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Histopathology of Gill Lesions in Channel Catfish Associated with *Henneguya*

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A number of epizootics occurred among farm-reared channel catfish (*Ictalurus punctatus* Rafinesque) in Mississippi during 1981–1983, with similar clinical signs. Mortality prevalences as high as 100% in affected populations were reported. Both fingerlings and market-size (>0.6 kg) channel catfish were affected. Gross lesions and clinical signs associated with the mortalities included swollen gills, lethargy, respiratory distress (crowding near aeration equipment), and sloughing of gill tissue. Neither pathogenic bacteria nor viruses were isolated from moribund fish. An investigation of the histopathology associated with a number of similar epizootics was undertaken in an effort to determine the factor(s) contributing to the loss of the fish.

Market-size and juvenile channel catfish were collected from ponds where epizootics were in progress. Fish from apparently healthy populations served as controls. Clinical signs and gross lesions were recorded at the pond site. Gill, posterior kidneys, liver, spleen, and intestinal tissues were removed from sampled fish and preserved in 10% neutral buffered formalin. These tissues were embedded in Paraplast (Lancer Co., St. Louis, Missouri 63178, USA), sectioned and stained with hematoxylin and eosin (Humason, 1967, Animal Tissue Techniques, W. H. Freeman and Co., San Francisco, California, 569 pp.). Gill samples were also fixed in glutaraldehyde and subsequently prepared for examination with a Zeiss EM109 transmission electron microscope.

A severe multifocal interlamellar hyperplasia (Fig. 1) was observed in the gills. Histozoic parasites were observed in the proliferative foci. These were presumptively identified as the plasmodium of a myxosporan. Examination of serially sectioned material collected during one epizootic revealed these organisms through the entire thickness of the gill filaments. Immediately surrounding the parasites was a zone of reactive host cells enclosed by a granulomatous tissue proliferation. In the more severe cases, the proliferative tissue occluded the lamellar troughs and overgrew the distal ends of the lamellae by several cells in thickness. These gill lamellae were often severely distorted.

Microscopic examination of one gill revealed several developmental stages of the plasmodium which allowed the identifi-

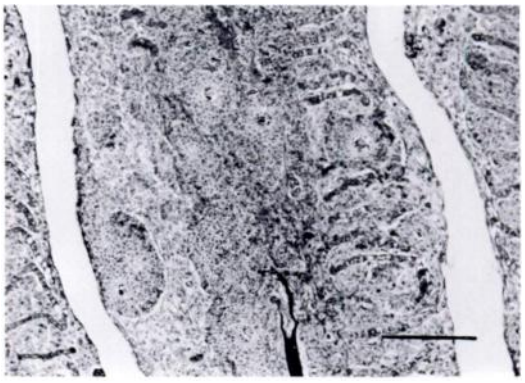

FIGURE 1. Gill filament from channel catfish with a severe multifocal interlamellar hyperplasia and histozoic parasites. (Bar = 100 μm.)

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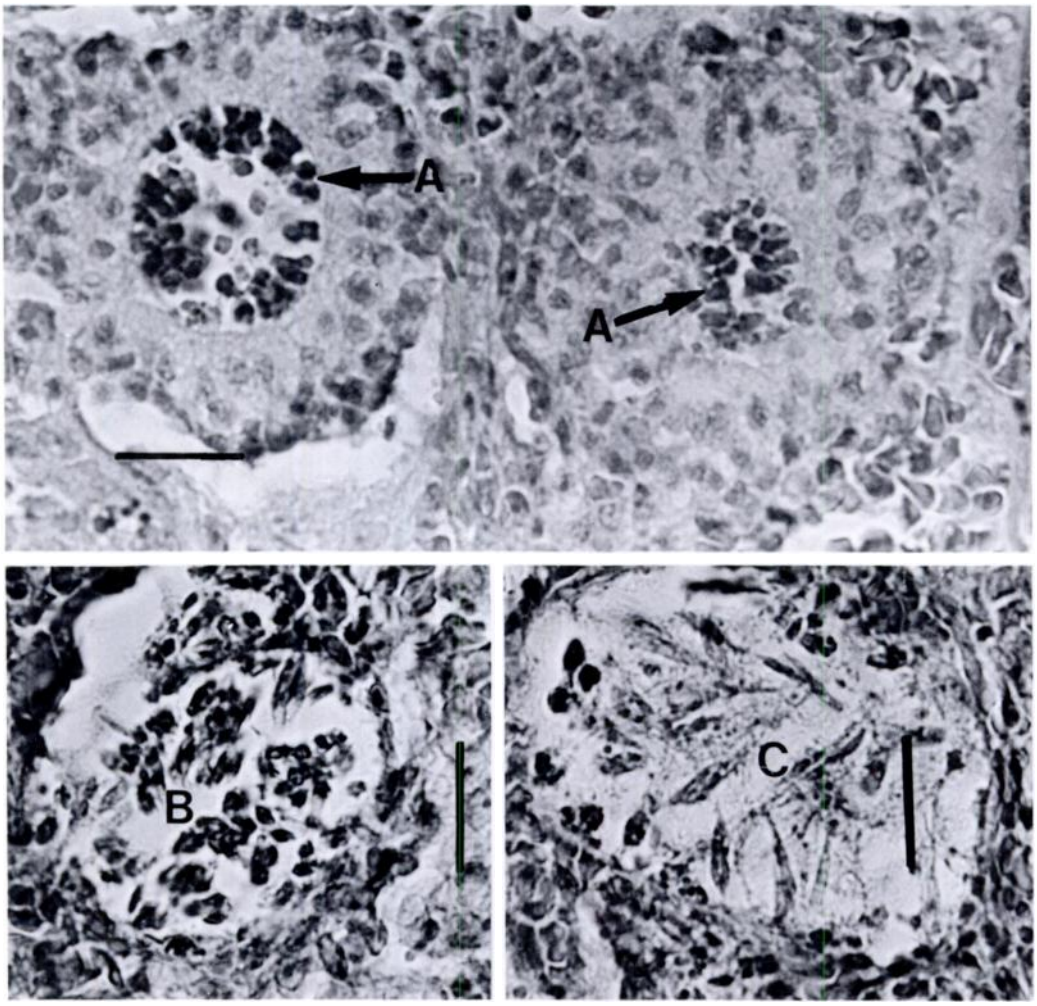

FIGURE 2. Development stages of *Henneguya* in the gills of channel catfish showing stages where generative cells predominate (A), stages with generative cells and apparently mature spores (B), and stages where mature spores predominate (C). (Bars = 25 μm.)

cation of the parasite as *Henneguya*. Figure 2 shows developing parasites at stages where generative cells predominate, at stages where generative cells and apparently mature spores are present, and at stages where spores predominate. Reactive cells surrounding the plasmodium appear to be replaced by connective tissue. Electron microscopy of a plasmodium (Fig. 3) shows an aggregation of generative cells as well as some cells suggestive of the enveloping cells described by Current and Janovy (1977, Protistologica 13: 157–167). *Henneguya exilis* Kudo, 1929 has been implicated as a pathogen capable of causing mortalities in channel catfish (McCraren et al., 1975, J. Wildl. Dis. 11: 2–7; Minchew, 1977, J. Protozool. 24: 213–220). Current and Janovy (1978, J. Protozool. 25: 56–65) described *H. exilis* as the parasite responsible for both the interlamellar and intralamellar clinial forms in channel catfish. They suggested that the different clinical forms were due to initial

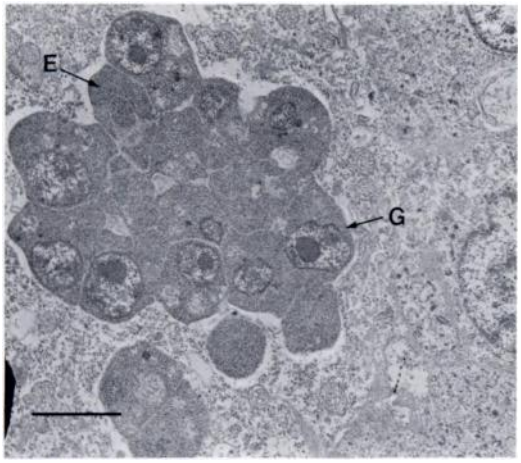

FIGURE 3. Electron micrograph of a *Henneguya* trophozoite in the gill of a channel catfish showing generative cells (G) and possibly enveloping cells (E). (Bar = 3 μm.)

site of infection, growth pattern within the host and the host immune response.

Proliferative lesions of the gills have been documented previously and are the subject of a review by Eller (1975, *In* Fish Pathology, Ribelin and Migaki (eds.), Univ. of Wisconsin Press, Madison, Wisconsin, pp. 305–330). Water quality problems, such as hydrogen sulfide (Wood, 1960, J. Water Pollut. Control Fed. 32: 994–999) or sodium cyanide (Eller, 1975, op. cit.), can produce a diffuse hyperplasia of the gills. In contrast, lesions produced in response to bacterial or protozoan pathogens are often focal or multifocal in nature (Wood and Yasutake, 1957, Prog. Fish-Cult. 19: 7–13; Wood, 1960, op. cit.). Proliferative lesions of the gills, due to a dietary pantothenic acid deficiency have been well documented for salmonids (Wolf, 1945, Fish. Res. Bull. 7, N.Y. State Cons. Dept., Albany, New York, 32 pp.; Eller, 1975, op. cit.; Poston and Page, 1982, Cornell Vet. 72: 242–261) and channel catfish (Murai and Andrews, 1975, Trans. Am. Fish. Soc. 104: 313–316; Murai and Andrews, 1979, J. Nutr. 109: 1140–1142). The lesions caused by pantothenic acid deficiency have been described as an interlamellar proliferation which develops first at the distal end of the gill filament. As the deficiency syndrome progresses, the lesion gradually encompasses the more proximal portion of the filament. In severe cases, the gill filaments take on a "clubbed" appearance and adjacent filaments may fuse. The gill lamellae of fish which are deficient in pantothenic acid have been described as sometimes being shortened but not structurally distorted. The severe proliferative gill lesions we observed in the present investigations were multifocal, with a trophozoite observed in the foci.

In summary, an interlamellar form of *Henneguya* and a severe multifocal interlamellar proliferation of the gill tissue were observed in a number of samples from channel catfish. Should fish be sufficiently infected with this parasite, develop the gill lesion, and subsequently be exposed to stressors such as low dissolved oxygen or handling, the potential for mortality exists.

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