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CONSTITUTIVE RELATIONSHIP OF TISSUE BEHAVIOR WITH DAMAGE ACCUMULATION OF HUMAN CORTICAL BONE

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Abstract

Microdamage accumulation has been identified as a major conduit for bone tissues to absorb fracture energy. Due to the poor understanding of its underlying mechanism, however, an adequate constitutive relationship between damage accumulation and the mechanical behavior of bone has not yet been established. In this study, the constitutive relationship between the damage accumulation induced by overload and the evolution of mechanical properties of bone with incremental deformation was established based on the experimental results obtained from a novel progressive loading protocol developed in our laboratory. First, a decayed exponential model was proposed to capture the damage accumulation (modulus loss) with increasing applied strain. Next, a power law function was proposed to represent the progression of plastic deformation with damage accumulation. Finally, a linear combination of the Kohlrausch-Williams-Watts (KWW) function and Debye function was used to depict the viscoelastic behavior of bone associated with damage accumulation. The results of this study may help develop a constitutive model for predicting the mechanical behavior of cortical bone tissues.

Keywords

Bone; Damage mechanics; Constitutive model; Viscous response; Plasticity

INTRODUCTION

The capacity to absorb energy (toughness) has often been used to evaluate bone quality. However, the constitutive relationship underlying such behaviors is still poorly understood. Previous studies have reported that microdamage accumulation is the major pathway of energy dissipation in bone (Courtney, et al., 1996, Jepsen and Davy, 1997, Kotha and

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Guzelsu, 2003, Martin, 2003, Zioupos and Casinos, 1998). McCalden et al. reported that aging could lead to a significant decrease in the plastic strain energy dissipation in bone (McCalden, et al., 1993). Similarly, we also found that the major difference between the elderly and younger tissues is the significantly reduced plastic deformation (Wang and Nyman, 2007). Moreover, evidence has shown that the viscous response of bone (hysteresis) increases with increasing post-yield deformation, suggesting an augmented participation of collagen in load bearing as damage accumulates in the tissue (Nyman, et al., 2005, Wang and Nyman, 2007, Yeni, et al., 2004). In fact, there are three distinct mechanical responses associated with the post-yielding of bone: (1) stiffness loss, (2) permanent deformation, and (3) hysteresis responses (Fondrk, et al., 1988, Pattin, et al., 1996).

In the past, continuum damage mechanics approaches have been used to develop the constitutive model for bone. Kachanov's damage model states that damage evolves as a power law function of the effective stress (Kachanov, 1986). Krajcinovic et al (Krajcinovic, et al., 1987) proposed a linear elastic damage model using a simple statistical mode of fiber pull-out. Based on the concept of effective stress/strain (Lemaitre, 1996), Fondrk et al. proposed a damage evolution model, representing both the modulus loss and viscoplastic (residual) deformation (Fondrk, et al., 1999). Moreover, Zysset et al and Curnier proposed a model based on a central assumption that plastic flow and damage accumulation are intrinsically related (Zysset and Curnier, 1996). These aforementioned models are all developed using assumed tissue behaviors and the material constants in these models are arbitrarily determined without direct physical meaning. Thus, lack of direct physical background is the major shortcoming of the models. To this end, this study was performed to explore the empirical constitutive relationship of bone in stiffness loss, plastic deformation, and viscous response during the post-yield deformation as a function of microdamage accumulation in the tissue.

EXPERIMENTAL OBSERVATIONS

To experimentally study the post-yield behavior of bone, we have developed a novel loading protocol (Wang and Nyman, 2007) that can examine the evolution of post-yield behavior of bone as a function of incremental strain. As shown in Figure 1, the loading protocol consists of a series of incremental, diagnostic loading cycles, each of which includes the following steps: (1) load the specimen in displacement control to a specified displacement level, (2) hold the displacement constant to determine the stress relaxation at the strain level, (3) unload in force control to zero force, (4) hold at zero force to determine the strain relaxation and residual strain (ϵ_{pi}), then (5) reload in displacement control to the next strain level with a constant increment. This procedure was repeated at a constant strain rate until failure of the bone specimen. This load-dwell-unload-dwell-reload protocol was designed to decouple the elastic, plastic, and viscous responses of bone while minimizing the effect of fatigue and viscoplastic creep during the test. At each applied strain level, the instantaneous elastic modulus (E_i) was determined as the slope of the line connecting the two equilibrium points at the ends of the two dwells in each cycle (i.e., the dotted line in Figure 1) to eliminate the hysteresis effect. The instantaneous stress (σ_{maxi}) was determined as the maximum stress, and the instantaneous strain (ϵ_i) is the maximum strain at each cycle. The instantaneous plastic (residual) strain (ϵ_{pi}) was determined at the end point of the strain relaxation dwell in each cycle. Moreover, the viscoelastic response of bone at each strain level was determined using the stress vs. time curve recorded during the stress relaxation dwell.

In this study, human cortical bone specimens were taken from the mid-diaphysis of tibiae of eight (8) middle-aged male donors (51.5 ± 3.3 years old). Cylindrical specimens (3mm in diameter and 5mm in length) were prepared for compression tests, whereas dog-bone specimens (2mm $\times$ 2mm $\times$ 10mm in gage region) were prepared for tensile tests. The axis of

all specimens was oriented along the longitudinal direction of bone. The deformation of the specimens was measured using extensometers and the force measured using the force transducer attached to the machine. The strain and stress were calculated by normalizing the gage deformation and force by the gage length and the gage cross-section area, respectively (Leng, et al., 2009, Nyman, et al., 2009a, Nyman, et al., 2008).

The results indicate that the instantaneous elastic modulus (E_i) of human cortical bone decreased exponentially with increasing strain in both tension and compression (Figure 2). In addition, the permanent strain (ϵ_{pi}) increased non-linearly with the increasing strain after yielding (Figure 3). Moreover, Figure 4 shows the typical stress relaxation vs. time curves at different strain levels during the period of relaxation dwells. For early cycles, the stress relaxation curves were almost like a straight line although the stress level increased. However, after the 4th cycle significant changes in the shape of stress relaxation curves could be observed, whereas the initial stress level gradually decreased with the applied strain. In summary, the post-yield behavior of bone with respect to a uniaxial loading can be characterized as follows: 1) an exponential decay of elastic modulus, 2) a non-linear increase of plastic deformation; and 3) a varying viscoelastic response.

EMPIRICAL CONSTITUTIVE RELATIONSHIPS

Elastic modulus loss vs. damage density

The previous experimental results indicate that the modulus loss starts at very low strain levels ($< 0.3\%$) and its relationship with the applied strain (ϵ_i) can be estimated using an exponential decay model (Figure 5) if a continuous modulus loss is assumed:

$$E_i = E_0 e^{-m\epsilon_i} \quad (\text{Eq. 1})$$

where E_0 is the initial elastic modulus, which can be approximately determined by extrapolation at zero strain, m is a material constant defining the acuteness of modulus loss. It is noted that E_0 ($\sim 22.1 \text{ GPa}$) is higher than the modulus value ($\sim 18 \text{ GPa}$) determined from the slope of initial linear elastic portion of stress-strain curves of the bone specimens (i.e., E_I in Figure 1).

Based on basic damage mechanics (Kachanov, 1986, Lemaitre, 1996), the microdamage density (D) can be simply defined as follows:

$$D = 1 - \frac{E_i}{E_0} \quad (\text{Eq. 2A})$$

where E_i is the instantaneous modulus and E_0 is the original modulus. Inserting Eq. 1 into Eq. 2A, the following relationship is obtained:

$$D = 1 - e^{-m\epsilon_i} \quad (\text{Eq. 2B})$$

D varies between 0 and 1. This equation suggests that the damage density is exponentially related to the applied strain (or degree of deformation).

Plastic deformation vs. damage accumulation

By plotting the plastic strain with respect to the microdamage density (D), the relationship of the plastic deformation with the instantaneous damage accumulation can be obtained (Figure 6). Limited plastic deformation was observed at the early stage of damage accumulation (D

< 0.2), followed by an accelerated increase in a nonlinear manner. We found that a power law function best fits the relationship:

$$\varepsilon_p = k \cdot D^n \quad (\text{Eq. 3})$$

where, both k and n are material constants, reflecting the sensitivity of the plastic deformation of bone to damage accumulation.

Viscous response vs. damage accumulation

Conventionally, an exponential relaxation function ($e^{-t/\tau}$, referred to as Debye model) is usually used to depict the relationship of stress relaxation with time (t), where τ is defined as the viscoelastic time constant. However, previous experimental results have showed that the viscoelastic behavior of bone can not be fully described by the Debye function, a linear combination of the Kohlrausch-Williams-Watts (KWW) function and the Debye function is needed (Sasaki and Enyo, 1995, Sasaki, et al., 1993):

$$\sigma = A_1 e^{-(t/\tau_1)^\beta} + A_2 e^{-t/\tau_2} + C \quad (\text{Eq. 4})$$

where, A_1 and A_2 represent the contribution to the total stress relaxation, and τ_1 and τ_2 are the time constants for the KWW and Debye processes, respectively, β is a material constant that determines the acuteness of the KWW stress relaxation, and C is a constant representing the stress contribution by the pure elastic deformation. By determining these material parameters at each strain level (cycle), their changes with respect to D were obtained (Figure 7). This model was selected because it not only fits well with our experimental data but also possesses physical meaning (Sasaki and Enyo, 1995, Sasaki, et al., 1993). The mechanism of the KWW relaxation process is believed to relate to the ultrastructural disorder of collagen fibrils (Sasaki, et al., 1993), whereas the Debye process is more associated with the microstructural (e.g., fluid flow in pores) and inelastic (e.g., creep) behavior of bone (Sasaki and Enyo, 1995).

By plotting A_1 and A_2 with respect to D (Figure 7), it was found that the magnitudes of the KWW (A_1) and Debye (A_2) functions were both negligible when $D < 0.3$, but increased afterward. However, the magnitude of the Debye stress relaxation (A_2) was increased less significantly than that of KWW (A_1), suggesting a much less contribution of the Debye process to the total relaxation process. It is presumable that the KWW process is dominant in bone viscoelastic behavior under the test condition of this study. Nonetheless, it should be pointed out that the Debye process may not be ignored for long term relaxation tests (Sasaki and Enyo, 1995).

Next, a bilinear relationship was observed between the time constants (τ_1 and τ_2) and the damage accumulation (D) for both the KWW and Debye processes (Figures 8), showing a sharp drop at the early stage of damage accumulation and then retaining either a much gradual declination (KWW) or a relatively consistent value (Debye). Such relationships can be expressed as:

$$\tau_1 = \begin{cases} B_1(D_{th} - D) + \tau_1^0 & D \leq D_{th} \\ \tau_1^0 - B_3(D - D_{th}) & D > D_{th} \end{cases} \quad (\text{Eq. 5A})$$

for the KWW process, and

$$\tau_2 = \begin{cases} B_2(D_{th} - D) + \tau_2^0 & D \leq D_{th} \\ \tau_2^0 & D > D_{th} \end{cases} \quad (\text{Eq. 5B})$$

for the Debye process. Where B_1 , B_2 , and B_3 are material constants determining the changing slopes, and τ_1^0 and τ_2^0 are the time constants, respectively. D_{th} is a threshold value at the transition of the bilinear behavior. It is noteworthy that the time constant for KWW process (τ_1) was much greater than that for Debye process (τ_2), again suggesting that the KWW relaxation dominates the viscoelastic behavior of bone.

Similarly, a bilinear relationship was also found between β and D (Figure 8). Following an initial quick decrease, the change of β decelerated significantly after D passed the threshold value (D_{th}). However, it is noted that β is defined within the range of $0 \leq \beta \leq 1$ for the KWW function (Sasaki and Enyo, 1995, Sasaki, et al., 1993). The value of β calculated from our experimental data fell in the range only after $D > D_{th}$, thus suggesting that the viscoelastic behavior of bone may not be captured using the KWW model when $D < D_{th}$. Thus, the change of β with D can be expressed using the following equation only in the range of $D \geq D_{th}$.

$$\beta = \beta_{th} - S(D - D_{th}) \quad D \geq D_{th} \quad (\text{Eq. 6})$$

where, β_{th} is a threshold value at $D = D_{th}$, S defines the slope of the curve.

DISCUSSION

By analyzing the experimental results obtained using the novel progressive loading technique, we have gained new insight into the effect of the overload induced damage accumulation on the mechanical behavior of cortical bone subjected to uniaxial loading. Similar to fatigue-induced damage accumulation (Burr, et al., 1998, Leng, et al., 2009, Nyman, et al., 2009b, Yeni, et al., 2007, Zioupos, et al., 2001), the overload-induced damage accumulation lead to significant degradation of mechanical properties of bone, such as stiffness, strength, toughness, and viscous response (Burr, et al., 1998, Leng, et al., 2009, Nyman, et al., 2009b, Yeni, et al., 2007, Zioupos, et al., 2001). Suitable formulae that describe the constitutive relationship between damage accumulation and these mechanical properties may assist to develop comprehensive constitutive laws for cortical bone tissues.

First, our results indicate that the exponential decay model (Eq. 1) could accurately describe the modulus degradation as a function of the applied strain. Since the modulus loss is correlated with microdamage accumulation (Burr, et al., 1998, Zioupos, et al., 1994), the exponent (m) in the model (Eq. 1) actually reflects the sensitivity of bone to damage accumulation. That is, a greater value of m means quicker damage accumulation and faster stiffness loss.

However, it is noteworthy that the initial modulus ($E_0 = \sim 22.1 \text{ GPa}$) predicted by the model is greater than the value ($\sim 18 \text{ GPa}$) measured using the slope of the initial linear portion of the stress-strain curve of this study and others (Reilly and Burstein, 1975, Rho, et al., 1998, Sobelman, et al., 2004). This is not surprising because former is predicted by extrapolation of the modulus degradation curve at zero strain ($\varepsilon = 0$), whereas the latter is measured during the loading process ($\varepsilon > 0.2\%$ in this study). Thus, it is possible that damage might be induced during the loading process. In addition, bone may contain pre-existing microdamages induced by daily activities, thereby causing a decrease in the initial modulus (Figure 2). In fact, the initial modulus predicted in this study is comparable to the mean

value (~23.5GPa) acquired from nanoindentation tests (Turner, et al., 1999), which are not sensitive to the pre-existing microdamage.

Another finding of this study is that the significant plastic deformation occurs only after considerable damage accumulation has occurred ($D > 0.2$), suggesting that the early-stage damage accumulation induces little plastic deformation to bone. Since considerable permanent deformation is unlikely to occur within the mineral and collagen phases, it may be induced only through the relative deformation between the two phases. Among all possible mechanisms, the interfacial rupture/sliding between the mineral and collagen phases may be a major source for the plastic deformation of bone in the later stage of deformation. Similar speculation is also proposed by some studies by investigating the internal strains of collagen fibrils in the loaded bone specimens (Gupta, et al., 2006).

In the power law equation (Eq. 3) representing the relationship between the plastic deformation and damage accumulation, the material constant k determines the extent of the plastic deformation, whereas the exponent n governs the shape of the plastic strain vs. damage accumulation curve. Since the plastic deformation is a function of D (Eq. 3) that is in turn determined by the exponent, m (Eq. 2B), a greater m may result in an earlier inception of the acute increase of plastic deformation. This argument can be verified using the experimental results reported in the other studies (Leng, et al., 2009).

The results of this study indicate that the stress relaxation becomes significant only when considerable damage accumulates in bone ($D > 0.3$) and most of it is attributed to the KWW relaxation process. However, Sasaki et al. reported that the KWW process contributes to 20–40% of the total stress relaxation for bovine bone samples (Sasaki and Enyo, 1995, Sasaki, et al., 1993). Two major reasons are deemed for the discrepancy. One is that only 0.5% strain was applied to the bone specimens in the Sasaki's experiments, which is still in the range of quasi-elastic deformation, whereas the bone specimens of this study were loaded to much greater strains (3~5%). In the former case, the involvement of the collagen phase (KWW behavior) in the load bearing of bone is still restricted by the mineral phase, thus longer time is needed to catch the relaxation characteristics by the Debye process. For the latter case, however, large damage accumulation led to significant viscoelastic responses by the KWW process. The other reason is that Sasaki's experiments lasted up to 100,000 seconds, while this study took only 120 seconds for stress relaxation. Since only short-term data of stress relaxation were produced in this study, they could not fully capture the characteristics of the Debye function.

Additionally, it was observed that the magnitude of A_1 and A_2 increased only when $D > 0.2$, suggesting that the contribution of both KWW and Debye functions to the stress relaxation is associated with marked damage accumulation. Also observed was that when $D < 0.3$ the values of β were greater than unity ($\beta > 1$), which are actually placed outside its valid range ($0 \leq \beta \leq 1$). All the observations suggest that under the condition of this study the stress relaxation occurs and is dominated by the KWW function only after marked damage accumulation takes place.

There are several limitations for this study. One limitation is that the time-dependent mechanical behavior of bone was not considered in this study, such as loading rate dependence (Courtney, et al., 1994, Gilbert and Merkhani, 2004) in addition to cyclic fatigue and viscoplastic creep. These topics actually are the future focus of our investigation. Another limitation is that this study only tested longitudinal mechanical properties of bone. Since bone is transversely isotropic, the orientation dependence of bone mechanical properties is expected. To address this issue, further investigation is required. Finally, it is hard to define the final failure of bone in compression since bone specimens lose their load

bearing capacity very gradually (Leng, et al., 2009), as encountered by other researchers (Reilly and Burstein, 1975). In contrast, failure in tension can be easily defined (Nyman, et al., 2009b). Although the post-yield behavior of bone is similar in compression and tension, its failure mechanisms is a little different between the two loading modes. This topic needs further investigation in the future.

SUMMARY

Based on the above results, the following mechanism of the post-yield behavior of bone can be speculated: At the early stage of deformation, microdamage accumulation induced by overload causes local ruptures of the mineral phase and collagen phase, thus leading to a rapid degradation of modulus. Meanwhile, the collagen phase that is exposed by the microdamages starts to participate in the load bearing process, thereby resulting in a sharp change of the viscous response of bone. However, the plastic deformation is still negligible at this stage. As the microdamage accumulation passes a threshold value (D_{th}), both plastic deformation and viscous responses become significant. In this case, the interfacial sliding between the mineral and collagen may serve as the major mechanism of plastic deformation, whereas the viscous interfacial friction (or deformation) may take place when the mineral-collagen interface begins to surrender under the load.

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Figure 1.

The progressive loading protocol including a series of diagnostic cycles of load-dwell-unload-dwell-reload was used in the compressive and tensile tests. E_I is the elastic modulus measured at the initial linear portion of the stress-strain curve. E_t is the instantaneous elastic modulus measured as the slope of the line connecting the ends of stress and strain relaxation dwells, ϵ_{ip} is the plastic strain measured at the end of the strain relaxation dwell, and ϵ_I is the maximum strain (instantaneous strain) applied in each diagnostic cycle.

Figure 2. Modulus loss as the function of applied strain. The elastic modulus decreased with the applied strain in the same trend irrespective of loading modes (*i.e.* compression and tension).

Figure 3.

Evolution of the permanent strain with respect to applied strain. Very limited plastic strain occurred in the early stage of damage accumulation ($D < 0.2$), and then increased in an accelerated and nonlinear manner.

Figure 4.

Typical stress relaxation vs. time curves at different strain levels (loading cycles) during the period of relaxation dwells. Little stress relaxation was observed at small strain levels, while prominent stress relaxation behavior became obvious after the 4th cycle.

Figure 5. Exponential decay model of modulus loss of bone with respect to applied strain. The model fits well with the experimental data.

Figure 6. Power law relationship between the plastic strain and the damage density in bone obtained by curve fitting of the experimental data.

Figure 7.
The contribution of the KWW process (A_1 -squares) and the Debye process (A_2 -circles) to the stress relaxation of bone. The KWW behavior appears to dominate the process.

Figure 8. Relationship of the viscoelastic parameters (τ_1 , τ_2 and β) with the damage density (D) in bone. They were estimated by curve fitting of the linear combination of the KWW and the Debye functions. All of them showed a bilinear relationship with D .