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Deleterious Effect of Westernized Diet Pattern, Red Meat and High Salt Intake on Left Ventricular Structure and Function in Hypertensive Black Sub-Saharan Africans: A Multicentric Cross Sectional Study

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Abstract

Diet is one of the mosaics of environmental factors that can affect heart health. This impact, positive or negative, could occur through left ventricular remodeling.

Aims: To study the correlation between different specific foods and dietary patterns as well as dietary salt intake with left ventricular structure and function in hypertensive black Sub-Saharan Africans.

Methods: 77 apparently healthy Congolese black hypertensive participants were evaluated. Dietary sodium intakes were calculated based on the amount of sodium excreted in 24-hour urine. Dietary habits were assessed using a Food Frequency Questionnaire (FFQ). Left ventricular structure and function were assessed by cardiac doppler ultrasound using Vivid T8 (GE) ultrasound.

Results: There was a significant and negative linear correlation between fruit consumption per week and left ventricular end-diastolic diameter (LVED) (r=0.27, p=0.017), left ventricular mass (LVM) (r=0.23, p=0.047), left ventricular mass indexed to height^{2,7} (LVMIh) (r=0.28, p=0.016), left ventricular mass indexed to body surface area (LVMIbsa) (r=0.28, p=0.015) and deceleration time (DT) (r=0.26, p=0.023); vegetable consumption per week and DT (r=0.33, p=0.004); and fish consumption and LVM (r=0.25, p=0.030). There was a significant and positive linear correlation between fruit consumption and ratio of peak early and late diastolic flow velocities (E/A) (r=0.28, p=0.013); meat consumption per week and Interventricular Septal Thickness (IVS) (r=0.27, p=0.016), sum of wall thickness (SWT) (r=0.26, p=0.013), LVM (r=0.32, p=0.005), LVMIh (r=0.33, p=0.003) and LVMIbsa (r=0.26, p=0.023); and fish consumption per week and LVMIh (r=0.36, p=0.001); NaCl g/24 h and IVS (r=0.29, p=0.012), PWT (r=0.33, p=0.003), SWT (r=0.33, p=0.004), LVM (r=0.34, p=0.002), LVMIh (r=0.32, p=0.005), LVMIbsa (r=0.36, p=0.001), RWT (r=0.25, p=0.031) and left atrial area (r=0.50, p<0.001). In a multiple linear regression analysis, fruit consumption, meat consumption and NaCl g/24 h were the independent determinants of LVM, LVMIh and LVMIbsa, explaining 53%, 51% and 52% of variability respectively.

Conclusions: Our results show a deleterious effect of westernized diet pattern, red meat consumption, and high salt intake on left ventricular structure and function in hypertensive black Sub-Saharan Africans. Our findings have implications for dietary choices with respect to consumption of specific food items. Our findings emphasize the importance of dietary measures for cardiovascular disease prevention. A larger-scale prospective study is needed to confirm these findings.

Keywords: Diet; Westernized dietary pattern; Mediterranean dietary pattern; Daily sodium chloride intake; Hypertension; Left ventricular mass; Left ventricular hypertrophy; Black; Sub-saharan African

Introduction

The associations between diet and morbidity and mortality have been widely reported in relation to diseases as diverse as cancer [1,2], sterility [3-5], dementia [6], cardiovascular disease [2,7], and more recently COVID-19 [8,9], to name a few. Unhealthy diet was responsible for an estimated 1 in 5 premature deaths globally from 1990 to 2016 [10]. Yet it seems that this association between diet and health has been known for millennia. Indeed, Hippocrates of Kos, Greek doctor of the century of Pericles (5th century BC), father of modern medicine and author of the oath that bears his name, recognized the central role of food in the prevention and treatment of disease. In the original version of this oath, the declaration "I will apply dietetic measures for the benefit of the sick according to my ability and judgment," precedes the declarations on drugs and surgery [11].

Cardiovascular Diseases (CVDs) constitute the leading cause of global mortality and are a major contributor to reduced quality of life [12]. Nearly 80% of global CVD deaths occur in Low and Middle-Income Countries (LMIC) where CVD and risk factor burden are on the rise as a result of an ongoing epidemiological transition [13]. Indeed, the days when infectious diseases predominated in these countries are over. Today the Cardiovascular diseases are supplanting tropical diseases. Significant dietary changes have been noted in Sub-Saharan Africa (SSA) [14-16] and have been considered by some authors as one of the causes of the surge in cardiovascular disease in this part of the old world [17,18]. Indeed, diet has a proven impact on cardiovascular disease. A healthy diet decreases the risk of coronary artery disease, in particular by improving the blood pressure, glycemic, lipid, oxidative and inflammatory profile. An unhealthy diet, on the other hand, would promote the onset of these diseases [19-24]. Almost all of studies on the impact of specific foods, nutrients, and drinks as well as dietary patterns on cardiovascular health [25-31] have been conducted in populations in developed countries. Aside from some recent data which give some fragmentary information on some populations in developing countries [32,33] data's are almost nonexistent for these countries. Furthermore, the ideal diet for specific patients, taking into account their risk factor profile, remains a matter of controversy [34,35].

In hypertensive patients, left ventricular hypertrophy (LVH) is a strong predictor of cardiovascular events including sudden death, congestive heart failure and stroke [36-38].

The purpose of this present study is to investigate the associations between daily sodium intakes, certain specific foods as well as different diet patterns (Mediterranean Diet Pattern (MDP), Westernized Diet Pattern (WDP) and Indeterminate Diet Pattern (IDP)) with left ventricular remodeling in Black Sub-Saharan African participants in Kinshasa, Democratic Republic of Congo (DRC).

Material and Methods

The present cross-sectional study involved 77 apparently healthy black Congolese hypertensive participants without a major morbid cardiovascular history. The participants were selected during outpatient consultations in the cardiology units of the University hospital of Kinshasa, the Centre Medical de Kinshasa (CMK), the General Reference Hospital of Kinshasa, the Ngaliema Clinic and the Lomo-Médical Clinic for 5 consecutive days from Tuesday, September 24, 2019, date of World Heart Day 2019.

Socio-demographic (i.e., age, sex), dietary, anthropometric, and echocardiographic data were collected for each participant. Socio-demographic and dietary data were collected by trained investigators, using a standardized questionnaire, in each of the 5 aforementioned recruiting hospitals. Anthropometric data and echocardiographic parameters were collected at the CMK during an individually negotiated appointment with each participant. The CMK is a private referral clinic working on international standards and norms. It has the best equipped cardiology unit in Central Africa, with highly qualified and regularly retrained staff.

Patient selection

All hypertensive patients aged 20 years and over who presented for an outpatient visit to the 5 aforementioned cardiology units during the study period were targeted to participate in this study. They were initially reviewed for clinical and/or paraclinical evidence of secondary hypertension, kidney and liver disease. Patients in whom no clinical or laboratory evidence of secondary hypertension, kidney or liver disease was found were selected for the study. The participants thus selected were invited, after free and informed consent, to participate in the present study. Patients with heart disease unrelated to arterial hypertension as well as those using diuretics were excluded.

Study procedures

Anamnestic data: The following information was gathered by a trained interviewer using an ad-hoc questionnaire. These are demographic data (age, sex), lifestyle data (high alcohol consumption, cigarette smoking, eating habits), medical history including cardiovascular risk factors (diabetes mellitus, dyslipidemia, hyperuricemia, menopause, duration of high blood pressure), a history of cardiovascular events (heart failure, ischemic heart disease, cardiovascular surgery, stroke, chronic kidney disease) and chronic use of certain drugs, in particular antihypertensive drugs, anti-diabetics, statins, anti-platelet drugs, hypouricemic drugs and combined contraceptives). Sedentary behavior was assessed using the World Health Organization's Global Physical Activity Questionnaire (WHO/GPAQ) [39]. Dietary behaviors were evaluated using a semi-quantitative Food Frequency Questionnaire (FFQ) [40]. The participants were interviewed on their average weekly consumption frequency of fruits, vegetables, legumes (soybeans, beans, peas, peanuts), cereals (rice, wheat, corn, sorghum), nuts, fish, dairy products and red meat.

Anthropometric data: The following anthropometric measurements were measured by a trained observer in accordance with WHO recommendations: body weight, height, and waist and hip circumference. Body mass index (BMI) was calculated as a derived variable as follows:

$$BMI = \text{Body weight} / \text{height}^2$$

The body surface area (BSA) was calculated using the DuBois formula [14] as follows:

$$BSA = \text{Height (cm)}^{0.725} \times \text{Weight (kg)}^{0.425} \times 0.007184$$

BMI was obtained by dividing the weight (kg) by the height (m) squared.

Blood pressure: Blood pressure was measured automatically and non-invasively by 24-hour Ambulatory Blood Pressure Monitoring (ABPM) using a TONOPORT V type recorder (GE Health care, Freiburg, GERMANY).

Echocardiographic data: A comprehensive echocardiographic assessment of left ventricular structure and function was performed for each participant as per the updated recommendations from the American Society of Echocardiography and the European Association

of Cardiovascular Imaging [41], using a Vivid T8 type ultrasound machine (GE Health Care, Freiburg, ALLEMAGNE) equipped with a 3.5 MHz probe.

The left ventricular dimensions (Interventricular Septum (IVS), End-Diastolic Diameter (EDD) and Posterior Wall Thickness (PWT)) were measured from a long-axis parasternal section on a two-dimensional image frozen in end-diastole, at the end of the mitral leaflets. The end of the diastole was detected by a simultaneous ECG. The values of the aforementioned dimensions were used for the calculation of the LVM according to the formula, as follows: LVM (g)=0.8 × 1.04 × [(LVED + IVS + PWT)^3 - (LVED)^3] + 0.6 g.

The LVM was then indexed to the body surface area expressed in square meters and to the height expressed in meters and raised to the power of 2.7. The relative wall thickness (RWT) was calculated by the quotient of double the PWT by LVED((2 × PWT) / LVED). Mitral flow analysis was performed in a four-chamber apical section using pulsed doppler, placing the measurement volume at the end of the mitral leaflets. The E waves (maximum speed of the E wave) and A (maximum speed of the A wave) as well as the deceleration time of the E wave (TDE) were thus recorded. The average of five consecutive measurements of various aforementioned parameters was recorded. Pulsed tissue doppler echocardiography recorded mitral annular velocities from the apical window. The sample volume was placed on the lateral side of the mitral ring. The area of the left atrium was measured planimetrically on an apical four-cavity section.

Laboratory measurements: Each participant received a suitable plastic container for urine collection, type V-Monovette® (Nümbrecht, Germany) with clear instructions on handling the container and how to collect 24-hour urine. The container and its contents (24 hour urine), were returned to the CMK medical laboratory, where the contents were mixed and the total volume recorded. From each container a quantity of 2 ml of urine was taken and stored at -20°C. Urinary sodium was assessed using Roche Hitachi (Indianapolis, IN, USA), an indirect ion-selective electrode to determine ion concentration.

The daily sodium chloride intake (NaCl g/24 h) was calculated from the daily sodium excretion using the formula [42]: NaCl (g/24h)=Na (mmol/24h). 58.4/1000.

This is consistent with the fact that 1 g of NaCl=17.1 mEq or 17.1 mmol of Na^+.

Operational definitions

Lifestyle data: Cigarette smoking was defined as having smoked daily for at least 30 days prior to the date of the interview, regardless of the type or number of cigarettes smoked [43].

Binge drinking was defined as consuming at least 2 glasses of beer or its equivalent daily for at least 12 months prior to the date of the interview [44].

Moderate consumption of red wine was defined as a daily consumption of red wine not exceeding 1 large glass (250 ml) of red wine [45].

Sedentary lifestyle has been defined as the habit of remaining seated, continuously or intermittently, for more than 7 hours a day [46].

A high consumption of a specific food was defined as an average of 4 to 7 days of weekly consumption of the considered food.

A low consumption of a specific food was defined as an average of fewer than 4 days of weekly consumption of the considered food.

Excessive sodium intake was defined as a daily intake of salt greater than or equal to 5 g regardless of the mode of ingestion [47].

The Mediterranean Diet Pattern (MDP) has been defined as comprising a high intake of at least four of the following: vegetables, legumes (including soybeans, beans, peas, peanuts), grains (including corn, wheat, rice, sorghum), tree nuts, fruits, fish; moderate consumption of red wine and small amounts of red meat and dairy products [48].

The Westernized Diet Pattern (WDP) has been defined as comprising a high intake of red meat and dairy products and a low intake of at least four of the following: vegetables, legumes, grains, fruits, nuts, fish [49].

The "Intermediate Diet Pattern" (IDP) was defined as a style of eating that did not meet the criteria of the two previous diet patterns.

Anthropometric parameters: Overweight was defined as BMI greater than 25 kg/m^2 of body surface area and less than 29.9 kg/m^2 body surface area [49].

Overall obesity was defined as a BMI equal to or greater than 30 kg/m^2 of body surface area [50]. Abdominal obesity was been defined as a waist circumference of >102 cm and >88 cm for men and women respectively [50].

Bioclinical data: Poor blood pressure control was defined as a mean nycthemeral systolic blood pressure greater than 130 mmHg and/or a mean diastolic nycthemeral blood pressure greater than 80 mmHg [51].

Echographic data: Normal LVM was defined as ≤ 115 g/m^2 or ≤ 48 g/m$^{2.7}$ for males and ≤ 95 g/m^2 or ≤ 44 g/m$^{2.7}$ for females; LVH was defined as LVM exceeding these values in males and female patients respectively. Three patterns of diastolic dysfunction (DD) were defined as follows [52,53]: abnormal relaxation (grade I of DD: E/A ratio <1 and prolonged deceleration time), pseudonormal relaxation (grade II: E/A ratio >1 and intermediate values of deceleration time), and restrictive patterns (reversible and irreversible, grade III-IV respectively; E/A ratio >2 and shortened deceleration time). The dilation of the Left Atrium (LA) was defined as a LA >20 cm^2 [54].

Statistical analysis

Data are presented as number (n) and relative frequencies (%) for categorical variables and average (± standard deviation) for quantitative variables. Student's t-test was performed to compare two means. The ANOVA test was used for multiple comparisons. The ANOVA tests which proved significant at the threshold of $p<0.05$ were supplemented by a post hoc Cheffée test. The influence of NaCl g/24 h and specific foods consumption on the left ventricular and diastolic parameters was investigated by linear regression in simple exploratory analysis respectively.

Correlation coefficients (r) were calculated to determine the degree of association between left ventricular and diastolic parameters, and salt and individual foods consumption. When differences were observed between left ventricular parameters and NaCl g/24 h and specific foods consumption, the effect of potential confounders including age, blood pressure, waist circumference, BMI and sedentary time was studied by adjustment in multiple linear regressions. The determination coefficients (R^2), were calculated finally to determine the degree of association between left ventricular parameters and the NaCl g/24 h and specific foods consumption. The significance threshold was $p<0.05$. Statistical analyzes were performed using XLSTAT 2020 and SPSS (Statistic Package for Social Sciences) for Windows version 24

software.

Ethical Considerations

This research was conducted in strict compliance with the recommendations of the Helsinki Declaration III. Approval to conduct the study was obtained from the ethics committee of the University of Kinshasa Public Health School prior to its commencement. All participants provided informed consent.

Results

Table 1 summarizes the general characteristics of the 77 hypertensive participants included in the final data base. The mean age of the study population was 48.7 ± 10.4 years with a male frequency of 52% (sex ratio 1M / 1F). The average weekly frequency of consumption of fruits, vegetables, red meat, fish and NaCl g/24 h was similar in both genders ($p>0.05$). In addition, we noted that the mean values of LVED, IVS, PWT, SWT and LVM were significantly higher in men than in women ($p<0.05$).

Table 2 summarizes the left ventricular parameters according to patients diet and shows that the patients on Western diet had significantly high average values of LVED, IVS, PWT, SWT, LVM, DT ($p<0.05$), on the other hand they had significantly lower values of E/A ($p=0.008$). We also note that patients on western diet had a significantly high frequency of diastolic dysfunction ($p=0.007$) and of LVH ($p=0.003$).

Table 3 shows that the mean values of the LV parameters were not different between patients with normal salt intake and those with excessive salt intake ($p>0.05$).

In table 4, shows a significant and negative linear correlation between:

- Fruit consumption per week and LVED ($r=0.27$, $p=0.017$), LVM ($r=0.23$, $p=0.047$), LVMIh ($r=0.28$, $p=0.016$), LVMIbsa ($r=0.28$, $p=0.015$), DT ($r=0.26$, $p=0.023$).
- Vegetable consumption per week and DT ($r=0.33$, $p=0.004$).
- Fish consumption and LVM ($r=0.25$, $p=0.030$).

And a significant and positive linear correlation between:

- Fruit consumption and E/A ($r=0.28$, $p=0.013$)
- Meat consumption per week and IVS ($r=0.27$, $p=0.016$), SWT ($r=0.26$, $p=0.013$), LVM ($r=0.32$, $p=0.005$), LVMIh ($r=0.33$, $p=0.003$), LVMIbsa ($r=0.26$, $p=0.023$)
- Fish consumption per week and LVMIh ($r=0.36$, $p=0.001$)
- Salt consumption per week and IVS ($r=0.29$, $p=0.012$), PWT ($r=0.33$, $p=0.003$), SWT ($r=0.33$, $p=0.004$), LVM ($r=0.34$, $p=0.002$), LVMIh ($r=0.32$, $p=0.005$), LVMIbsa ($r=0.36$, $p=0.001$), RWT ($r=0.25$, $p=0.031$), LAA ($r=0.50$, $p<0.001$).

In a multiple linear regression analysis (Table 5) fruits consumption, meat consumption and NaCl g/24 h emerged as independent determinants of LVM, LVMIh and LVMIbsa, explaining 53%, 51% and 52% of variability respectively.

Discussion

The present research work aimed to study the correlations between salt and specific foods consumption, as well as different dietary patterns with the left ventricular structure and function in a series of hypertensive black Sub-Saharan Africans. The study took place in Kinshasa, a crossroads of hundreds of ethnicities and cultures existing in the Democratic Republic of Congo, a country of varied foods and food habits. All the components of the studied diets (Mediterranean and Western) are widely available there, being produced locally or imported from other countries.

The present study highlighted significant favorable associations between fruit consumption, vegetable consumption and fish consumption and left ventricular structure and function parameters. The results suggest a deleterious effect of red meat, WDP and NaCl g/24 h on the left ventricular structure and function parameters. Fruit consumption, red meat consumption and salt emerged as food independent determinants of the LVM, explaining more than 50% of its variability.

Participants on a Mediterranean diet, compared to those on a westernized diet and on intermediate diet, had a significantly lower LVM and associated measurements (IVS, PWT, LVED), with a higher E/A and a shorter DT, suggesting a better left ventricular geometry and diastolic function.

These results agree with most previous studies, despite the great diversity of populations studied, methodologies, and operational definitions of the Mediterranean diet. Indeed, the concept of the Mediterranean diet deserves to be clarified from its historical, dietetic meaning and its operational definition. The Mediterranean diet recalls the traditional diet that prevailed in the olive growing areas of the Mediterranean basin before the mid-1960s [55]. From a dietetic point of view, it is a diet characterized by a relatively high vegetable fat intake (essentially from olive oil), which makes it palatable, low intake of saturated fat and is rich in dietary fiber. It is a diet rich in antioxidant compounds and bioactive elements with anti-inflammatory effects, and it has a low glycemic index [56]. From the point of view of the constitution of this diet, it is remarkable to note the great diversity of operational definitions reserved for it in the literature [57]. Galbete C, et al., through a recent general review of 27 meta-analyses, listed over 30 definitions [58]. The two indices that were the most used are the one of Trichopoulou A, et al., [48] and the one of Fung TT, et al., [59]. It has recently been recommended to use one of these two definitions in order not to compromise future comparability between studies [58]. The operational definition of the MDP used in this study is adapted from that of Trichopoulou A, et al., which seems closer to the traditional Mediterranean diet.

The Mediterranean diet has been shown to be beneficial on left ventricular remodeling. Gardener H, et al., in a multi-ethnic population-based Northern Manhattan Study, found that greater adherence to a Mediterranean diet is associated with decreased LVM [60]. The Multi-Ethnic Study of Atherosclerosis (MESA) and the Northern Manhattan study showed that adherence to the Mediterranean diet improved left ventricular remodeling [60,61].

The richness of this diet in antioxidant and anti-inflammatory substances, as well as its low glycemic index, provide the explanation for the favorable left ventricular profile associated with this diet. Indeed, oxidative stress, inflammation and insulin resistance are three common pathophysiological abnormalities in hypertensive patients [62-64], and play an important role in left ventricular remodeling [65]. The Mediterranean diet may, therefore, help to counter such pathophysiological anomalies.

Patients on the westernized diet had significantly greater LVM (crude and indexed to the body surface or to height) and associated measurements (IVS, PWT, LVED) compared to those of patients on the MDP and IDP. They also more often had diastolic dysfunction, with lower E/A and longer DT. These results agree with previous

Table 1: General characteristics of the study population according to sex.

Variables	All n=77	Male n=40	Female n=37	p-value
Age (years)	48.7 ± 10.4	50.2 ± 9.7	47.1 ± 10.9	0.189
Age groups (years)				0.139
≤ 40	21 (27.3)	7 (17.5)	14 (37.8)	
41-50	27 (35.1)	16 (40.0)	11 (29.7)	
>50	29 (37.7)	17 (42.5)	12 (32.4)	
BMI (kg/m²)	30.5 ± 4.7	30.7 ± 4.6	30.3 ± 4.7	0.672
WC (Cm)	102.9 ± 12.1	107.3 ± 13.6	98.7 ± 8.7	**0.004**
ST (hour/day)	9.4 ± 2.4	9.1 ± 2.2	9.8 ± 2.6	0.217
Insulin (pmol/l)	95.8 ± 48.5	93.2 ± 48.8	98.5 ± 48.7	0.634
HOMAIR	1.9 ± 0.9	1.8 ± 0.9	2.0 ± 1.0	0.439
Glycemia (mmol/L)	5.5 ± 1.6	5.7 ± 1.6	5.2 ± 1.6	0.187
HbA1C (%)	5.8 ± 0.9	6.1 ± 0.8	5.6 ± 1.0	**0.021**
Diet (days per week)				
Fruits	3.0 ± 1.8	3.0 ± 1.8	4.0 ± 1.8	0.163
Vegetables	5.0 ± 1.5	5.0 ± 1.5	5.0 ± 1.6	0.694
Legumes	4.0 ± 1.4	4.0 ± 1.2	4.0 ± 1.8	0.652
Cereals	5.0 ± 1.2	5.0 ± 1.4	5.0 ± 1.6	0.451
Nuts	2.0 ± 1.7	2.0 ± 1.5	2.0 ± 1.8	0.155
Fish	4.0 ± 1.5	4.0 ± 1.6	4.0 ± 1.4	0.250
Red meat	3.0 ± 1.4	4.0 ± 1.4	3.0 ± 1.5	0.311
Dairy products	3.0 ± 1.1	3.0 ± 1.2	4.0 ± 1.6	0.126
NaCl g/24 h	12.0 ± 8.3	12.9 ± 8.0	11.1 ± 8.6	0.329
Insulin resistance	18 (23.4)	8 (20.0)	10 (27.0)	0.592
Diet patterns				0.327
MDP	37 (48.1)	16 (40.0)	21 (56.8)	
WDP	21 (27.3)	13 (32.5)	8 (21.6)	
IDP	19 (24.7)	11 (27.5)	8 (21.6)	
Salt intake				0.328
Normal	21 (27.3)	9 (22.5)	12 (32.4)	
Excessive	56 (72.7)	31 (77.5)	25 (67.6)	
Echocardiographic parameters				
LVED (mm)	44.4 ± 4.3	45.4 ± 3.7	43.3 ± 4.6	**0.029**
IVS (mm)	11.5 ± 1.8	12.0 ± 1.7	10.9 ± 1.8	**0.007**
PWT (mm)	11.5 ± 1.7	11.9 ± 1.4	10.9 ± 1.9	**0.017**
SWT (mm)	22.9 ± 3.3	23.9 ± 2.9	21.9 ± 3.5	**0.007**
LVEF (%)	64.4 ± 5.5	64.1 ± 4.5	63.6.0	**0.125**
LVM (g)	184.6 ± 44.3	197.9 ± 37.5	169.8 ± 46.9	**0.005**
LVMIh (g/m$^{2.7}$)	45.8 ± 10.7	46.1 ± 8.3	45.4 ± 13.1	0.772
LVMIbsa (g/m²)	92.9 ± 20.2	98.3 ± 16.9	86.9 ± 22.0	**0.013**
RWT	0.53 ± 0.09	0.54 ± 0.09	0.51 ± 0.10	0.176
E/A	0.85 ± 0.33	0.80 ± 0.27	0.91 ± 0.38	0.173
DT (ms)	209.1 ± 42.0	215.8 ± 41.9	201.9 ± 41.3	0.146
LAA (cm²)	16.5 ± 4.2	17.2 ± 3.0	15.8 ± 5.1	0.171
DD	55 (71.4)	31 (77.5)	24 (64.9)	0.165
LVH	35 (45.5)	17 (42.5)	18 (48.6)	0.377
LV geometry				0.291
Normal	9 (11.7)	3 (7.5)	6 (16.2)	
Concentric remodeling	33 (42.9)	20 (50.0)	13 (35.1)	
Concentric LVH	35 (45.5)	17 (42.5)	18 (48.6)	

BMI=Body Mass Index; WC=Waist Circumference; ST=Sedentary Time; HOMAIR=Homeostatic Model Assessment for Insulin Resistance; HbA1C=glycated hemoglobin; NaCl g/24 h=Estimated daily sodium chloride intake; MDP=Mediterranean Diet Pattern; WDP=Westernized Diet Pattern; IDP=Intermediate Diet Pattern; LVED=Left Ventricular End-Diastolic Diameter; IVS=Interventricular Septal Thickness; PWT=Posterior Wall Thickness; SWT=Sum of Wall Thickness; LVEF=Left ventricular ejection fraction; LVM=Left Ventricular Mass; LVMIh=Left Ventricular Mass Indexed to Height^{2,7}; LVMbs=Left Ventricular Mass Indexed to Body Surface Area; RWT=Relative Wall Thickness; E=Mitral E Wave; E/A=ratio of peak early and late diastolic flow velocities; DT=Deceleration Time; LAA=Left Atrium Area; DD=Diastolic Dysfunction; LVH=Left Ventricular Hypertrophy; LV=Left Ventricle

Table 2: Left ventricular parameters according to diet pattern.

Variables	MDP n=37	WDP n=21	IDP n=19	p-value
LVED (mm)	43.0 ± 4.1	46.5 ± 4.3	43.2 ± 4.0	**0.021**
IVS (mm)	10.9 ± 1.9	12.4 ± 1.8	11.8 ± 1.0	**0.006**
PWT (mm)	10.9 ± 1.8	12.1 ± 1.5	11.8 ± 1.1	**0.012**
SWT (mm)	21.7 ± 3.7	24.5 ± 2.9	23.6 ± 1.9	**0.005**
LVM (g)	169.9 ± 46.5	213.5 ± 34.6	180.5 ± 32.1	**0.001**
LVMIh (g/m$^{2.7}$)	42.5 ± 12.2	50.8 ± 8.1	46.6 ± 8.3	**0.016**
LVMIbsa (g/m²)	86.4 ± 21.9	105.0 ± 16.9	92.2 ± 14.0	**0.003**
RWT	0.50 ± 0.09	0.55 ± 0.11	0.56 ± 0.07	0.079
E/A	0.97 ± 0.36	0.71 ± 0.22	0.79 ± 0.30	**0.008**
DT (ms)	194.9 ± 36.4	233.0 ± 39.9	210.4 ± 43.9	**0.003**
LAA (cm²)	16.9 ± 5.2	17.0 ± 3.1	15.3 ± 2.6	0.356
DD	22 (59.5)	20 (95.2)	13 (68.4)	**0.007**
LVH	11 (29.7)	16 (76.2)	8 (42.1)	**0.003**
LV geometry				**0.002**
Normal	8 (21.6)	1 (4.8)	0 (0.0)	
Concentric Remodeling	18 (48.6)	4 (19.0)	11 (57.9)	
Concentric LVH	11 (29.7)	16 (76.2)	8 (42.1)	

MDP=Mediterranean Diet Pattern; WDP=Westernized Diet Pattern; IDP=Intermediate Diet Pattern; LVED=Left Ventricular End-Diastolic Diameter; IVS=Interventricular Septal Thickness; PWT=Posterior Wall Thickness; SWT=Sum of Wall Thickness; LVM=Left Ventricular Mass; LVMIh=Left Ventricular Mass Indexed to Height^{2,7}; LVMbs=Left Ventricular Mass Indexed to Body Surface Area; RWT=Relative Wall Thickness; E/A=ratio of peak early and late diastolic flow velocities; DT=Deceleration Time; LAA=Left Atrium Area; DD=Diastolic Dysfunction; LVH=Left Ventricular Hypertrophy; LV=Left Ventricle

studies that have shown a significant association between the WDP and left ventricular structure and function abnormalities [66,67]. Pathophysiological disturbances related to WDP include increased production of reactive oxygen species and oxidative stress [68-70], the development of hyperinsulinemia and insulin resistance [68,69,71], low-grade inflammation [72,73] sympathetic nervous system [74] and renin-angiotensin-aldosterone system hyperstimulation [75]. All of these mechanisms are thought to be involved in the development of cardiovascular disease in general and LVH and diastolic dysfunction in particular [76-82].

Regarding salt consumption, there was no significant difference in left ventricular structural and functional parameters between patients with excessive salt intake and those without. However, NaCl g/24 h is significantly positively correlated with LVM and associated measurements (IVS, PWT, LVED), as well as with LAA. In addition, NaCl g/24 h has emerged as an independent determinant of LVM. This finding from our study is in accordance with studies that showed that high salt intake plays a role in the development of LVH independently

Table 3: Left ventricular parameters according to salt consumption.

Variables	Normal salt intake n=21	Excessive salt intake n=56	p-value
LVED (mm)	44.0 ± 3.3	44.5 ± 4.6	0.614
IVS (mm)	11.1 ± 1.9	11.6 ± 1.7	0.287
PWT (mm)	11.1 ± 1.9	11.6 ± 1.6	0.130
SWT (mm)	22.1 ± 3.6	23.3 ± 3.2	0.176
LVM (g)	174.2 ± 47.4	188.6 ± 42.8	0.208
LVMIh (g/m$^{2.7}$)	43.2 ± 10.7	46.8 ± 10.6	0.187
LVMIbsa (g/m^2)	88.4 ± 22.4	94.5 ± 19.3	0.242
RWT	0.50 ± 0.08	0.54 ± 0.10	0.069
E/A	0.82 ± 0.38	0.86 ± 0.31	0.650
DT (ms)	205.3 ± 32.9	210.6 ± 45.1	0.627
LAA (cm^2)	15.6 ± 2.6	16.9 ± 4.6	0.250
DD	17 (81.0)	38 (67.9)	0.200
LVH	9 (42.9)	26 (46.4)	0.492
LV geometry			0.483
Normal	4 (19.0)	5 (8.9)	
Concentric remodeling	8 (38.1)	25 (44.6)	
Concentric LVH	9 (42.9)	26 (46.4)	

LVED=Left Ventricular End-Diastolic Diameter; IVS=Interventricular Septal Thickness; PWT=Posterior Wall Thickness; SWT=Sum of Wall Thickness; LVM=Left Ventricular Mass; LVMIh=Left Ventricular Mass Indexed to Height$^{2.7}$; LVMbs=Left Ventricula Mass Indexed to Body Surface Area; RWT=Relative Wall Thickness; E/A=ratio of peak early and late diastolic flow velocities; DT=Deceleration Time; LAA=Left Atrium Area; DD=Diastolic Dysfunction; DD=Diastolic Dysfunction; LVH=Left Ventricular Hypertrophy; LV=Left Ventricle

or in addition to its effect on BP in human [83-86] and in animals [87]. Likewise, reduction of sodium intake showed positive results in terms of decreasing LVM [88,89]. There are some experimental studies that shed light on our understanding of the pathophysiological mechanisms explaining this relationship; for example, activation of local RAS leading to both myocardial hypertrophy and fibrosis [90,91] has been reported extensively in the literature. Chang RL, et al., have recently demonstrated the crucial role of IGF-IIRα (insulin-like growth factor-II receptor alternative spliced transcript) in enhancing cardiac hypertrophy under high-salt conditions [92] and Lang H, et al., recently demonstrated a role for the Uncoupling Protein 3 (UCP3) in this process too [93]. Old theories evoking the stimulation of the sympathetic system, hemodynamic changes (pressure and volume overload), enhanced protein synthesis and increased myocardial sodium influx have not been confirmed by experimental studies [94]; however, a large community-based experiment did find that urinary sodium excretion was not related to LVM [95]. In this study, urinary sodium excretion was measured from spot urine samples. Our study used analysis of sodium excretion in 24-hour urine, which is the gold standard for measuring salt intake [96]. The rationale for using 24-hour urine as a means of assessing daily sodium intake is that the kidney regulates the body's sodium balance, which implies that the sodium that is eliminated in the urine is equivalent to food ingestants (minus a negligible amount that is eliminated in the stool or sweat, about 7% of dietary sodium). This remains true in patients undergoing long-term treatment with a diuretic, if the dose of diuretic has not been changed recently [96]. Even if spot urine samples are considered an appropriate alternative for monitoring sodium intake, they are affected by the chronobiology of sodium excretion [82], and this methodological difference could explain the discordance with our findings. The differences in the ethnic makeup of the surveyed patients could also account for these discrepancies, given observed interethnic differences in salt sensitivity [97,98].

The present study has shown a significant negative correlation of fruit and fish consumption with LVM. In addition, a significant negative correlation has been found between fruit and vegetable consumption and DT. Fruit consumption was also positively correlated with E/A. Almost no studies have been conducted on the link between the consumption of fruits, vegetables, and fish as single foods and LVH. This is due to current recommendations that encourage dietary or food group studies rather than singular food studies, suggesting that analysis of the food model evaluating the overall quality of food is easier to translate into dietary recommendations than recommendations for single foods or nutrients [40,99]. This approach is reasonable but perhaps questionable, as it is clearly important to acknowledge unique aspects of foods and food components that may affect disease risk independent of the overall diet [100]. Focusing on isolated nutrients certainly cannot account for all interactions and may result in erroneous conclusions [101], but research focusing on single foods items could improve mechanistic understanding of the effects of diet patterns [100]. In a previous biracial population based study, we found that reduced fruit consumption was independently and significantly associated with echocardiographic LVH [102]. Studies have shown an inverse association between fruit consumption and the risk of cardiovascular events [25,103]. The cardioprotective mechanisms of fruits remain unclear, but their exceptional antioxidant and free radical scavenging properties are considered to be important in conferring protection [104]. These same properties could be the underlying mechanism of this inverse relationship between fruit consumption and LVH as it has been shown that oxidative stress plays a role in the genesis of LVH [65,105].

In this study, significant consumption of meat was found to be significantly positively correlated with LVM and associated measurements (IVS, PWT, LVED). A study performed by Haring B, et al., is one of few (if not the only example) that examined the relationship between the consumption of red meat as an individual food and the LVM. This study found no correlation between processed and unprocessed red meat consumption and LVM in American Indians [106]. This contradiction with the findings of our study could possibly be explained by race-related genetic factors that could confound the association between meat consumption and LVM. Another explanation for this contradiction might come from the fact that dietary intake was determined using FFQs, and some participants may not have adequately recalled dietary information on specific foods (recall bias). This bias may have reduced the observed associations, potentially causing an underestimation of true associations.

Study Strengths and Limitations

This study has strengths and limitations that should be noted. To our knowledge, this is the first study that has evaluated the implications of diet on left ventricular hypertrophy in hypertensive black Sub-Saharan Africans. The multicentric design and standardized echocardiographic measurements performed are also particular strengths of our study. Although echocardiographic measurements can be prone to errors due to signal noise, acoustic artifacts, and angle dependency, in the

Table 4: Correlation between ultrasound parameters and diet.

Variables	Fruit consumption r (p-value)	Vegetable consumption r (p-value)	Fish consumption r (p-value)	Meat consumption r (p-value)	Sal intake r (p-value)
LVED (mm)	-0.27 (**0.017**)	-0.08 (0.498)	-0.19 (0.105)	0.21 (0.064)	0.18 (0.114)
IVS (mm)	-0.11 (0.335)	-0.07 (0.550)	-0.24 (**0.033**)*	**0.27** (**0.016**)*	**0.29** (**0.012**)*
PWT (mm)	-0.17 (0.152)	0.04 (0.726)	-0.15 (0.189)	0.22 (0.060)	**0.33 (0.003)**
SWT (mm)	-0.15 (0.210)	-0.02 (0.882)	-0.21 (0.067)	**0.26 (0.023)**	**0.33 (0.004)***
LVM (g)	**-0.23 (0.047)**	-0.05 (0.653)	**-0.25 (0.030)**	**0.32 (0.005)**	**0.34 (0.002)***
LVMIh (g/m$^{2.7}$)	**-0.28 (0.016)**	-0.07 (0.538)	**0.36 (0.001)**	**0.33 (0.003)**	**0.32 (0.005)**
LVMIbsa (g/m^2)	**-0.28 (0.015)**	-0.06 (0.585)	-0.22 (0.050)	**0.26 (0.023)**	**0.36 (0.001)**
RWT	-0.05 (0.698)	0.03 (0.769)	-0.07 (0.521)	0.13 (0.265)	**0.25 (0.031)**
E/A	**0.28 (0.013)**	0.20 (0.085)	0.18 (0.118)	-0.15 (0.204)	0.02 (0.868)
DT (ms)	**-0.26 (0.023)***	**-0.33 (0.004)**	-0.20 (0.075)	0.17 (0.135)	0.17 (0.142)
LAA (cm^2)	-0.10(0.382)	0.07(0.521)	-0.04 (0.758)	0.02 (0.851)	**0.50 (<0.001)**

LVED=Left Ventricular End-Diastolic Diameter; IVS=Interventricular Septal Thickness; PWT=Posterior Wall Thickness; SWT=Sum of Wall Thickness; LVM=Left Ventricular Mass; LVMIh=Left Ventricular Mass Indexed to Height^{2,7}; LVMbs=Left Ventricular Mass Indexed to Body Surface Area; RWT=Relative Wall Thickness; E/A=ratio of peak early and late diastolic flow velocities; DT=Deceleration Time; LAA=Left Atrium Area

Table 5: Dietary determinants of left ventricular mass.

Variables	LVM			LVMIh			LVMIbsa		
	β	ES	P	β	ES	p	β	ES	p
Constant	158.343	24.642	**0.000**	35.472	6.057	**0.000**	86.027	11.374	**0.000**
Fruits consumption	-3.880	2.731	**0.016**	-1.299	0.671	**0.017**	-2.310	1.255	**0.010**
Vegetables consumption	1.975	3.237	0.544	0.235	0.796	0.769	0.799	1.494	0.594
Red meat consumption	8.150	3.399	**0.019**	2.508	0.836	**0.004**	2.729	1.565	**0.015**
Fish consumption	-5.350	3.289	0.108	0.067	0.808	0.934	-2.329	1.517	0.129
NaCl g/24 h	1.837	0.550	**0.001**	0.385	0.135	**0.006**	0.853	0.254	**0.001**
	R^2=0.53			**R^2=0.512**			**R^2=0.516**		
Overall p-value	**<0.001**			**0.001**			**<0.001**		

LVM=Left Ventricular Mass; LVMIh=Left Ventricular Mass Indexed to Height; LVMIbsa=Left Ventricular Mass Indexed to Body Surface Area; NaCl g/24 h=Estimated daily sodium chloride intake

present study, echocardiography was performed by an experienced cardiologist with post-graduate training in cardiac imaging. In this study, dietary behaviors were evaluated using a semi-quantitative Food Frequency Questionnaire (FFQ). This is a method widely used in epidemiology to assess the eating habits of respondents [40]. The FFQ used in the present study has a double limitation. First, this questionnaire explored the frequency rather than the actual amount of different foods consumed, assuming that frequency of consumption is a surrogate for quantity. Second, this questionnaire is based on the memory and skills of the interviewer. However, using the previous week as a reference period could reduce reporting biases due to memory. Finally, the cross-sectional design of this study does not allow formal establishment of any causal links in the observed associations, and the existence of potential confounding factors remains possible.

Conclusions

Our results showed a deleterious effect of westernized diet pattern, red meat consumption and high salt intake on left ventricular structure and function in hypertensive black Sub-Saharan Africans. Our findings have implications for dietary choices with respect to consumption of specific food items. Our data support current international hypertension guidelines [107-109] and lifestyle management guidelines that recommend limiting red meat consumption and encourage the consumption of fruits, vegetables and fish as one step towards maintaining and promoting cardiovascular health [110,111].

Author's Contributions

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