



Neurological diseases in cattle caused by plants and mycotoxins in Santa Catarina state, Brazil¹

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ABSTRACT.- Morais R.M., Wicpolt N.S., Molossi F.A., Ogliari D., Mori A.M., Surkamp V. & Gava A. 2019. **Neurological diseases in cattle caused by plants and mycotoxins in Santa Catarina state, Brazil.** *Pesquisa Veterinária Brasileira* 39(4):244-250. Laboratório de Patologia Animal, Centro de Ciências Agroveterinárias, Universidade do Estado de Santa Catarina, Av. Luiz de Camões 2090, Bairro Conta Dinheiro, Lages, SC 88520-000, Brazil. E-mail: aldo.gava@udesc.br

This study described the epidemiological and clinical-pathological aspects of 25 outbreaks of neurological diseases in cattle caused by plants and mycotoxins in Santa Catarina state. Six of them were due to *Sida carpinifolia* poisoning, five to *Solanum fastigiatum*, five to *Phalaris angusta*, three to *Claviceps paspali*, three to *Claviceps purpurea*, and three outbreaks were of unknown etiology. The clinical signs observed in the affected cattle were mild to severe and characterized by generalized muscle tremors, incoordination, hypermetria, wide-based stance, intentional head tremors, dull staring eyes, and frequent ear twitching, with convulsions in some cases. At necropsy, lesions were observed only for *P. angusta* poisoning, characterized by gray-greenish discoloration in thalamus and midbrain. Microscopically, rarefaction and/or disappearance of Purkinje neurons with substitution by Bergmann cells were observed for *S. carpinifolia* and *S. fastigiatum* poisoning. For *P. angusta* poisoning, thin granular brown-yellowish pigment was observed in the cytoplasm of some neurons. Gross and microscopic findings were not observed in three outbreaks of tremorgenic disease of unknown etiology. Experiments conducted with leaves, flowers and seeds of *Ipomoea indivisa* and *Ipomoea triloba*, as well as with maize and soybean residues contaminated with *Ipomoea* spp. did not reproduced clinical signs.

INDEX TERMS: Neurological diseases, cattle, mycotoxins, Santa Catarina, Brazil, plant poisoning, neuropathology, diseases of cattle, toxicoses.

RESUMO. - [Enfermidades neurológicas em bovinos causadas por plantas e micotoxinas no estado de Santa Catarina.] Descrevem-se os aspectos epidemiológicos e clínico-patológicos de 25 surtos de enfermidade neurológica em bovinos no estado de Santa Catarina causadas por plantas e micotoxinas. Destes, seis corresponderam a intoxicação

por *Sida carpinifolia*, cinco por *Solanum fastigiatum*, cinco por *Phalaris angusta*, três por *Claviceps paspali*, três por *Claviceps purpurea* e três surtos de etiologia não definida. Os sinais clínicos observados nos bovinos afetados eram de intensidade leve a acentuada e caracterizados por tremores musculares generalizados, incoordenação, hipermetria, aumento da base de sustentação, balanço contínuo de cabeça, olhar atento e movimentos frequentes de orelhas, e em alguns surtos convulsões. Por meio de necropsia foram observadas alterações somente na intoxicação por *P. angusta* as quais caracterizaram por coloração cinza-esverdeada no tálamo e mesencéfalo. Na histologia, rarefação e/ou desaparecimento de neurônios de Purkinje com substituição por células de Bergmann foram observadas na intoxicação por *S. carpinifolia* e *S. fastigiatum*. Na intoxicação por *P. angusta* foi observado no citoplasma de alguns neurônios do tronco encefálico com pigmentação finamente granular marrom-amarelada. Nos três surtos de enfermidade tremorgênica com etiologia não definida não foram observadas lesões macroscópicas e

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microscópicas. Experimentos com folhas, flores e sementes de *Ipomoea indivisa* e *Ipomoea triloba* e resíduos de milho e soja contaminados com sementes destas duas plantas não produziram alterações clínicas.

TERMOS DE INDEXAÇÃO: Doenças neurológicas, bovinos, micotoxinas, Santa Catarina, intoxicações por plantas, neuropatologia, doenças de bovinos, toxicoses.

INTRODUCTION

Bovine nervous system diseases caused by plants and/or mycotoxins are neurotoxicoses characterized by depression, muscle and head tremors, incoordination, ataxic gait with hypermetria or dysmetria, wide-based stance, and alertness with dull staring eyes. In some cases, when cattle are moved, clinical signs become exacerbated and animals tend to present decubitus with seizures and nystagmus. Deaths are rare and most often due to trauma resulting from falls or emaciation (Radostits et al. 2007).

In the South region of Brazil, *Solanum fastigiatum* (Riet-Correa et al. 1983b, Rech et al. 2006) and *Sida carpinifolia* (Furlan et al. 2009, Pedroso et al. 2010) are plants that cause storage disease and, consequently, severe neurological signs. *Phalaris angusta* (Gava et al. 1999) and *Claviceps paspali* (Riet-Correa et al. 1983a) have been described as causes of tremorgenic syndrome. *Aspergillus clavatus* can produce toxins that cause neurological disease when ingested by cattle (Loretti et al. 2003, Bezerra Junior et al. 2009). In the North and Northeast regions of the Country, *Ipomoea carnea* subs. *fistulosa* (Armién et al. 2007), *Turbina cordata* (Dantas et al. 2007), *Ipomoea riedelli*, *Ipomoea sericophylla* (Barbosa et al. 2007), and *Solanum paniculatum* (Guaraná et al. 2011) have also been reported as causing storage disease in ruminants. *Ipomoea asarifolia* has been described as responsible for causing tremorgenic syndrome in ruminants (Döbereiner et al. 1960, Medeiros et al. 2003, Tortelli et al. 2008). Pessoa et al. (2010) described eight outbreaks in cattle, sheep, horses, and mules that developed tremorgenic neurological signs after grazing pastures with predominance of the *Enteropogon mollis* and *Chloris* spp. species.

At necropsy, bovine carriers of these diseases do not usually show lesions, except in poisoning by *P. angusta*, in which gray-greenish discoloration in thalamus and midbrain is observed (Gava et al. 1999), and by *S. fastigiatum* and *S. paniculatum*, in which cerebellar atrophy may occur (Rech et al. 2006, Guaraná et al. 2011). Microscopically, in poisoning by plants that cause lysosomal accumulation in neurons and by *C. paspali*, degeneration and vacuolization of Purkinje neurons with axonal spheroids in the granular layer of the cerebellum are observed in the central nervous system (CNS) (Riet-Correa et al. 1983b, Rech et al. 2006, Armién et al. 2007, Barbosa et al. 2007, Dantas et al. 2007, Furlan et al. 2008, 2009). In poisoning by *A. clavatus*, histological findings are limited to rarefaction and/or absence of Nissl granules (chromatolysis) and central or peripheral pyknotic nuclei (Loretti et al. 2003).

This study described the epidemiological and clinical-pathological aspects of 25 outbreaks of neurological diseases in cattle caused by plants and/or mycotoxins diagnosed at the Laboratory of Animal Pathology of the "Centro de Ciências Agroveterinárias" (CAV-UDESC) and evaluates the toxicity of

Ipomoea triloba and *Ipomoea indivisa* as a possible cause of three tremorgenic disease outbreaks in cattle.

MATERIALS AND METHODS

Spontaneous disease. Surveys of 25 outbreaks of neurological diseases in cattle caused by plants and/or mycotoxins diagnosed from 1987 to 2017 were obtained from the Laboratory of Animal Pathology, "Centro de Ciências Agroveterinárias" of the "Universidade do Estado de Santa Catarina" (CAV-UDESC). In 22 of these outbreaks, diagnoses were performed based on epidemiological data, clinical-pathological aspects, and observation of the feed used in the farms and the grazing areas where the cattle were kept. Three of these outbreaks were of unknown etiology, and green leaves and seeds of *Ipomoea triloba* and *Ipomoea indivisa* provided to the animals were collected.

Twenty-five clinically ill head of cattle were necropsied and samples were collected from the CNS, liver, lungs, heart, spleen, lymph nodes, forestomach, abomasum, intestines, kidneys, parotid, thyroid, pancreas, and skeletal muscle. In addition to the samples collected at necropsy, CNS samples were also collected from four head of cattle with tremorgenic disease that had been referred to slaughter. All samples were fixed in 10% formalin, routinely processed, and stained using the hematoxylin-eosin (HE) method for reading under light microscopy.

Experimental study. Three bovines, one black-and-white Holstein Friesian and two Jersey, were used in the experimental study. Green leaves, flowers and seeds of *Ipomoea indivisa* (Aiea morning glory) collected in the municipalities of Concórdia and Abelardo Luz, Santa Catarina state, were provided *ad libitum* to the first bovine. The second bovine was fed maize (*Zea mays*) contaminated with seeds of *Ipomoea indivisa* and *Ipomoea triloba* collected in Concórdia. The third bovine was fed, *ad libitum*, green leaves, flowers and seeds of *Ipomoea triloba* (Aiea morning glory) and soybean (*Glycine max*) residue contaminated with seeds of *Ipomoea triloba* and *Ipomoea indivisa* collected in the municipality of Canoinhas. At the end of the afternoon, the calves were released for grazing in a paddock of kikuyu grass (*Pennisetum clandestinum*) with water *ad libitum*. Clinical examinations, which prioritized behavioral and locomotion assessment, were performed before and after administration of the plants and residues. Table 1 shows the experimental design.

Samples of green leaves, flowers and seeds of *Ipomoea* spp. were sent for botanical identification. This study was approved by the Animal Research Ethics Committee of the "Universidade do Estado de Santa Catarina" (CEUA-UDESC) under protocol no. 4893241116.

RESULTS

Epidemiology

Of the 25 outbreaks of neurological diseases in cattle associated with poisoning by plants and/or mycotoxins monitored by the Laboratory of Animal Pathology of the CAV-UDESC in Santa Catarina state, six were due to *Sida carpinifolia* poisoning, five to *Solanum fastigiatum*, five to *Phalaris angusta*, three to *Claviceps paspali*, three to *Claviceps purpurea*, and three outbreaks were of unknown etiology with suspicion of ingestion of leaves, flowers and seeds of *Ipomoea indivisa* and *Ipomoea triloba*.

Poisoning by *Sida carpinifolia* was observed in six farms located in the Alto Vale do Itajaí region from 2003 to 2005. The disease occurred in small properties where there was invasion of *S. carpinifolia*, which, in many cases, was the

Table 1. Experimental design with *Ipomoea indivisa*, *Ipomoea triloba*, and residues of maize and soybean contaminated with *Ipomoea* spp. seeds

Bovine	Breed	Age (months)	Weight (Kg)	<i>Ipomoea indivisa</i>	<i>Ipomoea triloba</i>	Maize residue	Soybean residue
				g/Kg bw day/ consumption days	g/Kg bw day/ consumption days	g/Kg bw day/ consumption days	g/Kg bw day/ consumption days
1	BWHF ^{ab}	12	170	-	-	18.58/6	-
2	Jersey ^b	12	160	86.53/22	-	-	-
3	Jersey ^b	12	160	-	113.03/6	-	22.13/5

^a Black-and-white Holstein Friesian, ^b male.

predominant vegetation. In some properties, the cattle were removed from the areas invaded by the plant, and the clinical signs regressed, but upon return the condition worsened. Poisoning by *Solanum fastigiatum* was observed in five rural properties located in the following municipalities and years: Erval Velho in 1997 and 2007, Chapecó in 2003, Água Doce in 2006, and Herval do Oeste in 2005 and 2007. The affected cattle grazed areas infested by large amounts of *S. fastigiatum*.

Poisoning by *Phalaris angusta* was reported in five farms located in the following municipalities and years: Capinzal in 1993, Canoinhas in 1996, Xanxerê in 1997 and 1998, and Porto União in 2010. In these outbreaks, the cattle grazed areas of soybean and/or maize stubble, and/or associated with pasture of common oat (*Avena sativa*) with presence of *P. angusta*.

The three outbreaks of poisoning by *Claviceps paspali* occurred in the municipalities of São Joaquim in pastures with *Paspalum dilatatum* in 1987 and 1988 and Otacílio Costa in pastures with *Paspalum paniculatum* in 2014. The affected cattle showed clinical signs two days after introduction in paddocks of *Paspalum* spp. in the seeding phase and infected with *C. paspali* sclerotia. Falls in steep areas and ditches were frequent in severe cases. When infestation with contaminated *Paspalum* spp. was low, the signs were mild and only ataxia and constant ear twitching were observed.

Outbreaks of poisoning by *Claviceps purpurea* in the neurological form have occurred in the municipalities of Água Doce and Lages in 2015 and Campos Novos in 2016. In the first two outbreaks, the cattle were grazing perennial ryegrass (*Lolium multiflorum*) pasture in the seed maturation phase, whereas in the third outbreak, the bovines were confined and fed pre-dried common oat (*Avena sativa*) in the seed maturation phase.

Three outbreaks of tremorgenic syndrome monitored from 2015 to 2017 were of unknown etiology. In the outbreak observed in Concórdia, 30 bovines aged 24 months were kept under semi-intensive management and received residues from maize dryer with large amounts of seeds of *I. triloba* and *I. indivisa*. They also had access to maize stubbles with large amounts of these seeds (Fig.1) and, according to the farm owner, the cattle ingested a large amount of this plant. In the outbreak observed in Abelardo Luz in April 2016, 10 crossbred cattle aged 18 months showed muscle tremors, frequent ear twitching, and ataxic gait. According to the farm owner, the disease began after introduction of the cattle in an area of maize remains contaminated by large amount of *I. indivisa*, which was in the phase of flowering and seed maturation (Fig.2). In the outbreak occurred in Canoinhas in May 2017, a batch of eight crossbred 12-month-old cattle showed clinical signs identical to those described in the outbreak observed in



Fig.1. *Ipomoea triloba* (Aiea morning glory or little bell) collected in the municipality of Concórdia, Santa Catarina state.



Fig.2. *Ipomoea indivisa* (Aiea morning glory or little bell) in maize stubble in the municipality of Abelardo Luz, Santa Catarina state.

Abelardo Luz; however, these cattle were kept in native field paddocks with no *Paspalum* spp. and, additionally, received dryer residue containing remnants of soybean grains and seeds of *I. triloba* and *I. indivisa*.

Tables 2 and 3 show the main epidemiological data and clinical findings (necropsy and histology), respectively.

Table 2. Epidemiological data relative to the outbreaks of neurological diseases in cattle caused by plants and/or mycotoxins diagnosed at the Laboratory of Animal Pathology, CAV-UDESC, Santa Catarina, from 1987 to 2017

Plant	Number of outbreaks	Number of bovines at risk/ natural death	Age	Time of year	Morbidity (%)	Mortality (%)	Clinical outcome
<i>Sida carpinifolia</i>	6	120/20	2-4 years	Year-round	Up to 90%	Up to 10%	Chronic
<i>Solanum fastigiatum</i>	5	100/4	>4 years	Year-round	Up to 50%	Up to 4%	Chronic
<i>Phalaris angusta</i>	5	150/2	>1 year	Jul./Sep.	10 - 100%	Up to 1.33% ^a	Acute
<i>Claviceps paspali</i>	3	50/0	>1 year	Mar./Apr.	30 - 100%	0	Acute
<i>Claviceps purpurea</i>	3	40/5	1-3 years	Oct./Nov.	3 - 10%	12.5%	Acute
Unknown etiology	3	48/0	1-2 years	Apr./May	80%	0	Acute

^a Traumas.**Table 3. Main clinical findings (necropsy and histopathology) for outbreaks of neurological diseases in cattle caused by plants and/or mycotoxins diagnosed in the Laboratory of Animal Pathology, CAV-UDESC, Santa Catarina state, from 1987 to 2017**

	Clinical signs	Necropsy findings	Histopathology findings
<i>Sida carpinifolia</i>	Dull staring eyes, continuous head and neck tremor, incoordination; convulsion and falls during moving in severe cases.	Unchanged	Thin granular vacuolization in the neurons of the central and peripheral nervous system; engorged Purkinje neurons, eosinophilic cytoplasm and irregular cytoplasmic borders; rarefaction of Purkinje neurons and proliferation of Bergman cells; axonal spheroids.
<i>Solanum fastigiatum</i>	Dull staring eyes, incoordination, varying degrees of hypermetria, wide-based stance, falls followed by generalized muscle tremors lasting few minutes, opisthotonus, and nystagmus.	Unchanged	Vacuolization of soma, necrosis and rarefaction of Purkinje neurons with Bergmann cell replacement.
<i>Phalaris angusta</i>	Frequent head tremors and ear twitching, marching gait, ataxia, varying degrees of hypermetria, convulsions, colliding against fences and walls, and/or falls in steep areas. Traumatic pectoral edema observed in one of the outbreaks.	Gray-greenish discoloration in thalamus and midbrain; increased volume of the ventral thoracic region with large amounts of fibrin and edema.	Some brainstem neurons with intracytoplasmic brown-yellowish granular pigment.
<i>Claviceps paspali</i>	Alertness with erect ears, hyperexcitability, rigid thoracic and pelvic limbs, wide-based stance, head and neck tremors, seizures, frequent exposure of the penis in bulls.	Unchanged	Unchanged
<i>Claviceps purpurea</i>	Dull staring eyes with alertness, incoordination, wide-based stance, convulsions with falls, diarrhea.	Unchanged	Unchanged
Unknown etiology	Dull staring eyes with head tremors, incoordination accentuated when animals were moved, wide-based stance, sternal decubitus.	Unchanged	Unchanged

Experiments conducted with green leaves, flowers and seeds of *I. triloba* and *I. indivisa*, as well as with maize and soybean residues contaminated with seeds of these two plants did not reproduce clinical signs.

DISCUSSION

In this study, *Sida carpinifolia* was the main plant responsible for causing neurological diseases in cattle in Santa Catarina state, followed by *Solanum fastigiatum*, *Phalaris angusta*, *Claviceps paspali*, and *Claviceps purpurea*. The outbreaks of *S. carpinifolia* occurred in small properties where the plant invaded the paddocks and was predominant in some situations, which, associated with hunger, were essential factors for occurrence of the diseases. These conditions were

also reported by Colodel et al. (2002), Furlan et al. (2009), and Bassuino et al. (2017) in poisoning in goats, cattle, and horses, respectively. The clinical signs observed in bovines in outbreaks of *S. carpinifolia* and *S. fastigiatum* are characteristic of cerebellar lesion. Because these diseases result in lysosomal accumulation and mainly affect the Purkinje neurons, the clinical signs were mainly head and neck tremors, ataxic gait, and frequent falls. One of the characteristics of poisoning by *S. fastigiatum* in the described outbreaks was convulsive episodes, lasting few minutes and with fast recovery. These characteristics have been described by Riet-Correa et al. (1983b) and Rech et al. (2006) in poisoning by *S. fastigiatum* and by Guaraná et al. (2011) in poisoning by *S. paniculatum*.

In poisoning by plants that cause lysosomal storage disease, especially those containing swainsonine toxins, it is

described that animals tend to be avid for these plants even when other feed is available. Tokarnia et al. (2012) interprets this avidity of the animals as a neurophysiological dependence due to neurological lesions, with loss of selective instinct and indiscriminate consumption of the plant. In poisoning by *S. carpinifolia*, ingestion was resumed even in areas with good feed availability, which demonstrates that cattle acquire the habit of ingesting the plant.

Deaths were recorded in cattle regarding poisoning by *S. fastigiatum*, *S. carpinifolia*, *P. angusta*, and *C. purpurea*. These deaths were associated with the lesions produced in the CNS, which are always present in poisoning by *S. carpinifolia* and *S. fastigiatum*. Some deaths observed in poisoning by *P. angusta* were always accidental, when cattle had access to steep areas. In the deaths caused by *Claviceps purpurea* in the neurological form, gross and microscopic lesions were not found, and no association with accidents was observed.

Outbreaks caused by ingestion of *P. angusta* have occurred from July to September. This plant presents an annual cycle and grows well in winter, when it is found invading other pastures, mainly of common oat, as well as in areas where maize or soybean had been cultivated in the summer. Thus, the diseases caused by this plant were observed only in winter and early spring. The onset of clinical signs may occur within the first days of plant ingestion, and their intensity depends on the soil conditions and moisture and stage of development of the plant (Gava et al. 1999), whereas in poisoning by *S. carpinifolia* and *S. fastigiatum*, the disease has occurred in different times of the year and, according to Riet-Correa et al. (1983b) and Furlan et al. (2008, 2009), clinical signs begin a few weeks after ingestion.

Poisoning by *C. paspali* was observed in cattle that ingested seeds of *Paspalum dilatatum* and/or *P. paniculatum* contaminated by the mycotoxin after the first days of introduction in the pasture. These outbreaks occurred in autumn, coinciding with periods of low rainfall with maturation of seeds of the genus *Paspalum* and proliferation of *C. paspali*. In the Southern Hemisphere, the described outbreaks have occurred in times of low rainfall between the end of February and the beginning of June (Riet-Correa et al. 1983a, Lopez et al. 1985, Botha et al. 1996, García et al. 2017).

The three outbreaks of nervous ergotism occurred in late spring, and the diagnoses were performed based on epidemiological data and clinical-pathological aspects. The cattle showed clinical signs while grazing ryegrass pasture (2 outbreaks) and being fed common oat with seeds contaminated by a fungus morphologically similar to *C. purpurea* in trough (1 outbreak). In Chile, cattle showed similar clinical signs after 30 days in a pasture of *Lolium perenne* and *Dactylis glomerata* contaminated by *C. purpurea* sclerotia (Poo & Araya 1989). The neurological or convulsive form is little observed in cattle, and occurs when consumption of *C. purpurea* sclerotia is relatively high (Clegg 1959).

Comparing the severity of the clinical condition manifested by the cattle, more severe clinical signs are verified in poisoning by *P. angusta*, often leading the animals to hurl themselves against obstacles. This explains why, in one of the outbreaks, due to the daily stalling of cattle, the animals had edemas of traumatic origin in the pectoral region. In poisoning by *S. fastigiatum*, the clinical signs were less severe than in poisoning by *P. angusta*; however, the clinical condition

worsened mainly when cattle had access to feed in the trough. This fact can be explained by Purkinje neuron lesion, which occurs in lysosomal storage diseases. As for tremorgenic disease caused by *C. paspali*, clinical signs ranged from mild to severe depending on the amount of *Paspalum* spp. in pasture.

In poisoning caused by *S. carpinifolia*, *S. fastigiatum*, *C. paspali*, and *C. purpurea* in the CNS, no gross lesions other than those resulting from traumas were observed. In *P. angusta* poisoning, the gross lesions observed included gray-greenish discoloration in thalamus and midbrain, which have also been previously reported by other authors (Gava et al. 1999, Sousa & Irigoyen 1999). In the present study, significant histological changes were observed in the CNS of bovines poisoned with *S. carpinifolia* and *S. fastigiatum*. Degeneration and vacuolization of the pericardium of neurons, especially of Purkinje cells of the cerebellum, are findings particularly common in lysosomal storage diseases (Riet-Correa et al. 1983b, Antoniassi et al. 2007, Barbosa et al. 2007, Dantas et al. 2007, Furlan et al. 2008, 2009). In both diseases, the cattle presented rarefaction and/or disappearance of Purkinje neurons, some with substitution by Bergmann cells - a finding that corroborates those of other studies and characterizes the chronicity of poisoning (Riet-Correa et al. 1983b, Rech et al. 2006, Furlan et al. 2008, 2009). In the present study, of all the bovines poisoned with *P. angusta*, only one animal showed pigmented brainstem neurons. According to Sousa & Irigoyen (1999) and Alden et al. (2014), poisoning by *P. angusta* and *P. aquaticum* microscopically shows thin granular brownish pigment in the cytoplasm of neurons, mainly in the cerebellum and brainstem.

In western Santa Catarina state, invasive plants such as *Ipomoea* spp. are commonly observed in plantations of maize and other cultures, and this plant is described by farmers as palatable for cattle of the region. In the epidemiological study, the main suspicion was that poisoning was caused by the plant and/or seeds, because in the three outbreaks of unknown etiology, the bovines showed neurological clinical signs after ingesting a large amount of leaves, flowers and seeds of *Ipomoea* spp. Nevertheless, experimental administration did not result in clinical changes. In experiments conducted with ruminants using *I. sericophylla*, *I. riedelii*, *I. asarifolia*, and *I. carnea* subsp. *fistulosa*, the clinical signs were chronic, being observed at least one week after consumption (Barbosa et al. 2007, Araújo et al. 2008, Ríos et al. 2012).

For the diagnosis of neurological diseases in cattle associated with plant and/or mycotoxin poisoning, the geographical distribution and seasonality, severity of clinical signs, and presence of one of the plants and/or fungi responsible for these diseases should be considered. Clinical signs such as muscle tremors, ataxia, and alertness are also observed in poisoning by the *Aspergillus clavatus* mycotoxin (Loretti et al. 2003, Bezerra Junior et al. 2009); however, this disease has not yet been diagnosed in Santa Catarina state, and in none of the outbreaks analyzed in the present study was there consumption of brewery residues. In Brazil, other diseases produce neurological clinical signs in cattle, such as poisoning by *I. asarifolia* (Döbereiner et al. 1960), *I. carnea* sub. *fistulosa* (Antoniassi et al. 2007), *I. riedelii*, *I. sericophylla* (Barbosa et al. 2007), *I. verbascoidea* (Lima et al. 2013), *S. paniculatum* (Guanará et al. 2011), and *Turbina cordata* (Dantas et al. 2007). In other countries, *Swainsonina*, *Oxitropis*, and *Astragalus* have been described as causing lysosomal storage

diseases (James et al. 1981, Smith 2006). These plants were not found in the farms of the present study. For differential diagnosis, gross and microscopic changes should also be considered. Blue-greenish coloration is observed only in poisoning by *P. angusta*, and cerebellar atrophy is found in poisoning by *S. fastigiatum* and *S. paniculatum*. Significant microscopic lesions occur in poisoning by *S. carpinifolia*, *S. fastigiatum*, *S. paniculatum*, *I. carnea* subsp. *fistulosa*, *I. riedelii*, *I. sericophylla*, *I. verbascoidea*, *S. paniculatum*, *Turbina cordata*, and *A. clavatus*, but are not observed in poisoning by *C. paspali*, *C. purpurea*, *P. angusta*, and *I. asarifolia*.

CONCLUSIONS

In Santa Catarina state, *Sida carpinifolia*, *Solanum fastigiatum*, *Phalaris angusta*, *Claviceps paspali*, and *Claviceps purpurea* are responsible for causing neurological diseases in cattle.

In experiments conducted with cattle, *Ipomoea triloba* and *Ipomoea indivisa* did not reproduce clinical signs.

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