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COLUMNARIS DISEASE

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Columnaris disease, caused by the bacterium *Flexibacter columnaris* may result in acute or chronic infections in both coldwater and warmwater fishes. It occurs both as external or systemic infections that result in significant losses of hatchery-reared fish, particularly at warm summer temperatures (Pacha and Ordal 1970; Becker and Fujihara 1978). Epizootics of columnaris disease frequently occur in natural populations and high losses of fish may be observed. Wood (1974) describes strains of high and low virulence; highly virulent forms attack gill tissue and the latter strains are primarily responsible for cutaneous infections. Some disagreements still exist concerning proper taxonomic placement of this organism (Snieszko and Bullock 1976). However, Bergey's Manual of Determinative Bacteriology (8th ed.) classifies it as a flexibacterium.

SIGNS OF INFECTION

In many species of fish, the first sign of the disease may be the appearance of discolored gray, patchy areas in the area of the dorsal fin. These characteristic "saddleback" lesions may progress until skin erosion exposes underlying muscle tissue. These lesions may become yellow and cratered and are often prominent in the mouth and head regions (Wood 1979). Virulent strains of *F. columnaris* may attack gill tissue and cause a "gill rot" condition (Wood 1974). Systemic infections due to less virulent strains may occur with no apparent external signs. However, cutaneous infections seem to be more prevalent in most species of fish.

DIAGNOSIS

A presumptive diagnosis of columnaris disease can be made by the detection of long, slender Gram-negative rods in smears of gills or scrapings obtained from cutaneous lesions. Frequently, material scraped from such lesions and examined under phase contrast microscopy in a wet mount will reveal the presence of unique characteristic “haystack” colonies that are of diagnostic significance. Isolation of the organism on cytophaga medium (Annacker and Ordal 1959) can be accomplished from gill or cutaneous lesions or from the kidneys of chronically infected fish. Colonies of *F. columnaris* exhibit a rough, rhizoid-margined growth that tends to extend into the agar (Snieszko and Bullock 1976). The organism may be differentiated from bacterial gill disease and coldwater disease on the basis of several characteristics. First, the causative organisms responsible for bacterial gill disease are not easily isolated on culture media. Unlike columnaris disease, bacterial gill disease causes no macroscopic gill necrosis. In the case of coldwater disease, the morphology of colonies on culture media is in the form of smooth, yellow colonies as compared to the rough-edged or rhizoid colonies of columnaris disease. Additionally, the columnaris organism differs in cell size, i.e. 0.5 0.7 x 4.0 8.0 nm as compared to 0.75 x 1.5 5.0 nm for the coldwater disease bacterium.

EPIZOOTIOLOGY

GEOGRAPHIC AND HOST RANGES

Flexibacter columnaris is a ubiquitous soil and water-borne bacterium and natural epizootics of the disease are common. Columnaris disease is found worldwide and infects practically all species of freshwater fishes and some amphibians (Snieszko and Bullock 1976; Becker and Fujihara 1978). It has been reported only in freshwater fishes. However, some marine fish are infected by myxobacterial diseases that are similar to columnaris (Bullock et al. 1971). Most hatchery-reared salmonids, coolwater species (such as the tiger muskellunge and the walleye), catfish and baitfish are highly susceptible under intensive culture conditions.

SOURCES AND RESERVOIRS OF INFECTION

When fish are under stress due to elevated temperatures, crowding, etc., *F. columnaris* may attack the fish and cause disease. Infected animals with gill or cutaneous lesions serve as a source of infection. In hatcheries with open water supplies, any species of infected fish in the water supply may serve as a reservoir of infection for the disease. Pacha and Ordal(1970) demonstrated that fish, such as catostomids, coregonids and cyprinids, may serve as reservoirs of infection.

Susceptibility AND Resistance **FACTORS**

There appears to be little or no species resistance to columnaris disease. The role of stressors is considered a key factor in outbreaks of disease. Stress may be provided by crowding, by holding fish at above normal temperatures, as

well as by physical injury due to handling (Wedemeyer 1974). In general, however, temperature seems to be the primary determining factor as to when infection may occur. Experimental studies by Holt et al. (1970) revealed that temperatures in excess of 12.2°C (54°F) were required to induce mortality in fish infected with *F. columnaris*.

MODES OF TRANSMISSION

Research has shown that *Flexibacter columnaris* can be transmitted from fish to fish directly through the water when virulent strains are used. Individual infected fish within a population harbor the bacteria over winter (Wood 1974) and serve as sources of infection during the following summer months when stresses occur due to overcrowding and water temperatures above 12.2°C (54°F), etc. Microcysts formed by *F. columnaris* have been shown experimentally to remain viable over a period of several years. Some uncertainty still exists as to the possibility that these forms are sources of infection under natural conditions (Wood 1979).

INCUBATION PERIOD

The period between exposure to *F. columnaris* and the outbreak of clinical disease varies, depending upon the virulence of the strain of bacteria and the ambient water temperature. Strains of high virulence may induce acute disease within 24 h, whereas less virulent forms may require from 48 h to several weeks (Warren 1981). Holt et al. (1975) have shown experimentally that a high degree of correlation exists between clinical disease and high water temperatures. Their studies also revealed that host species differ in the time from exposure to death. Existing data reveal that the disease has a pronounced seasonal occurrence. Both natural and hatchery epizootics are concentrated during the warm summer months.

METHODS OF CONTROL

PREVENTION

Avoidance of exposure to the disease is a primary method of prevention. This can be accomplished by the use of disease-free water or by the use of U.V. disinfected water supplies. The elimination of wild fish in an open water supply may be helpful when feasible. If water temperature manipulation is available, temperatures above 12.8°C (55°F) should be avoided since they favor development of the disease. Crowding or handling during these periods should be delayed when possible. If the fish must be handled or crowded, certain prophylactic treatments may be administered. These include copper sulfate (CuSO₄) baths for 20 min at 33 ppm or potassium permanganate (KMnO₄) at 2 ppm for indefinite periods (Snieszko and Bullock 1976). Copper sulfate should be used with care since it is highly toxic to fish in soft water. Similarly, KMnO₄ should be used with caution since it may also be toxic to certain species, particularly in soft waters with low levels of organic matter.

THERAPY

Most investigators have determined that treatment for columnaris disease should include both external bath and antimicrobial feed additive therapy to combat both cutaneous and systemic infections (Amend 1970). Compounds such as copper sulfate, potassium permanganate (Snieszko and Bullock 1976) and Diquat (Wood 1979) have been used. Quaternary ammonium compounds such as Roccal, Hyamine and Purina Four Power used at 2-3 ppm in one-hour flow-through treatments have been effective. However, one should consider water quality when making a choice among these compounds. Hardness, for example, may render some treatments ineffective at recommended levels or, in the case of copper sulfate, may be toxic at water hardnesses below 50 ppm CaCO₃. In waters of the northeast, for example, humic acid levels may be high and a permanganate demand of several ppm may have to be satisfied before any beneficial effects can be expected from potassium permanganate treatments.

Oxytetracycline (Terramycin) incorporated into the food at the rate of 4 gm/100 lbs of fish fed at 3% body weight per day is the usual antibiotic treatment used to accompany the chemical bath treatment (Wood 1974).

KEYS STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

Unless there is a closed or disinfected water supply, there is little likelihood of eliminating this pathogen from a culture facility. In the event that an open water supply exists, measures should be taken to prevent the introduction or immigration of any wild fish into the hatchery. If a closed water supply exists, steps should be taken to ensure that resident hatchery fish that may be carriers cannot migrate into the hatchery water supply. If cool water is available during periods of warm weather it should be used.

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