

**When
Moving
Hurts**



**Assess
Understand
Take Action**

GLOBAL YEAR AGAINST MUSCULOSKELETAL PAIN

OCTOBER 2009 – OCTOBER 2010

Osteoarthritis-Related Pain

Epidemiology

Osteoarthritis (OA) is the most common joint disorder and the leading cause of disability in older adults. Symptomatic knee OA occurs in approximately 37% of persons aged 60 years or older. From 1995 to 2005, the number affected with clinical OA grew from 21 million to nearly 27 million in the United States, reflecting the aging of the U.S. population. Pain from knee OA is a key symptom in the decision to seek medical care and an important antecedent to disability. Currently, no therapeutic strategies have been proven to alter structural progression. The rapid increase in the prevalence of this already common disease suggests that OA will have a growing impact on health care and public health systems in the future.

Pathophysiology

- The joint's intra-articular and periarticular structures, including menisci, adipose tissue, synovium, and periosteum, are innervated with nociceptors, while cartilage is aneural.
- The pathological structural changes of OA and inflammation with the related increase in cytokines lead to peripheral sensitization, manifesting as primary hyperalgesia, spontaneous pain, and pain with normally innocuous movement [5].
- The specific pathological features related to pain in OA are likely to be related to bone marrow lesions, synovitis, effusions, and possibly meniscal abnormalities.
- Changes in the central nervous system contribute to enhanced sensitivity to mechanical stimuli that develops outside the area of injury (secondary hyperalgesia), with increased responsiveness to peripheral input or central sensitization, manifested by clinical features such as referred or radiating pain and reduced pain thresholds in unaffected joints.
- Genetic contributions to pain sensitivity may play a role in OA. Recently, the *COMT* polymorphism val158met was demonstrated to be associated with hip OA-related pain.
- Psychological factors may also be an important component of the experience of OA pain, with small studies demonstrating increased affective and motivational response in the pain experience in those with OA.

Clinical Features and Diagnostic Criteria

- OA is generally localized to the hip, knee, or hand, and less commonly to other joints such as the shoulder, elbow, wrist, or ankle
- For a diagnosis of OA, patients must have pain and 5 of the following criteria:
 - age >50 years
 - stiffness <30 minutes
 - crepitus
 - bony tenderness
 - bony enlargement
 - no palpable warmth
 - erythrocyte sedimentation rate <40 mm/h
 - rheumatoid factor <1:40
 - synovial fluid signs of osteoarthritis
 - osteophytes

Treatment

- Systematic reviews show that opioid agonists (e.g., tramadol), acetaminophen (paracetamol), nonsteroidal anti-inflammatory drugs (NSAIDs), and interleukin-1 inhibitors reduce pain and in some cases improve function in people with osteoarthritis.
- Local treatments generally include intra-articular injection of corticosteroids or hyaluronic acid, and both improve pain and function in OA.
- Total joint replacement is considered when pain and functional limitations result in a diminished quality of life, when there is radiographic evidence of joint damage, and when there is moderate to severe pain that is not adequately relieved by nonsurgical approaches. Total joint replacement, generally of the hip or knee, is the primary surgical approach; it clearly reduces pain and improves function and quality of life in people with osteoarthritis.
- Physical therapy is effective in treatment of OA. Both strengthening and aerobic conditioning exercises reduce pain and improve function. Transcutaneous electrical nerve stimulation (TENS) reduces pain, cryotherapy improves function, and low level laser therapy reduces pain and improves function.
- Psychological management using cognitive-behavioral therapy reduces pain, with effects that are maintained through a 6-month follow-up.

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