Differential expression of muscle damage in humans following acute fast and slow velocity eccentric exercise

D Paddon-Jones, A Keech, A Lonergan & P Abernethy

School of Human Movement Studies, The University of Queensland, Brisbane, Queensland, Australia.

We sought to determine if the velocity of an acute bout of eccentric contractions influenced the duration and severity of several common indirect markers of muscle damage. Subjects performed 36 maximal fast (FST, n=8: 3.14 rad.s⁻¹) or slow (SLW, n=7: 0.52 rad.s⁻¹) velocity isokinetic eccentric contractions with the elbow flexors of the non-dominant arm. Muscle soreness, limb girth, plasma creatine kinase (CK) activity, isometric torque and concentric and eccentric torque at 0.52 and 3.14 rad.s⁻¹ were assessed prior to and for several days following the eccentric bout. Peak plasma CK activity was similar in SLW (4030±1029 U.L⁻¹) and FST (5864±2664 U.L⁻¹) groups, (p>0.05). Both groups experienced similar decrement in all strength variables during the 48 hr following the eccentric bout. However, recovery occurred more rapidly in the FST group during eccentric (0.52 and 3.14 rad.s⁻¹) and concentric (3.14 rad.s⁻¹) post-testing. The severity of muscle soreness was similar in both groups. However, the FST group experienced peak muscle soreness 48 hr later than the SLW group (24 hr vs. 72 hr). The SLW group experienced a greater increase in upper arm girth than the FST group 20 min, 24 hr and 96 hr following the eccentric exercise bout. The contraction velocity of an acute bout of eccentric exercise differentially influences the magnitude and time course of several indirect markers of muscle damage.


Corresponding author: djpaddon@utmb.edu

Introduction

The specificity of an exercise stimulus is an integral part of an athletic training program. Two of the major variables that influence exercise specificity are contraction mode and movement velocity. We recently demonstrated that an eccentric training program consisting of relatively fast velocity (3.14 rad.s⁻¹) eccentric contractions produced substantially greater improvements in muscle strength and also facilitated an increase in type IIb muscle fibres compared to slower eccentric contractions (0.52 rad.s⁻¹)¹. The mechanisms responsible for the failure of slower velocity eccentric contractions to produce a similar training effect remain uncertain. However, it is possible that the greater cumulative effects of muscle damage caused by the slow eccentric contractions inhibited adaptation to the eccentric training protocol.

Exercise-induced muscle damage is commonly observed following any number of activities that involve an eccentric component²⁻⁵. Further, symptoms can be induced following activities with diverse eccentric contraction velocities such as power-lifting and sprinting. Though the mechanisms and cellular processes contributing to an eccentric injury have been explored in detail²,⁶⁻⁸,
it remains unclear if the severity or duration of symptoms associated with exercise-induced muscle damage can be influenced by the contraction velocity of an acute bout of eccentric exercise.

Unlike concentric contractions, there is typically only small variation in maximum isokinetic eccentric torque production during slow (−0.52 rad.s⁻¹) to moderately fast (−3.14 rad.s⁻¹) eccentric contraction velocities¹,⁹,¹⁰. However, a number of potential mechanistic differences between fast and slow velocity eccentric contractions have been identified. These differences include an increased percentage of Type IIb muscle fibres following chronic exposure to fast but not slow eccentric contractions¹, greater muscle hypertrophy and strength gain following fast eccentric training¹¹, an increased contribution from series elastic tissues during faster eccentric contraction velocities¹², and a neurally-mediated inhibition of eccentric torque production at higher eccentric contraction velocities⁹,¹³,¹⁴.

The purpose of this study was to determine if the velocity of an acute bout of eccentric exercise influences the duration and severity of several common markers of muscle damage. We hypothesised that slower velocity eccentric contractions would produce greater muscle damage than a corresponding bout of faster velocity eccentric contractions.

**Methods**

Fifteen non-resistance trained male (n=8) and female (n=7) subjects participated in this study which complied with the requirements of The University of Queensland Medical Research Ethics Committee. Mean±SD age, height and weight was 24.1±4.8 y, 174.2±8.0 cm and 69.1±12.2 kg respectively. Subjects were counterbalanced on pre-test strength parameters and randomly assigned to a slow eccentric (SLW, n=7; 4 male, 3 female) or fast eccentric group (FST, n=8; 4 male, 4 female). Consistent with the contractions velocities used in our previous study, eccentric contractions were performed at 0.52 (slow) or 3.14 rad.s⁻¹ (fast). All subjects refrained from strenuous exercise for at least 10 days prior to the start of testing. A similar number of male and female subjects were included in each group. Specific gender differences following eccentric exercise in humans are equivocal. Some studies have identified an attenuated inflammatory response in women¹⁵ while others have noted that indices of muscle damage, force loss and plasma CK activity were not influenced by gender¹⁵-¹⁷. In most circumstances however, it appears that normal inter-subject variability is greater than any potential gender effect.

All subjects completed two familiarisation sessions 10 and seven days prior to the eccentric exercise intervention. During each session, subjects completed three maximal isometric contractions and three maximal concentric contractions at 0.52 and 3.14 rad.s⁻¹. However, due to the adaptive/protective effect afforded by even a small volume of previous exposure to eccentric exercise, no eccentric familiarisation was performed³. Pre-test blood samples for plasma CK analysis were obtained immediately prior to the first familiarisation session. Pre-test values for muscle soreness, isometric torque and eccentric and concentric 0.52 and 3.14 rad.s⁻¹ were obtained immediately prior to the eccentric exercise intervention.

The exercise protocol and equipment have been described previously¹,¹⁸,¹⁹.
Briefly, all exercise bouts were performed on an isokinetic dynamometer (Cybex 6000, Lumex Inc., Ronkonkoma, NY) using the subject’s non-dominant arm. Peak isometric elbow flexor torque and angle-specific concentric and eccentric torque values were assessed at 1.57 rad elbow angle. On each occasion, the highest torque attained during three maximal repetitions was recorded.

The eccentric intervention targeted the elbow flexors and consisted of six sets of six (6 sets x 6 reps) maximal eccentric contractions at a fast (FST: 3.14 rad.s⁻¹) or slow (SLW: 0.52 rad.s⁻¹) angular velocity. A 60-s rest period was provided between each set. During each eccentric repetition, subjects maximally resisted the dynamometer as it forced their arm from full flexion (0.7 rad) to near full elbow extension (2.9 rad).

With the exception of CK activity, all variables were assessed 20 min post-exercise and one day, two days, three days, four days, seven days and 10 days following the eccentric exercise bout (Figure 1). Muscle soreness was assessed via pressure algometry. With the subject in a supine position, arm fully extended and relaxed, the algometer (Pain Diagnostics & Thermography, Inc., Great Neck, NY, USA) was applied vertically with increasing force to the middle of the biceps brachii, 4 cm proximal to the elbow fold. Subjects indicated when they felt pain or tenderness, rather than a pressure sensation. The corresponding force gauge measurement was recorded and averaged over two trials. Plasma CK activity was measured spectrophotometrically (Cobas Mira, Roche Diagnostics, Switzerland) by means of an N-acetylcysteine activated, optimised ultraviolet test kit (Roche Diagnostics, Sydney, Australia). Venous blood samples (5 ml) were obtained from an antecubital vein of the non-exercised limb. Upper arm girth was measured midway between the lateral aspect of the acromial process and the lateral epicondyle of the elbow with the subject in a relaxed upright stance.

![Figure 1: The experimental protocol. Subjects performed 36 maximal fast (FST, n=8: 3.14 rad.s⁻¹) or slow (SLW, n=7: 0.52 rad.s⁻¹) eccentric contractions. Muscle soreness, limb girth, plasma creatine kinase (CK) activity, isometric torque and concentric and eccentric torque at 0.52 and 3.14 rad.s⁻¹ were assessed prior to and for several days following the eccentric bout.](image-url)
Data analysis
Within and between group comparisons were performed using two-way ANOVA. Two-tailed t-tests were used to compare demographic variables. A Bonferroni correction was applied to account for the multiple comparisons. Data are presented as means±SEM. Differences were considered significant at p< 0.05.

Results
Total work performed by the FST and SLW groups during the 36 repetition eccentric bout was 2857±635 J and 2524±404 J respectively (p>0.05), a non-significant difference equivalent to approximately four eccentric repetitions.

Pre-test eccentric torque measured at 0.52 rad.s⁻¹ was 55.7±9.5 Nm (FST) and 57.3±9.3 Nm (SLW) respectively (p>0.05). Pre-test eccentric torque measured at 3.14 rad.s⁻¹ was 50.4±8.5 Nm (FST) and 53.6±9.0 Nm (SLW), respectively (p>0.05). Peak eccentric torque loss occurred between 24 hr-48 hr in both groups. The recovery of eccentric torque at 0.52 and 3.14 rad.s⁻¹ occurred more rapidly in the FST group (Figure 2).

Pre-test concentric torque assessed at 0.52 rad.s⁻¹ was 52.3±9.4 Nm (FST), and 44.4±6.1 Nm (SLW), respectively (p>0.05). Pre-test concentric torque measured at 3.14 rad.s⁻¹ was 37.1±6.4 Nm (FST) and 39.9±5.8 Nm (SLW), respectively (p>0.05). The recovery of concentric torque at 3.14 rad.s⁻¹ occurred more rapidly in the FST group. However, neither group had fully recovered by Day 10 (Figure 3).

Pre-test isometric torque values were 60.0±9.2 Nm (FST) and 60.9±8.9 Nm (SLW), respectively (p>0.05). No significant differences between FST and SLW groups were observed throughout the post-testing period. Isometric torque had not fully recovered in either group by Day 10 (Figure 4).

Figure 2: Relative change in eccentric torque at A.) 0.52 rad.s⁻¹ and B.) 3.14 rad.s⁻¹ following an acute exercise bout of 36 maximal eccentric contractions performed at 0.52 rad.s⁻¹ (SLW; n=7) or 3.14 rad.s⁻¹ (FST; n=8) * denotes a significant difference between FST and SLW, (P<0.05).
Differential expression of muscle damage...

Figure 3: Relative change in concentric torque at A.) 0.52 rad.s⁻¹ and B.) 3.14 rad.s⁻¹ following an acute exercise bout of 36 maximal eccentric contractions performed at 0.52 rad.s⁻¹ (SLW; n=7) or 3.14 rad.s⁻¹ (FST; n=8). * denotes a significant difference between FST and SLW, (P<0.05).

Figure 4: Relative change in isometric torque (%) following an acute exercise bout of 36 maximal eccentric contractions performed at 0.52 rad.s⁻¹ (SLW; n=7) or 3.14 rad.s⁻¹ (FST; n=8).

Plasma CK values in the SLW and FST group increased dramatically following the eccentric exercise bout, reaching peak values of 4030±1029 U.L⁻¹ (SLW) and 5865±2664 U.L⁻¹ (FST) on day 4 (p<0.05). There were no significant differences between FST and SLW groups (p>0.05) (Figure 5). We have previously demonstrated that the contractions performed during the post-testing process do not significantly alter plasma CK activity (range: 92±15 U.L⁻¹ to 115±21 U.L⁻¹).

The SLW group experienced a greater increase in upper arm girth than the FST group at 20 min, 24 hr and 96 hr following the eccentric exercise bout. The FST group did not experience a significant increase in girth until 72 hr post exercise (Figure 6).

Muscle soreness was evident in both groups following the eccentric exercise...
Differential expression of muscle damage...

Figure 5: Change in plasma creatine kinase activity following an acute exercise bout of 36 maximal eccentric contractions performed at 0.52 rad.s⁻¹ (SLW; n=7) or 3.14 rad.s⁻¹ (FST; n=8).

Figure 6: Relative change in upper arm girth (%) following an acute exercise bout of 36 maximal eccentric contractions performed at 0.52 rad.s⁻¹ (SLW; n=7) or 3.14 rad.s⁻¹ (FST; n=8). * denotes a significant difference between FST and SLW, (P<0.05).

Figure 7: Force required to produce muscle soreness in the elbow flexor muscle group following an acute exercise bout of 36 maximal eccentric contractions performed at 0.52 rad.s⁻¹ (SLW; n=7) or 3.14 rad.s⁻¹ (FST; n=8). * denotes a significant difference between FST and SLW.

In the SLW group, muscle soreness peaked 24 hr post-exercise (p<0.05). In contrast, the FST group experienced greatest soreness 72 hr post-exercise (Figure 7).

Discussion

This study demonstrates that, while acute bouts of slow and fast velocity eccentric exercise have a similar effect on plasma CK activity and isometric torque, differences in rate of recovery of concentric and eccentric torque, muscle swelling and muscle soreness suggest that a number of common indices of muscle damage may be influenced by the contraction velocity of the initial eccentric exercise bout.

A substantial and prolonged reduction in muscle strength following acute
Differential expression of muscle damage...

intense eccentric exercise has been observed in many previous studies using a number of different exercise protocols\textsuperscript{4,5,10,21,22}. However, despite this widespread interest in the acute functional and morphological consequences of damaging eccentric muscle contractions, the influence of contraction velocity on common indices of muscle damage in humans has not been clearly defined. In the present study, both slow and fast eccentric exercise bouts resulted in a substantial muscle damage and functional impairment which was consistent with previous research employing similar protocols\textsuperscript{3,19}.

One of the novel findings of this study was the greater impairment in some, but not all, of the strength parameters following the slower velocity eccentric exercise bout. Though there are no clear mechanistic events which explain this result, the velocity-specific differences may have been facilitated by an inhibitory/protective neural mechanism which may be more prevalent during faster eccentric contraction velocities\textsuperscript{9,14,23}. In other words, a neurally-mediated inhibition of eccentric torque during higher velocity eccentric contractions may have resulted in the FST group experiencing a less severe overload stimulus, thereby facilitating less muscle swelling and a smaller decrement in strength than in the SLW group\textsuperscript{10,13,14}. This scenario is consistent with training studies that have examined strength and hypertrophy following several weeks of slow or fast velocity eccentric training\textsuperscript{1,11}. Specifically, in a previous study from our laboratory examining slow (0.52 rad.s\textsuperscript{-1}) and fast (3.14 rad.s\textsuperscript{-1}) eccentric training of the elbow flexors, we suggested that the failure of a 10-week slow velocity eccentric training protocol to induce hypertrophy and improve strength may have been facilitated by accumulative muscle damage and/or a failure to adapt to repeated slow velocity eccentric contractions\textsuperscript{1}. Another potential mechanism that may have contributed to the velocity-specific differences observed in the current study may be related to differential recruitment of the elbow flexors during slow and fast eccentric contractions. By examining changes in magnetic resonance imaging signal intensity, researchers have demonstrated that the biceps brachii is preferentially recruited during fast isotonic, eccentric contractions, whereas the brachialis is preferentially recruited during slower eccentric contractions\textsuperscript{24}. The impact and consequences of a differential recruitment pattern on additional variables such as strength remains largely unexplored.

While our data suggest that slow eccentric contractions may produce a greater decrement in some strength variables than fast eccentric contractions, other common markers of muscle damage appeared not to be influenced by the velocity of the initial damaging eccentric bout. The similar plasma CK response in FST and SLW groups suggests that both groups suffered a similar amount of structural muscle damage following the eccentric bout. Nevertheless, increases in plasma CK activity do not necessarily correlate well with the severity of muscle damage experienced and may not be a valid means of accurately quantifying muscle damage in applied research protocols where even small inter-subject differences in variables such as training history may have a large influence on plasma CK activity\textsuperscript{3,22,25}.

In the present study, the severity of muscle soreness was similar in both groups, but was evident for a longer period in the FST group. This result was conceptually consistent with a previous study that, although not equating the number of repetitions performed, reported muscle soreness following an
Differential expression of muscle damage...

isotonic bout of fast, but not slow, eccentric contractions of the elbow flexors\(^24\). It is possible that the use of pressure algometry to quantify muscle soreness at a specific location may not have been representative of the other areas which may have experienced more or less DOMS. However, this design does allow for the specific comparison of the same region of muscle, the only difference being the velocity of the damaging eccentric bout.

In contrast to the development of muscle soreness, the increase in upper arm girth following eccentric exercise was greater in the SLW group and did not correspond with the greater expression of muscle soreness in the FST group. While an increase in upper arm girth immediately following eccentric exercise is largely due to a transient increase in peripheral blood flow, a prolonged increase in girth is most likely attributable to muscle oedema and swelling\(^26,27\). Early research suggested that increased interstitial pressure may activate free nerve endings, thereby contributing to muscle soreness\(^28\). However, consistent with the present study, several investigators have been unable to confirm a relationship between oedema and muscle soreness\(^21,29,30\). This disparity may be due to the choice of muscle group investigated and the associated variation in the compliance of different anatomical compartments such as the elbow flexor (compliant) and anterior tibial (non-compliant) compartments\(^30\).

Finally, it must be noted that, while isokinetic contractions represent a convenient and easily quantifiable means of measuring strength, they bear little similarity to most muscle contractions performed during athletic events or activities of daily living\(^31\). Consequently, the practical implications of this study are perhaps necessarily limited to situations in which isokinetic contractions can be performed. This may include the use of eccentric contractions for rehabilitative purposes such as the treatment of tendonosis\(^32,33\).

In conclusion, the contraction velocity of an acute bout of eccentric exercise influences the magnitude and time course of several indirect markers of muscle damage. Fast eccentric contractions resulted in a longer period of muscle soreness, whereas slow eccentric contractions were associated with a greater increase in arm girth/swelling and a greater decrement in the ability to generate concentric and eccentric torque.

**Acknowledgments**

Our sincere thanks are extended to Margaret Barber, Robert Michaels and all our subjects for their valuable contributions to this project. This study was supported by funding from the Gatorade Sports Science Institute.

**References**

Differential expression of muscle damage...


