Effects of Athletic Strength and Endurance Exercise Training in Young Humans on Plasma Endothelin-1 Concentration and Arterial Distensibility

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Strength exercise training induces a decrease in arterial distensibility, whereas endurance exercise training causes an increase in arterial distensibility. Endothelin-1 (ET-1), which is produced by vascular endothelial cells, has potent vasoconstrictor and proliferative activity on vascular smooth muscle cells. We hypothesized that endogenous ET-1 participates in alteration of arterial distensibility by different exercise training types (i.e., strength and endurance exercise training). The purpose of the present study was to investigate plasma ET-1 concentration and arterial distensibility in strength- and endurance-trained athletes. Subjects were male strength-trained athletes (discus, hammer, or javelin throwers; 22.2 years; SA), male endurance-trained athletes (long- or middle-distance runners; 20.7 years; EA), and sedentary healthy men (20.6 years; sedentary control, SC). Maximum hand-grip strength was markedly greater in SA compared with EA and SC (55.3 vs. 41.1 vs. 40.5 kg, $P < 0.05$). Maximum oxygen uptake was markedly greater in EA than in SA and SC (60.9 vs. 43.1 vs. 43.6 ml/kg/min, $P < 0.05$). Arterial pulse wave velocity (PWV), which is an index of arterial distensibility, was significantly higher in SA than in EA and SC (688 vs. 529 vs. 601 cm/sec, $P < 0.05$). In EA, PWV was significantly lower in comparison to that in SC ($P < 0.05$). Thus arterial distensibility was lower in SA than in EA and SC and higher in EA than in SC. Plasma ET-1 concentration was significantly higher in SA compared with EA and SC (1.64 vs. 1.12 vs. 1.24 pg/ml, $P < 0.05$). Plasma ET-1 concentration tended to be lower in EA than in SC. These results suggest that the difference in plasma ET-1 level may participate in the mechanism underlying different adaptation of arterial distensibility between strength- and endurance-trained athletes. Exp Biol Med 231:789–793, 2006

Key words: pulse wave velocity; endothelial cell; thrower; runner

Introduction

Arterial distensibility is defined by changes of function and structure in artery (1, 2). The critical factors of arterial distensibility are mainly changes and amounts of elastic and collagen fibers and tonus on vascular smooth muscle cells (1, 2). Endurance exercise training induces an increase in arterial distensibility (3–5). On the other hand, it has been reported that strength exercise training decreases arterial distensibility (6, 7). Thus exercise training–induced adaptations in arterial distensibility depend on the type of exercise training. However, the mechanism of difference of these adaptations between two types of exercise training is unclear.

Endothelin (ET)-1 is a potent vasoconstrictor peptide produced by vascular endothelial cells (8–10), and exists in human vascular endothelial cells, having a potent vasoconstrictor effect (8, 11). Haynes and colleagues (12) reported that systemic administration of an ET receptor antagonist significantly decreases blood pressure and systemic vascular resistance in healthy humans. These observations suggest that endogenously generated ET-1 participates in the regulation of vascular tone in humans. Furthermore, ET-1 has potent proliferative activity on vascular smooth muscle cells (8, 9, 13). We previously reported that plasma level of ET-1 was decreased by aerobic exercise training in young and elderly humans (14, 15).

Because ET-1 implicates regulation of vascular tonus and proliferation on vascular smooth muscle cells, we hypothesized that ET-1 participates in the mechanism of

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Table 1. Physical Characteristics of Strength-Trained Athletes, Endurance-Trained Athletes, and Sedentary Control[a]

	Strength ($n = 5$)	Sedentary ($n = 5$)	Endurance ($n = 5$)
Age, years	22.2 ± 0.4	20.6 ± 1.5	20.7 ± 0.3
Height, m	1.77 ± 0.01	1.71 ± 0.02	1.72 ± 0.03
Weight, kg	90.4 ± 5.0	59.8 ± 3.5*	60.1 ± 2.2*
Body mass index, kg/m^2	29.8 ± 1.3	20.3 ± 0.7*	20.3 ± 0.4*
Chest, cm	103 ± 2	85 ± 3*	84 ± 2*
Waist, cm	90 ± 4	71 ± 2*	70 ± 1*
Hip, cm	106 ± 2	92 ± 2*	89 ± 1*
Upper arm, cm	35 ± 1	25 ± 1*	25 ± 1*
Lower arm, cm	30 ± 1	24 ± 1*	23 ± 0*
Thigh, cm	61 ± 2	47 ± 1*	50 ± 1*
Lower thigh, cm	42 ± 1	35 ± 1*	36 ± 1*
Systolic blood pressure, mm Hg	129 ± 3	110 ± 3*	110 ± 1*
Diastolic blood pressure, mm Hg	69 ± 2	61 ± 2*	57 ± 1*
Heart rate, beat/min	61 ± 2	63 ± 3	55 ± 5
Maximal oxygen uptake, ml/kg/min	43.1 ± 1.2	43.6 ± 3.2	60.9 ± 1.4*†
Hand grip strength, kg	55.3 ± 1.4	40.5 ± 2.5*	41.1 ± 3.2*

[a] Strength, strength-trained athletes; sedentary, sedentary control; endurance, endurance-trained athletes. Values are mean ± SE.
*$P < 0.05$ vs. strength, †$P < 0.05$ vs. sedentary.

different adaptation of arterial distensibility by the strength- and endurance-exercise training. The present study investigated whether the plasma ET-1 concentration with arterial distensibility differs between strength- and endurance-trained athletes. To test our hypothesis, we measured plasma ET-1 concentration and arterial pulse wave velocity (PWV), which is a traditional index of arterial distensibility, in strength-trained athletes and endurance-trained athletes.

Materials and Methods

Subjects and Protocol. Five male discus, hammer, or javelin throwers (strength-trained athletes; SA), five male long or middle distance runners (endurance-trained athletes; EA), and five sedentary healthy men (sedentary control; SC) volunteered to participate in the present study. Table 1 shows the physical characteristics of SA, EA, and SC groups. All athletes belonged to a track and field club in a university, and their competitive sport career was longer than 4 years (SA; 6.6 ± 0.8 years, EA; 8.8 ± 1.1 years). The subjects of SC had a sedentary lifestyle for at least for 3 years. All subjects were normotensive (<140/90 mm Hg) and free from the signs, symptoms, and history of any overt chronic diseases. None of the participants had a history of smoking, and none were taking any medications.

Before all measurements, subjects refrained from alcohol consumption and intense physical activity (exercise) for 24 hours and caffeine consumption for 4 hours to avoid the acute effects on arterial distensibility (16–18) and plasma ET-1 concentration (19, 20). Resting blood pressure, resting heart rate, maximal oxygen uptake, hand-grip strength, resting PWV, and resting venous plasma ET-1 concentration were measured in the SA, EA, and SC groups. All measurements were performed at a constant room temperature (25°C).

The present study was approved by the Ethical Committees of the Institute of Health and Sport Sciences of the University of Tsukuba. This study conformed with the principles outlined in the Helsinki Declaration. All subjects gave their written informed consent before inclusion in the study.

Measurement of Maximum Oxygen Uptake. Maximum oxygen uptake was determined incremental cycling to exhaustion (after 3 minutes at 80 W, with 30-W increases every 3 minutes) by monitoring breath-by-breath oxygen consumption and carbon dioxide production (AE280S; Minato Medical Science, Osaka, Japan), heart rate, and ratings of perceived exertion (Borg scale).

Measurement of Hand-Grip Strength. Maximum hand-grip strength was determined using a hand dynamometer (HK51020; SUNCREA Co., Ltd., Tokyo, Japan). Two maximum contractions, each lasting 3–5 seconds and at least 15 seconds apart, were performed in both limbs. The maximum strength score achieved from the two trials was taken as the maximum handgrip strength.

Measurement of Arterial PWV. Subjects were studied in a quiet, temperature controlled room (25°C) after an adequate resting period of at least 20 minutes. PWV was measured according to the previous studies (17, 21) with minor modification. Briefly, carotid and femoral arterial pulse waves were obtained in triplicate using arterial applanation tonometry incorporating an array of 15 transducers (formPWV/ABI; Colin Medical Technology, Komaki, Japan). Distance traveled by the pulse wave was assessed in triplicate with a random zero-length measurement over the surface of the body with a nonelastic tape measure. Pulse wave transit time was determined from the time delay between the proximal and distal "foot" waveforms. The foot of the wave was identified as the commencement of the

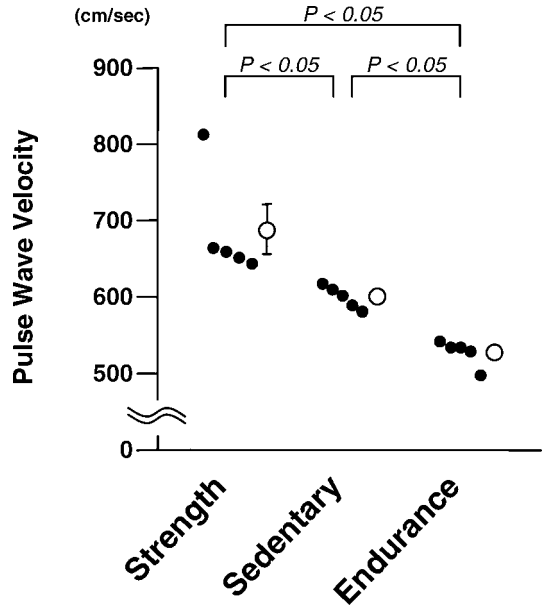

Figure 1. Arterial distensibility in strength-trained athletes (strength, $n = 5$), endurance-trained athletes (endurance, $n = 5$), and sedentary control (sedentary, $n = 5$). Arterial distensibility was estimated by PWV using applanation tonometry. Arterial distensibility is inversely related to PWV. PWV was significantly higher in strength compared with sedentary and endurance, and was significantly lower in endurance than in sedentary. Data are expressed as means and SE.

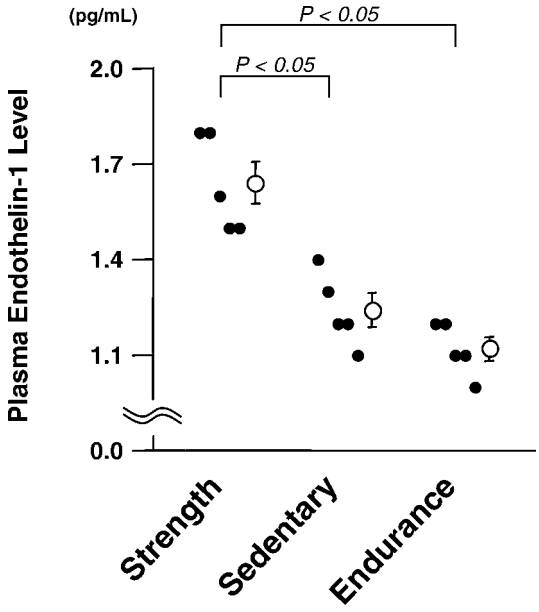

Figure 2. Plasma ET-1 concentration in strength-trained athletes (strength, $n = 5$), endurance-trained athletes (endurance, $n = 5$), and sedentary control (sedentary, $n = 5$). Plasma ET-1 concentration was significantly higher in strength compared with sedentary and endurance. Plasma ET-1 concentration in endurance tended to be lower than in sedentary, but the difference was not statistically significant. Data are expressed as means and SE.

sharp systolic upstroke, which was automatically detected by a band-pass filter (5–30 Hz). PWV was calculated as the distance divided by the transit time.

At the same time as PWV measurement, brachial arterial blood pressure was measured using oscillometry (21) (formPWV/ABI; Colin Medical Technology). Measurement of blood pressure was made in triplicate.

Measurement of Plasma ET-1 Concentration by Sandwich-Enzyme Immunoassay. Each blood sample was placed in a chilled tube containing aprotinin (300 kallikrein-inactivating units/ml) and EDTA (2 mg/ml) and then centrifuged at 2000 *g* for 15 minutes at 4°C. The plasma was stored at −80°C until assay. Plasma concentration of ET-1 was determined using a sandwich-EIA Kit (Immuno-Biological Laboratories, Fujioka, Japan). The assay procedure was carried out, as previously described, by our laboratory (19, 20).

Statistical Analysis. Values are expressed as means ± SE. Statistical analysis was carried out using analysis of variance followed by Fisher's protected least-significant difference test for multiple comparisons. $P < 0.05$ was accepted as significant.

Results

Physical characteristics of the SA, EA, and SC groups are shown in Table 1. There were no significant differences in age and height among the SA, EA, and SC groups. Body weight and body mass index were significantly higher in the SA group compared with the SC and EA groups. The circumstances of chest, waist, hip, arms, and thighs in SA were significantly greater than that in SC and EA. Maximum hand-grip strength was markedly greater in SA compared with SC and EA, whereas there was no significant difference in these parameters between SC and EA. These results indicated that strength-trained athletes exhibited the development of morphologic skeletal muscle and functional muscle power. Maximum oxygen uptake was markedly greater in EA compared with SA and SC. There was no significant difference in maximum oxygen uptake between SA and SC. These results indicated that endurance-trained athletes exhibited the increase in aerobic capacity.

Figure 1 shows PWV in the SA, SC, and EA groups. PWV was significantly higher in SA compared with SC and EA. PWV was significantly lower in EA than in SC. These results indicated that the arterial distensibility is lower in SA than in SC or EA and higher in EA compared with SC.

The plasma ET-1 concentrations in the SA, SC, and EA groups are shown in Fig. 2. Plasma ET-1 concentration was significantly higher in SA compared with SC and EA. Plasma ET-1 concentration in EA tended to be lower than in SC, although the difference was not statistically significant. Additionally, plasma ET-1 concentration was significantly related to PWV in all subjects ($r = 0.67$, $P < 0.05$).

Discussion

We demonstrated that plasma level of ET-1 was significantly higher in strength-trained athletes than in endurance-trained athletes or sedentary men and tended to

be lower in endurance-trained athletes compared with sedentary men. In the present study, arterial distensibility, as evidenced by PWV, was lower in strength-trained athletes than in endurance-trained athletes or sedentary men and higher in endurance-trained athletes compared with sedentary men. ET-1 has potent vasoconstrictor and potent proliferative activity on vascular smooth muscle cells (8–11, 13). It is now well established that vasoconstriction of arteries or proliferation on vascular smooth muscle cells decreases vascular distensibility (2). Thus, it appears that alteration of ET-1 is involved in adaptation of arterial distensibility. Therefore, we considered that the difference in plasma level of ET-1 between strength-trained athletes and endurance-trained athletes may partly participate in the mechanism underlying different adaptation of arterial distensibility.

Both an enhanced vascular constriction (2) and increased blood pressure (3) decrease arterial distensibility. Blood pressure increases with an increased peripheral vascular resistance by ET-1 (12). In the present study, baseline blood pressure in strength-trained athletes was significantly higher compared with endurance-trained athletes and sedentary men. Thus, it is possible that the difference in plasma ET-1 concentration may participate directly (i.e., central arterial constriction) and indirectly (i.e., increase in blood pressure by peripheral vascular constriction) in the mechanism underlying different adaptation of arterial distensibility or baseline blood pressure between strength- and endurance-trained athletes.

The mechanism underlying the increase in plasma ET-1 concentration in strength-trained athletes remains to be elucidated. It has been reported that arterial blood pressure increased to 320/250 mm Hg during a bout of high-intensity strength exercise (22). ET-1 has potent vasoconstrictor and potent pressure action (8–11). Therefore, it is possible that endogenous ET-1 production markedly increases during a bout of strength exercise. The intermittent repetition of strength exercise (i.e., strength exercise training) might cause an elevation of plasma ET-1 level in basal condition in strength-trained athletes. Furthermore, ET-1 has potent proliferative activity on vascular smooth muscle cells (8, 9, 13). Because strength exercise causes the marked increase in blood pressure (22), the thickness of vascular wall may thicken. Therefore, the increase in plasma ET-1 concentration in strength-trained athletes might be a reasonable adaptation to protect the vascular wall from intensive blood pressure elevation during strength exercise.

In the present study, arterial distensibility was lower in strength-trained athletes compared with endurance trained-athletes and sedentary men. Recently, Miyachi and colleagues (6) reported that arterial distensibility decreased by strength exercise training and the decreased arterial distensibility returned to the basal level (level before strength training) after cessation of strength training. Furthermore, we have reported that the plasma level of ET-1 decreased by aerobic exercise training in young humans and the decreased plasma ET-1 level returned to the basal level after cessation of the training (14). Taken together, we considered that alterations of arterial distensibility and plasma ET-1 concentration in strength-trained athletes are physiological adaptation during lifestyle of their competitive sports.

In conclusion, we demonstrated that plasma level of ET-1 differed between strength- and endurance-trained athletes and was significantly higher in strength-trained athletes than in endurance-trained athletes or sedentary men. On the other hand, the plasma level of ET-1 in endurance-trained athletes tended to be lower than in sedentary men. Arterial distensibility was downregulated in strength-trained athletes and upregulated in endurance-trained athletes. We propose that the difference in plasma ET-1 level may participate in the mechanism underlying different adaptation of arterial distensibility between strength- and endurance-trained athletes.

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