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Relationship between arterial stiffness and athletic training programs in young adult men

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ABSTRACT

Background: We examined the relationships of endurance- and strength-exercise training and the adolescent duration of training to arterial stiffness in young adult men. We hypothesized that young adults participating in endurance sports would decrease arterial stiffness, whereas those in strength-based sports would increase arterial stiffness. Additionally, we predicted that these trends would be more pronounced with increase in the duration of sport participation. **Methods & Results:** Subjects were male endurance-trained men with short (current age, 20yrs; age at beginning of competitive sport, 15yrs; sport careers, 5yrs; *n*=7, S-ET) and long (20yrs; 12yrs; 8yrs; *n*=7, L-ET) competitive sport careers, strength-trained men with short (20yrs; 16yrs; 4yrs; *n*=7, S-ST) and long (22yrs; 15yrs; 7yrs; *n*=7, L-ST) careers, and sedentary control men (age, 20yrs; *n*=7, C). The exercise training was associated with aortic pulse wave velocity (PWV), a traditional index of arterial stiffness, and the associations were statistically independent of blood pressure. Aortic PWV was lower in L-ET than C and ST. Aortic PWV in L-ST was greater than that of C. The associations of exercise training with systemic arterial compliance (SAC), which inversely correlates with arterial stiffness, were also positive and blood pressure-independent. SAC was greater in ET groups compared to C and ST groups. SAC in L-ST was lower than in C. **Conclusion:** These results suggest that changes in arterial stiffness associated with different training programs appear in young adults as in older humans, and these changes may begin in adolescence.

KEYWORDS

Adolescence; Artery; Compliance; Exercise; Young adults

INTRODUCTION

Increased arterial stiffness is an independent risk factor for the development of atherosclerosis and cardiovascular disease[1,2]. Additionally, increased arterial stiffness has been implicated in the development and progression of hypertension, left ventricular hypertrophy, myocardial infarction, and congestive heart failure[3]. Life style modifications, *e.g.*, sodium restriction, improve arterial stiffness and may reduce the risk of developing adverse complications[4]. Furthermore, we and others have demonstrated that endurance exercise training decreases arterial stiffness in young adults and older humans[5-9], whereas strength exercise training increases arterial stiffness[9-13]. However, the effects of adolescent exercise training on the arterial stiffness of young adults remain unclear.

We examined the relationship between both the type of exercise training (endurance or strength training) and the duration of training and arterial stiffness in young adult men. We hypothesized that young adults participating in endurance sports would decrease arterial stiffness, whereas those in strength-based sports would increase arterial stiffness. Additionally, we predicted that these trends would be more pronounced with increase in the duration of sport participation. Accordingly, we measured the aortic pulse wave velocity (PWV) and systemic arterial compliance (SAC) in young adult (from 19 to 23 years old) endurance- and strength- trained men participating in competitive sports from adolescence (from 12 to 18 years). The study subjects were divided into short- and long-career groups based on the median value of the respective sports careers duration, and arterial stiffness was compared.

METHODS

Subjects. Fourteen male long or middle distance runners (endurance-trained men, ET), 14 male shot put, discus, hammer, or javelin throwers (strength-trained men, ST), and 7 sedentary (untrained) healthy men (sedentary control; C) volunteered to participate in this

study. All of the trained men were intercollegiate athletes belonging to track and field teams. ST had been performing vigorous weight training (3 sessions per week) in addition to their athletic training. Both ST and ET were divided into short (S-ST and S-ET) and long (L-ST and L-ET) sport career groups, with the dividing line set at the median value (4.5 and 7.0 years) of sport careers duration. Athletes concurrently performing both types of training (*i.e.*, cross-training) on a regular basis were excluded. C was recruited through advertisements, and had sedentary lifestyles (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. Before all measurements, subjects refrained from alcohol consumption and intense physical activity (exercise) for 24 hours and caffeine consumption for 4 hours to avoid acute effects.

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 to the principles outlined in the Helsinki Declaration. All subjects gave their written informed consent before inclusion in this study.

Pulse wave velocity. Aortic PWV was measured at constant room temperature (25°C) as previously described by our laboratory[9]. Briefly, carotid and femoral artery pulse waves were obtained in triplicate using artery applanation tonometry incorporating an array of 15 transducers (formPWV/ABI; Colin Medical Technology, Komaki, Japan) after a resting period of at least 20 minutes. The distance traveled by the pulse waves were 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. PWV was calculated as the distance divided by the transit time.

Systemic arterial compliance. SAC was measured according to the previous studies with minor modifications[7,14]. Briefly, carotid artery pressure waveforms were obtained using applanation tonometry (formPWV/ABI; Colin Medical Technology) after a resting period. At the time of waveform recording, brachial arterial blood pressure was measured using oscillometry (formPWV/ABI; Colin Medical Technology). The pressure signal obtained by tonometry was calibrated by equating the carotid mean arterial and diastolic blood pressure to brachial artery values. SAC was calculated as follows:

SAC = Ad/(dP x R)

Where Ad is the area under an arbitrary portion of the diastolic pressure waveform, and dP is the pressure change in this portion. R is total peripheral resistance given as mean blood pressure divided by mean blood flow. Mean blood flow was measured using a Doppler-echocardiographic system as previously described by our laboratory (EnVisor; Koninklijke Philips Electronics, Eindhoven, Netherlands)[15]. The insertion point of the aortic valve tips at end-diastole was defined by two-dimensional imaging in the parasternal long axis view with a 3.5 MHz transducer, and the M-mode echocardiogram at that level was stored into the computer. Doppler ultrasonographic flow velocity curves in the ascending aorta were simultaneously recorded using a 1.9 MHz probe held in the suprasternal notch. Mean blood flow was calculated as a product of the aortic cross-sectional area and the mean flow velocity (Image J; NIH, Bethesda, MD).

Maximal oxygen uptake and maximal hand-grip strength. Maximal hand-grip strength and maximal oxygen uptake were measured after the measurements of PWV and SAC. Maximal hand-grip strength was determined using a hand dynamometer (HK51020; SUNCREA, Tokyo, Japan). Two maximal contractions, each lasting 3 to 5 seconds and at least 15 seconds apart, were performed by each hand. The maximal strength score

achieved from the 2 trials was taken as the maximal hand-grip strength.

Maximal oxygen uptake was determined 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 uptake and carbon dioxide production (AE280S; Minato Medical Science, Osaka, Japan), heart rate, and ratings of perceived exertion (Borg scale).

Serum cholesterol and triglycerides level. All participants were instructed to stop oral intake, without water, overnight 12 hours before sampling of blood. Serum concentrations of total-, HDL- and LDL- cholesterol and triglycerides were determined using the standard enzymatic techniques.

Statistical analysis. Data are expressed as means±SE. The differences in physiological characteristics among groups were tested using ANOVA. The comparisons of arterial stiffness between the groups were assessed by covariance analysis (ANCOVA) model that included systolic blood pressure and heart rate as covariate. Fisher's PLSD test was used for multiple comparisons. After Cook's D for influence was examined with the dividing line set at 0.5, correlation analyses were performed separately within the 2 exercise groups. $P<0.05$ was accepted as significant.

RESULTS

We divided subjects into groups based on the type of sport played (strength or endurance) and the duration of sport career (long or short), and compared the characteristics of these groups (Table 1). The mean duration of sport participation was approximately 3 years longer in the long career groups than the short career groups. Maximal hand-grip strength was greater in the ST groups than the C and ET groups. The absolute degree of maximal oxygen uptake was higher in both types of trained men, but the

body weight-corrected value was greater in the ET groups compared to both the C and ST groups. The weight, body mass index (BMI), chest, waist, hip, waist/hip ratio, and blood pressure were higher in the ST groups compared to the C and ET groups. Differences in serum total- and LDL-cholesterol and triglycerides levels were not present among the groups (Table 2). Serum concentrations of HDL-cholesterol in L-ET was greater compared to C and L-ST (Table 2).

We next measured aortic PWV, a traditional index of arterial stiffness, in all groups (Fig. 1A). When aortic PWV values were compared using ANCOVA that separately included systolic blood pressure and heart rate as covariate, exercise training was associated with aortic PWV (F=6.7, P<0.0001 and F=10.4, P<0.0001, respectively). The significant association between aortic PWV and training programs existed also after adjustment for both systolic blood pressure and heart rate (F=5.5, P=0.0022). In multiple comparisons, the aortic PWV was lower in L-ET than C, and the aortic PWV in L-ST was higher than that of C. The aortic PWV of the ST groups was higher than that of the ET groups. We also measured SAC, which is inversely related to arterial stiffness (Fig. 1B). Once again, there were differences between the groups when systolic blood pressure (F=13.7, P<0.0001), heart rate (F=12.4, P<0.0001), and both indices (F=7.4, P<0.0001) were included as covariate. Compared to the control group, SAC was greater in the ET groups and lower in L-ST. Additionally, the SAC of the ET groups was higher than that of the ST groups. Since BMI was higher in the ST groups, we also used BMI as covariate of ANCOVA. There were no significant associations of exercise program with the aortic PWV and SAC when BMI was entered as covariate.

After the Cook's D for influence was tested, we performed correlation analyses. The correlation coefficients (*i.e.*, r) between PWV and the duration of sport careers were 0.53 in the ST group (n=14, P=0.05) and 0.39 in the ET athletes (n=14, P=0.16). Those between SAC and the duration of sport careers were 0.33 (n=12, P=0.29) and 0.31 (n=13, P=0.30),

respectively.

DISCUSSION

We identified that the aortic PWVs of endurance- and strength-trained young adult men were lower and higher, respectively, compared to sedentary, healthy age-matched subjects. Furthermore, the SAC was greater in endurance-trained men and lower in strength-trained men than in sedentary peers. These associations were independent of systolic blood pressure and heart rate. Thus, overall, arterial stiffness was lower in endurance-trained men and higher in strength-trained men compared to sedentary healthy men. In addition, although the correlation coefficients were not strong and we could not rule out the variation in BMI, the differences in aortic PWV and SAC between sedentary men and both types of exercise trained men were more pronounced as the time of athletic participation increased. Thus, changes in arterial stiffness associated with different exercise training programs appear in young adults, and they may arise even in adolescents.

We and others previously reported that regular endurance exercise decreases[5-9] and regular strength exercise increases arterial stiffness[9-13]. Recently, greater emphasis has been placed on physical activity for cardiovascular health promotion in schools[16]. In the present study, we showed that changes in arterial stiffness are associated with endurance or strength training begun in adolescence, and such adaptations continue through young adulthood. Thus, our present results indicate that competitive sport careers begun in adolescence and continued can be associated with the changes in arterial stiffness.

Ferreira *et al.* reported that the level of maximal oxygen uptake in school age adolescents (13-16 years) was independently associated with carotid artery intima-media thickness (IMT) at middle age (36 years)[17]. Additionally, changes in maximal oxygen uptake from school to middle age were inversely associated with carotid, brachial and

femoral artery stiffness at middle age[18]. The present data demonstrate that arterial stiffness in endurance-trained young adults is lower than that of their sedentary peers, and changes in arterial stiffness is associated with training begun at school age. Taken together, these data suggest that endurance training in school-age youths decreases arterial stiffness, and continued endurance training should maintain this decrease and/or exert additive effects.

In the present study, PWV and blood pressure in strength-trained men with long sports careers was higher than in sedentary healthy men, whereas it was lower than that seen in older adults or hypertensive patients in previous studies[19]. To the best of our knowledge, it is unclear whether arterial stiffness is a predictor of cardiovascular disease also in strength-trained athletes. Although it is possible that the increased arterial stiffness is one of the physiological adaptations to the intense blood pressure increase during strength-based sports, the increases in arterial stiffness can be unfavorite adaptation for heart and vessels at resting condition. However, the type of strength training may also affect arterial stiffness; one study suggests that arterial stiffness may increase less in individuals performing eccentric strength training compared to concentric strength training[20]. Additionally, the present results may have limited applicability to specific athletes. Recently, we have reported that localized leg strength training may increase endothelial function without inducing aortic stiffening in older adults[21]. In general people (*i.e.*, non-specialty athletes), endurance training, which decreases arterial stiffness, is often performed in parallel with strength-training (*i.e.*, cross-training). It was demonstrated that regular rowing exercise, which includes components of aerobic endurance and muscular strength, may decrease arterial stiffness[22]. Intervention study has also reported that cross-training does not stiffen elastic arteries[23]. Taken together, it is possible that strength-based sports performed in parallel with endurance training could improve or, at least, does not increase arterial stiffness. The health organizations have recommended

resistance training based on the documented associations with metabolic risk factors as well as with the attenuation of osteoporosis and sarcopenia[24,25]. Also, we have showed that heart rate recovery immediately after exercise, which has been known to be a potent index of cardiovascular risk, is faster in the strength-trained athletes[26]. It is possible that strength exercise training may have unknown other positive effects even on cardiovascular system. We consider that the present findings should not discourage adolescents and young adults from participating in regular strength-based sports.

In the present study, BMI in sedentary control men and endurance-trained men was optimal, and there was no significant difference between the two groups. Thus, it is likely that BMI was not significantly related to the lower arterial stiffness in endurance-trained men, although we can not negate the relationships between BMI and the effects of adipose tissue on the individual level of artery stiffness. On the other hand, BMI was higher in the strength-trained athletes, and the adjustment for BMI removed the observable associations of sport careers with arterial stiffness. It is possible that the greater adipose tissue might increase arterial stiffness in the strength trained men. However, BMI is a poor measure of adiposity in this population because strength exercise training increases muscle mass and muscle fiber is heavier than adipose tissue. Indeed, the serum lipid profile, except for HDL-cholesterol, did not differ between groups. In the previous study, Miyachi et al has demonstrated that arterial stiffness is higher in strength-trained men by using the height and weight-matched groups[11]. Additionally, the intervention study has also reported that strength training did not increase body fat but it increased arterial stiffness[13]. It is difficult to completely rule out the effects of confounders. However, on the basis of the results from past studies plus the present results, it would be reasonable to consider that sport programs are, at least partly, associated with the changes in arterial stiffness by the adipose tissue-independent pathway.

The mechanism underlying the differences in arterial stiffness between endurance-

and strength-trained men remains 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). Endurance training decreases the plasma concentration of ET-1, a potent vasoconstrictor peptide[27,28], and it is also associated with increased plasma level of NO, an endothelium-derived relaxing factor, and NO bioavailability[27,29-32]. Therefore, it is possible that changes in ET-1 and NO production caused by exercise training could promote differential changes in arterial stiffness. Alternatively, strength training could cause increases in plasma norepinephrine concentrations[33,34] leading to chronically elevated sympathetic adrenergic vasoconstrictor tone and associated arterial stiffness.

The present investigation has the following study limitations. First, although the intergroup differences in BMI were existed, the correlation coefficients between the arterial stiffness measures and the sport careers duration were not strong. BMI was higher in strength-trained groups and there were no significant associations of exercise program with the aortic PWV and SAC when BMI was entered as covariate of ANCOVA model. The adjustment for BMI may attenuate these correlation coefficients. However, both the greater skeletal muscle mass and the higher fat mass would participate in their higher BMI. Also, fat mass in sedentary men could be higher compared with endurance-trained groups, whereas we did not find a significant difference in BMI between endurance-trained athletes and sedentary men. The sample size was small to examine the association between BMI and the sports career-related differences in arterial stiffness by using the multiple regression analysis. It would be of significance to examine the relations of these differences to fat mass and/or skinfold thickness using a large sample size. Second, we can not say that PWV and SAC mean the completely same thing for heavily muscled athletes as sedentary control and endurance-trained men. However, the results in the present study (*i.e.*, the increased arterial stiffness in strength-trained men) were not conflict

with the previous studies which measured carotid arterial compliance and β-stiffness index by using ultrasonography[10,11]. Moreover, the assessment of arterial stiffness in strength-trained men by SAC is consistent with that by β-stiffness index[12]. Thus, we consider that PWV and SAC are available also in heavily muscled athletes, although we can not negate that the muscularity could have artifactual effects also on the reported results by ultrasonography. Finally, the exercise form is different between endurance-trained athletes and strength-trained athletes. Thus, the specificity of each sport (endurance-trained athletes: a long or middle distance running, strength-trained athletes: an athletic throw) might influence the differences between the groups. For example, the contribution of arm is greater in the throw than the running although both sports use whole body. This would result in the differences in central hemodynamics during respective sport. It is possible that the sport types (endurance or strength) are not the only reason to explain the differences in arterial stiffness.

The Bogalusa Heart Study reported that carotid artery IMT in healthy young adults was associated with the cumulative burden of cardiovascular risk factors since childhood[35]. In addition, the Young Finns Study identified an association between school-age (from 12 to 18 years) risk variables and IMT at 36 years even after adjustment for contemporaneous risk variables[36]. Based on these and other previous reports, the American Heart Association has recommended cardiovascular health promotion in schools, and physical activity is an essential component of these recommendations[16]. However, the reported benefits of physical activity on blood pressure and serum cholesterol concentration are primarily limited to hypertensive or overweight adolescents[37], and further studies are required to examine the effect of physical activity and health promotion in schools. We demonstrated that long-term physical activity beginning in adolescence is associated with changes in arterial stiffness, an independent risk factor for atherosclerosis and other cardiovascular disease. The present data supports emphasizing endurance training as an

essential component of cardiovascular health promotion in schools, although further studies are needed to adequately address the role of strength training in such programs.

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FIGURE LEGENDS

Figure 1. Pulse wave velocity (A), a traditional index of arterial stiffness, and systemic arterial compliance (B), which is inversely related to arterial stiffness, in endurance- and strength-trained men. Data are expressed as means±SE. $^{*}P<0.05$ vs sedentary control; $^{\dagger}P<0.05$ vs sport career-matched endurance-trained men.

Table 1. Physiological characteristics of endurance-, and strength-trained, and sedentary men.

	Sedentary Men	Endurance-trained Men		Strength-trained Men		*F*-value
		Short Career	Long Career	Short Career	Long Career	
n	7	7	7	7	7	
Age, years	20.1 ± 0.6	19.9 ± 0.3	20.4 ± 0.5	19.7 ± 0.4	21.6 ± 0.6	2.2
Age at beginning of sport career, years		14.7 ± 0.7	12.1 ± 0.1‡	16.0 ± 0.4†	14.6 ± 0.5†‡	11.5$^{\#}$
Sport careers, years		5.1 ± 0.6	8.3 ± 0.5‡	3.7 ± 0.2†	7.0 ± 0.5‡	18.1$^{\#}$
Height, cm	173 ± 1	174 ± 2	171 ± 2	173 ± 2	179 ± 1	2.5
Weight, kg	66.2 ± 2.7	65.7 ± 2.6	60.5 ± 1.6	82.6 ± 3.8*†	92.5 ± 4.5*†‡	17.8$^{\#}$
Body mass index, kg/m^2	22.0 ± 0.7	21.8 ± 0.5	20.7 ± 0.4	27.5 ± 1.0*†	28.8 ± 1.2*†‡	21.0$^{\#}$
Chest, cm	87 ± 1	87 ± 2	85 ± 2	99 ± 2*†	105 ± 2*†‡	23.5$^{\#}$
Waist, cm	75 ± 1	73 ± 1	71 ± 1	86 ± 2*†	92 ± 4*†	15.0$^{\#}$
Hip, cm	94 ± 2	94 ± 1	89 ± 1	104 ± 2*†	107 ± 2*†	19.2$^{\#}$
Waist/Hip	0.79 ± 0.01	0.78 ± 0.01	0.79 ± 0.01	0.83 ± 0.01*†	0.85 ± 0.03*†	3.8$^{\#}$
Systolic blood pressure, mmHg	116 ± 2	121 ± 4	110 ± 1‡	127 ± 3*	131 ± 5*†	6.4$^{\#}$
Diastolic blood pressure, mmHg	63 ± 2	64 ± 2	57 ± 1‡	67 ± 3	70 ± 2*†	4.8$^{\#}$
Mean blood pressure, mmHg	83 ± 2	88 ± 3	80 ± 1‡	90 ± 3	94 ± 4*†	4.6$^{\#}$
Pulse pressure, mmHg	53 ± 1	57 ± 3	53 ± 0	60 ± 1*	62 ± 3*†	3.9$^{\#}$
Heart rate, bpm	61 ± 3	58 ± 3	53 ± 3	58 ± 3	62 ± 1	1.4
Maximal hand-grip strength, kg	43.7 ± 3.2	43.7 ± 1.1	40.9 ± 2.4	58.5 ± 2.6*†	55.5 ± 1.0*†	12.8$^{\#}$
Maximal oxygen uptake, L/min	2.99 ± 0.46	3.90 ± 0.35*	3.64 ± 0.37*	3.64 ± 0.42*	3.78 ± 0.30*	5.8$^{\#}$
Maximal oxygen uptake, mL/kg/min	45.1 ± 1.3	59.5 ± 1.2*	60.1 ± 1.1*	44.1 ± 1.0†	41.2 ± 1.4*†	54.8$^{\#}$

$^{*}P<0.05$ vs Sedentary men; $^{\dagger}P<0.05$ vs sport career-matched endurance-trained men; $^{\ddagger}P<0.05$ vs short sport career group; $^{\#}$statistical significance of *F*-value ($P<0.05$).

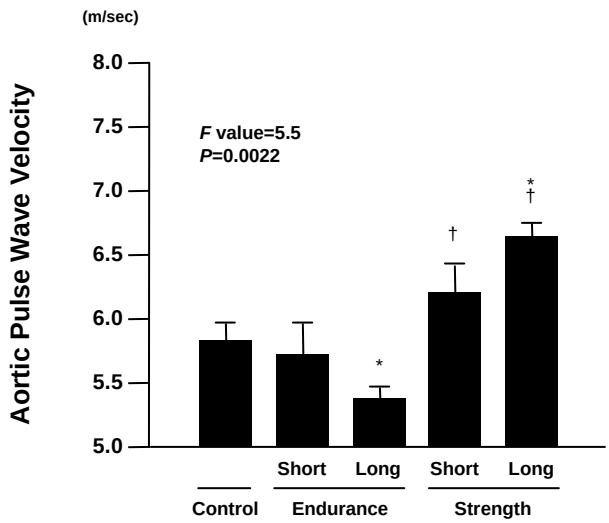
Table 2. Serum lipid profile of endurance-, and strength-trained, and sedentary men.

	Sedentary Men	Endurance-trained Men		Strength-trained Men		*F*-value
		Short Career	Long Career	Short Career	Long Career	
n	7	7	7	7	7	
Total cholesterol, mg/dL	156 ± 10	181 ± 9	180 ± 12	185 ± 9	164 ± 11	1.6
LDL cholesterol, mg/dL	83 ± 10	99 ± 10	96 ± 8	112 ± 12	99 ± 10	1.1
HDL cholesterol, mg/dL	55 ± 3	64 ± 4	74 ± 5[*]	57 ± 4	46 ± 2[†]	7.0[#]
Triglycerides, mg/dL	61 ± 4	92 ± 21	60 ± 18	92 ± 16	107 ± 15	1.8

[*]*P*<0.05 vs Sedentary men; [†]*P* <0.05 vs sport career-matched endurance-trained men; [#]statistical significance of *F*-value (*P*<0.05).

Figure 1
(AJH-D-06-0035)

A



B