by bacteria, since the dominant heterotroph *P. blanci* does not remain in this zone. Pant *et al*[2] found a high load of coliform bacteria in this system. Given that only a fraction of NPP is available for the herbivores, they appear to have adapted themselves to feed upon ditritus. Thus, detritus forms the major food source to macrobenthos and a supplementary one to zooplankton.

Exclusively, carp fish are found in the lake and among them the important food fish are: *Schizothorax, Tor* and *Cyprinus carpio*. The first one is a herbivore whereas the latter two are omnivores. Since the profundal zone is uninhabitable due to pollution, toxic gases and anoxic conditions[7], these fish remain more or less confined to the epilimnetic waters. Omnivorous fish feed on a variety of food items. However, the utilization of the food depends upon the relative abundance of the developing stages both of these fish and their prey species. Since most of the energy in the plankton component is dissipated through decomposer chains, very little is available to sustain the biomass of fish. It is therefore believed that the lake Nainital has lost the potential to increase the productivity of indigenous (carps) fish.

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LESION-PRODUCING NEMATODES OF *MUSA PARADISIACA*—HISTOPATHOLOGY AND ABUNDANCE

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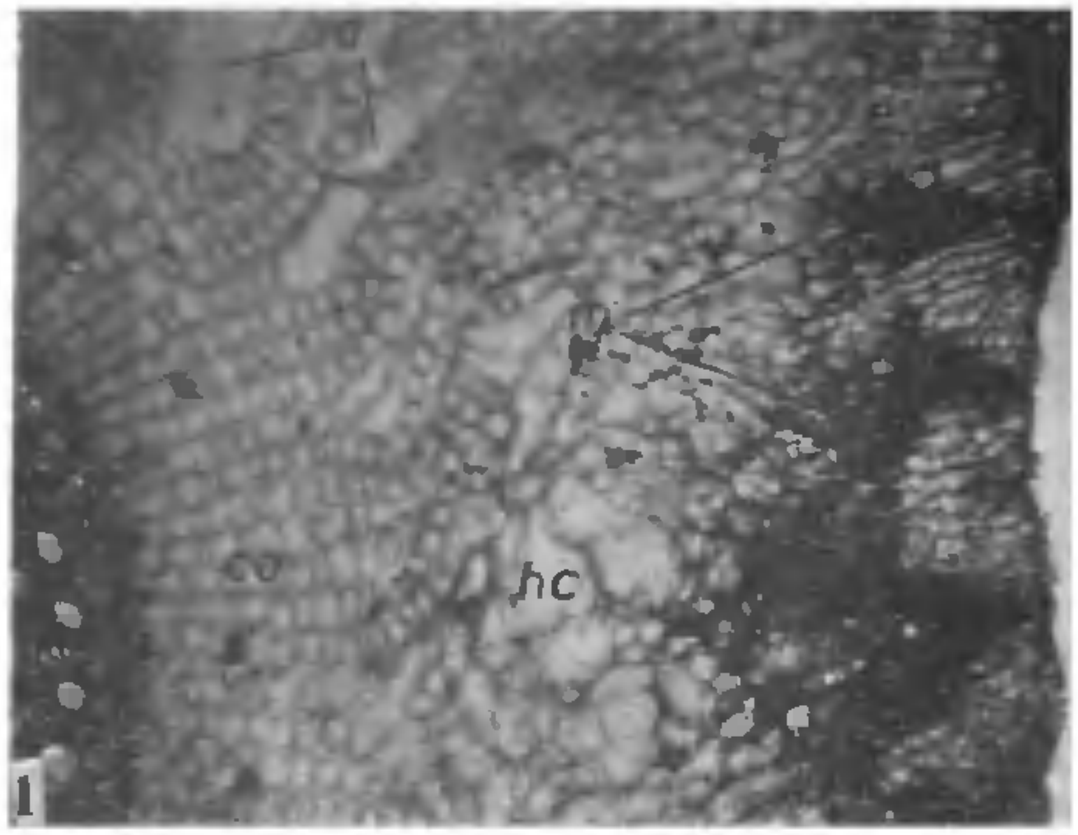
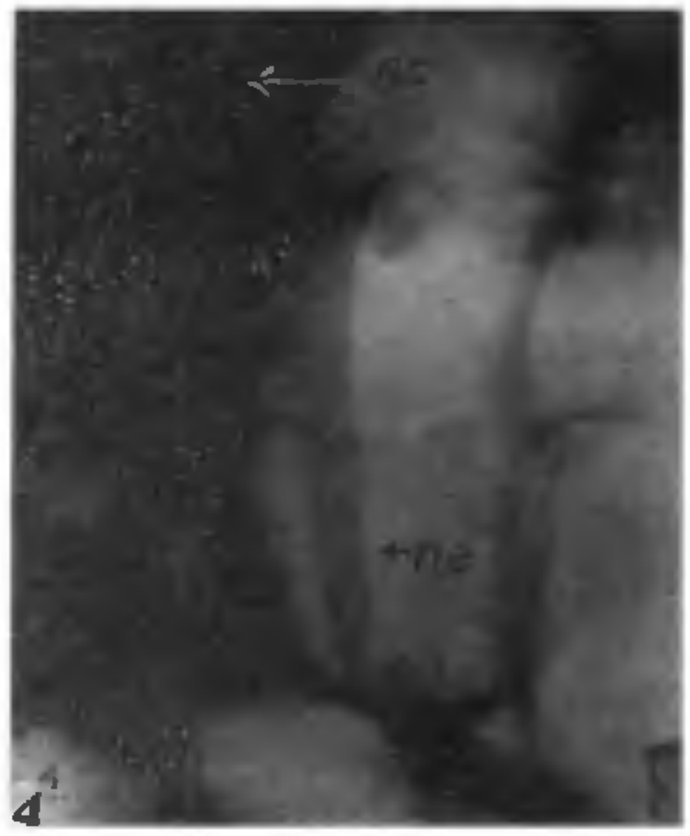
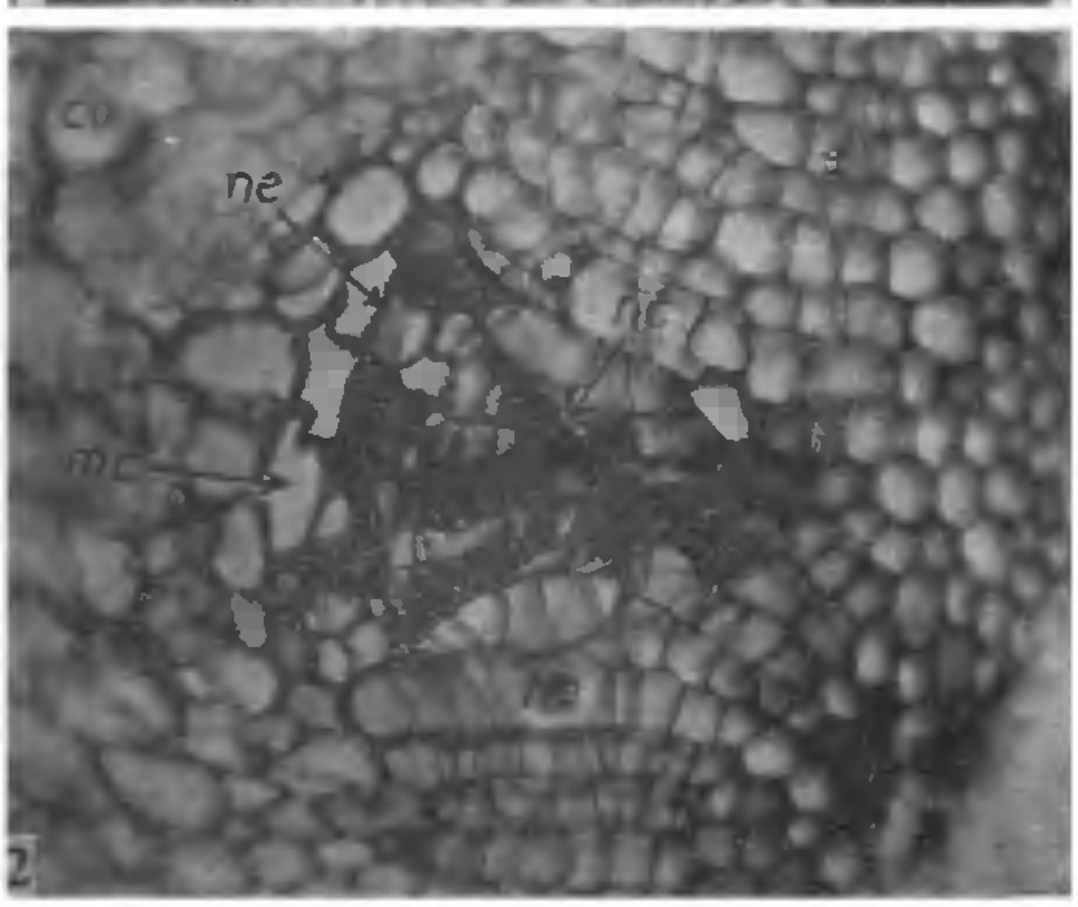
PLANT parasitic nematodes are considered[1] as the most important non-insect pests that hamper agriculture production in India. Cultivars of the banana plant, *Musa paradisiaca* in Kerala have been known[2] to be attacked by eight species of root-infesting nematodes, *viz. Radopholus similis, Helicotylenchus multicinctus, Helicotylenchus crenacauda, Rotylenchulus reniformis, Dolichodorus microannulatus, Xiphinema elongatum, Longidorus saginus* and *Meloidogyne incognita*. The first seven species contributed to necrosis and consequent lesioning of roots to a greater or lesser extent. Earlier studies on these nematodes carried out in this state reported their occurrence[2,3] and estimated their population[4,5]. The present study was to obtain information on the histological changes in roots of *M.paradisiaca* (cultivar 'palayamthodan') infested by these nematodes and to estimate their population under different intensities of necrotic lesioning.

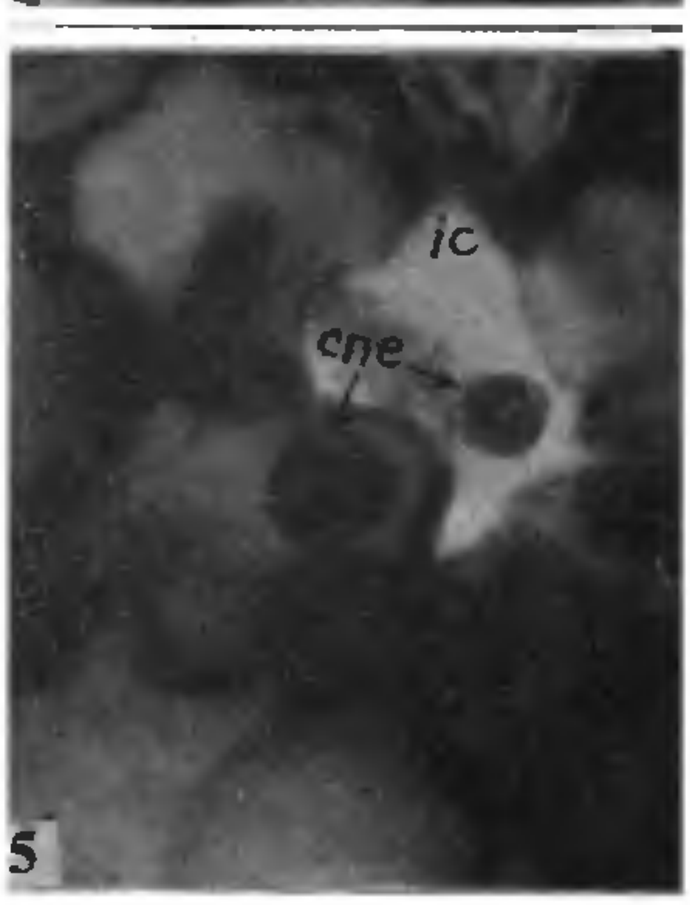
To study the histopathology of the infected roots, hand sections of fresh roots fixed in FAA (40% formalin 6.5 ml, glacial acetic acid 2.5 ml, 50% ethanol 100ml) were taken, stained with appropriate animal stains (0.1% acid fuchsin lactophenol) or plant stains (0.5% aqueous safranin) and examined. The weighed samples of the infected roots in sufficient quantities of water were mechanically macerated in a waring blendor and nematodes isolated from the macerated tissue by the seive method[6] utilising their natural motility and then identified to species level.

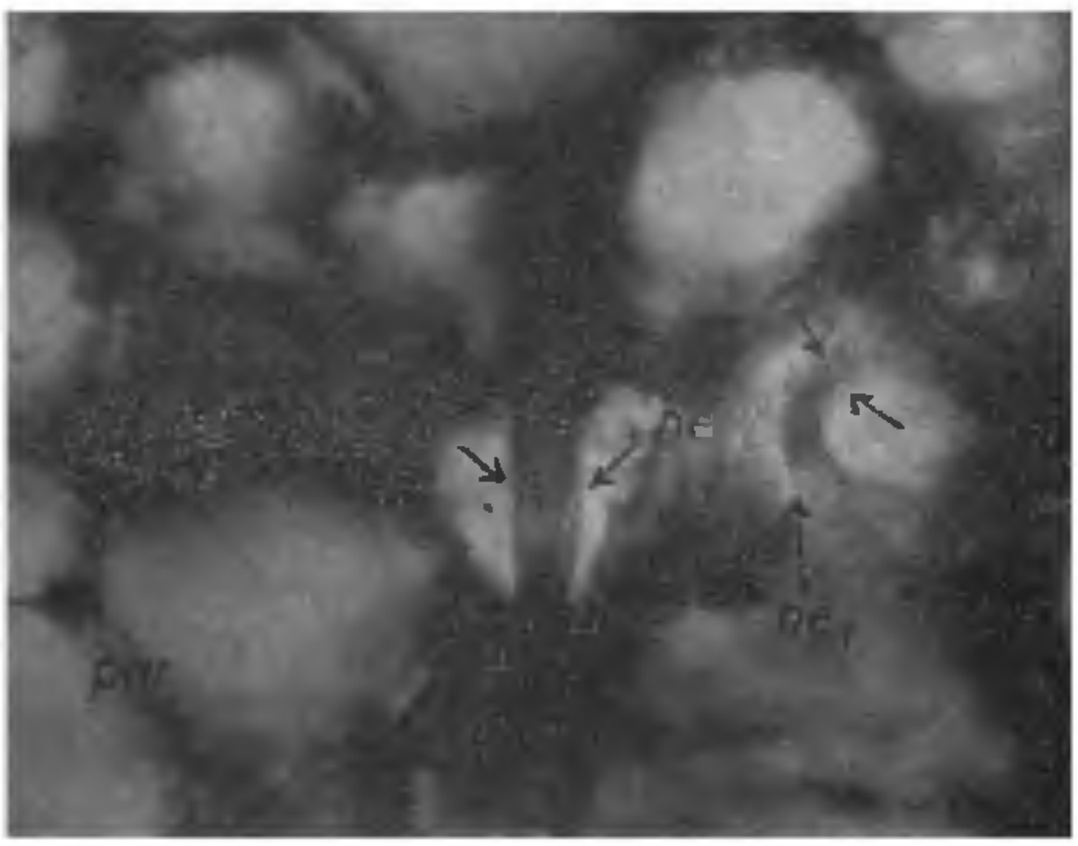
Reddish brown lesions were reported[5,7] on roots infested by the nematodes. When lesioned roots were examined, cell necrosis was localized in the epidermis and peripheral cortical region only (figure 1). Nematodes were located both intercellularly and intracellularly (figures 3 and 4) in the cortex. Intracellular location was not reported in *M. ornata*[6], but in the present study, this was more frequently observed (figures 4,5 and 6). Nematodes apparently ingested the cytoplasmic contents of the cell, the cell wall ruptured and an irregular cavity formed in place of the cell

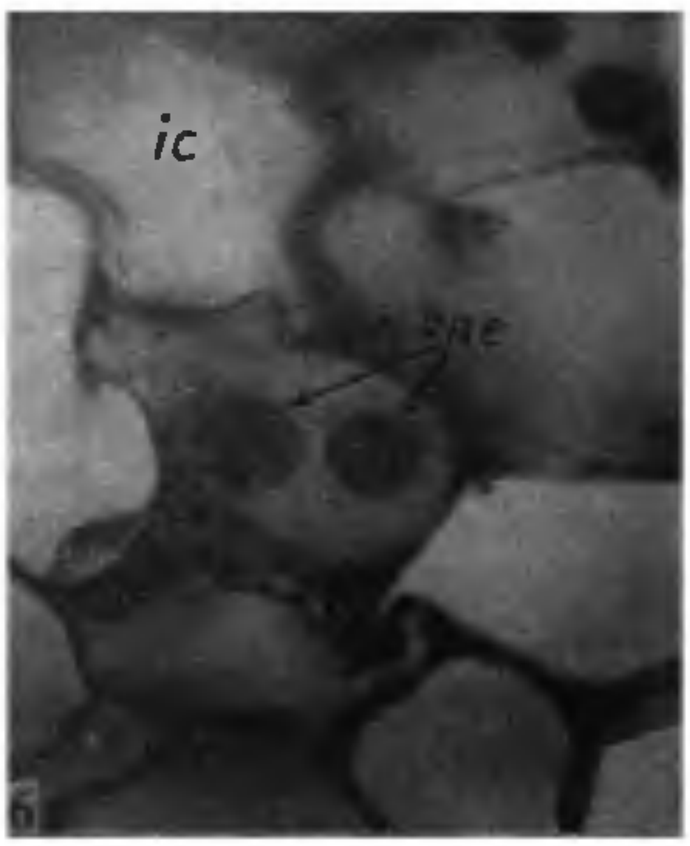












Figures 1–6. Cross section of Lesioned Banana Roots. **1.** Live root stained with aqueous safranin. **2–6.** Roots fixed in FAA and stained with 0.1% acid fuchsin-lactophenol. a–air cavities in cortex; co–cortex; one–cross section of nematode; e–exodermis; hc–cells with slight hypertrophy; ic–irregular cavity; in–infected area; mc–cavities with irregular walls; nc–region of necrosis; ne–nematode; ne$_1$–coiled nematode; par–parenchyma.

(figures 2 and 5), through which the nematodes moved (figure 5). Adjacent cavities coalesced forming tunnels, which had thick brown walls (figure 2). Hypertrophy of nucleus and nucleolus was reported in *M. ornata*[7], but this was not observed in *M. paradisiaca*. Ruptured cells turned brown or balck. Cells surrounding infected area changed their oval shape and became elongated and cylindrical (figure 2). In sections of roots, it was found that, up to three nematodes were often present in a coiled position within the cell (figure 3). More than two cross-sections of nematodes and rarely up to seven in the same cell were observed (figure 6). The latter probably belonged to two or three coiled nematodes.

The mean number of nematodes present in the roots differed from sample to sample (table 1) and these differences were related to the intensity of necrosis found on the root surface. When no visible lesions were found on the root surface, *i.e.*, at an early stage of infection, the total number of nematodes was very low and in this the proportion of *R. similis* and *H. multicinctus* was low compared to that of other species (sample 1 table 1). As the necrotic lesions appeared like streaks on the surface and latter, when the necrosis spread, the nematode population steadily increased (sample 2 to 4), and finally when the root surface turned totally brownish black (sample 5) the nematode population reached peak density. Further at this latter stage, only *R. similis* and *H. multicinctus* remained in the root. With the increase in the density of nematodes, a progressive simplifiction of the nematode community was noted in the root microhabitat. To the best of our knowledge this aspect has not been stressed by previous workers. The present as well as earlier[5,7,8] showed that the ability of *R. similis* and *H. multicinctus* to cause necrosis in banana roots was more than that of any other species. Probably severe necrotic conditions produced in the roots by *R. similis* and *H. multicinctus* made the roots unfavourable for other species of nematodes leading to the simplification of the nematode community. It may also be pointed out here that in an earlier study[5] the maximum number of *H. multicinctus* present in one gram of highly necrosed banana roots was 2200, while in the present study similar roots contained only a miximum of 950 nematodes per gram. This suggested that while roots could support a high population of a single species of nematode like *H. multicinctus*, only a smaller number could be supported when more species were present and probably also when *R. similis*

TABLE 1

Population of nematodes in 3 gm live roots of Musa paradisiaca

Sample No.	Mean diameter of root (mm)	Index of necrosis on root surface (nominal scale)	Mean no. of nematodes per 3 gm root	Mean percentage of individual nematode species	Mean population density of individual nematode species
1	3.7	No visible necrotic lesions	120	a 17.33 b 25.33 c 57.33	a 21 b 30 c 69
2	7.5	+	530	a 20.75 b 26.79 c 52.45	a 110 b 142 c 278
3	7.8	++	2020	a 61.39 b 32.17 c 6.43	a 1240 b 650 c 130
4	5.2	+++	2082	a 66.10 b 30.06 c 3.79	a 1377 b 626 c 79
5	7.5	++++	2823	a 61.45 b 38.50 c nil	a 1736 b 1087 c nil

Number of '+' signs indicate intensity of necrosis; a—*Radopholous similis*; b—*Helicotylenchus multicinctus*; c—Other nematodes. Figures represent mean of three replicates.

was one of them. The present study also suggested that nematode density was not strictly related to root diameter but was probably time-dependent *i.e.* the duration for which the root was subjected to infestation.

Earlier, it was found[5] that at Kariavattom *H. multicinctus* was the most predominant nematode affecting *Musa paradisiaca*. The present study showed that *R. similis* was the most abundant nematode in the necrosed roots of banana followed by *H. multicinctus*. Thus in the course of about five years *R. similis* attained a more dominant status as the root parasite of the banana plant, compared to *H. multicinctus*, in the same locality.

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EFFECT OF DIMETHOATE ON CHOLINESTERASE ACTIVITY IN THE ORGANS OF A TELEOST *H. FOSSILIS*

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COMPARED to the investigations on bioassay tests to assess the mortality rate and histopathological lesions, very little attention has been paid to study the effect of insecticides on the biochemical changes in the vital organs of the animals. Inhibition on acetylcholine sterase enzyme in the brain and serum has been reported in the fish exposed to organophosphorus compounds[1,2] which are widely used to protect the crops from the pests. The present study reports the changes in ChE activity on the liver, kidney and brain of a teleost *H. fossilis* exposed to sub-lethal concentration of dimethoate.

Maintenance and size range of the fish used in the experiment have been described earlier[3]. The fish were exposed to a sub-lethal concentration *i.e.* 10 ppm of dimethoate (30% E.C. Gujarat Agrochemical Industries, Ahmedabad) for 48 days. The 96 hr-TLm value had already been worked out[3]. The fish were sacrificed from the normal as well as treated tanks at eight-day intervals upto 48 days. ChE activity was estimated by using the method of Huerga *et al*[4].

Table 1 shows the ChE activity in the liver, kidney and brain of the normal as well as treated fish. During the first 16 days of treatment with 10 ppm of dimethoate, the ChE activity of all the tissues showed a decline. Thereafter the tendency was for recovery showing normal activity with minor fluctuations in the liver and the brain by the end of 24th day. The activity again declined in the tissues of treated fish.

TABLE 1

Effect of dimethoate (10.0 ppm) on the ChE activity in the organs of H. fossilis

Exposure period (days)	Liver	Kidney	Brain
Normal	0.33 ± 0.04	0.38 ± 0.04	0.55 ± 0.04
8	0.32 ± 0.02	0.33 ± 0.00	0.33 ± 0.04
16	0.31 ± 0.04	0.23 ± 0.03	0.24 ± 0.02
24	0.33 ± 0.00	0.26 ± 0.00	0.52 ± 0.04
32	0.20 ± 0.03	0.17 ± 0.00	0.45 ± 0.02
40	0.25 ± 0.03	0.21 ± 0.03	0.49 ± 0.02
48	0.21 ± 0.04	0.22 ± 0.04	0.50 ± 0.01

Activity of enzyme is expressed as μ mole/100 mg/hr values expressed are mean ± S.D. of 6 individuals.

The organophosphorus compounds are known to inhibit ChE activity[5,6]. These compounds which act as inhibitors combine with cholinesterase through the electrophilic *p* atom to form an irreversible enzyme inhibitor complex and the cholinesterase becomes unhydrolysed[7,8]. During the present work the activity of ChE however became normal in the liver and brain