

Blood Pressure Responses to Acute and Chronic Exercise Are Related in Prehypertension

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ABSTRACT

LIU, S., J. GOODMAN, R. NOLAN, S. LACOMBE, and S. G. THOMAS. Blood Pressure Responses to Acute and Chronic Exercise Are Related in Prehypertension. *Med. Sci. Sports Exerc.*, Vol. 44, No. 9, pp. 1644–1652, 2012. **Purpose:** Aerobic exercise is recommended as a way to prevent hypertension. However, about 25% of individuals receive minimal antihypertensive benefits associated with chronic exercise training. Thus, we attempt to identify those “nonresponders” to chronic exercise on the basis of their blood pressure (BP) responses to acute exercise (single session). Our primary objective was to correlate the magnitude of BP decrease after acute exercise to the magnitude of BP reduction after chronic exercise. Our secondary objective was to examine the correlates of BP reduction after acute and chronic exercise. **Methods:** Seventeen prehypertensive (120 to 139/80 to 89 mm Hg) males and females (45–60 yr old) underwent acute exercise assessments before an 8-wk walking/jogging program (four times per week, 30 min per session, 65% maximum oxygen consumption). BP, hemodynamics, HR variability, and baroreflex sensitivity were assessed before and after acute exercise and chronic training. **Results:** BP was significantly reduced $-7.2 \pm 1.2/-4.2 \pm 1.0$ and $-7.0 \pm 1.4/-5.2 \pm 1.2$ mm Hg relative to baseline after acute (30 min at 65% maximum oxygen consumption) and chronic exercise, respectively ($P < 0.01$). The magnitude of change in systolic BP after acute exercise was strongly correlated with change in resting systolic BP after chronic training, $r = 0.89$, $P < 0.01$. A similar correlation was observed with diastolic BP, $r = 0.75$, $P < 0.01$. After acute exercise, significant reductions in total power (ms^2) and baroreflex sensitivity were observed in both sexes ($P < 0.01$). However, after chronic exercise, only men demonstrated a significant reduction in the low-frequency-to-high-frequency ratio (-36%), $P < 0.01$. **Conclusions:** The magnitude of the acute BP-lowering with exercise may predict the extent of BP lowering after chronic training interventions in prehypertensive individuals. **Key Words:** AEROBIC EXERCISE, BLOOD PRESSURE, EXERCISE TRAINING RESPONSE, HR VARIABILITY, BAROREFLEX SENSITIVITY, ARTERIAL STIFFNESS

Lifestyle interventions and, in particular, chronic aerobic exercise may prevent or delay the development of hypertension (systolic blood pressure (SBP) > 140 mm Hg and diastolic blood pressure (DBP) > 90 mm Hg) (6,9). The prehypertension classification, defined by the Joint National Committee (Seventh Report) as resting SBP between 120 and 139 mm Hg or a DBP between 80 and 89 mm Hg, encompasses half of the population between 40 and 59 yr old in North America (6). It is now well established that resting

blood pressure (BP) can be transiently lowered (SBP/DBP = 8/9 mm Hg) after acute exercise (one bout of exercise), and this effect, termed “postexercise hypotension” (PEH), may persist for a few hours in individuals with hypertension (13,15). Repeated bouts of acute exercise are an important contributor to chronic adaptations, which may prevent or delay the development of hypertension. Four weeks of chronic aerobic exercise can lower resting BP by 10/6 mm Hg, and the reduction can persist for a period of 7 d (28). However, BP response to the acute and chronic exercise can vary considerably between individuals (SBP = -20 to 9 mm Hg, DBP = -11 to 11.3 mm Hg) (9). In fact, Hagberg et al. (14) identified that as many as 25% of individuals with elevated BP do not seem to elicit BP reduction after endurance training. The relation between BP responses to acute and chronic exercise remains obscure. Examining this relation can be an important step in identifying these “nonresponders” to chronic exercise.

The wide range in BP responses to aerobic exercise may relate to variations in underlying physiological adaptive processes (e.g., neural, hormonal, and local vasodilator substances) (15,26). Sex differences may also influence different cardiovascular regulation mechanisms after exercise (4).

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Currently, the relationship between the magnitude of BP response and mechanisms causing the reduction in BP in both sexes after acute and chronic exercise remains unclear. Furthermore, using acute exercise responses to identify the magnitude of chronic response before undergoing chronic exercise intervention would have important clinical implications. Our primary objective was to assess the relation between the degree of PEH with acute exercise and the magnitude of change in resting BP after chronic exercise. We hypothesized that the magnitude of BP reduction after acute exercise would be predictive of the BP reduction after chronic exercise. Our secondary objective was to examine the correlates of BP reduction after acute and chronic exercise in both sexes.

METHODS

Study population. Seventeen sedentary prehypertensive males ($n = 8$) and females ($n = 9$) age 45 to 60 yr were recruited. All subjects were nonsmokers, were free of cardiovascular disease and diabetes, and were not taking anti-hypertensive medications. All participants were untrained, which was defined by a score of three or less using the Rapid Assessment of Physical Activity survey (37). The female participants recruited were postmenopausal (≥ 2 yr). Written informed consent was obtained, and the Physical Activity Readiness Questionnaire was completed by all subjects (36). This study was approved by the institutional ethics board and conformed to the Helsinki Declaration on the use of human subjects for research.

Protocol. Baseline measures of anthropometrics and maximum oxygen consumption ($\dot{V}O_{2\max}$) were recorded during the first visit (Fig. 1). The second baseline visit (48 h later) assessed BP, baroreflex sensitivity (BRS), HR variability (HRV), cardiac output (CO), stroke volume (SV), and total peripheral resistance (TPR) at rest and in response to an acute exercise assessment protocol ($65\% \dot{V}O_{2\text{peak}}$ for 30 min).

All participants subsequently started an 8-wk walking/jogging program 2 to 3 d after the baseline tests. Study participants exercised four times per week, 30 min per session at

an HR equivalent to $65\% \dot{V}O_{2\text{peak}}$. Three of the four training sessions were performed at the University of Toronto Athletic Center under the supervision of study investigators, whereas the fourth session was unsupervised. All training sessions were recorded in a weekly training log, and all participants were assigned an HR monitor watch (Polar 810i; Polar Electro Oy, Kempele, Finland) to aid adherence to the assigned intensity. Furthermore, participants were asked to avoid any other strenuous physical activity other than the prescribed exercise.

Two to three days after the 8-wk training period, resting BP, anthropometrics, and arterial stiffness were recorded during the first follow-up visit; $\dot{V}O_{2\max}$ was measured 2 d later. Resting BP was also assessed at the end of weeks 1, 3, and 5 to determine the time course of the BP response to training. To avoid the influence of PEH from the acute exercise, the resting BP was taken 48 h after the previous exercise training session. All measures of BP were taken during late afternoon (5–9 p.m.) to minimize diurnal BP variability, and the participants were required to abstain from caffeine for 12 h before measurement.

Resting BP measurements. Participants sat in a quiet room by themselves for 20 min with an automated oscillometric brachial (Tango; SunTech, Morrisville, NC) BP device. BP readings were recorded from the left arm every 2 min throughout the last 10 min. Subjects were seated on a chair with their backs supported and their arms supported at heart level. The highest and lowest of the five readings were disregarded, and an average of the remaining three readings was used to determine resting BP (21). To enhance the reliability of our BP measurement, resting BP was recorded on both of the two baseline and the two follow-up visits.

$\dot{V}O_{2\max}$. $\dot{V}O_{2\max}$ was determined directly from a graded treadmill test using a calibrated metabolic cart (Moxus; AEI Technologies, Naperville, IL). The subjects selected the treadmill speed. Then incline was increased by 2% every 2 min until the eighth minute, after which it was increased by 1% every minute (1). HR was measured using an HR monitor (Polar 810i), and an ECG (Biopack; Kowloon, Hong Kong) tracing was monitored to ensure the safety of the participants.

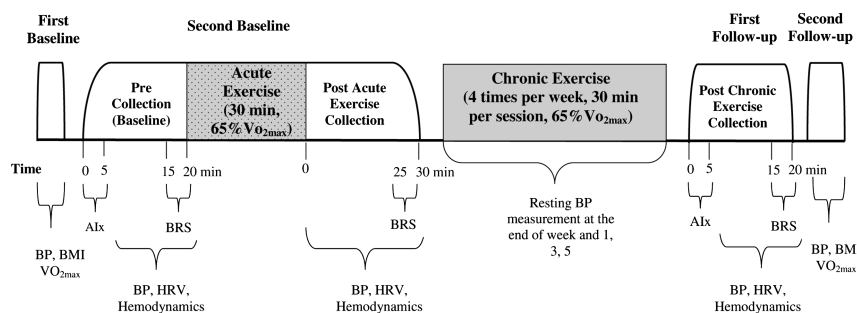


FIGURE 1—Graphical depiction of study timeline. BP, HRV, CO, SV, and TPR were measured throughout the pre- and post-acute exercise as well as post-chronic exercise collection periods. CO, TPR, and SV were collected every minute, whereas HRV and BP measures were collected continuously. Arterial stiffness was collected at the initial 5 min of the pre-acute exercise (baseline) and post-chronic exercise collection periods. BRS was collected for 5 min at the end of all the collection periods. All collections were recorded in a seated position with the subjects' backs supported. The first and the second baseline measurements were separated by 48 h. Similarly, the follow-ups after the chronic exercise were also separated by at least 48 h. The post-acute exercise collection was recorded immediately after the acute exercise.

$\dot{V}O_{2\max}$ was identified by either a plateau of $\dot{V}O_2$ values despite increasing exercise intensity, reaching the age-predicted maximum HR, or achieving an RER of 1.15 or higher.

Acute exercise assessments. Physiological changes in response to acute exercise were assessed with the acute exercise assessment protocol (Fig. 1), which included four stages: arterial stiffness measurement, preexercise collection (20 min), aerobic treadmill exercise (HR equivalent to 65% of $\dot{V}O_{2\max}$), and postexercise collection (30 min). The intensity, duration, and mode of the acute exercise were matched with the training stimulus, and both matched training guidelines (1). BP, hemodynamics, HRV, and BRS were recorded during the pre- and postexercise stages. Participants were required to abstain from caffeine for 12 h and vigorous physical activity 48 h before the acute exercise assessment. All acute exercise assessments were performed during late afternoon (5–9 p.m.) to avoid diurnal BP variation. Subjects were seated in a chair with their backs supported during both preexercise and postexercise collections. Arterial stiffness was assessed from the left radial artery using applanation tonometry (SphygmoCor; AtCor Medical, Sydney, Australia). A transfer function was used to generate a central arterial (aortic) wave form (18) from the average of 10 consecutive arterial wave forms. The augmentation index (AI_x), normalized to an HR of 75 bpm, was calculated as an index of arterial stiffness.

Continuous beat-to-beat BP recordings were obtained from the right hand (third digit) (Finometer MIDI Model-2; Finapres Medical Systems BV, Arnhem, The Netherlands), which was supported at heart level. Brachial BP was also recorded at 2-min intervals using an automated sphygmomanometer (Tango; SunTech) to adjust for any Finometer drift associated with long-duration recordings or fluctuations related to reapplication of the Finometer measurement cuff (18). Corrections were used if a difference greater than 10 mm Hg for SBP or 5 mm Hg for DBP was observed between the Finometer and automated brachial BP recordings. The reliability and reproducibility of AI_x (10) and the automated sphygmomanometer (Tango; SunTech) (3) have been demonstrated. Unpublished data from our laboratory indicate observed mean difference for AI_x and BP (Tango; SunTech) is $0.17\% \pm 4.31\%$ and $1.7 \pm 1.1/0.23 \pm 1.0$ mm Hg (SBP/DBP), respectively. CO and SV were recorded every minute using impedance cardiography (Sorba Medical Systems, Brookfield, WI). TPR was calculated using CO and corresponding BP values. Impedance cardiography has been reported to have a high test–retest reliability coefficient of $r = 0.90$ for assessing CO and SV (2).

HRV was used to assess autonomic nervous system function in accordance with the recommended standards (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (16)). HR was recorded using a three-lead ECG, and the data were sampled at 1000 Hz using LabView (version 7.1; National Instruments; Austin, TX). A 5-min HRV analysis was performed during the 5 min of the lowest BP after acute

exercise. The Kubios HRV software (Biosignal Analysis and Medical Imaging Group, Kuopio, Finland) was used to analyze R-wave to R-wave interval data using a fast Fourier transformation to obtain low-frequency (LF, 0.04–0.15 Hz) and high-frequency (HF, 0.15–0.50 Hz) spectral components. Statistics performed on HRV were transformed using natural logarithm, and the HRV results are reported in raw units.

BRS was evaluated using the sequence method during a 5-min interval (21). A custom program was used to identify three or more successive increases or decreases in BP (Finometer BP) with corresponding increases (++) or decrease (--) in R–R intervals (ECG). The threshold value for the sequence method is set to be 1 mm Hg for beat-to-beat SBP and 6 ms for R–R interval (21), and a lag of zero was selected.

Calculation of PEH. The change in the BP after chronic exercise was expressed as the difference between resting baseline BP and resting BP after week 8. The change in BP after acute exercise was quantified using two methods. First, the greatest magnitude of BP reduction (ΔSBP , ΔDBP) was determined as the difference between preexercise BP and the lowest 5-min rolling average BP in the postexercise period. Second, the area under the curve (AUC) quantifies the difference between preexercise BP and postexercise BP during a period of 30 min. The trapezoidal method was used by dividing the postexercise period into 5-min segments and summing the values as indicated in equation 1.

$$AUC (\text{mm Hg} \cdot \text{min}) = \sum_{n=1} \left(\frac{\Delta BP_n + \Delta BP_{n-1}}{2} \right) (t_n + t_{n-1}) \quad [1]$$

Statistical analysis. Statistical analyses were performed using SPSS version 15.0 (SPSS Inc., Chicago, IL). Data are reported as mean \pm SEM, and statistical significance was assumed at $P < 0.05$. The primary variables of interest were SBP and DBP after acute and chronic exercise. Sample size calculation was made by correlating the magnitude of BP reduction after acute and chronic exercise. The correlation coefficient was set to 0.65 with the α level set to 0.05 and the desired power set to 0.80. Seventeen participants were estimated to be needed. Data were normally distributed as determined using a Shapiro–Wilk normality test. Linear regression analyses were used to assess the relationship between the magnitude of BP reduction after acute and chronic exercise. Correlations were also used to assess the relationship between the change of BP after chronic exercise and the changes of BP cardiovascular regulatory mechanisms after acute exercise. BP responses to acute and chronic exercise were evaluated using two separate repeated-measure ANOVAs (2×2 , intervention \times sex) that examined physiological responses before and after exercise between males and females (See Supplemental Digital Content, <http://links.lww.com/MSS/A165>). A repeated-measure ANOVA was also used to examine the time course of BP

TABLE 1. Subject characteristics at baseline and 8-wk follow-up.

	Males (n = 8)		Females (n = 9)	
	Before Training	After Training	Before Training	After Training
Age (yr)	51 ± 2.4	51 ± 2.4	55 ± 1.3	55 ± 1.3
Height (m)*****	1.77 ± 0.24	1.77 ± 0.24	1.59 ± 0.24	1.59 ± 0.24
Weight (kg)*****	87.5 ± 4.7	86.0 ± 4.5**	71.8 ± 4.8*	72.4 ± 4.3*
BMI***	28.0 ± 1.0	27.4 ± 1.0**	28.6 ± 1.9	28.8 ± 1.9
Waist (cm)*****	99.4 ± 4.6	97.4 ± 4.6	93.5 ± 4.3	92.8 ± 4.3
Hip (cm)***	99.4 ± 3.7	97.3 ± 3.4**	103 ± 3.5	103 ± 3.2
Waist-to-hip ratio*****	1.0 ± 0.32	1.0 ± 0.32	0.90 ± 0.31	0.89 ± 0.30
VO _{2max} (mL·kg ⁻¹ ·min ⁻¹)*****	31.8 ± 1.6	33.7 ± 1.6	23.7 ± 1.5	25.3 ± 1.5
VT (mL·min ⁻¹)*****	1831 ± 171	1987 ± 193	1065 ± 56	1214 ± 57
SBP (mm Hg)****	126 ± 0.94	120 ± 1.8	129 ± 2.5	122 ± 3.2
DBP (mm Hg)****	81 ± 1.1	75 ± 1.1	80 ± 2.5	75 ± 2.0
Aortic SBP (mm Hg)****	118 ± 1.3	110 ± 1.9	118 ± 2.1	110 ± 3.2
Aortic DBP (mm Hg)	82 ± 1.9	78 ± 2.0	82 ± 2.7	76 ± 4.0

BMI, body mass index; VO_{2max}, maximal oxygen consumption; VT, ventilatory threshold; SBP, systolic blood pressure; DBP, diastolic blood pressure. Baseline SBP and DBP displayed in the table are from acute exercise assessment (second baseline visit). BP from baseline 1 (SBP/DBP: males = 128 ± 1.8/82 ± 2.8 mm Hg; females = 130 ± 2.2/80 ± 2.4 mm Hg) was not significantly different from that from baseline 2 ($P > 0.05$). Data are mean ± SEM.

* Significantly different between the sexes ($P < 0.05$).

** Significantly different after training within the sex group ($P < 0.05$).

*** Significant interaction of gender and intervention ($P < 0.05$).

**** Main effect of intervention ($P < 0.05$).

***** Main effect of sex ($P < 0.05$).

change throughout the training at baseline; weeks 1, 3, and 5; and follow-up. Bonferroni *post hoc* comparisons were used.

RESULTS

Subject characteristics. Overall, 54 individuals inquired about the study, and 17 participants (eight males, nine females) were recruited. The balance of individuals was disqualified because they failed to meet the study's inclusion criteria for age ($n = 6$), resting BP ($n = 11$), or medical history ($n = 5$), or they declined because of personal reasons ($n = 15$).

Anthropometric and descriptive results are presented in Table 1. All recruited participants ($n = 17$) completed the study, and their adherence to the training sessions was greater than 80%. This is determined by dividing the number of exercise sessions attended by the total number of exercise sessions. There were no adverse responses to exercise testing or training. The average age of the male and female subjects was 51 ± 2 and 55 ± 1 yr, respectively, $P > 0.05$. Males were larger compared with females (main effects of sex; height: $F_{1,15} = 26.7$, $P < 0.01$; weight: $F_{1,15} = 5.3$, $P < 0.01$; waist: $F_{1,15} = 5.1$, $P < 0.05$; waist-to-hip ratio: $F_{1,15} = 5.6$, $P < 0.01$). Body mass index (BMI) and waist-to-hip ratios classified both sex groups as being overweight (1). Only males reduced weight, BMI, and hip circumference with training; a significant interaction between inter-

vention and sex was found (weight: $F_{1,15} = 6.17$, $P < 0.01$; BMI: $F_{1,15} = 6.17$, $P < 0.01$; hip: $F_{1,15} = 5.27$, $P < 0.01$).

As expected, a main effect for sex in O_{2max} and ventilatory threshold (VT) was observed (O_{2max}: $F_{1,15} = 14.78$, $P < 0.01$; VT: $F_{1,15} = 18.21$, $P < 0.01$), but aerobic power for both sexes was in the 40th percentile relative to their sex and age-specific norms. After training, O_{2max} and VT were significantly improved (main effects of intervention; O_{2max}: $F_{1,15} = 17.2$, $P < 0.01$; VT: $F_{1,15} = 41.06$, $P < 0.001$) because their posttraining O_{2max} rose to the 50th percentile (1).

The average baseline BP for male and female subjects classified them as prehypertensive (6), and no sex difference was found ($P > 0.05$). No significant differences in resting BP measurements were observed between the repeated visits at baseline or at follow-up, $P > 0.05$ (Table 1). PEH was calculated using the baseline BP recorded during the acute exercise assessment (second baseline visit).

BP response to acute and chronic exercise. SBP and DBP were significantly decreased after acute and chronic exercise; no interaction between intervention and sex was found (Table 2). After chronic exercise, resting SBP and DBP were significantly decreased relative to baseline after 5 wk of training, with an earlier trend to reduce resting BP at weeks 1 and 3. Aortic SBP was significantly decreased after chronic training (main effect of intervention on aortic SBP: $F_{1,15} = 9.83$, $P < 0.01$). Significant AUC for ΔSBP ($86.2 ± 28$ mm Hg·min⁻¹) and ΔDBP ($67.4 ± 28$ mm

TABLE 2. Change of BP to baseline after acute and chronic exercise.

	After Acute Exercise	Week 1	Week 3	Week 5	After Chronic Exercise
ΔSBP (mm Hg)	-7.2 ± 1.2*	-1.0 ± 1.6	-2.0 ± 2.3	-4.5 ± 1.7**	-7.0 ± 1.4**
ΔDBP (mm Hg)	-4.2 ± 1.0*	-1.1 ± 1.5	-2.2 ± 1.0	-3.8 ± 1.3**	-5.2 ± 1.2**

Data are mean ± SEM; see Supplemental Digital Content, <http://links.lww.com/MSS/A165> for BP response by sex.

ΔSBP, change of SBP from baseline; ΔDBP, change of DBP from baseline.

* Significantly different from preexercise value ($P < 0.001$). Main effects of intervention after acute exercise; SBP: $F_{1,15} = 36.40$, $P < 0.01$; DBP: $F_{1,15} = 16.72$, $P < 0.01$.

** Significantly different from baseline value ($P < 0.05$). Main effects of intervention after chronic exercise; SBP: $F_{4,15} = 6.08$, $P < 0.01$; DBP: $F_{4,15} = 16.58$, $P < 0.01$.

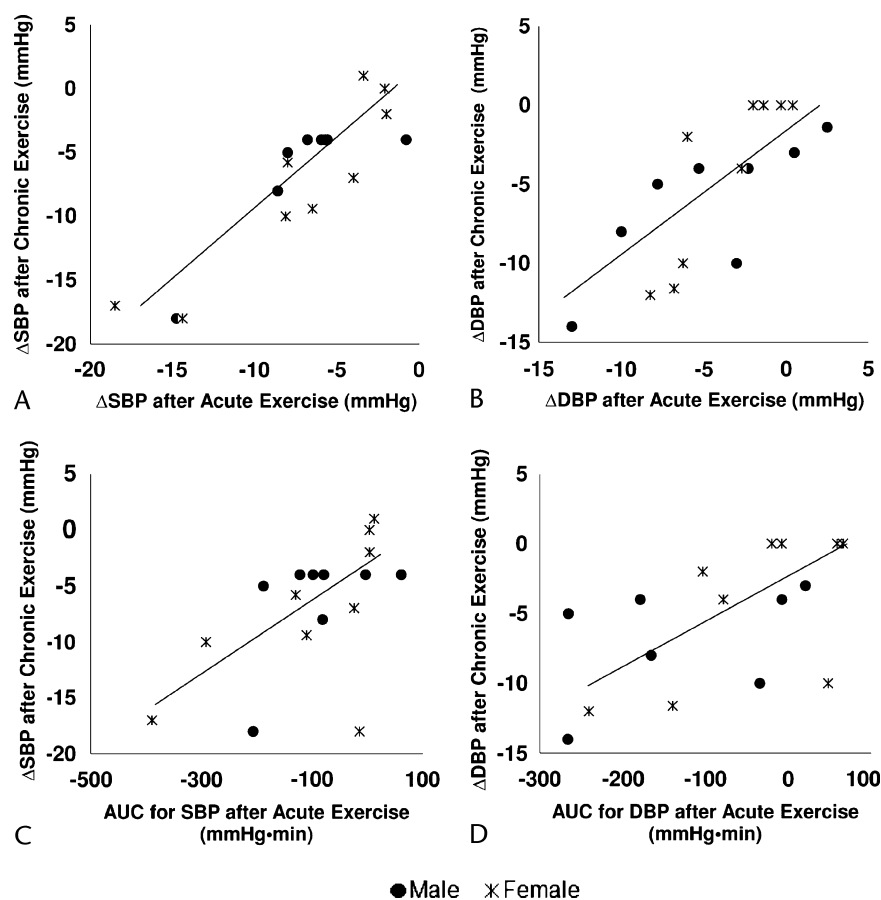


FIGURE 2—A. The magnitude of change (Δ) in SBP after acute exercise was significantly correlated with the magnitude of change (Δ) in resting SBP after chronic training ($r = 0.89, P < 0.01; 1.1 \pm 0.14 (B \pm SE), P < 0.01; \text{adjusted } r^2 = 0.78, P < 0.001$). Males: $r = 0.83, P < 0.01; 0.53 \pm 0.24 (B \pm SE), P < 0.001; \text{adjusted } r^2 = 0.82 (P < 0.01)$. Females: $r = 0.90, P < 0.01; 0.82 \pm 0.15 (B \pm SE), P < 0.001; \text{adjusted } r^2 = 0.78 (P < 0.001)$. B. Significant correlation between Δ DBP after acute exercise and resting Δ DBP after chronic training ($r = 0.75, P < 0.01; 0.722 \pm 0.19 (B \pm SE), P < 0.01; \text{adjusted } r^2 = 0.58, P < 0.001$). Males: $r = 0.77, P < 0.01; 0.75 \pm 0.29 (B \pm SE), P = 0.09$. Females: $r = 0.87, P < 0.01; 0.85 \pm 0.266 (B \pm SE), P < 0.01; \text{adjusted } r^2 = 0.79, P < 0.01$. C. The AUC for SBP after acute exercise was significantly correlated with Δ SBP after chronic training ($r = 0.60, P < 0.01; 0.60 \pm 0.10 (B \pm SE), P < 0.01; \text{adjusted } r^2 = 0.32, P < 0.01$). Males: $r = 0.57, P > 0.05$. Females: $r = 0.60, P > 0.05$. D. The AUC for DBP after acute exercise was significantly correlated with Δ DBP after chronic training ($r = 0.59, P < 0.01; 0.60 \pm 0.11 (B \pm SE), P < 0.001; \text{adjusted } r^2 = 0.31, P < 0.05$). Males: $r = 0.61, P > 0.05$. Females: $r = 0.62, P > 0.05$. The linear regressions are adjusted for baseline BMI.

Hg \cdot min $^{-1}$) were observed after acute exercise, $P < 0.05$; no effect of sex was found. The greatest reduction in Δ SBP after

acute exercise was positively correlated with AUC for Δ SBP ($r = 0.69, P < 0.05$) and Δ DBP ($r = 0.51, P < 0.05$) decrease

TABLE 3. Antihypertensive mechanisms after acute exercise.

	Males		Females	
	Baseline	After Acute Exercise	Baseline	After Acute Exercise
TPR (dyn \cdot s \cdot cm $^{-5}$)	2091 \pm 448	1823 \pm 350	2520 \pm 323	2135 \pm 265
CO (L \cdot min $^{-1}$)	4.5 \pm 0.70	5.1 \pm 0.83	3.7 \pm 0.73	4.0 \pm 0.75
HR (bpm)*	78 \pm 3.7	87 \pm 3.3	75 \pm 3.5	84 \pm 4.9
SV (mL)	57 \pm 11	52 \pm 11	50 \pm 5.8	47 \pm 6.4
MeanRR	778 \pm 35	706 \pm 17	790 \pm 35	713 \pm 33
STDRR*	32.5 \pm 4.4	26 \pm 2.7	26.7 \pm 4.4	20 \pm 3.4
LF (ms 2)*,**	587 \pm 186	265 \pm 60	227 \pm 186	80 \pm 14
LF (nu)	81.2 \pm 7.3	87 \pm 3.1	68.9 \pm 7.3	72 \pm 9.9
HF (ms 2)*	86.9 \pm 46	40 \pm 15	115.5 \pm 46	72 \pm 40
HF (nu)	18.8 \pm 7.3	12 \pm 3.1	31.1 \pm 7.3	28 \pm 9.9
TP (ms 2)*	1141 \pm 285	595 \pm 133	919 \pm 285	372 \pm 102
LF/HF**	10.3 \pm 2.1	10.6 \pm 2.1	3.4 \pm 2.1	6.5 \pm 2.0
BRS (–) (ms \cdot mm $^{-1}$ Hg)*	5.8 \pm 1.0	4.2 \pm 0.34	4.7 \pm 1.0	3.5 \pm 0.50
BRS (++) (ms \cdot mm $^{-1}$ Hg)*,**	6.3 \pm 0.57	4.1 \pm 0.23	3.5 \pm 0.57	2.3 \pm 0.32

Data are mean \pm SEM.

* Main effect of intervention ($P < 0.05$).

** Main effect of sex ($P < 0.05$).

TPR indicates total peripheral resistance; CO, cardiac output; HR, heart rate; SV, stroke volume; MeanRR, mean R–R interval; STDRR, standard deviation of the RR interval; LF, low-frequency power; LF (nu), low frequency (normalized units); HF, high-frequency power; HF (nu), high frequency (normalized units); BRS (–), baroreceptor sensitivity downward slope; BRS (++) , baroreceptor sensitivity upward slope.

TABLE 4. Antihypertensive mechanisms after chronic exercise.

	Males		Females	
	Baseline	After Chronic Exercise	Baseline	After Chronic Exercise
TPR (dyn·s·cm ⁻⁵)	2091 ± 448	1822 ± 675	2520 ± 323	2262 ± 462
CO (L·min ⁻¹)	4.5 ± 0.70	4.6 ± 0.70	3.7 ± 0.73	3.9 ± 0.53
HR (bpm)	78 ± 3.7	74 ± 1.8	75 ± 3.5	72 ± 2.8
SV (mL)	57 ± 11	62 ± 10	50 ± 5.8	51 ± 6.5
MeanRR	778 ± 35	810 ± 30	790 ± 35	796 ± 30
STDRR	32.5 ± 4.4	35.8 ± 4.0	26.7 ± 4.4	27.0 ± 4.0
LF (ms ²)****	587 ± 186	480.4 ± 89	227 ± 186	180 ± 89.4
LF (nu)	81.2 ± 7.3	80.1 ± 4.3	68.9 ± 7.3	66.4 ± 4.2
HF (ms ²)****	86.9 ± 46	176.6 ± 81	115.5 ± 46	114.5 ± 81
HF (nu)	18.8 ± 7.3	20.0 ± 4.2	31.1 ± 7.3	33.6 ± 4.2
TP (ms ²)	1141 ± 285	1342 ± 341	919 ± 285	659 ± 341
LF/HF***	10.3 ± 2.1	6.6 ± 1.3*	3.4 ± 2.1**	2.3 ± 1.3**
BRS (--) (ms·mm ⁻¹ Hg)	5.8 ± 1.0	5.8 ± 0.65	4.7 ± 1.0	4.4 ± 0.65
BRS (++) (ms·mm ⁻¹ Hg)	6.3 ± 0.57	6.0 ± 0.59	3.5 ± 0.57	3.7 ± 0.59
AI _x (%)****	14.6 ± 2.4	14.4 ± 2.0	31.6 ± 2.6	25.6 ± 2.1
AI _{x@75} (%)***	16 ± 2.1	14.8 ± 2.8	30.7 ± 2.2**	24.3 ± 3.0***

Data are mean ± SEM.

* Significantly different from baseline ($P < 0.05$).

** Significantly different between the sexes ($P < 0.05$).

*** Significant interaction of sex and intervention ($P < 0.05$).

**** Main effect of sex ($P < 0.05$).

AI_{x@75}, AI_x normalized to 75 heartbeats per minute.

after acute exercise. The magnitude of Δ SBP reduction after chronic exercise was significantly correlated with baseline BMI ($r = 0.52$, $P < 0.05$). Baseline SBP was not correlated with the magnitude of Δ SBP decrease after chronic exercise.

The reduction in SBP after acute exercise was strongly correlated with the chronic reduction in resting SBP after training ($r = 0.89$, $P < 0.01$), as shown in Figure 2. A similar correlation was also observed for Δ DBP ($r = 0.75$, $P < 0.01$; Fig. 2). AUCs for Δ SBP and Δ DBP were significantly correlated with reduction in resting Δ SBP ($r = 0.60$, $P < 0.01$) and Δ DBP ($r = 0.59$, $P < 0.05$), respectively, after training (Fig. 2).

Cardiovascular regulation and BP reduction after exercise. After acute exercise, SD of the R–R interval (STDRR), LF, HF, total power (TP), BRS (--), and BRS (++) were reduced; a main effect of intervention was present (STDRR: $F_{1,15} = 8.04$, $P < 0.01$; LF: $F_{1,15} = 5.68$, $P < 0.05$; HF: $F_{1,15} = 5.68$, $P < 0.05$; TP: $F_{1,15} = 14.58$, $P < 0.01$; BRS (--): $F_{1,15} = 5.70$, $P < 0.05$; BRS (++): $F_{1,15} = 5.62$, $P < 0.05$) (Table 3). A main effect of sex was observed for BRS (++) ($F_{1,15} = 5.07$, $P < 0.05$), LF ($F_{1,15} = 6.92$, $P < 0.05$), and LF-to-HF ratio (LF/HF: $F_{1,15} = 6.28$, $P < 0.05$). Females showed a lower BRS (++) , LF, and LF/HF than males. HR was elevated after acute exercise (main effect of intervention: $F_{1,15} = 22.54$, $P < 0.05$).

After chronic exercise, an intervention \times sex interaction was observed for arterial stiffness and LF/HF (LF/HF: $F_{1,15} = 4.78$, $P < 0.05$; AI_{x@75}: $F_{1,15} = 4.70$, $P < 0.05$) (Table 4). Males had a higher LF/HF but a lower arterial stiffness measurement (AI_{x@75}) than females at baseline. The LF/HF ratio significantly decreased only in the males; meanwhile, AI_{x@75} significantly decreased only in the females. A main effect of sex was observed for AI_x ($F_{1,15} = 6.30$,

$P < 0.05$), LF ($F_{1,15} = 6.92$, $P < 0.05$), and HF ($F_{1,15} = 6.92$, $P < 0.05$); males had a higher LF and lower HF than females. Changes in hemodynamics, HRV, and BRS after acute exercise did not significantly correlate with changes in BP after chronic exercise.

DISCUSSION

To our knowledge, this is the first study to quantify the relation between the decrease in BP after acute exercise and the resting BP reduction associated with chronic exercise. We observed that the magnitude of SBP reduction and AUC (SBP) after acute exercise was associated with the magnitude of resting SBP reduction after 8 wk of aerobic training. A similar relationship was observed for DBP. These observations have important clinical implications in the management of prehypertension and hypertension because the magnitude of the acute BP-lowering-effect exercise may predict the extent of BP lowering after chronic training interventions. Those individuals not responsive to this form of exercise may be prescribed alternative exercise or treatments such as diet or pharmacological interventions.

The significant reduction in Δ SBP (-7 mm Hg) and Δ DBP (-4 mm Hg) after acute exercise was comparable to previous studies that used similar doses of exercise prescription (26,32). The determination of PEH has yet to be standardized, and methods differ considerably between studies. Most studies report the greatest decrease over a certain time interval (13,26,32). However, these approaches are limited to arbitrary time points selected for comparison, potentially biasing results. For comparison purposes, we not only use this method but also report a novel and robust approach by calculating AUC to quantify PEH. Peak BP decrease was

significantly correlated with AUC for both SBP and DBP, which suggests that individuals with a greater peak decrease experienced a larger total effect.

Chronic exercise training elicited a significant reduction in resting BP (Δ SBP = -7.0 ± 1.4 mm Hg/ Δ DBP = -5.2 ± 1.2 mm Hg), typical of values reported for this age group (45–60 yr old) (40). The time course of BP change has varied among previous studies with reductions detected as early as 1 wk into the exercise program, whereas others reported significant BP reduction after 4 (7) to 6 (9,39) wk of chronic exercise. In our study, a trend to BP reduction occurred relatively early in the training period (week 3), with further significant reductions occurring at week 5 (Table 2). This is in contrast with the finding of Meredith et al. (28), which failed to observe further reductions in BP after the third week of aerobic training in healthy young normotensive individuals. It is possible that the subjects' age contributed to these differences secondary to different adaptive responses to training. Nevertheless, the time course of the resting blood change allowed us to verify that the greatest BP reduction was achieved at the end of the 8 wk of training.

Exercise intensity (50%, 70%, and 85% $\dot{V}O_{2max}$) and duration (30 and 45 min) seem to be weak predictors of BP reduction after acute exercise (26, 33). In our laboratory, we have observed a similar magnitude of PEH using moderate-intensity aerobic exercise (65% $\dot{V}O_{2max}$) and high-intensity interval exercise (five sets of 2:2 min at 85% and 40% $\dot{V}O_{2max}$) (21). Other exercise modalities such as the isometric handgrip have reported similar PEH magnitudes, independent of contraction and/or rest period frequency–duration relationships (29). However, unlike acute exercise, the BP response after chronic exercise has shown an exercise dose and BP response relationship. Jennings et al. (20) and Whelton et al. (39) reported that higher exercise frequencies per week (more than five times per week) and a longer exercise duration per session (>60 min) did not produce a greater reduction in BP in hypertensive patients compared with fewer bouts (three to four times per week) and shorter exercise durations (30–60 min). The relation of chronic exercise intensity to BP reductions is unclear, with some authors observing more change with an intensity greater compared with less than 70% $\dot{V}O_{2max}$ (14), whereas another sees no intensity effect (26). However, a higher intensity exercise (60%–75% $\dot{V}O_{2max}$) is required to have a greater gain in $\dot{V}O_{2max}$ (12), which is associated with a decreased risk of cardiovascular disease (27). Therefore, the exercise duration (30 min) and intensity (65% $\dot{V}O_{2max}$) prescribed in this study were expected to yield the greatest BP reduction as well as improvement in aerobic power.

Despite the potential benefits of BP lowering from acute and chronic exercise intervention in individuals with hypertension, considerable variability in responses exists (9,40). The SBP and DBP decrease after acute exercise ranged from -19 to 0 and -13 to 3 mm Hg, respectively. Similarly, after chronic exercise, the magnitude of change

in resting SBP and DBP varied from -14 to 0 and -17 to 7 mm Hg, respectively. However, the regression analysis revealed that the magnitude of the acute PEH was strongly related to the chronic reductions in BP secondary to exercise training. A 5-mm Hg reduction is clinically significant because it is associated with risk reduction for stroke and heart disease by 15%–20% (24). Therefore, we considered those individuals who had less than 5 mm Hg to have “minimal” response to exercise training. We observed five individuals who exhibited a minimal resting BP reduction of less than 5 mm Hg of SBP. Four of those five individuals also experienced a reduction of less than 5 mm Hg of SBP after chronic exercise. Therefore, a smaller magnitude of BP reduction after acute exercise may be an indicator to identify those individuals who are less responsive to chronic exercise training.

Previous studies have observed that a higher baseline SBP is also a predictor of SBP after exercise (32). However, this phenomenon is inconsistently observed (21). Using baseline BP as a predictor has been criticized because the predictor variable of baseline BP is mathematically related to the outcome variable (changes of BP) (35). The correlation between BP change (AUC) after acute and chronic exercise and measurement of chronic BP changes on separate occasions are alternative approaches.

The mechanisms contributing to the antihypertensive effects of acute and chronic exercise remain unclear, and BP changes are likely secondary to numerous factors. The antihypertensive mechanisms also differ with age, gender, and training status (4,13,15). Thus, these differences may have contributed to the lack of correlation between acute mechanisms and chronic BP reduction. The specific mechanisms that link BP reduction after acute and chronic exercise are unclear. From our data, a combination of vascular (TPR and AI_x) and neural (HRV and BRS) changes may be at play. Our data did not identify a common mechanism for the acute and chronic responses. This may suggest a central control mechanism that our measures do not address is operative.

Changes in hemodynamic response (CO and TPR) differ among study populations after acute exercise. The PEH in older hypertensive individuals (60–69 yr) was associated with a decrease in CO without changes in TPR (13). Conversely, PEH was observed in middle-age normotensive and prehypertensive (45–65 yr old) individuals achieved through a reduction in TPR with only modest increases in CO (26). It seems that often, the chronic exercise-associated BP reduction is mainly due to decreased TPR that is not met by an increase in CO (9). We observed a trend in reduced TPR after acute (13%) and chronic (11%) exercise; however, these reductions failed to reach significance because of the between-person variability of responses.

Arterial BP can also be modulated by changes in the distensibility of the artery and the timing of the reflected arterial pulse wave (10,34). Previous studies using arterial pulse wave analysis showed that arterial stiffness is decreased after chronic exercise (7,10). However, measurement

of arterial stiffness using AI_x or $AI_x@75$ after long-term training in prehypertensive males and females remains limited. Studies have reported significant reductions in AI_x after 6 and 12 months of aerobic training in the elderly (67 ± 6 yr) (34) and in patients with chronic kidney disease (30), respectively. We observed that arterial stiffness had a greater decrease in the female cohort (-22% relative to baseline) when compared with the males (-7.5% relative to baseline) after chronic exercise. This may be attributed to the higher arterial stiffness recorded at baseline in the postmenopausal females. Tabara et al. (34) reported that chronic exercise-induced changes in AI_x were significantly and inversely correlated with the preexercise AI_x ($r = 0.48$, $P < 0.01$). Postmenopausal females are known to have greater arterial stiffness values than age-matched males (38). This may be related to hormonal status because estrogen therapy can significantly reduce arterial stiffness in postmenopausal females (38).

The change in TPR and arterial stiffness are likely the result of both vasodilators and autonomic activity (15,26). The vasodilation may occur in response to increases in exercise-mediated vasodilator substances (e.g., nitric oxide, adenosine, prostaglandins) and an attenuated vascular response to sympathetic vasoconstriction. The neural component is the reduction of sympathetic vasoconstrictor nerve activity in the skeletal vascular bed, which has been demonstrated in humans (15). Training-associated reductions in sympathetic activity may lower vascular tone in skeletal muscles.

In our HRV analysis, the reduction in TP (ms^2) and LF (ms^2) in both sexes suggests a rebalancing of the sympathovagal modulation to the heart (16). Several other studies have reported similar findings after acute exercise (15,26,33). Interestingly, after chronic exercise, only males showed a significant shift toward more vagal dominance as indicated by HRV ($\sim 36\%$ decrease of LF/HF relative to baseline). This may reflect the higher LF/HF values for males at baseline. The greater decrease in LF/HF after chronic exercise in the males was not associated with greater TPR changes or arterial stiffness reductions in the males. On the basis of evidence from chronic training studies, improvements in endothelial function and arterial remodeling modulate TPR and arterial stiffness (10). Favorable vascular remodeling of elastin and collagen content can take place with exercise training to decrease arterial stiffness. In addition, increased lumen vessel diameter as a result of chronic training can also decrease TPR (15,26).

After acute exercise, BRS can be decreased for up to 60 min (31), and we found similar results. This change in BRS may reflect resetting the baroreflex to a lower pressure that leads to reduced sympathetic outflow from the CNS (15,26); however, the spontaneous BRS is an indirect measure of reflex gain. Respiratory pattern changes can influence BRS (11); however, few changes in resting respiratory patterns with training are observed (19). Nevertheless, baroreceptors play a vital role in PEH because Chandler

and DiCarlo (5) reported that PEH was only observed in rats with intact baroreceptors. BRS can be influenced by gender and age; postmenopausal females have been reported to have a blunted BRS compared with age-matched men (22), which is consistent with our findings. Some studies report increases in BRS (17,23), whereas we and others (8,25) found that BRS did not increase after chronic training in both sexes. Change in BRS may not be required for BP reduction after chronic exercise but may be operative in the PEH response.

This study benefited from a longitudinal study design, and the exercise training prescribed was closely supervised. The HR monitors issued to all participants allowed them to exercise at the prescribed intensity. We made an effort to ensure the accuracy of our BP values by recording BP at the same time of day and measuring resting BP on two separate days at baseline and follow-up. A limitation of the study is that a nonexercise control group was not included. Nevertheless, studies containing a control group have shown that the reduction of BP after acute and chronic exercises resulted from the exercise intervention (9,26). The magnitude of PEH response may be altered during the data reduction process. Diet was not controlled in this study, but participants were asked not to change their eating habits throughout the study. Diet can play a role in the reduction of BP (6); however, this study has shown that even without diet intervention, exercise alone can reduce BP significantly. Finally, the correlations between the decrease in BP after acute and chronic exercise may be restricted to an exercise program that is similar to the intensity and duration prescribed in this study. The ability to use cardiovascular variability measures as indirect indicators of autonomic function remains contentious.

CONCLUSIONS AND PERSPECTIVES

We conclude that the degree of BP reduction after acute exercise is related to the magnitude of change in BP observed after chronic exercise. Similar PEH responses in males and females were achieved using common mechanisms, yet sex differences were observed in how chronic reductions in BP were achieved. In addition, these results suggest that individual responsiveness to the BP-lowering effect of chronic exercise can be identified. Further studies are required to elucidate the mechanisms contributing to PEH and those leading to the chronic BP-lowering effect seen after exercise training.

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