Artery Research (2018) 23, 63–70

Available online at www.sciencedirect.com

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Resistance training-induced decreases in central arterial compliance is associated with increases in serum thromboxane B_2 concentrations in young men

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Received 22 February 2018; received in revised form 12 July 2018; accepted 14 August 2018

KEYWORDS
Resistance training;
Carotid arterial compliance;
Vasoconstrictive mediator;
Platelet aggregation;
Young men

Abstract *Background:* Reduction in central arterial compliance is an independent risk factor for cardiovascular disease, and is caused by high-intensity resistance training. The thromboxane has both potent vasoconstrictive and platelet aggregation effects, and is associated with cardiovascular diseases. However, whether thromboxane is involved in resistance training-induced decrease in central arterial compliance is unclear. The present study aimed to investigate relationships between circulating thromboxane levels and central arterial compliance in both cross-sectional and longitudinal (i.e., resistance training) designs.
Methods and results: First, in a cross-sectional study, we assessed association between circulating thromboxane concentrations and central arterial compliance in 63 young men, who showed significant negative correlation between those parameters. Second, in a longitudinal study, we examined effects of high-intensity resistance training on circulating thromboxane concentrations and central arterial compliance and relationship among changes from baseline in those parameters. Young sedentary men were assigned to control ($n = 7$) or training ($n = 17$) groups. Subjects in training group underwent four-week supervised high-intensity resistance training. Resistance training significantly elevated circulating thromboxane

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https://doi.org/10.1016/j.artres.2018.08.001

concentrations and decreased central arterial compliance; no significant change was observed in control group, and there was significant correlation between changes in those parameters. *Conclusions:* circulating thromboxane is possible mechanism explaining resistance training-induced decrease in central arterial compliance in young men.

Introduction

The guidelines of the American College of Sports Medicine and the American Heart Association recommend moderate- to high-intensity resistance training because resistance training can elicit substantial increases in physical fitness and some health-related factors such as muscle strength, bone mineral density, and insulin sensitivity.[1,2] In contrast, it has been reported that high-intensity resistance exercise and resistance training [≥75% one-repetition maximum (1RM)] decrease central arterial compliance (CAC),[3,4] although low-intensity resistance exercise and resistance training increase arterial compliance[5,6] and moderate-intensity resistance training does not change arterial compliance.[7] Moreover, a meta-analysis revealed that only high-intensity resistance training was significantly associated with a decrease in arterial compliance.[8] It is possible that decreased arterial compliance with both acute and chronic resistance exercise is related to high-intensity and is not found in moderate- and low-intensity exercise. Deteriorated vascular function (i.e., decreased CAC) is an independent risk factor for future cardiovascular diseases.[9] Thus, a decrease in CAC induced by resistance training may increase the future risk for cardiovascular diseases. However, the mechanisms underlying resistance training-induced decrease in CAC have not been clarified yet.

The arterial compliance is regulated by the composition of elastin and collagen (structural elements) and the vasoconstrictor tone exerted by smooth muscle cells (functional elements). The elastin/collagen composition of the arterial wall is a more slowly changing component that contributes to arterial compliance.[10] As such, it is unlikely that this may be a physiological mechanism underlying decrease in arterial compliance induced by short-term resistance training. In contrast, the functional elements are regulated by some vasoconstrictive mediators.[11,12] In particular, among vasoconstrictive mediators, thromboxane (TX) is produced from the platelets and has potent vasoconstrictory and platelet aggregation effects.[13–15] The TX receptor exists on vascular smooth muscle cell.[16] Thus, increased circulating TX concentrations are possibly associated with decreased arterial compliance. In contrast, high-intensity single bout of resistance exercise causes platelet aggregation,[17] which strongly suggests that high-intensity resistance exercise may increase circulating TX concentrations. However, the effect of high-intensity resistance exercise on circulating TX concentrations has not been clarified yet. Moreover, whether increased circulating TX concentrations are associated with resistance training-induced decrease in arterial compliance is unclear.

Accordingly, the aim of the present study was to investigate whether circulating TX concentrations are related to arterial compliance and whether the resistance training-induced decrease in arterial compliance is associated with changes in circulating TX concentrations. We hypothesized that (a) circulating TX concentrations are associated with arterial compliance and (b) resistance training increases circulating TX concentrations, which is associated with a resistance training-induced decrease in arterial compliance. To test our hypothesis, in experiment 1, we examined the relationship between circulating TX concentrations and arterial compliance in a cross-sectional study in young men. In experiment 2, we investigated the effects of a four-week-long resistance training on circulating TX concentrations and arterial compliance in young men.

Materials and methods

Subjects

In experiment 1, 63 young men (age, 20–36 years) were enrolled in a cross-sectional study. In experiment 2, 24 young men (age, 20–35 years) were enrolled in a longitudinal study. Applicants for control and resistance training were recruited as each subject in control (n = 7) and training (n = 17) groups, respectively. All subjects were recruited from the local community through flyers, e-mails, and information sharing. None of the subjects had participated in any resistance or endurance training regularly. All subjects were non-smokers and cardiovascular disease-free, as indicated by their medical history. None of the subjects were taking cardiovascular medications. The subjects were instructed to maintain current eating behaviors for the duration of the intervention. The present study was conducted in accordance with the Declaration of Helsinki and was approved by the ethical committee of the University of Tsukuba. All subjects provided informed written consent.

Sample size estimation

The sample size was calculated based on the previous studies[18,19] that circulating levels of vasoactive substances are associated with arterial compliance (experiment 1) and high-intensity resistance training decreases arterial compliance (experiment 2). Considering a power of 0.80 and an α level of 0.05, in experiment 1, a total sample size of 38 was found to be necessary by "bivariate normal model" using a general stand-alone power analysis program