Popping sarcomere hypothesis explains stretch induced muscle damage

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It has been known for over 100 years that active stretch of muscle, also known as eccentric or pliometric contraction, can lead to sore and stiff muscles, beginning the day after exercise and lasting up to a week. Mechanically eccentric contractions use muscles as brakes rather than motors, and occur in activities such as horse-riding, skiing and walking down hill. Histologically, such muscles show small areas of disrupted filament structure, confined to single fibres, and ranging in length from a single half sarcomere. Tension is also reduced more, and for longer, than after similar shortening contractions. Such exercise induces a rapid training effect, so that a second identical bout of exercise typically causes much reduced symptoms.

In 1990, it was suggested that the damage results from extremely non-uniform lengthening of sarcomeres, due to the instability of sarcomere lengths that results from the descending limb of the length-tension curve and the asymptote of the force-velocity curve (Morgan, 1990). Stretch of muscle beyond optimum length is concentrated in the sarcomere that has the lowest yield tension. This greater lengthening, on the descending limb of the length-tension curve, causes the isometric tension, and hence the yield point, to decrease. The assymptotic shape of the force velocity curve means that the sarcomere will be unable to support the existing tension at any velocity, and so will "pop", i.e. stretch rapidly and uncontrollably, limited only by passive viscosity and mass, until a length is reached where rising passive tension in that sarcomere increases to match the total tension being generated by the other un-lengthened sarcomeres. This will repeat with the next weakest sarcomere. The stretch then proceeds by popping sarcomeres in myofibrils, essentially one at a time in order from the weakest towards stronger. This explains why tension always rises during stretch, even beyond optimum length.

This hypothesis further postulated that the training effect consisted of growing extra sarcomeres in series to avoid stretch beyond optimum length. This was consistent with earlier observations that the number of sarcomeres in a fibre could change).

Since then, a number of results have supported this hypothesis. It has been shown in toad and rat muscle, that such stretch induced muscle damage is greater when the stretches are applied at longer length. It has been shown in rats and humans that training is accompanied by a shift in optimum length towards longer muscle lengths. In rats it has been confirmed that this is accompanied by an increase in the mean number of sarcomeres in the fibres of the muscle, and that the adaptation is ineffective if the stretches are moved to the same part of the length-tension curve rather than the same length.

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