

POISONOUS PLANTS AND EFFECTS ON ANIMALS

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ABSTRACT

Livestock animals are at the mercy of toxic plants when these plants are present in feeds or hay. Alfalfa hay or forage, which is commonly offered to livestock animals, may pose a risk if contaminated with poisonous plants. Undesirable effects may result from ingestion of large amounts of alfalfa by livestock but may also be associated with the ingestion of small amounts of highly toxic plants present in alfalfa. In many cases, livestock animals, especially horses are selective about what they eat, but instances such as herbicide applications may change palatability or increase toxicity in some plants. Most plant poisonings have no specific treatments and thus, are best prevented by avoiding exposure to toxic plants in the first place. Hay and feed should be carefully inspected for contamination with potentially toxic weeds before the first feeding and owners should establish excellent working relationships with suppliers and growers. Producers and farm managers, along with veterinarians and diagnosticians play important roles, and all contribute information that may be important to a poisoning case. Once all the information is available, all evidence is collected, and proper sampling of specimens has occurred, a summary of findings can be provided and will be instrumental in preventing reoccurrences.

Livestock poisoning associated with the ingestion of hay contaminated with nitrate accumulators, pyrrolizidine alkaloids, summer pheasant's eye, foxtail, and oleander are discussed. Awareness of poisonous plants growing in a certain geographical region and their associated clinical signs are instrumental in making a diagnosis, and initiate treatment. More importantly, the recognition of poisonous plants in hay or forage may help prevent plant poisonings in animals. In addition, botulism is briefly discussed.

Key Words: alfalfa, nitrate, pyrrolizidine alkaloids, summer pheasant's eye, bristlegrass, oleander, botulism, toxicosis

INTRODUCTION

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. While alfalfa is of high nutritional quality as animal feed, it may contain compounds that may inadvertently affect animals. Even though alfalfa hay is primarily used as animal feed for dairy cows, it is also included in rations for beef cattle, sheep, goats, chickens, turkeys, horses, and other farm animals.

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Nitrate accumulators: There are a number of common crop and pasture plants, and weeds that can accumulate toxic nitrate concentrations. Among weeds, pigweed (*Amaranthus* spp.), nightshades (*Solanum* spp.) and lamb's quarters (*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 lamb's quarters, thus, presenting a potential source for nitrate poisoning (Bedwell et al., 1995). Nitrate accumulates in the vegetative tissue, particularly in stems with less in the leaves. Seeds generally do not 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. Cattle are especially susceptible to nitrate toxicosis. The nitrite ion produces methemoglobin, which cannot react with oxygen, so anoxia occurs (Osweiler 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 depression, dyspnea, tremors, ataxia, rapid heartbeat, and terminal convulsions. Death may occur within 6-24 hours of ingestion. Diagnosis is based on appropriate clinical signs and laboratory analysis of nitrate and nitrite in serum, blood, ocular fluid, rumen contents, and forage. Forage nitrate levels of 0.3% 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 and can reduce the risk. Careful use of nitrogen fertilizers, harvest under appropriate conditions, supplementation of ration with corn, ensiling, and testing hay and forage for nitrate content are approaches to minimize the risk of nitrate poisoning in animals (Geurink et al., 1982).

Pyrrolizidine alkaloids: Pyrrolizidine alkaloid (PA) poisoning is of great economic importance as a cause of progressive liver disease in livestock animals. The disease has been reported from most areas of North America and is mostly caused by plants from the genus *Senecio*, but other plant genera such as *Amsinckia* and *Cynoglossum* spp. also contain the toxic alkaloids. Many of the groundsels and ragworts (*Senecio* spp.) contain PAs. *Senecio vulgaris* (common groundsel) is a common weed in hayfields in California and is also widely distributed along the East Coast and Canada. New plantings of alfalfa and alfalfa weakened by heavy weevil infestations are more susceptible to competition by common groundsel. PAs are hepatotoxic, 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". After the onset of clinical signs, the prognosis is poor (Schmitz, 1998). 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 that was eaten weeks or months before may no longer be available for evaluation. 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).

Summer Pheasant's eye: *Adonis* spp., including *Adonis aestivalis* (summer pheasant's eye, summer adonis) have long been utilized in European folk medicine for their cardiac-enhancing effects. Summer pheasant's eye was introduced into North America as a horticultural plant, escaped cultivation and is now naturalized in the western United States (Burrows GE et al., 2001). *Adonis aestivalis* has erect stems with terminal solitary flowers. The flowers have orange petals, and the leaves are simple alternate with blades two or three times pinnately dissected into linear segments (Fig 1).



Figure 1: Stand of *Adonis aestivalis* (summer Adonis, summer pheasant's eye) in Northern California. Associated grass hay resulted in fatal disease in horses.
Inset: flower with waxy, orange petals.

Adonis spp. are members of the Ranunculaceae family, and are considered unpalatable which is most likely the reason why reports of poisoning are rare. However, adonis poisoning has been reported in horses (Woods et al., in press), pigs (Davies et al., 1989) and sheep (Hurst, 1942). Plants of the *Adonis* genus contain a series of cardenolides similar to oleander and foxglove. The toxic cardenolides are highest in the leaves and flowers. Clinical signs of poisoning include gastrointestinal disturbances, such as colic, hemorrhagic enteritis, diarrhea or decreased gut motility. In addition, the cardiotoxins present in *Adonis* lead to a variety of tachy- and

bradyarrhythmias and at toxic doses death can occur. In the recent past, several horses died in California as a result of eating grass hay containing summer adonis. Poisoning of livestock with summer Adonis is becoming more feasible through contamination of fields harvested for animals feed. The plant may become better established in the western region of North America and may be more commonly found as a poisonous weed in hay. Recognition of summer adonis in hay can be instrumental in preventing poisoning. In addition, diagnostic procedures of a suspect adonis poisoning case in livestock include pathological and toxicological examination. Recently, a method was developed to identify the toxin of adonis in specimens from affected animals and suspect hay or forage to help establish a diagnosis and prevent further exposure (Filigenzi et al., 2003).

Bristlegrass: In the past years, the Toxicology laboratory of CAHFS diagnosed several cases of disease related to the exposure of horses to *Setaria* spp., also known as bristlegrass or bristly foxtail. The affected horses had a history of blisters and lesions in the mouth and ulcerations on the tongue and lips. Alfalfa hay fed to these horses contained large amounts of two different species of bristlegrass: *Setaria viridis* (green foxtail) and *Setaria glauca* (yellow foxtail, yellow bristlegrass, bristly foxtail). Both species have sharp and barbed bristles, particularly *S. glauca*. The bristles are capable of penetrating the mucous membranes and causing serious erosions of the mouth. Problems usually occur when the grass is cut late and a substantial number of panicles are present. The mechanical injury is a particularly serious problem in horses, but has also been reported in cattle (Fava et al., 2000). The lesions in the mouth will cause pain, resulting in a reluctance to eat and a loss of weight. The bristles can cause deep ulcerations of the tongue, gums and cheeks, which may result in abscesses. The bristles may be present in the wounds and need to be removed, if possible. If alfalfa hay is contaminated with bristlegrass, it should not be fed to livestock animals.

Oleander: The potential for exposure of animals to oleander is high. Oleander (*Nerium oleander*) is an ornamental, evergreen shrub that is very drought tolerant and often able to survive unattended in pasture lands where livestock graze. However, ingestion from clippings or dried leaves is the most common cause of oleander poisoning in animals (Galey et al., 1996). Modern agricultural practices, like crimping of hay can create a greater risk for oleander contamination from leaves blowing into the fields before harvest. Oleander is commonly found in the southern United States and most of California. The toxicity of oleander results from cardiac glycosides, with oleandrin being the most prominent one. Cardiac glycosides inhibit the Na^+/K^+ ATPase, ultimately resulting in cardiac arrhythmias. Death is typically due to ventricular fibrillation. The toxic cardenolides are present in all parts of oleander, and toxicity is retained with drying. The leaves and flowers have the highest concentrations of toxic cardenolides. 10–20 medium-sized leaves can be lethal to a horse, while as few as 8 leaves can cause death in a cow (Galey et al., 1998). Clinical signs of oleander poisoning include diarrhea, excess salivation, depression, and anorexia. As the disease progresses, the animals develop a variety of cardiac signs, including bradycardia and arrhythmias. At this stage of the disease, the animals may also show tremors and difficulty breathing. However, often the disease progresses so rapidly that the animal is found dead and clinical signs are not observed. Oleander leaves may be present in the gastrointestinal contents, but identification may not always be possible. Every effort should be made in a suspect oleander poisoning case to reach an accurate diagnosis so that adequate therapeutic measures can be initiated. In addition, the source of oleander has been identified and removed from animals.

Diagnosis of oleander poisoning has improved significantly since the development of several specific analytical methods (Tor et al., 1996; Puschner et al., 2001). Confirmation of the toxic glycoside oleandrin in biological samples, such as serum, urine and gastrointestinal contents provides invaluable information to the clinician in directing the clinical course and initiating adequate treatment in confirmed, non-fatal poisoning cases. Suspect contaminated plant material can also be analyzed for oleandrin to prevent fatal oleander poisoning in livestock.

Botulism: 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; Galey, 2001). 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. Botulism is usually a clinical diagnosis and supported by a thorough investigation to rule out the many other causes that result in clinical signs similar to botulism. It is very difficult to establish a definite diagnosis of botulism as laboratory tests are often not sensitive enough. As with many other toxicoses, prevention is the best way to deal with botulism. Forage should be evaluated with respect to carrion or other foreign matter.

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