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Research Paper

Continuum damage interactions between tension and compression in osteonal bone

*Mohammad J. Mirzaali, Alexander Bürki, Jakob Schwiedrzik[1], Philippe K. Zysset, Uwe Wolfram**

Institute for Surgical Technology and Biomechanics, University of Bern, Stauffacherstrasse 78, CH-3014 Bern, Switzerland

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ABSTRACT

Skeletal diseases such as osteoporosis impose a severe socio-economic burden to ageing societies. Decreasing mechanical competence causes a rise in bone fracture incidence and mortality especially after the age of 65 y. The mechanisms of how bone damage is accumulated under different loading modes and its impact on bone strength are unclear. We hypothesise that damage accumulated in one loading mode increases the fracture risk in another.

This study aimed at identifying continuum damage interactions between tensile and compressive loading modes. We propose and identify the material constants of a novel piecewise 1D constitutive model capable of describing the mechanical response of bone in combined tensile and compressive loading histories. We performed several sets of loading–reloading experiments to compute stiffness, plastic strains, and stress-strain curves.

For tensile overloading, a stiffness reduction (damage) of 60% at 0.65% accumulated plastic strain was detectable as stiffness reduction of 20% under compression. For compressive overloading, 60% damage at 0.75% plastic strain was detectable as a stiffness reduction of 50% in tension. Plastic strain at ultimate stress was the same in tension and compression. Compression showed softening and tension exponential hardening in the post-yield regime. The hardening behaviour in compression is unaffected by a previous overload in tension but the hardening behaviour in tension is affected by a previous overload in compression as tensile reloading strength is significantly reduced.

This paper demonstrates how damage accumulated under one loading mode affects the mechanical behaviour in another loading mode. To explain this and to illustrate a possible implementation we proposed a theoretical model. Including such loading mode dependent damage and plasticity behaviour in finite element models will help to improve fracture risk analysis of whole bones and bone implant structures.

*Corresponding author. Tel.: +41 31 631 59 59; fax: +41 31 631 59 60.
E-mail address: uwe.wolfram@istb.unibe.ch (U. Wolfram).
[1]Current address: EMPA, Swiss Federal Laboratories for Materials Science and Technology, Laboratory of Mechanics of Materials and Nanostructures, Switzerland.

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1. Introduction

Skeletal diseases such as osteoporosis expose a severe socio-economic burden to ageing societies (World Health Organization, 2003; Burge et al., 2007; Mithal et al., 2009). Osteoporosis is a progressive skeletal disease characterised by a loss of bone mass and impaired cellular repair mechanisms (Metcalfe, 2008; Teti, 2011). Both induce a loss of structural integrity and mechanical competence in whole bones. This decreasing mechanical competence causes a rise in bone fracture incidence and mortality (Marcus and Majumdar, 2001; Karlsson et al., 2005).

In case of the proximal femur, where the most detrimental fractures are observed, it is widely accepted that the load direction during a fall is distinct from the loading of everyday activities (Keyak, 2000). Regarding the elderly, even small loads in non-habitual load situations during daily activities such as collisions on the edge of a table, falling or simply lifting daily shopping off axis could damage the bone and, thus, increase the risk of fracture (Pinilla et al., 1996; US Department of Health, 2004). In fact, 90% of all hip fractures are related to falls and a third of all people over 65 years fall annually (Tinetti, 2003). It should be noted that every hip fracture is initiated by emerging microcracks (Fig. 1). The mechanisms how bone damage is accumulated under different loading modes and coupled into another loading mode and its impact on bone strength are unclear.

Pre-failure microdamage in bone is considered to be the most detrimental factor in defining its strength and toughness with respect to health and disease (Gupta and Zioupos, 2008). It accumulates as microcracks (Fig. 1) due to isolated, non-physiological overloading events in a quasi-static mode or after suffering a large number of physiological loading cycles in a fatigue mode (Schaffler et al., 1989; Moore and Gibson, 2003; Fleck and Eifler, 2003; Rapillard et al., 2006; Wolfram et al., 2011). Microdamage is not detectable using clinical imaging modalities but decreases bone's stiffness, strength, and toughness and eventually leads to collapse of whole bones (Fyhrie and Schaffler, 1994; Burr et al., 1997; Kopperdahl et al., 2000; Nalla et al., 2005; Ritchie et al., 2005; Ritchie, 2011).

Bone microdamage can be classified into microcracks and diffuse damage which are microcracks on a lower length scale (Fazzalari et al., 1998; Vashishth, 2007). Microcracks appear linear and spatially organised in 2D histological sections with a pertinent length of 10–70 μm (Reilly and Currey, 1999; Vashishth, 2007; Arlot et al., 2008; Zhang et al., 2010). In 3D, microcracks appear in approximately elliptical shape with an aspect ratio of 4:1 to 5:1 (Zioupos and Currey, 1994; O'Brien et al., 2000; Larrue et al., 2011). Their thickness is one to two orders of magnitude smaller than their length. Microcracks are associated and guided by micro-structural and ultra-structural features of bone (Rho et al., 1998; Jepsen et al., 1999; O'Brien et al., 2005, 2007). They appear at highly mineralised zones in bone tissue, between interstitial lamellae, along osteonal cement lines, at the boundaries of trabecular packages, at resorption cavities in trabecular bone, and, in case of sub-lamellar microcracking, along the canaliculi in cortical bone (Zioupos and Currey, 1994; Jepsen et al., 1999; O'Brien et al., 2005, 2007; Vashishth, 2007; Peterlik et al., 2006; Slyfield et al., 2012; Ebacher et al., 2012).

It was noted that microdamage is loading mode dependent as it appears to be different in bone regions loaded primarily in tension compared to regions loaded primarily in compression (Jepsen and Davy, 1997; Reilly and Currey, 1999). In histology studies, tensile microdamage appears to be more diffuse while compressive microdamage is rather expressed as microcrack (Boyce et al., 1998; Wang and Gupta, 2011) due to different yielding processes for tension and compression in bone (Reilly and Burstein, 1975; Currey, 2002). Microcracks open approximately normal to the loading direction under tension and show a cross-hatched pattern under compressive loading (Fig. 1; Chamay, 1970; Boyce et al., 1998). This loading mode dependent accumulation of microdamage eventually leads to the earlier noted difference in the post-elastic behaviour of cortical bone (Reilly and Burstein, 1975; Currey, 2002; Gupta and Zioupos, 2008; Nyman et al., 2009; Li et al., 2013).

Although important for understanding fragility of bones and bone-implant systems loaded along different modes, there is no substantial data on the mutual influence and mechanical consequences of bone microdamage accumulated in such different modes.

Continuum damage mechanics (CDM) is a well developed framework to describe the impact of emerging microcracks on the mechanical behaviour of a material. An extensive

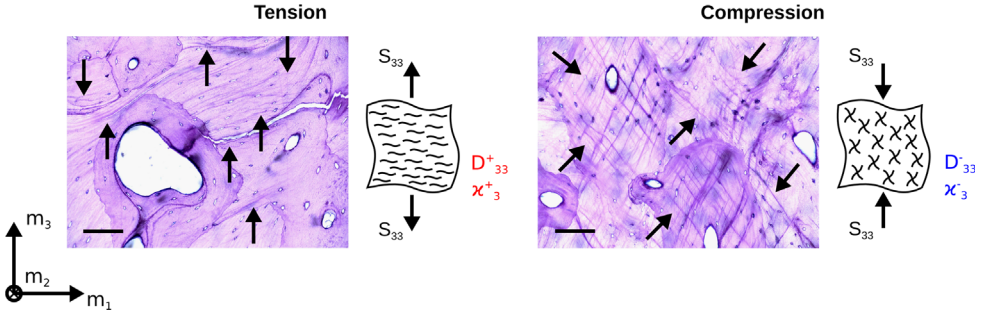

Fig. 1 – Basic Fuchsin stained, transverse histological sections of a sample overloaded in uniaxial tension (+) or uniaxial compression (−) are shown. Note, that small or perpendicular microcracks may close upon unloading (Sun et al., 2010) and are, thus, difficult to detect histologically. The images of the samples overloaded in tension are taken close to a macrocrack of which some effects are partially visible. The cracks in this modes were smaller and more diffuse than those found under compression. The scale bar is 0.1 mm long. (For interpretation of the references to color in this figure, the reader is referred to the web version of this paper.)