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Plants Affecting the Musculoskeletal System (2-Apr-2004)

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Lameness due to musculoskeletal disorders is relatively common in animals that consume poisonous plants. Many toxic plants cause lameness through muscular weakness induced by the debilitating effects of the toxins on other organs. For example, livestock with liver disease caused by eating tansy ragwort (*Senecio jacobea*) eventually have a severe weight loss that results in muscle weakness and lameness. Similarly, plants affecting the brain and peripheral nervous system can affect muscle function and cause secondary lameness. Relatively few toxic plants primarily effect the musculoskeletal system, but a few such as day blooming jessamine exert their primary effect on the muscles and bones [1]. In this chapter only those plants that are a primary cause of lameness are discussed.

Plant-Induced Calcinosis

A variety of plants contain calcinogenic glycosides that may be converted to a vitamin D-like substance in animals. Chronic exposure to these vitamin D analogs result in excessive amounts of calcium being absorbed from the intestinal tract and deposited in the tissues. Over time these calcium deposits in muscles cause chronic lameness and weight loss. Affected cattle and horses may survive for several years before they are unable to walk and become permanently recumbent.

Plants that have been incriminated as a cause of calcinosis in animals include *Solanum malacoxylon*, *S. sodomum* (sodom apple), *S. linnaeanum*, *trisetum flavescens* (golden oat grass), *Cestrum diurnum* (day-blooming jessamine), and *Nierembergia veitchii* [2-5]. Of these plants, only *C. diurnum* is known to cause calcinosis in horses in North America.

Day-Blooming Jessamine

Cestrum diurnum - Solanaceae (Nightshade family)

Habitat

Introduced from the West Indies, *C. diurnum* has become widely distributed through Florida, Texas, California, and Hawaii.



Habitat of Day-Blooming Jessamine. *Cestrum diurnum* - Solanaceae (Nightshade family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

This plant is a shrub or small tree up to 15 feet (4 to 5 meters) high, with alternate elliptic leaves that have a dark green glossy upper surface. The fragrant white tubular flowers are born in small clusters on axillary peduncles. Multiple green berries that turn black when ripe are produced after flowering (Fig. 9-1A).



Figure 9-1A. Day-blooming jessamine (*Cestrum diurnum*) (Courtesy Dr. Julia F. Morton, Miami, Florida). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Cestrum diurnum contains a toxin that has strong similarities to 1,25-dihydroxyc- holecalfiferol, the active metabolite of vitamin D [6,7]. Consequently, all animals eating cestrum while on a diet adequate in calcium and phosphorus absorb excessive amounts of calcium. Prolonged consumption of the plant results in the calcification of the elastic tissues of the arteries, tendons, and ligaments (calcinosis) [6,8,9]. The calcium deposited in the muscles, tendons and ligaments may become evident within 2 weeks of the animal starting to eat the plant. Generalized increased density of the bones (osteopetrosis) may also be related to hypoparathyroidism and hypercalcitoninism induced by the plant toxin [9].

Other members of the genus, *Cestrum nocturnum* (night-blooming jessamine) (Fig. 9-1B), *C. aurantiacum* (Fig. 9-1C), and *C. parqui* (green cestrum, willow-leaved jessamine) cause toxicity in livestock through the action of atropine-like alkaloids that are common in the family Solanaceae (see Chapter 3). These species of *Cestrum* have not been associated with calcinosis.



Figure 9-1B. Night-blooming jessamine (*C. nocturnum*). (Courtesy of Dr. Gerald D. Carr, Botany Department, University of Hawaii). - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 9-1C. Cestrum (*C. aurantiacum*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Clinical Signs

Chronic weight loss despite normal appetite, and generalized stiffness leading to severe lameness and prolonged periods of recumbency are characteristic. Lameness arises from pain in the ligaments and tendons where the calcium is deposited [8,9]. Heart murmurs may develop due to the calcification of the heart valves. Plasma calcium levels in animals with cestrum

poisoning are consistently elevated in the range of 11 to 16 mg/dL [8]. Other blood parameters, including phosphorus levels, are generally normal. Radiographically, a marked increase in bone density (osteopetrosis) is apparent, with increased calcification of cartilage and increased metaphyseal and epiphyseal trabeculae [9].

Severe calcification of the tendons, ligaments, and elastic arteries is often visible on postmortem examination. Mineralization of tissues is confined to those containing elastic tissue [8,9]. Chronic cases may show severe calcinosis of the aorta, pulmonary arteries, heart valves, and endocardium [8].

Recovery from *C. diurnum* poisoning is rarely reported because animals are usually affected chronically. Recovery is likely in less severely affected animals if they are denied further access to the plant and are given a balanced ration. Care should always be taken to ensure horses are not placed in pastures or pens surrounded by or containing day-blooming jessamine.

Plants Causing Muscle Degeneration

A variety of plant toxins are capable of causing muscle degeneration (myodegeneration) in addition to their other effects on different organ systems [1]. The golden chain tree (*Laburnum anagyroides*), scotch broom (*Cytisus scoparius*), and coyotillo (*Karwinskia humboldtiana*) are plants containing toxins that affect the nervous system and cause myodegeneration. Lupinosis is a degenerative disease of muscle and liver of sheep that graze the stubble of cultivated lupines containing fungal toxins (phomopsins) produced by the fungus *Phomopsis leptostromiformis* (*Diaporthe toxica*) [10,11]. Many of the toxic plants discussed in Chapter 6 that affect the nervous system also have secondary effects resulting in lameness.

Relatively few plants contain toxins that only cause degeneration of the musculature. A few plant toxins including gossypol found in cottonseed and tremetol in white snake-root (*Eupatorium rugosum*) cause degeneration of the heart muscle. In North America, mountain thermopsis (*Thermopsis montana*) and coffee weed or sickle pod senna (*Cassia* spp.) are examples of plants capable of causing primary muscle degeneration.

Senna, Coffee Weed, Coffee Senna

Cassia occidentalis (*Senna occidentalis*)

Sickle Pod, Coffee Weed, Coffee Bean

Cassia obtusifolia (*C. tora*) (*Senna obtusifolia*) - Fabaceae (Legume family)

Less common species

C. roemeriana - Twin-leaf senna

C. fasciculata - Showy partridge pea

C. lindheimeriana - Lindheimer senna

C. nictitans - Wild sensitive plant

Habitat

The *Cassia* spp. are common and troublesome weeds of the southeastern United States, Hawaii, Mexico, and most of the tropical world. As annuals they are opportunists, growing in waste areas, roadsides, fence lines, and are especially common as weeds of corn and soybean fields.



Habitat of Senna. *Cassia occidentalis* (*Senna occidentalis*) - Fabaceae (Legume family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

The *Cassia* spp. are woody, erect, lightly branched annual and occasionally perennial shrubs, 6 to 8 feet (2 to 3 meters) tall. The leaves are alternate, pinnate, consisting of four to five pairs of leaflets widely spaced along a common stalk. Each leaflet is rounded at the base and pointed at the other end. A raised gland is present near the upper side of the base of each petiole. The flowers are yellow and produced in loose clusters in the leaf axils. Thick, dark brown, slightly flattened and curved seed pods with paler longitudinal stripes along the edges contain dark brown seeds.

As the name implies, sickle pod cassia has much longer curved pods, up to 8 inches (20 cm) in length, that are distinctly sickle-shaped and contain many seeds (Fig. 9-2).



Figure 9-2. Sickle pod senna (*Cassia obtusifolia*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxins

Several compounds that bind strongly to cell membranes occur in *Cassia* spp., but the specific toxin responsible for muscle degeneration has not been identified [12-14]. The toxin induces acute muscle and liver degeneration that can be rapidly fatal in most animals [15-20]. The greatest concentration of the toxin appears to be in the seeds. *C. occidentalis* and *C. obtusifolia* are considered to be more toxic than other species, but all *Cassia* spp. should be considered toxic unless proven otherwise.

Cattle, sheep, goats, horses, pigs, rabbits, and chickens are susceptible to poisoning by *Cassia* spp. [13,19-26]. All parts of the plant are toxic, although most poisoning occurs when animals eat the pods and beans, or are fed green-chop containing cassia [27,28]. Ground beans of coffee senna fed to cattle at the rate of 0.5 percent of their body weight induces severe muscle degeneration [15]. Roasting of the beans partially reduces their toxicity such that goats fed 2.5 g/kg body weight of roasted beans were unaffected, whereas unroasted beans at this dosage were fatal [23,24,27]. Cassia poisoning in cattle may occur when 0.4 to 12.0 percent of body weight of the green plant is eaten [15,17,28]. At lower doses, *Cassia* spp. can cause diarrhea and decreased weight gain [27]. The plant is not very palatable and tends to reduce feed intake. As the amount of *Cassia* in the animal's diet increases muscle degeneration becomes a predominant characteristic of the poisoning and cause of the clinical signs. Experimentally, high doses of the plant (10 g/kg body weight daily for 3 days) induce acute liver degeneration and death before myodegeneration has time to develop [13].

Clinical Signs

In cattle a moderate to severe diarrhea develops shortly after consumption of the plant [30]. Abdominal pain, straining (tenesmus), and diarrhea are thought to be due to the irritant effects of anthraquinones in *Cassia* spp. Affected animals remain afebrile. Depending on the amount of plant or seeds consumed, muscle degeneration begins after several days, causing weakness and recumbency [20,21,31,32]. The urine may be coffee colored due myoglobinuria from acute muscle degeneration [13,14,30]. The levels of serum enzymes creatine kinase and aspartate transaminase are usually markedly elevated, reflecting acute muscle degeneration. Renal failure may develop secondarily to the myoglobinuria. In severe cases hepatic failure may be the predominant organ failure leading to death of the animal [14]. In more chronic cases, cardiomyopathy and hyperkalemia from muscle degeneration cause cardiac irregularities and contribute to the death of the animal [15,16,27]. Respiratory difficulty develops as a result of the degeneration of the intercostal and diaphragm muscles [22].

Horses may not exhibit the digestive and muscle degenerative signs of poisoning seen in cattle. Myoglobinuria may not develop in horses because they apparently succumb to liver degeneration sooner than to the degeneration of the musculature [19]. Poisoned horses are generally afebrile and severely ataxic and may die without showing other clinical signs. Serum liver enzymes may be elevated reflecting acute liver degeneration.

Gross lesions at postmortem examination consist primarily of pale skeletal muscles similar to those seen in white muscle disease associated with selenium and vitamin E deficiency. Skeletal muscle necrosis and renal tubular and hepatic centrallobular necrosis are characteristic histologic findings that differentiate cassia poisoning from vitamin E and selenium deficiency [15,16,20]. Confirmation of the diagnosis should be based on access to and consumption of *Cassia* spp. , along with the presence of degenerative lesions in the muscles, heart, and liver [16,18,27].

Treatment

There is no specific treatment for cassia poisoning. Affected animals should be removed from the source of the plants as quickly as possible and fed a nutritious diet. Supportive care of the recumbent animal will help prevent further muscle degeneration due to pressure necrosis. Where warranted, intravenous fluid may be helpful in maintaining renal function if myoglobinuria is present. Recovery depends on the severity of muscle and liver degeneration that has resulted. Rarely does an animal recover once it has become recumbent.

It is important to differentiate white muscle disease due to selenium and vitamin E deficiency from cassia poisoning because the use of selenium and vitamin E in cassia poisoning is contraindicated. Increased myodegeneration and higher mortality occur when selenium and vitamin E are used to treat cassia poisoning [15].

Golden Banner, Mountain Thermopsis, False Lupine, Yellow Pea

Thermopsis montana, *T. rhombifolia* - Fabaceae (Legume family)

Habitat

Golden banner is a common wildflower of the Rocky Mountains (*Thermopsis montana*) and the prairies (*T. rhombifolia*) from North Dakota to Oregon and Washington, and south to Nevada and Colorado.



Habitat of Golden Banner. *Thermopsis montana* - Fabaceae (Legume family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

These plants are perennials, arising from a rhizomatous root system, with erect, branching stems that reach a height of 12 to 18 inches (30 to 46 cm). Leaves are alternate, with three leaflets, unlike lupine that have five or more. Flowers are bright yellow, pealike, and produced in dense racemes from the leaf axils (Fig. 9-3A). The seed pods are densely haired, erect, and straight (*T. montana*) or curved (*T. rhombifolia*).



Figure 9-3A. Golden banner, false lupine (*Thermopsis montana*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

A variety of quinolizidine alkaloids including thermopsine, cytisine, N-methylcytisine, and anagryne have been isolated from all parts of the plant. The specific toxin responsible for the myopathy has not been identified [33-35]. The quinolizidine alkaloid fraction, however, induced the same lesions as did the entire plant when fed to cattle [34]. Experimentally, a dose of 1 g/kg body weight of dried plant given orally, once daily for 2 to 4 days, consistently induced muscle degeneration [3].

Clinical Signs

Cattle poisoned by *T. montana* initially show reluctance to move, walk with a stiff gait, and show muscle tremors when forced to move [33-37]. Severe depression, anorexia, arched back, and swollen eyelids have also been observed in affected animals. Depending on the quantity of plant consumed, animals become recumbent and eventually die. Severely affected animals may die acutely from respiratory arrest.

Levels of serum creatine kinase and aspartate transaminase are markedly elevated, reflecting acute muscle degeneration. Myoglobinuria has not been associated with poisoning caused by *Thermopsis* spp.

At postmortem examination, there are usually few gross signs. Microscopic muscle degeneration of the skeletal muscles is a characteristic finding, although there is usually no myocardial degeneration [34,34].

Scotch broom (*Cytisus scoparius*), a bushy shrub that was introduced from Europe, has occasionally been suspected of animal poisoning, especially in the horse. Having escaped from cultivation, scotch broom has become a troublesome weed along the west coast of North America from British Columbia to southern California. It grows 6 to 8 feet (2 to 2.5 meters) in height and has evergreen or deciduous palmate three-lobed leaves with showy yellow pealike flowers (Fig. 9-3B). Scotch broom contains several quinolizidine alkaloids including sparteine, cytisine, and isosparteine with the potential for similar toxic effects caused by golden banner (*Thermopsis montana*) and the golden chain tree (*Laburnum anagyroides*) (Fig. 9-3C).



Figure 9-3B. Scotch broom (*Cytisus scoparia*). Inset showing detail of the flowers. - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 9-3C. Golden chain tree (*Laburnum anagyroides*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Black Walnut

Juglans nigra - Juglandaceae (Walnut family)

Habitat

About 15 species of walnuts are widely distributed throughout the world; 6 species are native to North America. Most are deciduous trees growing to 60 feet in height. The black walnut is most commonly found in cultivation, where it is extensively used for its wood, aromatic oils, and edible nuts.

Description

Black walnuts are large trees with rough dark brown bark, with pinnate leaves to 50 cm long and 11 to 23 leaflets (Fig. 9-4). Male and female flowers are produced separately; the male flowers have 12-cm catkins, and the female flowers are 0.5 to 1 inch (1 to 2 cm) long with yellow-green stigmas. The fruits are ovoid, single hard-shelled nuts containing the edible fruit.



Figure 9-4. Black walnut leaves and fruits (*Juglans nigra*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

The toxin responsible for black walnut toxicosis in horses is not known. Juglone (5-hydroxy-1,4-naphthoquinone), present in the roots, bark, nuts, and pollen of the walnut tree, is possibly involved with poisoning in horses. Juglone is found in other members of the walnut family including English walnuts, butternuts, hickories, and pecans. Walnut trees are allelopathic, meaning they secrete chemical substances through their roots to inhibit the growth of other plants in the vicinity. Consequently, many plants will not grow under walnut trees.

Horses become poisoned if they are exposed to the wood shavings of black walnuts that are used for bedding [38,39]. Bedding containing as little as 20 percent of black walnut shavings can cause the development of laminitis in horses [40]. It is

not necessary for horses to eat walnut shavings to develop laminitis. Pollen and the leaves in the autumn are also toxic to horses [41].

The variability of laminitis, edema of the lower legs, colic, and other systemic signs associated with black walnut shavings is poorly understood. The clinical signs are not simply related to contact of the horse's skin with the walnut shavings. Purified juglone applied topically to the feet of horses causes mild dermatitis but does not cause laminitis [39]. However, horses experimentally treated with aqueous extracts of black walnut via nasogastric tube consistently develop acute laminitis, indicating that toxicity is due in part to the ingestion or inhalation of a toxic substance present in black walnut [42-46]. It has also been postulated that juglone or other substances act as haptens to induce toxicity [39,41]. Experimental evidence indicates that the toxin in black walnuts does not directly cause contraction of the digital vessels responsible for the laminitis, but it appears to enhance vasoconstriction of the blood vessels in the presence of catecholamines and corticosteroids [44,45,47].

Fallen walnuts that have become moldy may contain the mycotoxin penitrem A, which is a neurotoxin capable of poisoning dogs and other animals [48].

Clinical Signs

Naturally occurring black walnut toxicosis in horses is characterized by depression, edema of the lower legs, lameness, colic, and respiratory distress [38,40]. The severity of lameness depends on the duration and severity of laminitis. If affected horses are removed from the source of the black walnut shavings in the early stages of laminitis and are treated for the laminitis, they recover without the severe consequences of hoof deformity and third phalanx rotation attributable to laminitis.

Until the toxin and conditions causing black walnut toxicosis are better understood, it is a wise precaution to avoid bedding horses with wood shavings containing shavings from walnuts. Similarly black walnut trees should not be voluntarily planted in horse pastures. Fallen, moldy walnuts should also be removed to prevent animals gaining access to them.

Wood shavings from bitterwood trees (*Quassia simarouba*), a tree indigenous to Central and South America, also contain irritant compounds that can cause blister- like lesions on the lips, nose, and around the eyes of horses that are bedded on the shavings. Horses that eat the shavings may also develop lesions around the anus.

Hoary Alyssum

Berteroa incana - Brassicaceae (Mustard family)

Habitat

Introduced from Europe, hoary alyssum has become established as a common weed from Nova Scotia to Washington, the northern midwestern states, and west to northern California. It is an invasive weed of disturbed soils, waste areas, and roadsides and can become troublesome in alfalfa fields.



Habitat of Hoary Alyssum. *Berteroa incana* - Brassicaceae (Mustard family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Hoary alyssum is an erect, branching, annual, growing to 3 feet (1 meter) in height. The plant is densely haired, giving it a grayish green appearance. The leaves are alternate, narrow, and lanceolate and have smooth edges. The flowers, which are produced at the ends of the branches, are white, with four deeply divided petals. The round, slightly flattened seed pods with a central septum, contain up to six brown seeds.

Principal Toxin

No specific toxin has been identified in hoary alyssum. Both green and dried plants are toxic to horses. Hay containing hoary alyssum may remain toxic for up to 9 months [49]. The quantity of plant that has to be ingested to cause poisoning has not

been determined. In reported cases of hoary alyssum poisoning, the hay being fed contained up to 90 percent of the plant depending on the bale of hay [49]. The fact that the plant can contaminate alfalfa hay makes hoary alyssum poisoning of horses possible in any state to which the hay is transported.

Clinical Signs

Lameness due to laminitis and limb edema are associated with the consumption of hoary alyssum [47,48]. Depending on the quantity of plant consumed, horses may show signs ranging from stiffness, limb swelling, fever, diarrhea, laminitis, intravascular hemolysis, severe hypovolemic shock, and death secondary to endotoxemia [51]. Pregnant mares may undergo abortion or premature parturition [48]. Horses recover if they receive no further hoary alyssum and are treated symptomatically for laminitis and shock.

Flatweed, Cat's Ears

Hypochaeris radicata - Asteraceae (Sunflower family)

Habitat

Flatweed is a perennial native weed in Europe and now prevalent in many areas of Australia and North America, where it has become established in disturbed soils and overgrazed pastures.



Habitat of Flatweed, Cat's Ears. *Hypochaeris radicata* - Asteraceae (Sunflower family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Resembling the common dandelion (*Taraxacum officinale*), flatweed has multiple, basally clustered, irregularly lobed, 3 to 12 inch (7.5 to 30 cm), hairy leaves. Multiple branched flower stalks up to 2 feet (0.5 meter) in height, each bearing a single yellow dandelion-like flower, are produced each season (Fig. 9-5). Seeds are long-beaked, roughened, and tipped by a circle of bristles.



Figure 9-5. Flatweed, cat's ears (*Hypochaeris radicata*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

No specific toxin has been identified in flatweed. Horses preferentially grazing flatweed have been reported to develop a unique lameness syndrome described in Australia as Australian stringhalt [52,53]. A similar syndrome has been reported in horses grazing flatweed in California [54]. Attempts at experimentally reproducing the disease by feeding flatweed to horses have been unsuccessful, suggesting that other factors may be involved in the pathogenesis of the disease [52,53].

The hypermetria and hyperflexion of the hind legs associated with hoary alyssum is similar to stringhalt, a well-recognized lameness of horses [55].

Clinical Signs

Horses grazing flatweed over a period of several weeks may develop lameness characterized by high stepping and hyperflexion of the hind legs similar to that described for stringhalt [54,55]. Affected horses have difficulty in stepping backward; others have such severe hyperflexion of the hind limbs when walking that the abdomen is kicked. Left laryngeal

hemiplegia (roaring) associated with the Australian stringhalt syndrome has not been encountered in horses with this syndrome in North America [52]. Unless severely and chronically affected, horses tend to recover over a period of months once they are prevented from eating further flatweed.

Phytogenic Selenium Poisoning

Historically, selenium poisoning in animals has been associated with two disease syndromes referred to as alkali disease and blind staggers [56,57]. Both diseases were assumed to be associated with the chronic ingestion of forage and crop plants that had accumulated toxic levels of selenium from the soil in which they were growing. Records from 1860 indicate that cavalry horses in South Dakota suffered from chronic weight loss, lameness due to hoof deformity, and hair loss that was referred to as alkali disease [58]. In the 1930s in South Dakota and Wyoming, selenium poisoning (selenosis) was linked to animals grazing plants containing high levels of selenium. Since then, many reviews have stated the importance of chronic selenium poisoning in livestock raised on western rangelands of the United States [59-63]. However, the prevalence of confirmed selenosis in livestock is relatively low, and the economic significance of chronic selenium poisoning (alkali disease) is difficult to determine [64].

Blind staggers is a disease of cattle and sheep characterized by aimless wandering, walking in circles, disregard for objects in their paths, loss of appetite, and blindness [57]. However, as discussed later, there is good evidence that blind staggers is not related to selenium poisoning, and is more likely associated with sulfate toxicity.

Selenium-rich soils are generally alkaline and exist in areas with low rainfall where there is minimal leaching of selenium from the soil. In North America, selenium is most abundant in the cretaceous shales and glacial deposits of the great plains [65] (Fig. 9-6). Plant uptake of selenium is variable and depends on the chemical form of selenium, soil pH, temperature, moisture, and the species and stage of plant growth [66]. Selenium in its inorganic form as selenide or selenite is minimally absorbed by plants [67,68], whereas selenates, which occur in alkaline soils, are readily available to plants [68]. Total soil selenium content is, therefore, not a reliable predictor of selenium uptake by plants.



Figure 9-6. Seleniferous soils of North America. - To view this image in full size go to the IVIS website at www.ivis.org . -

Some plants require selenium for normal growth and are capable of accumulating 10 times the amount of selenium that is present in the soil. Referred to as obligate accumulators, these plants may contain up to 10,000 ppm dry matter of selenium [69] (Table 9-1). Obligate accumulator plants with 25 ppm or more of selenium may cause acute poisoning but are generally distasteful and not eaten by animals unless they are especially hungry. Recognition of selenium-accumulating plants or indicator plants, therefore, provides a means of identifying seleniferous soils and the potential for selenium poisoning of livestock kept in these areas. Other plants known as secondary or facultative accumulators will bind selenium in its organic forms if it is present in the soil but do not require selenium for growth. Crop plants and pasture grasses growing in seleniferous soils will accumulate toxic quantities of selenium. Phytogenic selenium poisoning is likely to occur after plants containing 5 to 40 ppm selenium have been consumed for several months [69]. High selenium content of water will compound a forage selenium toxicity problem by adding to the animal's total selenium intake.

Table 9-1. Obligate Selenium Accumulator Plants	
Plants That Accumulate Selenium	
Scientific Name	Common Name
<i>Astragalus</i> (24 spp.)	Milk vetches
<i>Conopsis</i> spp.	Golden weeds
<i>Xylorhiza</i> spp.	Woody aster
<i>Stanleya pinnata</i>	Princes plume

Table 9-1. Obligate Selenium Accumulator Plants	
Secondary Selenium Accumulators	
Scientific Name	Common Name
<i>Acacia</i> spp.	Acacia
<i>Artemisia</i> spp.	Sages
<i>Aster</i> spp.	Asters
<i>Atriplex</i> spp.	Saltbush
<i>Castilleja</i> spp.	Paintbrush
<i>Penstemon</i> spp.	Beard tongue
<i>Grindelia</i> spp.	Gumweed

Toxicity

Selenium has numerous complex effects on cellular function, many of which are poorly understood. It is well known that selenium inhibits cellular enzyme oxidation reduction reactions, especially those involving sulfur-containing amino acids [70,71]. This effect of selenium on sulfur alters the metabolism of sulfur-containing amino acids (methionine, cystine) thereby affecting cell division and growth. This causes degeneration and necrosis of the cells that form keratin (keratinocytes) [72,73]. By replacing sulfur in the keratin molecule, the primary constituent of the hooves and hair, selenium weakens the keratin structurally at the site of selenium incorporation into its structure. Consequently the hair and hoof wall tend to fracture at this site when subjected to mechanical stresses.

Selenium poisoning in animals is variable and depends on the amount and rate of absorption of selenium from the intestinal tract. Horses appear to be more susceptible to chronic selenosis than are cattle and sheep [74]. Some animals such as pronghorn antelopes appear capable of consuming diets high in selenium (15 ppm) for long periods without ill effect [75]. Individual animal susceptibility, the chemical form of selenium present, and the bioavailability of selenium as a result of the interaction with other elements such as sulfur or arsenic present in the diet are also important in the pathogenesis of selenium poisoning [57]. Elevated levels of selenium in animals are also immunotoxic, possibly through the peroxidative damage of free radicals on lymphocytes [76,77].

Chronic Selenosis (Alkali Disease)

Chronic selenosis is a disease of horses, cattle, pigs, sheep, and poultry that consume forages or cereal crops grown in seleniferous soils and that have accumulated toxic levels of selenium. Plants or diets with 5 to 50 ppm selenium are most likely to cause chronic selenosis [78-83]. Animals that consume greater than 50 ppm, or are inadvertently injected with an overdose of selenium, develop acute selenium poisoning. The signs of acute selenium poisoning are quite different from those of chronic selenosis, and are characterized by sudden death from cardiac insufficiency, and pulmonary congestion and edema [77,84]. Congestion, edema, and necrosis of the lungs, liver, and kidneys are the major lesions seen at postmortem examination in acute selenium poisoning.

Clinical Signs

Although early reports of chronic selenium poisoning described a wide variety of clinical signs including lameness, hair loss, blindness, aimless wandering, and head pressing, the most distinctive lesions are those involving the keratin of the hoof and hair [59,72,85]. The long hairs of the tail and mane tend to break off at the same place giving the animal a "bob" tail and "roached" mane, respectively (Fig. 9-7A). Lameness is due to abnormal hoof wall growth in all feet, which results in rapid uneven growth, circular ridges and subsequent cracking of the hoof wall (Fig. 9-7B). Some horses may slough the hoof wall entirely. Cattle will show similar defective hoof growth but rarely lose the hoof wall. Sheep do not seem to develop as severe lesions as cattle and horses but show marked reduction in fertility when grazing on selenium-rich pastures [84]. Chronic selenium poisoning has also been associated with reduced reproductive performance, anemia, liver cirrhosis, heart atrophy, and degeneration of bones and joints in horses and cattle [57,67].



Figure 9-7A. Broken tail hairs ("bobtail") in a horse with chronic selenium poisoning. - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 9-7B. Horizontal or circular hoof wall cracks resulting from chronic selenium poisoning. - To view this image in full size go to the IVIS website at www.ivis.org . -

Diagnosis

A diagnosis of selenium poisoning is best confirmed by submitting samples of hay, forages, water, serum, and liver for analysis. Western wheat grass accumulates selenium more readily than other common grasses and is therefore useful to submit for selenium analysis [87]. Forage selenium levels greater than 5 ppm should be considered potentially toxic [88]. Blood levels of 1 to 4 ppm are indicative of chronic selenium poisoning; serum levels up to 25 ppm have been reported in acute poisoning [57,89]. Liver and kidney levels greater than 4 ppm are indicative of selenium toxicosis [74,83,90]. High tissue levels of selenium may take 6 to 12 months to return to normal after the animal has been removed from the source of the selenium [57].

In chronic selenosis, levels of selenium in the tissues may be low, but hair and hoof samples retain high concentrations. Hair and hoof wall samples collected at the site where the hair is broken and where the hoof wall is cracked are useful in determining historical levels of selenium. Hoofwall containing 8 to 20 ppm of selenium is indicative of chronic selenosis [90,91]. Similarly, hair samples containing in excess of 5 to 10 ppm selenium indicate excessive selenium levels capable of causing toxicity [57].

Treatment

Successful treatment of selenium poisoning depends on early recognition of signs and the removal of livestock from the source of the excess selenium. Recovery from chronic selenium poisoning will occur in time if the animal is fed a diet low in selenium and high in sulfur-containing amino acids. Feeding a high-protein diet with adequate copper levels counteracts the effects of selenium on sulfur-containing amino acids. Feeding good-quality alfalfa hay, provided it is low in selenium, is helpful in providing adequate sulfur to counteract selenium in the diet. Alfalfa typically has low selenium levels (0.15 - 0.69 ppm), but can accumulate toxic levels (22 ppm) when growing in selenium-rich soils [83]. Adequate levels of copper in the diet has a protective effect against selenium poisoning [92]. Attention should be given to careful and regular trimming the deformed, overgrown hooves to avoid permanent lameness.

Selenium Deficiency

Deficiencies of selenium in livestock usually coincide with geographic areas that have high annual rainfall, which tend to leach selenium from acidic top soils [92]. (Fig. 9-7B and Fig. 9-7B). In deficient soils, plants may contain less than 0.05 mg/kg of selenium, making it necessary to supplement livestock rations with selenium to prevent deficiency symptoms. Diets containing 0.1 mg/kg (0.1 ppm) of selenium are generally considered adequate for normal growth. Selenium is an essential trace mineral required by all animals for normal growth. It is an important antioxidant, preventing intracellular oxidation, cellular degeneration, and cell membrane destruction. A deficiency of selenium in the diet of animals results in muscle degeneration, a disease of livestock known as muscular dystrophy or white muscle disease [92]. As the name implies, the muscles of selenium-deficient animals undergo degeneration and characteristically develop pale or white areas, especially in

the muscles of the limbs and heart.

Blind Staggers

Blind staggers in cattle and sheep, characterized by aimless wandering, circling, disregard for objects in their paths, loss of appetite, and blindness, was reported as a form of selenium poisoning [57]. The disease is characterized by front leg weakness, staggering gait, and eventual inability to stand. Weight loss accompanies poor appetite. Teeth grinding, an indication of abdominal pain, is common. Affected animals are often blind.

Recent review of the original micrographs used to document the cause of blind staggers indicates that the tissues were compromised by autolysis that led to misinterpretation [93]. The clinical signs and tissue findings more appropriately resemble those of sulfate toxicity [93,94]. Excessive consumption of sulfate (more than 2 percent of total diet) results in toxic levels of sulfide being absorbed from the rumen, which leads to polioencephalomalacia with clinical signs typical of blind staggers. This is substantiated by recent studies on sulfate toxicity in which cattle develop clinical signs of brain disease characterized by severe depression, uncoordination, and blindness [94-96]. Contributing to the history of the syndrome of blind staggers is the association of animals that eat two-grooved milk vetch (*Astragalus bisulcatus*), a known selenium accumulator. Sheep fed two-grooved milk vetch developed classical signs of blind staggers, suggesting high levels of selenium in the plant was the cause of the problem. However, two-grooved milk vetch also contains swainsonine, the alkaloid responsible for locoism, making the neurologic signs more likely due to the swainsonine than the selenium [97-99]. Furthermore the microscopic cytoplasmic vacuolation found in the sheep's tissues were typical of those seen in locoweed poisoning and not selenium poisoning. In light of current evidence, it is appropriate to assume that selenium poisoning is not the cause of blind staggers and that sulfate toxicity is the cause of this disease syndrome. Locoweed poisoning may be a confounding issue in that clinical signs are similar to those of animals showing blind staggers.

The following plants are described in some detail as their recognition is helpful in identifying areas in which there is potential for selenium poisoning. Many of these plants are require selenium in the soil for them to flourish, thereby serving as indicator plants for selenium rich soils.

Two-Grooved Milk Vetch

Astragalus bisulcatus - Fabaceae (Legume family)

Habitat

Many of the vetches prefer the dry, alkaline seleniferous soils at lower and middle elevations especially in western North America.



Habitat of Two-Grooved Milk Vetch. *Astragalus bisulcatus* - Fabaceae (Legume family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

This leafy-stemmed, perennial plant often grows as a large clump (Fig. 9-8). The leaves are pinnate with numerous leaflets. Flowers are in dense spikelike racemes, which become reflexed in age. Flower color may vary from purple to pink or white. The fruit is a pod, from 1 cm to 1.5 cm (11 to 15 mm) long on a stipe, 3 to 4 mm in length. The pod is one-celled with two grooves running lengthwise on the upper surface of the pod, which gives the plants its common name.



Figure 9-8. A: Two-grooved milk vetch (*Astragalus bisulcatus*). B: Inset showing two distinctive grooves on seed pods. - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxins

Approximately 24 species of *Astragalus* are known to accumulate toxic levels of selenium [2], (Table 9 - 2). Livestock will rarely eat these plants except under starvation conditions apparently because of their distasteful seleniferous odor. These

Astragalus spp. are useful indicator plants for soils high in selenium, and their presence can help identify pastures in which all plants may accumulate potentially toxic levels of selenium. Two-grooved milk vetch is one of the few known vetches to contain both selenium and swainsonine, the alkaloid responsible for locoism.

Table 9 - 2. Selenium-Accumulating Astragalus Species
<i>A. albulus</i>
<i>A. argillosus</i>
<i>A. beathii</i>
<i>A. bisulcatus</i>
<i>A. confertiflorus</i>
<i>A. crotalariae</i>
<i>A. diholcos</i>
<i>A. eastwoodae</i>
<i>A. ellisiae</i>
<i>A. grayi</i>
<i>A. haydenianus</i>
<i>A. moencoppensis</i>
<i>A. oocalycis</i>
<i>A. osterhoutii</i>
<i>A. pattersonii</i>
<i>A. pectinatus</i>
<i>A. praelongus</i>
<i>A. preussii</i>
<i>A. racemosus</i>
<i>A. recedens</i>
<i>A. sabulosus</i>
<i>A. toanu</i>

Rayless Goldenweed

(Oenopsis engelmannii) - Asteraceae (Sunflower family)

Habitat

Goldenweed prefers the dry, alkaline soils of the plains and foothills of central and southwest North America.



Habitat of Rayless Goldenweed. (*Oenopsis engelmannii*) - Asteraceae (Sunflower family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Goldenweed is a perennial shrub with a woody root stock. The stems are from 4 to 12 inches (10 to 30 cm) tall, have brown bark below, and are glabrous. The leaves are 1 to 2.75 inches (3 to 7 cm) long and 1 to 3 mm wide, narrowly linear, rigidly erect, and glabrous. The heads are few to many, with bracts imbricated in three or more lengths, somewhat oblong to lanceolate. The color of the disc flowers is yellowish, and the pappus is brown. There are no ray flowers. *Oenopsis foliosa* var. *monocephal* is similar to this species, except that it has longer involucre bracts, oblong lanceolate leaves which are over 6 mm wide, and usually one head to a stem. *O. foliosa* differs from the preceding species in that it has ray flowers 8 to 15 mm long and has broadly lanceolate leaves, with one to several flower heads per stem.

Principal Toxin

Jimmy weed may accumulate toxic levels of selenium that can induce chronic selenium poisoning in livestock grazing it and other forages in the area.

Woody Aster

Xylorrhiza glabriuscula (*Machaeranthera*) - Asteraceae (Sunflower family)

Habitat

Woody aster requires the alkaline seleniferous soils of western North America, often growing at altitudes of 5000 to 6500 feet (1,524 to 1,981 meters).



Habitat of Woody Aster. *Xylorrhiza glabriuscula* (*Machaeranthera*) - Asteraceae (Sunflower family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Woody aster is a low shrubby plant with a thick taproot and woody branching base. The leaves are 1 to 2 inches (2 to 5 cm) long and 2 to 6 mm wide. They are linear-oblongate to linear, hairy, and tipped with a callus point. The ray flowers are stiff and white (Fig. 9-9). *X. venusta* (*Machaeranthera* spp., old name) resembles the preceding with the exception that the involucre is longer and the disc is somewhat wider.



Figure 9-9. Woody aster (*Xylorrhiza glabriuscula*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Woody aster is an obligate selenium accumulator and may cause severe chronic selenium poisoning in livestock that are forced into grazing the plant when other forages are scarce.

Prince's Plume

Stanleya pinnata - Brassicaceae (Mustard family)

Habitat

Prince's plume requires the dry alkaline, selenium rich soils and shale rock formations of hills, valleys, and arroyo banks of western North America.



Habitat of Prince's Plume. *Stanleya pinnata* - Brassicaceae (Mustard family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

The plant is a coarse, herbaceous perennial, ranging from 1.5 to 5 feet (0.5 to 2 meters) in height. The stems are stout and mostly unbranched. The leaves are entire to pinnately compound, from 2 to 8 inches (5 to 20 cm) long. Many small flowers with yellow petals with long claws are arranged in a plume-like inflorescence (Fig. 9-10). The fruit is slender pod, nearly round in cross section, and with a stalk from 1 to 3 cm long.



Figure 9-10. Prince's plume (*Stanleya pinnata*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Prince's plume is an obligate selenium-accumulating plant capable of concentrating high levels of selenium. It is relatively unpalatable and rarely eaten by livestock. However, the presence of prince's plume is indicative of high selenium content in the soil, and other grasses and plants growing in the same area may also accumulate selenium that could cause chronic selenium poisoning in livestock.

White Fall Aster, Rough White Aster

Aster falcatus - Asteraceae (Sunflower family)

Habitat

Rough white aster is a fall blooming plant of the prairies and foothills of central and western North America.



Habitat of White Fall Aster. *Aster falcatus* - Asteraceae (Sunflower family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

White fall aster is a branched perennial plant with extensive root stalks. The leaves are 1 to 3 cm long, linear, and densely hairy. The inflorescence is a head with white ray flowers 3 to 4 mm long with many tawny pappus bristles (Fig. 9-11).



Figure 9-11. White fall aster (*Aster falcatus*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

White fall aster is a secondary or facultative selenium accumulator when growing in alkaline, selenium-rich soils. It may, therefore, cause chronic selenium poisoning of livestock.

Broom Snakeweed

Gutierrezia sarothrae - Asteraceae (Sunflower family)

Habitat

Broom snakeweed is a common inhabitant of the dry plains and hills of western North America, usually growing at altitudes from 4000 to 10,000 feet (1,219 to 3,048 meters).

Description

This is a herbaceous perennial that is shrubby and woody at its base. The stems are branching; the leaves linear and glabrous. The heads are many, usually in clusters at the ends of the branches. A given head will have no more than three to eight ray flowers and three to eight disc flowers. The flowers are yellow, with the disc flowers usually perfect (see Fig. 8-5A and Fig. 8-5B). The corollas have five lobes. The pappus is composed of several to many oblong scales.

Principal Toxin

The plant is a secondary selenium absorber. It has also been associated with abortion in cattle and sheep and may cause fatal liver disease (see Chapter 8).

Gumweed, Resin Weed

Grindelia spp. - Asteraceae (Sunflower family)

Habitat

Gumweed is common on the prairies, plains, roadsides and waste areas.



Habitat of Gumweed. *Grindelia* spp. - Asteraceae (Sunflower family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Gumweeds are biennial or perennial herbaceous plants with leafy stems. The leaves are alternate, simple, and more or less resinous dotted. The heads are solitary, with the bracts being imbricated in several series with the tips often recurved and covered with a very gummy resinous material (Fig. 9-12). The ray flowers, when present, are 8 to 10 mm long, lemon-yellow to bright yellow in color; the pappus awns are 2 to 3 mm long.



Figure 9-12. Gumweed (*Grindellia squarosa*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Gumweeds will accumulate selenium if growing in alkaline, selenium-rich soils, and therefore may cause chronic selenium poisoning in livestock. Horses frequently like to eat the flower heads.

Saltbush

Atriplex spp.

A. canescens - four-wing saltbush Chenopodiaceae (Goosefoot family)

Habitat

Saltbush is common on the dry plains and foothills of western North America.



Habitat of Saltbush. *Atriplex* spp. - Chenopodiaceae (Goosefoot family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

Saltbush is a perennial shrub growing to 6 feet (2 meters) in height that is more or less scaly and scurfy. The leaves are mostly alternate, often covered by a white powdery substance. The plants contain both male and female flowers, with staminate flowers in terminal panicles without bracts. They have a three- to five-parted perianth and three to five stamens. The pistillate flowers are subtended by two bracts, but are without a perianth. The ovary is one-celled with two stigmas. Four-wing saltbush has four distinct wings (bracts) to the seed capsule (Fig. 9-13).



Figure 9-13. Saltbush showing 4 wings (bracts) of the fruits (*Atriplex* spp.). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Saltbush will accumulate selenium when growing in selenium-rich soils and may cause chronic selenium poisoning of livestock under such growing conditions. Otherwise, the plant is considered a valuable range plant for grazing.

Saltbush Poisoning

Saltbush is also the name given to *Baccharis halimifolia*, a common shrub that grows in the coastal plains of Virginia, and from Florida to Texas. The plant is not a selenium accumulator but contains cardiotoxic glycosides. Cattle rarely eat saltbush unless other forages are scarce.

Indian Paintbrush

Castilleja spp. - Scrophulariaceae (Figwort family)

Habitat

Paintbrushes are widespread in the mountains and plains of western North America.



Habitat of Indian Paintbrush. *Castilleja* spp. - Scrophulariaceae (Figwort family). - To view this image in full size go to the IVIS website at www.ivis.org . -

Description

These perennial and occasionally annual plants are herbaceous, but many are woody at the base. The leaves are alternate and sessile. The irregular flowers are arranged in terminal, bracted spikes. The bracts are usually petal-like ranging from scarlet to yellow in color (Fig. 9-14). The calyx is tubular and four-lobed, more deeply cleft above and below than on the sides. The corolla is long and narrow, strongly two-lipped; the upper, called a galea (hood), is elongated and the lower lip is very short and three-toothed. The stamens number four, in pairs, and are enclosed by the galea. Paintbrush attaches to the roots of surrounding plants (sage species) in a symbiotic relationship.



Figure 9-14. Paintbrush (*Castilleja* spp.). - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Indian paintbrush species are selenium accumulators if growing in high selenium soils.

Beard Tongue

Penstemon spp. - Scrophulariaceae (Figwort family)

Habitat

Many different species of *Penstemon* are widespread in the mountains and plains of North America. Their habitat varies according to species preferences.

Description

These are usually herbaceous perennial plants that are erect and tufted; however, many are low and creeping. The leaves are opposite with the upper one sessile and often clasping. The inflorescence is a panicle of flowers ranging from blue to red (Fig. 9-15). The flowers are showy with a tubular corolla, which is bilabiate with the upper lip two-lobed and the lower lip three-lobed. There are four fertile stamens in two pairs with arched filaments. A fifth stamen, called a staminode, is represented by conspicuous sterile filament attached to the upper side of the corolla. It is widened and bearded at the apex, giving the plants their common name of beard tongue.



Figure 9-15. *Penstemon* (*Penstemon* spp.). Inset shows flower details. - To view this image in full size go to the IVIS website at www.ivis.org . -

Principal Toxin

Penstemon spp. will accumulate selenium if growing in selenium-rich soil. It is seldom a problem to livestock.

References

1. Dolahite JW, Henson JB. Toxic plants as the etiologic agent of myopathies in animals. *Am J Vet Res* 1965; 26:749-752.
2. Radostits OM, Blood DC, Gay CC, eds. *Veterinary Medicine*, ed 8. Philadelphia: Bailliere Tindall 1994; 1543-1544.
3. Krook L, Wasserman RH, Shively JN, et al. Hypercalcemia and calcinosis in Florida horses: implication of the shrub, *Cestrum diurnum*, as the causative agent. *Cornell Vet* 1975; 65:26-56.
4. Weisenberg M. Calcinogenic glycosides. In: Cheeke PR ed. *Toxicants of Plant Origin*, Vol II. Boca Raton: CRC Press 1989; 201-238.
5. Morris KML. Plant induced calcinosis: a review. *Vet Hum Toxicol* 1982; 24:34-48.
6. Kasali OB, Krook L, Pond WG, Wasserman RH. *Cestrum diurnum* intoxication in normal and hyperparathyroid pigs. *Cornell Vet* 1977; 67:190-221.
7. Wasserman RH. The nature and mechanism of action of the calcinogenic principle of *Solanum malacoxylon* and *Cestrum diurnum*, and a comment on *Trisetum flavescens*. In: Keeler RF, Van Kampen KR, James L, Eds. *Effects of Poisonous Plants on Livestock*. New York: Academic Press 1978; 545-553.
8. Krook L, Wasserman RH, McEntee K, et al. *Cestrum diurnum* poisoning in Florida cattle. *Cornell Vet* 1975; 65:557-575.
9. Krook L, Wasserman RH, Shively JH, et al. Hypercalcemia and calcinosis in Florida horses: implication of the shrub *Cestrum diurnum*, as the causative agent. *Cornell Vet* 1975; 65:26-56.
10. Allen JG, Steele P, Masters HG, Lambe WJ. A lupinosis-associated myopathy in sheep and the effectiveness of treatments to prevent it. *Aust Vet J* 1992; 69:75-81.
11. Williamson PM, Hight AS, Gams W, et al. *Diaporthe toxica* sp. nov., the cause of lupinosis in sheep. *Mycological Res* 1994; 98:1364-1368.
12. Hebert CD, Flory W, Seger C, Blanchard RE. Preliminary isolation of a myode- generative toxic principle from *Cassia occidentalis*. *Am J Vet Res* 1983; 44:1370-1374.
13. Rowe LD, Corrier DE, Reagor JC, Jones LP. Experimentally induced *Cassia roemeriana* poisoning in cattle and goats.

Am J Vet Res 1987; 48: 992-997.

14. Rowe LD. Cassia-induced myopathy. In: Keeler RF, Tu AT, Eds. Handbook of Natural Toxins. Toxicology of Plant and Fungal Toxins. New York: Marcel Dekker 1991; 335-351.

15. O'Hara PJ, Pierce KR, Reid WK. Degenerative myopathy associated with ingestion of *Cassia occidentalis*: clinical and pathologic features of the experimentally induced disease. Am J Vet Res 1969; 30:2173-2180.

16. O'Hara PJ, Pierce KR. A toxic cardiomyopathy caused by *Cassia occidentalis* II. Morphological studies in poisoned rabbits. Vet Pathol 1974; 11:97-109.

17. Mercer HD, Neal FC, Himes JA, et al. *Cassia occidentalis* toxicosis in cattle. J Am Vet Med Assoc 1967; 151:735-741.

18. Pierce KR, O'Hara PJ. Toxic myopathy in Texas cattle. Southwest Vet 1967; 20:179-184.

19. Martin BW, Terry MK. Toxicity of *Cassia occidentalis* in the horse. Vet Hum Toxicol 1981; 23: 416-418.

20. Henson JB, Dolomite JW, Bridges CH, et al. Myodegeneration in cattle grazing Cassia species. J Am Vet Med Assoc 1965; 147:142-145.

21. Rogers RJ, Gibson J, Reichman KG. The toxicity of *Cassia occidentalis* for cattle. Aust Vet J 1979; 55:408-412.

22. Colvin BM, Harrison LR, Sangster LT, Gosser HS. *Cassia occidentalis* toxicosis in pigs. J Am Vet Med Assoc 1986; 189:423-426.

23. Suliman HB, Wasfi AI, Adam SEI. The toxicity of *Cassia occidentalis* in goats. Vet Hum Toxicol 1982; 24:326-329.

24. Galal M, Adam SEI, Maglad MA, et al. The effects of *Cassia occidentalis* on goats and sheep. Acta Vet 1985; 35: 163-174.

25. Graziano MJ, Flory W, Seger CL, et al. Effects of *Cassia occidentalis* extract in the domestic chicken (*Gallus domestica*). Am J Vet Res 1983; 44:1238-1244.

26. O'Hara PJ, Pierce KR. A toxic cardiomyopathy caused by *Cassia occidentalis* II. Biochemical studies in poisoned rabbits. Vet Pathol 1974; 11:110-124.

27. Suliman HB, Shommein AM. Toxic effect of the roasted and unroasted beans of *Cassia occidentalis* in goats. Vet Hum Toxicol 1986; 28:6-11.

28. Nicholson SS, Thorton JT, Rimes AJ. Toxic myopathy in dairy cattle caused by *Cassia obtusifolia* in green-chop. Bovine Practitioner 1977; 12:120-123.

29. Putnam MR, Boosinger T, Spano J, et al. Evaluation of *Cassia obtusifolia* (sickle pod) seed consumption in Holstein calves. Vet Hum Toxicol 1988; 30:316-318.

30. Burrows GE, Edwards WC, Tyrl RJ. Toxic plants of Oklahoma: coffeeweeds and sennas. Oklahoma Vet Med Assoc 1989; 34:101-105.

31. Henson JB, Dolomite JW. Toxic myodegeneration in calves produced by experimental *Cassia occidentalis* intoxication. Am J Vet Res 1966; 27:947-949.

32. Schmitz DG, Denton JH. Senna bean toxicity in cattle. Southwest Vet 1977; 30:165-170.

Thermopsis (Yellow Banner)

33. Keeler RF, Johnson AE, Chase RL. Toxicity of *Thermopsis montana* in cattle. Cornell Vet 1986; 76:115-127.

34. Keeler RF, Baker DC. Myopathy in cattle induced by alkaloid extracts from *Thermopsis montana*, *Laburnum anagyroides*, and *Lupinus* spp. J Comp Pathol 1990; 103:169-182.

35. Baker DC, Keeler RF. Myopathy in cattle caused by *Thermopsis montana*. In: Keeler RF, Tu AT, Eds. Handbook of Natural Toxins. Toxicology of Plant and Fungal Toxins. New York: Marcel Dekker 1991; 61-69.

36. Spoerke DG, Murphy MM, Wruk KM, Rumack BH. Five cases of Thermopsis poisoning. Clin Toxicol 1988; 26:397-406.

37. Baker DC, Keeler RF. Thermopsis montana-induced myopathy in calves. J Am Vet Med Assoc 1989; 194:1269-1272.

Black Walnut

38. Uhlinger C. Black walnut toxicosis in ten horses. J Am Vet Med Assoc 1989; 195:343-344.

39. True RG, Lowe JE. Induced juglone toxicosis in ponies and horses. Am J Vet Res 1980; 41:944-945.

40. Ralston SL, Rich VA. Black walnut toxicosis in horses. J Am Vet Med Assoc 1983; 183:1095.

41. MacDaniels LH. Perspective on the black walnut toxicity problem - apparent allergies to man and horse. Cornell Vet 1983; 73:204-207.

42. Minnick PD, Brown CM, Braselton WE, et al. The induction of equine laminitis with an aqueous extract of the heartwood of black walnut (*Juglans nigra*). Vet Hum Toxicol 1987; 29:230-233.

43. MacDaniels LH. Perspective on the black walnut toxicity problem. Cornell Vet 1987; 29:230-233.

44. Galey FD, Beasley VR, Schaeffer D, Davis LE. Effect of an aqueous extract of black walnut (*Juglans nigra*) on isolated equine digital vessels. Am J Vet Res 1990; 51:83-88.

45. Galey FD, Twardock AR, Goetz TE, et al. Gamma scintigraphic analysis of the distribution of perfusion of blood in the equine foot during black walnut (*Juglans nigra*)- induced laminitis. Am J Vet Res 1990; 51:688-695.

46. True RG, Lowe JE, Heissen J. Black walnut shavings as a cause of laminitis. Proc Annu Meet Am Assoc Equine Pract

1978; 24:511-515.

47. Galey FD, Beasley VR, Twardock AR, Whiteley HE, et al. Pathophysiologic effects an aqueous extract of black walnut (*Juglans nigra*) when administered via nasogastric tube to the horse. In: James LF, Keeler R, Bailey EM, et al. Eds. Poisonous Plants Proceeding of the Third International Symposium. Ames: Iowa State University Press 1992; 630-635.

48. Richard JL, Arp LH, Bachetti P. The mycotoxin, penitrem A, as a cause of moldy walnut toxicosis in a dog. California Vet 1981; 6:12.

Hoary Alyssum

49. Geor RJ, Becker RL, Kanara EW et al. Toxicosis in horses after ingestion of hoary alyssum. J Am Vet Med Assoc 1992; 201:63-67.

50. Ellison SP. Possible toxicity caused by hoary alyssum (*Berteroa incana*). Vet Med/ Equine Practice 1992; 472-475.

51. Hovda LR, Rose ML. Hoary alyssum (*Berteroa incana*) toxicity in a herd of brood mares. Vet Human Toxicol 1993; 35: 39-40.

Flatweed

52. Huntington PJ, Jeffcott LB, Friend SCE, et al. Australian-stringhalt - epidemiological, clinical, and neurological investigations. Equine Vet J 1989; 21:266-273.

53. Pemberton DH, Caple IW. Australian-stringhalt in horses. Vet Annu 1980; 20:167-171.

54. Galey FD, Hullinger PJ, McCaskill J. Outbreaks of stringhalt in northern California. Vet Hum Toxicol 1991; 33:176-177.

55. Stashak TS. Lameness. In: Stashak TS., Ed. Adams' Lameness in Horses. Philadelphia: Lea & Febiger 1987; 723-725.

Selenium

56. Trelease SF, Beath OA. Selenium: Its Geological Occurrence and Its Biological Effects in Relation to Botany, Chemistry, Agriculture, and Medicine. Burlington, VT: Champlain Printers 1949; 1-12, 165-187.

57. Rosenfeld I, Beath OA. Selenium: Geobotany, Biochemistry, Toxicity and Nutrition. New York: Academic Press 1964; 1-7, 91-104, 141-163.

58. Durrell LW, Cross F. Selenium poisoning of livestock. Colorado State College, Extension Service Bulletin 1944;382-A.

59. Radostits OM, Blood DC, Gay CC, eds. Diseases caused by inorganic and farm chemicals - selenium poisoning. In: Veterinary Medicine, edn 8. London: Bailiere Tindall 1994; 1484-1486.

60. James LF, Panter KE, Mayland HF, et al. Selenium poisoning in livestock: a review and progress. In: Jacobs LW, Ed. Selenium in Agriculture and the Environment. Madison, Wisc: Am Soc Agron Soil Sci Soc of America; 1989; 23:123-131.

61. Whanger PD. Selenocompounds in plants and their effects on animals. In: Cheeke PR, Ed. Toxicants of Plant Origin, Vol III. Proteins and Amino Acids. Boca Raton: CRC Press 1989; 141-167.

62. James LF, Smart RA, Shupe JL, et al. Suspected phytogenic selenium poisoning in sheep. J Am Vet Med Assoc 1982; 180:1478-1481.

63. Edmondson AJ, Norman BB, Suther D. Survey of state veterinarians and state veterinary diagnostic laboratories for selenium deficiency and toxicosis in animals. J Am Vet Med Assoc 1993; 202:865-872.

64. Raisbeck MF, Dahl ER, Sanchez DA, et al. Naturally occurring selenosis in Wyoming. J Vet Diagn Invest 1993; 5:84-87.

65. Kubota J, Allaway WH. Geographic distribution of trace element problems. In: Dinauer RC, Ed. Micronutrients in Agriculture. Proceedings Madison, Wisc. Soil Science Society of America 1972; 525-554.

66. Meyer RD, Burau RB. The geochemistry and biogeochemistry of selenium in relation to its deficiency and toxicity in animals. Selenium in the environment: essential nutrient, potential toxicant. In: Proceedings of the University California, Division of Agriculture and Natural Resources. 1995; 38-44.

67. Ganje RJ, Whitehead EI. Selenium uptake by plants as affected by forms of selenium in the soil. Proc South Dakota Acad Sci 1958; 37:85-88.

68. Olson OE, Whitehead EI, Moxon AI. Occurrence of soluble selenium in soils and its availability to plants. Soil Sci 1942; 54:47-53.

69. Olson OE. Selenium in plants as a cause of livestock poisoning. In: Keeler RF, Van Kampen KR, James LF, Eds. Effects of Poisonous Plants on Livestock. New York: Academic Press 1978; 121-133.

70. Shrift A. Metabolism of selenium by plants and microorganisms. In: Klayman DJ, Gunther WHH, Eds. Organic Selenium Compounds: Their Chemistry and Biology. New York: John Wiley & Sons 1973; 693-726.

71. Underwood EJ, Ed. Trace Elements in Human and Animal Nutrition. New York: Academic Press 1977; 334-346.

72. O'Toole D, Raisbeck MF. Pathology of experimentally induced chronic selenosis (alkali disease) in yearling cattle. J Vet Diagn Invest 1995; 7:364-373.

73. Raisbeck MF, O'Toole D. Morphologic studies of selenosis in herbivores. In: Garland T, Barr AC, Eds. Toxic Plants and Other Natural Toxicants. New York: CAB International 1998; 380-388.

74. Crinion RA, O'Connor JP. Selenium intoxication in horses. Irish Vet J 1978; 32: 81-86.

75. Raisbeck MF, O'Toole D, Schamber RA, et al. Toxicologic evaluation of a high- selenium hay diet in captive pronghorn

- antelope (*Antilocapra americana*). J Wildlife Dis 1996; 32:9-16.
76. Raisbeck MF, Schamber RA, Belden EL. Immunotoxic effects of selenium in mammals. In: Garland T, Barr AC, Eds. Toxic Plants and Other Natural Toxicants. New York: CAB International 1998; 260-266.
 77. Smyth JBA, Wang JH, Barlow RM, et al. Experimental acute selenium intoxication in lambs. J Comp Pathol 1990; 102:199-209.
 78. Moxon AL. Alkali disease or selenium poisoning. South Dakota Agricultural Experimental Station Technical Bulletin 1937; 311:1-91.
 79. Flemming GA, Walsh T. Selenium occurrence in certain soils and its toxic effects on animals. Proc Royal Irish Acad 1957; 58:151-167.
 80. Knott SG, McCray CWR. Two naturally occurring outbreaks of selenosis in Queensland. Aust Vet J 1959; 35:161-165.
 81. Olsen OE, Embry LB. Chronic selenite toxicity in cattle. Proc South Dakota Acad Sci 1973; 52:50-58.
 82. Raisbeck MF, Dahl ER, Sanchez DA, et al. Naturally occurring selenosis in Wyoming. J Vet Diagn Invest 1993; 5:84-87.
 83. Witte ST, Will LA, Olsen CR, Kinker JA. Chronic selenosis in horses fed locally produced alfalfa hay. J Am Vet Med Assoc 1993; 202:406-409.
 84. James LF, Smart RA, Shupe JL, et al. Suspected phyto-genic selenium poisoning in sheep. J Am Vet Med Assoc 1982; 180:1478-1481.
 85. Raisbeck MF, O'Toole D, Belden EL, Waggoner JW. Chronic selenosis in ruminants. In: Garland T, Barr AC, Eds. Toxic Plants and Other Natural Toxicants. New York: CAB International 1998; 389-396.
 86. Glenn MW, Jensen R, Griner LA. Sodium selenate toxicosis: pathology and pathogenesis of sodium selenate toxicosis in sheep. Am J Vet Res 1964;25:1486-1494.
 87. Palmer IS. Water, soil, and plant selenium: analytical methodology. In: Selenium in the Environment: Essential Nutrient, Potential Toxicant. In: Proceedings of the University California, Division of Agriculture and Natural Resources 1995; 20-35.
 88. National Research Council. Selenium in Nutrition. Washington, DC: National Academy Press; 1983.
 89. Traub-Dargatz, Knight AP, Hamar DW. Selenium toxicity in horses. Compend Contin Educ Pract Vet 1986; 8:771-776.
 90. Osweiler GD, Carson TL, Buck WB, Van Gelder GA. Selenium. In: Osweiler GD, Carson TL, Buck WB, Van Gelder GA Eds. Clinical and Diagnostic Veterinary Toxicology, ed 3. Dubuque: Kendall Hunt Publishing 1985; 132-142.
 91. Hutline JD, Mount ME, Easley KJ, Oehme FW. Selenium toxicosis in the horse. Equine Pract 1979;1:57-60.
 92. Radostits OM, Blood DC, Gay CC, Eds. Diseases caused by inorganic and farm chemicals - selenium poisoning. In: Veterinary Medicine, edn. 8. London: Bailliere Tindall 1994; 1408-1425.
 93. O'Toole D, Raisbeck MF, Case JC, Whitson TD. Selenium-induced "blind staggers" and related myths. Commentary. Vet Pathol 1996; 33:104-116.
 94. Gould DH, McAllister MM, Savage JC, Hamar DW. High sulfide concentrations in rumen fluid associate with nutritionally induced polioencephalomalacia in calves. Am J Vet Res 1991; 52:1164-1169.
 95. Raisbeck MF. Is polioencephalomalacia associated with high sulfate diets? J Am Vet Med Assoc 1982; 180:1303-1305.
 96. Jeffrey M, Duff JP, Higgins RJ, et al. Polioencephalomalacia associated with the ingestion of ammonium sulfate by sheep and cattle. Vet Rec 1994; 134:343-348.
 97. Van Kampen KR, James LF. Manifestations of intoxication by selenium accumulating plants. In: Keeler RF, Van Kampen KR, James LF, Eds. Effects of Poisonous Plants on Livestock. New York: Academic Press 1978; 135-138.
 98. James LF, Van Kampen KR, Hartley WJ. *Astragalus bisulcatus*: a cause of selenium or locoweed poisoning. Vet Hum Toxicol 1983; 25:86-89.
 99. James LF, Hartly WJ, Van Kampen KR. Syndromes of Astragalus poisoning in livestock. J Am Vet Med Assoc 1981;178:146-150.

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