Biomaterials 35 (2014) 5472–5481

Contents lists available at ScienceDirect

Biomaterials

journal homepage: www.elsevier.com/locate/biomaterials

Fracture resistance of human cortical bone across multiple length-scales at physiological strain rates

Elizabeth A. Zimmermann [a], Bernd Gludovatz [a], Eric Schaible [b], Björn Busse [a,c], Robert O. Ritchie [a,d,*]

[a] Materials Sciences Division, Lawrence Berkeley National Laboratory, Berkeley, CA, USA
[b] Experimental Systems Group, Lawrence Berkeley National Laboratory, Berkeley, CA, USA
[c] Department of Osteology and Biomechanics, University Medical Center, Hamburg, Germany
[d] Department of Materials Science and Engineering, University of California, Berkeley, CA, USA

ARTICLE INFO

Article history:
Received 23 January 2014
Accepted 22 March 2014
Available online 13 April 2014

Keywords:
Bone
Strain rate
Fracture toughness
Plasticity
X-ray diffraction

ABSTRACT

While most fracture-mechanics investigations on bone have been performed at low strain rates, physiological fractures invariably occur at higher loading rates. Here, at strain rates from 10^{-5} to 10^{-1} s^{-1}, we investigate deformation and fracture in bone at small length-scales using *in situ* small-angle x-ray scattering (SAXS) to study deformation in the mineralized collagen fibrils and at the microstructural level via fracture-mechanics experiments to study toughening mechanisms generating toughness through crack-tip shielding. Our results show diminished bone toughness at increasing strain rates as cracks penetrate through the osteons at higher strain rates instead of deflecting at the cement lines, which is a prime toughening mechanism in bone at low strain rates. The absence of crack deflection mechanisms at higher strain rates is consistent with lower intrinsic bone matrix toughness. In the SAXS experiments, higher fibrillar strains at higher strain rates suggest less inelastic deformation and thus support a lower intrinsic toughness. The increased incidence of fracture induced by high strain rates can be associated with a loss in toughness in the matrix caused by a strain rate induced stiffening of the fibril ductility, *i.e.*, a "locking-up" of the viscous sliding and sacrificial bonding mechanisms, which are the origin of inelastic deformation (and toughness) in bone at small length-scales.

Published by Elsevier Ltd.

1. Introduction

Traumatic injuries, such as falls, often can lead to bone fractures. This fragility is especially significant in the elderly, where broken bones can be associated with a further deterioration in health [1]. As these traumatic injuries invariably result from loading over short time-scales, it is necessary to understand how the structural framework of the human body resists fracture at such physiologically high strain rates.

The fracture resistance of human cortical bone is a direct result of its hierarchically assembled structure of collagen and hydroxyapatite (HA) mineral, which spans multiple length-scales from molecular to near-macroscopic dimensions (Fig. 1) [2,3]. Basically, there are two major contributions to the fracture toughness of bone,[1] namely, intrinsic toughening mechanisms that promote "plasticity", *i.e.*, ductility in the mineralized tissue, and extrinsic toughening mechanisms that act to "shield" a growing crack from the global stresses and strains.[2] The intrinsic toughness represents the inherent fracture resistance of the material and is developed at small (sub-micron) length-scales; here, the fibril can elastically stretch through cooperative deformation between the mineral and collagen [8,9] as well as absorb further deformation through inelastic mechanisms, such as intra/interfibrillar sliding, breaking/reforming of sacrificial bonds, and even through the opening of dilatational bands at the mineral/collagen interface [8,10–12]. The extrinsic toughness of bone, conversely, is a primary function of how the microstructure can inhibit the growth of a crack; essentially, as a

* Corresponding author. Department of Materials Science and Engineering, University of California, Berkeley, CA, USA. Tel.: +1 510 486 5798; fax: +1 510 643 5792.
E-mail address: RORitchie@lbl.gov (R.O. Ritchie).

[1] The fracture toughness can be expressed as the critical value of the stress intensity K for unstable fracture in the presence of a pre-existing crack, *i.e.*, in mode I when $K = Y\sigma_{app}(\pi a)^{1/2} = K_{Ic}$, where σ_{app} is the applied stress, a is the crack length, and Y is a function (of order unity) of crack size and geometry. Alternatively, the toughness can be expressed as a critical value of the strain-energy release rate, G_c, defined as the change in potential energy per unit increase in crack area, or the nonlinear elastic version of G, the J-integral.

[2] Fracture resistance can be considered as a mutual competition between two classes of mechanisms: *intrinsic* mechanisms, which are microstructural damage mechanisms that operate ahead of the crack tip to promote cracking, and *extrinsic* mechanisms, which operate principally in the wake of the crack tip to inhibit cracking by "shielding" the crack from the applied driving force [4–7]. Whereas intrinsic toughening mechanisms, principally plastic deformation, act in general to resist intrinsic microstructural damage and thus are effective in inhibiting both the initiation and growth of cracks, extrinsic toughening mechanisms, *e.g.*, crack bridging, are only effective in inhibiting crack growth [6].

http://dx.doi.org/10.1016/j.biomaterials.2014.03.066
0142-9612/Published by Elsevier Ltd.

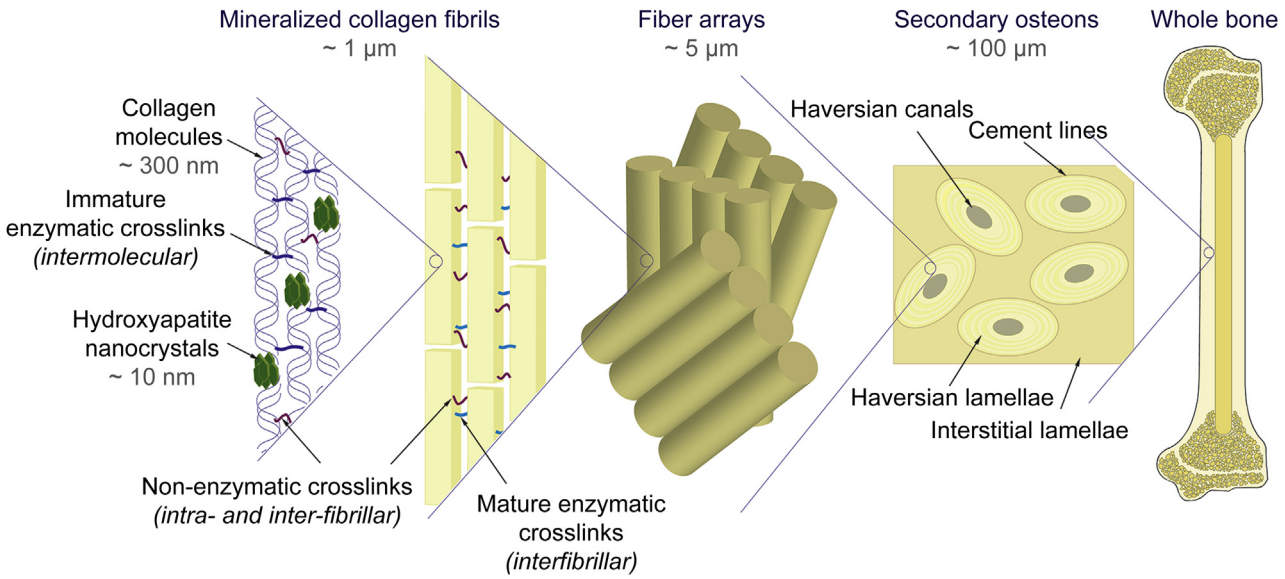

Fig. 1. The structure of human cortical bone spans multiple size-scales, which allows it to develop strength and resistance to fracture. At the smallest length-scales, human cortical bone is composed of an array of collagen molecules embedded with hydroxyapatite (HA) mineral crystals. The collagen and mineral comprises an array of mineralized collagen fibrils with various types of cross-links stabilizing the array and the individual fibrils. At higher length-scales, secondary osteons are the main motif at the microstructural scale. The osteons have a central vascular cavity (the Haversian canal) that is concentrically surrounded by lamellae, which are composed of collagen fibers. At the outer boundary of the osteon is a boundary called the cement line, which has a higher mineralization relative to the surrounding bone matrix. Adapted from Ref. [25].

crack begins to grow, the interaction of the crack path with the microstructure can lead to mechanisms such as crack deflection and bridging, which "shield" the crack tip from the full stress intensity, thereby increasing the bone toughness [6]. Because most cracks are on the micron-scale, these extrinsic toughening mechanisms are most effective when they interact with structural length-scales of comparable dimensions, *i.e.*, ~10s–100s μm, specifically with the osteonal systems through which bone remodels. The osteons consist of circumferential lamellar structures surrounding the Haversian canals (Fig. 1) and with an outer boundary separating the osteon from the interstitial matrix called the cement line, which is thought to have a relatively higher mineralization than the surrounding bone tissue[3] [14,16]. As a large population of the microcracks created in bone form within the interstitial bone (*i.e.*, the bone matrix between the osteons) [17], a growing crack which impinges on the osteonal borders (*i.e.*, the cement lines) is invariably subject to crack deflection and/or twisting,[4] often causing delamination along the cement lines; furthermore, the resulting intact material left between the microcracks can lead to so-called "uncracked ligament" bridges spanning the main crack wake, which further enhance the crack-tip shielding [19,20]. In this manner, through a combination of intrinsic "plasticity" mechanisms at small (sub-micron) length-scales and crack-tip shielding mechanisms at larger length-scales, healthy human cortical bone develops numerous potent mechanisms that can act to resist bone fracture.

Many studies on the strength and toughness properties of human cortical bone have shown succinctly that the complex hierarchical bone-matrix structure at both small and large length-scales is proficient in resisting the initiation and propagation of the major cracks that can cause bone fractures [19,21–26]. However, the reality is that most of these studies have been conducted at low strain rates on the order of 10^{-4} s^{-1} where it is easier to observe and collect data, whereas most physiological bone fractures are generally associated with much higher strain rates. For example, *in vivo* loading rate measurements on bone suggest strain rates of ~0.007–0.013 s^{-1} during walking or running and strain rates as high as ~0.02 s^{-1} during sprinting or downhill running [27–29]; other studies simulating a fall have shown that it takes ~6–10 ms for a falling femur to reach the peak load once it has begun to make contact with the ground [30], with an upper bound for these high strain rates to be ~25 s^{-1} for very high impacts [31]. As these physiologically realistic situations represent strain rates some four or more orders of magnitude higher than those used in most bone fracture experiments in the laboratory, characterizing and understanding the role of loading rate in influencing the multi-scale mechanisms by which bone resists fracture is clearly pertinent.

Previous studies have characterized the strength and toughness of bone at a wide range of strain rates [32–42]. The majority of studies point towards a ductile to brittle transition in bone, where there is a progressive decrease in the amount of post-yield ductility as well as increase in strength and modulus [34–37,40–42]. Notched toughness tests have also been performed at various strain rates and generally show a decrease in toughness at higher strain rates with a corresponding decrease in the accumulation of damage [32,33,38,39].

Consequently, we analyze here the mechanical response of bone over multiple length-scales at physiological strain rates of ~10^{-5}–10^{-1} s^{-1}. Using *in situ* synchrotron small- and wide-angle x-ray scattering/diffraction (SAXS/WAXD) during uniaxial tensile testing and fracture-mechanics-based fracture toughness analyses, we examine the specific roles of plasticity on intrinsic toughness at sub-micron dimensions and the role of crack-tip shielding on extrinsic toughness at the scale of ~1–100s μm to investigate whether the salient mechanisms of toughening in bone are still as effective in resisting bone fractures at physiologically high strain rates.

[3] The composition of the cement lines has been a matter of debate in the literature [13–16]. However, the general consensus is that the cement lines in healthy bone represent regions of high mineralization relative to the surrounding bone matrix or a collagen deficient feature in the bone microstructure [14,16].

[4] The deflection of a crack from a path of maximum tangential stress, essentially the path of maximum strain-energy release rate G, can lead to significant reductions in the crack-driving force experienced locally at the crack tip. Typically, an in-plane crack deflection of ~90° can reduce the stress intensity K at the crack tip by almost a factor of two; if out-of-plane twisting of the crack path occurs, the reduction in the crack-tip K can be even higher [18].