Developmental Orthopaedic Disease in Horses

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SUMMARY

This booklet was commissioned by the Rural Industries Research and Development Corporation in recognition of an industry need for up-to-date information on a common and costly disease. It is designed for horse breeders and stud managers in Australia, and provides a review of the current research on Developmental Orthopaedic Disease (DOD).

Chapter 1 provides an introduction to the syndrome of DOD. It describes the incidence of the problem both in Australia and overseas, and the approximate cost to the horse breeding and racing industries.

Chapter 2 examines the way foals grow and develop, and outlines both normal and abnormal bone development. It will aid breeders in identification of the individual diseases and provide an understanding of how they develop.

The current knowledge of the causes of DOD is discussed in Chapter 3. Many of the causes are not well understood, and information has often been extrapolated from research on similar diseases in the pig, dog, and chicken. There is an obvious need for further research into the causes of DOD in the horse.

Chapter 4 examines ways to minimise the incidence of DOD in young horses. Unfortunately, until all of the causes of DOD are identified, it is unlikely that we will be able to completely prevent its occurrence.

Chapter 5 gives a brief outline of equine nutrition in terms of the horse’s requirements, feed intake, and the nutrient content of feeds. It is not intended to be a complete review of the subject, and for a more comprehensive study, readers are encouraged to refer to the many available texts on horse nutrition.
Chapter 1.

What is Developmental Orthopaedic Disease?

1.1 Introduction
The term Developmental Orthopaedic Disease (DOD) was first used in 1986 to describe a range of skeletal problems associated with growth and development in the foal. The degree to which these disorders are related is the subject of much debate. Controversy also exists over the question of which diseases should be classed as DOD. However, the diseases listed below are generally recognised as being part of the DOD syndrome.

- **Osteochondritis dissecans** (OCD): thickening, cracking and tearing of the joint cartilage of growing horses. Loose fragments of cartilage or bone may be present in the joint space, causing swelling, pain and lameness.
- **Physitis**: inflammation, thickening and flaring of the growth plates in the lower end of the tibia (gaskin), radius (forearm), or cannon bones.
- **Angular limb deformities**: outward or inward deviation of the lower legs, involving the knee, hock, or fetlock joints. An angular limb deformity of the knee will result in either a knock-kneed or bow-legged appearance.
- **Flexural deformities** (contracted tendons, club foot): tightening or shortening of the flexor tendons at the back of the lower legs, producing a “knuckled over” or upright appearance to the limb. The knee, fetlock and coffin joints are most commonly affected.
- **Subchondral cystic lesions** (bone cysts): fluid-filled cysts occurring within a bone. The lesions most commonly affect the lower end of the thigh bone (femur) at the level of the stifle, but can also be found in the knee, fetlock, pastern and shoulder joints.
- **Cervical vertebral malformation** (wobbler syndrome): compression of the spinal cord in the neck, causing weakness, incoordination and an unsteady gait. This is usually most obvious in the hindlegs, but can progress to also involve the forelegs.
- **Cuboidal bone malformation**: malformation or collapse of the small bones within the hock or knee joints. This may cause lameness, sickle-hocks, or angular limb deformities.

It can be argued that not all cases of these disorders will be a problem of development per se. For example, congenital angular and flexural deformities may be caused by abnormal positioning in utero, and some cases of physitis, subchondral cystic lesions, and wobbler syndrome may be a result of trauma rather than abnormal development. However, most authorities working in this area agree that developmental problems lie behind the cause of the majority of cases.
1.2 Developmental Orthopaedic Disease in Australia

The incidence of DOD in Australia is not known and there are no surveys as to the incidence of this problem. However, interaction with veterinarians and stud managers in different regions of Australia has indicated that DOD appears to be an increasing problem. Recognising the problem for Thoroughbred breeders in the Hunter Valley, Mr Billy Neville, together with prominent stud managers, raised breeders’ concerns with Dr Bill Howey, a veterinarian with longstanding experience in the region. Discussions between Dr. Bill Howey and Professor Reuben Rose, the Research Manager of the Rural Industries Research and Development Corporation (RIRDC) Equine Research and Development Program, led to a workshop in Scone which aimed to establish a coordinated approach to the problem of DOD. The workshop was attended by stud managers, veterinarians, farriers, and researchers from the University of Sydney and the University of Glasgow. The major findings at this meeting were:

- Approximately 3000 Thoroughbred foals are born annually in the Hunter Valley
- 80% of foals will have some degree of angular limb deformity
- 40% of foals will require corrective hoof trimming for angular limb deformities
- 8% of foals will require surgery to correct angular limb deformities
- 3-5% of foals will have contracted tendons
- 6% of foals will become wobbler
- 5% of foals will develop OCD
- 10% of foals will not be sold at yearling sales because of DOD

Based on these findings it was estimated that DOD collectively costs the Hunter Valley Thoroughbred industry $9.8 million each year. This figure includes costs associated with diagnosis and treatment, increased labour, loss of sale value, and training fees. If these figures were applied Australia wide, the costs to the Thoroughbred industry alone could be as much as $60 million per year.

At the meeting it was agreed that two strategies are required for further investigation of the problem. Firstly, a retrospective audit of veterinary and stud records to accurately establish the current levels of DOD on Hunter Valley studs and to determine whether the incidence is increasing. A comparison with other Thoroughbred breeding areas in Australia such as Oakey, QLD, and Shepparton, Vic, will help to determine if there are regional differences in the incidence of DOD. Secondly, a prospective study extending over the next three to five years should be undertaken to allow many of the possible causative factors to be examined. This would be an expensive and demanding undertaking, but would provide detailed information on all variables, without being limited by existing records.

The following were regarded as important variables to be recorded:

- Mare; condition score during pregnancy
  - nutrition
  - resident or visiting
- Foal; date of birth
  - birth weight
  - sire
  - condition score and/or growth rate
nutrition  
exercise  
age at onset of DOD

1.3 Developmental Orthopaedic Disease Worldwide

DOD is not exclusively an Australian problem, or indeed, just a Thoroughbred problem. DOD is diagnosed world wide, and is commonly seen in the Thoroughbred, Standardbred, Quarter Horse and Warmblood breeds. In Ireland, nearly 68% of Thoroughbred foals surveyed developed some form of DOD (most commonly angular limb deformities and physitis), and 11.3% required treatment for the condition (O’Donohue et al 1992). A study in Kentucky, USA, found 10% of Thoroughbred foals born over a 4 year period on one stud developed OCD (Pagan and Jackson 1996). The most common location of lesions was the hock joint (4.4% of foals), followed by the fetlock (3.3%), and stifle or shoulder joints (2.2%). A radiographic survey of Standardbred horses in Quebec, Canada, found 25% had OCD and 11% had subchondral cystic lesions (Alvarado et al 1989). Other lesions found included degenerative joint disease, physitis, and sesamoiditis. Only 27% of horses surveyed had no abnormalities on Xray.

The different rates of incidence found in these studies may reflect differences in environment, genetics, nutrition, and management procedures, or may be a result of the different criteria used for diagnosis. Clearly, though, DOD is a significant problem in many of the major horse-breeding areas around the world.
Chapter 2.

Foal Growth and Development

2.1 How Foals Grow

At birth, a foal weighs approximately 10% of its expected mature weight. Most newborn Thoroughbred foals weigh around 50kg, and a foal weighing less than 35kg is unlikely to ever reach 15 hands high (Frape 1986). A large proportion of a foal’s birthweight is accumulated in the last 2-3 months of gestation, when the foetal demand for energy and other nutrients is at its greatest. During the tenth month of gestation skeletal development proceeds rapidly, and much calcium and phosphorus is deposited within the developing foal. This is also the time when nearly half of the foal’s copper, zinc, and manganese accumulation occurs (Ott 1994). Consequently, mare nutrition is of utmost importance during this period. Without an adequate supply of energy, calcium, phosphorus and other nutrients, the foetal development of the foal will be impaired.

Different tissues (bone, muscle, and fat) within the foal grow and develop at different rates. Bone is the earliest developing tissue; its major growth period begins in the final few months of gestation, and continues until around 12 months of age. Further bone growth does occur after this time but at a much slower rate compared to this early growth. The major growth period for muscle tissue begins 1-2 months before birth and by 2 years of age slows substantially. Fat is the latest developing tissue. Most fat deposition does not start until after birth and can continue throughout life.

Breed and genetics largely determine the rate of growth of the foal, but growth is greatly influenced by nutrition. Energy and protein are the major nutrients influencing growth rate of the foal. Maximum growth rate occurs when the foal is fed *ad libitum* high quality energy and protein. Similarly, if these nutrients are restricted to below requirements, growth rate falls. The amount of calcium, phosphorus and other nutrients needed in the diet is related to growth rate and therefore also to energy and protein intake. Therefore, foals growing at a rapid rate (ie on high energy diets) deposit greater quantities of bone, muscle and fat than those growing at slower rates, and hence have a greater requirement for calcium and phosphorus. Regular monitoring of growth rates can therefore be an important step in determining the nutrient requirements of growing horses.

Growth rate is greatest in the first few months of life, and then declines until maturity (Fig. 1). Unfortunately, the optimum growth rate required to maximise future performance and yet minimise developmental diseases has not been determined. However, most Thoroughbred foals at 3 months of age gain between 1 and 1.2kg of weight each day. By 6 months of age this drops to 0.65 to 0.85kg per day, and most foals now have reached around 46% of their mature weight and 84% of mature height (Frape 1986). It is not unusual for weanlings to experience a decrease in growth rate during the winter months of their first year, when pasture growth and quality is at its lowest. This is only a temporary drop, and growth rate increases with the onset of...
spring pasture growth. By 12 months of age most horses have reached 66% of their expected mature weight and 90% of mature height, and will be gaining approximately 0.5 to 0.65kg per day. This decreases to an average of 0.35kg per day at 18 months of age, and 0.2kg per day at 2 years.

**Insert Fig. 1 Daily Gain and Bodyweight of Thoroughbred Foals From Birth to 18 months.**

Weaning is an important time for feed intake and growth. Age at weaning appears to have little effect on mature height and weight, but nutrition at this time can be critical for future development. If foals have not been conditioned to eating creep feed before weaning, they may refuse to eat sufficient quantities of their new diet, causing a severe drop in growth rate. This is followed by a compensatory growth spurt, or “catch-up growth” once the foal begins to adjust to its new diet. These rapid growth spurts are undesirable as they are often associated with Developmental Orthopaedic Disease, particularly flexural limb deformities (contracted tendons and club foot). Therefore, feeding programs for young horses should aim to produce a steady increase in size and bodyweight, with avoidance of extremely rapid growth rates and growth spurts.

Regular monitoring of growth rates can be a very useful management tool for horse breeders. The most accurate method is simply to weigh each animal on a regular basis (fortnightly or monthly) and determine its average daily gain (ADG) in kg. If scales are not available, bodyweight can be estimated from a combination of condition score and height measurements (Table 1). Condition scoring involves assessing the degree of fat and muscle development at various points over the body, such as the neck, back, ribs, and pelvis. Figure 2 shows the condition scoring system of Carroll and Huntington (1988), which is probably the most commonly used system in Australia.

Table 1 Estimation of Weight (kg) from Height and Condition Score (CS)

<table>
<thead>
<tr>
<th>Height</th>
<th>CS 1</th>
<th>CS 2</th>
<th>CS 3</th>
<th>CS 4</th>
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<td>250</td>
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<td>420</td>
<td>470</td>
<td>520</td>
<td>575</td>
<td>650</td>
</tr>
</tbody>
</table>

(reproduced from Avery 1996)

**Insert Fig. 2 Condition Score Diagrams (modified from Avery 1996)**

### 2.2 Normal Bone Anatomy and Development

A typical long bone consists of a diaphysis (or shaft), the metaphyses and the epiphyses (Fig. 3). The epiphyses form the ends of the bone, and are covered with articular cartilage, which form the joints. The bony metaphysis and epiphysis are separated by the physis, or metaphyseal growth plate, which is made up of cartilage cells. The physis is responsible for increases in the length of the bone as the foal
grows. A similar growth plate exists at each epiphysis and provides for enlargement of the ends of the bone. The epiphyseal growth cartilage merges with the articular (joint) cartilage, and together these form the articular epiphyseal cartilage complex.

Insert Fig.3 Anatomy of a Long Bone

Growth in the long bones of a foal is accomplished by proliferation and enlargement of cartilage cells within the growth plates. These cells are then transformed into bone by a process called endochondral ossification. This process involves the calcification of cartilage cells, invasion by blood vessels and osteoblasts (bone-producing cells), and the deposition of new bone (Fig. 4).

Insert Figure 4 here

Longitudinal growth in the long bones continues until the physes close; that is, when the cartilage is completely replaced by bone. After this time, changes in the length of the bone cannot occur. Physeal closure occurs at a predetermined age peculiar to each bone type. For example, the growth plate at the distal (lower) end of the cannon bone closes at approximately 9 months of age, while the growth plate at the distal radius (lower forearm) closes at 2 to 2 1/2 years of age.

Physeal closure does not, however, mean that further bone development ceases. Bone is a dynamic tissue which is continually being remodelled in response to the loads or stresses placed on it. If exercise intensity is gradually increased over time, bone density and strength will increase in order to cope with the greater loads. The reverse is also true; an animal confined to a box or stall for greater than 3-4 weeks (eg due to illness or injury) will have a lower bone density and the bone will be structurally weaker. A sudden return to strenuous exercise could then result in trauma-induced damage to bones or joints.

2.3 Abnormal Bone Development

Osteochondritis dissecans

Osteochondritis dissecans, or OCD, results from an isolated disturbance to the process of endochondral ossification, and can occur in the epiphyseal or metaphyseal growth plates. The initial lesion probably develops before or shortly after birth, during the most rapid phase of growth. The exact mechanism remains unclear, but it appears that calcification of the cartilage cells does not occur and bone formation ceases (McIlwraith 1993). This results in a core or plug of cartilage left protruding into normal subchondral bone. The retained cartilage may die and become eroded or cracked, and loose flaps of cartilage or cartilage and bone may be present within the joint space (Fig 5).

Insert Figure 5 here

The clinical signs associated with OCD are quite variable. Foals may spend more time lying down or experience difficulties in keeping up with other horses in the paddock. Often there is swelling, stiffness or heat in the affected joint(s), and sometimes lameness will occur (Fig. 6). The most common site for OCD in the Thoroughbred is
the stifle joint, followed by the hock, shoulder, fetlock, and cervical spine. In Standardbreds, the hock is the most common site. It is common for OCD to affect several joints and often the same joint is affected in either the forelimbs or hindlimbs.

**Insert Fig. 6 Swelling of the Stifle Joint (gonitis) due to OCD**

**Physitis (Epiphysitis)**

Physitis is inflammation of the physis, or metaphyseal growth plate. It is generally believed to be a manifestation of OCD, although some researchers feel that biomechanical trauma (excessive exercise, excessive weight, or poor conformation placing stress on immature bone) may also produce the disease (Pool 1987). It most frequently affects the lower end of the radius (forearm), tibia (gaskin), and cannon bones, and usually is seen in foals between 4 and 10 months of age.

Clinical signs of physitis include enlarged or knobbly joints, often with heat or pain on palpation. Affected knee joints usually have a “dished-in” appearance, while fetlocks take on an hour-glass shape (Fig. 7). On Xrays, there is widening, flaring or irregularity of the growth plate, and callus formation.

**Insert Fig. 7 here**

**Angular Limb Deformities**

An angular limb deformity exists where the limb deviates axially from the normal vertical plane. The lower limb may deviate laterally (valgus deformity – where the limb deviates away from the body) or medially (varus deformity – where the limb deviates towards the body) (Fig. 8 & 9). The deformity most commonly involves the carpus (knee) or the fetlock but can also involve the hock.

**Insert Figures 8 and 9 here**

Congenital angular limb deformities are present at birth. They are generally believed to be a result either of malpositioning in the uterus, joint laxity, or incomplete ossification of the small cuboidal bones of the carpus (knee) or tarsus (hock). The latter 2 factors commonly are found in premature foals.

Acquired angular limb deformities become apparent in the first few weeks or months of life. They can be the result of OCD in the physis causing asymmetric growth in the long bone, or secondary to trauma or uneven weight distribution across the joint.

**Flexural Deformities**

Flexural deformities, or contracted tendons, result in an inability to straighten or extend the leg fully, with a “knuckled over” or upright appearance to the limb (Fig. 10).

**Insert Figure 10 here**

Congenital flexural deformities are present at birth, and can be a result of toxins or infections acting on the developing foal embryo, malpositioning in the uterus, or genetic factors. (Lewis 1995). The fetlock or carpal joints are most commonly affected, and the condition is usually bilateral.
Acquired flexural deformities are often secondary to pain associated with physitis, OCD or other injuries, or can result from a disparity between bone growth and tendon development. Areas most commonly affected are the fetlock and coffin joints. A flexural deformity of the coffin joint is characterised by a raised heel and a club-footed appearance.

Subchondral Cystic Lesions

This condition is characterised by the presence of radiolucent cyst-like structures in subchondral bone (Fig. 11). The presenting clinical sign is usually lameness, which often only becomes apparent after the horse begins training or suffers some other trauma or stress to the lesion. The stifle joint is most commonly affected, but lesions can also be found in the shoulder, fetlock, carpus (knee) and hock.

Insert Figure 11 here

Subchondral cystic lesions can be secondary to OCD, where cracks or fissures in articular cartilage allow joint fluid to be forced into the cavity during normal joint motion. The lesions may also occur as a result of biomechanical trauma to weight-bearing joint surfaces.

Cervical Vertebral Malformation

Cervical vertebral malformation (CVM), or “wobbler” syndrome, is characterised by a narrowing of the cervical vertebral canal, causing compression of the spinal cord. Clinical signs of CVM include progressive ataxia or loss of coordination of the hindlimbs and dragging of the toes. The clinical signs can be worsened by backing or turning the horse in small circles.

Controversy exists as to the cause of CVM. Many researchers believe CVM develops as a consequence of OCD in the cervical vertebrae (Jeffcott 1991). Other causes postulated include malpositioning in utero causing prolonged compression of vertebrae, or secondary degenerative joint disease as a result of chronic joint instability or other biomechanical disorders (Pool 1993).

Cuboidal Bone Malformation

The cuboidal bones are the numerous small bones that make up the carpus (knee) and tarsus (hock) joints. Cuboidal bone malformation occurs because of a delay in endochondral ossification of these bones. This can be a result of prematurity, hypothyroidism (abnormally low levels of thyroid hormones), or variations in the normal rate of ossification. The disease usually presents at or shortly after birth when the immature bone collapses under the stress of weightbearing (Fig. 12 & 13).

Insert Figures 12 and 13 here

Foals born many weeks premature will often have severely under-developed cuboidal bones (Fig. 14). The prognosis for survival and future athletic performance in these cases is poor.
Collapse of the cuboidal bones of the carpus generally results in a valgus (leg deviated away from the body) deformity, while collapse of the tarsal bones can cause lameness or a sickle-hocked appearance.
Chapter 3.

Causes of Developmental Orthopaedic Disease

DOD is recognised as having a multifactorial aetiology; that is, a number of different factors can influence the development of DOD. Problems with nutrition are generally recognised as one of the major causes, but trauma, rapid growth rates, large body size, hormonal aberrations and genetic predisposition are also important (Fig. 15).

3.1 Nutrition

Energy

Excess energy intake, particularly in the form of soluble carbohydrates, will consistently produce lesions of OCD. A study at the University of Melbourne compared the incidence of OCD in foals fed 100% and 129% of National Research Council (NRC) recommendations for energy (Savage et al 1993a). Eleven out of 12 foals on the high energy diet developed multiple OCD lesions. Only 1 of the 12 foals on the control diet showed evidence of OCD at post mortem. The lesions in the high energy fed foals were more numerous and more severe than in the control foal, and occurred irrespective of average daily weight gain.

Overfeeding the pregnant mare may also affect the incidence of DOD in her foals. Anecdotal evidence suggests that foals born to overfat mares are more likely to develop DOD than are those from mares in optimum condition. Some authors believe that overfat mares may predispose to foetal malpositioning, resulting in congenital flexural and angular limb deformities. Fat mares are more likely to experience problems with conceiving and maintaining pregnancy, as well as with foaling. They may also produce less milk due to the build up of fatty deposits within the udder, and this could lead to problems with compensatory “catch-up” growth once the foal is weaned.

Protein

Excessive dietary protein has also been proposed to cause DOD. There have been conflicting reports as to the effect of high levels of dietary protein, but recent studies have not found an association between feeding high levels of protein and the increased incidence of DOD (Savage et al 1993a). Further research is necessary.

Severe protein deficiency can result in impaired bone and muscle development in the horse. The degree of deficiency required to cause DOD would most likely result in poor condition and growth rates, and is unlikely to occur unless horses are receiving inadequate quantities of feed. However, there is some evidence to show that a mild protein deficiency combined with adequate energy intake may cause reduced bone growth without affecting weight gain, and this may predispose to DOD (Gibbs et al 1989).
**Calcium and Phosphorus**

Calcium and phosphorus together make up approximately 70% of the mineral content of the body and 50% of the skeleton. Both calcium and phosphorus are required for normal bone development. A deficiency of calcium or phosphorus will cause impaired endochondral ossification and decreased bone mineralisation, resulting in a wide range of skeletal disorders. These include lameness, decreased bone density, fractures, and DOD.

The ratio of calcium to phosphorus is an important factor to be considered when formulating diets for horses. Ideally, the calcium-phosphorus ratio should be in the range of 1.2:1 to 2:1. Diets with a calcium-phosphorus ratio less than 1:1 (where phosphorus intake is greater than calcium intake) may be detrimental, especially when fed to growing horses. Even if the total amount of dietary calcium is adequate, excessive phosphorus can inhibit the absorption of calcium, leading to a relative calcium deficiency and subsequent problems with bone development. Diets with a calcium-phosphorus ratio of approximately 1:2.1 (and nearly 4 times the recommended amount of phosphorus) have been experimentally shown to cause DOD. Five of 6 foals on this high phosphorus diet developed multiple OCD lesions, compared to only 1 of 12 foals fed to 100% NRC recommendations for calcium and phosphorus (Savage *et al* 1993b).

Diets likely to contain excessive phosphorus or a low calcium-phosphorus ratio are those with a high grain or bran content combined with little or no lucerne, clover or other calcium supplement.

Excessive dietary calcium may also be a cause of DOD, but there is some debate amongst researchers on this issue. High levels of calcium can lead to hormonal changes (increased calcitonin) which may inhibit the replacement of cartilage by bone, resulting in OCD (Krook and Maylin 1988). However, Savage *et al* (1993b) fed diets containing 3 ½ times NRC recommendations for calcium to 6 foals, and did not find an increased incidence of DOD in these foals. Further research in this field is necessary.

Much has been written about the calcium and phosphorus requirements of weaned, growing horses, but there is little information regarding the requirements of sucking foals. Mares’ milk in the first 4 weeks of lactation typically contains around 1.2g of calcium and 0.725g of phosphorus per kg of fluid milk (NRC 1989). If the foal consumes 15kg of milk, its intake will be 18g of calcium and 10.9g of phosphorus each day (this will vary according to milk composition and production, and foal appetite). It is assumed that this is sufficient to meet the requirements of the sucking foal, but there is little in the way of research data to confirm this. The NRC (1989) recommends that 4-month old weanlings receive 34g of calcium and 19g of phosphorus daily (based on 500kg mature weight and moderate growth rates). This decreases to 29g of calcium and 16g of phosphorus by 12-18 months of age.

**Trace Minerals**

Recent studies have shown a correlation between low copper diets and the incidence of DOD. An epidemiological study in Kentucky found low copper levels were the most consistent factor on stud farms with a high incidence of DOD (Gabel *et al* 1987). Experimentally, low copper diets have been shown to increase the incidence of
OCD, physitis, and angular and flexural limb deformities (Hurtig et al 1993). This is thought to be the result of a deficiency of the copper-dependant enzyme lysyl oxidase, which is essential for the maturation of cartilage and bone. A deficiency may result in a softening of articular cartilage and predisposition to fractures in the growth plates.

In contrast to sheep and cattle, high levels of molybdenum or sulphur in the diet do not appear to affect adversely the availability of dietary copper in the horse. However, excess zinc, iron and perhaps cadmium may decrease copper absorption or utilisation, resulting in a secondary copper deficiency. For example, young horses grazing pastures contaminated with zinc, iron and lead from industrial smelters near Newcastle, NSW, were found to have enlarged joints, flexural deformities, lameness, and multiple OCD lesions (Eamens et al 1984). On post mortem, tissue levels of zinc and lead were elevated, while copper was decreased. The authors concluded that a zinc-induced copper deficiency was responsible for the skeletal abnormalities.

Dietary zinc deficiency has also been postulated as a cause of DOD (Knight et al 1985). An unpublished study in the USA found that foals fed 152 ppm (mg/kg) zinc had less cartilage defects than those fed 42 ppm (Lewis 1995). Further research is required to confirm this finding.

The NRC (1989) recommends that all horses receive 40ppm zinc and 10ppm copper in the diet. The average 6 month old Thoroughbred weanling weighing 215kg and consuming 6kg of feed therefore would require 240mg of zinc and 60mg of copper per day. Many researchers, however, think that young horses consuming these levels (or lower) are more likely to develop DOD, and suggest that they receive 400mg of zinc and 150mg of copper each day (Huntington et al 1996). This equates to a concentration of approximately 100ppm zinc and 40ppm copper in supplemental feeds for young horses.

In the first 4 weeks after birth, mares’ milk contains around 2.5 mg of zinc and 0.45 mg of copper per kg of fluid milk (equivalent to approx. 25ppm zinc and 4.5ppm copper on a dry matter basis) (NRC 1989). Assuming the foal consumes 15kg of milk each day, its daily intake of zinc and copper will be only 37.5 mg and 6.75 mg respectively. The foal must rely on its own body stores of these two minerals until it begins to eat significant amounts of pasture or creep feed. It is therefore important that the pregnant mare receives adequate amounts of zinc and copper to allow the foetus to accumulate sufficient body stores before birth. Researchers in New Zealand have recently shown that copper supplementation of pregnant mares (an extra 250mg per day for 500kg mares) will increase the copper stores and decrease the incidence of physitis and cartilage lesions in their foals (Pearce et al 1997a, 1997b).

It should be noted that the figures used in the above calculations on milk composition are averages based on several different studies on the composition of mares’ milk. Milk composition will vary according to nutrient intake, stage of lactation, and individual mare factors. The number of previous lactations, breed, age, season of the year, and temperature may also have an effect on milk composition.
Pasture Composition

The type of pasture being grazed will affect total calcium and phosphorus intakes and therefore may influence the development of DOD. Legume pasture species (clovers and lucerne) contain much more calcium and less phosphorus than grasses (Table 3). Therefore, lactating mares and growing horses grazing grass-only pastures may suffer from a deficiency of calcium, whilst those on predominantly legume-based pastures may receive too much. For example, a 6 month old, rapidly growing weanling requires 36g of calcium each day. Assuming it eats 6kg of dry matter daily, its calcium intake will be 103g per day when grazing lucerne pasture, but only 22g per day when grazing phalaris. Both of these figures represent significant deviations from the recommended calcium intake and could potentially result in DOD.

Anecdotal evidence suggests that the incidence of DOD is reduced in times of drought, and increases in good seasons. Lush, rapidly growing pastures generally have a higher content of soluble carbohydrates and protein than the dry, mature pastures found in the drier times of the year. The higher incidence of DOD noted in good seasons may, therefore, be a result of increased intake of soluble carbohydrates, as this factor has been experimentally shown to cause DOD (Savage et al 1993a).

Lush pasture diets also may affect calcium balance and absorption. Dairy cows grazing lush pastures have a much higher incidence of milk fever (low blood calcium) than those grazing mature pastures or hay (Harris 1981). This is thought to be a result of changes in calcium absorption and excretion (Hyde 1994). There has been no published research on the effect of lush pasture on calcium balance in the horse. The value of pasture as a horse feed is often under-estimated in Australia. Good quality pasture can supply sufficient nutrients for mature horses, but growing horses and horses in training may require supplementation (Avery 1996). McLaughlin and McKiernan (1986) found that improved, irrigated pastures of kikuyu and white clover in the Hunter Valley provided sufficient protein and energy to support good rates of foal growth (average daily gain 1.93 kg in early lactation, 0.97 kg at weaning).

However, little work has been done in the Hunter Valley to determine the mineral composition of pastures throughout the year and their ability to meet the requirements of growing horses. In Camden, NSW, the composition of improved pastures on dairy farms was monitored for three years (Kellaway et al 1993). The pastures consisted of ryegrass, kikuyu, pigeon grass, summer grass, paspalum and white clover. The mineral content of these pastures often did not meet the NRC recommendations for calcium and phosphorus in horses, and was consistently deficient in copper (Trevor-Jones 1995).

The mineral content of pastures will vary according to soil type, plant species, season, and fertiliser history. If pasture is to provide the major part of the diet of broodmares and young horses, analysis of its mineral composition may be necessary to ensure that mineral intake is adequate.

3.2 Exercise and Biomechanical Trauma

Adequate exercise in foals is essential for normal bone and joint development. It has been stated that foals require at least 12 hours each day “at play” to allow normal development to occur (Gabel 1988). Research at the University of Melbourne has
shown how high intensity exercise can increase bone density and strength in young Thoroughbred horses (McCarthy & Jeffcott 1992). This occurs because bone is able to adapt to gradually increasing loads by increasing its cross-sectional area (thickness) and decreasing its porosity, resulting in stronger bone that is more able to withstand the stresses of training and racing.

A study in The Netherlands found that exercise had a protective effect on Warmblood foals fed high energy diets (Bruin & Creemers 1994). The “high intensity” exercise program consisted of 30 to 45 minutes of trotting and galloping three times a week in a training mill (lunging ring) with a sand-covered concrete base. The “low exercise” group of foals were walked in the training mill for an equal amount of time. Foals on the high intensity exercise program and high energy diet had a much lower incidence of OCD (6%) than those on the same high energy diet but with low intensity exercise (20%). Foals on a low energy diet and high intensity exercise program also had a relatively high incidence of OCD (13%). These findings suggest that increased exercise may be beneficial when animals are fed high energy diets, but detrimental if lesions are already present (Jeffcott 1997).

Biomechanical trauma may be caused by excessive bodyweight, abnormal conformation, excessive exercise, or increased or uneven weightbearing (eg lameness or pain in opposite leg). The result is a potentially damaging increase in stress or loads applied to bones and joints. Some DOD lesions may be entirely due to excessive biomechanical forces acting on normal cartilage, resulting in disruption of the blood supply to the cartilage and failure of ossification. Trauma to joints or bones already affected by DOD is likely to increase the severity of the lesions.

### 3.3 Body Size and Growth Rate

Extrapolation from studies in other species, particularly pigs and dogs, would suggest that large body size and rapid growth rates predispose to OCD. However, currently there is debate amongst researchers regarding the effect of rapid growth rate and large body size on the incidence of DOD in the horse.

It is generally believed that DOD is more common in large, rapidly growing horses, possibly as a result of excessive weightbearing and other biomechanical stresses on bones and joints. There is evidence to support this view. Pagan and Jackson (1996) in Kentucky found that Thoroughbred foals that developed hock OCD lesions were 5kg heavier than the population average at 25 days of age, and 14kg heavier at 240 days. Similarly, foals with stifle or shoulder OCD were 5.5kg heavier at 25 days and 17kg heavier at 120 days. Foals that developed early fetlock lesions, however, were of average size and growth rate. Other studies have found no difference in the size or average daily gain of horses with and without DOD (Savage et al 1993a, Bruin and Creemers 1994).

Compensatory growth spurts may affect the incidence of DOD. In one study, 6 foals were placed on a restricted feed intake sufficient to limit their growth to 0.3kg per day (Hintz et al 1976). After 4 months the feeding program was changed to allow ad libitum intake. Four of the 6 foals subsequently developed flexural deformities. None
of the control foals continuously fed the same diet ad libitum developed flexural deformities or other forms of DOD.

**3.4 Hormones**
Cartilage maturation and bone growth are controlled by the endocrine, or hormone, system. It is a complex system that has not yet been adequately studied in the horse. Extrapolation from other species suggests that insulin, thyroid hormones, growth hormone, parathyroid hormone, peptide growth factors and calcitonin are most likely to be involved with bone and cartilage development in the horse (Jeffcott 1997).

Some researchers believe that most nutritionally-induced effects on bone and cartilage growth are mediated by the endocrine system (Glade 1986). When diets containing high levels of carbohydrate are fed to horses, secretion of insulin and thyroid hormones will rapidly increase. This is quickly followed by an abnormally rapid removal of the thyroid hormones from the system (Glade 1987). This may have important consequences for bone and cartilage development because thyroid hormones are required for the maturation of cartilage. Horses with naturally low levels of thyroid hormones (hypothyroidism) often will show delayed cartilage maturation, abnormal ossification and OCD (Vivrette et al 1984).

There has been recent interest in the use of growth hormone, or somatotropin, in the horse. Research in the pig has shown that administration of somatotropin will increase the incidence and severity of OCD lesions (He et al 1994). Studies on the effect of somatotropin in the horse are currently underway but no results have been reported in growing horses.

**3.5 Genetics**
Many authors have suggested a genetic predisposition for DOD in horses. There have been several surveys of Standardbred horses in Europe and the USA which support this theory. In Denmark the yearling progeny of 9 Standardbred stallions were examined for OCD of the hock joint (Schougaard et al 1990). The progeny of two of the stallions had a much higher incidence of lesions (25.9% and 30.0% of yearlings) compared to yearlings sired by the other 7 stallions (3.4%-18.5%). Neither of the two stallions with the high incidence in their progeny had abnormalities on Xrays themselves. This suggests that selection of stallions to minimise DOD would need to made on progeny testing rather than Xrays of individual stallions.

An interesting study on the heritability of CVM was carried out in the USA. Two Thoroughbred wobbler stallions were mated to 12 wobbler mares (Wagner et al 1985). Of the 22 progeny, only one showed a compressive lesion in the cervical vertebrae, but 10 (45%) had OCD, 9 (40%) had physisis, and 7 (30%) had flexural deformities. The incidence of OCD, in particular, was much greater than would be expected in an average population of horses, and the authors suggested that breeding wobbler to wobbler will increase the incidence of DOD in foals.

The heritable nature of OCD has been proven in the dog, pig, and man. In the pig it is believed that genetic selection for faster growth, larger body size, and increased muscle mass has contributed to the high incidence of OCD in this species (Done &
Goody 1995). This could also be the case with the Thoroughbred, where it is often the larger, early maturing types that command the higher prices at elite yearling sales. In favouring these types we may be inadvertently selecting for an increased susceptibility to DOD.

Studies on the heritable nature of DOD in the Australian Thoroughbred have not been carried out, but there is some anecdotal evidence to suggest that some sires (and mares) may produce foals with a higher-than-average incidence of DOD. Considerable research would be necessary before any recommendations on sire selection could be made.
Chapter 4.

Prevention of Developmental Orthopaedic Disease

The following preventative measures are based on current knowledge on the causes of DOD and should help to minimise the incidence of the disease. There is still much to learn about DOD, and until the exact nature of the causes has been established, it is unlikely that we will be able to completely prevent its occurrence.

• Ensure a balanced diet is fed. Particular attention should be paid to the amounts of energy, calcium, phosphorus, copper and zinc in the diet. If in doubt have rations and pastures analysed.

• Don’t allow mares to become too fat. Monitor body weight or condition scores on a regular basis and adjust intake accordingly.

• Avoid rapid growth rates and growth spurts by regularly monitoring weight gain and condition score of foals and weanlings. It may be useful to wean rapidly-growing foals at an earlier age to minimise the impact of fast growth rates on the incidence of DOD. Particular attention should be paid to foals fostered onto Clydesdale or Clydesdale cross mares, as these produce more milk than the average Thoroughbred broodmare.

• If creep feed is provided to foals, care must be taken to avoid overfeeding, particularly in relation to energy, calcium and phosphorus. In some areas, pasture alone may not supply adequate amounts of minerals to foals, and the provision of mineral supplements will be required.

• Provide adequate exercise. Twelve hours or more of paddock exercise each day should be sufficient for foals and weanlings. Young horses on high energy diets (such as yearlings being prepared for sale) may benefit from a more intensive exercise program.

• Avoid excessive stress or trauma to young, growing bones and joints. Don’t allow young animals to exercise to fatigue or exhaustion. If foals must be confined for more than 2-3 weeks, try to provide a gradual return to exercise to allow bones to adapt to the increasing work load.

• Feed small, frequent meals to minimise the effects of abnormal hormone fluctuations.

• Avoid selection of bloodlines with a high incidence of DOD.
Chapter 5.

Equine Nutrition

Nutrient requirements of horses

The daily requirements of horses for energy, protein, calcium, phosphorus, zinc, and copper are presented in Table 2. For a more comprehensive list of nutrient requirements see the National Research Council’s Nutrient Requirements of Horses, 5th edition, 1989.

These figures provide the best estimate of the horse’s nutrient requirements through different stages of growth and reproduction. However, they are based on American conditions of environment, management and feeding, and in some cases information has been extrapolated from other species. A great deal more research is required to define the nutrient requirements of the horse under Australian conditions, particularly in relation to vitamin and mineral intake, and optimum growth rates.

Table 2 Daily Nutrient Requirements of the Horse (NRC 1989)

<table>
<thead>
<tr>
<th>Animal</th>
<th>Weight</th>
<th>Daily Gain</th>
<th>Energy</th>
<th>Crude Protein</th>
<th>Calcium</th>
<th>Phosphorus</th>
<th>Zinc</th>
<th>Copper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mature horse</td>
<td>500</td>
<td>NA</td>
<td>68.7</td>
<td>656</td>
<td>20</td>
<td>14</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>maintenance</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pregnant mare</td>
<td>500</td>
<td>NA</td>
<td>76.2</td>
<td>801</td>
<td>35</td>
<td>26</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>9 months</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pregnant mare</td>
<td>500</td>
<td>NA</td>
<td>82.5</td>
<td>866</td>
<td>37</td>
<td>28</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>11 months</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactating mare</td>
<td>500</td>
<td>NA</td>
<td>118.5</td>
<td>1427</td>
<td>56</td>
<td>36</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>0-3 months</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactating mare</td>
<td>500</td>
<td>NA</td>
<td>101.7</td>
<td>1048</td>
<td>36</td>
<td>22</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>3-6 months</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weanling 6mo</td>
<td>215</td>
<td>0.65</td>
<td>62.8</td>
<td>750</td>
<td>29</td>
<td>16</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>mod. growth</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weanling 6mo</td>
<td>215</td>
<td>0.85</td>
<td>72.0</td>
<td>860</td>
<td>36</td>
<td>20</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>rapid growth</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yearling 12mo</td>
<td>325</td>
<td>0.5</td>
<td>79.1</td>
<td>851</td>
<td>29</td>
<td>16</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>mod. growth</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yearling 12mo</td>
<td>325</td>
<td>0.65</td>
<td>89.2</td>
<td>956</td>
<td>34</td>
<td>19</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>rapid growth</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yearling 18mo</td>
<td>400</td>
<td>0.35</td>
<td>82.9</td>
<td>893</td>
<td>27</td>
<td>15</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>Not in training</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 year old</td>
<td>450</td>
<td>0.20</td>
<td>78.7</td>
<td>800</td>
<td>24</td>
<td>13</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>Not in training</td>
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<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Average nutrient content of feeds

Table 3 shows the average nutrient content of feeds commonly fed to horses. Nutrient content will vary according to soil type, fertiliser, stage of growth, harvesting conditions, storage conditions, and other factors.

Table 3 Average nutrient content of feeds (NRC 1989)
## Feed Type

<table>
<thead>
<tr>
<th>Feed Type</th>
<th>Dry Matter %</th>
<th>Digestible Energy MJ/kg/DM</th>
<th>Crude Protein %</th>
<th>Calcium g/kg</th>
<th>Phosphorus g/kg</th>
<th>Zinc mg/kg</th>
<th>Copper mg/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Grains</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oats</td>
<td>89</td>
<td>13.4</td>
<td>13.3</td>
<td>0.9</td>
<td>3.8</td>
<td>39</td>
<td>6.7</td>
</tr>
<tr>
<td>Barley</td>
<td>87</td>
<td>15.4</td>
<td>13.2</td>
<td>0.5</td>
<td>3.8</td>
<td>19</td>
<td>9.2</td>
</tr>
<tr>
<td>Corn</td>
<td>88</td>
<td>16.1</td>
<td>10.4</td>
<td>0.5</td>
<td>3.1</td>
<td>22</td>
<td>4.2</td>
</tr>
<tr>
<td>Sorghum</td>
<td>90</td>
<td>14.9</td>
<td>12.7</td>
<td>0.4</td>
<td>3.6</td>
<td>30</td>
<td>6.0</td>
</tr>
<tr>
<td>Wheat</td>
<td>89</td>
<td>16.2</td>
<td>14.6</td>
<td>0.5</td>
<td>4.2</td>
<td>37</td>
<td>5.5</td>
</tr>
<tr>
<td><strong>Hay/Chaff</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lucerne</td>
<td>91</td>
<td>9.5</td>
<td>18.7</td>
<td>13.7</td>
<td>2.4</td>
<td>31</td>
<td>17.7</td>
</tr>
<tr>
<td>Ryegrass</td>
<td>86</td>
<td>7.7</td>
<td>10.3</td>
<td>6.2</td>
<td>3.4</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>White clover</td>
<td>91</td>
<td>9.2</td>
<td>22.4</td>
<td>13.5</td>
<td>3.3</td>
<td>17</td>
<td>9</td>
</tr>
<tr>
<td>Oaten</td>
<td>91</td>
<td>8.0</td>
<td>9.5</td>
<td>3.2</td>
<td>2.5</td>
<td>45</td>
<td>4.8</td>
</tr>
<tr>
<td>Wheaten</td>
<td>89</td>
<td>8.0</td>
<td>8.7</td>
<td>1.5</td>
<td>2.0</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td><strong>Pasture</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lucerne</td>
<td>23</td>
<td>12.3</td>
<td>22.2</td>
<td>17.1</td>
<td>3.0</td>
<td>NA</td>
<td>10.8</td>
</tr>
<tr>
<td>Ryegrass</td>
<td>23</td>
<td>9.2</td>
<td>17.9</td>
<td>6.5</td>
<td>4.1</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>White clover</td>
<td>19</td>
<td>10.5</td>
<td>25.8</td>
<td>12.7</td>
<td>3.5</td>
<td>4</td>
<td>NA</td>
</tr>
<tr>
<td>Phalaris</td>
<td>23</td>
<td>10.6</td>
<td>17.0</td>
<td>3.6</td>
<td>3.3</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Fescue</td>
<td>31</td>
<td>9.3</td>
<td>15.0</td>
<td>5.1</td>
<td>3.7</td>
<td>22</td>
<td>NA</td>
</tr>
<tr>
<td>Timothy</td>
<td>27</td>
<td>9.9</td>
<td>12.2</td>
<td>4.0</td>
<td>2.6</td>
<td>36</td>
<td>8.9</td>
</tr>
<tr>
<td>Cocksfoot</td>
<td>24</td>
<td>9.6</td>
<td>12.8</td>
<td>2.5</td>
<td>3.9</td>
<td>NA</td>
<td>33.1</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bran</td>
<td>89</td>
<td>13.8</td>
<td>17.4</td>
<td>1.4</td>
<td>12.7</td>
<td>110</td>
<td>14.2</td>
</tr>
<tr>
<td>Linseed meal</td>
<td>90</td>
<td>12.7</td>
<td>38.4</td>
<td>4.3</td>
<td>8.9</td>
<td>NA</td>
<td>28.5</td>
</tr>
<tr>
<td>Soybean meal</td>
<td>89</td>
<td>14.7</td>
<td>49.9</td>
<td>4.0</td>
<td>7.1</td>
<td>57</td>
<td>22.4</td>
</tr>
<tr>
<td>Milk powder</td>
<td>94</td>
<td>17.0</td>
<td>35.5</td>
<td>13.6</td>
<td>10.9</td>
<td>41</td>
<td>NA</td>
</tr>
<tr>
<td>Molasses</td>
<td>74.3</td>
<td>14.7</td>
<td>5.8</td>
<td>10.0</td>
<td>1.0</td>
<td>21</td>
<td>65.7</td>
</tr>
</tbody>
</table>

NA: not available

**Expected daily feed consumption by horses**

Table 4 Expected Daily Feed Intake (% Body Weight) of Air Dry Feed (NRC 1989)

<table>
<thead>
<tr>
<th>Animal</th>
<th>Daily intake % body weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pregnant mare 9-11 months</td>
<td>1.5-2.0</td>
</tr>
<tr>
<td>Lactating mare 0-3 months</td>
<td>2.0-3.0</td>
</tr>
<tr>
<td>Lactating mare 3-6 months</td>
<td>2.0-2.5</td>
</tr>
<tr>
<td>Foal 3 months</td>
<td>2.5-3.5</td>
</tr>
<tr>
<td>Weanling 6 months</td>
<td>2.0-3.5</td>
</tr>
<tr>
<td>Yearling 12 months</td>
<td>2.0-3.0</td>
</tr>
<tr>
<td>Yearling 18 months</td>
<td>2.0-2.5</td>
</tr>
<tr>
<td>2 year old</td>
<td>1.75-2.5</td>
</tr>
<tr>
<td>------------</td>
<td>---------</td>
</tr>
<tr>
<td>24 months</td>
<td></td>
</tr>
</tbody>
</table>
Appendix

Glossary

ADG: average daily gain (kg).
Ad libitum: without limit.
Articular: relating to a joint.
Calcitonin: a hormone produced by the thyroid gland which has an important role in calcium regulation.
Carpus: the “knee” joint of the foreleg.
Cervical: pertaining to the neck.
Condition Score: a scoring system used to assess the body condition of a horse. Commonly ranges from 0 (very poor) to 5 (very fat).
Crude Protein: a close approximation of the protein content of a feed. Calculated by measuring the nitrogen content and multiplying by 6.25.
Diaphysis: the shaft of a long bone.
Digestible Energy: the proportion of energy in a feed which can be digested and utilised by the animal.
Distal: away from the centre of the body or origin; the lower part of a limb or bone.
Dry Matter: the plant tissue remaining after all the moisture has been driven off.
Endochondral: occurring within cartilage.
Epiphysis: the end section, or head, of a long bone.
Growth Hormone: a hormone secreted by the pituitary gland. Stimulates and controls the rate of growth.
Lateral: towards the side or outside; away from the midline of the body.
Medial: towards the midline of the body.
Metaphysis: the end of the shaft of a long bone; between the shaft and epiphysis.
Ossification: the process of bone formation.
Osteoblast: a bone-producing cell.
Parathyroid Hormone: a hormone secreted by the parathyroid gland. It regulates the resorption of calcium from bone.
Physis: the growth plate of a long bone, responsible for increases in length as the animal grows.
ppm: parts per million. Equivalent to milligrams per kilogram (mg/kg).
Radiograph: an X-ray film.
Radiolucent: permitting X-rays to pass through. Such tissue will appear dark on a radiograph, eg fluid or air.
Radius: the long bone of the forearm. Extends from the elbow to the carpus.
Sesamoiditis: inflammation of the sesamoid bones.
Somatotropin: growth hormone.
Subchondral: immediately below the cartilage.
Tarsus: the hock joint.
Thyroid Hormone: a hormone secreted by the thyroid gland, responsible for regulation of metabolism, growth and development.
Tibia: the long bone of the gaskin, extending from the stifle to the hock.
Valgus: angular limb deformity characterised by outward deviation of the distal limb.
Varus: angular limb deformity characterised by inward deviation of the distal limb.
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