

Aerobic exercise training and cardiovascular reactivity to psychological stress in sedentary young normotensive men and women

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Abstract

Forty-five (22 women) sedentary young (18–30 years old) nonsmoking normotensive volunteers engaged in either 6 weeks of aerobic training (AT), weight training (WT), or a no-treatment (NT) condition to determine whether AT lowers systolic (SBP) and diastolic (DBP) blood pressure, heart rate (HR), and rate-pressure product (RPP) during rest, psychological stress, and recovery periods. Estimated VO_2max increased for the AT (32.1 ± 1.1 to 38.4 ± 1.0 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$). A smaller increase for the WT (30.5 ± 1.1 to 33.8 ± 1.0) was likely due to increased leg strength, and VO_2max did not change for the NT (32.5 ± 1.1 to 32.9 ± 1.0). Heart rate and RPP levels were lower during psychological stress and recovery after training for AT relative to the WT and NT. Overall SBP was also lower in the AT relative to the NT but not the WT. In conclusion, aerobic training lowered cardiovascular activity levels during psychological stress and recovery in healthy young adults, implying a protective role against age-related increases in coronary heart disease for individuals who adopt aerobic exercise early in life and maintain the behavior across the life span.

Descriptors: Exercise, Cardiovascular reactivity, Stress, Blood pressure, Weight training

Participation in regular physical activity is associated with lower morbidity and mortality from coronary heart disease (CHD; Berlin & Colditz, 1990; Blair et al., 1995; Manson et al., 1999; Sesso, Paffenbarger, & Lee, 2000; Smith, Shipley, Batty, Morris, & Marmot, 2000) but the manner by which it reduces CHD risk has not been explained unequivocally. A number of possible mechanisms have been identified (Clayton, 1991; Grundy et al., 1999; Hagberg, Park, & Brown, 2000; Hardman, 1999; Jennings, 1995) including attenuation of cardiovascular reactivity to psychological stress and improved recovery (Dimsdale, Alpert, & Schneiderman, 1986). In this regard, it is well established that aerobic exercise training results in physiological adaptations that enable submaximal workloads to be negotiated with greater efficiency (e.g., at a lower heart rate and blood pressure; McArdle, Katch, & Katch, 1996), resulting in less strain on the cardiovascular system, and, importantly, it has been proposed that such improvements may generalize from ergogenic to

psychogenic challenges (Clayton, 1991; Sinyor, Schwartz, Peronnet, Brisson, & Seraganian, 1983).

This possibility is worthy of further investigation as exaggerated cardiovascular reactivity to psychological stress and inadequate recovery have been implicated in the etiology of hypertension and heart disease (Haynes, Gannon, Orimoto, O'Brien, & Brandt, 1991; Krantz & Manuck, 1984; Manuck & Krantz, 1986). As blood pressure levels and the incidence of hypertension rise throughout adulthood, particularly after 35 years of age (Kaplan, 1994), it may be that part of the age-associated increases in blood pressure are due to the effects of cardiovascular reactivity to chronic psychological stress. Therefore, regular aerobic exercise participation may delay, slow, or prevent increases in blood pressure associated with aging by contributing to a reduction in chronic cardiovascular stress reactivity. In this manner, regular participation in aerobic exercise may play a protective role in the development of hypertension and CHD. However, demonstration of such a protective role will, in part, require causal evidence that aerobic exercise participation reduces cardiovascular stress reactivity in sedentary young healthy normotensive individuals. If ameliorative effects occur in such individuals, maintenance of exercise across the life span by men and women who adopt exercise at an early age could serve a protective role as age becomes more of a risk factor. In this manner it would follow that increased benefits of exercise

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would occur as greater opportunity of prophylactic effects is present.

Empirical support for the association between aerobic fitness and cardiovascular reactivity to psychological stressors has been provided in several studies in which absolute and/or relative cardiovascular responses have been assessed (e.g., Crews and Landers, 1987; Holmes & Cappo, 1987; Light, Obrist, James, & Strogatz, 1987; McCubbin, Cheung, Montgomery, Bulbulian, & Wilson, 1992). However, causal inferences regarding the effects of aerobic exercise on reactivity are difficult to determine from these studies as they are cross-sectional in nature and any observed fitness-group differences in reactivity may be explained alternatively by constitutional differences that cannot be excluded (McCubbin et al., 1992).

In an attempt to overcome this limitation a number of investigators have conducted training studies that have also revealed heart rate (HR) levels and/or relative responses, and/or blood pressure levels, to be significantly lower during psychological stress in individuals who had completed aerobic exercise training (Cleroux, Peronnet, & de Champlain, 1985; de Geus, van Doornen, & Orlebeke, 1993; Georgiades et al., 2000; Holmes & McGilley, 1987; Kubitz & Landers, 1993; Sinyor, Peronnet, Brisson, & Seraganian, 1988; Stein & Boutcher, 1992; Throne, Bartholomew, Craig, & Farrar, 2000). However, the improvements observed in these studies also cannot be unambiguously attributed to aerobic training as the effects of retesting and/or the nonspecific effects of participating in a structured activity program (e.g., expectation, attention, time-out) were not excluded as an alternative explanation of the findings. Importantly, other studies have included appropriate activity control groups and, although not all have yielded positive findings (Roskies et al., 1986; Seraganian, Roskies, Hanley, Oseasohn, & Collu, 1987; Sinyor, Golden, Steinert, & Seraganian, 1986; Steptoe, Moses, Mathews, & Edwards, 1990), a number of investigators have reported significantly greater reductions in cardiovascular stress levels and/or relative responses during psychological stress in aerobically trained relative to weight-trained individuals (Blumenthal et al., 1988, 1990, 1991; Sherwood, Light, & Blumenthal, 1989). These latter findings substantiate a causal link between the physiological adaptations produced by aerobic exercise and attenuation of cardiovascular activity during psychological stress.

However, the evidence that aerobic training does indeed reduce cardiovascular reactivity to psychological stress, as observed for both absolute levels and relative responses, has typically been obtained from studies of middle-aged Type A men with and without borderline hypertension (Blumenthal et al., 1988, 1990; Sherwood et al., 1989) and pre- and postmenopausal women (Blumenthal et al., 1991). As such, the findings from these populations cannot be generalized to young normotensive individuals as there is evidence that they respond differently to psychological stressors. For example, greater cardiovascular reactivity has been observed in Type A relative to Type B individuals (Ward et al., 1986) and in borderline hypertensives relative to normotensives (Fredrikson & Matthews, 1990). Further, older individuals have been shown to exhibit greater relative systolic blood pressure (SBP) responses to psychological stressors than younger persons (Uchino, Uno, Holt-Lunstad, & Flinders, 1999). There is a need, then, to examine the effects of aerobic exercise training on cardiovascular reactivity in young normotensive adults. To date, younger normotensive individuals have been examined in only one study (Sinyor et al., 1986) but, in that

investigation, the sample was limited to men, and the psychophysiological stress assessment was limited to heart rate.

The purpose of this study, then, was to determine if cardiovascular activity levels during psychological stress and recovery are reduced in young healthy normotensive men and women after aerobic exercise training while employing adequate controls for competing explanations. A positive outcome would provide causal evidence for a possible protective effect of aerobic exercise participation, beginning in youth, against the development of hypertension and CHD with aging. Cardiovascular reactivity was assessed in terms of absolute levels in SBP, diastolic blood pressure (DBP), HR, and rate-pressure product (RPP). The latter measure was calculated as the product of HR and SBP and is an indirect measure of myocardial oxygen consumption and workload (McArdle et al., 1996). Aerobic fitness assessment and psychological stress testing were conducted before and after training in young nonsmoking normotensive men and women who were randomly assigned to one of three groups: aerobic training (AT), weight training (WT), and a no-training control (NT). Aerobic training was expected to produce adaptations that would lower cardiovascular activity levels during rest, psychological stress, and recovery, whereas WT and physical inactivity (i.e., NT) were not. Hence, the WT group was employed to control for the nonspecific effects of participating in a structured physical activity program (e.g., attention, expectation, and/or time out from stressful daily life). Therefore, as a result of training, the AT participants were expected to exhibit lower SBP, DBP, HR, and RPP at rest and during psychological stress and recovery relative to the WT and NT groups. No difference was expected between the WT and NT groups in any of the measures after the intervention period.

Method

Participants

The participants were 23 men and 22 women (19–29 years old, $M = 22.2$ years old, $SD = 2.8$) recruited from various employee programs at hospitals, medical centers, and universities in a large East Coast metropolitan area. Participants completed a health history questionnaire, a physical activity readiness questionnaire, and a blood pressure screening, which consisted of three determinations made at 2-min intervals in a seated position, to verify their eligibility to participate. All of the participants met the following inclusion criteria: (1) no personal or familial history of cardiovascular disease or diabetes, (2) normotensive (i.e., blood pressure $\leq 140/90$ mmHg), (3) no contraindications for exercise, (4) no participation in a cardiovascular or weight training program within the past 12 months, and (5) no use of tobacco products or medications that may affect cardiovascular function. Participants who met these initial inclusion criteria were also screened on aerobic fitness, as determined via a subsequent maximal graded exercise treadmill test. The test revealed that the participants had low aerobic fitness at the start of the study, ranking at or below the 35th percentile with respect to normative data (Pollock & Wilmore, 1990). The blood pressure screening showed that participants predominantly had optimal to normal blood pressure as classified in accordance with the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (1997) criteria; mean SBP and DBP were 119.4 mmHg ($SD = 5.3$) and 75.3 mmHg ($SD = 5.1$),

respectively. By self-reported race, the sample consisted of 36 Caucasians, 8 Blacks, and 1 Native American.

Procedures

During the initial visit to the laboratory participants were given a description of the study, provided written informed consent on a form approved by the institutional human subjects research review board, and completed the health history questionnaire, the exercise readiness questionnaire, and blood pressure screening. Individuals who satisfied the inclusion criteria were then scheduled for an exercise test to determine fitness level.

Aerobic fitness was assessed via a graded exercise test on a motor-driven treadmill (Quinton Treadmill System, model 18-60, Quinton Instrument Company, Seattle, WA) using the Bruce protocol (Bruce, Kusumi, & Hosmer, 1973). The protocol required participants to exercise during progressively more challenging 3-min stages until voluntary exhaustion was reached. Participants began the test by walking at 1.7 mph on a 10% grade for 3 min. For each stage thereafter, the treadmill grade was elevated an additional 2% and the speed was increased by 0.8 or 0.9 mph. Throughout the test the electrocardiogram (ECG) was monitored from standard limb and precordial leads. Aerobic fitness was measured as the time (expressed in seconds) that it took a participant to reach voluntary exhaustion (i.e., treadmill time). Gender-specific estimates of aerobic capacity (i.e., VO_2 max in $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) were predicted from treadmill time using a regression equation generated by Bruce et al. (1973).

Psychological stress testing was conducted between 1 and 3 days following the exercise test. After arriving at the laboratory the participant was seated in a straight-back chair in a quiet room where the testing occurred and a pressure cuff was attached around the biceps of the left arm for the measurement of blood pressure and HR. Additionally, the participant was given headphones to wear. After acclimating to the setting the participant rested with eyes open for 5 min. Tape-recorded task instructions were then delivered via the headphones, after which a 6-min mental arithmetic task was presented. The mental arithmetic problems consisted of 2- and 3-digit multiplication and division problems (e.g., 59×18 , $98/12$) that were projected, 10 at a time, onto a screen placed 15 ft in front of the participant. A new set of 10 problems was presented every 20 s whether or not all of the previous problems had been solved. Auditory distraction was presented via the headphones concurrently with the mental arithmetic problems during the last 4 min of the task in order to increase the attentional conflict and, thereby, increase the stressfulness of the task. In Min 3 and 4 the auditory stimulus was white noise that became progressively more irregular in intensity. In Min 5 and 6 the auditory stimulus consisted of recorded conversation of a mock business transaction that involved many mentions of numbers (e.g., model and serial numbers, telephone numbers, specifications, and prices). Participants were instructed to work as rapidly and accurately as possible, to give each answer verbally after it was obtained, and that their performance would be evaluated. The mental arithmetic task was immediately followed by a 90-s recovery period in which the subject was instructed to sit quietly and relax. Measures of SBP, DBP, and HR were obtained with a Dinamap Vital Signs Monitor (Critikon, Inc., model 8100, Tampa, FL) for each minute of rest and mental arithmetic. Blood pressure and HR measures were obtained every 30 s during recovery.

Treatment Groups

Participants were randomly assigned to one of three groups: aerobic training (AT, $N = 15$, 7 women), weight training (WT, $N = 15$, 9 women), or a no-treatment control (NT, $N = 15$, 6 women). Training was supervised and adherence to the training prescriptions was monitored via self-report exercise records that were validated by an exercise facility representative. Those in the AT group were instructed to exercise in an aerobic activity of their choice at an intensity of 70–85% of their maximal heart rate, as determined from the graded exercise test, for 20–30 min per session, 3–5 times weekly for 6 weeks. Participants engaged in such activities as jogging, biking, swimming, rowing, and stair climbing. Participants who were initially unable to exercise a minimum of 20 min within their target HR range were instructed to exercise for at least 10 min and to increase their exercise duration by 5 min each week. Participants were also instructed to begin each exercise session with 10 min of stretching and light-intensity exercise in order to warm up and to engage in 5 min of light activity following the aerobic exercise in order to cool down. Participants in the WT group were instructed to train 3–5 times per week for 6 weeks. Each training session included 10–15 min of warm-up activity followed by resistance exercises, which consisted of three sets of 8–12 repetitions to failure for each of the following muscles and muscle groups: biceps brachii, triceps brachii, pectorals, deltoids, hamstrings, and quadriceps. Session durations ranged from 40 to 45 min, which was similar to durations of the AT group. Strength was assessed for each of the six muscle groups before and after training as the amount of weight lifted in a one-repetition maximum (1-RM) effort. Participants were tested and trained on either Nautilus or Universal strength training equipment. Individuals in the NT group were instructed to refrain from any regular or new exercise participation, to maintain their customary level of activities of daily living, and to record any prolonged (i.e., >15 min in duration) physical activity performed during the day.

Assessments of aerobic fitness and cardiovascular reactivity to psychological stress were conducted between 1 and 3 days following completion of the 6-week training or waiting period. Testing procedures for the posttraining assessment were identical to those of the initial assessment with two minor exceptions. The sequence of arithmetic problems was rearranged for each task condition and, to reduce the effects of retesting, the volume of the white noise and conversation was increased.

Data Analysis

The effects of training on treadmill time and strength were examined first. To determine if the groups were initially similar on aerobic fitness, the treadmill times obtained before training were subjected to a one-way analysis of variance (ANOVA). To determine if training significantly improved aerobic fitness, the changes in treadmill times (i.e., posttraining – pretraining) for each group were subjected to separate one-tailed *t* tests. The test evaluated the hypothesis that the mean change score was significantly greater than zero. Additionally, the treadmill-time change scores were also subjected to a one-way ANOVA to determine if the groups differed. To determine if weight training improved strength, 1-RM change scores (i.e., posttraining – pretraining) for the six muscle groups were subjected to multivariate analysis of variance (MANOVA; Wilks = λ). A significant *F* ratio was

Table 1. Mean (SE) Treadmill Time and Estimated Aerobic Capacity (VO_{2max}) for the Aerobically Trained (AT), Weight-Trained (WT), and No-Training (NT) Groups Before (Time1) and After (Time2) Training

	AT		WT		NT	
	Time1	Time2	Time1	Time2	Time1	Time2
Treadmill time (s)	528 (11.1)	639 (13.6)	505 (20.3)	564 (16.9)	532 (14.9)	538 (10.8)
VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$)	32.1 (1.1)	38.4 (1.0)	30.5 (1.1)	33.8 (1.0)	32.5 (1.1)	32.9 (1.0)

Note: Aerobic capacity was estimated from total treadmill time (Bruce et al., 1973).

followed by a post hoc analysis using separate one-tailed *t* tests to determine the muscle groups in which strength improved.

Mean SBP, DBP, and HR were calculated for the last 2 min of the rest period (baseline), the 6-min stress period, and the 90-s recovery period. Also, RPP was calculated as the product of SBP and HR and averaged to yield mean values for the rest, stress, and recovery periods. To examine the effects of training on cardiovascular activity levels during rest, stress, and recovery, mean SBP, DBP, and HR were subjected to a $3 \times 2 \times 3$ (Group \times Session \times Period) MANOVA in which group was treated as a between-subjects factor, and session and period were treated as within-subject factors. Levels of session were the pretraining and posttraining assessments, and periods were the baseline, stress, and recovery phases within each of the two sessions. Because it was not appropriate to include RPP in the MANOVA (as it is structurally related to HR and SBP), it was analyzed in a separate mixed ANOVA employing the same three-factor design. When group differences were found at the pretraining assessment, posttraining values were subjected to an analysis of covariance (ANCOVA), with pretraining values serving as the covariate. In general, a group difference on the covariate would preclude use of ANCOVA (Miller & Chapman, 2001). However, an exception is permitted when the group difference on the covariate can be presumed to have arisen by chance, as it can be in the present study because of the random assignment of participants to groups. Significant multivariate tests were followed by univariate ANOVAs to determine which dependent measures were contributing to the effect. Significant univariate *F* tests were followed by post hoc analysis using Tukey's Honestly Significant Difference method. A critical alpha level of .05 was adopted for all significance tests. Effect sizes (ES) were calculated for pairwise comparisons via Hedges' *g* statistic (Hedges, 1981). Hedges' *g* is analogous to Cohen's *d* (Cohen, 1988); however, its calculation involves pooling standard deviations across the experimental groups and control group (Kirk, 1996).

RESULTS

Adherence to the exercise prescriptions was 100%. All participants in the AT group completed at least 18 exercise sessions during the 6-week intervention period and exercised at least 20 min each session ($M = 41.0$ min, $SD = 12.7$). Exercise intensity in the AT group was assessed via ratings of perceived exertion and averaged 13.9 ($SD = 0.8$) on a scale ranging from 6 through 20 (Borg, 1982). This mean rating corresponds to a perceived exertion of "somewhat hard." Each participant in the WT group also completed 18 exercise sessions. Participants in the NT group confined their activities to those of daily living. Preliminary analyses showed that there were no gender differences in

the effects of treatments on fitness (i.e., aerobic capacity and strength) or cardiovascular reactivity and recovery measures.¹

Training Effects on Aerobic Capacity and Strength

Mean ($\pm SE$) treadmill time and aerobic capacity (i.e., VO_{2max} , in $ml \cdot kg^{-1} \cdot min^{-1}$), estimated from treadmill time (Bruce et al., 1973), obtained from each group before and after training are presented in Table 1. The groups exhibited similar treadmill times prior to training. After training, treadmill times were significantly improved for the AT and WT, as indicated by treadmill-time change scores (i.e., posttraining – pretraining). These scores were significantly greater than 0 for the AT ($M = 111.3$ s, $SE = 7.3$) and WT ($M = 59.3$ s, $SE = 8.7$) groups, both $ts(14) \geq 6.8$, both $ps < .001$, but not for the NT ($M = 5.7$ s, $SE = 7.0$). Further, a one-way ANOVA revealed that the AT exhibited a significantly greater improvement in treadmill time than the WT and NT groups, $F(2,42) = 47.0$, $p < .001$. The WT and NT were also different. Additionally, the analysis of 1-RM values obtained from the WT before and after training revealed that the weight training produced a significant increase in strength, Wilks = $\lambda = .051$, $F(6,9) = 27.8$, $p < .001$. Follow-up one-tailed *t* tests revealed that 1-RM values were significantly greater after than before training for all muscle groups (see Table 2), all $ts(14) \geq 2.4$, all $ps < .025$.

Effects of Training on Cardiovascular Activity Levels During Rest, Stress, and Recovery

Mean ($\pm SE$) SBP, DBP, HR, and RPP obtained from each group during rest, stress, and recovery before and after training are presented in Table 3. The analysis confirmed that all four measures were significantly elevated during the mental arithmetic task relative to the resting baseline at both assessments for all groups, $F_s(2,41) \geq 51.8$, $ps < .001$, and that all four measures were significantly lower during recovery than during stress. However, except for DBP, none of the measures returned to resting levels during the recovery period.

¹Gender was initially included as a factor in the statistical design to determine if the men and women in each group exhibited similar changes from pretraining to posttraining in the dependent measures. Treadmill-time change scores (i.e., posttraining – pretraining) were analyzed in a 3×2 (Group \times Gender) ANOVA. This analysis did not reveal a significant interaction. A series of independent two-tailed *t* tests conducted on 1-RM change scores did not reveal a significant difference between the genders for any of the six muscle groups. SBP, DBP, and HR levels were analyzed in $3 \times 2 \times 2 \times 3$ (Group \times Gender \times Session \times Period) MANOVA. RPP was analyzed separately using the same design. These analyses did not yield a significant Group \times Gender \times Session interaction or a significant four-way interaction. Collectively, these results indicate that there were no gender differences in the effects of the treatments on aerobic capacity, strength, or cardiovascular activity levels during rest, psychological stress, and recovery. Hence, gender was dropped from the statistical designs that are reported in the body of the manuscript.

Table 2. Mean (SE) 1-Repetition Maximum (1-RM) Values (in Kilograms) Before and After Training for the Weight-Trained Group

	Before training		After training	
Biceps	35.7	(5.6)	47.0	(6.3)
Triceps	27.7	(4.4)	37.0	(4.8)
Deltoids	36.0	(4.1)	44.3	(4.6)
Pectorals	88.3	(17.7)	101.7	(18.3)
Quadriceps	73.3	(9.0)	87.0	(13.0)
Hamstrings	55.7	(6.1)	62.0	(6.3)

Note. 1-RM values obtained after training were significantly greater than those obtained before training for all muscle groups, all $t_s(14) \geq 2.4$, all $p_s < .025$.

MANOVA conducted on SBP, DBP, and HR revealed a significant Group \times Session interaction, $\lambda = 0.533$, $F(6,80) = 4.94$, $p < .001$, that was due to SBP, $F(2,42) = 9.74$, $p < .001$, and HR, $F(2,42) = 13.09$, $p < .001$. The interaction for DBP approached significance, $F(2,42) = 2.72$, $p < .08$. Post hoc analysis revealed that the groups were different on overall (i.e., averaged across the rest, stress, and recovery periods) SBP (i.e., $NT = AT > WT$) and HR (i.e., $AT > WT$, $AT = NT$, $WT = NT$) before training. To adjust for these initial group differences, posttraining measures were submitted to separate ANCOVAs with pretraining values serving as the covariate. The analysis of overall SBP showed that the AT and WT exhibited significantly lower levels than the NT after training (see Figure 1), but that the AT and WT did not differ, $F(2,41) = 12.00$, $p < .001$. The effect sizes for the AT-NT, WT-NT, and AT-WT comparisons were 1.02, 0.61, and 0.41, respectively. The other analysis revealed

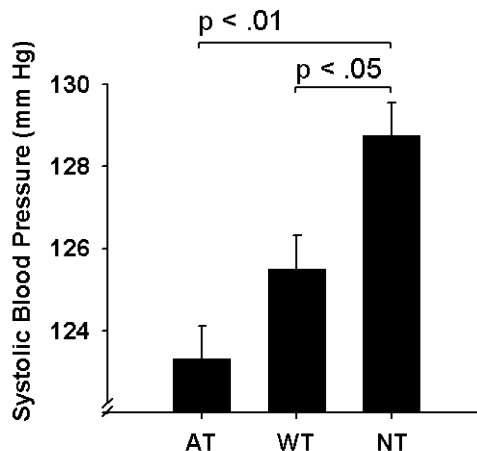


Figure 1. Mean (\pm SE) overall systolic blood pressure (i.e., averaged across rest, stress, and recovery) after treatment for aerobically trained (AT), weight-trained (WT), and no-training (NT) groups. Posttraining means were adjusted via ANCOVA for group differences in pretraining values.

that the AT exhibited a significantly lower overall HR than the WT and NT groups (see Figure 2), $F(2,41) = 14.11$, $p < .001$. The latter two groups did not differ. Effect sizes for the AT-NT, AT-WT, and WT-NT comparisons were 1.09, 0.69, and 0.41, respectively.

The analysis of RPP revealed a significant Group \times Session \times Period interaction, $\lambda = 0.744$, $F(4,82) = 3.26$, $p < .05$. Post hoc contrasts revealed that the groups were different on baseline (i.e., $NT > WT$, $AT = NT$, $AT = WT$), stress (i.e.,

Table 3. Mean (SE) Cardiovascular Activity Levels During Rest, Stress, and Recovery for the Aerobically Trained (AT), Weight-Trained (WT), and No-Training (NT) Groups Before (Time1) and After (Time2) Training

	AT		WT		NT	
	Time1	Time2	Time1	Time2	Time1	Time2
Resting baseline						
SBP (mmHg)	119.9 (1.5)	117.5 (1.2)	116.1 (1.1)	116.1 (1.1)	120.1 (1.4)	121.0 (1.2)
DBP (mmHg)	74.5 (1.3)	73.1 (1.0)	74.9 (1.3)	73.7 (1.3)	75.3 (1.4)	75.7 (1.2)
HR (bpm)	72.7 (0.9)	71.5 (1.1)	72.9 (1.3)	73.0 (1.0)	75.5 (1.4)	75.5 (1.2)
RPP (SBP \times HR)	8,728.1 (208.6)	8,409.1 (203.3)	8,483.6 (214.4)	8,484.4 (184.1)	9,080.8 (249.6)	9,140.3 (196.4)
Stress						
SBP (mmHg)	135.1 (1.7)	129.4 (1.6)	131.1 (1.3)	129.0 (1.5)	135.7 (2.0)	136.2 (1.8)
DBP (mmHg)	77.5 (1.0)	75.6 (1.1)	76.8 (1.0)	76.2 (1.5)	79.0 (1.3)	78.2 (1.3)
HR (bpm)	90.3 (1.9)	85.7 (1.5)	87.4 (1.2)	86.0 (1.3)	88.9 (1.7)	89.1 (1.8)
RPP (SBP \times HR)	12,269.6 (386.9)	11,139.0 (301.9)	11,506.7 (262.4)	11,159.4 (296.9)	12,127.9 (393.1)	12,211.3 (390.5)
Recovery						
SBP (mmHg)	130.5 (2.0)	125.3 (1.6)	128.2 (1.5)	126.3 (1.4)	130.7 (1.6)	132.0 (1.7)
DBP (mmHg)	73.6 (1.4)	73.1 (1.3)	74.4 (1.3)	73.9 (1.3)	76.7 (1.4)	76.9 (1.6)
HR (bpm)	87.2 (3.4)	84.0 (2.8)	84.4 (1.3)	84.0 (1.2)	83.7 (1.7)	85.4 (1.8)
RPP (SBP \times HR)	11,466.6 (615.3)	10,576.7 (452.2)	10,847.0 (256.4)	10,646.1 (259.2)	10,984.5 (334.3)	11,310.2 (337.9)

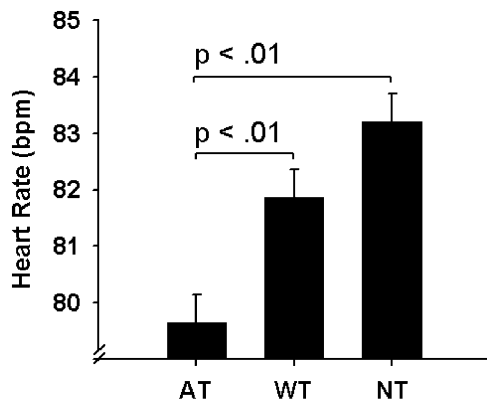


Figure 2. Mean (\pm SE) overall heart rate (i.e., averaged across rest, stress, and recovery) after treatment for aerobically trained (AT), weight-trained (WT), and no-training (NT) groups. Posttraining means were adjusted via ANCOVA for group differences in pretraining values.

AT = NT > WT), and recovery (i.e., AT > WT = NT) levels prior to training. To adjust for these initial group differences, posttraining values were subjected to a mixed 3 \times 3 (Group \times Period) ANCOVA with the pretraining baseline, stress, and recovery values serving as the covariates for their respective posttraining values. This analysis revealed a significant Group \times Period interaction, $F(4,83) = 2.47$, Greenhouse-Geisser $\epsilon = .998$, $p = .05$. Post hoc analysis revealed that the groups exhibited similar baseline values (see Figure 3). However, the AT exhibited a significantly lower RPP than the WT and NT during both stress and recovery. The WT and NT groups also differed during stress and recovery. Effect sizes for the AT-NT, AT-WT, and WT-NT comparisons during stress were 0.92, 0.46, and 0.45, respectively. For recovery, effect sizes for the AT-NT, AT-WT, and WT-NT comparisons were 0.79, 0.38, and 0.40, respectively.

Discussion

This study demonstrated that aerobic exercise training lowers cardiovascular activity levels during rest, psychological stress,

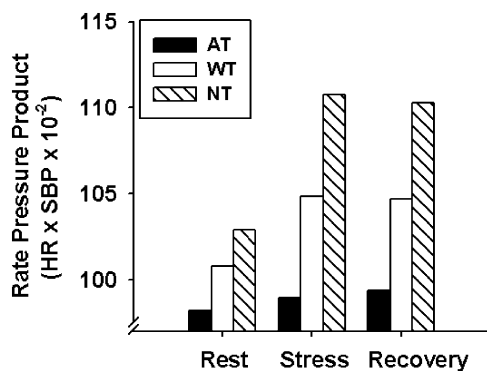


Figure 3. Mean rate-pressure product (RPP) during rest, stress, and recovery after treatment for aerobically trained (AT), weight-trained (WT), and no-training (NT) groups. Posttraining means were adjusted via ANCOVA for group differences in pretraining values; pretraining baseline, stress, and recovery values served as the covariates for their respective posttraining values. Groups were similar on resting RPP but different on stress and recovery RPP.

and recovery in previously sedentary young, nonsmoking, normotensive men and women. Beneficial effects were observed for SBP, HR, and RPP, but not DBP. Overall SBP and HR levels (adjusted for pretraining group differences) were significantly lower after training in the AT group as compared to the NT. These results indicate that aerobic exercise lowered SBP and HR levels during rest, stress, and recovery. Although the groups did not differ on resting RPP, the AT participants did exhibit significantly lower RPP levels (adjusted for pretraining group differences) during psychological stress and recovery relative to the NT group as a result of training. These findings indicate that aerobic exercise training lowered myocardial workload during psychological stress and recovery but not during rest. The AT-NT group difference in stress and recovery RPP emerged because the AT did not react to the cognitive task after training, whereas the NT did. The AT exhibited similar RPP levels during rest, stress, and recovery after training. In contrast, stress and recovery RPP levels were significantly higher than resting levels for the NT.

The posttraining improvements in overall HR as well as stress and recovery RPP levels in the AT group can be attributed to functional cardiovascular adaptations evoked by the aerobic exercise training. Alternative explanations—for example, nonspecific effects of participating in a structured activity program (e.g., attention, expectation, time-out) and the effects of retesting (e.g., familiarity with the tasks at posttest)—can be rejected because the improvements in HR and RPP were significantly greater for the AT than the WT and NT groups. In contrast, the lack of a difference between the AT and WT on overall SBP after training suggests that nonspecific effects could account for the improvement observed in these groups. However, a plausible alternative explanation is that weight training also produced adaptations in the cardiovascular system that lowered blood pressure. Well-controlled studies of normotensive and hypertensive individuals have shown that strength training does lower resting blood pressure (Hagberg et al., 1984; Hurley et al., 1988; Martel et al., 1999), although positive results have not been consistently demonstrated (Cononie et al., 1991; Hagberg et al., 1993). Hence, it is possible that strength training lowers the SBP baseline upon which relative responses are imposed, resulting in lower stress (and recovery) levels. Adaptations to strength training may also explain the difference between the WT and NT in stress and recovery RPP levels observed after training in this study. In this regard weight-trained participants exhibited lower RPP—and, hence, lower myocardial oxygen consumption and workload—during stress and recovery after training than the NT, although the effect sizes were not as great as those for the AT-NT contrast. In previous research, strength training has been included in the design as a control for nonspecific effects (Step toe et al., 1990), under the assumption that it does not produce adaptations that would alter cardiovascular stress levels or relative stress responses. This assumption may not be correct. Claytor (1991) has previously questioned the adequacy of weight training as a control for nonspecific effects. Presently, the evidence regarding the effects of weight training on cardiovascular activity during psychological stress and recovery is quite limited. Only two studies have allowed for a comparison of weight-training and no-training groups (Sinyor et al., 1986; Step toe et al., 1990). These studies did not reveal a significant group difference on heart-rate or blood-pressure reactivity assessed as a change from baseline. Absolute levels of cardiovascular activity during stress and recovery were not analyzed in either study. Additionally, it is

unclear if the weight-training treatments were effective in manipulating strength. Changes in strength were not assessed in either study.

The beneficial effects observed in the WT group relative to the NT are not likely due to an improvement in aerobic capacity. Although the weight-trained participants achieved a statistically reliable increase in treadmill running time and improved more than the NT (~60 s vs. 6 s), the enhanced endurance was likely due to an increase in leg strength (18.7% for the quadriceps and 11.3% for the hamstrings) rather than an increase in aerobic capacity. This interpretation is supported by the results of Marcinik et al. (1991), who found that a 33% increase in cycle ergometry endurance time in young untrained males following 12 weeks of circuit strength training was not associated with a change in treadmill VO_2max or cycle peak VO_2 . Similarly, Hickson, Rosenkoetter, and Brown (1980) found that, although peak VO_2 during cycle ergometry was significantly improved in young men after 10 weeks of strength training, true VO_2max , as measured during a maximal treadmill exercise test, was unchanged. In contrast to the WT and NT, the aerobically trained participants improved their treadmill time by an average of nearly 2 min and their estimated VO_2max by an average of 6.3 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. The improvement in estimated VO_2max in the AT group is similar to that observed in several other studies (Cleroux et al., 1985; Saraganian et al., 1987; Sinyor et al., 1986; Sothmann, Hart & Horn, 1992; Steptoe et al., 1990), in which mean increases of 5.9 to 7.0 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ were reported. Therefore, it appears warranted to conclude that the AT group experienced a change in cardiovascular conditioning that was not achieved by the two control groups.

In contrast to the other measures aerobic training did not alter DBP levels during rest, psychological stress, or recovery, although there was a trend toward significance. The Group \times Session interaction approached significance and, consistent with expectations, the AT group exhibited the greatest decrease in overall DBP levels. The small response magnitudes observed in this study may have hampered our ability to detect a training effect. Although the mental arithmetic challenge elicited a significant increase in DBP, the response was not remarkable—3.7 mmHg or less prior to training. Hence, there was little room for improvement. Another possible reason for the null result is that the training may not have been sufficiently long to produce adaptations that would affect DBP during stress and recovery.

The majority of well-controlled training studies in this area of research have involved middle-aged or older participants, many of whom also had one or more risk factors for exaggerated cardiovascular reactivity, hypertension, and heart disease (e.g., Type A personality, borderline hypertension, smoking; Albright, King, Taylor, & Haskell, 1992; Blumenthal et al., 1988, 1990, 1991, 1997; de Geus et al., 1993; Georiades et al., 2000; Roskies et al., 1986; Seraganian et al., 1987; Schaeffer et al., 1988; Sherwood et al., 1989; Stein & Boutcher, 1992; Throne et al., 2000). Although not all of the results are positive, these studies have frequently shown that aerobic exercise training lowers heart rate and/or blood pressure levels during psychological stress. Improvements have been more consistently reported for HR than blood pressure. Thus, the research supports the notion that aerobic exercise training may be prophylactic for individuals who, due to their age and the presence of other risk factors, are more vulnerable to hypertension and heart disease. However, there is less evidence on younger, nonsmoking, normotensive individuals, and there is a need to determine if the therapeutic potential of

aerobic exercise training extends to this population. Although they are less vulnerable to hypertension and heart disease, it is not likely that they are entirely free of risk because, presumably, they are exposed to chronic psychological stress. The present study provides the first well-controlled demonstration that aerobic training lowers SBP and HR during rest, psychological stress, and recovery in sedentary normotensive, nonsmoking individuals under 30 years of age. The only other studies of this population that are strong methodologically (e.g., employ a no-training control group, random assignment of participants to groups, and maximal graded exercise tests of aerobic capacity) have failed to reveal significant effects for blood pressure or heart rate levels (de Geus, van Doornen, de Visser, & Orlebeke, 1990; Kubitz & Landers, 1993). Although blood pressure and heart rate levels were not specifically examined in the one study (de Geus et al., 1990), resting blood pressure and HR, and blood pressure and HR reactivity quantified as a change from baseline, were not different after aerobic training relative to the no-training group. Similarly, Sinyor et al. (1986) did not observe a difference between aerobic-training and no-training groups in posttraining HR reactivity. However, in the aerobically trained group, HR stress responses were imposed on a significantly lower baseline level as compared to weight-trained and no-training control groups. This would imply that absolute HR levels during stress were also lower in the aerobically trained relative to the participants in the control groups. Relative to the no-training group, the training-induced reduction in resting HR was greater in their study (Sinyor et al., 1986) as compared to ours (12.0 bpm vs. 3.6 bpm). The large effect in their study may be due to a bias introduced by some participants self-selecting into treatments as the investigators attempted but could not achieve random assignment of participants to groups.

Further, two well-controlled studies have also shown that aerobic training lowers stress and recovery RPP levels in middle-aged Type A men (Blumenthal et al., 1988, 1990). The present study extends these findings to young, normotensive men and women. The magnitude of aerobic training effects observed in the young men and women of the present study were similar to those exhibited by the older Type A men in Blumenthal et al.'s (1988) study. This latter study employed a strength/flexibility training group as the control. The effect of aerobic training on RPP levels, estimated as the pre- to posttraining difference for the aerobically trained group minus the same difference for the weight-trained group, was approximately 590 and 535 for stress and recovery, respectively, in the present study. These estimates indicate that aerobic training lowered stress and recovery RPP levels 590 and 535, respectively, more than did weight training. In Blumenthal et al.'s (1988) study the corresponding estimates were 600 and 690 for stress and recovery RPP levels, respectively. However, larger effects were reported in their subsequent study (Blumenthal et al., 1990). In this latter study aerobic exercise lowered stress RPP levels approximately 1250 more than strength and flexibility training; recovery RPP levels were lowered by approximately 1165. The larger effects observed in the subsequent study cannot be explained by differences in methods or the magnitude of improvement in aerobic capacity achieved by the AT group. One possibility, though, is that strength and flexibility training yielded different effects in the two studies (Blumenthal et al., 1988, 1990), even though the reported training protocols were very similar. Muscular strength was not assessed before or after training in either study. Hence, it cannot be determined if training produced similar improvements in strength in the two studies. If weight

training does produce adaptations that lower SBP during rest, psychological stress, and recovery, as the results of this study suggest, then differences in the effectiveness of strength training in the Blumenthal et al. studies could account for the differences across studies in stress and recovery RPP. Less effective weight training would likely yield a greater AT-WT difference in improvements in stress and recovery RPP. Notably, the improvement in stress and recovery RPP achieved by the aerobically trained participants over the weight-trained in the Blumenthal et al. (1990) study were similar to the improvement achieved by the AT relative to the NT in the present study. Specifically, the AT-NT difference in the improvement (lowering) of stress and recovery RPP levels were 1179 and 1095, respectively, in the present study. Clearly, additional research involving strength training and no-training groups, with assessments of aerobic capacity and strength in identified target muscles before and after training, is needed to further assess the relative effects of aerobic and weight training on RPP stress responses.

Mechanisms mediating the effects of aerobic training on HR, SBP, and RPP in the present study are not known. However, with respect to HR, there are two possibilities. First, studies of borderline hypertensive rats have shown that trained animals exhibit higher vagal tone and lower sympathetic tone during foot shock stress than their nontrained counterparts (Cox, 1991). These findings would suggest that the lower overall posttraining HR observed in the AT was due to augmented vagal tone and/or reduced sympathetic tone. Although this seems reasonable, the limited evidence from training studies with humans does not support a neurogenic explanation. For example, there is no evidence that aerobic training alters the level of respiratory sinus arrhythmia (RSA), an index of vagal tone, during psychological stress or RSA reactivity (de Geus et al., 1990; Kubitz & Landers, 1993). Further, studies involving the measurement of plasma epinephrine and norepinephrine levels or preejection period (PEP) reactivity have failed to show that aerobic training alters sympathetic influences on the heart during psychological stress (Clayton, 1991; Cleroux et al., 1985; de Geus et al., 1990, 1993; Sinyor et al., 1988; Sothmann et al., 1992). However, evidence of lower sympathetic influence on the heart during psychological stress was observed in the aerobically trained individuals in one study (Sherwood et al., 1989), but the effect was dependent upon blood pressure status.

The second possibility is that the lower overall posttraining HR observed in the AT was secondary to an improvement in cardiac performance (i.e., enhanced stroke volume). Cardiac performance would be enhanced by any training-induced adaptations that increase ventricular filling or decrease myocardial work (i.e., due to a decrease in afterload). Aerobic exercise training is known to expand the plasma volume and will also lower HR during submaximal work (Blomqvist, 1983; McArdle et al., 1996). Both effects would increase end-diastolic volume. Additionally, a reduction in overall vascular resistance would increase ventricular filling and decrease afterload. There is evidence that aerobic training lowers total peripheral resistance (TPR) during rest and psychological stress (Georgiades et al., 2000). The evidence regarding TPR reactivity, calculated as a change score, is mixed (de Geus et al., 1990, 1993; Sherwood et al., 1989). Lastly, left ventricular hypertrophy, which is commonly observed in endurance trained individuals (McArdle et al., 1996), can also enhance stroke volume and, thereby, decrease HR. However, structural adaptations in the heart are not typically observed following brief training intervals (Ricci et al., 1982; Thompson

et al., 1981). It should be noted that a training-induced reduction in TPR could partially account for the lower overall SBP and the lower stress and recovery RPP levels observed in the AT after training. The lower HR could also have contributed to these effects by lowering cardiac output.

The improvements in SBP, HR, and RPP achieved as a result of aerobic training were modest in absolute magnitude. As compared to no training, aerobic training lowered overall SBP by 5.5 mmHg, overall HR by 3.6 bpm, and stress and recovery RPP levels by 1179 and 1095 (HR \times SBP), respectively. As the clinical significance of exercise-induced reductions in cardiovascular activity during stress is not presently known, the clinical significance of these findings is unclear. Several prospective studies have shown that exaggerated blood pressure responses to the cold pressor test (Keys et al., 1971; Menkes et al., 1989; Wood, Sheps, Elveback, & Schirger, 1984) and a mental arithmetic challenge (Matthews, Woodall, & Allen, 1993) predict future blood pressure levels and hypertension status. However, there are no studies to our knowledge that demonstrate that exercise-induced reductions in cardiovascular stress levels (or reactivity) lowers the future incidence of cardiovascular disease. Hence, there are no data to suggest what constitutes a minimal clinically significant reduction in cardiovascular stress levels. Nonetheless, the recent release of the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-7; Chobanian et al., 2003), which includes revised blood pressure classification guidelines, would seem to elevate the clinical importance of even modest changes such as those observed in the present study. The most notable change in the guidelines is the inclusion of a new category: Prehypertensive. The JNC is recommending that individuals with a systolic blood pressure of 120–139 mmHg or a diastolic blood pressure of 80–90 be classified as “Prehypertensive.” These blood pressure ranges were previously classified as normal to high-normal (Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, 1997). The new category was prompted by the recognition that prehypertensive individuals have a higher risk of progressing to hypertension. The risk of developing hypertension is doubled for individuals with a blood pressure in the range of 130–139/80–89 mmHg relative to those with lower values (Vasan, Larson, Leip, Kannel, & Levy, 2001, cited in JNC-7). The revised blood pressure classification clearly reflects greater alarm about blood pressures that were previously considered normal. The JNC recommends lifestyle modifications including increased physical activity for prehypertensive individuals. Although the effects in this study were modest in absolute terms, they do represent a relative benefit that aerobically trained young men and women would experience continuously during wakeful hours—that is, during rest, psychological stress, and recovery. As the relationship between blood pressure and cardiovascular disease events is continuous (Chobanian et al., 2003), it seems likely that a 5.5-mmHg reduction in SBP experienced continuously during waking hours across a long period of time would result in a meaningful reduction in the risk of CV disease.

An examination of effect sizes also indicates that the effects are meaningful. Effect sizes for overall SBP and HR exceeded 1.00 (i.e., 1.03 and 1.09, respectively) and were also substantial for stress and recovery RPP levels: 0.92 and 0.79, respectively. These values exceed the average of approximately 0.50 found in several surveys of effect sizes in behavioral research (Cooper & Findley, 1982; Crews & Landers, 1987; Haase, Waechter, & Solomon, 1982; Kirk, 1996; Sedlmeier & Gigerenzer, 1989). In

accordance with the widely accepted convention proposed by Cohen (1988, 1992), effect sizes ≥ 0.80 may be considered large. Additionally, the effect sizes observed in this study compare favorably to or exceed those reported in effect-size surveys of exercise studies. The median effect size observed in one survey was approximately 0.70 (Thomas, Salazar, & Landers, 1991), and the effect sizes obtained in the present study were as large or larger than 60% of the 93 effect sizes cited in this study. In a meta-analysis of studies on aerobic fitness and cardiovascular reactivity to psychological stress, Crews and Landers (1987) found mean effect sizes of 0.42 for SBP and 0.39 for HR. Hence, the large effect sizes observed in the present study suggest that reliable and meaningful effects are already emerging after a relatively short training period in young, normotensive individuals. In light of age-related changes in the cardiovascular system that result most notably in a rise in blood pressure and a decline in aerobic capacity (Docherty, 1990; Kaplan, 1994; McArdle et al., 1996), it is plausible that the effects of aerobic training on overall blood pressure and myocardial workload will become greater as the cardiovascular system ages.

Additionally, the lower stress HR levels induced by aerobic training may confer a benefit by lowering atherogenic activity. Beere, Glagov and Zarins (1984) have hypothesized that higher HR produces a pulsatile blood flow pattern (i.e., forward and reverse flows) that is associated with greater atherogenesis. In a test of this hypothesis they compared monkeys that had either

sinoatrial node ablation, sham surgery, or an atherogenic diet only on several measures of atherosclerosis. Although the groups did not differ on any measures, a post hoc comparison of low- and high-HR groups showed that the low-HR group had a significantly lower percentage of lesions with more than 25% stenosis, smaller average lesion areas, and a smaller average percent stenosis in the most occluded artery. Therefore, the lower stress HR levels induced by aerobic exercise training may render an arterial blood flow pattern that is less favorable to the formation of atherosclerotic plaques.

In summary, a 6-week program of aerobic exercise training lowered overall HR and SBP as well as stress and recovery RPP levels in young, nonsmoking, normotensive men and women. However, it did not alter SBP levels differentially from resistance training, nor did it alter DBP. The improvement in HR and RPP levels, although limited in absolute magnitude, seems remarkable in light of the young, asymptomatic population examined. As such, the results of this study suggest that aerobic exercise training could reduce HR, SBP, and RPP during the chronic psychological stress of normal daily living, even in young, healthy, normotensive men and women. Further, they provide strong support for the efficacy of regular physical activity participation in reducing the risk of coronary heart disease, especially if the behavior is maintained through the aging process when the risk of such occurrences is progressively higher and the opportunity for exercise-induced benefits is likely even greater.

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