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Aerobic Exercise Attenuates an Exaggerated Exercise Blood Pressure Response in Normotensive Young Adult African-American Men

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Abstract

An exaggerated exercise blood pressure response (EEBPR) may be associated with an increased risk of hypertension. We hypothesized that aerobic exercise training can decrease EEBPR and the risk for hypertension by decreasing arterial resistance. We studied the effects of aerobic training on the submaximal exercise blood pressure (BP) of eight normotensive young adult African-American men with an EEBPR. Subjects were trained on a stationary bicycle at an intensity of 70% peak oxygen uptake (VO_{2peak}) for 30 min, three times per week, for 8 weeks. BP, heart rate, cardiac output (CO), stroke volume (SV) and total peripheral vascular resistance (TPR) were measured at rest and during submaximal exercise at a work intensity of 50% VO_{2peak} . Significance of the training effects were evaluated by comparing the pre- and post-training measures (t -test, $p < 0.05$). A 15% post-training increase in VO_{2peak} (34.6 ± 1.4 to 40 ± 1.4 ml/kg/min) and a 9.5 ml post-training increase in mean resting stroke volume were found. A 16.2 mmHg decrement in mean systolic BP, an 11.5 mmHg decrement in mean diastolic BP, a 120 dyne/s/cm⁵ decrement in TPR and a 1.2 l/min increase in CO were detected during the post-training submaximal exercise tests. These results suggest that reductions in TPR may attenuate the EEBPR of normotensive African-American males following an 8-week training regimen of stationary bicycling at 70% VO_{2peak} . Aerobic exercise training may, therefore, reduce the risk of hypertension in normotensive African-American males by the mechanism of a reduction in TPR. Because of the limited number of subjects, the results of this study should be interpreted cautiously pending confirmation by a larger controlled trial.

Keywords

blood pressure; exercise training; African-Americans

INTRODUCTION

Hypertension affects over 60 million individuals in the USA and is the leading health problem in the adult African-American community [1]. However, the cause of the predilection for hypertension in African-Americans is uncertain. A recent investigation has shown that the arterial resistance of normotensive African-American youths with a family history of hypertension may be greater than that of a comparable group of Caucasian-Americans [2]. This finding suggests that arterial resistance may differentiate between African-American and other risk groups for hypertension. Because of the enormous impact of this disease, early identification of individuals at increased risk for hypertension may permit alterations of lifestyle behaviors that could reduce the risk for hypertension. An exaggerated blood pressure response to dynamic exercise appears to be a promising approach to early detection [3–5]. Based on a comprehensive review of the scientific literature, Jette *et al.* [4] have concluded that hyper-responders face a 2–10-fold greater probability of developing hypertension. Exaggerated exercise blood pressure responses (EEBPR) have been demonstrated in borderline hypertensives, normotensive individuals with parental histories of hypertension, and African-Americans [3–6]. Although exercise training at a level that increases aerobic functional work capacity appears to decrease the EEBPR of hypertensive subjects [7], no studies appear to have been performed to determine the effects of exercise training on normotensive subjects exhibiting EEBPR. Aerobic exercise seems to decrease arterial resistance generally [8]. However, the arterial resistance of normotensive African-American males seems to be greater than that of a comparable group of Caucasian-Americans during both exercise and rest [6]. Therefore, we hypothesized that aerobic exercise training may attenuate the EEBPR of normotensive African-American males by the mechanism of reducing arterial resistance.

METHODS

Subjects

Twenty-one healthy, normotensive African-American males between 18 and 26 years of age were recruited and then screened for an exaggerated blood pressure response to dynamic exercise. An EEBPR was defined as the calculated acute change in systolic blood pressure from sitting rest to 50% peak oxygen uptake (change in systolic blood pressure = exercise systolic blood pressure—resting systolic blood pressure) ≥ 50 mmHg. Further criteria for inclusion in the study were: non-smoking status, absence of alcohol abuse (less than two standard alcohol drinks a day), resting systolic/diastolic blood pressure $< 140/90$ mmHg, absence of all medication and no regular physical activity during the previous year (sedentary lifestyle). Eight subjects met the criteria and were used in this study. The Godin leisure time exercise questionnaire [9] was used to characterize the subject's physical activity lifestyle. Data obtained from the activity questionnaire were converted to MET-HOURS and a weekly score of 20 was defined as sedentary. The University Human Subjects 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.

Aerobic exercise training protocol

The aerobic program consisted of 8 weeks of bicycling three times per week, 30 min per session, at 70% of peak oxygen uptake ($\text{VO}_{2\text{peak}}$). Warm-up and cool down periods of 3–5 min were included prior to and subsequent to each training session. One of the investigators attended all exercise-training sessions and documented each participant's training intensity, duration and frequency.

Measurements

Aerobic fitness—During the first laboratory session, functional work capacity (aerobic fitness) was assessed during a progressive exercise test using an Ergoline 800S electronic-braked cycle ergometer (SensorMedics Corp., Anaheim, CA). Each subject began by exercising at 30 watts (W) for 3 min. The work rate was then increased by 30 W in successive 3-min stages until volitional exhaustion. Minute ventilation was measured using a FLO-1 volume transducer (PHYSIO-DYNE Instrument Corp., Quogue, NY). Percentage of expired oxygen (O_2) and carbon dioxide (CO_2) were measured using a paramagnetic O_2 analyzer and an infrared CO_2 analyzer connected to an on-line computerized PHYSIO-DYNE system (Quogue, NY) calibrated to known gases. Aerobic fitness ($\text{VO}_{2\text{peak}}$) was determined by recording the highest VO_2 value attained during the progressive exercise test. The same test procedures were carried out at the conclusion of the study to determine if $\text{VO}_{2\text{peak}}$ was altered by the exercise intervention.

Blood pressure and heart rate—Systolic and diastolic pressures were measured at the right arm by a Colin STBP-680 automated blood pressure monitor (Colin Medical Instruments, San Antonio, TX). In an earlier study, we reported that this automated monitor met the criteria established by the Association for the Advancement of Medical Instrumentation for systolic and diastolic blood pressure measures during periods of rest and dynamic exercise [10]. Mean arterial blood pressure (MABP) was calculated using the conventional formula: diastolic pressure + $\frac{1}{3}$ pulse pressure. Heart rates were determined from a CM5 electrocardiographic tracing using the Colin STBP-680 blood pressure monitor.

Cardiac output, stroke volume and total peripheral vascular resistance—

Cardiac output was measured non-invasively by using the CO_2 rebreathing technique of Collier [11]. End-tidal partial pressure of CO_2 (PCO_2) was measured using a rapid-response infrared CO_2 analyzer (PHYSIO-DYNE Instrument Corp., Quogue, NY). End-tidal PCO_2 was used to estimate arterial PCO_2 according to the formula of Jones *et al.* [12].

Each subject rebreathed into a 5-l latex bag containing CO_2 and O_2 to permit rapid equilibration with venous PCO_2 . A valid equilibrium during rebreathing was measured by determining a “plateau” in PCO_2 according to the criteria of Ashton and McHardy [13] and Jones *et al.* [12]. This criteria requires that during the first 6–8 s, an inspiration has to be followed by an expiration whose PCO_2 value is within ± 1 mmHg of the first recorded value. An automated gas mixing apparatus was used to adjust the initial gas volume and initial PCO_2 in the rebreathing bag according to the procedure described by Jones *et al.* [12]. If a trial had to be redone, end-tidal PCO_2 values were allowed to return to baseline values before the procedure was repeated. Cardiac output was calculated from the indirect Fick equation:

$$\text{CO} = V_{\text{CO}_2} / (C_{\text{VCO}_2} - C_{\text{aCO}_2})$$

where CO is cardiac output, V_{CO_2} is expired carbon dioxide, C_{VCO_2} is mixed venous CO_2 content, and C_{aCO_2} is arterial CO_2 content.

Stroke volume was calculated from the formula:

$$\text{Stroke volume} = \text{CO} / \text{heart rate} \times 1000 \text{ (expressed in ml)}$$

Total peripheral vascular resistance (TPR) was calculated from the formula:

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

Submaximal exercise test

Subjects were instructed to abstain from food, caffeine and physical activity for 3 h prior to performing the submaximal exercise test. The submaximal exercise test began with a 10-min sitting rest period during which time measures of systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MABP), heart rate (HR), cardiac output (CO), stroke volume (SV), and TPR were determined at minutes 8, 9 and 10. The mean value of the three measurements was then calculated for each variable. Following the rest period, the subject performed a submaximal exercise work-bout working at an exercise intensity of 50% $\text{VO}_{2\text{peak}}$ for a duration of 8 min. During the submaximal exercise test, steady-state values of SBP, DBP, MABP, HR, CO, SV and TPR were recorded at minutes 6, 7 and 8 of the work-bout. These identical test procedures were then used following the aerobic exercise-training program.

Statistical analysis

The study design consisted of pre- and post-test conditions with the subjects acting as their own control. Data were analyzed utilizing the StatView statistical program (Abacus Concepts, Inc., Berkeley, CA) using a Macintosh IIfx computer. Paired *t*-tests were used to identify pre- to post-test differences. A value of $p \leq 0.05$ was considered statistically significant. All data were expressed as mean $\pm$ SE.

RESULTS

Table I presents the body height, body weight, $\text{VO}_{2\text{peak}}$, peak HR and the resting measures of cardiovascular function in the eight normotensive African-American men who met the eligibility criteria for an EEBPR measured during the pre- and post-training periods of this study. At the beginning of the study, the subjects were found to have a mean age of 19.8 ± 0.3 years and a SBP change of 54 ± 1 mmHg at 50% $\text{VO}_{2\text{peak}}$ (average metabolic equivalent of 13.7 ± 0.2 ml/kg/min). A significant (14.9%) increase in aerobic capacity between pre-training ($\text{VO}_{2\text{peak}} = 34.6 \pm 1.4$ ml/kg/min) and post-training ($\text{VO}_{2\text{peak}} = 40.7 \pm 1.4$ ml/kg/min) conditions was detected. Following training, the resting SBP, DBP, MABP, HR, CO and TPR values were found to be unchanged. A significant increase in resting SV was detected after training (75.0 ± 5.7 vs 84.5 ± 6.8 ml).

Figures 1 and 2 summarize the cardiovascular responses during the submaximal exercise tests for the pre- and post-training conditions. The submaximal exercise test workload at 50% $\text{VO}_{2\text{peak}}$ was found to be 16.7% greater after training than before training (112.5 ± 7.5 vs 93.7 ± 8.8 W). The mean post-training values for submaximal exercise SBP, DBP and MABP at the same relative power output of 50% $\text{VO}_{2\text{peak}}$ were found to be 16.2, 11.5 and 13 mmHg lower than the pre-training values, respectively. The reductions in submaximal exercise blood pressures were not associated with changes in HR (128 ± 4.8 vs 128.8 ± 3.9 beats/min). A significant increase in the pre- vs post-training CO (14.2 ± 7.1 l/min vs 15.4 ± 0.8 l/min) and a significant decrease in the TPR (659 ± 104 dyne/s/cm⁵ vs 539 ± 86 dyne/s/cm⁵) were also detected.

DISCUSSION

The present study examined the effects of aerobic exercise training on an EEBPR in normotensive young adult African-American men. This type of hemodynamic response may increase one's predisposition for developing hypertension [3–5]. In the Healthy People 2000 National Health Promotion and Disease Prevention Report [14], it is acknowledged that although much is known about the physiological and biochemical benefits of regular physical activity, very little is known about the effects of exercise in minority populations. Indeed, the present study appears to be the first to examine the influence of regular physical activity on an EEBPR in normotensive African-Americans. As a strategy to reduce the incidence of hypertension in susceptible individuals, the American College of Sports Medicine [15] recommends regular physical activity at a level of intensity that promotes an increase in the aerobic functional work capacity. This recommendation suggests that regular physical activity may decrease the blood pressure response to dynamic work in individuals who are susceptible to hypertension.

In the present study, we hypothesized that an EEBPR to submaximal exercise could be attenuated by an aerobic exercise-training regimen that produced an increase in functional aerobic capacity. Our exercise intervention was found to be effective because the study subjects exhibited a 14.9% increase in functional aerobic capacity after 8 weeks of three work-bouts per week on a stationary bicycle performed at a workload of 70% $\text{VO}_{2\text{peak}}$. This training intervention was not associated with significant changes in the baseline resting BP values of the study subjects. This finding is consistent with those of Gilders *et al.* [16] and Tipton [17] who studied the resting BP values of Caucasian-Americans after aerobic training. Our results do not agree with the findings published by the HERITAGE Family Study [18] claiming that aerobic training was associated with significant reductions in resting SBP values in African-Americans. This difference may be attributed to the fact that the HERITAGE study used both normotensive and mildly hypertensive individuals as subjects while we examined only normotensive individuals. In the present study, there were no changes in resting BP and CO. The post-training resting data showed an 11.7% increase in SV ($p < 0.05$) and, although not statistically significant, a decrement in resting HR and an increment in resting DBP approaching significance ($p = 0.09$). Resting DBP is used as a measure of TPR and the post-training resting TPR was not different than the pre-training control. This finding diminishes the potential physiological significance of the resting DBP and points out why TPR needs to be measured independently. By itself, the trend of increased resting DBP may have been misinterpreted to suggest the potential for a training effect that could be considered adverse. On the other hand, a trend toward increased resting DBP coupled with an unchanged (or slightly decreased) resting SBP may be indicative of a trend toward decreased pulse pressure (PP). PP is considered by some to be the variable that provides the best estimate of the pressure component of arterial compliance [19]. In view of the significant increase in resting SV documented herein, an estimation of the pre-training/post-training arterial compliance change measured at rest may be indicative of an improvement in vascular structure–function relationships that could contribute to lessening blood pressure hyper-reactivity. A limitation of the present study is the absence of a control group and limited sample size that may have helped clarify whether or not a trend toward increased resting DBP was unique to the blood pressure hyper-responders. Therefore, the results of this investigation should be interpreted cautiously pending confirmation by a larger controlled study.

The decremental BP response to submaximal exercise at 50% $\text{VO}_{2\text{peak}}$ following aerobic training that we have documented herein supports the hypothesis that an EEBPR can be attenuated by aerobic training. At this work intensity, the mean SBP, DBP and MABP values of the study subjects were found to be 16.2, 11.5 and 13 mmHg lower, respectively,

after training. These findings are consistent with those of Wilmore *et al.* [18] who have reported that endurance training of normotensive and mildly hypertensive African-American men was associated with significant decrements in DBP and MABP during submaximal exercise at 60% of maximal of maximal oxygen uptake.

According to Ohm's law, reductions in blood pressure should be accompanied by reductions in CO and/or TPR. Increases in the post-training absolute power output and the CO values at 50% $\text{VO}_{2\text{peak}}$ were accompanied by a concomitant 18.1% reduction in TPR. Therefore, the reduction in submaximal exercise TPR appears to be the main mechanism for producing the lower BP values associated with dynamic exercise following aerobic training. Wilmore *et al.* [18] also reported a decrement in the BP response to submaximal exercise following training in African-Americans; however, they did not compute TPR.

Exploration of the mechanisms underlying the training induced reductions in submaximal exercise TPR was beyond the scope of the present investigation. However, we believe that increased endothelial nitric oxide synthesis [20], with concomitant vascular smooth muscle relaxation [21], and increased arterial compliance [22] offer the most plausible explanations for the decrements in submaximal exercise TPR associated with 8 weeks of aerobic exercise training.

In summary, we found that that aerobic exercise training (stationary bicycling) performed at a moderate level of intensity for 8 weeks increased the aerobic capacity and decreased the submaximal exercise TPR of a group of eight normotensive young adult African-American men who demonstrated an exaggerated systolic blood pressure response to exercise. These findings suggest that aerobic exercise may be an effective antihypertensive therapy for African-American men at risk for hypertension.

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Fig. 1.

Changes in pre-and post-training heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial blood pressure (MABP) values during exercise at 50% peak oxygen uptake ($\dot{V}O_{2peak}$), † $P < 0.05$.

Fig. 2. Changes in pre-and-post-training cardiac output (CO), stroke volume (SV), and total systemic vascular resistance (TPR) values during exercise at 50% peak oxygen uptake ($\dot{V}O_{2peak}$), † $p < 0.05$.

Table I

Mean values for body weight, height, heart rate, blood pressure, cardiac output, stroke volume, total peripheral resistance and $\text{VO}_{2\text{peak}}$ values during pre-and post-exercise training periods

Variable	Pre-trained	Post-trained
Height (cm)	178 $\pm$ 1.8	—
Weight (kg)	79.9 $\pm$ 5.3	79.2 $\pm$ 4.9
HR_{rest} (beats/min)	74.2 $\pm$ 3.5	68.7 $\pm$ 2.9
SBP_{rest} (mmHg)	129.5 $\pm$ 2.0	125.5 $\pm$ 3.6
DBP_{rest} (mmHg)	71.8 $\pm$ 2.0	78.3 $\pm$ 2.3
$\text{MABP}_{\text{rest}}$ (mmHg)	89.5 $\pm$ 2.0	95.2 $\pm$ 1.5
CO_{rest} (l/min)	5.5 $\pm$.4	5.7 $\pm$.4
SV_{rest} (ml)	75.0 $\pm$ 5.7	84.5 $\pm$ 6.8 ^a
TPR_{rest} (dyne s/cm ³)	1434.3 $\pm$ 105	1299 $\pm$ 107
HR_{peak} (beats/min)	189 $\pm$ 2.7	190 $\pm$ 2.5
$\text{VO}_{2\text{peak}}$ (ml/kg/min)	34.6 $\pm$ 1.4	40.7 $\pm$ 1.4 ^a

^aIndicates significant difference between treatment periods ($p < 0.05$).

SBP_{rest} , rest systolic blood pressure; DBP_{rest} , rest diastolic blood pressure; $\text{MABP}_{\text{rest}}$, rest mean arterial blood pressure; CO_{rest} , rest cardiac output; SV_{rest} , rest stroke volume; HR_{peak} , peak heart rate; $\text{VO}_{2\text{peak}}$, peak oxygen uptake.