Osteochondrosis is a common and important joint disorder that occurs in human beings and multiple animal species, particularly pigs, horses and dogs. It is defined as a focal disturbance of endochondral ossification and has a multifactorial aetiology, with no single factor accounting for all aspects of the disease. In human medicine osteochondrosis has been defined as an idiopathic condition characterized by disorderliness of endochondral ossification (Ytrehus, Carlson, & Ekman, 2007).

Although formation of fragile cartilage, failure of chondrocyte differentiation, subchondral bone necrosis, and failure of blood supply to the growth cartilage, all have been proposed as an initial step in the pathogenesis, the recent literature strongly supports failure of blood supply to growth cartilage as being the most likely. Localised failure of endochondral ossification ensues, which typically results in necrosis of affected cartilage. It can involve the physis (growth plate) or the articular-epiphyseal cartilage (Hill, Sutton, & Thompson 1998; Trostel, McLaughlin, & Pool 2002). In pigs the lesion is slightly different in that there is no evidence of necrosis of cartilage, but rather a focal failure of mineralisation of hypertrophic cartilage cells (Wooder 1997). A number of possible aetiologies and predisposing factors such as over nutrition, rapid growth, genetics [experimentally proven in pigs] (Hittmeier, Grapes, Lensing, Rothschild & Stahl, 2006), ischaemia, excess dietary calcium, hormonal influences, and trauma have been proposed (Trostel et al., 2002; Wooder 1997). In dogs osteochondrosis lesions are typically bilateral. Mild trauma to the necrotic cartilage may result in cleft formation of the affected cartilage with release of cartilage degeneration products into the joint causing a synovitis evident clinically as joint effusion and pain with lameness (Trostel et al., 2002).

Reports of osteochondrosis in cattle are infrequent, and most reported cases are associated with feedlot cattle raised under intensive conditions where increased growth rate is thought to play a major role in the aetiology. There have, however, been isolated reports of osteochondrosis of beef cattle kept on extensive farming systems in Australia. It was surmised that these cases
were a result of a genetically inherited disease (Hill et al., 1998).

Davies & Munro (1999) described an outbreak of osteochondrosis in beef bulls following failure to provide dietary mineral and vitamin supplementation. The bulls, varying from 4 to 14 months of age, were bedded on straw and divided into 8 groups of approximately 20 months, depending on age. Calves were either purchased at one week of age or as weaned calves and were introduced to the fattening ration when they were approximately 10 weeks old. Approximately two months before the start of the lameness problem, unknown to the owner, the mineral, vitamin, and avoparcin additive had not been included in the protein mix and this omission only became apparent during the investigation. Many of the animals appeared stiff and several showed obvious swelling of the hock joint. Two bulls were submitted for post mortem examination approximately 4 to 6 weeks after the problem was first recognized. They weighed 195kg to 212kg and were approximately 6 months old. Lesions were limited to the hock joints that were bilaterally distended by a large excess of straw amber colored synovial fluid. Approximately 2 cm by 3 cm of the articular cartilage of the lateral ridge of the tibial tarsal bone was ulcerated with shallow cavitation of the underlying bone. Several fissures up to 0.5 cm long were evident in the cartilage of the articular surface of the distal tibia. The joint capsule was thickened and new bone growth was evident around the joint margins.

Analysis of the metacarpal bone from the two bulls revealed adequate magnesium, phosphorus, and bone ash, but a slightly low calcium concentration. The vitamin A concentration was also low. Dietary analysis suggested inadequate calcium, sodium and copper intake and light deficiency of vitamin A, D and E. A balanced mineral and vitamin supplement was added to the diet when it became clear that the supplement had been omitted. A gradual clinical improvement was seen in the majority of the animals and after two to three weeks, the owner noted that growth rate and coat quality had significantly improved. However, 16 out of 76 of the 4-7 month old animals and two of 74 of the older animals were culled because of severe lameness problems. Blood samples collected from six animals, two days after introduction of a balanced mineral and vitamin supplement, revealed a calcium concentration within the reference range in five of the bulls. This outbreak provides evidence that mineral and vitamin imbalance is a likely contributing factor to the development of osteochondrosis in growing cattle.

Calcium deficiency with distorted calcium-phosphate ratio caused a severe outbreak of osteoarthritis in fattening bulls. Calcium deficiency caused more serious lesions in age groups 5 – 8 months than in age groups 12 – 18 months. Osteoarthritis lesions occurred in more than 80% of the animals with a calcium deficient diet (Heinola, Jikola, Nåkki & Sukura, 2006).

ENDOCONDORAL OSSIFICATION

There are two regions of specialized growth cartilage that are present at the ends of long bones during the period of skeletal growth and development. The growth plate is present on either side of the primary centre of ossification and is responsible for longitudinal growth. The growth cartilages are replaced by bone from both sides through a sequential process of cell proliferation, extra-cellular matrix synthesis, cellular hypertrophy, matrix mineralization, and vascular invasion that is termed endochondral ossification. The advantage of this process is that growth elongation is achieved by continuous addition of cartilage and subsequent replacement by bone in such a way that the individual is able to bear weight while growing. Vascular endothelial growth factor (VEGF), which is produced by hypertrophic chondrocytes, appears to be the factor responsible and necessary for vascular in growth into the growth plate, which is required for endochondral ossification (Gerber, Vu, Ryan, Kowalski, Werb, and Ferrara, 1999). The rate at which differentiation and growth of cartilage progresses, the direction of the process in three-dimensional space, and the time at which the process terminates are thought to be affected by genetic, nutritional, metabolic and mechanical factors (Ytrehus et al., 2007).

Although the epiphyseal growth cartilage matrix is macroscopically indistinguishable from the matrix of the overlying articular cartilage, it is distinguished histologically by the presence of vessels that invade
the cartilage from the surrounding perichondral plexus and run within channels termed cartilage canals, whereas articular cartilage is avascular (Shapiro, 1998). The functions of the cartilage canals are not fully understood, but they are suggested to serve three purposes: (1) to nourish chondrocytes beyond the reach of diffusive nutrients from the synovial fluid, (2) to play a role in the formation and maintenance of the secondary ossification center, and (3) to supply both cartilage and bone with mesenchymal stem cells.

As the individual grows, the growth rate of the cartilage diminishes compared with the rate of progression of the ossification front, causing the ossification front to advance toward the articular cartilage resulting in the layer of growth cartilage becoming progressively thinner. Eventually the growth cartilage is replaced by bone, and no cartilage canals remain.

**MANIFESTATIONS OF OSTEOCHONDROSIS**

The principal lesion of both articular and physeal osteochondrosis is a focal failure of endochondral ossification; that is, an area of growth cartilage fails to undergo matrix calcification or vascular invasion, and therefore does not become converted to bone. This is grossly visible in cut sections as a locally extensive zone of thickened cartilage accompanied by an irregular/uneven chondro-osseous junction.

Prior to the point of the disease at which a focal failure of endochondral ossification occurs and is grossly visible, the lesion in osteochondrosis is characterized histologically by a focal area of necrosis that is confined to the growth cartilage and involves neither the overlying articular cartilage nor the underlying subchondral bone. Lesions may develop in several locations in a single animal and often occur in bilaterally symmetrical sites. The primary changes of osteochondrosis in pigs are commonly observed by the time animals reach an age of two months and have been described as early as two weeks after birth (Hill, Kincaid, & Visco, 1990).

As early as 1978 Olsson & Reiland suggested that the pathophysiology of osteochondrosis is essentially the same in all species, including humans (Olsson, & Reiland, 1987).

In birds it was experimentally demonstrated that apoptosis of the endothelium lining cartilage canal vessels and, subsequently, the surrounding chondrocytes, initiates lesions of tibial dyschondroplasia, a disease that has many morphologic similarities to osteochondrosis in mammals.

**PROPOSED AETIOLOGIES OF OSTEOCHONDROSIS**

Osteochondrosis is regarded as having multifactorial aetiology, with no single factor accounting for all aspects of the disease (Schenck, & Goodnight, 1996). Suggested factors that play a role include rapid growth, heredity, anatomic characteristics, trauma, dietary factors, and a defect in vascular supply to epiphyseal cartilage.

**Rapid Growth**

Some large population surveys have revealed a positive genetic correlation between rapid growth and prevalence of osteochondrosis (Jørgensen & Andersen, 2000.)

**Heredity**

Differences in the prevalence of osteochondrosis between different breeds and different breeding lines of pigs, dogs, and horses strongly indicate that there is a heritable component to osteochondrosis. Many authors have suggested that the inherited factor is closely associated with a predisposition toward increased growth rate. In all species that have been examined there is compelling evidence that inheritance is important in the etiology of osteochondrosis.

**Anatomic Characteristics**

It was concluded that in the human knee the anatomic form and function of the joint determine the frequency of occurrence of osteochondritis (osteocondrosis) dissecan (Smillie, 1960). Based on necropsy findings, it was hypothesized that local traumatic factors, which may vary among joints, are important in the etiology of osteochondrosis in dogs (Olsson, 1987).

**Trauma**

Trauma has been one of the most widely proposed causes of osteochondrosis in all species (Ekman & Carlson, 1998). In support of a traumatic aetiology, predilection sites in all affected species tend to be located in areas of local biomechanical stress.
The role of trauma in the pathogenesis of osteochondrosis may depend on the stage of the disease that is considered. Although trauma may be involved in converting a subclinical osteochondrosis lesion to an osteochondrosis disseicans lesion, the severity of the trauma that is necessary usually is minimal and often includes only the forces involved in normal ambulation. There is, however, no clear evidence that acute macro trauma is involved in the initiation of primary lesions of osteochondrosis.

**Dietary Factors**

An imbalance in calcium-phosphorus supplementation, copper deficiency, zinc over supplementation, vitamin C deficiency, vitamin A deficiency, vitamin D deficiency, biotin deficiency, and chronic metabolic acidosis have all been suggested and investigated as aetiological factors of osteochondrosis (Nakano, Brennan & Aherne, 1987).

A lesion termed articular osteochondrosis has been associated with copper deficiency in several species, including deer, bison, and horses. The lesions are caused either by a primary copper deficiency or by exposure to factors that inhibit copper absorption or metabolism [e.g. zinc, cadmium, or inorganic sulphates] (Hurtig, Green, Dobson, Mikuni-Takagaki, & Choi, 1993). Hurtig et al. suggested a relationship between low copper intake in fast-growing horses, inferior collagen quality, biomechanically weak cartilage and bone, and lesions of osteochondrosis disseicans.

An outbreak of congenital skeletal defects in Holstein calves was associated with a manganese deficiency. Marginally, a low concentration of manganese was found in the livers of the calves, while high strontium concentrations were confirmed in the soil, plants and grass specimens, as well as in the bone ash of the ribs of two calves. Skeletal deformities include a dwarf-like appearance, joint laxity, and domed foreheads.

The need for manganese for normal skeletal development appears to be related to its role in proteoglycan biosynthesis. Manganese deficiency reduces the total amount of cartilage proteoglycans and results in qualitative changes in the proteoglycans present in epiphyseal growth plate cartilage (Liu, Heinrich, & Leach, 1994). Chondrodysplasia of calves in Northeast Victoria was most likely caused by a congenital manganese deficiency following prolonged drought (McLaren, Cave, Parker & Slowcombe, 2007).

**Primary Dyschondroplasia**

Olsson and Reiland (1978) proposed that osteochondrosis may begin as a generalized abnormality of chondrocyte development and maturation, leading to multifocally altered endochondral ossification. The cause of the change was not defined, but one suggestion was that it occurred secondary to ischaemia-induced necrosis of vascular channels. They further hypothesized that secondary cartilage necrosis may occur as a result of biomechanical stress.

**Ischaemic Necrosis**

Ytrehus et al., (2007) concluded that articular osteochondrosis should be defined as a focal ischaemic necrosis of growth cartilage initiated by necrosis of cartilage canal blood vessels. Because necrotic cartilage does not undergo mineralization or vascular
penetration, a focal failure of endochondral ossification occurs when the ossification front approaches the lesion.

INVESTIGATIONS INTO OSTEOCHONDROSIS IN SOUTH AFRICA

For over half a century supplementing cattle with phosphorus-based licks seemed to have resolved the musculo-skeletal abnormalities associated with mineral imbalances in the North West Province of South Africa and surrounding areas. However in 1982, farmers and local veterinarians began noticing a new phenomenon. Affected cattle would develop joint effusions of the weight-bearing joints in particular, such as the femoro-tibial joint, and would eventually have to be slaughtered as a result of severe lameness and loss of condition. This condition is reported to occur in areas as far aroad as Cradock in the Eastern Cape, Olfantshoek in the Northern Cape, Harrismith in KwaZulu-Natal, Theunissen and Boshoek in the Free State, Francistown and Lobatsi in Botswana, and Gobabis in Namibia.

The syndrome is found to be present in cattle of all age groups and classes, and seemingly in most breeds of cattle farmed in that area. Afrikaner cattle seem to be more resistant to this condition. The only factor in common between affected animals is the geographical area in which they live. Clinically affected animals show joint effusions of the stifles joint particularly, which appear to get progressively larger. Associated pain and inflammation result in lameness of varying degrees.

Mineral analyses of bone and rib biopsies to date have consistently shown mineral imbalances. Thus a nutritional imbalance is suspected to be playing the major role in this disease. Water samples collected from 27 affected farms were analysed. Bromine was high in all samples, with nickel, lead and selenium often in elevated concentrations. Very high water hardness values were consistently seen while Fluoride was notably absent. To date, however, there is no clear evidence of a correlation between the drinking water and this syndrome.

AETIOLOGY OF OSTEOCHONDROSIS IN SOUTH AFRICA

Up until recently, many of the farmers in the area have been using bone meal and coarse salt at a ratio of 1:1 as their major lick source, with other protein or energy sources (such as molasses) being added during the winter months. The bone meal is sourced from cattle from the same area.

The bioavailability of phosphorus and other minerals from licks may not be adequate from sources such as rock-based phosphates. Inorganic sources of phosphorus are considered to be less biologically available and have a greater risk of containing potentially toxic elements, such as heavy metals, than organic sources. Rock-based inorganic phosphate sources have taken precedence over bone meal as a phosphorus source in the last few years as a result of the risk of bovine spongiform encephalopathy (BSE) transmission in the feeding of bone meal. Currently only a few feed companies produce phosphorus for feed in SA with differences in the bioavailabilities of phosphate. Thus deficiencies of minerals such as phosphorus may occur in spite of lick supplementation (Prozesky 2006).

HYPOTHESIS

As it is known that: there are imbalances of various minerals in the affected areas including Mn, Mg, Fe, Zn, Pb, Co and P intrinsic to that area; that the lesions are not typical of aposphorosis; and that preliminary trials using anionic salts and P sources seemed to have some positive effect, but did not completely solve the problem, it is hypothesised that the syndrome may well be multifactorial and i mineral/vitamin related.

Preliminary experimental results with cattle on different licks indicate that there is a significant decline in the clinical occurrence of arthrosis in cattle on some licks.

REFERENCES


**CPD Questions follow on page 11**

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