Joint issues often contribute to attrition and performance and welfare issues in athletic horses. Even when horses recover from joint trauma such as infection or fracture, osteoarthritis (OA) most assuredly follows.

In this special report we’ll describe common joint issues that can negatively affect a horse’s musculoskeletal health, ultimately leading to OA. We’ll also share ways to prevent these conditions and minimize horses’ risk of lifelong joint swelling, pain, decreased range of motion, and loss of function, focusing on those backed by scientific data.

OSTEOCHONDRITIS DISSECONS (OCD)

Considered a developmental orthopedic disorder, OCD results from failure of endochondral ossification and is a primary cause of OA. Endochondral (occurring within cartilage) ossification is the process by which bone forms from cartilage early in a foal’s development.

In some cases endochondral ossification is disturbed, resulting in areas of malformed bone underlying the articular cartilage at the ends of long bones. Subchondral (beneath cartilage) bone cysts and fractures can occur that are covered by abnormally thick cartilage flaps.

These subchondral lesions usually lie dormant until the young horse begins training. At that time the cartilage flaps and abnormal subchondral bone can break free and float within the joint, causing trauma and inflammation. In addition to swelling, other signs of OCD include pain, lameness, and general poor performance.

Prevention An estimated 20-25% of newborn foals are at risk of developing some form of OCD (Bourebaba et al., 2019). Researchers believe diet, growth, hereditary factors, and trauma can all contribute. Nutritionally, foal diets should be designed to provide sufficient energy (calories) to support growth but limited easily digestible carbohydrates to regulate that growth. Mineral deficiencies such as low copper levels might contribute to OCD development.

“Other mineral deficiencies could contribute to OCD lesions, such as selenium,” says Mandy Peffers, BSc, MPhil, PhD, BVetMed, FRCVS, of the Institute of Life Course and Medical Sciences at the University of Liverpool, in the U.K. “Plus, some trace elements can interfere with uptake of important trace minerals like copper and selenium.”

While OCD has a genetic component, Peffers says heritability is likely poor. Nonetheless, if a foal has OCD, then breeders might consider avoiding repeat matings of its sire and dam.

POST-INJECTION JOINT FLARE/SEPSIS

Many athletic or arthritic horses receive intra-articular injections of anti-inflammatory medications such as triamcinolone acetonide (a corticosteroid), hyaluronic acid, or regenerative therapies such as stem cells and platelet-rich plasma. Entering a joint is never without risk of causing either a “flare” or an infection (sepsis).

Johnston et al. (2020) defined joint flares as inflammatory reactions characterized by transient synovial effusion (joint swelling) and lameness, ranging from mild to severe. In turn, sepsis results from bacterial inoculation and colonization of a synovial cavity (Byrne et al., 2020).

“In either case, inflammatory and degenerative cytokine mediators are stimulated, which can result in substantial irreversible damage to the synovium and articular cartilage,” says Peffers. “In fact, studies show that steroids (some more than others) can reduce chondrocyte proliferation (cartilage cell growth).”

Prevention “Proper joint preparation will help minimize joint infections,” says Peffers. “This could include clipping hair very short, scrubbing for five minutes with an appropriate reagent, applying an ethanol spritz, then using surgical gloves and sterile techniques.”

As an added layer of protection, some veterinarians elect to administer an antibiotic, most frequently amikacin, at the same time as the intra-articular medication. Considering the risk of joint sepsis is reportedly as low as 0.1% (Pezzanite 2021) and amikacin is potentially toxic to cartilage cells, the practice of rote intra-articular antibiotic administration is questionable.

OSTEOARTHRITIS (OA)

Even if a horse doesn’t have a history of OCD, joint infection, or fracture, any condition inciting inflammation can culminate in OA.

“Once considered a disease restricted to articular cartilage, we now know that OA is actually a ‘whole joint disease’ that may include changes to cartilage, underlying subchondral bone, the lining (synovium), joint capsule, and associated ligaments,” Peffers says.

“Additionally, we believe aging—when there is a loss of muscle—also contributes to OA. In complex joints such as the stifle there are other tissues also involved, including the meniscus and cruciate ligaments.”

Once any joint tissue becomes inflamed, a cascade of events ensues...
that results in cartilage degeneration—specifically, cartilage destruction manifesting as the loss of type II collagen and the extracellular matrix, including proteoglycans.

**Prevention** There is no cure for OA, which is why preventive measures to protect joints against inflammation are so important.

Peffers et al. performed a 2021 review of a decade’s worth of data on measures for maintaining distal (lower) limb functionality, which includes preventing OA. Based on their findings, they recommend:

**Hoof trimming** Peffers says correcting impaired conformation and reducing excess load on structures supporting joints in the distal limbs with routine (every four to six weeks) farrier appointments is critical. Farriers should consider factors such as breakover, initial contact, and center of force during the stance phase of locomotion, among others.

Prophylactic shoeing might also play a role in preventive joint care. Research in this area, however, is sparse, leaving voids in our knowledge about optimal types of shoeing (e.g., egg bar, toe wedges, heel wedges, etc.).

**Nutritional supplements** These dietary products are often administered once joint trauma has already occurred and the horse is showing signs of OA. Yamada et al. (2022) demonstrated that supplementing horses that had experimentally induced OA in the fetlock joint “may not be entirely effective to change the catabolic process in articular cartilage and progressive induced chondral damage.”

Some researchers suggest prophylactic administration might be of more benefit. “Because loss of extracellular matrix is a feature of cartilage degeneration, supplements that replace components of the extracellular matrix, such as glucosamine and chondroitin sulfate, are believed to be chondroprotective,” says Peffers.

In a 2017 study Van de Water et al. demonstrated the preventive effect of an oral joint health supplement containing glucosamine, chondroitin sulfate, hyaluronic acid, and other ingredients. It had anti-inflammatory effects, with PGE2 (a potent inflammatory mediator) levels lower in synovial fluid samples collected from treated horses than controls. “Hyaluronic acid suppresses inflammatory responses mediated through receptor-binding relationships with a number of molecules,” says Peffers. “Higher molecular weight hyaluronic acid promotes anti-inflammatory responses, but short hyaluronic acid oligosaccharides produce inflammatory reactions.”

**Exercise** Routine exercise might help prevent worsening OA. “In older horses, the ability to move helps reduce joint stiffness,” says Peffers. “These horses do better living outside with access to grass and opportunities to show natural behavior. We also know that joint loading is important for homeostasis, but it needs to be the correct type.”

**MORE RESEARCH NEEDED**

Despite the routine use of these preventive measures, evidence based on controlled clinical trials is lacking. “More research is needed, particularly well-powered, longitudinal studies, to make informed scientific conclusions regarding single and multimodal strategies applicable to clinical practice,” says Peffers.