rufus parasitised by the same species of nematode (*A. aptini*)[5, 6].

The most striking feature in this host-parasite relation was the occurrence of different stages of the nematode in the host of varying ages. The thrips pupae were most infected with parasitic adults and eggs, while in senescent adults (approximately 9th day), nematode larvae outnumbered the other stages. This variation at different stages of parasitic occurrence indicates that the parasite proliferates inside the host for the maintenance of its population, since the infection initially started with adults. It is curious to note that even after careful examination of hundreds of thrips, not even a single male was parasitized by the nematode. In the case of *A. rufus* also only the female thrips became infected by this parasite[7]. The specificity of these parasites infesting the females may be due to the restriction of its infestation to only the ovaries of the thrips host, the degree of ovarian damage depending largely on the density of the parasite in the host system.

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TOXICITY OF MERCURY ON THE OVARIES OF THE CARIDEAN PRAWN, *CARIDINA RAJADHARI* (BOUVIER)

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HIGHEST concentrations of mercury appear to occur in the Crustaceans either when they pass large quantities of water over their respiratory surfaces or bioaccumulated through food-chain[1, 2]. There is considerable evidence to indicate that mercury can cause structural damage to gill epithelia and kidney tubules and neurological disorders[3, 4]. It was reported that continuous accumulation of mercury may reduce the ability of every cell in the body of fish to maintain proper intracellular ionic composition[5]. The present study reports the toxicity of mercury on the ovaries of the freshwater prawn, *Caridina rajadhari* (Bouvier) (Crustacea, Decapoda, Atyidae).

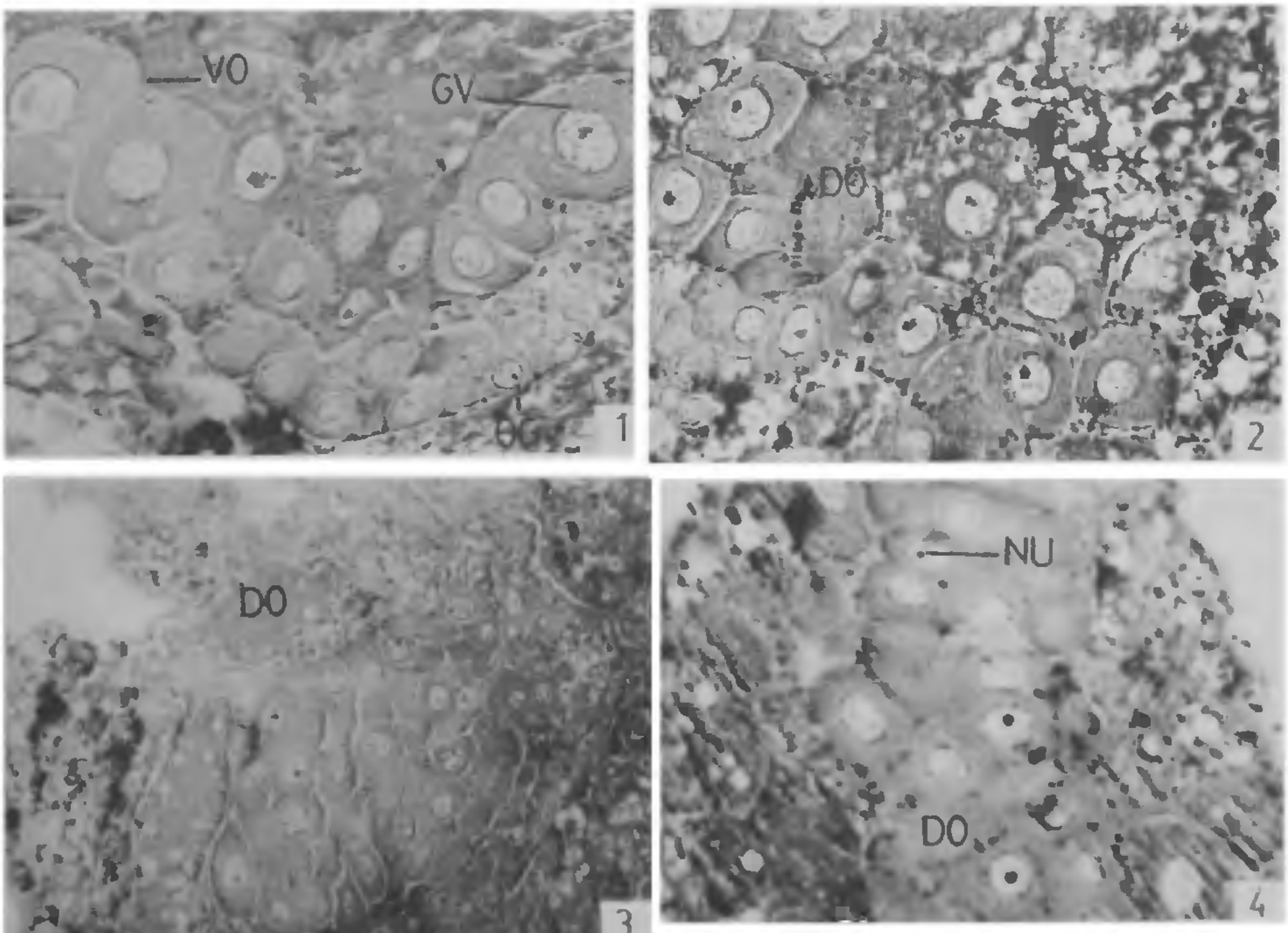
C. rajadhari was collected from Kham river, near Aurangabad, Maharashtra and they were acclimated to the laboratory conditions for a week. Acute bioassay studies were conducted according to standard methods[6]. All acute exposures with mercuric chloride were for 96 hr, static at $26 \pm 1°C$ in dechlorinated water. The survival data for 20 animals per each concentration were used to calculate the median lethal concentration (LC_{50})[7]. Change in colour was observed and the animals usually turn over prior to death, providing good indication of pending mortality. The LC_{50} values of mercury were found to be 0.009120 ppm for 24 hr, 0.006918 ppm for 48 hr, 0.005784 ppm for 72 hr and 0.004786 ppm for 96 hr.

Thereafter healthy, non-ovigerous, intermoult (stage-C) animals of identical size (mean total length = 2.5 cm) and ovarian stage (matured with green coloured ovary) were selected and divided equally into two groups: control and experimental. The experimental prawns were exposed to the sub-acute concentration of 0.00069 ppm of mercuric chloride for 30 days. Animals were fed on alternative days with wheat bran and green algae. The medium was changed on alternate days. After every 10th day, the ovaries from the living animals of both groups were dissected and fixed immediately in Bouin's fluid for 24 hr. The tissues were cut at 7 μm and stained with Harris' haematoxylin and eosin for histological observations.

On exposure to mercury, a burst of hyperactivity

involving darting movements and rapid flexures of caudal region were noticed followed by immobilization response. Loss of balance was then observed by upside down swimming, vigorous beating of both pleopods and mouth parts. After the loss of righting response, the prawns settled to the bottom and remained stationary on their back. Thereafter the pleopods ceased to beat, while the mouth parts continued their movement for a while, which also stopped as the animals succumbed to death.

In the ovaries of control prawns *C. rajadhari*, the oocytes appeared normal and maturation was well defined (figure 1). During sub-acute exposure of mercuric chloride, the initial changes noted were cellular swelling, increase in vacuolated oocytes, degenerations of oolemma and ovarian stroma (figure 2). Subsequently, oocytes lost their characteristic shapes. The degenerative changes of oocytes become more generalized after 20-day exposure with fusion of adjacent oocytes and extensive necrosis (figure 3). The process of degeneration still advanced with more nuclear pycnosis and cytolysis of oocytes after 30-day exposure (figure 4). Meanwhile the degenerated ovary was hypertrophied with haemocytes. The primary action of mercury must be on the membranes of cells and it interferes with a number of membrane func-



Figures 1–4. 1. Photomicrograph of T.S. of unexposed control ovary showing normal oocyte maturation (× 250), **2.** Sectional view of ovary after 10-day sub-acute exposure of mercuric chloride showing swelled and vacuolated oocytes (× 250), **3.** Sectional view of ovary after 20-day exposure of mercury chloride showing fusion of oocytes due to extensive necrosis of oolemma (× 150), **4.** Sectional view of ovary after 30-day exposure of mercuric chloride showing more advanced degeneration of oocytes (× 250). (DO—degenerating oocyte; GV—germinal vesicle; NU—nucleolus; OG—oogonia; VO—vitellogenic oocyte)

tions. Mercury inhibits the permeability of nutritionally important molecules such as sugars and amino acids[2].

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ACTIVITY LEVELS OF SUCCINATE DEHYDROGENASE (SDH) IN CELL-FREE SYSTEM UNDER DIETARY STRESS IN *ANABAS SCANDENS* (CUVIER)

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THE activity levels of succinate dehydrogenase (SDH) under experimental conditions in fish tissues have not been thoroughly investigated. Any alteration in the intermediary metabolism due to dietary stress is bound to affect the activity of this enzyme since SDH is one of the key enzymes of Krebs cycle. An attempt was therefore made to study the activity levels of SDH under high protein and high carbohydrate diet as well as under starvation in a freshwater carnivorous teleost *Anabas scandens*.

Four groups of fish (comprising ten per group) of individual average weight of about 30 g were kept in well-aerated aquaria and acclimatized to the laboratory conditions. They were subjected to various experimental conditions for eight weeks. The first group was fed with 65% protein diet and the second group 65% carbohydrate diet and the third group was starved. The fourth group served as control and this group was fed with a modified commercial fish meal (table 1). Fish were fed once a day *ad libitum*. After the experimental period the fish were killed and the tissues of liver, muscle, kidney, gills and brain were dissected and a 5% homogenate was prepared in ice cold 0.25 M sucrose. The homogenate was centrifuged at 2500 rpm for 15 min and the clear supernatant was used to assay SDH by the modified method of Nachlas *et al*[1]. The data was computed with reference to the mean value, percent change and standard deviation.

All the fish survived throughout the experimental period. Under 65% protein diet and 65% carbohydrate diet the activity levels of SDH showed an increase in all the tissues. The increase was greater in the brain than in other tissues. Under starvation the enzyme level decreased significantly in all the tissues except in liver (table 2).

Under high protein diet, protein catabolism and gluconeogenesis takes place in the tissues of *A. scandens*[2]. In the absence of external supply of carbohydrates the excess of aminoacids is channelled into Krebs cycle for the production of energy and supply of glucose to vital organs; hence the increase in the SDH activity levels in the tissues of this fish and this is more pronounced in the brain. It has been shown in rats that the increase in liver SDH activity could be correlated with an increase in dietary casein[3]. This finding supports the observation made in the present study.

Under carbohydrate loading the carbohydrate metabolism is accelerated due to the high influx of substrates and the SDH which plays a vital role in the oxidative metabolism of carbohydrates shows elev-

Table 1 *Composition of Diets*

Ingredients	Percentage of dry weight		
	High Protein diet	High Carbohydrate diet	Control diet
Casein*	65	-	25
Corn Starch	-	65	30
Cod liver oil	10	10	10
Alpha cellulose	15	15	15
Vitamin mix	2	2	2
Minerals	3	3	3
Agar	5	5	15

* Centron laboratories.