Muscular Strength is Inversely Associated with Aortic Stiffness in Young Men

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ABSTRACT

FAHS, C. A., K. S. HEFFERNAN, S. RANADIVE, S. Y. JAE, and B. FERNHALL. Muscular Strength is Inversely Associated with Aortic Stiffness in Young Men. *Med. Sci. Sports Exerc.*, Vol. 42, No. 9, pp. 1619–1624, 2010. Muscular strength is associated with reduced mortality. Paradoxically, strength training may increase central artery stiffness, a predictor of cardiovascular morbidity and mortality. However, the relationship between muscular strength and central arterial stiffness has yet to be defined. **Purpose**: The purpose of this study was to determine the relationship between muscular strength and central arterial stiffness in young men. **Methods**: Central and peripheral pulse wave velocity (PWV), augmentation index, muscular strength, and aerobic capacity ($\dot{V}O_{2peak}$) were measured in 79 young men (mean ± SD, age = 23 ± 4 yr). Height, weight, and brachial blood pressure were also recorded. Muscular strength was determined using a one-repetition maximum bench press and normalized to bodyweight. Spearman correlations were used to determine the relationships between relative strength, aerobic fitness, and hemodynamic/vascular measures. **Results**: There was a significant negative correlation between central PWV and strength ($r = -0.222$, $P < 0.05$). The relationship remained significant when controlling for aerobic fitness ($r = -0.189$, $P < 0.05$). Muscular strength was significantly higher ($P < 0.05$) in men with low central PWV (5.2 ± 0.4 m·s^{-1}) compared with men with high central PWV (6.6 ± 0.4 m·s^{-1}). **Conclusion**: These results show that there is a significant inverse association between muscular strength and aortic stiffness independent of aerobic fitness. **Key Words:** ABSOLUTE STRENGTH, RELATIVE STRENGTH, PULSE WAVE VELOCITY, WAVE REFLECTION

Muscular strength is inversely associated with incidence of diabetes mellitus, hypertension, and mortality, and these associations are independent of associations with cardiorespiratory fitness (33). The benefits of habitual resistance exercise (RE) are well documented and include increases in muscle hypertrophy and muscular strength (14,21,45). Habitual RE can also favorably modify traditional cardiovascular (CV) risk factors by lowering blood pressure (BP), fasting glucose, insulin levels, plasma triglycerides, and percent body fat while increasing basal metabolic rate and quality of life (51). As such, numerous professional health/medical associations recommend inclusion of RE for health promotion and disease prevention (1).

The impact of habitual RE on more novel CV risk factors, such as arterial stiffness, is less clear. Increased central arterial stiffness is currently recognized as an independent risk factor of stroke and is related to risk for coronary artery disease (5,30). Even in young prehypertensives, central artery stiffness is related to P-wave dispersion, an independent risk factor for atrial fibrillation (8) as well as target organ damage such as left ventricular hypertrophy (49) and carotid intima-medial thickening (27). Furthermore, increased central artery stiffness is associated with obesity, cholesterol, and diabetes in young individuals (17,18), providing evidence suggesting that both lifestyle behavior and genetic predispositions also impact arterial function and CV risk in young individuals.

An acute bout of RE has been shown to increase arterial stiffness (13,16,24). Moreover, chronic resistance training (RT) has also been shown to increase arterial stiffness (37), whereas resistance-trained athletes may have greater femoral and central arterial stiffness compared with sedentary controls (4), although these are not universal findings (6,44).

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In fact, there are currently more studies noting no change (6,7,11,25,34,42,43,52) or a reduction (19,39) in central artery stiffness with RT interventions than intervention studies noting increases in central artery stiffness (10,12,29,37). Furthermore, muscular strength is associated with reduced risk of hypertension, diabetes, and CV risk in epidemiological studies (33,35,48), although the mechanism for the reduced risk in unknown. The impact of muscular strength on central artery stiffness could be one such potential mechanism.

Although some studies suggest that RT is associated with increased central arterial stiffness, the relationship between central arterial stiffness and muscular strength *per se* has yet to be clearly defined. Therefore, the purpose of this study was to examine the relationship between muscular strength and central arterial stiffness. We hypothesized that increased muscular strength would be associated with increased central arterial stiffness on the basis of evidence that chronic RT may increase arterial stiffness.

METHODS

Participants. Seventy-nine young healthy men ($N = 79$; mean ± SD, age = 23 ± 4 yr) participated in this study. Before participation, all participants gave written informed consent. Participants completed a medical history questionnaire to screen for possible problems preventing full compliance with the study protocol. Exclusions criteria included any known CV, pulmonary, or metabolic disease (asthma, diabetes, hypertension, dyslipidemia, etc.), orthopedic problems, use of any medication, and smoking. This research was approved by the institutional review board of the University of Illinois at Urbana-Champaign.

Study design. Each participant made one visit to the laboratory. Participants were instructed not to consume caffeine or alcohol for 24 h before testing, not to eat anything at least 3 h before testing, and not to engage in exercise or any other strenuous physical activity for 24 h before testing. Anthropometric measures (height and weight) were assessed in the standing position. After this, participants rested supine for a minimum of 5 min before hemodynamic assessments. In the supine position, brachial blood pressure (BP), radial augmentation index (AIx), carotid-femoral pulse wave velocity (cPWV), and femoral pulse wave velocity (fPWV) were assessed. Next, each participant completed a graded exercise test ($\dot{V}O_{2peak}$) on cycle ergometer followed by assessment of their one-repetition maximum (1-RM) supine bench press. A measure of upper body strength was chosen because acute lower body exercise does not appear to impact central arterial stiffness (28), whereas acute upper body exercise has been shown to acutely increase central arterial stiffness (16). Furthermore, the 1-RM bench press is recognized as a safe and reliable proxy of overall muscular strength (22) and has been shown to be an excellent test of overall body strength (3).

Anthropometric measures. Standing height and weight were measured with participants wearing light-weight clothing and no shoes. Height was measured using a stadiometer with measures to the nearest 0.5 cm. Weight was measured on a standard beam balance scale to the nearest 0.5 kg. Body mass index (BMI) was calculated as weight (kg) divided by height in meters squared.

Hemodynamic measures. Systolic and diastolic BP (SBP and DBP, respectively) were measured using an automated oscillometric cuff (HEM-907 XL; Omron Corporation, Tokyo, Japan). All BP measurements were made in duplicate, and the average of the two values was recorded and used for subsequent analysis. Mean arterial pressure (MAP) was calculated as MAP = (1/3)SBP + (2/3)DBP; pulse pressure (PP) was calculated as PP = SBP − DBP.

Augmentation index (AIx) was assessed using an applanation tonometry and a high-fidelity strain-gauge transducer (Millar Instruments, Houston, TX). AIx was calculated as the ratio of amplitude of the pressure wave above its systolic shoulder (i.e., the difference between the early and the late systolic peaks of the arterial waveform) to the total PP. The result was expressed as a percentage and was used as an index of aortic pressure wave reflection.

Pulse wave velocity (PWV) was assessed using the same strain-gauge transducer. cPWV was obtained with measurements at the right common carotid artery and the right femoral artery. Femoral pulse wave velocity (fPWV) was obtained with measurements at the right femoral artery and the right posterior tibial artery. Distances from the suprasternal notch to the femoral artery, carotid artery to the suprasternal notch, and femoral artery to the posterior tibial artery were measured as straight lines with a tape measure. The distance from the carotid artery to the suprasternal notch was subtracted from the suprasternal notch to femoral artery segment length to account for differences in the direction of pulse wave propagation. PWV was calculated from the distances between the measurement points and the measured time delay between 10 proximal and distal waveforms (SphygmoCor, AtCor Medical, Sydney, Australia). The intraclass correlation coefficient (ICC, a measure of internal consistency) for PWV in our laboratory, calculated on two separate days, is 0.92.

Graded exercise test ($\dot{V}O_{2peak}$). After a brief warm-up consisting of cycling against no resistance (0 W) for 2 min, participants began cycling at 50 W. Every 2 min, the exercise intensity was increased by 30 W until volitional fatigue was reached. Expired gases were analyzed using a Quark b2 breath-by-breath metabolic system. The test was terminated when participants met three of the following five criteria: 1) a final rating of perceived exertion score of 17 or greater on the Borg scale (scale 6–20), 2) a respiratory exchange ratio greater than 1.1, 3) no change in heart rate with a change in workload, 4) a "plateau" (increase of no more than 150 mL) in oxygen uptake with an increase in workload, and 5) volitional fatigue, defined as an inability to maintain a pedal rate above 50 rpm. $\dot{V}O_2$ data were averaged over 10-s intervals, and $\dot{V}O_{2peak}$ was recorded as the highest $\dot{V}O_2$ reached during the last minute of exercise and

expressed in absolute terms ($L \cdot min^{-1}$) and relative to total body weight ($mL \cdot kg^{-1} \cdot min^{-1}$). The ICC for $\dot{V}O_{2peak}$ in our laboratory has ranged from 0.90 to 0.97 in previous studies.

One-repetition maximum (1-RM). The 1-RM of the supine bench press, a standard measure of upper body muscular strength, was used for muscular strength assessment (22). Participants first completed a brief warm-up consisting of 10 repetitions of a submaximal load. A second warm-up was completed (three to five repetitions) using a submaximal load within the participant's perceived capacity. Weight was added in 2.3- to 11.4-kg increments until participants could no longer successfully complete one repetition. The heaviest weight successfully lifted was recorded as the 1-RM (kg). Relative strength was calculated as 1-RM (kg) divided by body weight (kg) and expressed as a ratio. The ICC for 1-RM bench press in our laboratory is 0.98.

Statistical analysis. Descriptive statistics were calculated for all variables. Because the data were not normally distributed, Spearman correlations were used to determine the relationships between relative strength, aerobic fitness, and hemodynamic/vascular measures (SBP, DBP, MAP, PP, AIx75, cPWV, and fPWV). Owing to issues of colinearity, absolute $\dot{V}O_2$ was used when examining associations between strength and aerobic fitness. A partial correlation was used to remove the effect of absolute $\dot{V}O_{2peak}$, on the relationship between relative strength and vascular measures. Cluster analysis was used to separate young men into two groups according to cPWV (i.e., "low" and "high" cPWV). ANOVA was used to compare group differences in continuous variables. ANCOVA was used to statistically adjust for the influence of aerobic fitness on potential group differences in relative strength. Logistic regression was used to examine the relationship between relative strength and cPWV.

RESULTS

Participant characteristics are shown in Table 1. Relative strength was inversely correlated with cPWV ($r = -0.222$, $P < 0.05$) and positively correlated with absolute $\dot{V}O_{2peak}$ ($r = 0.284$, $P < 0.05$). Relative strength was not associated with fPWV, AIx75, SBP, DBP, MAP, or PP ($P > 0.05$). After adjusting for absolute $\dot{V}O_{2peak}$, there was still a significant association between relative strength and cPWV ($r = -0.189$, $P < 0.05$).

TABLE 1. Participant characteristics ($N = 79$).

	Mean ± SD
Age (yr)	23 ± 4
Height (cm)	178.5 ± 6.0
Weight (kg)	84.5 ± 16.5
BMI ($kg \cdot m^{-2}$)	26.5 ± 4.7
1-RM bench press (kg)	95.0 ± 30.4
Relative strength (1-RM/weight)	1.13 ± 0.31
$\dot{V}O_{2peak}$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	33.92 ± 6.80
SBP (mm Hg)	126 ± 9
DBP (mm Hg)	74 ± 7
MAP (mm Hg)	91 ± 8
PP (mm Hg)	52 ± 9
HR (bpm)	58 ± 9
AIx75 (%)	−11 ± 11
cPWV ($m \cdot s^{-1}$)	5.9 ± 0.7
fPWV ($m \cdot s^{-1}$)	8.5 ± 1.2

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure; HR, heart rate; AIx75, augmentation index at 75 bpm; cPWV, carotid-femoral pulse wave velocity; fPWV, femoral pulse wave velocity.

TABLE 2. Characteristics of participants classified as "low" and "high" cPWV.

	High cPWV ($n = 32$)	Low cPWV ($n = 47$)	*P* Value
Age (yr)	24 ± 4	22 ± 3	0.145
Height (cm)	177.8 ± 6.2	178.6 ± 6.1	0.592
Weight (kg)	84.7 ± 18.1	84.3 ± 15.2	0.918
BMI ($kg \cdot m^{-2}$)	26.69 ± 5.12	26.40 ± 4.49	0.789
1-RM bench press (kg)	87.5 ± 26.7	100.2 ± 31.9	0.069
Relative strength	1.05 ± 0.31	1.19 ± 0.31	0.051
$\dot{V}O_{2peak}$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	34.47 ± 7.72	33.54 ± 6.16	0.555
SBP (mm Hg)	128 ± 9	124 ± 9	0.071
DBP (mm Hg)	75 ± 7	73 ± 7	0.155
MAP (mm Hg)	93 ± 6	90 ± 7	0.067
PP (mm Hg)	53 ± 9	52 ± 9	0.577
HR (bpm)	60 ± 10	57 ± 8	0.221
AIx75 (%)	−10 ± −11	−12 ± −11	−0.630
cPWV ($m \cdot s^{-1}$)	6.6 ± 0.4	5.4 ± 0.4	0.000
fPWV ($m \cdot s^{-1}$)	8.7 ± 1.3	8.4 ± 1.2	0.415

Data are presented as mean ± SD.
BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure; HR, heart rate; AIx75, augmentation index at 75 bpm; cPWV, carotid-femoral pulse wave velocity; fPWV, femoral pulse wave velocity.

According to cluster analysis, 47 men were classified as "low" cPWV (5.4 ± 0.4 $m \cdot s^{-1}$), whereas 32 men were classified as "high" cPWV (6.6 ± 0.4 $m \cdot s^{-1}$). Low and high cPWV groups did not differ in age, BMI, SBP, DBP, MAP, PP, AIx75, fPWV, or $\dot{V}O_{2peak}$ (Table 2); cPWV was significantly different between groups ($P < 0.05$) (Table 2). After covarying for absolute $\dot{V}O_{2peak}$, group differences in relative strength were significant with the low cPWV group having higher strength than the high cPWV group (adjusted means = 1.19 ± 0.3 vs 1.05 ± 0.3, $P = 0.051$). According to logistic regression, after controlling for age, BMI, SBP, and absolute $\dot{V}O_{2peak}$, high relative strength was a significant predictor of lower prevalence of high cPWV (odds ratio = 0.14, 95% confidence interval = 0.02–0.92, $P = 0.04$).

DISCUSSION

The main finding of the present study is that relative upper body muscular strength is inversely associated with central arterial stiffness. Controlling for absolute aerobic fitness ($\dot{V}O_{2peak}$) did not alter this association, suggesting that the relationship between relative strength and central arterial stiffness is independent of aerobic fitness.

Although muscular strength only accounted for approximately 5% of the variance in cPWV, this is an important finding as we hypothesized that muscular strength and central arterial stiffness would show a positive association because of the independent increases of each that have been shown with regular resistance-type exercise (41). Previous research has noted numerous potential correlates of aortic stiffness, including (but not limited to) age, race/ethnicity,

steady (e.g., MAP) and pulsatile (e.g., PP) components of BP, blood lipids, BMI (e.g., obesity), sex, endogenous hormone levels (e.g., sex steroids), inflammation/oxidative stress, insulin resistance/glucose metabolism, and cardiorespiratory fitness (32,36). There may also be genetic determinants that contribute to aortic stiffness (31,46). Our novel findings build upon previous work and note for the first time that muscular strength is a correlate of aortic stiffness, independent of cardiorespiratory fitness. This is not only a novel but also an important finding because cardiorespiratory fitness attenuates or negates the positive association between muscular strength and CV health (35). There is also a moderate/strong relationship between cardiorespiratory fitness and central artery stiffness (20). Some of the studies that note a favorable effect of high muscular strength on clinical outcome have also noted that this was mediated by the influence of concomitant high cardiorespiratory fitness (35). Thus, our finding that muscle strength is independently related to central artery stiffness is an important contribution to our understanding. Our findings are also consistent with previous reports noting favorable independent associations between higher muscular strength and lower incidence of hypertension, diabetes, and CV mortality (33,35,48) and suggest that this may be mediated, in part, by the favorable influence of muscular strength on large artery stiffness.

Although initial intervention studies showed an increase in central artery stiffness with RT (12,37), there is now ample evidence to suggest that RT may not increase arterial stiffness. Several recent studies have reported no change (6,11,25,34,44,52) or improvements (19, 39) in arterial stiffness with RT. Recent cross-sectional findings note that young strength-trained men and women have similar cPWV and carotid compliance as sedentary men and women (23,26). Moreover, several studies have recently demonstrated improvement in other measures of vascular function such as endothelial function and limb blood flow following RT (2,9,25,40). Thus, contrary to previous reports on the negative relationship between RT and arterial stiffness, our findings suggest that increased muscular strength is associated with lower central artery stiffness. As noted by Casey et al. (6), the conflicting results from interventional studies regarding RT on arterial stiffness may be the result of high-intensity and concurrent high-volume protocols. Appropriately prescribed RT protocols may not cause unfavorable changes to the CV system. Different types of RT (body building vs power lifting vs fitness training) may impact the CV system in different ways. However, we did not collect data on what type of RT our participants were doing (if any); thus, we could not evaluate if different forms of RT affect arterial stiffness differentially. Given the large ranges in body weight, BMI, and level of muscle strength among our participants, one might guess our sample of individuals participated in a variety of different types of RT programs. It is also possible that age may modify this relationship. However, given the young age of our participants and the narrow age range in our study, it is unlikely that age affected our findings. There was also no significant relationship between age and arterial stiffness or age and muscle strength in our study, further suggesting that it did not influence our results. Nevertheless, future studies should examine potential differences among different strength training programs empirically.

An unexpected finding of the present study was the lack of relationship between peripheral (femoral) artery stiffness and muscular strength. Previous cross-sectional studies have noted that femoral artery stiffness may be higher in strength-trained men (4), although this too is not a universal finding (26). Unlike central artery stiffness, which has been shown to increase after an acute bout of RE, peripheral artery stiffness has been shown to decrease after both the upper body (16) and lower body (28) RE. Peripheral arteries have a greater smooth muscle component, and this may be why these arteries respond differently than the more elastic central arteries to RE/RT. However, we can only speak to the lack of a relationship between peripheral arterial stiffness and muscular strength, not the effects of RT. Thus, the favorable effect of muscular strength on arterial function may be relegated only to the central arteries and not extend to the peripheral vascular bed.

Another measure of arterial health is augmentation index (AIx). AIx is related to arterial stiffness and is defined as the ratio of the amplitude of the late systolic peak to the amplitude of the early systolic peak (47). AIx has been shown to be predictive of severe CV events (50). High cardiorespiratory fitness is associated with low AIx (15). Although most studies note that RT has no effect on AIx (6,7,25), the association of muscular strength with AIx was previously unknown. Consistent with findings obtained from RE interventions, we noted no cross-sectional association between muscular strength and AIx. Although muscular strength was associated with lower cPWV, this does not similarly translate into an inverse association between muscular strength and AIx. As stated previously, cPWV is a significant determinant of AIx. However, pressure from wave reflections can be influenced by several other factors, including left ventricular contractility, peripheral artery stiffness, peripheral vascular resistance, and endothelial function/arteriolar tone (38), and these other factors may be responsible for the lack of association between AIx and muscular strength in the present study as well as previously reported associations between AIx and cardiorespiratory fitness.

The question arises, what is the mechanism responsible for the association of muscular strength and aortic stiffness? It is possible that high muscular strength is a surrogate for low CV risk factor burden (i.e., low BP, low LDL cholesterol, high HDL cholesterol, low body fat, low inflammation, increased lean tissue, increased insulin sensitivity, etc.). These traditional and nontraditional risk factors may partly mediate the noted association between muscular strength and arterial stiffness. Indeed, there was a slight but nonstatistical difference in MAP between the low cPWV

group and the high cPWV group (P = 0.067; Table 2). Stiffness of an artery is influenced by the BP (distension pressure) and the structure of the vessel wall as well as the functional components of the smooth muscle cells. Arterial stiffness varies nonlinearly with distending pressure (MAP). With a rise in mean distension pressure, load bearing is transposed from more compliant elastin fibers to stiffer collagen fibers, passively increasing vascular stiffness. Many clinical studies that examine BP as a main outcome find small differences (~2–4 mm Hg) in BP to be clinically significant. It is likely that our study was underpowered to detect significant differences in BP between our groups stratified according to PWV. More research is needed to examine the mechanistic underpinnings of the association between muscular strength and arterial stiffness.

There are some limitations with the present study. First, women were excluded from this study, and the age range was relatively narrow (18–40 yr), which limits the generalizability of this study. Given age-associated increases in aortic stiffness and reductions in muscular strength, it is possible that the coupling between these two parameters may increase with advancing age. More research will be needed to examine this empirically. In addition, we have no measures of nutritional or exercise habits that can influence both arterial stiffness (53) and muscular strength (45). As alluded previously, hematological markers associated with vascular health would have also provided more insight regarding this relationship between arterial stiffness and muscular strength. However, we do not have data pertaining to fasting glucose or blood lipids, and this is a limitation. We did not carefully characterize the genesis of strength in our participants, and this too is a limitation. Whether "strength" arising from a particular type of RT program (i.e., bodybuilding vs power lifting vs fitness training) or a genetically inherent trait is superior to other forms with respect to having favorable effects on vascular properties requires additional study. All that can be ascertained from present findings is that upper body muscular strength *per se* is associated with lower aortic PWV, and this is independent of any possible association with cardiorespiratory fitness.

In conclusion, we were able to show an unexpected inverse relationship between central arterial stiffness and muscular strength. Therefore, the results of this study support current guidelines for adults to engage in regular RE as a means of improving muscular strength and lowering the risk for mortality and CV death.

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