

New Trends in Treating Muscle Injury

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Present-day scientists are studying the factors that aid and delay tissue repair, and especially how to prevent or diminish end-stage fibrosis. They realize nonsteroidal anti-inflammatory drugs (NSAIDs) and intramuscular corticosteroid injections that diminish the classic inflammatory symptoms of pain and swelling may be retarding tissue healing. Blocking the cyclooxygenase-2 pathway with these prostaglandin inhibitors [weakens the quality of muscle repair and diminishes normal function](#). These substances may [also be a factor in increasing fibrosis](#).^{1,2}

After muscle injury, there is an increase in a cytokine called transforming growth factor (TGF)-B1 that induces fibrosis. Fibrosis prevents patients from reaching their normal baseline of activity by preventing the formation of new axons toward myofibers, and [contributes to a decline in muscle contractility and range of motion](#).³ Gamma-interferon and decorin have proven to be antifibrotic, but more clinical studies are needed. There is currently research on the transplantation of stem cells for regeneration of muscle, although this has been limited to animal models.

One of the most exciting treatments for both muscle and tendon injuries has to do with the use of platelet-rich plasma (PRP). While not entirely proven, the early results for healing are very promising. Just imagine if after an acute injury to a tendon or muscle, your own blood is immediately used at the point of care, making rejection or an adverse reaction unlikely.

The possible theories as to how these platelets create healing are numerous. Some of the strong possibilities are that the PRP becomes activated by the collagen within connective tissue, releases growth factors and cytokines, stimulates local stem cells and enhances extracellular matrix gene expression. It inhibits excess inflammation, apoptosis and metalloproteinase activity. [The result is the regeneration of new tissue](#).⁴

It is fairly well-established that promoting angiogenesis (new blood vessel formation) is necessary for regeneration. New blood to the area activates muscle-derived stem cells that originate from the vascular endothelium, increasing more growth factors that activate dormant satellite cells. It is accepted that exercise causing muscular contraction induces the formation of new vessels and skeletal muscle perfusion. Another effect of exercise is the increase in the serum of matrix metalloproteinases, which digest fibrotic scar tissue, regulate the secretion of pro-regenerative growth factors and help to mobilize stem cells from the bone marrow.

While traditional treatment for muscle injury has always emphasized rest, ice, compression and elevation (RICE), it is possible that immediate rest and immobilization may retard regeneration and be responsible for scar tissue development. Rest may also jeopardize the recruitment of stem cells to the zone of injury in the earliest stages of regeneration. Studies are needed as to the timing and type of exercises that are safe to enhance regeneration as soon as possible.

Early mechanical load such as myofascial release, Graston Technique, ART and others might be considered local forms of exercise to increase blood flow. It appears that reducing excessive early inflammation may prevent dense scar-tissue formation. I always remember what [Dr. James Cyriax](#)

stated about treating a medial collateral sprain in a skier. He remarked that to prevent the formation of scar tissue, the ligament should receive friction massage while the patient is being carried down the hill on the stretcher.

References

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