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INTRODUCTION

A disease syndrome known as "fin rot" or fin erosion occurs in inshore fishes from Ventura south through northern Orange County. The syndrome has apparently been occurring in some localities since at least the mid-1950's (Young 1964). The disease is most prevalent in the Dover sole (Microstomus pacificus) and the white croaker (Genyonemus lineatus), although signs of fin erosion have now been documented in 36 species of local inshore fishes.

The cause or causes of the disease syndrome are presently unknown but hypotheses include (1) marine microbial infection, (2) microbial infection from pathogens of wastewater origin, (3) parasites, and (4) exposure to toxic materials in the sediments and water column. These possible causes may indirectly stem from more subtle responses of fish to man's activities, e.g., attraction and crowding of fish, elevated temperatures and stimulation of toxic and/or microbial agents via high organic content (or BOD) of localized waters or sediments. Any combination of these conditions is sufficient to cause diseases in marine fish. Once a disease appears in a population, its incidence becomes a function of migration, recruitment, predation pressure, and increased natural mortality.

Determination of the physical, chemical, and biological conditions associated with the disease syndrome is underway at the Southern California Coastal Water Research Project. We were engaged to further describe the disease histologically and suggest possible causes. To accomplish this, we examined fresh specimens from diseased and control areas for (1) associated microorganisms and (2) signs of tissue degeneration known to be caused by specific toxic agents (such as metals), microbial activity, and/or parasite activity. Dr. Juhee Kim, Department of Microbiology, California State University at Long Beach, conducted microbial assays. These will be described in another technical memorandum.

METHODS

A fish collecting trip was made aboard the Los Angeles County Sanitation Districts vessel "Sea-S-Dee" on 16 May 1972. The stations trawled are shown in Figure 1.

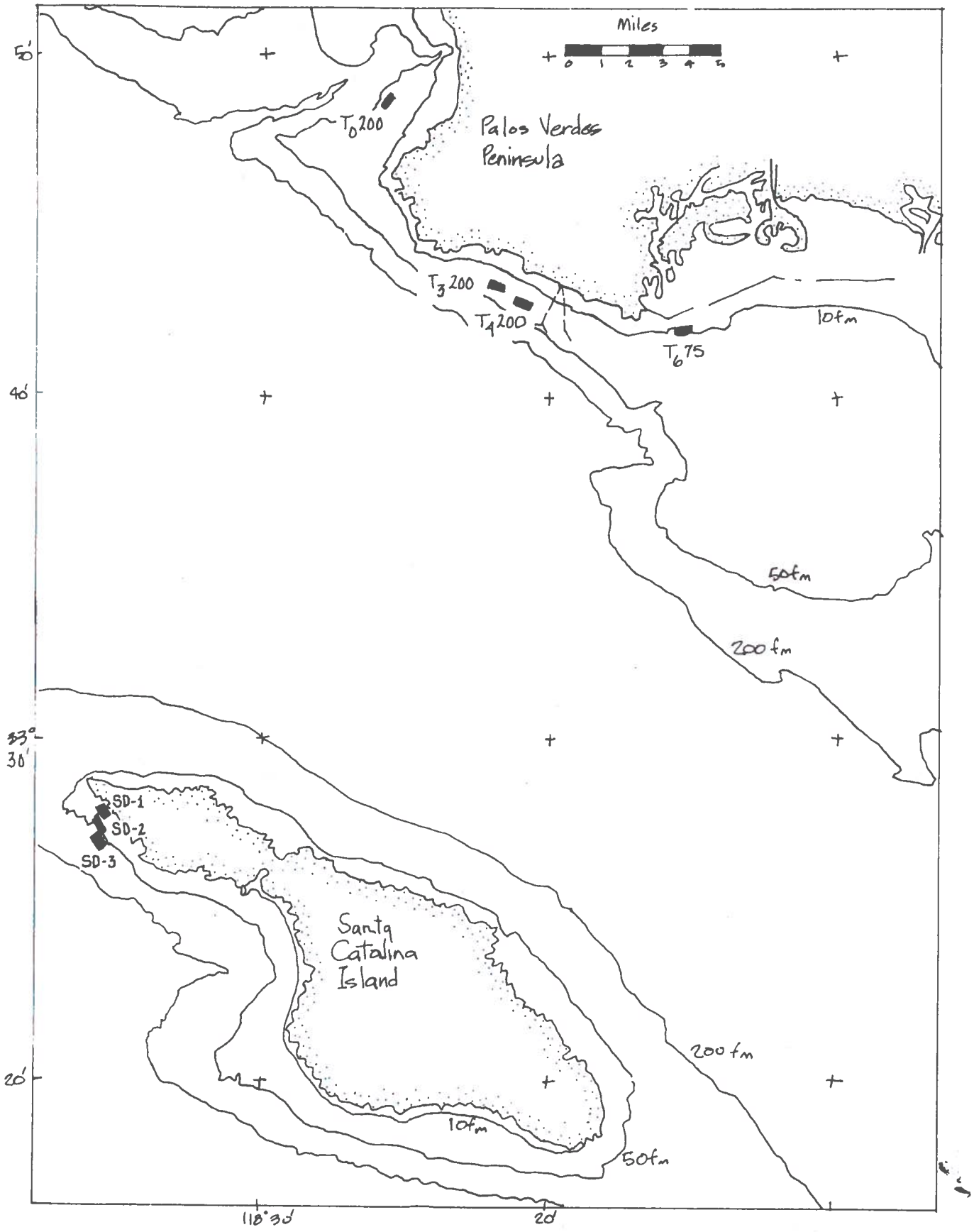

Figure 1. Stations from which diseased and apparently healthy fishes were collected for histopathological examination.

Selected specimens were photographed with High-Speed Ektachrome and were placed into 10 percent neutral-buffered formalin for subsequent histopathological examination. Several blood films were made of the kidney, liver, and spleen and the circulating blood. These were fixed in absolute methanol for subsequent staining and examination.

After preliminary examination of the samples obtained on 16 May 1972, it was apparent that samples of fish from a relatively unaffected area were necessary for comparison. These were obtained on 14 June 1972 from stations near Santa Catalina Island (Figure 1).

GROSS OBSERVATIONS

Twenty-three Palos Verdes specimens, representing seven species of fish were examined (Table 1). Of the four species of flatfish examined on 16 May 1972, the Dover sole was the only species that had fin lesions. The lesions appeared to begin, and were most common, on the middle portion of the dorsal and anal fins. The caudal fins were affected only in cases where the dorsal and anal fins were almost entirely missing. Pectoral and pelvic fins were only rarely affected.

The early changes appear to involve fraying of the fins, followed by denuding of the tips of the fin rays. The bony rays then begin to undergo degeneration, and the fin thickens due to fibroplasia in the skin between and around the fin rays. Simultaneously, melanophores appear to migrate toward the site of injury and form a black line along the edge of the eroding fins. However, these body defenses have little effect upon the progress of the lesion: After the initial injury, the cellular degeneration continues until the entire fin is gone.

Cow rockfish (Sebastes levis), vermilion rockfish (Sebastes miniatus), and white croaker also had fin lesions. In these species, the lesions were most commonly observed on the caudal and pectoral fins; however, lesions were also relatively common on the pelvic and anal fins. The fact that the lesions on the croaker were bloody, unlike those on the rockfish or Dover sole, may indicate a different pathogenesis.

The gross differences between the Dover sole taken off Catalina and those caught off Palos Verdes were striking. The Catalina Island fish were olive brown on the eyed side and very white on the blind side, while the fish collected off Palos Verdes were black on the eyed side and yellowish-white stippled with black on the down side. The fin rays of the eleven Catalina Island Dover sole examined (Table 2) tapered down to fine edges without fraying, but the Palos Verdes fish all had some variation of frayed fins, denuded fin ray tips, thickened fins, or markedly eroded fin edges with a band of melanosis.

HISTOPATHOLOGY

The lesions of significance in the flatfishes were restricted to the fins. These changes were manifested as thinning or loss of epidermis on the tips of the fins with resultant fibroplasia in the dermis and aggregation of melanophores along the edges of the denuded areas. Lymphocytic infiltration was sparse or absent. Application of various histological stains failed to demonstrate bacterial, fungal, or protozoal organisms associated with the fin lesions.

No differences were noted between the viscera of affected and non-affected fish. Both groups had large quantities of lipid in their hepatocytes and moderate numbers of encysted metacercaria in their livers and intestinal walls.

The gills of some fish from T_3200 and T_4200 (Figure 1) had mildly hyperplastic respiratory epithelium. However, it was not severe enough to have been causing difficult respiration. All of the gills examined were telangiectatic, probably due to the rapid pressure changes at the time of collection.

There were no significant abnormalities noted in any of the blood films or organ imprints. This suggests that the fin erosion in the Dover sole and the tail rot in the white croaker are localized rather than systemic disease symptoms.

CONCLUSIONS

The noninflammatory nature of the lesions and the absence of any demonstrable organism tend to rule against this being an infectious process; however, this conclusion should be verified. The location of the most severe lesions at the midportion of the dorsal and anal fins suggests that their development might be associated with movement of the fins and their contact with the bottom sediment.

The bottom sediment could cause the initial damage by extreme pH, high hydrogen sulfide content, or high trace metal content; any of these conditions could cause coagulation of the protective mucus on the skin and subsequent cellular necrosis of the fins. The undulation of the middle portion of the dorsal and anal fins during swimming and the use of these fins for burrowing into the sediment could cause this portion of the fish's skin to lose mucus rapidly and thus predispose the underlying epidermal cells to the chemical irritants in the sediment.

The skin of Dover sole is histologically different from that of the other flatfishes examined. The epidermis contains large aggregations of mucus cells, which coalese to form mucous cysts that discharge their contents onto the surface of the skin. However, no difference in numbers or location of these cysts on affected and nonaffected fish was noted.

Table 1

FISHES FROM PALOS VERDES EXAMINED FOR GROSS AND HISTOLOGICAL SIGNS OF FIN EROSION, 16 MAY 1972

Specimen No.	Species	Sta. No.	Gross Lesions
1	Bigmouth sole (*Hippoglossina stomata*)	$T_0$200	Metacercaria in stomach wall and anomaly of body wall
2	Dover sole	$T_0$200	None
3	Dover sole	$T_0$200	None
4	Dover sole	$T_0$200	None
5	Dover sole	$T_0$200	Fin erosion in posterior two-thirds of dorsal and anal fins
6	Curlfin sole (*Pleuronichthys decurrens*)	$T_0$200	None
7	English sole (*Parophrys vetulus*)	$T_0$200	None
8	Cow rockfish	$T_0$200	Fin erosion on anterior part of pectorals
9	Dover sole	$T_3$200	Marked erosion on dorsal and ventral fins; gills dark
10	Dover sole	$T_3$200	Moderate erosion of dorsal, anal, and caudal fins; gills dark
11	Dover sole	$T_3$200	Moderate erosion of dorsal, anal, and caudal fins
12	Dover sole	$T_3$200	Moderate erosion of dorsal, anal, and caudal fins
13	Dover sole	$T_3$200	Mild erosion of dorsal and anal fins; gills "burned" at tips
14	Dover sole	$T_3$200	Marked erosion of dorsal, anal and caudal fins
15	Dover sole	$T_4$200	Mild erosion of dorsal, anal, and caudal fins
16	Dover sole	$T_4$200	Moderate erosion of dorsal, anal, and caudal fins
17	Vermilion rockfish	$T_4$200	Moderate erosion of pectoral, pelvic and anal fins
18A	White croaker	$T_6$75	Tumor on mandible; moderate erosion of caudal fin (bloody)
18B	White croaker	$T_6$75	None

Table 1 (Cont.)

Specimen No.	Species	Sta. No.	Gross Lesions
19	Dover sole	$T_6$75	Depigmentation of eyed side
20	Dover sole	$T_6$75	Depigmentation of eyed side
21	Dover sole	$T_6$75	Tumor on caudal peduncle
22	Dover sole	$T_6$75	Tumor on caudal peduncle and caudal fin

Table 2

FISHES FROM SANTA CATALINA ISLAND EXAMINED FOR GROSS AND HISTOLOGICAL SIGNS OF FIN EROSION, 14 JUNE 1972

Specimen No.	Species	Sta. No.	Size (S.L., mm)	Gross Lesions
1	Dover sole	SD-3	164	None
2	Dover sole	SD-3	142	None
3	Dover sole	SD-3	140	None
4	Dover sole	SD-3	157	None
5	Dover sole	SD-2	139	None
6	Dover sole	SD-2	131	None
7	Dover sole	SD-3	146	None
8	Dover sole	SD-3	131	None
9	Dover sole	SD-3	132	None
10	Dover sole	SD-3	156	None
11	Dover sole	SD-3	152	None
12	Bigmouth sole	SD-1	168	None

The lesions on the ventral fins of the rockfish could develop in a manner similar to those on the Dover sole--through contact with the bottom sediment. The croakers, however, had bloody lesions that were common on their pectoral fins and the dorsal part of their caudal fins, which do not come in contact with the sediment. Although histological stains revealed no bacteria in these lesions, they look more like infectious lesions than do the erosions in the Dover sole and rockfish.

SUGGESTIONS FOR FUTURE RESEARCH

An attempt should be made to create fin lesions in the laboratory under controlled conditions. In addition, an attempt should be made to follow the development of the lesions in fish held in submerged cages in the area in which the fin erosion disease is most prevalent. The following experimental design is suggested.

A. Obtain unaffected fish from the Catalina Island area. These fish must be decompressed with care so as to keep extraneous variables to a minimum. The fish should be transported in water containing at least 1 ppm Furanace or Acriflavin to preclude the occurrence of bacterial disease.

B. The laboratory fish-holding containers should be made of glass, have at least a 25-gallon capacity and be supplied with running, filtered seawater. The TEST tanks should have at least a 3-inch layer of bottom sediment from the areas of greatest incidence of the fin erosion syndrome. The CONTROL tanks should have at least a 3-inch layer of bottom sediment from the areas of 0.00 percent incidence of the syndrome. The best site for collection of the control sediment would be the Catalina Island stations. The tanks should be stocked with five to ten fish 140 to 160 mm or smaller (too heavy stocking will lead to nipping, etc.). Each situation should be in duplicate.

C. The tank area should be in near darkness to simulate the bottom lighting. A photographic darkroom lighting system may be used; a flashlight could be used to view the fish. The lights must be turned off at night.

D. Both test and control fish should be fed the food they normally eat in the wild. The best diet would be polychaetes that have been held in the laboratory for a few weeks to be cleansed of extraneous material that might influence the test results.

E. The fish should be observed daily for development of fin erosion. No fish should be sampled in the first test period: The time sequence of the disease must be determined in the initial tests so as to be able to have sufficient numbers of fish available for histopathological examination.

F. Fish that die during the course of the observation period should be examined bacteriologically and histopathologically for the cause of death.

G. The first trial should be terminated at the time that 50 percent or more of the TEST fish exhibit fin erosion.

H. The second trial should be designed to determine the sequence of events occurring during the development of the fin erosion syndrome. It should be set up in the same manner as the first trial but have programmed into it the sampling of fish at regular intervals.

I. The "open water" trial should be done in live-cars (duplicate) resting on the bottom of the area of highest incidence and in live-cars suspended at least 5 feet above the bottom in the same area. Each live-car should contain fish obtained from the same unaffected area (Catalina Island) as the laboratory fish were captured.

J. Samples and observations should be made at biweekly intervals and they should be conducted in the same way as the laboratory tests.

K. The "open water" trial should be duplicated in a "clean" area (e.g., T_0200 or Catalina Island).

The tail rot syndrome in white croaker could be investigated in following manner.

A. Fish from a known affected area and a known unaffected area should be transported with due care into the laboratory. One tank should contain members of both groups. One tank each should contain several members of each group separately.

B. The idea here is to stress the fish sufficiently to bring on the syndrome in the fish from the affected area and see if it can be transmitted to fish from the clean area.

C. The fish should be maintained in an open water system (flow-through) and be fed the food they normally eat in the wild.

D. Samples should not be taken during the first trial.

E. The trial should be repeated for verification and sequential sampling to follow the histopathological and microbiological changes that occur.