted odds ratio (95% CI)		1	0.96 (0.76, 1.20)	0.95 (0.64, 1.42)	1.91 (0.63, 5.74)	0.35 (0.09, 1.36)	0.84
Multivariate-adjusted odds ratio (95% CI)		1	0.85 (0.60, 1.21)	0.87 (0.47, 1.64)	1.33 (0.26, 6.82)	0.38 (0.07, 2.02)	0.63
Sex							
Female	1688	1178 (49.2)	372 (52.0)	116 (52.0)	6 (50.0)	16 (55.2)	
Crude odds (95% CI)		1	1.14 (0.95, 1.36)	1.13 (0.86, 1.49)	1.09 (0.35, 3.39)	1.28 (0.61, 2.67)	0.14
Age-adjusted odds ratio (95% CI)		1	1.19 (0.98, 1.43)	1.29 (0.96, 1.73)	0.97 (0.30, 3.16)	1.36 (0.63, 2.94)	0.03
Multivariate-adjusted odds ratio (95% CI)		1	1.07 (0.82, 1.41)	1.28 (0.84, 1.94)	0.96 (0.06, 16.29)	1.54 (0.43, 5.53)	0.27
Male	1996	1371 (50.6)	468 (49.1)	130 (48.3)	19 (55.9)	8 (30.8)	
Crude odds (95% CI)		1	0.93 (0.80, 1.09)	0.92 (0.72, 1.17)	1.18 (0.60, 2.34)	0.44 (0.19, 1.01)	0.53
Age-adjusted odds ratio (95% CI)		1	0.95 (0.81, 1.12)	0.92 (0.72, 1.19)	1.20 (0.60, 2.44)	0.46 (0.19, 1.08)	0.67
Multivariate-adjusted odds ratio (95% CI)		1	0.85 (0.66, 1.11)	1.04 (0.69, 1.58)	0.96 (0.31, 2.92)	0.34 (0.09, 1.29)	0.71
Age groups							
< 60 years	1778	1238 (49.4)	400 (49.1)	110 (48.7)	15 (60.0)	15 (46.9)	
Crude odds (95% CI)		1	1.04 (0.87, 1.23)	0.95 (0.72, 1.26)	1.50 (0.67, 3.35)	0.95 (0.47, 1.92)	0.67
Age-adjusted odds ratio (95% CI)		1	1.07 (0.89, 1.28)	1.08 (0.81, 1.45)	1.43 (0.62, 3.30)	1.18 (0.57, 2.45)	0.31
Multivariate-adjusted odds ratio (95% CI)		1	1.03 (0.77, 1.38)	1.33 (0.84, 2.10)	1.03 (0.25, 4.19)	1.66 (0.47, 5.83)	0.34
≥ 60 years	1906	1311 (50.5)	440 (51.4)	136 (51.1)	10 (47.6)	9 (39.1)	
Crude odds (95% CI)		1	1.03 (0.87, 1.22)	0.99 (0.76, 1.28)	0.84 (0.35, 2.02)	0.57 (0.22, 1.48)	0.96
Age-adjusted odds ratio (95% CI)		1	1.06 (0.88, 1.27)	1.02 (0.78, 1.34)	0.87 (0.35, 2.18)	0.54 (0.21, 1.43)	0.65
Multivariate-adjusted odds ratio (95% CI)		1	0.92 (0.71, 1.20)	0.95 (0.63, 1.41)	0.98 (0.23, 1.19)	0.34 (0.07, 1.56)	0.71

Age-adjusted model was adjusted for coffee, age in years. Multivariate-adjusted model adjusting for coffee, age in years, highest education completed (none, primary school and above), monthly income (<$100,≥$100), ever used alcohol (no, yes), ever smoked (no, yes), physical Inactivity (no, yes), family history of CVD (no, yes), waist to hip ration(< 0.90(men) or 0.85 (women), ≥ 0.90 (men) and 0.85 (women), hypertension (no, yes), diabetes (no, yes)

Table 3 Odds ratio and 95% confidence interval of the associations between Tea intake and Stroke stratified by stroke subtypes, sex and age in a stepwise conditional logistic regression model

	All Stroke events	No tea	1 cup	2–3 cups	4–5 cups	6 cups and above	*P* for trend
All participants	3684	2085 (50.0)	912 (50.7)	531 (531)	54 (46.6)	102 (47.4)	
Crude odds (95% CI)		1	1.03 (0.92, 1.15)	0.98 (0.86, 1.12)	0.87 (0.60, 1.26)	0.90 (0.68, 1.18)	0.69
Age-adjusted odds ratio (95% CI)		1	1.03 (0.92, 1.16)	1.00 (0.87, 1.15)	0.94 (0.64, 1.39)	0.89 (0.67, 1.19)	0.99
Multivariate-adjusted odds ratio (95% CI)		1	0.83 (0.70, 0.98)	0.81 (0.66, 1.00)	0.90 (0.51, 1.60)	0.81 (0.51, 1.28)	0.03
Stroke subtypes							
Ischemic Stroke	2573	1402 (49.2)	652 (52.0)	397 (50.3)	44 (48.9)	78 (47.9)	
Crude odds (95% CI)		1	1.12 (0.98, 1.28)	1.05 (0.89, 1.23)	0.99 (0.65, 1.50)	0.94 (0.68, 1.29)	0.40
Age-adjusted odds ratio (95% CI)		1	1.14 (0.99, 1.31)	1.08 (0.91, 1.28)	1.06 (0.68, 1.65)	0.95 (0.68, 1.33)	0.22
Multivariate-adjusted odds ratio (95% CI)		1	0.90 (0.73, 1.11)	0.86 (0.66, 1.11)	1.03 (0.54, 1.95)	0.67 (0.39, 1.17)	0.10
Haemorrhagic Stroke	1101	679 (51.8)	256 (47.5)	132 (48.0)	10 (38.5)	24 (46.2)	
Crude odds (95% CI)		1	0.84 (0.69, 1.03)	0.85 (0.66, 1.11)	0.59 (0.27, 1.30)	0.80 (0.46, 1.38)	0.04
Age-adjusted odds ratio (95% CI)		1	0.83 (0.68, 1.02)	0.84 (0.64, 1.10)	0.66 (0.29, 1.50)	0.76 (0.43, 1.35)	0.05
Multivariate-adjusted odds ratio (95% CI)		1	0.71 (0.52, 0.96)	0.75 (0.51, 1.12)	0.53 (0.14, 2.06)	1.25 (0.53, 2.95)	0.19
Sex							
Female	1688	937 (49.4)	439 (51.6)	242 (50.4)	27 (50.9)	43 (45.3)	
Crude odds (95% CI)		1	1.09 (0.93, 1.29)	1.04 (0.85, 1.28)	1.06 (0.62, 1.83)	0.85 (0.57, 1.28)	0.52
Age-adjusted odds ratio (95% CI)		1	1.11 (0.93, 1.32)	1.08 (0.87, 1.34)	0.99 (0.56, 1.76)	0.77 (0.50,1.20)	0.46
Multivariate-adjusted odds ratio (95% CI)		1	0.90 (0.70, 1.14)	0.80 (0.59, 1.09)	1.61 (0.68, 3.82)	0.82 (0.42, 1.59)	0.31
Male	1996	1148 (50.6)	473 (49.9)	289 (48.9)	27 (42.9)	59 (49.2)	
Crude odds (95% CI)		1	0.97 (0.84, 1.13)	0.94 (0.78, 1.12)	0.73 (0.44, 1.22)	0.95 (0.65, 1.37)	0.27
Age-adjusted odds ratio (95% CI)		1	0.98 (0.83, 1.14)	0.94 (0.78, 1.14)	0.88 (0.52, 1.49)	0.99 (0.67, 1.46)	0.49
Multivariate-adjusted odds ratio (95% CI)		1	0.77 (0.60, 0.98)	0.84 (0.62, 1.13)	0.53 (0.24, 1.17)	0.80, (0.42, 1.51)	0.05
Age groups							
< 60 years	1778	1049 (50.9)	418 (47.7)	248 (47.9)	20 (39.2)	43 (44.3)	
Crude odds (95% CI)		1	0.89 (0.75, 1.04)	0.90 (0.73, 1.10)	0.62 (0.34, 1.15)	0.73 (0.48, 1.12)	0.05
Age-adjusted odds ratio (95% CI)		1	0.86 (0.72, 1.02)	0.91 (0.74, 1.13)	0.69 (0.36, 1.30)	0.76 (0.49, 1.19)	0.08
Multivariate-adjusted odds ratio (95% CI)		1	0.75 (0.57, 0.97)	0.86 (0.62, 1.19)	0.81 (0.29, 2.26)	0.80 (0.40, 1.61)	0.16
≥ 60 years	1906	1036 (49.2)	494 (53.6)	283 (51.2)	34 (52.3)	59 (50.0)	
Crude odds (95% CI)		1	1.14 (0.97, 1.34)	1.09 (0.90, 1.33)	1.12 (0.68, 1.85)	0.98 (0.66, 1.44)	0.16
Age-adjusted odds ratio (95% CI)		1	1.20 (1.01, 1.42)	1.11 (0.91, 1.37)	1.16 (0.69, 1.97)	0.98 (0.65, 1.48)	0.10
Multivariate-adjusted odds ratio (95% CI)		1	0.90 (0.71, 1.15)	0.82 (0.60, 1.11)	1.06 (0.51, 2.21)	0.76 (0.40, 1.44)	0.17

Age-adjusted model was adjusted for tea, age in years. Multivariate-adjusted model adjusting for tea, age in years, highest education completed (none, primary school and above), monthly income (<$100,≥$100), ever used alcohol (no, yes), ever smoked (no, yes), physical Inactivity (no, yes), family history of CVD (no, yes), waist to hip ration(< 0.90(men) or 0.85 (women), ≥ 0.90 (men) and 0.85 (women), hypertension (no, yes), diabetes (no, yes)

trend showed a reduction in the odds of stroke as the coffee and tea intake increased (Fig. 1).

Discussion

Leveraging well-phenotyped stroke case and control pairs, we examined the association of coffee and tea intake with stroke. We found a statistically insignificant but suggestively inverse association between coffee and tea intake and stroke, with generally lower levels of intake in this sample of indigenous Africans. The associations were consistent in stratified analyses of stroke subtype, sex, and age groups for tea intake, but not for coffee intake, where the suggestive inverse associations were attenuated, especially among women and those under 60 years. The inverse association between higher levels of coffee or tea intake and stroke risk observed in this study (though not significant) aligns with previously reported findings [13–16, 29–32]. For example, the overall relative risk of total stroke decreased with increasing coffee intake in a sample of Western Europeans and North Americans, and with tea intake among Chinese and South Americans [33]. In another study, an inverse dose-dependent relationship was observed between coffee intake and stroke incidence and stroke mortality among Japanese, Europeans, and Americans [14–16].

Additionally, a pooled meta-analysis reported an 8.0% decrease in stroke risk among coffee consumers, independent of stroke subtypes [34]. This association has also been explored to investigate its relation with mortality from chronic diseases in a large prospective U.S. cohort study [16]. They observed a dose-dependent inverse association between coffee intake and various causes of

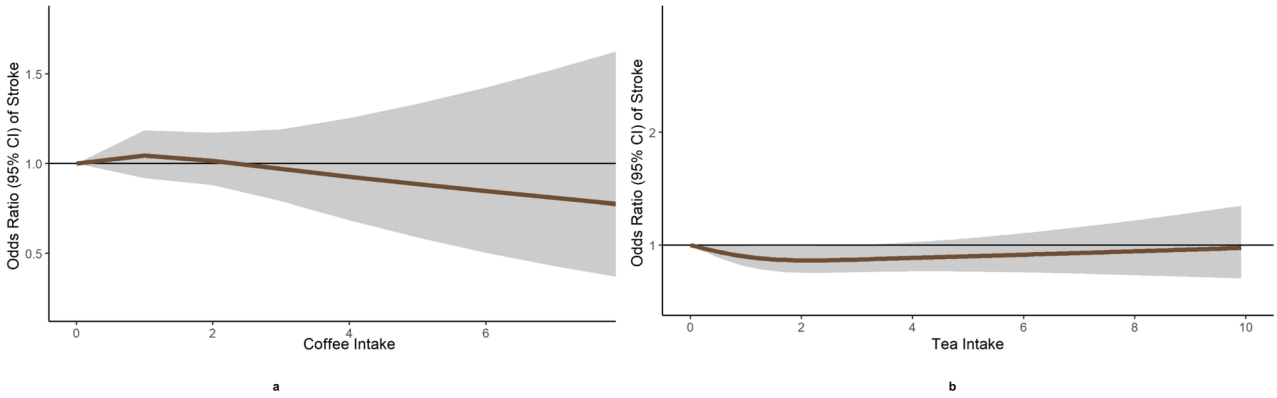

Fig. 1 Restricted cubic splines for the association between Coffee (**a**) and Tea **b**) consumption and the odds of stroke in the general population. The coffee line indicates the odds ratio (OR), and grey shades represent the 95% confidence intervals. The model was adjusted age in years, highest education completed (none, primary school and above), monthly income (<$100,≥$100), ever used alcohol (no, yes), ever smoked (no, yes), physical Inactivity (no, yes), family history of CVD (no, yes), waist to hip ration(< 0.90(men) or 0.85 (women), ≥ 0.90 (men) and 0.85 (women), hypertension (no, yes), diabetes (no, yes)

mortality, such as stroke, cancer, heart disease, respiratory issues, injuries and accidents, diabetes, and infectious diseases. Similarly, individuals who consumed six or more cups per day had a 10% (in men) and 15% (in women) lower risk of death related to chronic and acute health conditions, independent of the level of caffeination of coffee intake [16].

The suggestive inverse relationship between coffee and tea intake and stroke trends could be linked to several biological pathways. First, coffee and tea are rich in bioactive metabolites and phytochemicals such as epigallocatechin gallate (EGCG), which exhibit antioxidant, anti-inflammatory, and cardioprotective properties [30]. Polyphenolic molecules are broken down in the gut microbiota via various pathways [35, 36], into EGCG [37–39], which have protective hemodynamic potential and stability in the cardiovascular system and have been implicated in CVD, neurodegenerative disorders, and metabolic diseases [37, 40–42]. Additionally, EGCG can cross the blood–brain barrier, allowing it to exert direct neuroprotective effects and potentially limit neuronal damage during cerebrovascular processes [43, 44]. Second, most tea is flavonoid-enriched, which is an antioxidant and anti-inflammatory compound that could potentially contribute to the prevention of chronic diseases [13]. Flavonoids are naturally occurring polyphenolic compounds found in plant-based diets that are increasingly recognized for their broad therapeutic potential [45], lower risk of stroke [8] and hypertension [46]. They demonstrate antioxidant, anti-inflammatory, antimutagenic, and anticancer effects and have been shown to influence key cellular enzymes involved in oxidative stress and inflammatory pathways [45, 47, 48]. These bioactive compounds have become a key focus in nutraceutical, pharmaceutical, and functional food research because of their ability to support vascular health and lower the risk of chronic diseases such as coronary heart disease and stroke [49]. Although the exact molecular mechanisms by which flavonoids provide these benefits are not yet fully understood, ongoing research with oral contraception, molecular docking, and bioinformatics continues to deepen understanding of their biological interactions and therapeutic potential. Emerging evidence emphasizes that flavonoids are not only vital dietary components but also remain an area of ongoing research interest for future drug development to reduce the burden of chronic diseases [45].

Furthermore, the observed protective trend may be partly explained by improved hydration and expansion of intravascular volume, which have been reported to enhance blood rheology [50] and reduce the risk of clot formation, particularly in settings with high ambient temperatures. Similarly, high-volume intake of beverages such as coffee and tea (≥ 6 cups) may affect vascular parameters by expanding intravascular volume, stabilizing blood pressure, and improving circulation in dehydrated environments. Conversely, other studies have reported that higher coffee intake is associated with increased odds of stroke [29, 51]. Similarly, in our study, it is crucial to clarify that the direction of association between coffee intake and stroke risk differs between females and those < 60 years: females showed an increased stroke risk, whereas the inverse association was observed in the overall population and in other stratified analyses. This pattern may reflect sex-age-specific physiological responses to caffeine and other bioactive compounds in coffee. We hypothesize that young middle-aged adults in Africa may exhibit different patterns of caffeinated coffee intake [52], unsafe lifestyle behaviours or baseline cardiovascular risk profiles may be inherent, which may influence stroke risk profiles early in life [53]. Also, it is plausible that residual confounding or effect modification by unmeasured variables, such

as oral contraceptive use and menopause peculiar to women [54], stress levels [55], or genetic polymorphisms [56, 57] affecting caffeine metabolism, may contribute to the observed heterogeneity in stroke risk by sex and age group among coffee consumers. Additionally, hormonal fluctuations such as estrogen may influence caffeine metabolism in women [58, 59], thereby affecting vascular reactivity and hemodynamic responses.

Although the current findings indicated a possible inverse trend, this relationship was not statistically significant, and the findings of this study should be interpreted cautiously in light of the study's strengths and limitations. First, coffee and tea intake were self-reported by participants, introducing the potential for misclassification and reporting bias because participants may be aphasic and critically ill, particularly among participants with severe stroke-related impairments. Notably, we were unable to standardize what constituted a "cup" of coffee or tea across individuals, as this can vary widely depending on preferences, preparation methods, and serving sizes. Furthermore, the study did not account for the caffeine content of the beverages and other fizzy drinks consumed, which may differ substantially across types and brands. This study did not account for the various subtypes of coffee and tea in these associations, as this information was not reported during data collection. Recall bias is particularly concerning among stroke cases with severe disability, who may have had difficulty accurately reporting their usual beverage intake. Coffee and tea intake were assessed at a single time point, limiting our ability to capture changes in intake over time and potentially leading to exposure misclassification. Also, the observational design precludes causal inference, and mechanistic pathways were not directly assessed.

Additionally, the relatively low prevalence of coffee and tea intake in our study population may have reduced the statistical power to detect significant associations. Lastly, the case-control design, while efficient for studying rare outcomes like stroke, is inherently more susceptible to selection and recall biases compared to prospective cohort studies and cannot infer causality. The strength of this study lies in its statistical power, driven by the large sample size. Also, the stroke phenotype was well characterized, with reliable imaging confirming the stroke and its subtypes. The rigorous matching process between cases and controls effectively reduces confounding bias during statistical analysis, thereby improving the reliability of the findings. Furthermore, this is one of the first studies to assess the association of coffee and tea intake with stroke among indigenous Africans. While causality cannot be established from this study, the observed suggestive inverse association between coffee and tea intake and stroke may be supported by several biologically plausible mechanisms, warranting further investigation.

Conclusion

Coffee and tea intake showed a suggestive inverse association with stroke trend. These findings have laid the groundwork for additional studies to understand this phenomenon among Africans.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12913-026-14379-4.

Supplementary Material 1

Author contributions

O.J.A., A.P.O, O.M.A., and M.O. conceptualized and designed the study. O.J.A and A.P.O. performed the statistical analysis. O.J.A., M.O.O, and A.P.O. prepared the first draft of the manuscript. A.P.O. and O.M.A., M.O. revised the manuscript for important intellectual content. A.F., R.O., E.K., O.A., O.O., O.J.A., S.O.O., I.O., O.A., I.C., O.B., D.S., B.C.-T., O.O., I.O., A.A., G.O., A.S., C.J., L.T.A., Y.M., H.T., W.O., O.M.A., B.O, A.P.O, and M.O. critically reviewed the content. All authors approved the final version of the manuscript for submission and agree to be accountable for all aspects of the work and to ensure that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Data availability

The dataset used in this study is available upon request from the Stroke Investigation Research and Educational Network. (mayowaowolabi@yahoo.com).

Declarations

Ethical approval

The University of Ibadan/University College Hospital Ethical Review Committee is the overall (Institutional Review Board No.: UI/EC/13/0105) approved the study, and all participants provided written informed consent before participating. This study was conducted in full accordance with the Declaration of Helsinki.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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