Table 2 *Effect of NAA and 2,4-D on rooting in micropropagated shoots of Rosa hybrida cv. Landora cultured in liquid MS medium*

Treatment (mg l)	No. of shoots cultured	Shoots rooting (%)	Days to rooting	No. of roots per shoot (mean ± SE)
—	152	—	—	—
NAA (0.25)	176	63.6	14	3.66 ± 0.57
NAA (0.5)	160	80.0	12	3.88 ± 0.73
NAA (1.0)	154	81.8	12	2.58 ± 0.59*
2,4-D (0.25)	168	90.0	12	3.20 ± 0.26
2,4-D (0.5)	154	80.0	12	3.50 ± 0.56
2,4-D (1.0)	160	50.0	14	2.00 ± 0.62*
NAA (0.25) + 2,4-D (0.1)	154	90.9	8	6.00 ± 0.81
NAA (0.5) + 2,4-D (0.1)	140	80.0	10	5.10 ± 0.83*
NAA (1.0) + 2,4-D (0.1)	176	54 5	12	2.66 ± 0.69*

*Callusing at the base of shoots.

mortality. Similar results have been reported from micropropagation experiments in some difficult-to-propagate rose cultivars[3,5–8]. Roses being very popular as garden plants throughout the world, any such practice that can accelerate the propagation rate should be of high commercial value to the rose industry. It seems conceivable that in future many important cultivars of roses would be commercially propagated through tissue culture[3].

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DIABETOGENIC NATURE OF OCHRATOXIN A

S. SUBRAMANIAN, N. BALASUBRAMANIAN and S. GOVINDASAMY
Department of Biochemistry, University of Madras, Madras 600 025, India

OCHRATOXIN A, a hepatotoxic mycotoxin, is produced as a secondary metabolite by *Aspergillus ochraceus* and *Penicillium virridicatum*[1]. Ochratoxin A ingestion causes increase in blood glucose concentration[2]. *A. ochraceus* has been reported to grow on a wide variety of foodstuffs and feeds, including rice, wheat, corn and peanuts[3]. Ochratoxins and ochratoxin-producing fungi have been isolated and purified from food samples meant for human consumption in various parts of the world. Previous reports reveal that *A. ochraceus* contamination is very frequent in India, especially in the southern region, which is evident from the reported isolation and identification of ochratoxins in some warehouse samples in Tamil Nadu and Hyderabad[4,5]. Ingestion of mold-contaminated diet poses health hazards to human beings. This communication reports results of a study on the effect of ochratoxin A on liver carbohydrate, blood glucose and serum insulin levels.

The strain of *A. ochraceus* used was isolated in our laboratory from fungus-contaminated feed. The identification was confirmed by the Indian Agricultural Research Institute, New Delhi, India. The strain was found to produce ochratoxin A as a major secondary metabolite. Ochratoxin A was isolated from the culture filtrate by the method of Suzuki *et al.*[6] An authentic sample of ochratoxin A

supplied by Dr Timothy D. Phillips, Department of Veterinary Public Health, Texas A & M University, USA, was used as reference.

Weanling albino rats from our laboratory animal colony weighing 80 ± 15 g were used. The rats were divided into two groups, each comprising 15 rats. One group was administered orally with 0.5 ml of 0.1 M sodium bicarbonate solution[7] containing 100 μg of ochratoxin A per rat per day and the other group was maintained as control.

After eight weeks blood was collected from the tail of the rats and blood glucose concentration was estimated[8]. Since the fasting blood glucose concentration of the experimental group of rats was elevated, tolerance to glucose was tested. The rats were starved overnight and fasting blood samples were collected. Glucose solution (10%) was administered orally (3.5 g per kg body weight). Blood samples were collected thereafter over a period of 120 min every 30 min. One set of blood samples was collected with sodium fluoride as anticoagulant for glucose estimation and another set was collected for the assay of serum insulin by the method of Herbert *et al.*[9] Later the animals were killed and liver tissue was used for the estimation of total carbohydrates and glycogen[10].

The blood glucose and serum insulin levels of control and ochratoxin A-administered rats are given in table 1. The blood glucose and serum insulin levels during the glucose tolerance test are shown in figures 1 and 2 respectively. In control rats maximum blood glucose concentration was reached 60 min after glucose load and the concentration dropped back to fasting level by 120 min. But in the treated rats blood glucose concentration (maximum around 60 min) did not return to fasting level even at the end of 120 min, indicating an imbalance in glucose homeostasis during ochratoxin A toxicosis (figure 1). Though insulin level increased immediately after glucose load, the treated rats showed insufficient increase in insulin level (figure 2) to utilize the ingested glucose, which resulted in hyperglycemia. Total carbohydrates and glycogen were reduced in liver tissue of treated rats (table 2).

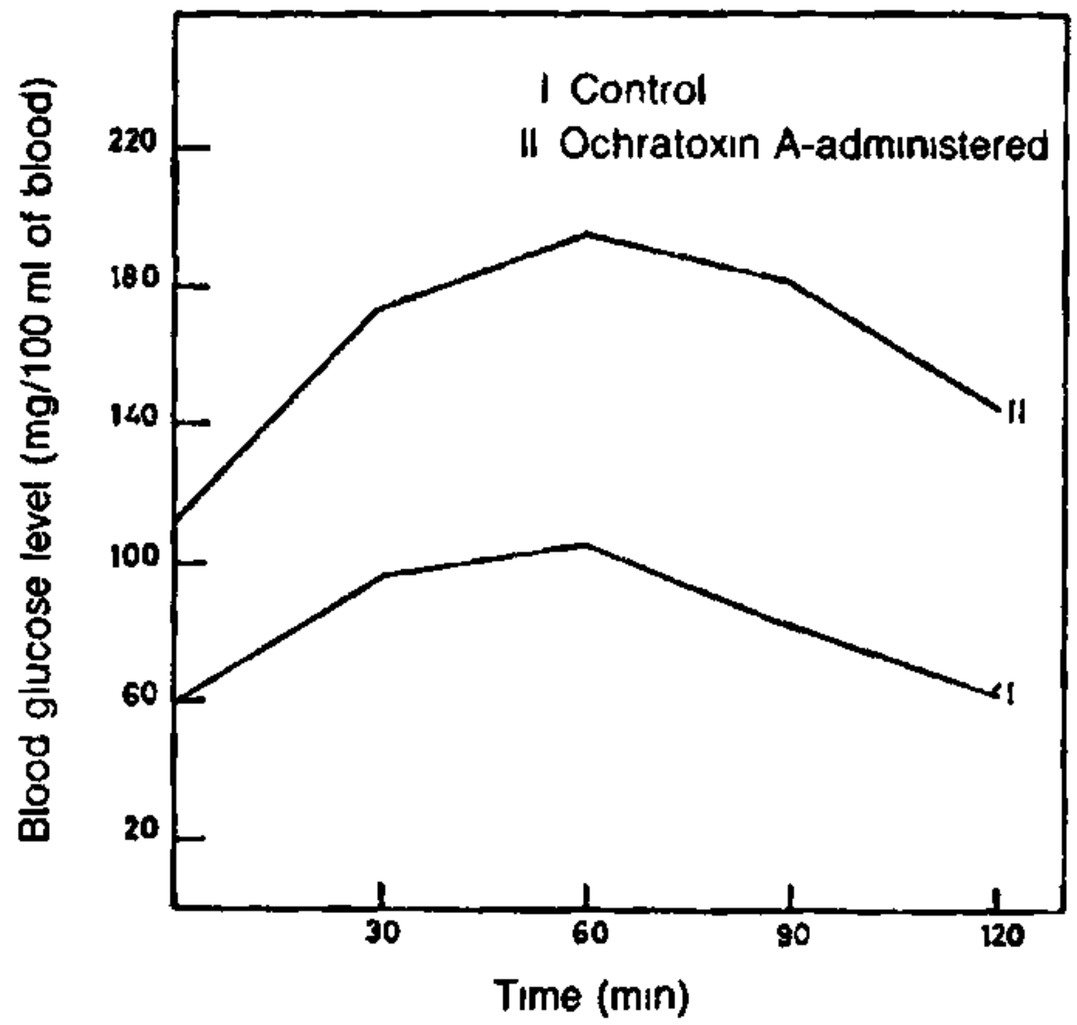

Figure 1. Blood glucose levels in control and ochratoxin A-administered rats during glucose tolerance test.

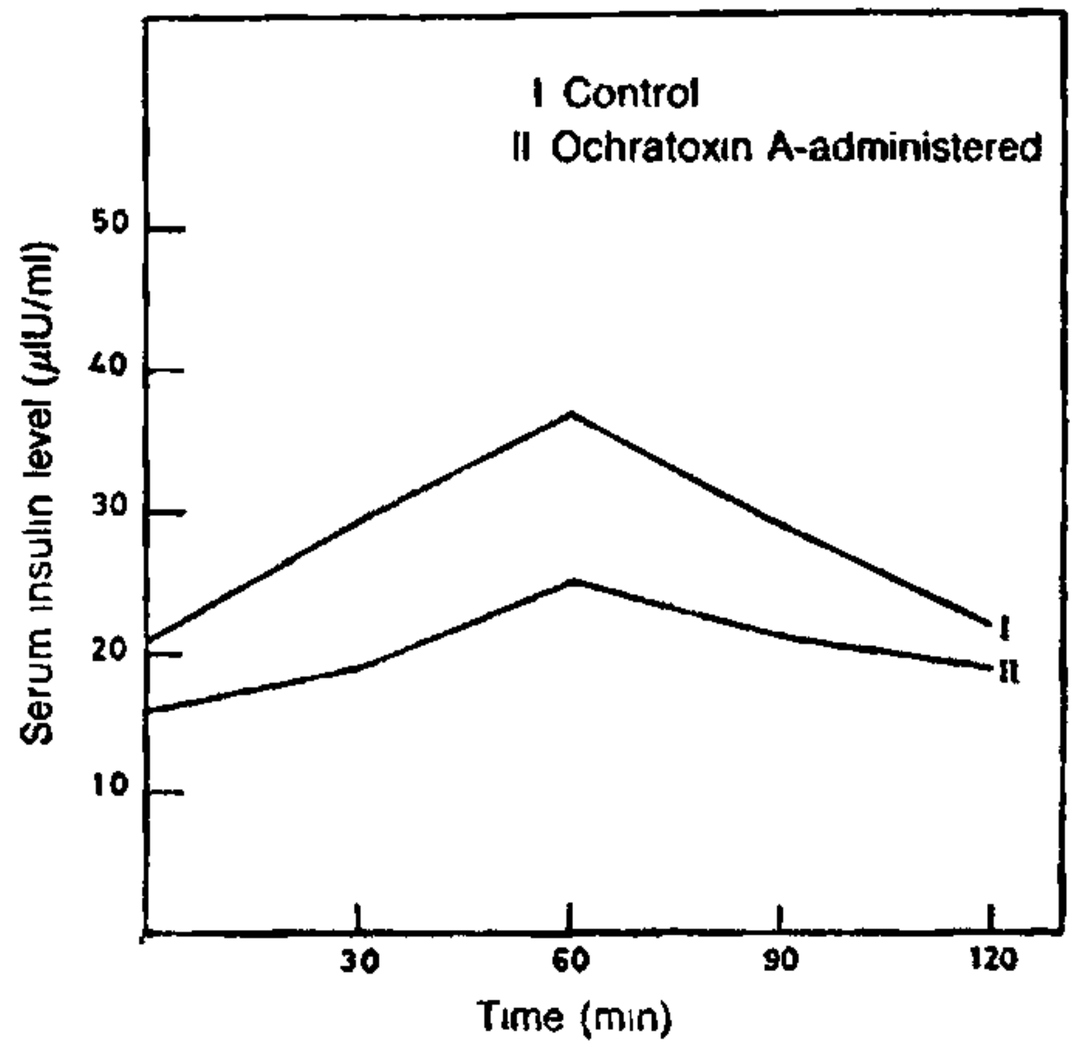

Figure 2. Serum insulin levels in control and ochratoxin A-administered rats during glucose tolerance test.

Table 1 *Blood glucose and serum insulin levels in control and ochratoxin A-administered rats*

Parameter	Control	Ochratoxin A-administered
Blood glucose (mg/100 ml)	59.20 ± 4.80	112.40 ± 5.10*
Serum insulin (μIU/ml)	21.30 ± 1.30	14.50 ± 1.80*

Values are mean of six observations.
*Statistical significance, $P < 0.001$.

Normally blood glucose level is maintained by the regulatory pancreatic hormones insulin and glucagon. Insulin and glucagon influence carbohydrate metabolism in an antagonistic manner. However, utiliza-

Table 2 *Liver total carbohydrates and glycogen in control and ochratoxin A-administered rats*

Parameter	Control	Ochratoxin A-administered
Total carbohydrates	62.40 ± 7.41	39.22 ± 4.76*
Glycogen	42.15 ± 3.15	20.50 ± 3.90*

Values are expressed as mg g of fresh tissue and are average for six animals in each group.
*Statistical significance, $P < 0.001$.

tion of blood glucose is mainly controlled by the action of insulin and glucose is the best inducer of insulin secretion from the islets of pancreas. The increase in blood glucose level during ochratoxin A toxicosis may be due to the decreased level of insulin, which in turn may be due to inhibited synthesis and/or reduced release of insulin from the pancreatic cells. Induction of hyperglycemia associated with hypoinsulinemia by mycotoxins like terreic acid[11] and penitrem A[12] has been reported. Enhanced blood glucose accompanied by depletion of hepatic glycogen in chicks during ochratoxin A toxicosis has also been noticed[13].

The liver functions as a 'glucostat' and plays a vital role in the maintenance of blood glucose level either by uptake from or by release of glucose into the blood. Ochratoxin A has been reported to cause injury to the liver[14]. It is important to note that though there is a reduction in the level of total carbohydrates in liver tissue during ochratoxin A toxicosis, it is only a reflection of glycogen decrease. The observed decrease in the level of glycogen may be due to the effect of ochratoxin A on glycogen metabolism. Other mycotoxins like aflatoxin[15] and patulin[16] have also been reported to lower hepatic glycogen.

Since ochratoxin A-administered rats exhibited a severe diabetic condition, it may be concluded that ochratoxin A, a major secondary metabolite of *A. ochraceus*, is diabetogenic in nature.

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CHROMOSOMAL STUDIES IN *FULVIA MUTICA* (BIVALVIA: CARDIIDAE) FROM JAPAN

USHA GOSWAMI
National Institute of Oceanography, Dona-Paula 403 004, India

CHROMOSOMAL studies on a large number of molluscs belonging to different families were made earlier[1-4]. Nakamura[2] listed cytogenetic information on 125 species of molluscs using the CISMOCH computerized index system. However, in family Cardiidae only five species were recorded. The present report of chromosome number and karyotype