Not All Forage Plants Are Safe for Horses

INTRODUCTION

Pasturing horses is the most economical and easiest way to feed. The most difficult thing about pasturing horses is their grazing behavior. Horses have two grazing habits that can make pasture management difficult. They are highly selective grazers, choosing some grasses or areas to graze heavily while avoiding others. They are close grazers, leaving very little of the grass above the soil surface.

Most horses if given the opportunity, will spend 14-18 hours a day eating forages, much like free-running horses (Ralston 1986). There are health and metabolic problems such as colic, gastric ulcers and behavior concerns when horses are fed large amounts of nutrient dense feeds like grain only once or twice a day and have limited access to forages. So it is important to supply equine with a safe constant forage supply.

Because horses have evolved to eat plant material, we must keep in mind that not all plants are safe for them to eat. Poisonous plants and weeds can cause metabolic disturbances or death in horses. Horses differ from other livestock in that anything that impairs the horse's tolerance for exercise and performance diminishes the horse's value. This paper will cover some disorders created by certain pasture and forage plants.

Mycotoxins (In Forages)

Fescue toxicosis in the horse has been recognized for decades. However, the mechanism of action and successful management practices are only now being reported. Fescue toxicosis in the horse is due to several alkaloids in the endophytic fungus found in fescue. These alkaloids can cause problems to a pregnant mare and her fetus. Common signs of fescue endophyte toxicity include prolonged gestation, lack of udder development, absence of milk production, abortion, thickened placentas, retained placentas, stillbirths, and foal mortality.

One of the most common effects in pregnant mares is reduction of prolactin hormone, which triggers udder development in late pregnancy. Prolactin is released from the pituitary gland and is influenced by other hormones, including dopamine, which inhibits prolactin release. Dopamine is a chemical normally produced in the body, affecting function of glands, muscles, organs, and nerves, and it acts as a neurotransmitter in the central nervous system. It is essential to normal nerve activity in the brain and some peripheral tissues. Its cellular receptor activity is affected by the endophytic fungus toxins.

Fescue toxicosis may be controlled by management or treatment. A rotational grazing technique allows use of fescue for growing horses. Even though endophyte-infected tall fescue hay may be less digestible in the horse than uninfected hay (Redmond et al., 1991), young growing horses being exercised can efficiently use the endophyte-infected fescue on a short-term basis (Pendergraft et al., 1993). Similar techniques can be used in mares. A forage legume, such as birdsfoot trefoil or red clover, seeded with tall fescue will substantially reduce the adverse effects of this poison. The primary method of transmitting the fungus is through infected seed. Fungus-free and fungus-resistant seed is now available and should be used. However, pastures seeded with fungus-free seed may eventually become-infected.

If mares are removed from fescue in late gestation, most signs of toxicosis can be reduced or eliminated. Withdrawal from infected fescue before parturition results in a rise in serum prolactin levels, allowing milk production (Redmond et al., 1991). Mares moved to endophyte-free pasture at 305 to 310 days of gestation delivered live foals and lactated normally.
Fluphanazine has been considered, however Equidone, domperidone has been very promising. Daily oral doses of 1.1 mg/kg body weight of domperidone prevented symptoms of fescue toxicosis in late gestation mares on endophyte-infested fescue forage (Cross et al., 1999).

**Ryegrass**

Ryegrass staggers usually occur in grazing animals in late summer when predominantly ryegrass pastures are dry and growing slowly. The condition occurs most commonly in sheep with occasional cases in cattle and horses. Most affected animals show no signs unless disturbed or excited. Symptoms vary from mild tremors when animals are at rest, to severe incoordination and falling when they move or are excited. Death from this condition is rare. The danger of ryegrass staggers in pastures greatly diminished with rain and increased plant growth.

Ataxia, tremors, and paralysis were observed in a group of horses and then several weeks later in a second group ingesting the same hay. The horses were ingesting ryegrass hay containing 5 to 6 mg lolitrem B/kg. A stallion ingesting 1.5 and 2.5 mg lolitrem B/kg also experienced ryegrass staggers. Trembling, hyperexcitability, and abdominal muscular spasms developed suddenly in ponies fed exclusively ryegrass seed cleanings shown to contain 5.3 mg lolitrem B/kg (Munday et al., 1985).

Ergot The ergot mold primarily infects rye and some other small grains. Some forage grasses including promegrass, bluegrass and ryegrass are also occasionally affected by the same mold. Seeds form a distinct dark purple to blackish mass when infected with this mold.

Two separate syndromes have been identified. Gangrenous ergotism, the most common form, is a chronic condition affecting cattle and occasionally swine. It is characterized by lameness, swelling around the fetlocks and eventual loss of hooves due to a dry gangrene. If severe, ears and tails can also be lost to dry gangrene. The nervous form of ergotism, although relatively rare, usually begins with diarrhea followed by convulsions, aggressiveness and eventual paralysis of the respiratory center. There is no effective treatment except animal removal from the offending feed source. Prevention is best accomplished by clipping potential problem pastures to prevent development of seed heads.

The nervous form of ergotism, although relatively rare, usually begins with diarrhea followed by convulsions, aggressiveness and eventual paralysis of the respiratory system. There is no effective treatment except removal of the offending feed source. Prevention is best accomplished by clipping potential problem pastures to prevent development of seed heads. Symptoms of ergot toxicosis developed in several horses fed Bermuda grass hay (Hintz, 1988).

**Mycotoxins -Corn Stocks**

Fumonisins are produced by Fusarium moniliforme, which causes “stalk rot” in corn. Fumonisin toxicosis in horses has primarily been caused by corn screenings or corn-containing feed. Fumonisin causes equine leukoencephalomalacia (ELEM), liver necrosis, and occasionally death in horses. Feeding corn stocks a forage source especially in wet areas of the county may be an issue in feeding horses.

Moldy corn poisoning, also known as encephalomalacia or blind staggers, is associated with consumption of corn that has been contaminated with the fungus Fusarium moniliforme. This fungus thrives on corn plants that have been stressed by drought, disease, or insects prior to harvest. High humidity and moisture encourage proliferation of the mold. Exposure to high doses of this fungus over a short period of time results in liver toxicity, while low doses ingested over a longer time result in brain damage or moldy corn poisoning. Clinical signs include decreased appetite; behavioral changes such as depression, anxiety, or
hyper-excitability; and neurologic signs such as circling, blindness, difficulty chewing or swallowing, muscle tremors, ataxia, and eventually coma. Depending on the amount of toxin ingested, moldy corn poisoning can take up to 75 days before the horse demonstrates clinical signs; once signs are seen, death can occur within two to three days.

**Alsike clover**

Alsike clover (Trifolium hybridum) creates a photosensitivity response secondary to damage in the liver caused by alkaloids in the plants. Legume hays have also been implicated in setting up these conditions for skin problems. (Nation, 1989). Chronic or nervous clinical signs and liver disease, including biliary fibrosis and epithelial proliferation, may occur (Nation, 1991). Photosensitivity - Certain plants contain photo-reactive pigments that are absorbed into the blood when a horse eats the plant. In the presence of ultraviolet light from the sun, these then react in areas of non-pigmented skin, and the horse's skin sunburns. Generally, a horse has to consume these plants for a few months before the liver effects are severe enough to allow accumulation of a by-product of plant chlorophyll breakdown in the blood. This compound is called phylloerythrin, and its accumulation in areas of non-pigmented skin causes photosensitivity.

**Sweet clover**

Sweet clover poisoning occurs as a result of molds that grow in poorly managed sweet clover silage or hay that is put up too wet. These molds produce the chemical dicoumarol which blocks normal blood clotting in animals that consume large quantities. Dicoumarol is commonly used in several commercial rodent poisons. If formed, the poison persists in hay or moldy silage and is readily eaten by animals. The signs of sweet clover poisoning include those of abnormal bleeding. The first signs are spontaneous nosebleeds and black tar like manure. Swelling of joints, lameness and difficult breathing can occur if heavy doses are consumed. Treatment consists of removal of the feed source and administration of vitamin K to restore normal blood clotting. Prevention includes avoiding moldy sweet clover silages and moldy hay that consists primarily of sweet clover.

**Red clover**

Slobbers - excessive salivation were observed in horses eating red clover or other legumes infested with Rhizoctonia leguminicola that produces slaframine (Socket et al., 1982). It thrives best in cool conditions with high moisture. Slaframine may be visible on a plant as bronze-colored or black spots or rings, and there is a quantitative lab test for its presence. This toxin is usually stable, it can be found in baled hay. Slaframine can break down over time. In one report, after ten months of storage, the toxin dropped from 100 mg/kg to 7 mg/kg (Hagler and Behlow, 1981). Some of the signs of slaframine are excessive salivation, increased tear production, increased urination, bloating with associated colic, diarrhea, feed refusal, or abortion. Its effects go away almost immediately after taking away the contaminated feed.

Nitrate poisoning can occur in cattle and horses grazing pastures or eating hays that have accumulated high levels of nitrates during growth. Nitrates accumulate in plants when excessive rates of fertilizer have been applied or when plants have been drought stressed. Nitrate levels tend to be higher in the lower one-third of the plant or stalks and accumulate more at night and on cloudy days. Some species of plants that are known to accumulate nitrates include Johnsongrass, sorghum, sweetclover, bromegrass, orchardgrass, lambsquarter, oat hay, rape, barley, wheat and corn. Ensiling forages suspected of having high nitrate levels usually reduces the chances for problems. However, hay continues to be dangerous as the accumulated nitrates decrease slowly over time (Stanton, 1995).

Low levels of nitrates can cause abortion without any other symptoms. Severely affected animals develop muscle tremors, lose coordination and become weak. Moving these animals will initiate difficult breathing
commonly followed by collapse and death. Nitrate poisoning is often confused with prussic acid poisoning but is distinguished by a marked difference in blood color of affected animals. Animals poisoned by nitrates will have chocolate brown blood while those poisoned by prussic acid will have bright red blood. Treatment by a veterinarian can be effective if initiated early. Prevention includes mixing affected forages with normal forages to dilute the nitrate levels. Raising the cutter bar 10-12 inches to avoid cutting the lower one-third of the plant and not cutting drought-stressed forages for several days after a rain also helps reduce problems. If high nitrate levels are suspected, samples of the forage should be submitted to a competent laboratory for analysis before the forage is fed. Horses can tolerate a higher amount of nitrate levels in feed than cattle.

Prussic acid poisoning is caused by a poison called cyanide that can be produced in several types of plants under certain growing conditions. All species of farm animals may be affected with this acute poisoning. The plants most commonly involved in prussic acid poisoning are Johnsongrass, sundangrass, common sorghum, arrowgrass, black cherry, choke cherry, pin cherry and flax. Johnsongrass is the most toxic of the sorghums and commonly causes poisoning when subjected to frost or drought conditions. Very young, rapidly growing plants are more likely to produce the poison. Feeding or grazing of these forages should be delayed until they are more mature. Feeding forages following heavy nitrogen fertilization, plant injury by trampling or stunting of plant growth due to adverse weather should be avoided. If large amounts of forages containing prussic acid are eaten, death can occur within a few minutes. Excess salivation, difficult breathing, muscle tremors and rapid heart rate all signal the onset of prussic acid poisoning. Shortly after these symptoms are seen the animal may go down and death will likely occur due to respiratory paralysis. Animals that live one to two hours after the onset of these signs will usually recover.

Prussic acid is quite volatile and there is little danger from feeding well-cured hay. However, ensiling affected forages does not appear to make it safe to feed. The following prevention measures will best control the problem: Do not graze pastures that are less than 18-24 inches tall or green chop plants over 18 inches tall for three days after a killing frost. Do not green chop plants less than 18 inches tall for three weeks after a killing frost. Feed grain before allowing animals to graze fields that may be high in prussic acid.

**Colic** - Alfalfa pasture is commonly involved in causing cattle to bloat. Cattle in early stages of bloat will show signs of abdominal pain, which include restlessness and kicking at the belly. Legumes tend to produce gas in the Equine digestive system. Horse do not bloat but do colic just another name for a sever stomachache.

**Respiratory Problems** - Chronic obstructive pulmonary disease (COPD) can be caused by various allergens. A horse can be afflicted by respiratory allergies that are often attributable to mold and mold spores in hay. It is an emphysematous-like condition called COPD or “heaves”. Chronic obstructive pulmonary disease in horses has been associated with poorly ventilated stables, exposure to dust and mold and as a sequel to bacterial and viral respiratory tract infections. The incidence and severity of COPD is dependent upon the quantity of antigenic material inhaled and the sensitivity of the individual horse to specific antigens. Be aware and look for molds, do some testing if you suspect problems, and consider ration changes.

Botulism occurs in horses that have ingested feed contaminated with the bacterium Clostridium botulinum toxins. The toxin forms when a decomposing animal or bird is accidentally baled with the hay. Wet or rotten hay, especially legume hay, can also grow this bacteria. Botulinum toxin is extremely potent and lethal, with resulting paralysis preceding death.

**Selenium toxixosis or deficiency** - The NRC lists the required amount of Se at 0.1 mg/kg of the diet. Foals deficient in Se might exhibit stiff and painful muscle disease and cardiac problems, while older horses have recurrent episodes of tying-up. Selenium deficiency occurs in certain geographic areas such as the
Northwest and the northeastern United States. However, caution must be taken not to supplement with too much selenium as toxicity can occur. According to the NRC, the maximum tolerable level is 2 mg per kg. Supplementing selenium to toxic levels could trigger several adverse effects, including colic, diarrhea, hair loss, laminitis and separation of the hooves from the coronary band. Plants in the western part of the country can accumulate selenium and if consumed over time can cause Se toxicity to horses.

Blister Beetle toxicity is typically found in the western U.S. However, alfalfa hay is shipped all over the country. Every horse owners should be aware of the possibility of blister beetles in hay. Cantharidin is the chemical in blister beetles that causes toxicity when consumed by horses. The adult beetles prefer to feed on alfalfa blossoms but will also feed on the leaves. They also feed on goldenrod, pigweed, and many other plants. Some species such as the striped blister beetle are gregarious and large numbers will feed in a small area of a field. This characteristic allows large numbers of beetles to end up in a small number of hay bales or even in just a few flakes of hay within one bale. Horses consume dead beetles that were killed during the hay making process and remained in the flakes of hay. The most common sign of cantharidin toxicity in horses is colic. This is induced by the painful and irritating effects of cantharidin on mucosal surfaces such as the lining of the stomach and intestines. Sudden death is also possible with ingestion of large doses. Other less common signs of cantharidin toxicity are neurologic disease, difficult urination and/or blood in the urine since the toxin is excreted in the urine, heart problems, and oral lesions. The concentration of cantharidin in blister beetles varies between species, and between males and females. There is no antidote so symptomatic therapy is the only option. Contaminated hay should be destroyed since cantharidin is very stable even with long term storage and can also poison other species of livestock. Producers of alfalfa hay can do a number of things to try to prevent contamination of their hay. (Kenny, Pears and Swinker 2002)

Metabolic Disorders related to feeding forages: Because horses are living longer we are having reports of metabolic and hormonal dysfunction (Peripheral Cushing's disease) in horses. Clinical signs of metabolic syndrome include obesity-associated laminitis and abnormal fat deposition. In addition, insulin resistance appears to be one of the criteria defining metabolic syndrome. When a horse has a history of chronic laminitis, the first concern would be addressing the metabolic issues contributing to the problem, usually obesity and/or pituitary dysfunction, both of which are treatable.

Horses that are truly insulin resistant/glucose intolerant there is no one "type" of hay guaranteed not to trigger a bout of laminitis. It depends more on the harvest conditions, not the species of grass, whether a batch of hay contains sufficient non-structural carbohydrates (NSC: starches, water soluble sugars and fructans) to cause problems. Most horses tolerate more than 20 percent NSC without adverse effects, and most grass hays, especially those from the Eastern states, contain only 7-18 percent NSC, with an average of 12 percent. Even legume hays, on average, contain less than 15 percent NSC. Oat hay, on the other hand, averages 22 percent NSC. (Equi-Analytical Laboratories' web site.)

Grasses accumulate NSC throughout the day, with the highest concentrations achieved late in the day if the sun shines. If temperatures are above freezing and adequate water is present, NSC are converted to cellulose and other structural carbohydrates overnight, resulting in very low sugar concentrations by daybreak. If this process is disrupted by drought or freezing temperatures overnight, NSC concentrations can increase significantly.

The grasses continue to "respire" after cutting until the hay is baled and "cured." The longer the hay is dried in the field, the lower the NSC will be. Sugars and fructans are water soluble, so if the hay is rained on or soaked in water, the overall NSC will also be reduced. "Warm season" grasses, such as coastal Bermuda and crabgrass, tend to accumulate lesser amounts of sugars than the "cool season" grasses like fescue, orchard grass, and timothy under adverse conditions.
If a horse is sensitive to NSC content, the "safest" hays are coastal Bermuda or timothy cut early in the day, after a warm night and recent rainfall. Hay dried in the field for at least a day or two—even rained on a bit—is considered safer. Western hay producers tend to cut their hay later in the day to prevent excessive drying, and they bale more quickly than is possible in the humid East, all of which tends to preserve a higher NSC content.

*Laminitis* is inflammation within the sensitive laminae of the feet. It can occur for many reasons, but as a nutritional problem it is commonly linked to grain-rich diets, ingestion of too much rich pasture, and obesity. Grain overload or a diet rich in high-carbohydrate feed (grain or lush pasture) initiates a series of metabolic and endocrine (hormone) disturbances in the body. A diet abundant in carbohydrates upsets normal intestinal bacteria, allowing more endotoxins from harmful bacteria to be absorbed into the bloodstream than can be neutralized by the liver.

**Mare Reproductive Loss Syndrome (MRLS)** At the end of April 2001, veterinarians reported a disease outbreak referred to as Mare Reproductive Loss Syndrome (MRLS), occurring among pregnant mares of all breeds in central Kentucky. Clinical signs of the disease both early and late fetal losses, the birth of weak foals, and a very small number of horses of different sexes and ages that exhibited pericarditis and unilateral uveitis. Infectious or contagious disease agents are not considered a primary cause, but environmental toxic agents remain the primary suspect. The University of Kentucky, College of Agriculture recommended that farms consider many contingency measures to reduce the risk of MRLS.

“Breeders can prevent mare reproductive loss syndrome (MRLS) by keeping horses away from caterpillars”, said Bruce Webb, PhD, a University of Kentucky researcher who has been studying the condition. Five years later, scientists suggest future studies are needed in the following topics:

Eastern Tent Caterpillars (ETC)--Researchers suspect that small lesions created in the mare's gastrointestinal (GI) tract by the caterpillars' setae (hairs) might allow bacteria to enter and circulate in the body to somehow reach the fetus and ultimately cause abortion. Webb said to keep pregnant mares off pastures with caterpillars for eight weeks after trees lining the pastures are sprayed with a caterpillar insecticide. Also, introducing a caterpillar virus early in the ETC season could dramatically decrease ETC numbers. Studying the Fetus In Utero--Cornell University's Don Schlafer. Schlafer is seeking funding for the research, which could be used to investigate fetal responses to viruses, bacteria, and other microbes that find their way to the pregnant uterus, as in MRLS.

**IMPLICATIONS**

When feeding horse forages there are a few things to keep in mind. There are molds and fungi that can produce toxins in forages that can make your horse sick or cause death. Learn to recognize clinical signs, and understand climatic conditions that may cause plants to be affected. Buy feeds and hays from reputable dealers and lab test suspicious feed.

**REFERENCES:**


Station, T., Colorado State University, College of Agriculture, Fact Sheet No. 1.610, Nitrate Poisoning, Fort Collins, Co. 1995.
