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Perspective Article

Hylonome

Collagen glycation and its role in fracture properties of bone

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Age-related non-traumatic fractures are a major public health problem. Although lower bone mass is the most commonly implicated variable for the age-related loss of bone strength and increased fracture incidence, recent evidence shows that the resistance of bone material against fracture (toughness) diminishes with age[1,2]. The mechanisms for the age-related loss of toughness are, however, unknown. Bone derives its resistance against fracture from collagen deformation[3] and its ability to form microcracks[4-6] and uncracked ligament bridges[7] during crack propagation. Collagen deformation, microcracking and uncracked ligaments are the primary toughening mechanisms in bone and any alteration in these toughening mechanisms will affect bone toughness. Using an *in vitro* ribosylation procedure, we demonstrate that the non-enzymatically (NEG) mediated accumulation of collagen cross-links stiffens the organic network reducing collagen deformation and measures of microcracking and crack bridging in bone. More significantly, NEG-induced collagen cross-links were highly correlated with the stiffness of the organic matrix in bone and the fracture properties of mineralized bone. Thus, NEG-mediated stiffening of the organic matrix causes loss of bone toughness. Furthermore, as post-yield and damage behavior of cancellous bone are independent of bone volume fraction[8] and similar to cortical bone[9,10], the organic matrix-mediated loss of toughness may be common to both cortical and cancellous bone. Consistent with this concept, the experiments conducted in our laboratory demonstrate that NEG induced cancellous bone fragility and that age-related loss of toughness properties are similar in both cortical and cancellous bone.

The author has no conflict of interest.

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