

CONTRACT REVIEW

HORSE NUTRITION RESEARCH IN AUSTRALIA
PAST FINDINGS - FUTURE DIRECTIONS

INTRODUCTION

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Pastures, cereal grains, cereal and legume hays are the basic sources of nutrients consumed by Australian horses. Enquiry into the ability of cereal hay to supply the nutritional requirements of heavy working horses led to the pioneering work at Roseworthy of Perkins (1915). He showed that horsemen need to know when the hay is cut since the digestibility of fibre in wheaten hay declined with each successive cut from 46 per cent in the first cut at full bloom to 26 per cent in the final cut made five weeks later.

With the virtual disappearance of heavy horses in the 1950's nutritional research lapsed until Bourke (1968) suggested rations for racehorses. Later Groenendyk and Seawright (1974) attributed lameness in horses grazed on tropical pastures to unavailability of calcium bound to oxalates.

The decade commencing in 1980 witnessed an upsurge in community involvement in horses for business or recreation. Individual racehorses were sold at auction for prices exceeding \$1 million and Gawler SA attracted the horses and riders from many nations to the world three day equestrian event in 1986. Community interest in rations for performance horses was recognised by Gollan (1983) who reviewed rations for working horses including show hacks, show jumpers, polo ponies and endurance horses.

The 1980s also marked an increase in the small number of workers in horse nutrition research in Australia and it is the aim of this contract to discuss research in intake of grazing horses, energy values of rice milling by-products, mycotoxins and vitamin and mineral requirements of horses. This in turn will point to future directions in horse nutrition research in the 1990s.

INTAKE STUDIES WITH GRAZING HORSES

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A large proportion of the Australian horse population obtains its nutrient requirements by grazing native and improved pastures. This applies not only to working horses from pastoral areas and riding horses kept in proximity to urban areas but also to a large proportion of the Australian Thoroughbred and Standardbred brood mare population. No data on pasture intake of grazing horses were available until a study conducted at the Queensland University Veterinary Science Farm was reported by Gallagher and McMeniman (1988). The methodology used in that study has been further, used and extended to examine the effects of physiological state, season and supplementation on intake of subtropical pasture by two groups of 14 thoroughbred type mares. A preliminary report on this work has been presented (Martin et al. 1989).

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The studies were conducted during winter, spring and summer months. The pastures in the paddocks that the horses grazed contained *Pennisetum clandestinum*, *Lotononis bainesii*, *Trifolium repens* and *Paspalum plicatum*. The quantities of dry matter on offer during these months varied; the lowest yield recorded was 600 kg/ha and the highest was 2 100 kg/ha. The pasture yield that reliably provided for liveweight maintenance of dry mares was 1 000 kg/ha.

Pasture intake was calculated from estimates of diet digestibility and faeces output. Diet digestibility was determined from concentrations of acid insoluble ash (AIA) in samples of the diet and in the faeces. Our data indicate that, providing the diet contains at least 25 g AIA/kg DM, accurate estimates of digestibility can be achieved. The indigestible marker Cr_2O_3 was used to determine faeces output. Samples of the diets consumed by the horses were obtained by closely observing what the mares were consuming and then hand plucking a similar representative sample "diet".

Occasionally the above method of estimating digestibility and intake gave obviously incorrect values for individual mares. This probably occurred because these animals consumed diets with different AIA concentrations to the representative diet selected by the observer. Some animals grazed apart from the main mob, especially the dry mares which appeared to be at the lower end of the pecking order. Individual mares were also observed consuming sticks, bark, dirt and other foreign materials that may have influenced the concentrations of AIA. To minimise this problem individual diets for each animal could be selected but this would be labour intensive.

The dry matter digestibility of unsupplemented diets varied from 42 to 59% and this was reflected in digestible energy values which ranged from 8.3 to 10.7 MJ DE/kg DM. On a comparative basis these diets were equivalent in energy content to, at the lower end of the scale, poor quality oaten chaff and, at the higher end, good quality lucerne chaff. The crude protein content of the diets varied over a small range (100 - 140 g/kg DM) while digestibility of the protein was in the range 54 to 75%. At each sampling crude protein intakes were in excess of NRC (1989) recommendations for all mares (dry, pregnant or lactating). These relatively high protein intakes were associated with consumption of legumes, *Lotononis* in the summer and white clover in the winter. McInness and McMeniman (1984) showed that the plasma urea nitrogen value obtained when horses were consuming recommended quantities of digestible protein was approximately 2.3mM/l. In all instances the values obtained from the grazing mares were above this, again suggesting that the digestible nitrogen intake was adequate.

The subtropical pastures supported adequate DE intakes in all classes of mares except for lactating animals during the late spring and early summer periods. Liveweight losses were recorded in these animals at this time but this did not appear to influence the mares' reproductive activity or foal growth rate. When it was possible to make comparisons, it was found that the DE intakes of lactating mares were approximately 30% higher than those of pregnant mares which were in turn 10% higher than those of dry mares. These results show that horses have the capacity to vary their intake of energy in an attempt to match their energy requirements,

Throughout the breeding season in 1987 a grain based protein supplement of pellets (250 g crude protein and 12 MJ DE/kg DM) was fed at the rate of 2 kg/head/d to half of the mares and the effect that this supplement had on foal growth rate (milk production) and mare reproductive activity was measured. It was found that, when fed this supplement, the mares exhibited a significant degree of substitution. The intake of one kg of pellets was associated with a reduction in pasture intake of 0.7 kg. However the performance of the supplemented mares was not superior to that of the unsupplemented ones in terms of either foal growth rate or mare reproductive activity.

A management practice that is used during some winters at the Veterinary School Farm is to offer a molasses supplement *ad libitum* to the brood mares to increase their digestible energy intake. Our studies using the tritium marker technique of Nolan et al. (1974) showed that mares consuming the protein supplement referred to above ingested 1.5 kg of molasses DM per day while those that were not given the protein supplement ingested 2.5 kg per day. Assuming the molasses contained 14.6 MJ DE/kg DM (NRC 1989), it would have provided 18 and 30% of the DE requirements of the supplemented and unsupplemented mares respectively during early lactation. The animal performance data obtained in 1987 suggests, however, that this supplement is not required, at least not if the pasture yield is around 1 000 kg/ha.

The results of our studies so far show that satisfactory performance of brood mares can be achieved on subtropical and semi-improved pastures and that supplementation with either protein or energy is usually not necessary.

COMPARATIVE ENERGY VALUES OF RICE MILLING BY-PRODUCTS FOR HORSES AND OTHER LIVESTOCK

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The major by-products of rice milling available for livestock feeding are rice hulls, rice pollard and broken white rice. Australian rice pollard results from the milling of brown rice to white following dehulling of paddy rice and it is made up of a mixture of rice bran and rice germ meal. Consequently it is free of rice hull fractions which often contaminate rice brans overseas.

Energy values for rice by-products can be estimated using prediction equations from chemical analyses. However rice by-products have analyses at the extremes of materials used for preparation of prediction equations and the results are not satisfactory. Values from digestibility trials with live animals are therefore preferred. Digestible energy (DE) values and metabolisable energy (ME) values for ground rice hulls, rice pollard and broken white rice determined using sheep, pigs and poultry have been published by Farrell and Warren (1982) and DE values for rice pollard and broken white rice determined using horses have been published more recently by McCarthy et al (1989). Furthermore DE values for rice pollard and broken white rice have also been determined for horses at University of Queensland (N.P. McMeniman, personal communication).

Rice pollard and broken white rice have been widely used for high energy horse feeds in Australia for many years and ground rice hulls have been used successfully in lower energy horse feeds at 25% inclusion in the pelleted part of the ration (Huntington and Hutton, 1986). There are no DE values for ground rice hulls determined for horses. However it is unlikely that the value would be very different to the figure determined with sheep. Available data for ME and DE values of rice by-products determined with sheep, pigs, poultry and horses are summarised in Table 1.

Clearly rice milling by-products are suitable ingredients for feeds for all livestock. Rice pollard and broken rice have a high DE content for all livestock whereas the DE in ground rice hulls is low and restricts the situations in which this material can be utilised as a stockfeed ingredient.

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Table 1 The ME and DE of rice milling by-products determined with sheep, pigs, poultry and horses

		MJ/kg dry matter			Reference
		Broken rice	Rice pollard	Rice hulls	
Sheep	DE	14.0	16.9	2.6	Farrell & Warren (1982)
	ME	13.7	16.0		
Pigs	DE	17.3	17.5	3.7	Farrell & Warren (1982)
Poultry	ME	14.6	14.5	2.2	Farrell & Warren (1982)
Horses	DE	15.1	14.3	-	McCarthy et al (1989)
	DE	18.2	15.6	-	McMeniman (pers. comm.)

TOXICANTS ENCOUNTERED BY HORSES IN FEED AND PASTURE

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In the preceding contributions attention has been directed to many of the nutrients that are required in a diet for normal growth, development and performance. However feedstuffs also contain a variety of other substances which can have deleterious effects if consumed in sufficient quantities. These components may be present in the equine diet because of the inherent genetic characteristics of the plant from which the feedstuff was derived, be part of the feed as a result of man's activities in agriculture, storage, processing, etc, or result from inadvertent or accidental contamination during feed preparation or from environmental pollution.

Before turning to the deleterious compounds that may occur in horse rations it is as well to remember that over zealous provision of feed supplements including vitamins, minerals, amino acids, anthelmintics and various 'health foods', intended to maintain and improve equine health and performance can result in just the opposite effect. For any chemical, even an nutrient, there is a level of intake that constitutes a potential hazard. Excessive intake of carbohydrate, for example, can result in laminitis. The severity of an intoxication will be dependent on the dose and duration of exposure and be modified by genetic and physiological attributes of the horse and the environment in which it is kept. The exposure of horses to dietary toxicants will be determined by feeding practices which are largely dictated by the conditions under which the horse is kept.

Stabled horses

In a stable, horses receive either manufactured feeds or feeds mixed on the premises from ingredients of plant origin which may contain antinutritional factors. These include tannins in sorghum, gossypol in cottonseed, glucosinolates and erucic acid in rapeseed and antitrypsin factors in soybeans and other legume seeds (Cheeke and Shull 1985). However as a result of selection of low toxin varieties and/or carefully controlled processing, these factors are unlikely to adversely affect feed quality.

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Careless harvesting of crops can result in grain contamination with toxic weed seeds. Adverse growing or storage conditions can favour mould growth in grain, Spores from mouldy feed can result in respiratory allergies with *Aspergillus fumigatus* being notable in this regard. In other circumstances mould growth may result in the formation of toxins or mycotoxins in the feedstuff. These compounds can accumulate in feedstuffs both before and after harvesting or processing in what otherwise appear to be wholesome feed ingredients. The mycotoxicoses likely to be encountered in the Australian environment have been reviewed by Bryden (1989). The recent identification of fumonisin by Marasas et al. (1988) as the causal toxin of leukoencephalomalacia (ELEM), a neurological disorder of horses consuming maize contaminated with the fungus *Fusarium moniliforme* is a major break through in equine mycotoxicoses. One case of ELEM has been reported in Australia but the toxicosis has been diagnosed on other occasions but not reported (A.P. Begg and D.R. Hutchins, pers comm). Aflatoxin is the only other mycotoxin of stored feed that has been studied in any detail in horses and although aflatoxicosis has been reported in other Australian farm livestock, it has not been reported in horses.

There are numerous other potentially toxic substances which are used in the production and storage of feed ingredients, in feedmills and on farms which can contaminate horse feeds, and others which can be present in bedding. These substances include herbicides, insecticides, rodenticides, fungicides, disinfectants and environmental pollutants, for example, heavy metals. All of these substances can be toxic at some level, but the question of practical importance is the extent to which they can impair performance at the low dose rates commonly encountered. The inclusion of these compounds in feeds is usually by accident. Inadvertent mixing errors or cross-contamination in a feed mill has resulted in ionophore toxicity in horses. Ionophores, including monensin, lasalocid and salinomycin are used as coccidiostats in poultry or as growth promoters in cattle. The horse is the most sensitive domestic species to these compounds with a LD₅₀ of 2 mg/kg bodyweight for monensin compared to 200 mg/kg in broiler chickens (Todd et al. 1984).

Feed related toxicities in the stable can be largely overcome by purchasing top quality feed or feed ingredients, practising feed storage bin or silo hygiene and adherence to manufacturers' recommendations in the use of potentially toxic compounds which are either added to feed or used around the stable.

Horses at pasture

If horses at pasture are given a supplement, as often occurs with yearlings and brood mares, these animals can be exposed to the same toxic insults as listed above. However exposure of these animals to feed-related toxins is more likely to come from grazing a plant containing either a plant toxin, a mycotoxin or a herbicide residue.

The natural toxicants in plants cover a wide range of chemical classes (Cheeque and Shull 1985). Seawright (1982) and Barry and Blaney (1987) have listed the many plants in the Australian environment that contain toxic principles. For example the evergreen garden shrub, oleander, although relatively unpalatable to horses, is extremely toxic due to a presence of cardiac glycosides. Kimberley horse or walkabout disease occurs in horses ingesting plants of the genus *Crotalaria* which contain hepatotoxic pyrrolizidine alkaloids. Plants including buckwheat, St John's wort, burr clover, lucerne and alsike clover have been shown to contain compounds that cause photosensitization in horses. Other plants induce disease by making nutrients unavailable to the animals consuming them. Consumption of these oxalate-containing plants by horses may result in oxalate-induced calcium deficiency and the resultant nutritional secondary hypoparathyroidism (*osteodystrophia fibrosa* or big head) can be overcome by supplementation with calcium and phosphorus (Barry and Blaney 1987).

Mycotoxicoses can also occur in grazing horses. Pasture grasses including perennial ryegrass, paspalum, tall fescue and lupins are all subject to fungal infection. The resulting toxicoses namely; ryegrass staggers, paspalum staggers, fescue poisoning and lupinosis, respectively, and the responsible fungi and toxins have been described (Bryden 1989). Ergotism can also develop in horses consuming ryegrass infected with *Claviceps purpurea*. Red clover infected with *Rhizoctonia leguminicola* is responsible for slobbers in horses due to the production of the mycotoxin, slaframine. Interestingly, this fungus also produces the mycotoxin swainsonine as do plants of the genus *Swainsona* (e.g. Darling pea). This toxin induces a neurological disorder commonly known as pea struck. There is another equine disorder, Australian stringhalt for which the cause has not been defined but it has been postulated that a plant associated mycotoxin may be involved (Pemberton and Caple 1980).

Management errors are usually the major contributing factor in toxicological problems associated with grazing horses. Instances of intoxication can be reduced by avoiding pastures containing toxic plants or judicious use of mechanical, chemical or biological methods to control the growth of potentially poisonous plants.

Within the context of the present discussion the possibility of equine toxicosis resulting from the water supply should be mentioned. Horses receive water from many sources. The chemical composition of water can differ considerably and abrupt changes in water source can result in digestive upsets. Water can also contain high levels of salt, bacteria, fluoride, nitrates and nitrites and heavy metals. In addition, water from dams can be contaminated with hepatotoxic algal toxins or phycotoxins. Obviously, in instances of equine intoxication, the water supply should be checked.

Despite the many toxicants that a horse can encounter there is a poor understanding of the metabolism of many of these compounds in horses. Moreover there has been no adequate assessment of the relationship between subclinical intake of commonly encountered toxicants and performance.

VITAMIN AND MINERAL NUTRITION OF THE HORSE

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There is a bewildering array of mineral and vitamin supplements marketed for horses in Australia. In Victoria alone there are at least 25 horse feeds registered under the Stock Foods Act, and more than 30 iron supplements, 50 vitamin preparations and 130 mineral and other nutritional feed additives. The element of chance and doubt in horse-racing, eventing and the show guarantees that any product which might offer some competitive advantage will have a place in the market. This state of affairs probably also reflects the relatively meagre state of knowledge about the mineral and vitamin requirements of horses. When compared with the use of biochemical indicators to survey disorders of minerals and vitamin nutrition in pigs, poultry and ruminants, the use of biochemical tests for the diagnosis of clinical and subclinical nutritional deficiency disorders in horses has not been extensively researched.

Horses are assumed to require the same minerals and vitamins as other species, but the amounts required for different types and weights, and for pregnancy, lactation and growth have not been closely defined. The most comprehensive review of the nutritional requirements of horses is published by the United States National Research Council (NRC 1989). In the eleven years since the

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previous review was published in 1978 few changes have been made to the recommended dietary mineral and vitamin allowances. The only major change has been a recommended increase in the amount of dietary vitamin E from 15 mg/kg DM to 80 mg/kg DM for horses being fed a high proportion of cereal grains. A summary of the recommended trace element and vitamin allowances for horses is given in Table 2.

Disorders of mineral and vitamin nutrition in horses in Australia

Many nutrients are involved in cartilage and bone metabolism. Skeletal disorders represent the most important mineral problems in horses in Australia. *Osteochondrosis dissecans* and *osteodystrophia fibrosa* are the main nutritional bone disorders in horses.

Osteochondrosis, or developmental orthopaedic disease, is a metabolic bone disease in the young, growing horse which results from a disturbance in the conversion of cartilage to weightbearing bone. The cause of the disorder is unknown, but overfeeding, copper deficiency, calcium deficiency, and calcium and zinc excess have been implicated. It is particularly common in thoroughbred racehorses which are fed high energy and protein diets to promote rapid growth so they can be sold as yearlings for racing as two-year olds. The disorder seems to be predisposed by genetic factors,, dietary carbohydrate intake and the endocrine responses to diets, The maturation of cartilage is controlled by the endocrine system involving growth hormone, insulin, insulin-like growth factors, and possibly several cytokines. In horses genetically predisposed to this disorder, mineral deficiencies and imbalances may exacerbate its development when they are fed high carbohydrate diets. Additional trace mineral supplementation, particularly with copper, may reduce its severity (Milne 1987).

Osteodystrophia fibrosa results from secondary hyperparathyroidism due to inadequate Ca and excessive P intake. This leads to excessive withdrawal of calcium from bone, lameness and enlargement of the facial bones and mandible with soft fibrous tissue. This disorder occurs in two separate management systems for horses in Australia. It occurs in permanently stabled horses which are fed cereal grains which are inadequate in calcium and high in phosphorus. It also occurs in horses grazing pastures containing a total oxalate concentration greater than 0.5% DM and a Ca:oxalate ratio less than 0.5 (McKenzie et al. 1981). In Queensland the disorder has been a problem in stock horses grazing introduced tropical pastures such as buffel grass (*Cenchrus ciliaris*). Other grasses containing oxalate include kikuyu (*Pennisetum clandestinum*), purple pigeon grass (*Setaria porphyantha*) and pangola grass (*Digitaria decumbens*). Unlike ruminants which are able to metabolize calcium oxalate in the rumen, horses are unable to absorb Ca in calcium oxalate from the duodenum.

Lameness and skeletal disorders in young horses grazing pastures contaminated with zinc (100 mg/kg DM) near industrial areas has been associated with reduced copper absorption (Eamons et al. 1984).

Other rare nutritional disorders recorded in newborn foals include goitre due to either iodine deficiency or excessive iodine intake by pregnant mares, and myopathy due to selenium deficiency in foals less than two months of age (Caple et al. 1978).

While clinical sodium deficiency in horses is not commonly recognised, it is recommended that all horses have free access to salt particularly when there may be increased sodium losses through sweating and when low sodium diets such as lucerne are fed.

Calcium and phosphorus nutrition

The Ca and P requirements of horses have been well established from practical feeding trials and nutritional balance studies (NRC 1989). The recommended dietary allowances for maintenance are 3.0 g Ca and 2.0 g P/kg DM, with increases up to 5.0 g Ca and 3.5 g P/kg DM for lactating mares, and up to 8.5 g Ca and 6.0 g P/kg DM for foals less than 3 months of age. Problems with Ca and P nutrition in stabled racehorses usually only occur when little or no lucerne is fed and where the diet contains high proportions of cereal grains and protein concentrates without adequate Ca supplementation (Caple et al. 1982).

Table 2 Dietary concentrations of trace minerals and vitamins adequate for maintenance and growth of horses

Trace mineral	(mg/kg)	Vitamin	(IU/kg)
Copper	10.0	Vitamin A	
Manganese	40.0	(maintenance and work)	1 400
Zinc	40.0	(growth)	1 800
Selenium	0.1	(reproduction, lactation)	2 500
Iodine	0.1	Vitamin D	275
Cobalt	0.1	Vitamin E	80
		Thiamin (B ₁)	3
		Riboflavin	2

Based on NRC 1989 (H.F. Hintz, personal communication)

Vitamin nutrition

The requirements of horses for many vitamins such as B₁₂, B₆, pantothenic acid, folic acid, biotin, and ascorbic acid (vitamin C) have not been established. There have been no reports of vitamin deficiencies affecting the health of horses in Australia. Early observations indicated that horses were not sensitive to cobalt deficiency because they continued to thrive in areas where sheep and cattle soon wasted and died from vitamin B₁₂ deficiency. Oral and parenteral vitamin B₁₂ and folic acid are commonly given to racehorses with the expectation that such supplementation will improve haematological function and their performance. However measurements of serum and erythrocyte folate and serum vitamin B₁₂ have indicated that vitamin B₁₂ supplementation does not appear to be justified and that only permanently stabled horses may require daily oral supplementation with folic acid (Roberts, 1983).

If horses have access to green pasture and sunshine it is unlikely they will require vitamin supplementation. Horses which are permanently stabled for long periods and fed a high proportion of concentrates (70%) sometimes develop inappetance which apparently responds to supplementation with B complex vitamins. It is unclear whether horses fed concentrates are susceptible to thiamin deficiency as few assessments of thiamin status have been made.

The horse apparently has a low requirement for vitamin D and normally has low circulating concentrations of 25-hydroxyvitamin D₃ which would be considered to indicate deficiency in other species. Rickets has not been reported in horses. The horse regulates its Ca metabolism primarily at the kidney rather than the intestine and intestinal Ca absorption in horses is apparently not regulated by vitamin D as in other species. Excessive administration of vitamin D to horses by injection has resulted in calcification of soft tissues, particularly the endothelium of the aorta.

Assessment of mineral and vitamin nutrition

Apart from analysis of diets, measurements of mineral and vitamin concentrations in blood, tissues and urine may be used to assess nutritional status. However the critical values indicating deficiencies or excesses for many minerals and vitamins in horses have not been established. There is a positive correlation between dietary Ca intake and urinary Ca excretion in horses, and urinalysis provides a useful indicator of Ca nutrition and whether P intake is excessive (Cagle et al. 1982). Blood glutathione peroxidase activity and selenium concentration is a good indicator of selenium nutrition (Cagle et al. 1978). Plasma copper and zinc concentrations are unreliable indicators of deficiencies since the levels may be affected by age, seasonal variations and other factors (Auer et al. 1988). However elevated plasma zinc concentration is a useful indicator of zinc toxicity (Eamons et al. 1984). The usefulness of plasma vitamin D metabolites for diagnosis of vitamin deficiency and toxicity has not been established. The values for serum and erythrocyte folate and serum vitamin B₁₂ indicating deficiency of these nutrients are unknown (Roberts 1983).

SUMMARY AND CONCLUSIONS

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The cornerstone of the Australian horse industry is the breeding of horses and it has been shown that satisfactory performance of brood mares can be achieved on subtropical semi-improved pastures. However most horse breeding enterprises are located in areas where mares graze temperate pastures and the role of these pastures in influencing reproductive performance is yet to be determined.

High levels of athletic activity are demanded by owners and trainers of racing and performance horses but few definitive studies of the energy and protein requirements of these horses have been conducted in Australia. The studies of the levels of digestible energy available from rice by-products suggest that rice can be considered as an alternative energy source to the traditional energy sources oats, barley and maize.

Toxicants which occur in feeds consumed in stables or by grazing horses have been discussed. Equine toxicoses can also result from intake of water, The metabolism of many of these compounds in horses is poorly understood as is also the effects of sub-clinical intakes of toxicants on equine performance. Skeletal disorders represent the most important mineral problems in Australian horses, Some progress has been made in assessing nutritional status from the prediction of mineral deficiency from blood, tissues or urine but the prediction of vitamin D or vitamin B₁₂ deficiency represents a continuing challenge for equine nutritionists.

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