Subchondral bone plate migration into trabecular bone in response to direct bone-on-bone contact wear in patients with severe knee osteoarthritis.

Meret Keiser¹, Stephan Preiss², Stephen J. Ferguson³, Vincent. A. Stadelmann¹ Department of Research and Development, Schulthess Klinik, Zürich, CH ²Department of Hip and Knee Surgery, Schulthess Klinik, Zürich, CH ³Institute for Biomechanics, ETH Zürich, Zürich, CH

Disclosures: V.A. Stadelmann: None. M. Keiser: None. S. Preiss: None. S.J. Ferguson: None

INTRODUCTION: Although it is well established that osteoarthritis (OA) is accompanied by changes in subchondral bone microstructure, often characterized by cortical thickening. However, there remains a gap in understanding the specific microstructural adaptations that occur around full-thickness cartilage defects in cases of severe knee OA. This study, employing high-resolution microCT scanning, aimed to investigate the intricacies of subchondral bone changes in response to direct bone-on-bone contact in individuals affected by severe OA.

METHODS: Ten tibial plateaus showing full-thickness cartilage defects were retrieved from total knee arthroplasty patients. All patients provided signed informed consent. Six 5x5 mm specimens were cut with a diamond saw from the defect, defect-edge, and cartilage region (Fig. 1) and two from the contralateral side. These were microCT-scanned at a resolution of 3.3 μm. Bone volume fraction (BV/TV) and Tissue Mineral Density (TMD) were analysed layer-by-layer to compute the specimens' density profiles and identify cortical and trabecular bone regions. Additionally, bone formation surrounding cortical porosity was deduced from the mineral density gradient adjacent to these pores.

RESULTS SECTION: The subchondral bone plate below full-thickness cartilage defects migrates into trabecular bone through direct thickening and through pockets of bone formation within the trabecular structures. In cases with deep wear grooves, trabecular pores occasionally connect with the joint space before they are filled with new bone. The spatial expansion of these effects shows a sharp boundary at the defect-edge, almost immediately disappearing under regions covered with even a minimal layer of cartilage: over 90% of the difference in cortical thickness happens within 1mm of the defect edge! Quantitatively, cortical bone was 4-fold thicker below defects than below cartilage (Fig. 2). BV/TV and TMD profiles significantly differed between defect, edge, and cartilage specimens, up to 5mm deep. Below defects, cortical porosity and trabecular BV/TV were 85% and 14% higher and TMD 6% lower compared to cartilage specimens. The TMD gradient around pores was greatest in the edge specimens.

DISCUSSION: this study introduces novel perspectives regarding bone microstructural adaptations beneath full-thickness cartilage defects in severe knee OA. Notably, our observations reveal subchondral plate migration triggered by trabecular osteogenesis beneath cartilage defects. The data also underscores heightened bone formation within defect and edge regions, a manifestation of wear-induced changes in native cortical bone and the ensuing trabecular corticalization, ultimately culminating in the migration of the cortical layer into the trabecular bone. A surprising finding was the limited extent of the transition between thickened and normal cortical bone plate. Most of the difference was in the close vicinity of the defect edge. These observations hold implications for refining therapeutic strategies and expanding our comprehension of the intricate interplay between bone remodeling and cartilage damage in severe OA.

SIGNIFICANCE/CLINICAL RELEVANCE: These findings are important for advancing our understanding and clinical approaches for severe knee OA. Knowing how bones change around damaged cartilage could lead to better treatments, enhancing the quality of life for those with this condition.

ACKNOWLEDGEMENTS: Support for this research was provided by Wilhelm Schulthess-Stiftung, Zürich, Switzerland. We thank Fabio Liechti for his efforts in the validation of the methodology.

IMAGES AND TABLES:

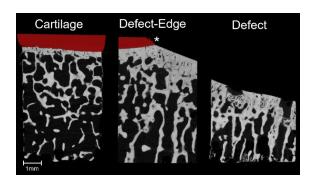


Fig. 1: MicroCT images of bone specimens, from left to right: cartilage, edge defect and defect specimen. The specimens are oriented with the joint space at the top and the level of the surgical cut at the bottom. Cartilage is enhanced in red. The * marks the start of the defect in the defect-edge specimen.

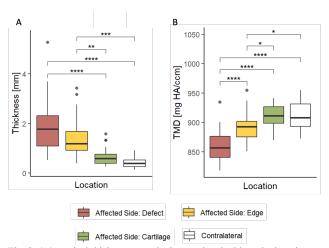


Fig. 2: (A) cortical thickness negatively correlated with probe location from defect to intact cartilage, while (B) tissue mineral density positively correlated. (Both observations p < 0.05).