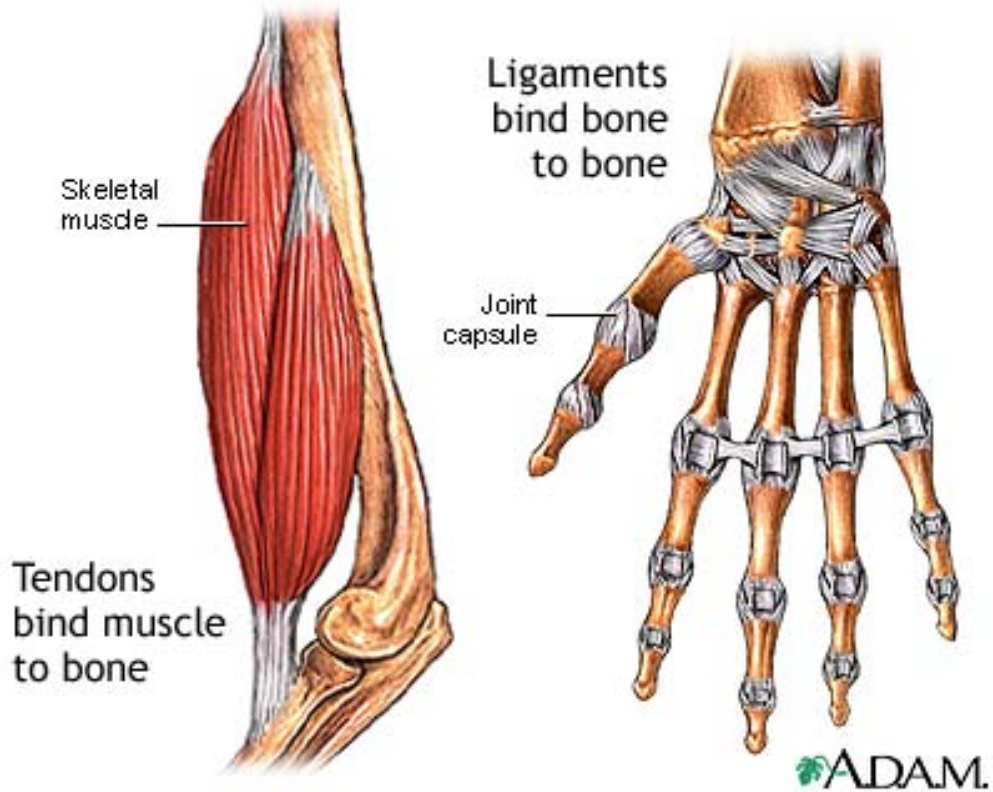
ME 482 Experimental Tissue Mechanics

Professor A. Wagoner Johnson

9/28 and 10/3/16: tendon and ligament – structure, composition, function, properties



http://www.nlm.nih.gov/medlineplus/ency/imagepages/19089.htm

Topics for tendon and ligament and corresponding sources. There is some overlap between sources.

- **Nordin and Frenkel**
- **(topics between pp 103-115)**
- Tendon – ligament composition table and corresponding text
- Tendon vs ligament fiber alignment – how does it affect stress-strain curve?
- Mechanical behavior tendon and ligament
- Failure mechanisms/modes
- Factors that affect mechanical properties (ageing, pregnancy and post-partum, mobilization, immobilization)
- **Other**
- Fiber recruitment model.

Nigg and Hertzog

2.5: Ligament, 2.6: tendon

- Morphology and histology 107-109 (use Nordin and Frenkel for composition info)
- 2.5.2 physical properties
- 2.6.2 physical properties

Hawkins – ligament biomechanics

- Fig. 5.1 and corresponding text
- Biomechanics pp 127-134
  - Especially failure modes and strain rate
- Basic effects of ageing, exercise and disuse

Cowin

What is tendon (ligament)?

- Dense, tough, fibrous tissue
- Connects muscle to bone (**ligament connects bone to bone**)
- Many different sizes and shapes depends on
  - location in body
  - requirements of bone and muscle to which it is attached.
- Tendon originates in muscle and may cross one or more joints before inserting in to bone.
- Sheaths protect it from injury during joint motion

What are the mechanical functions of tendon?

- To transmit forces that are generated in the muscle across one or more joints in order to stabilize them or to produce motion
- To attach/connect muscle to the bone
- To store elastic strain energy during motion.

Physiological functions

- crimping pattern (shown later) may help to absorb shock, but not exactly known.

- sensing of relative motion and position of adjacent parts of the body
  - (contrast to sensing external stimuli like in smell).

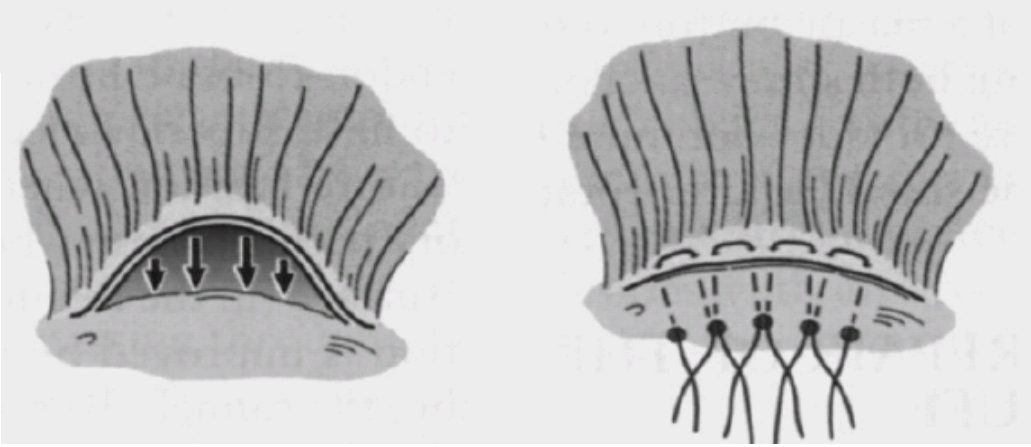
- Mechanoreceptors participate in a feedback system that changes muscle activity according to the resistance encountered.

Clinical significance of studying tendon (and ligament)

- Approximately 30% of work related injuries in the United States each year are to the upper extremity

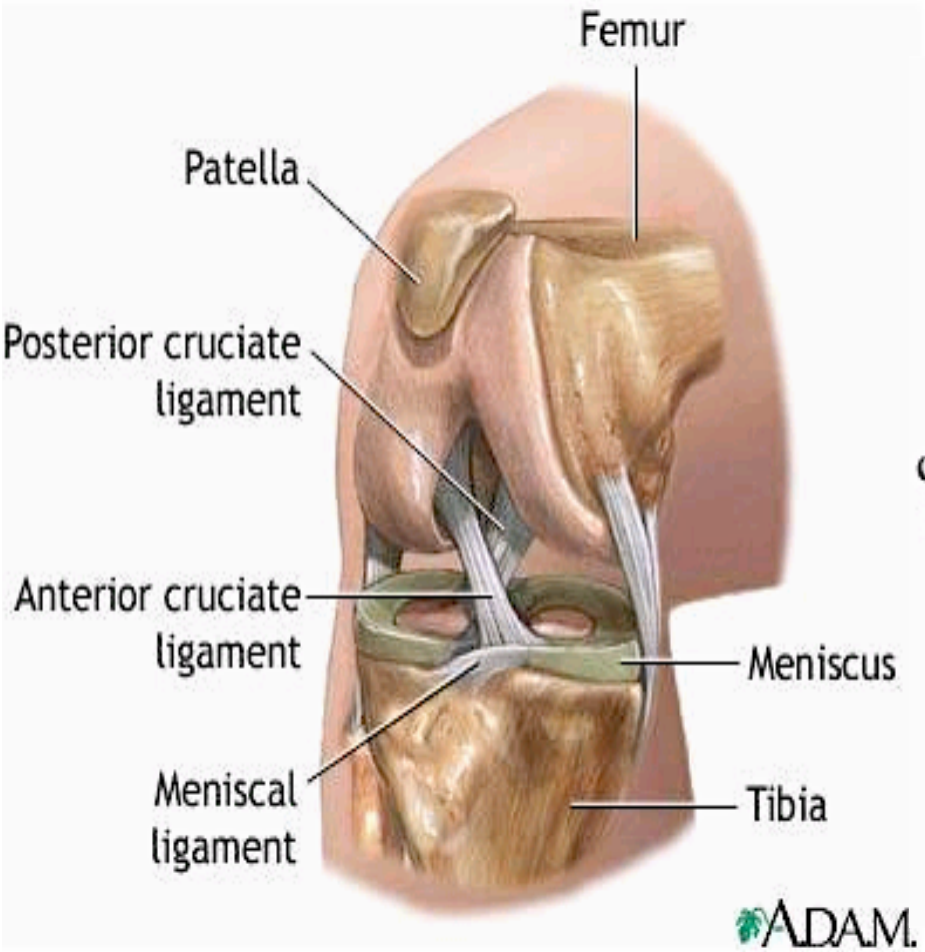
- Rotator cuff injuries of the shoulder account for a significant portion of the soft tissue injuries in the upper extremity

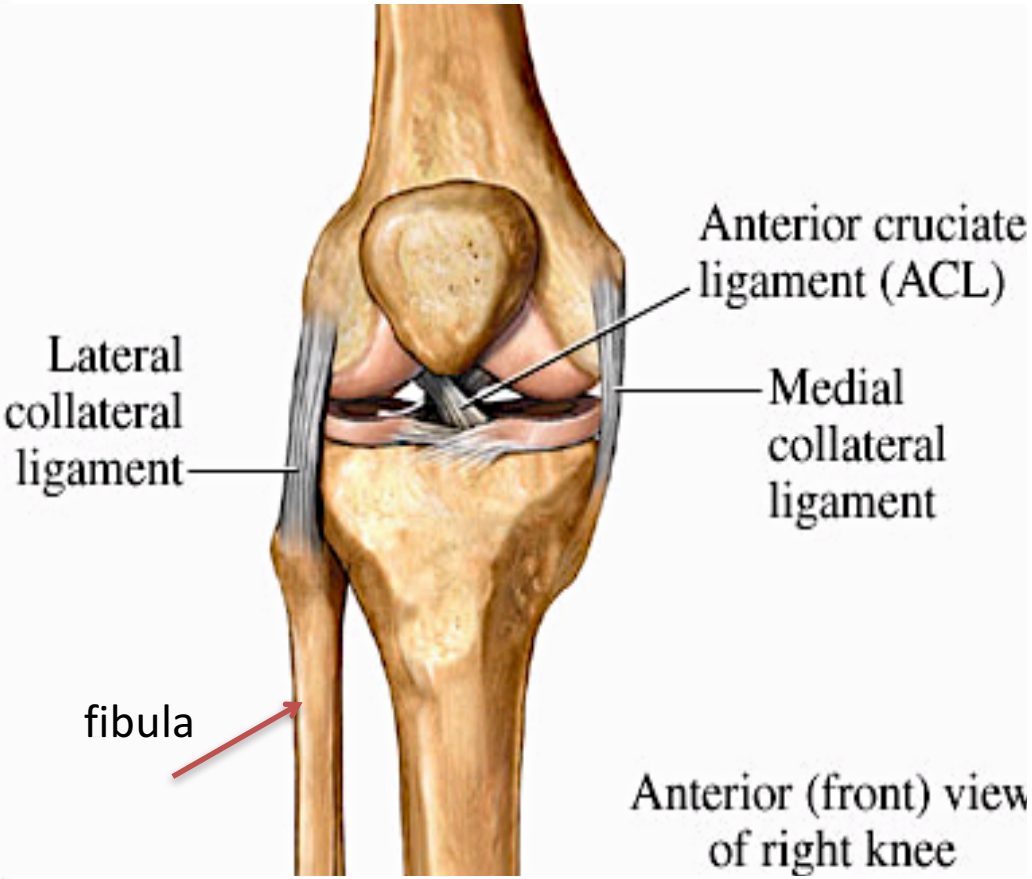
- At the rotator cuff, tendon to bone repair techniques have been plagued by a high rate of recurrent tears (up to 94%)



Courtesy Prof Guy Genin, Wash U.

Ligaments of the knee



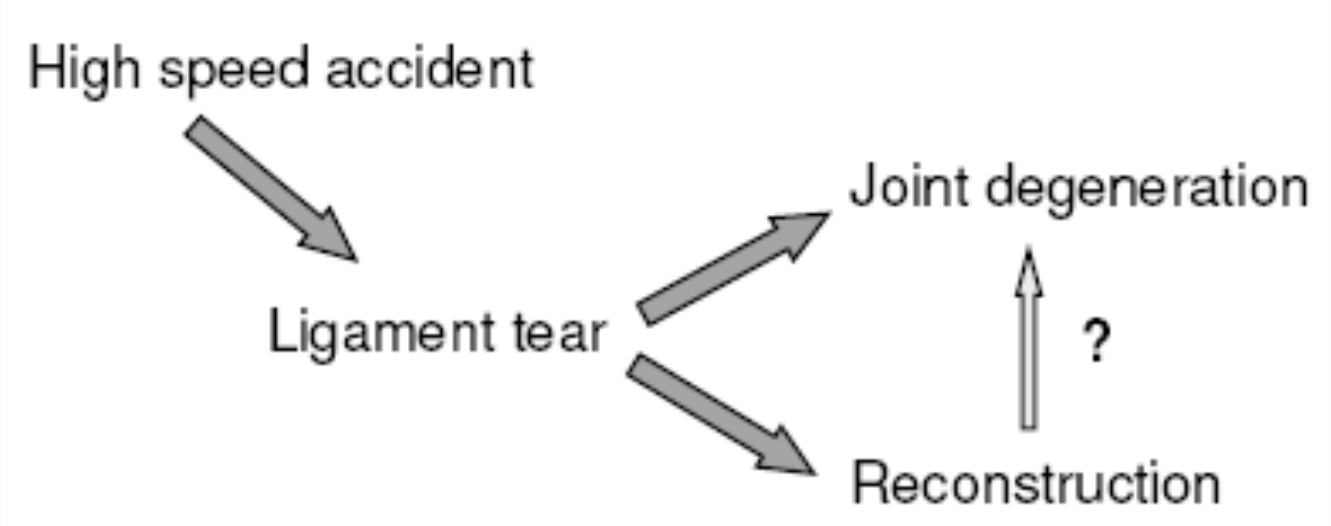


http://0.tqn.com/d/orthopedics/1/0/4/6/anatomy.jpg

http://blogs.pe.com/prosports/MCL%20image.jpg

Why are we interested in mechanical properties of ligaments, like ACL, and tendon??

- An ACL injury will not heal on it's own
- 75,000 to 100,000 ACL reconstructions per year
- Long term follow-up:
  - 25% unsatisfactory
  - 80% incidence of knee degeneration (arthritis) 30 years after ACL tear



Women have higher incidence of ACL injury for same activities

www.orthojournalhms.org/volume2/html/articles22.htm

TABLE 1 Risk factor for knee injury in female athletes

Risk Factor for Knee Injury	Reference
Female Gender	
NCAA injury rates differ, female > male	Arendt et al.[13]
High school injury rates differ, female > male	Chandy et al.[15]
	Powell et al.[14]
	Zillmer et al.[16]
Professional basketball injury rates differ, female > male	Zelisko et al.[17]
Sex Hormones	
Injury rate differs by menstrual cycle phase	Myklebust et al.[23]
	Wojtys et al.[24]
	Slauterbeck et al.[25]
No variation in ligament laxity in different phases	Belanger et al.[29]
Oral contraceptive use offered no injury protection	Arendt et al.[26]
Anatomic Considerations	
Q angle higher in injured female athletes	Shambaugh et al.[31]
Static postural faults correlated with higher injury rate	Loudon et al.[33]
Femoral notch parameters associated with injury (consensus paper)	Griffin et al.[20]
Notch size associated with increased injury risk	Shelbourne et al.[41]
Notch size not associated with increased injury risk	Schickendantz et al.[42]
	Teitz et al.[43]
Notch shape associated with increased injury risk	Ireland[44]
Neuromuscular Imbalances	
Proprioception deficits in ACL-deficient or ACL-injured	Barrack et al.[45]
	Wojtys et al.[46]
	Barrett et al.[47]
	Corrigan et al.[48]
Single leg stance deficits females > males	Hewett et al.[51,52]
Jumping and landing strategies differ female *vs.* male	Schultz et al.[53]
	Rozzi et al.[54]
Jump landing did not differ female *vs.* male	Fagenbaum et al.[64]
Females with greater valgus dominant knee	Ford et al.[57]
Females with highly dominant dominant leg	Hewett et al.[59]
Preseason strength and conditioning deficits associated with injury	Knapik et al.[66]
Preseason conditioning program associated with reduced injury	Hewett et al.[68]
	Caraffa et al.[69]
Quadriceps-dominant pattern in female athletes	Huston et al.[36]
Quadriceps-dominant pattern in elite athletes	Baratta et al.[63]
Eccentric muscle fatigue in female athletes	Nyland et al.[70]
Stiffness/elasticity differences females *vs.* males	Winter et al.[74]

Dugan, AS. Am J Phys Med Rehabil. 2005 Feb;84(2):122-30.

Tendon and Ligament Composition – why do ligaments have more PGs? (maybe more apparent in later slides)

Table 4.1 Structural Composition of Tendons and Ligaments		
Component	*ligament*	*tendon*
Cellular material (fibroblast)	20%	20%
Extracellular Matrix	80%	80%
water	60-80%	60-80%
solids	20-40%	20-40%
Collagen	70-80%	Slightly higher
Type 1	90%	90-95%
Type 3	10%	1-5%
Ground substance	20-30%	Slightly lesser

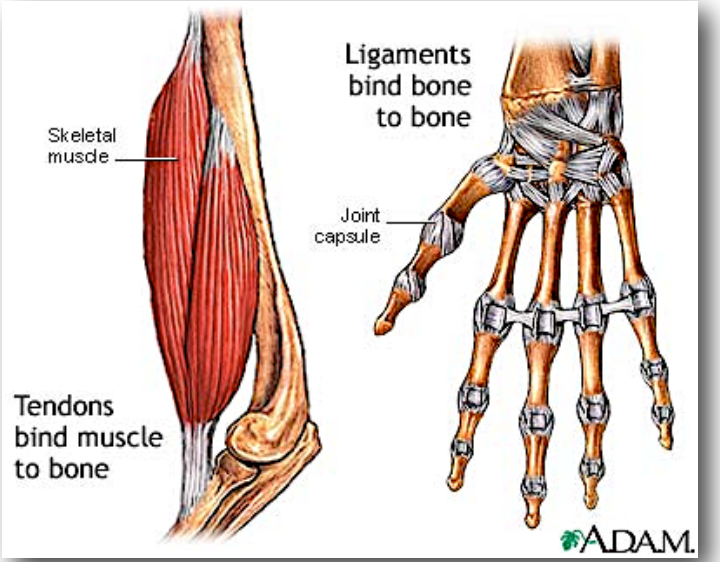


TABLE 4-1

Structural Composition of Tendons and Ligaments

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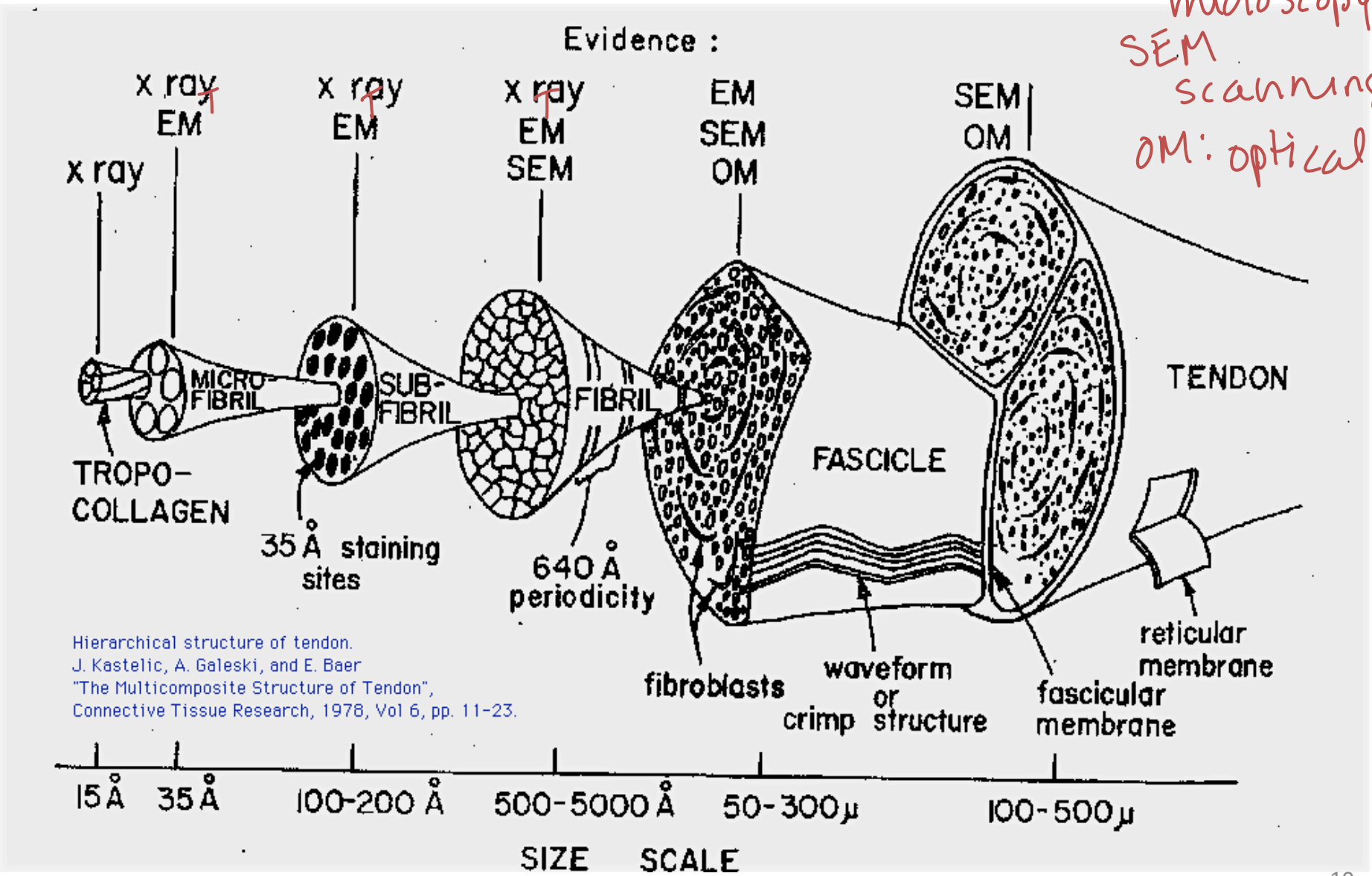
Collagen:	70–80%	slightly higher
Type 1	90%	95–99%
Type 3	10%	1–5%
Ground substance	20–30%	slightly lesser

Nordin and Frenkel

Tendon structure and *lengthscales*

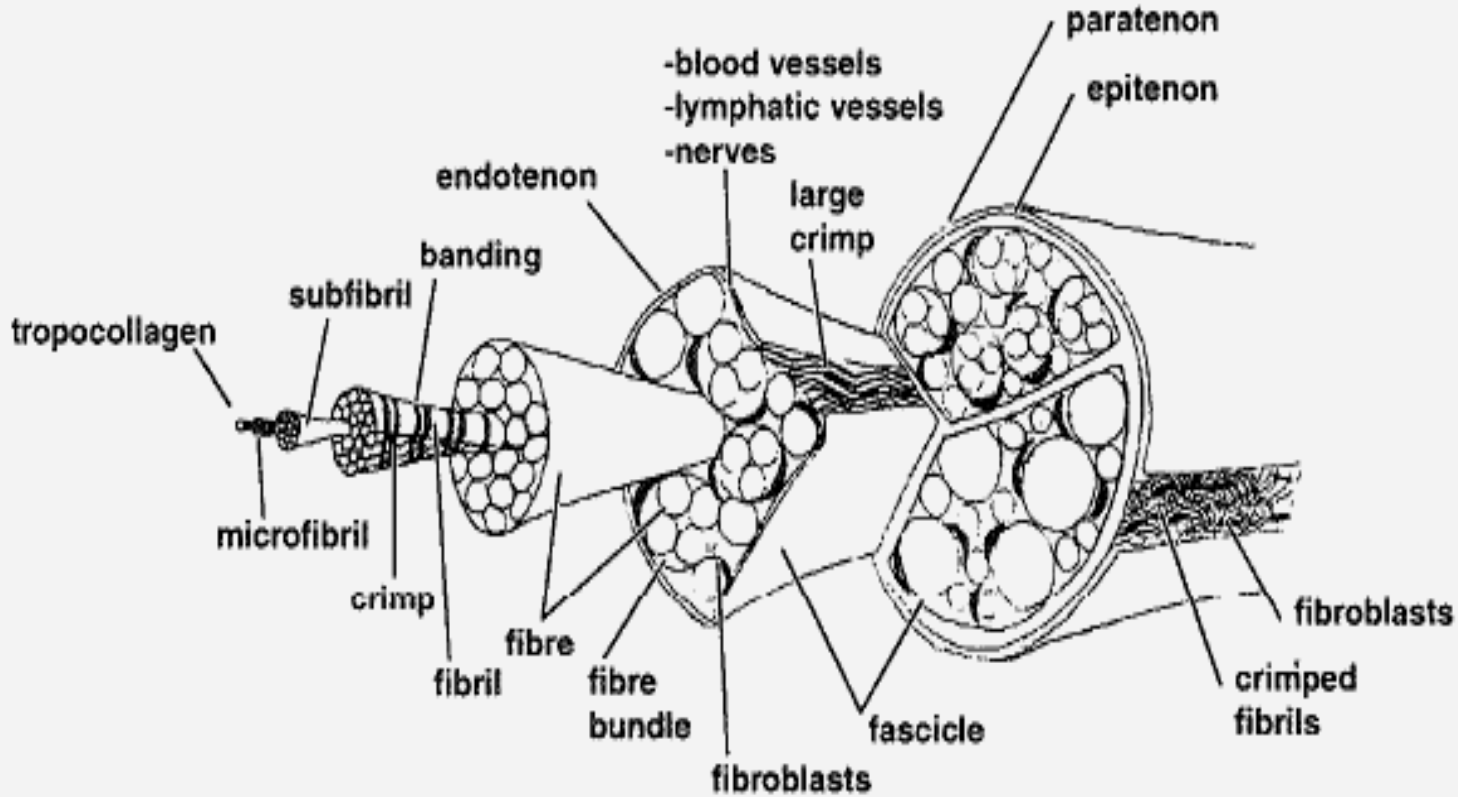
(use this version for studying for exam)

TEM transmission electron microscopy
SEM scanning EM
OM: optical



Hierarchical structure of tendon.
J. Kastelic, A. Galeski, and E. Baer
"The Multicomposite Structure of Tendon",
Connective Tissue Research, 1978, Vol 6, pp. 11-23.

Tendon and ligament Structure



Nigg and Hertzog

Figure 2.6.4 The structural hierarchy of a tendon, from the tropocollagen molecule to the entire tendon. Connective tissue layers or sheaths envelop the collagen fascicles (endotenon), bundles of fascicles (epitenon), and the entire tendon (paratenon). Note that blood and lymphatic vessels and nerves are cut in the cross-section within the endotenon (from Kastelic et al., 1978, with permission).

Summary text of hierarchy

- Tropocollagen 280nm long
  - Covalently crosslinked to neighboring molecules to form long strands of overlapping molecules
  - 5 tropocollagen molecules (in cross-section) make one microfibril
- Microfibril 3.5 nm in diameter
- Microfibrils aggregate to form subfibrils
- Subfibrils aggregate to form fibrils – characteristic periodic pattern for collagen seen here
- Fibrils form fiber bundles
- Fiber bundles aggregate and form fascicles – crimp pattern seen at this level
- Fascicles surrounded by sheath – *endotenon* – composed of collagen fibrils, proteoglycans, elastin
- Endotenon also contains blood and lymph vessels and nerves
- Fascicle bundles are surrounded by epitenon
- Several bundles are surrounded by paratenon – outermost sheath – crimp pattern seen at this level

Tendon and ligament Structure

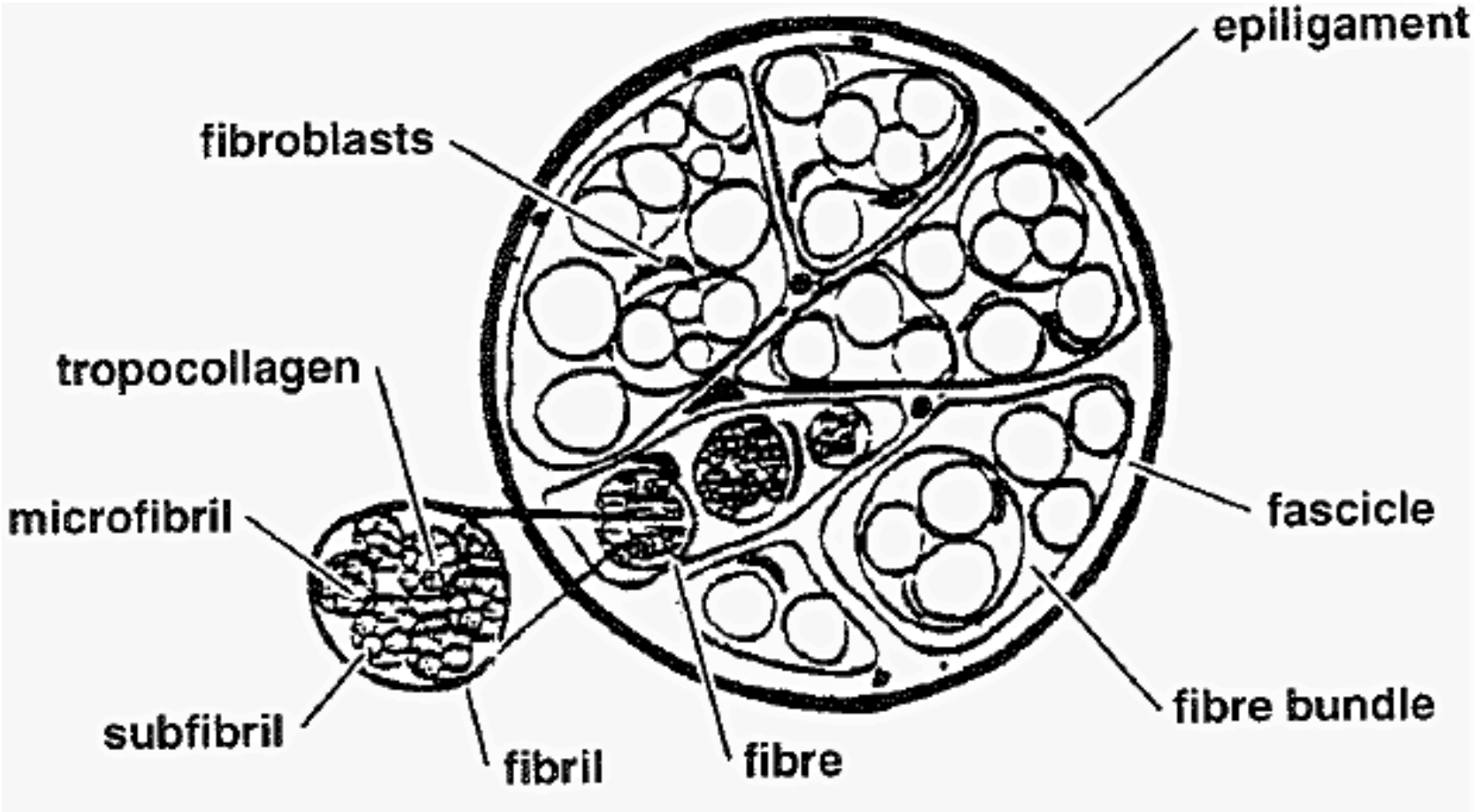


Figure 2.5.3 Schematic diagram of a ligament in cross-section.

Nigg and Hertzog

Summary of tendon (and ligament) structure

Component	Description	Size/Mass	Method by Which It is Seen	Biochemical and Morphologic Characteristics	Clinical Significance
Pro-alpha chain	Polypeptide	150kd	--	Every third amino acid in helical region glycine; rich in hydroxyproline and hydrixylysine	Mutation in just one amino acid can affect subsequent levels of organization and ultimate function
Procollagen	Three pro-alpha chains	450kd	Electron microscopy (rotary shadowing)	Heterotrimer a1(l)2a2(l); secreted by fibroblasts	--
Collagen monomer (tropocollagen)	Triple helix; formed by cleavage of N- and C-terminal propeptides	300kd, 300nm x 2nm	Electron microscopy (rotary shadowing)	Length ~ 4.5 D	Mutations affecting propeptidase activity can impede fibril formation and ultimate function
Fibril	Regular lateral and axial aggregate of collagen monomers	D period= 67nm, diameter=50-200nm	Electron microscopy; negative or positive staining	Repetitive quarter-stagger banding pattern gap and overlap by negative stain, bands (a, B, C. D. e) by positive stain; stabilized by convalent cross-links between collagen monomers; heterotypic structure includes type V collagen and decorin	Change in structure or amount of fibril-associated molecules can affect fibril structure and ultimate function, e.g., fragile skin, osteochondrodysplasias
Fibril bundle = fiber	Group of fibrils delineated by a cleft containing interfibrillar material, fibroblasts, and extended cell processes	Width ~ 20 µm	Light microscopy; collagen pink and cell nuclei blue with H&E stain	Wavy crimp pattern seen; interfibrillar material may include large proteoglycans, type VI collagen, elastin; cell processes in direct contact with other cells	Too small to hold a suture
Fiber bundle = fascicle	Group of fibers surrounded by endotenon	Diameter ~ 1 mm	Visible	Visible, linear elements of tendon; endotenon contains loose connective tissue and vasculature	Delamination separates fascicles
Whole tendon	Connects muscle to bone; surround by epitenon	Diameter from 0.1 to round or flat	Visible	Pinkish white, firm and tough; water content 55%; fibrocartilage at ends and where under compressive load	Tears may occur at ends or midsubstance, partial or complete

Table 1. Organization of type I collagen in tendon[3].

J Musculoskel Neuron Interact 2003; 3(4):323-325

Vasculature

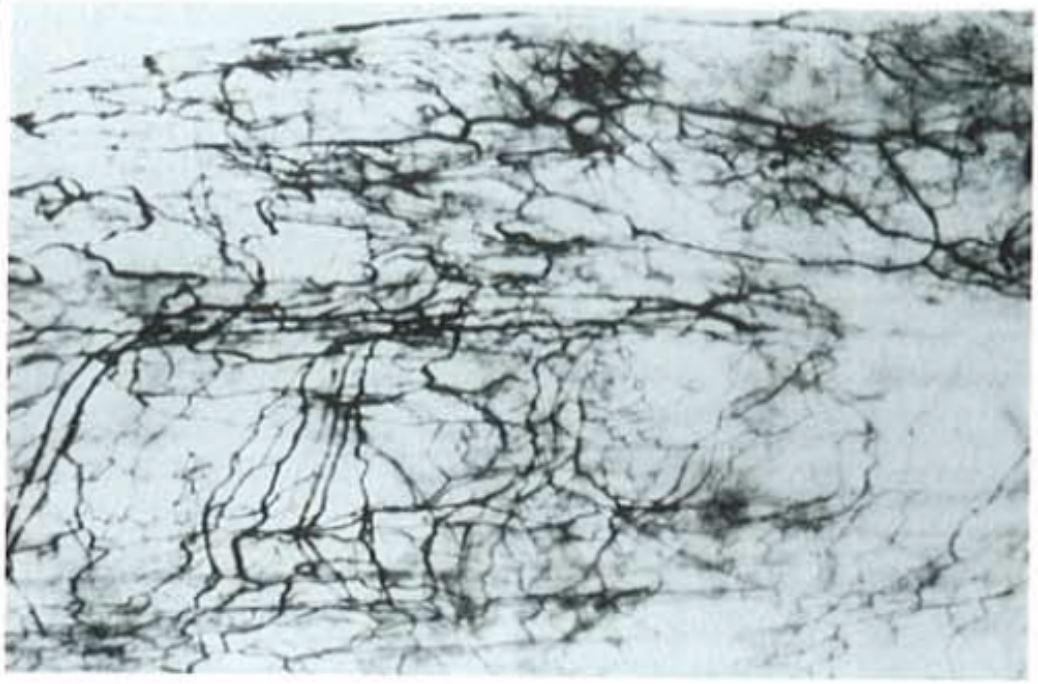


FIG. 4-5

India ink-injected (Spalteholz technique) into the calcaneal tendon of a rabbit, illustrating the vasculature of a paratenon-covered tendon. Vessels enter from many points on the periphery and anastomose with a longitudinal system of capillaries. *Reprinted with permission from Woo, S.L.Y., An, K.N., Arnoczky, D.V.M., et al. (1994). Anatomy, biology, and biomechanics of the tendon, ligament, and meniscus. In S.R. Simon (Ed.).* Orthopaedic Basic Science *(p. 52). Rosemont, IL: AAOS.*

Nordin and Frenkel

Vasculature

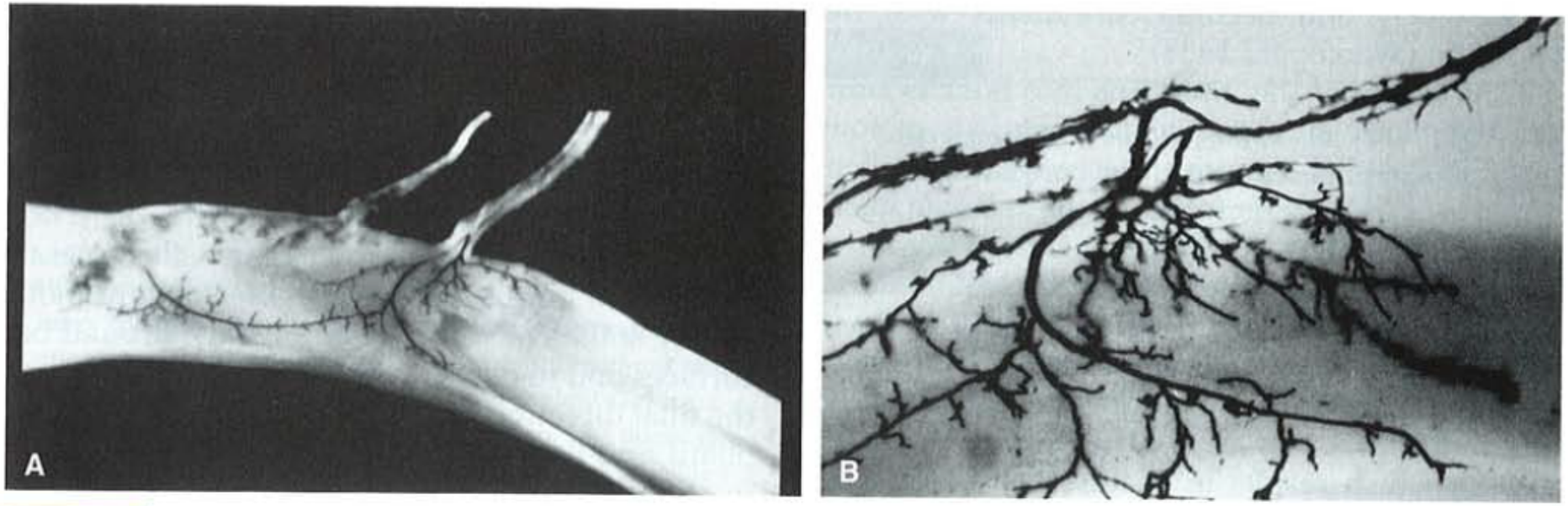
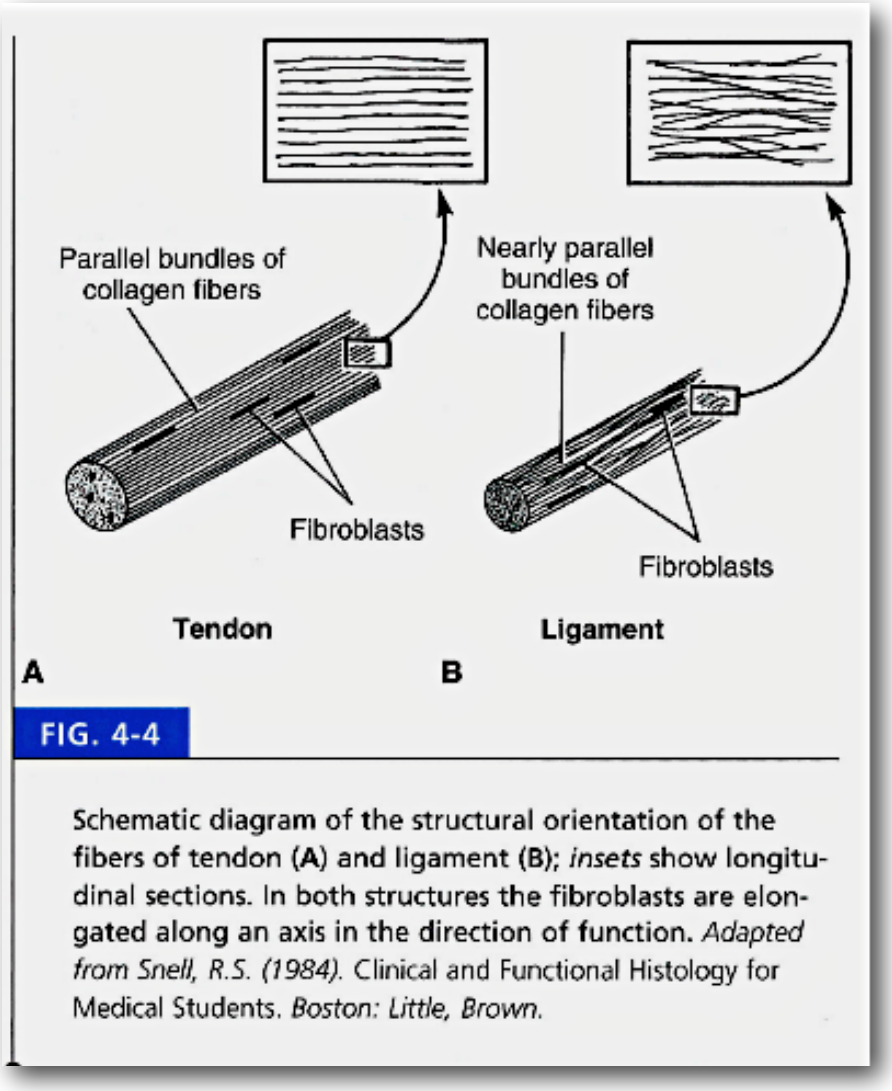


FIG. 4-6

A, India ink-injected specimen illustrating the vascular supply of the flexor digitorum profundus in a human through the vinculum longus. **B,** Close-up specimen (Spalteholz technique) showing the extent of the blood supply from the vinculum longus. The vessels in the vinculum divide into the dorsal, proximal, and distal branches, giving off vascular loops into the tendon substance. *Reprinted with permission from Woo, S.L.Y., An, K.N., Arnoczky, D.V.M., et al. (1994). Anatomy, biology, and biomechanics of the tendon, ligament, and meniscus. In S.R. Simon (Ed.).* Orthopaedic Basic Science *(p. 52). Rosemont, IL: AAOS.*

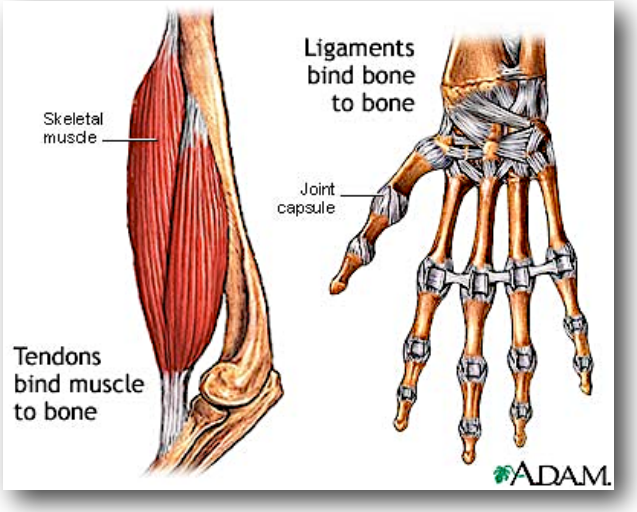
Nordin and Frenkel

Fiber orientation in tendon (and ligament)



Nordin and Frenkel

- Fiber bundles may be 1-20microns in dia. many centimeters long
- Fibroblasts (secrete ECM) align between bundles
- Fibers are orderly, parallel accommodate high uniaxial tensile loads
- For ligaments: fibers less ordered in order to accommodate some off-axis loading experienced by ligaments



Force deformation curves for ligament show initial non-linear behavior (fiber recruitment model). Similar for tendon (& AC)

<u>How might tendon and ligament differ?? (hint – good exam question)</u>

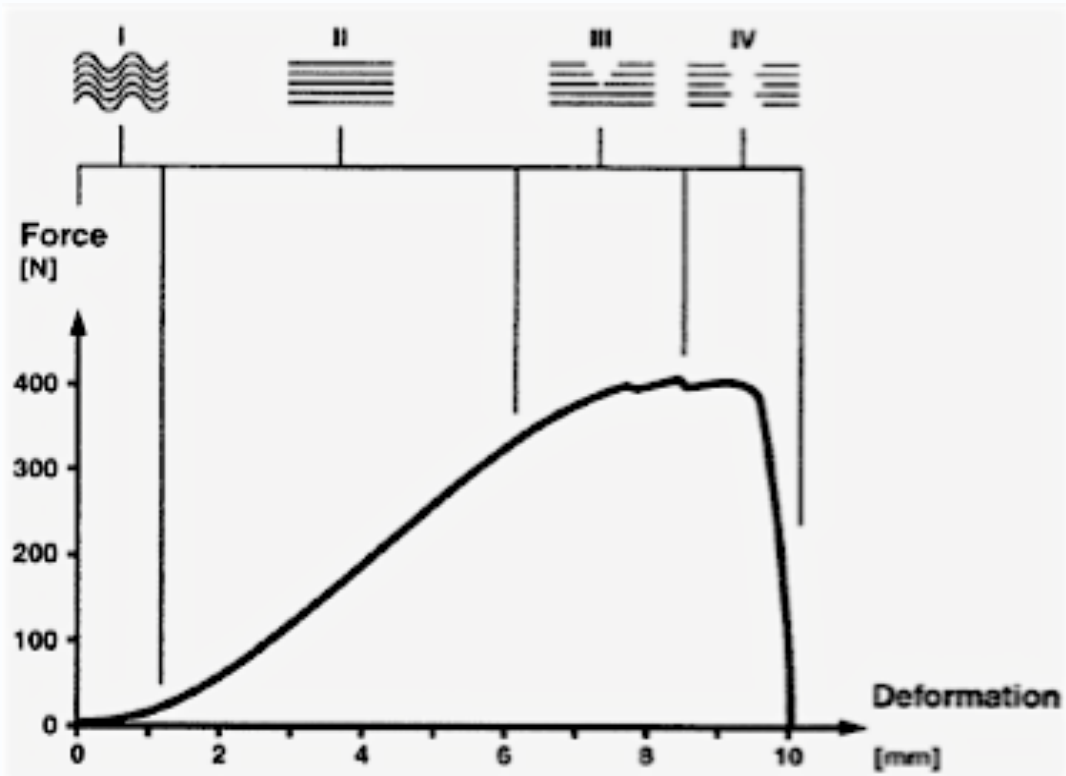


Figure 2.5.6

A typical force-deformation curve for a typical rabbit ligament under monotonic forcing. I = toe region; II = linear region; III = region of microfailure; IV = failure region. At top are schematic representations of fibres going from crimped (I) through recruitment (II) to progressive failure (III and IV).

Nigg and Hertzog

Crimp pattern gives rise to the non-linear toe region

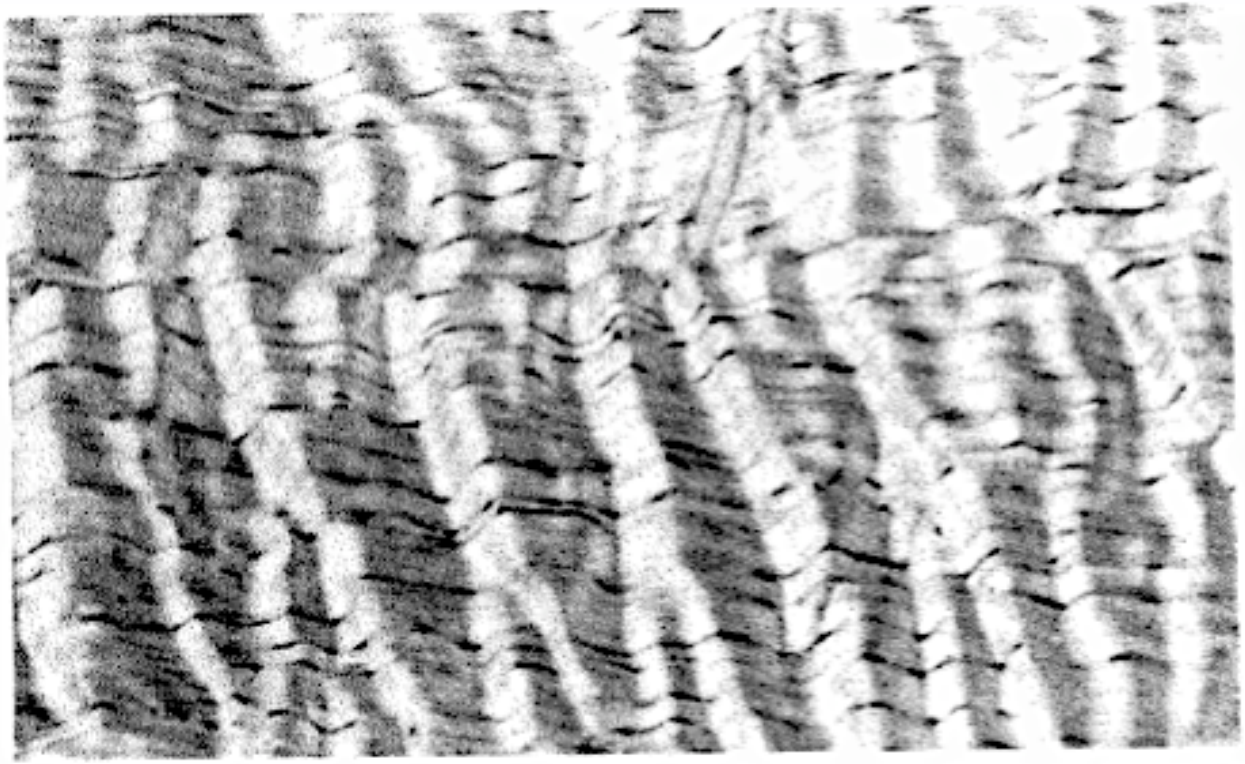


Figure 2.5.2 **Photograph illustrating crimped pattern of collagen in ligament. Fibroblasts may be seen interspersed between the collagen fibres.**

Nigg and Hertzog

Have a general sense of tensile strength & ultimate strain.

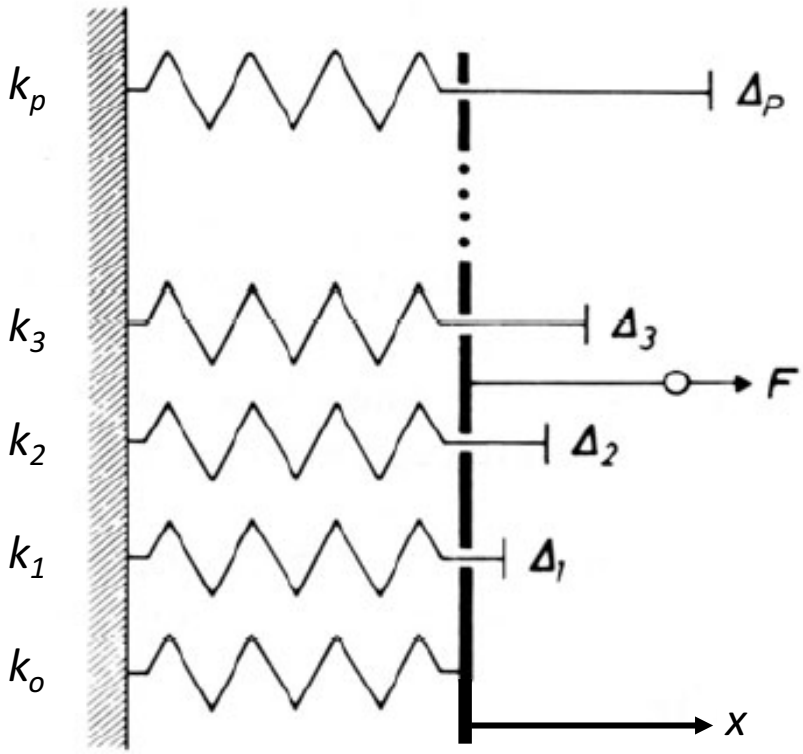
Tangent modulus varies A LOT, tensile strength and ultimate strain, not as much.

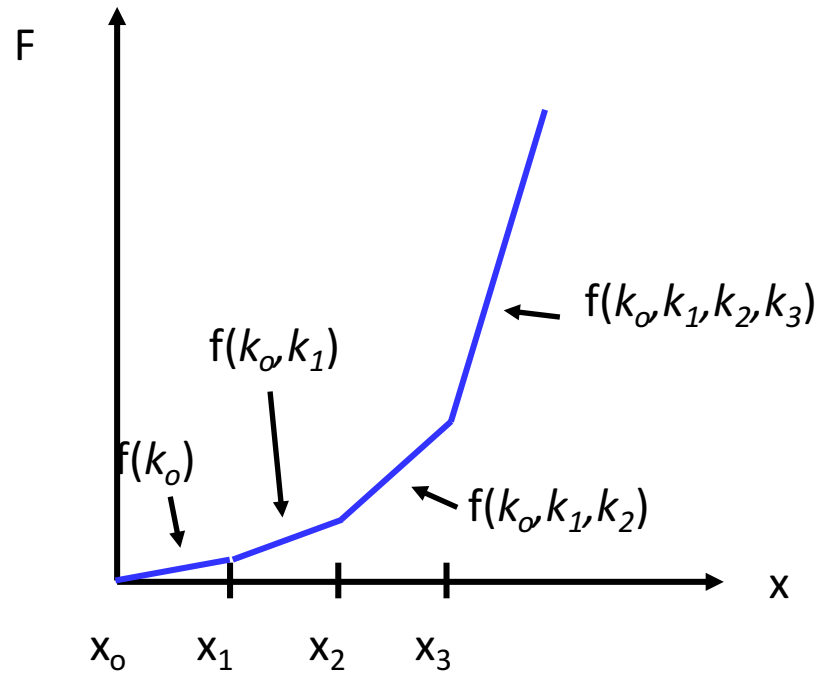
Table 16.2. Material Properties of Human and Animal Ligaments and Tendons (Mean ± SEM)

Specimen type	Source	Tangent modulus (MPa)	Tensile strength (MPa)	Ultimate strain (%)
Human MCL	Quapp & Weiss, 1998	332.2 ± 58.3	38.6 ± 4.8	17.1 ± 1.5
Rabbit MCL	Woo et al., 1990	740 ± 90	77.7 ± 1.9	12.9 ± 1.2
Human PCL (anterolateral bundle)*	Race & Amis, 1994	248 ± 119	35.9 ± 16.2	18.0 ± 5.3
Rabbit ACL	Woo et al., 1986	516 ± 64	62.3 ± 5.2	12.5 ± 1.5
Human ACL, LCL, PCL*	Butler et al., 1990	345.0 ± 22.4	36.4 ± 2.5	16.0 ± 0.8
Human patellar tendon*	Butler et al., 1990	643.1 ± 53.0	68.5 ± 6.0	13.5 ± 0.7
Goat patellar tendon**	Gibbons et al., 1991	1639.1 ± 435.9	126.8 ± 20.8	16.2 ± 3.9

Cowin & Doty

The toe region with non linear stiffness can be described using the fiber recruitment model (already saw)





$$F = \sum_{i=0}^{p} k_i \left(x - x_i \right)$$

After x_p, no more fibers recruited

Striking anisotropy for longitudinal vs transverse. Here MCL

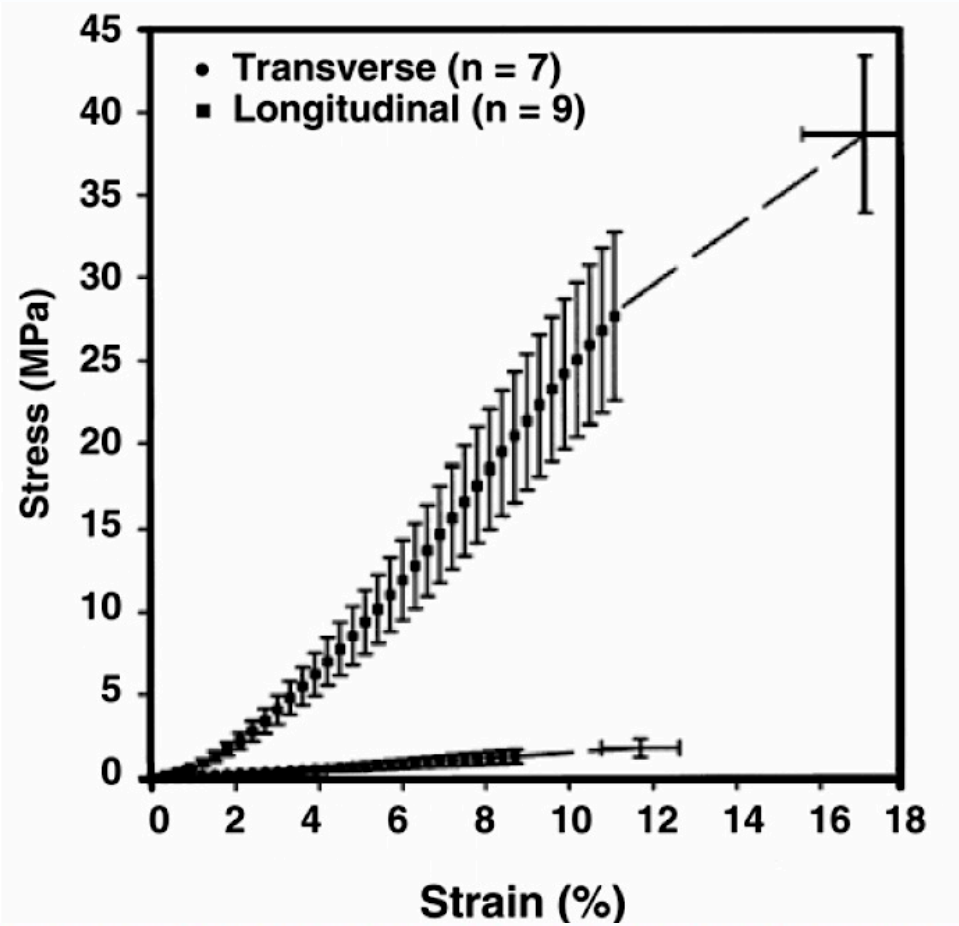
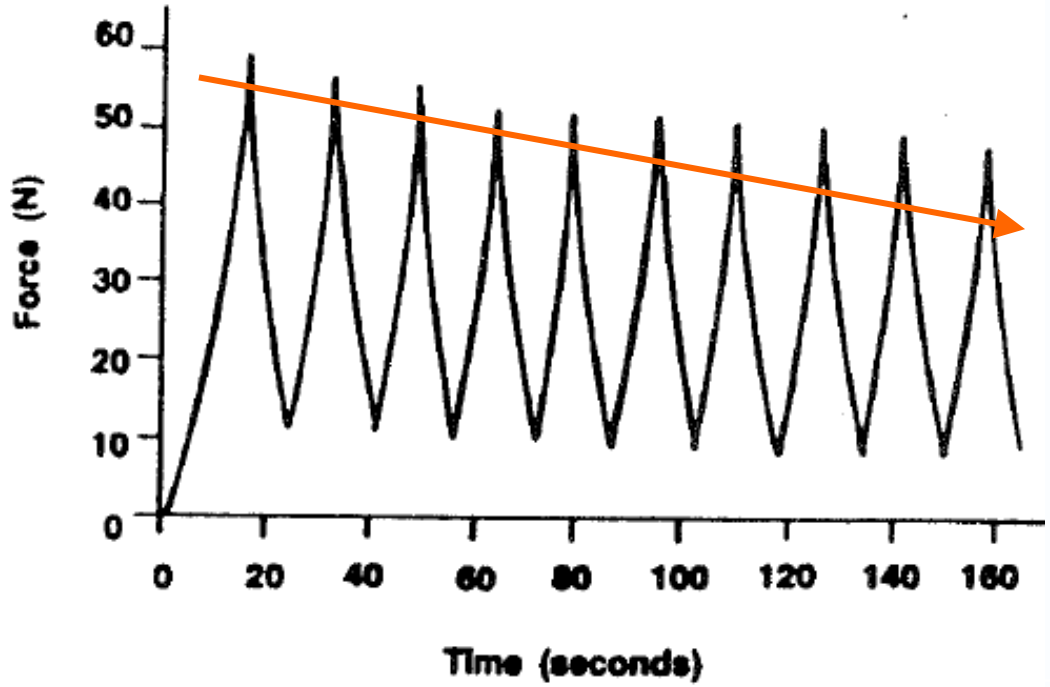


Figure 16.10. Stress–strain curves for human MCL tested parallel and transverse to the collagen fiber direction. The final point in each curve was the average failure point for the specimens. The error bars indicate the standard error for the average ultimate strain and tensile stress. The tangent modulus of the longitudinal curve was over an order of magnitude greater than the tangent modulus of the transverse curve. Reprinted with permission from Quapp and Weiss (1998).

Cowin and Doty

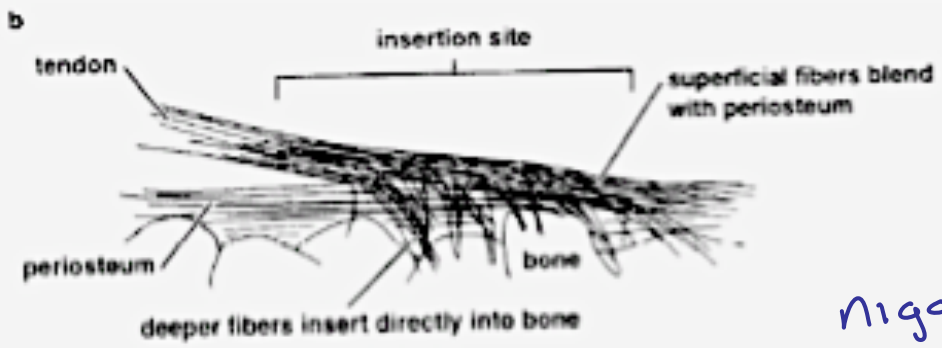
Ligament and tendon have viscoelastic behavior, show hysteresis and softening

Shown here specific for ligament, Hawkins



- *Ligament softening* - peak force for given displacement decreases with each cycle in vitro and in vivo
  - This occurs with daily cyclic activities – walking, running etc.
  - Laxity test in runners and basketball players after 10km race and 90 min playing time, respectively
  - Anterior posterior laxity increased > 18% (showed it was not due to muscle laxity)

Insertion structure in tendon and ligament is similar

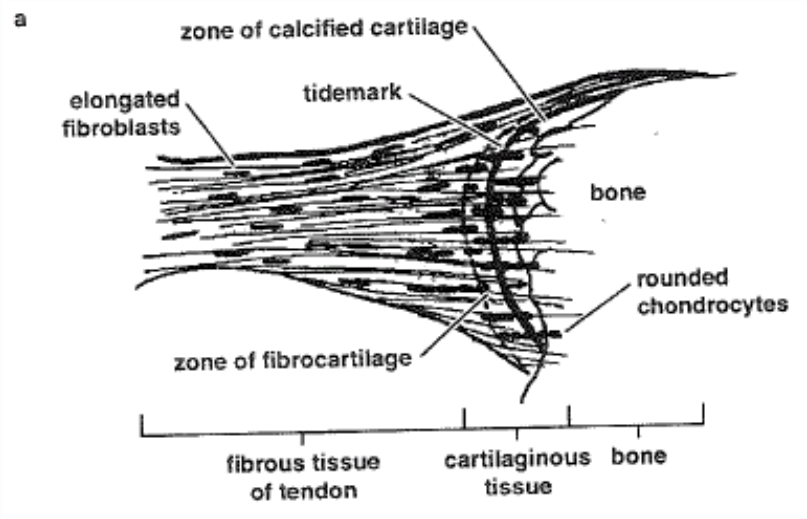


Nigg & Hertzogs

Figure 2.6.7 Schematic diagrams illustrating tendon insertion morphology. (a) The fibrous tissue of the tendon gradually changes to fibrocartilage, then to calcified fibrocartilage before inserting into the bone. (b) shows the tendon approaching bone at an acute angle, the superficial fibres blending directly with the periosteum while the deeper fibres insert into the bone (from Benjamin et al., 1986, with permission of Cambridge University Press).

- Insertion structure is functionally adapted – need to transmit loads between two materials of dissimilar structure and composition
- *Direct insertion: see next slide*
- Indirect insertion: insertion into periosteum (covers bone, liken to the *-tenon shealths) and the periosteum connects directly to bone.
  - Occurs in developing (young) mammals
  - When the tendon approaches at an acute angle
  - Another source says fibers penetrate periosteum and insert into bone

Direct insertion occurs in 4 zones



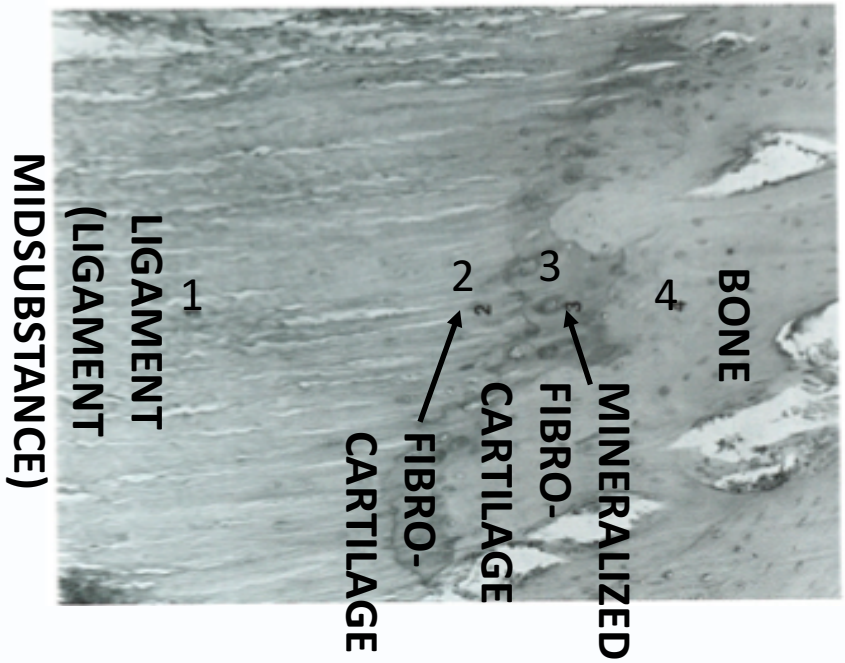


FIG. 4-7

Electron micrograph of a patellar tendon insertion from a dog, showing four zones (×25,000). *Zone 1,* parallel collagen fibers; *zone 2,* unmineralized fibrocartilage; *zone 3,* mineralized fibrocartilage; *zone 4,* cortical bone. The ligament-bone junction (not pictured) has a similar appearance. *Reprinted with permission from Cooper, R.R. & Misol, S. (1970). Tendon and ligament insertion. A light and electron microscopic study.* J Bone Joint Surg, *52A, I.*

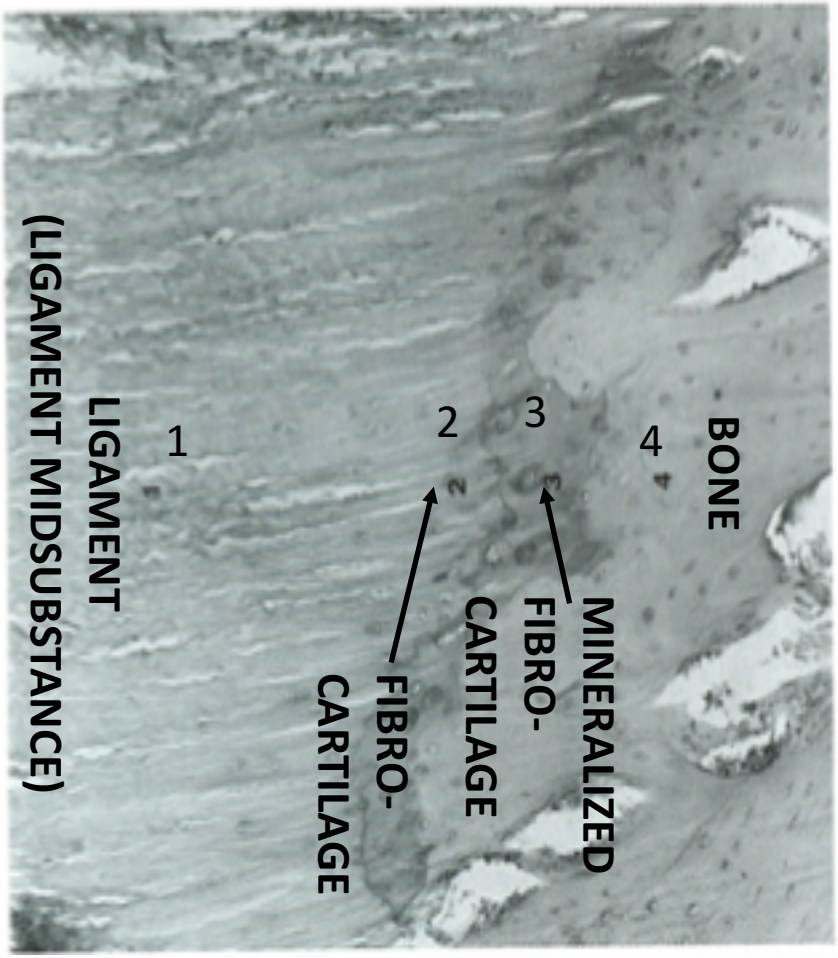
ZONE 1

Normal ‘midsubstance’ (basically is the tendon or ligament)

- organized parallel collagen bundles
- some elastin
- elongated fibroblasts also described spread out and attached
- tenocytes that gradually change to rounded chondrocytes

The four zones in direct insertion are described as:

- **ZONE 2**
- Non-mineralized fibrocartilage (fibrous cartilage)
- Collagen bundles continue from tendenous region
- Increased cell numbers
- Cells are larger and more ovoid in shape – more like chondrocytes than tenocytes
- Cells are arranged in columns like in deep zone region of cartilage near bone
- More dense with PGs, like cartilage
- No blood vessels, like cartilage

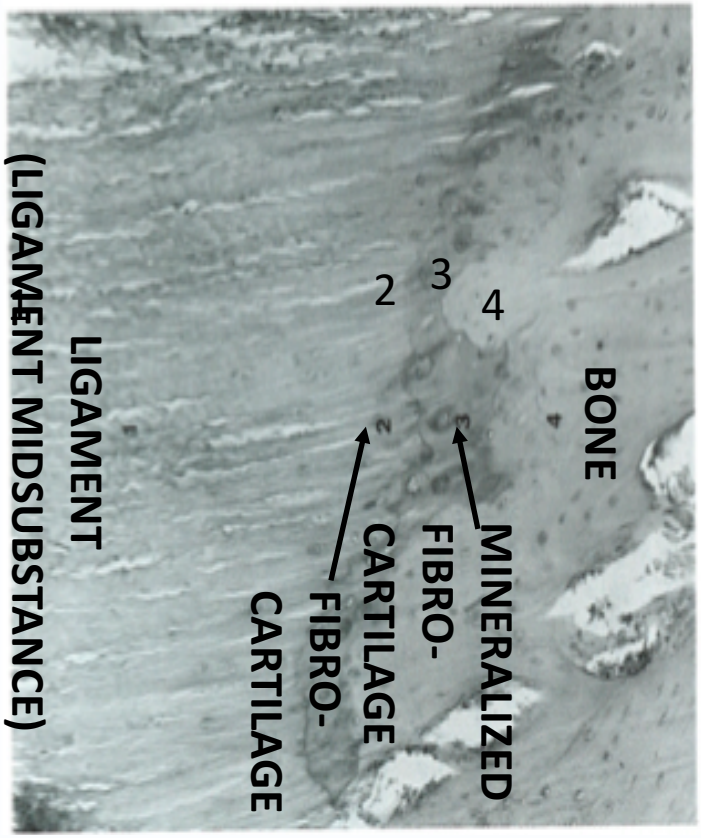


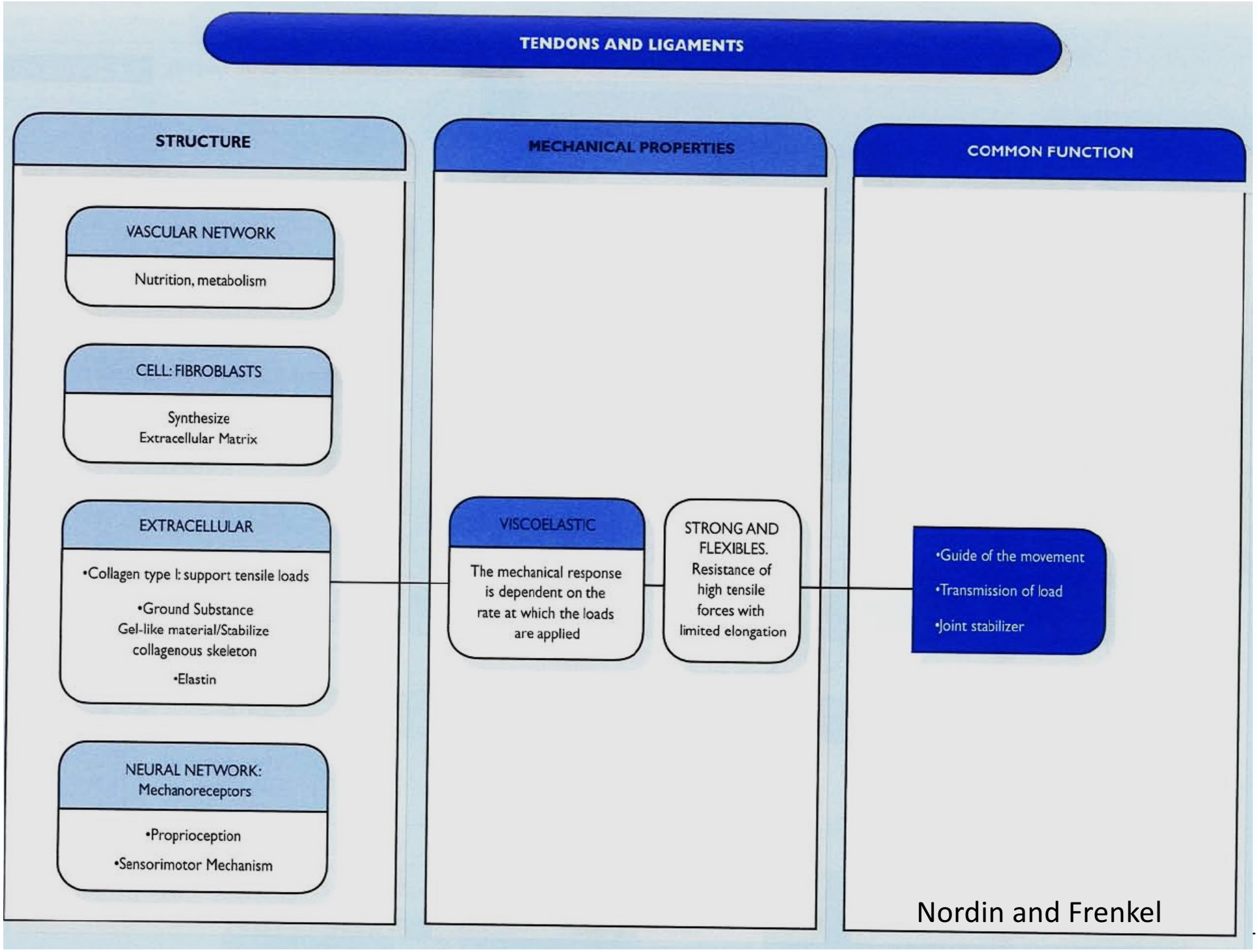
The four zones in direct insertion are described as:

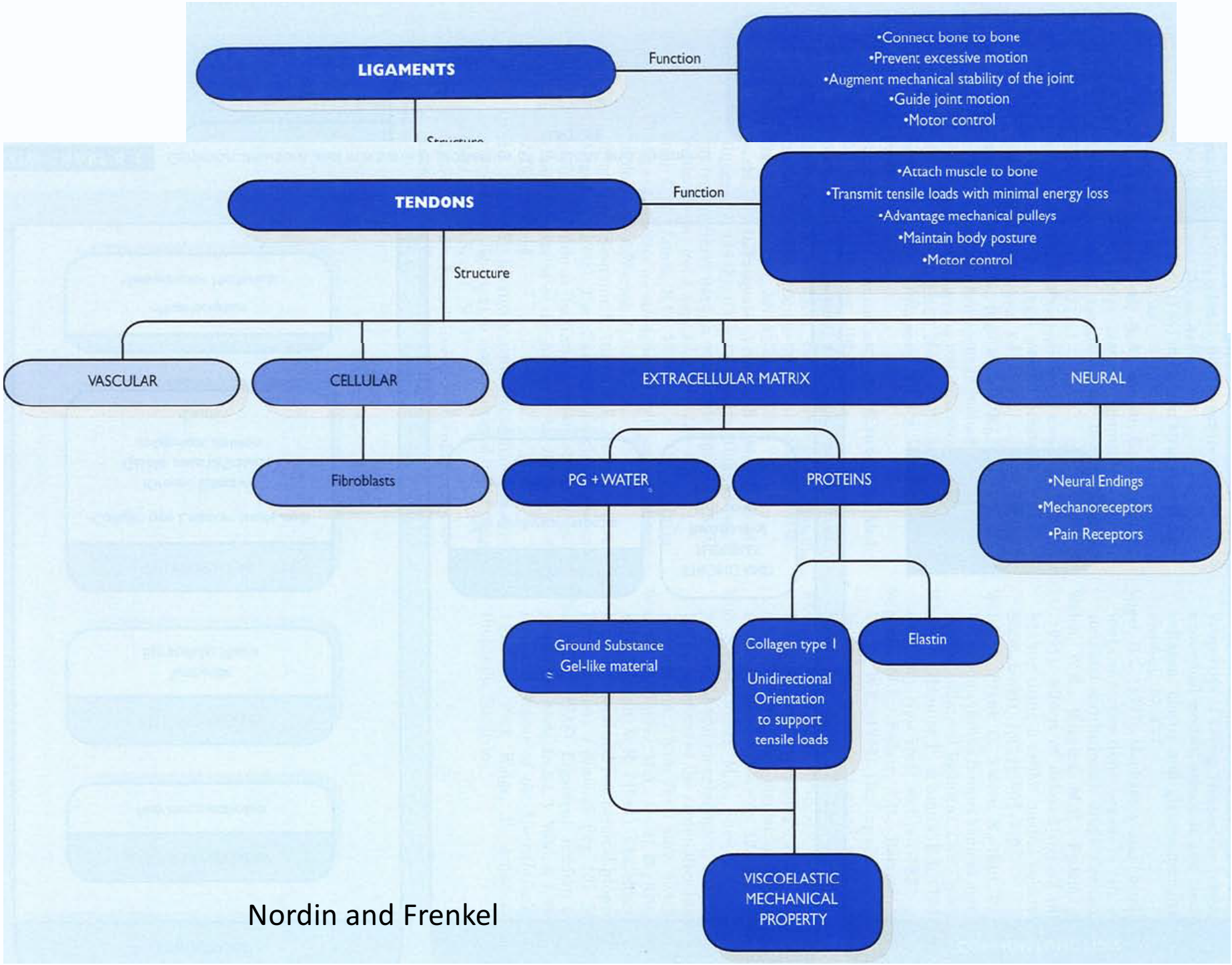
- ZONE 3:
- Mineralized fibrocartilage
- distinguished from Zone 2 by tidemark
- cell morphology same as Z2
- aggregates of mineral
- chondrocytes much less numerous

- ZONE 4
- Bony region
- ligament collagen blends into bone collagen

- The four zones span ~1mm and the thickness of each zone depends on age, the specific tissue of interest (therefore loading).
- The muscle-tendon junction is the 'myotendinous junction.' Will not go in to detail here.
- Thought that tendons with direct insertion (i.e. into the bone) have a larger change in angular movement during movements




TENDONS AND LIGAMENTS
STRUCTURE
VASCULAR NETWORK
Nutrition, metabolism
CELL: FIBROBLASTS
Synthesize
Extracellular Matrix
EXTRACELLULAR
•Collagen type I: support tensile loads
•Ground Substance
Gel-like material/Stabilize
collagenous skeleton
•Elastin
NEURAL NETWORK:
Mechanoreceptors
•Proprioception
•Sensorimotor Mechanism
MECHANICAL PROPERTIES
VISCOELASTIC
The mechanical response
is dependent on the
rate at which the loads
are applied
STRONG AND
FLEXIBLES.
Resistance of
high tensile
forces with
limited elongation
COMMON FUNCTION
•Guide of the movement
•Transmission of load
•Joint stabilizer
Nordin and Frenkel



Nordin and Frenkel

Later we will talk specifically about failure

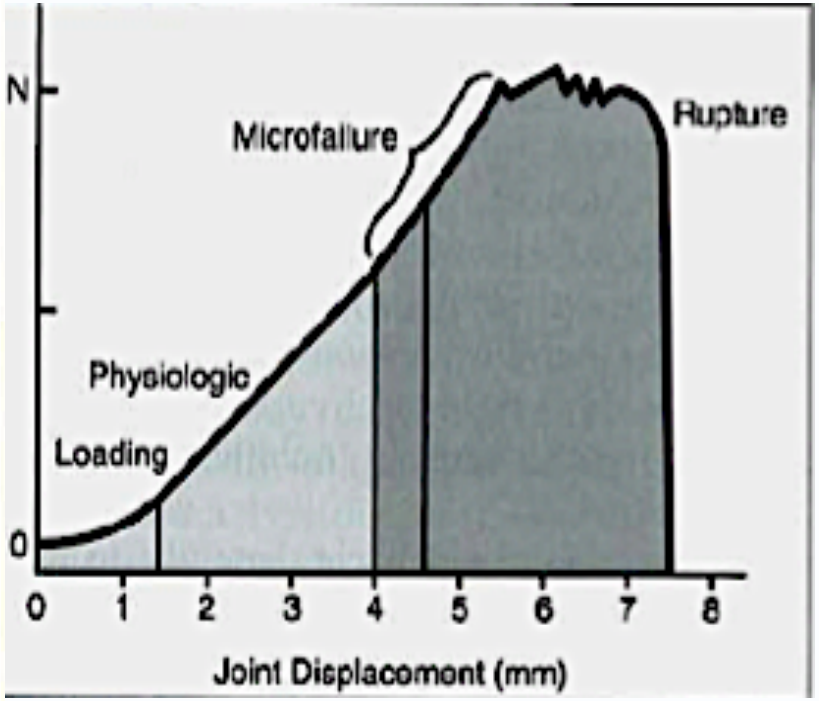
CASE STUDY 4-1

ACL Failure: Failure of the ACL Associated With High Strain and Stress

A 25-year-old male occasional soccer player injured his ACL as a result of an abnormal torque in rotation of the knee. The player locked his foot on the ground and pivoted on his lower limb to produce a high rotational torque on the knee, which increased tensile loads on the ACL.

The first region of the load-displacement curve shows a normal physiological loading response. In the microfailure region, the increase in strain deformation leads to high internal stress and finally a complete rupture. Experimental testing in vitro in human ACL yielded a point of failure between 340 and 390 N.

The knee with the ACL injury will increase intra-articular joint motion, producing abnormally high stresses on other joint structures such as cartilage, which can lead to osteoarthritis. A deficiency in joint stability that results from ACL impairments will increase the likelihood of experiencing the "giving way" sensation or functional instability, thus affecting activities of daily living such as gait, jogging, and squatting (Case Study Fig 4-1-1).



Nordin and Frenkel

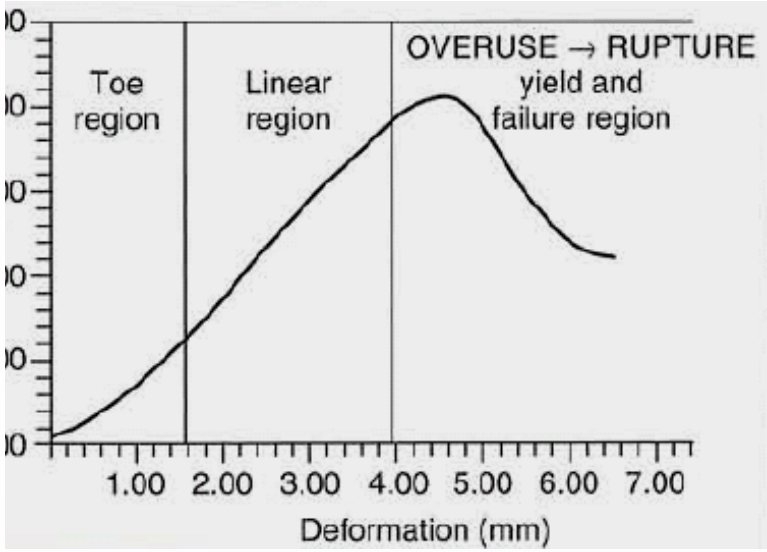
Later we will talk specifically about failure

CASE STUDY 4-2

Tendon Injuries: Achilles Tendon Injuries in Runners, Which Result From a High Strain Rate

A middle-aged male marathoner engaged in a strenuous running activity experienced pain and a popping sensation in his posterior calf. An overuse injury is diagnosed.

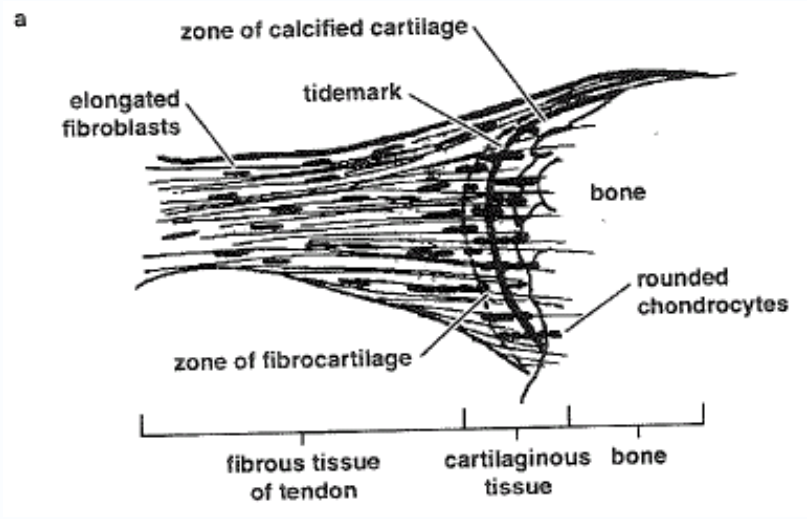
The first region of the load-deformation curve shows a normal physiological toe-loading response. In the linear region, high load is producing a higher deformation within the tendon structure. When the Achilles tendon is subjected to higher strain rates during frequent loading cycles and insufficient time is allowed for the healing process, the result is an overuse injury. Histological studies of these injuries reveal a pathological pattern described as "angiofibrotic hyperplasia," which suggests a degenerative process. This failure in tendon remodeling frequently occurs before the abrupt rupture of the tendon. Relative avascularity, inflammatory disease, and other local factors also contribute to midsubstance ruptures (Case Study 4-2-1).



Nordin and Frenkel

Start of lecture 10/3

RECALL: Direct insertion occurs in 4 zones



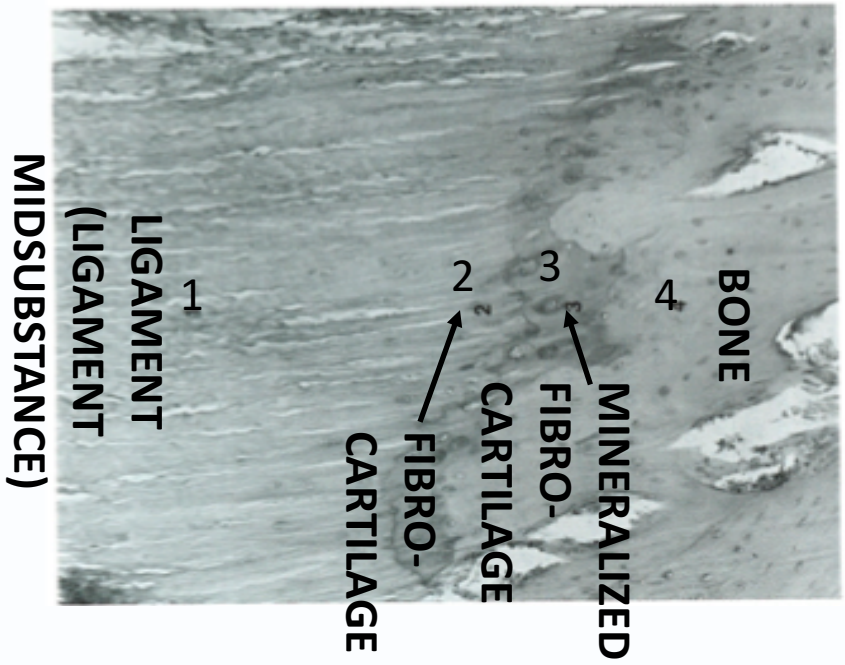


FIG. 4-7

Electron micrograph of a patellar tendon insertion from a dog, showing four zones (×25,000). *Zone 1,* parallel collagen fibers; *zone 2,* unmineralized fibrocartilage; *zone 3,* mineralized fibrocartilage; *zone 4,* cortical bone. The ligament-bone junction (not pictured) has a similar appearance. *Reprinted with permission from Cooper, R.R. & Misol, S. (1970). Tendon and ligament insertion. A light and electron microscopic study.* J Bone Joint Surg, *52A, l.*

ZONE 1

Normal 'midsubstance' (basically is the tendon or ligament)

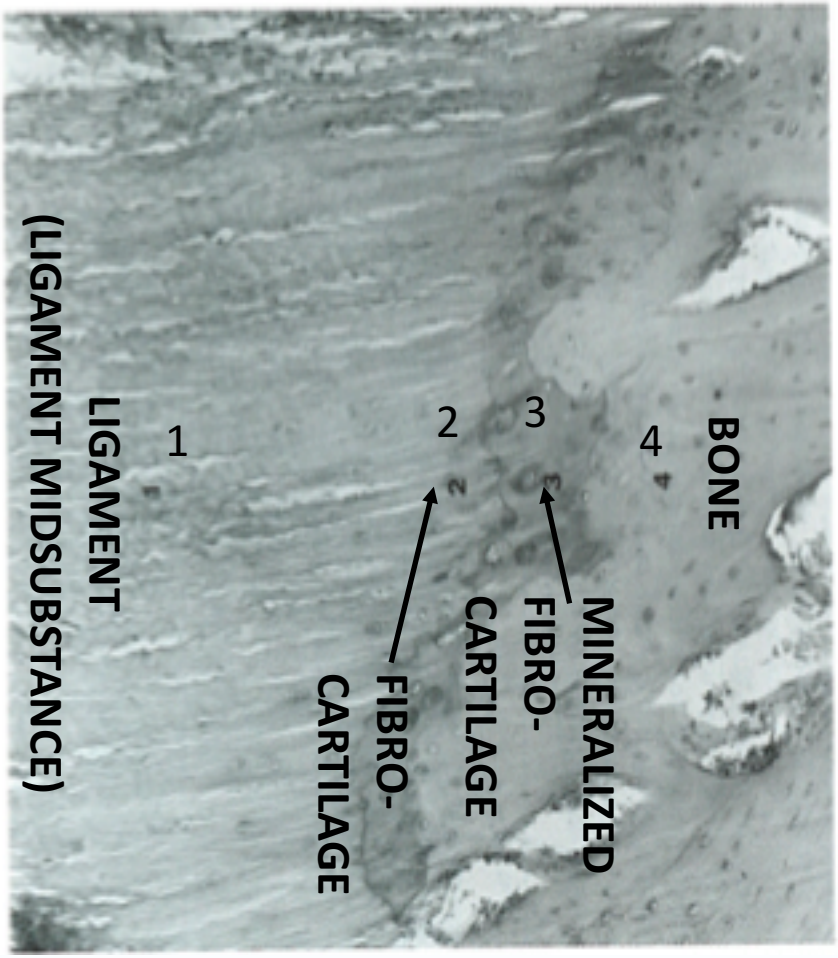
- organized parallel collagen bundles
- some elastin
- elongated fibroblasts also described spread out and attached
- tenocytes that gradually change to rounded chondrocytes

Nordin and Frenkel

RECALL: The four zones in direct insertion are described as:

ZONE 2

- Non-mineralized fibrocartilage (fibrous cartilage)
- Collagen bundles continue from tendenous region
- Increased cell numbers
- Cells are larger and more ovoid in shape – more like chondrocytes than tenocytes
- Cells are arranged in columns like in deep zone region of cartilage near bone
- More dense with PGs, like cartilage
- No blood vessels, like cartilage



Nordin and Frenkel

RECALL: The four zones in direct insertion are described as:

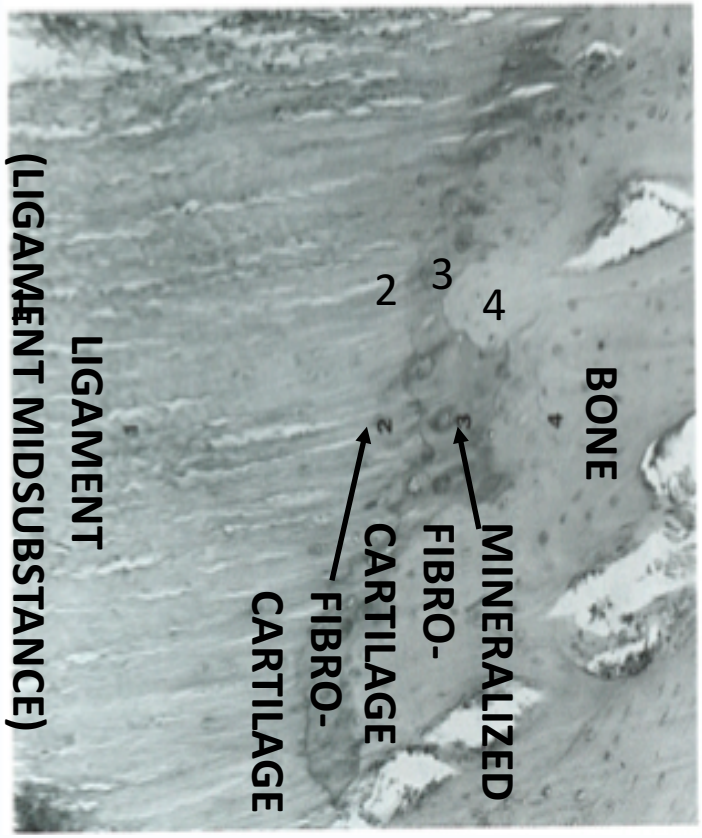
ZONE 3:

- Mineralized fibrocartilage
- distinguished from Zone 2 by tidemark
- cell morphology same as Z2
- aggregates of mineral
- chondrocytes much less numerous

ZONE 4

- Bony region
- ligament collagen blends into bone collagen

- The four zones span ~1mm and the thickness of each zone depends on age, the specific tissue of interest (therefore loading).
- The muscle-tendon junction is the 'myotendinous junction.' Will not go in to detail here.
- Thought that tendons with direct insertion (i.e. into the bone) have a larger change in angular movement during movements



Nordin and Frenkel

There are three **failure mechanisms/modes** for B-L-B complexes

(B–L–B = bone–ligament–bone)

- 3 Failure mechanisms/modes:
  - Ligamentous: midsubstance failure – fibers in tissue pull apart and tears
  - Cleavage at interface: ligament - bone interface failure at the insertion site with no bone fracture
  - Avulsion: tibia or femoral avulsion fractures

There are three known failure mechanisms/modes for B-L-B complexes

- <u>Ligamentous failure ("Mode III")</u>
  - Pulling apart of collagen fibers
  - Rupture of bundles is not localized
  - Mop-like gross appearance
  - More like physiological loading conditions

FIG. 6

Photomicrograph of a portion of the anterior cruciate ligament after failure. The mechanism of failure appeared to be a tensile, pulling-apart type of failure. The collagen fibers are shown to have ruptured at different portions of the ligament, giving an uneven cleavage line corresponding to the common "mop-end" gross appearance (trichrome, × 45).

Noyes et al 1974

There are three known failure mechanisms/modes for B-L-B complexes

<u>Cleavage at bone-ligament interface</u>

(see next slide for better image…)

- Failure at insertion
  - not in ligament itself or underlying bone
- Observed throughout four zones
- Preferred in mineralized fibrocartilage zone (3)
- Least common mode of failure

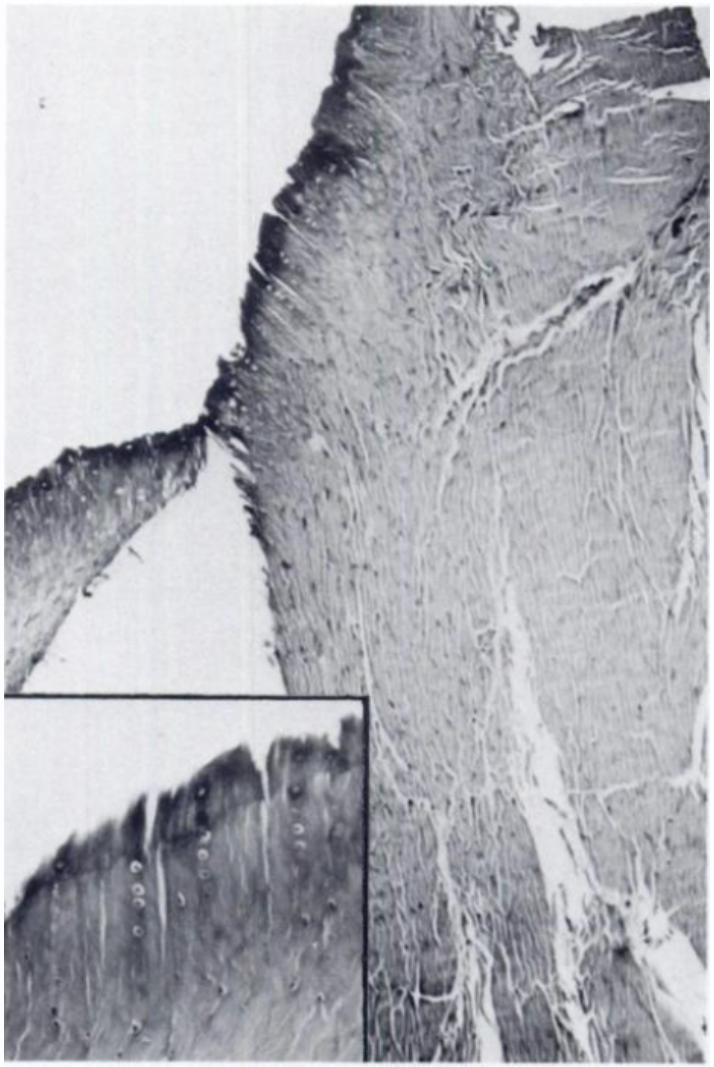
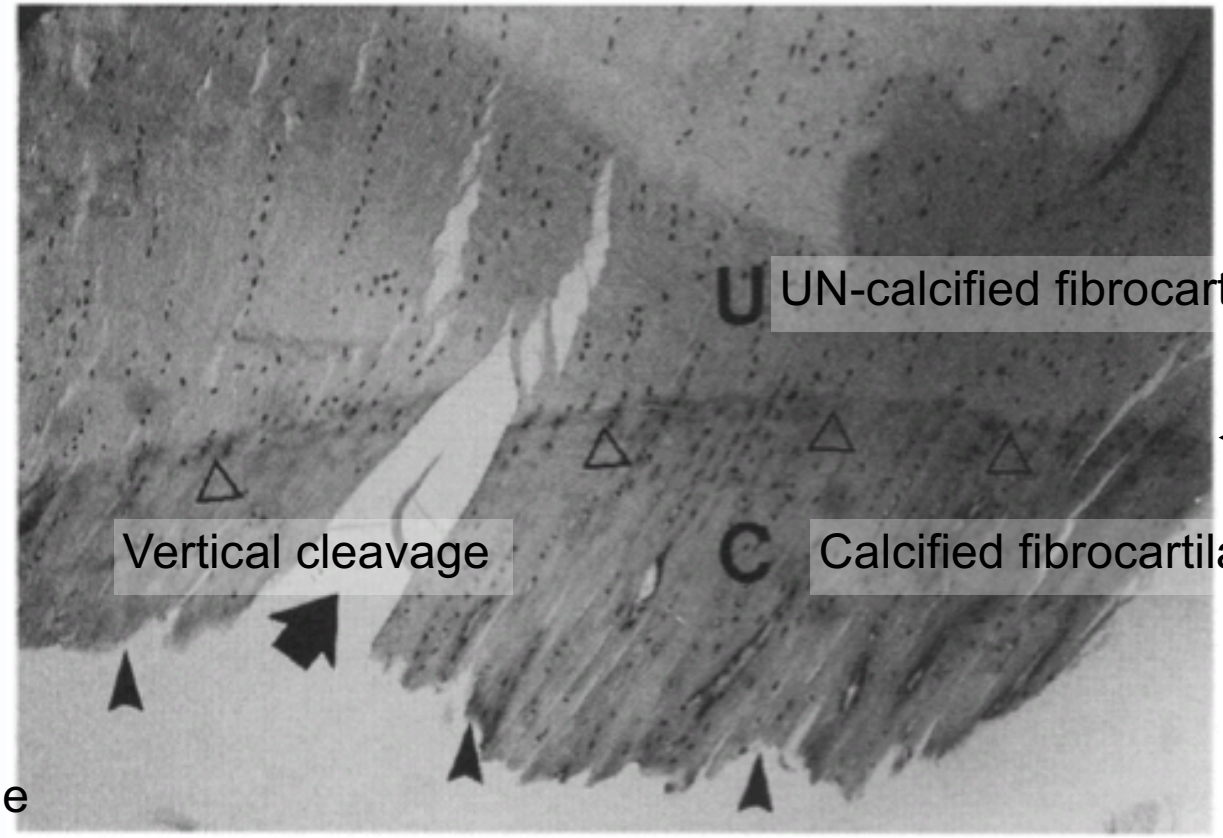


FIG. 8

Photomicrograph shows predilection for cleavage at the ligament-bone interface to occur through the mineralized fibrocartilage zone, in this case just distal to the blue line. Inset shows columnar arrangement of chondrocytes (periodic acid-Schiff, × 80).

A better image of "cleavage"



Ligament end of zone

UN-calcified fibrocartilage

tidemark

Vertical cleavage

Calcified fibrocartilage

Bone end of zone

Fig. 7. Failure at the femoral insertion of the posterior cruciate ligament. A rupture through the calcified fibrocartilage (arrowheads) is combined with a vertical cleavage tear (arrow) crossing the tidemark (empty arrowheads). U, uncalcified fibrocartilage; C, calcified fibrocartilage (HE, × 12.5).

J. Anat. (1996) **189**, pp. 127–133,

There are three known failure mechanisms/modes for B-L-B complexes

Avulsion fracture ("mode I")

- At site of ligament insertion
- Through cortical bone
- Through cancellous bone

- Recall
  - Zone 2 - Non-mineralized fibrocartilage
  - Zone 3 - Mineralized fibrocartilage
  - Zone 4 - bone

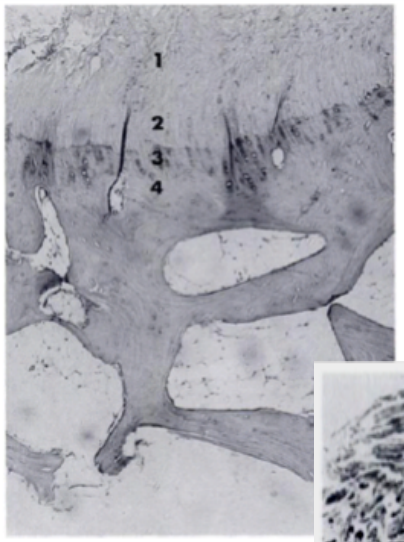
FIG. 7

Photomicrograph after tibial avulsion mode of failure shows that cleavage occurred through cancellous bone below the more dense cortical bone at the site of ligament insertion. The zonal arrangement at the bone-ligament interface is shown. Ligament = zone 1; fibrocartilage = zone 2; mineralized fibrocartilage = zone 3; and bone = zone 4 (hematoxylin and eosin, × 90).

Noyes et al 1974

Review points

- Tendon and ligament insertion into bone
  - has a functionally graded structure
  - 4 Zones:
    1. Ligament
    2. Fibrocartilage
    3. mineralized fibrocartilage
    4. bone
- Failure mechanism near this interface changes
  - Ligament tearing
  - Avulsion
  - “cleavage” – least common and can occur through all four zones



Why does failure mode change?

Failure mode is strain rate sensitive

- At <u>higher rates</u>, *tearing failure* more likely
- At <u>lower rates</u>, *avulsion failure* more likely
- Other studies suggest there is an additional switch between modes (Hyman, 2002)
- Still others say the sensitivity is lost at rates of ~100's/s (Bonner, 2015; McGovern 2002)

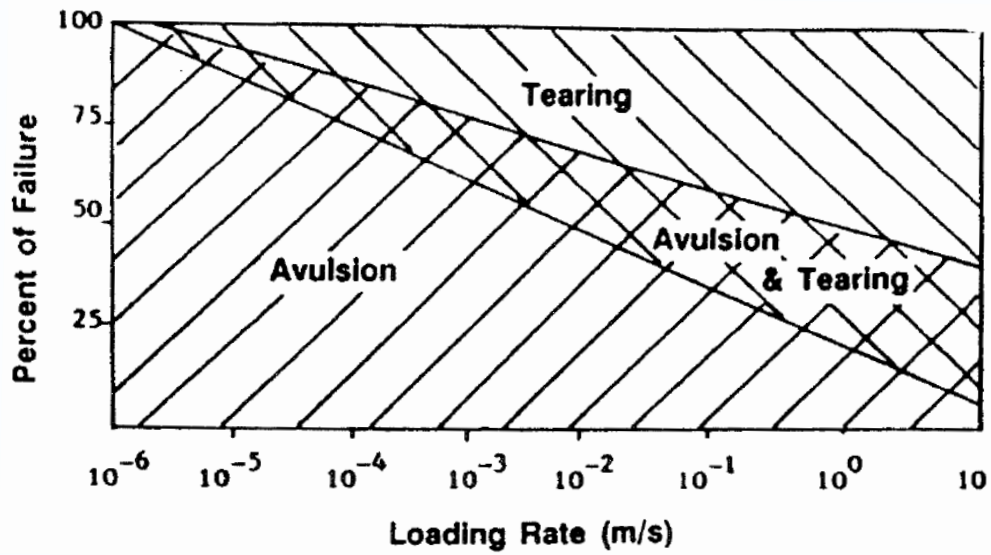


Figure 5.8 Experimental results indicate the probability of specific failure modes of bone-ligament-bone units as a function of loading rate. At slow loading rates, bony avulsion failures have the greatest probability of occurring. At fast loading rates, mid-substance failures are expected. (Adapted by permission from Crowninshield & Pope, 1976.)

Hawkins

What is strain rate sensitivity?

- Mechanical properties change with strain rate
- Typically yield stress increases, yield strain decreases, modulus may increase or stay constant.
- *How/Why might strain rate sensitivity influence the failure mode? (discuss)*

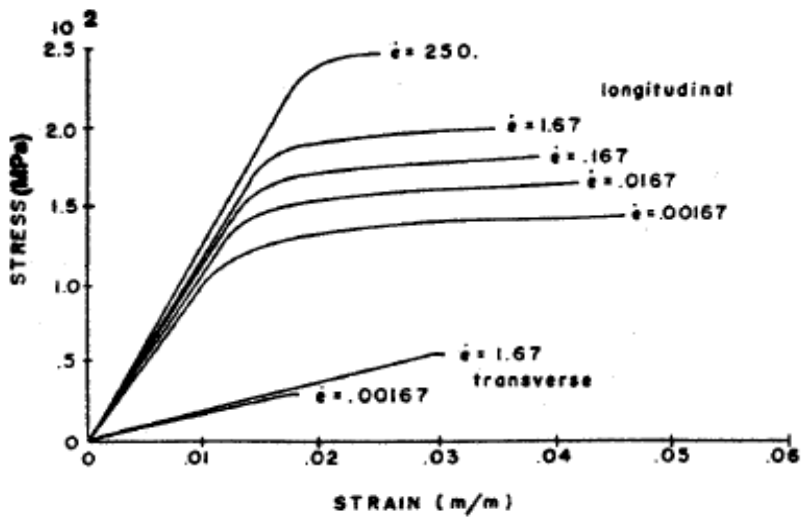


Figure 2
Stress-strain relationship of compact bovine bone [5].

Lee and Hyman, BMC 2002

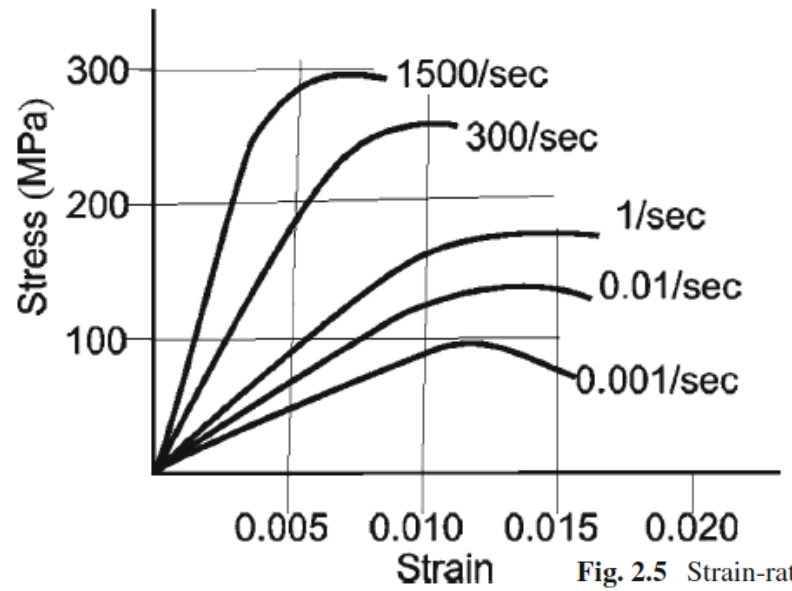
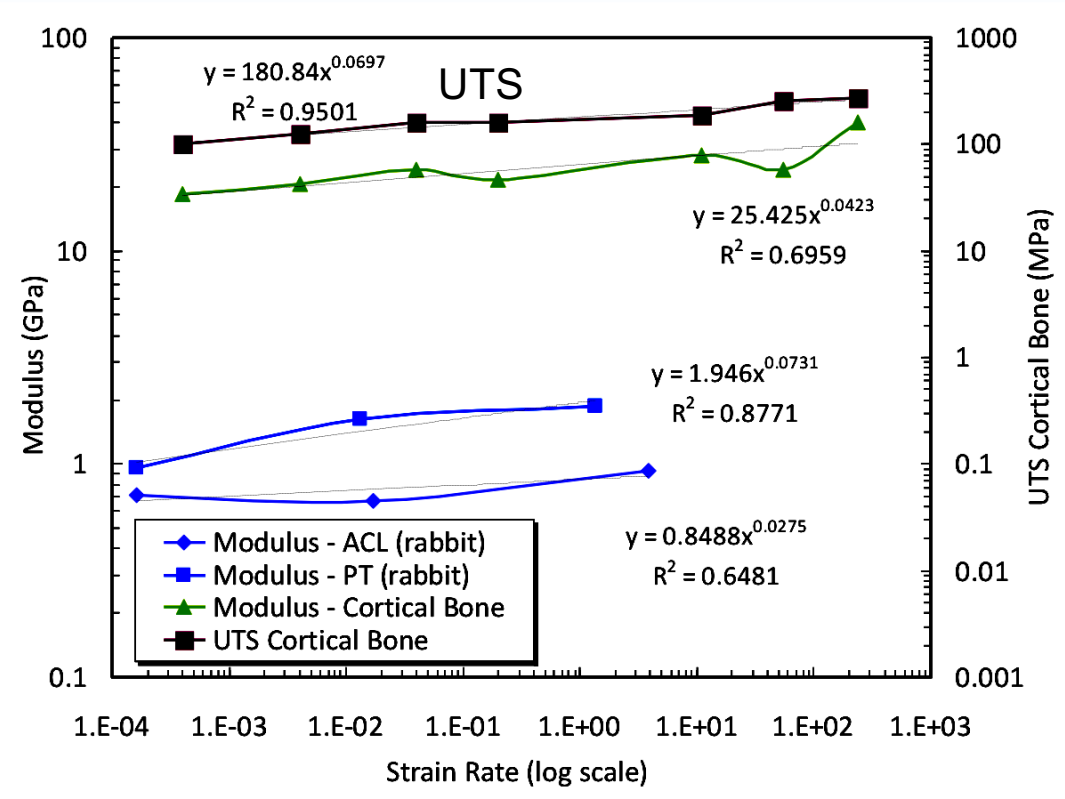


Fig. 2.5 Strain-rate sensitivity of cortical bone. As the strain rate increases, the ultimate strength increases and the ultimate strain decreases

S. Pal, *Design of Artificial Human Joints & Organs*, DOI 10.1007/978-1-4614-6255-2_2, © Springer Science+Business Media New York 2014

Strain rate dependence, fit to data as shown

$$\sigma = k\,\dot{\varepsilon}^{m}$$



ACL and PT data taken directly from Tables in Danto and Woo (PT and ACL) and fit to power curves. Data presented for bone were estimated from a figure in Mow and Huiskes (4-8) and fit to a power curve.

Caution

- This is one way to represent these data (metals follow this behavior)
- Does not include wide range of data from the literature
  - May not be the optimal representation for tissue
  - Strain rate sensitivity could be nonlinear (on log scale) for bone or tendon/ligament
  - Could have multiple regimes…
  - Literature is unclear – too many variables
    - Species (monkey, dog, rat, pig, human)
    - Specific ligament, etc.

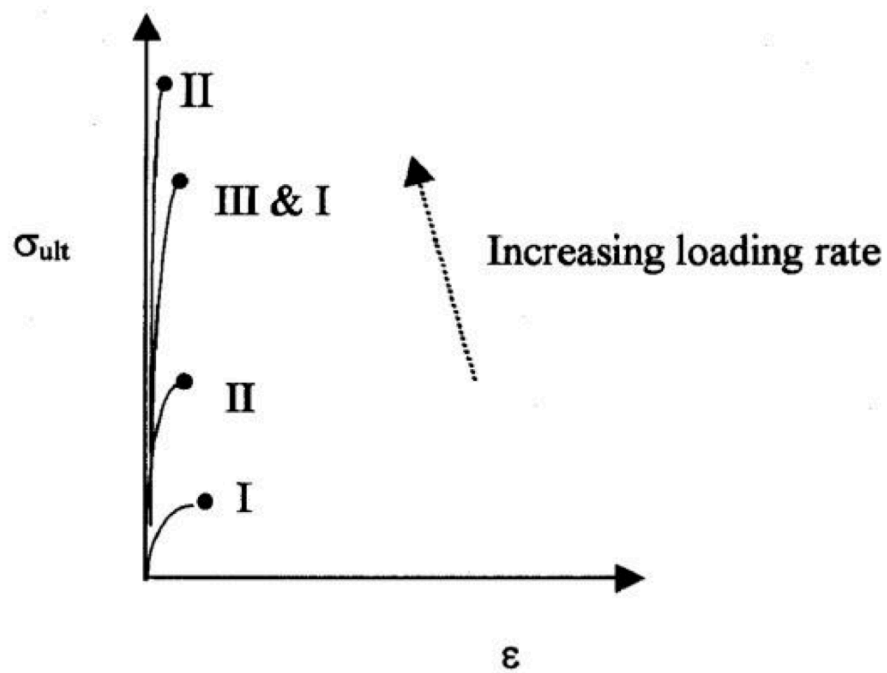
Relative strain rate sensitivities can be described by the exponent in the power law from previous slide. Note that this *specific* relationship is true for most metals, and while there is a strain rate dependence for bone and ligament, *it may not be linear (on log scale)*.

- Power law exponents:
  - property = constant*strain rate^{exponent}
  - From these few sources:
    - Bone UTS exponent ~0.06-0.07
    - Bone Modulus exponent (from estimate) ~ 0.04
    - PT modulus from mature rabbit exponent ~0.07
    - ACL modulus from mature rabbit exponent ~0.03
  - Superplastic metals have exponent of ~.3

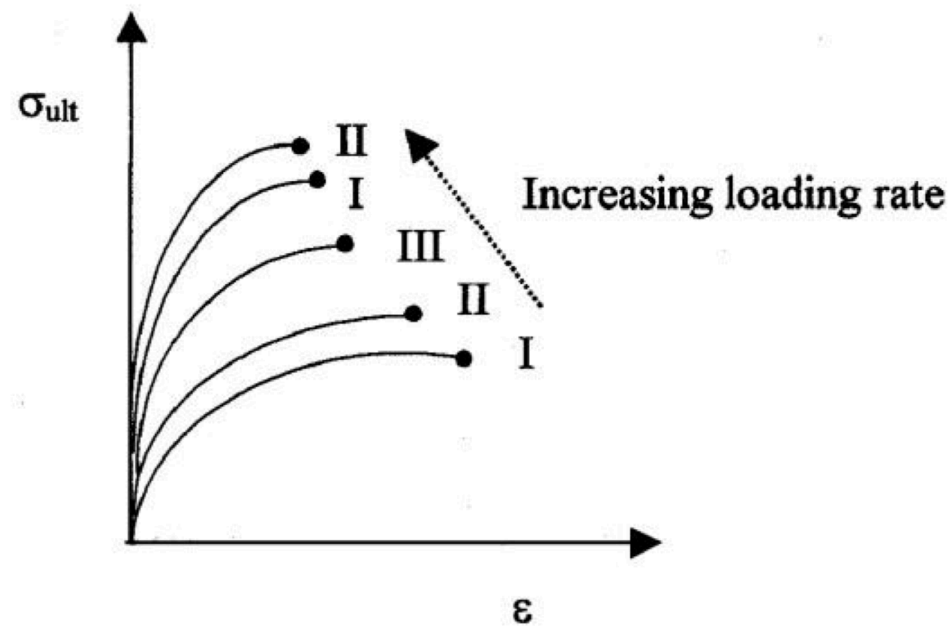
$$\sigma = k\,\dot{\varepsilon}^{m}$$

An interpretation of the mechanism behind the change in failure modes with a <u>qualitative</u> look at strain rate dependence of failure for bone vs ligament.

(Hyman, BMC 2002)



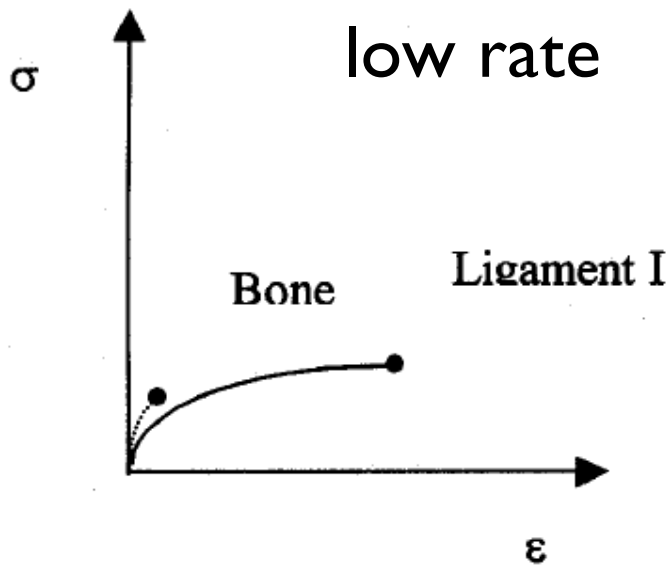
BONE



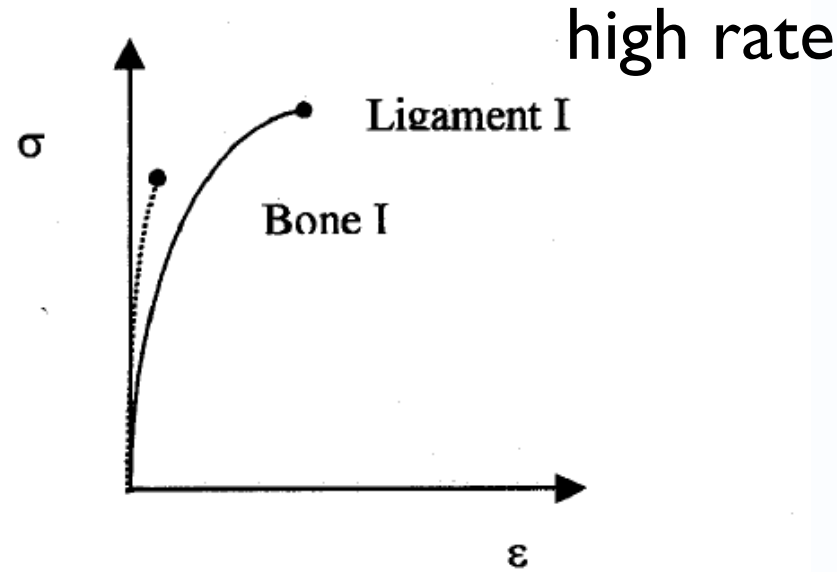
LIGAMENT

Don't worry about I, II, III etc right now…we will see the meaning over the next few slides.

Hyman's qualitative model: Mode I



Mode I. Avulsion > Ligament tearing

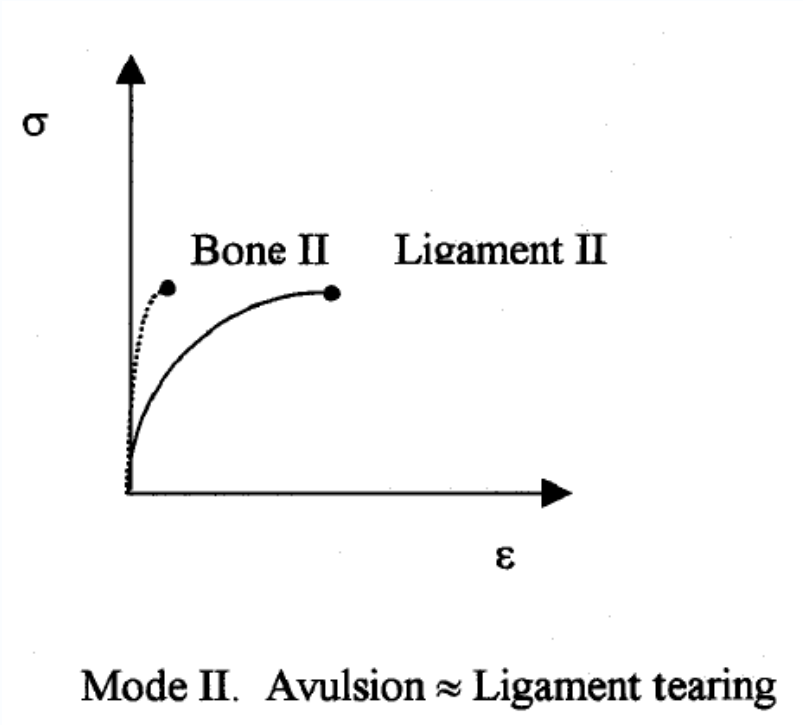


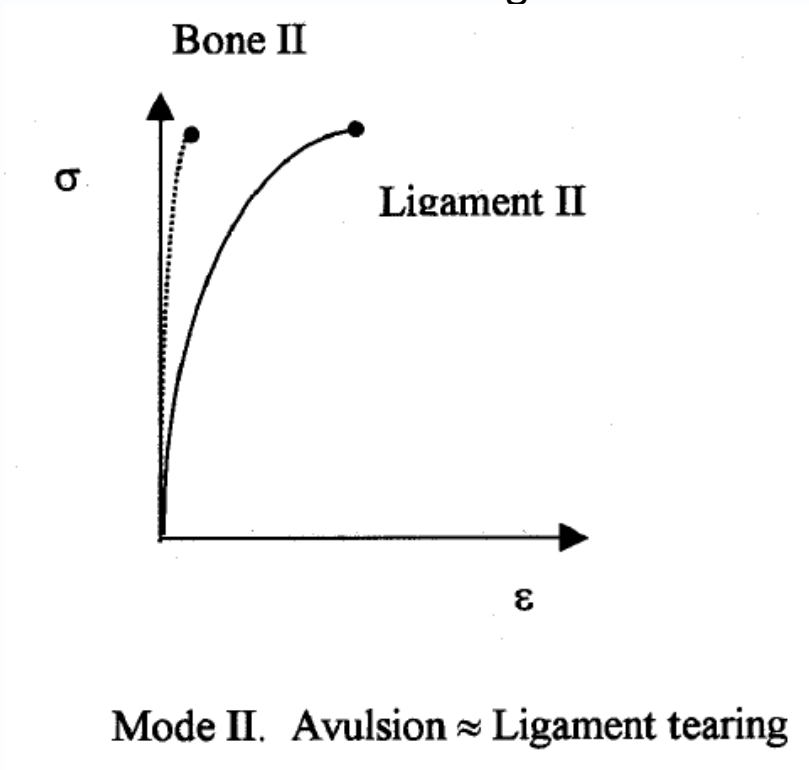
Mode I. Avulsion > Ligament tearing

- Avulsion more frequent at both low and higher rates
- UTS bone and ligament small (at low rate)
- Ligament UTS > Bone UTS (difference may be greater at high rates)
- Bone fails bc smaller UTS (weakest link)

Hyman's qualitative model: Mode II

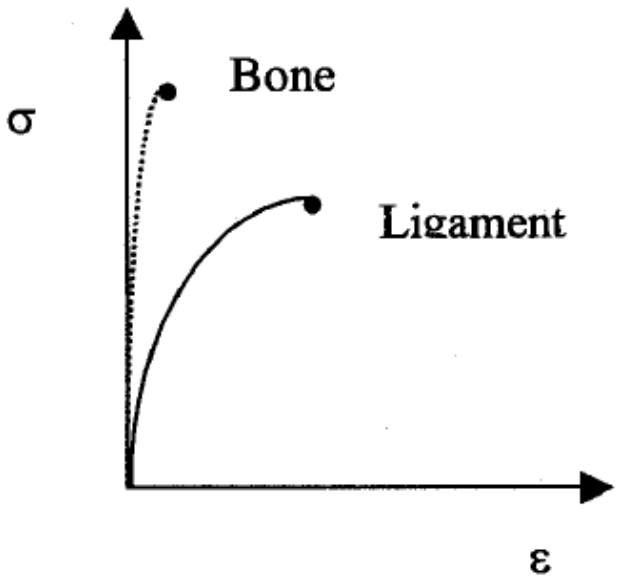
Higher strain rate…





- bone and ligament fail with equal probability
  - strain rates for which strength is same – may occur at more than one rate
- UTS determined by other parameters
  - Age, Health, etc

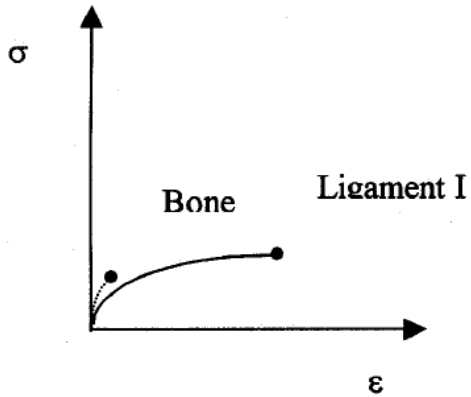
Hyman's qualitative model: Mode III



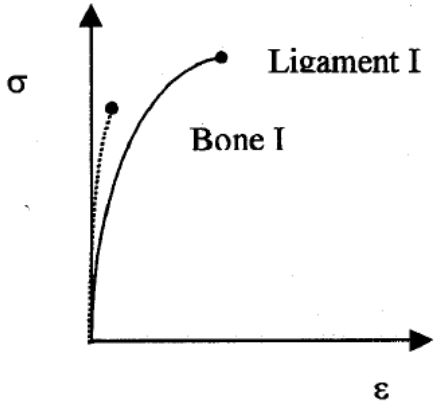
Mode III. Avulsion < Ligament tearing

- Ligament tearing
- UTS bone > UTS ligament
- Ligament fails

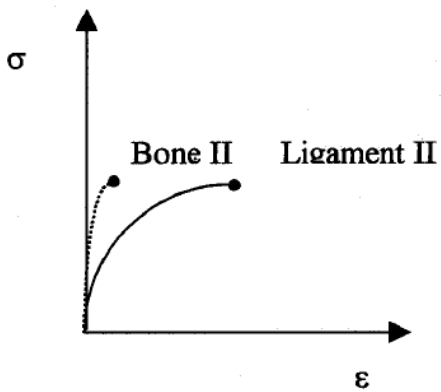
What might the UTS vs strain rate look like for bone and ligament?



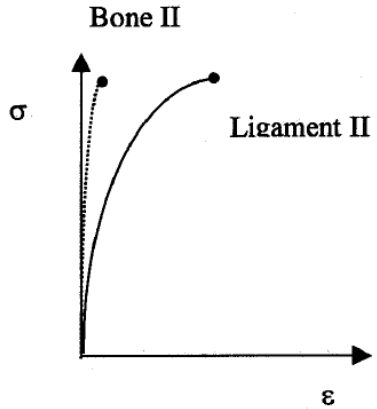
Mode I. Avulsion > Ligament tearing



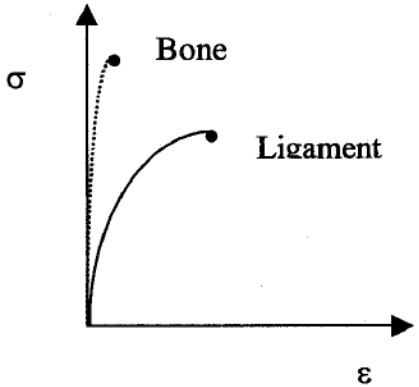
Mode I. Avulsion > Ligament tearing



Mode II. Avulsion ≈ Ligament tearing

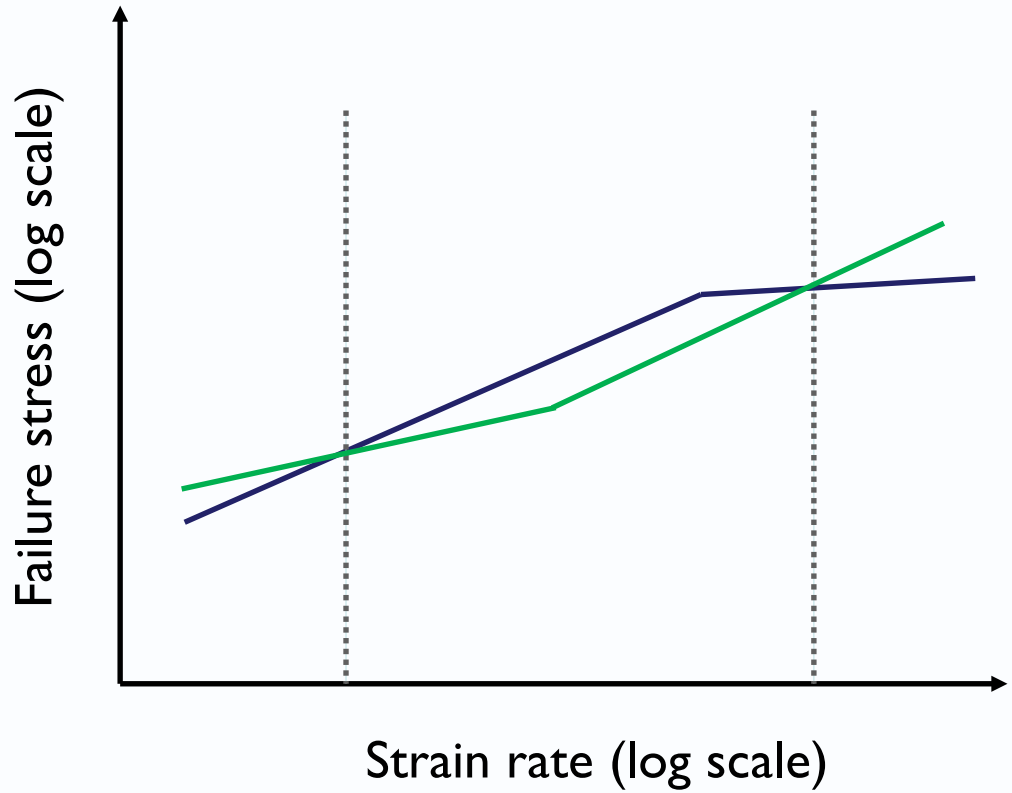


Mode II. Avulsion ≈ Ligament tearing

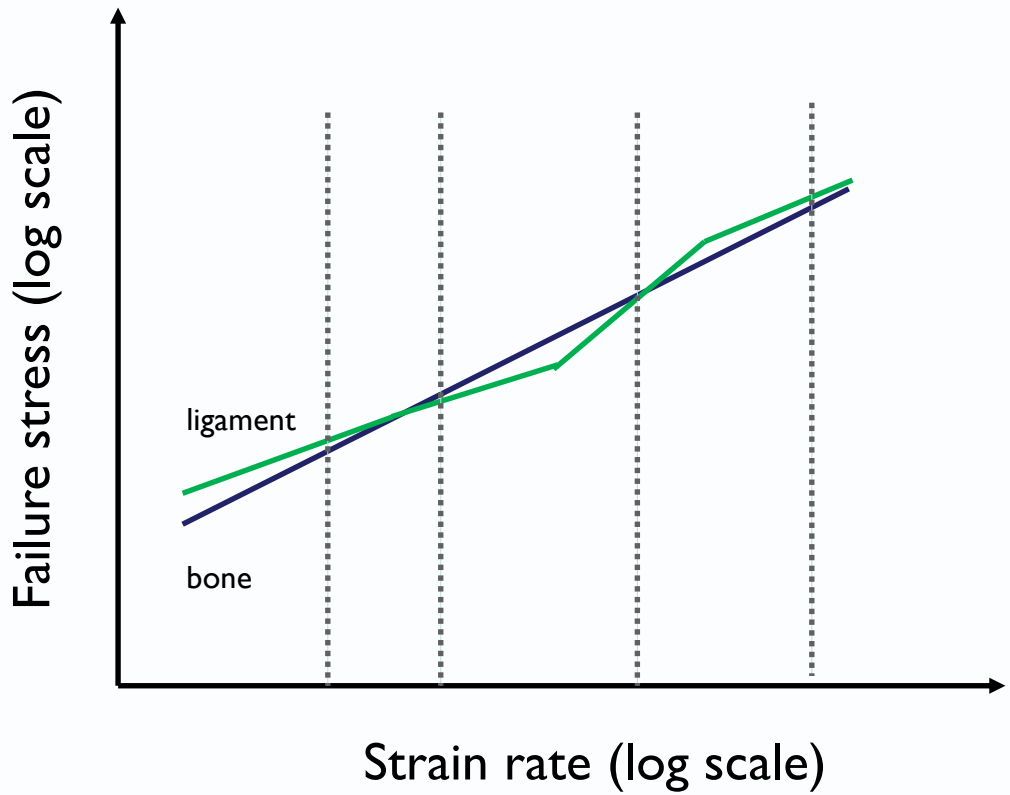


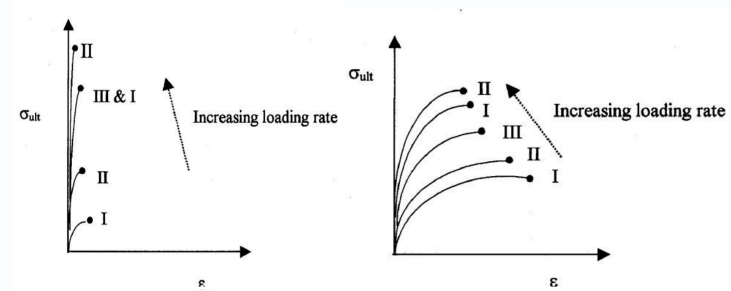
Mode III. Avulsion < Ligament tearing

What might the strain rate dependence of failure stress look like for two materials for which the failure mode changes?



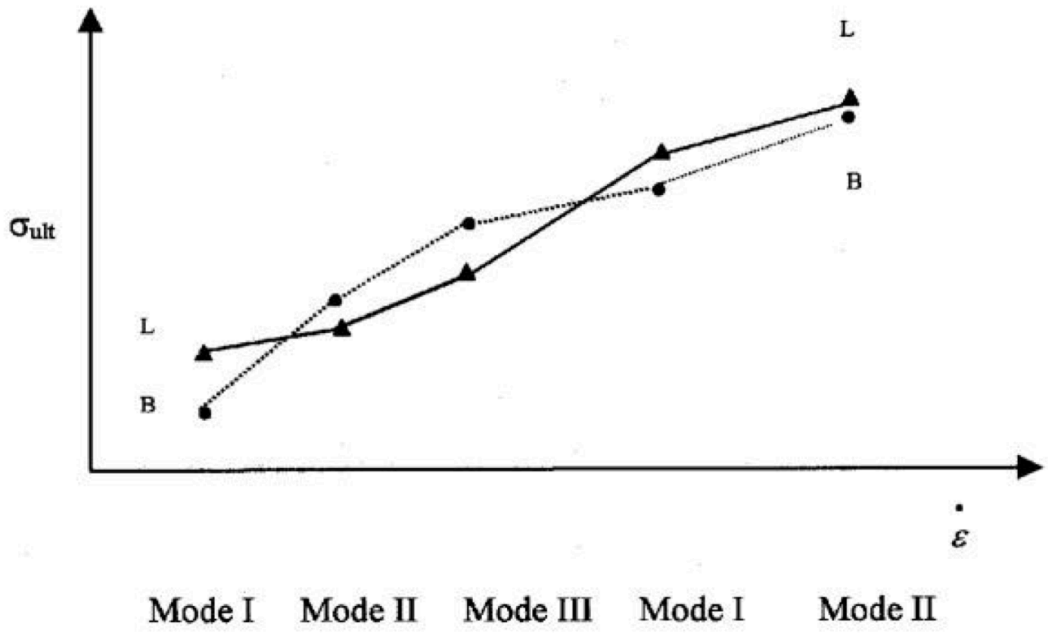
What might the strain rate dependence of failure stress look like for bone and ligament considering this failure analysis?





- Mode I (first)
  - Low strain rates
  - avulsion
- Mode II (first)
  - Tears = avulsion
  - Other parameters in play – individual variation
- Mode III
  - High rates
  - Bone stronger – ligament tears
- At higher rates ligament stronger again
- No quantitative ranges defined
- Evidence that rate dependence is lost for very high rates (100's -1000's)

Summary Hyman model – figure from hyman…



- Mode I (first)
  - Low strain rates
  - avulsion
- Mode II (first)
  - Tears = avulsion
  - Other parameters in play – individual variation
- Mode III
  - High rates
  - Bone stronger – ligament tears
- At *very* high rates ligament stronger again
- No quantitative ranges defined

Is it surprising that failure mode changes? Why?? Why not??

- The B-L-B complex involves two materials with substantially different properties (bone and ligament) plus a transition region (zones 2 and 3)
- Both bone and ligament are strain-rate dependent and the strain rate dependency is not well characterized and is difficult to test. Further, it may differ for different BLBs, species etc.
- *The key is that the relative strain rate dependency should dictate what 'link' fails under certain loading rates.*
- Other factors may also play a role (test conditions, gripping method, species, specific BLB, etc etc).
- **Conclusion – work to be done in this area….but we learned something about material behavior!**