Increased Serial Sarcomere Number does not Result in Increased Muscle Excursion after Tendon Transfer Surgery
Mitsuhiko Takahashi, Samuel R. Ward, Richard L. Lieber
University of California and Veterans Affairs Medical Center, San Diego, La Jolla, CA
rlieber@ucsd.edu

Introduction: Muscles adapt experimentally by adding sarcomere in series in response to stretch outside of their physiologic range in an animal tendon transfer model [1]. If this finding also applies to muscles after tendon transfer surgery, this would be desirable, as intraoperative measurements reveal that transferred muscles are typically overstretched to the point where they are predicted to produce less than 30% of their maximum active tension [2]. Increased sarcomere number in series is associated with increased muscle excursion and/or contraction velocity in normal muscles. However, there have been no functional studies of muscles after tendon transfer in an attempt to correlate change in serial sarcomere number with muscle excursion. Therefore, the purpose of this study was to measure muscle functional excursion after a tendon transfer surgery that results in increased sarcomere number.

Materials and Methods: These experiments were performed in accordance with the UCSD Institutional Animal Care and Use Committee. The hindlimbs of four male New Zealand White rabbits (body mass = 2.54±0.12 kg) were used. Briefly, the distal tendon of the EDII muscle was transferred to the ankle extensor retinaculum at a sarcomere length of 3.7 μm measured intraoperatively using a laser diffraction device [1]. Tendon attachment was secured with 5-0 Ethibond® suture. Under these conditions, the muscle adapts by adding ~1000 serial sarcomeres 1 week after surgery, corresponding to a ~30% increase. The contralateral muscle served as a control. Animals were reanesthetized 1 week after the surgery and each hindlimb was secured with Steinmann pins. Initial muscle length (Lm) was measured at 90° of both knee and ankle joints. The distal tendon was transected and clamped to a servomotor (Model 310B, Aurora Scientific, Ontario, Canada) at the distal muscle-tendon junction. Muscle isometric tension was measured at various lengths by stimulation of the branch from the popliteal nerve with a 650-ms train of 0.3-ms pulses delivered at 100 Hz. Active and passive Lm-tension curves were fit with quintic polynomials to enable calculation of excursion. After testing, muscles were dissected and fixed to perform muscle architectural analysis to permit quantification of serial sarcomere number. Paired t-test was used for comparison between the transferred and control muscles. Data are presented as mean±SEM.

Results: The general shape of the Lm-tension curves obtained from both the transferred and control muscles were similar. Maximum isometric active tension (P0) for the transferred muscle (11.06±1.95 N) was significantly (p < 0.05) reduced compared to that for the control (16.40±1.18 N) (Fig. 1). Lm-tension relationships expressed relative to percent P0 were used to compare excursions (Fig. 2). The Lm ranges where muscles were predicted to generate 50% of P0 were -2.41±0.34 to 2.67±0.33 mm (width: 5.09±0.67 mm) for the transferred muscle and -2.83±0.27 to 3.49±0.09 mm (width: 6.32±0.33 mm) for the control muscle. Surprisingly, in spite of the fact that muscle excursion was decreased in the transferred muscle, muscle architectural analysis demonstrated that the transferred muscle gained ~14% sarcomeres in series (4195±39 for the transferred muscle versus 3687±39 for the control; p < 0.05). In addition, the passive Lm-tension curve was shifted to shorter muscle lengths with a steeper inclination in the transferred muscle (Fig. 2).

Discussion: The most significant finding of this study was that, in spite of the increased serial sarcomere number produced by a stretched tendon transfer, muscle excursion actually decreased. This is surprising in light of the weight of literature suggesting that serial sarcomere number correlates linearly with muscle excursion. We currently have no definitive structural explanation for this result. Though stretch indeed induced addition of serial sarcomere, newly-synthesized sarcomeres might not be functional at this time-point, or surgical intervention may cause functional deficit at sarcomere level. Furthermore, isometric active tension including P0 was reduced throughout the Lm-tension curve accompanied by a sharp rise in passive tension.

Further experiments are required to understand the time-course and structural basis for these phenomena. However, the data are clinically significant in that they challenge the existing dogma regarding muscle adaptation to chronic stretch. Specifically, it should not be assumed that muscles will simply “re-optimize” to a new length after surgery.

In addition, it appears that muscles are equally adaptive with regard to passive tension as active tension. While the majority of the passive load bearing is probably extracellular in nature, the nature of collagen adaptation in a model such as this is not known.


Acknowledgements: This work was supported by NIH grants HD048501 and HD050837 and the Department of Veterans Affairs

Figure 1. Active Lm-tension curves on an “absolute” scale for the transferred muscle (solid lines) and for the control (dashed lines). P0 was predicted by the vertex of each curve.

Figure 2. Lm-tension curves averaged from four animals on a “relative” scale for the transferred muscle (A) and for the control (B). Passive Lm-tension curves are also drawn.