

## Review of “Developmental Orthopedic Disease (DOD) in Horses-A Multifactorial Process”

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In a talk presented at the seventeenth meeting of the Equine Nutrition and Physiology Society, Dr. Wayne McIlwraith defined various types of developmental orthopedic disease (DOD). He summarized current knowledge about **osteochondrosis**, one type of DOD, and reviewed what is known about the **causes of this problem**.

### What is osteochondrosis?

Osteochondrosis refers to a **defect that occurs during the process by which cartilage matures into bone**. The condition can lead to mild or severe lameness in fetlock, hock, shoulder, and stifle joints. Use of this and other terms has been somewhat undefined, but McIlwraith suggests the following clarification:

- **Osteochondrosis is the condition** in which there is a problem with cellular differentiation in growing cartilage, causing failure of some cartilage cells to ossify. Affected areas may develop weak, rough, thickened, separated, or fissured cartilage.
- **Osteochondritis is the inflammation** that results from these malformations and necrosis (tissue death) at the site of the defective cartilage.
- **Osteochondritis dissecans (OCD)** is the term used when the weak cartilage is crushed or cut (dissected). **Fissures and flaps may develop or small pieces of cartilage may break away, possibly disrupting joint function.**

The author points out that, while osteochondrosis has sometimes been used as a synonym for developmental orthopedic disease, the terms do not have the same meaning, since not all types of DOD stem from the cartilage maturation defects inherent in osteochondrosis.

### What causes osteochondrosis?

The only certain answer to this question, according to McIlwraith, is that **several factors are probably involved, and no one has a full understanding of the importance of, or interactions between, the various considerations**. Ongoing research is uncovering important facts, but a complete explanation has not yet been formulated. The following factors have been found to influence the development and severity of osteochondrosis in young horses.

- **Genetic predisposition.** Various studies have indicated that horses can inherit a tendency to develop osteochondrosis. However, some affected stallions and mares have no offspring with defects, and some horses without osteochondrosis have had defects in up to 40% of their offspring. Because of difficulties in interpreting research results, and because there are few data on the “normal” incidence of osteochondrosis, breeding recommendations cannot be based strictly on bloodlines.

- **Growth and body size.** The author says research has supported the common belief that osteochondrosis is found most often in large-framed and rapidly growing foals. Growth defects may occur as early as the first three months of life when growth is most rapid, although lameness is usually not noticed until a later age.
- **Mechanical stress and trauma.** According to McIlwraith, there is general agreement that mechanical stress and trauma can cause defective cartilage to tear or produce loose pieces. There is less understanding as to whether trauma and stress can be the initial cause of the defects. One school of thought is that imperfect areas of cartilage develop fairly commonly for unknown reasons, and inflammation and lameness are seen only when these defective areas are further damaged by stress. There may also be connections between cartilage defects and imperfect conformation. For instance, an upright and toed-out limb structure leads to greater strain on leg joints, possibly leading to cartilage damage or aggravation of existing lesions.
- **Defects in blood supply.** One theory reported by McIlwraith is that an injury or trauma may destroy the blood vessels supplying an area of developing cartilage, leading to tissue death. If this occurs early in the foal's life, it could conceivably lead to more serious defects than in an older horse where skeletal formation was more nearly complete.
- **Nutrition.** Studies of other species have linked osteochondrosis to overnutrition, and the same seems to be true for horses. In a study of weanlings, one group was fed 128% of the NRC requirement for digestible energy, another group was fed 126% of the NRC requirement for protein, and a third group received 100% of the NRC requirement for protein and digestible energy. McIlwraith states, "The number and severity of lesions found at post-mortem were much greater in the high digestible energy group than the control and high crude protein groups. The incidence of lesions in the foals fed high crude protein was not significantly different from that in the control foals." Although high levels of protein are often blamed for developmental problems, this study did not support that notion.
- **Mineral imbalances.** This paper mentions work done to establish the influence of calcium, phosphorus, copper, and zinc levels on osteochondrosis. Although some studies have been inconclusive, there is evidence that dietary copper deficiency may lead to an increase in osteochondrosis. Copper supplementation greatly reduced but did not eliminate all cartilage defects in foals. McIlwraith includes a warning that, while copper intake is important, overemphasis on the influence of any one mineral should not overshadow the importance of balanced nutritional management. Zinc, he says, appears to be the main copper antagonist in the equine, and he mentions that zinc:copper ratios of 4:1 or 5:1 are recommended for all horse breeds.
- **Endocrine factors.** Complex interactions exist among calcium availability, blood vessel penetration of cartilage, glucose/insulin response to feeding, and thyroid and parathyroid hormones. A recent study in Kentucky showed a high incidence of OCD on a farm where the feeding program produced a high glycemic response.
- **Exercise.** McIlwraith reviews a study in which foals were stalled with no exercise, stalled with increasing amounts of exercise, or left at pasture. All foals had osteochondrosis at 5 months. Number of lesions was the same for all treatments but severity tended to be worse in stalled foals. From 5 months to 11 months all horses were given light exercise. At the end of this period fewer lesions were evident, indicating that some lesions regress with time. It was concluded that level of exercise may influence the distribution and severity of lesions but may not play a causative role.

**Osteochondrosis** is a complex problem that has a **significant impact on the horse industry. It is not completely understood at this time**, and research continues into the many factors and interactions that influence this disease.