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Evidence of Microstructural Adaptation to Compression Loading The Optimal Cortical Structure?

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Introduction It has been hypothesized that specific strain features, such as strain mode (tension and compression) and strain magnitude are important in the maintenance of normal bone architecture (1,2). However, strain mode and strain magnitude coexist in varying degrees between regions of a given bone. Thus, it is unclear whether one or both of these strain-related features contribute to the signal that allows bone cells to recognize normal loading conditions. Using a simple bending system, the artiodactyl calcaneus, a previous study suggested that differences in osteonal microstructure between tension and compression cortices reflect the presence of strainmode-specific adaptation (4). This suggests that bone has the capacity to process local strain-related information to create a nonuniform microstructural organization that may be optimal for the prevailing loading conditions of the given bone (1,6). The present study further investigates this idea by examining an experimentally unperturbed model which has clearly defined strain differences between regions within the cortex of the bone. Methods The horse radius was used because it is subject to habitual compression loads in the caudal cortex and habitual tension loads in the cranial cortex, but lacks any firmly adherent fibrous structures. A radius from each of ten skeletally mature horses was sectioned transversely through the diaphysis at 50% and 65% of length (Fig 1). Each section was embedded in polymethyl methacrylate, polished, and prepared for imaging in the back scattered electron (BSE) mode in a JEOL scanning electron microscope. A total of 240 images representing nearly 900 square millimeters of cortical bone, were recorded on Polaroid 52 film at 50X magnification, and saved for analysis. Both cranial and caudal cortices of each bone were subdivided into three regions: periosteal (P), middle (M), and endosteal (E). The periosteal region was located immediately adjacent to the periosteal surface, the endosteal region was located adjacent to the endosteal canal without including any trabecular bone, and the middle region was located midway between the periosteal and endosteal regions. In no case did any of the regions overlap. Each image was analyzed for number of osteons, fractional crosssectional area of secondary osteons, and fractional cross-sectional area of cortical porosity. Using an ANOVA design, comparisons between tension and compression cortices, and also comparisons between P, M, and E regions were made.

Results Analysis of the data including all regions (P, M, E) combined showed nearly twice as many osteons per mm<sup>2</sup> in the compression cortex (18.1 ± 4.9) as in the tension cortex (9.4 ± 5.2) (p < 0.01). The fractional area of secondary osteons was also found to be nearly twice as great in the compression cortex (53.7% ± 12.1) compared to the tension cortex (26.9% ± 15.2) The porosity, however, showed no significant differences between compression (4.9% ± 2.3) and tension (4.3% ± 2.3) cortices. The porosity due only to resorption spaces was examined separately and was also not significantly different between cortices. This data implies that mean osteon diameter is approximately equal in both tension and compression cortices. Results of individual regions are summarized in Table 1. There were no osteonal microstructure or porosity differences between P. M. and E regions within a given cortex. Crosssectional shapes of osteons in the tension cortex were more irregular than those in the compression cortex.

Discussion These results suggest that remodeling occurs preferentially in regions loaded in compression compared to regions loaded in tension. However, since strain magnitudes are significantly greater in the compression cortex than in the tension cortex, it is difficult to relate the association of remodeling to either strain mode or strain magnitude. Strain magnitudes increase with distance from the neutral axis of bending, and thus individual regions within each cortex are expected to be subjected to different strain magnitudes. Since there are no significant differences between P, M, and E regions within a cortex, it is unlikely that there is a direct association between remodeling and strain magnitude. However, these data do not rule out the possibility that strain magnitude accentuates a mode-specific response.

Because the occurrence and propagation of microdamage in compression loading differs from that in tension loading, it has been proposed that bone might remodel to accomodate or correct these differences (2). Mori and Burr, using the canine radius, recently showed that resorption and thus remodeling, occur in close association with microdamage (3). However, they found fewer resorption spaces in the compression cortex than in the tension cortex. The implication that there should be fewer secondary osteons in the compression region seems to be in contradiction with the results of the present study, and others, which show more osteons in the compression cortex.

It has been proposed that bone remodels to achieve a cortical structure that optimizes one or more aspects of its strain environment (1,6). If this is true, the results of Mori and Burr (3) suggest that the incidence of microdamage has changed over the life of the bone. In other words, remodeling may have affected the structure of the bone tissue, not only to repair microdamage, but to adapt the cortical structure to minimize the recurrence of microdamage. In the mature horse radii used in this study, which have been subjected only to physiologic strain histories, the equal resorption space porosities between tension and compression cortices suggest that remodeling has reached an equilibrium state. Thus, the results of this study may represent an optimal configuration in which microdamage is held to a minimum, under normal physiologic conditions, in both tension and compression cortices.

In the horse radius, differences in osteon density between tension and compression cortices appear to be most closely associated with differences in strain mode rather than strain magnitude. Thus, some aspect associated with compression loading may stimulate the formation of secondary osteons within the compression cortex. The results of this study may reflect the capacity of bone to process local and regional strain-related information in order to achieve functional adaptation to regionally nonuniform loading conditions. Further studies in other simple, physiologically unperturbed, tension/compression systems are warranted. Comparison of these results with those of other similar systems will help to ultimately understand how mechanical stimuli affect skeletal maintenance, repair, and anaptation.

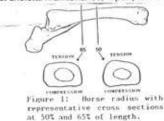


Table 1	ost/mm2		% Area		% Porosity	
Tension	mean	stds	mean	stds	mean	stds
Periosteal	8.6	(4.4)	23.8	(13.7)	3.3	(1.4)
Middle	9.5	(5.4)	25.4	(15.1)	4.1	(2.3)
Endosteal	10.1	(5.8)	31.4	(16.4)	5.7	(2.4)
Compressio	on					
Periosteal	20.1	(3.9)	56.4	(8.1)	3.7	(0.8)
Middle	18.9	(4.6)	57.0	(12.3)	4.3	(1.1)
Endosteal	15.3	(4.9)	47.6	(13.3)	6.7	(3.1)

% Area refers to fractional area of secondary bone

References 1) Rubin et al., 1990, J. Biomech. 23(Suppl. 1): 43-54. 2) Martin and Burr, Structure, 1989 Function and Adaptation of Compact Bone, Raven Press 3) Mori, Burr, 1993, Bone 14:103-109. 4) Skedros et al., 1993 ORS Abstract No. 533. 5) Schneider et al. 1982, Am. J. Vet. Res. 43(9): 1541-50. 6) Huiskes and Kulper, 1993 ORS Abstract No. 128.

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