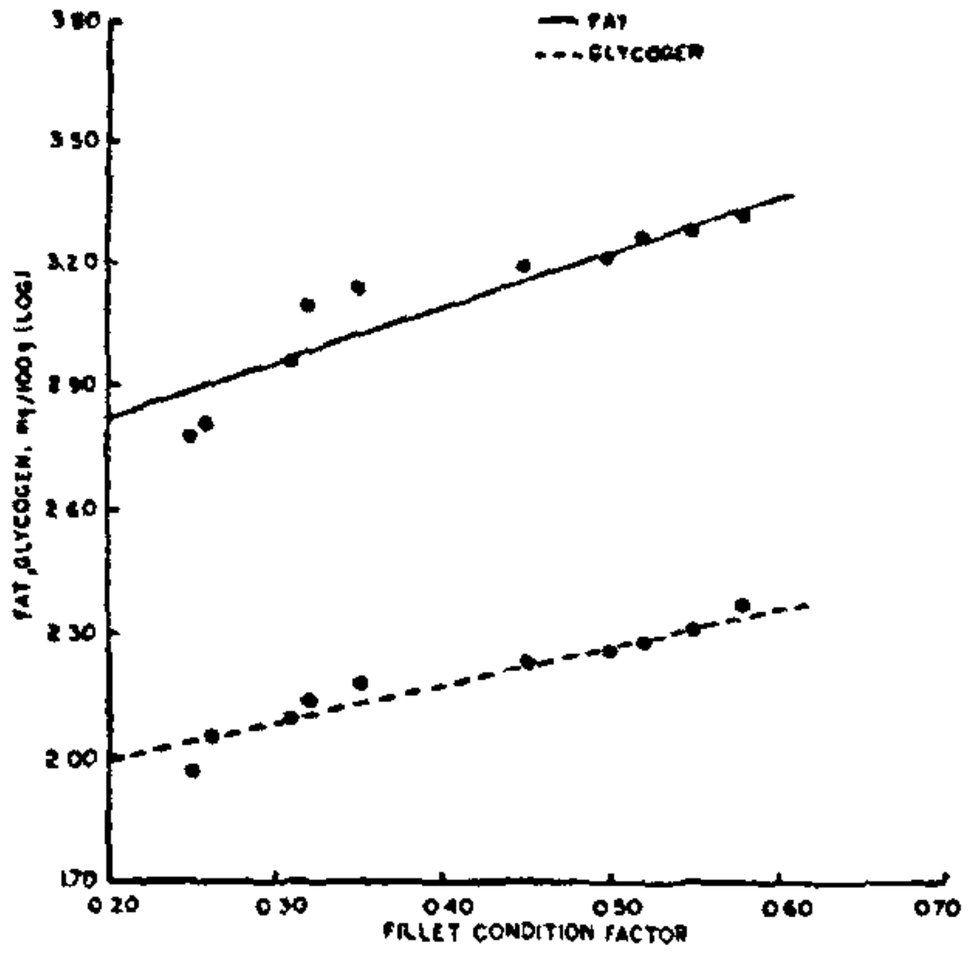

FIG. 1. Fat and glycogen concentrations in relation to fillet condition factor in *H. fossilis*.

sample used for chemical estimations was removed from the epaxial portion of the trunk. Procedure employed for the quantitative estimation of fat was the same as adopted by Jafri *et al.*[8]. Glycogen was isolated from the tissue through the method given by Ashman and Seed[9] and determined quantitatively using the technique of Montgomery[10]. The values of of fat and glycogen were recorded on fresh weight basis. Care was taken to analyse the specimens of approximately the same size ($21 \cdot 5 \pm 0 \cdot 5$ cm) to avoid the differences that are reported to be associated with the change in the length of fish[11–14].

The consistency with which the fat and glycogen concentrations of the flesh increased linearly with the fillet condition factor (Fig. 1) enabled the formulation of the regression models which permitted the prediction of the concentrations of these constituents for a given fillet condition factor of fish. The equation establishing the relationship between fat concentration and fillet condition factor was expressed as :

$$\log F = 2 \cdot 4901 + 1 \cdot 5023\ C$$

where, F was the fat (mg/100 g tissue) and C was the fillet condition factor. The correlation coefficient, 'r' being 0·884, significant at 0·001 level of probability.

The regression analysis of the relation between glycogen concentration and fillet condition factor was given by the equation :

$$\log G = 1 \cdot 7720 + 1 \cdot 0089\ C$$

where, G was the glycogen (mg/100 g tissue) and C was the fillet condition factor. The value of correlation coefficient (0 910) for this relationship was highly significant ($P < 0\ 001$). Given high strength of these correlations, a major advantage of fat and glycogen estimations is that they could possilby be used as indicators of growth in weight for a particular length, nutritional status of the fish and above all as indices of the relative fitness of different environments for pisciculture. These could be done for each commercially important species if ranges of fat and glycogen concentrations in the fish are established by prior experimentation. Obviosuly, such information would be of considerable utility to fish culturists.

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PHOSPHATASE ACTIVITY IN THE HEPATOPANCREAS AND THE LARVAL DIGENEAN PARASITES OF *LYMNAEA LUTEOLA*

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THE series of changes brought about in the carbohydrates of *Lymnaea luteola* during parasitic invasion has been reported[1,2]. Changes in the phosphatase activities of the digestive gland of other molluscan

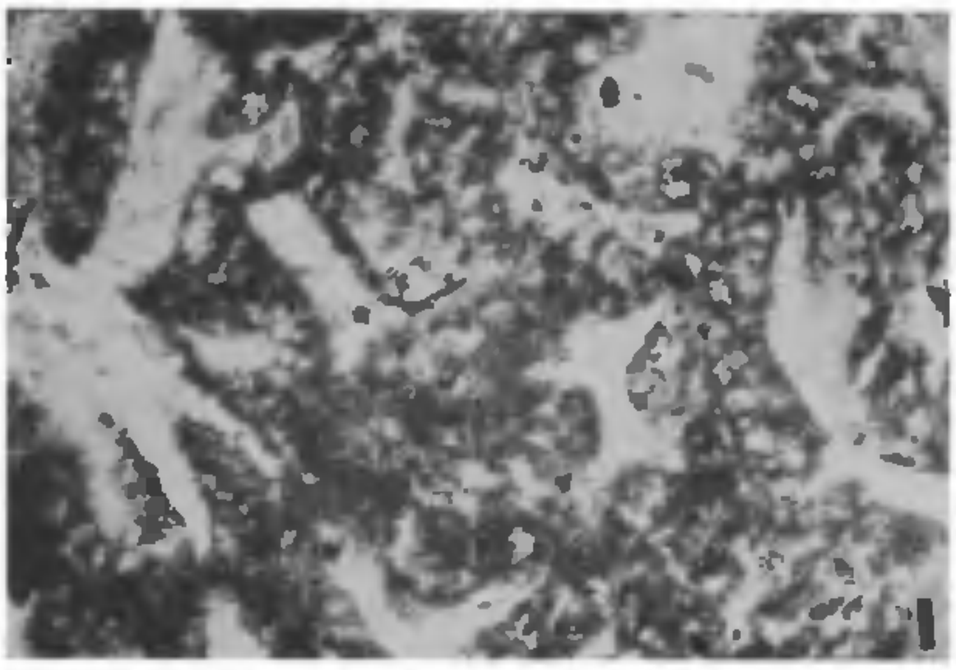

FIG. 1. AcP activity in the hepatopancreas of control *L. luteola* × 200.

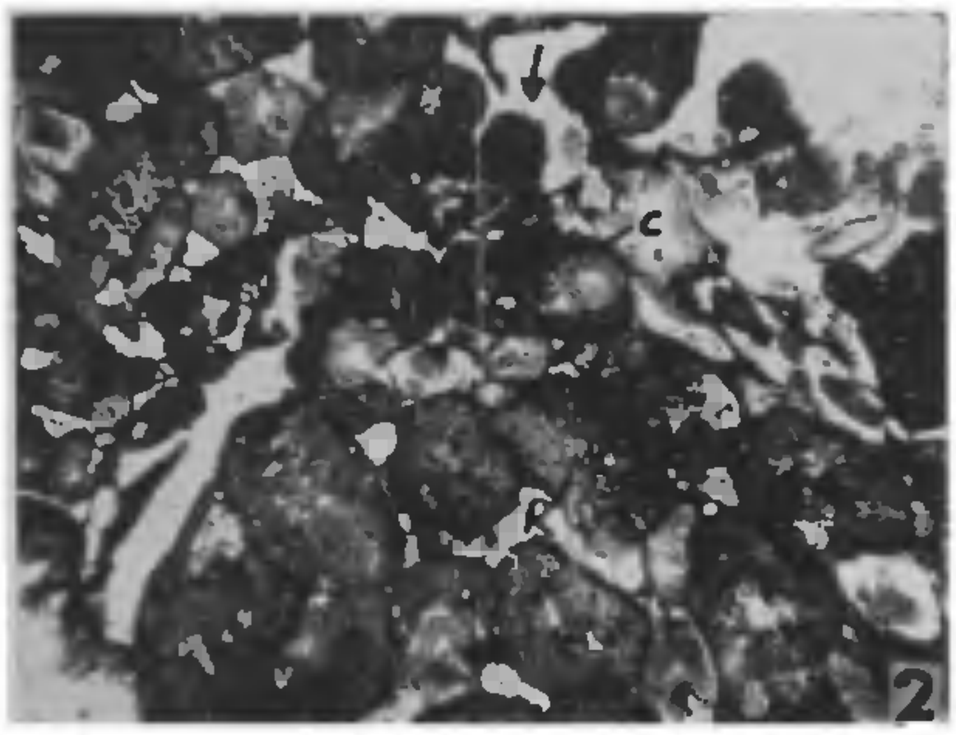

FIG. 2. AcP activity in the parasitized host. Note the heavy concentration of enzyme activity at the ruptured sites (marked with arrow) of the host and its presence on the body walls and in the caecum of the rediae (R) and cercariae (C) × 200.

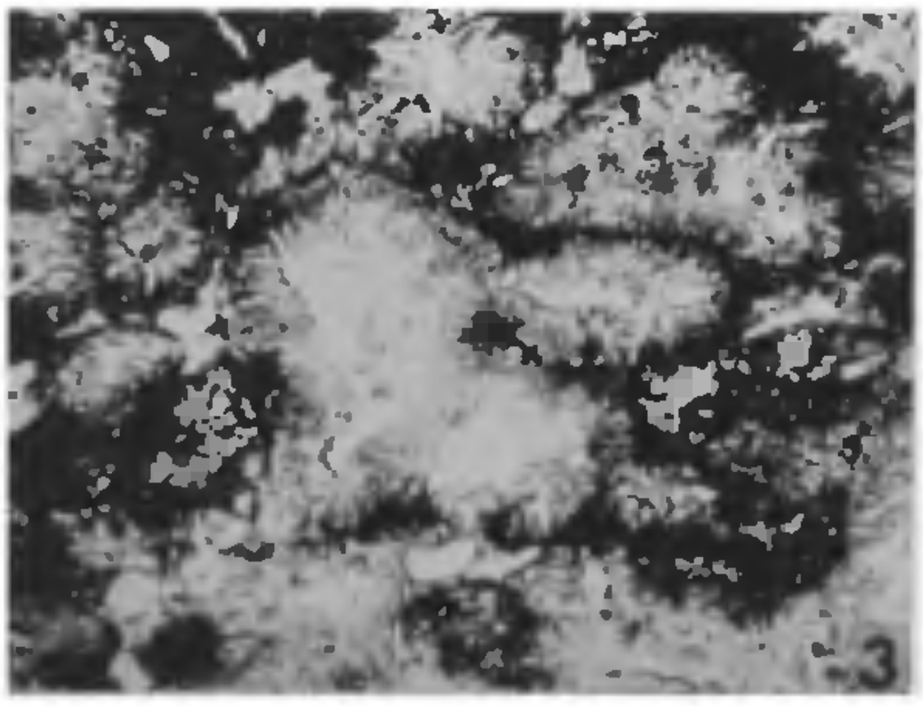

FIG. 3. AlP activity in the hepatopancreas of control *L. luteola*. Note the enzyme activity at the tunica propria × 200.

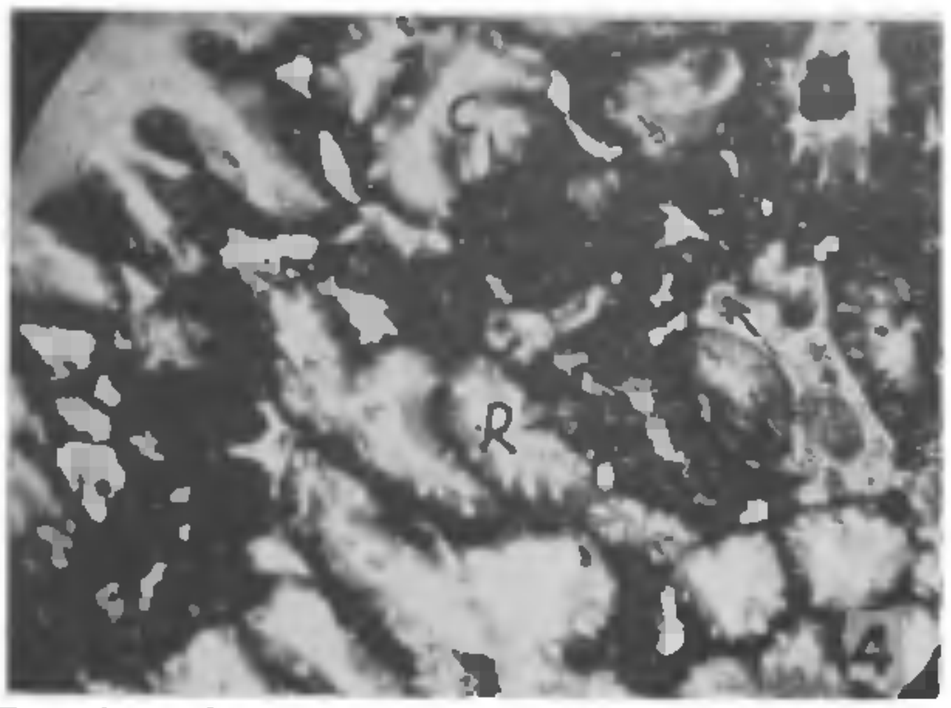

FIG. 4. AlP activity in the parasitized host. The enzyme activity in the rediae (R), Cercariae (C) and ruptured sites (marked with arrow) of the hepatopancreas can be seen × 200.

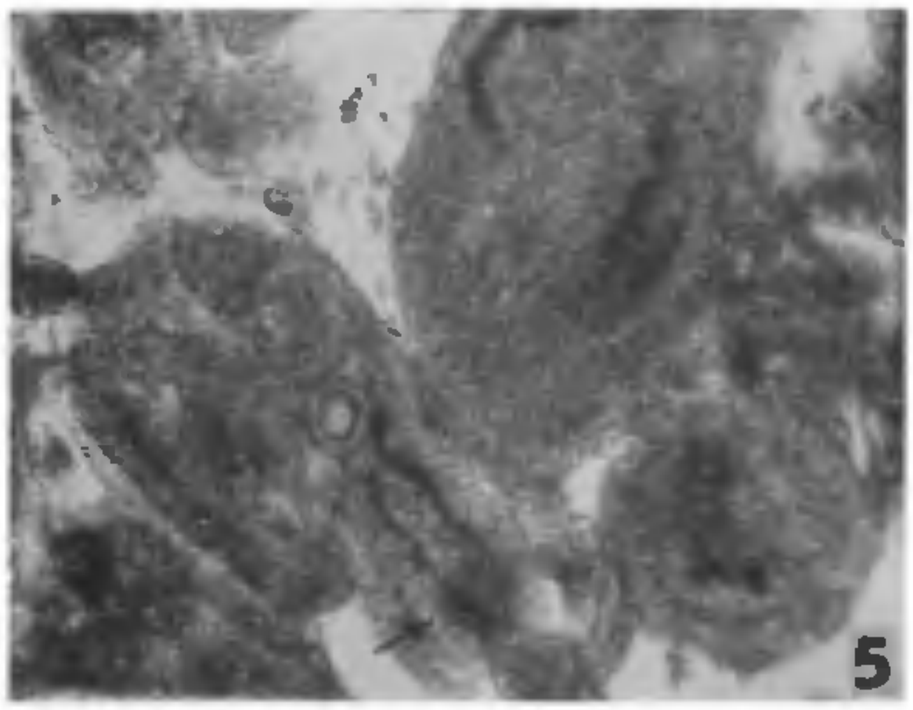

FIG. 5. AcP activity in the excretory tubules and caudal pocket (marked with arrow) of cercaria × 450.

hosts during larval trematode infections are known[3-5]. These studies are concerned with the localization of enzyme activity using histochemical techniques. The quantitative studies of these enzymes in the parasitized molluscan hosts were made by Marshall *et al.*[6], Michelson and Dubois[7] and Rama Krishna[8].

This communication deals with the phosphatase activity in the hepatopancreas of *L. luteola* during *Paramphistomum cervi* (larval forms) infection.

Freshwater snails, *L.* (*Pseudosuccinea*) *luteola f. typica* Lamarck from the ponds of the Osmania University were kept near electric light for 1 hr. Young snails free of natural infection were separated and maintained in the aquaria and were fed with *Spinacea oleracea* leaves. Five miracidia of *P. cervi* obtained by maintaining eggs in Tyrode's solution for 6–9 days and each snail was exposed to five miracidia to obtain cercaria. The hepatopancreas was dissected out, fixed in 3 changes of cold acetone at 3°C for 30 hr and paraffin sections of 6 μ were treated by lead nitrate method of Gomori[9] for acid phosphatase (AcP) and the calcium cobalt method[10] for alkaline phosphatase (AlP).

Slight AcP activity was observed in the hepatopancreas of normal *L. luteola* (Fig. 1), in the nuclei

of host cells and in the cytoplasm as finely granular brownish black precipitants. In the infected snails the activity increased in all these sites. The AcP activity was also found intertubularly in areas, where cells comprising the tubules are lysed or ruptured (Fig. 2). The AcP and AlP activities were also localized in the gut of rediae caecae, caudal pocket and the excretory canals of the cercariae. The AlP activity was greater than AcP activity in the normal *L. luteola* (Fig. 3), the activity is associated with the nuclear membrane, and the cytoplasm mainly in that portion of each cell distad to the tubular lumina (Fig. 3). In the hepatopancreas of infected snails, the AlP activity increased, rediae caused maximum damage to the hepatopancreas resulting in increased (Fig. 4) enzyme activity at the site of ruptured cells. The results indicate that AlP activity is more both in the normal and infected snails and, therefore, it is possible that its carbohydrate metabolism takes place at an alkaline pH. Increased AcP and AlP activities in the infected snail may be due to extensive hydrolytic cytolysis occurring in the hepatopancreas[3,4] and tissue destruction released, enzymes bound in the digestive gland. AcP and AlP was also observed in the excretory tubules of cercaria (Fig. 5) suggesting involvement in energy transfer and in selective excretion. The presence of these enzymes and especially AcP in the area of the caudal pocket may be associated with energy production.

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INFLUENCE OF HOST PLANTS IN THE CONTROL OF GREEN PEACH APHID *MYZUS PERSICAE* SULZ. WITH DIFFERENT CHEMICALS

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The variation in susceptibility of an insect to insecticides is largely governed by the external changes in the environment. Among the variety of environmental factors, the nature of host plants influences the susceptibility to insecticides to a greater extent[1–2]. *M. persicae* is a serious pest infesting chillies[3], tobacco[4], tomato[5] and many other crops. The present studies were undertaken to find out the influence of the host plant species on the susceptibility of the aphid to certain insecticides.

Experimental

Three field experiments were conducted in a randomized block design with four replications in three crops, *viz.*, chillies (*Capsicum annum* L.), tobacco (*Nicotiana tabacum* L.) and tomato (*Lycopersicum esculentum* Mill.) with a plot size of 25 m^2, at Thirumalairayapuram village where severe incidence of the pest was noticed. Five different spray formulations were tried on all the three crops. Five plants in each plot were selected at random and they were tagged. In each plant five leaves each from the top, middle and bottom regions were selected and the aphid population counted. Counts were made prior to spraying and after three, seven and fourteen days of the spray. The average population per plant after 14 days of the spray was worked out. Square root transformation was adopted for statistical interpretation.

The results presented in Table I indicate that pirimicarb 0·1% and FMC 35001–0·048% were highly effective for the control of *M. persicae* with all the three crops. All the five chemicals tested were highly effective on tobacco. Between tomato and chillies, insecticides pirimicarb and FMC 35001 were equally effective, whereas in the case of the other insecticides, the susceptibility of the aphid was greater when it occurred on tomato than on chillies. This indicates the possible existence of biotypes. All the insecticides excepting pirimicarb failed to register significant reduction in pest population on chillies. This is in general agreement with the large scale field observation. The effectiveness of pirimicarb has been already reported on chillies[6], tobacco[4] and tomato[7] against *M. persicae*.

Reports about the differential susceptibilities of an insect species infesting or reared on different host plants to one and the same insecticide are not infrequent. The susceptibility of *M. persicae*, to nicotine