WATCH OUT for those Pretty Plants, & Noxious Weeds!

by Beth Johnson, DVM

The Southeast region of North America has an abundance of forage and grasses that provide an excellent nutritional feedstuff for the small ruminant species that consume them. Unfortunately, with the good comes the bad and there are many toxic/poisonous plants that may cause intestinal upset, neurological disease, cardiac signs and even death when consumed. This article will attempt to familiarize the producer with some of the poisonous plants their animals may encounter, as well as, the symptoms and treatments available.

Poisonous plants fall into one of several categories depending on the toxic compounds they contain and the internal organs they affect.

Cyanogenic Glycosides: Many trees in the family Rosaceae (pictured in Figure 1)-(Choke cherry, service berry, peach, apple, wild cherry, etc.) and the Poaceae (Johnson grass, Sudan grass and many other grasses) have the greatest number of important cyanogenic plants affecting animals. These cyanogenic glycosides are capable of causing hydrogen cyanide (HCN), commonly called Prussic acid poisoning, in animals. The quantity of cyanogenic glycosides in plants varies with the stage of growth, time of year, soil mineral and moisture content, and time of day. Two major examples of this are 1) the extremely high in the cyanogenic glycosides and very palatable wilting leaves of the wild cherry tree, and 2) the broad leaf Johnson and Sudan grasses after a hard frost. In general, plant material containing more than 200ppm (20mg/100g of plant) has high potential for poisoning. Johnson or Sudan grass (Sorghum spp.) with levels > 500ppm HCN are highly toxic to ruminants.

Figure 1. Cherry tree

Clinical signs of HCN poisoning in ruminants can begin within minutes to hours of eating large amounts of cyanogenic plants. The onset of signs can be accelerated if the animal drinks water after eating the plants as hydrolysis speeds up the liberation of HCN from the glycosides in the rumen. Sudden death of the animal is often the only observed sign. If observed early enough, apprehension, dyspnea, open mouth breathing, ataxia, frequent urination, and dilated pupils, may be observed prior to death. Any stress on the animal exacerbates the signs and hastens death. The mucous membranes and venous blood may be bright cherry-red in color, but as the animal becomes anoxic, cyanosis is likely to develop.

Treatment involves the intravenous administration of sodium nitrite (10-20mg/kg) and sodium thiosulfate given at the rate 500mg/kg body weight.

Nitrate Poisoning

Many plants including barley, wheat, rye, corn, sorghum and Sudan grasses, and common annual weeds such as pig weed and Russian thistle can all accumulate toxic levels of nitrates. Application of nitrate fertilizers or drainage from fertilized fields and contaminated water sources can also be a source for nitrate. Drought conditions also promote accumulation of nitrate in plants. Nitrate itself is not overly toxic, but when the rumen microflora convert it to nitrite it becomes highly toxic.

Cattle, sheep and goats are the species most commonly affected by nitrate poisoning, with the fetus in utero being highly susceptible. Nitrite ions rapidly oxidize hemoglobin in red blood cells, forming methemoglobin, which cannot transport oxygen. When over 40% of hemoglobin is oxidised to methemoglobin, clinical signs of poisoning develop. Death occurs as methemoglobin levels reach 80%. Sudden deaths are common but if observed early enough sheep and goats may exhibit a rapid weak pulse, muscle tremors, tachypnea, brown mucous membranes, excessive salivation, staggering gait, disorientation, and frequent urination prior to coma and death.

Cold water with added oral broad-spectrum antibiotics can help decrease nitrate reduction to nitrite by rumen microorganisms. Diluted vinegar given orally via stomach tube has similar beneficial effects. Methylene blue has been used as a treatment but availability of this

Figure 2. Oleander bush

Figure 3. Taxus spp. "Japanese Yew"

Figure 4. Water Hemlock plant

product has become very difficult in recent years and is not recommended in food producing animals.

As a general rule, levels of nitrate over 0.5% in forages and water levels exceeding 200 ppm are potentially hazardous to pregnant animals especially if fed continuously. Forages containing in excess of 1% nitrate dry matter should be considered toxic. A nitrate level of the forage should be checked if there is suspicion that it is high in nitrates prior to feeding hay, silage, etc.

CARDIOTOXIC PLANTS

Oleander (*Nerium oleander*): Figure 2

Livestock are usually poisoned when they browse on oleander or when trimmings are carelessly thrown into animal enclosures. Potent cardiac glycosides are present in all parts of the plant. Oleander leaves remain toxic when dry. Cardiac dysrhythmias and heart block may be observed prior to death.

Yew (*Taxus* spp.): Figure 3

This ornamental plant is very common as a landscaping shrub due to its durability and evergreen characteristics. Yews contain a group of 10 or more toxic alkaloids, referred to as taxines. Taxine inhibits normal sodium and calcium exchange across the myocardial cells, preventing depolarization and causing arrhythmias. All parts of the plant, green or dried, are toxic. Livestock are frequently poisoned when fed clippings from cultivated yews!!!! The highest concentration of the alkaloids is generally found in the leaves in winter time. Adult cattle and horses have been fatally poisoned with as little as 8-16 oz of yew leaves or 0.1 to 0.5% of their body weight so you can just imagine how little is required to kill a sheep or goat.

Milkweeds (*Asclepias* spp.):

Many species of milkweed are found in North America, the most poisonous of which are those species with narrow, grass-like leaves. The principle toxins are cardenolides with digitalis-like properties. In addition to the cardiotoxic effects of the cardenolides common to most milkweeds, other glycosides and resinoids identified in milkweeds have direct effects on the respiratory, digestive, and nervous systems causing dyspnea, colic and diarrhea, muscle tremors, seizures, and head pressing. Milkweeds remain toxic when dry in hay which is extremely important as it is often present in pastures especially as a clump of plants in low lying areas which is incorporated in the hay.

The Monarch butterfly requires the milkweed in its life cycle to develop; therefore, there has been a recent push to encourage cultivation of this plant due to an indication that this butterfly has reduced numbers since this weed has been removed from pastures. I would encourage cultivation of this weed in a flower garden instead of pasture setting for several reasons mentioned above.

Water Hemlock (*Cicuta* spp.): Figure 4.

Native to North America, water hemlock is one of the most poisonous plants to all animals. All parts of the plant and especially the roots contain Cicutoxin that is rapidly absorbed from mucous membranes

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and acts on the central nervous system to produce rapid onset of ataxia, convulsive seizures and lateral recumbency, dilated pupils and death from respiratory paralysis. Animals consuming a sublethal dose, will recover if not stressed. There is no specific antidote but one may try a cathartic such as activated charcoal and supportive care.

Mountain Laurel, Rhododendron: (Figure 5 & 6)

There are many species of laurels and most are considered poisonous. The toxic principle is called andromedotoxin. Some of the laurels also contain a glucoside of hydroquinone. Poisoning can occur at any time of the year but is more commonly seen in the early spring or in wintertime when snow covers other vegetation. Sheep, goats, and cattle are commonly affected by grazing all portions of the plant, but particularly the leaves.

Symptoms include vomiting, bloating, salivation and abdominal pain as evidenced by straining. Eventually the animals become weak, stagger and become prostrate. Occasionally, pneumonia is present due to inhalation of rumen contents into the lungs during vomiting. It is important to provide oral support through use of a stomach tube due to the risk of inhalation which may occur if oral drenching is performed. Cathartics such as activated charcoal, mineral oil, etc. can be administered.

Oak (*Quercus* spp.):

All species of oak have the potential to poison animals, especially those eating large quantities of the young leaves. The principal toxins are gallotannins, found in the leaves, bark, and acorns of oaks. Tannic acid is an astringent causing necrosis of the intestinal mucosa and renal tubules. Goats and wild ruminants are apparently better able to detoxify tannic acid than other livestock because they have a tannin-binding protein in their saliva that neutralizes tannic acid.

Clinically, animals become depressed, anorexic and develop intestinal stasis. Excessive thirst and frequent urination may be observed. The feces are hard and dark initially, but a black tarry diarrhea often occurs later in the course of poisoning. Teeth grinding and a hunched back are often indicative of abdominal pain. Severe liver and kidney damage is detectable by marked elevations in serum liver enzymes, creatinine, and urea nitrogen. Icterus, red-colored urine, and dehydration are further signs of oak poisoning. Treatment is supportive to provide hydration.

Figure 5. Mountain Laurel in bloom

Figure 6. Rhododendron plant

Figure 7. Bracken Fern plant

Bracken Fern: (Figure 7)

The poisonous principle is the enzyme thiaminase which inactivates thiamine (Vitamin B1) in the horse and ruminants. In ruminants, an aplastic-anemia factor causes depression of the bone marrow. Sheep and goats are less susceptible to the toxic effects than cattle and horses.

All portions of the plant are toxic whether green or dry. Poisoning by the plant is cumulative and symptoms may not appear until several weeks or months later. Clinical cases are most often seen in the spring or late summer or fall, especially after periods of drought when other forage is short or not available. Animals have shown toxicity from consuming hay containing the dried plants.

Horses exhibit incoordination, often standing with their legs spread apart as if bracing themselves. The affected animal arches its back and neck into a crouching stance. Occasionally a fever is present up to 104°F. Prior to death horses may "head press" objects and have spasms with the head and neck drawn backwards. Cattle may exhibit two types of symptoms. The laryngeal form is often seen in younger animals and is characterized by edema of the throat region resulting in difficult and loud breathing. The enteric form may be preceded by the laryngeal form. Affected animals exhibit bloody feces, blood in the urine and excessive bleeding from fly bites. The blood is slow to clot since there is a deficiency of platelets. Death usually occurs within a few days after symptoms appear. Sheep have shown blindness due to degeneration of the retinal epithelial cells after grazing bracken fern.

Perilla Mint (Figure 8)

This toxic plant contains "perilla ketone" that is known to produce pulmonary edema and pleural effusion in a variety of animals but most often in cattle and horses. Usually seen in the late summer or fall.

Affected animals exhibit respiratory distress especially during exhaling and may even grunt during exhaling. During auscultation of the thorax, friction sounds are very common. A nasal discharge as well as an elevated temperature may also be present. Post mortem examination reveals pulmonary emphysema and edema with evidence of the plant and its seeds in the rumen.

Figure 8. Perilla Mint plant

Figure 9. Buttercup plant

TERATOGENIC PLANTS

Plants that are teratogenic and capable of causing abortions include lupines, locoweeds, tobacco, poison hemlock, rhododendrons and western false hellebore or skunk cabbage. Does may abort hairless kids with pronounced goiter after eating tansy mustard (*Descurainia pinnata)* in late pregnancy. Most of these plants are found out west, but in the Southeast the hemlocks and rhododendrons are the primary teratogenic plants.

Poison or Spotted Hemlock (*Conium maculatum*):

Originally introduced from Europe, poison hemlock has become a widely distributed noxious weed in North America. Eight piperidine alkaloids have been found in various parts of the plant. The mechanism of action of these alkaloids is complex effectively blocking spinal cord reflexes. Muscle tremors are followed by neuromuscular blockade and paralysis. Cyanosis, respiratory paralysis, and coma without convulsions precede death. Goats may recover from hemlock poisoning only to develop a strong craving for the plant, which ultimately proves fatal. Pregnant animals that survive the acute toxicity may abort. Lambs born to ewes fed poison hemlock in the 30 – 60th days of gestation develop excessive carpal joint flexure and lateral deviation.

HEPATOTOXIC PLANTS/ PHOTOSENSITIZATION

Several poisonous plants contain pyrrolizidine alkaloids which are absorbed from the digestive tract, are activated in the liver and cause damage to hepatocytes resulting in photosensitization, especially in horses. Photophobia, excessive tearing, and swelling and redness of non-pigmented skin, develop initially before the affected skin becomes necrotic and sloughs. White breeds of sheep often only develop lesions on the ears and face because of the protective fleece covering unless they have been recently sheered. Prior to the development of secondary photosensitization, liver enzymes are elevated, and when the liver is severely affected signs of hepatic encephalopathy may develop.

Cocklebur (*Xanthium strumarium*):

As common invasive weeds, cockleburs are poisonous to animals owing to the presence of the potent hepatoxin Carboxyactractyloside. The glycoside is present in high concentration in the seeds and the two-leafed cotyledon stage, but declines by the four-leaf stage and is absent in the mature plant. Acute diffuse central-lobular and paracentral coagulative necrosis of the liver are typical of cocklebur poisoning.

Buttercup: (figure 9)

A non-native invasive weed which can be found in many pastures during the spring. Buttercups (*Ranunculus* spp.) contain significant quantities of the irritating compound ranunculin and oily glycosides. Clinical signs of buttercup poisoning may be mild burning of the mouth, abdominal pain, vomiting and bloody diarrhea progressing to weakness, depression, labored breathing, and anorexia. Gross necropsy findings include inflammation and edema of the rumen, congestion of the lungs, liver, and kidneys; excessive fluid in the thoracic and abdominal cavities, and hemorrhages in the left ventricle of the heart.

Other common "weeds" or shrubs that cause toxicities include Pokeweed, yellow jasmine, boxwood, jimsonweed, nightshade, elderberry, crotalaria, coffee weed, among others. Among these more common poisonous plants there are many ornamental plants that are toxic. With the increase in landscaping around houses and farms, many of these are available to sheep and goats that escape their enclosure.

As you read this article, it makes you wonder how we are able to keep any of our sheep and goats alive. Fortunately, most animals have an innate ability to avoid the toxic plants. When food gets scarce; however, many of these plants are consumed. Take home message: provide adequate nutrition and hopefully you will never experience a toxicity.

Some excellent resources identifying poisonous plants can be found at:

https://www.famu.edu/cesta/main/assets/File/coop_extension/small%20ruminant/goat%20pubs/Poisonous_Plants_to_Livestock_Part_B.pdf

https://avmaspeakers.eventkaddy.net/event_data/28/session_files_published/2014_16103.pdf

https://carteret.ces.ncsu.edu/wp-content/uploads/2013/05/Poisonour-Plants-of-the-Southern-United-States.pdf?fwd=no

Beth Johnson, DVM , *is a Staff Veterinarian in the Kentucky Department of Agriculture and has 40 years of experience raising and treating small ruminants. Her family farms in Parksville, KY where she raises Gelbvieh cattle and Boer goats.*