ON THE FRACTURE MECHANICS OF BONE AND ITS BIOLOGICAL DEGRADATION

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The age-related deterioration of both the fracture properties and the architecture of bone, coupled with increased life expectancy, are responsible for increasing incidences of bone fracture in the elderly segment of the population. In order to develop effective treatments, an understanding of the mechanisms underlying the structural integrity of bone, in particular, its fracture resistance, is essential. The origins of the toughness of human cortical bone (and dentin, a primary constituent of teeth and simple analog of bone) are described in terms of the contributing micro-mechanisms and their characteristic length scales in relation to the hierarchical structure of these mineralized tissues. It is shown that although structure at the nanoscale is important, it is microstructural features at the scale of one to hundreds of microns (e.g., the Haversian systems present in the cortical bone of mammals and the tubule size and spacing in dentin) that are most important in determining fracture risk.¹⁻³ We specifically find that the origins of fracture resistance in materials such as bone are extrinsic, i.e., associated primarily with crack growth, and are related to such toughening mechanisms as gross crack deflection and crack bridging (Figs. 1-2), both processes that are induced by preferential microcracking (at cement lines in bone and at unfilled tubules in dentin).³ In particular, our results, in terms of full nonlinear elastic crack-resistance curve measurements, show that human cortical bone is actually much tougher than has been previously thought, because it is largely associated with the growth, rather than the initiation, of cracking. In this context, realistic short-crack measurements of both initiation and growth toughnesses performed on human and small animal bones and human and elephant dentin are used to evaluate the effects of aging and certain therapeutic treatments (e.g., steroids and bisphosphonates). These measurements are combined with structure characterization using UV Raman spectroscopy, small-angle x-ray scattering and transmission electron microcopy and imaging studies involving two-dimensional in situ fracture tests performed in an environmental scanning electron microscope (including quantitative electron backscattering analysis) and three-dimensional ex situ examination of crack paths derived using synchrotron x-ray computed tomography $(e.g., Figs. 1-2)^5$, to determine the microstructural features that underlie the toughness of bone and teeth and how these properties can degrade with biological factors.^{2,4}

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Fig. 1: Synchrotron x-ray computed tomography images of a crack path in human cortical bone (humerus, 37 yr donor) obtained in a notched 3-point bend geometry. The voxel size was 10 μ m. The crack propagation direction (L-R in the images) was perpendicular to the long axis of the bone, i.e., in the transverse (breaking) orientation. A 3-D image showing the Haversian system is shown on the left, and a subsurface slice is shown on the right. The crack has extended approximately 1 mm from the notch and has undergone several deflections, which significantly increases the measured toughness. The arrow in the right-hand image shows that one such deflection occurs at a sub-surface Haversian canal. Examination of the full series of sub-surface slices found that all crack deflections observed at the surface could be associated with cement lines, lamellar boundaries, or Haversian canals present in the in the Haversian system. (after ref. 5).



Fig. 2: Three-dimensional synchrotron x-ray computed tomography image of a crack path in human cortical bone (humerus, 37 yr donor), again in the transverse (breaking) orientation, showing the toughening obtained by crack deflection, and more importantly crack twisting, as the crack path encounters the interfaces of the Haversian canals (the cement lines). (Unpublished data from Advanced Light Source beamline 8.3.2: Barth and Ritchie).

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