Bone Composition in Osteoarthritis

Beamline: U10B

Technique: Infrared microspectroscopy

Researchers:
L. Miller, M. Ruppel, J. Novatt (NSLS)
D. Hamerman (AECOM)
C.S. Carlson (Univ. Minnesota)

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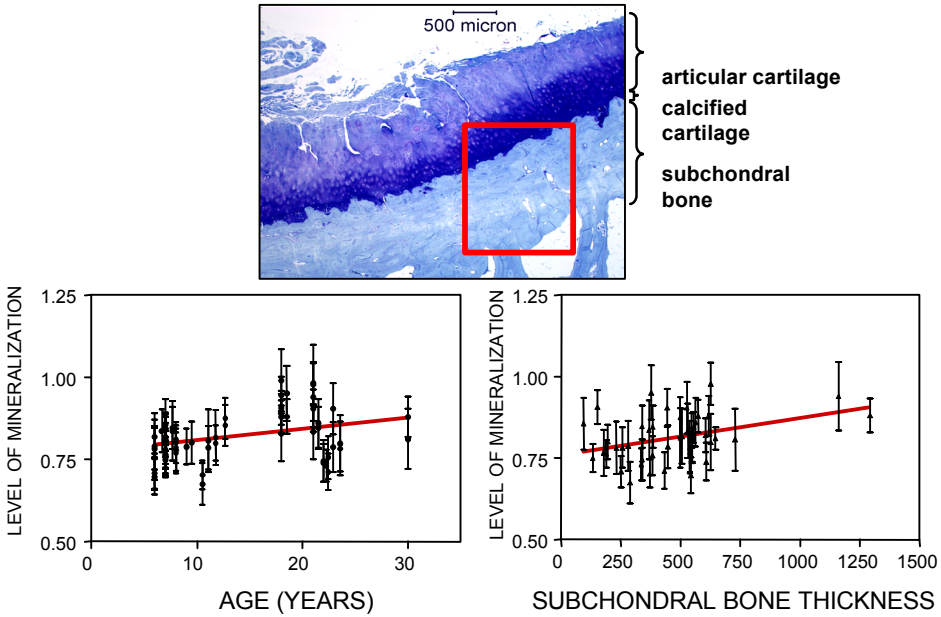
Motivation: Osteoarthritis (OA) is a prevalent joint disease that affects over 40 million Americans and is characterized by degeneration of the articular cartilage and thickening of the underlying subchondral bone. Although subchondral bone thickening has been implicated in articular cartilage degeneration, very little is known about the composition of subchondral bone in OA. In the present study, infrared microspectroscopy (IRMS) was used to determine the chemical composition of the calcified cartilage/subchondral bone plate in a monkey model of OA as a function of age and OA severity.



(Top) Light microscope image of a monkey knee joint illustrating the articular cartilage, calcified cartilage, and subchondral bone. (Bottom, left) Level of mineralization in the subchondral bone plate as a function of animal age. (Bottom, right) Level of mineralization in the subchondral bone plate as a function of subchondral bone thickness.

Results: Results demonstrated that the level of mineralization and carbonate content increased as a function of animal age. In addition, bone mineralization level increased as subchondral bone thickness increased. Dramatic increases in the mineralization level and carbonate accumulation were also observed as a function of the number of tidemarks. The presence of multiple tidemarks indicates the occurrence of one or more additional phases of cartilage calcification, suggesting that the observed compositional changes are due to cartilage mineralization. Our results support a re-activation of endochondral ossification that occurs with age, which is more pronounced in OA. Moreover, our findings show that the newly mineralized cartilage has an abnormally high mineral content. In summary, compositional analysis of the mineralized plate beneath the articular cartilage in OA is characterized by an overmineralized calcified cartilage and thickened subchondral bone, both of which likely put added mechanical stress on the joint, contributing to the progression of OA.