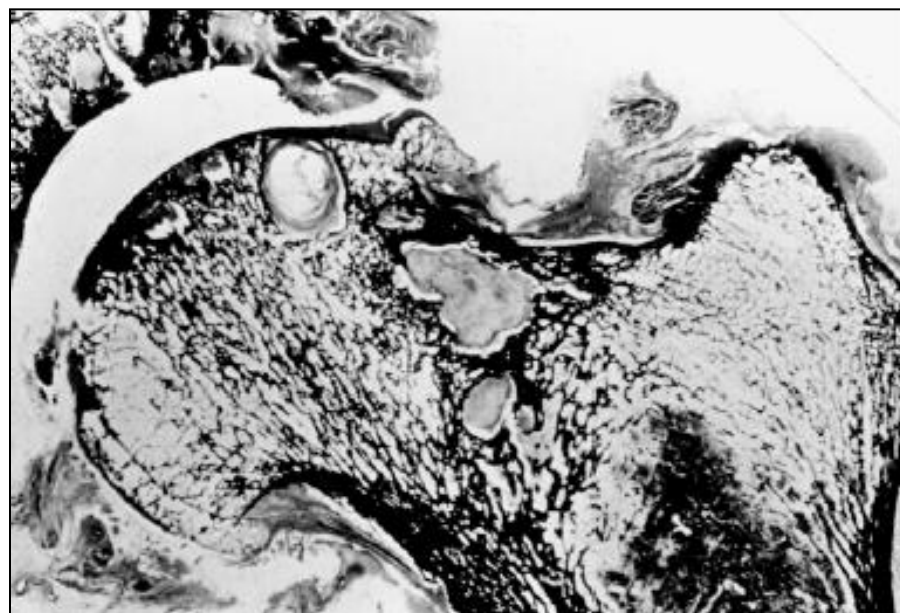




# Degenerative joint disease (DJD)

**รศ.นพ.อารี ตनावลี**  
**Aree Tanavalee, MD**

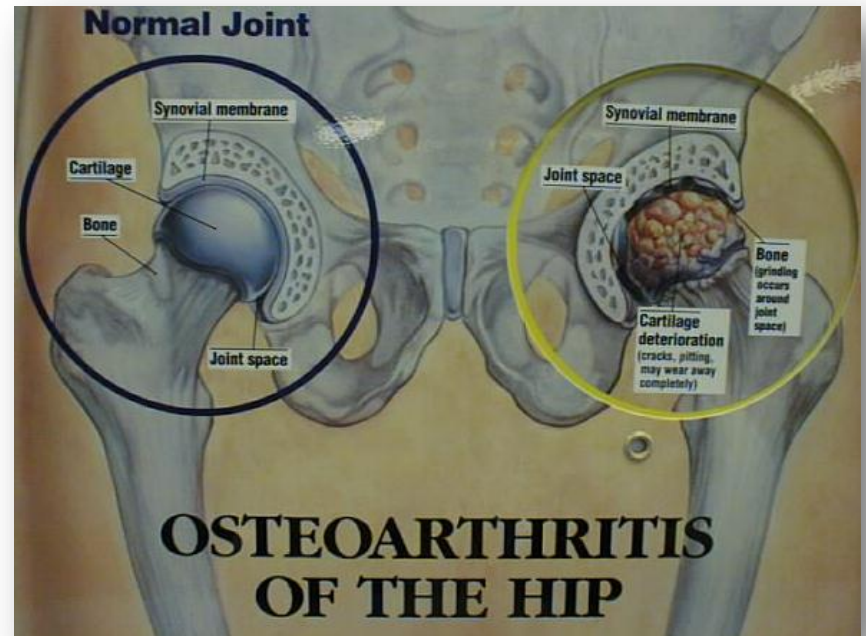
**Associate Professor**  
**Department of**  
**Orthopaedics**  
**Faculty of Medicine**  
**Chulalongkorn University**





# What is DJD?

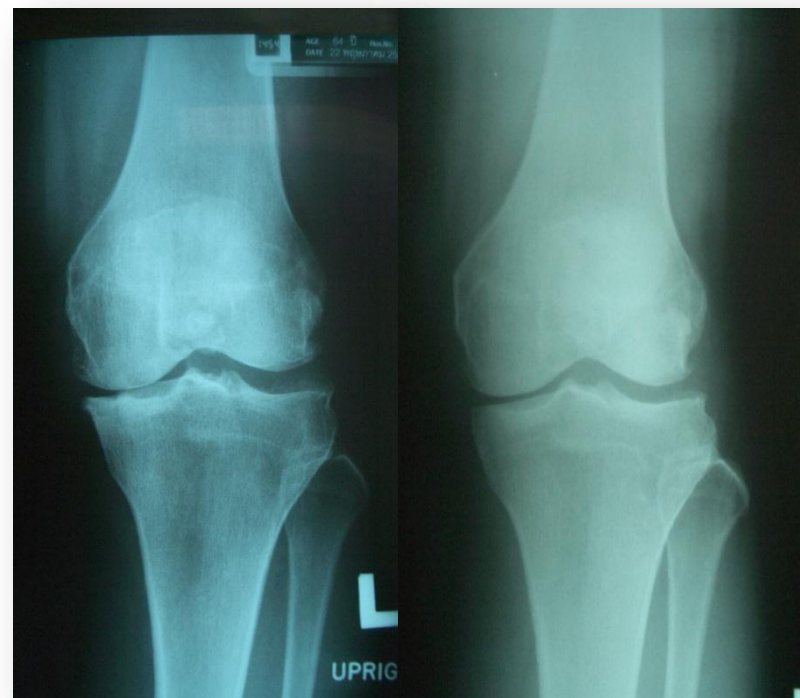
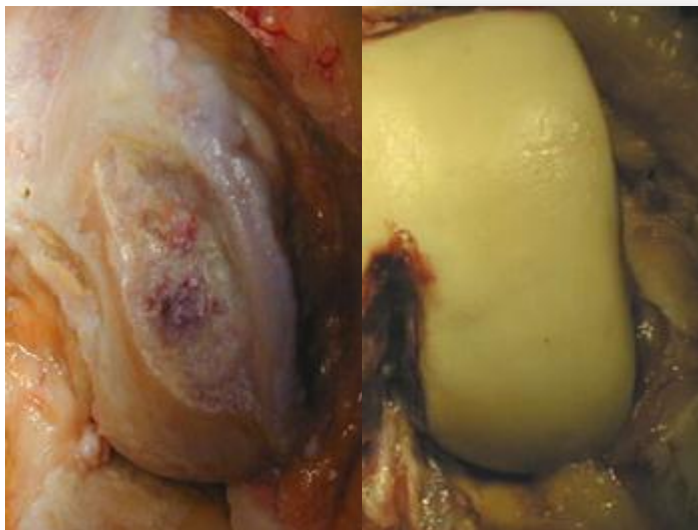
- Commonly known
  - Osteoarthritis, Osteoarthrosis
- Disease character:
  - Different etiologies
  - Similar findings
    - Biologic
    - Morphologic
    - Clinical outcomes





# What is DJD?

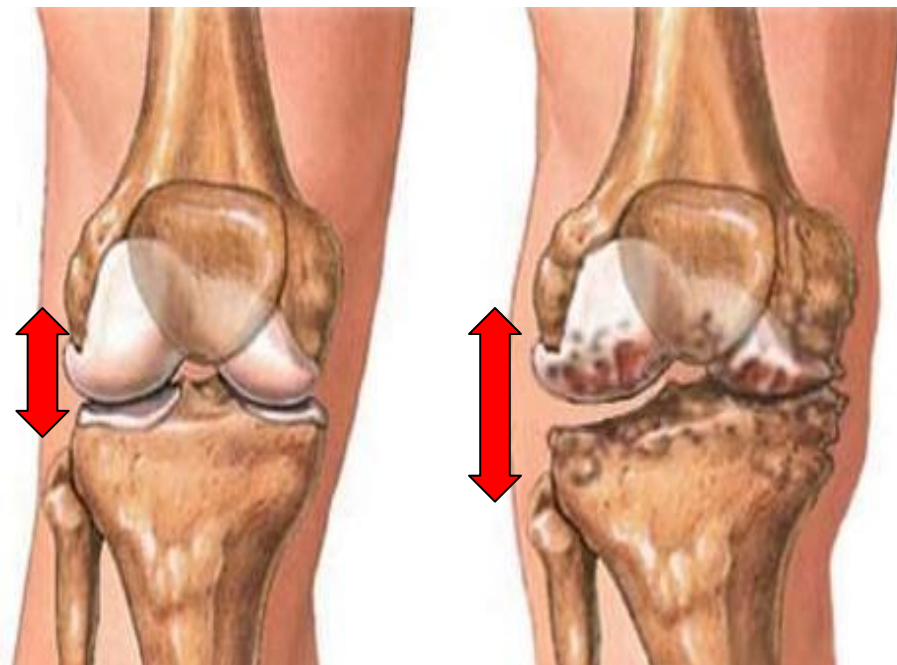
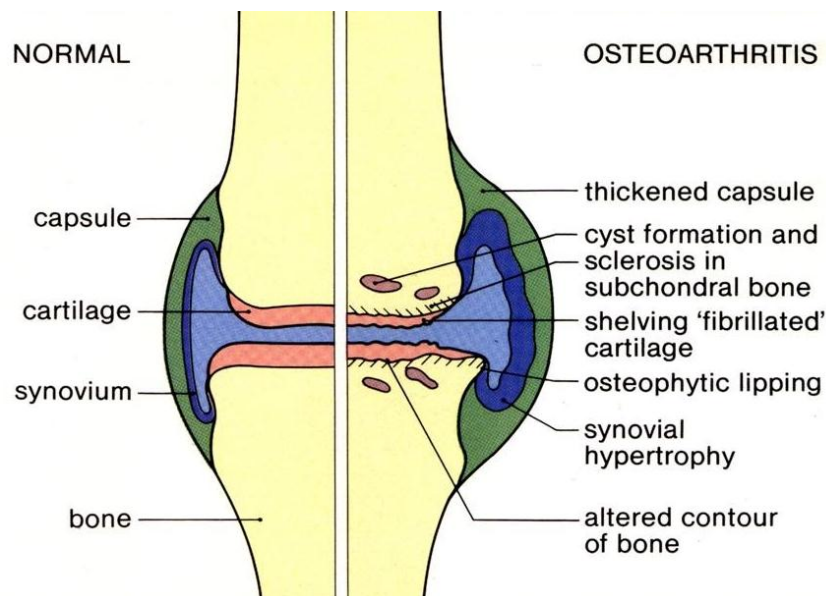
- Articular cartilage
  - Progressive loss
- Subchondral bone
  - Remodeling, sclerosis
  - Bone cysts, osteophytes





# What is DJD?

- Ligament
  - Attenuation
- Synovium and capsule
  - Thickening

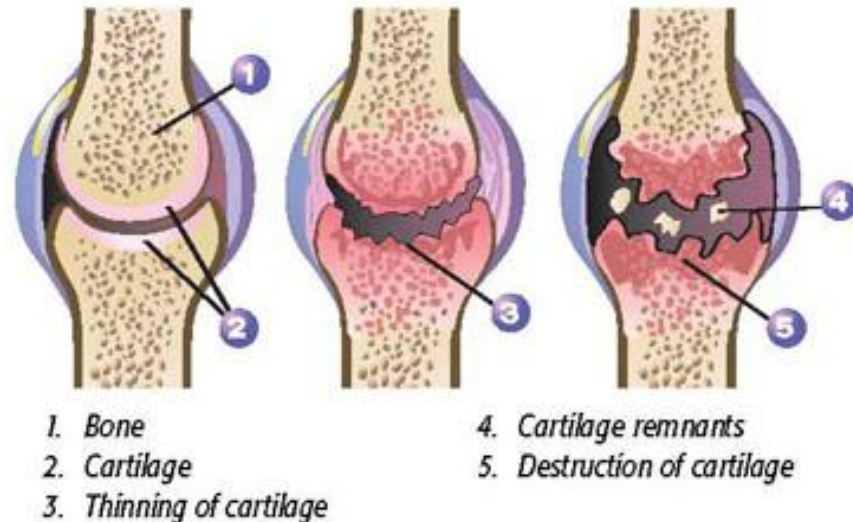






# Definition: DJD (Osteoarthritis)

- **Progressive disorder of the joints caused by**
  - **Gradual loss of cartilage**
  - **Reactive changes of bone**
- **Leading to joint**
  - **Destruction**
  - **Painful malfunction**





# Prevalence

- **Clinically defined OA.**
  - on the basis of symptoms and physical examination findings
  - Ages 25–74years
    - 12.1% of the US population
- **Radiographically defined OA**
  - According to the Kellgren/Lawrence scale (presence of osteophytes)



# Symptomatic OA

Anatomic site, age, years	Source (ref.)	% with symptomatic OA		
		Male	Female	Total
Hands, $\geq 26$	Framingham OA study (6)	3.8	9.2	6.8
Knees				
$\geq 26$	Framingham OA study (5)	4.6	4.9	4.9
$\geq 45$	Framingham OA study (5)	5.9	7.2	6.7
$\geq 45$	Johnston County OA Project (7)	13.5	18.7	16.7
$\geq 60$	NHANES III (4)	10.0	13.6	12.1
Hips, $\geq 45$	Johnston County OA Project (10)	8.7	9.3	9.2

Lawrence RC, Felson DT, Helmick CG, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II. Arthritis Rheum 2008;58(1):26–35.



# Radiographic OA

Anatomic site, age, years	Source (ref.)	% with mild, moderate, or severe OA		
		Male	Female	Total
Hands, $\geq 26$	Framingham OA study (6)	25.9	28.2	27.2
Knees†				
$\geq 26$	Framingham OA study (5)	14.1	13.7	13.8
$\geq 45$	Framingham OA study (5)	18.6	19.3	19.2
$\geq 45$	Johnston County OA Project (7)	24.3	30.1	27.8
$\geq 60$	NHANES III (4)	31.2	42.1	37.4
Hips, $\geq 45$	Johnston County OA Project (10)	25.7	26.9	27.0

Lawrence RC, Felson DT, Helmick CG, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II. Arthritis Rheum 2008;58(1):26–35.





# Comparing OA diagnosis

		S	R	
Anatomic site, age, years	Source (ref.)	Total	Total	
Hands, ≥26	Framingham OA study (6)	6.8	27.2	7
Knees				
≥26	Framingham OA study (5)	4.9	13.8	3
≥45	Framingham OA study (5)	6.7	19.2	
≥45	Johnston County OA Project (7)	16.7	27.8	
≥60	NHANES III (4)	12.1	37.4	3
Hips, ≥45	Johnston County OA Project (10)	9.2	27.0	



# Higher prevalence of OA

- **Genetic mutation**
  - Mutation in type II collagen
- **Heritability**
  - Radiographic OA of hands & knees
  - 39-65%
- **Knee**
  - History of meniscectomy
  - Repetitive kneeling and squatting



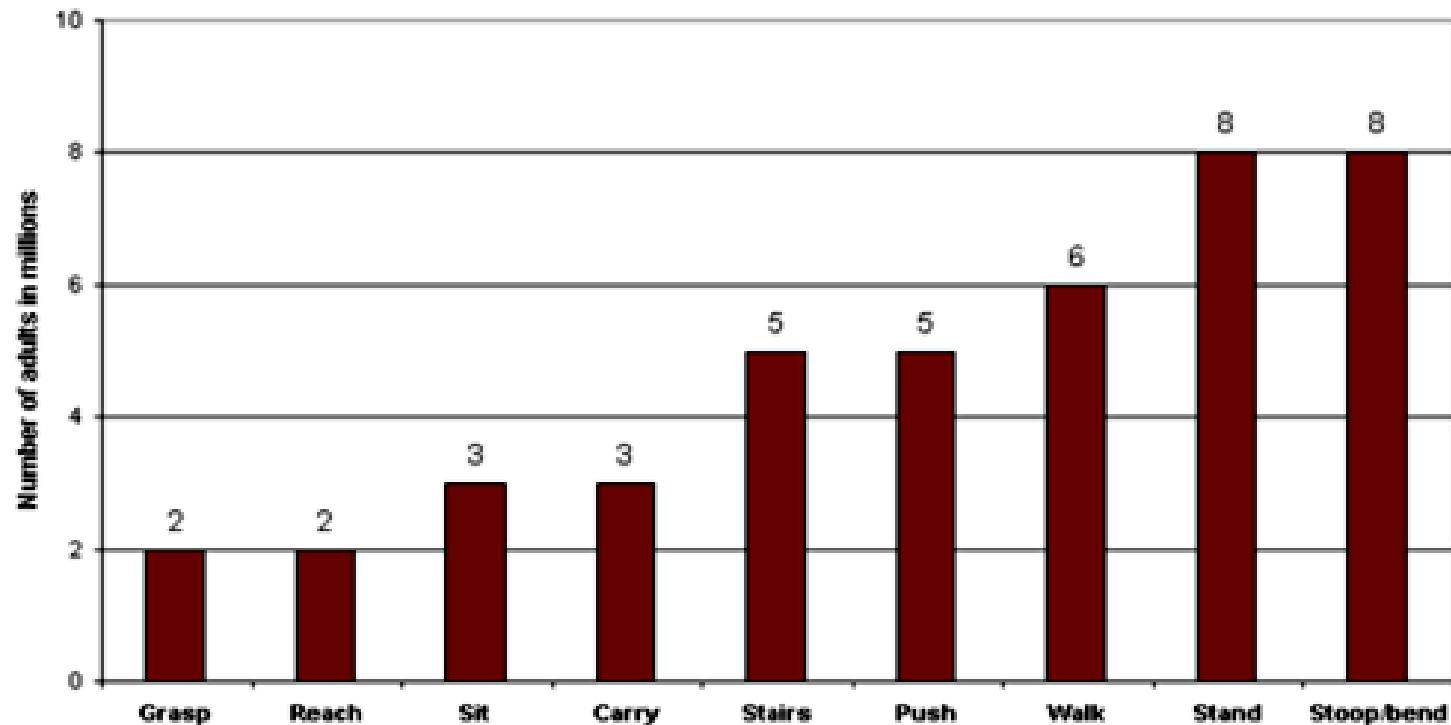
# Fact of OA

- **Obvious gross evidence**
  - Radiological OA in  $\geq 3$  joints
    - > 20% no history of joint pain
- **No gross evidence**
  - Etiology of pain is elusive
    - MRI & pain
      - Effusion
      - Bone edema
      - Thickened synovium





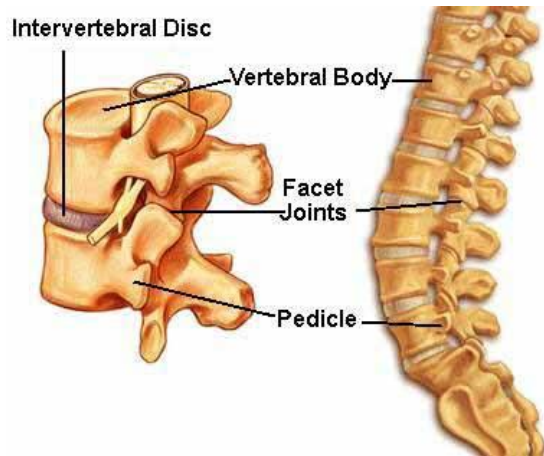
# Functional limitation caused by DJD





# Normal joint

- Painless
- Mobile
- Stable

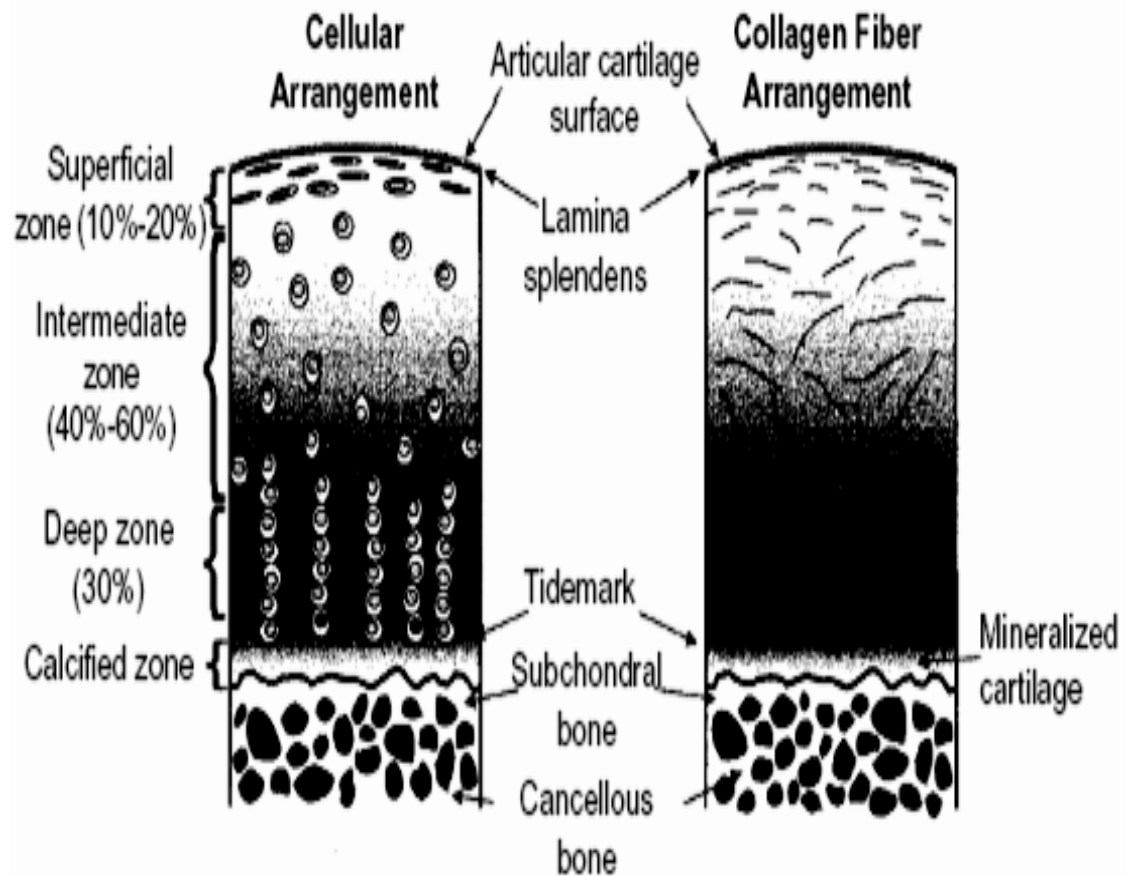






# Mature articular cartilage

- 5 layers
  - Avascular
  - Aneural
  - Alymphatic
- Matrix
- Chondrocyte
- Nutrients
  - Diffusion
  - Synovial fluid



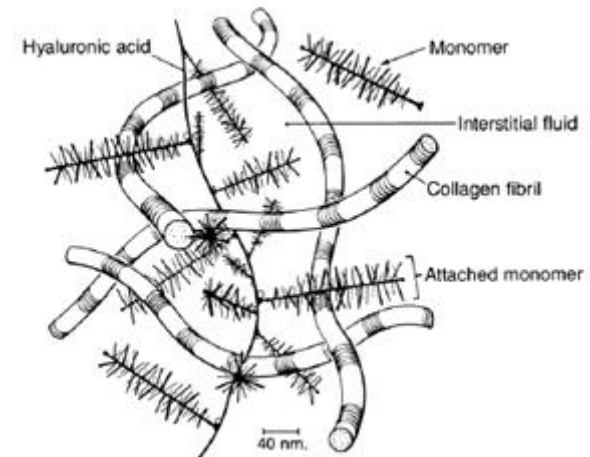
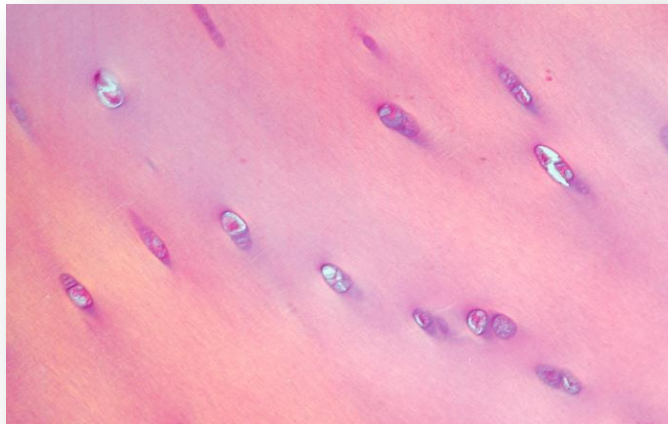
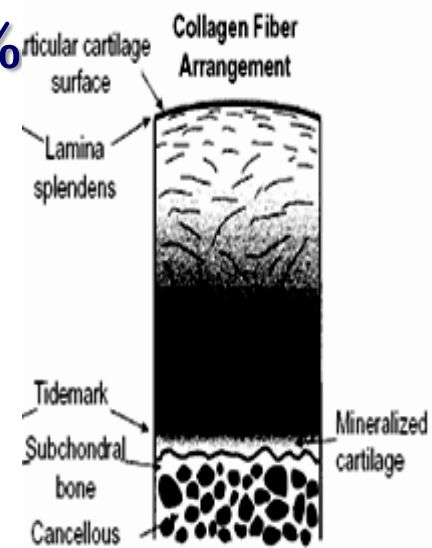


# Articular cartilage structure

- **Complex extracellular matrix 95%**

- **Water: 65-80%**
- **Collagen 10-20%**
  - **Type II 90-95%**
  - **Type V, VI, IX, X and XI**
- **Proteoglycans 10-15%**

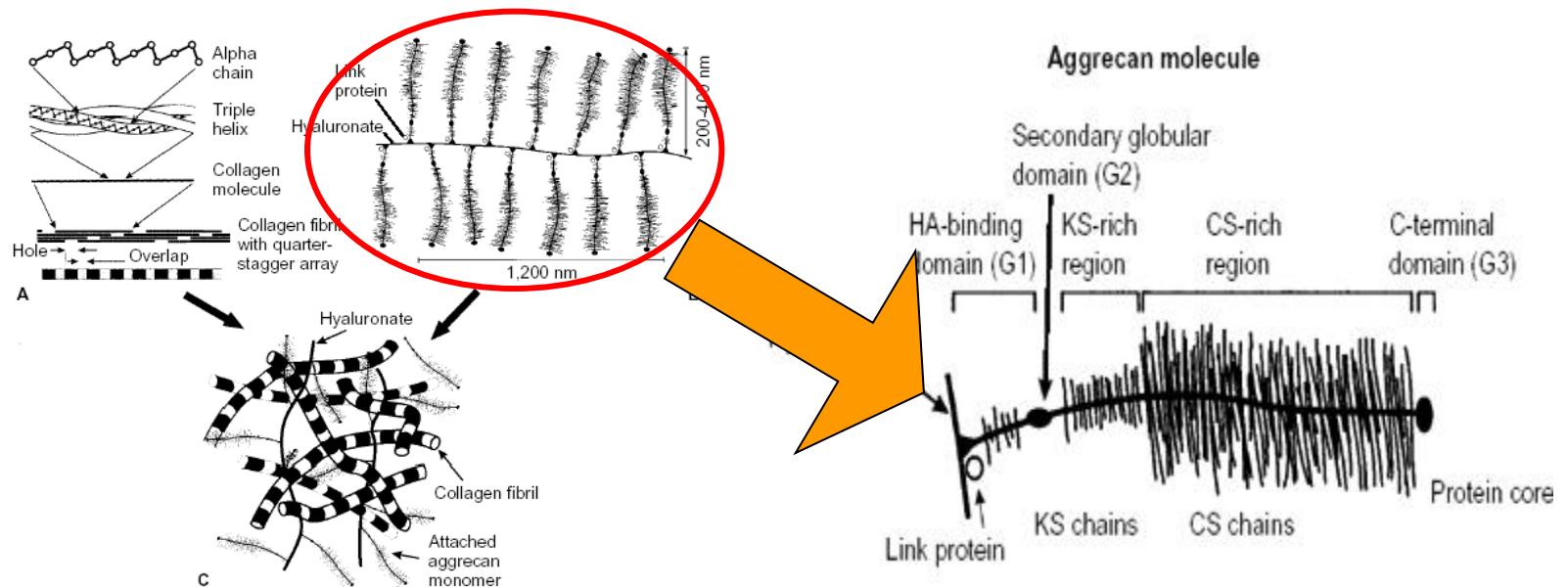
- **Cellular component 5%**





# Proteoglycans

- Large macromolecules
- Produced by chondrocyte
- Chains of glycosaminoglycans (GAGs) attached to a linear core protein

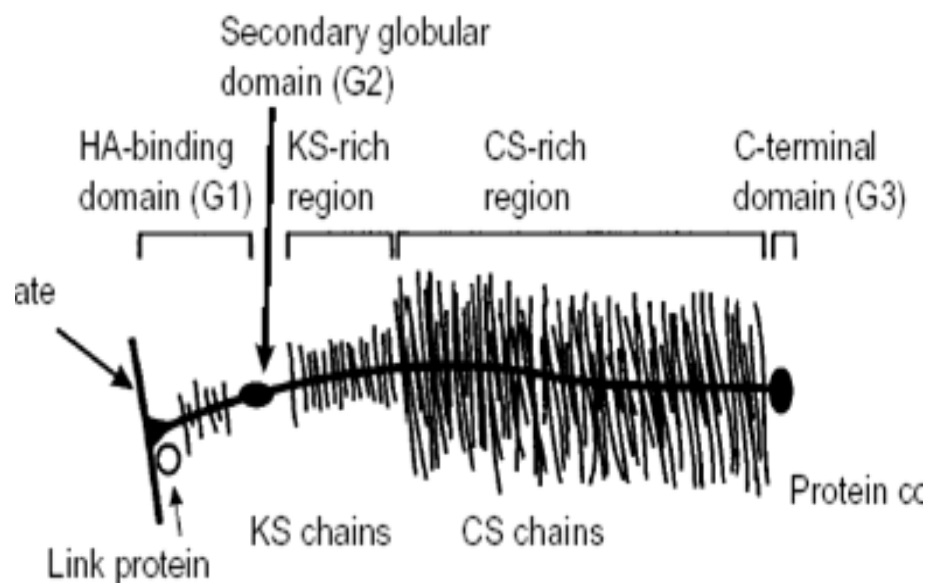




# Glycosaminoglycans (GAGs)

- **Disaccharide polymer**

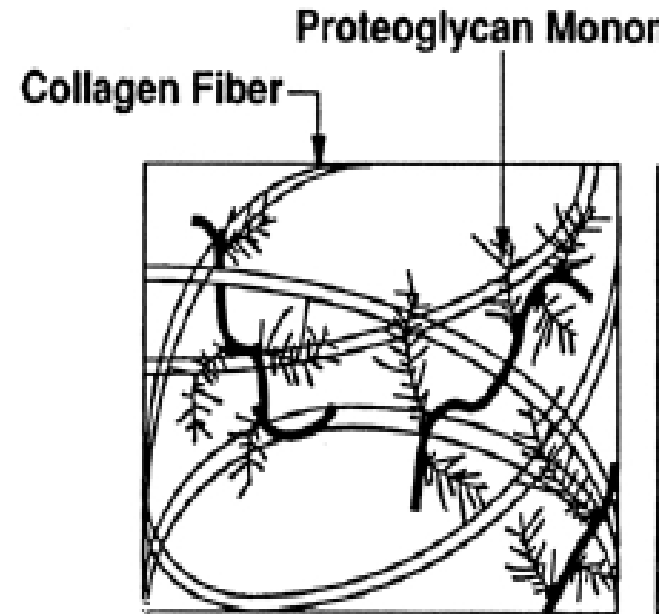
- **Chondroitin sulfate**
- **Keratan sulfate**
- **Dermatan sulfate**
- **Heparan sulfate**
- **Hyaluronic acid**





# Articular cartilage function

- **Water**
  - Deformation of cartilage surface due to stress
  - Nutrition
- **Collagen**
  - Framework strength
  - Tensile strength
- **Proteoglycans or aggrecan molecules**
  - Negatively charged hydrophilic
  - Compressive strength
- **Chondrocytes**
  - Matrix and collagen

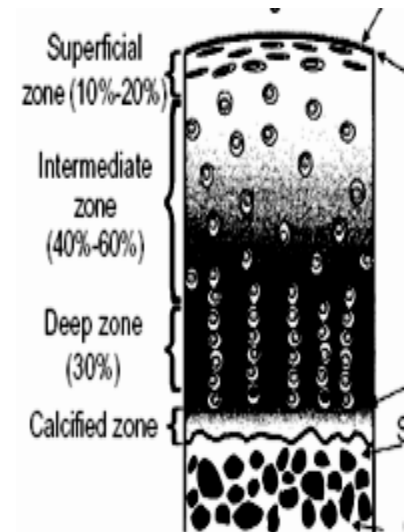
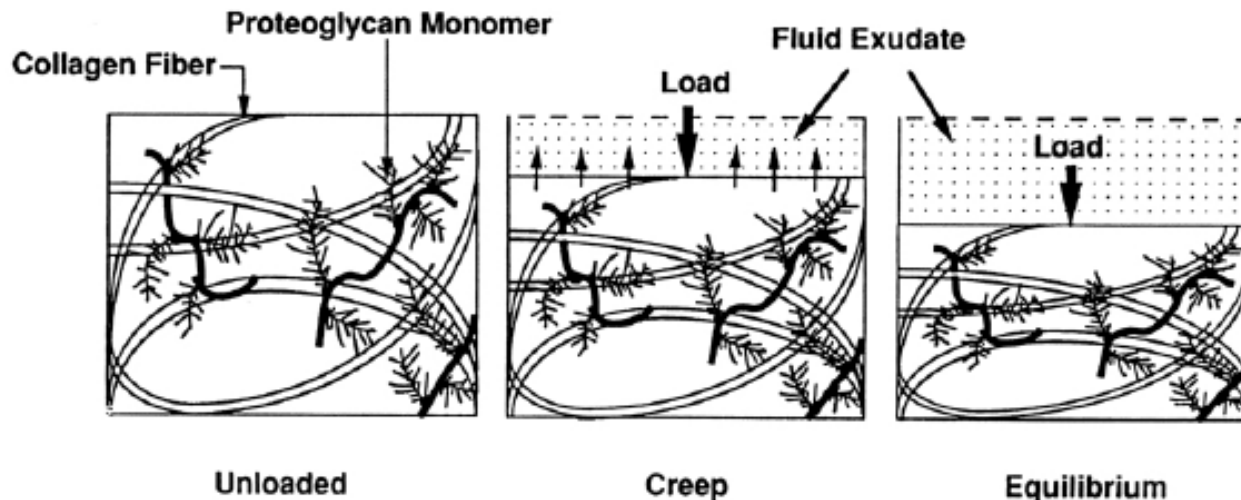






# Normal articular cartilage

- Load distribution
- Decrease friction
- Resistance to compressive, tensile, and shear forces
- Minimizing loads on subchondral bone

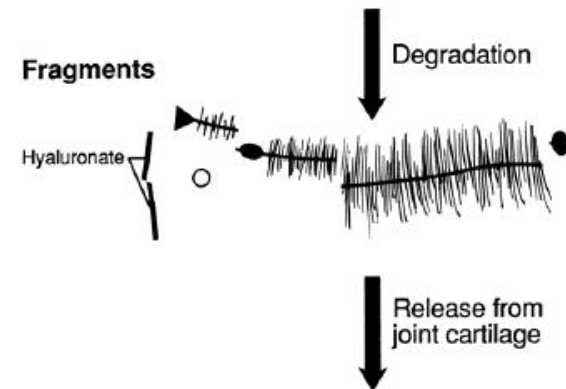
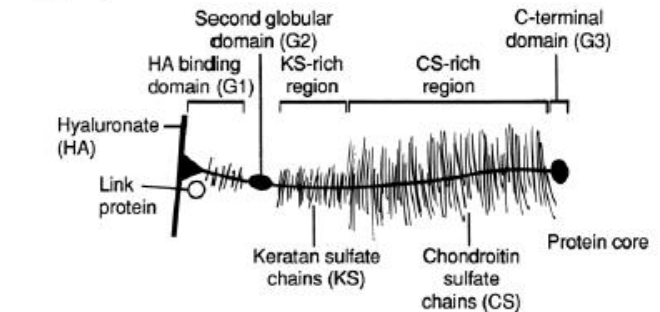




# What causes DJD?

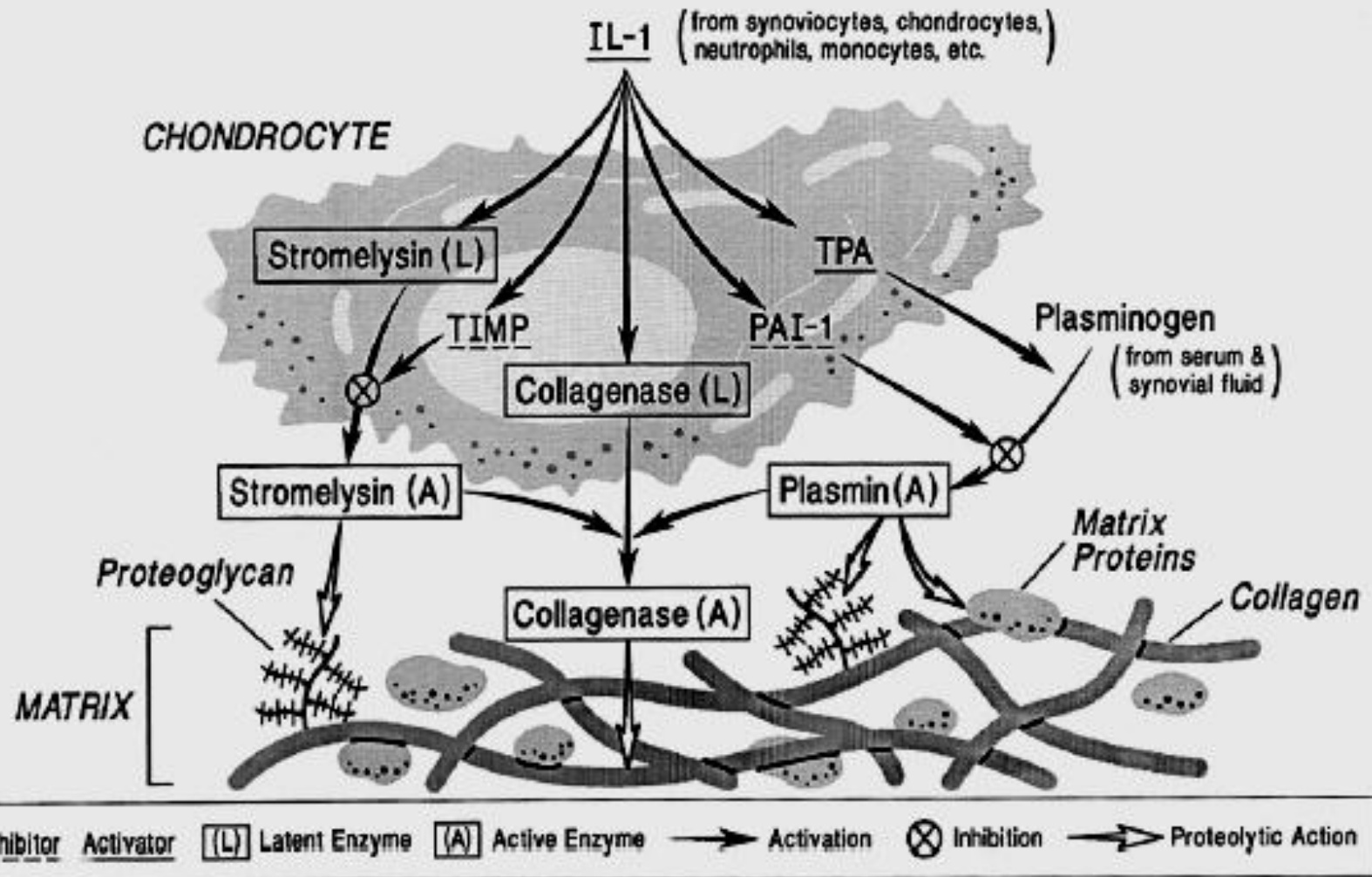
- Unknown cause
- Proteoglycans
  - Increase >> decrease
  - Shorter chain
- Water
  - Increase
- Biomechanical properties
  - Decrease

**Proteoglycan Aggregate**



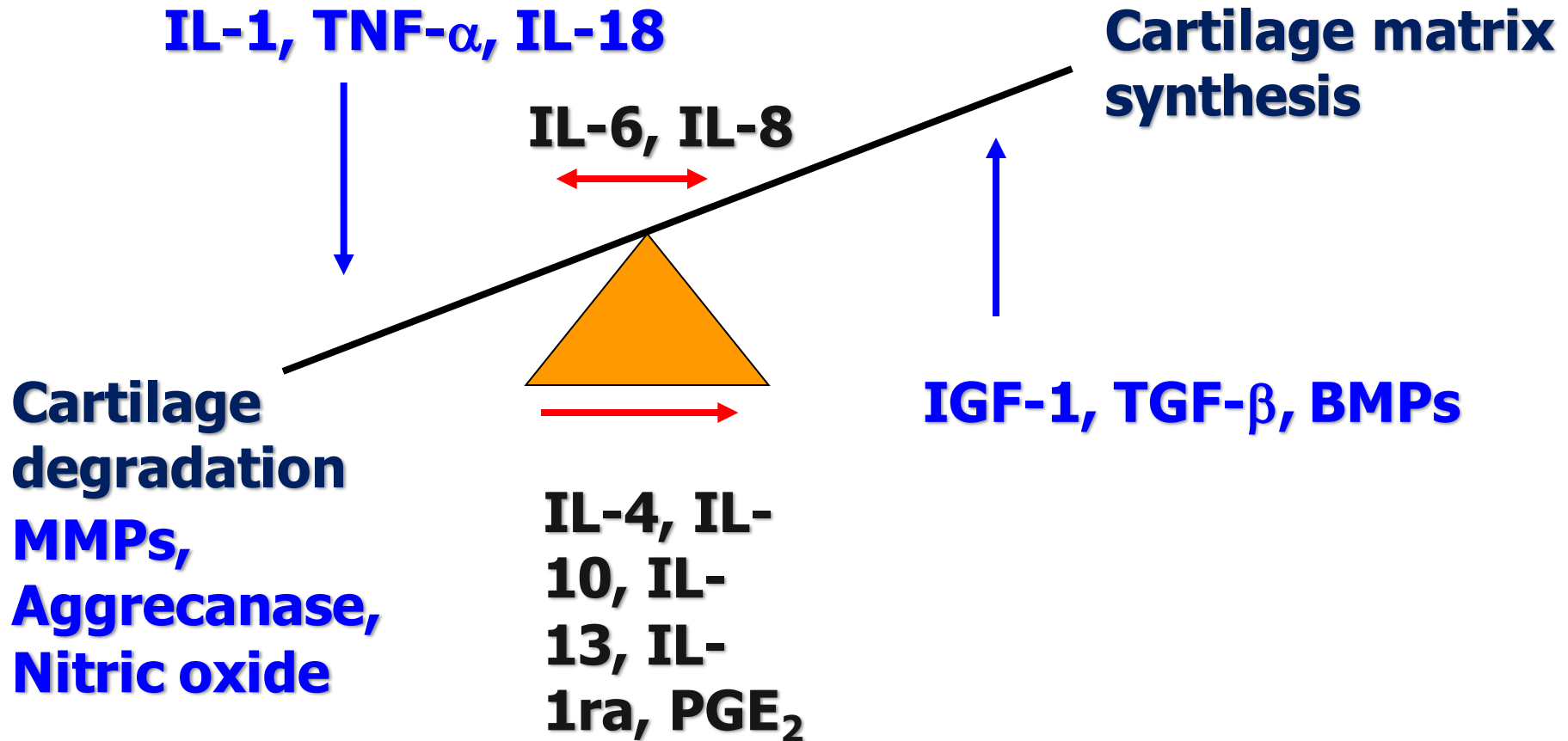


# Pathology of DJD





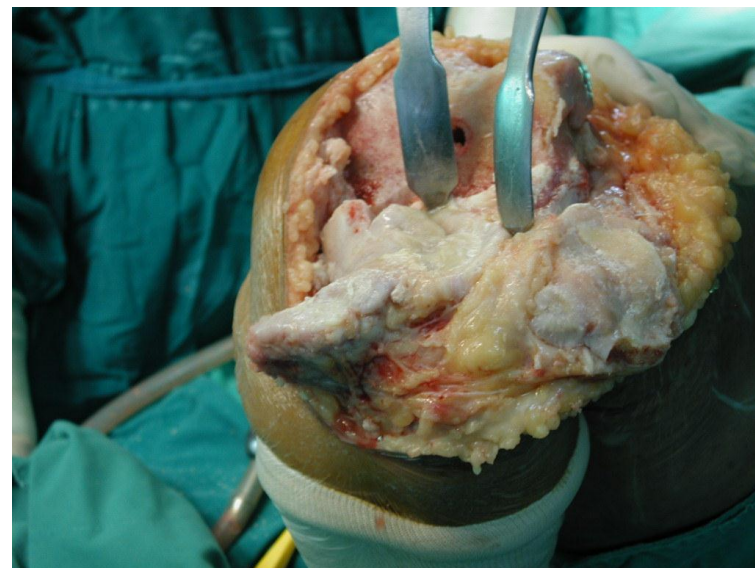
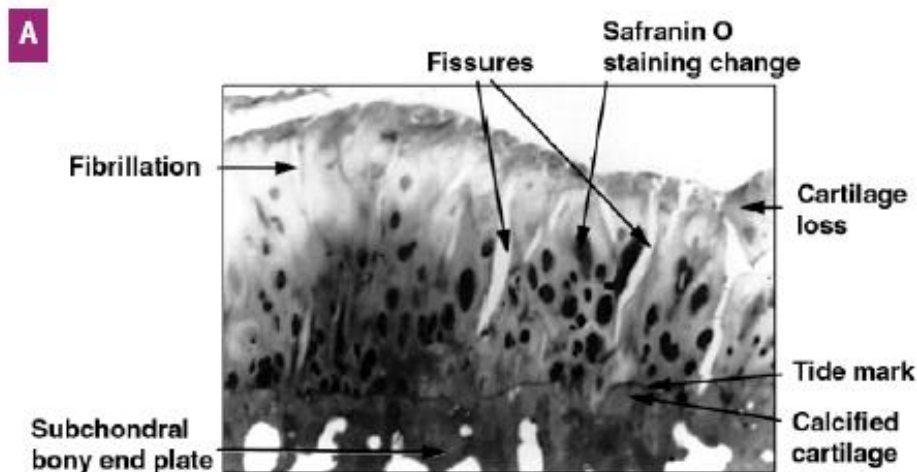
# Pathology of DJD





# Pathology of DJD

- Fissuring and focal erosive cartilage lesions
- Cartilage loss and destruction
- Subchondral bone sclerosis and cyst
- Large osteophyte formation

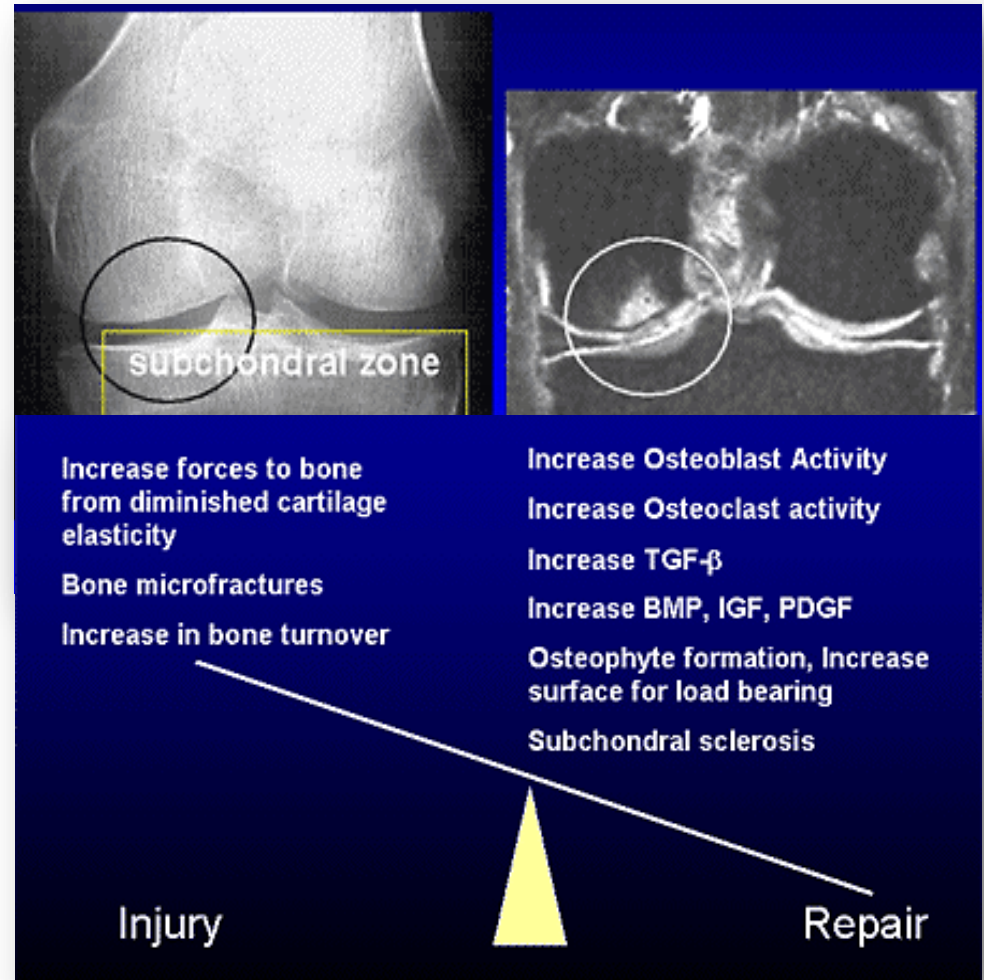






# Subchondral bone change

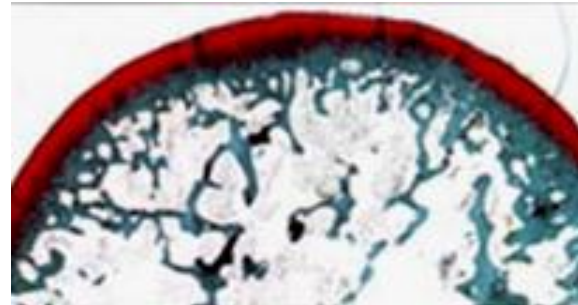
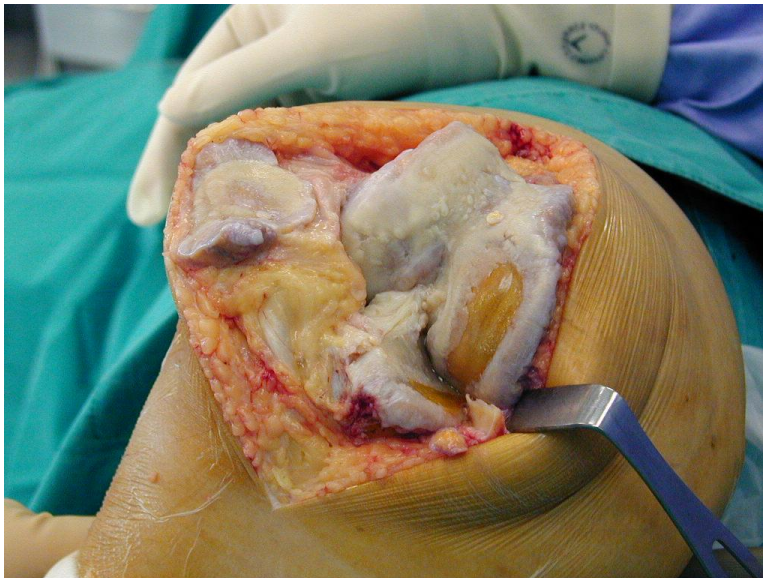
- Bone marrow edema
- Subchondral sclerosis
- Osteophyte





# What contributes poor articular cartilage repair?

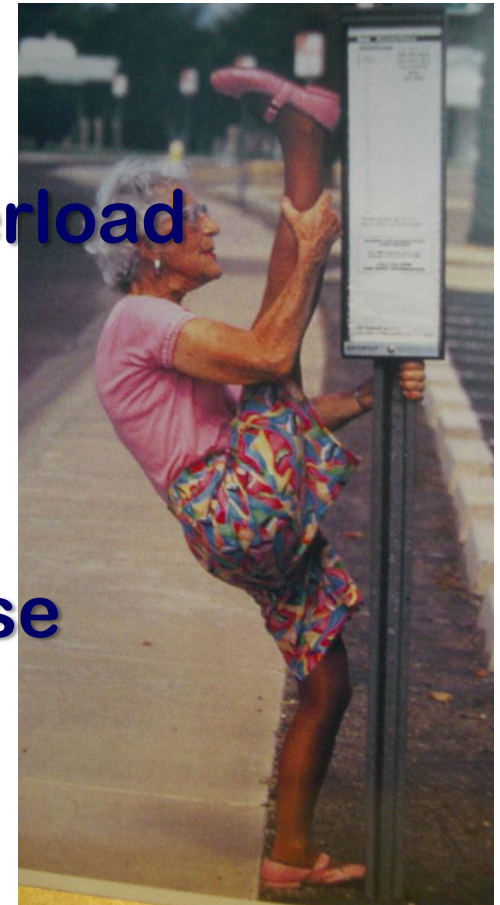
- High ratio of matrix/ cellular components
  - Poor ability of cartilage to repair
- Low mitotic activity of chondrocytes
  - Negligible healing of cartilage





# What are risk factors for OA

- Older age
- Female sex
- Repetitive stress and joint overload
- Genetic factors
- Major trauma
- Obesity
- Prior inflammatory joint disease
- Metabolic disorder
- Proprioceptive defects





# Etiologic factors in OA

- **Normal load**
  - Abnormal biomaterial (joint vulnerability)
- **Normal biomaterial**
  - Excessive mechanical stress (excessive joint loading)

## Joint vulnerability

- **Systemic factors**
  - Age
  - Gender
  - Genetic
- **Local factors**
  - Joint deformity
  - Malalignment
  - Previous injury of joint protective structures



# What are the symptoms of DJD?

- With or without symptoms for a long time
- Symptoms
  - Subtle development of morning stiffness
  - Pain with movement and activity
  - Improve with rest
  - Decreased range of motion
  - Abnormal sound
  - Unstable joints





# How is DJD diagnosed?

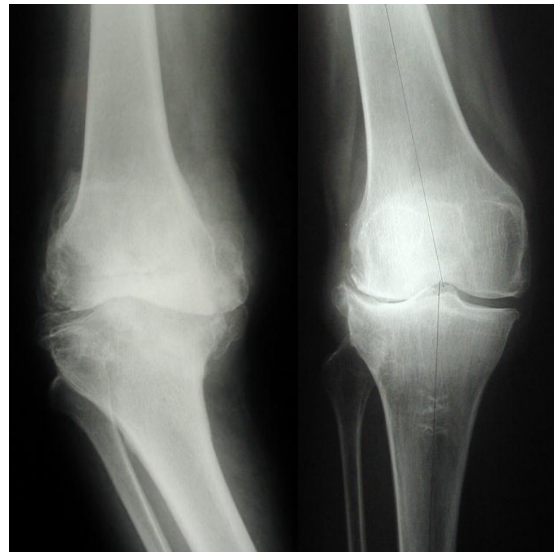
- **Symptoms and signs**
  - Joint pain
  - Restriction of motion
  - Crepitus with motion
  - Joint effusions
  - Deformity
  - Instability
- **Radiography**
  - Abnormal findings





# Radiographic findings

- **Subchondral bone changes**
  - Osteophyte
  - Cyst formation
  - Sclerosis
- **Narrowed joint spaces**
- **Deformity**





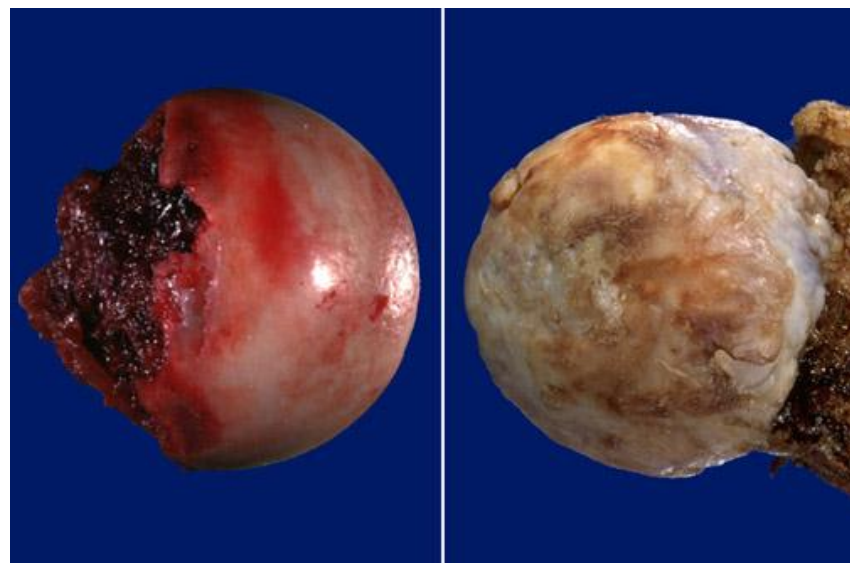
# Classification

- **Primary or idiopathic**
  - Most common type
  - No identifiable etiology or predisposing cause
- **Secondary**
  - Identifiable underlying cause



# Primary OA

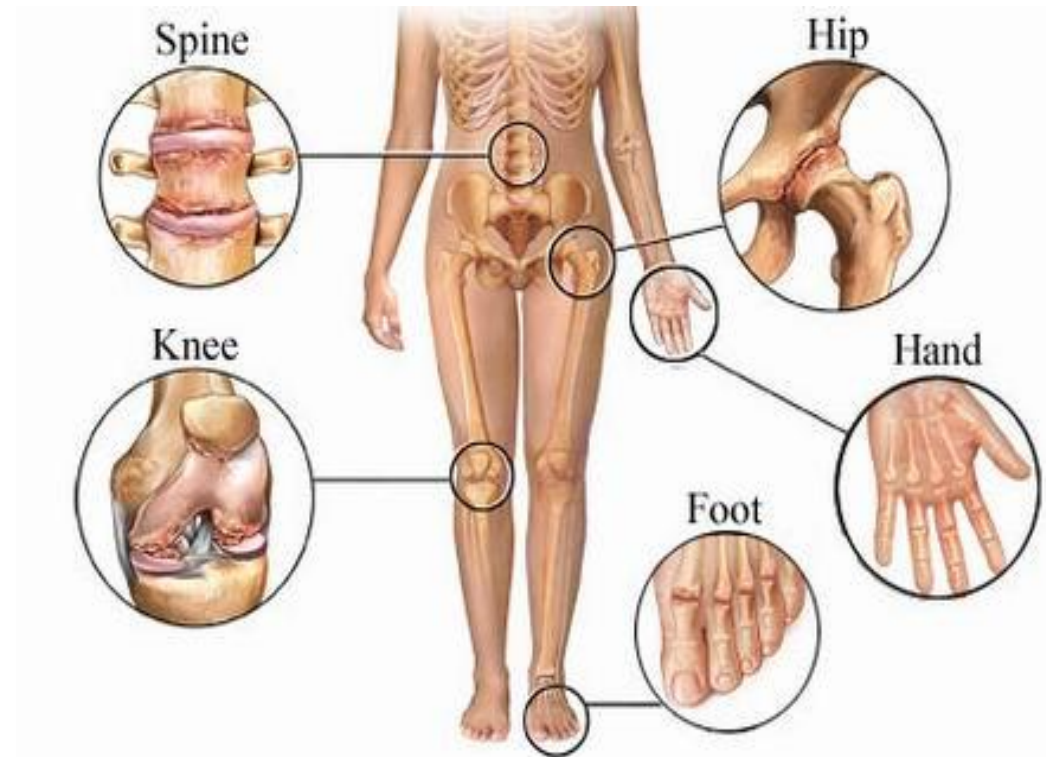
- Idiopathic
- Strong family history
- Alterations in structural macromolecules
- Mutations in the type II collagen gene
- Progressive wear and tear degenerative condition





# Primary OA

- **Common**
  - **Knee**
  - **Spine**
  - **Hip**
  - **IPJ of hand**
  - **IPJ of foot**







# Primary OA: knee

- **No relationship**
  - Premature arthrosis VS activity levels
- **Signs & symptoms**
  - Pain
  - Stiffness
  - Swelling
  - Limited ROM
  - Limping
  - Crepitation
  - Deformity





# Primary OA: knee





# Primary OA: knee







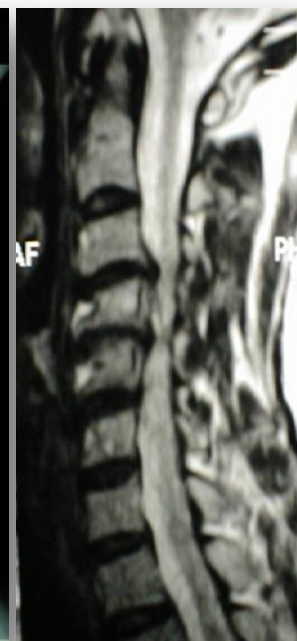
# Primary OA: knee





# Spine

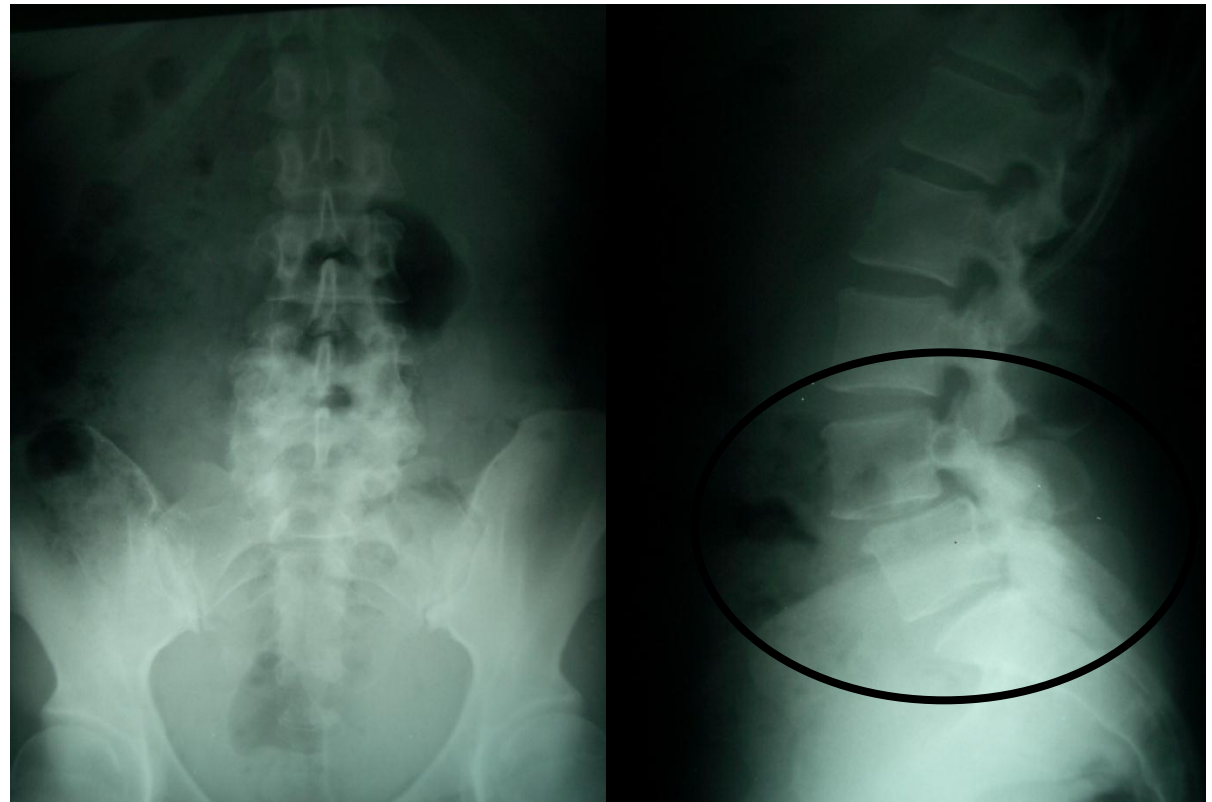
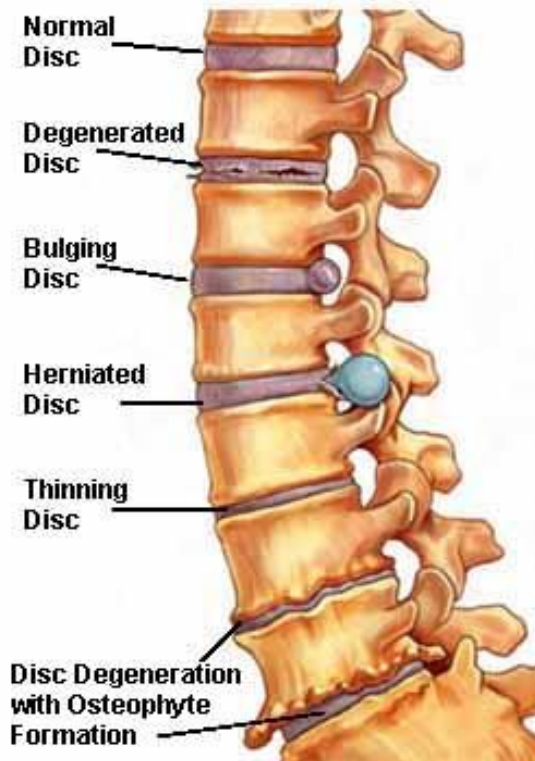
- **Spondylosis**
  - Pain
  - Stiffness
  - Limited ROM
  - Deformity
  - Radiculopathy
  - Myelopathy







# Spine





# Primary OA: hip

- Pain
- Stiffness
- Limited ROM
- Limping





# Primary OA: hip





# Primary OA: hand & finger

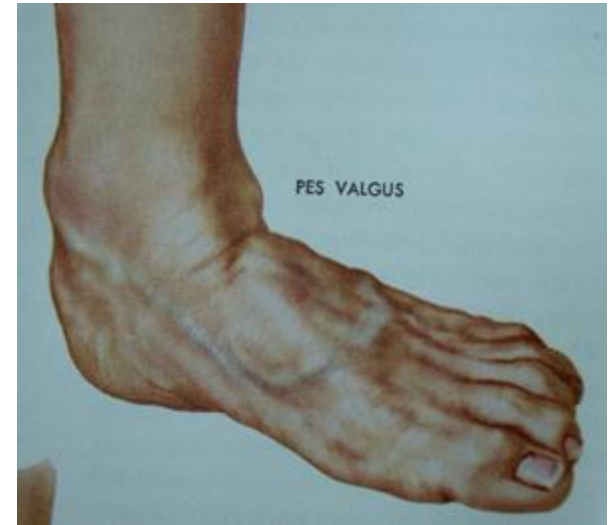
- Pain
- Swelling
- Limited ROM
- Heberden's nodes
- Bouchard's nodes







# Primary OA: other joints





# Secondary OA

- **Direct response**
  - Abnormal mechanical loading with articular cartilage
- **Various causes**
  - Metabolic condition
  - Anatomic factors
  - Traumatic events
  - Inflammatory disorders





# Secondary OA

Cause of 2 <sup>o</sup> OA	Presumed mechanism
Intra-articular Fx	Damage to cartilage / joint incongruity
High intensity impact joint loading	Damage to cartilage / subchondral bone
Ligament injury	Joint instability
Joint dysplasia	Abnormal joint shape / abnormal cartilage
Aseptic necrosis	Collapse of articular surface / joint incongruity
Joint infection	Destruction of cartilage



# Secondary OA

Cause of 2 <sup>o</sup> OA	Presumed mechanism
Crystal deposition	Accumulation of crystal in cartilage
Ochronosis	Deposition of homogentisic acid polymer in cartilage
Hemochromatosis	Unknown
Hemophilia	Multiple joint hemorrhage
Acromegaly	Overgrowth of cartilage produce joint incongruity
Paget's disease	Distortion or incongruity of joint



# Secondary OA

Cause of 2 <sup>o</sup> OA	Presumed mechanism
Ehlers-Danlos syndrome	Joint instability
Gaucher's disease	Bone necrosis or pathologic Fx leading to joint incongruity
Stickler's syndrome	Abnormal joint / cartilage development
Neuropathic arthropathy	Loss of proprioception and sensation result in increase impact loading and torsion ,instability, articular Fx



# Secondary OA



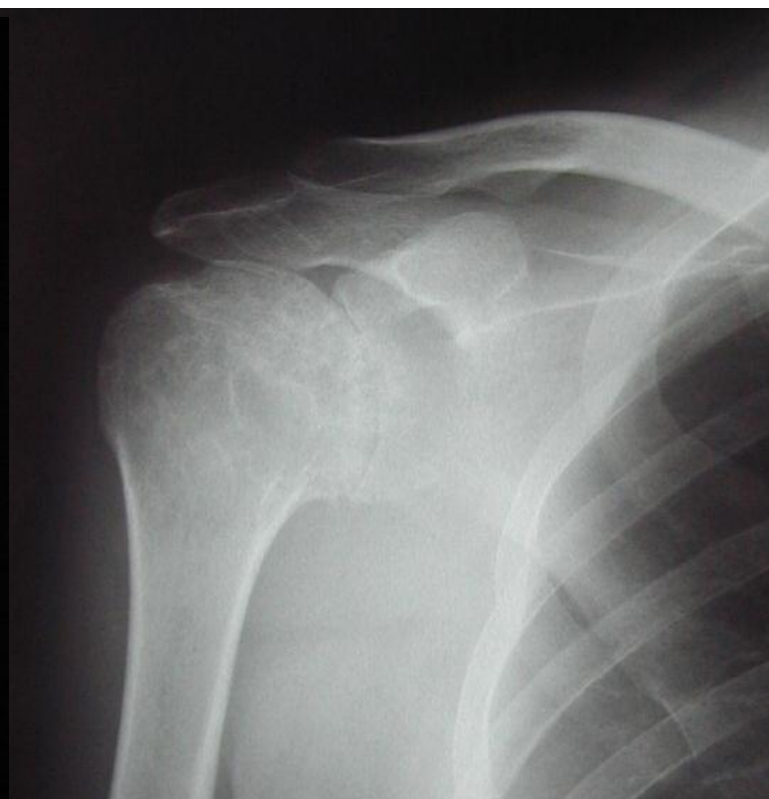


# Secondary OA





# Secondary OA







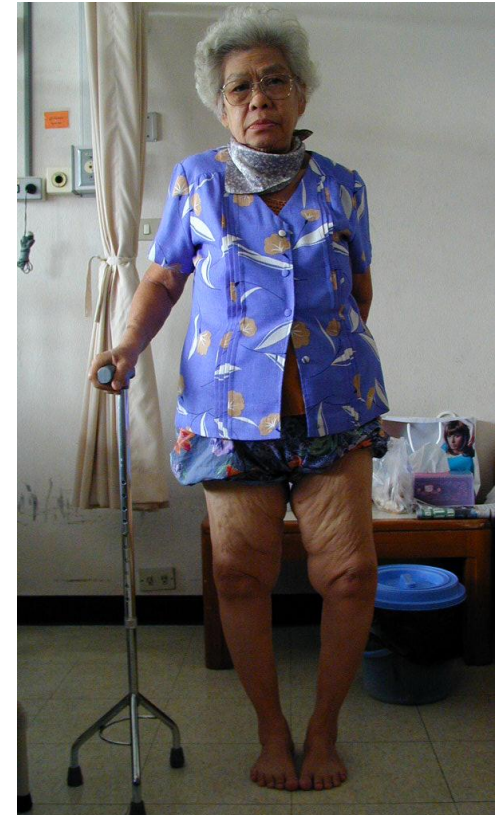
# How is DJD treated?

- **Goal of treatment**
  - **Decrease pain**
  - **Muscle strengthening**
  - **Improve or maintain joint function**
    - **Activity of daily living**
    - **Sports activity**



# Conservative treatment of OA

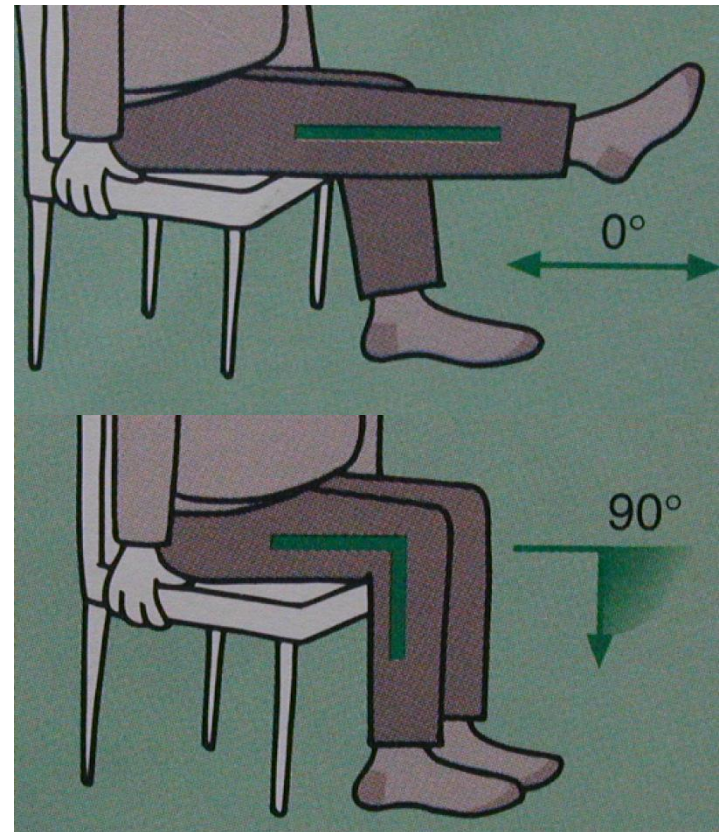
- Activity modification





# Conservative treatment of OA

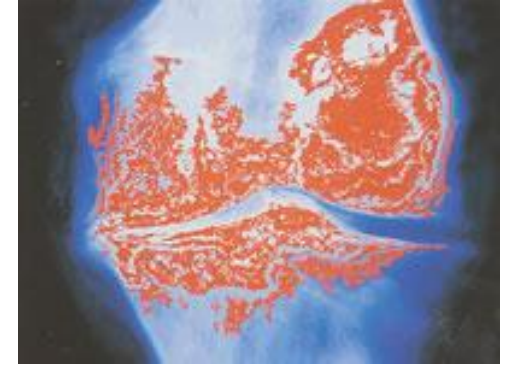
- Activity modification
- Analgesics
- Nonsteroidal anti-inflammatory drugs (NSAIDs)
- Appropriate physical therapy
- Weight loss
- Low impact exercise
- SySADOA





# Conservative treatment of OA

- **SySADOA**
- **Symptomatic Slow Acting Drug for Osteoarthritis**
  - Glucosamine
  - Chondroitin
  - Diacerein
  - Hyaluronic acid injection

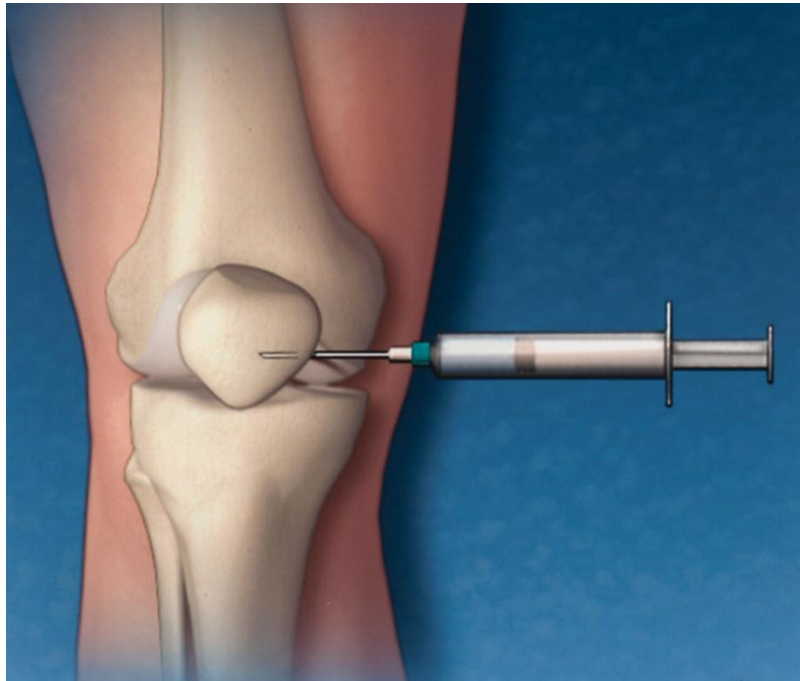






# Conservative treatment of OA

- Intra-articular administration of hyaluronic acid

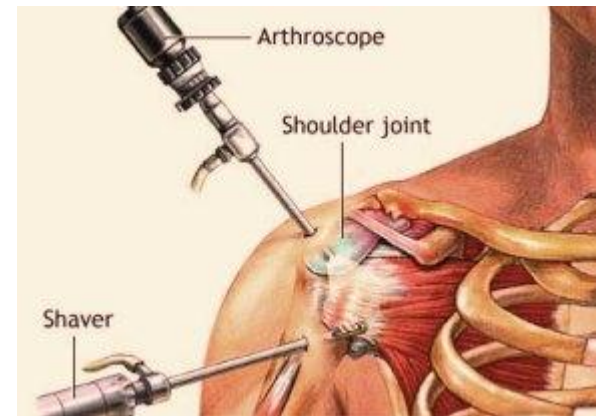
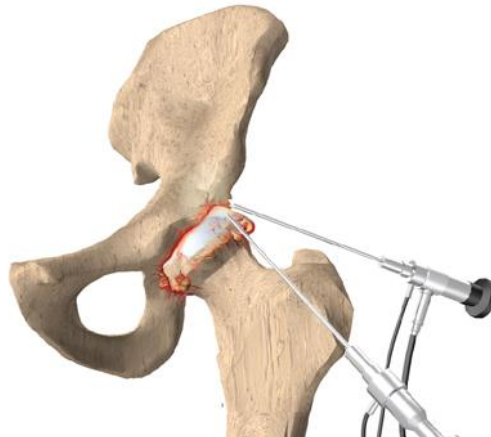






# Surgical treatment of OA

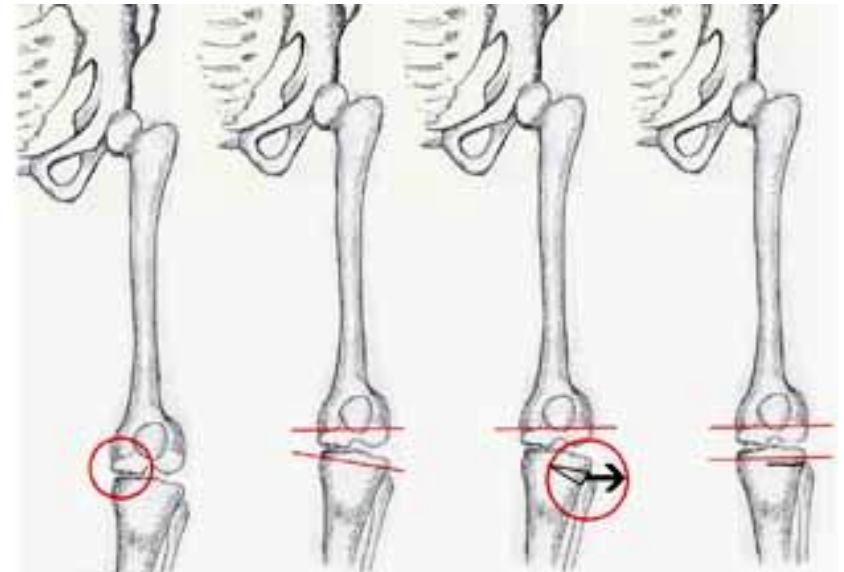
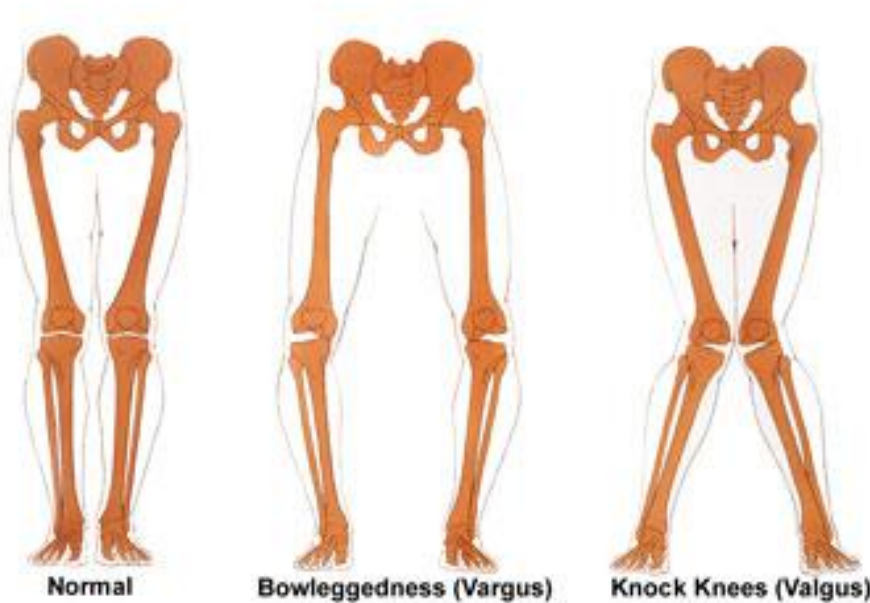
- **Arthroscopy**
  - Early symptom
  - Mild pathology –less deformity
  - Mechanical causes





# Surgical treatment of OA

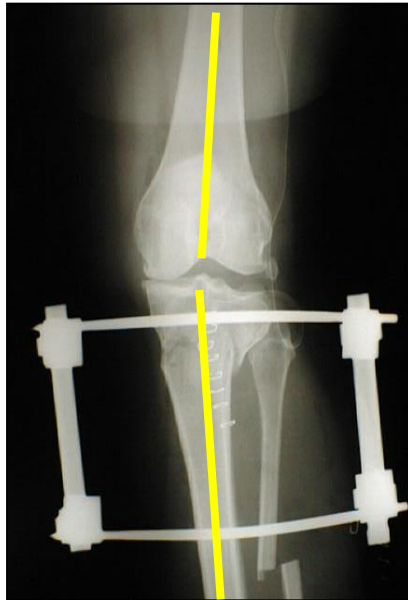
- **Realignment osteotomy**
  - Normalizes the biomechanical loads on the involved joint





# Surgical treatment of OA

- **Realignment osteotomy**
  - Normalizes the biomechanical loads on the involved joint





# Surgical treatment of OA







# Surgical treatment of OA







# Surgical treatment of OA

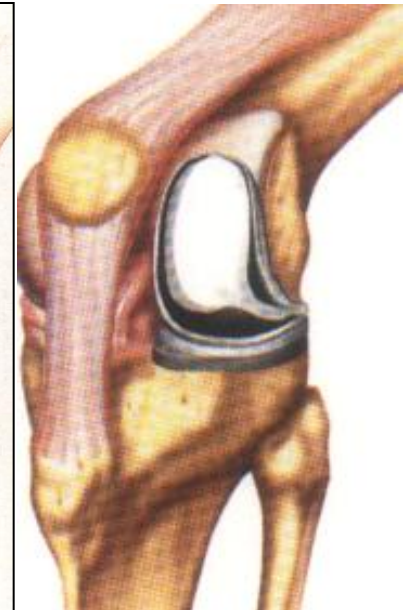
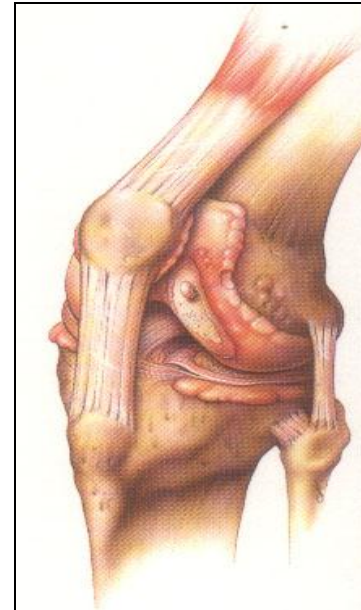
- **Autogenous chondrocyte transplantation**
  - Isolated, limited size (2- 7 cm<sup>2</sup>) chondral defects
- **Osteochondral autografts "mosaicplasty"**
  - Autografts of up to 10 mm is transferred into prepared defects





# Surgical treatment of OA

- **Knee arthroplasty**
  - Unicompartmental knee arthroplasty
  - Total knee arthroplasty





# Surgical treatment of OA

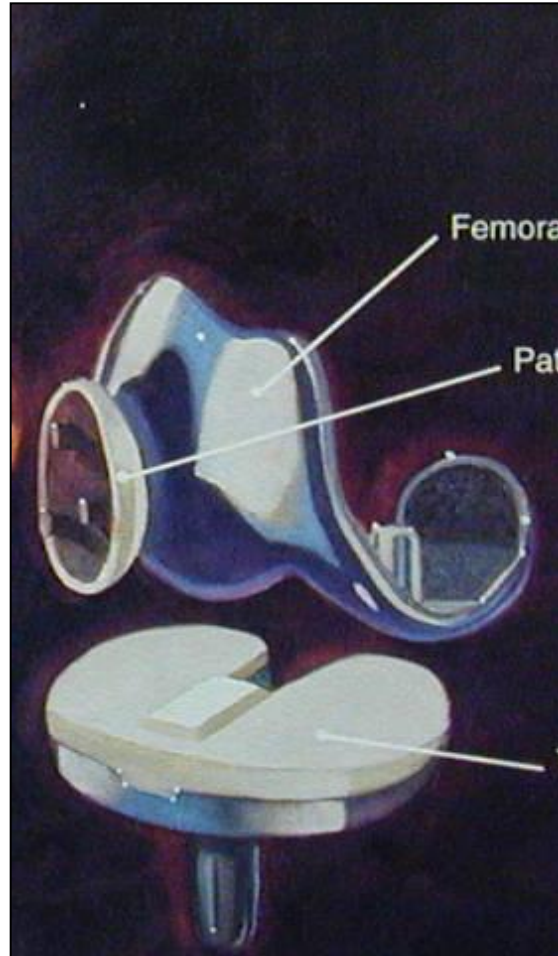
- **Conventional total knee replacement (arthroplasty)**







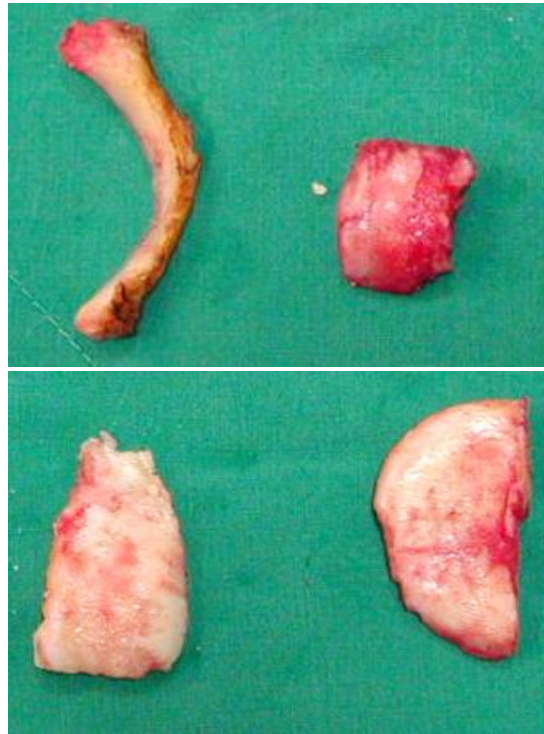
# Surgical treatment of OA





# Surgical treatment of OA

- Unicompartmental knee replacement (arthroplasty)







# Surgical treatment of OA



STD

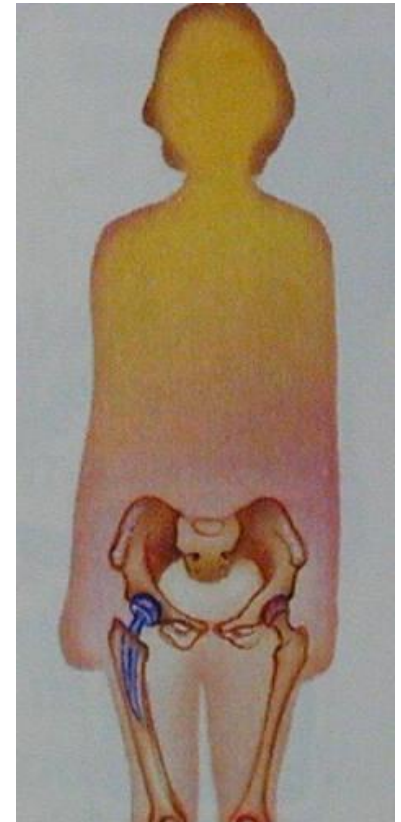
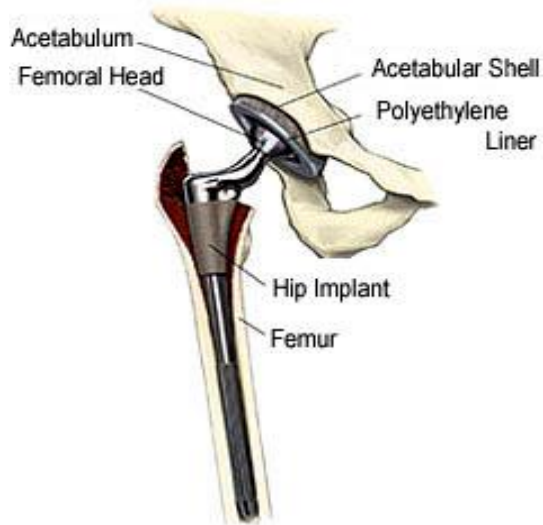
MIS: TKA

MIS: UKA



# Surgical treatment of OA

- **Total hip replacement (arthroplasty)**





# Surgical treatment of OA



**preoperative**



**postoperative**



# Surgical treatment of OA

- Conventional surgical technique

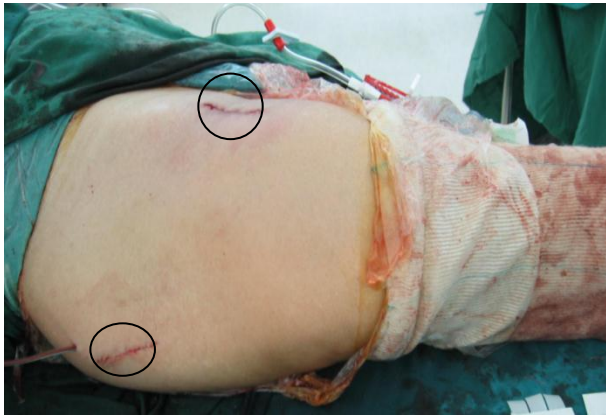






# Surgical treatment of OA

- Minimally invasive surgical technique







# Thank you



*Chulalongkorn University, Bangkok, Thailand*