THE ROLE OF MECHANOBIOLOGY IN THE PATHOGENESIS AND TREATMENT OF TENDINOPATHY

Steven P. Arnoczky, DVM, DACVS, DACVSMR
Laboratory for Comparative Orthopaedic Research
College of Veterinary Medicine, Michigan State University

While there is a significant amount of information available on the clinical presentation(s) and pathological changes associated with tendinopathy, the precise etiopathogenesis of this condition remains a topic of debate. Classically, the etiology of tendinopathy has been linked to the performance of repetitive activities (so-called overuse injuries). This has led many investigators to suggest that it is the mechanobiologic over-stimulation of tendon cells that is the initial stimulus for the degradative processes which have been shown to accompany tendinopathy. Although several studies have been able to demonstrate that the in vitro over-stimulation of tendon cells in monolayer can result in a moderate increase in the expression of some catabolic genes, the strain magnitudes and durations used in these in vitro studies, as well as the model systems, may not be clinically relevant. While different in vivo models of “over-use” injuries have been attempted, to date, no animal model has been shown to reproduce the full compliment of pathological changes associated with clinical cases of tendinopathy.

Using a rat tail tendon model, our lab has studied the in vitro mechanobiologic response of tendon cells in situ to various tensile loading regimes. From these studies we have forwarded the hypothesis that the etiopathogenic stimulus for the degenerative cascade which precedes the overt pathologic development of tendinopathy is the catabolic response of tendon cells to mechanobiologic under-stimulation as a result of microscopic damage to the collagen fibers of the tendon. Indeed, many of the catabolic changes seen in clinical cases of tendinopathy have been associated with the under-stimulation of tendon cells. However, to date, there is insufficient evidence to provide a direct link between the mechanical loading conditions of the tendon experienced during repetitive loading and the pathological response (e.g., not everyone involved in repetitive jumping sports will suffer from patellar tendinopathy).

Recently, we have expanded our studies to examine the measured and theoretical loads and strains experienced by the human patellar tendon during jumping activities. The results of these studies suggest that under certain conditions (joint position, tendon strain, etc) experienced during a jump-landing, the area of the patellar tendon commonly associated with patellar tendinopathy can be exposed to localized tissue strains sufficient enough to induce fibril damage. This, in turn, could lead to the mechanobiological understimulation of the tendon cells associated with these damaged fibrils and the initiation of a catabolic cascade. In addition, pilot studies have documented that decelerations reaching 10-15 g’s can occasionally occur in jump landings (in the vertical and/or horizontal planes) during routine volleyball maneuvers. When the resulting patellar tendon forces are applied rapidly and at a specific joint position, such loads could be sufficient to induce the aforementioned microdamage in the “at risk” area of the tendon.

Therefore, it is our theory that during repetitive activities one (or more) abnormal (overloading) cycles may occur during which individual collagen fibers can be damaged while the overall biomechanical status of the tendon is not clinically compromised. The outcome of this overloading cycle produces isolated tendon fibril(s) failure and a subsequent altered cell-matrix interaction leading to the mechanobiological understimulation of tendon cells in the damaged area. This, in turn, leads to the initiation of a catabolic cascade that decreases the
safety margin of the tendon making is more susceptible to additional damage at lower strains. We theorize that the association of increased frequency of training with the onset of tendinopathy could be related to the “probability” of experiencing an overload event as well as the impact of repetitive loading on the compromised tissue. Additional studies are being planned to further test this hypothesis.

References: