Subchondral Bone Reaction to Exercise

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Treadmill exercise of horses caused lameness, gross cartilage lesions and reduced osteocyte viability in their carpal and metacarpophalangeal joints. However, exercise had variable effects on bone formation and microdamage. Treadmill exercise can cause osteochondral injury similar to that seen in racehorses. Authors' address: Equine Orthopaedic Research Laboratory, Depts. of Clinical Sciences and Pathology, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, CO 80523. © 1999 AAEP.

1. Introduction

High-speed exercise is the cause of several types of joint injuries in horses. Subchondral bone is responsible for shock absorption and maintenance of joint shape, and proper adaptation is needed to avoid fatigue-related injuries such as osteochondral fragmentation, fracture and sclerosis. Young et al showed adaptive responses (increased bone formation) in third carpal and proximal sesamoid bones of exercised horses. However, osteocyte viability and microdamage in subchondral bone have not been evaluated. Reduced osteocyte viability and increased microdamage have been found in hip osteoarthritis and fracture in humans. Therefore, the purpose of this study was to investigate the effects of exercise on subchondral bone of equine carpal and metacarpophalangeal joints.

2. Materials and Methods

Six 2-year-old horses (TE) exercised on a high-speed treadmill for 6 months, while 6 others served as hand-walked controls (HW). The TE horses trotted for 2 min (10–12 mph), galloped for 3 min (26 mph), and trotted for 2 min each day, 5 days per week for 6 mon. Horses received fluorescent bone labels at 3 and 6 mon. Horses were graded for lameness and gross joint lesions at the end of the study, and radial carpal bones (CR), third carpal bones (C3) and third metacarpal condyles (MC3) were collected for analysis of bone and vascular morphometry, bone formation, microdamage, osteocyte viability and blood flow (as measured by flow cytometry of fluorescent spheres). Analysis of Variance was used to compare means between horses and treatments, and significance was set at p < 0.05. All data in this abstract are reported as means.

3. Results

TE horses were significantly lamer than HW horses after carpal (HW grade = 0.25 vs. TE grade = 2.38) and digital (HW grade = 0.29 vs. TE grade = 2.67) flexion tests. TE horses also had significantly more gross damage in the intercarpal joints than HW horses (HW grade = 0.167 vs. TE grade = 4.25). These lesions consisted of osteochondral fragmentation, articular cartilage erosion, and wear line formation.

CR and MC3 bones of TE horses had significantly
more percent bone area than those of HW horses (CR: HW = 71.21% vs. TE = 82.71%; MC3: HW = 67.97% vs. TE = 80.42%). MC3 bones of TE horses had significantly more bone formation at the time of labeling than HW horses (HW = 2.51% vs. TE = 5.73%). There was a trend towards more diffuse microdamage in C3 bones (HW = 6.10% vs. TE = 10.76%) and MC3 bones (HW = 0.026% vs. TE = 0.57%) of TE horses compared to HW horses. There were significantly more nonviable osteocytes in the subchondral bone of CR bones (HW = 189.45/mm² vs. TE = 380.02/mm²), C3 bones (HW = 144.50/mm² vs. TE = 368.58/mm²) and MC3 bones (HW = 161.46/mm² vs. TE = 255.33/mm²) of TE horses compared to HW horses.

There were no significant differences in the blood flow and vascularity between treatment groups.

4. Discussion
This study shows that subchondral bone of different joints can respond differently to exercise. The third metacarpal condyle may have a lower threshold for adaptive response to exercise than the carpal bones, as seen by its increase in percent bone area and bone formation. Microdamage was seen in the third carpal bone, indicating that this level of exercise may also lead to damage at this site. Therefore, the bones within the carpal and metacarpophalangeal joints may each require a specific intensity and duration of exercise to reduce the chances of injury.

Osteocyte viability was reduced in all bones of exercised horses. The consequences of this are unknown, but theories suggest that reduction in osteocyte viability may either be a signal to induce bone remodeling, or may prevent the detection and consequent repair of microdamage.

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References