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‘TRANSFORMING EDUCATION, TRANSFORMING LIVES…..’

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Learning Outcomes

- Describe types of tendon and ligament injury
- Discuss tendon and ligament healing including timescales and timeframes that facilitate appropriate healing
- Discuss their tolerances to loading

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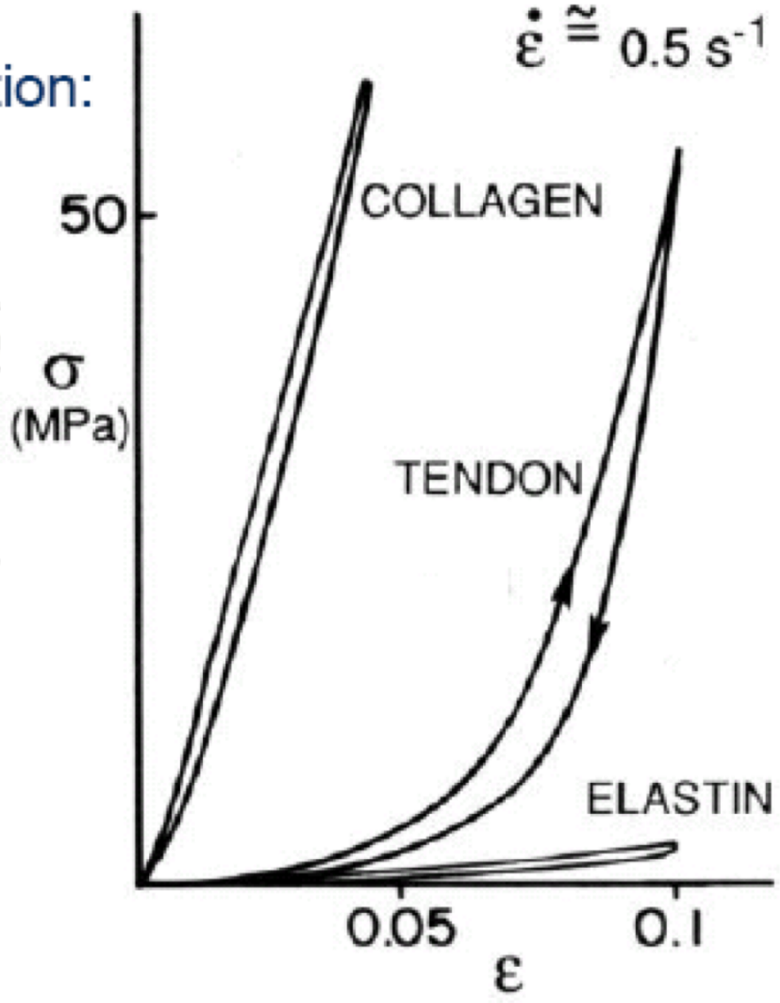
Tendon and Ligament Mechanics

Tendons and ligaments have similar composition:

- Mainly made of collagen

Component	Ligament	Tendon
Collagen (mostly as type I)	70–80	75–85
Elastin	10–15	<3
Proteoglycans	1–3	1–2

Different arrangement of the fibers



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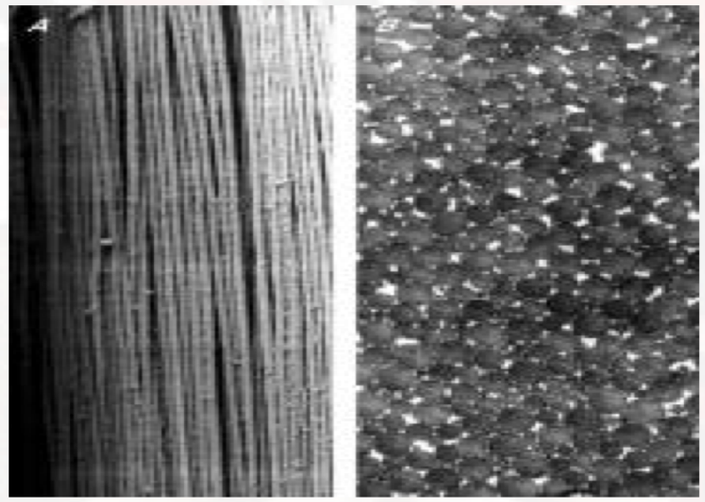
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Tendon and Ligament Alignment



Wavy



Patella:Tendon

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Ligament

- Attach Bone to Bone
- Sprained
- 3 Grades
  - Grade I (Pain no laxity)
  - Grade 2 (Pain & laxity)
  - Grade 3 (No Pain & lax+++)

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Ligament Damage and Failure

1 Reversible strain (physiological loading):

- Fibers progressively stretched and recruited
- Slope is increasing
- Tends to become linear

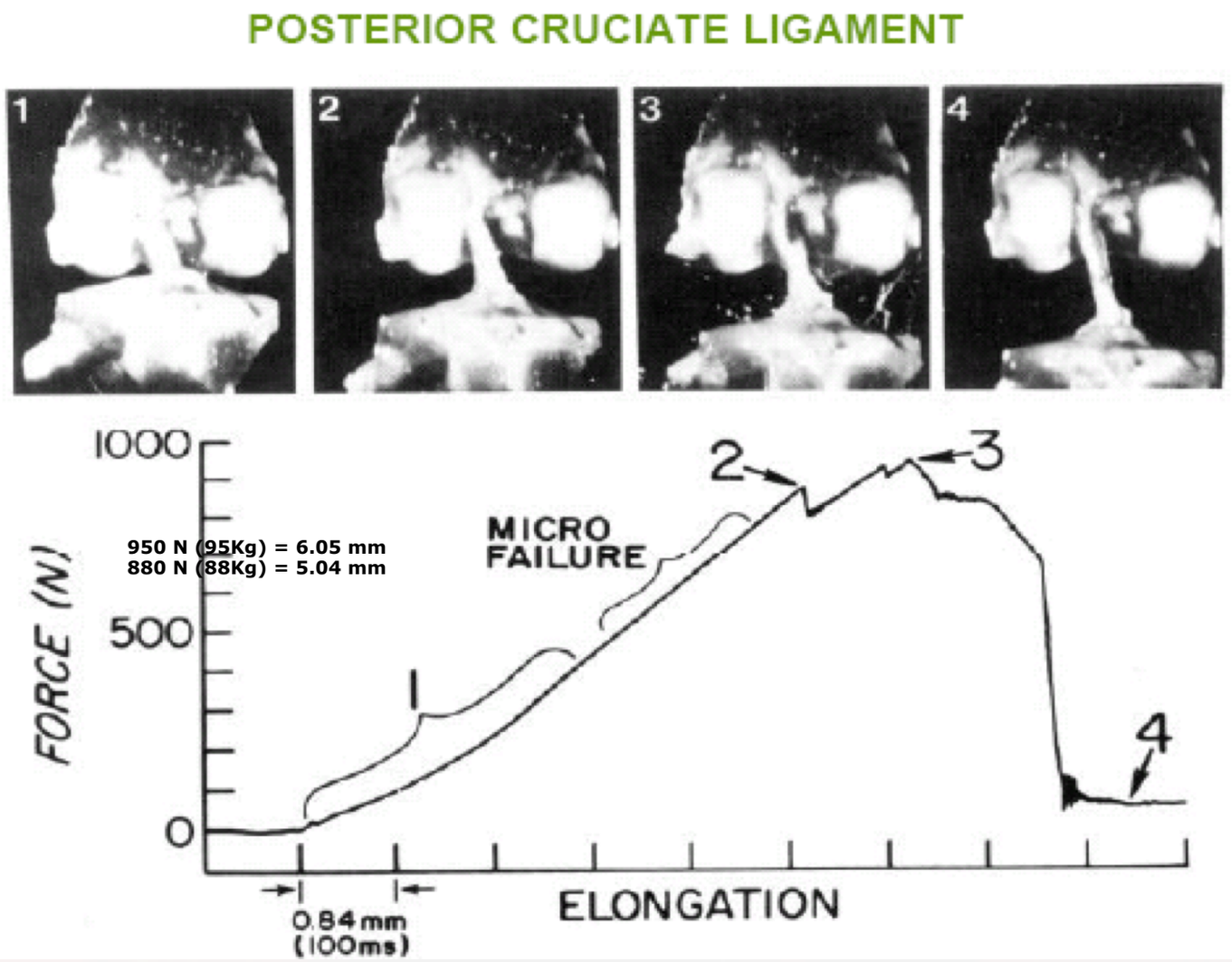
2 First fibers fail:

- First damage when curve still linear
- Load fluctuates

3 Peak load: most fibers stretched & ready to fail

4 Residual strength after damage:

- Ligament length is irreversibly increased (joint is loose)



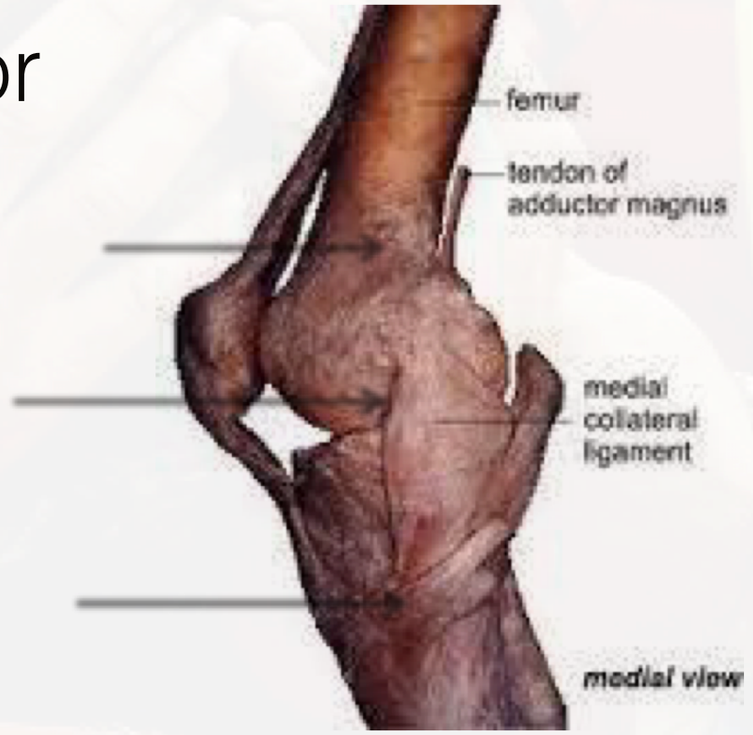
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Ligament Failure

- Speed of force determines where the ligament fails
  - High speed: Bone ligament interface
  - Low speed: Mid-belly (core or ligament)



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Use v Disuse

- Ligaments:
  - Increased structural strength and stiffness
  - ADL enough to maintain 80-90% potential strength/mechanical potential (Frank, 1996).
  - Exercise increases ligament strength and stiffness by up to an additional 10-20%

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Ligament Healing

- Ligaments:
  - When ligament tears, frayed stump ends are present
  - Ligament undergoes inflammatory process
  - Ligament stumps become surrounded by fluid, causing ends to become friable (easily crumbled)
  - Vascular permeability increases and permits normal inflammatory products to invade
  - Erythrocytes invade gap between stump ends

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Ligament Healing

Time	Activity
< 6hrs	Injury site fill with erythrocytes, leukocytes and lymphocytes. Ligament stumps become progressively more friable with the accumulation of serous fluids in the area
24/24	Monocytes and macrophages infiltrate the area . Fibroblasts begin to appear and become significant in number
48-72/24	Fibroblast produce the extracellular matrix
1-2/52	Fibrocytes and macrophages are numerous Random collagen fibres and abundant ground substance are seen. Fragile vascular granulation is seen Extracellular matrix continues to be synthesised by fibroblasts Macrophages, mast cells and fibroblasts continue to predominate. Vascular buds appear in the wound to communicate with existing capillaries Elastin is seen

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Ligament Healing

Time	Activity
2/7 to 6/52	Proliferation phase occurs Cellular and matrix structures replace the blood clot formed during inflammation
6/52 to 12/12	Macrophages and fibroblasts diminish
< 12/12	Collagen concentration stabilises with type I collagen replacing type III Collagen cross link increase in number Ligament becomes more normal
40-50/52	Near normal tensile strength restored

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Ligament Immobilisation

- Ligaments:
  - Major deterioration in first few weeks
  - Ligament cells produce inferior quality ligament material
  - Structural weakening at ligament complex
  - Osteoclast at bone-ligament that decreases the strength of insertional bone (concurrently)

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Tendons

- Types of Tendon Issue:
  - Strain (partial or rupture)
  - Tendinopathy (Overuse)

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Tendons

- USE v DISUSE:
  - Few good studies available
  - Increased number and size of collagen fibrils
  - Increased cross-sectional area
  - Exercise leads to increased collagen synthesis in growing tendons (Curwin et al,1988)
  - Exercise leads to increased number of fibroblasts in tendons (Zamora & Marini,1988)

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Acute Tendon Healing

Time	Activity
0-3/7	Cells that originate from extrinsic peri-tendinous tissue and from intrinsic tissue from the epitenon and endotenon are active
5/7	Wound gap is filled by phagocytes
1/52	Collagen synthesis is initiated , new collagen fibres placed in random and disorganised orientation
10/7	Collagen synthesis peaks
3/52	Endotenon provides significant fibroblast proliferation at the site. Significant revascularisation occurs. Synovial sheath is rebuilt: smooth gliding surface develops. Fibroblasts also start to become orientated in line with tendon axis
4/52	Fibroblasts predominate in the healing area. Collagen content increases. Collagen fully orientated with tendon axis

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Acute Tendon Healing

Time	Activity
5/52	Collagen synthesis is complete
6/52	Fibroblasts that have proliferated endotenon are the primary cells: simultaneously synthesising collagen whilst contributing to collagen re-absorption
2/12	Collagen is mature and re-aligned along the tendons axis
4/12	Fibroblasts have reverted to tenocytes , Type III collagen has been replaced by Type I Maturation is complete
40-50/52	Strength is 85-95% normal

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Tendon Terminology

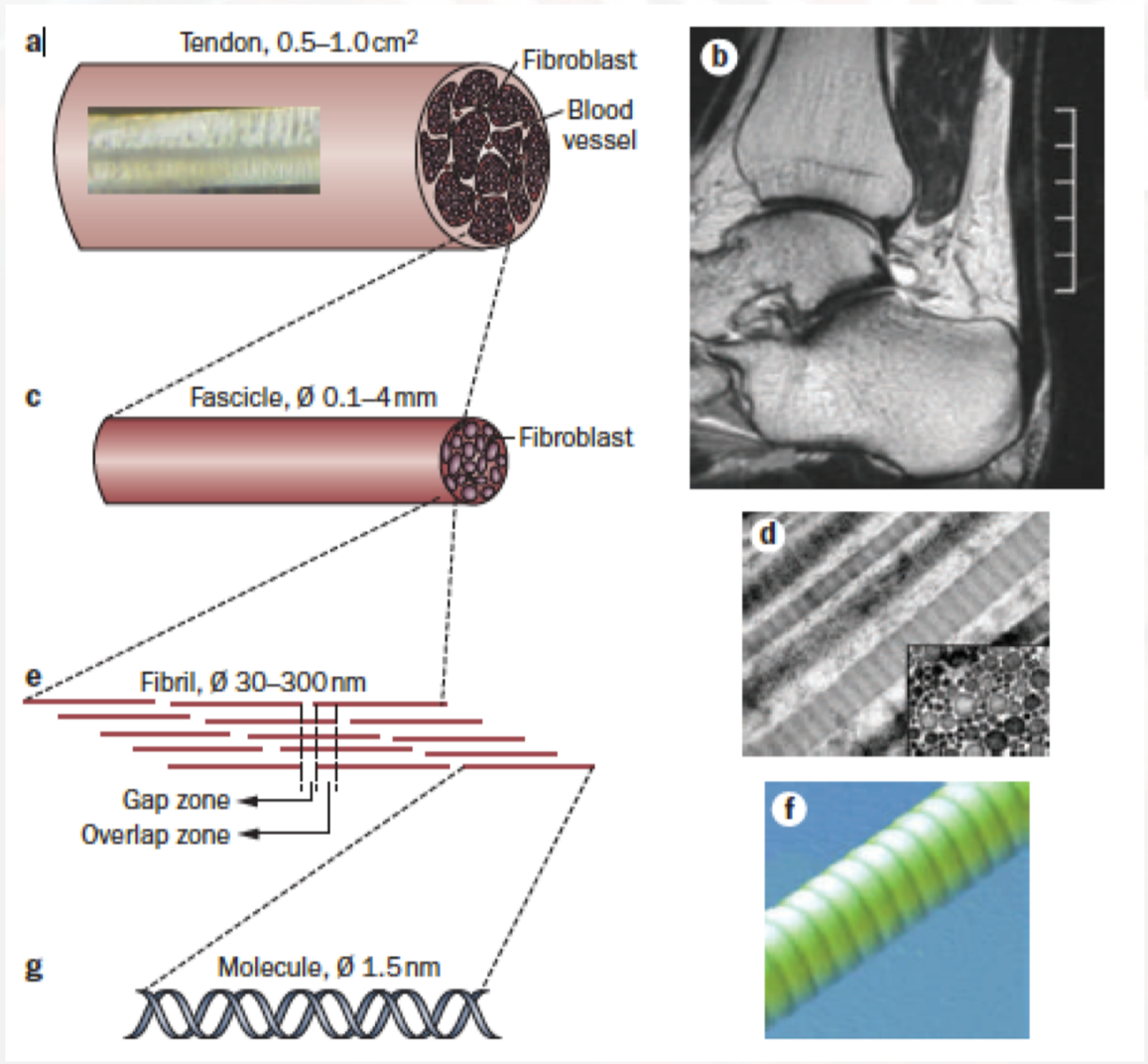
- **Tendinopathy** – condition associated with the tendon
- *Tendinosis* - degenerative
- *Tendinitis* - inflammatory
- *Tenosynovitis* – inflammation of the synovium around the tendon

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Tendon Structure

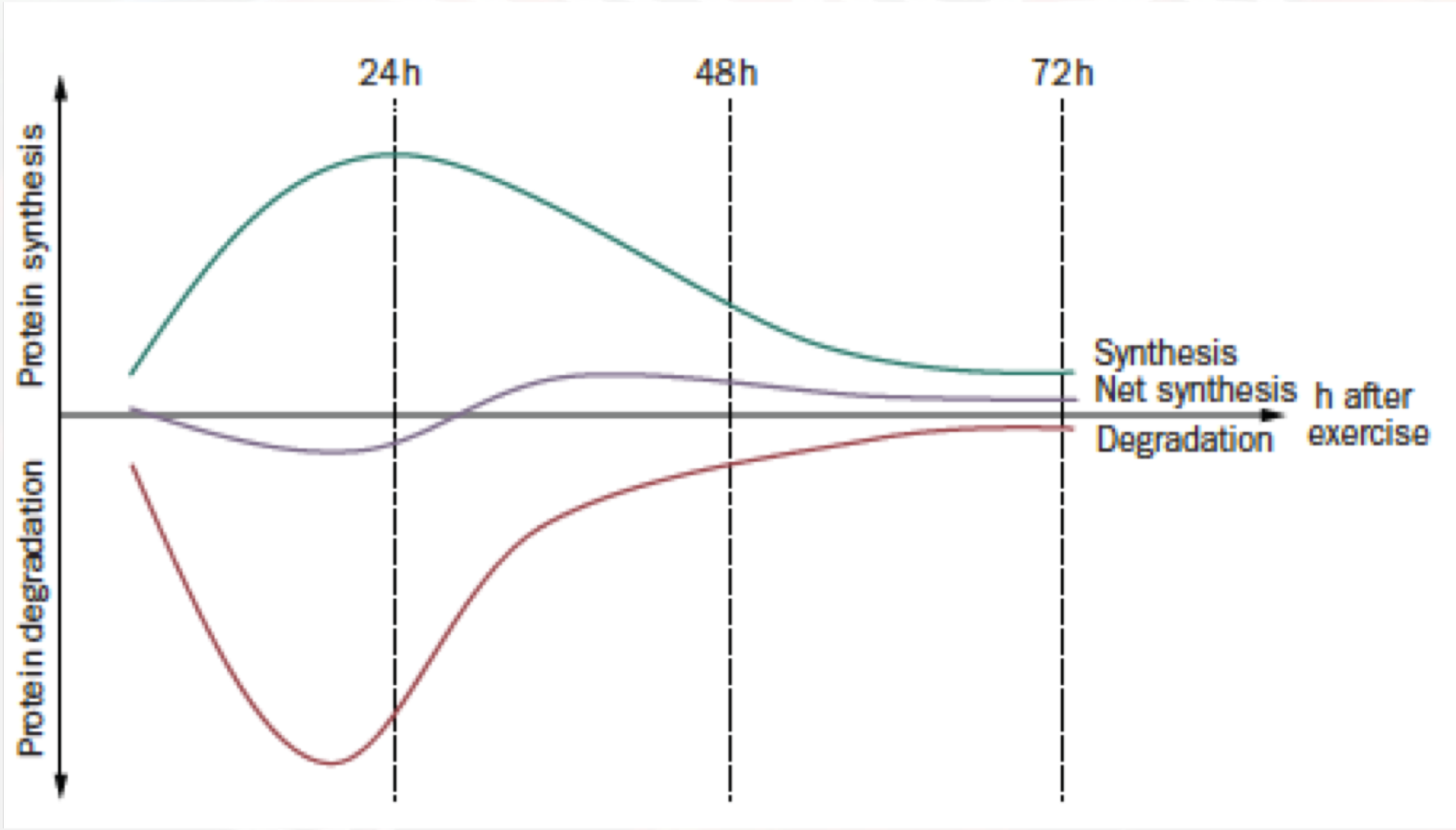


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Tendon Concepts - Normal



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Tendon Concepts - Abnormal

- Reactive tendon
- Tendon dysrepair
- Degenerative tendinopathy

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Tendon Concepts – Reactive tendinopathy

- Non inflammatory
- Acute Tensile/ Compressive Force
- Localised thickening at point of stress
- Increase CSA in response to load
- Normal tendon increases stiffness not CSA
- Able to return to normal state after deload
- Increased cytoplasmic organelles for protein production
- Large proteoglycans
- Matrix changes due to water uptake
- Collagen integrity maintained
- No neurovascular changes
- Minutes – few days

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Tendon Concepts – Dysrepair tendinopathy

- Attempt at tendon healing
- Similar to ReactiveT but with increased cell breakdown
- Increased cells numbers (Chrondrocytes and Myofibroblasts)
- Separation of collagen and disorganisation of matrix
- Increased ground substance
- Some may increase in neovascularization and neurals

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Tendon Concepts – Degenerative tendinopathy

- Areas of cell death
- Areas of acellularity
- Large areas of disordered matrix filled with vessels, matrix breakdown products and little collagen
- Considerable heterogeneity of the matrix
- Islands of degenerative pathology
- Interspersed with other areas of pathology and normal tendon
- 97% Achilles tendons that rupture are degenerative

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Clinical Concepts – Where are they

- Subjective Ax is Key
- Duration of symptoms
- Age factor
- Single bout or repeated
- Investigations - ultrasound/MRI? Doppler?
- Questionnaires
- Objective Markers? (Squeeze/SL Calf Raise)

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