Ultra-short Echo Time Magnetic Resonance Imaging of Rabbit Flexor Tendons in An Anterior Cruciate Ligament Reconstruction Model

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Target Audience: Researchers, orthopaedic surgeons and radiologists interested in the effects of load on tendon graft T2* values

Purpose: On commonly used magnetic resonance imaging (MRI) fast spin echo (FSE) images, tendon-grafts exhibit little signal due to their highly ordered collagen composition, yielding rapid shortening of T2 relaxation and loss of signal. When the graft is highly disrupted by edema or hemorrhage, it may appear abnormal on FSE but subtle injuries or plastic deformation may escape detection. As tendon-grafts mature, the process of ligamentization may also affect MRI appearance. Ultra-short echo time (UTE) MRI has the ability to assess tissue species with very short T2 relaxation times such as tendon-grafts (~4-6 ms), and permits calculation of a reproducible decay constant (T2*). UTE uses TEs that are 100–1,000 times shorter than those of conventional sequences and can detect signal from the tendon and other tissues before it has significantly decayed [1]. Prolongation of T2* may be used as an indicator of disruption of highly ordered tissues [1,2] such as tendon grafts. ACLR models using less than 5% tendon strain (which is needed to produce structural change in the tendon [3]) would not be expected to alter T2* due to lack of collagen distortion. The current study tested changes in UTE MRI T2* values in rabbit tendon grafts subjected to very low loads.

Methods: In vivo: Six 1.9 kg New Zealand White (NZW) rabbits underwent ACLR using a flexor tendon autograft positioned with an anterior femoral tunnel and an anatomic tibial tunnel. Half were immobilized with an external fixation device and half were allowed free motion after an initial 3 day post-operative immobilization. Five of the 6 rabbits were sacrificed at 6 weeks and MRI was performed immediately. Ex vivo in situ: Five contralateral knees from the in vivo study were harvested for ACLR and preloaded with 2N of tension applied to the graft at a limb position of 135 degrees. Following initial MRI, the tendon grafts were preloaded at the same angle to 17N and MRI was repeated. MRI: Scanning was performed on a 3T clinical system (GE Healthcare, Waukesha, FL). Multi-planar fast-spin-echo (FSE) images were acquired: echo time (TE): 24 ms, repetition time (TR): 4000 ms, receiver bandwidth (RBW): ±50 kHz, acquisition matrix (AM): 512x256-384, number of excitations (NEX): 1-2, field-of-view (FOV): 8cm, slice thickness (SL): 1-2.0 mm. Axial multi-slice multi-echo 2D UTE images oriented along the tendon graft were acquired for tendon T2* calculations: TE=0.05, 5.10, 15 ms, TR=250 ms, RBW=±62.5 kHz, AM=512x701, NEX=2, flip angle = 45°, ST=2mm, slice spacing = 1-2 mm. Image Analysis: Multiple tendon T2* values were calculated from the UTE images by fitting the TE to the corresponding signal intensity: SI(TE)=So e^{-TE/T2*}, where SI(TE) is the signal intensity at TE, So is proportional to proton density, T2* is the time constant, and C is a constant to account for noise. T2* values in each tension group were compared. Statistics: paired non-parametric t-test was performed to detect differences of tendon T2* values between the immobilized and mobilized in vivo tendons, and the unloaded and preloaded cadaveric tendons. Significance was set at p<0.05.

Results: In vivo: No significant difference of T2* was found between immobilized and mobilized tendon in vivo grafts, 3.27 ± 0.2 ms and 3.57 ± 0.8 ms, respectively, p=0.70 (Figure 1). Ex vivo in situ: No significant difference of T2* was found between unloaded and preloaded cadaveric tendon grafts, 3.08 ± 0.3 ms and 2.92 ± 0.5 ms, respectively, p=0.38 (Figures 1 and 2).

Discussion: The lack of detectable difference in tendon T2* values of the in vivo group suggests that 17N is not sufficient to disrupt the tendon structure, which agrees with prior studies [3]. The ex-vivo, in-situ pretension load of 17N is ~1% the ultimate load of a NZW flexor tendon [4] and is similar to that found during natural resting flexion of the graft as positioned described above. This level of preload induces a strain of only approximately 0.7%, which is below the threshold to induce collagen disruption [3] and therefore not likely to cause a detectable change of T2*. Our results demonstrate that immobilizing rabbit limbs did not produce detectable changes in the graft T2* values after 6 weeks compared with controls. Additionally, scanning with applied tension of approximately 17 N creates no substantial disruption to the tendon graft as shown by UTE MRI. We recognize that the actual graft strains during scanning are not known, and may be below than 17N due to creep and stress relaxation in vivo.

Conclusion: We have created an ACLR model with a preload and specific tunnel anatomy that does not alter UTE MRI T2* values under normal free cage motion conditions, presumed to be lack of sufficient collagen disruption. Histology will be necessary to demonstrate if minimal collagen disruption is present that is below the threshold needed to induce a change on UTE MRI. These results suggest that strict immobilization of the limb may not be required if the graft strain is less than 1%.


Figure 1: Graph demonstrating the T2* values of in vivo and ex vivo rabbit tendon grafts

Figure 2: Axial plane UTE MR images of a cadaveric tendon graft with regions of interest depicting T2* values for one tendon graft preloaded at A) 2N and subsequently at B) 17N.