

Stem cell therapy and targeted collagen reduction for tendon healing

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DESCRIPTION

Tendon injuries, ranging from acute ruptures to chronic tendinopathies, represent a significant burden in both sports medicine and occupational health. Historically, tendons have been notoriously difficult to treat due to their hypocellular nature and limited blood supply, which often results in the formation of disorganized scar tissue rather than functional regeneration. However, a new paradigm is emerging in orthopaedic research: the dual approach of Mesenchymal Stem Cell (MSC) therapy combined with targeted collagen reduction to optimize the healing environment.

The primary challenge in tendon healing is the "repair versus regeneration" conflict. Standard healing usually leads to fibrosis, where the body quickly fills a gap with inferior Type III collagen. Stem cell therapy aims to shift this balance toward true regeneration. Mesenchymal Stem Cells, often harvested from bone marrow or adipose tissue, serve as biological "factories. When injected into a site of tendinopathy, these cells do more than just differentiate into new tenocytes their most critical role is paracrine signalling-secreting growth factors and anti-inflammatory cytokines that modulate the local environment.

These MSCs release essential signals such as Transforming Growth Factor-beta (TGF- β) and Vascular Endothelial Growth Factor (VEGF). In a controlled environment, these factors encourage the production of, which is characterized by high tensile strength and a highly organized parallel alignment. By introducing a concentrated population of regenerative cells, surgeons can jumpstart the biological machinery required to rebuild a tendon that can actually withstand the mechanical loads of daily activity or high-performance sport.

While adding "good" cells is vital, addressing the "bad" environment is equally important. In chronic tendinopathy, the affected area is often congested with excessive, disorganized Type III collagen and a dense Extracellular Matrix (ECM) that inhibits cell migration. This is where the concept of targeted collagen reduction enters the surgical conversation. By utilizing specific enzymes, such as collagenase, or mechanical debridement,

clinicians can break down the pathological fibrotic tissue that characterizes "jumper's knee" or "tennis elbow."

Targeted reduction serves as a "biological clearing." By selectively removing the dysfunctional scar tissue, surgeons create a physical and chemical "niche" that is more receptive to stem cell integration. This process reduces the stiffness associated with fibrosis, allowing for better nutrient diffusion and physical space for new, healthy fibers to align. It effectively resets the wound-healing clock, transforming a stagnant, chronic lesion into an acute-like environment that is primed for the MSCs to begin their restorative work.

The true breakthrough lies in the combination of these two techniques. Targeted collagen reduction removes the physical barriers to healing, while stem cell therapy provides the biological impetus for growth. Without the reduction of old scar tissue, injected stem cells often struggle to survive or differentiate correctly in a hostile, fibrotic environment. Conversely, removing scar tissue without providing a regenerative stimulus often results in the body simply replacing the void with more scar tissue.

In this dual-action model, the biochemical environment is finely tuned. The reduction of dense ECM decreases the presence of inhibitory molecules, while the MSCs ensure that the subsequent healing phase produces a high ratio of Type I to Type III collagen. The result is a tendon with biomechanical properties-such as Young's modulus and ultimate tensile strength-that closely mimic the pre-injury state. This approach is particularly promising for rotator cuff repairs and Achilles tendon pathologies, where the failure rates of traditional "suture-only" repairs remain stubbornly high.

As we move through 2026, the focus is shifting toward bioactive scaffolds that can deliver these stem cells and collagen-modulating enzymes simultaneously. These scaffolds act as a temporary "house" for the cells, protecting them from mechanical shear forces while slowly releasing agents that prevent excessive scarring. This "precision orthopaedics" ensures that the treatment is not just reactive, but proactive in guiding the tissue toward a functional, rather than a fibrotic, outcome.

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CONCLUSION

In conclusion, the integration of stem cell therapy with targeted collagen reduction represents a sophisticated evolution in orthopaedic surgery. By addressing both the cellular deficiency

and the structural pathology of tendon injuries, this approach offers a pathway to faster recovery times, lower re-injury rates and a return to full function for patients who previously faced the limitations of permanent scarring.