North American Poisonous Snakes

<table>
<thead>
<tr>
<th>Major Species</th>
<th>Usual Time of Onset</th>
<th>Usual Duration (if survives)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All species</td>
<td>Minutes to hours</td>
<td>Several hours to days; potentially lethal</td>
</tr>
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</table>

Clinical Signs - Snake Bites - General

- Snakebite is not always easy to diagnose. The presence of a heavy haircoat and local swelling obscures fang marks. One or more fang puncture wounds may be present on the animal. The most common locations for fang marks in small animals are the face and legs. Horses are commonly bitten in the nose, head, and neck. Bites to the legs of horses are less common and may present with minimal swelling.
- If venom is directly injected into the blood stream, severe systemic signs and sometimes rapid death occur.

Crotalidae - Pit vipers, all have deep pit between eye and nostril. All pit vipers and a few nonpoisonous snakes have vertically elliptical pupils. Pit viper fangs are hollow and retractable, located near the front of the maxilla. The body of pit vipers is distinctly narrower than the back of the head giving them a more "arrow-like" shape than most other snakes.

Agkistrodon - Moccasins, copperheads
A. piscivorus - Cottonmouth moccasin
A. contortrix - Southern copperhead

Adults may attain length of 3.5 feet (average 2.5 feet). Dark forms have dumbbell, hourglass, or crossband figures of reddish brown or chestnut color. Head is generally lighter color than rest of body.

Crotalus - Rattlesnakes
C. horridus - Timber rattlesnake
C. willardi - Ridgenosed rattlesnake
C. adamanteus - Eastern diamondback rattlesnake
C. horridus atricaudatus - Canebrake rattlesnake

Adult C. horridus may attain lenght of 6 feet (average 3 feet). Occur in dark and light color phases, latter often confused with "diamondback". All rattlesnakes have rattle.

Sistrurus - Pygmy rattlesnake
S. catenatus - Massasaupas

Adults can reach 3 feet in length (average 22 - 30 inches). Have rattle, general body color is brown to gray.
**Toxic Principle**

- Pit viper venoms (rattlesnakes, cottonmouth, and copperhead) are primarily necrotizing and hemolyzing; however, other body systems can be involved. Venoms primarily contain enzymatic and proteinaceous toxins. Phospholipase A is a strong hemolytic, myotoxic agent that contributes to cardiotoxicity. L-amino-oxidase and other homologous enzymes may be responsible for the activation of tissue peptidases. Hyaluronidase (spreading factor) may enhance tissue penetration. Proteases are implicated in the anticoagulation events. Nonenzymatic polypeptides are also present, e.g., hemorrhagins, cardiotoxin, neurotoxin. Hemorrhagins in crotalid venom are vasculotoxic causing rapid hemorrhage and edema at the wound site, in addition to systemic hemorrhage and shock.

- In all cases the severity of the bite depends on the location and amount of toxin injected, the size of the animal bitten, and the specific toxicity of the venom. Copperheads (*A. contortrix*) are considered the least toxic, the Mojave rattlesnake (*C. scutulatus*) the most lethal, and the appressive Eastern rattlesnake (*C. adamanteus*) the most likely to envenomate its victim. The amount (if any) of venom injected is regulated by the snake. In Crotalidae up to 20% of bites do not result in envenomation.

- Depth of venom penetration is dependent on the length of the snake's fangs and the presence of "spreading factors".

- Behavioral differences between snakes exists, e.g., aggressiveness.

- Mortality in horses bitten by rattlesnakes has been reported at 10 - 30%. Often occurs 5 - 7 days past envenomation due to secondary complications, e.g., myopathy, hepatopathy.

- Major route of venom excretion is the kidneys. Variable amounts of biotransformation occur.

**Crotalidae Clinical Signs**

- When venom (pit viper) is injected into the tissues, pain, erythema, and edema develop as a result of the activation of bradykinins, histamine, and other inflammatory mediators. Swelling may become extensive and may be accompanied by petechia, ecchymosis, and hematomas.

- In general, crotalid venoms produce alterations in resistance (decrease in arterial pressure) and loss of integrity of blood vessels. Changes in the integrity of blood coagulation mechanisms often occur. In part, these are thought to be due to a thrombin-like enzyme, which acts directly on fibrinogen with subsequent abnormal clotting (Note - In general no change in PT or in the activity of factors VII, X, or XII occurs). DIC may occur.

- If signs of erythema and edema do not occur within 20 minutes, envenomation has probably not occurred; however, it is possible in a minority of cases to have systemic signs without local involvement.

- Systemic signs (dogs) are noted as the clinical course continues, these include hypotension with tachycardia, pulmonary edema, salivation, diarrhea, renal failure, and shock. Cardiac arrhythmias include premature ventricular contractions and ventricular tachycardia have been commonly reported. After a few hours hemolytic anemia often develops. In advanced cases, lethargy and paralysis develop. Death is due to respiratory and circulatory collapse.

- If strikes occur in the head and neck region, local swelling with secondary respiratory distress may be noted.

- Clinical signs in horses include initial excitement, anxiety, and pain. May act colicky. Lameness in all animals may be an early clinical sign.

- Clinical pathology changes: anemia can be profound due to hemolysis and extravasation.

**Elapidae** - Cobra family. The only North American representative is the coral snake (*Micrurus euryxanthus*). Distribution limited to southeast and southwestern USA. Small pair of fangs in the cranial maxilla; 20 - 44 inches in length; tend to be nonaggressive, reclusive. Snakes have to "chew" on prey in order to envenomate. Have bright rings of red and white or yellow, red and yellow bands touch. In nonvenomous snakes the yellow rings are separated on each side by a black ring. Nemonic device: "Red on yellow, kill a fellow; red on black, venom-lack".

Coral snake venom is primarily neurotoxic (due to a neurotoxic polypeptide). Cholinesterase activity is present in the venom of some species; however, this is not thought to be the major neurotoxic principle.
Elapidae Clinical Signs
Coral snake venom causes local pain and swelling. Within a half hour neurotoxic effects may be noted. Clinical signs in dogs receiving sublethal doses are lethargy, tremors, incoordination, and occasional hematuria. Lethal doses are associated with vomiting, salivation, defecation, generalized parasympathetic stimulation, followed by respiratory paralysis and death. Pharyngeal paralysis can result in aspiration pneumonia.

Therapy - Snake Bite - General
● Snake bite is an urgent emergency. In some cases, it is lethal, in many it can cause prolonged and disfiguring injury. Although the animal should receive veterinary care as soon as possible, this should be done while keeping the animal as quiet as possible.
● Even if the snake is killed for identification purposes, caution must be exercised in handling it after death. Envenomation is possible even after a poisonous snake has been decapitated.
● Objectives of therapy are: neutralize the venom, prevent shock, and prevent secondary infections; and sometimes to prevent the further spread of toxins, and remove the venom. The use of alcohol to clean the wound is contraindicated because of its vasodilatory effect, which would promote uptake and spread of venom.

Crotalidae Antivenins:
● Human and canine antivenin is available (Antivenin [crotalidae] - Polyvalent - Dodge Laboratories and Wyeth Laboratories). The manufacturer's recommended dose for dogs is 10 - 50 ml IV (check label for current recommendations). Actual dose, frequency, and duration of administration should depend on alleviation of clinical signs including swelling, pain, shock, etc. Dogs treated within 60 minutes of receiving lethal doses of venom showed increased survival rates. The effectiveness of antivenoms is greatest if administered within 4 hours of the bite. Has variable effectiveness at preventing necrosis. The use of antivenom in humans and domestic animals has been associated with severe anaphylactic reactions (serum sickness). Prettreatment with diphenhydramine is recommended. Epinephrine should be available.
● Canine
  ● Usually one bottle of antivenin is given, but 2 or 3 are sometimes needed.
  ● Antivenin can be mixed in fluids.
  ● Start with 5 ml over a 5-minute period. Monitor the inside of the pinna for hyperemia and the animal's overall appearance for any other evidence of a potential allergic reaction. If there is no reaction, the administration should continue. Antihistamines (such as diphenhydramine) may be used to offset allergic reactions. For anaphylactic reactions, the treatment should also include corticosteroids and epinephrine.
● Horse
  ● Usually 1 to 4 bottles of antivenin are used.
  ● Watch carefully for allergic reactions including anaphylaxis and treat as indicated (see above).
● Coral Snake Antivenin:
  ● Wyeth also manufactures an antivenin for coral snake bites. Keep in mind that signs may be delayed and that responses to antivenin may sometimes be poor after signs are present. Usually 2 vials of antivenin may be given IV before the onset of clinical signs. Start with a low dose and handle apparent allergic reactions as for crotalid antivenin above.
  ● Antibiotics: The prophylactic use of antibiotics is indicated. Broad spectrum antibiotics are commonly chosen.
  ● Respiratory assistance (ventilator) may be needed for 48 - 72 hours for animals with coral snake poisoning.
  ● Fluid therapy: Generally indicated in small animals. Hypotension is a common presenting sign. Maintain diuresis to facilitate excretion, renal damage has been reported in man.
  ● Corticosteroids: Use is controversial. Useful in treating shock but increases in mortality have been reported with their use. They can also alter results of laboratory tests that are otherwise useful in monitoring an animal's progress. Generally used for prevention of shock and hypotension. May not affect local swelling.
  ● Transfusions: Commonly indicated in dogs, if necessary, to treat anemia and hemorrhage.
  ● Supportive: The maintenance of a patent airway is critical. Large diameter tubing or opened syringe cases are commonly placed in the nostrils of horses bitten on the face to keep the airways open. Emergency tracheostomy may be required. Tranquilization in horses may be required.
  ● Tetanus antitoxin should always be given to the horse.

Therapies Generally Contraindicated
● Tourniquet - The use of tourniquets is controversial and usually they are avoided. When used they are most effective in first 30 minutes. Tourniquets increase local tissue damage due to hypoxia. The general location of snake bites (e.g., face) may prevent use. Recommended only for animals in which the tissues below the tourniquet will be sacrificed to save the animal's life!
● Incision and suction - Also controversial. Requires restraint of animal to be effective. Minimal benefit with regards to the local removal of venom. Not recommended unless pocket of venom will clearly be removable. Do not make cruciate incisions. They increase necrosis of the skin.
● Cryotherapy - Commonly associated with increased tissue damage. Not recommended.
● Surgical debridement - Use has not been substantiated. May result in serious scarring and loss of function. May not prevent systemic signs. Not recommended early in course of treatment for envenomation.
Brown Recluse Spider or Fiddle Back (*Loxosceles reclusa*)

<table>
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<tr>
<th>Major Species</th>
<th>Usual Time of Onset</th>
<th>Usual Duration (if survives)</th>
<th>Full Table for Toxicants that Affect Peripheral Circulation and/or that May Cause Reduced Lactation</th>
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<tbody>
<tr>
<td>All species</td>
<td>Minutes to hours</td>
<td>Days to months; rarely lethal; rarely recognized toxicosis</td>
<td></td>
</tr>
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Images


Description

Medium size, brown, on the dorsal surface most have a violin-shaped mark with the handle directed posteriorly.

Toxic Principle

Proteins include protease, hyaluronidase, sphingomyelinase D, and esterase. Have direct lytic effect on RBCs.

Signs

- The bite initially stings, then 1 of 2 forms may take place. The cutaneous form begins as edema, progresses to an ulcerated wound. The viscerocutaneous form, which is severe, produces hemolytic anemia, hemoglobinuria, icterus and hyperthermia.
- Ninety percent of the cases heal in 1 - 3 weeks. Some may need skin grafting.

Mechanism

- Unidentified venom component is cytotoxic to endothelial cells. Triggers intravascular coagulation and microthrombi formation within capillaries. Capillary occlusion, hemorrhage, and necrosis occur.
- Polymononuclear leukocytes and complement play important roles in potentiating the response to envenomation.

Treatment

- Steroids may be used to protect against systemic effects. Manage hemolytic anemia if needed by use of fluids and bicarbonate to minimize hemoglobin deposition in renal tubules and by blood transfusion if anemia is severe enough to justify.
- Early surgical excision or corticosteroids were not effective in experimental rabbit based models of loxoscelism. Allowing ulcerated areas to granulate is acceptable management.
- The leukocyte inhibitor, dapsone, was effective in reducing inflammation at the site of brown recluse venom injection.

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*Loxosceles reclusa*

**Brown Recluse Spider** - Two to four cm leg span. Color range: yellow-tan to dark brown. Has 6 eyes (most spiders have 8 eyes). Violin-shaped marking extending from eyes to abdomen.
Ergot Alkaloids and Gangrenous Ergotism (Saint Anthony's Fire)

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<th>Major Species</th>
<th>Usual Time of Onset</th>
<th>Usual Duration (if survives)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cattle sheep</td>
<td>Days to weeks</td>
<td>Weeks; potentially lethal</td>
</tr>
</tbody>
</table>

**Images**

- Ergot - Google Image Search. - To view this image in full size go to the IVIS website at www.ivis.org.

**Sources**

- The term ergot is used in 2 ways: 1) as the common name for the fungus *Claviceps* which invades the ovary of grasses and cereals and 2) used to refer to the sclerotium, the hard purple mass of mycelium hyphae that replaces the heads of grasses.
- In the midwest, *C. purpurea* is the most common fungus invading grasses. The host plant range includes: rye, triticale, barley, oats, brome grass, timothy and quack grasses.
- In commercial grain trade, wheat or rye are classified as "ergoty" if more than .3% by weight are comprised of ergot sclerotia and oats, triticale or barley are so graded when they contain more than .1%.
- In some parts of the world, *Claviceps* fungi have been isolated from sorghum grain that contained ergot alkaloids.
- Ergot poisoning, sometimes of epidemic proportions has occurred in persons due to the ingestion of smutted rye. Toxic amounts were associated with abortifacient effects and this effect led to its use to hasten labor. Unfortunately, this use led to excessive uterine contraction frequently and often caused ischemic damage to the child. In the present use of ergot alkaloids, the only rational objective is to prevent postpartum bleeding.

**Toxic Principle**

- The toxic action of ergot is due to the numerous alkaloids (ergotamine, ergocristine, ergonovine, etc.) present in the sclerotia and other components-choline, ACH, histamine, sterols. Over 40 alkaloids have been identified which are derivatives of lysergic acid, some of which are inactive.
- The important naturally occurring alkaloids are ergotamine and ergonovine. Of these, ergonovine is more readily absorbed. Both compounds are potent smooth muscle activators. LSD is also derived from ergot, and the smooth muscle contracting activity, although sometimes present, is not always seen. LSD causes depersonalization or hallucinations and may produce toxic psychosis.

**Absorption, Distribution, Metabolism and Excretion (ADME)**

- Metabolism (detoxification) of the ergot alkaloids is assumed to take place in the liver, since toxic effects are more likely in persons with liver dysfunction.
- Meat and milk residues have not been detected in any field outbreaks.

**Mechanism of Action**

- The wide spectrum of pharmacological activity is attributed in part to the alkaloids' agonist/antagonist activity at alpha adrenoreceptors, 5-HT receptors and dopamine receptors. The primary effects are central/ neurohormonal and peripheral. The peripheral effects include an oxytocic-like effect on the uterus and vasoconstriction from smooth muscle contraction.
- The central neurohormonal effect mimics the effects of dopamine. Dopaminergic action may result in a reduction in prolactin which
All smooth muscles, vascular and nonvascular, are contracted, and there is no dependence on innervation or any other chemical mediators.

Only at term is uterine muscle more sensitive to ergot than is other smooth muscle. Therefore in early pregnancy, ergot cannot be used as an abortifacient. Furthermore in early pregnancy, these alkaloids are more stimulatory to the cervix, than to the uterus. Ergot is not recommended in any context as an abortifacient. For the induction of labor, oxytocin is very much preferred.

The blood vessels in all vascular beds are constricted by the ergot alkaloids, but the larger arteries are most sensitive. Occlusion may occur, as shown by arteriograms. The result is ischemia of the tissues perfused by the artery. It is possible for blood flow to be reestablished without permanent damage, or the tissue may become gangrenous before the process reverses. The limbs, toes, ears and tail are often affected, but usually not all in the same animal.

### Toxicity

Animal responses vary with the total and individual alkaloid content, quantity of ergot in the feed, frequency of ingestion, climatic conditions (cool, wet weather) and animal species. All domestic animals, birds and man are susceptible. In human medicine, the gangrenous reaction sometimes occurs at therapeutic doses, although it occurs more often with excessive doses.

### Signs

- Ergot poisoning is commonly seen in cattle. Clinical signs include: reduced feed intake, unthriftiness, lameness, swelling and sloughing of feet below the fetlocks (generally the hind feet). Usually a line of demarcation separates the proximal viable tissue and the distal dry cold epidermis. Less commonly the ears and tail are sloughed.
- Affected extremities may become inflamed and then cold, with numbness and dry gangrene developing. Eventually, there is a painless loss of tissue. The gangrenous form results when animals ingest ergot alkaloids over periods of days or weeks.
- Other clinical signs may include poor milk production, nervous signs, and reduced conception.
- The primary signs in swine with ergotism are agalactia, early parturition, weak or dead pigs, infertility and **reduced rate of gain**.
- In sheep, necrosis of the tongue, gastroenteritis, abortions, gangrene and central nervous system involvement are reported.

### Diagnosis

- Ingestion of ergoty small grains (ergot does **not** occur in corn).
- Ergot alkaloids in grains may be identified in toxicology laboratories by gas chromatography-mass spectrometry or less sophisticated methods including thin-layer and high pressure liquid chromatography.

### Treatment

- Isolate from source.
- Control secondary bacterial infections.
- Move animals to a dry, warm environment.
- Keeping the animals warm is indicated to avoid the added insult of lower environmental temperature to the vascular insult. In warm weather, fly control is essential.
- Of several drugs and maneuvers tried to alleviate the vasoconstriction, only nitroprusside infusions showed any promise.

### Prevention

- Control of ergot - Climatic conditions are involved in yearly incidence.
- Mow grasses prior to flowering.
Ergot - Note the ergot structures extruding from the seed heads of barley (right). These structures replace the normal grain and are the toxic stage in the life cycle of ergot.
**Festuca Arundinacea - Tall Fescue**

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<th>Usual Time of Onset</th>
<th>Usual Duration (if survives)</th>
<th>Full Table for Toxicants that Affect Peripheral Circulation and/or that May Cause Reduced Lactation</th>
</tr>
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<tbody>
<tr>
<td>Cattle</td>
<td>Weeks to months</td>
<td>Weeks to months</td>
<td></td>
</tr>
</tbody>
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Images

- Tall Fescue, *Festuca arundinacea*. Source: Cornell University, Poisonous Plants Informational Database (www.ansci.cornell.edu/plants/index.html). - To view this image in full size go to the IVIS website at www.ivis.org.
- Tall Fescue, *Festuca arundinacea* - Google Image Search. - To view this image in full size go to the IVIS website at www.ivis.org.

Description

- Coarse perennial grass, deeply rooted, in vigorous clumps, reaching 3 - 4 feet in height when mature.
- Leaves broad, dark-green, ribbed, and rough on the upper surfaces.
- Inflorescence paniculate, nodding, to 1 foot in length
- Spikelets many-flowered.

Habitat

Tall fescue is a major forage grass, grown on an estimated 35 million acres and will tolerate being closely grazed. The grass is sometimes allowed to "accumulate or stockpile" for winter grazing. Fescue is sometimes cultivated as hay. Fescue is especially important in the transition area of the USA between North and South.

Toxic Principle

- The toxic principles are apparently produced by the endophytic (inside the grass) fungus, *Neotyphodium coenophialum*, previously known as *Acremonium coenophialum*; and before that, *Epichloe typhina*. This fungus is a *Clavicipitaceae* fungus which is transmitted in the seed (and not via spores) and can, produce ergot alkaloids. Ergovaline is the ergot alkaloid of greatest significance in endophyte-infected fescue. It is estimated that over 90% of tall fescue fields in the United States are infected with the endophyte to some extent, with an average of 62% of the plants infected within each field.
- Pyrrolizidine alkaloids including N-acetylloline, N-formylloline, and dimethyl-N-acetylloline have been isolated from Kentucky fescue. Also, the alkaloid perloline has been identified in endophyte-infected fescue. However, the significance of these types of alkaloids remains to be demonstrated.

Mechanisms of Action

Ergovaline is believed to account for the decrease in prolactin, and other effects of endophyte-infected fescue, most of which are compatible with the effect of ergot alkaloids (gangrene and low milk production, for example).

Signs

- The syndromes associated with tall fescue include:
  - Summer syndrome in cattle.
  - Fescue foot in cattle.
  - Fat necrosis in cattle.
  - Reproductive and lactation problems in cattle, horses and sheep
- Cattle:
  - **Summer syndrome:**
    - Poor weight gain, reduced feed intake
    - Reduced conception rates
    - Intolerance to heat
    - Failure to shed the winter hair coat (rough hair)
  - Elevated body temperature
Nervousness, excessive salivation
- More prevalent where fescue is present as a monoculture and where high nitrogen fertilization is used.

**Fescue foot:**
- Some fescue fields have a history of repeated fescue foot outbreaks, but in general, the outbreaks are non-recurring. Conditions which favor the fescue foot syndrome include the feeding of the fall regrowth or the total yearly accumulated fescue (longer stockpiling increases incidence).
- Lameness most often occurs with the onset of cooler or cold weather (below 60 °F and especially with snow or ice).
- Cattle on low-quality rations, and underweight are predisposed, animals in good flesh are not immune.
- Onset is 5 - 15 days, up to weeks after on pasture, occasionally where continuously pastured.
- Slight arching of the back.
- Rough hair coat.
- Soreness in 1 or both rear limbs, weight shifting, holding 1 foot up.
- Knuckling.
- Loose stools.
- Reddening of the coronary band, then swelling.
- Redness, swelling and then purple-black discoloration of the tail.
- Sloughing of the ear tips.
- Sloughing of the feet, usually at or just above the coronary band, sometimes, higher.

**Fat necrosis:**
- Lipomatosis or fat necrosis is defined as the presence of masses of hard or necrotic fat in the adipose tissue of the abdominal cavity.
- Affected fat has yellowish or chalky white irregular masses of very hard fat, especially in the mesenteries.
- These may be detected on rectal exam. Subcutaneous fat is not affected.
- Digestive disturbances, due to obstruction or intestinal constriction, scanty feces, bloating and occasionally death occur.
- Dystocia due to hardening of fat around the birth canal may occur also.
- Similarly urine retention has been encountered secondary to these fat deposits with associated postrenal uremia.
- Other signs associated with fat necrosis include; weight loss, poor appetite, listlessness and rough hair coat.
- Predisposing factors include the presence of fescue in monoculture and heavy nitrogen fertilization.

**Horse, sheep, cattle.**
- Predisposition to laminitis in horses has also been associated with exposure to endophyte-infected fescue.

**Reproductive and lactation problems** in cattle, horses and sheep: The primary problems are abortion, weak to stillborn young and moderate to severe agalactia. The effects result in the death of many neonates.
- Cattle.
  - Typically a farmer with cows on a problem pasture will have a 70 - 80% calf crop, which is made worse by fertilization, but improves with the addition of clover to the pasture. Dairy cows may have a marked decrease in milk production. Prolactin concentrations are depressed in cows ingesting affected fescue.
- Horses.
  - Horses may abort or more often have dystocia after a prolonged gestation, and may have a thick placenta that increases the need for human assistance during parturition. Serum cortisol concentrations did not increase normally near parturition in mares grazing a problem fescue pasture, and serum prolactin levels were extremely low. The low milk production may be attributable to the low prolactin levels. Prolactin is responsible for the initiation and maintenance of lactation and possibly for the initiation and maintenance of the corpus luteum as well. The decrease in prolactin may therefore also be responsible for the early abortion and fetal resorption in affected species, but this remains to be verified.
- Ewes.
  - Serum prolactin levels are depressed by ingestion of affected fescue.
  - Increased rate of return to estrus.

**Treatment of Fescue Foot**
- Remove from fescue pasture, when 1st signs occur.
- Rule out foot rot, frost bite, mechanical injuries such as stone bruising.

**Management**
- Mix legumes and other grasses with the fescue stand or supplement the diet with other feeds. This appears to work well with cattle but apparently dystocia in pregnant mares may occur with as little as 5 - 10% of the ground cover in a pasture as infected fescue.
- Rotational grazing.
- Do not fertilize with over 200 lb of nitrogen per acre per year.
Feeding hay may lessen problems; field cured fescue hay, processed in the early dough to boot stage, when quality is at a maximum is considered safe for feeding to cattle. However, toxic fescue cut for hay remains toxic.

Observe cattle daily for lameness or stiffness, especially during the first month on pasture and in cold weather. Observe in early AM before cattle warm out of the lameness. Move affected animals to another field or put them on another feed.

Calves on 1 or 2 lb of corn per day had less problems, than those getting no grain.

Adequate protein and mineral supplementation should be assured.

Avoid stress especially in cold weather.

Addition of a legume to the pasture sharply increases gains. Ladino clover also improves conception rates.

If new plantings of fescue are to be done, the use of old seed is recommended, since the fungus dies after 1 year under normal storage conditions in the lower South. New cultivars of fescue, such as Johnstone from Alabama and Triumph are relatively free of the endophyte. Some states require tags on bags of fescue seed to state the percent of endophyte infection. Strains should be chosen with 0 - 5% infection rate.

Tall fescue alkaloids - The authors are skeptical that these 2 compounds play a major role in the toxic effects of fescue.

Tall fescue, Festuca arundinacea.
**Juglans Nigra - Black Walnut - Toxicosis in Horses**

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<tbody>
<tr>
<td>Horses (cause of laminitis in horses)</td>
<td>12 - 24 hours</td>
<td>Days; rarely lethal unless 3rd phalanx rotates through the sole</td>
<td></td>
</tr>
</tbody>
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**Images**

- Black Walnut, *Juglans nigra* - U.S. G.S. Northern Prairie Wildlife Research Center. - To view this image in full size go to the IVIS website at www.ivis.org.

**Source**

- Black walnut (*Juglans nigra*) is native to the eastern United States, growing in the well-drained soil of the Appalachians and midwestern valleys.
- The wood is commonly used and prized as furniture and gun barrel stocks.
- The resultant shavings and sawdust are shipped wherever a market exists.

**Toxic Principle**

- Preliminary investigative work in an effort to identify the causative agent in walnut shavings laminitis was focused on "juglone" (5-hydroxy-1,4-napthaquinone), a napthaquinone toxin found in members of the walnut and hickory family.
- Juglone is also present in other members of the walnut family (Persian or English walnuts, butternuts, hickories, and pecans).
- The structure of juglone is as follows.

![Juglone](image)

**Toxicity**

- Experimental attempts to reproduce the clinical syndrome by using juglone were less than convincing.
- Experiments administering juglone in differing concentrations orally and topically (distal metacarpus to coronary band) have inconsistently produced clinical signs but not full-blown laminitis. Intravenous administration to one horse produced acute pulmonary edema and death.
- Juglone is believed to be responsible for the poor success of other vegetation beneath black walnut trees but is not likely to be responsible for black walnut toxicosis of horses.

**Toxicity**

- Field reports indicate that laminitis has occurred when bedding contained only 5 - 20% black walnut shavings.
- All parts of the tree may be toxic including the heartwood, leaves, roots, and bark.
- Every black walnut tree does not have naturally occurring toxicity, implying environmental factors may alter the tree's toxicity.
The toxin appears to act by causing a sensitization of vessels of the foot to the effects of adrenergic agonists such that acutely there is a reduction in the function of blood flow to the foot that reaches the dorsal laminae.

**Signs**

- Rapid onset, 12 - 24 hours after exposure.
- Commonly, several if not most horses of a group may be acutely affected with laminitis.
- Depression.
- Laminitis.
- Laminitic stance. Can see acute and/or chronic laminitis, and in either case, there can be rotation of P-3.
- Reluctance to move, resistance to attempts to pick up feet.
- Digital pulse evident, elevated hoof temperature in affected digits.
- Slight to moderate edema in limbs, from carpus and tarsus down.
- Increased respiratory rate and effort.
- Increased borborygmi.
- Elevated temperature and pulse.

**Lesions**

Necrosis of dorsal laminae followed by mitotic activity in an effort to repair the damage.

**Treatment**

- Remove black walnut shavings.
- Wash legs with mild soap.
- Mineral oil or activated charcoal and a saline cathartic; do not use mineral oil with a saline cathartic.

**Laminitis Treatment Can Include:**

- Phenylbutazone.
- Soft bedding (damp sand), or padded shoe.
- Removal of shoes.
- Acepromazine, or a more specific alpha blocker (i.e., prazosin), to restore circulation to the dorsal laminae and possibly heparin to prevent microthrombi formation.

**Prevention**

Do not use fresh black walnut shavings for bedding.
**Black Walnut, *Juglans nigra* L.**

**Growth Form** - Large tree up to 150 feet tall; trunk diameter up to 5 feet; crown broadly rounded; trunk straight, columnar, not buttressed at the base.

**Bark** - Black, thick, deeply furrowed.

**Twigs** - Stout, greenish or orange-brown, hairy, smooth and gray; pith brown, divided by partitions; leaf scars alternate, shield-shaped, elevated, with 3 bundle traces.

**Buds** - More or less rounded at the tip, pale brown, soft, hairy, up to 1/2 inch long.

**Leaves** - Alternate, pinnately compounded, with 15 - 23 leaflets; leaflets up to 3 1/2 inches long and 1 1/2 inches wide, broadly lance-shaped, pointed at the tip, rounded at the asymmetrical base, toothed along the edges, yellow-green and smooth on the upper surface, paler and hairy on the lower surface, turning yellow in the autumn.

**Flowers** - Borne separately but on the same tree, appearing when the leaves are partly grown, the staminate several in thick, yellow-green, hairy catkins, the pistillate much fewer in small spikes, neither of them with petals.

**Fruit** - In groups of 1 or 2, spherical, up to 2 inches in diameter, green or yellow-green, slightly roughened, the husk thick, the nut very hard, oval, dark brown, deeply ridged, the seed sweet.

**Wood** - Hard, heavy, course-grained, dark brown.

**Uses** - The wood is used for furniture, interior finishing, cabinets; the nuts are edible.

**Habitat** - Rich woodlands.

**Range** - Massachusetts across to Minnesota, south to Texas, east to Florida.

**Distinguishing Features** - The Black Walnut is recognized by its characteristic buds, its chambered pith, and its fruits.
**Berteroa Incana - Hoary Alyssum**

<table>
<thead>
<tr>
<th>Major Species</th>
<th>Usual Time of Onset</th>
<th>Usual Duration (if survives)</th>
<th>Full Table for Toxicants that Affect Peripheral Circulation and/or that May Cause Reduced Lactation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horses (leg swelling and laminitis)</td>
<td>1 - 2 days</td>
<td>Days to a week; rarely lethal</td>
<td></td>
</tr>
</tbody>
</table>

### Images

### Description
Hoary alyssum is an annual or perennial herb 30 - 90 cm tall with narrow, hairy, alternate leaves 1 - 7 cm long. Flowers are on racemes, very small and white with four deeply divided petals. Pods are hairy and approximately 0.5 cm long.

### Habitat
Hoary alyssum is found in north central and northeastern United States and Canada. It grows well on overgrazed pastures, poorly managed hayfields, and waste places.

### Toxic Principle
Unknown.

### Susceptible Species
Horses.

### Toxicity
The palatability of hoary alyssum is poor, and horses given the choice will usually ignore it in a pasture or preferentially eat another component of hay. In poor pasture or with very weedy hay, this is often not possible.

### Signs
- Fever, pounding digital pulses, limb edema, and laminitis may develop within 24 hours of consumption of hay containing more than 30% hoary alyssum. Death due to hoary alyssum toxicosis has been rare to date. Only mild limb edema has been reported in horses grazing heavily infested pastures.
- Response to the plant is variable. In field cases and feeding trials, fewer than 50% of horses consuming hoary alyssum showed any clinical signs.
- Signs normally resolve within 4 - 5 days following removal of the source of hoary alyssum unless laminitis has occurred. Recovery from laminitis may be very prolonged if severe.

### Lesions
Limb edema, laminitis with or without rotation of P3, petechiae and ecchymoses on serosal surfaces, and occasionally hemoperitoneum or hemothorax. Perivascular hemorrhage and hemosiderin-laden macrophages in the spleen may be seen histologically.

### Diagnosis
Clinical signs, lesions, and history of exposure.
Treatment

- Remove horses from infested pasture or source of hay.
- No standard treatment protocol has been established. Consider the use of activated charcoal for recent exposure.
- Treat for laminitis (see previous section on Black Walnut).

Prevention

- Diligent weed control in pastures and hay fields.
- Inspection of hay before purchase.

Mustard family, *Cruciferae*

Hoary Alyssum, *Berteroa incana* (L.) DC. 1, 2, seed; 3, seed pod; 4, seed pod split open; 5, upper part of flowering plant; 6, flower; 7, stem; 8, lower part of plant. **Annual, biennial, or perennial**, reproducing by seeds. The name hoary indicates many rough hairs on stems, leaves, and seed pods. **Stems** gray-green, hairy, 1 to 3 feet (30 to 90 cm) tall, with many branches near the top. **Leaves** gray-green, hairy, alternate, oblong, narrow, 1/2 to 3 inches (1.3 to 7.5 cm) long, with smooth edges. **Flowers** white, with 4 deeply divided petals, produced in long raceme. **Seed pods** hairy, swollen, oblong with short beak on the end. **Seeds** oblong, rough, dull gray-brown. **Found** in meadows, pastures, and waste places. Emerges early in the year, continues to grow throughout season, producing seeds until frost.
### References

#### North American Poisonous Snakes


#### Brown Recluse Spider (*Loxosceles Reclusa*)


#### Festuca Arundinacea - Tall Fescue


#### Juglans Nigra - Black Walnut - Toxicosis in Horses


#### Berteroa Incana - Hoary Alyssum


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