



## Osteoarthritis<sup>☆</sup>

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### Abstract

Osteoarthritis (OA), the syndrome of joint pain and dysfunction caused by joint degeneration, affects more people than any other joint disease. In most instances joint degeneration develops in the absence of an identifiable cause, but increasing age, excessive joint loading, and joint abnormalities and insults increase the risk of OA. Articular surface contact stress that causes tissue damage and compromises that ability of chondrocytes to maintain and restore the tissue has an important role in the development of joint degeneration. Current methods of attempting to restore an articular surface in osteoarthritic joints include penetrating subchondral bone, altering joint loading, osteotomies and insertion of soft tissue grafts. Dramatic advances in the prevention and treatment of OA are likely to stem from better understanding of the role of mechanical forces in the initiation and progression of joint degeneration.

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### Contents

1. Introduction . . . . .	151
2. Diagnosis . . . . .	151
3. Classification . . . . .	152
4. Prevalence, incidence, and costs. . . . .	152
4.1. Prevalence . . . . .	153
4.2. Incidence . . . . .	153
4.3. Costs . . . . .	154
5. Distribution among joints . . . . .	154

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6.	Risk factors . . . . .	154
6.1.	Age . . . . .	155
6.2.	Joint injury . . . . .	155
6.3.	Age and joint injury . . . . .	155
6.4.	Excessive repetitive joint loading . . . . .	155
6.5.	Joint dysplasia . . . . .	156
7.	Natural history . . . . .	156
8.	Restoring degenerated articular surfaces . . . . .	157
8.1.	Penetration of subchondral bone . . . . .	157
8.2.	Decreased articular surface contact stress . . . . .	159
8.3.	Osteotomy . . . . .	159
8.4.	Soft tissue grafts . . . . .	160
9.	Conclusions . . . . .	161
	Acknowledgement . . . . .	162
	References . . . . .	162

## 1. Introduction

Osteoarthritis (OA), the most common joint disease, is among the most frequent and symptomatic health problems for middle aged and older people [1–7]. It is characterized by joint pain and dysfunction; and, in its advanced stages, joint contractures, muscle atrophy and limb deformity [8–10]. OA is caused by joint degeneration, a process that includes progressive loss of articular cartilage accompanied by attempted repair of articular cartilage, remodeling and sclerosis of subchondral bone, and osteophyte formation [9,10]. Although increasing age and excessive articular surface contact stress increase the risk of degeneration in all joints, the pathophysiology of the joint degeneration that leads to the clinical syndrome of OA remains poorly understood. For this reason, efforts to prevent this disease or slow its progression are based more on speculation than science. Current treatments do not prevent or cure OA, and symptomatic treatments often fail to provide satisfactory pain relief. Once patients develop OA, they suffer from the disease for the remainder of their lives and the severity of pain and disability generally increase. The frequency and chronicity of OA and the lack of effective preventive measures or cures make this disease a substantial economic burden for patients, health care systems, businesses, and nations [2,3,5,6,11–14].

Improving the prevention and treatment of OA will require finding methods of preventing and slowing the

joint degeneration that causes OA and decreasing the joint pain and dysfunction. This manuscript was developed in part from the authors' previously published works [8,9,15–19]. It examines the clinical classification, incidence, prevalence, costs, distribution among joints, risk factors, and natural history of OA. The final section discusses approaches to restoring degenerated joint surfaces.

## 2. Diagnosis

Osteoarthritis results from degeneration of a synovial joint: a generally progressive loss of articular cartilage accompanied by attempted repair of articular cartilage, remodeling and sclerosis of subchondral bone, and in many instances the formation of subchondral bone cysts and marginal osteophytes [8,9]. In addition to the changes in the synovial joint, usually demonstrated by plain radiographs, diagnosis of the clinical syndrome of osteoarthritis requires the presence of chronic joint pain. Many patients with osteoarthritis also have restriction of motion, crepitus with motion and joint effusions. The most severely affected individuals develop joint deformities and subluxations.

Most people with osteoarthritis seek medical attention because of joint pain. They often describe the pain as a deep aching poorly localized discomfort that has been present for years. The pain may increase with changes in the weather, especially storms or a

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