Short communication

Spontaneous acute poisoning by *Crotalaria retusa* in sheep and biological control of this plant with sheep

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**ABSTRACT**

After the diagnosis of acute *Crotalaria retusa* poisoning, 21 healthy sheep from the affected flock were returned to the paddock and continued to consume the sprouting plants. Two years after returning these sheep to the paddock, very few plants were still alive, and after 3 years, no *C. retusa* plants were observed. The sheep had neither clinical signs nor biochemical alterations and delivered healthy lambs. It is concluded that resistant sheep can be used for the biological control of *C. retusa*.

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**Crotalaria retusa** is a weed native to Asia or coastal eastern Africa found in warm areas throughout the world. Acute poisoning by *C. retusa* in sheep (Nobre et al., 2005) and chronic poisoning in sheep (Dantas et al., 2004), cattle (Nobre et al., 2004a), and equids (Nobre et al., 2004b) occur in the semiarid range lands of Northeastern Brazil. Such poisoning is more frequent in equids, probably because the plant is more palatable to this species (Riet-Correa and Méndez, 2007) and because horses are more susceptible than cattle and sheep to monocrotaline poisoning (Cheeke, 1988, 1998). Recently, it was demonstrated that sheep are susceptible to acute intoxication by monocrotaline, with intoxication occurring after a single oral dose of approximately 205.2 mg/kg bw. However, sheep develop strong resistance to monocrotaline after the daily ingestion of non-toxic doses (136.8 mg/kg) (Anjos et al., 2010). Acute poisoning by *C. retusa* in sheep occurs after the ingestion of seeds, which contain higher concentrations of monocrotaline than other parts of the plant (Nobre et al., 2005; Anjos et al., 2010). Sheep ingesting high amounts of non-seeding plants apparently are not affected (Anjos et al., 2010). Sheep are also resistant to chronic *Senecio* spp. poisoning and have been used for the biological control of this plant (Méndez, 1993), although under certain conditions they can be intoxicated (Ilha et al., 2001; Schild et al., 2007).

The objective of this work was to document an outbreak of spontaneous acute poisoning by *C. retusa* in sheep and to determine whether it is possible to use resistant sheep for the biological control of this plant.

An outbreak of acute poisoning by *C. retusa* (Fig. 1) occurred in the municipality of Serra Negra do Norte in the state of Rio Grande do Norte, Brazil, between July and August 2007, in a flock of 150 Santa Inês and crossbred sheep. The flock had been transferred 20 days before the outbreak to an area in which a large amount of seeding *C. retusa* was present; this area had been used in previous years for rice, corn, and cassava cultivation. Thirty-four (22.7%) sheep were affected and died within approximately 30 days. The flock was moved from the paddock 20 days after the start of the outbreak; at this time 26 sheep had died, and 6 had clinical signs of poisoning. Two other sheep became affected after the withdrawal of the flock...
from the paddock. Of the 34 sheep that died, 5 were adults, and the others were 3–6 months old, including some nursing lambs. Males and females were equally affected. Clinical signs included abdominal distention with ascites, moderate jaundice, apathy and anorexia. The clinical course in most animals was 2–5 days, but one sheep died after a clinical manifestation period of 21 days. In the five sheep with acute clinical signs, the serum levels of aspartate aminotransferase (AST) and γ-glutamyltransferase (GGT) were elevated (Table 1).

Three sheep were necropsied, and their tissues were examined histologically. Sheep 1 and 2, which had displayed clinical signs for 3–4 days, had moderate jaundice of the subcutaneous tissue and petechial hemorrhages and ecchymoses of the subcutaneous tissue of the ventral and lateral regions of the abdomen and thorax. Moderate amounts of yellow translucent liquid were present in the abdominal and thoracic cavities. The liver was diffusely red with an enhanced lobular pattern and irregular red-dark areas alternating with pale areas (Fig. 2). Sheep 1 had

Fig. 1. Crotalaria retusa.
fibrin filaments in the capsular surface. Diffuse hemorrhages and edema were observed in the gall bladder (Fig. 2). Hemorrhages and edema were present in the mesentery and wall of the abomasums of both sheep. Sheep 3, which was found dead after a clinical course of 21 days, had some degree of autolysis. Ascites, hydropericardium, and an enhanced lobular pattern of the liver were observed at necropsy.

On histologic examination, the livers of Sheep 1 and 2 revealed diffuse periacinar necrosis and hemorrhage (Fig. 3) that occasionally extended to the mid-zone and was bordered by an area of swollen or vacuolated hepatocytes. Sheep 3 had fibrosis, mainly periportal; proliferation of epithelial bile duct cells; and megalocytosis. Different degrees of hemorrhage and edema were observed in lung, abomasum and intestine.

Three days after the diagnosis of the intoxication, 20 adult sheep and one ram from the affected flock were returned to the paddock, where most of the C. retusa had been consumed by the sheep. It was hypothesized that the surviving sheep had repeatedly consumed non-toxic doses of C. retusa and had become resistant, as suggested in previous experiments (Anjos et al., 2010), and therefore could consume the plant without risk of intoxication. The sheep stayed in the paddock until August 2010, during which period the paddock was inspected 11 times at regular intervals. At the two first visits, carried out one and three months after the reintroduction of the sheep into the paddock, the 20 sheep were bled for the determination of the serum activities of AST and GGT, which were within the normal ranges on both occasions.

**Table 1**

<table>
<thead>
<tr>
<th>Sheep</th>
<th>GGT (U/L)</th>
<th>AST (U/L)</th>
<th>Total bilirubin</th>
<th>Direct bilirubin</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>169</td>
<td>350</td>
<td>1.5</td>
<td>0.5</td>
</tr>
<tr>
<td>2</td>
<td>236</td>
<td>480</td>
<td>2.2</td>
<td>0.4</td>
</tr>
<tr>
<td>3</td>
<td>299</td>
<td>375</td>
<td>2.9</td>
<td>0.6</td>
</tr>
<tr>
<td>4</td>
<td>267</td>
<td>210</td>
<td>6.3</td>
<td>1.1</td>
</tr>
<tr>
<td>5</td>
<td>65</td>
<td>378</td>
<td>0.9</td>
<td>0.3</td>
</tr>
<tr>
<td>6</td>
<td>16</td>
<td>88</td>
<td>1.1</td>
<td>0.5</td>
</tr>
<tr>
<td>Normal values</td>
<td>20–52</td>
<td>60–280</td>
<td>1.71–8.55</td>
<td>0–4.61</td>
</tr>
</tbody>
</table>

*GGT = γ-glutamyltransferase, AST = aspartate aminotransferase.*

The paddock was flooded by severe rains in May 2008, and the sheep had to be removed. After 40 days, the 21 sheep were reintroduced into the paddock together with 100 other sheep. Over the next several months, a variable number of sheep was maintained in the paddock. During all visits, it was observed that the sheep continuously consumed the young leaves of the sprouting C. retusa, apparently preferentially to other plants. Due to the continuous consumption of the regrowth, the plants died, and increasing amounts of dry C. retusa were observed during the visits. The plants did not produce flowers or seeds, and after a period of 2 years, very few plants were still alive, and after 3 years no more plants were observed. Most ewes delivered healthy lambs during the experimental period. One ewe died with clinical signs characteristic of tetanus 10 days after lambing. This ewe was necropsied, and no gross or histologic lesions were observed in the liver. In a neighboring farm in a paddock grazed by cattle and invaded by C. retusa, the number of C. retusa plants varied during the 3-year period; the cattle remained healthy and apparently did not ingest the C. retusa.

The diagnosis of C. retusa poisoning was based on epidemiologic data, clinical signs and gross and histologic lesions, similar to those reported by Nobre et al. (2005). All cases were characteristic of acute poisoning, except Sheep 3, which survived for 21 days after observation of the first clinical signs and had lesions characteristic of chronic monocrotaline poisoning. Similar results have been observed experimentally in a group of eight sheep that were fed single doses of 3–4 g/kg body weight of C. retusa seeds. In those experiments, four sheep died acutely, two experienced chronic intoxication, and one had no clinical signs (Anjos et al., 2010).

The results obtained in this experiment, in which a flock continued to graze in a paddock invaded by C. retusa, demonstrate that sheep can be used for the biological control of this plant. However, some points have to be taken into account when considering the use of grazing sheep to control C. retusa. Sheep should be introduced into pastures with non-seeding C. retusa in order to allow sheep to adapt to the plant before being exposed to the mature
seeding plants with high monocrotaline levels. In a previous experiment, a sheep ingested large amounts of the aerial parts of *C. retusa* (285.6 kg in 270 days) without showing either clinical signs or lesions at the end of the experiment (Anjos et al., 2010). A method that could be used to induce resistance would be to introduce sheep gradually into pastures invaded by *C. retusa*, increasing the time spent in these pastures and the amount of plant ingested. It has been demonstrated that sheep ingesting low doses of *C. retusa* seeds develop resistance to doses that cause acute poisoning (Anjos et al., 2010). This biological control model for the control of *C. retusa* may be also applied to other Crotalaria species containing monocrotaline as the main alkaloid.

In accordance with Cheeke (1998) the pyrrolizidine alkaloid resistance of sheep and goats is largely a reflection of hepatic detoxifying enzymes. It is probably that resistance induced by the ingestion of non-toxic doses of monocrotaline is due to an adaptation of the cytochrome P450 enzyme system for the detoxification of monocrotaline or its metabolites in the liver.

**Conflict of interest**

The authors declare that there are no conflicts of interest.

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**References**


