

infoaging guides

DISEASES OF AGING



# OSTEOARTHRITIS

An introduction to aging science brought to you by the  
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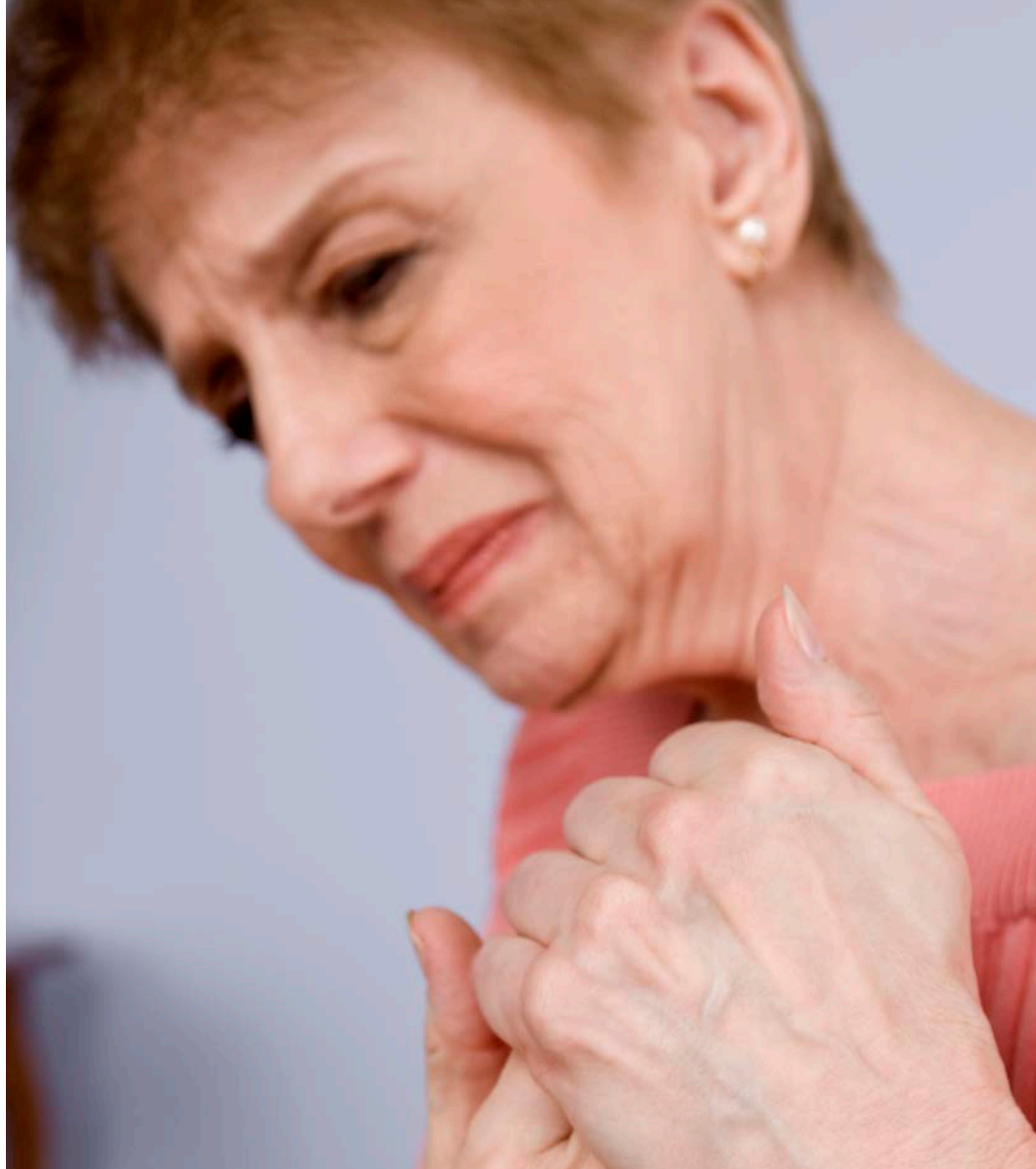
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## WHAT IS OSTEOARTHRITIS?

Osteoarthritis (OA) is the most common joint disease and involves damage to and loss of cartilage as well as overgrowth of bone within the joints. This overgrowth often takes the form of bony spurs. Some people with OA can also have *synovitis*, an inflammation in the joint lining. In the knee joint, there can be damage to the meniscus and joint ligaments. Damage to the cartilage, meniscus, ligament, and bone, as well as the synovitis can cause pain, deformities, and even disability. Indeed, osteoarthritis is the leading cause of disability among older adults.

Most of us will develop the early cellular changes of OA in our knees by age 60. Fortunately, not all of us will have changes severe enough to cause symptoms. As we age, the total number of our cartilage cells decreases. Further, these cells' ability to repair joint damage declines. If a proper balance between joint damage and joint repair is not maintained, the cartilage becomes worn or ragged. The cartilage cells, called chondrocytes, release cytokines, chemical messengers that activate neighboring chondrocytes as well as bony cells, or osteoblasts, and the joint lining cells. The bone cells respond by creating small bits of new bone that can irritate the joint. Likewise, the bone cells and joint lining cells release cytokines and growth factors, which can act on the chondrocytes and cause further destruction. And if you use a joint lined with ragged cartilage and bony spurs, you feel pain. Eventually, you may also experience chronic swelling that limits the joint's movement.

OA is distinct from rheumatoid arthritis, which involves



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disturbances in an individual's immune system. These disturbances can cause the person's body to have an inappropriate immune response to its own tissue.

### DIAGNOSING OSTEOARTHRITIS

Patient history and a physical examination provide most of the information needed to make a diagnosis of OA. Joint pain generally comes on gradually and may involve only a single joint. OA can

produce morning stiffness, which usually lasts only 15 to 30 minutes and typically improves with movement. Over time, the mobility of the affected joints decreases, and they can become tender to the touch. Cracking and crackling can be heard. Overgrowth of bone as well as the surrounding tissues can produce permanent swelling and joint enlargement.

There are no blood tests to diagnose OA, distinguishing it from rheumatoid arthritis and gout, for

which confirmatory blood tests are available.

Some imaging tests can confirm the suspicion of OA. They include:

- **X-Rays.** X-rays of the bones and joints in a patient suspected of having OA will show narrowing of the spaces that should be seen in joints, thickening of the bone that lines the joints and the formation of bone spurs. X-rays can also show bone changes indicative of OA, even in persons who report none of the usual symptoms.
- **Magnetic Resonance Imaging (MRI).** MRI studies are very sensitive tests. They are generally done to diagnose acute injuries but can often detect early signs of OA long before symptoms appear.
- **Ultrasound.** Ultrasound scans of osteoarthritic joints are particularly good at finding effusions, or fluid collections that might be too small to produce obvious swelling. Despite the sensitivity of ultrasounds and MRI, radiologists generally believe that ordinary X-rays are sufficient to make a diagnosis of OA.

## CAUSES AND RISK FACTORS

The causes of OA include:

- **Age.** The risk of OA increases with age, but after the age of 80 to 85, the risk falls a bit for reasons that are not clear.
- **Gender.** After the age of 55 to 60, women have a higher incidence of OA of the knee than men do. Some studies suggest that the decline in native estrogen after menopause might play a role in women's

greater risk, particularly since estrogen replacement therapy is associated with decreased risk.

- **Obesity.** In the Framingham OA Study, the risk of OA of the knees in women increased 40 percent for each ten-pound weight gain. Some researchers have estimated that if obesity were eliminated, the incidence of OA of the knee in both men and women would fall by 25 to 50 percent.
- **Previous injury.** Joint injury, such as a tear in a ligament or in the meniscus in the knee, increases the risk of developing OA. OA occurs more rapidly in older adults with joint injuries than in younger people. People who have held jobs requiring much physical labor, such as knee bending with heavy loads, can suffer chronic joint damage that leads to OA.
- **Muscle weakness.** Studies have shown that weakness in the quadriceps muscle that runs from the front of the thigh across the knee and down to the shin is associated with OA of the knee in people over 65. A decline in the stability of the knee with age also appears to increase the risk of OA.
- **Congenital abnormalities.** Congenital hip dysplasia, a structural abnormality, can lead to OA in later life, due to changes in the shape of the joint.
- **Joint infections.** Although it is unusual, joints can be infected by bacteria, viruses, fungi, and mycobacteria (the organisms that cause tuberculosis), all of which can penetrate joint spaces. This causes acute

damage and paves the way for chronic damage and future OA. Some of the most well known microbes that infect joints are the Lyme disease and gonorrhea bacteria.

- **Metabolic and hormonal disturbances.** A host of disorders feature OA as a complication. They include Paget's disease (in which bone is remodeled inappropriately), acromegaly (from excessive circulating growth hormone), and hemochromatosis (an iron storage disease).
- **Diet.** Studies have shown that diets deficient in vitamin C or vitamin D and perhaps vitamin K can increase the risk of progressive OA. In one study, evidence of continuing joint damage was reduced three-fold in those people with the highest dietary intake of vitamins C and D.

## PREVENTING OSTEOARTHRITIS

No studies have been done that can give a definitive solution to the challenge of preventing osteoarthritis. Maintaining an appropriate weight can decrease the risk of developing OA. Finding the correct balance between regular exercise and physical activity and allowing the joints to rest and recover from that activity may prevent OA. Avoidance of jobs and hobbies that require repetitive movements of the joints can also help. Interestingly, running does not lead to OA in people who have not experienced a joint injury.

## OSTEOARTHRITIS AND AGING

Aging, by itself, does not cause OA, but the changes that take place in cells and joints as we age allow OA to develop more easily.

Gout, repetitive trauma associated with jobs or other physical activities, and metabolic disorders are often associated with OA.

Aging can influence the choice of treatments for OA, since certain classes of pain relievers have adverse effects that can disproportionately affect older adults. For example, drugs such as ibuprofen, which belong to the category of non-steroidal anti-inflammatory drugs or NSAIDs, can produce kidney damage, gastrointestinal bleeding, and rarely, central nervous system and personality changes.

## TREATMENT

With the high cost of prescription (and over the counter) medications and possibility of adverse effects from medications, the American College of Rheumatology recommends beginning treatment for OA with non-drug methods. Studies support the value of carefully planned exercise programs in improving functional status and decreasing pain once OA has developed.

For example, a small Danish study of 25 patients with OA of the knees looked at the effects of a coordi-

nated training program focusing on fitness, balance, coordination, and muscle strength. At the end of one year, the patients experienced up to a 20% improvement in strength, a significant decrease in pain, and an increase of 13% in walking speed.

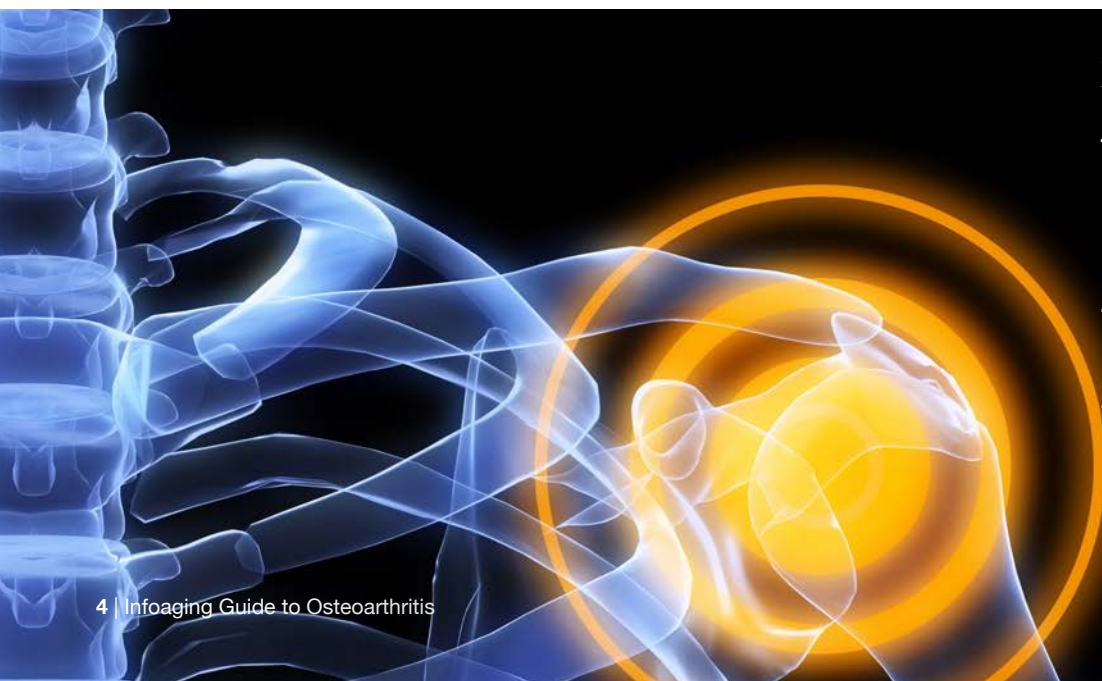
Another small study of 113 patients at the Veterans' Administration Hospital in Philadelphia compared an exercise program to an OA education program (talks on non-drug treatment of pain, preservation of joint function, and joint protection strategies). This study demonstrated that those who exercised had decreased pain, but both groups had improved strength, suggesting that patient education could play a role in improving quality of life for OA sufferers.

The Wake Forest School of Medicine in North Carolina, in conjunction with the University of Tennessee School of Medicine, conducted a larger study of 439 patients with OA of the knee, enrolling them in either an aerobic exercise program, a resistance exercise program, or an education program. Those who had entered the aerobic program had the largest improvements in

strength and endurance and the greatest decrease in pain. Those in the resistance training group derived intermediate benefits, and those in the education group had the smallest improvements. Other studies have shown, however, that participation in OA education programs can reduce pain in some sufferers.

In a follow-up study, recently published in the Archives of Internal Medicine, researchers at Wake Forest University examined the utility of exercise in improving OA. 250 persons with OA of the knee but no interference as yet in their activities of daily living (ADLs) were assigned to either a resistance exercise program, an aerobic exercise program, or a control group. They were followed for 18 months to determine what impact, if any, there was on their ADLs with either exercise program or with no exercise. Benefits were found with both forms of exercise. The likelihood of developing impediments in ADLs was 37 percent for those who exercised (either resistance or aerobic) and 52 percent for those in the control group.

Physicians recommend that people with OA rest their joints about every four to six hours. Exercise



X-rays of the bones and joints in a patient suspected of having OA will show narrowing of the spaces that should be seen in joints, thickening of the bone that lines the joints and the formation of bone spurs.

effectively squeezes the water out of the cartilage, and rest allows it to seep back in. A number of tissue studies in animal cartilage have demonstrated this mechanism. These joint rest treatments are aimed at both reducing pain and improving function.

Weight loss will certainly benefit those with OA. In the Framingham OA Study, an average of 10 pounds of weight loss over a 10-year period decreased the risk of OA of the knee by 50%.

Another study, called the [Arthritis, Diet, and Activity Promotion Trial \(ADAPT\)](#) demonstrated that exercise combined with moderate weight loss could improve function and reduce pain in knee OA. In ADAPT, patients with knee OA followed an 18-month program comprising physical exercise and a calorie-restricted diet. Results showed an average improvement of 24 percent in physical function and a 30.3 percent decrease in knee pain.

## MEDICATIONS FOR OA

Most painful conditions, including OA, involve some degree of inflammation. Inflammation is a complex immune response the body makes to injury, infection, or other damage. It includes the “recruitment” of various chemical substances in the body to combat the injury or infection. Though the inflammatory response is appropriate and often necessary, the substances or mediators it produces can worsen the damage. Medications that reduce the detrimental effects of the inflammatory response are often used for OA treatment. But in many individuals, medications that reduce pain without affecting inflammation can be just as effective as the anti-inflammatory medications. This suggests that

inflammation may not be the sole cause of pain for patients with OA.

Some of the medications used to treat OA include:

- **Traditional pain relievers.** Acetaminophen gives effective pain relief to many patients with OA. Because aspirin and medications such as ibuprofen are known to cause occasionally severe gastrointestinal bleeding, acetaminophen can be a safer choice. Acetaminophen can be taken at a dose of 1,000 mg, up to three times per day but should not exceed 3,000 mg in a day. Patients need to be aware of any other medications they are taking that may also contain acetaminophen (for example, some cold medications and some combination pain relievers) and include them when calculating their total dose for a day. Lower doses often give inadequate pain relief; higher doses can cause liver damage. Abusing alcohol while taking acetaminophen increases the risk of liver damage. Patients who try acetaminophen for OA should be aware that effective pain relief may take as long as two weeks.
- **Steroid injections.** Traditional treatments for flare-ups of joint pain have included injections of cortisone or other steroids into the joint spaces. These treatments can provide several

weeks of reduced pain. Individuals should receive no more than four injections in a single year.

- **Nonsteroidal Anti-Inflammatory Drugs (NSAIDs).** Topical or applied NSAIDs are available as creams and ointments and offer some relief from pain when rubbed over joints. Some absorption of the drugs through the skin and into the bloodstream is possible, so the fact that they are applied topically is not a guarantee that they will not cause gastrointestinal bleeding or kidney damage. In fact, a meta-study (review of a large number of studies) published in the June 2010 issue of the [Journal of Rheumatology](#), showed that although topical NSAIDs have fewer severe gastrointestinal adverse effects than the oral versions in older adults, a substantial proportion of these patients report systemic adverse effects from them. Further, about the same number of people (up to 21 percent) withdrew from topical therapy as did from oral therapy (up to 25 percent) because of adverse effects. Overall, however, it appears that topical NSAIDs are safer than the oral versions, particularly in adults over the age of 75 years.



- Whether oral or topical, traditional NSAIDs produce these adverse effects because they are not selective. While the medications inhibit inflammatory proteins, they cannot distinguish between those that are useful (cyclooxygenase-1 or COX-1, which protects the stomach and other organs) and those that are damaging (cyclooxygenase-2 or COX-2, which enhances inflammation and produces pain). Thus, they inhibit proteins released during inflammation that would actually prevent ulcers and gastrointestinal bleeding. Although NSAIDs do have potentially dangerous adverse effects, with careful management they can achieve excellent results, particularly in short-term treatment. When they are prescribed for long-term use, many physicians also prescribe an anti-ulcer medication to reduce the likelihood of gastrointestinal bleeding.

NSAIDs also include a class of drugs called COX-2 inhibitors, which generally cause fewer gastrointestinal side effects because they specifically

target the COX-2 protein. However, they do not lower the risk for other side effects such as kidney failure and cardiovascular events. Currently, the prescription drug celecoxib (Celebrex) is the only COX-2 inhibitor available to patients. Two others were pulled from the market when they were found to increase the risk of heart attacks, blood clots, and stroke. The U.S. Food and Drug Administration (FDA) now requires that the packaging for celecoxib and other NSAIDs contains a boxed warning indicating the risks associated with using these medications.

- **Hyaluronic acid.** Another treatment for OA is injection of hyaluronic acid. Hyaluronic acid is a normal component of joint fluid, and injecting it causes few adverse effects, although some individuals have experienced increased pain and swelling after an injection. Some people seem to benefit from these injections, but results from multiple studies show modest effects at best.

## THE FUTURE OF OSTEOARTHRITIS RESEARCH

At a recent American College of Rheumatology (ACR) symposium, presenters noted that the current understanding of OA is based in *pathomechanics*, which includes body mechanics, genetic factors, and any additional factors in the patient's behavior, lifestyle, or environment that might be contributing to the disease's development.

At another ACR meeting, lecturers presented new findings that suggested OA is actually an inflammatory disease, rather than simply a condition of wear and tear. As a result, much of future research will, in all probability, focus on the chemicals that cause inflammation in the body.

Other promising research areas include the body's stress response to an accumulation of unfolded or mis-folded proteins, genetic triggers of OA, activation of particular gene pathways to stop the progression of OA, and regeneration of healthy tissue through stem cell therapy.

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