Journal of the American Heart Association

ORIGINAL RESEARCH

Cardiometabolic Risk Factors and Preclinical Target Organ Damage Among Adults in Ghana: Findings From a National Study

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BACKGROUND: Although sub-Saharan Africa has a high prevalence of cardiovascular diseases (CVDs), there remains a lack of systematic and comprehensive assessment of risk factors and early CVD outcomes in adults in sub-Saharan Africa.

METHODS AND RESULTS: Using a stratified multistage random sampling method, we recruited 1106 men and women, aged >18 years, from the general population in Ghana to participate in a national health survey from 2016 to 2017. In Ghanaian adults, the age-standardized prevalence of known CVD risk factors was 15.1% (95% CI, 12.9%–17.3%) for obesity, 6.8% (95% CI, 5.1%–8.5%) for diabetes mellitus, 26.1% (95% CI, 22.9%–29.4%) for hypertension, and 9.3% (95% CI, 7.1%–11.5%) for hyperuricemia. In addition, 10.1% (95% CI, 7.0%–13.2%) of adults had peripheral artery disease, 8.3% (95% CI, 6.7%–10.0%) had carotid thickening, 4.1% (95% CI, 2.9%–5.2%) had left ventricular hypertrophy, and 2.5% (95% CI, 1.5%–3.4%) had chronic kidney disease. Three CVD risk factors appeared to play prominent roles in the development of target organ damage, including obesity for peripheral artery disease (odds ratio [OR], 2.22; 95% CI, 1.35–3.63), hypertension for carotid thickening (OR, 1.92; 95% CI, 1.22–3.08), and left ventricular hypertrophy (OR, 5.28; 95% CI, 2.55–12.11) and hyperuricemia for chronic kidney disease (OR, 5.49; 95% CI, 2.84–10.65).

CONCLUSIONS: This comprehensive health survey characterized the baseline conditions of a national cohort of adults while confirming the prevalence of CVD risk factors, and early CVD outcomes have reached epidemic proportions in Ghana. The distinct patterns of risk factors in the development of target organ damage present important challenges and opportunities for interventions to improve cardiometabolic health among adults in Ghana.

Key Words: adults ■ cardiovascular disease ■ Ghana ■ risk factors ■ target organ damage

Globally, cardiovascular diseases (CVDs) remain the leading cause of death. However, the CVD burden has not changed uniformly across world regions of different socioeconomic characteristics. Over the past decades, CVD mortality has dramatically declined in high-income countries, whereas it

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Supplementary Material for this article is available at https://www.ahajournals.org/doi/suppl/10.1161/JAHA.120.017492

Preprint posted on MedRxiv May 3, 2020. https://doi.org/10.1101/2020.04.28.20082446.

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For Sources of Funding and Disclosures, see pages 13 and 14.

JAHA is available at: www.ahajournals.org/journal/jaha

CLINICAL PERSPECTIVE

What Is New?

- Cardiometabolic risk factors previously identified in populations of the developed countries are now common among adults in Ghana.
- However, there are regional- and sex-specific patterns of risk factors in Ghana that are different from populations in the West.
- Obesity, hypertension, and hyperuricemia appear to play the most prominent roles in the early stage of cardiovascular disease development among Ghanaian adults.

What Are the Clinical Implications?

- Prevention strategies targeting the risk factors with the strongest association with early cardiovascular disease outcomes may reduce the cardiovascular disease burden in Ghana.
- As globalization is accelerating the epidemiologic transition in different countries and shifting much of the cardiovascular disease burden to low- and middle-income countries, disparities in health outcomes are expected to affect populations in low- and middle-income countries, particularly in sub-Saharan Africa.
- Findings from this national survey will establish a national cohort with continued follow-up to ascertain clinical outcomes and guide the development of specific prevention and intervention strategies to improve cardiometabolic health outcomes among populations in sub-Saharan Africa.

Nonstandard Abbreviations and Acronyms

DBP	diastolic blood pressure
GBD	Global Burden Disease
GHS	Ghana Heart Study
LVM	left ventricular mass
SSA	sub-Saharan Africa
TC	total cholesterol
TOD	target organ damage

has increased in low- and middle-income countries.[1] According to the GBD (Global Burden Disease) study in 2015, the age-standardized CVD prevalence is 6304 per 100 000 globally, with the highest rate in western sub-Saharan Africa (SSA) (9475 per 100 000).[2] There is a paucity of data on CVD epidemiological features, prevention, and treatment in many low- and middle-income countries, especially in SSA.[3] Thus, our understanding of CVD is disproportionately informed by studies conducted in high-income countries, findings of which may not be directly applicable to people in low- and middle-income countries. Given that the genetic characteristics and CVD risk factors differ across ethnicities and regions, systemic and comprehensive studies on CVD and the relevant risk factors in SSA are urgently needed to guide proper allocation of healthcare resource and public health policy for the region.

CVD is a multistage pathogenetic disorder spanning over a lifetime, often affected by a cluster of risk factors, including obesity, dyslipidemia, diabetes mellitus, and hypertension. Preclinical pathological changes in blood vessels, the heart, and kidneys, termed target organ damage (TOD), are common and typically lead to end-stage organ failure in the absence of intervention.[4] Although often asymptomatic, preclinical TOD can be considered an intermediate end point for CVD events in the pathogenesis of CVD. There is now a body of accumulating evidence that indicates CVD progression can be modified by earlier interventions.[5] In Ghana, the prevalence of some of the typical CVD risk factors has reached the levels in the developed countries.[6,7] Without adequate prevention and control measures, these risk factors are likely to transition into severe CVD burden in Ghanaian society.

To provide a comprehensive assessment of CVD risk factors and TOD prevalence, we conducted a population-based study in Ghana to guide the future development of intervention strategies to improve cardiometabolic health. Specifically, we investigated whether and to what extent the known CVD risk factors are associated with preclinical vascular, cardiac, and renal TOD among adults in Ghana.

METHODS

Data Sharing

Original raw data cannot be shared because of confidentiality agreement. Analytical codes and data set can be shared via standard data transfer agreement approved by Ghanaian institutions and Guangdong Provincial People's Hospital.

Study Participants

In this GHS (Ghana Heart Study), we used a 3-stage, stratified random sampling strategy to recruit a nationally representative sample of Ghanaian adults, aged ≥18 years. We first identified geographic regions (southern, middle, and northern) within Ghana where participants were selected to represent the southern (Greater Accra and Central regions), middle (Ashanti regions), and northern (Northern regions) populations in the country. Each of the 4 regions was further divided into urban areas and rural areas, according to

the most updated census data, from which one urban and one rural community were selected by simple random sampling method. This first stage leads to the selection of Dansoman (urban) and Damfa (rural) in the Greater Accra region, Kwadaso Estate (urban) and Dominase (rural) in the Ashanti region, Cape Coast (urban) and Awutu-Okwampa (rural) in the Central region, and Zogbeli (urban) and Sanzirugu (rural) in the Northern region. The second stage of sample selection consisted of households. In a given community, a listing of all households was prepared, and a subsample of these was selected by a systematic sampling method. The third stage of sample selection consisted of people within the selected households. All members aged ≥18 years within a household were listed, and a subsample of individuals was selected by simple random sampling method based on sex, age, ethnicity, and income. The exclusion criteria were excluding participants who were pregnant, had type 1 diabetes mellitus, or refused to give consent.

The sample size was calculated using the primary clinical target of hypertension with an estimated prevalence of 25.4% and a second target of smoking with an estimated prevalence of 4.3% (PASS 14.0 software). The population of Ghana was estimated at 24 658 823 on the basis of 2010 Population and Housing Census in Ghana.[8] We determined sample size for the various regions according to the population of the specific regions (the sample size was estimated to be 939+a 10% attrition rate, leading to 1032 participants). In addition, we also included 74 more participants who met the inclusion criteria in this study. All these 1106 participants gave written consent and completed the study. All procedures were performed according to the study protocol approved by the Committee on Human Research Publication and Ethics of School of Medicine and Dentistry, the Kwame Nkrumah University of Science and Technology, Kumasi, Ghana, and Research Ethics Committee of Guangdong Provincial People's Hospital, Guangdong Academy of Medical Sciences, Guangzhou, China. Informed consent was obtained from all the participants. The objectives and nature of the study were explained to all participants. This study was registered at http://www.chictr.org.cn as ChiCTR1800017374.

Cardiometabolic Risk Factor Measurements

A standard questionnaire was used to obtain information on demographics, personal and family medical history, and lifestyle risk factors, including smoking, drinking, and physical activity. Anyone who smoked any tobacco products, such as cigarettes, cigars, and pipes, either daily or occasionally, was described as a smoker. Drinking was defined as consumption of alcoholic drink, such as beer, wine, or spirit, either daily or occasionally. Adequate physical activity was defined as at least 150 minutes per week of moderate or vigorous activities, such as brisk walking.

Body weight, height, and waist circumference were measured with light clothes and bare feet. Body mass index was calculated as weight in kilogram divided by the square of the height in meters. Blood pressure was measured using the OMRON M6 devices with appropriate cuff sizes. Three blood pressure readings were taken from the left arm, with participants in the sitting position after 10 minutes of inactivity. The mean of the recorded readings was taken as the participant's blood pressure. Fasting plasma samples were collected to measure glucose, hemoglobin A1c, total triglyceride, total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol, lipoprotein (a), hs-CRP (high-sensitivity C-reactive protein), creatinine, and uric acid. The criteria used for diagnosing obesity, diabetes mellitus, hypertension, dyslipidemia, high hs-CRP, and hyperuricemia were provided in Table S1.

Preclinical TOD Measurements

The measurements for preclinical vascular, cardiac, and renal TOD were conducted by well-trained cardiologists using calibrated machines according to the relevant guidelines. For vascular TOD, ankle-brachial index was measured for each participant using the Boso (Bosch & Sohn, Germany) ankle-brachial index device. Carotid ultrasound was performed for both carotid arteries using the GE VIVID Q ultrasound system equipped with a 9L-RS 3.1- to 10.0-MHz linear probe to determine carotid intima-media thickness. For cardiac TOD, each participant had a transthoracic echocardiography examination by a cardiologist using the GE VIVID Q ultrasound system equipped with M4S-RS 1.5- to 3.5-MHz sector and linear probe. The left ventricular internal dimensions were measured using 2-dimensional guided M-mode imaging and used to estimate left ventricular mass (LVM). LVM index was calculated by dividing LVM by body surface area (see Data S1 for details). Renal TOD was evaluated using the estimated glomerular filtration rate, an important chronic kidney disease (CKD) marker. The criteria used for diagnosing peripheral artery disease (PAD), carotid intimal thickening, left ventricular hypertrophy (LVH), and CKD are provided in Table S1.

Statistical Analysis

Basic characteristics, cardiometabolic risk factors, and preclinical TOD measurements were described by sex and ethnicity. In our study, ethnicity was categorized into Akan and others, including Dagomba, Ewe, and Ga. Continuous variables were presented as means

Table 1. Characteristics of Adults Participating in the GHS, 2016 to 2017

Characteristic	Overall	Women	Men	*P*1 Value	Akan	Other Ethnicities	*P*2 Value
Basic characteristics							
No.	1106	642	464		639	467	
Age, mean (SD), y	46.9 (17.2)	47.5 (16.7)	46.1 (17.8)	0.17	47.0 (17.4)	46.8 (16.9)	0.89
Men, n (%)	464 (42.0)	0 (0.0)	464 (100.0)		246 (38.5)	218 (46.7)	0.008
Ethnicity, Akan, n (%)	639 (57.8)	393 (61.2)	246 (53.0)	0.008	639 (100.0)	0 (0.0)	
Region, n (%)				0.006			<0.001
Accra	352 (31.8)	187 (29.1)	165 (35.6)		172 (26.9)	180 (38.5)	
Ashanti	353 (31.9)	221 (34.4)	132 (28.4)		305 (47.7)	48 (10.3)	
Central	197 (17.8)	127 (19.8)	70 (15.1)		161 (25.2)	36 (7.7)	
Northern	204 (18.4)	107 (16.7)	97 (20.9)		1 (0.2)	203 (43.5)	
Community, urban, n (%)	889 (80.4)	504 (78.5)	385 (83.0)	0.08	520 (81.4)	369 (79.0)	0.37
Education, n (%)				<0.001			0.04
Primary school or less	478 (43.2)	340 (53.0)	138 (29.7)		258 (40.4)	220 (47.1)	
Middle school	452 (40.9)	232 (36.1)	220 (47.4)		268 (41.9)	184 (39.4)	
College or more	176 (15.9)	70 (10.9)	106 (22.8)		113 (17.7)	63 (13.5)	
Smoking, n (%)	44 (4.0)	8 (1.2)	36 (7.8)	<0.001	28 (4.4)	16 (3.4)	0.52
Drinking, n (%)	541 (48.9)	279 (43.5)	262 (56.5)	<0.001	396 (62.0)	145 (31.0)	<0.001
Physical activity ≥150 min/wk, n (%)	180 (16.3)	79 (12.3)	101 (21.8)	<0.001	57 (8.9)	123 (26.3)	<0.001
Measures of adiposity							
Height, mean (SD), cm	164.6 (8.4)	160.4 (6.2)	170.4 (7.5)	<0.001	164.0 (8.3)	165.4 (8.5)	0.13
Weight, mean (SD), kg	69.78 (15.0)	70.7 (16.2)	68.6 (12.9)	0.03	69.4 (14.7)	70.5 (15.2)	0.11
BMI, mean (SD), kg/m^2	25.8 (5.6)	27.4 (6.2)	23.6 (4.1)	<0.001	25.8 (5.6)	25.9 (5.7)	0.25
Obesity, n (%)	237 (21.4)	200 (31.2)	37 (8.0)	<0.001	131 (20.5)	106 (22.7)	0.07
Waist, mean (SD), cm	89.0 (14.3)	92.0 (15.0)	84.7 (12.2)	<0.001	89.6 (14.3)	88.12 (14.3)	0.29
Abdominal obesity, n (%)	405 (36.6)	367 (57.2)	38 (8.2)	<0.001	251 (39.3)	154 (33.0)	0.45
Glucose metabolism							
FBG, median (IQR), mmol/L	4.93 (4.70–5.30)	4.93 (4.60–5.30)	4.93 (4.70–5.40)	0.41	5.00 (4.70–5.40)	4.90 (4.70–5.20)	0.002
HbA1c, median (IQR), %	5.31 (5.00–5.68)	5.31 (5.00–5.70)	5.31 (5.00–5.60)	0.30	5.30 (5.00–5.70)	5.35 (5.00–5.60)	0.09
Diabetes mellitus, n (%)	120 (10.8)	78 (12.1)	42 (9.1)	0.13	83 (13.0)	37 (7.9)	0.01
Blood pressure, mean (SD), mm Hg							
SBP	128.3 (23.0)	126.3 (23.2)	131.0 (22.3)	<0.001	130.5 (23.8)	125.3 (21.4)	<0.001
DBP	78.9 (13.3)	78.7 (13.1)	79.2 (13.6)	0.32	79.9 (13.9)	77.5 (12.3)	0.002

(*Continued*)

Table 1. Continued

Characteristic	Overall	Women	Men	*P*1 Value	Akan	Other Ethnicities	*P*2 Value
Hypertension, n (%)	613 (55.4)	353 (55.0)	260 (56.0)	0.26	383 (59.9)	230 (49.3)	<0.001
Hypertension (JNC7), n (%)	450 (40.7)	272 (42.4)	178 (38.4)	0.37	276 (43.2)	174 (37.3)	0.03
Lipids							
TG, median (IQR), mmol/L	0.96 (0.72–1.24)	0.96 (0.72–1.27	0.95 (0.74–1.20)	0.80	0.97 (0.74–1.28)	0.94 (0.71–1.19)	0.004
TG ≥1.70 mmol/L, n (%)	103 (9.3)	60 (9.3)	43 (9.3)	0.94	70 (11.0)	33 (7.1)	0.03
TC, mean (SD), mmol/L	5.25 (1.22)	5.37 (1.27)	5.08 (1.13)	<0.001	5.37 (1.28)	5.08 (1.13)	<0.001
TC ≥6.22 mmol/L, n (%)	241 (21.8)	172 (26.8)	69 (14.9)	<0.001	174 (27.2)	67 (14.3)	<0.001
LDL-C, mean (SD), mmol/L	3.39 (1.07)	3.49 (1.12)	3.26 (0.98)	0.001	3.47 (1.10)	3.28 (1.01)	0.008
LDL-C ≥4.14 mmol/L, n (%)	214 (19.3)	144 (22.4)	70 (15.1)	0.004	140 (21.9)	74 (15.8)	0.02
HDL-C, mean (SD), mmol/L	1.37 (0.34)	1.40 (0.34)	1.33 (0.34)	0.001	1.41 (0.35)	1.31 (0.31)	<0.001
HDL-C <1.04 mmol/L, n (%)	161 (14.6)	79 (12.3)	82 (17.7)	0.01	75 (11.7)	86 (18.4)	0.004
Dyslipidemia, n (%)	451 (40.8)	278 (43.3)	173 (37.3)	0.07	275 (43.0)	176 (37.7)	0.10
Lipoprotein (a), median (IQR), mg/dL	38.6 (22.0–56.0)	38.7 (22.0–57.0)	38.2 (21.0–55.6)	0.75	38.0 (21.0–56.0)	40.0 (22.0–56.2)	0.34
Lipoprotein (a) ≥60 mg/dL, n (%)	251 (22.7)	149 (23.2)	102 (22.0)	0.73	145 (22.7)	106 (22.7)	0.95
Inflammation							
hs-CRP, median (IQR), mg/L	1.60 (0.60–3.46)	2.00 (0.80–3.80)	1.22 (0.50–2.65)	0.43	1.52 (0.60–3.40)	1.64 (0.70–3.59)	0.14
hs-CRP >3 mg/L, n (%)	321 (29.0)	213 (33.2)	108 (23.3)	0.001	183 (28.6)	138 (29.6)	0.50
Uric acid							
Uric acid, mean (SD), mmol/L	0.30 (0.08)	0.27 (0.07)	0.34 (0.07)	<0.001	0.30 (0.08)	0.30 (0.08)	0.79
Hyperuricemia, n (%)	148 (13.4)	92 (14.3)	56 (12.1)	0.36	87 (13.6)	61 (13.1)	0.92
Vascular TOD							
ABI, mean (SD)	1.01 (0.08)	1.00 (0.08)	1.02 (0.09)	0.001	1.02 (0.08)	1.00 (0.08)	0.006
PAD, n (%)	95 (8.6)	55 (8.6)	40 (8.6)	0.95	51 (8.0)	44 (9.4)	0.40
CIMT, median (IQR), mm	0.78 (0.60–1.00)	0.77 (0.60–1.00)	0.78 (0.60–1.00)	0.05	0.71 (0.60–1.00)	0.80 (0.60–1.00)	0.02
Carotid intimal thickening, n (%)	180 (16.3)	98 (15.3)	82 (17.7)	0.14	96 (15.0)	84 (18.0)	0.12
Cardiac TOD							
LVMI, mean (SD), g/m^2	72.4 (18.8)	68.7 (17.7)	77.5 (19.1)	<0.001	72.9 (19.6)	71.7 (17.6)	0.09

(*Continued*)

Table 1. Continued

Characteristic	Overall	Women	Men	*P1* Value	Akan	Other Ethnicities	*P2* Value
LVH, n (%)	66 (6.0)	47 (7.3)	19 (4.1)	0.03	40 (6.3)	26 (5.6)	0.82
Renal TOD							
Creatinine, mean (SD), μmol/L	77.0 (18.7)	68.3 (12.9)	88.9 (19.1)	<0.001	77.5 (18.9)	76.2 (18.5)	0.002
eGFR, mean (SD), mL/min per 1.73 m^2	91.5 (19.8)	91.8 (19.3)	91.2 (20.6)	0.08	90.2 (20.0)	93.3 (19.5)	0.001
CKD, n (%)	50 (4.5)	26 (4.0)	24 (5.2)	0.32	35 (5.5)	15 (3.2)	0.08

Data are presented as mean (SD) for normally distributed variables, median (IQR) for nonnormally distributed variables, and numbers (percentages) for categorical variables. *P1* is *P* value for sex difference calculated using linear regression model for continuous variables and logistic regression model for categorical variables, with adjustment for age, except for basic characteristics. *P2* is *P* value for ethnicity difference calculated using linear regression model for continuous variables and logistic regression model for categorical variables, with adjustment for age and sex, except for basic characteristics. ABI indicates ankle-brachial index; BMI, body mass index; CIMT, carotid intima-media thickness; CKD, chronic kidney disease; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; FBG, fasting blood glucose; GHS, Ghana Heart Study; HbA1c, hemoglobin A1c; HDL-C, high-density lipoprotein cholesterol; hs-CRP, high-sensitivity C-reactive protein; IQR, interquartile range; JNC7, Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; LDL-C, low-density lipoprotein cholesterol; LVH, left ventricular hypertrophy; LVMI, left ventricular mass index; PAD, peripheral artery disease; SBP, systolic blood pressure; TC, total cholesterol; TG, total triglyceride; and TOD, target organ damage.

and SDs for those with normal distribution or medians and interquartile ranges for those with nonnormal distribution. Categorical variables were shown in percentages. Age-standardized rates were calculated using the population distribution from 2010 Population and Housing Census in Ghana.[8]

The association between categorized risk factors and preclinical TOD was analyzed with the logistic regression model. The model for the association of overall obesity and abdominal obesity with TOD was adjusted for age, sex, ethnicity, living area, education, smoking, and physical activity. For the analysis of other risk factors, body mass index was further controlled in the model. The associations of risk factors with TOD were presented as odds ratios (ORs) and 95% CIs.

We also used restricted cubic spline regression with 3 knots at 10th, 50th, and 90th centiles to detect the possible nonlinear dependency of the relations between cardiometabolic risk factors and TOD measurements. The potential nonlinearity was examined by using a likelihood ratio test comparing the model with only a linear term against the model with linear and cubic spline terms. For the analysis of body mass index with TOD measurements, age, sex, ethnicity, living area, education, smoking, and physical activity were adjusted in the model. For the other risk factors, body mass index was additionally controlled in the model. Stratification analyses by sex were conducted to evaluate sex differences for the risk factor–TOD association.

The proportion of missing data ranged from 0.4% for systolic blood pressure and diastolic blood pressure (DBP) to 21% for carotid intima-media thickness. Missing values were imputed using random forest algorithm with R package missForest.[9] We conducted sensitivity analysis, excluding missing data to test if the associations of risk factors with TOD were robust. All analyses were conducted with the use of R 3.5.2 software (The R Foundation for Statistical Computing).

RESULTS

Basic Characteristics of Study Participants in Ghana

As shown in Table 1, of the 1106 participants aged 46.9±17.2 years, 42% were men, and 58% were Akan. Compared with women, more men were educated higher than middle school (70% versus 47%), were smokers (7.8% versus 1.2%), consumed alcohol (57% versus 44%), and were physically active (22% versus 12%). Compared with the other ethnicities, Akans were more likely to be educated higher than middle school (60% versus 53%) and to consume alcohol (62% versus 31%), and were less physically active (9% versus 26%).

Cardiometabolic Risk Factors of Study Participants in Ghana

As shown in Table 2, in Ghanaian adults, 15.1% (95% CI, 12.9%–17.3%) were overall obese, 25.2% (95% CI, 22.3%–28.0%) had abdominal obesity, 26.1% (95% CI, 22.9%–29.4%) were hypertensive, 6.8% (95% CI, 5.1%–8.5%) had diabetes mellitus, 34.4% (95% CI, 29.9%–39.0%) had dyslipidemia, 9.3% (95% CI, 7.1%–11.5%) had hyperuricemia, and 25.7% (95% CI, 21.5%–29.8%) had an hs-CRP >3 mg/L.

Compared with men, women were significantly more obese (overall obesity: OR, 5.21 [95% CI, 3.62–7.70]; abdominal obesity: OR, 17.50 [95% CI, 12.05–26.09]) and had higher risks of high TC (OR, 2.10; 95% CI, 1.53–2.90), high LDL-C (OR, 1.60; 95% CI, 1.17–2.21), and high hs-CRP (OR, 1.61; 95% CI, 1.23–2.13) after adjustment for age. Compared with the other ethnicities, Akan had higher risks of diabetes mellitus (OR, 1.71; 95% CI, 1.13–2.63), hypertension (OR, 1.75; 95% CI, 1.33–2.30), high total triglyceride (OR, 1.63; 95% CI, 1.06–2.55), high TC (OR, 2.19; 95% CI, 1.60–3.04), and high LDL-C (OR, 1.44; 95% CI, 1.05–1.99) after adjustment for age and sex. Urban participants have higher risks of obesity (OR, 1.85; 95% CI, 1.23–2.84), high TC (OR, 1.80; 95% CI, 1.20–2.75), and high LDL-C (OR, 1.85; 95% CI, 1.21–2.90) than those in rural areas, after adjustment for age and sex. Higher education (middle school or higher) was significantly associated with high TC (OR, 1.67; 95% CI, 1.22–2.30) after adjustment for age and sex.

Preclinical TOD of Study Participants in Ghana

As shown in Table 2, 10.1% (95% CI, 7.0%–13.2%) had PAD, 8.3% (95% CI, 6.7%–10.0%) had carotid thickening, 4.1% (95% CI, 2.9%–5.2%) had LVH, and 2.5% (95% CI, 1.5%–3.4%) had CKD in Ghanaian adults. Women had significantly higher risk of LVH than men (OR, 1.81; 95% CI, 1.06–3.22) after adjustment for age. Akan had lower estimated glomerular filtration rate than other ethnicities (90.2 versus 93.3 mL/min per 1.73 m^2; P=0.001). However, the association between ethnicity and CKD did not reach significant level (OR, 1.80; 95% CI, 0.96–3.53) after adjustment for age and sex.

Association of Cardiometabolic Risk Factors With Preclinical TOD

Among these known risk factors, obesity had the strongest association with PAD (OR, 2.22; 95% CI, 1.35–3.63), independent from age, sex, ethnicity, region, living community, education, smoking,

Table 2. Age-Standardized Prevalence of Cardiometabolic Risk Factors and TOD Among Adults Participating in the GHS, 2016 to 2017

Variable	Overall, % (95% CI)	Women, % (95% CI)	Men, % (95% CI)
Smoking	2.90 (1.91–3.89)	0.93 (0.15–1.72)	5.60 (3.53–7.67)
Drinking	45.91 (40.64–51.18)	38.40 (32.68–44.12)	53.68 (45.06–62.31)
Physical activity ≥150 min/wk	15.47 (12.61–18.33)	9.75 (7.18–12.32)	21.85 (16.68–27.01)
Obesity	15.12 (12.91–17.32)	22.60 (18.92–26.27)	4.91 (3.13–6.69)
Abdominal obesity	25.15 (22.33–27.96)	40.05 (35.25–44.85)	4.60 (2.95–6.25)
Diabetes mellitus	6.77 (5.06–8.49)	7.92 (4.91–10.92)	5.34 (3.41–7.27)
Hypertension	40.69 (36.20–45.17)	38.81 (32.73–44.89)	42.87 (36.12–49.62)
Hypertension (JNC7)	26.13 (22.86–29.39)	26.53 (22.22–30.83)	24.94 (20.13–29.75)
TG ≥1.70 mmol/L	6.75 (4.78–8.71)	7.51 (3.73–11.3)	6.79 (4.60–8.98)
TC ≥6.22 mmol/L	14.98 (12.46–17.5)	17.16 (14.19–20.14)	11.58 (7.79–15.37)
LDL-C ≥4.14 mmol/L	13.93 (11.44–16.42)	14.84 (12.02–17.66)	12.26 (8.35–16.17)
HDL-C <1.04 mmol/L	16.17 (12.52–19.82)	13.53 (8.99–18.08)	18.81 (13.17–24.46)
Dyslipidemia	34.41 (29.85–38.96)	34.84 (28.73–40.95)	33.34 (26.60–40.07)
Lipoprotein (a) ≥60 mg/dL	19.07 (15.80–22.34)	18.23 (14.16–22.29)	19.53 (14.47–24.60)
hs-CRP >3 mg/L	25.66 (21.51–29.81)	29.52 (23.18–35.85)	20.53 (15.14–25.91)
Hyperuricemia	9.28 (7.11–11.45)	11.37 (7.26–15.49)	7.66 (5.43–9.89)
PAD	10.09 (6.96–13.23)	9.96 (5.23–14.69)	10.05 (5.75–14.35)
Carotid hypertrophy	8.33 (6.67–10.00)	8.63 (5.69–11.58)	8.04 (6.12–9.95)
LVH	4.08 (2.93–5.23)	5.39 (3.61–7.17)	2.22 (1.01–3.44)
CKD	2.45 (1.54–3.36)	2.36 (1.22–3.51)	2.38 (1.37–3.39)

CKD indicates chronic kidney disease; GHS, Ghana Heart Study; HDL-C, high-density lipoprotein cholesterol; hs-CRP, high-sensitivity C-reactive protein; JNC7, Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; LDL-C, low-density lipoprotein cholesterol; LVH, left ventricular hypertrophy; PAD, peripheral artery disease; TC, total cholesterol; and TG, total triglyceride; TOD, target organ damage.

Table 3. Association Between Cardiometabolic Risk Factors and Specific TOD Among Adults Participating in GHS, 2016 to 2017

Variable	No.	Case, n (%)	OR (95% CI)	Case, n (%)	OR (95% CI)
		PAD		Carotid Intimal Thickening	
Overall obesity					
No	869	61 (7.0)	Reference	139 (16.0)	Reference
Yes	237	34 (14.3)	2.22 (1.35–3.63)	41 (17.3)	1.18 (0.75–1.84)
Abdominal obesity					
No	701	52 (7.4)	Reference	107 (15.3)	Reference
Yes	405	43 (10.6)	1.62 (0.95–2.80)	73 (18.0)	1.08 (0.69–1.69)
Diabetes mellitus					
No	986	83 (8.4)	Reference	146 (14.8)	Reference
Yes	120	12 (10.0)	1.1 (0.54–2.09)	34 (28.3)	1.22 (0.73–2.00)
Hypertension					
No	493	41 (8.3)	Reference	36 (7.3)	Reference
Yes	613	54 (8.8)	0.95 (0.58–1.55)	144 (23.5)	1.92 (1.22–3.08)
High TG					
No	1003	82 (8.2)	Reference	157 (15.7)	Reference
Yes	103	13 (12.6)	1.57 (0.79–2.92)	23 (22.3)	1.23 (0.69–2.14)
High TC					
No	865	64 (7.4)	Reference	127 (14.7)	Reference
Yes	241	31 (12.9)	1.76 (1.06–2.87)	53 (22.0)	1.25 (0.81–1.93)
High LDL-C					
No	892	65 (7.3)	Reference	135 (15.1)	Reference
Yes	214	30 (14.0)	1.90 (1.15–3.08)	45 (21.0)	1.28 (0.82–1.99)
Low HDL-C					
No	945	82 (8.7)	Reference	150 (15.9)	Reference
Yes	161	13 (8.1)	0.94 (0.48–1.72)	30 (18.6)	1.09 (0.63–1.83)
Dyslipidemia					
No	655	46 (7.0)	Reference	96 (14.7)	Reference
Yes	451	49 (10.9)	1.51 (0.97–2.34)	84 (18.6)	0.98 (0.67–1.43)
High Lp(a)					
No	855	65 (7.6)	Reference	133 (15.6)	Reference
Yes	251	30 (12.0)	1.44 (0.89–2.28)	47 (18.7)	0.92 (0.60–1.41)
High hs-CRP					
No	785	55 (7.0)	Reference	112 (14.3)	Reference
Yes	321	40 (12.5)	1.58 (0.98–2.51)	68 (21.2)	1.04 (0.70–1.55)
Hyperuricemia					
No	958	76 (7.9)	Reference	149 (15.6)	Reference
Yes	148	19 (12.8)	1.46 (0.81–2.53)	31 (20.9)	0.91 (0.55–1.48)
		LVH		CKD	
Overall obesity					
No	869	54 (6.2)	Reference	39 (4.5)	Reference
Yes	237	12 (5.1)	0.73 (0.35–1.41)	11 (4.6)	1.18 (0.53–2.46)
Abdominal obesity					
No	701	38 (5.4)	Reference	28 (4.0)	Reference
Yes	405	28 (6.9)	0.94 (0.51–1.70)	22 (5.4)	1.36 (0.65–2.85)
Diabetes mellitus					
No	986	58 (5.9)	Reference	37 (3.8)	Reference

(*Continued*)

Table 3. Continued

		LVH		CKD	
Yes	120	8 (6.7)	0.83 (0.34–1.76)	13 (10.8)	1.50 (0.71–3.03)
Hypertension					
No	493	9 (1.8)	Reference	5 (1.0)	Reference
Yes	613	57 (9.3)	5.28 (2.55–12.11)	45 (7.3)	3.38 (1.36–10.30)
High TG					
No	1003	64 (6.4)	Reference	42 (4.2)	Reference
Yes	103	2 (1.9)	0.22 (0.04–0.75)	8 (7.8)	1.67 (0.67–3.75)
High TC					
No	865	48 (5.5)	Reference	25 (2.9)	Reference
Yes	241	18 (7.5)	1.21 (0.64–2.22)	25 (10.4)	3.36 (1.72–6.66)
High LDL-C					
No	892	53 (5.9)	Reference	29 (3.3)	Reference
Yes	214	13 (6.1)	0.92 (0.46–1.74)	21 (9.8)	3.17 (1.61–6.23)
Low HDL-C					
No	945	57 (6.0)	Reference	41 (4.3)	Reference
Yes	161	9 (5.6)	0.66 (0.29–1.38)	9 (5.6)	1.18 (0.48–2.68)
Dyslipidemia					
No	655	42 (6.4)	Reference	17 (2.6)	Reference
Yes	451	24 (5.3)	0.62 (0.35–1.05)	33 (7.3)	2.34 (1.24–4.54)
High Lp(a)					
No	855	53 (6.2)	Reference	38 (4.4)	Reference
Yes	251	13 (5.2)	0.74 (0.37–1.38)	12 (4.8)	0.80 (0.38–1.58)
High hs-CRP					
No	785	46 (5.9)	Reference	25 (3.2)	Reference
Yes	321	20 (6.2)	0.91 (0.50–1.61)	25 (7.8)	1.84 (0.98–3.47)
Hyperuricemia					
No	958	59 (6.2)	Reference	26 (2.7)	Reference
Yes	148	7 (4.7)	0.60 (0.24–1.31)	24 (16.2)	5.49 (2.84–10.65)

Logistic regression model was used for statistical analysis. For overall obesity and abdominal obesity, age, sex, ethnicity, living area, education, smoking, and physical activity were adjusted in the model. For the other categorized risk factors, body mass index was additionally added into the model. CKD indicates chronic kidney disease; GHS, Ghana Heart Study; HDL-C, high-density lipoprotein cholesterol; hs-CRP, high-sensitivity C-reactive protein; LDL-C, low-density lipoprotein cholesterol; Lp(a), lipoprotein (a); LVH, left ventricular hypertrophy; OR, odds ratio; PAD, peripheral artery disease; TC, total cholesterol; TG, total triglyceride; and TOD, target organ damage.

and physical activity (Table 3). Hypertension had the strongest association with carotid intimal thickening (OR, 1.92; 95% CI, 1.22–3.08) and LVH (OR, 5.28; 95% CI, 2.55–12.11) after adjustment for potential confounding factors (Table 3). Hyperuricemia had the strongest association with CKD (OR, 5.49; 95% CI, 2.84–10.65) after adjustment for potential confounding factors (Table 3).

In Figures 1 through 4, we used restricted cubic spline to visualize the shape of the relation between the risk factors with strong association with TOD and TOD measurements. The directions of these associations were consistent with results of logistic regression analysis. DBP showed a J-shaped association with LVM index (*P* for nonlinearity <0.0001) after adjustment for age, sex, ethnicity, region, living community, education, smoking, and physical activity. The inflection points of DBP were 73 mm Hg for men and 63 mm Hg for women. The association of DBP with LVM index was relatively flat below the inflection point and then started to increase rapidly afterwards. Stratification analysis by sex showed no significant sex difference for these associations.

The sensitivity analysis, excluding missing data, showed that the above-mentioned associations were robust (Figure S1).

DISCUSSION

In this national study of adults in Ghana, we characterized the distribution of cardiometabolic risk factors and preclinical TOD among adults. Ghana is located in western SSA; our findings indicate that cardiometabolic

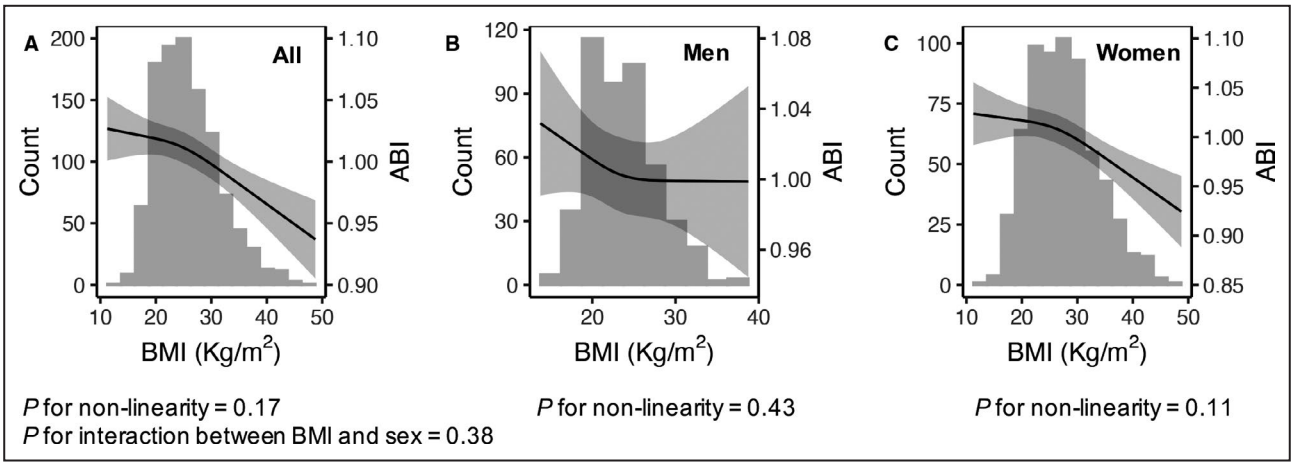

Figure 1. Association between body mass index (BMI) and ankle-brachial index (ABI) among adults who participated in GHS (Ghana Heart Study), 2016 to 2017.
A, Association of BMI with ABI in all population. **B**, Association of BMI with ABI in men. **C**, Association of BMI with ABI in women. Restricted cubic splines with 3 knots were used for association analysis with adjustment for age, sex, ethnicity, living area, education, smoking, and physical activity. Histogram represents the distribution of BMI in the study population.

risk factors previously identified in populations of the developed countries are now common among adults in Ghana SSA. In particular, obesity, hypertension, and hyperuricemia appear to be the risk factors having the strongest association with specific preclinical TOD.

With rapid rural-to-urban transitions in SSA, the cardiometabolic risk factors, such as obesity, hypertension, and diabetes mellitus, identified in the developed countries have not been rare in SSA.[6,10,11] Consistent with these previous reports from relatively small sample size, our study also indicated that the obesity rate in Ghanaian adults was as high as 15%, and that there were clear differences between women (23%) and men (5%). The age-standardized prevalence of hypertension in our study is ≈26%, based on Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, or ≈41%, based on 2017 American Heart Association guideline. Our study shows that type 2 diabetes mellitus prevalence is now 7% among adults in Ghana, compared with studies conducted from 1960s to 1980s showing type 2 diabetes mellitus prevalence of <1% in many regions of SSA.[11] In addition, dyslipidemia appears to be a most common risk factor in Ghanaian adults, consistent with findings from a recent meta-analysis in Africa.[12]

In comparison, according to the latest national estimates from National Health and Nutrition Examination Survey 2011 to 2014,[13] the prevalence of obesity was 58% among Black women, 38% in Black men, 34% in White men, and 33% in White women.[13] The prevalence of hypertension among Black individuals was 42.4% for adult men and 44% for adult women in 2016.[14] The prevalence of type 2 diabetes mellitus was 21.8% in Black individuals and 11.3% in non-Hispanic White individuals in National Health and Nutrition Examination Survey 2011 to 2012.[15] The prevalence of high TC was 32.6% in Black men, 37.0% in White men, 36.1% in Black women, and 43.4% in White women. Thus, the prevalence of obesity, diabetes mellitus, hypertension, and dyslipidemia in the population in Ghana is still lower than that in Black individuals. The prevalence of cardiometabolic risk factors and preclinical TOD reported herein was adjusted age standardized using the national population distribution. However, Ghana and the United States have different age distribution within the population, with a younger population in Ghana,[16,17] which may partly explain the observed lower prevalence in Ghana than in the United States.

Uric acid is the end product of purine metabolism in human body. Although the causal relationship between uric acid and CVD remains unclear, many epidemiological studies suggest the existence of a significant association between high uric acid and increased risk of CVD[18] as well as CKD.[19] In our study, hyperuricemia was defined according to the criteria adopted by the National Health and Nutrition Examination Survey. Our data indicate that 9% of adults are hyperuricemic in Ghana. As a comparison, hyperuricemia prevalence rates were 20% among adults in the United States in 2015 to 2016.[20]

Inflammation has been implicated in CVD pathogenesis, and plasma hs-CRP has been implicated as a marker of inflammation linked to CVD prospectively in several large cohorts of men and women in the United States,[21,22] although the optimal clinical utility of plasma hs-CRP remains controversial.[23] Previous studies in African populations have investigated the relationship between inflammation and infectious

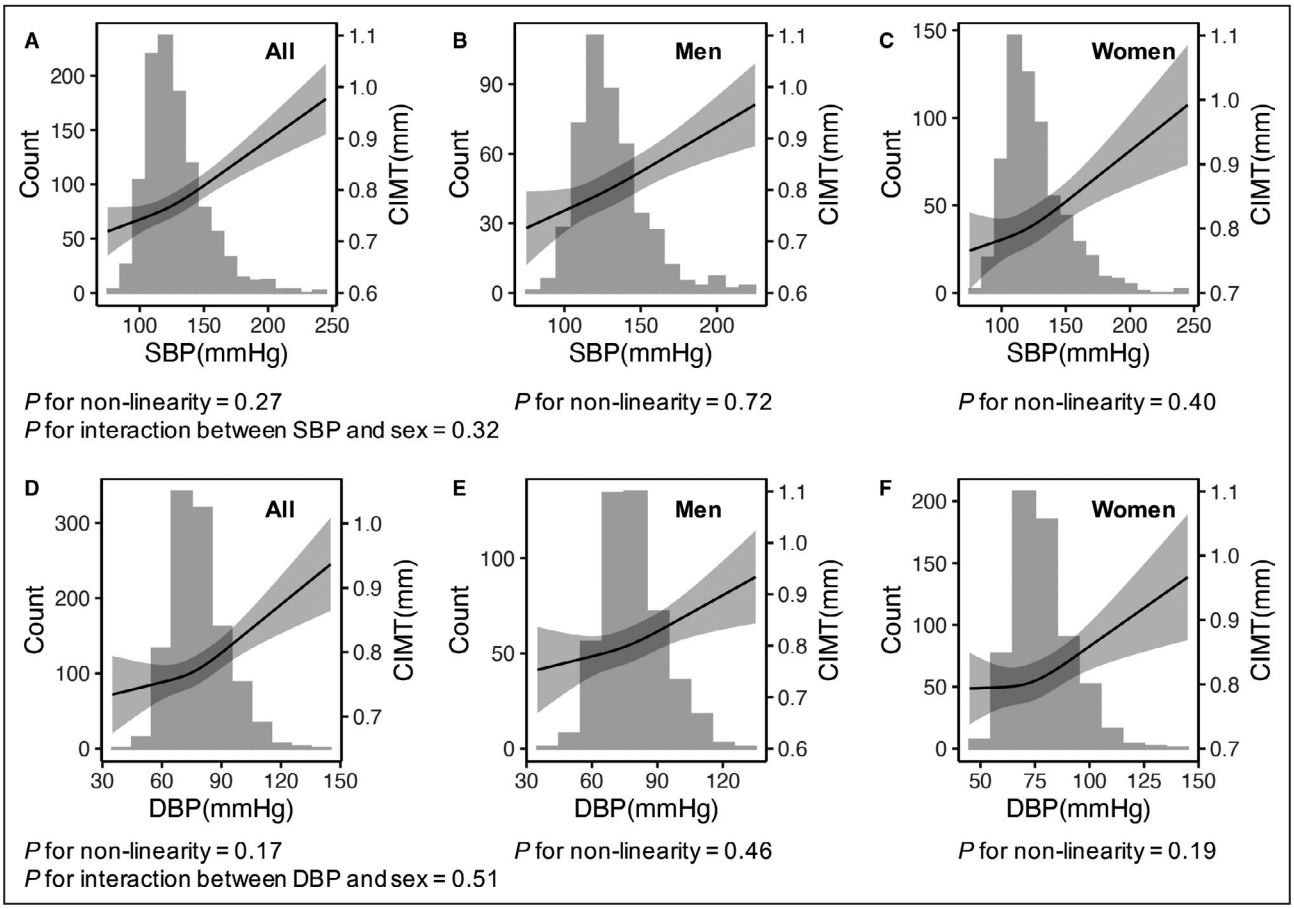

Figure 2. Association of systolic blood pressure (SBP) and diastolic blood pressure (DBP) with carotid intima-media thickness (CIMT) among adults who participated in GHS (Ghana Heart Study), 2016 to 2017.
A, Association of SBP with CIMT in all population. **B**, Association of SBP with CIMT in men. **C**, Association of SBP with CIMT in women. **D**, Association of DBP with CIMT in all population. **E**, Association of DBP with CIMT in men. **F**, Association of DBP with CIMT in women. Restricted cubic splines with 3 knots were used for association analysis with adjustment for age, sex, ethnicity, living area, education, smoking, physical activity, and body mass index. Histogram represents the distribution of SBP/DBP in the study population.

diseases[24]; however, the association between hs-CRP and CVD has not been well studied in SSA. Our study finds that the prevalence of high hs-CRP in Ghanaian adults is lower than that in Burkina Faso,[25] a geographical neighbor.

We also found clear sex and ethnicity disparities in CVD risk factors in Ghana. Women had higher rates of obesity, high TC, high LDL-C, and high hs-CRP than men, which also was reported in another study conducted in Ghana.[6] Akan had significant higher rates of diabetes mellitus, hypertension, high TC, high total triglyceride, and high LDL-C than other ethnicities in this study. The ethnicity disparities in hypertension and dyslipidemia were also reported in other Ghana studies.[26,27] These observed sex and ethnicity disparities may be partly explained by pathophysiological and sociocultural factors and their interactions. In our study, women had lower level of education and physical activity than men; more Akan drunk alcohol (62% versus 31%) and were physically inactive (91% versus 74%) than other ethnicities. Further investigations are needed to clarify the reasons causing such disparities and their effects on CVD development in Ghana. Despite the sex disparities in prevalence of CVD risk factors, we did not find significant sex difference for the association of CVD risk factors with TOD.

We further investigated the roles of CVD risk factors in the development of preclinical conditions of vascular, cardiac, and renal TOD. PAD and carotid thickening were chosen as the proxy measurements for vascular TOD. A recent systematic review shows that the prevalence of PAD in low- and middle-income countries is 3% at the age of 45 to 49 years and 15% at the age of 85 to 89 years, which is lower than in high-income countries, with smoking, diabetes mellitus, hypertension, and high TC being identified as the risk factors for PAD.[28] Our study showed that the age-standardized prevalence of PAD was ≈10% in the Ghanaian adult population. We identified obesity as the most important risk factor for PAD in Ghana. Also, we found that

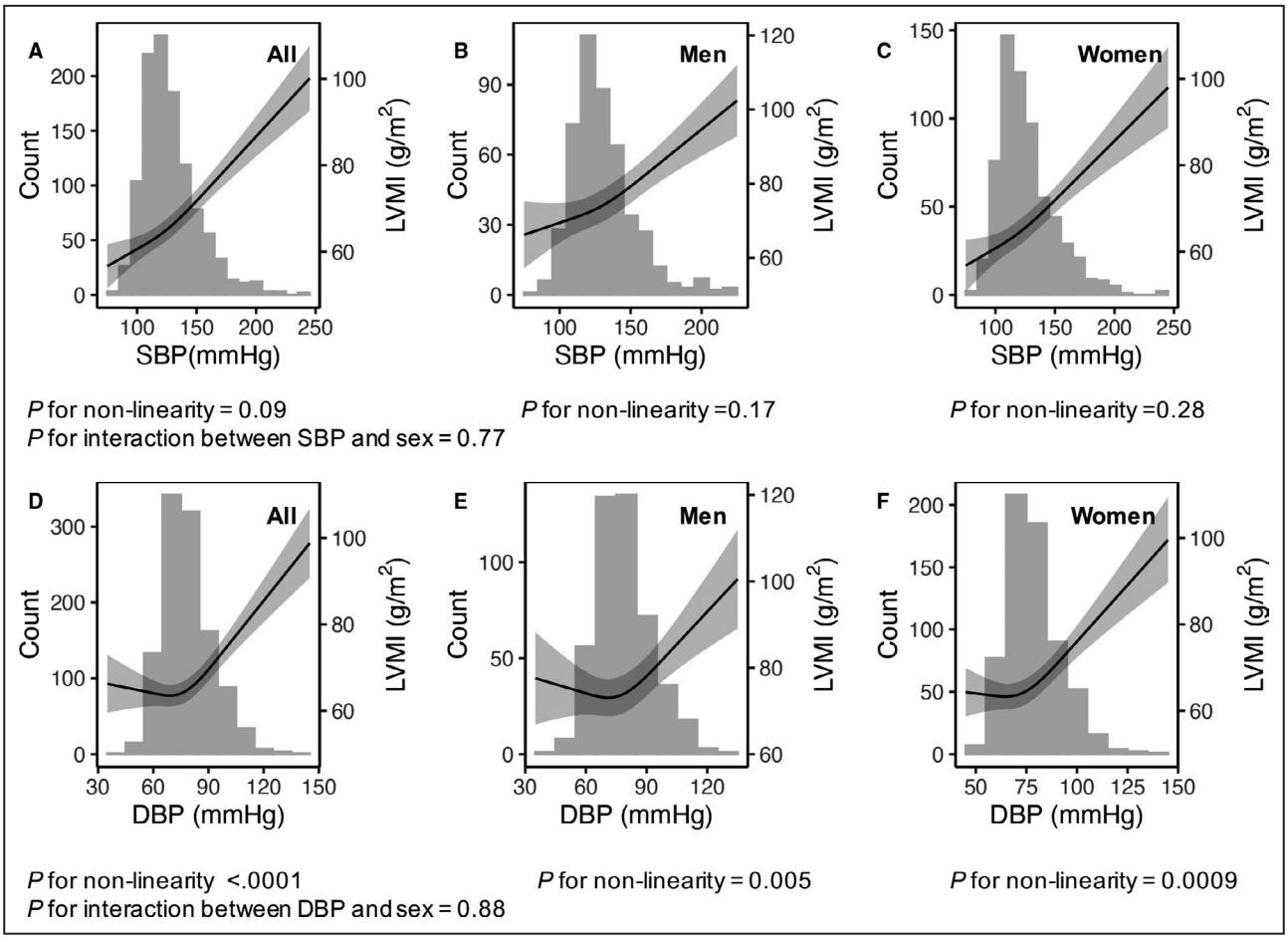

Figure 3. Association of systolic blood pressure (SBP) and diastolic blood pressure (DBP) with left ventricular mass index (LVMI) among adults who participated in GHS (Ghana Heart Study), 2016 to 2017.
A, Association of SBP with LVMI in all population. **B**, Association of SBP with LVMI in men. **C**, Association of SBP with LVMI in women. **D**, Association of DBP with LVMI in all population. **E**, Association of DBP with LVMI in men. **F**, Association of DBP with LVMI in women. Restricted cubic splines with 3 knots were used for association analysis with adjustment for age, sex, ethnicity, living area, education, smoking, physical activity, and body mass index. Histogram represents the distribution of SBP/DBP in the study population.

the age-standardized prevalence of carotid thickening was ≈8% in this population, and hypertension was considered to be the key risk factor, consistent with the previous report.[29]

It has been reported that Black people are more likely to develop LVH than White people in the United States.[30] The studies on Black populations living in Africa show that the overall prevalence of LVH, ascertained by echocardiogram, was 41% in Gambia,[31] 62% in Cameroon,[32] and 41% in Angola.[33] Our study indicated that the age-standardized prevalence of LVH in Ghana (≈4%) is significantly lower than that in other African countries. The hypertension prevalence reported previously in studies conducted in Gambia, Cameroon, and Angola was higher than that in our study, although these 3 studies had small sample size and selected participants from one single site. All of these differences in characteristics of participants in our study and others may lead to the different observed prevalence of LVH. Further studies with comprehensive comparison of sociodemographic and cultural factors in Ghana versus the other African countries are warranted. Consistent with the other studies,[34] we found that hypertension has the strongest association with LVH in Ghanaian population. Moreover, our spline regression analysis found a J-shaped association between DBP and LVM index, which needs to be researched in depth in the future studies.

Although the age-standardized prevalence of CKD was low in our population, hyperuricemia, high TC, and high LDL-C were significantly associated with CKD. The prevalence of CKD was 13% in the United States, according to the National Health and Nutrition Examination Survey 1999 to 2004 data,[35] and 11% in China in 2012, shown in a national survey.[36] The China national survey reported significant association of hypertension, diabetes mellitus, and hyperuricemia with CKD. Although hyperuricemia has a strong association with the risk of

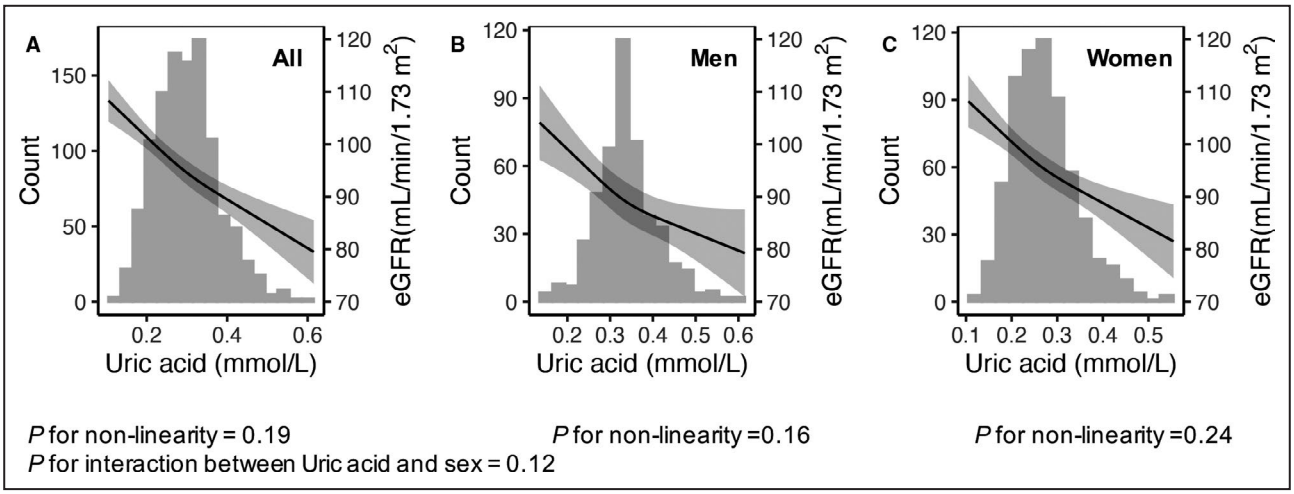

Figure 4. Association of uric acid with estimated glomerular filtration rate (eGFR) among adults who participated in GHS (Ghana Heart Study), 2016 to 2017.
A, Association of uric acid with eGFR in all population. **B**, Association of uric acid with eGFR in men. **C**, Association of uric acid with eGFR in women. Restricted cubic splines with 3 knots were used for association analysis with adjustment for age, sex, ethnicity, living area, education, smoking, physical activity, and body mass index. Histogram represents the distribution of plasma uric acid levels in the study population.

CKD (OR, 5.5 in our study; OR, 9.3 in the China survey), a recent mendelian randomization study concluded no causal effects of serum urate level on the risk of CKD.[37] Thus, the strong association of hyperuricemia with risk of CKD observed in multiple populations needs to be further investigated in future work.

Some limitations should be kept in mind when interpreting our findings. First, this study is a cross-sectional in design, precluding from direct causal inference of risk factors and outcome relations. Nevertheless, all CVD risk factors identified in the current study are consistent with those reported in large prospective cohorts of men and women in the developed countries. Second, the clinical outcomes were not ascertained by a repeated physical examination, which may result in measurement errors and potential underestimation of prevalence estimates of CVD risk factors. Similarly, CKD was assessed only by a single serum creatinine measurement in our study; we did not assess for proteinuria, which also indicates the presence of CKD, which may lead to underestimated prevalence of CKD.

In summary, in this first national survey of cardiometabolic health status among adults in Ghana, known CVD risk factors were highly prevalent. Specifically, obesity, hypertension, and hyperuricemia were important CVD risk factors associated with preclinical TODs among adults in Ghana, which should be the focus for interventions to improve both individual-cardiometabolic and population-cardiometabolic health in Ghana.

ARTICLE INFORMATION

Received May 8, 2020; accepted November 2, 2020.

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Acknowledgments

The authors would like to express their sincere gratitude to all the participants who were involved in this study, and the research assistants for the support they provided. Without their cooperation, this study would not have been successful.

Sources of Funding

This study was supported by Guangdong Academy of Medical Sciences and the China-Ghana West African Heart Center Cooperation Project (Dr Lin), People's Republic of China. Dr Liu was partially funded by the American Heart Association, the US Fulbright Scholar Program, and the National Institutes of Health.

Disclosure

In the design and conduct of the Ghana Heart Study, Dr. Liu served as Chief Scientific Consultant for Guangdong Provincial People's Hospital, Guangdong Academy of Medical Sciences.

Supplementary Material

Data S1
Table S1
Figure S1
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SUPPLEMENTAL MATERIAL

Data S1.

Supplemental Methods

Cardiometabolic risk factors definition

The cardiometabolic risk factors measured in this study include adiposity, blood pressure, diabetes mellitus, lipids, uric acid, and inflammation. Overall obesity was defined as BMI ≥ 30 kg/m^2. Abdominal obesity was defined as waist circumference > 88 cm for women and 102 cm for men. Diabetes mellitus was defined as fasting blood glucose level of 7.0 mmol/L or greater, HbA1c ≥ 6.5%, and/or use of insulin or an oral hypoglycaemic agent[38]. Hypertension was defined as systolic blood pressure (SBP) ≥ 130 mmHg and/or diastolic blood pressure (DBP) ≥ 80 mmHg or use of anti-hypertensive drugs according to the 2017 American College of Cardiology/American Heart Association Guideline for High Blood Pressure in Adults[39]. To facilitate comparison with previous studies, we also defined hypertension using the cutoff of 140 mmHg for SBP and/or 90 mmHg for DBP [40]. Dyslipidemia was defined as TG ≥ 1.70 mmol/L (150 mg/dL), TC ≥ 6.22 mmol/L (240 mg/dL), LDL ≥ 4.14 mmol/L (160 mg/dL), HDL <1.04 mmol/L (40 mg/dL), use of lipid-lowering medications[41], and/or Lipoprotein (a) [Lp(a)] ≥ 60 mg/dL[42]. A hsCRP > 3 mg/L[43] was considered as abnormal high. Hyperuricemia was defined as uric acid > 0.42 mmol/L (7.0 mg/dl) in men and > 0.34 mmol/L (5.7 mg/dl) in women, per the Third National Health and Nutrition Examination Survey laboratory definition[44].

Preclinical TOD definition

Each participant underwent physical examinations to evaluate for preclinical vascular (PAD and carotid), cardiac, and renal TOD. For vascular TOD, ankle brachial pressure index (ABI) was measured for each participant using the Boso (Bosch & Sohn, Germany) ABI device. Peripheral artery disease (PAD) was determined as either a left or right ABI < 0.9 [45]. Carotid ultrasound was performed for both carotid arteries using the GE VIVID Q ultrasound system equipped with a 9L-RS 3.1-10.0 MHz linear probe to determine carotid intima media thickness (CIMT). Carotid intimal thickening was defined as CIMT ≥ 0.9 mm [46].

For cardiac TOD, Left ventricular hypertrophy (LVH) was defined as left ventricular mass index (LVMI) > 95 g/m^2 for women and > 115 g/m^2 for men according to the American Society of Echocardiography (ASE) recommendation [47]. LVMI was calculated by dividing Left ventricular mass (LVmass) by body surface area (BSA). Left ventricular mass (LVmass) was calculated using the following formula:

LVmass = 0.8x(1.04x([LVIDD+PWTD+IVSTD]3-[LVIDD]3))+0.6 g.

Where: LVIDD = Left Ventricular Internal Diameter in Diastole

PWTD = Posterior Wall Thickness in Diastole

IVSTD = Interventricular Septum Thickness in Diastole

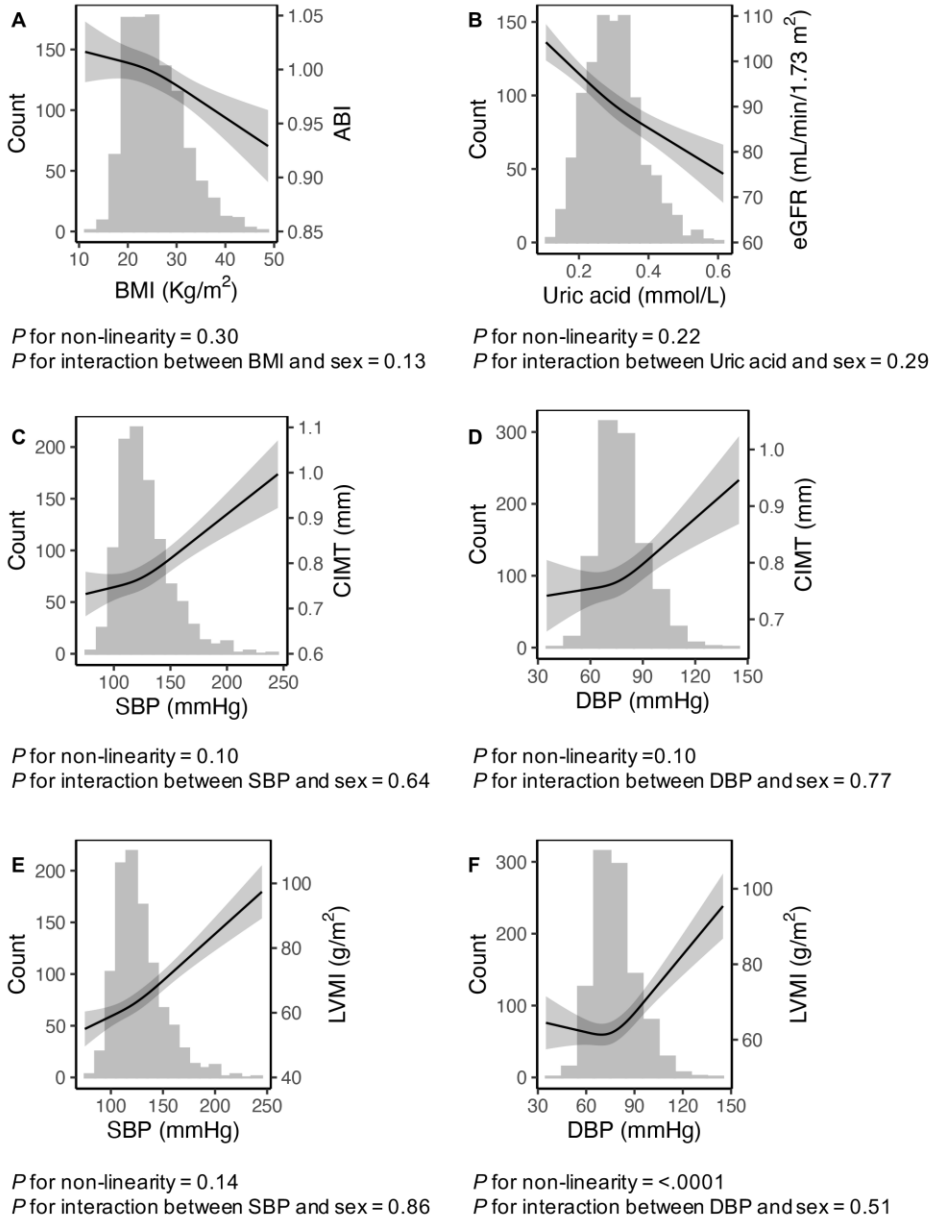
Renal TOD was evaluated using the estimated glomerular filtration rate (eGFR), an important chronic kidney disease (CKD) marker. The CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) equation was used to evaluate the eGFR[48]: eGFR = 141 × min (SCr/κ, 1) $^{\alpha}$ × max (SCr/κ, 1) $^{-1.209}$ × 0.993 ^{Age} × 1.018 [in women], where SCr is serum creatinine, κ is 0.7 for women and 0.9 for men, α is −0.329 for women and −0.411 for men, min indicates the minimum of SCr/κ

or 1, and max indicates the maximum of SCr/κ or 1. CKD was defined as eGFR of less than 60 mL/min/1.73 m^2.

Table S1. Definitions of cardiometabolic risk factors and preclinical target organ damage.

	Definitions
Overall obesity	BMI ≥ 30 kg/m^2
Abdominal obesity	Women: waist circumference > 88 cm
	Men: waist circumference > 102 cm
Diabetes mellitus	Fasting blood glucose≥7.0 mmol/L
	HbA1c ≥ 6.5%
	and/or use of insulin or an oral hypoglycaemic agent
Hypertension	SBP ≥ 140 mmHg
	and/or DBP ≥ 80 mmHg
	or use of anti-hypertensive drugs
Dyslipidemia	TG ≥ 1.70 mmol/L (150 mg/dL)
	TC ≥ 6.22 mmol/L (240 mg/dL)
	LDL ≥ 4.14 mmol/L (160 mg/dL)
	HDL <1.04 mmol/L (40 mg/dL)
	use of lipid-lowering medications
	and/or Lp(a)≥ 60 mg/dL
Abnormal high level of hsCRP	> 3 mg/L
Hyperuricemia	Women: uric acid > 0.34 mmol/L (5.7 mg/dl)
	Men: uric acid > 0.42 mmol/L (7.0 mg/dl)
Peripheral artery disease (PAD)	either a left or right ABI < 0.9
Carotid intimal thickening	Carotid intima media thickness (CIMT) ≥ 0.9 mm
Left ventricular hypertrophy (LVH)	Women: left ventricular mass index (LVMI) > 95 g/m^2
	Men: ventricular mass index (LVMI) >115 g/m^2
Chronic kidney disease (CKD)	eGFR<60 mL/min/1.73 m$^{2.}$

Figure S1. Sensitivity analysis excluding missing data for the association of cardiometabolic risk factors with preclinical target organ diseases measurements among all adults participated in Ghana heart study, 2016-2017.



Restricted cubic splines with three knots were used for association analysis with adjustment for age, sex, ethnicity, living area, education, smoking, physical activity, and BMI. Histograms represent the distribution of SBP/DBP in the study population. ABI, ankle-brachial index; CIMT, carotid intima–media thickness; LVMI, left ventricular mass index; eGFR, estimated glomerular filtration rate; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure.