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Normal Exercise Blood Pressure Response in African-American Women with Parental History of Hypertension

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Abstract

Background—Genetic and environmental hypotheses may explain why normotensive persons at high risk of developing hypertension often exhibit greater cardiovascular reactivity to stressors than those at low risk.

Methods—Pearson's correlation was used to evaluate reproducibility and independent *t* test to compare the cardiovascular responses to 30 W of exercise of normotensive young adult African-American women with positive and negative parental histories (PH) of hypertension (PH^+, n = 23; PH^-, n = 20).

Results—Correlations were significant for duplicate measurements. The effects of PH on blood pressure measured at rest and during exercise were not statistically significant ($P > 0.1$). A nearly significant trend for greater resting $\dot{V}O_2$ ($P = 0.08$) was detected in the PH^- than in the PH^+ group (3.67 ± 0.18 versus 3.26 ± 0.14 mL/kg/min).

Conclusion—A hyper-reactive blood pressure response to exercise, characteristic of the evolution of hypertension, may not be present among the normotensive female offspring of hypertensive African Americans. The significance of an 11% intergroup difference in the mean resting $\dot{V}O_2$ observed in this study is unclear.

KEY INDEXING TERMS

Hypertension; Exercise; African Americans; Parental history

Cardiovascular disease remains the leading cause of death in women in the United States, with hypertension being the leading health problem in adult African-American women.[1] Blood pressure responses to various stressors such as the cold pressor test,[2] mental challenge,[3] and physical exercise[4–6] have been used as early physiologic markers for the evolution of hypertension that could suggest effective preventive strategies. Jette et al[4] reported a 2- to 10-fold increase in the risk of hypertension for individuals characterized by a "hyperresponder" pressor response to exercise. An exaggerated exercise blood pressure

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response has been described in individuals with hypertension,[7] in the normotensive offspring of hypertensive parents,[8–11] and in normotensive African-American cohorts that have been compared with white American cohorts.[12–15] Although studies have been designed to investigate the pressor response to stressors known to elicit either beta-adrenergic (mental challenge) or alpha-adrenergic (cold stress) reactivity in the female offspring of hypertensive African-American parents,[16–18] how the blood pressure in African Americans responds to stress eliciting both beta- and alpha-adrenergic reactivity has not been addressed in that population. In the present study, we compared the blood pressure responses in normotensive young adult African-American women with and without a parental history of hypertension during physical exercise, which is known to elicit both beta- and alpha-adrenergic reactivity. We hypothesized that the normotensive offsping of hypertensive parents would have a greater pressor response to physical exercise than the offspring of normotensive parents.

Methods

Subjects

Forty-three healthy normotensive African-American women, 18 to 26 years of age, were recruited to participate in this study. Twenty-three of the women were identified as having one or both biological parents with essential hypertension, and the remaining 20 women were identified as having biological parents with normal blood pressure. The number of subjects included in the present study was based on data indicating that the critical detectable differences in total peripheral resistance (TPR) measured at rest for differentiating between groups of normotensive versus hypertensive subjects was 23%,[19] of normotensive versus borderline hypertensive subjects was 32%,[20] and of normal and borderline hypertensive salt-sensitive versus salt-insensitive subjects was 44%.[21] The parents of the participants were identified as normotensive (systolic pressure <140 mm Hg and diastolic pressure <90 mm Hg) or hypertensive (systolic pressure >140 mm Hg or diastolic pressure >90 mm Hg) by personal telephone calls, or on the basis of replies to a questionnaire given to participants to assess their parents' medical blood pressure histories. Most of the parents with hypertension had stage II (moderate) hypertension and all were receiving pharmacological treatments, with some parents receiving pharmacological and nonpharmacological treatments for the condition.

Criteria for subjects' participation in the study included nonsmoking status, physically inactive with a peak oxygen uptake ($\dot{V}O_{2peak}$) of 35 mL/kg/min or less, absence of alcohol abuse (less than two standard drinks per day), absence of medication (including birth control), body mass index less than 24, and resting systolic/diastolic pressures less than 140/90 mm Hg. The Howard University Human Participants Institutional Review Board approved all experimental procedures. The nature, purpose, and risks of the study were explained to each subject before written informed consent was obtained.

Experimental Test Conditions

Progressive Exercise Test of $\dot{V}O_{2peak}$—Initially, the subjects performed a progressive exercise test of $\dot{V}O_{2peak}$ on an Ergoline 800S electronic-braked leg cycle ergometer (SensorMedics, Anaheim, CA) to document that functional work capacity was similar in all subjects ($\dot{V}O_{2peak} \leq 35$ mL/kg/min). Each subject began by exercising at 30 watts (W) for 3 minutes. The work rate was then increased by 30 W in successive 3-minute stages until volitional exhaustion. Minute ventilation was measured using a FLO-1 volume transducer (PHYSIO-DYNE Instruments, Quogue, NY). Percentages of expired oxygen ($\dot{V}O_2$) and carbon dioxide (CO_2) were measured using a paramagnetic $\dot{V}O_2$ analyzer and an infrared

CO_2 analyzer connected to an online computerized PHYSIO-DYNE system and were calibrated to known medical grade gases.

Submaximal Exercise Test—To determine the cardiovascular responses to exercise, the subjects performed a single steady-state work bout on the cycle ergometer 5 to 10 days following the progressive exercise test of $\dot{V}O_{2peak}$. All subjects were given instructions to restrict physical activity and food for 3 hours and caffeine and alcohol products for 12 hours prior to entering the laboratory. To counteract any confounding effects of the menstrual cycle on the blood pressure response to submaximal exercise, all subjects were tested in the luteal phase of their menstrual cycle. After 15 minutes of seated rest position on the cycle ergometer, baseline measures of systolic blood pressure, diastolic blood pressure, mean arterial blood pressure (MABP), heart rate, cardiac output (CO), and TPR were recorded at minutes 14 and 15. The mean value of the two recordings was calculated for each variable. The subject was then required to exercise on the cycle ergometer at an absolute work intensity of 30 W for 10 minutes. During the work bout, blood pressure, heart rate, CO, and TPR were measured at minutes 9 and 10, with the calculated mean used for analysis.

Blood pressure was measured in the right arm using a SunTech automated blood pressure monitor (SunTech Medical Instruments, Raleigh, NC). The automated monitoring uses the method of gating the electrocardiogram R-wave with the Korotkoff sounds and has been validated against intra-arterial recordings during rest and exercise conditions.[22] MABP was calculated using the conventional formula: diastolic blood pressure + [1/3] pulse pressure. Heart rate was determined by the electrocardiogram using the automated blood pressure monitor.

Carbon monoxide was measured noninvasively by the CO_2 rebreathing technique of Collier.[23] End-tidal partial pressure of CO_2 (P_{CO_2}) was measured using a rapid-response infrared CO_2 analyzer (PHYSIO-DYNE Instrument, Quogue, NY). End-tidal P_{CO_2} was used to estimate arterial P_{CO_2} according to the formula of Jones et al.[24]

Each subject rebreathed into a 5-L latex bag containing CO_2 and $\dot{V}O_2$ to permit rapid equilibration with venous P_{CO_2}. A valid equilibrium during rebreathing was measured by determining a “plateau” in P_{CO_2} according to the criteria of Ashton and McHardy[25] and Jones et al.[24] These criteria require that during the first 6 to 8 seconds, an inspiration has to be followed by an expiration whose P_{CO_2} value is within ±1 mm Hg of the first recorded value. An automated gas mixing apparatus was used to adjust the initial gas volume and initial P_{CO_2} in the rebreathing bag according to the procedure described by Jones et al.[24] If a trial had to be redone, end-tidal P_{CO_2} values were allowed to return to baseline values before the procedure was repeated. CO was calculated from the indirect Fick equation:

$$CO=\dot{V}_{CO2}/(C_{VCO2}-C_{aCO2})$$

Where V_{CO2} is expired carbon dioxide, C_{VCO2} is mixed venous CO_2 content, and C_{aCO2} is arterial CO_2 content.

TPR was calculated from the formula:

$$TPR=(MABP)/CO \times 80(\text{expressed as dyne} \cdot s \cdot cm^{-5})$$

Statistical Analysis

The study design consisted of duplicate measurements of cardiovascular variables associated with resting and exercising conditions and comparison of means using the independent *t* test for the effects of parental history of hypertension on observed differences in $\dot{V}O_{2peak}$, heart rate, blood pressure, CO and TPR. Pearson's correlation coefficient was computed to evaluate reproducibility of the measured variables. ANOVA was used to assess statistical power. Data were analyzed utilizing the SPSS statistical program (SPSS, Chicago, IL). A *P*-value of 0.05 or less was considered statistically significant. All data were expressed as mean ± standard error (SE).

Results

Pearson's correlation coefficient ranged from 0.7 to 0.9 ($P < 0.01$) for heart rate, systolic pressure, diastolic pressure and CO at rest and at 30 W and for oxygen consumption at 30 W; the coefficient for oxygen consumption at rest was 0.3 ($P < 0.05$).

Table 1 presents the descriptive data of the groups with and without a parental history of hypertension. The intergroup differences in demographic and physiologic characteristics related to age, body height, body weight, body mass index, blood pressure, heart rate, and $\dot{V}O_{2peak}$ were found to be not significant.

Table 2 presents the cardiovascular measures at rest and during submaximal exercise pooled across subjects. During exercise corresponding to a metabolic activity of 44% $\dot{V}O_{2peak}$, the blood pressure, heart rate and CO were significantly greater than resting values, with the TPR values being significantly smaller. Resting measures of blood pressure, heart rate, and CO were not significantly different between the groups and the main effects of parental history on the cardiovascular measurements associated with resting and exercising conditions were not significant.

While the results in Table 2 showed submaximal exercise to significantly influence the cardiovascular responses across subjects, Table 3 illustrates that the cardiovascular responses to exercise in the groups with and without a parental history of hypertension were not significantly different.

Discussion

The method of comparing normotensive individuals with and without a family history of hypertension has been used to evaluate the etiologic role of factors such as disturbed vascular reactivity,[26] sodium intake and sodium transport,[27,28] and cardiovascular reactivity to stress.[8,10] It has been pointed out that the development of hypertension is predicted by the blood pressure response to the stress of physical exercise.[4–6]

The mechanism for an exaggerated blood pressure response to exercise is not clearly understood. To meet the increased metabolic demands of dynamic exercise, physiologic increases in CO are associated with decreases in TPR. Consequently, an exaggerated hyper-reactive blood pressure response to exercise must be a function of a greater-than-normal increase in CO and/or TPR. After reviewing the literature, the most likely explanation appears to be a failure to reduce TPR.[11,18] In a prior study, we found that normotensive college-age black subjects had a greater blood pressure response to submaximal exercise than a comparison group of white subjects[14]; however, a tendency approaching statistical significance was observed only for the black subjects having both a greater pressor response and a parental history of hypertension. Racial differences for heart rate and CO were not

observed, but the black subjects were found to have greater TPR than the white subjects both during resting and submaximal exercising conditions.

In the present study, we measured the blood pressure responses to submaximal exercise in normotensive young adult African-American women with and without a parental history of hypertension. The findings showed that the women with and without a parental history of hypertension, and similar levels of functional work capacity, exhibited no differences in their blood pressure responses to submaximal exercise at a work intensity of 30 W. Similar findings were found for measures of heart rate, CO, and TPR. Therefore, the findings of this study failed to support the hypothesis that women with a parental familial history of hypertension have greater blood pressure and TPR responses to dynamic exercise than women without such parental history. Our findings are consistent with those suggesting that genetic similarity may be more important than genetic variation across ethnic and racial groups. Among 109 regions of human DNA exhibiting substantial polymorphism, 85% of the genetic variation was found within, rather than between, the DNA samples of racial groups.[29] This finding supports the notion that the prevalence of hypertension may be better correlated with environment and lifestyle than with genetic variations.[30] However, racial differences in responses to angiotensin converting enzyme inhibitors are thought to result from both genetic and environmental factors.[31] Therefore, the relationship between a hyper-reactive blood pressure response to exercise and a familial genetic predilection for hypertension, which we found to be absent in normotensive young adult African-American women, may also be indicative of complex gene-environment interactions that are worthy of further investigation.

African Americans have a higher incidence of hypertension than other racial groups and some researchers have shown that normotensive men and women who exhibit exaggerated blood pressure responses to exercise may be at risk for future hypertension. The present studies have shown that young adult normotensive African-American women did not exhibit an exaggerated blood pressure response, albeit to a relatively low level of dynamic exercise. Therefore, the finding of nonsignificance of TPR reported herein may have been related to the low intensity of the workloads consisting of 30 W (approximately 44% $\dot{V}O_{2peak}$). However, in a previous study performed in our laboratory, we were able to detect exaggerated blood pressure responses to exercise at 50% $\dot{V}O_{2peak}$ in African-American men.[32] Prior studies have shown that neither African-American men nor women who are normotensive showed a greater blood pressure response to dynamic exercise than white Americans or Asian-Americans.[33] In that study, the dynamic exercise responses produced significant increments in both systolic and diastolic pressures at 70% of the subjects' $\dot{V}O_2$ reserve. In the present study, both of the parental history groups, positive and negative, exhibited significant decrements in diastolic pressures and TPR associated with increased systolic pressures. The finding of nonsignificance of TPR may also have been related to the statistical power of the present study that would, no doubt, have been increased by studying a larger sample size. The present sample size would have been sufficient to detect a 25%–30% difference in TPR. The intergroup difference in TPR at 30 W was not significant; however, the intergroup difference in the mean resting VO_2 was nearly significant (P = 0.08), albeit small (11%).

Relationships between age, height, weight, body mass index and resting energy expenditure, $\dot{V}O_2$, CO and TPR have previously been described in African-American[34,35] and other female -populations.[36,37] The closeness of the statistics to significance at P = 0.05 for resting $\dot{V}O_2$ suggests the possibility that inclusion of more subjects may have increased the statistical power of this measurement. However, the statistical power was observed to be a low value of 0.36 and β (power =1 − β) the probability of making a type II error was, therefore, large enough to not permit us to exclude the possibility of a type II error. Given

the small 11% intergroup difference, a much larger sample size would have been required to lessen β to the acceptable range of 0.2. The sample size of the present study was based on our review of the scientific literature, which suggested that the smallest detectable difference in TPR capable of differentiating groups of normotensive and borderline hypertensive subjects is 32%.[20]

A number of studies have shown that pulse pressure is inversely related to arterial compliance.[38] Therefore, our exercise studies, which appear to have been performed on African-American women at a greater pulse pressure than those performed by Wright et al[33] on African-American men and women, suggest that Wright and associates may have used a population of African-American subjects with a substantially smaller arterial compliance than our population. The aerobic capacity of our young adult African-American female subjects was low to moderate, and the fitness of those with a positive parental history of hypertension was, therefore, not found to be significantly different than that of those having a negative parental history. However, recent studies involving laboratory stress testing of young adult African-American women exhibiting low to moderate levels of aerobic capacity have shown that $\dot{V}O_{2peak}$ was positively correlated with the increase in TPR during mental stress testing, with an attenuated increase in systolic blood pressure after cold pressor testing, with recovery of blood pressure or TPR after mental stress testing, and with greater blood pressure responses during and after mental stress testing in women having a negative parental history of hypertension than in those having a positive parental history.[39] These findings suggest that low-intensity exercise performed at 30 W or 44% $\dot{V}O_{2peak}$ may not be as provocative as mental effort tests and cold pressor stressors for evaluating the risk of hypertension in populations of young adult African-American women exhibiting a low to moderate level of aerobic capacity.

In summary, we found a small nearly significant intergroup difference in VO_2 measured at rest related to parental history of hypertension that may have reflected interactions between variations in age, height, weight, and body mass index. However, we could not detect significant intergroup differences in measurements of cardiovascular variables such as blood pressure, CO, and TPR measured during resting or exercising conditions that were related to parental history of hypertension. These findings suggest that a hyper-reactive blood pressure response to exercise, characteristic of the evolution of hypertension, may not be present among the normotensive female offspring of hypertensive African-American parents and that the role of parental history of hypertension in exaggerated blood pressure responses to submaximal exercise may not be significant.

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Table 1

Descriptive Characteristics of Study Subjects with and without Parental History of Hypertension

Variable	Groups[a]	
	Without Parental Hypertension (n = 20)	With Parental Hypertension (n = 23)
Age (yrs)	20.6 ± 0.5	20.9 ± 0.5
Height (cm)	164.9 ± 2.0	165.4 ± 1.5
Weight (kg)	67.6 ± 2.6	69.4 ± 2.5
Body mass index	20.5 ± 0.7	20.9 ± 0.6
HR_{rest} (beats/min)	83.2 ± 3.1	79.0 ± 2.2
SBP_{rest} (mm Hg)	116.9 ± 2.5	117.3 ± 2.3
DBP_{rest} (mm Hg)	71.4 ± 1.1	72.5 ± 1.5
$MABP_{rest}$ (mm Hg)	86.4 ± 1.4	87.3 ± 1.4
HR_{peak} (beats/min)	181.4 ± 1.6	183.0 ± 1.9
$\dot{V}O_{2peak}$ (mL/kg/min)	25.8 ± 0.9	26.8 ± 0.8

DBP_{rest}, resting diastolic pressure; HR_{peak}, peak heart rate; HR_{rest}, resting heart rate; $MABP_{rest}$, resting mean arterial pressure; SBP_{rest}, resting systolic pressure; $\dot{V}O_{2peak}$, peak oxygen uptake.

[a] Mean ± standard error.

Table 2

Mean Cardiovascular Responses at Baseline and During Submaximal Exercise Pooled Across Subjects with and without Parental Hypertension

	Responses (n = 43)[a]	
Variable	**Baseline**	**30 Watts[b]**
SBP (mm Hg	117.1 ± 1.7	133.4 ± 2.4
DBP (mm Hg)	71.9 ± 1.0	69.3 ± 1.0
MABP (mm Hg)	86.8 ± 1.0	90.5 ± 1.2
HR (beats/min)	81.1 ± 1.8	111.8 ± 1.7
CO (L/min)	5.1 ± 0.1	8.6 ± 0.2
TPR (dyne/sec/cm^5)	1409.4 ± 39.3	867.9 ± 23.5

CO, cardiac output; DBP, diastolic pressure; HR, heart rate; MABP, mean arterial pressure; SBP, systolic pressure; TPR, total peripheral vascular resistance.

[a] Mean ± standard error.

[b] $P < 0.05$.

Table 3

Mean Cardiovascular Values for Subjects with Parental History of Hypertension (n = 23) and without Parental History of Hypertension (n = 20) at Resting and Submaximal Exercise (30 W) Conditions

	Without Parental Hypertension[a]		With Parental Hypertension[a]	
Variable	**Resting**	**30 Watts**	**Resting**	**30 Watts**
SBP (mm Hg)	116.9 ± 2.5	132.6 ± 3.7	117.3 ± 2.3	134.2 ± 3.6
DBP (mm Hg)	71.4 ± 1.1	68.5 ± 1.3	72.5 ± 1.5	70.1 ± 1.5
MABP (mm Hg)	86.4 ± 1.4	89.7 ± 1.5	87.3 ± 1.4	91.3 ± 1.8
HR (beats/min)	83.2 ± 3.1	111.6 ± 2.4	79.0 ± 2.2	112.0 ± 2.3
CO (L/min)	5.2 ± 0.2	8.7 ± 0.3	5.0 ± 0.2	8.4 ± 0.3
TPR (dyne/sec/cm^5)	1362.5 ± 44.5	842.0 ± 29.2	1456.7 ± 67.7	894.1 ± 36.5

CO, cardiac output; DBP, diastolic pressure; HR, heart rate; MABP, mean arterial pressure; SBP, systolic pressure; TPR, total peripheral vascular resistance.

[a] Mean ± standard error.