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Vascular Remodeling in Response to 12 wk of Upper Arm Unilateral Resistance Training

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Abstract

Participation in regular aerobic exercise has been shown to increase arterial size and that exercise-induced vascular remodeling may be regional rather than systemic. However, these issues have been minimally investigated concerning resistance training.

Purposes—To determine whether 1) resistance training of the nondominant arm elicits an increase in diameter of the brachial artery and 2) unilateral training induces arterial remodeling in the contralateral arm.

Methods—Twenty-four previously untrained participants, consisting of 18 females (aged 22.3 ± 5.1 yr) and 6 males (aged 21.7 ± 1.8 yr), participated in unilateral strength training of the biceps and triceps for 12 wk using their nondominant arm. Isotonic (one-repetition maximum, 1RM) and isometric (ISO) strength of the biceps were assessed before and after training on both arms. Brachial artery diameter and biceps muscle cross-sectional area (CSA) of both arms were also measured before and after training using magnetic resonance imaging (MRI).

Results—Brachial artery diameter increased 5.47% ($P < 0.05$) in the nondominant trained arm with no change observed in the dominant untrained arm. Biceps CSA increased 18.3% ($P < 0.05$) in the trained arm with no change ($P > 0.05$) in the untrained limb. Nondominant 1RM and ISO strength increased by 35.1% and 16.8%, respectively ($P < 0.05$ for both), although there were no significant changes ($P > 0.05$) in the contralateral arm. A modest correlation was found between the increases in CSA and in brachial artery diameter ($r^2 = 0.19$, $P = 0.039$).

Conclusions—These results indicate that upper arm vascular remodeling, manifesting as increased brachial artery diameter, can result from resistance training and that these changes are localized to the trained limb and associated with increases in CSA.

Keywords

BRACHIAL ARTERY; MAGNETIC RESONANCE IMAGING (MRI); ARTERIAL DIAMETER; CROSS-SECTIONAL AREA (CSA)

Exercise-induced vascular remodeling, as manifested in an increased arterial diameter, is generally accepted as an adaptive response to mediate the increases in transmural pressure and vessel wall stress associated with exercise training (14,20). As early as 1893, Thoma (27) observed a relationship between blood flow and vessel caliber. A classic study by Langille and O'Donnell (13) demonstrated that a chronic *reduction* in blood flow resulted in a significant reduction in vessel diameter. Further, the reduction in vessel diameter was shown to be endothelium-dependent. Other studies have found that *increases* in arterial diameter in response to increased blood flow are induced by an increased expression of endothelial nitric oxide synthase secondary to greater shear stress (8,10,14,23,30,31). More simply stated, it seems that the exercise training-induced enlargement of conduit vessels, i.e., arteries, is a chronic adaptation stimulated by nitric oxide release from the endothelium in response to increased shear stress. It is thought that this adaptive response serves to mediate the increases in transmural pressure and wall stress associated with regular exercise training and to restore shear stress values to baseline (8,14,30).

Cross-sectional studies of endurance athletes have shown an association between aerobic exercise training and improved vascular function as measured by flow-mediated dilation (FMD) (11,19,26) and/or an increased arterial diameter when compared with sedentary controls (6,9,12,22, 24,32). Increased FMD in response to training is less frequently reported in apparently healthy individuals, and there is some evidence that increases in FMD and arterial diameter may represent a continuum with arterial remodeling superseding FMD as the terminal adaptation (29). The assumption is that the increase in FMD dilation serves as a short-term adaptation to normalize shear stress levels until arterial remodeling affects a more permanent return to baseline levels (29).

Athletes such as distance runners and cyclists, whose training primarily involves the large muscles of the legs, have more consistently demonstrated an increased arterial diameter in the femoral as opposed to the carotid, aortic, subclavian, or brachial arteries (5–7,9,12,24). Interventional studies using dynamic aerobic exercise support these findings with significant increases in arterial diameter associated with training (4,6,15,16). The outcomes of these studies also suggest that increases in arterial diameter are more likely to be regional rather

than systemic (6,16). For example, Miyachi et al. (16) showed that one-legged cycle training resulted in a significant increase ($P < 0.05$) in femoral artery diameter in the trained but not the untrained leg in 10 previously untrained young men.

Resistance/strength training is an increasingly popular form of regular exercise, either alone or as an adjunct to an aerobic exercise program. It is well recognized that an appropriate resistance training program provides additive health and fitness benefits by increasing bone density and improving muscle tone and strength (1,2). However, its ability to promote vascular remodeling is not clear. Whereas resistance exercise increases transmural pressure and shear stress, it is associated with intermittent, postcontraction increases in blood flow, under high pressure compared with the continuous increase in blood flow under relatively low pressure seen with aerobic exercise. As such, resistance training may or may not produce the same vascular stimulus.

The few investigations of the effects of dynamic resistance training on vascular adaptations in apparently healthy populations have been equivocal. Cross-sectional studies comparing strength-trained individuals with sedentary controls have provided conflicting results. Bertovic et al. (3) found no group difference in transverse aortic diameter between untrained and strength-trained young men. In contrast, Miyachi et al. (18) measured femoral artery diameter in young and middle-aged men who were classified as either sedentary or resistance-trained. Compared with young and middle-aged sedentary men, femoral artery diameter was significantly greater in both age groups of resistance-trained men. Consistent with aerobic training studies, these limited data suggest that vascular remodeling in response to chronic resistance exercise may be localized to the limbs or muscles involved.

Interventional studies exploring the ability of resistance training to induce vascular remodeling are also limited and equivocal. Four months of supervised whole-body resistance training of 14 young men (aged 20–38 yr) significantly increased 1RM for all of the muscle groups tested, ranging from 20% to 47%, but did not increase brachial or carotid luminal diameter (17). In contrast, 12 wk of whole-body resistance training of 28 previously untrained young men (aged 23 ± 3.9 yr) resulted in a significant 5% increase in brachial artery diameter. Interestingly, this increase was observed at week 6 with no further changes after training (week 13) (21).

On the basis of these collective data, we hypothesized that resistance training would induce vascular remodeling, as manifested in an increased arterial diameter, and that this increase would only be observed in the trained arm and not the control/untrained arm. Therefore, the purposes of this study were to 1) determine whether brachial artery diameter increases in response to 12 wk of supervised unilateral resistance training of the nondominant arm and 2) compare the changes in brachial artery diameters between the trained nondominant arm and the untrained dominant arm.

METHODS

The data for this project were derived from a subset of participants from the Functional SNPs Associated with Muscle Size and Strength (FAMuSS) study (28). The FAMuSS Study

is a multisite controlled unilateral biceps resistance exercise study assessing four specific variables: 1) baseline biceps muscle strength, 2) baseline biceps muscle cross-sectional area (CSA), 3) posttraining biceps muscle strength, and 4) posttraining muscle CSA. The goal of the study is to search for relationships between these muscle traits and specific genetic markers (single nucleotide polymorphisms or SNP). The well-controlled design of the FAMuSS study also provided a unique opportunity to determine whether 12 wk of unilateral resistance training of the nondominant arm induces vascular remodeling and, if so, whether remodeling is confined to the trained limb or is also evident in the untrained limb, suggesting a systemic adaptation. The methods for the FAMuSS project have been detailed previously by Thompson et al. (28). However, a brief description is presented below:

Participants

Participants were apparently healthy adult males ($n = 6$) and females ($n = 18$). Baseline characteristics of the participants are presented in Table 1. Participants were excluded if they were <18 or >40 yr old; used medications known to affect skeletal muscle such as corticosteroids; had any restriction of activity; had chronic medical conditions such as diabetes; had metal implants in arms, eyes, head, brain, neck, or heart that would prohibit magnetic resonance imaging (MRI) testing; had performed strength training or employment requiring repetitive use of the arms within the prior 12 months; consumed on average more than two alcoholic drinks daily; or had used dietary supplements reported to build muscle size/strength or to cause weight gain such as protein supplements, creatine, or androgenic precursors. Before initiating the study, all participants were informed of all procedures and risks associated with the study and signed an informed consent in accordance with the Institutional Review Board for Human Subjects Experimentation at Central Michigan University.

Isometric biceps strength testing

Isometric strength of the biceps brachii of each arm was determined before and after 12 wk of strength training using a specially constructed, modified preacher bench and strain gauge (Model 32628CTL; Lafayette Instrument Company, Lafayette, IN). Baseline measures of isometric strength were assessed on three separate days, spaced no more than 2 d apart to control for learning effect. Posttraining measures of isometric strength were assessed immediately before the last training session and 48 h after the last training session. On each of the testing days, three maximal contractions were performed with each arm. To obtain three consistent peak force values, up to two more contractions were performed if a peak value deviated by more than 2.25 kg from the other two peak values. The average of the results obtained on the second and third pretraining testing days were used as the baseline criterion measurement, and the results obtained 48 h after the last training session were used for the posttraining criterion measurement.

One-repetition maximum biceps strength testing

Isotonic muscle strength of the elbow flexors of each arm was assessed before and after training by determining the one-repetition maximum (1RM) on a standard preacher bench. Baseline 1RM testing was performed during either the second or third strength testing visits.

Posttraining 1RM was measured 48–72 h after the final training session (either 48 h before or immediately after the MRI measurement to avoid the effect of prior testing on muscle size).

The 1RM test protocol was modified from Baechle (2). Briefly, each subject performed two warm-up sets with increasing weight, with 3 min of rest between each set. During the warm-up and the test itself, each subject was instructed to move through a full range of motion starting at 180° to full flexion. Care was taken to ensure that the subject completed the full range of motion. After the warm-up, subjects were instructed to attempt one full contraction at a higher weight. If the attempt was successful, the subject rested for 3 min and then performed another attempt at a higher weight (increments of 0.5–1.0 kg). This process was continued (usually three to five attempts) until they were unable to perform a full contraction. If the initial attempt was not successful, the weight was decreased and the same pattern was used. The test was terminated when the subject completed a full contraction with a given weight and failed at the next attempt.

Exercise training

Subjects underwent a gradually progressive, supervised strength training of their nondominant arm, as follows:

Weeks 1–4: three sets, 12 reps at 65%–75% of 1RM

Weeks 5–8: three sets, 8 reps at 76%–82% of 1RM

Weeks 9–12: three sets, 6 reps at 83%–90% of 1RM

The 1RM measured during pretraining testing was used to estimate the weight that could be lifted for 12, 8, and 6 repetitions using standard formulas (2). This protocol was designed to increase both muscle size using high repetitions, low intensity early in training, and strength using low repetitions, high intensity as training progressed (25). The primary interest was to train the elbow flexors, but the elbow extensors were trained as well to balance muscle strength across the joint.

All exercises were performed with the nondominant arm only. The exercises consisted of the biceps preacher curl, biceps concentration curl, standing biceps curl, overhead triceps extension, and triceps kickback. All exercises were performed with dumbbells (Powerblocks, Intellbell, Inc., Owatonna, MN), and some exercises used a preacher curl bench (Yukon International, Inc., Cleveland, OH). All training sessions were supervised and lasted approximately 45–60 min.

Arterial size: measurement of cross-sectional area

MRI (GE Signa 1.5T; GE Medical Systems, General Electric Company) was performed before and after training to assess brachial artery diameter and biceps CSA. Subjects were positioned in the magnet in the supine position with hands supinated. A phased array torso coil (GE Medical Systems, General Electric Company) was positioned so that it completely covered the antecubital fossa of the arm being scanned. A finger pulse probe was used for cardiac gating purposes.

An axial two-dimensional vascular time of flight was used to obtain the long axis of the brachial artery. This image allowed the technician to locate a point 1 cm above the bifurcation of the brachial artery. This was the landmark that was used for all subjects. Once the bifurcation was visualized, a sequential fast image slice acquisition time (ISAT) pulse that was spoiled gradient (SPGR) gated was used to obtain an image of the brachial artery echo time (TE) = minimum, repetition time (TR) = minimum, 60° flip angle, 2 mm by 0 skip, 256 × 224 matrix resolution, phase field of view 0.75, inversion number of excitation (INEX). MR images were then transferred to a compact disc for offline analysis.

Muscle size: measurement of CSA

MRI measurements were performed before and after exercise training to assess changes in the biceps brachialis CSA as previously described (28). Because of the concern that postexercise swelling can spuriously increase MRI measurements, pre-training and posttraining MRI were performed before or 24 and 48 h after the isometric or 1RM tests, respectively, and posttraining MRI was performed 48–96 h after the final training session. This ensured that temporary exercise effects, such as water shifts, were avoided while avoiding any reduction of muscle size from detraining. Pre- and posttraining MR images were obtained separately from both the dominant (untrained) and nondominant (trained) arms.

Because MR images were collected before and after training, it was important that each subject's positioning within the MR magnet be reliably reproduced to avoid coregistration errors. To accomplish this, the maximum circumference of the upper arm (i.e., the belly of the muscle) was measured with a vinyl, nonstretchable tape measure. The arm was abducted 90° at the shoulder, flexed 90° at the elbow, and the biceps maximally contracted for this measurement. The location of the maximum circumference, or the point of measure (POM), was then marked on the subject's skin using a radiographic bead (Beekley Spots; Beekley Corp., Bristol, CT). The radiographic bead was also used to standardize MRI measurements by comparing its measured CSA with that of the MRI-determined CSA.

Subjects had both arms scanned in the supine position with the arm of interest at their side and the center of the arm as close to the magnetic isocenter of the scanner as possible, palms facing up. The hand was supinated and taped in place on the scanner bed surface, and the POM centered to the alignment light of the MRI. A coronal scout image (six to nine slices) was obtained to locate the long axis of the humerus, followed by a sagittal scout image (six to nine slices) to align the eighth slice of the axial/oblique image with the POM. Fifteen serial fast-spoiled gradient images of each arm was then obtained (TE = 1.9 s, TR = 200 ms, flow artifact suppression, 30° flip angle) using the POM as the centermost point. These axial/oblique image slices (i.e., perpendicular to the humerus) began at the top of the arm and proceeded toward the elbow such that the belly of the muscle occurred at slices 8 and 9. Individual image slices were 16 mm thick with a 0-mm interslice gap, 256 × 192 matrix resolution, 22 cm × 22 cm field of view, number of excitations (NEX) × 6. This method images a 24-cm length of each arm. MR images were transferred to the central MRI facility at Yale University via either magneto-optical disk or compact disc read-only memory. Images were analyzed using a custom-design interactive processing and visualization

program that operates in Matlab (The Math Works, Inc., Natick, MA) running on a Windows-based personal computer platform. This software enables the user to assign regions of interest (ROI) in an image set by tracing region borders with a mouse. The muscle is easily identifiable on MR images, and its CSA is measured using this computerized planimetry technique. Once the ROI was defined, the program reported the number of pixels contained in the selected ROI. Based on the MR acquisition data (i.e., field of view and matrix resolution), the CSA (cm²) of the defined ROI was then calculated. When the pretraining CSA was subtracted from the posttraining CSA, the training effect could be compared between subjects. To optimize the accuracy of the muscle size calculation, a subset of data was analyzed by volumetric analysis. By analyzing the 15 successive slices throughout the scanned length of the upper arm each CSA was multiplied by the known slice thickness (1.6 cm) to yield a slice volume (cm³). Slice volumes were then summed over the length of the anatomical structure of interest.

Statistical analyses

To examine pre- and posttraining differences, two-tailed, paired *t*-tests were used to test for significance. In addition, Pearson correlations were used to determine significant relationships between variables of interest. Statistical significance for all tests was set at $P < 0.05$.

RESULTS

The descriptive characteristics for all participants, before and after training, are presented in Table 1. Briefly, in the trained arm, 12 wk of progressive resistance training significantly increased ($P < 0.05$) the diameter of the brachial artery, CSA of the biceps brachii, and both ISO and 1RM strength by 5.5% (axial plane), 18.3%, 16.8%, and 35.1%, respectively. There were no significant changes in these variables for the untrained arm or in any other variables including body weight.

In the ND-trained arm, correlations were found between pretraining brachial artery diameter and baseline measures of muscle CSA, as well as ISO and 1RM ($r^2 = 0.20$, $P = 0.027$; $r^2 = 0.21$, $P = 0.023$; and $r^2 = 0.19$, $P = 0.042$, respectively). In addition, the training-induced increase in CSA was correlated with the increase in brachial artery diameter ($r^2 = 0.19$, $P = 0.039$). In the dominant untrained arm, only baseline ISO strength was correlated with brachial artery diameter ($r^2 = 0.20$, $P = 0.030$).

DISCUSSION

This is the first study to examine the effects of a supervised program of unilateral upper arm resistance training on the diameter of the brachial artery. The most salient finding was that 12 wk of unilateral biceps resistance training produced a significant increase in the diameter of the brachial artery in the trained arm. In addition, the increase in brachial artery diameter was modestly associated with the increase in muscle CSA. These results demonstrate that resistance training can elicit vascular adaptations that were previously found primarily through aerobic exercise. These data also suggest that the vascular remodeling that occurs

with resistance training is specific to the limbs and/or muscles that are trained and associated with changes in muscle CSA.

Resistance training and arterial diameter

Twelve weeks of progressive unilateral resistance training of the biceps produced a 5.47% increase in the diameter of the brachial artery in the trained but not in the untrained arm. Miyachi et al. (18) reported that, compared with young and middle-aged sedentary men, femoral artery diameter was significantly greater in both age groups of resistance-trained men. Specifically, the femoral artery diameter of strength-trained young men was 3.3% greater than that of sedentary age-matched controls. Of equal if not greater interest, the femoral artery diameter of middle-aged resistance-trained men was greater than both sedentary young and middle-aged men (7.8% and 9.0%, respectively; $P < 0.05$). In contrast, Bertovic et al. (3) found no group difference in transverse aortic diameter between untrained and strength-trained young men. These very limited data suggest that vascular remodeling associated with resistance training, such as aerobic exercise training, may be more regional than systemic.

Interventional studies using resistance training are not only few in number but also equivocal. Miyachi et al. (17) reported no change in carotid luminal diameter after 4 months of a typical supervised whole-body resistance training of 14 young males (aged 20–38 yr) despite significantly increased 1RM for all of the muscle groups tested, ranging from 20% to 47%. In another study using a whole-body resistance training program, 28 previously untrained young men (aged 23 ± 3.9 yr) performed 12 wk of resistance training 5 d·wk⁻¹ using a repeating split-body 3-d cycle (21). Resting brachial artery diameter was measured at baseline, week 6 of training, and again at week 13. At week 6, mean brachial artery diameter increased ~5% but did not demonstrate further changes after that point.

A modest association ($P < 0.05$) was demonstrated between the increases in muscle CSA and brachial artery diameter in the trained arm. Although muscular strength (ISO and 1RM) was correlated with brachial artery diameter at baseline, the changes in strength did not show an association with the increase in brachial artery diameter. These data suggest that training and/or adaptations that induce muscular hypertrophy, but not necessarily strength, may also serve to increase brachial artery diameter. It is generally accepted that training volume, more so than the intensity of the training *per se*, is more conducive to muscle hypertrophy. It is also possible that resistance training that emphasizes volume and more muscle contractions may also be more likely to induce vascular remodeling. However, more work is needed in this area before definitive statements can be made about the nature of the training stimulus and arterial remodeling.

Aerobic exercise training and arterial diameter

The positive association between aerobic exercise training and arterial diameter is documented, with cross-sectional studies demonstrating 7% to 21% greater diameter of the femoral artery in endurance-trained athletes compared with sedentary controls (6,9,24). These studies, however, did not observe between-group differences in the brachial (6), aortic (9), subclavian (9), or common carotid (24) arteries. Because most athletes in these studies

were distance runners and cyclists, vascular remodeling may be primarily regional. Tinken et al. (29) have recently suggested that the lack of evidence for generalized remodeling may be related to the method used to assess arterial remodeling. They suggest, for example, that simple resting measures may be influenced by changes or variations in vasoconstrictor tone. They further recommend that inducing maximal/peak diameter change associated with reactive hyperemia is a better index of arterial remodeling because it is not influenced by this and other potential confounds.

Interventional studies have demonstrated that aerobic exercise training may induce vascular remodeling and increases in arterial diameter. Miyachi et al. (15) measured CSA of both the ascending and abdominal aorta before and after 8 wk of vigorous exercise training. The exercise training group showed increases of approximately 20% for both the ascending and abdominal aorta. Three months of aerobic leg training, primarily walking, resulted in a 6.7% increase in $\dot{V}O_{2\max}$ and a 9% increase in femoral artery diameter in a group of 22 previously untrained middle-aged men with no change in brachial artery diameter (6).

Collectively, these data not only support the hypothesis that regular aerobic exercise induces vascular remodeling but also this adaptation seems to be largely regional and specific to the conduit vessels in the exercising limbs and/or muscles. This was most clearly demonstrated by Miyachi et al. (16) who used unilateral cycling exercise to examine changes in femoral artery diameter in both the trained and untrained legs. Six weeks of one-legged cycle training resulted in a 16% increase in femoral artery CSA but no changes in the control leg.

Limitations and future directions

This study only examined the gross change in brachial artery diameter in response to resistance training. Changes in brachial artery structure such as intimal medial thickness as well as hemodynamic responses such as FMD were not examined. As such, we do not know if the increase in brachial artery diameter was due to changes in arterial structural, vascular tone, or both.

The duration of the present study was only 12 wk. Although a previous work (21) suggests that vascular remodeling with a whole-body resistance training protocol occurs within the first 6 wk of training, with no further changes at 12 wk, it is possible for further adaptive responses to occur with further training.

In summary, 12 wk of supervised unilateral biceps resistance training significantly increased brachial artery diameter by ~5.5% in the trained arm with no change in the con-tralateral arm. Muscular strength and CSA also increased significantly in the trained, but not the untrained, arm. A modest but significant correlation was found between the changes in muscle CSA and brachial artery diameter. These data suggest that resistance training of the elbow flexors not only increases muscle size and strength but may also induce vascular remodeling resulting in greater arterial diameter. These findings are also consistent with previous cross-sectional and interventional studies suggesting that vascular remodeling associated with exercise training, in general, and resistance training, in particular, is regional in nature and specific to the conduit vessels in the limb and/or muscles involved. Finally, the increase in brachial artery diameter was associated with the increase in muscle CSA.

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TABLE 1Descriptive characteristics for all subjects ($n = 24$) before and after training.

	Baseline	After Training
Age (yr)	22.2 $\pm$ 4.83	22.5 $\pm$ 5.16
Body weight (kg)	71.12 $\pm$ 15.68	71.30 $\pm$ 14.86
Height (cm)	167.51 $\pm$ 7.14	167.49 $\pm$ 7.11
Brachial artery diameter measured axially (cm)		
Untrained (DOM) arm	0.400 $\pm$ 0.088	0.402 $\pm$ 0.087
Trained (ND) arm	0.402 $\pm$ 0.078	0.424 $\pm$ 0.077 ^{*‡}
Brachial artery diameter measured coronally (cm)		
Untrained (DOM) arm	0.396 $\pm$ 0.094	0.397 $\pm$ 0.095
Trained (ND) arm	0.404 $\pm$ 0.083	0.425 $\pm$ 0.087 ^{*‡}
Biceps CSA (cm ²)		
Untrained (DOM) arm	15.05 $\pm$ 4.04	14.91 $\pm$ 4.12
Trained (ND) arm	14.81 $\pm$ 3.95	17.64 $\pm$ 5.63 ^{*‡}
Isometric strength (kg)		
Untrained (DOM) arm	31.81 $\pm$ 13.08	32.58 $\pm$ 13.88
Trained (ND) arm	29.76 $\pm$ 13.57	34.76 $\pm$ 13.52 [*]
Isotonic strength (1RM; kg)		
Untrained (DOM) arm	9.76 $\pm$ 4.15	10.37 $\pm$ 4.43
Trained (ND) arm	9.67 $\pm$ 4.30	13.05 $\pm$ 4.41 [*]

Values are means $\pm$ SD.^{*}Significant ($P < 0.05$) difference between pre- and posttraining values.[‡]Significant ($P < 0.05$) difference between trained and untrained arms at same time point.

DOM, dominant; ND, nondominant.