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


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R. A. Tibana1, N. M. F. de Sousa2, D. da Cunha Nascimento1, G. B. Pereira1, S. G. Thomas1, S. Balsamo1, H. G. Simoes1, J. Prestes1

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 ≤ 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 cardiovascular disease, including myocardial infarction, heart failure, stroke and kidney disease [5]. The treatment of SAH involves pharmacological and non-pharmacological therapies [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.
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]. Nevertheless, 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 JOHNSTON (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.
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 manufacturer’s instructions. All BP measurements 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

24h 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 (η²) 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 - β) achieved in this research was 0.72 for DBP and 0.82 for SBP. The level of significance was p < 0.05, and SPSS version 20.0 (Sommers, 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 BP 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 3 mmHg (SBP and DBP) demonstrated chronic reductions of approximately 10 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).

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 acute 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.

<table>
<thead>
<tr>
<th>Variables</th>
<th>n = 13</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>age, yrs.</td>
<td>35.7 ± 7.4</td>
<td>35.7 ± 7.4</td>
<td>35.7 ± 7.4</td>
</tr>
<tr>
<td>height, cm</td>
<td>159 ± 4</td>
<td>159 ± 4</td>
<td>159 ± 4</td>
</tr>
<tr>
<td>weight, kg</td>
<td>69.8 ± 15.6</td>
<td>69.8 ± 15.6</td>
<td>69.8 ± 15.6</td>
</tr>
<tr>
<td>body mass index, kg/m²</td>
<td>28.3 ± 5.4</td>
<td>28.3 ± 5.4</td>
<td>28.3 ± 5.4</td>
</tr>
<tr>
<td>waist circumference, cm</td>
<td>84.8 ± 9.7</td>
<td>84.8 ± 9.7</td>
<td>84.8 ± 9.7</td>
</tr>
</tbody>
</table>

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

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

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

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

<table>
<thead>
<tr>
<th></th>
<th>Systolic (mmHg/h)</th>
<th>ES</th>
<th>Diastolic (mmHg/h)</th>
<th>ES</th>
</tr>
</thead>
<tbody>
<tr>
<td>baseline AUC</td>
<td>108.5 ± 11.9 [34.2]</td>
<td>0.08</td>
<td>67.4 ± 9.1 [26.3]</td>
<td>0.03</td>
</tr>
<tr>
<td>after acute exercise AUC</td>
<td>105.7 ± 10.0 [37.3]</td>
<td>66.1 ± 7.8 [26.3]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>after chronic exercise AUC</td>
<td>107.9 ± 10.0 [34.1]</td>
<td>66.8 ± 7.8 [22.4]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>nighttime AUC</td>
<td>98.0 ± 13.0 [41.5]</td>
<td>0.08</td>
<td>59.3 ± 8.2 [21.5]</td>
<td>0.16</td>
</tr>
<tr>
<td>baseline AUC</td>
<td>95.3 ± 8.8 [28.0]</td>
<td>0.08</td>
<td>58.6 ± 6.1 [15.4]</td>
<td></td>
</tr>
<tr>
<td>after chronic exercise AUC</td>
<td>95.1 ± 10.6 [38.4]</td>
<td>57.0 ± 6.6 [22.3]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>daytime AUC</td>
<td>102.8 ± 11.9 [38.5]</td>
<td>0.01</td>
<td>63.6 ± 10.2 [28.3]</td>
<td>0.01</td>
</tr>
</tbody>
</table>

The values are expressed as mean ± standard deviation and [range] (n = 13). ES, eta-squared (η²) effect size.
Similar correlations were also observed during the night and daytime (Fig. 4). Similar correlations were also observed during the night and daytime (Table 4).

**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...
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 benefit other factors such as mortality and morbidity outcomes suggesting that small reductions in resting SBP and DBP of 3 mmHg 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: r = 0.003; r = 0.77; SBP 24 h post exercise: r = 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 [4], 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 Pesquisa, Maranhão, CFAPEM.

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

Affiliations

Graduation Program on Physical Education, Catholic University of Brasilia, Brasilia, Brazil
Graduation Program in Bioengineering, Sao Carlos, Brazil
Physical Education Department, Federal University of Maranhao, Sao Luis, Brazil
Physical Education, University of Toronto, Toronto, Canada
Physical Education, UNIEURO Universitary Center, Brasilia, Brazil

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