

# Aerobic exercise intensity and time of stressor administration influence cardiovascular responses to psychological stress

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## Abstract

This study examined cardiovascular responses as a function of time following exercise in which participants were exposed to a laboratory stressor. Ninety (42 women) young (18–35 years old) nonsmoking normotensive participants engaged in 30 min of high and low intensity (75–80% and 50–55%  $\text{VO}_2$  max) aerobic exercise and a sedentary control condition. Participants were randomly assigned to a laboratory stressor 5, 30, or 60 min following the exercise bout. Results indicate that low and high intensity exercise significantly reduce heart rate (HR) and systolic and diastolic blood pressure reactivity and HR recovery values. An inverse relationship between intensity of exercise and subsequent cardiovascular reactivity was found. These findings suggest attenuated stress responses following acute exercise depend both on exercise intensity and the time of exposure to psychological stress following exercise.

**Descriptors:** Exercise, Psychological stress, Cardiovascular reactivity, Blood pressure

Heightened cardiovascular reactivity to and inadequate recovery from psychological stress have been implicated in the etiology of hypertension and coronary heart disease (CHD; Krantz & Manuck, 1984; Lovallo & Gerin, 2003; Matthews, Wood, & Allen, 1993). Therefore, identifying effective interventions to reduce cardiovascular responses to psychological stress has become a vital public health concern. One strategy that has received increasing scientific and popular press attention is the role of exercise in attenuating cardiovascular responses to stress (Fillingim & Blumenthal, 1992; Sothmann et al., 1996). Regular aerobic exercise participation may serve a protective role in the development of hypertension and CHD by reducing the negative effects of chronic psychological stress or by attenuating acute stress reactivity that may be damaging to the cardiovascular system if elevated to excessive levels (Hamer, Taylor, & Steptoe, 2006; Spalding, Lyon, Steel, & Hatfield, 2004). However, a number of methodological shortcomings associated with studies examining the relationship between exercise and subsequent exposure to psychological stress have resulted in a limited under-

standing of how exercise may serve a protective role against exaggerated or excessive cardiovascular stress responses as well as the protective role of exercise in attenuating the risk for hypertension and CHD.

Currently, two shortcomings in this area of research include the inadequate assessment and manipulation of aerobic exercise and a lack of examination of the magnitude and length of attenuated stress responses following exercise (i.e., during the postexercise window). For instance, nearly every study to date incorporating an acute bout of aerobic exercise has relied on a postexercise assessment period of approximately 30 min in which to administer a laboratory stressor (e.g., mental arithmetic or Stroop task). The rationale behind selection of this time period is to allow participants the opportunity to reach baseline cardiovascular levels before exposing them to a psychological stressor. However, causal and mechanistic inferences are difficult to evaluate from such studies because of the limited assessment of cardiovascular stress levels at different time points following exercise. If an acute bout of aerobic exercise indeed serves to attenuate subsequent cardiovascular responses to stress, more than one postexercise assessment period is warranted to determine the length of time that reduced stress responses persist. From a mechanistic standpoint, it is important to study the effects of exercise on cardiovascular responses to stress from immediately postexercise to the conventional 30-min assessment period. Further, it is possible that exercise serves to attenuate cardiovascular responses to psychological stress at the well-documented 30 min assessment period (Crews & Landers, 1987;

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Hamer et al., 2006), but these effects may be relatively short-lived and thus of limited clinical relevance.

In light of these methodological considerations, the purpose of this study was to determine if cardiovascular responses during psychological stress and recovery are reduced following a low or high intensity bout of aerobic exercise in young healthy normotensive men and women. A secondary aim was to further establish the time course of stress responses by including assessments of cardiovascular responses to psychological stress at 5, 30, and 60 min postexercise. Addressing these shortcomings would provide stronger evidence for a protective effect of aerobic exercise on excessive or exaggerated stress responses, as well as elucidating a possible connection between exercise and the development of hypertension and CHD. It was hypothesized that the relationship between exercise intensity and cardiovascular stress reactivity and recovery values would differ based on the timing of the postexercise laboratory stressor. Specifically, participating in either a low or high intensity bout of aerobic exercise was expected to reduce cardiovascular reactivity and recovery values compared to a sedentary control condition at all three assessment periods. Based on studies suggesting a linear dose-response effect (Rejeski, Gregg, Thompson, & Berry, 1991; Roy & Steptoe, 1991), high intensity exercise was also expected to reduce cardiovascular reactivity and recovery to a standard laboratory stressor at 30 and 60 min following exercise cessation compared to low intensity exercise or a sedentary control condition. Similar to previous findings (Russell, Epstein, & Erickson, 1983), low intensity exercise was hypothesized to produce the greatest attenuations in cardiovascular responses to stress at the 5 min assessment period.

## Method

### Participants

Physically active male and female participants ( $N = 104$ ) between the ages of 18 and 35 years were recruited from a university and the surrounding area in the southwestern United States. All participants who agreed to be in the study signed an informed consent form that was approved by a university institutional review board. To be included in the study, all participants met the following inclusion criteria: (1) no significant health problems (e.g., bone or joint problem) that would influence their ability to exercise, (2) no history of psychological disorders (e.g., clinically diagnosed anxiety or depression), (3) no use of pharmacological drugs, medications, or tobacco products that would influence their cardiovascular responses to stress, and (4) normotensive blood pressure (BP) values (i.e., blood pressure  $\leq 140/90$  mmHg) at the initial testing session as determined by the average of three BP readings made at 2-min intervals in a seated position. Participants had normal BP values in accordance with the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (2003) criteria (Chobanian et al., 2003). Participants received either extra credit in a kinesiology course or monetary compensation (\$20) for their participation.

In addition to the meeting the initial screening criteria for health, all participants must have been physically active on a regular basis. To be considered physically active, the American College of Sports Medicine (2000) recommends 15 to 60 min of accumulated physical activity performed on most, if not all, days

of the week. Therefore, to be considered eligible for participation in this study, individuals must have reported having engaged in at least 30 min of low-to-moderate intensity (e.g., walking or jogging) physical activity at least three times per week for the past 3 months. This physical activity criterion was used for several reasons. First, for an adequate test of the effects of acute exercise on subsequent cardiovascular responses to psychological stress, it was important to limit the possibility that participants would perceive the acute exercise intervention as aversive. It is likely that sedentary individuals asked to participate in a vigorous bout of exercise would perceive such an intervention as stressful and would likely have aversive affective and/or psychophysiological responses as a result. Second, the use of physically active participants was used to maximize responsiveness of the autonomic nervous system during and following exercise (Kubitz & Landers, 1993). Using regularly physically active participants enhanced the likelihood that they would evidence adaptive cardiovascular responses to exercise (Heyward, 2002).

### Procedures

During the initial testing session, participants were given a general description of the study, provided written informed consent, completed a standard health and exercise history questionnaire and physical activity readiness questionnaire, and underwent blood pressure screening. Participants who met the inclusion criteria were then fitted with a Polar S810i HR monitor and asked to rest quietly for 10 min in order to determine resting heart rate (HR). Following this, participants were escorted to an adjacent room for a maximal oxygen consumption test on a treadmill.

Aerobic fitness was determined by a maximal oxygen consumption test using a modified Balke treadmill protocol (Balke & Ware, 1959). Specifically, participants began walking on the treadmill for 2 min at a level grade and a speed of 3 mph. After the initial 3–4 min warm-up, participants increased the speed of the treadmill until they reached their preferred running speed. Participants then continued running for 2 min, during which time the grade of the treadmill was increased by 2%. Thereafter, the grade of the treadmill was increased by 1% at the beginning of each additional minute of exercise until the criteria for establishing  $\text{VO}_2$  max were met (Taylor, Buskirk, & Henschel, 1955) or until the participant reached volitional exhaustion.  $\text{VO}_2$  max ( $\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ) was determined from direct breath-by-breath gas exchange data using a Parvo Medics True Max 2400 Metabolic Measurement Cart (ParvoMedics, Inc., Sandy, UT) and was established as the maximal average oxygen consumption over 30-s intervals.

Following the initial testing session, all participants were randomly assigned to one of three testing groups, which served as the between-subjects factor of the experiment. These groups consisted of the period of time postexercise that the laboratory stressor would be administered to participants. Specifically, participants were randomly assigned to exposure to a mental arithmetic task at 5, 30, or 60 min postexercise. Once they were assigned to a group, participants encountered the laboratory stressor at that same time point for all subsequent testing sessions. For the within-subjects portion of the study, participants completed a 30 min no-exercise sedentary control condition, a low intensity exercise condition (50%–55%  $\text{VO}_2$  max), and a high intensity exercise (75%–80%  $\text{VO}_2$  max) condition in a counterbalanced order, performed 2–3 days apart to allow adequate recovery from the previous exercise bout. In addition,

each session was conducted at approximately the same time of day to reduce potential diurnal variability in cardiovascular responses to stress. Participants were asked not to engage in any structured exercise or workouts on the day of their testing sessions and were asked not to use tobacco products, consume caffeine, or ingest any substances containing ephedrine for 3–4 h prior to every session. Verbal reports at the start of each testing session were used to determine compliance to these restrictions.

As a manipulation check of exercise intensity, Borg's (1982) rating of perceived exertion (RPE) was assessed every 5 min during the exercise conditions. The scale ranges from 6 (*very, very light*) to 19 (*very, very hard*). Participants were continually instructed to work at a level between 10 (*fairly light*) to 12 (*light*) during the low intensity condition and between 14 (*somewhat hard*) to 16 (*hard*) for the high intensity condition. The control condition consisted of reading magazines that were prescreened for potential excitatory material. Immediately following the 30-min treatment condition, participants were escorted to an adjacent room for the psychological stress testing.

### Laboratory Stressor

The laboratory stressor for the current study consisted of participants being asked to perform six 1-min serial subtraction problems verbally without stopping and without the aid of pencil and paper. They were presented with standardized task instructions asking them to continually subtract a random two-digit number from a random four-digit number during each minute. Participants were informed that the task was going to be videotaped to compare their responses to other participants in the study. The video camera served to enhance the "stressfulness" of the laboratory situation. They were instructed to work as rapidly and accurately as possible and were given prompts to speed up their responses at the start of minutes 2, 4, and 6. A modified version of this protocol has previously been shown to elicit measurable cardiovascular responses to stress (Sgoutas-Emch et al., 1994). To reduce habituation effects of retesting, the sequence of arithmetic problems was rearranged for each subsequent testing session.

### Measures

**Heart rate.** HR was monitored for 10 min at the beginning of each testing session to establish an initial pretreatment resting value, throughout the exercise and control conditions, and prior to, during, and following the psychological stress testing. HR was assessed using the Polar S810i HR monitor (Polar Electro Co., Lake Success, NY) at consecutive R-R intervals and downloaded using a Polar HR monitor computer interface for computer analyses with Polar HR monitor software. This HR device consists of a chest strap that contains electrodes and a transmitter, along with a receiver that stores the HR data. Average HR during the testing phases (before, during, and after exercise) and change in HR from prestress baseline values during the stress testing was assessed.

**Systolic and diastolic blood pressure (SBP and DBP).** SBP and DBP were assessed with an Omron Model HEM-780 portable automatic inflation BP monitor. BP readings were taken every 2 min during the initial 10-min rest period to establish a preexercise resting value, at the end of minutes 1, 3, and 5 of the 5 min immediately preceding the stressor (prestress baseline), and during minutes 2, 4, and 6 of the mental stress testing. In addition, BP measures were assessed every 2 min during a 10-min

recovery period following exposure to the laboratory stressor to assess participants' SBP and DBP recovery from the psychological stressor.

**Stressor difficulty.** Subjective ratings of the difficulty of the laboratory stressor were assessed following each condition. Participants were asked to rate the task on a 5-point Likert-type scale ranging from 1 (*not difficult at all*) to 5 (*very difficult*). A similar rating scale has been used in previous research of exercise and stress reactivity (Buckworth, Dishman, & Cureton, 1994).

### Data Analysis

Mean SBP, DBP, and HR were calculated by averaging measures taken during the 10-min initial resting period, 5-min prestress period, the 6-min stress period, and the 10-min recovery period. To obtain a measure of cardiovascular reactivity, task-induced delta ( $\Delta$ ) change scores were calculated as the mean mental arithmetic value minus the mean prestress baseline value. As a manipulation check of the effectiveness of the mental arithmetic in eliciting cardiovascular stress responses, a MANOVA was conducted to compare groups on  $\Delta$ SBP<sub>reactivity</sub>,  $\Delta$ DBP<sub>reactivity</sub>, and  $\Delta$ HR<sub>reactivity</sub> with group being the time of stressor administration following exercise. This analysis was expected to reveal significant increases in SBP, DBP, and HR during the mental arithmetic task compared to the prestress baseline period. To determine whether different patterns of cardiovascular reactivity emerged depending on the order in which participants completed the conditions, a 3 (Group)  $\times$  6 (Order) MANOVA was conducted. In addition, a repeated measures MANOVA was computed for  $\Delta$ SBP<sub>reactivity</sub>,  $\Delta$ DBP<sub>reactivity</sub>, and  $\Delta$ HR<sub>reactivity</sub> to examine whether the laboratory stressor was equally stressful across all treatment conditions (control, low intensity exercise, high intensity exercise) when collapsed across time of stressor administration (group). A one-way ANOVA was also conducted to compare participants' subjective rating of stressor difficulty.

To examine the effects of exercise on cardiovascular reactivity,  $\Delta$ SBP<sub>reactivity</sub>,  $\Delta$ DBP<sub>reactivity</sub>, and  $\Delta$ HR<sub>reactivity</sub> scores were subjected to a 3 (Group)  $\times$  3 (Condition) MANOVA with repeated measures on the condition factor. To compare the effectiveness of exercise in attenuating cardiovascular responses to stress after accounting for differences in baseline values due to exercise (i.e., differences in the prestress baselines), each cardiovascular response during the stressor was submitted to a 3 (Group)  $\times$  3 (Condition) MANCOVA with repeated measures on the condition factor. Cardiovascular responses at the pretreatment baseline (i.e., cardiovascular changes resulting from exercise) were used as the covariate.

To obtain a measure of cardiovascular recovery from stress, recovery  $\Delta$ change scores were calculated as the mean 10-min recovery value minus the mean task-induced mental arithmetic value. To examine the effects of exercise on recovery from the laboratory stressor and to determine whether the effects differ based on the timing of the laboratory stressor,  $\Delta$ SBP<sub>recovery</sub>,  $\Delta$ DBP<sub>recovery</sub>, and  $\Delta$ HR<sub>recovery</sub> scores were subjected to a 3 (Group)  $\times$  3 (Condition) MANOVA with repeated measures on the condition factor. To compare recovery values after accounting for elevations due to exercise,  $\Delta$ SBP<sub>recovery</sub>,  $\Delta$ DBP<sub>recovery</sub>, and  $\Delta$ HR<sub>recovery</sub> scores were submitted to a 3 (Group)  $\times$  3 (Condition) MANCOVA with repeated measures on the condition factor. Prestress baseline values (i.e., cardiovascular elevations due to exercise) were used as the covariates. Significant multivariate

tests were followed by univariate ANOVAs to determine which of the dependent measures were contributing to the effect. Significant univariate  $F$  tests were followed by pairwise comparisons using Tukey's Honestly Significant Difference. A critical alpha level of  $p < .05$  was adopted for all significance tests. In addition, effect sizes ( $ES$ ) were calculated for pairwise comparisons by using Hedges'  $g$  statistic (Hedges, 1981). The calculation of Hedges'  $g$  involves subtracting the means of two groups and dividing the mean difference by the pooled standard deviation across the exercise and control groups.

## Results

### Descriptive Statistics

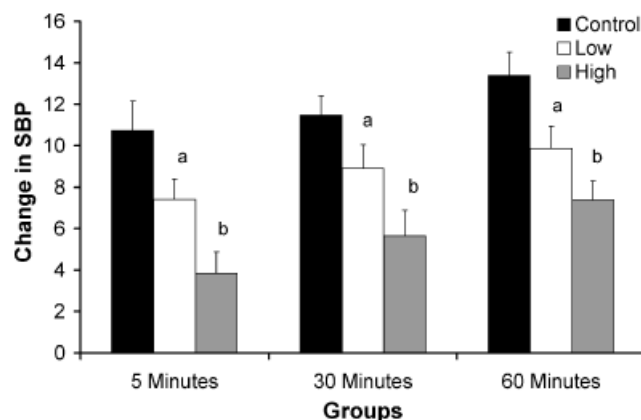
Of the 104 participants who were recruited to participate in this study, 90 ( $n = 42$  women) of these participants completed all phases of the experimental protocol. The 14 participants who failed to complete the entire protocol did not differ from those who completed the study in terms of gender, age, weight, BMI, or  $VO_2$  max,  $ps > .05$ . Descriptive statistics of age, weight, height, BMI,  $VO_2$  max, and resting and prestress cardiovascular values for participants in each group are presented in Table 1. Participants' age ranged from 18 to 34 years ( $M = 22.91$ ,  $SD = 4.18$ ) and by self-reported race, the sample consisted of 72% Caucasian, 20% Hispanic, 5% African American, and 3% Native American. No significant differences in age, gender, weight, BMI, or  $VO_2$  max were found between groups,  $p > .05$ . Because both men and women were included in this study, gender effects were assessed for all variables (Dishman, Jackson, & Nakamura, 2002). Analyses of reactivity and recovery to psychological stress including gender indicated a similar effect on all outcome variables for both men and women,  $p > .05$ . Therefore, data were collapsed across gender for all analyses.

### Manipulation Check of Laboratory Stressor

MANOVA revealed that the mental arithmetic task elicited significant increases in SBP,  $F(1,89) = 200.48$ ,  $p < .001$ , DBP,  $F(1,89) = 201.68$ ,  $p < .001$ , and HR,  $F(1,89) = 19.00$ ,  $p < .001$ . The 3 (Group)  $\times$  6 (Order) MANOVA revealed no significant effects for order of stressor administration,  $p > .05$ , which suggests that testing order did not influence cardiovascular reactivity to stress (i.e., no habituation to the laboratory stressor).

### Effect of Exercise on Reactivity to Stress

MANOVA results indicated a significant main effect for Group,  $F(2,156) = 3.09$ ,  $p < .001$ , and a significant Group  $\times$  Condition interaction,  $F(2,136) = 2.39$ ,  $p < .001$ , for cardiovascular reac-



**Figure 1.** Mean change (+SEM) in systolic blood pressure reactivity (mmHg) by group at 5, 30, and 60 min postexercise. Values not sharing a common superscript differ by  $p < .05$ .

tivity to the mental arithmetic task (see Figures 1–3). Post hoc analyses at 5 min indicated that, compared to control and low intensity exercise, high intensity exercise significantly attenuated SBP,  $t(68) \geq 3.36$ ,  $p \leq .001$ , and HR,  $t(68) \geq 2.08$ ,  $p < .05$ , reactivity. Furthermore, DBP reactivity was reduced following high intensity exercise compared to the control condition,  $t(68) = 5.70$ ,  $p < .001$ . SBP, DBP, and HR reactivity were all significantly attenuated in the low intensity condition compared with the sedentary control condition at 5 min,  $t(68) \geq 3.03$ ,  $p < .001$  (see Table 2).

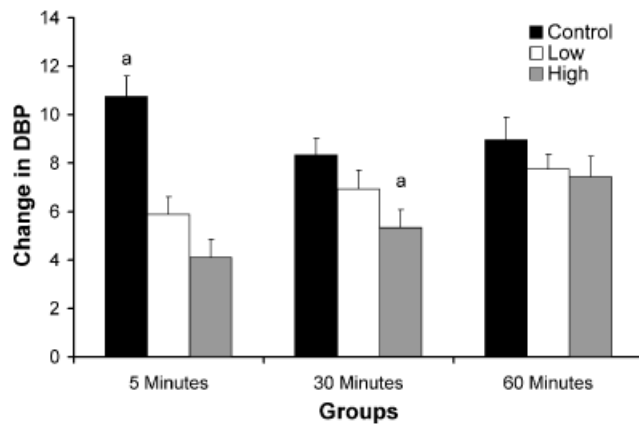
Further inspection of the Group  $\times$  Condition interaction showed that at 30 min postexercise, SBP reactivity was significantly lower in the low,  $t(68) = 2.30$ ,  $p < .05$ ,  $ES = 0.44$ , and high,  $t(68) = 5.51$ ,  $p < .001$ ,  $ES = 1.02$ , intensity exercise conditions compared to the sedentary control. High intensity exercise also resulted in reduced DBP,  $t(68) = 3.28$ ,  $p \leq .001$ ,  $ES = 0.81$ , and HR,  $t(68) = 2.43$ ,  $p < .05$ ,  $ES = 0.60$ , reactivity compared with the sedentary control condition. Although no differences emerged in DBP and HR reactivity between the low and high intensity exercise conditions at the 30-min assessment period, high intensity exercise resulted in attenuated SBP reactivity compared to low intensity exercise,  $t(68) = 3.09$ ,  $p < .01$ ,  $ES = 0.53$ .

At the 60-min assessment period, SBP reactivity was significantly lower in the high intensity condition compared to the other conditions,  $F(2,64) = 6.72$ ,  $p < .05$ . The low intensity condition also resulted in significantly lower SBP reactivity compared to

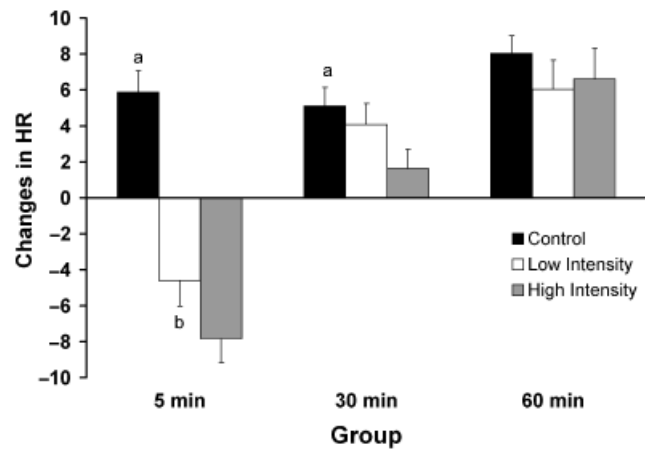
**Table 1.** Participant Demographic Means ( $SD$ ) Overall and by Grouping Variable (Time Following Exercise)

Variable	Total ( $N = 90$ )	5 min ( $n = 30$ )	30 min ( $n = 30$ )	60 min ( $n = 30$ )
Age (years)	22.91 (4.18)	22.70 (3.21)	23.60 (4.26)	21.87 (2.36)
Weight (kg)	74.97 (17.09)	73.66 (19.02)	75.93 (18.17)	75.33 (14.20)
Height (cm)	172.65 (10.34)	173.40 (8.77)	172.17 (11.98)	172.38 (10.34)
BMI ( $\text{kg}/\text{m}^2$ )	25.03 (4.62)	24.30 (4.93)	25.40 (4.31)	25.37 (4.67)
$VO_2$ max ( $\text{ml}/\text{kg}/\text{min}$ )	42.76 (8.42)	43.89 (9.06)	41.36 (8.30)	43.04 (7.96)
Resting SBP	115.69 (9.67)	114.17 (8.28)	115.78 (11.15)	117.12 (9.45)
Resting DBP	68.89 (6.23)	68.47 (6.23)	69.24 (7.16)	68.97 (5.31)
Resting HR	74.23 (9.57)	74.67 (10.74)	73.22 (9.03)	74.80 (9.09)
Prestress SBP	115.64 (9.33)	118.07 (8.54)	114.38 (9.85)	114.47 (9.37)
Prestress DBP	68.28 (6.06)	69.43 (6.26)	68.13 (6.34)	67.28 (5.56)
Prestress HR	82.98 (11.86)	91.03 (13.45) <sup>a</sup>	79.88 (7.52)	78.02 (9.58)

Note. BMI: body mass index. In each row of means, values not sharing a common superscript differ by  $p < .05$ .



**Figure 2.** Mean change (+SEM) in diastolic blood pressure reactivity (mmHg) by group at 5, 30, and 60 min postexercise. Values not sharing a common superscript differ by  $p < .05$ .



**Figure 3.** Mean change (+SEM) in heart rate reactivity (bpm) by group at 5, 30, and 60 min postexercise. Values not sharing a common superscript differ by  $p < .05$ .

the control condition,  $t(68) = 3.21, p < .01, ES = 0.56$ . No differences in DBP and HR reactivity between the three conditions at the 60-min stressor delivery period were observed,  $p > .05$ .

Results of the 3 (Group)  $\times$  3 (Treatment Condition) MANCOVA revealed that, after controlling for elevations in cardiovascular measures due to exercise, high intensity exercise resulted in significantly lower SBP reactivity to stress than did the control condition at 5 and 30 min following the treatment condition,  $p < .05$ . High intensity exercise also resulted in significantly lower DBP reactivity during the 5-min stressor delivery period compared to the sedentary control condition,  $p < .01$ . Both low and high intensity exercise resulted in attenuated HR reactivity during the mental arithmetic after adjusting for HR changes due to exercise at 5 min postexercise,  $p < .001$ .

#### Effects of Exercise on Recovery from Stress

The 3 (Group)  $\times$  3 (Condition) MANOVA with repeated measures on condition revealed a significant two-way interaction,  $F(12,164) = 2.76, p < .01$ . Univariate followups indicated that, at all time periods following exercise, both low and high intensity exercise resulted in significantly lower  $\Delta HR_{\text{recovery}}$  compared to the sedentary control condition,  $F_s(2,68) \geq 2.11, p \leq .01$  (see Table 3). High intensity exercise also resulted in significantly

lower  $\Delta HR_{\text{recovery}}$  following exposure to the stressor at 5, 30, and 60 min compared to low intensity exercise,  $p < .01$ . During recovery, no significant differences in  $\Delta SBP_{\text{recovery}}$  were found between the three treatment conditions,  $p > .05$ .

A significant condition main effect was found for  $\Delta DBP_{\text{recovery}}$  during the recovery period,  $F(2,86) = 3.13, p < .05$ . Specifically, when collapsed across groups, high intensity exercise resulted in lower  $\Delta DBP_{\text{recovery}}$  values compared to the sedentary control condition. The differences between high and low intensity exercise approached significance,  $p = .053$ , with lower  $\Delta DBP_{\text{recovery}}$  values for the high intensity condition.

Results of the 3 (Group)  $\times$  3 (Condition) MANCOVAs revealed that, after statistically controlling for exercise-induced changes in the cardiovascular measures prior to stressor administration,  $\Delta HR_{\text{recovery}}$  was significantly lower for both exercise conditions compared to the control condition at 5, 30, and 60 min postexercise,  $p < .01$ . Furthermore, there was a dose-response effect for exercise intensity, with high intensity exercise producing the lowest  $\Delta HR_{\text{recovery}}$  following stressor administration at 5, 30, and 60 min postexercise,  $p < .001$ . There were no significant effects for either  $\Delta SBP_{\text{recovery}}$  or  $\Delta DBP_{\text{recovery}}$  after controlling for prestressor values, though the overall Group  $\times$  Condition interaction approached significance for DBP,  $p < .08$ .

**Table 2.** Cardiovascular Responses ( $M \pm SD$ ) at Baseline and during Mental Arithmetic at Each Time Period Following Exercise

Measure	5 min ( $n = 30$ )		30 min ( $n = 30$ )		60 min ( $n = 30$ )	
	Prestress baseline	Mental arithmetic	Prestress baseline	Mental arithmetic	Prestress baseline	Mental arithmetic
<b>Control condition</b>						
SBP	113.37 $\pm$ 7.69	124.10 $\pm$ 12.2	115.63 $\pm$ 12.22	127.09 $\pm$ 13.96	114.77 $\pm$ 9.13	128.16 $\pm$ 10.69
DBP	66.50 $\pm$ 7.55	77.24 $\pm$ 8.11	68.03 $\pm$ 8.04	76.38 $\pm$ 8.32	67.70 $\pm$ 6.07	76.66 $\pm$ 7.35
HR	71.30 $\pm$ 10.39	77.17 $\pm$ 12.26	70.07 $\pm$ 9.81	75.17 $\pm$ 11.35	69.30 $\pm$ 9.61	77.33 $\pm$ 11.17
<b>Low intensity condition</b>						
SBP	118.10 $\pm$ 9.58	125.50 $\pm$ 11.21	109.6 $\pm$ 21.17	121.86 $\pm$ 11.59	114.03 $\pm$ 9.89	123.89 $\pm$ 10.87
DBP	70.43 $\pm$ 6.57	76.30 $\pm$ 7.04	68.03 $\pm$ 6.29	74.97 $\pm$ 6.27	67.53 $\pm$ 5.77	75.29 $\pm$ 6.50
HR	90.77 $\pm$ 19.5	86.17 $\pm$ 14.44	77.07 $\pm$ 8.55	81.13 $\pm$ 8.60	78.93 $\pm$ 12.3	84.97 $\pm$ 15.03
<b>High intensity condition</b>						
SBP	122.73 $\pm$ 10.1	126.57 $\pm$ 11.04	114.57 $\pm$ 9.64	120.21 $\pm$ 10.76	114.63 $\pm$ 10.06	122.01 $\pm$ 9.94
DBP	71.37 $\pm$ 7.64	75.47 $\pm$ 8.05	68.33 $\pm$ 6.39	73.67 $\pm$ 6.81	66.60 $\pm$ 6.46	74.03 $\pm$ 6.58
HR	111.03 $\pm$ 16.8	103.20 $\pm$ 14.08	92.50 $\pm$ 9.97	94.13 $\pm$ 10.55	85.83 $\pm$ 11.2	92.40 $\pm$ 14.23

**Table 3.** Psychophysiological Responses ( $\Delta$ Change Scores) during Recovery from Mental Arithmetic

Measure	Control	Low	High
$\Delta$ SBP <sub>recovery</sub>			
5 min	7.04 $\pm$ 6.34	8.41 $\pm$ 5.00	8.12 $\pm$ 5.42
30 min	7.97 $\pm$ 5.43	6.30 $\pm$ 5.15	5.40 $\pm$ 6.91
60 min	5.77 $\pm$ 4.58	5.30 $\pm$ 4.73	6.34 $\pm$ 3.90
$\Delta$ DBP <sub>recovery</sub>			
5 min	6.07 $\pm$ 4.64	5.54 $\pm$ 2.89	4.38 $\pm$ 4.01 <sup>a</sup>
30 min	4.78 $\pm$ 4.60	4.51 $\pm$ 3.99	2.04 $\pm$ 3.41 <sup>a</sup>
60 min	5.14 $\pm$ 4.71	4.33 $\pm$ 2.43	4.99 $\pm$ 3.95 <sup>a</sup>
$\Delta$ HR <sub>recovery</sub>			
5 min	-4.64 $\pm$ 5.85	-6.11 $\pm$ 5.24 <sup>a</sup>	-9.45 $\pm$ 4.64 <sup>b</sup>
30 min	-3.75 $\pm$ 4.70	-4.94 $\pm$ 5.24 <sup>a</sup>	-4.26 $\pm$ 4.54 <sup>b</sup>
60 min	-5.83 $\pm$ 4.37	-5.81 $\pm$ 5.26	-7.57 $\pm$ 5.64 <sup>b</sup>

Note. These delta ( $\Delta$ ) change values represent the mean 10-min recovery value minus the mean mental arithmetic level. In each row of means, values not sharing a common superscript differ by  $p < .05$ .

## Discussion

The primary purpose of this study was to determine if cardiovascular responses during psychological stress and recovery from that stress are reduced following a low or high intensity bout of aerobic exercise in young healthy normotensive men and women as a replication of previous work. A secondary aim was to extend previous research by further establishing the time course of stress responses by including assessments of cardiovascular responses to psychological stress at 5, 30, and 60 min postexercise. The results of this study provide further support for the position that an acute bout of aerobic exercise results in attenuated cardiovascular reactivity and improved HR recovery to psychological stress. Specifically, participants experienced reduced SBP, DBP, and HR reactivity and better HR recovery from a psychological stressor administered following low and high intensity exercise compared to a sedentary control condition. For high intensity exercise, beneficial effects were observed for SBP, DBP, and HR reactivity to stress at 5 and 30 min postexercise and SBP reactivity continued to be reduced at 60 min postexercise. Low intensity exercise resulted in attenuated SBP, DBP, and HR reactivity at 5 min following exercise, and continued to reduce SBP reactivity at 30 and 60 min. Even after statistically accounting for cardiovascular elevations due to the exercise bout, reductions in HR reactivity to stress were still observed for low and high intensity exercise 5 min following exercise cessation. Further, high intensity exercise resulted in lower SBP reactivity at 5 and 30 min and DBP reactivity at 5 min following exercise cessation even after accounting for the exercise-induced cardiovascular changes. In combination, this pattern of findings demonstrates that acute aerobic exercise results in lower cardiovascular responses during exposure to a psychological stressor. Furthermore, exercise also appears to enhance HR recovery following stressor cessation in a dose-response fashion, with high intensity exercise producing the greatest recovery. Though these effects were not seen for BP recovery, this may be partly due to the attenuated BP reactivity to the stressor following the exercise conditions in the first place. In other words, if there is a reduced cardiovascular response to a stressor following exercise, it would seem logical that the recovery values would not be as pronounced because of the diminished perturbation of homeostatic control.

The current findings further demonstrate that an acute bout of aerobic exercise results in a different pattern of cardiovascular

stress responses depending on the intensity of the exercise as well as the time after exercise cessation that individuals are exposed to psychological stress. The pattern of results in the current study supports a dose-response relationship between the intensity of exercise and subsequent cardiovascular reactivity to stress. An inverse relationship between the dose of exercise and subsequent SBP and DBP responses to stress has previously been reported (Rejeski et al., 1991; Roy & Steptoe, 1991). Rejeski et al. employed a design similar to the within-subjects portion of this experiment (i.e., the Condition factor), and found that exercise of higher intensity and longer duration resulted in lower BP responses compared to an attention placebo group. Exercise of low intensity and shorter duration resulted in intermediate effects. Future research should incorporate several different intensities and durations of exercise to adequately assess the nature of this trend.

It was initially hypothesized that low intensity exercise would provide greater reductions to psychological stress 5 min following exercise cessation compared with high intensity exercise. Because HR increases during low intensity exercise (i.e., less than 60%  $\text{VO}_2$  max) are primarily elicited by parasympathetic withdrawal whereas HR increases from exercise of greater intensities result from increased sympathetic influences on the heart, the heightened SNS activity from exercise was thought to contribute further to increases in stress responses. However, the findings revealed that high intensity exercise resulted in greater BP attenuations even though participants were still physiologically aroused from the exercise. Previous investigators (Russell et al., 1983) have attributed their lack of cardiovascular attenuations to stress at 5 min after high intensity exercise to the confounding influences of participants' recovering from exercise while they were exposed to the laboratory stressor. Although exercise attenuated cardiovascular reactivity immediately following exercise, absolute HR and BP levels were higher in the exercise conditions. One of the factors that may have contributed to the higher HR and BP levels immediately following exercise, especially in the high intensity condition, includes delayed autonomic recovery from the exercise session. It is obvious that immediately following exercise one's cardiovascular responses (e.g., BP and HR) will be heightened. Furthermore, this effect may be particularly pronounced as well as considerably extended following exercise of higher intensities due to excess postexercise oxygen consumption (EPOC). With low to moderate intensity exercise, the fast component of recovery oxygen consumption represents the primary component of recovery. Under this condition, complete recovery of oxygen debt often occurs within several minutes of exercise cessation (McArdle, Katch, & Katch, 2007). On the other hand, previous research has found that the slow component of recovery from intense exercise can take up to 1 full day to return to preexercise levels (e.g., Gore & Withers, 1990). Recent theories on this phenomenon have focused on the thermogenic, metabolic, cardiovascular, and endocrine adjustments that have to occur following the homeostatic perturbation that occurs following high intensity or long-duration exercise. These responses are even seen for at least 1 h following shorter "supramaximal" intervals of exercise (Bahr, 1992).

To offset this confounding influence of exercise toward cardiovascular stress responses, the current study used covariance to control for prestress baseline levels (i.e., the heightened cardiovascular responses resulting from exercise). The pattern of findings showed that high intensity exercise resulted in the greatest attenuations in cardiovascular reactivity at 5 and 30 min, whereas low intensity exercise reduced HR reactivity at 5 min postexercise, but

did not significantly reduce SBP or DBP reactivity. In contrast to the Russell et al. (1983) study, the results from the present study, with more study participants and thus greater statistical power, suggest that reductions in cardiovascular stress responses are evident even at 5 min following intense exercise.

At 30 min postexercise, both low and high intensity exercise resulted in reduced HR and BP levels during the laboratory stressor as well as improved HR recovery during the period following stressor exposure. This finding is consistent with previous studies that have reported decreases in BP levels following acute exercise (e.g., O'Connor & Davis, 1992; Raglin & Morgan, 1987). It is also consistent with previous findings in the literature (e.g., Crews & Landers, 1987; Hobson & Rejeski, 1993; Rejeski, Thompson, Brubaker, & Miller, 1992), that low and high intensity exercise both result in attenuated cardiovascular reactivity when the stressor is administered approximately 30 min after exercise.

One of the issues left unanswered thus far in the literature is the potential clinical significance of exercise-related reductions in psychological stress. Hobson and Rejeski (1993) argued that if the effect of exercise is limited to a very brief postexercise time period (e.g., less than 30 min), it may have limited clinical significance. If exercise results in attenuated cardiovascular responses to stress for a longer period of time following exercise cessation, then exercise may have clinical significance. Hamer and colleagues (2006) have suggested that the main benefits of chronic exercise on stress may be due to regular exercisers encountering stress during the postexercise window. Southard and Hart (1991) conducted a case study on a hypertensive male and reported reduced ambulatory BP responses by approximately 12 mm Hg for 9 h following 30 min of high intensity exercise. Ebbesen, Prkachin, Mills, and Green (1992) also explored the clinical significance of a delayed time course of stress responses following acute exercise and reported dampened BP responses to stress 3 h after either 1 or 2 h of aerobic work. The current findings suggest that higher intensity exercise will prolong the reductions in cardiovascular stress responses, particularly for blood pressure responses during and following psychological stress. Future research should continue to examine the length of time following exercise that attenuated cardiovascular stress responses persist. Using ambulatory monitoring in combination with psychological self-report following exercise could be particularly instructive regarding the time course of stress responses following exercise.

The current findings suggest that at 30 min postexercise, low intensity exercise resulted in a 5.2 mmHg attenuation in SBP reactivity to stress and high intensity exercise attenuated SBP by 6.9 mmHg. At 60 min postexercise, low intensity exercise reduced SBP reactivity by approximately 4.3 mmHg and SBP reactivity was further attenuated following high intensity exercise (i.e., 6.2 mmHg). During the recovery periods, low and high intensity exercise reduced absolute SBP values by 4.8 mmHg and 5.5 mmHg and by 3.8 mmHg and 6.6 mmHg, during the 30-min and 60-min assessment periods, respectively. Although the clinical significance of exercise-induced reductions in cardiovascular reactivity during stress is not firmly established, these reductions in BP appear to be of a magnitude that suggests clinical importance. For instance, Vemmos et al. (2004) studied the relationship between BP and mortality risk in 1121 patients hospitalized following a stroke. SBP was measured on admission to hospital, and patients were followed after 1 month and again after 1 year to evaluate mortality rates. After taking known risk factors into account, the relative risk of 1-month mortality increased by more than 10% for every 10 mmHg increase in SBP above 130 mm Hg

and the risk of death at 1 year increased by more than 7% for every 10 mm Hg increase in SBP. As the relationship between BP and cardiovascular disease event is continuous (Chobanian et al., 2003), it seems likely that a 4–5 mmHg reduction in SBP during exposure to a stressful encounter as well as BP reductions up to 60 min following exercise would result in a meaningful reduction in the risk of cardiovascular disease, particularly when coupled with the numerous other cardiopulmonary and health benefits of exercise. Repeated acute bouts may demonstrate a cumulative effect over time by helping in the day-to-day handling of stress. Based on the results of the current study and the known dose–response relationship between exercise intensity and fitness improvements, these effects may be even more pronounced for high intensity exercise. Future research is clearly warranted to help elucidate the implications for cardiovascular health and stress-related disorders.

Substantial controversy exists over the time course of post-exercise reductions in BP and adequate recovery following acute aerobic exercise (O'Connor & Davis, 1992; Taylor, 2000). Previous research examining postexercise BP levels has typically included a time period of approximately 20–30 min following exercise cessation to measure BP levels, similar to the time period that has been used most frequently in the exercise and stress responsivity literature (Hamer et al., 2006). Although a number of central and peripheral mechanisms have been advanced to explain the reductions in BP (Tipton, 1991; Whelton, Chin, Xin, & He, 2002) and HR (Carter, Banister, & Blaber, 2003; Goldsmith, Bigger, Steinman, & Fleiss, 1992; Smith, Hudson, Graitzer, & Raven, 1989) following exercise, it is currently unclear whether attenuated stress reactivity or enhanced recovery is caused directly by the same mechanisms (Boone, Probst, Rogers, & Berger, 1993; O'Sullivan & Bell, 2001). It is possible that a combination of hemodynamic, autonomic, and/or neuroendocrine influences may serve to explain both alterations in BP levels following exercise and the effects of exercise on subsequent responses to stress. For instance, West, Brownley, and Light (1998) found a reduction in vascular resistance during exposure to a mental challenge following acute exercise. Further, Brownley et al. (2003) reported reduced norepinephrine responses and enhanced  $\beta_2$ -mediated vasodilation concomitant with reduced BP responses to stress following exercise. Future research should address possible mechanisms underlying the effects of exercise on stress to help clarify the role of exercise in hypertension, CHD risk, and the etiology of other stress-related disorders and diseases.

In summary, the results of the present study support the hypothesis that an acute bout of exercise results in attenuated cardiovascular reactivity and enhanced HR recovery from subsequent exposure to a laboratory stressor. The results also indicate that the time course of stress-buffering effects of exercise may depend on exercise dose, as greater intensities of exercise seem to result in longer periods of reduced cardiovascular reactivity to stress. Future research should focus on clearly elucidating the time course of cardiovascular attenuations to psychological stress following acute exercise of different intensities and make a concerted effort to examine mechanistic issues to establish a causal relationship between acute exercise and cardiovascular reactivity to and recovery from psychological stress. Furthermore, it is vital that investigators now focus on extending these findings to at-risk populations (e.g., clinically anxious, depressed, posttraumatic stress disorder patients, obese, elderly) who may have compromised cardiovascular systems and thus may have the most to benefit from acute exercise-related reductions in cardiovascular responses to stress.

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