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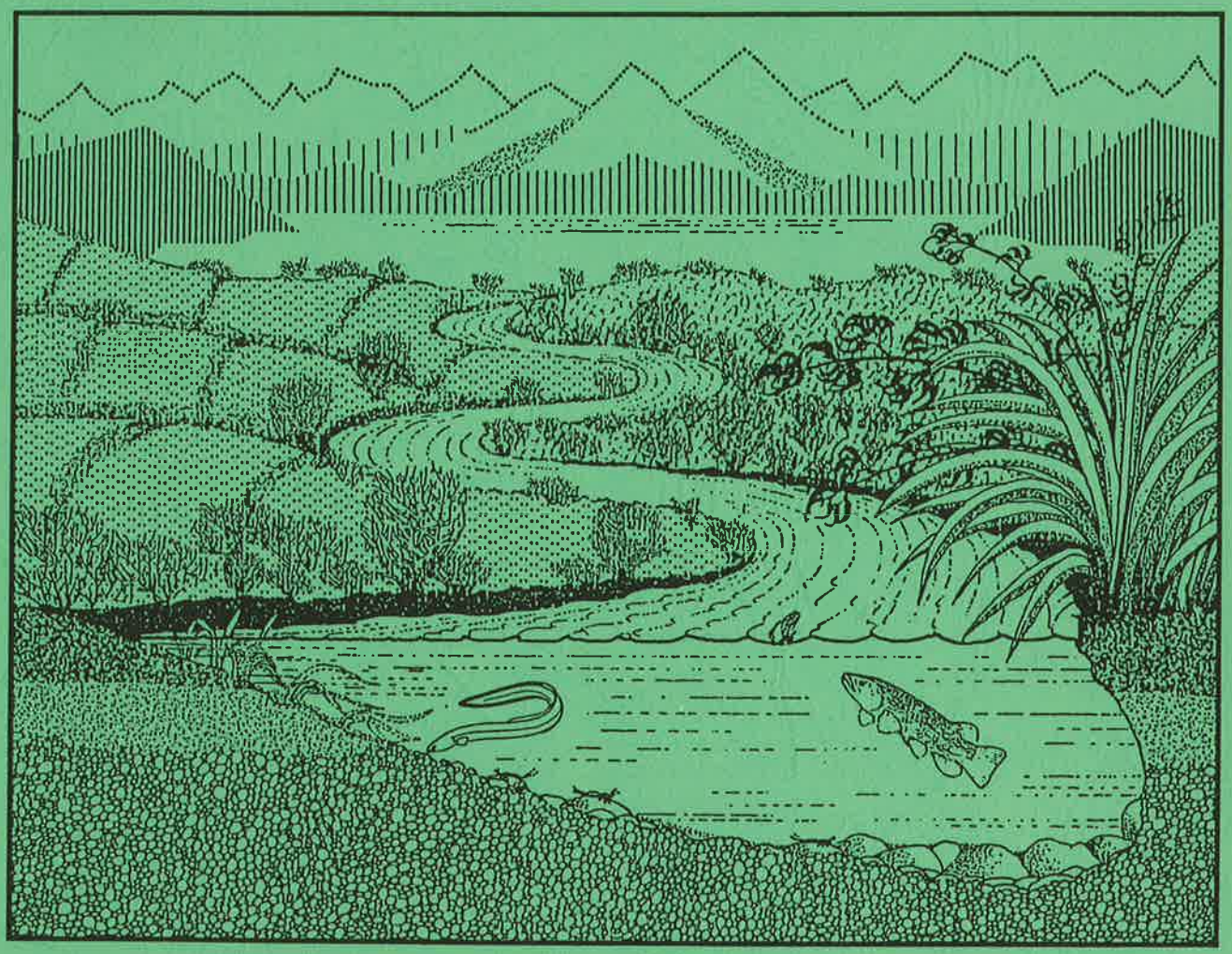
New Zealand Freshwater Fisheries Report No. 112

A guide to diseases of salmon in New Zealand



MAFFish

New Zealand Freshwater Fisheries Report No. 112

A guide to diseases of salmon in New Zealand

by

N.C. Boustead

Freshwater Fisheries Centre

MAFFish

Christchurch

August

1989

NEW ZEALAND FRESHWATER FISHERIES REPORTS

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FIGURE 1. Wake from a motor vessel travelling through a dense bloom of Heterosigma which has coloured the sea water brown. (See Section 3.22.)
Specimen origin: Big Glory Bay, New Zealand.

FIGURE 2. Red Skin Lesion. Typical skin lesions on chinook salmon reared in sea water. The skin damage is a clean wound revealing muscle underneath which is red with blood. (See Section 3.21.)
Specimen origin: New Zealand.

FIGURE 3. Vibriosis. Typical haemorrhages (arrowed) in the liver of a chinook salmon reared in sea water. (See Section 3.3.)
Specimen origin: New Zealand.

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SUMMARY

This report provides an introduction to some salmon diseases, their identification, prevention, and treatment. It is intended for people with an interest in practical salmon culture in New Zealand. Because there are numerous textbooks available which provide comprehensive descriptions of fish diseases, this guide is limited to a brief outline of the salmon diseases found in New Zealand and of some exotic diseases which are significant or particularly relevant to New Zealand.

The diseases are presented in two sections. The first section describes most of the diseases of salmon known in New Zealand at the time of writing (June 1989). The second section describes exotic salmonid diseases which are specified in New Zealand legislation but have not yet been reported in this country, and some other serious exotic diseases. Salmon culturists need to be aware of these diseases in order to meet the requirements of the Animals Act 1967 and the Freshwater Fish Farming Regulations 1983. The more common or important of these diseases are described by their cause, signs, diagnosis, pathogenicity, treatment, and prevention.

Management practices that can prevent the introduction of disease are outlined, together with the means to detect early signs of disease which can help minimise losses. Practical advice is provided on disinfection procedures for salmon ova, fish transporters, and equipment. The licensing of treatments by the Animal Remedies Board is explained and general guidelines on treating fish, with a list of remedies, are provided. Methods for the collection, preservation, and submission of samples for disease diagnosis are given. A list of some of the most relevant books, journals, and magazines is included to provide sources of further information.

1. INTRODUCTION

Salmon farming in New Zealand is carried out predominantly with chinook salmon (Oncorhynchus tshawytscha), and with a smaller number of sockeye salmon (O. nerka). There is some experimental rearing of Atlantic salmon (Salmo salar). Rapid growth in the number of salmon

farms over the last few years has created a demand for knowledge of the diseases which may occur, and this has led to the production of this guide.

The extent of losses from fish diseases is dependent not only on the virulence of the pathogen concerned, but also on the design and operation of the farm, and on the speed with which a disease is identified and treatment begun. The optimal design and operation of a fish farm lies beyond the scope of this publication. It is intended that this guide will assist salmon culturists in the recognition, treatment, and understanding of fish diseases, which will help to minimise the loss of fish from disease as well as assisting in the production of healthy salmon.

There is no doubt that as fish culture and fish health studies continue to grow, more information will be gained that should be included in future editions or updates of this guide. In addition, changes to the legislation are proposed (e.g., Animals Act 1967) which will make some of the information in this report obsolete, indicating the need for a revised edition. The author would be grateful to receive any comments or suggestions that would improve the value of this guide.

2. TREATMENT NOTICE

The use of drugs, chemicals, and vaccines must comply with current laws and regulations. Mention of product names does not imply endorsement by the Ministry of Agriculture and Fisheries. The Ministry does not accept liability for treatment advice contained in this publication. Details of recommended treatments are given in Appendix I.

3. SALMON DISEASES KNOWN IN NEW ZEALAND

3.1 Bacterial Gill Disease

Cause: Bacterial gill disease (BGD) is caused by the presence of a large number of long filamentous bacteria on the surface of the gills (Fig. 9). These bacteria were first called Myxobacteria, but they are

now described as Flexibacter or gliding bacteria. Recent studies in Japan and the United States have classified other bacteria causing BGD as Flavobacterium. Further studies or a revision of the taxonomy of Flexibacter will be required.

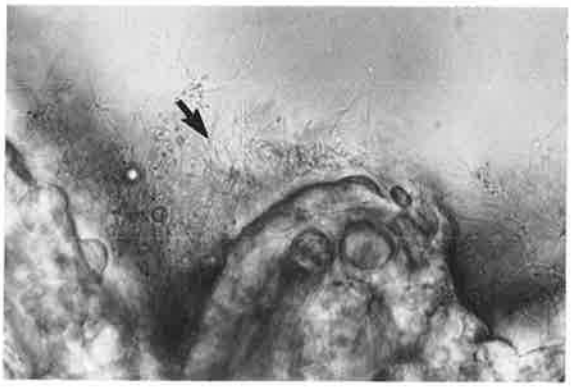


FIGURE 9. Bacterial gill disease. Arrow points to masses of the characteristic bacteria on the edge and surface of gill filaments, as seen in a wet mount preparation under a microscope. Specimen origin: New Zealand.

Signs: Normal swimming and shoaling behaviour in raceways or ponds changes when fish become infected with BGD. Infected fish swim more slowly, and position themselves closer to the edges and the surface. If the infection is severe, they will position themselves an equal distance apart and their opercula will remain more open than usual. They will also stop feeding and the daily mortality will increase rapidly.

Diagnosis: Microscopic examination of an unstained, wet mount preparation of the tips of the gills will show the presence of a large number of the characteristic long, slender bacteria. These will be seen covering the gills, lying between the gills, and lying on the surface of the slide. It would be possible for an experienced manager to make a presumptive diagnosis by recognising the change of behaviour and an increase in mortality, given a knowledge of the history of BGD at that site.

Pathogenicity: BGD is common in fry reared in fresh water, particularly from first feeding until about two months of age. Yearling sockeye may also become infected. BGD is more common in fish reared in hatcheries supplied with river water, but it may occur in fry in troughs supplied by bore water. Fish may be re-infected at hatcheries which have wild fish in the water supply. The occurrence of this disease is often considered indicative of poor water quality, occurring particularly when

ammonia levels and turbidity are high. Dusty food and crowding also can precipitate the disease in conditions of otherwise excellent water quality. Its onset can be very rapid, and the loss of stock will be substantial unless treatment is begun promptly.

Treatment: Chloramine-T is the therapy of choice and will readily control the disease. Other recommended treatments include diquat, and quaternary ammonium compounds such as Roccal or Hyamine 3500. The drug Furanace is effective, but it is expensive and continued use may create drug resistance in the bacteria.

Prevention: Attention to water quality, as discussed above, and the use of water supplies free of wild fry (bore or spring water) for the first few months, will reduce the likelihood of BGD occurring. Elimination of dusty food and reduced stocking densities also will help.

3.2 Columnaris

Cause: Columnaris is a bacterial disease (Fig. 10) caused by Flexibacter columnaris. This organism belongs to the same group of bacteria as those causing BGD and cold water disease. It occurs in fresh water.

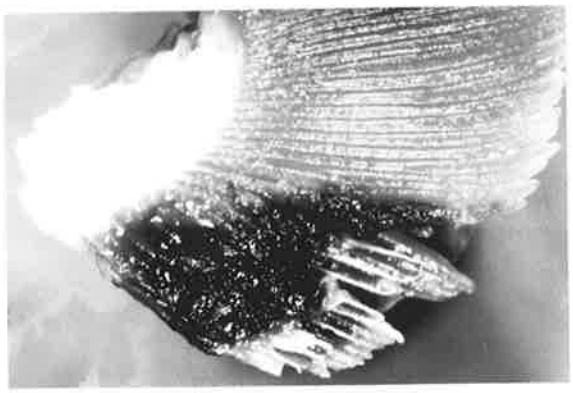


FIGURE 10. Columnaris lesions of bacteria and congealed blood causing erosion on the gills of an eel. (Photo courtesy of J. Bahler.) Specimen origin: New Zealand.

Signs: Columnaris has been found to cause erosion and bleeding of salmon gills in water at a temperature of 18°C. It also has occurred widely in cultured eels in the North Island. Columnaris may cause grey lesions to form on the dorsal surface, and these can progress to expose the underlying muscle tissue. The lesions may become yellowed and form craters.

Diagnosis: A presumptive diagnosis can be made from microscopic observation of the characteristic bacteria taken from lesions. The main difference between BGD and columnaris is that columnaris tends to cause necrosis and erosion of gill, skin, or muscle tissues, whereas BGD does not. Columnaris occurs at warm water temperatures, whereas BGD is usually found in cooler waters. Diagnosis can be confirmed from the pathology of the disease, and from isolation and identification of the bacteria.

Pathogenicity: Flexibacter columnaris is considered to be a ubiquitious soil and water-borne bacterium, and the disease can break out naturally. Stress factors such as crowding and an elevation in water temperature may precipitate the disease. Outbreaks of columnaris can arise quite rapidly, and require prompt recognition and treatment.

Treatment: Furanace has been used successfully to treat outbreaks of columnaris in eel culture, but this may lead to drug resistance. Other treatments recommended in the overseas literature include a combination of external bath treatment and the addition of antibiotics (oxytetracycline) to the food. Chemicals for bath treatment were copper sulphate, potassium permanganate, diquat, and quaternary ammonium compounds.

Prevention: Maintenance of a good rearing environment, where fish are not overcrowded, may assist in preventing the disease. As columnaris is most severe at high water temperatures, rearing of fish in cooler water is likely to prevent outbreaks of the disease, although it is recognised that lowering water temperatures is likely to be impractical.

3.3 Vibriosis

Cause: Vibriosis (Fig. 3) is caused by various species of Vibrio, including the bacteria V. anguillarum and V. ordalii. It occurs principally in sea water, although some outbreaks have been recorded overseas in fresh water. Both V. ordalii and V. anguillarum have been found in New Zealand (C. Anderson, Wallaceville Animal Research Centre, pers. comm.). These vibrios also have been described respectively as V. anguillarum biotypes I and II. V. ordalii also has been known as V. anguillarium type 1669. (Also see Hitra disease, Section 4.9.)

Signs: Signs of the disease are similar to those of some other bacterial septicaemias. Mortality can be abrupt, without any external symptoms, although haemorrhaging of the eyes, gills, vent, skin, and internal organs may occur. In chronic cases, haemorrhagic ulcers will form on the skin and in the muscle. These are usually bright red, and may be raised and boil-like. Haemorrhages in the muscle and liver have been typical of affected fish in New Zealand.

Diagnosis: Diagnosis is based on the isolation and identification of the causative bacteria from the blood, organs, or lesions of affected fish. This can only be done in a microbiology laboratory. Many other bacterial diseases cause very similar lesions, and laboratory tests are required to distinguish between them.

Pathogenicity: Vibrio bacteria are present in sea water, and they are pathogenic to many species of marine fish. The occurrence and severity of the disease may be influenced by factors such as transfer shock, overcrowding, low dissolved oxygen, high temperature, poor water quality, and fluctuating salinity.

Treatment: Chemotherapeutic agents are required to treat vibriosis. At present, only oxytetracycline (Terramycin) has been used in New Zealand, as a food additive. Other drugs recommended in overseas studies are Furazolidone and Oxolonic Acid.

Prevention: Avoidance of stress factors will help to prevent vibriosis, and vaccination with antisera against both species of Vibrio is practised widely. This involves a bath or spray treatment, and must be carried out at least 10-20 days before transfer to sea water. The vaccines must be licensed by the Animal Remedies Board before they can be used on fish. At the time of writing, only Biovax vaccine is licensed for use in New Zealand. It is manufactured by Biomed Research Laboratories, Seattle, and is distributed by Phoenix Pharm Distributors, P.O. Box 34-391, Birkenhead, Auckland.

3.4 Fin Rot and Cold Water Disease

Cause: Both of these conditions (Fig. 11) usually are found in yearling fish raised in fresh water. Fin rot is associated with high stocking densities or adverse rearing conditions, and may be followed by cold

water disease, which is associated with the bacterium Cytophaga psychrophila.

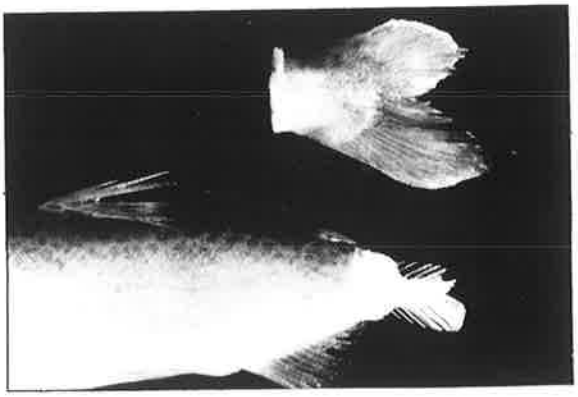


FIGURE 11. Fin rot and cold water disease. Top specimen shows the white appearance of the dorsal edge of the caudal fin. No cytophaga bacteria have been observed associated with this damage. Lower specimen shows a caudal fin which has been infected with fungi and cytophaga bacteria causing erosion to expose the vertebrae. Specimen origin: New Zealand.

Signs: Fin rot appears first as a conspicuous whitening along the edge of the caudal fin, which is due to a proliferation of cells. As the disease progresses, the fin becomes frayed and eroded. Cold water disease may erode the tail completely, leaving only a stump with the vertebrae showing. Muscle tissue is also exposed and eroded.

Diagnosis: No pathogens have been found to be associated with the early stage of this condition. Cold water disease can be recognised from the severe erosion about the tail region. Microscopic examination of the damaged tissues may reveal a large number of Cytophaga bacteria, which appear as long thin rods. Laboratory examination is required to confirm the identity of the bacteria.

Pathogenicity: Cold water disease has been observed at temperatures of 8-10°C. Mortality has been chronic, with a few mortalities occurring over a long period, and infected fish may survive for quite some time. Fish with the whitened discolouration of the tail will feed and behave normally, but they are more likely to be found at the lower end of a raceway. Secondary infection with fungi, recognisible as tufts like cotton wool, sometimes occurs on the damaged tissues.

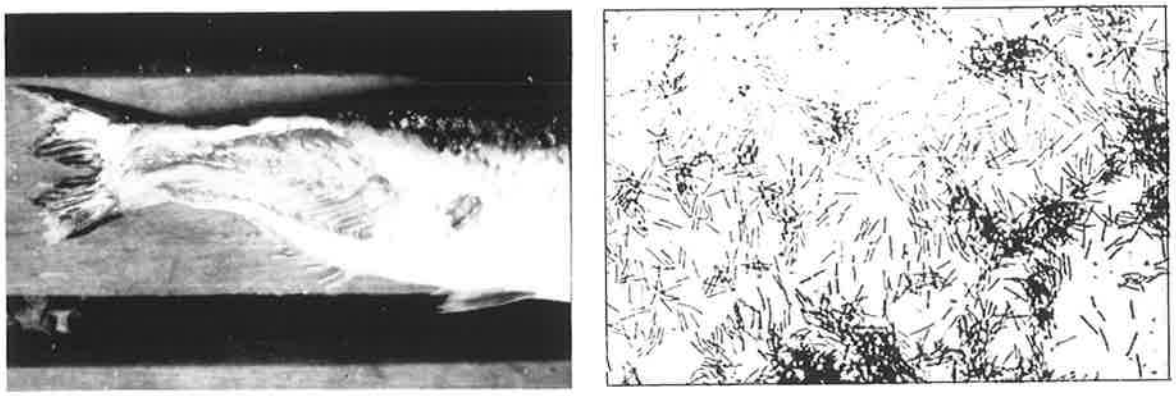
Treatment: The whitening of the caudal fin appears to be a cellular response to crowding and possibly to poor water quality. Treatment of this condition will be ineffectual, although the use of malachite green may help to prevent fungal infection.

External infections with Cytophaga bacteria may respond to Chloramine-T or quaternary ammonium compounds (Hyamine 3500 or Roccal). In advanced cases, where bacteria have been shown to be present, antibiotics may be required to control infection.

Prevention: This condition is aggravated by adverse rearing conditions. Appropriate preventative measures will need to be determined for each site, taking experience and water flows into account.

3.5 Marine Flexibacter

Cause: This bacterial infection occurs on salmon raised in sea water, and is associated with external lesions on the lateral and ventral surfaces (Fig. 12a), and on the gills and mouths of juveniles. The bacterium is distinctive, and may be the same as diseases described as Cytophaga or Sporocytophaga. Laboratory culture and characterisation will be required to determine its identity and pathogenicity.



(a) (b)

FIGURE 12a. Marine cytophaga bacterial infection on the edge of a lesion on the skin of a chinook salmon. Specimen origin: New Zealand.

FIGURE 12b. Photomicrograph of stained cytophaga bacteria from the fish illustrated in Figure 12a. Specimen origin: New Zealand.

Signs: The edge of external lesions will have a yellow or creamy mucoid appearance. Affected fish will be listless, and will swim apart from unaffected fish.

Diagnosis: Microscopic examination of material from the edge of the lesion will reveal numerous, very long, thin bacteria (Fig. 12b) similar to those causing BGD and cold water disease. Their identity can be confirmed from fresh samples in the laboratory.

Pathogenicity: This condition is described in overseas reports as being of minimal significance. It has been seen in New Zealand in fish which had been damaged while being graded, and it was considered to be a consequence of grading. It also has been found in association with lesions and significant mortalities of juveniles following transfer to warm marine waters.

Treatment: Treatment was not attempted in the fish which had been damaged during grading because the losses were not significant. Overseas reports recommend a bath treatment with oxytetracycline.

Prevention: Handling of the fish should be minimised during susceptible periods, e.g., following the introduction of fish to sea water, or when water temperatures are high.

3.6 Pasteurella

Pasteurella is another bacterial organism that can cause septicaemia in fish. It has been isolated once in New Zealand, from sea-cage fish with red lesions, although it was probably a secondary opportunistic pathogen. Overseas reports describe Pasteurella being associated with diseases of various fish, including white perch, yellow-tail, brown trout, and Atlantic salmon. Signs of the disease are similar to those of other bacterial septicaemias such as vibriosis, i.e., haemorrhaging around the operculum and fins, with external and internal lesions. Diagnosis is based on isolation and identification of the causative bacterium in a microbiology laboratory. Various antibiotics have been used in Japan to control the infection, but the effectiveness of these is becoming limited by the emergence of drug-resistant forms of the bacterium. Vaccines are now being developed.

3.7 Nocardiosis

Norcardia is a normal, soil-dwelling, filamentous bacterium that may opportunistically infect fish and higher vertebrates. This bacterial

infection was held responsible for losses of chinook salmon at one location in 1972 (P. M. Hine, MAFFish, pers. comm.). Only juvenile fish were affected, and no causative organism was isolated. The disease has not been seen since.

3.8 Whirling Disease

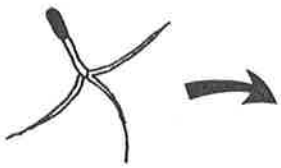
Cause: Whirling disease is caused by the myxosporean protozoan parasite Myxobolus cerebralis, which invades and develops in bone and cartilage of the head and spine of the fish. (Myxobolus cerebralis was originally named Myxosoma cerebralis.)

Life Cycle: An understanding of the complex life history of this parasite is necessary to appreciate its potential effects and the methods for its control (Fig. 13). (Note that the involvement of tubificids in the cycle is disputed by some research workers.)

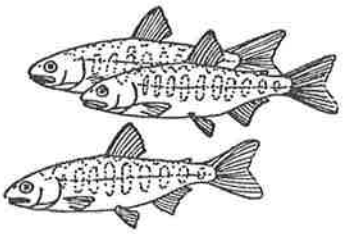
Signs: The classic sign of whirling disease is the tail-chasing behaviour for which the disease is named, i.e., fish adopt a crescent shape and swim repeatedly in tight circles of 10-30 cm diameter. Infection with M. cerebralis also can cause tail blackening and skeletal deformities. It is important to realise that, in New Zealand, the intensity of infection has been low, both in the percentage of fish infected and in the degree of infection as measured by spore numbers. Consequently, all of the salmon and most of the trout found to have whirling disease in New Zealand had none of the recognisable signs of the disease and appeared to be quite normal. The parasite was detected only by laboratory examination.

Diagnosis: Diagnosis of whirling disease is based on the characteristic spores isolated from infected bone or cartilage. Because of the possibility of confusing M. cerebralis either with other exotic myxosporeans from salmonids or with other myxosporeans from New Zealand native fish, diagnosis should be supported by histological demonstration of spores in damaged cartilage. The most sensitive techniques for isolation of the spores are by plankton centrifugation, enzyme digestion, or a combination of both.

Pathogenicity: Whirling disease is a disease of juvenile fish and it can cause mortality in the first six months of life. Older fish may

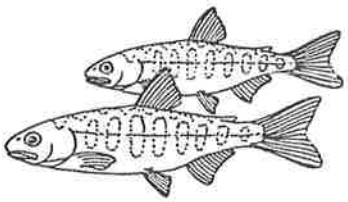


Mature actinosporean is released, or tubificid is ingested to infect other salmonids.



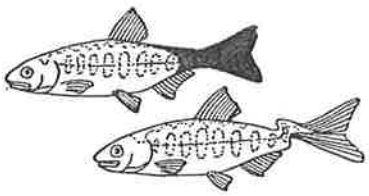
Healthy susceptible salmonids.

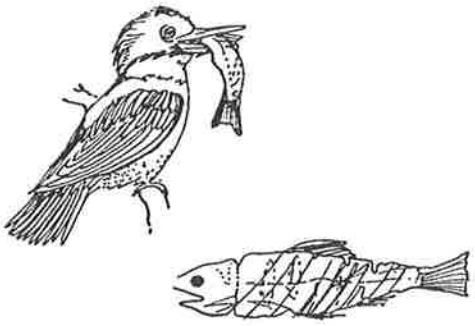
Spore Development to an actinosporean in the tubificid takes 3-4 months.



Infected salmonids may appear normal, or may develop black tail, skeletal deformities, and whirling behaviour, 30 to 50 days after infection.

Tubificid worms become infected by spores





Formation of spores in cartilage takes 3-4 months.

Spores are released from infected fish at death or after ingestion by a predator.

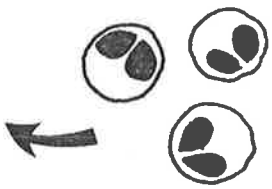


FIGURE 13. Life cycle of Myxobolus cerebralis.