WHY SUTURE-BASED ROTATOR CUFF REPAIRS OFTEN FAIL: MECHANICAL AND BIOLOGICAL FACTORS

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INTRODUCTION

Rotator cuff repairs continue to experience unacceptably high failure rates, with structural re-tear or non-healing reported in a substantial fraction of cases despite advances in surgical technique. These failures are often not due to inadequate initial repair strength – in fact, modern suture-anchor constructs are extremely strong – but rather due to the mechanical and biological consequences of how sutures interact with tendon tissue during healing.[1] High-strength sutures and tightly tied knots can inadvertently damage the very tissue they are meant to fix, leading to compromised biology and eventual mechanical failure. This white paper examines evidence from experimental, biomechanical, and clinical studies to elucidate why suture-based rotator cuff repairs fail, focusing on post-operative loading, stress concentrations and tissue necrosis, gap formation, the trade-off between fixation strength and biology, and the limitations of current suture constructs. Finally, we introduce the SINEFIX™ implant system as a novel approach aiming to improve outcomes by mitigating these failure mechanisms.

POSTOPERATIVE LOAD DYNAMICS AND SUTURE STRENGTH

Early postoperative rehabilitation of rotator cuff repairs (especially passive motion protocols) imposes relatively low loads on the repair construct. Studies have shown that gentle passive range-of-motion exercises generate forces on the order of only tens of Newtons, well below the failure strength of typical repairs. In practical terms, the initial loads across the repair during the early healing phase are low, and modern #2 high-strength sutures (e.g. FiberWire) have tensile strengths far exceeding these loads. For example, a standard suture-anchor repair can withstand on the order of 150–300 N of force before failing, whereas early passive motion might impart <50–60 N. Thus, initial failure by overload is uncommon—contemporary repairs generally provide sufficient mechanical strength for the controlled rehabilitation period. This underscores that something other than simple lack of strength is at play in many repair failures. The key issues lie in how the loads are transmitted to the tendon and how the tendon tissue responds.

STRESS CONCENTRATION AND TENDON NECROSIS AT SUTURE SITES

Suture-based fixation inherently concentrates force at a few discrete points where the sutures pass through the tendon or attach to bone. Finite element analyses and cadaveric studies demonstrate that peak stresses around suture anchors often exceed the physiological tolerance of tendon, leading to local fiber disruption and micro-tearing in the tendon matrix.[2] In other words, even if the overall load is within safe limits, the stress is not evenly distributed – a phenomenon sometimes called the “anchor effect” or stress riser effect. As a result, a stiff, high-tension suture can effectively act like a cheese wire, cutting through collagen fibers—the so-called *“cheese-wiring” effect*—which has been directly observed as localized collagen damage and cell death at the suture-tendon interface.

A well-documented biological consequence of these focal stress concentrations is the formation of an acellular zone in the tendon around the suture. In a landmark study, Wong *et al.*[3] showed that when a suture is passed through tendon under tension, a region completely devoid of living cells develops around the suture within minutes to hours. Remarkably, this necrotic, cell-free zone can persist

for long periods (up to at least one-year post-repair in their observations), indicating that the initial insult of the suture can cause irreversible local tissue loss. The extent of cell death correlates with the degree of suture tension: tighter sutures create larger zones of ischemia and necrosis in the surrounding tendon tissue.[3] This has been demonstrated in animal models as well—for instance, in a rabbit rotator cuff repair model, excessive initial suture tension led to obvious perisuture tendon necrosis within days of surgery. Essentially, an overly tight suture strangulates the tissue, cutting off blood supply and killing tendon cells in its immediate vicinity.

The creation of an acellular, non-viable region around suture material is highly detrimental to repair integrity. That segment of tendon cannot participate in healing or bear load. It becomes a structurally weak link—a focal point prone to failure. Even as overall tendon healing progresses around it, the necrotic zone represents a local deficit in material properties and biology. In summary, high suture-induced stress leads to localized tendon necrosis (via ischemia and direct tissue cutting), which undermines the repair from within. This mechanism helps explain why even technically "strong" repairs (in terms of suture material) may still fail at the suture-tendon interface.

GAP FORMATION AND MICROMOTION AT THE TENDON-BONE INTERFACE

One critical consequence of suture cutting and tissue necrosis is the formation of gaps at the repair interface. As the repaired tendon is cyclically loaded in the post-op period (even at low forces from passive motion), the damaged or dead tissue around the suture cannot effectively carry load.[3] The sutures begin to cut through the compromised tendon, or the tendon tissue adjacent to the sutures plastically stretches out. Over time, this process leads to a gradual separation (micromotion) between tendon and bone, manifesting as a small gap. Initially the gap may be microscopic, but with repeated loading it can widen. Once a detectable tendon-bone gap forms, the mechanical integrity of the repair plummets. The repair is no longer a snug tendon-to-bone apposition but rather a loosely approximated interface with interposed gap; as a result, load is no longer effectively transferred across the repair.

Even a relatively small gap is a major predictor of repair failure. Laboratory studies have shown that the appearance of even a few millimeters of gap dramatically weakens the repair's resistance to further loading, often precipitating rapid failure if loading continues. In essence, the repair loses its initial stiffness once a gap opens, leading to a vicious cycle: gap leads to stress concentration on the remaining intact fibers, which leads to further tissue failure and gap enlargement. Moreover, gap formation is biologically deleterious—if the tendon is not in contact with bone, the healing tissue must bridge the gap with scar tissue, which is biomechanically inferior. In vivo imaging studies and clinical observations support this: for example, repairs performed under high tension (which often corresponds to inherent gap or poor footprint contact) are associated with higher re-tear rates.[4] In a 2021 clinical study, patients whose tendons had to be repaired under substantial tension had a retear rate of ~29%, compared to only ~10% retear when tendons could be repaired with minimal tension (full footprint coverage).[5] This underscores that incomplete contact or gapping at the repair site correlates with failure. Thus, gap formation—often driven by suture cut-through and local tissue loss—is a central mechanical failure mode in rotator cuff repairs. Reducing micromotion and preventing gaps during the healing period is crucial for a successful outcome.

FIXATION STRENGTH VS. BIOLOGICAL PRESERVATION

Given the above, a paradigm emerges: more fixation strength is not always better, if achieved at the cost of biological health of the tendon. There is a threshold beyond which increasing the mechanical rigidity or load of the repair yields diminishing returns or even becomes counterproductive. The goal of any rotator cuff repair construct is to be *just strong enough* to withstand expected physiological loads while preserving the tissue's capacity to heal. Once a repair meets the baseline strength needed for early rehabilitation (which most modern repairs do), further increasing the number of sutures, anchors, or the tightness of sutures does not necessarily improve success—in fact, it may strangle the tendon and impair healing. Excessive suture material and overly tight constructs elevate the risk of stress concentration and necrosis, as described above. This can create a mechanically robust construct on day 0 that paradoxically fosters a biologically hostile environment, leading to later failure.

Evidence suggests that an optimal repair balances mechanics and biology. Sufficient initial strength is necessary—to maintain fixation during the healing period—but the quality of that fixation is paramount, meaning how the load is distributed and whether

the tendon's blood supply and cells are preserved. Ultimate healing (tendon reattachment) is a biological process; no matter how strong a repair is at time zero, it will fail eventually if the tendon cannot biologically integrate with bone. Therefore, approaches that achieve adequate fixation with minimal tissue damage are preferred over simply maximally stiff, high-tension constructs. Surgeons are advised to avoid over-tensioning the repair—*"strong yet gentle"* should be the guiding principle. In practical terms, that means using just enough sutures/anchors to stabilize the tendon without overly constricting it, and distributing forces to avoid any one point bearing too much load. The concept of biomechanical gentleness has emerged: a repair that is forgiving (compliant) enough to cushion the healing tendon from stress spikes, while still preventing gross displacement. Indeed, recent improvements like more elastic suture materials and devices that spread out fixation forces are driven by this recognition that biological preservation is as important as raw strength.

LIMITATIONS OF CONVENTIONAL SUTURE CONSTRUCTS (FIBERWIRE AND OTHERS)

Current rotator cuff repair techniques predominantly rely on suture anchors and advanced suture materials (e.g. non-absorbable polyblend sutures like FiberWire, FiberTape, etc.). These high-strength sutures have revolutionized our ability to secure tendons, as they can sustain loads far above those encountered in early rehab. However, their very strength and stiffness can be a double-edged sword. FiberWire, for instance, is a braided ultrahigh-molecular-weight polyethylene (UHMWPE) suture with minimal elongation. When tied tightly through tendon, it does not elongate or yield under load, meaning all motion is transferred as shear at the suture-tendon interface. This can produce very high local stresses. Experimental comparisons between FiberWire and newer "gentler" sutures illustrate this issue: Owens et al. (2019)[6] found that in cyclic loading of repaired cadaveric tendons, FiberWire cut through an average of ~3.7 mm of tendon tissue, whereas a slightly elastic high-strength suture (Dynacord) cut through only ~2.7 mm.7 This ~28% reduction in tendon damage with a more compliant suture was statistically significant ($p = 0.012$). Moreover, in that study 2 out of 7 FiberWire repairs experienced complete tendon cut-through (>5 mm, essentially total stitch pull-out), while none of the Dynacord repairs did.[6] In other words, the stiffer FiberWire was more prone to "cheese-wire" straight through the tendon, whereas the slight elasticity of Dynacord allowed it to maintain fixation without sawing through. This highlights a limitation of traditional suture: beyond a certain point, increasing suture tensile properties offers no benefit because tendon tissue is the weak link—the tendon will yield before the suture breaks.

Another limitation of conventional repairs is the focal load distribution. Single-row suture anchor repairs concentrate force at just a couple of points on the tendon, where each suture anchor grips a small area of tendon. This point-loading can create stress concentrations and risk pull-out at those points. Techniques have evolved to mitigate this; for example, double-row and suture-bridge repairs spread the fixation over a broader footprint on the tuberosity, compressing a larger area of tendon to bone. Biomechanical studies confirm that double-row constructs often exhibit higher initial fixation strength and less gap formation than single-row, precisely because the load is shared across more sutures and a wider area. However, even multi-row suture constructs still have multiple individual sutures piercing the tendon, so the issue of localized suture-tendon interface damage is reduced but not eliminated. The tendon underneath each suture loop can still experience high pressure. Similarly, the widespread adoption of *tape sutures* (wider flat braided materials) instead of round cords is an attempt to distribute pressure on the tendon over a wider surface area and reduce cut-through. These innovations—double-row fixation, suture tapes, more elastic sutures—improve the situation but do not fully solve the core problem: the need to pass materials through tendon inherently creates stress concentration and can impair local circulation.

It is telling that even with excellent surgical technique (e.g. anatomic footprint coverage, multiple anchors, strong sutures), clinical failure rates remain significant, especially for large tears or poor-quality tendons. This suggests that limitations of biology and mechanics at the tissue-suture interface are still at play. Researchers have even explored biologic augmentations, such as compliant adhesive layers or patches, to offload the sutures. For instance, inserting a thin adhesive film between tendon and bone to bond the two has shown the potential to distribute load across the entire footprint (mimicking a native enthesis) rather than through just a few sutures.[7] Finite element models predict that an optimally designed adhesive interlayer could increase repair strength up to ten-fold compared to suture-alone, and early prototype tests have indeed demonstrated substantially improved failure loads with

adhesive-augmented repairs.[7] While these approaches are still experimental, they underscore a critical point: how the load is transferred can be as important as how much load the construct can bear. Conventional suture repairs, even with strong materials, are fundamentally limited by stress focusing and the biological toll on the tendon.

A NOVEL APPROACH—THE SINEFIX™ IMPLANT SYSTEM

To address the shortcomings of suture-based fixation, new fixation concepts are being developed. One such innovation is the SINEFIX™ implant system. SINEFIX represents a completely different approach to rotator cuff repair: instead of threads cutting through tendon, it uses a small polyether-ether-ketone (PEEK) implant that effectively "staples" the tendon to bone over a broad area. In practice, the device consists of a base that sits on the tendon surface and small fixation prongs that are driven into bone, clamping the tendon down. By securing a wide swath of tendon tissue against the bone, SINEFIX design is aimed at eliminating the point-loading of sutures—there are no concentrated suture anchor points, and at distributing the pressure is evenly across the repair footprint.

Biomechanical evaluations have compared SINEFIX to a conventional double-row suture anchor repair (a medial row of anchors with a lateral row bridging configuration). The results are promising. Under cyclic loading meant to simulate early postoperative forces (for example, 10–62 N cyclic loads for several hundred cycles), repairs done with SINEFIX showed minimal gap formation, while standard double-row suture repairs had significantly larger displacement. Specifically, at 62 N of load, SINEFIX repairs exhibited on average only about 1.5 mm of gap, whereas the double-row suture repairs opened to around 3.2 mm of gap.[8] This more than 50% reduction in gapping (1.5 mm vs 3.2 mm, $p = 0.001$) indicates a much more secure tendon-bone contact during cyclic motion. Maintaining close contact with minimal micromotion is crucial for allowing the tendon to biologically integrate, so this finding is significant.

Importantly, this improved stability did not come at the expense of ultimate strength. In the same tests, the maximum pull-out force (ultimate failure load) of SINEFIX repairs was about 215 ± 55 N, which was on par with—in fact numerically higher than—the ~166 ± 15 N average failure load of the double-row suture repairs (though the difference was not statistically significant).[8] In other words, SINEFIX provided at least equivalent peak strength to traditional repairs while dramatically reducing gap formation. This balance of high strength and low gap formation suggests that SINEFIX may achieve a more favorable mechanical environment for healing. The failure mode for SINEFIX was via the implant pulling out from bone at high load (tendon tissue remained intact under the staple), whereas the suture repairs tended to fail by sutures cutting through tendon or anchors pulling out.

From a biological perspective, the broad, flat fixation provided by SINEFIX may preserve the tendon's microcirculation. There are no strangulating sutures passing through the tendon substance; instead, pressure is spread out and there are no acute pressure points. The SINEFIX concept explicitly prioritizes even pressure distribution to avoid focal ischemia. By maintaining a large area of gentle contact between tendon and bone, it aims to prevent the creation of any acellular, necrotic zones in the tendon. Notably, the design intent is to maintain blood flow in the tendon under the implant and allow cell survival, in stark contrast to the localized tissue death that a tight suture loop can cause. A recent description of the device after its regulatory approval highlighted that SINEFIX has the potential to create a flat and even contact of tendon and bone, distributing shear stress uniformly and not cause point pressure peaks, while also maintaining blood circulation in the tendon. In essence, SINEFIX is designed to provide a mechanical buttress for the repair without "choking" the tendon's biology.

It embodies the principle that maximizing the quality of the repair (even load sharing, tissue preservation) can be more impactful than simply maximizing the quantity of fixation.

CONCLUSION

Suture-based rotator cuff repairs fail not because we lack strong enough sutures or anchors, but often because of the *side effects* of those sutures on the tendon tissue over time. The postoperative mechanical environment of a repair involves low loads that modern constructs can handle, yet highly localized stresses at suture-tendon contact points can irreversibly damage the tendon's cells and collagen. This leads to the formation of dead zones in the tendon and progressive cheese-wiring cut-through, which in turn causes gap formation at the healing interface—a harbinger of eventual failure. Piling on more sutures or stiffer materials in pursuit of higher ultimate strength can backfire by worsening these stress concentrations and strangling the tendon's blood supply. Thus, an evidence-based perspective is that optimal rotator cuff repair requires a balance: enough mechanical stability to hold the tendon in place, but not so much local pressure that the tendon's biology is compromised.

Current best practices (such as using multiple anchors in a double-row configuration, wider sutures, and avoiding over-tensioning) seek to strike this balance, yet conventional repairs remain fundamentally limited by the need to puncture and tie down the tendon. Emerging solutions like the SINEFIX implant illustrate a new paradigm: fix the tendon without lacerating it. By distributing load broadly and preserving tissue viability, such approaches aim to maintain secure tendon-to-bone contact throughout healing—which is ultimately the key to a successful repair.[9] In summary, the failure of suture repairs is often a consequence of their very design: high-strength sutures concentrate force and can damage the tendon they hold. The future of rotator cuff repair will likely lie in technologies and techniques that provide sufficient strength in a more tendon-friendly manner, ensuring that biology and mechanics work together rather than at odds. For surgeons and researchers, the mandate is clear: *protect the tendon while repairing it*. Only by doing so can we reduce failure rates and improve long-term outcomes in rotator cuff surgery.

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