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Rotator Cuff Biology and Biomechanics: a Review of Normal and Pathological Conditions

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Abstract The glenohumeral joint is a complex anatomic structure commonly affected by injury such as tendinopathy and rotator cuff tears. This review presents an up-to-date overview of research on tendon biology and structure, shoulder joint motion and stability, tendon healing, and current and potential future repair strategies. Recent studies have provided information demonstrating the serious impact on uninjured tissues after a rotator cuff tear or other cause of altered shoulder joint mechanics. Another major focus of recent research is biological augmentation of rotator cuff repair with the goal of successfully reinstating normal tendon-to-bone structure. To effectively treat shoulder pathologies, clinicians need to understand normal tendon biology, the healing process and environment, and whole shoulder stability and function.

Keywords Rotator cuff · Tendon · Shoulder · Biomechanics · Orthopedic surgery · Overuse injury · Tendinopathy

Introduction

The complex structure of the glenohumeral joint confers the shoulder with the most mobility of any major joint in the human body. This characteristic is primarily due to a limited interface between the humerus and the scapula, requiring the presence of a large network of ligaments, tendons, and other connective tissue elements to provide stability and allow functional movement. However, these tissues also have a propensity for injuries and degenerative conditions due to acute trauma or chronic overuse. Recent studies have investigated shoulder biomechanics, interactions between tissues within the joint, the responses of these tissues to stress, and the repair of the rotator cuff tendon after injury. Clinical studies, cadaver studies, and animal models offer new clues regarding the biology, biomechanics, and pathology of shoulder injury, influencing the development of clinical solutions to musculoskeletal problems.

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Anatomy and Background

The Shoulder Girdle

The shoulder is formed by connections between the scapula, the clavicle, and the humerus [1]. Scapular landmarks include the coracoid process superiorly, the glenoid cavity laterally, the subscapular fossa anteriorly, and the supraspinous and infraspinous fossae divided by the scapular spine posteriorly. The scapular spine extends laterally to a free end, the acromion, which articulates with the lateral end of the clavicle. On the anterior proximal humerus, from medial to lateral, are the lesser tuberosity, the bicipital groove, and the greater tuberosity.

The medial side of the humeral head is composed of articular cartilage, which is integral to providing a smooth gliding surface between the humeral head and the glenoid fossa [2]. It is a solid matrix composed predominantly of type II collagen and the proteoglycan (PG) aggrecan that can be divided into zones from the articular surface to the subchondral bone. Most superficially, the densely packed collagen is oriented parallel to the articular surface and PG

content is low. In the middle zone, fibers are more randomly oriented and PG content is the highest. The deep zone contains large collagen fibers oriented perpendicular to the subchondral bone and low PG levels.

Glenohumeral Joint and Ligaments

The glenohumeral (GH) joint is formed by the articulation of the humeral head with the glenoid cavity. Although the humeral head is much larger than the glenoid cavity, their curvatures differ by as little as 1 % [3]. The shape and concavity of the glenoid fossa may vary between individuals; however, the cavity is typically 5°–7° retroverted relative to the mediolateral axis of the scapula [4].

A variety of ligaments provide support to the shoulder, including the superior and inferior acromioclavicular (AC) ligaments; the coracoclavicular (CC) ligament; the coracohumeral (CH) ligament; the coracoacromial (CA) ligament; and the superior, middle, and inferior GH ligaments [1]. The CA ligament acts as the roof of the subscapular space and forms the CA arch along with the acromion and coracoid process. The labrum is a band of fibrocartilaginous tissue that can vary in shape and attaches around the margin of the glenoid cavity. The articular capsule of the GH joint attaches to the labrum, scapular neck, and anatomical neck of the humerus. It is stabilized by the GH ligaments anteriorly and CH ligament superiorly and also by the CA and AC ligaments. The inferior portion of the capsule is not reinforced, resulting in the axillary recess.

The Rotator Cuff

There are four muscles that constitute the rotator cuff [1, 5]. The supraspinatus originates from the supraspinous fossa of the scapula; its tendon passes through the subscapular space and inserts on the superior and middle facets of the greater tuberosity. The infraspinatus and teres minor both originate from the infraspinous fossa and fibrous septum, and their tendons insert on the middle and inferior facets of the greater tuberosity, respectively. The subscapularis originates from the subscapular fossa, and its tendon inserts on the lesser tuberosity. The rotator cuff is unique in that its tendons fuse to form a continuous structure near their insertions. Its bursal surface is covered by deep extensions from the CH ligament, while its articular surface is lined by the joint capsule.

The description of an anatomic footprint has aided in diagnosing and repairing rotator cuff tears [6]. The subscapularis has the largest footprint, inserting along the medial aspect of the bicipital groove. The second largest is that of the infraspinatus; its anterior border overlaps with the posterior border of the supraspinatus insertion. Between the articular surface and the inferior insertion of the infraspinatus is a gap called the bare area. The supraspinatus insertion, the third largest, extends from the lateral aspect of the bicipital groove to this bare area. The overlap zone between the supraspinatus and infraspinatus is located just anterior to the tip of the bare area and serves as an arthroscopic landmark. The supraspinatus, however, inserts closer to the articular surface. Finally, the teres minor has the smallest insertional footprint, directly inferior to the infraspinatus.

The tendon-to-bone insertion site is divided into four zones: tendon midsubstance, fibrocartilage, calcified fibrocartilage, and bone, with a gradual and continuous change in composition [7]. Zone 1, the tendon midsubstance, is composed primarily of collagen types I and XII and the PGs decorin and biglycan and contains spindle-shaped cells. As the tendon progresses toward the bony insertion, it takes on a more fibrocartilaginous form, composed mostly of collagens II, IX, and X, and the PG aggrecan and its cells have a rounded shape. Collagen orientation also transitions from well aligned in zone 1 to randomly oriented in zone 4.

A five-layer structure has been described at the confluence of the supraspinatus and infraspinatus tendons [5]. Layer 1 is the most superficial and contains fibers from the CH ligament. In layer 2, fibers come directly from the tendons and are parallel and densely packed. Layer 3 corresponds to the gross overlap of the tendons, and the fibers in this layer are more loosely packed than in layer 2. Layer 4 contains loose connective tissue and thick fibers from the deep extension of the CH ligament. Finally, layer 5 is the true capsular layer with randomly oriented fibers.

There are other adjacent structures in the shoulder that have an interrelated connection with the rotator cuff. One such structure is the long head of the biceps tendon (LHBT), which originates at the supraglenoid tubercle of the scapula. Its tendon perforates the articular capsule, travels distally in the bicipital groove, and ultimately, inserts on the radial tuberosity. In the bicipital groove, the LHBT is ensheathed by fibers from the supraspinatus and subscapularis tendons [8].

Biomechanics of the Glenohumeral Joint

General Properties of Tendons and Ligaments

Rotator cuff tendons are characterized by their nonlinear, viscoelastic, and heterogeneous material properties. As the primary component of the extracellular matrix (ECM) of both the tendon and its insertion, collagen is important to many of these properties. The hierarchical arrangement of collagen molecules, fibrils, and fibers allows several steps of deformation under axial loading, including uncrimping of wavy fibrils, straightening of twisted triple-helical molecules, and eventually molecular uncoiling. These effects provide the tendon with significant extensibility and strain hardening [9]. Cyclic

loading induces collagen alignment in the direction of the force, increasing tendon strength and creating nonlinear stiffness in response to strain [10, 11].

Components of the rotator cuff have unique anatomies, conferring specific mechanical properties and strain distributions across regions and surfaces. For example, the supraspinatus tendon consists of anterior and posterior subregions, with a significantly higher modulus of elasticity occurring in the anterior than the posterior subregion. This helps preserve the shape of the tendon during shoulder motion; however, dissimilar rates of deformation may play a role in initiating tears [12]. The inferior GH ligament can also be divided into three anatomical regions with variations in strain to failure. The weakest portion is the ligament midsubstance, suggesting larger strains must occur near the insertion sites [13]. However, this ligament interacts significantly with surrounding capsular tissue and should be evaluated in the context of a single sheet of fibrous tissue rather than discrete regions [14]. Likewise, the complex interactions between each component of connective tissue should be acknowledged during mechanical analyses.

Role of Tendons and Ligaments in Glenohumeral Joint Motion and Stability

The inherent bony stability of the shoulder is poor, as the articular surface of the proximal humerus and the glenoid are mismatched in size. The addition of the fibrocartilaginous labrum in conjunction with the joint capsule and GH ligaments aids in shoulder stability. Labral tissue increases the depth of the glenoid by 50 % and, together with compressive forces of the rotator cuff, imparts a concave compression on the humeral head into the glenoid. Maintaining a negative intra-articular pressure in a closed system within the capsule also helps to prevent translation of the humeral head [15, 16]. These soft tissue connections allow for a large range of motion and define roles for tendons and ligaments as specific active and passive restraints, respectively, during movement.

Early studies of shoulder stabilization involved dissection of various connective tissue components in cadavers. At 0° of abduction, the subscapularis muscle plays the primary stabilizing role; at 45° of abduction, the subscapularis, middle GH ligament, and a portion of the inferior GH ligament provide stability; and nearing 90° of abduction, the inferior GH ligament prevents dislocation [17]. Biomechanical loading studies offer additional information regarding directional stability. The LHBT provides more than 30 N of anterior stabilization in neutral rotation, with the subscapularis providing the majority of the stabilization in external rotation. Ligaments play a bigger role in stability as they become loaded at higher displacements [18]. The supraspinatus and biceps muscles are important active stabilizers in inferior stabilization, with the inferior GH ligament passively stabilizing in external rotation [19]. Posteriorly, the subscapularis muscle resists subluxation, with the CH ligament contributing in neutral rotation [20]. Release of the CA ligament to treat impingement causes GH laxity both anteriorly and inferiorly, indicating its importance as a static restraint [21].

Anatomical positioning of the rotator cuff muscles and the LHBT creates an ideal configuration to actively compress the humeral head into the cavity of the glenoid [22]. Shoulder anatomy also provides the rotator cuff muscles with short lever arms, establishing a stable and dynamic fulcrum during abduction (reviewed by Lugo et al.) [23]. Interestingly, individual shoulder anatomy, particularly acromion length and glenoid inclination, may predispose a shoulder to either osteoarthritis or rotator cuff tears, as the two pathologies occur together infrequently [24].

Anterior-Posterior Force Balance

Force couples occur when two opposing muscle groups create a given moment around a fulcrum. The rotator cuff creates a force couple around the GH joint, with coordinated activation and inactivation of agonist and antagonist muscles. The anterior-posterior force balance is defined by the subscapularis anteriorly and infraspinatus posteriorly (Fig. 1). Cadaver studies determined that GH joint motion is not affected as long as this force balance is intact [25]. Quantitative analysis confirmed that the direction and magnitude of joint reaction forces were most affected by the integrity of the anterior-posterior force balance, with no significant change after incomplete or complete tear of the supraspinatus tendon [26]. This dynamic relationship is an important aspect of understanding normal shoulder motion as well as how a disrupted force balance can play a role in shoulder pathologies, discussed below.

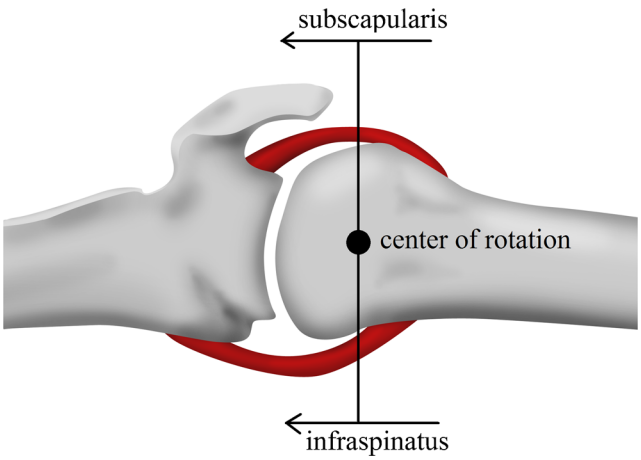

Fig. 1 Transverse plane force couple. The infraspinatus tendon posteriorly balances against the supraspinatus tendon anteriorly

Interaction of Tendons with Adjacent Tissues

Rotator Cuff Tendon Response to Stress

While tendons and ligaments are important for creating a stable joint, they are also dynamic tissues that respond to loading and change due to age and use. Tendon tissue adapts to mechanical loading via temporary upregulation of metabolic activity, specifically in collagen expression and synthesis regulated by tenocytes experiencing strain [27]. In opposition, another response to exercise is an increase in the expression of matrix metalloproteinases, promoting collagen turnover. A positive net balance requires a period of rest, without which the tendon will undergo continuous loss of collagen [28]. Consequently, the tendon milieu changes significantly in response to overuse, creating a mechanically inferior connective tissue. Rotator cuff overuse has been modeled in an established rat model in order to study the effects of such chronic use common in athletes and manual laborers performing repetitive overhead activities. Initial studies demonstrated increased tendon size and deteriorated mechanical properties after completion of an overuse exercise protocol [29]. Rotator cuff tenocytes undergo phenotypic change toward chondrocyte-like behavior, with increased PG expression, glycosaminoglycan accumulation, and upregulated SOX9 [30, 31•]. Significant upregulation of pro-inflammatory cytokines and apoptotic genes occurs over time as microinjuries develop and accumulate [32, 33]. Increased mechanoreceptor expression suggests that proprioception or pain is amplified after excessive physical activity [34]. Together, these changes help define and model the degenerative, tendinopathic state of the human rotator cuff.

For the human patient, the diagnosis of tendinosis or tendinopathy is characterized by a chronic condition typically lacking frank inflammation [35]. Characteristic pathological changes include decreases in tenocyte numbers and more rounded cell shape, increased apoptosis, increased PG content and disorganized collagen, and increased adrenergic receptors [36]. Therefore, the terminology and treatment approach for an inflammatory condition (tendinitis) is largely unwarranted. More recently, the field has also revised the way it defines rotator cuff impingement. Over four decades ago, contact between the rotator cuff tendons and the acromion or the undersurface of the AC joint was identified as a primary causative for rotator cuff disease [37]. Indeed, compressive forces initiate major physiological changes in the tendon, reminiscent of overuse pathology [38]. However, three recent reviews independently found no evidence to correlate tendon impingement and tendinopathy, particularly that associated with aging. There is also no proven correlation between the shape of the acromion and a positive surgical outcome or degree of shoulder pain with rotator cuff pathology, suggesting that preventing contact between the two tissues is not the most critical treatment [39–41]. In fact, the rotator cuff contacts the CA arch in normal shoulders in both cadaveric studies and in healthy human subjects [40].

Fatigue damage initiates with isolated changes in microstructure and develops into severe matrix disruption and kinked deformations [42]. This degeneration as well as biological changes due to aging or overuse can predispose a tendon to failure. The severity and location of tears within rotator cuff tendons vary considerably, being either full or partial thickness and located on either the bursal or articular side of the tendon. However, partial thickness and bursal side tears in particular have been shown to induce significant localized tendon strain concentrations in regions adjacent to the tear, leading to tear propagation [43•].

Rotator Cuff Tears: Changes in Joint Kinematics

As reviewed above, the rotator cuff musculature provides balanced forces that impart mobility and stability to the GH joint. Disruption of this innate force couple results in abnormal joint kinematics, as the stable fulcrum for rotation of the humeral head in the glenoid is lost [44]. Such force changes are dependent on tear size and location. Anterior tears of the supraspinatus insertion are more likely to be symptomatic and progress as a result of increased regional strain patterns due to joint force imbalance, potentially causing additional pain and requiring surgical intervention [45]. However, completely repairing a rotator cuff after a chronic multi-tendon tear can be challenging due to tendon retraction and stiffening. Instead, restoring the balance of the anterior-posterior forces by repairing only the infraspinatus in a supraspinatus-infraspinatus tear may be sufficient to restore shoulder function [46].

Joint Damage After Rotator Cuff Tears

Disruption of the force balance and normal shoulder stability and motion after loss of tendon function initiates changes in almost all adjacent tissues. Rotator cuff tears are often accompanied by tears in the glenoid labrum. Superior humeral head translation and loading of the LHBT due to decreased stabilization cause displacement of the labrum and increased labral tissue strain [47]. A number of studies utilizing the rat model further investigated these and other changes. Without repair, rotator cuff tears can cause cartilage degeneration in the labrum, putting it at risk for injury [48•]. Returning to a high level of activity increases the severity of this damage, significantly decreasing the expression of cartilage matrix proteins such as type II collagen and aggrecan in the glenoid [49]. Articular cartilage of the humeral head also shows surface irregularities, loss of PGs, and clonal chondrocyte formation 12 weeks after rotator cuff transection [50]. Mechanical properties of adjacent untorn tendons, including the LHBT and the subscapularis, also deteriorate, becoming stiffer at both the

insertion and the midsubstance [51, 52••]. Functional impairment of muscles associated with torn tendons decreases their potential to produce normal forces after repair, largely due to atrophy and fatty infiltration [53•]. Moreover, chronic fibrosis increases muscle stiffness, increasing tension at the repair site and impeding the repair process [54]. Adjacent muscles react in a compensatory way, becoming hypertrophic [55]. Clearly, normal shoulder stability and motion defined by the rotator cuff are crucial in maintaining the health of the entire shoulder.

Long Head of the Biceps Tendon Lesions and Tenodesis

Biceps tendon injuries are associated with rotator cuff tears, with increasing damage occurring as tear size increases [56, 57]. The rat model reveals an increase in size of the LHBT of up to 220 % after rotator cuff injury, as well as worsening mechanical properties over time [58]. Inflammation causes friction between the tendon and the bicipital groove, creating significant pain. Tenodesis or tenotomy is an extremely successful treatment option, with persistent pain occurring with an incidence of only 0.2 % [59]. Unlike major tendons of the rotator cuff, severing the LHBT does not dramatically alter dynamic GH movement, supporting the use of tenotomy in conjunction with rotator cuff repair to reduce pain and increase function [60•]. A recent prospective, randomized study determined no difference in shoulder function, strength, or patient satisfaction between the two procedures. However, a shorter surgical time and faster pain relief for those treated with tenotomy may make this technique more advantageous [61•]. Interestingly, detachment of the LHBT in the presence of a multi-tendon cuff tear results in improved shoulder function and less joint damage in the rat model, indicating that early management of biceps pathology may diminish long-term effects of rotator cuff tears to adjacent tissues (Fig. 2) [62•]. Overall, the procedure is gaining popularity and is well accepted by most patients with high satisfaction levels.

Rotator Cuff Repairs

Current Practice for Rotator Cuff Repairs

Rotator cuff tears are one of the most common shoulder injuries, affecting more than 40 % of patients over the age of 60 and resulting in 30,000–75,000 repairs performed annually [63]. The theoretical advantages of arthroscopic repair have led to its increased popularity over an open approach. Data suggest that arthroscopy should be performed for repair of small and medium tears, while for large and massive tears, open repairs may result in superior outcomes in some cases [64–66]. Double-row suture anchors, particularly with rip-stop fixation, allows for better anatomic footprint coverage, greater resistance to suture pull through, and increased compression throughout the tendon. Although radiological, clinical, and biomechanical data exist supporting its use over single-row repairs of large and massive tears, statistically significant data are not available for use in repairs of small and medium tears [67–69]. Following surgery, there is a period of protection that is suggested in order to allow for optimal healing without sacrificing range of motion (ROM) [70]. Currently, surgeons often recommend 6 weeks of immobilization in a sling, especially in large and massive tears, after which active ROM exercises are gradually incorporated with resistance exercises initiated around 3 months post-repair.

Despite optimization of mechanical construct and post-operative rehabilitation, failure rates range widely from 9 to 94 % [71, 72••]. There are a number of factors that have been implicated in the failure of cuff repair, including tear size, tissue quality, fatty infiltration, age, diabetes, smoking, osteoporosis, and duration from onset of symptoms to time of surgery [66, 73, 74, 75••, 76]. Of these, the most important predictors of outcome have been found to be age, tear size, extent of fatty infiltration, muscle atrophy, and amount of retraction at the time of surgery [75••, 76].

Rotator Cuff Tendon Healing

Rotator cuff tendon healing involves three overlapping stages: inflammatory, fibroblastic, and remodeling, detailed in Table 1 [66, 76]. During these stages, there are complex interactions between a variety of tissue growth factors and cells, ultimately resulting in tissue that is markedly different from that of normal, uninjured tendon. The healing tendon is composed primarily of collagens I and III, which are more characteristic of scar tissue. Studies have shown that despite advancements in surgical techniques and rehabilitation protocols, the healed tendon fails to recreate the normal transition seen in uninjured tissue. Instead of four zones, a three-layer fibrovascular construct is formed that has significantly less fibrocartilage than normal. Developing ways to restore the composition and structure of the normal transition site will be crucial in reducing the failure rates of rotator cuff repair procedures.tgroup

The growth factors that have been studied include IGF-1, FGF-2, MMP-3, TIMP, VEGF, TGF-β1, PDGF, CTGF, and BMP12-13. The exact roles that these factors play, however, are still unknown. Tenocytes from irreparable cuff tendons can be stimulated to produce the ECM proteins of a healthy tendon after local delivery of PDGF [77•]. This would suggest that not only do cuff tendons have healing capabilities but also that this healing environment can be manipulated.

In the rat rotator cuff injury model, TGF-β1, an important signaling molecule in remodeling, is localized to the repair tissue and correlates with a peak in cellular activity [78]. TGF-β3, which is involved in tissue regeneration, is not detected at the repair site; however, it has been shown to

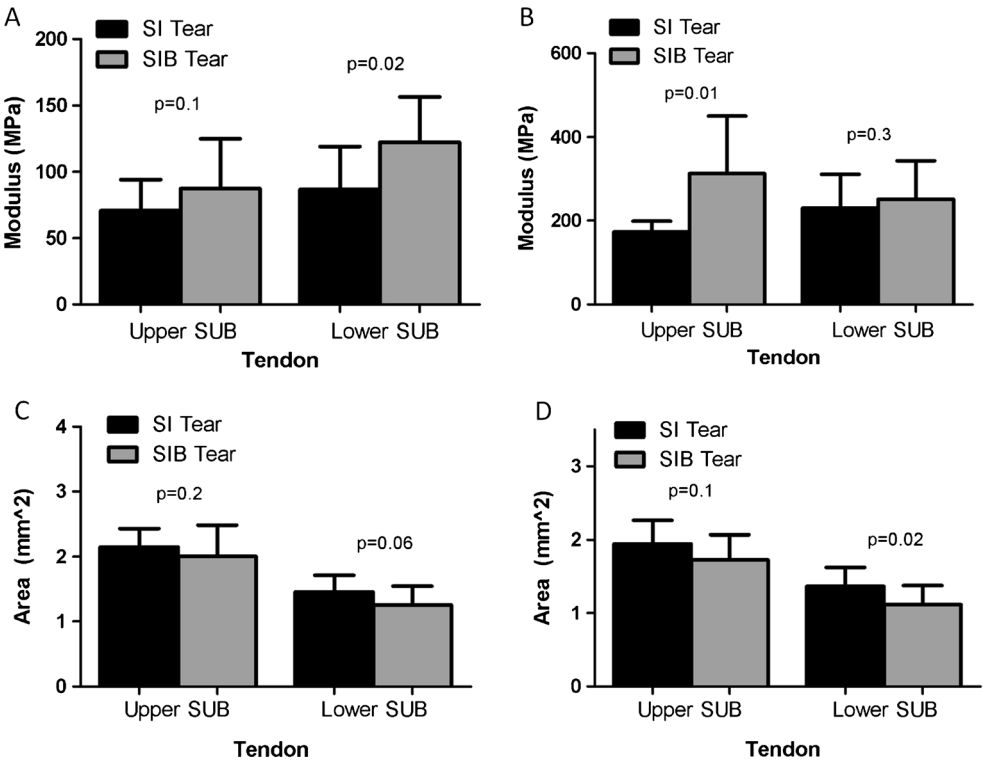

Fig. 2 Biceps tenotomy prevents damage to uninjured tendon in the presence of a rotator cuff tear. Compared to the group without tenotomy, the groups with tenotomy showed **a** increased elastic modulus for the lower subscapularis insertion site and **b** increased elastic modulus for the upper subscapularis midsubstance. **c** No significant change in area was seen at the insertion site, but **d** the cross-sectional area of the lower subscapularis midsubstance was decreased within the group that received tenotomy. Data are shown as mean+SD. *SI* supraspinatus and infraspinatus; *SIB* supraspinatus, infraspinatus, and LHBT; *SUB* subscapularis. (Reproduced with permission from [62•])

improve tendon-to-bone healing after exogenous delivery [79]. Matrix protein expression also fluctuates during healing. While type III collagen is predominantly expressed at early time points (day 10–14), by later time points (day 52) there is also a large quantity of new type I collagen. This suggests that while a significant period of healing occurs early, it is an ongoing process.

The Future of Rotator Cuff Repairs: Changing the Healing Environment

Recently, research has focused on augmenting the biological environment in the healing tendon. Based on their success in other orthopedic procedures, two biological therapies—platelet-rich plasma fibrin matrix (PRPFM) and porcine small

Table 1 Phases of tendon healing

Phase	General characteristics and cell activities	Time scale	Cells involved	Proteins expressed
Inflammatory	Hematoma formation, release of proinflammatory molecules, neovascularization, cell recruitment and infiltration	Days	Fibroblasts, macrophages, phagocytes, neutrophils	Collagen type III, MMP9 and 13, NOS, VEGF, TGFβ, BMP-12, bFGF, IGF
Fibroblastic	Continued release of growth factors, cell proliferation, and disorganized matrix production	Weeks	Fibroblasts	Collagen types I and III, MMP2, substance P, PDGF, bFGF, TGFβ
Remodeling	Reorganization of collagen, GAG production, decreased cellularity and vascularity	Up to 1 year	Fibroblasts, tenocytes	Collagen type I, MMP2 and 14, TGFβ

intestine submucosa (SIS)—were tested in rotator cuff repairs. Numerous studies, however, have shown that PRPFM does not improve clinical healing rates or outcomes and that porcine SIS not only fail to decrease surgery failure rates but can also lead to increased pain, decreased function, and sterile effusions [63, 66, 73]. Investigators have also studied the effects of other growth factors and cell therapies, including PDGF, TGF-β1, bone marrow-derived mesenchymal stem cells treated with insulin, and FGF-2-coated sutures [73, 77•, 78, 80]. These treatments beneficially alter the healing tissue by causing it to express proteins and exhibit mechanical properties closer to that of native cuff tendons. Studies evaluating the efficacy of current FDA-approved ECM and synthetic scaffold devices have shown mixed results. The acellular human dermal matrix device GraftJacket, for example, has good biocompatibility and leads to significant improvement in shoulder scores, pain scores, and healing rates on postoperative MRI [63, 81]. In vitro studies, however, have failed to show a significant biomechanical improvement. Based on animal and clinical studies, the synthetic polycarbonate polyurethane scaffold Biomerix RCR Patch has a biocompatible host response and improves outcome scores [63]. The results of many current studies, however, are difficult to interpret because of poor study design (e.g., lack of control group, minimal rotator cuff tear), limited scope of investigation (e.g., single growth factor therapy), questionable translation into clinical efficacy (e.g., insufficient follow-up periods), and finally, and possibly most importantly, because the mechanisms by which these augmentation devices function are not fully understood.

Conclusions

Rotator cuff tears are a common musculoskeletal condition, which leads to pain, restricted motion, and lost productivity. Understanding tendon biology and pathology as well as joint biomechanics is fundamental for providing adequate treatment of shoulder injuries. Here, we have updated current concepts regarding rotator cuff impingement, the role of the LHBT in rotator cuff tears, the importance of maintaining force balances within the joint, and how changes in joint mechanics impact surrounding tissues. Additionally, we suggest that biological augmentation of cuff repair is critical for improving clinical outcomes. Further understanding of appropriate tendon healing biology will be necessary to refine these therapeutic approaches.

Compliance with Ethics Guidelines

Conflict of Interest Julianne Huegel and Alexis A. Williams declare that they have no conflict of interest.

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Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance

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