

In: **Veterinary Toxicology**, V. Beasley (Ed.)

Publisher: International Veterinary Information Service (www.ivis.org), Ithaca, New York, USA.

Thiaminase-Containing Plants and Other Substances (9-Aug-1999)

V. Beasley

Department of Veterinary Biosciences, College of Veterinary Medicine, University of Illinois at Urbana-Champaign, Urbana, IL, USA.

Chapter Sections

Pteridium Aquilinum (Also Called *Pteris Aquilina*) - Bracken fern

Equisetum Arvense - Horsetail

Thiaminase and Thiamine (Vitamin B1) Deficiency in Cats, Dogs, Fur-Bearing and Marine Mammal Species

Pteridium Aquilinum (Also Called *Pteris Aquilina*) - Bracken Fern

Major Species	Usual Time of Onset	Usual Duration (if survives)	Full Table for Thiaminase-Containing Plants and Other Substances
Horses	Weeks to months	Weeks to permanent damage; potentially lethal	

Synonyms - Brake fern, hog brake

Images

- Bracken fern (*Pteridium aquilinum*). Knight A.P. and Walter R.G. (Eds.). A Guide to Plant Poisoning of Animals in North America. Ithaca: International Veterinary Information Service (www.ivis.org), 2003. - To view this image in full size go to the IVIS website at www.ivis.org . -
- Bracken fern (*Pteridium aquilinum*). Knight A.P. and Walter R.G. (Eds.). A Guide to Plant Poisoning of Animals in North America. Ithaca: International Veterinary Information Service (www.ivis.org), 2003. - To view this image in full size go to the IVIS website at www.ivis.org . -
- Bracken fern, *Pteridium aquilinum*. 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 . -
- Bracken fern, *Pteridium Aquilinum* - 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 . -
- Bracken fern - *Pteridium Aquilinum* - Google Image Search. - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

- Native perennial herb, which spreads by means of a deep, stout, especially toxic horizontal rhizome. The rhizome is often below plow depth and results in growth of large patches of the plant.
- Triangular coarse, leaves (fronds) tend to be erect. Fronds are palatable.
- Up to 6 feet tall.
- Spores in late summer; sporangia are protected by the rolled under edges of the blades.

Habitat

- Worldwide, especially forested areas, common where recently cleared, in abandoned fields, open woods, chiefly on dry sandy soils or gravelly areas.
- Often in dense stands.
- Has caused serious losses in the northeastern, southeastern, midwestern, and far western USA. Poisoning most commonly reported in Pacific Northwest and north central Midwest.

Species Affected

- Cattle most commonly affected. Reports of poisoning in horses, swine, and sheep are less frequent.
- Seasonal tendency - Late summer to early fall.
- Horses and cattle are primarily affected, and the syndromes differ:
 - Horses - Mainly neurologic.
 - Cattle - Mainly bone marrow damage.
- Sheep and swine rarely eat bracken, but exposed swine (at least) may sometimes experience a thiaminase mediated syndrome.

Signs

- In the horse:
 - Horses generally must consume bracken fern for 1 - 2 months before the onset of clinical signs occurs. Clinical signs can also occur even when horse is no longer on bracken fern.
 - Emaciation.
 - Loss of weight occurs despite maintenance of appetite until the late stages of the disease.
 - Lethargy.
 - Incoordination, especially when forced to walk. Can progress to overt staggers - Occasionally referred to as "bracken staggers".
 - Wide stance, arched back, crouched stance.
 - Severe tremors, unable to arise, injuries from attempts to get to feet; most pronounced when attempt to work animal.
 - Bradycardia with arrhythmias seen early in disease course. Weak, fast pulse, terminally.
 - Convulsions, recumbency, and opisthotonus terminally. Hyperthermia also observed terminally.
 - Hemolytic crisis rarely reported.
 - If not treated, death in 2 - 10 days (occasionally survive up to 30 days or more after onset).
- In cattle:
 - Considered a "radiomimetic" disease in cattle and sheep.
 - Thiamine production in the rumen results in resistance to the thiaminase syndrome seen in horses.
 - Signs of poisoning begin 1 - 2 months after being turned into bracken containing pasture, or after being given bracken-containing hay.
 - Deaths have occurred at 7 - 8 weeks after first access. Acute course of illness-usually with death within 4 - 8 days of the first onset of clinical signs. Disease develops as a result of bone marrow suppression.
 - Early fever (106 - 108° F).
 - Loss of condition, anorexia.
 - Anemia (late).
 - Bracken fern-induced hematuria in cattle has been called bovine enzootic hematuria.
 - Leukopenia.
 - Thrombocytopenia, blood-tinged nasal discharges, bloody or "tarry" feces, blood clots in feces, hematuria. Prolonged clotting times, defective clot retraction.
 - Edema of larynx and dyspnea.
 - Differential diagnosis in cattle includes: septicemia, anaplasmosis, moldy sweet clover ingestion, and leptospirosis.
 - Bladder and upper gastrointestinal carcinoma in cattle exposed to bracken fern for 1 - 6 years.
- In swine:
 - Depressed growth rate, dyspnea, weakness, recumbency, and death.
 - Clinical signs are the result of thiamine deficiency.
 - Elevated pyruvate levels.

Diagnosis

- Evidence of significant ingestion.
- Appropriate clinical signs and lesions.
- Thiamine analysis of whole blood (avoid hemolysis) of monogastrics may help support the diagnosis. Contact Dr. Dwayne Hamar, Department of Pathology, Veterinary Diagnostic Laboratory, Colorado State University, USA.

Lesions

- In horses:
 - Enteritis, with some pericardial and epicardial hemorrhages.
- In cattle:
 - Generalized hemorrhages, anemia, aplastic marrow.
 - Ulceration of the abomasum.
 - Lesions in urinary tract most often involve the bladder. Lesions range from hyperplastic and hemorrhagic inflammation to frank neoplasias.
- In sheep:
 - Great Britain-ingestion of bracken fern has been associated with retinal atrophy, "bright blindness". Characterized by loss of rods and cones and outer nuclear layers. Experimentally reproduced by feeding bracken to sheep.
 - Hemorrhagic syndrome similar to cattle.
- In swine:
 - Enlarged, mottled heart, congestive heart failure.
 - Lungs edematous, ascites, and enlarged gall bladder.
 - Degeneration of cardiac muscle fibers.

Treatment

- In horses:
 - Saline cathartic.
 - Activated charcoal.
 - Thiamine HCl - At a dose of 0.25 - 0.5 mg/kg, IV or IM, daily for several days, up to 2 weeks.
- In swine:
 - Treatment of swine is similar to that described for horses.
- In cattle:
 - Blood transfusions - 1 gallon per day at onset.
 - Broad spectrum antibiotics.
 - d,1-batyl alcohol: Treatment with d,1-batyl alcohol is not consistently effective.
 - Preparation of d,1-batyl alcohol for intravenous administration: add 5 grams of Tween-80 or Tween 20 emulsifier to 100 ml saline and dissolve in a water bath. After heating, add 2 grams of d,1-batyl alcohol and shake vigorously periodically until homogeneous. After cooling in cool water, the mixture should be only mildly opalescent. If not, reheat with shaking and cool again. This approximately 2% batyl alcohol solution may be given intravenously.
 - Preparation for subcutaneous administration: the concentrated alcohol may be diluted in olive oil at a rate of 1 gram batyl alcohol to 10 ml of the oil.
 - The dose administered is 1 gram of d,1-batyl alcohol intravenously or subcutaneously daily for five days. d,1-batyl alcohol available from Sigma Chemical Co., St. Louis, MO.
 - Protamine sulfate (1%), a heparin antagonist administered as a 10 ml injection, in conjunction with blood transfusions may be of benefit.
 - Phenothiazine added to the diets of mice and rats reduced the incidence of bracken fern-induced tumors. May be result of increased liver microsomal enzyme activity and subsequent metabolism. Questionable efficacy in ruminants (sheep) experimentally.

Major Areas of the Distribution of Bracken Fern. Spores on Underside of Fronds





Bracken Fern. Note the large pinnately divided leaf-like frond, the spore-bearing frond (lower left), fiddlehead (lower center), unfolding fiddlehead (lower right), and the underground rootstock (lower right) of this primitive plant.

Equisetum Arvense - Horsetail

Major Species	Usual Time of Onset	Usual Duration (if survives)	Full Table for Thiaminase-Containing Plants and Other Substances
Horses	Weeks to months	Weeks to permanent damage; potentially lethal	

Synonyms - Scouring rush, maretail, and jointed rush

Images

- Horsetail, *Equisetum Arvense*. Knight A.P. and Walter R.G. (Eds.). A Guide to Plant Poisoning of Animals in North America. Ithaca: International Veterinary Information Service (www.ivis.org), 2003. - To view this image in full size go to the IVIS website at www.ivis.org . -
- Horsetail, *Equisetum Arvense*. Knight A.P. and Walter R.G. (Eds.). A Guide to Plant Poisoning of Animals in North America. Ithaca: International Veterinary Information Service (www.ivis.org), 2003. - To view this image in full size go to the IVIS website at www.ivis.org . -
- Horsetail, *Equisetum Arvense*. 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 . -
- Horsetail - *Equisetum Arvense* - Google Image Search. - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

- Perennial herb, very short or up to 4 feet tall.
- Hollow stems-green, segmented.
 - Harsh in texture due to large amount of silica.
 - Stems readily separate at segmentations (nodes).
- Leaves-vestigial in whorls at the nodes.

Habitat

- Native - Midwest - West - All USA - Especially cooler areas.
- Moist, wet areas.
 - E.g., meadows, along ditches, ponds, etc.
 - Tolerates soils low in fertility.
 - Low lying areas in pastures and fields.

Toxic Principle

- Thiaminase in nonruminants.
- Unknown in ruminants.
- Remains toxic when dried.

Susceptible Species

- Horses primarily affected, especially younger animals.
- Rarely cattle, sheep.
- Poisoning on pasture, but mostly in winter when contaminated hay is fed.

Diagnosis (See Bracken Fern [*Pteridium*])

Signs

- Clinical signs develop after eating several days-onset is characterized by a loss of weight.
 - Weight loss becomes chronic with continued ingestion.
- The syndrome progresses at a rate dependent on intake.
- Signs of a nervous disorder and muscular weakness.
- After approximately 1 month, incoordination may affect hind limbs first.
- At 40 - 45 days, paresis occurs and continued ingestion results in convulsions and death within 1 - 15 days. Progressive muscular rigidity.
- Tenesmus and constipation marked.

Lesions

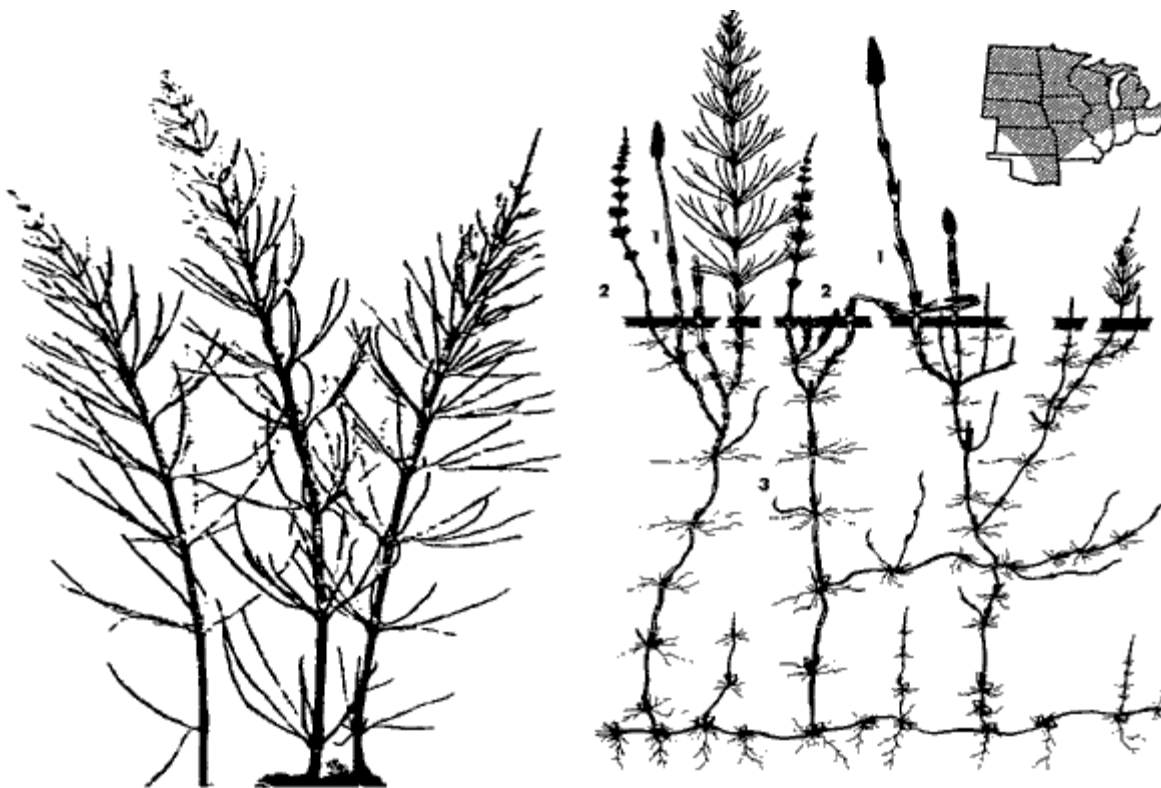
- Pale and flabby skeletal musculature.
- Hydroperitoneum, congestion, and inflammatory edema of the meninges.
- Congestion of lungs, proximal renal tubular epithelial degeneration.

Treatment

- Remove from source.
- Supplement with good diet.
- Inject thiamine HCl at 0.25 - 0.5 mg/kg IV or IM for several days.

Prognosis

Favorable unless the animal is unable to arise at outset.



Field Horsetail, *Equisetum arvense* L. **1**, fertile or reproductive shoots; **2**, sterile or vegetative shoots; **3**, rhizomes. **Perennial**, reproducing by spores instead of seeds, and by rhizomes, to which are attached small tubers. **Stems** tough and wiry, hollow, jointed, and of two types: *fertile*, producing fruiting heads and having large, easily separable joints, not branched; *sterile or vegetative*, having much smaller joints, with lateral branches in whorls around the main stem. **Leaves** on sterile stems only, in the form of cupshaped toothed sheaths at the joints. **Fruiting heads** contain masses of tiny pale greenish spores in small pine-cone-like structure. **Found** mostly on wet, sandy, or gravelly soil. It is poisonous to livestock when eaten in large quantities. There are several species of *equisetum* in the area, all of which can be recognized as horsetails from this illustration.

Thiaminase and Thiamine (Vitamin B₁) Deficiency in Cats, Dogs, Fur-Bearing and Marine Mammal Species

Major Species	Usual Time of Onset	Usual Duration (if survives)	Full Table for Thiaminase-Containing Plants and Other Substances
Cats, dogs, foxes, mink, captive marine mammals	Weeks	Days to permanent damage; potentially lethal	

Introduction

Thiamine (vitamin B₁) deficiency occurs in cats fed all fish diets that contain thiaminase, and occasionally dogs fed cooked meats (cooking can destroy thiamine). Occasionally, it occurs in chronic anorexia, or extensive diuresis without vitamin supplementation (especially in cats which have higher thiamine requirements than dogs).

Sources

- Many kinds of fish, from both fresh and salt waters, e.g., carp, catfish, smelt, whitefish, white bass, herring, and others, include thiaminase.
- Thiamine may also be destroyed by excessive heating or prolonged storage of foods.

Mechanism of Action

See Bracken fern handout.

Susceptible Species

- Cats, dogs.
- Mink (Chastek paralysis), foxes (Chastek paralysis), seals, sea lions, bottle-nose dolphins, etc.

Clinical Signs

- Thiamine deficiency predominately affects the nervous, gastrointestinal, and cardiovascular systems.
- In the earliest stages, cats develop anorexia, salivation, vomiting, and weight loss. Ataxia and dyspnea may develop.
- Other clinical signs include a mild vestibular ataxia (frequently asymmetrical, no loss of strength, circling, falling, or tipping toward the side of the lesion). Sometimes signs of acute vestibular disturbances accompany a prolonged period of inappetence, and complete remission of signs follows thiamine therapy.
- Cats may also develop circling, dysmetria, dilated pupils, and hypersensitivity.
- Generalized convulsions frequently are elicited by handling the cat. Convulsions in cats are characterized by kyphosis (ventroflexion of the head), hyperesthesia, and unresponsive pupils. Seizures are often generalized and are "grand mal" and of short (< 30 seconds) duration.
- Occasionally, retinal hemorrhages occur.
- Cardiovascular abnormalities in the cat include bradycardia or tachycardia with arrhythmias.
- Pupils may be dilated and poorly responsive to light.
- Terminally, there is semicoma, continual crying, opisthotonus, and persistent extensor tonus. Can develop 3 - 4 days after the initial clinical signs.
- Dogs develop upper motor neuron paraparesis that progresses to tetraparesis and convulsions. Dogs have persistent anorexia, with paraparesis developing several days after the onset of anorexia.
- Thiamine deficiencies in nondomestic carnivores can produce a dry, unkempt coat, and paralysis. The condition is also known as Chastek paralysis in the fox.

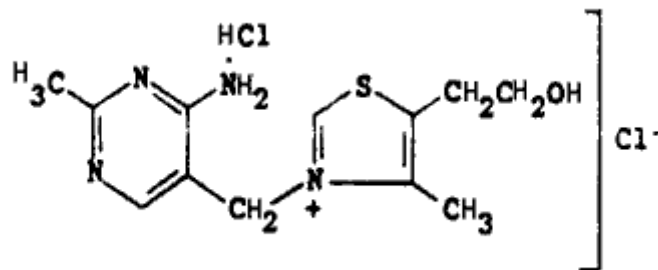
Lesions

- Symmetric periventricular polioencephalomalacia is the characteristic lesion in thiamine deficiency. Lesions can vary in severity from bilateral spongy change to frank necrosis.
- Unlike hypoxia, and some toxin-induced changes, these lesions are primarily located in the brainstem. Terminally, there is a bilaterally symmetrical hemorrhagic necrosis of the brain stem periventricular grey matter, which includes lateral geniculate nuclei, oculomotor nuclei, and vestibular nuclei.
- Cats may also have ulceration of the mucosa of the pylorus.
- Generalized vascular dilatation including retinal, brain, and spinal cord vasculature.

Diagnosis (See Bracken Fern [*Pteridium*])

Therapy

- Treatment for thiamine deficiency should be instituted in any animal in which it is suspected based on history and clinical signs.
- Therapy with 2 mg/kg/day of thiamine hydrochloride (IM) early in the disease will generally result in clinical improvement. A single dose of 250 mg was tolerated and effective in a thiamine-deficient border Collie.



Thiamine HCl (Vitamin B1)

- Thiamine deficiency also occurs naturally in mink as well. Has been described in captive seals, sea lions, and bottle-nosed dolphins.

References

***Pteridium Aquilinum* (Also Called *Pteris Aquilina*) - Bracken Fern**

1. Cheeke PR, Shull LR. Natural Toxicants in Feeds and Poisonous Plants. Westport, Connecticut: AVI Publishing, 1985.
2. Evans WC, Widdop B, Harding JDJ. Experimental poisoning by bracken rhizomes in pigs. *Vet Rec* 1972; 90:471-475.
3. Oehme FW. Plant toxicities. In: Robinson, ed. Current Therapy in Equine Medicine. Philadelphia: WB Saunders, 1987; p.672-682.
4. Osweiler GD, Ruhr LP. Plants affecting blood coagulation. In: Howard JL, ed. Current Veterinary Therapy II Food Animal Practice. Philadelphia: WB Saunders, 1986; p.404-406.
5. Parker WH, McCreg CT. Bracken (*Pteris aquilina*) poisoning of sheep in the North York moors. *Vet Rec* 1965; 77(30):861-865.
6. Watson WA, Barnett KC, Terlecki S. Progressive retinal degeneration (bright blindness) in sheep: A review. *Vet Rec* 1972; 91:665-670.

***Equisetum Arvense* - Horsetail**

1. Henderson, JA, Evans, EV, McIntosh, RA. The antithiamine action of *Equisetum*. *JAVMA* 1952; 120:375-378.

Thiaminase and Thiamine (Vitamin B1) Deficiency in Cats, Dogs, Fur-Bearing and Marine Mammal Species

1. Geraci JR. Thiamine deficiency in seals and recommendations for its prevention. *JAVMA* 1974; 165:801-803.
2. Holzworth J. Diseases of the Cat Medicine and Surgery. Philadelphia: WB Saunders, 1987; p. 28-30.
3. Loew FM, Martin CL, Dunlop RH, et al. Naturally occurring and experimental thiamine deficiency in cats receiving commercial cat food. *Can Vet J* 1970; 11(6):109-113.
4. Read DH, Jolly RD, Alley MR. Polioencephalomalacia of dogs with thiamine deficiency. *Vet Pathol* 1977; 14:103-112.

Kochia

1. Gould DH, McAllister MM, Savage JC, et al. High sulfide concentrations in rumen fluid associated with nutritionally induced polioencephalomalacia in calves. *Am J Vet Res* 1991; 52:1164-1169.
2. McAllister MM, Gould DH, Hamar DW. Sulphide-induced polioencephalomalacia in lambs. *J Comp Pathol* 1992; 106:267-278.
3. Cummings BA, Gould DH, Caldwell DR, et al. Ruminant microbial alterations associated with sulfide generation in steers with dietary sulfate-induced polioencephalomalacia. *Am J Vet Res* 1995; 56:1390-1395.
4. Cummings BA, Caldwell DR, Gould DH, et al. Identity and interactions of rumen microbes associated with dietary sulfate-induced polioencephalomalacia in cattle. *Am J Vet Res* 1995; 56:1384-1389.
5. Gould et al. *J Vet Diag Invest* 1997 in press.

All rights reserved. This document is available on-line at www.ivis.org. Document No. A2607.0899.

