

Damage to human muscle from eccentric exercise after training with concentric exercise

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1. It is known that a period of eccentric exercise provides protection against damage to muscle from subsequent eccentric exercise. Here we ask, does concentric exercise do the opposite, make muscle more prone to damage?
2. The triceps surae muscle group of one leg in each of eight human subjects was subjected to 30 min of concentric exercise per day, for 5 days. At the end of the training period there was a small but significant increase in passive torque in the exercised muscle ($P < 0.05$), with no changes in the untrained muscle.
3. After a single period of eccentric exercise, angle–torque curves for muscles of both legs shifted in the direction of longer muscle lengths, suggestive of an increase in series compliance. The shift in the concentrically trained muscle was significantly greater over the first 48 h post-exercise ($P < 0.05$).
4. The volume of the trained leg increased significantly more than the untrained leg for five subjects over 72 h post-exercise ($P < 0.05$). Peak torque fell, passive stiffness increased and both muscles became sore, but with no significant differences between the two legs.
5. It is concluded that a period of concentric exercise increases the susceptibility of muscle to changes associated with the damage from eccentric exercise.

We have all experienced the feeling of exhaustion and muscle weakness after a period of intense exercise. However, if we carry out a particular kind of exercise, eccentric exercise, where muscles are lengthened while they contract, we will not only experience the usual sensations of fatigue but the exercised muscles will become sore the next day. Eccentric contractions are involved in all activities where the contracting muscles exert a braking action to control motion of the body. So, for example, knee extensors control the forward motion of the body when we walk down a steep slope. Other activities involving eccentric contractions include horse-riding and skiing.

While it has been known for a long time that eccentric exercise is followed by delayed-onset muscle soreness (DOMS) (Hough, 1902), the initial event which brings about the soreness remains the subject of debate. Both chemical and mechanical mechanisms have been considered (for a review, see Armstrong *et al.* 1991). In our studies we have been seeking evidence in support of a hypothesis put forward by one of us (Morgan, 1990), which proposes a non-uniform distribution of the length change during the eccentric contraction, leading to overextension of some sarcomeres in muscle fibres and eventual damage in these fibres.

It is a common experience that a second period of eccentric exercise a week after the first is accompanied by much less soreness (Jones & Newham, 1985; Newham *et al.* 1987; Clarkson & Tremblay, 1988). Some kind of adaptation process has taken place within the muscle. It has been hypothesized (Morgan, 1990) that the adaptation is the result of incorporation of additional sarcomeres into the exercised muscle fibres. The length change during a subsequent eccentric contraction therefore brings fewer sarcomeres into a length range where they become unstable and overextended.

Direct support for such a mechanism is provided by the experiments of Lynn & Morgan (1994). They showed that, after a week of training, muscle fibres of knee extensor muscles in rats, trained to run downhill on a treadmill, contained significantly more sarcomeres than muscles fibres of sedentary animals or animals trained to run uphill over the same period. Interestingly, the uphill-trained animals had fewer sarcomeres per fibre than either the sedentary or downhill-trained rats. This finding suggested that the postulated adaptation process was bidirectional and could lead to either an increase or a decrease in sarcomere number depending on the kind of exercise. Where the exercise was predominantly concentric (uphill running), it would be

advantageous for the muscle to be able to generate significant levels of force at short muscle lengths. This could be achieved by reducing the number of sarcomeres in series in muscle fibres and therefore shift the length–tension relationship of the muscle in the direction of shorter muscle lengths. It would also increase efficiency by reducing the mass of muscle required for a given level of force.

Here we describe experiments designed to test the hypothesis that human subjects who have undergone regular concentric exercise in the ankle extensor muscles of one leg will, after a test bout of eccentric exercise, show more severe changes associated with damage in that leg compared with the untrained leg. The principal measure used was a shift in the length–tension relationship of the muscle. Previous experiments from our laboratory have shown that this is a reliable indicator of damage immediately after eccentric exercise, an indicator which is independent of fatigue (Wood *et al.* 1993; Jones *et al.* 1997; Talbot & Morgan, 1998). Other measures of damage sought were the drop in force, rise in passive tension, degree of swelling and level of muscle soreness. These latter indicators are delayed in onset, or, in the case of force, complicated by the effects of fatigue.

METHODS

A total of eight subjects participated in the study, four male (mean age, 24 years) and four female (mean age, 22 years). All subjects were in good health, did not participate in regular exercise programmes other than that of this study, and none had any musculo-skeletal defects. All gave informed consent to participate in the study which had previously been approved by the local ethics committee.

The equipment

The muscle group studied was triceps surae. The testing equipment used was similar to that described in Jones *et al.* (1997). It consisted of an adjustable chair supported by a steel frame to which was bolted a rotatable footplate. The axle about which the footplate rotated was aligned with the ankle joint and four strain gauges were cemented to the shaft of the axle to allow measurement of torque. Once a subject's foot was firmly strapped to the footplate the ankle angle could be systematically altered by rotating the footplate. Ankle angle was the angle subtended between the footplate and the shin, keeping the angle at the knee constant at 140 deg by means of a knee brace.

The angle–torque relationship for the triceps was determined by measuring torque over a range of ankle angles from 90 to 50 deg, a decrease in angle representing an increase in muscle length (Fig. 1). The muscle was contracted using double-pulse stimulation of the tibial nerve at the popliteal fossa using 1 ms pulses 20 ms apart. Current strength was sufficient to produce a maximum contraction and was typically about 30 mA. Previous experience had shown that reproducible angle–torque curves could be constructed using these stimulus parameters.

For each contraction, peak torque was measured and then plotted against ankle angle (Fig. 1). A Gaussian curve was fitted to torque values greater than 75% of the peak and this provided a convenient means of locating the optimum angle for torque generation, which typically lay in the range of ankle angles of

65–75 deg. Curve fitting was done using a computer program, IGOR Pro (WaveMetrics, Lake Oswego, OR, USA) running on an Apple Macintosh computer.

As well as constructing angle–torque curves, measurements were made of passive torque, leg volume and muscle soreness. Passive torque was measured by moving the ankle joint in 10 deg steps over the range 50–90 deg while subjects remained completely relaxed. Each movement was carried out at the same speed and was completed in about 1 s. Tension was then allowed to fall to a steady level before the next movement was made. Values were measured immediately before each movement. Leg volume below the knee was measured by placing the leg in a Perspex container filled with water and noting the amount of water displaced by the leg. Values for passive torque and leg volume were compared before and after the eccentric exercise. Soreness ratings were noted each day after the eccentric exercise, and subjects were asked to rate pain intensity on a scale of 1–10 during contraction or palpation of the muscle and during normal walking.

The experiment

The experimental protocol which was used with each subject was carried out in four stages. Initially, control angle–torque curves were constructed for triceps of both legs, repeated 3–4 times over several days to establish repeatability of the measurements. In stage two, the triceps of one leg was subjected to a regular training regime carried out over 1 week. The exercise required the subjects to plantarflex one foot, lifting the weight of the body, so that they were now standing on the toes of that foot. Then by rotating their body and leaning over, subjects transferred their weight to the other foot which was brought down flat on the ground, without any dorsiflexion or plantarflexion. This cycle was repeated at an approximate rate of fifteen contractions per minute for 30 min, each day, over 5 consecutive days. To ensure that their muscles were generating sufficient levels of force during the contractions, subjects carried a 10 kg weight in a backpack. The triceps of one leg therefore underwent regular concentric exercise while the other leg remained untrained and was subjected to just the normal, everyday activity.

Stage three was carried out 3–4 days after the end of the period of concentric training. The muscles of both legs were subjected to a period of eccentric exercise. Subjects were required to walk backwards on a moving treadmill (Heartmaster, Tetley Technologies). The treadmill was inclined at an approximate angle of 13 deg, and moved at a speed, which was typically 3.5 km h⁻¹, to achieve a stepping rate of about thirty per minute for each leg. Subjects were asked to step backwards, downhill, one leg after the other, using a toe-to-heel action to ensure that the triceps of each leg was stretched while bearing the weight of the body. Subjects were asked to exercise both legs as evenly as possible over the 1 h period of exercise.

In stage four, angle–torque curves were measured immediately before, immediately after and 2 h after the eccentric exercise. Additional curves were constructed on each of the following 3 days.

Statistics

For all parameters measured, means and standard errors of the mean were calculated. Student's paired *t* tests were used to compare measurements from before and after training and for comparisons between the two legs. A three-factor analysis of variance with interactions (ANOVA) was used to test the significance of the changes following the eccentric exercise. The factors were time, muscle (trained or untrained) and subject. Where

an ANOVA was significant ($P < 0.05$), an LSD (least significant difference) *post hoc* test was used to look for significant differences between the two legs at different times after the exercise. The analysis program used was Data Desk (Ithaca, NY, USA)

RESULTS

Effects of training

The possibility was considered that training had produced changes in some of the parameters that were to be measured after the eccentric exercise. However, comparisons between muscles of both legs over the training period showed no significant shifts in the optima of angle–torque curves, no changes in peak torque or leg volume and no subject reported that the training had made their muscles sore. There was, however, a small but significant increase in passive torque in the trained muscle (see below).

Changes after eccentric exercise

The angle–torque curve. We have shown previously that accompanying the damage of eccentric exercise there is a shift to the right of the length–tension curve of the muscle as a result of the increased compliance provided by the injured muscle fibres (Wood *et al.* 1993; Jones *et al.* 1997; Talbot & Morgan, 1998; see also Katz, 1939). Shifts in the curves measured here were similarly used as indicators of damage.

An example of an angle–torque curve for one subject measured immediately before and immediately after the period of eccentric exercise is shown in Fig. 1. There were two effects of the exercise. One was a large drop in torque, presumably due to the combined effects of muscle fatigue and damage to muscle fibres. For this subject torque had dropped from 30 N m at the optimum angle to 19 N m, a fall of 37%. Secondly, the curve measured after the exercise showed a shift to the right, that is, towards longer muscle lengths. The optimum angle for torque generation had shifted from an ankle angle of 70 to 65 deg, that is, the ankle had to be dorsiflexed by 5 deg more after the exercise to reach the optimum angle.

Figure 1. Changes in angle–torque curves after eccentric exercise

Curves for triceps surae of a subject immediately before (●) and after (○) a period of eccentric exercise. Isometric torque at each ankle angle was produced by double-pulse stimulation of the tibial nerve. Gaussian curves were fitted to values above 75% of the optimum torque. The arrows indicate the angle for optimum torque. This example is from a muscle which had undergone 5 days of concentric training. Note both the drop in torque and shift in the direction of longer muscle lengths of the angle–torque relationship after the eccentric exercise.

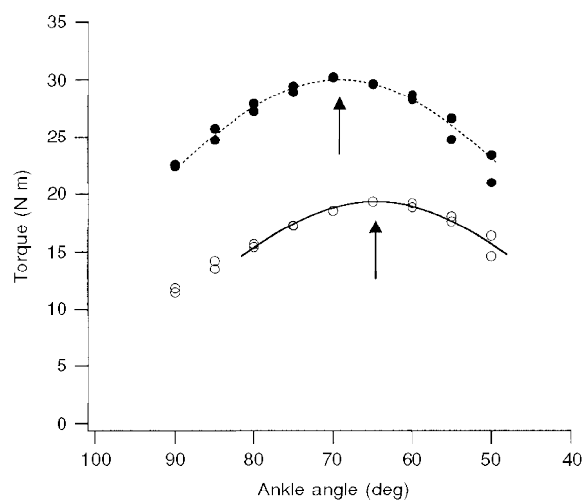
Our working hypothesis was that the periods of concentric exercise carried out with the triceps of one leg during the week before the single bout of eccentric exercise increased the susceptibility to damage. This prediction was borne out when the shifts in optimum angles for the trained and untrained muscles of each subject were compared.

Shift in optimum angle. After the eccentric exercise muscles of both legs showed shifts to the right in their angle–torque curves. The pooled data for the eight subjects are shown in Fig. 2. The largest mean shift in the curve was measured immediately after the eccentric exercise in muscles of both legs. It then reversed back to control values over the next 24 h for the untrained muscles and over 48 h for the trained muscles. More importantly for our hypothesis, the mean shift measured 2 h after the eccentric exercise was significantly larger in the muscles which had undergone concentric exercise training.

For the concentrically trained muscles the mean shift for the eight subjects was 4.4 ± 1.0 deg. The control muscles showed a mean shift of 2.8 ± 0.8 deg. The difference between the two legs measured at 0, 2, 24 and 48 h was significant (three-factor ANOVA; $P < 0.05$).

Peak torque. Immediately after the eccentric exercise peak torque dropped to a mean of $69 \pm 4.4\%$ of control in the trained muscles and to $68 \pm 5.2\%$ in the untrained muscles (Fig. 3). Torque recovered over the next 72 h to near-control values. The drop in torque was not significantly different between the trained and untrained muscles. There was a slight but non-significant trend towards a more rapid recovery in the untrained muscle (three-factor ANOVA; $P = 0.14$).

Leg volume. Another indicator of damage was an increase in leg volume as a result of muscle swelling. This was measured for only five subjects for the full post-exercise period up to 72 h. Mean leg volume was 4.2 ± 0.25 l before the eccentric exercise and the first signs of swelling appeared at 2 h after the exercise (Fig. 4). Swelling continued to increase to reach a peak at 72 h post-exercise,



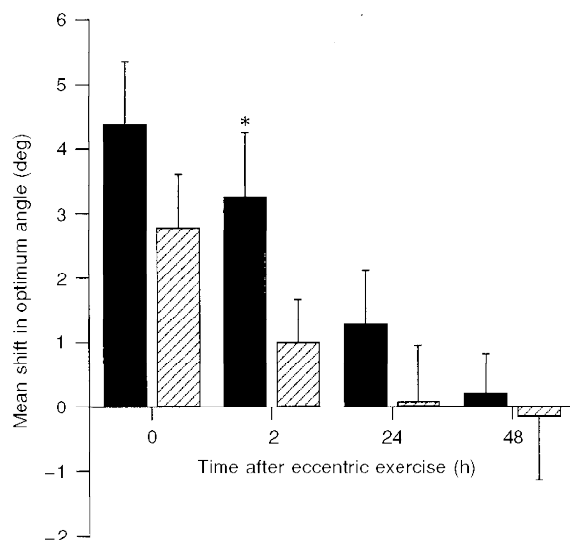


Figure 2. Shifts in optimum angle after eccentric exercise

Mean (\pm s.e.m.) shift in optimum angle of angle-torque curves for triceps surae of all eight subjects after a 1 h period of eccentric exercise in concentrically trained muscles (■) and untrained muscles (▨). All values are expressed relative to the optimum angle before the exercise. * Significant difference between trained and untrained muscles ($P < 0.01$).

where the trained leg had swollen by a mean of $2.8 \pm 0.2\%$ while the untrained leg had swollen by a mean of $1.8 \pm 0.3\%$ (Fig. 4). There was a significant difference in volume between the two legs over the period 2–72 h (three-factor ANOVA; $P < 0.05$). Swelling for the control leg had typically subsided by about the end of 1 week while for the trained leg it persisted for longer.

Passive torque. All of the torque values cited above were active torques. However, passive torque was also measured, over the range 50–90 deg, while the subject sat completely relaxed. Passive torque was the only measured parameter that changed during the training period. By the end of the training period, mean passive torque had risen by a small but significant amount in the trained leg at all angles (three-factor ANOVA; $P < 0.05$). There was no significant change in mean passive torque for the untrained muscles. Passive torque rose further in muscles of both legs after the eccentric exercise and reached its peak at 24 h post-exercise. However, for the group the difference between the two legs post-exercise was not significant, keeping in mind that for the trained muscles passive torque was already higher than its initial control value.

Soreness. All subjects experienced DOMS in triceps muscles of both legs after the eccentric exercise. It first became noticeable 1 day after the exercise, reached its peak at 48 h and had subsided by the end of 1 week. Subjects found it difficult to distinguish between soreness levels for the two legs. Values scored on a scale of 10 averaged 7.2 ± 0.4 for the trained muscle and 7.0 ± 0.6 for the untrained muscle. Soreness levels were not significantly different for the two legs.

DISCUSSION

The main result achieved in this study was that rather mild concentric exercise, carried out by the muscle of one leg, for 30 minutes per day, for 5 days, when compared with the other, untrained muscle, rendered it more susceptible to changes associated with the damage of eccentric exercise.

Our interpretation of these findings is based on a hypothesis which aims to explain the initial event in eccentric exercise that leads to muscle damage and DOMS. According to this hypothesis, muscle fibres do not lengthen uniformly during an eccentric contraction because of variations in sarcomere

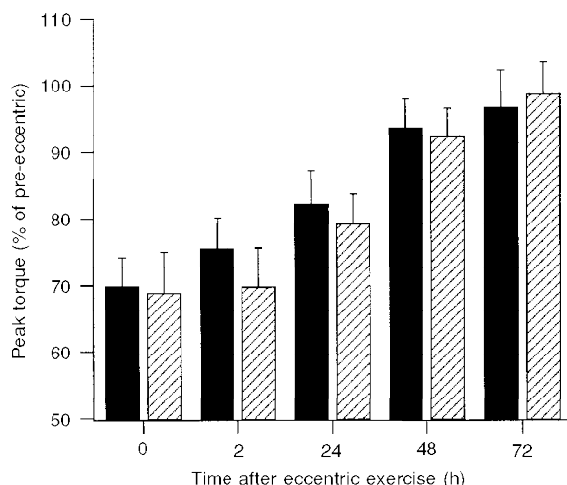


Figure 3. Fall in peak torque after eccentric exercise

Pooled data showing means (\pm s.e.m.) for the changes in peak torque at various times after the eccentric exercise in both the concentrically trained muscles (■) and untrained muscles (▨). Torque is expressed as a percentage of the pre-eccentric exercise torque.

lengths and non-uniform areas of cross-section along myofibrils. Regions of the myofibril containing weak sarcomeres will lengthen at the expense of adjacent areas. If these weak sarcomeres are at a length corresponding to the plateau or descending limb of their length–tension relationship, they will become even weaker as they elongate and so lengthen progressively faster. Once they have reached the ‘yield point’ in their force–velocity relationship for lengthening, they will continue lengthening, uncontrollably, until further elongation is halted by the rising tension in passive supporting structures. There is evidence that most of the overextended sarcomeres reinterdigitate on muscle relaxation, while a few fail to do so and become disrupted (Talbot & Morgan, 1996; Jones *et al.* 1997). Such disruptions place extra strain on neighbouring sarcomeres leading to spread of the damage during subsequent eccentric contractions.

During repeated eccentric contractions, as the areas of disruption become larger, a point is reached where membranous elements within the muscle fibres, associated with the sarcoplasmic reticulum and t-tubule systems, become damaged. This would be followed by uncontrolled movement of calcium ions into the sarcoplasm and development of a contraction clot. On occasion the entire fibre may die. The link between muscle damage and DOMS is thought to be provided by the inflammatory process triggered by the damage (Smith, 1991) and it is this which is thought to be responsible for the muscle swelling.

We propose that training led to adaptive changes in the muscle directed at optimizing muscle performance during the concentric exercise and in the process rendered it more vulnerable to the damage of eccentric exercise. Lynn & Morgan (1994) reported that a group of rats, which had run uphill on a treadmill for 15–30 minutes per day for 1 week, had fewer sarcomeres in muscle fibres of knee extensors than in muscles from a sedentary group. A reduction in the number of sarcomeres in series in muscle fibres will result in a shift of the length–tension relationship in the direction of shorter lengths. That, in turn, will allow the muscle to develop more force at short lengths, given that during uphill

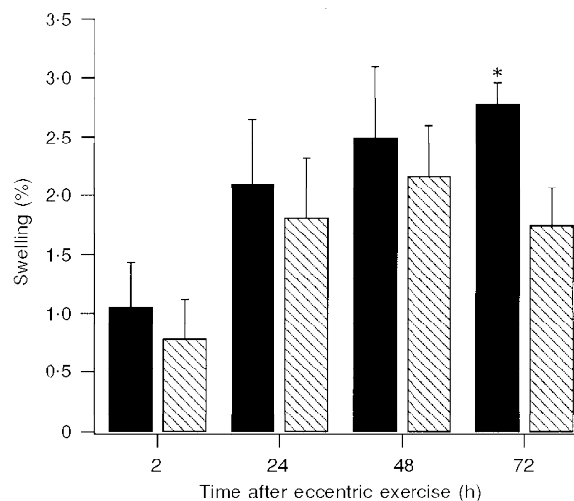
running knee extensors undergo a predominantly shortening contraction. A reduction in the number of sarcomeres in muscle fibres will, according to our hypothesis, make the muscles more susceptible to the damage of eccentric exercise.

The interpretation of our findings is based on differences in indicators of damage in the trained muscles compared with the untrained muscles. Thus after the acute bout of eccentric exercise, the trained muscles exhibited a greater shift in their length–tension relationship and more swelling than the untrained muscles. The greater shift in the length–tension relationship suggests a larger increase in series compliance in the trained muscle as a result of more disruption of sarcomeres in muscle fibres. Swelling was significantly greater in the trained muscle at 72 h after exercise (Fig. 4). This presumably reflects the more extensive repair processes occurring at this time (Smith, 1991). Another indicator of damage is a drop in force. A drop in force immediately after the exercise will be due to both damage and fatigue. In our experiments, the drop in torque after the eccentric exercise was not significantly different for trained and untrained muscles. This raises the possibility that the extra work carried out by the trained muscles may have made them less susceptible to fatigue and so, even though they may have become more damaged by the eccentric exercise than the untrained muscles, this did not show up in the overall drop in force. Finally, it should be remembered that a drop in force is not a very reliable indicator of the amount of damage. Hesselink *et al.* (1996) found a poor correlation between muscle damage evaluated histologically and the measured drop in force.

If, as we propose, the training led to a reduction in sarcomere numbers in muscle fibres and so made them more vulnerable to the damage of eccentric exercise, it might have been expected that at the end of the training period these muscles would show a shift in the direction of shorter muscle lengths in their length–tension curve. There was, in fact, a small, but insignificant, shift for the trained muscles. Passive torque, on the other hand, increased significantly in the trained muscles. How can these two findings be reconciled? Assuming that the shift in the passive curves

Figure 4. Increases in leg volume after eccentric exercise

Pooled data showing the mean (\pm S.E.M.) amount of swelling of muscles of both legs for 5 subjects at various times after the eccentric exercise. Swelling is expressed as percentage increase in leg volume from the pre-exercise value. ■, trained muscles; ▨, untrained muscles. * Significant difference between trained and untrained muscles ($P < 0.01$).



was due to reductions in the number of sarcomeres in muscle fibres, a comparable shift should have been seen in the active curves. The mean shift in the passive curve represents, for a given level of torque, approximately a 1 deg change in ankle angle. Control angle–torque curves for the eight subjects were distributed over a similar range. In other words, a shift of 1 deg would be lost in the variability of the measurement. It would require a larger sample and, preferably, more severe training exercise to demonstrate a significant shift in angle–torque curves during training.

There is recent evidence supporting our findings (Ploutz-Snyder *et al.* 1998). It was found that subjects who underwent 9 weeks of concentric training of knee extensors of one leg showed, after an acute period of eccentric exercise in both legs, a greater reduction in load bearing in the trained muscles. Interestingly, magnetic resonance imaging was reported to show the same intensity of damage in both legs but in the concentrically trained muscles the damage was more widespread.

To conclude, our results support the hypothesis that a concentric exercise training regime produces changes in a muscle which will make it more prone to the damage from eccentric exercise. This conclusion has implications for all regular exercise enthusiasts, particularly elite athletes such as triathletes and marathon runners who are required to carry out activities which are likely to include both concentric and eccentric exercise. It may be beneficial for such individuals to review their training programmes, in the light of these ideas, and to try to achieve a programme which promotes enhancement of muscle performance without rendering the muscle more susceptible to the damage of eccentric exercise.

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