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CORR Insights®: Muscle Fibers are Injured at the Time of Acute and Chronic Rotator Cuff Repair

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Where Are We Now?

While rotator cuff repair has an excellent track record in terms of pain relief, recovery of strength after surgery is not as consistent [7]. Some patients have persistent symptoms [4]. With time, torn rotator cuff muscles atrophy and undergo fatty infiltration [5]. This process worsens with time and as the tendon retracts medially. Even a successful repair defined as successful tendon healing at best stabilizes these muscle changes and does not reverse them [2]. Despite our technological

and recent biologic advances, overall the rate of tendon retear is still high, and repairs of chronic tears are more likely to fail [9]. There is some evidence that lengthening of the tendon for repair could cause traction related muscle injury leading to muscle cell plasma membrane disruption and permanent injury [1]. Some surgeons prefer to perform a medialized repair when significant tension is noted while reducing a chronically retracted tendon with the goal of decreasing tension on the repaired tendon [3]. This may be a reason to consider intercalary grafting of tears that cannot be sufficiently lateralized without undue tension.

Where Do We Need To Go?

The authors of the current study should be commended on their work that sheds light on muscle cell injury that can occur with rotator cuff repairs of any chronicity, though they specifically studied chronic repairs. In their rat study model, they found not surprisingly that chronic rotator cuff tears resulted in a decrease in muscle mass of up to 35% and tendon length of 20%. This is consistent with other studies evaluating those properties of torn muscle-tendons units [6, 8]. Most interestingly they found that repairing both acute and chronic tears resulted in significant rates of muscle fiber cell damage along the distal aspect of the tendon. After chronic rotator cuff tendon repair, there was additional muscle cellular injury in the proximal portion of the muscle that was not present in the acute repairs. One question remaining from their study's findings is that the sham surgery group also had a 27% rate of muscle damage. This raises the question of whether the muscle-splitting surgical approach resulted in secondary inflammatory cytokine induced cellular injury in the

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rotator cuff musculature. Another open question might be how much of the muscle injury seen after repair is due to mechanical tension, as opposed to other biologic causes.

How Do We Get There?

What makes the current study interesting is not only that it provides us with answers at a cellular level for clinical findings many of us experience with our rotator cuff repair patients, however it also provides a stimulus for many future research studies. One study that can be perhaps performed with the same model would be to determine the time frame from which a torn tendon progresses from the acute to the chronic stage when more extensive muscle damage occurs with repair. Likely this is a continuum, and it would be clinically usefully to have a time-dependent graph demonstrating these muscle changes. Similar studies could also help elucidate what other tendon factors might affect post-repair muscle damage such as tear size and retraction as well as if certain tendons are more predisposed to muscle injury

than others. Also of interest, would be testing chemical compounds which may provide muscle cell membrane protection and membrane repair of damaged cells or even genetic up-regulation of endogenous membrane-sealing proteins. Lastly, human studies involving muscle biopsy from repairs both that were acute and chronic as well as those that failed to heal would add to the direct clinical applicability of the current study's findings.

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