Degenerative joint disease (DJD)

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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 25–74 years
  • 12.1% of the US population

• Radiographically defined OA
  – According to the Kellgren/Lawrence scale (presence of osteophytes)

## Symptomatic OA

<table>
<thead>
<tr>
<th>Anatomic site, age, years</th>
<th>Source (ref.)</th>
<th>% with symptomatic OA</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Male</td>
</tr>
<tr>
<td>Hands, ≥26</td>
<td>Framingham OA study (6)</td>
<td>3.8</td>
</tr>
<tr>
<td>Knees</td>
<td></td>
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<tr>
<td>≥26</td>
<td>Framingham OA study (5)</td>
<td>4.6</td>
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<tr>
<td>≥45</td>
<td>Framingham OA study (5)</td>
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<td>≥45</td>
<td>Johnston County OA Project (7)</td>
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<tr>
<td>≥60</td>
<td>NHANES III (4)</td>
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Radiographic OA

<table>
<thead>
<tr>
<th>Anatomic site, age, years</th>
<th>Source (ref.)</th>
<th>% with mild, moderate, or severe OA</th>
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<td>Male</td>
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<tr>
<td>Hands, ≥26</td>
<td>Framingham OA study (6)</td>
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<tr>
<td>Knees†</td>
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<tr>
<td>≥26</td>
<td>Framingham OA study (5)</td>
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</table>

Comparing OA diagnosis

<table>
<thead>
<tr>
<th>Anatomic site, age, years</th>
<th>Source (ref.)</th>
<th>Total</th>
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<tr>
<td>Hands, ≥26</td>
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<td>Johnston County OA Project (7)</td>
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<td>Johnston County OA Project (10)</td>
<td>9.2</td>
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</table>
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 ≥ 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
Pathology of DJD
Pathology of DJD

IL-1, TNF-α, IL-18

IL-6, IL-8

Cartilage degradation
MMPs, Aggrecanase, Nitric oxide

IL-4, IL-10, IL-13, IL-1ra, PGE₂

IGF-1, TGF-β, BMPs

Cartilage matrix synthesis
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

<table>
<thead>
<tr>
<th>Cause of 2º OA</th>
<th>Presumed mechanism</th>
</tr>
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<tbody>
<tr>
<td>Intra-articular Fx</td>
<td>Damage to cartilage / joint incongruity</td>
</tr>
<tr>
<td>High intensity impact joint loading</td>
<td>Damage to cartilage / subchondral bone</td>
</tr>
<tr>
<td>Ligament injury</td>
<td>Joint instability</td>
</tr>
<tr>
<td>Joint dysplasia</td>
<td>Abnormal joint shape / abnormal cartilage</td>
</tr>
<tr>
<td>Aseptic necrosis</td>
<td>Collapse of articular surface / joint incongruity</td>
</tr>
<tr>
<td>Joint infection</td>
<td>Destruction of cartilage</td>
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<td>Crystal deposition</td>
<td>Accumulation of crystal in cartilage</td>
</tr>
<tr>
<td>Ochronosis</td>
<td>Deposition of homogentisic acid polymer in cartilage</td>
</tr>
<tr>
<td>Hemochromatosis</td>
<td>Unknown</td>
</tr>
<tr>
<td>Hemophilia</td>
<td>Multiple joint hemorrhage</td>
</tr>
<tr>
<td>Acromegaly</td>
<td>Overgrowth of cartilage produce joint incongruity</td>
</tr>
<tr>
<td>Paget’s disease</td>
<td>Distortion or incongruity of joint</td>
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<td>Ehlers-Danlos syndrome</td>
<td>Joint instability</td>
</tr>
<tr>
<td>Gaucher’s disease</td>
<td>Bone necrosis or pathologic Fx leading to joint incongruity</td>
</tr>
<tr>
<td>Stickler’s syndrome</td>
<td>Abnormal joint / cartilage development</td>
</tr>
<tr>
<td>Neuropathic arthropathy</td>
<td>Loss of proprioception and sensation result in increase impact loading and torsion</td>
</tr>
<tr>
<td></td>
<td>instability, articular Fx</td>
</tr>
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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

- **Realignement osteotomy**
  - Normalizes the biomechanical loads on the involved joint
Surgical treatment of OA
Surgical treatment of OA

- **Autogenous chondrocyte transplantation**
  - Isolated, limited size (2-7 cm$^2$) 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