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RESIDUAL STRENGTH OF EQUINE BONE IS NOT REDUCED BY INTENSE FATIGUE LOADING: IMPLICATIONS FOR STRESS FRACTURE

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Abstract—Fatigue or stress fractures are an important clinical problem in humans as well as racehorses. An important question in this context is, when a bone experiences fatigue damage during extreme use, how much is it weakened compared to its original state? Since there are very limited data on this question and stress fractures are common in racehorses, we sought to determine the effect of fatigue loading on the monotonic strength of equine cortical bone. Beams were machined from the dorsal, medial and lateral cortices of the third metacarpal bones of six thoroughbred racehorses. Beams from left and right bones were assigned to control and fatigue groups, respectively ($N = 18$ each). The fatigue group was cyclically loaded in three-point bending at 2 Hz for 100,000 cycles at 0–5000 microstrain while submerged in saline at 37°C. These beams, as well as those in the control group, were then monotonically loaded to failure in three-point bending. The monotonic load–deflection curves were analyzed for differences using three-factor (fatigue loading, anatomic region, and horse) analysis of variance. The mean failure load was 3% less in the fatigue group, but this reduction was only marginally significant. Neither elastic modulus nor yield strength was significantly affected by the fatigue loading. The principal effects of fatigue loading were on post-yield behavior (yield being based on a 0.02% offset criterion). The work done and the load increase between yield and failure were both significantly reduced. All the variables except post-yield deflection were significantly affected by anatomic region. In summary, loading equivalent to a lifetime of racing does not significantly weaken equine cortical bone *ex vivo*. The clinical implication of this may be that the biological repair of fatigue damage can actually contribute to stress fracture if pressed too far.

INTRODUCTION

Racehorses are exceptional athletes in that they place extraordinary stress on their musculoskeletal systems while training and racing. Consequently, they are prone to stress fractures at several skeletal sites (Nunamaker *et al.*, 1990). The third metacarpal or 'cannon' bone is one of the most common stress fracture sites in racehorses; 70% of thoroughbred racehorses suffer from either stress fractures of their cannon bones or a pathophysiologic response to fatigue damage known as 'bucked shins'. Therefore, this bone has been the subject of several experimental studies designed to elucidate its structure (Nunamaker *et al.*, 1989; Stover *et al.*, 1992), mechanical properties (Les *et al.*, submitted), *in vivo* strain magnitudes (Biewener *et al.*, 1983; Nunamaker *et al.*, 1990; Turner *et al.*, 1975), and fatigue properties (Gibson *et al.*, 1995; Nunamaker *et al.*, 1991). While peak periosteal surface strains during vigorous activities are between 2000 and 3000 microstrain in most animals, cannon bone strains exceeding 5000 microstrain have been measured in horses running at racing speeds (Nunamaker *et al.*, 1990). Racehorses begin training and racing at a relatively early age, and the mechanical demands of these activities may be responsible for producing a unique type of periosteal bone formation and primary bone structure (Stover *et al.*, 1992). Bone formation actually begins within the periosteum, off the surface of the bone, producing islands of woven bone. These become incorporated into the primary bone structure as layers of woven bone separated by rows of primary osteons. This type of bone is different from human and canine circumferential lamellar primary bone, which lack the woven bone layers, and from bovine plexiform bone, in which circumferentially extended vascular spaces containing multiple blood vessels form in place of primary osteons.

Fatigue loading of bone and other materials introduces microscopic damage (Carter and Hayes, 1977b). This damage may be responsible for the reduction in elastic modulus which is commonly produced by repetitive loading (Carter and Hayes, 1977a). Fatigue loading also activates bone remodeling (Burr *et al.*, 1985; Mori and Burr, 1993), presumably for the purpose of repairing the fatigue damage. It has been proposed that under normal circumstances the generation of damage by loading and its repair by remodeling are able to reach an equilibrium state in which the damage burden waiting to be repaired is tolerable (Martin, 1992). It has also been proposed that when loading is excessive, accelerated remodeling not only removes damage at a higher rate, but also increases the rate of damage production (Martin, 1995). This is because the porosity associated with remodeling decreases the elastic modulus of, and increases the strain in, the bone. According to this theory, stress

fractures do not usually represent simple fatigue failure of the bone material, but are the result of a 'vicious circle' in which the biological repair mechanism (remodeling) cannot achieve a new equilibrium with damage production.

If this theory is correct and applicable to racehorses, one would suppose that fatigue loading equivalent to many, many miles of running at racing speeds would, in the absence of remodeling, not necessarily lead to fracture. Put another way, one may hypothesize that a large amount of fatigue loading of equine cannon bone tissue would not seriously alter its failure stress if the loading were done *ex vivo*. At 12 m per stride and 1400 m per race, 100,000 cycles of loading would approximate 850 races, more than any horse would be likely to accumulate during its career, including training activities. We therefore conducted an experiment to answer the question, will 100,000 cycles of flexural fatigue loading at 0–5000 microstrain, in the absence of biological repair processes, significantly reduce the strength or elastic modulus of equine cannon bone tissue?

METHODS

Left and right cannon bones were obtained at necropsy from six thoroughbred racehorses using an approved institutional protocol. The mean age of these animals was 3.5 (range: 3–5) yr. One was female, two were males, and three were geldings. All had trained and raced until shortly before their deaths. The whole bones were stored frozen at -4°C. Subsequently, three beams measuring $4 \times 10 \times 100$ mm were machined from each bone, taking care to keep the specimens wet. The long axes of these beams were parallel to that of the bone, and the 10×100 mm faces were parallel to the periosteal surface in one of three cortical quadrants: medial, dorsal, and lateral (Fig. 1). The total number of beams was 36: 3 beams/bone × 2 bones/horse × 6 horses. Having established that left–right differences in fatigue and modulus properties are absent in such beams (Gibson *et al.*, 1995), we sorted the beams into fatigue and control groups such that the fatigue group contained the contralateral mates to the beams in the control group. All beams were stored frozen prior to mechanical testing.

All mechanical testing was carried out in symmetric three-point bending in accord with ASTM standard D790 with a span of 64 mm. The testing was done on a model 809.10 MTS servohydraulic testing machine running under version 1.4c Testware SX software on a Compac 386 computer. The supports were fixed 9.5 mm diameter stainless steel cylinders. In a previous investigation, we demonstrated that sliding contact at the supports of beams loaded in four-point bending creates wear grooves that introduce a nonlinearity to the force–deflection curve (Griffin *et al.*, submitted). In the present study, specimen wear at the outer supports was prevented by placing a layer of Teflon tape between the supports and the beam. (Since there was little relative motion between the beam and the central support, wear was not an issue there.) Testing was done in a saline bath at 37°C. Load was measured by the machine's 2500 N load cell; deflection of the beam was measured with a Schaevitz model DC-E-125 LVDT in direct contact with the beam surface opposite the center support. The periosteal face of the beam was loaded in tension.

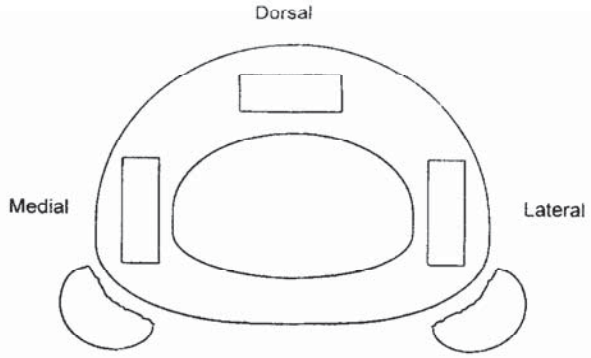

Fig. 1. Diagram showing the location of the beams cut from the medial, dorsal, and lateral cortices of the equine third metacarpus. The beams' long axes were parallel to the bone axis; the diagram represents cross-sections of the bone diaphysis (*c.* 50 mm in diameter) and the beams (4×10 mm) in their approximate original locations.

The fatigue group beams were sinusoidally loaded for 100,000 cycles at 2 Hz under load control. (At approximately 12 m per stride and 1400 m per race, 100,000 cycles would correspond to about 850 races or equivalent training sessions.) The peak applied load was that required to produce 5000 microstrain on the tensile surface as determined from beam theory and an initial load–deflection curve. The deflection rate for this initial load cycle was 3.4 mm s^{-1} (20,000 surface microstrain per s), and loading was reversed as soon as the target deflection was reached. The minimum load for subsequent cycling was 10 N. Using a data acquisition rate of 200 Hz, the first 100 load–deflection cycles were saved, then 10 cycles were recorded after every 100 cycles to $N = 1000$, every 1000 cycles to $N = 10{,}000$, and so on through the end of the cyclic loading. Immediately following fatigue loading, the fatigue group beams were loaded monotonically to failure at 1.00 mm s^{-1} while recording their load–deflection curve. The control group beams were not fatigued, but simply loaded monotonically to failure at 1.00 mm s^{-1} following immersion in a saline bath at 37°C for the 13.9 h required to cyclically load the fatigue group. This precaution was taken because we have shown that mineral leaching accompanying saline immersion for 6 d can significantly reduce beam stiffness (Gustafson *et al.*, 1995), and we are unsure of the minimal immersion time for such an effect.

Several variables were calculated for the monotonic failure test of both the fatigue and control groups (Fig. 2). Using beam theory, elastic modulus was determined by performing linear regression on the initial portion of the load–deflection curve (from 20 to 200 N) and using the beam dimensions. The load and deflection at failure were obtained from the load–deflection curve. Load and deflection at yield were determined using the 0.02% offset strain criterion. Work-to-yield and work-to-failure were calculated as the areas under the load–deflection curve to the 0.02% offset and fracture points, respectively. Post-yield load, deflection, and work were computed as the

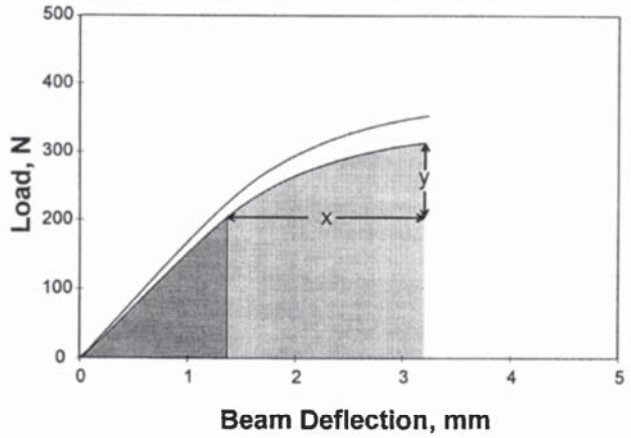

Fig. 2. Typical monotonic bending load–deformation curves for a left–right pair of dorsal beams. Their end points represent failure. Note the substantial nonlinearity in both curves. The curve which rises higher is that of the control beam. The lower curve represents a beam previously loaded for 100,000 cycles at 5000 microstrain. For the fatigued beam, the distance x is the post-yield deflection, from the 0.02% offset yield point to failure. The distance y indicates the post-yield load rise. The dark and light-shaded regions indicate the work-to-yield and the post-yield work, respectively.

differences between the failure and yield load, deflection, and work values, respectively. (The term 'yield' is used imperfectly here; see the Discussion section).

Two analysis of variance (ANOVA) models were used to analyze the results. Inspection of the monotonic test data revealed more regional differences in mechanical properties than differences between the fatigue and control groups. Initially, a three-factor (fatigue loading, anatomical region, horse) ANOVA was done for a completely randomized design. Because of the significant effect of horse in the initial ANOVA, the analysis was repeated, blocking on horse to adjust for this animal-to-animal variability. In all cases, standard deviations were used to express the variability of the data and $p < 0.05$ was the criterion for statistical significance.

RESULTS

For both the fatigue and control groups the load–deflection curves were nonlinear but lacked a distinct yield point (Fig. 2). The initial three-factor ANOVA showed that all three regions were significantly different from one another with respect to all the variables in Table 1 (Table 3, 'no blocking' rows). Fatigue loading reduced the failure load by 3% ($p = 0.048$) and work-to-failure by 19% ($p = 0.013$), but modulus and the other variables in Table 1 were not affected. All the variables in this table varied significantly among the different horses.

When the ANOVA was repeated, blocking on horse to adjust for this animal-to-animal variability (Table 3, 'blocking' rows), the regional differences remained significant, but the effects of fatigue loading on strength and work-to-failure were no longer quite significant ($p = 0.060$ and $p = 0.054$, respectively). Modulus again proved unaffected by fatigue. This was further demonstrated by the 'flatness' of modulus vs cycle number plots (Fig. 3). In summary, fatigue loading did not affect the elastic modulus of the beams, and any effect on strength and work-to-failure is dubious, but all three regions of the cannon bone were different from one another with respect to all the variables in Table 1. The lateral cortex was strongest and stiffest, and the anterior cortex was weakest and most compliant.

The post-yield behavior provided the only variables significantly affected by fatigue loading (Fig. 2, Table 2). The initial three-way ANOVA showed that both region and fatigue loading significantly affected the post-yield load increase, and in this case horse was not a significant factor (Tables 2 and 3). The post-yield load rise was greatest in the lateral region and least in the anterior region, and was diminished 16% by the cyclic loading. Post-yield work was reduced 6% by fatigue loading and also varied by region. In this case, horse was a significant factor, but blocking on horse did not remove the significance of the fatigue result.

Since post-yield load and deflection were not significantly related to horse, there was no particular reason to block on this variable, but we show such results for the sake of completeness (Table 3). It should also be noted

Table 1. Monotonic failure and modulus data

Region	Fail. load (N)	Yield load (N)	Fail. defl. (mm)	Elastic mod. (GPa)	Work-to-failure (J)	Work to yield (J)
Dorsal						
Control	336 ± 22	217 ± 23	3.78 ± 0.88	16.0 ± 1.9	842 ± 199	159 ± 20
Fatigue	308 ± 28	223 ± 33	3.36 ± 0.73	15.4 ± 1.9	636 ± 181	177 ± 34
Medial						
Control	383 ± 18	261 ± 15	4.14 ± 0.66	18.1 ± 01.0	1121 ± 219	205 ± 15
Fatigue	380 ± 19	265 ± 23	4.21 ± 0.71	18.5 ± 1.2	1112 ± 253	207 ± 27
Lateral						
Control	405 ± 21	273 ± 14	4.36 ± 0.60	20.7 ± 1.0	1292 ± 242	209 ± 18
Fatigue	403 ± 26	289 ± 32	3.98 ± 0.42	20.1 ± 1.0	1137 ± 167	228 ± 40
All regions						
Control	375 ± 35	250 ± 30	4.09 ± 0.72	17.9 ± 1.9	1185 ± 282	191 ± 28
Fatigue	364 ± 48	259 ± 39	3.85 ± 0.70	18.1 ± 2.4	962 ± 305	204 ± 38

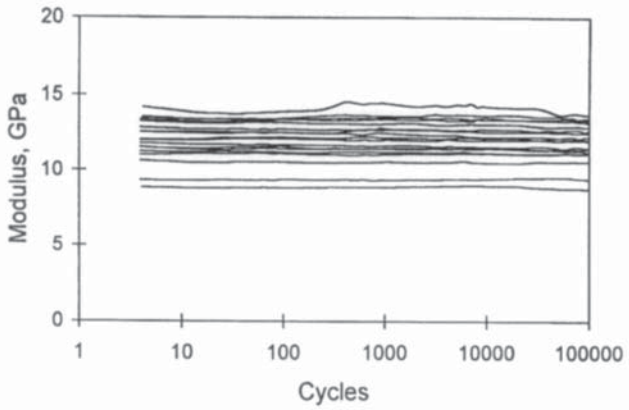

Fig. 3. Elastic modulus vs cycle number for all the fatigue group specimens. The curves begin at cycle 5 because each modulus value is an average over nine cycles, sampled periodically as explained under the Methods section. The data points have been connected by lines for ease in visualizing the behavior of individual specimens. There was no change in any of the individual moduli over 100,000 cycles. The variability among specimens is primarily due to regional differences.

that there were no interactions between region and fatigue in any of the analyses.

DISCUSSION

We sought to determine the effects of 100,000 cycles of bending to 5000 microstrain on the elastic modulus and strength of bone tissue from the equine cannon bone. We found that the fatigue loading did not change the elastic modulus, or the yield or failure strength, supporting the concept that the biological response to fatigue loading contributes to stress fracture. On the other hand, the fatigue loading substantially altered the post-yield work and load rise, indicating that some sort of fatigue damage had been introduced. While all the mechanical properties which we measured were functions of cortical location, the effects of fatigue loading were similar in each region, suggesting that the regional variations which existed in the bone structure did not affect susceptibility to the kind of fatigue damage which occurred.

There are several limitations to our experiment. The specimens came from a relatively small sample of six horses. We found that there were some significant differences from horse to horse, but our sample is too small to know whether these differences are due to age, gender, or other factors. The various mean values reported here may not be representative of thoroughbred racehorses in general. On the other hand, the focus of the study was the effects of fatigue loading on monotonic mechanical properties, horse was not a significant factor with respect to one of the two variables which fatigue did affect (post-yield load rise), and controlling for horse-to-horse variations only strengthened the effect of fatigue. Therefore, there is reason to believe that a larger study would produce a similar result.

The 0.02% offset 'yield' point which we defined, and the variables we have defined as characterizing 'post-yield' behavior, may be more properly described simply in terms of nonlinearity rather than yielding. Our specimens' unloading curves were intermediate between the classically plastic and pseudoplastic behaviors described by Fondrk *et al.* (1988), following Rabotnov (1980). It is unclear how much of the nonlinearity is due to slipping ('flow' or plasticity) of elastic components within the bone, and how much is due to rupturing of such components ('other damage' or pseudoplasticity). We therefore have used the term 'post-yield' as a matter of convenience rather than with a strict mechanism in mind.

Another limitation is that stemming from the mode of loading and the nature of the test specimens. In the living horse the tissue in the three cortices is certainly not loaded the way it was loaded in our beams, but it is not at all clear how the tissue in these sites is loaded *in vivo*. *In vivo* strain gauge data of Turner *et al.* (1975) indicated mediolateral bending of the equine metacarpus during trotting, with superimposed compression and the lateral side in slight tension. Biewener *et al.* (1983) found that gait analysis showed saggital plane bending in the cannon bone during various gaits, but *in vivo* strain gauge data suggested uniaxial compression dominated the loading. Nunamaker *et al.* (1990) found that the principal direction of the strain on the dorsolateral cortex changed by 40° during a transition from trotting to racing speed.

Table 2. Post-yield behavior data

Region	Post-yield load (N)	Post-yield defl. (mm)	Post-yield work (J)	% work-to-failure that is post-yield
Dorsal				
Control	119 ± 16	1.78 ± 0.91	682 ± 219	79.5 ± 8.0
Fatigue	84 ± 19	2.15 ± 0.11	460 ± 191	69.2 ± 14.6
Medial				
Control	122 ± 5	2.00 ± 0.58	916 ± 215	81.1 ± 3.7
Fatigue	115 ± 12	2.13 ± 0.13	905 ± 249	80.4 ± 5.4
Lateral				
Control	132 ± 17	2.31 ± 0.62	1083 ± 250	83.3 ± 4.2
Fatigue	114 ± 11	2.10 ± 0.18	909 ± 136	79.9 ± 2.1
All regions				
Control	124 ± 14	2.03 ± 0.71	894 ± 273	81.3 ± 5.5
Fatigue	104 ± 20	2.13 ± 0.14	758 ± 286	76.5 ± 10.1

Table 3. p-values for ANOVA F-scores

Variable	Factor		
	Fatigue	Region	Horse
Failure load			
No blocking	0.048	0.0000	0.003
Blocking	0.060	0.0000*	—
Yield load			
No blocking	0.137	0.0000	0.004
Blocking	0.140	0.0000*	—
Failure defl.			
No blocking[1]	0.086	0.004	0.001
Blocking	0.161	0.009†	—
Elastic modulus			
No blocking	0.488	0.0000	0.001
Blocking	0.549	0.0000*	—
Work-to-failure			
No blocking[2]	0.013	0.0000	0.005
Blocking	0.054	0.0000†	—
Work-to-yield			
No blocking	0.118	0.0008	0.049
Blocking	0.113	0.0001†	—
Post-yield load			
No blocking	0.002	0.009	0.362
Blocking	0.0002	0.002†	—
Post-yield defl.			
No blocking[3]	0.454	0.307	0.059
Blocking	0.564	0.482	—
Post-yield work			
No blocking[4]	0.011	0.0000	0.005
Blocking	0.030	0.0000†	—

* All three regions different.

† Dorsal different from medial and lateral, which were not different from each other.

Note. The following footnotes indicate the only interactions which were significant in the above analyses: [1]region–horse interaction, $p = 0.046$; [2]region–horse interaction, $p = 0.017$; [3]fatigue–horse interaction, $p = 0.048$; [4]region–horse interaction, $p = 0.034$.

Thus, the strain distribution in the different cortices of the equine cannon bone is poorly understood. Because similar problems are encountered in other species, few studies of the material properties of bone are able to closely approximate normal *in vivo* strain patterns. Given these difficulties, we felt it was most useful to load all the specimens the same way with respect to their histologic structure, i.e. with the neutral plane parallel to the periosteal surface, the layers of woven bone, and the rows of primary osteons.

The prediction of strength and modulus reductions following prescribed amounts of repetitive loading is an important problem which has not been adequately solved for any material (Ben-Amoz, 1990). For the case of bone, there are very limited data from which to develop a residual strength theory. Carter and Hayes (1977a) examined the residual tensile strength of bovine femoral specimens following fatigue in rotating bending at a frequency of 125 Hz and 4800 microstrain. Under these conditions, specimens containing only primary bone and loaded for 25,000 cycles lost about 8% of their tensile strength. Specimens containing substantial amounts of secondary bone were less fatigue resistant. These are apparently the only published data on the residual strength of bone, and it would be difficult to apply them to horse racing because the 125 Hz frequency was nonphysiologic and the equine cannon bone has a different histologic structure than cow bone (Stover *et al.*, 1992). Our specimens of equine bone lost only 3% of their strength after 100,000 cycles of loading, and this change was not statistically significant. Therefore, there is reason to hypothesize that the equine cannon bone is more fatigue resistant than the bovine specimens. This may be related to the differences in primary bone structure noted in the introduction, or to remodeling activity, or both.

We found no reduction in the elastic modulus of the fatigued equine beams. Carter and Hayes (1977a) did not report on the elastic modulus of their bovine specimens, but they found that the *secant* modulus was clearly reduced by less than 1000 cycles of fatigue loading. Examination of their stress–strain curves suggests that most of this change involved a transition from nearly linear to obviously nonlinear behavior, rather than reduction of the initial slope. Our specimens were quite nonlinear to begin with (Fig. 2), and the primary result of fatigue was to change the nature of the additional load that could be supported following yield.

There are three important differences between the two experiments which could explain their different results. The first is the different modes of loading, and there are insufficient data to know which way this would bias the result. The second is the greater loading frequency of the bovine test. Caler and Carter (1989) have shown that creep and fatigue phenomena are interrelated such that lower frequencies ought to produce more damage than higher frequencies. Therefore, our 2 Hz experiment should have reduced residual strength more than if it had been conducted at 125 Hz. Finally, the third difference is that our specimens were largely composed of secondary bone, which should have reduced their fatigue resistance relative to Carter and Hayes' primary bone specimens (Carter *et al.*, 1976). Clearly, additional experiments need to be done under comparable methodologies to sort these matters out, but a working hypothesis could be that equine cortical bone structure (both primary and secondary) is more fatigue resistant than bovine structure. Obviously, both need to be compared to the human case, and the advantages and disadvantages of remodeling to produce an osteonal structure need to be more fully studied in these different systems.

The only sure effects of fatigue loading which we measured were related to post-yield behavior. One of the first studies to recognize the importance of bone's post-yield behavior was that of Burstein and co-workers (1972). More recently it has become clear that 'yielding' in bone is fundamentally different from the classical flow plasticity seen in metals, which involves slippage along crystalline interfaces. Instead, 'yielding' in bone seems to involve damage in the form of rupture of elastic structural components (Fondrk *et al.*, 1988). Because the effects on modulus and strength were less, the damage in our equine specimens, containing much secondary bone, was apparently different in some respects from that in the bovine specimens containing only primary bone tested by Carter and Hayes (1977a).

Clearly, much remains to be learned about the effects of histologic structure on the fatigue behavior of bone. Other studies in our laboratories are attempting to elucidate these effects. In the meantime, the present study has provided the first data on the residual strength of bone at physiologic strain rates and, for the racehorse, strain magnitudes. The results suggest that equine bone may be quite resistant to *ex vivo* fatigue damage, and that remodeling may therefore play an important role in stress fracture etiology.

REFERENCES

Ben-Amoz, M. (1990) A cumulative damage theory for fatigue life prediction. *Engng Fract. Mech.* **37,** 341–347.

Biewener, A. A., Thomason, J., Goodship, A. and Lanyon, L. E. (1983) Bone stress in the horse forelimb during locomotion at different gaits: a comparison of two experimental methods. *J. Biomechanics* **16,** 565–576.

Burr, D. B., Martin, R. B., Schaffler, M. B. and Radin, E. L. (1985) Bone remodeling in response to *in vivo* fatigue microdamage. *J. Biomechanics* **18,** 189–200.

Burstein, A. H., Currey, J. D., Frankel, V. H. and Reilly, D. T. (1972) The ultimate properties of bone tissue: the effects of yielding. *J. Biomechanics* **5,** 35–44.

Caler, W. E. and Carter, D. R. (1989) Bone creep-fatigue damage accumulation. *J. Biomechanics* **22,** 625–635.

Carter, D. R. and Hayes, W. C. (1977a) Compact bone fatigue damage—I. Residual strength and stiffness. *J. Biomechanics* **10,** 325–337.

Carter, D. R. and Hayes, W. C. (1977b) Compact bone fatigue damage: a microscopic examination. *Clin. Orthop. Rel. Res.* **127,** 265–274.

Carter, D. R., Hayes, W. C. and Schurman, D. J. (1976) Fatigue life of compact bone—II. Effects of microstructure and density. *J. Biomechanics* **9,** 211–218.

Fondrk, M., Bahniuk, E., Davy, D. T. and Michaels, C. (1988) Some viscoplastic characteristics of bovine and human cortical bone. *J. Biomechanics* **21,** 623–630.

Frost, H. M. (1960) Some aspects of the mechanics and dynamics of blood–bone interchange. *Henry Ford Hospital Med. Bull.* **8,** 36–51.

Gibson, V. A., Stover, S. M., Martin, R. B., Gibeling, J. C., Gustafson, M.B. and Griffin, L.V. (1995) Fatigue behavior of the equine third metacarpus: mechanical property analysis. *J. orthop. Res.* **13,** 861–868.

Griffin, L. V., Gibson, V. A., Gibeling, J. C., Martin, R. B. and Stover, S. M. The influence of wear grooves and friction on the mechanical response of bone during four point flexural loading. *J. Biomechanics*, submitted.

Gustafson, M. B., Gibson, V. A., Stover, S. M. and Martin, R. B. (1995) Requirement of calcium buffering to maintain equine cannon bone stiffness in saline. *Trans. orthop. Res. Soc.* **20,** 297.

Hoshaw, S. J., Cody, D. D., Saad, A. M. and Fyhrie, D. P. (1995) Decrease in canine proximal femoral ultimate strength and stiffness due to fatigue damage. *Proc. 19th Ann. Meeting of the American Society of Biomechanics*, 24–26 August, Stanford, CA, pp. 143–144.

Les, C. M., Stover, S. M., Keyak, J. H., Taylor, K. T. and Willits, N. H. The distribution of material properties in the equine metacarpus. *J. Biomechanics*, submitted.

Martin, R. B. (1992) A theory of fatigue damage accumulation and repair in cortical bone. *J. orthop. Res.* **10,** 818–825.

Martin, R. B. (1995) A mathematical model for fatigue damage repair and stress fracture in osteonal bone. *J. orthop. Res.* **13,** 309–316.

Mori, S. and Burr, D. B. (1993) Increased intracortical remodeling following fatigue damage. *Bone* **14,** 103–109.

Nunamaker, D. M., Butterweck, D. M. and Black, J. (1991) *In vitro* comparison of thoroughbred and standardbred racehorses with regard to local fatigue failure of the third metacarpal bone. *Am. J. Vet. Res.* **52,** 97–100.

Nunamaker, D. M., Butterweck, D. M. and Provost, M. T. (1989) Some geometric properties of the third metacarpal bone: a comparison between the thoroughbred and standard racehorse. *J. Biomechanics* **22,** 129–134.

Nunamaker, D. M., Butterweck, D. M. and Provost, M. T. (1990) Fatigue fractures in thoroughbred racehorses: relationships with age, peak bone strain, and training. *J. orthop. Res.* **8,** 604–611.

Rabotnov, Y. N. (1980) *Elements of Hereditary Solid Mechanics.* Mir, Moscow.

Stover, S. M., Pool, R. R., Martin, R. B. and Morgan, J. P. (1992) Histologic features of the dorsal cortex of the third metacarpal bone mid-diaphysis during postnatal growth in thoroughbred horses. *J. Anat.* **181,** 455–469.

Turner, A. S., Mills, E. J. and Gabel, A. A. (1975) *In vivo* measurement of bone strain in the horse. *Am. J. Vet. Res.* **36,** 1573–1579.