Title:

Knee joint neuromuscular activation performance during muscle damage

and superimposed fatigue.

Claire Minshull[1], Roger Eston[2], David Rees[3], Nigel Gleeson[4]

[1]School of Science and Technology, Nottingham Trent University, Nottingham, NG11 8NS, United Kingdom. [2]School of Health Sciences, City East Campus, University of South Australia, Adelaide, South Australia 5000, Australia. [3]National Centre for Sports Injury Surgery, RJAH Orthopaedic Hospital, Shropshire, SY10 7AG, United Kingdom. [4]School of Health Sciences, Queen Margaret University Edinburgh, Edinburgh EH21 6UU, United Kingdom

Running Title:

Muscle damage and fatigue.

ABSTRACT

This study examined the concurrent effects of exercise-induced muscle damage and superimposed acute fatigue on the neuromuscular activation performance of the knee flexors of nine males (age: 26.7 ± 6.1yrs; height 1.81 ± 0.05m; body mass 81.2 ± 11.7kg [mean $\pm$ SD]). Measures were obtained during three experimental conditions: (i) FAT-EIMD, involving acute fatiguing exercise performed on each assessment occasion plus a single episode of eccentric exercise performed on the first occasion and after the fatigue trial; (ii) FAT, involving the fatiguing exercise only and; (iii) CON consisting of no exercise. Assessments were performed prior to (pre) and at 1h, 24h, 48h, 72h, and 168h relative to the eccentric exercise. Repeated-measures ANOVAs showed that muscle damage within the FAT-EIMD condition elicited reductions of up to 38%, 24% and 65% in volitional peak force, electromechanical delay and rate of force development compared to baseline and controls, respectively ($F_{[10, 80]} = 2.3$ to 4.6; $p < 0.05$) with further impairments (6.2% to 30.7%) following acute fatigue ($F_{[2, 16]} = 4.3$ to 9.1; $p < 0.05$). By contrast, magnetically-evoked electromechanically delay was not influenced by muscle damage and was improved during the superimposed acute fatigue ($\sim$14%; $F_{[2, 16]} = 3.9$; $p < 0.05$). The safeguarding of evoked muscle activation capability despite compromised volitional performance might reveal aspects of capabilities for emergency and protective responses during episodes of fatigue and antecedent muscle damaging exercise.

INTRODUCTION

Skeletal muscle is susceptible to ultrastructural damage following unaccustomed high-intensity eccentric exercise (Proske & Morgan 2001; Newham, McPhail, Mills & Edwards, 1983). This may be evident in the sports performer who commences training, match play or rehabilitation following a prolonged period of inactivity or injury-related deconditioning. Exercise-induced muscle damage (EIMD) may be attributed to excessively stressing relatively smaller numbers of active motor units and fibres (McHugh, Connolly, Eston & Gleim, 2000), particularly type-II fast-twitch fibres (Brockett, Morgan, Gregory & Proske, 2002), whose recruitment also corresponds to the most rapid and forceful activation patterns and net joint moments (Enoka, 1996) and to the capacity for effective joint stabilisation. Major functional consequences of EIMD include immediate and prolonged reductions in peak isometric muscle force ranging from 50% to 70% (Sayers & Clarkson, 2001; Rinard, Clarkson, Smith & Grosman, 2000; Cleak & Eston, 1992), reductions in peak power ranging from 15% to 20% (Byrne & Eston, 2002; Sargeant & Dolan, 1987) and an impairment in balance performance (Twist, Gleeson & Eston, 2008).

During neuromuscular conditioning interventions that involve multiple training sessions within a week, symptoms of EIMD may be carried forward into subsequent exercise sessions. Given the likely sequelae of EIMD, it is plausible that muscle symptomatic of damage might exhibit different neuromuscular responses to subsequent high-intensity fatiguing exercise compared to 'fresh' muscle. Currently, it is not known if EIMD-inciting exercise influences the

fatigue related-responses of the key knee musculature responsible for maintaining dynamic joint stability.

The efficacy of dynamic joint stabilisation during sudden loading is dependent on the temporal parameters related to the initiation, development and magnitude of the muscle force response (Blackburn, Bell, Norcross, Hudson, Engstrom, 2008; Linford et al., 2006). Several studies have shown the significant prophylactic effects of neuromuscular training programs on anterior cruciate ligament injury (Hewett, Ford & Myer, 2006). Scenarios associated with reduced levels of neuromuscular performance, such as EIMD and fatigue, might conversely compromise the system's capability to rapidly resist threats of joint injury posed by external forces. A high incidence of serious musculoskeletal injuries during pre-season training (Hawkins, Hulse, Hodson & Gibson, 2001) associated with periods of antecedent deconditioning and EIMD might exemplify this process. Similarly, unaccustomed exercise and EIMD during the rehabilitation process may attenuate recovery and hinder the dynamic stabilisation of the musculoskeletal system (Gleeson, Eston, Marginson & McHugh, 2003).

Electromechanical delay (EMD), defined as the time delay between the onset of muscle electrical activity and the onset of tension development in skeletal muscle (Norman & Komi, 1979), is considered to be an important marker of the temporal limitations for protective neuromuscular responses (Blackburn et al., 2008; Minshull, Gleeson, Walters-Edwards, Eston & Rees, 2007; Linford et al., 2006; Chan, Lee, Wong & Yeung, 2001). Increases in EMD latency have been observed following fatiguing exercise (e.g. Minshull, Eston, Bailey, Rees & Gleeson, 2011;

Minshull, et al., 2007; Chan et al., 2001) and following muscle damage in the biceps brachii (Howatson, 2010). To our knowledge, however, investigation into the effects of EIMD and fatigue on the temporal capacity of the neuromuscular system, to initiate and muster muscle force is limited to only one previous study (Behm, Baker, Kelland & Lomond, 2001), which reported no impairment to electrically-evoked EMD following EIMD in the elbow flexors and sub-maximal fatiguing exercise. No studies have investigated changes in these indices of performance in the dynamic knee stabilisers at functional joint angles and following maximal fatiguing protocols. There is some suggestion that sub-maximal static hold-to-fatigue times in muscles symptomatic of EIMD might be shorter compared to fresh muscle (Behm et al., 2001). Also, recent findings following evoked muscle activation, have suggested that fatigue in isolation provokes a maintenance of evoked EMD performance (Minshull et al., 2011; 2007). It is not yet known, however, whether or not these combined exercises stresses compromise the neuromuscular activation capability in a cumulative way, particularly in the knee flexor muscles and at functional joint angles that have been shown to be vulnerable. From a functional perspective, a worst case scenario might be even greater diminished capabilities for neuromuscular activation and perhaps greater threat to dynamic joint stabilisation.

The aim of this study was to examine the effects of EIMD with superimposed fatigue on neuromuscular activation performance of the knee flexors in males. An additional aim was to document any concomitant effects in the antagonist muscle group (knee extensors) subsequent to EIMD in the knee flexors.

METHODS

Participants

Nine male (age: 26.7 ± 6.1yrs; height 1.81 ± 0.05m; body mass 81.2 ± 11.7kg [mean ± SD]) competitive team games players (football, basketball, field hockey) who participated at least three times per week and who were free from injury provided written informed consent to participate in this study. Participants had not engaged in systematic resistance training of the involved musculature for 6-months prior to assessment and were instructed to refrain from strenuous physical activity for the 24-hours prior to the testing, maintain normal exercise levels and to avoid pain-relieving medications throughout the study. Assessment protocols were approved by the Institutional Ethics Committee for Human Testing.

Experimental Procedures and Design

On each test occasion and prior to testing, participants were asked to rate their perceived soreness and provide a fingertip blood sample before being secured in a prone position on a custom-built dynamometer (Minshull et al., 2009). The bi-lateral lever-arms of the dynamometer were attached to the legs of the participant by means of padded ankle-cuffs and adjustable strapping just proximal to the lateral malleolus. The dynamometer and knee joint's axes of rotation were aligned as closely as possible. Adjustable strapping across the mid-thoracic spine, pelvis and posterior thigh proximal to the knee localised the action of the involved musculature. A functionally relevant knee flexion angle of 25 degrees (0.44 rad), (0° = full knee extension) associated with the greatest mechanical strain on key ligaments (Li et al., 1999) was identified for each participant during activation of

the involved musculature using a goniometer system and was maintained throughout testing.

The experimental design comprised three treatment conditions: (i) an exercise-induced muscle damage and superimposed fatigue condition (FAT-EIMD) in which a fatiguing task of static maximal voluntary muscle activation of the knee flexors of the preferred leg was performed prior to (pre) and at 1h, 24h, 48h, 72h and 168h following a single EIMD protocol that was performed on the first assessment occasion; (ii) a control condition (CON) of equivalent duration to the FAT-EIMD condition consisting of no exercise; (iii) a further control condition (FAT) of equivalent duration to the FAT-EIMD condition, consisting of the static fatiguing exercise tasks only. The static fatiguing exercise task consisted of 30-seconds sustained maximal activation of the musculature followed by a 5-second rest period and a further 5-second period of maximal effort. This intermittent pattern of exercise was selected to enable multiple estimates of magnetically-evoked performance to be obtained. The EIMD protocol consisted of a warm-up (5 sub-maximal and 5 maximal eccentric activations) of the knee flexors of the preferred leg, followed by 6 sets (each separated by 1 minute) of 10 repetitions of maximal eccentric activations ($60°·s^{-1}$; movement range of 70° to 10° of knee flexion (0° = full extension)) using an isokinetic dynamometer (Kin-Com, Chattecx, Chattanooga, USA). In an attempt to ensure maximal muscle activations, strong verbal encouragement was provided throughout, in addition, real-time feedback of the level of muscle activation was observed by the participants on a computer screen, which they were encouraged to exceed, or at the very least maintain if greater force production wasn't possible. Following

testing of the knee flexors, assessments of ipsilateral antagonist (knee extensor)
muscle performance were obtained from seven participants using the same
protocol for knee flexor testing whilst in a prone position.

Treatment conditions were separated by one week; CON and FAT conditions
were presented in random order and the FAT-EIMD condition was performed last
to avoid carry-over effects from muscle damage (Brown, Child, Day & Donnelly,
1997). Performance measures were recorded prior to and immediately following
each static fatiguing exercise task (or equivalent period of rest). Magnetic
stimulation was performed first on each test occasion in order to minimise the
possible potentiating effects of prior MVMA (maximal voluntary muscle
activation) associated with the volitional testing. Experimental protocols are
shown in Figure 1. This was a fully-repeated experimental measures design and
as such participants attended the lab a total of nine times for testing (excluding
familiarisation).

Assessment of Neuromuscular Performance

Supra-maximal magnetic stimulation of the sciatic nerve and associated activation
of the knee flexors was achieved by means of a double wound coil (120 mm) that
was powered by a Magstim 200 stimulator (Magstim Co. Ltd., Whitland, Dyfed,
Wales). The protocol deployed to elicit and verify supra-maximal stimulation was
in accordance with the methodology described previously (Minshull et al., 2007).
Briefly, the magnetic coil was placed over the L4-L5 region (or in the femoral
triangle, just lateral to the femoral artery for knee extensor activation) and small
iterative positional changes of the coil were made that were commensurate with

increasing size of responses during a series of discrete stimulations. The optimum site for stimulation of the nerve was defined as the site that elicited the largest twitch force and M-wave amplitude and this optimised coil position was maintained manually throughout the remainder of the test. Supra-maximal stimulation was verified by contemporaneous visual inspection of the data during a sequence of seven discrete stimulations of increasing intensity that commenced at 40% of the Magstim 200's maximal capacity output with subsequent increments of 10% to 100% of capacity and by retrospective analyses of M-wave and peak twitch force data. Sequential stimulations were separated by at least 10-seconds to enable neuromuscular recovery.

Following a series of sub-maximal warm-up muscle activations an auditory signal was given randomly within 1-4 seconds that instructed the participants to flex (or extend for knee extensor testing) the knee joint as rapidly and forcefully as possible against the immovable restraint offered by the apparatus. Another auditory signal was given to the participant after 3-seconds of MVMA to cue neuromuscular relaxation. Intra-trial MVMA replicates were each separated by at least 10-seconds.

Indices of Neuromuscular Performance

Electromyographic activity (EMG) was recorded from the m. biceps femoris (and m. vastus lateralis) during the estimation of volitional static peak force (PF_V) and magnetically-evoked peak twitch force (P_TF_E) using bipolar rectangular surface electrodes (self-adhesive, Ag/AgCl; 10 mm diameter; Unilect, UK) that were applied longitudinally over the belly of the muscle parallel to the orientation of the muscle fibres. The raw unfiltered EMG signals, which incorporated minimal

intrusion from induced currents associated with external electrical and electromagnetic sources and noise inherent in the remainder of the recording instrumentation were passed through a differential amplifier (input impedance 10,000 MΩ, CMRR 100 dB, gain of 1000), filtered (Butterworth 2nd order; 1kHz cut-off frequency) (Cambridge Electronic Design, UK)) and were analogue-to-digitally converted at 2.5 kHz sample rate, ensuring a significant margin of reserve between the highest frequency expected in the EMG signal and the Nyquist frequency (Gleeson, 2001). The inter-electrode distance was 30 mm and a reference electrode was placed 30 mm lateral and equidistant from the recording electrodes. Standardised skin preparation techniques yielded inter-electrode impedance of less than 5 kΩ. Electrode placement was standardised across inter-day testing by marking the skin with indelible ink and mapping to anatomical landmarks. The m. biceps femoris was selected as an important contributor to restraint of anterior tibio-femoral displacement and lateral rotation of the femur relative to the tibia since both processes have been implicated in ACL injury (Li et al., 1999).

PF$_V$ and P$_T$F$_E$ were recorded as the mean of the highest force response during two intra-trial replicates. Volitional rate of force development (RFD$_V$) was calculated as the average rate of force increase between 25% and 75% of PF$_V$. Volitional and magnetically-evoked electromechanical delay (EMD$_V$ and EMD$_E$, respectively) were computed as the mean response of two intra-trial muscle activations in which the time delay between the onset of electrical activity and the onset of force was recorded. Onset of electrical activity and muscle force were defined as the first point in time where the recorded signals exceeded consistently

the 95% confidence limits of the background electrical noise amplitude (Minshull et al., 2009; 2007).

Indirect Markers of Muscle Damage

Subjective assessments of soreness were obtained by asking participants rate their soreness on visual-analogue scale (Price, McGrath, Rafii & Buckingham, 1983) following stretching of the involved musculature and active knee flexion. Statements on the scale included 'my muscles don't feel sore at all' and 'my muscles feel so sore that I don't want to move them', corresponding to numerical ratings (shielded from the participant) of 0 and 10, respectively, displayed on a 100cm line. Plasma creatine kinase levels, used as an indirect indicator of muscle ultrastructural integrity, were assessed from samples of whole fresh blood that were taken from fingertip capillary punctures. The initial sample of blood was discarded and a 32 μl sample was pipetted onto a test strip and analysed using a calometric assay procedure (Reflotron, Boehringer Mannheim, Germany). In the event that the creatine kinase concentrations exceeded the range of the analyser (2300 U/L), blood samples were diluted using purified water in quantities of 32 μl. The diluted concentrations value was then corrected to account for the number of dilutions made. Values were subjected to logarithmic transformations (CK_{log}) for statistical analysis (Brown et al., 1995).

***** Figure 1 near here *****

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Statistical Analyses

Effects of EIMD and fatigue on each index of neuromuscular performance were evaluated using separate three (condition: CON; FAT; FAT-EIMD) by six (time: pre; 1h; 24h; 48h; 72h; 168h) by two (static fatiguing exercise: pre; post) fully repeated-measures ANOVAs. Perceived soreness and CK_{log} levels were assessed by separate three (condition: CON; FAT; FAT-EIMD) by six (time: pre; 1h; 24h; 48h; 72h; 168h) fully repeated-measures ANOVAs. Comparisons between muscle groups (knee flexors, knee extensors) of the dominant leg following eccentric exercise were evaluated for each index of performance by separate two (condition: CON; EIMD) by six (time: pre; 1h; 24h; 48h; 72h; 168h) by two (muscle group: knee flexors; knee extensors) fully repeated measures ANOVAs, where CON represents the mean of scores for CON and FAT obtained prior to the static fatiguing exercise task. The assumptions underpinning the use of repeated measures ANOVA were checked and violations corrected by the Greenhouse-Geisser adjustment of the critical F-value, as indicated by $_{GG}$. Statistical significance was accepted at $p < 0.05$.

The experimental design offered an approximate 0.80 power of avoiding a Type-II error when employing a least detectable difference of 16 N, 8 ms and 350 N.s-1 for PF_V, EMD_V and RFD_V respectively and 3.13 N, 3.5 ms and 208 N.s-1 for P_TF_E, EMD_E and RFD_E, respectively.

Knee Flexor Neuromuscular Performance

The FAT-EIMD condition showed eccentric exercise-induced reductions in PF_V compared to baseline scores ($F_{[10, 80]}$ = 4.6; p <0.001) that were most prominent at 48h. Reductions in PF_V were 28.3%, 26.0%, 36.9%, 32.9% and 17.8% at 1h, 24h, 48h, 72h, 168h, respectively, compared to baseline levels (Figure 2). During the static fatiguing task, PF_V performance was decreased by on average 11.2% within FAT condition and by 6.2% in muscle symptomatic of damage during FAT-EIMD condition ($F_{[2, 16]}$ = 9.1; p <0.001).

***** Figure 2 near here *****

Eccentric exercise-induced reductions were also observed in RFD_V performance compared to baseline, that were most prominent at 48h ($F_{[10, 80]}$ = 2.8; p <0.001). Reductions in RFD_V performance were 38.4%, 50.0%, 65.3%, 59.1% and 30.6% at 1h, 24h, 48h, 72h, 168h, respectively, compared to baseline levels within FAT-EIMD (Figure 3). The static fatiguing exercise tasks elicited further mean reductions of 30.7% and 10.0% in RFD_V in FAT and FAT-EIMD conditions, respectively, compared to baseline ($F_{[2, 16]}$ = 4.3; p < 0.05) (Figure 3).

***** Figure 3 near here *****

EMD_V performance was impaired following eccentric exercise within the FAT-EIMD condition ($F_{[10, 80]}$ = 2.3; p <0.05), which was most prominent at 24h and

48h where it represented a 23.5% - 22.0% increase in latency, respectively (Table 1). The fatiguing static exercise was associated with impaired EMD_V performance of on average 21.3% within FAT condition and by 16.9% in muscle symptomatic of damage during the FAT-EIMD ($F_{[2, 16]} = 4.7$; p <0.05).

***** Table 1 near here *****

Knee Flexor Magnetically-Evoked Neuromuscular Performance

Results showed that EMD_E performance was not significantly influenced by eccentric exercise. However, the fatiguing static exercise was associated with improved performance, which was a similar in both muscle symptomatic (FAT-EIMD) and asymptomatic of damage (FAT) (average ~14% improvement) (Figure 4).

***** Figure 4 near here *****

There were no significant interaction effects for P_TF_E. A significant main effect for static fatiguing exercise only indicated that P_TF_E performance associated with post measures was superior compared to pre measures ($F_{[5, 40]} = 17.6$; p < 0.005) (Table 1).

Knee Flexor and Extensor Comparisons

Knee extensor RFD_V performance was maintained during the CON condition for both muscle groups, however, during the FAT-EIMD condition reductions in RFD_V performance were observed at all assessment points following the eccentric exercise compared to pre- exercise levels ($F_{[5, 30]} = 4.4$, p < 0.01).

Reductions in knee extensor RFD_V performance were 18.0%, 14.1% and 16.0% at 48h, 72h and 168h, respectively, which is of smaller magnitude to the changes observed in the knee flexors (Table 2). No other EIMD-related changes in volitional or magnetically-evoked indices of performance were observed for the knee extensors.

***** Table 2 near here *****

Indirect Indicators of Muscle Damage

CK_{log} and perceived soreness levels remained constant over both CON and FAT conditions, the FAT-EIMD condition showed increased group mean values that commenced at 24h ($F_{[10, 80]} = 15.3$; p <0.00; $F_{[10, 80]} = 7.0$; p <0.001, respectively) (Table 1; Figure 5). Peak values were observed at 72h and 48h, for CK_{log} and perceived soreness, respectively.

***** Figure 5 near here *****

DISCUSSION

Knee Flexor Neuromuscular Performance

To our knowledge, this is the first study to investigate the concurrent effects of fatigue and muscle damage the neuromuscular performance of the knee flexors. EIMD alone elicited changes in neuromuscular performance that were large biostastically (e.g PF_V relative experimental effect size >1.5 at 48h) and biologically meaningful (PF_V >36% decrease compared to baseline at 48h). Following EIMD, the magnitude of deterioration in EMD_V performance during the fatiguing task was also large (relative experimental effect size >1.1) and represented a further 16.9% change. The concurrent and detrimental effects on neuromuscular performance of these exercise endeavours might have important implications to dynamic knee joint stability (Minshull et al 2007).

The current results strongly suggest that the eccentric exercise protocol induced muscle damage, characterised by an increase in plasma CK_{log} values, ratings of perceived soreness (peak 48h) (Table 1; Figure 5). In addition, whereas performance over the control conditions showed no change, a significant and prolonged decrease in PF_V of the knee flexors (Figure 2) of a similar level to those reported previously (e.g. Byrne & Eston, 2002; Brockett et al., 2001) was observed. The EMD_V and RFD_V performance capabilities were also substantively impaired by up to 23.5% and 65.3% at 24h and 48h, respectively. The superimposed acute fatigue induced further decrements to all indices of volitional

neuromuscular performance, however, magnetically-evoked performance was preserved or enhanced, in the case of EMD_E.

The decrements to the volitional neuromuscular performance following eccentric exercise is likely to reflect a complex interaction of several factors, which may include structural damage to a proportion of active muscle fibres and associated organelles (Hortobagyi et al., 1998), preferential recruitment of slow-twitch muscle fibres (Warren, Hermann, Ingalls, Massell & Armstrong, 2000) and potential conscious or unconscious inhibition as a result of the firing of group III and IV afferents associated with the delayed pain response (Twist et al., 2008; Prasartwuth, Taylor & Gandevia, 2005). The functional importance of such large deficits in knee flexor tempero-force capabilities, coupled with possible losses to proprioceptive acuity and motor control following eccentric exercise (Twist et al., 2008; Saxton et al., 1995) could result in extended periods of elevated threat to effective stabilisation of the knee joint during critical episodes of mechanical loading in sports performances or in rehabilitative conditioning (Minshull et al., 2011; Blackburn et al., 2008). It might be speculated that in these circumstances, the delayed pain response that may peak commensurate with reduced neuromuscular performance capabilities may provide a useful subjective marker by which to manage the complexity and intensity of subsequent exercise endeavours.

Given the likely sequelae to EIMD, coupled with the expectation of increased susceptibility of fast twitch fibres to damage (Brockett et al., 2002; Linnamo, Bottas & Komi, 2000), the preservation of magnetically-evoked indices of

performance observed in the current study is perhaps contrary to expectation.

Eccentric exercise within the FAT-EIMD condition was regulated by voluntary

efforts and, although not measured in the current study, subconscious protective

inhibitory processes driven by pain and swelling. may have limited the

recruitment of the total pool of fast twitch motor units (Prasartwuth et al., 2005).

By contrast, evoked activation of the fastest motor units (Verges et al., 2008),

including those that may have evaded damage, might explain the observed

maintenance of evoked performance. Under these conditions, the net result

following stressful exercise may be a 'reserve capacity' of high-threshold motor

units that can be deployed during perceived threat to the joint system (Minshull et

al., 2007).

The FAT-EIMD condition was used to investigate the concurrent effects of an

episode of muscle damage interspersed amongst serial bouts of fatigue on

neuromuscular performance. The static exercise induced fatigue in the knee

flexors, characterised by an immediate reduction in PF_V that was similar in

magnitude across both FAT and FAT-EIMD conditions (11.2%; 6.2%,

respectively). While the musculoskeletal system might cope with acute deficits in

capability, the combination of insidious longer-term effects of muscle damage and

superimposed periods of acute fatigue might present a greater challenge to

dynamic joint stability. For example, the combined effects of EIMD and fatigue

on PF_V represent ~43% reduction in performance at 48h (36.9% plus 6.2%),

whereas in FAT the fatigue task alone elicited 11.2% reduction in PF_V

performance (Figure 2; Table 1). The equivalent comparison for EMD_V is ~47%

vs. 28.9% impairment during FAT-EIMD and FAT, respectively. Thus much

slower muscle activation and reduced force production, particularly at vulnerable

joint positions, may present insurmountable challenges to to maintaining dynamic

knee joint integrity.

Interestingly, despite the substantive impairment to voluntary contractile

capabilities, EMD_E performance during fatiguing exercise was improved to a level

that was superior to all corresponding volitional activation times. The level of

potentiation to EMD_E performance (~14%) is similar to reported previously

subsequent to acute fatiguing exercise only (21% and 10%: Minshull et al., 2007;

2011, respectively). Improvement of EMD_E times is likely to represent a net

result of the metabolic and mechanical effects of damage that needs to be

counterbalanced by correspondingly greater level of potentiation during maximal

muscle activation. Whilst metabolically mediated increases in sensitivity of

muscle contractile proteins to Ca^{2+} during maximal muscle actions (Rassier &

MacIntosh, 2000) may have contributed to quicker EMD_E performance, enhanced

muscle stiffness that might have been effected by intra-muscular swelling (Foley,

Jayraman, Prior, Pivarnik & Meyer, 1999) and a rightward EIMD-related shift in

the length-tension curve (Brockett et al., 2001; Morgan & Allen, 1999), might

also have helped to preserve evoked performance in the knee flexors at extended

and vulnerable joint positions. These data, describing a preservation and, or,

enhancement of evoked performance might offer further evidence of protective

inhibitory mechanisms under routine volitional exercise conditions (Hopkins &

Ingersoll, 2000) to maintain a reserve capacity of high threshold motor units.

Knee Extensor Neuromuscular Performance

Exercise-induced muscle damage in the agonist muscle group (knee flexors) was
associated with a minor decrease in RFD_V capabilities of up to 18.0% (at 48h) in
the knee extensors of the sub-sample of seven participants (Figure 3). Whilst
speculative, the contributing mechanisms might include intramuscular swelling
(Foley et al., 1999) and pain-driven inhibitory effects on the central drive to the
reciprocal antagonist musculature (Jaskolski, Andrzejewska, Marusiak, Kisiel-
Sajewicz, Jaskólska, 2007). From the current data it is not possible to discern the
relative contributions of these mechanisms, yet from a functional perspective, it is
interesting to note a possible down-regulation of knee extensor performance
commensurate with peak impairments to knee flexor performance (48h). Given
that activation of the knee extensors at vulnerable joint angles may be associated
with anterior tibio-femoral shear forces and a susceptibility to ACL injury (Aune.,
Schaff, Nordsletten, 1995), the observed reduction in RFD_V might be favourable
for knee joint stabilisation in circumstances where knee flexor activation
performance is impaired.

Conclusions

Knee flexor volitional neuromuscular activation performance was substantially
impaired following both eccentric exercise and acute fatigue. These effects of
concurrent EIMD and muscular fatigue experienced within contemporary
rehabilitation or training programmes may be sufficiently great to critically
amplify the challenges of maintaining synovial joint integrity during rapid
episodes of mechanical loading. Compensatory mechanisms that might offset
these changes may include the timing of the DOMS response, an improvement in

EMD$_E$ latencies and a down-regulation of antagonist knee extensor performance. Ultimately, increased risk of injury is likely to reflect the complex interaction of several factors, some of which may include neuromuscular conditioning, susceptibility to fatigue and an ability to deploy the full motor unit capacity of the neuromuscular system at crucial times. It must be noted that the current sample was relatively small and the magnitude of changes observed are likely to represent a 'worst-case' scenario within contemporaneous exercise settings. Future research might also scrutinise the efficacy of rehabilitative conditioning strategies in patients experiencing fatigue and, or, symptoms of EIMD.

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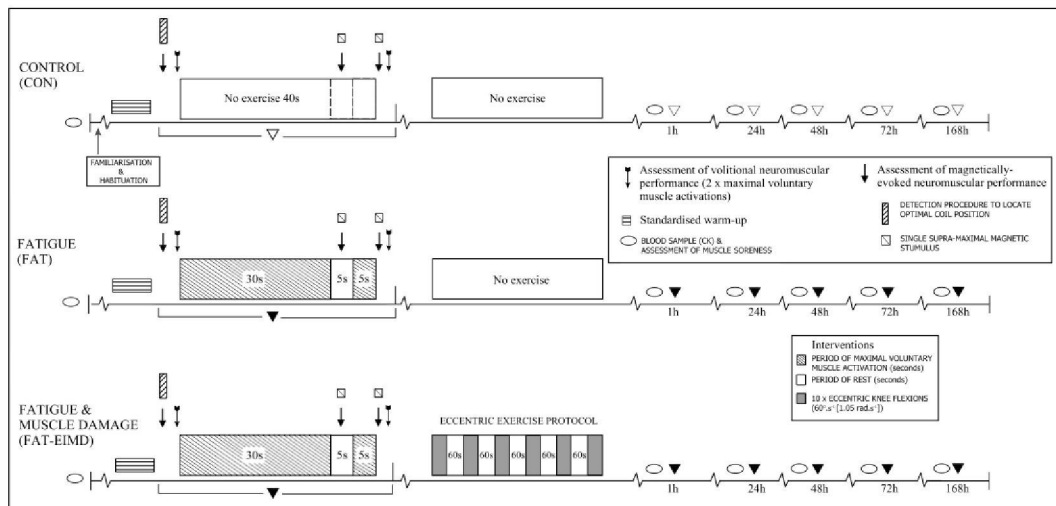
Figure 1. Schematic of the protocol. CON: no exercise; FAT: fatigue only; FAT-EIMD: eccentric exercise and fatigue.
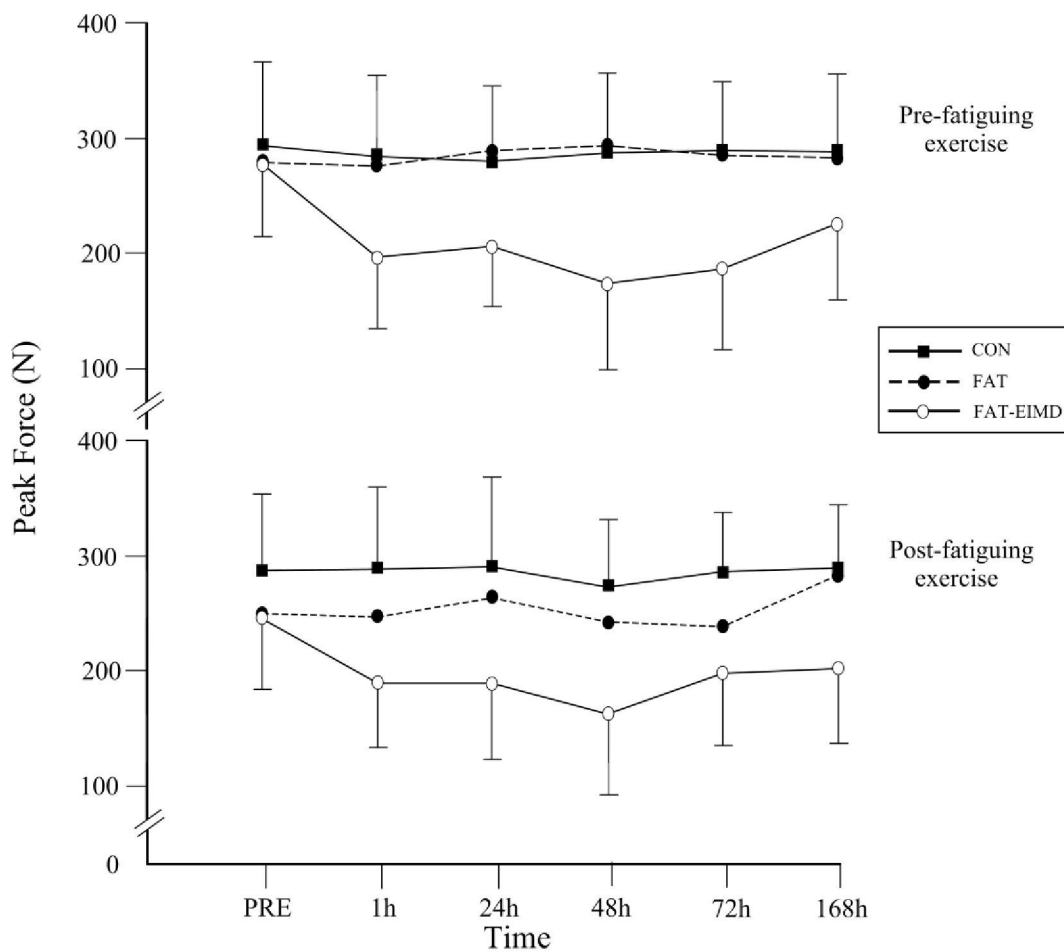

Figure 2. Volitional peak force (PFv) performance of the knee flexors over the three treatment conditions (group mean+s) (some s bars removed for clarity). CON: no exercise; FAT: fatigue only; FAT-EIMD: eccentric exercise and fatigue. Upper trace: prefatigue measures; Lower trace: post-fatigue measures.

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Figure 3. Volitional rate of force development (RFDv) performance of the knee flexors over the three treatment conditions (group mean+s) (some s bars removed for clarity). CON: no exercise; FAT: fatigue only; FAT-EIMD: eccentric exercise and fatigue. Upper trace: pre-fatigue measures; Lower trace: postfatigue measures.
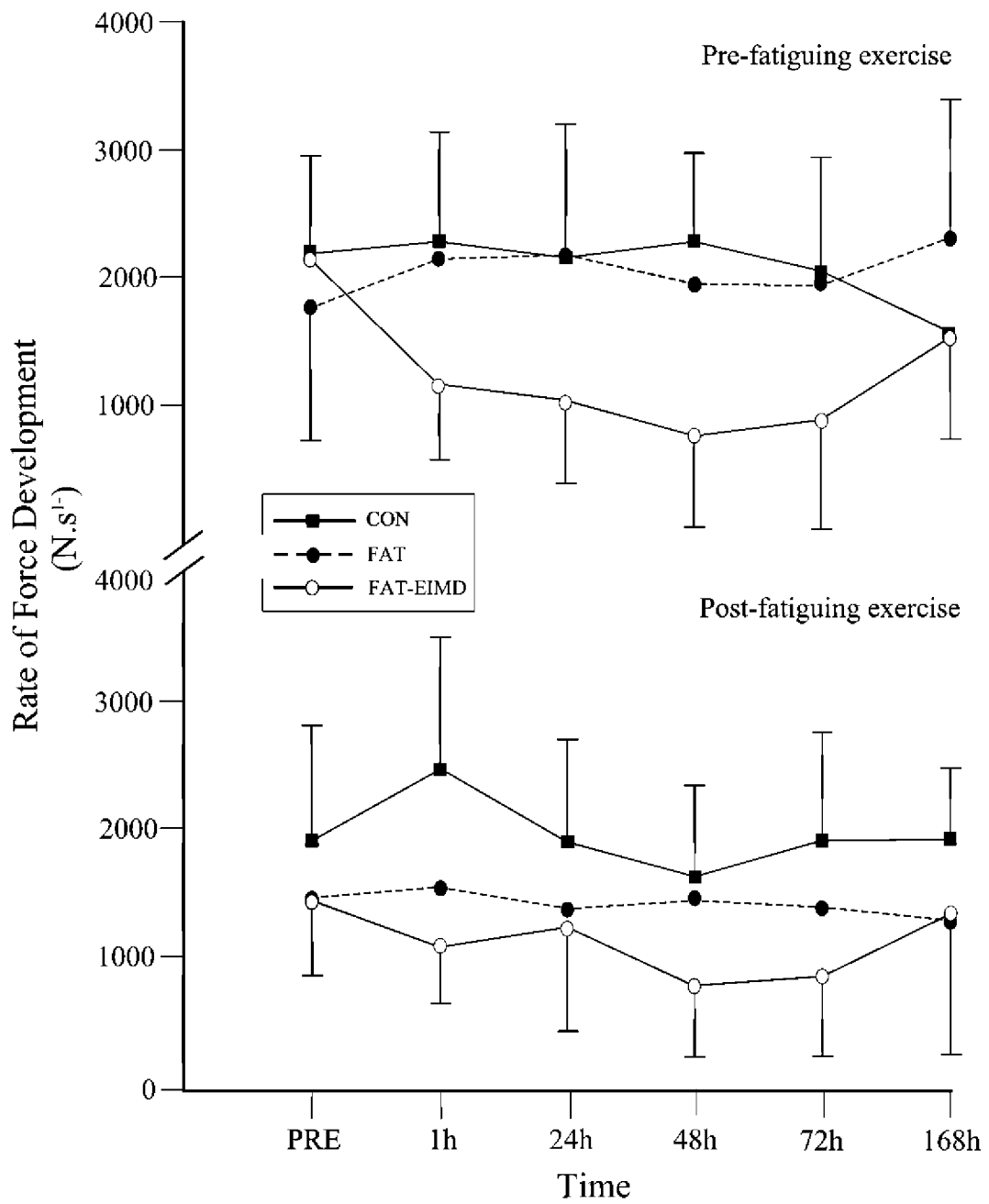
Figure 4. Magnetically-evoked electromechanical delay (EMDE) performance of the knee flexors over the three treatment conditions (group mean+s) (some s bars removed for clarity). CON: no exercise; FAT: fatigue only; FAT-EIMD: eccentric exercise and fatigue. Upper trace: pre-fatigue measures; Lower trace: post-fatigue measures.
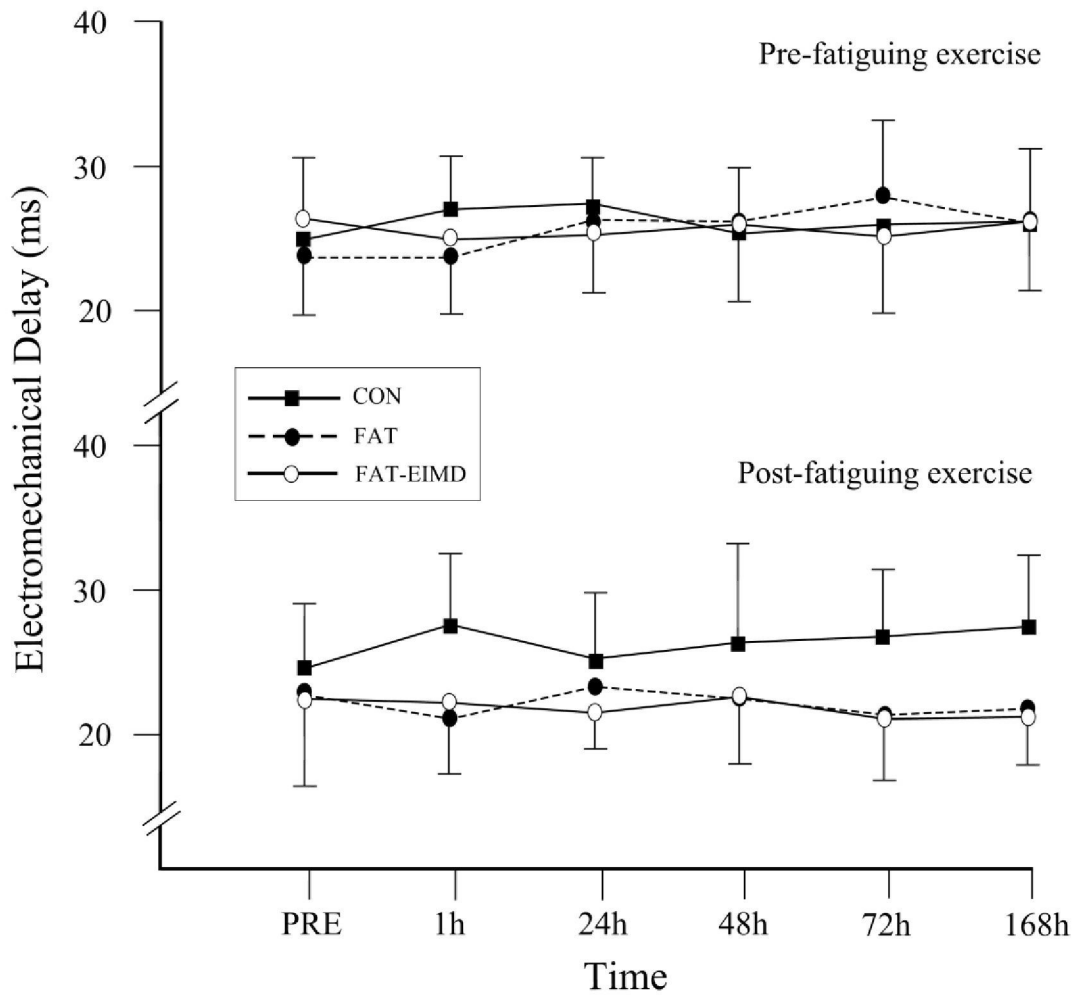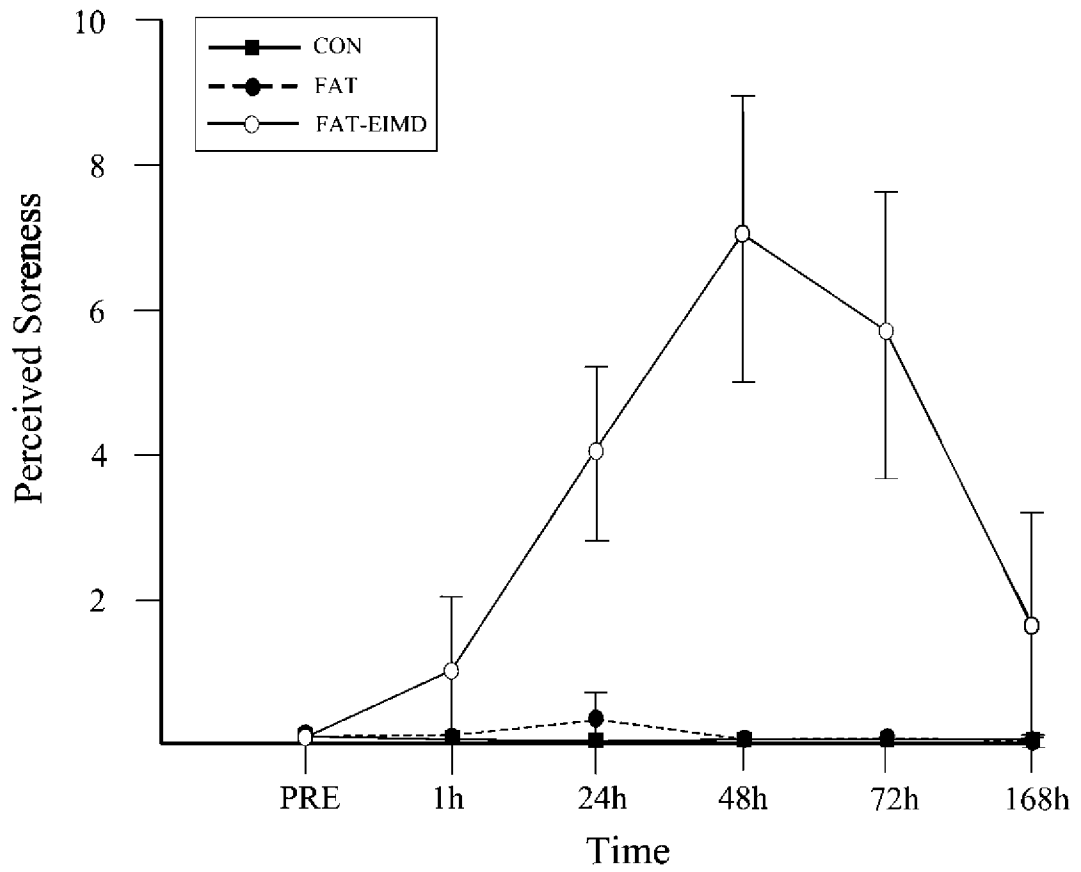
Figure 5. Ratings of perceived soreness of the knee flexors over the three treatment conditions (group mean+s). CON: no exercise; FAT: fatigue only; FAT-EIMD: eccentric exercise and fatigue.

Table I. Group mean (+ s) knee flexor volitional electromechanical delay (EMDV), magnetically-evoked peak twitch force (PTFE) and creatine kinase concentrations during the 'fatigue-muscle damage' condition, a: pre fatiguing exercise; b: immediately following fatiguing exercise.

Index			Time					
			Pre	1h	24h	48h	72h	168h
EMD_V	(ms)	a	47.3 ± 10.3	53.9 ± 13.9	58.4 ± 14.0	57.5 ± 11.7	49.0 ± 12.9	51.2 ± 22.9
		b	56.9 ± 12.8	60.2 ± 27.5	68.2 ± 31.4	69.0 ± 28.8	61.4 ± 33.4	68.8 ± 40.1
P_TF_E	(N)	a	34.1 ± 5.2	23.9 ± 5.8	27.9 ± 5.2	24.6 ± 6.6	28.8 ± 9.5	31.2 ± 5.0
		b	42.0 ± 16.0	34.8 ± 11.6	39.4 ± 9.6	33.3 ± 8.5	40.4 ± 14.0	39.5 ± 12.3
CK	(U/L)		94 ± 26	119 ± 32	277 ± 90	1121 ± 214	2824 ± 369	1284 ± 202

Table II. Group mean (+ s) knee extensor volitional peak force (PFV), electromechanical delay (EMDV) and rate of force development (RFDV) during the 'fatigue-muscle damage' condition

Index		Time					
		Pre	1h	24h	48h	72h	168h
PF_V	(N)	395.6 ± 107.5	410.1 ± 124.7	411.9 ± 113.5	431.1 ± 167.7	414.9 ± 120.0	423.8 ± 138.4
EMD_V	(ms)	32.7 ± 7.6	34.9 ± 10.0	31.6 ± 9.8	34.2 ± 13.1	28.5 ± 6.0	30.0 ± 9.2
RFD_V	(N.s^{-1})	2841 ± 1606	2774 ± 1593	2622 ± 1289	2384 ± 1411	2740 ± 1730	2388 ± 1775