# Repeated high-force eccentric exercise: effects on muscle pain and damage

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NEWHAM, D. J., D. A. JONES, AND P. M. CLARKSON. *Repeated high-force eccentric exercise: effects on muscle pain and damage.* J. Appl. Physiol. 63(4): 1381–1386, 1987.—Five women and three men (aged 24–43 yr) performed maximal eccentric contractions of the elbow flexors (for 20 min) on three occasions, spaced 2 wk apart. Muscle pain, strength and contractile properties, and plasma creatine kinase (CK) were studied before and after each exercise bout. Muscle tenderness was greatest after the first bout and thereafter progressively decreased. Very high plasma CK levels (1,500–11,000 IU/l) occurred after the first bout, but the second and third bouts did not significantly affect the plasma CK. After each bout the strength was reduced by ~50% and after 2 wk had only recovered to 80% of preexercise values. Each exercise bout produced a marked shift of the force-frequency curve to the right which took approximately 2 wk to recover. The recovery rate of both strength and force-frequency characteristics was faster after the second and third bouts. Since the adaptation occurred after the performance of maximal contractions it cannot have been a result of changes in motor unit recruitment. The observed training effect of repeated exercise was not a consequence of the muscle becoming either stronger or more resistant to fatigue.

training; fatigue; elbow flexors; creatine kinase

PREVIOUS STUDIES have shown that exercise involving high-force eccentric muscle contractions can produce temporary muscle pain and damage. Evidence of damage includes disruption of muscle fibers (8, 14, 21), increased circulating levels of muscle proteins (3, 9, 18, 19), muscle uptake of radionuclides (17, 20, 25), and changes in voluntary strength and contractile properties in the immediate postexercise period (5, 22).

One interesting feature is the rapidity with which the pain and muscle damage are reduced or abolished by repeated exercise (2–4, 7, 11, 13). In our previous work (11), release of creatine kinase (CK) and muscle tenderness were measured after one bout of exercise involving contractions at 50% maximum force. When the exercise was repeated 1 wk later both pain and CK release were much reduced. This training effect was found to last ~6 wk, indicating a considerable and long-lasting adaptation (11). There are a number of possible explanations for the training which include the following.

*1*) There may be a change in the pattern of motor unit recruitment. Previous studies have used submaximal contractions that allow the possibility that training may cause a change in the order of motor unit recruitment such that either susceptible fibers are spared on the second and subsequent occasions or more fibers are recruited and the force-fiber ratio is reduced. This can be investigated by using maximal contractions.

*2*) There may be some adaptation in the muscle fibers such that they become more resistant to the fatiguing and damaging effects of eccentric exercise. This might be evident as a change in the strength and contractile properties of the muscle.

*3*) It has been suggested that eccentric exercise preferentially damages a population of fibers that are nearing the end of the cycle of growth and replacement (1). In this case, a decrement in force generation would occur after the first exercise which would be absent on subsequent occasions.

The purpose of the present investigation was to test these possibilities. To this end we have compared the changes in strength and contractile properties with the pain and plasma CK response after three bouts of maximal eccentric contractions of the elbow flexors spaced at two weekly intervals.

## METHODS

*Subjects.* Eight subjects (5 females and 3 males) ranging in age from 24 to 43 yr took part in the study. They were healthy and active but none was participating in any training programs, and they had not participated in studies involving eccentric contractions for at least 3 mo. Their physical characteristics are shown in Table 1. All were fully informed of the nature and risks of the procedures to be used. The study had the approval of the Committee for the Ethics of Human Procedures at University College Hospital.

*Exercise.* To exercise the elbow flexors the subject sat in an adjustable chair with the upper arm supported on a shelf, the height of which could be adjusted to bring the upper arm to an angle of 90° with the body. An inextensible cord passed from a wrist cuff to a pulley attached to the wall in front of the subject and then to the experimenter. The latter was able to forcibly extend the elbow and overcome the maximal effort of the subject by means of a winch with a mechanical advantage of ~10:1. The signal from the strain gauge was amplified and displayed on an ultraviolet recorder. Details of the procedure have been given elsewhere (12).

TABLE 1. *Physical characteristics of the subjects*

| Subj. No. | Sex | Age | Ht, m | Wt, kg |
|-----------|-----|-----|-------|--------|
| 1 | M | 43 | 1.76 | 71.9 |
| 2 | M | 41 | 1.68 | 73.4 |
| 3 | F | 37 | 1.67 | 68.4 |
| 4 | F | 35 | 1.76 | 65.3 |
| 5 | M | 39 | 1.64 | 69.9 |
| 6 | F | 24 | 1.56 | 47.7 |
| 7 | F | 26 | 1.60 | 48.3 |
| 8 | F | 40 | 1.58 | 49.6 |

The subjects performed one eccentric maximal voluntary contraction every 15 s for 20 min. Each contraction lasted ~2 s. The range of elbow movement was from full flexion to full extension with care being taken not to hyperextend the elbow. The exercise was repeated on three occasions that were separated by an interval of 2 wk.

*Force measurement.* Isometric maximum voluntary contraction (MVC) force was measured before and immediately after each exercise period and repeated at daily intervals thereafter. Three attempts were made on each occasion, and the highest was taken as the maximal force. When recording force from voluntary contractions of painful muscles there is the possibility that the contractions are submaximal, even with well-motivated subjects. To determine whether this was the case electrical stimulation (at both 1 and 100 Hz) was superimposed on the voluntary contractions. With this technique additional force is generated by the electrical stimulation only if the voluntary contractions are submaximal (23).

The force-frequency relationship was determined by percutaneous electrical stimulation of the biceps at 1 Hz (for 5 s) and 10, 20, 50, 80, and 100 Hz for 2 s each. Damp electrodes ($8 cm^2$) were bandaged onto the upper arm, over the proximal and distal ends of the biceps. Square-wave pulses of 50 $\mu$s and up to 100 V were used (Digitimer dual high-voltage stimulator, Hertfordshire, UK). The force at each frequency was expressed as a percent of that generated by stimulation at 100 Hz (1/100%, 10/100%, etc.). Care was taken to ensure that at least 25% of the elbow flexors were stimulated, comparing the stimulated contractions with the MVC.

For all the isometric measurements the wrist was tied and the upper arm supported to prevent movement of the arm in any direction. The shoulder was prevented from forward movement by being clamped to the back of the chair. In our experience daily testing in this way neither causes muscle pain or damage nor does it modify the response to eccentric exercise.

*Plasma CK.* Blood was collected from an antecubital vein into heparinized tubes before and at daily intervals after each exercise period. CK activity was determined by the Department of Chemical Pathology, University College Hospital, using a Boehringer Mannheim kit method with $N$-acetylcysteine activation. The normal range using this method is 60-190 IU/l.

*Muscle tenderness.* This was measured daily after the exercise by recording the force required to elicit tenderness at fixed sites on the skin over the biceps and brachioradialis (22). Twelve test sites were spaced ~2 cm apart over the muscle surface and marked with indelible

ink. At each site a gradually increasing force was applied through a flat plastic disc of 2 cm diam attached to a force transducer with digital readout (Penny & Giles Transducers, Christchurch, Dorset, UK). The subject was asked to indicate when the sensation of pressure changed to discomfort, and the force at that point was recorded. Tenderness was recorded as absent if not reported before 30 N. The value of the force (N) has been subtracted from 30 to give a tenderness score.

*Statistics.* Data were analyzed using repeated-measures analysis of variance. Statistical significance was set at $P < 0.05$.

## RESULTS

*Maximal voluntary force.* Superimposition of electrical stimulation on the voluntary contractions demonstrated that all subjects were able to fully activate their muscles during the isometric contractions despite any discomfort.

The values for MVC, expressed as a percent of the initial preexercise force, are presented in Fig. 1. There was a large decrease in voluntary force (~50%) immediately after the three exercise periods. There was no significant difference between the extent of force loss after the three bouts. Recovery was slow and there were differences between the rate of recovery from the first compared with that from the second and third occasions. After the first exercise force had hardly improved after 24 h (49% initial), whereas after *exercise bouts 2* and *3* there was recovery to 66 and 69%, respectively. The values for *bouts 2* and *3* at this time differed significantly from *bout 1* but not from each other. After the first bout of exercise the MVC remained significantly lower than the initial force until 1 wk after the third and final exercise. There was no evidence of an increase in strength as a result of the exercise in any of the subjects.

*Contractile properties.* After exercise there was a significant decrease in force generation at low frequencies of stimulation. A representative index of this is the force generated by 20 Hz stimulation expressed as a percent of that at 100 Hz (20/100%). These values are presented in Fig. 2. A large decrease in the 20/100% value to ~40% was seen immediately after each exercise bout, there being no significant difference between the decrement seen on the three occasions. As with the MVC measurements (Fig. 1) there was a faster recovery after the second and third exercise bouts compared with the first. The 20/100% value was significantly less than the initial value until the 9th day after the first exercise but was not significantly different from preexercise values by the 4th and 3rd days after the second and third bouts of exercise.

*Plasma CK.* Although there was a large intersubject variation in the CK response, all showed a substantial increase after *bout 1*, the peak values ranging from 1,570 to 10,904 IU/l, which was much greater than the response seen after *bouts 2* and *3* (Fig. 3 and Table 2). The plasma CK only showed a significant increase after the first exercise.

*Muscle tenderness.* Muscle tenderness values are presented in Fig. 4. A significant difference in tenderness was found among the three bouts, with each resulting in
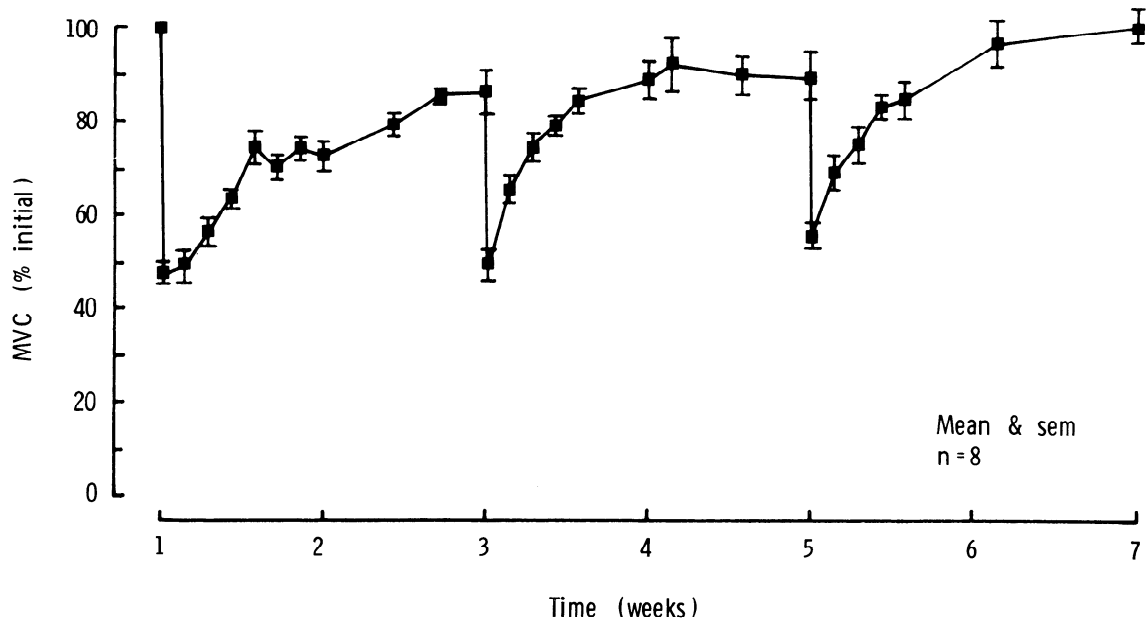
FIG. 1. Maximal isometric strength during training period. Exercise was performed in 1st, 3rd, and 5th wk. MVC, maximum voluntary contraction.
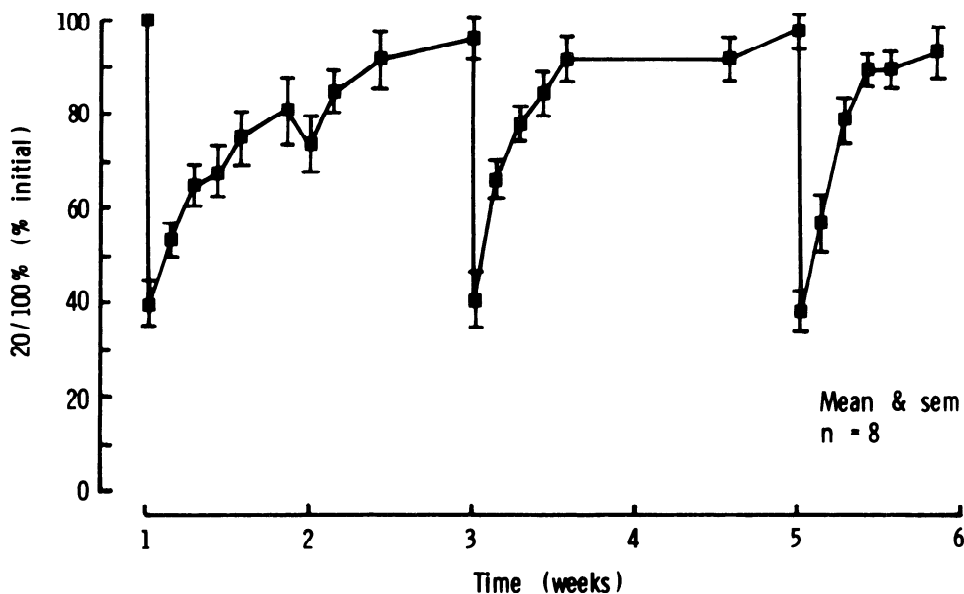


FIG. 2. Force-frequency relationship during training period. Exercise was performed in 1st, 3rd, and 5th wk.

progressively less tenderness. The peak pain, which occurred on *day 2* after the first exercise and *day 1* after the second and third occasions, was significantly less on the second compared with the first occasion and less again on the third compared with the second occasion. The sums of the pain scores over the 4 days after exercise were reduced, being 43 and 35% of the initial response after the second and third bouts, respectively.

There were no obvious relationships among the variables measured such as the extent of force loss and subsequent pain or increase in plasma CK. Changes in force and contractile properties were similar in all the subjects (Figs. 1 and 2), but there was considerable variation in the pain and release of CK. There was no obvious relationship between the extent of the response and parameters such as age, sex, or general level of physical fitness of the subjects (Tables 1 and 3).

## DISCUSSION

The training effects of repeated high-force, eccentric exercise were very obvious both from subjective reports and objective measurements of pain and muscle damage. Muscle pain and stiffness were much reduced after the second and third bouts of exercise compared with the first.

The most striking training response was seen in the CK efflux. The enzyme release after the first exercise was similar in magnitude and time course to those that we have previously reported (14, 18, 19), being both very large and characteristically delayed for several days after eccentric exercise. Despite variation between individuals in the extent of the release, the plasma levels were always greatest, by up to two orders of magnitude, after the first bout of exercise. Although the mechanism of the exercise-
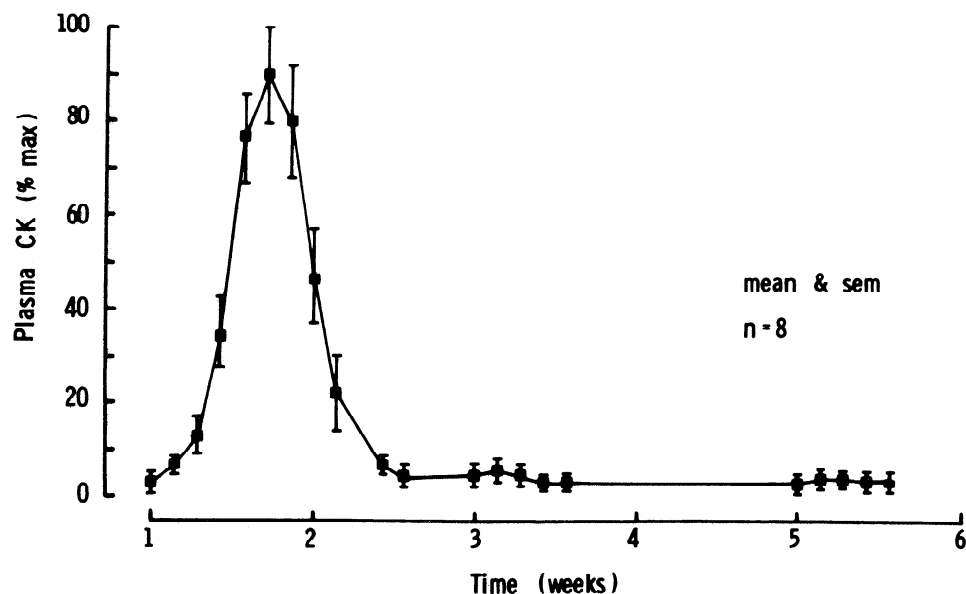
FIG. 3. Plasma creatine kinase (CK) during training period. Note marked training effect seen here in comparison with force measurements. Exercise was performed in 1st, 3rd, and 5th wk.

TABLE 2. *Range of plasma CK values before and after the three exercise bouts*

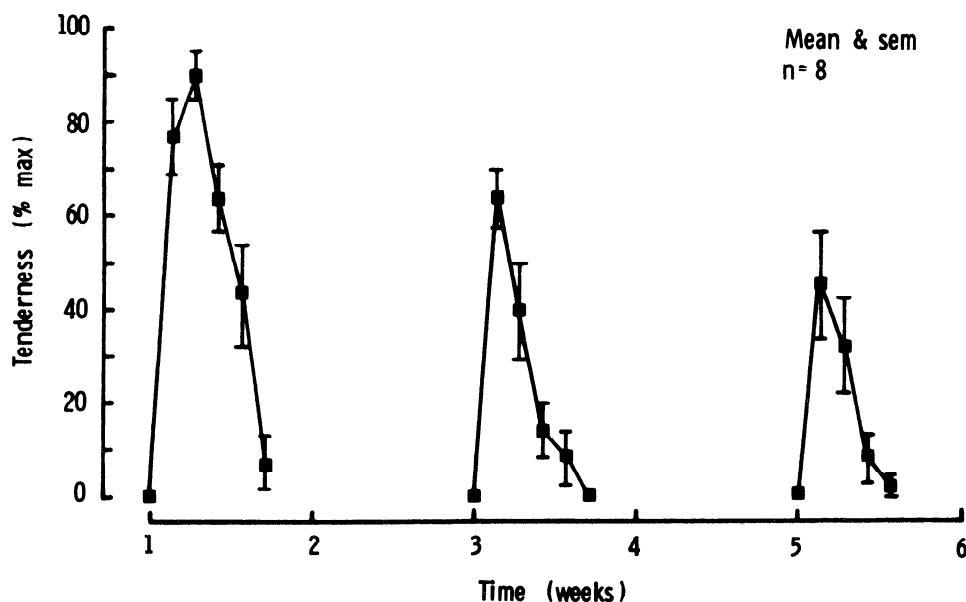| Exercise Bout | Preexercise | Postexercise, days | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| 1 | 57–250 | 88–1,133 | 81–3,074 | 177–7,978 | 735–10,904 | 1,854–6,074 | 1,402–4,535 | 709–3,313 | 365–2,222 |
| 2 | 90–208 | 97–328 | 64–327 | 49–250 | 52–237 | 65–175 | | | |
| 3 | 36–185 | 36–230 | 31–137 | 31–167 | | | | | |

CK, creatine kinase, in IU/l.



FIG. 4. Muscle tenderness during training period. Pain progressively decreased after each exercise bout. Exercise was performed in 1st, 3rd, and 5th wk.

induced enzyme efflux is unknown, it is generally assumed to reflect some form of membrane damage.

Suprisingly, in view of the major changes in pain and enzyme release, the training had no effect on the maximum isometric strength, the extent of force loss after the exercise, or the changes in contractile properties of the muscles. Other workers have reported similar reductions in the MVC immediately after a single bout of submaximal eccentric contractions (5, 16) and it has often been assumed that the apparent force loss was largely due to the pain preventing subjects from fully

activating their muscles (7, 15, 24). In the present study the strength measurements were shown to be from fully activated muscles, and thus the force loss was the result of changes in the contractile elements.

Although both the pain and CK release responded to training, it is unlikely that these were causally related. Whereas the CK response was virtually eliminated after the first bout of exercise, the discomfort was only reduced in a stepwise manner. Furthermore, the CK release and pain are always separated in time (see Figs. 3 and 4) and are often dissociated in magnitude. Thus one individual

TABLE 3. *Relationship between muscle strength, fatigue, damage, and pain after eccentric contractions*

| Subj. No. | MVC | | 20/100%, % initial | Peak CK, IU/l | Peak Tenderness Score |
|---|---|---|---|---|---|
| | n | % initial | | | |
| 1 | 310 | 47.8 | 53.7 | 5,586 | 12.6 |
| 2 | 263 | 43.9 | 72.9 | 1,854 | 9.0 |
| 3 | 186 | 57.8 | 75.2 | 6,074 | 14.0 |
| 4 | 167 | 51.4 | 71.6 | 5,464 | 10.2 |
| 5 | 152 | 56.3 | 52.0 | 9,426 | 17.9 |
| 6 | 129 | 57.1 | 58.9 | 1,570 | 15.0 |
| 7 | 115 | 55.9 | 56.5 | 10,904 | 10.6 |
| 8 | 103 | 60.7 | 74.6 | 2,494 | 14.9 |

Relationship between initial strength of subjects and changes in force generation, plasma creatine kinase (CK), and muscle pain after first bout of exercise. Force measurements are those before and immediately after exercise period. MVC, maximum voluntary contraction; 20/100%, force generated by 20 Hz of stimulation expressed as percent of that at 100 Hz (% initial).

might have considerable pain but little or no evidence of muscle membrane damage.

There is evidence that the pain and stiffness experienced after eccentric contractions are a consequence of shortening of the noncontractile material that is arranged in parallel with the contractile material (10, 13). This may be a response to some form of damage to the connective tissue, and if so the training could have caused some adaptation of this tissue.

We have previously studied the training effect using eccentric contractions at 50% maximum (11) and found results similar to those reported here. With submaximal contractions it was possible that changes might have occurred in the recruitment pattern so that, after training, less susceptible motor units were used. Alternatively, more motor units could be recruited, thereby reducing the force per fiber. The results of the present study, using maximum contractions, show that neither of these possibilities can be the entire explanation since maximal contractions require the activation of all motor units so there is no possibility of change in the extent or order of recruitment.

The training program did not increase the voluntary strength of the muscle, nor did it prevent the loss of force or change in contractile properties (shown by the 20/100% value, Fig. 2) that occurred to a similar extent after each bout of exercise. This demonstrates that the adaptation did not involve any change in the strength or contractile properties of the muscle or its ability to withstand fatigue.

The present results provide some evidence in favor of the possibility, suggested by Armstrong (1), that the first bout of exercise causes damage and destruction to a population of susceptible fibers, possibly those near the end of their life cycle. There was a notable difference in the time course of recovery of force generation after the first exercise bout compared with the second and third, being slower after the first bout (Figs. 1 and 5). This difference in rate of recovery was also evident with the 20/100% value (Fig. 2). The slow recovery of force and contractile properties was associated with the release of CK from the muscle. It is possible that the loss of force and release of soluble constituents represent the removal
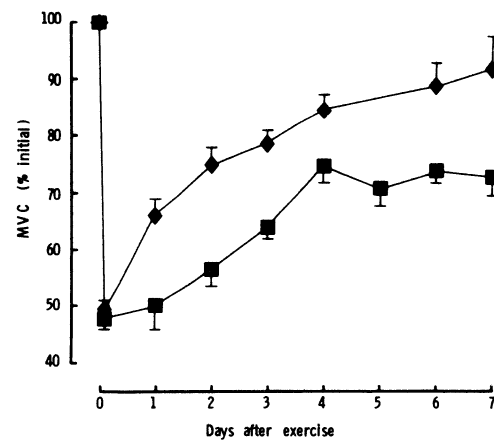


FIG. 5. Changes in maximum voluntary contraction (MVC) force (means ± SE) after first (*squares*) and second (*diamonds*) exercise bouts. Values after second exercise bout were significantly greater than after first at all times shown with exception of those on *day 4*.

and replacement of irreparably damaged fibers that may have been particularly susceptible to damage, possibly being near the end of the natural cycle of cell turnover. This could equally well apply to either the entire fiber or a part of it. After the initial damage they may fail to repair and subsequently undergo degeneration, recovering slowly and releasing soluble enzymes in the process. The more resilient fibers, or parts of fibers, remain and these are able to withstand the effects of eccentric exercise without undergoing a process of degeneration and enzyme release.

This seems to be the first report in which the time course of the recovery of both strength and contractile properties has been followed for more than a few days. The MVC was very slow to recover, only returning to ~80% of the initial value in 2 wk. Interestingly, the subjects were aware of impaired muscle function only after the first exercise bout in which most pain was experienced. They therefore felt that the training had improved their performance, yet objective testing showed appreciable and long-lasting weakness.

This study does not identify the mechanism by which the pain and damage are reduced by training. However, it does suggest that the prevention of enzyme release with training may be due to the removal of contractile material that is particularly sensitive to damage.

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