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## COLUMNARIS DISEASE OF FISHES

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FISH DISEASE LEAFLET 45      February 1976

UNITED STATES DEPARTMENT OF THE INTERIOR  
Fish and Wildlife Service  
Division of Cultural Methods Research  
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This leaflet supersedes Fishery Leaflet 461 (1958) and Fish Disease Leaflet 16 (1969), both with the same title and prepared by the senior author of the present leaflet.

## INTRODUCTION

Columnaris disease is a chronic to acute infection that affects salmonids and many species of warmwater fishes. The first description of the disease was given by Davis (1922) who named the disease and bacterium from the columnar arrangement of cells as seen in wet mounts. The bacterium causing columnaris disease was first isolated by Ordal and Rucker (1944). They identified the organism as belonging to the group known as slime bacteria or myxobacteria; because it produced fruiting bodies and microcysts, they named it Chondrococcus columnaris. Garnjobst (1945), who was unable to find fruiting bodies, renamed it Cytophaga columnaris. However, in the recently revised Bergey's Manual of Determinative Bacteriology (Buchanan and Gibbons 1974) the columnaris organism and other fish pathogenic myxobacteria have been reclassified as flexibacteria. The forms of flexibacteria pathogenic to fish were recently reviewed by Bullock et al. (1971), McCarthy (1975), and Pacha and Ordal (1970).

## ETIOLOGY

The causative agent of columnaris disease is a long, thin (0.5 to 0.7 X 4.0 to 8.0  $\mu$ m), gram-negative bacterium that moves by a creeping or flexing action. It is now classified as Flexibacter columnaris.

Although we do not agree completely with the present

taxonomic position of the columnaris agent, Bergey's Manual is considered authoritative by most microbiologists; we therefore accept the designation Flexibacter columnaris.

## SIGNS OF THE DISEASE

Columnaris disease begins as an external infection, with lesions developing on body surface and gills. The type of lesions varies with the fish. In scaleless fish such as catfish (Ictalurus sp.), initial lesions are small and circular with gray-blue necrotic centers and red margins surrounded by a ring of inflamed skin. As the disease progresses, lesions spread and may cover most of the body. In scaled fish, such as the bluegill (Lepomis macrochirus), necrotic lesions begin at the outer margins of the fins and spread toward the body.

In Pacific salmonids and warmwater pondfishes, columnaris disease often causes extensive gill necrosis. In such cases, necrosis starts at the margins of the filaments and progresses toward the arches, resulting in the loss of sections of the filaments.

In systemic infections, there are no characteristic external lesions, but typical myxobacterial cells can be seen in smears, or easily isolated on suitable media.

## DIAGNOSIS AND DETECTION

Presumptive diagnosis of columnaris disease is based on the presence of long, thin, gram-negative bacteria in

necrotic lesions on the surface of the body or gills. The tendency of the columnaris bacterium to form mounds or columns, as detected in wet mounts of diseased tissue, also aids diagnosis.

Definitive diagnosis is accomplished by isolation of Flexibacter columnaris, usually on the medium developed by Anacker and Ordal (1955). This isolation is followed by a slide agglutination test with rabbit anti-F. columnaris serum. Colonies of F. columnaris on Anacker and Ordal's medium appear as rough, rhizoid growths that tend to adhere to agar. Also, cells grown on agar or in broth media are usually rough and granular in suspension and are not suitable for slide agglutination tests. Smooth suspensions may be prepared by growing cultures in broth, centrifuging the cultures, resuspending the cells in saline, and heating this suspension for 5 min at 55 C.

At present, there is no described procedure for detecting F. columnaris carriers.

#### PATHOLOGY AND PATHOGENESIS

The pathological changes associated with columnaris disease were described in detail by Davis (1953). Bacteria apparently gain entrance to the dermal tissues as a result of injury to the epidermis, multiply in the connective tissue, and reach the musculature where they form clean, shallow, red ulcerations. The capillaries

become congested and disintegrate, and blood fills the margins of the lesion. There is some phagocytosis in fresh lesions; however phagocytes are apparently destroyed in advanced stages of the disease.

Columnaris disease is common in salmonids held at water temperatures above 15 C. Progress of the disease is faster at the higher temperatures. In acute cases, caused by virulent strains, the gills are the only organs with gross lesions. When gill damage is extensive death is probably caused by asphyxiation and partial loss of excretory function. Although the mode of pathogenesis in systemic infections is still unknown (Pacha and Ordal 1970), a recent review by Morita (1975) indicated that the pathogenicity of myxobacteria can be explained by the release of powerful proteolytic enzymes by cell autolysis. Release of these enzymes explains the histolysis and necrosis observed in columnaris disease. In fish with such lesions, the body is likely to be depleted of plasma proteins and minerals.

#### SOURCES AND RESERVOIRS OF THE PATHOGEN

Flexibacteria are common inhabitants of soil and water. They are usually found on the surface of fishes, particularly on the gills. Most hatchery-reared salmonids contain circulating antibodies to fish-pathogenic flexibacteria (Bullock 1972, Fujihara and Tramel 1968, Fujihara et al. 1965). The presence of these antibodies indicates that healthy adult fishes serve as

carriers of this pathogen. In areas where columnaris disease is common and losses of fish are considerable, strains of Flexibacter columnaris with high virulence are present (Pacha and Ordal 1970).

#### TRANSMISSION

Experiments indicate that Flexibacter columnaris can be transmitted through water. Virulent strains infect fish when added to water; less virulent strains produce infection only when they are injected. Catostomids, cyprinids, and coregonids may serve as reservoirs of infection (Pacha and Ordal 1970). The stress of crowding fish (Wedemeyer 1974), handling them, or holding them at above-normal temperatures, as well as the stress of external injury, facilitates the transmission and outbreak of columnaris disease.

#### HOST AND GEOGRAPHIC RANGE

Columnaris disease is common throughout the world, and affects virtually all species of freshwater fishes, including ornamental fishes. Catadromous fishes, such as eels, are very susceptible when held in fresh or brackish water (Wakabayashi et al. 1970, Hine and Boustead 1974). The disease is common in pond-fish culture in the United States (Meyer 1970) and in carp culture in Europe (Spangenberg 1975).

#### CONTROL

##### Prevention

Since Flexibacter columnaris is shed into water from carrier fish, columnaris disease may be avoided by utilizing water supplies containing no fish.

Injury, crowding, and unfavorably high temperatures contribute to outbreaks and should therefore be avoided.

Davis (1922, 1953) reported that, after handling of fishes, copper sulfate can be used for prevention as a 20-min bath at 37 ppm (1:30,000) or added to pond water at 0.5 ppm. He also recommended a dip of 1 to 2 min in a dilution of 1:2000. Rogers (1971) suggested the addition of potassium permanganate to pond water at 2 ppm for an indefinite period.

Immunization against columnaris disease is possible, by either the oral or parenteral route (Fujihara and Nakatani 1971, Schachte and Mora 1973).

##### Treatment

In vitro, Flexibacter columnaris is susceptible to a wide variety of drugs and antibiotics (Kincheloe 1962, Fijan and Voorhees 1969). Practical control of outbreaks of columnaris disease is possible with a number of drugs. It must be remembered that external treatments are possible only in early stages of the disease,

when infection is still superficial. One such treatment for salmonids is the herbicide Diquat (Chevron Chemical Company, Ortho Division, San Francisco, California) diluted to 8.4 ppm of commercial solution. This equals 2 ppm of the Diquat cation. One hour treatments may be repeated on 4 consecutive days.

Among the older chemicals used for treatment of outbreaks of columnaris disease in pond fishes are two that are also recommended for prevention: copper sulfate at 0.5 ppm (Davis 1922, 1953) and potassium permanganate at 2 ppm (Rogers 1971); the chemicals are added to ponds for indefinite periods.

Oxytetracycline (Terramycin) given orally with food at a rate of 8 g per 100 kg fish per day for up to 10 days is very effective in early as well as advanced outbreaks (Wood 1968).

Other effective new drugs, given orally or used as a bath, are nifurpirinol or Furanace (Amend 1972, Ross 1972, Williams 1973, Amend and Ross 1970); nifurprazine--Aivet in Japan and Carofur in Germany (Shiraka et al. 1970, Deufel 1974)--and oxolinic acid (Endo et al. 1973).

Nifurpirinol and nifurprazine can be added to water at 1 ppm for 5 to 10 min or 0.05 to 0.1 ppm for an indefinite period. In oral administration, these nitrofurans are used at a rate of 2 to 4 mg per kg fish with food for

3 to 5 days. Oxolinic acid is used as a bath at 1 ppm for 24 h. Sulfonamides, such as sulfamerazine and sulfamethazine, can be used orally with food at a rate of 10 to 20 mg/kg fish per day but are less effective than other drugs.

None of these drugs have been cleared by the Food and Drug Administration for control of columnaris disease.

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