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Muscle function after exercise-induced muscle damage

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**MUSCLE FUNCTION AFTER EXERCISE-INDUCED MUSCLE
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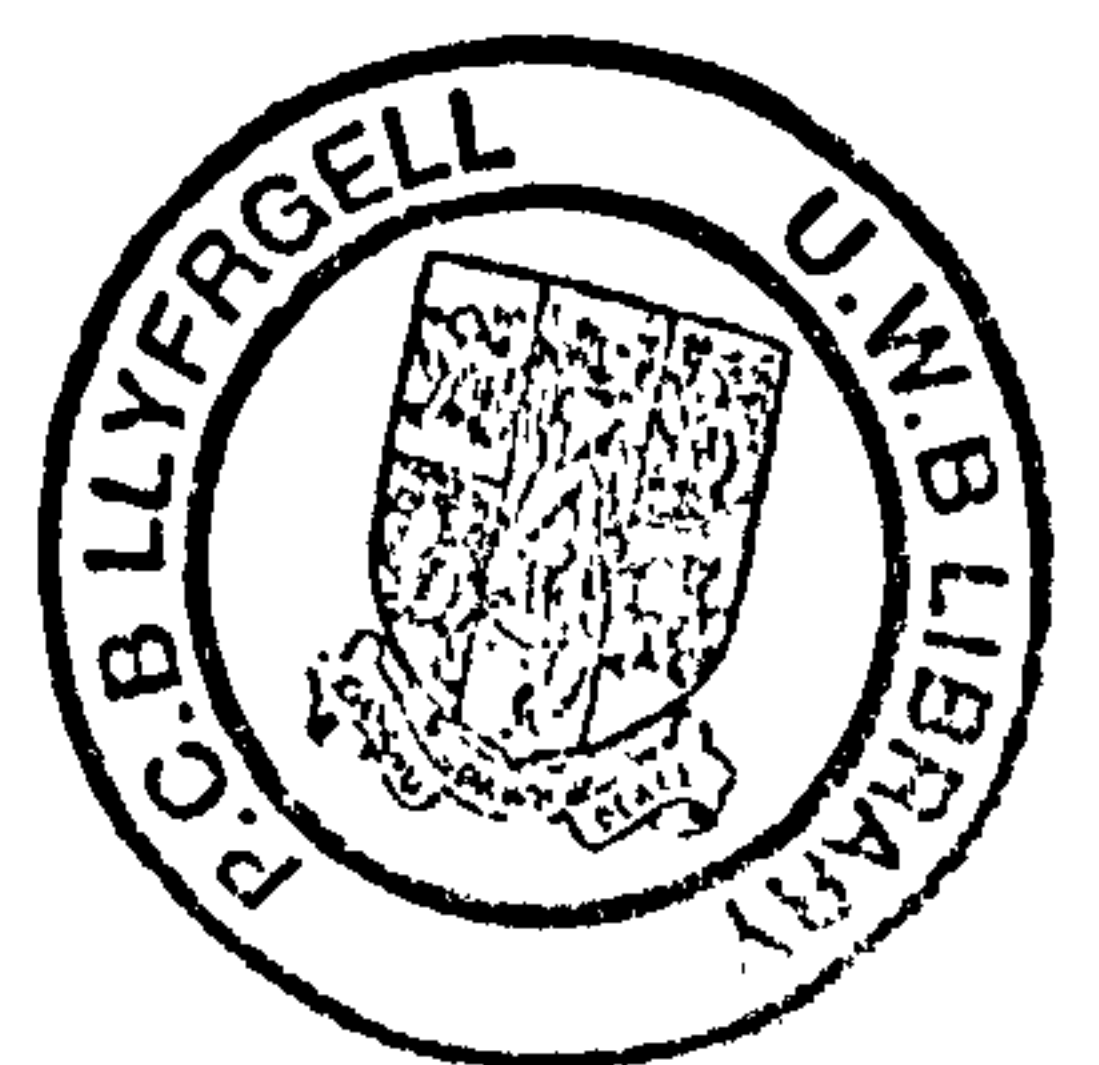
Christopher Byrne

Submitted for the degree of Doctor of Philosophy, May 2001

SCHOOL OF SPORT, HEALTH AND EXERCISE SCIENCES

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For my parents, Janet and Kevin, and my sister, Nicola

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Wisest is he who knows he does not know

SUMMARY

Muscle function after exercise-induced muscle damage has traditionally been evaluated by measures of isometric strength at a single joint angle or muscle length. The thesis investigates the effect of muscle damage on other muscle function parameters such as, isometric strength as a function of muscle length, concentric strength as a function of angular velocity, strength across muscle actions, the stretch-shortening cycle, power output, and fatigability.

Study 1

The first part of this study aimed to determine how the muscle length at which strength is measured affects reductions in isometric strength following eccentric exercise-induced muscle damage. The damaging exercise protocol consisted of 100 maximal voluntary eccentric actions of the knee extensors, performed in the prone position through a range of motion from 40° to 140° (0° = full extension) at an angular velocity of 90 deg·s⁻¹. Isometric strength of the knee extensors was measured at short muscle length (10° knee flexion) and optimal length (80°). A significantly greater relative loss of strength was observed at short versus optimal muscle length (76.3 ± 2.5% vs. 82.1 ± 2.7% of pre-exercise values, $P < 0.05$) over the seven day testing period following eccentric exercise. The second part of the study investigated isometric strength at optimal length and concentric strength at slow (30 deg·s⁻¹) and fast (180 deg·s⁻¹) angular velocities of movement. No differences were apparent in the magnitude and rate of recovery of strength across isometric (82.1 ± 2.7%) and slow (86.6 ± 2.0%) and fast (84.3 ± 1.5%) concentric muscle actions. Both the popping sarcomere hypothesis of

muscle damage and a failure in excitation-contraction coupling are possible explanations for the reduction in strength being affected by the muscle length at which it is measured. Both would be expected to affect strength to a greater extent at short versus optimal muscle lengths.

Study 2

The second study investigated knee extensor muscle strength during isometric, concentric and eccentric muscle actions and vertical jump performance under conditions of squat jump (SJ), countermovement jump (CMJ) and drop jump (DJ). These measures were taken before, 1 hour after, and on days 1, 2, 3, 4 and 7 following a damaging exercise protocol consisting of 100 barbell squats (10 sets x 10 reps @ 70% body mass load). Strength was significantly reduced for four days, however, no differences were observed in the magnitude or rate of recovery of isometric strength at 80° knee flexion and concentric and eccentric strength at 90 deg's⁻¹. Vertical jump performance was significantly reduced for three days and was dependent on the type of jump being performed. The relative decline in SJ performance was significantly greater than that in CMJ performance ($91.6 \pm 1.1\%$ vs. $95.2 \pm 1.3\%$ of pre-exercise values, $P < 0.05$) and the relative decline in SJ was significantly greater than that in DJ performance ($91.6 \pm 1.1\%$ vs. $95.2 \pm 1.4\%$, $P < 0.05$). No differences were observed in the relative decline in CMJ and DJ performance ($95.2 \pm 1.3\%$ vs. $95.2 \pm 1.4\%$, $P > 0.05$). The stretch-shortening cycle (SSC) of muscle function is utilised in CMJ and DJ but not in SJ. The SSC has a clear purpose: to allow the final phase (concentric action) to take place with greater force or power output, as compared to the condition where the movement is initiated by a concentric action alone.

Utilisation of the SSC in performance seems to attenuate the detrimental performance effects of exercise-induced muscle damage.

Study 3

The third and final study investigated the effects of exercise-induced muscle damage on maximal power output and knee extensor fatigability under isometric and dynamic conditions. Under isometric conditions, strength was assessed at 40° and 80° knee flexion and fatigability was assessed by a sustained 60s maximum voluntary contraction (MVC) at each joint angle. For dynamic conditions, maximum power output and fatigue were assessed during a maximal 30s cycle ergometer test. These measures were taken before, 1 hour after, and on days 1, 2, 3, and 7 following a damaging exercise protocol consisting of 100 eccentric squats (10 sets x 10 reps @ 80% concentric 1 RM). Isometric strength was significantly reduced ($P < 0.05$) for seven days but no significant differences were observed in the magnitude of strength loss and the pattern of recovery between the two joint angles.

Fatigability was quantified as the slope (b) of a linear regression line fitted to the torque and power decay during the 60s MVC and the 30s cycle test, respectively. Prior to muscle damage, subjects were significantly less fatigable ($P < 0.05$) at 40° ($b = -2.39 \pm 0.26$) versus 80° ($b = -5.50 \pm 0.72$). After muscle damage, subjects became significantly less fatigable at both 40° and 80° with recovery taking three days at 40° and seven days at 80°. Before damaging exercise, a greater rate of fatigue was observed under dynamic ($b = -12.75 \pm 2.3$) versus isometric (80°) conditions ($b = -5.50 \pm 0.72$). Isometric and dynamic fatigue

followed a similar temporal pattern after damaging exercise. When the effects of muscle damage on strength at 80° and maximal power output were compared, differences in the extent of performance loss and the time course of recovery were observed. At 1 hour post-exercise, strength was affected to a greater extent (30% reduction) than power (13% reduction) and whereas strength followed a linear recovery pattern, power suffered further decrements at day 1 (18%) and day 2 (16%) before starting to recover. The results indicate that under conditions of voluntary activation muscle becomes weaker but less fatigable under isometric and dynamic conditions following exercise-induced muscle damage.

The lower starting torque / power output and the slower rate of decline in torque / power output observed in post-damage fatigue curves may be a phenomenon of selective type II fibre damage. Evidence suggests that type II fibres are selectively damaged during eccentric exercise and therefore post-damage fatigue curves may be missing their contribution to performance. The different recovery patterns observed for isometric and dynamic performance may indicate an inability to maintain central motor drive during complex dynamic tasks when damage is present.

CHAPTER 1

INTRODUCTION

- 1.1 Exercise-induced muscle damage**
- 1.2 Markers of muscle damage**
 - 1.2.1 Direct versus indirect markers**
 - 1.2.2 Ultrastructural changes**
 - 1.2.3 Strength loss**
 - 1.2.4 Range of motion**
 - 1.2.5 Delayed-onset muscle soreness (DOMS)**
 - 1.2.6 Muscle protein release**
 - 1.2.7 Summary of the markers of muscle damage**

1.1 EXERCISE-INDUCED MUSCLE DAMAGE

Unaccustomed exercise, high intensity exercise, or an increased training workload often results in exercise-induced muscle damage. This is especially the case when the exercise involves eccentric muscle actions. Eccentric muscle actions occur when the muscle is lengthened during activation, for example, when the elbow flexors lower a weight. Conversely, a concentric muscle action occurs when the muscle shortens during activation, for example, when the elbow flexors raise a weight. When the muscle is activated and there is no change in muscle length and hence no movement, an isometric muscle action occurs. Studies comparing the effects of isometric, concentric, and eccentric muscle actions have consistently shown that eccentric actions result in the greatest evidence of muscle damage (Komi and Viitasalo, 1977; Davies and White, 1981; Newham *et al.*, 1983a; 1983b; McCully and Faulkner, 1986; Jones *et al.*, 1989; Golden and Dudley, 1992; Warren *et al.*, 1993a; Gibala *et al.*, 1995; Hesselink *et al.*, 1996). Eccentric muscle actions occur frequently in everyday activities and sporting pursuits. They provide the braking force in the knee extensors when walking down stairs and elbow flexors when lowering objects. In sports, eccentric muscle actions are a common feature in weightlifting exercises, they provide the braking in racquet sports, gymnastics, skiing, and descents in mountaineering. They are also an essential part of the stretch-shortening cycle, a natural form of muscle function (Komi, 1984), commonly referred to as the '*wind up*', which is used in jumping and throwing, and also in running. Consequently, muscle damage is a common experience following heavy or unaccustomed muscular work in these activities.

1.2 MARKERS OF EXERCISE-INDUCED MUSCLE DAMAGE

1.2.1 Direct versus indirect markers

There are many symptoms or manifestations of exercise-induced muscle damage, not just damage to the muscle fibre structure. There is strength loss, muscle soreness, stiffness and swelling, and increases in blood levels of myofibre proteins. As such, a wide variety of criteria have been used to quantify muscle damage. Direct evidence of muscle damage is gained from histological analysis using light or electron microscopy of muscle biopsy samples taken from humans or samples ranging from whole muscle to single fibres taken from animals. Indirect markers of muscle damage include methods for evaluating muscle function, such as strength and range of motion, and also muscle soreness and blood levels of myofibre proteins.

1.2.2 Ultrastructural changes

Qualitative and quantitative evidence of muscle damage in humans has been gained by needle biopsy following eccentric exercise (Friden *et al.*, 1983; Newham *et al.*, 1983a; Jones *et al.*, 1986; Friden and Lieber, 1992; Gibala *et al.*, 1995; Hortobagyi *et al.*, 1998). Damage is not widespread throughout a muscle or even throughout a single muscle fibre. It is often only focal, affecting one or two adjacent myofibrils or only part of a single sarcomere, although more widespread areas are observed. Characteristic features of damaged muscle include Z-line streaming and broadening, A-band disruption, misalignment of the myofibrils, and sometimes complete disruption of the myofibrils. The extent of damage seems to progress considerably in the post-exercise period. Newham *et al.* (1983a) reported that immediately post exercise, 16% of sampled fibres had focal changes, 16% had extensive changes, and 8% had very extensive changes. Focal areas were classified as areas of disruption

affecting one or two adjacent myofibrils and one or two adjacent sarcomeres; extensive areas as, disruption affecting more than two adjacent sarcomeres and more than two adjacent myofibrils; and very extensive areas as, fibres which contained more than one extensive area. Damage progressed so that at 30 hours post exercise, extensive and very extensive areas of damage had increased to 23% and 28%, respectively. Similarly, Jones *et al.* (1986) reported that small focal areas of fibre damage became more extensive in the days after eccentric exercise and lead to muscle fibre destruction 6-10 days after exercise. Friden *et al.* (1983) also reported an increase in the extent of damage from 1 hour post exercise to 3 days post exercise.

More recent studies applying rapid fixation techniques to single muscle fibres while still active during an eccentric action (Brown and Hill, 1991; Talbot and Morgan, 1996) have confirmed the earlier observation of Newham *et al.* (1983a) that actin and myosin filaments appear pulled apart in damaged sarcomeres. When muscle fibres were stretched beyond their optimum length (i.e. on the descending limb of the length-tension curve) there were sarcomeres with myofilaments not overlapping in one or both halves of the sarcomere (Brown and Hill, 1991; Talbot and Morgan, 1996). It has also been observed that over-extended whole sarcomeres are surrounded by normal sarcomeres that are essentially shorter than expected for the overall length of the fibre (Brown and Hill, 1991), or in the case of over-extended half sarcomeres, the other half of the sarcomere had typically shortened (Talbot and Morgan, 1996; Jones *et al.*, 1997).

1.2.3 Strength loss

Measures of isometric strength have provided the primary means of determining muscle function after muscle damage in both human and animal studies (Warren *et al.*, 1999). Strength loss is greatest immediately after eccentric exercise and can be reduced by over 50% (Clarkson *et al.*, 1992). The recovery of strength is prolonged, taking hours (Davies and White, 1981; 1982; Newham *et al.*, 1983b), days (Friden *et al.*, 1983; Sargeant and Dolan, 1987; Golden and Dudley, 1992; Gibala *et al.*, 1995; Hortobagyi *et al.*, 1998), or even over a week or more (McCully and Faulkner, 1985; Newham *et al.*, 1987; Cleak and Eston, 1992; Howell *et al.*, 1993). In humans, strength typically follows a linear recovery pattern after the initial post-exercise decrement. The time course of strength loss and histological evidence of muscle damage is not the same. As has been documented, damage becomes worse in the days following exercise, during the time that strength is recovering. Lowe *et al.* (1995) reported that muscle strength was not affected further when damage progressed and protein degradation rates were elevated 24–72 hours after eccentric exercise. Hesselink *et al.* (1996) observed only a weak relationship between the percentage of histological damage and isometric strength following eccentric exercise, suggesting that mechanisms other than structural damage are involved in the loss of muscle strength.

1.2.4 Range of motion

Range of motion is another aspect of muscle function that is adversely affected by exercise-induced muscle damage. Two aspects are affected: i) the ability to fully shorten the muscle and ii) spontaneous muscle shortening. These have most often been studied in the elbow flexors because of the ease of measurement in this muscle

group (Howell *et al.*, 1985; Jones *et al.*, 1987; Clarkson *et al.*, 1992; Cleak and Eston, 1992). The ability to fully shorten the muscle has been assessed by measuring the flexed arm angle (Clarkson *et al.*, 1992). The angle increases, with the greatest increase occurring immediately post exercise, followed by a gradual linear recovery. The pattern and time course of recovery is similar to that of strength loss and recovery, suggesting that strength loss may be responsible for the inability to fully shorten the muscle. The observation that some sarcomeres adopt a shortened position after eccentric exercise (Brown and Hill, 1991; Talbot and Morgan, 1996; Jones *et al.*, 1997) would reduce their ability to produce force at short muscle lengths and could account for the inability to fully shorten the muscle.

Spontaneous muscle shortening is the most common measure of impaired range of motion after eccentric exercise. In the elbow flexors it is assessed by measuring the resting arm angle, which decreases immediately and has been shown to reach its lowest value 1 to 2 days later (Jones *et al.*, 1987), 3 days later (Clarkson *et al.*, 1992) and 4 days later (Cleak and Eston, 1992). The shortening is not due to a normal muscle contraction since it is electrically (EMG) silent (Howell *et al.*, 1985; Jones *et al.*, 1987). The absence of electrical activity and the observation that muscle shortening shares a similar time course to muscle stiffness led Jones *et al.* (1987) to suggest that shortening of non-contractile elements acting in parallel with the muscle fibres, such as muscle connective tissue, could be responsible for the muscle shortening. Shortening of non-contractile connective tissue in series with the muscle fibres, such as tendinous attachments, has also been suggested to occur (Jones *et al.*, 1989).

1.2.5 Delayed-onset muscle soreness (DOMS)

Muscle soreness is the most commonly used marker of exercise-induced muscle damage in human studies (Warren *et al.*, 1999). Objective measures of soreness have been gained by using a myometer to measure the applied force to a muscle group at the pain threshold (Newham *et al.*, 1983b; Jones *et al.*, 1987; Newham *et al.*, 1988; Jones *et al.*, 1989; Cleak and Eston, 1992; Eston *et al.*, 1996; Semark *et al.*, 1999) and subjective measures of soreness have been gained by numerical scales, questionnaires, and visual analogue scales (Clarkson *et al.*, 1992; Howell *et al.*, 1993; Mair *et al.*, 1992; MacIntyre *et al.*, 1996; Child *et al.*, 1998; Hortobagyi *et al.*, 1998). Both forms of measurement have demonstrated that muscle soreness following eccentric exercise has a characteristic time course. Exercised muscles are pain free for approximately 8 hours and then soreness increases and peaks over the next one or two days (Newham *et al.*, 1983b; Jones *et al.*, 1987; Newham, 1988; Newham *et al.*, 1988; Jones *et al.*, 1989; Clarkson *et al.*, 1992; Cleak and Eston, 1992; Howell *et al.*, 1993; Mair *et al.*, 1994; Eston *et al.*, 1996; Child *et al.*, 1998; Hortobagyi *et al.*, 1998; Semark *et al.*, 1999). Jones and Round (1990) observed that a person will go to bed with only minor discomfort but wake the next morning with severe, and in some cases, almost disabling pain first appreciated when trying to get out of bed. All discomfort usually subsides within four days (Newham, 1988). Thus, the term delayed-onset muscle soreness (DOMS) is appropriate in describing the typical time course of the sensation, but Jones *et al.* (1987) stated that the term 'soreness' conveys little about the nature of the sensation. The sensation of soreness comprises muscle tenderness, pain on palpation, and also mechanical stiffness in the muscle that results in pain when it is stretched.

Delayed-onset muscle soreness shares a poor temporal relationship with histological evidence of muscle damage and measures of muscle function. Function is impaired before soreness arises and damage becomes worse when soreness has dissipated.

Several authors have suggested that DOMS originates from damage to non-contractile connective tissue (Asmussen, 1956; Jones *et al.*, 1987; Jones *et al.*, 1989).

The spontaneous muscle shortening immediately post-exercise suggests that connective tissue is damaged during eccentric exercise. Jones *et al.* (1989) suggested that damage occurring to connective tissue such as tendinous attachments during exercise, may give rise to an inflammatory reaction, oedema, distension and stiffening of the connective tissue matrix. Secondary responses such as these are suggested as causative of DOMS due to an increase in intramuscular pressure and inflammation of the connective tissue, which serves to sensitise pain receptors and give rise to painful sensations, especially when the muscle is palpated or stretched (Howell *et al.*, 1985; Jones *et al.*, 1987; Jones *et al.*, 1989; Smith, 1991).

1.2.6 Muscle protein release

Following muscle damage there is a release of muscle proteins into the blood. The most commonly measured protein is creatine kinase (CK) but lactate dehydrogenase, myoglobin, myosin heavy chain, and 3-methylhistidine have all been observed to be elevated following damaging exercise (Paul *et al.*, 1989; Mair *et al.*, 1992; 1994).

Following high force eccentric exercise there is typically a delay of about 2 days before any change in CK activity is observed, and then CK activity reaches its peak 4–7 days after exercise (Friden *et al.*, 1983; Jones *et al.*, 1986; Newham *et al.*, 1987; Clarkson *et al.*, 1992; Mair *et al.*, 1992; Saxton and Donnelly, 1996; Child *et al.*, 1998). Following downhill running (Byrnes *et al.*, 1985; Balnave and Thompson,

1993; Eston *et al.*, 1996), isometric exercise (Clarkson *et al.*, 1987), and weightlifting exercise involving both concentric and eccentric actions (Paul *et al.*, 1989), the increase and peak in CK activity occurs much more rapidly, occurring in the first 24–48 hours. The reason why different exercise regimes produce different temporal patterns of CK release is unknown, although CK activity reflects not only its release into the blood but also its clearance from the blood (Clarkson *et al.*, 1992). CK activity provides an indirect measure of muscle damage but does not correlate with histological evidence of damage, measures of muscle function, nor muscle soreness.

1.2.7 Summary of the markers of muscle damage

Due to the many symptoms of exercise-induced muscle damage, the phenomenon has been quantified in many different ways. The various markers of damage have been demonstrated to follow different temporal patterns, suggesting they are unrelated to each other. In their critical review of the measurement tools used in the study of eccentric exercise-induced muscle damage, Warren *et al.* (1999) stated that measures of muscle function provide the best means of evaluating the magnitude and time course of muscle damage resulting from eccentric actions. However, muscle function has mainly been assessed by measures of isometric strength. The functional performance of damaged muscle when tested in different ways shall be the focus of the thesis.

CHAPTER 2

REVIEW OF LITERATURE

- 2.1 Mechanical factors associated with damage**
 - 2.1.1 Force**
 - 2.1.2 Force/Activation**
 - 2.1.3 Length**
 - 2.1.4 Popping sarcomere hypothesis**
- 2.2 Neural factors associated with damage**
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 - 2.3.4 Isokinetic strength**
 - 2.3.5 Dynamic performance measures**
 - 2.3.6 Fatigability**
 - 2.3.7 Motor control**
 - 2.3.8 Research implications**

2.1 MECHANICAL FACTORS ASSOCIATED WITH DAMAGE

2.1.1 Force

The ability to generate high muscle force during an eccentric action has been implicated as a causative factor in muscle damage (Asmussen, 1956; Newham *et al.*, 1983a; 1983b; Friden *et al.*, 1983; McCully and Faulkner, 1985; Sargeant and Dolan, 1987; Armstrong *et al.*, 1991; Warren *et al.*, 1993b). The classical force-velocity relationship indicates that during an eccentric action the force can reach a value of about 1.8 times the isometric force (Jones and Round, 1990). Although the torque-velocity relationship deviates from the classical force-velocity relationship, well-motivated subjects can achieve greater torques during an eccentric action compared to either an isometric or concentric action (Tesch *et al.*, 1990; Westing *et al.*, 1991; Kellis and Baltzopoulos, 1998). This places a greater mechanical stress on the muscle fibres during eccentric actions and this may account for the greater evidence of damage observed after these muscle actions. The mechanism of force generation during an eccentric action differs from isometric and concentric actions. During lengthening, the cross-bridges are detached mechanically rather than undergoing a detachment that involves ATP splitting, as with shortening. The compliant portion of individual cross-bridges is also stretched further during an eccentric versus an isometric action (Jones and Round, 1990). Enoka (1996) stated that this loading profile undoubtedly places high stresses and strains on the involved structures and may contribute to the tissue damage that occurs with eccentric actions.

2.1.2 Force/Activation

The amplitude of the surface electromyogram (EMG) is often used to assess the magnitude of motor unit activation during voluntary actions. EMG amplitude during

a maximum voluntary eccentric action is often much less than during a maximum voluntary isometric or concentric action (Tesch *et al.*, 1990; Westing *et al.*, 1991; Kellis and Baltzopoulos, 1998). Furthermore, it is well documented that for a given force production, less motor unit activation is required for eccentric versus concentric actions (Bigland and Lippold, 1954; Komi *et al.*, 1987; Tesch *et al.*, 1990; Westing *et al.*, 1991; Kellis and Baltzopoulos, 1998). That is, the force/EMG ratio is greater under eccentric conditions, suggesting that during eccentric actions the force is being generated by a lower number of active motor units. This implies that the mechanical stress per active fibre will be greater under eccentric versus isometric or concentric conditions.

2.1.3 Length

Force may not be the sole mechanical factor responsible for the damage observed after eccentric actions, there also seems to be an important length-dependent factor. McCully and Faulkner (1986) using an *in situ* muscle preparation reported that damage resulted from eccentric actions eliciting 85% of maximum isometric force (P_0) but not from isometric or concentric actions eliciting 85% P_0 . The authors concluded that a characteristic of eccentric actions other than peak force appears to be implicated in the damaging process. Newham *et al.* (1988) reported greater evidence of muscle damage after eccentric exercise performed at long muscle lengths than at short lengths. Isometric actions performed at long muscle length have also been shown to result in greater changes in the markers of damage compared to isometric actions at short length (Jones *et al.*, 1989). This was despite isometric actions at long length generating 70-80% of the force observed at short length. In the same study, concentric actions, which produced greater forces than isometric actions,

resulted in no evidence of damage. Therefore, Jones *et al.* (1989) observed a non-linear relationship between force and evidence of damage, indicating an important length-dependent component to muscle damage. It is apparent that high force generation and activity at long muscle length are important determinants in inducing muscle damage. Eccentric actions combine these two factors. The lengthening process itself has been ruled out as a cause of damage, since no changes in the markers of muscle damage were observed after 80 passive lengthening actions of the elbow flexors (Jones *et al.*, 1989).

2.1.4 Popping sarcomere hypothesis

Jones *et al.* (1989) stated that the length-dependent component to muscle damage implies a change in some structure that is particularly under stress when the muscle is held at long sarcomere lengths, such as a structure in series with the contractile elements. Jones *et al.* suggested that in a muscle held at long length, due to the variation in sarcomere spacing along the length of a muscle fibre, some sarcomeres would be on the plateau of their length-tension curve whereas others will be on the descending limb. Sarcomeres on the descending limb will be weaker than their counterparts on the plateau and as such, an unstable state could result in the weaker sarcomeres being extended during an eccentric action to a length at which structural damage occurs. Morgan (1990) proposed a very similar hypothesis, which has become known as the ‘popping sarcomere hypothesis’. The hypothesis states that:

- 1) Rapid lengthening of muscle at long length does not involve uniform lengthening of sarcomeres, but consists primarily of instantaneous, uncontrolled extension of individual half-sarcomeres in each myofibril, one at a time, in order from the weakest towards the strongest (‘popping’). Such a

popped sarcomere will be extended beyond filament overlap, where its tension is borne by passive components.

- 2) Some such sarcomeres do not fully return to the normal inter-digitating pattern on relaxation and remain over-extended.
- 3) Repeated active lengthening would quickly stretch those sarcomeres, and so place extra tension on neighbouring sarcomeres causing them to be more likely to pop during a subsequent eccentric action and become disrupted.
- 4) This would lead to a growing region of disrupted sarcomeres, which could eventually lead to tearing of membranes (sarcolemma, transverse tubules, sarcoplasmic reticulum), causing a loss of calcium homeostasis in the fibre and eventual fibre de-generation.

Based on Morgan (1990) and Morgan and Allen (1999).

Additionally, the hypothesis has been extended to suggest that as a result of weak sarcomeres becoming over-extended, the remaining functional sarcomeres will adopt a shorter length to compensate (Wood *et al.*, 1993; Jones, 1996; Jones *et al.*, 1997; Whitehead *et al.*, 1998; Morgan and Allen, 1999). When muscle fibres were stretched beyond their optimum length (i.e. on the descending limb of the length-tension curve), over-extended whole sarcomeres were shown to be surrounded by normal sarcomeres that were essentially shorter than expected for the overall length of the fibre (Brown and Hill, 1991), or in the case of over-extended half sarcomeres, the other half of the sarcomere had typically shortened (Talbot and Morgan, 1996; Jones *et al.*, 1997).

2.2 NEURAL FACTORS ASSOCIATED WITH DAMAGE

2.2.1 Selective type II motor unit recruitment

As already stated, surface EMG amplitude is lower for maximal eccentric actions and a lower level of activation is required to achieve a given force (Bigland and Lippold, 1954; Komi *et al.*, 1987; Tesch *et al.*, 1990; Westing *et al.*, 1991; Kellis and Baltzopoulos, 1998). Enoka (1996) stated that this reduced activation might take the form of a lower level of activation distributed across the entire population of motor units or the activation of only a subset of the entire population. Several studies have indicated preferential recruitment of type II motor units during sub-maximal eccentric actions (Nardone and Schieppati, 1988; Nardone *et al.*, 1989; Howell *et al.*, 1995; McHugh *et al.*, 2000). However, these results have not been confirmed for either sub-maximal eccentric actions of the elbow flexors (Moritani *et al.*, 1988; Potvin, 1997) or maximal eccentric actions of the knee extensors (Tesch *et al.*, 1990). As such, the possibility of selective recruitment of type II motor units remains uncertain.

2.2.2 Selective type II fibre damage

Although there is uncertainty regarding selective type II fibre recruitment during eccentric exercise, it appears that type II fibres are more susceptible to damage during eccentric exercise (Friden *et al.*, 1983; Jones *et al.*, 1986; Lieber and Friden, 1988; Friden and Lieber, 1992; MacPherson *et al.*, 1996). Friden *et al.* (1983) reported three times more evidence of damage in type II versus type I muscle fibres of the knee extensors, and two-and-a-half times more damage in type IIb versus type IIa fibres. Similar structural changes were observed in rabbit tibialis anterior muscle after an extensive bout of eccentric actions (Lieber and Friden, 1988). These authors

reported that 80% of the abnormalities were found in fibres with narrow Z-lines and only 3 M-lines visible, which corresponds to type IIb fibres. Jones *et al.* (1986) stated that it remains to be seen whether the preferential damage to type II fibres is due to different patterns of use (selective recruitment) during eccentric exercise or a greater intrinsic susceptibility to damage of the type II fibres. Friden and Lieber (1992) suggested the latter, stating that the broader Z-lines in type I fibres may make them less vulnerable to repetitive, high-tension stress and subsequent damage than type II fibres.

2.3 MUSCLE FUNCTION AFTER DAMAGE

Warren *et al.* (1999) gave an operational definition of muscle function as, the ability to exert force under a given set of conditions, that is, over a given range of motion or at a fixed muscle length, at a given velocity or at a given external load, at a given level of activation and over a given number of contractions. The authors state that from a functional standpoint, muscle damage can be defined as a prolonged (i.e. lasting days or weeks) impairment of the ability of a muscle to produce force.

However, most of our knowledge relating to muscle function after muscle damage has been gained from measures of force under isometric conditions at a fixed muscle length. It is not clear how the impaired ability to produce force affects strength at different muscle lengths, strength during different types of muscle actions or at different angular velocities of movement, dynamic performance measures, or the fatigability of muscle. Factors such as selective type II fibre recruitment; selective type II fibre damage; sarcomere shortening; and impaired excitation-contraction coupling, have implications for muscle function to be affected in different ways, depending on how it is assessed. What follows is a review of research investigating

muscle function after exercise-induced muscle damage. Gaps in our knowledge of muscle function after damage and implications for research will be highlighted.

2.3.1 Isometric strength

In human studies, isometric strength has been quantified by measures of maximal voluntary contraction (MVC) torque at a single joint angle (Komi and Viitasalo, 1977; Davies and White, 1981; 1982; Newham *et al.*, 1983b; Newham *et al.*, 1987; Jones *et al.*, 1989; Clarkson *et al.*, 1992; Cleak and Eston, 1992; Balnave and Thompson, 1993). In animal studies, maximal isometric tetanic force or torque (P_0) is the equivalent of MVC torque. P_0 is measured *in situ* (i.e. the muscle retains normal blood supply but the nerve and/or distal tendon are exposed), *in vitro* (i.e. the muscle is completely isolated from the animal), or *in vivo* (i.e. the muscle-joint system is still intact) (Warren *et al.*, 1999). As with MVC, P_0 is reduced immediately after eccentric exercise and recovery is prolonged (McCully and Faulkner, 1985; 1986; Warren *et al.*, 1993a; Lowe *et al.*, 1995; Hesselink *et al.*, 1996; Ingalls *et al.*, 1998).

A problem with the use of MVC is that torque output is affected by subject motivation. In the study of muscle damage, muscle soreness could inhibit the performance of an MVC and result in a sub-maximal effort, leading to erroneous conclusions regarding the functional status of damaged muscle. In an attempt to overcome this, an experimental technique has been used to assess the magnitude of voluntary activation during an MVC by delivering single electrical impulses to the muscle. This technique is known as percutaneous twitch superimposition or twitch interpolation (Rutherford *et al.*, 1986). Sensitive force measurements allow for the

determination of any additional force increment in response to the electrical impulse and a measure of voluntary activation (VA) can be gained by a simple ratio:

$$\text{VA (\%)} = (\text{MVC} / \text{MVC} + \text{additional force}) \times 100$$

Adapted from Kent-Braun (1999).

As can be seen, any additional force produced by the superimposed electrical impulse will result in less than full (100%) voluntary activation. Evidence from studies employing this technique have reported that well motivated subjects can achieve full voluntary activation in the fresh state (Belanger and McComas, 1981; Gandevia and McKenzie, 1988) and in the damaged state (Rutherford *et al.*, 1986; Newham *et al.*, 1987; Saxton and Donnelly, 1996). When subjects were unable to achieve full voluntary activation in the fresh state, the level of voluntary activation did not change in the days following muscle-damaging exercise (Gibala *et al.*, 1995). These results suggest that the immediate and prolonged loss of isometric strength observed after muscle damage is caused by factors within the muscle (i.e. peripheral mechanisms) rather than factors proximal to the neuromuscular junction (i.e. central mechanisms). This is supported by the close association between the changes in MVC and electrically elicited tetanic torque (Davies and White, 1981). Davies and White (1981) reported a high correlation ($r = 0.85$) between high frequency (50 Hz) torque and MVC following damaging exercise. A simple ratio can be used to highlight any central contribution to strength loss (MVC torque / tetanic torque). A reduction in this ratio would indicate an increased contribution of central mechanisms to strength loss, for example a reduction in voluntary effort.

Animal studies have allowed the peripheral mechanisms responsible for the immediate and prolonged loss of strength to be determined. Damage to contractile elements and/or series elements or alternatively, an inability to fully activate the contractile elements could explain the loss of strength. Comparing P_0 to the force elicited by caffeine exposure provides insight into the mechanisms responsible for the strength loss. Caffeine exposure acts at the level of the sarcoplasmic reticulum to cause calcium release and a contracture of the muscle. If a damaged muscle has a caffeine-elicited force that is high relative to P_0 , then a failure of activation will be indicated rather than an inability to produce force because of damage to the contractile machinery. An inability to activate the contractile elements could result from either impaired excitation-contraction (E-C) coupling and/or an inability to conduct action potentials. E-C coupling has been defined as the sequence of events that starts with the passage of the action potential along the plasmalemma and ends with the release of calcium from the sarcoplasmic reticulum (Ingalls *et al.*, 1998). Warren *et al.* (1993a) reported that caffeine elicited force was 118% of P_0 immediately after 20 eccentric actions of isolated mouse soleus muscle. Prior to the eccentric actions, caffeine elicited force was 65% of P_0 . This suggests a failure to activate the contractile machinery rather than damage was responsible for the 43% decrement in P_0 observed in the study. Furthermore, Warren *et al.* (1993a) reported that the ability of damaged muscle to conduct action potentials was not impaired, suggesting that impaired E-C coupling was the primary mechanism responsible for the force loss. Additionally, Ingalls *et al.* (1998) reported a disproportional reduction in P_0 (approximately 51%) compared with caffeine-induced force (approximately 11-21%) after 150 eccentric actions in mouse EDL muscle. Again, this suggests an inability to fully activate the contractile machinery rather than damage is the primary

mechanism responsible for the loss of force after muscle damage. Ingalls *et al.* (1998) estimated that at least 75% of the reduction in P_O was due to E-C coupling impairment immediately post-exercise, and although the contribution declined with recovery, the authors estimated that E-C coupling accounted for at least 57% of the reduction in P_O at 5 days post-exercise.

Morgan and Allen (1999) in their review of the mechanisms underlying force loss after eccentric exercise stated that failure at any stage in the chain of events leading to muscle contraction will lead to a reduction in force. They proposed that the possible causes of force loss can be divided into five categories:

- 1) Changes in the central nervous system, motor nerve, or neuromuscular junction.
- 2) Inexcitable muscle cells, presumably due to gross cellular damage.
- 3) Failure or reduction of calcium release.
- 4) Changes in the calcium sensitivity of the contractile machinery.
- 5) Disorganisation of the contractile machinery.

Morgan and Allen (1999) concluded that changes in E-C coupling (category 3) and changes in sarcomere structure (category 5) appear to be the main contributors to the impaired ability to produce force after eccentric exercise. The authors stated that both of these mechanisms would affect force loss in a way that is dependent on the muscle length at which force is measured.

2.3.2 Length-dependence of isometric strength loss

According to the popping sarcomere hypothesis of muscle damage (Morgan, 1990; Morgan and Allen, 1999), the length-tension relationship of muscle undergoes a shift

to the right, towards longer muscle lengths, following eccentric exercise. That is to say, a longer muscle length is required to achieve the same myofilament overlap after exercise compared to before exercise. Such an effect would cause the joint angle for optimum force production to occur at a longer muscle length for *in vivo* studies and the optimal muscle length (L_0) to occur at a longer length for *in vitro* studies. Shifts in the optimum joint angle and L_0 towards longer muscle lengths have been reported for the human ankle extensors (Jones *et al.*, 1997; Whitehead *et al.*, 1998) and toad sartorius muscle (Wood *et al.*, 1993; Talbot and Morgan, 1998), respectively. The functional consequence of a shift in the length-tension curve is that strength loss will be affected by the muscle length at which it is measured. A greater relative loss of strength at short length versus optimal or long muscle length would indirectly indicate a rightward shift in the length-tension curve. Evidence from studies employing this method have observed the greatest relative loss of strength to occur at short muscle length in the human elbow flexors (Saxton and Donnelly, 1996) and knee extensors (Child *et al.*, 1998).

The shift in the length-tension relationship is hypothesised to be the result of over-extended sarcomeres causing the remaining functional sarcomeres to adopt a shortened position (Jones *et al.*, 1997). According to the popping sarcomere hypothesis, when the over-extended sarcomeres re-interdigitate, a corresponding reversal of the shift in the length-tension relationship, a recovery of optimal joint angle or L_0 , and a proportional loss of strength at short, optimal, and long muscle lengths should occur. Talbot and Morgan (1998) reported a shift in L_0 of toad sartorius muscle that reversed within 3 to 5 hours and immediate shifts in optimal angle of the ankle extensors, with reversals after two days, have been reported (Jones

et al., 1997; Whitehead *et al.*, 1998). When the indirect method of determining a shift in the length-tension relationship is employed, it is not clear if the greater loss of relative strength is an acute effect only (Child *et al.*, 1998) or persists for several days (Saxton and Donnelly, 1996).

2.3.3 Contractile properties

The force frequency relationship (FFR) is a convenient way of characterising the contractile properties of muscle (Jones, 1996). The FFR is a plot of the force that is produced (y-axis) when a muscle is stimulated with a range of frequencies (x-axis), usually 1 to 100 Hz. Force rises steeply as the stimulation rate is increased from 1 to 50 Hz and then reaches a plateau from 50 to 100 Hz (Edwards *et al.*, 1977a).

Edwards *et al.* (1977b) first reported that the FFR demonstrates a rightward shift after fatiguing exercise, due to relatively lower forces being generated at low frequencies of stimulation. Jones (1996) reported that the main features of this type of fatigue, termed low frequency fatigue (LFF), are as follows:

- 1) The forces at low frequencies of stimulation are the most severely affected;
- 2) Recovery is slow, taking hours or, in severe cases, days for a full recovery;
- 3) The effect persists in the absence of gross metabolic or electrical disturbance to the muscle.

Low frequency fatigue is most pronounced after eccentric versus isometric or concentric exercise (Davies and White, 1981; 1982; Newham *et al.*, 1983b; Jones *et al.*, 1989) and can take over a week to recover (Newham *et al.*, 1987; Jones *et al.*, 1989). The ratio of force generated at 20 compared to 50 or 100 Hz is often used as a measure of LFF. Edwards *et al.* (1977b) suggested that LFF is caused by impairment of E-C coupling so that less calcium is released per action potential. Due to the

sigmoidal relationship between force and intracellular calcium (Allen *et al.*, 1992), a reduction in calcium on the right side of the relationship will have little effect on force, whereas on the steep left portion of the curve a small reduction will have a large effect on force. Jones (1981) suggested that LFF could be a consequence of damage to the structure of the muscle fibre and the E-C coupling mechanism, possibly the sarcoplasmic reticulum. An alternative or adjunctive explanation is that shortened sarcomeres cause the FFR to shift to the right, since the FFR is shifted to the right at short muscle lengths (Edwards *et al.*, 1989a; Sacco *et al.*, 1994). Jones (1996) suggested that two processes may be occurring in damaged muscle that lead to LFF, one being a reduction in calcium release and the other, a redistribution of sarcomere lengths.

2.3.4 Isokinetic strength

In contrast to isometric strength, much less is known about dynamic strength following exercise-induced muscle damage. Clarkson and Newham (1995) in their review of muscle damage research stated that the majority of studies have examined the effect of eccentric exercise on isometric force generation and that it is assumed these changes are comparable to those in eccentric and concentric force, but there is no direct evidence that this is so. Of the studies that have investigated isometric, concentric, and eccentric strength, Golden and Dudley (1992) observed a faster recovery of eccentric and isometric strength versus concentric strength, whereas, Hortobagyi *et al.* (1998) observed no differences in the magnitude or rate of recovery of strength across muscle actions.

Golden and Dudley (1992) also observed that concentric strength at $180 \text{ deg}\cdot\text{s}^{-1}$ was slower to recover than concentric strength at $60 \text{ deg}\cdot\text{s}^{-1}$. This velocity-dependent effect of strength loss has been observed by other authors (Friden *et al.*, 1983; Eston *et al.*, 1996). Friden *et al.* (1983) investigated the effects of 30 minutes eccentric cycle ergometer exercise on knee extensor isometric strength and isokinetic concentric strength at $90 \text{ deg}\cdot\text{s}^{-1}$, $180 \text{ deg}\cdot\text{s}^{-1}$, and $300 \text{ deg}\cdot\text{s}^{-1}$. Measurements were recorded immediately post-exercise, at day 3 and day 6 post-exercise. No comparisons were made between isometric strength and the different angular velocities or between the different angular velocities themselves. Friden *et al.* reported that isometric strength and concentric strength at $90 \text{ deg}\cdot\text{s}^{-1}$ and $180 \text{ deg}\cdot\text{s}^{-1}$ were not significantly different from their baseline measurements at day 6, but strength at $300 \text{ deg}\cdot\text{s}^{-1}$ was significantly below its own baseline value at this time. The results were from 9 subjects immediately post-exercise, 7 subjects at day 3 post, and 4 subjects at day 6 post. Repeated paired sample t-tests were used to determine if strength measurements were significantly different from baseline. The 12 t-tests performed by Friden *et al.* increased the probability of making a Type 1 error (rejecting a true null hypothesis) from 0.05 to 0.46 (Huck, 2000). Although the results of Friden *et al.* need to be treated with caution, the reports of selective type II fibre recruitment and/or selective type II fibre damage during eccentric exercise provide reason to suggest that strength at high angular velocities of movement may be affected to a greater extent than either strength at low velocities of movement or isometric strength. It is quite apparent that the torque-angular velocity relationship is an area of research that requires further attention.

2.3.5 Dynamic performance measures

Clarkson and Newham (1995) stated that in functional terms, the ability to generate power and do work is more important than isometric force generation, but this has been studied infrequently. Indeed, only one study, with a very small sample size ($n=4$), has examined the ability of damaged muscle to generate power. Following a bout of downhill walking, Sargeant and Dolan (1987) reported an immediate, significant, and sustained reduction in short-term (20s) power output, measured concentrically on an isokinetic cycle ergometer. Reductions persisted for four days and were accompanied by reductions in isometric strength of a similar magnitude. Clarkson and Newham (1995) stated that this area warrants further investigation.

Other dynamic performance measures such as, vertical jumping and sprinting have also been studied infrequently. Only one study has investigated the effect of muscle damage on sprinting performance. Semark *et al.* (1999) observed no difference in either sprint time or acceleration over 30 metres following 70 drop jumps. It is likely that the exercise regime was not intense enough to induce damage and functional changes in the trained subjects that participated in the study. This is certainly an area that requires further investigation since sprinting performance is an important component of many sports. For vertical jump performance, Mair *et al.* (1995) reported a median decrease of 6.3 cm in one-legged vertical jumping performance immediately after eccentric exercise of the knee extensors, which had recovered four days later. Following exhaustive stretch shortening cycle (SSC) exercise of the knee extensors, Horita *et al.* (1999) reported that drop jump performance was reduced for up to two days. Vertical jumping is a convenient model to study muscle function with and without use of the SSC. The SSC is utilised in countermovement and drop

jump performance but not in squat jump performance. Chambers *et al.* (1998) reported that following a 90 km foot race, a mode of exercise known to induce muscle damage (Sherman *et al.*, 1984), squat jump performance remained significantly lower for longer (18 days) compared to countermovement jump (11 days) and drop jump (3 days) performance. Again, this is an area of dynamic muscle function that has been studied infrequently and requires further attention.

2.3.6 Fatigability

Only two previous studies have examined the susceptibility of damaged muscle to fatigue (Davies and White, 1981; Balnave and Thompson, 1993). Both of these studies used an isometric electrically elicited fatigue test consisting of trains of stimuli at 20 Hz, lasting 300 ms and repeated every second for two minutes. Davies and White (1981) reported that damaged muscle was weaker but no more fatigable, whereas Balnave and Thompson (1993) reported that damaged muscle was weaker but less fatigable. The fatigability of damaged muscle using voluntary isometric actions has not been studied. Assessing the fatigability of damaged muscle using dynamic muscle actions provides further scope for research.

2.3.7 Motor control

Only a handful of studies have investigated motor control after muscle damage (Saxton *et al.*, 1995; Brockett *et al.*, 1997; Pearce *et al.*, 1998), all of which used the elbow flexors. Brockett *et al.* reported that subjects consistently placed the damaged elbow flexors in a more extended position for over four days, when trying to match the joint angle of the non-damaged arm. This indicates that subjects thought the muscle was shorter than it actually was. Both Saxton *et al.* and Brockett *et al.*

reported an impairment of force sense. Brockett *et al.* reporting that subjects consistently undershot the target force with the damaged arm. Additionally, Pearce *et al.* reported a prolonged impairment in motor control, as assessed by tracking error during a one-dimensional elbow flexion/extension visuomotor pursuit task. Brockett *et al.* suggested that damage or disturbance to the muscle spindles and tendon organs are likely to be responsible for the impairment in motor control after eccentric exercise.

2.3.8 Research implications

Warren *et al.* (1999) conducted a major review of the measurement tools used in muscle damage research and concluded that in addition to the 'standard' isometric torque measurement, characterisation of the torque producing capability as a function of velocity, activation, muscle length, and bout duration should be pursued. From the preceding review of literature it is quite apparent that the majority of research assessing muscle function after exercise-induced muscle damage has focussed on maximal isometric strength measurement at a single joint angle. The thesis aims to advance knowledge of muscle function after exercise-induced muscle damage by assessing function in a way that is both physiologically valid and practically relevant.

The following specific questions were addressed:

- 1) Is the loss of isometric strength after eccentric exercise-induced muscle damage dependent on the muscle length at which it is measured? (Chapter 3)
- 2) Are isometric strength, slow concentric strength, and fast concentric strength affected to a similar extent after eccentric exercise-induced muscle damage? (Chapter 3)

- 3) Is strength loss following exercise-induced muscle damage dependent on the type of muscle action being performed (isometric, concentric, eccentric)?
(Chapter 4)
- 4) Is vertical jump performance following exercise-induced muscle damage dependent on the type of jump being performed (squat jump, countermovement jump, drop jump)? (Chapter 4)
- 5) Is muscle more susceptible to fatigue after eccentric exercise-induced muscle damage? (Chapter 5)
- 6) Does eccentric exercise-induced muscle damage affect the ability of muscle to generate power output to a similar extent as isometric strength? (Chapter 5)

CHAPTER 3

THE EFFECT OF ECCENTRIC EXERCISE-INDUCED MUSCLE DAMAGE ON ISOMETRIC KNEE EXTENSOR TORQUE AT SHORT AND OPTIMAL MUSCLE LENGTHS

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3.5.5 Summary

The contents of this chapter form the basis of a study to appear in the *Scandinavian Journal of Medicine and Science in Sports* (accepted August 2000). Characteristics of isometric and dynamic strength loss following eccentric exercise-induced muscle damage.

3.1 ABSTRACT

Angle-specific isometric strength and angular velocity-specific concentric strength of the knee extensors were studied in eight subjects (5 males and 3 females) following a bout of muscular damaging exercise. One hundred maximal voluntary eccentric actions of the knee extensors were performed in the prone position through a range of motion from 40° to 140° (0° = full extension) at 90 degs⁻¹. Isometric strength was measured whilst seated at 10° and 80° knee flexion, corresponding to short and optimal muscle length, respectively. Isokinetic concentric strength was measured at 30 and 180 degs⁻¹. Plasma creatine kinase (CK) activity was also measured from a fingertip blood sample. These measures were taken before, immediately after and on days 1, 2, 4, and 7 following the damaging exercise. The eccentric exercise protocol resulted in a greater overall loss of relative strength ($P < 0.05$) at short muscle length ($76.3 \pm 2.5\%$ of pre-exercise values) compared to optimal length ($82.1 \pm 2.7\%$). There were no differences in the relative strength loss between isometric strength at optimal length and isokinetic concentric strength at 30 and 180 degs⁻¹. Creatine kinase activity was significantly elevated above baseline at days 4 ($P < 0.01$) and 7 ($P < 0.01$). The greater relative loss of strength at short muscle length persisted throughout the seven-day testing period. The popping sarcomere hypothesis predicts that functional sarcomeres adopt a shortened state following eccentric exercise and this mechanism along with a failure in excitation-contraction coupling could explain the present results since they both affect strength in a way that is dependent on muscle length.

3.2 INTRODUCTION

3.2.1 Length-dependence of isometric strength loss

Strength loss following muscle damage has mainly been assessed by measuring isometric force at a single muscle length (Warren *et al.*, 1993; Ingalls *et al.*, 1998) or a single joint angle (Newham *et al.*, 1983; Jones *et al.*, 1989; Clarkson *et al.*, 1992; Cleak and Eston, 1992) before and after the damaging exercise. However, the popping sarcomere hypothesis of muscle damage suggests that the length-tension relationship of muscle undergoes a shift to the right, towards longer muscle lengths, following eccentric exercise (Morgan and Allen, 1999). That is to say, a longer muscle length is required to achieve the same myofilament overlap after exercise compared to before exercise. Such an effect would cause the joint angle for optimum force production to occur at a longer muscle length and would cause the force loss associated with eccentric exercise to be affected by the muscle length or joint angle at which it is measured. A shift in the length-tension curve towards longer muscle lengths would cause a significantly greater loss of relative force to occur at short muscle lengths compared to optimal muscle length, due to their respective locations on the length-tension curve. Short muscle lengths being located on the ascending limb and optimal lengths on the plateau of the curve. This is exemplified in Figure 3.1.

The first part of this study aimed to determine how reductions in isometric force following eccentric exercise are affected by the muscle length at which force is measured. According to the popping sarcomere hypothesis (Morgan, 1990), lengthening of active muscle does not occur by uniform lengthening of all

sarcomeres, but by a non-uniform distribution of sarcomere length change. With some sarcomeres rapidly over-extending ('popping') beyond filament overlap and becoming permanently over-extended. The hypothesis suggests that over-extended sarcomeres lead to the remaining functional sarcomeres adopting a shorter length to compensate. It is this mechanism which is suggested to cause the length-tension curve to shift towards longer muscle lengths. By measuring isometric force at short versus optimal muscle length before and after eccentric exercise we can indirectly determine whether the length-tension curve has shifted towards longer muscle lengths. A significantly greater loss of relative force at short versus optimal muscle length would indicate a rightward shift in the length-tension curve, towards longer muscle lengths. Evidence from studies employing this method have observed the greatest relative loss of force to occur at short muscle length following eccentric exercise of the human elbow flexors (Saxton and Donnelly, 1996) and knee-extensors (Child *et al.*, 1998). However, it is not clear if the greater force loss is an acute effect only (Child *et al.*, 1998) or persists for several days (Saxton and Donnelly, 1996) after eccentric exercise. This study aimed to determine the magnitude and time course of the effect over a seven-day period following eccentric exercise.

3.2.2 Isometric versus dynamic strength loss

The purpose of the second part of this study was to compare the effect of exercise-induced muscle damage on isometric and concentric strength at slow and fast angular velocities of movement. Warren *et al.* (1999) recently advocated the investigation of strength as a function of velocity in addition to the 'standard' measurement of

isometric strength. This followed evidence from several studies which have suggested that the recovery of strength may be dependent upon the type of muscle action (isometric vs. concentric vs. eccentric) and/or the angular velocity of movement (slow vs. fast) (Friden *et al.*, 1983; Golden and Dudley, 1992; Gibala *et al.*, 1995; Eston *et al.*, 1996). In addition, it has been suggested that type II muscle fibres are more susceptible to damage during eccentric exercise (Friden *et al.*, 1983; Jones *et al.*, 1986; Lieber and Friden, 1988; Friden and Lieber, 1992; MacPherson *et al.*, 1996) and type II motor units are perhaps selectively recruited for eccentric actions (Nardone and Schieppati, 1988; Nardone *et al.*, 1989; Howell *et al.*, 1995; Enoka, 1996; McHugh *et al.*, 2000). With type II fibres playing a significant role in determining muscle strength at high angular velocities of movement (Thorstensson *et al.*, 1976; Ivy *et al.*, 1981), the possibility exists that dynamic strength at high angular velocities may be affected to a greater extent by muscle damage than strength at low angular velocities or even isometric strength.

3.2.3 Research hypotheses

The following hypotheses applied to this investigation:

It is hypothesised that:

- a) The relative loss of isometric strength following eccentric exercise-induced muscle damage will be significantly greater at short versus optimal muscle length;
- b) The relative loss of concentric strength at a high angular velocity of movement will be significantly greater than either concentric strength at a low angular velocity or isometric strength.

3.3 METHODS

3.3.1 Participants and design

Eight healthy participants, five males and three females (age 21.4 ± 3.5 yr (mean \pm SD), ht 1.73 ± 0.13 m, mass 68.1 ± 5.0 kg) were involved in the study. All individuals were moderately active but had not participated in any resistance training for six months prior to the study, and none had any musculo-skeletal defects. Each individual gave written informed consent to participate in the study, which had previously been approved by the School ethics committee. The muscle group studied was the knee extensors. Using a single-group design, joint angle-specific isometric strength, angular velocity-specific concentric strength, and plasma creatine kinase (CK) activity were measured before and after a bout of maximal repetitive isokinetic eccentric exercise. Isokinetic eccentric exercise and all strength measurements were performed with the non-dominant limb using a Kin-Com (500H, Chattecx, Chattanooga, TN, USA) isokinetic dynamometer. Each subject was evaluated for each criterion measure prior to, immediately afterwards, and on days 1, 2, 4, and 7 following the eccentric exercise bout.

3.3.2 Positioning and procedure for muscle function measurement

Participants were tested in the seated position with the lateral femoral epicondyle aligned to the dynamometer's axis of rotation. The pelvis, chest, and active limb were secured with restraining straps to prevent extraneous movement. The pad of the lever arm was positioned at a distal point on the tibia near the malleoli. The dynamometer lever arm length and the vertical, horizontal, and seat positions were recorded for each individual in order to replicate the exact testing position from trial

to trial. All participants performed at least two familiarization sessions during which they were introduced to standardized written instructions to work as hard and fast as possible against the resistance of the dynamometer, the isometric and isokinetic testing protocols, and the use of visual feedback to enhance torque output from one repetition to the next (Baltzopoulos *et al.*, 1992).

3.3.3 Angle-torque relationship

Selection of two joint angles for isometric torque measurement, corresponding to short muscle length and optimal muscle length, were established from the angle-torque relationship of the non-dominant limb of the knee extensors of four participants by testing each person eight times. Full knee extension (0°) was entered as a reference value into the Kin-Com visual display. This was used to set angles for isometric torque measurement at 12° increments throughout a 96° range of motion. Two maximal voluntary contractions (MVCs) of three seconds duration were performed at each joint angle in a random order with a one-minute rest period between successive attempts. The highest peak torque elicited from the two attempts was used as the criterion score.

3.3.4 Isometric strength measurement

Participants performed isometric MVCs of the knee extensors at 10° and 80° knee flexion corresponding to short and optimal muscle length, respectively. The testing positions were obtained by entering full knee extension (0°) as a reference value into the Kin-Com visual display. The reproducibility of this method was checked on each testing occasion by noting the Kin-Com angle display when the lever arm was at true

90° (determined by spirit level). The pre-test angle display at true 90° was used as the criterion. If any difference existed, the process was repeated until the criterion was achieved. Three sub-maximal and one maximal practice repetitions acted as warm-up at each testing position. Three MVCs of three seconds duration were performed at each joint angle in a random order with a one-minute rest period between repetitions. The highest peak torque from the three contractions was used as the criterion score for short and optimal muscle length, respectively.

3.3.5 Isokinetic concentric strength measurement

Concentric peak torque of the knee extensors was measured at angular velocities of 30 and 180 degs⁻¹. The problem of torque overshoot or artefact becomes increasingly important when testing isokinetic strength at high angular velocities. We were unable to distinguish between muscular torque and torque overshoot at velocities above 180 degs⁻¹ and so this angular velocity was the highest measured. Range of motion for the dynamic contractions was from 90° to 10° knee flexion. Three sub-maximal and one maximal practice repetitions acted as warm-up for each velocity. The highest peak torque elicited from three MVCs was used as the criterion score for each angular velocity. A continuous protocol was employed with a passive return to the start angle following each MVC. One minute of rest was allowed between repetitions and each angular velocity. The order of testing between isometric and dynamic torque was randomised. However the slower angular velocity was always tested before the faster angular velocity during the dynamic protocol as this has been shown to enhance reproducibility (Wilhite *et al.*, 1992).

3.3.6 Creatine kinase measurement

Plasma creatine kinase (CK) activity was determined from a fingertip blood sample.

A warm fingertip was cleaned with a sterile alcohol swab and allowed to dry.

Capillary puncture was made with a softclix lancet and a sample of whole fresh blood (32µl) was pipetted from a capillary tube onto the test strip and analysed for CK activity via a colorometric assay procedure (Reflotron, Boehringer Mannheim, Lewes, UK). This system uses a plasma separation principle, which is incorporated in the reagent carrier on the test strip.

3.3.7 Isokinetic eccentric exercise protocol

Each subject performed a bout of 100 isokinetic eccentric MVCs at an angular velocity of 90 degs⁻¹ using the Kin-Com dynamometer. The eccentric actions were performed as 10 sets of 10 repetitions with 10 seconds rest between repetitions and one minute between sets. Participants exercised in the prone position through a range of motion from 40° to 140° (0° = full extension) of knee flexion. Each eccentric action was followed by a passive return to the start angle. The testing position and range of motion were selected to exercise the knee extensors at long muscle length. Eccentric exercise performed at long muscle lengths results in greater functional impairment and evidence of muscle damage than eccentric exercise performed at short muscle lengths (Newham *et al.*, 1988; Child *et al.*, 1998).

3.3.8 Statistical analysis

The strength data were analysed using a series of two-factor analyses of variance.

Isometric data was analysed by a two factor (6 x 2; Measurement Time x Angle)

fully repeated measures analysis of variance (RM ANOVA). Isometric and dynamic strength were compared using a two-factor (6 x 3; Measurement Time x Contraction Mode) RM ANOVA. Plasma CK activity was analysed using a single-factor RM ANOVA. The assumption of sphericity was tested by the Mauchly Test of Sphericity. Any violations of this assumption were corrected by using the Greenhouse-Geisser adjustment to raise the critical value of F, as indicated by (GG) . Statistical significance was set at the 0.05 alpha level. Post-hoc tests were conducted using paired sample t-tests. The Bonferonni technique was used to eliminate the problem of an inflated Type 1 error risk by adjusting alpha depending on the number of pairwise comparisons. For example, if 5 pairwise comparisons were made the adjusted alpha would be: $0.05 / 5 = 0.01$.

3.4 RESULTS

3.4.1 Angle-torque relationship

Peak isometric torque was generated in the region of 75-85° knee flexion. This is in agreement with previous results using the same muscle group (Newham *et al.*, 1991).

The two angles selected for torque measurement prior to, and following the eccentric exercise bout were 10° and 80°, corresponding to short and optimal muscle length, respectively. Their positions were on the ascending limb and the plateau of the angle-torque relationship, respectively. Isometric peak torque at 10° was approximately 30% of isometric peak torque at 80° (see Figure 3.1).

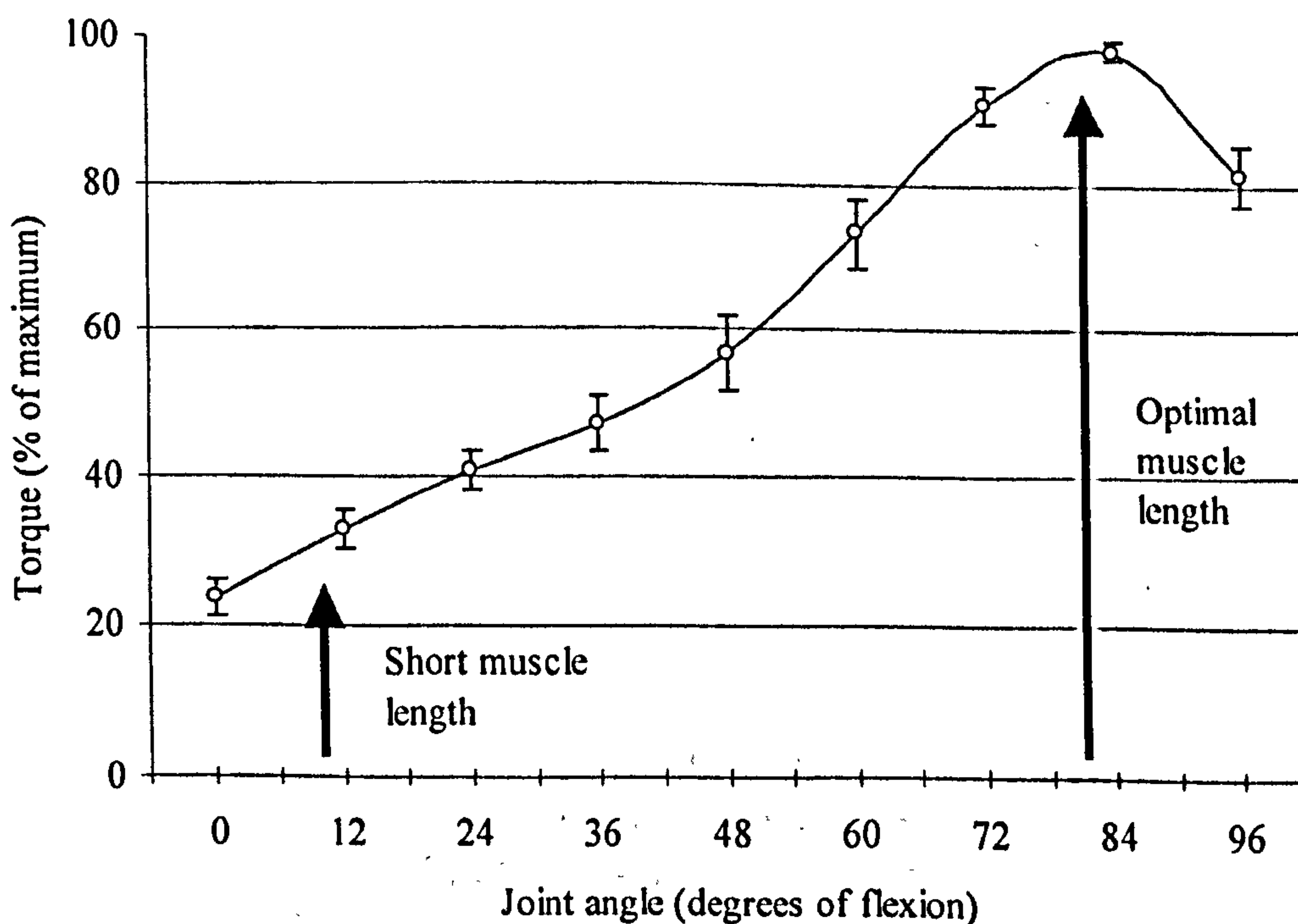


Figure 3.1: Angle-torque relationship of the knee extensors determined through isometric MVCs at each joint angle. Torque values are means (\pm S.E.M.) expressed as a percentage of maximum torque.

3.4.2 Isometric strength at short and optimal muscle length

Absolute values for isometric peak torque were 127 ± 32 Nm and 422 ± 54 Nm (mean \pm SEM) for short and optimal muscle lengths, respectively. Isometric strength changed significantly over time ($F_{5,35} = 27.2, P < 0.001$) and between angle ($F_{1,7} = 7.0, P < 0.05$). The interaction of time x angle on isometric peak torque approached significance ($F_{5,35} = 2.2, P = 0.07$).

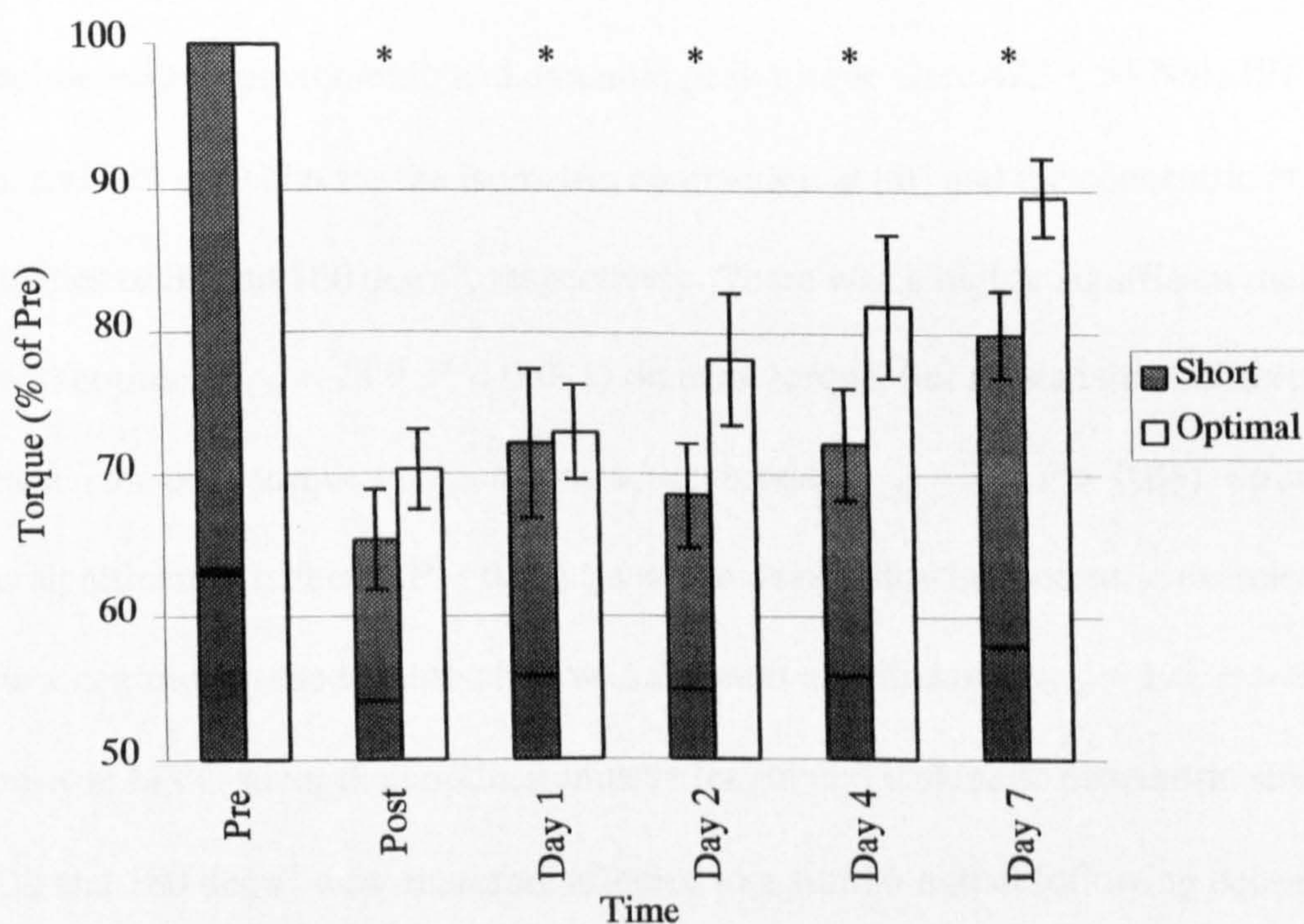


Figure 3.2: Changes in isometric strength at short and optimal muscle length across time following 100 eccentric MVCs of the knee extensors. Torque values are means (\pm S.E.M.) expressed as a percentage of the pre-eccentric exercise torque.

* Significantly different ($P < 0.05$) from pre-exercise.

Strength loss was immediate and prolonged with recovery incomplete ($P < 0.05$)

seven days after eccentric exercise. The main effect for angle indicated that the

overall relative decline in isometric strength was significantly greater at short versus

optimal muscle length. Isometric torque at short muscle length was reduced to an

overall mean of $76.3 \pm 2.5\%$ of pre-exercise values compared to $82.1 \pm 2.7\%$ at optimal muscle length. As there was no significant interaction of time x angle, we did not conduct post-hoc tests to determine differences between means at the various time points following eccentric exercise. However, the cell means demonstrate a clear trend showing that isometric torque at short muscle length was reduced to a greater extent throughout the 7 day testing period (see Figure 3.2 and Table 3.1).

3.4.3 Isometric and isokinetic concentric strength

Absolute values for isometric and dynamic peak torque were 422 ± 54 Nm, 397 ± 62 Nm, and 224 ± 49 Nm for the isometric contraction at 80° and the concentric angular velocities of 30 and 180 $\text{deg}\cdot\text{s}^{-1}$, respectively. There was a highly significant main effect for time ($F_{5,35} = 28.9, P < 0.001$) on peak torque, but no statistical difference between the peak torque for each contraction mode ($F_{2,14} = 2.1, P > 0.05$). Strength was significantly reduced ($P < 0.05$) for seven days following eccentric exercise. The time x contraction mode interaction was also non-significant ($F_{10,70} = 1.6, P > 0.05$). Isometric MVC strength at optimal muscle length and isokinetic concentric strength at 30 and 180 $\text{deg}\cdot\text{s}^{-1}$ were therefore affected to a similar extent following eccentric exercise-induced muscle damage (see Figure 3.3 and Table 3.1).

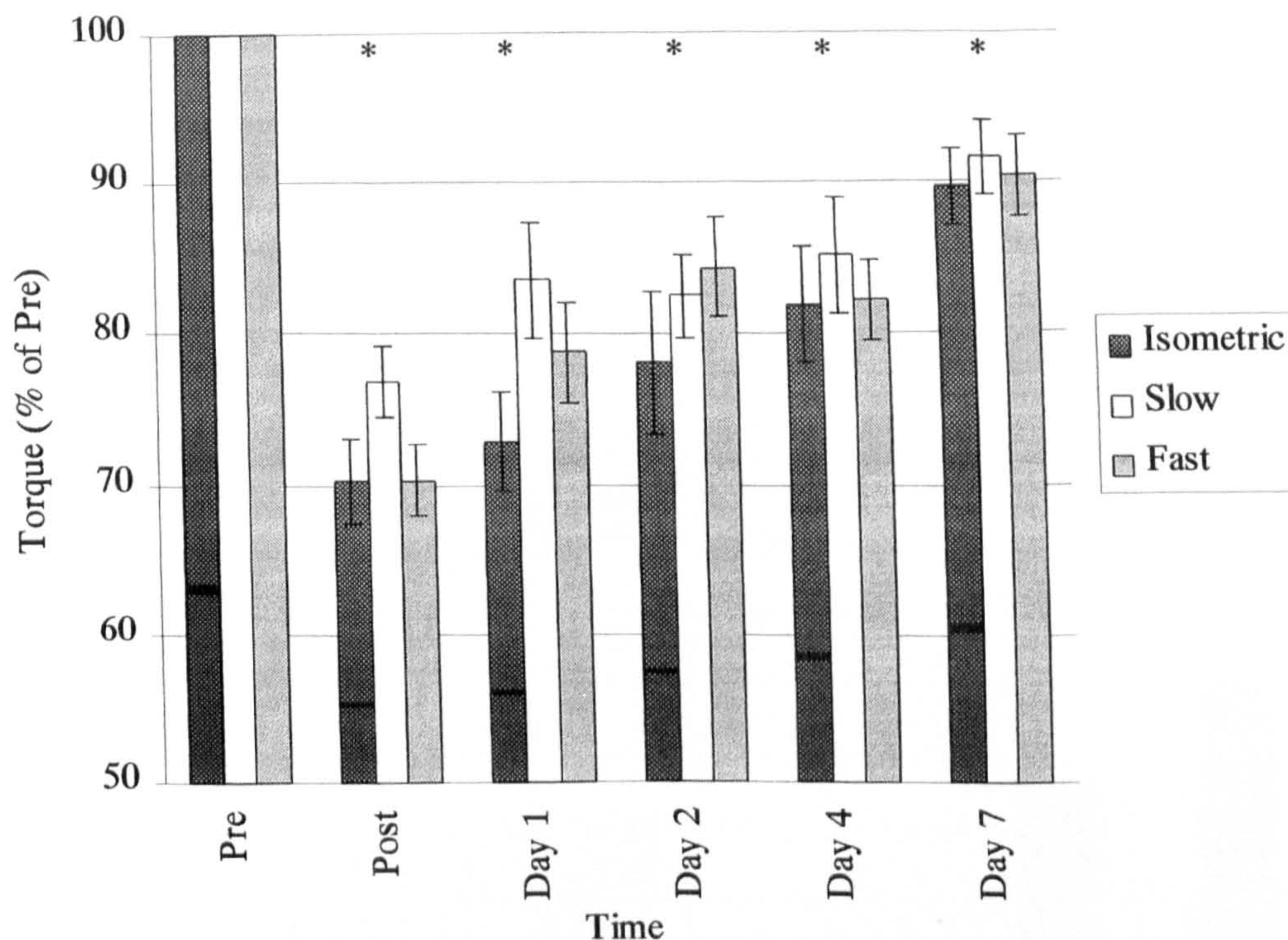


Figure 3.3: Changes in isometric strength at optimal muscle length (80°) and concentric strength at 30 deg s^{-1} (slow) and 180 deg s^{-1} (fast) across time following 100 eccentric MVCs of the knee extensors. Torque values are means (\pm S.E.M.) expressed as a percentage of the pre-eccentric exercise torque. *Significantly different ($P < 0.05$) from pre-exercise.

Table 3.1: Changes in isometric strength across time at short and optimal muscle length and concentric strength at 30 deg s^{-1} and 180 deg s^{-1} following 100 eccentric MVCs of the knee extensors. Torque values are means (\pm S.E.M.) expressed as a percentage of the pre-eccentric exercise torque.

	Pre	Post	Day 1	Day 2	Day 4	Day 7	Mean
Isometric	100	65.4	72.1	68.5	72.1	79.9	76.3*
(10°)		± 3.5	± 5.2	± 3.6	± 3.9	± 3.1	± 2.4
Isometric	100	70.3	72.9	78.1	81.9	89.6	82.1
(80°)		± 2.8	± 3.3	± 4.7	± 5.1	± 2.7	± 2.7
Concentric	100	76.8	83.6	82.5	85.2	91.7	86.6
(30 deg s^{-1})		± 2.3	± 3.8	± 2.8	± 3.8	± 2.5	± 2.0
Concentric	100	70.3	72.9	84.4	82.2	90.4	84.3
(180 deg s^{-1})		± 2.4	± 3.5	± 3.3	± 2.6	± 2.7	± 1.5

*Significantly different ($P < 0.05$) from isometric (80°)

3.4.4 Creatine kinase activity

Plasma CK activity changed significantly over time ($F_{(GG)5,35} = 18.3, P < 0.001$).

Creatine kinase activity was significantly higher than baseline on days 4 ($P < 0.05$) and 7 ($P < 0.05$) after the eccentric exercise (see Figure 3.4).

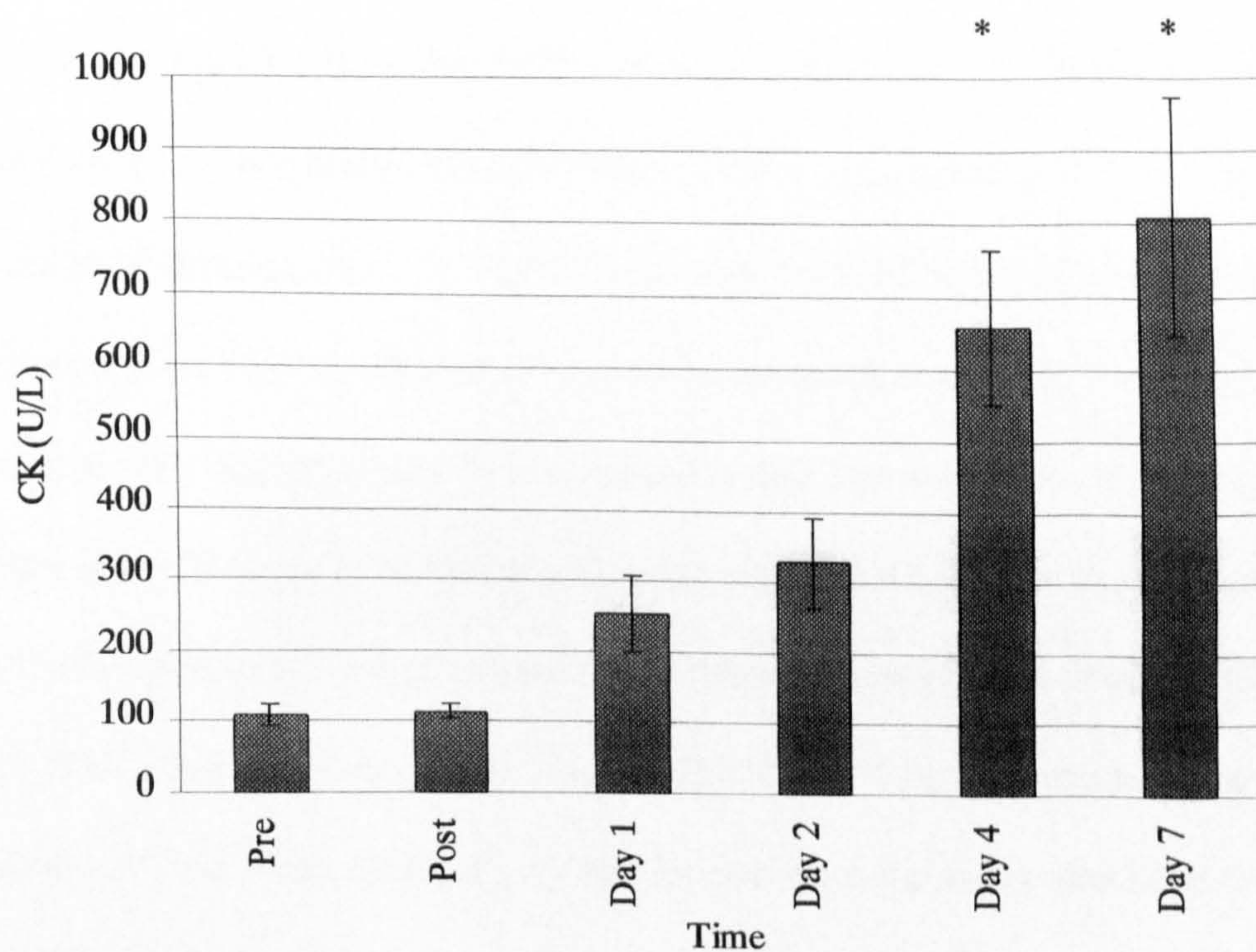


Figure 3.4: Changes in plasma CK activity across time following 100 eccentric MVCs of the knee extensors. CK values are means (\pm S.E.M.). *Significantly different ($P < 0.05$) from pre-exercise.

3.5 DISCUSSION

3.5.1 Evidence of muscle damage

The eccentric exercise protocol resulted in an immediate and prolonged reduction in muscle strength and an increase in circulating levels of the myofibre protein creatine kinase. Both of these measures are commonly used as indicators of exercise-induced muscle damage (Warren *et al.*, 1999). Immediate post-exercise values for strength (expressed as a percentage of pre-exercise values) ranged from 65.4 - 76.8% for all methods of measurement. By day 7 these values ranged from 79.9 - 91.7%. Creatine kinase activity was significantly elevated above baseline at days 4 and 7 post-exercise. The highest values were recorded at day 7 post-exercise. The delayed response in CK activity is typical following high-force eccentric exercise and has been shown to peak between 4 and 7 days post-exercise (Friden *et al.*, 1983; Jones *et al.*, 1986; Clarkson *et al.*, 1992). It is possible that CK activity reached a peak during this time in our study. Using a very similar eccentric exercise protocol as the one used in this study, Child *et al.* (1998) reported peak CK activity to occur at 5 days post-exercise. In our study plasma CK activity had a poor temporal relationship with the functional measures of muscle strength. Immediately and 24 hours post-eccentric exercise when muscle strength was affected to the greatest extent, CK activity was not significantly elevated above baseline. By day 7 when muscle strength was returning to pre-exercise values, CK activity was at its highest. For this reason, Warren *et al.* (1999) questioned the usefulness of myofibre proteins as criteria for measuring muscle damage.

3.5.2 Length-dependence of isometric strength loss

The results suggest that reductions in isometric strength following eccentric exercise are dependent on the muscle length at which strength is measured. Isometric strength was reduced to a significantly greater extent at short versus optimal muscle length ($76.3 \pm 2.5\%$ vs. $82.1 \pm 2.7\%$). Previous studies using the elbow flexors (Saxton and Donnelly, 1996) and the knee extensors (Child *et al.*, 1998) have also observed a length dependence of strength loss following eccentric exercise. Saxton and Donnelly (1996) reported a greater relative loss of strength at short versus long muscle length, which persisted for 4 days, whereas Child *et al.* (1998) only observed this effect immediately post exercise. Due to the non-significant interaction of time x angle in our study we were not able to determine the time course of the greater relative strength loss at short muscle length. However, a clear trend was evident suggesting that the greater loss of strength at short compared to optimum muscle length persisted throughout the seven day testing period.

Greater strength loss at short versus optimal muscle length indirectly supports the hypothesis that the length-tension curve shifts to the right, towards longer muscle lengths following eccentric exercise. Indeed, shifts in the optimum of the length-tension curve have been reported following eccentric exercise of the human ankle extensors (Jones *et al.*, 1997; Whitehead *et al.*, 1998) and whole toad sartorius muscle (Wood *et al.*, 1993; Talbot and Morgan, 1998). Morgan and Allen (1999) suggest that over-stretched sarcomeres failing to produce active tension and a reduction in sarcomere length of the remaining functional sarcomeres are the factors responsible for changes in the length-tension curve. Over-stretched sarcomeres would

result in a reduction in the number of cross-bridges available for force generation and this could explain the overall strength loss associated with exercise-induced muscle damage. A reduction in sarcomere length would account for the greatest loss of strength at short muscle lengths.

There are several consequences of the remaining functional sarcomeres becoming shorter at a fixed muscle length. The activation curve of muscle shifts to higher calcium levels at short sarcomere lengths (Endo, 1973), very high stimulation rates are required to achieve maximum force at short sarcomere lengths (Rack and Westbury, 1969), and the force frequency relationship is shifted to the right at short muscle (sarcomere) lengths (Edwards *et al.*, 1989a; Sacco *et al.*, 1994). The impact of these factors on muscle function will be dependent on the muscle length at which force is measured. For example, strength will be reduced at all muscle lengths but the greatest loss of strength will occur at short muscle lengths. Low frequency fatigue (LFF), a selective loss of force at low stimulation frequencies and a common feature of damaged muscle, will be evident at all lengths, but again will be greater at short muscle lengths (Edwards *et al.*, 1989b).

A disturbance in excitation-contraction coupling so that less calcium is released per action potential is often cited as the mechanism underlying strength loss and LFF (Edwards *et al.*, 1977b; Jones, 1981; Newham *et al.*, 1983; Warren *et al.*, 1993a; Ingalls *et al.*, 1998). E-C coupling failure would be expected to have its greatest effect on strength at short muscle length due to the higher level of activation needed for force generation at short muscle lengths. Although this failure alone could

explain the greater strength loss at short muscle length, it would be compounded by a redistribution of sarcomere lengths. Jones (1996) suggested that there may be two processes occurring in damaged muscle that lead to an impaired muscle function, one being a reduction in calcium release, the other a redistribution of sarcomere lengths.

3.5.3 Isometric versus dynamic strength loss

Our results suggest that isometric strength at optimal muscle length and concentric strength at slow and fast angular velocities were affected to a similar extent in terms of magnitude and rate of recovery following exercise-induced muscle damage (isometric $82.1 \pm 2.7\%$ vs. slow $86.6 \pm 2.0\%$ vs. $84.3 \pm 1.5\%$). There have been reports suggesting that strength loss and recovery may be dependent on the type and/or angular velocity of muscle action (Friden *et al.*, 1983; Golden and Dudley, 1992; Gibala *et al.*, 1995; Eston *et al.*, 1996). Friden *et al.* (1983) reported a slower restoration of concentric strength at $300 \text{ deg}\cdot\text{s}^{-1}$ compared to either 180 , $90 \text{ deg}\cdot\text{s}^{-1}$ or isometric strength, and Golden and Dudley (1992) reported a slower recovery of concentric strength at 180 versus $60 \text{ deg}\cdot\text{s}^{-1}$. For eccentric strength, Eston *et al.* (1996) reported a slower restoration of strength at 160 versus $30 \text{ deg}\cdot\text{s}^{-1}$. Evidence suggesting that type II motor units are selectively recruited (McHugh *et al.*, 2000) and fast type II fibres are selectively damaged (Friden *et al.*, 1983; Lieber and Friden, 1988) during eccentric exercise may account for the slower recovery of strength at the higher angular velocities in these studies. It is possible that the highest angular velocity used in the present study ($180 \text{ deg}\cdot\text{s}^{-1}$) may not have been high enough to detect any disproportionate performance changes due to selective type II fibre damage. However, the highest correlations between performance and percentage type

II fibres have been shown to occur at 180 deg s^{-1} (Thorstensson *et al.*, 1976; Ivy *et al.*, 1981) and so this velocity would seem to be the most appropriate to detect any disproportionate performance decrements attributable to selective type II fibre damage.

A potential mediating factor when assessing dynamic strength at different angular velocities is the joint angle (muscle length) at which strength is measured. The joint angle at peak torque is dependent on the angular velocity of movement, occurring later in the range of movement as the angular velocity increases (Thorstensson *et al.*, 1976). For the knee extensors this means that peak torque occurs at a shorter muscle length the higher the angular velocity. Thus, analysis of peak torque data irrespective of angular position may lead to erroneous conclusions about muscle function due to the length-dependent nature of strength loss following eccentric exercise. Future studies could make a more meaningful comparison of dynamic muscle function across angular velocities by measuring peak torque at a pre-determined joint angle, thereby controlling for muscle length.

3.5.4 Central factors and strength loss

Only peripheral mechanisms (i.e. at muscle level) have so far been discussed in accounting for the immediate and prolonged loss of strength. However, due to the use of voluntary contractions, central factors must also be considered. The inhibitory effects of muscle soreness could have adversely affected subject motivation and strength output. In an attempt to minimise the contribution of central factors participants were always given standardised written instructions, verbal

encouragement, and used visual feedback in an attempt to achieve the highest torque output and full voluntary activation of the knee extensors. Nevertheless, without the use of electrical muscle stimulation techniques it cannot be said for certain that participants achieved full voluntary activation. Evidence from studies employing superimposed electrical stimulation on isometric MVCs have shown that subjects can fully activate painful muscles at both optimal (Newham *et al.*, 1987; Jones *et al.*, 1989; Gibala *et al.*, 1995) and short (Saxton and Donnelly, 1996) muscle lengths. However, research has yet to demonstrate whether subjects can fully activate painful muscles during dynamic actions.

An impaired central drive so that full voluntary activation was not achieved at short muscle length could explain our results. Such a mechanism was evident in Olympic athletes suffering from the 'overtraining syndrome' (Koutedakis *et al.*, 1995), which exhibits symptoms of muscle damage, muscle soreness, and muscle weakness (Budgett, 1990). These athletes were able to achieve full voluntary activation during isometric MVCs at 80° knee flexion, but not at 10°. Koutedakis *et al.*, (1995) hypothesised that athletes in this state may not be able to recruit all motor units or achieve the high firing frequencies necessary for full voluntary activation at short muscle lengths. The present study was not designed to distinguish between central and peripheral mechanisms, however, it is suggested that both operated with peripheral mechanisms dominant.

3.5.5 Summary

These findings suggest that isometric strength loss following muscle damage is dependent on the muscle length at which strength is measured. Relative strength loss being greater at short versus optimal muscle length. A redistribution of sarcomere lengths, a failure in E-C coupling, and/or an impaired central motor drive are possible explanations for the present findings. Isometric and concentric strength at low (30 degs⁻¹) and high (180 degs⁻¹) angular velocities appeared to be affected to a similar extent in terms of magnitude and rate of recovery following muscle damage.

CHAPTER 4

THE EFFECT OF EXERCISE-INDUCED MUSCLE DAMAGE ON ISOMETRIC AND DYNAMIC KNEE EXTENSOR STRENGTH AND VERTICAL JUMP PERFORMANCE

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4.2 Introduction

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4.2.2 Dynamic strength and muscle damage

4.2.3 The stretch-shortening cycle and muscle damage

4.2.4 Research hypotheses

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4.3.3 Vertical jump height measurement

4.3.3.1 Squat jump

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4.3.3.3 Drop jump

4.3.4 Creatine kinase measurement

4.3.5 Muscle damaging exercise

4.3.6 Statistical analysis

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4.4.2 Vertical jump performance

4.4.3 Creatine kinase activity

4.5 Discussion

4.5.1 Evidence of muscle damage

4.5.2 Muscle damage and isometric, concentric, and eccentric strength

4.5.3 Maximal eccentric actions and voluntary activation

4.5.4 Muscle damage and vertical jump performance

4.5.5 Complex muscle function and muscle damage

4.5.6 Summary

4.1 ABSTRACT

The effect of exercise-induced muscle damage on knee extensor muscle strength during isometric, concentric, and eccentric actions and on vertical jump performance under conditions of squat jump, countermovement jump, and drop jump, was studied in eight participants (5 males and 3 females). Isometric strength was measured at 80° knee flexion (0° = full extension) and isokinetic concentric and eccentric strength were measured at an angular velocity of 90 deg·s⁻¹. The squat jump (SJ) was performed from a static squatting position; the countermovement jump (CMJ) from a standing position with a preparatory downward movement; and the drop jump (DJ) from a 0.6m high platform, with a drop to the floor followed by an immediate vertical rebound. Plasma creatine kinase (CK) was also measured from a fingertip blood sample. These measures were taken before, 1 hour after, and on days 1, 2, 3, 4 and 7 following a bout of 100 barbell squats (10 sets x 10 reps @ 70% body mass load), which was used to induce muscle damage. No significant differences ($P > 0.05$) were apparent in the magnitude and rate of recovery of strength between isometric, concentric, and eccentric muscle actions. Strength was reduced for 4 days ($P < 0.05$) following the damaging exercise. The overall decline in vertical jump performance was dependent on jump method. Squat jump performance was affected to a greater extent than CMJ ($91.6 \pm 1.1\%$ vs. $95.2 \pm 1.3\%$ of pre-exercise values, $P < 0.05$) and DJ performance ($91.6 \pm 1.1\%$ vs. $95.2 \pm 1.4\%$, $P < 0.05$). Overall jumping performance was significantly ($P < 0.05$) reduced for 3 days after the damaging exercise. Creatine kinase was significantly elevated ($P < 0.05$) above baseline at 1-hour post, peaked at day 1, and remained significantly elevated at days 2 and 3 post exercise. Strength loss following exercise-induced muscle damage was

shown to be independent of the muscle action being performed. However, the detrimental performance effects of muscle damage were attenuated when the stretch-shortening cycle was utilised in vertical jumping performance.

4.2 INTRODUCTION

4.2.1 Isometric strength and muscle damage

In functional terms, exercise-induced muscle damage is usually quantified by measures of isometric maximal voluntary contraction (MVC) torque (Warren *et al.*, 1999). Measures of isometric strength provide the primary means of determining muscle function after muscle damage. Previous research has demonstrated that isometric strength is reduced immediately after exercise and recovers over a time course of hours (Davies and White, 1981; Newham *et al.*, 1983b), days (Friden *et al.*, 1983; Gibala *et al.*, 1995; Hortobagyi *et al.*, 1998) or even over a week or more (Cleak and Eston, 1992; Howell *et al.*, 1993). The extent of isometric strength loss is dependent on the muscle length or joint angle at which it is measured, being more pronounced at short muscle lengths (Saxton and Donnelly, 1996; Child *et al.*, 1998; Byrne *et al.*, 2000, Chapter 3 of Thesis). Strength loss seems unrelated to soreness inhibition since comparable changes are observed between MVCs and stimulated force at high frequencies (Davies and White, 1981). Moreover, electrical stimulation superimposed on MVCs demonstrates that motor unit activation is similar at times when muscles are pain free pre-exercise and when they are suffering from DOMS post-exercise (Newham *et al.*, 1987; Jones *et al.*, 1989; Gibala *et al.*, 1995; Saxton and Donnelly, 1996). This suggests that subjects can fully activate painful muscles during isometric MVCs.

4.2.2 Dynamic strength and muscle damage

Much less is known about dynamic muscle strength following exercise-induced muscle damage. It is generally assumed that changes in isometric strength are comparable to

those in concentric and eccentric strength, despite a lack of evidence (Clarkson and Newham, 1995). Of the studies that have investigated isometric, concentric, and eccentric strength, Golden and Dudley (1992) observed a faster recovery of eccentric and isometric strength versus concentric strength, whereas, Hortobagyi *et al.* (1998) observed no differences in the magnitude or rate of recovery of strength across muscle actions. Differences in activation patterns between isometric and dynamic muscle actions (Tax *et al.*, 1990; Nakazawa *et al.*, 1993; Enoka, 1996; Kellis and Baltzopoulos, 1998) may provide reason for the extent of strength loss and the rate of recovery to be dependent upon the type of muscle action being performed. Eccentric strength may be particularly prone to exercise-induced muscle damage. Although well motivated subjects achieve greater muscle torques during eccentric versus isometric or concentric actions, it is difficult to achieve full voluntary activation during maximal eccentric actions (Dudley *et al.*, 1990; Westing *et al.*, 1990). A neural force limiting mechanism is thought to be responsible for the reduced muscle activation (EMG) observed during eccentric actions (Tesch *et al.*, 1990; Westing *et al.*, 1991; Enoka, 1996; Kellis and Baltzopoulos, 1998). This mechanism may be especially active during eccentric strength testing following damaging exercise in an attempt to limit further damage. Therefore, the first part of this study aimed to determine whether the magnitude and rate of recovery of strength loss following damaging exercise is dependent upon the type of muscle action being performed (isometric, concentric, eccentric).

4.2.3 The stretch-shortening cycle and muscle damage

The isolated muscle actions discussed above rarely occur alone in normal human muscle movements (Komi, 1984). Instead, natural muscle function occurs in a sequence of eccentric stretch, isometric coupling and concentric shortening of muscles, known as the stretch-shortening cycle (Hill, 1970; Komi, 1984). Komi (1984) stated that the stretch shortening cycle (SSC) of muscle function has a clear purpose: to allow the final phase (concentric action) to take place with greater force or power output, as compared to the condition where the movement is initiated by a concentric action alone. This has been demonstrated in experiments on isolated muscles (e.g. Cavagna *et al.*, 1968) and also during *in-vivo* human leg actions, such as the greater jump height achieved during a countermovement jump (with SSC) as compared to a squat jump (no SSC) (Asmussen and Bonde-Petersen, 1974; Komi and Bosco, 1978; Bobbert *et al.*, 1996). The mechanisms underlying performance enhancement during the SSC and their relative contributions are highly debatable, but four mechanisms have been identified: the time available to develop force; storage and reutilization of elastic energy; potentiation of the contractile machinery; and contribution of reflexes (see Ingen Schenau *et al.*, 1997a).

Vertical jumping is a convenient model to study performance with and without use of the SSC. Three types of jump can be studied: the squat jump (SJ) which is considered a pure measure of concentric muscle performance, the countermovement jump (CMJ) which utilises the SSC by a downward countermovement from an erect standing position, and the drop jump (DJ) which involves rebounding vertically following a drop from a specified height and also utilises the SSC. To date, no previous research has

examined dynamic muscle function using a vertical jumping model following exercise-induced muscle damage. It is conceivable that muscle damage may have differing effects on performance depending on the type of jump being performed. The jumps differ not only in their use of the SSC (SJ vs. CMJ and DJ) but also in the mechanisms underlying performance enhancement during the SSC (CMJ vs. DJ) (Ingen Schenau *et al.*, 1997b). Therefore, the second part of this study aimed to determine the effect of exercise-induced muscle damage on vertical jump performance under SJ, CMJ, and DJ conditions.

4.2.4 Research hypotheses

The following hypotheses applied to this investigation:

It is hypothesised that:

- a) Strength loss following exercise-induced muscle damage will not be dependent on the muscle action being performed.
- b) Vertical jump performance will be affected to a greater extent under conditions of squat jump than either countermovement jump or drop jump following exercise-induced muscle damage.

4.3 METHODS

4.3.1 Participants and design

Eight healthy participants, five males and three females (age 29.5 ± 7.1 yr (mean \pm SD), ht 1.74 ± 0.08 m, mass 69.5 ± 12.0 kg) were involved in the study. All individuals were moderately active but had not participated in any resistance training of the lower limbs for six months prior to the study, and none had any musculo-skeletal defects. Each individual gave written informed consent to participate in the study, which had previously been approved by the School ethics committee. The muscle group studied was the knee extensors. Each subject was evaluated for each criterion measure prior to, 1 hour afterwards, and on days 1, 2, 3, 4, and 7 following a bout of resistance exercise designed to induce the symptoms of exercise-induced muscle damage.

4.3.2 Isometric, concentric and eccentric strength measurement

All strength measurements were made on the non-dominant limb using a Kin-Com (500H, Chattecx, Chattanooga, TN, USA) isokinetic dynamometer. All subjects performed a familiarisation session during which they were introduced to standardised written instructions to work as hard and fast as possible against the resistance of the dynamometer and to use visual feedback to enhance torque output.

Following a warm-up consisting of three sub-maximal and two maximal contractions, subjects performed 2 sets of 3 maximal voluntary contractions (MVC) for each muscle action. Isometric, concentric, and eccentric muscle actions were presented in a random order. Isometric strength was measured by performing an MVC for 3 seconds at 80°

knee flexion (0° = full extension). A 30 second rest interval separated each repetition and a 3 minute rest interval separated sets. Isokinetic concentric and eccentric strength measurements were performed at an angular velocity of $90 \text{ deg}\cdot\text{s}^{-1}$, with 30 seconds rest between contractions and 3 minutes rest between sets. The range of motion was from 90° knee flexion to 10° knee flexion for concentric actions and 10° knee flexion to 90° knee flexion for eccentric actions. The highest peak torque from the two sets of contractions was used as the criterion measure for isometric, concentric and eccentric strength, respectively.

4.3.3 Vertical jump height measurement

All jumps were performed on an electronic timing mat system (Ergo Tester, Globus, Italy). The timing system measures flight time and is triggered by the feet of the subject at the moment of take-off and is stopped at the moment of contact upon landing. The method described by Komi and Bosco (1978) was used to calculate the height of rise of the centre of gravity. Firstly, by measuring flight time (t_{air}) in seconds, the vertical take-off velocity (V) of the centre of gravity is calculated as follows:

$$V = 0.5 (t_{\text{air}} \times g)$$

Where g = acceleration of gravity ($9.81 \text{ m}\cdot\text{s}^{-2}$)

Then, the height of rise of the centre of gravity can be calculated as follows:

$$\text{Height (m)} = \frac{V^2}{2g}$$

The computation assumes the jumper's position on the platform is the same at take-off and landing. Therefore, subjects were instructed to place hands on hips and to keep their body erect throughout. Upon landing, contact was made with the toes and the knees were fully extended. Any jumps that were perceived to deviate from the vertical plane were repeated. To determine whether jumping performance following exercise-induced muscle damage was influenced by the type of jump being performed, we measured jumping performance under three different conditions: squat jump; countermovement jump; and drop jump. No use of the SSC is permitted in the squat jump whereas in the countermovement and drop jump the SSC is utilised in performance.

4.3.3.1 Squat jump

The squat jump was performed from a squatting position with the knees flexed to approximately 90°. Subjects were asked to hold this position for three seconds and then upon the verbal command, "GO", the subjects jumped vertically for maximal height. It was imperative that no countermovement was made at the start of the jump. If such a movement was made the jump was repeated.

4.3.3.2 Countermovement jump

Subjects started from an erect standing position with knees fully extended (knee = 180°). Upon the verbal command, "GO", subjects made a downward countermovement to the same starting position as the squat jump (knee = 90°) and then jumped vertically for maximum height in one continuous movement.

4.3.3.3 Drop jump

Subjects were instructed to drop from a 0.6m high box to the floor and then jump vertically for height as soon as possible after landing, with minimum ground contact time. As a measure of reactive strength the jump is associated with very high muscle forces, very high power output around the knee joint and very high angular velocities of movement (Bobbert *et al.*, 1987).

4.3.4 Creatine kinase measurement

Plasma CK activity was determined from a fingertip blood sample. A warm fingertip was cleaned with a sterile alcohol swab and allowed to dry. Capillary puncture was made with a softclix lancet and a sample of whole fresh blood (32 μ l) was pipetted from a capillary tube onto the test strip and analysed for CK activity via a colorometric assay procedure (Reflotron, Boehringer Mannheim, Lewes, UK). This system uses a plasma separation principle, which is incorporated in the reagent carrier on the test strip.

4.3.5 Muscle damaging exercise

Each subject performed one hundred barbell squats, performed as 10 sets of 10 repetitions. The load on the barbell corresponded to 70% of the subjects body mass. Subjects started the movement with the barbell resting on their shoulders, body erect, legs fully extended (knee = 180°) and the toes pointing forward. The movement consisted of an eccentric action of the knee extensors to lower the barbell to a knee angle of approximately 90° and a concentric action to raise the barbell to the starting position. This ensured that the knee extensors were exercised at long muscle length during the

eccentric phase. Since this form of exercise was unaccustomed to all subjects, the load was moderate to ensure correct technique and to avoid injury. However, the volume of exercise was high, since evidence suggests that muscle damage increases progressively with the number of eccentric muscle actions (Hesselink *et al.*, 1996).

4.3.6 Statistical analysis

Strength and vertical jump data were analysed using two separate, two factor (Time x Muscle Action and Time x Jump Method) fully repeated measures ANOVAs. Ground contact time during the drop jump and plasma CK activity were analysed using two separate, single factor repeated measures ANOVAs. The assumption of sphericity was tested by the Mauchly Test of Sphericity. Statistical significance was set at the 0.05 alpha level. Post-hoc tests were performed with paired-sample t-tests using the Bonferonni correction technique.

4.4 RESULTS

4.4.1 Isometric, concentric and eccentric strength

Absolute values for isometric, concentric, and eccentric strength were 416 ± 49 Nm (mean \pm SEM), 384 ± 33 Nm, and 511 ± 55 Nm, respectively. There was a highly significant main effect for time on muscle strength ($F_{6,42} = 18.2, P < 0.001$). Reductions in strength persisted for 4 days and were approximately 20% lower at 1 hour post-exercise ($P < 0.05$), 25% lower at day 1 ($P < 0.05$), 21% lower at day 2 ($P < 0.05$), 15% lower at day 3 ($P < 0.05$), 13% lower at day 4 ($P < 0.05$), and 5% lower at day 7 ($P > 0.05$). No significant differences in strength (expressed as a % of pre-exercise strength) were apparent between muscle actions ($F_{2,12} = 1.2, P > 0.05$) and the time x muscle action interaction was also non-significant ($F_{12,84} = 1.0, P > 0.05$). The results suggest that isometric, concentric, and eccentric strength were affected to a similar extent, in terms of magnitude and rate of recovery, following the damaging exercise (see Figure 4.1 and Table 4.1).

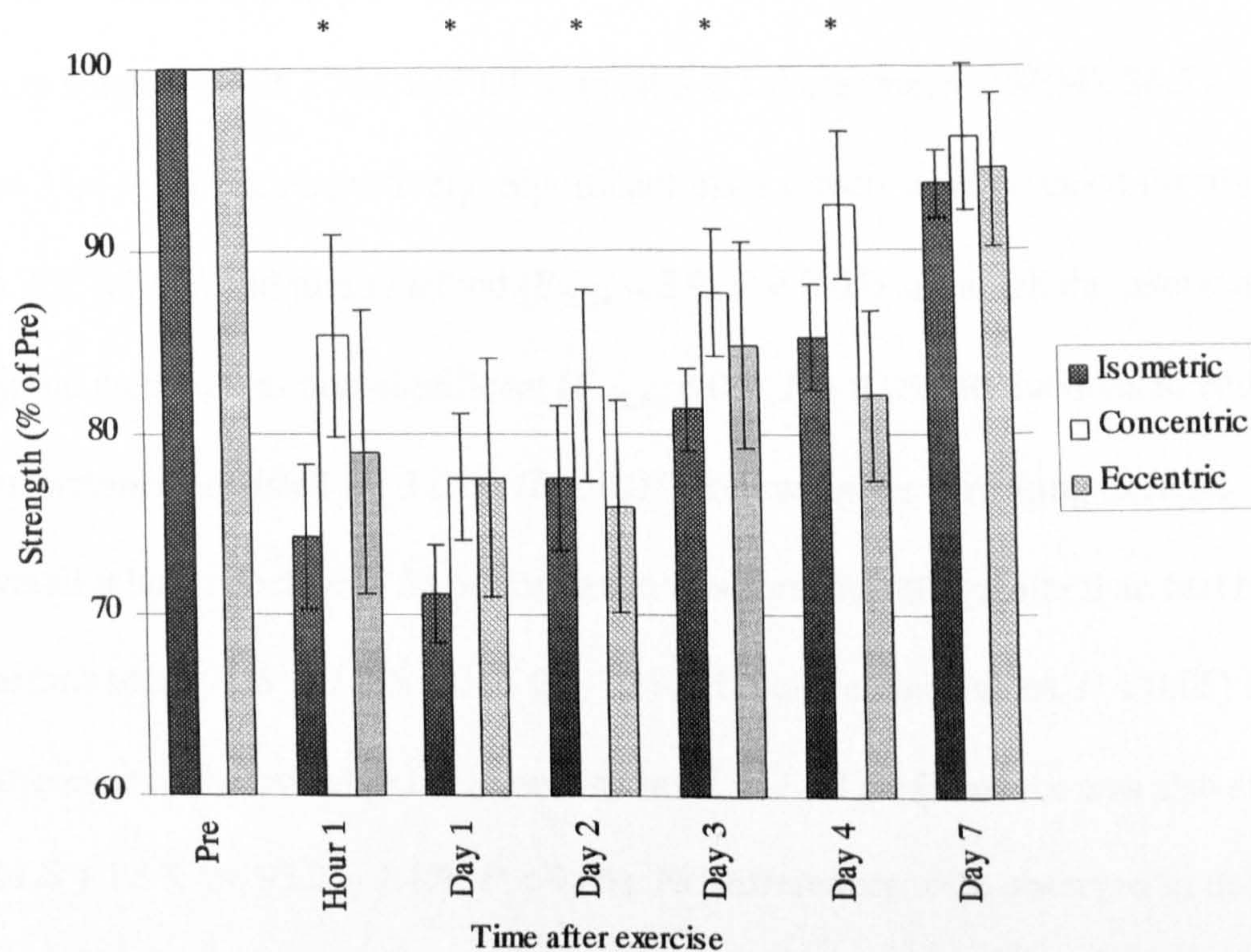


Figure 4.1: Changes in knee extensor muscle strength following exercise-induced muscle damage. Values are means (\pm SEM) expressed as a percentage of pre-exercise strength. *Significantly different ($P < 0.05$) from pre-exercise.

Table 4.1: Knee-extensor muscle strength following exercise-induced muscle damage. Values are means (\pm SEM) expressed as a percentage of pre-exercise strength.

	<i>Pre</i>	<i>Hour 1*</i>	<i>Day 1*</i>	<i>Day 2*</i>	<i>Day 3*</i>	<i>Day 4*</i>	<i>Day 7</i>	<i>Overall Mean</i>
<i>Isometric</i>	100	74.3 ± 4.0	71.2 ± 2.7	77.6 ± 4.0	81.3 ± 2.2	85.3 ± 2.8	93.6 ± 1.9	88.8 ± 3.2
<i>Concentric</i>	100	85.4 ± 5.5	77.6 ± 3.5	82.6 ± 5.2	87.7 ± 3.4	92.5 ± 4.0	96.1 ± 4.0	84.8 ± 4.4
<i>Eccentric</i>	100	79.0 ± 7.8	77.6 ± 6.6	76.0 ± 5.9	84.9 ± 5.6	82.1 ± 4.6	94.4 ± 4.2	83.3 ± 1.6

*Significantly different ($P < 0.05$) from pre-exercise.

4.4.2 Vertical jump performance

Jump heights for SJ, CMJ, and DJ were 34.3 ± 1.6 cm (mean \pm SEM), 36.2 ± 1.8 cm, and 33.6 ± 2.0 cm, respectively. Significant main effects were revealed for time ($F_{6,42} = 13.7, P < 0.01$) and jump method ($F_{2,14} = 3.9, P < 0.05$), although the interaction of time \times jump method was non-significant ($F_{12,84} = 0.7, P > 0.05$). Reductions in vertical jump performance persisted for 3 days ($P < 0.05$) following the damaging exercise. The overall relative decline in SJ performance was significantly greater than that in CMJ performance ($91.6 \pm 1.1\%$ vs. $95.2 \pm 1.3\%$ of pre-exercise values, $P < 0.05$) and the difference in the overall relative decline in SJ and DJ performance was also significant ($91.6 \pm 1.1\%$ vs. $95.2 \pm 1.4\%$, $P < 0.05$). No differences were observed in the overall relative decline in CMJ and DJ performance ($95.2 \pm 1.3\%$ vs. $95.2 \pm 1.4\%$, $P > 0.05$). Figure 4.2 and Table 4.2 display the relative changes in the 3 jump methods over time after the damaging exercise. Figure 4.3 displays the main effect for jump method. Ground contact time during the drop jump prior to exercise was 337 ± 31 ms (mean \pm SEM). An increase in contact time, approaching significance ($F_{6,42} = 2.0, P = 0.08$), was observed in the days following the damaging exercise (see Figure 4.4).

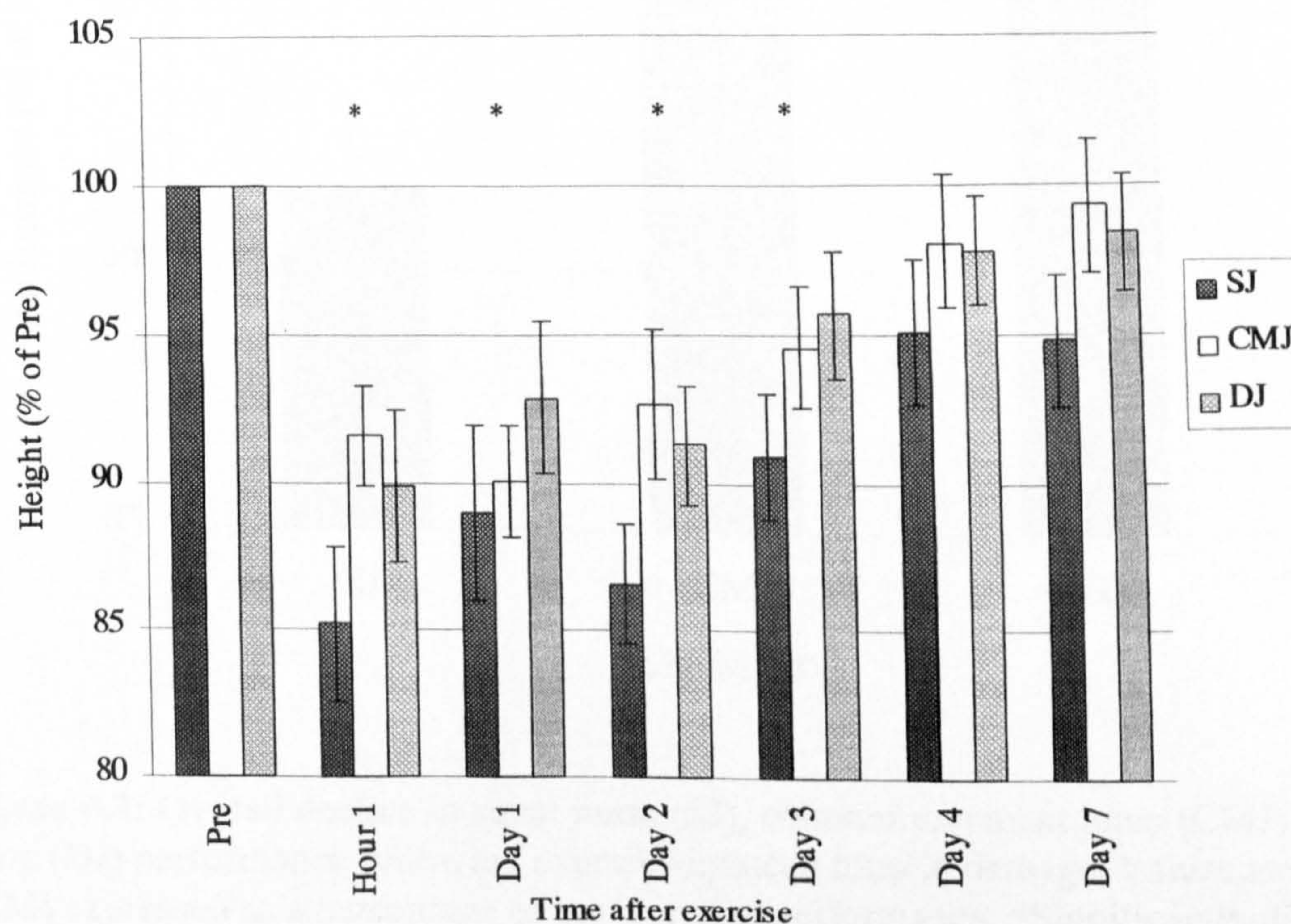


Figure 4.2: Changes in vertical jump performance following exercise-induced muscle damage. Values are means (\pm SEM) expressed as a percentage of pre-exercise performance. *Significantly different ($P < 0.05$) from pre-exercise performance.

Table 4.2: Vertical jump performance following exercise-induced muscle damage. Values are means (\pm SEM) expressed as a percentage of pre-exercise performance.

	<i>Pre</i>	<i>Hour 1*</i>	<i>Day 1*</i>	<i>Day 2*</i>	<i>Day 3*</i>	<i>Day 4</i>	<i>Day 7</i>	<i>Overall Mean</i>
<i>SJ</i>	100	85.2 ± 2.6	89.0 ± 3.0	86.6 ± 2.0	90.9 ± 2.1	95.1 ± 2.4	94.8 ± 2.2	91.6 ± 1.1
<i>CMJ</i>	100	91.6 ± 1.7	90.1 ± 1.9	92.7 ± 2.5	94.6 ± 2.0	98.1 ± 2.2	99.3 ± 2.2	95.2 ± 1.3
<i>DJ</i>	100	89.9 ± 2.6	92.9 ± 2.6	91.3 ± 2.0	95.7 ± 2.1	97.8 ± 1.8	98.4 ± 1.9	95.2 ± 1.4

*Significantly different ($P < 0.05$) from pre-exercise.

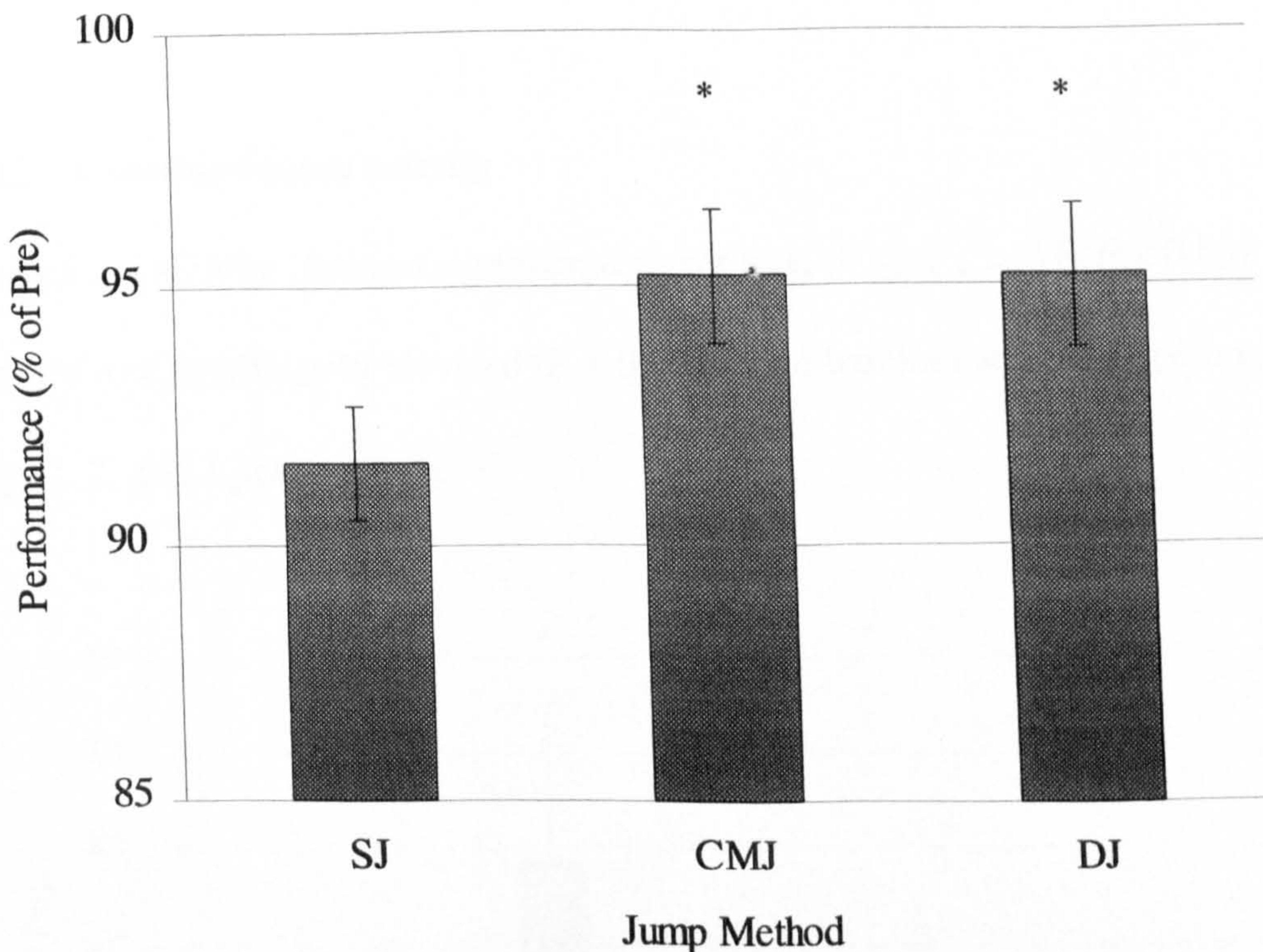


Figure 4.3: Overall decline in squat jump (SJ), countermovement jump (CMJ), and drop jump (DJ) performance following exercise-induced muscle damage. Values are means (\pm SEM) expressed as a percentage of pre-exercise performance. *Significantly different ($P < 0.05$) from SJ.

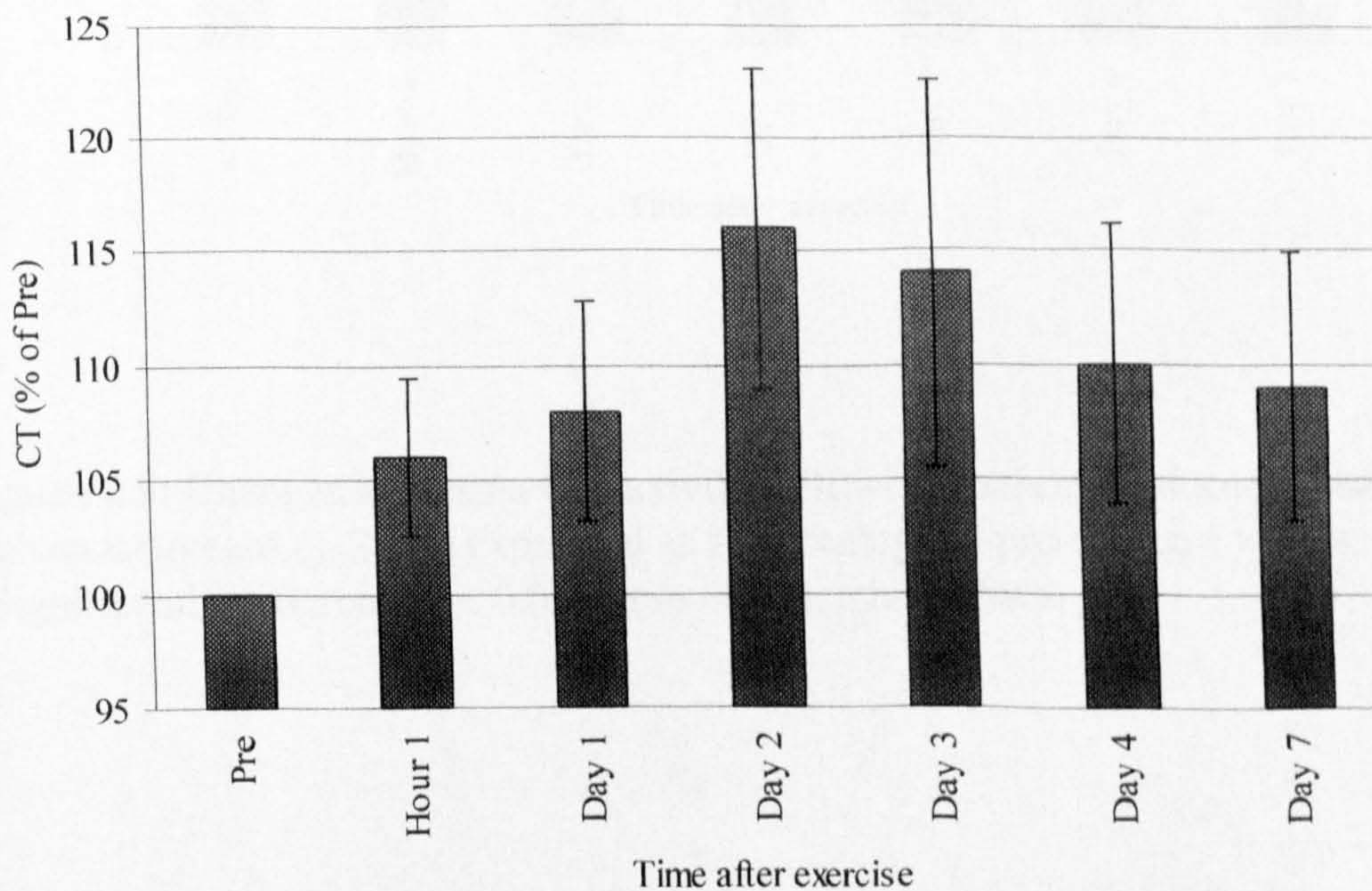


Figure 4.4: Ground contact time during the drop jump. Values are means (\pm SEM) expressed as a percentage of pre-exercise performance.

4.4.3 Creatine kinase activity

Plasma CK activity changed significantly over time ($F_{(GG)6,36} = 8.6, P < 0.05$). CK activity was significantly elevated ($P < 0.05$) above baseline at 1 hour post-exercise and days 1, 2, and 3 post-exercise.

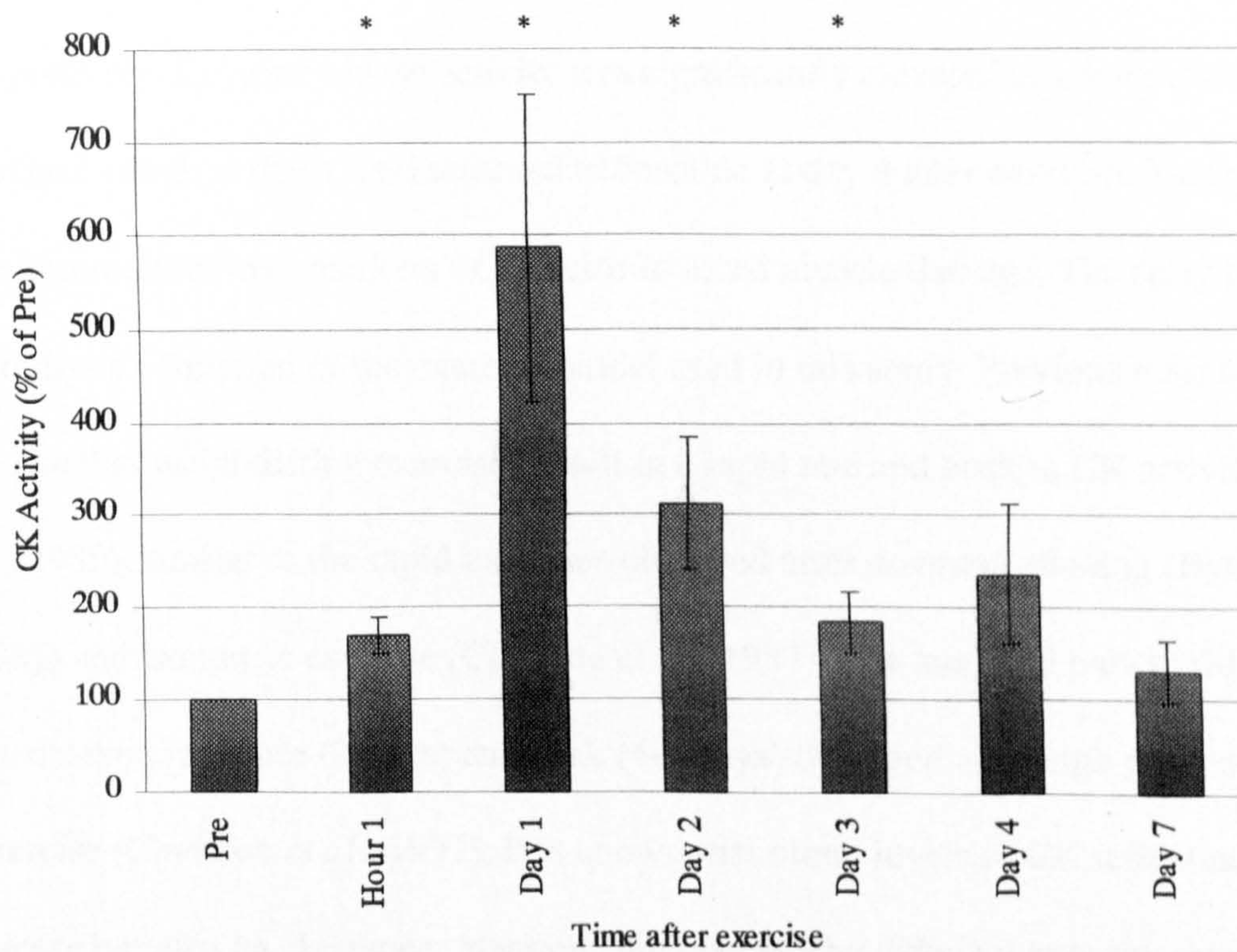


Figure 4.5: Changes in plasma CK activity following exercise-induced muscle damage. Values are means (\pm SEM) expressed as a percentage of pre-exercise values. *Significantly different ($P < 0.05$) from pre-exercise values.

4.5 DISCUSSION

4.5.1 Evidence of muscle damage

The exercise protocol resulted in an immediate and prolonged reduction in muscle function and a release of the myofibre protein creatine kinase. Strength measurements and vertical jump performance were significantly reduced for 4 days and 3 days, respectively. Creatine kinase activity was significantly elevated at 1-hour after exercise, reached a peak at day 1, and returned to baseline at day 4 after exercise. These symptoms are common indirect markers of exercise-induced muscle damage. The early peak in CK activity is a function of the exercise model used in this study. Previous research has shown that weightlifting exercises result in a rapid rise and peak in CK activity (Paul *et al.*, 1989), similar to the rapid increases observed after downhill running (Byrnes *et al.*, 1985) and isometric exercise (Clarkson *et al.*, 1987). The temporal pattern differs from the delayed response (2 days) and peak (4-7 days) observed after high force eccentric exercise (Clarkson *et al.*, 1992). It is known that blood levels of CK reflect not only its release but also its clearance; however, the reason why different exercise regimes produce different time courses in the CK response is unclear (Clarkson *et al.*, 1992).

4.5.2 Muscle damage and isometric, concentric, and eccentric strength

No significant differences were apparent in the overall reduction in strength (expressed as a % of pre-exercise) between isometric ($88.8 \pm 3.2\%$), concentric ($84.8 \pm 4.4\%$), and eccentric ($83.3 \pm 1.6\%$) muscle actions. Furthermore, there was no significant time x muscle action interaction. Previous research comparing these strength measures following damaging exercise is very scarce. Golden and Dudley (1992) measured

isometric and concentric and eccentric strength at 60 and 180 deg·s⁻¹. These measures were affected to a similar extent but demonstrated contrasting recovery patterns.

Concentric strength was slower to return to baseline and this was most evident at 180 deg·s⁻¹. In contrast, Hortobagyi *et al.* (1998) found no differences between isometric and concentric and eccentric strength at 30 deg·s⁻¹, the three strength measures being affected to a similar extent in terms of magnitude and time course of recovery. The magnitude of the effect size for the non-significant main effect in the present study was 0.15, which, according to Cohen (1992) corresponds to a less than small effect size. According to Stevens (1996) a very large sample size ($n = 106$) would be required to give the study sufficient power (0.80) to detect a small effect size at alpha 0.05. The results display no significant or practical differences between the muscle actions and are in agreement with the results of Hortobagyi *et al.* (1998). However, when the torque-velocity relationship is studied in more detail, that is, when more than one angular velocity of movement is studied, strength loss may be dependent on the type of muscle action and/or the angular velocity of movement (Golden and Dudley, 1992).

4.5.3 Maximal eccentric actions and voluntary activation

The pre-exercise strength values revealed a 19% and 25% greater peak torque for eccentric actions (511 ± 55 Nm) compared to isometric (416 ± 49 Nm) and concentric actions (384 ± 33 Nm), respectively. Although this indicates that subjects were highly motivated, research suggests that it is difficult for subjects to achieve full activation and the true maximum torque during a voluntary eccentric contraction (Tesch *et al.*, 1990; Westing *et al.*, 1990; 1991; Enoka, 1996; Kellis and Baltzopoulos, 1998). Furthermore,

inhibition may particularly effect maximal eccentric contractions after damaging exercise in an attempt to limit further damage. Although subjects may not be able to achieve full voluntary activation pre-damage, it is not known if subjects can achieve the same activation levels after damage. For isometric MVCs subjects can achieve full voluntary activation both before and after damage (Newham *et al.*, 1987; Jones *et al.*, 1989; Saxton and Donnelly, 1996). Superimposed electrical stimulation or artificial activation needs to be applied to dynamic actions to determine the true functional state of damaged muscle. Voluntary actions alone, although ecologically valid, may not be giving a true insight into the functional status of damaged muscle.

4.5.4 Muscle damage and vertical jump performance

Reductions in vertical jump performance were immediate, long lasting, and dependent on the type of jump being performed. The reduction in jumping performance can most likely be explained by the observed reduction in strength of the knee extensors.

However, since muscle damage affected SJ performance to a greater extent than either CMJ or DJ performance, the way in which strength is utilised seems an important determinant of performance. The CMJ and DJ utilise the SSC in performance whereas the SJ does not. In a SSC the final concentric muscle action is enhanced by a preceding eccentric muscle action. This is accomplished by the preceding eccentric action producing a greater active state and muscle force at the beginning of the concentric action, compared with a movement that involves only a concentric action such as the SJ (Bobbert *et al.*, 1996; Ingen Schenau *et al.*, 1997a). The net effect is that during the concentric phase of a CMJ and DJ, the contractile element can produce more force at a

given length or shortening velocity compared to the SJ (Ingen Schenau *et al.*, 1997a).

This potentiating mechanism seems to attenuate the detrimental performance effects of exercise-induced muscle damage. A long-standing theory accounting for strength loss following muscle damage is that the excitation-contraction coupling system is impaired (Edwards *et al.*, 1977b), probably due to damage to the sarcoplasmic reticulum so that less calcium is released per action potential. This defect would manifest itself more during SJ performance than either CMJ or DJ performance because of the greater active state that is built up prior to shortening in SSC movements.

The present results are in line with those of Hortobagyi *et al.* (1991) following intensive SSC exercise and Chambers *et al.* (1998) following an ultramarathon foot race.

Although these studies did not directly study the effects of exercise-induced muscle damage, the exercise protocols used are known to induce the symptoms of muscle damage. Hortobagyi *et al.* reported that when squat jump performance was reduced by 11% and isometric MVC by 20% following 50 drop jumps, CMJ and DJ performance were unaffected. Chambers *et al.* reported that following a 90 km foot race, SJ performance remained significantly lower for longer (18 days), compared to CMJ (11 days) and DJ (3 days). These results suggest that when the SSC is utilised in performance the detrimental effects of muscle damage are attenuated.

4.5.4 Complex muscle function and muscle damage

According to Komi and Gollhofer (1997), an effective SSC requires three fundamental conditions: a well timed pre-activation of the muscle(s) before the eccentric phase; a short and fast eccentric phase; and an immediate transition (short delay) between stretch (eccentric) and shortening (concentric phase). The authors suggest that the DJ meets these criteria but the CMJ does not. The drop jump provides the opportunity to study natural muscle function as it occurs in the sporting context. The ability to reverse a movement from an eccentric to a concentric contraction at high speed, reactive strength (Young *et al.*, 1999), relies on a contribution from both voluntary and reflex actions. The present results revealed that contact time in the drop jump increased by up to 15 % (day 2) after damaging exercise. It is likely that the stretch, transition, and shortening phases, of the SSC, all lengthened during impact due to the loss of eccentric, isometric, and concentric strength, respectively. Previous research investigating drop jump performance following damaging exercise has reported reduced stretch reflex sensitivity and reduced muscle stiffness regulation (Horita *et al.*, 1996; Nicol *et al.*, 1996; Horita *et al.*, 1999) which serve to deteriorate the force potentiating mechanisms during the SSC. The authors propose that muscle damage affects the stretch reflex and stiffness regulation through muscle spindle disfacilitation and through inhibition due to activation of III and IV afferent nerve endings. Horita *et al.* (1999) also suggested that muscle damage induces modifications in the pre-landing motor control, possibly brought about by central inhibition due to muscle soreness. With current research revealing that force and position sense (Brockett *et al.*, 1997) and motor skill (Pearce *et al.*, 1998) are adversely

affected by muscle damage, it is apparent that exercise-induced muscle damage has a multi-faceted effect on muscle function.

4.5.5 Summary

Strength loss following exercise-induced muscle damage was similar in magnitude and recovery rate for isometric, concentric, and eccentric actions. Vertical jump performance was affected to a greater extent under squat jump conditions than either countermovement or drop jump. The stretch-shortening cycle possibly attenuates the detrimental performance effects of exercise-induced muscle damage.

CHAPTER 5

EFFECT OF ECCENTRIC EXERCISE-INDUCED MUSCLE DAMAGE ON KNEE EXTENSOR ISOMETRIC AND DYNAMIC FATIGUE, ISOMETRIC STRENGTH AND MAXIMAL POWER OUTPUT

5.1 Abstract

5.2 Introduction

5.2.1 Isometric fatigue and muscle damage

5.2.2 Isometric versus dynamic fatigue

5.2.3 Maximal isometric strength versus maximal power output

5.2.4 Research hypotheses

5.3 Methods

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5.3.3 Isometric fatigue measurement at 40° and 80° knee flexion

5.3.4 The Wingate anaerobic test

5.3.5 Creatine kinase measurement

5.3.6 Muscle damaging exercise

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5.4.1 Isometric strength at 40° and 80° knee flexion

5.4.2 Isometric fatigue at 40° and 80° knee flexion

5.4.3 Isometric fatigue versus dynamic fatigue

5.4.4 Maximal isometric strength versus maximal power output

5.4.5 Creatine kinase activity

5.4.6 Statistical analysis

5.5 Discussion

5.5.1 Observations on pre-exercise muscle function

5.5.2 Evidence of muscle damage

5.5.3 Muscle damage and fatigue susceptibility

5.5.4 Muscle damage and maximal strength and power

5.5.5 Summary

5.1 ABSTRACT

The effect of eccentric exercise-induced muscle damage on angle-specific isometric knee extensor strength and fatigue, and peak power and fatigue was studied in seven participants (5 males, 2 females). Isometric strength was measured at 80° and 40° knee flexion, corresponding to optimal and short muscle length, respectively. Isometric fatigue was assessed by a 60s sustained isometric maximal voluntary contraction (MVC) at each muscle length and during the 30s Wingate cycle test. The rate of fatigue was calculated as the slope of a linear regression line (b) fitted to the 60 data points of the MVCs and the 30 data points of the Wingate test. Plasma creatine kinase (CK) was also measured from a fingertip blood sample. These measures were taken before, 1 hour after, and on days 1, 2, 3, and 7 following 100 eccentric squats (10 sets x 10 reps @ 80% concentric 1 RM). Prior to damaging exercise, the knee extensors fatigued at a significantly ($P < 0.05$) greater rate at optimal muscle length versus the shortened length. Following damaging exercise, strength was reduced ($P < 0.05$) for 7 days and the rate of fatigue was lower for 3 days ($P < 0.05$) and 1 day ($P < 0.05$) at optimal and short muscle length, respectively. Wingate fatigue was lower ($P < 0.05$) for 3 days. Maximal power output was reduced to a lesser extent ($P < 0.05$) than strength at optimal length at 1-hour post and although the time course of recovery was equal, the pattern of recovery differed. Creatine kinase activity was significantly elevated above baseline values at 1-hour ($P < 0.05$) and day 1 ($P < 0.05$). Damaged muscle was weaker but had an improved ability to maintain force and power, albeit at lower than pre-exercise levels. The lower rate of fatigue in damaged muscle can be explained by selective damage or fatigue in type II fibres, which would reduce their contribution to the post-exercise fatigue curves.

5.2 INTRODUCTION

5.2.1 Isometric fatigue and muscle damage

Fatigue has been defined as an inability to maintain the required or expected force or power output (Edwards, 1981). The susceptibility of damaged muscle to fatigue is an aspect of muscle function that has been studied infrequently (Warren *et al.*, 1999).

Only two previous studies have examined this aspect of muscle function (Davies and White, 1981; Balnave and Thompson, 1993), both using an electrically elicited isometric fatigue test consisting of trains of stimuli at 20 Hz, lasting 300 ms and repeated every second for two minutes. Davies and White (1981) reported that damaged muscle was weaker but no more fatigable, whereas Balnave and Thompson (1993) reported that damaged muscle was weaker but, surprisingly, less fatigable. Davies and White (1981) stated that with damaged muscle, one is investigating muscle weakness rather than fatigue, that is, the inability to generate, rather than maintain, force.

Volitional fatigue has not been studied previously in damaged muscle. During 60s sustained maximal voluntary contractions (MVCs) of the knee extensors in the fresh state, Bigland-Ritchie *et al.* (1978) reported that central fatigue consistently accounted for up to 30% of the total force loss, even in apparently well motivated subjects. It is possible that this proportion would increase following muscle damage, due to the inhibitory effects of muscle soreness accompanying damage, and result in the fatigability of damaged muscle *increasing* rather than decreasing. Furthermore, an aspect of fatigue during MVCs is that susceptibility to fatigue is influenced by muscle length. Human muscle has been reported to be less fatigable when tested at short versus optimal muscle length during MVCs of the ankle dorsi flexors (Fitch

and McComas, 1985) and elbow flexors (McKenzie and Gandevia, 1987). Therefore, the first part of this study aimed to investigate fatigue susceptibility of the knee extensors during sustained 60s isometric MVCs when performed at optimal muscle length and at a shortened length for torque generation. This was investigated before and for seven days after exercise-induced muscle damage.

5.2.2 Isometric versus dynamic fatigue

In the fresh state, human skeletal muscle has been shown to fatigue more rapidly under dynamic versus isometric conditions. During all out cycle ergometry, Sargeant *et al.* (1981) reported a rapid loss of power so that by 30 seconds, power was reduced to 50-60% of the maximum value. In comparison, sustained isometric MVCs demonstrate a modest loss of force so that by 30 seconds force is reduced by about 20% (Bigland-Ritchie *et al.*, 1978). However, these relationships have not been determined for damaged muscle. The study of power output in addition to isometric force allows for the investigation of possible differential effects of muscle damage on isometric and dynamic muscle performance. The generation of power output by muscle shortening is more energetically demanding and requires higher rates of ATP regeneration than isometric force generation (Woledge, 1985). Hesselink *et al.* (1996) suggested that damage could impair normal metabolism and concomitant performance since changes in metabolism have been reported by Van der Meulen *et al.* (1992) after eccentric exercise. Vollestad (1997) stated that the assessment of power output can provide additional information regarding any changes involved in energy release and utilization, since changes are more easily detected by shortening compared with isometric contractions. Therefore, the second part of this study aimed to determine the effect of muscle damage on the rate of fatigue during a 60s

isometric MVC and during a maximal intensity 30s cycle ergometer test, to determine any differential effects of muscle damage on isometric and dynamic muscle performance.

It is very interesting to note that Maughan *et al.* (1997) suggested that the rapid and marked rise and subsequent decline in force and power output observed during intense muscle contractions, such as sustained MVCs or maximal cycling, may be closely related to the activation and rapid fatigue of type II fibres. Especially when evidence suggests that type II fibres are selectively damaged during eccentric exercise (Friden *et al.*, 1983; Jones *et al.*, 1986; Lieber and Friden, 1988; Friden and Lieber, 1992; MacPherson *et al.*, 1996). Such a scenario would serve to maintain force and power in damaged muscle, albeit at lower levels, since the type II fibre contribution to performance will be reduced and performance will reflect the more fatigue resistant type I and IIa fibres. In this context the fatigability of damaged muscle will be *reduced*.

5.2.3 Maximal isometric strength versus maximal power output

Previously, only Sargeant and Dolan (1987) have investigated the ability of damaged muscle to generate maximal power output. Following a bout of downhill walking in just four subjects, the authors reported an immediate, significant, and sustained reduction in short-term power output, measured concentrically on an isokinetic cycle ergometer. Reductions persisted for four days and were accompanied by reductions in maximal isometric strength of a similar magnitude. Consequently, Clarkson and Newham (1995) have stated that this area warrants further investigation and commented that it is not known if the time course of recovery for power generation is

the same as for isometric strength. Therefore, the final aim of this study was to compare the decrement and time course of recovery of maximal isometric strength and power output following exercise-induced muscle damage.

5.2.4 Research hypotheses

The following hypotheses applied to this investigation:

It is hypothesised that:

- a) **Fatigue during sustained isometric MVCs will be greater at optimal versus short muscle length both before and after damaging exercise. However, damaged muscle will be less fatigable at both muscle lengths;**
- b) **Fatigue will be greater during the cycle power test versus the isometric test and this will be evident both before and after damaging exercise;**
- c) **Isometric strength will be affected to a greater extent than power output after muscle damage but the two parameters will share a similar pattern and time course of recovery.**

5.3 METHODS

5.3.1 Participants and Design

Seven healthy participants, five males and two females (age 22.6 ± 4.4 yr (mean \pm SD), ht 1.78 ± 0.08 m, mass 75.7 ± 11.7 kg) were involved in the study. All individuals were moderately active but had not participated in any resistance training of the lower limbs for six months prior to the study, and none had any musculo-skeletal defects. Each individual gave written informed consent to participate in the study, which had previously been approved by the School ethics committee. The muscle group studied was the knee extensors. Each subject was evaluated for each criterion measure prior to, 1 hour afterwards, and on days 1, 2, 3, and 7 following a bout of resistance exercise designed to induce the symptoms of exercise-induced muscle damage.

5.3.2 Isometric strength at 40° and 80° knee flexion

Isometric strength and fatigue measurements were made on the knee extensors of the non-dominant limb using a Kin-Com (500H, Chattecx, Chattanooga, TN, USA) isokinetic dynamometer. Participants performed isometric MVCs of the knee extensors at 80° and 40° knee flexion, corresponding to optimum muscle length and a shortened length for torque generation. The testing positions were obtained by entering full knee extension (0°) as a reference value into the Kin-Com visual display. The reproducibility of this method was checked on each testing occasion by noting the Kin-Com angle display when the lever arm was at true 90° (determined by spirit level). The pre-test angle display at true 90° was used as the criterion. If any difference existed, the process was repeated until the criterion was achieved. Three sub maximal and one maximal practice repetitions acted as warm-up at each testing

position. Three MVCs of three seconds duration were performed at each joint angle, presented in a random order, with a one-minute rest period between repetitions. The highest peak torque from the three contractions was used as the criterion score for short and optimal muscle length, respectively.

5.3.3 Isometric fatigue at 40° and 80° knee flexion

Fatigue was assessed by having subjects perform a 60s MVC at both 40° and 80° knee flexion. A target of 100% of the current MVC was entered into the Kin-Com visual display which gave real time feedback of the subjects torque production and the target. The test started when subjects achieved the 100% MVC target torque. Tests at 40° and 80° were presented in a mixed order and were separated by 20 minutes of recovery. Torque was recorded each second during the 60s contraction. The scenario of performing a 60s MVC has been described by Bigland-Ritchie *et al.* (1978), "To maintain a 60s MVC of the quadriceps takes a considerable effort. During the first 30s discomfort is mild but between 30 and 45s pain in the thigh becomes increasingly severe. There is a progressive change in the perceived sensations from the leg with the result that a subject, without visual feedback, is uncertain of the force exerted towards the end of the contraction."

Fatigue was quantified as the slope of the regression line, determined through linear regression analysis of the 60 data points. The slope of the line or regression coefficient (b) represents the rate of change in y with a unit change in x . In this study, for every unit change in x (time), y (torque) decreased by b . Therefore, since torque declined over time during the fatigue tests, b was always a negative value. As such, the less negative the number, the less fatigable the knee extensors were.

5.3.4 The Wingate anaerobic test

The Wingate anaerobic test was used to assess power output of the legs. Use of the test is widespread (Winter, 1996) and it is considered the most sensitive and reliable assessment of anaerobic performance presently available (Bird and Davison, 1997). The test was performed according to British Association of Sport and Exercise Sciences (BASES) guidelines (Bird and Davison, 1997). Prior to testing, a fingertip blood lactate sample was taken using a softclix lancet, capillary tube, and an Accusport Lactate Analyser (Boehringer Mannheim, Lewes, UK). Subjects performed a 5 minute warm up on a cycle ergometer at 100 Watts, which included a flat out sprint at 3 minutes for 5 seconds, followed by 5 minutes of rest. Subjects were then transferred to the test ergometer (Monark 814 E). The seat height was adjusted for comfort and was recorded, toe clips were secured, the resistive load attached, and a restraining harness was positioned to prevent subjects rising from the seat. The resistive load corresponded to 10% and 8% of body mass for males and females, respectively (Bird and Davison, 1997). Subjects began pedalling at 50-60 RPM with the external load supported. Upon the command "3, 2, 1, GO", the load was applied abruptly, subjects pedalled flat out for 30 seconds, and power output data was recorded every second for 30 seconds. A warm-down consisting of 2 minutes of cycling at 100 Watts was then performed, followed by a post-exercise blood lactate sample at 3 minutes. Peak power was quantified as the highest power output value in the first 5 seconds of the test. Regression analysis was performed on the 30 data points and the regression coefficient (*b*) was used to quantify fatigue.

On a Monark cycle ergometer, one revolution of the pedal crank moves a point on the flywheel a distance of 6m. Therefore, an expression for power output (W) can be produced:

$$1W = 1J \cdot s^{-1} = 1Nm \cdot s^{-1} \quad \text{then} \quad W = \frac{R}{60} \times 6m \times \text{Load (N)}$$

From Winter (1996).

5.3.5 Creatine kinase measurement

Plasma CK activity was determined from a fingertip blood sample. A warm fingertip was cleaned with a sterile alcohol swab and allowed to dry. Capillary puncture was made with a softclix lancet and a sample of whole fresh blood (32 μ l) was pipetted from a capillary tube onto the test strip and analysed for CK activity via a colorometric assay procedure (Reflotron, Boehringer Mannheim, Lewes, UK). This system uses a plasma separation principle, which is incorporated in the reagent carrier on the test strip.

5.3.6 Muscle damaging exercise

Each subject performed 10 sets of 10 repetitions of the eccentric phase of the barbell squat exercise. The load on the barbell corresponded to 80% of the subject's concentric one repetition maximum. Subjects started the movement with the barbell resting on their shoulders, body erect, legs fully extended (knee = 180°) and the toes pointing forward. The movement consisted of an eccentric action of the knee extensors to lower the barbell to a knee angle of approximately 90°. The barbell was then raised to the starting position by two individuals acting as spotters. Two minutes of rest separated each set.

5.3.7 Statistical analysis

Isometric strength and fatigue data were analysed by two separate, two factor (Time x Angle) repeated measures ANOVAs. A comparison of isometric strength and Wingate peak power and isometric fatigue and Wingate fatigue was analysed by two separate, two factor (Time x Mode) repeated measures ANOVAs. Creatine kinase activity and post-Wingate blood lactate data were analysed using separate single factor repeated measures ANOVAs. The assumption of sphericity was tested by the Mauchly Test of Sphericity. Any violations of this assumption were corrected by using the Greenhouse-Geisser adjustment to raise the critical value of F, as indicated by (GG) . To determine the regression coefficient (b) to quantify fatigue, individual regression analyses were performed on performances during the isometric endurance test and the Wingate test. Statistical significance was set at the 0.05 alpha level. Post-hoc tests were performed with paired-sample t-tests using the Bonferonni correction technique.

RESULTS

5.4.1 Isometric strength at 40° (short muscle length) and 80° (optimal muscle length) knee flexion

Absolute values for isometric peak torque at 40° and 80° were 302 ± 19 Nm (mean \pm SEM) and 495 ± 33 Nm, respectively. There was a highly significant main effect for time ($F_{5,30} = 50.8, P < 0.001$) but the main effect for angle was non-significant ($F_{1,6} = 2.5, P > 0.05$). The time x angle interaction was also non-significant ($F_{5,30} = 1.0, P > 0.05$).

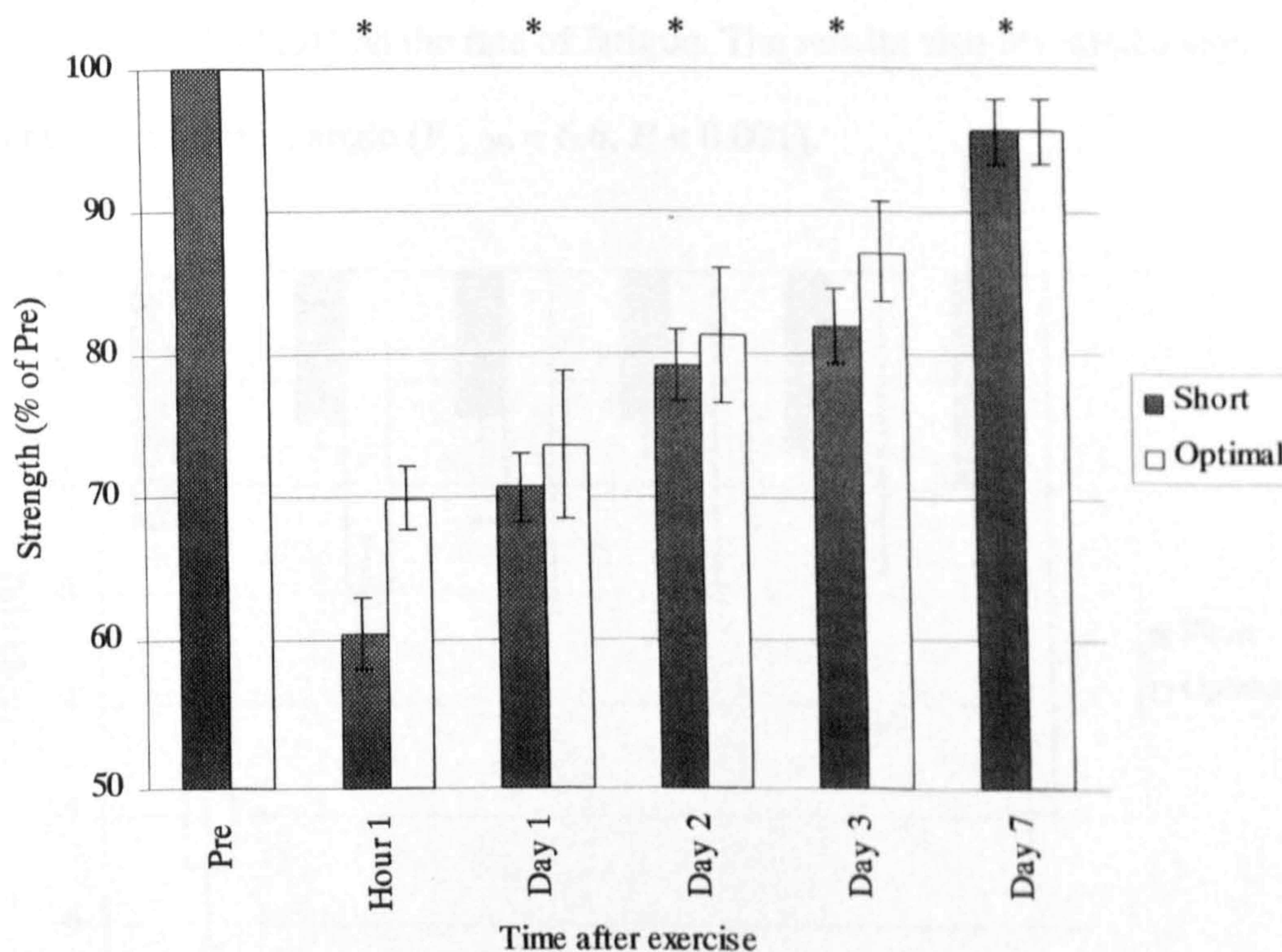


Figure 5.1: Changes in isometric strength at 40° (short) and 80° (optimal) knee flexion following exercise-induced muscle damage. Values are means (\pm SEM) expressed as a percentage of pre-exercise strength. *Significantly different ($P < 0.05$) from pre-exercise.

Strength was significantly reduced 1-hour post and remained significantly reduced for 7 days following the damaging exercise (see Figure 5.1). Strength was

approximately 35% lower at 1-hour post ($P < 0.05$), 28% lower at day 1 ($P < 0.05$), 20% lower at day 2 ($P < 0.05$), 15% lower at day 3 ($P < 0.05$), and 5% lower at day 7 ($P < 0.05$). In contrast to previous studies (Saxton and Donnelly, 1996; Child *et al.*, 1998; Byrne *et al.*, 2000, Chapter 3 of thesis), no disproportionate loss of strength was evident in the shortened position. However, the extent of shortening (40° knee flexion) was not as acute as the shortened positions used in these previous studies.

5.4.2 Isometric fatigue at 40° and 80° knee flexion

Significant main effects were revealed for time ($F_{5,30} = 23.2, P < 0.001$) and angle ($F_{1,6} = 17.0, P < 0.01$) on the rate of fatigue. The results also revealed a significant interaction of time x angle ($F_{5,30} = 6.6, P < 0.001$).

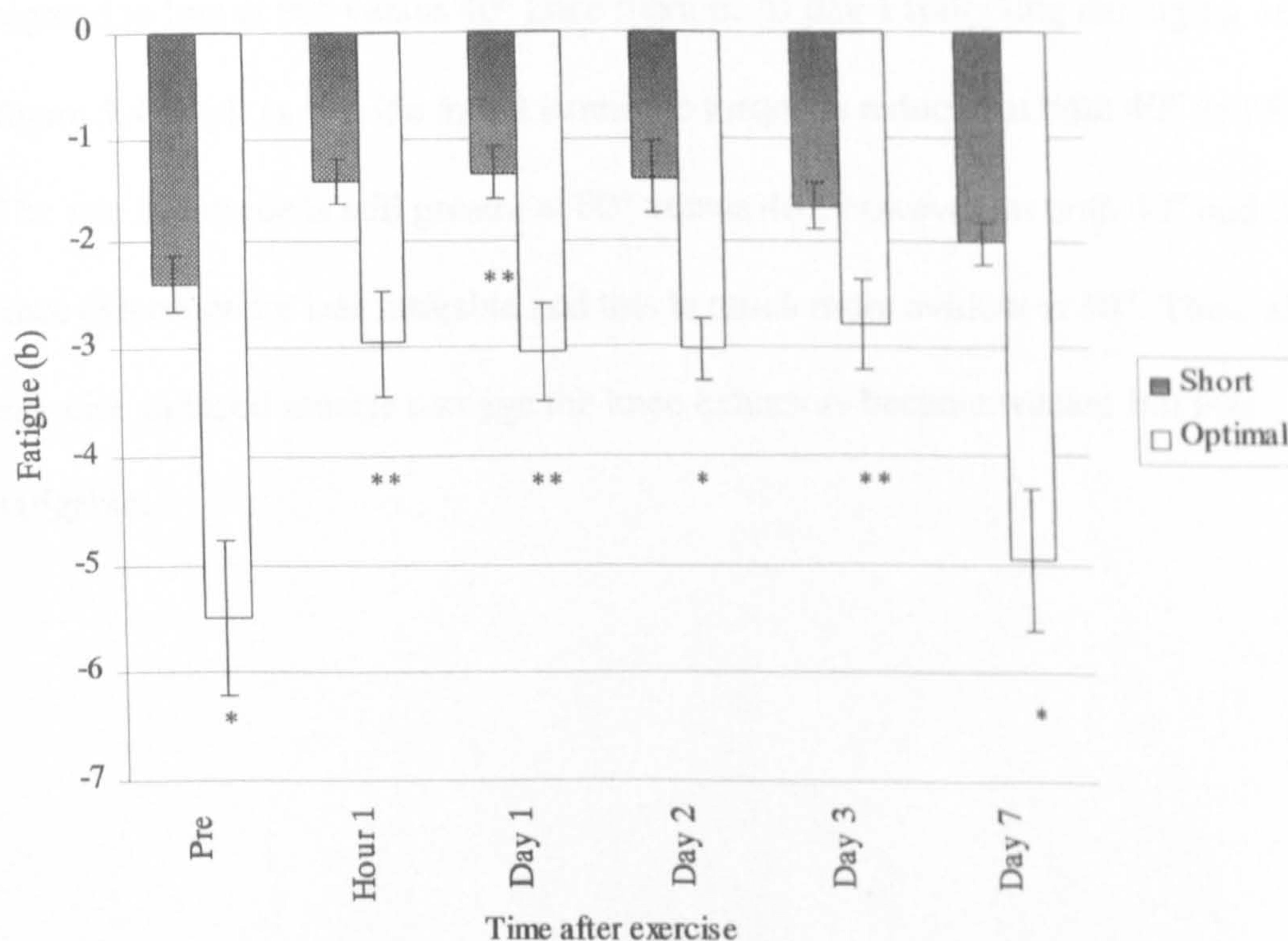


Figure 5.2: Fatigability of the knee extensors during 60s MVCs at 40° (short) and 80° (optimal) knee flexion before and after exercise-induced muscle damage. Fatigability is expressed as the regression coefficient (b), the less negative the coefficient the less fatigable the muscles. *Significantly different ($P < 0.05$) from 40°. **Significantly different ($P < 0.05$) from pre-exercise.

Prior to damaging exercise, the knee extensors were significantly less fatigable ($P < 0.05$) at 40° ($b = -2.39 \pm 0.26$) than at 80° ($b = -5.50 \pm 0.72$). Following damaging exercise the knee extensors became less fatigable, however, the magnitude and time course of this effect was dependent on joint angle (see Figure 5.2). At 80° the knee extensors were significantly less fatigable ($P < 0.05$) at hour-1, day 1, and day 3. At 40° the knee extensors were significantly less fatigable ($P < 0.05$) at day 2 only.

Figures 5.3 and 5.4 show the response of one subject to the isometric fatigue tests before damaging exercise and 1 day afterwards. Before damaging exercise, figure 5.3 displays the greater initial isometric torque and the greater rate of fatigue at optimum versus the shortened muscle length, as evidenced by the steeper slope of the regression line at 80° versus 40° knee flexion. At day 1 following damaging exercise figure 5.4 displays that the initial isometric torque is reduced at both 40° and 80° . The rate of fatigue is still greater at 80° versus 40° , however, at both 40° and 80° the knee extensors are less fatigable and this is much more evident at 80° . Thus, after exercise-induced muscle damage the knee extensors became weaker but less fatigable.

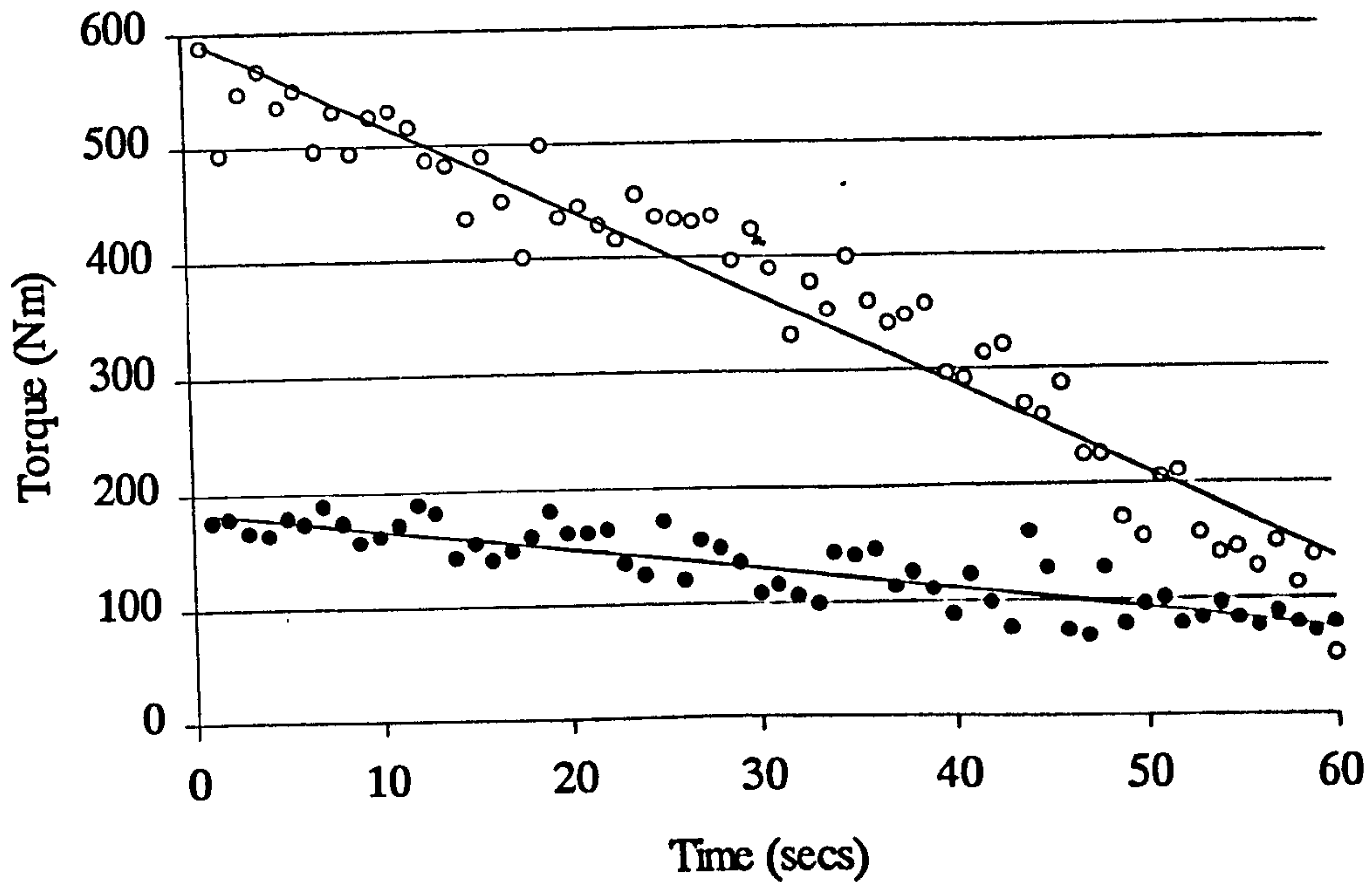


Figure 5.3: Pre-damage data from one subject (MO) showing the rate of fatigue during the 60s MVC fatigue test at 40° (filled circles) and 80° (open circles) knee flexion. Regression lines fitted to the data represent $b = -1.84$ at 40° and $b = -7.68$ at 80°.

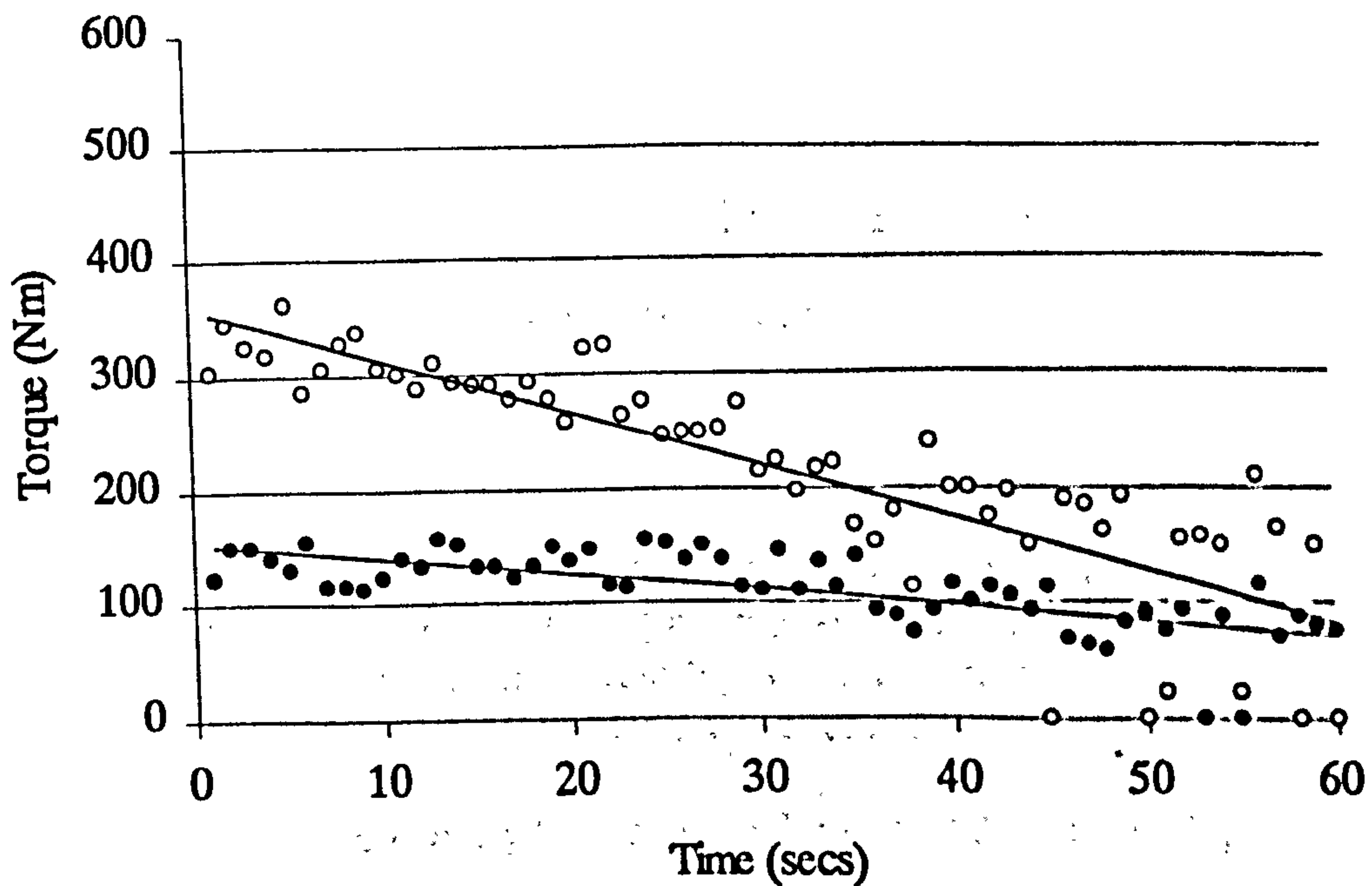


Figure 5.4: Data from one subject (MO) at day 1 after damaging exercise, showing the decline in isometric torque during the 60s fatigue test at 40° (filled circles) and 80° (open circles) knee flexion. Regression lines fitted to the data represent $b = -1.40$ at 40° and $b = -4.58$ at 80°.

5.4.3 Isometric fatigue versus dynamic fatigue

Significant main effects were revealed for time ($F_{5,30(GG)} = 11.7, P < 0.05$) and mode ($F_{1,6} = 10.4, P < 0.05$), although the interaction of time x mode was non-significant ($F_{5,30} = 1.3, P > 0.05$). Figure 5.6 and Table 5.1 illustrates the effect of muscle damage on fatigue during the 60s isometric MVC test and the Wingate power test.

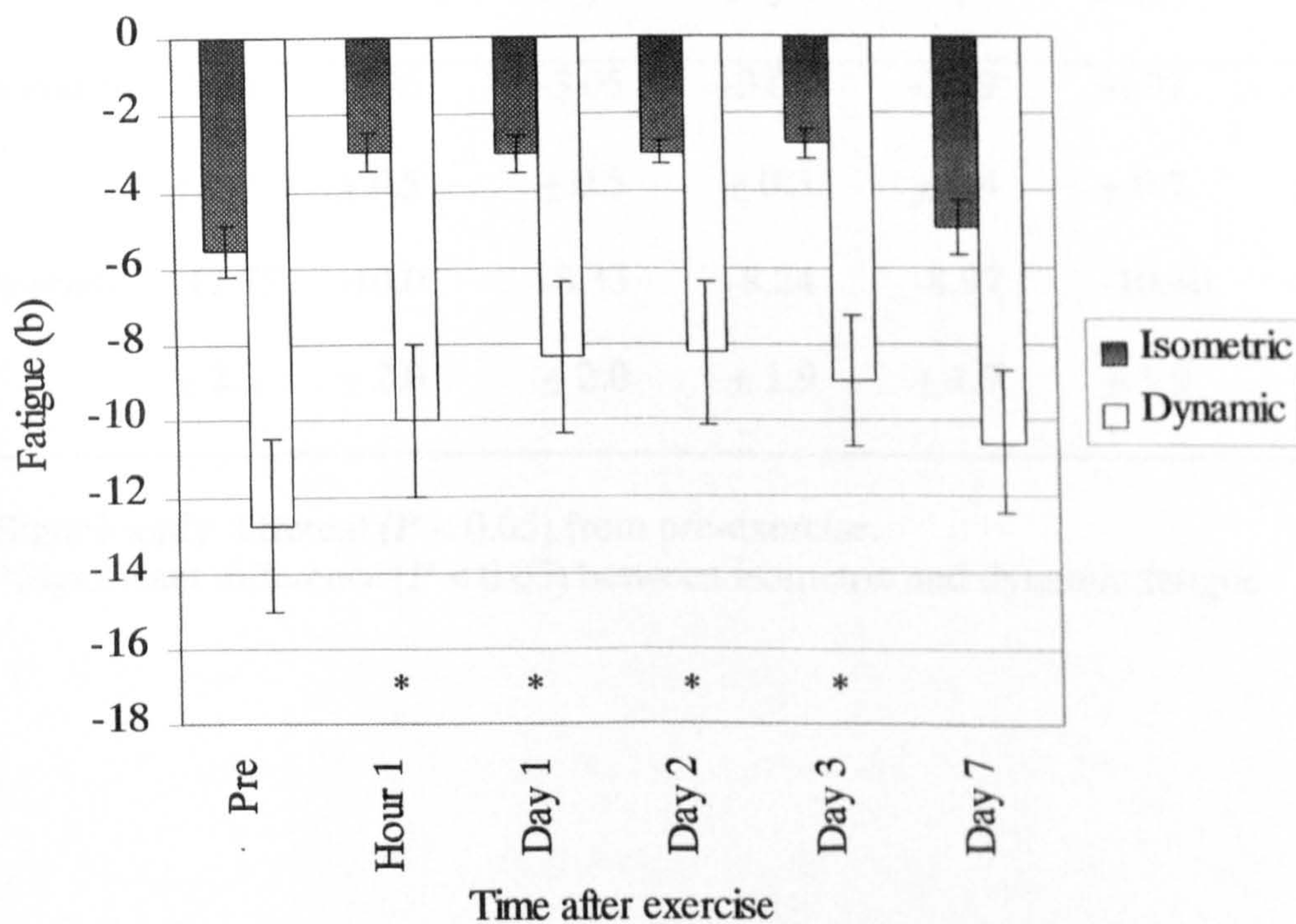


Figure 5.5: Changes in fatigability during the 60s isometric test at 80° knee flexion and the Wingate power test following exercise-induced muscle damage. Fatigue is expressed as the regression coefficient (b). Values are means (\pm SEM).

*Significantly different ($P < 0.05$) from pre-exercise.

Subjects became significantly less fatigable for over 3 days following exercise-induced muscle damage. Overall, subjects were significantly less fatigable during the isometric test ($b = -3.72 \pm 0.5$) than the Wingate test ($b = -9.81 \pm 1.9$). Due to the non-significant interaction of time x mode, the difference in fatigability during the

two exercise modes before and after muscle damage could not be determined.

However, there was a clear trend for Wingate fatigue to be much greater than

isometric fatigue before exercise and, although fatigability during both modes of

exercise reduced, this trend continued.

Table 5.1: Changes in fatigability during the 60s isometric MVC test at 80° knee flexion and the Wingate power test following exercise-induced muscle damage. Fatigue is expressed as the regression coefficient (*b*). Values are means (\pm SEM).

	<i>Pre</i>	<i>Hour 1*</i>	<i>Day 1*</i>	<i>Day 2*</i>	<i>Day 3*</i>	<i>Day 7</i>	<i>Mean**</i>
<i>Isometric</i>	-5.50	-2.96	-3.05	-3.02	-2.79	-4.97	-3.72
	± 0.7	± 0.5	± 0.5	± 0.3	± 0.4	± 0.7	± 0.5
<i>Dynamic</i>	-12.75	-10.0	-8.33	-8.24	-8.97	-10.60	-9.81
	± 2.3	± 2.0	± 2.0	± 1.9	± 1.7	± 1.9	± 1.9

*Significantly different ($P < 0.05$) from pre-exercise.

**Significant difference ($P < 0.05$) between isometric and dynamic fatigue.

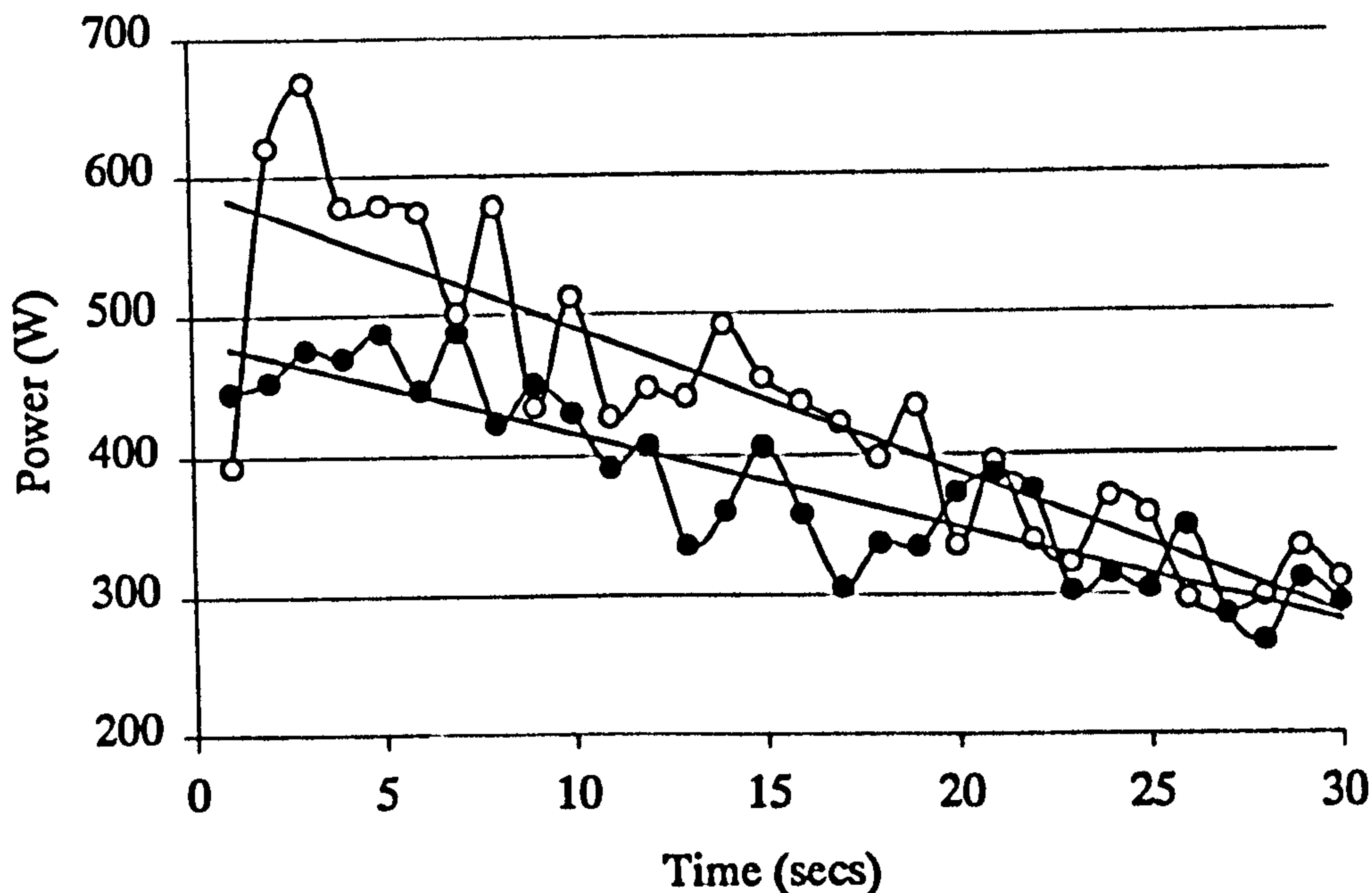


Figure 5.6: Data from one subject (AR) showing the decline in power output during the 30s Wingate test before damaging exercise (open circles) and 1 day afterwards (closed circles). Regression lines fitted to the data represent pre-damage $b = -10.24$ and day 1 post $b = -6.73$.

Figure 5.7 illustrates fatigue curves from 1 subject during the 30s Wingate test before and 1 day after damaging exercise. During the pre-exercise test there is an initial high peak in power followed by a rapid decline and then a more gradual decline. The post damage test displays a lower peak power and a more gradual decline in power, similar to the second half of the pre-exercise fatigue curve. The response to dynamic exercise was similar to isometric exercise; the damaged muscles became weaker but less fatigable. Post-Wingate blood lactate levels did not change significantly following exercise-induced muscle damage. A non-significant main effect for time ($F_{5,30} = 1.2, P > 0.05$) was revealed. Post-Wingate blood lactate values were 10.6 ± 0.6 (mean \pm SEM) before damaging exercise, 11.1 ± 1.2 at 1 hour post, 9.6 ± 1.0 at day 1 post, 9.7 ± 1.0 at day 2 post, 9.1 ± 0.7 at day 3 post, and 10.6 ± 1.2 at day 7 post.

5.4.4 Maximal strength versus maximal power

A significant main effect was revealed for time ($F_{5,30} = 20.4, P < 0.01$). Although the main effect for mode was non-significant ($F_{1,6} = 2.6, P > 0.05$) the interaction of time x mode was significant ($F_{5,30} = 4.8, P < 0.05$). Figure 5.8 and Table 5.2 illustrate the effect of muscle damage on the ability to generate isometric strength and dynamic peak power.

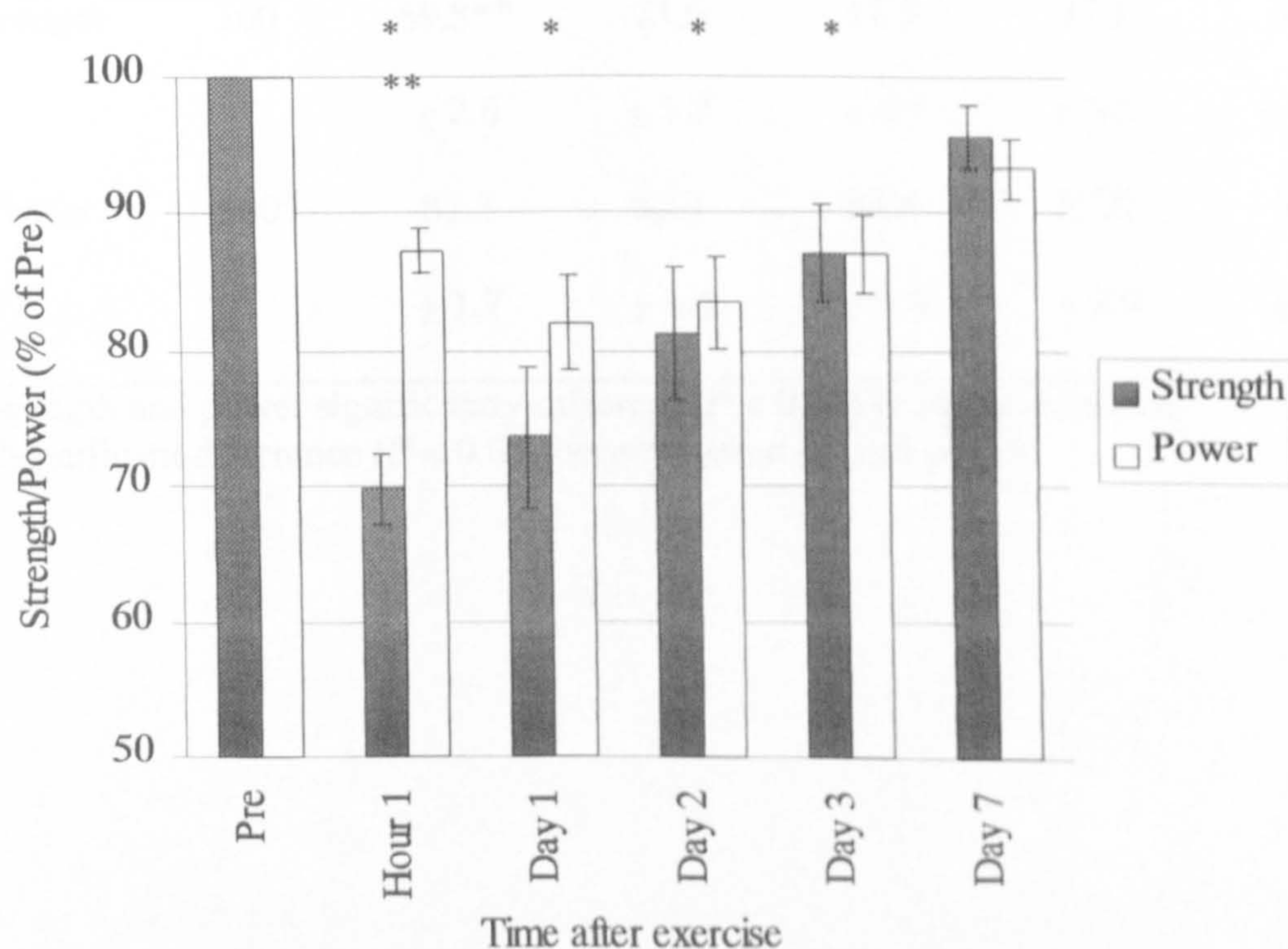


Figure 5.7: Changes in isometric strength at 80° knee flexion and Wingate peak power following exercise-induced muscle damage. Values are means (\pm SEM) expressed as a percentage of pre-exercise values. *Significantly different ($P < 0.05$) from pre-exercise. **Significant difference ($P < 0.05$) between strength and power.

The interaction of time x mode was the result of initial differences (at 1 hour post) in the effect of muscle damage on strength (30% reduction) and power (13% reduction) and subsequent differences in the pattern of recovery. Strength followed a linear

recovery pattern at day 1 (26%) and day 2 (19%), whereas power suffered further decrements at day 1 (18%) and day 2 (16%) before starting to recover.

Table 5.2: Changes in isometric strength at 80° knee flexion and Wingate peak power following exercise-induced muscle damage. Values are means (\pm SEM) expressed as a percentage of pre-exercise values.

	<i>Pre</i>	<i>Hour 1*</i>	<i>Day 1*</i>	<i>Day 2*</i>	<i>Day 3*</i>	<i>Day 7</i>
<i>Strength</i>	100	69.8**	73.6	81.3	87.1	95.6
		± 2.6	± 5.2	± 4.7	± 3.5	± 2.3
<i>Power</i>	100	87.3	82.1	83.5	87.0	93.3
		± 1.7	± 3.4	± 3.4	± 2.9	± 2.2

*Strength and power significantly different ($P < 0.05$) from pre-exercise.

**Significant difference ($P < 0.05$) between strength and power.

5.4.5 Creatine kinase activity

A significant main effect for time ($F_{5, 30}$) was revealed. Creatine kinase activity was significantly elevated above baseline at 1-hour post ($P < 0.05$) and day 1 post ($P < 0.05$).

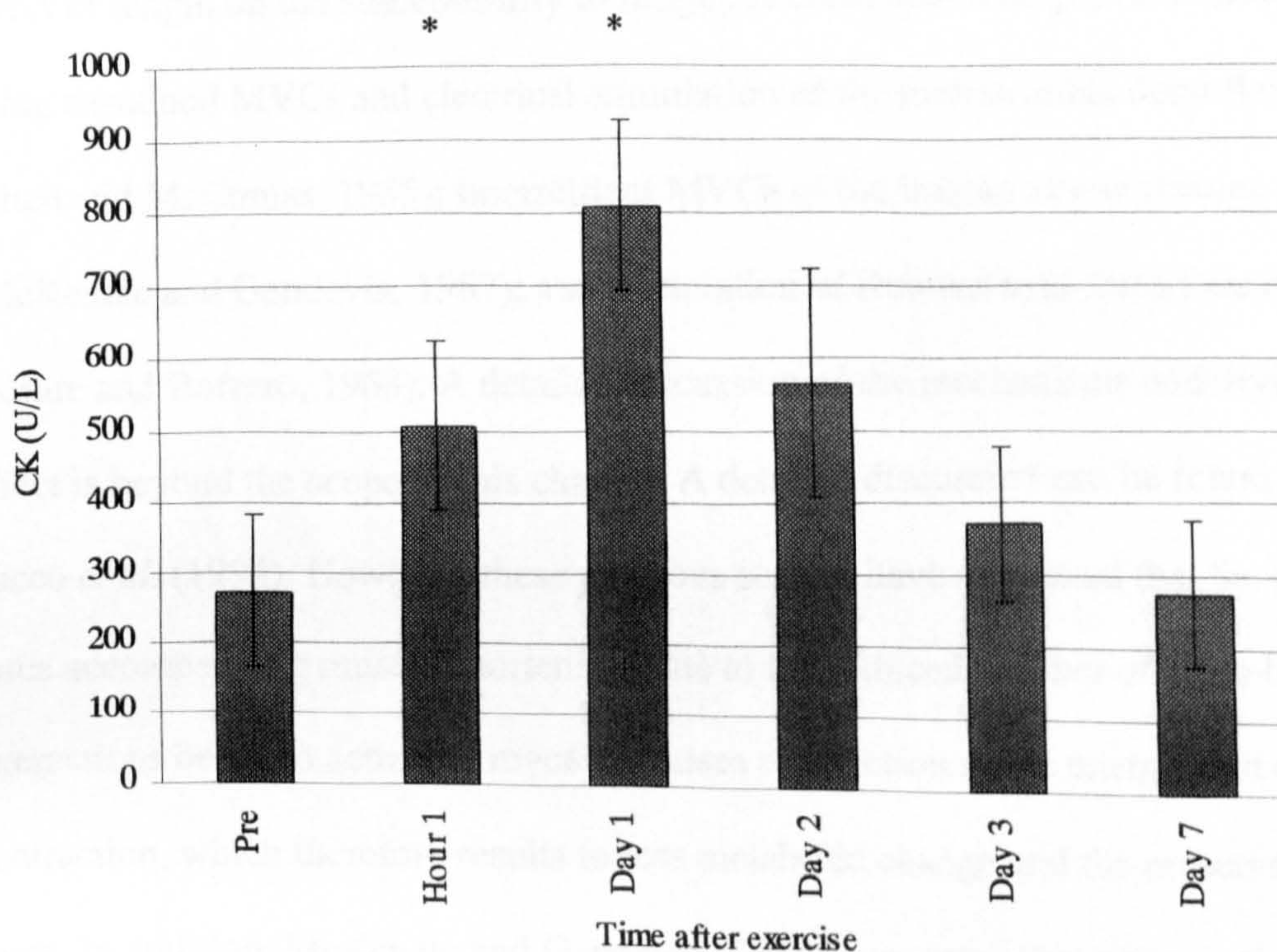


Figure 5.8: Creatine kinase activity following exercise-induced muscle damage. Values are means (\pm SEM). *Significantly different ($P < 0.05$) from pre-exercise values.

5.5 DISCUSSION

5.5.1 Observations on pre-exercise muscle function

The knee extensors were significantly less fatigable at a shortened muscle length compared to the optimal length for torque generation during a 60 second MVC. This effect of length on the susceptibility to fatigue is consistent with previous studies using sustained MVCs and electrical stimulation of the human ankle dorsi flexors (Fitch and McComas, 1985); intermittent MVCs of the human elbow flexors (McKenzie and Gandevia, 1987); and stimulation of isolated toad sartorius muscle (Aljure and Borrero, 1968). A detailed discussion of the mechanisms underlying this effect is beyond the scope of this chapter. A detailed discussion can be found in Sacco *et al.* (1994). However, these previous studies have suggested that the reduced force accompanying muscle shortening, due to the reduced number of cross-bridge interactions between actin and myosin, causes a reduction in the energy cost of contraction, which therefore results in less metabolic change and the preservation of force. In addition, McKenzie and Gandevia (1987) suggested that preservation of blood flow at short muscle lengths, due to the lower force and hence intrinsic pressure, might serve to preserve force. Gandevia and McKenzie (1988) provided evidence that full activation can be achieved at short muscle length and stated that the reduced fatigue at short length is not simply a spurious effect of incomplete activation of the motor units. More recent studies however have challenged these earlier findings and assumptions. Using ^{31}P -nuclear magnetic resonance spectroscopy techniques, no differences were reported for the energy cost of contractions at short and optimal length of the ankle dorsi flexors (Baker *et al.*, 1992; Sacco *et al.*, 1994). Additionally, Sacco *et al.*, using intermittent electrical stimulation, reported a greater rate of fatigue at short versus optimal muscle length.

A greater rate of fatigue was observed during 30 seconds of maximal cycling compared to 60 seconds of MVC. This is consistent with previous observations on maximal cycling (Sargeant *et al.*, 1981) and sustained MVCs (Bigland-Ritchie *et al.*, 1978). More recently, James *et al.* (1995) reported a 50% loss of power during electrically stimulated isokinetic contractions compared with a 25% loss for stimulated isometric contractions during 2 minutes of knee-extensor fatiguing contractions. Fatigue seems to affect the ability of a muscle to shorten and generate power to a greater extent than its ability to generate isometric force. This is possibly due to a change in the force-velocity relationship brought about by a slowing of the contractile properties of the muscle (de Haan *et al.*, 1989; Jones, 1993). This effect would result in a reduction of the maximum shortening velocity and power output, which would be proportionately greater than the loss of isometric force (de Haan *et al.*, 1989; Jones and Round, 1990; Jones, 1993; James *et al.*, 1995).

5.5.2 Evidence of muscle damage

The eccentric exercise protocol produced changes in all muscle function variables. During sustained MVCs and the Wingate test, the knee extensors were significantly less fatigable for 3 days and 7 days after eccentric exercise, respectively. Maximal isometric strength and power output were reduced by 35% and 23% at 1-hour post-exercise, respectively, and had not returned to pre-exercise values by day-7. Creatine kinase activity was significantly elevated at 1-hour after exercise, reached a peak at day-1, and then gradually returned to baseline values. The immediate and sustained loss of muscle function and the increase in blood levels of creatine kinase are common indirect markers of exercise-induced muscle damage (Warren *et al.*, 1999).

5.5.3 Muscle damage and fatigue susceptibility

As stated earlier muscle fatigue has been defined as an inability to maintain the required or expected force or power output (Edwards, 1981). The knee extensors were significantly less fatigable under isometric and dynamic conditions for over 3 days following eccentric exercise. This is in agreement with the findings of Balnave and Thompson (1993) who raised the question: how can a muscle be less fatigable at the conclusion of prolonged eccentric exercise than it was at the beginning. Research investigating fatigue mechanisms during maximal exercise and selective fibre changes occurring in eccentrically exercised muscle help to answer this question. The loss of force during isometric contractions (Soderlund *et al.*, 1992) and power output during maximal cycling (Casey *et al.*, 1996a) can be attributed to declining rates of phosphocreatine (PCr) and glycogen utilization in type II muscle fibres. Evidence suggests that type II fibres are selectively damaged during eccentric exercise (Friden *et al.*, 1983; Jones *et al.*, 1986; Lieber and Friden, 1988; Lieber and Friden, 1992; MacPherson *et al.*, 1996) and this is possibly due to selective type II recruitment. If type II fibres are fatigued, for example due to excitation-contraction coupling impairment, or if they are damaged, then the contribution of this fibre type to the post eccentric exercise fatigue curves will be diminished. As such the fatigue curves will be without the marked rise and rapid decline in force / power that is the result of the activation and rapid fatigue of type II fibres (Maughan *et al.*, 1997) and will appear less fatigable. Balnave and Thompson (1993) suggested that the fatigue curves describe the extent of muscle damage rather than the true fatigability of the muscle. The present results support the statement of Davies and White (1981) that with damaged muscle one is investigating muscle weakness rather than muscle fatigue, that is an inability to generate, rather than maintain, force.

Linear regression analysis was used to quantify fatigue since this method takes into account all of the data points rather than a simple percentage decline based on the highest and lowest data points. However, linear regression analysis may have been inappropriately applied to the Wingate data, where a curvilinear relationship appeared to exist (see Figure 5.6, page 99). A more appropriate statistical model may have been the use of trend analysis and curvilinear regression. It appears that various methods of quantifying fatigue have been used in the muscle function literature and at present there appears to be no standard or widely accepted method.

One aspect of muscle damage that might be expected to make muscle more fatigable is that it impairs the repletion of muscle glycogen (O'Reilly *et al.*, 1987; Van der Meulen *et al.*, 1992; Hesselink *et al.*, 1998). Low muscle glycogen levels have been reported to significantly reduce endurance time during a sustained isometric contraction at 60% of MVC (Maughan, 1988). In the present study fatigability was not assessed by endurance time, however, it would be interesting to investigate this aspect of fatigue in damaged muscle. Following a marathon run, Nicol *et al.* (1991) reported a 39% reduction in endurance time during a sustained isometric contraction at 60% of pre-marathon MVC. The greater neural activation (EMG) required to achieve a given sub-maximal force after damaging exercise (Komi and Viitasalo, 1979; Newham *et al.*, 1983) would be expected to increase the perception of effort during exercise and reduce endurance time.

5.5.4 Muscle damage and maximal strength and power

The results revealed a reduced ability of damaged muscle to generate isometric strength and power output. At 1-hour post exercise, the ability to generate isometric

strength was affected to a greater extent ($69.8 \pm 2.6\%$ of pre-exercise) than power output ($87.3 \pm 1.7\%$). The two parameters then followed different temporal patterns of recovery; strength recovering linearly, whereas power declined further at days 1 and 2 before starting to recover. Power eventually recovered with a similar time course to that of isometric strength. It is not surprising that power was affected to a lesser extent than strength, since additional, non-damaged muscle groups other than the knee extensors, are involved in the generation of power output during cycling. However, the different temporal pattern of recovery may indicate that exercise-induced muscle damage affects isometric strength and dynamic power output to different extents. The difference might result from peripheral changes or an inability to maintain central motor drive during complex motor tasks. Reductions in muscle glycogen and high-energy phosphate levels have been reported to occur 24 hours after eccentric exercise (Van der Meulen *et al.*, 1992; Hesselink *et al.*, 1998). During maximal isometric and dynamic muscle actions the energy demand will be being met by anaerobic utilization of PCr and glycogen. However, the generation of power output is more energetically demanding than isometric force generation (Woledge, 1985) and changes in energy metabolism are more easily detected by shortening compared with isometric actions (Vollestad, 1997). An impairment of metabolism could result in a change in the force-velocity relationship, as seen with non-damaging fatiguing exercise (de Haan *et al.*, 1989; Jones, 1993), which could serve to reduce maximum power output to a greater extent than isometric force. No changes in post-Wingate blood lactate levels were observed in this study, indicating that anaerobic glycogen metabolism was not impaired. It is possible that reduced levels of ATP and PCr in the days after damaging exercise affected the ability to generate power output to a greater extent than isometric force.

Alternatively, there may have been a reduction in central drive, resulting from voluntary conscious or subconscious reflex mechanisms. The reductions in power following the initial (1-hour) decrement occurred at a time when muscle soreness was present (days 1 and 2). Sargeant and Dolan (1987) also observed further reductions in power output in the days following the initial decrement. Voluntary effort may have been reduced at these times due to sensations of soreness, or alternatively, afferent signals from the working muscles and tendons may have produced reflex inhibition. An additional factor to consider is that motor control is impaired following muscle damage (Pearce *et al.*, 1998) and this would be expected to affect a complex dynamic motor task such as cycling, to a greater extent than a simple static task such as knee extension.

5.5.5 Summary

In fresh muscle a length-dependent effect of fatigue susceptibility was observed during sustained maximal isometric contractions. Damaged muscle became weaker but less fatigable under isometric and dynamic conditions. This suggests an inability to generate rather than maintain force and power. Maximal strength and power, although sharing a similar time course of recovery, differed in their pattern of recovery. Strength recovered linearly after the initial insult whereas power declined further in the following days before starting to recover.

CHAPTER 6

CONCLUSIONS

- 6.1 New contributions to the literature**
 - 6.1.1 Length-dependence of isometric strength loss**
 - 6.1.2 Dynamic performance with and without use of SSC**
 - 6.1.3 Length-dependence of knee extensor fatigue**
 - 6.1.4 Isometric and dynamic fatigue of damaged muscle**
 - 6.1.5 Isometric strength and power generating ability of damaged muscle**

- 6.2 Study limitations**
 - 6.2.1 Sample size, effect size and statistical power**
 - 6.2.2 Non-determination of the sites and mechanisms of muscle function impairment**

- 6.3 Future directions**
 - 6.3.1 The angle-torque relationship after muscle damage**
 - 6.3.2 The torque-velocity relationship after muscle damage**
 - 6.3.3 Dynamic performance measures after muscle damage**
 - 6.3.4 Fatigability after muscle damage**
 - 6.3.5 Creatine supplementation and muscle function after damage**

6.1 NEW CONTRIBUTIONS TO THE LITERATURE

6.1.1 Length-dependence of isometric strength loss (chapter 3)

Measures of isometric strength have provided the primary means of determining muscle function after exercise-induced muscle damage. There is typically an immediate and prolonged reduction in isometric strength after damaging exercise. Strength has traditionally been measured at a single joint angle or muscle length. However, recent evidence suggests that the optimal joint angle or muscle length for the generation of strength is shifted towards longer muscle lengths, due to a shift to the right of the length-tension curve, after eccentric exercise (Wood *et al.*, 1993; Jones *et al.*, 1997; Talbot and Morgan, 1998; Whitehead *et al.*, 1998). The functional consequence of a shift in the length-tension curve is that strength loss will be affected by the muscle length at which it is measured, being disproportionately reduced at short muscle lengths versus optimal or long lengths. Indeed, Saxton and Donnelly (1996) reported that elbow flexor strength was disproportionately reduced at short muscle length versus long muscle length for over four days after 70 maximal eccentric actions.

In chapter 3 it was reported that after 100 maximal eccentric actions of the knee extensors, isometric strength was disproportionately reduced ($P < 0.05$) at short muscle length ($76.3 \pm 2.5\%$ of pre-exercise values) compared to optimal length ($82.1 \pm 2.7\%$). These values represent the average decline in strength for the seven-day assessment period. There was a clear trend for strength at short muscle length to be disproportionately reduced throughout the seven-day period, although this could not be determined statistically. Both the popping sarcomere hypothesis and impaired excitation-contraction coupling could explain the

present results since they would both be expected to affect strength in a way that is dependent on muscle length.

6.1.2 Dynamic performance with and without use of SSC (chapter 4)

Dynamic performance measures have very rarely been used in the assessment of muscle function after exercise-induced muscle damage. This is surprising since measures of isometric strength may not give an accurate reflection of performance impairment in dynamic movements that are used in sporting activities. Vertical jumping offers a convenient way to study dynamic performance with and without use of the stretch-shortening cycle (SSC). The SSC is a natural form of muscle function that occurs in a sequence of eccentric stretch, isometric coupling, and concentric shortening, and allows the final concentric action to take place with greater force or power output compared to the condition where the movement is initiated by a concentric action alone (Komi, 1984). A higher performance in vertical jumping can be achieved when the SSC is utilised in performance, for example, the greater jump height achieved during a countermovement jump (with SSC) compared to a squat jump (without SSC) (Asmussen and Bonde-Petersen, 1974; Komi and Bosco, 1978; Bobbert *et al.*, 1996). The drop jump, which involves dropping from a specified height and rebounding vertically for maximal height also utilises the SSC. The response of these three jump types to exercise-induced muscle damage had not been studied previously.

In chapter 4 it was reported that squat jump performance was affected to a greater extent than countermovement jump ($91.6 \pm 1.1\%$ vs. $95.2 \pm 1.3\%$, $P <$

0.05) and drop jump performance ($91.6 \pm 1.1\%$ vs. $95.2 \pm 1.4\%$, $P < 0.05$) over the seven-day assessment period. The results suggest that the detrimental performance effects of exercise-induced muscle damage are attenuated when the SSC is utilised in performance.

6.1.3 Length-dependence of knee extensor fatigue (chapter 5)

The ability of muscle to maintain force is enhanced when it is tested at short compared to optimal muscle length. This reduced fatigability has been demonstrated in isolated toad sartorius muscle (Aljure and Borrero, 1968), human ankle dorsiflexors (Fitch and McComas, 1985), human inspiratory muscles (McKenzie and Gandevia, 1987), and human elbow flexors (McKenzie and Gandevia, 1987). In chapter 5 it was reported that the human knee extensors were significantly less fatigable at a shortened length compared to optimal length for torque generation during a sustained 60s isometric MVC. Torque was measured every second and the slope of a linear regression line (b) was fitted to the 60 torque points. A greater rate of fatigue was observed at the optimal muscle length versus the shortened length ($b = -5.50 \pm 0.72$ vs. -2.39 ± 0.26 , $P < 0.05$).

6.1.4 Isometric and dynamic fatigue of damaged muscle (chapter 5)

The fatigability of damaged muscle had only previously been assessed by electrically elicited isometric fatigue tests consisting of trains of stimuli at 20 Hz, lasting 300 ms and repeated every second for two minutes (Davies and White, 1981; Balnave and Thompson, 1993). The fatigability of damaged muscle during voluntary isometric muscle actions and during dynamic performance was compared in chapter 5. The rate of fatigue was assessed during a 60s sustained

isometric MVC at optimal muscle length and during a 30s maximal cycle ergometer test by fitting a linear regression line to the 60 torque points of the MVC and the 30 power output points of the cycle test. Prior to damaging exercise, subjects were less fatigable during the MVC compared to the cycle test ($b = -5.50 \pm 0.7$ vs. -12.75 ± 2.3) and this trend continued after the damaging exercise. Over the seven-day assessment period subjects were significantly less fatigable during the MVC compared to the cycle test ($b = -3.72 \pm 0.5$ vs. -9.81 ± 1.9 , $P < 0.05$). During both exercise modalities, subjects became weaker and less fatigable after damaging exercise. The pattern of change in fatigability did not differ between the two exercise modes. The results of chapter 5 indicate that damaged muscle has an impaired ability to generate, rather than maintain, torque / power output.

6.1.5 Isometric strength and power generating ability of damaged muscle (chapter 5)

Only one study had previously investigated the ability of damaged muscle to generate power output. Sargeant and Dolan (1987) using a very small sample size ($n = 4$) reported an immediate, significant, and sustained reduction in short term power output, measured concentrically on an isokinetic cycle ergometer. The authors commented that reductions in power were accompanied by reductions in isometric strength of a similar magnitude but no comparison of the ability of damaged muscle to generate maximal isometric strength and power output was made. Such a comparison was made in chapter 5. The results of which revealed that maximal isometric strength and power output followed different temporal patterns of recovery after the initial eccentric exercise-induced

insult. Strength recovered linearly from 1-hour post exercise, whereas power output declined further at days 1 and 2 before starting to recover. Power output eventually recovered with a similar time course to that of isometric strength.

6.2 STUDY LIMITATIONS

6.2.1 Statistical power, sample size and effect size

Statistical power refers to the probability of the study leading to significant results, that is rejection of the null hypothesis (Cohen, 1992). Huck (2000) stated that an inverse relationship exists between sample size and the probability of a type II error (failing to reject a false null hypothesis). That is, if power is high, the chances of not rejecting a false null hypothesis are low, whereas if power is low, the chances of reaching a fail to reject decision are high. There is also a direct relationship between sample size and the probability of rejecting a false null hypothesis. That is, a study with an insufficient sample size will probably lead to a fail to reject decision. Therefore it is desirable for researchers to use sample sizes that are large enough to give their statistical tests adequate power to detect important and noteworthy deviations from the null hypothesis and make it unlikely that an important finding is missed due to a type II error.

In chapter 3 a main effect for joint angle (muscle length) was reported. Isometric strength was disproportionately reduced ($P < 0.05$) at short muscle length ($76.3 \pm 2.5\%$ of pre-exercise values) compared to optimal length ($82.1 \pm 2.7\%$) over the seven-day testing period. A trend was observed for strength at short muscle length to be disproportionately reduced throughout the seven-day testing period. However, due to the interaction of time x angle only approaching significance

($P = 0.07$) it was not possible to conduct follow-up tests to determine at which time points, if any, significant differences in strength loss existed between short and optimal muscle lengths. The effect size for the interaction was 0.24 and the power of the test was 0.66. According to Cohen (1992) this corresponds to a small effect size and the smaller the effect size, the larger the sample size necessary if the study is to have sufficient power to detect that effect size. It is possible that the sample size ($n = 8$) did not afford sufficient power to detect the effect size and allow for the determination of potential differences in strength loss at the various time points after eccentric exercise.

6.2.2 Non-determination of the sites and mechanisms of muscle function impairment

When using voluntary muscle actions, the performance output is the integrated result of the total chain of events leading to muscle force production. The immediate and prolonged impairment of force production after exercise-induced muscle damage could result from a failure of any one link or site in the chain. Techniques such as superimposed electrical stimulation and EMG can be used to categorise the mechanisms of force loss into central or peripheral factors, depending on whether the site is proximal or distal to the neuromuscular junction. However, the aim of the thesis was not to determine the sites and mechanisms of muscle function impairment after muscle damage and thus the role of possible sites and mechanisms have merely been speculated. Vollestad (1997) suggested that voluntary muscle actions can serve as the first choice of methods, before additional investigations are added to examine possible sites and mechanisms of muscle function impairment in more detail.

6.3 FUTURE DIRECTIONS

6.3.1 The angle-torque relationship after eccentric exercise

The popping sarcomere hypothesis predicts a shift in the length-tension relationship of muscle after eccentric exercise so that the optimum muscle length or the optimal angle for the generation of force occurs at a longer muscle length. Direct evidence of a shift in the optimal angle has been reported in isolated toad sartorius muscle (Wood *et al.*, 1993; Talbot and Morgan, 1998) and human ankle extensors (Jones *et al.*, 1997; Whitehead *et al.*, 1998). Such a shift has not been demonstrated in other muscle groups commonly used in muscle damage research such as, the knee extensors and elbow flexors. Demonstration of a shift in these muscle groups would require the angle-torque relationship to be studied in detail (torque measured at small angle increments) and preferably with the use of tetanic stimulation. There is uncertainty regarding the time course of the shift, whether it demonstrates a reversal after 1-2 days or persists for as long as the loss of strength.

6.3.2 The torque-velocity relationship after muscle damage

Differing reports have emerged from the literature regarding the effect of muscle damage on the torque-velocity relationship. Strength loss and recovery has been shown to be dependent on the muscle action being performed (Golden and Dudley, 1992) and the angular velocity of movement (Friden *et al.*, 1983; Golden and Dudley, 1992; Eston *et al.*, 1996). In contrast, strength loss and recovery has been shown to be independent of the muscle action being performed (Hortobagyi *et al.*, 1998; Chapter 4) and the angular velocity of movement (Chapter 3). None of these studies however, determined if subjects achieved full voluntary

activation during dynamic strength testing. It is possible that the ability to achieve full voluntary activation is reduced as the complexity and velocity of the task increase. The only way to determine this is by applying the methods used to determine voluntary activation during isometric actions to dynamic actions. That is, the use of superimposed electrical stimulation on voluntary dynamic actions.

Newham *et al.* (1991) superimposed tetanic stimulation (100 Hz) for 250 ms on knee extensor isometric and isokinetic concentric actions at 20 and 150 deg·s⁻¹, ranging from 5-100% of maximum torque. The inverse linear relationship between the additional torque generated by the stimulation and level of voluntary activation was similar for all three conditions and similar to that observed when single electrical impulses are superimposed on isometric actions (Rutherford *et al.*, 1986). When muscle actions were truly maximal, no additional torque was generated under isometric and concentric conditions. Newham *et al.* (1991) assessed voluntary activation before and after 4 minutes of fatiguing exercise (maximal concentric knee extensions). Before fatigue there was little evidence of incomplete activation, whereas after fatigue all subjects showed evidence of incomplete activation during both isometric and concentric actions at 20 deg·s⁻¹, but not during concentric actions at the higher velocity of 150 deg·s⁻¹. Thus, superimposed electrical stimulation can be used to highlight the role of central factors in strength loss for both isometric and concentric actions. Use of the technique in the study of the concentric portion of the torque-velocity relationship would allow for the determination of central and peripheral factors in the immediate and prolonged loss of strength after muscle damage.

Furthermore, the level of voluntary activation achieved at different angular

velocities would also highlight any differences in central and peripheral contributions to strength loss across angular velocities.

Superimposed tetanic stimulation has also been used to assess voluntary activation during maximal eccentric actions at 60, 180, and 360 deg·s⁻¹ (Westing *et al.*, 1990). The authors reported that superimposed stimulation increased torque above voluntary levels for maximal eccentric actions but not for isometric or concentric actions. This suggests that maximal voluntary eccentric torque does not reflect the true maximal torque-producing ability of the muscle. It appears that a protective neural force-limiting mechanism is active to prevent full voluntary activation. It is not known what effect exercise-induced muscle damage has on the level of voluntary activation that can be achieved during eccentric strength testing. The application of superimposed tetanic stimulation to maximal eccentric actions across a range of velocities would resolve this issue.

An alternative to the use of electrical stimulation superimposed on voluntary actions is the use of artificial activation (by tetanic stimulation) to study the torque-velocity relationship. Although the voluntary torque-velocity relationship deviates from the classical force-velocity relationship, due to inhibition of eccentric and slow concentric torque, this is not the case when the muscles are artificially activated (Dudley *et al.*, 1990). Dudley *et al.* (1990) reported that the artificially activated torque-velocity relationship of the knee extensors was remarkably similar to that of isolated muscle, with torque changing three times more across concentric and eccentric angular velocities under artificial versus voluntary conditions. Artificial stimulation would prove extremely useful in

providing information on the inherent torque producing ability of damaged muscle across angular velocities and muscle actions.

6.3.3 Dynamic performance measures after muscle damage

One hypothesis for the enhanced performance of skeletal muscle during stretch-shortening movements is that the pre-stretch allows for the attainment of a higher active muscle state and a corresponding higher level of force prior to the start of the concentric action. In chapter 4 it was reported that vertical jumping performance was affected to a lesser extent by exercise-induced muscle damage under conditions of countermovement and drop jump compared to squat jump, and it was speculated that this was due to use of the SSC in the former two jumps. If the attainment of a higher active muscle state and force is responsible for performance potentiation in SSC movements and the results observed in chapter 4, then as long as the starting force prior to a concentric action is equal, the type of muscle action preceding the concentric action should be irrelevant. That is, concentric performance should not be dependent on whether it is preceded by an isometric or eccentric action. Isokinetic dynamometry allows for concentric muscle actions to be preceded by either an isometric pre-load or an eccentric action. Studies using this technique have reported greater torque output for concentric actions preceded by isometric or eccentric actions compared to pure concentric actions (Jensen *et al.*, 1991; Svantesson *et al.*, 1994). It would be interesting to compare isokinetic performance before and after exercise-induced muscle damage under the three conditions of:

- i) Concentric only
- ii) Concentric preceded by isometric pre-load
- iii) Concentric preceded by eccentric action

(with isometric pre-load equalling the torque at the start of the concentric phase of the eccentric-concentric action)

Such a study would have the potential to determine whether the generation of a high active muscle state and/or muscle stretch potentiate muscle performance and if these factors have a role to play in reducing the impairment of muscle function after exercise-induced muscle damage. A study examining these three conditions in the fresh state, using isokinetic concentric barbell squats, reported a greater work output for both the concentric isometric pre-load and eccentric-concentric conditions over the pure concentric condition (Walshe *et al.*, 1998). Additionally, the authors reported greater work output for eccentric-concentric versus concentric isometric pre-load condition, suggesting that the type of muscle action preceding the concentric action is an important determinant of performance and not just the attainment of a high active muscle state and force.

In chapter 5 it was reported that the ability to generate maximal isometric strength and power output differed in their pattern of recovery after exercise-induced muscle damage. Maximal power output declined further in the days after eccentric exercise whereas isometric strength followed a linear recovery pattern. Superimposed electrical stimulation has recently been applied to study the extent of voluntary activation in the knee extensors during dynamic isokinetic cycling before and after fatigue (Beelen *et al.*, 1995). A similar technique must be

applied to the study of power output before and after muscle damage if we are to elucidate the mechanisms responsible for the immediate and prolonged loss of maximal power after damaging exercise.

6.3.5 Fatigability after muscle damage

In chapter 5 it was reported that damaged muscle had an improved ability to maintain maximal isometric torque. This unlikely finding was most likely due to an inability to generate a high torque at the start of the fatigue test because of the reduction in maximal isometric strength. Perhaps a clearer insight into the ability of damaged muscle could be gained by comparing the rate of fatigue or endurance time during the maintenance of absolute torque levels and torque levels relative to the current MVC. It would be expected that after exercise-induced muscle damage the ability to maintain an absolute torque level will be decreased due to the loss of maximum isometric strength. However, it is uncertain whether similar results would be gained during the maintenance of relative torque.

6.3.6 Creatine supplementation and muscle function after damage

Due to the discrepancy between the magnitude of ultrastructural muscle damage and force loss after eccentric exercise, some authors have speculated that impaired metabolism may contribute to the loss of muscle function (Hesselink *et al.*, 1996). In addition to the impaired muscle glycogen resynthesis observed after eccentric exercise it has been reported that levels of high-energy phosphates (ATP and PCr) are reduced by eccentric exercise and are not restored to control levels 24 hours afterwards (Van der Meulen *et al.*, 1992; Hesselink *et al.*, 1998).

Considering that PCr availability is thought to limit maximal exercise performance, a logical inference is that elevating the level of PCr prior to eccentric exercise may preserve muscle function after eccentric exercise.

Creatine (Cr) supplementation has been reported to increase the muscle content of Cr and the content in the form of PCr (Harris *et al.*, 1992). In addition, creatine supplementation has been shown to improve performance during repeated bouts of maximal isokinetic cycling (Birch *et al.*, 1994; Casey *et al.*, 1996b). Repeated maximal exercise performance is thought to be improved by an enhanced PCr resynthesis during exercise and recovery (Greenhaff *et al.*, 1994). It remains to be seen whether creatine supplementation prior to exercise-induced muscle damage can help to preserve muscle function after damage.

CHAPTER 7

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