

THE EFFECTS OF EXERCISE-INDUCED MUSCLE DAMAGE ON AGILITY AND SPRINT RUNNING PERFORMANCE

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The purpose of this investigation was to assess the effects of exercise-induced muscle damage (EIMD) on agility and linear sprint running performance. Twelve healthy adults were randomly allocated to a treatment group ($n=7$; age, 21 ± 1.2 years; height, 1.77 ± 0.11 m; body mass, 70.8 ± 8.2 kg) where they completed 100 plyometric jumps, or to a control group ($n=5$; age, 24 ± 0.7 years; height, 1.77 ± 0.07 m; body mass, 69.2 ± 8.8 kg). Perceived muscle soreness, isokinetic peak torque of the knee extensors at 60 and 270 deg \cdot s⁻¹, sprint running time over 5 and 10 m, a timed agility test and ground contact time at the agility turn point were recorded at baseline and then 24, 48 and 168 hours following muscle-damaging exercise. Perceived muscle soreness was significantly increased ($p < 0.05$) and isokinetic peak torque at 60 and 270 deg \cdot s⁻¹ was significantly decreased ($p < 0.05$) at 24 and 48 hours in the treatment group following plyometric exercise. Significant increases ($p < 0.05$) were also observed for 5 m (6%) and 10 m (5%) sprint time, agility time (8%) and ground contact time at the agility turn point (21%), peaking at 48 hours in the treatment group. All variables remained unchanged in the control group ($p > 0.05$). These findings indicate that agility and sprint performance is reduced following EIMD, and provides further evidence that performance of activities requiring rapid generation of force is impaired following muscle-damaging exercise. [*J Exerc Sci Fit* • Vol 7 • No 1 • 24–30 • 2009]

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Introduction

Participation in unaccustomed, eccentrically biased exercise often results in ultrastructural damage to skeletal muscle (Friden & Lieber 2001; Paul et al. 1989; Friden et al. 1983). Symptoms of this exercise-induced muscle damage (EIMD) include elevated muscle proteins in the blood, delayed-onset muscle soreness, swelling, a decreased range of motion, and impairment of proprioceptive function and neuromuscular control (for a review, see Byrne et al. 2004). However, perhaps of greatest consequence to sports performers is the immediate

and long-lasting reduction in strength and power commonly observed following muscle-damaging exercise. Traditionally, such observations have demonstrated significant reductions in isometric force generating capability (Byrne & Eston 2002a,b; Sayers & Clarkson 2001; Clarkson et al. 1992; Cleak & Eston 1992a). However, the relevance of such observations to sports performers is questionable, as such static movements rarely occur during athletic competition. Moreover, evidence that type II muscle fibers are more susceptible to EIMD (Brockett et al. 2001) would make the investigation of more dynamic movements warranted.

Several studies have consistently shown that EIMD reduces peak torque produced on an isokinetic dynamometer. However, results regarding whether there is any significant difference between performance losses at different angular velocities of movement have been equivocal, with some studies reporting greater strength loss at higher (Eston et al. 1996; Golden & Dudley 1992; Friden et al. 1983) or lower (Michaut et al. 2002;



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Deschenes et al. 2000; Gibala et al. 1995) angular velocities of movement and others reporting no difference (Byrne et al. 2001; Sherman et al. 1984). Although these measurements involve the use of a dynamic muscle action, the validity of these measurements are questionable when considering that angular velocity for knee flexion can be up to $975 \text{ deg} \cdot \text{s}^{-1}$ during activities such as sprinting (Baltzopoulos & Gleeson 2008), a velocity approximately twice as high as can be measured through the use of isokinetic dynamometry. Accordingly, the effects of EIMD on measures more closely associated with sporting performance requiring a rapid generation of force have been sought.

To date, decrements in various vertical jump parameters (Marginson et al. 2005; Byrne & Eston 2002a; Avela et al. 1999; Horita et al. 1999; Chambers et al. 1998) and cycle ergometer-derived peak power output (Nottle & Nosaka 2007; Twist & Eston 2005; Byrne & Eston 2002a) have been reported. Currently, only two studies (Twist & Eston 2005; Semark et al. 1999) have examined the effects of EIMD on sprint running performance, eliciting conflicting results. Furthermore, no study has examined the effects of EIMD on the ability to change direction rapidly. As the ability to generate rapid force and employ a high degree of neuromuscular control are significant contributing factors to sprint and agility performance (Young et al. 2002), the potential for EIMD to influence these attributes is feasible. Therefore, the aim of the present study was to examine the effects of EIMD on agility performance and to further explore its effects on sprint running performance.

Methods

Experimental design and participants

Twelve healthy participants were randomly allocated to a treatment ($n=7$; age, 21 ± 1.2 years; height, 1.77 ± 0.11 m; body mass, 70.8 ± 8.2 kg) or control group ($n=5$; age, 24 ± 0.7 years; height, 1.77 ± 0.07 m; body mass, 69.2 ± 8.8 kg). All were recreationally active and asymptomatic at the time of testing. To avoid the protective effects of a prior bout of EIMD, participants had not performed any systematic resistance training in the previous 6 months (McHugh et al. 1999). Each participant provided written informed consent to participate in the study, which was approved by the Ethics Committee of the Department of Sport and Exercise Sciences, University of Chester, UK. Measurements of participants' perceived muscle soreness, isokinetic peak torque of the knee extensors, sprint running time over

5 and 10 m, and agility performance were recorded at baseline and then 24, 48 and 168 hours following plyometric exercise.

Assessment of isokinetic strength

Isokinetic peak torque in the knee extensors of the dominant leg was measured at 60 and 270 $\text{deg} \cdot \text{s}^{-1}$ using an isokinetic dynamometer (Biodex Medical Systems Inc., Shirley, New York, USA). Participants were tested in the seated position with the lateral femoral epicondyle aligned to the dynamometer axis of rotation. The upper body and active limb were secured with restraining straps to prevent extraneous movement (Baltzopoulos & Gleeson 2008). The pad of the dynamometer lever arm was positioned at the distal point on the tibia above the malleoli, with the dynamometer lever arm length and the vertical, horizontal and seat positions recorded for each participant to replicate testing positions at each time interval. Range of motion for each contraction was set prior to testing for each individual, and limb mass was assessed to enable gravity correction of values (Gleeson & Mercer 1996). Participants performed a familiarization trial consisting of 3 submaximal and 1 maximal practice repetitions, which also served as a warm up at each angular velocity (Byrne et al. 2001). During testing, subjects performed 5 maximal repetitions at each angular velocity with a 2-minute interval between sets of repetitions, with the highest peak torque achieved during the repetitions recorded. Participants performed the slower angular velocity first to enhance the reproducibility of results (Wilhite et al. 1992). Visual feedback displaying real-time force was used to encourage maximal efforts (Gleeson & Mercer 1996).

Assessment of sprint performance

Following a standardized warm up and practice trial, participants performed 2 sets of a single 10 m sprint from a standing start on an indoor non-slip surface interspersed with a 5-minute recovery. Sprint times were recorded using six electronic photo cells (Time-it; Eleiko Sport, Halmstad, Sweden) positioned at 0 (start), 5 and 10 m. Times for sprint performance were recorded to the nearest 0.01 second via telemetry to a handheld system. The fastest times recorded over 5 and 10 m were used for analysis.

Assessment of agility performance

Participants performed 2 sets of the Agility-505 test (Draper & Lancaster 1985) from a standing start on an indoor non-slip surface (Figure 1), separated by a 5-minute recovery. The test required participants to sprint

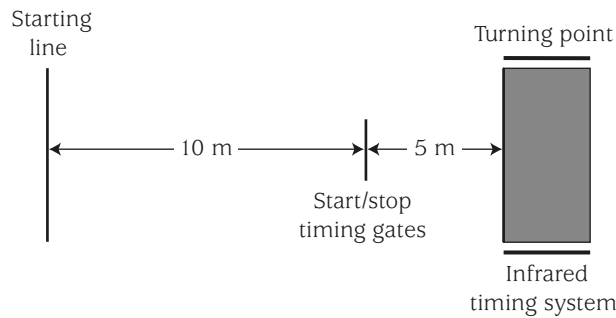


Fig. 1 Diagram of the agility performance test (15 m sprint followed by a 180° turn on the dominant limb and a 5 m return sprint).

15 m, perform a 180° turn on the dominant limb and sprint 5 m toward the start position through two electronic photo cells (Time-it; Eleiko Sport). An infrared system (Optojump; Microgate S.r.l., Bolzano, Italy) was positioned at the turning point of the agility test in order to measure the ground contact time of the dominant limb during the turning maneuver. Agility performance time and ground contact time at the agility turn point were recorded to the nearest 0.01 second, with the fastest time recorded representing optimum agility performance.

Assessment of perceived muscular soreness

Participants reported perceived muscle soreness of the knee extensors using a visual analog scale (VAS). The VAS is numbered from 1 to 10 (on the reverse side of a sliding scale unseen by the participant) with 0 indicating no muscle soreness and 10 indicating that the muscles are too sore to move. Participants were instructed to adopt a knee angle of approximately 90° and then indicate the level of perceived soreness in the knee extensors based upon the rating scale. This method has been used successfully in previous studies to indicate muscle soreness (Marginson et al. 2005; Twist & Eston 2005).

Muscle-damaging exercise

Participants in the treatment group performed 10 sets of 10 maximal vertical jumps interspersed with 1 minute of passive recovery between sets. Prior to starting the exercise, a maximal vertical counter-movement jump was performed and its height recorded on an infrared timing system (Optojump; Microgate S.r.l.). The participants were then instructed to attempt to attain this height for each subsequent jump to encourage maximal effort. On landing, participants were instructed to adopt a knee joint angle of approximately 90° before

performing the next jump. This protocol has been used successfully to induce EIMD in previous studies (e.g. Twist & Eston 2005).

Statistical analysis

Changes in muscle function were assessed using a mixed model three factor [group (2) × angular velocity (2) × time (4)] analysis of variance (ANOVA), with repeated measures on angular velocity and time. All other variables were analyzed using separate mixed model two factor (group × time) ANOVAs. Assumptions of sphericity were assessed using Mauchly's test of sphericity, with any violations adjusted by use of the Greenhouse-Geisser correction ($_{GG}$). In the presence of a statistically significant F ratio, *post hoc* analyses were carried out using paired-samples *t* tests adjusted using the Bonferroni correction. Correlation analyses using the Fisher *Zr* transformation were performed to determine the overall relationship between muscle soreness and sprint times and muscle soreness and agility performance in the treatment group across each of the four testing occasions. Statistical significance was set at $p=0.05$.

Results

No significant differences were observed for any recorded baseline values between the treatment and control groups ($p>0.05$).

Muscle soreness

ANOVA showed a significant interaction of time by group on muscle soreness ($F_{3, 30}=36.136, p<0.05$). Muscle soreness in the treatment group was significantly higher than baseline at 24 and 48 hours following the muscle-damaging exercise before returning to baseline levels at 168 hours (Table). Significant positive correlations ($p<0.05$) were also observed between perceived muscle soreness and 5 m ($r=0.82$) and 10 m ($r=0.89$) sprint time, agility turn time ($r=0.82$) and ground contact time at the agility turn point ($r=0.94$) in the treatment group.

Peak isokinetic strength

ANOVA showed a significant interaction of time by speed by group for isokinetic peak torque of the knee extensors ($F_{3, 30}=5.633, p<0.05$). Isokinetic peak torque at 60 and 270 $\text{deg} \cdot \text{s}^{-1}$ in the treatment group was significantly reduced at 24 and 48 hours ($p<0.05$) following EIMD, whilst the control group remained unchanged ($p>0.05$). Furthermore, isokinetic peak torque was

Table. Muscle soreness and isokinetic peak torque of the knee extensors following the plyometric protocol*

	Baseline	24 hr	48 hr	168 hr
Muscle soreness				
Control	0±0.0	0±0.0	0±0.0	0±0.0
Treatment	0±0.0	6.5±1.5 [†]	6.1±2.1 [†]	0±0.0
Isokinetic peak torque 60 deg·s ⁻¹ (N·m)				
Control	202.5±41.1	189.3±39.9	198±47	203.4±52.7
Treatment	206.7±54.3	168.1±44.6 [†]	162.1±39.7 [†]	210.4±51.7
Isokinetic peak torque 270 deg·s ⁻¹ (N·m)				
Control	119±25	116.4±25.6	118.2±31.2	119.5±33.1
Treatment	124.4±22.9	113.4±21.4 [†]	109.1±17.1 [†]	125.7±25.4

*Data presented as mean ± standard deviation; [†]significantly different from baseline.

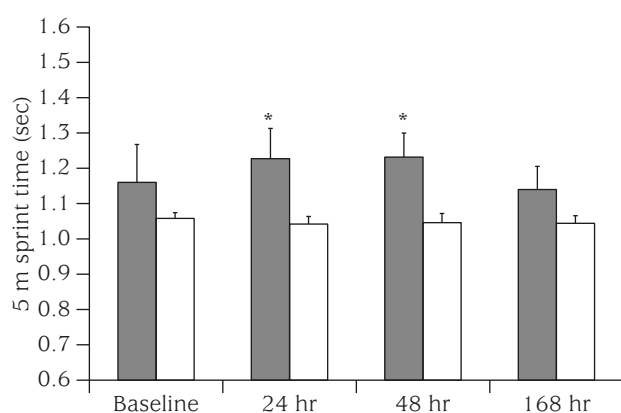


Fig. 2 Changes in 5 m sprint performance are presented as mean ± standard deviation for the treatment (■) and control (□) groups. *Treatment group significantly different from baseline.

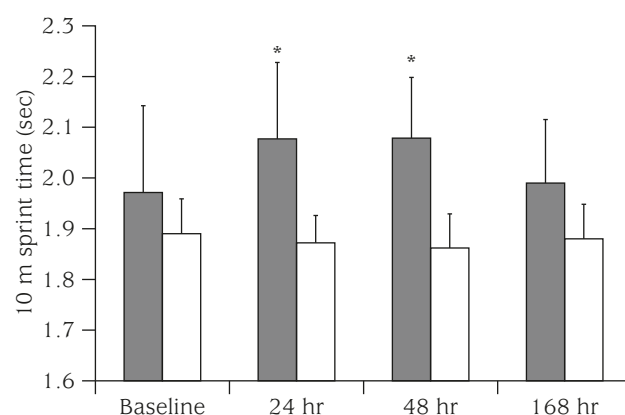


Fig. 3 Changes in 10 m sprint performance are presented as mean ± standard deviation for the treatment (■) and control (□) groups. *Treatment group significantly different from baseline.

reduced to a greater extent at the slower angular velocity of movement at 24 and 48 hours following muscle-damaging exercise (Table).

Sprint performance

ANOVA revealed a significant interaction of time by group for sprint performance over 5 m ($F_{3, 30} = 5.427$, $p < 0.05$) and 10 m ($F_{3, 30} = 8.172$, $p < 0.05$). Sprint time in the treatment group over 5 m (Figure 2) and 10 m (Figure 3) was significantly increased ($p < 0.05$) at 24 and 48 hours following EIMD before returning to baseline levels at 168 hours, whereas the control group remained unchanged ($p > 0.05$).

Agility performance

ANOVA revealed a significant interaction of time by group on agility performance time ($F_{3, 30} = 7.163$, $p < 0.05$). Performance was significantly ($p < 0.05$) slower when compared to baseline in the treatment group at 24 and 48 hours following EIMD before returning to

baseline levels at 168 hours (Figure 4), whereas the control group remained unchanged ($p > 0.05$).

Ground contact time at the agility turn point

ANOVA revealed a significant interaction of time by group on ground contact time at the agility turn point ($F_{(GG) 1.69, 16.88} = 15.635$, $p < 0.05$). Contact time was significantly increased ($p < 0.05$) in the treatment group at 24 and 48 hours following EIMD before returning to baseline levels at 168 hours (Figure 5), whereas the control group remained unchanged ($p > 0.05$).

Discussion

Changes in perceived muscle soreness and isokinetic peak torque in the treatment group provide indirect evidence to suggest that EIMD was present following the plyometric exercise. Muscle soreness peaked at 24 hours, remained elevated at 48 hours, and then

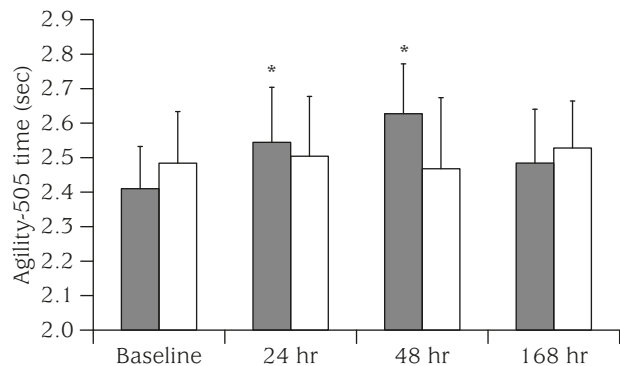


Fig. 4 Changes in agility performance time are presented as mean \pm standard deviation for the treatment (■) and control (□) groups. *Treatment group significantly different from baseline.

returned to baseline levels at 168 hours. These results are consistent with previous research which has reported a similar temporal pattern of muscle soreness following eccentrically-biased exercise (Twist & Eston 2005; Clarkson et al. 1992; Cleak & Eston 1992a,b). Isokinetic peak torque in the treatment group was significantly reduced at 24 and 48 hours following plyometric exercise and had returned to baseline at 168 hours. Furthermore, changes in peak torque of the knee extensors appeared to be dependent on the angular velocity of the contraction performed. Isokinetic peak torque in the treatment group was reduced to a greater extent at 60 deg \cdot s⁻¹ (23% and 28% at 24 and 48 hours, respectively) than peak torque at 270 deg \cdot s⁻¹ (10% and 14% at 24 and 48 hours, respectively). These findings support previous observations that strength loss following eccentric exercise is greater at lower angular velocities of movement (Michaut et al. 2002; Deschenes et al. 2000; Gibala et al. 1995), and may contradict the notion that type II muscle fibers were preferentially damaged during eccentric exercise. However, slower movements might be impaired following EIMD because of a possible force-related mechanism leading to an inverse relationship between activation failure and the velocity of movement (Michaut et al. 2002). Slower movement velocities, which demonstrate a greater capacity to generate force, might be regulated by the intensity of volitional activation against resistance and not ultimately by the velocity of movement (Deschenes et al. 2000). Neural inhibition might also be more pronounced in such actions in an attempt to prevent further damage (Westing et al. 1991). Therefore, it is possible that the isokinetic testing procedures used in the current study were unable to detect specific damage to fast-twitch muscle fibers.

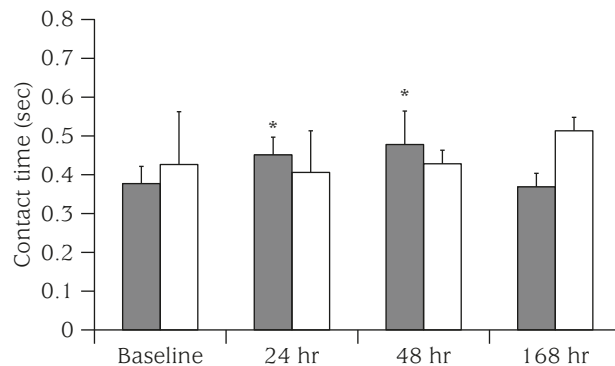


Fig. 5 Changes in ground contact time at the agility turn point are presented as mean \pm standard deviation for the treatment (■) and control (□) groups. *Treatment group significantly different from baseline.

Sprint times in the treatment group were 5% and 6% slower over 5 m and 5% slower over 10 m at 24 and 48 hours, respectively. These findings are consistent with those of Twist and Eston (2005) who reported that linear sprint running time over 10 m was significantly reduced by approximately 3% following 100 countermovement jumps. In contrast, Semark et al. (1999) reported that completing 70 drop jumps did not significantly impair sprint running performance over 5 m, 10 m, 20 m and 30 m. However, the authors also reported that serum creatine kinase was not significantly elevated over the period of testing, possibly indicating that there was not sufficient EIMD to influence sprint performance. When considering the changes in muscle strength and soreness in the present study and in serum creatine kinase and muscle soreness in the study of Twist and Eston (2005), it would appear that eccentric exercise does impair sprint performance providing sufficient muscle damage is present.

To date, the effects of EIMD on agility performance have not been investigated. In the present study, consistent with reductions in both strength and running speed, there was a significant reduction in agility performance following the plyometric exercise. In the treatment group, agility time assessed using the agility-505 test increased from baseline by approximately 5% and 8% at 24 and 48 hours, respectively. It is possible that the prolonged reductions in sprint speed may explain the observed decrements in agility turning time as both showed similar decrements in performance and time-course of recovery. However, the slightly higher decrement in agility performance at 48 hours compared to sprint performance (8% cf. 5%) may suggest that increases in sprint time were not solely responsible for decrements in agility performance.

Following the plyometric exercise, the treatment group demonstrated a significant increase in ground contact time at the agility turn point of approximately 16% and 21% at 24 and 48 hours, respectively. The changes in contact time observed in the present study are comparable with studies which have demonstrated increased contact time during the drop jump (Byrne & Eston 2002a; Avela et al. 1999). These studies have suggested that following eccentric exercise, damaged muscle displays a reduced tolerance to impact forces during stretch-shortening cycle movement. Consequently, decreases in strength and reflex activity result in a reduced ability to utilize ground impact forces and thus an increased contact time during the braking and push-off phases of the jump. During the similar action of deceleration and acceleration when turning, where the knee extensors switch rapidly from an eccentric to a concentric action, it is likely that damaged muscle would display similar properties of reduced efficiency resulting in an increase in contact and turning time. It is suggested that the reduced reflex sensitivity observed following EIMD is a result of a combination of increased stiffness regulation in the knee joint and a centrally-mediated, force-inhibiting neural mechanism which acts to prevent further damage to the muscle (Avela et al. 1999; Horita et al. 1999). More specifically, it has been hypothesized that there is an increased inhibitory drive to the α motor-neuron-pool in response to delayed-onset muscle soreness (Horita et al. 1999). The strong significant correlation between soreness and ground contact times ($r=0.94$, $p<0.05$) observed in the current study would seem to support this hypothesis and thus may partially explain the observed decreases in agility performance.

It is also possible that a reduced reflex sensitivity during the stretch-shortening cycle may have impaired the ability of the participants' damaged muscle to utilize ground impact forces during the 5 m and 10 m sprints. As such, participants may have produced less force during the propulsive phase of the leg movement and also increased their contact time with the ground. Interestingly, Horita et al. (1999) have also reported that following EIMD, participants adopt a more extended knee angle during landing in the drop jump. If such a change were to exist during the stretch-shortening cycle during sprinting, it is possible that force production and stride length and frequency would be altered. This has previously been observed during submaximal running following EIMD (Chen et al. 2007; Dutto & Braun 2004). However, it remains to be elucidated if these kinematic changes transfer to sprint running performance.

Nonetheless, a combination of these factors would be likely to affect maximal sprint running performance.

The observed increase in turning time and subsequent decrease in agility performance observed in the present study may be further explained by changes in neuromuscular control following EIMD. Several studies have reported disturbances in the ability to match both force and position in damaged versus non-damaged limbs (e.g. Paschalis et al. 2008; Proske et al. 2004; Brockett et al. 1997; Saxton et al. 1995), suggesting that EIMD affects proprioception. Whilst the precise mechanism is not entirely clear, it may be that pain results in a reduced motor system excitability to protect the muscle during the time in which it is being repaired, which in turn affects motor performance (Proske et al. 2003). Indeed, a recent study by Twist et al. (2008) has demonstrated that unilateral balance performance is impaired following a similar plyometric exercise protocol. Accordingly, the effects of reduced strength and impaired neuromuscular control are likely to impair agility performance by affecting balance and body control.

In conclusion, this study suggests that eccentric exercise results in immediate and prolonged reductions in strength, speed and agility which peaks at approximately 48 hours. Possible explanations for this reduced performance include neural inhibition in response to muscle soreness, reduced reflex sensitivity during the stretch-shortening cycle, and reductions in proprioceptive function. These outcomes have prospective detrimental effects on individuals who wish to perform dynamic, multi-joint actions in a sporting context. As a result, sports performers and coaches should be cognizant of the functional constraints following eccentric exercise and accommodate training and competition schedules accordingly.

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