Acute muscle damage as a stimulus for training-induced gains in strength

JONATHAN P. FOLLAND, JAMES CHONG, ELIZABETH M. COPEMAN, and DAVID A. JONES

Chelsea School Research Centre, University of Brighton, Eastbourne, UNITED KINGDOM; and School of Sport & Exercise Sciences, University of Birmingham, Birmingham, UNITED KINGDOM

ABSTRACT

FOLLAND, J. P., J. CHONG, E. M. COPEMAN, and D. A. JONES. Acute muscle damage as a stimulus for training-induced gains in strength. Med. Sci. Sports Exerc., Vol. 33, No. 7, 2001, pp. 1200–1205. Purpose: The purpose of this study was to investigate the effect of a single acute bout of maximal eccentric work upon the strength gains during 9 subsequent weeks of strength training. Eccentric work causes acute muscle damage that may initiate compensatory hypertrophy and enhance training-induced gains in strength. Methods: Twenty-six healthy adults (21 ± 1 yr, 7 women) trained the elbow flexors 3 d per week for 9 wk. One arm (C) performed purely conventional isotonic training, i.e., lifting and lowering. The other arm (E) began with a single bout of maximal eccentric work but thereafter undertook identical isotonic training. Every week dynamic lifting strength (1 RM) and isometric strength were measured. Results: The results indicated that an acute bout of eccentric muscle damage does not accentuate training-induced gains in strength. Isometric strength of arm E fell by 15 ± 2% (mean ± SEM) 2 d after the bout of eccentric work, and, 4 d afterward, plasma creatine kinase levels were 1502 ± 397 IU·L⁻¹. Although arm E displayed rapid gains in strength from 2 d after the bout of eccentric work, these were not sustained, and for several weeks arm E showed significantly smaller gains in strength than arm C (isometric strength, 2 wk; dynamic lifting strength, 5 wk). Conclusions: After 9 wk of training, the gains in both isometric and dynamic lifting strength were similar for the two arms. A single bout of damaging eccentric work did not enhance the response to conventional strength training and significantly compromised strength gains for several weeks. Key Words: MUSCULAR STRENGTH, STRENGTH TRAINING, SKELETAL MUSCLE, MUSCLE DAMAGE

The precise stimulus for strength gains and muscle hypertrophy in response to high resistance work remains unknown. Metabolite accumulation has been suggested to be important (30), but this was not found to be the case in a 9-wk study comparing high and low fatiguing protocols (9). High mechanical stress, with its associated damage and repair processes that may initiate compensatory hypertrophy, is another possible stimulus (13,21).

During eccentric work, skeletal muscle generates considerably greater forces than concentric or isometric work (~40% in the elbow flexors (8)). Several studies have compared eccentric and concentric work regimes, but the consensus is that purely eccentric work is no more effective at increasing strength than concentric training (4,16,18). Some studies have found a combination of concentric and eccentric contractions to be more effective at increasing strength than either concentric or eccentric contractions alone (6,14). It is not clear, however, whether this was as a result of a prolonged activity or because of an interaction between the two types of training.

Unaccustomed eccentric work can result in considerable, although temporary, muscle damage, as indicated by disruption of the sarcomere architecture (10), elevation of circulating muscle proteins (24,25), and a marked reduction (up to 50%) of force-generating capacity (3). However, in the period after a bout of eccentric contraction-induced damage, skeletal muscle exhibits rapid recovery of muscle strength (24) and regeneration of the muscle architecture (17). In mice, 5 d after eccentric contraction-induced damage, protein synthesis was elevated by 83% (20).

In this study, it was hypothesized that skeletal muscle may be particularly sensitive to a training stimulus at the time when it is regenerating rapidly. Conventional training at this time may lead to supercompensation, and this is certainly a popular view among bodybuilders. Therefore, we investigated the effect of a single bout of maximal eccentric work at the start of a 9-wk strength training program on the gains in isometric and dynamic lifting strength.

METHODS

Approach to the problem. The large individual variation in the response to strength training (14,4) makes the comparison of strength training protocols between groups of subjects particularly difficult. In contrast, intrasubject comparisons, where opposite limbs are trained with different methods, should highlight the experimental variable. However, there is controversy regarding the possibility of crossover effects with some studies reporting a transfer of strength gains between limbs (19,22), whereas other studies have found no evidence for this effect (7,12). Nevertheless, cross-over effects that are ascribed to a neurological adaptation could confound this type of intrasubject design. To minimize the influence of neurological adaptations, young
healthy and physically active subjects, who might have less scope for changes in learning and coordination, were recruited. Furthermore, to evaluate the capacity available for neurological adaptation, the ability of a subsample of subjects to activate the agonistic muscle group was tested before the training.

**Subjects.** Twenty-six healthy young (21.4 ± 0.7 yr, mean ± SEM) adults completed the study. The subjects were volunteers from among the University’s staff and students. All the subjects were recreationally active, but none had experience of systematic strength training. Subjects’ physical characteristics were recorded pretraining (Table 1), with somatotype measured according to Heath and Carter (15). The study was approved by the local Ethics Committee and subjects gave their written informed consent before participating.

**Strength training.** Subjects trained the elbow flexors of both arms unilaterally three times per week (Monday, Wednesday, Friday) for 9 wk. All of the training was carried out on a “preacher curl” type training machine that isolated the movement to the elbow flexors and could be adjusted to the individual’s size. The training machine afforded a range of movement from 2.80 to 0.7 rad (160° to 40°) of elbow flexion. Eccentric and concentric contractions were performed at a velocity of ~1.57 rad·s⁻¹. All of the training sessions were supervised with every lift recorded and encouragement throughout all of the training.

One arm of each subject was randomly assigned to perform purely conventional isometric training (C), completing 4 sets of 10 repetitions, lifting and lowering, at 75% of maximum (1 RM) during each training session. The one repetition maximum of each arm was reassessed on a weekly basis (see below).

The other arm (E) was subjected to a single bout of maximal eccentric work, but from 4 d after the eccentric bout carried out identical conventional isometric training. For the eccentric contractions, a lever was added to the training machine to enable the experimenter to forcibly extend the elbow of the subject. The eccentric bout involved one repetition every 10 s for 10 min, making a total of 60 repetitions. Subjects were instructed to maximally resist the downward movement of the training apparatus. Visual feedback of force and verbal encouragement were provided to the subjects throughout the eccentric exercise to maintain a high level of motivation. To provide direct visual feedback for each contraction, the voltage output from a strain gauge on the training machine was amplified and displayed on a strip of lights.

**Strength testing.** Pretraining measurements were taken on three occasions in a 10-d period before the training commenced. Data from the first of these were discarded and the second and third were averaged and used as the measure of pretraining strength. Posttraining measurements were taken 3 d after the last training session.

**Isometric strength.** Isometric strength of the elbow flexors was measured with an adapted conventional strength testing chair (26). The upper arm was positioned perpendicular to the frontal plane on a rigid surface. The wrist was strapped to a strain gauge fixed to an adjustable rotating plate that could be set to joint angles of 1.05, 1.40, 1.75, 2.09, and 2.44 rad. Movement of the torso was prevented by lap and shoulder straps. This apparatus afforded spatial adjustment in all three dimensions for different body sizes, and could be precisely repositioned.

Pre- and post-training isometric force of each arm was measured first at a joint angle of 2.09 rad (120°) and then at four other angles 1.05, 1.40, 1.75, and 2.44 rad (60°, 80°, 100°, and 140°). The order of the other four angles and the two arms was randomized for each subject and then maintained on successive testing occasions. At each angle, maximum isometric voluntary force was measured. This involved three maximal efforts of elbow flexion of at least 2-s duration with 20 s of rest between each contraction. The highest of three maximum voluntary contractions (MVCs) was the measure of maximum strength for each angle. In addition, isometric strength measurements for both arms were also taken at the criterion angle (2.09 rad) every week (Monday) throughout the training period, as well as 2 d after the eccentric exercise bout for arm E.

**Dynamic lifting strength (1 RM).** Subjects’ maximal lifting strength, also known as the one repetition maximum (1 RM), was tested on the training machine pre- and post-training and every week throughout the training program. Starting with ~90% of their previous maximum, subjects lifted successively heavier loads until failure. There was 30 s of rest between lifts, and 0.25- or 0.5-kg increments were used to increase the weight.

**Muscle soreness and plasma creatine kinase.** Two days after the eccentric exercise, subjects were asked to rate the soreness of their elbow flexors on a five-point scale from normal to extremely sore. Four days after the eccentric exercise, venous blood samples were collected in heparinized tubes from 23 subjects. Plasma samples were frozen and later analyzed for CK activity with a Sigma Diagnostics Kit (St Louis, MO).

**Activation.** Several studies have concluded that the majority of untrained healthy subjects can fully activate their major muscle groups (1,2,11,28). Nevertheless, to confirm this assumption for the present study, and to measure the scope for neurological adaptation, the activation level of 10 subjects was measured pretraining. On the third of the pretesting occasions, electrically stimulated twitches were

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**TABLE 1. Subjects’ physical characteristics (mean ± SEM).**

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Mass, kg</th>
<th>Height, m</th>
<th>Somatotype Rating</th>
<th>% Body Fat</th>
</tr>
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<tbody>
<tr>
<td>Female</td>
<td>7</td>
<td>60.9 ± 1.5</td>
<td>1.66 ± 0.02</td>
<td>3.1 ± 0.4</td>
<td>4.4 ± 0.3</td>
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<tr>
<td>Male</td>
<td>19</td>
<td>71.9 ± 5.8</td>
<td>1.78 ± 0.02</td>
<td>2.8 ± 0.2</td>
<td>4.8 ± 0.2</td>
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superimposed on three MVCs to estimate the level of elbow flexor activation (28). Two conducting rubber electrodes were applied proximally and distally to the surface of the biceps brachii. A CED-1401 (Cambridge Electronic Design Ltd., Cambridge, U.K.) triggered the electrical stimuli (pulse width 50 μs, up to 200 V, Digitimer DS7, Welwyn Garden City, United Kingdom) at a frequency of 1.25 Hz, and twitch magnitude was manipulated by changing the current (range, 28–50 mA). The resting twitches on average evoked a force of 10–15% MVC. The size of the twitches during the voluntary contractions were compared with those at rest before the contraction to calculate the level of muscle activation.

**Statistical analyses.** Paired Student’s t-tests were used to test for significance within and between groups. All of the changes in strength reported here are relative to the pretraining values. Relative gains in strength were used to compare the two training protocols. Strength gains at different angles were compared with analysis of variance and a post hoc Scheffe test. The Pearson product moment correlation was used to examine the relationship between gains in strength and other variables. Values are expressed as means ± SEM, and the 0.05 level of confidence was accepted for statistical significance.

**RESULTS**

**Damage after eccentric exercise.** After the bout of maximal eccentric exercise, subjects showed the characteristic signs of muscle damage. Two days after the exercise, there was a significant decrease in isometric strength of 14.9 ± 2.3% (P < 0.001; range –2.6 to –42.2% at 2.09 rad) and, on average, arm E was subjectively rated as “very sore.” Four days after the damaging exercise, plasma CK levels were 1502.1 ± 396.7 IU·L⁻¹ (range, 173.5–7477.6 IU·L⁻¹). The normal plasma CK range in healthy subjects is 60 –190 IU·L⁻¹. There was only a weak correlation between the fall in isometric strength and the level of plasma CK (r = −0.31).

**Activation.** By using twitch superimposition, it was found that all of the tested subjects could achieve >95% of full muscle activation. On average, these untrained subjects achieved 97.1 ± 1.5% (N = 10) of full muscle activation before the training.

**Training.** On average, subjects attended 25 of 27 training sessions in the 9-wk period. Over the whole training period, all the subjects increased their one repetition maximum on the training machine and therefore their training load.

**Dynamic lifting strength (1 RM).** The mean 1 RM increased significantly for both arms (P < 0.01): from 9.1 ± 0.5 kg at the start of the training to 12.8 ± 0.7 kg after 9 wk training for arm E, and from 8.8 ± 0.5 to 12.8 ± 0.6 kg for arm C. The mean percentage increase in the 1 RM throughout the training is shown in Figure 1. During the first 5 wk of the training, arm E showed significantly smaller improvements than arm C (P < 0.05). However, after the entire 9 wk of training, there were no significant differences between the arms, and they could lift (1 RM) on average 42.4 ± 2.6% (E) and 41.6 ± 3.4% (C) more than pretraining.

**Isometric strength.** There were no significant differences between the absolute isometric strengths of the two arms pre- or post-training, and both increased significantly from pre- to post-training (P < 0.001, Table 2). The time course of the relative changes in strength for both arms is displayed in Figure 2. Due to the bout of eccentric muscle damage, arm E was significantly weaker than arm C and below its pretraining strength for at least 2 wk. However,
from 2 d after the damage session, arm E improved rapidly during the following 3 wk (2.61% per training session) and after only 3 wk of training the two arms were of similar strength (Fig. 2). Arm E briefly rose above arm C, after 35 and 42 d of training, but this difference was not significant or sustained. Post-training, arm E had improved by 18.4 ± 3.0% and arm C by 16.6 ± 2.2%. This was equivalent to overall gains in strength of 0.74% and 0.66% per training session for arms E and C, respectively.

The individual responses to strength training were highly variable (mean of both arms, range, 3.4–35.1). The gains in strength were unrelated to gender, somatotype, body mass, height, or pretraining strength. The gains in isometric strength of arm E after 9 wk were not related to any marker of the damage sustained after the eccentric exercise bout (fall in isometric strength, $r = 0.18$; CK release, $-0.10$).

**The length-tension relationship.** Changes in the isometric strength at each joint angle of both arms are illustrated in Figure 3. There were no significant differences between the strength changes of the two arms at any angle. The strength increases for both arms were significantly greater ($P < 0.05$) at the longest muscle length (2.44 rad) than at any other angle: 32.1 ± 6.9% for arm E and 33.9 ± 5.8% for C.

**DISCUSSION**

The muscle damage sustained after the eccentric work appeared to be substantial with a significant loss of strength and subjective discomfort 2 d after the exercise and high plasma CK levels 4 d afterward. However, these are indirect indices of muscle damage that require further comment. There is little doubt that muscle soreness and CK release are caused by eccentric exercise, yet these parameters do not seem to be directly related to structural damage of skeletal muscle tissue (5). Changes in strength after eccentric exercise are also not entirely coincident with myofibrillar disruption (10,23), although the significant fall in strength we have observed is strongly suggestive of structural damage to skeletal muscle tissue. It was notable that the decrease in strength and the level of plasma CK were smaller than reported in similar studies (24,25,27).

The observed increases in isometric strength of 0.74% and 0.66% per training session, averaged over the whole 9 wk, are similar to a comparable training study of the elbow flexors (0.81% per training session (7)). In accordance with several previous reports, the subjects in the present study were able to activate their elbow flexors to a very high level before the training (1,11,28). This implies that there is almost no scope for a training-induced increase in the level of agonist activation. Although other neurological adaptations could contribute to gains in strength (i.e., increased motor unit synchronization (29), the level of antagonist/synergistic activation (12)) the high level of activation tends to suggest that the neural contribution to the observed gains in strength was limited. A limited neural contribution implies that significant cross-over effects, which tend to be attributed to neural adaptations, were unlikely. Another consequence of a limited neural contribution to strength gains is a greater contribution from morphological adaptations including hypertrophy, although the relative contribution of neural and hypertrophic adaptations to work-induced changes in strength remains controversial.

It was notable that gains in strength for both arms were significantly greater at the longest muscle length. Furthermore, the gains in isometric strength at this long length were the most similar to the increase in 1 RM. It is likely that the large increase in strength at the longest muscle length was due to the mechanics of the training apparatus. At long muscle lengths, the muscle must overcome the inertia of lifting the weight at the start of the lift while at a considerable mechanical disadvantage.

The first major finding of the present study was that a bout of maximal eccentric muscle damage did not potentiate the response to conventional strength training. There were no beneficial effects of the eccentric exercise bout on isometric or dynamic lifting (1 RM) strength gains any point during the 9 wk of training. After the damaging bout, isometric strength gains in the damaged arm were rapid in the first 3 wk, but these were not sustained above those found with conventional isotonic training. The strength gains of arm E were unrelated to the magnitude of the initial strength decrement after the bout of eccentric exercise. The training of arm C, which consisted of lifting and lowering, clearly involved an eccentric component that could in theory have confounded the study design. However, the eccentric component of arm C was far from the maximal eccentric bout performed by arm E, and the strength measurements of this arm showed no evidence of muscle damage in the early stages of the training program.

Eccentric contraction-induced muscle damage can cause disruption of the contractile apparatus, fiber necrosis, and regeneration (17). It was hypothesized that initiation of the regenerative processes might facilitate greater adaptations to strength training. However, this was not the case, suggesting that recovery from eccentric muscle damage and the adap-
tations to strength training are distinct processes. Although it is possible that the muscle damage in this study was not sufficient to initiate significant overcompensation, the lack of a relationship between any of the measures of damage and gains in strength suggests that above average levels of damage were of no advantage. Alternatively, disruption to the contractile apparatus may have been a relatively minor component of the muscle damage sustained, when it may be overcompensation of this material that is required for hypertrophy and gains in strength.

The second major finding of the present study was the reduced strength gains for several weeks after the eccentric exercise bout. The gains in dynamic lifting strength of arm E were significantly compromised for at least 5 wk after the bout of maximal eccentric work. The isometric strength gains of arm E were also significantly lower than arm C for at least 2 wk. Therefore, during the initial stages of strength training, eccentric-contraction–induced muscle damage inhibits the acquisition of strength and reduces strength performance for several weeks although providing no positive effect in the 9 wk after the eccentric exercise bout. Clearly for individuals starting a strength training program, this strategy seems to be entirely disadvantageous, particularly considering the discomfort involved with a bout of maximal eccentric work.

This result casts some doubt on the common practice of occasional acute bouts of eccentric work in habitual strength trainers and bodybuilders, but there are a number of reasons why the findings of the present study may not be transferable to these different populations. First, the process of strength gain, which is at a much slower rate, may be very different in habitual strength trainers. The morphological and hypertrophic adaptations of habitual strength trainers may involve different processes and stimuli to that of individuals in the initial stages of training. Second, as a result of long-term training, the response of habitual trainers to eccentric contraction induced injury, in terms of their susceptibility to muscle damage and the form of damage sustained, may be significantly different to that of novice strength trainers. At the present time, firm conclusions regarding the efficacy of this training strategy in habitual strength trainers cannot be drawn.

In summary, a bout of eccentric work designed to initiate muscle damage significantly inhibits training-induced gains in strength for several weeks, although providing no benefit during the 9 wk after the eccentric work. The authors recommend that novice strength trainers do not use this training strategy. The findings also cast doubt over the use of this strategy by habitual strength trainers, although further work is required to clarify this issue.

Address for correspondence: Dr Jonathan P. Folland, Chelsea School Research Centre, University of Brighton, Gaudick Road, Eastbourne, East Sussex, BN20 7SP, United Kingdom; E-mail: j.folland@bton.ac.uk.

REFERENCES


