Practical Applications of an Instability Model of Equine Osteoarthritis

Emily J. Simmons, DVM; Alicia L. Bertone, DVM, PhD; and Steven E. Weisbrode, VMD, PhD

Disruptions of normal joint congruency (i.e., ligamentous injury, angular limb deformities) can lead to joint instability and laxity, altering normal wear of the articular cartilage and causing osteoarthritis to develop. We have established a clinically applicable model of osteoarthritis to evaluate the physiology and therapeutic modalities for this joint disorder. Authors’ address: Dept. of Clinical Sciences, The Ohio State University, 601 Vernon Tharp St., Columbus, OH 43210. © 1997 AAEP.

1. Introduction
Musculoskeletal disorders, including osteoarthritis (OA), are among the leading causes of morbidity in the equine racing industry. These injuries are related to excessive joint motion and excursion caused by uneven ground, overtraining, immaturity, poor conformation, or improper shoeing. The canine Pond–Nuki model, simulating anterior cruciate ligament rupture of the human athlete, has been widely used to mimic OA. No such instability model has been documented in the horse, despite the wide clinical occurrence of OA, particularly in equine athletes. Our purpose was to establish an equine instability model, similar to naturally occurring OA, that can be used in clinical physiologic and therapeutic studies.

2. Materials and Methods
Six mature (>3 years) horses, healthy and sound upon physical examination, lameness examination, joint circumference, range of motion, and radiographs (four standard views) of bilateral metacarpophalangeal (MCP) joints were used.

Instability was surgically induced by transection of the lateral collateral and lateral collateral sesamoidean ligaments in a MCP joint of each horse. Horses were walked on a treadmill for 2 weeks after surgery and then trotted for 6 weeks. Lameness examinations were performed every 7 days after surgery. Stress radiographs were taken immediately before, after, and 8 weeks following surgery. Synovial fluid was collected before surgery and at 8 weeks for analysis [white blood cell (WBC) and total protein (TP)]. Euthanasia was performed 8 weeks after surgery. MCP joints were opened and evaluated for evidence of osteophytes, score lines, and erosions. Cartilage was collected for histology and tissue culture. Synovial membrane was collected for histology (H&E).

3. Results
Lameness was significantly increased (mean = 1.6/5) over the 8-week period (p < 0.0001). Joint circumference was significantly greater in the OA (32.8 ± 1.4 cm) versus the contralateral joint (26.0 ± 0.8 cm);
Range of motion was significantly less (mean = 20°) in the OA joint (p < 0.0001). Stressed laxity measurements at 8 weeks were significantly greater in the OA (6.2 ± 0.7 mm) compared with the contralateral joints (2.08 ± 0.03 mm; p < 0.0001). Synovial fluid analysis (WBC and TP) showed no significant difference between presurgery (600 ± 124 cells/µl; 2.0 g/dl) and 8-week samples (267 ± 99 cells/µl; 2.0 g/dl). The number (mean = 2) and size (mean = 2.67 mm) of osteophytes on radiographs at 8 weeks were significantly greater for the OA joints (p < 0.0001). A gross evaluation showed the number of score lines/cm (mean = 2.8) and the number (mean = 2.6) and size (mean = 3 mm) of osteophytes to be significantly greater in the OA compared with the contralateral joints (p < 0.0001). Newly synthesized prostaglandin was significantly greater at 18 and 72 h of cartilage explant culture in the OA (133.3 ± 8.2 and 384.6 ± 155.4 cpm/µg protein) compared with the contralateral joints (59.0 ± 12.1 and 283.0 ± 83.9 cpm/µg protein, respectively; p < 0.0001). Articular cartilage from OA joints demonstrated fissures with adjacent chondrone formation and reduced interterritorial matrix staining.

4. Discussion

Our instability model established classic OA in the equine MCP joint with typical clinical signs of lameness, joint enlargement, and decreased range of joint motion. Articular cartilage degeneration was documented by the formation of osteophytes, score lines, and surface erosions. The consistency of clinical signs and gross lesions indicate the comparable nature of this model to naturally occurring disease and its usefulness for clinical trials. Minimal synovial membrane changes were produced, which is typical of naturally occurring OA.4,5 Pilot studies using this model suggest that OA joints have increased vascular tone as a result of increased arterial resistance that decreased the synovial fluid production. These data may alter the clinical pharmacologic actions of intra-articular medications. In conclusion, this equine instability model induced clinical and gross features of classic OA, providing a practical modality for future diagnostic, therapeutic, and physiologic trials.

References