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Decreased exercise blood pressure in older adults after exercise training: contributions of increased fitness and decreased fatness

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Abstract

Objective—To describe the contribution of changes in fitness and fatness resulting from exercise training on changes in submaximal exercise blood pressure (BP) during treadmill testing.

Design and Setting—Prospective, randomized, controlled trial

Participants—Sedentary older adults (n=115), with untreated prehypertension or mild hypertension

Intervention—Six-month supervised aerobic and strength training

Main Outcome Measurement—BP was assessed at rest and during each stage of a maximal graded exercise test (GXT) that determined VO₂peak. General and regional fatness was assessed by anthropometry, dual-energy x-ray absorptiometry, and magnetic resonance imaging. BP changes were calculated for each GXT stage and multivariate regression models were used to describe the association of changes in exercise BP with changes in fitness and fatness.

Results—After training, exercisers versus controls had significantly increased VO₂peak and significantly lower measures of general and regional fatness. Also, stage-specific SBP was significantly lower at stage 3 (−9.4 vs. −1.6 mmHg, *p*=0.03) and stage 4 (−7.9 vs. −1.2 mmHg, *p*=0.03). Pooled regression analysis across all stages showed that exercisers had a 7.1 mmHg reduction in SBP, but this reduction fell short of statistical significance (*p*=0.12) compared to controls. A 1.0 ml/kg/min increase in VO₂peak and a 1.0 cm decrease in waist circumference independently predicted a 1.0 mmHg decrease in exercise SBP (*p*=0.04 and *p*=0.001, respectively).

Conclusions—Decreased exercise SBP was independently associated with decreased waist circumference, a marker of abdominal obesity, and increased fitness. Our findings suggest that exercise training improves multiple factors that have independent influence on SBP.

Keywords

hypertension; exercise test; randomized controlled trial; obesity; physical fitness

CONFLICTS OF INTEREST

All authors of this manuscript declare no completing interests.

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INTRODUCTION

Exercise training decreases resting, ambulatory, and exercise blood pressure (BP) in individuals with hypertension.[1–3] Measurement of exercise BP provides additional information about hypertensive status because exercise BP is thought to be less responsive to external stressors and anxiety compared to resting BP, and thus may help distinguish true from ‘white-coat’ hypertension.[4] Furthermore, BP response during exercise testing is a valuable prognostic tool that predicts long-term mortality and cardiovascular events independently of resting BP.[5–7] Lastly, exercise BP reflects patients’ exposures to BP at varying levels of exertion without using an ambulatory BP monitoring device.

Understanding how increased fitness and decreased fatness achieved through exercise training contribute to changes in exercise BP is important for maximizing the effectiveness of interventions and explaining variability in response.[1] Reductions in resting BP due to exercise training have been associated with favorable changes in fitness and obesity.[8, 9] However, previous studies of the effect of exercise training on BP during exercise testing did not report these associations.[10–14] Since exercise BP is valuable for long-term patient prognosis beyond resting BP, understanding pathways to reducing exercise BP could add important information about intermediate targets in fitness and fatness for exercise interventions. Our aim was to describe the contributions of increased fitness and decreased fatness due to a 6-month aerobic and strength training intervention on reductions in exercise BP.

METHODS

Participants

Subjects in the present study completed a randomized controlled trial of exercise for mild hypertension.[9] Briefly, sedentary adults, aged 55–75, with untreated prehypertension or mild hypertension were randomized to a 6-month supervised exercise program or to a usual care control group. Eligible participants had to have a resting systolic BP (SBP) between 130–159 mmHg or a diastolic BP (DBP) from 80–99 mmHg on 2 consecutive visits and an average in this range over 4 weekly visits. Subjects using a single antihypertensive medication were allowed to undergo the same study screening after a 2-week washout period approved by their physician. Major exclusions included known clinical cardiovascular disease or other serious illnesses, smoking, diabetes mellitus, or self-report of moderate-intensity exercise more than 90 minutes per week at baseline

Exercise Training Intervention

The exercise intervention followed the American College of Sports Medicine guidelines for hypertension and included 3 supervised sessions per week.[1] Each session included a 10–15 minute warm-up, 45 minutes of aerobic exercise at a target heart rate between 60–90% of maximum heart rate based on baseline exercise testing, and a cool down. Additionally, each session included 7 weight training exercises (latissimus dorsi pull down, leg extension, leg curl, bench press, leg press, shoulder press, and seated mid-rowing) performed as 2 sets of 12–15 repetitions at 50% of 1-repetition maximum. Weight was increased when subjects could easily complete 15 repetitions. Participants were expected to attend at least 62 sessions in the 26-week period (80%) and an extra month was allowed for participants to reach the required number of sessions if necessary. All subjects, including controls, were given written exercise guidelines from the National Institute of Aging during screening (<http://www.nia.nih.gov/HealthInformation/Publications/ExerciseGuide>). Controls received no further intervention.

Measurement of Exercise Blood Pressure and Fitness

Subjects completed a graded exercise test (GXT) to measure aerobic fitness by peak oxygen ($\text{VO}_{2\text{peak}}$) at baseline and at 6 months. The GXT used a modified Balke protocol where walking began at 4.8 km/h and 0% grade, increased 2.5% every 3 minutes, and stopped at volitional fatigue. Maximum effort was validated by reaching 18 on the Borg Rating of Perceived Exertion scale, respiratory exchange ratio of 1.1 or greater, and/or heart rate or VO_2 plateau. Blood pressure was taken during the last minute of each stage by an aneroid cuff by study staff experienced in measuring GXT BP. To limit the effects of habituation, all participants underwent an initial practice exercise test during screening, before baseline measurement.

Measurement of Resting Blood Pressure

Resting BP was assessed by an automated device (Dinamap MPS Select; Johnson & Johnson, New Brunswick, NJ) at the same time of day. Resting BP was taken 3 times with 1 minute between readings and averaged. If BP differed by more than 5 mmHg between readings, additional readings were taken until 3 readings within 5 mmHg were obtained.

Measurement of Fatness

We obtained several measures of general and abdominal fatness. Weight, height, and waist circumference were measured by trained study staff. Percent total body fat and lean body mass were assessed by dual x-ray absorptiometry (DEXA) (GE Lunar Prodigy; General Electric Medical Systems, Milwaukee, WI). Images of abdominal, visceral, and subcutaneous fat were measured with magnetic resonance imaging (MRI) (Siemens Vision 1.5T, Siemens Medical Systems, Iselin, NJ). An experienced reader measured fat volume from these images using the National Institutes of Health application (<http://rsb.info.nih.gov/nih-image/>).

Statistical Analysis

Baseline and 6-month change in study outcome variables were compared by study group using independent t-tests. All variables were checked for normality.

Six-month changes in exercise SBP and DBP were calculated for each stage that a participant completed during both the baseline and 6-month GXT. For GXT stages 1 through 5, we tested for significant change in exercise BP from baseline within each intervention group using one-sample t-tests and for significant difference between intervention groups with independent t-tests. This was not done beyond stage 5 because too few participants completed these stages during the baseline GXT. Next, Pearson correlation coefficients were calculated comparing change in exercise BP to changes in parameters of fitness and fatness using pooled data from all subjects for stages 1 to 5. METs for stages 1 through 5 were calculated from directly measured oxygen uptake during the GXT and averaged across all participants at each stage.

To assess the contributions of changes in fitness and fatness to changes in exercise BP, we used generalized estimating equations (GEE) linear regression across all GXT stages, which account for the correlation between measurements from the same individual at different stages.[15] First, we examined the average effect of the intervention across all stages. Further GEE models added changes in fitness and fatness parameters to assess their associations with exercise BP. Changes in different measures of fatness (e.g. body mass index, waist circumference, abdominal fat) were collinear. Therefore, variable choices for fatness measure in the multivariate models were based on the results of the correlation analysis and R^2 . Analyses were repeated controlling for GXT stage. Data analysis was

performed using the Stata 10 Statistical Package (Statacorp®, College Station, Tx). Type I error rate was set at $\alpha = 0.05$.

RESULTS

Baseline Characteristics

Of 115 subjects randomized, 104 (90%) completed the study ($n = 51$ exercisers, 25 men and 26 women; and $n = 53$ controls, 26 men and 27 women). Mean age was 64.6 ± 5.7 years, and the ethnic composition was 87% non-Hispanic white, 11% African American, 1% Asian American, and 1% Hispanic.

At the time of randomization, 56 subjects had resting SBP ≥ 140 mmHg and 7 subjects had resting DBP ≥ 90 mmHg. As reported elsewhere, there were no significant differences at baseline between study groups on any variable reported herein.[9] Compliance to the exercise intervention was excellent and subjects attended 88% of the prescribed exercise sessions. Only 1 of the 51 participants did not complete at least 80% of the prescribed sessions. Exercise heart rate for the aerobic phase of the workout was within the target range 98% of the time.

Effect of Exercise Training on Blood Pressure, Fitness, and Fatness

Baseline and change measurements for BP, GXT, and body fatness are reported in Table 1. At baseline, no significant differences were observed. At follow-up, exercisers significantly decreased resting DBP compared to controls. Exercisers significantly increased maximum exercise SBP, VO₂peak, and stages completed in the GXT compared with controls. Exercisers significantly improved all measures of fatness and body composition compared to controls, except waist-hip ratio.

Compared to baseline, exercisers had significant reductions in SBP at stages 2, 3, 4, and 5 and controls at stage 2(Figure). The 6-month change in exercise SBP differed significantly between exercisers and controls at stages 3 ($p=0.03$) and 4 ($p=0.03$). While DBP decreased significantly from baseline among exercisers at stages 1 through 5 and did not differ significantly for controls at any stage, there were no statistically significant differences between groups.

Correlation of Changes in Exercise Blood Pressure with Fitness and Fatness

Pearson correlation coefficients of change in stage specific SBP and changes in other variables are shown in Table 2. Reductions in weight, body mass index, and waist circumference were significantly associated with lower SBP across all 5 stages. The reduction in exercise SBP was significantly correlated to increases in VO₂peak at only 2 stages and change in exercise SBP did not correlate with change in resting SBP at any stage. A reduction in exercise DBP was only significantly correlated to reductions in abdominal total fat (3 stages), abdominal subcutaneous fat (3 stages), increases in VO₂peak (2 stages), and reduction in abdominal visceral fat (1 stage) (data not shown).

Contribution of Changes in Fitness and Fatness to Changes in Exercise Blood Pressure

Table 3 reports results of the regression analyses. The model represents average group effects across all stages. Model 1 estimates the average change in exercise SBP between baseline and 6 months in exercisers versus controls. In controls, average exercise SBP decreased by 3.1 mmHg. In exercisers, average exercise SBP decreased by an additional 4.0 mmHg, but this result fell short of statistical significance ($p=0.12$). Next, change in VO₂peak and the measures of adiposity listed in Table 2 were added to the model to assess the association of the change in exercise SBP by changes in fitness and fatness. A 1.0 ml/kg/

min increase in VO₂peak was significantly associated with a decrease of 1.2 mmHg in exercise SBP ($p=0.008$) (model 2). Measures of fatness were highly collinear and change in weight, body mass index, total body fat, total abdominal fat, subcutaneous abdominal fat, visceral abdominal fat, and waist circumference significantly predicted decreased exercise SBP. Waist circumference was the strongest predictor of decreases in exercise SBP (by R²), with a -1.0 cm decrease in waist circumference being associated with a 1.1 mmHg decrease in exercise SBP ($p<0.001$) (model 3). A final model (model 4) includes change in VO₂peak, change in waist circumference, and intervention group as covariates. A 1.0 ml/kg/min increase in VO₂peak and a 1.0 cm decrease in waist circumference independently predicted a -1.0 mmHg decrease in exercise SBP ($p=0.04$ and 0.001 , respectively). The analysis was repeated controlling for GXT stage and results were similar.

Models were repeated for changes in exercise DBP. Average decrease in exercise DBP across all stages among controls was 1.8 mmHg and exercisers decreased an additional 3.7 mmHg ($p=0.07$). Change in exercise DBP across all stages was not associated significantly with any measure of fitness or body composition. Change in total abdominal fat was most strongly associated with changes exercise DBP of all of the fatness measures, but the trend fell short of statistical significance ($p=0.06$).

DISCUSSION

The main finding in this study is the independent effects of changes in fitness and measures of body composition on changes in exercise BP. This parallels the observation that improvements in adiposity and exercise training have independent effects on reductions in resting BP.[2,3,16] Though the association of increased fitness and decreased exercise SBP is not surprising, the more novel finding is that reductions in obesity also predicted changes in exercise SBP. These findings suggest that exercise training has multiple, additive pathways through which it can improve submaximal exercise BP. In our study, exercisers achieved a 4.0 ml/kg/min increase in VO₂peak and a 3 cm decrease in waist circumference, which would translate to a total decrease in exercise SBP of 7 mmHg. Though our trial did not provide dietary advice about weight loss, the fat-reducing effect of exercise, particularly abdominal fat, appears to have an independent, beneficial effect on exercise BP. The independent effects of increased fitness and decreased fatness may also explain variation in the exercise BP-lowering effect of exercise training across individuals.

Our results, that exercise training produces modest decreases in exercise SBP at fixed, submaximal workloads, are consistent with previous reports.[10–14] When the results of 10 studies of the effect of aerobic exercise training on exercise SBP at a specified workload were combined, a statistically significant average decrease of -7 mmHg in exercise SBP was observed.[1] These studies were heterogeneous with respect to age, hypertension, and length and type of aerobic exercise intervention. Our study differs from these studies in two ways. First, we measured exercise BP over a range of workloads rather than a single workload. Interestingly, while exercise SBP was lower among trained subjects at all stages, this decrease reached statistical significance at higher levels of work compared to lower levels of work. This may reflect larger effect sizes as the absolute exercise SBP increases. A second distinction is that subjects in our study participated in both aerobic and resistance training. While the independent effect of resistance training on resting SBP in randomized trials appears to be at best a modest fall in BP[17], the separate effect of resistance training on exercise BP herein is unknown because we used a combined program. The resistance component may have contributed to reduced exercise SBP through effects on body composition, though future research needs to confirm this possibility. Though we did not see a significant change in exercise DBP comparing exercisers and controls, the intervention group significantly decreased exercise DBP compared to baseline for stages 1 through 5

(Figure). We also saw that changes in exercise DBP were somewhat associated with measures of fatness, but not as strongly as observed with exercise SBP. This may result from the stability of DBP over increasing exercise workloads, thereby having a lesser potential to be affected by an exercise intervention.

Blood pressure measured during a GXT may be useful for quantifying the effect of exercise training on BP during normal, daily exertion, especially at the lower GXT stages. Though ambulatory BP monitoring can be used for this purpose, a commonly administered exercise test performed before and after training can also reflect non-resting BP. Future studies should examine the correlation between changes in ambulatory BP and exercise BP obtained during GXT.

We observed that the effect of exercise training on maximal exercise BP can lead to false conclusions because trained subjects complete a greater number of stages during a GXT following exercise training. Maximal exercise BP was achieved at a higher stage of work and may thus appear to have increased in our trained subjects. By computing stage-specific changes in BP over a range of workloads, we were able to account for the level of work and gain insight into how non-resting BP may change with exercise training. Furthermore, the hemodynamic response at maximal exertion does not reflect common levels of work that an individual experiences on an average day. Hence, changes in exercise BP at fixed, submaximal workloads during a GXT may be more informative when evaluating a training program and also more clinically relevant to decreased daily exposure to BP.

Strengths of our study include the randomized, controlled trial design, the 6-month, supervised intervention, high compliance to exercise intensity and frequency, standardized measurement of BP, measurement of VO₂peak, and excellent characterization of fatness by several measures of anthropometry and high-tech imaging methods. Our major study limitation is that the parent study was powered to detect a difference in resting BP, which averaged the value of at least 4 measurements obtained on separate days. Conversely, exercise BP at each stage was a single measure, which is less precise. This may have reduced our ability to detect a significant result and account for our observation that the overall decrease in exercise BP across all GXT stages among exercisers fell short of statistical significance versus controls. A post-hoc power calculation estimated we would need a sample size of at least 67 patients per intervention group to achieve 80% power.

In conclusion, exercise training has a beneficial effect on exercise BP at fixed stages of work during a GXT that is independently associated with changes in fitness and fatness. The separate effects of increased fitness and decreased fatness on exercise BP emphasize the need to achieve both targets to achieve maximal decreases in BP at varying levels of exertion through exercise training and may help explain variability in response to exercise training across individuals.

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TOPIC BOX 1**WHAT IS ALREADY KNOWN ON THIS TOPIC**

Exercise training reduces submaximal exercise systolic blood pressure, yet the relative contributions of increased fitness and decreased fatness to changes in exercise blood pressure are unknown. Understanding these relationships could inform exercise training targets and explain individual variability in the effectiveness of exercise training for blood pressure reduction.

TOPIC BOX 2**WHAT THIS STUDY ADDS**

Following 6-months of exercise training, decreased waist circumference and increased VO₂peak were each independently associated with decreased systolic blood pressure during a graded exercise stress test. These findings suggest that individuals both increasing fitness and decreasing fatness will have the greatest reductions in exercise blood pressure.

Figure. 6-month Change in Blood Pressure by Stage of a Graded Exercise Stress Test

Abbreviations: SBP, systolic blood pressure; DBP diastolic blood pressure

* $p < 0.05$ comparing exercisers and controls

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Mean (SD) Baseline and 6-month Change from Baseline Measurements in Participants of a 6-month Exercise Intervention (n=51) and Controls (n=53)

Table 1

	BASELINE		6-MONTH CHANGE FROM BASELINE			
	Exercisers (n = 51)	Control (n=53)	p-value	Exercisers (n = 51)	Control (n=53)	p-value
<i>Resting Blood Pressure</i>						
Resting SBP, mmHg	140 (8)	142 (8)	0.34	-5.3 (10.1)	-4.5 (8.2)	0.65
Resting DBP, mmHg	77 (7)	76 (9)	0.78	-3.7 (4.8)	-1.5 (4.9)	0.02*
<i>Measures from the Graded Exercise Stress Test</i>						
VO ₂ peak, ml/kg/min	24.4 (5.2)	24.2 (5.3)	0.85	+4.0 (2.9)	-0.1 (2.3)	<0.001*
Stages completed during GXT	5.1 (1.5)	4.8 (1.5)	0.33	+1.1 (0.8)	-0.2 (1.0)	<0.001*
Maximum exercise SBP, mmHg	222 (22)	226 (19)	0.39	+5.7 (14.8)	-1.0 (16.0)	0.03*
Maximum exercise DBP, mmHg	99 (12)	97 (12)	0.28	-1.5 (16.8)	-1.7 (11.5)	0.94
<i>Measures of Fatness</i>						
Weight, kg	83 (15)	85 (19)	0.61	-2.3 (3.0)	-0.5 (2.4)	0.002*
Body mass index, kg/m ²	29.4 (3.8)	29.7 (5.0)	0.75	-0.8 (1.1)	-0.2 (0.9)	0.001*
Total body fat (DEXA), %	38 (9)	38 (10)	0.91	-3.5 (2.5)	-0.2 (1.9)	<0.001*
Lean body mass (DEXA), %	59 (9)	59 (10)	0.85	+3.5 (2.4)	+0.2 (1.9)	<0.001*
Abdominal fat, total (MRI), cm ²	433 (116)	450 (165)	0.55	-52 (49)	-7 (50)	<0.001*
Abdominal fat, subcutaneous (MRI), cm ²	285 (105)	306 (133)	0.39	-26 (32)	-3 (32)	<0.001*
Abdominal fat, visceral (MRI), cm ²	147 (67)	143 (69)	0.77	-27 (31)	-4 (26)	<0.001*
Waist-hip ratio	0.89 (.10)	0.90 (0.10)	0.76	-0.01 (0.03)	-0.01 (0.03)	0.52
Waist circumference, cm	94.0 (11.8)	95.0 (14.0)	0.69	-2.9 (4.3)	-0.8 (3.4)	0.009*

Abbreviations: GXT: graded exercise test, DEXA: dual-energy x-ray absorptiometry, MRI: magnetic resonance imaging.

* $p < 0.05$

Table 2

Pearson Correlation Coefficients (p-value) of Change in Stage Specific Systolic Blood Pressure and Change in Selected Variables following a 6-Month Randomized Trial of an Aerobic and Strength Training Intervention Stage

	SBP				
	Stage 1 METS = 4 n=99	Stage 2 METS = 4.5 n=98	Stage 3 METS = 5 n=92	Stage 4 METS = 6 n=78	Stage 5 METS = 7 n=50
Δ VO ₂ peak	-0.13 (0.21)	-0.18 (0.07)	-0.38 (0.0002)*	-0.39 (0.0004)*	-0.25 (0.08)
Δ Weight	0.25 (0.01)*	0.32 (0.001)*	0.34 (0.0009)*	0.31 (0.005)*	0.29 (0.04)*
Δ Body mass index	0.22 (0.03)*	0.30 (0.003)*	0.33 (0.002)*	0.30 (0.008)*	0.28 (0.05)*
Δ Total body fat	0.12 (0.23)	0.17 (0.09)	0.25 (0.02)*	0.25 (0.03)*	0.36 (0.01)*
Δ Lean body mass	-0.11 (0.28)	-0.16 (0.12)	-0.24 (0.02)*	-0.24 (0.04)*	-0.35 (0.01)*
Δ Abdominal fat, total	0.12 (0.23)	0.26 (0.009)*	0.24 (0.02)*	0.41 (0.0002)*	0.35 (0.02)*
Δ Abdominal fat, visceral	0.12 (0.25)	0.18 (0.08)	0.021 (0.05)*	0.39 (0.0005)*	0.31 (0.03)*
Δ Abdominal fat, subcutaneous	0.09 (0.36)	0.27 (0.009)*	0.20 (0.06)	0.30 (0.008)*	0.25 (0.09)
Δ Waist-hip ratio	0.17 (0.09)	0.07 (0.47)	0.06 (0.60)	0.08 (0.48)	0.025 (0.08)
Δ Waist circumference	0.30 (0.002)*	0.23 (0.03)*	0.27 (0.01)*	0.24 (0.04)*	0.37 (0.009)*
Δ Resting SBP	0.06 (0.58)	0.07 (0.47)	0.05 (0.67)	0.11 (0.32)	0.24 (0.09)

* p<0.05

Table 3

Generalized Estimating Equations Linear Regression Models Predicting Change in SBP following a 6-Month Randomized Trial of an Aerobic and Strength Training Intervention

	Covariates	Δ SBP (mmHg)	p-value
Model 1	Intercept [*]	-3.1	0.09
	Intervention group ^{**}	-4.0	0.12
Model 2	Intercept	-3.2	0.07
	Intervention group	+1.1	0.74
	Δ VO ₂ peak (ml/kg/min)	-1.2	0.008 [†]
Model 3	Intercept	-2.1	0.22
	Intervention group	+1.5	0.55
	Δ Waist circumference (cm)	+1.1	<0.001 [†]
Model 4	Intercept	-2.3	0.18
	Intervention group	+2.1	0.48
	Δ VO ₂ peak (ml/kg/min)	-1.0	0.04 [†]
	Δ Waist circumference (cm)	+1.0	0.001 [†]

^{*} controls

^{**} difference, exercisers to controls

[†] p<0.05