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Left ventricular mass independently associates with 24-hour sodium excretion in young masked hypertensive adults: The African-PREDICT study

Bianca van der Westhuizen [a,1], Aletta E. Schutte [a,b,1], Lebo F. Gafane-Matemane [a,b,1], Ruan Kruger [a,b,*,1]

[a] Hypertension in Africa Research Team, Faculty of Health Sciences, North-West University, Potchefstroom, South Africa
[b] MRC Extramural Research Unit for Hypertension and Cardiovascular Disease, North-West University, Potchefstroom, South Africa

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ABSTRACT

Background: Due to the known contribution of excess sodium intake on elevations in blood pressure, salt reduction regulations are being introduced in countries all over the world. To study the contribution of sodium intake on cardiovascular disease development, we determined whether left ventricular mass associates with sodium excretion in young adults free from overt cardiovascular disease and those with masked hypertension.
Methods: We included 681 participants (41% men and 50% black) in a cross-sectional analysis from the African-PREDICT study with complete 24-hour urine collections and successful ambulatory blood pressure monitoring (>70% valid readings). The participants were categorized as normotensive (n = 534) or masked hypertensive (n = 147). In addition, we determined left ventricular mass index (LVMI) along with traditional risk factors.
Results: Masked hypertensive individuals had higher sodium excretion (149 vs. 128 mmol/L/day) and LVMI (78.1 vs. 69.6 g/m^2) than normotensives. In single, partial and multiple regression analyses, LVMI independently associated with higher sodium excretion in the total group of young adults ($\beta = 0.089$; $p = 0.011$). This result was also evident among masked hypertensives ($\beta = 0.215$; $p = 0.008$), but not in normotensives ($\beta = 0.054$; $p = 0.134$).
Conclusion: Our results indicated that higher sodium excretion (reflecting a higher salt intake) may contribute to increased left ventricular mass, potentially driven by the early development of masked or undetected hypertension.

1. Introduction

The effects of a diet high in sodium on elevating blood pressure are well reported [1–3]. As in numerous countries around the world, the South African Government has also implemented a sodium reduction regulation (R.214) to curb the high prevalence of hypertension [4–6] enforcing mandatory reduction in the sodium content of certain processed foods [7]. Despite the well-known link between hypertension and excess sodium intake, a review highlighted an excess sodium intake in the diet has direct adverse effects on target organs, beyond the increased risk for hypertension [8].

The diagnosis of hypertension evolved in the past years and the classification thereof became more complex. Masked hypertension entails the combination of non-elevated clinical blood pressure with elevated ambulatory blood pressure [9]. Masked hypertensives are at great risk of target organ damage such as increased left ventricular mass and left ventricular dysfunction [10–12]. Masked hypertension is less easily diagnosed and are therefore untreated with individuals being unaware of their increased cardiovascular risk [13], hence the importance of investigating this group of people in a young population. A recent study, that included the same sample population as the current study, reported 16.4% of the population had masked hypertension, and that masked hypertensive individuals were more likely to present with increased left ventricular mass index (LVMI) compared to normotensive individuals [14]. There was also found to be no ethnic-specific difference in masked hypertension frequency in young black and white individuals in this population group [15]. Another study using a sub-sample of individuals within the present research project reported a 133 mmol/d dietary intake of sodium, which is well above what the World Health Organization (WHO) recommends [16].

In light of these findings, the aim of the current study was to investigate whether left ventricular mass relates to 24-hour urinary sodium

* Corresponding author at: Hypertension in Africa Research Team, North-West University, Private Bag X6001, Potchefstroom 2520, South Africa.
E-mail address: ruan.kruger@g.nwu.ac.za (R. Kruger).
[1] All authors takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

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excretion in young healthy adults, with a secondary aim of investigating differences between individuals with normal blood pressure as well with masked hypertension.

2. Methods

2.1. Study population

Data was obtained from participants enrolled in the African-PREDICT (Prospective study on the Early Detection and Identification of Cardiovascular disease and Hypertension) study. This study aims to monitor and track young, healthy, normotensive adults with the prospect of identifying novel and early markers of early cardiovascular risk. The inclusion criteria were young (20 to 30 years), apparently healthy black and white men and women with office blood pressure <140/90 mm Hg and no known CVD. Further details regarding the inclusion criteria were described elsewhere [15].

We included cross-sectional data from 555 participants with complete 24-hour urine collections and successful ambulatory blood pressure monitoring (ABPM) (>70% valid readings). The participants were categorized as normotensive (n = 534) or masked hypertensive (n = 147). Normotensive status was defined as having a clinic BP < 140/90 mm Hg and normal ABPM (24-h readings <130/80 mm Hg, daytime readings <135/85 mm Hg and nighttime readings <120/70 mm Hg). Masked hypertension status was defined as participants having normal clinic BP < 140/90 mm Hg, but an increased ABPM (24-hour readings ≥130/80 mm Hg and/or daytime readings ≥135/85 mm Hg and/or nighttime readings ≥120/70 mm Hg) [17].

Informed consent was obtained from each participant and the study protocol conformed to the ethical guidelines of the Declaration of Helsinki as reflected in a priori approval by the Health Research Ethics Committee of the North-West University (NWU-00001-12-A1).

2.2. Data collection and procedures

2.2.1. Demographic and anthropometric measurements

Demographic data, such as sex, age and ethnicity were collected by means of a demographic and lifestyle questionnaire.

All anthropometric measurements were conducted in accordance to the International Standards for Anthropometric Assessment [18]. Weight (kg) was measured to the nearest 0.01 kg (SECA electronic scales, SECA, Birmingham, UK), height (m) to the nearest 0.1 cm (SECA stadiometer, SECA) and waist circumference (cm) was measured in triplicate using a non-flexible tape measure (Holtain, Crymych, UK), and recorded to the nearest 0.1 cm. Body mass index (BMI) [weight (kg) / height (m^2)] was also calculated.

2.2.2. Cardiovascular measurements

For the office brachial blood pressure measurements, the participants were seated and in a relaxed state while the blood pressure were taken in duplicate (on each arm) with a 5 minute rest period between them. The Dinamap® Procare blood pressure monitor (GE Medical Systems, Milwaukee, USA) with appropriate cuff sizes were used to determine blood pressure which were taken four times, twice on each arm. The mean of the four measurements were used as office blood pressure. Normotensive status was based on clinic blood pressure <140/90 mm Hg and normal (24-hour readings <130/80 mm Hg, daytime readings <135/85 mm Hg and nighttime readings <120/70 mm Hg). Masked hypertension status was classified as participants having normal clinic BP < 140/90 mm Hg, but an elevated ABPM (24-h readings ≥130/80 mm Hg and/or daytime readings ≥135/85 mm Hg and/or nighttime readings ≥120/70 mm Hg) [17]. The Card(X)plore devices (MediTech, Budapest, Hungary), were used to determine the 24-hour ABPM and were programmed to take recordings every 30 min during the day (06h00 to 22h00) and every hour at night (22h00 and 06h00).

A standard transthoracic echocardiogram was conducted for each participant [19]. The data was then analysed using the EchoPAC software (GE, version 10.8.1) to determine measures of left ventricular structure and function. Each participant was scanned while in a partial left decubitus position with the head of the examining table modestly elevated. Using 2D echocardiography recordings, we measured left ventricular dimensions according to the recommendations of the American Society of Echocardiography, by one specialist clinical technologist [19,20]. From these dimensions we reported the interventricular septum thickness at end-diastole (IVSd), the left ventricular posterior wall thickness at end-diastole (LVPWd) and the left ventricular internal diameter during end-diastole (LVIDd). The LVIDd was additionally indexed by body height in meter. Left ventricular mass was consecutively calculated by the corrected Devereux formula and was normalised for body surface area, hence LVMI [21]. The relative wall thickness (RWT) was defined as the ratio of twice the posterior wall thickness and the left ventricular diastole diameter.

2.2.3. Biochemical sampling and analysis

Fasting blood samples were collected, prepared and stored at −80 °C until analysed. We used the Cobas Integra® 400 plus (Roche, Basel, Switzerland) to analyse, glucose, lipids, creatinine and high-sensitivity C-reactive protein. The CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) equation was used to estimate glomerular filtrate rate (eGFR) [22].

2.2.4. 24-hour urine collections

Each participant collected a 24-hour urine sample according to the WHO [23]. Urine samples were aliquoted and placed in a −20 °C freezer until analyses. Urinary sodium and potassium were measured by means of ion-selective electrode potentiometry on the Cobas Intergra® 400 plus (Roche, Basel, Switzerland). Sodium and potassium excretion was calculated as set out in Swanepoel et al. [16].

2.3. Statistical analysis

Statistical analyses were performed using IBM® SPSS® version 25 (IBM, Armonk, NY, USA). Normality was tested using the Kolmogorov-Smirnov test and visual inspection of histograms. Parametric tests were applied when data were normally distributed and a logarithmic transformation was performed when data were not normally distributed. Parametric data were reported as the mean and standard deviation, where non-parametric data were presented by the geometric mean with 5th and 95th percentiles. Data are presented in the total group and by stratification according to normotensive and masked hypertensive status. Additionally, we stratified the demographic information of the population by ethnicity and added as a Supplementary Table. Comparisons between the two stratified groups (masked hypertension vs. normotensive) were assessed with independent *t*-tests for continuous variables and the Chi-square test for categorical variables. Unadjusted correlations were plotted using GraphPad Prism version 6.0 (GraphPad Software Inc., CA, USA). Partial correlations were performed to determine the relationships of LVMI, RWT and LVIDd with sodium excretion with adjustment for sex, age and ethnicity. Multiple regression analyses were performed to determine independent associations of LVMI and LVIDd with sodium excretion. Covariates considered for entry in the models included sex, age, ethnicity, waist circumference and 24-h systolic blood pressure as independent variables A p-value of <0.05 was considered statistically significant.

3. Results

Table 1 shows masked hypertensive individuals had higher sodium excretion (149 vs. 128 mmol/L/day) as well as a higher LVMI (78.1 vs. 69.6 g/m^2) than their normotensive counterparts. LVIDd (2.78 vs. 2.86 cm/m) as well as RWT (0.35 vs. 0.36 cm) was also observed to be higher in the masked hypertensive individuals. Both IVSd (0.78 vs. 0.84 cm) and LVPWd (0.81 vs. 0.91 cm) were higher in the masked hypertension group. Overall, 147 (21%) participants had masked hypertension, with the majority (67%) being men. White (54%) participants had a higher prevalence of masked hypertension than their counterparts. Individuals with masked hypertension had higher body weight, BMI and waist circumference (all $p < 0.001$) than the normotensive group. The Supplementary Table shows ethnic comparisons with no difference in LVMI (70.9 vs. 71.8 g/m^2), LVIDd (2.78 vs. 2.88 cm/m) as well as sodium excretion (136 vs. 129 mmol/L/day) between the black and white groups. RWT was higher in the black participants (0.37 vs. 0.34 cm) and 24-hour systolic blood pressure was higher in the white participants ($p = 0.005$).

Fig. 1 illustrates the unadjusted correlations of LVMI and LVIDd with sodium excretion in all three groups, respectively. In the total group ($r = 0.14$; $p < 0.001$) as well as the masked hypertensive group ($r = 0.26$; $p = 0.001$), a positive correlation was observed between LVMI and sodium excretion, but not in normotensives ($p = 0.53$). LVIDd positively correlated with sodium excretion in the masked hypertensive group ($r = 0.21$; $p = 0.011$) only. No correlation was evident between RWT and 24-h sodium excretion in either group (data not shown).

In partial regression analysis, with adjustments for age, sex and ethnicity (Table 2), the previous correlation between LVMI and 24-h sodium excretion remained in the total ($r = 0.11$; $p = 0.006$) and masked hypertensives ($r = 0.221$; $p = 0.008$). We found no significant correlation between RWT and 24-h sodium excretion in any of the groups. In addition, LVIDd correlated with sodium excretion in the masked hypertensive group ($r = 0.212$; $p = 0.011$) only.

Table 3 presents the multivariable-adjusted linear regression analysis. We confirmed a positive association between LVMI and sodium excretion in the total group ($\beta = 0.089$ [0.02; 0.16]; $p = 0.011$) as well as the masked hypertension group ($\beta = 0.215$ [0.06; 0.39]; $p = 0.008$). Male sex, but not ethnicity, also contributed to the independent association between LVMI and sodium excretion in the total and masked hypertension groups. LVIDd showed a positive association with

Table 1
Demographics of the total study population and stratified according to normotension and masked hypertension.

	Total (n = 681)	Normotensive (n = 534)	MHT (n = 147)	p value*
Age (years)	24.9 ± 3.03	24.8 ± 2.95	24.9 ± 3.04	0.286
Men, n (%)	278 (40.8)	179 (33.5)	99 (67.3)	<0.001
Black, n (%)	341 (50.1)	273 (51.1)	68 (46.3)	0.352
Socioeconomic status				
Low, n (%)	251 (36.9)	203 (38.0)	48 (32.7)	0.089
Middle, n (%)	166 (24.4)	131 (24.5)	35 (23.8)	
High, n (%)	260 (38.2)	199 (37.3)	61 (41.5)	
Anthropometric measures				
Weight (kg)	70.8 [69.3; 72.2]	67.5 [66.2; 68.9]	83.2 [79.5; 87.0]	<0.001
Height (cm)	168 [167; 169]	167 [166; 168]	172 [171; 174]	<0.001
Body mass index (kg/m^2)	24.9 [24.6; 25.1]	24.2 [23.7; 24.7]	27.8 [26.7; 29.1]	<0.001
Waist circumference (cm)	79.4 [78.3; 80.5]	77.1 [76.1; 78.1]	88.7 [86.1; 91.5]	<0.001
Systolic blood pressure				
Clinic (mm Hg)	117 ± 11.2	114 ± 9.72	128 ± 9.27	<0.001
24-hour (mm Hg)	117 ± 9.42	113 ± 6.87	129 ± 6.94	<0.001
Daytime (mm Hg)	121 ± 9.75	118 ± 7.51	133 ± 8.62	<0.001
Night time (mm Hg)	108 ± 10.5	104 ± 7.64	121 ± 8.06	<0.001
Diastolic blood pressure				
Clinic (mm Hg)	78 ± 6.95	76 ± 6.28	83 ± 6.95	<0.001
24-hour (mm Hg)	69 ± 5.56	67 ± 4.69	74 ± 5.26	<0.001
Daytime (mm Hg)	73 ± 6.10	72 ± 5.35	78 ± 6.68	<0.001
Night time (mm Hg)	60 ± 6.70	58 ± 5.36	66 ± 6.90	<0.001
Heart rate				
Clinic (beats/min)	64.9 ± 10.2	64.9 ± 10.2	64.9 ± 10.5	0.957
24-hour (beats/min)	75.1 ± 10.7	75.1 ± 10.5	75.3 ± 11.5	0.857
Daytime (beats/min)	79.4 ± 11.2	79.6 ± 10.9	78.9 ± 12.3	0.530
Night-time (beats/min)	66.9 ± 11.6	66.7 ± 11.0	67.7 ± 13.5	0.394
Left ventricular structure				
LV mass index (g/m^2)	71.4 ± 16.6	69.6 ± 15.6	78.1 ± 18.8	<0.001
Relative wall thickness (cm)	0.35 [0.35; 0.34]	0.35 [0.34; 0.36]	0.36 [0.35; 0.37]	0.014
LVIDd (cm/m)	2.80 ± 0.24	2.78 ± 0.23	2.86 ± 0.27	0.05
IVSd (cm)	0.79 [0.76; 0.81]	0.78 [0.75; 0.81]	0.84 [0.78; 0.95]	<0.001
LVPWd (cm)	0.84 [0.59; 1.19]	0.81 [0.79; 0.84]	0.91 [0.85; 0.95]	<0.001
Biochemical markers:				
Total cholesterol (mmol/L)	4.19 [4.11; 4.27]	4.12 [4.03; 4.21]	4.45 [4.27; 4.64]	0.014
HDL (mmol/L)	1.30 ± 0.38	1.33 ± 0.38	1.18 ± 0.36	<0.001
LDL (mmol/L)	2.84 ± 0.93	2.78 ± 0.91	3.02 ± 0.97	0.139
Triglycerides (mmol/L)	0.84 [0.62; 1.13]	0.79 [0.50; 1.22]	1.09 [0.98; 1.20]	<0.001
C-Reactive protein (mg/L)	1.07 [0.94; 1.18]	1.02 [0.89; 1.17]	1.17 [0.94; 1.46]	0.187
Glucose (mmol/L)	4.53 [4.39; 4.68]	4.45 [4.28; 4.64]	4.84 [4.70; 4.97]	0.001
eGFR (mL/min/1.73 m^2)	111 [110; 113]	112 [110; 113]	109 [106;112]	0.095
GGT (U/L)	20.5 [19.9; 21.9]	19.3 [18.3; 20.3]	28.3 [25.3; 31.8]	<0.001
Cotinine (ng/mL)	3.21 [2.82; 4.01]	3.22 [2.64; 3.91]	3.96 [2.65; 5.91]	0.237
Urinary excretion profile:				
Na^+ excretion (mmol/L/day)	132 [125; 139]	128 [121; 135]	149 [133; 167]	0.036
K^+ excretion (mmol/L/day)	41.4 [39.4; 43.5]	40.3 [38.1; 42.6]	45.8 [41.0; 51.2]	0.022
Na^+:K^+	3.19 [3.06; 3.33]	3.17 [3.02; 3.33]	3.26 [2.99; 3.55]	0.978

Data presented as mean ± standard deviation or geometric mean [5th and 95th percentile intervals] for logarithmically transformed variables, or number of participants and (percentages).
Abbreviations: eGFR, estimated glomerular filtrate rate; GGT, gamma-glutamyl transferase; HDL, high-density lipoprotein; IVSd, interventricular septum thickness at end-diastole; K, potassium; LDL, low-density lipoprotein; LVPWd, left ventricular posterior wall thickness at end-diastole; LV, left ventricular; LVIDd, left ventricular internal diameter at end-diastole; MHT, masked hypertension; Na, sodium; Na:K, sodium to potassium ratio.
* Indicated p-values for differences between normotensive and masked hypertensive individuals.

sodium excretion in the masked hypertensive group (β = 0.206 [0.01;0.36], p = 0.011) only.

4. Discussion

A robust positive association of both LVMI and LVIDd with higher 24-hour urinary sodium intake in the total group and more evidently in the masked hypertension group were observed in this population group. The results from our study are in line with similar results reported in a normotensive population of young adults (similar in age of the current study) and showed a positive relationship between sodium intake with LVMI [24]. The current study, however, found no relationship between potassium and LVMI (data not shown). Similar to our study, data from another study suggested that in prehypertensive and hypertensive patients, an increase in sodium intake was positively associated with increased left ventricular mass [25]. Those studies also emphasized that an unhealthy diet (characterized by high sodium intake), worsens the existing negative effects of high blood pressure on left ventricular mass [24,25]. A number of other cross-sectional studies also showed a positive correlation between 24-hour urinary sodium excretion and left ventricular mass [24,26–28]. In normotensive individuals it was reported that very little changes in cardiac structure as a result of excess sodium intake was observed, with the exception of slight modifications in diastolic parameters [25], however diastolic function was not assessed in this study. However, we reviewed LVIDd as a measure of LV dilation and confirmed a positive association with 24-hour urinary sodium excretion in the masked hypertensive group, suggesting potential early onset volume-loading hypertension in this group. Some studies also showed a reduction in salt intake contributes to a decrease left ventricular mass in hypertensive individuals [28–30]. It is important to note that the populations studied in these studies were comprised of black and white Americans as well as Europeans. Studies within

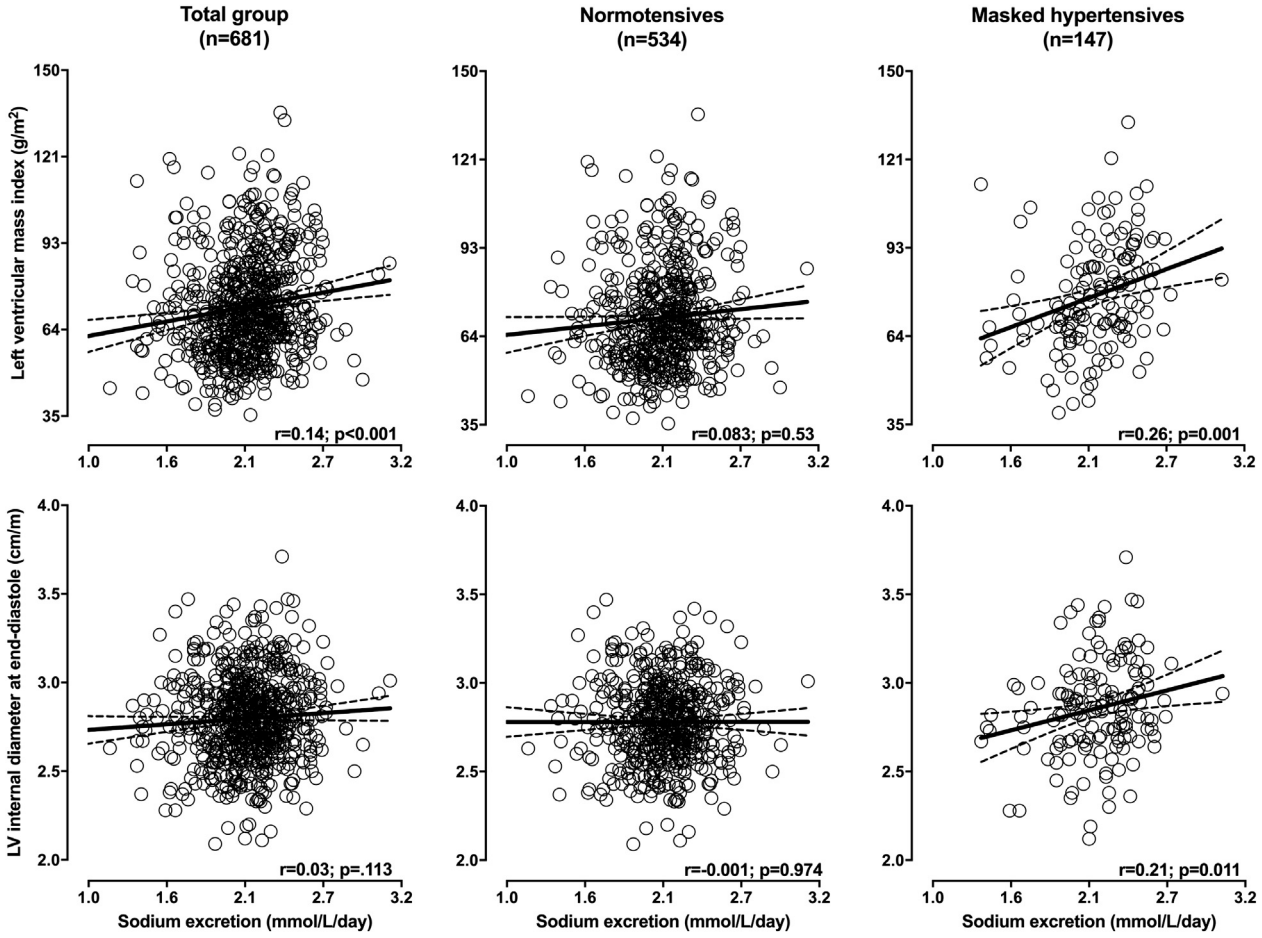

Fig. 1. Unadjusted correlation of LVMI and LVIDd with sodium excretion in the study population.

sub-Saharan Africa populations investigating these associations are scant. In addition, we did not observe any finding with RWT as a measure of cardiac wall remodeling, but we emphasize the potential early development of left ventricular hypertrophy among these young masked hypertensive adults [14]. The underlying mechanism regarding how sodium intake modulates myocardial structure was suggested to be a dysregulation of the renin-angiotensin-aldosterone system [31]. Some individuals may have an impaired down regulation of angiotensin II synthesis when challenged with high salt intake. Therefore, high levels of angiotensin II in relation to urinary sodium excretion were associated with left ventricular hypertrophy in individuals on a high salt intake diet [31]. With our positive association between LVIDd and 24-hour urinary sodium excretion, we may indicate early onset volume-loading consequences on the heart, especially in masked hypertensives.

In a group of apparently healthy young individuals, 21% were reported to present with masked hypertension, which subjects them to future risk for hypertension-related target organ damage and cardiovascular disease [32,33]. A review reported an overall prevalence of 8.5–16.6% in the general population [10]. These population groups were however older than the current study's adults, highlighting the high prevalence of undetected and early development of hypertension among young adults in a South African context. LVMI was higher (although still in normal ranges) in the masked hypertensives compared to the normotensive group, which is in line with previously reported findings [34,35]. There is a consensus that dietary sodium intake plays a significant role in the development of high blood pressure in the general populations as well as the severity of hypertension [1–3]. Additionally, subclinical target organ damage as a result of high sodium intake and subsequent elevations in blood pressure, may put this young adult population at higher risk for early cardiovascular morbidities.

Limitations of our study could include the use of a single 24-hour urine sample to determine sodium excretion. However, by including a

Table 2
Partial correlations of left ventricular structure with sodium excretion in the total population, normotensives and masked hypertensive individuals.

		LV mass index (g/m^2)	RWT (cm)	LVIDd (cm/m)
Na^+ excretion (mmol/L/day)	Total group	r = 0.105	r = 0.038	r = 0.061
	(n = 681)	p = 0.006	p = 0.322	p = 0.113
	Normotensive	r = 0.064	r = 0.054	r = −0.001
	(n = 534)	p = 0.142	p = 0.213	p = 0.974
	Masked hypertensive	r = 0.221	r = −0.018	r = 0.212
	(n = 147)	p = 0.008	p = 0.832	p = 0.011

Adjustments applied for sex, age, ethnicity.
Abbreviations: LVIDd, left ventricular internal diameter at end-diastole, LV mass index, left ventricular mass index; Na^+, sodium; RWT, relative wall thickness.

Table 3
Multiple regression analysis of LV mass index with sodium excretion in the normotensive and masked hypertensive individuals.

LV mass index (g/m^2)						
	Total group (n = 681)		Normotensive (n = 534)		Masked hypertensive (n = 147)	
R^2	0.23		0.21		0.20	
Adjusted R^2	0.22		0.20		0.16	
	Beta (95% CI)	p-Value	Beta (95% CI)	p-Value	Beta (95% CI)	p-Value
Na^+ excretion (mmol/L/day)	0.089 (0.02; 0.16)	0.011	0.054 (−0.02; 0.13)	0.13	0.215 (0.06; 0.39)	0.008
Sex (women/men)	0.413 (0.34; 0.49)	<0.001	0.416 (0.33; 0.50)	<0.001	0.343 (0.21; 0.57)	<0.001
Age (years)	0.023 (−0.05; 0.09)	0.51	0.016 (−0.06; 0.09)	0.39	0.067 (−0.10; 0.25)	0.41
Ethnicity (black/white)	−0.002 (−0.07; 0.09)	0.96	0.003 (−0.07; 0.08)	0.95	−0.015 (−0.20; 0.16)	0.86
Waist circumference (cm)	0.005 (−0.08; 0.09)	0.91	0.016 (−0.08; 0.11)	0.73	−0.045 (−0.23; 0.14)	0.65
24 h SBP (mm Hg)	0.082 (−0.01; 0.07)	0.067	0.068 (−0.03; 0.21)	0.16	0.056 (−0.17; 0.34)	0.52
LVIDd (cm/m)						
	Total group (n = 681)		Normotensive (n = 534)		Masked hypertensive (n = 147)	
R^2	0.06		0.05		0.16	
Adjusted R^2	0.06		0.04		0.08	
	Beta (95% CI)	p-Value	Beta (95% CI)	p-Value	Beta (95% CI)	p-Value
Na^+ excretion (mmol/L/day)	0.051 (−0.01; 0.03)	0.18	−0.005 (−0.02; 0.01)	0.90	0.206 (0.01; 0.36)	0.012
Sex (women/men)	0.022 (−0.02; 0.03)	0.60	0.014 (−0.02; 0.03)	0.77	0.005 (−0.05; 0.05)	0.95
Age (years)	0.119 (0.02; 0.20)	0.002	0.122 (0.01; 0.15)	0.005	0.119 (−0.01; 0.26)	0.15
Ethnicity (black/white)	0.053 (0.02; 0.06)	<0.001	0.002 (−0.02; 0.02)	0.96	0.195 (0.01; 0.29)	0.019
24 h SBP (mm Hg)	0.174 (−0.02; 0.23)	0.54	0.155 (0.02; 0.23)	0.001	0.066 (−0.04; 0.09)	0.43

Abbreviations: CI, confidence interval; LV, left ventricular, LVIDd, left ventricular internal diameter during end-diastole; Na, sodium; SBP, systolic blood pressure.

sufficient number of people, a single 24-hour urine collection should be adequate in estimating the mean sodium excretion for a population, with little error of the mean [36], and is still regarded as the gold standard for sodium excretion estimation, which is recommended by the WHO. Additionally, the cross-sectional design of the study makes the establishment of casualty difficult, but the positive association between LVMI and high sodium intake in this population still provides valuable information on the harmful role of excessive sodium consumption, above that of increased blood pressure. The African-PREDICT study is designed as longitudinal, which makes future studies on the reporting of the follow-up data possible. This will allow for further investigations on the progression of left ventricular mass increase and the role of sodium within this population.

In conclusion, we confirmed positive associations of left ventricular mass and size with 24-hour sodium excretion, especially in masked hypertensive adults. Our findings support global initiatives in reducing the general population's sodium intake to curb the increasing cardiovascular morbidity and mortality rates due to unhealthy lifestyle and diet.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2018.11.116.

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Disclaimers and conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

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