The Pathogenesis of Osteochondrosis

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The osteochondroses are a heterogeneous group of lesions occurring in growth (epiphyseal) cartilage of immature animals and are characterized by focal or multifocal delay in endochondral ossification, which is the process by which growth cartilage is converted to bone. The sites of occurrence of these lesions include the growth plate (physis) and the epiphyseal cartilage of the articular-epiphyseal cartilage complex (AE complex). The lesions are common, often occur in bilaterally symmetrical sites, and represent an important orthopedic entity that has a number of different clinical manifestations in pigs, dogs, horses, cattle, and poultry. The hallmark of the uncomplicated gross lesion of osteochondrosis is focal or multifocal retention of growth cartilage due to its failure to become mineralized and replaced by bone by endochondral ossification. In the growth plate, this is due to an accumulation of viable hypertrophic chondrocytes, whereas in the AE complex, it is due to necrosis of epiphyseal cartilage.

The etiology of osteochondrosis appears to be multifactorial, with genetics, rapid growth rate, vascular factors, and trauma all being implicated. Trauma is the most widely proposed etiology in both humans and animals; however, its most likely role is as a final insult to compromised epiphyseal cartilage rather than an as initiating factor in the development of early lesions. Rapid growth and nutritional factors also are cited. Although osteochondrosis occurs in all species during the period of rapid growth and is most common in species that grow rapidly, most experimental work evaluating these has failed to document rapid growth as a cause of the disease. In addition, little evidence is available to indicate that the lesions of osteochondrosis result from a specific nutritional deficiency.

Recent experimental work in pigs and horses has demonstrated that a defect in vascular supply to growth cartilage, resulting in localized areas of ischemic necrosis, is important in the initiation of preclinical lesions. Although articular cartilage is avascular throughout life, the subarticular epiphyseal cartilage of the AE complex and the

epiphyseal cartilage of the growth plate both are highly vascularized tissues and are dependent on a vascular supply for viability. The blood vessels supplying growth cartilage run in channels that are termed cartilage canals. During growth, the epiphyseal cartilage gradually becomes reduced in volume and requires the nutritional support of fewer vascular channels, until it becomes entirely avascular and eventually is completely replaced by bone. It is during the time frame in which the epiphyseal cartilage is supplied by blood vessels that subclinical lesions of osteochondrosis occur. This time frame varies based on site, age, and species, which likely explains, along with biomechanical factors, why different species have different lesion predilection sites. Although the nature of the insult to the vessel(s) is unclear, the result is a well-demarcated area of necrosis of epiphyseal cartilage that is centered on necrotic blood vessels, is visible only microscopically, and is termed *osteochondrosis latens*. When the ossification front reaches the area of necrosis, failure of endochondral ossification occurs, resulting in an area of retained necrotic epiphyseal cartilage that is visible grossly. The lesion at this stage is termed *osteochondrosis manifesta*. This lesion is highly vulnerable to traumatic clefting through the area of necrosis, termed *osteochondrosis dissecans*, even with physiologic pressure. Once clefting occurs and subchondral bone is exposed, the lesion is painful and results in clinical signs of lameness. If cleft formation (the most common sequel) does not occur, necrotic epiphyseal cartilage becomes surrounded by subchondral bone, gradually diminishes in size, and eventually becomes completely replaced by bone. Predilection sites for both clinical and subclinical lesions in swine include the AE complex of the distal femur (especially the medial femoral condyle) and humerus (condyles and head).

Osteochondrosis dissecans (OCD) is the name given to lesions of osteochondrosis at the AE complex that form clefts in the necrotic cartilage with subsequent fracture of the overlying articular cartilage. OCD can be accompanied by pain, joint effusion, and nonspecific secondary lymphoplasmacytic synovitis. Free-floating cartilage fragments mice occasionally interfere with mechanical movement of the joint. The disease is extremely common in young breeding pigs and is a significant cause of lameness in this species. The articular cartilage defect in OCD has poor healing capabilities, and such joints commonly develop some degree of degenerative joint disease.

Epiphysiolysis is the separation of the epiphysis from the metaphysis due to the formation of a horizontal fissure through an abnormal physis. . The condition is most common in pigs and dogs. In market-weight pigs and in young gilts, the femoral head may be involved whereas, in sows, separation of the ischial tuberosity (site of insertion of semimembranosis, semitendonosis, and biceps femoris muscles) at its growth plate (often bilaterally) is a common cause of caudal weakness and inability to stand and often occurs at parturition. Because these lesions have primarily been studied in their chronic stages, the nature of the initial lesion is unclear; however, it is strongly suspected that it occurs secondary to an extensive area, or to multifocal coalescing areas, of physeal osteochondrosis.

Although lesions of osteochondrosis form during development in juvenile animals, recent work supported by Zinpro Corporation has demonstrated that forelimb lesions that are considered to be severe enough to contribute to clinical lameness are present in high producing sows across multiple parities. Strategies to reduce the prevalence of osteochondrosis include exclusion of animals with joint lesions from the breeding program and minimizing physical stress that occurs during transportation or under housing conditions of overcrowding and/or hard flooring.