Introduction: The effects of exercise on bone in general are well understood. However, the effects of exercise on subchondral bone have been rarely investigated. Since physical fitness is of growing importance, and since subchondral bone changes can often lead to the initiation and progression of osteoarthritis, it appears that characterizing the response of subchondral bone to exercise is of importance. The racehorse is a potential model for studying subchondral bone change in response to exercise. The distal third metacarpal condyles of the metacarpal bones of horses can fracture or secrete progressive subchondral bone sclerosis and necrosis. This occurs naturally and rapidly. The purpose of this study was to determine the effects of treadmill exercise on the subchondral bone of third metacarpal condyles of horses to characterize changes that might lead to disease.

Materials and Methods: Twelve horses were used in the study, six of which were randomly chosen to be exercised on a high-speed treadmill five days per week for six months. The institutional Animal Care and Use Committee approved all procedures. The exercise routine included 2 minutes at a trot (4-6 km/hr), 3 minutes at a gallop (10-13 km/hr) followed by 2 minutes at a trot. The remaining six horses served as handwalked controls. All horses were given calcine (20 mg/kg, IV) on days 81 and 91, and oxytetracycline (25 mg/kg, IV) on days 181 and 191 for labeling of active bone formation. All horses were also evaluated clinically at the end of the project, and then anesthetized for evaluation of subchondral bone blood flow. Twenty-five million red fluorescent microspheres were injected into the median artery as a reference sample was simultaneously withdrawn from the medial palmar digital artery. Subchondral bone samples were collected for histomorphometric, bone formation, microdamage, osteocyte viability and blood flow analyses. Subchondral bone plate (SCP) and trabecular bone (TB) areas were outlined on each section. Morphometry and microdamage were evaluated nondecalcified sections that were stained in 1% undecylated basic fuchsin. Bone area (B.Ar/T.Ar - %), vascular area (Vs.Ar/B.Ar - %), diffuse staining microdamage (Ddx.Ar %), microcracks (Cr.Dn - #/mm²), delamination cracks (D-Cr.Dn-#/mm²), and the length of each type of microcrack were determined. Nondecalcified sections were then viewed under UV light for measurement of the number and length of staining labels. Osteochondral sections were also evaluated for osteocyte viability by staining for lactate dehydrogenase activity. The numbers of viable and nonviable osteocytes were determined in each section. Blood flow in each osteochondral section was determined by counting microsphere in each section and normalizing to blood flow within the limb. Analysis of variance was used for determination of treatment on outcome, and P was set at 0.05.

Results: There was significantly more bone (B.Ar/T.Ar) within the SCP and TB areas of the third metacarpal condyles of exercised horses compared to control horses (P=0.0092 and P=0.0016, respectively). (Figure 1) Increased bone area was seen in the palmar aspect and the proximodorsal aspect of the condyle. (Figure 1) There was no significant difference in vascular area between exercised and control horses. There was a trend towards more bone formation in the SCP of exercised horses compared to controls (P=0.0100). There was a trend towards greater microdamage number and size in the SCP and TB of exercised horses compared to controls (P=0.06 and P=0.0993, respectively). There was a significantly lower percentage of viable osteocytes in the SCP of exercised horses (P=0.0215). There were no significant differences in blood flow between the two groups.

Discussion: The third metacarpal condyles of exercised horses showed a modeling response as demonstrated by increased bone area and active formation. The stresses placed on the palmar aspect of the condyle by the proximal sesamoid bones, and on the proximodorsal aspect of the condyle by the first phalanx, were enough to induce bone formation. Furthermore, an area of relatively low bone content occurred between the areas of increased bone content. This density gradient is in an area in which cartilage erosion and osteochondral fracture occur. There appeared to be no vascular response in the subchondral bone of the third metacarpal condyle, however, the method of measuring blood flow in this study may be too insensitive to detect differences. Increased vascularity associated with bone formation at this site may have occurred earlier in the study and was missed at the time of sample collection. Microdamage was not significantly different between treatment groups in this study, but it was evaluated at only one time point in the study. It may be that significant microdamage also occurred earlier in exercised horses, thus leading to the modeling response seen at the end of the study. Osteocyte viability was reduced in the subchondral bone of exercised horses. This may be a pathologic response, or a normal apoptotic response leading to stimulation of bone modeling.

In conclusion, treadmill exercise led to active bone formation in the palmar aspect to the third metacarpal condyles. The stimulus for this response may be mediated by microdamage and/or osteocyte viability. Regardless, the exercising horse represents an overload model in which osteochondral damage can be induced without surgical intervention. This model may help investigators identify the point at which normal bone adaptation becomes pathologic. This is important in order to identify the significance of clinical diagnostic tests such at CT, MRI and biochemical markers of joint disease.


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