Articular-Cartilage Lesions of the Knee and Osteoarthritis in Athletes: 
An Overview

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In 1743, William Hunter, English surgeon and anatomist, stated, “From Hippocrates to the present age, it is universally allowed that ulcerated cartilage is a troublesome thing and once destroyed, it is not repaired.”1,p6 Two hundred sixty years later, his statement still holds true. Articular cartilage does not repair itself to its original form once injured. Although there are many techniques to repair articular lesions, a method to completely restore damaged cartilage remains elusive. Athletes are especially susceptible to cartilage lesions. These injuries cause pain, swelling, mechanical symptoms, and functional disability and can lead to osteoarthritis. An important area of musculoskeletal research is in the treatment of these injuries and prevention of osteoarthritis.

Structure and Composition

Articular cartilage is a viscoelastic material that reduces stress to underlying bone during functional activities. Normal articular cartilage is composed of water, a matrix, and chondrocytes (cartilage cells). The percentage of water by mass is about 65–80%. The matrix contains Type II collagen and proteoglycans (protein molecules). The proteoglycans are linked to hyaluronate, and together with collagen fibrils they form a lattice-type framework that gives cartilage its tensile strength. The chondrocytes are mesenchymal cells that are responsible for synthesizing the matrix. The number of chondrocytes in a person’s body is set at birth. Once these cells are injured, they have a very limited capacity to heal themselves. Their healing response is age dependent; older athletes’ chondrocytes have a limited capacity for healing lesions and are susceptible to further damage. The components of cartilage are arranged in a complex pattern that produces the functional and biomechanical properties of cartilage (Figure 1). When damaged, cartilage loses its resilience and optimal load-bearing characteristics.2

Pathogenesis

The cartilage on the joint surface is called hyaline or articular cartilage. Injuries to articular cartilage result from mechanical trauma. Repetitive compressive forces or sudden impacts cause many articular lesions. High shear forces at the subchondral bone junction are especially
The cartilage injuries can be divided into three types: microdamage to the cells without visible damage (bone bruise on MRI), chondral fracture, or fracture of the articular surface with subchondral bone penetration. The healing response depends on the depth of the lesion and the specific injury mechanism (e.g., acute vs. chronic).

Partial-thickness lesions do not have the capacity to heal. Mature articular cartilage is avascular, and the healing mesenchymal cells cannot enter the site of injury. Full-thickness injuries that involve the subchondral bone behave differently. Once the subchondral bone has been penetrated, the lesion has the capacity to mount a healing response. The reparative process includes a fibrin clot that contains mesenchymal cells, which can synthesize fibrocartilage. The replacement cartilage is not equivalent to the original, however. The new cartilage is fibrocartilage, or “scar” cartilage. It has biomechanical properties inferior to those of the original hyaline cartilage. This means that a layer of fibrocartilage is more susceptible to recurrent injury and predisposes adjacent cartilage to damage, which is a major problem in young athletes. Athletic individuals who continue to participate in strenuous impact-loading activities after having sustained a knee articular-cartilage lesion risk further damage and the development of osteoarthritis.

Clinical Features

An athlete with an articular-cartilage defect might present with an acute injury or with a prolonged history of pain with an insidious onset. The pain might be