Lack of Independent Relationships Between Left Ventricular Mass and Cardiovascular Reactivity to Physical and Psychological Stress in the Coronary Artery Risk Development in Young Adults (CARDIA) Study

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The objective of this study was to determine whether exaggerated blood pressure (BP) reactivity to stress and psychosocial characteristics are related to left ventricular mass (LVM) in a large cohort of young adults. Analyses were conducted with 3,742 participants of the CARDIA study (945 white men, 1,024 white women, 781 black men, and 993 black women), evaluated in 1990 to 1991 with echocardiographic measurement of LVM. Analyses were stratified by gender and race. The relationships of LVM/height$^2$ and cardiovascular reactivity to physical and psychological stressors (treadmill exercise, cold pressor, video game, and star-tracing tasks), were examined in both univariate and multivariate analyses adjusting for baseline BP, weight, and other relevant biobehavioral variables.

The relationships between LVM and several psychosocial characteristics (hostility, anger suppression, anxiety, depressive symptoms, and education) were also assessed.

Systolic blood pressure (SBP) reactivity to exercise was significantly related to LVM in black and white men; LVM was 10% greater among white men with exaggerated (upper quintile) peak exercise SBP than among other white men. SBP reactivity to the cold pressor test was related to LVM in all race/gender groups, although the relationship remained significant only among white men and women in the multivariate analysis. Diastolic blood pressure (DBP) reactivity to the video game was related to LVM only among black men in adjusted analyses. After adjusting for resting BP, weight, and other covariates in linear multiple regression models, SBP reactivity to exercise explained only 3% of the variance in LVM among white men. Otherwise, reactivity to other stressors or psychosocial variables accounted for no more than 1% of the variance in LVM.

It was concluded that among a cohort of young adults, blood pressure reactivity to physical and mental stressors did not add substantially to the prediction of LVM when resting BP, weight, and other covariates were taken into account. Am J Hypertens 1996;9:915–923

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Increased left ventricular mass (LVM) has recently emerged as an independent risk factor for cardiovascular disease (CVD). It has also been hypothesized that increased LVM may either contribute to the development of hypertension or predict its development. It is therefore important to determine how increased LVM evolves over time. Studies have shown that resting systolic blood pressure (SBP) is related to LVM, along with several other factors including obesity, age, and possibly alcohol consumption.

Other factors that may also be related to increased LVM include blood pressure reactivity to stress and certain psychosocial characteristics. Several small studies have demonstrated a significant relationship between exaggerated systolic blood pressure (SBP) reactivity during exercise and LVM. However, a larger study found that this relationship is at best modest when adjusting for resting BP, age, and body mass. Two recent studies have shown that blood pressure reactivity to other stressors, such as mental arithmetic and cold pressor stimulation, are associated with increased LVM, but these studies did not adequately control for resting SBP and other potential confounding factors. In another study, LVM was shown to be related to job strain. Other psychosocial factors that have been associated with CVD and/or hypertension, such as depression, hostility, and anxiety, may also be related to LVM. However, this possibility has not been explored to date.

The Coronary Artery Risk Development in Young Adults (CARDIA) study provides an opportunity to assess the relationships between LVM and both reactivity and psychosocial characteristics in a large, population-based sample that is approximately balanced by gender and race (black and white). In addition to obtaining echocardiographic measures of LVM, the CARDIA study has measured blood pressure reactivity to exercise, blood pressure reactivity to psychological stressors, and psychosocial factors (trait anxiety, hostility, anger suppression, and depression). The purpose of the present study was to examine the independent relationships between LVM and these factors.

METHODS

Subjects The CARDIA study is a longitudinal epidemiologic study designed to determine the precursors of CVD risk factors in young adults and to determine the relationships between behavioral factors and changes in CVD risk factors over time. The subjects for CARDIA were initially recruited in 1985 and 1986 from four U.S. cities: Birmingham, AL; Chicago, IL; Minneapolis, MN; and Oakland, CA. A total of 5,115 young adults between the age of 18 and 30 years were recruited, approximately balanced for age (18 to 24 and 25 to 30 years), sex (race: black and white), and educational level (high school graduate or less, and beyond high school). The study design and baseline characteristics of the participants have been previously described in detail. The first and second examinations for the study were performed in 1985 to 1986 and 1987 to 1988, respectively.

Echocardiography was performed at the third examination, 5 years after initial recruitment (1990 to 1991). A total of 85% of the initial cohort (4352) returned for this examination. Of these, 610 were excluded from the present analysis for the following reasons: 211 due to missing LVM data; 147 due to pregnancy at any examination; 183 due to non-attendance at the second examination; and 69 due to use of medications for hypertension. These exclusions left 3742 participants for analysis, including 945 white men, 1024 white women, 781 black men, and 992 black women. For all examinations, the study was approved by the institutional review boards for each of the four testing centers, and participants gave informed consent.

Data Collection Due to the design of the CARDIA study, not all variables of interest were measured concurrently. For the present analysis, data were taken from the first three examinations: exercise testing from the first examination, cardiovascular reactivity to psychological stress at the second examination, and echocardiography, psychosocial measures, and covariates at the third examination. Symptom-limited graded exercise treadmill testing was performed according to a standard protocol. The testing consisted of nine increasingly difficult 2-min stages. Blood pressure and heart rate were obtained immediately prior to the test, at the end of each stage, at maximum exercise, and every minute for 3 min after exercise. Testing procedures for cardiovascular reactivity to psychological stress involved three different tasks: Atari Breakout (video) game, a mirror-image star-tracing task, and a cold pressor task (hand immersion technique). These tasks have been commonly used in studies of cardiovascular reactivity. An 8-min habituation/baseline period was followed by presentation of the video game and star-tracing task in randomized order for 3 min each. The cold pressor task, consisting of approximately 30 s of hand immersion in a cold water tank, followed the other two tasks. Time intervals between tasks consisted of 2 min for recovery readings and approximately 1 min for repositioning for the next task. Blood pressure was measured at 1-min intervals during the last 4 min of the 8-min habituation/baseline period, and once during each min of the video game and star-tracing tasks. Only one blood pressure reading was taken during the cold pressor task. Blood pressures...
and heart rates for the habituation/baseline period, 
video game, and the star-tracing task were recorded 
with an automated blood pressure monitor (2600B 
Vita-Stat, SpaceLabs Inc., Redmond, WA). Blood 
presures during cold pressor were recorded manu-
ally with a mercury sphygmomanometer by trained 
and certified technicians, so as to minimize the dur-
ation of participants’ hand immersion. Cuff infla-
tion during the cold pressor began after 30 sec of im-
ersion, and the participant’s hand was removed imme-
diately following completion of cuff deflation. Stan-
dardization of the reactivity protocol was accom-
plished by centralized training of technicians at all 
four centers, quality assurance site visits, and the 
use of audio tapes for instructions to participants. 
Automated blood pressure monitors were calibrated 
weekly.

Echocardiographic techniques have been de-
scribed previously. M-Mode and 2-D echocardi-
ograms were obtained with participants in the left 
lateral decubitus position. Recordings were per-
formed with respiration suspended at mid-expira-
tion.supervideo tape recordings were sent to the 
Echocardiographic Reading Center at the University 
of California at Irvine for centralized interpretation. 
Measurements were made from digitized images 
using a Dextra D-200 offline image analysis system 
(Dextra Medical, Lakewood, CA). Quality control 
measures for the center were extensive. M-mode 
LVM was calculated using the following formula:

\[ LVM (g) = 0.8 \times [ (VSTd + LVIDd + PWTd)^3 - (LVIDd)^3] + 0.6 \]

where VSTd = ventricular septal thickness at end-
diastole in centimeters, LVIDd = LV internal dimen-
sion at end-diastole in centimeters, and PWTd = pos-
terior wall thickness at end-diastole in centimeters, 
all measured using the American Society of Echocar-
diography convention.

Psychological assessment was performed using 
well standardized measures of relevant personality 
traits. Questionnaires included the Cook-Medley Hos-
tility subscale of the MMPI,28 the Spielberger Anger-
in subscale designed to assess anger reactivity,29 
the Spielberger Trait Anxiety scale,30 and the CES-D 
scale assessing depressive symptoms.31 Education 
was measured as the number of years of schooling at 
the third examination.

Covariates potentially related to LVM were also 
assessed at the third examination. These included 
age, weight, height, cigarette smoking status (pre-
cent, past, or never), alcohol intake (in ml/day), 
clinic baseline SBP obtained according to stan-

dardized epidemiologic methods32 and self-reported 
physical activity.33

Statistical Analysis Using the formula developed by de Simone and colleagues, LVM was normalized for height (LVM/height^2). Normalized values for LVM were then used in all subsequent analyses. Average levels of LVM and the covariates used in the multivariate analyses were determined for each race/gender group, and 2 (black/white) x 2 (men/women) analyses of variance (ANOVAs) were performed to determine differences among these groups. The remaining analyses were stratified by race and gender, given the differences among race/gender groups with regard to risk factors and LVM, as well as race/gender differences in reactivity (unpublished data). The peak blood pressure level measured during physical stress and the average level measured during psychological stress were generally used as indicators of reactivity. Change scores were also determined by subtracting the baseline blood pressure level from the above levels. Change scores for the cold pressor test were calculated in the same way, subtracting the baseline level measured by the Vita-Stat monitor from the manual blood pressure measure; given the frequent calibration of the Vita-Stat machines, it is unlikely that the difference in blood pressure determination techniques had any effect on the results of the study. Quintiles of each of these measures were calculated for each race/gender group. Individuals in the upper quintile were classified as exaggerated reactors to exercise or psychological stress. In most analyses, individuals with exaggerated BP reactivity were compared to the lower four quintiles (normal reactivity). T tests were used in the unadjusted models, and analysis of covariance was used in the models adjusting for covariates. Multiple linear regression (adjusting for covariates) was used to evaluate the relationships between LVM and psychosocial factors, as well as to determine the relative contribution of reactivity to the prediction of LVM. Given the large number of comparisons, a P value of .05 was considered significant.

RESULTS

LVM, Covariates, and Upper Quintiles of BP Reactivity The values for normalized LVM (LVM/height^2) and LVM covariates for the race/gender groups are shown in Table 1. Men had higher LVM than women, and blacks had greater LVM than whites. In a similar fashion, main effects for both race and gender were found. As testing SBP. Interactions between race and gender were found for weight and physical activity. Men were heavier and more physically active than women, black women were less physically active and heavier than white women. \( \chi^2 \) analyses showed that whites were also less likely to be current smokers than blacks.
The range of BP levels for each quintile of peak SBP response during exercise for the four race/gender groups are shown in Table 2. The SBP levels for the upper quintile are similar to levels defined by other studies as exaggerated SBP responses to exercise.10,11,12

**LVM and Exercise Testing**  The unadjusted and adjusted levels of LVM for the exaggerated and normal SBP reactivity during exercise were determined (Table 3). LVM was greater for exaggerated reactivity among white men only. In the adjusted analysis, LVM was 10% greater among white men with exaggerated reactivity than among normal reactors. The variable that explained the greatest amount of variance in all race/gender models was weight (for example, for white men, R² increment of 0.27 out of a total R² of 0.30).

**LVM and Reactivity to Psychological Stress**  The relationship of LVM and SBP reactivity to cold pressor is shown in Table 4. Unadjusted analyses were significant in all race/gender groups, and the relationships remained significant for white men and women after adjustment for covariates. The difference in LVM between exaggerated and normal reactivity was greatest among white women (7.1%). Exaggerated DBP reactivity to cold pressor was associated with greater LVM among black men only. However, this relationship did not remain significant when adjusted for covariates (P = .053; data not shown).

Exaggerated SBP reactivity to the video game was not significantly related to LVM in either univariate or multivariate analyses (data not shown). The relationship of LVM to DBP reactivity to the video game is shown in Table 5. Exaggerated reactivity was associated with greater LVM among both black men and black women in the unadjusted analyses; however, this relationship remained significant only among black men in the adjusted analyses. LVM was 8.1% greater among black men with exaggerated DBP reactivity than normal reactors in adjusted analyses. Among black men, exaggerated SBP reactivity to star-tracing was associated with greater LVM in unadjusted analyses (P = .0003), but not in adjusted analyses (P = .07; data not shown). The relationship of LVM to DBP reactivity to the star-tracing task is not significant in all race/gender groups. However, the relationship remained significant for white men and women at high levels of LVM. The variable that explained the greatest amount of variance in all race/gender models was weight (for example, for white men, R² increment of 0.27 out of a total R² of 0.30).

**TABLE 1. AVERAGE VALUES OF LEFT VENTRICULAR MASS AND COVARIATES BY RACE AND GENDER**

<table>
<thead>
<tr>
<th>Race</th>
<th>Gender*</th>
<th>Interaction*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Men</td>
<td>White Men</td>
<td>Black Women</td>
</tr>
<tr>
<td>No.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV Mass (g)/height² (m)</td>
<td>781</td>
<td>945</td>
</tr>
<tr>
<td>Resting SBP (mm Hg)</td>
<td>113.3 (11.0)</td>
<td>109.9 (10.3)</td>
</tr>
<tr>
<td>Weight (lb)</td>
<td>192.7 (38.3)</td>
<td>178.4 (30.9)</td>
</tr>
<tr>
<td>Physical activity</td>
<td>486.4 (349.2)</td>
<td>462.0 (288.4)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>29.3 (3.7)</td>
<td>30.5 (3.1)</td>
</tr>
<tr>
<td>Alcohol intake (ml/day)</td>
<td>350.4 (253.8)</td>
<td>30.0 (25.5)</td>
</tr>
<tr>
<td>Smoking status (%)</td>
<td>41.1 (25.6)</td>
<td>31.1 (23.6)</td>
</tr>
</tbody>
</table>

* p values from 2 × 2 ANOVA models. Data presented as n (%) SD.

† p = 0.05 for association.

**TABLE 2. EXAGGERATED BP REACTIVITY TO STRESS: RANGE OF EACH QUINTILE OF PEAK SYSTOLIC BLOOD PRESSURE DURING EXERCISE BY RACE AND GENDER**

<table>
<thead>
<tr>
<th>Peak SBP During exercise</th>
<th>Black Men</th>
<th>White Men</th>
<th>Black Women</th>
<th>White Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Men</td>
<td>126–180</td>
<td>128–180</td>
<td>96–148</td>
<td>104–130</td>
</tr>
<tr>
<td>White Men</td>
<td>126–180</td>
<td>128–180</td>
<td>96–148</td>
<td>104–130</td>
</tr>
</tbody>
</table>

All values are in mm Hg.
### TABLE 3. DIFFERENCES IN LEFT VENTRICULAR MASS* FOR EXAGGERATED AND NORMAL SBP REACTIVITY TO EXERCISE: UNADJUSTED AND ADJUSTED ANALYSES

<table>
<thead>
<tr>
<th>Blood Pressure Reactivity</th>
<th>Exaggerated</th>
<th>Normal</th>
<th>P Value</th>
<th>Exaggerated</th>
<th>Normal</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Black Men</td>
<td></td>
<td></td>
<td>Black Women</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>38.21 (0.80)</td>
<td>37.49 (0.37)</td>
<td>.01</td>
<td>36.05 (0.70)</td>
<td>34.83 (0.33)</td>
<td>.12</td>
</tr>
<tr>
<td>Adjusted†</td>
<td>37.51 (0.62)</td>
<td>37.64 (0.37)</td>
<td>.89</td>
<td>35.19 (0.72)</td>
<td>35.02 (0.33)</td>
<td>.93</td>
</tr>
<tr>
<td>Adjusted‡</td>
<td>37.35 (0.80)</td>
<td>37.67 (0.36)</td>
<td>.71</td>
<td>35.31 (0.68)</td>
<td>34.99 (0.31)</td>
<td>.67</td>
</tr>
<tr>
<td>Adjusted§</td>
<td>37.61 (0.86)</td>
<td>37.76 (0.46)</td>
<td>.87</td>
<td>35.67 (0.73)</td>
<td>35.34 (0.40)</td>
<td>.64</td>
</tr>
</tbody>
</table>

|                           | Black Women |        |         | Black Women |        |         |
| Unadjusted                | 39.37 (0.65) | 34.66 (0.29) | <.001 | 32.77 (0.63) | 32.14 (0.30) | .36 |
| Adjusted†                 | 39.22 (0.56) | 34.69 (0.29) | <.001 | 32.30 (0.63) | 32.25 (0.30) | .95 |
| Adjusted‡                 | 38.48 (0.61) | 34.86 (0.28) | <.001 | 32.26 (0.60) | 32.26 (0.28) | .98 |
| Adjusted§                 | 38.40 (0.62) | 34.92 (0.32) | <.001 | 32.70 (0.63) | 32.48 (0.31) | .74 |

* All values are LVM (grams/kg)† for 10. Data presented as mean (SD). Exaggerated Reactivity defined as upper quartile.
† Adjusted for resting SBP.
‡ Adjusted for resting SBP and weight.
§ Adjusted for resting SBP, weight, age, alcohol consumption, smoking status, and physical activity.

As shown in Table 6, unadjusted analyses revealed an association between increased LVM and exaggerated SBP reactivity in black men and women; after adjustment, the association was not significant in any race/gender group.

After controlling for resting BP, weight, and other covariates in multiple linear regression models, the only significant relationship was between SBP reactivity to cold pressor and LVM among white females only (P < .01). However, reactivity in this case explained less than 1% of the variance in the overall model (R² increment of 0.006); again, weight was by far the strongest predictor of LVM for all race/gender groups (R² increments of 0.12 to 0.21 out of total R² range of 0.19 to 0.29).

### TABLE 4. DIFFERENCES IN LEFT VENTRICULAR MASS* FOR EXAGGERATED AND NORMAL SBP REACTIVITY TO COLD PRESSOR: UNADJUSTED AND ADJUSTED ANALYSES

<table>
<thead>
<tr>
<th>Blood Pressure Reactivity</th>
<th>Exaggerated</th>
<th>Normal</th>
<th>P Value</th>
<th>Exaggerated</th>
<th>Normal</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Black Men</td>
<td></td>
<td></td>
<td>Black Women</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>40.51 (0.89)</td>
<td>37.15 (0.36)</td>
<td>&lt;.001</td>
<td>37.92 (0.64)</td>
<td>34.99 (0.32)</td>
<td>.001</td>
</tr>
<tr>
<td>Adjusted†</td>
<td>39.62 (0.67)</td>
<td>37.20 (0.38)</td>
<td>.02</td>
<td>37.21 (0.67)</td>
<td>34.86 (0.32)</td>
<td>.005</td>
</tr>
<tr>
<td>Adjusted‡</td>
<td>39.54 (0.45)</td>
<td>37.25 (0.37)</td>
<td>.06</td>
<td>36.94 (0.81)</td>
<td>34.62 (0.30)</td>
<td>.02</td>
</tr>
<tr>
<td>Adjusted§</td>
<td>39.72 (0.59)</td>
<td>37.33 (0.48)</td>
<td>.06</td>
<td>37.08 (0.66)</td>
<td>35.08 (0.46)</td>
<td>.02</td>
</tr>
</tbody>
</table>

|                           | Black Women |        |         | Black Women |        |         |
| Unadjusted                | 39.54 (0.71) | 35.19 (0.49) | <.001 | 34.35 (0.71) | 31.93 (0.28) | .01 |
| Adjusted†                 | 37.29 (0.74) | 35.20 (0.39) | .02 | 34.37 (0.67) | 32.78 (0.24) | .02 |
| Adjusted‡                 | 37.41 (0.50) | 35.23 (0.28) | .01 | 34.21 (0.72) | 31.81 (0.28) | .002 |
| Adjusted§                 | 37.26 (0.72) | 35.35 (0.37) | .01 | 34.29 (0.74) | 32.02 (0.51) | .004 |

* All values are LVM (grams/kg)† for 10. Data presented as mean (SD). Exaggerated Reactivity defined as upper quartile.
† Adjusted for resting BP.
‡ Adjusted for resting BP and weight.
§ Adjusted for resting BP, weight, age, alcohol consumption, smoking status, and physical activity.
TABLE 5. DIFFERENCES IN LVM FOR EXAGGERATED AND NORMAL DBP REACTIVITY TO VIDEO GAME: UNADJUSTED AND ADJUSTED ANALYSES*

<table>
<thead>
<tr>
<th>Blood Pressure Reactivity</th>
<th>Exaggerated</th>
<th>Normal</th>
<th>P Value</th>
<th>Exaggerated</th>
<th>Normal</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Black Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>39.89 (0.74)</td>
<td>37.03 (0.39)</td>
<td>.0007</td>
<td>36.38 (0.67)</td>
<td>34.50 (0.34)</td>
<td>.01</td>
</tr>
<tr>
<td>Adjusted†</td>
<td>39.78 (0.88)</td>
<td>37.03 (0.42)</td>
<td>.008</td>
<td>34.66 (0.77)</td>
<td>34.75 (0.35)</td>
<td>.93</td>
</tr>
<tr>
<td>Adjusted‡</td>
<td>39.74 (0.87)</td>
<td>37.04 (0.41)</td>
<td>.008</td>
<td>34.79 (0.72)</td>
<td>34.71 (0.33)</td>
<td>.93</td>
</tr>
<tr>
<td>Adjusted§</td>
<td>39.98 (0.92)</td>
<td>36.98 (0.51)</td>
<td>.004</td>
<td>35.17 (0.78)</td>
<td>35.18 (0.42)</td>
<td>.99</td>
</tr>
<tr>
<td><strong>White Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>34.37 (0.61)</td>
<td>35.29 (0.30)</td>
<td>.15</td>
<td>33.39 (0.60)</td>
<td>31.93 (0.30)</td>
<td>.03</td>
</tr>
<tr>
<td>Adjusted†</td>
<td>35.35 (0.73)</td>
<td>35.59 (0.32)</td>
<td>.78</td>
<td>33.95 (0.72)</td>
<td>32.79 (0.32)</td>
<td>.03</td>
</tr>
<tr>
<td>Adjusted‡</td>
<td>35.20 (0.69)</td>
<td>35.62 (0.30)</td>
<td>.59</td>
<td>33.19 (0.66)</td>
<td>31.88 (0.30)</td>
<td>.10</td>
</tr>
<tr>
<td>Adjusted§</td>
<td>35.40 (0.71)</td>
<td>35.72 (0.34)</td>
<td>.69</td>
<td>33.33 (0.70)</td>
<td>32.07 (0.33)</td>
<td>.11</td>
</tr>
</tbody>
</table>

* All values are LVM (grams/height²7/5) (millimeters). Data presented as mean (SE). Exaggerated reactivity classified as upper quintile.
† Adjusted for resting BP.
‡ Adjusted for resting BP and weight.
§ Adjusted for resting BP, weight, age, alcohol consumption, smoking status, and physical activity.

LVM among black females (r = 0.08, P = .01). After adjusting for significant covariates, β-coefficients were calculated to determine the relationships between LVM and psychosocial traits (Table 7). Lower education remained related to LVM among black and white women, and also was significant among black men. Depressive symptoms were related to LVM among white women only. However, the R² increment for these variables was less than 0.01 in the overall models (total R² range of 0.22 to 0.28; data not shown).

DISCUSSION

The present study does not support the hypothesis that exaggerated blood pressure reactivity to certain physical and psychological stressors independently contribute in a substantial way to increased LVM in healthy young adults. While univariate analyses show a number of statistically significant associations, these relationships are inconsistent when examined across race/gender groups, and the associations with LVM...

TABLE 6. DIFFERENCES IN LEFT VENTRICULAR MASS FOR EXAGGERATED AND NORMAL DBP REACTIVITY TO STAR-TRACING: UNADJUSTED AND ADJUSTED ANALYSES*

<table>
<thead>
<tr>
<th>Blood Pressure Reactivity</th>
<th>Exaggerated</th>
<th>Normal</th>
<th>P Value</th>
<th>Exaggerated</th>
<th>Normal</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Black Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>39.55 (0.74)</td>
<td>36.99 (0.39)</td>
<td>.002</td>
<td>36.73 (0.68)</td>
<td>34.49 (0.34)</td>
<td>.004</td>
</tr>
<tr>
<td>Adjusted†</td>
<td>39.35 (0.73)</td>
<td>37.02 (0.41)</td>
<td>.02</td>
<td>35.72 (0.78)</td>
<td>34.62 (0.35)</td>
<td>.22</td>
</tr>
<tr>
<td>Adjusted‡</td>
<td>39.06 (0.84)</td>
<td>37.11 (0.41)</td>
<td>.05</td>
<td>35.77 (0.73)</td>
<td>34.60 (0.33)</td>
<td>.16</td>
</tr>
<tr>
<td>Adjusted§</td>
<td>38.18 (0.90)</td>
<td>37.16 (0.50)</td>
<td>.05</td>
<td>36.22 (0.78)</td>
<td>34.94 (0.43)</td>
<td>.012</td>
</tr>
<tr>
<td><strong>White Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>36.59 (0.61)</td>
<td>35.24 (0.30)</td>
<td>.05</td>
<td>32.82 (0.64)</td>
<td>32.05 (0.29)</td>
<td>.28</td>
</tr>
<tr>
<td>Adjusted†</td>
<td>36.02 (0.71)</td>
<td>35.36 (0.31)</td>
<td>.41</td>
<td>32.75 (0.74)</td>
<td>31.47 (0.31)</td>
<td>.35</td>
</tr>
<tr>
<td>Adjusted‡</td>
<td>35.61 (0.67)</td>
<td>35.46 (0.30)</td>
<td>.85</td>
<td>32.07 (0.70)</td>
<td>32.11 (0.29)</td>
<td>.95</td>
</tr>
<tr>
<td>Adjusted§</td>
<td>36.11 (0.68)</td>
<td>35.41 (0.33)</td>
<td>.35</td>
<td>32.19 (0.71)</td>
<td>32.30 (0.32)</td>
<td>.99</td>
</tr>
</tbody>
</table>

* All values are LVM (grams/height²7/5) (millimeters). Data presented as mean (SE). Exaggerated reactivity classified as upper quintile.
† Adjusted for resting BP.
‡ Adjusted for resting BP and weight.
§ Adjusted for resting BP, weight, age, alcohol consumption, smoking status, and physical activity.
are modest when other factors related to LVM are taken into account. The most prominent factor associated with LVM is weight, with all other factors (including resting SBP) contributing, at most, 3% to 5% to the explanation of the variance in LVM in multivariate models.

The strongest finding in the study is that increased LVM is independently associated with increased maximal SBP change during exercise in men. However, this factor contributed only 3% to the explanation of the variance of LVM among white men and less than 1% in black men. This finding is similar to the relationship found in a healthy cohort from the Framingham Heart Study,13 which found that white men and women with exaggerated SBP responses to exercise had only 5% to 8% greater LVM after adjusting for age, body mass, and resting SBP. In the present study, this difference among white men was somewhat higher (10%), but was not found among either white women or black men and women. Both the present study and the Framingham study were conducted with large, healthy cohorts, which may explain the differences in findings relative to other studies.12

The results for the psychological stressors were similar to the results for exercise testing. Of a total of 21 analyses conducted in this study for the three stressors, 10 were significant in the unadjusted analyses (five for cold pressor, two for the video game stressor, three for the star-tracing task). However, after adjustment for covariates, only three remained significant (two for the cold pressor task, one for the video game stressor). Even where these relationships remained statistically significant, the differences in LVM between exaggerated and normal reactivity were modest, ranging from 5.4% to 8.1%. Perhaps more importantly, only one of 24 relationships in this study was significant in multiple linear regression analyses, and in this case (SBP reactivity to cold pressor among white women), reactivity accounted for less than 1% of the variance in LVM. Previous studies of reactivity and LVM have included either children with a family history of hypertension,14 or patients with white coat hypertension.15 The differences in subject characteristics between these studies and the present study may partially account for the differences in findings.

Among the psychosocial variables, only lower education was consistently related to increased LVM. This relationship was significant among three of the four race/gender groups, however, like blood pressure reactivity to stress, this variable contributed little explanatory value in the multivariate models.

There are several limitations to the present study, including the relatively short protocol for the psychological stressors, and the lack of a more provocative psychological stressor that might have induced significant anger or anxiety. In addition, reactivity to exercise and psychological stressors were not measured concurrently with LVM. The stability of both of these measures over time is open to question; the relationships between LVM and reactivity may therefore have been weakened by the nonconcurrent measurement of these variables. However, as these reactivity measures were assessed prior to LVM evaluation, the findings might also be seen as the ability of physical and psychological reactivity to predict LVM. In addition, although there have been no longitudinal studies of change in LVM among young adults, a cross-sectional study indicated that LVM is relatively stable from late adolescence to early old age.23 Future studies would benefit from monitoring LVM change over time to determine whether reactivity to stress predicts later increases or rates of change in LVM.
In summary, blood pressure reactivity to physical and mental stressors adds little to resting blood pressure and weight in the prediction of LVM among young, healthy adults. The psychological characteristics of depression, anger suppression, hostility, and anxiety also do not independently add to the prediction of LVM. These negative findings indicate that reactivity and psychological characteristics exert their influence on cardiovascular disease through mechanisms other than increased LVM. Future studies might benefit from serial measurements of LVM over longer follow-up periods, in order to determine whether blood pressure reactivity to stress or psychological characteristics are associated with the development of left ventricular hypertrophy.

REFERENCES

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