Osteoarthritis (OA) is a joint disease characterized by progressive degeneration and loss of articular cartilage, ultimately resulting in severe pain and disability. Articular cartilage derives its functional mechanical properties from its extensive extracellular matrix (ECM) of type II collagen and proteoglycans. In OA, ECM degeneration is characterized by extensive proteolysis of the type II collagen network and proteoglycans (1, 2). Relationships between these structural changes and altered cartilage mechanical properties have been observed at all stages of OA degeneration. This study investigated the changes of mechanical properties and surface roughness in OA progression.

Animal samples were obtained following the Guidelines of the Internal Committee for the Care and Use of Laboratory Animals (NOM-069-ZOO-1999). Normal articular and osteoarthritic cartilage was obtained from femoral condyles of male adult Wistar rats (120-150 g) and rats with OA induced by partial meniscectomy (5, 10, 20 and 45 days after surgery compared with normal). Full-thickness rat normal and osteoarthritic articular cartilages from weight bearing areas were fixed with 4% PBS paraformaldehyde at 4°C, and cryosectioned in 6 µm. The samples were scanned with an atomic force microscope (AFM) (Autoprobe CP Research, Thermomicroscopes) operating in contact mode.

In normal cartilage it was observed collagen fibers in double or triple junction, which provides strength and stability to the cartilage. In OA cartilage (45 days after induction) we saw that the integrity of these junctions were lost.

Using AFM, we can get new images of microstructures that help us to understand the biological processes occurring in the OA pathogenesis. These results are part of a comprehensive study on osteoarthritis in the Wistar rat model; which provides an insight of OA pathogenesis.

References

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Fig. 1: Normal cartilage. Left 2D image, double or triple very tight binding of the collagen fibers is observed. In right side 3D image, viewed from another perspective or triple pairing arrangement of the collagen fibers.

Fig. 2: OA cartilage. Left 3D image of a sample of OA cartilage 45 days after induction, where the disorder of the collagen fibers is observed, we also observed a similar formations holes in the extracellular matrix. In right side another 3D perspective in which the loose arrangement of the collagen is clearly observed.