

## **Osteoarthritis Epidemiology, Risk Factors, and Pathophysiology**

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### THESE AUTHORS NOTE:

“Osteoarthritis (OA) is the most prevalent form of arthritis and a major cause of disability in people aged 65 and older.” OA affects the majority of adults over age 55.

58% of those older than 70 years have symptomatic OA.

10-30% of those with OA have significant pain and disability.

OA is “the clinical and pathologic outcome of a range of disorders that results in structural and functional failure of synovial joints. OA occurs when the dynamic equilibrium between the breakdown and repair of joint tissues is overwhelmed.”

The risk of OA has 2 major categories: systemic factors and local factors:

#### 1) Systemic Factors:

- A)) Ethnicity
- B)) Age: “The presence of radiographic OA rises with age at all joint sites.”
- C)) Gender
- D)) Hormonal Status
- E)) Genetic Factors  
OA has a major genetic component
- F)) Bone Density
- G)) Nutritional Factors  
There is evidence that OA is linked to free radicals, and that high dietary antioxidants (especially vitamins C and D) are protective against the development of OA. “Chondrocyte senescence is thought to be the result of chronic oxidative stress.”

#### 2) Local Factors:

- Local factors “result in abnormal biomechanical loading of affected joints.”
- A)) Obesity
- B)) “Altered joint biomechanics”
  - ligamentous laxity
  - malalignment

- impaired proprioception  
With aging, there is a decline in proprioception, causing decreased neurologic responses, impairing proprioceptive joint-protective mechanisms. Consequently, reduced proprioception advances OA.
  - muscle weakness
- C)) Prior joint injuries  
D)) Occupational Factors  
E)) Effects of sports and physical activities  
F)) Developmental abnormalities

“If systemic factors are in place, the joint may be thought of as vulnerable, and thus local biomechanical factors will have more of an impact on joint degeneration.”

“Injuries that alter mechanical or joint alignment may also predispose individuals to OA at other sites.”

“Other risk factors for posttraumatic arthritis include high body mass, high level of activity, and residual joint instability or malalignment.”

Obesity increases the risk of OA. Importantly, the increased risk includes joints that are not weight bearing, like hand OA. This suggests that “obesity may predispose to OA, perhaps via an inflammatory or metabolic intermediary.” [I suggest prostaglandin E2 (PGE2)]. “This means that obesity plays a role not only as a local process but systemically as well.”

Repetitive occupational stresses increase OA.

In the absence of systemic factors, moderate exercise, such as running, does not cause joint degeneration. However, there is increased OA in male runners who exceed more than 20 miles per week.

High-intensity direct joint impact or torsional loading can increase the risk of OA in the affected joint.

Loss of normal joint biomechanics result in increased joint vulnerability to OA.

Joint malalignment, or proprioceptive deficits predispose the joint to the development of OA.

“Impaired proprioception has been seen in patients with OA compared with age-matched controls, which may also indicate that proprioceptive loss preceded disease development.”

“Joint immobilization has been shown to be detrimental, reducing cartilage thickness and proteoglycan content.”

Intense exercise, especially in the elderly, can accelerate cartilage breakdown and OA.

Muscle weakness predisposes individuals to the development of OA because greater stress loads are borne by the joints, accelerating joint damage.

“Adequate muscle strength and bulk are protective to the joint.”

“Cartilage is avascular, and therefore chondrocytes receive nutrients and eliminate waste by diffusion through the synovial fluid and by facilitated imbibition.”

Osteoarthritis of a joint typically involves all of these tissues of the synovial joint, including:

- 1) Articular cartilage
- 2) Subchondral bone
- 3) Synovial tissue
- 4) Ligaments
- 5) Joint capsules
- 6) Muscles that cross the joint

A decreased range of joint motion leads to muscle atrophy and loss of joint protection, increasing the risk of OA.

Although OA is considered to be a non-inflammatory arthritis, as cartilage destruction proceeds, mild to moderate inflammatory reactions are found in the synovial membranes.

As the OA catabolic process progresses, the synoviocytes begin to make and release the pro-inflammatory eicosanoid hormone prostaglandin E2 (PGE2).

**[Recall that PGE2 is derived from the omega-6 fatty acid arachidonic acid]**

#### KEY POINTS FROM DAN MURPHY

- 1) “Osteoarthritis (OA) is the most prevalent form of arthritis and a major cause of disability in people aged 65 and older.” OA affects the majority of adults over age 55.
- 2) OA is “the clinical and pathologic outcome of a range of disorders that results in structural and functional failure of synovial joints. OA occurs when the dynamic equilibrium between the breakdown and repair of joint tissues is overwhelmed.”
- 3) Both systemic factors and local factors will increase the risk of osteoarthritis.
- 4) Systemic Factors:
  - A)) Ethnicity
  - B)) Age: “The presence of radiographic OA rises with age at all joint sites.”
  - C)) Gender

- D)) Hormonal Status
- E)) Genetic Factors  
OA has a major genetic component
- F)) Bone Density
- G)) Nutritional Factors  
There is evidence that OA is linked to free radicals, and that high dietary antioxidants (especially vitamins C and D) are protective against the development of OA. "Chondrocyte senescence is thought to be the result of chronic oxidative stress."
- 5) Local Factors:  
Local factors "result in abnormal biomechanical loading of affected joints."
- A)) Obesity
- B)) "Altered joint biomechanics"  
  - ligamentous laxity
  - malalignment
  - impaired proprioception  
With aging, there is a decline in proprioception, causing decreased neurologic responses, impairing proprioceptive joint-protective mechanisms. Consequently, reduced proprioception advances OA.
  - muscle weakness
- C)) Prior joint injuries
- D)) Occupational Factors
- E)) Effects of sports and physical activities
- F)) Developmental abnormalities
- 5) "If systemic factors are in place, the joint may be thought of as vulnerable, and thus local biomechanical factors will have more of an impact on joint degeneration."
- 6) "Injuries that alter mechanical or joint alignment may also predispose individuals to OA at other sites."  
**[Altered alignment or mechanics predispose joints to osteoarthritis]**
- 7) "Other risk factors for posttraumatic arthritis include high body mass, high level of activity, and residual joint instability or malalignment."  
**[Important: joint instability and malalignment increase risk of OA]**
- 8) Obesity increases the risk of OA in both weight-bearing and non weight-bearing joints. This suggests that "obesity may predispose to OA, perhaps via an inflammatory or metabolic intermediary." **[I suggest prostaglandin E2 (PGE2)].** "This means that obesity plays a role not only as a local process but systemically as well."
- 9) Repetitive occupational stresses increase osteoarthritis.

- 10) High-intensity direct joint impact or torsional loading can increase the risk of OA in the affected joint.
- 11) Loss of normal joint biomechanics result in increased joint vulnerability to osteoarthritis. **[Important]**
- 12) Proprioceptive deficits predispose the joint to the development of osteoarthritis.  
**[This is important because the subluxation is not only a mechanical alignment lesion, but also has associated aberrant proprioception]**
- 13) "Impaired proprioception has been seen in patients with osteoarthritis compared with age-matched controls, which may also indicate that proprioceptive loss preceded disease development."
- 14) Muscle weakness predisposes individuals to the development of OA because greater stress loads are borne by the joints, accelerating joint damage. "Adequate muscle strength and bulk are protective to the joint."
- 15) "Cartilage is avascular, and therefore chondrocytes receive nutrients and eliminate waste by diffusion through the synovial fluid and by facilitated imbibition."  
**[Important: reduced motion impairs joint nutrition, accelerating OA]**
- 16) Both immobilization and decreased range of joint motion leads to muscle atrophy and loss of joint protection, increasing the risk of osteoarthritis.
- 17) Although OA is considered to be a non-inflammatory arthritis, as cartilage destruction proceeds, mild to moderate inflammatory reactions are found in the synovial membranes.
- 18) As the OA catabolic process progresses, the synoviocytes begin to make and release the pro-inflammatory eicosanoid hormone prostaglandin E2 (PGE2).  
**[Recall that PGE2 is derived from the omega-6 fatty acid arachidonic acid]**

#### COMMENTS FROM DAN MURPHY

For decades, at least since the Renaissance seminars in the 1970s (Flesia and Riekeman), chiropractors have maintained that the spinal subluxation accelerated spinal joint degeneration and osteoarthritis. Components of the subluxation include altered alignment, altered movement, muscle atrophy, reduced range of joint motion and aberrant proprioception. These components of the subluxation are the same factors that this article associates with an increased risk of osteoarthritis. This supports the teachings of the Renaissance seminars of the 1970s, and the phases of subluxation degeneration. It supports the contention that uncorrected subluxations predispose those joints to osteoarthritis.