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Effect of Scapular Dyskinesia on Supraspinatus Repair Healing in a Rat Model

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Abstract

Background—Rotator cuff tears are common conditions that often require surgical repair to improve function and relieve pain. Unfortunately, repair failure remains a common problem following rotator cuff repair surgery. 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 understood making clinical management difficult. Specifically, altered scapular motion (termed scapular dyskinesia) may be one important and modifiable factor contributing to the risk of repair failure. Therefore, the objective of this study was to determine the effect of scapular dyskinesia on supraspinatus tendon healing following repair.

Methods—A rat model of scapular dyskinesia was used. 70 adult male Sprague-Dawley rats (400–450 grams) were randomized into two groups: nerve transection of the accessory and long-thoracic nerves (SD) or sham nerve transection (Sham Control). Following this procedure, all rats underwent unilateral detachment and repair of the supraspinatus tendon. All rats were sacrificed at 2, 4, and 8 weeks after surgery. Shoulder function, passive joint mechanics, and tendon properties (mechanical, histological, organizational, and compositional) were evaluated.

Results—Scapular dyskinesia alters joint function and may lead to compromised supraspinatus tendon properties. Specifically, diminished mechanical properties, altered histology, and decreased tendon organization was observed for some parameters.

Conclusion—This study identifies scapular dyskinesia as one underlying mechanism leading to compromise of supraspinatus healing following repair. Identifying modifiable factors that lead to compromised tendon healing will help improve clinical outcomes following repair.

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Conflict of Interest: None

Level of evidence—Basic Science, *in-vivo* Animal Study.

Keywords

scapular dyskinesis; tendon; rotator cuff repair; animal model; rat; shoulder

Introduction

Rotator cuff tears are common conditions that often require surgical repair to improve function and relieve pain. Unfortunately, despite successes in relieving pain, the success associated with repair integrity is mixed, with 5-95% of patients having recurrent tears,^{4, 11, 13, 14, 17} resulting in decreased shoulder strength. Several factors may contribute to repair failure including age, tear size, and time from injury; however, mechanical mechanisms contributing to repair failure are not well-established, making clinical management difficult. Identifying modifiable factors that lead to compromised tendon healing will help improve clinical outcomes following repair.

Tendon healing following repair does not regenerate the normal tendon-bone interface and instead leads to the formation of a fibrovascular scar.^{12, 25} This scar is mechanically inferior to native tendon and is therefore prone to re-rupture. Several augmentation (conservative and surgical) strategies have been developed in an attempt to improve healing rates and re-establish tendon insertion architecture. Conservative rehabilitation protocols are typically implemented pre-operatively in an attempt to correct deficits and post-operatively in an attempt to improve healing and outcomes.²² Biologics, such as growth factors, cytokines, and stem cells, and tissue grafting approaches have been introduced at the repair site in an attempt to promote healing and strengthen repairs.^{2, 3, 34, 37, 44}

Mechanical loading plays an important role in tendon-to-bone healing following repair.⁴³ However, the optimal loading conditions in the treatment of healing repair tissues are not well-defined. Specifically, previous studies have shown conflicting results, regarding immobilization, passive motion, and increased loading.^{5, 7, 16, 18, 19, 31} In general, low loading regimens and immobilization seems to promote healing while excessive or abnormal joint loading may have detrimental effects. The goal of pre- and post-operative rehabilitation protocols is to optimize the mechanical loading environment of the tendon and improve healing potential.

At the shoulder, abnormal scapulothoracic joint kinematics (termed scapular dyskinesis) contributes to tendon injury.^{24, 27} Specifically, the abnormal loading environment in the presence of scapular dyskinesis alters the tendon composition and diminishes its mechanical properties.³⁶ It is likely that scapular dyskinesis is also detrimental to the supraspinatus tendon following surgical repair, compromising healing. Specifically, in the presence of scapular dyskinesis, the mechanical loading environment of the tendon is likely abnormal in both amount (i.e., overload) and type (i.e., compressive, shear) of loading. These mechanical changes may undermine optimal tendon-to-bone healing. In particular, abnormal joint mechanics may be one mechanical mechanism by which failed healing following cuff repair occurs.

Studies in the knee have demonstrated that deficits in strength and function have negative consequences on the long term outcomes following ACL reconstruction.^{9, 29} However, in the shoulder, the consequences on rotator cuff tendon healing and functional outcomes following rotator cuff repair in the presence of scapular dyskinesis have not been evaluated. Physical therapy and rehabilitation^{6, 22, 23} aimed to re-educate scapular muscles and correct positional abnormalities is the current treatment for scapular dyskinesis. Successful pre-operative scapular rehabilitation may be necessary to achieve successful outcomes post-operatively.

Therefore, the objective of this study was to determine the effect of scapular dyskinesis on supraspinatus tendon to bone healing following tendon repair utilizing an established animal model.^{35, 36} We hypothesized that scapular dyskinesis will result in H1) diminished joint function and passive joint mechanics and H2) decreased supraspinatus tendon-to-bone healing following tendon repair due to the compromised mechanical environment present during healing.

Materials and Methods

Study Design

A rat model was used in this Institutional Animal Care and Use Committee approved study. Seventy male Sprague Dawley rats (400-450 g) were randomized into 2 groups: nerve transection to create scapular dyskinesis (SD + Supra Repair group, n=35) or sham nerve transection (Sham + Supra Repair Group, n=35). For the nerve transection surgery, the spinal accessory and long thoracic nerves were visualized and transected as described by Reuther et al.³⁶ For the sham nerve transection surgery, the nerves were visualized but were not transected. Following this procedure, all rats underwent unilateral detachment and repair of the supraspinatus tendon as described by Thomopoulos et al..⁴² Pre- and post-operative analgesics (buprenorphine, 0.05 mg/kg) were administered up to 2 days following surgery. Animals then returned to unrestricted cage activity and were sacrificed 2, 4, and 8 weeks following surgery and either frozen (for mechanical testing at 4 and 8 weeks, n=10) or fixed in formalin (for histology and immunohistochemistry at each time point, n=5)

Detachment and Repair Surgery

Briefly, the supraspinatus tendon was visualized and a grasping stitch placed, using a 5-0 polypropylene suture (Surgipro II, Covidien, Mansfield, MA, USA). The tendon was then detached at its insertion using a #11 scalpel blade and allowed to freely retract. For repair, a 5mm diameter burr (Multipro 395, Dremel, Mt. Prospect, IL, USA) was then used to remove any remaining fibrocartilage at the insertion site. A 0.5 mm anterior to posterior hole was drilled through the greater tuberosity of the humerus distal to the insertion site and suture was passed through the bone tunnel, and the tendon reapposed to the insertion site. Closure was achieved by suturing the deltoid muscle and the skin was closed using staples.

Quantitative Ambulatory Assessment

Forelimb ground reaction forces (medial/lateral, braking, propulsion, and vertical) were quantified prior to sacrifice using an instrumented walkway.³⁸ Data was collected one day

prior to surgery to obtain baseline, uninjured values and then collected at days 5, 7, 14, 28, 42, and 56 following surgery. All data was normalized by body-weight.

Passive Joint Mechanics

Passive range of motion measurements were performed prior to sacrifice.^{36, 39} Data was collected one day prior to surgery to obtain baseline, uninjured values and then at 2, 4, and 8 weeks following surgery. All data was normalized to baseline values.

Sample Preparation for Mechanical Testing

Following ambulatory and passive joint mechanics measurements, animals were sacrificed and frozen for subsequent mechanical testing. At the time of testing, the animals were thawed and the humerus was dissected out with the supraspinatus tendon intact. Upon dissection, successful denervation was confirmed in all animals in the SD + Supra repair group through observation of serratus anterior and trapezius muscle thinning and atrophy. The tendon was fine dissected under a microscope to remove muscle and any excess, non-loadbearing tissue near the repaired insertion site. Suture from the surgical repair was left embedded within the tendon to avoid damage due to dissection. Cross-sectional area was measured using a custom laser device.¹⁰

Tendon Mechanical Testing

Elastic and viscoelastic mechanical properties of the insertion and mid-substance of the supraspinatus tendon were determined using uniaxial tensile testing.¹⁵ Briefly, Verhoeff stain lines were placed along the length of the supraspinatus tendon to divide the insertion and mid-substance regions for local optical strain measurements. The humerus was fully embedded in a holding fixture using polymethylmethacrylate (PMMA) and inserted into a custom testing fixture and secured to a base fixture. The proximal tendon was annealed in sand paper and gripped using custom serrated grips. The specimen was immersed in PBS at 37°C during testing. Tensile testing consisted of: preconditioning for 10 cycles from 0.1 N to 0.5 N, stress relaxation to 5% strain at a rate of 5 %/sec for 600 sec, and ramp to failure at 0.3%/sec. Elastic properties were calculated using a linear regression from the linear region of the stress-strain curves. For viscoelastic parameters, percent relaxation was determined using the peak and equilibrium loads.

Tendon Histology

Histologic analysis was performed to examine cellular and organizational changes in the supraspinatus tendon. Tissues were harvested immediately after sacrifice and processed using standard paraffin procedures. Sagittal sections (7 um) were collected and stained with Hematoxylin–Eosin (H&E). Stained supraspinatus tendon sections were imaged at the insertion site and mid-substance using a microscope at 200X and 100X magnification using traditional and polarized light, respectively. Cell density and cell shape were independently graded by three blinded investigators, who were provided with previously prepared standard images, 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. Previous studies indicate that cell shape and cell density are important indicators in assessing tendon damage and healing.^{8, 40} Polarized

light images were analyzed using custom software to evaluate the angular deviation of the collagen orientation, a measure of fiber distribution spread.¹⁵

Tendon Immunohistochemistry

The distribution of extracellular matrix (ECM) proteins was localized using immunohistochemical techniques.³⁶ Tissue specimens were stained for a protein consistent with cartilage (collagen type II), a protein consistent with scar formation (collagen type III), a protein consistent with tendon (the proteoglycan decorin), and an inflammatory marker (IL1- β) (S-Table 1). The proteins were visualized using DAB, which makes the antibody-protein conjugate turn brown. Given the potential variations in staining intensities, all sections were stained at the same time with the same antibody. Negative controls were used and no background staining was observed. The insertion site and mid-substance of each tendon were evaluated separately. Staining results were independently graded by three blinded investigators, who were provided with previously prepared standard images, using a scale of 0-3 (0=undetectable, 1=low, 2=medium, 3=high), and the mode was used as the final score.

Statistics

Statistical analysis was performed according to our specific study hypotheses rather than by the global study design. For ambulatory assessment, measurements were occasionally absent for a specific animal on a specific day. Therefore, multiple imputations were conducted using the Markov chain Monte Carlo method for missing data points (~10%). 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 t-tests (n=10). Based on previous data on rat tendon mechanical properties by Gimbel et al, Peltz et al, and Perry et al.,^{15, 30, 33} a priori power analysis was performed to determine sample size to achieve a power of 0.8. Histology and immunohistochemistry scores were evaluated using Mann-Whitney tests (n=3-5). Significance was set at $p<0.05$; trends at $p<0.1$.

Results

Ambulatory Data

Shoulder joint function was significantly altered in the SD + Supra Repair group compared to the Sham + Supra Repair group (Figure 1). Gross observation demonstrated alteration in scapular movements, consistent with scapular “winging” as observed in the human. Specifically, propulsion force was significantly increased in the SD + Supra Repair group compared to the Sham + Supra Repair group at every time point, except 5 days post-injury (Figure 1A). Braking force was significantly decreased in the SD + Supra Repair group compared to the Sham + Supra Repair group at 6 and 8 weeks post-injury (Figure 1B). No differences in vertical or medial/lateral forces were observed between groups (Figure 1C, 1D). No significant differences in spatio-temporal parameters (step width, stride length, or speed) were observed between groups (data not shown), demonstrating lack of a difference in compensation between groups by the uninjured contralateral limb, as observed by Perry et al.³²

Passive Joint Mechanics

No differences in passive joint mechanics were observed between groups at any time point (S-Table 2).

Tendon Mechanics

Mechanical properties were altered in the SD + Supra Repair group compared to the Sham + Supra Repair group for some parameters. Specifically, insertion cross-sectional area was significantly decreased in the SD + Supra Repair group compared to the Sham + Supra Repair group at 4 weeks, with a similar trend at the tendon mid-substance (Figure 2A, 2B). No differences in insertion elastic modulus were observed between groups (Figure 3A). Mid-substance elastic modulus was significantly decreased in the SD + Supra Repair group compared to the Sham + Supra Repair group at 4 weeks (Figure 3B). No differences in maximal load were observed (data not shown). For viscoelastic properties, a trend toward increased percent relaxation in the SD + Supra Repair group compared to the Sham + Supra Repair group was observed at 8 weeks post-injury (Figure 4).

Tendon Histology

No differences in cellularity were observed between groups (Table 1). A trend toward a more rounded cell shape was observed at the supraspinatus insertion in the SD + Supra Repair group compared to the Sham + Supra Repair group at 4 weeks (S-Figure 1). No differences in collagen fiber organization were observed at any time point, except for a trend toward greater disorganization in the mid-substance at 2 weeks (S-Figure 2).

Tendon Immunohistochemistry

No differences were observed at any time point or in any region for the proteins studied (S-Table 3).

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 abnormal scapulothoracic joint kinematics (termed scapular dyskinesis) and shoulder injury.²⁷ Using an established animal model, we prescribed scapular dyskinesis and rigorously evaluated the effect on supraspinatus tendon healing following cuff repair.

Consistent with our first hypothesis, scapular dyskinesis significantly altered joint function. Specifically, propulsion force was significantly increased in the SD + Supra Repair group compared to the Sham + Supra Repair while braking force was significantly decreased at later time points. These changes indicate an alteration in the loading environment at the shoulder due to scapular dyskinesis and may place the healing supraspinatus tendon at increased risk of re-rupture. These findings are consistent with but less dramatic than previous findings by Reuther et al. that have also identified significant changes in joint function in propulsion force and in additional parameters, such as vertical ground reaction force, due to scapular dyskinesis alone.³⁶ A higher propulsion force may place increased

stresses on the healing supraspinatus, compromising the mechanical integrity of the repair. Decreased braking force, which was observed only at later time points, may be indicative of a functional adaptation to prevent further damage. During forward locomotion, the rat undergoes large amounts of forward flexion, requiring substantial rotation of both the humerus and scapula at the shoulder joint. In this scapular dyskinesis model, the scapular rotators, the trapezius and serratus anterior, are not functioning and the scapula is unable to rotate, which likely led to the observed alterations in braking and propulsion forces. However, with no change in the medial or lateral forces, the results indicate that the serratus anterior and trapezius did not participate in these actions, or were compensated for by surrounding structures. Interestingly, no changes in vertical forces were observed, contrary to previous findings from Reuther et al. using this model.³⁶ Vertical force corresponds to weight-bearing and pain and it is possible that the pain due to the repair surgery was greater than that due to the effect of scapular dyskinesis on function, masking any underlying alterations due to scapular dyskinesis. Loading environment is important in healing tissues and can differ in amount/magnitude (i.e., immobilization, exercise, over use) and type (i.e., tensile, compressive, shear). In this study, scapular dyskinesis likely alters the type of loading the tendon experiences much more than amount/magnitude, thus altering the mechanical integrity of the healing tendon.

Contrary to our first hypothesis, scapular dyskinesis had no effect on passive joint mechanics. Sarver et al previously demonstrated that increased stiffness and decreased range of motion is observed following cuff repair.³⁹ This may be attributed to scar formation in the healing tissue and joint capsule or muscular adaptations in response to the repair surgery. While tendon stiffening, capsular tightening, and muscular dysfunction are likely to contribute to changes in glenohumeral joint stiffness and range of motion, the excessive scar formation present following repair surgery may mask these underlying issues and therefore differences in passive joint mechanics between groups were not identified. It is likely that the surgical repair has a greater effect on passive joint mechanics than the effect of scapular dyskinesis.

Consistent with our second hypothesis, scapular dyskinesis was detrimental to supraspinatus tendon mechanical properties for some parameters. Specifically, mid-substance modulus was significantly diminished at 4 weeks in the SD + Supra Repair group compared to the Sham + Supra Repair group. However, this change did not persist at 8 weeks. Additionally, a trend toward a more rounded cell shape was observed in the supraspinatus insertion at 4 weeks, which may be indicative of compressive loading. In the mid-substance region, the tendon was also more disorganized in the SD + Supra Repair group compared to control at 2 weeks and is consistent with the mechanical changes observed at this time point. The location specific tendon changes (mid-substance region) are similar to findings observed previously by Reuther et al. in the supraspinatus tendon in the presence of scapular dyskinesis³⁶ and may be due to its anatomic location under the acromial arch during forward flexion, resulting in impingement and an altered loading environment. Surprisingly, decreased cross-sectional area was observed in the SD + Supra Repair group compared to the Sham + Supra Repair group at 4 weeks. This finding may be related to the reduced subacromial space present in the SD + Supra Repair group due to the diminished upward rotation of the scapula, allowing less space for tissue proliferation and matrix production.

This finding did not persist at 8 weeks and an improvement in tissue elasticity is apparent (no difference in elastic modulus). This may be related to the compensatory decrease in braking force observed at later time points (6 and 8 weeks), reducing stress on the repair site and preserving mechanical properties. However, a trend toward increased percent relaxation (a viscoelastic parameter) was observed in the SD + Supra Repair group compared to the Sham + Supra Repair group at 8 weeks post-injury. Previous studies have found increased percent relaxation in injured tendon, indicative of inferior tissue properties and poor healing.^{1, 8}

Contrary to our second hypothesis, no differences were observed in insertion modulus or maximal load. While increased insertion modulus may be an indicator of tendon healing, Reuther et al. has previously demonstrated that scapular dyskinesis has no effect on native insertion site properties.³⁶ Additionally, while maximal load is an important indicator of repair strength, the typical loads at which the supraspinatus operates is not near this threshold. Material properties, such as elastic modulus and percent relaxation, which highlight the quality of the tissue, may be a better indicator of repair integrity and functionality than maximal load at failure. Fatigue properties, which may be more consistent with modes of failure seen in the clinical condition, could be evaluated in future studies. In general, these findings indicate that there may be mechanical consequences associated with poor scapulohoracic joint kinematics for the healing supraspinatus tendon following repair.

Interestingly, no differences were observed in protein expression, as measured by immunohistochemical techniques. Tendon healing is characterized by a reactive scar formation and involves excessive inflammation, cell proliferation, and increased matrix synthesis characterized by increased deposition of type III collagen.²⁰ This heightened response to tendon injury and repair may mask any effect of scapular dyskinesis in this study. As observed with passive joint mechanics, surgical repair may have a greater effect on tendon composition than the effect of scapular dyskinesis.

This study has several limitations. First, the use of a quadruped animal does not exactly replicate the human shoulder. However, the presence of the acromial arch and its position over the rotator cuff is similar to the human shoulder and is essential in our model to evaluate the effect of scapular dyskinesis on supraspinatus healing as it passes underneath it. Secondly, acute transection of the supraspinatus tendon and immediate repair does not exactly replicate the human condition. Specifically, rotator cuff tears are typically chronic in nature. However, using an acute transection and repair allows us to examine the mechanical and biologic healing response in a more controlled manner to address our study hypotheses. Thirdly, acute transection of both the spinal accessory and long thoracic nerve to induce scapular dyskinesis does not exactly mimic the most common clinical scenario of scapular dyskinesis. Specifically, the etiology of scapular dyskinesis could be attributed to nerve injury in only approximately 6% of cases⁴¹ and several other factors may contribute to scapular dysfunction clinically. However, nerve injuries, due to a traumatic blow, a severe stretch or traction, or laceration, are common in sports²⁶ and can be found secondary to various surgical interventions.^{21, 28} This model of scapular dyskinesis, created by utilizing a nerve injury mechanism, is appropriate because it does mimic the clinical condition of scapular “winging” and does provide a consistent, repeatable method of inducing scapular

dyskinesia, allowing us to address our primary hypothesis. Specifically, this model was designed to investigate the effect of scapular dyskinesia on tendon healing and was not developed to address the etiology of scapular dyskinesia. Importantly, underlying mechanisms and cause and effect relationships can only be addressed in an animal model where time from injury can be controlled and function and properties can be evaluated over time. Future studies could model a more mild case of scapular dyskinesia through surgical transection of one nerve (as opposed to two) or through temporary paralysis of the scapular muscles through Botulinum toxin (Botox) injections. Lastly, while immunohistochemistry is a standard technique to localize proteins in tendon, additional quantitative measures, such as the enzyme-linked immunosorbent assays (ELISA), could be measured to further elucidate biochemical outcomes.

Conclusion

In summary, results suggest the functional consequences associated with scapular dyskinesia may compromise supraspinatus tendon healing following repair by diminishing some tendon mechanical properties. Identification of abnormal joint mechanics as a potential mechanical mechanism of failed rotator cuff healing will help guide clinicians in prescribing treatment strategies for patients with cuff tears. This study supports the need to evaluate joint kinematics and to consider correcting these abnormalities prior to surgical repair. This may allow for optimization of the mechanical loading environment and result in improved tendon healing rates.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

(A) The SD + Supra Repair group had increased propulsion force compared to the Sham + Supra Repair group at most time-points (except 5 days following repair). (B) The SD + Supra Repair group had decreased breaking force compared to the Sham + Supra Repair group at later time-points (6 and 8 weeks). (C) No difference in vertical force was observed. (D) No difference in medial/lateral force was observed. Data is shown as mean $\pm$ standard deviation.

Figure 2.

(A) The SD + Supra Repair group had decreased cross-sectional area at the insertion compared to the Sham + Supra Repair group at 4 weeks following repair. (B) A trend toward decreased cross-sectional area was observed in the SD + Supra Repair group compared to the Sham + Supra Repair group at the mid-substance at 4 weeks following repair. Data is shown as mean $\pm$ standard deviation.

Figure 3.

(A) No differences were observed in insertion elastic modulus. (B) The SD + Supra Repair group had decreased elastic modulus compared to the Sham + Supra Repair group at 4 weeks following repair. Data is shown as mean $\pm$ standard deviation.

Figure 4.

A trend toward increased percent relaxation was observed in the SD + Supra Repair group compared to the Sham + Supra Repair group at 8 weeks following repair. Data is shown as mean ± standard deviation.

Table I**Tendon Histology**

Results for passive joint mechanics demonstrated no differences between groups. Data is shown as normalized by baseline values (change from baseline) and as mean $\pm$ standard deviation.

Histology	Group	Region	2 weeks	p-value	4 weeks	p-value	8 weeks	p-value
Cell Shape	SD+Supra Repair	INS	1 (1-2)	0.11	2 (2-2)	0.08+	2 (2-2)	0.37
	Sham+Supra Repair		2 (2-2)		1 (1-1)		2 (1.75-2)	
	SD+Supra Repair	MID	2 (1.75-2)	0.5	2 (2-2)	0.50	3 (2-3)	0.27
	Sham+Supra Repair		2 (1-2)		2 (2-2)		2 (2-3)	
Cell Density	SD+Supra Repair	INS	2 (2-3)	0.42	3 (2-3)	0.50	2 (1-2)	0.45
	Sham+Supra Repair		2 (2-3)		3 (2-3)		2 (1-3)	
	SD+Supra Repair	MID	3 (3-3)	0.50	3 (3-3)	0.16	2 (2-2)	0.35
	Sham+Supra Repair		3 (3-3)		3 (2-3)		2 (2-3)	

Table II

A trend toward a more rounded cell shape was observed in the SD + Supra Repair group compared to the Sham + Supra Repair group at 4 weeks following repair. Data is shown as median and interquartile range (+p<0.1).

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