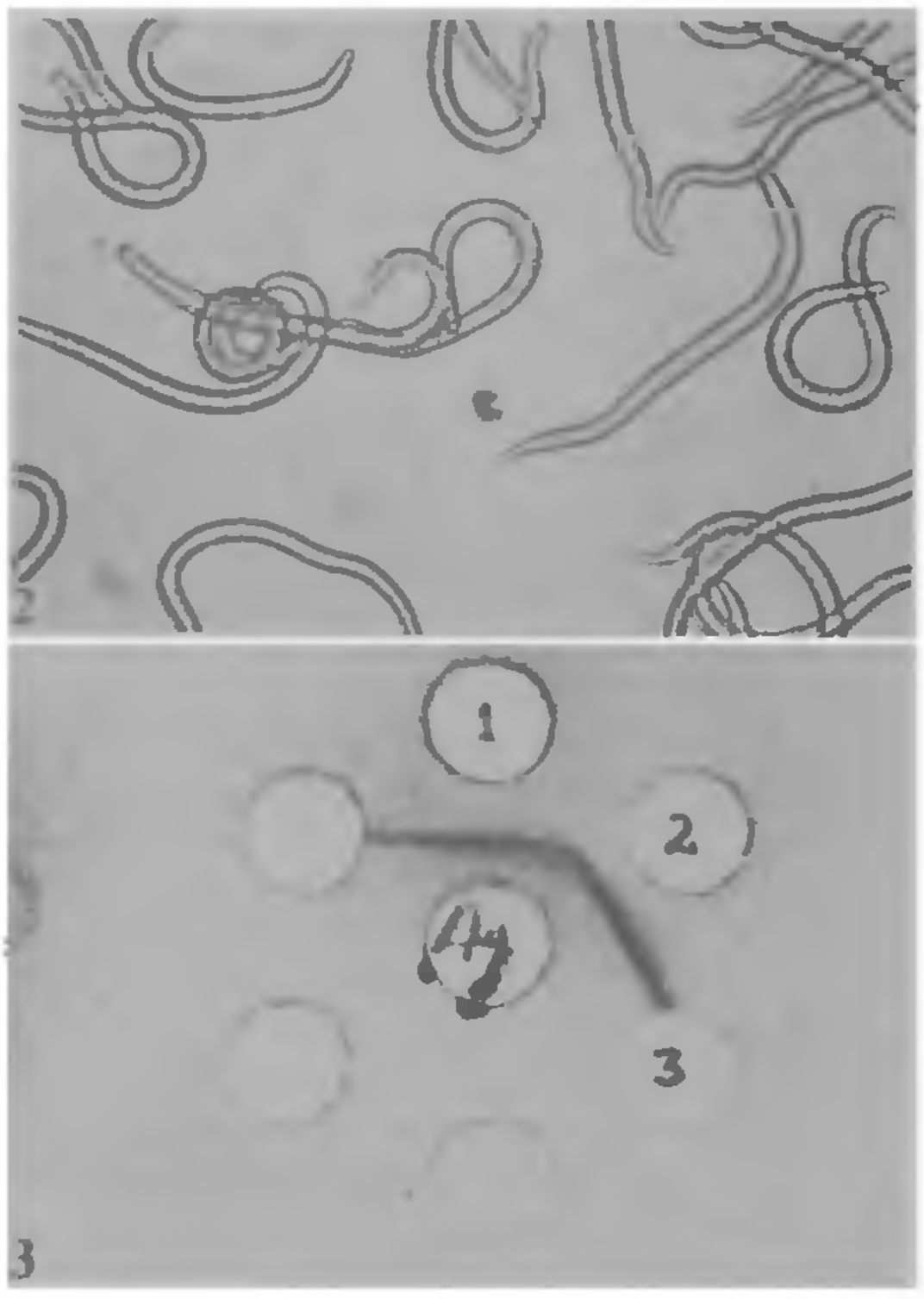

Figures 2 & 3. 2. Hatched and viable *T. canis* larvae. **3.** AGD test showing the precipitin lines centre well (Ag)—ES antigen; 1 and 2 —*T. canis* infected mice sera and 3 —Normal control (uninfected) mouse serum.

The medium was changed every 15 days and the spent pooled medium was stored at −20°C. This spent medium after filtration through millipore filter (0.45 μm), was precipitated with saturated ammonium sulphate solution. The precipitate was removed by centrifugation and was dissolved and dialyzed against normal saline. The final volume was 1/20th of the original fluid. The antigen was checked in agar-gel diffusion (AGD) test[8], which gave precipitin lines against the sera of *T. canis* infected mice (figure 3). The control antigen prepared similarly from the uninfected medium did not show any reaction against *T. canis* infected or normal mouse sera. Twelve Swiss mice were infected orally with *T. canis* larvae (600–700 larvae/mouse) and the sera collected from the 67th to 314th post-infection day, were tested for antibodies in AGD test against the ES antigen. Sera from 7 non-infected (control) mice of the same age and sex were also included. Eleven out of the 12 sera from *T. canis* infected mice reacted with the antigen while all the 7 sera from the control mice were negative.

Employing this method, hatching of *T. canis* larvae was successfully tried seven times in the laboratory. The ES antigen was prepared twice and yielded good reaction against *T. canis* infected mouse sera in the AGD test.

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EFFECT OF MALATHION ON BLOOD GLUCOSE, LIVERGLYCOGEN, PLASMA CORTICOSTERONE AND ELECTROLYTES CONCENTRATIONS AND EOSINOPHIL COUNT IN ADRENALECTOMISED RATS

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ORGANOPHOSPHATE insecticides are known to cause hyperglycaemia and also the increased liver glycogen[1, 2]. Previous studies with malathion in this laboratory have shown that when it was administered

intraperitonially at 170 mg/kg in rats, produced increase in plasma corticosterone along with typical signs of hyperglycaemia, increase in liverglycogen, and eosinopaenia[3]. Ghafghazi and Mennear[4] found that the hyperglycaemic effect due to cadmium acetate is mediated through adrenal glands. In contrast, KaCew and Singhal[5] reported that the hyperglycaemic effect of P, P-DDT is not mediated through adrenal glands and it is due to inhibition of gluconeogenic enzymes. The present study was therefore conducted in adrenalectomised rats administered malathion to elucidate the possible involvement of adrenal glands in mediating these effects.

The experiment was conducted on Wister strain of male albino rats weighing 120 to 150 g. The animals procured from the animal house of this Institute were maintained on standard feeding schedule. The feed and water were provided *ad lib.*

Malathion [(0,0-Dimethyl S (1,2-dicarbo ethoxyethyl) phosphorodithioate] technical grade (97.2%) after dissolving in arachis oil was administered intraperitoneally. The animals were divided into four groups: (1) sham-operated control (2) sham-operated administered malathion (3) adrenalectomised control and (4) adrenalectomised administered malathion. Each group consisted of six animals. The rats were adrenalectomised bilaterally according to the method outlined by Zarrow *et al*[6]. In the case of sham-operated animals, the entire operation of adrenalectomy was performed except removal of adrenal glands. The operated animals were maintained for five days by providing 1% sodium chloride solution and 5% glucose in the case of adrenalectomised animals and only glucose solution in the case of sham-operated animals. After five days, these animals were used for experimentation. Malathion was administered at a dose of 170 mg/kg which is approximately 1/7th of LD-50 (1150 mg/kg). Controls received equivalent volume of arachis oil without malathion. All the animals were sacrificed 2 hr after malathion administration[3]. The rats were anaesthetised with pentobarbitone at 50 mg/kg given intraperitoneally before sacrifice. The chest was opened and the blood was collected directly from the heart in heparinised test tubes. A part of it was utilized to estimate blood glucose[7] and eosinophilcounting[8] and the remainder was centrifuged and the plasma obtained was used to estimate corticosterone[9] and sodium and potassium levels. After opening the peritoneal cavity, liver was taken out for estimating glycogen[10]. The experimental data were analysed statistically[11] and student's *t* test was applied to determine the significance.

Malathion produced a significant hyperglycaemic effect in sham-operated animals (141.8 mg/100 ml) whereas it failed to show this effect in adrenalectomised rats (66.4 mg/100 ml). This indicates the involvement of adrenal glands in hyperglycaemic effect caused by malathion. Malathion treatment caused a significant increase of liverglycogen in sham-operated animals (50.3 mg/g) but in adrenalectomised animals, the glycogen level decreased (17.5 mg/g). Malathion administration caused a significant increase in the level

Table 1 *Effects of malathion treatment on blood and liver components*

	Adrenalectomy	Control	Malathion treated	% control
Blood glucose (mg/100 ml)	–	76.1 ± 3.4	141.8 ± 5.6**	186
	+	70.5 ± 3.5	66.4 ± 2.8	94
Liver glycogen (mg/g)	–	26.6 ± 1.2	50.3 ± 1.2**	189
	+	25.3 ± 2.2	17.5 + 2.6**	69
Plasma corticosterone (μg/100 ml)	–	32.2 ± 2.9	63.7 ± 3.5**	210
	+	7.2 ± 0.5	7.6 ± 0.5	106
Eosinophil count (counts/cmm)	–	876 ± 14	136 ± 6**	15
	+	1206 ± 113	275 ± 28**	23
Plasma sodium (mEq/l)	–	170.0 ± 3.6	157.0 ± 8.5	92
	+	177.0 ± 4.5	180.0 ± 4.8	102
Plasma Potassium (mEq/l)	–	5.4 ± 0.1	5.2 ± 0.1	96
	+	6.5 ± 0.5**	6.1 ± 0.2	94

Animals were sacrificed 2 hr after the administration of malathion.
** $P < 0.01$ as compared to their respective controls in all attributes except plasma potassium which is compared with sham-operated.

of plasma corticosterone in sham-operated animals, whereas it produced no change in corticosterone level in adrenalectomised rats since the animals had no in tact adrenal cortex. Malathion produced a significant eosinopaenic response in both sham-operated and adrenalectomised rats. The eosinophil counts reduced from 876 to 135 counts/cmm in sham-operated animals and from 1206 to 275 counts/cmm in adrenalectomised animals, indicating that malathion has eosinopaenic effect through some direct nonspecific toxic action. Malathion did not produce any significant change in the levels of plasma sodium and potassium in both sham-operated and adrenalectomised rats. The level of plasma potassium was significantly higher in adrenalectomised rats (6.5 mEq/l) compared to that of sham-operated (5.4 mEq/l), an effect similar to adrenalectomy[12].

The involvement of adrenal glands in mediating the hyperglycaemic effect was observed with other insecticides and herbicides like guthion diquat and paraquat[1, 13]. In contrast hyperglycaemia induced by P, P-DDT was not mediated through adrenal glands[5]. Guthion failed to produce increase of liverglycogen in adrenalectomised animals[1]. Speirs and Mayer[14], reported a decrease of eosinophils after administration of benzyl alcohol which was due to some direct action but not through adrenal glands.

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A NEW DISEASE OF WHITE BUTTON MUSHROOM (*AGARICUS BISPORUS*)

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DURING the cultivation of *Agaricus bisporus* from October to January some note-worthy diseases were earlier recorded[1–3] but a disease caused by *Gliocladium virens* Miller, Giddens and Foster is being reported for the first time from India[4] or from any other country.

The disease is characterised (figure 1) by the formation of brown necrotic lesions from the margin of the pileus which migrated deep into the juncture of pileus and stipe, causing browning and necrosis in the stipe due to which there was splitting of the fungal hyphae of the stipe. The diseased fruit bodies did not produce any characteristic odour. The normal course of the development of sporophores was checked and the mushroom appeared ugly. Pathogenicity of *G. virens* was also tested by inoculating the fruit bodies of *A. bisporus* (figure 2). Preliminary studies show that the disease can be controlled by spraying benlate (methyl

Figures 1 & 2. 1. Diseased fruit body of *Agaricus bisporus*. 2. Symptoms of disease developed three days after artificial inoculation.