Absence of Age-Related Increase in Central Arterial Stiffness in Physically Active Women

Hirofumi Tanaka, Christopher A. DeSouza, Douglas R. Seals

Abstract—Increased arterial stiffness is thought to contribute to the increased incidence of cardiovascular disease with age. Little, however, is known about the influence of aging on central and peripheral arterial stiffness in females. Moreover, it is unknown whether physical activity status influences age-related increases in arterial stiffness in females. Arterial pulse wave velocity (PWV) and augmentation index (AI, applanation tonometry) were measured in 53 healthy females, including 10 premenopausal (Pre-S) and 18 postmenopausal (Post-S) sedentary women, and 9 premenopausal (Pre-PA) and 16 postmenopausal (Post-PA) physically active women. In the sedentary women, there were no age-related differences in arterial blood pressure, but aortic PWV and carotid AI (measures of central arterial stiffness) were higher (P<.01) in Post-S versus Pre-S (1065±110 versus 690±80 cm/sec and 16.5%±1.8% versus 0.3%±1.6%, respectively); however, there were no significant differences in leg and arm PWV (measures of peripheral arterial stiffness). Systolic and mean arterial blood pressures were higher (P<.05) in Post-PA versus Pre-PA. Despite this and in contrast to the sedentary women, aortic PWV and AI were not different in Post-PA versus Pre-PA. Stepwise multiple regression indicated that maximal oxygen consumption, plasma total cholesterol, and plasma LDL-cholesterol were significant independent predictors and together explained up to 50% of the variability in central arterial stiffness. We concluded that (1) central, but not peripheral, arterial stiffness increases with age in sedentary healthy females in the absence of age-related increases in arterial blood pressure; (2) significant age-related increases in central arterial stiffness are not observed in highly physically active women; and (3) aerobic fitness and plasma total cholesterol and LDL-cholesterol levels are significant independent physiological correlates of central arterial stiffness in this population. (Arterioscler Thromb Vasc Biol. 1998;18:127-132.)

Key Words: exercise ■ aging ■ pulse wave velocity ■ augmentation index ■ arterial compliance

The stiffness of the “central” arteries (eg, aortic, carotid) increases with age in males,1-3 as indicated by an increase in PWV or earlier pressure wave reflections (ie, increased AI).1-3 These increases in arterial stiffness are thought to contribute to age-related increases in the incidence of cardiovascular disease.4-5 Much less is known about the influence of aging on arterial stiffness in females. A recent report from the Baltimore Longitudinal Study of Aging (BLSA)6 found that aortic PWV and carotid AI increased progressively with age in 50 healthy females (26 to 96 years) in whom only modest age-related increases in blood pressure were observed. No data are available, however, regarding the effects of aging on peripheral arterial stiffness in healthy females. This is noteworthy in that the elastic properties of arteries are not necessarily uniform,1 and aging has been reported to have different effects on the stiffness of peripheral (eg, brachial and radial) and central arteries in men.6

Regular physical activity is associated with reduced risk of cardiovascular disease.7-8 In the BLSA mentioned above, older adult males who performed endurance exercise on a regular basis demonstrated lower levels of aortic PWV and carotid AI than their sedentary peers.7 These observations suggest that habitual aerobic exercise may delay or prevent age-associated increases in central arterial stiffness. However, the absence of data on corresponding endurance-trained young adults precluded the ability to assess this possibility. Moreover, these data on males cannot necessarily be generalized to females because certain unique age-associated factors, such as menopause and hormone supplementation, could independently affect the elastic properties of arteries.

Accordingly, the aims of the present investigation were to determine (1) if central and/or peripheral arterial stiffness increases with age in sedentary healthy females in the absence of age-related increases in arterial blood pressure; (2) if these increases in arterial stiffness with age are not observed in highly physically active women; and (3) the key physiological correlates of central and peripheral arterial stiffness in healthy females varying in age and physical activity status.

Methods

Subjects
A total of 53 healthy women were studied. They were divided into two groups according to their physical activity status. The sedentary
groups consisted of 10 Pre-S and 18 Post-S women, none of whom performed regular exercise. The physically active groups consisted of 9 Pre-PA and 16 Post-PA women who had been performing endurance exercise training for at least the past 2 years (mean, 13±1 year), and were actively competing in running road races. On average, the endurance-trained women exercised for 6±1 h/wk. Pre-PA and Post-PA were matched for age-adjusted running performance (Masters Age-Graded Tables, National Masters News, Van Nuys, CA) as described previously. All subjects were free of overt cardiovascular disease as assessed by medical history questionnaire and had plasma lipid and lipoprotein concentrations all within the normal range. Postmenopausal women were further evaluated by physical examination and by resting and maximal exercise ECGs. None of the subjects smoked or took medications (other than hormone replacement). All women in the postmenopausal groups were postmenopausal at least 2 years (mean, 10±1 year). All premenopausal women were eumenorrheic as assessed by self-report of menstrual cycles, and were not taking oral contraceptives. Among the 34 postmenopausal women, 18 (10 sedentary and 8 active) used hormone replacement and 16 (8 sedentary and 8 active) did not. We observed no influence of hormone replacement use. Therefore, the data were pooled and presented together. Before participation, a verbal and written explanation of the procedures and potential risks was provided. All subjects gave their written informed consent to participate. This study was reviewed and approved by the Human Research Committee of the University of Colorado at Boulder.

Measurements

Measurement of arterial stiffness was conducted after an abstinence of caffeine and an overnight fast of at least 12 hours. Subjects were familiarized with all pertinent procedures before making the measurements. During the experimental session, each subject rested supine for at least 15 minutes in a quiet, temperature-controlled room. Blood pressure was measured by auscultation over the brachial artery in the last 5 minutes according to American Heart Association guidelines. Postmenopausal women were further evaluated by physical examination and by resting and maximal exercise ECGs. None of the subjects smoked or took medications (other than hormone replacement). All women in the postmenopausal groups were postmenopausal at least 2 years (mean, 10±1 year). All premenopausal women were eumenorrheic as assessed by self-report of menstrual cycles, and were not taking oral contraceptives. Among the 34 postmenopausal women, 18 (10 sedentary and 8 active) used hormone replacement and 16 (8 sedentary and 8 active) did not. We observed no influence of hormone replacement use. Therefore, the data were pooled and presented together. Before participation, a verbal and written explanation of the procedures and potential risks was provided. All subjects gave their written informed consent to participate. This study was reviewed and approved by the Human Research Committee of the University of Colorado at Boulder.

Arterial Applanation Tonometry

The pressure waveform and amplitude were obtained from the right common carotid artery with a pencil-type probe incorporating a high-fidelity strain-gauge transducer (model TCB-500, Millar Instruments), as previously described by Kelly et al. This instrument was based on the principle of applanation tonometry as used in ocular tonometry for the measurement of intraocular pressure. In principle, the flattening or applanation of the curved surface of a pressure-containing structure under the detecting device allows direct measurement of arterial pressure pulse within the structure. This tonometer has been shown to register a pressure wave with harmonic content that does not differ from that of an intra-arterially recorded wave, and the use of the tonometer on an exposed artery records a waveform identical to that recorded intra-arterially. Waveforms were recorded on a Gould recorder at a high speed of 100 mm/sec. All measurements were performed by the same investigators. Recordings were taken only when reproducible signals could be obtained with high-amplitude excursion. The peak of the R wave from the simultaneously recorded ECG was used as a timing marker. A minimum of 20 consecutively recorded pulse waves were analyzed and averaged as previously described. All the analyses were performed manually by the same investigator who was blinded to the group assignment. The measured pressure waveform consists of both a “forward” or “incident” wave, and a “reflected” wave that is returning from a peripheral site. The reflected wave is superimposed on the incident wave such that the pulse and systolic pressures are increased. This increase is defined as a pressure pulse AI, and it is calculated as pressure wave above its systolic shoulder (ΔP) divided by pulse pressure (ΔP). The shoulder was defined as the first concavity on the upstroke of the wave and separates the initial systolic pressure rise from the late systolic peak. The carotid AI has been proposed as an indicator of the magnitude of wave reflections, which is closely linked to arterial stiffness. In the present study, carotid AI was used as a measure of the stiffness of the central arteries. The reliability of the AI measurement in our laboratory was established by sequential measurement on 8 adult men and women of varying age on two separate days. Carotid AI was 5.0%±3.2% versus 4.8%±2.9% for trial 1 versus trial 2 (not significant); the mean coefficient of variation was 7%.

PWV

PWV is measured from the foot of pressure waves recorded at two points along the path of the arterial pulse wave, and is calculated from the measurement of pulse transit time (or time delay) and the distance traveled between two arterial recording sites. Two identical transcufcutaneous Doppler flowmeters (model 810-A, Parks Medical) were used to obtain the pulse wave (1) between the aortic arch and the femoral artery (aortic PWV); (2) between the femoral and posterior tibial artery (leg PWV); and (3) between the brachial and radial artery (arm PWV), as previously described by Avolio et al. Distance traveled by the pulse wave was assessed in duplicate with a random zero length measurement over the surface of the body with a nonelastic tape measure. The peak of the R wave from the simultaneously recorded ECG was used as a timing marker. A minimum of 20 simultaneously recorded waveforms were analyzed and averaged as described previously. All the analyses were performed by the same trained technician who was blinded to the group assignment. Aortic PWV was used as a measure of the stiffness of the central arteries, whereas leg and arm PWV were used as measures of peripheral arterial stiffness.

Aortic pressure waves were digitized for off-line analysis with signal-processing software (WINDAQ, Dataq Instruments). PWV was calculated from distance (cm) divided by transit time (sec). Transit time was determined from the time delay between the proximal and the distal foot waveforms. The foot of the wave was identified as the commencement of the sharp systolic upstroke. The test-retest reliability of our PWV measurements was established using the experimental approach described for AI above. The mean PWV combined for three sites was 573±56 versus 586±54 cm/sec for trial 1 versus trial 2 (not significant). The coefficients of variation of aortic, arm, and leg PWV measurements were similar with mean values of 8% in each case.

Potential Physiological Correlates of Arterial Stiffness

Body fat percentage was estimated from the hydrostatic weighing technique. Body mass index was calculated according to the formula of body mass (kg)/height (m²). Waist circumference was measured at the narrowest part of the torso, and hip circumference was measured at the maximal extension of the buttocks. VO₂max was assessed with on-line computer-assisted open-circuit spirometry during incremental treadmill exercise as described in detail previously. Dietary sodium intake was determined using 3-day food intake records, and 24-hour urinary sodium excretion was determined with the use of flame photometry. Fasting plasma concentrations of cholesterol, glucose, and insulin were performed in the clinical laboratory affiliated with the General Clinical Research Center at the University of Colorado Health Sciences Center as described previously. All measurements of metabolic variables on the premenopausal women were performed during the early follicular phase of the menstrual cycle.

Statistical Analyses

The respective influences of aging and physical activity were assessed with two-way ANOVA (age and physical activity). When indicated
by a significant F-value, a post-hoc test using Scheffé’s method was performed to identify significant differences among group means. ANCOVA, using systolic blood pressure or body fatness as a covariate, was used to analyze the effect of age on arterial stiffness. Univariate correlation and regression analyses were performed to determine the relation between arterial stiffness measurements and selected physiological variables. Stepwise regression analyses were used to determine significant, independent physiological correlates for each of the arterial stiffness measurements. Age was not included in the regression analyses because it did not have a continuous distribution. All data are reported as the mean±SE. Statistical significance was set at P<.05 unless indicated otherwise.

Results

Arterial Stiffness in Premenopausal versus Postmenopausal Sedentary Women

Table 1 shows the physical characteristics and blood pressure at rest of the sedentary women. Body mass index, percent body fat, and waist-to-hip ratio were higher (P<.01) in Post-S relative to Pre-S. There were no differences in height, body mass, fat-free mass, resting heart rate, or arterial blood pressure between the two groups. Post-S had a lower V̇O₂max (P<.001) than Pre-S.

Aortic PWV and carotid AI were higher (P<.01) in Post-S than in Pre-S (1060±58 versus 690±80 cm/sec and 16.4%±1.4% versus 0.3%±1.6%, respectively) (Fig 1). When ANCOVA was performed using either BMI, percent body fat, or waist/hip as a covariate, the difference between Post-S and Pre-S remained statistically significant (P<.05). In contrast, there were no significant differences in leg and arm PWV in the two groups.

Arterial Stiffness in Premenopausal versus Postmenopausal Physically Active Women

Physical characteristics of the physically active women are presented in Table 2. There were no significant differences in height, body mass, fat-free mass, BMI, waist-to-hip ratio, and resting heart rate between Post-PA and Pre-PA. Post-PA had higher (P<.01) percent body fat than Pre-PA. Although well within the normotensive range, systolic and mean arterial blood pressure were higher (P<.05) in Post-PA than in Pre-PA whereas no significant difference was observed for diastolic blood pressure. Post-PA had a lower V̇O₂max (P<.001) than Pre-PA.

In contrast to the sedentary women, there were no significant differences in either aortic PWV or carotid AI between Post-PA and Pre-PA (Fig 2). When ANCOVA was performed using systolic blood pressure as the covariate, aortic PWV and carotid AI were 671.5±56.0 and 611.8±37.9 cm/sec and 6.4%±1.6% and 3.9%±2.3% in Post-PA and Pre-PA, respectively (both not significant). Importantly, aortic PWV and carotid AI were ~30% and 50% lower (P<.01), respectively, in Post-PA versus Post-S.

Physiological Correlates of Arterial Stiffness

Univariate correlation analyses were performed to determine which physiological variables were most closely associated with arterial stiffness.

Table 1. Physical Characteristics of the Sedentary Women

<table>
<thead>
<tr>
<th></th>
<th>Pre-S (n=10)</th>
<th>Post-S (n=18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>28±2</td>
<td>59±2*</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.66±0.02</td>
<td>1.62±0.02</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>62.5±4.2</td>
<td>69.7±2.8</td>
</tr>
<tr>
<td>Fat free mass, kg</td>
<td>44.3±1.9</td>
<td>42.5±1.2</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>22.9±1.5</td>
<td>26.6±1.0*</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>28±3</td>
<td>38±2*</td>
</tr>
<tr>
<td>Waist, cm</td>
<td>75.1±2.8</td>
<td>87.2±2.5*</td>
</tr>
<tr>
<td>Waist/hip</td>
<td>0.75±0.01</td>
<td>0.82±0.02*</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>111±2</td>
<td>118±2</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>73±2</td>
<td>76±1</td>
</tr>
<tr>
<td>MABP, mm Hg</td>
<td>86±2</td>
<td>90±1</td>
</tr>
<tr>
<td>Resting HR, bpm</td>
<td>61±2</td>
<td>62±3</td>
</tr>
<tr>
<td>V̇O₂max, mL·kg⁻¹·min⁻¹</td>
<td>33.8±2.1</td>
<td>22.3±1.1*</td>
</tr>
</tbody>
</table>

Data are mean±SE. SBP indicates systolic blood pressure; DBP, diastolic blood pressure; MABP, mean arterial blood pressure; and HR, heart rate. *P<.01 vs premenopausal group.

Table 2. Physical Characteristics of the Physically Active Women

<table>
<thead>
<tr>
<th></th>
<th>Pre-PA (n=9)</th>
<th>Post-PA (n=16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>31±1</td>
<td>59±2*</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.65±0.01</td>
<td>1.66±0.01</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>52±0.17</td>
<td>57±1.5</td>
</tr>
<tr>
<td>Fat free mass, kg</td>
<td>44±2.5</td>
<td>43±0.1</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>19±0.5</td>
<td>20.7±0.4</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>15±2</td>
<td>24±1*</td>
</tr>
<tr>
<td>Waist/cm</td>
<td>66±1.6</td>
<td>70.5±1.0</td>
</tr>
<tr>
<td>Waist/hip</td>
<td>0.74±0.01</td>
<td>0.74±0.01</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>106±3</td>
<td>118±2*</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>70±2</td>
<td>76±1</td>
</tr>
<tr>
<td>MABP, mm Hg</td>
<td>82±3</td>
<td>90±2*</td>
</tr>
<tr>
<td>Resting HR, bpm</td>
<td>56±1</td>
<td>53±2</td>
</tr>
<tr>
<td>V̇O₂max, mL·kg⁻¹·min⁻¹</td>
<td>54.3±1.6</td>
<td>36.6±2.0*</td>
</tr>
</tbody>
</table>

Data are mean±SE. SBP indicates systolic blood pressure; DBP, diastolic blood pressure; MABP, mean arterial blood pressure; and HR, heart rate. *P<.05 vs premenopausal group.
arterial stiffness. Because the correlated variables and correlation coefficients were similar in the sedentary and physically active groups and when grouped separately by age (ie, premenopausal and postmenopausal), the data were pooled and presented together.

The relations between aortic PWV and selected correlates of interest are depicted in Fig 3. Aortic PWV and carotid AI were significantly ($P < .01$) related to body fat percentage ($r = 0.54$ and $0.47$), waist circumference ($r = 0.58$ and $0.42$), waist-to-hip ratio ($r = 0.44$ and $0.42$), $\dot{V}O_2\text{max}$ ($r = -0.66$ and $-0.53$), systolic blood pressure ($r = 0.46$ and $0.42$), mean arterial blood pressure ($r = 0.40$ and $0.42$), plasma total cholesterol ($r = 0.56$ and $0.52$), and plasma LDL-cholesterol ($r = 0.56$ and $0.48$), respectively. Aortic PWV and carotid AI were not significantly related to height, diastolic blood pressure, resting heart rate, dietary sodium intake, urinary sodium excretion, plasma HDL-cholesterol, or fasting glucose or insulin concentrations. A modest, but significant correlation ($r = 0.43$, $P < .01$) existed between carotid AI and aortic PWV. There were no significant correlations between arm or leg PWV and the other subject characteristics, with the exception of correlations between systolic blood pressure and leg PWV ($r = 0.40$, $P < .01$) and between fasting insulin concentration and arm PWV ($r = -0.40$, $P < .01$). Arm and leg PWV were not significantly related to aortic PWV.

To establish which of these correlates were independent predictors of arterial stiffness in the overall population, we performed stepwise regression analyses. The variables that entered for carotid AI were $\dot{V}O_2\text{max}$, which explained $25\%$ of the variability ($P < .001$), and total cholesterol, which explained an additional $5\%$ of the variability ($P < .05$). For aortic PWV, $\dot{V}O_2\text{max}$ appeared first in the analysis and explained $41\%$ of the variability ($P < .0001$); an additional $8\%$ of variability in aortic PWV was explained by LDL-cholesterol ($P < .05$). No other variables entered were significant predictors.

**Discussion**

**Arterial Stiffness in Premenopausal versus Postmenopausal Sedentary Women**

Healthy postmenopausal sedentary women demonstrated significantly higher levels of aortic PWV and carotid AI compared with premenopausal females, indicating an age-associated increase in central arterial stiffness. A novel aspect of the present study was that the elevated central arterial stiffness was observed with age in the sedentary women in the absence of increases in arterial blood pressure. In previous studies, reporting stiffening of central arteries with age in females, at least modest increases in arterial blood pressure, an important correlate of arterial stiffness, were present. Thus, our data indicate a primary effect of aging in the increase in the stiffness of central arteries observed in healthy sedentary postmenopausal women. This increase in arterial stiffness, however, may precede an age-related elevation in blood pressure in these women.

There are several possible explanations for the influence of aging on the loss of elasticity of central arteries. The most likely explanation appears to be age-associated structural changes in the arterial wall. Aging is associated with a decrease in elastin and an increase in collagen and connective tissues in the arterial wall. Fragmentation of the internal elastic lamina with age has also been observed in human thoracic aorta. Age-related increases in central arterial stiffness do not appear to be dependent on the presence of clinical atherosclerotic disease. The stiffening of arteries has been observed in a Chinese population in whom the prevalence of atherosclerosis is very low as well as in rigorously screened US men and women. However, in the present study, there was a modest correlation between arterial stiffness and plasma cholesterol levels. There-
fore, we cannot exclude the possibility that subclinical atherosclerosis contributed to the age-related elevation in central arterial stiffness in the sedentary women.

In contrast to the central arteries studied, there was no obvious age-associated increase in the stiffness of the peripheral arteries in the present population of healthy normotensive females. In hypertensive subjects, it has been reported that the common carotid arteries demonstrate a greater increase in rigidity with age than the common femoral arteries.18 Such differential effects of aging on the stiffness of central versus peripheral arteries may be related to their distinct roles in hemodynamic regulation. Compared with the central arteries whose cushioning function damps fluctuations in flow, the peripheral arteries do not exhibit the same extent of pulsatile changes in diameter19 and, as such, may not undergo the adaptations leading to a loss of elasticity.

Our findings of an increase in central arterial stiffness with age even in healthy normotensive women may have a number of clinical implications. Arterial stiffness recently has been identified as a risk factor for cardiovascular disease,4 the prevalence of which increases markedly after menopause.20 The stiffening of arteries is thought to result in impairments in baroreflex sensitivity, which could play a role in the increased prevalence of orthostatic hypotension observed with age.21–23 Moreover, increased central arterial stiffness would act to increase the afterload imposed on the left ventricle, contributing to ventricular hypertrophy, and eventually, left ventricular ischemia and dysfunction, which are known to increase with age in women.24 Importantly, our results suggest that even healthy normotensive postmenopausal women face the clinical risks of these increases in central arterial stiffness.

**Arterial Stiffness in Premenopausal versus Postmenopausal Physically Active Women**

In the present study, we found that aortic PWV and carotid AI levels were ~30% to 50% lower in Post-PA compared with Post-S. Our results support the recent findings of Vairkevicious and colleagues from the BLSA,5 in which physically active older males demonstrated ~25% to 35% lower levels of carotid AI and aortic PWV than their sedentary age-matched peers. However, because no data on active young adults were presented in their study, it is possible that young active males might also have exhibited reduced arterial stiffness compared with age-matched sedentary males. Thus, similar increases in central arterial stiffness with age may have been present in active and sedentary males.

The present study extends our current understanding of the relation between physical activity, aging, and arterial stiffness in at least two ways. First, by establishing that central arterial stiffness is similar in active versus sedentary young adults, our findings indicate that the lower levels of central arterial stiffness observed in middle-aged and older physically trained subjects appear to be caused by an absence of an increase with age. Second, our study demonstrates this beneficial association between regular physical activity, aging, and arterial elasticity in women, in whom the age-related risk of cardiovascular disease is markedly elevated after menopause.20

It is not clear how regular physical activity may prevent increases in central arterial stiffness with age. One possibility is that arterial blood pressure does not increase as much with age in physically active women. However, in the present study the active postmenopausal women demonstrated higher levels of systolic and mean arterial blood pressure and pulse pressure than the active premenopausal women. Thus, there was an absence of central stiffening with age despite a modest elevation in arterial blood pressure. This apparent dissociation between age-related elevations in arterial pressure and stiffness may be explained by the fact that the resting blood pressure of the active postmenopausal women was still within the normotensive range (Table 2). Another possibility is that regular physical activity minimizes age-related structural changes in the arterial wall. In this regard, the endurance-trained state has been shown to be associated with an elevated overall content of elastin and a reduced calcium content of elastin in rat aorta.25

A third possibility is that regular physical activity may act to maintain endothelium-dependent vasodilation with age, as recently reported.26

**Physiological Correlates of Arterial Stiffness**

Several factors in addition to arterial blood pressure are known to be related to the elasticity of arteries. We found that central arterial stiffness was significantly related to body fitness, aerobic fitness, systolic blood pressure, and the plasma lipid and lipoprotein profile in the present study sample of healthy females. Stepwise regression analysis revealed that aerobic fitness, plasma total cholesterol, and plasma LDL–cholesterol levels were the significant independent correlates of central arterial stiffness.

Aerobic fitness is a major determinant of overall physiological functional capacity, and low levels of aerobic fitness have recently been identified as a risk factor for cardiovascular as well as all-cause mortality.8,27 The present finding of a significant inverse relation between measures of central arterial stiffness and maximal oxygen consumption in general supports recent results from the BLSA.2 However, whereas an association between aortic PWV and aerobic fitness was observed primarily in the older cohort in that study, we found significant relations ($r=-0.5$ to $-0.7$) in each age and activity subgroup, as in the overall pooled population. Thus, collectively, the present findings and those from the BLSA2 suggest that central arterial stiffness may be one factor responsible for the inverse relation between premature mortality and aerobic fitness in middle-aged and older men and women.

Finally, increased arterial stiffness has been proposed as a potential mechanism in the initiation and progression of atherosclerosis,4,28 although the exact mechanistic link has not been established. The strong univariate correlations between plasma lipid and lipoprotein concentrations and central arterial stiffness, and in particular, the independent association with plasma total cholesterol and LDL–cholesterol levels suggest that dyslipidemia may play a role in the relation between central arterial stiffness and the risk of atherosclerosis in women.4

**Summary and Conclusion**

In summary, we have shown that central, but not peripheral, arterial stiffness increases with age in sedentary healthy females in the absence of age-related increases in arterial blood pressure. The age-associated stiffening of central arteries did not
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occur in physically active females, suggesting that high levels of physical activity may prevent increases in arterial stiffness with advancing age in women. Finally, aerobic fitness, total cholesterol, and LDL-cholesterol were found to be significant independent physiological correlates of central arterial stiffness in healthy females varying in age and physical activity status. Our findings suggest that an absence of increases in arterial stiffness with age may contribute to the lower incidence of cardiovascular disease observed in postmenopausal women who exercise regularly.

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References


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