EQUINE DISEASE QUARTERLY

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COMMENTARY

IN THIS ISSUE

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College of Agriculture, Food and Environment
Department of Veterinary Science

LLOYD'S

Lloyd's—An international insurance market for bloodstock and other specialist insurance.

Proper nutrition is key to producing and maintaining horses of all types, while poor nutrition or feeding management can increase the risk of various health disorders. In this issue, Dr. Clair Thunes focuses on refeeding the starved horse, and Dr. Carrie Finno addresses vitamin E deficiency. Both articles highlight consequences of inadequate or imbalanced nutrition.

Nutrient deficiencies may be primary or secondary. Primary deficiencies result when the diet contains inadequate amounts of one or more nutrients. Starvation can occur when feed resources are scarce, producing a deficiency in calories, protein, and nutrients. Primary deficiencies in specific nutrients, such as vitamin E, occur with improperly balanced diets. Secondary deficiencies occur when the diet is nutritionally adequate, but some other factor affects nutrient intake or absorption. For example, starvation can occur if a horse is unable to eat due to a mouth injury, even though feed is available. Secondary deficiencies may be caused by dietary substances or medications that interfere with the absorption of a specific nutrient. Excessive concentrations of dietary zinc inhibit copper absorption and lead to copper deficiency in growing horses despite adequate dietary copper.

From a nutritionist's perspective, identification of dietary deficiencies requires a diet evaluation. This involves determining the amount and composition of the feeds provided. Commercial feed laboratories offer affordable and rapid analysis for many important nutrients including protein, calcium, phosphorus and several trace minerals. In contrast, selenium, iodine, and most vitamins require a specialized laboratory. It is important to remember that most nutritional deficiencies manifest after months to years of dietary inadequacy. Therefore analysis of the current diet may not necessarily reflect a past diet.

MATT BARTON

Due to variation in nutrient pools within the body and stability of various minerals and vitamins, laboratory submission of whole blood, serum, or liver may be required. Prior to sample collection, the laboratory should be consulted to confirm the type of sample needed and the optimal conditions for shipping samples to the laboratory. Dr. Finno indicates that baseline concentrations of circulating vitamin E may indicate the need for supplementation. However, blood concentrations

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INTERNATIONAL

Third Quarter 2020

The International Thoroughbred Breeders Federation, International Collating Centre, Newmarket, United Kingdom, and other sources reported the following equine disease events.

Thailand and Malaysia recorded outbreaks of African horse sickness (AHS). Thailand reported outbreaks in August (one case in a horse) and September (two cases in captive Chapman's zebras). Malaysia confirmed its first ever incursion of AHS in August; five horses on a single premises. Affected equids in the outbreaks in both countries were euthanized.

Belgium, France, Germany, Italy, Sweden, and the USA confirmed outbreaks of equine influenza. These ranged from one (Belgium, France and Italy), three (Sweden), four (Germany), to at least nine in the USA, where the disease is endemic.

Strangles is considered endemic in many countries with outbreaks reported from Belgium (two), Canada (one), France (nine), the Netherlands (12, one involving 15 cases), Switzerland (eight), and the USA (41 outbreaks in 17 states, the majority with multiple cases).

Equine herpesvirus 1 (EHV-1) related diseases are endemic in most countries. Cases were confirmed in France, Japan, Republic of South Africa (RSA), Sweden, Switzerland, the UK, and the USA. Single outbreaks of respiratory disease were recorded in France, RSA, and the UK. One case of EHV-1 abortion was confirmed in Japan in an unvaccinated mare. EHV-1 neurologic disease was reported by Sweden (single case), Switzerland (three outbreaks), and the USA (seven outbreaks involving five states, with two outbreaks in California and Maine); the majority involved single cases.

Equine herpesvirus 4 (EHV-4) respiratory disease was recorded by France (20 outbreaks, mostly single cases), Japan (one outbreak involving eight unvaccinated horses) and the Netherlands (five outbreaks, one involving seven 4-5 month old foals). Switzerland reported an outbreak of EHV-4 neurologic disease.

Belgium and the USA confirmed cases of equine herpesvirus 2 and/or 5. Infection with EHV-2 was diagnosed in a 2-month-old foal in Belgium, and numerous infections by either virus were diagnosed in several states in the USA, some associated with respiratory disease.

A case of adenovirus infection was reported in a foal in Ireland.

The USA confirmed four outbreaks of equine infectious anemia, three in Texas, one involving two cases, and one in Georgia. Three outbreaks involved single cases.

Equine piroplasmosis was recorded in Finland (one case), RSA (disease is endemic) and Switzerland (one outbreak).

Denmark reported the isolation of *Taylorella equigenitalis*, the cause of contagious equine metritis, from three stallions.

Equine coital exanthema (equine herpesvirus 3) was diagnosed in Argentina (two outbreaks involving 29 Thoroughbreds) and the USA (one case).

Belgium confirmed single cases of leptospiral abortion on two premises. A case of *Leptospira* associated recurrent uveitis was diagnosed in a stallion by France.

Salmonellosis was reported by Switzerland (two outbreaks) and the USA (10 cases, two involving serogroup C1, five serogroup D1, and three weren't serogrouped).

France confirmed 19 outbreaks of rotavirus diarrhea in foals, the majority were single cases. A total of 17 cases were diagnosed by the USA, the majority in 60- to 90-day-old foals in Kentucky involving G3 and/or G14 genotypes.

One case of proliferative enteropathy caused by *Lawsonia intracellularis* was reported by the USA.

Canada (four outbreaks, all single cases) and the USA (17 outbreaks in 10 states, one involving 10 cases) confirmed outbreaks of equine neorickettsiosis (Potomac Horse Fever).

Eastern equine encephalomyelitis was reported by Canada (two outbreaks, each involving single cases) and the USA (104 cases in 11 states), with highest numbers in Michigan (33), Wisconsin (20), and Florida (13).

West Nile encephalitis was recorded by Canada (one case), France (three outbreaks, one case each), Germany (eight outbreaks), Hungary (one outbreak), Italy (nine outbreaks), Portugal (one case), Spain (13 outbreaks, three were single cases), and the USA (27 cases in seven states, four on one premises).

Equine Disease Quarterly

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A case of rabies was confirmed in a Warmblood horse in Maryland, USA.

Switzerland confirmed two outbreaks of Borna disease, and one outbreak of anaplasmosis.

Vesicular stomatitis continued to spread in the USA in the third quarter of 2020, extending to Arkansas, Missouri and Oklahoma, in addition to five previously reported states. A total of 198 equine outbreaks were recorded, all associated with the Indiana serotype.

Rhodococcus equi related diseases were confirmed by the USA.

Prepubic Tendon Rupture

The prepubic tendon extends from the brim of the pelvis and joins with the abdominal muscles to support the abdomen and abdominal viscera. Mares can rupture (tear) the prepubic tendon, either partially or fully, and the associated muscles of the abdominal wall. Prepubic tendon rupture is an uncommon but serious condition in late pregnancy that may have a poor prognosis and can result in death of the mare and fetus.

The exact cause of prepubic tendon rupture is not known, however, the condition is more common in older mares that have had multiple pregnancies, draft breeds, mares carrying twins, and mares with placental abnormalities such as hydrops (hydroallantois or hydroamnion).

Mares with prepubic tendon ruptures exhibit signs of colic-like pain, elevated heart and respiratory rates, and reluctance to move or lie down. Changes in the shape of the ventral abdomen (underbelly) may be first noted, followed by a rapidly progressive swelling (edema) that can extend from the udder towards the chest. The udder may be swollen and teats may point toward the mare's head instead of downwards, and blood may drip from the teats. Touching the ventral abdomen is often resented. Some mares will adopt a 'sawhorse' stance with a tilted pelvis and elevation of the tailhead. Other mares may die after a short illness. Diagnosis of prepubic tendon rupture is difficult to confirm and is usually based on characteristic clinical signs.

The choice of therapy for mares with prepubic tendon rupture varies. It will depend on the extent of the rupture, ability to control the mare's pain, and stage of gestation. The relative value of the mare and foal may also be considered. Complete rupture of the prepubic tendon has a poor prognosis for the mare because the tendon cannot be surgically repaired.

Conservative therapy will usually involve restricting the mare to stall rest, carefully applying 'belly bandages' to support the ventral abdomen, and alleviation of pain using a variety of drugs. Progestins may be given to keep the uterus in a quiet (not contracting) state. Close monitoring of the fetus to detect fetal stress is required with frequent ultrasound examinations via the mare's abdomen to monitor fetal heart rate and activity.

Stage of gestation is an important factor when therapy options are being considered. If the mare is close to term (330 days of gestation or later), induction of labor or a caesarean section may be considered. If the mare is induced, then foaling must be assisted as she has limited or no ability to contract abdominal muscles during active labor. If the foal is successfully delivered, it is considered at high risk of developing problems such as neonatal sepsis. Colostrum supplementation may be needed, and the foal may not be able to suckle the mare due to severe udder swelling.

For mares that rupture the prepubic tendon before 330 days of gestation and in which pain can be managed, a decision may be made to support the mare so the fetus can reach 330 days of gestation or longer. The last part of pregnancy is very important in determining viability of the foal, as most of the fetal maturation that ensures the foal is ready for life outside the uterus happens in the last 10 days of gestation. For mares in which pain cannot be well managed, euthanasia should be considered.

Mares with prepubic tendon rupture that survive foaling or caesarean section should not be bred again. Some mares that survive foaling may subsequently need to be euthanized due to intractable pain.

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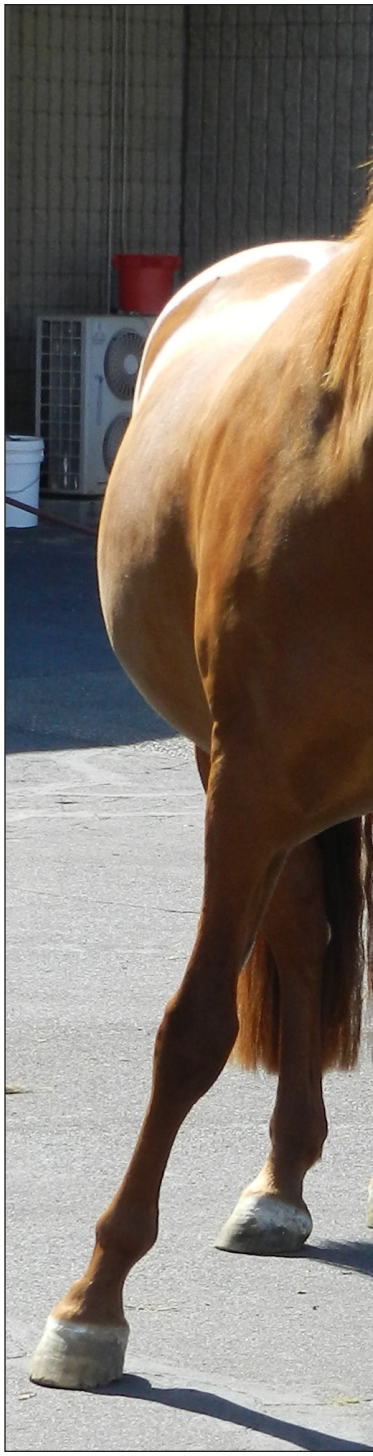

Abnormal stance of a horse with equi
degenerative myeloencephalopathy
deficiency.
CARRIE FINNO

Consequences of Vitamin E Deficiency

ine neuroaxonal dystrophy/equine
(eNAD/EDM) from vitamin E

Vitamin E is an essential nutrient for horses. This antioxidant helps maintain normal neurological function by limiting the damage caused by oxidative stress and free radicals. The best source of vitamin E is fresh green grass. Horses that graze on lush green pastures often have adequate access to vitamin E. However, many stabled horses are not maintained on pasture. Vitamin E levels decrease and eventually are lost as soon as grass is harvested, so even the highest-quality hay will not meet vitamin E requirements. Some horses maintained on diets deficient in vitamin E can experience significant health consequences.

Vitamin E helps a horse's muscles and nerves function properly. A dietary deficiency can lead to three specific diseases: (1) equine neuroaxonal dystrophy/equine degenerative myeloencephalopathy (eNAD/EDM), (2) equine motor neuron disease (EMND) and (3) vitamin E deficient myopathy (VEM). These diseases are typically diagnosed based on clinical signs, exclusion of other disorders, and low concentrations of vitamin E in the blood.

Equine neuroaxonal dystrophy/equine degenerative myeloencephalopathy occurs in young animals and has a genetic predisposition. Affected horses may stand with forelimbs too far apart (or too close together), have difficulty navigating curbs or hills, or lack coordination while walking and making tight turns. Signs of the disease can vary, some horses show mild performance issues, while others are severely debilitated. Although the exact cause of the degeneration is unknown, a specific interaction between genetics and nutrition during the first few years of life is required for this disease to manifest.

Researchers in the Finno Laboratory are working to develop a genetic test for eNAD/EDM. Until one is available, the recommendation is to provide vitamin E supplementation to horses that do not have regular access to pasture, especially pregnant mares and newborn foals. This may not eliminate the disease, but it appears that less severe neurological signs may develop if foals receive supplementation during the early stages of life.

Equine motor neuron disease typically occurs in older horses that have been vitamin E deficient for more than 18 months. The disease affects lower motor neurons, which are nerves that supply the direct neurological input into all muscles. In their absence, the associated muscles atrophy, resulting in the clinical signs of weakness and weight loss characteristic of this disease. Affected horses may carry their heads low and lie down for longer periods. Research from Cornell University has shown that approximately 40% of horses affected by EMND improve with vitamin E supplementation, 40% stabilize but remain disfigured, and 20% progress in disease severity.

Vitamin E deficient myopathy is typically found in horses with a shorter duration of vitamin E deficiency. Affected horses exhibit muscle weakness, low muscle vitamin E concentrations and mitochondrial alterations (i.e. changes to the "power house" energy storage unit of the cell) in skeletal muscle but show no evidence of neurologic impairment. With proper supplementation, affected animals are generally able to recover within three months.

These diseases are currently the only conditions definitively associated with vitamin E deficiency. At this time, nothing is known about vitamin E metabolism in horses, the appropriate dosage, or its actual efficacy in preventing or treating other neurological conditions. In humans, the same enzymes that metabolize vitamin E also metabolize approximately 50 percent of therapeutic drugs, so supplementation with vitamin E could potentially alter the effects of other drugs if a horse is on multiple therapies. Therefore, while vitamin E supplementation may not necessarily be harmful with respect to other neurologic diseases, it is unlikely to be beneficial.

Since there are no effective treatments for most diseases caused by vitamin E deficiency, prevention is key. If horses are not able to access fresh pastures to graze, vitamin E must be supplemented in their diets. However, not all vitamin E supplements are created equally. For detailed information on

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vitamin E supplementation, please refer to a recent issue of the UC Davis Horse Report: https://ceh.vetmed.ucdavis.edu/sites/g/files/dgvnsk4536/files/inline-files/Horse_Report_Fall_2018_web.pdf. Baseline blood vitamin E concentrations should always be assessed before initiating any vitamin E supplementation. Owners should consult with their veterinarians to determine if vitamin E supplementation is required and identify the best supplementation regimen for their particular horse.

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Re-feeding the Starved Horse

Finding yourself in the position of providing care to a starved horse can be an emotional situation for any horse enthusiast. The desire to do everything possible to return the horse to full health, including providing ample nutrition, can be the initial impulse. Sadly, the desire to feed the horse well can have catastrophic consequences resulting in a re-feeding syndrome that may result in the need for euthanasia. While it may be tempting to provide unlimited access to feed, when it comes to feeding starved horses, less truly should be the rule of thumb.

Horses rely on body stores of inert carbohydrate and fat to fuel the large number of metabolic processes within the body. These stores are constantly replenished through the diet. In a horse that is starved, these stores become depleted and so instead, protein is utilized for energy. All protein in the body is actively contributing to important functions and is not stored for the purpose of providing energy. In this scenario, the body is not picky about what source of protein gets used for energy. Therefore, as protein is burned as a fuel source, skeletal muscle and vital organs become negatively impacted.

When a starved horse is "re-fed" and provided carbohydrates, in particular glucose, blood insulin increases. This helps move glucose from the circulation into cells and with it electrolytes. This can result in inadequate levels of key electrolytes such as phosphorus, potassium and magnesium in the circulation as well as leaving red blood cells without adequate energy. These red blood cells are then unable to adequately release oxygen to various tissues resulting in heart, kidney and respiratory organ failure and ultimately death.

Research studies have shown that a greater success rate is achieved when starved horses are rehabilitated using forages containing low non-structural carbohydrate (NSC) as this limits the insulin response. If using grass hays, they should be tested for NSC content and ideally have an NSC content of less than 10 percent. If only untested grass hay is available, feeding alfalfa may be a better choice as alfalfa has a low NSC content and also tends to provide good amounts of magnesium and phosphorus.

Very small amounts of feed should be fed that provide no more than about 50 percent of the horse's calculated daily digestible energy (DE) requirement. Different research studies have used different feeding protocols. One fed alfalfa at 50 percent of the calculated daily DE requirement for the first 3 days of re-feeding and then increased the amount to 75 percent for days 4 and 5 before increasing to a full 100 percent on day 6. Another study fed frequent handfuls of grass hay for the first day and then provided netted hay that was hung outside the stalls so that the horses had to eat the hay through the bars. These researchers introduced 0.5 pounds of complete feed and increased this by the same amount every 3 days until they were being fed no more than 3 lbs at any feeding.

Re-feeding syndrome can develop as quickly as the first day of re-feeding or may not become apparent for 3 to 4 weeks. The keys to successful rehabilitation lie in starting off with very small meals that provide limited calories and utilizing forages and feeds with an NSC content at or below 10 percent. Sticking to these guidelines despite the natural desire to want to lavish the horse with food will provide the greatest chance of success in nursing the starved horse back to health.

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COMMENTARY: *continued from the front page*

are not useful in assessing the status of many nutrients. For some, homeostatic mechanisms maintain relatively consistent blood levels, even in the face of significant dietary deficiencies. For nutrients such as selenium, indicators of functional status may be more useful than serum selenium concentrations alone. Even for nutrients whose levels fluctuate in the blood with dietary intake, a blood level is only useful if it can be interpreted in relation to horse health. Laboratories should have established or referenced normal ranges to evaluate blood mineral or vitamin concentrations. Ideally, reference ranges should be developed from normal healthy horses of different physiological states, as age and physiological function (gestating, exercising, growing) affect nutrient needs. Unfortunately specific reference ranges for blood constituents in equines are very limited.

Horses are often described as fragile, but they are also extremely resilient and adaptable. Horses evolved to deal with times of food abundance as well as food scarcity. Short periods of food scarcity may result in nutritional deficiency; food abundance allows for restoration of body reserves. Clinical diseases only typically will develop as a result of chronic deficiencies. As both articles in this issue point out, overzealous feeding and supplementation can have negative consequences. For this reason, dietary changes, even those aimed at correction of nutritional deficiencies, should be made gradually. Perhaps, optimal horse feeding is more like the tortoise and less like the hare.

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Abnormal stance of a horse with equine neuroaxonal dystrophy/equine degenerative myeloencephalopathy (eNAD/EDM) from vitamin E deficiency.
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