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Oxidative stress and arterial stiffness in strength- and endurance-trained athletes

Youngju Choi [a], Seiji Maeda [a,*], Takeshi Otsuki [b], Asako Miyaki [a], Nobutake Shimojo [c], Mutsuko Yoshizawa [a], Hitoshi Shiraki [a], Ryuichi Ajisaka [a]

[a] *Division of Sports Medicine, Graduate School of Comprehensive Human Sciences, University of Tsukuba, Ibaraki, Japan*
[b] *Graduate School of Health and Sport Sciences, Ryutsu Keizai University, Ibaraki, Japan*
[c] *Division of Cardiovascular Medicine, Graduate School of Comprehensive Human Sciences, University of Tsukuba, Ibaraki, Japan*

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Abstract *Background:* Endurance exercise training decreases arterial stiffness, whereas high-intensity strength exercise training increases arterial stiffness. However, the mechanisms underlying the adaptations to the 2 types of exercise training remain unclear. Increased oxidative stress induces vasoconstriction and endothelial dysfunction. Plasma advanced oxidation protein products (AOPP)—a novel marker of oxidative stress—have recently been reported to be positively associated with arterial stiffness in healthy subjects. We hypothesized that AOPP are involved in the adaptation of arterial stiffness in different types of exercise training.
Methods: We investigated plasma AOPP concentration and arterial stiffness in strength- and endurance-trained athletes. The subjects included young strength-trained athletes (SA group) (shot put, hammer, or javelin throwers; $n = 12$), endurance-trained athletes (EA group) (long- or middle-distance runners; $n = 10$), and sedentary individuals (Control group) ($n = 12$). We measured aortic pulse-wave velocity (PWV), systemic arterial compliance (SAC), and plasma AOPP concentrations.
Results: PWV was higher in the SA than in the EA or control groups (SA: 6.48 ± 0.47, EA: 6.00 ± 0.67, Control: 5.65 ± 0.52 m/s, mean $\pm$ SD), and SAC was lower in the SA than in the EA or control groups (SA: 1.04 ± 0.24, EA: 1.56 ± 0.44, Control: 1.38 ± 0.35 ml/mmHg); thus, arterial stiffness was higher in the SA group. Plasma AOPP concentrations were higher in the SA group than in the EA group (SA: 31.7 ± 8.5, EA: 23.9 ± 6.9, Control: 27.2 ± 3.9 μmol/l). We found that plasma AOPP levels tended to be related to SAC ($P = 0.073$, $r = -0.31$).
Conclusions: The present study provides a possibility that exercise training-induced oxidative stress may be partly involved in the mechanism underlying the adaptation of arterial stiffness in strength- and endurance-trained athletes.

* Corresponding author. Tel.: +81 29 853 2683; fax: +81 29 853 2986.
E-mail address: maeda@taiiku.tsukuba.ac.jp (S. Maeda).

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Introduction

The central artery acts as a conduit that carries blood to tissues and organs and performs a buffering function to level off fluctuations in blood pressure, which are generated by cardiac pulsation and intermittent blood flow. Increase in arterial stiffness reduces this buffering function and subsequently leads to an increase in blood pressure. Exercise training can affect arterial stiffness. We and other groups showed that endurance exercise training decreased arterial stiffness.[1–3] In contrast, high-intensity strength exercise training may increase arterial stiffness,[3–8] although there are some conflicting reports.[9–11] The mechanisms underlying these different adaptations to the 2 types of exercise training remain unclear.

Increased oxidative stress induces vasoconstriction and endothelial dysfunction.[12–14] Thus, oxidative stress may play an important role in the regulation of vascular function.[15] Plasma advanced oxidation protein products (AOPP)—a novel marker of oxidative stress—have been recently reported to be positively associated with arterial stiffness in healthy subjects.[16] AOPP are formed during oxidative stress by the reaction of plasma proteins with chlorinated oxidants and are novel markers of oxidant-mediated protein damage.[17] Furthermore, AOPP act as mediators of inflammation.[17] Since inflammation is thought to contribute to the process of arterial stiffening,[18] alteration in the plasma AOPP concentration may have important clinical significance in vascular function.

A large number of studies have shown that endurance exercise training leads to decreased oxidative stress,[19–24] although there are some conflicting reports.[25–28] On the other hand, it has been reported that high-intensity strength exercise training increases oxidative stress.[29,30] Therefore, exercise training-induced oxidative stress may play a role in the difference in the adaptation of arterial stiffness observed between strength exercise training and endurance exercise training. Furthermore, the plasma AOPP concentrations may be implicated in arterial stiffness.[16] Thus, we hypothesized that AOPP participates in the adaptation of arterial stiffness to different types of exercise training. The purpose of this study was to investigate plasma AOPP concentration and arterial stiffness in strength- and endurance-trained athletes. We measured aortic pulse-wave velocity (PWV), which is an index of arterial stiffness; systemic arterial compliance (SAC), and plasma AOPP concentrations in strength- and endurance-trained athletes.

Methods

Subjects

The subjects of this study comprised (1) 12 young male strength-trained athletes who were shot put, hammer, or javelin throwers (the SA group), (2) 10 male endurance-trained athletes who were long- or middle-distance runners (the EA group), and (3) 12 sedentary individuals who were used as the controls. All the athletes were members of an intercollegiate track and field team. Members of the SA group had been mainly undergoing high-intensity resistance training on an average of 5.2 ± 0.1 sessions per week (3.1 ± 0.3 h/session); those of the EA group had been mainly undergoing aerobic training on an average of 5.3 ± 0.4 sessions per week (2.7 ± 0.3 h/session); and those of the control group had led a sedentary lifestyle (no regular physical activity) for at least 2 years. None of the subjects exhibited signs or symptoms, or had a history of any chronic diseases. All of them were non-smokers, and none of them were under any medications, anabolic steroids, or antioxidant-containing dietary supplements at the time of the study. The subjects were asked to refrain from alcohol consumption and intense physical activity (exercise) for 24 h and caffeine consumption for 4 h prior to the tests in order to rule out possible acute effects. All measurements were taken at a constant room temperature (25 °C).

The present study was approved by the Ethical Committee of the University of Tsukuba. This study conformed to the principles outlined in the Helsinki Declaration, and all the subjects provided their written informed consent prior to participation in this study.

Aortic pulse-wave velocity

Aortic pulse-wave velocity (PWV) was measured after a minimum resting period of 20 min by applanation tonometry as previously described with minor modifications in the method.[31] In brief, carotid and femoral arterial pulse waves were obtained in triplicate using arterial applanation tonometry incorporating an array of 15 transducers (form PWV/ABI; Colin Medical Technology, Komaki, Japan). The distance between the 2 applanation sites was assessed in triplicate with a random zero-length measurement of the surface of body, using a non-elastic tape. Pulse wave transit time was determined on the basis of the delay in time between the proximal and distal 'foot' waveforms. The foot of the pulse wave was identified as the commencement of the sharp systolic upstroke, which was automatically detected. Aortic PWV was calculated as the distance divided by the transit time and was expressed in metres per seconds.

At the same time as PWV measurement, the resting brachial arterial blood pressure and heart rate were measured in triplicate using oscillometry and ECG (form PWV/ABI; Colin Medical Technology, Komaki, Japan).

Systemic arterial compliance

Systemic arterial compliance (SAC) in the carotid artery was measured by applanation tonometry and Doppler echocardiography as previously described, with minor modifications in the methods.[32,33] In brief, carotid arterial pressure waveforms were obtained by applanation tonometry (form PWV/ABI; Colin Medical Technology, Komaki, Japan) after a minimum resting period of 20 min. At the time of waveform recording, the systolic, diastolic, and mean blood pressure (SBP, DBP, and MBP, respectively) in the brachial arterial were measured by oscillometry (form PWV/ABI; Colin Medical Technology, Komaki, Japan). The pressure signal obtained by tonometry was calibrated by equating the carotid MBP and DBP to the brachial arterial values. SAC was calculated as follows: $SAC = A_d/(dP \times R)$,