

SPELLING

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Short spells of 2-6 weeks have little effect on cardiovascular and muscular fitness. However, whenever exercise level is reduced, bone is lost from the skeleton. Overall bone size does not change, but porosity increases as minerals, especially calcium, are lost. Lack of exercise is the most significant factor stimulating bone loss, leading eventually to osteoporosis. Stall rest is not indicated except on veterinary advice because such confinement can result in resorption of bone. If a horse is being spelled to alleviate pain from shin-soreness, a recurrence frequently occurs as soon as exercise is resumed, if the horse is confined such that exercise is prevented.

To avoid bone damage, calcium intake must be maintained and exercise should be reintroduced gradually. As a rule of thumb, for every month of conditioning lost, the horse should drop back a month in work load. Nutrition management also affects bone density. If chronic deficiencies and imbalances occur in the diet, both bone density and peak performance can be adversely affected. Diets which contain excessive amounts of raw grains, chloride or sulphur, and those that are deficient in sodium and potassium, reduce body calcium, phosphorus and magnesium levels. Young horses on such diets lose bicarbonate and calcium in the urine and long term this can lead to bone demineralization and weakening of the skeleton. Increasing the 'available' calcium in the diet minimises the reduction in bone density in during early training. The 'availability' of minerals is determined by the form and level of all other minerals in the diet. Chelation, the process by which minerals are bound to amino acids, increases hoof growth and hip height in yearlings when compared to those on inorganic mixtures of minerals. Young horses on unbalanced diets also suffer more acidosis and their blood glucose levels take longer to recover after fast exercise.

Diseases caused by calcium deficiency may be clinical or subclinical. The classic disease is 'big head' or 'bran disease', but calcium deficiency has also been linked to ruptured tendons, spontaneous fractures, nasal discharges, tying up, poor performance and reduced exercise tolerance. If the body calcium stores become depleted, it can take up to 12 months for levels to be restored.

Requirements increase when horses are spelled, but even when in full work many horses are calcium deficient, despite the widespread use of supplements. However, as well as the absolute amount of calcium in the diet, the ratio of calcium to phosphorus is critical. Hence a calcium deficiency may be due to inadequate levels in the diet; high dietary phosphorus or a reduced ability to absorb calcium.

Horses may begin spelling in a state of calcium depletion for the following reasons:

- grains and bran are high in phosphorus;
- high grain diets increase body acid levels and when this acid is excreted in the urine, it drags calcium with it;

- many pasture grasses are high in phosphorus and contain oxalates, bran contains phytates. Phytates and oxalates bind calcium - preventing absorption. Oxalates are present in kikuyu, buffel grass, pangola, green panic and setaria. Horses on these feeds may require up to 100 grams of extra calcium per day - the equivalent of 1/3 kg of lime.

Longer spells of between 2 and 12 months are generally prescribed for horses recuperating from surgery or more serious athletic injuries. Injury and surgery both increase body tissue breakdown and recovery is hastened if a balanced, highly digestible, complete feed is supplied.

Pasture: It is common practice to remove shoes or racing plates and turn spellers out onto pasture. Most racing diets contain a minimum amount of roughage and a sudden change to a largely roughage diet can cause horses to lose condition or develop deficiency states. Roughage digestion depends on fermentation by the millions of bacteria that live in the hindgut. On a low roughage diet (less than 3.5kg per day) the number of these organisms is low, so when horses are suddenly changed to a largely pasture diet, they are unable to fully digest the grass - leading to reduced protein and energy intake.

Pasture and hay grown in areas with soil deficiencies often have similar deficiencies. If horses are grazing deficient pastures and being fed hay grown on the same soils, the problems are compounded. Analysis of both pasture and soil will yield valuable information and allow the development of management strategies to address nutrient imbalances.

In group feeding situations supplements can be difficult to control. 'Aggressive' feeders who consume other horse's feeds may receive supplements not intended for them. If all feeds contain the same supplement then 'greedy' feeders may receive a double dose if they rapidly consume their own feed followed by that of their friends.

The incidence of colic and choke is also higher in group feeding systems. Gastro-intestinal emergencies are reduced when horses are changed from a pelleted to an extruded feed. This is thought to be due to a slower rate of feeding and more saliva production with extruded feeds.

Biotin: Essential amino acids are of equal importance for hoof growth. Stable floors, hard work and frequent plating exact a toll on hoof quality and strength. Added to this is the increased body acid level associated with raw grain training diets. Crumbly white lines, dropped soles and wall cracks have all been linked to high grain diets. Trace minerals such as zinc and biotin also improve hoof wall strength and the diet must include these. Because hoof growth is slow and the benefits of biotin - in terms of hoof quality and strength - are not seen for up to 9 months, it is prudent to maintain intake during spelling.

Oil: Similarly, to receive the advantages of added oil, it must be fed for at least 3 weeks. It takes 3 weeks for the intestine and muscles to fully absorb and utilise oils. Although all oils offer cool, safe energy and delay the onset of fatigue during

exercise, only Omega 3 oils improve circulation and oxygen delivery by improving red blood cell function. Red cells have a turnover of around 3 weeks in the racehorse, so Omega 3 oils must be fed for 3 weeks before the new Omega 3-enriched red blood cells enter the circulation.

When pasture is good, the hard feed does not need to provide a lot of energy and a concentrate may be all that is required. Excessive weight gain should be avoided as it prolongs the time to reach racing fitness after a spell. Diet analysis is a valuable management tool, allowing accurate measure of energy and protein levels and whether deficiencies and /or excesses are occurring.

'Shin-soreness' or 'bucked shins' occurs in 70-80% of all 2 year old Thoroughbred racehorses during their first preparation. It is thought to be a response by immature bone to the acute stress of fast exercise. Although damage is not permanent, the recuperative period accounts for 12,000 lost training days and millions of dollars annually. The major risks in order of precedence are high-speed work; immaturity; too much work too soon and low bone strength/density.

The cannon bone of the young racehorse responds to exercise by producing new bone. This new bone is low density and fatigues sooner than more mature bone. When bone fatigues it deforms, so shear forces occur between the areas of new, low mineralised bone and older fully mineralised bone. The resulting inflammation causes pain and swelling. If the stress is not severe it stimulates new bone production, if severe, it leads to type 2 and 3 shin soreness.

There are 3 types of disease:

Type 1: Occurs after exercise in horses exposed to fast exercise for the first time, irrespective of age. Horses are sensitive to any pressure on the front of the cannon bone. Type 1 generally responds to reduced exercise intensity or rest - allowing the newly formed bone time to mature before it is loaded again with concussive forces of fast work.

Type 2: is a chronic form of type 1 that has failed to heal. There is a hard painful callus on the front of the shin. These respond to reduced workload, but may be refractory.

Type 3: Is characterised by fractures on the front and sides of the bone. These usually heal with rest, but surgery may be necessary.

Other sites which may be affected include the carpal bones, femur, humerus, sesamoids and the bone of the foot. If both legs are affected or several sites are injured, there may be gradual loss of performance, but no lameness. Local pain, tenderness, swelling and Xray changes may or may not be present. Scintigraphy or bone scans give the most consistent and earliest means of diagnosis. Rest and reduced-intensity, controlled exercise shorten rehabilitation time. Bone scans have shown that it takes one to 6 months for the bone to return to normal. Too rapid a return to too high a level of exercise will result in recurrence.

Prevention is important. The rate of response of the young skeleton should be addressed when exercise regimes are formulated. When a young horse enters training, bone is first resorbed and density is lowest around 8 weeks into training - making the young horse least able to withstand bone strain at the time that fast work is being introduced. Bone strength is proportional to the density cubed. Density is increased about 16 weeks into the preparation. Submaximal exercise does not increase the density of bone and training at high speeds above 12m/sec is necessary to increase bone mass and density. As speed increases, so does the risk of bucked shins. A trade-off is required between maximising the bone response and minimising the risk of bucked shins.

Because a small number of high intensity loads per day enhance bone growth, the distance over which horses gallop need only be 200 to 400m. Once a loading has been repeated often enough for the bone to adapt, subsequent repetitions will not increase the adaptive response. A reduction in exercise-induced lamenesses – without compromising bone density - can be achieved by limiting excessive amounts of fast work. Further studies are necessary to determine the minimum amount of fast work necessary to improve strength of the cannon bone and the duration and speed that provides an unacceptable risk of causing bone damage.

As a result of training, bone enlarges and increases in density, so for a given load, there will be less strain. The strain required to stimulate bone density and strength is 1500-3000 $\mu\epsilon$. Below 100-300 $\mu\epsilon$ resorption occurs. The faster the speed, the greater the bone's response, so it is important to control the speed to a slow gallop for 4-5 weeks and progressively increase speed every 4-5 weeks. At full gallop the strain on a mature thoroughbred is around 3500 $\mu\epsilon$, whereas it may be more than 5000 $\mu\epsilon$ in a 2-year old.

The track surface should allow the foot to rotate rather than slide. The incidence is about twice as high and occurs with fewer miles of fast work on a dirt than either wood fibre or grass.

Calcium: Supplements: Although horses adapt to pasture diets, for optimum growth, performance and fertility, nutrient supplementation is required. The most common trace mineral deficiencies involve selenium, iodine and copper. Deficiencies of minerals such as iron, zinc, calcium, phosphorus, magnesium, copper, potassium and vitamins A, B, D and E also commonly occur in pastured horses. Unfortunately the clinical signs caused by deficiencies are often similar to those caused by excesses eg iodine and vitamin A. Of equal importance is the potential for minerals to interact with each other.

An excess of zinc will prevent copper absorption so copper deficiency occurs, even though intake is adequate. All the possible interactions are not fully understood, but what is known is that supplementing individual minerals ignores these relationships.

The effects of many borderline imbalances are often sub clinical - ie, they do not affect overall health, but they do reduce performance and ability to reach genetic

potential - and they are difficult to diagnose. However intakes above requirements cannot improve performance and, for many vitamins and minerals, a surplus is as dangerous as a deficiency.

The safety margin for selenium, iodine and vitamin A is very narrow. In addition, the higher the desired level of performance and growth, the narrower the optimal range of a nutrient. A recent survey found that 78% of horses fed supplements receive excess energy, protein, calcium, phosphorus, copper, manganese and zinc - at levels high enough to unbalance the diet.

Excessive levels of individual nutrients occur up to 10 times more frequently when supplements are used - and deficiencies may still exist. In addition, when several supplements are used in an attempt to meet individual requirements, overlap can occur and the risk of subclinical excesses and toxicities increases.

Essential amino acids: Although energy levels of hard feed do not need to be high when pasture is good, the vitamin, mineral and amino acid balance of the feed is important - for all spellers, but especially for 2 year olds. If essential amino acids are not provided, horses lay down more 'cover' and less muscle. The percent protein of a feed does not indicate whether all essential amino acids are included.

Muscle and bone protein building is so specific that even if the diet provides adequate levels of 9 of the 10 essential amino acids, but only 50% of the 10th, protein synthesis will be reduced by 50%. If one essential amino acid is deficient, the others cannot be used and are stored as fat. Analysis of diets given to horses that are laying down too much cover - instead of gaining in height and muscle development - have revealed essential amino acid deficiency.

Recent research has discovered that even if the diet contains recommended levels of essential amino acids, they may not be available to the horse. The absorption of amino acids is affected by whether the feed is digested in the small intestine or the hindgut. Protein needs to be digested in the small intestine. Any not digested passes into the hindgut where it is converted to ammonia and lost to the horse. So although the diet may meet essential amino acid requirements, these are lost to the horse if they escape digestion in the small intestine.

Feed processing: Fortunately recent research has developed processing methods which increase digestion in the small intestine to over 90%. Refining and advancing time-honoured practices, wet-extrusion cooks grains and protein meals at the correct temperature and for the precise time required to improve digestion without damaging nutrients. Older methods - such as boiling barley, grinding corn, crushing oats and pelleting feeds - damage nutrients because temperature and duration of cooking were not known or controlled and have little effect on digestibility, as shown in the graph below.

Group feeding: Mitavite combines time-honoured methods with the latest scientific research to produce a range of fully pressure-cooked and extruded feeds. Risks associated with supplements are eliminated and natural chelated mineral proteinates, as used in Mitavite feeds, prevent vitamin and mineral interaction and improve absorption.

Feeds such as Mitavite Athlete (a concentrate containing balanced levels of all vitamins, minerals and essential amino acids) are ideal when pasture or roughage quality and quantity is good. The major nutrients that should be maintained during spelling are included. Mitavite Athlete has been formulated to be fed with hay, chaff or pasture as a nutrient-dense concentrate containing all supplements. It can also be combined with grains for extra energy for horses entering pre-training prior to going back in.

Mitavite Breeda contains energy, essential amino acids and meets vitamin and mineral requirements to restore body levels depleted during training and improve post-operative recovery in horses convalescing after surgery.