

Role of microstructure in the aging-related deterioration of the toughness of human cortical bone

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Abstract

The aging-related deterioration of the fracture properties of bone, coupled with higher life expectancy, is responsible for increasing incidence of bone fracture in the elderly; consequently, an understanding of how these fracture properties degrade with age is essential. In this study, *ex vivo* fracture experiments have been performed to quantitatively assess the effect of age on human cortical bone in the proximal–distal orientation, i.e., longitudinally along the osteons. Because cortical bone exhibits rising crack-growth resistance with crack extension, the toughness is evaluated in terms of resistance-curve (R-curve) behavior, measured for bone taken from wide range of age groups (34–99 years). Using this approach, both the crack-initiation and crack-growth toughness are determined and are found to deteriorate with age; the initiation toughness decreases some 40% over six decades from 40 to 100 years, while the growth toughness is effectively eliminated over the same age range. The reduction in crack-growth toughness is considered to be associated primarily with a degradation in the degree of extrinsic toughening, in particular, involving crack bridging in the wake of the crack. An examination of the micro-/nano-structural changes accompanying the process of aging, using optical microscopy, X-ray tomography, nanoindentation and Raman spectroscopy, is shown to support such observations.

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1. Introduction

There is mounting evidence that the traditional thinking concerning “bone quality”, which has largely focused on bone mass or bone mineral density as a predictor of fracture risk, is insufficient [1–3]. This is particularly a concern as aging-related changes to the musculoskeletal system are known to increase the susceptibility to bone fracture [1]; indeed, for the very elderly, the consequent fractures can lead to mortality [4]. This has led to a renewed interest into how aging can alter the various mechanical properties of bone, and in particular the fracture resistance. Specifically, there have been several recent studies that have focused on age-related issues and have shown a significant deterioration in the fracture toughness of bone with age (e.g., [5–13]).

In order to characterize the deterioration of bone with age, generally the fracture toughness, K_{Ic} , or the strain-energy release rate, G_c , has been used as a single-parameter approach to characterize the resistance to fracture. However, in many materials, including cortical bone, so-called *extrinsic* toughening mechanisms, such as constrained microcracking or crack bridging [14], are active. In general, crack propagation can be considered as a mutual competition between two classes of mechanisms: *intrinsic* mechanisms that operate ahead of the crack tip, and affect the material's inherent resistance to fracture and damage, and *extrinsic* mechanisms that principally operate in the wake of the crack tip, and “shield” the crack from the applied driving force [15–17]. Whereas intrinsic mechanisms primarily govern the crack-initiation toughness, extrinsic mechanisms, specifically crack bridging in bone [18], operate in the crack wake and govern the crack-growth toughness. As the effect of extrinsic mechanisms is dependent on the size of the crack and stable crack growth can occur prior to unstable fracture, this requires a “resistance-curve” approach

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to evaluate the fracture toughness [19]. Despite this, R-curves have only been utilized in relatively few studies [13,14,18,20,21] to characterize human bone fracture.

Mechanistically, it has been suggested that the elevated bone turnover in older bone, although beneficial in repairing damage [22], may have a deleterious effect on the toughness due to the formation of resorption cavities and hence increased porosity. Elevated turnover also results in a higher density of secondary osteons [23], and associated cement lines which are known to provide weak interfaces, and hence preferred (weaker) paths, for cracking [13,24–27]. In the present paper, we seek to investigate the ex vivo R-curve fracture toughness properties of human cortical bone as a function of age, with the aim of elucidating the role that these age-related changes in the microstructure play. Furthermore, we show that the age-related changes in macroscopic

properties can be linked to changes of the hierarchical structure of bone at the micro- and nano-scales [28] (Fig. 1). This is achieved specifically at the nanoscale using both nanoindentation, to obtain moduli of the collagen fibers that constitute the osteons, and deep-ultraviolet (UV) Raman spectroscopy, to ascertain corresponding changes in the cross-linking in the collagen.

2. Experimental methods and materials

2.1. Fracture toughness testing

Fresh frozen human cortical bone taken from the humerus of nine cadavers (donor age: 34 to 99 years) was used. Blocks of bone were obtained by carefully sectioning the medial cortices of the mid-diaphyses of the humeri. Seventeen ($N=17$)

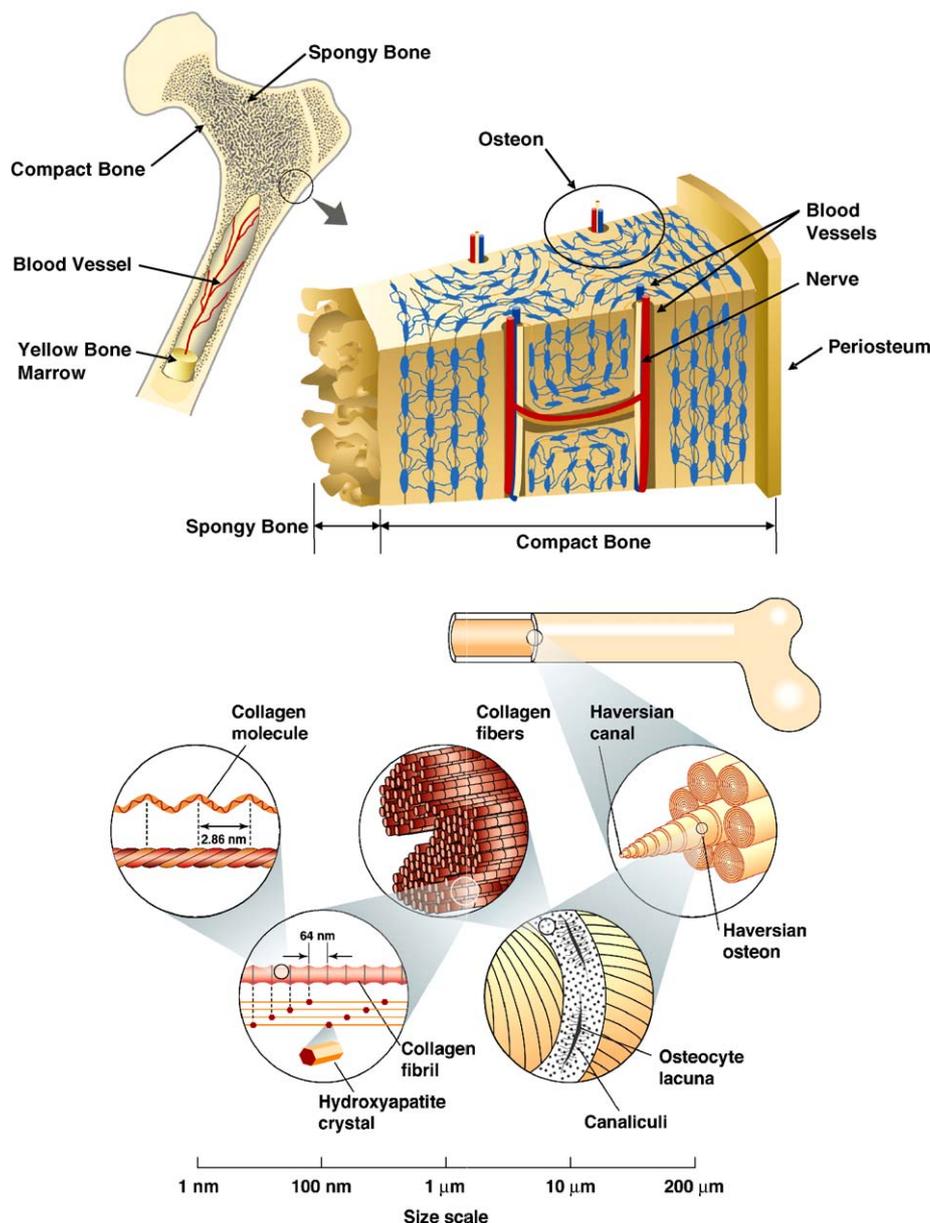


Fig. 1. Hierarchical structure of human cortical/compact bone.

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