

## ■ ORIGINAL ARTICLES

# Acute effect of resistance exercise on arterial stiffness in healthy young women

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**ABSTRACT:** The purpose of this study was to examine the acute effects of resistance exercise on arterial stiffness in 10 healthy young women. The cardio-ankle vascular index (CAVI) was used as a measure of arterial stiffness. Resistance and aerobic exercises were performed on different days and the change in CAVI from baseline was evaluated at 15 and 30 min after exercise. The CAVI was significantly lower than that at baseline at 15 min after both resistance (change from  $5.5\pm 0.9$  to  $4.9\pm 0.7$ ) and aerobic (change from  $5.7\pm 0.6$  to  $5.2\pm 0.5$ ) exercise. At 30 min after exercise, the CAVI had recovered to baseline values for both types of exercise. Therefore, both resistance and aerobic exercise produced a transient decrease in arterial stiffness, with the effect being dissipated by 30 min after exercise.

**Key words:** resistance exercise; arterial stiffness; cardio-ankle vascular index (CAVI)

## INTRODUCTION

Prolonged aerobic exercise generally reduces arterial stiffness<sup>1)</sup>. This effect is mediated by an elevation in blood nitric oxide (NO) levels, a vascular endothelial cell-derived vasodilator, and decreases in the blood concentration of endothelin, a vasoconstrictor, which, together, reduce the tonus of vascular smooth muscle, thereby increasing central arterial compliance<sup>2)</sup>. On the other hand, an increase in arterial stiffness has been reported after a prolonged program of resistance exercise, resulting from an increase in sympathetic activity<sup>2)</sup>, which

triggers adrenergic vasoconstriction<sup>3)</sup>. Repeated episodes of exercised-induced acute hypertension lead to changes in arterial structure and resistance, which, over time, are likely to result in an overall increase in arterial stiffness<sup>3)</sup>. However, the intensity and duration of resistance exercise needed to induce arterial stiffness are not fully understood.

This issue is clinically important when we consider that prolonged high-intensity resistance exercise is recommended as an effective intervention for the prevention and amelioration of sarcopenia, defined as an aging-related decrease in muscle strength and muscle

mass, and locomotive syndrome<sup>4</sup>). An increase in arterial stiffness and the consequent increase in blood pressure with prolonged resistance exercise, however, could have negative consequences in aging individuals. Therefore, these negative effects of resistance exercise need to be considered to recommend a safe and effective program of exercise to aging individuals. To this end, our aim was to obtain basic data regarding the acute effects of resistance exercise on arterial stiffness in young healthy individuals to determine the short-term time course of these effects.

## SUBJECTS AND METHOD

### Participants

Ten healthy, female, third-year university students with no history of neuro-musculoskeletal disease and no smoking habits and who did not participate in a regular exercise program formed our study cohort (Table 1). All participants provided informed consent. The present study was conducted according to the provisions of the Declaration of Helsinki.

### Study Design and Protocol

This study used a randomized crossover design,

with each participant completing the aerobic and resistance exercise programs on two different days. All participants performed the two exercise programs, with a washout period, for 5 and 7 days. The order of the programs was randomized across participants.

The program of resistance exercise focused on large muscle groups of the trunk and extremities, targeted using the following 6 exercises: chest press, leg press, abdominal crunches, leg curls, latissimus dorsi pull-downs, and knee extension. All exercises were performed using the Body Masters Basix Series (Body Masters Sports Industries Inc., RAYNE, LA, USA). The resistance load was set to 80% of one repetition maximum<sup>5</sup>), with 3 sets of 12 repetitions completed for each exercise, with a short break between sets, over a 40-min duration. Aerobic exercise was performed on a cycle ergometer (AEROBIKE 75XL II, Konami Sports Life, Kanagawa, Japan). Participants cycled at a rate of 50 rpm for 40 min. The resistance load was calculated using the Karvonen method<sup>6</sup>) to maintain a target heart rate of 60% maximum.

### Assessment of Arterial Stiffness

The cardio-ankle vascular index (CAVI) was used as an index of arterial stiffness<sup>7</sup>). A blood

**Table 1. Characteristics of the study participants**

Items	Units	Mean	SD
Age	(years)	20.0	0.7
Body height	(cm)	160.3	4.1
Bodyweight	(kg)	53.5	7.6
Resting systolic blood pressure	(mmHg)	118.8	12.3
Resting diastolic blood pressure	(mmHg)	73.3	7.2
Resting heart rate	(beat/min)	68.5	7.4

n = 10 (all female). SD, standard deviation

pressure and pulse-wave velocity testing device (VeSeraVS-150, FUKUDA DENSHI, Tokyo, Japan) was used for the measurement of CAVI and blood pressure. Blood pressure measurements were obtained by attaching blood pressure cuffs to both upper arms and ankles. The pulse-wave velocity was measured using a heart sound microphone, positioned over the second rib and the sternum, and electrocardiographic electrodes placed on both volar aspects of the wrists. Measurements were obtained in the supine position at three time points: prior to exercise (baseline) and at 15 and 30 min after exercise.

### Statistical Analysis

We first evaluated the change in CAVI from baseline at 15 and 30 min after exercise completion using a repeated measures one-way analysis of variance, with Dunnett's test used to evaluate the effects of the two exercise programs. We then compared the CAVI at baseline and at 15 and 30 min after exercise between the two exercise programs using two-way analysis of variance and Bonferroni's test for multiple comparisons. Statistical analyses were performed using IBM SPSS Statistics 24 (IBM Corp., Armonk, N.Y., USA), with the statistical significance set at 5%.

## RESULTS

Table 2 reports the CAVI at baseline and at 15 and 30 min after exercise completion for both exercise programs. We observed no significant difference in the CAVI at baseline or at the 15 and 30 min time points after exercise completion between the two exercise programs. The CAVI was lower than at baseline at the 15-min time point after exercise for both resistance and aerobic exercise (change from  $5.5 \pm 0.9$  to  $4.9 \pm 0.7$  and from  $5.7 \pm 0.6$  to  $5.2 \pm 0.5$ , respectively;  $p < 0.05$ ). The CAVI recovered to baseline values at the 30-min time point after

exercise, for both types of exercise.

## DISCUSSION

The CAVI was lower than at resting baseline at 15 min after completion of both the resistance and aerobic programs of exercises. The fact that we examined the response to exercise in only healthy participants and after only one bout of each exercise type might explain the absence of the previously reported increase in arterial stiffness after high-intensity exercise<sup>3,8</sup>. A previous study demonstrated that resistance exercise performed at 70% of 1 RM increased blood flow to a level 38× higher than that at rest<sup>8</sup>. This exercise-induced increase in blood pressure is caused by an increase in shear stress on the vascular wall, which affects vascular endothelial function, resulting in increased vascular resistance. This acute effect of exercise on the tonus of the arterial wall would explain the transient increase in the CAVI that we measured 15 min after exercise completion, regardless of the type of exercise. The effects of exercise-induced elevation in muscle temperature and blood levels of the vasodilator NO, previously reported as having a vasoconstrictor effect on endothelial cells of blood vessels after aerobic exercise, may also have contributed to the increase in CAVI at the 15-min time point after exercise.

The recovery of the CAVI to baseline values at 30 min after exercise completion reflects the stress reduction in arterial vessels as the shear stress on the vessel walls dissipates and blood concentrations of NO and the muscle temperatures return to resting levels. Therefore, in healthy young women, the increase in CAVI at 15 min after exercise is a transient phenomenon. The mechanisms underlying this acute, short-term, effect of exercise might be different than those for repeated exercise. Therefore, the effect of repeated bouts of exercise on the CAVI will need to be

**Table 2. Variations of the cardio-ankle vascular index before exercise and at 15 and 30 min after exercise completion**

	Before exercise	15 min after exercise completion	30 min after exercise completion	P value
R.E. group	5.5 (0.9)	4.9 (0.7)*	5.3 (0.7)	< 0.05
A.E. group	5.7 (0.6)	5.2 (0.5)*	5.5 (0.9)	< 0.05

Data are presented as the mean (standard deviation). R.E., resistance exercise; A.E., aerobic exercise. The p-value reported is for the repeated measures one-way analysis of variance. \*Results of multiple comparisons of values measured before exercise and at 15 min after exercise completion ( $p < 0.05$ ).

investigated by sex and by age. Effects of aging on the relationship between exercise and the CAVI will also need to be investigated to accumulate evidence regarding the safety and efficacy of resistance exercise in order to develop evidence-based exercise therapy programs for the prevention and amelioration of sarcopenia and locomotive syndromes.

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