Biologic Augmentation in Rotator Cuff Repair
Should We Do It, Who Should Get It, and Has It Worked?

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Abstract
Rotator cuff tears are a common pathologic entity, and rotator cuff repairs are a frequently performed procedure. Given the high rate of structural failure of repair, biological augmentation of repairs is increasingly important. Biological augmentation primarily enhances the healing response and secondarily provides a mechanical bridge for tension free repair. Understanding biology of tendons and tendon healing aids in determining an optimal environment for repair. The basic principles of rotator cuff repair are aimed at achieving high initial fixation strength of the repair, restoring the anatomic footprint of the cuff tendon, minimizing gap formation, and maintaining mechanical stability until biologic healing occurs. Methods of augmentation come in many different forms and can be categorized by cell type and mechanism of delivery. Cell types include individual growth factors, stem cells, or a combination of both. Vehicles range from in situ delivery, such as microfracture, direct injection, or scaffold materials that are biologic or synthetic.

Rotator cuff tears are a common pathologic entity, and the incidence of rotator cuff tears increases with age. The incidence of rotator cuff tears in the general population is approximately 20% regardless of symptomatology but approaches 50% in individuals over 80 years old.1 The most common cause of rotator cuff tears is degenerative in nature. Rotator cuff tears are less often caused by a specific traumatic event. Most often symptoms are insidious in onset, although they can become acutely worse after a traumatic event.

Not only are rotator cuff tears a common problem, but rotator cuff repairs are a common procedure performed, with over 75,000 rotator cuff repairs annually in the USA.2 Outcomes after rotator cuff repair are typically very good, with greater than 80% to 90% of patients experiencing long-term relief of pain and return of function. While there is a high rate of clinical success, follow-up imaging studies have shown that there may be a low rate of structural success of the repair, with imaging often showing incomplete healing or re-tearing. A number of factors increase the chances of having a structural failure or poor clinical outcome, including age greater than 65 years, large or massive tear, greater than 50% fatty atrophy or tendon retraction, osteoporosis, and comorbidities, such as diabetes or smoking.3

With the high rate of structural failures after rotator cuff repair and the prevalence of patients at increased risk of failure, a significant amount of time and resources have been dedicated to find ways to improve outcomes with augmentation. The goal of biologic and pharmacologic augmentation is two-fold: primarily to enhance the healing response and secondarily to provide a mechanical bridge for tension free repair.

Anatomy
Treatment of rotator cuff tears requires a good understanding of the anatomy of the rotator cuff and in particular the supraspinatus tendon, as this is most commonly affected in rotator cuff tears. Dimensions of the footprint have been extensively studied, as restoring the footprint during cuff repair plays an important role in tendon healing. The supraspinatus tendon is 10 mm to 14 mm thick and inserts on the superior facet and the superior half of the middle facet of the greater tuberosity.4 The rotator cuff insertion anterior-posterior dimension is 37.8 mm, and the medial-lateral dimension is 14.7 mm. The suprascapular nerve innervates the supraspinatus muscle, and the suprascapular artery is the blood supply. The supraspinatus tendon has two primary functions: abduction
of the arm and stabilization of the glenohumeral joint. Rotator cuff tears usually lead to pain and loss of function, with the patient unable to elevate the arm above 90°.

At the microscopic level, the tendon fibrils of the supraspinatus tendon inserts directly into the greater tuberosity of the proximal humerus through four zones: tendon, unmineralized fibrocartilage, mineralized fibrocartilage, and finally bone. This organization is not seen in healed large rotator cuff tears. Instead, there is a disorganized fibrovascular scar, and this disorganized scar may play a role in failure after repair. Biological augmentation seeks to replicate the organized tendon-bone interface in order to improve healing and repair strength.

### Biology

Tendons are made up of fibrous connective tissue that transmits the force of muscle contraction to bone in order to move an extremity. The tendon is made up of densely packed collagen fiber bundles aligned parallel to the tendon axis. The collagen fibers are surrounded by a tendon sheath made up of the extracellular matrix (ECM).

There are three components of a tendon at the most basic level: collagen, the tenocyte, and the extracellular matrix. Collagen is the basic unit of the tendon and structurally consists of a single polypeptide alpha chain arranged into a triple helix with hydrogen bond cross-links. The triple helix structure is arranged parallel to the direction of action and is held together by intermolecular bonds into fibrils. There are different types of collagen; most common in tendons is type I collagen. The endotenon and epitenon, which surrounds the type I collagen, primarily consists of type III collagen. Type III collagen is weaker, and synthesis of type III collagen balances the activity of MMP. These enzymes also play a role in the development of tendon degeneration. The expression of MMP3, TIMMP2, and TIMMP3 is decreased in torn rotator cuff tendons.

Del Buono and coworkers found that administering endogenous MMP inhibitor at the greater tuberosity was associated with reduced collagen degradation and increase collagen organization at 4 weeks in an animal rotator cuff repair model.

Tendon biology can be described by three basic principles: structure, biomechanics, and healing response to stress. Structurally, tendons are made up of fascicles surrounded by epitenon. The fascicles are a collection of collagen fibrils surrounded by an endotenon. The collagen fibril is a collection of collagen fibers that is the triple helix arrangement of collagen. Between the collagen fibers are the tenocytes and the ECM. In regards to biomechanics, the collagen fibers are crimped at resting state, and in response to stress, the fibers elongate. The interactions between collagen fibrils at a macrolevel dictates the strength of the tendon, resulting in the stress strain curve. The tendon healing response is mediated by the tenocyte, which responds to mechanical load and shear by releasing growth factors that stimulate the healing process.

The mechanical properties of the tendon depend on the intramolecular and intermolecular bonds within the tendon. The load-elongation curve (or stress-strain curve) represents how the tendon changes in length in response to a given force. On the load-elongation curve, the x-axis represents strain, which is the change in length over the original length of the tendon. The y-axis represents the stress, which is the force per unit area. When placed under tension, the tendon fibers straighten, and the system becomes stiffer. The slope of the load elongation curve corresponds to the stiffness. There are three regions of the curve that can be observed prior to tendon rupture: initial toe region, linear region, and failure region. In the initial toe region, there is a flattening of the crimp pattern with little force required to elongate the tissue. This protects the tendon from large loads during normal joint motion. In linear region, most of the fibers are aligned in parallel to the longitudinal axis of tension and elongation of the helical structure on fibrillar and macromolecular levels occur. In the failure region, further loading results in small reductions in stiffness, which represents the failure of a few fibers. Eventually, there is major failure of tendon as the fibers recoil and rupture.

Under normal conditions, the tendon undergoes continual remodeling, including degradation and rebuilding. This remodeling is mediated by matrix metalloproteinases (MMPs) located in the extracellular matrix. MMPs degrade collagen in the ECM, whereas tissue inhibitor of MMP (TIMMP) balances the activity of MMP. These enzymes also play a role in the development of tendon degeneration. The expression of MMP3, TIMMP2, and TIMMP3 is decreased in torn rotator cuff tendons.

Tendon failure typically occurs as a result of repetitive loading at submaximal failure loads, which leads to fatigue and eventual failure of the tendon. The pathogenesis of rotator cuff tendon tears is multifactorial and can be broadly categorized into intrinsic and extrinsic factors. Extrinsic factors involve impingement from structures surrounding the rotator cuff. Subacromial impingement leads to the compression of the rotator cuff tendon within the subacromial space from anatomical or biomechanical abnormalities. Internal impingement occurs in patients whom are overhead throwing athletes and involves compression of the insertion of the supraspinatus between the humeral head and the postero-superior glenoid rim with the arm in external rotation at 90° abduction in the throwing motion. These impingement conditions are influenced by a number of anatomical variables, including spurring at the anterior
edge of the acromion or at the acromioclavicular joint, the shape of the acromion, or scapular kinematics.

Intrinsic degeneration occurs from changes within the tendon. Degeneration of the tendon occurs when tensile loads exceed the tendon’s intrinsic healing and adaptive responses. This balance depends on the tendon’s vascular-ity, morphology (collagen content or elasticity), and genetic predisposition. The increased amount and duration of load on a tendon results in activation of protein kinases, oxygen free radicals, and apoptotic mediators, which when persistently activated cause tendon cells to undergo apoptosis.10

For large and massive rotator cuff tears in particular the healed tendon is more disorganized as compared to the pre-injury tendon. After rotator cuff repair, a reactive scar is formed.11 The normal insertion site with transition of unmineralized fibrocartilage to mineralized fibrocartilage to bone structure is not present in the repaired tendon. The repaired tendon is composed of a fibrovascular scar with a large proportion of type III collagen. A number of causes have been identified as to why the repaired tendon is disorganized, including disorganized expression of cytokines in the healing response, presence of inflammatory cells at the tendon-bone interface, slow and limited bony ingrowth between the tendon and prepared tuberosity, and insufficient number of undifferentiated stem cells at the healing tendon-bone interface.11

Clinical Presentation and Management

Patients with rotator cuff tears usually have an insidious onset of progressive pain and weakness, and they often complain of pain that occurs at night. There is usually a loss of active motion; however, passive motion is usually preserved. Nonetheless, pain can lead to adhesive capsulitis and loss of passive motion. The diagnostic workup includes radiographs to rule out other pathology, such as acromioclavicular joint arthritis or glenohumeral joint arthritis, which usually presents as loss of both active and passive range of motion, as well as MRI. The majority of patients with rotator cuff tears can be treated non-operatively. Patients with partial rotator cuff tears and older patients with insidious onset of symptoms should be given an initial trial of non-operative management, which includes use of nonsteroidal anti-inflammatory drugs and physical therapy to strengthen the remaining intact rotator cuff and scapular stabilizers. Indications for surgery include symptomatic traumatic tears and atraumatic tears that have failed conservative management. Symptomatic traumatic tears are more likely to fail non-operative management and are more common in young patients or older patients with acute loss of strength after trauma. Petersen and Murphy prospectively followed 36 patients with acute symptomatic rotator cuff tears and found that those repaired prior to 4 months after injury had better outcomes than those repaired after 4 months.12 Contraindications to surgical management include the presence of an active infection and concomitant stiffness, secondary to adhesive capsulitis. Preoperative stiffness must be corrected prior to rotator cuff repair to eliminate a potential cause of postoperative stiffness.

Techniques for Rotator Cuff Repair

The techniques for rotator cuff repair have developed over time in an effort to maximize tendon healing. Muller, in 1889, described the repair of a rotator cuff tear after a shoulder dislocation using catgut sutures.13 In 1906, von Perthes described the reattachment of the supraspinatus tendon to the greater tuberosity using staples. The repair was performed in conjunction with repair of an avulsed capsule and labrum from the glenoid rim for unstable shoulders.13 Codman, in 1911, described the deltoid splitting approach with suture repair of the ruptured supraspinatus tendon. He reported on the results of two patients with a ruptured supraspinatus tendon whom postoperatively had improved pain and complete restoration of abduction compared to one additional patient who refused surgery and had no return of function and continued to have pain 4 years after the onset of symptoms.14 In 1938, Outland described a suture repair of chronic supraspinatus tendon rupture to the greater tuberosity using drill holes, which is essentially the same transossous technique employed years later.15 Neviaser was the first to report using graft to augment cuff repair in 1971.16 He reported using an intraarticular portion of the biceps tendon as a free graft to repair a chronic massive rotator cuff tear. Later, in 1978, Neviaser went on to report a technique and preliminary results of a chronic massive rotator cuff repair using freeze-dried rotator cuff allograft.17 The first to report using arthroscopy for the treatment of rotator cuff tears was Ellman in 1987. He described performing subacromial decompression for the treatment of partial and full thickness tears.18 In 1990, Levy described a “mini-open” arthroscopic subacromial decompression with deltoid splitting rotator cuff repair.19 Lastly, in 1998, Gartsman presented results of a series of patients using an all-arthroscopic repair of full thickness tears.20

The basic principles of rotator cuff repair are aimed at achieving high initial fixation strength of the repair, restoring the anatomic footprint of the cuff tendon in a tension free manner, minimizing gap formation, and maintaining mechanical stability until biologic healing occurs.21 With these goals in mind, one should follow a stepwise surgical approach to arthroscopic management of rotator cuff tears. First, perform a complete subacromial debridement and bursectomy. Second, perform adequate mobilization of the cuff by releasing adhesions as needed. Third, define the edges of the cuff tear and identify the tear pattern. For retracted tears margin, convergence techniques can be used to bring the tear closer to the footprint. If it is not possible to bring the cuff to the footprint after initial capsular releases, interval slide techniques can be used to reduce tension and increase mobility.

Footprint reconstruction should be performed to provide high initial fixation strength and to recreate, if possible,
the broad anatomic footprint, which is thought to improve the likelihood of healing. The different constructs of footprint reconstruction include single row, double row, and transosseous equivalent repairs. With single row fixation, the anterior and posterior suture anchors are placed 1 cm lateral to the articular margin of the humeral head. With the double row construct, a lateral row is placed 1 cm lateral to the medial row of anchors approximately at the lateral edge of the repair footprint. The double row construct has clear biomechanical advantages, including increased strength, less gap formation, and better anatomic footprint reconstruction. However, the clinical advantages of using this technique are not yet clear in the literature. Demand and colleagues found an advantage in double row repair for massive rotator cuff tears. DeHaan and associates performed a meta-analysis and found a trend towards functional improvement with double row repairs for large to massive tears, although not statistically significant. Transosseous equivalent is a modified double row repair technique and is biomechanically equivalent to the open transosseous repair.

**Outcomes After Repair**

There is a high rate of clinical success after rotator cuff repair, with various studies showing a greater than 80% excellent or satisfactory outcome at greater than 10 years after surgery. Cofield and coworkers reported 80% excellent or satisfactory outcomes after 13-year follow-up but found that older patients with massive tears had significantly worse outcomes. Wolf and colleagues reported 94% good or excellent results with average 6-year follow-up. Finally Galatz and associates reported 72% of patients had an ASES score greater than 90 for large and massive cuff tears treated arthroscopically after 3-year follow-up.

Failure after rotator cuff repair can be attributed to extrinsic or intrinsic causes, with intrinsic causes further subclassified into intra-articular or extra-articular causes. Extrinsic causes can include cervical spine disease and suprascapular neuropathy. While for intrinsic causes, the intra-articular pathology could include glenohumeral arthritis, adhesive capsulitis, instability, labral pathology, or biceps tendinopathy. Extra-articular component includes persistent subacromial impingement, acromioclavicular arthropathy, or persistent rotator cuff defect. There are a number of factors associated with clinical and structural failure after rotator cuff repair: age greater than 65 years, large and massive tears greater than 3 cm, moderate to severe muscle atrophy, greater than 50% fatty infiltration of involved cuff, tear retraction greater than 2.5 cm, and diabetes. These factors have consistently been found to increase a patients risk of failure.

**Biological Augmentation**

The main goal in biological augmentation is to enhance the body’s natural healing response. This can be applied to each of the phases of tendon healing. The inflammatory phase promotes early scar formation and angiogenesis. The proliferative phase stimulates the production of collagen and ECM. The remodeling phase involves the conversion of type III collagen to type I collagen, and ECM and fibrous tissue becomes more organized. Augmentation involves supplementing components of the healing response and providing a scaffold for local delivery of these components as well as structural support for the repair. Increased structural support mechanically offloads the repair to allow early shoulder movement.

Biologic augmentation seeks to enhance the natural healing response, recreate normal anatomy, provide cell types involved in the healing response, and act as a vehicle for local delivery and structural support. Primary healing after tendon injury occurs in three overlapping phases. The first stage is the inflammatory stage, which occurs from day 1 to day 3. Injury to blood vessels in the area leads to hematoma, and this clot activates the release of chemotactic factors. Inflammatory cells are recruited, including red blood cells, platelets, neutrophils, monocytes, and macrophages. Platelets deposit fibrin and fibronectin and secrete IGF-1, PDGF, and TGF-β. Macrophages secrete TGF-β1, which promotes the formation of scar tissue. The proliferative phase occurs between day 3 and the sixth week. There is continued recruitment and proliferation of fibroblasts via expression of cytokines, and these then synthesize collagen type III. The final phase, the remodeling phase, occurs between the sixth week and 1 year. Consolidation occurs during this phase around the tenth week and involves the conversion of type III collagen to type I collagen. By 1 year, maturation occurs with gradual change in fibrous tissue to scar-like tendinous tissue. The hallmark of this stage is the reorganization of collagen fibers and the decreased cellularity and vascularity within the tendon.

At each stage of the healing process, different growth factors play a key role in the normal healing response. Expression follows a very specific timeline during cuff healing. Bone morphogenetic protein (BMP) stimulates bone formation, and BMP-1, BMP-12, BMP-13, and BMP-14 are expressed in the acute phase of healing after a rotator cuff tear. BMP-12 and BMP-13 are important regulators of fibrocartilage, neotendon, and ligament formation. bFGF affects proliferation and collagen secretion of rotator cuff tendon cells. Expression in vivo peaks between 5 and 9 days after tendon rupture. PDGF promotes expression of other growth factors and recruits inflammatory cells to the site of repair. Expression peaks between 7 and 14 days after injury. TGF-β plays a role in normal fetal development of tendons, as well as in modulation of scar tissue in tendon-bone healing. TGF-β is present during the acute phase of healing in three isoforms: TGF-β1, TGF-β2, and TGF-β3. IGF-1 is upregulated in the initial inflammatory phase of tendon healing and stimulates chemotaxis and proliferation.
of fibroblasts and inflammatory cells to the tear area, while VEGF stimulates angiogenesis.\textsuperscript{33}

**Methods of Augmentation**

Methods of augmentation come in many different forms but can be categorized by the type of cell delivered and the mechanism of delivery. Cell types include individual growth factors, stem cells, or a combination of both, such as in the use of platelet rich plasma. Examples of individual growth factors that can be used are as mentioned previously and include the BMP family, bFGF, PDGF, and TGF-β. Potential stem cell sources include bone marrow derived mesenchymal stem cells (BMSC), bone marrow cells (BMC), and muscle-derived stem cells (MDSC). A variety of techniques have been used to deliver and localize the cell types for augmentation. Vehicles range from in situ delivery, such as microfracture; direct injection, such as plasma rich platelets, or scaffold materials, which are biologic or synthetic (Fig. 1).\textsuperscript{34,35}

**Microfracture**

Snyder and Burns\textsuperscript{36} coined the term “crimson duvet” to describe the resulting clot obtained after microfracture of the greater tuberosity, just lateral to the site of rotator cuff repair. It is a cheap and technically easy method of delivery of growth factors to the repair site using a simple microfracture technique. A crimson colored clot is produced from the bone marrow vents punctured in the greater tuberosity. Perforations enter cancellous bone, and bone marrow then flows out to cover the denuded tuberosity and repaired rotator cuff tendon.\textsuperscript{36} This resultant “super clot” contains mesenchymal stem cells and platelets with growth factors and other vascular elements. The fibrin clot serves as a temporary scaffolding material, acts as a reservoir for growth factors, and a surface that guides cells by encouraging their migration and proliferation. This is important as the rotator cuff repair site is initially relatively avascular. At 3 months post-repair, a robust vascular response occurs at the suture anchor site in the greater tuberosity, providing blood supply to the tendon-bone interface.

Snyder and Burns\textsuperscript{36} also reported on a few case series in patients in which serial postoperative MRIs and second look arthroscopies were performed. In one patient who underwent serial MRI after rotator cuff repair, gradual reconstitution of the footprint was observed. Four patients had second look arthroscopies, which were indicated for various reasons, and two of these patients underwent biopsies of the microfracture site. A rich vascular blanket was observed covering the cuff edge at 2 weeks postoperatively. Fibrovascular tissue surrounding the repaired cuff and suture anchors was observed at 8 weeks. Biopsy performed at 5 weeks postoperatively showed a rich fibrin matrix infiltrated with healthy fibroblasts, lymphocytes, and abundant vascular elements. At 3 years, biopsy revealed a normal appearing cuff tendon attachment.

**Platelet-rich Plasma**

Platelet-rich plasma (PRP) has also been investigated as a method of delivering autogenous growth factors directly at the repair site in the form of a fibrin clot. Platelet-rich plasma contains many cellular components known to play a role in cuff healing, such as PDGF, VEGF, TGF, FGF, and

![Figure 1](image-url) Overview of biological augmentation vehicles for delivery.
IGF. However, recent randomized controlled trials failed to show that PRP augmentation improved outcomes after rotator cuff repair.37,38

Rodeo and coworkers37 looked at 79 patients undergoing arthroscopic rotator cuff repair and randomized them to receiving platelet-rich fibrin matrix (PRFM) at the tendon-bone interface or repair without PRFM. PRFM is a PRP variant that is a fibrin matrix formed by activation of a fibrin-clotting cascade initiated by adding calcium CaCl2 and a second centrifugation step. PRFM is made intraoperatively by using 9 mL of peripheral venous blood. All patients underwent a standard arthroscopic rotator cuff repair. A suture anchor was placed in the tuberosity and a suture passed through the PRFM implant and tendon and then tied, placing the PRFM between tendon and bone at the tendon-bone interface. Tendon healing was then evaluated at 6 and 12 weeks postoperatively using ultrasound. No difference in tendon-to-bone healing or ASES scores was found between both groups at all-time points. Complete tendon-to-bone healing (i.e., intact repair) was found in 67% in the PRFM group and 81% in the control group. In fact, the investigators found that their regression analysis suggested that PRFM may have a negative effect on healing but hypothesized that there may not be sufficient platelet concentration in PRFM and suggested that further studies were required to evaluate the role of PRFM in rotator cuff repair.

Castricini and associates38 also examined the use of PRP in augmentation of rotator cuff tears. They looked at 88 patients with small to medium sized rotator cuff tears and randomized to arthroscopic double-row repair with autologous PRFM augmentation or without PRFM. PRFM preparation was similar as was described previously. At 16-month follow-up, no significant difference was found in both Constant-Murley score and MRI tendon score, which was based on tendon thickness, coverage of greater tuberosity, and intensity of signal.

**Scaffolds**

There are a wide variety of products available as biologic scaffolds from different companies. The products can be classified into two broad categories: biologic or synthetic.34,39 Biological scaffolds can be further categorized based on cell type and source. Various cell types used include small intestine submucosa, dermis, pericardium, and fascia lata. Cell sources can be either human, (i.e., autograft or allograft) or animal with xenografts, such as porcine, bovine, or equine.

Basic principles are found in common between the different cell types and sources. They consist of a protein based extracellular matrix and are composed primarily of type I collagen fibers. Scaffolds are processed to minimize host response, which lessens degradation of ECM by host immune response, incorporates the scaffold into host tissue, and induces growth factors in the healing response. Advantages of biologic scaffolds over synthetic scaffolds are due to host cell integration, with a three dimensional protein microstructure and natural porosity. This allows a larger space for host cell attachment, proliferation and migration, and induces new tissue formation faster. Disadvantages include poorer mechanical properties, unclear degradation rate, and variations in biocompatibility.39 Biomechanical studies have shown a lower elastic moduli than intact tendon, which can lead to graft rupture under minimal stress.40 Biologic scaffolds have a non-specific induction ability, an undefined degradation rate, and vary in biocompatibility depending on the source tissue leading to inflammatory response and implant rejection. Scaffolds have the potential to be seeded with transduced fibroblasts with genes for specific growth factors to potentially provide more prolonged delivery.39

The GraftJacket® (Wright Medical Technology, Inc., Memphis, TN) is a biologic scaffold that has been extensively studied. GraftJacket® is an allograft constructed from tissue bank human skin.41 Epidermal and dermal cells are removed with processing, and this acellular freeze dried patch is available in 5 x 5 cm and 5 x 10 cm sizes with an average of 1.0 mm thickness. A thicker version called “maximum force” averages 1.5 mm thick and is available in 4 x 7 cm or 5 x 5 cm sheets. Finally, a 2.0 mm thick product is available named GraftJacket® extreme and comes in a 4 x 7 cm sheet size. The components of the GraftJacket® include collagen types I, II, IV, and VII, elastin, chondroitin sulfate, proteoglycans, and fibroblast growth factor. The product has an intact basement membrane, and vascular channels are preserved, which aids in host incorporation.

Barber and coworkers52 looked at GraftJacket® in a randomized controlled trial in patients with large rotator cuff tears measuring at least 3 cm in width with two tendon involvement. Patients were randomized to arthroscopic single-row repair with GraftJacket® augmentation or no augmentation. An onlay technique was used to augment the repair from the musculotendinous junction across the bone-tendon interface to cover the entire tuberosity laterally. Mean follow-up was 24 months, with MRI obtained on average 14.5 months after surgery. Cuff repairs with GraftJacket® augmentation had significantly higher ASES scores (98.9 versus 94.8) and Constant scores (91.9 versus 85.3), and on follow-up MRI, a high percentages of patients in the augmentation group had intact cuff, 85% versus 40%. No adverse events were reported related to the implanted matrix.

Another biologic scaffold that has been evaluated with less clinical success is the Restore Orthobiologic Implant. Restore is composed of porcine small intestine submucosa and was available in 63 mm diameter circular implant with 10 layers. It was composed of over 90% collagen, 5% to 10% lipids, and a small amount of carbohydrates, with bioactive molecules including bFGF, VEGF, and TGF-β1. Walton and associates61 looked at the results of 15 patients with large rotator cuff tears who had poor quality tendon augmented with Restore and compared them to matched
controls retrospectively. In the xenograft group, 4 patients had a severe postoperative inflammatory reaction requiring open debridement and at 2 years postoperatively had significantly less lift-off strength and internal rotation and adduction strength. The re-tear rate based on MRI at 2 years was 6 of 10 in the xenograft group and 7 of 12 in the control group. The investigators thus recommended against the further use of Restore Orthobiologic Implant.

Synthetic scaffolds are manufactured from chemical compounds and consist of polymers from a variety of materials, including polyester, polypropylene, polyacrylamide, dacron, carbon, silicone, and nylon. Strategies that have shown early success in animal studies and small clinical case series. Continuing development and testing of biologic augmentation therapy, and a better understanding of the healing cascade.

The advantage of synthetic scaffolds is that they are mechanically stronger, of consistent quality, and there is no risk of disease transmission with use. However, disadvantages are biocompatibility. There is a foreign body reaction which can lead to infection, decreased stability, synovitis, osteolysis, and osteoarthritides. Although the outcome data on these products is limited, there have been some positive results in animal studies.

17. Nevisier JS, Nevisier RJ, Nevisier TJ. The repair of chronic massive ruptures of the rotator cuff of the shoulder by use