Model of flexural fatigue damage accumulation for cortical bone

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Summary: Analytical models that predict modulus degradation in cortical bone subjected to uniaxial fatigue loading in tension and compression are presented. On the basis of experimental observations, damage was modeled as self-limiting for tension but not for compression. These mechanistic uniaxial damage models were then developed into a model for flexural fatigue of cortical bone based on laminated beam theory. The unknown coefficients in the uniaxial damage models were obtained by successfully fitting the resulting equations to uniaxial fatigue data from the literature on human cortical bone in tension and compression. Then the predictions of the flexural model for the behavior of human cortical bone were compared with experimental results from a small but independent set of specimens tested at three different ranges of load in our laboratory. The behavior of the modulus degradation curves and the flexural fatigue lives of the specimens were in excellent agreement with the predictions of the model.

The mechanisms for fatigue damage accumulation in cortical bone are of interest to researchers and clinicians who wish to determine the causes of, and preventive measures for repetitive stress injuries. Cyclic loading of cortical bone produces observable damage in the form of microcracks, osteon debonds, and pulled-out osteons, which are thought to degrade mechanical integrity (2-8,12-15, 20). The literature dealing with the fatigue of engineering composites and other structural materials is extensive; however, the fatigue processes of bone are not as well understood.

Previous studies of cortical bone have demonstrated that its fatigue behavior in tension is different from that in compression (2, 3, 17). Fatigue lives are longer in uniaxial compression than in tension, and the shapes of the modulus degradation curves are different. In tension, there are generally three distinct regions of behavior with increasing numbers of cycles. The first is an initial reduction in modulus, the second is a plateau and the third is a rapid reduction in modulus before failure. In compression, on the other hand the modulus diminishes very slowly at first but then at an ever increasing rate until failure.

Carter and Hayes (2) also observed differences in the appearance of microdamage associated with fatigue in tension and compression. In tension, most cracks were found in the interstitial matrix or in osteonal cement lines. In compression, the cracks tended to run from one vascular channel to another. We have seen the same behavior in the tensile and compressive regions of specimens of human femur fatigued in four-point bending (Fig. 1). Although it seems reasonable to attribute the differences in modulus degradation in tension and compression to differences in the nature of the damage, there has not been a theory that supports this hypothesis, to our knowledge.

Our efforts to interpret the results of our flexural experiments (6) led us to ask how bone's flexural fatigue behavior is related to its uniaxial fatigue behavior. We postulated that a mechanistic model could be devised to explain the different modulus degradation curves for tension and compression on the basis of the different kinds of cracking that occur in these two modes of loading. Assuming that the damage in four-point bending experiments is predominantly due to axial stresses, we also conjectured that flexural fatigue behavior could be predicted from uniaxial data with use of a damaged-beam theory.

THEORY

We assumed that cortical bone could be modeled as a fiber-matrix composite. The fibers were considered complete osteons, and the matrix was the interstitial bone, composed of primary bone and remodeled osteon fragments. We also assumed that damage could be partitioned into components that independently affect the fibers and the matrix and the "damaged modulus" could be obtained from an isostrain rule of mixtures similar to that used in modeling ceramic matrix composites (18) [Figure 1]

$$E=(1-D_f)E_fV_f+(1-D_{,,})E_{,,}V_{,,} \quad (1)$$

Here, E is the elastic modulus, V is the volume fraction, and D represents damage. The subscripts m and f refer to matrix and fibers (intact osteons), respectively. D_1 and $D_{,,}$ have values that range from 0, in an undamaged state, to 1, in a failed state.

For ceramic matrix composites loaded in tension, the initial damage is caused by a system of micro-cracks in the matrix (11,21). The number of these cracks does not increase indefinitely but approaches a limit known as the saturation damage state (9,21). Similarly, in zero-tension fatigue loading of cortical bone — on the basis of the observations of Carter and Hayes (2) and our own observations in tensile regions of flexural specimens — we postulate that the loss of stiffness is caused primarily by cracks that form in the interstitial matrix (Fig. 1). We believe these cracks are self-limiting in length because they consistently appear as seen in Fig. 1A, Therefore, we suggest that damage to the matrix evolves as

$$\frac{dD_m}{dN} = K_1(D_s - D_m) \quad (2)$$

Here, D_m , represents the matrix damage, N is the cycle number. K_1 is the parameter of the rate of matrix damage, and $D_s = K_2 \sigma_a^q$ represents the saturation level of matrix damage, with K , and q being constants. The saturation damage is assumed to be a function of the applied stress amplitude.

Most matrix cracks intercept osteon cement lines (19) and can be expected to produce stress concentrations at the point of impingement. Matrix damage also results in load transfer from the matrix to the osteons. These factors should eventually lead to the tensile failure of the osteons. As the osteons break, the load is further redistributed to neighboring osteons, producing positive feedback and resulting in osteon failure at an ever increasing rate. Therefore, the fiber damage, D_f is assumed to accumulate as

$$\frac{dD_f}{dN} = (K_3 D_f + K_4 D_m) \quad (3)$$

where K_3 is a coefficient of the rate of osteonal damage, and K_4 is a parameter of the damage due to interaction of the osteon and matrix. Equations 2 and 3 can be solved simultaneously in closed form to obtain

$$D_m = K_2 \sigma_a^q (1 - \exp [-K_1(N/N_f)]) \quad (4)$$

$$D_f = \frac{K_2 K_4 \sigma_a^q}{K_1 + K_3} \left\{ \left[\frac{K_3 \exp(-K_1(N/N_f))}{K_1 + K_3 \exp(K_3(N/N_f))} \right] - (K_1 + K_3) \right\} \quad (5)$$

Where N_f is the fatigue life in tension. Here, the life fraction, N/N_f is the variable of integration rather than N .

Microcracks appear less likely to stop at the osteonmatrix interfaces in compression than they do in tensile loading (2) (Fig.1). We postulate, therefore, that cracks in bone fatigued in compression tend to grow in length and number without bound degrading the material in a positive feedback manner. We consequently model the damage in compression, D_c , to evolve as

$$\frac{dD_c}{dN} = K_5(D_{sr} - D_c) \quad (6)$$

Here, $1 < p$ is a parameter of the rate of compression damage and D_c represents incipient damage in the form of stress concentrator, such as Haversian or Volkmann's canals, which initiate or extend D_c . Assuming that $D_c = K_6 \sigma_d^p (N/N_{fc})^{K_5} - 1$, where K_6 and p are constants. Eq. 6 is readily solved to obtain

$$D_c = K_6 \sigma_d^p (\exp [K_5(N/N_{fc})] - 1) \quad (7)$$

where N_A is the fatigue life in compression. For compression, D_c is used for both damage terms in Eq. 1 because there do not appear to be distinct damage modes in osteons and interstitial matrix in this mode of loading.

The flexural fatigue model was developed on the basis of laminated beam theory (1). It is important to note that the plies of the model do not represent lamellae in the bone but rather regions of approximately homogeneous stresses, for computational purposes. The laminated beam theory assumes perfect bonding of the plies, which ensures that compatibility of the strains is not violated. The constitutive equations assume perfect bonding of the plies, which ensures that compatibility of the strains is not violated. The constitutive equations for the laminated beam theory are

$$T = A\varepsilon_o + B\kappa \quad (8a)$$

$$M = B\varepsilon_o + C\kappa \quad (8b)$$

where T is the axial force in the beam, M is the moment, ε_o is the midplane strain, and κ is the beam curvature, $(1/l^2) d^2x/dx^2$, where x is the beam deflection and l is position along its length. The constants A , B , and C in Eq. 8a and b are functions of the ply geometry and elastic properties:

$$\alpha = \sum_{k=1}^n b E_k (h_k - h_{k-1}) \quad (9a)$$

$$\beta = \frac{1}{2} \sum_{k=1}^n b E_k (h_k^2 - h_{k-1}^2) \quad (9b)$$

$$\gamma = \frac{1}{3} \sum_{k=1}^n b E_k (h_k^3 - h_{k-1}^3) \quad (9c)$$

In Eq. 9a-c. h is the beam width. E_A is the elastic modulus of the k th ply (assuming isotropic behavior), and h_A is the distance to the ply from the geometric midplane of the beam (Fig. 2). Assuming that the beam is simply supported, the normal force, T , in Eq. 8a is 0. The differential equation for the deflection, δ , is then obtained by solving for E , and i with

$$\frac{d^2 \delta}{dx^2} = \frac{M}{\gamma - (\beta^2/\alpha)} \quad (10)$$

A numerical integration technique developed for beams with a variable flexural rigidity along their length was used to solve the beam equations for deflection (16). The beam is discretized into 14 variable length segments, each composed of 20 plies of equal thickness (Fig. 2). For four-point bending with symmetrically placed supports and a center span of $L/2$, the applied load for the beam as calculated with beam theory is

$$P = \frac{2E_{bn} b h^2 \epsilon_{\max}}{3L} \quad (11)$$

Here, $\epsilon_{\max}$, is the desired peak flexural strain, and b , h , and L are the width, depth, and supported length of the beam, respectively. [Figure 2]

Once the load, P , has been obtained, the moments are calculated at the ends and midpoint of each segment. Before Eq. 10 can be solved, the flexural rigidity — $[EI]$ of each segment must be obtained by solving Eq. 9a-c. Initially, every ply within each segment is undamaged. with a bone composite elastic modulus value. E_c , given by the isostrain rule of mixtures:

The stresses for each ply of the beam segments are calculated with Hooke's law:

$$\sigma(y) = E_{bn} \left[\varepsilon_o + y \left(\frac{Ma}{\gamma\alpha - \beta^2} \right) \right] \quad (13)$$

where y is the distance from the geometric midplane of the beam to the center of the ply (Fig. 2A). The ply stresses for the current cycle are used to determine properties of the plies for the subsequent cycle from the appropriate damage model (tension or compression). To minimize computation, the symmetry of the beam is exploited.

Once the flexural rigidity is calculated for each segment, Eq. 10 can be integrated twice to obtain the values for beam deflection and slope. The first integration uses the trapezoidal rule to obtain the slope of the i th segment:

The second integration uses Simpson's rule, because the slope is a piecewise quadratic function, yielding

which requires that there be three integration points for each segment (Fig. 2). The flexural rigidity of adjacent segments will vary, therefore, two values of flexural rigidity are calculated at each integration point to ensure that the entire beam is integrated. The equation for the deflection, δ , of the beam as a function of longitudinal position, x , is then where C_1 and C_2 are the integration constants. At $x =$

$$E_{bn} = E_f V_f + E_m V_m \quad (12)$$

$$\varphi_i = \varphi_{i-2} + \left\{ \left[\frac{(M_{i-1}/(\gamma - \beta^2/\alpha)_{i-1})}{+ (M_{i-2}/(\gamma - \beta^2/\alpha)_{i-2})} \right] / 2 \right\} (x_i - x_{i-1}) \quad (14)$$

$$\delta_i(x) = \Psi_i + C_1 x + C_2 \quad (16)$$

$$\Psi_{i+4} = \Psi_i + \frac{1}{6} (\varphi_{i+4} + 4\varphi_{i+2} + \varphi_i) (x_{i+4} - x_i) \quad (15)$$

and $x = L$, the displacements are 0. This provides the boundary conditions required to solve for C_1 and C_2 . Because $\delta(0) = \delta(L) = 0$ and $\delta'(0) = 0$, it is apparent from Eq. 16 that $C_2 = 0$ and $C_1 = -\frac{P(L)IL}{EI}$. [Table 1]

The beam is "loaded" numerically one cycle at a time until failure. Failure of an individual ply in tension or compression is modeled by setting the elastic modulus of that ply equal to 0 (1). Beam failure was assumed to occur when the normalized beam modulus reached a value of 0.35. This value was arbitrary but practical because one to two cycles later the beam's deflections were far beyond the limits of beam theory.

To test the model, we obtained values for its parameters from experimental results in the literature, predicted the flexural fatigue behavior of human femoral bone, and compared these predictions with the results of our own independent experiments. We also studied the sensitivity of the model's predictions to variations in its parameters.

The fiber volume fraction (V_f) was somewhat arbitrarily assumed to be 0.65. Kerley (10) measured the volume fraction of primary bone in 50-year-old human femurs to be about 0.15. If 0.20 of the volume fraction were osteon fragments, then intact osteons would comprise the remaining 0.65. Using this value in Eq. 12 with $V_{f,0} = 1 - V_f$, $E_{f,0} = 16$ GPa, and E_f equal to the mean initial flexural modulus from our fatigue testing of human beams, we obtained $E_r = 14$ GPa.

The number of cycles to ply failure was estimated from a power-law fit of the data of Pattin et al. on uniaxial tension and compression fatigue for human femoral tissue (17). The resulting expressions are similar to Basquin's law:

$$\text{Tension: } N_{ft} = 0.5 \left(\frac{\sigma_a}{118.96} \right)^{-18.2} \quad (17a)$$

$$\text{Compression: } N_{fc} = 0.5 \left(\frac{\sigma_a}{217.06} \right)^{-10.6} \quad (17b)$$

Again, σ_a , is the stress amplitude in megapascals, which is simply the peak value of stress in a zero-tension or zero-compression fatigue test conducted under load control. To obtain values for the remaining parameters in Eqs. 4, 5, and 7, these equations were fitted to the curves for modulus reduction versus cycle number of Pattin et al. with use of the Newton computation option of the nonlinear regression procedure in SAS (version 6.09: SAS Institute, Cary, NC, U.S.A.) (16,17). Ten tensile and six compression fatigue specimens were used for this procedure. [Figure 3] [Table 2]

A Plackett-Burman analysis of variance technique was used in the parametric sensitivity test (7,22). In general, the model's parameters, chosen as described above, were systematically increased by 20% or to the upper 95% confidence limit from the regression, whichever was smaller. V_f was increased to 0.80, and E_f and $E_{,,}$, were increased to 16.5 and 20 GPa, respectively.

EXPERIMENTAL METHODS

Nine beams with nominal dimensions of 4 X 10 x 100 mm were machined from the mid-diaphysis of six human femurs obtained from unembalmed adult cadavers and stored frozen at -4°C . The gender and ages of these highly remodeled specimens were unknown, but the means and SDs of their porosity, initial elastic modulus, and basic fuchsin-stained microcrack density were $6.9 \pm 1.5\%$, 15.9 ± 1.4 GPa, and $2.37 \pm 1.95/\text{mm}^2$, respectively. The beams were tested to fatigue failure in four-point bending in accordance with American Society for Testing and Materials Standard D790 and a protocol developed in our laboratory (6). The tests were done in load control with the specimens submerged in saline solution

at 37°C and the periosteal side of the beam in tension. Roller-bearing supports with a diameter of 9,53 mm were symmetrically spaced 32 and 64 mm apart, and the load profile was a 2sine wave. Tests were done with ranges of load that initially produced three different initial peak strains (n = 3 in each case): 6,000, 6,500, and 7,500 microstrain. In all cases, the low end of the load range was 10 N. Specimens from the medial, lateral, and anterior cortices were randomly distributed among the experimental groups.

RESULTS

The damage model coefficients obtained by regression from the uniaxial fatigue data of Pattin et al. (17) (Table 1) were used in the models of tension and compression fatigue damage to calculate modulus reduction versus N for various applied stress amplitudes. The fits to the experimental data were $R^2 = 0.86$ for zero-tension fatigue (Fig. 3) and $R^2 = 0.97$ for zero-compression fatigue (Fig. 4). These correlations suggest that most of the variability of the data of Pattin et al. can be explained by the proposed mechanistic models of tension and compression fatigue (Eqs. 4, 5, and 7).

More importantly, the model's predictions for beams fatigued in four-point bending under load control also show good agreement with our independently obtained experimental data (Table 2 and Fig. 5). The predictions with regard to changes in modulus as a function of cycle number fall within the range of data for each of the three ranges of load. For two of the three initial ranges of strain, the mean fatigue life was less than 1/10th of a SII from the predicted value.

The sensitivity analysis of the model's parameters showed that fatigue life was sensitive to changes in E , and EI (p values are shown in Table 1): the stiffer the bone components, the shorter the fatigue life. The prescribed increases in K_2 , K_4 , and IC , all increased fatigue life.

Fatigue life also increased as the exponent q increased. but it decreased as p increased.

Changing the fiber volume fraction, V_p , on the other hand, did not significantly affect fatigue life. [Figure 4] [Figure 5]

DISCUSSION

On the basis of the types of cracking that occur in uniaxial tension and compression. We proposed a mechanistic model that successfully fits the different modulus degradation curves reported by Pattin et al. (17) for uniaxial tensile and compressive fatigue of human femoral bone. We also proposed that flexural fatigue behavior could be predicted by assuming uniaxial failure mechanisms and using a laminated beam model. We obtained strong support for this when we compared the predictions of the beam model, calibrated to the data of Pattin et al. (17), with our own independently obtained data for flexural fatigue.

The work presented here has several limitations. First, the model does not take into account damage resulting from shear stresses. This may become problematic if the model is extended to three-point or cantilever bending, in which the beam is subject to direct shear. Also, the model is essentially elastic and attributes all reductions in modulus to cracking, debonding, or fiber breakage. However, cortical bone has significant anelastic damage mechanisms (5.12). Although such nonlinearity is somewhat accounted for by using the secant modulus to describe the evolution of damage, this treatment is not rigorous.

Our data on flexural fatigue are limited to nine specimens, and the 6,000 and 6500 microstrain groups each (inadvertently) contained two specimens from the same femur. There are some differences in the methods by which the data for bending, tensile, and

compressive Fatigue were obtained, e.g., in our tests the beam was immersed in a saline solution, whereas Pattin et al. used a water drip (17). Furthermore, the data for uniaxial fatigue were obtained for initial strains well below the initial range of 6000-7500 microstrain used in the flexural fatigue experiments. This may be why the 7,500 microstrain curve appears to over-predict the modulus reduction that occurs early in fatigue life. Another limitation is that we made no attempt to account for possible regional variation of fatigue behavior of the bone, partly because the data of Pattin et al. did not include this information. We plan to study regional variability in future experiments.

The parallels we have drawn between damage modes in fiber-matrix composites and osteonal bone remain to be substantiated. In particular, the self-limiting nature of damage under tensile loading and the existence of a "saturation damage state" in bone need to be critically examined. We also plan to experimentally determine the $\epsilon_{f,cr}$, E_f , and V_f in future experiments.

It is noteworthy that the analysis is not inherently wedded to a fiber-matrix or osteonal structure and could apply as well to alternating layers of tough and brittle materials. The discretization of the beam into plies was for computational purposes, as is routinely done for continuous media analysis such as finite differences or finite elements. This method is commonly used to model failure of composite systems (19,21). Discretization of the beam into plies produces an artificial "ratcheting" of the beam's stiffness versus cycle curve; this artifact is reduced by increasing the number of plies. Our model was partially based on that of Ramakrishnan and Jayaraman (18), but their model did not contain a mechanistic basis for the differing rates of damage accumulation in fibers and matrix.

Our relatively simple modeling method was chosen to study the behavior of machined fatigue specimens. It could readily be modified to study other loading modes, such as bending superimposed on compression or reversed bending. The bending fatigue of a long bone that is reasonably straight could also be simulated by modifying Eq. 9a-c to account for varying ply width. However, if the bone has substantial curvature or is thick in comparison with its length, the assumptions of beam theory are no longer valid and other methods of analysis will be required.

The model exhibits increased fatigue life when the elastic moduli of the matrix, and especially the fibers, are reduced. This is in concert with our observation that the dorsal region of the equine third metacarpal bone has *a* significantly longer fatigue life but is more compliant than the lateral region (6). This behavior is not surprising when one considers that, under load control, more compliant specimens require less load to reach a prescribed initial strain value. Thus, the stresses and strain energy to which compliant specimens are subjected during the fatigue test are less than those for stiff specimens, and the propagation of damage is presumably reduced in similar measure. This may apply *in vivo* because the weight of the subject serves as "load control."

Finally, this model can be extended to include other modes of loading, particularly **if** data can be obtained on the effects of shear damage. Other fatigue phenomena can also be modeled, including modulus reduction and residual strength after sequential cyclic loading, with use of an arbitrary sequence of load ranges.

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FIG. I. Microcracks observed in human femoral bone fatigued to Failure and *en bloc* stained in basic fuchsin. A: Interstitial damage characteristic of the tensile side of a flexural specimen. The cracks do not usually penetrate osteonal cement lines. B: On the compression side, cracks typically travel across cement lines from one haversian canal to another.

FIG. 2. A: The geometric configuration of the laminated beam model. (Adapted from *Analysis and Performance of Fiber Composites*. 2nd ed. Agarwal BD, Broutman LI, p 149. Copyright 1990. Reprinted by permission of John Wiley & Sons, Inc., New York, NY, U.S.A.) B: Two representative 20-ply segments are shown. Segmental properties are calculated at the segment center. h = height of the beam, and a = length of each segment. C: Two segments are again depicted. The segment integration points required for the numerical solution technique are shown for the left segment.

FIG. 3. Examples of the model fit of tensile fatigue data from Pa itin et al. (17). The initial strain for the model approximately matches that of the data: data for 3,224. 3.547.4144 and 4.942 microstrain and predictions for 3,200. 3.550, 4.100. and 4,900 microstrain are shown.

FIG. 4. Examples of the model fit of compressive fatigue data from Pattin et al. (17). Data for 4.078.4,573.6.110.6.089 and 6,652 microstrain and predictions for 4.000. 4.500, 6,100. and 6.500 microstrains are shown.

FIG.5. The model's predictions of modulus reduction versus cycle *number* for human femoral bone in four-point bending compared with experimental data. The initial strains for the model and specimens are shown on each graph. Data are represented by symbols, and model predictions by solid lines. In each case, there are three specimens.

Parameter	Value	P value
K_1	5.40	0.593
K_2	8.60×10^{-5}	0.005
K_3	10.50	0.051
K_4	3.60×10^{-7}	0.035
K_5	1.60×10^{-8}	0.476
K_6	2.70	0.019
p	3.00	0.018
q	2.05	0.002
V_f	0.65	0.296
E_f (GPa)	14	0.001
E_m (GPa)	16	0.004

TABLE 1. Model parameters determined by regression from the data of Partin et al. (17), and p values for change in fatigue life in the parametric sensitivity test.

	Initial flexural strain (microstrain)		
	6,000	6,500	7,500
Model	560	3,230	9,660
Data mean	808	3,320	10,282
SD	425	2,084	7,701

TABLE 2. Fatigue life in cycles-to-failure: model predictions compared with experimental means and SDs.