Intoxication by *Ipomoea sericophylla* and *Ipomoea riedelii* in goats in the state of Paraíba, Northeastern Brazil

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**Abstract**

A disease of the nervous system was observed in goats from two farms of the semiarid of the state of Paraíba, northeastern Brazil. *Ipomoea sericophylla* was found in one farm and *I. riedelii* in the other. Both plants were administered experimentally to five goats each. Both plants induced clinical signs similar to those observed in spontaneous cases. Two goats died spontaneously and five were euthanatized. Three goats recovered after the withdrawal of the plants. Histological examination showed that all goats that died spontaneously or were euthanized had diffuse vacuolation of neurons, macrophages of lymphatic tissues, and epithelial cells of pancreas, thyroid, renal tubules and liver. On electron microscopy of Purkinje cells, numerous dilated membrane bordered vacuoles were identified as lysosomes. On lectin-histochemical analysis, cerebellar cells gave positive reactions to *Concanavalia ensiformis*, *Triticum vulgaris*, and succinyalted-*T. vulgaris*, which indicate the storage of \( \alpha\)-D-mannose, \( \alpha\)-D-glucose, \( \beta\)-D-N-acetyl-glucosamine, and acetyl-neuraminic acid. The chemical analysis of *I. sericophylla* and *I. riedelii* showed 0.11 and 0.14% of swainsonine, respectively. The latter also contained calystegines B1, B2 and C1. It is concluded that *I. sericophylla* and *I. riedelii* cause a lysosomal storage disease.

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**Keywords:** Storage diseases; Swainsonine; Ipomoea sericophylla; Ipomoea riedelii; Toxic plants

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Acquired glycoprotein storage diseases are caused by plants of the genera *Swainsona*, *Oxytropis*, *Astragalus* (James et al., 1970; James and Panter, 1989; Dorling et al., 1978; Hartley et al., 1989), *Ipomoea* (de Balogh et al., 1999; Armieén, 2000; Rodriguez Armesto et al., 2004) and *Sida carpinifolia* (Driemeier et al., 2000) which contain swainsonine a well known inhibitor of lysosomal \( \alpha\)-mannosidase and Golgi mannosidase II (Huxtable and Dorling, 1982; Molyneux et al., 1995b; de Balogh et al., 1999; Colodel et al., 2002a; Haraguchi et al., 2003).

*Ipomoea carnea* Jacq. subsp. *fistulosa* (Mart.ex Choisy) D.F. Austin has been recognized as toxic since 1960 in northeastern Brazil (Tokarnia et al., 1960) affecting goats, sheep and cattle. More recently the disease was characterized as a glycoprotein storage diseases (Armieén, 2000)
caused by the presence of swainsonine in the plant (de Balogh et al. 1999; Haraguchi et al., 2003; Medeiros et al., 2004). Another plant causing glycoprotein storage diseases in goats (Driemeier et al., 2000), sheep (Seitz et al., 2005), and horses (Loretti et al., 2003) in southern Brazil is Sida carpinifolia, which also contains swainsonine (Colodel et al., 2002a).

The object of this paper is to report the clinical signs, histological and ultrastructural findings, and lectin-histochemical analysis of the cerebellar cells in the spontaneous and experimental intoxication by Ipomoea sericophylla and I. riedelii in goats, as well as the determination of the active principle of these plants.

1. Material and methods

1.1. Study of the spontaneous disease

Two farms, one in the municipality of São Sebastião do Umbuzeiro, and another in the Municipality of Zabelé, state of Paraíba, Brazil where the disease was occurring were visited to determine the epidemiology of the disease and observe the clinical signs. One goat with clinical signs from the farm in the municipality of São Sebastião do Umbuzeiro was euthanized by electric shock following anesthesia with sodium pentobarbionate and necropsied. After fixation, the CNS was sectioned in 4–5 mm transverse sections. Transverse sections taken from the cervical, thoracic and lumbar spinal cord, medulla oblongata, pons, rostral colliculi, thalamus, basal nuclei and internal capsule, cortex, cerebellar peduncles, and cerebellum were examined histologically. Longitudinal sections of the spinal cord were also studied. Samples of liver, kidney, lung, lymph nodes, spleen, thyroid, adrenal, stomach, small and large intestine, cardiac, skeletal muscles and peripheral nerves were also fixed in 10% neutral formalin. All tissues were embedded in paraffin, sectioned at 6 μm, and stained with hematoxylin and eosin. Selected sections of the CNS were also stained with Luxol fast blue for myelin.

Other farms in the municipalities of São Sebastião do Umbuzeiro, Zabelé and Monteiro were visited to ask the farmers about the occurrence of the disease and for observation of the paddocks.

1.2. Identification of the plant

Samples of the whole plant of the two different species of Ipomoea found in each farm were sent for botanical identification to the Empresa Pernambucana de Pesquisa Agropecuária, Recife, state of Pernambuco, Brazil.

1.3. Experimental reproduction of the disease

The plants used in the experiments were collected in the municipalities of São Sebastião do Umbuzeiro (I. sericophylla), and Zabelé (I. riedelii). For the experiments with fresh plants they were collected weekly and kept at 3–4 °C until administered. For the experiment with dry plants, the plants were dried in the shade, ground and mixed with concentrate ration.

Fourteen 4–12-month old Moxotó goats were used; seven (nos 1–7), to reproduce the intoxication by I. sericophylla and seven (nos 8–14) to reproduce the intoxication by I. riedelii. General information for the experimental studies is summarized in Table 1. Data on the amount of plants consumed were collected daily, and the goats were weighted at start and end of experiment.

1.3.1. Experiments with I. sericophylla

Goats 1 and 2 consumed the fresh plant as the only food for 24 and 34 days, respectively. From day 34 to day 40, goat 2 received the fresh plant ad libitum, but also commercial concentrate ration in an amount equivalent to 1% live weight. Goats 1 and 2 were euthanized 1 day after the end of plant consumption. Goat 3 received the fresh plant ad libitum, and commercial concentrate ration in an amount equivalent to 1% live weight, during 38 days. After this period, the plant was substituted by Cynodon dactylon (Tifton grass) hay. After the withdrawal of the plant the animal was examined daily to observe regression of the clinical signs. Goats 4 and 5 received, daily, a mixture of 50% dry ground I. sericophylla and 50% concentrate ration, in amounts equivalent to 0.8% of their live weight, which represents a daily dose of 4 g/kg bw of dry plant. They also received C. dactylon hay ad libitum. Goat 4 received the plant for 53 days. After this period the animal was observed for regression of the clinical signs, fed with 1% bw of concentrate ration and C. dactylon hay ad libitum. Goat 5 was euthanized and necropsied, 1 day after the withdrawal of the plant. The period of plant administration was determined by the intensity of clinical signs or by the disposition of the animals to ingest it. Goats 6 and 7 were used as controls, receiving concentrate ration (1% bw) and C. dactylon hay ad libitum.

1.3.2. Experiments with I. riedelii

Goats 8 and 9 consumed the fresh plant as the only food for 22 days. Goat 10 received the fresh plant ad libitum during 60 days, but also commercial concentrate ration in an amount equivalent to 1% live weight. Goats 8 and 9 died spontaneously and goat 10 was euthanized 3 days after the end of consumption. After this period the plant was substituted by C. dactylon hay. Goats 11 and 12 received, daily, a mixture of 50% dry ground I. riedelii and 50% concentrate ration, in amounts equivalent to 0.6% of their live weight, which represents a daily dose of 3 g/kg bw of dry plant. They also received C. dactylon hay ad libitum. Goat 11 received the plant for 46 days. After this period the animal was observed for regression of the clinical signs, fed with 1% bw of concentrate ration and C. dactylon hay ad libitum. Goat 12 received the plant for 39 days, and was
euthanized and necropsied 1 day after the withdrawal of the plant. The period of plant administration was determined by the intensity of clinical signs or by the disposition of the animals to ingest it. Goats 13 and 14 were used as controls, receiving concentrate ration (1% bw) and C. dactylon hay ad libitum.

1.3.3. Clinical, macroscopic and microscopic examinations

During the experiments the animals were examined daily, performing a detailed nervous system examination, including the head raising test and the stand up test. The first consists of raising the head of the animal for about 60 s and then suddenly releasing it to observe the presence of cerebellar signs (Pienaar et al., 1976). The stand up test consists in putting the animal in lateral recumbence then observing the time to get up and the presence of coordination problems (Armien, 2000).

Goats 1, 2, 5, 10 and 11 were euthanized by electric shock following anesthesia with sodium pentobarbitone. These goats and goats 8, 9, and 12, which died spontaneously, were necropsied. Histological examination was performed with the methods reported for the spontaneous case.

1.3.4. Electron microscopy

Samples of the cerebellum of goats 5 and 10 were fixed in 2% glutaraldehyde with 2% paraformaldehyde, in 0.4 M cacodylate buffer (pH 7.4), post fixed in 1% osmium tetroxide buffered in 0.4 M sodium cacodylate (pH 7.4), and embedded in Epon 812. Semithin sections were stained with methylene blue. Ultrathin sections were stained with lead citrate and uranyl acetate and examined with a EM 109 Zeiss transmission electron microscope at 80 kw.

1.3.5. Lectin histochemistry

Paraffin wax-embedded sections (5 μm) from goats 1, 5, 9 and 10 were used. After dewaxing, sections were treated with 0.3% hydrogen peroxide in methanol for 30 min at room temperature (to inhibit the endogenous peroxidase), rinsed several times in 0.01 M PBS (pH 7.2), and treated with 0.1% bovine serum albumin in PBS for 15 min. The sections were then incubated for 1 h at room temperature with biotinylated lectins. Nine lectins (Vector Laboratories, Burlingame, CA, USA) with different specificities were used, as follows: Con A (C. ensiformis), binding specificity α-D-Man and α-D-Glc; DBA (Dolichos biflorus), binding specificity α-D-GalNAc; SBA (Glycine max), binding specificity α-D-GalNAc, and α-D-GalNAc and α- and β-Gal; PNA (Arachis hypogaea), binding specificity β-D-Gal and (1–3)Gal NAc; RCA-I (Ricinus communis-I), binding specificity β-D-Gal and α-D-Gal; UEA-1 (Ulex europaeus-I), binding specificity α-L-Fuc; WGA (Triticum vulgaris), binding specificity α-D-GlcNAc and NeuNAc; sWGA (Succinyl-WGA), binding to β-(1–4)β-D-GlcNAc; and BS-I

Table 1
Mean daily and total dose of I. sericophylla and I. riedelii consumed by the experimental goats, weight of the animals at the start and end of the administration, clinical signs, and outcome of the intoxication

<table>
<thead>
<tr>
<th>Goat no.</th>
<th>Plant material</th>
<th>Initial weight (kg)</th>
<th>Final weight (kg)</th>
<th>Daily dose (g/kg)</th>
<th>Total dose (g/kg)</th>
<th>Ingestion period (days)</th>
<th>Onset of clinical signs (days)</th>
<th>Duration of clinical signs (days)</th>
<th>Euthanasia (days after the end of ingestion) or death</th>
<th>Recovery (days after the end of ingestion)</th>
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<td>1</td>
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<td>7</td>
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<td>112</td>
<td>2688</td>
<td>24</td>
<td>22</td>
<td>4</td>
<td>1</td>
<td></td>
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<tr>
<td>2</td>
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<td>59</td>
<td>2419</td>
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<td>950</td>
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<td>28</td>
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</tr>
</tbody>
</table>

a Ipomoea sericophylla.
b Ipomoea riedelii.
c Until withdrawal of the plant.
(Bandeirea simplicifolia-I), binding to α-D-Gal. The optimal lectin concentration was 30 μg/ml in PBS for all lectins, except PNA, which was applied at a concentration of 10 μg/ml. The slides were incubated with an avidin–biotin–peroxidase complex (ABC) (vector) for 45 min. The horseradish peroxidase was activated by incubation for 1–2 min with a diaminobenzidine commercial kit (DakoCytomation, Carpinteria, CA, USA). Specimens were rinsed in distilled water, dehydrated with graded ethanol solutions, cleared in xylene and mounted in Permount (Fisher Scientific International, Inc., Liberty Lane Hampton, NH, USA). Controls for lectin staining included: exposure to horseradish-peroxidase and substrate medium without lectin; and blocking by incubation with the appropriate blocking sugars (0.1–0.2 M in PBS) for 1 h at room temperature before applying to the sections. The intensity of lectin binding was subjectively scored from 0 (none) to 3 (strongly positive). Two cerebells of slaughtered, 1 year old, Moxotó goats were used as control.

1.4. Identification of the active principles of the plants

Swainsonine content of I. sericophylla and I. riedelii was measured by liquid chromatography–mass spectrometry using procedures previously described (Gardner et al., 2001). Duplicate samples (100 mg) of the air-dried plant material were extracted and the swainsonine isolated by ion-exchange resin. The final aqueous extract was then analyzed by liquid chromatography–mass spectrometry. The presence of swainsonine was verified using gas chromatography–mass spectrometry after a portion of the aqueous extract was dried and the residue derivatized by addition of N,O-bis(trimethylsilyl)trifluoroacetamide (BSTFA) and pyridine to convert swainsonine to its trimethylsilyl ether derivative. Calystegines were analyzed by gas chromatography–mass spectrometry by the method of Molyneux et al. (1995). Dried, ground leaves (approximately, 1 g) were extracted with methanol (MeOH) for 16 h in a Soxhlet apparatus, and the alkaloidal fraction was purified by ion-exchange chromatography on a 5×0.5 cm column of Dowex 50WX8 (NH4+ form). The 0.5% aqueous ammonium hydroxide eluate was evaporated to dryness to give the base fraction, containing any alkaloids and basic amino acids. Portions of this material were used for gas chromatography–mass spectrometric (GC–MS) analysis of the trimethylsilyl (TMSi) derivatives, prepared by treatment with N-methyl-N-trimethylsilyl-trifluoroacetamide (MSTFA) in pyridine at 60 °C for 1 h. Analyses were performed on a Hewlett-Packard 5890 Series II instrument equipped with a 5971 mass-selective detector operating at 70 eV, an on-column injector, and a 60 m×0.32 mm i.d. SE-30 fused Si column. The column was temperature programmed from 120–300 °C at 10°C/min.

2. Results

2.1. Identification of the plant

The plant collected in the municipality of São Sebastião do Umbuzeiro was identified as I. sericophylla Meisn (Fig. 1). Voucher specimens kept in the herbarium Dármando de Andrade Lima were identified as: São Sebastião do Umbuzeiro, Paraíba, N° 66099 (IPA), and São Sebastião do Umbuzeiro, Paraíba, N° 66102 (IPA). The plant collected in the municipality of Zabelé was identified as I. riedelii Meisn (Fig. 2). Voucher specimens were identified as Monteiro, Paraíba, N° 60345 (IPA), and Monteiro, Paraíba, N° 60344 (IPA).

2.2. Spontaneous cases

The farm in the municipality of São Sebastião do Umbuzeiro was visited in June 2001. In the farm, 17 out of 18 goats of different ages showed clinical signs characterized by rough hair coat, depression, weight loss, and nervous signs including difficulties in rising, ataxia, hypermetria, wide-based stance, intention tremors, spastic paresis mainly in the hind legs, abnormal postural reactions, nystagmus, head tilting, depressed tongue or lip tonus, and occasionally other signs of cranial nerves impairment. Clinical signs got worst when the goats were disturbed or frightened. The animals had been affected for more than 6 months. A goat with non-reversible clinical signs (Figs. 3 and 4) was euthanized. No macroscopic lesions were observed. The main histologic lesions were in the cerebellum with the loss of Purkinje cells, which had been substituted by Bergman glia (Fig. 5). Wallerian-type degeneration, due to neuronal loss was observed mainly in the cerebellar white matter. In this year, probably because the rainy season was already finished no species of Ipomoea were found. In the following year during the rainy season, it was observed that the paddock was severely invaded by I. sericophylla. The
farmer reported that the goats consumed the plant in preference to other forages.

A farm in the municipality of Zavelé, which was neighboring to São Sebastião de Umbuzeiro, was visited in April 2002. The farm had 80 goats, 46 sheep, and 24 cattle. One female, 1-year-old, goat showed clinical signs similar to those previously mentioned. This goat was brought to the University of Campina Grande, and two and a half years later showed the same clinical signs. In the year 2001, two goats were affected in this farm, in 2000, six were affected, and more than 20 had been affected in 1999. Most cases had permanent clinical signs and were culled. The disease was more frequent in 1–2 years young goats. Sheep and cattle were not affected. The paddock was severely invaded by *I. riedelii*. The farmer reported that the goats developed a preference to consume the plant, which he defined as addiction.

During the rainy season of 2003, another 10 farms were visited in the three neighboring municipalities of Zavelé, São Sebastião do Umbuzeiro, and Monteiro to ask the farmers about the occurrence of the disease and for observation of the paddocks. In four farms only *I. riedelii* was found in the paddocks, and in one only *I. sericophylla* was found. In the other farms both plants were present in the paddocks in different amounts, but generally *I. riedelii* was more frequent. In one farm there was one goat with clinical signs, and in another two goats were affected. Four farmers did not observe affected animals on their farms, and the other four reported that the disease occurred in previous years.

![Fig. 2. Ipomoea riedelii. Meisn. Municipality of Zavelé, State of Paraíba, Brazil.](image)

![Fig. 3. Goat intoxicated spontaneously by *I. sericophylla* showing loss of balance due to severe intention tremors after being disturbed.](image)

![Fig. 4. Goat intoxicated spontaneously by *I. sericophylla* showing ataxia.](image)

![Fig. 5. Cerebellum from the goat observed in Figs. 3 and 4. There is a severe loss of Purkinje cells, which had been substituted by Bergmann glia (arrow). HE. ×10.](image)
2.3. Experimental reproduction of the disease

2.3.1. Plant consumption, clinical signs, gross and microscopic lesions

The daily and total dose of *I. sericophylla* or *I. riedelii* consumed by the experimental goats, the weight of the animals at the start and end of the experiment, the observation of the first clinical signs, and outcome of the intoxication are presented in Table 1.

In goats 1–5, 11 and 12, first clinical signs were observed 18–28 after ingestion (Table 1). They were characterized by depression, sometimes rough hair coat, and by spending more time to rise after the stand up test. Four–7 days later, goats 2–5, 11 and 12 showed intention tremors, and loss of equilibrium and falling to the side or backward after the head raising test. Other clinical signs, observed 7–15 days after the observation of the first signs, were incoordinated, hypermetric or lateral gait, wide based standing, spastic paresis, ataxia, abnormal postural reactions, nystagmus, inappetency, rough hair coat, weight loss, and, in goat 1, lateral recumbence. Goats 8 and 9 that consumed fresh *I. riedelii* as the only food showed rough hair coat and depression on day 11 after administration, in the following days they showed weight loss and weakness; they were found dead in the morning of day 23. No clinical signs were observed in goat 10 after the ingestion of fresh *I. riedelii* for 60 days (Table 1).

Goat 4 had diarrhea on the seventh day after the start of the plant administration. Despite continuation of the plant consumption the animal recovered 6 days later. Diarrhea was again observed on day 34, but the goat recovered in 3 days. Diarrhea was also observed in goat 5 from day 6 to day 14; on day 20 it had diarrhea again, but recovered 2 days later. Goat 11 had diarrhea 19 days after the plant ingestion, recovering spontaneously in 4 days. Goat no. 5 had diarrhea and depressed food consumption after 14 days of consumption, recovering spontaneously in 2 days. The four animals continued to consume the plant during the periods that they had diarrhea.

No clinical signs were observed in the control goats 6, 7, 13 and 14.

At necropsies of goats 1, 2, 5, 10, and 12 no significant gross lesions were observed. Goats 8 and 9 had ascites, dehydration, hydropericardium, emaciation, and serous atrophy of the pericardial fat.

Histologic lesions were vacuolation of the perikaria in neurons in all regions of the nervous system, but more prominent in the Purkinje cells (Fig. 6) of the cerebellum and in neurons of the cerebellar nuclei. Axonal spheroids were observed in the granular layer of the cerebellum, cerebellar white matter, cerebellar peduncles and cerebellar nuclei (Fig. 7). Cytoplasmic vacuolation was also observed in parenchymal cells of various organs including pancreatic acinar cells, hepatocytes, epithelial cells of the kidney, follicular epithelial cells of the thyroid gland, Kupffer cells and macrophages of the lymphatic tissues. Those lesions were observed in all goats, including goat 9 that had no clinical signs.

2.3.2. Electron microscopy

The electron microscopy of the Purkinje cells showed that the vacuoles observed under light microscopy correspond to dilate lysosomes surrounded by a single layered membrane (Fig. 8). The vacuoles were empty or contained membranous fragments or small amounts of a finely granular material. The axonal spheroids in the granular layer were filled with membrane bound residual bodies and some mitochondria (Fig. 8).

2.3.3. Lectin histochemical findings

The cytoplasm of Purkinje cells, and cytoplasm of multiple cells of cerebellar granular and molecular layers of
all affected goats examined (nos 1, 5, 9, and 10) stained strongly with Con A, WGA, and sWGA. These cells did not stain in corresponding slides from control animals. Lectin binding permitted the demonstration of stored material in numerous cells that appeared normal with the haematoxylin and eosin stain. Additionally, a partial weak reaction of Purkinje cells to UEA-I, PNA, RCA-I, SBA and DBA was observed in some cells from affected and control goats.

2.4. Identification of the active principle of the plants

The chemical analysis of the plants showed 0.11% swainsonine in I. sericophylla and 0.14% swainsonine in I. riedelii. Small amounts of calystegines B₁, B₂ and C₁ were also present in I. riedelii but no calystegines were detected in I. sericophylla.

3. Discussion

The results reported in this paper demonstrated that I. riedelii and I. sericophylla cause a swainsonine induced glycoprotein storage disease.

The spontaneous disease, caused by I. sericophylla in the municipality of São Sebastião do Umbuzeiro, and by I. riedelii in the municipality of Zabelé, state of Paraíba, is also observed in other farms in the region, but in most of these farms both plants are found together. The intoxication is seasonal occurring during the rainy season, which extends from December–January to April–May when the plants are growing. A similar disease is caused in the Brazilian semiarid by Ipomoea carnea subsp fistulosa (Tokarnia et al., 1960; Armién, 2000; Riet-Correa et al., 2003), which also contains swainsonine (de Balogh et al., 1999; Armién, 2000; Haraguchi et al., 2003; Medeiros et al., 2004). I. carnea subsp fistulosa also affects cattle and sheep (Tokarnia et al., 1960). I. riedelii and I. sericophylla occur only during the raining season, but I. carnea subsp fistulosa is a plant found mainly around water fountains or partially flooded areas during the whole year, and the intoxication can occur at any time of the year.

Nervous signs observed in the intoxication by Ipomoea spp. are characteristic of lesions in the cerebellum and brain stem. Hindquarter ataxia and spastic paresis are due to proprioceptive and upper motor neuron lesions, respectively; nystagmus, head tilting, decrease tonus of the tongue and/or lip are signs of cranial nerves impairment; and wide stance, loss of equilibrium, hypermetria and intention tremors are due to cerebellar lesions. Such clinical signs are similar to those observed in the intoxication by other swainsonine-containing plants, including I. carnea, S. carpinifolia, Swainsona spp., Oxytropis spp. and Astragalus spp. (James et al., 1970; James and Panter, 1989; Dorling et al., 1978; Hartley et al., 1989; de Balogh et al., 1999; Driemeier et al., 2000; Armién, 2000). In the experimental reproduction of the disease these signs were reversible in goats ingesting the plants for periods of 38–53 days, and showing clinical signs for 20–38 days (Table 1). On the other hand, many spontaneous cases had irreversible nervous signs, suggesting that the ingestion of the plant was for longer periods. In a recent experiment comparing the toxicity of I. riedelii and I. sericophylla, clinical signs became irreversible only in one goat out of four after the ingestion of I. riedelii containing 0.01% swainsonine for periods of 141 days, and in three goats ingesting I. sericophylla containing 0.05% swainsonine, during 75–127 days. Clinical signs started after 36–38 days of I. sericophylla ingestion and after 99–135 days of I. riedelii ingestion. This information is important for the control of the intoxication. If the animals are withdrawn from the area invaded by the plants after the first signs of intoxication they will recover. Also goats showing clinical signs for less than 30 days can recover. The cause of the irreversibility of the nervous signs after some period is the neuronal loss, which was observed, mainly in the cerebellum, in the goat intoxicated spontaneously by I. sericophylla. Because both toxic species of Ipomoea reported in this study only occur during the rainy season, which in the region extends from December–January to May–June is also probably that those irreversible signs are caused also by the consumption of the plant during more than one rainy season. One clinical sign not observed in the spontaneous cases is the diarrhea observed in goats 4, 5, 11 and 12 that ingested the dry plant. Because of the absence of diarrhea in the goats ingesting the fresh plant and also in the spontaneous cases, it is probably that this sign is caused by some chemical modification of the plant, during the drying process.

One probably important factor in the occurrence of the disease is the possibility of the goats developing a preference and apparent craving to graze these plants,
which was mentioned by the farmers. Everist (1981) reported that sheep develop a morbid appetite for *Swainsona* spp. and would eat nothing else when the plant is available. This morbid appetite, sometimes reported as addiction, had been reported also in the intoxication by other swainsonine-containing plants, including *Swainsona* spp. (Huxtable and Dorling, 1982), locoweeds (Ralphs et al., 1990, 1991), *I. carnea* (Tokarnia et al., 1960; de Balogh et al., 1999; Riet-Correia et al., 2003) and *S. carpinifolia* (Colodel et al., 2002b). In animals ingesting locoweeds it is suggested that this behavior is due to feed preference and habituation, not to a true addiction (Ralphs et al., 1990, 1991). Because of the potential development of a preference to graze *Ipomoea* spp. it should be recommended that affected goats not be allowed to return to areas where these plants occur after the clinical recuperation. It is very probable also that goats that consume *Ipomoea* spp. containing swainsonine induce other goats to ingest the plant by social facilitation, as occurs with locoweeds (Ralphs et al., 1994).

The pattern of lectin staining observed in Purkinje cells partially agrees with the results reported for locoweed, swainsonine, *S. carpinifolia* and *Ipomoea* spp. toxicosis and for mannosidosis in humans, cats, and calves (Alroy et al., 1985; de Balogh et al., 1999; Driemeier et al., 2000; Armien, 2000; Rodriguez Armesto et al., 2004). The reaction was clear to sWGA and WGA which indicates the presence of different calystegines in *Sida carpinifolia* poisonous, but not in the samples analyzed in 2003. The toxic effects of calystegines to animals had not been determined, but they are strong inhibitors of glycosidases, mainly β-glucosidase, and α-galactosidase. Recently, the occurrence of ataxia in cattle was associated with the presence of different calystegines in *Ipomoea* spp (Dorling et al., 2004).

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**References**


