

FORCE DEPRESSION IN SINGLE MYOFIBRILS AND SARCOMERES

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INTRODUCTION

It is well accepted that the steady-state isometric force following shortening of an activated muscle is smaller than the corresponding steady-state force obtained for a purely isometric contraction at the corresponding length (Abbott and Aubert, 1952; Marechal and Plaghki, 1979; Herzog & Leonard, 1997). This phenomenon is referred to as force depression.

Despite an abundance of experimental observations, the origin of force depression is still a matter of debate. One of the hypotheses proposed to explain force depression is sarcomere length non-uniformity (Edman et al., 1993; Morgan et al., 2000). According to this hypothesis, during shortening on the descending limb of the force-length relationship, sarcomeres are assumed to shorten by different amounts because of instability. Some sarcomeres shorten a slight amount, whereas others shorten more than average; these sarcomeres may shorten to a degree that places them on the ascending limb of the force-length relationship. This behaviour leads to a situation in which the tension produced is smaller than that produced at the corresponding length during an isometric contraction in which sarcomere lengths are assumed to be relatively uniform.

The aim of this study was to investigate this hypothesis by testing force depression in single myofibrils and tracking at the same time the changes in individual sarcomere lengths. If the sarcomere length non-uniformity hypothesis was true, and force depression was caused by sarcomere length

non-uniformity exclusively, the following predictions should hold: (i) myofibrils should show force depression, (ii) there should be an increase in sarcomere length non-uniformity after shortening, and (iii) force depression should not be observed in individual sarcomeres.

METHODS AND PROCEDURES

Myofibrils isolated from rabbit psoas muscle were fixed to a glass needle and a motor at one end and to a nanolever at the other end, allowing for length changes and force measurements, respectively. The striation pattern of myofibrils was projected onto a linear photodiode array for determination of individual sarcomere lengths.

Myofibrils ($n=11$) were activated at an average sarcomere length (SL) of $2.8\mu\text{m}$ and then shortened at a speed of $0.1\mu\text{m/s/sarcomere}$ to an average SL of $2.4\mu\text{m}$. Myofibrils were then held isometrically for 1min, and then deactivated. After a rest period of 5 mins, myofibrils were reactivated at the final SL of $2.4\mu\text{m}$ in order to obtain an isometric reference force. Forces were normalized by myofibril cross-sectional area and expressed as stress ($\text{nN}/\mu\text{m}^2$).

Force depression for a myofibril was defined as the difference in the steady-state isometric force following shortening, and the purely isometric reference contraction at $2.4\mu\text{m}$ sarcomere length.

Since myofibrils are formed of sarcomeres arranged in series, measuring the force at the end of a myofibril gives the instantaneous

force in each sarcomere; and therefore, by measuring individual sarcomere lengths, we can determine force depression for individual sarcomeres.

RESULTS AND DISCUSSION

All eleven myofibrils showed force depression averaging $31.0 \pm 3.9\%$ of the isometric reference force (figure 1).

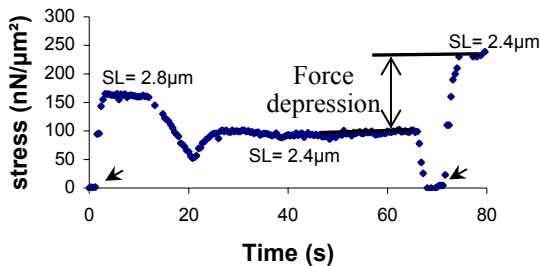


Figure 1. Myofibril response when activated at an average SL of $2.8\mu\text{m}$, then shortened to an average SL of $2.4\mu\text{m}$, deactivated, then activated. Arrows indicate the activation time.

This result indicates that the origin of force depression must be within the sarcomeric structure. In order to test if force depression might be caused by the development of sarcomere length non-uniformities, we tracked sarcomere lengths prior to, during and after myofibril shortening. According to the sarcomere length non-uniformity theory, we would expect an increase in the sarcomere length distribution after shortening and an abolishment of force depression in individual sarcomeres. However, this was not the case. First, sarcomere lengths after shortening were non-uniform (the standard deviation (SD) of the sarcomere lengths was $0.10\mu\text{m}$) but this non-uniformity was not greater than the non-uniformity observed before shortening ($\text{SD} = 0.12\mu\text{m}$; figure 2), or the non-uniformity obtained at $2.4\mu\text{m}$ after the purely isometric reference force ($\text{SD} = 0.11\mu\text{m}$). Second, all individual sarcomeres ($n=60$) showed force depression ranging from 6.1% to 54.3%.

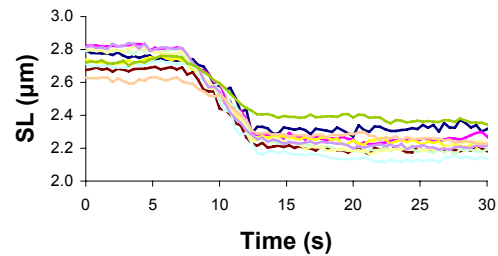


Figure 2. Sarcomere lengths prior to, during and after shortening from a typical myofibril.

This result suggests that force depression cannot be explained by the sarcomere length non-uniformities. Changes in the kinetics of cross bridges leading to a decrease in the force produced per cross bridge or the number of attached cross-bridges should be considered. It has been proposed (Marechal and Plaghki, 1979) that shortening might induce an inhibition of cross-bridge attachment in the newly formed overlap zone and therefore resulting in a decrease in the number of attached cross-bridges and force.

CONCLUSION

Force depression does not depend on sarcomere length non-uniformity, but rather seems an inherent property of the molecular mechanism underlying contraction.

REFERENCES

- Abbott, BC and Aubert, XM (1952). *J. Physiol* 117:77-86.
- Edman K et al. (1993). *J Physiol* 466:535-552.
- Herzog, W and Leonard, TR (1997). *J Biomech* 30:865-872.
- Marechal, G and Plaghki, L (1979). *J Gen Physiol* 73:453-467.
- Morgan DL et al. (2000). *J Physiol* 522:503-513.

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