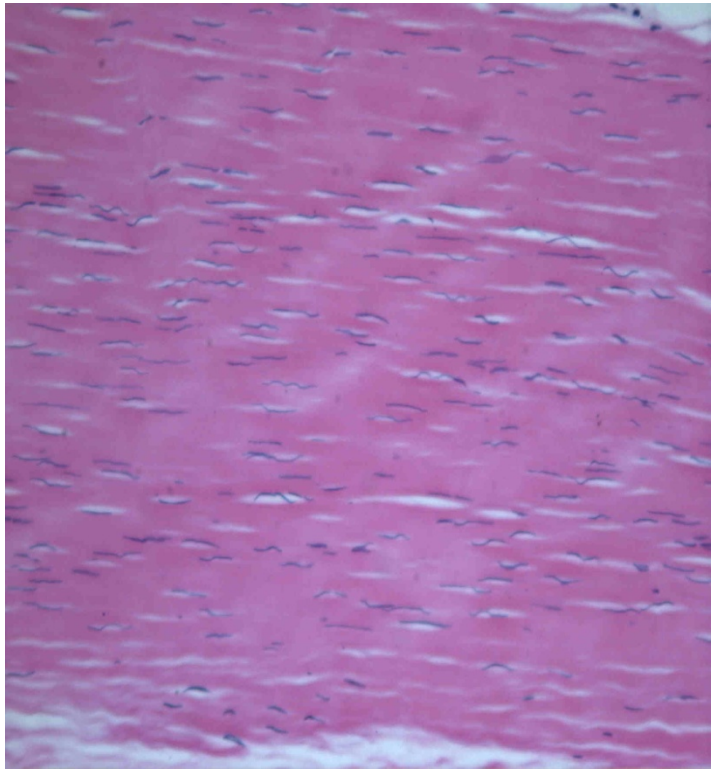
New insights into the mechanisms of tendon injury

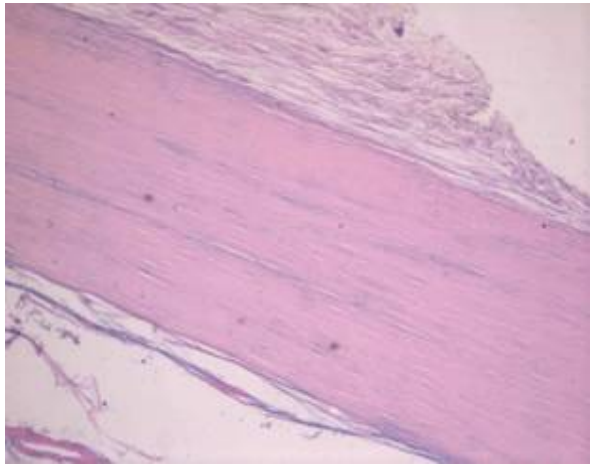
What is normal tendon?

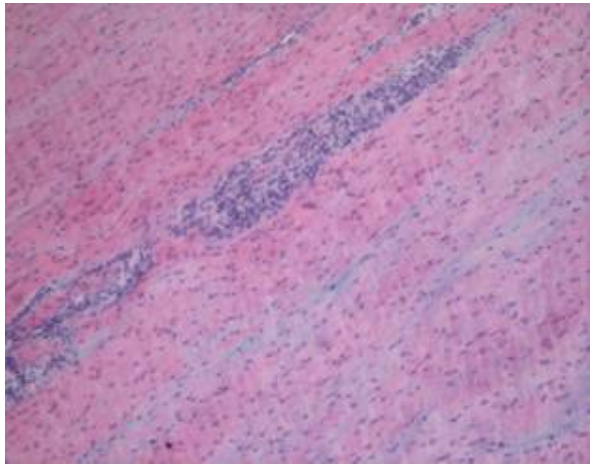
- A extracellular matrix tissue
  - Type 1 collagen
- Small amounts of CRITICAL proteins
  - Ground substance
    - Proteoglycans, glycoproteins
- Structures that make the tendon a living structure
  - Cells, nerves, vessels



Tendon pathology

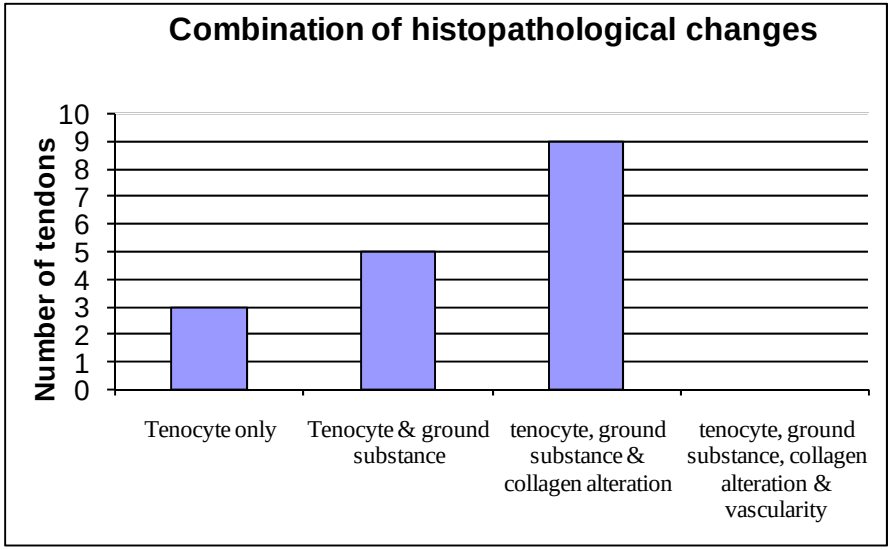
- Metaplastic change to **fibrocartilage**
  - Cell proliferation
    - Rounder
  - Ground substance increase
    - Larger (compressive) proteoglycans
  - Collagen degradation
    - Loss of Type 1
  - Neurovascular proliferation



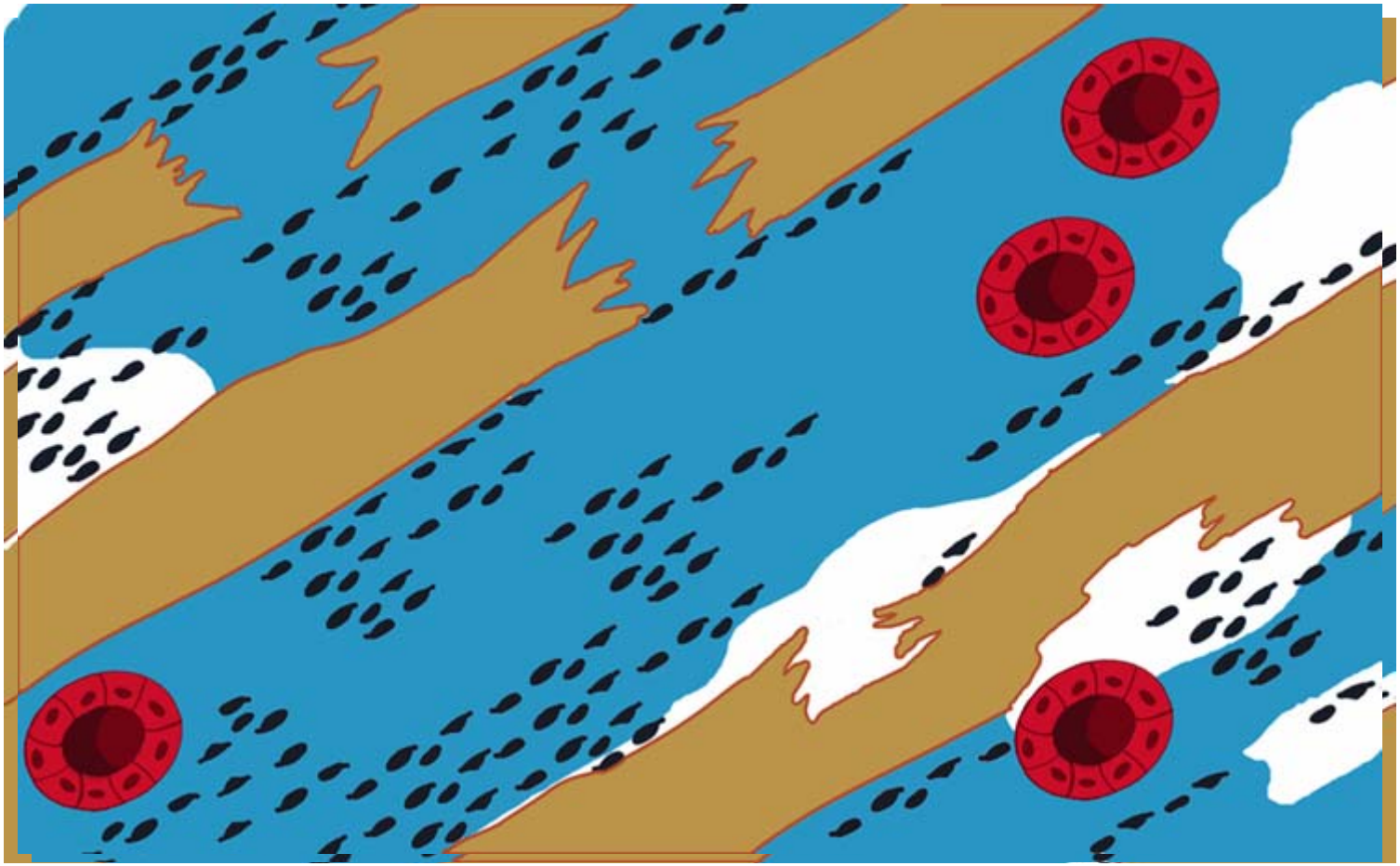


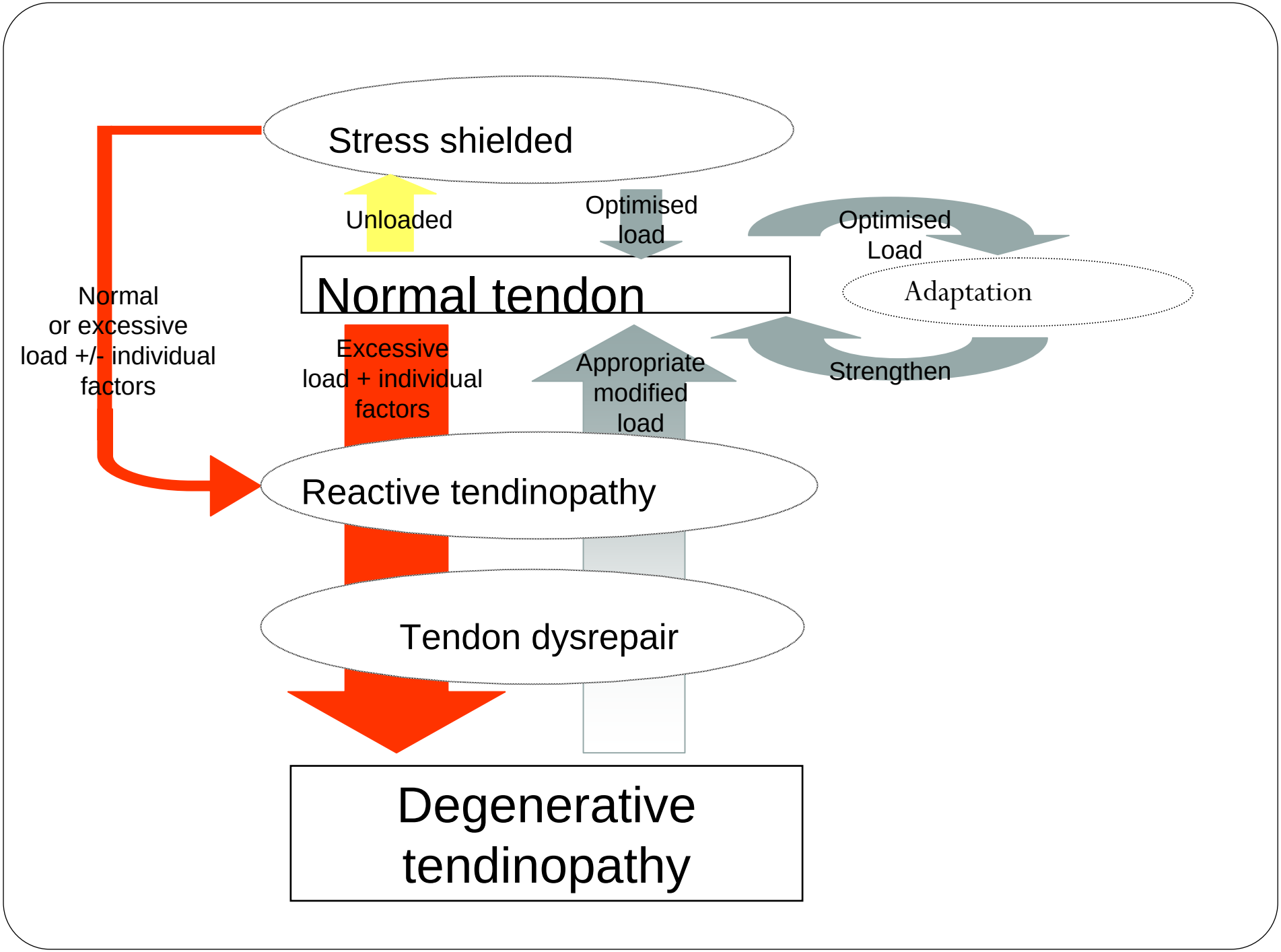
What happens first in tendinopathy?

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



How does pathology develop?



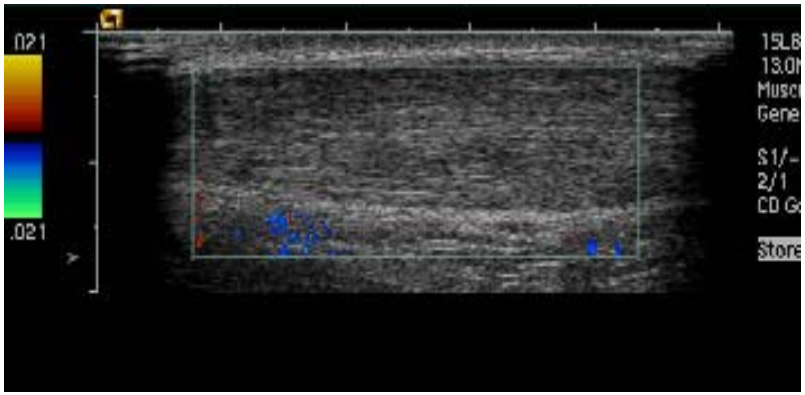

Stress shielded
Unloaded
Optimised load
Optimised Load
Adaptation
Normal tendon
Normal or excessive load +/- individual factors
Excessive load + individual factors
Appropriate modified load
Strengthen
Reactive tendinopathy
Tendon dysrepair
Degenerative tendinopathy

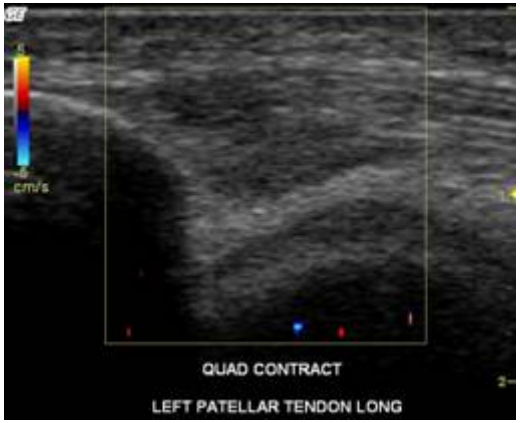
Model of tendinopathy

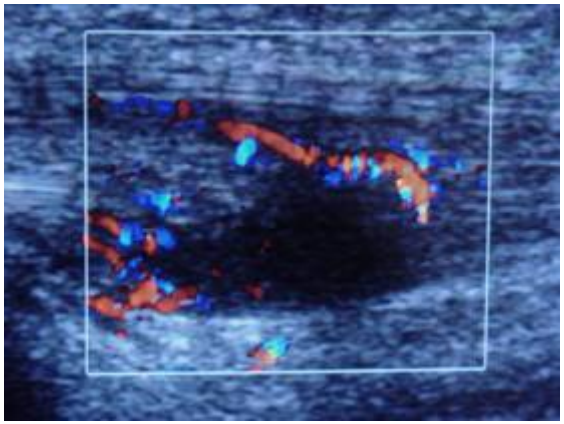
	Reactive	**Tendon dysrepair**	**Degenerative**
Tendon response	Adapting to load	Attempting to heal	Gives up on healing
Pathology	Cells active Increased ground substance production	Cells active Continues GS and collagen production but fails to gain structure	Cells die, no protein production
Age/load	Younger or short term load	Older and/or ongoing strain	Oldest and/or further strain
Capacity to repair	Full	Limited	None May progress to rupture
Prevalence	Common, not seen	Less common presents clinically	Uncommon, presents clinically
Pain	If extensive, very painful	Sometimes	Often grumbly

Clinical perspective

Imaging







Evidence for this model

Patellar tendon transition over one volleyball season

Start of season

	Normal	Reactive (cell-PGs)	Degenerative (matrix-collagen)
Normal	226 .78	58 .2	5 .02
Reactive	35 .26	72 .54	26 .2
Degenerative	4 .02	25 .16	129 .82

End of season

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

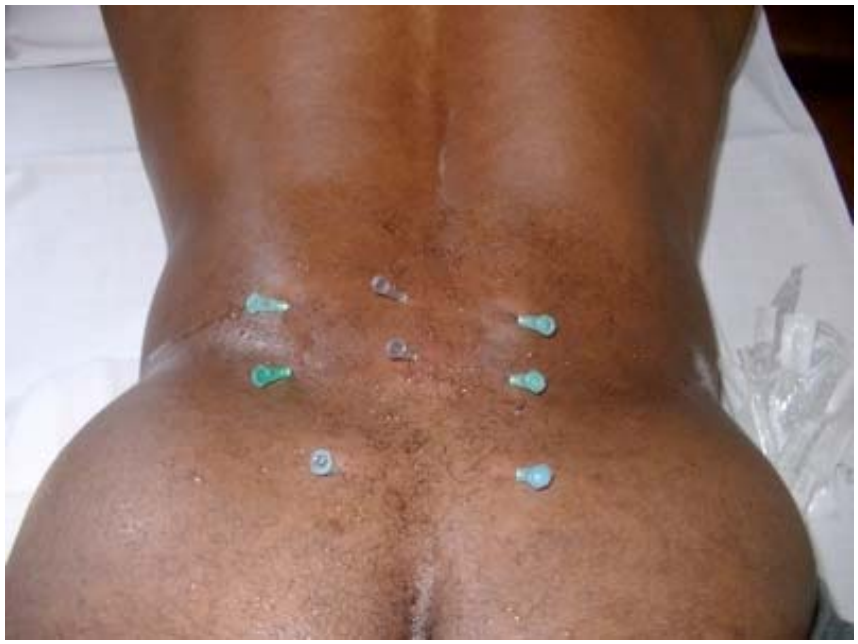
How should you treat the stages?

- **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?

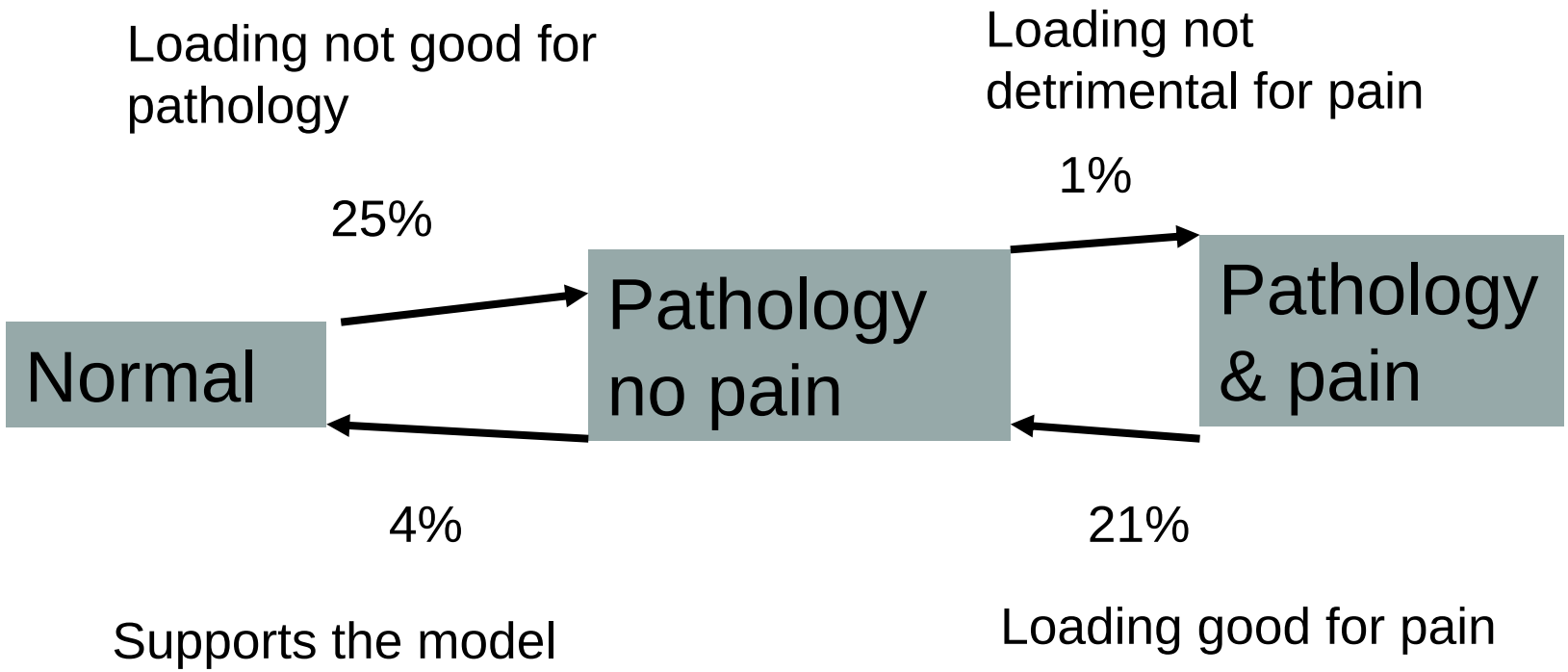


Well, what about pain?

- 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



Can pain preceed 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 midsubstance
  - 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

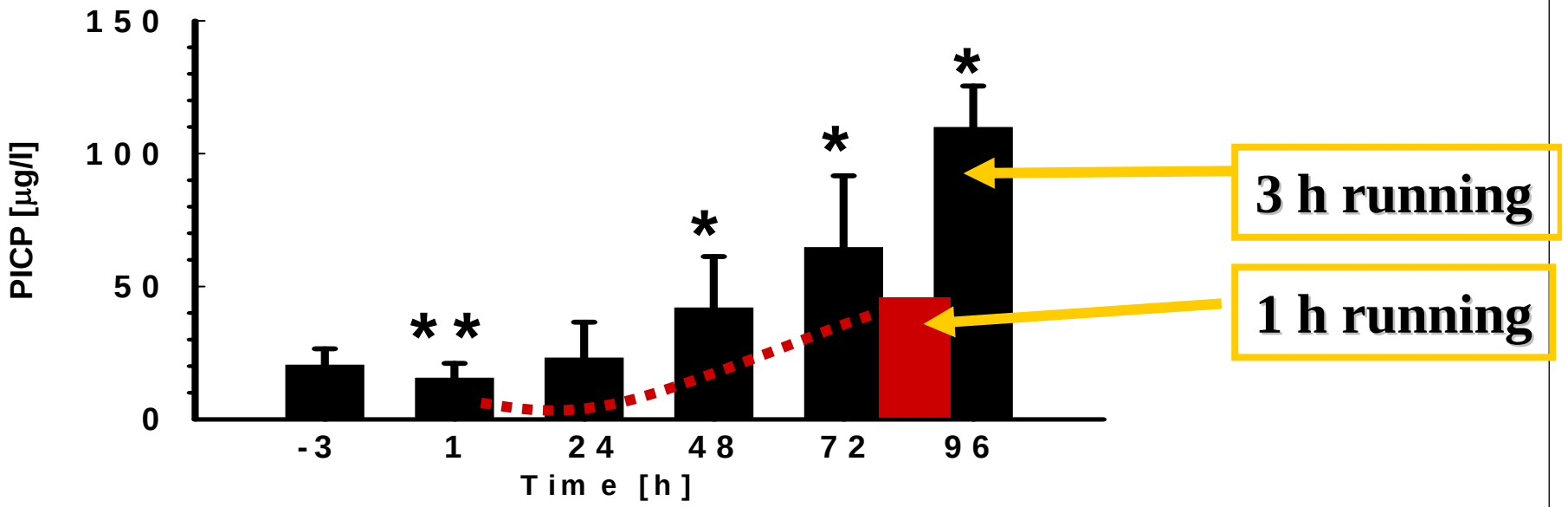
D Stretch-shorten cycle

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?

Slowly!!!!!



Collagen type-I formation in peritendon tissue

Effect of acute, prolonged exercise in healthy subjects

Langberg et al. J.Physiol.521:299-306,1999, Heinemeier et al.JAP,2003

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

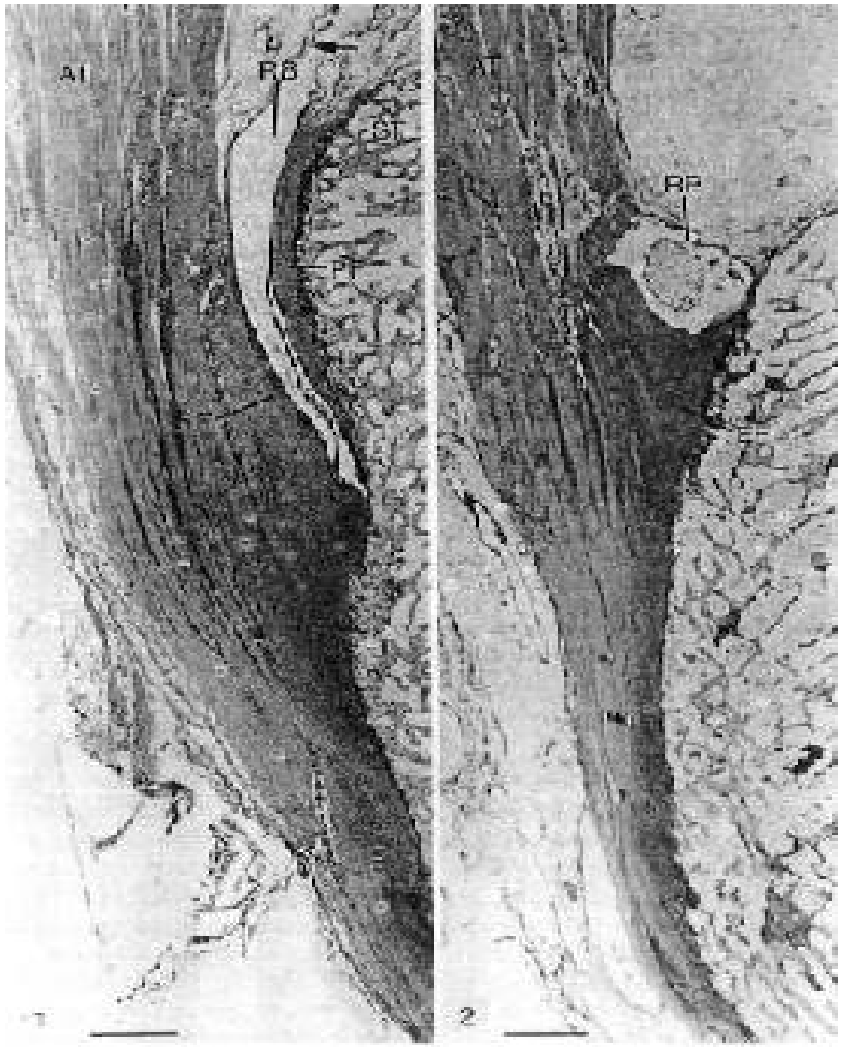
- Long thin springs are vulnerable to overload in the middle
  - Sustained in the midtendon
  - Shorter tendons may be more vulnerable at the insertions

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

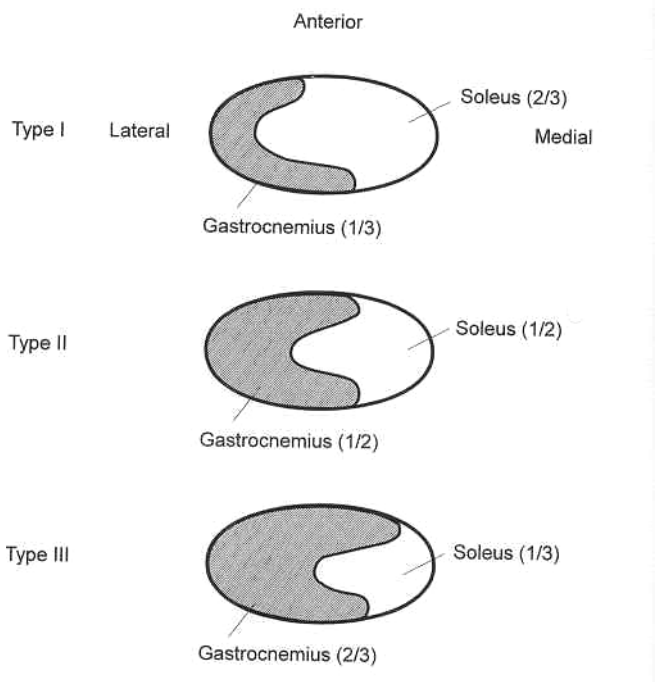


Decompressing the Achilles insertion

- 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*

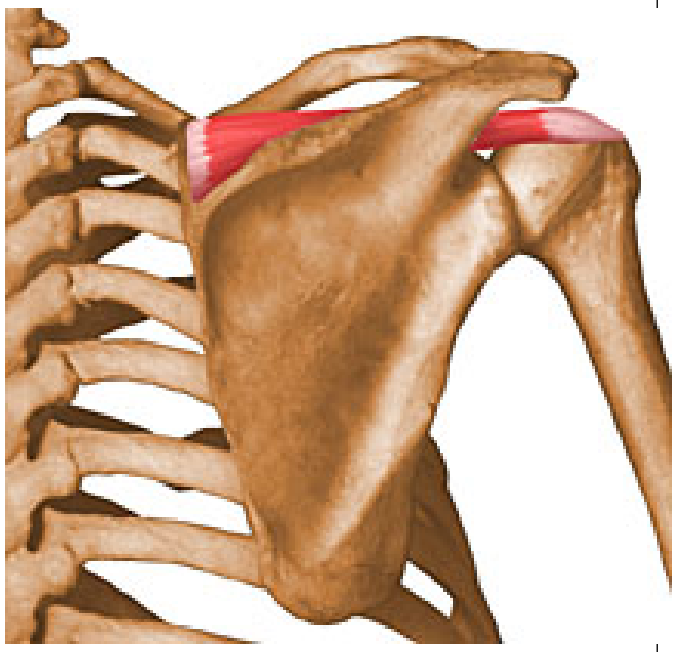


Compression in Achilles tendinopathy

- 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 impotrant