

Function analysis

Load and activity balance optimizes tendon/ligament healing

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Tendons and ligaments perform primarily mechanical functions; tendons transmit forces from muscles to bones, ligaments prevent excessive motion between bones. To function effectively, tendons and ligaments must develop and maintain mechanical properties that enable them to withstand the mechanical loading to which they are exposed. They do so by adapting their structure, and consequently their mechanical properties, in response to mechanical loading. If a tendon or ligament fails to adapt sufficiently, or if adaptation to increased loading cannot keep pace with the creation of loading-induced damage, injury may occur. Rehabilitation of the weakened tendon or ligament following injury involves maintaining a delicate balance between providing mechanical stimuli to guide the healing process and protecting the tendon or ligament from loading that could cause further damage.

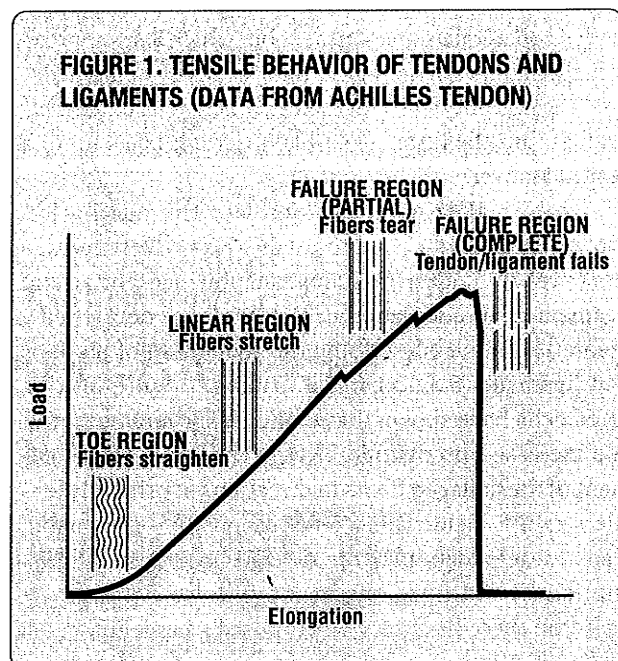
Basic biomechanics

Tendons and ligaments experience primarily tensile loading along their length. Since tendons form a direct connection between muscle and bone, they are loaded in nearly uniaxial tension when muscles contract. Ligaments are also loaded in tension, but the direction of loading varies as external loading creates multiple joint motions. Tendons and ligaments go through many cycles of loading and unloading during the course of a day. Each loading cycle generates stresses and strains that may create damage in the tendon or ligament while also serving as mechanical stimuli for adaptation.

Tendons and ligaments are composed of cells called fibroblasts embedded in an extracellular matrix that is produced, organized, and maintained by the cells. The mechanical behavior of tendons and ligaments derives primarily from type I collagen fibers, a matrix component that makes up 70% to 90% of the tissue's dry weight. The fibers generally align along the tendon's or ligament's axis, providing maximum stiffness and strength in the princi-

ple direction of tensile loading. The fibers have a "crimped" or wavy appearance when unloaded. As load is applied, the crimps straighten and the fibers rotate towards the direction of loading. This creates a "toe region" behavior (the standard term used to describe the initial region of the load versus the elongation curve) in which the tendon or ligament elongates under little force (Figure 1). Once the crimps have straightened, the fibers stretch with constant resistance creating a "linear region" behavior. Finally, fibers or groups of fibers begin to tear in a "failure region" behavior. As fibers fail, the load-bearing capacity of the tendon or ligament drops, and further stretching eventually results in complete failure of the structure.

The main microstructural determinants of tendon and ligament tensile behavior are collagen fiber content, fiber orientation, and fiber stiffness, which depends on collagen type, fiber size, and the density of cross-links between fibers.¹ Tendons tend to have a higher collagen content and

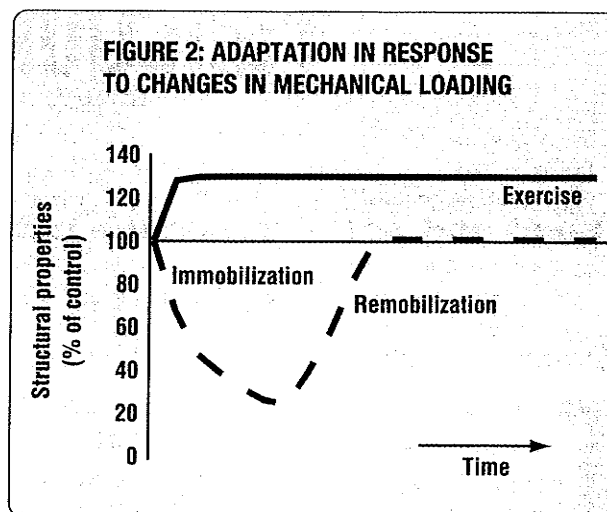


more uniform axial fiber alignment than ligaments. This results in a higher material stiffness and strength for tendons than for ligaments. Microstructure and mechanical properties also vary substantially among tendons and ligaments. The medial collateral ligament, for example, has a higher material stiffness and strength than the anterior cruciate ligament due to its higher collagen content, larger fibers, and better fiber alignment. These differences in composition and microstructure reflect differences in mechanical loading. Tendon and ligament cells are sensitive to their local loading environment, and they alter the extracellular matrix in response to changes in mechanical loading.

Adaptation to activity

Animal studies have shown that tendons and ligaments respond to exercise, immobilization, and remobilization by altering their microstructure and mechanical properties. Exercise can increase a tendon's or ligament's size, stiffness, and strength by improving fiber alignment and by increasing collagen content, fiber thickness, and cross-linking between fibers.² Immobilization, on the other hand, results in decreased tendon or ligament size, stiffness, and strength as mature collagen is replaced by new fibers that are small, poorly cross-linked, and randomly oriented.^{3,4} Remobilization appears to fully reverse the effects of immobilization, allowing a tendon or ligament to regain its normal structure and mechanical properties as the collagen network matures.⁴ Tendons and ligaments recover their properties at approximately the same rate at which they are lost during immobilization, but their attachments to bone take much longer to recover: The medial collateral ligament in a rabbit, which loses approximately half its strength after nine weeks of immobilization, regains normal strength after nine weeks of remobilization, but the ligament's tibial insertion takes up to a year to recover.⁴

Theoretical models can emulate the adaptation process and may be used to predict how different exercise or rehabilitation programs can influence tendon and ligament adaptation and healing. We have developed a theoretical model describing the adaptation of tendons and ligaments to mechanical loading.⁵ Studies of cultured cells have shown that cyclic tensile strains stimulate increased production of type I collagen and alignment of the collagen fibers in directions of principle tensile strain.^{6,7} Removal of loading, on the other hand, leads to degradation of the collagen network and disruption of the fiber alignment and cross-linking.⁸⁻¹⁰ Based on these findings, we proposed a model in which cyclic tensile strains are the primary mechanical stimu-

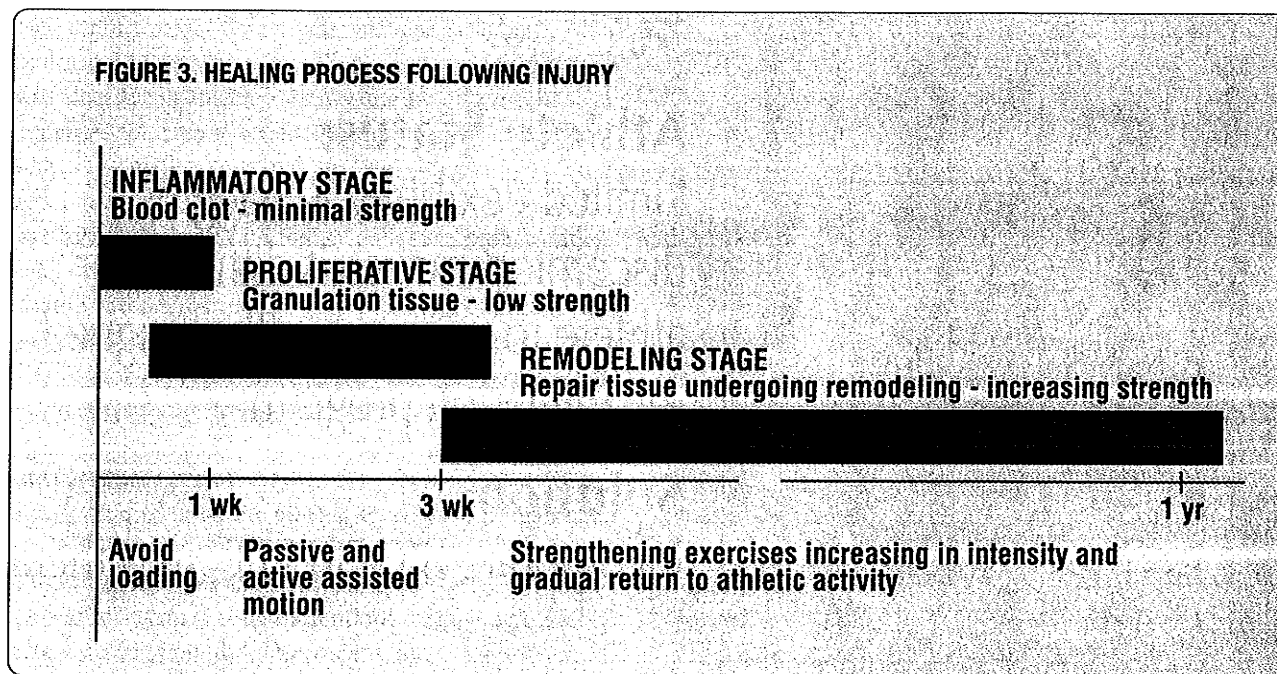


lus to which tendon and ligament cells respond. During normal activities, tendons and ligaments experience tensile strains in the 1.5% to 3% range.^{11,12} The properties of a tendon or ligament remain stable for strains in this physiologic range. When strains fall consistently below this range, the tendon or ligament loses stiffness and strength to raise the strains back into the physiologic range. For strains above this range, stiffness and strength increase to bring the strains back down into the physiologic range.

We have shown that simulations of exercise, immobilization, and remobilization using our model produce results in agreement with the results of experimental studies as described earlier (Figure 2).⁵ Exercise creates increased strains, which stimulate a moderate increase in tendon or ligament size, stiffness, and strength. Immobilization causes a drastic decrease in strains, leading to a rapid loss of size, stiffness, and strength. When loading is restored through remobilization, increased strains stimulate a rapid recovery of normal properties. Because our model can accurately reproduce the exercise, immobilization, and remobilization effects observed in experimental studies, we believe cyclic tensile strains are a likely mechanical stimulus guiding tendon and ligament adaptation to mechanical loading.

Although tendons and ligaments can clearly adapt to mechanical loading, it appears that there are sometimes limits to the extent to which a particular tendon or ligament can adapt. We have found that although the Achilles tendon has a material stiffness and strength similar to other tendons,¹³ it experiences much higher stresses than other tendons and must therefore experience significantly higher strains. These high strains likely contribute to the incidence of Achilles tendon injuries.¹⁴

FIGURE 3. HEALING PROCESS FOLLOWING INJURY



Mechanics of injury

While mechanical loading creates strains that may stimulate adaptation, mechanical loading can also create damage that leads to injury. A single high load (how high depends on which tendon/ligament—the strain, rather than the load, matters most) can produce strains in the failure region (>10%) resulting in sprains, partial tears, or even complete ruptures. Sprains and partial tears involve the disruption of collagen fibers without failure of the whole tendon or ligament. Sprains can occur in joints throughout the body and most frequently affect ligaments of the ankle.^{15,16} Partial tears occur clinically in many different tendons and ligaments. Ruptures often affect ligaments in the knee, where high strains are created by external loading. Tendon ruptures occur less frequently since most tendons are loaded by muscle contractions, which do not create strains high enough to rupture the tendon. However, tendons such as the patellar and Achilles tendons can rupture when muscle contraction occurs in conjunction with opposing external forces.

In addition to the traumatic overload injuries described above, tendons and ligaments can suffer injury through repetitive loading at lower strains. Repetitive loading injuries can affect both tendons and ligaments. Elbow and shoulder ligaments can develop overuse injuries from repetitive overhand motions, and the patellar tendon can develop tendinitis from repetitive jumping. Cyclic strains create microdamage, which is normally repaired by cells in and around the damaged tissue. Sudden increases in the strain magnitude or number of loading cycles, however,

can create damage faster than it can be repaired. The damage elicits attempts at repair, which often fail, causing pain, swelling, and degradation of the tendon's or ligament's mechanical properties. Reduced mechanical properties result in even higher strains if the level of activity is not reduced. A vicious cycle ensues in which damage accumulates and the structural properties of the tendon or ligament progressively degrade. When its mechanical properties degrade due to repetitive loading, the tendon or ligament becomes more susceptible to traumatic overload injuries. Rupture of the Achilles tendon, for instance, usually occurs only after the tendon has suffered some prior, often asymptomatic, degeneration.¹⁷

Healing response during rehabilitation

After a traumatic incident such as acute rupture or surgical repair, tendons and ligaments go through the same healing process as other soft tissues. This process has three overlapping stages: inflammation, proliferation, and remodeling (Figure 3). The inflammatory stage begins at the time of the traumatic event and lasts three to seven days. This stage is characterized by an infusion of blood, fluids, platelets, fibrin, cells, and other inflammatory products. A blood clot forms, tenuously connecting the broken ends of ruptured fibers. The proliferative stage begins as early as three days after the traumatic event and lasts two to three weeks. In this stage, fibroblasts migrate into the area, proliferate, and produce randomly oriented collagen fibrils. The collagen and other extracellular matrix components compose a vascular granulation tissue that replaces the ini-

tial blood clot. In the remodeling stage, which begins three to four weeks after the traumatic event and can continue for more than a year, cell density decreases and the production of matrix components slows. The collagen fibrils assemble into larger fibers with more extensive cross-linking and align in directions of tensile stress or strain. By the end of the remodeling phase, the tendon or ligament will ideally have reestablished normal structural and mechanical properties.

Mechanical loading is needed for tendons and ligaments to heal properly. Immobilization leads to decreased collagen content, smaller fibers, reduced cross-linking, and poor fiber alignment in the repaired tissue.¹⁴ Early active and passive motion, on the other hand, enhance fiber size and alignment, improving both the short- and long-term mechanical properties of healing tendons and ligaments.¹⁸ Early mobilization also helps to reduce the formation of adhesions.¹⁹ Mechanical loading guides the healing process by providing the same mechanical stimuli that guide tendon and ligament adaptation. These mechanical stimuli are especially critical during the proliferation and remodeling phases.

Although mechanical loading is essential for successful tendon and ligament healing, the healing tissue must also be protected from loading that might cause further injury. During the inflammatory stage, the blood clot stabilizing the injury site has almost no mechanical strength. The tendon or ligament must therefore be shielded from loading as much as possible during this stage. The randomly oriented collagen fibrils produced during the proliferative stage improve the tissue strength, but it remains far below that of normal tendons and ligaments. Passive motion and active assisted motion can be applied during this stage, but care must be taken to limit the range of motion. During the remodeling phase, the strength of the healing tissue progressively improves. Loading can therefore be increased gradually throughout this period. Eventually, as the healing tissue remodels, its strength returns to normal, and the patient is able to return to full activity.

The ideal healing process replaces the damaged tissue with normal tendinous or ligamentous tissue. Tendons and ligaments, however, often heal by scarring instead. While the scar remodels as described above, it is unclear

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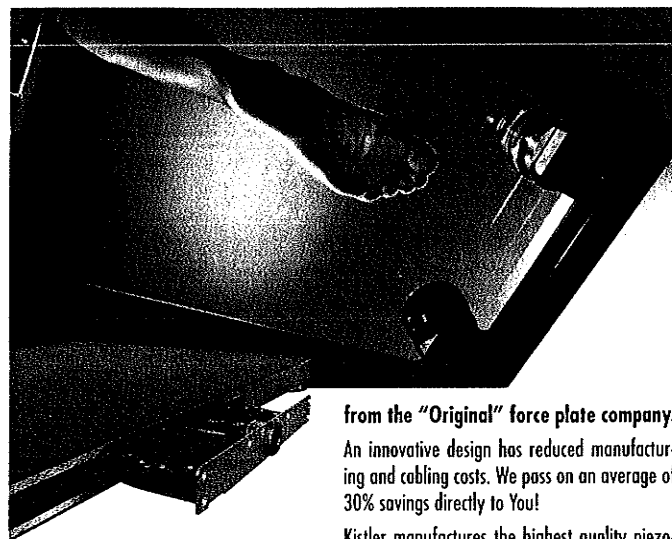
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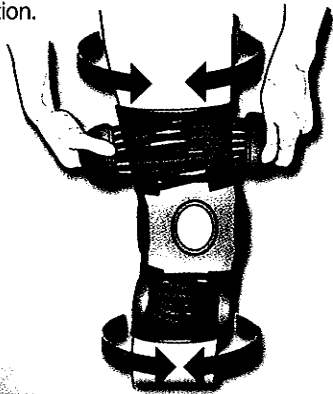


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whether it can attain the same strength as normal tissue. One goal of rehabilitation should therefore be to minimize scarring to allow the formation of more normal tissue. To accomplish this goal, the inflammatory process must be brought under control with rest, ice, and, possibly, anti-inflammatory medications, as well as protection of the injured tissue from loading that might cause further damage and overstimulate the repair response. To achieve optimal long-term results, therefore, overly aggressive rehabilitation programs should be avoided.

Avoidance of excessive loading is also critical in the rehabilitation of chronic overuse injuries. These injuries occur because the tendon or ligament has been unable to repair tissue damaged by repetitive microtrauma. The injured tissue usually exhibits degenerative changes including disruption of the collagen fiber network and focal scarring, indicating that attempts at repair have been made.²⁰ Only with adequate reductions in loading can subsequent repair attempts succeed.

Balancing act

Tendon and ligament rehabilitation involves maintaining a delicate balance between providing sufficient loading to guide the healing process and avoiding loading that might further damage the healing tissue. Loading should be minimal during the inflammatory phase of healing, which can last up to a week after acute injury or surgical repair. Once the inflammation is under control, early mobilization will enhance the healing process. Loading should initially be minimized through the use of passive or active assisted range-of-motion exercises. As the repair strengthens, loading should be gradually increased with closed chain strengthening exercises, eccentric loading, and return to athletic activity. The exact timing and specific exercises required for optimal healing will differ depending on which tendon or ligament is being rehabilitated. 

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References for this article are available at www.biomech.com.