BLOOD PRESSURE RESPONSES OF SEDENTARY AFRICAN AMERICAN WOMEN DURING CYCLE AND TREADMILL EXERCISE

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The purpose of this study was to evaluate the BP responses of African American women at two exercise intensities on two different modes of exercise.

Although African Americans display augmented BP reactivity to various stressors, little research has been conducted on a potential mode dependency exercise BP response in African Americans. Moreover, because women may show greater BP reactivity than men, research with African American women may be particularly informative. Therefore, the purpose of this study was to evaluate the BP responses of African American women at two exercise intensities on two different modes of exercise. Two questions were asked. First, at fixed HR values for a given intensity, do the BP responses of African American women depend on the mode (cycle vs treadmill) and intensity (low vs moderate) of exercise? Second, what is the relationship between baseline SBP and the magnitude of SBP reactivity for cycle and treadmill exercise in this population?

METHODS

Subjects
Forty-four African American women volunteered to participate. The number of subjects recruited was based on a power estimate of .80 to detect a potential exercise mode by intensity SBP interaction. Criteria for participation included nonsmoking, 20 to 45 years of age, physically inactive as
assessed by global items on the modified Community Healthy Activities Model Program for Seniors (CHAMPS) Physical Activity Questionnaire, \(^{13}\) resting BP <140/90 mm Hg, \(^{14}\) absence of medication, and absence of any medical or orthopedic condition aggravated by exercise. Subjects were asked to restrict food and caffeine for three hours before testing. Two subjects did not comply with the caffeine restriction and were rescheduled. The study protocol conformed to the ethical guidelines of the Declaration of Helsinki. \(^{15}\)

**Exercise Testing**

Subjects reported to the laboratory where they read and signed a university-approved informed consent, were weighed (kg) and had their height (cm) measured (Seca 700 beam scale, Brooklyn, NY), and were fitted with a HR monitor (Polar E200, Lake Success, NY). Subjects then completed a medical history and the modified CHAMPS, \(^{13}\) allowing for a 10-minute seated rest period, after which baseline HR and BP were assessed. Systolic blood pressure (SBP) and DBP were measured at rest and during exercise by manual auscultatory cuff sphygmomanometry (Omron Sprague Rappaport stethoscope and nylon cuff, Bannockburn, Ill) by using standardized procedures. \(^{16}\) All subjects exercised on a cycle ergometer (Monarch 818E, Varberg, Sweden) and treadmill (Landice L9, Randolph, NJ) during one visit.

To control for a potential exercise mode order effect, subjects were assigned an exercise mode sequence in a pseudorandom manner based on recruitment (ie, subject one treadmill first, subject two cycle first, etc). Low- and moderate-intensity target HR values were calculated for the initial mode by using the heart rate reserve \(^{17}\) (HRR) method with adjustments to the HRR formula \(^{18,19}\) to improve the estimate of 40% and 60% maximal oxygen consumption (VO\(_{2\text{max}}\)) for cycle or treadmill exercise.

For the cycle ergometer, subjects pedaled at 60 revolutions per minute (rpm) with the power output increased by 30 watts (W) every two minutes until HR targets were attained. To control for the effect of isometric hand gripping on BP, subjects were instructed to lightly rest their palms on the cycle handlebar without gripping. For the treadmill, subjects walked at three miles per hour (mph) with 1% or 2% grade increases every two minutes until HR targets were reached. Cycle and treadmill calibration settings were checked every five tests.

During exercise, once HR reached a plateau within ±5 beats of the low-intensity target (40% HRR), rating of perceived exertion \(^{20}\) (RPE) and BP were measured. One minute later, HR was rechecked for steady state, and BP was measured a second time. The workload was then increased until the HR stabilized within ±5 beats of the moderate-intensity target (60% HRR), and the data collection procedure was repeated followed by a three-minute cool down. Between bouts, subjects rested quietly in a seated position for 10 minutes and began the second exercise bout after HR and BP returned to within ±5 of resting values. The HR values recorded during the first exercise bout served as targets for the second bout. \(^{8}\) After the second bout, subjects again rested quietly for 10 minutes, and HR and BP were measured a final time. Water and towels were provided during recovery. All baseline, exercise, and recovery HR and BP assessments were taken twice separated by a one-minute interval. Average baseline values were used for target HR calculations and average exercise plateau values for cardiovascular response calculations in all statistical analyses.

**STATISTICAL ANALYSES**

**Preliminary Analyses**

Data were analyzed by using SPSS version 12.0 (SPSS, Chicago, Ill). A \(P\) value ≤.05 was considered statistically significant. To verify whether pseudorandom assignment to an exercise mode sequence had controlled for order effects on the dependent variables, independent \(t\) tests were conducted that compared the BP responses of subjects assigned to the cycle first vs subjects assigned to the treadmill first. For each variable, four \(t\) tests were conducted corresponding to the four mode-by-intensity data collection points: cycle low, cycle moderate, treadmill low, and treadmill moderate. Holm’s Bonferroni procedure \(^{21}\) was used to adjust the \(\alpha\) level for multiple \(t\) tests. No between-group differences were detected for SBP \((P> .05)\) or DBP \((P> .05)\). In a similar manner, independent \(t\) tests were performed for HR and RPE, and no differences were found \((P> .05)\). Therefore, order effects did not appear to influence the data.

**Mode-Intensity BP Differences**

Separate 2 (mode: cycle and treadmill) \(\times\) 2 (intensity: 40% and 60% HRR) repeated measures analyses of covariance (ANCOVA) were used to examine SBP and DBP reactivity, respectively. Blood pressure (BP) reactivity was calculated by using the following formula: \([\% = \left(\frac{X_{\text{stressor}} - X_{\text{resting}}}{X_{\text{resting}}} \right) \times 100])\). \(^{22,23}\) Baseline values served as covariates. Because age and BMI are related to hypertension in this population, \(^{24}\) these variables were also controlled for in the analysis and thus served as additional covariates. Tukey’s procedure \(^{25}\) was used to screen for outliers. No outliers were found.

**Regression Analyses**

Regression analyses were fitted for 60% HRR SBP reactivity with age, BMI, and baseline SBP as independent variables. Partial correlation coefficients were computed. Assumptions were tested by using standardized procedures. \(^{26}\) No outliers were detected. Examination of normality led to a log\(_{10}\) transformation of age and BMI. Tests of other
assumptions were unremarkable. No evidence of multicollinearity was present as tolerance values for all independent variables were >.9.  

RESULTS

Mode-Intensity BP Differences

Table 1 displays subject descriptive characteristics. The $2 \times 2$ repeated measures SBP ANCOVA showed significant main effects for mode ($P \leq .05$) and intensity ($P \leq .05$), but no significant mode $\times$ intensity interaction ($P > .05$). These results indicate that independent of baseline SBP, BMI, and age, SBP reactivity was higher during cycling compared to treadmill exercise at both levels of intensity, and SBP reactivity was higher at 60% HRR compared to 40% HRR regardless of mode (see Figure 1). The $2 \times 2$ repeated measures DBP ANCOVA revealed a significant main effect for mode ($P \leq .05$), which indicated that independent of baseline DBP, BMI, and age, DBP reactivity was higher during cycling compared to treadmill exercise regardless of intensity. No other significant effects were found (see Figure 2). In sum, SBP and DBP were consistently higher on the cycle.

Regression Analyses

In Table 2, baseline SBP contributed to the model at 60% HRR, $R^2 = .66$, $R^2_{adj} = .44$, $F(3,40) = 10.55$, $P \leq .001$. This model accounts for 66% of the variance in cycle SBP reactivity at 60% HRR. A negative correlation independent of age or BMI, indicated that higher baseline SBP predicts lower SBP reactivity for both modes ($P \leq .01$).

DISCUSSION

This study compared BP during low- and moderate-intensity cycle and treadmill exercise in a volunteer sample of sedentary African American women. Results indicate that SBP reactivity increased from 40% to 60% HRR and was higher for cycle than treadmill exercise, while DBP reactivity was higher for cycling, but remained unchanged from 40% to 60% HRR. Regression analyses revealed that only baseline SBP significantly contributed to the prediction of 60% HRR for SBP. Several factors might account for the higher SBP and DBP reactivity during cycling. For example, in contrast to treadmill exercise, cycling is associated with decreased mechanical efficiency, greater non-exercising muscle vasoconstrictive responses, smaller working muscle mass offering greater resistance to blood flow, and decreased vagal stimulation. Additionally, unlike treadmill exercise, as intensity increases, cycling produces greater lower body intramuscular tension and greater upper body isometric contraction.

![Figure 1. Plot of mean SBP reactivity for cycle and treadmill modes at 40% and 60% intensities. SBP=systolic blood pressure;HRR=heart rate reserve.](image-url)
Another, albeit speculative, explanation is that more participants were working above their lactate threshold during higher intensity cycling, as SBP tends to increase non-linearly above the lactate threshold,\textsuperscript{32} and thresholds tend to differ between exercise modes.\textsuperscript{8} However, cycle and treadmill HR and RPE were nearly identical at the time of BP assessments, so SBP mode differences may not be entirely related to changes in blood lactate. Finally, BP response differences between cycling and treadmill exercise may be related to mechanical factors interacting with the increased blood volume and left ventricular volume overload associated with higher BMI.\textsuperscript{33} That is, since cycling offers greater resistance to blood flow, the higher reactivity associated with cycling may be an expression of the underlying pathology associated with the development of future hypertension in some individuals.

The present findings are in contrast to those of Fahrenberg et al.,\textsuperscript{34} who found baseline SBP positively related to reactivity for recumbent cycling in male normotensives and borderline hypertensives exercising at a fixed intensity of 100 Watts. In the present study, relative intensities were used to control for between-subject threshold levels. A discrepancy exists where those with higher baseline BP did not display higher levels of exercise BP reactivity.

The higher cycling SBP mean reactivity at 60% HRR invalidates assumptions that SBP responses will be the same for the cycle and treadmill at a given HR at moderate exercise intensity, a result in line with those of Kelly et al.\textsuperscript{8} Since most exercise programs are designed around a target HR, differences in SBP mode responses must be understood to avoid exaggerated SBP and associated complications in hypertensive-prone individuals such as sedentary African American females.

In interpreting the results, readers should consider some limitations. First, the modified CHAMPS\textsuperscript{13} responses were based on physical activity recall, and data based on recall should warrant some caution. Second, although inten-

| Table 2. Multiple regression analyses with SBP reactivity as dependent variable |
|---------------------------------|----------------|---------------|----------------|----------------|
|                                | Cycling 60% HRR | Treadmill 60% HRR |
| % correct=66                   | % correct=16    |                |
| $R^2=.66$                      | $R^2=.16$       |                |
| Independent variables          | 95% CI          | P value        | 95% CI        | P value        |
| Age                            | (-3.91 to 46.09)| NS            | (-18.66 to 29.42) | NS           |
| BMI                            | (-1.43 to 55.69)| NS            | (-12.43 to 44.61) | NS           |
| SBP baseline                   | (-.99 to -.50)  | .002          | (-.59 to .09) | .01           |

HRR=heart rate reserve; % correct=percentage correctly predicted; 95% CI=95% confidence interval; NS=not significant.
sity was established using the HRR along with appropriate cycle and treadmill correction formulas.\textsuperscript{18,19} The use of HR to prescribe exercise intensity can lead to errors in estimating workload relative to VO\textsubscript{2max}. The results, however, do not appear to be overly influenced by such error, because RPE, another variable closely linked with exercise intensity, was nearly identical between exercise modes. Finally, the generalizability of the results is somewhat limited by the sampling procedure. It is possible that subjects who volunteered may have differed in some way from those who met the criteria but did not participate in the study.

In conclusion, this study provided a direct comparison of BP responses during low and moderate cycling and treadmill exercise in sedentary, normotensive African American women, an understudied population in exercise physiology. The results indicate: 1) after controlling for baseline BP, BMI, and age, exercise BP reactivity is mode dependent with reactivity higher on the cycle compared to the treadmill at low and moderate intensities. Mode and intensity do not appear to produce an interaction effect for either SBP or DBP reactivity; and 2) an inverse relationship exists between moderate-intensity SBP reactivity and baseline SBP after controlling for BMI and age on the cycle and treadmill. Additional study in this area is important because further understanding of exercise BP responses can help suggest safer health-related exercise intervention strategies for individuals susceptible to the development of hypertension. Future studies should compare normotensive and hypertensive African American females across modes at various intensities to clarify potential BP response differences in these groups. Finally, exercise mode and the relationship between baseline BP and BP reactivity may be important considerations with respect to inconsistencies in the exercise BP literature.

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**REFERENCES**


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