



Effect of Scapular Dyskinesis on Supraspinatus Tendon Healing in a Rat Model

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Introduction

Rotator cuff tears are common conditions that often require surgical repair to improve function and relieve pain. Unfortunately, despite pain relief, the success associated with repair integrity is mixed, with 5-95% of patients having recurrent tears¹⁻³, resulting in decreased strength and function. Several factors may contribute to repair failure including age, tear size, and time from injury. However, the mechanical mechanisms resulting in repair failure are not well-established making clinical management difficult. Specifically, altered scapular motion (termed scapular dyskinesia) may be one important and modifiable factor which can place the healing tendon at risk for re-injury⁴. Therefore, the objective of this study was to determine the effect of scapular dyskinesia on supraspinatus tendon healing following repair. We hypothesized that scapular dyskinesia will result in H1) diminished joint function and passive joint mechanics and H2) decreased supraspinatus tendon healing following repair due to the compromised mechanical environment present.

Methods

Experimental Design: A rat model of scapular dyskinesia was used. This condition was created by denervating the trapezius and serratus anterior muscles through surgical transection of the spinal accessory and long thoracic nerve, respectively. 70 adult male Sprague-Dawley rats (400-450 grams) were randomized into two groups: nerve transection (SD) or sham nerve transection (Control). Following this procedure, all rats underwent unilateral detachment and repair of the supraspinatus tendon. All rats were sacrificed at 2, 4, or 8 weeks after transection and frozen (for mechanical testing at 4 and 8 weeks, N = 10) or fixed in formalin (for histology at each time point, N = 5).

Ambulatory Assessment: Forelimb gait and ground reaction forces were quantified over time in all animals using an instrumented walkway. Data was collected one day prior to nerve transection to obtain baseline ambulatory values and then collected at days 3, 7, 14, 28, 42, and 56 post-injury. Parameters were normalized to body weight.

Passive Joint Mechanics: Passive shoulder joint range of motion and stiffness were measured over time using a custom instrument and methodology⁵. Measurements were taken one day prior to nerve transection, and at days 14, 28, and 56 days post-surgery. Briefly, under anesthesia, the forearm was secured into a rotating clamp at 90° of elbow flexion and 90° of glenohumeral forward flexion. The scapula was manually stabilized to isolate glenohumeral motion. The arm was then rotated through internal and external rotation three times. A bilinear fit utilizing least-squares optimization was applied to calculate joint stiffness in the toe and linear regions for both internal and external rotation. Parameters were normalized to baseline values.

Tendon Mechanical Testing: At the time of testing, the animals were thawed and the humerus was dissected with the supraspinatus intact. Stain lines, for local optical strain measurement, were placed on the tendon. Cross sectional area was measured using a custom laser device. To determine biomechanical properties, tensile testing was performed as follows: preload, preconditioning, stress relaxation, and ramp to failure. Stress was calculated as force divided by initial area, and 2D Lagrangian optical strain was determined.

Histology: Tendons were harvested immediately after sacrifice and processed using standard paraffin procedures. Sagittal sections (7 μ m) were collected, and stained with Hematoxylin-Eosin (H&E). Cell density and cell shape were graded by three blinded investigators, using a scale of 1-3 (1 = low, 2 = moderate, 3 = high) for cellularity and 1-3 (1 = spindle shaped, 2 = mixed, 3 = rounded) for cell shape. Polarized light images were analyzed using custom software to evaluate tendon organization, as previously described⁷.

Statistics: For the ambulatory assessment, multiple imputations were conducted using the Markov chain Monte Carlo method for missing data points. For both ambulatory assessment and passive joint mechanics, significance was assessed using a 2-way ANOVA with repeated measures on time with follow-up t-tests between groups at each time point. Tissue mechanics between groups were assessed using a t-test

at each time point. Histology scores were evaluated using a Mann-Whitney test. Significance was set at $p < 0.05$, trends at $p < 0.1$.

Results

Joint function was significantly altered in the SD group compared to control (Figure 1). Specifically, braking force was significantly decreased at later time points (6 and 8 weeks post-injury) and propulsion force was significantly increased at all time points (except 3 days post-injury). No differences were observed in passive joint mechanics (not shown).

Elastic and viscoelastic parameters were altered in the presence of scapular dyskinesis (Figure 2). Specifically, mid-substance modulus was significantly diminished at 4 weeks in the SD group compared to control, while no differences were observed at 8 weeks. However, a trend toward increased percent relaxation, a measure of inferior viscoelastic properties, was observed in the SD group compared to control at 8 weeks. Interestingly, tendon cross-sectional area was significantly decreased at the insertion at 4 weeks post-injury, with a similar trend at the mid-substance (not shown). Tendon organization and cell shape were also altered (not shown). Specifically, a trend toward greater disorganization was observed at the mid-substance in the SD group compared to control at 2 weeks post-injury. Additionally, a trend toward a more rounded cell shape was observed at the insertion in

the SD group compared to control at 4 weeks post-injury. No differences were observed in cell density (not shown).

Discussion

While the prevalence of rotator cuff repair failures is well-documented, the mechanical mechanisms by which failure occurs are not well-established, making clinical management difficult. Previous studies have demonstrated a strong association between scapular dyskinesis and shoulder injury. Using this animal model, we prescribed scapular dyskinesis and rigorously evaluated the effect on supraspinatus tendon healing following cuff repair in a controlled manner. Results demonstrated that scapular dyskinesis alters joint function and leads to compromised supraspinatus tendon properties. Loading environment is particularly important in healing tissues and in this study, scapular dyskinesis likely placed abnormal and excessive loads on the healing tendon, thus compromising its mechanical integrity. Specifically, tendon mechanical, organizational, and histological properties were diminished in the presence of SD, indicative of diminished tissue healing. Identification of scapular dyskinesis as a potential mechanical mechanism of failed rotator cuff healing will help guide clinicians in prescribing treatment strategies for patients with cuff tears. Specifically, successful pre-operative scapular rehabilitation may be necessary to achieve successful outcomes post-operatively.

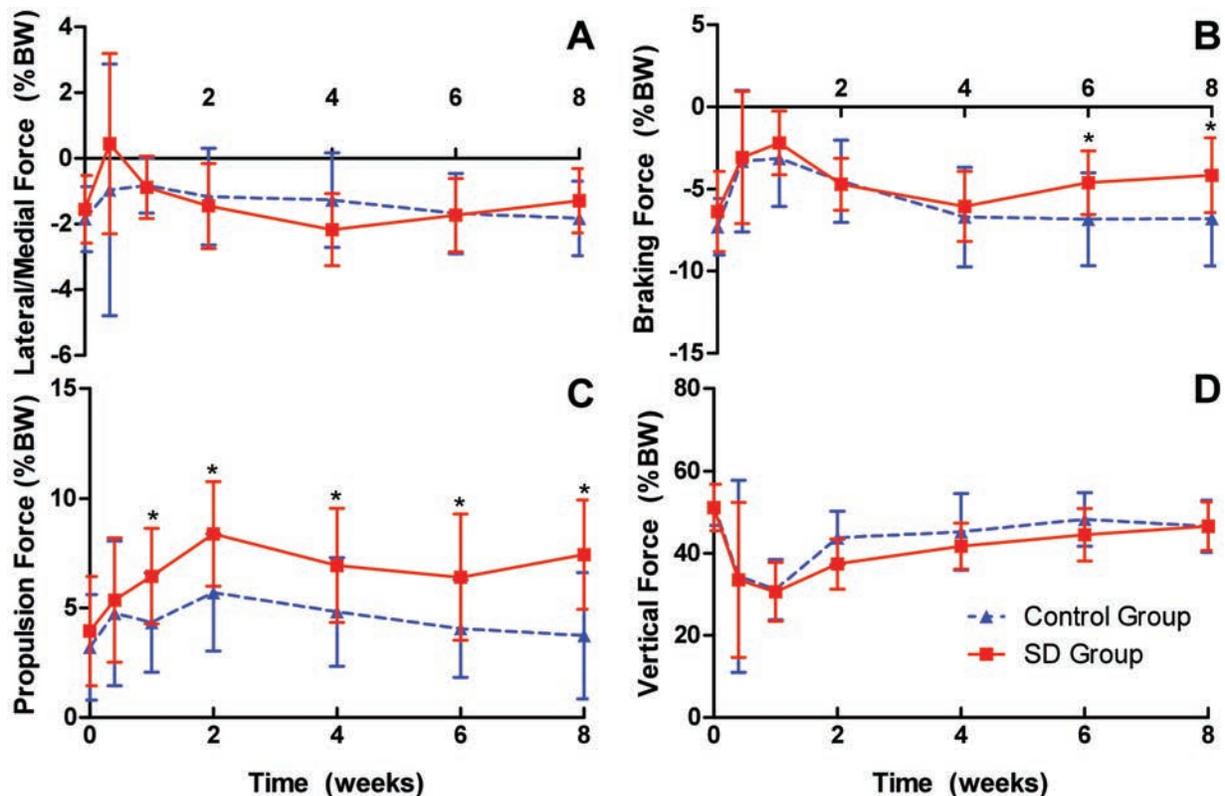


Figure 1. (A) No differences were observed in medial-lateral forces between groups. (B) The SD group had a significantly decreased braking force compared to control at 6 and 8 weeks. (C) The SD group had a significantly increased propulsion force compared to control at all time-points (except 3 days post-injury). (D) No differences were observed in vertical force between groups. Data is shown as mean and SD (* $p < 0.05$).

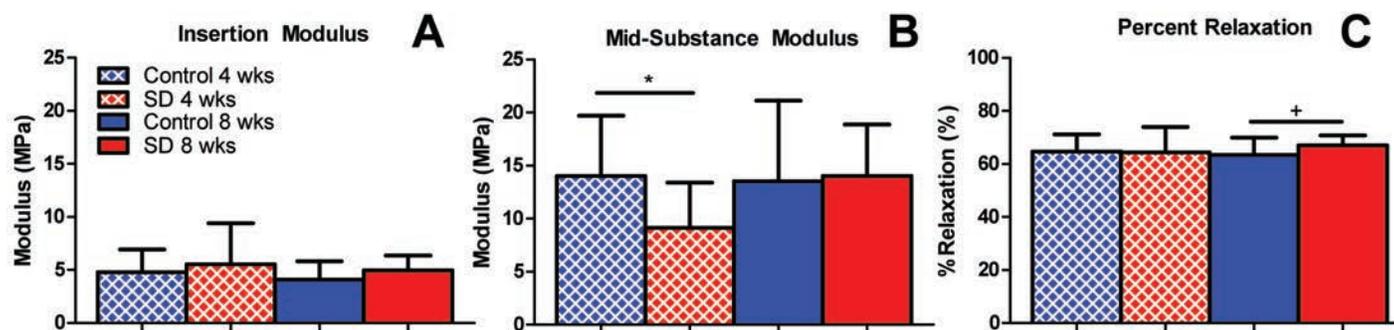


Figure 2. (A) No differences were observed at the insertion site at any time point. (B) A significantly decreased mid-substance modulus was observed at 4 weeks, in the SD group compared to control. (C) A trend toward increased percent relaxation was observed at 8 weeks, in the SD group compared to control. Data is shown as mean and SD (* $p < 0.05$, + $p < 0.1$).

Clinical Significance

This study identifies scapular dyskinesis as a mechanical mechanism for compromised supraspinatus healing following repair. Identifying modifiable factors that lead to compromised tendon healing will help improve outcomes following repair.

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