SARCOMERE LENGTH AND FORCE BEHAVIOR IN SKELETAL MUSCLE MYOFIBRILS

Taiki Ino, Timothy Leonard, and Walter Herzog
University of Calgary, Faculty of Kinesiology
Email: taiki.ino1@ucalgary.ca

Summary

This study was aimed at investigating the effects of local sarcomere deactivation on sarcomere length and force behavior in skeletal muscle myofibrils. Using rabbit psoas muscle myofibrils, a novel technique involving targeted fluid delivery was employed to deactivate one (or more) sarcomeres in an otherwise fully activated myofibril. The deactivated sarcomeres lengthened slightly (from ~2.1µm to ~2.7µm) but did not "pop," preventing overstretch leading to muscle injury. Interestingly, at ~2.7µm, where the passive force from titin is negligible, deactivated sarcomeres still bore significant stress (~200 nN/µm²). We propose that activation establishes a stable connection between actin and titin, resulting in a stable truss that prevents sarcomere popping and muscle damage even after deactivation. These findings highlight a potential safety mechanism in skeletal muscle and contribute to our understanding of mechanisms of contraction.

Introduction

Muscles can produce active and passive forces. Active and passive forces depend on the length of sarcomeres [1]. Active force is generated through cross-bridge cycling, while passive force in single sarcomeres and myofibrils depends exclusively on the protein titin [2]. Within a myofibril, sarcomeres are arranged in-series, thus they transmit identical forces at any instant in time. When active forces between sarcomeres differ, passive forces need to compensate by sarcomere elongation until passive forces reach the required magnitude. These changes in sarcomere length have been thought to lead to so-called sarcomere "popping" and muscle injury [3]. We developed a novel technique to locally deactivate one (or more) sarcomeres in an otherwise fully activated myofibril with the purpose to test the idea that deactivated sarcomeres will "pop" when active force is eliminated.

Methods

We used single myofibrils from rabbit psoas muscle. A single myofibril is suspended between a needle (for length control) and a cantilever pair (for force measurement). A large and a small-bore pipette are used for activation and deactivation of the myofibril and single sarcomeres, respectively (Fig 1)

Fig 1: Experimental setup showing a single myofibril attached between a force lever and a glass needle for length control.

Results and Discussion

We found that locally deactivated sarcomeres did not overstretch in an otherwise fully activated myofibril as predicted by the popping sarcomere theory [3]. Deactivated sarcomeres lengthened by about 0.6µm from about 2.1 to about 2.7µm (Fig 2). At 2.7µm, sarcomeres from rabbit psoas have negligible passive force, thereby questioning how these deactivated sarcomeres could sustain stresses of more than 200 nN/µm^2. We propose that activated sarcomeres form a linked framework between actin and titin that provides increased passive force, thus preventing "popping" and sarcomere damage under these experimental conditions.

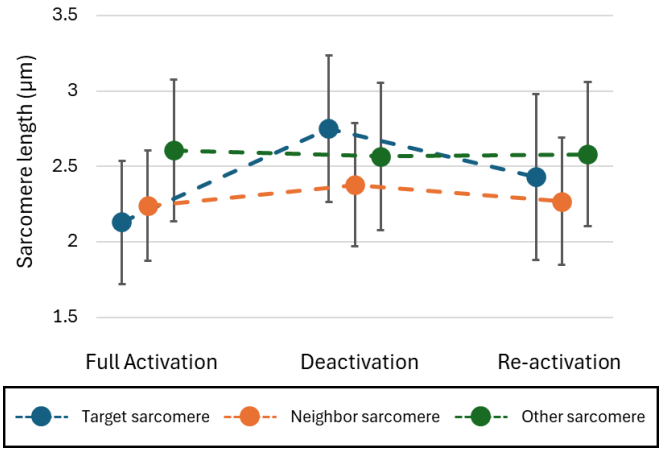

Figure 2: Sarcomere length changes as a function of experimental conditions. The target sarcomere is the one being deactivated. The neighbor sarcomeres are the sarcomeres on either side of the target sarcomere. The other sarcomeres are sarcomeres not immediately neighboring the target sarcomere.

Conclusions

We show the novel result that there is an in-built safety mechanism in previously activated sarcomeres that prevents them from being overstretched and damaged when deactivated. We propose that activation causes a titin-actin linkage that provides stability to sarcomeres that is not abolished immediately by deactivation.

Acknowledgments

CIHR and KDDS-University of Calgary scholarship to T.I.

References

[1] Gordon AM et al., *J. Physiol*. 184:170-92, 1966.
[2] Trombitas K, et al., *J Cell Biol*. 140:853-9, 1998.
[3] Morgan DL, *Biophys J*. 57:209-221, 1990.