

Relationship Between Arterial Stiffness and Athletic Training Programs in Young Adult Men

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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 have decreased arterial stiffness, whereas those in strength-based sports would have increased arterial stiffness. In addition, we predicted that these trends would be more pronounced with an increase in the duration of sport participation.

Methods: Subjects were male endurance-trained men with short (current age, 20 years; age at beginning of competitive sport, 15 years; sport careers, 5 years; $n = 7$, S-ET) and long (current age, 20 years; age at beginning of competitive sport, 12 years; sport careers, 8 years; $n = 7$, L-ET) competitive sport careers, strength-trained men with short (current age, 20 years; age at beginning of competitive sport, 16 years; sport careers, 4 years; $n = 7$, S-ST) and long (current age, 22 years; age at beginning of competitive sport, 15 years; sport careers, 7 years; $n = 7$, L-ST) careers, and sedentary control men (aged, 20 years; $n = 7$, C).

Results: 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 (BP). 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 BP independent. The SAC was greater in the ET groups compared with C and ST groups. The SAC in L-ST was lower than in C.

Conclusions: These results suggest that changes in arterial stiffness associated with different training programs appear in young adults as well as in older humans, and these changes may begin in adolescence. Am J Hypertens 2007;20:967-973 © 2007 American Journal of Hypertension, Ltd.

Key Words: Adolescence, artery, compliance, exercise, young adults.

Increased arterial stiffness is an independent risk factor for the development of atherosclerosis and cardiovascular disease.^{1,2} In addition, 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 (eg, sodium restriction) improve arterial stiffness and may reduce the risk of developing adverse complications.⁴ Furthermore, we and other investigators 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 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 have decreased arterial stiffness, whereas those in strength-based sports would have increased arterial stiffness. In addition, we predicted that these trends would be more pronounced with an increase in the duration of sport participation. Accordingly, we measured the aortic pulse wave velocity (PWV) and systemic arterial compliance (SAC) in young adults (19 to 23 years old) endurance- and strength- trained men participating in competitive sports from adolescence (12 to 18 years). The study subjects were divided into short and long

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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. The ST group had been performing vigorous weight training (three sessions per week) in addition to their athletic training. Both ST and ET groups 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 (ie, cross-training) on a regular basis were excluded. Controls were 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 h and caffeine consumption for 4 h 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 min. 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. The PWV was calculated as the distance divided by the transit time.

Systemic Arterial Compliance

The 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, bra-

chial arterial blood pressure (BP) 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 BP to brachial artery values. The SAC was calculated as follows: $SAC = Ad/(dP \times R)$, where Ad is the area under an arbitrary portion of the diastolic pressure waveform; dP is the pressure change in this portion; and R is total peripheral resistance given as mean BP 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; National Institutes of Health, 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 sec and at least 15 sec apart, were performed by each hand. The maximal strength score achieved from the two 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 h 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 \pm SE. The differences in physiologic 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 BP 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 two exercise groups. $P < .05$ 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-to-hip ratio, and BP were higher in the ST groups compared to the C and ET groups. Differences in serum total cholesterol, LDL-cholesterol, and triglycerides levels were not present among the groups (Table 2). Serum concentrations of HDL-cholesterol in the L-ET group was greater compared with C and L-ST groups (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, which separately included systolic BP and heart rate as covariates, exercise training was associated with aortic PWV ($F = 6.7, P < .0001$ and $F = 10.4, P < .0001$, respectively). The significant association between aortic PWV and training programs existed also after adjustment for both systolic BP and heart rate ($F = 5.5, P = .0022$). In multiple comparisons, the aortic PWV was lower in the L-ET than C groups, and the aortic PWV in the L-ST group was higher than that of C group. 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 BP ($F = 13.7, P < .0001$), heart rate ($F = 12.4, P < .0001$), and both indices ($F = 7.4, P < .0001$) were included as covariate. Compared to the control group, SAC was greater in the ET groups and lower in the L-ST group. In addition, the SAC of the ET groups was higher than that of the ST groups. Because BMI was higher in the ST groups, we also used BMI as a covariate of ANCOVA. There were no significant associations of exercise program with the aortic PWV and SAC when BMI was entered as covariate.

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

	Sedentary men	Endurance-trained men		Strength-trained men		F value
		Short career	Long career	Short career	Long career	
<i>n</i>	7	7	7	7	7	
Age (y)	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 (y)		14.7 ± 0.7	12.1 ± 0.1†	16.0 ± 0.4†	14.6 ± 0.5†‡	11.5#
Sport careers (y)		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-to-hip ratio	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 (mm Hg)	116 ± 2	121 ± 4	110 ± 1†	127 ± 3*	131 ± 5*†	6.4#
Diastolic blood pressure (mm Hg)	63 ± 2	64 ± 2	57 ± 1†	67 ± 3	70 ± 2*†	4.8#
Mean blood pressure (mm Hg)	83 ± 2	88 ± 3	80 ± 1†	90 ± 3	94 ± 4*†	4.6#
Pulse pressure (mm Hg)	53 ± 1	57 ± 3	53 ± 0	60 ± 1*	62 ± 3*†	3.9#
Heart rate (beats/min)	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#

* $P < .05$ v sedentary men; † $P < .05$ v sport career-matched endurance-trained men; ‡ $P < .05$ v short sport career group; # statistical significance of F value ($P < .05$).

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

	Sedentary men	Endurance-trained men		Strength-trained men		F value
		Short career	Long career	Short career	Long career	
<i>n</i>	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

* $P < .05$ v sedentary men; † $P < .05$ v sport career-matched endurance-trained men; ‡ statistical significance of F value ($P < .05$).

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 ($n = 14$, $P = .05$) and 0.39 in the ET athletes ($n = 14$, $P = .16$). Those between SAC and the duration of sport careers were 0.33 ($n = 12$, $P = .29$) and 0.31 ($n = 13$, $P = .30$), respectively.

Discussion

We identified that the aortic PWVs of endurance- and strength-trained young adult men were lower and higher, respectively, compared with 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 BP and heart rate. Thus, overall, arterial stiffness was lower in endurance-trained men and higher in strength-trained men compared with 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 other investigators previously reported that regular endurance exercise decreases⁵⁻⁹ and regular strength exercise increases arterial stiffness.⁹⁻¹³ Recently, greater emphasis has been placed in schools on physical activity for cardiovascular health promotion.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 al¹⁷ reported that the level of maximal oxygen uptake in school-age adolescents (13 to 16 years old) was independently associated with carotid artery intima-media thickness (IMT) at middle age (36 years old). In addition, 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 or exert additive effects.

In the present study, PWV and BP 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 physiologic adaptations to the intense BP increase during strength-based sports, the increases in arterial stiffness may not be an 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 with concentric strength training.²⁰ In addition, 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 the general population (ie, nonspecialty athletes), endurance training, which decreases arterial stiffness, is often performed in parallel with strength training (ie, cross-training). It was demonstrated that regular rowing exercise, which includes components of aerobic endurance and muscular strength, may decrease arterial stiffness.²² An intervention study²³ has also reported that cross-training does not stiffen elastic arteries. Taken together, it is possible that strength-based sports performed in parallel with endurance training could improve or, at least, 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 shown 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 ath-

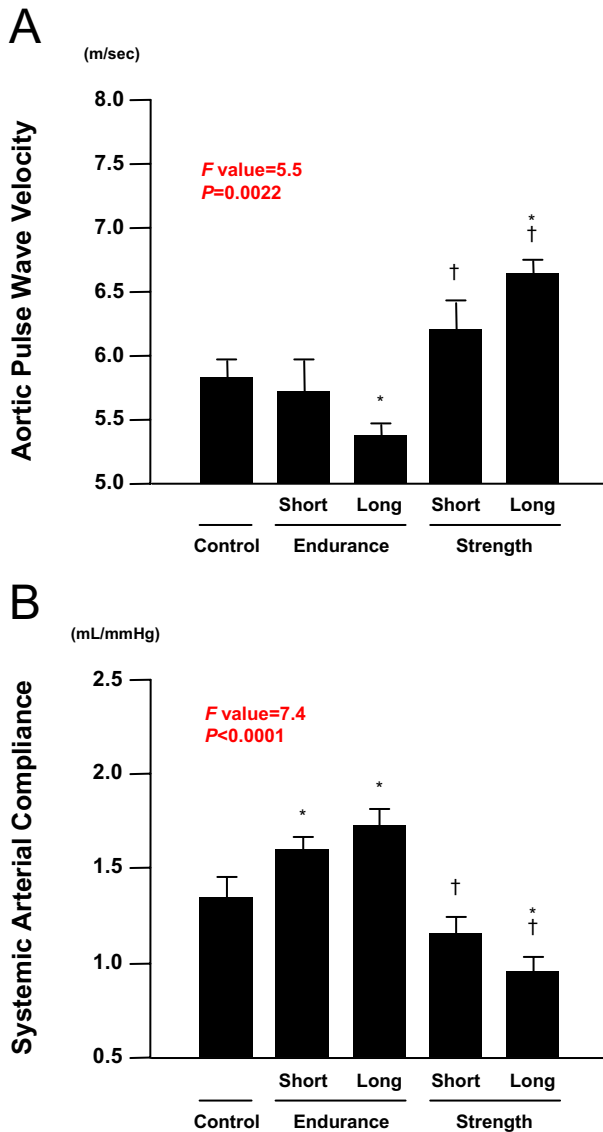


FIG. 1 Pulse wave velocity (A), a traditional index of arterial stiffness, and systemic arterial compliance (B), which is inversely related to arterial stiffness, in endurance- and strength-trained men. Data are expressed as means \pm SE. * $P < .05$ v sedentary control; † $P < .05$ v sport career-matched endurance-trained men.

letes.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 cannot 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. The serum lipid profile, except for HDL-cholesterol, did not differ between groups. In a previous study Miyachi et al¹¹ demonstrated that arterial stiffness is higher in strength-trained men by using the height- and weight-matched groups. In addition, 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 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 intergroup differences in BMI existed, the correlation coefficients between the arterial stiffness measures and the duration of the sport careers were not strong. The BMI was higher in the 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 the 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 too 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 or skinfold thickness using a larger sample size. Second, we cannot say that PWV and SAC mean the same thing for heavily muscled athletes as for sedentary control and endurance-trained men. However,

the results in the present study (ie, the increased arterial stiffness in strength-trained men) were not in conflict with previous studies that 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 of the β -stiffness index.¹² Thus, we consider that PWV and SAC are available also in heavily muscled athletes, although we cannot negate that the muscularity could also have artifactual effects 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 the arm is greater in the throw than running, although both sports use the whole body. This would result in the differences in central hemodynamics during each 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 (12 to 18 year olds) risk variables and IMT in 36 years olds, 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 BP 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 support 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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