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MORTALITY IN CAPTIVE ATLANTIC COD, *Gadus morhua*, ASSOCIATED WITH FIN ROT DISEASE

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Abstract: A total of 320 of 621 (52%) captive Atlantic cod, *Gadus morhua*, died from fin rot over an 8-year period; mortality reached high proportions in 1979 when 110 of 168 (66%) succumbed. Lesions were confined to the fins, skin and dermal musculature and were observed 3-10 days after the fish were placed in laboratory aquaria. Erosion of the fins and caudal peduncle, accompanied by petechiae and ulceration also were apparent in the trunk region. Most deaths occurred within 2 months after capture. Infection was associated with depressed hematocrit, hemoglobin and total plasma protein and an increase of circulating immature erythrocytes and neutrophils. Mortality was probably due to physiological stress resulting from excessive blood loss. Three genera of bacteria, mostly *Pseudomonas*, but also *Aeromonas* and *Vibrio*, were isolated from fin rot tissues and possibly are the causative agents of the disease.

INTRODUCTION

Fin or tail rot is a disease of both fresh water and marine fish.[4,5,8,9,10,11,12,13,15,17,19] The causative agent of the disease has not been clearly established. Initially, it appears as a slight discoloration of the outer edges of the fins. As the infection progresses, fins lose their epidermis and become frayed, exposing the fin rays. Eventually, the fin rays drop off and only the stump of the fin remains. Fin rot has been reported previously in marine fish from several geographical areas.[4,5,7,13,14,15,17,19]

During studies on parasites of marine fish in the northwestern Atlantic Ocean, Atlantic cod *(Gadus morhua)* were captured by research vessels and returned alive to the laboratory. Some of these fish succumbed to fin rot disease. This study reports on mortality in Atlantic cod associated with fin rot disease, histopathologic observations of the lesions and identification of the presumed aetiological agents.

MATERIALS AND METHODS

Atlantic cod (36-92 cm) were captured on the continental shelf off Newfoundland by Canadian research vessels using an otter trawl. Bottom (280-350 m) temperatures at which fish were taken varied from 2.5 to 4.1 C. The fish were retained for 3 to 10 days on board ship in circular tanks (1.4 m diameter × 0.9 m deep) provided with running sea water. At the laboratory, they were held subsequently in running sea water (0-5 C) in tanks 2.8 wide × 2.8 long × 1.2 m deep and fed capelin *(Mallotus villosus)* about thrice weekly. Blood (about 1 ml) was removed from 15 fish prior to death, which varied from 3-20 days after they were placed in laboratory aquaria, and assessed for changes in hematocrit, hemoglobin, total plasma protein and numbers of leucocytes by conventional laboratory procedures. Tissues, which included heart, liver, spleen, kidney, intestine, gill and skin from 6 cod showing signs of fin rot were fixed in Bouin's fluid and sections prepared by conventional

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histologic methods. Tissue sections were stained by various stains including hematoxylin and eosin, Mallory's triple stain and Giemsa colophonium.

Procedures for isolation of bacteria from tissues of 6 cod required sterilization of the external surface of the fin with a hot scalpel, insertion of a sterile probe into the underlying tissue and streaking it on trypicase soya agar (Difco). Plates were incubated at 18 C and taxonomic procedures for the bacteria isolated followed those of Gilespie *et al.*[2]

RESULTS

Mortality in captured Atlantic cod, associated with fin rot, is shown in Table 1. A total of 320 of 621 (52%) cod succumbed over an 8-year period. The number of deaths each year was variable but it reached alarming proportions in 1979 when 110 of 168 (66%) died. Early signs of fin rot were noted when some of the cod were placed in laboratory aquaria. Frayed and discolored extremities of the fins, especially the caudal fin, and the presence of petechiae on the lateral wall of the body characterized this initial phase. As the infection spread to the distal areas of the fins, erosion of the epidermis became apparent. The petechiae on the body wall enlarged as papular elevations and became ulcerated eventually with a pale central core and hyperemic margins (Fig. 1). In fish which succumbed, the skin and musculature were eroded, leaving the fin rays exposed. The terminal extremity of the caudal peduncle detached occasionally (Fig. 2) and hemorrhage was apparent. The ulcers on the body were enlarged conspicuously and outlined (Fig. 1).

The earliest deaths among cod in 1979 occurred after 5 days and by 12 days, 22 fish died. Deaths continued daily, 40 in the first month and 78 of the 82 fish after 2 months. The water temperature during this period varied from 3 C in December, 1979 to 0 C by the end of January, 1980. Fin rot infections were observed among cod taken from areas adjacent to Newfoundland and included the Gulf of St. Lawrence, Grand Bank, Flemish Cap and the Labrador Sea shortly after they were placed in laboratory aquaria.

There was alteration of blood parameters in cod with terminal signs of fin rot (Table 2). Hematocrit, hemoglobin and total plasma protein levels were lower in diseased fish than in apparently healthy animals. There were many immature erythrocytes in cardiac blood smears of moribund fish and while there was no apparent change in total white cells counts, an approximate decrease of

TABLE 1. Mortality from fin rot disease in Atlantic cod held in laboratory aquaria.

Year	No. died/total no.
1973	19/27
1974	17/53
1975	16/47
1976	41/86
1977	27/61
1978	40/84
1979	110/168
1980	50/95
1973-1980	320/621 (52%)

TABLE 2. Blood parameters in 15 Atlantic cod which died from fin-rot disease.

	%RBC	Hb (gm%)	gm Plasma protein	WBC/100 RBC	
				L-M[1]	N[2]
Cod with fin rot	18.3 ± 8.76	5.0 ± 2.16	15.0 ± 7.29	9.4 ± 6.7	15.6 ± 6.7
Control cod	33.4 ± 5.27	5.95 ± 1.15	57.4 ± 5.86	17.9 ± 3.4	3.8 ± 3.1

[1]lymphocyte-monocyte
[2]neutrophil

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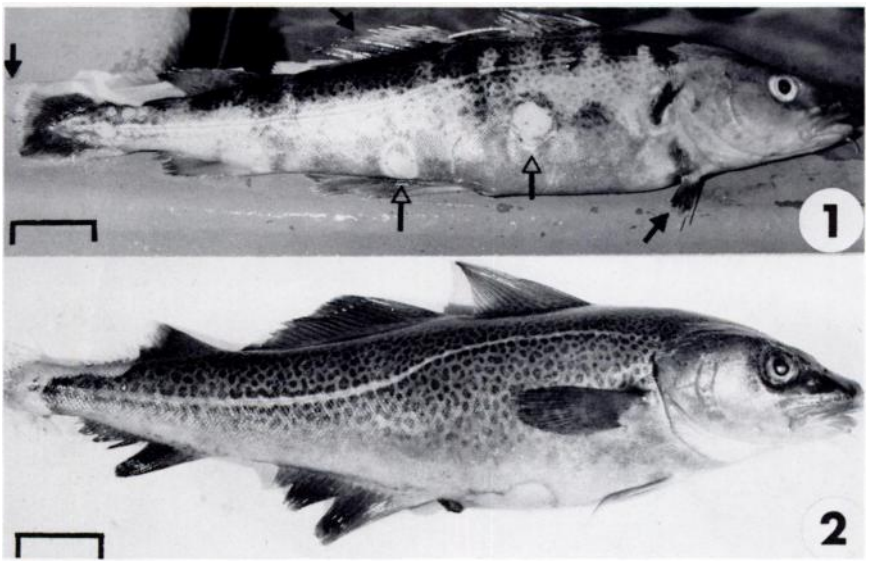

FIGURE 1. Atlantic cod showing signs of fin rot. Note fin erosion (solid arrow heads) and ulcers (hollow arrow heads).

FIGURE 2. Cod with eroded caudal peduncle.

Scale bars. Figure 1 = 4 cm; Fig. 2 = 3 cm.

50% in the number of monocytes and lymphocytes was accompanied by an increase of neutrophils.

Histopathologic findings were compatible with gross lesions. Examination of histologic sections of skin revealed sloughing of the outer epidermal layers and the presence of rod-shaped bacteria. Dermal capillaries were dilated and congested and characterized the early skin infections. Petechiae and papular elevations contained a serosanguineous fluid. Hemorrhages were also apparent in the stratum spongiosum of the dermis (Fig. 3). In small ulcers, epidermal necrosis was observed and as the underlying musculature became involved, edema was evident (Fig. 4). In some instances, hyperplasia and excessive tissue were also evident in the statum compactum of the dermis. As the ulcers enlarged, the epidermis was eroded completely and subcutaneous muscle necrosis occurred (Fig. 5). There was some evidence of an inflammatory response in the vicinity of the ulcer and it was characterized by hemorrhage, inflammatory cells (mainly monocytic-lymphocytic type) and necrosis of the muscle fibers (Fig. 6). Bacteria also were observed in the musculature. No histopathologic changes were observed in sections of heart, liver, spleen, kidney, intestine and gill and bacteria were not seen in these tissues.

The bacteria isolated from the fin lesions were Gram negative, rod-shaped and motile. *Pseudomonas* accounted for 72% of the isolates and *Vibrio* and *Aeromonas* for 28%.

DISCUSSION

Observations in the present study suggest that fin rot disease of cod from the Newfoundland-Labrador area is

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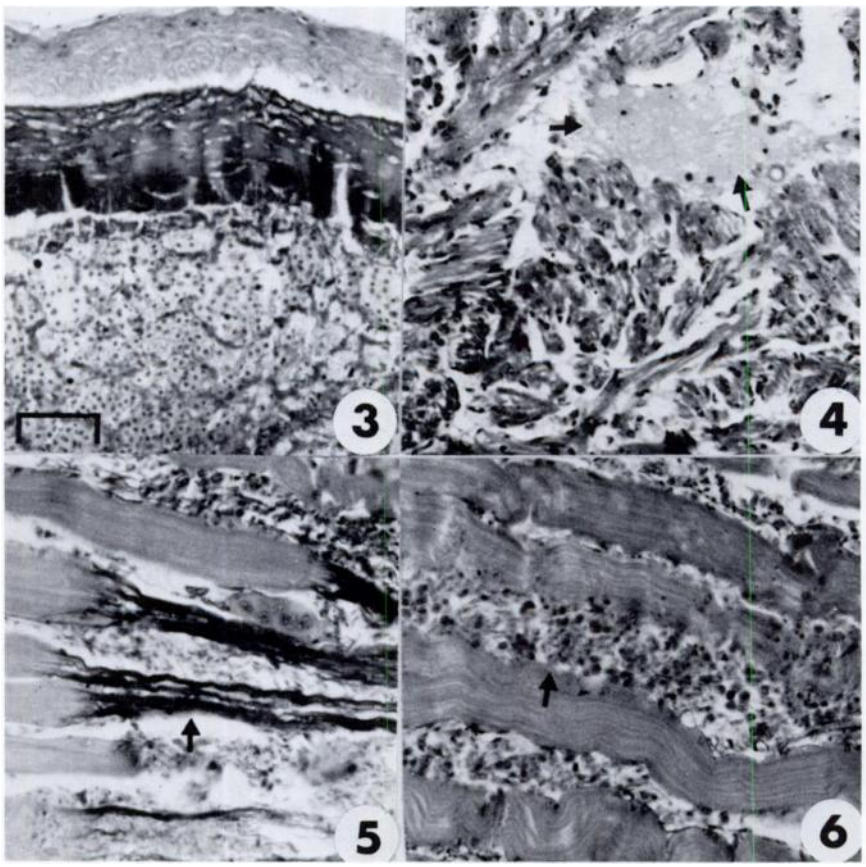

FIGURE 3. Dermal hemorrhage adjacent to an ulcer.

FIGURE 4. Intramuscular edema (arrows) beneath ulcer.

FIGURE 5. Skeletal muscle necrosis (arrow) at the base of a dermal ulcer.

FIGURE 6. Inflammatory response (arrow) in subcutaneous muscle beneath ulcer.

Scale bars. Figs. 3-6 = 50 μm.

similar to that reported by Oppenheimer[13] and by Jensen and Larsen.[3,4] Fin rot, moreover, is not restricted to cod, but also is found in many other species of fish including 7 spotted *(Anarhichas minor)* and 11 striped wolffish *(A. lupus)*, 5 American plaice *(Hippoglossoides platessoides)*, 3 yellowtail flounder *(Limanda ferruginea)* and 2 Greenland halibut *(Reinhardtius hippoglossoides)* after they were placed in laboratory aquaria in late winter, 1978 (Khan, unpubl.). Mahoney *et al.*[9] reported that 22 species of marine and euryhaline fish from the New York Bight were infected with fin rot disease and it appears that many other species of teleosts are susceptible.[10,14,15,17,19]

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In the present study, bacteria, mainly *Pseudomonas* sp., were associated with fin rot disease. Several genera of bacteria are associated with sea water[6] and the skin of fish[2,16] and some authors report species of *Aeromonas,*[13,17] *Vibrio,*[5] or *Aeromonas, Vibrio* and *Pseudomonas* as the disease agent.[9] Although Danish workers noted bacteria *(Vibrio anguillarum)*[6,7] and viruses[5] in fin rot lesions, neither were implicated as the cause of the disease. Moreover, Wellings *et al.,*[19] did not find bacteria, fungi or parasites in eroded fin tissue of two pleuronectid fish. Murchelano[11] and Murchelano and Ziskowski[12] concluded from their studies in the New York Bight that stress induced by pollutants might be the underlying cause of the disease. Overstreet and Howse,[14] Mearns and Sherwood[10] and a recent review by Sindermann[18] suggest that fin rot tends to be associated with degraded habitats. Couch[1] induced fin erosion in spot *(Leiostomus xanthurus)* following exposure to polychlorinated biphenyls. We, however, have observed that fin rot in cod occurs only when the fish are brought into the laboratory in winter months at low temperatures (0 to 4 C). There is some evidence that the immune response of cod is considerably reduced or absent at these temperatures under laboratory conditions (Khan, unpubl.). During the past three years over a thousand cod have been collected in traps in the summer (9 to 14 C) and held in captivity in the laboratory. Fin rot has been observed rarely in these fish. Winter flounder, on the other hand, show signs of fin rot mainly during the summer months and less often in winter in the Newfoundland area (Fletcher, unpubl.). Moreover, the fish are collected from areas that are apparently free of pollution. In light of the above reports and our observations, a field study on fin rot is a prerequisite towards understanding the factors associated with its primary cause, occurrence and distribution.

The bacteria, noted in fin rot lesions in cod, probably gained entry into the tissues through mechanical injury caused by the otter trawl as well as by the spines of redfish *(Sebastes marinus)* and thorny skate *(Raja radiata)* in the cod end. These abrasions, which result in removal of scales and protective mucous and in epidermal performations, permit invasion by opportunistic bacteria. The abrasions caused by redfish are so extensive that cod caught in trawls are not tagged where redfish are in excess of 20% of the catch (Lear, unpubl.). Since the clinical signs of fin rot develop rapidly and kill the fish (mortality is probably due to physiologic stress resulting from excessive blood loss) in a short time, it is not surprising that diseased fish or fish with healed lesions were not observed among the numerous cod examined in a tagging program (Lear, unpubl.). Fin rot disease, consequently, could be one of the major factors responsible for low tag returns (~10%, Lear, unpubl.) if mortality observed in the laboratory is similar to that which occurs in the field.

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