Vascular endothelium-derived factors and arterial stiffness in strength- and endurance-trained men

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Otsuki T, Maeda S, Iemitsu M, Saito Y, Tanimura Y, Ajisaka R, Miyauchi T. Vascular endothelium-derived factors and arterial stiffness in strength- and endurance-trained men. Am J Physiol Heart Circ Physiol 292: H786–H791, 2007. First published September 22, 2006; doi:10.1152/ajpheart.00678.2006.—Arterial stiffness is higher in strength-trained humans and lower in endurance-trained humans. However, the mechanisms underlying these different adaptations are unclear. Vascular endothelium-derived factors, such as endothelin-1 (ET-1) and nitric oxide (NO), play an important role in the regulation of vascular tonus. We hypothesized that endogenous ET-1 and NO participate in the adaptation of arterial stiffness in different types of exercise training. The purpose of this study was to investigate plasma ET-1 and NO concentrations and arterial stiffness in strength- and endurance-trained men. Young strength-trained athletes (SA; n = 11), endurance-trained athletes (EA; n = 12), and sedentary control men (C; n = 12) participated in this study. Maximal handgrip strength in SA and maximal oxygen uptake in EA were markedly greater than in C. Aortic pulse-wave velocity, which is an established index of arterial stiffness, was higher in SA and lower in EA than in C. Additionally, we measured systemic arterial compliance (SAC) using carotid artery applanation tonometry and Doppler echocardiography, because arterial stiffness is a primary determinant of compliance. SAC was lower in SA and higher in EA compared with that in C. Plasma ET-1 concentrations were higher in SA compared with C and EA. We did not find significant differences in plasma NO concentrations (measured as the stable end product of NO, i.e., nitrite/nitrate). The relationships of plasma ET-1 concentrations to aortic pulse-wave velocity and SAC were linear. These results suggest that differences in endogenous ET-1 may partly participate in the mechanism underlying different adaptations of arterial stiffness in strength- and endurance-trained men.

arterial compliance; endothelin-1; nitric oxide; pulse-wave velocity

AN INCREASED VASCULAR TONUS enhances arterial stiffness (28, 33). The increased central arterial stiffness reduces arterial buffering function of the pulsation of blood pressure and flow (i.e., arterial compliance). Increased arterial stiffness and reduced arterial compliance implicate in the pathophysiology of cardiovascular disease and have been identified as an independent risk factor for cardiovascular disease (2, 12). In addition, exercise training can affect arterial stiffness. Our laboratory and other groups have reported that arterial stiffness is decreased by endurance training (3, 9, 32) and increased by strength training (1, 21). However, the mechanisms underlying the different adaptations to these two types of exercise training are unclear.

Vascular endothelial cells play an important role in the regulation of vascular activity by producing vasoactive substances, such as endothelin-1 (ET-1) and nitric oxide (NO). ET-1 is a potent vasoconstrictor peptide produced by vascular endothelial cells (22). It has been reported that systemic administration of an endothelin receptor antagonist significantly decreases systemic blood pressure and peripheral vascular resistance in healthy humans, strongly suggesting that endogenously generated ET-1 contributes to basal vascular tonus in humans (7). Previous study has demonstrated that plasma ET-1 concentrations increase in some human cardiovascular diseases (4). We have reported that plasma ET-1 concentrations increase with aging even in normal subjects (18, 23). On the other hand, endurance exercise training decreases plasma ET-1 concentrations (16, 18). NO produced by vascular endothelial cells has a potent vasodilator effect and plays an important role in the regulation of platelet-vessel wall interactions and vascular resistance and growth (24). The plasma NO [measured as the stable end product of NO, i.e., nitrite/nitrate (NOx)] concentrations decrease in some human cardiovascular diseases, e.g., essential hypertension (25), and NO bioavailability may decrease with aging (31). We have previously demonstrated that plasma NO (NOx) concentrations are increased by endurance training in healthy humans (16, 19). It has also been reported that endurance training is associated with improved NO bioavailability in healthy humans (8, 10). Therefore, altered plasma ET-1 and NO concentrations may have important clinical significance for both the physiological and pathological states.

Since ET-1 and NO are implicated in the regulation of vascular tonus, it is reasonable to hypothesize that ET-1 and NO participate in the mechanisms underlying the different adaptations of arterial stiffness to strength and endurance training. However, the relationships of the plasma concentrations of these endothelium-derived factors to the adaptations of arterial stiffness by different types of exercise training remain unclear. The primary aim of this study was to investigate plasma ET-1 and NO concentrations and arterial stiffness in strength- and endurance-trained men. To test our hypothesis, we measured plasma ET-1 and NOX concentrations; aortic pulse-wave velocity (PWV), a traditional index of arterial stiffness; and systemic arterial compliance (SAC) in strength-trained and endurance-trained men.

METHODS

Subjects. Eleven male shot put, hammer, or javelin throwers (strength-trained athletes; SA), twelve male long or middle distance...
runners (endurance-trained athletes; EA), and twelve sedentary (un- 
trained) control men (C) volunteered to participate in this study. All 
of the athletes were intercollegiate athletes belonging to track and 
field teams, and their competitive sports careers were longer than 2 
years. The training volume and intensity in the SA group were 5.1 ± 
0.1 sessions/wk (3.1 h/session) at an average intensity of 15 on the 
rating of perceived exertion scale (i.e., hard). Those in the EA group 
were 5.5 ± 0.3 sessions/wk (2.4 ± 0.3 h/session) and the rating of 
15–17 (i.e., hard–very hard). Athletic training in the SA group con-
sisted of throwing, sprint, and plyometric training and skill practice. 
The SA group had been performing vigorous whole-body weight 
training (3 sessions/wk) in addition to their athletic training. The 
training in the EA group mainly consisted of some kind of 
running training, such as long-distance running and interval training. 
The athletes who had been concurrently and regularly performing 
both types of training (i.e., cross-training) were excluded. The C 
group had a sedentary lifestyle (no regular physical activity) for at 
least 2 years. All subjects were free of signs, symptoms, and history 
of any overt chronic diseases. None of the participants had a history 
of smoking and none were currently taking any medications, anabolic 
steroids, or other performance-enhancing drugs. Before all measure-
ments, subjects refrained from alcohol consumption and intense phys-
ical activity (exercise) for 24 h and caffeine consumption for 4 h to 
avoid immediate (acute) effects.

This study was approved by the Ethics Committee of the Institute of 
Health and Sport Sciences of the University of Tsukuba. This study 
conformed to the principles outlined in the Helsinki Declaration. All 
subjects provided written, informed consent before inclusion in this 
study.

**Blood biochemistry.** All participants were instructed to stop oral 
take, without water, overnight 12 h before blood sampling. Each 
blood sample was placed in a chilled tube containing aprotinin (300 
kallikrein-inactivating Unit/ml) and EDTA (2 mg/ml) and was then 
centrifuged at 2,000 g for 15 min at 4°C. The plasma was stored at 
−80°C until assay. Plasma concentrations of ET-1 were determined 
by using a sandwich-EIA Kit (Immuno-Biological Laboratories, 
Fujio, Japan). The ET-1 assay was carried out as previously de-
scribed by our laboratory (17). The intra-assay coefficients of varia-
tion of the ET-1 assay were 11% in our laboratory (18). Plasma 
concentrations of NOx were determined by using a NO (NO2 
/NO3) assay kit (R&D Systems, Minneapolis, MN). The NOx assay was 
carried out according to the manufacturer’s instructions. Serum con-
centrations of cholesterol, triglycerides, and insulin and plasma con-
centrations of glucose were determined by using the standard enzy-
matic techniques.

**Aortic PWV.** Aortic PWV was measured at constant room temper-
ature (25°C) using applanation tonometry as previously described (5) 
with minor modifications. Briefly, carotid and femoral arterial pulse 
waves were obtained in triplicate using arterial applanation tonometry 
in incorporating an array of 15 transducers (formPWV/ABI; Colin Med-
cial Technology, Komaki, Japan) after a resting period of at least 20 
min. The distance traveled by the pulse waves 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 
notch. Mean blood flow was calculated as a product of the aortic 
cross-sectional area and the mean flow velocity (ImageJ; National 
Institute of Health, Bethesda, MD).

**Maximal handgrip strength and maximal oxygen uptake.** Maximal 
handgrip strength and maximal oxygen uptake were measured after 
blood sampling, and PWV and SAC measurements were taken. Maximal handgrip strength was determined by using a hand dyna-
rometer (HK51020; SUNCREA, Tokyo, Japan). Two maximal con-
tractions, each lasting 3 to 5 s and at least 15 s apart, were performed 
by each hand. The maximal strength score achieved from the two 
trials was taken as the maximal handgrip strength.

Maximal oxygen uptake was determined by using incremental 
cycling to exhaustion (a 3 min at 80 W, with a 30-W increase every 
3 min) 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).

**Statistical analysis.** Data are expressed as means ± SE. Statistical 
analysis was carried out using one-way ANOVA followed by Fisher 
protected least significant differences test for multiple comparisons.
The blood pressure-independent effects of training on PWV and SAC 
were tested by using analysis of covariance (ANCOVA). Stepwise 
regression analyses were used to determine significant, independent 
physiological correlates for each of the arterial stiffness measurements. 
The partial correlation analysis was used to determine whether the 
associations of plasma levels of vascular endothelium-derived factors 
with PWV and SAC were independent of blood pressure. P < 0.05 was 
accepted as significant.

**RESULTS**

Table 1 summarizes the characteristics of all groups. Body 
weight, body mass index (BMI), and body circumferences 
were greater in SA compared with C and EA, although there 
were no significant differences in age and height. Serum HDL 
cholesterol concentrations were higher in EA than in SA. 
Maximal handgrip strength was greater in SA than in C and 
EA. Maximal oxygen uptake was higher in EA compared with 
C and SA. Table 2 shows the hemodynamics of all groups. 
Blood pressure was higher in SA compared with C and 
EA. Maximal handgrip strength and maximal oxygen uptake were measured after 
blood sampling, and PWV and SAC measurements were taken. Maximal handgrip strength was determined by using a hand dyna-
rometer (HK51020; SUNCREA, Tokyo, Japan). Two maximal con-
tractions, each lasting 3 to 5 s and at least 15 s apart, were performed 
by each hand. The maximal strength score achieved from the two 
trials was taken as the maximal handgrip strength.

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**RESULTS**

Table 1 summarizes the characteristics of all groups. Body 
weight, body mass index (BMI), and body circumferences 
were greater in SA compared with C and EA, although there 
were no significant differences in age and height. Serum HDL 
cholesterol concentrations were higher in EA than in SA. Maximal handgrip strength was greater in SA than in C and EA. Maximal oxygen uptake was higher in EA compared with 
C and SA. Table 2 shows the hemodynamics of all groups. 
Blood pressure was higher in SA compared with C and EA. Maximal handgrip strength and maximal oxygen uptake were measured after 
blood sampling, and PWV and SAC measurements were taken. Maximal handgrip strength was determined by using a hand dyna-
meter (HK51020; SUNCREA, Tokyo, Japan). Two maximal con-
tractions, each lasting 3 to 5 s and at least 15 s apart, were performed 
by each hand. The maximal strength score achieved from the two 
trials was taken as the maximal handgrip strength.
VASCULAR ENDOTHELIUM AND ARTERIAL STIFFNESS

Table 1. Characteristics of strength- and endurance-trained men and sedentary control men

<table>
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<th>Sedentary</th>
<th>Endurance</th>
<th>Strength</th>
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<tbody>
<tr>
<td>Age, yr</td>
<td>21.3±0.7</td>
<td>20.0±0.2</td>
<td>21.0±0.5</td>
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<tr>
<td>Height, cm</td>
<td>172±2</td>
<td>171±2</td>
<td>177±1</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>65.7±2.0</td>
<td>61.8±1.5</td>
<td>87.3±1.5†</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>87±1</td>
<td>85±1</td>
<td>103±2‡</td>
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<tr>
<td>Chest, cm</td>
<td>75±1</td>
<td>71±1</td>
<td>90±3†</td>
</tr>
<tr>
<td>Waist, cm</td>
<td>95±1</td>
<td>91±1*</td>
<td>106±2†</td>
</tr>
<tr>
<td>Hip, cm</td>
<td>26.8±0.4</td>
<td>25.5±0.5</td>
<td>35.0±1.3†</td>
</tr>
<tr>
<td>Thigh, cm</td>
<td>50.7±0.7</td>
<td>50.3±0.6</td>
<td>58.9±1.0†</td>
</tr>
<tr>
<td>Total cholesterol, mg/dl</td>
<td>178±9</td>
<td>181±8</td>
<td>170±9</td>
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<tr>
<td>HDL cholesterol, mg/dl</td>
<td>61±4</td>
<td>70±4</td>
<td>51±3†</td>
</tr>
<tr>
<td>LDL cholesterol, mg/dl</td>
<td>103±11</td>
<td>98±6</td>
<td>102±9</td>
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<tr>
<td>Glucose, mg/dl</td>
<td>90±3</td>
<td>90±2</td>
<td>89±3</td>
</tr>
<tr>
<td>Insulin, µU/ml</td>
<td>11.7±1.2</td>
<td>9.0±1.3</td>
<td>9.2±1.4</td>
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<tr>
<td>Maximal oxygen uptake, ml·kg⁻¹·min⁻¹</td>
<td>45.2±1.6</td>
<td>59.4±1.0†</td>
<td>43.0±1.0†</td>
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<tr>
<td>Maximal hand-grip strength, kg</td>
<td>42.6±2.1</td>
<td>42.1±1.5</td>
<td>58.0±1.7†</td>
</tr>
</tbody>
</table>

Values are means ± SE. Sedentary, sedentary control men; Endurance, endurance-trained men; strength, Strength-trained men. *P < 0.05 vs. Sedentary; †P < 0.05 vs. Endurance.

Table 2. Hemodynamics of strength- and endurance-trained men and sedentary control men

<table>
<thead>
<tr>
<th></th>
<th>Sedentary</th>
<th>Endurance</th>
<th>Strength</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure, mmHg</td>
<td>119±3</td>
<td>115±3</td>
<td>128±4†</td>
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<tr>
<td>Mean blood pressure, mmHg</td>
<td>86±2</td>
<td>84±2</td>
<td>90±2</td>
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<td>Diastolic blood pressure, mmHg</td>
<td>66±2</td>
<td>61±2</td>
<td>68±2†</td>
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<tr>
<td>Pulse pressure, mmHg</td>
<td>53±1</td>
<td>54±1</td>
<td>60±2†</td>
</tr>
<tr>
<td>Heart rate, beat/min</td>
<td>65±4</td>
<td>59±3</td>
<td>60±2</td>
</tr>
</tbody>
</table>

Values are means ± SE. *P < 0.05 vs. Sedentary; †P < 0.05 vs. Endurance.

independent of MBP (F = 15.5, and P < 0.05) and DBP (F = 13.8, and P < 0.05). To confirm these relationships, we measured their SAC, because arterial stiffness is a primary determinant of the compliance. The variability of SAC was also shown to be associated with exercise training by using ANOVA (Fig. 1B; F = 9.0, and P < 0.05). SAC correlated statistically independent of MBP (r = −0.36, and P < 0.05) but was not statistically associated with DBP (r = −0.28, and P = 0.103). The effect of exercise training on SAC, which was tested by using ANCOVA, was also independent of MBP and DBP (F = 6.7, and P < 0.05; and F = 6.8, and P < 0.05, respectively). In multiple comparisons, aortic PWV was higher in SA than in C and EA; PWV was also lower in EA compared with C. SAC was lower in SA and higher in EA compared with that in C. Figure 2 shows plasma ET-1 levels in all groups. Plasma ET-1 concentrations were higher in SA compared with C and EA. We did not find the significant relation of plasma ET-1 concentrations to MBP (r = 0.29, and P = 0.086) and DBP (r = 0.26, and P = 0.140). The relationships of plasma ET-1 concentrations to aortic PWV and SAC were linear (Fig. 3). Additionally, partial correlation analysis revealed that plasma ET-1 concentrations were related to aortic PWV, even after adjusting for MBP (r = 0.38, and P < 0.05) and DBP (r = 0.46, and P < 0.05), respectively. We also found that the association of plasma ET-1 concentrations with SAC was statistically independent of MBP (r = −0.34, and P < 0.05) and DBP (r = −0.41, and P < 0.05). We did not find a

Fig. 1. Aortic pulse-wave velocity (A) and systemic arterial compliance (B) in strength-trained athletes (SA), endurance-trained athletes (EA), and sedentary control men (C). Data are expressed as means ± SE. Aortic pulse-wave velocity, an index of arterial stiffness, was higher in the SA group and lower in the EA group compared with that in the C group. Systemic arterial compliance was lower in the SA group and higher in the EA group than in the C group.

Fig. 2. Plasma endothelin-1 concentrations in the SA, EA, and C groups. Data are expressed as means ± SE. Plasma endothelin-1 concentrations were higher in the SA group compared with the EA and C groups.
significant effect of training on plasma NOx concentrations (Fig. 4).

To establish which factors were independent predictors of arterial stiffness in overall population, we performed stepwise regression analyses of age, BMI, blood pressure, and heart rate; serum concentrations of cholesterol, triglycerides, and insulin; and plasma concentrations of glucose, ET-1, and NOx to arterial stiffness. Coefficients of determination ($r^2$) were 0.61 (aortic PWV) and 0.59 (SAC), respectively. The variables that were entered for aortic PWV were plasma ET-1 concentrations, age, SBP, and BMI. Plasma concentrations of ET-1 and serum concentrations of HDL and LDL cholesterol and insulin were entered for SAC.

**DISCUSSION**

It is now well established that increased vascular tonus and proliferation of vascular smooth muscle cells increase arterial stiffness and consequently decrease arterial compliance (28, 33). ET-1 has potent vasoconstrictor activity and potent proliferative effects on vascular smooth muscle cells (22). Furthermore, it has been reported that arterial stiffness was increased by intra-arterial infusion of ET-1 and decreased by the administration of ET-1 receptor antagonist (20, 34). These findings suggest that endogenous ET-1 participates in the regulation of arterial stiffness. In this study, we found that aortic PWV was higher and SAC was lower in strength-trained men and that aortic PWV was lower and SAC was higher in endurance-trained men. We have also demonstrated that plasma concentrations of ET-1 were significantly higher in strength-trained men than in endurance-trained or sedentary men. The relationships of aortic PWV and SAC to plasma ET-1 concentrations were linear. Furthermore, stepwise regression analysis and partial regression analysis revealed that the associations of plasma ET-1 concentrations with aortic PWV and SAC were independent of blood pressure. These results suggest that differences in plasma ET-1 levels may be involved in the mechanism underlying adaptations of arterial stiffness to different types of exercise training.

Previous studies have reported the effects of ET-1 infusion, endothelin receptor blockade, and inhibition of NO production on arterial stiffness (11, 20, 30, 34, 35). Recently, we have demonstrated that the inhibition of NO production appeared to have no effect on the acute decrease in middle-sized arterial stiffness with low-intensity, short-duration exercise, although its decrease was induced mainly by regional factors (30). Wilkinson et al. (35) have reported that PWV increased by only 3% during the intra-arterial infusion of NO synthase inhibitor (10 μM/min). In the same experimental setting, they have also demonstrated that the administration of ET-1 (10 pM/min) increased PWV by 12% and that treatment with endothelin A receptor antagonist (40 nM/min) reduced PWV by 12% (20). These findings suggest that ET-1 may be a more important factor to the regulation of arterial stiffness than NO (20). In the present study, we showed the increased arterial stiffness and plasma ET-1 concentrations in strength-trained men. On the basis of the results from past studies plus the present results, we propose that the higher ET-1 production in strength-trained men increases arterial stiffness.

The mechanism underlying the increased plasma ET-1 concentrations in strength-trained men remains unclear. It has been reported that arterial blood pressure increases to 340/240 mmHg during bouts of high-intensity strength exercise (15). After such high-intensity strength exercise, arterial stiffness increases acutely for at least 30 min, although arterial blood pressure rapidly returns to the preexercise basal level (6). Since ET-1 has potent vasoconstrictor and pressor activities (22), it is possible that endogenous
ET-1 participates in increased blood pressure and arterial stiffness during and following high-intensity strength exercise. The intermittent repetition of high-intensity strength exercises (i.e., strength training) might cause an elevation of basal plasma ET-1 levels in strength-trained men.

The conclusions drawn in this study come from examination of plasma ET-1 concentrations. It is generally accepted that ET-1 acts predominantly in autocrine and paracrine manners, and the secretion of ET-1 by endothelial cells is polarized toward the underlying vascular smooth muscle (22). Consequently, plasma levels are largely the result of spillover from the vascular endothelium into the bloodstream. Since only ~20% of generated ET-1 is secreted intraluminally (36), plasma ET-1 concentrations are very low. However, the plasma levels of ET-1 are associated with disease severity (4) and increase with aging even in normal subjects (18, 23). Therefore, the alteration of plasma ET-1 concentrations may have important clinical significance in physiology and/or pathophysiology. Taken together, these data suggest that the differences in plasma ET-1 concentration may be involved in the different adaptations of arterial stiffness to strength training and endurance training.

Increased arterial stiffness implicates in the pathophysiology of cardiovascular disease (2, 12). In this study, arterial stiffness was higher and arterial compliance was lower in strength-trained men compared with endurance-trained men and sedentary control men. Additionally, blood pressure was also higher in the strength-trained men. It is possible that the increased arterial stiffness is one of the physiological adaptations to the intense increase in blood pressure during strengthening exercise. However, at resting conditions, it is unlikely that the increased arterial stiffness and blood pressure are beneficial to vessels and heart. The Bogalusa Heart Study has reported that intima-media thickness of carotid arteries in healthy young adults was associated with the cumulative burden of cardiovascular risk factors since childhood (13). The Young Finns Study has identified an association between school-age risk variables and intima-media thickness of carotid arteries at 36 yr, even after adjustment for contemporaneous risk variables (27). Recently, Miyachi et al. (21) have reported that the decreased arterial compliance caused by strength training dissipated to the basal level (the level before strength training) 2 mo after cessation of strength training in young healthy men. Furthermore, we have reported that the decreased plasma ET-1 levels observed in endurance training returned to the basal level 2 mo after cessation of training (16). Therefore, we believe that increased arterial stiffness and plasma ET-1 concentrations in strength-trained men are physiological adaptations to their competitive sports.

The present investigation has the following study limitations. First, blood pressure in strength-trained men was higher than in endurance-trained men and sedentary peers. This might influence the main results in this study. However, in this study, we performed statistical analysis in consideration of the differences in blood pressure. ANCOVA revealed that the effects of exercise training on arterial stiffness were statistically independent of blood pressure. Additionally, stepwise regression analysis was used to determine the blood pressure-independent association between plasma ET-1 concentrations and arterial stiffness. Although the sample size in the present study may not be enough to use this method, the results of partial correlation analysis did not conflict with that of stepwise regression analysis. The conceivable next steps would be to compare arterial stiffness and plasma ET-1 concentrations between blood-pressure-matched groups to determine the independent role of ET-1 on arterial stiffness. Second, it is difficult to identify which arteries determine the value of SAC. Stergiopulos et al. (29) has reported that the aortic occlusion at the aortic trifurcation did not change SAC but that the occlusion at the level of proximal descending aorta induced a 40% reduction of SAC. Thus it is considered that compliance of the descending aorta at least participates in SAC and that ascending aorta and/or the ramified arteries such as carotid artery would also be implicated in SAC.

In conclusion, the present study has demonstrated that plasma concentrations of ET-1 were significantly higher in strength-trained men than in endurance-trained or sedentary men. We have also demonstrated that arterial stiffness was increased in strength-trained men and reduced in endurance-trained men. The association of arterial stiffness with plasma ET-1 concentrations was linear and statistically independent of blood pressure. We propose that differences in plasma concentrations of ET-1 may partly participate in the mechanism underlying different adaptations of arterial stiffness to strength and endurance training.

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