

POISONOUS WEEDS AND TOXIC FACTORS IN HAY CROPS: WHY YOU SHOULD WORRY

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ABSTRACT

Noxious weeds can result in clinical signs of poisoning when consumed by livestock in pasture or hay. It is important to establish an accurate diagnosis of plant poisoning in order to provide adequate treatment to affected animals, carefully and accurately assess the potential for the transfer of toxins into edible products and prevent further exposures. Important steps in prevention are recognition and knowledge of which poisonous plants occur on range or pasture and which animals are most susceptible to the toxic effects. In addition, producers and veterinarians must work together effectively to handle possible publicity and medico-legal issues that result from exposure to toxic plants. In California, the most common weeds with serious concerns to livestock are nitrate accumulators, common groundsel, oleander, and bristlegrass. The severity of plant poisoning is illustrated by specific cases.

Key Words: nitrate, common groundsel, oleander, bristlegrass, poisonings, toxic plants

INTRODUCTION

Exposure to toxic plants can result in significant losses of livestock. Adverse effects may result from a single ingestion of a large amount of, or a highly, toxic plant, or from exposure to small amounts over weeks to months leading to chronic illness and often death. The latter situation is very difficult to manage and diagnose as exposure to toxic plant material often ended prior to the onset of clinical signs of disease; in addition, at that time, treatment options are often limited or non-existent. Plant poisonings in livestock may be underreported as diagnostic work-up is often only done when the economic loss is sufficient to justify a thorough investigation. There are several factors that must be met to result in animal poisoning. Foremost, there is the requirement that a sensitive species of animal ingest or otherwise be exposed to a toxic plant; for example, exposure to plants with high nitrate concentrations is only of concern in ruminants but would not be a risk to horses. Furthermore, growing conditions as well as maturity stages of plants influence toxin concentrations and thus risk for poisoning. With few exceptions, livestock will not eat poisonous plants unless forced to by hunger or if incorporated into rations. Thus, the single most important way to avoid poisonings is exposure prevention by proper range and pasture management practices and by providing ample forage and favor encourage consumption of non-toxic plants. Areas infested with poisonous plants should be avoided when trailing, holding, or unloading animals. Supplemental feed may protect stock if these conditions cannot be avoided, but there are instances (for example, herbicide applications) that can change palatability or increase toxicity of some plants. Additionally, when toxic weeds are embedded into cubes or pellets, animals cannot avoid exposure.

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While there are many indications for plant poisonings, the most obvious cases involve sudden onset of disease in a group of animals ingesting the same feed. A diagnosis of poisoning is often supported when common feed or environmental conditions are found. A toxicosis is also suggested when the animal dies suddenly, because many plant toxins result in acute poisoning. Overall, the success in correctly and rapidly diagnosing a plant poisoning depends on available specimens, time since exposure, and the extent of diagnostic work-up. In many cases, initial clinical signs are non-specific (such as diarrhea) and post-mortem lesions may be absent leading the diagnostician to no additional toxicology testing. However, veterinary toxicology laboratories can provide testing for many of the most commonly diagnosed plant toxins and should be consulted. The best way to support a diagnosis of a plant poisoning is to confirm the presence of a toxic plant in the animal's environment, to confirm that the plant has been ingested (by noting that the candidate plants have been chewed and/or finding plant fragments in gastrointestinal tract samples) and to correlate clinical findings, when possible, with those known to be associated with the suspect plant. Confirmation of plant poisoning is especially important in insurance or legal investigations.

Poisonous weeds that have been found in alfalfa or other hay include nitrate-accumulator plants, pyrrolizidine alkaloid containing plants, oleander, and grasses that can cause mechanical irritation. Awareness of poisonous plants growing in a certain geographical region and their associated clinical signs are instrumental in making a diagnosis and initiate treatment. Most importantly, recognition of poisonous plants in hay or forage may help prevent plant poisonings in animals.

NITRATE-ACCUMULATING PLANTS

Nitrate-nitrite poisoning can result in high mortalities in cattle and other ruminants (Costagliola et al. 2014). The most common nitrate accumulating weeds identified in alfalfa hay are pigweed (*Amaranthus retroflexus*) and lamb's quarters (*Chenopodium spp.*). Among crop plants, especially oat hay and sorghum have been incriminated with nitrate toxicosis, but alfalfa itself may contain potentially toxic nitrate concentrations. Nitrate accumulates in the vegetative tissue, particularly in stems with less in the leaves (Bedwell et al. 1995). Thus, grass species have a greater tendency to accumulate nitrate compared to broadleaf plants. Several other factors may promote nitrate accumulation in forages including fertilizer rate, stage of maturity, amount of sunlight, herbicide treatment, and drought. In general, any stress condition can increase nitrate levels. Thus, testing for nitrate is especially recommended before feeding stressed forages. In addition, it is important to assess nitrate concentrations in water as the toxic effects of nitrate are additive taking into account all sources of nitrate (total from feed and water). During normal digestion, ruminal microbes convert nitrate (NO_3) to nitrite (NO_2), and further to ammonia,



Figure 1. *Amaranthus retroflexus*, redroot pigweed. Courtesy of Dr. Joe DiTomaso.

which is incorporated into protein. Rapid ingestion of large amounts of nitrate allows the converted nitrite to be rapidly absorbed into the bloodstream before it can be converted to ammonia. Nitrite then oxidizes hemoglobin to methemoglobin which causes the clinical manifestation of intoxication due to its inability to carry oxygen. Nitrate poisoning is an acute disease in ruminants, especially cattle. Clinical signs of acute nitrate poisoning include depression, respiratory distress, 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, ocular fluid, rumen contents, and forage. Lab testing can help decision making on whether forages are safe to feed. Forage nitrate levels of 0.3% and above (dry matter basis) are potentially dangerous, with acute poisoning likely to occur if the nitrate level exceeds 1%.

Table 1. Interpreting nitrate (differing reporting units) forage tests

<i>NO3 (dry matter)</i>	<i>NO3-N (dry matter)</i>	<i>KNO3 (dry matter)</i>	<i>Feeding Recommendations</i>
< 5,000 ppm (0.5%)	< 1,200 ppm (0.12%)	< 8,100 ppm (0.81%)	Generally Considered Safe for Livestock
> 5,000 ppm (0.5%) but < 10,000 ppm (1%) ppm	> 1,200 ppm (0.12 %) but < 2,300 ppm (0.23%)	> 8,100 ppm (0.81%) but < 16,000 ppm (1.62%)	Caution: Problems can occur at this level
>10,000ppm (1%)	> 2,300 ppm	>16,200ppm (1.62%)	Do not feed

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.

PYRROLIZIDINE ALKALOID CONTAINING PLANTS

Pyrrrolizidine alkaloids (PAs) are highly toxic chemicals present in many plants belonging to the families of Asteraceae, Boraginaceae and Fabaceae (Wiedenfeld 2011). In our region, the most common PA containing plants are common groundsel (*Senecio vulgaris*), fiddleneck (*Amsinckia intermedia*), and tansy ragwort (*Senecio jacobaea*). These weeds are most commonly found in spring cut alfalfa hay. Plants containing PAs are normally avoided due to unpalatability, except under drought conditions when little other forage is available or when incorporated into hay cubes or pellets. Most reported poisonings in animals result from ingestion of hay and other feed ingredients contaminated with PA-containing weeds in forms that prevent animals from exercising discrimination (Molyneux et al. 1991; R. Gardner et al. 2006). Cattle and horses are most susceptible to the toxic effects while sheep, goats and small herbivores (e.g. rabbits, guinea pigs, hamsters) are resistant (Cheeke 1994).

PAs are metabolized in the liver of animals to highly reactive compounds that lead to progressive liver disease after several months of PA exposure. Thus, PA poisonings are usually chronic in nature, but onset of clinical signs may be sudden. Clinical signs include loss of condition, anorexia, dullness, constipation or diarrhea. Tenesmus and passing of blood stain feces may occur and is often followed by rectal prolapse especially in cattle. Ascites, icterus, and intermittent photosensitization may be seen. Animals may become progressively weaker and reluctant to move or exhibit signs of hepatic encephalopathy (especially horses) including head-pressing, aimless wandering, ataxia, or even frenzied and aggressive behavior. Pica may be noted and death seen suddenly or after prolonged recumbency with coma subsequent to high levels of ammonia. Photosensitization secondary to liver failure may also be observed in horses and cattle. Identification of PA-containing weeds in alfalfa and detection of PAs in forage are important measures to prevent poisonings. 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).



Figure 2. *Senecio vulgaris*, common groundsel.
Courtesy of Dr. Joe DiTomaso.

OLEANDER

Oleander (*Nerium oleander*) poisoning is a common poisonous plant problem encountered in California. Oleander is an ornamental, evergreen shrub. Oleander leaves are leathery and dark gray-green and have a prominent midrib with secondary veins that are parallel with each other. In livestock, oleander poisoning often results from accidental ingestion of plant clippings, plant material baled with hay, or when chopped into silage. Modern agricultural practices, like crimping of hay can create a greater risk for oleander contamination from leaves blowing into the fields before harvest. Most commonly, horses, cattle, goats, llamas, and alpacas are affected, but oleander poisoning has also been reported in sheep, cats, dogs, birds and humans. The toxicity of oleander results from several cardiac glycosides that are present in all parts of the plant, but in highest concentrations in leaves and flowers. The most prominent of those glycosides is oleandrin. Cardiac glycosides cause poisoning by inhibiting Na^+/K^+ ATPase resulting in electrolyte disturbances that alter the electrical conductivity of the heart. Oleander is an extremely toxic plant; as little as 0.005% of an animal's body weight in dry oleander leaves may be lethal, which is estimated to be 5-10 leaves for an adult horse or cow (Galey et al. 1996). The plant remains toxic when dried.

Animals exposed to oleander are often found suddenly dead or present with rapidly developing nonspecific signs that may resemble colic. When clinical signs are observed, they include diarrhea, excess salivation, depression, staggering and anorexia (Galey et al. 1996; Soto-Blanco et al. 2006). As the disease progresses, the animals develop a variety of cardiac signs, including bradycardia, tachycardia and arrhythmias. At this stage of the disease, the animals may also show tremors and difficulty breathing. In camelids (alpacas, llamas), renal failure is often seen several days after exposure to oleander. However, in many cases the disease progresses so rapidly that the



Figure 3. Oleander (*Nerium oleander*) contamination of hay.
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animal is found dead and clinical signs are not observed. Oleander poisoning typically occurs as a herd problem; therefore, if one animal is found to be intoxicated by oleander the owner should be alerted to look for additional animals that have potentially been exposed. It is critical to establish a firm diagnosis of oleander intoxication by analyzing biological specimens such as serum, urine, or stomach contents of affected animals for oleandrin (Tor et al. 2005). Suspect contaminated plant material can also be analyzed for oleandrin to prevent fatal oleander poisoning in livestock.

GRASSES THAT CAUSE MECHANICAL IRRITATION

Plants with sharp and barbed bristles or spines can cause abrasions and ulcerations of the mucosa of the lips, oral cavity, tongue, gingiva and oropharynx. The resulting clinical signs of loss of appetite, drooling, oral ulcers, and granulation tissue are particularly serious in horses but have also been reported in cattle (Fava et al. 2000; Kutasi et al. 2017). The oral ulcers are painful and need veterinary care. Deep ulcerations of the tongue, gums, and cheeks, which may lead to abscesses, may also be noticed. Importantly, differential diagnoses include highly contagious viral diseases such as vesicular stomatitis,



Figure 4. Bristlegass (*Setaria sp.*) contamination of hay.
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and exposure to blister beetles or chemicals. Thus, it is important to quickly assess the hay for weed contamination.

Setaria viridis (green foxtail) and *Setaria glauca* (yellow foxtail, yellow bristlegrass, bristly foxtail) have been identified in alfalfa hay. The seed head is a dense hairy panicle 5–30 cm long with spikelets of approximately 2 mm length. The morphology of these species allows them to cause a continual irritation of the mucosal membrane. Mechanical pressure when picking up feed and during mastication of hay is responsible for forcing the awns through the mucus membranes. *Setaria* spp. do not cause lesions deeper in the oropharynx or other parts of the alimentary tract. It is likely that oral grinding and salivary softening of the plant awns make them less harmful when they reach the esophagus and stomach. If hay is contaminated with bristlegrass, it should not be fed to livestock animals. A veterinarian should be consulted to assess the severity of damage and initiate appropriate treatment.

CONCLUSION

Toxic plants can be devastating if they are grazed upon or unintentionally incorporated into hay or silage. The recognition of poisonous plants in hay or forage is essential to the prevention of plant poisonings in animals. Veterinarians and diagnosticians, along with producers and farm managers play important roles as guardians, and all can contribute important information to diagnoses of plant poisonings. Once all the information is available, all evidence is collected, and proper sampling of specimens has occurred, a summary of findings can be provided that will be instrumental in preventing reoccurrences.

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