MR measurements of muscle damage and adaptation after eccentric exercise

Jeanne M. Foley, Roop C. Jayaraman, Barry M. Prior, James M. Pivarnik, and Ronald A. Meyer.

Electrical stimulation of muscle fibers during eccentric exercise results in acute muscle damage manifested as pain and swelling (1). The extent of this damage is thought to depend on the number of fibers recruited during eccentric contraction, exercise intensity, and exercise duration (2). Recent experiments have used magnetic resonance imaging (MRI) to assess the extent of muscle damage and adaptation after eccentric exercise (3, 4).

Foley, Jeanne M., Roop C. Jayaraman, Barry M. Prior, James M. Pivarnik, and Ronald A. Meyer. MR measurements of muscle damage and adaptation after eccentric exercise. J. App. Physiol. 87(6): 2311–2318, 1999.—The purposes of this study were, first, to clarify the long-term pattern of T2 relaxation times and muscle volume changes in human skeletal muscle after intense eccentric exercise and, second, to determine whether the T2 response exhibits an adaptation to repeated bouts. Six young adult men performed two bouts of eccentric biceps curls (5 sets of 10 at 110% of the 1-repetition concentric maximum) separated by 8 wk. Blood samples, soreness ratings, and T2-weighted axial fast spin-echo magnetic resonance images of the upper arm were obtained immediately before and after each bout; at 1, 2, 4, 7, 14, 21, and 56 days after bout 1; and at 2, 4, 7 and 14 days after bout 2. Resting muscle T2 [27.6 ± 0.2 (SE) ms] increased immediately postexercise by 8 ± 1 ms after both bouts. T2 peaked 7 days after bout 1 at 47 ± 4 ms and remained elevated by 2.5 ms at 56 days. T2 peaked lower (37 ± 4 ms) and earlier (2–4 days) after bout 2, suggesting an adaptation of the T2 response. Peak serum creatine kinase values, pain ratings, and flexor muscle swelling were also significantly lower after the second bout (P < 0.05). Total volume of the imaged arm region increased transiently after bout 1 but returned to preexercise values within 2 wk. The exercised flexor compartment swelled by over 40%, but after 2 wk it reverted to a volume 10% smaller than that before exercise and maintained this volume loss through 8 wk, consistent with partial or total destruction of a small subpopulation of muscle fibers.

Within the past decade, it has been established that the T2 (transverse or spin-spin) relaxation time of muscle in proton MR images increases during exercise, returning to resting values within 1 h postexercise. This acute T2 change appears to arise from changes in intracellular water chemistry (24). It also scales with exercise intensity and therefore may serve as an index of muscle recruitment (1, 7, 12). More recently, it has been observed that a second T2 increase develops gradually from 1 to 6 days after eccentric but not after concentric or isometric exercise (8, 26). The time course and magnitude of this delayed T2 increase as well as its relationship to other markers of muscle damage have been described for the early postexercise period, with the general conclusion that the chronic T2 phenomenon reflects edema (21, 26). However, a few studies have monitored MR relaxation times in small groups of two to three subjects over longer periods and have documented persistent elevations in muscle T2 as long as 2–3 mo after a single bout of eccentric exercise (8, 26). Thus muscle T2 apparently may remain elevated long after swelling resolves, suggesting that this residual T2 increase cannot be attributed to extracellular water accumulation and could possibly reflect a long-lasting adaptation.

Most markers of eccentrically induced muscle damage show a long-lasting protective effect against further damage in the weeks and even months after a single bout of damaging eccentric exercise (14, 28). To date, no studies have examined whether the delayed T2 elevation is also attenuated after repeated bouts of eccentric exercise. Several models have been proposed to explain how an initial bout of injurious eccentric exercise is able to drastically reduce the damage resulting from a second bout repeated weeks or months after the initial bout (for reviews see Refs. 4 and 14). One theory proposed to explain this protective effect invokes the existence of a small subpopulation of mechanically vulnerable fibers that are irreversibly damaged by the excessive strain of eccentric contractions (2). This hypothesis would predict some loss of mass and volume in exercised muscle after a single bout of intense eccentric contractions. Several studies have reported small reductions in muscle mass in animal models after recovery from a damaging bout of eccentric exercise (2, 13, 18). Biopsies and serum markers have also shown evidence of muscle fiber destruction in two eccentric exercise studies on human subjects (13, 17). Muscle mass or volume reductions below preexercise values have not been documented in any human studies to date, although this may be a reflection of the low precision of

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the anthropometric and ultrasound measurement methods used.

The purposes of the present study were to clarify the long-term time course of T2 values in human muscle after intense eccentric exercise, to determine whether the delayed T2 response exhibits an attenuation adaptation to repeated bouts, and to precisely quantify the muscle volume changes induced by these exercises. T2-weighted MR images were used to determine T2 values and compartment volumes in the arm muscles of human subjects after a bout of severe eccentric exercise and again after a second bout 8 wk after the initial exercise. The specific hypotheses being tested were the following: 1) T2 will remain elevated in the exercised muscle 8 wk after the initial exercise bout, 2) peak T2 values achieved after bout 2 will be lower than after bout 1, and 3) volume of the exercised muscle compartment will be reduced after recovery from the initial exercise bout.

The results showed a small but persistent T2 elevation lasting at least 2 mo after the initial exercise, coincident with a measurable and enduring reduction in size of the exercised muscles. Peak T2 elevation was markedly reduced after the second compared with the first exercise bout, confirming an adaptation similar to that reported for other measures of muscle damage.

METHODS

Subjects. Six young adult men aged 21–25 yr were recruited from the university community. Subjects were screened for medical and orthopedic conditions that would preclude severe arm exercise or MR imaging (MRI) procedures. Also excluded were persons who had participated in a weight lifting program or regularly performed rigorous arm exercise for recreational or occupational purposes within the past year. The university committee for research involving human subjects approved procedures for the study, and all subjects gave informed written consent before participation. Physical characteristics of the subjects are provided in Table 1.

Exercise protocol. Each subject was tested for the one repetition maximum (1 RM; Table 1) for the standing concentric biceps curl by using a weighted dumbbell. The 1-RM lift was done with the right arm starting from full extension of the elbow and moving to full flexion; a trainer then removed the weight from the subject’s grip to ensure only concentric contraction of the biceps muscle. The subject was coached to use strict form on the lift, including maintaining an upright posture and keeping the elbow directly at the side of the body. Weight loads commenced at 15% of body weight for this 1-RM test. After a 1.5-min rest, the weight was incremented by 2.5 lb, and the lift was repeated until the subject was unable to complete a lift to full elbow flexion with proper form.

At this time, subjects were also introduced to a muscle soreness scale (4) in which the perception of pain in the biceps muscle was rated on a scale from 1 (“no soreness”) to 10 (“extremely sore”). Subjects rated soreness while holding the unweighted arm in a relaxed, extended position at the side of the body.

A preliminary MRI scan and blood draw were followed by an exercise bout consisting of 5 sets of 10 eccentric biceps curls of the left arm at 110% of the concentric 1-RM. Lifts were begun with the elbow fully flexed. The weight was lowered smoothly to a 4-s count to the point of full elbow extension. A trainer removed the weight at the bottom of the curl, and the subject returned the unweighted arm to the flexed position, receiving the weight to begin the next repetition after a 4-s rest. A 1.5-min rest separated each set of 10 repetitions. Immediately after the exercise, MRI scans were repeated. Pain ratings, MRI scans, and blood draws were repeated at the same time of day 1, 2, 4, 7, 14, 21, and 56 days after the eccentric exercise bout. Throughout this period, subjects were instructed to refrain from taking any anti-inflammatory medications or using ice, massage, or other analgesic agents. Weight lifting or other vigorous arm exercise was also prescribed during this period.

Eight weeks after the initial bout, the eccentric exercise protocol was repeated. MRI scans, blood draws and pain ratings were taken at 2, 4, 7, and 14 days postexercise with subjects following the same instructions regarding antiinflammatories, analgesics, and exercise. All six subjects completed the first phase of the study; one subject dropped out just before the repeat-bout phase.

MRI methods. Subjects were positioned headfirst and prone, with the left arm extended overhead, in a 1.5-T Signa whole body imager (General Electric, Milwaukee, WI). The upper left arm was encased in a standard linear transceiver (General Electric extremity) coil centered on an indelible-ink landmark drawn one-third of the distance along a line from the middle of the cubital fossa to the acromion process of the scapula. A series of 8 fast spin-echo axial images 10 mm thick and 5 mm apart was taken from a 12-cm-long region centered on this landmark. A 16 × 16-cm field of view was used, with two echoes (repetition time = 1500 ms; echo times = 30 and 60 ms), a 256 × 128 matrix, and one excitation. Acquisition time for each series was 3 min 30 s. Typical images from the center of this region are shown in Fig. 1.

Mean values of T2 were determined for the entire elbow flexor muscle compartment in each slice. Data from the two echoes were fitted to a monoexponential decay algorithm as described previously (7). The pixel counts and mean T2 value for the biceps brachii and brachialis muscles in each of the eight slices were combined to give a volume-weighted average T2 value for the elbow flexor muscles over the entire imaged region, which represented about two-thirds of the full length of these muscles.

Volumes of the whole imaged arm region as well as the bone, subcutaneous fat, exercised (flexor), and unexercised (extensor) muscle compartments (Table 1) were determined by manually tracing these regions on each slice. The resulting cross-sectional pixel counts were multiplied by pixel area (field of view/matrix) and then multiplied by the interslice interval (2 cm). The term “volume” is used to refer to this
estimate of the volume of that section of the muscles contained within the imaged region, recognizing that this region contained the belly of the muscle but did not cover the entire length of the muscle compartments. All volume analysis was done by a single investigator to ensure consistency across subjects and time. Complete preexercise scan sets from all subjects were analyzed twice, on separate days, to check repeatability of the manual tracing method. Group means of the two trials agreed to within 0.1%. Each pair of volume values agreed to within ±1% on each individual subject, with the mean of absolute values of differences between trials equaling 0.8 ± 0.2 (SE)% (n = 6).

Serum creatine kinase (CK) determinations. For each blood draw, a 4-ml volume of blood was withdrawn into a heparinized tube from a single venipuncture of the antecubital vein of the unexercised arm. Samples were immediately centrifuged, and the serum fraction was stored briefly on ice and then frozen at −20°C until chemical analysis. CK activity was determined in duplicate 0.02-ml aliquots at 37°C by using standard photometric techniques and a Sigma diagnostic test kit (CK-10, Sigma Diagnostics, St. Louis, MO).

Statistical analysis. All data are reported as means ± SE, with differences evaluated by two-way analysis of variance [bout (df = 1) × day (df = 4)] with the α-level of significance set at $P < 0.05$. Only the five levels of the "day" factor common to both bouts (days 0, 2, 4, 7, and 14) were included in the ANOVA. (Outcome measures for day 1 postexercise were omitted after the second bout because none of the tested parameters peaked before day 2.) Where justified by ANOVA results, specific comparisons arising from the hypotheses were tested by using the t-statistic for a one-tailed test on paired differences with the Bonferroni adjustment for mul-

![Fig. 1. Representative midbrachial axial plane magnetic images (repetition time = 1,500 ms; echo time = 30 ms) from 1 subject before (top left image; Pre) and at each test interval during the 8 wk after initial eccentric exercise bout (test day noted next to image; D1–D56). In these T2-weighted images, lighter contrast corresponds to longer T2 relaxation times. Images are oriented so that anterior direction is to the right and the lateral direction is upward.](http://jap.physiology.org/DownloadedFrom)
multiple comparisons. Because of the failure of one subject to participate in the second exercise bout, interbout comparisons utilized data only from those five subjects who completed both exercise bouts. Our hypotheses that T2 values would remain elevated and that the volume of the exercised muscle compartment would be reduced after 8 wk of recovery from the initial exercise bout were tested by one-tailed paired t-tests on values for all six subjects for day 56 vs. preexercise.

RESULTS

Initial exercise bout. The image series in Fig. 1 shows the progression of swelling and T2 (as reflected by image contrast) in the elbow flexor muscles of one subject during the 8-wk period after the first bout of eccentric exercise. Figure 2 displays the time course of the group mean changes in these two parameters as well as serum CK concentrations and subjective pain ratings. Note that "overall arm swelling" in Fig. 2B refers to general swelling of the entire imaged section of the arm; specific swelling of the exercised muscle compartment is displayed in Fig. 3 to facilitate comparisons of this parameter between the two exercise bouts.

Group means of the preexercise (baseline) values are summarized in Table 2, along with peak changes from baseline after each of the two bouts. Statistically significant changes from baseline occurred in all parameters except overall arm swelling (Table 2, Figs. 2 and 3). The effectiveness of the exercise regimen in producing damage is most evident from the 130-fold increase in serum CK after exercise bout 1, although substantial elevations were observed in all measured parameters.

The time courses of changes in the indexes of muscle damage varied widely, with pain peaking earliest at 48 h postexercise, followed by serum CK 2 days later, and finally by T2 and overall arm swelling, which both peaked toward the end of the first week (Fig. 2). Roughly the same order was followed in the recovery to baseline values, with the exception that the T2 and swelling recovery time courses diverged after simultaneously reaching peak values. Arm volume returned to normal during the second week postexercise, whereas T2 declined more gradually and remained elevated by nearly 10% (29.9 ± 0.3 vs. 27.6 ± 0.2 ms, P < 0.05) 8 wk after the initial bout of eccentric exercise.

A transient or acute T2 increase of 8.4 ± 1.3 ms was observed in MR images taken immediately after exercise. Transient swelling was also observed immediately after exercise in both the nonexercised extensor and

Table 2. Baseline values and peak values after each exercise bout

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Preexercise Values</th>
<th>Peak Value After Bout 1</th>
<th>Peak Value After Bout 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>T2 of flexor muscles, ms</td>
<td>27.6 ± 0.2</td>
<td>46.7 ± 3.8*</td>
<td>37.6 ± 3.9†</td>
</tr>
<tr>
<td>Serum CK, IU/l</td>
<td>156 ± 45</td>
<td>21,000 ± 3,500*</td>
<td>2,600 ± 1,200†</td>
</tr>
<tr>
<td>Pain rating, 1–10 scale</td>
<td>1 ± 0</td>
<td>8.0 ± 0.4*</td>
<td>5.6 ± 0.5†</td>
</tr>
<tr>
<td>Arm volume, ml</td>
<td>820 ± 40</td>
<td>960 ± 60</td>
<td>900 ± 50</td>
</tr>
<tr>
<td>Flexor muscle volume, ml</td>
<td>245 ± 10</td>
<td>350 ± 30*</td>
<td>270 ± 15†</td>
</tr>
</tbody>
</table>

Values are means ± SE for 6 subjects per group (baseline and bout 1) or 5 subjects per group (bout 2). CK, creatine kinase. *Significantly different from baseline, P < 0.5. †Significantly different from bout 1, P < 0.5; analysis conducted only on those 5 individuals who completed both bouts.
exercised flexor muscle compartments (volume increases of 9 ± 4 and 18 ± 4%, respectively). However, the persistent later swelling was exclusively localized to the exercised flexor muscle compartment. By 48 h postexercise, the volume of the nonexercised triceps muscle had returned to preexercise values and remained near baseline values for the remainder of the 2-mo period (data not shown). In contrast, by 48 h the exercised biceps/brachialis muscle compartment had swelled by over 40% (Fig. 3; also Fig. 1, Pre vs. D2). Flexor muscle volume peaked just before peak serum CK occurrence and then dropped precipitously during the second week after exercise to a value <90% of baseline volume (217 ± 7 ml at 14 days vs. 245 ± 10 ml preexercise; P < 0.05). This volume loss was still evident 8 wk after the exercise (227 ± 6 ml at 56 days; P < 0.05 vs. preexercise). Furthermore, a volume loss was detected in five of the six subjects. The one subject whose biceps size remained unchanged also had the smallest changes in the all measured markers of damage.

Repetitive exercise bout. The transient or acute T2 increase immediately after the second bout of eccentric exercise was 8.4 ± 0.5 ms, which was not different from the acute change after the first bout. Figure 4 shows the delayed T2 response and the other markers of damage after the second bout of eccentric exercise compared with the first bout conducted 8 wk earlier.

Significant main effects of bout and time were observed for all tested parameters except for overall arm swelling. No bout by time interaction occurred for T2 or flexor volume, thus allowing testing of the comparisons suggested by the initial hypotheses. A significant interaction was noted for CK values; this finding is explained by the fact that CK values achieved statistically significant changes across time after bout 1 but not after bout 2 (Table 2, Fig. 4). Subjective pain ratings also exhibited a bout by time interaction, suggesting that the significance of the peak pain ratings after bout 2 was due at least in part to expectations raised by subjects' recall of the severity of the pain induced by the first exercise bout.

The magnitude of the peak increase was significantly lower for each index of muscle damage except overall arm swelling (Table 2, Fig. 4). The serum CK rise was most drastically attenuated, achieving a peak value nearly 10-fold lower than that produced by bout 1. Peak T2 increase and peak overall arm swelling after bout 2 averaged less than one-half the amount of change induced by the initial bout. Despite these large reductions in objective measures of muscle injury, peak subjective pain ratings were only marginally lower compared with the first bout (Table 2, Fig. 4). Peak responses also occurred earlier after the second bout, again with the exception of perceived soreness, for which the time course as well as magnitude closely paralleled that of bout 1 (Fig. 4).

The exercised flexor muscle compartment swelled only about one-third as much after the second vs. initial exercise bout (Fig. 3). During the recovery from bout 2, this muscle compartment reverted to the reduced size that had been maintained during the second month after the initial exercise bout, although no additional persistent volume loss occurred. Also, muscle T2 values recovered rapidly after the second bout, but the plateau of this recovery corresponded more closely to the chronically elevated T2 after bout 1 (mean T2 at 56 days after bout 1 = 29.9 ± 0.3 vs. 30.7 ± 0.9 ms at 14 days after bout 2) rather than to the preexercise baseline T2 value (27.6 ± 0.2 ms).
DISCUSSION

Muscle volume changes. A unique aspect of this study was quantification of arm and internal muscle compartment volumes via MR images. Arm circumference measurements to track swelling caused by eccentric exercise have been documented in numerous studies (e.g., Refs. 4, 11, 25). Results typically show that the arm circumference increases immediately after exercise. This acute swelling subsides quickly, followed by a gradual, weeklong rise to a maximum that occurs 1–2 days after peak serum CK levels. Although our MR measurements of brachial volume provide a more precise and reproducible estimate of total arm swelling, our arm volume data merely confirm these prior results.

Of more interest was our observation of a persistent 7–10% volume deficit in the exercised muscle compartment during the period from 2 to 8 wk postexercise. The variability of whole arm volume measurements, including those by MRI, precludes the detection of very small volume changes; consequently no previous studies have monitored external arm volumes after the recovery to preexercise values during the first 2 wk after exercise. Several prior studies have attempted to quantify internal muscle compartment volumes by using Doppler ultrasound to measure muscle thickness and assuming a cylindrical muscle geometry, but the crudeness of the resulting volume estimates prevents reliable discrimination of subtle volume changes such as those reported in the present study. One of these studies did report measurable acute swelling in the flexor muscles immediately after exercise but detected no delayed swelling in the 1- to 10-day postexercise period (11). Using similar sonographic methods, Nosaka and Clarkson (21) did observe delayed flexor muscle thickness increases peaking at 3–4 days postexercise; however, ultrasonography was not performed beyond 5 days postexercise in their study. MR images were also taken out to 58 days in that study, but neither arm circumference nor muscle compartment areas were quantified. Another sonographic study by Chleboun et al. (3) followed compartment volume of the elbow flexor muscles for 11 days after eccentric exercise in untrained female subjects. This study reported a significant volume increase in the exercised muscles, peaking at 26% above baseline by 4 days and then returning to near baseline values by 11 days. The time course of this ultrasound data corresponds well with our MRI results (Fig. 4), although, again, volumes were not tracked beyond 11 days postexercise.

Other groups employing MRI methods have anecdotally observed muscle volume increases in the week postexercise, but have either not quantified the changes (26) or examined neither internal compartment nor long-term total arm volume changes (22). An MRI study of leg muscle by Dudley et al. (5) quantified cross-sectional areas in the images, noting significant swelling peaking 4 days after eccentric exercise and returning to baseline by day 10, although no measurements were made beyond 10 days after exercise.

The observed volume loss is consistent with the existence of a small population of “susceptible” or “vulnerable” fibers within a muscle compartment, a hypothesis initially advanced by Armstrong and colleagues in 1983 (2). These authors observed necrosis of ~5% of fibers in muscles of rats mildly eccentrically exercised by downhill running. A more recent study of mice by this group also reported apparently irreversible muscle fiber damage as well as muscle mass and protein content reduction 14 days after in vivo eccentric exercise (16). A recent report by McCully and Faulkner (18) also documented muscle mass decreases in mice after eccentric but not concentric or isometric exercise. In this study, muscle mass loss peaked at 25% in extensor digitorum longus muscle 7 days after an intense bout of lengthening contractions in situ.

Two human studies have also reported evidence of this phenomenon. In one study, the appearance of myosin heavy chain fragments in the blood 3–5 days after peak appearance of the smaller, cytoplasmic CK protein was interpreted as proof of irreversible degradation of muscle fibers (17). In the other report, biopsies of human calf and biceps muscles after eccentric exercise showed evidence of muscle fiber degeneration at 21 days but not as early as 7 days after exercise (13). To our knowledge, the results of the MR volume assessments in the present study represent the first quantitative evidence of muscle volumetric loss after recovery from damaging eccentric exercise in human subjects.

T2 as indicator of muscle damage. Immediate postexercise T2 elevation has been used in many recent studies as an index of exercise intensity (e.g., Refs. 1, 7, and 12). We observed that the acute T2 increase was identical after both exercise bouts, providing evidence that the identical loads, sets, repetitions, and timing of the two exercise regimens represented the same relative as well as absolute work intensities in both cases. Although we did not measure muscle strength after the exercise bouts, others have reported no strength change resulting from a single bout of intense eccentric exercise (27).

The delayed rise in T2 relaxation time in the eccentrically exercised muscle compartment paralleled other
objective measures of muscle damage in this study for the first week after exercise. The persistence of the T2 elevation after other indicators of injury have subsided suggests that the long-term T2 response may reflect a more permanent change in the muscle, whereas the response in the first week tracks transient damage. The coincidence of the residual T2 elevation with the observed persistent loss in flexor muscle volume supports this conclusion. We propose that the long-lasting attenuation effect induced by a single, damaging bout of eccentric exercise is an adaptation reflected by both the chronic T2 elevation and the loss of volume in the exercised muscle. The mechanism of the residual T2 elevation remains unclear, although edema is unlikely to be a contributing factor because swelling resolves within 2 wk postexercise. The direction of the T2 change is consistent with previous reports that fast-twitch fibers are preferentially damaged by eccentric exercise (9, 13, 28) and that fast-twitch muscle appears to have shorter T2 values compared with those of slow-twitch fibers (6, 15). However, the size of the apparent volume loss (≤10%) and the small magnitude of the fiber type difference in T2 (2–3 ms) are insufficient to account for all of the observed residual T2 increase. Further study will be needed to determine the combination of mechanisms accounting for the total effect.

Adaptation to eccentric exercise. The existence of a protective effect conferred by a single bout of eccentric exercise against muscle damage from a later bout was first described nearly 30 years ago when Nuttall and Jones (23) reported smaller increases in two muscle enzymes in the blood of subjects in response to an eccentric test after training involving a substantial eccentric component. Many subsequent studies have clarified the nature and extent of this protective adaptation, noting that peak changes in numerous objective measures of muscle damage were smaller and occurred earlier after a second compared with an initial bout of eccentric exercise (for review see Ref. 28). The present study provides the first evidence that this adaptation extends to the delayed T2 response.

Several theories have been advanced regarding the mechanism underlying this protective adaptation or “repeated-bout effect” (e.g., Refs. 10, 14, 19, 20). One model consistent with the data reported here postulates that the excessive strain produced by a bout of unaccustomed eccentric exercise could mechanically disrupt the sarcomeric and/or connective tissue structure enough to result in complete and irreversible necrosis of the most severely damaged fibers (2). Our results showing a maintained volume loss exclusively in exercised muscles provide additional evidence in support of the susceptible fiber theory, although it is possible that this volume loss could be explained by reduced diameter rather than number of fibers, or by partial fiber loss due to focal area damage.

A muscle volume loss of 7–10% as observed here is consistent with the data cited above in animal and human models. Because the elbow flexor muscles comprise <30% of the volume of the imaged brachial region (Table 1), it is credible that arm circumference or cross-sectional area measurements would not detect what would translate into a total arm volume loss on the order of 2%. The persistence of the flexor volume loss to at least 8 wk postexercise is also consistent with this and other reports on the months-long duration of the protective effect. Destruction of most of the vulnerable fiber population by the first bout of exercise would explain not only the nearly 90% reduction in serum CK accumulation after the second bout but also the protection against further biceps/brachialis muscle volume loss after the second bout, as seen in Fig. 3. Faster recovery after the second bout is also predicted by this model: although the second eccentric bout produces some damage, the extent of trauma is consistent with partial, reversible fiber disruption that would be likely to cause smaller and earlier CK release than processes involving complete digestion of severely damaged cells.

In summary, this study confirms the utility of T2 relaxation time as a marker of muscle damage due to eccentric exercise and provides the first evidence that the change in this MR parameter is diminished after a second bout of eccentric exercise, in agreement with the reported changes in other markers of damage. Volume assessment of internal tissue compartments in MR images also shows a 7–10% reduction in size of the exercised muscles that is maintained during the period from 2 to 8 wk after the initial exercise bout. We interpret this shrinkage as possible evidence of the loss of a subpopulation of mechanically weak fibers, an adaptation consistent with the enduring protection against further damage.

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Address for reprint requests and other correspondence: J. M. Foley, Dept. of Kinesiology, Rm. 3 IM Sports-Circle, Michigan State Univ., East Lansing, MI 48824.

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