THE RELATIONSHIP BETWEEN SARCOMERE LENGTH AND FORCE IN RABBIT PSOAS MYOFIBRILS

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INTRODUCTION

HE Huxley and Hanson (1954) and AF Huxley and Niedergerke (1954) showed that contraction in isolated myofibrils and single fibers, respectively, was not associated with an appreciable shortening of the A-band region of sarcomeres. They concluded independently that contraction in skeletal muscle occurs through the sliding of two sets of filaments, actin and myosin. In 1957, AF Huxley formulated a theory for contraction and force production in skeletal muscle, the cross-bridge theory, which explained how the relative sliding of actin and myosin was powered. The cross-bridge theory contains several basic assumptions including the idea that the cross-bridges are arranged uniformly on the myosin filament, and that they can attach to uniformly-spaced active sites on the actin filament. Also, on average, all cross-bridges are assumed to produce about the same amount of force and act independently of each other. These assumptions led to the prediction that the force in a sarcomere should be linearly related to the amount of actin/myosin overlap on the descending limb of the force-length relationship. In 1966, Gordon et al. tested this prediction and found it to be correct for single fibers of frog whose sarcomere lengths were controlled in the mid-portion of the fiber. Although individual sarcomere lengths could not be measured or controlled with this approach, the linearity of myofilament overlap and force has been accepted as fact in the muscle mechanics community. The purpose of this study was to test the relationship between sarcomere length and force in isolated myofibrils in which sarcomere lengths can be measured for each and all sarcomeres of the preparation.

METHODS

Myofibrils from rabbit psoas were isolated using standard chemical and mechanical isolation procedures. Myofibrils were attached to a nanolever force transducer (Fauver et al., 1998) at one end, and a glass needle at the other end. The glass needle was attached to a computer controlled micro manipulator to produce prescribed length changes of the myofibril. Individual sarcomere lengths were measured by projecting the striation patterns of the sarcomeres onto a linear photodiode array, and digitizing this area at 20 Hz (Blyakhman et al., 2001). Measurements were obtained from 8 myofibrils containing 7-30 sarcomeres in series. Typical recordings were made for isometric contractions of the myofibril on the descending limb of the force-length relationship, followed by stretches of approximately 10% of myofibril length.

RESULTS AND DISCUSSION

For all myofibrils, we observed that sarcomere lengths prior to stretching on the descending limb of the force-length relationship were non-uniform, but steady. During stretch, all sarcomeres of the myofibrils were stretched by varying amounts (Figure 1). In the isometric phase following the stretch, sarcomeres remained at non-uniform, but perfectly constant lengths. Differences in the sarcomere lengths following myofibril stretch typically
exceeded 0.5 um. In the example shown in Figure 1, the shortest and longest sarcomere lengths following stretching are about 2.4 and 3.1 um, respectively, which, according to the myofilament overlap theory, corresponds to a difference in active force capability of 48% of the maximal isometric force at optimal length. The sarcomeres in a myofibril are arranged strictly in-series, therefore, the force at steady-state must be the same in each sarcomere. So, how can we possibly explain a difference of almost 50% of force between the shortest and longest sarcomere?

One explanation might be that the force deficit in the long sarcomere might be taken up by passive structures. However, we know that for sarcomere length of 3.1 um and less, passive force in rabbit psoas myofibrils barely reach 5% of the maximal isometric force (Bartoo et al., 1993), so passive forces are not sufficient to explain this discrepancy. Another explanation might be that sarcomeres in a myofibril have differing amounts of contractile proteins and that the difference in myofilament overlap might be accounted for by the number of contractile proteins. However, if this assumption was correct, the amount of overlap (or sarcomere length) before and after stretch should be exactly proportional, but it is not (Figure 1). At this point, we do not know why sarcomeres of vastly different length can produce identical forces, when the theory predicts that forces should differ by as much as 50%.

We propose that sarcomeres at different lengths can produce the same amount of force following active stretching because of the well-known force enhancement effect (Herzog, 1998). This theory needs rigorous testing in the future.

**SUMMARY**

We found strong evidence that sarcomere length does not uniquely determine sarcomere force. Future research will need to address systematically the origins of this highly unexpected result.

**REFERENCES**

HE Huxley and J Hanson, Nature 173:973-976, 1954
AF Huxley Prog Biophys Biophys Chem 7:255-318, 1957

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**Fig. 1:** Sarcomere lengths of a single myofibril as a function of time