



- **FACT SHEET No. 6**

Exercise, Sports, and Joint Pain

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Osteoarthritis (OA) is a complex disease of the joint leading to a severe disability in elderly people. This disability results in pain during motion and loss of joint function. In some OA subphenotypes of the disease, the mechanical pain can be associated with inflammatory and/or neuropathic pain [1]. It is now well accepted that OA is more than a joint disease. OA is associated with obesity, metabolic syndrome, and cardiovascular diseases [2]. The links between these conditions are systemic mediators, i.e., adipokines, myokines, cytokines, which are released in the blood stream by joint tissues as well as muscles and fat [3].

OA Is More Than a Cartilage Disease

Osteoarthritis pain has been classically attributed to structural damage in a joint or joints. In OA, cartilage is progressively degraded and undergoes structural modifications like fibrillation, fissuration, and erosion. This cartilage degradation leads to the release in the joint cavity of products of degradation—osteochondral fragments and microcrystals—which trigger synovial membrane inflammation [4]. The inflamed synovium is directly associated with chondrolysis and inflammatory pain.

Further, cartilage disappearance is associated with subchondral bone changes, i.e., bone sclerosis and microfractures, and both contribute to mechanical pain. However, disparity between the severity of structural damages and the severity of symptoms implies that factors other than the joint pathology itself contribute to the pain. Peripheral and central sensitization have been suggested as two of the underlying mechanisms that contribute to OA pain. The peripheral nociceptors may be sensitized by, for example, inflamed synovium and damaged subchondral bone. Continuous and intense nociceptive input



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from the OA knee joint may drive central sensitization and subsequent modification of central pain-transmitting neurons and may be clinically associated with neuropathic pain [1].

State-of-the-Art Management of OA Is Crucial

The management of OA represents a challenge for the scientific community. Recently, different OA phenotypes have been described, including obesity-related OA, mechanical-induced OA, and aging-related OA. This suggests that OA treatment could be stratified and tailored to the relevant phenotype. A key challenge will be to identify phenotypes for particular treatments.

Until now, the management of OA has consisted mostly of symptom management, i.e., reduction of pain and improvement of joint function, which relies on the combination of non-pharmacologic and pharmacologic approaches. Although important, the control of symptoms is not the only goal that needs to be achieved in OA patients. Indeed, the ideal treatment for OA should preserve the joint structures, keeping in mind safety and an improved quality of life for patients.

Recently, the Osteoarthritis Research Society International (OARSI) published guidelines for nonsurgical management of knee OA [5]. The originality of these guidelines is that they provide separate recommendations for each of four clinical subphenotypes: knee-only OA without comorbidities, knee-only OA with comorbidities, multi-joint OA without morbidities, and multi-joint OA with comorbidities. The core treatments appropriate for all individuals include land-based exercises, weight management, strength training, water-based exercises, self-management, and education.

References

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