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THE HISTOPATHOLOGY OF FIN ROT DISEASE IN WINTER FLOUNDER FROM THE NEW YORK BIGHT

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Abstract: The histopathology of fin rot disease in winter flounder, *Pseudopleuronectes americanus*, from the New York Bight is described. Fin rot in winter flounder was characterized by progressive loss of portions of the anal and dorsal fins. Microscopic findings included epidermal hyperplasia accompanied by dermal fibrosis, hyperemia, and hemorrhage. Bacteria were not observed *in situ* and a pronounced leukocytic inflammatory response was not noted.

INTRODUCTION

Fin rot or tail rot is characterized by the progressive destruction of fin tissue and may be found in association with many fish diseases, notably those of bacterial etiology.[1,2] The location of affected fins varies with the species of fish and the causative agent. Bullock[1] states that tail rot is more prevalent in marine fish than in aquarium fish or salmonids. In the present study, destruction of the anal and dorsal fins was observed more frequently than destruction of the caudal fin. Studies of fin rot in marine fish have yielded a variety of marine-occurring bacteria.[5,6,8,10,12] The specific etiology of fin rot has not been determined, although bacteria have been implicated. Mahoney[6] induced fin erosion in mummichogs (*Fundulus heteroclitus*) by inoculation with species of *Aeromonas, Pseudomonas,* and *Vibrio* subsequent to abrasion of the fin surface.

Studies of disease prevalence in fish from the New York Bight have indicated that fin rot disease in winter flounder (*Pseudopleuronectes americanus*) is more prevalent in the Bight apex than adjacent coastal areas to the north and south.[13] In the apex area, 371/2632 (14.1%) of the winter flounder examined had fin rot. Only 26/1857 (1.9%) of the winter flounder examined from adjacent coastal areas had the disease (difference significant at $p < 0.01$). The ecology of the apex area has been altered substantially as a consequence of the disposal of sewage sludge, harbor dredge spoils, and industrial wastes.[7] Histopathological assessment of fin rot disease in winter flounder has been made for over a year and is the subject of this report.

MATERIALS AND METHODS

Fish were collected by otter trawl during cruises to the New York Bight (Fig. 1) conducted from March 1973 through June 1974. Diseased fish (35) with grossly dissimilar lesions were obtained from the Bight apex and Raritan and Sandy Hook Bays, N.J. (13 in winter and 22 in summer); normal fish (5) were obtained from Great Bay, N.J., a relatively "pristine" area in southern New Jersey having a similar fauna and hydrographically compatible to the "stressed" areas. Fish were measured and portions of diseased fish tissue were fixed immediately in 10% seawater-formalin. Subsequent to fixation, tissues were decalficied in RDO (DuPage Kinetic Laboratories)* for 2-4 h (or when soft

* References to trade names do not imply endorsement of commercial products by the National Marine Fisheries Service.

enough to cut satisfactorily). Tissues were processed routinely at 6 μm sections were stained with hematoxylin and eosin, Feulgen stain, periodic acid Schiff, and tissue Gram stain.

RESULTS

The gross examination of fish with advanced fin rot disease revealed extensive loss of fin rays and fin tissue (Fig. 2). Lesions of the mid-portion of the anal and dorsal fins appeared to be more prevalent than caudal fin lesions; no frequency tabulations were made, however. In some fish, destruction of the fin rays did not keep pace with that of the overlying epithelium and the denuded rays projected beyond the distal margin of the remaining fin stump. All fish examined, regardless of the time caught and severity of the disease, had lesions bordered by a whitish tissue. Fish collected in the spring, after their period of winter torpor, displayed substantial quantities of this tissue.

Microscopic examination of the fin lesions substantiated the gross observations (Fig. 3, 4 and 5). All lesions were characterized by epidermal hyperplasia and dermal fibrosis. The thickness of the hyperplastic epidermis and fibrotic dermis varied with the severity of the disease and the time of the year. Hyperemia and focal hemorrhage were common in the dermis of the fin stump. Occasionally a focal leukocytic inflammatory response was observed.

Melanophores were very prominent around blood vessels in the fibrotic dermis, especially deep within the tissue. Eosinophilic granule cells (EGC), normally seen in the epidermis adjacent to the basement membrane, appeared to be

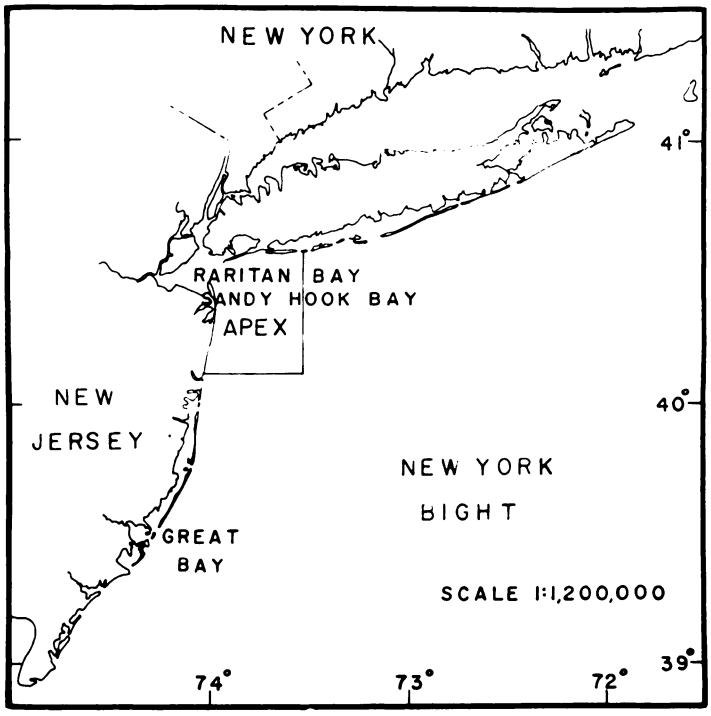

FIGURE 1. New York Bight apex and adjacent coastal areas.

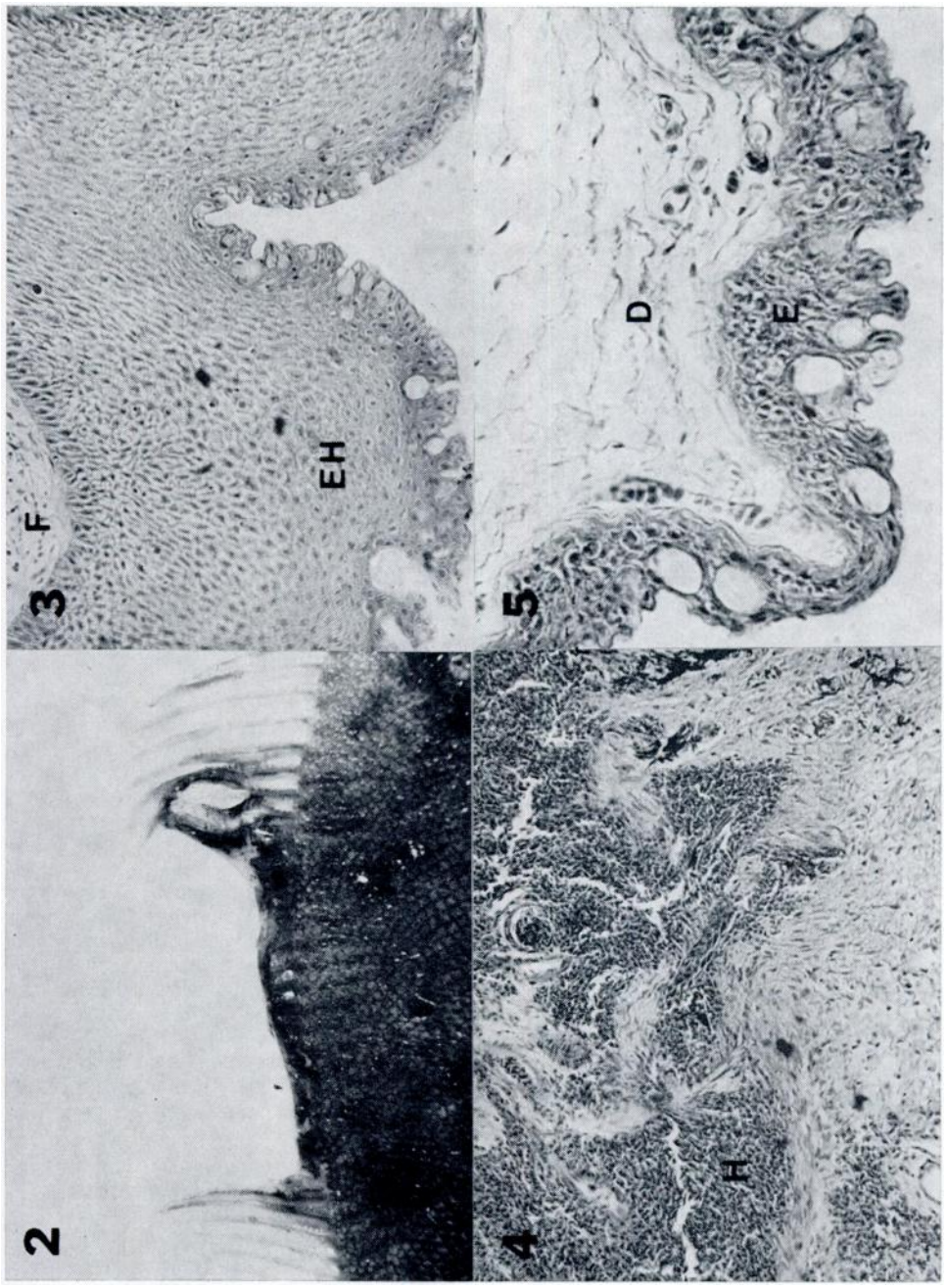

FIGURE 2. Winter flounder with fin rot of dorsal fin.
FIGURE 3. Epidermal hyperplasia, EH and fibrosis, F (X200).
FIGURE 4. Hemorrhage, H in fibrotic dermis (X200).
FIGURE 5. Normal epidermis, E and dermis, D (X200).

more abundant in diseased tissues. The cells were not restricted to the area of the basement membrane but occurred throughout the epidermis. The EGC were morphologically similar to those described by Roberts[9] in the epidermis of the plaice (*Pleuronectes platessa*). Mucus cells also appeared to be more abundant in the epidermis of fish with advanced fin rot disease. It was not possible to demonstrate the presence *in situ* of bacteria, fungi, or other parasites in any of the histologic preparations.

DISCUSSION

In contrast to the diseased winter flounder examined by Levin,[5] the winter flounder examined in this study all had primarily resolving and not acute lesions. Epithelization of the perimeter of the lesion accompanied by dermal fibrosis was a more conspicuous histologic finding than necrosis of the epidermis and was evident in fish caught during both summer and winter. Necrotic fin lesions with no evidence of healing and characteristic of the acute disease rarely were observed in winter flounder. Summer flounder, *Paralichthys dentatus,* frequently were noted with acute lesions. The pathogenesis of fin lesions in winter flounder is difficult to determine histopathologically using randomly caught fish. Laboratory studies are required to assess the role of temperature and other variables on tissue responses of migratory poikilothermic vertebrates.

The fact that bacteria were not found in any of the winter flounder fin tissues sectioned does not obviate the possibility of a bacterial etiology. Unless they are present in substantial numbers, bacteria are difficult to see in tissue sections. It is also possible that bacteria no longer were present in the healing lesions of the winter flounder examined. In the acute fin lesions of summer flounder, bacteria were readily demonstrable. The lack of a pronounced leukocytic response in both summer flounder and winter flounder suggests that the necrotic process is not primarily microbial. The inflammatory responses of fishes to microbes vary extensively, however, and some bacteria and fungi elicit no detectable cellular responses.[3] Although water temperature affects the appearance of the piscine inflammatory response,[4] fish used in this study were caught in both winter and summer.

The severely altered environment in which the diseased winter flounder were collected, in itself, may be adequate to initiate a necrotic process in fin tissue. The distal fin tissue is thin and could become necrotic from prolonged contact with a chemical irritant. Alternate cycles of aseptic necrosis and resolution may be all that are required to produce the lesions that were observed. A proliferative inflammatory response induced by chemical irritants and characterized by epidermal hyperplasia and fibrosis occurs in mammals and also could occur in fish.[11] Ischemic necrosis with subsequent dermal fibrosis provides an alternative hypothesis to one necessitating direct contact with an external irritant. Distal fin tissues could become ischemic from a variety of causes; the fins are terminal in terms of their blood supply and, therefore, may be more vulnerable than other tissues.

The winter flounder is an important recreational and commercial fish in the New York Bight. It is the only pleuronectid in the Bight which has a high prevalence of fin rot disease. The other flounders of economic significance are the summer flounder and yellowtail flounder (*Limanda ferruginea*). Both summer and yellowtail flounders are seasonal migrants; more summer flounders have fin rot than yellowtail flounder. The most abundant resident flounder in the Bight is the windowpane (*Scophthalmus aquosus*). Windowpane flounder rarely are found with fin rot. It is of interest to speculate that their abundant slime is an effective barrier to the biotic or abiotic agents which initiate the disease.

Bacteriologic and immunologic studies are being conducted to elucidate the possible role of bacteria in fin rot disease. Bacteria readily can be isolated from superficial fin lesions (*Pseudomonas, Achromobacterium, Flavobacterium,*

Vibrio), but not from viscera (Murchelano, unpublished). Bacterial agglutinin titers in sera from fish with fin rot were not consistently high against organisms isolated from diseased fin tissue (Robohm, unpublished). Future bacteriologic studies must include the use of more nutritionally appropriate initial culture media and extensive culture of acute lesions.

One only can speculate on the eventual course of fin rot disease in wild fish. Presently, it does not appear that fin rot in winter flounder is a rapidly progressive disease which ultimately causes death. Behavioral changes consequent to extensive loss of fin tissue may render individual fish more vulnerable to predation or affect their ability to capture prey. There are many facets of fin rot disease of winter flounder from the New York Bight which warrant further study.

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