A MODEL TO ASSESS FATIGUE DAMAGE OF HUMAN CORTICAL BONE

H. Najmi[1] and A. Varvani-Farahani[2]

Dept. of Mechanical and Industrial Engineering, Ryerson University, 350 Victoria Street, Toronto, Ontario, M5B 2K3

[1]Graduate student (hnajmi@aluma.com)

[2]Associate Professor (avarvani@ryerson.ca)

INTRODUCTION

Cortical bone has different types and structures. Haversian system or secondary osteon type of cortical bone shown in Figure 1 is the major bone type found in adult humans. Haversian system is the result of remodeling phenomenon in bone which gives it better resistance to microcracking and fatigue fracture [1-6].

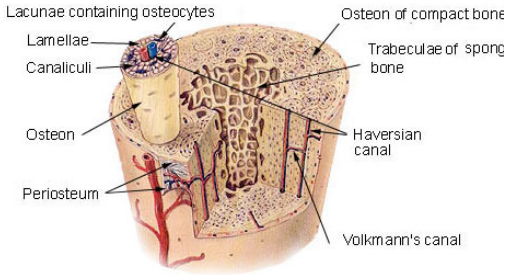

Figure 1: Haversian system (Secondary Osteon) bone type.

Many researchers have tried to develop a fatigue model mostly by using curve fitting techniques on fatigue test results [7-10]. Bone structure changes as a result of age, temperature, physical activity, sickness and remodeling. This will directly affect the mechanical properties of bone such as stiffness or fatigue strength. Bone under tensile cyclic loading generates many microcracks which causes reduction in stiffness.

The present study intends to model fatigue response of human cortical bone as a natural composite material at which minerals embedded in collagen strings constructing Osteons are reinforced fibres lying in interstitial bone (cement lines) as matrix in bone structure. In the proposed damage model, present authors incorporated stiffness reduction of bone materials as function of mechanical properties of osteons and interstitial bone.

FATIGUE DAMAGE MODEL

Damage mechanism of cortical bone is characterized by three phases of fatigue damage progress within interstitial cement lines (matrix), Osteons (fibres) and Osteon-cement interface [11]. In phase I, initiation of microcracks within cement matrix results in a drop in stiffness of bone structure as number of load cycles increases. Stiffness loss changes in slope of damage-number of cycle diagrams in the secondary phase where microcracks grow along the interface. In the phase III, stiffness further declines rapidly leading to failure.

The proposed damage model addresses three phases of damage in cortical bone and is developed based on an earlier fatigue damage model for composite materials [12]:

$$D = E_m^* D_m + E_f^* D_f \qquad (1)$$

where D is equal to $1\text{-}E_n/E_0$ and is the damage accumulated in bone and has a value of 0 at start to a maximum of 1.0 at fracture point.

E_n is the modulus of elasticity at n_{th} cycle and E_0 is the initial modulus of bone.

The backbone elements of Eq. (1) are constructed based on the fact that the stiffness of the composite just prior to failure is obtained by subtracting the summation of the accumulated damage in fibre and that of matrix from the initial composite normalized stiffness (E/E_0=1). It is assumed that the matrix is severely damaged prior to final failure and fibres are degraded up to a point where the composite can no longer withstand the applied load [12].

Term E^*_f represents the ratio of the product of Osteon modulus of elasticity E_f and its volume fraction V_f over the initial modulus of elasticity of bone (E_fV_f/E_0). Similarly, E^*_m corresponds to the same ratio for the matrix (cement lines). Modulus of elasticity of the fibres (osteons), E_f, is highly dependent on mineral composition and collagen fibres arrangement in osteons as function of age. A model to calculate E_f of Osteons is earlier proposed by Krenchel and Katz in [2].

Volume fraction of osteons, V_f, is a function of physical activity and health, gender and most importantly age. Remodeling phenomenon in bone can also change V_f. Remodeling not only help the crack arresting mechanisms but also reorient the minerals which are very stiff to the direction most beneficial to resist the applied load. Rate of remodeling is an unknown property in bone biomechanics as of yet which has a great impact in fatigue life of bone.

Taylor and Lee [6] measured the primary bone percentage in human cortical bone and they found its variations with donor age. Volume fraction of osteons was further calibrated based on donor age as:

$$V_f = \alpha\left(1-\exp(-4\frac{Age}{100})-2.0\times10^{-4}\times Age\right) \qquad (2)$$

Coefficient α accounts for other factors affecting V_f such as bone heath and gender and is about unity.

Damage evolution in matrix (cement lines), D_m, and fibers (osteons), D_f, as number of cycles increases are described using equations (3) and (4), respectively as:

$$D_m = r\cdot\left((1-f)\left(1-\exp\left(-\frac{\sigma_a}{2}\frac{n}{N_f}\right)\right)+f\frac{n}{N_f}\right) \qquad (3)$$

and

$$D_f = r\cdot\left((1-f)\cdot\exp\left(\sigma_a\left(\frac{n}{N_f}-1\right)\right)+f\frac{n}{N_f}\right) \qquad (4)$$

Both equations (3) and (4) have a linear part which presents the secondary part of the fatigue diagram. In these equations, n is the load cycle number, N_f is the number of cycles to failure and is estimated as [14]:

$$N_f = 0.5\left(\frac{\sigma_a}{118.96}\right)^{-18.2} \qquad (5)$$

In equations (3) and (4), r is the ratio of applied stress, σ_a, to the initial modulus of bone and varies between 0 and 1:

$$r = 100\frac{\sigma_a}{E_0} \qquad (6)$$

Equations (3) and (4) mathematically represent a damage function with a starting point at zero and a maximum value of 1.0. Mathematical terms have been identified through a rigorous study of many fatigue curves and models and meaningfully selected to best model the fatigue behavior of human cortical bone loaded under tensile fatigue loading conditions.

The modified form of Eq (1) for damage assessment of cortical bone includes the effect of Osteon-cement interfacial bonding by introducing factor f. Factor f is described as a function of interface shear strength, volume fraction of Osteon and cement, and the applied shear stress along the interface. This factor varies between zero and unity and is also influenced by the donor age [13].

RESULTS

The proposed damage model was found to be valid for tensile fatigue loading where stress ratio R>0 and is more accurate within the strain

range of 2500-4000 με which compares to loadings in living life.

A computer program was used for iteration of variables required for damage assessment of cortical bone material as the number of loading cycles progressed.

Figure 2 compares actual test data and predicted fatigue data using modified damage model. Experimental fatigue data for human cortical bone have been collected from previous works [9,10,16,18] and for each set of data the model input parameters were calculated, analyzed to find out the trends and proximity to real life values. Most samples showed good agreement as compared with the damage curves predicted using the modified damage model. Four different bone samples with various mechanical properties loaded at different cyclic stress magnitudes are presented here (See Figure 1).

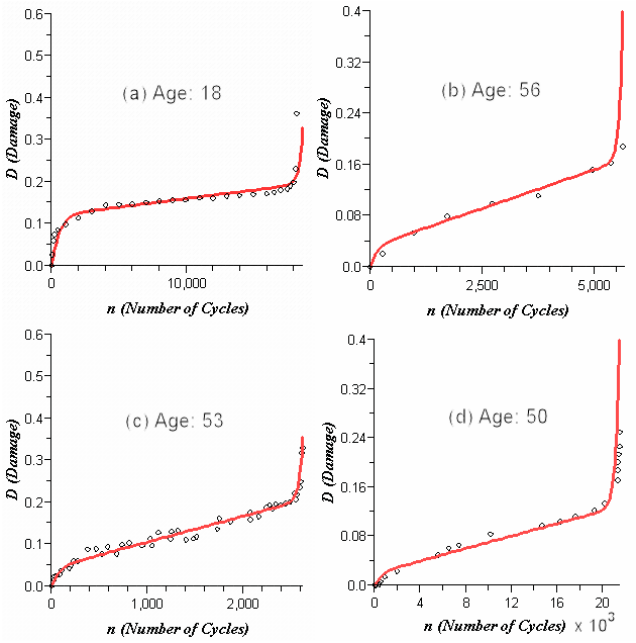

Figure 2: Experimental data [9, 10, 16 and 18] (shown as circles) versus predicted fatigue damage curves (solid lines)

Table 1 presents mechanical properties and fatigue life data of various cortical bone samples fatigue tested under different cyclic applied stresses. Experimental test data have been taken from references 9,10,16, and18.

CONCLUSION

Bone is stronger in compression than in tension and as such a model for tensile fatigue response can unconservatively predict the shortest fatigue life of bone under real loading conditions. The present study developed a fatigue damage model to predict the fatigue response of human cortical bone. The proposed damage model considers bone as a natural composite material consisting of Osteon fibres embedded in interstitial bone and separated by cement lines. Predicted fatigue damage results were found in good agreement with experimentally obtained damage results [9,10,16,18].

One of the complications involved with damage analysis of cortical bone is the lack of material properties (e.g., stiffness) of the Osteons. Osteons exist in different mineral arrangements which greatly affect their stiffness values. Further future investigation is required to precisely and accurately compute this important property of the bone. Moreover, the effects of biological parameters (age, nutrition, physical activity, volume fraction of Osteons and the bone density) are crucial on damage assessment of cortical bones.

Acknowledgements: The financial support by Natural Sciences and Engineering Research Council (NSERC) of Canada is very much appreciated.

Table 1: Mechanical properties of various bone samples tested at different cyclic stresses.

Parts (Fig. 2)	*Source of Test Data*	*Donor Age*	*Applied Stress* σ_a **(MPa)**	*Initial Modulus of Elasticity* E_0 *(GPa)*	*Number of Cycles to Fracture* (N_f)	*Osteon Modulus of Elasticity* E_f *(GPa)*	*Osteon Volume Fraction* V_f	*Interface Strength (f)*
a	*Pattin et al. [9]*	18	70.7	21.4	18658	22.5	51%	0.23
b	*Zioupos et al. [9]*	56	72.8	13.2	5677	13.9	88%	0.25
c	*Cotton et al. [16]*	53	72	20.0	2626	18.0	87%	0.45
d	*Griffin et al. [18]*	50	69.2	15.9	21500	15.9	85%	0.25

REFERENCES

[1] Burr DB, Martin RB, Schaffler MB & Radin EL (1985). *J. Biomechanics*, vol. 18, pp. 189–200.

[2] Currey JD (2002). Bones, Structure and Mechanics, Princeton University Press, Princeton, NJ.

[3] Carter DR, Hayes WC & Schurman DJ (1976). *J. Biomechanics*, vol. 9, pp.211–218.

[4] Schaffler MB, Jepsen KJ (2000). *International Journal of Fatigue*, vol.22, pp. 839-846.

[5] Taylor D, Prendergast PJ (1997). Proceedings of the Institution of Mechanical Engineers Part H – *Journal of Engineering in Medicine*, vol. 211, pp. 369–375.

[6] Taylor D, Lee TC (2003). *Journal of Anatomy*, vol. 203, PP.203-211.

[7] Carter DR, Hayes WC (1977). *Journal of Biomechanics*, vol. 10, pp. 325–337.

[8] Carter DR, Hayes WC (1976). *Journal of Biomechanics*, vol.9, pp. 27-34.

[9] Pattin CA, Caler WE, & Carter DR (1996). *Journal of Biomechanics*, vol. 29, pp. 69-79.

[10] Zioupos P, & Casinos A (1998). *Journal of Biomechanics*, vol.31, pp.825-833.

[11] Reifsnider K (1991). *Fatigue of composite materials*, Elsevier, Amsterdam.

[12] Ramakrishnan V, & Jayaraman N (1993). *Journal of Materials Science*, vol.28, pp.5592-5602.

[13] Diab T, Sit S, Kim D, Rho J, Vashishth D (2005). *European Journal of Morphology*, vol. 42, pp. 53 – 59.

[14] Griffin LV, Gibeling JC, Martin RB, Gibson VA, Stover SM (1997). *Journal of Orthopedic Research*, vol.15, pp. 607-614.

[15] Hiller LP, Stover SM, Gibson VA, Gibeling JC, Prater CS, Hazelwood SJ, Yeh OC, Martin RB (2003). *Journal of Orthopaedic Research*, vol. 21, pp. 481-488.

[16] Cotton JR, Winwood K, Zioupos P, Taylor M (2005). *Journal of Biomechanical Engineering*, vol.127, pp. 213-219.

[17] Burr DB, Turner CH, Naick P (1998). *Journal of Biomechanics*, vol. 31, pp. 337–345.

[18] Griffin LV, Gibeling JC, Martin RB, Gibson VA, Stover SM (1999). *Journal of Biomechanics*, vol. 32, pp. 105-109.

[19] Pidaparti RM, Wang QY, Burr DB (2001). *Biomedical Materials and Engineering*, vol.11, pp. 69-78.