Length-tension relations are altered in regenerating fast- but not slow-twitch muscles of the rat

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Treatment of rat muscles with the myotoxin bupivacaine, results in complete degeneration of fibres and subsequent full regeneration within 60 days. These newly regenerated muscles have been shown to be more resistant to repeated muscle lengthening actions (eccentric contractions) than uninjured muscles, although the mechanisms underlying this protection are unclear. An alteration in the length-tension relationship and a resultant shift in the optimal length (L_o) for maximum isometric force production might protect regenerated muscles against damage from repeated lengthening actions. To test this hypothesis rats were deeply anaesthetised with an intraperitoneal (i.p.) injection of sodium pentobarbital (60 mg/kg) and the extensor digitorum longus (EDL) and soleus muscle of the right hind limb surgically exposed, and injected intramuscularly with the myotoxin bupivacaine hydrochloride. The rats were then allowed to recover for 7, 14, 21 or 60 days post surgery. The rats were then anaesthetised with sodium pentobarbital (60 mg/kg, i.p.) and isometric contractile properties of isolated muscles assessed in vitro at 25^{o}C. Muscles of the contralateral hind limb were not treated and served as control. Rats were killed by surgically excising the heart whist still anaesthetised deeply. EDL muscles recovered from myotoxic injury more rapidly than soleus muscles, with mass of EDL muscles restored to control levels by 21 days post-injury. In contrast, soleus muscles were restored to only 81% of control at 60 days post-injury. L_o of EDL muscles was ~2 mm (8%) longer than control ($P < 0.05$) at 60 days post injury and had a length-tension relationship shifted towards longer muscle lengths. No change in L_o or length-tension characteristics were observed in soleus muscles at any time point post injury. The results support the hypothesis that newly regenerated EDL, but not soleus muscles have longer L_o and length-tension relations located at a longer muscle length, a likely mechanism contributing to the regenerated EDL muscles protection from contraction-mediated injury.

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