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Plants containing urinary tract, gastrointestinal, or miscellaneous toxins that affect livestock

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Key Points:

- Plants that affect the urinary tract and kidneys include oxalate containing plants, oaks, plants that cause hemolysis, calcinogenic plants, pigweed and others.
- Many plants affect gastrointestinal tract even though their other prominent lesions results in their discussion in other systems.
- Buttercup, nightshade, mustard, bindweed and castor bean are plants that produce gastrointestinal lesions.
- Miscellaneous plants such as cynogenic plants, and bracken fern also commonly poison livestock.

Synopsis:

Whether exposed by grazing toxic range or pasture plants or by eating contaminated feed there are a number of plant toxins that produce urinary tract disease, gastroenteritis, and other miscellaneous or multi-systemic diseases. As with many toxic plant poisonings, diagnosis can be challenging. The best diagnosis requires incorporation of field studies, clinical signs, gross and microscopic pathology as well as chemical identification of plants, toxins and metabolites in animal samples. The objectives of this review are to briefly introduce selected poisonous plants that commonly poison livestock in North America; describe the clinical and pathologic lesions they produce in livestock; and present current technology available to identify poisoning, treat affected animals, and minimize or avoid poisoning additional animals.

Urinary system toxins or nephrotoxic plants:

Oxalate Containing Plants: Many commonly used plants contain soluble or insoluble oxalates. The insoluble oxalates such as calcium oxalate are present as crystals in plants. These crystals are very irritating to mucosal membranes and when eaten damage the oral cavity and gastrointestinal tract. Such plants are not palatable and generally avoided; however, if animals are forced to eat them, they develop oral mucosal hyperemia, swelling and marked hypersalivation. Plant soluble oxalates include sodium and potassium oxalate and oxalic acid. These are quickly absorbed and if plant concentrations are high, they cause systemic poisoning. Though many plants contain some oxalates, only a few species of the *Agave*, *Beta*, *Bassia*, *Chenopodium*, *Halogeton* (Figure 1), *Oxalis*, *Rhuem*, *Rumex*, *Sarcobatus*, and *Setaria* genera contain enough to be reported as toxic. The type of poisoning depends both on the animal species poisoned and plant oxalate concentrations. As ruminants adapt and metabolize oxalates, they are much less susceptible to chronic poisoning. However, all animals can be fatally poisoned if they ingest too much too quickly. Horses and other monogastric animals are more likely to develop chronic poisoning (low oxalate doses for extended durations). Plants with >10% soluble oxalates are usually nephrotoxic causing fatal renal failure. Lower soluble oxalate concentrations can produce secondary hyperparathyroidism, resorption and loss of bone with secondary osseous proliferation and dysplasia. This is because soluble oxalates effectively bind and sequester calcium and magnesium. This can result in functional deficiencies including altered neurologic function.[1] As calcium supplementation does not alter acute poisoning and nephrosis, additional mechanisms of toxicity were found to contribute to nephrosis. An oxalate metabolite, calcium oxalate monohydrate (COM) has been shown to damage mitochondrial function- impairing oxidative phosphoralyation.[2] COM crystals also alter membrane structure and function resulting in physical damage as well as increased reactive oxygen species that further damage cells.[3] Additionally oxalate poisoning reduces activities of tricarboxylic acid cycle enzymes (succinate dehydrogenase, isocitrate dehydrogenase, malate dehydrogenase and others). This combined with increased oxygen stress (decreased antioxidant enzymes and glutathione with increased reactive oxygen species and lipid peroxidation) certainly contribute to disease. The impact and contribution of these different toxic mechanisms is unknown.[4,5]

Acute oxalate poisoning is characterized by hypocalcemia, lethargy, anorexia, muscle tremors, weakness, stiffness, diarrhea, ataxia, tachypnea, dyspnea, tetany, recumbency, rumen atony, coma and death. Animals that are forced to move may develop hyperesthesia and seizures. Animals that survive these early changes develop azotemia and if severe they will develop clinical renal failure and potentially encephalopathy. Gross lesions in most animals are minimal and characterized by gastritis or rumenitis with serosal edema and hemorrhage. Secondary

accumulation of pleural fluids and ascites, as well as pulmonary edema may also occur. The kidneys are often pale and swollen. In chronic poisoning the kidneys often become pale, firm, and shrunken.[5] Histologic lesions are associated with deposition of birefringent COM crystals in the renal tubules, abomasal mucosa, and gastric vasculature. There is degeneration and necrosis of the tubular epithelium of the proximal convoluted tubules. Affected tubules are typically distended with crystalline material and have flattened and degenerative cortical tubular epithelium in acute cases (Figure 2). Animals that survive for several days will have gross changes of thinning of the renal cortex with discoloration between the cortex and medulla. The discoloration results from the accumulation of crystalline materials in the tubules.

Chronically poisoned animals, especially horses, show weakness, stiffness, intermittent lameness, inability to work, weight loss, and swelling of the osseous structures of the head. Grossly and histologically they develop fibrous osteodystrophy with swelling of the nasal bones, maxilla, and mandible (Figure 3). Associated histologic changes include increased osteoclast activity, dystrophic mineralization with proliferation of fibrous connective tissue.[6]

Oxalate poisoning is best diagnosed by associating histologic COM crystals with the clinical signs, and field evidence of exposure and consumption of oxalate containing plants. Treatment has been largely unsuccessful, though oral calcium therapy may also be useful in binding soluble oxalate in the rumen to prevent further absorption. Certainly, allowing ruminants to adapt with limited exposures to produce a low dose exposures to soluble oxalates for 8 to 25 days will decrease the risk of poisoning.[5] Additionally risk assessment can be facilitated by analyzing the oxalate concentrations of potentially toxic plant populations.

Oaks: (*Quercus* spp.) are common throughout the world and they include hundreds of species of shrubs and trees. All species are considered toxic with seedlings, early bud growth, and acorns being highly toxic and the cause of most intoxications. Poisoning generally occurs in early spring or fall when toxic acorns or early growth are available and alternative forages are limited. Toxicity has been attributed to tannins; however, purified tannins are only toxic at relatively high doses, suggesting there is probably an unidentified toxic component. Tannins are polyphenolic compounds that cross-link proteins and other macromolecules resulting in cellular degeneration and necrosis.[7] Poisoning occurs when large doses are ingested over several days to weeks. Primarily a ruminant toxin, calves are highly susceptible and goats seem to be able to metabolize tannins and are less susceptible to poisoning.[8,9] Though oaks predominantly cause gastrointestinal mucosal damage and nephrosis, at some doses and durations they can cause liver necrosis. Initial signs of poisoning include lethargy, constipation, tenesmus, polydypsia, polyuria, and a brown discoloration to the urine. These are followed by hemorrhagic diarrhea, abdominal pain, rumen atony and anorexia. Oak induced renal disease is characterized by isosthenuria, glucosuria, proteinuria and hematuria with serum biochemical changes of hyperkalemia, hyperphospatemia, azotemia and hypercreatinemia. Gross lesions include oak material/acorns in the rumen contents, subcutaneous edema, perirenal edema, mesenteric edema, ascites, hydrothorax, and ulcerative hemorrhagic rumenitis, gastritis, and enteritis. Microscopic lesions are characterized by diffuse cortical renal tubular degeneration and necrosis with tubular casts (Figure 4). If present, hepatic changes include hepatocellular degeneration and necrosis with focally extensive hemorrhage.

A most likely diagnosis of poisoning is made by associating the clinical presentation, pathologic findings, and evidence of ingestion. Chemical detection of tannin metabolites has limited use as they are readily eliminated and often undetectable in tissues of poisoned animals. Poisoning can be prevented by limiting intake of oak materials to less than 50% of the diet. Treatment of poisoned animals is generally symptomatic and supportive for the gastric and renal damage.

Hemoglobin-induced nephrosis: *Acer rubrum* (red maple) causes severe hemolysis and nephrosis in horses. Toxicity in other livestock has not been reported. In horses, zebras and donkeys small doses of less than 1.5 gm leaf/kg BW are reported to be poisonous. The wilted and shed leaves are most toxic. Although only red maple has been associated with poisoning in horses, red-maple hybrids should be considered toxic until proven otherwise. Most poisoning occurs in the fall, after storms cause branches to fall into paddocks or when trimmings are fed to animals. Though the cause has not been identified, oxidative hemolysis usually develops within 24 hours of exposure. Clinically this is seen as anorexia, depression, intravascular and extravascular hemolysis, icterus, anemia, hemoglobinuria, respiratory distress, Heinz body anemia, colic, laminitis, coma and death. Post-mortem findings include icterus, enlarged and swollen spleen, liver and kidneys and they often have red urine. Treatment is symptomatic and includes blood transfusions, fluid replacement and oxygen therapy. Antioxidants such as ascorbic acid may also be helpful.

Allium spp. (onions) contain an oxidant (N-propyl disulfide) that causes acute hemolytic anemia. Because only large amounts of onion contain enough toxin, it rarely poisons livestock.

Amaranthus spp. (pigweeds): The *Amaranthus* genus includes over 60 species and hybrids that have global distribution. These are annual weeds with prolific seed production that allows them to spread and dominate paddocks, fields and disturbed areas along fences, ditches and roads. Though all can accumulate nitrates, only *A. retroflexus* (red pigweed) and several additional species have been reported to be nephrotoxic and cardiotoxic in livestock. [7,10-12] Clinical poisoning appears to require plant ingestion for several days to several weeks. Additionally the signs of poisoning can be delayed 5 to 10 days after exposure, suggesting the elimination of these toxins is slow. The clinical signs of nitrate associated poisoning include sudden onset of tachypnea, weakness, and recumbency. The syndrome progresses rapidly to death or to a full recovery within 24 hours or less. Pathologic lesions are minimal or absent, but can be of darkened, brownish blood in tissues. Pigweed associated renal disease requires extended ingestion, but once initiated, clinical poisoning quickly progresses in 1 to 2 days from weakness, muscle tremors, ataxia, knuckling of pasterns to recumbence, paralysis, hemorrhagic diarrhea, hemorrhages, coma, and death. Serum biochemistry changes include increases in potassium, phosphorous, blood urea nitrogen, and creatinine. Gross histologic lesions are predominantly of fluid accumulation with straw-colored fluid in the abdominal and thoracic cavity with pale, potentially swollen kidneys, and prominent perirenal edema. Microscopically there is marked degeneration and necrosis of the convoluted renal tubular epithelium with interstitial edema. Many renal tubules are dilated and contain proteinaceous debris and casts. The disease in surviving animals often progresses into interstitial renal fibrosis.[7,12-15] The cardiotoxic effects usually affect pigs resulting in sudden death. Histologically these animals have localized areas

of myocardial hemorrhage, necrosis with subsequent fibrosis and scarring. Many other tissues may be subsequently congested and edematous with occasional effusions.[16,17]

Identifying poisoning is made by linking exposure and ingestion with the clinical signs and lesions. Nitrate poisoning can be confirmed by monitoring serum methemoglobinemia, analyzing serum or ocular fluid for nitrates, analyzing forages for nitrates and correlating these with the clinical disease. Identifying nephrotoxic or cardiotoxic diseases is more challenging as these lesions are correlated with field studies that verify ingestion and clinical presentation. As treatment has had no overall effect on survival, prevention by avoiding exposure is recommended. As animals with pigweed-induced renal disease that survive are likely to have diminished renal function, the prognosis is guarded.

Calcinogenic glycoside containing plants**:** *Solanum malacoxylon, S. verbascifolim, S. torvum, Nierembergia veitchii, Trisetum flavescens, and Cestrum diurnum* contain glycosides of 1,25-dihydroxycholecalciferol (calcitriol) or physiologically similar compounds that act as active vitamin D (cholecalciferol). This results in hypercalcemia and calcification of many tissues and organs. Cholecalciferol increases calcium (Ca) absorption in the intestinal tract; increases Ca resorption from bone; and decreases renal Ca excretion resulting in marked hypercalcemia and hyperphosphatemia. This results in hyperostosis and eventually metastatic calcification.

Clinical poisoning is first seen as depression, weakness, weight loss, infertility, anorexia, cardiac arrhythmias and impaired stilted gait, lameness, recumbence and death. Biochemically there is both hypercalcemia and hyperphosphatemia and with progressing renal disease increases in BUN, creatinine and phosphorus. At necropsy mineralization seen as gritty, white deposits are often seen in kidneys, intestines, stomach, heart, lungs, arteries, bones, tendons and ligaments. Renal lesions include mineralization of the renal tubular basement membranes, glomerular tufts, and Bowman's capsule. Mineralization may involve many other tissues such as bronchioles, alveoli, endocardium, vessel walls and walls of the intestine and stomach. Secondary changes include hyperplastic thyroid cells and atrophy of the parathyroid.

Mineralization is generally not reversible and many tissues may remain mineralized for years. Mineralization in the walls of the aorta and tendons are especially persistent.[18-20] Consequently avoiding exposure is essential. Most of these calcinogenic plants are toxic when grazed and they are also toxic when included in green forage; however, their calcinogenic potential decreases when they are stored for extended periods in dried feeds.

Plants that contain Gastrointestinal Toxins:

Mechanical damage (foxtail, bristlegrass, sandbur, cheatgrass, etc): These are poor-quality forages and their awns often embed in the mucous membranes of the gums and tongue, causing ulcers and abscesses. Affected animals salivate excessively and they may have difficulty eating. Awns may also migrate deep into tissues producing infection, abscesses and loss of function. As many of these are invasive species, they often displace good forage and also contaminate harvested feeds.

Ranunculaceae (buttercup family): Buttercups often contain ranunculin that is quickly converted to a potent mucosal irritant, protoanemonin. Clinical signs of poisoning include blistered lips, stomatitis, gastroenteritis, increased salivation, abdominal pain and diarrhea. Most poisonings occur in sheep because buttercup is not very palatable to other livestock. Dried plants appear to be non-toxic.

Nightshades *Solanum rostratum* (buffalo bur), *S. ptycanthum* (black nightshade), *S. dulcamara* (bittersweet), *S. elaeagnifolium* (silverleaf nightshade), *S. carolinense* (Carolina horse nettle), *S. dimidiatum* (western horse nettle), *S. triflorum* (cutleaf nightshade): The nightshades are a diverse group of toxic plants resulting in several poisoning syndromes. Some steroidal glycoalkaloids cause severe gastroenteritis. Others contain cholinesterase inhibitors that cause neurologic disease. The most common toxin is an alkaloid named solanine that is a potent mucosal irritant resulting in severe gastroenteritis (Figure 5). Solanine concentrations are highest in the berries that often poison both livestock and humans. Poisoned animals often develop anorexia, increased salivation and slobbering, abdominal pain, diarrhea, dilation of pupils, dullness, depression, weakness, progressive paralysis, prostration and rarely death. Treatment generally is symptomatic and most animals quickly recover when exposure is discontinued.[7]

Ricinus communis (castor bean) is an ornamental that in many places has become a weed. The seeds are very toxic and they often contaminate feeds and food. The toxin, ricin, inhibits protein synthesis (by inhibiting ribosomal function) and it can cause severe immunologic disease with anaphylaxis. Poisoning develops within 12 to 48 hours after ingestion as animals develop a dull appearance, depression, anorexia, thirst, weakness, colic, trembling, sweating, incoordination, difficult breathing, progressive CNS depression, fever, bloody diarrhea, convulsions and death. Most recognized poisonings are fatal and treatment for those identified early is primarily directed to reduce absorption. Activated charcoal and cathartics may be likely choices for livestock.[21,22]

Convolvulus arvensis (field bindweed, morning glory) is an invasive, noxious weed that invades and often dominates many pastures, paddocks and disturbed areas. Poisoning appears to exclusive for horses as there are no reports of toxicity in other species. Horses grazing extensively on bindweed may develop diarrhea, colic, gastrointestinal ulceration and intestinal thickening and fibrosis. The resinoid convolvulin and several tropane alkaloids have been suggested as the toxins, but this has not been confirmed experimentally.[23] More work is needed to better understand this poisoning, to determine the pathogenesis, and to identify methods to avoid poisoning.

Brassica spp (white mustard, yellow mustard, wild mustard, charlock, black mustard, Indian mustard); *Erysemum cheiranthoide* (wormseed mustard); *Raphanus raphanastrum* (*w*ild radish); *Thlaspi arvense* (fanweed, field pennycrest); *Barbarea vulgari* (yellow rocket, wintercrest) and others: Most mustards accumulate nitrates which accounts for most of their toxicity. However, several additional toxins have been proposed and various syndromes have been associated with mustard ingestion. However, the only proven toxicities are related to

nitrates and isothiocyanates (goitrogenic). The remaining poisoning syndromes are variable with diverse clinical signs and lesions. Those relating to this section are variable, but many are associated sporadic diseases such as wooden tongue and digestive-tract irritation.[24,25] These syndromes are poorly described and their intermittent occurrence has made identifying the cause and character of poisoning difficult. In most cases animals recover if they are removed from the source.

Miscellaneous Toxic Plants:

Cyanogenic Plants: Thousands of plants, including those used for food and feeds, contain cyanogenic glycosides. However, those associated with poisoning include many types of cherries (*Prunus* spp. Figure 6), elderberry (*Sambucus* spp.), service berry (*Amelanchier alnifolia),* various sorghum, Johnson and Sudan grasses (*Sorghum* spp.), corn (*Zea* spp.), vetches (*Vacia sativa),* white clover (*Trifolium repens),* birdsfoot trefoil (*Lotus* spp.), and arrowgrass (*Triglochin* spp.). Sequestered in plants as glycosides such as amygdalin, prunasin, and lucumin, that are hydrolyzed producing toxic cyanide or prussic acid. Hydrolysis primarily occurs when the plant is damaged by crushing, chewing, freezing, or wilting. The cyanide slowly degrades with drying or ensiling. The concentration of cyanogenic glycosides is highest in young rapidly growing plants and often increases with stresses such as frost, drought, or herbicide treatment.[26,27] Cyanide displaces oxygen by avidly binding with iron in cellular cytochrome oxidase. This inhibits cellular respiration. Unused oxygen accumulates as oxyhemoglobin making blood and tissues of poisoned animals "cherry red." Low, non-lethal doses of cyanide have also been associated with goiter, spinal cord degeneration and cystitis. It has been suggested these changes are due to neurologic myelin damage.[28]

Clinical poisoning is characterized by hyperventilation, dyspnea, anxiousness, hypotension and staggering followed by convulsions, paralysis and death. The blood and tissues may be bright red immediately after death, but this darkens between 2 and 6 hours post mortem. Petechial hemorrhages may also be found in the abomasum, endocardium and epicardium (probably due to stress and agonal struggling). Poisoned animals may also have an almond smell, but this dissipates rapidly post mortem. Diagnosis is made by associating the clinical signs and lesions with evidence of plant consumption. As cyanide quickly dissipates from tissues after death, tissues such as liver, muscle and rumen contents must be collected within a couple of hours , frozen in sealed, air- tight containers and quickly analyzed. Intravenous sodium nitrite and sodium thiosulfate oxidize hemoglobin forming methemoglobin. As methemoglobin binds cyanide protecting the cytochrome oxidase system from cyanide this has been used as a treatment. Of course the best treatment is to avoid harvesting and feeding these plants when they are likely to be toxic. As some plants are only sporadically toxic, risk assessments can be made by testing potentially toxic feeds for their cyanogenic potential.[26]

Bracken Fern (*Pteridium aquilinum* Figure 7) has subspecies and varieties throughout the world. They grow in semi-shaded, well drained fields and rangelands. They are perennials and though they are prolific spore producers, they spread primarily through a dense rhizome network. In disturbed areas that have been burned or disturbed, bracken fern expands and dominates the entire plant community. Animals are poisoned when they eat bracken fern while grazing and when it contaminates feeds.[29] Bracken fern toxins include cyanogenic glycosides, thiaminases, steroidal

and radiomimetic toxins, and carcinogens. Some have been characterized, but still others have only been suggested. Of these the best described is ptaquiloside, a norsequiterpene glucoside that is mutagenic, clastogenic and carcinogenic. It damages rapidly dividing cells in the bone marrow and gastrointestinal tract. It is also a potent carcinogens that produces esophageal, gastric and urinary tract neoplasms.[30] Ptaquiloside concentrations are highest in the vegetative plant parts as the rhizomes, roots and spores contain very little. Several thiaminases have also been isolated from bracken fern.[31] Thiaminase concentrations are highest in the rhizomes. Prunasin, a cyanogenic glycoside, has also been identified in bracken fern.[32] It has been suggested that chronic cyanide poisoning may account for equine neurologic disease related to bracken fern poisoning. Several syndromes have been associated with bracken fern poisoning. These include acute hemorrhagic disease, bovine enzootic hematuria, bright blindness, upper alimentary carcinomas and thiamine deficiency. Current evidence suggests these different toxicologic presentations are dependent on dose, duration and species intoxicated.

Acute hemorrhagic disease and enzootic hematuria is common ruminant syndrome with a continuum of presentations varying from hemorrhagic disease, chronic intermittent hematuria with anemia and thrombocytopenia. Hemorrhagic disease most often occurs during late summer when other feed is scarce, or when animals are fed hay containing bracken fern. Poisoning livestock must ingest bracken fern for several weeks to years before they become weak and rapidly lose weight. This is followed by fever, dyspnea and anemia. Hemorrhages develop next ranging from minor mucosal petechia to effusive bleeding that is often seen as large blood clots in the feces. Coagulation times are prolonged and bleeding is extensive even from small wounds such as small insect bites or other minor scratches. Once animals develop clinical disease, poisoning is almost always fatal. Post mortem examinations usually reveal multiple serosal and soft tissue hemorrhages and bruises, intestinal hemorrhage, hematuria with hemorrhagic uroepithelim in the bladder and urethra. Later the uroepithelium may also contain numerous vascular, fibrous, or epithelial neoplasms. Though less common, neoplasms in the upper gastrointestinal tract have also been reported. Enzootic hematuria is characterized by intermittent urinary hemorrhage that is due to extensive epithelial and mesenchymal neoplastic transformation of the urinary tract. The neoplastic lesions are of mixed origin as they include vascular, mesenchymal or epithelial differentiations. The adjacent urinary epithelium is often ulcerated and there are large submucosal hemorrhages. In most cases, mixtures of hemorrhagic and neoplastic lesions are present making separation of these syndromes artificial. Both the hemorrhagic syndrome and uroepithelial neoplasms have been reproduced experimentally with bracken fern and ptaquiloside.[33] The clinical presentation is dose related as high doses of short duration produce acute poisonings seen as hemorrhagic disease. This is due to ptaquiloside's cytotoxic effects on proliferating bone marrow stem cells. Microscopically this is seen as depletion of bone marrow megakaryocytes followed by panhypoplasia. The leukogram often has a mixed response. In the initial phase there is a pronounced monocytosis that is followed by granulocytopenia and thrombocytopenia. Final phases include marked thrombocytopenia with anemia, leucopenia, and hypergammaglobulinemia. Urinalysis generally includes hematuria and proteinuria. Affected animals have both an increased susceptibility to infection and a tendency for spontaneous hemorrhage.[34] Ptaquiloside is excreted in the urine and milk of poisoned animals.[35] Ptaquiloside neoplastic transformation may be enhanced by bovine papilloma virus infection; however, this is probably secondary as bracken fern-associated myelodysplasia and subsequent immunosuppression probably promote papilloma virus infection.[36]

Bright blindness is another bracken fern syndrome that may be a less common component of ptaquiloside toxicity characterized by progressive retinal degeneration of sheep. There is marked degeneration and thinning of the retina that is seen as tapetal hyper reflectivity. Affected sheep may be partially visual and in efforts to see they adopt a characteristic wide-eyed or alert attitude. The pupils respond poorly to light. Ophthalmoscopic findings are those of retinal degeneration characterized by a hyper reflective fundus with narrowing of arteries and veins and pale tapetum nigrum with patchy spots of gray. Histologically the lesion is seen as severe atrophy of the retinal rods, cones, and outer nuclear layer that is most pronounced in the tapetal portion of the retina. Affected animals may also have many of the other bracken fern-associated lesions including bone marrow suppression, hemorrhage, immunosuppression and urinary tract neoplasia. Retinal degeneration has also been experimentally reproduced in sheep using both powdered bracken fern and purified ptaquiloside.[37,38]

Bracken staggers or neurologic disease is the bracken fern poisoning syndrome that is described in monogastric animals. It was first recognized as a neurologic disease when horses consumed contaminated hay. Subsequent studies showed that horses fed diets of 20-25% bracken fern for 3 or more months developed the characteristic staggers. The clinical presentation of bracken staggers in horses includes anorexia, weight loss, incoordination, and a crouching stance while arching the back and neck with splayed feet. When forced to move, affected animals tremble and if they are severely poisoned, horses develop tachycardia and arrhythmias. These signs may progress to convulsions and opisthotonos and such severely affected animal generally die. As this disease is similar to vitamin B_1 deficiency and most animals respond with thiamine therapy, the pathogenesis of these changes has been attributed to bracken fern thiaminases and anti-thiamine factors. Perhaps partially due to availability and feeding practices, horses seem to be particularly susceptible to bracken fern induced neurologic disease.[29,39] The disease is rare in pigs and the signs are less distinct including anorexia, weight loss and sudden onset of dyspnea, recumbency, and death. In ruminants thiamine deficiency is generally associated with polioencephalomalacia which is not a common finding in ruminant bracken fern poisoning; however, impaired thiamine metabolism in sheep has been associated with consumption of bracken fern and rock or mulga fern (*Cheilanthes sieberi*) in Australia.[40]

As bracken fern poisoning, other than thiamine deficiency, is essentially untreatable; poisoning is most easily controlled by preventing exposure. Bracken fern is usually grazed for want of alternative forages. Avoiding exposure by improving pasture management and increasing the production of alternative forage is essential. Recent work has found that some bracken populations contain very low or no ptaquiloside. More work is needed to identify these populations, determine why they are not toxic and use this information to predict or reduce toxicity. As with most toxic plants the initial step should be to remove poisoned animals from bracken fern containing pastures. Treatment of thiamine deficiency in horses is effective if the diagnosis is made early. Thiamine therapy should include animals similarly exposed but not yet showing signs, as they may develop disease days or weeks after removal from the source of bracken. Antibiotics may be useful to prevent secondary infections. Blood or even platelet transfusions may be appropriate. Most animals that develop hemorrhagic and neoplastic disease do not recover.

Conclusions: Plants often contain toxins that damage the urinary tract and gastrointestinal systems. These selected plants represent some of the most common toxic plants that affect these

systems in livestock in North America. Diagnosis depends on correlating disease and lesions with exposure and chemical analysis of tissues and feeds.

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Figures:

Figure 1: Halogeton (*Halogeton glomeratus)* is a noxious Eurasian weed. Since its introduction in North America it has spread to cover millions of acres. It tends to grow as a monoculture in disturbed areas along roads, in holding pens and heavily used areas around loading pens or watering tanks. Catastrophic poisonings most often occur when hungry sheep are unloaded in such halogeton infested areas.

Figure 2: Photomicrograph of calcium oxalate monohydrate crystals and resulting crystal-associated nephrosis in a sheep poisoned with halogeton.

Figure 3: Horse with oxalate related fibrous osteodystrophy. This as a case is best palpated but there is bony proliferations on the lateral surfaces of the maxilla and mandible.

Figure 4: Kidney of a cow poisoned by oak acorns. Notice the extensive necrosis of the proximal convoluted tubules with eosinophilic granular casts.

Figure 5: Intestine of a Syrian hamster treated with ground cutleaf nightshade (*Solanum triflorum).* Notice the extenvise hemorrhagic enteritis with necrosis of enterocyte deep within the intestinal crypts.

Figure 6: Chokecherry (*Prunus virginiana)* is a common shrub or small tree that grows along rivers, stream and cannel banks. All parts contain cyanogenic glycosides except the fleshy part of the berry (the pits are toxic). Ruminants are most susceptible to poisoning and though chokecherry is not very palatable, cattle will ingest toxic doses when other forages are scarce.

Figure 7: Bracken fern (*Pteridium* spp.) is a prolific perennial that dominates many plant communities. It spreads primarily by an expanding rootstock though it may also produce spores.