

Down on the Farm

Focus on Physisitis

The good news is this skeletal disorder of growing foals is almost always self-resolving

by **KAREN BRIGGS**

Developmental orthopaedic disease (DOD) remains one of the top concerns of breeders worldwide. A series of related syndromes, rather than a single condition, DOD encompasses anything that contributes to poor skeletal development in foals—angular limb deformities, osteochondrosis, osteochondritis dessicans (OCD), contracted tendons, cervical malformations, subchondral bone cysts, club foot and physisitis, among them.

Physisitis (fie-SITE-is), sometimes called physal dysplasia, is one of the many manifestations of DOD. Essentially, it's an inflammation of the growth plates in the long bones of a growing foal. Physisitis was formerly known as epiphysitis—which most horsemen had just learned to pronounce when the terminology was changed! Dr. C. Wayne McIlwraith, BVSc, Ph.D., of Colorado State University, one of the United States' leading orthopedic researchers, explains, "(Epiphysitis) was not an accurate description of where the problem was—the epiphysis is the

site of secondary ossification in a bone, while the physis is the actual growth plate."

When we focus in on physisitis, it's helpful first of all to understand how bones grow and develop in a foal.

Normal Bone Development

Development of the skeleton of a foal begins in the first month of gestation. A miniature 'framework' of cartilage is formed initially and, as the fetus grows, the cartilage undergoes a gradual and progressive change into bone—a process called endochondral ossification.

The first area of the fetal cartilage to ossify (be replaced by bone) is the central shaft, or diaphysis, of the long bones of the limbs. Secondary ossification follows at the epiphyses near each end of the bone. By the time the foal is born, almost all of the original cartilage has been transformed into bone, leaving 'growth cartilage' at only two sites—the growth plate or physis and a layer between the articular (joint) cartilage and the bone of the epiphysis. It's from the first of these two sites, the growth plate, that the bone grows in length as the foal matures—and it's from here that

growth plate abnormalities, or physisitis, arise.

After birth, the foal's bones continue to lengthen and develop for at least another 18 months. The growth plates produce chondrocytes (cartilage cells) in the layer nearest the epiphysis. As they multiply, these cells are pushed towards the main shaft of the bone by the next generation of dividing cells, and they gradually enlarge and assemble into columns. Calcium and phosphate (and a few other trace minerals) are deposited in the matrix between the cell columns, and the cell columns which remain between the lines of newly calcified cartilage matrix are then invaded by blood vessels. Over time, the mixture of calcified cartilage and immature bone is replaced by mature bone, and the limb lengthens as the foal grows. When maturity is reached and bone growth ceases, the horse is left with cartilage only at the articular surfaces of the joints, and in a few select areas such as the muzzle and ears.

The growth process is such that the rate of production of cartilage cells at the epiphyseal end of the growth plate is almost exactly matched by the rate

at which cartilage is replaced by bone at the shaft end. Or, at least, that's how it works when everything goes according to plan.

Where It Goes Awry

Osteochondrosis is the term used to describe a bone maturation process gone wrong. In osteochondrosis, the cartilage progresses through all the normal stages to begin with—but then calcification doesn't occur, blood vessels fail to invade, the normal columnar bone structure fails to organize, and the cartilage thickens and projects into the bone of the epiphysis or the diaphysis.

When osteochondrosis affects the growth of the joint surface cartilage at the ends of the bones, a piece of the thickened, abnormal cartilage sometimes separates from the bone. It may remain loosely attached to the articular surface, or it may break away completely and float freely in the joint capsule. Either way, it causes intense irritation to the joint, pain, and lameness. This is what veterinarians call osteochondritis desiccans, or OCD.

Physisitis, on the other hand, is more subtle, because it occurs in the growth plate or physis. Foals with physisitis have unusually 'knobby' joints (particularly the knees and/or fetlocks), and enlarged growth plates may give the knee a characteristic 'dished-in' appearance and fetlocks an hourglass shape. On x-rays, the growth plates appear widened, asymmetric, flaring or corroded.

Physisitis affects different joints at different ages. Growth plate abnormalities in the cannon bone usually surface between four and nine months of age, while in the upper fore- and hind-leg (the radius and tibia) they are more likely to appear in yearlings. There is often detectable heat in the area, with the youngster resisting palpation, and there may or may not be lameness. Sometimes, the only obvious sign is that the foal does not play actively with the herd.

It's estimated that between 73 and 88 percent of developmental bone disorders of growing horses are flexural deformities (such as contracted tendons) and physisitis. No one factor—nutrition, genetics, environment or bad karma—can create DOD on its own,

and prevention is proving a challenge. Still, researchers are making progress; here's some of what we do know.

Cause and Effect

GENETIC PREDISPOSITION: Radiographic studies have demonstrated that certain stallions produce foals with a higher frequency of OCD, compared with other stallions of the same breeds—sometimes even when the stallion himself shows no signs of the condition. Anecdotal evidence in North America seems to indicate a higher incidence of physisitis, particularly in heavy-bodied breeds such as Quarter Horses and Paints. While it seems probable that heredity plays a role in physisitis, it's not likely that anyone will be able to develop a screening program for stallions or mares to prevent the condition from showing up in their foals.

MECHANICAL STRESS AND TRAUMA: Injuries to the growth plates and/or articular cartilage can result from the sort of rough play that foals frequently engage in, and clinical signs of OCD (when flaps or fragments of cartilage separate from the bone) can arise from these injuries. It's thought that shear forces may disrupt capillaries in the bone and give rise to cartilage cell damage. How common this is, however, no one is sure.

GROWTH AND BODY SIZE: Numerous studies of both Thoroughbreds and Standardbreds have suggested that major growth spurts have been associated with a high incidence of OCD lesions. In Sweden, a higher incidence of osteochondrosis of the hock joint was found to be linked to foals who had a larger than average birth weight, large skeletal frame, and demonstrated a higher average daily weight gain.

Sudden growth spurts, which can occur when a foal is weaned and placed on a grain ration, are suspected of having a large role to play in the development of physisitis. Many researchers recommend feeding a creep ration before the foal is weaned, so as to accustom him to carbohydrate intake (as well as compensate for the declining quality of his dam's milk) and avoid a sudden growth spurt at weaning time.

EXCESS ENERGY: Overnutrition as a cause of physisitis was originally

extrapolated from work in dogs and pigs. Though excesses of protein have often been blamed for high incidences of DOD, the more likely culprit appears to be excesses of digestible energy (DE), especially when coupled with restricted exercise. An Australian study demonstrated that high energy diets (120 percent of National Research Council recommendations) consistently produced lesions in weanlings.

Excess digestible energy almost invariably comes from the feeding of grains and concentrates, rather than hay. Nutritionist Sarah Ralston, VMD, Ph.D., of Rutgers University, notes that when hay is digested, it is broken down into fatty acids, while grain is broken down to its component sugars. Both of these can yield energy, but sugars influence the balance of insulin and the thyroid hormone, thyroxin, in the body—excess thyroxin production is thought to increase the risk of OCD. Thus, the more energy provided by grain and the less provided by forage, the more likely it is that OCD will result.

MINERAL IMBALANCES: Calcium and phosphorus are integral to bone development. Both the levels of these two minerals in the diet, and the ratio of one to the other, is important. Current recommendations for growing horses are a Ca:P ratio of 1.8:1—though anywhere between 1.2:1 and 2.5:1 is considered okay. High phosphorus levels (those where there is more phosphorus than calcium) have been implicated in OCD lesions, and one study did consistently produce lesions in young foals fed five times the recommended level of phosphorus. Foals fed three times the recommended levels of calcium, however, didn't develop lesions.

Mineral intake is an area where oversupplementation is common. Many people operate under the "if some is good, more is better" principle! Calcium is commonly oversupplemented in horses, and while it by itself may not produce DOD, it can interfere with the absorption of zinc, copper, manganese and iron—all trace minerals which have been sus-

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pected of having a role to play in DOD.

Copper, a component of normal bone collagen and elastin synthesis (the latticework of bone formation), is a mineral which has gotten a lot of attention in the study of DOD. In one study, an artificially-induced copper-deficient diet (1.7 parts per million) did produce OCD-like lesions and flexural deformities in foals. Other studies have also produced a higher incidence of lesions in foals with copper-deficient diets—but it should be noted that the majority of those lesions turned up in the spine, not in the limbs. Unfortunately, higher levels of copper intake have not been shown to completely prevent OCD. Copper supplementation may help (most researchers now recommend an intake of 25 mg/kg or higher), but it will not, by itself, eliminate the problem.

Zinc, a mineral which competes for the same transport mechanisms as copper, has also been implicated in DOD. Excessive zinc intake, as was noted in one study where foals were raised near a zinc smelter, can cause DOD lesions. Currently, the NCR recommends a copper to zinc ratio of about 1:4, so if copper levels are raised in the diet, zinc

should be as well. Many nutritionists now recommend feeding broodmares 25-30 mg/kg of copper and 65 mg/kg of zinc during late gestation and lactation.

ENDOCRINE FACTORS: Stressed foals who have consistently high levels of cortisol in their systems may also be at higher risk for DOD. McIlwraith notes that cortisol inhibits ossification, and that long-term use of corticosteroids can have a similar effect.

Prognosis of Phytitis

Most researchers agree that phytitis, by itself, tends to resolve on its own. Unless there are complicating factors, such as cystic lesions in the bone, it should gradually disappear as growth slows and the physes close.

"Phytitis is definitely one of the less severe forms of DOD," says McIlwraith. "It can sometimes be a warning sign of other, more serious manifestations of DOD cropping up, but it's not necessarily tied in with other DOD problems."

What to do when a foal develops phytitis? In most cases, says McIlwraith, nothing. "It all depends on how bad it is, of course. If there's swelling at the growth plates, but no detectable lameness, it will resolve on its own as the foal grows. If the problem is more severe, and the horse is lame, then you have to consider restricting exercise somewhat. But because too much confinement can predispose the horse to more severe problems, such as contracted tendons or flexural limb deformities, you have to balance confinement with controlled exercise—sometimes alternating stall rest with exercise in a small paddock."

McIlwraith points out that if a foal is sore, then phytitis is probably not the only problem he has—generally, at that point, a veterinarian should examine the animal for evidence of OCD and subchondral bone cysts.

In any case where phytitis is diagnosed, radiographic monitoring is a good idea—both to determine how well the abnormalities are resolving, and to detect any other DOD conditions that may be associated with it. Some forms of DOD can, if necessary, be treated surgically, but phytitis by itself is best treated by a correctly balanced diet, good management and time.

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