Residual force enhancement following eccentric induced muscle damage

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ABSTRACT

During lengthening of an activated skeletal muscle, the force maintained following the stretch is greater than the isometric force at the same muscle length. This is termed residual force enhancement (RFE), but it is unknown how muscle damage following repeated eccentric contractions affects RFE. Using the dorsiflexors, we hypothesised muscle damage will impair the force generating sarcomeric structures leading to a reduction in RFE. Following reference maximal voluntary isometric contractions (MVC) in 8 young men (26.5 ± 2.8 y) a stretch was performed at 30 °/s over a 30° ankle excursion ending at the same muscle length as the reference MVCs (30° plantar flexion). Surface electromyography (EMG) of the tibialis anterior and soleus muscles was recorded during all tasks. The damage protocol involved 4 sets of 25 isokinetic (30 °/s) lengthening contractions. The same measures were collected at baseline and immediately post lengthening contractions, and for up to 10 min recovery. Following the lengthening contraction task, there was a 30.3 ± 6.4% decrease in eccentric torque (P < 0.05) and 36.2 ± 9.7% decrease in MVC (P < 0.05) compared to baseline. Voluntary activation using twitch interpolation and RMS EMG amplitude of the tibialis anterior remained near maximal without increased coactivation for MVC. Contrary to our hypothesis, RFE increased (~100–250%) following muscle damage (P < 0.05). It appears stretch provided a mechanical strategy for enhanced muscle function compared to isometric actions succeeding damage. Thus, active force of cross-bridges is decreased because of impaired excitation–contraction coupling but force generated during stretch remains intact because force contribution from stretched sarcomeric structures is less impaired.

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1. Introduction

Force generation is highly dependent upon the muscle's contractile history, such that isometric force of activated skeletal muscles following stretch is greater than that produced at the same muscle length prior to stretch (Abbott and Aubert, 1952; Rassier et al., 2003). Increased force during the isometric steady state phase following stretch when compared with values at the same length without a prior stretch is termed residual force enhancement (RFE). Mechanisms of RFE are believed to be a combination of both active and passive properties of muscle force generating and transmitting structures (Herzog and Leonard, 2002). Force enhancement is evident in various preparations (Joumaa et al., 2008; Julian and Morgan, 1979; Leonard et al., 2010; Rassier et al., 2003; Rassier and Pavlov, 2012; Telley et al., 2006) and also in studies on humans (Lee and Herzog, 2002; Pinniger and Cresswell, 2007; Tilp et al., 2009). Whereas stretch leads to RFE, repeated unaccustomed lengthening contractions result in muscle damage, by decreasing force generation through mechanical disruption of sarcomeres (Hough, 1900) and impairing excitation–contraction (E–C) coupling (Allen, 2001). Because both muscle actions have opposing effects on muscle force generation it is important to understand the influence of muscle damage on RFE.

Proposed mechanisms of the muscle's history dependent behaviour leading to RFE are not fully understood [for review see (Campbell and Campbell, 2011; Edman, 2012)]. Residual force enhancement initially was thought to occur through development of sarcomere length non-uniformities that increased the amount of filament overlap thereby elevating force production (Julian and Morgan, 1979). Current suggested mechanisms of RFE include an increased proportion of strongly bound cross-bridges following stretch (Rassier and Herzog, 2004), increased average steady state force per cross-bridge, and the engagement of force transmitting elements (Edman et al., 1982; Herzog and Leonard, 2002), but there is no consensus on these concepts. Recently, the role of the contractile protein titin, has gained considerable attention as a Ca2+ modulated contributor to force enhancement following stretch (Herzog et al., 2012). Because RFE may depend on the same structures that are damaged following repeated lengthening...
contractions, there is potential for impaired RFE following eccentric exercise. The initial insult of muscle damage causes impaired isometric force due to damaged and overstretched sarcomeres failing to re-interdigitate (Morgan and Proske, 2004). This increases series compliance (Gregory et al., 2007), shifting the force-length relationship to longer muscle lengths for optimal force production. As well, impaired E–C coupling (Ingalls et al., 1998; Warren et al., 2001) through reduced Ca\(^{2+}\) release and decreased myofibrillar Ca\(^{2+}\) sensitivity (Balog, 2010; Bruton et al., 2008) leads to impaired torque production and low-frequency torque depression (Bruton et al., 2008; Edwards et al., 1977).

Residual force enhancement has been observed previously in the human tibialis anterior muscle (Pinniger and Cresswell, 2007; Tilp et al., 2009). In addition, following lengthening induced damage of the tibialis anterior, voluntary activation and neuromuscular activation (i.e. EMG amplitude) are well maintained (Power et al., 2010, in press). Hence, dorsiflexion provides an apt model to study the effects of muscle damage on RFE in humans. This study aims to determine changes in neuromuscular properties of the dorsiflexors following damaging lengthening contractions. We hypothesised muscle damage would severely impair force generating and transmitting structures leading to a reduction in RFE.

2. Materials and methods

2.1. Participants

All participants were healthy and free from musculoskeletal disorders (eight males; 26.5 ± 2.8 y, 178.1 ± 3.4 cm, 82.6 ± 7.9 kg). This study was approved by the local University’s Review Board for Research Involving Human Subjects and conformed to the Declaration of Helsinki. Informed, oral and written consent was obtained from all participants before testing.

2.2. Experimental arrangement

All testing was conducted on a HUMAC NORM dynamometer (CSMi Medical Solutions, Stoughton, MA) using the non-dominant foot. Participants sat in a slightly reclined position with the hip, knee, and ankle angles at 100°, 140°, and 30° plantar flexion (PF), respectively. All voluntary and evoked isometric dorsiflexion contractions were performed at an ankle joint angle of 30° of PF. Lengthening contractions began at the neutral ankle angle (0° PF) until 30° of PF.

2.3. Electromyography

(EMG) Electromyographic signals were collected using self-adhering Ag–AgCl surface electrodes (1.5 × 1 cm; Kendall, Mansfield, MA). Prior to electrode placement, the skin was cleaned aggressively with pre-soaked alcohol swabs. A monopolar electrode configuration was used with the active electrode positioned over the proximal portion of the tibialis anterior at the innervation zone (~7 cm distal to the tibial tuberosity and ~2 cm lateral to the tibial anterior border) (Botter et al., 2011) and a reference surface electrode was placed over the distal tendinous portion of the tibialis anterior at the malleoli. The surface active electrode for the soleus was positioned ~2 cm distal to the lower border of the medial head of the gastrocnemius and a reference was placed over the calcaneal tendon.

2.4. Experimental procedures

Stimulated contractions of the dorsiflexors were evoked electrically with two round carbon rubber electrodes (3 cm diameter; Emergi, St. Paul, Minnesota, USA), coated in conductive gel positioned at the optimal site for twitch torque and secured with tape. The anode was positioned anterior, and the cathode posterior to the fibular head over the deep branch of the common fibular nerve. The operator applied manual pressure over the cathode during stimulation. A computer-triggered stimulator (model DSTAH, Digitimer, Welwyn Garden City, Hertfordshire, UK) set at 400 V provided the electrical stimulation using a pulse width of 100 μs. Peak twitch torque (P\(_{\text{t}}\)) was determined by increasing the current until a plateau in dorsiflexor P\(_{\text{t}}\) and tibialis anterior compound muscle action potential (M-wave) peak to peak amplitude were reached, and then the current was further increased by at least 15% to ensure supramaximal stimulation. This stimulation intensity was used for the evoked doublet (P\(_{\text{dt}}\)) (two pulses at a 10 ms interpulse interval) to assess voluntary activation. Finally, a 10 Hz (5 pulses over 0.5 s) and 50 Hz stimulus (25 pulses over 0.5 s) train was delivered to assess peak tetanic torque by increasing the current until there was a plateau in evoked 50 Hz torque (Experimental timeline; Fig. 2).

Two reference baseline maximal voluntary isometric contractions (MVC) were performed, each of 3–5 s duration with 3 min of rest between each contraction. Participants were provided visual feedback of the torque tracing on a computer monitor, and were exhorted verbally during all voluntary efforts. Voluntary activation was assessed using the modified interpolated twitch technique (Gandevia, 2001). Values from the MVC with the highest peak torque were recorded. Next, electrical stimulations at tetanic frequencies were delivered to determine 10 Hz and 50 Hz peak

Fig. 1. Schematic diagram of the experimental set up. Participants sat in a slightly reclined position with the hip, knee, and ankle angles set at 100°, 140°, and 30° plantar flexion (PF), respectively. All voluntary and evoked isometric dorsiflexion contractions were performed at an ankle joint angle of 30° of PF. Lengthening contractions began at the neutral ankle angle (0° PF) until 30° of PF.
torque ratio. The residual force enhancement protocol (Fig. 3) involved maximal activation of the dorsiflexors at the neutral ankle angle (0°) for 1 s, followed by a 1 s stretch at 30°/s, ending with a 3 s isometric hold at the same ankle angle as the reference MVC (30° PF). Although the magnitude of RFE is based on stretch amplitude (Edman, 2012), we chose in this study to end the stretch at 30° PF to optimise isometric torque production, and to highlight strength deficits following damage at the optimal muscle length for torque production.

2.5. Damage protocol

Participants performed 4 sets of 25 eccentric isokinetic dorsiflexion contractions at 30°/s through a 30° range of motion with each set separated by ~30 s of rest. The voluntary and electrically evoked responses of the dorsiflexors were recorded at baseline, during the protocol, immediately following each of the 4 sets, immediately following task termination (Post), and during recovery at 5 and 10 min (Fig. 2).

Fig. 2. Schematic diagram of the experimental timeline. Grey bars are isometric maximum voluntary contractions (MVC). Open torque profiles are electrically evoked contractions (twitches, doublet, 10, 50 Hz). Open arrows are electrically evoked twitches; and filled arrows are electrically evoked doublets. The stretch protocol used in the calculation of residual force enhancement (RFE) was performed through a 30° range of motion at 30°/s. Recovery time points: immediately post, 5 and 10 min.

Fig. 3. (A) Raw data depicting the calculation of residual force enhancement at baseline. (B) Residual force enhancement following muscle damage.
2.6. Data reduction and analysis

Torque and position data were sampled at 500 Hz. Signals were converted to digital format using a 12-bit analogue-to-digital converter (model 1401 Power, Cambridge Electronic Design, Cambridge, UK). Residual force enhancement (defined as the per cent increase in isometric torque following stretch, relative to the reference MVC) was calculated by determining the mean torque value over 0.5 s around the peak torque of the reference MVC, and divided by the mean torque value for 0.5 s during the steady state of the MVC following the stretch (Fig. 3). Surface EMG signals were pre-amplified (× 100), amplified (× 2), band-pass filtered (10–1000 Hz), and sampled online at 2500 Hz using Spike 2 software (version 6.10, Cambridge Electronic Design Ltd). Tibialis anterior and soleus EMG were recorded during the dorsiflexor reference MVC and expressed as a root mean squared (RMS) value over a 0.5 s epoch about the peak torque. Following stretch, EMG RMS amplitude was analysed over a 0.5 s epoch. Soleus EMG during those periods was used to calculate antagonist coactivation as a soleus/tibialis anterior EMG ratio × 100% (Power et al., 2010).

2.7. Statistical analysis

Using SPSS software (version 16, SPSS Inc. Chicago, IL) a repeated measures ANOVA was performed. The level of significance was set at \( P < 0.05 \). When a significant effect was observed a post hoc analysis using unpaired t-tests was performed with a Bonferroni correction factor to determine where significant differences existed. A power calculation was performed to ensure there was sufficient power (1–0.91 to 0.95) to detect significant differences in RFE. Results in the table are presented as means ± standard deviations (SD), and figures as means ± standard errors (SE) values.

3. Results

3.1. Baseline values and residual force enhancement

 Eccentric torque values were ~40% greater than those during the isometric contractions (Table 1). Torque was consistently higher in the steady state (isometric) phase following stretch (44.2 ± 12.7 Nm) compared to the reference isometric MVC (39.4 ± 9.8 Nm) (\( P < 0.05 \)) (Fig. 4) resulting in a mean force enhancement of 10.3 ± 5.5%. RMS EMG amplitude and coactivation for the tibialis anterior and soleus muscles were the same for the isometric reference MVC and the steady state isometric phase following stretch (\( P > 0.05 \)). Baseline measures are presented in Table 1.

3.2. Muscle damage protocol

Participants completed all contractions of the protocol resulting in a 29.3 ± 6.4% decrease in peak eccentric torque (\( P < 0.05 \)) and 30.9 ± 11.3% decrease in MVC torque (\( P < 0.05 \)) compared to baseline. Concurrently, RMS EMG amplitude of the tibialis anterior decreased by 22.1 ± 19.5% throughout the protocol relative to MVC values obtained at baseline (\( P < 0.05 \)), and coactivation was similar to values obtained at baseline (\( P > 0.05 \)).

3.3. Markers of damage

Participants reported mild to no muscle soreness for up to 10 min into recovery, but during the two days following the protocol, all reported muscle soreness which peaked at 24 hr and returned to mild soreness at 48 hr. With a significant effect of time (\( P < 0.05 \)), dorsiflexion MVC torque decreased during the task (~36%), and remained blunted throughout recovery (30.9 ± 11.3%) (\( P < 0.05 \)) (Fig. 5). The slight, albeit, non-significant recovery of the reference MVC can be attributed to the transient effects of muscle fatigue which are recovered fully by 10 min following lengthening contractions, and the remaining torque

Table 1

<table>
<thead>
<tr>
<th>Electrically Evoked Contractions</th>
<th>Peak Twitch Torque</th>
<th>10 Hz Torque</th>
<th>50 Hz Torque</th>
<th>Voluntary Activation</th>
<th>MVC Per cent Co-Activation</th>
<th>Reference MVC Torque</th>
<th>Steady State MVC Torque</th>
<th>Peak Torque During Stretch</th>
<th>Residual Force Enhancement</th>
<th>Passive Force Enhancement</th>
</tr>
</thead>
<tbody>
<tr>
<td>MVC Preceding Stretch</td>
<td>5.6 ± 1.9 Nm</td>
<td>15.3 ± 3.1 Nm</td>
<td>22.3 ± 4.0 Nm</td>
<td>96.5 ± 3.7%</td>
<td>20.0 ± 6.6%</td>
<td>39.4 ± 9.8 Nm</td>
<td>44.2 ± 12.7 Nm</td>
<td>55.0 ± 12.4 Nm</td>
<td>10.3 ± 5.5%</td>
<td>1.4 ± 0.66 Nm</td>
</tr>
<tr>
<td>MVC Succeeding Stretch</td>
<td>7.1 ± 1.9 Nm</td>
<td>17.7 ± 3.1 Nm</td>
<td>24.2 ± 4.0 Nm</td>
<td>98.5 ± 3.7%</td>
<td>20.0 ± 6.6%</td>
<td>39.4 ± 9.8 Nm</td>
<td>44.2 ± 12.7 Nm</td>
<td>55.0 ± 12.4 Nm</td>
<td>10.3 ± 5.5%</td>
<td>1.4 ± 0.66 Nm</td>
</tr>
</tbody>
</table>

Fig. 4. (A) Residual force enhancement. (B) Isometric strength following muscle damage expressed relative to baseline preceding stretch. (C) Isometric strength during the steady state succeeding stretch following muscle damage expressed relative to baseline. Maximal voluntary isometric strength was reduced more than isometric strength following stretch leading to an increase in force enhancement. Effect of time (*) values are Means ± SE.
deficit being attributed to muscle damage (Power et al., 2010, in press). Voluntary activation was >95% (Table 1) at baseline and did not change significantly following task termination and throughout recovery ($P > 0.05$). The incomplete recovery of MVC and elevated soreness suggests that muscle damage was present (Clarkson and Hubal, 2002; Warren et al., 1999).

3.4. Residual force enhancement following muscle damage

Voluntary activation ($P > 0.05$) and RMS EMG ($P > 0.05$) of the tibialis anterior remained near maximal without increased coactivation (soleus:tibialis anterior) during all dorsiflexion contractions ($P > 0.05$) throughout recovery. Contrary to our hypothesis, RFE was increased significantly ($P < 0.05$) following muscle damage (~100–250%) (Fig. 4) in that torque values were consistently higher in the steady state (isometric) phase following stretch compared to the reference isometric MVC. Residual force enhancement was increased following damage 25.0 ± 9.8% due to a 10% greater decline in reference MVC (39.4–25.3 Nm) compared to steady state torque following stretch (44.2–32.0 Nm). Passive force enhancement was calculated as the difference between resting torque after stretch and resting torque after a reference MVC. Passive force enhancement contributed ~25–30% of force enhancement and was similar at baseline (1.4 ± 0.66 Nm) and was unchanged following muscle damage (1.10 ± 0.39 Nm) ($P > 0.05$).

3.5. Excitation contraction coupling disruption

Low frequency torque depression (10:50 Hz) presented a significant effect for time ($P < 0.05$) and decreased to 71.3 ± 11.3% of baseline by 10 min. The alterations in the 10:50 Hz ratio were manifested by the greater reduction in 10 Hz evoked torque compared with the 50 Hz (Fig. 6a–c). The 10 Hz torque was significantly reduced by 40.5 ± 16.1% throughout recovery and 50 Hz was decreased by 16.6 ± 5.1% ($P < 0.05$). Thus, there was significant low frequency torque depression following the lengthening contractions and damage induced impairments in E–C coupling. For $P_t$ there was a main effect for time ($P < 0.05$). Twitch torque was potentiated to 137.6 ± 28.1% following the first set of lengthening contractions (Fig. 7), but by 10 min recovery $P_t$ was reduced to 77.2 ± 26.5% of baseline.

4. Discussion

We investigated the effects of repeated unaccustomed lengthening contractions on residual force enhancement. In contrast to our hypothesis, residual force enhancement increased following muscle damage. The driving force behind this finding is the greater decrease in MVC torque preceding stretch compared with the MVC torque following stretch. This suggests that RFE provided a prophylactic effect in preserving isometric strength following muscle damage.

During lengthening contractions, and similar to previous investigations (Lee and Herzog, 2002), peak eccentric torque

![Fig. 5](image-url) Maximal voluntary isometric strength was reduced following the first set of lengthening contractions and remained blunted throughout recovery. Effect of time (*), values are Means ± SE.

![Fig. 6](image-url) (A) Low frequency torque depression as a combined consequence of impaired (B) 10 Hz and (C) 50 Hz torque following muscle damage. There was a significant increase in low-frequency torque depression as shown by the decreased 10:50 Hz which was present throughout the task and recovery. The decreased ratio was driven by the 10 Hz component. Effect of time (*), values are Means ± SE.
was reached at the end of active lengthening. Prior to stretch, dorsiflexor MVCs at a shortened muscle length (neutral ankle angle; 0° PF), were lower than the reference MVC at 30° PF. During stretch, torque increased throughout the range of motion and was greater than the isometric reference MVC. In the current study, a 6–19% force enhancement was present (Fig. 4), which was similar to reports on the adductor Pollicis 14% (Lee and Herzog, 2002), knee extensors 4–5% (Shim and Garner, 2012), plantar flexors ~7–13% (Pinniger and Cresswell, 2007), and dorsiflexors ~9–16% (Pinniger and Cresswell, 2007; Tilp et al., 2009). However, force enhancement is not always evident following stretch. The incidence of RFE is equivocal in large muscle groups; for example RFE in the quadriceps muscle group was not present in one study (Hahn et al., 2007) while others reported force enhancement (Hahn et al., 2010; Shim and Garner, 2012).

The steady state phase of RFE can last up to 30 s immediately following stretch in some muscles (Abbott and Aubert, 1952). We did not observe this ‘long lasting’ force enhancement but rather a shorter term enhancement similar to that reported by Tilp et al. (2009), who also tested the dorsiflexors. There was a marked increase in eccentric torque throughout the range of motion, peaking at the end of stretch, then decreasing but remaining elevated over the reference isometric MVC for the duration of the effort (Fig. 4). We used a conservative approach to calculating RFE by comparing a 0.5 s epoch of torque following stretch to 0.5 s effort (Fig. 4). This indicates the possible role for neuromuscular fatigue which can be defined as any activity-induced transient reduction in the generation of torque or power, and can be manifested through both central and peripheral factors (Enoka and Duchateau, 2008). As indicated by the maintenance of agonist RMS EMG amplitude and voluntary activation (assessed using the interpolated twitch technique) which were similar to baseline values, with no change in antagonist coactivation, factors proximal to the neuromuscular junction (Power et al., 2010) do not seem to be pertinent in explaining (i.e. central fatigue) torque loss during the damaging protocol for this muscle group. Fatigue recovers relatively quickly following task cessation while damage has longer lasting impairments on muscle function (Choi and Widrick, 2009). By 10 min following task termination, recovery of fatigue is typically reached and muscle damage and impaired E–C coupling are primarily responsible for long lasting impairments in function (Power et al., 2010). This would explain the slight, non-significant, recovery of reference MVC up to 10 min.

Peripheral factors leading to a long-lasting reduction in MVC are decreased sarcoplasmic reticulum Ca2+ release and decreased myofibrillar Ca2+ sensitivity (Allen et al., 2008) inhibiting cross-bridge force production. Muscle damage and the subsequent impairment in force generated by the contractile machinery are frequently reported following repetitive lengthening contractions (Clarkson and Hubal, 2002) and are likely responsible for the prolonged torque loss in the present study. Mechanical disruptions from repetitive lengthening contractions also lead to E–C uncoupling, which was likely the peripheral mechanism for the reduced torque following repetitive lengthening contractions (Allen, 2001; Ingalls et al., 1998; Warren et al., 2001). We observed a greater reduction in 10 Hz than 50 Hz evoked torque during recovery from repetitive lengthening, which is an indication of impaired E–C coupling (Brunton et al., 2008).

Force enhancement has been observed in reduced preparations and in situ human studies (Campbell and Campbell, 2011; Herzog et al., 2012) and is well regarded as a fundamental property of muscle unaccounted for by the current cross bridge and sliding filament theory of muscle contraction. The current study cannot elucidate the exact mechanisms of increased force enhancement following muscle damage, but these are likely related to stretch-induced reduction of series compliance. This would retain an optimal angle of torque production following the shift in the force-length relationship found after damage. As well, following damage, the structural component of sarcomeres may be maintained, contributing to the increased force developed after stretch. Therefore, active force of cross-bridges is decreased because of impaired E–C coupling but force generated during stretch remains intact because force contribution from stretched sarcomeric structures is less impaired. Following stretch, isometric torque was impaired less by damage than the reference MVC suggesting that stretch provided a prophylactic effect to maintain torque during the isometric steady state phase. Whole body functional tasks show similar results, for example following muscle damage a squat jump without prior stretch is impaired more than a counter movement jump which relies on a quick stretch component before jumping (Byrne and Eston, 2002; Marginson et al., 2005). Therefore, stretch appears to attenuate detrimental effects of muscle damage on subsequent torque production.

![Fig. 7. Twitch torque was initially potentiated throughout the lengthening contraction task. Following task termination, twitch torque was reduced from baseline and remained blunted throughout recovery. Effect of time (s), values are Means ± SE.](https://example.com/image-url)
Conflict of interest statement

The authors have no known conflict of interest.

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