

Osteochondrosis in horses –
How serious is this developmental problem and what are we doing about it?

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Introduction

Horse breeding as an international industry has made an enormous investment in science over the last three decades and this has reaped considerable benefits. For example reproductive efficiency (i.e. conception and birth rates) has been significantly improved in most breeds of horses and losses due to twinning almost eliminated. There have also been considerable advances in perinatology with improved rates of foal survival. However, the situation in relation to growth and skeletal development in young horses is far less satisfactory. This is largely because this area has not been designated for detailed investigation and research. The result is that there is too much "art" and not enough "science" in the rearing/growing of foals. Perhaps this is why the incidence of developmental problems (e.g. Osteochondrosis, phytitis, angular limb deformities) is unacceptably high in many breeds of horse. Many of these conditions can lead to weakness, impaired skeletal integrity, lameness and poor performance potential.

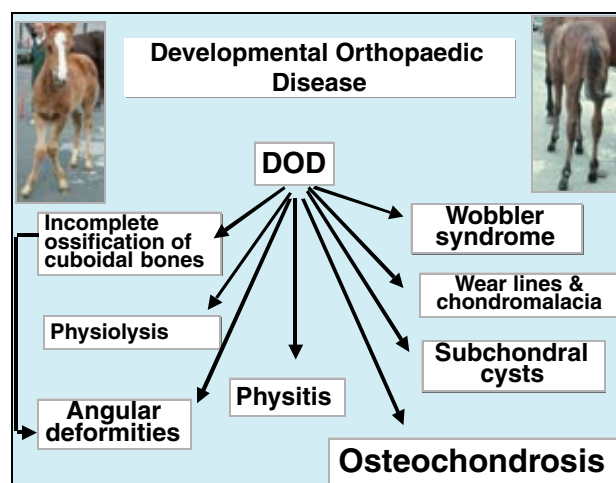


Fig 1
Range of conditions involved with developmental orthopaedic disease (DOD) in horses which includes Osteochondrosis

In the United States the term "Developmental Orthopaedic Disease" (DOD) was coined to describe a whole range of skeletal conditions from angular deformities to osteochondrosis (Fig. 1). One of the difficulties with this umbrella (or "dustbin") term is that including them altogether gives the impression that they have a common aetiology. The specific aetiopathogenesis is not known, but a common causation is most unlikely. The conditions are all associated with disturbances of growth cartilage and more than one can occur at the same time. (Fig.2).

Terminology of Osteochondrosis

Osteochondrosis has been recognised as a problem in rapidly growing animals of many domestic species. In some species (e.g. poultry and pig) a large volume of research has been carried out. This is not yet the case in the horse. Equine osteochondrosis is a developmental skeletal problem that costs the horse industry millions of pounds each year. It occurs worldwide in many



Fig 2 A rapidly growing Thoroughbred foal aged 7 months exhibiting clinical signs of DOD including osteochondrosis of hocks and stifles, flexural deformities of the forelimbs, phytitis of distal radial epiphysis

Epidemiological aspects

The topic of osteochondrosis in horses is currently of considerable interest around the world. It is accompanied by an extensive amount of scientific literature dating back to the first reported occurrence of the problem some 50 years ago in the stifle (Nilsson 1947). Despite the large numbers of scientific papers relatively few studies on pathogenesis have been undertaken. In particular there have been many radiological surveys demonstrating incidence of breed and sex predisposition and the common predilection sites (Fig. 5). The epidemiological data available suggests the condition is often present in the horse population at unacceptably high levels (i.e. 10-25%) across a range of different breeds. This means there must be horrendous economic wastage at a time when the horse industry can least afford it. However, stud farms that carefully monitor bodyweight and growth parameters in foals and then prevent bursts of rapid growth do appear to show lower incidence of the problem.

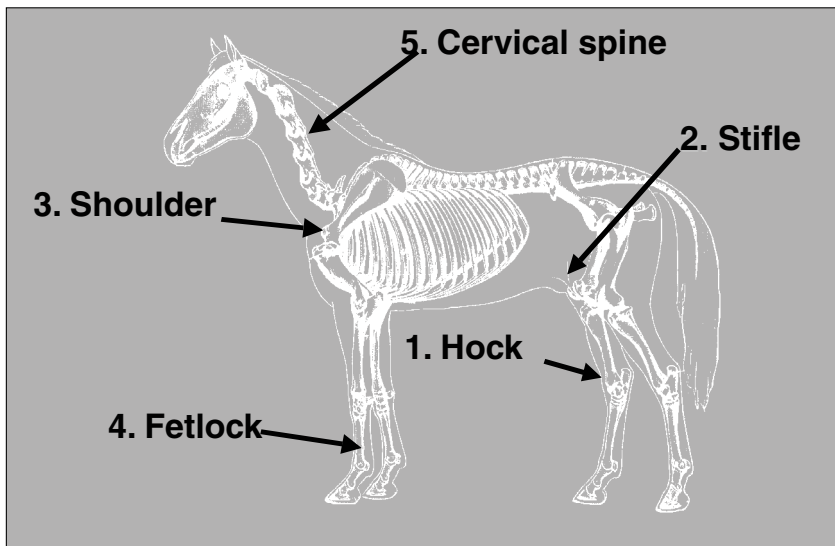


Fig 5 Predilection sites of osteochondrosis in the horse

Clinical aspects

The clinical signs of equine osteochondrosis are difficult to characterise specifically because of the wide range of lesions and sites involved. In severe cases, other signs of DOD also may be apparent (e.g. physal dysplasia, angular limb deformities and contracted tendons). Furthermore, lesions of dyschondroplasia do not always progress to osteochondrosis and produce clinical signs. These signs may begin with mild stiffness/lameness, but if there is superimposed biomechanical trauma, the joint damage progresses to pain and lameness or loss of performance.

The most common sign of osteochondrosis is a non-painful distension of an affected joint (e.g. gonitis, bog spavin). Clinical signs may be divided broadly into two categories; those presented by foals usually under six months of age and those occurring in older animals. Often the first sign noted in foals is a tendency to spend more time lying down. This is accompanied frequently by joint swelling, stiffness and difficulty keeping up with other animals in the paddock. An accompanying sign may be the development of upright conformation of the limbs, presumably as a result of rapid growth. Fetlock osteochondrosis is particularly seen in younger animals (i.e. < 6 months of age).

Marked lameness is not usually a feature of equine osteochondrosis, although it does occur with damage in some sites. For example, lesions in the shoulder frequently produce moderate to severe lameness, muscle atrophy and pain on joint flexion. In the stifle some cases of subchondral bone cysts in the medial femoral condyle present with lameness severe enough to suspect a fracture, but without a discernible site of pain or any joint swelling. The origin of pain in osteochondrosis presents a fascinating question. Horses often exhibit very severe pathological changes without showing much pain or distress. A very different situation from that observed in some other species and sites (e.g. canine elbow).

The main signs in yearlings or older animals are stiffness of joints, flexion responses and varying degrees of lameness. These signs are usually associated with the onset of training and therefore suggest a biomechanical influence and an activation of subclinical or "silent" lesions.

Factors contributing to pathogenesis

The primary lesion is usually seen as a small core of retained cartilage in the articular/epiphyseal growth cartilage (Fig. 6).

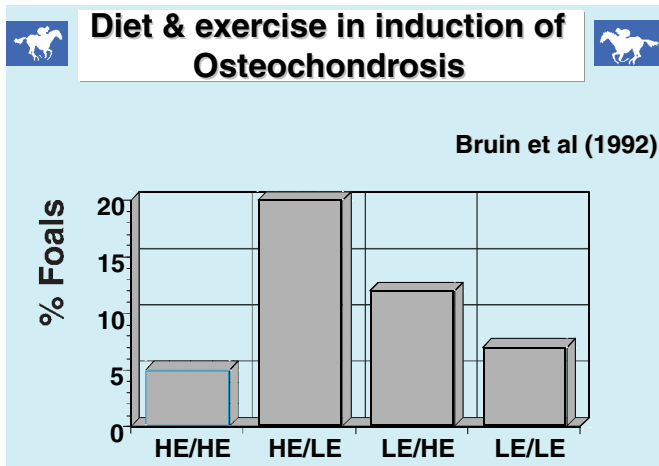


Fig 9 Effect of diet and exercise on the incidence of osteochondrosis in Dutch Warmblood horses (from Bruin and Creemer 1994).
 [HE/HE = High energy/high exercise HE/LE = High energy/low exercise
 LE/HE = Low energy/high exercise LE/LE = Low energy/low exercise]

The foals on the high intensity exercise programme fed a low energy diet actually had a higher level of osteochondrosis (13 per cent) than foals fed a high energy diet during high intensity exercise. Foals fed the low energy diet while on the low energy exercise regime also had a low incidence of osteochondrosis. Foals fed high energy and provided with little exercise had a high incidence of osteochondrosis. These findings indicated that increased exercise may be beneficial when high energy diets are chosen, but detrimental if lesions are already present, as purported in the group in which foals were fed a low energy diet, but exercised more frequently and intensively.

Diagnosis and clinical aids

Clinical diagnosis can often be made on the basis of signalment and signs. More definitive diagnosis requires the use of some specific clinical aids. Radiographic examination has been the traditional method of confirming diagnosis, but it should be remembered that early lesions involving cartilage without significant subchondral bone damage will not be visualised. In the distal limb oblique views may be helpful and in the hock as the commonest site of a lesion is the distal intermediate ridge of the tibia the best view is a plantarolateral/dorsomedial oblique. Ultrasound examination of the swollen joints can also be helpful and is capable of showing up articular damage and joint mice. The most accurate way to confirm diagnosis is by arthroscopy (McIlwraith 1990) and nowadays most of the predilection sites are accessible with the exception of the cervical articulations.

Other aids include nuclear imaging (scintigraphy) which will usually have negative results unless there is active secondary bone damage. Magnetic resonance imaging (MRI) is a modality that would be ideal for diagnosis of both early and late lesions, but is currently unavailable to equine veterinary practice. Clinical pathology and the evaluation of synovial fluid may be helpful, but is used largely to eliminate inflammatory causes of swollen joints.

Therapy and management

Management of clinical cases of osteochondrosis will depend on the site and severity of signs. In mild cases a conservative approach may be appropriate. In young animals (< 12 months) this would involve restricted exercise for some weeks combined with a reduction in feed intake to slow the growth rate down. Particular care should be taken to ensure appropriate mineral supplementation if this is indicated (e.g. suspected copper deficiency). It is controversial as to whether correcting the diet, once signs have developed, will actually assist resolution, but it may help limit or prevent further cases on the stud farms. Spontaneous recovery of mild cases can certainly occur. Intra-articular medication with hyaluronic acid may be beneficial and injection of long acting corticosteroids will assist in reducing swelling and improving any associated synovitis.

Those cases considered for surgery nowadays are mainly treated arthroscopically. This technique has been successful in most affected sites in particular the hock, stifle and fetlock. In addition to removing damaged cartilage and loose pieces of subchondral bone (i.e. joint mice) the bone overlying the lesion is curetted and the joint flushed extensively. Prognosis should be good in all but those cases with severe joint disruption or secondary arthrosis (degenerative joint disease).

Treatment of osteochondrotic lesions in the shoulder are often more problematic to treat surgically as arthroscopic access is more difficult, there is usually more extensive subchondral bone damage often with multiple cyst formation. The prognosis is therefore always rather guarded.

Future investigations

It is well accepted that dyschondroplasia is a multifactorial problem and there is good evidence that growth, nutrition, genetics, hormones and biomechanics all influence the condition. One of the most important keys to the whole problem is the ability to identify the first signs of a focal failure in the process of endochondral ossification (Fig. 10).

The primary lesion develops in growth cartilage and therefore a major area of investigation must involve the study of chondrocyte metabolism in growing foals. This will need to include molecular investigation of growth factor effects which are likely to have a direct effect on

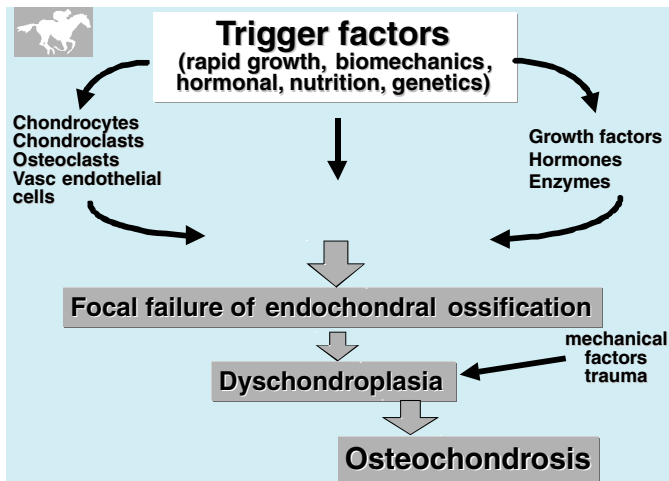


Fig. 10 Simplified representation of a hypothesis for the pathogenesis of dyschondroplasia and osteochondrosis.

chondrocytes which may influence their metabolism. Insulin may have a role and the insulin-like growth factors and their binding proteins are profoundly affected by insulin states. The transforming growth factors (TGF) may well stimulate proliferation and the expression of the tissue inhibition of metalloproteinases thereby affecting the synthesis of extracellular matrix and collagen. The direct investigation of the chondrocytes themselves and the secretion of metalloproteinases and cysteine proteases is also important as they are capable of cleaving the components of extracellular matrix in preparation for angiogenesis and mineralisation. Another important facet of the whole process is to understand more about the process of endochondral ossification. Work to identify certain markers (e.g. alkaline phosphatase, collagen type X secretion) has already begun (Henson et al 1995; Henson et al 1996). From these studies it should be possible to go on and investigate the influence of other factors (e.g. copper, zinc, growth hormone, thyroid function) on growth cartilage metabolism. The future is hopeful and there is much to learn from the condition in other species, in particular poultry and pigs.

Osteochondrosis prophylaxis

Despite the lack of precise information on the aetiology of dyschondroplasia there are a number of practical pieces of advice. Many veterinarians and studfarm managers have devised their own programmes to limit or prevent osteochondrosis (e.g. copper supplementation to the diet). The following steps for prophylaxis should be considered:-

1. ensuring balanced nutrition and a steady growth pattern;
2. avoiding excess carbohydrate and energy in the diet;
3. providing adequate exercise after weaning;
4. ensuring correct dietary copper status in the feed;
5. avoiding breeding from known genetic 'carriers'.

The evidence for genetic involvement implicates certain stallions, but unless a system of progeny testing is devised, the elimination of those stallions is fraught with difficulty. Radiological surveys will not identify with certainty horses having osteochondrosis because many mild or early lesions only involve cartilage and therefore will not be demonstrable radiographically.

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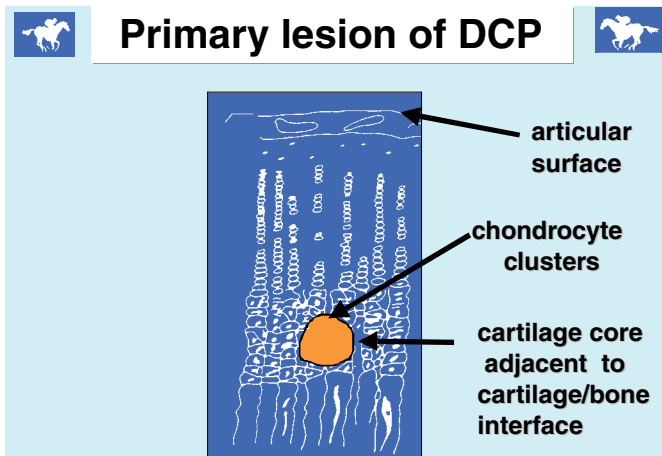


Fig.6 Example of an early lesion of dyschondroplasia in the articular epiphyseal growth cartilage of a foal showing a retained core of cartilage adjacent to the cartilage/bone interface.

At the predilection sites this can go onto develop a more serious lesion with secondary damage to subchondral bone and joint surface. Although the initiating cause of the dyschondroplasia is unknown, it is generally assumed to be multifactorial (Fig. 7) and includes:-

- Nutrition

There are many nutritional aspects to consider, but over-nutrition has been most widely reported as a cause of osteochondrosis. There is a lower incidence of the condition in foals fed according to the nutritional requirements of the National Research Council (NRC) (1989).

Factors involved in aetiology

- growth & body size
- nutrition
 - overfeeding
 - mineral imbalance
 - toxicity
- endocrinological involvement
- heredity/genetic predisposition
- biomechanics/exercise

Fig.7 List of some of the suspected aetiological factors in dyschondroplasia in horses.

Some dietary factors associated with the onset of osteochondrosis in horses

- excess digestible energy (DE)
- excess crude protein (CP)
- imbalanced Ca & P :-
 - excess phosphorus
 - relative deficiency Ca
 - excess calcium
- excess Zn/Cd
- deficiency of copper

Fig.8 Some dietary factors associated with the onset of osteochondrosis in horses.

These provide an average daily gain in weanlings of between 0.65 kg/day for moderate growth and 0.85 kg/day for rapid growth.

Diets with excessive levels of carbohydrate and oil (i.e. based on 128 per cent NRC requirements for digestible energy) fed to foals can cause widespread lesions of osteochondrosis (Savage et al 1993a). This means that excessive feeding of young horses for weanling and yearly sales is probably detrimental to skeletal development and may lead to clinical problems of developmental orthopaedic disease when they enter training. High energy diets appear to predispose to osteochondrosis, irrespective of the average daily gain. High or compensatory average daily gains were thought originally to predispose to lesions of osteochondrosis by increasing the stresses on growth cartilage. This may be important in some cases, but it is likely that factors associated with the intake of high energy diets cause abnormal development in the growth cartilage, producing osteochondrosis.

Despite proposals that excessive protein in the diet causes osteochondrosis, this has not been confirmed as a causative factor, nor does it appear to have deleterious effects on growth. However, diets with very high levels of quality protein are expensive and therefore wasteful.

An imbalance in mineral homeostasis is another important facet of the pathogenesis of osteochondrosis. Foals fed excessive amounts of phosphorus (i.e. four times the NRC recommendations) will consistently show lesions of osteochondrosis, although no clinical signs of nutritional secondary hyperparathyroidism ensue provided adequate calcium levels are fed (Savage et al 1993b). Excessive dietary calcium has also been proposed as a cause of hypercalcaemia, leading to osteochondrosis and osteoclerosis. There is no evidence to support this theory in foals, but it has been confirmed in fetal lambs whose dams were fed excessive calcium. The levels of calcium recommended by the NRC appear to be adequate and supplementation (e.g. three to four times the NRC recommendations) does not appear to be detrimental to the skeletal development of foals. However, increasing the calcium:calorie ratio to foals fed excessive energy (e.g. 128 per cent and 340 per cent NRC recommendations for digestible energy and calcium, respectively) does not appear to be protective. Lesions of osteochondrosis still develop, which is presumably a manifestation of the excessive energy level.

Copper deficiency in young horses has also been suggested as a cause of developmental orthopaedic disease and, in particular, phytitis and osteochondrosis (Knight et al 1985). Copper levels in the feed approximately the value recommended by the NRC (i.e. 10ppm dry matter) were not associated with an increased incidence of osteochondrosis in foals, as long as the energy and phosphorus components approximated the level recommended by the NRC. However, it has been reported in the USA that copper levels up to 25 to 50 ppm can be used in the diets of young horses, and this may decrease the incidence of developmental orthopaedic disease, especially phytitis. Horses have a high resistance to chronic copper toxicity and can withstand levels of up to 800 ppm, and so it seems very unlikely that levels of 25 to 50 ppm copper in the feed will have any deleterious effects. The mare can also be fed a higher copper diet during gestation; however, the copper level of mares' milk is low, irrespective of the dietary copper status of the mare. Consequently, to increase the copper status of the foal, supplemental dietary manipulation at foal level may be necessary (e.g. copper sulphate supplement in low energy creep feed), but recent work in New Zealand does not confirm that copper supplementation will reduce the incidence of Osteochondrosis (Pearce et al 1998).

No evidence exists that molybdenum interferes with copper metabolism in the horse, as persistent, protein-bound thiomolybdates that occur in ruminants have not been identified. It is unlikely that diets with excessive molybdenum could cause a copper deficiency and therefore developmental orthopaedic disease in horses.

- Heredity/genetic predisposition

Some useful genetic studies have been carried out on equine osteochondrosis in Scandinavia and the USA, mainly involving the hock. Radiographic surveys have shown a correlation between the incidence of osteochondrosis in the progeny and particular sires in both Standardbred and other breeds. The most definitive investigations have been carried out in Sweden and an extensive progeny testing genetic survey revealed a heritability coefficient of 0.24 to 0.27 for osteochondrosis (Philipsson et al 1993).

The evidence currently available suggests that it is not possible to formulate a programme of screening for osteochondrosis in stallions and mares that will ensure freedom from the condition in the offspring. Wagner et al (1986) performed a unique study by breeding 12 mares with wobbler syndrome to two affected stallions. The resulting offspring did not show signs of ataxia, but seven developed contracted tendons, four had osteochondrosis in the cervical spine and five had physal dysplasia.

- Endocrinological factors

The endocrinological control of skeletal growth is extremely complex and few studies have been documented in the horse. However, by extrapolation from other species, the hormones most likely to be involved with endochondral ossification are insulin, thyroxine, growth hormone, parathyroid hormone and calcitonin.

Diets containing high levels of carbohydrate predispose to post prandial hyperinsulinaemia/ hyperglycaemia and a higher than normal incidence of osteochondrosis (Ralston 1996). This type of diet may also invoke a temporary hypothyroaemia which could have adverse effects on chondrocyte maturation and extracellular matrix structure. No reports of any association between either growth hormone or parathyroid hormone and abnormal skeletal development in horses have been published. It is likely that a range of peptide growth factors and their receptors will play an important role in the control of growth and endochondral ossification (e.g. insulin-like growth factors, transforming growth factor β , fibroblast growth factor and the bone morphogenetic proteins). It has been suggested that excess calcium in the diet, leading to hypercalcaemia with decreased chondrocyte maturation and bone resorption, may cause dyschondroplasia (Krook and Maylin 1988). However, this has not been proven in the horse. A good deal more research into many of these endocrinological factors and their relationship with dyschondroplasia still needs to be done.

- Biomechanics/exercise

Controversy surrounds the subject of exercise and the subsequent effects on the presence and severity of osteochondrotic lesions. In a study in the Netherlands, Bruin and Creemers (1994) showed the effects of varying levels of exercise on the incidence of osteochondrosis in foals maintained on either low or high energy diets (Fig 9).

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breeds and its incidence appears to be steadily increasing. The primary lesion in growth cartilage involves focal damage during the process of endochondral ossification. It occurs most frequently adjacent to the joint surface in the articular/epiphyseal cartilage complex and results in the development of a cartilage core that can then progress to subchondral bone damage, serious cartilage pathology and production of bony fragments (joint mice) (Fig. 3).

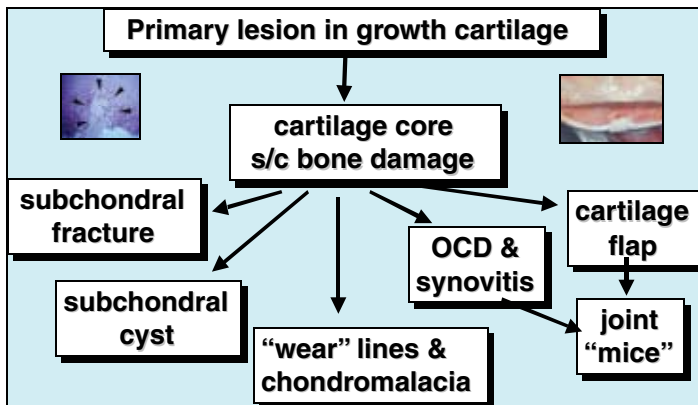


Fig 3 Schematic representation of the possible initiation of dyschondroplasia and osteochondrosis in horses

Most of the investigations reported in the literature on equine osteochondrosis have concentrated on the aspects of clinical diagnosis and treatment. The condition can be diagnosed and treated fairly effectively, but no systematic attempts at prevention have been employed. This cannot be done until the pathogenesis of the primary lesion is better understood (Jeffcott 1991). It is widely accepted that the initial lesion occurs in the proliferative or hypertrophic zone of articular/epiphyseal cartilage although the primary insult or "trigger" is still not known. The term "osteochondrosis" is therefore not strictly the most appropriate one for these early lesions, and "dyschondroplasia" is a much better alternative. If the primary lesion of dyschondroplasia progresses to involve more extensive pathology then such terms as osteochondrosis dissecans, osteochondritis dissecans, subchondral lesions, can be used. Finally, these lesions can further develop into chronic degenerative joint disease (e.g. arthropathy) and in other clinical scenarios (e.g. wobbler syndrome).

Growth patterns of foals

Osteochondrosis has traditionally been associated with large framed animals and during periods of rapid growth. However, not all surveys of growth measurements of normal foals and those with DOD confirm this opinion. Work studies in the Thoroughbred in Ireland (Jelan et al 1996) showed a steady almost linear increase from birth to 20 months of age and no significant difference between normal and DOD affected foals. The pattern of average daily gain (ADG) involves four phases of growth - birth to one month, 1-12 months, 12-15 months and 15-20 months (Fig. 4).

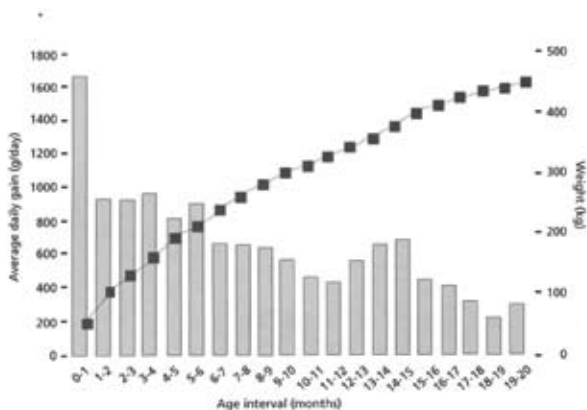


Fig. 4 Growth patterns in Thoroughbred foals (Jelan et al 1996). Average body weight and daily weight gain (ADG) in 798 foals born between 1988 and 1994.

In the States on a farm in Kentucky it was shown that growth rate and management can affect the incidence of certain types of DOD, but these differences were not dramatic (Pagan and Jackson 1996). A study in Sweden (Sandgren et al 1993) indicated that larger foals (6-16 months of age) were slightly more prone to osteochondrosis and had higher ADGs. Studies where growth rate has been slowed and then stimulated into compensatory growth have not shown the expected higher incidence of osteochondrosis. Recent studies in the States (Pagan et al 1996) suggest that growth rate per se in Kentucky was more a function of season of the year than age. So the picture of how growth rates affect the incidence of osteochondrosis is not clear cut, but larger and rapidly growing animals are probably more prone to the condition.