POISONOUS PLANTS THAT CONTAMINATE HAY AND FORAGES IN THE WESTERN UNITED STATES[1]

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ABSTRACT AND INTRODUCTION

In the Western United States poisonous plants are estimated to cost the animal industry more than 200 million dollars annually.[1] The majority of these losses are attributed to livestock grazing on rangelands. Little has been done to determine the costs of livestock poisoned by contaminated feeds which are probably more frequent and costly. Grazing livestock generally avoid eating free standing poisonous plants when there are adequate and acceptable alternative forages. However, feeding pressure, palatability and subsequent poisonings are much different when toxic plants contaminate prepared and stored feeds. Alternatively, under some conditions, safe forages can produce and/or accumulate toxins and poison livestock. Although animal deaths and loss of production is costly, there are additional larger economic losses attributed to decreased product quality and loss domestic and international markets. Identifying toxic plants and recognizing when forages may be toxic is essential in avoiding these problems. To better ensure the quality and safety of prepared and stored forages the objectives of this presentation are to: 1) review some basics of toxic plant identification in forages; 2) introduce common toxins and poisonous plants that are likely to contaminate hay and prepared forages; and 3) identify the conditions when feeds are likely to be contaminated.

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. These plants are often weedy in nature, their growth is not uniform, and the subsequent contamination is 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 is often a herculean task as numerous samples may need to be examined to identify the 4 or 5 contaminated bales from a field of hay. Optimally it is best to inspect fields before the forage is harvested and processed as identification of standing toxic plants is relatively easy. Certainly knowing and identifying all potentially toxic plants can be challenging. Local extension agents and veterinarians are good sources as they are often familiar with local plants and problems. Unknown plants may be

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collected and sent to a local laboratory, plant taxonomist or herbarium. Positive identification is more likely if an entire plant is submitted. Flowers and seed pods are often essential for making a definitive identification. If a local herbarium is not available, the sample should be dried and pressed by placing it between newspapers pressed down under a couple of large books for two to three days. The plant can then be taped to a piece of cardboard, or placed in a folder, and mailed to the appropriate laboratory (see Table 1).

Some plants, even within the same species, may or may not be poisonous as the types and concentrations of toxins vary. Such plants might need to be analyzed chemically to confirm they present a risk. For chemical analysis potentially toxic plants can be collected green and frozen. If plant samples cannot be maintained frozen until analysis, many can be dried before shipment. Consult the appropriate laboratory to ensure samples are properly collected, preserved and most likely to be diagnostic (Table 1). Some forages can accumulate toxins such as nitrates or cyanogenic glycosides. These forages should also be appropriately sampled and as they also may not be homogenously distributed throughout the feed. Most laboratories understand this and multiple samples are included in the analysis. As previously mentioned be sure to check with the laboratory to ensure the submission is complete, collected and shipped properly, and that the laboratory will be ready to receive the package (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	http://www.pprl.ars.usda.gov
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, collection of appropriate samples for chemical analysis and microscopic analysis are critical to identify the actual cause (see Tables 1 and 2). As discussed with plant samples submission, expert consultation is needed to make the proper sample collection, preservation and submission. Some samples 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 extensive chemical 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.

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 delievery
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.[2,3]

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.[4] 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.[5] Recently bloat resistant cultivars of 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 been seen when animals are fed high quality hay. Frothy bloat can also been seen in animals fed grain diets. These cases is probably related to both rapid fermentation, acidosis, and microbial production of foam stabilizing compounds.[6]

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-fatal animal may develop secondary septicemia and hepatic abscessation. 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 modulate rumen microflora.[7]

Nitrate-nitrite poisoning: Another relatively common toxicity occurs with certain pasture and cultivated forages accumulate toxic nitrate concentrations. Ruminants are especially sensitive as they quickly reduce nitrates to nitrite which is absorbed and oxidizes hemoglobin producing methemoglobin. Methemoglobin is nonfunctional and 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 1.[8] 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 1: *Kochia (Kochia scoparia) is an annual weed originally introduced from Asia. It is a rapidly growing, branching plant, with alternate hairy leaves. It can grow up to 2 m tall and 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.[8]

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

Pyrrolizidine alkaloid (PA) containing plants: This group of plants is large and distributed worldwide where it includes over 6000 species that may contain over 650 different PAs. In the United States the list is shorter, but PA containing plants can be found in every state. As a group they 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 2: *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 coverd 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 of maternal toxicity.[9] Selected PA containing plants (Table 4), include hound's tongue (*Cynoglossum officinale*- Figure 2), and several different *Senecio* species (See tansy ragwort- *Senecio jacobea*- Figure 3). 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.[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 3: *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.

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

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

Poison hemlock- *Conium maculatum*: Poison hemlock (Figure 4) is an introduced biennial or perennial plant that grows along roads, ditches and fences. It commonly invades 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) affect poisoned animals similarly 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, these 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 4: *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 removed from the source of the toxins and allowed to recover with minimal stress. Supportive care might include a cathartic or administration of activated charcoal to minimize absorption.

Black nightshade *(Solanum nigrum),* jimson weed (*Datura stramonium)* and henbane (*Hyoscyamus niger*): Black nightshade (Figure 5) is found along fences and roads throughout North America. It contains several glycoalkaloids, including solanine, hyoscine and hyoscamine. These toxins are similar to atropine and alter the function of the autonomic nervous system. Consequently they alter 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 test race horses. 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 (hypscyamine, hyoscine and atropine) are similar to those in black nightshade and jimson weed, so cause similar diseases to these plants. Henbane is generally not palatable unless it is included in prepared forages.

Figure 5: *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 6) 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 6: *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 7) are generally used as hay. Other cyanogenic plants such as chokecherry (*Prunus virginiana* Figure 8) and service berry (*Amelanchier alnifolia)* often grow along fences and field margins and can inadvertently be included.

Figure 7: *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 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.

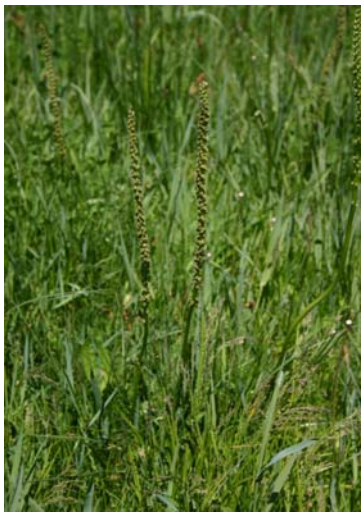

Figure 8: *Arrowgrass (Triglochin maritima) is found throughout North America in moist marshlands and pastures. It is a perennial grass-like plant and the leaves are between 15 and 30 cm long, linear, unjointed and sheathed at the base. The flower is composed of a pediceled raceme that may grow up to 1.5 m tall. The greenish flowers are inconspicuous and ripen into greenish fruitsCyanide 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.

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 9*)* and yellow star thistle (Figure 10*)* 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 substancia nigra and the globus pallidus, resulting in the morphological description of negropallidal encephalomalacia. No other disease or toxin has been shown to have such a site specific effect.

Figure 9: *Russian knapweed (Centaurea repens, right) 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 altenate 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 10: *Yellow star thistle (Centaurea solstitialis, left) 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.

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