# Correlation between Acute and Chronic 24-Hour Blood Pressure Response to Resistance Training in Adult Women

DOI 10.1055/s-0034-1382017
Int J Sports Med 2015; 36: 82–89



**Publisher and Copyright**
© 2015 by
Georg Thieme Verlag KG
Rüdigerstraße 14
70469 Stuttgart
ISSN 0172-4622



Thieme

# Correlation between Acute and Chronic 24-Hour Blood Pressure Response to Resistance Training in Adult Women

Authors
R. A. Tibana[1], N. M. F. de Sousa[2], D. da Cunha Nascimento[1], G. B. Pereira[3], S. G. Thomas[4], S. Balsamo[5], H. G. Simoes[5], J. Prestes[5]

Affiliations
Affiliation addresses are listed at the end of the article

Key words
- cardiovascular response
- exercise
- blood pressure
- resistance exercise
- hypotension

## Abstract

The purpose of the present study was to correlate the acute and chronic decrease in blood pressure (BP) following resistance training (RT). 13 normotensive women (18–49 years) completed an acute whole body RT session with 3 sets of 10 repetitions at 60% 1RM and then 8 weeks of RT as follows: 3/week, 3 sets of 8–12 repetitions maximum. Systolic (SBP) and diastolic BP (DBP) were measured up to 60 min and 24 h following RT (acute and chronic). The greatest acute decrease of SBP (108.5 ± 7.0 mmHg) and DBP (71.5 ± 6.4 mmHg) values over the 60-min period were reduced compared to pre-exercise (117.3 ± 11.7 and 79.3 ± 8.2 mmHg, respectively; $p < 0.05$). The chronic effect on resting BP was observed only for those presenting acute post-exercise hypotension (PEH). The change in both SBP and DBP following acute RT was correlated with the chronic change in resting SBP and DBP ($r > 0.5$; $p \leq 0.05$). The change in 24 h BP after acute RT was correlated with the chronic reduction in SBP ($r = 0.74$) and DBP ($r = 0.80$). The magnitude of PEH is a promising candidate for the prediction of individual BP-related training efficacy.

## Introduction

Systemic arterial hypertension (SAH) is defined as a chronic medical condition with a prevalence of one billion hypertensive individuals worldwide, being responsible for approximately 7.6 million deaths per year [10]. Moreover, alarming data revealed a 22% prevalence of hypertension in a Brazilian population (individuals aged ≥ 18 years) [28]. Hypertension predisposes persons to increased risk of cardiovasular disease, including myocardial infarction, heart failure, stroke and kidney disease [5]. The treatment of SAH involves pharmacological and non-pharmacological theraphies [23]. Among non-pharmacological methods, physical training has been indicated as one of the most important interventions for preventing or controlling high blood pressure (BP) [21, 33].

Aerobic training has been the focus of substantial studies and has been recommended as an adjunct treatment for SAH [24]. However, there is evidence to support that resistance training (RT) may elicit both acute and chronic BP reduction [7, 8, 25]. In this sense, our group's studies revealed that acute and chronic RT was effective in decreasing systolic (SBP), diastolic (DBP) and mean BP (MBP) over 24 h and at night in overweight/obese middle-aged women and in women with metabolic syndrome [32, 33].

On the other hand, even within homogeneous groups, the magnitude of training effects (referring to performance and to health-related factors) varies widely and includes low and/or even nonresponders [18, 20, 29, 31]. Recently, Hecksteden et al. [18] reported that the magnitude of postexercise hypotension (PEH) following acute, maximal exercise is associated with the training-induced decrease of BP following a walking/running program (45 min, 4 times per week at 60% heart rate reserve) over 4 weeks in healthy untrained subjects aged 30–60 years. Previously, Liu et al. [20] found that the magnitude of the acute BP-lowering with submaximal exercise may predict the extent of resting BP lowering following an 8-week walking/jogging training program (4 times per week, 30 min per session, 65% maximum oxygen consumption) in prehypertensive individuals. Taken together, these results suggest that the acute BP-lowering effect can be predictive of chronic exercise-induced BP change. However, the aforementioned studies were performed with aerobic training, while the analysis of BP was conducted during a limited period after exercise.

accepted after revision
April 23, 2014

Bibliography
DOI http://dx.doi.org/10.1055/s-0034-1382017
Published online: August 21, 2014
Int J Sports Med 2015; 36: 82–89 © Georg Thieme Verlag KG Stuttgart · New York
ISSN 0172-4622

Correspondence
***Dr. Jonato Prestes***
Graduation Program on Physical Education
Catholic University of Brasilia
Q.S. 07, Lote 01 – Bloco G
71966-700 Brasilia
Brazil
Tel.: +55/21/55 61 3356 9350
Fax: +55/21/55 61 3356 9350
jonatop@gmail.com

It should be noted that monitoring BP over 24 h enables the recording of the circadian rhythms under daily living conditions, with most individuals displaying diurnal and nocturnal variations in SBP and DBP [30]. Strong evidence suggests a better correlation of 24 h BP measurements with overall target organ damage score, left ventricular mass, impaired left ventricular function, albuminuria, brain damage, microvascular disease and retinopathy [12]. Nevethless, to the best of our knowledge no previous study has been designed to correlate the acute effects of RT on 24 h BP with the magnitude of change in resting 24 h BP following chronic training.

We studied adult women because it has been shown that the residual lifetime risk for developing hypertension is 90 %, suggesting that this would be a very important period of life for prevention [34]. Moreover, according to Floras [11] women are clearly underrepresented in the present literature for this area. Therefore, the aim of the present study was to evaluate the acute and chronic effects of RT on BP in adult women and assess the correlation between the degree of PEH following acute exercise and the magnitude of change in resting BP after chronic RT. The initial hypothesis of the study *is* that the magnitude of BP reduction after an acute RT session *is* predictive of the BP reduction after chronic training.

## Material and Methods

### Subjects

19 untrained females aged 18–49 years were recruited by convenience through advertisements and phone calls. Subjects with physical disabilities, diagnosis of diabetes, cardiovascular diseases, hypertension (systolic blood pressure > 140 mmHg and diastolic blood pressure > 90 mmHg), musculoskeletal disease, recent use of any medication and smoking or drug/alcohol abuse were excluded from the trial. 6 women were excluded because of hypertension and/or use of anti-hypertensive medication. All study participants responded to the short-version of the International Physical Activity Questionnaire (IPAQ), were considered sedentary and were untrained according to American College of Sports Medicine [19], as they had no previous experience with RT.

All participants signed an informed consent document, and the study was approved by the Catholic University of Brasilia Research Ethics Committee for Human Use (protocol #376/2010) and conformed to the Helsinki Declaration on the use of human subjects for research. Our study meets the ethical standards of the journal [16].

### Experimental design

Subjects completed 2 weeks of familiarization prior to testing. During the familiarization weeks, individuals were advised regarding the execution of proper technique, and completed 3 sessions per week, with 1 exercise for each main muscle group (same exercises of the RT) performing 3 sets of 10–12 submaximal repetitions at 60 % of estimated 10 repetitions maximum (10RM). After the familiarization period, subjects completed a 1-repetition maximum test (1RM) to determine the maximal strength in each exercise. 3 days later participants performed an acute RT session. In the exercise session, participants remained seated quietly for 15 min before completing 3 sets of 10 repetitions of the following exercises: machine leg press, leg extension, leg curl, chest press, front lat pull-down and machine shoulder press at 60 % 1RM followed by 15 repetitions of abdominal crunches. A rest interval of 1-min was allowed between sets and exercises (the total duration of the session was 30 min). Subjects were instructed to perform each repetition at a moderate speed (i.e., 2 s concentric and 2 s eccentric) to avoid Valsalva maneuver and were supervised by a researcher experienced in physical training. All subjects were encouraged to avoid smoking, alcohol and caffeine consumption as well as unusual physical activity before each trial. Individuals from the present study completed the RT session between 8:00 and 9:00 p.m., and we observed an immediate postexercise hypotension response characterized by a decrease in SBP, DBP and MBP. Subjects were also instructed to go to bed at 11:00 p.m. and awake at 6:00 a.m. on experimental days. The phase of the menstrual cycle was not controlled [17], even though there is conflicting evidence showing that the menstrual cycle may affect BP [22].

### Maximal strength testing

After 2 weeks of familiarization with the exercises, 1RM tests were performed on 4 different days separated by a minimum of 72 h. All tests were performed with 10-min rest intervals between each exercise. The order of the exercises was as follows: chest press, front lat pull-down and machine shoulder press (days 1 and 2; test and re-test); machine leg press, leg extension and leg curl (days 3 and 4; test and re-test). The protocol consisted of a light warm-up of 10 min of treadmill running followed by 8 repetitions at 50 % of estimated 1RM (according to the participants' capacity verified in the adaptation weeks). After a 1-min rest, subjects performed 3 repetitions at 70 % of the estimated 1RM. Following 3 min of rest, participants completed 3–5 1RM attempts interspersed with 3–5-min rest intervals, with progressively heavier weights (5 %) until the 1RM was determined. The range of motion and exercise technique were standardized according to the descriptions of Brown and Weir [2]. The intraclass correlation for the tests was: chest press, $r = 0.97$; front lat pull-down, $r = 0.99$; machine shoulder press, $r = 0.98$; machine leg press, $r = 0.97$; leg extension, $r = 0.98$; and leg curl, $r = 0.99$.

### Resistance training

All participants subsequently started a RT program lasting 8 weeks with 3 sessions/week. Training machines were from JOHNSON (Landmark Drive, Cottage Grove, USA). All training sessions were carefully supervised by 3 experienced professionals (ratio of supervision 1:2–1 supervisor for 2 participants). Participants were required to complete at least 85 % of the exercise sessions. **Fig. 1** shows the exercise order that was strictly followed. The RT was divided into A (Monday) and B (Tuesday) and whole body (Friday) regiments. Abdominal crunches (3 sets of 15 repetitions) were included. For all listed exercises, 3 sets with 8–12 RM were performed, with 1-min rest interval between each set and exercise. Training loads were monitored in each session according to the increase in muscle capacity of the participants. The mean duration to complete one repetition was 3–4 s (both concentric and eccentric phases of the movement), and training sessions lasted ≈ 40–45 min. The loads were updated when necessary to keep the number of repetitions within the same range of RM and to provide a progressive overload. Additionally, correct breathing patterns were instructed to prevent Valsalva maneuver.

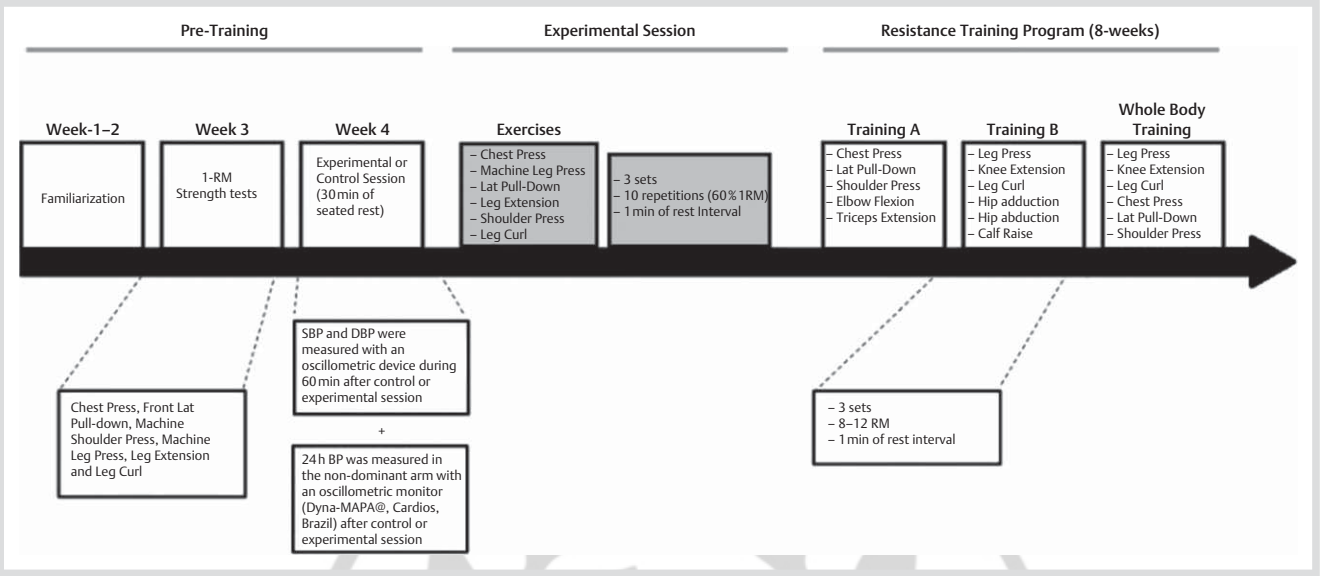


**Fig. 1** Graphical description of study timeline. Clinical and ambulatory BP were measured throughout the pre- and post- acute exercise or control sessions as well as post-chronic (8 weeks) exercise collection periods. The control and the experimental session measurements were separated by 48 h. BP = Blood Pressure; DBP = Diastolic Blood Pressure; RM = Repetitions Maximum; SBP = Systolic Blood Pressure.

### Blood pressure measurement

Systolic BP and DBP were measured with an oscillometric device (Microlife 3AC1-1, Widnau, Switzerland) according to the recommendations of the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure [5]. The cuff size was adapted to the circumference of the arm of each participant according to the manufacture's instructions. All BP measures were assessed in triplicate (measurements separated by 1 min) with the mean value used for analysis. Blood pressure measurements were performed after 15 min of seated rest (0); and 10 min; 30 min and 60 min after the exercise session. During BP measurements participants remained seated quietly under a controlled room temperature.

### Twenty-four hour blood pressure

24 h BP was measured in the non-dominant arm with an oscillometric monitor (Dyna-MAPA®, Cardios, Brazil), validated by the European Society of Hypertension. The monitor was programmed to take measurements every 15 min during the daytime (7:00 a.m.–10:00 p.m.) and every 30 min during the nighttime (11:00 p.m.–6:00 a.m.). All 24 h measurements of BP were taken during weekdays (i. e., Monday–Friday) and were initiated between 9:00 and 10:00 p.m. All participants were advised to maintain their normal activities and diet (this was guaranteed by a dietary recall follow-up), refrain from programmed exercise, avoid smoking, alcohol and caffeine consumption, and to stop and relax the arm during each measurement. Data from 24 h monitoring were calculated and analyzed as follows: mean of all measurements during the 24 h period; mean of all measures performed during the daytime and mean of all measures performed during the nighttime as proposed by Tibana et al. [32]. The percent decline in nocturnal BP was calculated as follows for SBP: the percent decline in nocturnal BP (%) = (Daytime BP − Nighttime BP) × 100/Daytime BP. Subjects were assigned into groups based on the percent decline in their nocturnal BP as follows: inverted dippers: no nocturnal decline in SBP; nondippers: percent decline in nocturnal BP ≥ 0% and ≤ 10% for SBP; dippers: > 10% and < 20% for SBP; extreme dippers: percent decline in nocturnal BP ≥ 20% for SBP. These cut-off points were based on the results of the previous studies that investigated the relationship between the nocturnal decline in BP and cardiovascular complications [11].

### Statistical analysis

The results are expressed as means ± standard deviation (SD). Data were normally distributed as determined using a Shapiro-Wilk test. A paired t test was applied to compare baseline blood pressure values with post exercise values and resting values after chronic exercise. ANOVA with repeated measures was used to compare the area under the curve (AUC) of 24 h, daytime and nighttime blood pressure between baseline values, after acute exercise and after chronic training. Tukey's post-hoc test was applied in the event of significance. Cohen's d and eta-squared ($\eta^2$) effect sizes were calculated using Cohen's [6] convention. Pearson's product moment correlations and chi-square for associations were used to explore the relationship between the magnitude of blood pressure reduction and the percent decline in nocturnal BP after acute and chronic exercise. The power of the sample size was determined using G*Power version 3.1.3 (Erdfelder, Faul, & Buchner, 1996; Kiel, Germany), based on the correlation between the magnitudes of change in chronic and acute exercise for SBP and DBP. Considering the sample size of this study and an alpha error of 0.05, the greatest decrease (greater individual decrease of clinical BP reached after exercise) power ($1 - \beta$) achieved in this research was 0.72 for DBP and 0.82 for SBP. The level of significance was $p \leq 0.05$, and SPSS version 20.0 (Somers, NY, USA) software was used.

## Results

All recruited participants (n = 13) completed the study, and their adherence to the training sessions was greater than 85%. The anthropometric characteristics of the subjects are presented in **Table 1**. The average baseline blood pressure classified the subjects as normotensive (**Table 2**).

Blood pressure during the post-exercise acute session and after the training period are summarized in **Table 2**. Although there were no significant changes for SBP and DBP at 10, 30 and 60 min after acute exercise compared with pre-exercise values, the greatest decrease (greater individual decrease of clinical BP reached after exercise) in SBP and DBP (considering all time-points) after acute exercise was 9 ± 11 mmHg (p = 0.015) and 9 ± 7 mmHg (p = 0.002), respectively (**Table 2**). There was no chronic effect of training on resting SBP (p = 1.000) and DBP (p = 0.701). Moreover, there was no significant change in SBP and DBP AUC following acute and chronic exercise (**Table 3**). However, considering only the subjects who presented a decrease in SBP greater than 10 mmHg after acute exercise (n = 6), a chronic effect of training was evident in resting SBP (123.8 ± 12.6 mmHg to 117.3 ± 9.7 mmHg after training, p = 0.013) even though no chronic effect in resting DBP was observed (80.3 ± 9.6 mmHg to 77.3 ± 7.3 mmHg after training, p = 0.084).

**Fig. 2** shows that there was a considerable heterogeneity in SBP and DBP responsiveness to RT. The subjects exhibiting the greatest acute reduction in BP also had the highest chronic reduction. Study participants with an acute reduction of approximately 10 mmHg (SBP and DBP) demonstrated chronic reductions of approximately 3 mmHg (SBP and DBP). However, participants with acute reductions lower than 10 mmHg did not change the chronic BP following 8 weeks of training (**Fig. 2**). In this sense, the subjects B, J, K, L and M could be classified as SBP nonresponders and the subjects B, E, I, K and M as DBP nonresponders (**Fig. 2**). The responsiveness of blood pressure to acute exercise was significantly higher for the responders group (SBP, $p = 0.002$ and DBP, $p = 0.043$). The groups did not differ ($p > 0.05$) in terms of age, anthropometric and baseline blood pressure measurements. The chi-square analysis revealed no statistically significant association between the SBP and DBP in the responders group, $\chi^2(1) = 1.592$, p = 0.207). Only 3 subjects were non-responders to SBP and DBP, and 6 subjects were responders to SBP and DBP. Overall, only 2 subjects (15.4 %) were classified as dippers at baseline conditions. However, following acute exercise, 6 subjects (46.2 %) were classified as dippers and after 8 weeks of training, 7 (53.8 %) were classified as dippers (5 were dippers also following acute exercise). In dippers the acute BP response to exercise was correlated with the chronic BP response to training ($p = 0.048$). Moreover, there was no association between the SBP responders groups and dippers after acute exercise ($p = 0.053$) or after 8 weeks of training ($p = 0.725$). In this study, no subject was classified as reverse dipper or extreme dipper.

The Pearson product moment correlations between the magnitudes of acute and chronic changes in clinical and AUC BP are presented in **Table 4**. The reduction in SBP and DBP following exercise was well correlated (r > 0.5; p ≤ 0.05) with the chronic reduction in resting SBP and DBP after training. **Fig. 3** shows the correlation between the magnitudes of change in chronic exercise training and the greatest decrease after acute exercise for SBP and DBP (r = 0.81, p = 0.001 and r = 0.69, p = 0.032, respectively). Similar correlations were also observed between chronic and 10, 30 and 60 min after acute exercise (**Table 4**). The magnitudes of changes in 24 h AUC for SBP and DBP after the acute session were also significantly and strongly correlated with the chronic reduction in resting SBP (r = 0.74, p = 0.004) and DBP

**Table 1** Baseline subject characteristics.

| Variables | n = 13 |
|---|---|
| age, yrs. | 35.7 ± 7.4 |
| height, cm | 159 ± 4 |
| weight, kg | 69.8 ± 15.6 |
| body mass index, kg/m$^2$ | 28.3 ± 5.4 |
| waist circumference, cm | 84.8 ± 9.7 |

**Table 2** Resting and post-exercise blood pressures measurements.

| | Systolic (mmHg) | ES | Diastolic (mmHg) | ES |
|---|---|---|---|---|
| **baseline resting value** | 117.3 ± 11.7 [43.0] | – | 79.3 ± 8.2 [26.0] | – |
| **after acute exercise** | | | | |
| 10 min after exercise | 113.1 ± 4.9 [16.0] | 0.36 | 75.2 ± 5.3 [17.0] | 0.49 |
| 30 min after exercise | 111.7 ± 8.9 [30.0] | 0.48 | 76.3 ± 8.5 [31.0] | 0.47 |
| 60 min after exercise | 114.4 ± 7.3 [28.0] | 0.24 | 78.8 ± 7.1 [22.0] | 0.07 |
| greatest decrease | 108.5 ± 7.0* [26.0] | 0.78 | 71.5 ± 6.4* [18.0] | 1.11 |
| **after chronic exercise** | | | | |
| resting value | 117.3 ± 9.3 [32.0] | 0.00 | 78.8 ± 7.2 [23.0] | 0.11 |

The values are expressed as mean ± standard deviation and [range] (n = 13). *Significantly different from baseline resting value ($p \leq 0.05$); ES, Cohen's *d* effect size

**Table 3** Area under the curve (AUC) for resting and post-exercise blood pressure measurements.

| | Systolic (mmHg/h) | ES | Diastolic (mmHg/h) | ES |
|---|---|---|---|---|
| **24-h** | | 0.08 | | 0.03 |
| baseline AUC | 108.5 ± 11.9 [34.2] | | 67.4 ± 9.1 [26.3] | |
| after acute exercise AUC | 105.7 ± 10.0 [37.3] | | 66.1 ± 7.8 [26.3] | |
| after chronic exercise AUC | 107.9 ± 10.0 [34.1] | | 66.8 ± 7.8 [22.4] | |
| **nighttime** | | 0.08 | | 0.16 |
| baseline AUC | 98.0 ± 13.0 [41.5] | | 59.3 ± 8.2 [21.5] | |
| after acute exercise AUC | 95.3 ± 8.8 [28.0] | | 58.6 ± 6.1 [15.4] | |
| after chronic exercise AUC | 95.1 ± 10.6 [38.4] | | 57.0 ± 6.6 [22.3] | |
| **daytime** | | 0.01 | | 0.01 |
| baseline AUC | 102.8 ± 11.9 [38.5] | | 63.6 ± 10.2 [28.3] | |
| after acute exercise AUC | 103.0 ± 10.7 [28.7] | | 63.6 ± 7.4 [20.1] | |
| after chronic exercise AUC | 103.0 ± 11.6 [43.8] | | 62.2 ± 6.6 [18.1] | |

The values are expressed as mean ± standard deviation and [range] (n = 13). ES, eta-squared ($\eta^2$) effect size

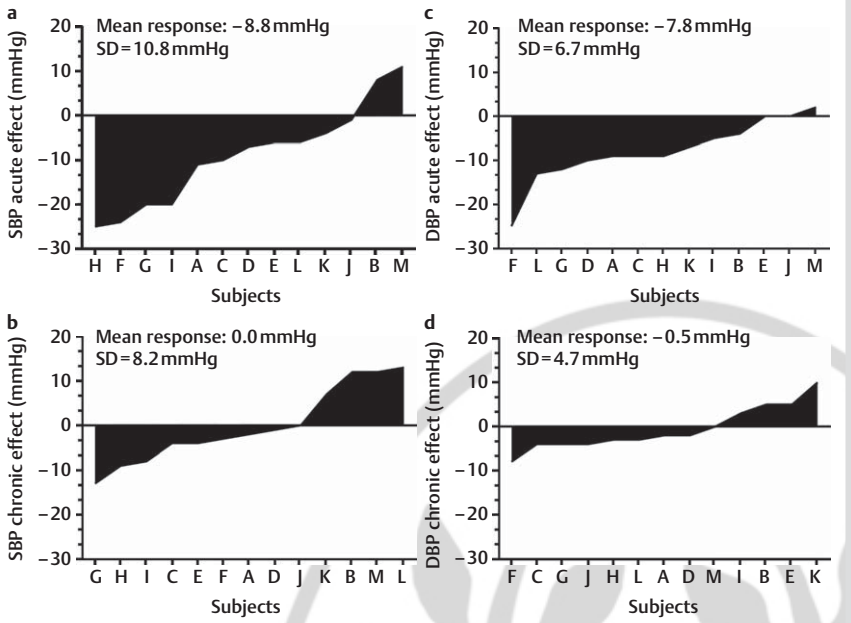


**Fig. 2** Heterogeneity of acute systolic (SBP; **a**) and diastolic blood pressures (DBP; **c**) responsiveness and chronic SBP **b** and DBP **d** effect to a standardized resistance training regimen. The acute effect was calculated as the difference between the baseline and the greatest decrease of blood pressure achieved 60 min after exercise. The chronic effect was calculated as the difference between the baseline and the resting value after 8 weeks of training. The letters A until M represent the subjects of the study. SD, standard deviation.

**Table 4** Pearson product moment correlation of the magnitude of change (Δ) in systolic (SBP) and diastolic (DBP) blood pressures after acute exercise with the magnitude of change in resting SBP and DBP after chronic exercise and Δ area under the curve (AUC) after acute exercise with Δ AUC after chronic exercise.

| | SBP | | DBP | |
|---|---|---|---|---|
| | *r* | *p*-value | *r* | *p*-value |
| **Δ values after chronic exercise vs.** | | | | |
| Δ 10 min after exercise | 0.77 | 0.002 | 0.61 | 0.028 |
| Δ 30 min after exercise | 0.82 | 0.001 | 0.56 | 0.039 |
| Δ 60 min after exercise | 0.73 | 0.004 | 0.55 | 0.042 |
| Δ greatest decrease | 0.81 | 0.001 | 0.69 | 0.032 |
| **Δ AUC after acute vs. chronic exercise** | | | | |
| 24 h | 0.71 | 0.010 | 0.78 | 0.003 |
| nighttime | 0.89 | <0.0005 | 0.61 | 0.027 |
| daytime | 0.60 | 0.031 | 0.91 | <0.0005 |

(r=0.80, p=0.001) (**Fig. 4**). Similar correlations were also observed during the night and daytime (**Table 4**).

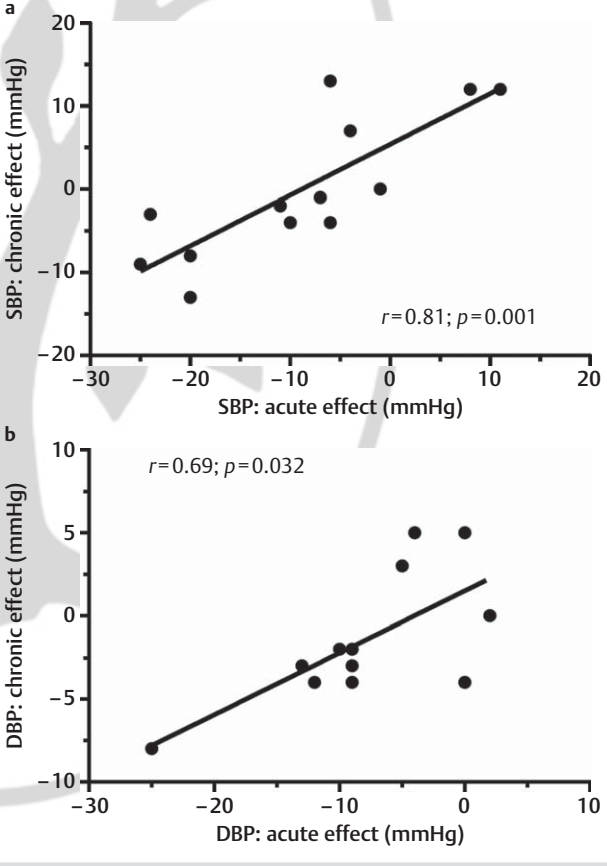


**Fig. 3** Correlation between the magnitudes of change in chronic exercise training and the greatest decrease after acute exercise for systolic (SBP; **a**) and diastolic blood pressure (DBP; **b**).

## Discussion

The main purpose of the present study was to evaluate the acute and chronic effects of RT on BP in middle-aged women and assess the correlation between the degree of PEH following acute exercise and the magnitude of change in resting BP after chronic RT. The novel finding of the present investigation was that the reduction in both SBP and DBP after an acute RT session was strongly correlated with the chronic reduction in resting SBP and DBP in middle-aged women. Our data reinforces previous suggestions regarding the importance of RT in the management of hypertension [18,20], meaning that the magnitude of the acute BP-lowering following exercise may predict the extent of BP lowering after chronic training. An interesting finding was that the greatest value of BP decrease (greater individual decrease of clinical BP reached after exercise) produced the highest correlations with chronic BP reduction, reinforcing the notion that the timing for PEH may vary between individuals and should be used in future studies. Moreover, we also found that some individuals were characterized as responders to SBP or DBP, while some were not. Nevertheless, following the acute exercise, 6 subjects were classified as dippers and after 8 weeks

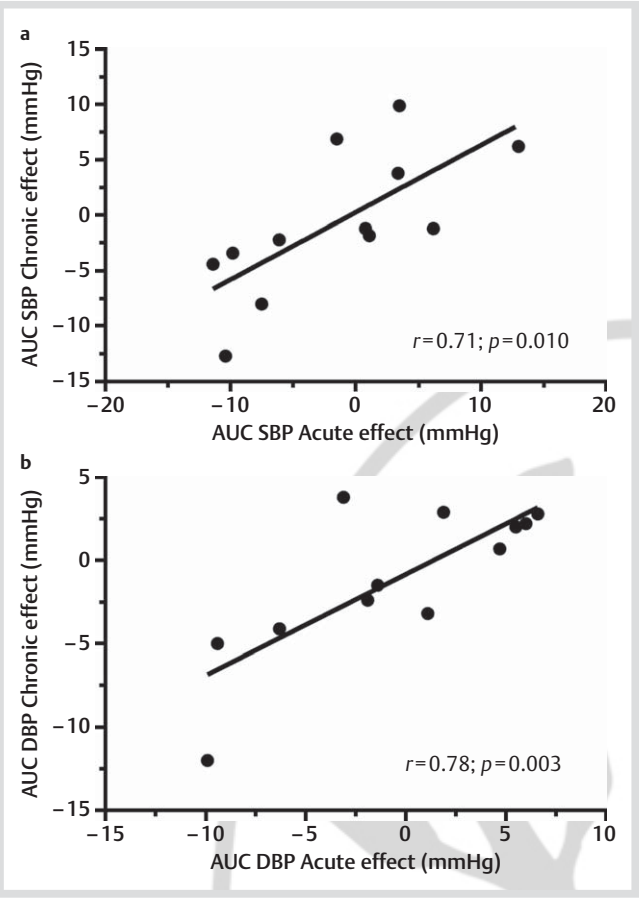


**Fig. 4** Correlation between the magnitudes of change in area under the curve (AUC) for chronic resistance training and AUC for 24 h following acute exercise for systolic (SBP; **a**) and diastolic blood pressure (DBP; **b**).

of training, 7 were classified as dippers (5 were dippers also following acute exercise). There was an association between the dippers following acute exercise and after 8 weeks of training. Importantly, Floras [11] reported that although these categories have been selected arbitrarily, and despite the absence of reproducibility of "dipping" status in a substantial minority of subjects studied, such classification appears to have prognostic value. Thus, even without a statistically significant decrease in BP following acute and chronic exercise, other factors may indicate a decreased cardiovascular risk, as shown by the 7 dippers following 8 weeks of RT. It should be noted that PEH is less consistent and of lower magnitude in normotensive compared to hypertensive subjects [21], which may have affected our results. Apart from the well-documented effects of RT for the maintenance of functional capacity, and prevention of sarcopenia and osteoporosis, a body of evidence shows that RT may also beneficially affect metabolic and cardiovascular health [23, 32]. In this sense, Tibana et al. [32] evaluated the behavior of BP following an acute RT (3 sets with 60% of 1RM for whole body exercises) session in 13 overweight or obese adult women. The results indicate that both SBP and DBP decreased in the nighttime period (−4.2 mmHg and −4.1 mmHg, respectively) and in the overall 24 h period (−3.6 mmHg and −4.5 mmHg, respectively) following the acute RT session compared to the control trial. In addition, Tibana et al. [33] analysed the effects of 8 weeks of RT program (3 sets with 8–12 RM, 3 times per week of whole body RT) on 24 h BP in 17 adult women with (n = 9) and without metabolic syndrome (n = 8). The authors found decreased mean and diastolic night-time BP (−3.9 mmHg, p = 0.04; −5.5 mmHg, p = 0.03, respectively) after 8 weeks of RT only in middle-aged women with metabolic syndrome.

Furthemore, a recent meta-analysis from Cornelissen et al. [8] reported that moderate-intensity RT can reduce SBP (−1.8 mmHg) and DBP (−3.2 mmHg). In the present study, we found that there was a considerable heterogeneity in SBP and DBP responsiveness to RT. For example, the women who presented an acute reduction of approximately 10 mmHg (SBP and DBP) exhibited a chronic reduction of approximately 3 mmHg (SBP and DBP). However, women presenting an acute reduction lower than 10 mmHg showed no change in chronic BP after 8 weeks of RT. The clinical importance of these BP reductions can be estimated from large, prospective intervention studies investigating morbidity and mortality outcomes suggesting that small reductions in resting SBP and DBP of 3 mm Hg can reduce the risk of coronary heart disease by 5%, stroke by 8% and all-cause mortality by 4% [35].

To the best of our knowledge, this is the first study to quantify the correlation between the decrease in BP after an acute RT session and the resting BP reduction associated with chronic training (both up to 60 min following exercise and during 24 h measures). Recently, Hecksteden et al. [18] reported that the magnitude of postexercise hypotension is associated with the training-induced decrease of BP (SBP 60 min post exercise: p = 0.003; r = 0.77; SBP 24 h post exercise: p = 0.017; r = 0.67). Similarly, Liu et al. [20] found that the magnitude of the acute SBP and DBP lowering with submaximal exercise are correlated with the extent of SBP and DBP lowering following 8 weeks of an aerobic training program (4 times per week, 30 min per session, 65% maximum oxygen consumption) in prehypertensive individuals (SBP, r = 0.89; p < 0.01; DBP, r = 0.75, p < 0.01). However, unlike the present study, these studies were performed with aerobic exercise, and the analysis of BP was performed with clinical measurement. We demonstrated that both clinical and 24 h BP response after acute RT were strongly correlated with the chronic reduction in resting SBP and DBP after training (AUC 24 h SBP, r = 0.74, p = 0.004, AUC 24 h DBP, r = 0.80; p = 0.001; greatest decrease of SBP after acute RT, r = 0.81, p = 0.001 and DBP after acute RT r = 0.69, p = 0.032).

Despite the fact that the decline in BP is likely multifactorial, some proposals indicate a decreased cardiac output and peripheral vascular resistance due to lowered sympathetic activity, reduced transduction for vascular tone [3, 4, 13], higher activity of the plasma kallikrein system mediating nitric oxide release [14], and alterations in cerebral blood flow induced by exercise [4]. Additionally, sustained post-exercise vasodilation of the previously active skeletal muscle is primarily the result of histamine H1- and H2-receptor activation [15] following aerobic exercise, but not RT. Moreover, the mechanisms underlying the relationship between post-exercise hypotension and BP reduction following chronic RT are still unknown. It is even possible that acute responders may present higher nitric oxide (NO) release, which has also been related to the intensity in which exercise is performed [26, 27]. Thus, the NO release capability would also be related to exercise-induced chronic training adaptations. Differences in genetic markers of responsiveness to chronic exercise may play a role although the contribution appears to be small. For example the endothelial nitric oxide synthase −786 T > C polymorphism and the exercise-induced blood pressure and NO responses among men with elevated BP [1]. However, aerobic endurance training has been shown to

decrease resting plasma norepinephrine levels as well as muscle sympathetic nerve activity. In contrast, RT may not change sympathetic tone [9].

This study has a number of limitations, such as the reduced number of subjects (although the achieved power was acceptable), absence of a control group, the lack of mechanistic measures and control of the menstrual cycle. Moreover, because the sample comprised adult women, the findings should not be generalized to hypertensive women. Although diet was not controlled in this study, participants were asked to maintain their normal eating habits. Therefore, a randomized controlled follow-up study with more individuals, including a hypertensive group, and longer intervention periods are needed to reinforce the importance of these results.

## Conclusions

In summary, for these adult women, the degree of BP reduction after acute RT is correlated to the magnitude of change in resting BP observed after 8 weeks of RT. This finding makes the magnitude of acute postexercise hypotension following RT a promising candidate for the prediction of individual blood pressure-related training efficacy and may contribute to an individual optimization of training prescription. For example, those subjects who did not present acute PEH could have their training variables (number of sets, repetitions and exercises) modified to improve their training results. In addition, using the greatest value of BP decrease produced after exercise reinforces that the timing for PEH may vary between individuals and should be used in future studies. Finally, more studies should be designed to investigate the acute and chronic effects of RT on the differences in percent decline between nocturnal and daytime BP. It is possible that the different individual reponsiveness to training may require different dose-response prescription for the achievement of optimal results.

## Acknowledgments

This Research was Supported by the Fundação de Amparo à Pesou, Maranhao, CFAPEM.

**Conflicts of interest:** The authors declare no conflict of interest.

**Affiliations**
[1] Graduation Program on Physical Education, Catholic University of Brasilia, Brasilia, Brazil
[2] Graduation Program Interunits in Bioengineering, Sao Carlos, Brazil
[3] Physical Education Departement, Federal University of Maranhao, Sao Luis, Brazil
[4] Physical Education, University of Toronto, Toronto, Canada
[5] Physical Education, UNIEURO Universitary Center, Brasilia, Brazil

## References

1 *Augeri AL*, *Tsongalis GJ*, *Van Heest JL*, *Maresh CM*, *Thompson PD*, *Pescatello LS*. The endothelial nitric oxide synthase −786 T>C polymorphism and the exercise-induced blood pressure and nitric oxide responses among men with elevated blood pressure. Atherosclerosis 2009; 204: e28–e34
2 *Brown LE*, *Weir JP*. ASEP procedures recommendation I: Accurate assessment of muscular strength and power. JEPonline 2001; 4: 1–21
3 *Brum PC*, *Da Silva GJ*, *Moreira ED*, *Ida F*, *Negrão CE*, *Krieger EM*. Exercise training increases baroreceptor gain sensitivity in normal and hypertensive rats. Hypertension 2000; 36: 1018–1022
4 *Chen CY*, *Bonham AC*. Postexercise hypotension: central mechanisms. Exerc Sport Sci Rev 2010; 38: 122–127
5 *Chobanian AV*, *Bakris GL*, *Black HR*, *Cushman WC*, *Green LA*, *Izzo JL Jr*, *Jones DW*, *Materson BJ*, *Oparil S*, *Wright JT Jr*, *Roccella EJ*. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure: the JNC 7 report. Hypertension 2003; 42: 1206–1252
6 *Cohen J*. Statistical Power Analysis for the Behavioral Sciences. New York: L. Erlbaum Associates, 1988; 559
7 *Cornelissen VA*, *Fagard RH*, *Coeckelberghs E*, *Vanhees L*. Impact of resistance training on blood pressure and other cardiovascular risk factors: a meta-analysis of randomized, controlled trials. Hypertension 2011; 58: 950–958
8 *Cornelissen VA*, *Smart NA*. Exercise training for blood pressure: a systematic review and meta-analysis. J Am Heart Assoc 2013; 2: e004473
9 *Cornelissen VA*, *Fagard RH*. Effect of resistance training on resting blood pressure: a meta-analysis of randomized controlled trials. J Hypertens 2005; 23: 251–259
10 *de la Sierra A*, *Zamorano JL*, *Ruilope LM*. Application of hypertension guidelines in clinical practice: implementation of the 2007 ESH/ESC European practice guidelines in Spain. J Hypertens 2009; Suppl 27: S27–S32
11 *Floras JS*. Blood pressure variability: A novel and important risk factor. Can J Cardiol 2013; 29: 557–563
12 *Giles T*. Relevance of blood pressure variation in the circadian onset of cardiovascular events. J Hypertens 2005; 23: S35–S39
13 *Halliwill JR*. Mechanisms and clinical implications of post-exercise hypotension in humans. Exerc Sport Sci Rev 2001; 29: 65–70
14 *Halliwill JR*, *Minson CT*, *Joyner MJ*. Effect of systemic nitric oxide synthase inhibition on postexercise hypotension in humans. J Appl Physiol 2000; 89: 1830–1836
15 *Halliwill JR*, *Buck TM*, *Lacewell AN*, *Romero SA*. Postexercise hypotension and sustained postexercise vasodilatation: what happens after we exercise? Exp Physiol 2013; 98: 7–18
16 *Harriss DJ*, *Atkinson G*. Ethical standards in sport and exercise science research: 2014 update. Int J Sports Med 2013; 34: 1025–1028
17 *Hartwich D*, *Aldred S*, *Fisher JP*. Influence of menstrual cycle phase on muscle metaboreflex control of cardiac baroreflex sensitivity, heart rate and blood pressure in humans. Exp Physiol 2013; 98: 220–232
18 *Hecksteden A*, *Grütters T*, *Meyer T*. Association between postexercise hypotension and long-term training-induced blood pressure reduction: a pilot study. Clin J Sport Med 2013; 23: 58–63
19 *Kraemer WJ*, *Adams K*, *Cafarelli E*, *Dudley GA*, *Dooly C*, *Feigenbaum MS*, *Fleck SJ*, *Franklin B*, *Fry AC*, *Hoffman JR*, *Newton RU*, *Potteiger J*, *Stone MH*, *Ratamess NA*, *Triplett-McBride T*. American College of Sports Medicine position stand. Progression models in resistance training for healthy adults. Med Sci Sports Exerc 2002; 34: 364–380
20 *Liu S*, *Goodman J*, *Nolan R*, *Lacombe S*, *Thomas SG*. Blood pressure responses to acute and chronic exercise are related in prehypertension. Med Sci Sports Exerc 2012; 44: 644–1652
21 *Macdonald JR*. Potencial causes, mechanisms, and implications of post exercise hypotension. J Hum Hypertens 2002; 16: 225–236
22 *Minson CT*, *Halliwill JR*, *Young TM*, *Joyner MJ*. Influence of the menstrual cycle on sympathetic activity, baroreflex sensitivity, and vascular transduction in young women. Circulation 2000; 101: 862–868
23 *Morais PK*, *Campbell CS*, *Sales MM*, *Motta DF*, *Moreira SR*, *Cunha VN*, *Benford RE*, *Simões HG*. Acute resistance exercise is more effective than aerobic exercise for 24h blood pressure control in type 2 diabetics. Diabetes Metab 2011; 37: 112–117
24 *Pescatello LS*, *Franklin BA*, *Fagard R*, *Farquhar WB*, *Kelley GA*, *Ray CA*. American College of Sports Medicine position stand. Exercise and hypertension. Med Sci Sports Exerc 2004; 36: 533–553
25 *Rossi AM*, *Moullec G*, *Lavoie KL*, *Gour-Provençal G*, *Bacon SL*. The evolution of a Canadian hypertension education program recommendation: the impact of resistance training on resting blood pressure in adults as an example. Can J Cardiol 2013; 29: 622–627
26 *Santana HA*, *Moreira SR*, *Asano RY*, *Sales MM*, *Córdova C*, *Campbell CS*, *Espindola FS*, *Sposito AC*, *Nóbrega OT*, *Simões HG*. Exercise intensity modulates nitric oxide and blood pressure responses in hypertensive older women. Aging Clin Exp Res 2013; 25: 43–48
27 *Santana HA*, *Moreira SR*, *Neto WB*, *Silva CB*, *Sales MM*, *Oliveira VN*, *Asano RY*, *Espíndola FS*, *Nóbrega OT*, *Campbell CS*, *Simões HG*. The higher exercise intensity and the presence of allele I of ACE gene elicit a higher post-exercise blood pressure reduction and nitric oxide release in elderly women: an experimental study. BMC Cardiovasc Disord 2011; 11: 71
28 *Schmidt MI*, *Duncan BB*, *Hoffmann JF*, *Ld Moura*, *Malta DC*, *Carvalho RM*. Prevalence of diabetes and hypertension based on self-reported morbidity survey, Brazil, 2006. Rev Saude Publica 2009; 43: 74–82

29 *Scharhag-Rosenberger F*, *Meyer T*, *Walitzek S*, *Kindermann W*. Time course of changes in endurance capacity: a 1-yr training study. Med Sci Sports Exerc 2009; 41: 1130–1137
30 *Singh A*, *Gianos E*, *Schwartzbard A*, *Black H*, *Weintraub H*. Use of ambulatory blood pressure monitoring to guide hypertensive therapy. Curr Treat Options Cardiovasc Med 2013; 15: 746–760
31 *Tajra V*, *Tibana RA*, *Vieira DCL*, *Farias DL*, *Teixeira TG*, *Funghetto SS*, *Silva AO*, *Sousa NMF*, *Willardson J*, *Karnikowski MGO*, *Prestes J*. Identification of high responders for interleukin-6 and creatine kinase following acute eccentric resistance exercise in elderly obese women. J Sci Med Sport 2013; in press
32 *Tibana RA*, *Pereira GB*, *Navalta JW*, *Bottaro M*, *Prestes J*. Acute effects of resistance exercise on 24-h blood pressure in middle-aged overweight and obese women. Int J Sports Med 2013; 34: 460–464
33 *Tibana RA*, *Pereira GB*, *de Souza JC*, *Tajra A*, *Vieira DC*, *Campbell CS*, *Cavaglieri CR*, *Prestes J*. Resistance training decreases 24-hour blood pressure in women with metabolic syndrome. Diabetol Metab Syndr 2013; 5: 27
34 *Vasan RS*, *Beiser A*, *Seshadri S*, *Larson MG*, *Kannel WB*, *D'Agostino RB*, *Levy D*. Residual lifetime risk for developing hypertension in middle-aged women and men: The Framingham Heart Study. JAMA 2002; 287: 1003–1010
35 *Whelton PK*, *He J*, *Appel LJ*, *Cutler JA*, *Havas S*, *Kotchen TA*, *Roccella EJ*, *Stout R*, *Vallbona C*, *Winston MC*, *Karimbakas J*. Primary prevention of hypertension: clinical and public health advisory from The National High Blood Pressure Education Program. JAMA 2002; 288: 1882–1888