

Journal club:

JL Cook, CR Purdam. Br J Sports Med
2009;43:409-416

Is tendon pathology a continuum? A pathology model to
explain the clinical presentation of load-induced tendinopathy.

Dr. J. Gielen

Is tendon pathology a continuum? A pathology model to explain the clinical presentation of loadinduced tendinopathy.

- JL Cook, CR Purdam. Br J Sports Med 2009;43:409-416
- Tendon load >
 - Anabolic
 - Catabolic
- Key factors etiopathogenesis:
 - Energy storage and release
 - Tendon compression

Individual Factors influence amount of tendon pathology and onset

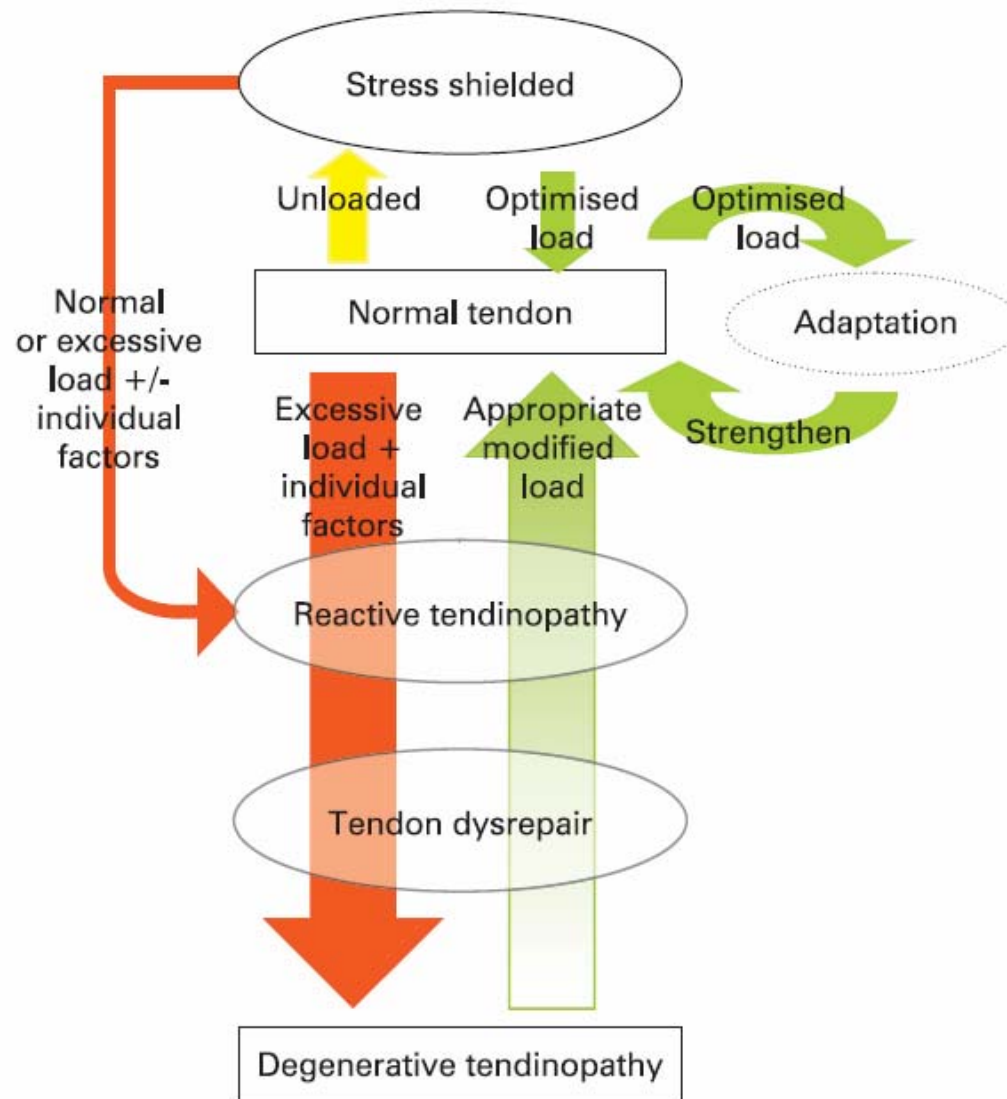
- These have to be considered when developing a treatment plan
 - First time tendinopathy in young athlete ><
 - Chronic tendinopathy in postmenopausal woman
- Pathology and response to therapy are different in these presentations
- Interventions tailored to the pathology

Tendon Pathology Concepts

- **Degeneration**: hypoxic, hyaline, mucoid
 - End stage pathology, disintegration of the matrix
- **Healing** phase: angiofibroblastic hyperplasia
 - Active cells, increased protein production but disorganisation and neovascularisation
- Tendon **unloading** ~ chronic overload
 - Decreased mechanical integrity of the tendon
 - Reversible

New Model of Tendon Pathology

- Continuum with three stages
 - **Reactive** tendinopathy
 - Tendon **dysrepair** (failed healing)
 - **Degenerative** tendinopathy
- Load: stimulus that drives a tendon forward or back along the continuum
 - Reduced load: tendon returns to a previous level of structure and capacity within the continuum



Pathological, imaging and clinical manifestations? Reactive Tendinopathy

- Acute tensile or compressive overload
 - Non-inflammatory proliferative response in cell and matrix
 - Adaptation: homogeneous thickening of a portion of the tendon
- $><$ normal tendon adaptation to tensile load:
 - Tendon stiffening without change in thickness

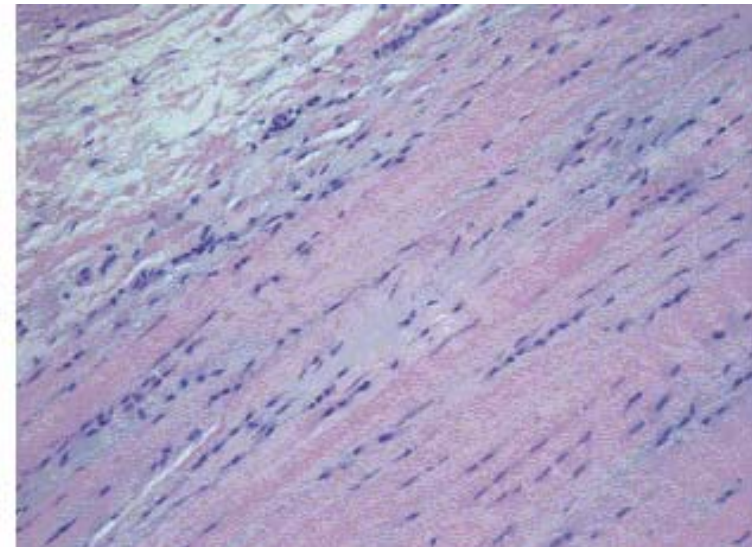
Reactive Tendinopathy

- Clinically
 - Acute overload of physical activity or direct blow
- Pathology
 - Non-inflammatory quick response: metaplastic change in cells and cell proliferation
 - Fibroblasts: chondroid shape, cytoplasmic organelles for increased protein production
 - Proteoglycans > increased bound water
 - Glycoproteins (hyaluronan)
 - Maintained collagen integrity, some longitudinal separation
 - No change in neurovascular structures
- Short term adaptation to overload
- Potential to revert to normal if overload is reduced

Reactive Tendinopathy

- Imaging (US and MRI)
 - Fusiform swelling with increased diameter
 - Intact collagen fascicles (US)
 - Increased water bound in proteoglycans
 - Hypoechoogenic (US) between intact collagen structures
 - Minimal or no increased SI (MRI)

Late Reactive Tendinopathy

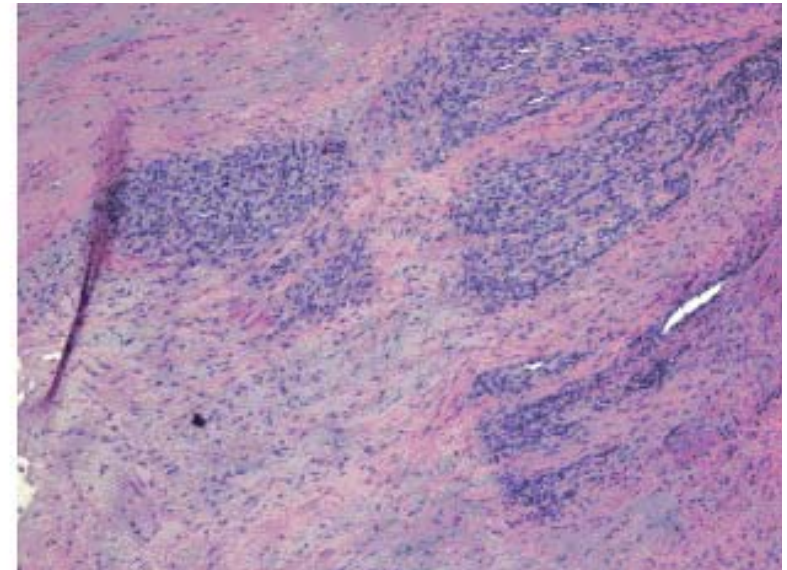
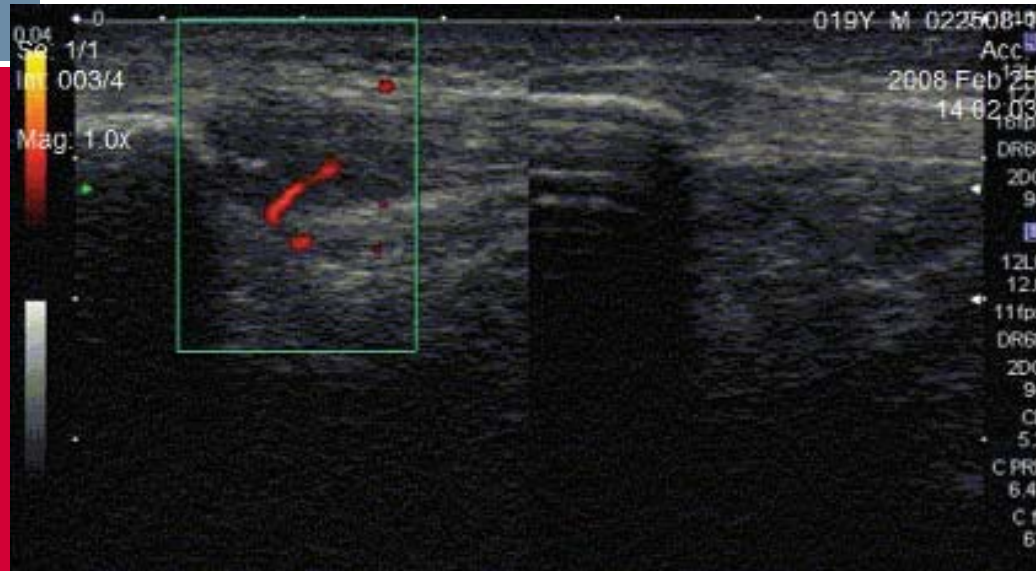


- Increased:
 - cell number, cell rounding, ground substance

Tendon Dysrepair

- Attempt of tendon healing with greater matrix breakdown
- Pathology
 - Increased number of cells: chondrocytic, myofibroblasts
 - Increased protein production:
 - Proteoglycan: separation of collagen and disorganisation of the matrix
 - Collagen
 - Increased vascularity and neuronal ingrowth may exist

Tendon Dysrepair, Early Degenerative



- Increased cell number, loss of collagen, increased number of vessels

Tendon Dysrepair

- Imaging
 - Collagen disorganisation
 - US:
 - small areas of hypoechogenicity
 - Increased vascularity: power Doppler
 - MRI:
 - Swelling with increased signal within the tendon
- Clinical
 - Reversibility of the pathology is possible with load management and exercise to stimulate matrix structure

Degenerative Tendinopathy

- Pathology
 - Progression of matrix and cell changes
 - Areas of cell death: apoptosis, trauma, tenocyte exhaustion
 - Areas of acellularity
 - Large areas of disordered matrix filled with vessels, matrix break down products and little collagen
 - Heterogeneous matrix: degeneration interspersed with reactive and dysrepair areas
 - Little capacity for reversibility

Degenerative Tendinopathy

- Imaging
 - US:
 - hypoechoic areas without collagen reflections
 - Power Doppler: Numerous large vessels
 - MRI:
 - Focal areas with increased signal in a thickened area
- Clinical
 - Chronic overload in athletes or primarily in older persons
 - Rupture is possible (97% of ruptured tendons have degenerative changes)

Placing Clinical Treatments in the Model

- Where is a tendon in the pathological spectrum?
 - Two clear groups on basis of clinical and imaging findings
 - Reactive/early tendon dysrepair
 - Young patient acute overload
 - Late tendon dysrepair/degenerative
 - Old patient with acute overload
 - Young patient with chronic overload
 - Large areas of swelling
 - Distinguishing between tendons that can return to normal and that can not!!!

Placing Pain the Model

- Pain can occur at any point
 - Dissociation of pain and pathology
 - Normal tendons on US: may be painful
 - Degeneration with rupture: may be painfree
- Source:
 - neurovascular ingrowth in tendon dysrepair/degeneration
 - Other? ((paratenonitis?))

Treatment

Reactive tendinopathy/early tendon dysrepair

- Physical
 - Load reduction
 - Tendon load without energy storage and release
 - Cycling, strength-based weight training
 - Aggravation due to
 - High load elastic or eccentric loading with little recovery time
- Pharmacotherapy
 - Ibuprofen: no detrimental effect on tendon repair
 - Peritendinous injection of corticosteroids to decrease pain

Treatment

Late tendon dysrepair/degenerative tendinopathy

- Physical treatment
 - Frictions: variable results
 - ESWT: pain relieving, not consistent superior to placebo
 - Ultrasound: increased protein production, variable results
- Surgery: return to sports in 50-80% at previous level

Treatment Late tendon dysrepair/degenerative tendinopathy

- Pharmacotherapy
 - Glucose and blood: induces matrix changes
 - Injections regardless the substance: induces matrix changes
 - Sclerosing therapy: effective in pain treatment and structure

Placing Exercise in the Model

- Eccentric exercise affects pain and structure
 - Increased collagen production in abnormal tendons
 - Improved tendon structure in short and long term
 - Pain relieving in 4-6 weeks
- Reactive/early dysrepair: probably no improved outcome
- Degenerative phase: positive stimulus for cell activity and matrix restructuring