Poisonous Plants Affecting Cattle in Central-Western Brazil

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Abstract

Poisonous plants affecting cattle in the Brazilian Central-Western region are reviewed. The most important poisonous plants in the region are Brachiaria spp., which cause hepatogenous photosensitization, and Palicourea marcgravii and Mascagnia pubiflora, which cause sudden death precipitated by exercise. Enterolobium contortisiliquum, Stryphnodendron obovatum, and Stryphnodendron fissuratum are trees whose pods cause digestive signs, photosensitization, and abortion. Vernonia mollissima and V. rubricaulis cause acute liver necrosis, but V. rubricaulis is much more important than V. mollissima as a cause of cattle losses. Other less important plants are Solanum glaucophyllum, which causes soft tissue calcification; Ipomoea carnea subsp. fistulosa, which causes nervous signs; Senna occidentalis and Senna obtusifolia, which cause segmental muscular necrosis; Tetrapterys multiglandulosa, which causes cardiac fibrosis, abortion, and neonatal mortality; Polygala klotzschii, which causes lymphatic tissue necrosis; and Pterodon emarginatus, a tree whose leaves cause liver necrosis. Rare outbreaks of poisoning by Lantana tiliae folia, Cestrum laevigatum, Pteridium caudatum, and Crotalaria spp. also are observed.

Keywords: Brachiaria spp., Enterolobium contortisiliquum, Mascagnia pubiflora, Palicourea marcgravii, Stryphnodendron spp., Vernonia rubricaulis

Introduction

The Central-Western (Centro-Oeste) region of Brazil (figure 1), the most important area for cattle production in the country, is composed of the states of Goiás, Mato Grosso, and Mato Grosso do Sul and the Federal District, where Brasilia, the Brazilian capital, is located. The Central-Western region represents 18.86 percent of the Brazilian territory. The region is formed by three different biomes: Cerrado, Pantanal, and, in northern Mato Grosso, the Amazon rainforest. Similar to savannas, the Cerrado originally covered the entire area of Goiás and large parts of Mato Grosso and Mato Grosso do Sul. The Pantanal, the world’s largest plain covered with water, is a tropical wetland that lies mainly in Mato Grosso do Sul and extends into Mato Grosso. The climate is humid subtropical and tropical with annual rainfall of around 800 to 1,600 mm and a rainy season from October to March with a dry
season for the rest of the year. The soils are generally very old, chemically poor, and deep. Large areas of the Cerrado and the Amazon rainforest have been deforested for agriculture (soybean, cotton, sunflower, and other crops) and for the cultivation of pastures (mainly Brachiaria spp. and Panicum maximum) for cattle production. The cattle industry is very important in the Central-Western region of Brazil, with nearly 70 million head of mainly beef cattle, which represents 36 percent of the Brazilian cattle population. The objective of this paper is to review toxic plants affecting cattle in the Brazilian Central-Western region.

Figure 1. Map of Brazil showing the different regions, Brasília (★), and the states of Goiás, Mato Grosso, and Mato Grosso do Sul.

**Brachiaria** spp.

Brachiaria spp. (Poaceae) are the most important toxic plants in the Brazilian Central-Western region. *Brachiaria decumbens* cv. Ipean, originally from Africa, was first introduced to Brazil in 1962. In 1972, *B. decumbens* cv. brasilisk was introduced from Australia and spread rapidly throughout the region (Seiffert 1980). *B. brizantha* cv. Marandú, which has gradually replaced *B. decumbens*, was introduced in the 1980s. *B. humidicola* was introduced from Africa in 1983. Other species of *Brachiaria* include *B. purpurascens*, *B. dictyoneura*, *B. ruziziensis*, *B. radicans*, *B. extensa*, *B. plantaginea*, *B. dura*, and *B. milliformis* (Seifert 1980). There are about 60 million hectares of cultivated pastures in the Cerrado. Of this total, 51 million hectares consist of *Brachiaria* spp., approximately 30 million hectares of *B. brizantha*, 15 million hectares of *B. decumbens*, and 6 million hectares of *B. humidicola* and other *Brachiaria* (Macedo 2005). Most outbreaks of poisoning are caused by *B. decumbens* (Camargo et al. 1976; Döbereiner et al. 1976b; Nobre and Andrade 1976; Fagliari et al. 1983, 1993b; Lemos et al. 1996, 1997; Brum et al. 2007; Mustafa 2009; Souza et al. 2010). Outbreaks caused by *B. brizantha* (Mustafa 2009, Lemos et al. 2011, Riet-Correa et al. 2011) are less frequent. Few outbreaks have been reported from *B. ruziziensis* (Nazário et al. 1985, Purchio et al. 1988) but this species is rarely used in pastures of the region. Poisoning by *B. humidicola* has been reported in sheep (Schenk and Schenk 1983), buffalo (Láu 1990), and horses (Barbosa et al. 2006) but not in cattle.

The toxicity of *Brachiaria* spp. is due to lithogenic steroidal saponins, protodioscin being the main saponin found in the different species (Brum et al. 2007, 2009; Santos Jr., 2008; Mustafa 2009; Castro et al. 2011). A toxicity threshold is yet to be established, but for sheep, it was suggested that pastures with saponin concentrations of more than 1 percent cause toxicity in naive sheep (Riet-Correa et al. 2011). There is no information on saponin concentrations in pastures causing poisoning in cattle. In general, saponin concentrations are higher in growing plants, but outbreaks occur throughout the year, probably due to unexplained rises in saponin concentrations in the plant. Most data show that sprouting pastures have higher saponin concentrations than mature ones (Santos Jr. 2008, Castro et al. 2011), and green leaves contain more saponin than mature or senescent leaves (Barbosa-Ferreira et al. 2011).

*Brachiaria* poisoning has been reported in cattle, sheep, and goats. Sheep are more susceptible to poisoning than cattle (Riet-Correa et al. 2009, Lemos et al. 2011). Young cattle up to 2 years of age are more frequently affected, but adult cattle may also be affected. The poisoning occurs mainly in calves near weaning or just weaned (Nobre and Andrade 1976, Fagliari et al. 1993a, Souza et al. 2010), but suckling animals up to 30 days old also are affected (Fagliari et al. 1983, Lemos et al. 1996).

Castro et al. (2011) demonstrated that there are differences in susceptibility between animals of the same species. Sheep raised on *Brachiaria* spp. pastures are less susceptible to poisoning than naive sheep raised on other pastures (Castro et al. 2011), and it has been suggested that resistance is genetic (Riet-Correa et al. 2011). In cattle, *Brachiaria* spp. poisoning was more frequent in the years after the introduction of *B. decumbens* (1960 to 1980). Subsequently, outbreaks were less frequent, apparently due to the death of susceptible animals or to the development of poisoning resistance. Another possibility is that the decrease in the number of...
outbreaks is a result of the replacement of *B. decumbens*, which is more toxic, by the less toxic *B. brizantha* and *B. humidicola* (Castro et al. 2011, Riet-Correa et al. 2011). It has also been suggested that buffalo and probably some sheep are resilient, i.e., when poisoned these animals show histologic lesions and high GGT serum concentrations but they do not show clinical signs (Saturnino et al. 2010, Castro et al. 2011, Riet-Correa et al. 2011).

Poisoning can occur at any time of the year and at any stage of the plant growth (Castro et al. 2011, Souza et al. 2011), but some authors mention a higher frequency of the poisoning during pasture sprouting (Riet-Correa et al. 2009, Castro et al. 2011). Frequency of the poisoning varies; in 29 cattle outbreaks in Mato Grosso do Sul, the morbidity rate ranged from 0.2 to 50 percent, and the fatality rate from 44.4 to 100 percent (Souza et al. 2010).

In cattle, main clinical signs are hepatogenous photosensitization with dermatitis mainly on the muzzle, ears, flanks, perineum, udder, and in areas of white skin. More acute cases show edema of the brisket and ears or other body parts, and a common finding in chronic cases is thickening and scar retraction of the ears, which are deformed and crooked (Fagliari et al. 1993b, Lemos et al. 1997, Souza et al. 2010). A clinical condition characterized by progressive weight loss and death after a few months without photosensitization has been reported in cattle over 2 years of age grazing *B. decumbens* (Riet-Correa et al. 2002, Souza et al. 2010).

In cattle, a subclinical disease characterized by lower productivity has also been reported (Fagliari et al. 1993b, Fioravanti 1999, Moreira et al. 2009a). However, Castro et al. (2011) found no significant differences in weight gains when young sheep raised on *Brachiaria* pastures were introduced to *B. brizantha, B. decumbens,* or *Panicum maximum* pastures. Sheep introduced to an *Andropogon gayanus* pasture showed lower weight gain than the others. In the same experiment naïve sheep introduced to *B. decumbens* pastures showed clinical signs of poisoning.

Increased serum activities of gamma glutamyl transferase (GGT) and aspartate aminotransferase (AST) and increased serum concentrations of total, direct, and indirect bilirubin have been detected in natural and experimental *Brachiaria* spp. poisoning in cattle and sheep (Fagliari et al. 1994, Fioravanti 1999, Mendonça et al. 2008, Santos Jr. 2008, Saturnino et al. 2010, Castro et al. 2011). Increased serum GGT levels are the best predictors of the onset of the poisoning, tending to remain high for a longer period (Fagliari et al. 1994, Santos Jr. 2008, Saturnino et al. 2010, Castro et al. 2011). However, increased AST and GGT levels did not correlate with the severity of poisoning (Santos Jr. 2008, Saturnino et al. 2010, Castro et al. 2011).

The main macroscopic lesions, in addition to dermatitis, are jaundice, an enlarged yellowish or brownish liver, a distended gallbladder with edematous wall, subcutaneous yellowish edema, ascites, hydropericardium, and hydrothorax. On histologic examination, the liver shows vacuolation, swelling or necrosis of individual hepatocytes, bilestasis, lymphoplasmocytic cholangitis and pericolangitis and, in chronic cases, a varying degree of periportal fibrosis. The most characteristic lesions of *Brachiaria* spp. poisoning are refringent crystals or negative images of these crystals in the bile ducts, macrophages, and hepatocytes (Lemos et al. 1996, 1997; Driemeier et al. 2002; Santos Jr. 2008), and the occurrence of macrophages with foamy cytoplasm, sometimes containing crystals (Lemos et al. 1997; Driemeier et al. 1998, 1999, 2002; Gomar et al. 2005; Santos Jr. 2008; Moreira et al. 2009b).

To control poisoning, cattle should be removed from toxic pastures. The only treatment is symptomatic. It is very important to keep the animals in the shade and provide them with food and water. In the future, main preventive measures should be based on the selection of resistant or resilient animals and on the development of *Brachiaria* species or varieties with low saponin concentrations.

**Palicourea marcgravii**

*Palicourea marcgravii* (Rubiaceae) (figure 2) is the most important native toxic plant in Brazil. Until the 1990s, *P. marcgravii* was the most important toxic plant in the Central-Western region. The plant is found mainly in the native forest, and it cannot survive in pastures without shade. With the deforestation and the substitution of native forests by *Brachiaria* spp. pastures, the poisonings have become less frequent, but they still remain a concern in Goiás, Mato Grosso (mainly in the north of the state), and in the Federal District, but not in Mato Grosso do Sul (Tokarnia and Döbereiner 1986, Tokarnia et al. 1990). Because it is palatable and very toxic, sudden deaths occur in all places where the plant is found. The toxic compound of *P. marcgravii* is fluoroacetate (Oliveira 1963, Krebs et al. 1994), and doses of 0.6 to 2 g of fresh leaves per kg bodyweight (g/kg) are lethal to cattle (Tokarnia and Döbereiner 1986, Tokarnia et al. 1990).
In most cases, clinical signs are hyperacute, and they appear when the animals are exercising. The characteristic signs are loss of balance, ataxia, tachycardia, tachypnea, muscular tremors, and falling down. Some animals rise but immediately fall down again, staying in lateral recumbence and pedaling in an attempt to stand up. Nervous signs like opisthotonous and bruxism also are observed. These severe signs last, in most cases, 1 to 10 minutes before death. In some animals, early clinical signs are mild, and they consist of reluctance to walk or moving slowly, lying down frequently and for long periods, heart palpitations with dilated and visible pulsing jugular vein, and dyspnea. In these cases, death occurs after 12 to 55 hours. Some animals are found dead without exercise. Affected animals rarely recover (Tokarnia and Döbereiner 1986, Tokarnia et al. 1990). There are no significant gross lesions. Histologically, nearly 60 percent of the animals had necrosis and hydropic degeneration of the epithelium of distal convoluted tubules of the kidney (Tokarnia and Döbereiner 1986).

Clinical signs linked to exercise and to the presence of the plant are suggestive of the diagnosis. The histologic lesion of the kidneys is characteristic but is not observed in all cases. Other plants cause sudden death in Brazil with similar signs and lesions, but only *P. marcgravii* and *Amorimia (Mascagnia) pubiflora* are found in the Central-Western region of Brazil.

Poisoning from these plants is very difficult to control except by the use of fences to isolate areas with the plant. Under an agreement with CSIRO, Australia, and the USDA-ARS Poisonous Plant Research Laboratory in Logan, UT, our research group in Brazil (Institute of Science and Technology for the Control of Plant Poisoning), isolated fluoroacetate-degrading microorganisms to be used as a rumen inoculate to reduce losses from fluoroacetate-containing plants (Camboim et al. 2012).

**Amorimia (Mascagnia) pubiflora**

*Amorimia pubiflora* (Malpighiaceae) (figure 3) is a very important toxic plant in Mato Grosso do Sul, Goiás, and Mato Grosso (Tokarnia and Döbereiner 1973, Tokarnia et al. 1990, Lemos et al. 2011). Most outbreaks occur during the dry period. Morbidity varies from 1 to 3.5 percent and case fatality rate is nearly 100 percent but some animals recover if they do not exert themselves (Tokarnia and Döbereiner 1973, Tokarnia et al. 1990, Lemos et al. 2011). The toxic compound in *A. pubiflora* is unknown, but it is probably fluoroacetate. Clinical signs of poisoning by *A. pubiflora* are very similar to those mentioned in *P. marcgravii* poisoning. There are no significant lesions at necropsy. Histologic lesions of vacuolar degeneration and necrosis of distal convoluted tubules of the kidney were observed in 66 percent of cattle poisoned experimentally (Tokarnia and Döbereiner 1973) and also in some spontaneous cases (Ricardo Lemos, 2011, unpublished).
The control of poisoning by A. pubiflora is difficult. Hand removal of the plant is a good control measure for small areas, but it is not feasible in most areas. Amorimia spp. have a persistent root crown that facilitates regrowth after removal of the plant or use of herbicides. The use of fences to isolate areas with the plant is a good control measure for some farms. Goats develop considerable resistance after the continuous ingestion of small amounts of Amorimia (Mascagnia) rigida, another poisonous plant, and that resistance is transferred by transfaunation of the ruminal content, suggesting that resistance is due to ruminal microorganisms that degrade fluoroacetate (Franklin Riet-Correa et al., 2011, unpublished data).

**Enterolobium contortisiliquum and Stryphnodendron spp.**

In the Central-Western region of Brazil, there are a group of leguminous trees belonging to the family Fabaceae, subfamily Mimosoideae, including *Enterolobium contortisiliquum* (= *Enterolobium timbouva*) (figure 4) (Tokarnia et al. 1960, 1999; Grecco et al. 2002; Mendonça et al. 2009; Lemos et al. 2011), *Stryphnodendron obovatum* (Brito et al. 2001a,b), and *Stryphnodendron fissuratum* (figure 5) (Ferreira et al. 2009) that produce pods during the dry season whose consumption has been associated with digestive signs, photosensitivity, and abortion in cattle. Poisoning by *E. contortisiliquum* pods has been frequently diagnosed in Mato Grosso (Tokarnia et al. 1960, 1999; Grecco et al. 2002; Mendonça et al. 2009) and Mato Grosso do Sul (Lemos et al. 2011). The intoxication occurs when the animals ingest the fallen pods from August to November. In field outbreaks of poisoning by *E. contortisiliquum*, the main clinical signs are digestive signs (mainly diarrhea), photosensitization, and abortion, but experimentally only digestive signs (Tokarnia et al. 1960, 1999; Grecco et al. 2002; Mendonça et al. 2009; Lemos et al. 2011) and mild photosensitization (Grecco et al. 2002, Lemos et al. 2011) have been produced. The main histologic lesions are degeneration and necrosis of the epithelium of the forestomachs with formation of intraepithelial vesicles. In the liver, swelling, vacuolization, and individual necrosis of hepatocytes, and in some cases, discrete proliferation of epithelial bile duct cells are observed. The abortive properties of *E. contortisiliquum* were demonstrated in guinea pigs (Bonet-Raposo et al. 2008) but not in cattle (Tokarnia et al. 1999, Lemos et al. 2011). Cattle become tolerant to the toxicity of the pods if they ingest successive non-lethal doses (Tokarnia et al. 1999). Triterpenoid saponins (Carvalho 1981) pathogenic for guinea pigs (Bonet-Raposo et al. 2008) were isolated from *E. gummiferum* pods. Mimaki et al. (2003, 2004) identified enterolosaponin A and contortisilioside B, which were toxic to macrophages, and contortisilioside A and C, toxic to macrophages and murine lymphoma cells.

![Figure 4. Enterolobium contortisiliquum. The cattle are consuming pods (inset) of the tree.](image-url)
spontaneous poisoning were apathy, anorexia, aggressiveness, jaundice, drooling, incoordination, dysmetria, retraction of the abdomen, uneasiness, pasty black feces with strings of mucus or blood, diarrhea, edema of the dewlap, and hepatogenous photosensitization. At necropsy, jaundice, edema of the subcutaneous tissue (mainly of the cervical region), hemorrhages of serous membranes, ascites and hydrothorax, edema of the mesentery, perirenal edema, increased size of liver and kidney, reddening of the ruminal mucosa, and abomasum ulcers were observed. The main histologic lesions are degeneration and necrosis of the epithelium of the forestomachs with formation of intraepithelial vesicles, swelling, vacuolization, and individual necrosis of hepatocytes, and mild nephrosis (Ferreira et al. 2009). Experimentally, digestive signs were reproduced by the administration of pods to two bovines, but photosensitization was not observed before the animals died. Farmers in the state of Mato Grosso do Sul have observed abortion from poisoning by \textit{S. fissuratum}, and the abortive properties of the pods were demonstrated in goats (Alburquerque et al. 2011) and cows (Ricardo Lemos, 2011, unpublished). Triterpenoid saponins were isolated from \textit{S. fissuratum} (Haraguchi et al. 2006, Yokosuka et al. 2008).

To control poisoning by \textit{Stryphnodendron} spp. and \textit{E. contortisiliquum}, it is necessary to prevent the ingestion of the pods by cattle. However, frequently the ingestion of pods of \textit{E. contortisiliquum} by cattle causes no clinical signs, which can be due to the development of resistance, to the ingestion of low doses, or to both. In recent experiments, no significant differences in toxicity were found between plants from different regions (Franklin Riet-Correa and Ricardo A. Lemos, 2011, unpublished data).

\textbf{Vernonia mollissima and Vernonia rubricaulis}

\textit{Vernonia mollissima} and \textit{V. rubricaulis} (Asteraceae) (figure 6) have been reported as toxic for cattle in Mato Grosso do Sul (Döbereiner et al. 1976, Tokarnia and Döbereiner 1982), but since these reports, no more outbreaks of poisoning by \textit{V. mollissima} have been reported in this region (Lemos et al. 2011). In contrast, poisoning by \textit{V. rubricaulis} is an important cause of death in cattle in Mato Grosso do Sul (Brum et al. 2002, Lemos et al. 2011).

Poisoning by \textit{V. mollisima} occurs mainly when the plant is sprouting or during periods of forage scarcity from August to September (Dobereiner et al. 1976a). Poisoning by \textit{V. rubricaulis} occurs annually in the late dry or early wet season (August to December), when the plant is green and other forages are dry. Some conditions are important for the occurrence of the intoxication: management practices that encourage sprouting of the plant (e.g. mechanical removal or fire); exposing naïve animals to the plant either by transporting them from other ranches or moving them into unfamiliar pastures; and excessive stocking rates on pastures. Morbidity varied from 2 to 21 percent, and case fatality is...
nearly 100 percent (Brum et al. 2002, Lemos et al. 2011).

Clinical signs are characterized by apathy, tremors, dry muzzle (dehydration), dry feces with blood, and aggressive behavior. Histological findings are severe coagulation necrosis, mainly in the centrilobular region of the liver, and hemorrhages, occasionally affecting the whole lobule. Cases with massive necrosis of the liver and bleeding are frequent (Brum et al. 2002, Lemos et al. 2011). The toxic compound of the plant is unknown.

To prevent intoxication, it is necessary to avoid grazing *V. rubricaulis* during sprouting of the plant after burns, mowing, and rotational grazing. Caution is necessary when animals are moved from areas where *V. rubricaulis* does not exist into paddocks invaded by this plant.

**Solanum glaucophyllum (=Solanum malacoxylon)**

*Solanum glaucophyllum* (Solanaceae) (figure 7) is a toxic plant of the Brazilian Pantanal. It causes a chronic condition characterized by soft tissue calcification, hypercalcemia, hypercalcitoninism, and osteopetrosis. The disease occurs most frequently from June to October during the dry season and mainly during periods of forage scarcity. The plant is commonly found in low-lying flood plains and in marshy areas near rivers and streams (Döbereiner et al. 1971, Tokarnia and Döbereiner 1974, Lemos et al. 2011), but in one outbreak, the area was deforested to plant a pasture, and *S. malacoxylon* invaded this area in large amounts (Lemos et al. 2011). In three outbreaks reported recently, morbidity varied from 1.7 to 3.75 percent and lethality from 2 to 100 percent (Lemos et al. 2011). As the plant is commonly found in the Brazilian Pantanal, the intoxication is known by farmers and practitioners, and cases of intoxication are probably underreported (Lemos et al. 2011). The poisoning was reported also in buffalo from January to March during the rainy season (Santos et al. 2011).

Clinical signs are characterized by progressive weight loss, stiff gait and lameness, tucked-up abdomen, kyphosis, and heart murmurs. Animals tend to remain recumbent and have a difficult time standing up. Other signs are dyspnea and an increase in the size and rigidity of the arteries, particularly in the facial arteries, and in the iliac arteries by rectal palpation. The clinical course is chronic, and death can occur up to 4 months from extreme malnutrition and cachexia if animals are not removed from pastures where the plant occurs (Döbereiner et al. 1971, Tokarnia and Döbereiner 1974).

The lesions observed at necropsy are characterized by thick, hard, and inelastic arterial walls, excluding the pulmonary artery. The tunica intima of the arteries appears wrinkled and covered with mineralized deposits. There also is calcification of the bicuspid and aortic valves and occasionally the endocardium, and mineralization in the lungs, especially on the borders of the diaphragmatic lobes. The renal cortices have focal white areas of mineralization in the cortex and white streaks of mineralization in the medulla. Histologically, there is edema and fragmentation of elastic fibers in arteries of diverse organs, with granular deposits and mineral plaque. There also is calcification of tendons and ligaments (Döbereiner et al. 1971, Tokarnia and Döbereiner 1974, Lemos et al. 2011). The toxins in *Solanum malacoxylon* are derived glycosides of 1,25-(OH)2 D3 (calcitriol) (Wasserman 1978).

There is no treatment. If animals are removed as soon as they show clinical signs, they can clinically recover and gain weight, but the lesions are not reversible. Avoiding grazing of cattle in areas severely invaded by the plant is the only way to prevent poisoning.

**Ipomoea carnea subsp. fistulosa**

*I. carnea subsp. fistulosa* (Convulvulaceae) (figure 8) is very common in the Brazilian Pantanal and farmers in the region reported the occurrence of poisoning in cattle during the dry period. The disease occurs when there is low forage availability. Most animals recover if they are removed from the paddocks invaded by this plant. One outbreak of poisoning by this swainsonine-containing plant was
reported in cattle in the Pantanal floodplains from June to September 2006 (dry season) in an area severely invaded by *I. carnea* with low forage availability (Antoniassi et al. 2007). In three other outbreaks between 2008 and 2009, some animals developed a preference for *I. carnea* and continued eating it even in the rainy season, when there was greater availability of forage in the pasture, which resulted in intoxication and deaths during this period (Fábio S. Mendonça, 2011, unpublished). Neurological signs, characteristic of cerebellar and brainstem alterations, were mainly hypermetria, ataxia, and intention tremors. Severe weight loss was also observed. No gross lesions were observed at necropsy. The main histologic lesions were vacuolation of the pericaryon of neurons and in the cytoplasm of epithelial cells of the thyroid, kidney, and pancreas. One affected bovine removed from the area invaded by *I. carnea* subsp. *fistulosa* recovered clinically within 15 days (Antoniassi et al. 2007).

**Senna occidentalis** and **Senna obtusifolia**

*Senna occidentalis* (figure 9) and *Senna obtusifolia* (figure 10) (Fabaceae) are common weeds in Central-Western Brazil that occasionally cause poisoning in cattle grazing these plants. In two outbreaks of intoxication by *S. occidentalis* when it was in seed in Mato Grosso do Sul, morbidity varied from 6.4 to 17 percent and lethality was 100 percent, but in another outbreak, morbidity was 62 percent. This high morbidity probably was due to the high stocking rate in a paddock severely invaded by *S. occidentalis*, with low forage availability due to previous grazing (Lemos et al. 2011). Poisoning by contamination of grains by seeds of *S. occidentalis* as observed in different species in other Brazilian regions has not been reported in the Central-Western region. Poisoning by *S. obtusifolia* has been diagnosed recently in Mato Grosso, and the poisoning was reproduced experimentally by the administration of daily doses of 15 g of green leaves with pods per kg BW for 6 days (Fernando Furlan, 2011, unpublished data).

Main clinical signs are diarrhea, muscle weakness, ataxia of the hind limbs, restlessness, and recumbency, followed by death after a clinical manifestation period of 4 to 12 days. Occasionally, in some outbreaks, some animals recovered after being recumbent for some days. At necropsy, pale areas of some muscles and bleeding and congestion of the fascia are observed. The bladder contains dark urine, and in the case of *S. occidentalis* poisoning, seeds of this plant can be observed in the reticulum. Occasionally, pale areas in the myocardium and passive chronic liver congestion are observed. Main histological findings are segmental necrosis of the muscles. Centrilobular necrosis and dilatation of the renal tubules with presence of hyaline casts are occasionally observed. To prevent intoxication, it is necessary to avoid grazing animals in paddocks.
severely invaded by *S. occidentalis* or *S. obtusifolia* and avoid feeding grain contaminated with *S. occidentalis* seeds.

The toxins in the plants have not been completely isolated or identified, but anthraquinones (Lewis and Shibamoto 1989) and a compound named diantrons (Haraguchi et al. 1996) were suggested as possible toxins in *Senna* spp.

**Pterodon emarginatus**

*Pterodon emarginatus* (Fabaceae) (figure 11) is a tree found in the three states in the Brazilian Central-Western region. The intoxication seen in cattle in Mato Grosso (Arruda et al. 2008) and Mato Grosso do Sul (Lemos et al. 2011) occurs when cattle ingest leaves of trees that have been cut for wood. Farmers claim that the poisoning is frequently observed between July and September (dry season) after windstorms. The poisoning causes hepatogenous photosensitization and hepatic encephalopathy with nervous signs including incoordination, apparent blindness, and depression. The death occurs after a clinical manifestation period of 12 to 72 hours. The liver is enlarged with increased lobular pattern and a diffuse mottled appearance with red areas intercalated with yellow areas. Severe hemorrhages in the abdominal and thoracic serosa also were observed. The main histologic lesion is centrilobular (periacinar) liver necrosis. The poisoning was reproduced experimentally in bovines after the administration of 3 g of fresh leaves/kg BW, but the toxicity seems to be very variable (Edson M. Colodel and Ricardo Lemos, 2011, unpublished data). The toxic compound of *P. emarginatus* is unknown.

**Polygala klotzschii**

*Polygala klotzschii* (Polygalaceae) is a small spiny shrub reported as toxic for cattle in the 1970s in a small area of Mato Grosso do Sul (municipalities of Amambai, Guatemi, Anaurilândia, and Nova Andradina). No more outbreaks have been reported since then, and the poisoning is apparently of reduced importance. The plant is low in palatability, and the intoxication occurs mainly during the dry season when forage is scarce. Cattle of all ages are affected, morbidity is variable, and case fatality is high (Tokarnia et al. 1976). The toxic compound is 5-metoxi-podophyllotoxin, which belongs to the group of the podophyllins. The intoxication is acute and characterized by anorexia, salivation, severe depression, diarrhea, incoordination, and death within 10 to 38 hours. Gross lesions are ascites, hydrothorax, petechial hemorrhages in the trachea, endocardium and gut, enlarged and reddish lymph nodes, distention of the gall bladder, and congestion of lung, liver, kidney, and brain. Omasal content is dry, and the plant can be found in the rumen. The most characteristic histologic lesion is diffuse necrosis of lymphocytes, mainly in the germinative centers of the follicles of the spleen, lymph nodes, Payer patches, and other lymphatic tissues (Tokarnia et al. 1976).

**Tetrapterys multiglandulosa**

*Tetrapterys multiglandulosa* (Malpighiaceae) (figure 12) is a well-known toxic plant in southeastern Brazil causing three clinically different diseases, which can occur in isolation or together: a perinatal form with abortion or neonatal death, a nervous form with vacuolation (status spongiosus) of the nervous system, and a cardiac form with fibrosis of the heart,
causing sudden death or congestive heart failure. Poisoning by *T. multiglandulosa* was diagnosed twice on a farm in Mato Grosso do Sul (Carvalho et al. 2006). In the first case, a herd of 290 pregnant cows was affected; 7 cows (2.4%) died of cardiac insufficiency, and 230 cows (79.3%) aborted or delivered weak calves that died after parturition. Forty days later on the same farm, only non-pregnant heifers were affected by neurological disturbances and cardiac insufficiency: 9 of 285 heifers showed clinical signs and died. Main lesions in cows and calves were necrosis and fibrosis of the myocardium, chronic passive congestion of the liver, pulmonary edema, and status spongiosus in the white matter of the brain (Carvalho et al. 2006).

Other Toxic Plants

One outbreak of poisoning by *Lantana tilaefolia* (Verbenaceae) was reported in Mato Grosso in a herd of 400 cows introduced to a paddock severely invaded by this plant. Sixty animals showed severe photosensitization and jaundice, and 59 died. Clinical signs were observed within 10 days after the introduction of the cows to the paddock, and the clinical manifestation period was between 1 and 8 days. *Lantana* spp. contains the triterpenes Lantadene A and Lantadene B, which affect the periportal hepatocytes causing colestasis. The disease was produced experimentally by the administration of a single dose of 30 g/kg BW of fresh *L. tilaefolia* in one calf and 4 daily doses of 4 g/kg BW in another (Tokarnia et al. 1984).

Poisoning by *Cestrum laevigatum* (Solanaceae) has been reported in southern Mato Grosso do Sul. The disease occurs in hungry animals mainly in the presence of sprouting plants after cutting or fire or after exposure of naive animals to the plant (Purisco et al. 1998). The poisoning is characterized by acute liver necrosis. Clinical signs and pathology, reported elsewhere, are similar to other plants causing acute liver necrosis (Riet-Correa et al. 2009). Prevention or reduction of losses is accomplished by eradicating the plants, by avoiding overgrazing in invaded areas, or by the use of fences in areas invaded by the plant (Purisco et al. 1998). The toxin contained in *C. laevigatum* is unknown, but this plant probably contains kaurene glycosides similar to those reported in *Cestrum parqui* (Oelrichs et al. 1994).

An outbreak of acute poisoning by *Pteridium caudatum* (Polypodiaceae) was observed in Mato Grosso do Sul in the Amazonic Region in a herd of 306 cattle. After 40 days grazing in a degraded pasture severely invaded by this plant, 22 bovines were affected, and 20 died after a clinical manifestation period of nearly 3 days. Clinical signs were depression, intolerance to exercise, pale mucosa, fever (41-42°C), hemorrhages, and increased time for blood coagulation. Hemorrhages were observed at necropsy and on histologic examination there was severe aplasia of the bone marrow (Fernando Furlan, 2011, unpublished).

In Mato Grosso do Sul, three outbreaks of liver fibrosis and interstitial pneumonia in cattle, similar to those observed on pyrrolizidine alkaloids poisoning, were associated with the ingestion of *Crotalaria* spp. (Leguminoseae), but the species of the plant was not identified (Lemos et al. 2011).

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