TOXIC WEEDS AND THEIR IMPACT ON ANIMALS

Birgit Puschner, Amy Peters, Leslie Woods

ABSTRACT

Agriculturally grown plants are potentially contaminated with poisonous weeds. Many potentially toxic plants are found in pastures and crops. Consequently, forage crops at harvest or when directly grazed may contain toxic amounts of poisonous plants. Unfortunately, efficient prevention of contamination with certain poisonous plants is not always feasible or practical, and thus, contaminated feed presents a realistic threat to the livestock industry. Certain toxic plants remain toxic when dried, while others lose toxicity upon drying. Similar results have been observed with ensiling. Each toxic plant needs to be evaluated carefully with regards to its toxicity potential when fresh, dried or ensiled.

Key Words: summer pheasant's eye, poison hemlock, nitrate, pyrrolizidine alkaloids, oleander, toxicosis

INTRODUCTION

Forage crops may contain compounds that may inadvertently affect animals. Several factors contribute to an animal being poisoned by plants. Fundamentally, there is the requirement that a sensitive species of animal ingest, or otherwise be exposed to, a toxic plant at an appropriate time. There are many examples of species differences with regard to sensitivity to the toxic effects of plants. In addition, it is possible for animal species to adapt to a potentially toxic plant if exposure is allowed to occur over a period of time. Disease problems are most commonly caused by forage contamination with poisonous weeds. Even though animals are selective about what they eat, there are instances (for example, herbicide applications) that may change palatability or increase toxicity of some plants. If weeds are embedded in hay cubes, animals may not be able to avoid ingestion of the weeds.

Diagnosing a plant poisoning can be extremely difficult. In many cases, clinical signs are non-specific (such as diarrhea) and post-mortem lesions are not characteristic. Specialized veterinary toxicology laboratories may provide testing for plant toxins, but the assays do not cover the wide variety of plant toxins present in the US. In many cases, 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 (noting that the candidate plants have been chewed and/or finding plant fragments in gastrointestinal tract samples), and to correlate clinical findings, where possible, with those known to be associated with the suspect plant. If diagnostic tests are available, the diagnosis can be confirmed. This is especially important in insurance or

legal investigations. Producers and farm managers, along with veterinarians and diagnosticians play important roles, and all contribute information that may be important to diagnose 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.

Plants of specific concern to contaminate forage crops in CA and the Pacific Northwest include summer pheasant's eye and poison hemlock. Very little information is available with regards to the toxicity potential of summer pheasant's eye to various animal species. While such information is available for poison hemlock, there is limited information with regards to the toxicity potential of silage contaminated with poison hemlock. Recent research results of studies conducted with summer pheasant's eye and poison hemlock are presented. Other toxic weeds of concern to animal health and production include nitrate-accumulating plants, pyrrolizidine alkaloid containing plants and oleander. These plants can be devastating if grazed or found in hay or silage. Thus, recognition of poisonous plants in hay or forage is instrumental to prevent plant poisonings in animals.

Summer pheasant's eye (*Adonis aestivalis*)

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). There is concern that 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 or any other poisonous plant in hay is instrumental in preventing poisoning. *Adonis* spp. are members of the buttercup family (Ranunculaceae) 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., 2004), pigs (Davies et al., 1989) and sheep (Hurst, 1942). Plants of the *Adonis* genus contain cardiac glycosides similar to the toxins present in oleander and foxglove. Clinical signs of summer adonis poisoning in horses include gastrointestinal disturbances, such as colic, hemorrhagic enteritis, diarrhea or decreased gut motility. In addition, the cardiotoxins present in *Adonis* can lead to a variety of cardiac arrhythmias and death. In recent years, several horses died in California as a result of eating grass hay containing summer adonis. Acute and chronic feeding studies conducted in cattle indicated that cattle are less susceptible to summer pheasant's eye than horses. The variable susceptibility of ruminant and monogastric animals may be a result of ruminal metabolism, but other mechanisms cannot be ruled out.

Poison hemlock (*Conium maculatum)*

Poison hemlock (*Conium maculatum*) is found throughout the United States in pastures and crops and is considered very toxic. Consequently, forage crops at harvest or when directly grazed could contain toxic amounts of poison hemlock. Although livestock seldom eat hemlock because of its strong odor, they will ingest it if no other forage is available or if it ends up as a contaminant in hay or silage. Poisonings have been described in many species, including cattle (Galey et al., 1992), sheep (Panter et al., 1988), horses (Keeler,et al., 1980), pigs (Widmer,

1984), goats (Copithorne, 1937) and poultry (Frank et al., 1987). The toxicity of poison hemlock is caused by a variety of piperidine alkaloids, with coniine and γ-coniceine representing the most predominant toxins. The concentrations of alkaloids depend on various factors, including temperature, moisture, time of day, plant growth stage, and plant tissue. In general, the concentrations of coniine increase during the ripening and drying periods, while the concentrations of γ-coniceine decrease. There are no specific methods reported that examined methods to degrade piperidine alkaloids or reduce their toxicity. It is known that drying of fresh *C. maculatum* under the sun for seven days produces an important loss of alkaloids, although contaminated hay has caused deaths in cattle. The toxicity of *C. maculatum* in silage is not known, but livestock may be exposed to silage contaminated with poison hemlock. Silage making has been used as a means to reduce mycotoxin concentrations and prevent formation of mycotoxins in a variety of crops (Scudamore et al, 1998). The fermentation process leads to a conversion of carbohydrates to organic acids assuring preservation. In addition, variable degradation of proteins to amino acids and other compounds occurs. The exact mechanisms that lead to the detoxification of mycotoxins during fermentations have not been established, but may be a result of enzymatic transformation as well as spontaneous degradation under acidic conditions. A study was conducted to examine the feasibility of ensiling poison hemlock as a method for degradation of toxins and whether silage containing poison hemlock can safely be fed to livestock. The major piperdine alkaloids, coniine and γ-coniceine concentrations were determined before, during and at the end of the fermentation process to evaluate the effect of the ensiling process on the toxicity potential of *Conium maculatum*. The concentration of γ-coniceine decreased during the ensiling process, while the concentration of coniine increased. Despite the apparent reduction in γ-coniceine concentrations during ensiling, ensiled poison hemlock cannot be considered safe for animals. The remaining coniine concentrations after the ensiling process pose a significant risk. The practice of ensiling poison hemlock contaminated forage is therefore not recommended. Careful inspection and testing of forage that may be contaminated with poison hemlock is suggested to avoid intoxication in livestock.

Nitrate accumulating plants

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. 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 alkaloid accumulating plants

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).

Oleander (*Nerium oleander)*

Oleander (*Nerium oleander*) intoxication is one of the most common plant-associated poisonings in California. The California Animal Health and Food Safety Laboratory at UC Davis has diagnosed oleander poisoning in over 100 cases since 2005. The affected species included horses, cattle, goats, llamas, and alpacas. Oleander intoxication in several other species such as sheep, cats, dogs, birds and humans have also been reported in the literature. Most often, intoxication in animals results from accidental ingestion of plant parts (Galey et al., 1996). As few as 5 -10 leaves of oleander can cause severe diarrhea, cardiac arrhythmias and death in cattle and horses within 2 to 8 hours of consumption (Galey et al., 1998). Oleandrin is the primary cardiotoxic glycoside in oleander. There are no published studies regarding effective treatment of oleander intoxication in animals. Generally, the treatment has consisted of symptomatic and supportive care. Unfortunately, there is no specific treatment for oleander poisoning. The best approach to oleander intoxication is avoiding exposure to this extremely toxic plant 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. Highly specific and sensitive diagnostic testing is available to detect oleandrin in suspect plant material and specimens from animals, including gastrointestinal contents, liver, serum and urine (Tor et al., 2005).

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