

THE EFFECT OF MUSCLE-DAMAGING EXERCISE ON MAXIMAL INTENSITY CYCLING AND DROP JUMP PERFORMANCE

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The purpose of this study was to assess the effects of muscle-damaging exercise on the time to peak power during maximal intensity cycling and drop jump performance. Nineteen subjects were randomly assigned to a treatment ($n=10$, age 22.6 ± 2.8 years, body mass 70.7 ± 12.9 kg, stature 1.76 ± 0.10 m) or control group ($n=9$, age 20.8 ± 2.5 years, body mass 74.2 ± 10.2 kg, stature 1.76 ± 0.07 m) and randomly performed a 10-second cycle ergometer sprint, against a load corresponding to $0.70 \text{ N} \cdot \text{m} \cdot \text{kg}^{-1}$ for males and $0.67 \text{ N} \cdot \text{m} \cdot \text{kg}^{-1}$ for females, and a drop jump from a height of 50 cm. Indirect markers of muscle damage (perceived muscle soreness and isokinetic knee extensor torque at 60 and 360 $\text{deg} \cdot \text{s}^{-1}$) were also assessed. All measures were taken before and at 24, 48, 72 and 168 hours following a plyometric exercise protocol comprised of 10×10 maximal countermovement jumps. Repeated measures ANOVA showed significant interactions of time by group on all dependent measures ($p < 0.05$). Peak power output, time to peak power output, drop jump height, ground contact time during the drop jump, and strength at both joint angular velocities were all adversely affected in the treatment group, with no significant changes in the control group. These results provide further evidence that dynamic muscle performance is compromised in the days following muscle-damaging exercise.

Keywords: exercise-induced muscle damage, peak power, peak torque

Introduction

Eccentric-biased exercise has been shown to result in symptoms of muscle damage, including muscle soreness (Newham et al. 1987), reduced range of motion (Cleak & Eston 1992), and losses in force-generating capability (Clarkson et al. 1992). It is the loss in force

generation that potentially has the most significant consequences for athletic performance.

Several studies have demonstrated an immediate and prolonged reduction in peak power output during maximal intensity cycling (Twist & Eston 2005; Byrne & Eston 2002b; Sargeant & Dolan 1987) and impairment of vertical jump performance (Byrne & Eston 2002b; Avela et al. 1999; Horita et al. 1999) following muscle-damaging exercise. However, although these modes of assessing dynamic muscle function are well-accepted, it may be that other indices are of greater significance to athletic performance. The ability to generate force rapidly is an inherent characteristic required in many

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athletic pursuits, and is often of greater significance to athletic performance than maximal strength. This has been suggested because in many activities the contact time for force application is relatively short (<200 ms) in comparison to the time taken to attain peak force (>300 ms) (Zatsiorsky 1996). As such, the maximal rate of force development (RFD) has been proposed as a measure of neuromuscular performance that correlates strongly with more performance-related measures (Young et al. 1995), and has consequently been shown to be impaired following muscle-damaging exercise (Minshull et al. 2007; Miles et al. 1997). Nonetheless, the relevance of RFD in assessing athletic performance is often criticized due to the low external validity associated with isometric dynamometry (Wilson & Murphy 1996).

More recently, the time to peak power during maximal intensity cycling has been proposed as a more ecologically relevant measure of athletic performance (Bell & Cobner 2007; Winter & MacLaren 2001). However, this parameter of maximal intensity cycling is not frequently reported and, as far as the authors are aware, has not been investigated following muscle-damaging exercise. Similarly, the stretch-shortening cycle provides a sound model to study neuromuscular function (Komi 2000) and several authors have reported the impairment of vertical jump performance following muscle-damaging exercise, and in particular the increase in contact time during drop jump procedures (Byrne & Eston 2002a; Avela et al. 1999; Horita et al. 1999). Such alterations are associated with decreased strength, reflex activity and initial stiffness (Avela et al. 1999; Horita et al. 1999). Kraemer and Newton (1994) proposed that the contact time between the landing and take-off phases of a drop jump is a marker of the maximal rate at which the muscle can generate force.

Although the general consensus is that exercise-induced muscle damage results in a decrease in torque-generating capability, findings are equivocal in relation to its effect on the torque-angular velocity relationship of muscle. Using isokinetic dynamometry, several studies have reported that there is no velocity-dependent reduction in strength-generating capability (Byrne et al. 2001; Sherman et al. 1984). In contrast, some studies have observed a faster recovery of strength at faster velocities (Michaut et al. 2002; Deschenes et al. 2000),

while others have suggested that slower velocities of movement show a quicker return to baseline values (Golden & Dudley 1992; Fridén et al. 1983). However, few studies have considered the assessment of angular velocities which are ecologically more consistent with those posed in athletic activities. Therefore, the purpose of this study was to investigate the effects of muscle-damaging exercise on the time to peak power during maximal intensity cycling and drop jump performance.

We hypothesized that after a bout of rapid lengthening of the antigravity musculature (plyometric jumps), the resultant exercise-induced muscle damage of the knee and hip extensors would impair the ability of those muscles to generate peak force and reduce the rate at which this force could be generated.

Methods

Participants and experimental design

A sample of healthy participants (12 males, 7 females) who participated in recreational level team sports but who had not participated in any resistance training of the lower limbs within 6 months prior to the assessment were randomly assigned to a treatment ($n=10$, age 22.6 ± 2.8 years, body mass 70.7 ± 12.9 kg, stature 1.76 ± 0.10 m) or control group ($n=9$, age 20.8 ± 2.5 years, body mass 74.2 ± 10.2 kg, stature 1.76 ± 0.07 m). The study was approved by the research ethics committee of the School of Health and Applied Sciences, University of Chester, before starting the study. All risks associated with the experimental procedures were explained prior to involvement in the study and each participant was asked to complete a written informed consent form before assessment.

One week before commencing the study, all participants attended familiarization trials for maximal intensity cycling, isokinetic strength and drop jump procedures. Following the familiarization procedures, the treatment group was evaluated for each of the specific measures before and then 24, 48, 72 and 168 hours following a bout of plyometric exercise designed to bring about the symptoms of exercise-induced muscle damage. The control group underwent the same measures over the same time period, without the bout of plyometric

exercise. Each subject performed the criterion measures in a random order with an appropriate and standardized recovery between each to allow optimal recovery.

Maximal intensity exercise performance

An electronically braked cycle ergometer (Lode Excalibur Sport, Lode Medical Technology, Groningen, The Netherlands) was used to assess instantaneous power output corrected for flywheel acceleration. A 5-minute cycle at 100 W with a flat-out sprint at 3 minutes followed by a 5-minute passive recovery including stretching exercises served as a warm-up. Participants were required to perform a 10-second maximal effort sprint with prior acceleration of the limbs, against a load corresponding to $0.70 \text{ N} \cdot \text{m} \cdot \text{kg}^{-1}$ for males and $0.67 \text{ N} \cdot \text{m} \cdot \text{kg}^{-1}$ for females (Lode Medical Technology). The test commenced with a rolling start where the participant cycled at a constant load of 50 W for 30 seconds. The experimenter provided a 3-second countdown at the end of the rolling start, after which the load was automatically applied by the software (Lode Medical Technology) and the subject accelerated as quickly as possible and maintained a maximal effort for 10 seconds. Data were recorded every 0.2 seconds over the 10 seconds of effort, with the maximum value being used to reflect peak power output (PPO). Time to peak power was also recorded to the nearest 0.2 seconds. As a rolling start may cause difficulty in precisely identifying the start of the 10-second sprint (Winter & MacLaren 2001), and hence accurately measuring time to peak power, the onset of the power generation was identified visually as the time at which power output first deviated above the rolling start value. Recorded times were then corrected to the nearest 0.2 seconds to enable a more accurate measurement of the time to peak power.

Drop jump procedures

Following habituation to the test procedures, participants were asked to perform a drop jump from a height of 50 cm (Horita et al. 1999), which was recorded using an infrared timing system (Optojump, Microgate S.r.l., Bolzano, Italy) interfaced to a laptop (Sony Vaio, PCG-4G1M, Sony, Tokyo, Japan). Participants dropped from the pre-specified height and then jumped for maximal height following minimal contact with the ground. Vertical takeoff velocity (v) was calculated from the flight

time (t_{flight}), i.e. $v = 0.5(t_{\text{flight}} \times g)$, where g is the acceleration due to gravity ($9.81 \text{ m} \cdot \text{s}^{-2}$). Vertical jump height was then calculated from the equation: $\text{height} = v^2/2g$ (Byrne & Eston 2002a). In addition, the contact time on landing was recorded as an index of the maximal rate of force development (Kraemer & Newton 1994).

Peak isokinetic knee extensor torque measurement

Peak knee extensor torque was measured using an isokinetic dynamometer (Biodex 3, Biodex Medical Systems, Shirley, NY, USA) at both slow ($60 \text{ deg} \cdot \text{s}^{-1}$) and fast ($360 \text{ deg} \cdot \text{s}^{-1}$) velocities. Testing was preceded by a standardized warm-up of 4 minutes of cycling at 50 W on a cycle ergometer (Lode Corival, Groningen, The Netherlands) followed by static stretching exercises of the knee extensor/flexor muscle groups. The participant was positioned in a sitting position with hip of the test limb fixed at 85 deg (full hip extension = 180 deg) respectively. The ankle was secured to the input arm of the dynamometer on the tibia above the malleoli, with the rotational axis of the dynamometer aligned with the lateral femoral epicondyle to ensure that only motion around the knee joint was possible. The upper body and contralateral limb were secured to avoid any extraneous movement, with the subject asked to maintain the arms positioned across the chest. The total range of motion for each subject was manually determined by the investigator, while the mass of the limb was recorded by the dynamometer to enable gravitational correction of peak torque values. Following habituation trials at each test velocity, participants performed five maximal efforts at each velocity, separated by a 2-minute recovery. Visual feedback, displaying real-time force, was used to encourage maximal efforts. Participants were consistently encouraged to exceed target values, based on those achieved during the familiarization session.

Perceived muscle soreness

Each participant was asked to indicate perceived muscle soreness of the knee extensors using a visual analogue scale (VAS). The scale was numbered from 0 to 10 (on the reverse side of the sliding scale) with 0 indicating no muscle soreness and 10 signifying that the muscle was too sore to move. With hands on hips and squatting to an approximate knee angle of 90 deg, participants

were asked to indicate the level of perceived soreness based on the rating scale. This corresponded to the location of perceived muscle soreness on the continuum. This technique has been used successfully in previous studies (e.g. Twist & Eston 2005).

Muscle-damaging exercise

Participants performed 10 sets of 10 maximal vertical jumps, interspersed with 1-minute recovery periods between each set. Prior to starting the exercise, a maximal vertical counter-movement jump was performed and the height recorded on an infrared timing system (Optojump, Microgate S.r.l.). The participant was asked to maintain this target height for each subsequent jump. This technique has been used successfully in previous studies (e.g. Twist & Eston 2005).

Statistical analysis

Paired samples *t* tests were performed on baseline measures between treatment and control groups to ensure that no significant differences existed. All measures over time were compared in relation to baseline measures. Changes in perceived muscle soreness, peak power, time to peak power, jump height and contact time were analyzed using five separate two-way (Time

[5] × Group [2]) analyses of variance (ANOVA). Isokinetic strength data were analyzed using a three-way (Group [2] × Speed [2] × Time [5]) ANOVA with repeated measures on the last two factors. Assumptions of sphericity were assessed using the Mauchly's test of sphericity, with any violations adjusted by use of the Huynh-Feldt correction. Corrected values are indicated by the suffix _{HF}. Paired *t* tests with Bonferroni correction were used to follow up significant results. Correlations between perceived muscle soreness and performance parameters were assessed using the Fisher *z* transformation procedures. In all cases, the alpha level was initially set at *p* = 0.05.

Results

Paired samples *t* tests on baseline values indicated that no significant differences existed between the treatment and control groups (*p* > 0.05). This suggested that the random allocation of subjects was successful in forming two groups with similar baseline characteristics. Absolute values for all measures are shown in Table 1. Figures 1–6 show how these values have changed relative to the baseline values.

Table 1. Absolute change in performance measures for treatment and control groups following plyometric exercise*

	Baseline	24 hr	48 hr	72 hr	168 hr
Peak torque 60 deg · s ⁻¹ (N · m)					
Treatment	178.9 ± 12.6	158.3 ± 10.8	155.9 ± 11.2	165.6 ± 8.2	178.8 ± 11.6
Control	197.9 ± 14.3	195.9 ± 14.5	196.5 ± 13.5	200.6 ± 16.0	201.1 ± 16.3
Peak torque 360 deg · s ⁻¹ (N · m)					
Treatment	94.0 ± 6.4	83.9 ± 7.0	88.2 ± 6.1	89.9 ± 7.1	94.9 ± 6.9
Control	89.4 ± 6.4	90.3 ± 7.4	94.3 ± 8.2	95.2 ± 6.7	91.0 ± 8.7
Peak power output (W)					
Treatment	851.0 ± 67.5	797.8 ± 63.6	781.7 ± 69.3	795.9 ± 62.1	853.8 ± 70.1
Control	966.8 ± 101.2	987.2 ± 101.9	993.9 ± 100.7	1014.7 ± 107.1	1013.4 ± 105.7
Time to peak power (s)					
Treatment	4.6 ± 0.3	4.7 ± 0.3	5.0 ± 0.3	4.5 ± 0.2	4.4 ± 0.3
Control	5.7 ± 0.5	5.5 ± 0.5	5.5 ± 0.5	5.4 ± 0.4	5.5 ± 0.4
Jump height (cm)					
Treatment	31.2 ± 2.0	28.9 ± 2.0	29.1 ± 2.0	29.2 ± 1.8	30.1 ± 1.9
Control	30.0 ± 2.1	30.5 ± 2.2	29.6 ± 2.2	29.6 ± 2.2	29.4 ± 2.2
Contact time (s)					
Treatment	0.44 ± 0.03	0.49 ± 0.02	0.48 ± 0.03	0.48 ± 0.02	0.45 ± 0.03
Control	0.40 ± 0.04	0.38 ± 0.04	0.40 ± 0.04	0.37 ± 0.03	0.39 ± 0.04

*Data are expressed as mean ± standard error of the mean.

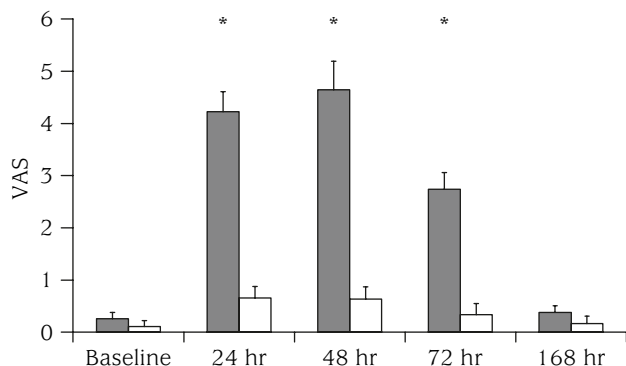


Fig. 1 Changes in visual analogue scale (VAS) for treatment (■) and control (□) groups following 100 plyometric jumps. VAS values are expressed as mean ± standard error of the mean. *Significantly different from baseline.

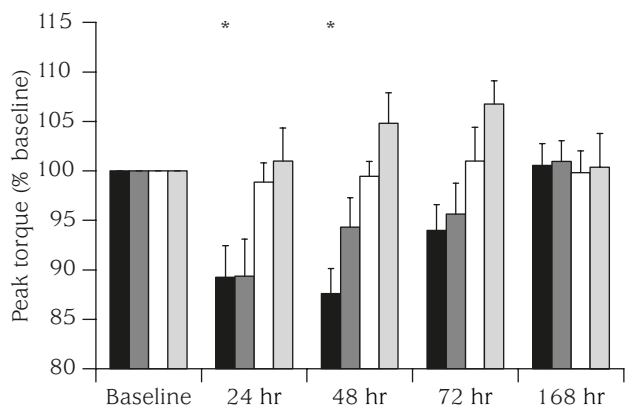


Fig. 2 Changes in relative isokinetic knee extensor peak torque at slow ($60 \text{ deg} \cdot \text{s}^{-1}$) (■ treatment, □ control) and fast ($360 \text{ deg} \cdot \text{s}^{-1}$) (■ treatment, □ control) velocities following 100 plyometric jumps (values are presented as a percentage of baseline). Values are expressed as mean ± standard error of the mean. *Significantly different from baseline.

Perceived muscle soreness

A significant interaction of time and group ($F_{2,57, 43.7_{HF}} = 32.9, p < 0.05$) demonstrated that while the control group remained relatively unchanged, the treatment group displayed an increase in perceived muscle soreness (VAS). Values peaked at 48 hours following plyometric exercise and had returned to baseline values at 168 hours (Figure 1).

Peak isokinetic knee extensor torque

Repeated measures ANOVA showed a significant interaction effect of time and group on peak torque ($F_{3,9, 66.0_{HF}} = 7.6, p < 0.05$), indicating a significant loss of strength in the treatment group (Figure 2). However,

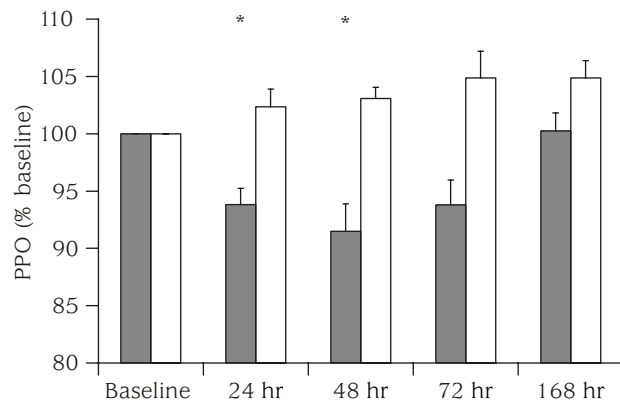


Fig. 3 Changes in relative peak power output (PPO) for treatment (■) and control (□) groups (values are presented as a percentage of baseline). Peak power values are expressed as mean ± standard error of the mean. *Significantly different from baseline.

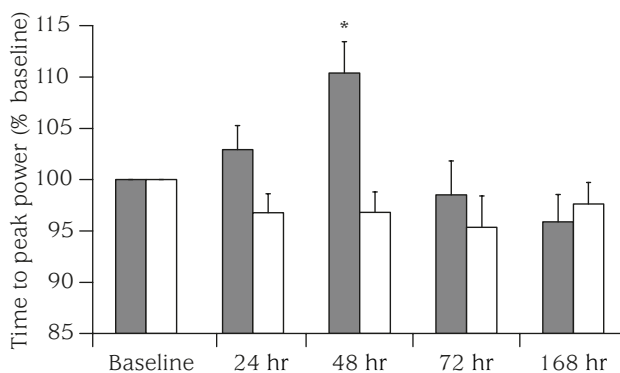


Fig. 4 Changes in relative time to peak power for treatment (■) and control (□) groups (values are presented as a percentage of baseline). Values are expressed as mean ± standard error of the mean. *Significantly different from baseline.

the interaction of speed by time by group was not significant ($F_{3,4, 58.6_{HF}} = 1.01, p > 0.05$), suggesting that recovery of peak torque following plyometric exercise was not dependent on the velocity of movement.

Peak power during a 10-second cycle ergometer sprint

The significant interaction of time and group ($F_{4, 68} = 7.7, p < 0.05$) indicated that relative PPO was reduced in the treatment group, while the control group remained unchanged. The relative PPO values were significantly lower at 24 hours and 48 hours following the plyometric exercise protocol, with values recovering by 168 hours (Figure 3).

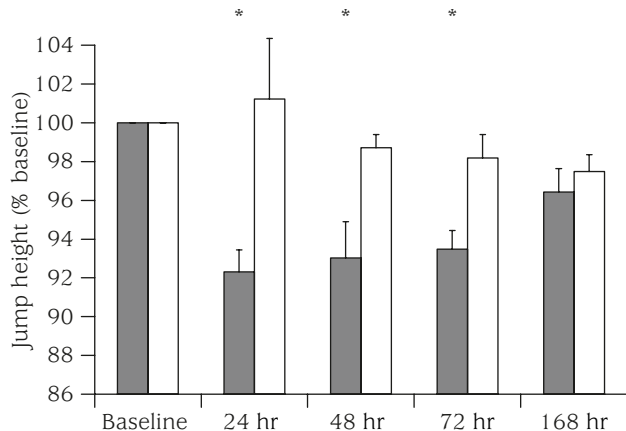


Fig. 5 Changes in relative drop jump height for treatment (■) and control (□) groups (values are presented as a percentage of baseline). Values are expressed as mean ± standard error of the mean. *Significantly different from baseline.

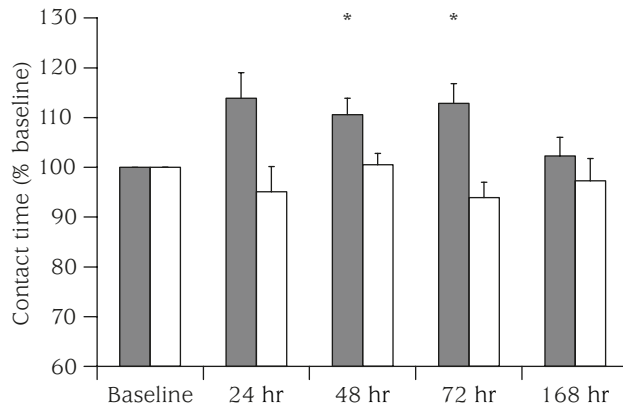


Fig. 6 Changes in relative drop jump contact time for treatment (■) and control (□) groups (values are presented as a percentage of baseline). Values are expressed as mean ± standard error of the mean. *Significantly different from baseline.

Table 2. Relationships between perceived muscle soreness and neuromuscular performance measures in the treatment group ($n = 10$) following plyometric exercise*

	Peak torque at 60 deg · s ⁻¹	Peak torque at 360 deg · s ⁻¹	Peak power output	Time to peak power	Drop jump height	Contact time
Visual analogue scale	-0.82 [†]	-0.68 [†]	-0.82 [†]	0.64 [†]	-0.75 [†]	0.64 [†]

*Correlation coefficients (r) calculated using the Fisher z transformation procedure; [†] $p < 0.05$.

Time to peak power during a 10-second cycle ergometer sprint

Repeated measures ANOVA showed that there was a significant interaction of time and group for time to peak power ($F_{2,4, 41.4} = 3.5, p < 0.05$), indicating that time to achieve peak power during maximal intensity cycling increased following plyometric exercise (Figure 4). Values increased from baseline by 10.4% at 48 hours, but appeared to have returned to baseline by 72 hours following the plyometric exercise protocol.

Drop jump performance

The significant interaction of time and group ($F_{4, 68} = 6.7, p < 0.05$) indicated that jump height in the treatment group decreased and remained below baseline values at 24, 48 and 72 hours following the plyometric exercise protocol (Figure 5). The corresponding ground contact times for the treatment group increased significantly from baseline ($F_{4, 68} = 3.7, p < 0.05$). Values increased by 10.6% and 12.8% at 48 hours and 72 hours respectively, while control group values remained unchanged (Figure 6).

Relationship between functional measures and perceived muscle soreness

Correlations using the Fisher z transformation indicated significant relationships between muscle soreness and all measures of neuromuscular performance ($p < 0.05$). Values are shown in Table 2.

Discussion

Increases in perceived muscle soreness following the plyometric exercise protocol were consistent with those previously reported (Cleak & Eston 1992; Newham et al. 1987), as were the reductions in isokinetic strength (Byrne & Eston 2002a; Byrne et al. 2001; Deschenes et al. 2000; Fridén et al. 1983). These observations indirectly support the presence of muscle damage following the plyometric exercise protocol.

While supporting previous studies which have demonstrated an immediate and prolonged reduction in the ability to generate peak power during maximal intensity cycling (Twist & Eston 2005; Byrne & Eston

2002b; Sargeant & Dolan 1987), as far as the authors are aware, this is the first study to investigate the effects of muscle-damaging exercise on the time to peak power during maximal intensity cycling. In a study by Byrne and Eston (2002b), which observed a reduction in PPO during 30-second Wingate performance, data indicated that the time to achieve peak power might also have been increased, although the software used in the study was unable to assess data with sufficient frequency to enable a true analysis of the variable. The data presented here suggest that reductions in PPO were accompanied by concurrent increases in the time to achieve peak power.

Previous findings have illustrated an immediate and prolonged reduction in RFD of up to 65% at 48 hours following an eccentric loading protocol (Minshull et al. 2007). This would appear to be consistent with the timing of the greatest increase in time to peak power in the current study. However, time to peak power values in this study appeared to have recovered by 168 hours, whereas in Minshull et al.'s study (2007), the recovery of RFD was incomplete at this time point.

Differences in the response of RFD and time to peak power may be explained by the opposing modes of dynamometry used to evaluate each parameter and the sensitivity of the recording instrumentation. Studies assessing RFD have used dynamometers with a load cell permitting signal amplification of muscle force in excess of 2 kHz, which contrasts to the sampling rate of 5 Hz used in the current study. However, although a higher level of sensitivity is achievable, the ecological validity of isometric dynamometry and RFD has been questioned (Wilson & Murphy 1996). In isometric actions, the stiffness of the series elastic component determines the rate at which forces are transferred from the muscle contractile component to the skeletal system. Therefore, it might be that muscle-damaging exercise will affect responses of isometric and dynamic muscle function differently.

The loss in isokinetic force-generating capability is consistent with the characteristics of low frequency fatigue (Jones 1996) and might be expected to contribute to the observed reductions in PPO. Animal studies, which have indicated a reduced excitation-contraction (E-C) coupling efficiency due to a reduction in calcium release per action potential, have shown that maximal

force production is concurrently impaired for several days following eccentric exercise (Ingalls et al. 1999; Warren et al., 1993). Therefore a reduction in force-generating capability due to E-C coupling failure would unsurprisingly reduce the ability of the muscle to produce power.

It is plausible that repeated stretching of the quadriceps during plyometric jumping might have led to preferential disruption in type II muscle fibers (Brockett et al. 2002; Fridén & Lieber 1992; Lieber & Fridén 1988; Fridén et al. 1983) as a result of early fatigue and temporary increases in muscle stiffness caused within these fibers by the eccentric component (Enoka 1996). These fibers would then be less able to contribute to force and power generation following the damaging exercise and therefore increase the time taken to achieve peak power. These suggestions are consistent with the immediate and prolonged exacerbation in PPO and time to peak power during the 10-second cycle sprint observed in this study.

It has been suggested that following muscle-damaging exercise, voluntary activation might be impaired as a result of increased muscle pain and tenderness leading to a reduction in neural drive to the muscle (Gandevia et al. 1996; Westing et al. 1991). Hence, it is possible that a reduced voluntary effort was manifested as a reduction in maximal cycling performance. The high correlations between perceived muscle soreness, peak power and time to peak power indirectly support this suggestion. In contrast, Prasartwuth et al. (2005) recently reported that the relationship between muscle soreness and voluntary activation only exists in the acute period following muscle damaging exercise, i.e. up to 24 hours. Using motor nerve stimulation, the authors concluded that prolonged losses in voluntary activation (hence, maximal force production) were more attributed to disruption to the E-C coupling mechanism and Ca^{2+} homeostasis. However, these findings were reported using isometric contractions and might not explain the changes associated with dynamic muscle activity given the difference in activation patterns between both muscle actions (Enoka 1996).

Drop jump height and ground contact time were both impaired in the treatment group following the plyometric exercise. These findings are consistent with previous studies that reported a change in drop jump

performance following muscle-damaging exercise (Byrne & Eston 2002a; Avela et al. 1999; Horita et al. 1999).

The reduction in jump height did not follow a similar pattern to that of peak power during maximal intensity cycling, with the greatest decrement occurring at 24 hours following the muscle-damaging exercise. It is assumed that this response is an artefact of the loss in force-generating capability of the knee extensors following muscle-damaging exercise. However, this observation would also suggest that the mechanisms to explain the responses between cycling and drop jump performance are not similar, even though both tests are influenced by lower limb power. Indeed, it might be that the response to muscle-damaging exercise is test-specific.

The drop jump provides a suitable model to study dynamic muscle action, incorporating eccentric lengthening, isometric coupling and concentric shortening of the knee extensors within the movement (Komi 2000). Studies which have demonstrated increased contact time have proposed that, following eccentric exercise, damaged muscle displays a reduced tolerance to impact forces during stretch-shortening cycle movement. This results in an increased contact time during the deceleration and push-off phases of the jump (Byrne & Eston 2002a; Horita et al. 1999). This is attributed to a reduced stretch reflex and muscle and joint stiffness brought about by inhibition of the muscle spindle (Komi 2000). In addition, several authors have reported the loss of concentric, isometric and eccentric strength following muscle-damaging exercise (Byrne & Eston 2002a; Hortobágyi et al. 1998; Golden & Dudley 1992), which would undoubtedly exacerbate the stretch-shortening capabilities of the muscle and result in an increased contact time. The observations in this study further confirm that exercise-induced muscle damage not only impairs maximal strength and power, but also the rate at which these values are achieved.

In conclusion, the impairment of maximal intensity cycling performance is underpinned by the loss of force-generating capability by the neuromuscular system. This study was unable to determine the specific mechanism to explain the loss in neuromuscular performance, although it is proposed that the changes are a result of complex interactions of peripheral and centrally mediated mechanisms. The strong correlations between perceived muscle soreness and dynamic

muscle performance suggest that inhibitory mechanisms play a crucial role following muscle-damaging exercise, in an attempt to limit further damage to the neuromuscular structures. It also appears that, although the same mechanisms are apparent, the temporal pattern of recovery is dependent upon the mode of dynamic exercise and assessment following exercise-induced muscle damage. Nonetheless, a reduction in PPO and the rate at which force is generated has significant consequences for athletic performance and would also influence the types of training adopted by athletes in the days following muscle-damaging exercise.

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