ACTIVE AND PASSIVE FORCE ENHANCEMENT IN RABBIT PSOAS MYOFIBRILS

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INTRODUCTION

It has been accepted that the steady state force produced by a muscle following active stretch is greater than the corresponding purely isometric force (Abbott and Aubert, 1952; Herzog and Leonard, 2002). This phenomenon, called residual force enhancement, has been described in a variety of muscle preparations, ranging from human muscles (Lee and Herzog, 2002), isolated muscles (Abbott and Aubert, 1952; Herzog and Leonard, 2002) and single fibers (Rassier et al., 2003). The mechanisms of residual force enhancement are not known. It has been proposed that it has a passive component, related to titin filaments, and an active component, related to cross-bridge formation and actin-myosin interaction (Herzog and Leonard 2002).

The aim of this study was to investigate 1) residual force enhancement at the myofibril level and 2) the amount of contribution of the passive and active components to the residual force enhancement in single myofibrils. Residual force enhancement is accompanied by an increase in passive force after deactivation. This so-called passive force enhancement reflects the contribution of the passive component to the total residual force enhancement (Herzog and Leonard, 2005). The contribution of the active component can be approximated by subtracting the passive force enhancement from the total residual force enhancement. This calculation is possible in myofibrils where all sarcomeres are arranged in series, and therefore mechanical properties can be directly related to the intra-sarcomeric passive (titin) and active (cross-bridges) components.

METHODS

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

Myofibrils (n=6) were fixed at a sarcomere length (SL) of 2.4µm then stretched passively, in a relaxing solution, to a SL of 3.4µm; the stretch was held for 1min (Fig.1). After a period of ten-minute rest, the myofibril was chemically activated, by replacing the relaxing solution by an activating solution with a high calcium concentration, then stretched to a SL of 3.4µm. The stretch was held for 1min; deactivation occurred 30s after the stretch (Fig. 1).

Residual force enhancement was defined as the difference between the steady state force obtained after stretch (Fs, Fig. 1) and the force obtained before stretch (Fi, Fig. 1) corrected for the final sarcomere length according to the force length relationship (Fc, Table 1).

Passive force enhancement was measured as the difference between the steady state force reached after passive stretch (Fp, Fig. 1) and

the force after active stretch and deactivation (Fa, Fig. 1). Forces were normalized to the myofibril cross-sectional area.

RESULTS AND DISCUSSION

All six myofibrils showed residual force enhancement and passive force enhancement, as shown for a typical myofibril in figure 1. This result is in accordance with previous results obtained in whole muscles and single fibres (Herzog et al., 2002; Rassier et al., 2003).

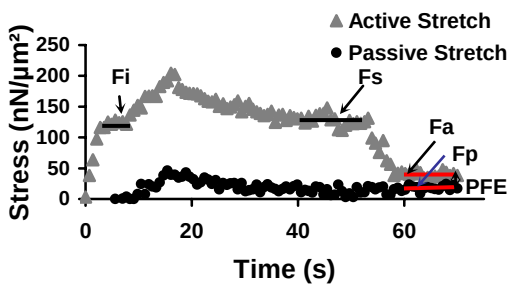

Figure 1: Typical myofibril response when stretched passively (●) and actively (▲). Passive force enhancement (PFE) is Fa-Fp. Residual force enhancement is Fs-Fc, where Fc is Fi minus the decrease in force when going from a SL of 2.4µm to a SL of 3.4µm, according to the force length relationship.

The average residual force enhancement and passive force enhancement were respectively 140 ± 24 nN/µm^2 and 39 ± 8 nN/µm^2 (Table 1). Accordingly, the contribution of the passive component to the residual force enhancement cannot exceed 28 ± 5% of the residual force enhancement. The contribution of the active component to the residual force enhancement would then be more than 70%.

Knowing that titin is the main structure responsible for passive force in single myofibrils, we propose that titin is responsible for about 28% of the residual force enhancement for the conditions tested here. Titin's stiffness might be calcium-dependent and therefore increasing upon muscle activation (Labeit et al., 2003).

The active component of the residual force enhancement is directly related to the cross-bridges. It might be caused by an increase in the number of attached cross-bridges induced by a decrease in the rate of detachment (Herzog and Leonard, 2002).

	Fi	Fs	Fc	FE	PFE
Mean	177	199	57	140	39
SD	24	30	8	24	8

Table 1: Forces (Mean ± SD) produced by 6 myofibrils normalized to the cross-sectional area (nN/µm^2).

CONCLUSION

The active component, represented by the cross-bridges, is the major contributor to residual force enhancement in myofibrils stretched from 2.4 to 3.4µm average sarcomere length. It accounts for more than 70% of the force enhancement while the passive contribution is about 28%.

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