DISCUSSION

An acute infection of Thelohanellus ophthalmicus Haldar, Das and Chandra, 1983 is observed in the gills of *Catla catla*. Degeneration, atrophy and other necrobiotic changes as well as inflammation and repairing of the host tissue as host response, confirms the pathogenic nature of this parasite.

The epithelium of gills forms a barrier between the fish's blood and the surrounding water. Gaseous exchange needed to sustain life takes place through this barrier. Any morphological alteration by the parasites hinders the respiratory, secretory and excretory function of this organ.

Clubbing of the gill filaments due to excessive proliferation of interlamellar cells is an adaptive measure to protect the gill filaments from continual irritation caused by the lack of an adequate external gill covering. Clubbing of the gill lamellae was also observed by Takashima (1982).

Large amount of mucus is secreted which is an indication of the irritant nature of the parasite. It is presumed that abundant mucus mechanically disrupts gill function and causes asphyxiation of the fish.

Host response to this parasite is quite adequate as large number of neutrophils, eosinophils, lymphocytes and plasma cells are observed in the immediate vicinity of the cyst. Repairing of the infected tissue is also clearly observed in the gills.
A large number of inflammatory cells are present along with the degenerating spores. A few fibroblasts are also seen in this repairing zone indicating a strong host response for this parasite. This is not typical of a granulom formation as observed by Dykova and Lom (1978) in the gills of a perch as tissue response to *H. psorospermica*, as the pseudoepithelial cells are not observed surrounding this mass in the present study. Dykova and Lom found that when the cyst is full of mature spores an inflammatory reaction is mounted resulting in the rapid replacement of the cyst by granulomatous tissue. Their observations are in agreement with those of Finn and Nielsen (1971a, b) and Lucky (1970) who found that host tissue response to *Myxobolus ellipsoides* begins only when the parasite has reached a certain minimum size. Dykova and Lom stated that at first relatively small cysts are overlaid by massive hyperplasia of the adjacent epithelium. This is followed by invasion of the parasitic mass by macrophages, which remove the spores by phagocytosis. Later infiltration by fibroblasts and histiocytes complete the typical granuloma formation. The outer layers of the granulomas are formed by pseudo-epithelial cells derived from mesenchmal cells. In contrast to this in the present studies, cysts are not invaded by inflammatory cells; instead, infiltration of neutrophils, eosinophils, lymphocytes and plasma cells is observed close to the cyst. The present author presumes that spores from the cyst have moved out of the cyst into this inflamed mass and later infiltrated by fibroblasts thus a repairing tissue is seen adjacent to the cyst. Almost
similar findings were given by Hoffman, Putz and Dunbar (1965), while discussing the histopathology of *Myxosoma cartilaginii* in centrarchid fish.

Recently Kalavati and Narasimhamurti (1985) studied the histopathological changes in the gills of *Channa punctatus* infected with *Hennequya waltairensis* but the host response was poor in that case. They did not observe any granulomatous tissue in the gills although they observed hypertrophy of the host tissue and vacuolisation of the associated cytoplasm. They also observed that when the cysts were mature, degenerative changes appeared more conspicuous and were associated with accumulation of macrophages; the rupture of the cyst was associated with haemorrhage. Similar observations were made with *H.*psorospermica by Dykova and Lom (1978) but they did not observe associated haemorrhage. However, Greven (1956) reported the occurrence of haemorrhage with this parasite. Rupturing of the cyst is not observed in the present studies.

An early healing response to *Hennequya ameirusis* in the barbels of *Ameirus nebulosus* was seen by Nigrelli and Smith (1940). However, they could not observe later stages of repair. Besides the mucoid material in the cysts, they observed lymphocytes, some fibroblasts and occasionally melanophores.

**Conclusion**

The present studies and an evaluation of the literature leads the author to conclude that myxozoans are pathogenic in
nature. They cause atrophy and degeneration of gill filaments along with some other necrobiotic changes. Thus, the function of the gills is impaired by reduction of the functional respiratory surface and also by impairment of circulation. Large amount of mucus is secreted which is an indication of the irritant nature of the parasite. Excessive mucus secretion causes asphyxiation of the fish. When the cysts are filled with mature spores, neutrophils, plasma cells, lymphocytes, eosinophils accumulate near the cyst as a host response. Plasma cells may be secreting antibodies in response to the antigens from the parasites. Repairing of the host tissue also takes place as fibroblasts and lymphocytes are seen among the degenerating spore mass. This shows an early healing response from the host tissue.