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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15 April 1983

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**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 mouse 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, 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

during the study period for 90 days with a daily
administration of 1 mg/kg body weight. The rats were
kept on a regular diet and provided with water ad
libitum. The animals were divided into four groups
containing 6 rats each. Group 1 received saline
solution only. Groups 2, 3 and 4 received 1, 3 and 6
mg/kg body weight of malathion, respectively.

**EFFECT OF MALATHION ON BLOOD GLUCOSE, LIVER GLYCOGEN, PLASMA CORTICOSTEROIDE AND ELECTROLYTE CONCENTRATIONS AND EOSINOPHIL COUNT IN ADRENALECTOMISED RATS**

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Organophosphate insecticides are known to cause hyperglycaemia and also the increased liver glycogen1,2. Previous studies with malathion in this laboratory have shown that when it was administered
intraperitoneally at 170 mg/kg in rats, produced increase in plasma corticosterone along with typical signs of hyperglycaemia, increase in liverglycogen, and eosinopenia. Ghafghi and Mennear found that the hyperglycaemic effect due to cadmium acetate is mediated through adrenal glands. In contrast, KaCew and Singhal 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. 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. 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 and eosinophil counting and the remainder was centrifuged and the plasma obtained was used to estimate corticosterone and sodium and potassium levels. After opening the peritoneal cavity, liver was taken out for estimating glycogen. The experimental data were analysed statistically 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>
<thead>
<tr>
<th>Table 1</th>
<th>Effects of malathion treatment on blood and liver components</th>
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<tr>
<td></td>
<td>Adrenalectomy</td>
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<tr>
<td>Blood glucose (mg/100 ml)</td>
<td>-</td>
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<tr>
<td>Liver glycogen (mg/g)</td>
<td>-</td>
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<td>Plasma corticosterone (µg/100 ml)</td>
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<tr>
<td>Eosinophil count (counts/mm³)</td>
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<tr>
<td>Plasma sodium (mEq/l)</td>
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<tr>
<td>Plasma Potassium (mEq/l)</td>
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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 intact adrenal cortex. Malathion produced a significant eosinopenic response in both sham-operated and adrenalectomised rats. The eosinophil counts reduced from 876 to 135 counts/cm³ in sham-operated animals and from 1206 to 275 counts/cm³ in adrenalectomised animals, indicating that malathion has eosinopenic effect through some direct non-specific 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 liver glycogen in adrenalectomised animals.1. Speirs and Mayer 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 recorded1–3 but a disease caused by Gliocladium virens Miller, Giddens and Foster is being reported for the first time from India4 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.