Joint angle-torque characteristics of the knee extensors following eccentric exercise-induced muscle damage in young, active women

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Abstract

Functional disruption following eccentric exercise-induced muscle damage is characterized by an immediate and prolonged loss in force-generating capacity, a disproportionate loss of strength at short muscle lengths, and a rightward shift in muscle length-tension relationship, in favor of a longer muscle length for optimal force generation. The purpose of this study was to examine progressive changes in muscle joint angle-torque characteristics following strenuous eccentric exercise in the quadriceps group, in female athletes. Seventeen physically active female athletes completed eccentric exercise designed to induce muscle damage. Isokinetic dynamometry was used to determine characteristics of muscle strength loss for the following dependent variables: change in maximal force, force at optimal and short muscle lengths, and shift in length-tension relationship at 1 hour, 24 hours, 48 hours, 72 hours, and 96 hours following damaging exercise. Perceived soreness and creatine kinase activity were also measured at these times. Significant increases in perceived soreness and creatine kinase activity, and a significant reduction in force-generating capacity were observed. A greater strength loss at short muscle lengths was observed following damaging exercise [17.2% vs. 25.7% relative force reduction for optimal and short (71° and 21° from full extension) muscle lengths, respectively]. A significant rightward shift in optimal joint angle for force generation was also observed, indicating that maximal force was generated at a longer muscle length subsequent to damaging exercise. This paper demonstrates a progressive rightward shift in the muscle length tension relationship.

Keywords: Muscle damage; Soreness; Strength loss

Introduction

It is well documented that unaccustomed and predominantly eccentric exercise leads to symptoms of exercise-induced muscle damage (EIMD), including increases in perceived soreness, rating of perceived exertion, and circulating myoprotein activity, as well as a decrease in time to exhaustion during exercise. EIMD is also characterized by reductions in muscle strength and a shift in the length-tension relationship of the muscle. Although the exact mechanisms of EIMD are not fully known, the most likely initial event in injury is related to the mechanical disruption of the muscle during contraction, particularly during eccentric actions.

It is postulated, that due to their nonuniformity of length, sarcomeres within the same myofibril function at different points of the LT curve during muscular contraction. During eccentric muscle actions, a greater number of sarcomeres function on the plateau or descending limb of the LT curve, at a point beyond their optimum force-generating length, an effect that does not occur during shortening actions because there is no overextension of sarcomeres. Once overextended, sarcomeres are unable to generate tension and lengthen rapidly.
until a point where lengthening is halted by rising passive tension in the remainder of the muscle fiber. Consequently, sarcomeres that are able to generate force must compensate for those that cannot, increasing the force generating requirements of these sarcomeres, and subsequently, making them more susceptible to damage. This is exacerbated during eccentric contractions when the muscle is at a long length, such as in the knee extensors during walking downstairs or downhill walking and running. Nonuniform, rapid lengthening of sarcomeres can continue throughout contraction, progressing from weakest to strongest. Upon completion of contraction, most sarcomeres will reinterdigitate and resume normal function, although some fail to reinterdigitate, and disruption of force-generating structures is often observed following strenuous exercise. This can lead to spontaneous muscle shortening, which may be a result of changes in calcium ion concentration, and can be observed through changes in resting joint angle.

Damaging eccentric exercise is often characterized by a shift in the LT relationship of the muscle, with the point of optimal force generation occurring at a longer muscle length. This phenomenon was initially observed by Katz in animal models. Although the shift in the LT relationship has been shown in several studies, reports that show a detailed shift of the LT relationship across subsequent 24-hour periods are limited. Several studies have also inferred a shift in optimal muscle length from isometric contraction data, which indicate a disproportionate strength loss at short muscle lengths. Studies that have assessed the shift in the LT relationship of the muscle, with the point of optimal force generation occurring at a longer muscle length. This phenomenon was initially observed by Katz in animal models. Although the shift in the LT relationship has been shown in several studies, reports that show a detailed shift of the LT relationship across subsequent 24-hour periods are limited. Several studies have also inferred a shift in optimal muscle length from isometric contraction data, which indicate a disproportionate strength loss at short muscle lengths. Other studies have observed the shift in the LT relationship in animal models. Studies that have assessed the shift in the LT relationship in animal models. Other studies have observed the shift in the LT relationship in animal models. Studies that have assessed the shift in the joint angle-torque relationship in humans have also primarily involved male participants and these studies did not assess changes in the knee extensors.

To date, there is a paucity of data that show the absolute change in daily peak torque in conjunction with the shift in the LT relationship, following a bout of plyometric exercise, in young and active female participants. The purpose of this study was therefore to examine the LT characteristics of strength loss in the knee extensors following strenuous eccentric activity in young, physically active women, in order to investigate the temporal pattern of muscle function characteristics over a prolonged time course. A further unique aspect of this study was to assess the torque changes across a dynamic range of knee extension assessed during isokinetic dynamometry. Following the bout of plyometric exercise, we hypothesized that there would be an immediate decrease in peak torque accompanied by an initial rightward shift in muscle LT relationship, and there would be a further shift in joint angle-torque relationship, which would be observable over the 4-day course of the investigation.

Materials and methods

Participants

Data were collected from 17 physically active (minimum three occasions × 30 minutes per week) female volunteers (age, 21.5 ± 2.2 years; stature, 1.66 ± .054 m; and mass, 64.1 ± 6.1 kg), who were free from injury and were not engaged in specific lower limb weight training or plyometric exercise. The sample used in this study was sufficient to exceed 80% power, based on results from a previous study investigating strength loss following strenuous exercise. Participants provided signed informed consent to participate in the study, which had received approval from the University of Exeter School of Sport and Health Sciences ethics committee. Volunteers were asked to maintain normal hydration and food intake patterns, and were instructed to refrain from alcohol, nonsteroidal anti-inflammatory drugs, and nutritional supplements for the duration of the test. Participants were also asked to avoid any exercise or therapeutic treatments that may have affected their normal recovery pattern. Data pertaining to all indices of muscle damage were collected prior to, and 24 hours, 48 hours, 72 hours, and 96 hours after muscle damage. Data were also collected 1 hour after plyometric exercise in order to provide an indication of immediate muscle strength loss while minimizing the potentially confounding factor of acute fatigue following the exercise protocol.

Design

Following the collection of baseline data, participants completed a plyometric drop jump protocol, designed to induce muscle damage similar to that experienced following training or competition in a number of sports.

Muscle soreness

Perceived soreness of the knee extensors was assessed using a 10-cm visual analog scale, with 0 indicating no pain, and 10 indicating the worst soreness experienced following exercise. Briefly, participants were instructed to complete an unweighted squat, holding a knee angle of approximately 90° for a period of 2 seconds, and mark perceived soreness on the visual analog scale. The distance from 0 (no pain) was measured to give an indication of perceived soreness following exercise. This method has been used successfully in previous studies.

Plasma creatine kinase

After cleaning a finger with a sterile alcohol swab, a capillary puncture was made using a Hemocue lancet (Hemocue, Sheffield, UK). A 30-µL blood sample was separated by centrifuge and analyzed using spectrophotometry (Jenway Ltd., Dunmow, UK) in accordance with manufacturer guidelines (Randox Laboratories, Co. Antrim, UK).

Assessment of strength across joint angle

Isokinetic dynamometry (Biodex System 3; Biodex Medical Systems Inc., New York, USA) was used to determine muscle strength and the shift in muscle LT relationship. Participants were seated upright with the upper body and the
experimental leg secured to reduce extraneous movement. The axis of rotation of the knee of the dominant leg was aligned with the dynamometer, and body position was recorded and maintained throughout the experimental period. All participants completed five consecutive, maximal voluntary isokinetic concentric contractions at 60 deg.sec\(^{-1}\) through an 80\(^\circ\) range of movement from knee flexion on the dominant leg. The best of five maximal, gravity-corrected concentric contractions of the knee extensors was taken as the criterion measure of muscle strength. Upon completion of contractions, analysis of participant torque data through the full range of knee extension indicated that average peak knee extensor muscle torque was generated at 71 (±1.4)\(^\circ\) from full knee extension. This value was taken as the optimal angle for force generation. Isokinetic muscle torque at short length was assessed at 21\(^\circ\) of knee extension, which represented the point at which force generation was 30% of maximum at baseline, in accordance with the methods of Byrne et al.\(^1\)

**Plyometric exercise protocol**

Participants performed 10 × 10 plyometric drop jumps from a 0.6-m box. They were instructed to stand erect on the box, step off with one foot, land both feet together, adopt an approximately 90\(^\circ\) squat position, prior to performing a maximal vertical jump. Jump repetitions and sets were separated by 10 seconds and 1 minute, respectively. This exercise protocol has been used previously to induce minor muscle damage.\(^2\)\(^1\)\(^2\)\(^3\) The plyometric jumping protocol was demonstrated and monitored by an experienced strength and conditioning practitioner.

**Analysis of data**

Joint angle-dependent changes in torque were analyzed using a two-factor, 2 × 6 (joint angle × time) repeated measures analysis of variance (ANOVA). A one-way repeated-measures ANOVA was used to analyze creatine kinase activity, perceived soreness, and optimal muscle torque (SPSS version 15.0; SPSS Inc., Chicago, IL, USA). Plasma creatine kinase activity data were log transformed to satisfy assumptions of sphericity. For all variables, the Mauchly sphericity test was applied to test for homogeneity of variance. Where this was not confirmed, the Greenhouse-Geisser value was used to adjust degrees of freedom to increase the critical F-ratio. A modified Tukey post hoc analysis was used to examine changes in peak torque and shifts in joint angle-torque relationship across time. Significance was set at \(p \leq 0.05\) a priori. (All data are presented as ± standard error of the mean).

**Results**

**Creatine kinase**

Creatine kinase activity peaked at 24 hours following exercise, and returned to baseline levels after 72 hours [(F\(_3\), \text{49.} = 26.9, p < 0.01), Table 1].

**Muscle soreness**

Muscle soreness peaked at 48 hours following exercise, and returned to baseline levels at 96 hours [(F\(_5\), \text{80.} = 90.0, \(p < 0.01\)), Table 1].

**Muscle strength by joint angle**

Significant main effects of time (F\(_3\), \text{72.} = 28.8, \(p < 0.01\)) and joint angle (short vs. optimal; F\(_1\), \text{24.} = 4.2, \(p \leq 0.05\)) effects were also observed on peak isokinetic muscle torque following plyometric exercise. There was no time × joint-angle (short vs. optimal) interaction effect on peak torque (F\(_3\), \text{72.} = 1.8, \(p > 0.05\)). Tukey post hoc analysis indicated that the proportion of peak torque compared to baseline was significantly lower at 1 hour, 24 hours, 48 hours, 72 hours, and 96 hours following the muscle-damaging plyometric protocol. The proportional loss in peak torque was also greater for short muscle length at each time point (Fig. 1).

The optimal joint angle (muscle length) for generating peak torque changed across time (F\(_5\), \text{80.} = 2.8, \(p < 0.05\)). Follow-up analysis indicated that the optimal muscle length was different at all time points following damaging exercise. The shift in optimal muscle length was accompanied by a significant reduction in isokinetic muscle function (F\(_2\), \text{5.} = 29.7, p < 0.01; Fig. 2).

Peak torque occurred at 71 (±1.4)\(^\circ\) from full knee extension prior to damaging exercise, with a peak muscle torque of 152.9 (±5.6) N.m\(^{-1}\). Following EIMD, significant decreases in peak isokinetic muscle force and a rightward shift in the

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**Table 1**

<table>
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<tr>
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<th>Pre</th>
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<th>24</th>
<th>48</th>
<th>72</th>
<th>96</th>
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<tbody>
<tr>
<td>Maximum torque (N.m)</td>
<td>152.9 (5.6)</td>
<td>120.6 (5.9)(^a)</td>
<td>116.1 (6.1)(^b)</td>
<td>103.4 (6.3)(^b)</td>
<td>120.9 (7.3)(^b)</td>
<td>131.9 (7.3)(^b)</td>
</tr>
<tr>
<td>Optimal angle ((^\circ))</td>
<td>71 (1.4)</td>
<td>75.5 (1.8)(^a)</td>
<td>75.3 (1.4)(^b)</td>
<td>75.3 (1.6)(^b)</td>
<td>76.5 (1.3)(^b)</td>
<td>77.7 (2.0)(^b)</td>
</tr>
<tr>
<td>Torque at optimal angle (71(^\circ); N.m)</td>
<td>152.9 (5.6)</td>
<td>120.3 (6.0)(^a)</td>
<td>115.7 (6.1)(^b)</td>
<td>103.0 (6.3)(^b)</td>
<td>119.9 (7.6)(^b)</td>
<td>129.6 (6.9)(^b)</td>
</tr>
<tr>
<td>Soreness</td>
<td>0.5 (0.1)</td>
<td>2.9 (0.4)(^a)</td>
<td>6.1 (0.4)(^b)</td>
<td>7.0 (0.3)(^b)</td>
<td>3.9 (0.3)(^b)</td>
<td>1.3 (0.1)(^b)</td>
</tr>
<tr>
<td>CK (U.l(^{-1}))</td>
<td>109.0 (7.6)</td>
<td>148.3 (12.4)(^a)</td>
<td>277.5 (29.7)(^b)</td>
<td>154.3 (14.3)(^b)</td>
<td>106.1 (9.9)</td>
<td>107.0 (12.2)</td>
</tr>
</tbody>
</table>

Data are presented as mean (±standard error of the mean).

CK = creatine kinase.

\(^a\) Peak torque at the initial optimal angle is also presented.

\(^b\) Represents significant difference from baseline (\(p < 0.05\)).
joints. Observations of an actual, rather than an inferred shift in the LT relationship in the human knee extensors are sparse, and recognition of a continuing progressive rightward shift in the LT relationship is lacking. In the current study, the use of isokinetic dynamometry to observe dynamic torque changes during knee extension at 60 deg.sec\(^{-1}\) allowed the change in strength loss following muscle damage to be presented alongside the observed shift in LT relationship. These data support previous observations that the LT curve shifts rightward following eccentric exercise.

The initial shift in the LT relationship is most likely caused by an increase in muscle compliance as a result of disruptions to individual sarcomeres resulting from strain following eccentric exercise.\(^\text{24}\) In this study, an immediate initial shift in LT relationship is observed, which was maintained for 48 hours. Further progressive shifts were observed at 72 hours and 96 hours (Table 1). More prolonged shifts in the LT relationship may be indicative of muscle sarcomeres that are still deformed, but may also be attributed to an increase in the number of sarcomeres in series.\(^\text{25}\) These data indicate that the rightward shift in the LT relationship remains significant 96 hours after the bout of plyometric exercise and may indicate an addition of sarcomeres within the muscle fiber, as suggested by Morgan and Proske.\(^\text{25}\) Although the addition of sarcomeres following EIMD has been observed in animal muscle following downhill running,\(^\text{26}\) evidence indicating the addition of muscle sarcomeres in such a short time frame in human muscle is not available, and should be considered in future research. Though shifts in LT relationship may be associated with changes in the stiffness properties of the muscle and tendon, observations that the change in optimal torque generating length occurs without a change in muscle length,\(^\text{9}\) and that changes in muscle compliance are not accompanied by changes in tendon properties cast doubt upon this assertion, although an increase in muscle stiffness may increase individual susceptibility to further EIMD.\(^\text{27}\)

The shift in LT relationship may contribute to the explanation of disproportionate strength loss observed at short muscle lengths, with the rightward shift resulting in fewer myofilament cross-bridges being formed while the muscle is at a short length.\(^\text{25}\) Alternatively, changes in the excitation-contraction coupling process may affect calcium release and in turn affect force generating capability, particularly at short muscle lengths. Balog\(^\text{28}\) has recently described that a fall in intracellular calcium resulting from reduced calcium release from the sarcoplasmic reticulum following fatiguing exercise has a relatively small effect on high force elicited at high stimulation frequencies, such as those at optimal muscle lengths. At lower stimulation frequencies, the

Fig. 1. Changes in peak torque at short (21°) and optimal (71°) joint angles across time. *Represent significant difference compared to baseline; + represents significant difference between joint angle (\(p < 0.05\)).

Discussion

Following the bout of plyometric exercise, significant increases in perceived soreness and creatine kinase activity were observed. The temporal pattern of these changes is consistent with previous studies, which have examined EIMD in active female participants.\(^\text{21,22}\) Alterations in the muscle LT relationship, which in particular is regarded to be one of the best indicators of EIMD,\(^\text{24}\) in combination with the increase in soreness and creatine kinase levels, confirmed the occurrence of muscle damage in the female participants in this study.

In the current study, decrements in peak isokinetic torque were immediately apparent, although a comparison of data from studies investigating postexercise strength loss at different muscle lengths is difficult due to the lack of studies that have investigated these characteristics in human knee extensor muscle. Byrne et al\(^\text{1}\) indicated torque losses of 23.7 ± 2.5% versus 17.9 ± 2.7% at short versus optimal muscle lengths respectively, immediately following muscle damaging exercise. The current data indicate comparable strength losses of 29.7 ± 4.6% versus 19.4 ± 2.6% at short and optimal muscle lengths immediately (1 hour) following damaging exercise (Fig. 1). A significant difference in strength loss between short and optimal muscle lengths persists throughout the testing period (\(p < 0.05\)), which is also consistent with the observations of Byrne et al.\(^\text{1}\)

Greater strength losses at shorter muscle lengths have been used to infer a rightward shift in the LT relationship of the muscle to favor a longer optimal tension-generating length.\(^\text{3}\) Observations of an actual, rather than an inferred shift in the LT relationship in the human knee extensors are sparse, and recognition of a continuing progressive rightward shift in the LT relationship is lacking. In the current study, the use of isokinetic dynamometry to observe dynamic torque changes during knee extension at 60 deg.sec\(^{-1}\) allowed the change in strength loss following muscle damage to be presented alongside the observed shift in LT relationship. These data support previous observations that the LT curve shifts rightward following eccentric exercise.

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Fig. 2. Shift in optimal muscle length 1 hour, 24 hours, 48 hours, 72 hours, and 96 hours (A–E, respectively) after exercise-induced muscle damage. The letters A and B in each figure panel represent the points of peak isokinetic torque at baseline and after the plyometric exercise for each time point.
same decline in intracellular calcium results in a more substantial decline in force, with the rightward shift in the LT relationship exacerbating force decline for a given stimulation frequency.\textsuperscript{28,30}

The influence of changes in neuromuscular function should be considered as a further factor in the disproportionate muscle strength loss observed in this study. It has been observed that twitches at short muscle lengths are shorter in duration and smaller than at long muscle lengths, and therefore require higher frequencies of stimulation to produce a given force.\textsuperscript{28,31} More neural drive may consequently be required to produce maximal muscle activation at short muscle lengths.\textsuperscript{58} There is some evidence to suggest that neural drive is inhibited following damaging exercise to prevent further damage.\textsuperscript{32} In this case, the required increase in neural stimulation may be inhibited, and would be particularly manifest at short muscle lengths as a result of the frequency of stimulation required to fully activate the muscle.

In conclusion, this study observed rather than inferred a progressive and long-lasting rightward shift in muscle LT relationship toward a longer optimal muscle length following eccentric EIMD. The change in torque characteristics was assessed during dynamic knee extension using isokinetic dynamometry. As the shift in LT relationship was apparent until the final observation point of this study, the duration of shift in length-tension relationship remains unclear. A continued observation beyond the 96-hour time frame used in this study may provide further information pertaining to the duration of LT shift, and the time-course of adaptation to this type of exercise. A disproportionate loss of force at a short muscle length was also observed, which may be a result of reductions in the ability to form myofilament cross-bridges, or alterations in the excitation-contraction coupling process, due to a reduction in intracellular calcium availability. Although similar LT changes have been observed previously, data pertaining to progressive, day-to-day shifts in muscle LT relationship are rare, and to the best of our knowledge this is the first such observation in female, human knee extensor muscle.

Conflicts of interest

The authors perceive there to be no conflicts of interest related to this study.

Acknowledgments

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