Unveiling Stress-Dependent Contributions of Passive Structures to Muscle Short-Range Stiffness in Cat Soleus Muscle

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Summary

Muscle short range stiffness (SRS) provides stability during movement and is assumed to result from stretching actin-myosin cross-bridges, with a minimal contribution from passive structures. However, previous studies compare passive and active contributions to SRS at matched muscle lengths that do not generate substantial passive stress. This does not account for the stress dependent stiffness of passive structures. Thus, our goal was to compare SRS at matched levels of active and passive stress in cat soleus muscle. We found that SRS was only up to 30% greater in active compared with passive muscle at matched levels of stress, highlighting a potential role of passive structures on SRS.

Introduction

Muscle short-range stiffness (SRS) is important for movement stability and postural control. SRS describes muscles' initial response to rapid perturbations in length and is assumed to result from stretching actin-myosin cross-bridges [1]. Passive structures (i.e. extracellular matrix, titin) are assumed to have a minimal contribution to SRS based on comparisons at matched muscle lengths with varying levels of activation [2]. Previous studies only measure SRS at muscle lengths with minimal passive stress. However, passive stiffness increases with stress [3] and internal stresses due to muscle activation may cause passive structures to contribute to SRS. Therefore, our goal was to compare SRS at matched levels of active and passive stress in cat soleus muscle. We hypothesized that SRS would increase with both active and passive stress, which would suggest that the contribution of passive structures to SRS cannot be ignored.

Methods

The hindlimbs of anesthetized cats (4 females) were placed in a 37C saline bath with knee and ankle joints fixed. The soleus and its distal tendon were dissected from surrounding tissues, remaining connected to a bone chip of the calcaneus. Force-length properties were characterized by systematically lengthening the muscle and measuring force by stimulating a distal branch of the sciatic nerve (40Hz) (Fig 1A). SRS was measured at four active and four passive conditions at lengths corresponding to 25-100% optimal force (F_{max}) (Fig 1B). A rapid change in length (2mm at 2m/s) was applied to measure SRS of the muscle tendon unit and the stiffness of the tendon-aponeurosis was subtracted [4]. A linear mixed effects model was used to describe SRS as a function of stress (continuous) and activation (fixed), with a random effect of cat.

Results and Discussion

SRS increased with active and passive stress (Fig 1C-D). SRS increased more rapidly with active compared to passive stress (slope=5.23mm), resulting in 30% greater SRS at ~F_{max}. Although our passive and active trials placed the muscle at different lengths, we achieved similar stresses and found a stress dependent component of SRS that is independent of activation. While we cannot conclude that passive structures contribute to SRS during active conditions, our findings highlight the possibility that passive structures may be the major contributor to SRS even during active conditions.

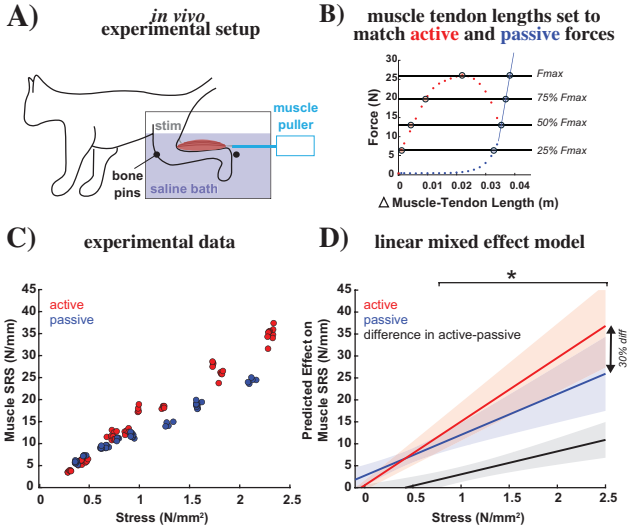

Figure 1: A) Experiment setup. B) Initial force-length characterization. C) SRS vs active and passive stress for one cat. D) Model predictions (solid line) and 95% confidence intervals (shaded region) for grouped data. *Significant effect ($p<0.05$).

Conclusions

Our findings highlight a potential contribution of passive structures to muscle SRS. Increased internal stresses during muscle activation may cause passive structures in series with muscle fibers (i.e. collagen fibers, internal tendons) to contribute to SRS in a way that is not captured when measuring passive and active SRS at matched muscle lengths. Future work is needed to isolate the mechanisms of SRS in passive and active muscle, especially when using SRS in impaired populations with alterations in passive mechanics.

Acknowledgments

Thank you to funding (R01 AR071162, T32 HD07418) and our experimental team (Drs. Mahrous, Chardon, Wang).

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