Left Ventricular Hypertrophy in Blacks and Whites
Different Genes or Different Exposure?

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The evidence that blacks carry greater left ventricular (LV) mass than whites is almost indisputable, as recently confirmed by new epidemiological findings and analysis of previous literature.\textsuperscript{1,2} There are only a few studies denying this ethnic difference, a finding possibly attributable to selection of participants and distribution of risk factors.\textsuperscript{2} The conclusion that LV mass is greater in blacks than in whites is now confirmed by a new epidemiological study using a different (and possibly more accurate) method to calculate LV mass, with MRI at high magnetic field.\textsuperscript{3} It is relevant that the criteria for definition of MRI LV hypertrophy derived from analysis of normal population-specific distribution are not substantially different from those derived from distribution criteria in echocardiographic studies using 2D-targeted M-mode tracing, especially in men:\textsuperscript{4,5} 111 g/m\textsuperscript{2} in men (versus 112 g/m\textsuperscript{2} with MRI) and 106 g/m\textsuperscript{2} in women (versus 89 g/m\textsuperscript{2} with MRI), a gender difference that might reflect differences in ethnicity and body size distribution. The consistency of findings obtained using different methods (ultrasound and MRI) further increases the confidence that according to the present predictive models, ethnicity is an independent contributor to LV mass; but how much variability can be explained primarily by ethnicity remains to be clarified.

In a recent article from the HyperGEN network, Kizer et al\textsuperscript{6} reported that LV mass remained greater in 1060 hypertensive black individuals than in 580 white participants, even after adjusting for sex, body build, blood pressure (BP), diabetes, duration of hypertension, and antihypertensive treatment. However, even in that study, African-American ethnicity explained <1% of variability of LV mass once the effect of the considered confounders was taken into account. Similarly, variability of relative wall thickness depending on African-American ethnicity was 2%. In addition to the HyperGEN regression model, Drazner et al\textsuperscript{3} also analyzed socioeconomic factors as additional confounders for ethnicity-related LV mass differences.

Differences in socioeconomic status are able to track different distributions of risk factors and behavior in their management, a well-known problem,\textsuperscript{6,7} justifying this important analysis. Compared with the regression models adjusting for confounders, considerations of socioeconomic factors did not change the influence of ethnicity on prevalence of LV hypertrophy, defined by normalization of LV mass for body surface area, a method that takes away the effect of obesity.\textsuperscript{5} In contrast, the ethnic difference in the prevalence of LV hypertrophy was reduced when accounting for socioeconomic factors, when definition of LV hypertrophy was based on normalization for height\textsuperscript{8}, a method that includes obesity-related LV hypertrophy and, at least in populations with high prevalence of obesity (which is the case in the Dallas Heart Study), produces the highest population attributable to risk.\textsuperscript{9} These findings and considerations of population risk attributable to LV hypertrophy\textsuperscript{8} indicates that obesity is a major health problem in black US citizens and supports the hypothesis that a potent interaction among different risk factors might be operating in this ethnic group, which is not completely highlighted by the current regression modeling, as a consequence of the high prevalence of obesity. Thus, understanding the ethnic distribution of LV hypertrophy might be even more complex than represented in well-conducted epidemiological studies.

Compared with whites, blacks have more clustering of cardiovascular risk factors, including hypertension, obesity, and insulin resistance, but less severe lipid abnormality\textsuperscript{10} and lower waist girth for comparable levels of body mass index (BMI), possibly because of the racial differences in body composition,\textsuperscript{10} suggesting the need to define race-specific partition values.\textsuperscript{11} Interaction between obesity and hypertension is likely crucial to fully elucidate the ethnic differences because these 2 conditions are likely to have multiplicative effects. In Drazner’s work,\textsuperscript{3} using definition by LV mass/height\textsuperscript{12}, the prevalence of LV hypertrophy in the lowest thirtile of BMI was <10% versus a prevalence of \(\approx 18\)% in the lowest thirtile of BP (<120 mm Hg). In the second thirtiles, this difference in prevalence was reduced (\(\approx 22\)% for BMI versus 28% for systolic BP) and was minimized in the highest thirtiles (LV hypertrophy present in 56% for BMI versus 62% for systolic BP). This finding confirms that at the lowest levels of systolic BP or BMI, the hypertrophic stimulus produced by systolic BP is greater than the one of body size. At the highest systolic BP and BMI, the impact of body size becomes almost as important as BP and is associated with substantially high prevalence of LV hypertrophy. It is very likely that an interaction between the 2 stimuli occurs.

Increased sympathetic nervous activity and a high level of neurohormonal activation are reported in central obesity,\textsuperscript{12,14} a condition substantially more frequent in blacks than in other ethnic groups, also predisposing to hypertension.\textsuperscript{15,16} Even in the absence of hypertension and obesity, complex hemody-
namic patterns associated with central fat distribution are potentially able to amplify the effect of pressure load. The high transcriptional activity related to central fat should also be considered as a potential booster shot for myocardium to enhance response to hemodynamic stimuli.

The doubt about whether the ethnic differences in LV geometry are related to real different genetic signaling to produce more contractile elements in blacks can find a partial answer looking at children and adolescents. Dekkers et al reported that ethnicity effect on LV mass begins in early adolescence and remains independent of socioeconomic status and anthropometric and hemodynamic stimuli, and that differential cardiac growth in whites and blacks can be explained mainly by body growth and increase in adiposity. Thus, most probably, at birth, the number of cardiomyocytes is comparable in both ethnic groups, similar to what happens between genders, whereas some difference might be present in the force of contraction, as suggested by genetic analyses. At puberty, different influences occur to produce more hypertrophy in cardiomyocytes of blacks than in whites.

Therefore, to definitively separate genetic trait from environmental influence, a goal that can be clinically, socially, and economically relevant, very selected, and well matched, risk factor–free populations should be studied and potential interaction taken into account. At the level of present knowledge, environmental influence appears to be critical to explain most of the ethnic differences in LV mass.

References
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