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# NATURAL TOXICANTS AFFECTING ANIMAL HEALTH AND PRODUCTION

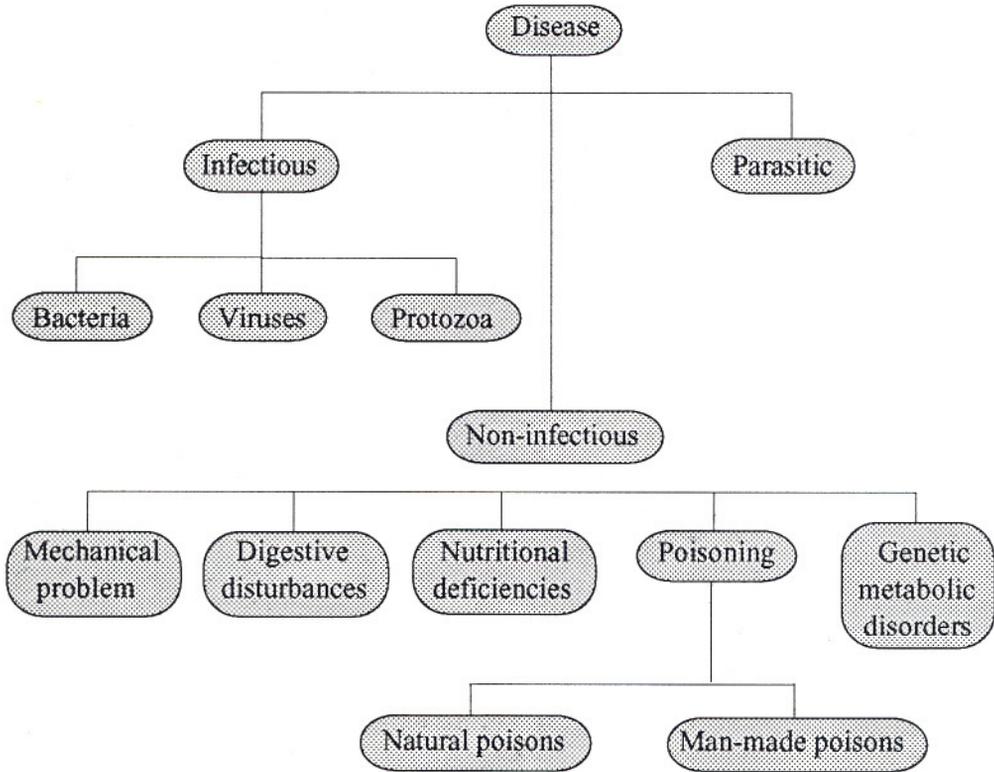
by

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## Introduction

Disease is one of the major constraints in animal production and causes a loss of capital in the form of direct and latent costs. The outbreak of haemorrhagic septicaemia in 1989 in Kinta Valley alone resulted in an estimated loss of RM500,000.00. The direct cost is the actual loss of capital in terms of veterinary therapy, culling and mortality. The direct cost is easily calculated and rapidly recognised by the farmers. However, it is much more difficult to quantify the real latent cost of diminished production due to physical malformations, abortions, photosensitisation and reduction of growth. It is impossible to assign a precise economic value of these losses for the livestock industry.

Livestock diseases may be infectious, non-infectious or parasitic (Figure 1) and this lecture is confined to the non-infectious diseases caused by natural toxicants or poisons. A number of fatalities in domestic animals involving natural toxicants have been reported in local veterinary journals (Arokiasamy, 1968; Seiler *et al.*, 1979; Salam Abdullah and Lee, 1981; Abas Mazni *et al.*, 1983; Salam Abdullah *et al.*, 1987). However, these few available reports may not reflect accurately the number of incidents involving natural toxicants in this country.



**Figure 1.** General classification of livestock disease

### **Types of natural toxicants**

Toxicants or poisons are naturally-occurring or man-made chemicals which, following their entry via any route and in relatively small quantities into the body, produce biochemical abnormalities and/or physical lesions (disease conditions).

Natural toxicants or biotoxins affecting animal health and production are:

- Zootoxins -(animal origin)
- Bacterial toxins (bacterial origin)
- Mycotoxins (fungal origin)
- Phytotoxins (plant origin)
- Phycotoxins (algae toxins)

Since it is not possible to review all the above toxins in this lecture, I will confine the discussion to mycotoxins and phytotoxins. These two toxins often cause severe poisoning and death of animals in this country but were somewhat neglected by local veterinarians.

## Mycotoxins

Mycotoxins are secondary toxic metabolites of fungi. These fungal toxins are capable of producing acute or chronic toxic effects in animals and man at different levels of exposure. Mycotoxins are carcinogenic, mutagenic, teratogenic and oestrogenic. Toxic syndromes following the intake of mycotoxins by animals and man are known as "mycotoxicoses".

Although mycotoxicoses have been known for a long time("Holy fire" in the middle ages in Europe) caused by the fungus *Claviceps purpurea*, these diseases remained neglected until the discovery of aflatoxin in the early 1960's during which 100,000 turkey poults were lost due to a toxic peanut meal (Sargeant *et al.*, 1962). The discovery of aflatoxin has generated a lot of interest in scientific research on mycotoxins since many foodstuffs and foodstuff ingredients may become contaminated with mycotoxins. To date a few hundred mycotoxins produced by the fungi *Aspergillus*, *Penicillium* and *Fusarium* species have been discovered (Van Egmond *et al.*, 1990).

The earliest recognised mycotoxicosis in recorded history was ergotism. Outbreaks of gangrenous ergotism and convulsion in domestic animals in the sixteenth century were linked to fungal contamination of the cereals (Radeleff, 1970).

Table 1 summarises some mycotoxins of importance in veterinary medicine and the diseases produced by these natural toxins in various species of domestic animals.

**Table 1.** Mycotoxinoses of importance in domestic animals

Toxins	Fungus	Species affected	Disease produced
Aflatoxins	<i>Aspergillus flavus</i> ; <i>Aspergillus parasiticus</i>	duck, poultry, cattle, swine, dog, horse.	Aflatoxicosis
Ochratoxin A	<i>Aspergillus ochraceus</i>	swine	Renal tubular necrosis
Sporodesmin	<i>Pythomyces chartarum</i>	sheep, cattle	Facial eczema
Zearalenone (F-2)	<i>Fusarium graminearum</i>	swine	Reproductive disorders
Trichothecene T-2 toxin	<i>Fusarium tricinctum</i>	cattle, swine, poultry	Gastroenteritis, haemorrhage

### Nature of the diseases

From a number of field outbreaks in domestic animals, mycotoxicosis is characterised as:

- a veterinary health problem whose true cause is not readily identified.
- neither an infectious nor a contagious disease.
- not responding to the treatments with antibiotics or any other drugs.
- linked with the consumption of certain feedstuff with signs of fungal growth.

### Conditions favourable for fungal growth

It has been estimated that 1 percent of the world supply of grain and oilseed is rendered unfit due to fungal invasion. Fungal invasion of foods and feeds may produce either toxic or non-toxic effects. The following storage factors are favourable in the initiation and development of fungal growth:

- A proper substrate (carbohydrate in a readily available form).
- Moisture in the grain (10.0 to 18%)
- Relative humidity (10%) in the storage atmosphere

- Adequate temperature for growth. It varies with the fungi (*Aspergillus flavus* can elaborate toxin from 12 - 47° and some *fusaria* may be active at or near freezing temperatures.
- A supply of oxygen.

The effect of different storage conditions on the production of aflatoxin in compound poultry feeds has been studied by a group of Indian scientists recently. Their results showed that none of the samples (240) which they have collected at random from various poultry farms were free from aflatoxins and levels in different samples ranged between 7 and 11,600 ppm (Jindal *et al.*, 1993). The duration of storage and aflatoxin production were directly proportional. Levels of aflatoxin were higher in the temperature range of 22° - 24°C and in the humidity range of 76-80% (Table 2).

**Table 2.** Effect of different storage factors on aflatoxin levels in feed samples

Factors		Mean aflatoxin content (ppb)
Period of storage (days)	1-10	291.70 (142) <sup>a</sup>
	11-20	557.47 (78)
	21-30	827.55 (14)
	31-40	908.28 (6)
Temperature (°C)	19-21	596.18 (9)
	22-24	840.06 (124)
	25-27	809.81 (33)
	28-30	658.67 (12)
	31-33	588.43 (25)
	34-36	528.35 (21)
Humidity (%)	37-39	502.25 (16)
	56-60	350.42 (25)
	61-65	580.65 (98)
	66-70	698.18 (89)
	71-75	793.25 (16)
Floor condition	76-80	808.75 (12)
	Pucca	528.76 (196)
Ventilation	Kacha	863.74 (44)
	Ventilated	564.42 (167)
	Non-ventilated	728.03 (73)

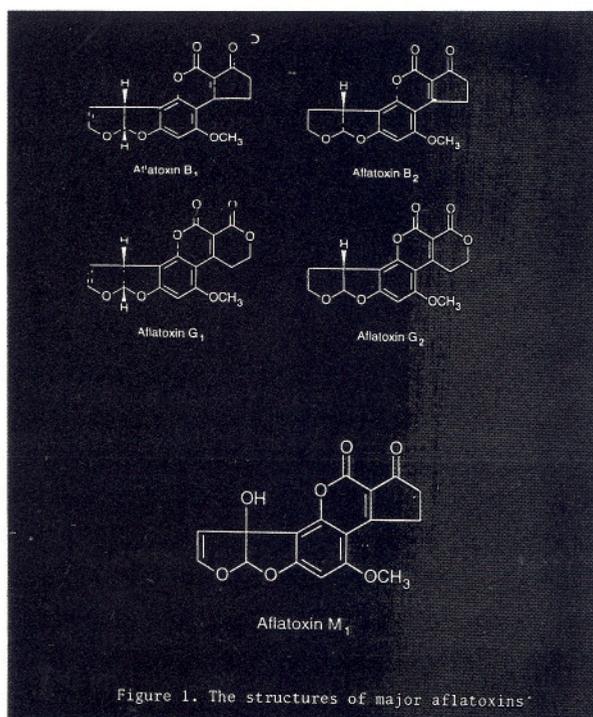
<sup>a</sup>Number in parentheses indicate number of samples in each category. From Jindal *et al.* (1993).

## Naturally occurring mycotoxins in feedstuffs and foods

The most commonly encountered mycotoxins in feedstuffs and food are aflatoxins, zearalenone, deoxynivalenol (vomitoxin), and fumonisins (Richard *et al.*, 1993). These mycotoxins have been proven as causes of, or implicated in mycotoxicoses of either animals or humans. Only aflatoxins and zearalenone will be reviewed in this lecture.

### Aflatoxins

Aflatoxins are fluorescent compounds chemically classified as difurocoumarolactones, biosynthesised by the producing fungi via the pollyketide pathway (Smith and Moss, 1985). Aflatoxins are produced by two major species of *Aspergillus*, *A. flavus* and *A. parasiticus*. The chemical structures of major aflatoxins found in feedstuffs and foods are shown in Figure 2. The most potent and most frequently detected is aflatoxins B<sub>1</sub>. This aflatoxin is considered to be the most potent carcinogen known. Its metabolite called, aflatoxin M<sub>1</sub> which occurs in various animal tissues and fluids including milk is also carcinogenic.



**Figure 2.** The structures of major aflatoxins

## Action levels of aflatoxin

The toxic effects of aflatoxin are dose and time dependent. Susceptibility and manifestation of aflatoxicosis vary with the species and breed of animals. The dietary levels of aflatoxin toxic to various species and the duration of feeding required to produce toxicity symptoms are summarised in Table 3.

The action level of aflatoxin has been revised by the Food and Drug Administration (FDA) from 30 ppb to 20 ppb for all foods, including animal feeds. The revision was based on the FDA's improved analytical capability and the agency's aim of limiting aflatoxin exposure to the lowest possible level (Price *et al.*, 1993).

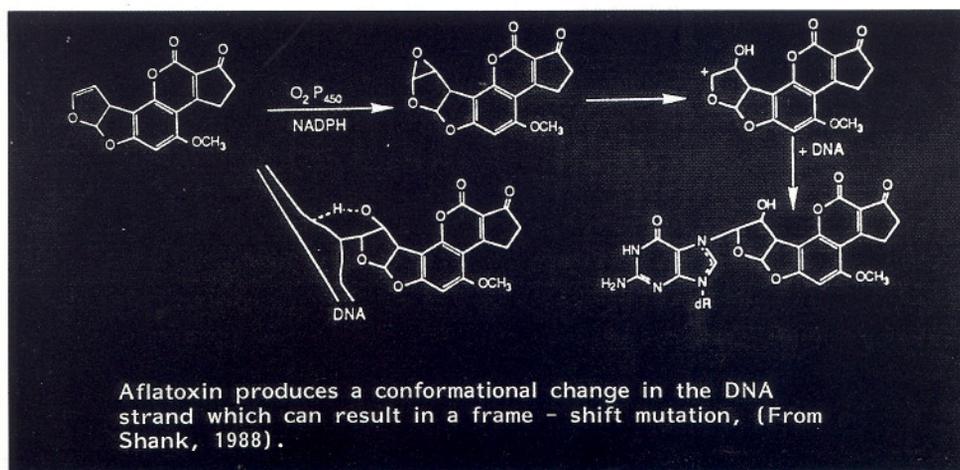
**Table 3.** Dietary aflatoxin levels causing toxicity in domestic animals

Animal	Aflatoxin level, (ppm)	Feeding time	Toxicity symptoms
Calves (Weaning)	2.2 0.22-0.44	16 wks 16 wks	Death Growth suppression, liver damage
Steers (2 yrs old)	0.22-0.66	20 wks	Liver damage
Cows (heifers)	2.4	7 mo	Liver damage, clinical illness
Pigs (6 weeks old)	0.41-0.69	3-6 mo	Growth suppression, liver damage
Chickens (1 week old)	0.84	10 wks	Growth suppression liver damage
Ducklings	0.30	6 wks	Death, liver damage

From Wogan (1968)

## Mechanism of action of aflatoxin

Many of the clinical signs and postmortem lesions in aflatoxicosis such as hepatic necrosis and fatty changes have been linked with the impairment of protein synthesis and ability to mobilise fat due to the depression of messenger - RNA synthesis (Buck *et al.*, 1976). The carcinogenic effect of aflatoxin B<sub>1</sub> is due to the metabolic activation of this mycotoxin (Shank, 1988). Aflatoxin B<sub>1</sub>, like most environmental carcinogens are lipophilic and stable compounds which are difficult to be excreted by the body. The oxidative enzyme, cytochrome P450 monooxygenase isozymes bound to the endoplasmic membrane of the liver play a critical role in the metabolic activation of aflatoxin B<sub>1</sub>. When an atom of oxygen is added to the aflatoxin B<sub>1</sub>, an electron is lost from one of the carbon atoms in aflatoxin B<sub>1</sub>, converting it to an electrophilic carcinogen. Reaction of this electrophile with protein and nucleic acid also inactivates the electrophile and is detrimental to the survival of the cell. It results in either mutation or death of the cell, Figure 3 shows the conformational change in the DNA strand by aflatoxin B<sub>1</sub> which can result in a frame shift mutation.



**Figure 3.** Aflatoxin produces a conformational change in the DNA strand which can result in a frame - shift mutation, (from Shank, 1988).

## Analysis of aflatoxins

The condition or disease caused by these toxins are not pathognomonic, thus, to determine the cause of the specific condition or disease requires confirming the presence of the toxin (s) in a representative sample of the feed, food, tissue or fluid.

The procedure for the analysis of aflatoxins in feedstuff or food samples is shown in Figure 4. Briefly, the samples must be adequately ground and thoroughly mixed before subsamples are taken for the extraction with chloroform. A portion of the extract is placed on a column of silica gel and the lipids and pigments are eluted from the column before elation of the aflatoxins. After the aflatoxins are eluted they can be analysed by either TLC, HPLC or gas chromatography - mass spectrometry (GC-MS).

## Treatment

There is no specific treatment for aflatoxicosis. Easily digested low-fat diets, lipotropic agents and avoidance of stress should be beneficial in cases where aflatoxin-induced liver damage has occurred.

## Zearalenone

It is a non-steroidal estrogenic mycotoxin. The structures of this mycotoxin and its major metabolites ( $\alpha$ -zearalenol and  $\beta$ -zearalenol) are shown in Figure 5. Stob *et al.* (1962) isolated and crystallised this mycotoxin, a resorcylic acid lactone from laboratory cultures of "*Fusarium roseum*" (*F. graminearum*). Another zearalenone producing fungus is *F. culmorum*. These two species of fungus are distributed worldwide (Marasas *et al.*, 1984).

Zearalenone is heat-stable and insoluble in water. It persists in animal feeds and human foods prepared from contaminated grain. Zearalenone is the only known phytoestrogen produced by a fungus and is unique in that one of its derivatives,  $\alpha$ -zearalenol, is useful commercially as a growth promoter in cattle.

The estrogenic and anabolic effects of zearalenone is due to the increase in weight and activity of the uterus (Buck *et al.*, 1976).

### Sample Preparation

1 - to 2 kg samples ground to pass a No. 20 sieve.  
Mix thoroughly.

50 - g subsample

Place in 500-mL, glass-stoppered Erlenmeyer flask with 250 mL of H<sub>2</sub>O,  
25 g of diatomaceous earth, and 250 mL of CHCl<sub>3</sub>. Shake for 30 min.

Filter and collect first 50 mL extract.  
Place on column.

### Column Preparation

Place glass wool loosely in bottom of 22 - x 300-mm chromatographic tube and  
add 5 g of anhydrous Na<sub>2</sub>SO<sub>4</sub> and then CHCl<sub>3</sub> until tube is half-full.

Add 10 g of silica gel 60. Wash sides with CHCl<sub>3</sub> and  
drain to aid settling of silica gel, leaving - 5 to 7 cm above silica gel.

Carefully add 15 g of anhydrous Na<sub>2</sub>SO<sub>4</sub>. Drain to top of Na<sub>2</sub>SO<sub>4</sub>

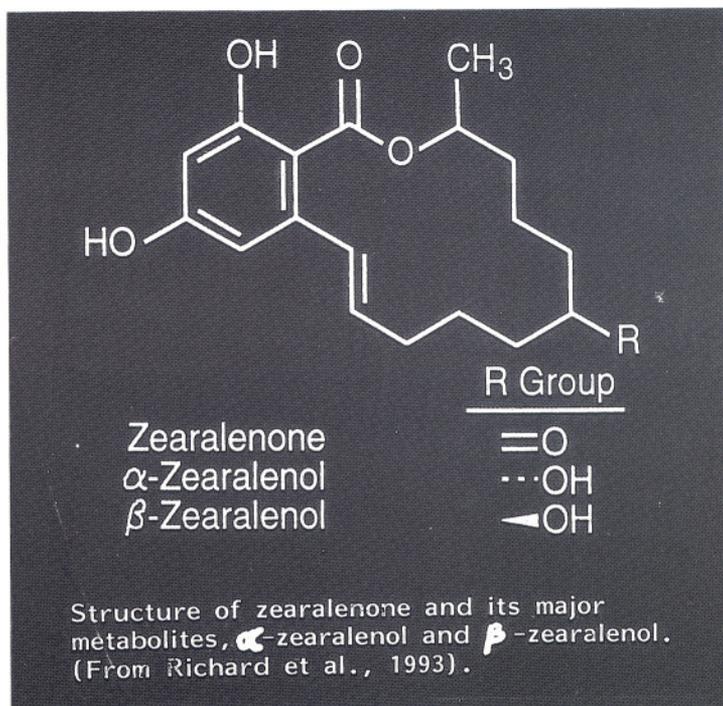
Add 50 mL of extract from above

Wash with 150 mL of hexane,  
followed by 150 mL of anhydrous ether.  
Discard

Elute aflatoxins with 150 mL of MeOH-CHCl<sub>3</sub> (3:97).  
Collect entire fraction

Evaporate to dryness and quantitatively transfer to vial.  
Evaporate solvent and seal vial until use in quantitative analysis.

**Figure 4.** Diagrammatic procedure for preparation, extraction, and cleanup of aflatoxins from grains or feeds for analysis.



**Figure 5.** Structure of zearalenone and its major metabolites,  $\alpha$ -zearalenol and  $\beta$ -zearalenol (from Richard *et al.*, 1993)

### Phytotoxins

Phytotoxins are complex molecules of high toxicity elaborated by certain plants. It is not fully understood why certain plants contain compounds which are highly toxic to animals and man. It has been suggested that the toxic compounds may be:

- intermediates of plant metabolic stores of energy, or are necessary to the plant in some other ways;
- incidental metabolic products which have no significance to the plant itself;
- intermediates of metabolism representing unwanted excretory products of the plants; and
- a protection to the plant, by making it unpalatable, or in extreme cases poisonous, restricting its use as food by animals and man.

There are about 7000 species of poisonous plants in the world (Culvenor, 1970), and more species of the poisonous plants are found in the hot humid tropical region than in temperate countries (Morton, 1971). Definitely it is not possible to discuss all of them within an hour and therefore I will confine my lecture this morning to only one of them. The toxic plant selected for the discussion today is Signal grass (*Brachiaria decumbens*).

### Signal grass (*Brachiaria decumbens*)

Research conducted locally has shown that *Brachiaria decumbens* or signal grass is well adapted to the local conditions and give impressive yields of both green and dry matter. (Table 4). Thus, this grass is planted in almost all livestock farms in Malaysia. In fact, in recent years increasing interest in developing *Brachiaria decumbens* pasture has been shown by various countries in Asia, Africa, South and Central America. High productivity, tolerance towards low fertility conditions, drought resistance and relative freedom from pest and diseases account for the current interest. Apparently, *Brachiaria decumbens* could provide all the forage requirement of ruminant in the tropics and this has helped to promote the growth of this livestock sector. However, this interest has been short-lived because numerous reports have shown that *B. decumbens* is hepatotoxic to sheep and goats (Abas Mazni *et al.*, 1983, Salam Abdullah *et al.*, 1987).

**Table 4.** Average dry matter yield of various fodder cut at six weeks

Fodder	Average dry matter yield (ton/hectare/year)
Signal grass	25.5
Para grass	12.1
Guinea grass	18.6
Napier grass	23.2
Splendida grass	17.6
Kazungula grass	18.2

(From A-Z on Pasture and Fodder. Department of Veterinary Services Malaysia, 1991).

### Toxic effects of *B. decumbens*

Toxic signs developed in sheep as early as 2 weeks after grazing on *B. decumbens* pasture. The affected sheep exhibited signs of jaundice, emaciation, photosensitisation and edema of the ears, submandibular area and eyelids (Figure 6) (Salam Abdullah, 1990).



**Figure 6.** Affected sheep showing oedema of the ears, submandibular areas and eyelids

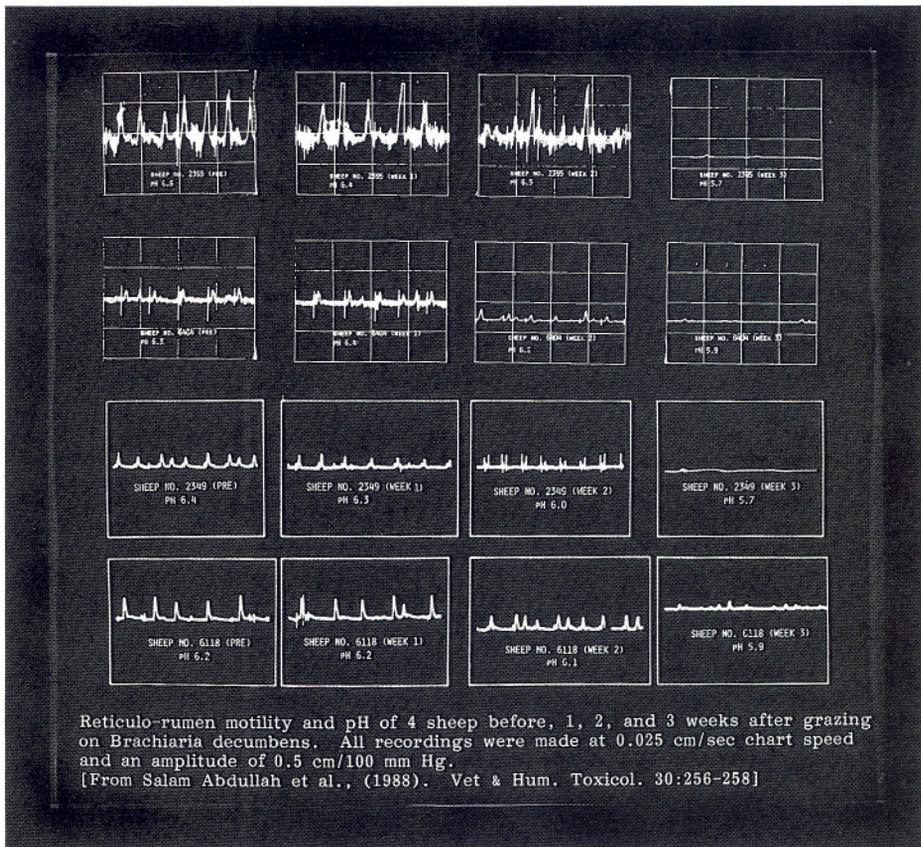
Photosensitisation occurred in all intoxicated sheep as erythema and oedema of the affected part, especially the lightly coloured or unpigmented parts. (Figure 7). In severe cases there was exudation and necrosis of the affected parts and the animals usually died within 4 weeks after grazing on this grass. Necropsy revealed varying degrees of jaundice of the subcutaneous, omental and mesentric fat as well as serous and mucous membranes. The livers were enlarged and firm, mottled and interic with distended gall bladders. Kidney

were also enlarged and congested. In addition, affected sheep also showed neurological disorders such as stamping of forelegs, stargazing, incoordination, head pressing against the wall and circling movements. The neurological dysfunction correspond with pathological changes observed in the brain, particularly in the white matter (Salam Abdullah *et al.*, 1989).



**Figure 7.** Photosensitisation of unpigmented areas in affected sheep

*Brachiaria decumbens* toxicity also affect reticulo-rumen motility and microbial activity of sheep. Ruminal stasis occurred within 3 weeks of grazing on this grass (Figure 8) and the toxicity also produced changes in the rumen microbial population (Figure 9) and the levels of volatile fatty acids of the affected sheep. (Salam Abdullah *et al.*, 1990).



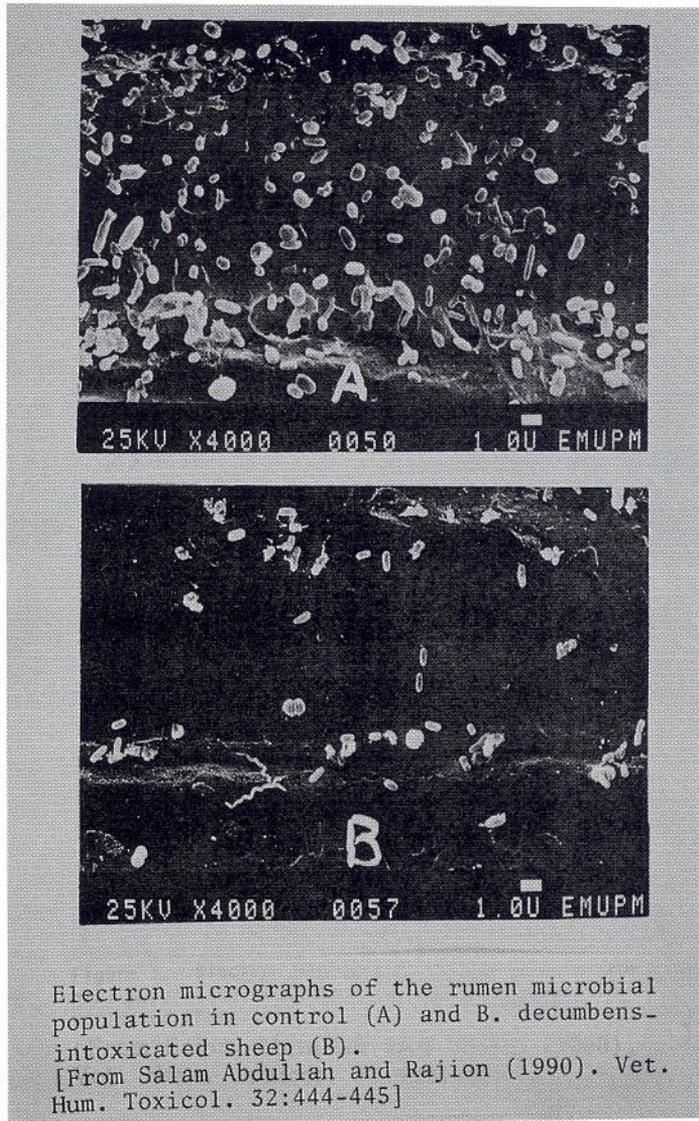
**Figure 8.** Reticulo-rumen motility and pH of 4 sheep before, 1, 2, and 3 weeks after grazing on *Brachiaria decumbens*. All recordings were made at 0.025 cm/sec chart speed and an amplitude of 0.5 cm/100 mm Hg.

## Clinical biochemistry

Experimental *B. decumbens* toxicity in sheep showed that the plasma icteric index of affected sheep increased almost 40 folds during the 8-week period of the study. This increase coincided with the appearance of jaundice.

Other significant changes in the serum biochemistry of the affected sheep include increased in the total bilirubin levels, blood urea nitrogen (BUN) creatinine, activity of the enzymes such as aspartate aminotransferase (AST), glutamate dehydrogenase (GLDH) and gamma glutamyl transferase (GGT).

All intoxicated sheep also showed decreased in bromosulphalein (BSP) clearance. The BSP retention which increased progressively each week (approximately 6 percent during the pre-grazing period to approximately 50 percent at the end of the experiment) was an indicative of immediate liver damage occurring in the affected sheep.



**Figure 9.** Electron micrographs showing the rumen microbial population in sheep, non-feeding (A) and feeding on *B. decumbens* (B)

## Genetic resistance

One important observation was that not all sheep developed the toxic signs after grazing this grass. A few of the experimental sheep did not develop toxic signs at all which suggests strongly that there may well be individual or genetical resistance towards *B. decumbens* toxicity (Salam Abdullah *et al.*, 1990).

## Toxic compounds

It has been reported that the ethanolic extract of rumen liquor from *B. decumbens*, intoxicated sheep contains a hepatotoxic substance or substances causing marked enlargement of the liver and severe necrosis of hepatocytes of rats (Salam Abdullah, 1987). The infusion of rumen liquor from *B. decumbens* intoxicated sheep into the rumen of cattle caused hepatic and renal dysfunction, whereas the grass itself when fed directly to cattle did not produce toxic symptoms (Noordin *et al.*, 1989). These observations suggested strongly that the grass is not toxic *per se* but certain compounds which as a result of ruminal activities of the sheep were converted to their derivatives responsible for causing the toxicity.

Spectroscopic examinations of purified extracts of the above rumen liquors revealed the presence of a mixture of sapogenins, 3-spirostanols (Salam Abdullah *et al.*, 1992) and later identified as epi-sarsasapogenin and epi-smilagenin (Figure 10) (Nordin Lajis *et al.*, 1993). The same compounds were also isolated from the bile of lambs with alveld (a hepatogenous photosensitisation disease which develops after grazing *Nartheccium ossifragum*) by a group of scientists in New Zealand (Miles *et al.*, 1993).

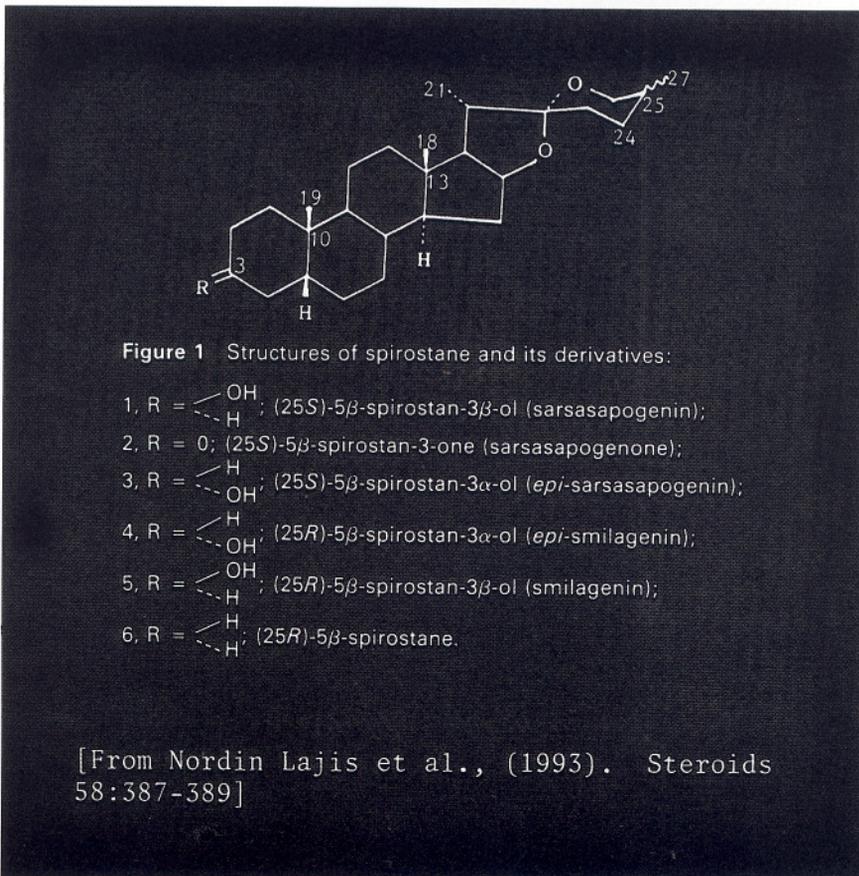
A list of plants causing hepatogenous photosensitisation in which steroidal sapogenins were implicated is shown in Table 5.

## Mechanism of toxicity

The mechanism of toxicity of *Brachiaria decumbens* is still poorly understood. However, histopathological examinations of the livers and kidneys of affected sheep suggest that *B. decumbens* toxin(s) directly damage the cells of the liver and kidney.

## Treatment of *B. decumbens* poisoning

To date there is no effective antidote for *B. decumbens* poisoning. Therapeutic trials using zinc sulphate in drinking water (1 g/litre) provided a significant degree of protection to the sheep from the toxic effects of *B. decumbens* (Salam Abdullah *et al.*, 1994). The mechanism of protection by zinc from the toxic effect of *B. decumbens* is not known. However zinc was known to bind saponins in the gastrointestinal tract (Price *et al.*, 1987) and perhaps zinc also binds saponins and renders them less toxic in the rumen of *B. decumbens* intoxicated sheep.



**Figure 10.** Structures of spirostane and its derivatives

**Table 5.** Steroidal sapogenins implicated in hepatogenous photosensitisation

Plant	Foliage	Bile crystals	Rumen fluid	Reference
<i>B. decumbens</i>	-		3-spirostanols (epi-sarasapogenin, epi-smilagenin)	Salam Abdullah <i>et al.</i> , 1992 Nordin Lajis, <i>et al.</i> , 1993
<i>P. coloratum</i>	diosgenin + yamogenin			Patamalai <i>et al.</i> , 1990
<i>P. schinzii</i>	diosgenin	epismilagenin		Miles <i>et al.</i> , 1991, 1992
<i>P. dichotomi- florum</i>	diosgenin	epismilagenin		Miles <i>et al.</i> , 1991, 1992 Holland <i>et al.</i> , 1991
<i>T. terrestris</i>	diogenin + tigogenin			Miles <i>et al.</i> , 1993
<i>N. ossifragum</i>		episarsapogenin + smilagenin		Miles <i>et al.</i> , 1993

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## SENARAI SYARAHAN INAUGURAL

1. Prof. Dr. Sulaiman Haji Mohd. Yassin  
*'The Challenge To Communication Research in Extension'* 22 Julai 1989
2. Prof. Ir. Abang Abdullah Abang Ali  
*'Indigenous Materials and Technology For Low Cost Housing'* 30 Ogos 1990
3. Prof. Dr. Abd. Rahman Razak  
*'Plant Parasitic Nematodes, Lesser Known Pests of Agricultural Crops'* 30 Januari 1993
4. Prof. Dr. Mohamed Sulaiman  
*'Numerical Solution of Ordinary Differential Equations : A Historical Perspective'* 11 Disember 1993
5. Prof. Dr. Mohd. Ariff Hussein  
*'Changing Roles of Agricultural Economics'* 5 Mac 1994
6. Prof. Mohd. Ismail Sayyed Ahmad  
*'Marketing Management : Prospects and Challenges for Agriculture'* 6 April 1994
7. Prof. Dr. Mohd. Mahyuddin Mohd. Dahan  
*'The Changing Demand for Livestock Products'* 20 April 1994
8. Prof. Dr. Ruth Kiew  
*'Plant Taxonomy, Biodiversity and Conservation'* 11 Mac 1994
9. Prof. Ir. Dr. Mohd. Zohadie Bardaie  
*'Engineering Technological Developments Propelling Agriculture Into The 21st Century'* 28 Mei 1994
10. Prof. Dr. Shamshuddin Jusop  
*'Rock, Mineral and Soil'* 18 Jun 1994