Differential Impact of Risk Factors on Stroke Occurrence Among Men Versus Women in West Africa

The SIREN Study

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Background and Purpose—The interplay between sex and the dominant risk factors for stroke occurrence in sub-Saharan Africa has not been clearly delineated. We compared the effect sizes of risk factors of stroke by sex among West Africans.

Methods—SIREN study (Stroke Investigative Research and Educational Networks) is a case-control study conducted at 15 sites in Ghana and Nigeria. Cases were adults aged >18 years with computerized tomography/magnetic resonance imaging confirmed stroke, and controls were age- and sex-matched stroke-free adults. Comprehensive evaluation for vascular, lifestyle, and psychosocial factors was performed using validated tools. We used conditional logistic regression to estimate odds ratios and reported risk factor specific and composite population attributable risks with 95% CIs.

Results—Of the 2118 stroke cases, 1193 (56.3%) were males. The mean±SD age of males was 58.1±13.2 versus 60.15±14.53 years among females. Shared modifiable risk factors for stroke with adjusted odds ratios (95% CI) among females versus males, respectively, were hypertension [29.95 (12.49–71.77) versus 16.1 0(9.19–28.19)], dyslipidemia [2.08 (1.42–3.06) versus 1.83 (1.29–2.59)], diabetes mellitus [3.18 (2.11–4.78) versus 2.19 (1.53–3.15)], stress [2.34 (1.48–3.67) versus 1.61 (1.07–2.43)], and low consumption of green leafy vegetables [2.92 (1.89–4.50) versus 2.00 (1.33–3.00)]. However, salt intake and income were significantly different between males and females. Six modifiable factors had a combined population attributable risk of 99.1% (98.3%–99.6%) among females with 9 factors accounting for 97.2% (94.9%–98.7%) among males. Hemorrhagic stroke was more common among males (36.0%) than among females (27.6%), but stroke was less severe among males than females.

Conclusions—Overall, risk factors for stroke occurrence are commonly shared by both sexes in West Africa favoring concerted interventions for stroke prevention in the region. **(*Stroke*. 2019;50:820-827. DOI: 10.1161/STROKEAHA.118.022786.)**

Key Words: diabetes mellitus ■ female ■ hypertension ■ male ■ risk factors ■ sex

Recent secular trends indicate an unequivocal surge in stroke incidence, prevalence, morbidity, and mortality within low- and middle-income countries in sub-Saharan Africa.[1–3] Combating this surge will require the identification and targeting of population subsets in sub-Saharan Africa, which are susceptible to stroke through particular biological or social

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characteristics interacting with established stroke risk factors. A key demographic factor contributing to potentially different stroke risk is sex. We have previously reported an association of IL-6 (interleukin-6) rs1800796 and cyclin-dependent kinase inhibitor (CDKN2A/CDKN2B) rs2383207 with ischemic stroke in indigenous West African males but not females.[4] The interaction between sex and the dominant risk factors underpinning stroke among Africans has not been clearly deciphered, thus undermining efforts at controlling the burden of stroke.[3,5,6] Studies have identified sex differences in risk factor profile,[7–12] stroke presentation and severity,[11,13,14] and choice and response to therapy.[7,15–18]

Reasons for these sex-related differences are multifactorial and have been the subject of many studies.[11,18–21] Understanding what reduces or eliminates sex differences is valuable because it can point to the underlying mechanism for the disparity[8,19,21] and can lead to the identification of modifiable factors and potential interventions. The effect of sex on stroke risk has not been well characterized among Africans, and the existing studies have not provided conclusive evidence.[20,22–24] Therefore, we sought to compare the effect sizes of vascular risk factors of stroke by sex among West Africans.

Methods

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Study Design

The SIREN study (Stroke Investigative Research and Educational Networks) is a multicenter case-control study involving 15 sites in Ghana and Nigeria (Table I in the online-only Data Supplement). The study commenced in August 2014, and the study protocol has been published.[25] In brief, stroke cases included consecutive consenting adults aged >18 years with first clinical stroke within 8 days of current symptom onset or last seen without deficit with neuroimaging confirmation with computerized tomography or magnetic resonance imaging scan within 10 days of symptom onset (Table II in the online-only Data Supplement for eligibility criteria).

Controls were consenting stroke-free adults recruited via robust control recruitment from the community and participating hospitals. Stroke-free status was confirmed using the 8-item questionnaire for verifying stroke-free status validated in 3 major languages spoken in West Africa (Ashanti, Yoruba, and Hausa).[26] Controls were matched by age (±5 years), sex, and ethnicity to minimize the potential confounding effect of these variables on the relationship between stroke and the main environmental risk factors (Tables II through IV in the online-only Data Supplement). Ethical approval was obtained for all study sites, and informed consent was obtained from all subjects. To minimize investigation bias, cost of neuroimaging, echocardiography, carotid Doppler, lipid profiling, and other investigations were covered for all eligible patients who could not afford these procedures. Ghana has universal health coverage, whereas in Nigeria, patients make out-of-pocket payments for all investigations and treatments.

Stroke diagnosis and phenotyping (Figure I in the online-only Data Supplement) were based on clinical evaluation and brain neuroimaging (computerized tomography or magnetic resonance imaging), electrocardiography, transthoracic echocardiography, and carotid Doppler ultrasound performed according to the standard operating procedures. Ischemic stroke was subtyped clinically using the Oxfordshire Community Stroke Project criteria,[27] and presumed etiological subtypes were defined using the TOAST (Trial of ORG 10172 in Acute Stroke Treatment)[28] and the ASCO (Atherosclerosis, Small Vessel Disease, Cardiac Source, and Other)[29] criteria. Intracerebral hemorrhage was classified etiologically into SMASH-U (Structural, Medication-Related, Amyloid Angiopathy, Systemic/Other Disease, Hypertension and Undetermined Causes).[30] Stroke severity was measured by the modified National Institutes of Health Stroke Scale and the Stroke Levity Scale.[31]

Data Collection

We collected basic demographic and lifestyle data including ethnicity and native language of the subjects and their parents, socioeconomic status, cardiovascular risk profile, and dietary patterns. We used validated instruments to assess physical activity, stress, depression, cigarette smoking, and alcohol use.[32] We collected blood samples early in the morning after overnight fast in cases (postacute phase when fasting is feasible) and controls for measurement of blood glucose and lipid profile (total cholesterol, LDL-C [low-density lipoprotein cholesterol], HDL-C (high-density lipoprotein cholesterol), triglyceride, and glycosylated hemoglobin) using a uniform standard operating procedure across all study sites.

Definition of Risk Factors

- Hypertension: Blood pressure (BP) was recorded at baseline and daily for 7 days or until death. A cutoff of ≥140/90 mm Hg for up to 72 hours after stroke, a history of hypertension, or use of antihypertensive drugs before stroke or >72 hours after stroke was regarded as an indicator of hypertension. Adjustments to systolic BP based on reported associations between premorbid BP and acute poststroke BP in the OXVASC (Oxford Vascular Study) were also applied in sensitivity analyses.[33] Definition of hypertension in controls was self-reported history of hypertension or use of antihypertensive drugs or average BP at first clinical encounter ≥140/90 mm Hg.
- Diabetes mellitus (DM) was defined based on history of DM, use of medications for DM, a glycosylated hemoglobin >6.5%, or a fasting blood glucose levels > 7.0 mmol/L at first encounter in controls or measured after the postacute phase in cases due to the known acute transient elevation of glucose as a stress response after stroke.[34]
- Dyslipidemia was defined as total cholesterol ≥5.2 mmol/L, HDL-C ≤1.03 mmol/L, triglyceride ≥1.7 mmol/L, or LDL-C ≥3.4 mmol/L according to the NCEP guidelines (National Cholesterol Education Program) or use of statin before stroke onset.[35]
- Cardiac disease was defined based on history or current diagnosis of atrial fibrillation, cardiomyopathy, heart failure, ischemic heart disease, and rheumatic heart disease. Cases had electrocardiography and echocardiography done to ascertain diagnosis where feasible.
- Obesity: We assessed both waist-to-hip ratio and body mass index. Individuals were classified using the World Health Organization guidelines using cutoffs of 94 cm (men) and 80 cm (women) for waist circumference, 0.90 (men) and 0.85 (women) for waist-to-hip ratio, and 30 kg/m^2 for body mass index (obesity).[36]
- Individuals were classified as physically active if they were regularly involved in moderate exercise (walking, cycling, or gardening) or strenuous exercise (jogging, football, and vigorous swimming) for 4 hours or more per week.[32]
- Dietary history included regularity of intake of food items such as meat, fish, green leafy vegetables, addition of salt at table, nuts, sugar, and other local staple food items. Regular intake was defined as intake on daily, weekly, or at least once monthly versus none in a month.
- Alcohol use was categorized into current users (users of any form of alcoholic drinks) or never/former drinker, while alcohol intake (or drinking) was categorized as low drinkers (1–2 drinks per day for female and 1–3 drinks per day for male) and high drinker (>2 drinks per day for female and >3 drinks per day for male).
- Smoking status was defined as current smoker (individuals who smoked any tobacco in the past 12 months) or never/former smoker.[32]
- For psychosocial risk factors, we adapted measures of psychosocial stress and depression in the INTERSTROKE study.[32]
- Family history of cardiovascular risk/diseases was defined based on self-reported history of any of hypertension, DM, dyslipidemia, stroke, cardiac disease, or obesity in participants' father, mother, sibling, or second-degree relative.

Statistical Analysis

We assessed the bivariate association between risk factors and stroke status (case versus control) using McNemar test for paired categorical outcomes with stratification by sex (male versus female). Mantel-Haenszel χ^2 is used to compare categorical variables. Further analysis to determine the adjusted associations between the risk factors and stroke occurrence for the total sample and stratified by sex were made using conditional logistic regression with adjustment for potential confounders that were not used in the matching except baseline age was included to adjust for residual confounding due to the nonexact age matching. We have also tested for the interaction between sex and each of the covariates. The adjusted models included selected covariates depending on whether they are confirmed confounders in the bivariate analysis and considerations from the literature on stroke risk factors. In addition, the final adjusted models were assessed for collinearity using variance inflation factor and goodness of fit using residual analysis, Pearson χ^2, and deviance statistics. We fixed the type I error rate at 5%, and no adjustment was made for fitting multiple models to arrive at the final model.

The odds ratio and 95% CI in the final models were estimated using conditional likelihood. We calculated the adjusted population attributable risks (PARs) with their respective 95% CI for each exposure variable included in the best-fitted adjusted models and a composite PAR for all risk factors. The PARs were estimated as the proportion of the risk of the stroke in the population that is attributable to the individual risk factors (ie, the proportion of cases that would not occur in the population if the factor were eliminated).[37] The 95% CI for the PAR was obtained using the AF R-package,[38] where the variance is estimated via the delta method. The advantage of the AF package is it allows for empirical variance estimator to be used in building the 95% CI. Composite PARs for the dominant risk factors for stroke and sex were calculated using the ATTRIBRISK R package with its 95% CI computed via the bootstrap method. All statistical tests of hypotheses are 2-sided. Statistical analyses and graphics were produced with SAS 9.4 and R statistical program (version 3.4.2)

Results

Demographic and Clinical Characteristics

Out of 2118 stroke cases, 1193 (56.3%) were males. The mean±SD age of males compared with females was 58.09±13.16 versus 60.15±14.53, $P \leq 0.0001$. Compared with males, females had lower educational attainment, were less likely to earn >$100 a month, and used alcohol less as shown in Table 1.

Risk Factors for Stroke by Sex

The 5 shared modifiable risk factors associated with stroke occurrence with adjusted odds ratios (95% CI) among females and males, respectively, were hypertension [29.95 (12.49–71.77) versus 16.10 (9.19–28.19)], dyslipidemia [2.08 (1.42–3.06) versus 1.83 (1.29–2.59)], DM [3.18 (2.11–4.78) versus 2.19 (1.53–3.15)], stress in the preceding 2 weeks of stroke [2.34 (1.48–3.67) versus 1.61 (1.07–2.43)], and low consumption of green leafy vegetables [2.92 (1.89–4.50) versus 2.00 (1.33–3.00)], Table 2, Figures II and III in the online-only Data Supplement. Furthermore, cardiac disease [1.82 (1.00–3.27) versus 1.75 (0.97–3.170)] for stroke occurrence did not show a statistically significant difference, while cigarette smoking, high salt, higher income, and meat consumption were independently associated with stroke among males. Compositely, 6 modifiable factors, hypertension, dyslipidemia, DM, cardiac diseases, stress and low consumption of green leafy vegetables, were associated with a combined PAR of 99.1% (96.0%–99.8%) among females, whereas 9 factors, hypertension, dyslipidemia, DM, physical inactivity, tobacco smoking, stress, table added salt, low consumption of green leafy vegetables, and regular meat consumption, accounted for a PAR of 98.3% (97.1%–99.2%) among males. Tests for interactions between sex and individual risk factors were significant only for monthly income and table added salt (Table 2).

There were intercountry differences in the effect sizes between the sexes, for instance, Nigerian men had higher incomes and consumed more red meat than Ghanaian males. Ghanaian women had higher effect sizes for hypertension, low consumption of green leafy vegetable, low physical activity, and lower effect of stress than Nigerian females (Table V in the online-only Data Supplement). Hypertension had a greater effect size in females than in males using different definitions in sensitivity analyses (Table VI in the online-only Data Supplement).

Stroke Types by Sex

Ischemic stroke was more common among females at 72.4% versus 64.0% among males, $P<0.001$. Partial anterior circulation infarction strokes were more common among males (35.7%) than among females (27.9%), whereas lacunar infarctions were more frequent among females (45.7%) than among males (38.3%) using the Oxfordshire Community Stroke Project classification. Etiologic subtypes of ischemic stroke according to ASCO and TOAST classification by sex are shown in Table 3. Hypertension-related hemorrhagic stroke was more common among males than among females. Strokes were more severe among women than among men.

Discussion

We have characterized the similarities and differences in the effect sizes of risk factors associated with stroke occurrence by sex among West Africans in the largest cohort of patients with stroke in sub-Saharan Africa. Six potentially modifiable risk factors, hypertension, dyslipidemia, DM, cardiac diseases, stress, and low consumption of green leafy vegetables, were independently associated with stroke occurrence among females. Male West Africans had a wider repertoire of factors associated with stroke occurrence than females with effect sizes of shared vascular risk factors being stronger among females. Overall, hypertension was the most dominant risk factor associated with a high odds ratio of 16.1 among males and 30.0 among females; however, our sensitivity analyses using 4 different definitions for hypertension produced estimates that ranged between 5.3 and 17.4 for males and 4.2 and 32.4 for females. Although effect sizes of risk factors overlapped, tests for interactions between sex and individual risk factors were significant only for monthly income and added table salt.

Traditional/Sociocultural Risk Factors

Beyond the differences in the effect sizes, the traditional risk factors of hypertension, DM, and dyslipidemia were associated with stroke in both males and females consistent

Table 1. Demographic and Variables for Stroke by Sex (Cases Versus Controls)

	Cases			Controls		
Variable	Women, N (%)	Men, N (%)	*P* Value	Women, N (%)	Men, N (%)	*P* Value
Total	925	1193		925	1193	
Age <50 y	209 (22.6)	306 (25.6)	0.1041	238 (25.7)	341 (28.6)	0.1440
No education	252 (27.2)	91 (7.6)	<0.0001	295 (31.9)	117 (9.8)	<0.0001
Income <100$	499 (53.9)	379 (31.7)	<0.0001	548 (59.2)	588 (49.3)	<0.0001
Hypertension	872 (94.3)	1125 (94.3)	0.7645	570 (61.6)	637 (53.4)	<0.0001
Dyslipidemia	743 (80.3)	915 (76.7)	0.0527	574 (62.1)	723 (60.6)	0.5276
Diabetes mellitus	377 (40.7)	419 (35.1)	0.008	132 (14.3)	150 (12.6)	0.2559
Cardiac Disease	120 (12.9)	128 (10.7)	0.1131	54 (5.8)	55 (4.6)	0.2068
WH raised	716 (77.4)	821 (68.8)	<0.0001	632 (68.3)	657 (55.1)	<0.0001
No physical activity	52 (5.6)	45 (3.8)	0.0444	28 (3.0)	21 (1.8)	0.0553
Tobacco use in 12 mo	7(0.8)	60(5.0)	<0.0001	1 (0.1)	26 (2.2)	<0.0001
Used alcohol before	174 (18.8)	583 (48.8)	<0.0001	152 (16.4)	514 (43.1)	<0.0001
Stressed	185 (20.0)	247 (20.7)	0.7249	113 (12.2)	163 (13.6)	0.3415
Depressed	70 (7.6)	88 (7.4)	0.8229	59 (6.4)	70 (5.8)	0.5883
Cardiovascular disease in family	366 (39.6)	481 (40.3)	0.6518	273 (29.5)	330 (27.7)	0.3883
Added table salt very often	62 (6.7)	108 (9.0)	0.0471	33 (3.6)	83 (6.9)	0.0006
Green vegetable consumption ≤1 per month	313 (33.8)	399 (33.4)	0.7865	219 (23.7)	289 (24.2)	0.3303
Greens weekly	367 (39.6)	454 (38.0)		286 (30.9)	366 (30.6)	
Greens daily	168 (18.1)	208 (17.4)		343 (37.1)	406 (34.0)	
Confectionary consumption	239 (25.8)	351 (29.4)	0.0113	263 (28.4)	411(34.4)	0.0002
Meat consumption	692 (74.8)	905 (75.8)	0.0606	624 (67.4)	894 (74.9)	<0.0001

WH indicates waist-hip ratio.

with previous findings.[8,20] The effect size of association between cardiac diseases and stroke occurrence reached statistical significance among females but not among males. There are hints of potential differences in lifestyle and dietary practices by sex that may influence stroke occurrence via a nexus of cultural and socioeconomic factors. For instance, male patients with stroke reported a higher proclivity to adding salt at table and consuming meat more regularly than females.[39] In addition, males were more likely to consume alcohol and smoke cigarette. We found associations between higher income among males and stroke, while low educational attainment and stroke risk was observed among females. It has been observed that relatively affluent, well-educated population may have difficulty in identifying and avoiding high-salt foods even if they perceive it is a health issue.[40,41] Higher-salt consumption has been associated with stroke occurrence[5,42]; however, the mechanistic pathways for this association are not clear but have been posited to be either indirectly via effects on BP or via yet-to-be-defined alternative mechanisms.

Role of Stress

Stress was independently associated with stroke occurrence in both sexes. However, the effect size and PARs were higher among females than among males. Despite the prevalence and potency of this risk factor, little is known about the mechanisms that link stress with stroke.[43] Interestingly, a recent study has shed light on the role of chronic stress and creation of an atherosclerotic milieu via elaboration of vasculotoxic and proatherogenic cytokines.[44] The resting metabolic activity within the amygdala is significantly associated with the risk of developing cardiovascular disease independently of established cardiovascular risk factors. Furthermore, the link between amygdala activity and cardiovascular disease events is posited to be mediated by arterial inflammation.[44]

Stroke Type/Subtypes

There were differences in proportions of primary stroke types by sex. The female participants were older and more likely to have ischemic stroke. With advancing age, ischemic stroke is more likely than hemorrhagic stroke and vice versa.[45] Males significantly had more hemorrhagic strokes causally associated with hypertension than females, but no significant differences in etiologic subtypes of ischemic stroke were observed. Intriguingly, although hemorrhagic strokes, which are often more severe, were more common among males than among females and the usually less

Table 2. Odds Ratio and Population Attributable Risk With 95% CI Estimates of Stroke Risk Factors by Sex

Label	Female		Male		Interaction Between Sex and Risk Factor
	Odds Ratio (95% CI)	PAR (95% CI)	Odds Ratio (95% CI)	PAR (95% CI)	*P* Value*
Age ≥50 y	7.93 (2.09 to 29.98)	67.5 (56.5 to 78.4)	3.20 (0.98 to 10.46)	51.1 (37.9 to 64.4)	0.13
Education	1.33 (0.85 to 2.09)	18.6 (–9.4 to 46.7)	1.46 (0.76 to 2.80)	29.9 (–9.2 to 69.2)4	0.9
Monthly income >$100 (USD)	0.85 (0.59 to 1.24)	–7.4 (–35.8 to 21.1)	1.87 (1.35 to 2.58)	31.4 (18.4 to 44.4)	0.03
Hypertension	29.95 (12.49 to 71.77)	92.7 (89.7 to 95.7)	16.10 (9.19 to 28.19)	89.7 (85.2 to 94.2)	0.21
Dyslipidemia	2.08 (1.42 to 3.06)	41.6 (26.6 to 56.5)	1.83 (1.29 to 2.59)	34.8 (19.7 to 49.8)	0.56
Diabetes mellitus	3.18 (2.11 to 4.78)	27.2 (21.0 to 33.2)	2.19 (1.53 to 3.15)	18.1 (10.9 to 25.2)	0.41
Cardiac disease	1.82 (1.00 to 3.27)	5.1 (0.30 to 9.8)	1.75 (0.97 to 3.17)	4.6 (–0.9 to 10.2)	0.61
Raised waist-to-hip ratio	1.69 (1.07 to 2.68)	36.1 (5.8 to 66.4)	1.35 (0.96 to 1.89)	19.1 (4.7 to 35.1)	0.38
No physical activity	2.02 (0.90 to 4.52)	2.8 (0.2 to 5.4)	2.70 (0.77 to 9.46)	2.2 (–0.3 to 4.7)	0.92
Stress	2.34 (1.48 to 3.67)	14.3 (6.3 to 22.2)	1.62 (1.07 to 2.43)	9.2 (2.1 to 16.3)	0.21
Family history of cardiovascular diseases	1.44 (0.97 to 2.14)	11.9 (–4.2 to 28.1)	1.19 (0.84 to 1.68)	6.9 (–6.2 to 20.1)	0.33
Sprinkled salt	6.06 (2.23 to 16.44)	7.4 (5.6 to 9.3)	1.37 (0.78 to 2.40)	2.9 (–0.5 to 6.4)	0.02
Green leafy vegetables	2.92 (1.89 to 4.50)	20.2 (14.9 to 25.4)	2.00 (1.33 to 3.00)	15.6 (8.6 to 22.7)	0.18
Confectionary sugar/syrups	1.34 (0.92 to 1.95)	7.5 (–0.4 to 15.6)	1.07 (0.76 to 1.50)	2.3 (–8.5 to 13.2)	0.30
Meat	1.75 (1.17 to 2.62)	35.4 (16.2 to 54.6)	1.38 (0.89 to 2.14)	23.5 (–11.8 to 58.8)	0.46
Composite PAR		99.1 (98.3 to 99.6)		97.2 (94.9 to 98.7)	

PAR indicates population attributable risk.
**P* value from conditional logistic regression for the interaction between sex and each risk factor.

severe lacunar ischemic strokes were more common among females, we found overall that females had more severe strokes at presentation. The striking differences observed between males and females with regards to primary stroke types, Oxfordshire Community Stroke Project stroke classification, etiologic subtypes of hemorrhagic strokes, and stroke severity are quite significant. First, differential distribution and impact of risk factors may account for the differences in primary stroke types and severity.[46] There is preliminary evidence[4,47] in support of a genetic basis for the sex disparity in stroke occurrence; thus, further studies are needed to elucidate the sex-specific genetic mechanisms underlying the pathobiology of stroke and its different subtypes.[11,19] Several studies have shown differing incidences for ischemic versus hemorrhagic stroke by sex.[11,19] Second, preventive measures with their associated economic impacts might depend on the specific strokes being targeted for prevention. For instance, given that females tended to have more severe strokes in our study, it might be useful to explore further and identify sex-specific risk associations for severe strokes for evidence-based prevention strategies.

Biological Differences

The biological and social explanations for these observations require further investigations. However, the influence of estrogen and testosterone on the endothelium and the vascular system, the role of risk factors unique to women such as the use of oral contraceptives, hormone replacement therapy, and pregnancy, systemic delays in the recognition, and insufficient treatment of conventional stroke risk factors in women have all been considered as probable explanations.[19] Efforts to characterize the possible role of these different factors have been hampered by the paucity of data on sex differences in age-specific stroke incidence, as outlined in systematic reviews.[11,19] The inherent difficulties in conducting long-term longitudinal follow-up cohort incidence studies and the persistent misperception that stroke is a rare disease in women may in part be responsible for the paucity of available data.

Strengths and Limitations

This is one of the largest studies to examine the impact of sex on factors associated with stroke risk among West Africans. Previous studies in this population have been limited by sample size and had no control group. A limitation of the case-control design is that causality between putative risk factors and event/outcome outcomes cannot be established. However, because control participants were recruited predominantly from the community, a health volunteer effect cannot be entirely ruled out as influencing the effect sizes observed. We performed individual matching of cases to controls (age, sex, and ethnicity not risk factor status) in a 1:1 fashion and used conditional logistic regression analysis to attain unbiased odds ratios. Due to the severity of strokes, responses to questions on lifestyle and dietary behavioral information were obtained from 1621 valid proxies with the remainder from patients themselves. We have previously

Table 3. Stroke Types and Subtypes, Stroke Levity Scale, and Severity of Stroke by Sex

Parameters	Female, n (%); N= 922	Male, n (%); N= 1190	*P* Value*
Stroke type			<0.001
Ischemic	668 (72.4)	762 (64.0)	
Hemorrhagic	254 (27.6)	428 (36.0)	
OCSP classification			0.0264
Total anterior circulation infarction	78 (14.1)	91 (14.2)	
Partial anterior circulation infarction	197 (35.7)	179 (27.9)	
Posterior circulation infarction	63 (11.4)	78 (12.2)	
Lacunar infarction	214 (38.3)	293 (45.7)	
ASCO classification			0.1899
Atherosclerosis	109 (28.0)	100 (20.3)	
Small-vessel disease	200 (51.4)	293 (59.4)	
Cardioembolic	66 (16.9)	87 (17.6)	
Others	14 (3.6)	13 (2.6)	
TOAST Ischemic Stroke Subtypes			0.299
Large artery atherosclerosis	211 (37.5)	203 (30.8)	
Cardioembolism	39 (6.9)	63 (9.6)	
Small-vessel disease	195 (34.6)	261 (39.7)	
Other determined cause (dissection, vasculitis, others)	1 (0.1)	0 (0.0)	
Undetermined cause (≥2 causes identified, negative evaluation, incomplete evaluation)	117 (20.8)	131 (19.9)	
SMASH-U hemorrhagic subtypes			0.0013
Structural	15 (6.9)	6 (1.6)	
Medication related	0 (0.0)	3 (0.8)	
Amyloid angiopathy	5 (2.3)	1 (0.3)	
Systemic disease	0 (0.0)	1 (0.3)	
Hypertension	193 (88.9)	335 (94.4)	
Undetermined	4 (1.8)	9 (2.5)	
Stroke Levity Scale			0.0065
Mild	108 (12.9)	202 (18.9)	
Moderate	287 (34.4)	340 (31.8)	
Severe	439 (52.6)	526 (49.3)	
Modified NIHSS			0.0099
1–5	105 (13.9)	182 (18.6)	
6–14	293 (38.9)	382 (39.1)	
15–25	243 (32.3)	287 (29.4)	
>25	112 (14.8)	126 (12.9)	

ASCO indicates Atherosclerosis, Small Vessel Disease, Cardiac Source, and Other; NIHSS, National Institutes of Health Stroke Scale; OCSP, Oxfordshire Community Stroke Project; SMASH-U, Structural, Medication-Related, Amyloid Angiopathy, Systemic/Other Disease, Hypertension and Undetermined Causes; and TOAST, Trial of ORG 10172 in Acute Stroke Treatment.

**P* Mantel-Haenszel χ^2.

reported that the associations observed among proxies were in the same direction as for patients with direct assessment.[5]

Conclusions

Overall, risk factors for stroke occurrence are commonly shared by both sexes in West Africa favoring concerted interventions for stroke prevention in the region.

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Disclosures

None.

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