and although normal RPF was seen by Mogensen[5,6], no significant increase in the RPF was reported in diabetes. Most workers suggest either reduced RPF or an increased kidney size together with other metabolic and endocrine changes to be behind this high GFR. However, from these findings it can be said that at least in experimental diabetes it may be that both increased kidney size and reduced RPF and hence increased filtration pressure due to constriction of the vas efferens are responsible for the high GFR. Other metabolic and endocrine changes are not excluded.

These results also show that the kidney functions well during this period of diabetes, although the reduced blood flow to the kidney may indicate the growing tendency of atherosclerosis in renal vessels. Alloxan and streptozotocin in diabetogenic doses seem to have no direct effect on kidney function and the use of very high doses may be responsible for the lesions seen by some other workers. Work now in progress to investigate the response of the diabetic kidney to diuresis seems also to confirm these findings.

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Haemagglutinating activity of modeccin[1]

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Summary. Modeccin, the toxin of *Adenia digitata,* agglutinates erythrocytes from several mammalian species. The haemagglutinating activity is enhanced by neuraminidase and is inhibited by galactose and by galactose-containing sugars.

Modeccin is a highly toxic lectin purified recently from the roots of *Adenia digitata*[2-5], similar but not identical to ricin and abrin (for review on these see Olsnes and Pihl[6]).

Ricin and abrin are distinct from the agglutinins contained in the same plants, but still agglutinate red blood cells. It was observed in our laboratory that modeccin agglutinates rabbit erythrocytes[5], whereas Refsnes et al.[2], working with higher concentrations of the toxin, could observe only indirect agglutination of human erythrocytes. The present experiments were undertaken to elucidate further the haemagglutinating properties of modeccin and to compare them with the properties of ricin.

Materials and methods. Modeccin was prepared as described previously[5], and ricin as described by Nicolson et al.[7,8]. Blood was collected on 0.4% Na-citrate in 0.90% NaCl; erythrocytes were washed 4–5 times by successive resuspensions and centrifugations in 20 mM Na-phosphate buffer, pH 7.1, in 0.14 M NaCl containing bovine serum albumin (15 µg/ml) and were finally resuspended in the same solution at a concentration of 1.2%. Haemagglutinating activity was assayed in Greiner microtiter plates: each well contained 50 µl of erythrocyte suspension and 50 µl of resuspending buffer containing, when appropriate, 2.5 units of neuraminidase (from *Vibrio comma,* Boehringwerke)

Table 1. Haemagglutinating activity* of modeccin and ricin

Erythrocytes	Modeccin		Ricin	
	without neuraminidase	with neuraminidase**	without neuraminidase	with neuraminidase**
Human A	125	7.8	7.8	1.95
B	250	15.6	7.8	1.95
AB	250	15.6	3.9	1.95
0	250	15.6	15.6	1.95
Rabbit	31.2	31.2	1.95	1.95
Pig	62.5	31.2	3.9	3.9
Horse	125	7.8	7.8	0.98
Guinea-pig	125	15.6	15.6	0.98
Mouse	250	62.5	15.6	1.95
Rat	500	125	31.2	1.95
Sheep	No agglutination***	31.2	62.5	7.8
Ox	No agglutination***	31.2	250	31.2

*Expressed as the lowest concentration (in µg/ml) of the lectin giving visible agglutination. **2.5 units/ml. ***With modeccin up to 500 µg/ml.

Table 2. Inhibitory effect of sugars on the haemagglutinating activity of modeccin*

Modeccin (μg/ml)	62.5	31.2
Sugars	Sugars concentration (mM) inhibiting agglutination	
D-galactose	12.5	3.1
D-fucose	50	12.5
Lactose**	not tested	12.5
Melibiose	25	3.1
Raffinose	50	12.5

*Assayed on rabbit erythrocytes. **Lactose at 25 mM and higher concentrations causes by itself some haemagglutination.

and serial dilutions (1:1, starting from 500 μg/ml of final volume) of the lectins. Sugars were tested for inhibitory effect at serial dilutions (1:1, starting from 50 mM final) with 100 μl of erythrocyte suspension in a final volume of 200 μl.

Results and discussion. The haemagglutinating activities of modeccin and ricin are reported in table 1. Modeccin agglutinates erythrocytes from several mammalian species with no specificity for human blood groups. Maximal agglutination was observed with rabbit erythrocytes, whereas sheep and ox erythrocytes were not agglutinated by modeccin at the highest concentration tested. In all cases except rabbit erythrocytes, agglutination was enhanced by neuraminidase, to an extent variable from species to species. In all cases the agglutinating activity of modeccin was lower than that of ricin, which also was enhanced by neuraminidase, except in the case of rabbit and pig erythrocytes.

Several sugars were tested for inhibitory effect on haemagglutination of rabbit erythrocytes by modeccin. The following sugars had no effect at concentrations up to 50 mM: D-ribose, D-xylose, L-arabinose, D-fructose, D-glucose, 1-O-methyl-α-D-glucopyranoside, 1-O-methyl-β-D-glucopyranoside, D-mannose, D-glucosamine, N-acetyl-D-glucosamine, D-galactosamine, N-acetyl-D-galactosamine, maltose, sucrose, cellobiose, trehalose, melezitose. Only fucose, galactose and galactose-containing di- and trisaccharides were inhibitory (table 2).

Our results demonstrate that modeccin has an haemagglutinating activity, which is different on erythrocytes from different species. Previous apparent inconsistencies[2,5] are thus accounted for by the fact that Refsnes et al.[2] used human erythrocytes, whereas we used rabbit erythrocytes[5], which are more sensitive to the agglutinating lectin. The inhibitory effect of galactose and of galactose containing sugars indicates that modeccin, like ricin, binds to galactosyl residues on the red cell membrane, which is consistent with previous findings with Ehrlich[5] and HeLa[2] cells. The lower agglutinating activity of modeccin as compared with ricin may indicate that, on the membrane of erythrocytes, there are fewer receptors for modeccin than for ricin, as is the case in HeLa cells[9]. The enhancement of agglutination by neuraminidase is consistent with results obtained with modeccin on human erythrocytes and HeLa cells[2], and with ricin on Novikoff tumour cells[10], and may be due to unmasking of galactosyl receptors on the cell membrane[9].

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The effect of dietary fat on the anticoagulant activity of aflatoxin B_1

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Summary. A single i.p. dose of aflatoxin B_1 had no significant effect on the thrombotest clotting times of monkeys subsisting on low-fat and high-fat dietary regimens, respectively. There was a significant interaction between aflatoxin and dietary fat level.

Haemorrhage has been a common clinical and pathological sympton associated with aflatoxicosis in many animal species[2-6]. This bleeding often precedes death[5,7,8]. Related to these findings are reports that aflatoxin lengthened blood clotting time in the dog, rat, chicken, guinea-pig and monkey[5,9-12]. In a review by Schoental[13], emphasis was placed on the need for studies on the anticoagulant property (amongst other pharmacological properties) of aflatoxin. It has been proposed that the aflotoxins as coumarin compounds, having a central 5-methoxy moiety, act as anticoagulants[9,14]. However, the aflatoxins have been reported to be much more effective than coumarin in prolonging the time necessary for blood clotting to occur in the rat[9,15-16]. In this paper, we report our observations on the influence of dietary fat on the anticoagulant activity of aflatoxin B_1 in the Nigerian monkey (*Cercopithecus aethiops*, Tantalus).

Materials and methods. The experimental animals were obtained from the stock quartered at the Primate Colony of the Department of Biochemistry of Ibadan University. They had weights between 2.8–3.5 kg. Their ages were not known because none of them was born in captivity. All monkeys were in apparent good health and were individually housed in cages which were supplied with food trays. Tap water was available ad libitum. Monkeys were alloted into 6 treatment groups of 6 animals in each. Groups I, II and III were maintained on a stock, low-fat (2.2%) diet, while groups IV, V and VI received a high-fat (18.1%) diet.