ETH zürich

Connective Tissue

Materials and Mechanics in Medicine HS 2019

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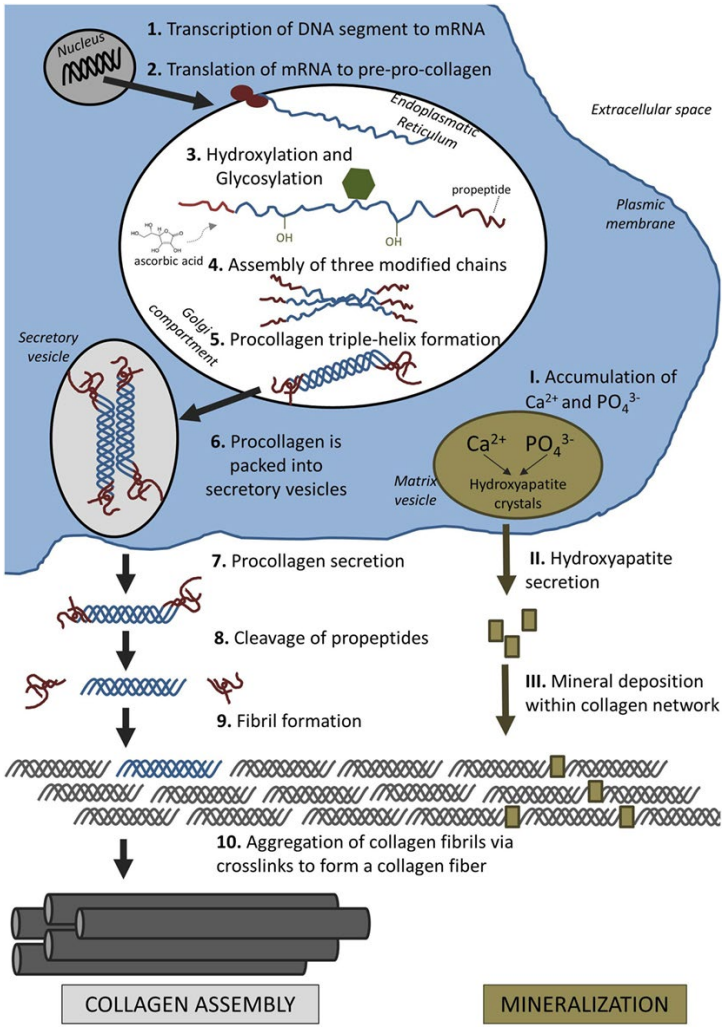
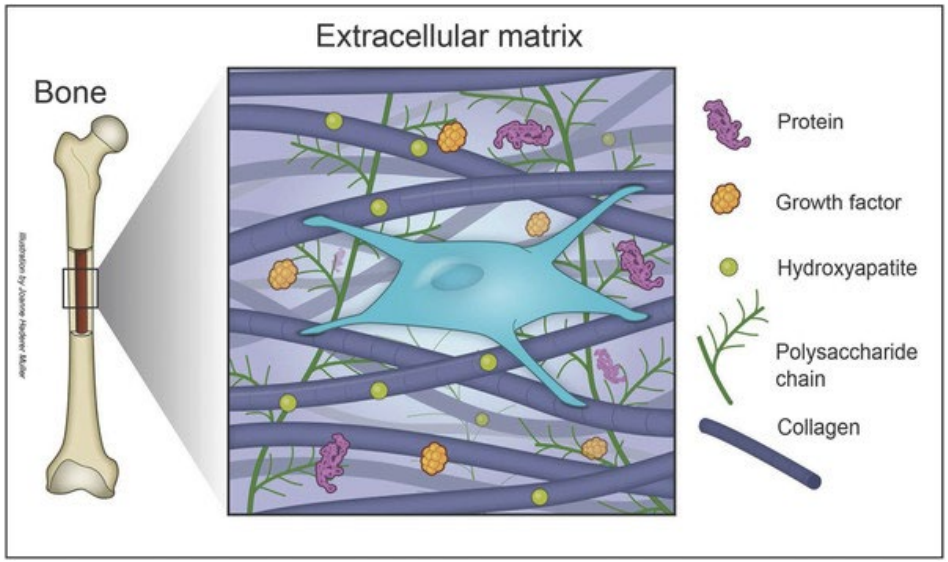
Bone ECM



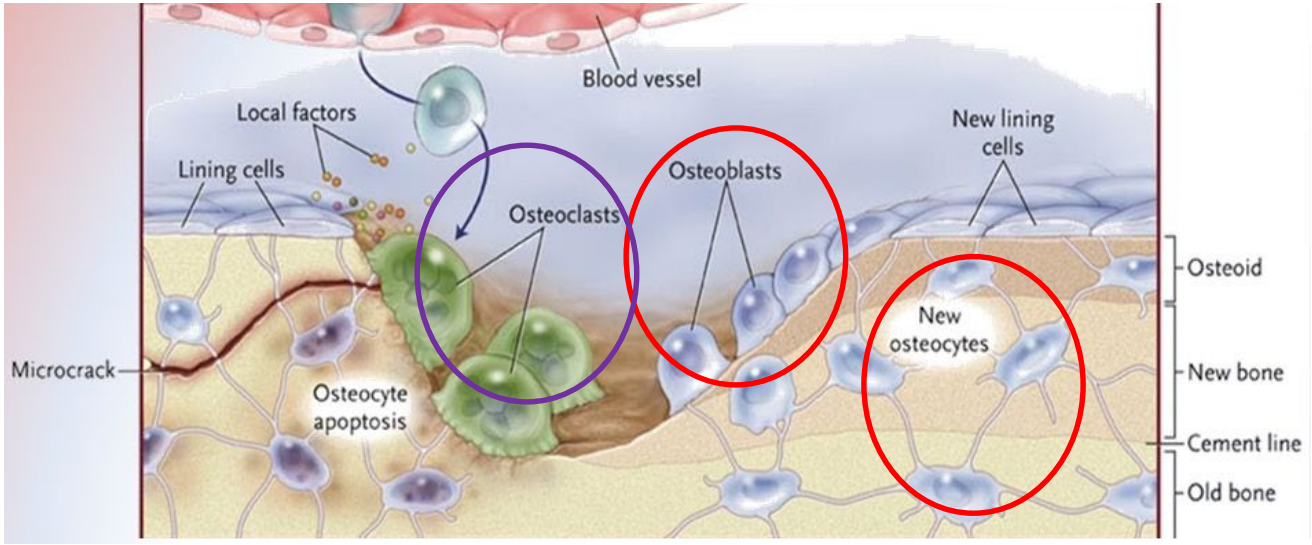


Collagen I: flexible, resilient, organic (27%)

HA: hard, rigid, inorganic (60%)

GAGs: shock absorption & nutrient transport (1.5%)

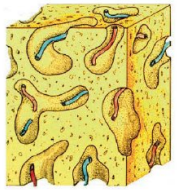
Bone Cells


Blood vessel
Local factors
Lining cells
Osteoclasts
Osteoblasts
New lining cells
Microcrack
Osteocyte apoptosis
New osteocytes
Osteoid
New bone
Cement line
Old bone

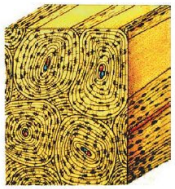
Bone Structure

- All bone has a similar composition, but can be differently structured
- Woven bone: immature bone with randomly oriented fibrils
- Lamellar bone: mature bone with highly orientated fibers arranged

Woven bone

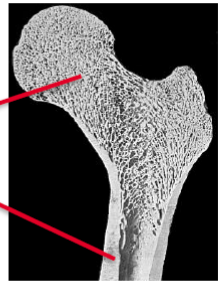


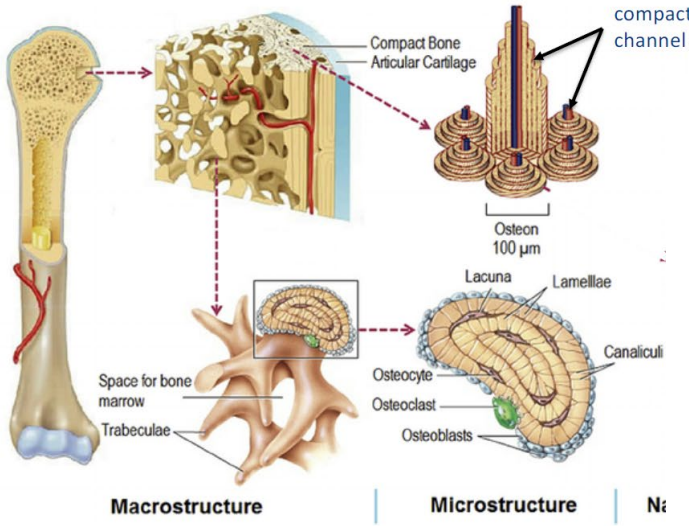
Lamellar bone



Trabecular bone

Cortical bone

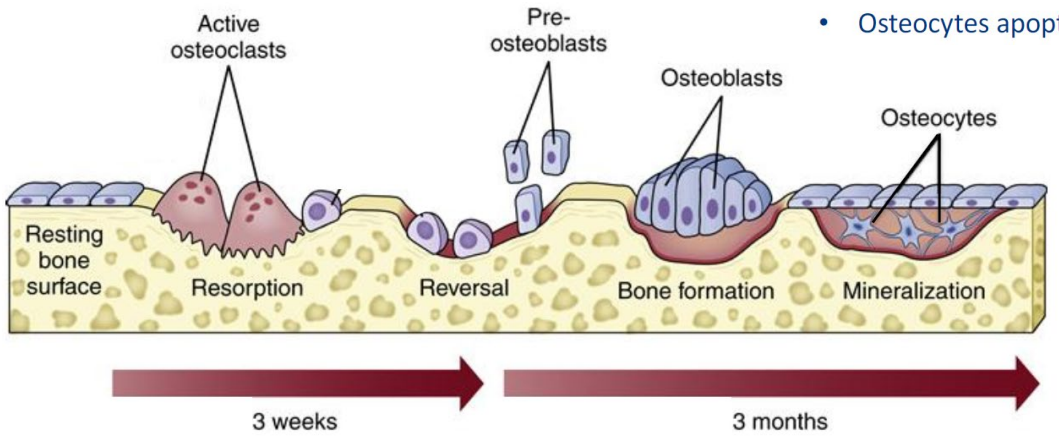




- Cortical bone (compact bone), the dense cortex
- Trabecular bone (cancellous bone, spongy bone), the inner porous structure

Bone Remodeling

- Living osteocytes inhibit bone resorption
- Osteocytes apoptose from microcrack formation or nutrient deprovation



Bone remodeling cycle

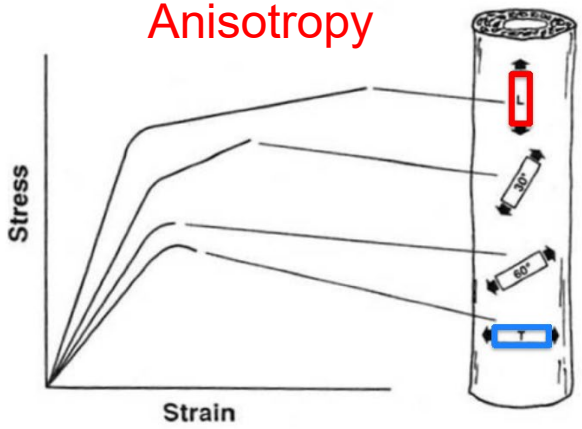
- Reduces collagen cross-linking, keeping our bones 'young and tough'

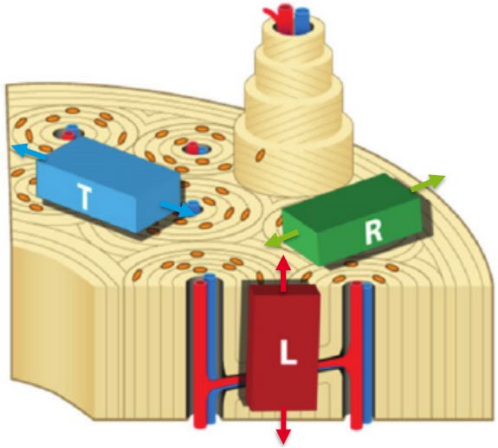
1. First, osteoclasts begin bone resorption, creating the pit.
2. Then osteoblasts enter the pit and produce bone matrix, which then calcifies
3. Osteoblasts become bone lining cells or osteocytes that reside in the ECM

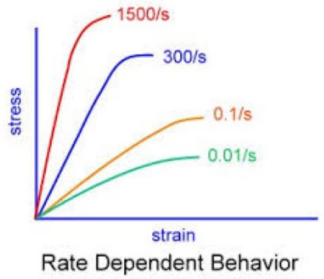
- Osteocyte apoptosis (nutrient deprivation / μcracks) initiates bone remodeling
- Osteoblasts lay down osteoid, which remodels into lamellar bone
- Trabeculae are remodeled appositionally / Cortical bone via 'cone digging'
- Bone remodeling is load-driven (more load = more bone, no load = less bone) Wolff's law

Bone Mechanical Properties

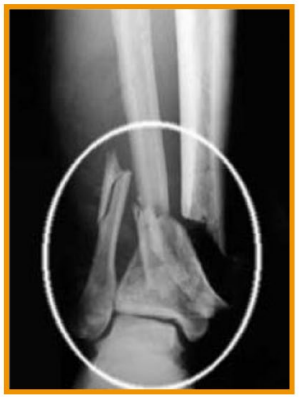
Anisotropy

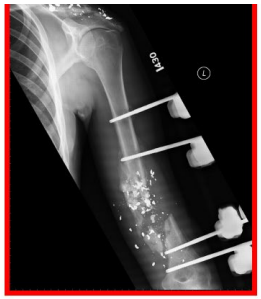


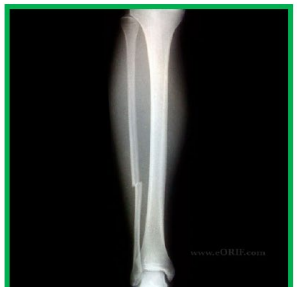




Rate Dependent Behavior

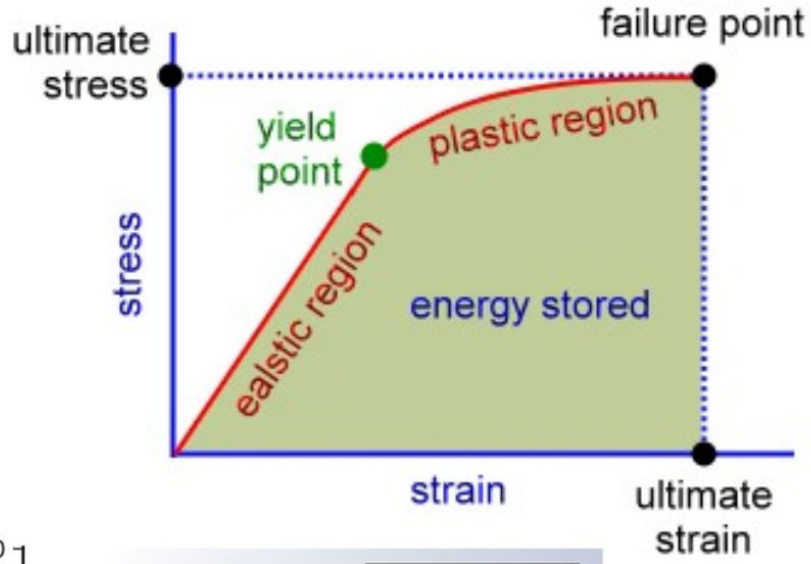


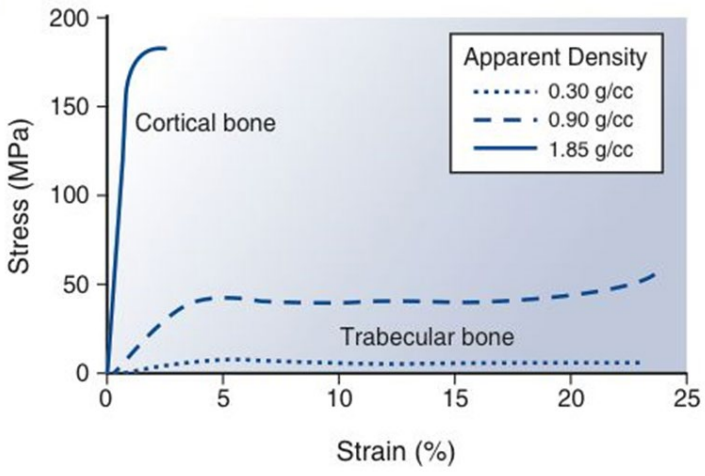




- Fracture viscoelasticity ≈ energy release
- higher strain rates equal complicated fractures and more tissue damage

Recap: Stress-Strain Curve





- Elastic region: capacity to return to original shape
- Yield point: until this point, deformation is reversible
- Plastic region: residual deformation is permanent
- Failure point: structure will fail, i.e. bone will fracture
- Ultimate stress: failure stress
- Ultimate strain: failure strain
- Energy stored: toughness

trabecular vs coritcal bone?

Cartilage Composition

Collagen
- 15-22% vol/vol
- Primarily type II

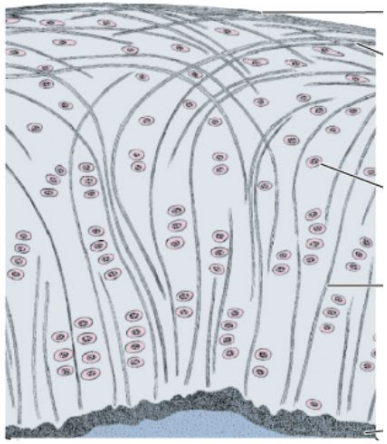
Proteoglycans
- 4-7% vol/vol
- Primarily aggrecan

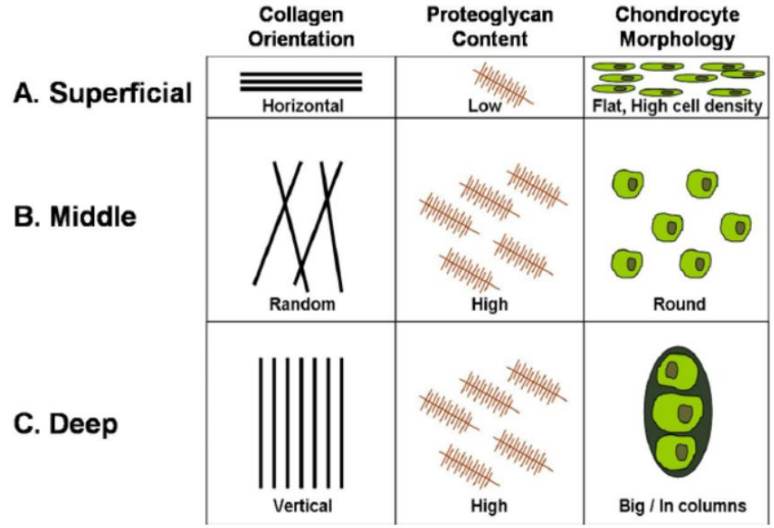
Water
- 60-85% vol/vol

Chondrocytes
- < 10% vol/vol
- Low density

No blood vessels

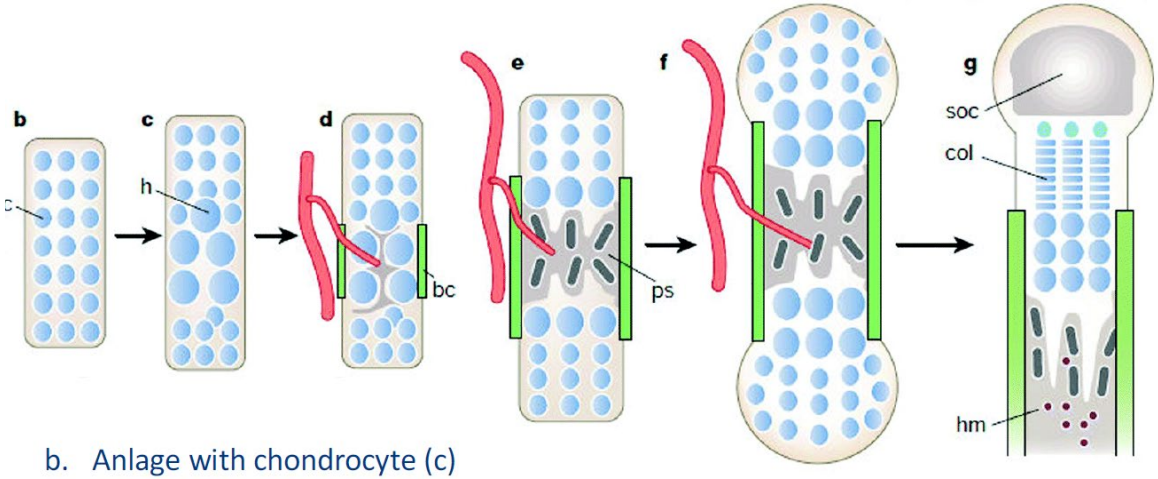
No innervation



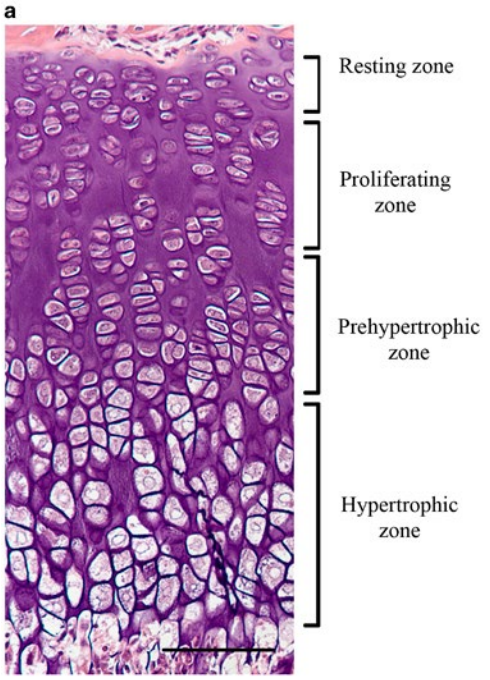


Type II collagen is arranged as 'Benninghoff Arcades'
- Enable swelling pressure
- Resist superficial tension

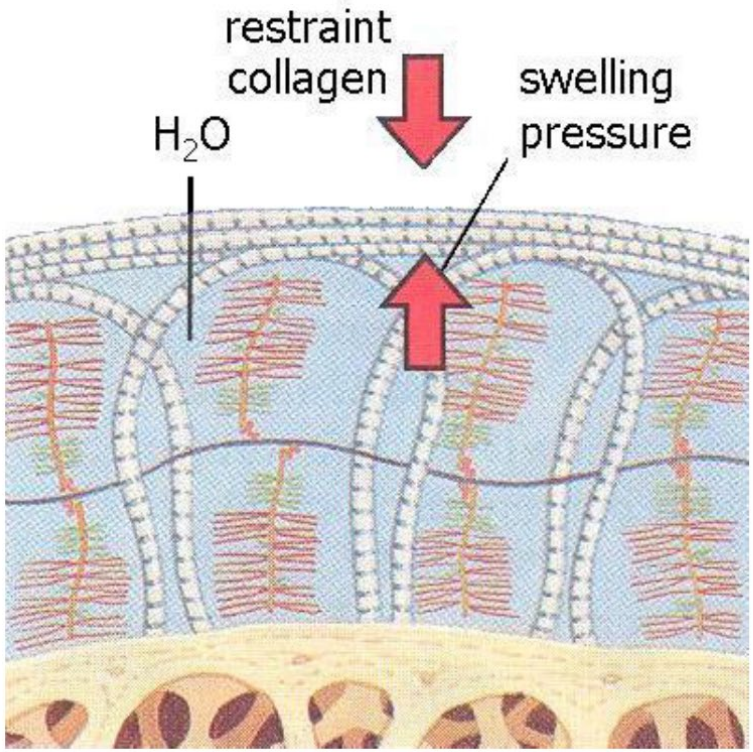
Cartilage/Bone Development



b. Anlage with chondrocyte (c)
c. Chondrocytes hypertrophy (h) and recruitment of blood vessels
d. Bone collar (bc) is formed from periosteum, vascularization
e. Primary ossification center (ps) is formed
f. Chondrocytes proliferate and lengthen bone
g. Secondary ossification center (soc) forms and growth plate forms with typical columns of proliferating chondrocytes (col), articular cartilage develops similar distribution



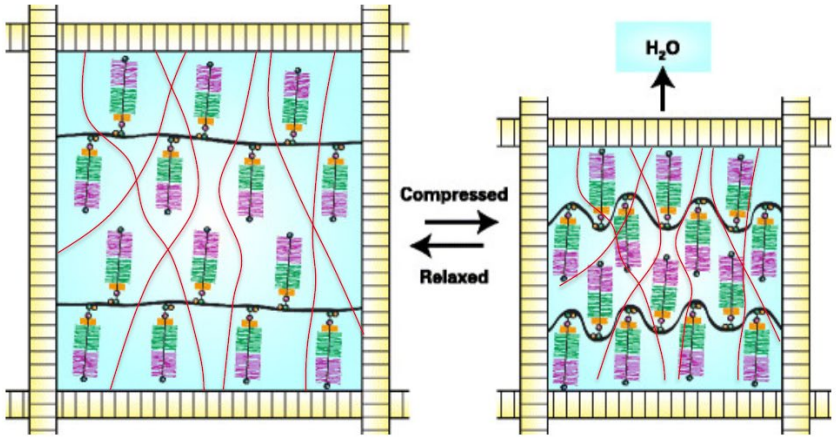
Cartilage Load-Bearing: Swelling Pressure



1. Proteoglycans have fixed negative charges
2. Positive ions are attracted
3. This increases osmotic pressure
4. Is neutralized by absorbing water
5. Creates swelling of the tissue
6. Collagen resists swelling
7. Results in swelling pressure

Cartilage Load-Bearing: Donnan Effect

Collagen Hyaluronan Keratan sulfate GAGs: Chondroitin sulfate 1 & Chondroitin sulfate 2



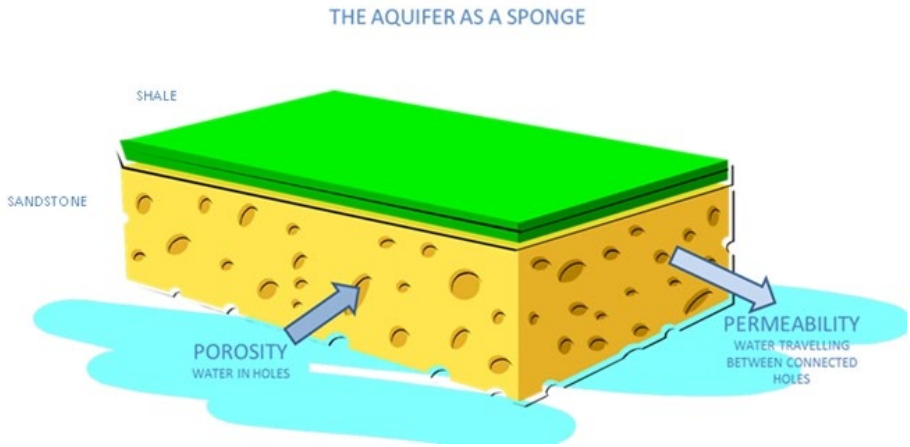
- Proteoglycans link to collagen
- Swelling draws water into the tissue until equilibrium
- Swelling is balanced by tensile forces in the collagen fibrils

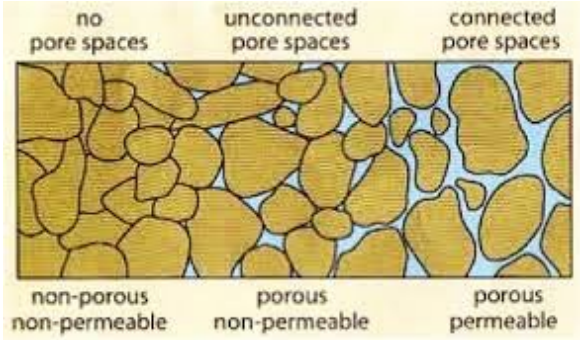
- Compression → water exudation → proteoglycans are closer → increased FCD & swelling potential → new equilibrium
- Reversible: removal of load restores original equilibrium

Cartilage Load-Bearing

- *Permeability = resistance to fluid flow =* $\frac{\textit{fluid speed}}{\textit{pressure gradient}}$ $(m^4/N{\cdot}s)$
- *With pressure gradient* $\approx \frac{P_1 - P_2}{h}$
- Permeability decreases with increasing strain
- Permeability decreases with increasing ΔP (frictional drag)

Healthy Cartilage:
high porosity and **low** permeability

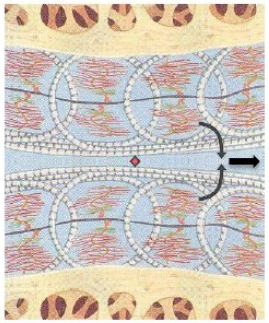




Cartilage Mechanical Properties

With cartilage compression

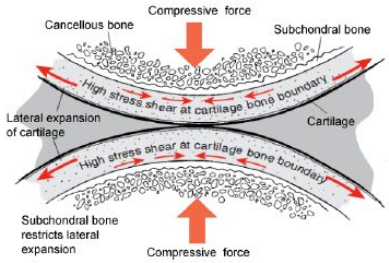
1. Efflux of interstitial fluid
2. Redistribution of fluid, free molecules and ECM
3. New equilibrium
4. Increased Fixed Charge Density, stiffness ↗



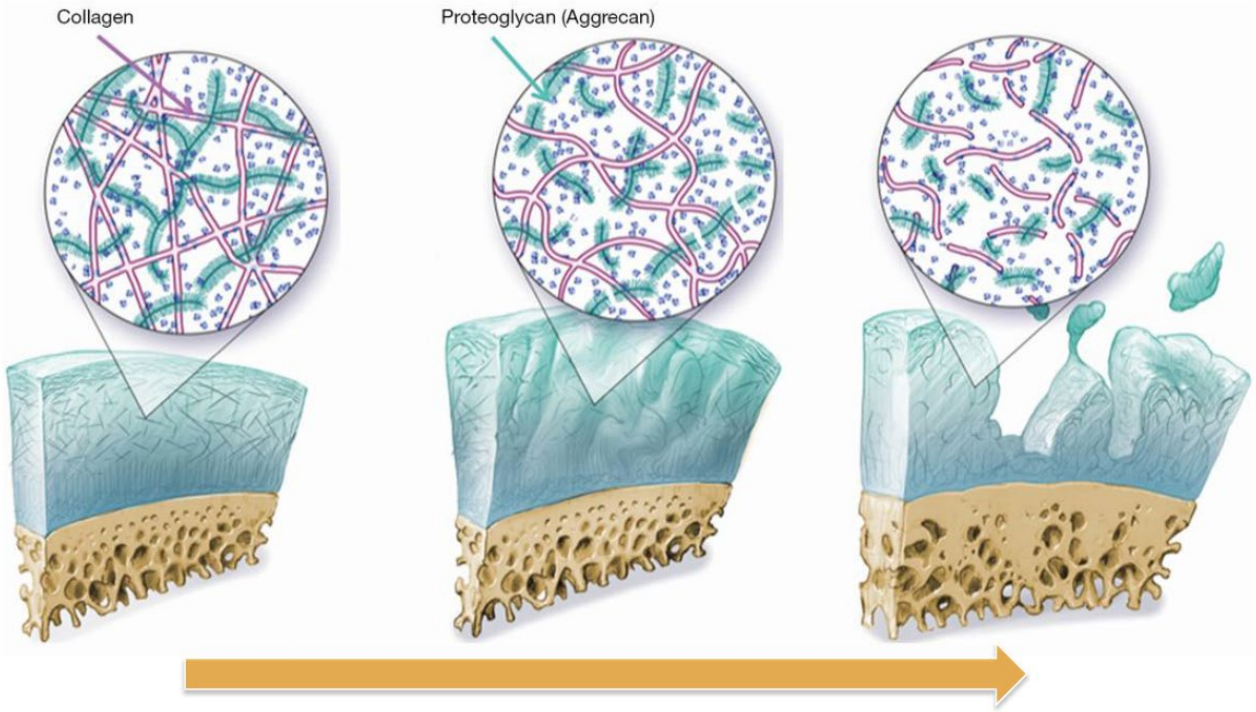
creep, stress relaxation

Shear forces

- Surprisingly high in articular cartilage
- Shear stiffness originates from collagen & collagen-proteoglycan interaction



Cartilage Degeneration



1. Disruption of collagen – GAG ECM
2. Tissue swelling
3. Proteoglycan washout
4. Increased permeability
5. Decreased cartilage stiffness
6. Tissue fibrillation
7. Increased shear stress & wear
8. Subchondral bone thickening

Cartilage Repair

- Cartilage has very limited repair capacity
  - Low cell number
  - Low metabolism
  - Avascular
- Surgical techniques:
  - Microfracture
  - Lesion filling
  - Temporary joint distraction
  - *Implants (treated in coming lectures)*