FURUNCULOSIS

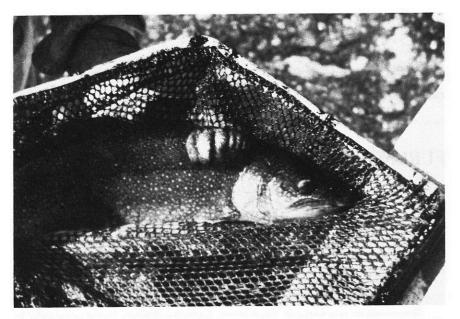
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Furunculosis is a serious, septicemic, bacterial disease found principally in salmonid fishes. but it may also occur in goldfish and other cyprinids. The common name of the disease is derived from the presence of "blisters" or furuncules on the surface of chronically infected salmonids (Snieszko and Bullock 1975). However, this sign is not diagnostic of this disease inasmuch as it may be encountered in fish infected with other pathogens. It should be pointed out that, in acute cases of furunculosis, the furuncules may not be present.

The disease is caused by a Gram-negative bacterium, **Aeromonas salmonicida** described by Griftin et al. (1953). It has recently been demonstrated (Paterson et al. 1980) that the ulcer disease attributed to Hemophilus piscium is, in fact, caused by a strain of **A. salmonicida**. Numerous reports in the literature describe the epizootiology and control of the disease (McCraw 1952; Herman 1968; and Bullock et al. 1971). Furunculosis is found worldwide with few exceptions and causes disease in many species of coldwater and warmwater fishes. In trout hatcheries in North America, it accounts for a high percentage of the fish losses attributable to infectious diseases.

SIGNS OF INFECTION

Clinically-infected fingerlings will usually exhibit hemorrhages at the base of fins and erosion of the pectoral fms. Bloody or hemorrhagic vents and petecbial hemorrhages on the ventral surface are frequently observed. In chronically infected adults, typical "furuncules" or blisters on the skim containing an amorphous yellow substance and blood may be present. This is rarely seen in small or fingerling fish since an acute infection frequently causes massive bacteremia and death before gross lesions develop (Snieszko and Bullock 1975). Internal examinations frequently reveal a bloody fluid in the body cavity. Petechial hemorrhages are commonly observed in the body wall and viscera.



Furunculosis, a bacterial fish disease, may cause large lesions on the external body surface such as those on this lake trout. (U.S. Fish and Wildl. Serv.)

DIAGNOSIS

Positive diagnosis of furunculosis depends upon isolation and identification of the causative agent, A. salmrmicida. The organism is typically a Gramnegative, non-motile rod that ferments selected carbohydrates, produces cytocbrome oxidase, and produces a water soluble brown pigment on several types of isolation agar. Care must be exercised, however, in the identification of non-motile cytochrome oxidase-positive, Gram-negative rods since a number of atypical and achromogenic variants have been reported (Elliot and Shotts 1980a; Paterson et al. 1980) in several species of fish. If an atypical A. salmonicida, such as that encountered in ulcerative disease of goldfish is suspected, enriched isolation media may be required. Elliot and Shotts (1980a) reported that either chocolate agar or tryptic soy agar plus 5% defibrinated sheep's blood was required for adequate growth of isolates. Isolates from suspect variants of A. salmonicida should be given sufficient culture time to allow for those strains which slowly produce a brown, water soluble pigment to do so (Elliot and Shotts 1980b). In such cases, or when rapid identification is needed, the fluorescent antibody technique (FAT) may be used (McDaniel 1979).

It is generally accepted that asymptomatic carriers are very difficult to detect. If it is necessary to establish the absence of carriers in a potential broodstock population, the use of serum agglutination techniques or corticosteroid techniques described by Bullock and Stuckey (1975) might be employed. Sampling of intestinal contents has also been suggested for carrier detection but information is lacking on the reliability of this technique.

EPIZOOTIOLOGY

GEOGRAPHIC AND HOST RANGES

Furunculosis is primarily a disease of salmonid fishes. However, epizootics have been diagnosed in numerous cool and warmwater species, including Esocids and Cyprinids. With the exception of Australia and New Zealand, the disease is distributed worldwide wherever trout and salmon are reared (Snieszko and Bullock 1975).

SOURCES AND RESERVOIRS OF INFECTION

Aeromonas salmonicida is considered to be an obligate pathogen of fish. It has not been found when diseased or carrier fish are absent. The organism may survive for days or weeks in water but cannot persist indefinitely in the absence of carrier fish (Snieszko and Bullock 1975).

SUSCEPTIBILITY AND Resistance FACTORS

Most species of salmonids and many cool and warmwater species are susceptible to the disease. In New York State, for example, the intensive culture of tiger muskies has encountered significant problems with furunculosis. In addition, forage minnows fed to muskellunge have been shown to be carriers and the sources of infection.

Various state fisheries research groups have been working to develop resistant strains of fish through selective breeding. Ehlinger (1964) in New York, reported the first successful work in achieving resistance in brook and brown trout to furunculosis. Field testing in New York, Pennsylvania, and Minnesota has shown that both species possess a high degree of resistance to the disease. Pennsylvania and Missouri have been progressing steadily toward the development of IPN and BKD resistant strains of trout. It should be kept in mind, however, that in this method of fish disease management, resistant stocks are generally considered to be carriers of the disease. As such, they might serve as sources of infection for other susceptible stocks with which they might come in contact.

In addition to species susceptibility to the disease, the problem of antibiotic resistance by the furunculosis bacterium exists. Resistance to both Terramycin and sulfamerazine has become widespread. This is believed to be primarily due to the questionable use of low levels of these compounds as prophylactic measures in the absence of acute disease outbreaks.

MODES OF TRANSMISSION

Transmission generally occurs as a result of contact with diseased or carrier fish, but can occur through water passed from one pond or raceway to another. Contaminated clothing or equipment may also transfer the disease from one culture unit to another. The possibility also exists that fish-eating birds may transfer the disease either by contact or by dropping infected fish into an uninfected pond (Snieszko and Bullock 1975). If eggs from carrier broodstocks are not disinfected prior to incubation, the organisms may be transferred on the surface of the eggs (Wood 1974). Japanese investigators have conducted studies that indicate *A.* salmonicida is not an invasive pathogen. According to their work, infection occurs experimentally only when the pathogen is ingested or has access to external injuries on the fish (Sakai 1979).

Incubation PERIOD

The incubation period for acute cases of furunculosis is probably from 2-4 d. However, in chronic cases, particularly at lower temperatures, the period may be extended by several weeks (Groberg et al. 1978). Furunculosis is usually seasonal with the highest incidence of disease during the midsummer months of July and August. Incidence is related to temperature; the disease is most prevalent in the range from 12.8°C (55°F) to 21.1°C (70°F). At low temperatures, chronic furunculosis has been observed in landlocked salmon cultured in the Adirondack Mountains of New York at water temperatures of 0.5° C (33°F) to 1.6° C (35°F).

METHODS OF CONTROL

PREVENTION

A basic step in the prevention of serious communicable fish diseases is the 'adherence to a sound program ofhatchery inspections and a disease classification system. As a minimum, all lots of fish in a hatchery should be inspected at least once per year for the presence of disease. Utilizing the data generated from these inspections, transfers of suspect or known carrier fish from hatchery to hatchery should be avoided. All eggs from susceptible species should be routinely disinfected using organic iodine compounds at 100 ppm of active iodine for 10 min (Amend 1974) on water hardened eggs. The hatchery water supply should be kept free of fish. Barriers should be provided to prevent the introduction of potential wild carrier fish into the hatchery. Resistant strains of fish should be utilized as a disease management tool where appropriate. If eggs must be imported from outside of the hatchery system, insist that only eggs supplied from inspected and certified furunculosis-free sources be used.

THERAPY

Epizootics of the disease may be treated through the addition of drugs to the fish feed. Terramycin (oxytetracycliie) should be added to feed at the rate of 3.0 g/IOO lb fish, administered daily for 10 d to affected fish. Sulfamerazine should be administered at the rate of 5-10 g/IOO lb fish and fed for 10 or 15 consecutive days. Care should be taken to determine if the strain of furunculosis involved is resistant to either or both of the compounds used for therapy.

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

IMMEDIATE

Use only stocks certified free of furunculosis as sources of eggs for introduction into the hatchery. This practice must be accompanied by the disinfection of all eggs brought into the hatchery. In those cases where hatcheries must use open water supplies, stock only disease-free fish in the headwaters above the hatchery.

Long TERM

If complete eradication of the disease is required, removal of all fish and complete disinfection of the contaminated hatchery may be necessary. This approach should be complemented by an annual disease inspection program which will provide the basic information for disease classification of hatcheries. With these tools, it will then be possible to restrict transfers of fish, thus avoiding contamination of facilities which do not already have the disease.

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