

POISONOUS PLANTS THAT CONTAMINATE HAY AND FORAGES IN THE WESTERN UNITED STATES¹

Bryan L. Stegelmeier²

ABSTRACT

When alternative forages are available, grazing animals generally avoid eating poisonous plants. However, when toxic plants are included in prepared feed and hay, they are often consumed, and they can poison animals. Alternatively, some normally safe forages can under certain conditions, produce and accumulate toxins and poison livestock. As poisoning often results in fatal or costly debilitating disease, identifying toxic plants and their toxins in prepared forages is essential. The purpose of this presentation is to review basic principles of identifying contaminated feeds, review how to collect and where to have samples analyzed using case studies. Participants of these discussions should become familiar with common poisonous plants in the western United States and know how to get help in identifying and avoiding poisoning.

Key Words: poisonous plants, hay, bloat, nitrate, pyrrolizidine alkaloids, poison hemlock, black nightshade, milkweed, cyanogenic plants, knapweed, yellowstar thistle, black henbane, *Salvia reflexa*

INTRODUCTION

Case Presentation: A producer seeded a 120-acre irrigated field with Roundup Ready® alfalfa in late spring. After planting a storm developed and high winds further seeded the field from every weed available from the adjacent marginal property. Consequently, a forest of weeds and a little alfalfa sprouted and quickly grew. The producer consulted with the seed company and they decided the problem might be best resolved if they cut the contaminated hay and sprayed the field with herbicide when the second crop started growing. They cut the weedy hay and stacked it over on the corner with the intention of composting it. However, it was a challenging year and hay was in short supply. A relative had some hungry cows and needed feed. So, after consulting with the experts, they tested the hay for nitrates and decided to try and feed it. They were cautious and planned to transition to the weedy hay slowly while monitoring for signs of toxicity. So, they diluted the hay 1:1 with grass hay and spread it out through their feedlot. Within 12 hours of feeding, half of the 500-cow herd became anorexic, depressed with signs of colic or malaise. Though much of the hay was consumed, the unconsumed part was removed. About 200 of the exposed cows were severely poisoned and they became reluctant to stand, recumbent, and bellowing in pain. A 172 that were fatally poisoned and most died within 72 hours of

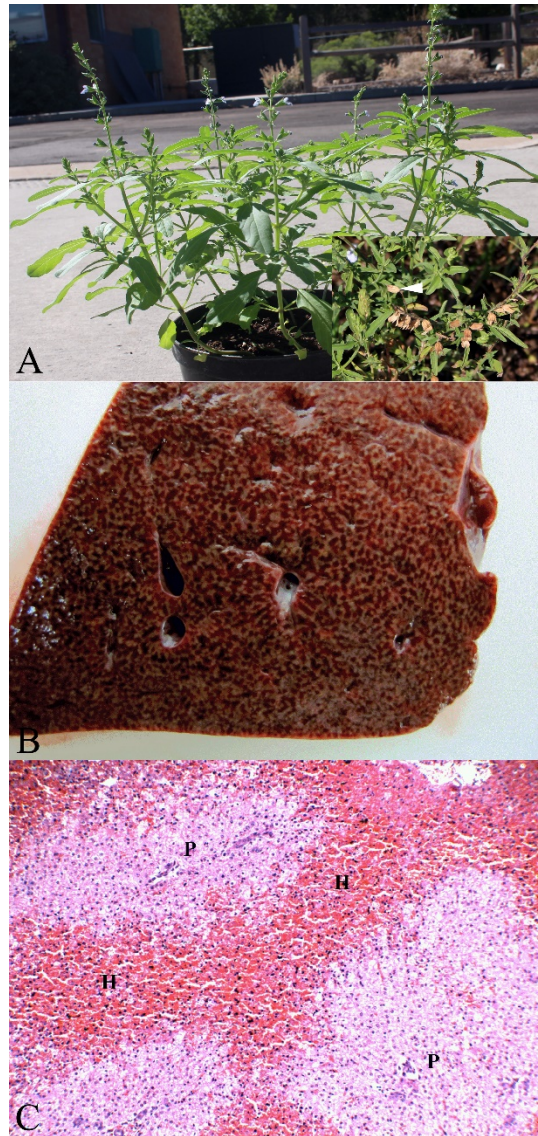
¹ Portions including tables and photographs have been published in Rangelands 34:2-11, 2012

² B.L. Stegelmeier, DVM PhD Diplomat ACVP
USDA/ARS Poisonous Plant Research Laboratory
1150 East 1400 North, Logan UT 84341
Telephone: 435-752-2941
Email: bryan.stegelmeier@usda.gov Internet Page: www.ppri.ars.usda.gov

In: Proceedings, 2021 Western Alfalfa & Forage Symposium, Reno, NV 16-18 November 2021. (<http://alfalfa.ucdavis.edu>).

exposure. Three of those that died were necropsied and all had massive liver necrosis. The timing and presentation suggest poisoning relating to the weedy bales. Likely toxic plants that could produce such severe liver necrosis include annual kochia, cocklebur, and *Wedelia glauca*. As kochia had been linked to several similar catastrophic poisonings and many of the bales were estimated to contain 70% or more kochia, that was considered a likely cause. However, the massive liver necrosis resembled cocklebur poisoning making it also a likely cause. *Wedelia* was considered unlikely as it has been associated with liver disease, but not in North America. Consequently, the remaining contaminated hay was collected, sampled, and fed to both naïve cows and several cows that were involved in the original case. None of the hay samples contained carboxyatractylocide (the cocklebur toxin) or any of the other aflatoxins that were tested. The previously poisoned cattle would not eat the contaminated hay. Grinding and administered the hay by pumping it into the rumen also did not produce disease. When cattle forced to eat the hay, they would carefully pick through it. The remaining uneaten feed was collected and found to contain significant amounts of *Salvia reflexa* or lance leaf sage. When those plant parts or other plants that were collected from near the original field were dosed to cattle it produced hepatic necrosis (see figure 1). Using a mouse bio-model the *Salvia* toxins were identified and their presence was confirmed in both the original contaminated hay and rumen contents from those poisoned cattle.¹

Figure 1: *Salvia reflexa* and *S. reflexa*-induced disease in cattle. A- *S. reflexa* grown from seeds recovered from contaminated hay. Notice the blunt tipped leaves. The flowers are pale blue in clusters on the tips of the branches. The inset is an older plant with 0.5 cm calyxes (arrowhead). The calyxes and the leaves are the most toxic and were plentiful in the fines or remaining feed previously exposed cattle would not eat. B- is the liver from a cow that was poisoned with *S. reflexa*. Notice the marked lobular pattern caused by lobular necrosis and hemorrhage. C- photomicrograph of the liver shown above. Notice the hemorrhage and necrosis (H) with relative sparing of portal liver cells (P).



This is a great example of a lot of work and great success as both the cause of poisoning was identified and testing was developed to identify the plant in contaminated feed and in the tissues of poisoned animals. Sadly, this is unusual as many other possible poisonings remain unsolved. The objectives of this presentation are to provide you with some direction to help you identify common plant poisoning and more importantly how to assemble a team of collaborators to help you come up with answers that will help you identify potential problems, diagnose poisoning when it occurs and develop methods to minimize the effects of poisoning.

SAMPLING AND PLANT IDENTIFICATION

Many toxic plants are exotic, noxious weeds that invade pastures and fields and many have the potential to be harvested with the desired forage. The subsequent contamination is nearly always patchy. Additionally, the nature of harvesting and storing forages smashes, fractures, ensiles and possibly cooks fragile plant parts resulting in destruction of many of the characteristic plant structures that are essential for identification. To overcome these problems, adequate sampling of prepared forages is difficult. For example, hundreds of samples may need to be examined to identify the 4 or 5 contaminated bales from a field of hay. As free standing plants are much easier to identify, it is most effective to inspect fields before the forage is harvested and processed. As knowing and identifying all potentially toxic plants can be challenging, consulting with local extension agents and veterinarians who are familiar with local plants and problems can be often helpful. Unidentified plants may be collected and sent to a local laboratory, plant taxonomist or herbarium. Positive identification is more likely if an entire plant especially flowers and seed pods are submitted. If a local herbarium is not available, the sample can be dried and pressed between papers pressed two or three days down under several weights such as a couple of large books. The pressed plant can then be taped to a piece of cardboard, or placed in a folder, and mailed to the appropriate laboratory (see Table 1).

Toxin concentration in plants often varies and some populations may not be poisonous. In many cases plants can be analyzed chemically to determine their toxicity and potential risk. For chemical analysis potentially toxic plants can be collected green and frozen. If plant samples cannot be maintained frozen until analysis, some plants can be dried before shipment. It is advised that the appropriate laboratory consulted to ensure samples are properly collected, preserved, shipped, and received at the laboratory (Table 1). Under some growing conditions, many normally safe forages can accumulate toxins including nitrates or cyanogenic glycosides. Under such questionable conditions, these forages should be sampled and analyzed to determine their potential toxicity. As toxin distribution may not be homogenous, analyzing multiple samples is also recommended. Most laboratories include multiple samples in their analysis and report a range of results to ensure the resulting recommendations are reliable (see Table 2).

Table 1: List of potential references and resources

	Phone Number	Web Page
Local Extension Agent		http://www.csrees.usda.gov/Extension
USDA/ARS Poisonous Plant Research Laboratory	435-752-2941	https://www.ars.usda.gov/pacific-west-area/logan-ut/poisonous-plant-research/docs/bulletin-415/
State Animal Disease Diagnostic Laboratory		http://www.aavld.org/mc/page.do?sitePageId=33930&orgId=aavld (Laboratories Accredited by American Association of Veterinary Diagnostic Laboratories)
Local Herbarium		http://herba.msu.ru/mirrors/www.helsinki.fi/kmus/botmus.html (List of public herbaria throughout the world)
Microscopic analysis of feces and ingesta to detect toxic plant parts- Texas Veterinary Medical Diagnostic Laboratories	888-646-5623	http://tvmdlweb.tamu.edu

DIAGNOSIS OF POISONED LIVESTOCK

When livestock are poisoned, clinical studies and post mortem examination are essential to obtain a good diagnosis. As many nutritional, genetic and infectious diseases can cause clinical diseases and lesions similar to those produced by toxic plants, veterinary examination and collection of the appropriate samples for chemical analysis and microscopic analysis are critical. As discussed with plant samples submission, expert consultation may also be needed to make the proper sample collection, preservation and submission. Most states have excellent animal diagnostic laboratories and if available submission of severely affected, moribund animals or fresh animals that have recently died provides the best chance of obtaining a definitive diagnosis and cause of death. Expertise is needed to collect the proper samples as many are unique and not immediately obvious. For example, the ocular fluid is relatively protected within the sclera and other eye tissues so that it changes very slowly after the animal dies. Subsequently elevated ocular nitrates are excellent post mortem indicators of plant-associated nitrate poisoning. In many cases the cause of death is not obvious even with post mortem and histologic studies. Such negative results should not be discouraging as they can be used to exclude many potential diseases and narrow the list of possible causes. Many studies result in a most likely diagnosis and when integrated with local field studies can be useful to formulate an appropriate action to avoid future problems or minimize the impact of that likely toxic plant.²

Table 2: Partial list of tests, samples, sample size and preferred method of shipping for investigation of potential poisoning by plant toxins.*

Test	Sample	Size	Shipping
Blood counts	EDTA containing blood tube (often a purple topped tube)	3-5 ml	Chilled shipped on ice
Serum biochemistries	Clot tube (often a red-topped tube)- contains no anticoagulant. The serum is separated from the cellular portions after clotting.	5-10 ml	Chilled on ice or frozen for extended delivery
Microscopic evaluation of tissues	Various tissues (liver, heart, lung, kidney, GI tract, brain, and any lesions)	2X2X4 cm pieces	Fixed in formalin
Post mortem or necropsy	Dead or moribund animal	Whole animal	Fresh
Chemical evaluation of serum, blood, eye fluid, urine or milk	Serum, whole blood, urine or milk. The whole eye is the best sample to send if nitrate poisoning is suspected.	20 ml	Stored in tubes or plastic bags and shipped on ice or frozen
Chemical evaluation of tissue	Various tissues	2X2X4 cm pieces	Stored in plastic bags and shipped frozen
Chemical evaluation of feces or gastrointestinal contents	Feces or ingesta	1 kg (about a sandwich bag full)	Stored in plastic bags and shipped frozen
Plant identification	Whole plant	Whole plant including flowers, pods, leaves, stems and roots	Fresh if delivered that day, dried if hand delivered later, pressed

			and dried if sent through the mail
Plant chemical analysis	Whole plants	5 or 6 whole plants	Fresh if delivered that day, dried if mailed or frozen if they can be maintained frozen during shipping
Hay for weed contamination and weed identification	Stored baled hay	5 or 6 bales	Dry
Hay for nitrate analysis	Hay	Several representative samples. These can be core samples. 0.5-1 kg	Dry
Prepared feeds	Feeds	Representative feed samples such as cubed feed, 0.5-1 kg	Dry
Silage or green chopped feed	Feeds	Representative feed samples- 3-5 kg	Frozen

***Be sure to check with the laboratory as they often require specific sampling, sample preparation and shipping. Label all materials with indelible ink; provide date, owner, location and contact information.**

SPECIFIC POISONOUS PLANTS AND PLANT ASSOCIATED TOXINS

The following are some of the common toxins and poisonous plants that commonly contaminate feeds and forages in the western United States. This list is not all inclusive and regional problems may involve different plants. Additional texts, reviews and web pages contain additional information on these and other toxic plant problems.^{3,4}

Plant Associated Bloat and Rumen Acidosis: Bloat is probably the most common fatal plant poisoning. Bloat is a common name for increased rumen pressure or tympany. It has various causes, but the most common plant associated bloat is frothy bloat. Common plants associated with bloat include alfalfa, ladino clover, red and white clover, green cereal crops, rape, kale and others. Most contain soluble and quickly digested protein, saponins and hemicelluloses that alter rumen surface tension and stabilize surface foam.⁵ Tannin containing legumes such as sainfoin, crown vetch, milk vetch, fenugreek, and birdsfoot trefoil have similar proteins, but they are more slowly digested and rarely cause bloat.⁶ Recently bloat resistant cultivars of alfalfa and other desirable species have been developed to minimize bloat incidence and severity. Bloat is most often seen when animals are allowed to graze rapidly growing lush pastures, but it can also be seen when animals are fed high quality hay. Frothy bloat can also be seen in animals fed grain diets. These cases are probably related to both rapid fermentation, acidosis, and microbial production of foam stabilizing compounds.⁷

Clinically, bloat is seen as marked distension of the abdomen with prominent rumen distension seen in the left paralumbar fossa. This distension and pressure results in dyspnea (labored breathing) seen as grunting and open mouthed breathing, extension of the neck, and regurgitation of rumen contents. If untreated, animals may quickly die. Death often occurs within hours of exposure. Emergency treatment is to reduce rumen pressure via a trocar, cannula, or rumenotomy. In less severe cases rumen pressure can be relieved using a stomach tube. Antifoaming agents such as vegetable and mineral oils with various surfactants such as poloxalene are also commonly used. Preventing bloat is difficult and most strategies only

reduce the incidence. Supplemental feeding, mixed seeding, and strip grazing can be used to limit the doses of quickly growing legumes to less than 30% of the diet. Antifoaming agents mixed with minerals can be used to reduce the risk. Timed release rumen capsules containing ionophores are also helpful.

Rumen acidosis is a complex disease involving the interaction of feed and rumen microflora. Simplistically it results when forages or mixed rations include quickly fermentable carbohydrates causing expansion of fermenting rumen microflora, production of lactic acid, and subsequent damage to the rumen mucosal structure and function. Many cases are fatal and non-fatally poisoned animals may develop secondary septicemia and hepatic abscesses. Treatment is symptomatic and most poisoning can be avoided by controlling feed rations, allowing cattle time to adapt to concentrate rations, and the use of ionophores and antibiotics to influence rumen microflora.⁸

Nitrate-nitrite poisoning: Under some weather and growth conditions certain pasture and cultivated forages accumulate toxic nitrate concentrations. Ruminants are especially sensitive as they quickly reduce nitrates to nitrite. The reduced nitrite is absorbed and oxidizes hemoglobin producing methemoglobin. Methemoglobin will not bind oxygen. In monogastric animals such as horses the nitrates are not so easily reduced and much higher doses are required to be toxic. Signs of nitrite poisoning include weakness, trembling, brown or cyanotic mucous membranes, dyspnea, brown discolored blood, abortion and death. Poisoning is cumulative and all other potential sources such as water, and feed additives should be included when considering poisoning. Nitrates accumulate in all plant parts but may be especially high in stalks and leaves. Seeds or grains are generally safe. Plants predisposed to accumulate toxic nitrate concentrations (>0.5%) are listed in Table 3 and Figure 2.⁹ Nitrate accumulation is provoked by nitrogen fertilization, drought or frost stress and some herbicide treatments. Water may be contaminated with fertilizer, silage-pit or feedlot runoff.

Poisoned animals may be treated with intravenous methylene blue (8 mg/kg in cattle). As methylene blue is rapidly cleared, treatment may need to be repeated every 2 hours. Other suggested treatments include oral gavage with mineral oil, antibiotic and vinegar to reduce nitrite production and enhance toxin movement through the gastrointestinal tract.



In most cases the animals die quickly precluding treatment. The diagnosis postmortem is best made analyzing the eye for nitrates. The whole globe should be submitted as the nitrates in this tissue are slow to be altered by autolysis and saprophytic bacteria (see Table 2).

Figure 2: Kochia (*Kochia scoparia*) is an annual Asian weed that grows rapidly into a 2 m large branching plant, with alternate hairy leaves. It is commonly found in disturbed areas of fields, pastures and rangelands. In some parts of the world it is used as forage, but in North America it has been associated with liver disease, photosensitivity, polioencephalomalacia, and nitrate poisoning. Other than nitrate poisoning, the toxin or mechanism of toxicity has never been proven for kochia.

Nitrate poisoning can be prevented by recognizing crops, weeds, and forages that are likely to accumulate nitrates and avoiding contact with susceptible species. Forage nitrate concentration

of >0.5% and water concentrations >200 ppm should be considered dangerous. Contaminated forages can still be used if they are diluted with good feed or fed to less susceptible species.

Table 3: Partial list of plants predisposed to accumulate toxic nitrate concentrations (>0.5%) that may contaminate hay and prepared forages.⁹

Sudan grass (<i>Sorghum bicolor</i>)	Fescue (<i>Festuca</i> spp.)	Dock (<i>Rumex crispus</i>)
Johnson grass (<i>Sorghum halepense</i>)	Alfalfa (<i>Medicago sativa</i>)	Nightshade (<i>Solanum</i> spp.)
Sorghum (<i>Sorghum</i> spp.)	Sweet clover (<i>Melilotus officinalis</i>)	Soybean (<i>Glycine max</i>)
Fireweed (<i>Kochia scoparia</i>)	Flax and linseed (<i>Linum</i> spp.)	Pigweed (<i>Amaranthus</i> spp.)
Beets (<i>Beta</i> spp.)	Lambsquarter (<i>Chenopodium album</i>)	
Rape (<i>Brassica</i> spp.)	Canadian thistle (<i>Cirsium arvense</i>)	
Corn (<i>Zea</i> spp.)	Jimson weed (<i>Datura stramonium</i>)	
Rye (<i>Secale cereal</i>)	Wild sunflower (<i>Helianthus</i> spp.)	
Wheat (<i>Triticum</i> spp.)	Cheeseweed (<i>Malva parviflora</i>)	
Oats (<i>Avena sativa</i>)	Smartweed (<i>Polygonum</i> spp.)	

Pyrrolizidine alkaloid containing plants: This group of over 6000 plants contain over 650 different toxic pyrrolizidine alkaloids (PAs). In the western United States, there are several indigenous PA containing plants, but those that commonly contaminate hay and poison animals tend to be exotic, invasive, noxious weeds. As a group, PA containing plants are not very palatable, but when they are included in hay or other prepared forages, they are readily accepted and eaten. The PA toxins primarily damage the liver, resulting in non-specific but suggestive histological changes (liver necrosis, fibrosis, biliary hyperplasia and megalocytosis). These lesions are dependent on the dose and duration of poisoning. High doses produce extensive damage or necrosis of the liver. This is seen clinically as acute liver failure with jaundice (icterus), coagulopathy and hepatic encephalopathy. Lower doses are more common and also damage the liver. However, in these cases the liver damage is less severe, but often results in compromised animals with reduced hepatic function. Such animals may decompensate and develop clinical liver disease months and even years after the initial exposure. Clinical disease in these animals includes icterus, photosensitivity, weight loss and poor production. Such liver failure is often precipitated by a stressful event such as pregnancy, lactation, poor nutrition or bad weather. This delayed onset of disease complicates diagnosis as the causative contaminated feed is gone and cannot be examined.



Figure 3: Hound's tongue (*Cynoglossum officinale*) is a biennial noxious weed originally from Euroasia that has spread throughout North America. The leaves of the first year rosette are long (40+ cm) and broad, hence the common name. In its second year the plant produces flowering stems that are about 0.5 m tall and topped with reddish purple flowers that ripen into small 8-10 mm nutlets that are covered with hooked barbs that easily attach to hair and clothing.

Most PA containing plants are invasive noxious weeds of varying toxicity making it difficult to predict the risk and the extent or effect of poisoning. Animal and individual susceptibility to poisoning also differs. Horses and cattle are more susceptible than sheep and goats. Young animals are often much more susceptible than mature animals and there are reports of transmammary neonatal poisoning without

clinical signs of maternal toxicity.¹⁰ Selected PA containing plants (Table 4), include hound's tongue (*Cynoglossum officinale*- Figure 3), and several different *Senecio* species (See tansy ragwort- *Senecio jacobaea*- Figure 4). PA containing plants are the poisonous plant most likely to contaminate grain and herbal products. Consequently human poisonings and deaths have been reported.¹⁰ PA containing plants remain toxic in prepared feeds and food with minimal degradation in most ensiling processes.¹¹ Cereals products can be contaminated by dust alone when they are co-harvested with PA containing plants. PAs have also been identified in honey, milk and animal tissues (see Figure 6- *Amsinckia*).¹²

As most animals that develop clinical disease die, treatment of poisoned animals is limited. Though little is known about sub-clinically poisoned animals, it is speculated that they have permanent loss of hepatic function and are not likely to perform to their full potential. Consequently avoiding exposure is the suggested treatment. As with many other toxic plants, the best way to ensure feed quality is to inspect the fields for invasion by these plants prior to harvest.

Figure 4: Tansy ragwort (*Senecio jacobaea*) is a noxious weed that is a native of the British Isles that has spread into Western Europe, South Africa, Australia, New Zealand and North America. It commonly invades fields and pastures in the Pacific Northwest. In spite of extensive control efforts using chemical and biologic agents, it continues to be a problem. Tansy ragwort is a tall (0.5-1.5 m), erect plant that is unbranched except at the inflorescence. Poisoning occurs when plants contaminate feeds, when grazing animals cannot easily differentiate the early rosette from grasses and clovers, or when no other forages are available. *S. jacobaea* contains six major alkaloids of which several have been shown to cross the placenta and to be secreted in milk.





Figure 5: Fiddleneck (*Amsinckia intermedia*) is an annual that has spread over much of the southwest. The flower is yellow or orange and it is shaped like the neck of a fiddle resulting in the common name- fiddleneck. Poisoning is sporadic and most often affects highly susceptible young animals especially horses. When other forages are lacking, resistant animals often seem to eat fiddleneck without problems, but it is likely that such subclinical poisonings have permanent sequelae that may not be seen until months or years later. Fiddleneck contains various PAs including intermedine and echiumine. Similar Australian plants suggests that these toxins have the potential to contaminate honey and pollen.

Table 4: Partial list of pyrrolizidine alkaloid containing plants that have poisoned livestock and may contaminate hay and prepared forages.¹²

<i>Senecio douglasii</i> var. <i>longilobus</i>	Woody groundsel
<i>S. glabellus</i>	Butterweed
<i>S. intergerrium</i>	Lamb's tongue groundsel
<i>S. jacobaea</i>	Tansy ragwort
<i>S. plattensis</i>	Prairie ragwort
<i>S. riddellii</i>	Ridell's ragwort
<i>S. spartioides</i>	Broom groundsel
<i>S. vulgaris</i>	Common groundsel
<i>Crotalaria retusa</i>	Wedge leaf rattlebox
<i>C. sagittalis</i>	Rattlebox
<i>C. spectabilis</i>	Showy crotalaria or rattlebox
<i>Amsinckia intermedia</i>	Fiddleneck, tarweed
<i>Cynoglossum officinale</i>	Hound's tongue
<i>Echium vulgare</i>	Blue weed, Viper's bugloss
<i>Symphytum officinale</i>	Comfrey

Poison hemlock- *Conium maculatum*: Poison hemlock (Figure 6) is an introduced biennial or perennial plant that grows along roads, ditches and fences. It is a prolific seed producer allowing it to spread and invade in large patches into adjacent fields and pastures. Animals most often avoid eating poison hemlock unless there are no other forages available. The toxins (conine and gamma-coniceine) are similar to nicotine causing muscle tremors, salivation, incoordination,



dyspnea, increased defecation and urination and death. If not acutely fatal, abortions and birth defects also occur.

The leaves and stems are toxic and may contaminate hay and silage. The seeds are highly toxic and can contaminate cereal grains. Fortunately, poison hemlock toxins are not as stable as many other plant toxins and they degrade over several months in most hay and prepared feeds. However, this degradation may not be uniform, resulting in some portions of the prepared feed having residual toxicity. Nearly always the fresh plant material is uniformly toxic and most poisonings occur when animals are fed contaminated fresh, chopped forages.¹³

Figure 6: Poison hemlock (*Conium maculatum*) is an erect biennial that grows up to 2 m tall. Originally from Europe, it now grows throughout North America and it is commonly found along ditches and roads where the ground is moist. The stems are smooth and hollow and they often have purple spots. The leaves are carrot-like, alternate, pinnately dissected and coarsely toothed. The inflorescence is a flat topped umbel with multiple small white flowers.

There are no specific treatments for poison hemlock poisoning and most animals recover if they are allowed to recover with minimal stress. Supportive care might include a cathartic or administration of activated charcoal to minimize absorption. The contaminated feed should be removed to prevent further poisoning.

Black nightshade (*Solanum nigrum*), jimson weed (*Datura stramonium*) and henbane (*Hyoscyamus niger*): Black nightshade (Figure 7) is found along fences and roads throughout North America. It contains several glycoalkaloids, including solanine, hyoscyne and hyoscamine. These toxins are similar to atropine and alter the function of the autonomic nervous system. Clinical signs include reduced salivation and decrease gastrointestinal motility. Under some conditions this can result in epidemic incidences of colic. Similar problems have been documented to occur when grains contaminated with jimson weed seeds were fed to horses in the Midwest.¹⁴ Nightshade toxins are stable and if they are included in forages they remain toxic. They are especially bothersome to the racehorse industry as these toxins can cross react in the drug screens that are used to test racehorses. In most of these cases, identifying contaminating

plants in feeds is difficult. Consequently, such false positives usually result in disqualification of those animals.¹⁵

Henbane is a noxious weed that also grows along fences, roadsides and waterways. It can invade fields and pastures and can contaminate hay and other prepared forages. The henbane toxins (hyoscyamine, hyoscyne and atropine) are similar to those in black nightshade and jimson weed, so they cause similar diseases to these plants. Henbane is generally not palatable unless it is included in prepared forages.

Figure 7: Black nightshade (*Solanum nigrum*) is a spineless erect or trailing annual plant that grows along fences and roads and on the disturbed margins of fields. It has a small white flower that ripens into a 0.5 to 1 cm round smooth green fruit that contains the toxin, solanine. When the fruits turn black, they are edible.



Milkweeds (*Asclepias* spp.) and other cardiac glycoside containing plants: Milkweeds (Figure 8) can be found throughout the world as they grow along roadsides, waterways and in disturbed areas. Most species contain cardenolides or cardiac glycosides, but the concentrations of these vary between the species and plants. These toxins are similar to digoxin (the toxin in



foxglove, *Digitalis lannata*) that is widely used to treat congestive heart failure.

Cardenolides inhibit the sodium-potassium pumps on myocardial membranes resulting in altered conduction and contractility. Similar toxins can also be found in oleander (*Nerium oleander*), lily of the valley (*Convallaria majalis*), dogbane or Indian hemp (*Apocynum cannabinum*), and pheasant's eye (*Adonis microcarpa*). Milkweeds are most toxic when in the vegetative stage and they remain toxic when included in hay and other forages.

Signs of poisoning are usually sudden death, dyspnea or labored respiration, pulmonary edema, muscular tremors and a weak rapid pulse. As no specific treatment is available in large animals, prevention is essential. As

milkweeds tend to grow in small patches they can be manually removed or treated with various herbicides.

Figure 8: Milkweed (*Asclepias subverticillata*) - Milkweeds are found throughout the world along road, waterways, and in disturbed areas on the margins of fields and pastures. They are erect perennials that generally have broad veined leaves. Most contain milky sap or latex and the flowers are usually terminal umbels colored from greenish white to red.

Cyanogenic glycosides containing plants: More than 2000 plants throughout the world have been shown to contain cyanogenic glycosides. Only a handful of these have been associated with poisoning in livestock in the western United States and several of these are often used as

chopped green forage and to make hay. Johnson grass (*Sorghum halapense*), Sudan grass (*Sorghum vulgare*), forage sorghums (*Sorghum* spp.), and arrowgrass (*Triglochin* spp. Figure 9) are generally used as hay. Other cyanogenic plants such as chokecherry (*Prunus virginiana* Figure 10) and service berry (*Amelanchier alnifolia*) often grow along fences and field margins and can inadvertently be included.



Figure 9: Arrowgrass (*Triglochin maritima*) is found throughout North America in moist marshlands and pastures. It is a perennial grass-like plant and the leaves are between 15 and 30 cm long, linear, unjointed and sheathed at the base. The flower is composed of a pediceled raceme that may grow up to 1.5 m tall. The greenish flowers are inconspicuous and ripen into greenish fruits. Cyanide is highly toxic to all animals as it inhibits cellular respiration. Affected animals cannot use oxygen and develop “cherry red” tissues and blood. Low, non-lethal doses of cyanide have been associated with lathyrism-like disease, goiter, birth defects such as arthrogryposis, and spinal cord degeneration and cystitis. The mechanism of many of these changes is due to damage to nerve coverings called myelin sheaths.



Figure 10: Chokecherry (*Prunus virginiana*) is a small tree or shrub that grows in thickets and along waterways. It can grow up to 5 m tall and the leaves are ovate to obovate with minimally serrate margins. The bark is grey with obvious lenticles. The inflorescence is a cylindrical raceme of white fragrant flowers that ripen into dark purple round fruits. The fruit is the only edible portion.

To be toxic cyanogenic glycosides must be hydrolyzed to cyanide or prussic acid. This conversion is facilitated when the plant is damaged as by crushing, chewing, freezing, or wilting. Drying or ensiling the plants decreases the cyanogenic potential as the cyanide is slowly degraded and released over time. The concentration of cyanogenic glycosides in plants varies and can be higher in young plants growing rapidly in cold moist weather; when heavily fertilized; when frosted or drought stressed; or when the plants are treated with certain herbicides.

Treatment of cyanide poisoning is difficult as most animals quickly die. As cyanide is quickly dissipated from tissues, tissues such as liver, muscle and rumen contents must be collected within a couple of hours of death, frozen in sealed, air tight containers and quickly analyzed. If poisoned animals are found, recommended treatments include intravenous sodium nitrite (22 mg/kg) and sodium thiosulfate (600 mg/kg). These oxidize hemoglobin forming

methemoglobin. Methemoglobin avidly binds cyanide protecting the cytochrome oxidase system of cellular respiration from its effects. The best treatment is to avoid harvesting and feeding these plants when they are likely to be toxic. Potential toxic feeds can be tested for their cyanogenic potential.

Russian knapweed (*Rhaponticum repens*) and yellow star thistle (*Centaurea solstitialis*):

Russian knapweed (Figure 11) and yellow star thistle (Figure 12) produce a unique disease that is specific for horses. These plants are both invasive species that grow in disturbed areas, pastures and paddocks throughout the western United States. Though several toxins have been identified and proposed, none have been definitively confirmed as the cause of this disease. This is largely due to the extended duration of exposure that is required to produce the disease. All parts of the plants are toxic and they remain toxic when dried and included in hay. Poisoning is seen after the horses have eaten the plant for between 30 and 60 days. Affected animals lose the ability to prehend and chew food and to drink water. The lesions are degeneration and necrosis of specific locations in the brain known as the substantia nigra and the globus pallidus, resulting in the morphological description of nigropallidal encephalomalacia. No other disease or toxin has been shown to have such a site-specific effect.

Figure 11: Russian knapweed (right) (*Centaurea repens*) is a Russian plant that has invaded many parts of the world. It is a perennial erect plant whose branches can grow to about 1 m tall. The leaves are alternate with toothed margins. The thistle like flowers are 1-2 cm in diameter and range from lavender to white. The seeds are white with bristles on one end.



Figure 12: Yellow star thistle (left) (*Centaurea solstitialis*) is a noxious weed from the Mediterranean that is well established in the western United States. It grows in disturbed areas along fields, roads and waste areas. It is an annual branching herbaceous weed that grows about 30 cm tall. The leaves vary from deeply lobed at the base to linear and entire on the stems. The flowers are yellow and the bracts



are tipped with stiff yellow spines (10-20 mm).

There is no effective treatment for poisoning. Once clinical signs begin, poisoning is nearly always fatal. Affected horses may be treated and maintained with liquids and oral liquid diets, but they do not recover and nearly always continue to deteriorate until they must be euthanized.¹⁶ The best treatment is management to control the plants to minimize exposure and prevent them from contaminating forages.

SUMMARY

Many poisonous plants remain toxic and can poison animals when they are included in hay or prepared feeds. Hay fields should be closely inspected and potentially poisonous plants should be excluded from harvesting. Other, presumably safe, forages may accumulate saponins, highly soluble carbohydrates, nitrates or cyanogenic glycosides and this potential should be recognized. Such forage may be tested and treated accordingly to minimize the risk of poisoning.

REFERENCES

1. Panter KE, Stegelmeier BL, Gardner DR, et al. Clinical, pathologic, and toxicologic characterization of *Salvia reflexa* (lance-leaf sage) poisoning in cattle fed contaminated hay. *J Vet Diagn Invest*. May 2021;33(3):538-547.
2. Stegelmeier BL, Davis TZ, Clayton MJ, Gardner DR. Identifying Plant Poisoning in Livestock in North America. *Vet Clin North Am Food Anim Pract*. Nov 2020;36(3):661-671.
3. Knight AP, Walter RG. *A Guide to Plant Poisoning*. Teton Newmedia; 2001:1-367.
4. Burrows GE, Tyrl RJ. *Toxic Plants of North America*. Wiley Blackwell 2013:1-1277.
5. Kahn CM, Line S, Atello SE. Bloat (Ruminal Tympany). *The Merck Veterinary Manual*. Courier Kendallville Inc.; 2011:205-209.
6. Lees GL. Condensed tannins in some forage legumes: their role in the prevention of ruminant pasture bloat. *Basic Life Sciences*. 1992;59:915-934.
7. Cheng KJ, McAllister TA, Popp JD, Hristov AN, Mir Z, Shin HT. A review of bloat in feedlot cattle. *J Anim Sci*. 1998;76(1):299-308.
8. Underwood WJ. Ruminal lactic acidosis .1. epidemiology and pathophysiology. *CompendContEducPractVet*. 1992;14(8):1127.
9. Burrows GE, Tyrl RJ. Plants causing sudden death in livestock. *Veterinary Clinics of North America - Food Animal Practice*. 1989;5(2):263-289.
10. Huxtable RJ. Human health implications of pyrrolizidine alkaloids and herbs containing them. In: Cheeke PR, ed. *Toxicants of Plant Origin*. CRC Press; 1989:41-86.
11. Candrian U, Luthy J, Schmid P, Schlatter C, Gallasz E. Stability of pyrrolizidine alkaloids in hay and silage. *Journal of Agricultural and Food Chemistry*. 1984;32(4):935-937.
12. Stegelmeier BL, Edgar JA, Colegate SM, et al. Pyrrolizidine alkaloid plants, metabolism and toxicity. *Journal of Natural Toxins*. 1999;8(1):95-116.
13. Panter KE, Keeler RF, Baker DC. Toxicoses in livestock from the hemlocks (*Conium* and *Cicuta* spp.). *JAnimSci*. 1988;66(9):2407-2413.
14. Vanderhoff BT, Mosser KH. Jimson weed toxicity: management of anticholinergic plant ingestion. *American Family Physician*. 1992;46(2):526-530.
15. Respondek F, Lallemand A, Julliard V, Bonnair Y. Urinary excretion of dietary contaminants in horses. *Equine Vet J Suppl*. Aug 2006;(36):664-7.
16. Panter KE. Neurotoxicity of the knapweeds (*Centaurea* spp.) in horses. In: James LF, Evans JO, Ralphs MH, Child RD, eds. *Noxious Range Weeds*. Westview Press; 1991:316-324.