1. Fundamentals of tendon

Introduction

Tendons were once thought to be relatively inert. They were thought to be a tissue whose only function and capacity was simply connecting muscles to bones. Prepare to get inspired!

Tendons are incredibly complex structures capable of both withstanding and transferring massive loads to enable the performance of amazing physical features. Olympic sprinters use their Achilles tendon like a spring for efficient and fast movements, withstanding loads of up to 15 times their body weight. According to Beyer et al. (2015), "tendon tissue is uniquely designed to withstand considerable forces to produce joint movement, and it may at times see loads exceeding 9 kN". In fact, if they had to contract and relax their gastrocnemius muscle during running, it would take minutes to run 100 metres!

Their superb performance is reliant on a relatively isometric calf complex contraction during contact and push and on their amazing Achilles tendon acting as a wonderful spring, with fascicles that slide and glide. A football player can cut and change direction very quickly thanks to their patellar tendon. However, tendons are not only responsible for athletic pursuits, they are integral for simple, everyday activities such as walking.

Figure 1: Sprinting

(Ruiz, 2019). F.C. Barcelona Brand Center

Tendons are found in many different shapes and sizes around the body, and their shape and structure reflect their function. Apart from having different shapes and lengths, they may lie around bony pulleys, enclose sesamoid bones or may be located within a muscle (intramuscular tendons.) To understand such a varied and important structure, understanding tendon composition and function (and differences between tendons) is

vital. Understanding this is also crucial for the clinician who desires to understand why tendon injuries occur and foresee who may be the most susceptible. Moreover, this text provides the framework for planning effective, progressive and thorough rehabilitation.

This module is designed to provide the basics of lower limb tendon structure and function as well as to explain some of our latest understanding of bioplasticity. You don't know what bioplasticity is? Keep reading!

Normal tendon structure

Most textbooks start with a description of the collagen within tendons. We are going to start with the cell, because we believe the cell is central to controlling the health of the tendon. In fact, it is cell changes that precede any changes to collagen in pathological tendons (Cook, 2004).

The cell is responsible for making the entire tendon structure; all of the biological materials, including collagen and proteins, are made entirely by the resident cells (Milz, 2009). These resident cells are predominantly tenocytes. They are described as spindle-shaped and, compared to many tissues, they are sparse in number. There is a lot we do not know about the tenocyte, but it is clear from laboratory-based studies, as well as in vivo observation, that this cell is sensitive to its mechanical and biochemical environment (Banes et al., 1999).

The cells communicate with each other, thus enabling co-ordinated reactions (Milz, 2009). One method of communication between cells is via gap junctions. These are specialised intercellular connections between the cytoplasm of two cells, allowing molecules, ions and electrical impulses to pass through. These gap junctions allow for a continuous pathway between cells – so you can see how the cell is truly in charge and aware of everything that is happening in the whole tendon. The cells communicate along rows and between rows. The gap junctions in tendons, also termed "connexins", are composed of either connexin 32 or 43. The gap junctions/connexins form rows and networks. Tenocyte rows are linked longitudinally by both connexin 32 and 43, but laterally by connexin 43 only.

Every time you rush to catch the bus, I want you to imagine your tenocytes communicating with each other! They are connected via connexins (32 and 43 along rows, 43 between rows.) This helps to have a co-ordinated response to load!

Interestingly enough, these gap junctions appear to have different roles in cell response to mechanical loading. While each gap junction/connexin responds to load (termed mechanotrandsuction), the integration of signals – the co-ordinated response enhances this. Do you remember connexion 32? This connexion is stimulatory, while connexion 43 is inhibitory. This form of communication is one of the ways cells control and co-ordinate their response.

What is mechanotransduction?

It is the mechanism by which cells convert a mechanical stimulus to a biological response. Simply put, it has to do with how a cell detects a mechanical stimulus and how it responds (usually by converting it to an electrical or chemical signal that, in turn, causes something to happen.)

Actually, tenocytes can sense mechanical load through direct contact to collagen and through fluid flow (cilia and cytoskeleton.) This means that when you move, fluid gets pushed perpendicular to the tendon and the cell can also sense this. This also means that tenocytes can detect tensile strain and stress (we will call this 'compression'.)

The cytoskeleton is the internal architecture that gives a cell shape and structure. Deformation of the cell is detected by the cytoskeleton and converted into a biochemical response.

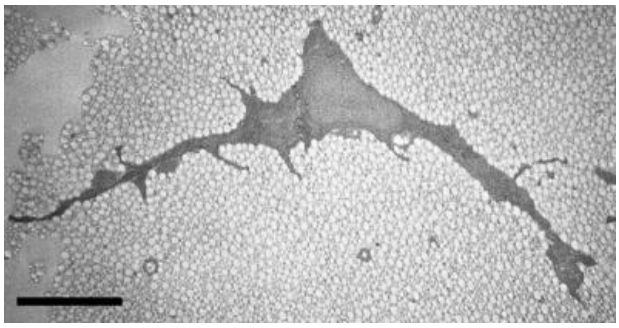
Strain is defined as the force tending to pull or stretch something.

Compression is the force applied perpendicular to the tendon.

Tenocytes have other ways of communicating and sensing load too. They have processes that reach out to touch a type of collagen termed 'cilia.' The amount of strain (load) directly impacts on cilia expression and this in turn affects signalling.

Because the cell is central to governing the health of the tissue, understanding its role becomes even more important to understand how pathology develops and how a tendon adapts. We have to consider the rest of the tendon. The tenocyte is located between collagen fascicles. So, let's learn about collagen now!

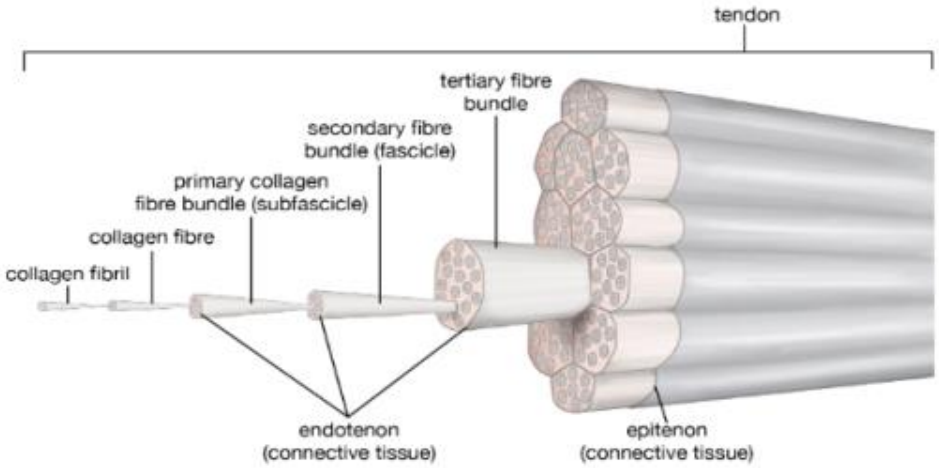
Figure 2: image of cross-sectioned collagen fibrils and a tenocyte showing long and thin cytoplasmic processes



(Franchi et al., 2007)

Tendon tissue that is not cellular is termed 'extracellular matrix' (ECM.) In general, tendon consists of ECM that is mainly type I collagen (60 - 90%), as well as proteoglycans, glycoproteins, elastins. Collagen is arranged in a hierarchical pattern. Tenocytes form procollagen, which is formed together as tropocollagen (Kjaer, 2004). These molecules are made up of a three polypetdice chain, formed in a triple-helix structure (Kadler, 1996). The smallest functional unit of a tendon is a collagen fibril. Enzymes bind the tropocollagen molecules together as a fibril. Fibrils are arranged longitudinally and overlap each other and allow for gliding between molecules as tendons elongate (Robbins, 1988). These small fibrils are bound together into fibers, which are then grouped into larger bundles termed "fascicles." (Figure 3)

Figure 3: Tendon Structure



(Roger, 2016)

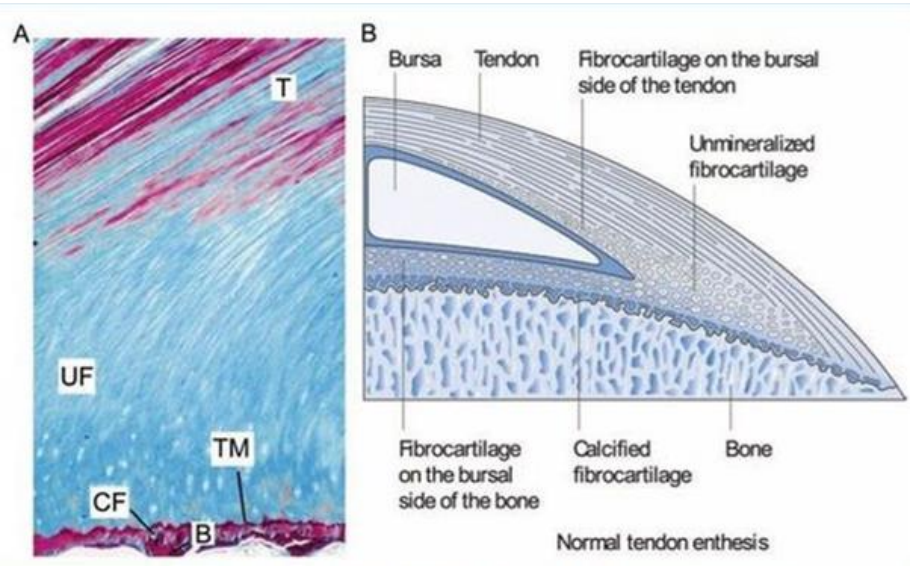
In addition to collagen, the ECM contains ground substance that consists of proteoglycans and glycoproteins. Proteoglycans, such as decorin or aggrecan, attract water, and may be thought of as important lubricators or spacers within collagen fibrils. Simply put, decorin is a small proteoglycan and can bind a small amount of water. On the other hand, aggrecan is capable of binding large amounts of water. A normal tendon has predominantly decorin and, thus, it has only a small amount of water. (Remember this when we start talking about changes in tendon pathology.) Proteoglycans are vital to maintaining the elasticity of tendons as well as to resisting compressive loading. Glycoproteins have a role similar to that of proteoglycans.

Compared to other body tissues, tendons are relatively avascular and aneural. Bloody supply may be variable and be influenced by exercise and muscle contraction. Tendons have mainly afferent nerve supply, and most nerves do not pierce the tendon itself. In fact, many tendons may have almost no nerve supply at all to the centre of the tendon. In contrast, the peritendon is richly innervated and supplied with blood vessels.

Now this makes sense! Can you imagine those nerves and vessels getting squashed every time we use our tendons? The outside covering of a tendon has a rich nerve supply. We will talk about pain and nociception in Module 2. For now, we want you to be aware that there are sensory nerves (afferent nerves also termed 'free nerve endings') that are limited to the periphery and that are capable of sending information about changes in the tendon that may be dangerous. Actually, the outside covering of tendon has a huge competence for nociceptive… bigger than viscera, joint, fat, and ligaments. We will explain the importance of this in Module 2.

Before delving into how tendons respond to different stimuli, it is important to understand the different structural regions. Most tendons consist of an aponeurosis (attachment to muscle), mid tendon, and enthesis. The enthesis is the transition from tendon to bone and it can be fibrous or fibrocartilaginous. A fibrous enthesis can be observed when a tendon attaches to the metaphysis or diaphysis of a bone, and it is typically a long attachment. Fibrocartilaginous entheses attach to the epiphysis or apophysis, and have 4 distinct zones of transition: from normal tendon to fibrocartilage, then to mineralized fibrocartilage and then to the bone. A number of other structures may be located around the enthesis, such as a fat pad or bursae. These have been termed the 'enthesis organ' by Benjamin and colleagues, as together they play an important role in load transmission and dispersion (Benjamin & McGonagall, 2001).

Figure 4: Normal Tendon Enthesis



(Brukner et al., 2018)

Tendons may be enveloped in a peritenon, which is made up of the paratenon (a looser outer layer) and the epitenon (a thinner inner layer.) The peritenon allows for gliding

within surrounding tissues and it is an important functional component of many tendons in the body. The paratenon may also include synovial cells – in this case, it is referred to as 'tenosynovium.' It is important to mention that the function of a tendon is often reflected on its peritenon. The tendons around the feet, ankles and hands have complex and thick peritenons, as there is a significant longitudinal movement around the surrounding tissues. By contrast, the patellar tendon has almost no peritenon at all.

The intra-muscular tendon is best described as a scaffold. It acts as a central supporting strut to which muscle fibres attach (Brukner, 2017). The intra-muscular tendon is different from the free tendon both in structure (it is unlikely to have well aligned bundles and specialised IFM), and in function (Brukner, 2017). Clinically, injury to the intra-muscular tendon manifests as a severe strain and it is probably the improvements in imaging that have allowed us to understand and diagnose these injuries. At a structural level, injury involves bleeding and oedema, in contrast with tendinopathy. Intra-muscular tendon injury should be conceived of as different from free tendon injury. Rehabilitation of intra-muscular tendon will not be covered in this course.

Normal tendon function

The function of a tendon is related to its shape and properties. Larger tendons will be able to withstand more load (e.g. the Achilles tendon), whereas longer tendons can store more energy. Tensile stress is calculated by dividing the load placed on a tendon by the cross sectional area. Because of this, the larger the cross sectional area, the more the load can be dispersed, and thus, the lower the stress. Tensile strain refers to how much the tendon elongates compared to its unloaded length. In this sense, thinking of the tendon as a spring can help to understand how shape relates to load tolerance.

Clinicians can think of tendons as energy storage tendons (such as those of the lower limb), or positional tendons (e.g. hand, finger tendons.) Lower limb tendons present elastic function best. During running, the Achilles tendon absorbs and recovers force as it lengthens during the early stance phases, which then released during the propulsive phase (push). Tendons of the upper limb are structurally different from lower limbs. In general, principles that apply to the lower limb tendons cannot be applied to upper limb tendons. Finger and hand tendons are very long and thin, which allows for large ranges of motion and dextrous function. Gliding is critical to their function; however, withstanding large amounts of energy storage loads is less important.

Tendons transfer the elastic energy of muscle onto their very stiff bony attachment to create joint movement. They also provide a transition of material characteristics from very compliant muscle to very stiff bones (Screen, 2015). The benefit of this transition is that it reduces focused areas of stress where failure may occur.

Clinical take-home message

Not all tendons are the same around our body. Their function is related to their composition. Tendons that act like a spring (energy storage tendons such as the Achilles) have a different make-up and role from those of positional tendons, such as the supraspinatus.

This means that not all tendons can be treated the same way!

Understanding tendon loads

Tendons are subject to different types of load. Broadly speaking, these can be broken down into four types: tensile, compressive, friction or shearing, and mixed loads.

The first thing that you need to know when you are assessing and managing tendons is what tendon load is. Tendons can withstand four types of load:

1. Tensile load: This is where we "ask" the tendon to store and release energy like a spring. This can be achieved by movements that involve velocity. The Achilles tendon can do movements like hoping or running movements. If we think of the patellar tendon, tensile load not only results from jumping, there is also a fast change of direction involved, or lunging or reaching. This occurs in tennis, for example. This is what high tendon load is. As we can see, it is really important to understand which loads we use on tendons as a spring, because it is the loads that end up being really provocative for tendons.
2. Compressive load: the tendon is squished against the bone. A good example of this occurs in the Achilles tendon, when it lengthens on dorsiflexion. Now you can start to understand why interventions like heel raises can help, whereby people are put out of dorsiflexion and, therefore, out of compression.
3. Friction or sharing load: Here the tendon withstands low load in repetitive movements. A good example is the case of cycling, where a person might go into repeated plantar and dorsiflexion, though the Achilles tendon is not used like a spring, but as a shearing of the outside layer of the tendon against the tendon. This is a provocative load which causes a paratendonitis. Movement is a provocative load for these kinds of conditions. However, it is not so for Achilles tendinopathy, with a calf raise for example. Which in fact are critical to use in Achilles tendinopathy.
4. Mixed load: The tendon combines the first two types of load: Fast energy storage movement while someone is in a compressed position. Pushing off from a dorsiflexion position like a fast change direction, can be an example for this type of load.

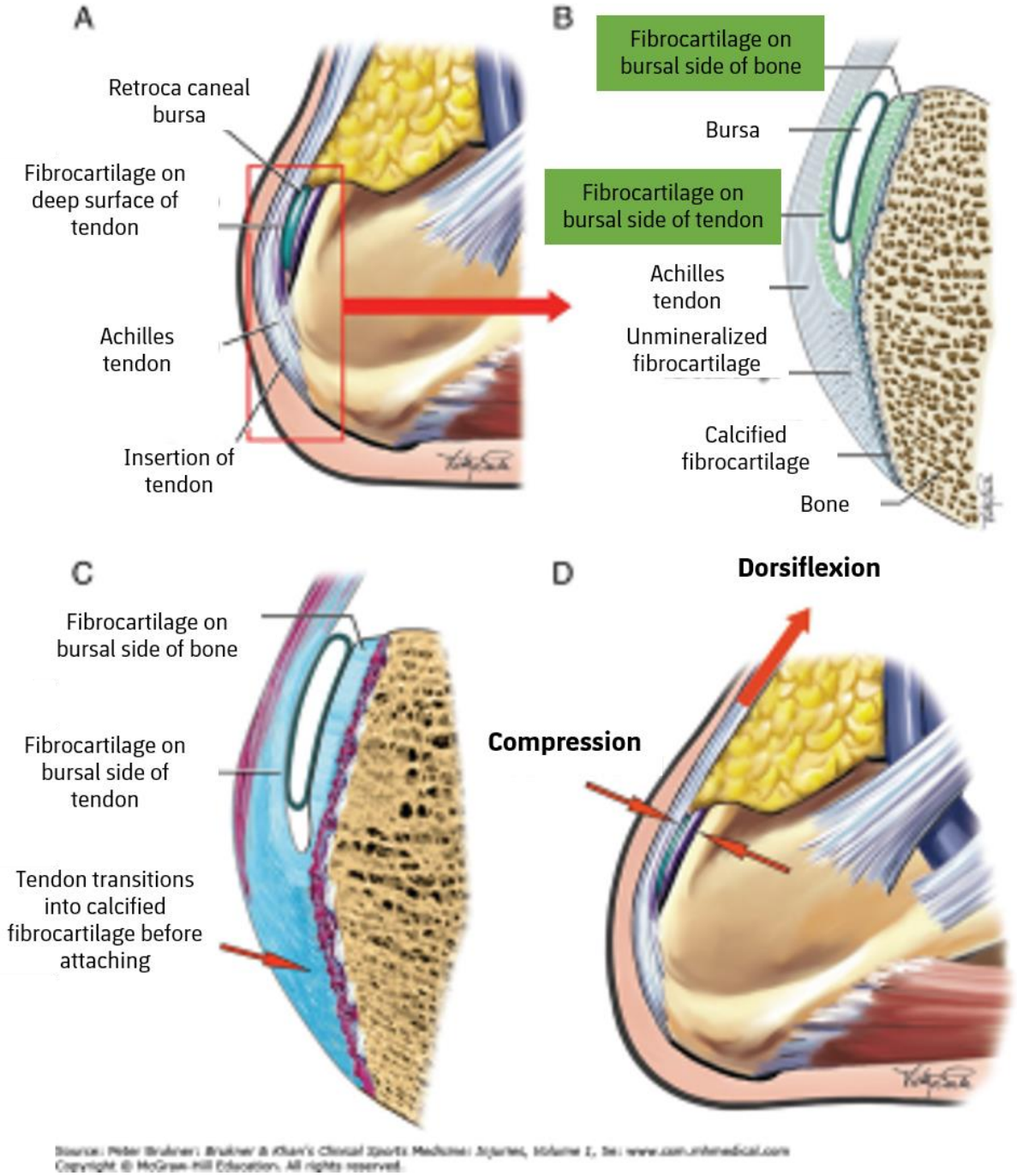
It is important to understand this type of load, because it is why we can see Achilles tendinopathy throughout an athlete's lifespan. This condition can be seen in young athletes who run and jump, but it can also be observed in older or more sedentary people. This is so because the Achilles tendon must be used like a spring in any common life activity which involves the lower limbs producing force against the flour. In contrast with the patella tendon, this type of load can only be seen in young people who jump, the only ones capable of bearing high loads through their patella tendon, to be able to quickly jump and change directions. On the other hand, running does not mean high patella tendon load. Thus, it is important to understand what high tendon load is for each tendon.
Understanding this will allow practitioners to decide which type of load and with what intensity to start working with, which will depend on the athlete or patient. If fast energy storage loads are provocative, then a static or a slow load must be used to start the process. Therefore, in order to remove loads, if we understand which loads are provocative, advisory can be highly targeted, by reducing fast loads and giving some advice on how to intervene over compressive loads.

This is why differential diagnosis matters; it is necessary to decide treatment direction. By knowing the four types of tendon load, practitioners can determine whether or not the person in front of them is describing high tendon load. In this way, it will be possible to know which loads to remove and which ones to start rehabilitations with.

Tensile loads may be thought of as spring loads. These are the ones that first come to our mind when thinking about tendon loads. However, they are often misconceived. Tensile loads may be different, based on factors such as the rate of loading. Although lifting a heavy weight may be a significant tensile load, when performed slowly, there is a very slow rate of loading, which results in minimal energy storage requirement. It contrasts with jumping, where the rate of loading is much faster and thus results in higher energy storage loads. Tendons are built to withstand these energy storage loads; however, there may be a 'sweet spot' between too much or too little to maintain homeostasis.

Compressive loads usually occur around a boney fulcrum or another external source, such as retinacula or fascia. Examples of these loads include the insertion of the Achilles tendon in dorsiflexion or over the greater trochanter in the gluteal tendons during pelvic drop or adduction. Compression loads lead to increases in larger proteoglycan production. This may be an attempt by the tendon to resist the compressive loads.

Figure 5: Achilles tendon insertion dorsiflexion compression effect



Source: Brukner et al., 2018

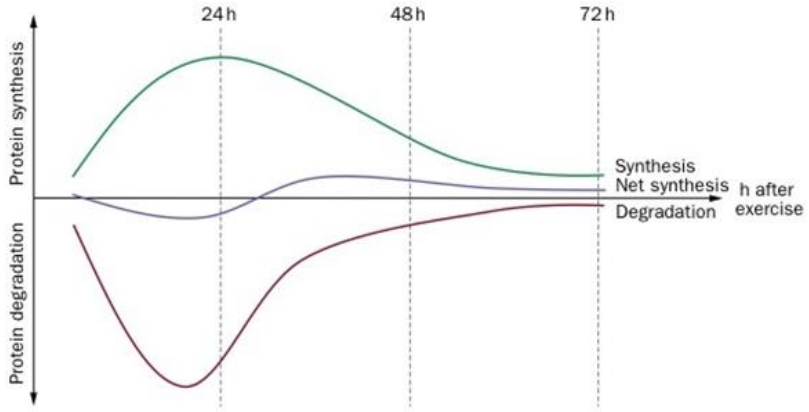
Friction or shearing loads occur most commonly in tendons with a significant peritenon. These loads are more pronounced when the tendon undergoes large, repetitive range of motion within the tendon (and not necessarily under significant load.) Excessive friction loads may lead to irritation or inflammation of the peritenon structures. Eventually, the peritenon structure may change because of friction or shearing loads.

Of course, all of these load types do not occur in isolation but are usually mixed. However, when combined, it may be more difficult for the tendon to uphold normal homeostasis. An example in which a combination load occurs is when a person runs up a hill. The

running places a tensile load on the tendon, while compression load is also created as the foot is more dorsiflexed while running up hill. This combination load is sometimes seen as a precursor to symptoms (other factors may also be involved, of course.)

Structurally speaking, tendons respond to load in a complex cell-driven response that is not fully understood. Tenocytes alter their shape and action in response to load, and play an important role in mechanotransduction. Generally speaking, tensile loading leads to temporary changes in collagen degradation, synthesis and ECM protein production. These changes may occur for 24 to 72 hours before returning to normal levels. Increased collagen production is often cited in relation to load. However, after puberty it is likely that most of the increased produced collagen be not actually integrated into the ECM, and after adolescence collagen turnover is minimal. As we can see, it seems that collagen matrix has limited scope for change in relation to load once adulthood is reached. On the other hand, other proteins, such as the proteoglycans and glycoproteins discussed above, may be more load-responsive and allow for adaptation. An increase in PG production is a normal response to load, though, again, how this normal response may become pathological is not fully understood. As well as these more global changes, tendon loading leads to increased production of growth factors, cytokines, as well as to the release of substance P and calcitonin gene-related peptides (CGRP). All of these factors are unregulated temporarily before returning to normal levels in the short term.

Figure 6: Schematic representation of collagen synthesis and degradation



(Chuah, 2019)

The influence of different contraction types on tendon responses has been suggested as an important consideration. Nevertheless, contraction type does not seem to affect tendon responses; total tendon stress and strain are more implicated.

Clinical implications of understanding tendon load in presentation

For the treating clinician it is vital to understand how different loads affect different tendons and different people. To make a diagnosis they must be able to understand and work out what loading factors were present in a patient's history and how these may have led to the symptoms. For instance, in the

absence of significant or changing tensile or compressive forces, it may be more pertinent to consider differential diagnoses to tendon conditions, as these usually occur as a result of loading. Understanding the different loads is also critical to specific ruling in of a diagnosis, even if pain and symptoms may be located in a similar region. An example of this may be a recreational cyclist that presents with Achilles pain – cycling does not put the Achilles tendon under significant energy storage load, however with poor bike set up or technique, the tendon may be exposed to friction/shearing loads if the cyclist has their heel off the back of the pedal and repetitively falls into dorsiflexion. Only by understanding loads is the clinician able to differentiate these points.

In addition to diagnosis, understanding load is also critical to managing symptoms and effective rehabilitation prescription. The first step in any musculoskeletal management is to manage symptoms – in tendon injuries this is managing pain. Clinicians must be able to identify what loads are causing pain and how they can be managed, minimised or modified. Sometimes this can be obvious (e.g. if someone has just started a running program for the first time and they experience Achilles symptoms) but other times it may be more complex, (such as the busy schedule of an elite athlete with many different training types). For the best rehabilitation the clinician must identify the current loading capacity of the patient, the goal capacity and how to progress the patient through the journey from one to the other.

Changes that occur in pathology

There is considerable debate about how exactly pathology develops in tendons and its relevance to clinical presentations. As in many other musculoskeletal conditions, the relevance of structural pathology has been recently questioned. Given this, while discussing tendon pathology, it is important to remember this is independent of pain and symptoms.

The final stage of tendon pathology has been extensively researched and described. Tenocytes change their shape and become larger and rounder. More tenocytes are present in pathological tendons, and cell activity becomes altered, with increased protein production and metabolic activity. In addition to elevated tenocyte levels and activity, more proteoglycans (PGs) and glycoproteins are present, especially aggrecan and versican – 2 larger PGs. This results in more bound water, increasing the volume of tendons. Collagen production is increased, with more type III collagen produced than in normal homeostasis.

Perhaps the most common feature of tendon pathology is collagen matrix disorganisation. The disorganisation is characterised by loss of collagen alignment, thinner collagen fibrils and increased percentage of type III collagen. More importantly, this disorganisation occurs in an isolated segment of the tendon, and not across the tendon. The disorganised section of the tendon has diminished the load bearing capacity and may become 'mechanically silent.' Despite this most tendons will maintain sufficient aligned fibrillar structure despite a pathological lesion (Docking et. al, 2016). As a result of the large scale changes to the ECM, an ingrowth of nerves and vessels occurs as

pathology progresses and may occur as they 'grow' into the spaces left due to structural disorganisation.

The final stage tendon pathology is now well understood; however, the pathoaetiology of those changes and its relevance to pain and symptoms is poorly understood. I It was previously thought that collagen tearing, and degeneration of collagen fibers were responsible for tendon pathology (some models refer to micro-tearing of collagen). Failed healing has also been suggested as a cause of tendon pathology. These two, however, do not account for symptoms, or explain the other factors present in the tendon pathology process. Also, collagen injury as the primary event in tendon pathology development has not been supported, and changes in collagen structure are preceded by cellular changes and alterations to ground substance.

The role of inflammation in tendon pathology has been continually debated across research and clinical fields. Several studies have shown elevated levels of inflammatory cells and mediators present in pathological tendons; however, some of these are also present in normal responses to loading – i.e. a mechanical stimulus may result in elevated inflammatory cytokines, as a normal response. Also, all tissue injury states will have inflammatory processes, and at this stage evidence to suggest inflammation as a driver of symptoms or pathology is scarce. Anti-inflammatory treatment methods such as rest and medications are not considered to be effective in treatment. An association has been found between inflammatory cells and blood vessels in the tendon; however, neovascularisation is only seen in late stage pathology, and there is a poor correlation between this and clinical symptoms.

At this stage, it is safe to say that inflammatory processes are present in tendon pathology, but their role in pathogenesis and/or symptom development is not confirmed at this time. The tenocyte is the most mechanically responsive element in the tendon and may be responsible for any changes in inflammatory markers or cells. Thus, it may be used for autocrine or paracrine signalling, as opposed to an inflammatory process.

The clinical relevance of staging

The continuum model of tendon pathology was first presented by Cook & Purdam in 2009 and was updated in 2016. The model proposed three distinct stages of pathology that helped explain how pathology develops and its relevance clinically. More importantly, the model focused on tendon pathology, but it was not designed to describe the pain process that occurred in tendinopathy.

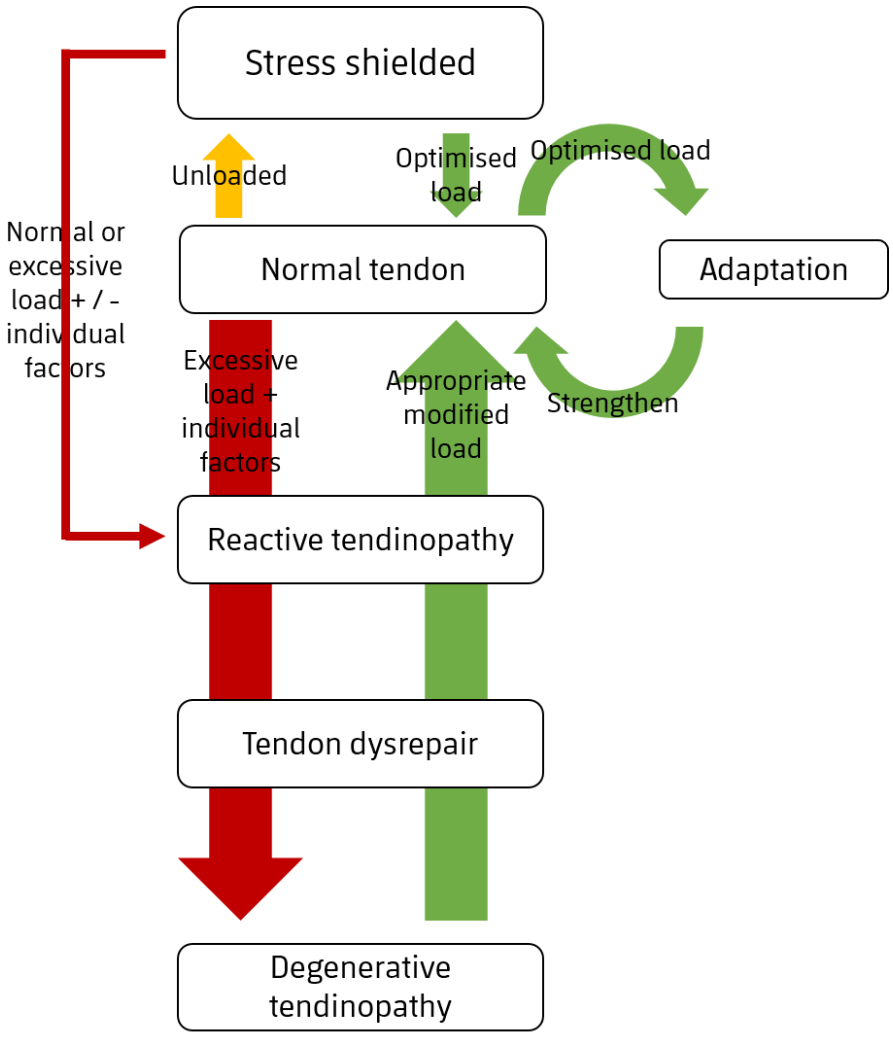
Reactive Tendinopathy

The first stage in the development of tendon pathology is reactive tendinopathy. A reactive tendinopathy is a short-term response to an overloading stimulus which causes a hypercellular response and adaptive tendon thickening (to reduce stress). Tenocytes change their shape to become more chrondroid and produce larger proteoglycans. Because of this, more water is drawn into the tendon thus the volume increases (and cross sectional area increases) (Magnusson, 2010). Moreover, there is minimal or no damage to the collagen matrix at this stage. Inflammatory cells and markers may be

present at this stage; however, there is no evidence today that suggests that this is the driver behind pathogenesis.

A reactive tendinopathy may not show significant changes on imaging beyond an increase in tendon thickness, as this stage is not characterised by collagen disruption. On ultrasound tissue characterisation there may be evidence of a change in the quality of tendon structure, with increased type II signal area, but no large areas of significant degenerative pathology (type III and IV will be observed).

Figure 7: Tendinopathy cycle



Source: Cook, et al., 2016

For the clinician, reactive tendinopathy is often associated with a painful presentation – an acute bout of overload will often bring on pain and other symptoms with this stage. Exposure to unaccustomed exercise, or commencing a type of exercise after a period of rest is a common source of overload. A direct blow to the tendon may also cause a reactive tendinopathy (Garau, 2008).

Despite the cellular reaction that occurs in a reactive tendon, the tendon may 'return' to normal structure (as no collagen disruption occurs) if the overloading stimulus is

removed, and the response is allowed to settle. If the response is not correctly managed, the pathology may further progress.

Tendon Dysrepair

If the original reactive response is not settled, fibrillary disorganisation begins, and the tendon falls into dysrepair. Matrix disorganisation remains localised, and large scale areas of degeneration are not seen. This stage shares similarities with models of tendinopathy described as 'failed healing' (Fu et al., 2010). Clinically, this stage is often not seen, and it is rarely associated with symptoms.

Degenerative Pathology

This stage has been extensively studied in the published literature. Larger scale matrix degradation, collagen disorganisation and even cell death are characteristic of degenerative tendon pathology. As degenerative pathology progresses, blood vessels and nerves grow into the empty spaces of the deformed matrix. All degenerative pathology is irreversible – the area of pathology becomes mechanically silent and does not transmit load. The area of degeneration is usually contained to a smaller section of the tendon, and as such the whole cross-sectional area of the tendon is rarely affected. For the clinician, the degenerative stage is most often encountered in someone with a long history of tendon loading, or symptoms that have led to the degradation of the matrix over time. Despite this, there is controversy whether the area of structural disorganisation is a source of symptoms, given the high rates of asymptomatic pathology in pain- free people, as well as lack of load bearing capacity of degenerative areas.

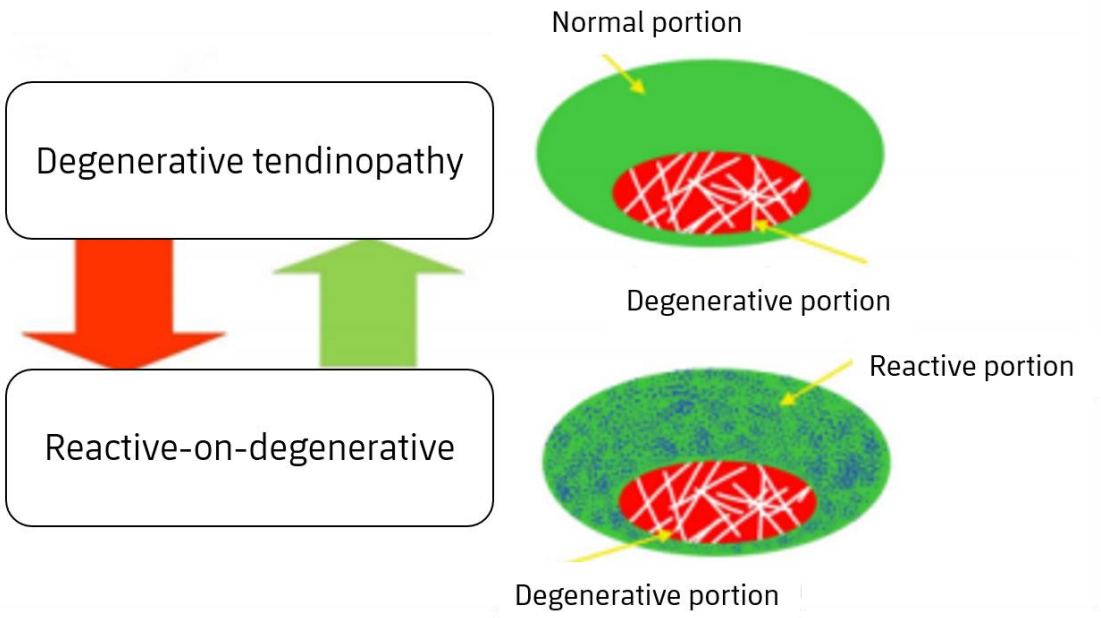
Based on the continuum model of pathology developed by Jill Cook and Craig Purdam to stage tendon pathological changes, we must remember that pathology does not equal pain.

If we are considering the relevance of pathology in staging and managing of the person in front of us, it really comes down to lots of person-related factors. This means that if we consider people with tendon pain, they will present two clinical presentations.

The first one is reactive tendinopathy. Young athletes with first presentation of tendon pain usually have an acute overload that may have been recruited for their first state level team, or they might have played a tournament and had a huge increase in their load. And what we see is a big increase in pain related to that load. You might have experienced this yourself if you overdo it in some way. If you cannot play golf because you are not used to playing 18 holes, you can certainly have some medial elbow tendon symptoms. Where you might notice that if you do not play for a few days, your pain settles down. Pain has a profound effect on changing your behaviour; as a result, we can see that reactive tendon apathy is generally found in younger people, and the same happens with acute onset of load and first-time presentation. This is a very recoverable tendon from a pathological perspective. What we see is intact college and fibers. If this people want to be imaged, there might be some swelling. Which is an important term to use in language to educate patients. You can remind people that this is not an inflammatory process. They are cells that have attracted water. Therefore, you can say to people that this is a water response within the tendon, but we still have lots of lovely load bearing intact tendon

within that tendon. Our second key clinical presentation is a mixed presentation of reactive on degenerative. This means that a person will have an area of pathology that on imaging would be called degenerative. They would have malalignment of college and fibers, but they would have lots of load bearing tendon around that area. Thus, the reactive on degenerative is an overload of the remaining part of normal tendon; that is, they have a reactive on the background of a degenerative pathological area of their tendon. This is by far the most common clinical scenario that we will see. So that people have had a history of tendon pain on and off over the years. If you look at them, they can have variable lumps and bumps that can cause atrophy to their muscles. It is important to understand what our goals are from recovering the person in front of us. Which are, recovering their pain and their function, not their pathology. So, someone with a reactive tendinopathy can completely return to normal. It is imperative to avoid invasive or aggressive interventions in the tendon. Itis predominantly a load management option and we have to make sure that athletes, parents and coaches understand how critical it is to reduce the overload and then address any deficits to return that person back to their function. When you have someone with a reactive-on-degenerative tendon, you need to know that you will never change that degenerative area of pathology.

Figure 8: Schematic representation of 'reactive-on-degenerative' tendinopathy



Source: Cook, et al., 2016

What we are trying to do is to reduce their pain and restore their function so we do not seriously image this person because changing their pathology is not important.

The other clinical importance of staging comes down to listening to our tendon. So, someone that has a reactive tendon can have very acute pain and we want to settle their pain down as much as we can. People that have had years of symptoms might have some morning pain and stiffness in the Achilles. Your goal is to return them to their normal

baseline level of low and stable symptoms. And that may not be zero. Some people might complain about the some pain one, two or ten minutes of morning stiffness. And if that is their baseline, that is the outcome for this person. So, it is not about telling people how much pain they should put up with, but Itis about understanding the people in front of you staging their pathology, pain and function and helping them address their deficits.

Adaptation and bioplasticity at a local level

It appears that our tendons are capable of remarkable adaption. There is evidence in the patellar and Achilles tendons that most of the tendons increase their cross-sectional area to compensate the areas of disorganisation. This increase in tendon size is a way of reducing tendon stress. This has fantastic implications for patients!

However, we hope you have learnt from this chapter that not all tendons are the same. The supraspinatus tendon does not seem to get thicker with pathology. Further, the tibialis posterior tendons and gluteal tendons may also differ.

Implications or clinicians

In order to summarize module 1, it is important to consider the next three key points:

1) The pathogenesis of tendon pathology is from the cell. That is, the cell drives all the consequent changes in the tendon.
   Inflammation probably exists at very low levels and probably does not have any implications in terms of pathology and pain. Collagen tearing exists; however, it is a consequence of those changes driven by the cell.

2) The second point I want to make is that vascularity is a terminal part of tendon pathology and it is neither responsible for driving a reparative response, nor for pain. Therefore, vascularity is a good marker of tendon degeneration rather than a marker that is implicated clinically in any aspect of the pathology or the pain.

3) The third point I want to make is that degeneration is final. That is, once a person has reached degeneration and that is marked by large areas of vascularity, there is nothing that you can do about this. In other words, it is a condition that will stay and its implications are relatively low for the tendon. As we discussed before, the tendon has already adapted to having an area of pathology and is quite capable of taking large amounts of load. Therefore, we should not be focusing on pathology, tendon injury and tendinopathy because they have no implications in the long term either for the ability of the person to recover from their pain or for the tendon to deal with the loads that are placed on it.

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