for a period of one year. After this period the activity was reassessed by the same method. In all, three experiments were carried out, and the data based on these studies are presented in Table I.

TABLE I

Effect of seed treatment with antibiotics against H. oryzae *and seed germination*

Treatment	Efficacy soon after seed treatment	Per cent seed germination	Efficacy after one year seed storage	Per cent seed germination
Piomy	+	90	..	88
Aureofungin	+	91	..	85
Check		85		76

+ denotes effective — denotes ineffective

It is evident from Table I, that piomy and aureofungin are rendered biologically inactive on the seed after one year, suggesting their degradation in storage and consequent loss of fungicidal properties.

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TOPOGRAPHICAL RELATIONSHIP BETWEEN THE BLASTOCYST AND THE UTERUS IN THE RHINOLOPHID BAT, *RHINOLOPHUS ROUXI* (TEMMINCK)

THE mammalian blastocyst bears a specific topographical relationship with the morphology of the uterus at the time of implantation, and this is usually constant within the family and sometimes within the order (Mossman[1,2,3]). Among the bats, the orientation of the embryonic mass of the implanting blastocyst is mesometrial in Megachiroptera (Keibel[4], Moghe[5], Mossman[1]), and antimesometrial in the Microchiroptera (Duval[6], Ramaswami[7], Wimsatt[8], Gopalakrishna[9]). Most of the earlier work on the embryology of Microchiroptera relates to members of the family Vespertilionidae. During the past 20 years the embryology of members of several families of Microchiroptera has revealed that the orientation of the embryonic mass differs not only in different families of Microchiroptera (Gopalakrishna[10,11], Gopalakrishna and Moghe[12]) but also in different individuals of the same species, in a few cases (Gopalakrishna and Khaparde[13], Gopalakrishna and Karim[14]). Where the orientation of the embryonic mass has been shown to be variable in the same species, the blastocysts described were at a very early stage of development, and the embryonic mass in all the cases was still spherical. Gopalakrishna and Khaparde[13], and Gopalakrishna and Karim[14] suggested that the embryonic knob in these bats may rotate within the blastocyst, as in the case of the mouse (Kirby, Potts and Wilson[15]).

While making a detailed study of the early development of the Rufous Horse-shoe Bat, *Rhinolophus rouxi*, the author examined eight late implanted bilaminar blastocysts, all nearly at the same stage of development. In every case the blastocyst was in a large implantation chamber of the uterus, and the wall of the blastocyst was in intimate contact with the endometrium since the uterine epithelium was lost from all sides of the implantation chamber. The embryonic area had expanded into a flat disc. Among the eight blastocysts, the embryonic disc was oriented towards the antimesometrial side in two, towards the mesometrial side in one, towards the lateral side in two, towards the medial side in two and between the lateral and mesometrial side in one.

Whereas the earlier descriptions of variable orientation of the embryonic mass referred to early blastocysts having spherical embryonic mass[13,14], the present study refers to advanced blastocysts with expanded emoryonic disc. Hence the explanation that the embryonic mass may rotate within the blastocyst covering is not tenable in the present case. The final position of the placental disc in *Rhinolophus rouxi* is however invariably mesometrial (Gopalakrishna

and Bhiwgade[16]). In all bats, except *Tadarida braziliensis cynocephala* (Stephens[17]), the final placental disc is invariably formed on the side towards which the embryonic mass is directed at the stage of implantation. In *Tadarida braziliensis cynocephala*[17] the early orientation of the embryonic mass is antimesometrial but the final placenta is formed on the mesometrial side.

Rhinolophus rouxi is evidently an exception among Chiroptera where, regardless of the variable orientation of the embryonic disc in advanced implanted blastocysts, the final placental disc is invariably located on the mesometrial side. Our knowledge of the embryology of bats does not suggest any explanation of this anomaly except to presume that the allantois grows towards the mesometrial side through the exocoelom formed by the separation of the yolk sac splanchnopleaure from the chorion in all the blastocysts except in those in which the embryonic disc is already oriented towards the mesometrial side. In the latter case, the allantois grows as in other bats (except *Tadarida braziliensis cynocephala*[17]) towards the dorsal side of the embryonic disc since the yoll sac lies on the ventral side of the embryonic disc.

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CHANGES IN TISSUE GLYCOGEN OF A FRESH WATER CATFISH *HETEROPENUSTIS FOSSILIS* (BLOCH) DUE TO MERCURY INTOXICATION

THERE has been much anxiety in many countries due to adverse effects arising from heavy metal intoxication as a result of rapid industrialization. Due to mercury intoxication minamata disease, congenital mental retardation, injury to kidney, suppression of urine and ultimately death from kidney failure, corrosion of gastrointestinal tract, etc., are caused[1]. Though heavy metal intoxication has been studied by numerous investigators,[2–6] the effect of mercury intoxication on glycogen content of fish tissues has not been studied. In the present investigation the effect of various concentrations (5, 25 and 50 ppm) of mercuric nitrate on glycogen content of brain, liver, kidney and muscle of *H. fossilis* has been investigated.

Fifteen live *H. fossils* (18–20 cm) were acclimatized in the laboratory for 3-4 days. A group of 5 fishes was kept in the glass aquaria containing 5, 25 and 50 ppm of mercuric nitrate for 2-3 hours. Their liver, brain, kidney and muscle were homogenised and centrifuged for 20 minutes at 3,000 rpm in 5 ml of 1·5% KOH and processed for glycogen content[7]. Each experiment was repeated 4 times and the data were subjected to statistical analysis. Glycogen content of the above-mentioned tissues of untreated fish was taken as the control.

Mercury intoxication has brought about a number of significant changes in the glycogen content of liver, brain, kidney and muscle of *H. fossils*. It has been noted that 5 ppm of mercury increases the glycogen content of the liver, brain and muscle but the glycogen content decreases in liver and muscle as the concentration of mercuric salt increases to 25 and 50 ppm (Table I). Moreover, the kidney and brain responded differently with the mercury salt. The glycogen content of kidney decreased with the increase in the concentration of mercuric nitrate. However, in the brain, the glycogen contnet increased with 5 and 25 ppm of mercury intoxication and then decreases when the concentration was enhanced to 50 ppm