WHIRLING DISEASE

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Whirling disease is a parasitic infection of trout caused by the protozoan Myxosoma cerebralis. Signs of the disease are the result of the parasite feeding on the cartilage of young host fish. A common sign of the disease is rapid, tail-chasing behavior when fish are frightened or trying to feed. The course of the disease ranges from sub-clinical infections to acute disease with mortalities of fry and fingerlings.

SIGNS OF INFECTION

The characteristic sign from which the disease gets its name is the rapid tail-chasing behavior observed in some species and sizes of trout. In surviving fish as the disease progresses, skeletal deformation, including misshapen heads, jaws, and gill covers, and spinal curvature may develop. Trout exposed early in life sometimes develop a “blacktail” due to loss of control of chromatophores on the caudal penduncle. Acutely-infected fry reared in contaminated water may show no signs before they suffer a high mortality. When older fish are exposed, they exhibit less whirling behavior and blacktail, but any skeletal deformities will remain for life. Fish with light infections show none of these signs, but will carry spores throughout their life.

DIAGNOSIS

Infection by M. cerebralis should be strongly suspected whenever signs of whirling, blacktail, or skeletal deformities are seen. Infections are confirmed either by the demonstration of spores, or by demonstration of the immature forms of the parasite in histological sections (see McDaniel 1979, for specific methodology).
An early sign of whirling disease caused by *Myxosoma cerebralis* is a loss of nerve control over the posterior one-third of the body. This young trout shows the typical “black tail” that results. (U.S. Fish and Wildl. Serv.)

*Myxosoma cerebralis* infections cause extensive damage to the skull of developing salmonids. Resulting pressures on nerves may destroy control of muscle groups and lead to grossly deformed bodies in surviving fish. (U.S. Fish and Wildl. Serv.)
EPIZOOTIOLOGY

GEOGRAPHIC AND HOST RANGES

The disease was first reported in Europe about 1904. Its present range, as defined by Halliday (1976), includes Africa, North and South America, Asia, New Zealand, and Europe. In North America, it has been reported from the following states: California, Connecticut, Massachusetts, Michigan, Nevada, New Jersey, Ohio, Pennsylvania, West Virginia, Virginia, and New Hampshire (K. Wolf, USFWS, Kearneysville, WV, personal communication).

Susceptible hosts include all species of salmon, trout, and grayling. Although M. cerebralis has been reported in several non-salmonid species (Halliday 1976), there is reason to question the accuracy of these reports.

SOURCES AND RESERVOIRS OF INFECTION

Infected trout or salmon, contaminated water supplies, and contaminated mud are known to be sources and reservoirs of infection. It has been reported (Christensen 1972) that the spores may survive for 10-15 yr in contaminated mud. Yoder (1972) reported the spread of M. cerebralis from a contaminated hatchery by approximately 6 miles in 28 months following the introduction of the disease to the hatchery. In spite of repeated attempts to destroy the infected fish population and to disinfect the stream, the infection persists and presents a constant threat for further spread of the disease. Bogdanova (1970) reported that, in the USSR, infection rates in natural waters may reach 100% in non-anadromous salmonids, but that the intensity of infection was so low that no clinical signs were observed. Even so, asymptomatic carriers present a threat of infection to hatcheries.

MODES OF TRANSMISSION

The exact route of infection has yet to be demonstrated, but it is suspected that spores are released from dead and decaying fish or shed by living fish. Freshly-shed spores are not immediately infective and must spend time (4-5 months) aging in mud before they develop infectivity (Hoffman and Putz 1969). Transmission of whirling disease with fertilized eggs is highly unlikely if care is used to avoid contaminated water and mud during the spawning and egg-packing procedures. Spores can survive as long as two months in frozen infected fish.

Other recent work suggests that tubificid worms may be involved in the transmission of M. cerebralis (K. Wolf, USFWS, Kearneysville, WV, personal communication).

SUSCEPTIBILITY

Rainbow trout, brook trout, Atlantic salmon, kokanee salmon, and European grayling may become severely diseased. Brown trout, coho salmon, and lake trout are very resistant to this disease and, under experimental conditions, develop less than 1% of the average spore numbers found in rainbow trout (J. O’Grodnick, Pa. Fish and Game Commission, Bellefonte, PA, personal communica-
icitation). According to O’Grodnick: “since M. cerebralis has already become established in certain geographical areas, policies of eradication are not practical and abandonment of existing facilities is not economically justifiable. An acceptable management alternative may be the rearing of proven resistant salmonids in contaminated hatcheries. State agencies could arrange production schedules so that resistant species which do not develop clinical whirling disease are substituted for susceptible rainbow trout or brook trout. Coho salmon, brown trout, and lake trout can be reared in contaminated hatcheries with no whirling disease development. The number of spores developed by these species is quite low and it would be unlikely that establishment of whirling disease in large water areas would be possible by stocking these fish. Also, hatchery effluent-receiving streams would receive fewer spores from very resistant production fish and contamination would be reduced. Since the value of the coho salmon and lake trout to the Great Lakes fishery is well documented, a careful evaluation of the policies regarding rearing these resistant species in known M. cerebralis contaminated waters should be made”.

Fish are most susceptible to infection during the first 12 months of life. Sac fry at an age of 3 d are the youngest fish known to become infected (Puta and Hoffman 1966). Fish of 4.5 months of age or older do not develop acute clinical signs even though they may still become infected and serve as asymptomatic carriers (Hoffman and Byrne 1974: Hoffman 1976).

INCUBATION PERIOD

Infected fish may show signs of infection 28 wk after exposure (Hoffman 1976). Spore formation in infected fish takes 52 d at 17°C, 3 months at WC, and 4 months at 7°C (Hilliday 1973). Acutely infected fish may not develop any overt signs of the disease.

METHODS OF CONTROL

PREVENTION

The only means of preventing infections of M. cerebralis is to keep the parasite away from any susceptible fish. The importation of infected fish or the use of contaminated water should be avoided.

THERAPY

No effective therapy is known

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

IMMEDIATE

There is no way known to eradicate the parasite from infected fish. Therefore, the only way that the parasite can be eradicated is to eliminate infected fish and to remove, seal off, or destroy all other infected material, such as mud and
contaminated water. All of the fish in ponds known to be infected must be eliminated. Incineration or deep burial with quicklime (CaO) is recommended but dressed carcasses may be used for food, provided they are cooked and that careful control is maintained over the disposal of the heads and offal. In certain cases, special landfill or burial permits may be necessary. Smoking also kills the parasite (Wolf and Markiw 1982). After the fish have been disposed of, disinfection of the contaminated facility can be considered if a water source free of contamination is available. Contaminated earthen ponds should be replaced with concrete or other impervious surfaces such as plastic or fiberglass, if possible. If not, the pond should be drained and thoroughly cleaned including removal of as much mud as practical. Quicklime at 380 g/m² (3,360 lb/acre) or calcium cyanamide at 500 g/m² (4,231 lb/acre) have been effective disinfectants when spread evenly on drained, wet pond bottoms and dikes, and if treatments are repeated several weeks or months later, preferably in the spring and autumn (Christensen and Bogdanova 1973; Hoffman 1976; Schaperclaus 1954).

Concrete surfaces or structures should be wet when disinfected with these chemicals.

Calcium cyanamide is no longer commercially available, thus quicklime is recommended (Schaperclaus, Kulow and Schreckenbach 1979). When using any chemicals, take adequate safety precautions and follow all label instructions. In addition, be sure that applications will have no adverse effects on downstream waters.

After a facility has been disinfected, small test plantings of susceptible rainbow trout should be used to see if the job was successful. The test fish should be young fingerlings less than 4 months of age and should be left in the test ponds at least 4 months.

The hatchery may be restocked the following season with uninfected fish. The young fish should be kept in fiberglass, metal, or concrete facilities as long as possible. Earthen ponds should not be used until the fish are at least 8 months old unless it is certain that the disease has been eliminated from the hatchery. Rearing tanks and ponds should be kept as clean as possible, and all dead fish should be removed daily. Although fish in these ponds may become lightly infected, they will usually show no signs of disease (Hoffman 1976). However, the population must be considered to be potential carriers and should not be planted into areas known to be free of whirling disease until at least 2 yr of rearing and repeated inspection work can certify that the population is free of the infection.

If the water supply is known to be contaminated with the parasite, it should be treated in an attempt to kill or remove the spores. Filtration of the water through pore sizes of less than 10 nm in diameter will remove the parasite. Irradiation of water with ultraviolet light at 35,000 microwatts/cm² after filtration through 25 nm filters is effective (Hoffman 1976). Sand gravel filters may be used as prefilters to ultraviolet irradiation. However, no examples of effective water supply decontamination for whirling disease on a hatchery scale are available.

**LONG TERM**

Prevention is the most effective control. If an outbreak occurs, eradication should be attempted. In enzootic areas where the disease has spread to natural
waters, and where there is no possibility of total fish stock destruction and disinfection, fish can be raised in the presence of the parasite. These fish will become infected, but if everything possible is done to lessen the degree of infection, most of the infected fish will be asymptomatic. These fish should be stocked under strict control and only into known infected areas, or be processed and used as table fish. It is important that the water supplies be made free of infection and that earthen ponds be replaced with concrete or completely disinfected. If fish must be raised in contaminated earthen ponds, the fish should be first raised to at least 7-13 cm (3-5 in) long and to at least 8 months of age in uncontaminated water before moving them to the contaminated earthen ponds. Covering the bottoms of contaminated earthen ponds with plastic and use of spore-free water has been effective in preventing clinical signs of whirling disease in Denmark (Hoffman 1976).

REFERENCES


