New insights into the mechanisms of tendon injury
What is normal tendon?

- A extracellular matrix tissue
  - Type 1 collagen
- Small amounts of CRITICAL proteins
  - Ground substance
    - Proteoglycans, glycoproteins
- Structures that make the tendon a living structure
  - Cells, nerves, vessels
Tendon pathology

- Metaplastic change to fibrocartilage
  - Cell proliferation
    - Rounder
  - Ground substance increase
    - Larger (compressive) proteoglycans
  - Collagen degradation
    - Loss of Type 1
  - Neurovascular proliferation
What happens first in tendinopathy?

- Does a tendon go from normal to pathological in one step?
  - What happens first?
    - Collagen tear?
    - Vascular ingrowth?
    - Cell reaction?
    - Ground substance increase

![Combination of histopathological changes](chart)
How does pathology develop?
Normal tendon

Excessive load + individual factors

Normal or excessive load +/- individual factors

Stress shielded

Optimised load

Reactive tendinopathy

Appropriate modified load

Tendon dysrepair

Degenerative tendinopathy

Optimised Load

Adaptation

Strengthen
## Model of tendinopathy

<table>
<thead>
<tr>
<th></th>
<th>Reactive</th>
<th>Tendon dysrepair</th>
<th>Degenerative</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tendon response</strong></td>
<td>Adapting to load</td>
<td>Attempting to heal</td>
<td>Gives up on healing</td>
</tr>
<tr>
<td><strong>Pathology</strong></td>
<td>Cells active</td>
<td>Cells active Continues GS and collagen production but fails to gain structure</td>
<td>Cells die, no protein production</td>
</tr>
<tr>
<td></td>
<td>Increased ground substance production</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Age/load</strong></td>
<td>Younger or short term load</td>
<td>Older and/or ongoing strain</td>
<td>Oldest and/or further strain</td>
</tr>
<tr>
<td><strong>Capacity to repair</strong></td>
<td>Full</td>
<td>Limited</td>
<td>None May progress to rupture</td>
</tr>
<tr>
<td><strong>Prevalence</strong></td>
<td>Common, not seen</td>
<td>Less common presents clinically</td>
<td>Uncommon, presents clinically</td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td>If extensive, very painful</td>
<td>Sometimes</td>
<td>Often grumbly</td>
</tr>
</tbody>
</table>
Clinical perspective
Imaging
Evidence for this model
Patellar tendon transition over one volleyball season

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Reactive (cell-PGs)</th>
<th>Degenerative (matrix-collagen)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Start of season</td>
<td>Normal</td>
<td>Reactive</td>
<td>Degenerative</td>
</tr>
<tr>
<td>Normal</td>
<td>226 .78</td>
<td>58 .2</td>
<td>5 .02</td>
</tr>
<tr>
<td>Reactive</td>
<td>35 .26</td>
<td>72 .54</td>
<td>26 .2</td>
</tr>
<tr>
<td>Degenerative</td>
<td>4 .02</td>
<td>25 .16</td>
<td>129 .82</td>
</tr>
<tr>
<td>End of season</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Malliaras et al in press BJSM
Typical presentations

- **Reactive**
  - Younger (15-25yrs)
  - Rapid onset generally related to load
  - Fusiform swelling of tendon 3-4cm
  - Aggravated by exercise

- **Tendon dysrepair**
  - Young adult (20-35yrs)
  - Past history with load related exacerbations
  - Fusiform swelling of tendon 3-4cm
  - Less irritable

- **Degenerative**
  - Older (30-60yrs)
  - Long history of symptoms
  - Variable swelling and lumps/bumps
  - Exhibit unloading strategies or atrophy
How should you treat the stages?

- **Reactive** tendons need to be calmed down and unloaded
  - How?
    - NOT eccentrics. decrease load
  - This is maybe how passive therapies work
    - They treat the right tendon with no treatment

- **Tendon dysrepair** needs to be stimulated to make good structure
  - How? Load probably eccentrics

- **Degenerative** tendons need to be stimulated to manufacture protein and structure matrix
  - Throw anything at it

Could have parts of a tendon that are in different phases
Well, what about pain?

- Pain in tendon pathology is difficult to explain
- A majority are not painful
- Why are some tendons painful?
  - Multiple possibilities
    - Neurovascular change
    - Cytokines
    - Cell driven?
Well, what about pain?

- Tendon pain may have several sources
  - Generated by nerves associated with vessels
    - Association is not clear or strong
  - Maybe by the tendon cells themselves
    - ? a factor in reactive tendinopathy
      - Pain only seems to be present when most of the tendon is involved
      - Young tendons, pranged tendons, remaining bits of degenerative tendons
  - Calming the cells may decrease the pain

Ohberg, Danielson, Alfredson
Linking load, pathology and pain - patellar tendon

- Normal
  - Loading not good for pathology: 25%
  - Supports the model

Pathology
- Pathology no pain: 4%
- Loading not detrimental for pain: 1%

Pathology & pain
- Loading good for pain: 21%
Can pain precede pathology?

- Early stage disease?
  - Incorrect diagnosis, minor disease, peritendon pathology?
- 26 players that had imaging normal tendinopathy at start of volleyball season
  - 17 men, 9 women
  - 33 tendons
    - 7 bilateral
  - Malliaras et al 06

- Became normal (lost pain)
  - 6 (18%)
- Stayed same
  - 11 (33%)
- Developed ultrasound changes
  - All proliferative in appearance, one hypoechoic
  - Without pain
    - 9 (27%)
  - With pain
    - 7 (21%)
How does load affect tendon?

- Positive
  - Without load tendons lose function
  - Tendon degradation in matrix and cell
  - Person changes
    - Functional and musculotendinous deterioration in the individual

- Negative
  - Overload leads to tendinopathy
    - Pathology and pain
      - Not necessarily together
  - More load, more prevalent
    - Young and past elite athletes
      (Kujala et al 07)
  - Aspects of load may be critical
    - Distance runners OR 31.2 (frequency)
    - Sprinters OR 14.9 (load)?
      (Kujala 05)
What types of tendon load are there?

- Tensile load thought to be the primary overload
- Compression is implicated in many tendinopathies
- Load may vary in different parts of the tendon
Is load homogeneous throughout the tendon?

- **Mid tendon**
  - Achilles is the only tendon to fail in the midsbstance
  - Not hypovascular
- **Tensile load**
- **Insertion**
  - Not commonly where tendon inserts, but just proximal to it
- **Compressive load**
- **Peritendon**
  - Posterior gliding membranes and anterior fat structures
    - Complex and multifunctional
    - Mechanoreceptive, nociceptive structures and macrophages Shaw et al 07
- **Friction**
What is high tensile load?

A Eccentric contraction
B Fast contraction
C High weights
D Stretch-shorten cycle
What is high tensile load?

- Any activity that requires the tendon to store and release energy
- Anything else is easy for a tendon
  - High weights, eccentric movement
How does tendon respond to load?

Collagen type-I formation in peritendon tissue
Effect of acute, prolonged exercise in healthy subjects


Slowly!!!!!!
Cell response to load

- Cell produces proteins to adapt ECM
  - Both collagen production and breakdown in early stages then more production
    - More than 4 weeks
      - Kjaer and associates
  - Indications that tendon size is responsive to load
    - Bigger tendons in athletes
      - Kongsgaard et al 06
    - ? Possible after puberty Smith 02
Quick and dirty response to load

- Back to the spring analogy
  - If a spring is stretched too much or too often, then it must be strengthened
    - In a tendon a thicker tendon is stronger
  - This can be a very quick response
    - Hyaluronon can be made in minutes and aggrecan in 2 days
    - After one bout of eccentric exercise in tendinopathy
      - Signal increased by 17%, volume by 31% after 30 minutes
      - Shalabi et al 2003
Clinical application

- High loads must be continued for extended periods for adaptation to occur
- Exercise prescription is critical
  - Frequency of high load
    - In normal and pathological tendons
    - HIGH TENDON LOAD
      - Every third day to start
      - Pathological tendons may never cope with twice daily exercise
Clinical perspective

- Loading the spring when it is stretched further is more damaging than loading it when it is shorter
- Tendons succumb to pathology when they repeatedly undergo stretch-shortening at length
  - Jumping athletes, not runners, get patellar tendinopathy
  - Hockey players and sprinters get hamstring tendinopathy
    - Not lawn bowls because length but no energy storage
  - Change of direction (soccer players) get adductor tendinopathy
    - Kicking in ARF
  - Court sports and runners get Achilles tendinopathy
Compression with tensile load

Force increased with load at length
  - Long thin springs are vulnerable to overload in the middle
    - Sustained in the midtendon
    - Shorter tendons may be more vulnerable at the insertions

Load at length also induces compression
  - Tendons insert into a depression after a bony elevation
    - Load at length will compress the tendon
Compressive load

- Enthesis adapts to the strains on the tissue
  - The bone, bursa and cartilage changes are dependent on
    - The inherent characteristics
    - Loading history

- Primarily compressive pathology proximal to the insertion
Decompressing the Achilles insertion

- Only 30% improved with standard Achilles program
- 27 participants (34 tendons)
  - Chronic Achilles insertional tendinopathy
    - Mean 26 months
- Eccentric program to flat
  - I.e. eccentrics with reduced compression
  - VAS 72 at baseline

- VAS decreased to 21
- 19 participants (23 tendons satisfied)
  - 70% of group improved
- 9 (11 tendons not satisfied)
  - VAS significantly improved (58) but not back to previous levels of activity
- Outcome not influenced by bone spurs, Haglund's or bursal pathology
  - Jonnson et al 08
Does compression explain some mid tendon Achilles tendinopathy?

- Loading a spring causes thinning in the midtendon, therefore internal compression
- Midtendon is made up of fibres from soleus and gastroc
  - Internal interface between soleus and gastrocnemius Bojsen
    – Moller et al 2004
Compression in Achilles tendinopathy

- The posterior retinaculum that prevents bowstringing in plantar flexion may compress the tendon in this position
- Sedentary people may spend time in plantar flexion
- 44% of sedentary people failed to improve with eccentric (tensile) loading
  (Sayana et al. 07)
What about supraspinatus?

- Why does it commonly have a tendinopathy?
  - No spring like energy storage
    - Other larger tendons take this role
    - More stabiliser
  - Likely compressed
    - Anatomically from winding over the tuberosity
    - Pathologically from the decrease in acromio-humeral distance
- That’s why scapular retraining works
  - Alters compressive loads
- Anatomical compressive loads known in tib post
What about unloading?

- Rest is clearly catabolic for connective tissue
  - No stimulus for protein production or for structure
  - No maintenance of muscle tendon unit or musculoskeletal capacity
Critical time for tendons

- After period of decreased load (hence a rapid increase in load)
  - Injury of any sort
  - Off-season
  - Load tolerance of the muscle-tendon unit is decreased
  - Most symptoms in athletes start at this time

- Clinical perspective
  - Ramp back after low load periods especially in those with known pathology
Summary

- Load is inherent for a tendon
  - Maintaining homeostasis
  - Becoming pathological
    - Different parts of the tendon respond differently
    - Type of load is critical
    - Quantity of load critical
  - Not sure what is most important