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STREPTOCOCCAL INFECTIONS OF FISHES

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INTRODUCTION

Streptococcosis is a septicemic disease that affects both freshwater and saltwater fishes; it was first reported in rainbow trout, *Salmo gairdneri* (Hoshino et al. 1958). Additional outbreaks have since been described in cultured golden shiners (*Notemigonus crysoleucas*), yellowtails (*Seriola quinqueradiata*), and eels (*Anguilla japonicus*), and in a variety of feral saltwater fishes.

ETIOLOGY

Three groups of streptococci have been isolated from diseased fishes: alpha hemolytic (Minami et al. 1979), beta hemolytic (Minami et al. 1979; Boomker et al. 1979; Robinson and Meyer 1966), and nonhemolytic (Plumb et al. 1974). A comparative study of strains isolated from diseased fish by Japanese and American workers revealed Japanese strains similar to, but not identical with, *Streptococcus faecalis* and *S. faecium*, and American strains similar to *S. agalactiae* (Kusuda and Komatsu 1978). However, strains from fishes did not conform to previously described species.

CLINICAL SIGNS AND PATHOLOGY

Pathological changes vary with species affected; however, unilateral or bilateral exophthalmus and hemorrhages in the eye chamber, inside opercula, and at the base of fins are common. Golden shiners developed numerous raised lesions on the dorsolateral body areas, but no internal signs (Robinson and Meyer 1966); yellowtails exhibited congestion in intestine, liver, spleen, and kidney (Kusuda et al. 1976); and a variety of saltwater species showed hemorrhagic enteritis, bloody peritoneal fluid, and pale livers, but macroscopically normal kidneys (Plumb et al. 1974). Infected cultured eels showed numerous diffuse hemorrhages on the ventral body surface (Kusuda et al. 1978).

Histological changes in diseased rainbow trout included severe congestion and hemorrhaging in the capillaries of the sclera and choroid body of the eye. The lens showed fragmentation of fibers and contained masses of globular debris. Small petechial hemorrhages occurred in gills, and focal vacuolization and necrosis of fibers in skeletal muscle. Hemorrhages occurred in heart muscle and kidney. Diseased yellowtails showed severe epithelial destruction of intestine, focal necrosis of liver, and epithelial degeneration in heart and kidneys. Blood vessels of musculature and subepithelium were inflamed (Hoshino et al. 1958).
DIAGNOSIS

Presence of typical clinical signs and gram positive cocci in kidneys or other internal organs constitutes a presumptive diagnosis. Definitive diagnosis requires isolation and determination of the cultural characteristics of the *Streptococcus*.

SOURCE AND RESERVOIR OF INFECTION

Available information indicates that causative bacteria occur in seawater and mud (Kitao et al. 1979). Fish that survive epizootics serve as reservoirs of infection. Minami (1979) found that fish-pathogenic streptococci survived for 6 months in frozen fish held for use as food for yellowtails.

RANGE

Outbreaks have been reported in cultured freshwater fishes in the United States, South Africa, and Japan, and in cultured saltwater fishes in Japan. Outbreaks have also occurred in several species of feral saltwater fishes off the coast of Florida and Alabama.

MODE OF TRANSMISSION

Initial infection may occur from contact with carrier fish or from infected food fish and water. Robinson and Meyer (1966) transmitted the disease by placing an infected golden shiner in an aquarium with healthy ones. Experimental infections have also been established by injection of $10^4$ to $10^5$ bacteria (Cook and Lofton 1975), or by exposure of fish for 10 min to $10^6$ cells (Robinson and Meyer 1966). Kimura and Kusuda (1979) showed that experimental infection was associated with rapid growth of streptococci in the intestine, suggesting the presence of an exotoxin. They also showed that the injection of cell-free spent medium into fish before the cells were injected increased the virulence of streptococci.

INCUBATION PERIOD

Healthy golden shiners died within 4 to 5 days after being exposed to diseased shiners (Robinson and Meyer 1966). Cook and Lofton (1975) found that injection of $10^5$ to $10^6$ streptococcal cells caused death in 1 to 3 days in susceptible saltwater species and in 7 to 9 days in less susceptible species.

PERIOD OF COMMUNICABILITY

Infection is communicable as long as infected fish are present. Because causative bacteria occur in the environment, disease outbreaks may occur at any time, particularly when fish are stressed.
HOSTS

Natural outbreaks have occurred in golden shinners, rainbow trout, yellowtails, Japanese eels, menhaden (*Brevortia patronus*), hardhead catfish (*Arius felis*), striped mullet (*Mugil cephalus*), pinfish (*Lagodon rhomboides*), Atlantic croaker (*Micropogon undulatus*), spot (*Leiostomus xanthurus*), stingray (*Dasyatis* sp.), and silver trout (*Cynoscion notius*). Experimental infections have been established in green sunfish (*Lepomis cyanellus*), bluegills (*L. macrochirus*), and American toads (*Bufo americanus*).

CONTROL

If causative streptococci are present in mud and water where fish are cultured, avoidance is not a practical means of disease prevention. Cultural practices that effectively reduce stress should help prevent outbreaks.

A daily treatment with erythromycin at the rate of 25 mg per kilogram of fish for 4 to 7 days controlled streptococcal infections in yellowtails better than did daily treatments of oxytetracycline at 50-80 mg/kg, or ampicillin at 200 mg/kg (Shiomitsu et al. 1980). Kashiwagi et al. (1977) showed that 50 mg/kg of sodium nifurstyrenate per day also controlled streptococcal infections in cultured yellowtails. Oxytetracycline and chloramphenicol at 50 mg per gallon of aquarium water controlled streptococcal infections in golden shiners (Robinson and Meyer 1966).

ANNOTATED BIBLIOGRAPHY


Smears were taken from eyes, spleen, and blood of rainbow trout (collected from a fish farm) that were classified as clinically sick, under treatment, recovered, and apparently healthy. Smears were examined microscopically and the bacteria present identified as aeromonads or streptococci. Aeromonads apparently occurred often in the eye and spleen, but not in the blood, of fish of all groups—including those that were apparently healthy. Streptococci were present in the blood, eye, and spleen.

Osmotic fragilities and erythrocyte sedimentation rates (ESR) were measured in healthy, diseased, treated, and recovered trout (*Salmo gairdneri*). Bacterial infection with *Aeromonas* and *Streptococcus* resulted in increased fragility of erythrocytes and elevated ESR's. Treatment with antibacterial agents reduced both disease signs. Possible reasons for such changes are discussed. Caution is urged in the use of apparently healthy fish for the unqualified interpretation of hematological results.


When excessive mortalities occurred in rainbow trout (*Salmo gairdneri*) at a trout farm, a fecal *Streptococcus* belonging to the Lancefield group D was isolated from the spleen, liver, and kidneys of affected fish. This organism was pathogenic for trout but not for Mozambique tilapia (*Tilapia mossambica*), banded tilapia (*Tilapia sparrmanii*), carp (*Cyprinus carpio*), or largemouth bass (*Micropterus salmoides*). Isolation and biochemical characteristics of the organism are described. Symptoms, gross pathology, and histopathology of the disease are described and discussed. The disease resembled hemorrhagic septicemia and was believed to be associated with stress.


A *Streptococcus* isolated from moribund fish in a 1972 Alabama-Florida fish kill produced mortalities in five species of fish. In pathogenicity studies conducted on two of the species with six streptococci unrelated to fish, results were negative.


A systemic streptococcal infection in cultured rainbow trout is described, including symptoms and pathology of affected trout and characteristics of the causative bacterium.

Effect of sodium nifurstyrenate and two therapeutic agents for experimental streptococcal infection were studied with yellowtails. Influence of a challenge dose on the protective effect of sodium nifurstyrenate was examined and compared with that of two other therapeutical agents. Dosage and period of administration were also examined. Sodium nifurstyrenate showed a remarkable therapeutic effect when administered for 3 or 5 days at a daily rate of 50 mg/kg body weight. Ampicillin showed no therapeutic effect after daily administration for 5 days at 50 mg/kg body weight, and chloramphenicol showed only a slight therapeutic effect when fed 5 days at daily rates of 50 or 25 mg/kg body weight.


The antibacterial and bactericidal activity of sodium nifurstyrenate and three therapeutic drugs were studied with Streptococcus sp. The minimum inhibitory concentration against the Streptococcus was 0.15-0.62 µg/mL for sodium nifurstyrenate, 0.31-0.62 µg/mL for aminobenzylpenicillin, 1.2-2.5 µg/mL for chloramphenicol, and 0.075-0.15 µg/mL for tetracycline. A bactericidal effect against Streptococcus was observed at 5 µg/mL of sodium nifurstyrenate for 8 h and at 5 µg/mL of ampicillin for 24 h, but not at 10 µg/mL of chloramphenicol or tetracycline for 24 h.


Intramuscular injection of 10^6 to 10^7 cells of a pathogenic Streptococcus did not produce mortality in yellowtails. However, when cell-free supernatant containing the exotoxin was injected before injection of bacteria, 10^6 to 10^7 cells of either virulent or avirulent Streptococcus produced clinical signs and death.


Over a 10-month period, a Streptococcus pathogenic for yellowtails was isolated from seawater and mud near yellowtail culture pens. The authors suggested that the presence of the Streptococcus in the environment influenced disease outbreaks.

The concentration of Streptococcus in yellowtails after experimental challenge is described. After percutaneous challenge, streptococcal counts reached $10^7$ cells/g of kidney tissue and $10^5$ and $10^6$ cells/g in other internal organs. Intestinal Streptococcus counts reached $10^7$/g within 72 h after oral challenge.


Strains of Streptococcus isolated from diseased fishes in Japan and the United States showed Japanese strains similar to S. faecalis and S. faecium, and American strains were identified as S. agalactiae.


Characteristics of a new organism pathogenic for cultured yellowtails are described, as well as symptoms and clinical signs of disease. The organism was identified as a group III Streptococcus, but was not identical with formerly established species.


A Streptococcus that appeared to be the causative agent of an epizootic in cultured eels was isolated from the kidney of moribund eels from two farms. The pathology of the disease, and the morphology, physiology, and biochemistry of the bacterial isolate are described.


A Streptococcus similar to S. faecalis and S. faecium, a pathogen of yellowtails, was isolated from several species of fish used in the diet of yellowtails. The pathogen survived at refrigerator temperatures and freezing for 6 months. It was suggested that contaminated fishes may serve as a source of oral infection.

A beta hemolytic Streptococcus, similar to S. equisimilis, was isolated from diseased yellowtails. Previously, an alpha hemolytic Streptococcus from diseased yellowtails had been isolated.


Fish kills in estuarine bays along the Florida and Alabama gulf coast were investigated in August and September 1972. Moribund fish of eight species were examined. A nonhemolytic group B type I_b Streptococcus sp. was isolated from over 90% of the fish examined. Parasites and toxicants were eliminated as possible causes of the kills.


Descriptions of two epizootics among golden shiners caused by a group B Streptococcus.


Natural streptococcal infections in cultured yellowtails were effectively controlled by a 4- to 7-day treatment of erythromycin at daily rates of 25-50 mg/kg. The drug was mixed in minced mackerel with a formula feed and a binder. Erythromycin was superior to a daily treatment of 50-80 mg/kg with oxytetracycline, and also controlled outbreaks in which ampicillin was not effective.