

# ORIGINAL REPORTS: CARDIOVASCULAR DISEASE AND RISK FACTORS

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## ACUTE EXERCISE AND POSTEXERCISE BLOOD PRESSURE IN AFRICAN AMERICAN WOMEN

**Background:** Limited data suggest that physical activity increases postexercise blood pressure in African-American women. The purpose of this study was to evaluate the postexercise blood pressure response to acute exercise in normotensive young adult African-American women.

**Methods:** Eight healthy women (age  $22.5 \pm 0.9$  years) performed a cycle ergometer bout of 30 minutes at 60% of peak ventilatory oxygen uptake ( $VO_2$  peak). Control arterial blood pressure, heart rate, lower leg blood flow, cardiac output, spectral analysis of blood pressure, heart rate variability, and baroreceptor sensitivity were measured for 5 minutes before exercise and were compared to postexercise measurements performed at rest intervals of 15–20, 35–40 and 55–60 minutes after exercise.

**Results:** Exercise performed at 60%  $VO_2$  peak produced an arterial pressure of  $172 \pm 10/70.1 \pm 4.0$  mm Hg. Postexercise recovery values were not significantly different than the baseline control values.

**Conclusion:** These results do not support the hypothesis that acute physical activity exerts an adverse effect on postexercise blood pressure in African American women. (*Ethn Dis.* 2007;17:664–668)

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

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### INTRODUCTION

Improvements in cardiovascular risk factor profile, cardiovascular morbidity and mortality,<sup>1</sup> and all-cause mortality<sup>2</sup> have been proposed as significant factors contributing to the long-term beneficial effects of physical activity. Postexercise reduction in blood pressure, wherein arterial pressure decreases below the pre-exercise control value, is also thought to contribute to the long-term benefits of exercise.<sup>3</sup> A reduction in postexercise systemic vascular resistance has been reported after maximal exercise.<sup>4</sup> On the other hand, no change in postexercise forearm vascular resistance has been observed after submaximal exercise.<sup>5</sup> High postexercise cardiac output and stroke volume values have been reported after submaximal and maximal supine bicycle exercise.<sup>6</sup> A neural hypothesis for explaining postexercise reduction in blood pressure is suggested by findings of decreased postexercise sympathetic nerve activity of the lower leg muscles in borderline hypertensive subjects<sup>7</sup> and by increased postexercise sympathetic modulation of heart rate in heart rate variability studies of normotensive subjects.<sup>8,9</sup> Postexercise reduction in blood pressure has been attributed to changes in baroreceptor sensitivity in some studies,<sup>10,11</sup> but an absence of such changes in baroreceptor activity has also been reported.<sup>12,13</sup>

Postexercise reduction in blood pressure may occur within the initial minutes after exercise<sup>14–16</sup> or at some

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point within 30–60 minutes of stopping exercise.<sup>7,12,17–21</sup> Postexercise reduction in blood pressure has also been demonstrated after a single bout of aerobic exercise in young, middle-aged, and older subjects.<sup>22</sup> Detection of postexercise reduction in blood pressure has been reported to be less consistent in normotensive individuals than in hypertensive individuals<sup>23–25</sup> and both absent and present in normotensive, premenopausal women.<sup>23,24,26</sup> In contrast, normotensive, premenopausal African American women exhibited a hyperreactive blood pressure response after a single bout of exercise.<sup>23</sup>

These findings suggest a neural cardiovascular control mechanism, which suggests that postexercise reduction in blood pressure may be absent in African-American women. The present study was, therefore, designed to examine the blood pressure and heart rate variability of normotensive, premenopausal African American women before and at intervals after a session of submaximal exercise.

## METHODS

### Subjects

Eight healthy African American women volunteered to participate in the study. All participants were premenopausal, not taking any medications (such as oral contraceptives), nonsmoking, and abstaining from alcohol. No participant engaged in regular physical activity. Participants were excluded if the average of three sitting systolic and diastolic blood pressure readings on separate days was  $\geq 140$  and 90 mm Hg, respectively. Blood pressure screenings were performed by a research assistant using a SunTech Tango (SunTech Medical Inc., Raleigh, NC) automated blood pressure monitor. The institutional review board at Howard University granted ethical approval, and informed consent was obtained from all subjects before they participated in the study.

### Study Protocol

Subjects participated in three separate sessions in the laboratory. The first session was for the participant to perform the peak oxygen uptake ( $\text{VO}_{2 \text{ peak}}$ ) test and dual energy x-ray absorptiometry measure of body composition. The two subsequent laboratory visits consisted of a control session of 60 minutes of supine rest or an experimental session which consisted of 30 minutes of cycling at 60%  $\text{VO}_{2 \text{ peak}}$ , followed by 60 minutes of supine rest. The control session and experimental sessions were randomly assigned and performed during the luteal phase of the participant's menstrual cycle. The laboratory was maintained at 21°C, and the participants were instructed not to eat and restrict physical activity for 4 hours before the study sessions.

### Peak Oxygen Uptake Test Session

$\text{VO}_{2 \text{ peak}}$  was measured during a standardized incremental cycle task with a SensorMedics Ergoline 800 ergometer (SensorMedics Corp., Yorba Linda, CA).

Subjects were asked to cycle continuously at 70–75 rpm, at a starting work rate of 25 W. The work rate was increased by 25 W every 3 minutes until volitional fatigue. During the incremental exercise test, expired gas fractions of  $\text{VO}_2$ , carbon dioxide, and minute ventilation were measured using the method of open-circuit spirometry (Max II metabolic system, Physio-Dyne, Quogue, NY). The  $\text{VO}_2$  value achieved during the last minute of the incremental exercise test was defined as  $\text{VO}_{2 \text{ peak}}$ .

### Experimental Test Session

Continuous blood pressure was measured noninvasively using an application tonometer (Colin Medical Instruments Corp., San Antonio, TX) that was positioned and secured on the skin over the subject's left radial artery. Heart rate data were collected from an electrocardiogram signal simultaneously with blood pressure. Respiratory excursions were also recorded by means of a piezoelectric thoracic belt. The three analog signals were sampled at 500 Hz/channel using a BioPac MP150 data acquisition system (BIOPAC Systems, Inc., Goleta, CA) and stored for subsequent analyses. Baseline blood pressure, heart rate, and respiration measures were collected during the last 5 minutes of a 10-minute supine rest position. After baseline recordings, the participants performed 30 minutes of submaximal exercise on the cycle ergometer at a work intensity of 60%  $\text{VO}_{2 \text{ peak}}$ . After exercise, each participant was placed in a postexercise supine rest position for 60 minutes. Postexercise measurement of blood pressure, heart rate, and respiration were recorded across the following time intervals: 15–20, 35–40, and 55–60 minutes. Averages of the baseline and postexercise data recordings were used for statistical analysis.

Estimates of cardiac output were made using a SORBA model CIC-1000 impedance electrocardiography system, software version 7.2 (SORBA Medical Systems, Inc., Brookfield WI). Participants were prepared with skin electrodes

placed on the forehead and on the left side of the body at the base of the neck, along the mid-axillary line at the level of the xiphoid process, and along the mid-axillary line on the upper thigh. The SORBA system continuously sampled signals for cardiac output that were collected and averaged across the aforementioned baseline and postexercise time intervals.

Power spectral analysis of heart rate variability (HRV) and blood pressure variability (BPV) was used to derive measures of autonomic modulation.<sup>27</sup> Power spectra of R-R intervals of 0.15 to 0.4 Hz were defined as the high-frequency (HF) component of HRV (denoted as  $\text{HF}_{\text{RR}}$ ), representing primarily parasympathetic modulation. The low-frequency (LF) component of HRV (0.04–0.14 Hz) is a mixture of both parasympathetic activity and sympathetic activity. Unlike parasympathetic activity, the sympathetic activity is not easily separated from the power spectrum of HRV.<sup>28</sup> Sympathovagal balance was computed as the ratio between the LF and HF spectra of HRV.<sup>29</sup> BPV in the LF range of 0.04 to 0.15 Hz represents sympathetic vasomotor activity.<sup>30,31</sup> All spectral data were log transformed to remove skewness and minimize the large standard deviations customarily present in these data. The digitized R-R intervals and in-phase systolic peaks obtained after the spectral analysis were used to determine baroreflex sensitivity through the index method. This method is based on the calculation of the transfer function or modulus between systolic blood pressure and pulse interval powers in the LF band (0.04 to 0.14 Hz), where the coherence between the two signals is highest. The cross-spectrum between pulse interval and systolic blood pressure (alpha index) was computed in the frequency regions defined as LF (0.1 Hz) and HF (0.3 Hz) bandwidths.<sup>32</sup> Spectral analysis was carried out using five-minute data collection at baseline and during the varying postexercise rest periods. Studies of HRV, BPV, and baroreflex sensitivity were determined using the Nevrokard software (Medistar, d.o.o., Ljubljana, Slovenia).

**Table 1. Physical characteristics of study subjects (N=8)**

Variable	Findings
Age (yr)	22.5 ± 0.9
Height (cm)	166.8 ± 3.1
BMI (kg · m <sup>-2</sup> )	26.8 ± 1.6
Weight (kg)	75.5 ± 5.9
Body fat (%)	37.6 ± 2.7
VO <sub>2peak</sub> (ml · kg <sup>-1</sup> · min <sup>-1</sup> )	27.1 ± 1.6
HR <sub>peak</sub> (b · min <sup>-1</sup> )	188.7 ± 2.6
HR <sub>rest</sub> (b · min <sup>-1</sup> )	64.1 ± 4.2
SBP <sub>rest</sub> (mm Hg)	107.5 ± 3.9
DBP <sub>rest</sub> (mm Hg)	70.2 ± 2.6

Values are means ± SE. BMI, body mass index; VO<sub>2 peak</sub>, peak oxygen uptake; HR<sub>peak</sub>, heart rate peak; HR<sub>rest</sub>, heart rate rest; SBP<sub>rest</sub>, systolic blood pressure rest; DBP<sub>rest</sub>, diastolic blood pressure rest.

Lower leg blood flow was measured with a noninvasive Hokanson EC-6 Plethysmograph, (Hokanson Inc., Bellevue, WA). In the supine position the subject's left heel was elevated above the level of the heart on a foam block to enable venous drainage. Occlusion cuffs were applied to the ankle (arterial cuff) and to the lower thigh 5 cm proximal to the knee joint (venous cuff). A mercury-filled silastic rubber strain gauge, the circumference of which was 1–2 cm less than the widest circumference of the lower limb, was applied in a double-loop fashion at the widest girth of the calf muscle. The mercury-filled strain gauge was fixed in position with tape. Subjects were asked to remain motionless during the measurement period. With the arterial cuff inflated to 200 mm Hg, the venous cuff was inflated to 50 mm Hg for 10 seconds and deflated for five seconds intermittently, for a total of 60 seconds during the last minute of the baseline and postexercise rest recordings. Lower leg

blood flow was determined using NIVP3 software (Hokanson Inc., Bellevue, WA) as the slope of the increase in limb circumference across time.<sup>33</sup>

**Control Test Session**

The control session consisted of blood pressure measurements in the absence of physical exercise. The subject was placed in the supine rest position, which consisted of a 10-minute baseline period followed by 60 minutes of continuous rest. During the baseline period, blood pressure was measured at minutes 5 through 10. After baseline blood pressure recording, resting blood pressure was continuously measured at the following time intervals: 15–20, 35–40, and 55–60 minutes.

**Data Analysis**

All data were expressed as means plus or minus standard errors. A one-way repeated measures analysis of variance was used to evaluate differences in heart rate, blood pressure, cardiac output, HRV, BPV, baroreflex sensitivity, and lower leg blood flow at baseline and postexercise measurement intervals. For significant main effect, post hoc assessment with individual time point comparisons was carried out using the Newman-Keuls F test. Significance for all analyses was set at *P*<.05.

**RESULTS**

Table 1 summarizes the physical characteristics of the study subjects. Values for the percentage of body fat and body mass index indicate that the subjects were mostly overweight or obese.

The maximum workload attained during the incremental exercise test was 135±4.5 W, and heart rate increased from 64±4.2 to 188.7±2.6 b · min<sup>-1</sup>. During steady state work at an exercise intensity of 60% VO<sub>2 peak</sub> (65±4.5 W), the heart rate and blood pressure were 142.1±1 b · min<sup>-1</sup> and 172±10/70.1±4.0 mm Hg, respectively.

Average postexercise systolic and diastolic blood pressure values at 20, 40, and 60 minutes were not significantly different than the baseline blood pressure values (Table 2). These values were similar to those of the exercise session.

Table 3 illustrates the central and peripheral hemodynamic responses and neural responses at baseline and across all time points following exercise. The postexercise heart rate during the 15- to 20-minute interval was significantly higher than the baseline value (66.5±3.2 vs 75.1±3.7 beats/minute).

**DISCUSSION**

The main finding of the present study is that among normotensive, young adult, overweight or obese African American women, blood pressure after a session of exercise at 60% of VO<sub>2 peak</sub> was not reduced and did not differ from that of control subjects who did not exercise. Our results suggest that postexercise reduction in blood pressure is absent in normotensive, premenopausal African American women. This finding is in agreement with similar studies of non-African American women demonstrating that exercise does not reduce blood pressure acutely.<sup>24,34</sup> However, in other studies, the average

**Table 2. Blood pressure values (mean ± SE) during control and experimental exercise sessions**

Blood Pressure	*Post-control Baseline	*Post-control 20 min	*Post-control 40 min	Pre-exercise 60 min	Postexercise Baseline	Postexercise 20 min	Postexercise 40 min	Postexercise 60 min
Systolic (mm Hg)	102.0 ± 2.9	102.6 ± 3.5	102.6 ± 4.1	102.0 ± 2.2	108.8 ± 5.3	112.5 ± 5.6	112.8 ± 3.6	110.6 ± 3.9
Diastolic (mm Hg)	57.8 ± 2.2	59.7 ± 2.1	60.5 ± 2.1	58.8 ± 2.7	60.5 ± 3.9	57.6 ± 3.4	56.5 ± 2.7	54.7 ± 3.3

\* Post-control measurements were made at intervals after the baseline control.

**Table 3. Mean ( $\pm$  SE) neural responses and central and peripheral hemodynamic values at baseline and during postexercise (N=8)**

Variable	Pre-exercise Baseline	Postexercise 20 min	Postexercise 40 min	Postexercise 60 min
Heart rate (b·min <sup>-1</sup> )	64.1 $\pm$ 3.2	75.1 $\pm$ 3.7*	70.6 $\pm$ 4.3	70.7 $\pm$ 4.4
Heart rate variability				
HF, nu	62.3 $\pm$ 7.4	57.5 $\pm$ 7.0	57.7 $\pm$ 7.0	57.7 $\pm$ 7.0
LF, nu	41.8 $\pm$ 8.0	71.7 $\pm$ 34	72 $\pm$ 35	72.2 $\pm$ 35
LF/HF	0.84 $\pm$ 0	2.8 $\pm$ 2.2	2.9 $\pm$ 2.3	2.9 $\pm$ 2.3
Blood pressure variability				
LF, nu	95.3 $\pm$ 16	92.8 $\pm$ 18	89.8 $\pm$ 20	90.4 $\pm$ 20
BRS	8.4 $\pm$ 2.4	4.8 $\pm$ 1.0	7.8 $\pm$ 1.5	6.8 $\pm$ 1.2
Cardiac output (L · min <sup>-1</sup> )	4.7 $\pm$ 0.3	4.5 $\pm$ 0.4	4.8 $\pm$ 0.5	4.9 $\pm$ 0.5
LLBF (ml · 100 ml <sup>-1</sup> · min <sup>-1</sup> )	7.4 $\pm$ 1.9	8.2 $\pm$ 1.6	7.7 $\pm$ 1.8	7.2 $\pm$ 1.1

\* Significantly different than control

HF, high frequency; LF, low frequency; nu, normalize unit; BRS, baroreceptor sensitivity; LLBF, lower leg blood flow.

decrease in systolic and diastolic blood pressure was 8/9 mm Hg<sup>4,19,35-38</sup> in normotensive subjects. While the post-exercise blood pressure did not decrease in the present study, postexercise diastolic blood pressure at the 55- to 60-minute interval was 5.8 mm Hg lower than the baseline value, which may be of significance.

In contrast to both our results and those of others, Pescatello et al<sup>23</sup> reported an increase in postexercise blood pressure in normotensive, premenopausal African-American women.

In summary, we found that young adult, normotensive, African-American women exercising at an intensity of

60% VO<sub>2 peak</sub> for 30 minutes did not exhibit postexercise reduction in blood pressure for 60 minutes after exercise. Future studies should determine whether the absence of postexercise reduction in blood pressure may be a physiological marker for the predilection for cardiovascular disease in this high-risk population.

**ACKNOWLEDGMENTS**

This study was supported by a grant from the Howard University Mordecai Wyatt Johnson Fund (V. Bond, PI, and R.M. Millis, Co-PI).

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## POSTEXERCISE BLOOD PRESSURE IN AFRICAN AMERICANS - Enweze et al

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