Tendon Transfer Surgery Increases Passive Tension of Muscle, Muscle Fiber Bundles, and Muscle Fibers

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Introduction: Muscles can adapt to altered length change by increasing or decreasing serial sarcomere number [1]. The functional result of this adaptation is for the muscle to work on or around optimal sarcomere length. If this finding applies to muscles after tendon transfer surgery, this would be desirable, as intraoperative measurements reveal that transferred muscles may be overstretched to the point where they produce less than 30% of their maximum active tension [2]. This dramatic structural reorganization in response to stretch may alter muscle passive tension, which can also influence function. However, passive tension after tendon transfer has not been thoroughly studied. Since muscle tension can be regulated at the fiber, fiber bundle or even whole muscle level, it is important to know which (if any) of these scales is affected. Therefore, the purpose of this study was to measure the passive tension of the entire muscle, fiber bundle and single fiber after stretched tendon transfer surgery that resulted in increased sarcomere number.

Methods: These experiments were performed in accordance with the UCSD Institutional Animal Care and Use Committee. The hindlimbs of male New Zealand White rabbits (body mass = 2.54 ± 0.31 kg SD) were used. Briefly, the distal tendon of the EDI1 muscle was transferred to the ankle extensor retinaculum at a sarcomere length of 3.7 µm measured intraoperatively [3]. Under these conditions, the muscle increased sarcomere number by ~30% within the first week after surgery. Animals were re-anesthetized 1 week (n = 9) or 4 weeks (n = 8) after the surgery. After each hindlimb was secured with Steinmann pins, the distal tendon was transected and clamped to a servomotor (Model 310B, Aurora Scientific, Ontario, Canada) at the distal muscle-tendon junction. Muscle passive tension was measured at various muscle lengths (Lm). After testing, a small fiber bundle was taken from the middle part of the musculature and immediately immersed in relaxing solution to measure passive mechanical properties [4]. Fiber bundles and single fiber segments were dissected from the bundle and transferred to a chamber filled with physiological solution. The ends of each were attached between an ultrasonic force transducer (Model 405, Aurora Scientific, Ontario, Canada) and a micro-manipulator. Diameter was measured and used to calculate fiber or bundle cross-sectional area. Sarcomere length was measured by laser diffraction. Samples were stretched in 250-µm increments with a two-minute stress relaxation period between each stretch, after which sarcomere length and tensile force were recorded. The elastic modulus (Assyst/Axstrm) was determined within the sarcomere length physiologic range (2.5 to 4.0 µm). Raw data were expressed as tangent modulus to account for the elastic modulus of single fiber (open bars) and fiber bundle (filled bars) from transferred muscles compared to control muscles (Fig. 1). In addition, passive tension of the transferred muscle was larger at week 1 compared to week 4 over most of the range studied. In agreement with muscle properties, the elastic modulus of fiber bundles from transferred muscles were nearly double those of control muscles at both week 1 and week 4 (Fig. 2, filled bars, p < 0.001 for both time points). There was a trend of shorter resting fiber bundle sarcomere length from transferred muscles compared to control muscles (data not shown). The elastic modulus of single fibers from transferred muscle was significantly higher at week 1 compared to control muscles (p < 0.001) but not at week 4 (Fig. 2, open bars).

Discussion: This study demonstrated that muscles become stiffer after tendon transfer, primarily through alteration of ECM, during which time the muscle adapted to stretch by adding sarcomeres in series (data not shown). Considering the rapid synthetic activity required to add sarcomeres after stretch, it is no wonder that muscle also rapidly alters its ECM. Interestingly, muscle passive tension increased within the first one week then decreased slightly by week 4. The passive tension increase may represent a protective response against acute stretch outside of the physiologic range and it may then be attenuated as muscles generate enough serial sarcomeres to cope with the stretch. In surgical limb distraction models, it has been shown that stretch induces an increase in intramuscular connective tissue, which results in increased muscle passive tension [5]. While there was no information regarding passive tension at different time points in that study, it is possible that the same underlying biological processes are in place. The nature of these processes is not known. It is most likely that the intimate interaction between muscle cell and ECM through the combined effect of integrin and dystrophin-associated protein complex are involved. These transmembrane force transmitting proteins have already been implicated in the pathogenesis of the muscular dystrophies [6, 7]. Further studies are required to understand both the biological and the biomechanical interaction between muscle cells and the ECM.

Results: For whole muscles, Lm-passive tension relationships were shifted to shorter muscle lengths with steeper inclinations in the transferred muscles compared to the control muscles (Fig. 1). In addition, passive tension of the transferred muscle was larger at week 1 compared to week 4 over most of the range studied.

Figure 2: Elastic modulus of single fiber (open bars) and fiber bundle (filled bars) from the control and transferred muscles.

Figure 1: Average Lm-passive tension relationships for the transferred (diamond) and control (circle) muscles at week 1 (solid lines) and week 4 (dashed lines). Lm is expressed relative to optimal length (Lo) where active tension was maximal.

References:

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