Demographics and Correlates of Five-Year Change in Echocardiographic Left Ventricular Mass in Young Black and White Adult Men and Women: The Coronary Artery Risk Development in Young Adults (CARDIA) Study

Julius M. Gardin, MD,* Debra Brunner, MA,† Pamela J. Schreiner, PhD,‡ Xiaoyuan Xie, MD,§ Cheryl L. Reid, MD,* Karen Ruth, MS,§ Diane E. Bild, MD, PhD, MPH,¶ Samuel S. Gidding, MD¶

Irvine, California; Minneapolis, Minnesota; Chicago, Illinois; Bethesda, Maryland; and Wilmington, Delaware

OBJECTIVES

The goal of this study was to determine the presence and correlates of change (Δ) in left ventricular (LV) mass by echocardiography in young adults.

BACKGROUND

Left ventricular mass is known to be a powerful independent predictor for cardiovascular disease events in adults. However, little is known about Δ in LV mass over time in young adults.

METHODS

Coronary Artery Risk Development in Young Adults (CARDIA) is a multicenter, longitudinal, population-based study of black and white men and women who were ages 23 to 35 at the time of their initial two-dimensionally directed M-mode echocardiography exam (year 5); half the cohort had a repeat echocardiography exam five years later (year 10). Data were analyzed from 1,189 participants who had paired echocardiography studies. To minimize reader variability, blinded measurements on initial and repeat echocardiography were performed nearly contemporaneously by the same reader.

RESULTS

In multilinear regression analyses, significant (p < 0.05) predictors of year 10 two-dimensional guided M-mode LV mass included initial LV mass, initial body mass index (BMI) and change in BMI for all race/gender subgroups. Initial systolic blood pressure (SBP) was a significant predictor of year 10 LV mass in white men and black women; change in SBP was significant in black women with a trend towards significance in white women. Left ventricular mass remained constant in all race/gender subgroups, except black women, where it increased (by 5.9 g [mean]). Black women also had the largest increases in BMI and SBP. In black women, a five-year weight gain of 20 pounds and a 15-mm Hg increase in SBP would be expected to be associated with a 9% to 12% increase in LV mass.

CONCLUSIONS

Particularly in black women, weight and blood pressure control may be important community health and treatment goals to prevent LV hypertrophy. (J Am Coll Cardiol 2002;40:529–35) © 2002 by the American College of Cardiology Foundation

Left ventricular (LV) mass, as determined by M-mode echocardiography, is known to be a powerful independent predictor for cardiovascular disease (CVD) morbidity and mortality in older adults (1,2). Left ventricular mass has been positively associated with multiple factors, including age, male gender, black race, height, weight and arterial blood pressure (BP) (3–7). Whereas longitudinal data on LV mass exist for adolescents (8,9), little is known about the correlates of change (Δ) in LV mass over time in young adults.

The purpose of this study was to examine the correlates of five-year Δ echocardiography LV mass in the Coronary Artery Risk Development in Young Adults (CARDIA) cohort, a National Heart, Lung, and Blood Institute-sponsored, prospective multicenter study of development of CVD risk factors in a biracial cohort. The hypothesis was that LV mass at year 10 would be associated with initial LV mass, race, gender, BP, physical activity and body mass index (BMI). Additional analyses examined: 1) Δ LV mass as associated with Δ in BMI, systolic blood pressure (SBP), physical activity, cigarette use and alcohol use, and 2) change in CVD risk status as related to five categories of change in LV mass, ranging from a >20% decrease to a >20% increase.

METHODS

Subjects. Initially, 5,115 participants who were 18 years old to 30 years old at the time of enrollment (1985 to 1986) were recruited and examined at four CARDIA field centers, located in Birmingham, Alabama; Chicago, Illinois; Min-
neapolit, Minnesota; and Oakland, California. The overall design and objectives of the CARDIA study have been presented elsewhere (10). In 1990 to 1991, during year 5 of the CARDIA study, a total of 4,243 participants underwent two-dimensionally (2D) directed M-mode echocardiograms at the four field centers using a previously described protocol for multicenter studies (6,7,11). During the CARDIA year 10 (1995 to 1996) examination, echocardiography studies were repeated at the Chicago and Minneapolis field centers.

Of 1,536 participants who had year 5 and 10 echocardiograms at the two field centers, a final cohort of 1,189 (206 black men, 338 white men, 277 black women and 368 white women) are included in these analyses. Exclusions included characteristics that could affect serial assessment of LV mass (pregnancy or taking antihypertensive medication, n = 75), missing data (for one or more independent variables or missing data for LV mass calculation, n = 154) or poor echocardiogram LV quality scores (n = 263). One other individual was excluded because of a gender change.

The final cohort was compared with the remainder of the year 5 CARDIA cohort for comparability in three ways: those who did not return for year 10 follow-up, those from centers that did not have repeat echocardiography exams and those who had poor echocardiogram quality scores. Participants who were pregnant or taking antihypertensive medications were excluded, and p = 0.05 determined significant differences between the final cohort and others. Those without a year 10 echocardiography exam had higher BP (difference of 2.7 mm Hg for SBP, 2.9 mm Hg for diastolic blood pressure [DBP]), were slightly younger (0.3 years), had attained slightly lower levels of education (0.2 years) and were somewhat heavier smokers (1.3 cigarettes/day), on average, than the final cohort. Participants from Birmingham and Oakland—those who did not have a year 10 echocardiography exam—had higher BP (2.5 mm Hg for SBP, 4.3 mm Hg for DBP), BMI (0.6 kg/m²) and higher education level (0.4 years), shorter stature (0.5 cm), lower activity levels (32 exercise units [EU]) and reported lower alcohol intake (1.8 ml/day) and lower cigarette use (0.8 cigarettes/day) than the final cohort. Those who had poorer quality echocardiograms had higher body weight (9 lbs), BMI (1.7 kg/m²), higher DBP (1.3 mm Hg), higher cigarette use (1.8 cigarettes/day), shorter stature (0.7 cm) and lower education level (0.4 years). For other study variables, the final study cohort was comparable to those who did not participate (p > 0.05).

**Echocardiography.** Two-dimensional, M-mode (2D-guided) and Doppler echocardiograms were recorded onto super-VHS tape at each field center using a standardized protocol (7,11). Videotapes were sent to a central reading center (at University of California, Irvine) where measurements were made on an off-line analysis system and entered directly into a database. All studies were read by experienced echocardiographic sonographers who had undergone standardized training. Discrepancies in blind duplicate readings were adjudicated by one of two experienced physician-echocardiographers (J.M.G., C.L.R.) (7,11). To reduce interreader differences (including temporal drift) and to eliminate possible bias due to different reading systems, year 5 echocardiograms were reread in year 10 nearly contemporaneously with the year 10 echocardiograms using the same Freeland-TomTec off-line systems (Indianapolis, Indiana). Measurements from year 5 and year 10 studies, both made in year 10, served as the basis of the analyses presented in this report. Readers were blinded as to the year of recording, and demographic and other identifiers associated with each echocardiogram.

Quality control measures included standardized training of echocardiogram technicians and readers, technician observation by trained echocardiographers, periodic reader review sessions, phantom studies on ultrasound equipment, quality control audits and blind duplicate readings to establish inter- and intrareader measurement variability and technician recording variability (7,11). Coefficients of variation for measurement of LV mass were 10.6% for interreader, 9.5% for intrareader, 13.5% for intratechnician and 10% for intertechnician.

This report focuses on five-year change in 2-D guided M-mode echocardiogram measurements of LV mass (Δ LV mass). M-mode measurements were made according to the American Society of Echocardiography convention (12) and LV mass derived from the necropsy-validated formula described by Devereux et al. (13):

\[
\text{LV mass (g)} = 0.80 \times 1.04 \left( (\text{VSTd} + \text{LVIDd} + \text{PWTd})^3 \right) - (\text{LVIDd})^3 + 0.6
\]

where VSTd is ventricular septal thickness at end-diastole (cm), LVIDd is LV internal dimension at end-diastole (cm) and PWTd is LV posterior wall thickness at end-diastole (cm).

**Covariates analyzed.** Independent variables included in this analysis were: 1) race, 2) gender, 3) age (in years), 4) initial systolic and diastolic BP, measured at year 5 (in mm Hg), 5) five-year change in SBP (in mm Hg), 6) initial BMI (i.e., weight/height² in kg/m²) (10), 7) five-year change in BMI (kg/m²), 8) initial LV mass (in grams), 9) initial physical activity (in units)—the total of the heavy and moderate intensity scores calculated from responses to the
CARDIA physical activity questionnaire in year 5, a modification of the Minnesota leisure time questionnaire, which included intensity and duration of various activities (14), but not occupational physical activity, 10) five-year change in physical activity, 11) initial cigarette smoking level (cigarettes/day), 12) five-year change in smoking level, 13) education level (years), 14) alcohol use, calculated from a formula to provide total alcohol consumption in ml/day, and 15) five-year change in alcohol use. Change variables were calculated as year 10/year 5 measurements. The independent variables were selected a priori based on previous CARDIA and other studies (1,6,7,9,14).

Pulse pressure was initially considered for inclusion among independent variables. Pulse pressure and pulse pressure were significantly related to LV mass for black women. However, with the inclusion of systolic BP and systolic BP, the associations were no longer significant. We also examined other measures of obesity—including ponderal index, subscapular skin folds and waist-to-hip ratio—but no measure explained more of the variance in LV mass than did BMI.

Statistical analyses. Univariate descriptive statistics were computed for year 5 LV mass, year 10 LV mass, Δ LV mass and the independent variables. Bivariate Pearson correlation coefficients were computed between year 10 LV mass and Δ LV mass for each of the predictor variables. Multiple linear regression was used to estimate the effect of each predictor variable adjusted for the other predictors. Squared partial correlation coefficients were calculated to determine the proportion of variance in year 10 LV mass explained by each independent variable, controlling for the other variables. The total adjusted R-square is the proportion of variance explained by the full model, adjusted for degrees of freedom. Percent change in LV mass was determined as 100 × [(year 10 LV mass) − (year 5 LV mass)]/(year 5 LV mass). Trends were based on linear regression with equal spacing of the five Δ groups.

RESULTS
Year 5 and year 10 LV mass, and five-year Δ in LV mass by race/gender subgroup. Figure 1 displays data for year 5 LV mass and five-year change in LV mass, by race/gender subgroup. The Δ LV mass was significant only in black women, with a mean increase of 5.9 g (5.8%, p < 0.0001). Differences within gender and within race for unadjusted LV mass were significant (p < 0.05), with blacks having a higher average Δ LV mass than whites and women higher than men (data not shown). These differences were predominantly driven by Δ in black women.

Correlation and multivariate linear regression analyses of year 10 LV mass by race/gender subgroup. Table 1 displays the mean and SD for age, BMI, weight, SBP, DBP and physical activity at year 5, year 10 and for the Δ over time (year 10 − year 5), as well as educational status during year 5. Black women had the greatest five-year Δ BMI (increase of 1.7 kg/m²) and Δ SBP (increase of 3.9 mm Hg). Whites were more highly educated and had greater declines in physical activity. Cigarette smoking and alcohol use varied substantially by race and gender (data not shown).

Bivariate Pearson correlations between year 10 LV mass and each covariate were computed and are included in Table...
2. Initial LV mass was the strongest bivariate correlate of year 10 LV mass for all race/gender groups (range of $r = 0.72$ to 0.78). Initial BMI was a significant correlate of year 10 LV mass in all subgroups (range of $r = 0.40$ to 0.51). Initial SBP was also a significant correlate of year 10 LV mass for each subgroup, particularly for black women ($r = 0.29$). Other covariates were inconsistently related to year 10 LV mass across race/gender subgroups. The five-year BMI changes were significantly correlated with year 10 LV mass only in whites, while five-year systolic BP changes were related to year 10 LV mass only in women. Age was a significant correlate of year 10 LV mass in black men only.

Table 2. Bivariate Pearson Correlations of Year 10 LV Mass (g)
With Predictor Variables by Race/Gender Group

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Black Men</th>
<th>White Men</th>
<th>Black Women</th>
<th>White Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean  SD</td>
<td>Mean  SD</td>
<td>Mean  SD</td>
<td>Mean  SD</td>
</tr>
<tr>
<td>Yr 5 LV mass</td>
<td>0.72§</td>
<td>0.78§</td>
<td>0.78§</td>
<td>0.74§</td>
</tr>
<tr>
<td>Yr 5 BMI</td>
<td>0.50§</td>
<td>0.40§</td>
<td>0.51§</td>
<td>0.43§</td>
</tr>
<tr>
<td>BMI change</td>
<td>0.06</td>
<td>0.12†</td>
<td>0.06</td>
<td>0.21§</td>
</tr>
<tr>
<td>Yr 5 SBP</td>
<td>0.18†</td>
<td>0.19§</td>
<td>0.29§</td>
<td>0.14‡</td>
</tr>
<tr>
<td>SBP change</td>
<td>0.12*</td>
<td>−0.01</td>
<td>0.15†</td>
<td>0.16†</td>
</tr>
<tr>
<td>Yr 5 activity</td>
<td>0.13*</td>
<td>0.11†</td>
<td>0.02</td>
<td>0.04</td>
</tr>
<tr>
<td>Activity change</td>
<td>−0.05</td>
<td>0.07</td>
<td>0.05</td>
<td>−0.01</td>
</tr>
<tr>
<td>Yr 5 alcohol ml/day</td>
<td>0.07</td>
<td>0.05</td>
<td>−0.06</td>
<td>0.01</td>
</tr>
<tr>
<td>Alcohol change</td>
<td>−0.01</td>
<td>−0.03</td>
<td>0.12*</td>
<td>0.02</td>
</tr>
<tr>
<td>Yr 5 cig/day</td>
<td>−0.08</td>
<td>−0.01</td>
<td>0.01</td>
<td>0.12‡</td>
</tr>
<tr>
<td>Cig/day change</td>
<td>0.08</td>
<td>0.05</td>
<td>−0.05</td>
<td>−0.02</td>
</tr>
<tr>
<td>Yr 5 age</td>
<td>0.26§</td>
<td>−0.00</td>
<td>0.04</td>
<td>0.06</td>
</tr>
<tr>
<td>Yr 5 education</td>
<td>0.12*</td>
<td>0.02</td>
<td>−0.09</td>
<td>−0.07</td>
</tr>
</tbody>
</table>

BMI = body mass index; cig = cigarettes; LV = left ventricular.

Associations of the covariates with year 10 LV mass adjusted for each other were examined using multiple linear regression. The range of total $R^2$ for the multivariate model within each race/gender subgroup indicated that between 58% and 65% of the total variation in year 10 LV mass was explained by the covariates (Table 3). In all race/gender subgroups, initial LV mass was, by far, the strongest predictor of year 10 LV mass (squared partial correlation =

Table 3. Multiple Linear Regression Coefficients Relating Year 10 LV Mass to Predictor Variables, by Race/Gender Group

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Black Men</th>
<th>White Men</th>
<th>Black Women</th>
<th>White Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean  SD</td>
<td>Mean  SD</td>
<td>Mean  SD</td>
<td>Mean  SD</td>
</tr>
<tr>
<td>LV mass (g), yr 5</td>
<td>0.63§</td>
<td>0.74§</td>
<td>0.83§</td>
<td>0.71§</td>
</tr>
<tr>
<td>BMI (kg/m²), yr 5</td>
<td>1.97§</td>
<td>0.85†</td>
<td>0.45†</td>
<td>0.79§</td>
</tr>
<tr>
<td>BMI change (kg/m²)</td>
<td>1.67†</td>
<td>1.79‡</td>
<td>1.83§</td>
<td>1.35§</td>
</tr>
<tr>
<td>SBP (mm Hg), yr 5</td>
<td>0.17</td>
<td>0.35‡</td>
<td>0.42‡</td>
<td>0.14</td>
</tr>
<tr>
<td>SBP change (mm Hg)</td>
<td>0.26</td>
<td>0.08</td>
<td>0.38‡</td>
<td>0.26*</td>
</tr>
<tr>
<td>Activity (EU), yr 5</td>
<td>0.00</td>
<td>0.01†</td>
<td>0.01</td>
<td>0.00</td>
</tr>
<tr>
<td>Activity change (EU)</td>
<td>0.01</td>
<td>0.01†</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Age (yrs), yr 5</td>
<td>1.59‡</td>
<td>0.20</td>
<td>−0.36</td>
<td>0.00</td>
</tr>
<tr>
<td>Education, yr 5</td>
<td>0.10</td>
<td>−0.39</td>
<td>0.24</td>
<td>0.64</td>
</tr>
<tr>
<td>Cig use (cig/day), yr 5</td>
<td>−0.09</td>
<td>0.00</td>
<td>0.11</td>
<td>−0.01</td>
</tr>
<tr>
<td>Cig use change (cig/day)</td>
<td>0.39</td>
<td>−0.18</td>
<td>0.11</td>
<td>−0.46*</td>
</tr>
<tr>
<td>Alcohol (ml/day), yr 5</td>
<td>0.02</td>
<td>−0.13*</td>
<td>−0.02</td>
<td>0.06</td>
</tr>
<tr>
<td>Alcohol change (ml/day)</td>
<td>−0.01</td>
<td>−0.05</td>
<td>−0.04</td>
<td>−0.03</td>
</tr>
<tr>
<td>Total $R^2$ for model</td>
<td>0.58</td>
<td>0.63</td>
<td>0.65</td>
<td>0.58</td>
</tr>
</tbody>
</table>

* $p < 0.10; † p < 0.05; ‡ p < 0.01; § p < 0.001$. Each column above reflects a separate multiple linear regression analysis. BMI = body mass index; cig = cigarettes; EU = exercise units; LV = left ventricular; SBP = systolic blood pressure.
0.37 for black men, 0.53 for white men, 0.48 for black women and 0.45 for white women, all \( p < 0.0001 \).

Multiple linear regression results (Table 3) further indicate that initial BMI was a significant predictor of year 10 LV mass in all subgroups. However, there were differences in the magnitude of the relation. The regression coefficient for year 5 BMI was 1.97 for black men compared with 0.45 for black women (\( p = 0.02 \) for differences in coefficient); predictor differences were similar, but not as strong, in whites. The \( \Delta \) BMI was a significant predictor in all subgroups. Initial SBP and \( \Delta \) SBP were strong predictors of year 10 LV mass in black women; initial SBP was significant for white men, whereas \( \Delta \) SBP trended towards significance for white women. For other variables, either no significant correlations were identified or inconsistent trends among the race/gender subgroups were observed.

**Additional longitudinal analyses.** Three additional analyses were performed to further examine the longitudinal relation of risk factor covariates to LV mass. First, the relation of \( \Delta \) LV mass with \( \Delta \) BMI, \( \Delta \) SBP, \( \Delta \) physical activity, \( \Delta \) cigarette use and \( \Delta \) alcohol use was examined in each race/gender subgroup. The \( \Delta \) LV mass was positively correlated with \( \Delta \) BMI in all subgroups—most strongly in black women (range of \( r = 0.14 \) to 0.29, \( p < 0.05 \) to 0.001). The \( \Delta \) LV mass was also positively correlated with \( \Delta \) SBP in black women (\( r = 0.16, p < 0.01 \)) and white women (\( r = 0.13, p < 0.05 \)) and negatively correlated with cigarettes/day in white men and women (\( r = -0.11 \) and \( -0.12 \), both \( p < 0.05 \)).

In the second analysis, the entire cohort was stratified into three groups, i.e., those with a significant increase, no change or a significant decrease in LV mass. Table 4 displays the relation of increases (\( >20\% \) and \( 10\% \) to \( 20\% \)), no change (i.e., within \( 10\% \)) and decreases (\( >20\% \) and \( 10\% \) to \( 20\% \)) in LV mass to covariates. A significant trend toward increasing BMI, SBP and weight over five years was observed in those whose LV mass decreased versus increased.

Finally, to assess the impact of regression to the mean, \( \Delta \) LV mass was adjusted for initial (year 5) and five-year change covariates in participants with low (lower quartile), average (second and third quartiles) and high (upper quartile) measurements of initial LV mass, adjusting for initial age, race and gender. Participants with low initial LV mass measurement showed a significant five-year increase (mean = 7.31 g) in LV mass (\( p < 0.0001 \)), whereas participants with high initial LV mass measurements showed a significant decrease (mean = 8.05 g) in LV mass (\( p < 0.0001 \)). In the middle reference group, participants had a relatively small increase (mean = 2.55 g) in LV mass (\( p = 0.004 \)). These data suggest regression to the mean partially confounded longitudinal analysis, particularly that presented in Table 4.

**DISCUSSION**

The unadjusted five-year mean \( \Delta \) (increase) in echocardiography LV mass in black women was 5.9 g; changes in LV mass were smaller (<1.7 g) and not statistically significant among black men, white men and white women. For all four race/gender subgroups, the most important predictors of year 10 LV mass were initial LV mass and initial BMI. For both white and black women, \( \Delta \) SBP was a significant predictor of year 10 LV mass, whereas initial SBP was a predictor in black women. The above variables explained 58% to 65% of the variation in LV mass, largely because initial LV mass accounted for 37% to 53% of the variance,
with risk factors accounting for 10% to 21% of the variation. Given that the coefficient of variation for intrareader measurements of LV mass in CARDIA is in the range of 10% to 12% (7), the unmeasured component of variation is small. Thus, our findings suggest that LV mass is dependent on body size and blood pressure, which are, in turn, likely influenced by a combination of environmental and genetic factors (15). In particular, changes in body size may alter intravascular blood volume, thus contributing to the observed change in mass. These CARDIA findings are consistent with important relations of body size and blood pressure to LV mass reported in serial assessments in healthy adolescents (8,9,16). Although we were unable to assess the role of familial or genetic traits, these factors may contribute to the remainder of the observed variance in five-year \( \Delta \) LV mass.

This study suggests that, for most healthy young adults, there is little change in LV mass once somatic growth is complete. Well over 50% of the cohort had a \( \Delta \) LV mass over the five-year interval that was within the coefficient of variation of LV mass measurement. This is in agreement with the fact that, as a whole, the cohort experienced little change in BP and, with the exception of black women, modest increases in body weight.

The current report on five-year \( \Delta \) echocardiography LV mass from the CARDIA study is unique in a number of respects. First, data are reported from a large multicenter study of young adults, approximately half of whom are Caucasian and the other half African American, while half are men and half are women. Second, two echocardiograms were obtained separated by a five-year interval, but measured nearly contemporaneously by the same readers, in order to minimize effects of possible temporal drift in reading approach and interreader variability (17). This reading methodology greatly minimized interreader and temporal drift.

**BP and LV mass.** Blood pressure within the normal range appears to explain a relatively small proportion of the observed variance in LV mass (5,7–9,16). Regardless of whether one studies single BP measurements or averages of BP over time, correlations of resting BP measurements with LV mass appear to be in the range of \( r \leq 0.30 \) (18–20). Because BP changes in individuals are small over five-year intervals, the full impact of BP on LV mass may be expressed over a lifetime. Published data suggesting that hypertension predicts development of LV hypertrophy, and vice versa, raises the possibility that both hypertension and LV mass are associated with common antecedent factors, e.g., genetic influences (20,21). A role for genetic factors is further suggested by other studies (19,20). Of note, hypertension was uncommon among CARDIA participants. Specifically, hypertension—defined as SBP \( \geq 140 \) mm Hg, or DBP \( \geq 90 \) mm Hg, or taking antihypertensive medications—was present in 1.6% of the cohort during year 5 (3.1% of blacks, 0.6% of whites) and 3.8% during year 10 (7.0% of blacks, 1.6% of whites). None of the participants in this cohort had a BP \( \geq 160/100 \) mm Hg. Individuals on antihypertensive therapy were excluded from this study because of the potential confounding effect of treatment.

A few studies have demonstrated apparent regression of LV mass after treatment with antihypertensive medications (22,23). Of considerable interest, there is preliminary evidence that regression of electrocardiographic LV hypertrophy and echocardiography LV mass are associated with improvement in survival (24,25). Positive effects on LV function are associated with improvement of both LV geometry and mass reduction (26). However, whether regression in LV mass is a direct consequence of hypertension treatment, or an independent predictor of improved prognosis, remains an area of active investigation (27).

**Obesity and LV mass.** Evidence of the relation between obesity and echocardiography LV mass has been reported in a small controlled, randomized trial of the effects of weight reduction on echocardiogram measurements in overweight patients with hypertension (28). In a report from Framingham, hypertension and obesity each had independent associations with LV mass and wall thickness, and the strengths of association were additive, but not multiplicative (29). Relations between body size and LV mass are complex with separate relations for lean body mass and fat mass (30,31). Obesity may contribute more to LV mass by increasing LV volume than by increasing wall thickness.

**Study limitations.** In this study “regression to the mean” is a potential contributor to the observed five-year \( \Delta \) LV mass (32–34). Individuals with the largest values for mass at year 5 tended to have lower values at follow-up; conversely, individuals with the lowest values at year 5 tended to have increases in LV mass. A recent study that compared echocardiograms done within one to two months of each other showed relatively good correlation of measurements and suggested an LV mass decrease of 6.8% associated with regression to the mean, slightly larger than the value of 6.2 g (4% to 5% depending on gender) found in this study (32,33). Regression to the mean may have important clinical significance, e.g., in echocardiography studies designed to study potential regression of LV mass in response to medication or other therapeutic interventions. A change in LV mass of 17 g has 80% confidence and a change in LV mass of 35 g has 95% confidence of being clinically significant (32). Another limitation is that the study cohort had slightly lower BP than the unexamined portion of the cohort. Because the analysis was longitudinal, it is unclear whether this impacted analyses. However, this data set may underestimate the prevalence of LV hypertrophy in the general population.

**Implications in young black women.** Of all race/gender groups in the CARDIA cohort, black women demonstrated the greatest five-year \( \Delta \) LV mass. This increase in LV mass was positively associated with the greatest five-year \( \Delta \) BMI and \( \Delta \) SBP. Thus, sequelae of the five-year \( \Delta \) in BMI and SBP may be particularly important in black women (35). For example, if we consider a hypothetical black woman
who had gained 20 pounds (Δ BMI = + 3.42 kg/m²) over the five-year period, our data predict a five-year Δ LV mass of +6.3 g. If, in addition, she demonstrated a five-year Δ SBP of +15 mm Hg, Δ LV mass would be expected to increase by an additional 5.7 g. Because the 25th to 75th percentile for LV mass in black women is 99 g to 136 g, this black woman’s Δ LV mass of 12 g represents a 9% to 12% increase over five years. Particularly for young black women, weight and BP control should be important treatment goals for primary care physicians (including obstetricians) and community-based programs.

Reprint requests and correspondence: Dr. Julius M. Gardin, Division of Cardiology, St. John Hospital & Medical Center, 22201 Moross Road, PBII, Suite 470, Detroit, Michigan 48236. E-mail: Julius.gardin@stjohn.org.

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