Acta Biomaterialia 8 (2012) 1093–1100

Contents lists available at SciVerse ScienceDirect

Acta Biomaterialia

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

Sub-lamellar microcracking and roles of canaliculi in human cortical bone

Vincent Ebacher [a], Pierre Guy [b], Thomas R. Oxland [b], Rizhi Wang [a,*]

[a] Department of Materials Engineering, University of British Columbia, Vancouver, BC, Canada V6T 1Z4
[b] Department of Orthopaedics, University of British Columbia, Vancouver, BC, Canada V6T 1Z4

ARTICLE INFO

Article history:
Received 16 June 2011
Received in revised form 21 September 2011
Accepted 10 November 2011
Available online 17 November 2011

Keywords:
Bone fracture
Cortical bone
Inelastic deformation
Microcracking
Canaliculi

ABSTRACT

Bone is a tough biological material. It is generally accepted that bone's toughness arises from its unique hierarchical structure, which in turn facilitates distributed microcracking prior to fracture. Yet, there has been limited progress on the detailed roles of the structural elements in the microcracking process. The present study examines the structure–microcracking relations at the lamellar and sub-lamellar levels of human cortical bone subjected to compressive loading. Laser scanning confocal microscopy revealed a clear influence of the local structure and porosity of the Haversian systems' lamellae on microcrack development. In particular, crack initiation and growth under transverse compression were associated with stress concentration at canaliculi. Later stages of microcracking showed extensive sub-lamellar cracks forming cross-hatched patterns and regularly spaced 0.5–1.7 μm apart. The density, size and regularity of the crack patterns suggest enhanced inelastic deformation capacity through cracking control at the level of mineralized collagen fibril bundles. The present study thus improves the current understanding of the nature of inelastic deformation and microcracking in bone and further suggests that bone's resistance to fracture is achieved through microcrack control at multiple length scales.

1. Introduction

Bone is a nanocomposite of carbonated apatite nanocrystals and organic phases mainly composed of fibrous collagen protein. It is known for its unique hierarchical structure which has been hypothesized to give rise to its exceptional mechanical performance [1–4]. Although highly mineralized and with an extensive porosity network, human cortical bone exhibits remarkable inelastic deformation [4–8]. Such deformation is critical to its resistance to fracture as it relaxes stress concentrations [8–12]. However, how bone's different structural levels, from the mineralized collagen fibrils to the Haversian systems (or secondary osteons) and their lamellar organization, are involved in the deformation process is still poorly understood.

Bone's inelastic deformation at the nano-scale has been associated with slip at mineral/collagen interfaces [7,13], increased energy dissipation arising from nanostructural heterogeneities [14], interfibrillar shear sliding [15–17], and unfolding of non-collagenous proteins (NCPs) [18,19]. At the micro-scale, the inelastic deformation is accompanied by distributed microcracks [6,20] which, in human cortical bone, are present within both interstitial and osteonal lamellae [21,22]. Microcrack initiation has been linked to Haversian canals [21,23] and osteocyte lacunae [24,25]. Long microcracks (~100 μm), forming at later stages of cracking [26], interact with osteons [27] and the hyper-mineralized [28] cement lines [29,30]. "Bridges" at various length scales also hinder crack growth [11,31,32]. Despite these progresses, the nature of microcracking (distributed microcracks) at the lamellar, sub-lamellar, and fibrillar levels is still largely unknown [8,33,34].

Our recent study has shown the role of the osteonal lamellae in redistributing stress around each Haversian canal through the stable development of multiple intralamellar microcracks [8]. However, where those cracks initiated and how they developed within the bulk remained unclear [8,34]. Canaliculi are fine channels (~200 nm) [35] connecting the osteocyte lacunae together. Despite their high distribution density (1×10^6 canaliculi mm^{-3} [1]) and potential as stress concentrators [36], their roles in the microcracking process remain hypothetical [37,38]. A detailed study of bone's microcracking process would clarify the relations between microcracks and those fine structures.

The present microscopy study focused on the structure–microcracking relations at the lamellar and sub-lamellar levels of human cortical bone. The purpose was to investigate the morphology and the development of microcracks within the osteonal lamellar structure and with respect to the canaliculi network. This was carried out through imaging with a laser scanning confocal microscope following longitudinal and transverse compressive loading. The work is the first step to bridge the gap between

* Corresponding author. Address: University of British Columbia, Department of Materials Engineering, 309-6350 Stores Road, Vancouver, BC, Canada V6T 1Z4. Tel.: +1 604 822 9752; fax: +1 604 822 3619.
E-mail address: rzwang@mail.ubc.ca (R. Wang).

1742-7061/$ - see front matter © 2011 Acta Materialia Inc. Published by Elsevier Ltd. All rights reserved.
doi:10.1016/j.actbio.2011.11.013