

# Left Ventricular Mass of Normotensive Adolescent Progeny of Nigeria Hypertensives

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## Abstract

Background: Left ventricular mass is known to be influenced by genetic factors. This study was carried out to determine the influence of parental history of hypertension on the left ventricular mass of normotensive adolescent offspring of Nigerian Hypertensives. Method: The study population comprised 91 normotensive adolescents (13-19yrs) with positive family history of hypertension in one or both biological parents, and 92 age and gender matched normotensive controls whose parents were normotensive. Anthropometric parameters and blood pressure were obtained and electrocardiogram performed for each individual. Left ventricular mass was determined by echocardiography. Result: LVM and LVM index were significantly higher in the subjects than in the controls,  $179.10 + 40.67\text{g}$  vs.  $147.70 + 30.98\text{g}$   $p < .000001$  and  $106.97 + 18.49\text{g/m}^2$  vs.  $87.95 + 16.54\text{g/m}^2$ ,  $p < .0001$  respectively. Offspring of maternal hypertensives had significantly higher LVM and LVMI than those with paternal hypertension,  $187.46 + 38.19\text{g}$  vs.  $173 + 31.75\text{g}$   $p < .001$  and  $110.15 + 20.35\text{g/m}^2$  vs.  $104.31 + 17.54\text{g/m}^2$ ,  $p < .001$  respectively. However, when LVM was indexed to height<sup>2.7</sup> no difference was observed between the LVM of these cohorts. The case prevalence of LVH determined by echocardiography was 19% in the subjects and 3% in the controls,  $p < .00001$ . Conclusion: This study demonstrates that normotensive offspring of Nigerian hypertensives have larger LVM and both maternal and paternal hypertension seem to confer equal susceptibility to LVH.

## INTRODUCTION

Left ventricular hypertrophy is a major and independent risk factor for cardiovascular morbidity and mortality<sup>1</sup>, even among normotensive individuals.<sup>2</sup> Left ventricular mass is influenced by both genetic and environmental factors as well as by lifestyle. Established predictors of left ventricular mass include age, gender, race, anthropometric factors (BMI, BSA, weight) and systolic blood pressure. Other postulated determinants of left ventricular mass include smoking, alcohol consumption; dietary sodium intake, physical activity and various hormonal peptides.<sup>3-6</sup> Genetic and population based studies across Europe and the United States have demonstrated that inherited factors are also important determinants of left ventricular mass.<sup>7-9</sup>

However, in most parts of Africa particularly Nigeria there is paucity of studies showing the influence of genetic factors as determinants of left ventricular mass. We therefore studied the influence of parental history of essential hypertension as a surrogate of genetic influence on the ventricular mass.

## CONCEPTUALIZATION OF THE PROBLEM

This study was powered to evaluate the influence of family history of hypertension on the LVM of normotensive offspring, the family history of hypertension being a surrogate marker of genetic influence. Left ventricular mass and frequency of LVH were determined by echocardiography. LVH was also assessed by electrocardiography. The influence of age, sex weight and blood pressure as covariates were also evaluated. Ethnicity was not evaluated because we were studying a homogenous African population. There was no attempt to correlate parents LVM and environmental factors to the LVM in offspring.

## METHODS

### STUDY DESIGN

One hundred and eighty three normotensive (systolic blood pressure (SBP)  $< 140$  and diastolic blood pressure  $< 90$  mmHg) individuals between the ages of 13 and 19 were recruited for this study. These were the offspring of patients who attended the medical outpatient clinic of a government owned academic hospital in an urban centre in Nigeria.

Subjects were consecutively recruited through consenting parents. The subjects, 91, comprised normotensive individuals with a positive family history of hypertension in one or both biological parents. Family history of hypertension was defined as medical history of essential hypertension (BP  $\geq$  140/190mmHg) in one or both biological parents and or history of use of anti-hypertensives. The controls, 92, consisted of age and gender matched normotensive progeny of normotensive parents. The subjects were further divided into 3 groups viz; 39 were from hypertensive mothers, 38 had hypertensive fathers while in 14 of them both parents were hypertensive (fig. 1).

### SAMPLE SIZE

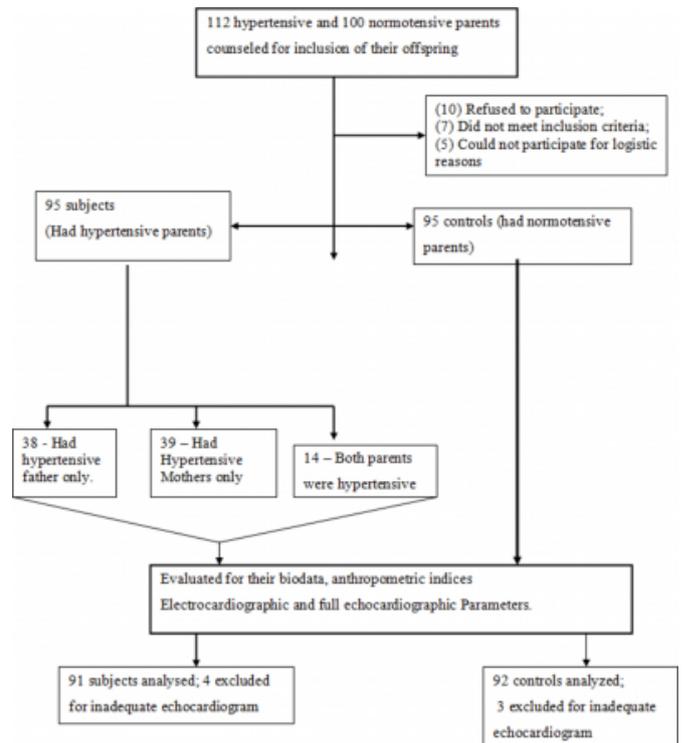
This was calculated using the formula for survey among a healthy population:  $n = Z^2pq/d^2$ , where n is the sample size,  $Z = 1.96$  (at 95% confidence limit), p = estimated disease prevalence (assumption of 50% with a precision of  $\pm 10\%$  for an unknown prevalence was used as there are no Nigerian prevalence figures);  $q = 1-p$  and d is the difference between two sub-samples (0.05). This calculation yielded a sample size of 96.

### EXCLUSION CRITERIA

Individuals with any cardiovascular, renal or metabolic diseases were excluded from the study. Smokers, competitive athletes and individuals on steroids were also excluded.

**Figure 1**

Figure 1: consort – Like diagram of study design



Written informed consent was obtained from participants and the study was approved by the ethical committee of the Hospital.

The bio-data, medical, family and social history of participants and their parents were obtained and entered into a standard format.

### ANTHROPOMETRIC INDICES

Weight (to nearest 0.5kg) and height (to nearest 0.1cm) were measured without footwear, headgear or heavy clothes. Body surface Area<sup>10</sup> was calculated using the formula (weight<sup>0.425</sup> (kg) X height<sup>0.75</sup> (cm) X 71.84/10000m) and expressed in meters squared. Body Mass Index (BMI)<sup>11</sup> was also calculated.

### BLOOD PRESSURE

Blood pressure was measured after a 5-minutes rest in the sitting position using an Accoson branded mercury sphygmomanometer on both arms, using appropriate cuff sizes. Subsequent measurements were done on the arm with a higher blood pressure value. Three blood pressure measurements were taken at 1-2 minutes intervals. Systolic and diastolic blood pressures were determined at Korotokoff phases I and V respectively. The average of the 3 measurements was used in the analysis.

**ELECTROCARDIOGRAPHY**

A standard resting 12 lead ECG was done for all subjects and controls using the Kenz 110 model ECG machine. Lead placement was according to the recommendation of the American Heart Association.<sup>12</sup>

LVH was diagnosed using the Sokolow-Lyons criteria (SV1 + RVs> 35mm) and the Cornell Voltage criteria (SV3 + RaVL>28mm for males and >20mm females).

**ECHOCARDIOGRAPHY**

Echocardiography was done with a Hewlett Packard Sonos 2000 model echocardiography machine using a 3.5 MHz transducer. Images were acquired in the left decubitus position and 2-D directed M-mode echocardiograms of the left ventricle were obtained at end inspiration from the parasternal long-axis. Measurements were taken according to the recommendations of the American Society of Echocardiography, (ASE).<sup>13</sup>

Left ventricular mass was calculated by the modified cubed formula according to the ASE convention.<sup>13</sup>

$$LVM = 0.8\{1.04 (LVIDD+PWT)^3 -(LVIDD^3) - 13.6\} + 0.6$$

LVM was indexed to BSA to derive LVMI

LVM was also indexed to height<sup>2.7</sup>, the power of allometric growth relationship between LVM and body height. Value greater than 51g/m<sup>2.7</sup> was regarded as LVH.<sup>14</sup>

**STATISTICAL ANALYSIS**

Data obtained was analyzed with SPSS version 10.0 software. Continuous variables are presented as mean ± S.D. Chi square and ANOVA were used to compare differences in proportions between groups. Simple and stepwise regression analyses were used to calculate relationship between LVM and independent variables. Statistical significance was said to be present at p<0.05.

**RESULTS**

**GENERAL CHARACTERISTICS OF THE STUDY POPULATION**

The age, gender distribution, anthropometric indices were comparable in subjects and controls Table 1.

**Figure 2**

Table 1: General Characteristics and Echocardiographic Features of the study Population

| Parameter                                   | Subjects         |                  | P - value  |
|---|------------------|------------------|------------|
|   | Mean ± SD (n=91) | Mean ± SD (n=92) |            |
| Age (yr)                                    | 16.21 ± 1.88     | 16.45 ± 1.91     | 0.42       |
| Gender                                      |                  |                  |            |
| Males                                       | 46 (50.5%)       | 50(45.65%)       | 0.47       |
| Females                                     | 45(49.5%)        | 42(45.65%)       | 0.47       |
| Weight (kg)                                 | 59.88 ± 12.96    | 60.67 ± 12.62    | 0.69       |
| Height (m)                                  | 1.67 ± 0.08      | 1.68 ± 0.07      | 0.34       |
| BMI (kg/m <sup>2</sup> )                    | 21.43 ± 4.01     | 21.40 ± 3.88     | 0.96       |
| BSA (M <sup>2</sup> )                       | 1.69 ± 0.19      | 1.69 ± 0.18      | 0.57       |
| SBP (mmHg)                                  | 112.90 ± 8.59    | 106.24 ± 5.96    | <0.0001    |
| DBP (mmHg)                                  | 71.24 ± 5.11     | 68.71 ± 4.36     | <0.0001    |
| IVS (cm)                                    | 1.13 ± 0.12      | 0.99 ± 0.09      | <0.0001    |
| PWT(cm)                                     | 0.95 ± 0.12      | 0.89 ± 0.09      | <0.0001    |
| LVDD(cm)                                    | 4.29 ± 0.32      | 4.21 ± 0.27      | 0.20       |
| LVDS(cm)                                    | 2.93 ± 0.43      | 2.89 ± 0.51      | 0.59       |
| LVM(g)                                      | 179.10 ± 40.67   | 147.70 ± 30.98   | <0.0001    |
| LVMI(g/m <sup>2</sup> )                     | 106.97 ± 18.49   | 87.95 ± 16.54    | <0.0001    |
| LVM/Ht <sup>2.7</sup> (g/m <sup>2.7</sup> ) | 44.86 ± 8.75     | 36.36 ± 7.13     | <0.0001    |
| LVH   | 19%              | 3%               | <0.0000001 |

Legend: IVS: = Interventricular septum; PWT = Posterior Wall Thickness; LVDD: = Left ventricular Diameter in Diastole; LVDS = Left ventricular Diameter in Systole; LVM: = Left Ventricular Mass; LVMI = Left Ventricular Mass Index; LVM/Ht<sup>2.7</sup> = Left ventricular Mass Indexed to Height 2.7; LVH = LVM/Ht<sup>2.7</sup> >51g/m<sup>2.7</sup>

Both SBP and DBP were significantly higher in the subjects than in the controls, p<0.00001 and p<0.00001, respectively as shown in Table 1. There was no gender difference in the anthropometric indices and blood pressure measurements.

Echocardiographic parameters are shown in Table 1. Subjects had significantly higher mean values of IVS, PW, LVM, LVMI, and LVM/Ht<sup>2.7</sup> than the controls. These parameters increased with age in both groups. (Table 2)

**Figure 3**

Table 2: Age Distribution Of Echocardiographic Parameters

| Parameters                   | Subjects (Mean ± SD); (%) |                     | p-value | Controls (Mean ± SD), (%) |                    | p - value |
|------------------------------|---------------------------|---------------------|---------|---------------------------|--------------------|-----------|
|                              | (13-15yrs) (n = 42)       | (16-19yrs) (n = 42) |         | (13-15yrs) (n = 39)       | (16-19yrs) (n= 53) |           |
| IVS (cm)                     | 1.10±0.12                 | 1.16±0.11           | <0.005  | 0.96±0.09                 | 1.01±0.09          | <0.005    |
| PW (cm)                      | 0.90±0.13                 | 0.98±0.11           | <0.005  | 0.86±0.09                 | 0.91±0.08          | <0.0001   |
| LVDD (cm)                    | 4.16±0.25                 | 4.41±0.31           | <0.0001 | 4.08±0.21                 | 4.30±0.28          | <0.0001   |
| LVDS (cm)                    | 2.76±0.31                 | 2.94±0.51           | 0.38    | 2.71±0.49                 | 2.91±0.61          | 0.34      |
| LVM (g)                      | 163.19±38.57              | 192.48±37.82        | <0.0001 | 131.81±24.46              | 158.96±30.35       | <0.001    |
| LVMI (g/m <sup>2</sup> )     | 100.65±18.15              | 112.28±17.24        | <0.005  | 80.19±13.71               | 93.45±16.28        | <0.001    |
| LVM/Ht (g/m <sup>2.7</sup> ) | 41.68±8.25                | 47.54±8.33          | <0.005  | 33.44±5.16                | 38.43±7.64         | <0.01     |
| LVH                          | 2 (10.5)                  | 17 (89.5)           | <0.001  | -                         | 3 (100)            | -         |

Legend: IVS: = Interventricular septum; PWT = Posterior Wall Thickness; LVDD: = Left ventricular Diameter in Diastole; LVDS = Left ventricular Diameter in Systole;

LVM: = Left Ventricular Mass; LVMI = Left Ventricular Mass Index; LVM/Ht<sup>2.7</sup> = Left ventricular Mass Indexed to Height 2.7; LVH = LVM/Ht<sup>2.7</sup> >51g/m<sup>2.7</sup>.

Mean LVM value was significantly higher in males than in females in both groups viz; 194.02 ± 36.79g vs. 163.81 ± 39.19g, p<0.001 in the subjects and 155.97 ± 34.95g vs. 137.65 ± 21.86g, p<0.001 in the controls. Mean LVMI (LVM indexed to body surface area) was also significantly higher in males than females viz; 112.74 ± 13.23g/m<sup>2</sup> vs. 101.06 ± 21.24g/m<sup>2</sup>, p<0.005 for the subjects and 91.22 ± 16.83g/m<sup>2</sup> vs. 83.96 ± 15.49g/m<sup>2</sup>, p<0.05 for the controls (Table 3).

**Figure 4**

Table 3: Gender Distribution Of Echocardiographic Parameters

| Parameters                                 | Subjects (Means ± SD): (%) |                  | p-value | Controls (mean ± SD): (%) |                  | p-value |
|--|----------------------------|------------------|---------|---------------------------|------------------|---------|
|  | Males (n = 46)             | Females (n = 45) |         | Males (n = 50)            | Females (n = 42) |         |
| IVS (cm)                                   | 1.17±0.11                  | 1.10±0.12        | 0.24    | 1.00±0.09                 | 0.87±0.09        | 0.11    |
| PWT (cm)                                   | 0.96±0.11                  | 0.93±0.14        | 0.60    | 0.91±0.09                 | 0.87±0.08        | 0.28    |
| LVDD (cm)                                  | 4.41±0.31                  | 4.18±0.27        | <0.005  | 4.27±0.30                 | 4.14±0.22        | 0.09    |
| LVDS (cm)                                  | 2.91±0.33                  | 2.86±0.44        | 0.43    | 2.86±0.51                 | 2.83±0.49        | 0.46    |
| LVM (g)                                    | 194.02±36.79               | 163.81±39.12     | <0.001  | 155.97±34.95              | 137.65±21.86     | <0.001  |
| LVMI (g/m <sup>2</sup> )                   | 112.74±13.23               | 101.06±21.24     | <0.005  | 91.22±16.83               | 83.96±15.49      | <0.05   |
| LVMHt <sup>2.7</sup> (g/m <sup>2.7</sup> ) | 46.08±7.11                 | 43.62±10.00      | 0.21    | 37.06±7.67                | 35.50±6.42       | 0.33    |
| LVH  | 13 (68.4)                  | 6 (31.6%)        | <0.01   | 2 (66.7)                  | 1 (33.3%)        | 0.04    |

Legend: IVS: = Interventricular septum; PWT = Posterior Wall Thickness; LVDD; = Left ventricular Diameter in Diastole; LVDS = Left ventricular Diameter in Systole; LVM: = Left Ventricular Mass; LVMI = Left Ventricular Mass Index; LVM/Ht<sup>2.7</sup> = Left ventricular Mass Indexed to Height 2.7; LVH = LVM/Ht<sup>2.7</sup> >51g/m<sup>2.7</sup>

Subjects with maternal hypertension alone had the highest mean LVM, which differed significantly from the mean value of subjects with paternal hypertension alone, 187.46 ± 38.19g vs. 173.93 ± 31.75g, p<0.001. Subjects in whom both parents were hypertensive had a comparable mean LVM with those with maternal hypertension alone, 183.96 ± 36.14g vs. 187.46±36.14g, p<0.68. However, their mean LVM was significantly higher than those with paternal hypertension alone, 183.96 ± 36.14g vs. 173.93 ± 31.75, p<0.002 (Table 4).

**Figure 5**

Table 4: Parental History Of Hypertension And Derived Echocardiographic Parameters

| Parameters                                 | Hypertensive mother      | Hypertensive Father       | p-value |
|--|--------------------------|---------------------------|---------|
|  | Means ± SD; (%) (n = 39) | Means ± SD; (%) (n = 38)  |         |
| LVM (g)                                    | 187.46±38.19             | 173.93±31.75              | <0.001  |
| LVMI (g/m <sup>2</sup> )                   | 110.15±20.53             | 104.31±17.54              | <0.001  |
| LVMHt <sup>2.7</sup> (g/m <sup>2.7</sup> ) | 46.48±8.60               | 45.01±9.69                | 0.76    |
| LVH  | 9 (23.1)                 | 5 (13.1)                  | 0.21    |
| Parameters                                 | Hypertensive mother      | Both parents hypertensive | p-value |
|  | Means ± SD; (%) (n=39)   | Means ± SD; (%) (n=14)    |         |
| LVM (g)                                    | 187.46±38.14             | 183.96±36.14              | 0.68    |
| LVMI (g/m <sup>2</sup> )                   | 110.15±20.53             | 108.14±18.21              | 0.38    |
| LVMHt <sup>2.7</sup> (g/m <sup>2.7</sup> ) | 46.48±8.60               | 44.29±7.80                | 0.76    |
| LVH  | 9 (23.1)                 | 5 (35.7)                  | 0.18    |
| Parameters                                 | Hypertensive father      | Both parents hypertensive | p-value |
|  | Means ± SD; (%) (n = 38) | Means ± SD; (%) (n = 14)  |         |
| LVM (g)                                    | 173.93±31.75             | 183.96±36.14              | <0.002  |
| LVMI (g/m <sup>2</sup> )                   | 104.31±17.54             | 108.14±18.21              | <0.001  |
| LVMHt <sup>2.7</sup> (g/m <sup>2.7</sup> ) | 45.01±9.69               | 44.29±7.80                | 0.68    |
| LVH  | 5 (13.1)                 | 5 (35.7)                  | 0.15    |

Legend: LVM = Left Ventricular Mass, LVMI = Left Ventricular Mass Index, LVMHt<sup>2.7</sup> = Left ventricular Mass Indexed to Height 2.7, LVH = LVMHt<sup>2.7</sup> >51g/m<sup>2.7</sup>

Also the mean LVMI was significantly higher in offspring of maternal hypertensives compared to those of paternal hypertensives, 110.15 ± 20.53g/m<sup>2</sup> vs. 104.31 ± 17.54g/m<sup>2</sup>, p<0.001. This parameter was comparable in offspring with maternal hypertension alone and in those in whom both parents were hypertensive, 110.15 ± 20.53g/m<sup>2</sup> vs. 108.14 ± 18.21g/m<sup>2</sup>, p<0.38. On the other hand mean LVMI value was higher in offspring in whom both parents were hypertensive than in those with paternal hypertension alone viz; 108.14 ± 18.21g/m<sup>2</sup> vs. 104.31 ± 17.54g/m<sup>2</sup>, p<0.001 (Table 4)

LVM indexed to Ht<sup>2.7</sup> was significantly higher in the subjects than in the controls p<0.00001 Table 1. There was significant age but no gender differences in the mean value of this parameter (Table 2 & 3). The mean value of this parameter was comparable amongst the three sub groups of subjects with history of parental hypertension viz; 46.48 ± 8.60 and 45.01 ± 9.69, p=0.76 for maternal and paternal hypertensives respectively, 46.48 ± 8.60 and 44.29 ± 7.80, p=0.76 for maternal hypertensives and in those whom both parents were hypertensive and 45.01 ± 9.69 and 44.29 ± 7.80, p=0.68 for paternal hypertensives and those in whom both parents are hypertensive respectively. (Table 4)

Table 5 summarizes the correlation between LVM and independent variables. LVM had the best correlation with BSA, weight, height and age in both groups. It had a moderate correlation with DBP and male gender though weak was statistically significant. BMI and female gender had the weakest correlation with LVM. In a multiple regression model only age, weight and SBP were found to significantly correlate with LVM (R<sup>2</sup> = 0.65, p<0.01) and SBP (R<sup>2</sup> = 0.10, p<0.01).

**Figure 6**

TABLE 5: Pearson’s correlation between Left Ventricular Mass and selected independent clinical variables.

| Variable      | Subjects                |         | Controls                |         |
|---------------|-------------------------|---------|-------------------------|---------|
|               | Correlation coefficient | p-value | Correlation coefficient | p-value |
| Age           | 0.81                    | <0.0001 | 0.89                    | <0.0001 |
| Male gender   | 0.31                    | 0.03    | 0.37                    | 0.01    |
| Female gender | 0.11                    | 0.07    | 0.27                    | 0.07    |
| Weight        | 0.87                    | <0.001  | 0.82                    | <0.0001 |
| Height        | 0.73                    | <0.001  | 0.67                    | <0.001  |
| BMI           | 0.28                    | 0.10    | 0.36                    | 0.16    |
| BSA           | 0.79                    | <0.0001 | 0.84                    | <0.0001 |
| SBP           | 0.61                    | <0.0001 | 0.42                    | <0.0001 |
| DBP           | 0.41                    | <0.001  | 0.33                    | <0.0001 |

Legend: BMI = Body Mass Index; BSA = Body Surface Area; SBP = Systolic Blood

**ELECTROCARDIOGRAPHIC LVH**

Using the Sokolow-Lyon criteria, the case prevalence for LVH was significantly higher in the subjects than in the controls, 27% vs. p<0.001. The Cornell Voltage criteria yielded a case prevalence of LVH of 18% and 3% for subjects and controls respectively p<0.001.

**ECHOCARDIOGRAPHIC LVH**

The case prevalence of echocardiographic LVH defined as  $LVM/Ht^{2.7} > 51g/m^{2.7}$  was 19% amongst the subjects, which differed significantly from the 3% amongst the controls, p<0.0000001. Table 1.

Prevalence of LVH was higher in the older age group. Seventeen (89.5%) of the subjects with echocardiographic LVH were within the 16-19 year age group while 2 (10.5%) were within the 13-15 years age group (Table 2). The prevalence of LVH was also higher in males than in females viz, 13(68.4%) vs. 6(31.6%) p<0.0001(Table 3).

Mean SBP was significantly higher in subjects with LVH than in subjects who did not have LVH,  $118.76 \pm 7.18mm/Hg$  vs.  $112.90 \pm 8.59mm/Hg$ , p<0.0001. DBP was comparable in subgroups,  $73.19 \pm 4.26mm/Hg$  vs.  $71.24 \pm 5.11mm$ , p=0.39. In the controls mean SBP and DBP were comparable in those with LVH and those without LVH viz  $107.89 \pm 5.56mmHg$  vs  $106.24 \pm 5.96mmHg$ , p= 0.73 for SBP and  $70.16 \pm 4.27mmHg$  vs  $68.13 \pm 4.36mmHg$ , p= 0.68 for DBP.

LVH was commoner in subjects in whom both parents were hypertensive, 5(35.7%) than in the other sub-groups viz 9(23.1%) for offspring of maternal hypertensives and 5(13.1%) in those from paternal hypertensives .However these differences were not significant (Table 4).

**DISCUSSION**

The present study was aimed at determining the influence of family history of hypertension on LVM in adolescent Nigerians.

**LVM AND FAMILY HISTORY OF HYPERTENSION**

In this study, offspring of hypertensive parents had a significantly larger LVM than controls. This correlates with findings from other studies.<sup>15-17</sup> This study however did not reveal any differences between maternal and paternal influences on LVM contrary to what had been documented by previous studies.<sup>17-18</sup> Kuznetsova et al<sup>18</sup>, studying multiple adult offspring of hypertensives, their parents and first degree relatives simultaneously found stronger maternal-offspring LVM correlation than paternal-offspring LVM correlation, suggesting that maternal influences on LVM were stronger. In this study our cohort was the adolescent age group only and parental LVM and those of first degree relatives were not determined and thus could be not correlated with the LVM of offspring. These differences in methodology and analysis probably can account for the observed difference.

**LVM AND DEMOGRAPHIC PARAMETERS**

In this present study LVM increased with increasing age as reported by earlier studies.<sup>16,17,19</sup> However, the rate of increase in LVM with increasing age is blunted in older individuals especially in the presence of other risk factors.<sup>20,21</sup> Dannenberg and colleagues<sup>22</sup> demonstrated that LVM did not increase with age in a healthy sub-sample of the Framingham study suggesting that most of the supposed physiological increase in LVM is caused by other determinants. This is buttressed by studies in young subjects where age-associated increase in LVM is partly explained by body size and blood pressure<sup>20,23</sup> and this was also demonstrated in this study.

Males also had larger LVM than females as reported by other studies.<sup>16,17</sup> Di Simone et al<sup>20</sup> showed that gender difference in LVM is marginal in pre-pubertal individual but becomes more significant after puberty and parallels the sex difference in growth of body size. Hormonal factors may

account for this gender difference. Estrogen is known to down regulate the expression of genes for LVH and thus reduces the risk of LVH.<sup>24</sup> This also could explain the higher prevalence of LVH in males than in females in this study. The mean LVM obtained in this study was higher than those obtained in two Caucasian studies.<sup>15,16</sup> This difference may be genetic since it is established that Africans have larger LVM than Caucasians.<sup>15,16</sup>

### LVM AND ANTHROPOMETRIC INDICES

Anthropometric indices such as weight, height, BMI and BSA affect LVM. However, there is a lack of consensus amongst researchers on which of these indices correlates best as determinant of LVM. Three studies<sup>14,23,25</sup> in children and adolescents found weight to be the best determinant of LVM. Other investigators found BMI a better determinant of LVM especially in the presence of obesity.<sup>26,27</sup> Brandao et al<sup>28</sup> and Devereux et al<sup>29</sup> found BSA to be best determination of LVM in adolescents. In Nigeria Oladokun and Omokhodion<sup>30</sup> and Araoye et al<sup>31</sup> arrived at a similar conclusion. In this study LVM correlated best with BSA and weight but poorly with BMI, similar to the findings of Oladokun and Omokhodion.<sup>30</sup> The major drawback of BSA as a correlate of LVM is that in the presence of obesity LVH may be masked.

In the multiple regression analysis weight and not BMI was found to be a stronger determinant of LVM. This suggests that excess body weight may contribute to the development of increase in LVM and eventual LVH.

### LVM AND BLOOD PRESSURE

Blood pressure especially SBP had a close relationship with LVM in this study, with larger LVM occupying the upper quartiles of SBP curve, similar to findings from other studies.<sup>16,31,32</sup> This relationship is physiologically explainable by the fact that increased afterload associated with elevation of blood pressure is a stimulus for hypertrophy to reduce systolic wall stress of the left ventricle.<sup>33</sup>

Although SBP and to a minor extent DBP are stronger predictors of LVM<sup>25</sup>, Urbina et al<sup>19</sup>, in the Bogalusa Heart Study, found univariate association between SBP and DBP and LVM but did not find blood pressure significant in a multivariate analysis that included anthropometric variables. In this present study, multiple regression analysis involving weight and SBP showed that only weight and to minor extent SBP were determinants of LVM. This suggests that blood pressure might play some physiologic role in

determining LVM and its role may be only complementary to that of weight.

### LEFT VENTRICULAR HYPERTROPHY

The prevalence of echocardiographic LVH in this study was 19% amongst the subjects. This is lower than the prevalence rate of between 65 – 72% in Americans of African decent.<sup>35</sup> This wide difference could be due to the smaller sample size of the present study.

Secondly, Americans of African decent are a select group, with a hybrid of genetic influences due to inter-racial marriages. Thus their genetic make-up may not be exactly similar to those of homogeneous Africans. In addition the prevalence of obesity is higher in Americans of African decent than in Black Africans. They are also more exposed to environmental risk factors for cardiovascular diseases such as high salt intake from processed foods, smoking and sedentary living. All these might explain this difference in prevalence. Paucity of prevalence studies in Africans and Nigerians especially makes it difficult to compare data derived from this study.

### CONCLUSION

This study demonstrates the influence of family history of hypertension on LVM. Maternal and paternal histories of hypertension appear to have equal influence on LVM of offspring. Weight and to a minor extent SBP are other predictors of LVM.

### LIMITATIONS

The number of subjects in this study is small compared to other family studies on LVH and this may have affected some of the results. Secondly an overestimation of LVM in this study is possible due to inability to control for unknown environmental and genetic factors that may influence of the study population. The lack of local prevalence data may have introduced some bias in the calculation of sample size but this does not negate the differences demonstrated in the two groups studied. Further studies should include parents as well so that their LVM can be correlated with those of their offspring.

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