**Continuum of Tendinopathy Concept and Updates**

Widely debated, the continuum of tendon pathology, injury and repair has taken many twists and turns over the last 30 years.

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The term "tendontitis" (inflammation of the tendon) is considered in many academic circles as a misnomer over the last decade, in light of histological evidence to suggest that there is no evidence to suggest an inflammation is the key element of tendon pathology, with the term "tendinopathy" (pathology of the tendon) being preferred.

Leading expert in tendinopathy, Jill Cook, has unearthed some further evidence in the last 5 years and proposed further updates to the widely accepted *Continuum of tendinopathy*.

**Pathophysiology**

Fundamentally, the seminal ‘continuum model of tendinopathy’ proposed by Purdam and Cook (2009) lays out a framework of developing tendinopathy from acute, reactive tendinopathy to tendon disrepair to degenerative tendinopathy (See figure 1)



Figure 1: Continuum of tendinopathy (Cook and Purdam, 2009)

In review of this model, three stages of tendon pathology are considered:

1. **Reactive tendinopathy**

Fundamentally, a tendon stretch and deform in response to strain and resist stress due to its tensile nature and has the ability to return to its original state when the load is removed. But this response is age dependent, complex and is bound by the tendons own physiological limits.

There are many factors involved regarding the mechanical limitations of a tendon but essentially if the tensile limits are breached or there is compressive overload, we enter a stage of **reactive tendinopathy.**

This is a non-inflammatory response whereby there is a short term proliferative adaptation of the cellular matrix. That is, that the tendons cross sectional area may increase in a general/diffuse fusiform manner to reduce stress or mechanical properties will appropriately adapt by increasing the stiffness.

If the load is appropriately modified, the collagen integrity can be maintained and the morphology of the tendon returned to its original state

**Imaging Findings:** The tendon is swollen in a fusiform manner; the diameter is increased on both magnetic resonance imaging (MRI) and ultrasound (US) scans. Ultrasound shows reflection from intact collagen fascicles, with diffuse hypoechogenicity occurring between intact collagen structures. Magnetic resonance imaging will show minimal or no increased signal at this stage.

1. **Tendon Dysrepair**

This stage was proposed to represent the attempt made by the body at tendon healing, similar to reactive tendinopathy, but when there is chronic overloading of the tendon and there is greater cellular matrix breakdown.

At this stage, changes are more focal and varied than the reactive stage with an increase in vascularity and associated neural ingrowth. In the continuum, some reversibility of the tendinopathy is considered possible with appropriate load management to stimulate changes to the matrix structure.

**Imaging Findings:** The imaging changes reflect increased matrix disorganisation, and these tendons are swollen, with increasing evidence of collagen disorganisation. On US there is some discontinuity of collagen fascicle and small focal areas of hypoechogenicity. The increase in vascularity may be evident on colour or power Doppler. On MRI the tendon is swollen and there is increased signal within the tendon.

1. **Degenerative tendinopathy**

This represents the latter stages of tendinopathy, whereby areas of cell death and the tendon matrix is decimated, leading to a disorganisation of the tendon matrix, loss of collagen and extensive neovascularisation (growth of vessels). The risk of rupture is markedly increased. It is suggested in the original model that using interventions/treatments at this stage which have been shown to increase cellular activity, increase collagen production and restructure the matrix are appropriate for this stage. *However, it has since been suggested when revisiting the continuum in 2016 by Cook et al, that unfortunately this stage has limited reversibility and therefore interventions designed to change the structure of the degenerative portion as less critical.*

**Imaging Findings:** The compromised matrix and the vascular changes can be extensive. These appear on ultrasound scans as hypoechoic regions with few reflections from collagen fascicles. Numerous and larger vessels are usually visible on Doppler US. Magnetic resonance imaging demonstrates increased tendon size and intratendinous signal. The changes are more focal rather than spread throughout the tendon.

**Update**

Since 2009, it has been challenged that identifying structural changes alone is no longer the principal take home approach in classifying tendinopathy. In the continuum revision/update in 2016, emphasis was given to the complex interplay between structure, pain and function.

With all things considered, tendinopathy is principally considered a pain condition. However, treatment solely directed at pain often does not complement improving function (albeit challenging the pain aspect will in principle allow the patient to better tolerate the interventions required to improve function and structure.

It is suggested from the update that the continuum is carefully considered to help guide the clinician to the appropriate clinical treatment depending on the stage of the pathology and potentially the priority to the patient, but to fundamentally aim for little pain and good function.

**Pain**

It is now suggested that pain appears to be primarily caused by the functional cell of the tendon (tenocyte) signalling to the mechanoreceptors (Sensory cell that responds to mechanical pressure or distorsion) in or near the paratenon (The outer layer of the tendon matrix containing the ingrowth of nerves and vessels). This stimulation of the mechanoreceptors in turn stimulates the peripheral nerve to interpret this as pain.

This explains why structural pathology deeper in the tendon can often not elicit pain due to its remoteness from the paratenon.

In the reactive stage, the diffuse increase in tendon size may irritate the paratenon and account for the presentation of pain.

**Function**

Dysfunction has historically carried less weight in tendinopathy that pain and structure which have been deemed as more important. However, improving function will help the athlete to be able to effectively return to sport (albeit removing the barriers to improve function are also crucial). Also, a dysfunctioning tendon is likely to predispose the athlete to reinjury.

**Structure**

As mentioned earlier, because the degenerative tendon has limited reversibility, attempts to regenerate these areas of degeneration are likely to be futile. The original continuum suggested that treatments should be focused to stimulate collagen generation in the degenerative

regions and restructure the matrix but this has since been deemed ineffective.

Instead, the update suggests that treatment should be focused on building the load capacity of the less/unaffected tendon portion of the tendon and effectively increasing the cross sectional dimension of the organised aligned collagen.

Hence…. Treat the doughnut (Area of normal/less affected tendon) and not the hole (Area of degeneration

**References**

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