Scientific Contributions

Differences in Left Ventricular Structure Between Black and White Hypertensive Adults

The Hypertension Genetic Epidemiology Network Study


Abstract—The degree to which ethnic differences in left ventricular structure among hypertensive adults are independent of clinical and hemodynamic factors remains uncertain. We assessed whether left ventricular mass and geometry differ between black and white hypertensives after accounting for differences in such factors. Our study group comprised 1060 black and 580 white hypertensive participants free of valvular or coronary disease in a population-based cohort. Blood pressure was measured during a clinic visit and echocardiography was performed using standardized protocols. After controlling for clinical and hemodynamic parameters (cardiac index, peripheral resistance index, and pulse pressure/stroke index), both left ventricular mass and relative wall thickness were higher in blacks than whites (173.9±30.9 versus 168.3±24.3 grams, \(P=0.006\), and 0.355±0.055 versus 0.340±0.055 grams, \(P<0.001\)). Similarly, the adjusted risk of having left ventricular hypertrophy, whether indexed by height\(^2\) or by body surface area, was greater for blacks than for whites (odds ratio: 1.80; 95% CI: 1.29 to 2.51; and odds ratio: 2.50; 95% CI: 1.58 to 3.96, respectively), and this was also true for concentric geometry (odds ratio: 2.28; 95% CI: 1.22 to 4.25). Further adjustment for relatedness in this genetic epidemiological study did not attenuate these differences. Our findings confirm the strong association between black ethnicity and increased left ventricular mass and relative wall thickness in hypertensive adults and demonstrate that these differences are independent of standard clinical and hemodynamic parameters. Whether such differences relate to distinct ambulatory pressure profiles or an ethnic propensity to cardiac hypertrophy requires further investigation. (Hypertension. 2004;43:1182-1188.)

Key Words: ethnicity • hypertrophy • vascular resistance

Hypertension constitutes a major health concern for Americans of every ethnic category, but in no group is the disease more pervasive or its consequences more devastating than among blacks.\(^1\) Black hypertensive Americans exhibit greater progression to end-stage renal disease, as well as heart failure, coronary heart disease, and stroke, than their white counterparts.\(^1\) Most strikingly, they experience rates of stroke and coronary heart disease mortality that are 50% and 80% higher, respectively, than those of American whites.\(^1\)

One manifestation of hypertension’s end-organ effects, left ventricular (LV) hypertrophy, represents an important index of preclinical disease that carries incremental prognostic value beyond that afforded by traditional coronary risk factors.\(^2\) In a large cohort of blacks, LV hypertrophy proved to be an even more powerful predictor of mortality than coronary artery disease and LV ejection fraction.\(^3\) These observations have fueled interest in exploring differences in LV mass and geometry between blacks and whites. Previous studies have found LV relative wall thickness (RWT) to be higher in black normotensive\(^4–6\) and hypertensive\(^6–10\) subjects compared with whites. Among both normotensive and hypertensive patients, some studies have reported ethnic differences for LV mass,\(^9–14\) but others have not.\(^5–8,15\)

Important limitations of available ethnic studies have been the variably selected nature of some study populations and limited adjustment for effects of other variables known to be independent determinants of LV mass and geometry.\(^16–21\) Accordingly, we investigated ethnic differences in LV mass and geometry while accounting for established or potential risk factors in a large population-based cohort with mostly treated hypertension.

Methods

General Study Characteristics

The Hypertension Genetic Epidemiology Network (HyperGEN) study is a component of the National Heart, Lung, and Blood...
Institute Family Blood Pressure Program, whose objective is to investigate the genetic basis of hypertension in population-based cohorts. HyperGEN relied principally on a sib-pair design that recruited hypertensive members of sibships in which ≥2 siblings with hypertension of unknown cause and onset by age 60 agreed to participate. Hypertensive subjects had systolic blood pressure ≥140 mm Hg and/or diastolic pressure ≥90 mm Hg or received antihypertensive treatment. Four HyperGEN field centers participated in the ancillary echocardiographic study. Details of recruitment, including proportion of black and white participants, have been reported.28 For the present analyses, hypertensive patients with ≥2+ valvular regurgitation or any degree of valvular stenosis, or with overt coronary artery disease (CAD) were excluded.

The HyperGEN examination obtained standardized measurements of blood pressure at rest using an automated device, the Dinamap (Critikon, Inc.). Diabetes was diagnosed by American Diabetes Association criteria22 and hypercholesterolemia as fasting serum cholesterol ≥240 mg/dL.

### Echocardiographic Methods

As previously described,24 25 echocardiograms were performed by a standardized protocol that included M-mode, 2-dimensional (2D), and color Doppler recordings from the parasternal long-axis and short-axis windows, as well as 2D and color Doppler evaluations from the apical window to yield 2-, 3-, and 4-chamber images.

### Echocardiographic Measurements

Correct orientation of planes for 2D and Doppler imaging was confirmed using standard procedures.26 All studies were interpreted at the Echocardiography Core Laboratory using a computerized review station. Readings were blinded to clinical characteristics of participants, but not to center. LV internal dimension and septal and posterior wall thicknesses were measured on up to 3 cardiac cycles at end-diastole and end-systole by American Society of Echocardiography recommendations.27 28

### Calculation of Derived Variables

LV mass was calculated using the equation:

\[ 0.80 \times 1.04 \left( IVSTD + LVIDd + PWTD \right) - (LVIDd)^3 + 0.6 \]

which yields values closely related (R=0.90) to necropsy LV weight.29 LV hypertrophy was defined by sex-specific partition values for LV mass indexed by height\(^2\) and body surface area.\(^30\) Extended method details can be found in an online supplement available at http://www.hypertensionaha.org. RWT was calculated as posterior wall thickness/LV internal radius at end-diastole. Together with sex-specific partition values of LV mass index, a RWT cutoff of 0.43\(^\text{11}\) was used to identify LV geometric patterns.23 End-diastolic and end-systolic LV volumes calculated by the Teichholz method\(^33\) were used to calculate LV ejection fraction, whereas pulsed wave Doppler at the aortic anulus was used to compute stroke volume and cardiac output. Total peripheral resistance was calculated from the ratio of mean arterial pressure (automated recorder) and cardiac output. The ratio pulse pressure/stroke index was used as a measure of arterial stiffness.\(^34\)

### Measures of Myocardial Performance

Assessment of myocardial contractility was based on the relationship between LV mid-wall shortening and circumferential end-systolic stress.\(^35\)\(^36\)

### Statistical Analysis

The Student \( t \) test was used to compare continuous variables, and \( \chi^2 \) or Fisher exact test for categorical variables. Adjusted comparisons of continuous variables were performed by analysis of covariance. Independent correlates of LV mass and RWT were identified by multiple linear regression using the backward elimination procedure (\( P<0.05 \)). Multiple logistic regression was used to adjust for potential confounders of associations between ethnicity and LV hypertrophy and concentric geometry. Adjustment for relatedness was performed as described in the online supplement.

### Results

Of 2172 hypertensive HyperGEN participants in the Birmingham, Minneapolis, Salt Lake City, and Winston-Salem field centers, \(~99\%\) underwent echocardiography, and \(98\%\) of these individuals (\( n=2098 \)) had measurable LV mass; 376 participants with CAD, 126 with valvular heart disease, and 87 with uncertain CAD or ethnicity status were excluded. Table 1 presents the demographic and clinical characteristics of the resulting cohort of 1640 subjects, 1024 of whom belonged to 505 sibships. Blacks were younger and more often women than whites. They also had higher clinical blood pressures and body mass index, greater prevalences of diabetes mellitus and smoking, were less likely to have hypercholesterolemia, receive antihypertensive medication, or to have completed college, and received different therapeutic regimens than whites.

Echocardiographic characteristics also differed between groups (Table 2), with blacks demonstrating higher wall thicknesses and LV mass than whites. Blacks also had slightly greater cardiac index without difference in peripheral resistance index (PRI) compared with whites. Similarly, there was no ethnic difference in pulse pressure/stroke index (PP/SI). LV ejection fraction was minimally lower in black participants and LV mid-wall shortening was significantly reduced in this group. A minority of patients in either group (\(<10\%\) ) had reduced LV ejection fraction, with no patients having an LV ejection fraction \(<45\%\). Stress-corrected mid-wall shortening did not differ significantly between groups.

### Table 1. Demographic and Clinical Characteristics by Ethnicity

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Blacks (N=1060)</th>
<th>Whites (N=580)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>50.4±10.5</td>
<td>59.1±9.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Male, %</td>
<td>31.8</td>
<td>43.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>134±22</td>
<td>131±19</td>
<td>0.003</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>76±12</td>
<td>72±11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mean arterial pressure, mm Hg</td>
<td>96±14</td>
<td>91±12</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pulse pressure, mm Hg</td>
<td>58±17</td>
<td>59±15</td>
<td>0.149</td>
</tr>
<tr>
<td>Hypertension duration, y</td>
<td>13.4±11.3</td>
<td>14.1±11.2</td>
<td>0.245</td>
</tr>
<tr>
<td>Diabetes mellitus, %</td>
<td>21.3</td>
<td>13.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hypercholesterolemia, %</td>
<td>23.8</td>
<td>44.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ever smoker, %</td>
<td>51.3</td>
<td>35.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.67±0.09</td>
<td>1.68±0.09</td>
<td>0.005</td>
</tr>
<tr>
<td>Body mass index, kg/m(^2)</td>
<td>32.9±7.5</td>
<td>31.3±6.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Body surface area, m(^2)</td>
<td>1.99±0.24</td>
<td>1.97±0.23</td>
<td>0.317</td>
</tr>
<tr>
<td>Education (≥4 y of college), %</td>
<td>35.3</td>
<td>42.8</td>
<td>0.003</td>
</tr>
<tr>
<td>Antihypertensive medication, %</td>
<td>84.6</td>
<td>89.3</td>
<td>0.008</td>
</tr>
<tr>
<td>&gt;2 Antihypertensive medications, %</td>
<td>8.8</td>
<td>7.4</td>
<td>0.337</td>
</tr>
<tr>
<td>Angiotensin-converting enzyme inhibitor, %</td>
<td>28.4</td>
<td>36.6</td>
<td>0.001</td>
</tr>
<tr>
<td>( \beta )-blocker, %</td>
<td>13.2</td>
<td>23.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Calcium-channel blocker, %</td>
<td>43.1</td>
<td>25.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Thiazide diuretic, %</td>
<td>24.1</td>
<td>18.5</td>
<td>0.011</td>
</tr>
</tbody>
</table>
Ethnic comparison of LV structure, adjusting for factors traditionally recognized to influence LV mass and wall thickness, is presented in Table 3. As observed for the unadjusted comparisons, mean LV mass and wall thicknesses remained significantly greater in blacks, but LV internal dimension was significantly smaller in the latter group.

After multivariable adjustment that included antihypertensive medications, indices of hemodynamic status and vascular stiffness did not differ significantly between blacks and whites (Table 3). Heart rate and cardiac index were virtually identical in the 2 ethnic groups, and peripheral resistance was modestly but not significantly greater among blacks. Mean LV ejection fraction was similar in blacks and whites, but LV mid-wall shortening was significantly lower in blacks without significant between-group difference in stress-corrected mid-wall shortening.

To investigate further ethnic differences in LV structure, we assessed which variables among demographic characteristics, measures of body size, severity and duration of hypertension, presence of diabetes mellitus, systemic hemodynamics, vascular stiffness, and antihypertensive treatment were independent linear correlates of LV mass and RWT in the entire cohort (Table 4). Body mass index, height$^2$, and mean arterial pressure were the strongest positive correlates of LV mass.

**TABLE 3. Echocardiographic Characteristics by Ethnicity Adjusted for Potential Clinical Confounders**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Blacks N=1060</th>
<th>Whites N=580</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV mass, g</td>
<td>173.1±44.9</td>
<td>168.2±37.4</td>
<td>0.021</td>
</tr>
<tr>
<td>LV internal dimension (diastole), cm</td>
<td>5.09±0.48</td>
<td>5.12±0.47</td>
<td>0.251</td>
</tr>
<tr>
<td>Posterior wall thickness, mm</td>
<td>9.0±1.2</td>
<td>8.7±1.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Interventricular septal thickness, mm</td>
<td>9.6±1.3</td>
<td>9.4±1.2</td>
<td>0.002</td>
</tr>
<tr>
<td>Relative wall thickness</td>
<td>0.36±0.05</td>
<td>0.34±0.05</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Heart rate, min$^{-1}$</td>
<td>70.5±11.6</td>
<td>67.6±12.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cardiac index, L/min</td>
<td>2.73±0.58</td>
<td>2.67±0.58</td>
<td>0.011</td>
</tr>
<tr>
<td>Peripheral resistance index, dynes·sec·cm$^{-5}$·m$^{-2}$</td>
<td>2922±724</td>
<td>2890±707</td>
<td>0.403</td>
</tr>
<tr>
<td>Pulse pressure/stroke index, mm Hg·mL$^{-1}$·m$^{-2}$</td>
<td>1.52±0.52</td>
<td>1.54±0.50</td>
<td>0.619</td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>63.0±5.8</td>
<td>63.5±6.0</td>
<td>0.069</td>
</tr>
<tr>
<td>LV mid-wall shortening, %</td>
<td>17.3±1.8</td>
<td>17.7±1.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stress-corrected mid-wall shortening, %</td>
<td>105.4±10.5</td>
<td>106.4±10.8</td>
<td>0.090</td>
</tr>
</tbody>
</table>

*Adjusted for age, sex, body mass index, diabetes mellitus, mean arterial pressure, duration of hypertension, and antihypertensive treatment.
†Adjusted for all covariates in * except mean arterial pressure.
‡Adjusted for all covariates in * except body mass index.
§Adjusted for all covariates in * except body mass index, as well as for heart rate, hypercholesterolemia, and smoking.
¶Adjusted for all covariates in * except mean arterial pressure.
mass, whereas female gender showed a substantial negative relationship. Moreover, black ethnicity was also significantly associated with LV mass after control for all significant variables. When PRI was considered in lieu of mean arterial pressure, this variable was also significantly correlated to LV mass (Table 4). In the case of RWT, PP/SI was the strongest positive correlate, followed by black ethnicity. Height and female gender showed negative associations with RWT.

We used the significant correlates of LV mass and RWT, as well as nonsignificant indices of hemodynamic status and vascular stiffness, in parsimonious models to adjust for aggregate confounding in the observed ethnic differences. After adjustment for the variables in Table 4, as well as PP/SI, mean LV mass was significantly higher in black than in white HyperGEN participants (Figure 1a), without evidence of gender–ethnicity interaction. Adjustment for systolic pressure instead of mean arterial pressure or for additional covariates did not meaningfully influence the results. Moreover, the observed ethnic difference persisted when relatedness was taken into account (Figure 1a).

Similarly, mean RWT remained significantly greater among blacks after adjustment for covariates in Table 4, as well as mean arterial pressure and cardiac index (Figure 1b). Adjustment for other clinical covariates, or replacement of mean arterial pressure by systolic blood pressure, had no substantive effect on the results, nor was there a statistical interaction between ethnicity and gender. Furthermore, the ethnic difference in RWT persisted when relatedness was taken into account (Figure 1b).

Last, racial–ethnic differences remained significant when LV mass and RWT were dichotomized. The prevalence of LV hypertrophy in blacks was at least 1.8-fold higher than in whites regardless of the indexation used and independent of clinical and hemodynamic variables, whereas concentric geometry was more than twice as frequent in blacks when covariates were taken into account (Figure 2).

**Discussion**

In this large population-based cohort of predominantly treated hypertensive patients, blacks had higher mean LV mass index and RWT than whites—differences that persisted after adjustment for conventional demographic and clinical risk factors. Accordingly, black participants had prevalences of LV hypertrophy and concentric geometry that approached or exceeded twice those of whites. Measures of systemic hemodynamics and vascular stiffness were similar in black and white hypertensive patients, and observed ethnic differences in LV mass and geometry were also independent of these parameters. Moreover, relatedness did not meaningfully influence the results in this population drawn from sibships.

To our knowledge, ours is the largest cohort of hypertensive patients in whom black–white differences in LV structure have been evaluated. This study has important strengths, including population-based recruitment, standardized blood-pressure measurement, and systematic assessment of hemodynamics and vascular stiffness.

Our findings confirm previous investigations by documenting that RWT is higher in black than white American hypertensive adults.4-9 They also demonstrate that the greater LV wall thickening in blacks is independent of potential differences in vascular hemodynamics and stiffness. Moreover, against a backdrop of previous conflicting reports, our
LV Hypertrophy (He²⁻)

<table>
<thead>
<tr>
<th></th>
<th>OR</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crude</td>
<td>1.80</td>
<td>1.41-2.31</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Multivariable-Adjusted*</td>
<td>1.80</td>
<td>1.29-2.51</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

LV Hypertrophy (BSA)

<table>
<thead>
<tr>
<th></th>
<th>OR</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crude</td>
<td>2.16</td>
<td>1.50-3.12</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Multivariable-Adjusted†</td>
<td>2.50</td>
<td>1.58-3.96</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Concentric Geometry (RWT ≥ 0.43)

<table>
<thead>
<tr>
<th></th>
<th>OR</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crude</td>
<td>1.93</td>
<td>1.19-3.14</td>
<td>0.008</td>
</tr>
<tr>
<td>Multivariable-Adjusted‡</td>
<td>2.28</td>
<td>1.22-4.25</td>
<td>0.010</td>
</tr>
</tbody>
</table>

Figure 2. Prevalence of LV hypertrophy and concentric geometry in blacks vs whites. BSA indicates body surface area; CI, confidence interval; Ht, height; LV, left ventricular; OR, odds ratio; RWT, relative wall thickness. Adjusted for age, sex, body mass index, diabetes mellitus, mean arterial pressure, duration of hypertension, hypercholesterolemia, smoking, education, treatment with angiotensin-converting enzyme inhibitors, β-blockers, calcium-channel blockers, and thiazide diuretics, cardiac index, and pulse pressure/stroke index. †Adjusted for all covariates in * except for body mass index. ‡Adjusted for all covariates in † as well as for height. §Logarithmic scale.

Results buttress the contention that LV mass is greater in black than white American hypertensives receiving antihypertensive treatment.

In both normotensive and hypertensive blacks, total peripheral resistance has been previously documented to be higher than in their white counterparts. Whether this represents a difference in hypertension pathogenesis or severity is unclear. Vascular stiffness, by contrast, is associated with aging and atherosclerosis, as well as with increased intra-arterial distending pressure. In the present study, the higher arterial pressure in black than in white patients was caused by a nearly significant, modestly higher mean value for PRI, without significant difference between groups in cardiac index or PP/SI.

Previous studies have reported total peripheral resistance to be associated with both greater LV wall thickness and mass. Higher peripheral resistance results in LV hypertrophy by increasing systemic blood pressure and hence LV systolic pressure. Systolic LV wall stress, the principal stimulus to myocyte hypertrophy, is therefore amplified considerably, leading to a corresponding increase in LV mass. Because increased resistance to flow may be accompanied by a reduced stroke volume, high peripheral resistance may also be associated with reduced LV chamber volume and, thereby, with elevated RWT.

In our cohort, PRI was independently correlated with LV mass but not with RWT. Despite previously reported ethnic differences in peripheral resistance and the modestly higher PRI among blacks in our study, the higher LV mass in blacks was statistically independent of this factor. Cardiac index was also a significant correlate of LV mass, in keeping with previous studies showing an association between stroke volume and LV mass through its relationship with LV internal diameter. PP/SI, in turn, was the strongest positive correlate of RWT in our study, consistent with previous reports.

Significant hemodynamic and clinical predictors accounted for only a modest proportion (37.2%) of the variability in LV mass in our cohort. Our multiple R of 0.61, however, approaches values derived in other populations using similar methods. The somewhat smaller multiple correlation observed herein may relate to our more narrowly defined sample, which excludes normotensive individuals with, on average, lower body mass index, diabetes prevalence, and LV mass, as well as arterial pressure.

Having largely excluded potential effects of conventional risk factors, as well as of hemodynamic and vascular parameters and relatedness, on the observed black–white differences in LV structure in this study, a fundamental question is whether these differences relate to an ethnicity-specific genetic predisposition, to unmeasured confounders, or to a combination thereof. Poorer access to care among blacks could potentially have led to underestimation of hypertension duration in this group, and we cannot exclude the possibility that longer duration of undetected hypertension among blacks may have contributed to the results. In addition, our study did not assess ambulatory blood pressure, which would have helped elucidate the basis for our findings. Blacks often have higher nighttime blood pressure than whites with similar daytime blood pressure. Moreover, blacks show greater vascular reactivity than whites. Intermittent periods of blood pressure elevation in response to stress or intervals of nocturnal hypertension might explain the higher LV mass and RWT in blacks. In another study in the HyperGEN cohort, however, handgrip and mental arithmetic did not lead to a differential increase in vascular reactivity in blacks. Nevertheless, the present study cannot address the role of differential diurnal blood pressure patterns in the ethnic differences described. Nor can the relative contribution of genetic and environmental influences be unraveled. Such issues are in need of further investigation.

Whatever the causes behind the ethnic differences in LV mass and geometry, these findings have potentially far-reaching implications. LV hypertrophy carries independent prognostic information over and above conventional cardiovascular risk factors for black and white cohorts alike. The higher prevalence of cardiac hypertrophy among hypertensive blacks in this study supports the need for improved attainment of blood pressure goals in these patients. It also suggests that increased screening for end-organ damage may be warranted in this population. In this regard, the fact that electrocardiography is insensitive, and less specific in blacks than whites, for detection of LV hypertrophy, may make echocardiographic evaluation especially valuable in blacks.

Among the study’s limitations is its cross-sectional design, which can reveal associations between variables but cannot demonstrate that the observed relationships are causal. Furthermore, because most HyperGEN participants were recruited from sibships with unexplained, relatively early-onset, and predominately treated hypertension, these findings may not apply to all hypertensive patients.
Perspectives
This investigation shows important differences between treated black and white hypertensive patients in LV mass and geometry that are independent of clinical covariates and indices of systemic hemodynamics. Whether an inherent ethnic propensity or greater severity of hypertension and other factors is operative in the genesis of LV remodeling in blacks remains unresolved. Additional studies are required to better understand ethnic differences in hypertension etiology and severity, and how these influence the process of cardiac hypertrophy. Such information could help narrow the gap in adverse outcomes that currently characterizes the natural history of hypertension in the 2 ethnic groups.

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Methods

Calculation of Derived Variables

LV mass measurements derived from M-mode or 2D LV linear showed good interstudy reproducibility ($\rho=0.93$) in a separate cohort of 183 hypertensive patients assessed at the Echocardiography Reading Center.¹ We defined LV hypertrophy based on two criteria: (1) LV mass/height $^{2,7}$, which normalizes cardiac mass for ideal body size estimated from body height, using previously derived partition values of 49.2 g/m$^{2.7}$ and 46.7 g/m$^{2.7}$ in men and women;² and (2) LV mass/BSA, applying cut points of 104 g/m$^2$ in women and 116 g/m$^2$ in men to identify LV hypertrophy independently of obesity.²

Statistical Analysis

Because the HyperGEN cohort is composed of sibships, phenotypic observations in individual members of this population are not fully independent. Accordingly, supplemental analyses of the relationship of ethnicity to LV mass and RWT were performed using the MIXED procedure in SAS, version 8.2. Non-independence among family members was adjusted for by using a “sandwich estimator” that asymptotically yields the same parameter estimates as ordinary least squares or regression methods, but with standard errors and, consequently, hypothesis tests, that account for relatedness of participants. The method is general, assuming the same degree of dependency among all members within a sibship.
References


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