



## **TOXIC PLANTS AND COMPOUNDS**

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Fall is the time of year where we see a greater incidence of sheep exposure to toxic plants and chemicals. Unfortunately in most cases the first signs of toxicity are sudden death and depending on the plant or compound death rates can be catastrophic. The most important aspect of treating toxicity is simply to prevent it. This requires a knowledge of potentially toxic plants and chemicals and situations which may lead to toxicity. When we encounter toxicity the cause is either ignorance on the part of the shepherd not realizing that a plant is toxic or accidental exposure where a gate is left open and sheep gain access to areas of the farm that they are not supposed to. If recent exposure to a toxic plant or chemical is suspected treatment with activated charcoal is indicated. Activated charcoal is used to absorb or bind-up the toxic compounds before they are absorbed by the intestine. We also administer mineral oil to speed intestinal transit time to minimize absorption by the intestine. Activated charcoal and mineral oil are generally effective in all exposures to toxic plants or compounds but must be given as quickly as possible after ingestion. In most cases, when clinical signs of toxicity develop it is too late to give charcoal or mineral oil as the absorption and damage has already occurred. The following is a list of common toxic plants and compounds that we encounter, situations where toxicity can occur, clinical signs of toxicity and treatment options. This list is not all inclusive as there are many plants and compounds that can be toxic. This is only a list of those we most frequently encounter. Your local Veterinarian or Extension Office is a good resource for potentially toxic plants that are prevalent in your area.

**Copper** - Without a doubt copper is the leading cause of toxicity that we see in sheep. The causes of toxicity are too high of a level of copper in the feed or feed that is manufactured for cattle or horses that gets fed to sheep. The organ of toxicity is the liver. Clinical signs include rapid breathing, trembling, anemia (lack of red blood cells), red colored urine and death. Treatment is generally unrewarding and sheep that survive may be chronic poor-doers for the remainder of their life.

**Lead** - Lead toxicity occurs when sheep ingest peeling lead paint or exposure to old batteries. Lead causes liver and kidney damage and clinical signs include blindness, seizures, anemia, neurological signs and sudden death. Treatment of clinical cases is generally unsuccessful. Removing non-clinical, exposed sheep from the source is very successful in preventing new cases.

Salt - Salt toxicity occurs either from mixing errors resulting in too much salt in mixed feed or water deprivation. Sheep will not overeat on free choice salt unless it is unavailable for a long period of time. Water deprivation can occur in situations when water sources freeze or in cases of stray voltage preventing access to water. The mechanism of action is increase salt in the blood which causes an increase level of salt in the brain causing cerebral edema or brain swelling. Clinical signs include convulsions, blindness, paralysis, inability to stand and death. Treatment of affected animals consists of injecting dexamethasone, 2mL/100# of body weight, I.V. or I.M. and gradually increasing water intake. Offering unrestricted access of water to a water deprived sheep will result in more rapid clinical signs and higher death rate. Water must be reintroduced in small amounts gradually to prevent over consumption.

Nightshade - Nightshades can cause variable toxicity to sheep. This is dependant on the environment and the maturity of the plant. Sheep avoid nightshade but will consume it in the fall as the nightshade plants stay green longer than grasses. Sheep will also consume it if it is present in harvested hay. Clinical signs include depression, weakness, anorexia, abdominal pain and diarrhea. Treatment of exposed sheep with activated charcoal will minimize clinical signs.

Oak (acorn) - Oak toxicity is one of the most common plant poisonings that we experience in sheep. This occurs usually in the fall when sheep are grazing pastures with oak trees when the acorns drop. If sheep consume acorns in the fall or buds in the spring they will experience oak toxicity. Clinical signs include abdominal pain, constipation, dark tarry feces, teeth grinding and death. The organ of toxicity is the kidney. A post-mortem exam can confirm the diagnosis with large numbers of acorns in the rumen. Treatment of clinical cases is unsuccessful as the kidney damage is severe and irreversible. Removing non-affected animals from oak pasture will prevent further deaths.

Photosensitization - Photosensitization occurs when white-face sheep ingest either a photodynamic pigment or a plant that is hepatotoxic (toxic to the liver). Common examples of these plants in the U.S. are St. John's Wart, Buckwheat, Bishop's Weed and Dutchman's Britches. One to ten days after ingestion sheep will develop swelling in their face, eyelids and muzzle. They are sensitive to light and have sore feet from inflammation at the coronary band of the hoof. Only white-face sheep are susceptible. Treatment includes locking sheep in a shaded environment and injecting dexamethasone (if ewes are not-pregnant) until the sheep return to normal. Often many sheep will be affected but very few will die. Death occurs as a result of the extensive muzzle swelling constricting airways. Prevention involves removal of the toxic plant or removal of the sheep to a different pasture. Generally these plants are not very palatable and sheep will avoid these unless there is a shortage of grass in the pasture.

Nitrate - Nitrate toxicity occurs when sheep graze nitrate accumulating plants such as pigweed, lambsquarter, dock, corn or sorghum or when sheep gain access to nitrogen fertilizer or when sheep consume water contaminated with nitrate. Nitrate accumulating plants accumulate nitrate from the soil into the plant which causes toxicity when grazed. The nitrate is converted to nitrite in the rumen when

changes the hemoglobin in red blood cells preventing it from carrying oxygen. Sheep die quickly from lack of oxygen despite increase respiratory rate. Upon necropsy the blood is chocolate brown rather than bright red. Treatment with methylene blue is effective however generally sheep die so quickly that we do not have an opportunity to treat any. Removal from the source is very effective and treating potential exposures with methylene blue and activated charcoal are very effective in preventing new cases. Ensiling nitrate accumulating plants will decrease the nitrate present and allows safe consumption.

Teratogens - These are plants or chemicals that can cause birth defects in the fetus if the ewe is exposed during pregnancy. The most notable is skunk cabbage (*veratrum californicum*). If ewes consume skunk cabbage between day 12-36 days of gestation we can see facial and leg deformities. Deworming ewes in the first trimester of pregnancy with Valbazen will cause toxicity to the fetus that can result in early embryonic death and abortion. Moldy feed can contain zearalaone which is a biologically active estrogen that can interfere with breeding and conception if moldy feed is fed during breeding season.

Ionophores - Toxicity from Ionophores (Rumensin, Bovatec) occurs as a result of mixing errors or consumption of a concentrated premix that is intended to be used in a diluted form. Rumensin is more toxic than Bovatec. Inclusion rates of 3x the recommend cattle rate can cause toxicity in sheep. The tissue of toxicity is muscle. Exposure to toxic levels causes a severe decrease in feed consumption, muscle weakness and death from heart muscle injury. Treatment is unsuccessful. Preventing access to toxic premixes and correct inclusion rates are important to avoid toxicity.

Japanese Yew - This is an ornamental shrub that is commonly used in landscaping. It is highly toxic to sheep and other ruminants. Consumption of as little as .5% of body weight is lethal. Yew is readily consumed by sheep if trimmings are fed or if they escape their pen in the winter. Clinical signs include sudden death, incoordination, diarrhea and slow heart rate. The organ of toxicity is the heart. Death may occur up to two days following ingestion. Treatment of sheep with clinical signs is rarely successful. Treatment of exposed, non-clinical sheep with activated charcoal and mineral oil will help prevent absorption.