

Tensegrity to Tendinosis

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ABSTRACT

Biotensegrity or hierarchical tensegrity can explain how prestressed structures function in animals to transfer the stresses created by gravity, movement, digestion and emotional factors to the extracellular matrix (ECM). Tensegrity's connection to the ECM is explored, and the mechanotransduction of signals that prompt cellular changes in entheses, ligaments and tendons is discussed. The relationship between stress to the ECM and its effects on development of tendinosis at the cellular level is introduced. Recent findings demonstrating that tendinosis begins long before the patient experiences pain are examined, and the importance of balance between stress and rest in recovery is explored.

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KEYWORDS: biotensegrity, collagen, ECM, extracellular matrix, Prolotherapy, tendinosis, tensegrity.

TENSEGRITY

The word tensegrity can be used to describe many ideas employed in architectural design, bicycle wheels, spider webs and toys and even Carlos Castaneda's "warrior's path with heart." In architecture and animals tensegrity is associated with a type of structure in which the integrity is based on a balance between tension and compression components. Buckminster Fuller in 1948 coined the term tensegrity after he saw a sculpture created by Kenneth Snelson for Black Mountain College. (Wikipedia)

Steve Levin, MD, first presented it to the orthopedic medical community at a meeting of the North American Academy of Manipulative Medicine in 1980. It often is illustrated using Snelson sculptures (*See Figure 1.*) or architectural models such as the Wright flyer. (*See Figure 2.*) These models have some distinct relationships:

- Loading members only in pure compression or pure tension, which means the structures will fail only if the cables yield or the rods buckle.
- The structure has mechanical stability, which means the members remain in tension/compression as the stress on the structure increases.
- The cables are prestressed, which means the cables are rigid in tension. Tensional forces naturally transmit themselves over the shortest distance between two points. This makes them precisely positioned to withstand stress.
- These features of tensegrity mean that no structural member experiences bending.

Donald Ingber, MD, a Harvard researcher in the biological sciences working at about the same time, wrote about the concept of mechanotransduction or biotensegrity in 1993.¹ The idea of using *hierarchical tensegrity structures* as a means of explaining how the animal model of tensegrity works was in the biochemical and biophysical literature, but a recent article by Dr. Ingber discusses many biological

INTRODUCTION

I first started my "case for Prolotherapy" wanting to further examine several areas of interest to me, such as the method by which fibroblasts morph into myofibroblasts, new developments in understanding tendinosis, and the differences between ligaments and tendons. In the five to seven years since I looked into these areas for the book I co-authored, *Principles of Prolotherapy*, I have read numerous articles and research papers that have helped me in treating my patients and I wanted to share this information. As my research progressed in each of these areas of musculoskeletal medicine a common thread emerged—tensegrity. Tensegrity explains how altering the extracellular matrix (ECM) with a needle leads to wound healing, why excessive tendon stress leads to tendinosis and how the mechanochemistry of tendons and ligaments defines their function. It also explains why fibroblasts change to myofibroblasts and shorten ligaments and how tenocytes repair overuse injuries in tendons.

This paper will superficially explore some of the major new research about tensegrity as it applies to the ECM, the entheses, ligaments, tendons and tendinosis.



Figure 1. This is an example of sculpture tensegrity by Kenneth Snelson at the First Bank Building in downtown Denver. Ref: Ravin T. AAMM. 2003

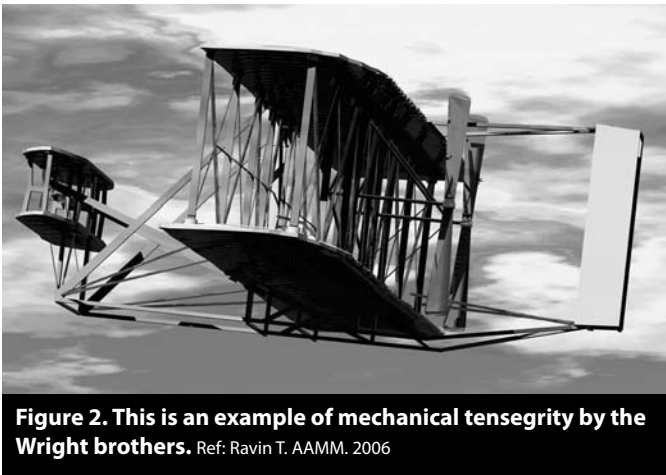


Figure 2. This is an example of mechanical tensegrity by the Wright brothers. Ref: Ravin T. AAMM. 2006

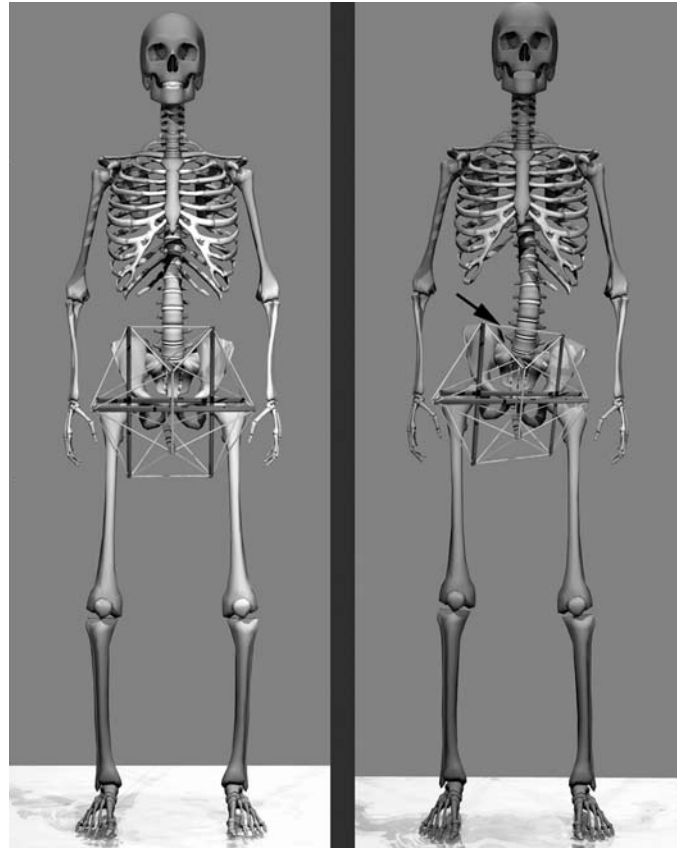


Figure 3. This is one example of biotensegrity by Tom Ravin, MD. Ref: Ravin T. AAMM. 2004

applications of this idea.² Knowledge gleaned from study of hierarchical tensegrity structures explains how large land animals can be mechanically strong, flexible and lightweight and yet respond by changing the shape of the body to accommodate specific tasks, such as altering the bony architecture because of gravity or guiding the bony growth from fetus to adulthood. Tensegrity enables the immediate response to changes in force by altering the stiffness in direct proportion to the applied mechanical stress.³

Hierarchical tensegrity structures have all the features of architectural models but also have prestressed elements of different size scales.² (See Figure 3.) One element can be as large as the bones, ligaments, muscles and tendons of the legs and within it can be an element that might

include a single muscle bundle and its tendon attachment to the bone. (See Figure 4.) Smaller still is a collagen fiber of the tendon attaching to the cell wall of a fibroblast in the ECM.⁴ Hierarchical tensegrity at the cellular level affects cell wall function that alters the cytoplasm, including the actin and α -actinin that give the cell its shape and mobility. The tensegrity continues even further to the intracellular proteins that change shape and function depending on stresses to the cytoskeleton and this explains how our tissues respond to growth, work, play and injuries. The whole cell itself is the final sensor because it integrates multiple local signals with other environmental inputs before reacting to the stress. (See Figure 5.)

EXTRACELLULAR MATRIX

The ECM is the principal extracellular component of all tissues and organs. Its main components—collagen, elastin, proteoglycan, fibronectin and laminin—allow it to play a pivotal role in hierarchical tensegrity. As a structural material, it controls the spatial organization in the tissue, from nanometer, micrometer, millimeter, and centimeter

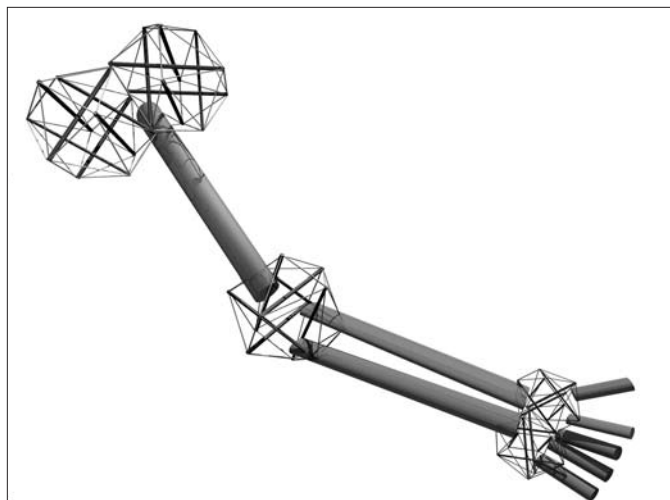


Figure 4. This example of biotensegrity by Tom Ravin M.D. conveys the idea of progressively smaller units that extend to the smallest joints. There are prestressed elements beyond the joints down to the extracellular matrix and into the cell

Walls. Ref: Ravin T. AAMM. 2004

to meter length scales. It is the connection between the nanometer features and the larger-scale organization that controls the motility and positions of cells, their geometry and mechanical connectivity. This ability to alter the composition and organization lends itself to a wide range of forms and functions ranging from solid in bone to pliable in tendons and cartilage.⁵ The major components of the ECM are incredibly stable and over time may develop covalent bonds in response to stress that can change their functional properties. An illustrative example is provided by collagen, the most abundant molecule in the ECM. The half-life for collagen before turnover through degradation by the matrix metalloproteinases is 2–4 years in bone, 10–15 years in skin and ~100 years in tendon.⁶

Strains of the ECM of only a few percent translate into very small alterations in the cytoskeleton and these tiny mechanical stimuli can be transduced into chemical and electrical signals, causing a number of cellular responses.³ The most common are the chemical ones, such as stretch-sensitive ion channels and G-protein coupled receptors. Despite the prevalence of these receptors, only a relatively small set, the integrins and cadherins, appears to be capable of responding to mechanical cues. (See Figure 5.) Integrins have elements that connect to the fibronectin in the ECM and molecular components that transverse the cell walls and attach to the intracellular focal adhesion complexes.⁷ These dynamic protein complexes consisting

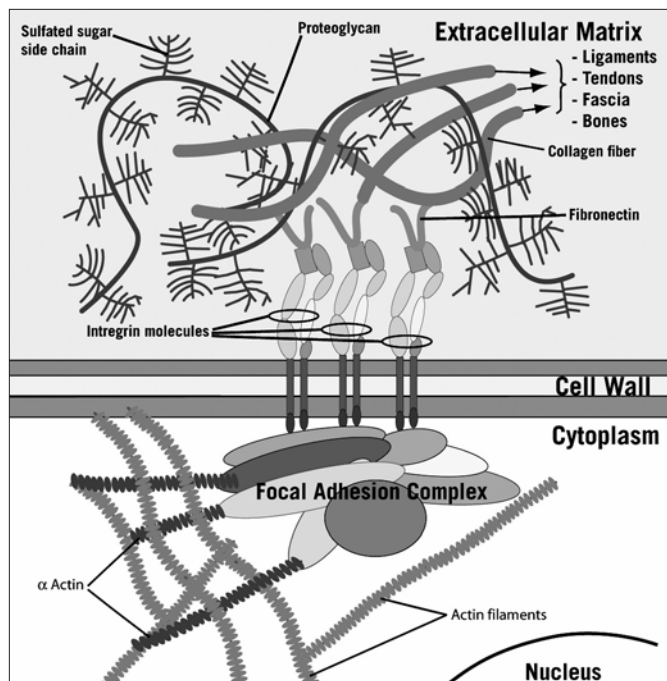


Figure 5. The smallest collagen fibers of the ligaments, tendons, fascia and bones in the ECM attach to the proteoglycans and to fibronectin. Proteoglycans are long sugar molecules (10,000 to 25,000 units) combined with long protein molecules and have many sulfated sugars as side chains. These molecules are rigid and resist compression and the sulfated sugars allow the ECM to change the turgor. There are two molecules that connect the ECM with the cytoplasm of the cell—these are the integrin molecules and the focal adhesion complexes. The integrin molecule spans the cell wall and attaches to the focal adhesion complex in the cytoplasm. The α -actin molecule, which has some contractile elements, attaches to the focal adhesion complex and the actin filaments. The actin filaments then transmit the forces throughout the cell to the nucleus and to other signaling organelles. This arrangement allows the biotensegrity of whole body to almost instantly transmit information from the outside world to the inside of a cell and *visa versa*. Ref: Ravin T. AAMM. 2011

of multiple integrins linked to a focal adhesion complex, as an integrated unit, provide the mechanical link between the cytoskeleton and the ECM.⁵ This bridging between cellular components and the ECM enables the this complex to serve as the conduit through which signal mechanotransduction occurs in response to physical force. The extent and degree of the stress can alter the configuration of the focal adhesion complex such that in one situation it might cause unraveling of a protein molecule and reveal a hidden binding site or in another alter the nuclear membrane. These simple and other more complex reactions enable the focal adhesion complex to act as a strain gauge and allow for varying degrees of response.

In another situation the strain might stabilize the integrin-ECM connection and provide a way to concentrate intracellular stresses on some molecules while shielding most of the other cellular components.⁵ This gives the ECM and focal adhesion complex the ability to organize and modify their structures instantaneously at the cellular level and still respond to long-term low-level stresses that create physiological changes, such as alterations in the shape of a bone, at the tissue level. All of these features cause the ECM to also be a source of “tissue memory” by binding, integrating and controlling the presentation of growth factors and other ligands to cell wall receptor sites. This allows the ECM to act in some ways like DNA.^{8,9}

Entheses, Ligaments and Tendons

ENTHESES

Both ligaments and tendons share the enthesis organ’s biology and biomechanics.¹⁰ The entheses also include structures adjacent to the entheses themselves to help reduce stress concentration at the attachments sites. The ligaments and tendons (LTs) are similar in the way they link the soft to the solid structures. The entheses provide strong and stable anchorage for these structures but also protect them from damage and injury by aiding in the smooth transfer of force between the soft and hard tissue. The gross structure of the enthesis reveals a flaring in order to increase the surface area of the attachment. Individual entheses also combine to form a larger and stronger attachment site called a *conjoined enthesis*, just as the sartorius, gracilis and semitendinosus combine on the tibia at the *pes anserinus*.¹¹ In the last ten years there has been an increasing interest in the concept of “*conjoined LTs enthesis*,” or where the tendons and ligaments blend together as they attach to bone. This is demonstrated at the lateral epicondyle where the common extensor tendon merges imperceptibly with the lateral collateral ligament and the annular ligament. Another example is the conjoined LT enthesis in the shoulder where the distal glenohumeral ligaments meld with the rotator cuff tendons on the humerus. The plantar aspects of the foot have multiple layers of conjoined tendons and LT combinations, such as where the long and short plantar ligaments, tibialis posterior and the peroneus longus share entheses to all the tarsal bones except the talus.¹²

Benjamin, Shaw and others have studied the enthesis in detail and have found that it is more than just the LT attachment site. It includes the bony prominences such as the superior tuberosity adjacent to the Achilles tendon that acts like a pulley, reducing stress on the tendon in dorsiflexion. The Achilles tendon also has Kager’s fat pad, the tip of which presses into the retrocalcaneal bursa. These particular structures are adapted to resist compression or shear when the foot is dorsiflexed and reduce friction and the build up of heat. LTs often attach to bone near tuberosities or are sunken into pits, which also act like pulleys to dissipate stress away from the attachment site itself. It is interesting that the Achilles tendon fat pad is the only part of the normal Achilles tendon enthesis organ that is innervated. This makes it the probable source of pain from the normal tendon.¹³

LTs are designed to bear and transmit high tensile loads along their longitudinal axes so they have structural characteristics that confer a greater stiffness and resistance in the axial dimension. The highly nonlinear response of the LT tissues is due to the hierarchical tensegrity structure of the collagen network. The structural integrity and the viscoelastic characteristics of LTs result mainly from the interaction between collagenous proteins and non-collagenous proteins-proteoglycans in the ECM. This interaction allows both reversible (slip-links) and irreversible (rupture) detachment of the glycosaminoglycans (GAG) from the proteins.¹⁴ There also are other cellular and molecular tensile stress absorbers that are discussed in the tendon and tendinosis sections.

LIGAMENTS

Recent ligament research supports the idea that ligaments have two functions and anatomies. One function is that they are the static stabilizers of joints and the second function is sensory or proprioceptive. The presence of mechanosensors implies a sensory role for some ligaments and that this afferent information could regulate the stiffness of the muscles surrounding the joint and improve its stability.¹⁵

The anatomic vision of ligament anatomy—that ligaments are static structures that fail by fracturing like pieces of soft iron (as illustrated in Strollers’ book) and that they are avascular—is being replaced as their microanatomy is unraveled.¹⁶ Immunohistologic chemistry and newer microimaging techniques available in the last fifteen to

twenty years are showing them to be dynamic structures closely connected to the tensegrity transduction system and the ECM.

It now is clear that there are two types of ligaments: those that consist of densely packed collagen fibres and fascicles and those with the fascicles surrounded by an area of loose connective tissue called the epifascicular region. The ratio of epifascicular to fascicular regions varies greatly in ligaments and when individual ligaments are subjected to scrutiny it can be observed that some ligaments have a lot of epifascicular space and others very little. The ligaments that are predominantly densely packed collagen fibers have only a minimal epifascicular region and almost no innervation. In the other type of ligaments the fascicular bundles and the epifascicular spaces are nearly equal in size. The ligaments with more epifascicular space contain the innervation and blood supplies. The majority of fascicles at the entheses have a high percentage of epifascicular space.^{17, 18}

The ligaments with a high percentage of fascicles are the static stabilizers or, in tensegrity terms, the tension elements. The ligaments with a high percentage of epifascicular space are involved in gathering afferent information such as tension, position and speed of movement. The sensory elements in the ligament are similar to those in the skin. A brief review of these sensors is helpful in understanding how ligaments function:

- Free nerve endings are pain fibers and are mostly in the epifascicular region with just a few in the fascicles.
- The Ruffini corpuscles function as pressure sensors and low vibration sensors and have a low threshold in relation to pressure. They are slow adapting and respond to static conditions of position and stretch.
- The Pacini corpuscles sense pressure and higher vibrations. They sense dynamic changes such as changes in velocity or acceleration and deceleration.
- Golgi apparatuses sense tension. These are slow adaptors and give information about passive stretch and active contraction and they inhibit muscle contraction.

In the wrist there are many ligaments and these have been studied extensively in research correlating their anatomy with their function. In particular the epifascicular anatomy of wrist ligaments was correlated with their known functions. The dorsal ligaments of the wrist have densely

placed collagen fibers and limited innervation and function mainly to constrain the scapholunate relative motion. The volar ligaments, which work together to support the wrist throughout its entire range of wrist motion, have a high percentage of epifascicular space and are among the most innervated ligaments in the wrist.^{18, 19}

The wrist ligaments have been the subjects of the most extensive studies comparing form to function, but similar research currently exists for the posterior cruciate ligament (PCL).²⁰ In the anterior cruciate ligament (ACL) almost 1% of the total area is nerve.²¹ These studies also have demonstrated the presence of blood vessels throughout the ligaments, particularly in the epifascicular space. So the idea that ligaments have limited blood supplies only applies to the densest regions of the fascicular portions of the ligaments. Immediately following injury the blood supply is derived from the epifascicular and epiligamentous tissues. The injured, frayed or disrupted area is pink or even red from the blood clots and increased blood supply. As the wound heals and the energy demands of the wound decrease, the hypervascular region disappears.²²

TENDONS

The basic functions of tendons are simple enough. They just connect muscles to bones and transmit forces so that they create movement. The musculoskeletal physician, however, needs to know more about how these complex and elegant structures actually go about these tasks. It seems that at almost every level of this tensegrity structure something important to know has been learned in the last two to eight years. Understanding more about tendons in general might be helpful in making decisions about how best to treat tendinosis.

A quick review of tendon anatomy will help reveal where the new research and some old ideas can aid in our understanding of the structure and function of these complex entities. Tendons have many hierarchical tensegrity scales beginning with individual collagen fibers, which attach to the cell walls of tenocytes and tenoblasts by way of the focal adhesion complex. In the ECM three collagen molecules arrange in helices that are held together by hydrogen bonds in a coiled shape and are called tropocollagen. Five tropocollagen molecules constitute a microfibril and multiple microfibrils aggregate and form fibrils. The fibril is defined as a collection of fibers that is surrounded by an endotendon.¹⁰ Fibrils are grouped into fibres; collections of fibres form bundles and bundles

form fascicles. The endotendon is mostly ECM with most of the tenocytes (TC), tenoblasts (TB), blood vessels and nerves. Some tendons have little or no endotendon and are thought to be involved in transferring stress.¹⁸ There is a helical organization of the whole tendon, from the shiny white tendon to the collagen molecules. This allows the collagen molecules to behave like cables or ropes that deform under tensile stress and improve the handling of their loads.²³ These loads are defined as tensile stress, which is a measure of the internal forces acting within a deformable body. (Wikipedia)

Many of the tendons that are of particular interest to a musculoskeletal physician have to do with joints used in throwing and walking. These muscles do not transmit their load just to bone but also to adjacent muscles and “non-muscular tissue” or the fascia. Wood Jones first explored this idea in the 1940s. A good example of this is where the gluteus maximus attaches to the tendon of the tensor fascia lata and the iliotibial band and not to the greater trochanter.²⁴ This anatomic arrangement links muscles together to form “mechanical chains” and most muscles in the legs have some direct attachment to the fascia.²⁵ These muscle and tissue arrangements allow the muscles to distribute tensile stress to the fascicles, then to the fibrils and eventually to the microfibrils and the tropocollagen and finally to the collagen molecules in the ECM that attach to the integrin complex on the cell wall.

Another feature of tendons is the ability of the fascicles to slide independently, which allows them to transmit tensile stress while changing direction, such as going around the medial malleolus. This feature also allows them to change shape as the muscle contracts and also creates a space for the blood vessels.²⁶ The fibrils also can slide relative to adjacent fibrils and this may account for up to 50% of a tendon’s ability to absorb tensile strain.²⁷

As tensile stress is further distributed down the tensegrity scale another feature of tendons comes into play—the crimp in the collagen. At rest, LTs’ collagen fibrils are in a wavy or crimped configuration. The crimp acts like a shock absorber or a buffer, permitting slight elongation to occur without fibrous damage. The crimp performs like a shock absorber that stores the stretch energy and when the stress is released the elastic recoil returns up to 90% of this stored energy.²⁸ As the tendon is stretched, the crimps begin to disappear progressively or individually rather than simultaneously from the ends toward the

centre of the collagen fascicle. The collagen crimp allows for a graded response to acute stretching and works with increasing stress up to about 4% of the LTs’ length.²⁹

As the length of the tendon exceeds 4% the tensile stress shifts to the collagen fibril, which is the primary force-transmitting unit of the tendon.³⁰ The stress at this level leads to some slippage of the ECM’s protein and GAG molecules relative to each other as their crosslinks break.³¹ This level of tensile stress also causes the tenocytes and fibroblasts that are in the fibrils and inter-fascicular spaces to be deformed. The collagen molecules of the ECM that attach to the integrin molecules on the cell surface sense the collagen stress and signal the cells to produce more collagen and ECM. This induces a two- or three-fold increase in collagen formation that peaks around 24 hours after exercise and remains elevated for up to 80 hours. The degradation of collagen proteins also increases in response to exercise and is a physiological response to healing. This illustrates that to increase tendon collagen some period of rest (from 36 to 72 hours) is required. This time frame may be compressed with training and conditioning. In any case, without sufficient rest a continuous loss of collagen is likely to occur.³² (See Figure 6.)

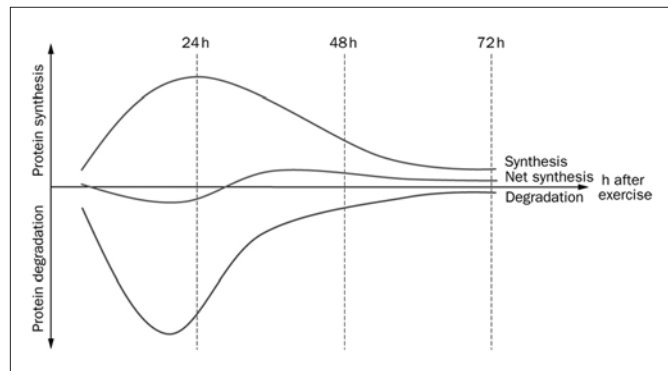


Figure 6. Acute exercise in humans is followed by an increase in both the synthesis and degradation of collagen in tendons. Notice that there is more degeneration than synthesis until 36-72 hours after the stress. If the rest periods are too short degradation will prevail and lead to tendinosis.

Ref: Magnusson PS. The pathogenesis of tendinopathy; balancing the response to loading. *Nature Reviews Rheumatology*. 2010.

The tenocytes also have mechanisms for continuing to dissipate the stress on the tendons. Tenocytes are like myofibroblasts and respond to rapid length increases with a rapid force increase associated with elastic resistance. This is followed by rapid loss of tissue tension associated with both viscoelastic relaxation and actin cytoskeleton disruption. The recovery of the tension is biphasic,

described as rapid active response (RAR) and gradual active response (GAR), restoring the tissue tension and rebuilding the actin cytoskeleton. The RAR occurs within seconds of a sudden stretch and is completed in less than a minute, and GAR begins a few minutes post-stretch and lasts more than 20 minutes. Both of these responses are mediated by Ca^{++} channels and reflect the distortion of the cell walls. Both result in cytoskeletal structural changes that include increased actin molecules in the direction of the force and are proportional to the applied stress.³³

Cytoskeletal disruption may shield cell-cell adhesions, cell-matrix adhesions and internal cellular components connected to the actin cytoskeleton from large stresses. This suggests that the structure, mechanics and biochemistry of myofibroblasts combine in an intricate choreography to enable stress release for protection of internal structures followed by rapid tissue tension recovery as the cell rebuilds and remodels its cytoskeleton.^{34, 35}

TENDINOSIS

Painful tendon “tendinosis” was considered not too long ago to be an inflammation that could be treated with rest, stretching, NSAIDs or cortisone. In the last five years, however, there has been an explosion of research and ideas on the nature and treatment of tendinosis. The observation today is that tendinosis is present in 35% of adults over the age of 35 years and that 50% of the shoulders of 65-year-olds have evidence of tendinosis even though they are asymptomatic.³⁶

The factors that lead to tendinosis can be divided into two major groups: intrinsic and extrinsic. The intrinsic ones seem obvious but often are overlooked. These issues may be major causes of asymptomatic tendinosis that is present in the older population.³⁷ These include:

- Genetic variation sequence of amino acids in Type I collagen as well as the ratios of Type I to other types of collagen.³⁸
- Endocrine issues such as estrogen in younger women and older men.³⁹
- Metabolic issues such as obesity, diabetes mellitus, lipid disorders and hypertension that alter microvascularity.⁴⁰

The extrinsic factors are also familiar to most of us in the musculoskeletal medicine community. These include:

- Using tendons too much, too hard and for too long, all at one time.
- Training errors that may be the result of a bad habit, which for many individuals can be hard to identify. In the adult athlete who has been doing a sport for many years these bad habits often are the result of compensation for another old injury. To find these training errors coaching and video often are the best answers.
- Training in either very hot or cold conditions. There is developing evidence that heat injury to tendons is a real problem. The heat generation is the result of friction in the tendinous structures and increased metabolic activity. The heat inside the tendon can easily achieve a temperature of greater than 42.5°C with continuous hard exercise. It is above this temperature that the tendon proteins begin to denature.^{37, 41}
- Training or competing while on fluoroquinolone drugs and possibly on statins in the older populations.⁴²
- The understanding of the pain in tendons has undergone a major shift in the last ten years. The working theory that the pain is the result of inflammation has shifted to a new theory that it is the result of degeneration. Research to explain the nature of the chronic arthroidides that involve the entheses resulted in the paradigm shift. The nature of tendon pain and dysfunction was evaluated anatomically and pathologically and it was clear that normal tendons under stress showed evidence of restructuring. It also was evident that healthy exercise led to both synthesis and degradation of collagen but synthesis prevailed. *Figure 6* shows the overall picture and illustrates the fine line between progressive growth and degeneration. The balance between synthesis and degradation is taking place at the cellular level and if the balance is tipped to degeneration by any of the factors discussed above, the process becomes progressive. The disruption of one adhesion complex causes the adjacent elements to take up the load and then they fail. This begins a cascade that becomes self-sustaining and eventually leads to tendinosis. The degenerative changes progress until pain develops and finally the tendon ruptures.⁴³

Tendinosis creates a wide variation in cell density, ranging from areas of near zero cells to areas with many cells that are metabolically active. These areas also have collagen fascicles with unequal crimping, loss of transverse bands, ruptured fibers and increased type III collagen with decreased crosslinks. The abnormal microenvironments

and altered tensegrity of tendinosis induce differentiation of some tenoblasts into adipocytes, others to chondroblasts and even to osteoblasts, which explains the existence of lipid accumulation, mucoid formation and tissue calcification in areas of tendinosis.⁴⁴ In these areas of tendinosis there is a considerable increase in both nerves and vessels.⁴⁵

Rest alone is not the solution to tendinosis. *Figure 7* illustrates that some stress is necessary for the tendon to regenerate. The challenge for the patient and clinician is to find the right balance between stress and rest in all activities.

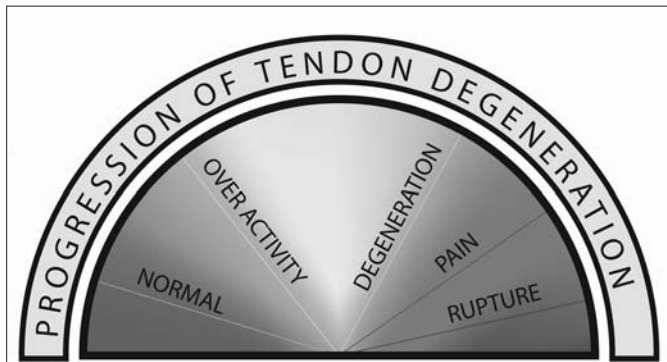


Figure 7. This image portrays the progression of tendinosis from (on the left) a normal response to stress by a progressively weaker and painful tendon to a structure that ruptures with the slightest additional stress. The image should give one an idea of the time for progression and recovery. It may be possible to go from normal to very painful or even rupture in one day but recovery is slower.

Ref: Ravin T. AAMM. 2011

A CASE FOR PROLOTHERAPY

Hierarchical tensegrity explains why it makes more sense to treat lax ligaments or compression elements before fixing tendons. If the ligaments are lax, there will be more stress on the tendons. This is particularly true in the case of conjoined LTs, where the injury or degeneration of one component directly affects the other. Prolotherapy allows the physician to repair the compression elements when they are stretched or torn.

In all the musculoskeletal system problems that we diagnose and treat, the ECM plays a critical role that is just beginning to be unraveled. Understanding how the tissues relate to the ECM and the ECM relates to the cells opens up whole new avenues for understanding the

musculoskeletal system. The ECM allows the smallest movements of our body to be transmitted almost instantly to every cell by way of tensegrity and prestressed elements. It now is clear that altered movement patterns not only waste energy but also change how our whole body works. It now is possible to imagine how postural decompensation can affect every cell in the body and how this could be critical in both health and disease. The developing awareness of the ECM as a key part in the mechanotransduction of stress from the tissue level to the cell opens up new avenues to explain how tissues heal their wounds and repair themselves.

Understanding the different functions of ligaments and their innervations should begin to influence how we treat them and with what. The idea that there are two different ligament types in at least three joints means that probably all joints have similar arrangements. Do we treat them all the same with our cocktail of choice or should we consider the function of the ligament before we start the injection? The ligament examination now takes on a whole new meaning. It will be interesting to see if the newer ultrasound and MRI machines and techniques will be useful defining the ligament function or functions and aid us in treatment decisions. “Proliferative therapy” seems the right phrase to explain what we are doing here by stimulating the growth of new ligament, and in 99% of the treatments the response to therapy is consistent with the well-established understanding of tissue repair and wound healing.

Tendons provide clear examples of hierarchical tensegrity in both health and disease. The new information on tendons, from the entheses to the tenocyte cytoskeleton, challenges all of us to integrate these ideas into our practices. This newer information should begin to help us reconcile the clear differences among the clinical history, physical examination, MRI, ultrasound and surgical findings. The wide discrepancies among these methodologies need to be reconciled to improve the diagnosis of tendinosis.

The current therapy for tendinosis once tendons are degenerated may best be described as regenerative. Rest now seems essential to healing. The question is how we balance stress and rest. Beyond rest, the treatment options for tendinosis seem to be equal to the number of doctors treating. This emphasizes the need for a better and more complete diagnosis of tendinosis. Understanding of

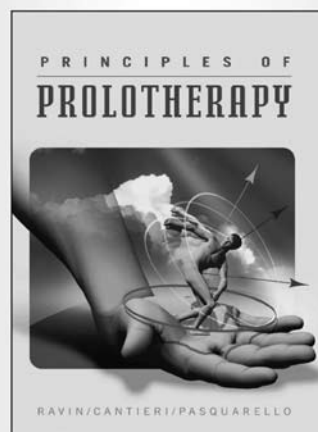
tensegrity can shed light as physicians seek to understand how ligaments repair, why tendinosis occurs and how cells respond to the stresses of living, working and playing. ■

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