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WHIRLING DISEASE OF TROUT

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INTRODUCTION

Whirling disease of trout is a serious hatchery disease in Europe and has recently spread to Russia, Italy, and the United States.

IDENTIFICATION

This disease derives its name from the rapid, tail-chasing type of whirling which is often seen when the fish is frightened or trying to feed. This whirling differs from the horizontal spiraling of the fish along its long axis which is characteristic of infectious pancreatic necrosis (Fishery Leaflet 453). The whirling symptom is associated with lesions and disintegration of the cartilaginous skeletal support of the organs of equilibrium caused by invasion of a parasite protozoan. The damage to the head skeleton is evident in older trout as a depression in the head or as misshapen jaws. Sometimes the spinal column is affected resulting in spinal curvature. Pressure on the nerves which control the caudal pigment cells results in "blacktail" in many fish. Symptoms may appear as early as two weeks after the fry start feeding and in heavily infected groups of fish there may be many mortalities. The whirling

and black tail are less striking as survivors grow older and these symptoms tend to disappear in one to two years. Sunken heads and spinal curvatures do not disappear, however.

CAUSE OF THE DISEASE

Myxosoma cerebralis (Lentospora c.) a myxosporidian protozoan which was discovered in Europe in 1903 is the causative organisms. The development of the parasite takes four months at which time the spores can be demonstrated in wet mounts made from scrapings of cranial skeleton and auditory capsule (organ of equilibrium). The spores are about 10 microns in diameter and possess two polar capsules. Prior to this, verification can be made only by histological methods.

SOURCE AND RESERVOIR OF INFECTION

As far as is known this parasite attacks only salmonid fishes. Infected yearlings in the water supply are the usual source. The spores are very resistant and probably survive drying and/or freezing. It is difficult to eradicate the disease from earthen ponds.

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MODE OF TRANSMISSION

Although there is some evidence that spores are released through the intestine, it is probable that most fry become infected from accidentally ingesting spores that have been released from dead trout four months of age or older which are crushed or decomposed. The ingested parasite leaves its spore capsule in the intestine and migrates through the intestinal wall to the head cartilage where development continues.

INCUBATION PERIOD

The typical symptoms usually appear at one to two months after exposure to the disease. However, mortalities of fry may occur before this.

PERIOD OF COMMUNICABILITY

Spores remain in infected fish at least three years and probably longer.

SUSCEPTIBILITY AND RESISTANCE

Rainbow and eastern brook trout are susceptible to the disease. Very young fry are most susceptible; occasionally year-old fish may become infected but older fish do not become diseased. Brown trout become infected but do not develop disease conditions and, therefore, may act as carriers. Salmon and grayling have been found infected in Europe.

RANGE

This disease was known only in Europe until recent years. Apparently it originated in Central Europe and spread to other European countries, including Russia. It has been reported in Italy and the United States in the last few years.

METHODS OF CONTROL

A. Preventive measures

Destroy all fish from ponds containing fish known to be infected. Incineration or deep burial is recommended.

Fish must be reared in spring or well water for the period of maximal susceptibility (8 mo.). No adequate filtering device is available for stream water. Earthen ponds must be used during this important period.

All concrete ponds must be cleaned thoroughly and disinfected with calcium cyanamide (0.08 lb/sq. ft. on wet concrete). Quicklime and sodium hypochlorite are chemicals second choice.

Earthen ponds are usually responsible for the perpetuation of the disease. Drain the ponds and immediately apply calcium cyanamide as above. Allow to stand a month or more and clean out muck as thoroughly as possible--by digging deeply or plow under in farm field. Fill ponds with water, drain and repeat treatment. Ponds may be used six weeks later. It may be necessary to repeat this treatment the following year.

The hatchery may be restocked the following season with fish from known uninfected sources. The young fish should be kept as long as possible in metal or concrete facilities. Keep ponds cleaned and remove mortalities each day. Use earthen ponds only for eight-month or older until it is certain that the disease has been eliminated.

Brown trout and older rainbow and brook trout, although not obviously diseased, may act as carriers.

B. Therapy

No proven chemotherapy is available present although Scolari (1954) cites partial prophylaxis.

ANNOTATED BIBLIOGRAPHY

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Plehn, Marianna.

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Schäperclaus, Wilhelm.

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Scolari, C.

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Mortalities but not all symptoms were suppressed with Stovarsole (acetarsone) at the rate of 10 mg. each day per kg weight in the food. Drug was administered for three consecutive days with weekly intervals between medications.

Tack, E.

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Gives an account of first epizootics of this disease in Russia. Recommends that no fish be transferred from an affected hatchery. Describes the possibility of some spores being eliminated with the feces. She found the disease in rainbow trout primarily, but also brown trout and salmon.