HISTOPATHOLOGY OF AN ACUTE FIN LESION IN THE SUMMER FLOUNDER, *Paralichthys dentatus*, AND SOME SPECULATIONS ON THE ETIOLOGY OF FIN ROT DISEASE IN THE NEW YORK BIGHT

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Abstract: The histopathology of acute fin rot disease in summer flounder, *Paralichthys dentatus*, from the New York Bight is described. Grossly, caudal and dorsal fin lesions appeared ragged or frayed with no evidence of resolution. Microscopically, there was epidermal and dermal necrosis, congestion, edema, focal and diffuse hemorrhage, and Zenkers necrosis of underlying muscle. Gram-negative bacteria were present in the fin tissues as well as in heart muscle and liver parenchyma. The inflammatory response consisted mostly of macrophages. The significance of the acute disease in summer flounder is discussed in relation to the etiology of fin rot disease in winter flounder from the Bight.

INTRODUCTION

Fin rot, or fin erosion, a disease characterized by the progressive loss of fin tissue, recently has been noted in trawl surveys of demersal fishes from a number of geographically disparate marine environments.[1,2,3,5,6] In many instances, a high prevalence of fin rot disease in the catch has been associated with a deteriorated environment. An intensive study of fin rot in winter flounder, *Pseudopleuronectes americanus*, from the severely degraded New York Bight has established a significantly higher disease prevalence in the Bight apex than in adjacent coastal areas.[6] A histopathologic study of fin tissues from Bight winter flounder revealed that the lesions were more typically resolving than acute.[4] This paper presents the results of a histopathologic study of acute fin lesions from two summer flounder, *Paralichthys dentatus*, collected from the New York Bight and held in a laboratory aquarium.

MATERIALS AND METHODS

The summer flounder were obtained from Raritan Bay, New Jersey, on 12 November, 1974. Ambient water temperature was 10 C. Two summer flounder measuring 14 cm and 16 cm and with early signs of fin erosion on their anal, caudal, and dorsal fins were placed in a 302 L cylindrical, polypropylene tank for transport to the laboratory. The tank was continuously flushed with seawater provided by the deck pump of the vessel. At the laboratory, the fish were placed in an aquarium constructed of fiberglass-lined plywood measuring 1.2 x 1.2 x 1.2 m. The aquarium contained several inches of fine sand and was provided with water from a seawater well. The temperature of the seawater ranged from 12 to 14 C. After the fish were in the aquarium for 22 days, it was apparent that the disease had progressed. When they were placed in the aquarium, fin damage consisted only of distal erosion; when they were killed the erosion on all

fins had progressed medially and on the caudal fin also had affected the peduncle. The abdomen was opened from the vent to the gill arch and the fish were placed in 10% seawater formalin for later histopathologic examination. Pieces of fin and visceral tissues were excised, routinely dehydrated, embedded in paraffin, decalcified in RDO, sectioned at 6 μm, and stained with hematoxylin and eosin, Giemsa, and tissue Gram stain.

RESULTS

Early caudal and dorsal fin lesions (Figs. 1, 2) were characterized grossly by loss of epidermal, dermal, and fin ray tissues and a generally ragged or frayed appearance. As the disease progressed, the loss of epidermal and dermal fin tissues became more pronounced. This was particularly apparent on the caudal fin where these tissues were absent totally from the fin itself and were stripped substantially from the adjacent peduncle. Microscopic examination of sections of peduncle from this region (Fig. 3) showed the absence of epithelium and basal lamina. Edematous and partially necrotic dermal connective tissue comprised the perimeter of the peduncle. Many blood vessels in the dermis were congested and in some areas focal hemorrhage was noted. Large aggregates of slender, rod-shaped bacteria (Fig. 4) were present on the surface of the irregular and necrotic dermis and discrete colonies of pleomorphic, coccobacilli (Fig. 5) were present within the dermis. The bacteria stained Gram negative. Macrophages were the most numerous of the inflammatory cells observed, although lymphocytes and granulocytes also were present. Some of the underlying lateral muscles appeared normal whereas others contained fibers which were atrophic or necrotic (Fig. 6). No histopathologic changes were observed in sections of gill, heart, intestine, kidney, liver, or spleen; however, small aggregates of Gram negative bacilli were noted between muscle fibers in the ventricle of the heart and also in liver sinusoids.

DISCUSSION

In contrast to other histopathologic studies of fin rot disease in pleuronectids —southern California coastal waters,[3] the New York Bight,[4] and Puget Sound, Washington (Wellings, unpublished)—in this study bacteria were found in necrotic fin tissues of diseased fish. The histopathologic differences between the fin lesions of the winter flounder from the New York Bight examined earlier[4] and the summer flounder examined herein were substantial. The lesions of the summer flounder were acute or "nonproliferative".[6] They contained abundant bacteria and the histopathologic changes observed probably were consequences of their presence. Although no bacteria were observed in the winter flounder fin lesions, the lesions were characterized by

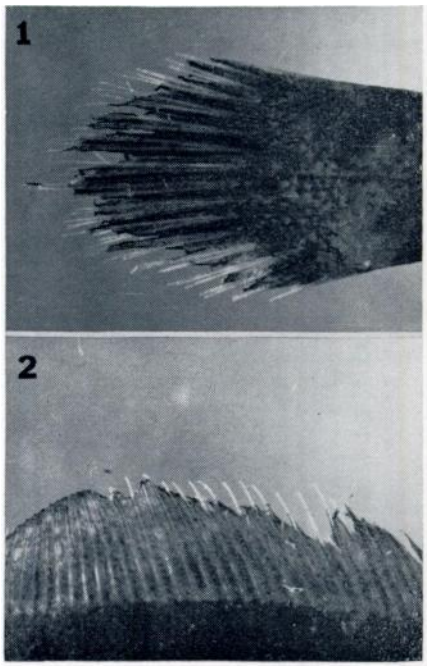

FIGURE 1. Erosion of caudal fin of summer flounder.

FIGURE 2. Erosion of dorsal fin of summer flounder.

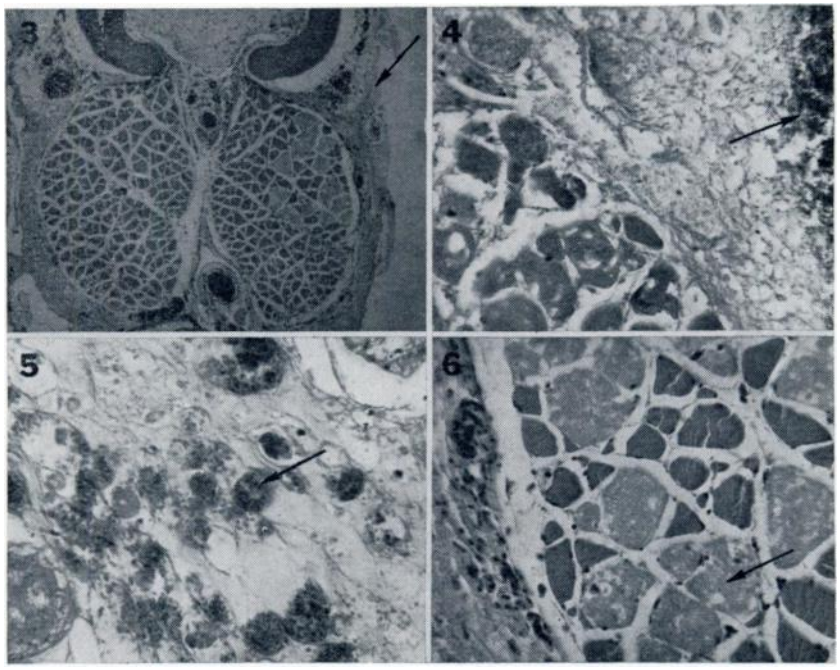

FIGURE 3. Section from caudal peduncle of summer flounder with extensive erosion of fin and caudal peduncle. Note absence of epithelium and basal lamina (arrow), congestion, hemorrhage and necrotic muscle fibers. H & E stain; X100.

FIGURE 4. Aggregates of elongate bacilli (arrow) present on necrotic dermis of peduncle shown in Fig. 3. H & E stain; X450.

FIGURE 5. Colonies of coccobacilli (arrow) in necrotic dermis of peduncle shown in Fig 3. H & E stain; X1000.

FIGURE 6. Higher magnification of muscle bundle shown in Fig. 3. Note necrotic muscle fibers (arrow). H & E stain; X450.

fibrosis and epidermal hyperplasia, suggesting a chronic or "proliferative"[6] response. The question arises as to why winter flounder are found with chronic lesions and summer flounder are found with acute lesions.

The migratory patterns of winter and summer flounder in the New York Bight are temporally reversed. Winter flounder migrate seaward in the summer and summer flounder migrate shoreward. Summer flounder, therefore, are abundant in the ecologically degraded survey area during the period of maximum water temperature. Since water temperatures are elevated, it also is likely that bacterial populations are at their maximum density. Recently acquired data indicate that the monthly prevalence of fin rot disease in summer flounder increases with their duration of residence in the impacted area (Murchelano and Ziskowski, unpublished). The lesions of the two summer flounder examined in this report probably were acquired during their onshore residence. Placing the flounder in a laboratory aquarium may have accelerated the disease described but did not initiate it. Chronic fin lesions are not seen in summer flounder because at the end of

the summer the fish migrate to deeper, offshore waters. Unfortunately, trawl surveys have been limited to nearshore areas.

Fin rot disease studies in the New York Bight have focused on winter flounder because the prevalence of the disease is highest and the lesions are the most conspicuous in this species. However, because of the timing of their shoreward migration, summer flounder may be more useful in establishing the etiology of fin rot disease. Bacteriologic and histopathologic studies of summer flounder from the New York Bight with fin rot should be informative.

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