Effect of resistance training on muscle use during exercise

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Ploutz, Lori L., Per A. Tesch, Ronald L. Biro, and Gary A. Dudley. Effect of resistance training on muscle use during exercise. *J. Appl. Physiol.* 76(4): 1675–1681, 1994.—This study examined the effect of resistance training on exercise-induced contrast shift in magnetic resonance (MR) images. It was hypothesized that a given load could be lifted after training with less muscle showing contrast shift, thereby suggesting less muscle was used to perform the exercise. Nine males trained the left quadriceps femoris (QF) muscle 2 days/wk for 9 wk using 3–6 sets of 12 knee extensions each day. The right QF served as a "control." Exercise-induced contrast shifts in MR images evoked by each of three bouts of exercise (5 sets of 10 knee extensions with a load equal to 50, 75, and 100% of the maximum pretraining load that could be lifted for 5 sets of 10 repetitions) were quantified pre- and posttraining. MR image contrast shift was quantified by determining QF cross-sectional area (CSA) showing increased spin-spin relaxation time. One repetition maximum increased 14% in the left trained QF and 7% in the right untrained QF. Left QF CSA increased 5%, with no change in right QF CSA. Left QF CSA showing contrast shift was less after each bout of the exercise test posttraining. This was also true, to a lesser extent, for the right QF at the higher two loads. The results suggest that short-term resistance training reduces MR image contrast shift evoked by a given effort, thereby reflecting the use of less muscle to lift the load. Because this response was evident in both trained and contralateral untrained muscle, neural factors are suggested to be responsible. The consequence of this adaptation could be to increase "stress" per unit area of active muscle during the course of training and thereby evoke hypertrophy.

magnetic resonance imaging; neuromuscular adaptation

INCREASES IN STRENGTH in excess of what can be explained by muscle hypertrophy have frequently been reported to occur early in resistance training (22, 23, 27, 30, 32, 36). The findings that maximal integrated electromyogram (iEMG) is increased and the slope of the iEMG-force relationship at submaximal loads is decreased after training have been used to suggest that neural factors account for the increased strength (8, 20, 32). These are not universal findings, however, as others report no change in iEMG after training (5, 19). Instead, some authors have suggested that resistance training alters muscle architecture (increased angle of pennation), which allows for greater force to be generated without a greater muscle cross-sectional area (CSA) (24, 25). Adding to the confusion is the suggestion that pennation angle may be neglected when muscle force output is estimated (37). Regardless of the mechanism, the consequence of an increase in strength with little or no increase in muscle size should be that less muscle is used to lift a given submaximal load after training. This, however, has not been previously demonstrated as there has been no method to quantify muscle use in an absolute sense.

Magnetic resonance (MR) imaging allows for unparalleled visualization of whole muscle groups, as well as the individual muscles of a given limb segment. Additionally, exercise-induced contrast shifts in spin-spin relaxation time (T_2)-weighted images are reflective of muscle use (1, 2, 15–18, 40, 44). The extent of increase and the absolute value of surface iEMG and T_2 have been shown to be correlated with resistance exercise intensity (load) (1). Additionally, contrast shifts are greater with concentric than eccentric contractions, in concert with the knowledge that muscle activation (iEMG) and use (MR imaging) are greater for concentric contractions (1, 40). Recently, a method has been developed that allows the area of muscle showing such a contrast shift in T_2-weighted MR images to be quantified and thus yields an absolute area of muscle that has previously performed contractile activity. Adams et al. (2) have demonstrated that the CSA of muscle demonstrating such a contrast shift has physiological relevance because it was directly related to force development during isometric contractions of the quadriceps femoris (QF) muscle evoked by surface electromyostimulation.

The interest of this study was to use the powerful technology of MR imaging to examine the effect of short-term resistance training on muscle use during exercise. Resistance training was designed to increase QF strength, yet evoke modest skeletal muscle adaptations that could account for the increase in strength or the expected reduction in exercise-induced contrast shift. For this same reason, the trained and contralateral untrained QF muscles were studied. It was hypothesized that a given load would be lifted after training with less QF showing contrast shift, thereby suggesting less muscle was used to perform the exercise. It was also hypothesized that this would occur in the contralateral untrained QF if the cross-over effect with regard to strength was realized. The results support both hypotheses. It is suggested that this reflects an alteration in neural activation after training because hypertrophy or changes in architecture would not be expected to provide for more advantageous force transmission in the untrained QF. The physiological consequence of continuously using less muscle early in resistance training to lift a given load is suggested to be increased stress per unit area of "active" muscle, thereby gradually evoking hypertrophy.

METHODS

General design. Subjects participated in four to five orientation sessions over 3 wk to familiarize them with knee extension

TABLE 1. *General design of exercise test and MR imaging schedule pre- and posttraining*

Exercise
Unilateral knee extensions with QF muscle 5 × 10 with each leg, alternating between right and left QF
Test schedule
MR images of QF at rest
5 × 10 with 50% of pretraining 5 × 10 RM load
MR images of QF
45 min rest
5 × 10 with 75% of pretraining 5 × 10 RM load
MR images of QF
45 min rest
5 × 10 with 100% of pretraining 5 × 10 RM load
MR images of QF

MR, magnetic resonance; QF; quadriceps femoris; 5 × 10, 5 sets of 10 repetitions; RM, repetition maximum.

exercise using unilateral concentric contractions. The interest was to train them to exercise, not exercise train them. After orientation, two biopsies were obtained from the left vastus lateralis muscle to evaluate fiber type, fiber area, and fiber type-specific succinate dehydrogenase (SDH) activity. Three days later subjects were tested for 5 × 10 repetition maximum (RM), the heaviest load that could be lifted for 5 sets of 10 repetitions with 2 min between sets, and 1 RM, the heaviest load that could be lifted one time. Three days later subjects performed an exercise test using each of three intensities of knee extension exercise (50, 75, and 100% of 5 × 10 RM), and exercise-induced contrast shifts in MR images of the QF muscles were quantified after each bout. After all pretesting, subjects trained the left QF 2 days/wk for 9 wk using concentric knee extensions. After training, the muscle biopsies and the 1 RM were repeated. Subsequently, the exercise test and MR imaging were repeated with the same absolute loads that were used pretraining (50, 75, and 100% of pretraining 5 × 10-RM load).

Subjects. The subjects consisted of nine males [77 ± 3 (SE) kg] who were untrained in lower-body resistance exercise. The exercise history of the subjects ranged from sedentary for >15 yr to active but not currently participating in organized resistance training. The procedures, purpose, and risks associated with participation were explained, and informed written consent was obtained. This study was approved by the Human Research Review Board at the Kennedy Space Center, Florida.

Strength testing. Strength testing and training were conducted on a modified Nautilus knee extension machine. The machine was equipped with a hydraulic device that lowered the weight so that subjects performed concentric contractions only. Subjects participated in approximately five orientation sessions designed to familiarize them with knee extension exercise so that reasonable measures of the 5 × 10-RM load and the 1-RM load could be obtained. After orientation, the left and right QF muscles were separately tested for the 5 × 10-RM load. This strength measure was obtained to establish the loads used for the exercise test (see below). Five days later, each QF was separately tested for the 1-RM load. After training, the 1-RM load was again determined to measure strength after 9 wk of training.

Exercise test protocol. The tests were performed at the MR imaging facility so that subjects could be imaged immediately (~1.5 min) after exercise. Subjects performed 5 sets of 10 unilateral concentric contractions on the modified Nautilus knee extension machine at each of three intensities: 50, 75, and 100% of the pretraining 5 × 10-RM load (Table 1). Two minutes of rest were taken between sets and 45 min between bouts. MR images of both QF muscles were taken at rest before any exercise and immediately after completion of the five sets with a given load. For example, subjects performed 5 sets of 10 repetitions with each QF muscle, alternating legs, with the 50% of the pretraining 5 × 10-RM load. MR images of both QF muscles were taken after the last set. Subjects rested in a seated posture for 45 min, then performed 5 sets of 10 repetitions with each QF muscle with the 75% load, and again MR images were taken. This pattern was repeated with the 100% load. Fisher et al. (15) have shown that the half life of recovery of the exercise-induced contrast shift is 5–7 min (15); thus 45 min of rest ensured that 97–98% of the contrast shift had been resolved before the start of the next exercise bout. This exercise test protocol was repeated after training. Because of the increase in strength after training, an additional bout was performed with a load equivalent to 120% of the pretraining 5 × 10-RM load.

Training protocol. Subjects trained the left QF using the modified Nautilus knee extension machine. Training was designed to increase strength and yet evoke modest adaptations in skeletal muscle. Accordingly, subjects trained 2 days/wk for only 9 wks using concentric contractions. Subjects performed a warm-up set and 3 sets of 12 repetitions with a weight that induced failure within each set during *weeks 1* and *2*. Every 2 wk the number of sets was increased by one while a load was maintained that induced failure within each set. Therefore, at *weeks 7–9*, subjects were performing six sets. Two minutes of rest were taken between sets. All training was performed at the Kennedy Space Center.

Imaging techniques and analyses. MR images were collected using a 1.5-T superconducting magnet (General Electric, Milwaukee, WI). Images were taken of the QF muscles because of their marked involvement in knee extension exercise. Seventeen transaxial images 1 cm thick were obtained between the knee joint and the head of the femur at 0.5-cm intervals. Ink marks on the thigh aligned with the cross-hairs of the imager and a foot brace allowed for similar positioning of the thigh in the magnet bore for each scan. Two T_2-weighted images (repetition time = 2,000 ms, echo time = 30 and 60 ms) were collected within a 40-cm rectangular field of view body coil. A 256 × 256 matrix resolution and one excitation were used. Total scan time was 4:40 min. All MR images were transferred to a Macintosh computer for calculation of muscle CSA and T_2 using a modified version of the public domain NIH Image software package. By tracing around an image of the QF, a region of interest (ROI) was established. The QF CSA was measured from this ROI after spatial calibration. A T_2 value (2/3 of the signal decay time) was calculated for each pixel in the ROI from the formula $T_2 = (t_a - t_b)/\ln (i_a/i_b)$, where t_a and t_b are spin-echo collection times and i_a and i_b are signal levels. Resting muscle was assumed to have a T_2 value between 20 and 35 ms based on multiple observations that the resting T_2 of muscle is 28 s with an SD of 3–4 s (2). Pixels out of this range were assumed not to be muscle, and this CSA (usually ~3–4 cm^2) measured at rest was subtracted from subsequent postexercise images taken on that day. T_2 values after exercise that were greater than the resting mean + 1SD T_2 were considered to be elevated. The area of pixels showing elevated T_2 after exercise was expressed in square centimeters and taken to be the CSA of QF showing recent contractile activity. Six slices were selected, beginning with the first slice not containing gluteal muscle and continuing for the next five slices toward the knee. This section represents the area of maximal CSA of all of the individual QF muscles (32). QF CSA and "active" CSA measurements were averaged over these six slices.

TABLE 2. *QF muscle 1 RM and CSA pre- and posttraining*

	Pretraining	Posttraining
QF 1 RM, kg		
Right	71±4	76±4*
Left	70±5	80±5*
QF CSA, cm^2		
Right	85±4	84±4
Left	77±4	82±4*

Values are means ± SE. CSA, cross-sectional area. * Significantly different from pretraining values.

Biopsy techniques. Two biopsies were taken from the left QF before and after the 9 wk of training. The muscle samples were removed from the left vastus lateralis by using the pecutaneous needle biopsy technique (3), as modified by Evans et al. (14), and processed for histochemical analysis as described previously (9). Muscle fiber CSA was assessed using a modified version of NIH Image. Images were obtained with a microscope (model BH 2, Olympus Optical, Tokyo, Japan), a charge-coupled device camera (model WV-5000, Panasonic Industrial, Secaucus, NJ), a computer (Macintosh Quadra 700 with 21-in. color display, Apple Computer, Copertino, CA) with frame-grabber board, and a 512 Mbyte optical disk drive. Regions of a section were excluded from analyses if they contained oblique or histologically abnormal fibers, that is, fibers that had interrupted cell membranes, freeze damage, or were oblique, meaning they had a common orientation (21). At least 150 fibers in each section were assessed for area and type. This provided a minimum of 20 fibers of types I, IIa, IIab, and IIb. In most cases 60–80 fibers were analyzed for the type I and IIa fibers.

Sections were also assayed for SDH activity as described previously (4). The assay and a blank were run for 10 min at room temperature in the dark. Sections were air dried and mounted. Within 1 day, digital images of the sections were captured to disk for subsequent analysis. The quantitative histochemical assay for SDH was found to be linear for up to 15 min with human skeletal muscle. The change in optical density (OD) over 10 min minus the blank was used to calculate the steady-state activity of SDH in OD units (OD U/min × 10^{-4}). Fiber type-specific SDH was determined by matching fibers assayed for SDH with those in serial sections assayed for myofibrillar adenosinetriphosphatase activity. Becasue of the inherent difficulty in matching a given fiber in serial sections and because of the few type IIab and type IIb fibers pre- and posttraining, respectively (see Table 4), <20 fibers in some subjects were assessed for types IIab and IIb SDH activity. Densiometric measurements of the SDH sections were made by NIH Image after calibration of OD. Neutral density filters were used to determine the relationship between gray levels and OD. OD pixel values of a blank field were subtracted from pixel values of a given image to correct for camera field variations. All microscope illumination was provided through a narrow-pass interference filter with peak emission at 570 nm.

Statistical analyses. Repeated-measures analyses of variance (ANOVAs) were run with the BMDP statistical package. Because subjects served as their own controls, all factors were repeated. In the case of a three-factor interaction, the analyses were broken down into either two-way or one-way ANOVAs. Tukey's highly significant difference post hoc tests were used when necessary (the 1-factor ANOVA had more than 2 levels). Where missing data occurred, the computer program removed the subject from the analysis, thus necessitating that we combine the IIb and IIab fiber types for the overall SDH analyses because subjects did not exhibit both fiber types at all times.

TABLE 3. *Muscle fiber type percentage and CSA in biopsies of left vastus lateralis muscle pre- and posttraining*

	Pretraining	Posttraining
Fiber type, %		
I	40±3	39±3
IIa	36±2	33±2
IIab	8±1	22±3*
IIb	16±4	5±2*
Fiber CSA, μm^2		
I	6,237±202	6,017±459
IIa	7,505±619	8,489±812
IIab	6,819±946	7,064±605
IIb	5,513±708	5,387±415

Values are means ± SE. * Significantly different from pretraining.

TABLE 4. *Muscle fiber type succinate dehydrogenase activity in biopsies of left vastus lateralis muscle pre- and posttraining*

Type*	Pretraining	Posttraining	*P*
I	164±15 (47, 8)	170±10 (49, 8)	0.6302
IIa	139±13 (40, 8)	146±11 (34, 8)	0.5733
IIb	84±10 (29, 5)	116±20 (19, 4)	0.0891
IIab	122±17 (13, 6)	118±07 (31, 6)	0.8739

Values are means ± SE in optical density (OD) units per min × 10^{-4}. Nos. in parentheses, average no. of fibers, subjects analyzed. *P*, probability of difference pre- to posttraining for each individual fiber type. * Overall significant difference among individual fiber types.

RESULTS

Training. Subjects lifted a mean load of 51 ± 3 kg for each of 3 sets of 12 repetitions during *week 1* of training. During the last week of training, the subjects were lifting a load of 68 ± 4 kg for 6 sets of 12 repetitions.

Muscle strength and size. There was a time effect ($P = 0.0004$) for 1-RM load with no side effect ($P = 0.1634$) and no interaction ($P = 0.0711$) (Table 2). Strength increased 14% in the left trained QF and 7% in the right untrained QF.

QF CSA did not change over the 9-wk training period in the right untrained leg. The QF muscle group of the left trained leg significantly increased in size from pre- to posttraining (77 ± 4 to 82 ± 4 cm^2; Table 2).

Muscle biopsies. There was no change in fiber area for any fiber type after training ($P = 0.1132$; Table 3). There was a decrease in the percentage of type IIb fibers ($P = 0.0068$), with a concomitant increase in type IIab fibers ($P = 0.0056$). There were no changes in the percentage of type I and IIa fibers ($P = 0.6992$ and 0.2311, respectively; Table 3).

The single-fiber SDH analyses demonstrated a difference among each of three fiber types, I, IIa, and IIb + IIab, both pre- and posttraining ($P = 0.0000$ for both); however, there was no time effect (overall $P = 0.3407$). Type IIab and IIb fibers were combined for the overall model because several of the subjects did not exhibit both type IIb and IIab fibers pre- and posttraining. Because fiber type-specific SDH activity could not be evaluated in

the overall model, one-way ANOVAs were run on each of the four individual fiber types (Table 4). None showed an increase at the conventional $P < 0.05$ level.

MR image contrast shift. There was a significant three-way interaction, time × bout × side ($P = 0.0007$); therefore, two two-way ANOVAs were run and demonstrated a two-way interaction for time × bout for the right and left legs ($P = 0.0163$ and 0.0000, respectively). To evaluate main effects, one-way ANOVAs were run for time at each bout. There was no changes in the resting T_2 or QF CSA with an elevated T_2 at rest for either the right or left leg ($P = 0.7147$ and 0.4877, respectively) after training. The CSA of the left trained QF showing contrast shift, and thereby use, after performance of 5 sets of 10 repetitions of knee extensions with a load equal to 50, 75, and 100% of pretraining 5 × 10-RM load was reduced after training ($P = 0.0356$, 0.0012, and 0.0000, respectively; Fig. 1*B*). The right untrained QF showed the same response, although to a lesser extent, at the two higher intensities (75 and 100% of pretraining 5 × 10-RM load) ($P = 0.0283$ and 0.0136, respectively; Fig. 1*A*). Figure 2 shows a representative MR image taken after the 120% bout posttraining. Note that the left trained QF shows much less contrast shift, i.e., appears lighter, than the untrained right QF.

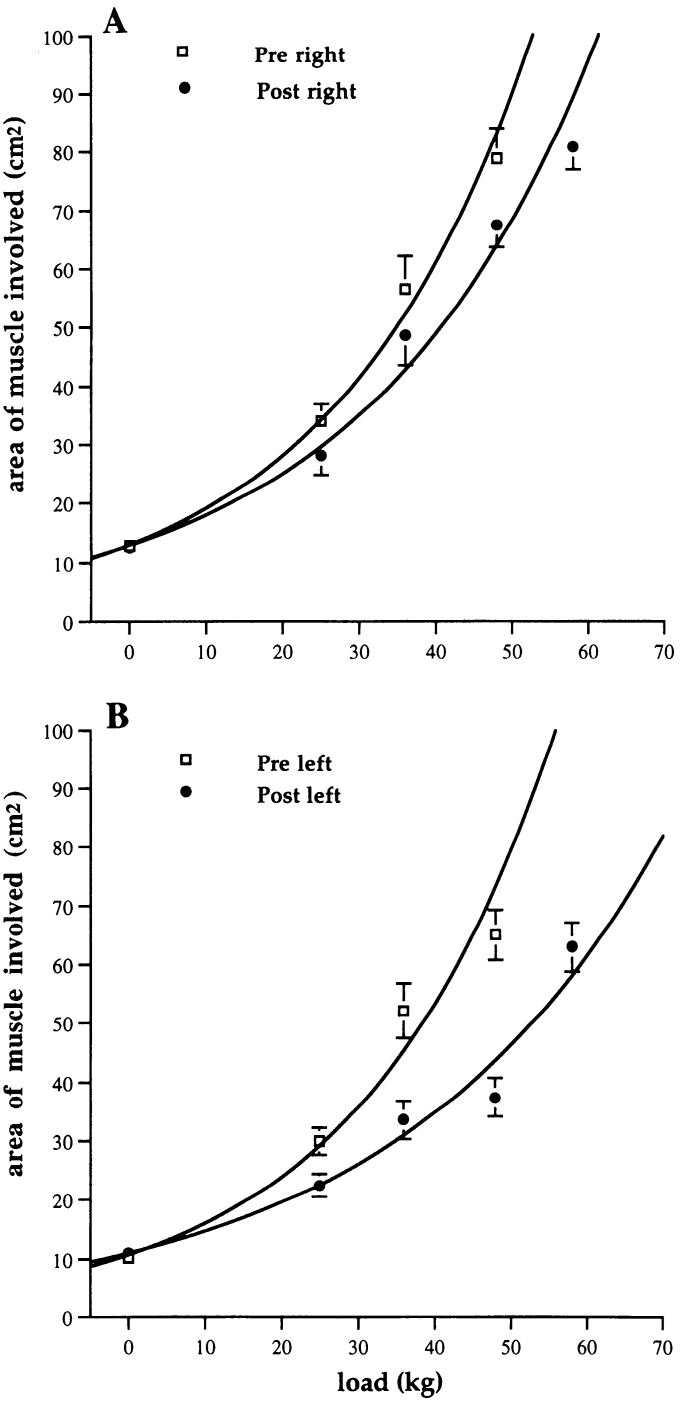

FIG. 1. Average cross-sectional area (cm^2) of right untrained (*A*) and left trained (*B*) quadriceps femoris (QF) muscles showing magnetic resonance (MR) image contrast shift, and thereby use, plotted as function of load lifted during exercise test pre- and posttraining.

DISCUSSION

The interest of the present study arose from the observation made on numerous occasions that increases in strength early in resistance training are not obligatory to muscle hypertrophy (7, 22, 23, 27, 32, 36). Although not put forth previously to our knowledge, it seemed that this would result in less muscle being used to lift a given load after training. We thus set out to address this issue using the powerful technology of MR imaging.

The resistance training used in this study was intended to increase strength without inducing marked adaptations within muscle that would confound interpretation of our MR imaging studies (see below) and/or account for improved performance. To this end, short-term training with concentric contractions was used (7, 22, 23, 27, 32, 36). The trained and contralateral untrained muscle groups were also compared pre- vs. posttraining because increases in strength in the untrained QF, if realized due to the cross-over effect (5, 12, 19, 22, 38, 39), would be highly unlikely to have occurred due to changes within the muscle. The findings in this study that the 1-RM load was increased 14% in the trained QF, with a 5% increase in QF CSA, and that the 1-RM load increased 7% for the right untrained QF with no increase in its CSA indicate that the training goals were accomplished. The nature and magnitude of these responses are also comparable to those reported previously by others (7, 12, 23, 24, 32, 38, 39).

The most important finding of this study was that short-term resistance training reduced exercise-induced contrast shift in MR images of both the trained and untrained QF muscles in both an absolute and relative sense (Figs. 1, *A* and *B*, and 2, Table 5). It is generally accepted that this contrast shift is due to recruitment of muscle and the associated metabolic demand, although the exact biochemical basis is unknown (1, 17). It is highly unlikely that the energy cost of the exercise differed to such an extent to account for the reduced contrast shift after training found in this study. It is also doubtful that alterations in the ability of the trained QF to supply or use energy during exercise were responsible for the marked reduction in contrast shift because *1*) we found no change in SDH activity in the trained QF, *2*) others have reported that short-term resistance training does not appreciably alter the ability of muscle to supply

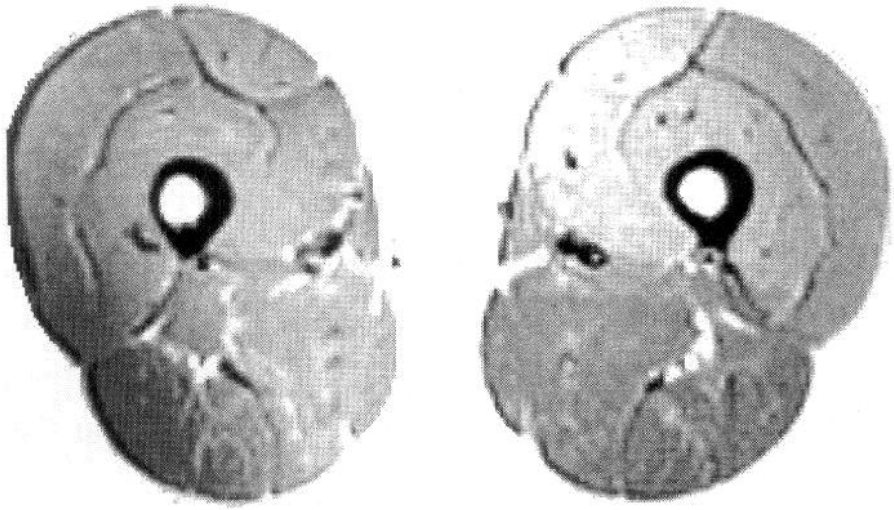

FIG. 2. Representative transaxial spin-spin relaxation time (T_2)-weighted MR image of left and right QF muscles posttraining, taken immediately after performance of 5 sets of 10 knee extensions with a load equal to 120% of pretraining 5 × 10 repetition maximum. Right QF (*right*) shows greater exercise-induced contrast shift (appears lighter) than left QF (*left*).

energy via anaerobic mechanisms (22), and *3*) muscle fiber type composition was not markedly changed by training. In addition, the finding of reduced contrast shift in the untrained QF could hardly be accounted for by skeletal muscle adaptations. Thus, we interpret the finding that the trained and untrained QF muscles showed less exercise-induced contrast shift after training to reflect an alteration in recruitment whereby less muscle was used to perform the exercise.

The findings in the present study that suggest that less muscle was used to lift the same load after training are indirectly supported by the implications of some, but not all, EMG studies. Several authors have reported a significant increase in the maximal iEMG and a decrease in the slope of the EMG-isometric tension relationship after resistance training such that, for a given submaximal load, EMG was decreased after training (20, 32). Yet, in one of these studies, the increase in maximal iEMG was nearly twofold greater in the untrained than in the trained limb (32). Several other authors have reported no change in EMG after training in which strength was increased more than could be accounted for by hypertrophy (5, 19). This controversy may be reflective of the technical difficulty in making repeat surface iEMG measurements over a prolonged time. It has been reported that the coefficient of variation is 23% for recurrent EMG measurements (43).

Alternatively, an increase in muscle fiber angle of pennation independent of hypertrophy has been suggested by some to occur early in resistance training, the result being enhanced force transmission simply by mechanical means (24, 25). This would in effect reduce the amount of muscle that would need to be recruited to lift a given submaximal load. It is unlikely, however, that this could fully account for the improved performance found in the present study because increased strength was found in the untrained contralateral QF.

TABLE 5. *Relative CSA of QF muscle showing MR image contrast shift and thereby use in exercise test pre- and posttraining*

Load	QF Muscle	Pretraining	Posttraining
50%	Right	38±3	32±3
	Left	38±3	27±2
75%	Right	62±5	54±4
	Left	62±5	39±3
100%	Right	83±4	74±3
	Left	74±4	43±3

Values are means ± SE in %. Load is percentage of pretraining 5 × 10 RM load.

The neural adaptation underlying the use of less muscle to lift a given load after training could involve desynchronization of motor unit activity (26). There is controversy regarding this concept, as one author has reported increased synchronization of motor units with resistance training (31), whereas others have shown greater tension development during more asynchronous stimulation (6, 28) or discounted the probability that synchronization could account for their finding of a greater increase in strength than muscle size after short-term resistance training (33). Desychronization among motor units would be expected to enhance force development and possibly delay the onset of fatigue (6, 28, 34, 35). As a consequence fewer motor units would have to be used to perform the same exercise after training, thereby diminishing exercise-induced contrast shifts in T_2-weighted MR images.

Muscle fiber CSA was not increased after short-term resistance training in the present study, as has been reported previously by others (7). This finding may seem at odds with the significant 5% increase in CSA of the "trained" QF. It should be appreciated that high-resolution MR images provide the opportunity to measure whole muscle CSA with remarkable precision (11, 33), especially when multiple images are analyzed as was done in this study. There is, in contrast, 10–15% variability in measurements of muscle fiber CSA with the biopsy technique because of, in large part, regional differences in fiber CSA within muscle (10). We did find a decrease in the percentage of type IIb fibers with a concomitant increase in type IIab fibers after training, consistent with the findings of others (41, 42). These results suggest that fiber type transformation among the fast-twitch subtypes can occur after training that requires performance of only several hundred intense contractions per week.

That the training performed in this study required increased use of type IIb fibers and their subsequent transformation to the type IIab isozyme is supported by our data, which show a trend toward increased SDH activity in type IIb fibers ($P = 0.089$). In light of the fact that only four subjects exhibited type IIb fibers posttraining, the power of this statistical test must be quite low. Yet there was a 91% chance ($P = 0.089$) that this was not due to random variation. The novel finding of a hierarchy of SDH activity among the I, IIa, and IIb fiber types by microdensiometric techniques (Table 4) lends credence to our method of analysis of muscle fiber aerobic enzyme content because these results are comparable to those reported for freeze-dried single fibers analyzed with microfluorometric techniques (12) and with a recent report of greater single-fiber SDH activity in type I than in type II human muscle fibers (29).

In summary, the data support our hypothesis that less muscle is required to lift the same load after short-term resistance training. Because the increase in muscle size alone cannot account for the increase in strength of the trained leg and because there was little adaptation in the muscle itself, we support the notion that neuromuscular activation is altered after training. This is further supported by the untrained "cross-educated" leg, which has previously been postulated to enjoy a neural benefit from the contralateral training. It is suggested that the physiological consequence of using less muscle to lift a given load during training may be increased stress per unit area of active muscle, which gradually induces muscle hypertrophy.

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REFERENCES

1. **Adams, G. R., M. R. Duvoisin, and G. A. Dudley.** Magnetic resonance imaging and electromyography as indexes of muscle function. *J. Appl. Physiol.* 73: 1578–1583, 1992.
2. **Adams, G. R., R. T. Harris, D. Woodard, and G. A. Dudley.** Mapping of electrical muscle stimulation using MRI. *J. Appl. Physiol.* 74: 532–537, 1993.
3. **Bergström, J.** Muscle electrolytes in man. *Scand. J. Lab. Invest.* 14: 1–110, 1962.
4. **Blanco, C. E., G. C. Sieck, and V. R. Edgerton.** Quantitative histochemical determination of succinic dehydrogenase activity in skeletal muscle fibres. *Histochem. J.* 20: 230–243, 1988.
5. **Cannon, R. J., and E. Cafarelli.** Neuromuscular adaptations to training. *J. Appl. Physiol.* 63: 2396–2402, 1987.
6. **Clamann, H. P., and T. B. Schelhorn.** Nonlinear force addition of newly recruited motor units in the cat hindlimb. *Muscle Nerve* 11: 1079–1089, 1988.
7. **Colliander, E. B., and P. A. Tesch.** Effects of eccentric and concentric muscle actions in resistance training. *Acta Physiol. Scand.* 140: 31–39, 1990.
8. **DeVries, H. A.** "Efficiency of electrical activity" as a physiological measure of functional state of muscle tissue. *Am. J. Phys. Med.* 47: 10–22, 1968.
9. **Dudley, G. A., R. S. Staron, T. F. Murray, F. C. Hagerman, and A. Luginbuhl.** Muscle fiber composition and blood ammonia levels after intense exercise in humans. *J. Appl. Physiol.* 54: 582–586, 1983.
10. **Elder, G. C. B., K. Bradbury, and R. Roberts.** Variability of fiber type distributions within human muscles. *J. Appl. Physiol.* 53: 1473–1480, 1982.
11. **Engstrom, C. M., G. E. Loeb, J. G. Reid, W. J. Forrest, and L. Avruch.** Morphometry of the human thigh muscles. A comparison between anatomical sections and computer tomographic and magnetic resonance images. *J. Anat.* 176: 139–156, 1991.
12. **Enoka, R. M.** Muscle strength and its development. New perspectives. *Sports Med.* 6: 146–168, 1988.
13. **Essen, B., E. Jansson, J. Henriksson, A. W. Taylor, and B. Saltin.** Metabolic characteristics of fibre types in human skeletal muscle. *Acta Physiol. Scand.* 95: 153–165, 1975.
14. **Evans, W. J., S. D. Pinney, and V. R. Young.** Suction applied to a muscle biopsy maximizes sample size. *Med. Sci. Sports Exercise* 14: 101–102, 1982.
15. **Fisher, M. J., R. A. Meyer, G. R. Adams, J. M. Foley, and E. J. Potchen.** Direct relationship between proton T_2 and exercise intensity in skeletal muscle MR images. *Invest. Radiol.* 25: 480–485, 1990.
16. **Fleckenstein, J. L., L. A. Bertocci, R. L. Nunnally, R. W. Parkey, and R. M. Peshock.** Exercise-enhanced MR imaging of variations in forearm muscle anatomy and use: importance in MR spectroscopy. *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 153: 693–698, 1989.
17. **Fleckenstein, J. L., R. G. Haller, S. F. Lewis, B. T. Archer, B. R. Barker, J. Payne, R. W. Parkey, and R. M. Peshock.** Absence of exercise-induced MRI enhancement of skeletal muscle in McArdle's disease. *J. Appl. Physiol.* 71: 961–969, 1991.
18. **Fleckenstein, J. L., D. Watumull, L. A. Bertocci, R. W. Parkey, and R. M. Pershock.** Finger-specific flexor recruitment in humans: depiction by exercise-enhanced MRI. *J. Appl. Physiol.* 72: 1974–1977, 1992.
19. **Garfinkel, S., and E. Cafarelli.** Relative changes in maximal force, EMG, and muscle cross-sectional area after isometric training. *Med. Sci. Sports Exercise* 24: 1220–1227, 1992.
20. **Häkkinen, K., M. Alén, and P. V. Komi.** Changes in isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of human skeletal muscle during strength training and detraining. *Acta Physiol. Scand.* 125: 573–585, 1985.
21. **Hather, B. M., C. E. Mason, and G. A. Dudley.** Histochemical demonstration of skeletal muscle fiber types and capillaries on the same transverse section. *Clin. Physiol. Oxf.* 11: 127–134, 1991.
22. **Houston, M. E., E. A. Froese, S. P. Valeriote, H. J. Green, and D. A. Ramey.** Muscle performance, morphology and metabolic capacity during strength training and detraining: a one leg model. *Eur. J. Appl. Physiol. Occup. Physiol.* 51: 25–35, 1983.
23. **Ikai, M., and T. Fukunaga.** A study of training effect on strength per unit cross sectional area of muscle by means of ultrasonic measurement. *Int. Z. Angew. Physiol. Einschl. Arbeitsphysiol.* 28: 173–180, 1970.
24. **Jones, D. A., and O. M. Rutherford.** Human muscle strength training: the effects of three different training regimes and the nature of the resultant changes. *J. Physiol. Lond.* 391: 1–11, 1987.
25. **Jones, D. A., O. M. Rutherford, and D. F. Parker.** Physiological changes in skeletal muscle as a result of strength training. *Q. J. Exp. Physiol.* 74: 233–256, 1989.
26. **Komi, P. V.** How important is neural drive for strength and power development in human skeletal muscle? *Biochem. Exercise* 16: 515–529, 1986.
27. **Komi, P. V.** Training of muscle strength and power: interaction of neuromotoric, hypertrophic, and mechanical factors. *Int. J. Sports Med.* 7: 10–15, 1986.
28. **Lind, A. R., and J. S. Petrofsky.** Isometric tension from rotary stimulation of fast and slow cat muscles. *Muscle Nerve* 1: 213–218, 1987.
29. **Martin, T. P., R. B. Stein, P. H. Hoeppner, and D. C. Reid.** Influence of electrical stimulation on the morphology and metabolic properties of paralyzed muscle. *J. Appl. Physiol.* 72: 1401–1406, 1992.

30. **Maughan, R. J.** Relationship between muscle strength and muscle cross-sectional area: implications for training. *Sports Med.* 1: 263–269, 1984.
31. **Milner-Brown, H. S., R. B. Stein, and R. G. Lee.** Synchronization of human motor units: possible roles of exercise and supraspinal reflexes. *Electroencephalogr. Clin. Neurophysiol.* 38: 245–254, 1975.
32. **Moritani, T., and H. A. deVries.** Neural factors versus hypertrophy in the time course of muscle strength gain. *Am. J. Phys. Med.* 58: 115–130, 1979.
33. **Narici, M. V., G. S. Roi, L. landoni, A. E. Minetti, and P. Cerretelli.** Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. *Eur. J. Appl. Physiol. Occup. Physiol.* 59: 310–319, 1989.
34. **Oslon, C., D. Carpenter, and E. Henneman.** Orderly recruitment of muscle action potentials. *Arch. Neurol.* 19: 591–597, 1968.
35. **Rack, P. M. H., and D. R. Westbury.** The effects of length and stimulation rate on tension in the isometric cat soleus muscle. *J. Physiol. Lond.* 204: 443–460, 1969.
36. **Sale, D. G., J. D. MacDougall, A. R. M. Upton, and A. J. McComas.** Effect of strength training upon motorneuron excitability in man. *Med. Sci. Sports Exercise* 15: 57–62, 1983.
37. **Scott, S. H., and D. A. Winter.** A comparison of three muscle pennation assumptions and their effect on isometric and isotonic force. *J. Biomech.* 24: 163–167, 1991.
38. **Scripture, E. W., T. L. Smith, and E. M. Brown.** On the education of muscular control and power. *Stud. Yale Psych. Lab.* 2: 114–119, 1894.
39. **Shaver, L. G.** Cross transfer effects of conditioning and deconditioning on muscular strength. *Ergonomics* 18: 9–16, 1975.
40. **Shellock, F. G., T. Fukunaga, J. H. Mink, and V. R. Edgerton.** Acute effects of exercise on MR imaging of skeletal muscle: concentric vs eccentric actions. *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 156: 765–768, 1990.
41. **Staron, R. S., M. J. Leonardi, D. L. Karapondo, E. S. Malicky, J. E. Falkel, F. C. Hagerman, and R. S. Hikida.** Strength and skeletal muscle adaptations in heavy-resistance-trained women after detraining and retraining. *J. Appl. Physiol.* 70: 631–640, 1991.
42. **Staron, R. S., E. S. Malicky, M. J. Leonardi, J. E. Falkel, F. C. Hagerman, and G. A. Dudley.** Muscle hypertrophy and fast fiber type conversions in heavy resistance-trained women. *Eur. J. Appl. Physiol. Occup. Physiol.* 60: 71–79, 1989.
43. **Veiersted, K. B.** The reproducibility of test contractions for calibration of electromyographic measurements. *Eur. J. Appl. Physiol. Occup. Physiol.* 62: 91–98, 1991.
44. **Weidman, E. R., H. C. Charles, R. Negro-Vilar, J. J. Sullivan, and J. R. MacFall.** Muscle activity localization with ^{31}P spectroscopy and calculated T_2-weighted ^{1}H images. *Invest. Radiol.* 26: 309–316, 1991.