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S E M I N A R

aus

Halbleiterphysik und Nanotechnologie

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"Nanostructural Mechanisms of Mineralized Tissue Toughness and Their Modification in Metabolic Bone Disease"

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Bone tissue types consist of a nanoscale mesh of collagen fibrils impregnated with mineral (carbonated apatite) particles inside and on their surface, along with a small fraction of noncollageneous proteins. These constituent elements assemble into fibre bundles, lamellae and osteons forming a hierarchical structure and mechanical deformation at interfaces between elements at each level is expected to be critical to the high work to fracture of such systems. While these interfacial mechanisms are clearly demonstrated at the microscale (crack reorientation inside and between lamellae, and crack bridging) the corresponding mechanisms at the nanoscale are controversial especially in the realm of macroscopic inelasticity. We investigate the operative toughening mechanisms in antler, an exceptionally tough, low mineralized bone type. Using in situ tensile testing combined with synchrotron small angle X – ray scattering and diffraction, the fibrillar and mineral strain are measured in the zone of inelastic deformation during uniaxial and cyclic loading. Evidence of fibrillar plasticity is clearly seen, with minimal hysteresis, suggesting a permanent structural deformation at the nanoscale. By modifying a previously proposed two-level staggered model for tensile deformation of nanocomposites, we find that these results are very well explained by an interfacial sliding between the mineral and collagen components inside the fibril. The model can be generalized to macroscopically higher mineralization where it matches experimental measurements of elastic moduli well. We then demonstrate that these nanoscale mechanisms can be severely disrupted in metabolic bone diseases, using a murine model of hypophosphatemic rickets (HPR) generated by ENU-mutagenesis. The strain in the fibrils increased and effective fibril modulus decreased, both significantly, in rachitic bone compared to wild type. We propose a structural mechanism of partial (patchy) mineralization in HPR mice to explain these results. Our results show the ability of in situ synchrotron X-ray scattering techniques, when combined with micromechanics, to identify toughening mechanisms at the nanoscale, as well as the functional reasons for reduction of skeletal tissue mechanical competence in bone diseases at this length scale.