ORIGINAL RESEARCH

Acute and chronic effects of resistance exercise on blood pressure in elderly women and the possible influence of ACE I/D polymorphism

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Abstract: This study investigated the chronic effect of blood pressure (BP) and post-exercise hypotension (PEH) during resistance training (RT) and its relation with the angiotensin-converting enzyme (*ACE*) gene insertion/deletion (I/D) polymorphism in hypertensive elderly women. Participants were divided into two groups: an experimental group (EG) with exercise and a control group (CG) without exercise. The EG performed one adaptation month and one repetition maximum load (1RM) test at the end of this period. After the first month, the EG conducted a three-month program of RT at 60%, 70%, and 80% of 1RM, respectively, for each month. The CG was evaluated at the end of each month. Systolic (SBP) and diastolic (DBP) blood pressure (Microlife BP 3AC1-1) were measured, with the subject in a seated position, during an acute session for both GE and CG as follows: every 5 minutes for 20 minutes at pre-exercise rest, immediately after the resistance exercise and control, and every 15 minutes during 1 hour of recovery after exercise and CG. Analysis of covariance showed reduction in SBP and DBP ($P \leq 0.05$) rest values after the RT program. PEH was observed only for the EG in acute sessions, for SBP after the second and third months ($P \leq 0.05$), and for DBP after the second and fourth months ($P \leq 0.05$). No significant differences in main effects and interaction effects between blood pressure and ACE I/D were observed. The occurrence of chronic reduction of blood pressure and PEH through EG may have a protective effect on the cardiovascular system with no ACE I/D polymorphism influence for this population.

Keywords: post-exercise hypotension, resistance exercise, angiotensin-converting enzyme, genetics, polymorphism

Introduction

Hypertension is a multifactorial and multicausal syndrome characterized by high blood pressure (BP) levels ($\geq$140/90 mmHg), usually associated with metabolic, hormonal, and structural disorders, and representing a primary risk for coronary disease.[1] The non-pharmacological treatment of hypertension through physical exercise can produce significant hemodynamic changes including increases in muscle blood flow, nitric oxide production, and α1 and α2 adrenergic receptors density in skeletal muscles.[2,3]

The importance of performing aerobic and resistance exercise to prevent the risk of stroke by the promotion of post-exercise hypotension (PEH) is evidenced by several studies.[4–8] However, although post-aerobic exercise hypotension is well established,[1,9] few studies have investigated post-resistance exercise hypotension; those that have show conflicting results, with some studies showing reduction,[10–13] maintenance,[14] or even increase[15] in BP after a resistance exercise session.

Beyond the multifactorial issues mentioned above, genetic characteristics may be related to blood pressure (BP) and cardiovascular diseases (CVDs). A study conducted with 496 subjects observed associations between CVD and polymorphisms of genes related to the renin–angiotensin–aldosterone system (RAAS),[16] which plays an important role in circulatory homeostasis. In humans, plasma levels of angiotensin-converting enzyme (ACE) may be related to the insertion/deletion (I/D) polymorphism of the *ACE* gene, located on chromosome 17.[17,18] Rigat et al[18] investigated the *ACE* gene I/D polymorphism in 80 healthy subjects and observed that DD homozygotes showed a greater concentration of circulating ACE than the ID heterozygotes and II homozygotes genotypes. Moreover, another study found a positive association between ACE I/D polymorphism and hypertension.[19]

Floras et al[20] observed that the effect of 45 minutes of acute submaximal treadmill exercise on PEH magnitude may be linked both to initial BP levels and to genetic factors. Thus, research on chronic diseases such as hypertension requires a more detailed assessment of gene polymorphisms in RAAS. Taken together, the occurrence of PEH and the possible association with genetic factors or even with ethnic and genetic factors[21] would aid in prescribing more precise exercise regimes for elderly.

To the authors' knowledge, there have been no scientific studies relating the decrease in BP after chronic resistance training and ACE I/D polymorphism. It would be useful to analyze the BP throughout a resistance exercise program and its association with ACE I/D polymorphism at different intensities in hypertensive women in order to clarify this issue. Thus, the purpose of this study was to investigate the chronic effect and PEH occurrence during a 4-month period of resistance training and its relation with ACE I/D polymorphism in hypertensive elderly women.

Methods
Experimental approach to the problem

Subjects were divided into two groups to perform resistance training (experimental group [EG], n = 32) and control sessions without exercise (control group [CG], n = 32) for 16 weeks. The exercise sessions were performed three times per week. Three sets of ten exercises, using an adapted protocol,[22] were performed in the following order: lat pull-down, knee extension, chest press in a vertical machine, hip abduction, knee flexion, abduction of the shoulders with free weights, free-standing calf raises, sit-ups, trunk extension, and leg press 45°. The velocity of execution adopted was 2 seconds for both concentric and eccentric phases (2:2). Before and after each exercise session, stretching exercises for relevant major muscle groups were performed. Load progression occurred each month in order to respect physiological adaptations, as well as to establish the usefulness of the protocol on the variables measured. The weight machines used were by Righetto® (São Paulo, Brazil), and all tests were conducted around 3 pm.

Subjects

After informed consent was signed, 64 elderly female subjects (67.1 ± 6.2 years; 66.4 ± 13.4 kg; 142.2 ± 5.7 cm) were selected to participate (Table 1). The women had been sedentary for at least 6 months and previously diagnosed with hypertension controlled with the use of antihypertensive medication. To participate in the intervention, the subjects underwent a cardiac evaluation consisting of a resting electrocardiogram and an effort test on a treadmill. The study was approved by the ethics committee of the Catholic University of Brasilia, Brasilia, Brazil (process number 075/2006).

Experimental procedures
Determination of one repetition maximum (1RM) load

Tests were conducted as per the protocol of Kraemer and Fry[23] after 3 weeks of training that aimed to promote neural adaptation and efficiency of motor control.[24] No 1RM tests were performed for the abduction of the shoulder, sit-ups, trunk extension, and free-standing calf exercises.

Resistance exercise sessions

The EG performed 60 sessions of resistance exercise lasting 40 minutes, at the same time of day (2.40 to 4.40 pm) on different days, as follows:

- Month 1: subjects were submitted to an adaptation period of 12 sessions of resistance exercise at light intensity, performing ten repetitions in each set with a 30-second rest interval between sets. At the end of this first month, 1RM was applied as per the previous description.
- Month 2: 16 sessions of resistance exercise at 60% of 1RM; 12 repetitions with a 60-second rest interval between sets.
- Month 3: 16 sessions of resistance exercise at 70% of 1RM; ten repetitions with a 60-second rest interval between sets.
- Month 4: 16 sessions of resistance exercise at 80% of 1RM; eight repetitions with a 90-second rest interval between sets.

Extraction of DNA: genotyping

Venous blood samples (5 mL) were collected from the antecubital vein of each subject, drawn by a specialized

Table 1 Descriptive characteristics of experimental (EG) and control (CG) groups (n = 64)

Patient characteristics	EG n = 32	CG n = 32	T-value	P-value
II genotype (n)	11	10	–	–
ID genotype (n)	9	11	–	–
DD genotype (n)	12	11	–	–
Age (years)	67.5 ± 7.0	66.8 ± 5.4	0.49	0.63
Body weight (kg)	65.4 ± 14.3	67.4 ± 12.6	−0.62	0.54
Height (cm)	153.3 ± 5.9	151.2 ± 6.2	1.42	0.16
BMI (kg · [m²]⁻¹)	27.8 ± 5.5	29.4 ± 4.6	−1.32	0.19
Resting SBP (mmHg)	134.5 ± 14.6	131.8 ± 16.9	0.67	0.51
Resting DBP (mmHg)	76.0 ± 9.2	74.3 ± 7.4	0.82	0.42
Resting MBP (mmHg)	94.9 ± 10.0	93.4 ± 9.5	−0.54	0.59
Resting HR (bpm)	72.2 ± 13.5	73.7 ± 9.4	−0.33	0.75
Resting DP (mmHg · bpm)	9544.3 ± 1927.0	9686.9 ± 1549.1	0.63	0.53

Notes: Data are presented as mean ± standard deviation. II genotype: two alleles with 287 bp insertion in *ACE* gene; DD genotype: two alleles with 287 bp deletion in *ACE* gene; ID genotype: one allele with 287 bp insertion and one allele with 287 bp deletion in *ACE* gene.
Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; DP, double product; HR, heart rate; MBP, mean blood pressure; SBP, systolic blood pressure.

professional and stored in sterile vacuum tubes containing ethylenediamine tetraacetic acid (EDTA) anticoagulant. Then, DNA extraction was performed by the salting-out method,[25] and high-molecular weight genomic DNA was extracted from peripheral leukocytes. The method used for DNA quantification was electrophoresis on agarose gel (1%), stained with 3 μL of ethidium bromide to estimate the concentration of DNA extracted in advance.

DNA amplification: polymerase chain reaction (PCR)
For DNA amplification, the PCR technique, which amplifies the DNA fragment containing the insertion polymorphism, was applied. The methodology of Zhao et al[26] was adopted to assess the direct (forward) and reverse primers.

Classification of *ACE* gene polymorphism
The samples were classified by PCR into one of three possible genotypes for the polymorphism of ACE: two homozygotes (DD and II) and one heterozygote (ID). By ultraviolet light after electrophoresis at 80 V in 1% agarose gel, it was possible to visualize the PCR products. The identification of genotypes was performed by viewing the presence of the alleles D and I; the presence of only a fragment of 190 base pairs characterizes the DD genotype and the presence of only a fragment of 490 base pairs characterizes the II genotype. The ID heterozygotes were identified by the presence of both fragments. The analysis of genotype identification was performed separately by two researchers. To increase the specificity of genotyping, an additional confirmatory PCR was performed with all samples carrying the DD genotype, using a pair of primers specific for the insertion, as used in

previous studies.[27,28] Samples carrying the ID or II genotype were used as positive controls during this reamplification.

Procedure for measurements of BP and heart rate (HR)
Measurements of systolic (SBP) and diastolic (DBP) BP were evaluated using an automatic BP device (BP 3AC1-1; Microlife, Switzerland) following the guidelines of a previous study.[29] HR was measured using a specific monitor (FS 3, Polar Sport Tester, Finland). The variables were measured with the subject in a seated position at the end of each month for both GE and CG as follows:

- EG: measurement of BP and HR every 5 minutes for 20 minutes at pre-exercise rest, as well as during and immediately after resistance exercise, and every 15 minutes during 1 hour of post-exercise recovery.
- CG: measurement of BP and HR every 5 minutes for 20 minutes at rest before the session, as well as immediately after and every 15 minutes for 1 hour after the control session.

Statistical analyses
Statistical analyses were performed using Statistical Package for Social Sciences (SPSS) for Windows (v 10.0; IBM Corporation, Armonk, NY, USA). The descriptive analyses were presented as mean ± standard deviation (SD). The assessment of normality was obtained by analyses of skewness and the Kolmogorov–Smirnov test. To analyze homoscedasticity, the Levene's test was applied.

Analysis of variance (ANOVA) for repeated measures and Bonferroni test of multiple comparisons were adopted to

verify the chronic effect on BP and PEH at different moments in the months of training in the EG and CG. The Student's *t*-test for independent samples was applied to assess differences between the groups. Finally, an analysis of covariance ([ANCOVA] group versus genotype versus months of training time) was applied with the Bonferroni post hoc test to analyze the effects of training on SBP and DBP. The level of significance adopted was $P \leq 0.05$.

Results

Both SBP and DBP were reduced with resistance training when the rest values from the first and fourth months were compared (Tables 2 and 3; $P \leq 0.05$). PEH was observed after acute sessions at the end of the second and third months for SBP analysis (F[15.930] = 14.5, $P = 0.001$) for EG. At months 1 and 2, SBP did not differ between groups. At the acute session at the end of month 3, there was a significant difference between groups in the recovery period of 30 ($P = 0.009$) and 45 ($P = 0.05$) minutes. At month 4, significant differences between groups at all times of recovery were observed ($P \leq 0.05$). The CG showed neither PEH nor differences among the rest BP in the 4 months (Table 2). The differences in SBP in the EG and CG are shown in Table 2.

PEH for DBP was observed after the acute sessions at the end of the second and fourth months for the EG (F[15.930] = 7.10; $P = 0.001$). At month 1, there was no significant difference between groups. At month 2, there were significant differences between groups at rest ($P = 0.001$) and at the recovery period of 60 minutes ($P = 0.001$). At months 3 and 4, there were no significant differences between groups. The CG showed neither PEH nor differences among the rest BP for the

four months (Table 3). DBP analyses are also shown in Table 3.

The SBP and DBP responses during resistance training and in the control group without exercise, and their interactions to the ACE I/D polymorphism are showed in Tables 4 and 5, respectively. An influence of rest DBP values was observed by months (F[1.54] = 108.76; $P = 0.001$) and used as a covariate to the ACE I/D polymorphism interactions. ANCOVA did not show difference in main effects and interaction effects of the ACE I/D polymorphism and PEH for both SBP and DBP (Tables 4 and 5, respectively) for the EG and the CG.

Discussion

This study evaluated the chronic effects of resistance exercises on BP and the PEH (acute effect) during resistance training in hypertensive physically inactive women aged 60–75 years; the possible relation between PEH and the ACE I/D polymorphism was also investigated. Our main findings were that ANCOVA showed a chronic reduction in rest values of SBP and DBP only for the EG (Tables 2 and 3; $P \leq 0.05$). Also, PEH was observed only for the EG at acute seccions, for SBP after the second and third months (Table 2; $P \leq 0.05$) and for DBP after the second and fourth months (Table 3; $P \leq 0.05$). No significant differences in main effects and interaction effects between BP and genotypes were observed (Tables 4 and 5). These chronic and acute patterns were not evidenced in the CG.

The findings of this study about the response of SBP to resistance exercise corroborate the results in the scientific literature about the acute[7,30] and chronic[12,22,31] benefits of this type of exercise. We found magnitude of decrease in the PEH of approximately 13.0 mmHg in month 2 and about 5.6 mmHg in month 3 for SBP in the EG (Table 2).

Table 2 Systolic blood pressure responses (mmHg) in the experimental (EG) and control (CG) groups

Month	Rest	Recovery period			
		15 minutes	30 minutes	45 minutes	60 minutes
EG					
1	134.5 ± 14.6	133.6 ± 13.6	132.2 ± 13.4	132.2 ± 13.5	133.5 ± 12.6
2	134.5 ± 14.6	126.4 ± 16.5*∽	122.4 ± 13.8*∽	121.5 ± 14.8*∽	128.2 ± 14.4*∽
3	124.9 ± 11.8∽,†	121.6 ± 11.9∽,†	119.3 ± 13.0*∽	120.9 ± 14.1∽	122.8 ± 14.0∽,†
4	120.2 ± 11.8∽,†,¤	118.8 ± 11.7∽,†	117.5 ± 12.1∽	117.9 ± 11.4∽	120.3 ± 12.0∽,†
CG					
1	131.8 ± 16.9	127.6 ± 16.6	129.7 ± 17.0	128.2 ± 18.1	127.8 ± 16.8
2	129.1 ± 15.8	128.8 ± 18.7	129.6 ± 17.3	128.6 ± 16.6	127.8 ± 16.8
3	129.1 ± 15.8	128.7 ± 18.7	129.6 ± 17.3#	128.6 ± 16.7#	128.4 ± 18.0
4	132.3 ± 17.6	130.4 ± 19.9#	128.7 ± 18.3#	128.2 ± 19.5#	127.7 ± 16.7#

Notes: Data are presented as mean ± standard deviation. *$P \leq 0.05$ in relation to rest; ∽$P \leq 0.05$ in relation to month 1; †$P \leq 0.05$ in relation to month 2; ¤$P \leq 0.05$ in relation to month 3; #$P \leq 0.05$ in relation to the same moments in EG.

Table 3 Diastolic blood pressure responses (mmHg) in the experimental (EG) and control (CG) groups

Month	Rest	Recovery period			
		15 minutes	30 minutes	45 minutes	60 minutes
EG					
1	76.0 ± 9.2	75.4 ± 10.0	74.5 ± 11.3	73.9 ± 9.6	76.5 ± 10.1
2	80.9 ± 11.1∞	73.0 ± 10.0*	73.2 ± 9.5*	75.9 ± 9.9*	81.0 ± 11.1∞
3	74.5 ± 9.5†	74.5 ± 9.5	74.2 ± 9.5	74.2 ± 9.5	74.1 ± 9.5∞,†
4	72.4 ± 9.3∞,†	72.2 ± 10.3	69.7 ± 9.2*,∞,†,¤	70.3 ± 9.7*,∞,†,¤	72.5 ± 10.1∞,†
CG					
1	74.3 ± 7.4	72.5 ± 7.2	74.0 ± 7.8	73.4 ± 7.6	73.3 ± 7.5
2	73.0 ± 7.2#	72.9 ± 7.9	72.1 ± 7.8	72.6 ± 8.0	72.7 ± 6.9#
3	73.5 ± 7.4	72.8 ± 8.1	72.2 ± 8.1	72.6 ± 7.1	72.2 ± 7.6
4	73.8 ± 7.8	73.7 ± 7.9	73.6 ± 8.6	73.5 ± 8.4	72.8 ± 8.4

Notes: Data are presented as mean ± standard deviation. *$P \leq 0.05$ in relation to rest; ∞$P \leq 0.05$ in relation to month 1; †$P \leq 0.05$ in relation to month 2; ¤$P \leq 0.05$ in relation to month 3; #$P \leq 0.05$ in relation to the same moments in EG.

In regard to our DBP results, there were significant reductions in moments of acute post-exercise, similar to the results of other studies.[20,32] In general, we observed a PEH magnitude of approximately 7.9 mmHg in month 2 and about 2.7 mmHg in month 4 for the subjects who trained (Table 3).

It is clear from the literature that PEH is an acute benefit from physical exercise. Its mechanisms are not clear, but may be related to multifactorial origins.[21] Anyway, reductions in sympathetic activation, in cardiac output and the maintenance of the peripheral vascular resistance that decrease after exercise, may be the physiological mechanisms to promote PEH.[1,9,33]

The average reduction in SBP at the end of the training period was 14.3 mmHg, a relevant result with regard to non-pharmacological approaches for prevention, treatment, and control of hypertension in elderly women. A population study conducted by Stamler[33] showed that small decreases in BP can protect the cardiovascular system. Reductions in SBP of 2–5 mmHg may decrease the risk of infarction in 6%–14% and the risk of coronary heart disease in 4%–9%, also reducing mortality from all causes by 3%–7%. These data are relevant when analyzing the results of the present study, in which a significant

reduction in SBP of approximately 14.0 mmHg as a result of chronic resistance training for 4 months was observed. Additionally, a chronic reduction of 3.6mmHg was observed in the EG. This chronic effect reinforces the importance of resistance training in order to prevent hypertension and promote the health of elderly hypertensive people.

Our results from SBP and DBP responses after resistance exercise and control sessions showed no relation to the ACE I/D polymorphism (Tables 4 and 5). In fact, ACE I/D polymorphism and PEH of SBP showed a tendency of association only at month 2, with more PEH for the II genotype subjects.

Although the training protocols were different, our findings corroborate to some extent the results of Dengel et al,[34] which showed no significant interaction between genotype and aerobic training in relation to SBP, DBP, and mean BP. To the contrary, relatively recent results[35,36] showed that 10 weeks of training on a cycle ergometer (aerobic exercise) significantly decreased the levels of SBP and DBP only in patients with genotypes II and ID, and not in the subjects with the DD genotype of *ACE*.

Despite the absence of association between ACE I/D genotypes and DBP in our study, Blanchard et al[37] showed

Table 4 ANCOVA results and their interactions with SBP

Variables	F-value	P-value	η^2
Genotypes	0.55	0.58	0.020
Groups × genotypes × PEH	1.26	0.23	0.105
Groups × genotypes × months	1.53	0.13	0.124
Groups × genotypes × PEH × months	1.31	0.12	0.108

Abbreviations: ANCOVA, analysis of covariance; PEH: post-exercise hypotension; SBP, systolic blood pressure.

Table 5 ANCOVA results and their interactions with DBP

Variables	F-value	P-value	η^2
Genotypes	2.99	0.06	0.100
Groups × genotypes × PEH	1.15	0.32	0.096
Groups × genotypes × months	1.03	0.42	0.087
Groups × genotypes × PEH × months	0.92	0.57	0.079

Abbreviations: ANCOVA, analysis of covariance; DBP, diastolic blood pressure; PEH, post-exercise hypotension.

a greater DBP drop in subjects with DD genotype in relation to genotypes II and ID after mild aerobic training at 40% VO_2 max for men after aerobic exercise. The study by Kim[38] was closer to ours in regard to population evaluated and type of exercise, but not with respect to the result. This author observed that, in adult women, the DD genotype showed greater reduction of DBP, compared to the II group, when performing aerobic and resistance exercise two to three times a week.

The I/D polymorphism of the *ACE* gene might be related to different effects of resistance exercise related to health, such as strength gains, muscle hypertrophy,[38,39] and BP response to exercise.[40] In addition, it appears that the DD genotype of the *ACE* gene is associated with a higher incidence of hypertension.[41] Investigations on the interaction of different I/D genotypes of *ACE* in response to resistance exercise are of great importance in regard to health promotion in the general population and particularly in the elderly, who are prone to development of hypertension. However, the present study showed only a tendency of PEH to be different between the DD and ID genotypes to the SBP in the second month of training, showing differences neither in the interaction between the II, ID, and DD *ACE* genotypes in relation to SBP in other months nor to DBP.

There are a small number of papers linking ACE I/D polymorphism to resistance training, which limits the discussion of our findings.[38,39] It is also relevant to mention the limitations of the present study due to the sample characteristics. It is possible that environmental factors may have influenced the results and hindered a proper association between the variables investigated. Also, more participants in the ACE activity analysis, and in other polymorphisms, could provide better interpretations. In addition, diet and hydration were not rigorously controlled. These facts reinforce the need for future studies.

In summary, chronic reductions of resting BP and PEH after acute sessions during the resistance training were evidenced, which may be interpreted as a protective effect on the cardiovascular system for elderly hypertensive women. Finally, there were no significant associations between PEH and the ACE I/D polymorphism.

Practical applications

Our findings may contribute to promoting chronic reductions on resting BP values and PEH (acute reductions on BP) obtained from resistance exercise training for the hypertensive population. In addition, the progression of loads adopted in the present study (light loads at adaptation period; 60% 1RM at month 2; 70% 1RM at month 3; and 80% 1RM at month 4) may be helpful to professionals who want to guarantee cardiovascular safety and strength gains in a hypertensive elderly population.

Disclosure

The authors report no conflicts of interest in this work.

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