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Damage accumulation of bovine bone under variable amplitude loads ☆

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ABSTRACT

Stress fractures, a painful injury, are caused by excessive fatigue in bone. This study on damage accumulation in bone sought to determine if the Palmgren-Miner rule (PMR), a well-known linear damage accumulation hypothesis, is predictive of fatigue failure in bone. An electromagnetic shaker apparatus was constructed to conduct cyclic and variable amplitude tests on bovine bone specimens. Three distinct damage regimes were observed following fracture. Fractures due to a low cyclic amplitude loading appeared ductile ($4000\ \mu\epsilon$), brittle due to high cyclic amplitude loading ($>9000\ \mu\epsilon$), and a combination of ductile and brittle from mid-range cyclic amplitude loading ($6500-6750\ \mu\epsilon$). Brittle and ductile fracture mechanisms were isolated and mixed, in a controlled way, into variable amplitude loading tests. PMR predictions of cycles to failure consistently over-predicted fatigue life when mixing isolated fracture mechanisms. However, PMR was not proven ineffective when used with a single damage mechanism.

1. Introduction

This study considers cyclic constant and cyclic variable amplitude loading fatigue tests on bovine bone specimens, and it is motivated by the prevalence of stress fractures caused by repeated high-cycle loading. According to Verheyen et al. (2006), stress fractures occur in up to 21% of American military recruits and 45% of Israeli military recruits. Athletes such as dancers, figure skaters and gymnasts have a high incidence of stress fracture as well. However, cross country and track runners seem to experience stress fractures more often than other athletes. In a study conduct by Bennell et al. (1996), 21% of competitive runners acquired stress fractures in just a twelve month period. As we age, our bone's elastic modulus, strength and toughness decline. The elastic modulus, as well as the ultimate tensile strength, both decrease by 2% each decade between the ages of 20 and 90 years old (Mow and Hayes, 1997). These properties are extremely important to the fatigue behavior of bone, which is why the growth of microcracks is also an issue at older ages. This is especially a concern in post-menopausal, osteoporotic females. Humans are not the only mammals that acquire stress fractures; animals have a high incidence as well. Racing animals, such as horses and grey hounds, acquire stress fractures very frequently. The constant training and fatigue that human athletes, soldiers and animal athletes go through to excel at their activities is the main factor in acquiring stress fractures (Verheyen et al., 2006).

Stress fractures are microcracks within the bone that are caused from excessive fatigue. When the osteoblasts, cells that form or synthesize bone, cannot keep up with the microdamage that these continuous stresses create, the bone begins to experience fatigue. Bone fatigue is the cycle-by-cycle accumulation of damage from varying stresses and strains, so that immediate failure does not occur (Cui, 2002). The initial microcracks can eventually propagate and develop into a complete fracture in the bone. Stress fractures in humans are most commonly located in the tibia, tarsals, metatarsals and the femur (Patel et al., 2011; Sanderlin and Raspa, 2003). This type of injury is often very painful. Diagnosis is very difficult, and the best way to detect them is by plain radiography, magnetic resonance imaging, or triple-phase bone scintigraphy (Patel et al., 2011). Currently, the only way to treat a stress fracture is activity modification, analgesic drugs to relieve the pain, and pneumatic bracing to help with the healing process.

1.1. Constitutive fatigue model

When studying fatigue in bone, it is imperative to understand how bone breaks and what actually occurs as the fatigue damage accumulates. There is a strong consensus that bone acts like a fiber reinforced composite material when subjected to stresses and strains (Alto and Pope, 1979; Bell et al., 1999; Gibson et al., 1995; Guo et al., 1998; Martin et al., 1996; Moyle et al., 1978). Varvani-Farahani et al. compared the specific structures within bone and engineered

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composite (Varvani-Farahani and Najmi, 2010). In human cortical bone, osteons carry the brunt of the load and play the role of the reinforcing fiber within the interstitial bone tissue, which acts as the matrix. Additionally, the cement lines between the individual osteons act as the weak interfaces in fiber-reinforced composites. Bone and engineered composites display similar mechanical properties. Both are much stronger when loaded parallel to their fiber or osteonal structures. In bone, these structures run along the longitudinal axis, and are therefore stronger in the longitudinal direction.

When subjected to axial cyclic loading, bone undergoes three stages in the process leading up to, and resulting in, complete fracture (Currey, 1998; Gupta and Zioupos, 2008; Varvani-Farahani and Najmi, 2010). This is very similar to the process that composite materials undergo during characterization of fatigue damage under axial loading conditions. When loaded, both materials begin with an elastic region that strains linearly until it hits a yield point. A region of plastic flow follows this yield point. In this phase, large amounts of energy are absorbed due to a variety of toughening mechanisms. Finally, once the material cannot absorb any more energy, a catastrophic failure takes place.

1.1.1. Stage I

The first phase is a reversible deformation process prior to crack initiation that results in a very quick decline in stiffness due to the presence and initiation of microcracks in the interstitial bone, or matrix (Gupta and Zioupos, 2008; Schaffler et al., 1995; Varvani–Farahani and Najmi, 2010). Frost was the first to propose and report the idea of microcracks in 1960 (Frost, 1960). His theory was that microcracks signal and initiate their own healing due to osteocyte apoptosis. His concepts and findings were later confirmed by many studies (Bentolila et al., 1998; Mori and Burr, 1993; Verborgt et al., 2000). Researchers believe that even healthy bones contain microdamage. Burr stated that microcracks were a naturally occurring defense mechanism against extraneous forces applied to bones (Burr, 2011). Cracks are able to absorb and dissipate energy, lessening the amount of energy that the interstitial matrix and osteons have to absorb. Within healthy bone, microdamage is in equilibrium with the repair and remodeling system, osteoblasts and osteoclasts. These defects enable the bone to deform reversibly in the beginning phase until the bone hits a yield point.

1.1.2. Stage II

The slow process of stage II begins after the bone has hit this yield point. While the stiffness of the material continues to decrease, it is at a much slower rate than that of the elastic region. In this stage, the material still has its structural integrity, but is now permanently damaged (Gupta and Zioupos, 2008). The majority of this damage tends to occur at the cement lines (Varvani-Farahani and Najmi, 2010). Bone absorbs a great deal of energy in this phase and decreases in stiffness and residual strength. What is occurring during this time to cause this type of behavior, is the question that many researchers have spent their careers trying to pinpoint. It has been found that this particular behavior is primarily due to a combination of mechanisms.

Gupta and Zioupos presented two different methods in 2008 describing the failure of bone (Gupta and Zioupos, 2008). The first is the stress based method, which takes into account the stress intensity factor, K_c. This theory says that a crack is initiated when the stress at the crack tip reaches or exceeds the material's critical value. Peterlik et al. found that the fracture process depends on the direction in which the crack travels (Peterlik et al., 2006). The angle of the fibril microstructure, at the point damage originated, determines whether the crack propagates longitudinally, tangentially, or radially. Gupta and Zioupos also stated that the way in which cracks propagate through bone, and lead to complete failure, is directly influenced by the heterogeneity of the microstructure of the material (Gupta and Zioupos, 2008). The second method is an approach based on energy, including the critical strain energy release rate, and the work to fracture of a specimen. The required critical levels of energy per unit area to fracture the material are determined by these variables. Engineered composites, as well as bone, have very weak interlamellar surfaces with the ability and tendency to absorb energy and divert the main crack. This in turn slows down the start and propagation of cracks within bone. Burr stated that the ability for bone to absorb energy before failure is what determines the fracture resistance for the material (Burr, 2011).

A typical trend seen in fatigue testing is an increase in toughness as stiffness decreases. This phenomenon is precisely what is seen in this second phase of bone fatigue. Toughness is the amount of energy required to run a crack through a material. This observed toughening is due to bone's many mechanisms for deterring the propagation of cracks and damage. For this reason, Gupta and Zioupos stated that the initiation of a crack in the material is not nearly as significant as the propagation of the crack through the material (Gupta and Zioupos, 2008). Bone does not prevent the initiation of damage; it only delays the growth of fracture with its micro hierarchical structure, preventing cracks from growing to catastrophic size. The microstructure usually delays the growth of cracks long enough for the bone to repair itself. Our bodies have also developed mechanisms that attempt to prevent fracture. These toughening mechanisms increase the amount of energy that is required to grow the crack and fracture the material. Researchers have categorized the observed mechanisms into two groups, intrinsic and extrinsic (Kruzic and Ritchie, 2008; Launey et al., 2010; Nalla et al., 2003, 2005a,b; Ritchie et al., 2005). Intrinsic toughening mechanisms take place ahead of the crack tip. Extrinsic toughness develops from the applied stress behind the crack tip. Along with the crack diversion or deflection, other toughening mechanisms include microdamage, nucleation, crack bridging, sacrificial bonds, interfibrillar shearing, and fiber pull-out (Koester et al., 2008; Nalla et al., 2005b; Ritchie et al., 2009, 2005).

Microdamage is one of the main tools that bones use to defend against crack propagation. It dissipates energy, preventing and delaying catastrophic failure. Not only are microcracks found in healthy bone, but they have also been reported to develop after crack initiation, in an attempt to slow down and deter crack growth (Hansen et al., 2008). However, this mechanism also reduces the total amount of energy that can be absorbed by the bone overall. Microdamage causes the mechanical properties of bone to decline, which in turn results in fracture. The way in which the cracks are formed, the location they are in, the stimulus that caused them to form, and the way in which they are mended are all factors that are taken into account when determining the type of microcrack that is observed. There are two types of microcracks that occur in cortical bone (Burr, 2011). The first are linear microcracks. These occur most often and are typically found in the mineralized interstitial bone. These cracks are a clear indicator that the bone tissue has deformed before the main crack initiates and are due to compressive stresses. The second is diffuse microdamage, which is a collection of small cracks that are caused by tensile loads. When these cracks are close enough, they have the tendency to nucleate, coalesce and grow in size. Vashishth reported this in 1996 and found that these mechanisms stabilized the progression of fracture by absorbing energy and taking it away from the main crack (Hansma et al., 2005).

Ritchie et al. showed that another mechanism, crack bridging, increased fracture resistance (Kruzic and Ritchie, 2008; Launey et al., 2010; Nalla et al., 2003, 2005a,b; Ritchie et al., 2005). There are two forms of crack bridging. The first is collagen-fibril bridging. These by themselves do not play a large role in crack shielding. The second form is uncracked-ligament bridging which can sustain considerable loads.