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Burden of cardiometabolic risk factors and preclinical target organ damage among adults in Freetown, Sierra Leone: a community-based health-screening survey

James Baligeh Walter Russell [1], Theresa Ruba Koroma [1], Santigie Sesay [2], Sallieu Kabay Samura,[3] Sulaiman Lakoh [1], Ansumana Bockarie [4], Onome Thomas Abir,[5] Joseph Sam Kanu [6], Joshua Coker,[1] Abdul Jalloh [1], Victor Conteh [1], Sorie Conteh [1], Mohamed Smith [1], Othman Z Mahdi,[7] Durodami R Lisk [1]

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For numbered affiliations see end of article.

Correspondence to
Dr James Baligeh Walter Russell;
jamesbwrussell@gmail.com

ABSTRACT

Objective To investigate the prevalence of cardiometabolic risk factors (CMRFs), target organ damage (TOD) and its associated factors among adults in Freetown, Sierra Leone.

Design This community-based cross-sectional study used a stratified multistage random sampling method to recruit adult participants.

Setting The health screening study was conducted between October 2019 and October 2021 in Western Area Urban, Sierra Leone.

Participants A total of 2394 adult Sierra Leoneans aged 20 years or older were enrolled.

Outcome measure Anthropometric data, fasting lipid profiles, fasting plasma glucose, TOD, clinical profiles and demographic characteristics of participants were described. The cardiometabolic risks were further related to TOD.

Results The prevalence of known CMRFs was 35.3% for hypertension, 8.3% for diabetes mellitus, 21.1% for dyslipidaemia, 10.0% for obesity, 13.4% for smoking and 37.9% for alcohol. Additionally, 16.1% had left ventricular hypertrophy (LVH) by ECG, 14.2% had LVH by two-dimensional echo and 11.4% had chronic kidney disease (CKD). The odds of developing ECG-LVH were higher with diabetes (OR=1.255, 95% CI (0.822 to 1.916) and dyslipidaemia (OR=1.449, 95% CI (0.834 to 2.518). Associated factors for higher odds of Left Ventricular Mass Index by echo were dyslipidaemia (OR=1.844, 95% CI (1.006 to 3.380)) and diabetes mellitus (OR=1.176, 95% CI (0.759 to 1.823)). The odds of having CKD were associated with diabetes mellitus (OR=1.212, 95% CI (0.741 to 1.983)) and hypertension (OR=1.163, 95% CI (0.887 to 1.525)). A low optimal cut-off point for ECG-LVH (male 24.5 mm vs female 27.5 mm) was required to maximise sensitivity and specificity by a receiver operating characteristics curve since the odds for LVH by ECG were low.

Conclusions This study provides novel data-driven information on the burden of CMRF and its association with preclinical TOD in a resource-limited setting. It illustrates the need for interventions in improving cardiometabolic health screening and management in Sierra Leonean.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ A major strength of this study is its community-based design and the first study of its kind on a larger population in Sierra Leone.
⇒ The study was statistically powered to produce results that are representative of adults in Sierra Leone.
⇒ The study is limited as it could not infer direct causality between risk factors and effect outcomes.
⇒ Since some of the outcomes (fasting plasma glucose, HbA1c and fasting lipid profile) are limited by the reliance on single time point measurements, it may result in measurement errors and the potential of underestimating cardiometabolic risk factors.
⇒ Chronic kidney disease (CKD) assessment by single serum creatinine without assessing for proteinuria, which also indicates the presence of CKD, will lead to an underestimation of CKD.

INTRODUCTION

Cardiometabolic diseases (CMD) are a group of complex disorders, including cardiovascular diseases and diabetes mellitus. The spectrum of CMD begins with insulin resistance, a trait that is expressed early in life and later will progress to clinically identifiable high-risk states of pre-diabetes, then to type 2 diabetes mellitus and cardiovascular diseases (CVD).[1]

CVD is of great interest because its insidious progression is marked by a multistage

pathogenesis that is often heralded by asymptomatic changes in the heart, kidney and blood vessels.[1 2] The associated risk factors of CMD are a cluster of obesity (particularly central adiposity), dyslipidaemia, psychosocial stress, unhealthy lifestyles like physical inactivity, lack of consumption of fruits/vegetables, cigarette smoking and harmful alcohol consumption.[2 3] These risk factors are associated with dysfunctional biomedical processes within the body, with the potential of triggering cardiovascular diseases (CVD) and their related complications of chronic non-communicable diseases (NCDs).[4–6]

According to WHO, NCDs are the leading causes of morbidity and mortality, with more than three-quarters of NCD deaths occurring in low-income and-middle-income countries.[3] In 2017, the Global Burden of Disease Study reported a dramatic increase in the total number of deaths in NCD by 22.7% (21.5%–23.9%) from 2007 to 2017, while the disability-adjusted life-years related to CVDs were 73.3%. During the same period (2007–2017), there was an estimated increase of 7.61 million deaths, with the highest rate in western sub-Saharan Africa (SSA).[7] This epidemiological transition from communicable to NCDs in SSA could account for the exponential increase in cardiometabolic risk factors (CMRFs).[8] The recent demographic change witnessed in urban settings of many LMICs may be attributed to adopting western lifestyle behaviours, including poor eating habits, harmful alcohol consumption and cigarette smoking.[9–11] These settings will also be a favourable platform for developing CMRFs and their attending target organ damage (TOD). While there is recognition of the rising burden of NCDs across Africa, scanty information exists in most SSA countries because of the absence of well-developed health programmes for the comprehensive evaluation and management of high-risk individuals.[12 13] Our understanding of this spectrum of diseases is disproportionately informed by studies conducted in developed countries. Such findings may not be entirely applicable to individuals in developing countries. Reasons for this could be related to differences in genetic characteristics and CVD risk factors across countries and regions.[14]

Sierra Leone is one of the least developed countries in the world, with a double burden of communicable and NCDs. The 11 years of devastating civil war (1991–2002) disrupted the health system, and its long-term effects were still seen during the public health crisis caused by the 2013–2016 Ebola outbreak.[15 16] Since the civil war, Sierra Leone has experienced significant urbanisation in recent years, and this demographic evolution has impacted the socioeconomic growth recovery of the nation. This type of chaotic urbanisation, also referred to as a 'complex urban health crisis', is seen in other SSA countries because it serves as a harbinger that is accelerating NCD burden and is an existential threat to the health and development of a nation.[17–19]

In Sierra Leone, the evaluation of CVDs has been conducted by several small studies but with very little information on the assessment of the CMRF burden.[20–23] Although a recent survey in a provincial district setting (rural and urban) suggested a high prevalence of CMRF, there are limited data estimating preclinical TOD in this West African country.[24] In addition, there is no report of a direct evaluation of CMRF in any settlement in the capital city of Sierra Leone. This study aimed to comprehensively evaluate the prevalence of cardiovascular risk factors and TOD in a population-based study in Freetown, Sierra Leone. The study also investigated how these known CVDRFs are associated with preclinical cardiac and renal TOD among adults aged 20 years or more.

METHODS

Patient and public involvement

Patients and the public were not involved in the planning, designing, conducting, reporting or information dissemination.

Study setting and design

This population-based cross-sectional study was a health screening survey conducted between October 2019 and October 2021 among adults living in Western Area Urban, Freetown, Sierra Leone. It was a screening and awareness programme for NCDs in Western Area Urban, initiated and funded by Ecobank Sierra Leone Limited. Freetown is the capital city of Sierra Leone, with an estimated 1.5 million inhabitants.[25] Freetown is important because of its densely heterogeneous population and the main business centre of Sierra Leone. It sets the trend for the rest of the country as its demographic distribution is similar to other larger cities. All ethnic groups in the country can be found in Freetown, with Krio and English being the primary spoken languages.

Sample size calculation, participant recruitment and selection

The study was designed to provide results that truly represent the adult population in Sierra Leone. A month before the awareness and screening campaign for NCDs, citizens within the Freetown municipality were informed about these activities by repeated mass communication through national radio and television stations. We used a stratified random sampling strategy to recruit adult Sierra Leonean participants aged ≥20. Western Area Urban—Freetown is divided into eight official electoral constituencies (Central I, II, East I, II, III and West I, II, III), and the first stage in the sampling strategy was to select all eight constituencies. This was followed by subdividing each constituent region into subzones using the 2015 census data,[25] and subsequently, one of the subzonal communities was selected by simple random sampling. The selected communities were namely: Calabar Town—East III, Low-Cost Housing Community—East II, Ginger Hall Community—East I, Mountain Cut Community—Central II, PWD/Pademba Road community—Central I, Brookfield's Community—West II and Aberdeen Community—West III. Potential participants within each selected subzonal community were line-listed at their

community health centre, and simple random sampling methods were used to select these enlisted individuals. The following participants were excluded: Pregnant and lactating mothers, those with mental illness/dementia and persons unwilling to grant consent.

The sample size was calculated using the clinical estimated prevalence of 22% for hypertension in Sierra Leone.[26] The minimum sample size was assessed using the Leslie Kish formula[27]:

$$n = \frac{Z^2 \times p(1-p)}{d^2}$$

where n is the sample size (number of adult participants), p is the expected prevalence of hypertension in an adult population ($p = 0.22$) and d is the precision (if 5%, d=0.05). The Z value is 1.96 for a 95% CI.

$$n = 263$$

To minimise bias and allow attrition of non-response and non-availability of data, the sample size was oversampled by 20%.

$$n = 263 + 52.7 = 316.42$$

Using a design effect of eight subzonal communities to adjust the sample size:

$$n = 316.42 \times 8.0 = 2531.36 \approx 2531$$

Procedure and data collection

All eligible potential participants in each selected community were invited to participate in the 'awareness and screening campaign for NCD' on a designated date at the National Victoria Park. The WHO stepwise approach guided the process of data collection for this study. Medical students, doctors and nurses were trained on the campaign's conduct, including data collection. (Flow chart during the campaign is shown in figure 1).

Demographic and health history

A standard questionnaire was used to obtain information on demographics (age, sex and education), lifestyle (fruit and vegetable consumption, smoking status and physical activity) and medical history (family history of hypertension and diabetes mellitus). Translators were used for participants who could not understand English. An OMRON M3 electronic sphygmomanometer with an appropriate cuff size was used to record a participant's blood pressure in the sitting position, and measurements were taken after at least 3–5 min of rest. The mean of the two recorded readings was taken as the participant's blood pressure. Body weight, height and waist circumference (WC) were measured with light clothes and bare feet.

Outcome measures and definition

i. Hypertension was defined as an average systolic blood pressure (SBP) of 140 mm Hg or higher, or diastolic blood pressure (DBP) of 90 mm Hg or greater or a participant-reported current use of antihypertensive medication.[28]
ii. A participant who smoked more than 100 sticks of cigarettes in their lifetime and still smoking at the interview was referred to a smoker, while an ex-smoker was someone who had stopped smoking at least 28 days before the interview.[29]
iii. Data on alcohol consumption were based on the WHO step survey tool.[30]
iv. Physical activity was classified into 'low', 'moderate' and 'vigorous'.
   A. Low physical activity: sedentary lifestyles at work and home.
   B. Moderate physical activity: brisk walking, domestic house chores and general house task such as roofing and painting, moderate farm work like weeding.
v. Vigorous physical activity: running, briskly ascending and descending hill tasks, intense farm working and carrying masses >20 kg.[31]
vi. The body mass index (BMI) was calculated as a ratio of the weight in kilograms and the square of the height in metres. BMI-based body habitus (in kg/m^2) was classified as underweight (BMI<18.5), normal weight (BMI=18.5–24.9), overweight (BMI=25.0–29.9) and obese (BMI≥30).[32]

CMRFs definition

The CMRFs measured in this study include blood pressure, fasting blood sugar, HbA1c (Glycated Haemoglobin), WC, BMI and serum lipids.

vi. Overall obesity was defined as BMI≥30 kg/m^2.
vii. Abdominal obesity was defined as WC >88 cm for women and 102 cm for men.[33]
viii. Diabetes mellitus was defined as a fasting blood glucose (FBG) level of 7.0 mmol/L or greater, HbA1c≥6.5%, or the use of insulin or an oral hypoglycaemic agent. Pre-diabetes was defined as FPG between 6.1 mmol/L (110 mg/ dL) and 6.9 mmol/L (124.9 mg/dL).[34]

At the health screening venue (Victoria Park), consented and enrolled participants who had completed their screening questionnaires were referred for cardiac evaluation, ECG, echocardiographic and to an accredited reference laboratory for blood sample collection.

Clinical biochemistry measurements

Participants' blood samples were collected from the median cubital vein between 8:00 and 10:00 hours, after overnight fasting for 8–10 hours. These samples were processed within 4 hours of collection per manufacturers' instructional protocols, using Beckman Coulter: AU480 Chemistry System. Glucose, total cholesterol (TC), triglycerides, high-density lipoprotein (HDL-C) and low-density lipoprotein (LDL-C) were analysed. America Diabetes Association cut-points were used to evaluate lipid panel markers and DM abnormalities. Dyslipidaemia was defined as TG≥1.70 mmol/L (150 mg/dL), TC≥6.22 mmol/L (240 mg/dL), LDL≥3.3 mmol/L (130 mg/dL), HDL<1.04 mmol/L (40 mg/dL), use of

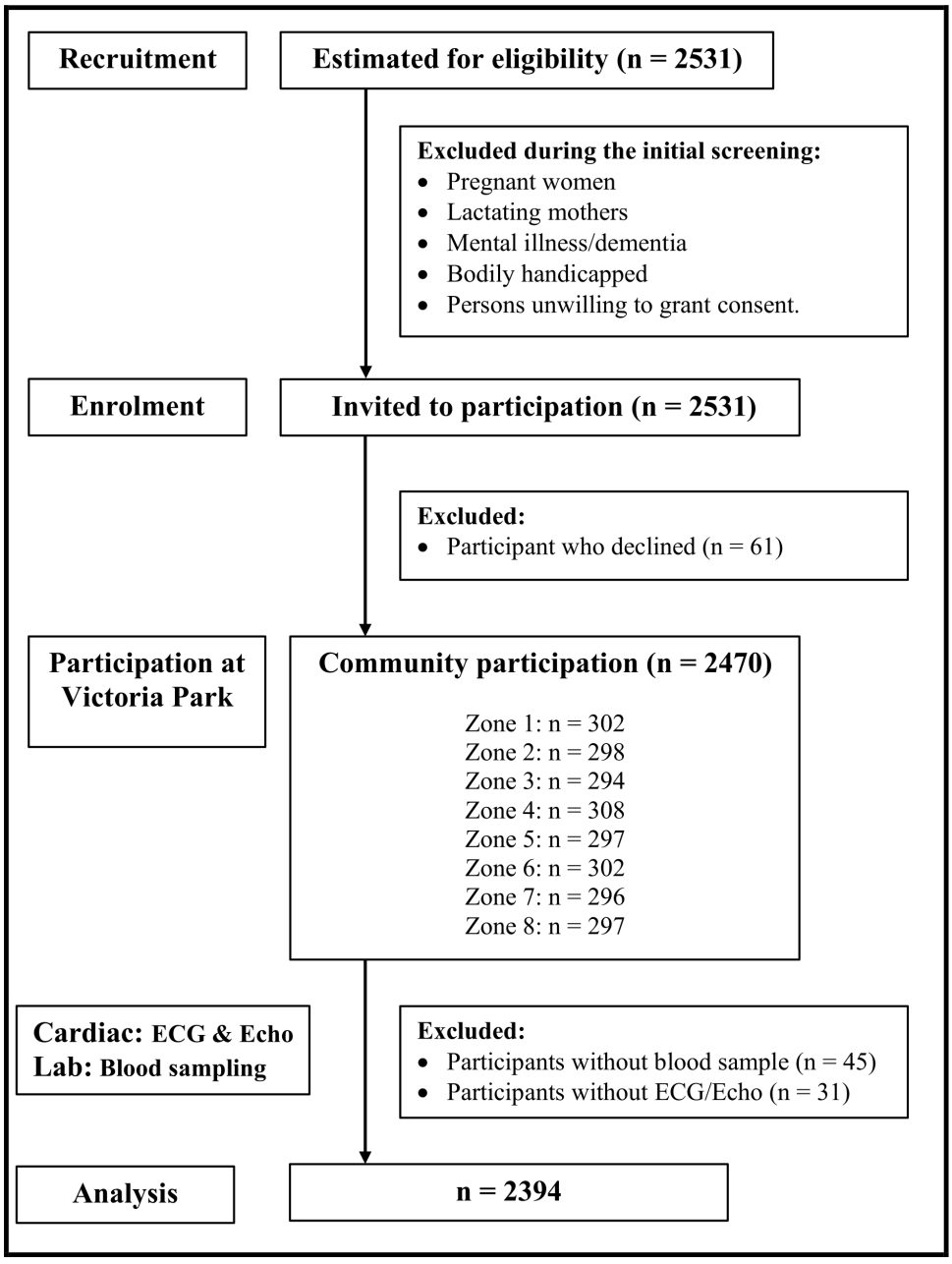

Figure 1 Steps involved during recruitment of participants and final analysis of data.

lipid-lowering medications, was considered an abnormal high.[35]

Preclinical TOD definition

A cardiologist evaluated each participant for cardiac TOD using transthoracic echocardiography (GE vivid e ultrasound system equipped with MSR-RS 1.5 to 3.5 MHz sector and linear probe). The recommended formula for calculating left ventricular mass was used by measuring the two-dimensional guided M-mode imaging. The LVM index was calculated by dividing LVM by body surface area (see online supplemental method 1). Cardiac TOD for 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.[36] Renal TOD was evaluated by using the estimated glomerular filtration rate (eGFR), an essential chronic kidney disease (CKD) marker[37] (online supplemental method 2).

Statistical analysis

Data analysis was done by using IBM SPSS Statistical V.2.6 and STATA V.17 software. Baseline characteristics, CMRFs and TOD characteristics were analysed by sex and zones. Categorical variables were expressed as numbers and percentages, and the Pearson χ^2 test was used to assess the difference. Continuous variables were expressed as mean±SD and compared using a one-way analysis of variance. Median and IQR were used when necessary. Multivariable logistic regression was done to determine associations between demographic characteristics and cardiovascular risk factors. A two-tailed p≤0.05 was

considered statistically significant. Subsequently, receiver operating characteristics (ROCs) were conducted to evaluate and compare the sensitivity of the different CMRFs. A multivariate binary logistic regression with a forced entry for all independent variables assessed the odds of targeted organ damage (LVH, LVMI and CKD) and its association with CMRFs. To determine the influence of potential confounders on the association between CMRFs and targeted organ damage, the following models were generated: model 1 adjusted for age and sex; model 2 adjusted for age, sex, marital status, education level, income and occupation.

RESULTS

Basic characteristics of the study

A total of 2531 participants were recruited into the study, with a response rate of 94.6%. We excluded 54 participants who were absent on the 'screening and awareness campaign' day, 53 who refused blood sampling by venous puncture, and 30 whose ECG and echocardiographic data were missing. Finally, 2394 participants (52.2% female) with a mean age of 41.9±12.3 years (p=0.550) were included in the analysis. Participants from the eight subzonal communities were equally selected without significant differences in population distribution (p=0.950). The baseline sociodemographic and clinical characteristics of all the participants are shown in tables 1 and 2. While unemployment (38.8%) and being single (39.9%) were high among the study participants, we also noted that most of the participants were earning <SLE500 (<US$30) a month. According to WHO criteria, 91.1% of the study population consumed less than three servings of vegetables and fruits per week. Compared with women, more men were physically active (54.4% vs 45.6%) and consumed alcohol (51.5% vs 48.1%).

CMRFs of study participants

The prevalence of hypertension was 35.3%, diabetes mellitus was 8.3%, and combined overweight and obesity (O/O) was 35.6%. In comparison with women, gender differences were not significant in SBP (128.9±23.5 mm Hg vs 128.4±23.3 mm Hg, p=0.516) and DBP (86.0±11.2 mm Hg vs 85.0. ± 12.6 mm Hg, p=0.188). Anthropometric data also revealed significant gender differences in waist-height ratio (WHtR) risk (p<0.001), WC (p<0.001) and WHR (p<0.001).

Association between demographic characteristics and cardiovascular risk factors

The association between baseline demographic characteristics and cardiovascular risk factors were presented in online supplemental table 1. Multivariate logistic regression analysis for hypertension showed that the age group (30–39 years) (OR=0.163; 95% CI: (0.079 to 0.336), p<0.001), high income >SLE 2,000 (OR=0.574; 95% CI: (0.421 to 0.782), p<0.001), unemployed (OR=2.100; 95% CI (1.407 to 3.134), p<0.001) and self-employed (OR=1.912; 95% CI: (1.282 to 2.849), p=0.001) were independently associated with hypertension. The OR of having hypertension was strongest with unemployment. Diabetes mellitus shows a significant association with the age group 30–39 years (OR=0.093; 95% CI: (0.023 to 0.378), p<0.001), income >SLE 2000 (OR=0.548; 95% CI: (0.348 to 0.865), p<0.001), income SLE1100–SLE2000 (OR=0.376; 95% CI: (0.239 to 0.591), p<0.001) and income SLE500–SLE1000 (OR=0.376; 95% CI: (0.239 to 0.591), p<0.001). Dyslipidaemia was associated with the age group 40–49 years (OR=0.255; 95% CI: (0.121 to 0.537), p<0.001), and all occupational groups, including self-employment (OR=5.210; 95% CI: (3.123 to 8.691), p<0.001), unemployment (OR=2.440; 95% CI: (1.469 to 4.052) p=0.001), retired (OR=2.085; 95% CI: (1.276 to 3.408), p=0.003), student (OR=4.389, 95% C.I. (1.778 to 10.834)). Overweight/obesity was significantly associated with all educational levels: primary education (OR=5.781; 95% CI (4.181 to 7.994), p<0.001), secondary education (OR=7.595, 95% CI (5.378 to 10.726), p<0.001), tertiary education (OR=2.220, 95% CI (1.605 to 3.071), p>0.001) and unemployment (OR=0.647, 95% CI (0.452 to 0.925), p<0.001). For alcohol, the regression analysis shows an independent association in all age groups, and all educational levels, while smoking as a risk factor was only associated with participants earning SLE1100–SLE2000. WC was associated with the age group 40–49 years and the various cadres of occupation.

Preclinical TOD of the study participants

In this study, 16.1% had ECG-LVH, while 14.2% had an abnormal LVMI by two-dimensional echo measurement. The participants' impaired kidney function (eGFR) was 11.4%, with eGFR stage II being the highest at 7.1%. Men had a significantly higher risk of eGFR staging than women (p<0.018)

Association of CMRFs with preclinical tissue organ damage

Tables 3–5 show the multivariate binary logistic regression analysis results between CMFRs and the indices of TOD. The OR of CMRF in relation to TOD is also shown in online supplemental figure 1.

In table 3, diabetes mellitus (OR=1.176, 95% CI (0.759 to 1.823)) and dyslipidaemia (OR=1.844, 95% C.I. (1.006 to 3.380)) were strongly associated with LVH. After adjusting sex and age for potential confounders in table 4, model 1 showed that DM was the only CMRF associated with LVH, while in model 2, alcohol and diabetes were associated with LVH. Table 5 shows that the multivariate binary logistic regression analysis for LVMI shows a strong association with diabetes mellitus (OR=1.176, 95% C.I (0.759 to 1.823)) and dyslipidaemia (OR=1.844, 95% C.I (1.006 to 3.380)). On adjusting age and sex to determine the influence of potential confounders, model 1 analysis established an association of diabetes mellitus, dyslipidaemia and WC with LVMI, while model 2 showed an additional cofounder of alcohol being associated with LVMI. For CKD (table 5), the multivariate analysis shows that the odds of having CKD was strongly

Table 1 Sociodemographic and clinical characteristics of participants

Characteristics	Total n (%)	Female n (%)	Male N (%)	
No	2394	1250	1144	P value (χ^2)
Age, by group				
20–29 years	447 (18.7)	255 (57.0)	192 (43.0)	<0.001 (28.9)
30–39 years	703 (29.4)	383 (54.5)	320 (45.5)	
40–49 years	684 (28.6)	359 (52.5)	325 (47.5)	
50–59 years	356(14,9)	141 (39.6)	215 (60.4)	
>60 years	204 (8.5)	112 (54.9)	92 (45.1)	
Income (currency=Leone)				
0–500	920(38,4)	504 (54.8)	416 (45.2)	0.052 (7.715)
500–1000	666 (27.8)	319 (47.9)	347 (52.1)	
1100–2000	490 (20.5)	256 (52.2)	234 (46.2)	
>2000	318 (13.3)	171 (53.8)	147 (46.2)	
Education level				
None	618 (25.8)	318 (51.5)	300 (48.5)	0.095 (6.361)
Primary	479 (20.0)	273 (57.0)	206 (43.0)	
Secondary	835 (34.9)	432 (51.7)	403 (48.3)	
Tertiary	462 (19.3)	227 (49.1)	235 (50.9)	
Marital status				
single	955 (39.9)	504 (52.8)	451 (47.2)	0.699 (1.428)
Married	737 (30.8)	372 (50.5)	365 (49.5)	
Seperated/divorce	586 (24.5)	310 (52.9)	276 (47.1)	
Widow	115 (4.8)	63 (54.8)	52 (45.2)	
Occupation				
Employed	500 (20.9)	302 (60.4)	198 (39.6)	<0.001 (27.6)
Self employed	531 (22.2)	241 (45.4)	290 (54.6)	
Unemployed	930 (38.8)	466 (50.1)	464 (49.9)	
Retired	167 (7.0)	96 (57.5)	71 (42.5)	
Student	264 (11.0)	145 (54.9)	119 (45.1)	
Blood pressure (mm Hg)				
Normal	1061 (44.3)	510 (48.1)	551 (51.9)	0.002 (15.15)
Prehypertension	489 (20.4)	278 (56.9)	211 (43.1)	
Hypertension stage 1	644 (26.9)	360 (55.9)	284 (44.1)	
Hypertension stage 2	200 (8.4)	102 (51.0)	98 (49.0)	
Diabetes (mmol/l)				
Normal (<6 mmol/L)	2084 (87.1)	1080 (51.8)	1004 (48.2)	0.234 (2.9)
Pre-diabetes (6.0–6.9 mmol/L)	103 (4.3)	51 (49.5)	52 (50.5)	
Diabetes (>7.0 mmol/L)	199 (8.3)	115 (57.8)	84 (42.2)	
Fruits/vegetable				
<3 serving	2182 (91.1)	1143 (52.4)	1039 (47.6)	0.592 (0.28)
>3 serving	212 (8.9)	107 (50.5)	105 (49.5)	
Alcohol				
Never	1486 (62.1)	783 (52.7)	703 (47.3)	0.234 (2.9)
Current previous	652 (27.2)	316 (48.5)	336 (51.5)	
Previous	256 (10.7)	151 (59.0)	105 (41.0)	
Smoking				

Continued

Table 1 Continued

Characteristics	Total n (%)	Female n (%)	Male N (%)	
No	**2394**	**1250**	**1144**	**P value (χ^2)**
Never	2073 (86.6)	1088 (52.5)	985 (47.5)	0.797 (0.45)
Current	198 (8.3)	100 (50.5)	98 (49.5)	
Ex smoker	123 (5.1)	62 (50.4)	61 (49.6)	
Daily physical activity				
Low	895 (37.4)	481 (53.7)	414 (46.3)	
Moderate	939 (39.2)	515 (54.8)	424 (45.2)	
Vigorous	511 (21.3)	233 (45.6)	278 (54.4)	
Lipids				
Total cholesterol (TC) (≥6.2 mmol/L)				
Normal	2163 (90.4)	1171 (54.1)	992 (45.9)	<0.001 (32.25)
High	231 (9.6)	79 (34.2)	152 (65.8)	
LDL-C (≥3.3 mmol/L)				
Normal	2077 (86.8)	1083 (52.1)	994 (47.9)	0.858 (0.03)
High	317 (13.2)	167 (52.7)	150 (47.3)	
HDL-C (≤1.04 mmol/L)				
Normal	2129 (88.9)	1142 (53.6)	987 (46.4)	<0.001 (15.68)
High	317 (13.2)	108 (40.8)	157 (59.2)	
Triglyceride (≥1.7 mmol/L)				
Normal	1862 (77.8)	1011 (54.3)	851 (45.7)	<0.001 (14.58)
High	530 (22.1)	238 (44.9)	292 (55.1)	
Measures of adiposity				
BMI (kg/m^2)				
Underweight	38 (1.6)	21 (55.3)	17 (44.7)	0.066 (7.2)
Normal	1502 (62.7)	794 (52.9)	708 (47.1)	
Overweight	612 (25.6)	329 (53.8)	283 (46.2)	
Obese	240 (10.0)	106 (44.2)	134 (55.8)	
Waist circumference (≥94 cm men, ≥80 cm women)				
Normal	1882 (78.6)	870 (46.2)	1012 (53.8)	0.003 (8.569)
Abnormal	512 (21.4)	274 (53.5)	238 (46.5)	
WHtR risk				
Normal (≤0.5)	1050 (43.9)	36 (3.4)	1014 (96.6)	<0.001 (1786.73)
Increased risk (0.51–0.59)	1276 (53.3)	1146 (89.8)	130 (10.2)	
High risk (>0.6)	68 (2.8)	68(100)	0 (0.0)	
WHR (≥0.90 men, ≥0.85 women)				
Low (≤0.5)	763 (31.9)	753 (98.7)	10 (1.3)	0.210 (3.12)
Moderate (0.51–0.59)	273 (11.4)	271 (99.3)	2 (0.7)	
High (≥0.6)	207 (8.6)	207(100)	0 (0.0)	
Target organ damage				
ECG-LVH (mm/mV)				
No	2009 (83.9)	951 (47.3)	1058 (52.7)	0.315 (1.010)
Yes	385 (16.1)	193 (50.1)	192 (49.9)	
LVMI (g/m^2)				
No	2055 (85.8)	969 (47.2)	1086 (52.8)	0.127 (2.329)
Yes	339 (14.2)	175 (51.6)	164 (48.4)	

Continued

Table 1 Continued

Characteristics	Total n (%)	Female n (%)	Male N (%)	
No	2394	1250	1144	P value (χ^2)
eGFR stages (mL/min/1.73 m^2)				
Stage I	2123 (88.7)	1124 (52.9)	999 (47.1)	0.018 (10.06)
Stage II	169 (7.1)	77 (45.6)	92 (54.4)	
Stage III	96 (4.0)	49 (51.0)	47 (49.0)	
Stage IV	6 (0.3)	0 (0.0)	6 (100)	
eGFR (mL/min/1.73 m^2)				
Normal	2120 (88.6)	1022 (48.2)	1098 (51.8)	0.251 (1.318)
Abnormal	274 (11.4)	122 (44.5)	152 (55.5)	

BMI, body mass index; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVH, left ventricular hypertrophy; LVMI, Left Ventricular Mass Index; WHtR, Waist-Height-Ratio.

associated with diabetes mellitus (OR=1.212, 95% CI (0.741 to 1.983)), hypertension (OR=1.163, 95% CI (0.887 to 1.525)), alcohol (OR=1.003, 95% CI (0.772 to 1.303)), low HDL-C (OR=1.261, 95% CI (0.881 to 1.804)), high LDH-C (OR=1.355 95% CI (0.754 to 2.433)) and TC (OR=1.170, 95% CI (0.663 to 2.066)). Regression (model 1 and model 2) adjustment analysis demonstrated diabetes mellitus and high low HDL-C as the strongest determinant for LVH.

The relationship between clinical sensitivity and specificity for ECG-LVH as a TOD by gender was evaluated using the ROC curve. A low optimal cut-off point for ECG-LVH (male 24.5 vs female 27.5 mm) was required to maximise sensitivity and specificity (figure 2). Additional information on the sensitivity and specificity of parameters related to TOD is shown in online supplemental table 2.

DISCUSSION

Health screenings are essential for identifying the burden of cardiovascular risk factors and their complications

Table 2 Mean (±SD) of specific demographic, clinical and biochemical characteristics of participants stratified by sex

Characteristics	Total, mean (±SD)	Female, mean (±SD)	Male, mean (±SD)	P value
Age (year)	41.9 (12.3)	42 (12)	42 (13)	0.550
BMI (kg/m^2)	24.8 (4.7)	24.62 (4.4)	25.04 (4.99)	0.029
WC (cm)	87.1 (8.3)	93.63 (4.47)	80.0 (5.01)	<0.001
WHtR	0.5 (0.05)	0.54 (0.03)	46 (0.03)	<0.001
WHR	0.88 (0.05)	0.94 (0.06)	0.81 (0.06)	<0.001
SBP (mm Hg)	127.8 (23.3)	128.9 (23.5)	128.4 (23.3)	0.516
DBP (mm Hg)	85.7 (11.3)	86 (11)	85 (12)	0.188
Triglyceride	1.65 (0.34)	1.63 (0.32)	1.66 (0.35)	0.013
Total cholesterol (mmol/l)	4.97 (0.72)	4.89 (0.66)	5.05 (0.77)	<0.001
HDL-C (mmol/l)	1.29 (0.27)	1.31 (0.24)	1.28 (0.29)	0.016
LDL-C (mmol/l)	2.99 (0.66)	3.00 (0.65)	2.98 (0.66)	0.315
TC/HDL	4.25 (2.29)	4.06 (1.98)	4.46 (2.58)	<0.001
LDL/HDL	2.51 (1.25)	2.47 (1.19)	2.56 (1.30)	0.085
Non-HDL-C	3.67 (0.84)	3.59 (0.75)	3.77 (0.92)	<0.001
Non-HDL/HDL	3.25 (2.29)	3.06 (1.98)	3.46 (2.58)	<0.001
FBS (mmol/l)	5.10 (1.64)	5.16 (1.67)	5.04 (1.61)	0.080
HbA1c (%)	5.21 (1.03)	5.21 (1.09)	5.20 (0.96)	0.964
Creatinine level (ummol/l)	79.63 (21.19)	86 (22)	73 (17)	<0.001
eGFR (mL/min/1.73 m^2)	98.37 (15.63)	100 (15)	97(16)	<0.001

BMI, body mass index; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; FBS, Fasting Blood Sugar; HbA1c, Glycated Hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SBP, systolic blood pressur; TC, total cholesterol; WC, waist circumference; WHtR, waist-height ratio.

Table 3 Association between cardiometabolic risk factors and specific target organ damage (ECG-LVH)

ECG-LVH						
Variable	**Total no (%)**	**Yes no (%)**	**No (%)**	**OR (95% CI)**	**Model 1**	**Model 2**
Overweight/obese						
No	1598 (66.8)	239 (15.0)	1359 (85.0)	Ref.		
Yes	796 (33.2)	146 (18.3)	650 (81.7)	0.793 (0.631 to 0.996)	0.784 (0.625–0.984)	0.693 (0.542–0.885)
Alcohol						
No	1486 (62.1)	236 (15.9)	1250 (84.1)	Ref.		
Yes	908 (37.9)	149 (16.4)	759 (83.6)	0.938 (0.748 to 1.176)	0.880 (0.688–1.125)	1.032(0.756–1.409)
Smoking						
No	2073 (86.6)	328 (15.8)	1745 (84.2)	Ref.		
Yes	321 (13.4)	57 (17.8)	264 (82.2)	0.871 (0.637 to 1.176)	0.751(0.518–1.088)	0.764 (0.526–1.109)
Diabetes						
No	2187(91,7)	356 (16.3)	1831 (83.7)	Ref.		
Yes	199 (8.3)	27 (13.6)	172 (86.4)	1.255 (0.822 to 1.916)	1.276 (0.833–1.955)	1.325 (0.861–2.035)
Hypertension						
No	1550 (64.7)	237 (15.3)	1313 (84.7)	Ref.		
Yes	844 (35.3)	148 (17.5)	696 (82.5)	0.843 (0.671 to 1.058)	0.859 (0.680–1.084)	0.856(0.676–1.084)
Waist circumference						
Normal	1882 (78.6)	300 (15.9)	1582 (84.1)	Ref.		
Abnormal	512 (21.4)	85 (16.6)	427 (83.4)	0.952 (0.730 to 1.240)	0.968 (0.736–1.273	0.948 (0.717–1.254)
Total cholesterol						
Normal	2163 (90.4)	344 (15.9)	1819 (84.1)	Ref.		
Abnormal	231 (9.6)	41 (17.7)	190 (82.3)	0.742 (0.454 to 1.210)	0.901 (0.624–1.302)	0.860(0.593–1.247)
LDL-C						
Normal	2077 (86.8)	327 (15.7)	1750 (84.3)	Ref.		
High	317 (13.2)	58 (18.3)	259 (81.7)	0.647 (0.379 to 1.107)	0.834(0.610–1.142)	0.811(0.586–1.122)
Low HDL-C						
No	1985 (82.9)	311 (15.7)	1674 (84.3)	Ref.		
Yes	409 (17.1)	74 (18.1)	335 (81.9)	0.835 (0.630 to 1.107)	0.841 (0.636–1.113)	0.852 (0.643–1.129)
Dyslipidaemia						
No	1882 (78.6)	300 (15.9)	1582 (84.9)	Ref.		
Yes	512 (21.4)	85 (16.6)	427 (83.4)	1.449 (0.834 to 2.518)	0.968 (0.736–1.273)	0.948 (0.717–1.254)

HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVH, left ventricular hypertrophy.

Table 4 Association between cardiometabolic risk factors and specific target organ damage (LVMI)

LVMI						
Variable	**Total no (%)**	**Yes no (%)**	**No (%)**	**OR (95% CI)**	**Model 1**	**Model 2**
Overweight/obese						
No	1598 (66.8)	210 (13.1)	1388 (86.9)	Ref.		
Yes	796 (33.2)	129 (16.2)	667 (83.8)	0.790 (0.621 to 1.005)	0.777 (0.612–0.987)	0.712 (0.540–0.921)
Alcohol						
No	1486 (62.1)	208 (14.0)	1278 (86.0)	Ref.		
Yes	908 (37.9)	131 (14.0)	777 (85.5)	0.945 (0.745 to 1.199)	0.915 (0.705–1.186)	1.088(0.781–1.516)
Smoking						
No	2073 (86.6)	289 (13.9)	1784 (86.1)	Ref.		
Yes	321 (13.4)	50 (15.6)	271 (84.4)	0.883 (0.634 to 1.229)	0.654 (0.448–0.953)	0.668 (0.457–0.977)
Diabetes						
No	2187 (91.7)	313 (14.3)	1874 (85.7)	Ref.		
Yes	199(8,3)	25 (12.6)	174 (87.4)	1.176 (0.759 to 1.823)	1.197 (0.770–1.863)	1.259 (0.805–1.970)
Hypertension						
No	1550 (64.7)	209 (13.5)	1341 (86.5)	Ref.		
Yes	844 (35.3)	130 (15.4)	714 (84.4)	0.857(0.674 to 1.088)	0.869 (0.680–1.111)	0.867 (0.676–1.112)
Waist circumference						
Normal	1882 (78.6)	265 (14.1)	1617 (85.9)	Ref.		
Abnormal	512 (21.4)	74 (14.5)	194 (84.0)	0.956 (0.724 to 1.264)	1.017 (0.762–1.358)	1.010 (0.751–1.357)
Total cholesterol						
Normal	2163 (90.4)	302 (14.0)	1791 (86.02)	Ref.		
Abnormal	231 (9.6)	37 (16.0)	264 (83.3)	0.646 (0.387 to 1.079)	0.908 (0.619–1.332)	0.861 (0.584–1.269)
LDL-C						
Normal	2077 (86.8)	286 (13.8)	1791 (86.3)	Ref.		
High	317 (13.2)	53 (16.7)	264 (83.3)	0.501 (0.280 to 0.894)	0.810 (0.585–1.123)	0.821 (0.585–1.152)
Low HDL-C						
No	1985 (82.9)	271 (13.7)	1714 (86.3)	Ref.		
Yes	409 (17.1)	68 (16.6)	341 (83.4)	0.768 (0.573 to 1.028)	0.789 (0.590–1.056)	0.801 (0.597–1.073)
Dyslipidaemia						
No	1882 (78.6)	265 (14.1)	1617 (85.9)	Ref.		
Yes	512 (21.4)	74 (14.5)	438 (85.5)	1.844 (1.006 to 3.380)	1.017 (0.762–1.358)	1.010 (0.751–1.357)

HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVMI, Left Ventricular Mass Index.

Table 5 Association between cardiometabolic risk factors and specific target organ damage (CKD)

CKD						
Variable	**Total no (%)**	**Yes no (%)**	**No (%)**	**OR (95% CI)**	**Model 1**	**Model 2**
Overweight/obese						
No	1598 (66.8)	168 (10.5)	1430 (89.5)	Ref.		
Yes	796 (33.2)	106 (13.3)	690 (86.7)	0.750 (0.578 to 0.974)	0.774 (0.596–1.005)	0.778 (0.589–1.028)
Alcohol						
No	1486 (62.1)	172 (11.6)	1314 (88.4)	Ref.		
Yes	908 (37.9)	102 (11.2)	1844 (89.0)	1.003 (0.772 to 1.303)	0.915 (0.688–1.218)	0.810 (0.565–1.161)
Smoking						
No	2073 (86.6)	229 (11.0)	1844 (89.0)	Ref.		
Yes	321 (13.4)	45 (14.0)	276 (86.0)	0.784 (0.554 to 1.111)	0.678 (0.448–1.024)	0.665 (0.439–1.008)
Diabetes						
No	2187 (91.7)	254 (11.6)	1933 (88.4)	Ref.		
Yes	199 (8.3)	19 (9.5)	180 (90.5)	1.212 (0.741 to 1.983)	1.200 (0.730–1.975)	1.208 (0.730–1.997)
Hypertension						
No	1550 (64.7)	186 (12.0)	1364 (88.0)	Ref.		
Yes	844 (35.3)	88 (10.4)	756 (89.6)	1.163 (0.887 to 1.525)	1.126 (0.852–1.486)	1.118 (0.843–1.483)
Waist circumference						
Normal	1882 (78.6)	213 (11.3)	1669 (88.7)	Ref.		
Abnormal	512 (21.4)	61 (11.9)	451 (88.1)	0.926 (0.683 to 1.254)	0.870 (0.634–1.193)	0.825 (0.598–1.139)
Total cholesterol						
Normal	2163 (90.4)	247 (11.4)	1916 (88.6)	Ref.		
Abnormal	231 (9.6)	27 (11.7)	204 (88.3)	1.170 (0.663 to 2.066)	0.876 (0.566–1.356)	0.862 (0.555–1.339)
LDL-C						
Normal	2077 (86.8)	239 (11.5)	1838 (88.5)	Ref.		
High	317 (13.2)	35 (11.0)	282 (89.2)	1.355 (0.754 to 2.433)	1.000 (0.682–1.465)	0.951 (0.641–1.409)
Low HDL-C						
No	1986 (82.9)	235 (11.8)	1751 (88.2)	Ref.		
Yes	409 (17.1)	39 (9.5)	370 (90.5)	1.261 (0.881 to 1.804)	1.277 (0.893–1.827)	1.275 (0.890–1.825)
Dyslipidaemia						
No	1882 (78.6)	213 (11.3)	1669 (88.7)	Ref.		
Yes	512 (21.4)	61 (11.9)	451 (88.1)	0.706 (0.399 to 1.252)	0.870 (0.634–1.193)	0.825 (0.598–1.139)

CKD, chronic kidney disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

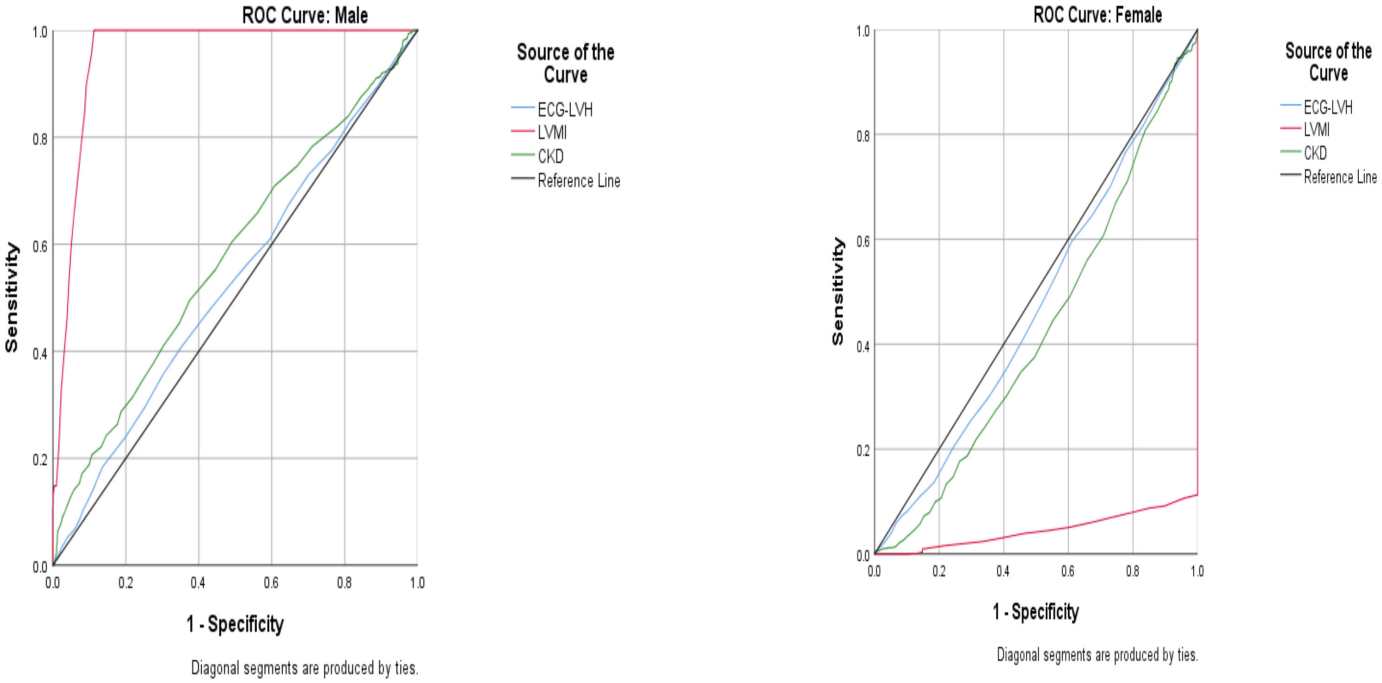

Figure 2 Area under the curve for specific TOD (LVH, LVMI, eGFR). CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; LVH, left ventricular hypertrophy; LVMI, Left Ventricular Mass Index; ROC, receiver operating characteristics; TOD, target organ damage.

(kidney disease, stroke and coronary artery disease), as these may adversely influence the quality of life of an individual.[38] This notwithstanding, population-based screening remains limited in LMICs. Our study provides the largest data on CMRFs in health screening for NCD in Sierra Leone.

It is the first study to characterise the distribution of CMRFs and preclinical TOD among adults in Sierra Leone. Our findings indicate that CMRFs are common among adult Sierra Leoneans with hypertension, diabetes and dyslipidaemia having the strongest association with specific preclinical TOD. The study suggests a high prevalence of CMRFs for CVD, as many Sierra Leonean adults have at least one significant risk factor: hypertension (35.6%), diabetes mellitus (8.3%), overweight/obesity (37.3%), abdominal obesity (21.4%), dyslipidaemia (21.4%) and alcohol consumption (37.7%). The reported prevalence in our study is consistent with findings from other studies in SSA.[39–41] In Sierra Leone, the observed patterns of CMRFs indicate that a demographic health transition might be occurring faster than previously reported.[22 24 26] Therefore, our study has contributed critical evidence on the burden and distribution of CMRFs among adults living in an urban setting in SSA.

This study's prevalence of hypertension (35.5%) is similar to other community-based studies in SSA.[42–44] This prevalence of hypertension was identical to the previous WHO STEPS survey in 2009 that reported 37% in males and 33% in females.[22] The study design and age population of 25–65 years used in the STEPs survey make it difficult to compare with our study. In Sierra Leone, a much higher prevalence of hypertension (49.6%) was recently reported in a provincial district by Odland *et al*. In comparison, a lower rate of hypertension (22%) was reported by Geraedts *et al* when compared with this study.[24 26] The disparity may be attributed to the age differences of the studied cohorts (20 years and above in our study, unlike 40 years and above in the reported survey by Odland *et al*[24]). The difference may also be ascribed to the study design, sociodemographic characters and lifestyle patterns of the study participants. Our estimated prevalence (8.3%) of type 2 diabetes mellitus is higher than the prevalence reported from other studies in Sierra Leone—3.5% in 2009, 5.5% in 2021, 6.2% in 2017, 2.4% in the urban population and 0% in the rural population in 1997.[23 24 45] The high urban prevalence of diabetes in our study was partly due to the combined use of FBG and HbA1c, unlike other studies conducted in Sierra Leone and the greater variance in fasting glucose among urban participants. Additionally, the high prevalence of DM in this study compared with previous studies could be partly attributable to previous studies being 10–15 years earlier. Even though the prevalence of diabetes is higher in this study, the small population size and methodology used in previous studies would make comparisons difficult.

Overweight (26.5%) and obesity (10.0%) were surprisingly more common in our study, as Sierra Leone is one of the poorest countries in the world. The estimated 36.5% of overweight/obese reported in this study is higher than the 25% reported by Odland *et al*, the first study to evaluate CVDRFs in a larger sample size in Sierra Leone.[24] Our study's estimated finding of O/O is consistent with other studies from Ghana, Nigeria and Ethiopia.[46–48] Our study's high proportion of individuals with increased BMI

may suggest an upward trend in this risk factor, thereby supporting the hypothesis of rapid urbanisation and a westernised lifestyle. Previous studies have indicated that WC, as an indicator of abdominal obesity, correlates positively with a risk for cardiovascular diseases.[49] Abdominal obesity was more common in our study, with men more likely than females to be affected, probably because of the tendency of central obesity. Similarly, a study conducted in Ethiopia showed WC to be associated with hypertension.[50]

Even though BMI is an independent cardiometabolic risk for cardiovascular diseases, there is evidence strongly suggesting WHtR>0.5 as the highest predictor of all CMRFs for both sexes, even more than BMI and WC combined.[51 52] When WHtR was analysed, more than half of the study participants were categorised into 'increased risk (53.3)' and 'high risk (2.8)'. WHtR as a predictor of cardiovascular events was generally higher in our study than BMI and WC. This result confirmed earlier findings in existing literature.[52] The results of WHtR>0.5 allow us to conclude that more adults Sierra Leoneans are at 'early health risk' for cardiovascular disease.

The previous perception that dyslipidaemia was rare among black Africans is now being discredited by several studies showing a high prevalence of dyslipidaemia among black Africans.[53 54] In this study, elevated TG (22.1%) was the most prominent form of dyslipidaemia, followed by an elevated LDL-C and HDL-C, with women having the highest prevalence of all measures of dyslipidaemia in comparison to men. This observed pattern of dyslipidaemia prevalence in our study is similar to a survey conducted in Ghana[55] but inconsistent with results documented by Asiki *et al*[54] and Gebreegziabiher *et al*,[56] where the most prevalent dyslipidaemia markers were HDL-C, TC and LDL-C. Despite the observed disparities in the different measures of dyslipidaemia, studies have reported a high prevalence of all forms of dyslipidaemia among women.[57–60] This study further demonstrated that women were more likely to have high levels of low HDL-C although the widely accepted belief that HDL is male specific.[54 56] These findings illustrate the importance of health screening for dyslipidaemia as many of the study participants are dyslipidaemic.

WHO has identified several major risk factors for a cardiovascular disorder, including smoking, alcohol consumption, unhealthy diets and physical inactivity.[3 31] About one-third of the participants had consumed alcohol in this study, and the rate/frequency of consumption was high. Our report is higher than the WHO-reported general prevalence of alcohol consumption in most SSA countries.[3] The increased consumption of alcohol in our study could partly be attributed to our youthful participants that comprised about half of the cohort with the ability to afford its cost. It was observed in this study that one-third of participants do not engage in any form of exercise, with women being less educated, unemployed and physically inactive than their men counterparts. These results are consistent with reports from a Ghanaian study.[55] Cigarette smoking was generally uncommon in our study, but the impact of 'Shesha pipe smoking' among young age must be evaluated.

Our analysis to identify the association between CMRFs and some demographic variables revealed that hypertension was associated with the youthful age group, non-employment status and increased income. Nevertheless, diabetes mellitus was associated with the young and increased income. Dyslipidaemia was associated with middle age and non-employment. Education level, all age groups and being a student were associated with alcohol consumption. Earning more income was associated with smoking, while young age and all employment status were associated with WC. Our study's findings are consistent with several SSA studies[4 12 61 62] and confirm our earlier statement that CMRFs are the principal causes of cardiovascular diseases in Sierra Leone.

We investigate the role of CMRFs in developing preclinical TOD. Studies on LVH are scarce in Africa because of the non-availability of electrocardiograms and echocardiograms in many settings. However, few studies on the black population living in Africa show an overall prevalence of LVH of 4.1% in Ghana, 62% in Cameroon, 41% in the Gambia and 41% in Angola.[55 63–65] In our study, the prevalence of LVH by ECG and LVMI was 16.1% and 12.4%, respectively. Our findings were higher than the Ghanaian study[64] but comparatively lower than other African reports. The odds of having LVH either by ECG or LVMI were further evaluated in this study, and our findings demonstrated a strong association with diabetes and dyslipidaemia. Other studies have found hypertension to have a strong association with LVH, even though it is inconsistent with our findings. The weak association of hypertension with LVH in this study could be attributed to our youthful study population (about half of the population is less than age 40 years), as most of the hypertensives were young. Studies have reported that LVH in hypertensives is increased several fold with ageing and in hypertensives with risk factor-adjusted cardiovascular morbidity, which was unlike our study.[66 67]

Using the regression model adjustment analysis, diabetes mellitus was identified as the strongest determinant for LVH in our study. Other studies have reported LVH to be common among patients with diabetes, with LVH being a strong predictor of cardiovascular disease in diabetics.[68 69] Since hypertension is a low predictor of LVH in this study, an ROC curve was performed to show the relationship between clinical sensitivity and specificity for ECG-LVH cut-off. This demonstrated that a low cut-off points for ECG-LVH (male 24.5 mm vs female 27.5 mm) were required to maximise sensitivity and specificity. This analysis suggests that LVH may occur at a much lower cut-off for Sierra Leoneans and that the standard cut-off points for LVH may fail as a screening tool for TOD in this setting. These findings need further research and in-depth evaluation in future studies. The prevalence of CKD in our population was 11.6%, and the odds of having CKD were strongly associated with diabetes mellitus, hypertension, alcohol, low HDL-C, high LDH-C and TC.

Regression (model 1 and model 2) adjustment analysis demonstrated diabetes mellitus and high low HDL-C as the strongest determinant for LVH. These findings confirmed the recent results by Coker *et al* who reported diabetes mellitus as the second most common cause of CKD for admission into a tertiary hospital in Sierra Leone, while Kachimanga *et al* reported a high prevalence of 29.9% CKD in Rural Sierra Leone.[70 71] Hence, the strong association of diabetes mellitus as a risk factor for CKD observed in this study is a wake-up call for action on kidney disease screening and prevention programmes in Sierra Leone.

Our findings should be interpreted within the context of the following limitation. Since the study is cross-sectional in design, it could not conclude direct causality inference of risk factors and effect outcomes. Additionally, as a health screening study, some of the clinical outcomes were not repeated, and this may result in measurement errors, with the potential of underestimating CMRFs. CKD assessment by single serum creatinine without assessing for proteinuria, which indicates the presence of CKD, will also lead to an underestimation of CKD. However, the findings in our study are consistent with other large prospective studies in LMICs.

Conclusion

The study provides novel data-driven information on the burden of cardiometabolic risk and its association with TOD, as it is the first health screening survey on a larger population in Sierra Leone. This study's relatively high prevalence of CMRFs indicates that CVD is increasing in Sierra Leone, a country whose health services are already overburdened by tuberculosis, malaria and HIV/AIDS. Despite the various assumptions underlying these projections, the importance of this work cannot be overestimated. The result of this study could serve as the basis for advocacy with an urgent call for action in establishing programmes that would improve the control and management of CMRFs and CVD, along with other NCDs.

Author affiliations
[1]Department of Internal Medicine, College of Medicine And Allied Health Sciences, University of Sierra Leone, Freetown, Sierra Leone
[2]Directorate of Non-Communicable Diseases and Mental Health, Sierra Leone Ministry of Health and Sanitation, Freetown, Sierra Leone
[3]Department of Mathematics and Statistics, Fourah Bay College, University of Sierra Leone, Freetown, Sierra Leone
[4]School of Medical Sciences, Department of Internal Medicine, University of Cape Coast, Cape Coast, Ghana
[5]Department of Pharmacology and Therapeutics, Faculty of Basic Medical Sciences, College of Medicine and Allied Health Sciences, University of Sierra Leone, Freetown, Sierra Leone
[6]Department of Community Health, Faculty of Clinical Sciences, College of Medicine & Allied Health Sciences, University of Sierra Leone, Freetown, Sierra Leone
[7]Department of Internal Medicine, Choithrams Memorial Hospital, Freetown, Sierra Leone

Twitter James Baligeh Walter Russell @James B.W. Russell@AProfJamesBWRu

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Contributors JBWR and SS contributed equally to this work. JBWR, SS, TRK and SC were responsible for the design of the study. VC and MS were responsible for coordinating data project acquisition and community recruitment of participants. SKS, TRK and JK performed the statistical analyses. The first draft was written by JBWR, SS, AB and OTA. OZM, JC, AJ and SL reviewed and edited the final manuscript. DRL reviewed all stages of the drafted manuscript for important intellectual content. All authors contributed to data interpretation, critically reviewed the first draft, approved the final version and agreed to be accountable for the work. JBWR is the guarantor of this study.

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Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

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Ethics approval This study involves human participants and was approved by Government of Sierra Leone Office of the Sierra Leone Ethics and Scientific Review Committee. Ministry of Health and Sanitation Sierra Leone. An ID for our ethics approval was not given and this is the standard practice of the ethics committee. Participants gave informed consent to participate in the study before taking part.

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Data availability statement Data are available on reasonable request. The anonymised dataset supporting this study's findings is available on reasonable request from the corresponding author as cited in the publication.

ORCID iDs
James Baligeh Walter Russell http://orcid.org/0000-0003-4732-6588
Theresa Ruba Koroma http://orcid.org/0000-0002-6826-7102
Santigie Sesay http://orcid.org/0000-0003-0899-3935
Sulaiman Lakoh http://orcid.org/0000-0002-7639-0004
Ansumana Bockarie http://orcid.org/0000-0003-1173-1493
Joseph Sam Kanu http://orcid.org/0000-0003-0799-6907
Abdul Jalloh http://orcid.org/0000-0001-6596-1097
Victor Conteh http://orcid.org/0000-0003-0560-8894

Sorie Conteh http://orcid.org/0000-0002-2827-9707
Mohamed Smith http://orcid.org/0000-0003-3004-2298
Durodami R Lisk http://orcid.org/0000-0002-7085-3055

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