J. Vet Malaysia (1990) 2(2): 135-138

INDIVIDUAL VARIATION IN RESISTANCE TO *BRACHIARIA DECUMBENS* TOXICITY IN SHEEP

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SUMMARY: The offspring of rams which had shown resistance to *Brachiaria decumbens* poisoning in earlier experiments exhibited variability in response when fed the grass. Three out of seven sheep showed no ill effects at all. The affected individuals showed slight pathological changes in the liver with no signs of jaundice and neurological disorders even after 8 weeks of grazing on *B. decumbens*. These preliminary observations suggest that there may be a genetic factor producing resistance which have been transmitted by the sires to their offsprings.

Key words: *Brachiaria decumbens*, toxicity, sheep

INTRODUCTION

Signal grass (*Brachiaria decumbens*), widely grown on livestock farms in Malaysia, has been confirmed to be hepatotoxic and nephrotoxic to sheep (Abas Mazni *et al.* 1983, Salam Abdullah, 1987, Salam Abdullah, *et al* 1987, Noordin, 1988). Incidences of toxicity caused by this species of grass in sheep and goats have also been reported elsewhere (Briton and Pattridge, 1941; Opasina, 1985). Ruminal stasis has been shown to occur within 3 weeks and death normally occurred within 4 to 6 weeks after grazing on this grass (Salam Abdullah *et al.* 1988; Noordin 1988). The affected sheep also showed neurological disorders such as stamping of fore-legs, stargazing, incoordination, head pressing against the wall and circling movement (Salam Abdullah *et al.*, 1989).

Although *B. decumbens* is highly toxic to sheep, not all sheep developed toxic signs even after eight weeks of grazing on this grass. This paper describes the effects of feeding *B. decumbens* to the offsprings of these sheep which had shown resistance towards poisoning by this grass.

MATERIALS AND METHODS

Experimental Animals

The sheep used in this experiment were offsprings obtained by mating three rams which had previously shown resistance towards *B. decumbens* toxicity with local Malin (Malaysian Indigenous) ewes. Seven one-year-old male (3) and female (4) sheep were allowed to graze freely on *B. decumbens* pasture for 8 weeks. Water and mineral licks were provided *ad libitum*. Their body weights were recorded at regular intervals.

Samples Collection and Analysis

Blood samples were collected weekly for the enzyme serum aspartate aminotransferase analysis. The enzyme assay was performed using Roche Diagnostic Kits (F. Hoffman-La Roche Co. Ltd. Diagnostica, Bassel, Switzerland) and split-beam spectrophotometer (Spectronic 1001, Wilton Ray Co. Rochester, New York).

Gross and Histopathology

Two sheep showing toxic symptoms were sacrificed for post mortem examination. Samples of the liver and the skin of the affected parts were taken for histopathological examination.

RESULTS

The initial and final body weight of the sheep and the levels of serum aspartate aminotransferase for each sheep during the experimental period are shown in Table 1. Four sheep (one male and three females) showed high levels of enzyme serum aspartate aminotransferase and photosensitization reactions on the ears, eyelids, around the muzzles and nose after seven weeks of grazing on *B. decumbens*. One of the four affected sheep which showed evidence of jaundice, emaciation and general weakness at the 7th week died a few days later. None of the affected sheep showed any signs of neurological disorders.

The remaining three sheep (two males and one female) did not show any signs of *B. decumbens* toxicity and in fact they had gained between 3 and 5 kg in body weight within eight weeks. The levels of enzyme serum aspartate aminotransferase in these three sheep were within normal limits which is about 122 ± 5 IU.

Autopsy findings of the two affected sheep includes poor body condition with skin around the muzzle, nose eyes thickened and scabby with purulent discharge. Removal of the scabs revealed raw and bleeding surfaces. The livers were moderately enlarged with numerous small pale areas of about 1-2cm diameter distributed diffusely throughout the liver parenchyma. However, none of the carcasses were icteric.

Histologically, most perilobular hepatocytes particularly around the portal triads were severely degenerated. The cytoplasm were eosinophilic while the nuclei were hyperchromatic. Individual necrosis were observed among the hepatocytes in these areas. The central lobular hepatocytes were less affected but some were showing hydropic degeneration.

Thick scabs were found covering the severely thickened epidermis of the affected skin and severe hydropic degeneration was observed in some of the epidermal cells.

DISCUSSION

The symptoms and histopathological changes in the liver, kidney and brain of *B. decumbens* toxicated sheep have already been described (Salam Abdullah *et al.* 1988, 1989). However, those observed in the affected animals in the present experiment were less severe compared to the previous reports. For example, neurological disorders were absent and the carcase of those autopsied were not icteric. Some aspects of the pathological lesions observed in the present study were also different and milder than those observed and reported earlier by Zamri-Saad *et al.*, (1987).

In addition to the milder pathological changes in the liver and the absence of jaundice and neurological disorders in the affected sheep of the present study three out of seven did not show any symptom indicative of *B. decumbens* toxicity at all. In fact they gained weight normally purely feeding on the grass without any feed supplementation. These preliminary

observations suggest that there may be factors(s) producing resistance towards *B. decumbens* toxicity in the offsprings of rams which were not affected by *B. decumbens* toxicity in our earlier studies. This factor which may be genetically controlled must have been transmitted by the sires to their offsprings. The variation in susceptibility of individual members of a particular strain or group of animals of a particular breed to any toxic compound has been

TABLE 1
Body weight and levels of serum aspartate aminotransferase of sheep grazing on *B. decumbens*

Sheep No.	Sex	Body weight (kg)		Serum Aspartate Aminotransferase(IU)							
		Initial	Final	Week							
				1	2	3	4	5	6	7	8
3142*	Male	11.0	14.6	54.65	54.00	68.79	83.66	87.42	42.54	64.70	48.66
3155	Female	9.5	9.8	54.56	34.05	67.64	58.82	79.83	92.00	549.20	Died
3153	Female	13.5	10.0	42.69	47.99	61.04	198.60	158.30	142.50	245.40	697.20
3144*	Female	16.5	19.6	47.84	57.99	55.87	53.24	70.12	82.34	63.24	97.74
3149	Male	11.0	13.0	45.76	45.78	47.96	82.62	64.77	164.16	115.96	149.28
3160*	Male	14.5	19.4	50.94	54.67	32.04	53.19	91.27	86.64	40.08	86.52
3151	Female	11.0	12.2	60.12	31.24	29.40	37.49	7192.0	36.39	172.24	618.60

**Sheep showing resistance to B. decumbens toxicity*

reported to be greatly influenced by the genetically determined structure of the protein receptor because most toxic compounds must ultimately combine with their target receptor in order to produce their toxic manifestations (Doull, 1975). Similar explanation has been suggested for the development of resistance to the coumarin rodenticide warfarin in rats in Scotland and Denmark (Doull, 1975). The decreased response to *B. decumbens* toxicity manifested by the animals used in the present study may be similarly explained. Further investigations involving more animals and their backcrosses are in progress to test the proposed hypothesis.

ACKNOWLEDGEMENT

The authors wish to thank Mr. Shokri Jusoh and Mr. Zainal Abidin Jamin for their technical assistance. This study is supported by International Foundation for Science Research Grant No. B/1132-1.

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RINGKASAN

Anak kepada bebiri jantan yang telah menunjukkan ketahanan terhadap keracunan Brachiaria decumbens dalam ujikaji awal menunjukkan kepelbagaian gerak balas apabila diberi makan rumput ini. Tiga daripada 7 bebiri tidak menunjukkan kesan buruk langsung. Individu yang terkesan menunjukkan sedikit perubahan patologi dalam hati tanpa petanda jaundis atau gangguan neurologi, walaupun mereka telah meragut B. decumbens selama 8 minggu. Cerapan awal ini menyarankan kemungkinan adanya faktor genetik menghasilkan ketahanan yang telah dipindahkan daripada baka jantan kepada anak mereka.