INCREASED RESIDUAL FORCE ENHANCEMENT FOLLOWING MUSCLE DAMAGE

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INTRODUCTION
By lengthening an already activated skeletal muscle, the force produced during stretch is greater than isometric force at the same muscle length. Any increase in force during the steady state phase following stretch is termed residual force enhancement (RFE). This intrinsic muscle property is believed to be a function of the muscles force transmitting structures, and is evident in various muscle preparations and in vivo human studies [1,2]. Repeated unaccustomed lengthening contractions result in muscle damage and impaired functional performance [3] owing to reduced force generation capacity and increased series compliance. However it is unknown how muscle damage affects RFE. This study investigated changes in neuromuscular properties of the dorsiflexors following high intensity lengthening contractions in young men. We hypothesised muscle damage associated with lengthening contractions would severly impair force generation and force transmitting structures leading to a reduction in RFE.

METHODS
We tested the dorsiflexors of 8 young men (26.5±2.8y) on a HUMAC NORM dynamometer (Stoughton, MA). Following determination of electrically evoked muscle properties (twitch, 10 Hz, 50 Hz), baseline reference maximal voluntary isometric contractions (MVC) were performed with 3 min of rest between all efforts. Following the MVCs a stretch was performed at 30%/s over a 30° range of motion ending at the same muscle length as the reference MVCs. The reference MVC always preceded the stretch and RFE was calculated as shown in figure 1. RMS EMG of the tibialis anterior and soleus muscles was recorded during all voluntary efforts. The muscle damage protocol involved 4 sets of 25 isokinetic (30%/s) lengthening contractions. The same measures collected at baseline were collected immediately post lengthening contractions, 5 min and 10 min into recovery.

RESULTS
Baseline neuromuscular measures are presented in table 1. Throughout the protocol 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. Because the MVC remained 29.3±11.9% lower (p<0.05) than baseline at 10 min of recovery, it was presumed significant muscle damage had occurred. Voluntary activation and RMS EMG of the tibialis anterior remained near maximal without an increase in coactivation of the antagonist (soleus:tibialis anterior). Contrary to our predicted results, RFE was significantly increased (p<0.05) following muscle damage (~100-300%). Despite damage associated eccentric force loss during stretch, the isometric reference MVC was further impaired thus driving the relationship between stretch and MVC for an increased RFE in the damaged muscle.

![Figure 1. RFE calculation example (a). Group data for RFE at baseline and following muscle damage (b).](image)

DISCUSSION AND CONCLUSIONS
Residual force enhancement was increased following muscle damage. It appears stretch provided a mechanical strategy for enhanced muscle function over isometric actions following muscle damage. Relatively, isometric force was reduced more than force following stretch. A potential explanation may relate to the intact force generators being unable to produce isometric force similar to baseline. Thus the active stretch may have involved structural properties of those ‘damaged force generators’ which contributed to enhanced RFE following muscle damage.

REFERENCES

Table 1: Baseline neuromuscular properties.

<table>
<thead>
<tr>
<th>Twitch Torque</th>
<th>10 Hz Torque</th>
<th>50 Hz Torque</th>
<th>Voluntary Activation</th>
<th>MVC Torque</th>
<th>Residual Force Enhancement</th>
<th>Eccentric Torque</th>
<th>Percent Co-Activation</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.6±1.9Nm</td>
<td>15.3±3.1Nm</td>
<td>22.3±4.0Nm</td>
<td>96.5±3.7%</td>
<td>39.4±9.8Nm</td>
<td>12.3±7.6%</td>
<td>55.0±12.4Nm</td>
<td>20.0±6.6%</td>
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