

ANTI-NUTRITIONAL FACTORS IN ALFALFA HAY

Birgit Puschner¹

ABSTRACT

It is not surprising when disease outbreaks are traced back to a plant in such general use for forage and hay as alfalfa. Undesirable effects may result from ingestion of large amounts of alfalfa by livestock. A variety of anti-nutritional factors in alfalfa have been identified. In many instances, alfalfa is contaminated with poisonous weeds. One of the most common weeds found in alfalfa hay is *Amaranthus retroflexus*, or redroot pigweed, a potential nitrate accumulator. In addition, *Senecio vulgaris*, or common groundsel has been found in alfalfa hayfields. *Senecio vulgaris* can contain toxic pyrrolizidine alkaloids. Alfalfa hay may contain blister beetles (*Epicauta* spp.). Blister beetle poisoning is a significant problem in the US. Other anti-nutritional factors in alfalfa include bloat-producing saponins, photodynamic agents leading to photosensitization, coumestans inducing phytoestrogenism, and selenium accumulation resulting in chronic selenosis. Exposure to botulism toxin may occur if alfalfa hay is contaminated with decayed organic matter, or carcasses.

Key Words: alfalfa, nitrate, phytoestrogens, blister beetle, selenium, botulism, toxicosis

INTRODUCTION

Alfalfa hay is used primarily as animal feed for dairy cows, but also for beef cattle, sheep, goats, chickens, turkeys, horses, and other farm animals. Alfalfa is one of the most widely grown crops in the United States and produces forage higher in protein and minerals, but lower in fiber and, therefore, higher in net energy than many other forages. While alfalfa is of high nutritional quality as animal feed, it may contain compounds that may inadvertently affect animals.

Common crop and pasture plants and numerous weeds may accumulate toxic levels of nitrate. Among weeds, pigweed (*Amaranthus* spp.), nightshades (*Solanum* spp.) and goosefoot (*Chenopodium* spp.) have been found to contain nitrate at a potentially toxic concentration. Among crop plants, especially oat hay, and sorghum have been incriminated with nitrate toxicosis, but alfalfa itself may contain potentially toxic nitrate concentrations. Alfalfa hay may be contaminated with pigweed or goosefoot, thus, presenting a potential source for nitrate poisoning (Bedwell et al., 1995). Nitrate accumulates in the vegetative tissue, particularly in stalks with less in the leaves. Seeds do not generally contain toxic nitrate levels. Heavy fertilization of pastures, herbicide treatment, drought, cloudy weather, and decreased temperature may increase the nitrate concentrations in plants. Nitrate poisoning is primarily a problem in ruminants because of the reduction of nitrate to nitrite by ruminal microorganisms. The nitrite ion produces methemoglobin, which cannot react with oxygen, so anoxia occurs (Oswailer et al., 1985). Methemoglobin leads to dark brown or chocolate-colored blood, a common feature of nitrate/nitrite poisoning. Clinical signs of acute nitrate poisoning include diarrhea, salivation,

¹ B. Puschner, DVM, Ph.D., Diplomate of the American Board of Veterinary Toxicology, California Animal Health and Food Safety Laboratory System, University of California, West Health Sciences Dr., Davis, CA 95616. Published In: Proceedings, 2000 National Alfalfa Symposium, 10-12 December, 2000, Las Vegas, NV.

dyspnea, tremors, ataxia, rapid heartbeat, and terminal convulsions. Death may occur within 6-24 hours. Diagnosis is based on appropriate clinical signs and laboratory analysis of nitrate and nitrite in serum, blood, aqueous humor, rumen contents, and forage. Forage nitrate levels of 0.5% and above are potentially dangerous, with acute poisoning likely to occur if the nitrate level exceeds 1%. Forage management techniques can affect the concentration of nitrate.

Senecio vulgaris (common groundsel) is a common weed in hayfields in California and possibly other states. New plantings of alfalfa and alfalfa weakened by heavy weevil infestations are more susceptible to competition by common groundsel. *Senecio* spp. can contain pyrrolizidine alkaloids (PA). PAs are hepatotoxins, causing irreversible liver damage (Cheeke 1998). Horses and cattle are the major livestock species poisoned by PAs. Sheep, goats and small herbivores (e.g. rabbits, guinea pigs, hamsters) are resistant to PA toxicity due to detoxification processes in the liver (Cheeke, 1994). Clinical signs of chronic PA poisoning may often not appear for 2-8 months after the first ingestion of PA containing plants. Affected animals lose condition, and develop icterus. Cattle may also develop photosensitization. Neurological signs are commonly seen in horses, and the condition is called "walking disease". The presumptive diagnosis is based on clinical signs, and gross as well as histologic lesions (Hopper 1978). Identification of PA-containing weeds in alfalfa and detection of PAs in forage are important to establish an accurate diagnosis. However, due to the prolonged delay in onset of clinical signs the affected hay may no longer be available for testing. While there is some degradation of PAs in silage, the PA content of hay remains constant over many months. Silage contaminated with more than 5% *Senecio* spp. is considered unsafe for cattle or horse feeding (Candrian et al., 1984).

Consumption of alfalfa hay containing blister beetles (*Epicauta* spp.) can be fatal to horses and other livestock animals (Beasley et al., 1983). Blister beetles contain a natural toxin, called cantharidin. Although horses are considered most susceptible to cantharidin, similar levels of toxicity have also poisoned sheep, goats, cattle, and chickens. Clinical signs of cantharidin poisoning include severe irritation and inflammation of digestive and urinary tracts, increased temperature, diarrhea, increased heart rate, and shock. Death may occur within 24 hours. Adult blister beetles concentrate in alfalfa fields during blooms to feed on pollen and plant nectar, and are often harvested with the alfalfa. Modern agricultural practices, like crimping of hay can create a greater risk for intoxication preventing escape of beetles. Cantharidin can continue to be toxic in bales after beetles have died. Larvae develop in the soil and feed on grasshopper eggs. Thus, blister beetle populations may be increased in years following a high grasshopper population. Several management options exist to help reduce the risk of blister beetle contamination in alfalfa hay (Hutchison et al., 1990).

Bloat primarily occurs on legume pastures. Alfalfa is one of the most important bloating species in temperate regions (Hall et al., 1994). Bloat is a distension of the rumen as a result of gases trapped in the form of a stable foam. Lush legume pastures, especially immature foliage can contain high concentrations of soluble protein, which produce a stable rumen foam. Legume hay rarely causes bloat, since drying results in protein denaturation. In recent years, bloat-safe alfalfa has been developed, based on the observation that the major foaming agents involved are proteins (Goplen et al., 1993). In addition, supplementation of cattle with monensin reduces bloat incidence, due to decreased gas production and reduced foam stability (Tanner et al., 1995).

Alfalfa hay and silage have induced photosensitization in livestock (Casteel et al., 1995; House et al., 1996). Causative agents in alfalfa hay have not been determined. Consumption of alfalfa silage has not resulted in liver disease. Thus, alfalfa silage may contain primary photosensitizing agents. Clinical signs of photosensitization include inflammation and ulcerated lesions of non-pigmented skin, and photophobia.

Plant estrogens are referred to as phytoestrogens. The phytoestrogens in alfalfa are coumestans, and are synthesized by plants from cinnamic acid (Adams, 1989). Coumestan-induced infertility is generally considered as temporary. However, problems in breeding have occurred with cattle and sheep. Coumestans suppress estrus and inhibit ovulation, and can lead to precocious mammary and genital development in heifers (Adams, 1995). However, the infertility does not persist if the animals are subsequently maintained on nonestrogenic pastures.

Alfalfa is a passive selenium accumulator (Rosenfeld et al., 1964). Thus, alfalfa may accumulate potentially toxic selenium concentrations if grown on soils that contain bioavailable selenium. Selenium poisoning in livestock occurs in acute and chronic forms. Alfalfa hay that contains more than 5 ppm Se may cause chronic selenosis (National Research Council, 1983). Alkali disease, the classical syndrome associated with chronic selenosis is characterized by weight loss, hair loss, and hoof deformities. In addition, excessive dietary selenium has been associated with abortion and teratogenesis (Raisbeck et al., 1993). In high-selenium regions, evaluation of selenium concentrations in locally grown alfalfa may need to be incorporated into an overall workup.

Botulism can cause deaths in a large number of animals. The disease is characterized by progressive, flaccid paralysis, and is observed in all species of animals (Rocke, 1993). The most common form of botulism is the ingestion of preformed toxin associated with carcasses or decayed organic matter. Thus, exposure to poorly ensiled small grain haylages, carcasses, poultry litter, or bones from dead animals may result in exogenous botulism poisoning (Rings, 1987). If alfalfa is contaminated with a carcass, botulism toxin may be present in the feed. Diagnosis of botulism is still difficult and usually requires a thorough investigation to rule out other causes of weakness and death in livestock.

REFERENCES

Adams NR: 1989, Phytoestrogens. **In:** Cheeke PR (ed.). Toxicants of plant origin, Vol IV. CRC Press, Boca Raton, FL.

Adams NR: 1995, Detection of the effects of phytoestrogens on sheep and cattle. *J Anim Sci* 73:1509-1515.

Beasley VR, Wolf GA, Fischer DC et al.: 1983, Cantharidin toxicosis in horses. *JAVMA* 182:283-284.

Bedwell CL, Hamar DW, Hoesterey ML et al.: 1995, Comparison of four methods for forage nitrate analysis. *J Vet Diagn Invest* 7:527-530.

Candrian U, Luthy J, Schmid P, et al.: 1984, Stability of pyrrolizidine alkaloids in hay and silage. *J Agric Food Chem* 39:930-933.

Casteel SW, Rottinghaus GE, Johnson GC et al.: 1995, Liver disease in cattle induced by consumption of moldy hay. *Vet Hum Toxicol* 37:248-251.

Cheeke PR: 1998, Natural toxicants in feeds, forages, and poisonous plants. 2nd ed., pp.338-352. Interstate Publishers, Inc., Danville, IL.

Cheeke PR: 1994, A review of the functional and evolutionary roles of the liver in the detoxification of poisonous plants, with special reference to pyrrolizidine alkaloids. *Vet Hum Toxicol* 36:240-247.

Goplen BP, Howarth RE, Lees GL: 1993, Selection of alfalfa for a lower initial rate of digestion and corresponding changes in epidermal and mesophyll cell wall thickness. *Can J Plant Sci* 73:111-122.

Hall JW, Majak W, Stout DG et al: 1994, Bloat in cattle fed alfalfa selected for a low initial rate of digestion. *Can J Anim Sci* 74:451-456.

Hooper PT: 1978, Pyrrolizidine alkaloid poisoning – pathology with particular reference to differences in animal and plant species. **In:** RF Keeler, KR Van Kampen, and LF James (eds). *Effects of poisonous plants on livestock*. Academic Press, NY.

House JK, George LW, Oslund KL et al.: 1996, Primary photosensitization related to ingestion of alfalfa silage by cattle. *JAVMA* 209:1604-1607.

Hutchison WD, Murphy MJ, Tuft GN: 1990, Blister beetles in alfalfa. Management options to minimize poisoning in horses. Publication of the Minnesota Extension Service, University of Minnesota.

National Research Council: 1983, Selenium in nutrition. National Academy Press, Washington, DC.

Osweiler GD, Carson TL, Buck WB et al.: 1985, Clinical and diagnostic veterinary toxicology, 3rd ed., pp. 460-467. Kendall/Hunt Publishing Co., Dubuque, IA.

Raisbeck MF, Dahl ER, Sanchez DA et al.: 1993, Naturally, occurring selenium in Wyoming. *J Vet Diagn Invest* 5:84-87.

Rings DM: 1987, Bacterial meningitis and diseases caused by bacterial toxins. *Vet Clin North Am* 3:85-97.

Rocke TE: 1993, *Clostridium botulinum*. **In:** Gyles CL, Thoen CO (eds). *Pathogenesis of bacterial infestions*. 2nd ed., pp. 86-96. Iowa State University Press, Ames, IA.

Rosenfeld I, Beath OA: 1964, Selenium: geobotany, biochemistry, toxicity, and nutrition. Academic Press, NY.

Tanner GJ, Moate PJ, Davis RH et al.: 1995, Proanthocyanidins (condensed tannins) destabilize plant protein foams in a dose dependent manner. Aust J Agric Res 46:1101-1109.