

ATTENUATION OF EXAGGERATED EXERCISE BLOOD PRESSURE RESPONSE IN AFRICAN-AMERICAN WOMEN BY REGULAR AEROBIC PHYSICAL ACTIVITY

Introduction: A hyperreactive blood pressure response to exercise is a predictor of developing hypertension. The present study determined the influence of physical activity on an exaggerated exercise blood pressure response (EEBPR) in normotensive African-American women.

Methods: We screened 36 women 18–26 years of age for EEBPR defined as a ≥ 50 mm Hg difference in systolic blood pressure at rest and during exercise at 50% peak oxygen uptake (VO_{2peak}). Seven subjects demonstrated an EEBPR and participated in the study. Study participants trained for eight weeks on a bicycle ergometer at a work intensity of 70% VO_{2peak} . Blood pressure, heart rate, cardiac output (CO), stroke volume (SV), and total peripheral vascular resistance (TPR) were determined at baseline and during submaximal exercise at power outputs of 30 W and 50% VO_{2peak} . Subjects served as their own controls, and data were evaluated by using a paired *t* test at $P < .05$.

Results: Effectiveness of the intervention was shown by a significantly greater VO_{2peak} associated with significant decrements in systolic and mean arterial pressures at power outputs of 30 W and 50% VO_{2peak} . A significant decrement in heart rate was observed during exercise at 30 W. Significant increments in CO and SV and decrement in TPR were found during exercise at 50% VO_{2peak} .

Conclusion: The reduction in TPR associated with regular aerobic physical activity may attenuate the EEBPR and decrease the risk for hypertension in normotensive, young-adult, African-American women. (*Ethn Dis.* 2005;15 [suppl 5]:S5-10–S5-13)

Key Words: Exercise, African-American Women, Blood Pressure

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INTRODUCTION

Hypertension is a major risk factor for cardiovascular disease and is the leading cause of death in women in the United States. Epidemiologic data indicate that hypertension is the single most important risk factor for the markedly excessive rates of premature death and disability from heart attack, heart failure, stroke, and renal failure in African-American women.¹ Because the cause of hypertension is often unclear, considerable interest has been directed toward identifying risk factors for the evolution of hypertension and implementing strategies to prevent or delay the onset of disease. One approach used in predicting future hypertension is to assess the blood pressure response to acute bouts of exercise. During dynamic exercise, systolic pressure normally increases within limits of an increase in oxygen uptake (VO_2) with little or no change in diastolic pressure. The magnitude of increase in systolic pressure during exercise that exceeds the expected magnitude for a given increase in VO_2 describes an exaggerated exercise blood pressure response (EEBPR)² and has been shown to be a predictor of future hypertension in middle-aged women.³ As a strategy to reduce the incidence of hypertension in susceptible individuals, the American College of Sports Medicine recommends a lifestyle

of regular physical activity, and studies have shown that physical activity is associated with a lowering of resting blood pressure in women.^{4–6} Therefore, the present study was designed to determine whether regular aerobic exercise exerts an antihypertensive effect in sedentary, normotensive, young-adult, African-American women with EEBPR.

METHODS

Study Participants and Methods

The Howard University Human Participants Institutional Review Board approved the study protocol, and each subject provided informed consent. Thirty-six healthy, normotensive, young-adult, African-American females, 18–26 years of age, were recruited and screened for EEBPR. We defined an EEBPR as the calculated acute change in systolic blood pressure from sitting rest to 50% VO_{2peak} (change in systolic blood pressure = exercise systolic blood pressure minus resting systolic blood pressure) ≥ 50 mm Hg. Among the subjects screened, seven meet the EEBPR criteria and served as participants. Inclusion criteria also included nonsmoking status, absence of alcohol abuse (fewer than two standard alcohol drinks a day), resting systolic/diastolic blood pressure $< 140/90$ mm Hg, absence of medication (including birth control), and lack of regular physical for six months before the study.

The EEBPR was documented during a progressive exercise test of peak oxygen uptake (VO_{2peak}). Prior instructions were given to the participants to fast for three hours and restrict physical

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activity for 12 hours before entering the laboratory. The progressive exercise test of $\text{VO}_{2\text{peak}}$ was carried out by using an electronic-braked cycle ergometer (Ergoline 800S, SensorMedics Cooperation, Anaheim, CA) that began at an initial three-minute work intensity of 30 W then increased by 30-W increments at three-minute intervals until volitional fatigue. The VO_2 value achieved during the last minute of the progressive exercise test was used to define the $\text{VO}_{2\text{peak}}$ value. The VO_2 was assessed with the Max-II metabolic system (Physio-Dyne Instruments, Quoque, NY) that was calibrated with known gases.

Brachial artery blood pressure was measured simultaneously with exercise minute VO_2 values using a SunTech Tango exercise blood pressure monitor (SunTech Medical Instruments, Morrisville, NC). During exercise the Tango system automatically acquired blood pressure readings at one-minute intervals during the procedure. In a recent study, the SunTech Tango exercise blood pressure monitor was compared against brachial artery pressure measured with an indwelling catheter during treadmill exercise, and it was found to reliably identify systolic and diastolic blood pressure values.⁷ Blood pressure was measured in the right arm in accordance with the guidelines from the 2003 Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure.⁸ Mean arterial blood pressure (MABP) was calculated as diastolic pressure + 1/3 pulse pressure (systolic pressure minus diastolic pressure). During exercise, heart rate was measured by the electrocardiogram incorporated in the SunTech Tango exercise blood pressure monitor.

Approximately 3–6 days after the progressive exercise test of $\text{VO}_{2\text{peak}}$ /EEBPR, participants performed a submaximal exercise test working at a power output level of 30 W and 50% $\text{VO}_{2\text{peak}}$. The submaximal exercise test consisted

Table 1. Characteristics of subjects before and after aerobic exercise training

Variable	Pre-Trained	Post-Trained
Age (y)	20 ± 0.89	NC
Height (cm)	167.4 ± 3.6	NC
Weight (kg)	76.9 ± 3.8	76.2 ± 3.9
HR _{rest} (bpm)	88.1 ± 2.6	83.5 ± 3.9
SBP _{rest} (mm Hg)	116.4 ± 3.5	115.5 ± 3.3
DBP _{rest} (mm Hg)	73.2 ± 2.0	74.1 ± 2.7
HR _{peak} (bpm)	182 ± 2.2	181.5 ± 2.2
$\text{VO}_{2\text{peak}}$ (mL · kg ⁻¹ · min ⁻¹)	26.4 ± 0.9	29.9 ± 1.2*

Data in means ± standard errors.

* Indicates significant difference between treatment periods ($P < .05$).

NC, no change; HR_{rest}, baseline heart rate; bpm, beats per minute; SBP_{rest}, baseline systolic blood pressure; DBP_{rest}, baseline diastolic blood pressure; HR_{peak}, peak heart rate; $\text{VO}_{2\text{peak}}$, peak oxygen uptake.

of a 10-minute baseline period, followed by the subject working for eight minutes at the absolute workload of 30 W and eight minutes at the relative workload at 50% $\text{VO}_{2\text{peak}}$. Blood pressure, heart rate, and cardiac output were measured at baseline (min 8–10) and during min 6–8 of the absolute and relative workloads. Blood pressure, heart rate, and VO_2 were measured as described earlier. Cardiac output (CO) was assessed noninvasively by using the CO_2 rebreathing method of Collier,⁹ with the procedures of the technique described by our laboratory and other laboratories.^{10–13} Stroke volume was calculated by dividing the estimated CO value by the heart rate value. Total peripheral vascular resistance (TPR) was estimated by using the formula:

$$\text{TPR} = (\text{MABP}/\text{CO}) \times 80$$

(values expressed as dyne/sec/cm⁵).

After the submaximal exercise test the participants performed an exercise training program that consisted of cycling on a bicycle ergometer three times per week, for 30 min per session, at a work intensity of 70% $\text{VO}_{2\text{peak}}$ for 8 weeks. Exercise sessions were carried out in the research laboratory and monitored by research assistants. Following the 8-week training period, the initial $\text{VO}_{2\text{peak}}$ /EEBPR test and the submaximal exercise absolute and relative workload tests were repeated. The $\text{VO}_{2\text{peak}}$ /EEBPR and submaximal exer-

cise tests were conducted during the luteal phase of the menstrual cycle to prevent any confounding hormonal effect on the observed blood pressure.

To analyze data for significant differences between pre- and post-test periods, the paired *t* test was used. Statistical analyses were conducted by using the SPSS v. 10 software (SPSS Inc., Chicago, Ill), and data are presented as means ± standard errors. Statistical significance was established at $P < .05$.

RESULTS

Table 1 presents physiologic characteristics of the study participants. The mean age was 20 ± 0.89 years, with no observed differences in the body weight, blood pressure, and heart rate values between the pre- and post-training study periods. After training, a significant 8.8% increase was seen in functional work capacity ($\text{VO}_{2\text{peak}} = 26.4 \pm .9 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ vs. $29.9 \pm 1.2 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) ($P = .006$).

The pre- and post-training cardiovascular responses at baseline and during submaximal exercise at 30 W and during 50% $\text{VO}_{2\text{peak}}$ are illustrated in Figures 1 and 2. Baseline cardiovascular measures of arterial pressure, heart rate, cardiac output (CO), stroke volume (SV), and total peripheral resistance (TPR) were not significantly different than those measured after exercise

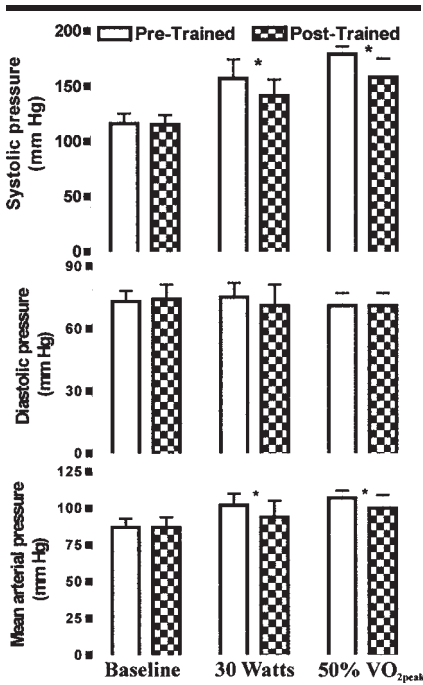


Fig 1. Blood Pressure Responses to Exercise Training. The effects of eight weeks of aerobic exercise measured at rest (baseline), at an absolute workload of 30 W and at 50% of peak oxygen uptake (VO_{2peak}) in African-American women with an exaggerated blood pressure response to exercise

training. During submaximal exercise at the same absolute workload of 30 W, systolic pressure, MABP and heart rate were significantly lower after training than before training. Training did not alter diastolic pressure, SV, TPR, or CO. However, the pre- vs post-training differences in CO approached significance ($t=2.1, P=.07$). Similarly, an exercise training effect was also noted for the blood pressure response during exercise at 50% VO_{2peak}. The systolic pressure and MABP were decreased by 11.7% (179.1 ± 2.7 vs. 158.0 ± 6.5 mm Hg) and 6.5% (107.2 ± 1.9 vs. 100.0 ± 3.5 mm Hg), respectively. This training-induced decrement in blood pressure was associated with a significant increment in CO (9.3 ± 3 vs 10.4 ± 5 L·min⁻¹) and significant decrement in TPR (924.9 ± 44.5 vs 784.0 ± 55.7 dyne/s/cm⁵).

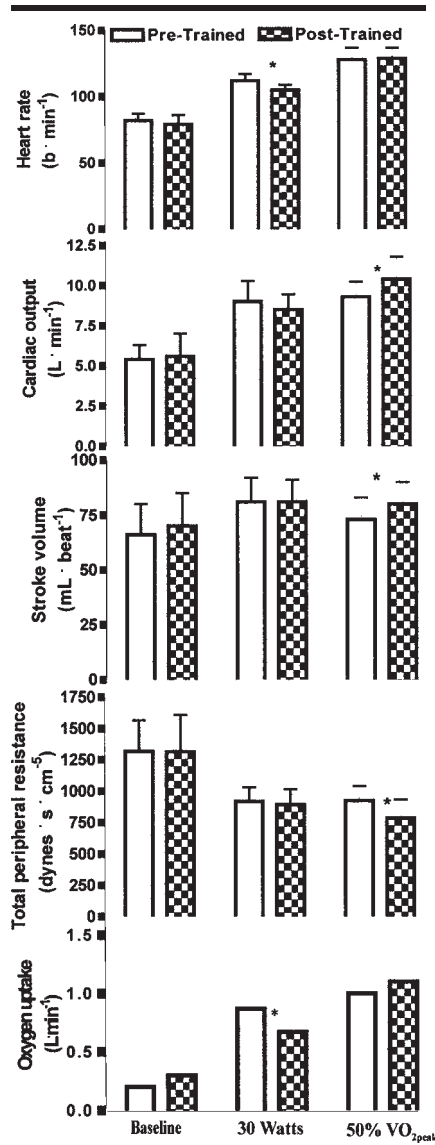


Fig 2. Cardiovascular Responses to Exercise Training. The effects of eight weeks of aerobic exercise measured at rest (baseline), at an absolute workload of 30 W and at 50% of peak oxygen uptake (VO_{2peak}) in African-American women with an exaggerated blood pressure response to exercise

DISCUSSION

Exaggerated exercise blood pressure response (EEBPR) is an independent risk factor for predicting developing hypertension.³ The present study provides evidence that aerobic physical activity may decrease exercise blood pressure in sedentary African-American

women presumed to be at high risk for developing hypertension because of EEBPR. These results agree with the findings of another study conducted by our research team where, in normotensive, young-adult, African-American males, eight weeks of aerobic exercise training attenuated their EEBPR.¹⁰ In the present study, a significant 8.8% increase in VO_{2peak} occurred after eight weeks of moderate exercise training. The post-training systolic and MABP values measured during submaximal exercise were significantly smaller than the pre-training values. In the resting (baseline) condition, measurements of blood pressure and heart rate were not different than those measured after training. This lack of a training-induced change in baseline hemodynamic measures was expected because the study group consisted of normotensive individuals with an average blood pressure of 116/73 mm Hg. This blood pressure value is substantially below the classification of normal blood pressure (120/80 mm Hg) reported in the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure.⁸ However, the absence of a change in resting heart rate after training was unexpected because the conventional effect of aerobic exercise is to decrease resting heart rate. Although our findings showed a trend toward a lower post-training resting heart rate, the limited sample size may have imposed a limitation on our ability to detect this difference with certainty.

During exercise at the absolute workload power output of 30 W, we detected decrements in systolic and MABP and heart rate. This finding is similar to the Heritage Family Study, wherein a decrement in systolic pressure was observed at an absolute workload of 50 W in hypertensive African-American women subjected to 20 weeks of exercise training.¹⁴ We were unable to detect a post-training reduction in

TPR at 30 W. However, exercise at 50% $\text{VO}_{2\text{peak}}$ evoked a post-training reduction in both systolic pressure and TPR. Our definition of EEBPR was based on the systolic pressure change at 50% $\text{VO}_{2\text{peak}}$. Therefore, we were able to detect lowering of TPR at the 50% $\text{VO}_{2\text{peak}}$ level of exercise required to demonstrate the EEBPR and not at 30 W. Exercise at 30 W was associated with significantly lower total oxygen consumption values than exercise at 50% $\text{VO}_{2\text{peak}}$ (Figure 2). These findings suggest that the variability in MABP and cardiac output associated with exercise at 30 W may have been an insufficient workload to stimulate the endothelium-mediated vasodilatory mechanisms thought to protect against EEBPR.

Dysregulation of TPR by a defect in endothelium-derived nitric oxide vasodilation, mediated by cyclic guanosine monophosphate (cGMP), has been described in subjects exhibiting EEBPR.¹⁵ Endothelium-derived nitric oxide activity has been reported to be increased by exercise training,¹⁶ which could, therefore, explain the decrement in TPR and attenuated EEBPR reported herein. In addition, the statistically significant pre- vs post-training systolic pressure values were likely also physiologically significant because hyperreactors were converted to normal reactors after training, which indicates the potential for a reduction in their risk for developing hypertension.

To our knowledge, this article is the first report demonstrating the effects of physical activity on EEBPR in women. These findings are novel and significant because we found that moderate aerobic exercise training attenuated EEBPR in normotensive, African-American women independent of a change in their resting blood pressure values. More than 60% of women in the United States are less active than they should be, and the number of adults, especially women engaging in physical exercise, is continuing to decline.¹⁷ Studies examining

the relationships between levels of physical activity and attitudes toward exercise in African-American women have reported more sedentary behaviors associated with less positive attitudes toward exercise.^{18,19}

In conclusion, the present study demonstrates that a physical activity intervention may attenuate EEBPR. This effect appears to be mediated by decreased TPR in normotensive, young-adult, African-American women presumed to have a high risk for hypertension because of their EEBPR. This finding suggests that African-American women who are capable of a lifestyle change that incorporates regular physical activity may lessen their EEBPR and their risk for developing hypertension. An increase in physical activity may help decrease the excess burden of cardiovascular disease in African-American women. With our current knowledge of negative behaviors and attitudes about physical activity in African-American women, we recommend more basic behavioral and social science research to understand why the lifestyles of African-American women do not involve sufficient physical activity.

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