Sudden Bovine Death from *Mascagnia rigida* in Northeastern Brazil

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ABSTRACT. One outbreak of sudden death occurred in the Agreste region of Paraíba, Brazil in a herd of 129 cattle. When the herd was driven from the area, 40 cattle had clinical signs characterized by instability, staggering and lying down or falling. Twenty cattle died and the other 20 recovered. Dry *Mascagnia rigida* collected in the paddock was administrated to 3 groups of 2 rabbits each at doses of 1.25, 2.5 or 5 g/kg bw. All rabbits ingesting 2.5 and 5 g/kg bw died acutely between 3 h 25 min and 16h 15 min after dosing. *M rigida* collected in another farm where the disease was not observed was given to 3 groups of 2 rabbits each at doses of 5, 10 or 20 g/kg bw. Only 1 rabbit died suddenly 3 h and 25 min after ingestion of 20 g/kg bw. These results tend to support *M rigida* as a cause of sudden death in cattle and rabbits They also demonstrate variations in the toxicity of the plant, which seems reflective of the variation in disease occurrence in different areas where it occurs.

It is estimated that sudden death following exercise are responsible for approximately 60% of the cattle losses from toxic plants in Brazil (1). At least 12 plant species, belonging to 3 families, cause sudden death: 4 species of *Palicourea* (Rubiaceae); 5 species of *Mascagnia* (Malpighiaceae); 2 species of *Arrabidaea* (Bignoniaceae); and *Pseudocalymma elegans* (Bignoneaceae) (1). *Palicourea marcgravii* is probably the most important Brazilian toxic plant for cattle. It causes severe losses over the whole country, except in the 3 states of the Southern Region and in the state of Mato Grosso do Sul (1). Three other plants that cause sudden death are important: *Mascagnia rigida* in Northeastern Brazil, and *Arrabidaea bilabiata* and *Arrabidaea japurensis* in the Amazonic region. *Palicourea juruana* and *Palicourea grandiflora* also cause sudden deaths in the Amazonic region, but they are less important (1). *Policourea aeneofusca* and *Mascagnia elegans* occur in the state of Pernambuco, northeastern Brazil, the first in the coastal region and the second in the semiarid region. *Mascagnia pubiflora* is an important toxic plant in Mato Grosso do Sul, Central-western Brazil, but it also occurs in the states of São Paulo, Goias and Minas Gerais. *Mascagnia* aff *rigida* causes sudden deaths in the state of Espirito Santo (1). Another not determined species of *Mascagnia* causes sudden death in the states of Santa Catarina and Rio Grande do Sul, southern Brazil (2). Intoxication from *Pseudocalyma elegans* has been reported only from the state of Rio de Janeiro (1)

Mascagnia rigida (Juss) Griseb is the most important toxic plant of Paraíba (3). It is reported to cause sudden death in cattle (4) and probably in goats (5). Experimentally, clinical signs could be observed 24-48 h after animals had been exposed to the plant, but in spontaneous outbreaks cattle ingesting the plant for long periods only had clinical signs when they were to exercised (1,4). Affected animals may suddenly staggers, tremble and fall with convulsions. Opisthotonos, bellowing and tachycardia with irregular heart rhythm are also observed. They may die in 1 min to 2 h after the first clinical signs. Animals less severely affected are reluctant to move and can recover if not forced to walk (1,4,6). Macroscopic lesions are not observed. The main histologic lesion, in some cattle, is the hydropic-vacuolar degeneration and necrosis of epithelial cells of the distal convoluted tubes in the kidney; most epithelial cells in the affected tubes had the disappearance of the cytoplasm and nuclear picnosis. In some animals heart muscle fibers had vacuolation and focal areas of intracellular edema, increased eosinophilia of the cytoplasm, and occasionally picnosis. Liver cells are occasionally swollen or vacuolated (1,4). The plant shows great variation in its toxicity; the toxic principle is unknown (1). Rabbits are susceptible to the intoxication and the fruits are approximately 20 times more toxic than the leaves (7).

The state of Paraíba, Northeastern Brazil is divided from West to East into 4 main regions: Zona da Mata, Agreste, Cariri (or Borborema), and Sertão. The regions of Cariri and Sertão, cover 82.7% of the area of the State and have a semiarid climate with low rainfall and large drought periods. The Zona da Mata has a wet tropical climate, and the Agreste is the region of transition between the Zona da Mata and the Cariri where the climate is changing gradually from wet tropical to semiarid. *Mascagnia rigida* is found in the whole state, except in the Zona da Mata, which represents 7.6% of the territory (3); however, the occurrence of sudden death due to intoxication is variable between regions and between farms in the same region.

The objectives of this paper are to report an outbreak of *M rigida* intoxication in the Agreste region and to compare the toxicity of the plant causing the outbreak with plant from other regions where the disease does not occur.

CASE REPORT

An outbreak of sudden death occurred on a farm in the municipality of Alagoa Nova, in the Agreste region of Paraíba, in a herd of 129 cattle. When the herd was driven from the area, 40 cattle had clinical signs characterized by instability, staggering, breathing difficulties, and lying down or falling. Twenty cattle died. The other 20 were reluctant to move, but recovered when left alone. After the outbreak the pastures were inspected. *Mascagnia rigida* (Fig 1) was found in the paddock where the disease occurred. A voucher specimen was collected and identified as Brazil, Paraíba, Medeiros et al, Nº 21112 (PEL).

EXPERIMENTAL PROCEDURE

Mascagnia rigida was collected in the Agreste region, from the paddock where the disease was observed. Other samples of the plant were collected from a farm in the municipality of

São José do Bom Fim, in the Sertão region, where the disease had not been observed recently. At the time of collection in June 2000, many plants were blooming and others were seeding. After collection the plants were dried in the shade and ground.

Dried powered leaves were suspended in water and given by stomach tube to adult rabbits weighting 1,2-3 kg. The plants from the Agreste region were dosed to 3 groups of 2 rabbits each. Each group received doses of 5 g/kg bw, 2.5 g/kg bw or 1.25 g/kg bw. The plants collected in the Sertão were also dosed to 3 groups of 2 rabbits each. Doses given to each of the latter group were 5 g/kg bw, 10 g/kg bw or 20 g/kg bw. The dose of 10 g/kg bw was divided in 2 doses and the dose of 20 g/kg into 3 doses. These doses were given at intervals of 8 h.

All animals treated with 2.5 or 5 g/kg bw of the *M rigida* collected on the farm where the outbreak occurred developed clinical signs between 3 h 24 min and 15 h 50 min after dosing. These clinical effects were characterized by general uncontrolled violent movements followed by falling with dyspnea, a few shrieks and death in 1-3 min (Table 1). Enlarged and congestive livers were observed at necropsy in rabbits 2 and 4 (Table 1). On histologic examination, mild hepatic degeneration and congestion were observed in the livers of rabbits 1, 2 and 4. The 2 rabbits treated with 1.25 g/kg bw had no clinical signs.

Of all of the rabbits treated with the plants collected in the Sertão region, only ½ rabbits dosed with 20 g/kg died (Table 1). Clinical signs were similar to those previously mentioned, and the rabbit died 3 h and 25 min after dosing. No significant lesions were observed on necropsy of this rabbit.

Table 1. Experimental intoxication in rabbits with dried powered leaves of Mascagnia rigida with plants collected on a farm in the Agreste region where sudden deaths were observed in cattle, and on another farm in the Sertão region were sudden deaths were not observed.

Rabbit No	Weight (kg)	Region plant collected	Dose (g/kg bw)	Time between dosing and clinical signs	Clinical period
1	2.7	Agreste	5	10 h 50 min	1min
2	3	Agreste	5	7 h 24 min	3 min
3	2.9	Agreste	5	3 h 24 min	1 min
4	2.6	Agreste	2.5	6 h 55 min	2 min
5	2.7	Agreste	2.5	15 h 15 min	3 min
6	2.5	Agreste	1.25	No signs	None
7	2.9	Agreste	1.25	No signs	None
8	2.8	Sertão	5	No signs	None
9	2.1	Sertão	5	No signs	None
10	1.2	Sertão	10	No signs	None
11	1.25	Sertão	10	No signs	None
12	1.25	Sertão	20	3 h 27 min	2 min
13	1.2	Sertão	20	No signs	None

DISCUSSION

The clinical signs presented by the affected cattle, and the experimental production of sudden deaths in the experimental rabbits with *M rigida* collected on the farm, support that the disease was caused by ingestion of this plant. The clinical signs and rapid development of the intoxication observed in the experimental rabbits are very similar than those caused in rabbits by other plants that cause sudden death in cattle in Brazil (1) and by the ingestion of fluoracetate in many species (8). The active principle of *M rigida* has not been identified, but the clinical signs in cattle and rabbits suggest the toxic principle may be fluoracetate.

Figure 1. Masagnia rigida with fruits and flowers. Municipality of Alagoa Grande, State of Paraíba, Brazil.

In the outbreak being reported, 50% of cattle with clinical signs recovered, and the signs were only observed after exercise. These aspects of the intoxication by *M rigida* are different from the intoxication produced by *Palicourea* spp, mainly *Palicourea marcgravii*. In the later, all affected cattle die, and clinical signs appear without previous exercise (1). These differences between the 2 plant species could be due to differences in the active principle or in its concentration.

A large variation was found in the toxicity of *M rigida* collected from the 2 different places. The plants from the paddock where the outbreak occurred were nearly 8 times more toxic than the plants collected where the disease did not occur. This wide variation in toxicity would seem responsible for the varied occurrence of the disease in the different areas of the state of Paraíba where *M rigida* grows. In previous experiments with *M rigida* collected in the state of Ceara, rabbits died with doses of 4 mg/kg bw, but in experiments with cattle wide variations in *M rigida* toxicity were also observed. Some cattle become intoxicated with single doses of 12.5 to 50 g/kg bw, but others had no clinical effects from single doses of 10 to 94 g/kg bw (4,6). Also repeated doses had variations in toxicity; 1 bovine dosed with 5 g/kg bw daily died in 3 d, but another ingesting the same daily dose was not affected after 30 d (4). The reasons for such variations in *M rigida* toxicity are not known, but this is an important issue to be considered in the epidemiology and control of these intoxications. The only current control measure is to move the animals, with minimal exercise, to an area free of the plant and to wait 8-14 d for further transportation of the herd (4).

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MYCOTOXINS IN FOOD

The Joint FAO/WHO Expert Committee on Food Additives met in Geneva February 6-15, 2001 to evaluate certain mycotoxins that may contaminate food. The committee was charged with assessing and characterizing the risks associated with consuming foods contaminated by the mycotoxins under consideration. Evaluations were made on aflatoxin M_1, fumonisins, (FB_1, FB_2, FB_3), ochratoxin A, and trichothecenes (deoxynivalenol, T-2 toxin and HT-2 toxin). The report addressed several concerns about each mycotoxin: explanations of the mycotoxin, absorption through excretion, toxicological studies, and final evaluation. Along with the mycotoxin evaluations, the committee put forth general considerations on analytical methods, sampling, associated intake issues, and control. A report on the meeting can be found at http://www.fao.org/ES/esn/jecfa/jecfa56.pdf.

In particular, good surveillance data should be generated using validated analytical methods to ensure reliable results. Methods used should be validated though collaborative studies of analytical performance; however, the committee recognized that it may not always be possible to use a validated method due to expense or an official method being irreconcilable to a particular toxin-matrix combination. The committee also stressed the importance of laboratories' participation in inter-laboratory comparison studies to ensure analytical quality assurance.

Accurate determination of the occurrence of mycotoxins in foods requires practical, economically feasible sampling plans. To date, there is not enough information on sampling variability and surveillance data to ensure effectiveness. The committee recommended a clearly defined sampling plan with samples from selected food populations taken from defined locations. Similarly, the committee recommended that food consumption data be taken from national food balance sheets. The estimate of relative health risks associated with specific proposed maximum limits for a particular mycotoxin was calculated by combining mean data on food consumption with weighted mean contamination levels.

Also supplied were general principles on the prevention and control of mycotoxins. Prior to harvest, appropriate agricultural practices should be applied including breeding plants to resist fungus and competitive exclusion with the use of nontoxigenic strains in the field. Postharvest, it is important to dry commodities and keep them dry (below 0.70 water activity) during storage to prevent fungal growth and mycotoxin formation. Cleaning grains and keeping them cool during storage also reduces insect and fungal growth.

Carcinogenic potency to the liver (cancers/year/100,000 persons/ng/kg of body weight/day) of aflatoxin M_1 estimated from epidemiological and toxicological studies on aflatoxin B_1 was 0.001 and 0.03 in humans, respectively. It was concluded that the additional risks for liver cancer predicted with use of the proposed maximum levels of aflatoxin M_1 of 0.05 and 0.5 ug/kg are very small ie 29 cancers/1000 million persons/year.

The provisional maximum tolerable daily intake (PMTDI) for fumonisins B_1, B_2, and B_3 alone or in combination was 2 ug/kg body weigh/day on the basis of NOEL of 0.2 mg/kg of body weigh/day and a safely factor of 100. For deoxynivalenol, the PMTDI was 1 ug/kg of body weigh/day and a safety factory of 100. For T-2 and HT-2 toxins, alone or in combination, the PMTDI was 0.060 mg/kg of body weigh/day on the basis of the LOEL of 0.029 mg/kg of body weight/day and a safety factor of 500.

For ochratoxin A, the Committee retained the previously establish provisional tolerable PTWI of 10 ng/kg body weight/week, pending the result of ongoing studies on the mechanisms of nephrotoxicity and carcinogenicity, and recommended a further review of ochratoxin A in 2004.

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