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Abstract—Increased left ventricular mass (LVM) and lower socioeconomic status (SES) are predictors of cardiovascular morbidity and mortality. Blacks and Hispanics are more likely to have higher LVM and lower SES. The relation between SES, race–ethnicity, and LVM has not been fully explored. Data were used from the NOMAS population-based sample of 1916 subjects living in Northern Manhattan. SES was characterized on the basis of educational attainment and divided into 4 categories. Echocardiography-defined LVM was indexed according to height at the allometric power of 2.7 and analyzed as a continuous variable. LVM varied by race in our cohort (blacks 48.9 g/m$^{2.7}$, Hispanics 48.4 g/m$^{2.7}$, whites 45.6 g/m$^{2.7}$; $P=0.004$). Using ANCOVA, there was a significant inverse and graded association between mean LVM and SES for the total cohort. Mean LVM was 48.4 g/m$^{2.7}$, 48.6 g/m$^{2.7}$, 47.1 g/m$^{2.7}$, and 45.3 g/m$^{2.7}$ for the lowest to the highest educational level category (P trend$=0.0004$). This relationship remained among normotensives (P trend$=0.0005$) and was present for blacks (P trend$=0.009$), but not for whites (P trend$=0.86$) or Hispanics (P trend$=0.47$). The difference in mean LVM between the highest and lowest categories of education was 5.3 g/m$^{2.7}$ for blacks, 0.0 g/m$^{2.7}$ for whites, and 1.0 g/m$^{2.7}$ for Hispanics. Lower SES is an independent predictor of increased LVM among hypertensive and normotensive blacks. **(*Hypertension*. 2004;43:775-779.)**

Key Words: socioeconomic factors ■ hypertrophy ■ epidemiology ■ race

Despite recent declines in mortality, cardiovascular disease remains the leading cause of death in the United States today.[1] Blacks are known to have significantly higher rates of heart disease and stroke mortality compared with whites.[1,2] The substrate for increased cardiovascular disease mortality among blacks has been inadequately defined and does not appear to be fully explained by traditional risk factors such as smoking, hypertension, hypercholesterolemia, and diabetes.

Increased left ventricular mass (LVM) is a predictor of cardiovascular morbidity and mortality independent of arterial hypertension and other traditional risk factors.[3,4] Blacks are more likely to have increased LVM than whites.[5,6] Increased LVM may account for part of the increased cardiovascular mortality among blacks, and little is known about LVM among Hispanics. Unfortunately, the mechanism by which increased LVM imparts an elevated cardiovascular risk and why its prevalence may be higher among minorities remain largely unknown. An association between lower socioeconomic status (SES) and poorer health, including all-cause mortality and increased cardiovascular morbidity and mortality, has been observed.[7–9] Whether environmental precursors (including underlying socioeconomic factors) contribute to the differential burden of LVM has not been fully investigated. In the United States blacks are known to have considerably lower SES than whites.[2,9] Lower SES is associated with increased psychological stress, increased cardiovascular reactivity, and increased incidence of hypertension,[10–15] all of which may potentially contribute to the development of increased LVM. The aim of the present study is to examine the association between SES, as defined by educational level, and LVM. We will also investigate any influence of race–ethnicity on the potential relation between LVM and SES.

Methods

Subjects were participants in the Northern Manhattan Study (NOMAS), a population-based prospective cohort study designed to investigate cardiovascular and stroke incidence, risk factors, and prognosis in a multiethnic sample from northern Manhattan. The methods of subject recruitment and enrollment into NOMAS have

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From the Department of Medicine (C.J.R., R.R.S., S.H., M.R.D.), Columbia University, New York, NY; Department of Epidemiology (A.V.D.-R.), University of Michigan, Ann Arbor; Neurological Institute (B.B.-A., R.L.S.), Columbia University, New York, NY; Division of Epidemiology (R.L.S.), Columbia University Mailman School of Public Health, New York, NY; Department of Sociomedical Science (B.B.-A.), Columbia University Mailman School of Public Health, New York, NY; and Sergievsky Center (R.L.S.), College of Physicians & Surgeons, Columbia University, New York, NY.

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Correspondence to Dr Carlos J. Rodriguez, Division of Cardiology, Columbia University, College of Physicians & Surgeons, 630 West 168th Street, New York, NY 10032. E-mail cjr10@columbia.edu

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TABLE 1. Cohort Demographics by Race–Ethnicity

	Total Cohort (N=1916)	Whites (N=377)	Blacks (N=417)	Hispanics (N=1081)	*P* Value
Characteristic					
Age	68±10	73±10	70±10	66±9	<0.0001
BMI (kg/m^2)	27.6±5.3	25.8±5.1	27.9±6.2	28.2±4.9	<0.0001
Physical activity (h/wk)	2.5±4.2	3.2±4.0	2.9±1.9	1.9±3.3	<0.0001
SBP (mm Hg)	144±21	140±20	147±21	144±21	<0.0001
	%	%	%	%	
Women	61	59	66	61	<0.049
Educational level					<0.0001
< High school	54	15	31	77	
Completed high school	18	24	32	11	
Some college	12	19	19	6	
≥College graduate	16	41	19	6	
Hypertension	56	42	64	59	<0.0001
Diabetes	21	14	24	23	0.0003

41 subjects are included in the total cohort who could not be classified as black, white, or Hispanic.

been described elsewhere.[16,17] Briefly, random digit dialing of approximately 25 000 households was performed and community participants were enrolled in NOMAS if they: (1) had never had a stroke diagnosed; (2) were older than age 40; and (3) resided in Northern Manhattan for ≥3 months in a household with a telephone. NOMAS subjects with previous myocardial infarction were excluded from this study. Ninety-one percent of those called participated in a telephone interview, and 75% of those who were eligible and invited to participate came to Columbia University Medical Center (CUMC) for an in-person evaluation (overall participation rate 68%). The study was approved by the Institutional Review Board at CUMC. All participants gave consent directly or through a surrogate when appropriate. As part of NOMAS, 3298 participants underwent extensive in-person evaluation, and transthoracic echocardiograms were performed on 2003 eligible subjects. Echocardiograms that were technically adequate for analysis were obtained in 1916 subjects and are included in this study.

Blood pressure was measured with mercury sphygmomanometers and cuffs of appropriate size. Hypertension was defined as a blood pressure recording ≥140/90 mm Hg (based on the average of 2 blood pressure measurements during one sitting by a trained research assistant), the patient's self-report of a history of hypertension, or antihypertensive medication use. Diabetes mellitus was defined by the patient's self-report of such a history, use of insulin or hypoglycemic agent, or fasting glucose ≥126 mg/dL. Physical activity was assessed with a standardized questionnaire that recorded the frequency and duration of 14 different recreational activities during the 2-week period before the interview. These analyses used the total duration of physical activity in hours per week. Height and weight were determined by the use of calibrated scales. Assessments were conducted in English or Spanish, depending on the primary language of the participant. Race–ethnicity was based on self-identification through a series of interview questions modeled after the 2000 US census and conformed to the standard definitions outlined by Directive 15.

Transthoracic echocardiography was performed and measurements were taken by standard two-dimensional (2-D) protocols according to the guidelines of the American Society of Echocardiography (ASE). Left ventricular diastolic dimension (LVDD), left ventricular systolic dimension (LVSD), interventricular septal thickness (IVS), and posterior wall thickness (PWT) were measured in all patients. LVM was calculated with the use of the corrected ASE method: $0.8\times(1.04\times[(IVS+LVDD+PWT)^3-LVDD^3]+0.6)$.

LVM was then indexed to body size by dividing raw LVM by height to the allometric power of 2.7 and analyzed as a continuous variable.[18,19] Interpretation of echocardiographic studies was performed off-line by researchers blinded to the subject's clinical and demographic characteristics. Four readers over the period of 1993 to 2000 were involved in the analysis of all the echocardiographic studies. For quality-control measures, all readers were trained by senior echocardiographers (S.H. or M.D.T.) and interobserver reliability was periodically assessed among the readers by use of intraclass correlation coefficients for the variables measured, which ranged between 0.59 and 0.74.

Educational level was used as the indicator of SES and classified into 4 categories: "less than high school" included those who never went to high school or had completed only part of high school, "completed high school" included those who had completed high school or other vocational training beyond primary, "some college" included those who had some level of tertiary education, and "college graduate or more" included those who had completed college alone or with a higher degree such as a masters or doctorate.

Means±SD were calculated for continuous variables, and proportions were used for categorical variables. The distribution of sociodemographic and stroke/cardiovascular risk factors was evaluated in the total cohort and among the 3 race–ethnic groups. Unadjusted analyses were performed using ANOVA and linear regression models for continuous variables and the χ^2 test for categorical variables. Unadjusted comparisons of mean LVM across each SES group were performed using ANOVA. Tests for trend were performed using educational levels as ordinal variables. ANCOVA was then used to analyze the association between SES and LVM after adjusting for potential confounding demographic and medical variables, including age, gender, systolic blood pressure, diabetes, physical activity (hours per week), and body mass index (BMI). Least-square adjusted means for the covariates mentioned were computed as an estimate of the marginal means that would be obtained with a balanced population that does not differ with respect to any of the covariates. Statistical significance was determined at the $\alpha=0.05$ level using two-sided tests. Statistical analyses were conducted using PROC GLM from SAS 8.2 computer software (SAS Institute).

Results

Demographic characteristics of the study population are shown in Table 1. On average, our cohort was elderly (mean

TABLE 2. LVM by Socioeconomic Status and Race–Ethnicity

	Unadjusted Mean LVM±SD (adjusted mean LVM)*			
	Total Cohort N=1916	Whites N=377	Blacks N=417	Hispanics N=1081
Educational Level				
<High school	48.9±15.5 (48.4)	47.3±14.8 (45.3)	52.2±18.2 (50.0)	48.5±15.1 (48.3)
Completed high school	48.5±15.6 (48.6)	48.0±15.5 (45.5)	48.5±16.4 (50.2)	48.9±15.0 (49.6)
Some college	47.0±17.3 (47.1)	48.0±20.0 (46.7)	46.4±17.7 (47.7)	47.9±14.1 (48.3)
≥College graduate	44.2±14.5 (45.3)	42.3±13.5 (45.3)	46.4±16.8 (44.7)	47.2±14.4 (47.3)
P trend	<0.0001 (0.0004)	0.048 (0.86)	0.012 (0.009)	0.43 (0.47)

41 subjects are included in the total cohort who could not be classified as black, white, or Hispanic. LVM indicates left ventricular mass.

*Values in parentheses are mean left ventricular mass adjusted for age, gender, systolic blood pressure, diabetes, physical activity, and BMI.

age 68±10), mostly Hispanic, and with less than high school education. Our Hispanic population was predominantly foreign-born and consisted of 62% Dominicans, 13% Puerto Ricans, 11% Cubans, and 14% other Hispanics. There was a high prevalence of hypertension (56%) in the total cohort. Whites were significantly older. Blacks had a significantly greater percentage of female participants. Hispanics were more likely to be in the lowest SES category and least likely to be in the highest SES category. Conversely, whites were more likely to be in the highest SES category and least likely to be in the lowest category. Blacks tended to be in the mid-range category. Blacks and Hispanics had a higher prevalence of diabetes and hypertension, were less likely to be physically active, and were more likely to be overweight. LVM varied among race–ethnic groups, with blacks and Hispanics having a higher burden of LVM compared with whites (blacks 48.9±17.4 g/m$^{2.7}$, Hispanics 48.4±15.0 g/m$^{2.7}$, whites 45.6±15.0 g/m$^{2.7}$, P=0.004).

In the total cohort, using unadjusted analysis there was a significant inverse and graded association between SES and mean LVM, ie, subjects of lower SES have higher degrees of LVM. Mean LVM for those in the highest SES category was 10% lower than those in the lowest SES category (P trend<0.0001) (Table 2). On multivariate analysis, this relationship remained independent of age, gender, systolic blood pressure, diabetes, physical activity, and BMI.

On race–ethnic group comparison, using an adjusted multivariate analysis, SES was inversely associated with LVM in blacks (P trend=0.009) but not in whites (P trend=0.86) or Hispanics (P trend=0.47) (Table 2). The difference in adjusted mean LVM between the highest and lowest categories of education was 5.3 g/m$^{2.7}$ for blacks, 0.0 g/m$^{2.7}$ for whites, and 1.0 g/m$^{2.7}$ for Hispanics. This was equivalent to an 11% difference in adjusted mean LVM between the highest and lowest SES categories among blacks.

Table 3 shows the relationship between SES and LVM among the subgroup of 841 subjects without clinical hypertension (SBP was 152±21 mm Hg in hypertensives and 130±14 mm Hg in nonhypertensives; P<0.0001). Differences in unadjusted mean LVM showed that among normotensives, those in the highest SES category had an 11% lower mean LVM than those in the lowest SES category (P trend <0.0001). On race–ethnic-specific multivariate analyses, the inverse relationship between LVM and SES again remained significant only among normotensive blacks. Normotensive blacks in the lowest SES category had an 18% higher mean LVM compared with blacks in the highest category (P trend=0.006).

Discussion

The present study demonstrates that in a population-based sample of adults free of clinically overt cardiovascular disease, an independent inverse and graded relationship exists

TABLE 3. LVM by Socioeconomic Status and Race–Ethnicity Among Normotensives

	Unadjusted Mean LVM±SD (adjusted mean LVM)*			
Educational Level	Total Cohort N=841	Whites N=219	Blacks N=151	Hispanics N=445
<High school	45.1±13.2 (44.7)	46.9±14.1 (44.4)	46.2±16.9 (45.6)	44.9±12.8 (44.8)
Completed high school	45.6±15.8 (45.1)	45.5±15.6 (42.7)	46.1±17.0 (46.7)	45.5±15.0 (46.2)
Some college	41.9±14.6 (42.6)	40.5±18.3 (40.7)	41.8±13.0 (42.1)	44.7±12.9 (45.2)
≥College graduate	40.0±11.6 (41.0)	39.2±10.6 (41.3)	37.6±9.2 (37.2)	45.7±16.3 (45.1)
P trend	<0.0001 (0.0005)	0.002 (0.23)	0.01 (0.006)	0.84 (0.99)

26 subjects are included in the total cohort who could not be classified as black, white, or Hispanic. LVM indicates left ventricular mass.

*Values in parentheses are mean left ventricular mass adjusted for age, gender, systolic blood pressure, diabetes, physical activity, and BMI.

between the level of SES, as defined by educational level, and the degree of LVM among blacks. This association remained among normotensive blacks and with adjustment for systolic blood pressure. Lack of access to care or poor medical compliance may not fully explain the observed LVM–SES differences. If low SES is a marker for lack of access to care or poor compliance, then one would expect to observe SES differences in whites or Hispanics as well, which we did not find. Blacks had greater LVM than whites only for the lowest educational category, suggesting that LVM differences by race are not a biological constant. Little is known about LVM among Hispanics, although it has been suspected that Hispanics have higher LVM than whites.[19] Our study confirms that Hispanics carry a higher burden of increased LVM than whites at a level similar to that of blacks.

Explanations for the greater LVM differences among blacks remain speculative, but several different factors may play a role. Sympathetic stimulation is one mechanism through which low SES could be associated with greater LVM. Sympathetic stimulation has been shown to vary with environmental exposures, such as job stress and lower SES.[12,14] Evidence suggests that lower SES is associated with a disproportionate cumulative burden of stressful life conditions.[12–15] Chronic intermittent adrenergic stimulation, in the absence of overt hypertension, can cause increased LVM, and the presence of increased LVM may precede the onset of clinically overt hypertension.[20–22] Lower SES, particularly among blacks, may be associated with increased psychosocial stress and adrenergic stimulation, both of which may produce increased LVM. Being black and of low SES may be a different experience than being white or Hispanic of low SES. Low SES blacks, for example, may be subjected to greater stress producing experiences than low SES whites, resulting in stronger SES patterning of LVM among blacks than among whites. Increased adrenergic receptor sensitivity to norepinephrine infusion has been shown in blacks compared with white hypertensive subjects. These pathways may be either more sensitive (requiring a lower threshold to "trigger") or more responsive (have a higher "gain") or both among blacks leading to increased LVM. Thus, blacks of lower SES may carry a higher sympathetic tone than whites or Hispanics of lower SES, and their LVM may also be more responsive to its effects. Regardless of the mechanisms involved, our observations raise the clinical issue of whether blacks of lower SES require more careful assessment for prehypertension/hypertension and subsequent target organ damage.[23]

SES may also be related to dietary behaviors. Studies have suggested that blacks retain sodium more avidly than whites,[24] and increased sodium retention is associated with increased LVM by way of mechanisms involving circulating volume expansion and possibly activation of the renin-angiotensin system.[25] Dietary differences (in the intake of sodium for example) may be greater by SES among blacks than among whites and could also contribute to the greater LVM differences by SES observed in blacks.

Conventional clinical assessment of blood pressure may underestimate the severity and duration of high blood pressure, particularly among subjects of lower SES. Greater LVM among blacks compared with whites is unexplained by differences either in rest or in mean daytime blood pressure.[26] However, ambulatory blood pressure that remains elevated at night (non-dipping) rather than exhibiting its normal nocturnal decrease is a strong correlate for the presence of increased LVM and predictive of the LVM differences between blacks and whites.[27] Because nondipping status is related to sympathetic tone, and because blacks are more likely to be nondippers,[28,29] ambulatory blood pressure measures and diurnal variation may better explain race–ethnic and socioeconomic differences in LVM. Ambulatory blood pressure may also explain the observed LVM–SES relation among normotensives. Patients with a normal clinic blood pressure may show abnormalities on ambulatory blood pressure monitoring. This phenomenon of "masked hypertension" may have more clinical relevance because ambulatory blood pressure has a stronger impact than resting blood pressure on end-organ damage and cardiovascular outcomes.[6] The development of hypertension may be less well recognized or the severity of hypertension underestimated among blacks of lower SES when compared with their white or Hispanic counterparts in an urban community. These issues warrant further investigation.

Important strengths of this study are its population-based nature and the presence of a tri-ethnic sample. Several limitations exist. First is the lack of variability in educational level observed among Hispanics (nearly 80% were in the lowest educational category), which may have limited our ability to detect SES effects in this group. Income data were not available in our cohort. Educational level was used as a proxy for SES and remains a reasonable but limited measure of SES.[7] Educational level may not be a good predictor of SES among an immigrant population, possibly explaining the lack of an association between SES and LVM among our predominantly immigrant Hispanic cohort. Finally, at a given educational level, race–ethnic groups may differ in terms of wealth, buying power, living conditions, or access to resources in ways that could not be fully measured.

Perspectives

This study is the first, to our knowledge, to demonstrate that lower SES is an independent predictor of increased LVM among hypertensive and normotensive blacks. Although our results do not establish a causative role for SES in the pathogenesis of increased LVM, they do suggest that a link between SES and LVM exists and open important avenues for further research. It is possible that conditions of lower SES more adversely affect blacks in terms of increased LVM and its sequelae. Thus, this group may warrant more aggressive cardiovascular risk monitoring and intervention.

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