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EQUINE ROUNDS

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New York State College of Veterinary Medicine ● Cornell University

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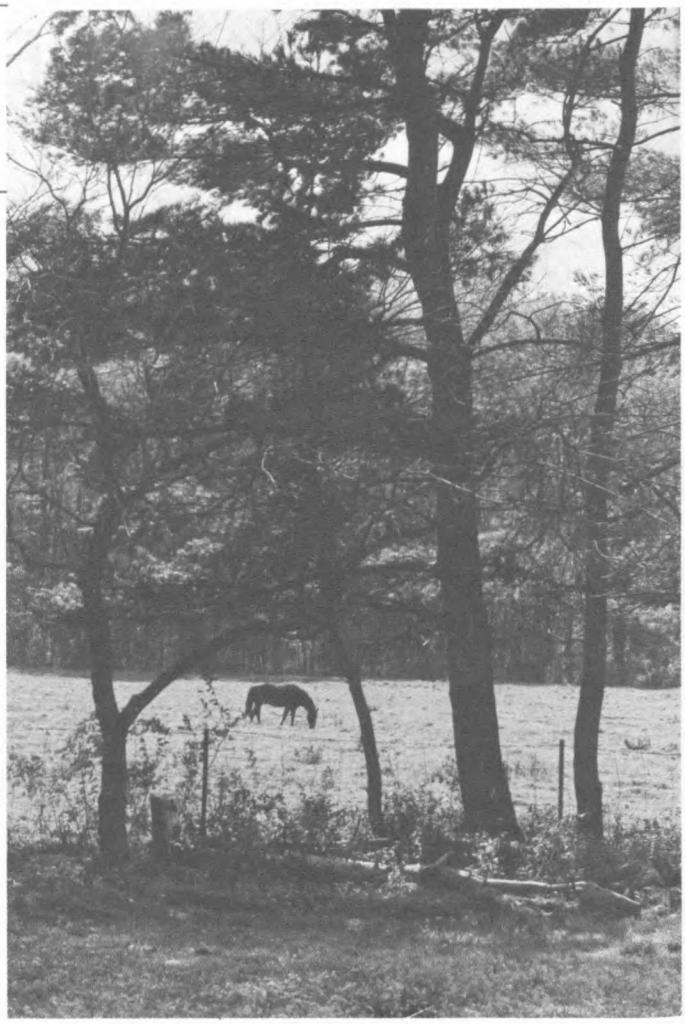

CAUTION: HAY

By Mike Golden '89

Special thanks to Dr. John King and Dr. Harold Hintz

Hay commonly makes up a large part of a horse's diet and for this reason it is critical that we feed a high quality, safe product. Occasionally though, organisms within the hay may endanger the horse's health. In considering safety, it is important to remember that by feeding moldy or dusty hay, we aggravate the horse's respiratory system, thus accentuating any pre-existing problems such as coughing or heaves. Far less frequently, feeding such low quality forages may produce any one of the following conditions.

Moldy hay may contain a product that limits the availability of its vitamin E. The importance of reduced vitamin E intake will vary according to the age of the animal. Younger animals have a higher requirement for vitamin E and show clinical signs of deficiency more often. A deficiency of vitamin E in younger animals may be seen as White Muscle Disease (WMD), a condition in which a deficiency in vitamin E and/or selenium leads to muscular degeneration. Foals may be born with this condition if there is deficient placental transfer of vitamin E. Reduced vitamin E in mares' milk can produce WMD in nursing foals. In weanlings, direct intake of moldy hay which is vitamin E deficient may produce WMD. In all cases, early recognition and veterinary medical treatment is critical for recovery.

Sweet clover is a legume primarily grown in Canada due to its hardiness, but it is used less frequently in the United States. Moldy sweet clover causes internal hemmorhage. This condition is a result of the action of the mold on cumarol, a natural compound in sweet clover that is converted to dicumarol, an anticoagulant. Moldy sweet clover poisoning is a potentially fatal disease unless recognized and treated with vitamin K and blood transfusions.

A soil fungus, Rhizoctonia legumicola can infect legumes, especially red clover, during conditions of high humidity. This organism produces an alkaloid, slaframine, that causes excessive salivation in the horse. Exposure to the fungus can occur with legumes fed as pasture or more commonly, as hay. With a change to unaffected pasture or hay, horses will recover.

Blister beetles, primarily found in southwest alfalfa hay, contain a poisonous subtance called "cantharidan" which is very irritating to the digestive tract and causes severe kidney damage. Ingestion of more than a few beetles could cause acute signs of shock and death within hours. If less toxin is ingested, the horse will show signs of colic and mouth irritation and may be helped with prompt medical attention.

The feeding of silage has proven useful in horses where allergic conditions such as heaves necessitate dust-free management. Botulism, a highly fatal, paralyzing disease, caused by ingestion of a preformed toxin has appeared in horses fed big bale silage. The source of the toxin Clostridium botulinum, may gain access to the fodder via decaying organic material but more likely through soil in which the organism lives. To inhibit clostridial growth in silage a low pH of 4.2-4.6 and a high dry matter content of more than 25% must be achieved by wilting for 36-48 hours prior to ensiling. Before feeding, the aroma of the silage should be checked for any secondary fermentations that may indicate the presence of mold and potential toxin formation. It is difficult to maintain a delicate quality control on every sample fed and horse owners should be aware that this type of silage has inherent risks.

The key to avoiding dust- and mold-related problems is to follow recommended harvesting practices and store hay in a clean, dry environment. Conventional field cured hay should be dried down to a moisture of 15% to 18% for safe storage. If facilities are available for heat finishing hay, it may be baled and placed in a dryer when moisture is between 40-45%. Organic acid treatment has proven successful in controlling microorganisms in hay stored at up to 30% moisture. Organic acids, as normal by-products of digestion, will not harm horses. Adherence to these techniques, careful examination of hay before feeding, and careful use of such feeds as silage and sweet clover will make for a safe and nutritious feed.

Mike Golden begins his second year in the DVM program this fall. He is particularly interested in equine medicine.

BRACKEN FERN POISONING

By Eden Bermingham '88

With thanks to
Dr. Robert Hillman and Dr. Mary Smith

The bracken fern Pteridium aquilinum is a tall fern with erect, coarse, triangular fronds. It is widely distributed in upland pastures, recently cleared land, abandoned fields, and open woods. Horses grazing these areas may inadvertently ingest bracken fern which contains the enzyme thiaminase. This enzyme destroys the necessary B_1 vitamin, thiamin, and if ingested in large enough quantities can reduce the blood thiamin to a fatal level.

Most of the poisoning cases occur when horses are fed a poor to inadequate diet of hay containing large amounts of bracken fern. They may also accidently ingest it in their bedding. Although bracken fern doesn't usually grow on lawns, lawn and yard clippings can be dangerous when fed to horses as they may contain other potentially toxic plant species.

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The amount of bracken fern necessary to produce symptoms is very large -- generally the horse must eat hay containing more than 20 percent bracken fern daily for at least a month to produce clinical symptoms. The signs first noted are a loss of condition and weight as well as minor incoordination which is shown by a swaying from side to side as the horse is walked. This incoordination increases as the toxicity progresses. Afflicted horses are also disinclined to move and stand with their legs apart as though bracing themselves. They may sometimes assume a crouching attitude with an arched back. Widespread muscular twitches may also develop and progress into tremors so severe that the horse cannot stand. Although most affected horses lose weight, their appetite remains normal until the signs become very severe. The most significant physiological finding is a markedly lowered thiamin level in the blood with a concurrent increase in pyruvate concentration and a decrease in the number of platelet components. As the symptoms worsen, some horses may make violent attempts to get up, severely injuring themselves in the process. Death is usually preceded by convulsions and may occur several days to several weeks after the onset of clinical signs.

The treatment for bracken fern poisoning, massive doses of thiamine, has been successful in all but the most advanced cases where the horse is already convulsing. After the initial treatment, there is usually marked improvement within 1 to 2 days and a complete recovery within 2 to 4 days.

Information found in John M. Kingsbury's Poisonous Plants of the United States and Canada, Prentice-Hall, Inc., 1964.

Eden Bermingham '88 hopes to specialize in equine medicine.

BACK TO BASICS

By Luba Drouin '87

One of a horseowner's biggest concerns is the nutrition of their horse and, not surprisingly, most people are guilty of "overnutrition". One such area of concern is in the oversupplementation of vitamins and minerals. These supplements are costly, may not be necessary for most horses, and worse yet, they may cause more harm than good.

Remembering what vitamins horses require is as easy as "A-B-C-D-E" and add on K.

Vitamin A: The vitamin A content of good quality hay is usually adequate for mature horses. However, hay that has been severely weathered or stored for a few years has a much lower vitamin A level and may need to be supplemented with synthetic vitamin A. Concentrates such as corn and oats are low in vitamin A. In young growing horses where half the diet may constitute concentrates, a supplement may have to be added. A suggested guideline is 1000 I.U. of vitamin A per pound of concentrate ration. Vitamin A, like vitamins D, E, and K, is stored in the fat deposits of the body so high levels of supplementation may build to toxic levels.

Vitamin B: The B vitamins are a complex of vitamins such as thiamin, nicotinic acid, riboflavin, pantothenic acid, peridoxine, biotin, and folic acid. B vitamins are also obtained directly from feeds as well as being synthesized in the large intestine. Feeding a good quality ration will provide the horse with the necessary vitamins. However, there is some question whether a young growing horse can synthesize all of the B vitamins it needs. Many owners supplement the ration of their young horses with B vitamins.

Vitamin C: Vitamin C is produced by the horse. Whether or not a vitamin C deficiency can occur in the horse is not known. It is a water soluble vitamin and as such is readily excreted by the body, so adding it to the feed will probably cause no harm. However, adding vitamin C may be costly while providing few if any benefits.

Vitamin D: Vitamin D is an important player in the Ca + P scenario. Vitamin D is definitely needed for maximum calcium and phosphorus absorption and utilization. Without vitamin D, there is an excess amount of Ca + P excreted in the feces. It is rare that horses fed natural feedstuffs can be deficient in vitamin D. Horses receive their vitamin D from suncured hay or directly from the sun by action of ultraviolet light on compounds in the skin. As an example, a mature 1000-pound horse requires about 3300 I.U. of vitamin D per day. A daily ration of 15 pounds of suncured hay will provide 13,000 I.U./1000-pound horse, an approximately six-fold increase over his requirement.

A major concern today is excess vitamin D. This is mainly associated with oversupplementation, because natural feed stuffs rarely contain excessive amounts. Some supplements contain 800,000 I.U./lb. and coupled with the natural amounts found in feed and the fat soluble character of the vitamin, toxic levels become a possibility. Toxic levels can decrease the growth rate, cause weight loss, diarrhea, kidney damage and tissue mineralization.

Vitamin E: Good growing pastures are an excellent source of this vitamin for horses. However, effective vitamin E utilization seems to be dependent on the presence of adequate selenium, and selenium is deficient in soil in the Northeast, and therefore in the hay and pasture. The cost of this vitamin may be well worth its benefit.

Vitamin K: Plants have a high content of vitamin K and a substantial amount of this vitamin is produced by the horse. Vitamin K has an important function in the clotting mechanism. However, certain synthetic forms and/or dosages of vitamin K can be toxic.

Along with the required vitamins, there are many required minerals. The three minerals that have received much attention in recenty years are calcium, phosphorus and, especially in the Northeast, selenium.

Calcium and Phosphorus: There are still many discrepencies about the actual Ca + P needs of the horse, but most researchers agree inadequate Ca/P ratios play an important role in the cause of skeletal disease of growing horses. There are at least three conditions that must be met in order to assure proper calcium and phosphorus utilization. First, an adequate level of both Ca + P must be available in the diet. Second, the ratio between the two must be suitable. Third, vitamin D must be available in adequate amounts. Excess levels of phosphorus can lead to an inadequate level of Ca even though the level of calcium is adequate for the horse. This is because excess phosphorus ties up a certain amount of calcium and makes it unavailable to the animal for use. If alfalfa hay, which is very high in Ca, is fed, calcium supplementation is probably not necessary.

Selenium: Selenium is the newest trace mineral added to horse rations. Soil in the Northeast is deficient in selenium and therefore crops such as hay and grains raised in that area may be deficient in selenium also.

Sodium selenite and sodium selenate are the two inorganic selenium compounds that can be purchased and added to the animals' ration. Their purchase usually requires a prescription from your veterinarian but it is a good idea to supplement selenium only under the guidance of your veterinarian. There is a narrow range between the required level and the toxic level of selenium. Toxic levels can result in respiratory failure, blindness, sloughing of hoofs and death. For that reason, extra caution should be taken that selenium is used only when there is some certainty that selenium deficiency exists on the farm and that the supplement is mixed and used properly.

Iodine: Goiters have been reported in foals where the dams were fed kelp (seaweed) meal. In a case of two foals with enlarged thyroid glands from a New York State farm, Drs. J. D. Driscoll, H. F. Hintz and H. F. Schryver, recommended that the iodine supplementation be discontinued and the kelp product removed from the foal's diet. In addition, the use of kelp in the diet of pregnant mares was terminated. (There were given as much as 12 oz. daily during the last third of pregnancy and studies have shown that the feeding of 40 mg. daily to a pregnant mare could result in a goitrous foal.) Decreasing the intake of dietary iodine resulted in a gradual decrease in the size of the thyroid gland.

The basics of good equine nutrition are good quality hay and concentrates for energy as needed. Rapidly growing young horses and lactating mares may require additional supplementation of vitamins and minerals, but horses not under such stresses and fed maintenance diets won't require much additional supplementation. Many people tend to oversupplement their horse's diet. In fact, they may be spending a fortune on supplements that are not only unnecessary, but potentially harmful.

Luba Drouin is beginning her final year in the DVM program and is interested in equine practice after graduation.

SELENIUM DEFICIENCY: A NUTRITIONAL PROBLEM FOR FOALS

by Andrea Looney '89
With Thanks to Dr. William Rebhun

The horse's diet must be able to develop and maintain the highly supportive and propulsive framework of the equine body, promoting the sound bone and muscle growth that are fundamental to a long and successful athletic life. The horse depends upon a high quality feeding program and vitamins and minerals play an increasingly vital role in this program. One significant vitamin-mineral pair, vitamin E and selenium, are rapidly gaining in importance to horsemen since dietary deficiencies of these nutrients in the equine diet can cause the nutritional muscle degeneration known as White Muscle Disease (WMD).

Andrea Looney '89 is a first year student interested in equine or companion animal practice after graduation.

Selenium and vitamin E help stabilize cells and cell membranes, thus maintaining the integrity of individual cells such as muscle fibers. Both prevent the buildup of toxic waste products, peroxides, within the cells. Vitamin E is a primary defense mechanism, inhibiting or at least minimizing the formation of lipid peroxides, while selenium functions as a component of a tissue enzyme (glutathione peroxidase) which destroys peroxides by converting them to less toxic alcohol compounds. With adequate amounts of both of these "antiperoxide" nutrients in the body, disruption of cellular membranes and injury to tissue as a whole is averted.

Problems relating to inadequate selenium arise mainly in the Northeast and Great Lakes states where the soil is selenium deficient. Animals fed the selenium-deficient grains and forages grown on this soil frequently develop White Muscle Disease. According to a recent publication by Drs. Stephen Dill and William Rebhun at Cornell, this nutritional disease characterized by myo-(muscle) degeneration, appears to pose a significant threat to foals. Weanlings fed solid feed and the foals of mares fed diets of locally grown crops and feeds are those most often afflicted with the disease.

As the result of an inadequate dietary intake of selenium, tissue peroxides accumulate, causing muscle protein coagulation and fragmentation of muscle cell membranes. Fibers become calcified, swollen, and undergo necrosis. Affected muscle masses include those of the front and hind limbs, muscles of the heart and ribs (including the diaphragm), and muscles of the neck and tongue.

The first sign of WMD is usually some form of muscle dysfunction, often weakness. Affected foals have trouble rising or may be unable to rise without assistance. If ambulatory, a stiff, awkward gait may be observed and the limb muscles may be tense or painful. Young foals may lose milk via the nose and mouth while nursing; older foals fumble with or drop feed while eating, with roughage accumulating in the cheek cavities. These are clinical presentations of dysphagia, or difficulty in swallowing, caused by weakened tongue and pharyngeal muscles. Ineffective nursing and swallowing leads to weight loss and dehydration, along with severe electrolyte imbalances. Foals often have difficulty in breathing due to affected respiratory musculature. Urine is a dark yellow to brown color attributable to myoglobin released from the muscle cells during their degradation. Elevated levels of serum creatine kinase (CK) and aspartate aminotransferase (SAST) are fairly consistently found. These are two muscule enzymes typically released into the blood during muscle cell damage.

Early diagnosis is crucial if treatment is to be successful. Therapy consists of: (1) injectable vitamin E-selenium administered intramuscularly, (2) fluid therapy to correct electrolyte imbalances, (3) milk and oral supplements (given through nasogastric tube to dysphagic foals), and (4) antiinflammatory drugs used to lessen muscle pain and swelling. Foals must be kept in well-bedded stalls during recovery and physical exertion must be minimized to prevent further muscle trauma.

Prevention is, of course, the best medicine. To assess selenium status, forages, grains, and soil can be analyzed for selenium levels (deficiency is less than .1 ppm). Also, blood selenium or glutathione peroxidase activity can be measured in both mares and newborns. For those mares with low levels of blood selenium and glutathione peroxidase activity, feed can be supplemented with a commercial additive of 1-2 mg. selenium daily. Foals from these mares can be given an IM injection of a vitamin E-Se preparation at birth, usually repeated at 2 and 6 weeks of age. While forms of selenium toxicity do exist (mainly in the central-western United States), and care should be taken in selenium supplementation, White Muscle Disease can be prevented and treated with vitamin E-selenium preparations under the guidance of your veterinarian. As long as therapy is initiated early, the chances for recovery are relatively good.

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A Newsletter for Horsepeople

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Guest Editor: Herbert F. Schyver, DVM, Ph.D.
Student Editors : Matthew Belman '89
Corine Farewell '89
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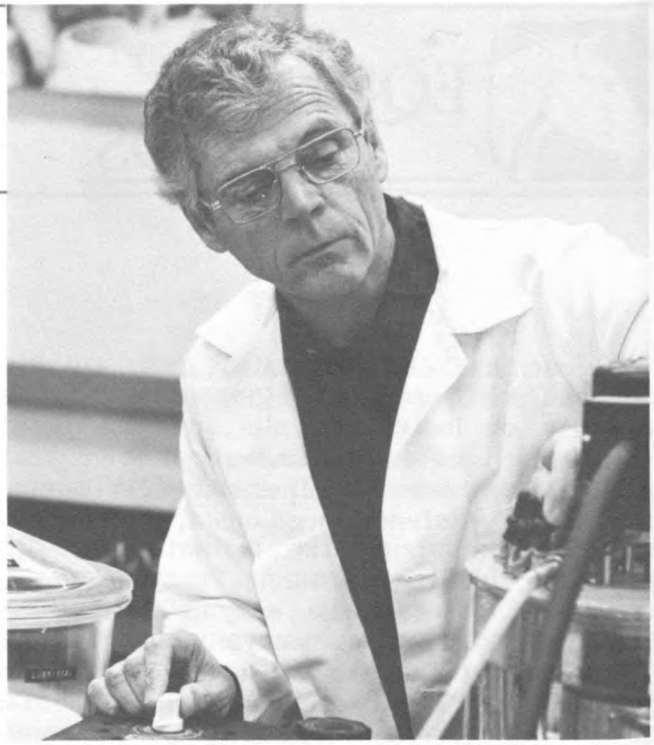

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IT'S FOAL SEASON AT THE EQUINE RESEARCH PARK. IF YOU'RE INTERESTED IN PURCHASING A STANDARDBRED OR THOROUGHBRED FOAL, CONTACT:
DR. TOM LITTLE, EQUINE RESEARCH PARK, BLUEGRASS LANE
CORNELL UNIVERSITY, ITHACA, NEW YORK 14850
OR CALL: 607/253-3081 or 607/255-7753

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