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Strain Distribution of Intact Rat Rotator Cuff Tendon-to-Bone Attachments and Attachments With Defects

This study aimed to experimentally track the tissue-scale strains of the tendon–bone attachment with and without a localized defect. We hypothesized that attachments with a localized defect would develop strain concentrations and would be weaker than intact attachments. Uniaxial tensile tests and digital image correlation were performed on rat infraspinatus tendon-to-bone attachments with defects (defect group) and without defects (intact group). Biomechanical properties were calculated, and tissue-scale strain distributions were quantified for superior and inferior fibrous and calcified regions. At the macroscale, the defect group exhibited reduced stiffness (31.3 ± 3.7 N/mm), reduced ultimate load (24.7 ± 3.8 N), and reduced area under the curve at ultimate stress (3.7 ± 1.5 J/m²) compared to intact attachments (42.4 ± 4.3 N/mm, 39.3 ± 3.7 N, and 5.6 ± 1.4 J/m², respectively). Transverse strain increased with increasing axial load in the fibrous region of the defect group but did not change for the intact group. Shear strain of the superior fibrous region was significantly higher in the defect group compared to intact group near yield load. This work experimentally identified that attachments may resist failure by distributing strain across the interface and that strain concentrations develop near attachment defects. By establishing the tissue-scale deformation patterns of the attachment, we gained insight into the micromechanical behavior of this interfacial tissue and bolstered our understanding of the deformation mechanisms associated with its ability to resist failure.

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Introduction

The tendon-to-bone attachment transfers load from muscle to bone. The loading requirements of the attachment involve tensile forces (e.g., from muscle contraction), compressive forces (e.g., from joint motion), and shear forces (e.g., from tendon fibril sliding) [1,2]. The attachment withstands these loads through its hierarchical, structural, and compositional features that may act to reduce stress and control strain at the interface [3–7]. At the macroscale, the tendon-to-bone attachment splays outwardly, increasing its cross-sectional area (CSA) from tendon to bone to reduce localized stress at the interface [8,9]. At the microscale, a gradient of unmineralized and mineralized fibrocartilage [6,10,11], as well as collagen fibril interdigitations and alignment, aid in reducing or redirecting localized stress [4,9,11–15]. At the nanoscale, pattern

variations in mineral accumulation around collagen fibers and within the collagen molecular structure may also modulate macroscale attachment biomechanics [4].

The hierarchical structures of the tendon–bone attachment are theorized to limit failure and stress concentrations at the interface [3,4,9–11]. The alignment of collagen fibrils in the rotator cuff attachment, measured using polarized light microscopy and modeled using finite element analysis, has been identified as a mediator of stress concentrations at the tendon-to-bone attachment [9]. In addition, mineral accumulation and collagen fibril orientation has been shown, using modeling, as some of the key factors of the attachments' mechanical properties [4]. Multiscale regression modeling has also been used to probe how the composition, structure, and dynamic processes of tendon and the tendon-to-bone attachment establish their mechanical properties [16]. These studies utilized computation and regression modeling to explain how the intact structures prevent stress concentrations at the attachment and have motivated many experimental studies to define the structure–function relationships of the tendon-to-bone attachment

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[17–19]. Experimental work has shown that, at the microscale, there exists a zone of high compliance within the fibrocartilage gradient that may reduce stress concentrations and mitigate failure at the attachment [14,15].

Regardless of the attachments' ability to reduce localized stress, rotator cuff tendon injuries are common and occur predominantly near the tendon–bone attachment [20–24]. To understand the high incidence of rotator cuff injury, numerous studies have uncovered how various conditions, such as tear size, abduction angle, and joint rotation, affect tissue-scale strain of the rotator cuff tendons [3,25–30]. Several of these investigations showed that, after injury, both structure and function are compromised, eliciting further damage via increased strain and tear propagation within the tendon midsubstance [26,31,32]. However, to date, the tissue-scale strain distribution of the rotator cuff tendon-to-bone attachment has yet to be elucidated. Therefore, this study aimed to identify the tissue-scale strain at the rotator cuff tendon-to-bone attachment with and without a localized defect.

The objectives of this study were to (1) determine the biomechanical properties and (2) regional tissue-scale strain patterns of intact and defect tendon–bone attachments in the infraspinatus of adult rats. We hypothesized that: (1) attachment defects would result in reduced ultimate load and stiffness due to reduced CSA, but not ultimate stress, strain at ultimate stress, Young's modulus, and area under the stress–strain curve (AUC) at ultimate stress compared to intact attachments, and (2) localized strain concentrates near the defect during uniaxial loading.

Methods

Defect Model and Sample Preparation. Adult, female Long Evans rats ($N=14$, 5–6 months old, 315.8 ± 12.67 g) were euthanatized and immediately frozen at -20°C . One animal was excluded due to data acquisition error, leaving $N=13$ paired intact and defect attachments for biomechanical and strain comparisons. Rats were thawed at 4°C prior to testing. This study was designed using paired comparisons of the left and right shoulders, with one shoulder (randomly assigned) serving as an uninjured control (intact group) and the other undergoing an attachment-site defect (defect group). For the defect group, the forearm was externally rotated, and a skin incision was made to expose the deltoid. The deltoid was then retracted, and the infraspinatus tendon–bone attachment was surgically exposed, visualized using a stereoscope (Stemi 2000 CS, Zeiss; Jena, Germany), and a 0.30-mm diameter cylindrical defect was made at the infraspinatus attachment using a biopsy punch (Robbins, Chatham, NJ). The biopsy punch was placed at the superior-edge of the tendon, nearest to the supraspinatus attachment, and pressed firmly until the punch completely penetrated the fibrocartilage, tendon, and cortical bone. After the defect was made, infraspinatus tendon-to-bone attachments were finely dissected under a stereoscope to remove the surrounding musculature, capsular tissue, epitenon, neighboring attachments, and bone. Tendon-attachment-bone complexes were kept hydrated in phosphate-buffered saline (PBS) prior to mechanical testing and tested within 48 h of dissection.

Prior to mechanical testing, stereoscopic images of the lateral and inferior sides of the attachment were taken (DSLR, Nikon, Minato, Tokyo, Japan), and ImageJ was used to measure the length of these sides [33]. The attachment was assumed to be rectangular for CSA approximation. For the Defect group, the diameter of the defect was measured in the lateral view and subtracted from the lateral attachment width.

To prevent failure at the growth plate during testing, a hole was made in the diaphysis using a rotary tool (Dremel, Racine, WI), and a steel wire was passed through and wrapped to secure the humeral head. The distal humerus was potted using poly-methyl methacrylate (Ortho-Jet BCA, Lang Dental, Wheeling, IL) in a 1.5 mL centrifuge tube, and guide needles were used to confirm that each sample was oriented at 0° deg of shoulder abduction, as

abduction angle is known to affect cuff tendon mechanics [29,34]. Abduction and rotation angles were chosen to mimic neutral shoulder position.

Mechanical Testing. After potting, individual 1.5 mL tubes were secured to a custom-built attachment with set screws. The custom attachment was used to ensure that individual tendons, both left and right, were consistently aligned in the direction of applied loading when gripped and attached to the material test stand (Instron 5943, Norwood, MA). Tissue paper (Kimwipes, Kimberly-Clark, Irving, TX) soaked in PBS was placed on each side of thin-film grips (FC-20, IMADA, Northbrook, IL) to prevent slipping from the grips during testing, and the thinnest portion of the tendon midsubstance was securely gripped. The attachments were speckle-coated with Verhoeff's stain [35]. Uniaxial tensile tests were performed in a custom PBS bath at room temperature. Image capture was performed using a high-speed camera (Basler A102f; Exton, PA with Navitar 6000 Video Zoom Microscope Lens; Rochester, NY) and custom LABVIEW program (National Instruments, Austin, TX). A 0.2 N tare load was applied, attachment-to-grip length was measured as initial length (l_0), and five preconditioning cycles were performed (0 – 0.05 mm at 0.01 mm/s). After a hold for 90 s, samples were loaded in uniaxial tension to failure at 0.01 mm/s.

Data Analysis of Mechanical Properties and Regional Tissue-Scale Strain. Ultimate load and stiffness, as well as ultimate stress, strain at ultimate stress, Young's modulus, and the AUC at ultimate stress, were computed for intact and defect groups (MATLAB, Natick, MA). The load (F) for each sample was divided by its original CSA to estimate maximum stress (σ)

$$\sigma = \frac{F}{\text{CSA}} \quad (1)$$

Initial attachment-to-grip length and crosshead displacement were used to calculate Lagrangian strain and was used to compute Young's modulus and AUC at ultimate stress. Strain at ultimate stress was calculated as the strain corresponding to the highest stress on the stress–strain curve. The elastic region was defined by manually choosing four points after the toe region and before yield, and stress–strain curves were generated using individual attachment CSA (measured prior to start of test). Stiffness and Young's modulus were calculated as the slope of the elastic region of the load–displacement and stress–strain curves, respectively. AUC at ultimate stress was calculated as the area under the stress–strain curve prior to the maximum stress.

Analysis of tissue-scale strain (ϵ) was determined at discrete loads including: toe region ($1/8$ ultimate load), at the start of the elastic region ($1/4$ ultimate load), near the end of the elastic region ($1/2$ ultimate load), and near the yield point ($3/4$ ultimate load). Strain fields were computed using incremental digital image correlation, with one image used per ~ 0.1 mm displacement (Vic-2D, Correlated Solutions, Columbia, SC). The images at each analyzed load were included using a custom Python script (v3.6.1, Python Software Foundation, Wilmington, DE). Pixel-wise longitudinal strain (ϵ_{xx}), transverse strain (ϵ_{yy}), and the absolute value of shear strain (ϵ_{xy}) were calculated by Vic-2D. Strain fields for ϵ_{xx} , ϵ_{yy} , and ϵ_{xy} for all intact and defect samples were created at each analyzed fraction of the ultimate load using a custom Python script.

Vic-2D regions of interest, i.e., regions analyzed for tissue-scale strain, were drawn in the tendon, attachment, and bone regions. Out of focus and defect regions were excluded from Vic-2D regions of interests. The zero-strain image at the start of each test was used to determine region boundaries. Using ImageJ, lines were drawn along the superior and inferior boundaries of each attachment, and a straight line was drawn through the middle of each attachment to separate the superior and inferior regions.

Fig. 1 Study design. Infrapinatus attachments were divided into two experimental groups: intact and defect. For the defect group, a biopsy punch was used to create a superior-edge, partial-width, full-thickness tendon-to-bone attachment defect. Uniaxial tensile tests were performed with simultaneous image capture to obtain: biomechanical properties, qualitative tissue-scale strain fields, and quantitative regional tissue-scale strain of intact and defect groups. For regional tissue-scale strain, the average median strain was compared for fibrous, calcified, superior, and inferior regions within and between intact and defect groups. The white cylinder represents the biopsy punch used to create the defect. The dashed rectangle is a zoomed in depiction of the attachment. The dashed circle represents the superior edge defect. The dashed line separates superior from inferior, and the solid line separates the fibrous attachment from the calcified attachment; together creating the four quadrants. T, tendon; B, bone; M, muscle.

Similarly, using ImageJ, the fibrous and calcified regions were defined by drawing a segmented line along each tendon-to-bone attachment (fibrous-calcified line). Strain data 0.3 mm above or below the fibrous-calcified line was included for regional analyses. Using these boundaries, Vic-2D strain values were separated into fibrous, calcified, superior, and inferior regions via a custom Python script (Fig. 1).

Statistical Comparisons. All statistical comparisons were performed using Prism (v7, GraphPad, La Jolla, CA). Paired *t*-tests were used to compare mechanical outcomes between intact and defect groups. Repeated measure (load; region) two-way analysis of variances with Sidak's correction for multiple comparisons were used to compare mean strain between fibrous and calcified regions at toe, elastic, and yield loads and to compare regions between intact and defect attachments near yield. All data presented are average median strain $\pm$ 95% confidence intervals.

Results

Cross-sectional area was significantly reduced for the defect group compared to the intact group (defect: $1.5 \pm 0.2 \text{ mm}^2$; intact:

Fig. 2 Biomechanical strength and AUC at ultimate stress decreased in tendon-to-bone attachments with defects. Structural properties of (a) ultimate load and (b) stiffness significantly decreased in the defect group compared to the intact group. (c) AUC at ultimate stress significantly decreased in the defect group, while other material properties of (d)–(f) Young's modulus, strain at ultimate stress, and ultimate stress were not significantly different between groups. Black lines indicate significant difference ($p < 0.05$); data shown are mean $\pm$ 95% confidence intervals.

$2.1 \pm 0.3 \text{ mm}^2$; $p = 0.0112$). The defect model depicted in the study design (Fig. 1) resulted in two qualitatively distinct groups, one with bridged defects in which the tendon was still intact on the superior side of the defect, and the other with edge defects in which the attachment experienced a notch-like defect. All intact samples failed via bone avulsion at the greater tuberosity, while a combination of failure patterns at the attachment, tendon midsubstance, growth plate, and greater tuberosity were observed for the defect group.

Biomechanical Properties. Stiffness, ultimate load, and AUC at ultimate stress were significantly reduced in the defect group (Fig. 2). Ultimate load, stiffness, and ultimate stress were significantly higher for the edge defect compared to the bridged defect (Supplemental Fig. 1 is available under the “Supplemental Data” tab for this paper on the ASME Digital Collection). No other biomechanical outcomes calculated in this study were significantly different between intact and defect groups and between edge and bridged defects (Fig. 2).

Regional Tissue-Scale Strain. Near yield, intact attachments showed a uniform distribution of longitudinal (ϵ_{xx}), transverse (ϵ_{yy}), and shear (ϵ_{xy}) strain (Figs. 3(a)–3(e)), while strain concentrations were evident for each strain component in the defect group (Figs. 3(b)–3(f)). Longitudinal strain in the defect group appeared localized, with regions of higher and lower strain (Fig. 3(b)) that was not evident in the intact group (Fig. 3(a)). Qualitatively, transverse strain appeared localized at the fibrous-calcified interface (Fig. 3(d)), and the magnitude of transverse strain in the fibrous region increased with increasing load for the defect group (Supplemental Figs. 2(b) and 3(c) are available under the “Supplemental Data” tab for this paper on the ASME Digital

Fig. 3 Near yield load, tissue-scale strain was heterogeneous and concentrated at attachment defects. Representative strain fields for (a), (c), and (e) intact and (b), (d), and (f) defect attachments are shown. Longitudinal (ϵ_{xx} ; (a) and (b)), transverse (ϵ_{yy} ; (c) and (d)), and shear (ϵ_{xy} ; (e) and (f)) strain fields were generated near yield load. Strain magnitude is represented by the colored scale bars. Images shown are representative for each group. Coordinates for measured strain are shown in panel C. (a) and (b) T, tendon; B, bone. (b), (d), and (f) The defect is marked with a dashed white circle.

Collection). For the intact group, transverse strain remained heterogeneous at the fibrous-calcified interface (Fig. 3(c)) and did not increase with increasing load for either the fibrous or calcified regions (Supplemental Figs. 2(b), 3(c), and 3(d) are available under the “Supplemental Data” tab for this paper on the ASME Digital Collection). A banding pattern of high and low shear strain across the attachment was observed for the intact group (Fig. 3(e)), whereas concentrations of shear strain at the fibrous-calcified border were apparent for the group (Fig. 3(f)). No noticeable differences in transverse, shear, or longitudinal strain were observed between the bridged and edge defects (Supplemental Fig. 4 is available under the “Supplemental Data” tab for this paper on the ASME Digital Collection).

No differences in average median longitudinal strain in fibrous and calcified regions of and between intact and defect groups were observed near yield load (Fig. 4(a)). In the fibrous region near yield load, average median transverse strain was significantly decreased in the defect group compared to the intact group (Fig. 4(b)). Additionally, average median transverse strain was significantly reduced in the fibrous region compared to the calcified region of the defect group (Fig. 4(b)). Average median shear strain in the fibrous region was significantly higher in the defect group compared to the intact group near yield load (Fig. 4(c)).

No differences between superior and inferior fibrous regions of intact and defect groups were observed for average median transverse strain (Fig. 5(a)), and no differences between superior and inferior fibrous regions were observed within intact and defect

Fig. 4 Near yield load, transverse and shear strain were concentrated in the fibrous region of attachments with defects. Average Median tissue-scale (a) longitudinal strain (ϵ_{xx}), (b) transverse strain (ϵ_{yy}), and (c) shear strain (ϵ_{xy}) were compared between intact and defect groups for the fibrous and calcified regions of tendon-to-bone attachments. Black lines indicate significant difference ($p < 0.05$). Data shown are average median strain $\pm 95\%$ confidence intervals.

Fig. 5 Near yield, shear strain localizes near tendon-to-bone attachment defects. (a) Average median tissue-scale transverse and (b) shear strain for the superior and inferior fibrous regions were compared. Black lines indicate significant difference ($p < 0.05$); data shown are average median strain $\pm 95\%$ confidence intervals.

groups for average median transverse and shear strain (Figs. 5(a) and 5(b)). However, average median shear strain was significantly increased in the superior fibrous region of the defect group compared to the same region in the intact group (Fig. 5(b)).

Discussion

This study quantified the biomechanical properties and regional tissue-scale strain at the rotator cuff tendon-to-bone attachment with and without a full-thickness defect. We found that the intact attachment distributes heterogeneous strain across the entire attachment, whereas an attachment with a defect develops stress concentrations near the defect. These findings emphasize the importance of the tendon-to-bone attachment in mitigation of stress concentrations and failure at the attachment.

Damage to rotator cuff tendons presents in the clinic and typically requires surgical reattachment of the tendon back to bone [20]. Tissue-scale studies have identified a multitude of structural and biomechanical properties of the intact, damaged, and repaired rotator cuff tendon-to-bone attachment; while native attachments resist failure, damaged or repaired attachments are mechanically weaker and often experience poor reintegration during healing [1,19,25,36,37]. To improve reintegration of the damaged attachment, a better understanding of the mechanical requirements of the attachment is necessary. Therefore, understanding the mechanical properties and distribution of strain at the intact and damaged tendon-to-bone attachment is important for the characterization of the attachment and for identifying methods to resist failure at the damaged attachment.

Previously, research by Andarawis-Puri et al. and Bey et al. have shown that increased damage correlates with increased tissue-scale strain [26–30,34]. Additionally, Thomopoulos et al. has modeled the collagen fiber orientation of the tendon-to-bone attachment and identified a potential role of the attachment to alleviate stress at the interface. This study used uniaxial biomechanical testing with digital image correlation to quantify biomechanical properties and regional tissue-scale strain of the rat infraspinatus tendon-to-bone attachment with and without a localized defect [21].

This study highlighted the importance of the attachment's structure in distributing strains across the entire surface of the attachment, potentially serving to prevent failures at the tendon-to-bone attachment. Attachment defects led to decreased failure load but not decreased ultimate stress suggesting that, even with defects, the tendon-to-bone attachment was still able to mitigate failure, albeit less than intact attachments. The intact attachment may resist failure, as suggested by increased AUC at ultimate stress compared to attachments with defects. One explanation of decreased AUC at ultimate stress in the defect group is that the defect may disrupt the hierarchical toughening mechanisms of the tendon-to-bone attachment, such as collagen alignment [9]. Another potential cause of reduced AUC at ultimate stress may be that strain concentrations, which increase localized stress, are created nearest the defect attachments as loading increases [26]. From these findings, it was evident that once the attachment is disrupted at the tissue-scale, an atypical attachment-site failure occurs at reduced ultimate loads. However, the location and size of injury that induces the highest stresses at the attachment remains to be uncovered. Future work using finite element analysis could model the development of stress concentrations at attachments with and without varying defect types and locations.

Limitations

The attachment is not symmetric or flat, and analyzing strain in two dimensions may impart error in our analysis. The attachment has a splayed geometry, with out-of-plane fibrocartilaginous interfaces that may introduce varying levels of local compression and tension [8,9]. These problems were controlled in our study by testing at specific abduction and rotation angles, and, by comparing

median strain, we attempt to limit bias from localized regions of high or low strain. Another limitation is that the tendon-to-bone attachment may not deform in a continuous manner; fibril sliding and fascicle recruiting may be responsible for the large variability in strain calculations [2]. Additionally, creating the defect introduced some variability that resulted in two subgroups: bridged and edge defect. The bridged group retained a thin part of the attachment on the superior-most edge, while the edge group completely removed the superior attachment and the rest of the attachment remained as one body. Although these two groups were biomechanically different (Supplemental Fig. 1 is available under the "Supplemental Data" tab for this paper on the ASME Digital Collection), for this study they were considered as one group because the strain measurements excluded a majority of the superior-most attachments in the Bridged group.

Conclusions

This work aimed to experimentally understand the theory that the tendon-to-bone attachment mitigates strain concentrations. The biomechanical properties and tissue-scale strain distributions of intact attachments and attachments with defects showed that intact attachments resist failure by evenly distributing strain across the interface, while attachments with defects have reduced load capacity as well as developed strain concentrations. Notably, this study was performed on rat tendon-to-bone attachments, which enables future work to investigate the strain properties of tendon-to-bone attachments in various scenarios of healing and repair. Our data suggest that future repair strategies should aim to redistribute or absorb the increased strain experienced at the defect site.

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Nomenclature

CSA = cross-sectional area

mm = millimeter

s = seconds

ϵ_g = grip-to-grip strain

ϵ_{xx} = tissue-scale longitudinal strain

ϵ_{xy} = absolute value of tissue-scale shear strain

ϵ_{yy} = tissue-scale transverse strain

σ = stress

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