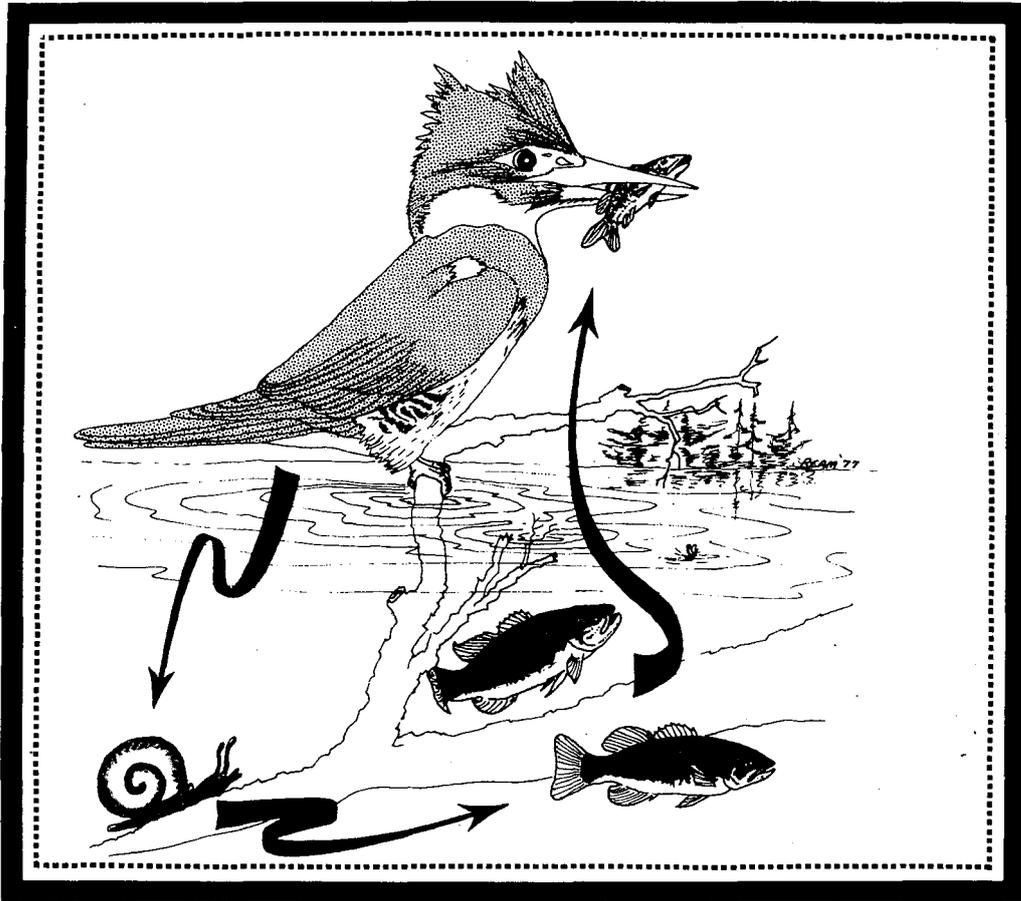


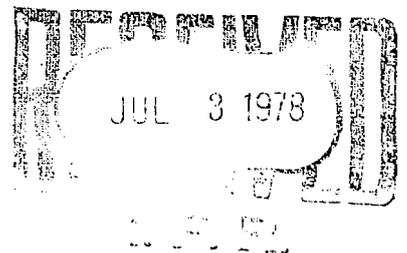
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Manual of ----
**COMMON DISEASES,
PARASITES, AND ANOMALIES
OF MICHIGAN FISHES**



**Michigan Department
Of Natural Resources
Fisheries Division**



MICHIGAN DEPARTMENT OF NATURAL RESOURCES

llon

INTEROFFICE COMMUNICATION

Lake St. Clair Great Lakes Station
33135 South River Road
Mt. Clemens, Michigan 48045

JH

- J.R.H.
STAVE
- ASA

TO: Dave Weaver, Regional Fisheries Program Manager, Region III
Ron Spitler, Fisheries Biologist, District 14
Ray Shepherd, Fisheries Biologist, District 11

FROM: Bob Haas, Biologist In Charge, Lake St. Clair Great Lakes Station

SUBJECT: Impact of the red worm parasite on Great Lakes yellow perch

I recently received an interim report from the State of Ohio on red worm infestation of yellow perch in Lake Erie. The report is very long and tedious so I want to summarize for you some of the information which I think is important. The description of the red worm parasite in our MDNR disease manual is largely outdated by this work.

First, the Nematodes or round worms, locally called "red worms", were positively identified as Eustrongylides tubifex. The genus Eustrongylides normally completes its life cycle in the proventriculus of fish-eating birds. E. tubifex was fed to domestic mallards and the red worms successfully matured but did not reach patency (females with obvious egg development). Later lab examination of various wild aquatic birds collected on Lake Erie showed that the red-breasted merganser is the primary host for the adult worms.

Next, large numbers of perch were (and are still) being examined for rate of parasitism and its potential effects. In addition to red worm, the Lake Erie perch sampled were rather heavily infected with a liver tapeworm, Trienophorus nodulosus and another body cavity nematode, Philometra cylindracea. An annual mean infestation rate of 77 percent was found for a 2,000-fish sample collected in 1978. These fish were infected with at least one of the three parasites. Red worm infections were found in 46 percent of the total perch sampled and there is evidence for a synergistic effect of multiple parasite species infections.

Red worms are usually seen by fishermen when in the perch's body cavity or flesh. The worms migrate from their normal position when the perch dies and its temperature exceeds 17°C. Presumably this is a normal red worm reaction when the host fish has been swallowed by a fish-eating bird.

Dave Weaver, Ron Spitler, Ray Shepherd
November 1, 1979
Page 2

Red worms first enter yellow perch in their third life stage when about 9 mm in length. The earlier two stages are unknown but the best guess is that they have an Oligochaete host rather than the more typical crustacean host. Once inside the perch, red worms evoke a host tissue reaction which results in growth of a cancerous tumor around the parasite. This tumor usually becomes benign when the parasite has reached full fourth stage growth (up to 93 mm long). These tumorous capsules are found in the mesenteries (85%), liver (10%), gonads (3%) and on the body wall. The body wall tumors are severely damaging to the perch and probably fatal which might account for their low representation in the total sample.

Laboratory studies showed that the red worms could live in yellow perch for at least 18 months and that already infected perch could be further infected by ingesting additional parasites. The field data also suggests that infestations of 6-8 red worms per individual perch are lethal, and I would say there isn't any question that infestations of parasites resulting in serious cancer growths would be physiologically damaging to the host. It is too early in the Lake Erie study to estimate the overall effects of these three parasites on the condition of yellow perch population, but they must be significant.

RCH:bb

Bob

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M - 4 1990

FISH HEALTH LABORATORY
Wolf Lake State Fish Hatchery
34270 C.R. 652
Mattawan, MI 49071

May 17, 1990

TO: All Fisheries Division Offices and/or Units

FROM: John G. Hnath, Fish Pathologist
Fish Health Laboratory

SUBJECT: Identification of Michigan's Redworm

Bill Bryant did some careful study of the common redworm of perch from Saginaw Bay, and concluded that there was some "problem" with the identification of the parasite. He sent samples of the worm to Dr. Glenn Hoffman (who wrote the book on parasites of fishes), and Dr. Hoffman identified them as Eustrongylides sp. This constitutes a positive identification from the nation's top taxonomist, and represents a change from what we have previously referred to as Philometra cylindracea.

You should make note of this, and change the wording in your copies of Fisheries Management Report No. 8, June 1977, Manual of Common Diseases, Parasites, and Anomalies of Michigan Fishes, by L.N.Allison, J.G.Hnath, and W.G.Yoder, on page 91.

See attached for more information.

cc: Westers
Bryant
file



MICHIGAN DEPARTMENT OF NATURAL RESOURCES

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November 19, 1980

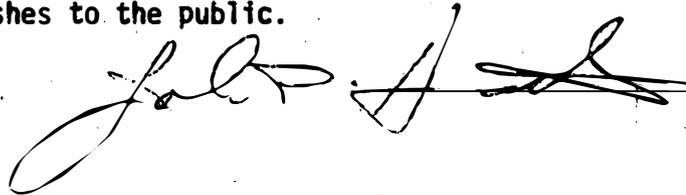
TO: HATCHERY AND FIELD BIOLOGISTS
FROM: JOHN G. HNATH, FISH PATHOLOGIST, WOLF LAKE STATE FISH HATCHERY
SUBJECT: FUNGUS INFECTIONS OF FISHES

Attached please find pp 55 and 56 as revised for your copy of the Manual of the Common Parasites, Diseases, and Anomalies of Michigan Fishes.

Also attached please find some additional information on the nature of fungal infections, as taken from Neish, G.A., and G.C. Hughes. 1980. Fungal Diseases of Fishes, Book 6 of Diseases of Fishes, S.F. Snieszko and H.R. Axelrod eds. T.F.H. Publications.

I think you will find the information interesting and informative, and that it will be helpful in explaining fungal problems of fishes to the public.

Your comments or criticisms are solicited.



FUNGUS INFECTIONS OF FISHES

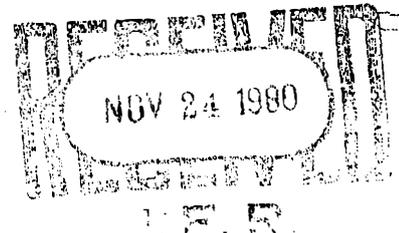
Although there are several genera of fungi capable of infecting fish, the vast majority of externally visible fungus infections seen in Michigan are caused by the genus Saprolegnia. Saprolegnian fungi are normal and ubiquitous in freshwater, and any body of water capable of supporting fish will have the fungi. Although fish are continuously exposed to these potential pathogens, the fish develop infections only when they are debilitated in a way that impairs the normal defense functions to a degree which allows infection to start.

With salmonids, there is good evidence that specific strains of Saprolegnia have a predilection towards parasitism of fish. This has been documented for Pacific Salmon in Canada. There is also a seasonal variation in inoculum potential, and the inoculum potential may be increased by the presence of infected fish in a given area.

"A reasonable, if unproven, assumption is that fish are, to a greater or lesser degree, continually challenged by potentially parasitic fungi. We are...therefore, forced to the conclusion that the major factor determining whether an infection is initiated is the condition of the host."

Under certain circumstances saprolegnia can act as the lethal primary pathogens, or as the most serious pathogen in a mixed infection.

Fungal infections are initiated by the spores only, not by the hyphae.



The fact that fungal infections of fish are frequently associated with wounds and lesions or handling infers that these fungi are wound parasites. And it is true that the integument and mucous both act to prevent infection through both physical and biochemical barriers.

Sexually mature fish are more susceptible to fungal infections than immature fish.

Various stresses, both external and internal, increase the susceptibility of fish to fungal infections. This can be demonstrated by application of Seyle's stress theory as described below and on the attached diagram.

Various stressors, and this could include a variety of both external and internal stimuli acting singly or synergistically, operate through the pituitary-interrenal axis to produce an increase in the level of plasma corticosteroids. Suitable stressors include crowding, injury, suboptimal water temperatures, handling, or the presence of noxious chemicals in sublethal concentrations. A fungal infection itself is also a stressor once it is initiated. An increase in plasma corticosteroid levels can impair the inflammatory responses and lead to an increase in corticosteroid-regulated protein catabolism and gluconeogenesis. This can ultimately lead to a protein deficiency which contributes to the wasting of skeletal muscle and leads to a decrease in antibody production and collagen synthesis. Lack of collagen, in turn impairs the ability of a fish to heal wounds and ulcers.

What we're describing here of course, is a normal metabolic pathway. The difference which would lead to the initiation of infection is quantitative, not qualitative, and would be associated with periods in the life of a fish during which, for some reason, there were especially high levels of plasma corticosteroids. These periods might be associated with chronic stress such as might be found, for example, in a hatchery where naturally aggressive and territorial fish are crowded together in unnaturally high concentrations. High levels of plasma corticosteroids might also be associated with the fishes' osmoregulatory function with the necessity to catabolize protein to obtain energy (as, for example, during periods of inanition), or with the inability of fishes to clear the hormone. In the case of Pacific Salmon, periods of especially high corticosteroid levels occur during the downstream migratory period, which is associated with the par-smolt transformation and during the upstream migratory period which is associated with sexual maturation.

Another important (and not unrelated) factor to be considered is the ascorbic acid metabolism of a fish. Fish in general, have a dietary requirement for Vitamin C. In the case of maturing salmon, these reserves become depleted because of inanition and the ability of the fish to repair tissue damage is greatly impaired at a time in their lives when they are quite likely to suffer damage to the integument. Such an explanation does not apply to young salmon, of course, unless they have not had access to an adequate diet, but in this regard it should be noted that an increase in levels of plasma corticosteroids will also cause depletion of ascorbic acid reserves. Presumably then, even if the salmon are being maintained on a marginally adequate diet,

under sufficiently stressful conditions, they could conceivably suffer from a de facto ascorbic acid deficiency.

In summary, we believe that there is a direct link between increased plasma corticosteroid levels in fish and their susceptibility to saprolegniosis. These higher hormone levels may occur in response to the physiological requirements of a fish at certain periods in its life (e.g. smoltification, sexual maturation), may be related to stress-induced increased in pituitary-interrenal activity, or come about as a result of both factors acting synergistically. As plasma corticosteroid levels increase, particularly if associated with a period of inanition, the fish become increasingly susceptible to infection and, at the same time, less able to maintain the integrity of their integument. This combination of factors renders them susceptible to infection by saprolegnian fungi, and other ubiquitous facultative pathogens. Variation in the stress response of different individuals, species, or populations of fishes to various internal and external stressors at different periods in their lives may well explain, to some extent, a number of the apparently contradictory results obtained by investigators who have carried out infection experiments.

Saprolegnia infections once started tend to be progressive and terminal. The fungal growth spreads outwards from the initial point of infection and as the infection progresses the fish becomes progressively weakened. The fungi are not tissue specific and are thus capable of attacking virtually any tissue. Yet the response of the fish to fighting a fungus infection is very slight once the infection has started. The fungi produce no toxins, and the damage done to the fish is directly related to tissue damage in the immediate area of the infection. The death of the host is a function of the growth rate of the fungus, the initial site of infection, type and quantity of tissue destroyed, and the ability of the individual fish to withstand the stress of the disease. The major ions in the serum of infected fish are all significantly reduced in concentration and it is suggested that the primary cause of death is osmoregulatory breakdown resulting in a lethal hemodilution. Severe hypoproteinemia and a significant reduction in the albumin to globulin ratio were reflected in the...infected fish.

There is not currently an ideal therapeutant for saprolegnian infections.

Fungus, or water mold is frequently observed on fish in Michigan, both in natural waters and in fish hatcheries. The appearance of grayish-white, furry or cottony-like patches is indication of a fungus infection. Several types of fungus are found on fish, and Saprolegnia parasitica is the most common one in Michigan. Water with a high silt or dirt particle content will mask the normal white fungus color to brownish or gray color as the particles collect on the fungus.

The fungus growth consists of a mass of filaments each of which is about 20 microns in diameter (1/5 the diameter of a human hair)(3). The fungus reproduces asexually by means of flagellated zoospores which are produced in enormous numbers in enlarged, club-shaped ends of the filaments. These zoospores swim actively in the water until they contact a fish. The fungus attaches to fish and fish eggs by means of small root-like filaments which penetrate the skin. As these filaments grow through the skin, they cause the death of the surrounding tissue and form large necrotic areas, the spread of which may eventually cause the death of the fish. Saprolegnia produce no toxins, and the damage to the fish results from direct tissue damage at the infection site. Virtually any tissue may be attacked and the time of death will be predicated by the type of tissue destroyed, the growth rate of the fungus, and the ability of the fish to fight the stress of infection.

Fungus attacks on fish may be primary or secondary in nature. Any physical injury, such as produced during spawning or migrating activity, or infection by external parasites may enable fungus to gain a foothold on the fish. Once the protective mucous covering of the fish is broken, an opportunity is afforded for the zoospores to germinate and penetrate the epithelium at the point of injury. (1). However, fungus seldom develops on strong fish, even in injuries. It develops rapidly in fish that have been weakened by stress such as spawning activity, disease, overcrowding, etc. It is not

unusual to see large steelhead and brown trout in streams during spawning runs with large patches of fungus. The fish are weak from the stresses of spawning activity, and in the case of the steelhead, they have come from a lake into a stream where the water chemistry is different, thus adding another stress. Most fish do not recover from the infection which tends to be progressive and terminal. Fungus among steelheads in spawning runs is not limited to Michigan streams, but also occurs in Wisconsin and streams of the Pacific coast (personal communication).

Control of fungus on fish eggs can be achieved by mechanical and chemical methods. Mechanical methods involve the removal of dead and infected eggs. Chemical control is less time-consuming, even with daily treatments. Formalin at 1:6000 dilution for 15 minutes daily is effective.

The treatment of fungused fish is not highly successful since the use of the most effective chemical (malachite green) was banned by the FDA. Formalin has been used on fish at a 166 ppm bath or constant flow for one hour daily as needed with limited success.

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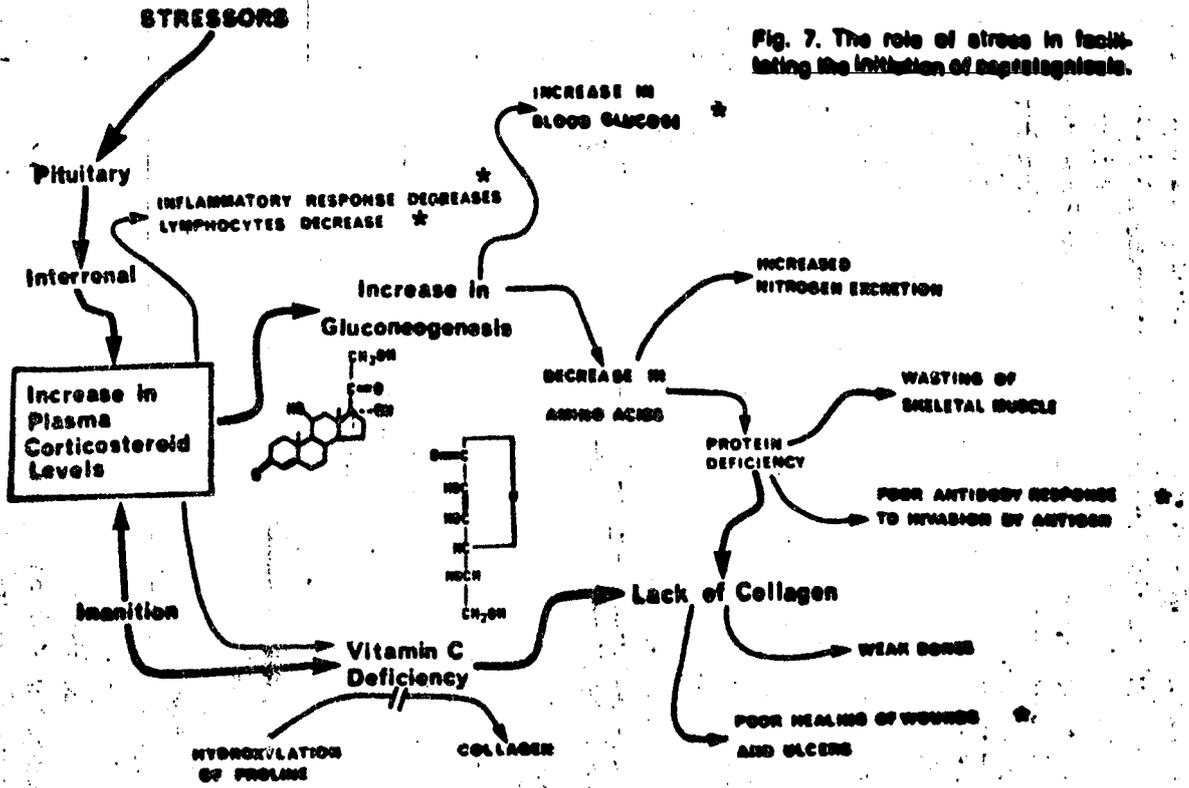


Fig. 7. The role of stress in facilitating the initiation of pathological.

* indicates those factors which reduce resistance to infection.

Fisheries Management Report No. 8

June 1977

MANUAL OF
COMMON DISEASES, PARASITES, AND ANOMALIES
OF MICHIGAN FISHES

by

Leonard N. Allison, PhD, John G. Hnath,
and Warren G. Yoder

MICHIGAN DEPARTMENT OF NATURAL RESOURCES
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ACKNOWLEDGMENTS

It is with deep appreciation and gratitude that the authors would like to acknowledge the assistance of the following people in the preparation of this manual:

Dr. Glenn L. Hoffman, Fish Farming Experimental Station, P. O. Box 860, Stuttgart, Arkansas, for the photographs of whirling disease.

Mr. Gerry Ream, Michigan Department of Natural Resources, Fisheries Division, for the cover design and line drawings of Ergasilus and yellow grub.

Mr. David G. MacLean, Michigan Department of Natural Resources, for his advice and critical review of the manuscript.

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MANUAL OF
COMMON DISEASES, PARASITES, AND ANOMALIES
OF MICHIGAN FISHES

INTRODUCTION

Fishing is a popular recreation in Michigan and many fishermen are intensely interested in the welfare of the fish. Consequently, the fish they catch are often scrutinized for anything unusual, and fisheries biologists, fish hatcherymen, and personnel of the pathobiology laboratory are consulted for identification of these conditions. Because fish, like humans, are attacked by a wide variety of bacteria, viruses, parasitic worms, tumors, and deformities, many inquiries are received every year.

Over the years a number of the more common afflictions of fish in Michigan were photographed by the staff of the Grayling Research Station. The illustrations should facilitate identification of the various parasites, diseases, and anomalies that affect Michigan fishes.

Since it would not be practical to include all known diseases of fish in manual form, this work was designed primarily to assist fisheries personnel to identify the common afflictions of fish in Michigan. Each subject is discussed briefly, including salient identification features, life cycles of parasites, and specific therapeutic measures for hatchery fish. References are included for those readers who desire to consult technical reports. The specific identity of many parasites, bacterial and viral diseases, and tumors can only be accomplished through complex laboratory techniques. Any unusual specimens not described in this manual should be taken to the fish pathobiology laboratory. Live specimens are most desirable, but fresh, refrigerated, or iced material is good, although material preserved in 70 percent alcohol or 10 percent formalin may be suitable in some cases. If there is any question about

the method of preservation, the pathobiology laboratory should be contacted for instructions. Valuable specimens may be destroyed through poor preservation.

All species of fish are vulnerable to invasion by parasites, depending somewhat on the habits of the species of fish and the type of lake or stream where they live. Many of the parasites that attack fish must live part of their lives in snails, so fish in shallow lakes with many aquatic plants, where most snails prefer to live, may be very heavily parasitized, and fish in deep lakes with few plants are lightly infested. In lakes that have deep water and weed beds in shallow areas, fish that prefer to live in shallow water may be heavily infested, while fish that prefer deep water may be lightly infested with parasites. Fish in streams that are cold and have few weeds have few parasites as compared to fish in warm, sluggish streams where weeds are abundant. The stage of the parasite in snails must have a certain degree of warmth for development.

Parasites usually do not destroy the animals they infest (hosts); if they did, they would soon eliminate their homes, and thus, themselves. Normally, most parasites do not visibly affect the health of fish, unless present in enormous numbers. One notable exception is the cataract worm. An intermediate stage of this parasite infests the crystalline lens of the eyes of fish, thus blinding it and making it easy prey for gulls, which are the final hosts where the parasite matures. Many people believe that parasites of fish may infest humans, thus they discard fish if a parasitic worm is observed. Actually, there are several parasites of Michigan fishes that are capable of infesting humans, but all are easily destroyed by normal cooking, smoking, or pickling practices. One parasite of the flesh of fish, the broad tapeworm of man (Diphyllobothrium latum), has been listed in textbooks as endemic in the Portage Lake area of the Keweenaw Peninsula. This information was taken from a study made about 40 years ago. A survey made in 1965 of fish from this area failed to find a single fish infested with this parasite.

Fish are no different than other animals where disease is concerned. A healthy animal is more resistant to disease than a weak animal, or an animal temporarily under stress. Fish are confined to living in water and are subjected to all the many natural and unnatural changes that occur. The body temperature of fish is not constant, as it is in mammals and birds, but is very close to the temperature of the water. Thus its metabolism is fast or slow, depending upon the water temperature. If the temperature changes very rapidly, physiological processes are drastically altered, sometimes causing death. Such things as low oxygen, excess silting, lightning, excessive current, supersaturation by gas, and pollutants of all kinds exert stresses on fish. If fish are not damaged directly by these things, they may be weakened, their resistance lowered, and they are then vulnerable to attacks by parasites, bacteria, fungus, etc.

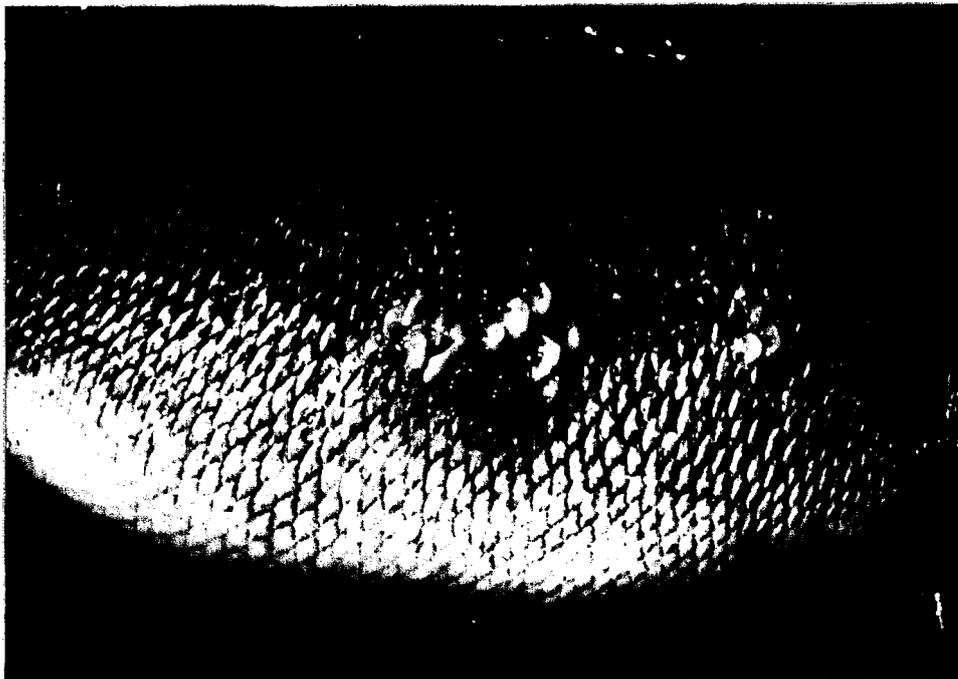
A certain terminology is used to describe various stages in the life cycle of parasites and other conditions relating to pathology. Some of these terms are defined below:

1. Parasite: A plant or animal living upon or within another plant or animal and usually at its expense.
2. Host: A plant or animal upon or within which the parasite lives.
3. Intermediate host: A host that harbors a larval stage of a parasite. Some parasites have one or more intermediate hosts before they reach the final host.
4. Final host: The host that harbors the adult, or sexually mature, parasite.
5. Cercaria: Free swimming, larval stage of a trematode. It is released from snails where it developed.
6. Metacercaria: Larval stage of a trematode, living in the skin, flesh, or vital organs of the intermediate host. It developed from a cercaria that burrowed into the fish and it becomes an adult when eaten by the final host.

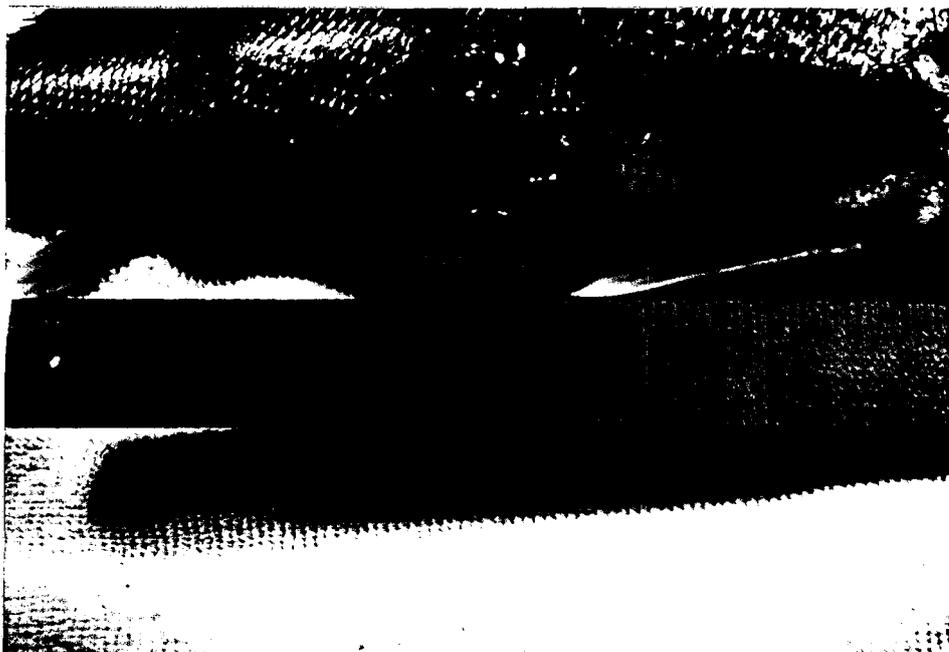
7. Plerocercoid: Larva of a tapeworm found in the flesh or vital organs of the intermediate host. When ingested by the final host it matures in the intestine of the final host.
8. Lesion: The easily visible or the microscopic changes which occur in the tissue and cells during disease, or as a result of injury.

Since the purpose of this manual is to assist the person with limited training in fish diseases to identify common afflictions of Michigan fishes, the diseases are grouped as to their location on the fish, either external or internal. Those diseases common to hatchery fish are so indicated and methods of control are included.

LYMPHOCYSTIS



Typical lesions of lymphocystis in walleye.



Dermal sarcoma lesions in walleye.

Lymphocystis is a virus-caused disease of the higher order of fish (Percidae and Centrarchidae). The incidence of the disease in walleye, Stizostedion vitreum, may be high in some locations and affected fish are discarded by fishermen.

The lesions of lymphocystis disease are raised nodular masses of generally light-colored tissue which superficially resemble warts. The wart-like growths are usually located on the skin or on fins but often may be restricted to a small area of a single fin (4). The growths are caused by virus infected cells which enlarge greatly. Color of the lesion is usually light and may be white, gray, or cream colored (5). There is a tendency towards opalescence, and larger lesions may show pink due to the vascular network. Lymphocystis cells may occur internally, but the infection is characteristically a disease which involves the skin (5).

Transmission of the virus is by the bursting and/or sloughing of host cells and release of the virus particles. This can occur intermittently through the duration of infection, or it can be massive as upon death and decomposition of the host fish (5). In temperate freshwater fishes, lymphocystis disease usually appears in the spring, reaches a maximum development during the summer and gradually disappears in fall and winter (2). There is some evidence that abrasion of the skin leads to increased incidence of infection (1).

There is no control of this disease other than the removal and destruction of infected fish.

A similar condition, also caused by a virus, is Dermal Sarcoma, and it may be found either separately or in conjunction with lymphocystis.

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COLUMNARIS



Fingerling coho salmon with Columnaris infection.
Note the lighter colored areas which are masses of
bacteria on the surface of the skin.

The symptoms of columnaris disease are easily recognized. An indication of the disease is the appearance of a grayish-white spot on some part of the head, gills, fins, or body, which is usually surrounded by a zone with a distinct reddish tinge (2). The spots superficially resemble lesions produced by Saprolegnia. However, the characteristic fuzzy appearance of a fungus infection is lacking. The columnaris lesions on different species of fish vary in size, location, and appearance. The typical lesions on coho salmon are on the body, usually originating at the fins (3). Lesions at the anal fin are most common, and frequently on other fins with some lesions originating on the head. The fin involved becomes frayed and the rays separate. Then a grayish-white area develops on the body at the base of the fin. This infected area rapidly expands, sometimes encircling the body before the fish dies (3). In some fish with severe caudal lesions, the entire tail structure disintegrates and some tissues of the caudal peduncle are exposed.

On fingerling rainbow trout the lesion usually originates on the back of the fish, progressing down each side and resembling a saddle.

The lesions on crappies are usually confined to the fins and gills and only rarely extend to the body (2). The lesions on bullheads appear as numerous small circular areas with sharp distinct outlines.

A scraping of material from a columnaris lesion examined with the aid of a microscope will reveal bacteria with a very characteristic appearance. They are long, thin, rod-shaped motile bacteria. The bacterial clumps or aggregates form columns or dome-shaped masses. (This is the basis of the name Columnaris.)

Salmon hatcheries of the west coast are concerned with two general strains of columnaris disease--high virulence and low virulence (5). Outbreaks involving the high virulent strains occur when the average water temperatures reach 60 F and outbreaks involving the low virulent strains occur when the average temperature approaches 70 F. Strains of higher

virulence appear to primarily attack the gills, while strains of lower virulence attack both body tissues and the gills.

Columnaris bacteria are found in various internal organs and the blood during disease outbreaks (5). It appears that the bacteria found systemically enter the blood stream through external lesions and are not directly involved in causing death. This is important because such bacteria are able to survive the usual external treatments and probably serve as a source of reinfection of the external tissues.

External infections may be treated with dips in copper sulfate 1:2,000 for 1-2 minutes or with malachite green 1:15,000 for 10-30 seconds (4). Diquat at a strength of 8.4-16.8 ppm for 1 hour for 4 consecutive days will control columnaris if given early in the outbreak (5). Terramycin, active, at a level of 4 g/100 pounds of fish per day in the food for 10 days is very effective and has the advantage of "clearing" the fish internally (5). Reinfection can occur as early as 4 or 5 days following treatment if the bacterium is still prevalent in the water supply and the water is warm.

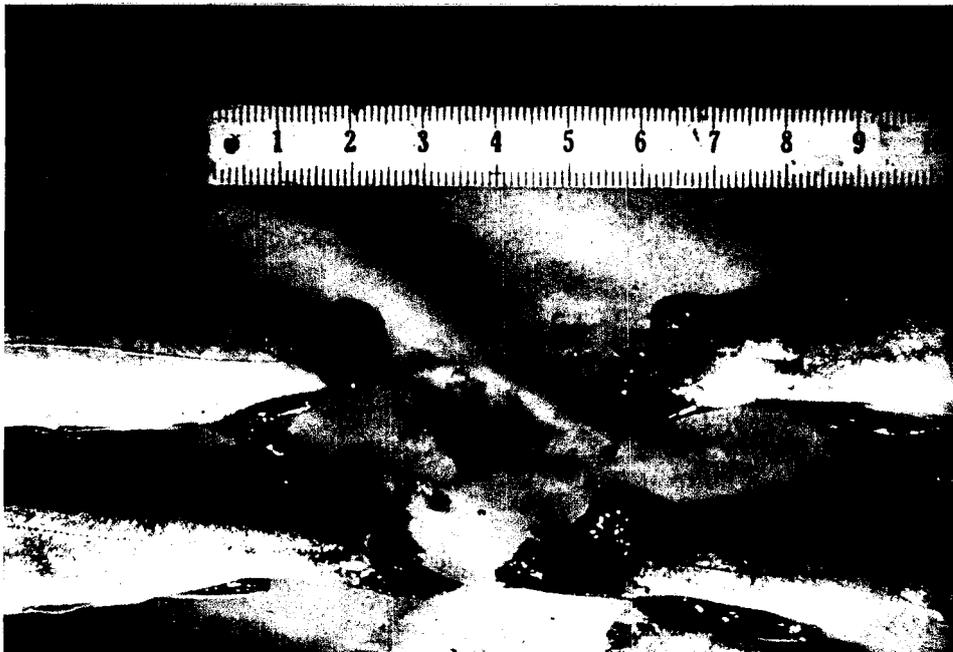
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FIN AND TAIL ROT



Tail rot on splake trout.



Close up of tail rot on splake trout.

These two diseases are discussed as one because of the similarity of symptoms and because both are probably caused by bacteria, the exact identity of which is not known. The symptoms of fin rot usually begin in the dorsal fin where the first sign of the disease is a noticeable white line along the outer margin of the fin. This white line is formed by a thickening of cells apparently as a reaction to the invading bacteria. As the disease progresses, the bacteria invade further toward the base of the fin, and the white line shows the degree to which the disease has advanced. The epithelium of the fin is destroyed and the fin rays are exposed, eventually becoming frayed and broken. Bacteria may invade the connective tissue of the fins, and destroy the cartilages at the base, causing the fins to drop off. In severe cases fish mortality may be heavy; in other cases mortality may be low and most fish recover and regenerate their fins.

Fingerling rainbow trout frequently show a light-colored thickening on the edges of the dorsal fins. If the margin of the fin is smooth and the fin rays are not exposed, fin rot is not present. Apparently, overcrowding and consequent fin-nipping causes this affliction which is not an infection.

Peduncle disease begins similarly to fin rot, but the adipose fin is first attacked. The infection progresses to the caudal peduncle where the muscles are destroyed by the bacteria, eventually leaving the vertebral column exposed and the tail still attached. The disease is always fatal.

No effective treatment has been found for peduncle disease. Leitritz (5) recommends a 1:2,000 copper sulfate dip, and Irwin (4) has shown terramycin to be very effective in aquaria treatments of bacterial fin rot.

Both of these diseases may be associated with unsanitary hatchery conditions or poor water quality in nature (2).

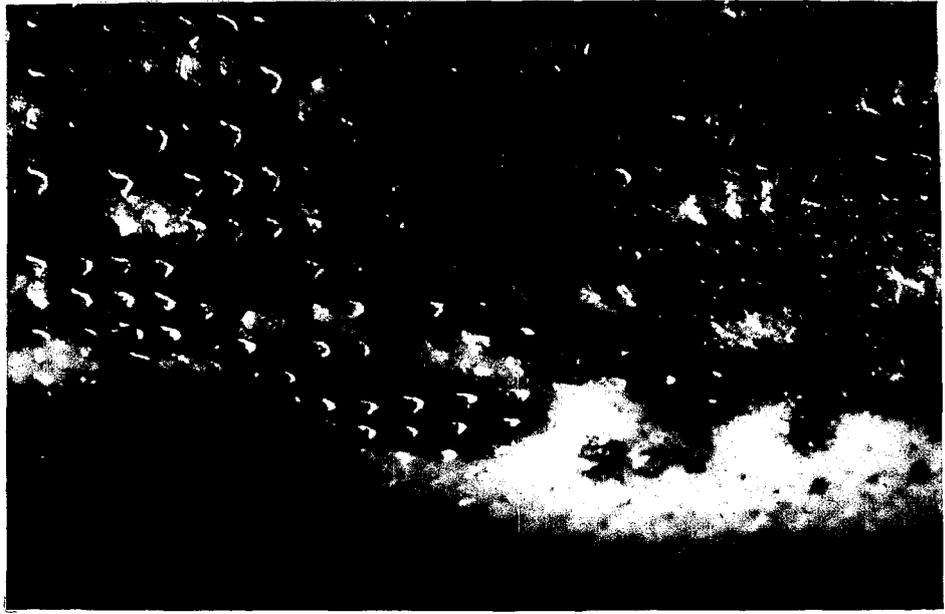
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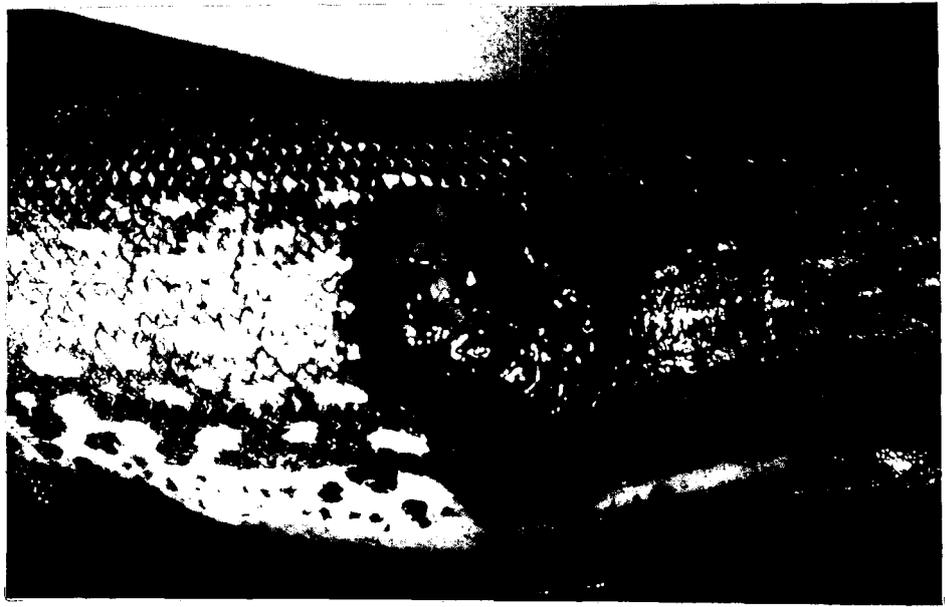
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RED SORE DISEASE

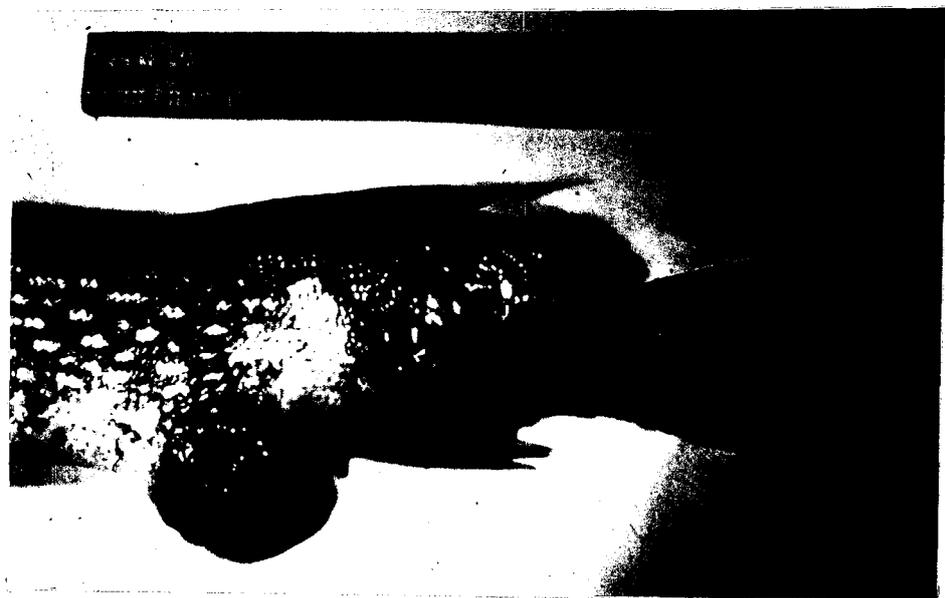
Early lesion of
lymphosarcoma
on northern pike.



Advanced lesion of
lymphosarcoma.



Multiple (terminal)
lesions of
lymphosarcoma.



A red sore disease of the pike, Esox lucius, has frequently been observed by anglers in Michigan. The disease is specific to northern pike and muskellunge in which it may reach an incidence level of 16% of the adult esocid population.

Actually two forms of a red sore disease may be seen. One form is a bacterial infection caused by bacteria of the Aeromonas hydrophila group which also cause infectious dropsy of carp, redmouth disease of rainbow trout, and red leg disease in frogs. As this condition is not often seen in Michigan, the reader is referred to the literature for more information.

The other form of red sore is viral in nature, and more properly referred to as lymphosarcoma. This disease is circumpolar in distribution in the northern hemisphere and widely distributed in the USA and Canada. Nearly all watersheds so far examined in Michigan have this condition. This disease is known to be fatal to muskellunge, but its effect on pike is not fully understood.

Lymphosarcoma is a tumorous condition which appears as welt-like sores on the flank, fins, or head. The tumor may appear as a cluster of pink blisters which may rupture, resulting in a sore resembling a lamprey scar. The tumor may also appear as a series of bluish blisters surrounding a "cream-like core." Or the tumor may most typically appear as what we have traditionally called "red sore."

Studies to date indicate that the disease is a contact-transmitted virus spread from fish to fish during the spawning act. The disease begins as a skin lesion which invades the underlying tissues and muscle. Two sequels are then possible: (1) the tumor may progress to involve internal organs and subsequent death of the animal, or (2) the tumor may regress and heal. The highest incidence of the disease is seen in spring, suggesting that the syndrome may cause death of most tumor-bearing fish during the summer months.

There is no evidence at this time that this disease affects humans or other animals. However, heavily infected fish are not considered very palatable and are being discarded by most fishermen.

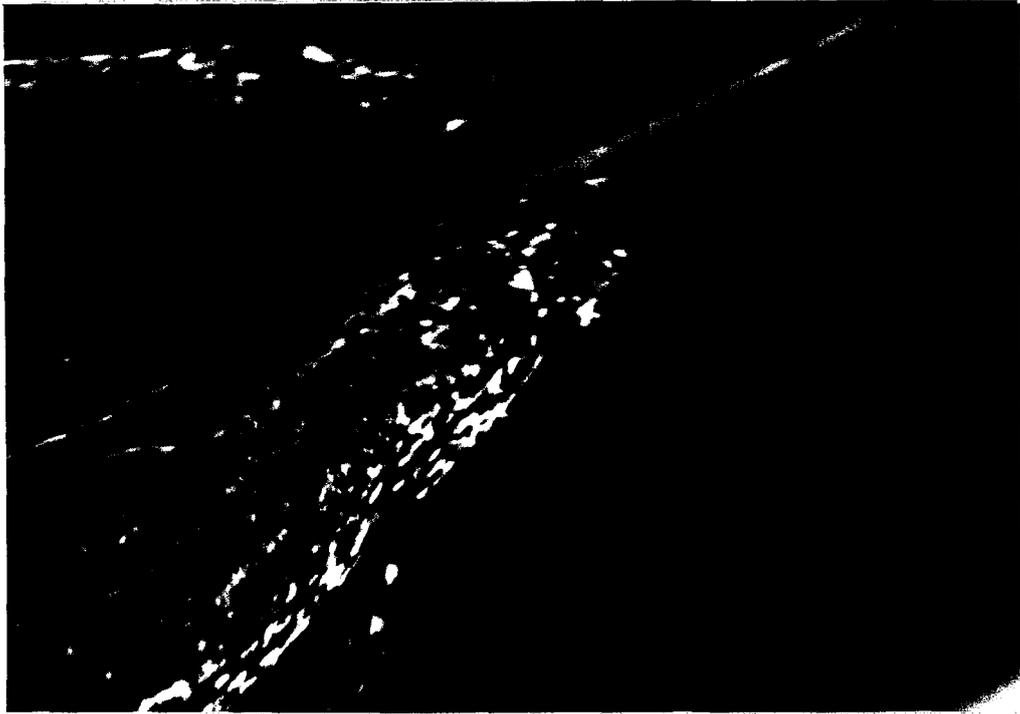
In general, the warmwater fish are most susceptible in the spring although cases have been observed during the winter months. The infection frequently occurs during the winter months, with the disease not breaking out until spring (2).

The best method of control for red sore is to avoid transfer of fish from waters which have the disease to waters free of the disease.

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CHILODON



Northern pike infested with Chilodonella sp.
Note the gray-white areas which are clusters of many parasites.

These ciliated protozoans are most frequently found on warmwater fish such as pike and carp, although infestation of trout fry in hatcheries is not uncommon.

The parasites are tiny, 50 to 70 microns long, and cannot be seen without magnification, although heavily parasitized fish may show blotchy gray areas on the surface of the skin. Under magnification the parasites may be seen as tiny, motile, oval bodies covered with fine cilia.

When Chilodonella occurs in very great abundance on a fish, particularly on the gills, it causes the fish to produce great quantities of mucous which impair respiration. Affected fish may become lazy, lie on their sides, rise to the surface, and eventually die. The parasite shows a preference for debilitated and undernourished fish. It is frequently observed on northern pike in the spring of the year as they enter a marsh for spawning. When they leave the marsh after spawning, the number of infected fish is considerably less and no infestations have been reported in Michigan during summer months.

The protozoan reproduces on the fish by simply dividing in half (binary fission), and has a resistant cyst which is formed to carry the parasite through unfavorable environmental conditions.

Chilodon can be controlled in hatchery fish by a 1:4,000 formalin bath for one hour. One treatment is usually sufficient.

These parasites are not harmful to man.

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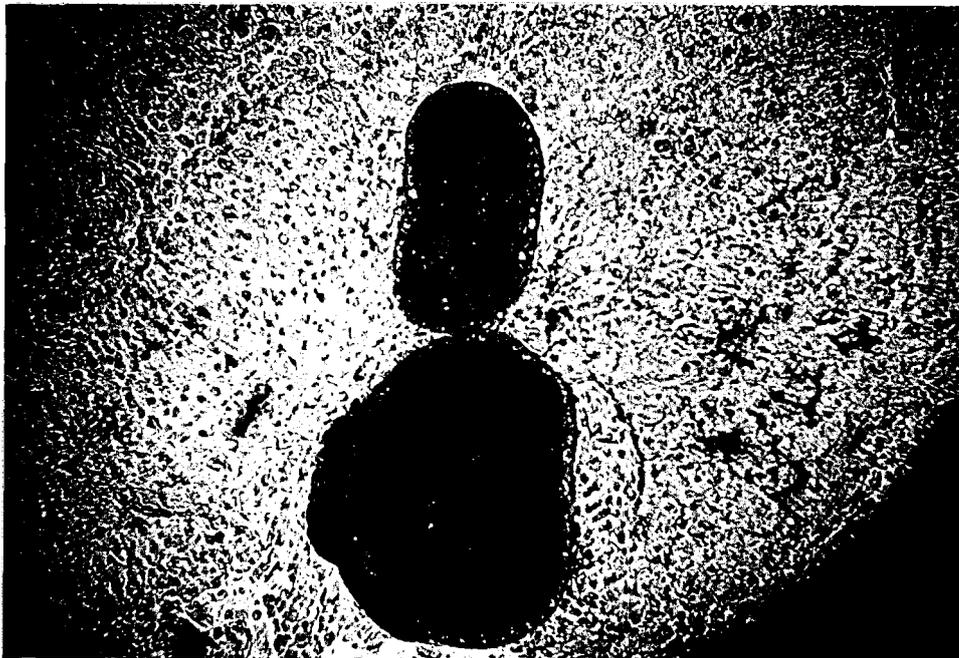
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ICH



Excised fin showing multiple tiny white spots of the parasite "Ich" (Ichthyophthirius multifiliis).



Photomicrograph of two live "Ich" specimens, greatly enlarged. Note the characteristic horseshoe-shaped nucleus.

A common disease of hatchery and aquaria fish is white spot, a condition caused by large ciliated protozoans. The adults of this parasite (Ichthyophthirius multifilis) are up to 1 mm in diameter, and may be seen with the unaided eye as tiny white spots on affected fish. The parasites live under the epithelial layers of the skin, fins, and gills of many species of fish, especially young fish. They are found more frequently on warm-water fishes than on fish from coldwater because low temperatures inhibit their activity.

When the parasite has grown to maturity it leaves the host and becomes enclosed in a cyst. Within this cyst multiplication occurs resulting in the production of from 400 to 2,000 young parasites. These are also ciliated, and when they leave the cyst, swim actively until contacting a fish. If a fish is not found within a few days, the parasites die. If they find a fish, they burrow into the skin, migrate for a time, then grow to maturity. The entire life cycle takes from 4 days to 3 weeks, depending on water temperature.

White spot can be very serious, causing high mortality, especially when fish are under crowded conditions and heavy infestation occurs.

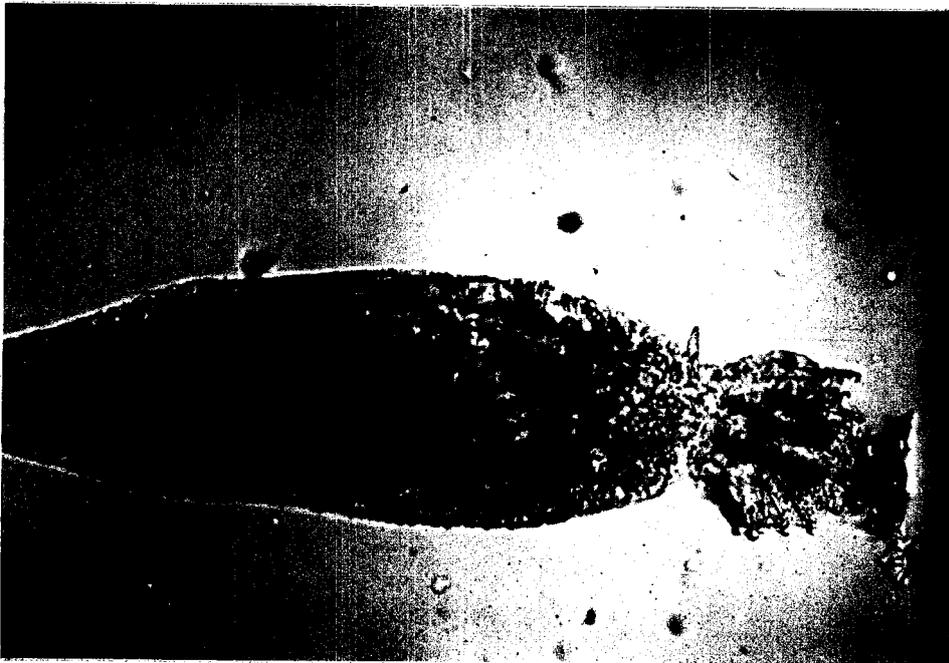
Since the parasite on fish is embedded in the skin, it is protected from the external treatments given for external parasites. The most effective control measure is to place parasitized fish in rapidly flowing water and hold there until all the parasites have disappeared.

The parasite is harmless to man, and is killed by normal cooking.

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"GYROS"



Photomicrograph of Gyrodactylus elegans showing hooks on posterior disc and hooks on embryo in body of adult parasite.

"GYROS"

GYRODACTYLUS ELEGANS, DACTYLOGYRUS sp.

Fish culturists frequently observe the symptoms of "Gyro" infestation. "Gyros," Gyrodactylus elegans and Dactylogyrus sp. seldom become a serious menace to fish in nature. However, they may become a problem in hatcheries where fish are closely crowded and the worms may be easily spread from one fish to another (1). Infested fish can be seen "flashing" as they rub themselves against the sides and bottom of the trough or stream in an effort to rid themselves of the parasites.

"Gyro" infestation can be identified only with the use of a microscope as the worms are quite small, 0.5 to 0.8 mm long (0.02 inch). The posterior end of these worms is disc shaped and equipped with hooks which are used to hold the parasite to the host. These hooks penetrate the skin or gill tissues, creating open sores which frequently become infected with fungus, and may serve as portals of entry for pathogenic bacteria and viruses.

Gyrodactylus may be distinguished from worms belonging to the genus Dactylogyrus by its absence of eye spots in the anterior end. Gyrodactylus may live almost anywhere on the host but it is usually most abundant on the fins, especially the dorsal and caudal. The frayed fins of heavily parasitized fish may also be an indication of infestation by "Gyros." The spread of infestation may be rapid as Gyrodactylus does not require intermediate hosts in its life cycle. "Gyro" gives birth to living young which are already well developed and immediately become attached to a fish host.

Dactylogyrus is easily identified by the presence of two pairs of eyes which appear as small black dots near the anterior end (2). This genus differs from Gyrodactylus by laying eggs, which become attached to the gills of the host. After the young hatch they require some time to grow to maturity. For this reason they do not multiply as fast as Gyrodactylus, however, the damage to the gills may be more injurious than fin damage. In addition, the eggs of Dactylogyrus may resist treatment and hatch later, so treatment must therefore be repeated to

kill the newly hatched young before they mature and lay eggs.
Effective control of "Gyrodactylus" adults and young is obtained by subjecting fish to a 1:4,000 formalin solution for one hour (1).

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BLACK SPOT



Black spot in rock bass. Note the darkly pigmented spots, especially around the eye and mouth. Each of these spots is an encysted larval trematode.

These parasites are easily discerned as obvious pigmented cysts (the size of a pinhead) slightly raised from the skin or fins, or sometimes in the mouth or flesh. The parasites commonly infest rock bass and other sunfish, bass, pike, perch, minnows, and other fish species. Various species of black spot are found in practically all parts of the world. The black spots are actually pigmented cysts of larval trematodes which mature in fish-eating birds. The life cycle follows the generalized account (snail to fish to bird) as given on page 94 for yellow grub, except that the bird host is the kingfisher.

Fish may be heavily infested, yet the parasites do relatively little damage in most cases and do not obviously affect growth or longevity of the fish. There is some evidence, however, that a massive infestation on a young fish may cause excessive blood loss, physiological stress, and even death.

These trematodes are incapable of infesting man, and even a heavily infested fish is safe to eat. It may be more aesthetically pleasing to skin a very heavily infested fish before eating. In any case, cooking kills the parasite.

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PARASITIC COPEPODS

Gill lice (Salmincola edwardsii) on gills of brook trout.



Close up of gill lice on gills. Note the egg sacs on the female parasites.



Salmincola siscowet on the skin of lake trout.



Brook trout from Michigan waters and hatcheries are often seen with small white or yellow, grub-like organisms attached to the gills, fins, or general body surface. The best known species of gill lice is Salmincola edwardsii which occurs only on brook trout in Michigan. Salmincola siscowet is found on the body of lake trout with other species of Salmincola occurring on other salmonid fishes (3).

Studies (1) of S. edwardsii on brook trout have indicated that rather heavy infestations, up to 125, do not appear to affect the growth of the fish. Mortalities have occurred among hatchery brook trout from 2 to 3 inches long when the fish are first attacked by these parasites. At this time extensive bleeding is noted from the gills. In these instances infested adult brook trout were in the spring pond that supplies water to the raceways holding the young fish.

The life cycle (S. edwardsii) is simple. The female, which is usually seen, bears a pair of long egg sacs within which the eggs undergo complete development. The young hatch and swim freely until they can become attached to a fish host. They die in about 2 days if they do not find a brook trout. On the fish they mate, the males die and the females develop into adults in about 30 days. The female produces a second pair of egg sacs 2 to 3 weeks after the first has hatched and dies shortly after these last eggs hatch. Adult gill lice are about one-fourth of an inch (6 mm) long. The complete life cycle may take from 1 to 6 months depending upon species and temperature.

The larva is able to rasp a hole in the surface tissue of fish by means of its mouth parts and attachment filament. The imbedded filament soon enlarges, anchoring its parasite firmly in place (3). Once imbedded, the organs of locomotion degenerate and all evidence of segmentation is lost.

The adult copepods are very resistant to chemicals, and to date no external treatment has been found to eliminate these parasites that will

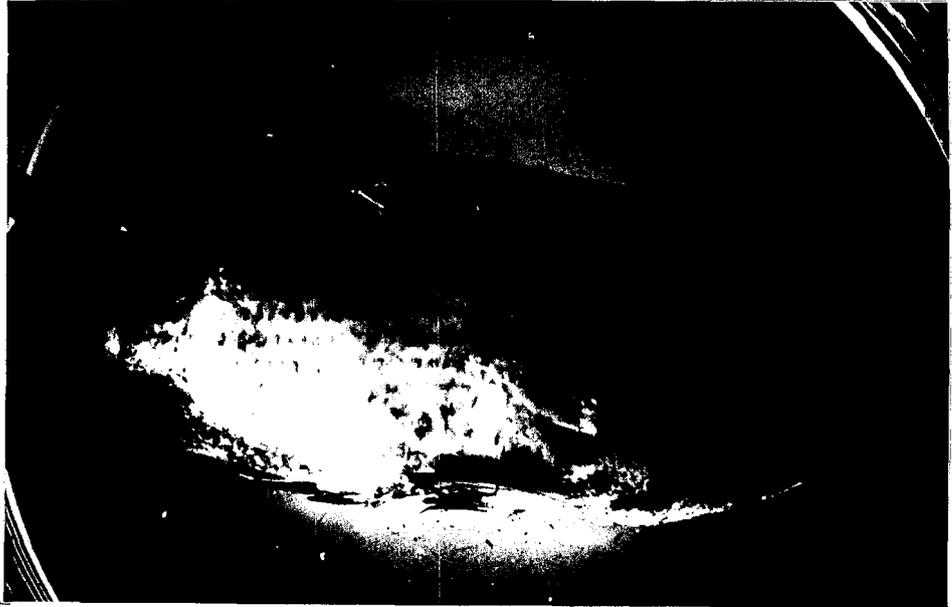
not also kill the fish (4). Infestation by this parasite can be avoided by using a water supply free of infested fish. If the water supply is free of infested fish, and fish in the raceways become infested, all fish should be removed, the raceways allowed to dry completely, then refilled and stocked with clean fish. Or, the water with diseased fish may be treated every several days with 1:4,000 formalin, or 2% salt (NaCl) for about 30 days to kill the delicate free-swimming young copepods before they become established on the fish.

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ANCHOR WORM

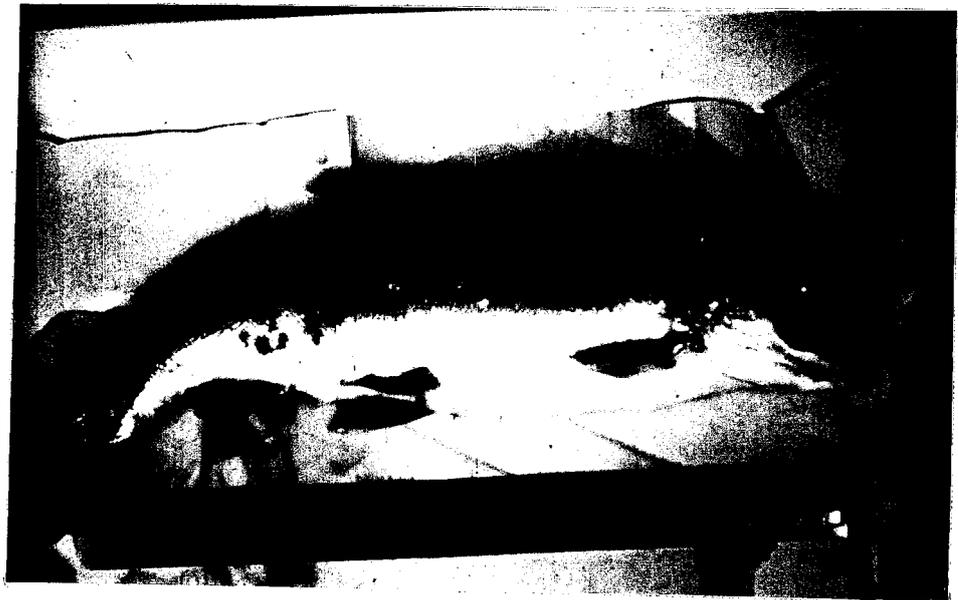
Lernaea cruciata on
rock bass.



Close up of Lernaea
on rock bass.



Lernaea sp. on
rainbow trout.



The genus Lernaea contains species of copepods parasitic on fish. L. cruciata is the most common in Michigan waters. Identification of Lernaea is based on the morphology of the adult female which is seen protruding from the skin of the host (4). It has a slender, wormlike body with the head imbedded in the flesh of the host which causes unsightly lesions (1). The embedded head bears branching processes that resemble antlers or an anchor, hence the name "anchor worm."

Species of Lernaea have life cycles characterized by an increase in complexity through molting of the hard, inflexible exoskeleton. The eggs hatch in 1 to 3 days, releasing larvae which are free-swimming. The larvae pass through five successive stages before the female attaches to a fish (2), where they penetrate the skin and attain a permanently fixed position. Then they increase in length up to 3/4 of an inch (22 mm), and develop the imbedding anchors (4). After reaching adulthood, egg sacs and eggs are formed completing the life cycle.

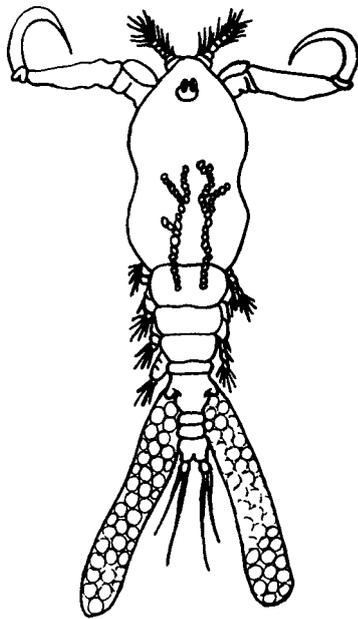
Early infections may cause the fish to swim about erratically, flashing against the sides and bottoms of ponds and aquariums. The parasites cause hemorrhagic and ulcerated areas at the point of penetration, spoiling the appearance of the fish. The main injuries are caused by loss of blood and openings in the skin which allow entry of secondary infections, such as other parasites, bacteria, fungi, and possibly viruses. Lernaea may occur at the base of the fins or scattered about the body surface. Occasionally it penetrates the eye and causes blindness.

Anchor worm transmission can occur through water supplies containing the free-swimming infestive stages or through the introduction of infested fish. Once Lernaea becomes established it is difficult to control. Prevention, more effective than control, can be effected by isolation of newly acquired fish, excluding fish from contaminated water, filtration, and filling ponds early to insure that the life span of the free-living period is exceeded (4).

References:

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ERGASILUS



***Ergasilus versicolor* (female)**
From Wilson, 1911

Copepods of the family Ergasilidae appear as small, elongated white spots on the gills of fish. Ergasilus, whose second antenna has been changed into a stout prehensile claw, adheres firmly to the gills of the host, destroying and digesting the epithelial covering of these organs. In heavy infestations its activity soon leads to secondary infection by bacteria and fungi and to the development of adhesions between the gill lamellae. Respiration is impaired, the fish refuse to feed, lose weight and their general health deteriorates seriously (3). Ergasilus has a wide range of host specificity, but tends to parasitize fish found in waters of intermediate and warmer temperatures (1). Ergasilus infestations can be particularly heavy among young, plankton-feeding fish in shallow waters.

Ergasilus is the only cyclops-like, parasitic copepod which bears egg sacs (1). The identification is based upon the females, as males are rarely seen. The female body narrows posteriorly and has a total length of 1/8 to 1/4 inch (1.5 to 2.5 mm), including egg sacs. The mouth parts are well developed and modified for biting.

Copulation occurs during the free-swimming stage, after which the male dies. The female then enters the gill cavity where she is retained by the gill rakers, then she creeps to the gills and attaches by the clasper-like claws (2). The eggs are laid in egg sacs where embryonic development takes place and the free-swimming larva hatches from the egg. Eggs are produced at intervals of 3 to 12 days, depending on species and temperature, and up to one million eggs may be produced during the 1-year life span of the female (1). The larva passes through four stages, accompanied by molting (2). The parasite overwinters as the mature female stage.

Control methods are as recommended for Salmincola (pp. 39 and 40).

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ARGULUS



Magnified view of the ventral side of two argulids.
Note the suction cups used for attachment to the fish.

Argulus is the only large (1/4 to 1/2 inch [6 to 10 mm]), long, external parasite, excluding the fish leech, that can move freely over the surface of the fish (1). These are large copepods and consequently, they are conspicuous objects on the fish that they inhabit. There are many species of argulids, one of the more common (Argulus appendiculatus) being found on largemouth bass, yellow perch, white crappie, and catfishes, among others. Fish with advanced infestations are characterized by erratic swimming, especially flashing, and reduced growth (1).

This parasite is easily recognized by the flat, leaf-like carapace (shell covering the outside of the body) which covers the whole animal, and the posterior heart-shaped incision (3). A small bilobed abdomen projects posteriorly. The anterior appendages are modified to form attachment organs for clinging to the host (2). The maxillae (pair of mouth parts) are united to form a stiletto which is retractable into a tube. The stiletto is long and sharp, well capable of penetrating the skin of fish (3). Lice prefer those parts of the skin best supplied with blood vessels like the mouth region, the operculum and the base of the various fins. The stiletto punctures the skin and the parasite sucks up tissue juice and blood. After a period of feeding they leave their host and swim about unattached (3). The feeding sites become ulcerated and hemorrhagic, providing ready access to secondary infections by other parasites, bacteria, fungi, and viruses (1).

Upon maturity the adult female leaves the host and lays eggs in masses on rocks, logs, vegetation, or other hard substances. The larvae pass through a number of stages until they reach their adult form. Argulids are parasitic from the time of hatching, leaving the host only to molt and reproduce.

Where Argulus is a problem, the following control methods will help if applied early enough.

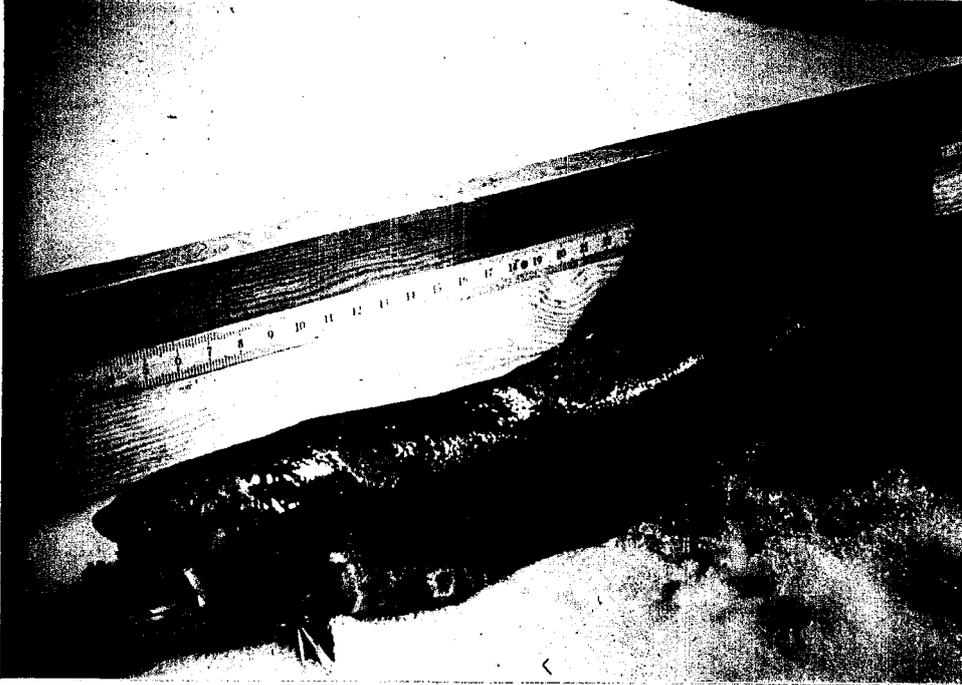
- (a) Removal of hard substances, submerged vegetation; tarring of concrete structures will prevent their use for egg deposition.

- (b) Lowering the temperature, or fertilizing to darken the water, will lengthen the duration of egg development.
- (c) Complete drying of ponds will kill eggs, larvae, and adults within 24 hours.

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FUNGUS



Severe fungus infestation on a mature steelhead rainbow trout. Such a heavily diseased fish has little chance of recovery.



Fungus on center fish (smelt); fish to the left and right are free of fungus.

Fungus, or water mold, is frequently observed in Michigan, both in natural waters and in fish hatcheries. The appearance of grayish-white, furry or cottony-like patches is an indication of a fungus infection, most likely by Saprolegnia parasitica (4). Several other genera and species of fungus are found on fish but S. parasitica is the most common one in Michigan. Water with a high silt or dirt particle content will mask the white fungus color to a brownish color as the particles collect on the fungus.

The fungus growth consists of a mass (mycelium) of nonseptate filaments (hyphae) each of which is about 20 microns in diameter (3). The fungus reproduces asexually by means of flagellated zoospores which are produced in enormous numbers in enlarged, club-shaped ends of the hyphae (1). Saprolegnia also reproduces sexually through the formation of egg-like oogonia, which, after being fertilized, develop into mycelia. The fungus attaches to fish and fish eggs by means of small, root-like filaments which penetrate the skin. As the filaments grow through the skin, they cause the death of the surrounding tissues and form large necrotic areas, the spread of which may eventually cause the death of the fish.

Fish eggs that die during incubation may become infected with fungus. Surrounding eggs may also be infected and die. Without control measures every egg can be lost to the expanding fungus growth. Saprolegnia cannot begin to develop on a normal, healthy egg unless there is some foreign organic matter adhering to the surface (1).

Fungus attacks on fish are considered to be secondary invaders. Any physical injury, such as produced during spawning, or migrating activity, or infection by external parasites may enable fungus to gain a foothold on the fish. Once the protective mucous covering of the fish is broken, an opportunity is afforded for the zoospores to germinate and penetrate the epithelium at the point of injury (1). However, fungus seldom develops on strong fish, even in injuries. It develops rapidly on fish that have been weakened by stresses, such as spawning activity, disease, overcrowding, etc. It is not unusual to see large steelhead and brown trout in streams during spawning runs with

large patches of fungus. The fish are weak from the stresses of spawning activity, and in the case of the steelhead, they have come from a lake into a stream where the water chemistry is different, thus adding another stress. Most fish recover from the infection when they return to original habitat and resume normal feeding habits. Fungus among steelheads in spawning runs is not limited to Michigan streams as it also occurs in Wisconsin and streams of the Pacific coast (personal communication).

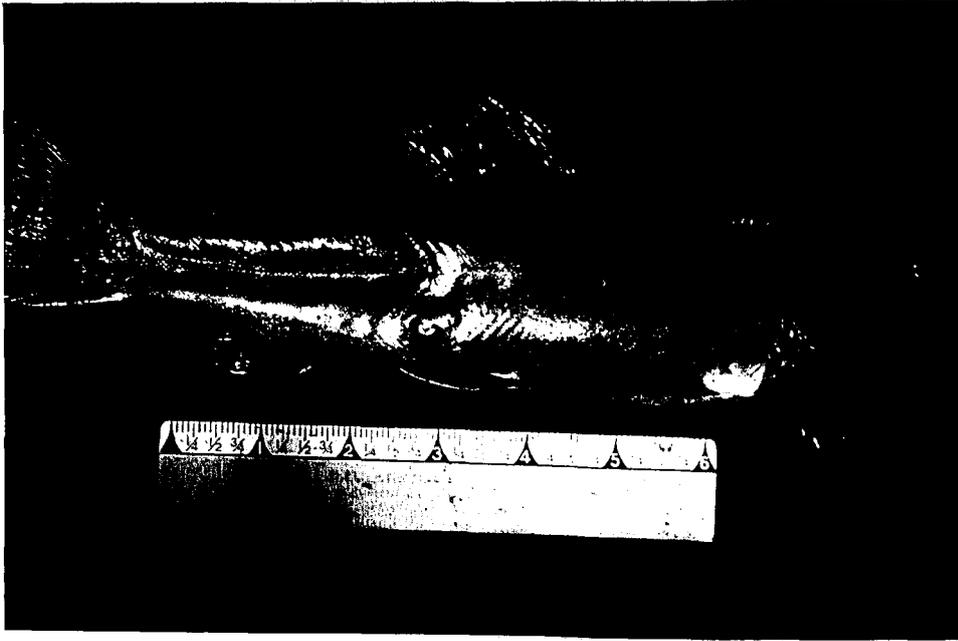
Control of fungus on eggs can be achieved by mechanical and chemical methods. Mechanical methods involve the removal of dead and infected eggs. Chemical control is less time consuming, even with daily treatments. A daily flush treatment with 3 oz of a mixture of 1.5 oz (40.5 g) zinc-free malachite green in 1 gallon (3.8 liters) water and the rate of water flow at 6 gpm can be used. Formalin at 1:600 for 15 minutes daily is also effective.

The treatment of fungused fish may be accomplished with an immersion of fish in a 1:15,000 dilution of malachite green, zinc-free, for 10-60 seconds (1). The success of the treatment is indicated by the color of the fungus when the fish (or fish eggs) are returned to clear water. The fungus should retain the green color. Care should be taken to accurately time the immersion as malachite green can be toxic to fish, especially minnows. Also, it may be toxic to rainbow trout, especially those longer than 3 inches. A few fish should be exposed to the chemical and observed for 24 hours before a large number are treated.

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FURUNCULOSIS



External blister of furunculosis on brook trout



Dissection of brown trout showing inflammation in the body cavity. Note hemorrhages on swim-bladder.

Furunculosis is the common name given to a bacterial disease that, under certain conditions, produces boils or furuncles on the skin of fish (3, 5). It reaches epidemic proportions only among trout and salmon, especially in hatcheries. The common name is misleading because the boils of furunculosis are quite different in structure from boils associated with humans. Also, the disease is really systemic in nature as it affects all parts of the body, especially the vital organs. It is not unusual for mortalities from furunculosis to occur without evidence of boils on the skin.

The causative organism is a bacterium, Aeromonas salmonicida, that is transmitted through the water and enters the fish through breaks in the skin and possibly the alimentary tract. It is carried to all parts of the body in the blood stream (1). When the bacteria multiply rapidly, they clog capillaries causing tiny hemorrhages, and escape to spread into surrounding tissues. When this happens in muscle tissues, the tissue is broken down and eventually forms a boil at that location or, if in vital organs, the fish dies. When capillaries are ruptured throughout the body, a generalized septicemia results. Among small fingerlings the septicemia usually causes mortality before the muscles are attacked and boils are formed. Internal symptoms are general inflammation of the body wall and lower intestines, small hemorrhages in the fatty tissue among the pyloric caeca, hemorrhages of the capillaries of the swim bladder, bright red spleen and swollen kidneys. Positive identification is made through bacteriological techniques.

Furunculosis is endemic in a number of Michigan lakes and streams, and fish mortalities are not unusual during periods of high water temperatures, although epidemics are not common. Mortalities from this disease among brook trout in natural waters are uncommon, perhaps because they prefer colder water than do brown trout. Mortalities from furunculosis among rainbow trout are rare as they appear to be relatively immune to this disease.

At hatcheries where fish are crowded in unnatural conditions, mortality can be quite high. However, furunculosis can be readily diagnosed

and controlled by therapeutic measures. In Michigan, sulfamerazine, 12 g per 100 pounds of food, fed for 3 consecutive days, followed by terramycin, 2.5 g per 100 pounds of fish for 4 consecutive days, has been effective in control of the disease in hatcheries. Longer treatments with sulfamerazine (1) and terramycin (6) have been recommended but, since none of the treatments eradicate the disease, and reinfection can occur at some hatcheries, our therapy is designed for control.

Another material, furoxone, at the rate of 1.14 g per 100 pounds of fish for 15 days has been successful (4).

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KIDNEY DISEASE



Both photos: Bacterial kidney disease in coho salmon. Note the large pus and bacteria-filled swellings on the kidneys of both fish.

Kidney disease in trout and salmon is caused by a bacterium thought to be a species of Corynebacterium. The disease is actually systemic but derives its name from pus-filled lesions in the kidney. In later stages various internal organs are affected, the body cavity may be filled with fluid, and lesions occur in the flesh and skin. Presence of the disease is verified by identifying the gram-positive diplobacillus in stained smears from lesions.

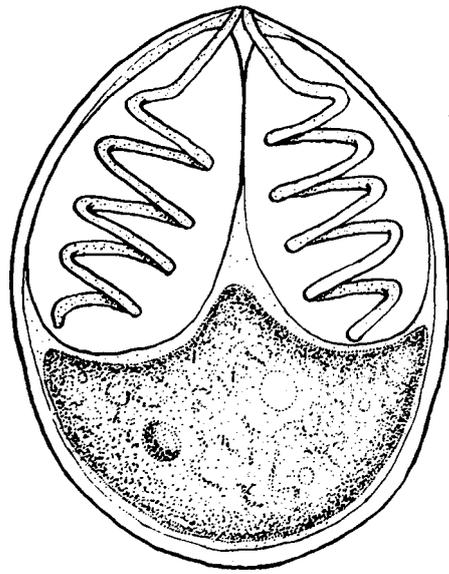
Although epizootics of this disease have been reported from natural waters, only a small percent of coho salmon in the Great Lakes have exhibited kidney disease during the lake phase of their life cycle (3). In hatcheries, however, it can cause severe mortalities among rainbow trout, brook trout, splake, and coho salmon. This can be a serious problem because completely successful therapy has not been found and the mode of transmission has not been definitely established. There is evidence that the bacterium may enter the skin through abrasions or injuries from ectoparasites, and by egg transmission.

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SPOROZOA



Myxosoma cerebralis

Sketch of a spore of Myxosoma cerebralis
from rainbow trout. •



Photomicrograph of stained spores of M. cerebralis.
Photo courtesy of Dr. G. L. Hoffman.

SPOROZOA

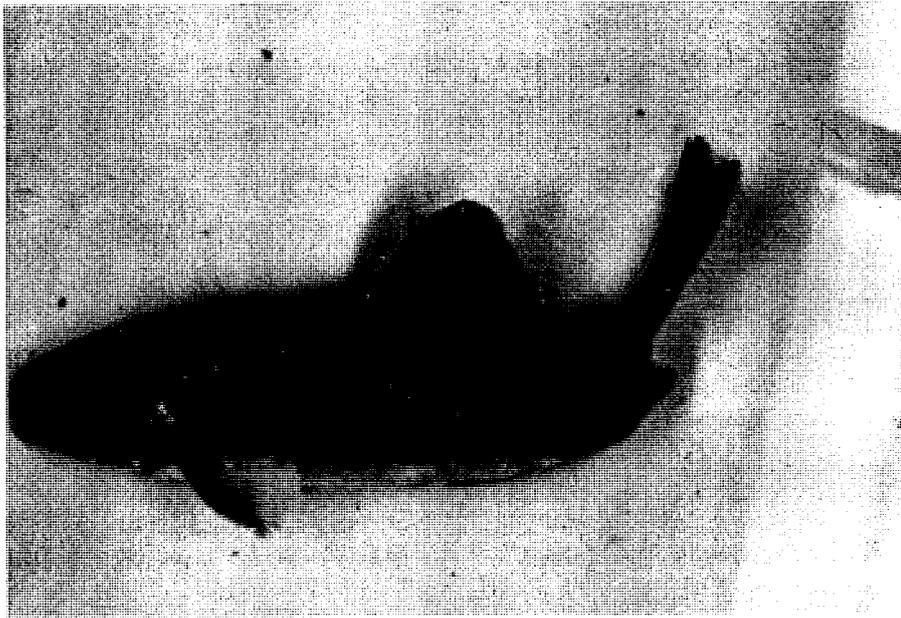
PROTOZOA

Myxosoma cerebralis, causative agent of whirling disease, attacks the cartilage of young salmon and trout, especially in the skull, and is a very serious problem among hatchery fish. The destruction of cartilage results in malformation of the head and curvature of the spine. In the head, damage to the auditory capsule, which contains the organs of balance, causes the fish to gyrate wildly with a peculiar whirling, tail-chasing motion, especially when they are suddenly frightened or attempt to capture food. Because of this behavior the disease is commonly called "whirling disease."

M. cerebralis originated in Europe and was identified in trout in the United States in 1956 in Pennsylvania. Since that time it has spread, through transfers of fish, to a number of states, including Michigan. Eradication is very difficult. The best method of control is to avoid transfer of infested fish. Since the spores withstand freezing, the parasite can be carried in fish for the market to various locations in the world and can infect natural waters if garbage containing uncooked heads or carcasses is discarded near natural drainage areas.

During the growth of the parasite much host cartilage is eroded and the skeleton weakened, resulting in the symptoms: whirling, black tail, gaped jaws, misshapen heads and trunks.

Trout infested during the first few weeks of life display the most severe disease symptoms because of the greater proportion of cartilage present in younger fish. The tail chasing, whirling, and black tail become evident at about 40 to 60 days after exposure. Small fish may become exhausted, and fall to the bottom of the pond until they regain their strength. Positive diagnosis of this disease is made by crushing or grinding cartilage from the auditory capsule with water to free the spores. Several drops of the slurry are then examined under a microscope at 450 \times . The spores are 10 microns in diameter. Those small fish which survive the early disease stages may show such permanent damage as sunken heads, misshapen jaws, and spinal curvature. When older fish



Trout infested with *M. cerebralis*, or whirling disease. Note the spinal deformities in both fish. Photos courtesy of Dr. G. L. Hoffman.

become infested they may not be seriously affected because the ossification of the skeleton prevents massive infestation (2). Such fish, however, may act as "carriers."

M. cerebralis has infested rainbow trout (Salmo gairdneri), brook trout (Salvelinus fontinalis), brown trout (Salmo trutta), and chum salmon (Oncorhynchus keta) (2), all salmonids native to North America. Rainbow trout are the most seriously affected by this disease, brook trout somewhat less severely, and brown trout may show no symptoms at all but may act as "carriers."

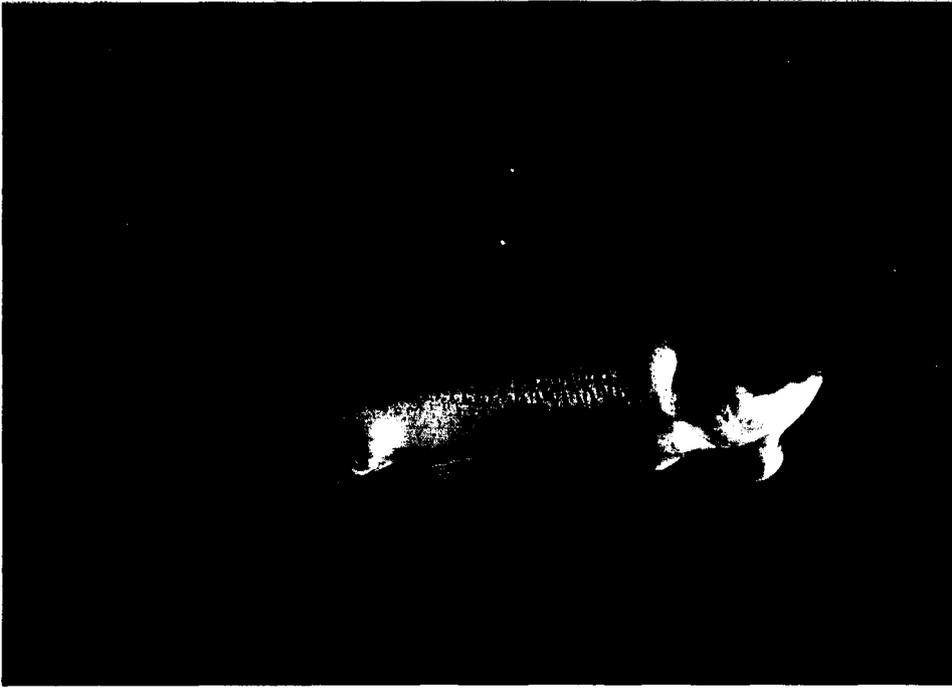
There is no proven chemotherapy for whirling disease. Current control measures are preventive: destroy all fish from infested ponds, drain ponds, apply a disinfectant and let stand a month or more. Fill the pond with water, drain, and repeat the treatment.

Species of other sporozoa have been found in nearly every kind of fish examined. Almost all tissues and organs of fish have been found parasitized, although each species of myxosporidian has its specific site of infestation in one or a few species of fish.

Large or small cysts may be formed on the body, gills, eyes, internal organs, or may cause severe curvature of the spine, nervous disorders, impaired kidney function, or other maladies in fish.

Since individual sporozoa are not visible with the unaided eye, microscopic examination is necessary to verify the identity of these parasites. Only when mature spores are found can a positive identification be made. Living spores seen under magnification (450×) are colorless and of a distinct form with oval, round, or pyriform shapes most common. Each spore has one to four polar capsules with coiled filaments, and each genus has a characteristic shape. References should be consulted for specific identifications.

The infestation is spread to other fish when an external cyst breaks and releases spores into the water. If the protozoans are internal, the spores are liberated after death and disintegration of the host or with feces. When a spore is ingested, it produces a motile form in the digestive tract. This form penetrates the gut, and migrates to the



"Tumors" caused by Myxobolus sp. on minnow.



Internal cysts of Glugea hertwigi in the body cavity of smelt.
Note: In all myxosporidian infestations, cysts as above are composed of thousands of individual microscopic parasites.

tissue of its choice where it grows and produces more spores which may become enveloped in host tissues to form a pustule or cyst.

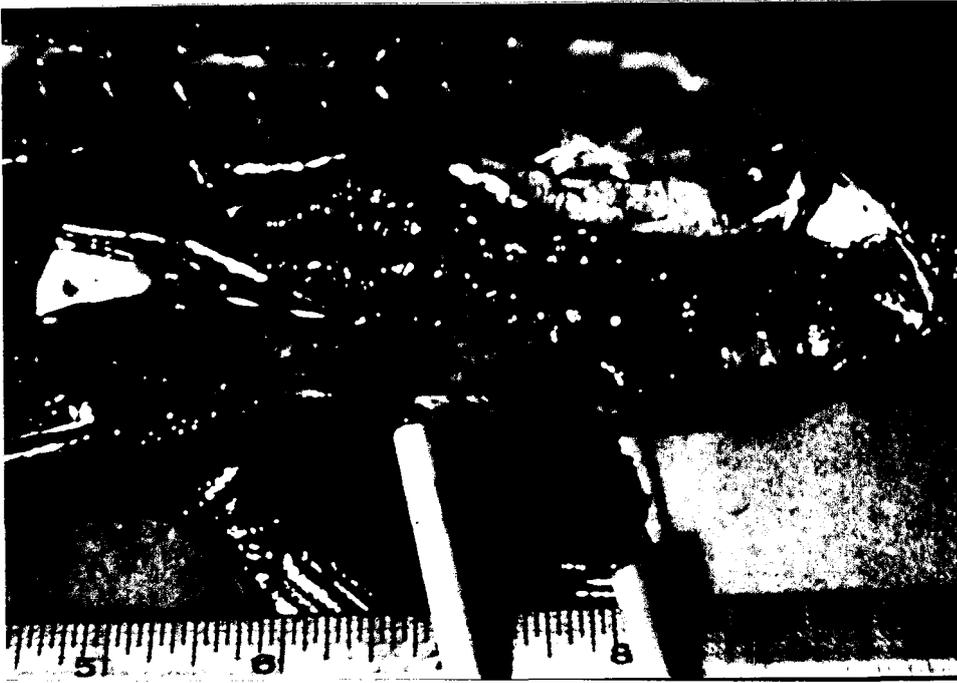
A sporozoan commonly found in the body cavity of smelt in Lake Erie is Glugea hertwigi. It occurs in white cysts and can cause severe mortalities. This parasite has been reported from eastern North America, Europe and the Far East. Sporozoans of fish are not known to be infective to man; as with other parasites of fish, they are destroyed by normal cooking.

References:

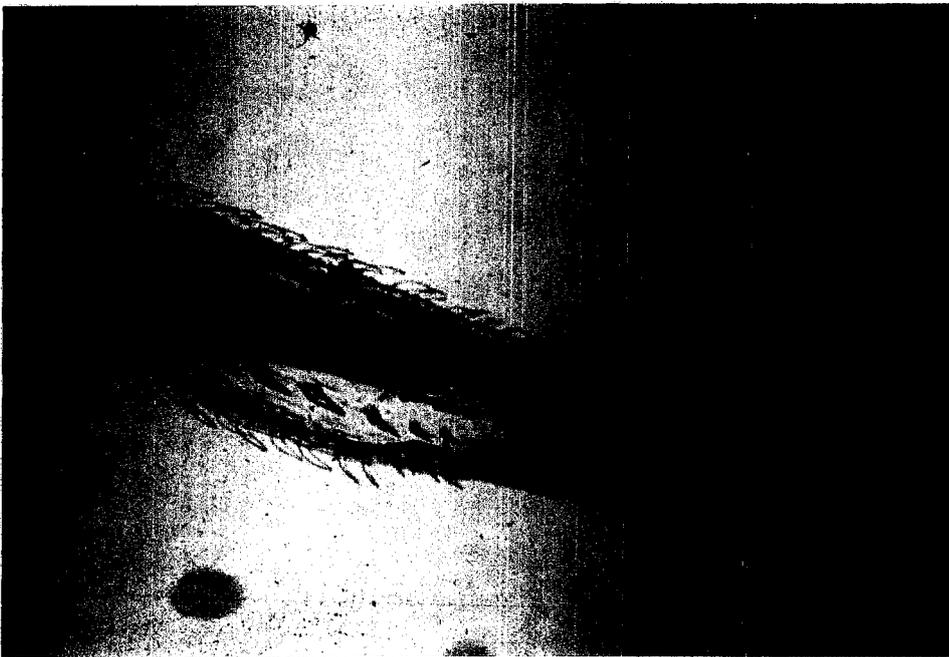
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ACANTHOCEPHALA



Acanthocephalans (Echinorhynchus salmonis) in the gut of coho salmon.



Photomicrograph of the proboscis of an acanthocephalan from smelt.

There are many species of Acanthocephala and many fish species the world over harbor these worms. Salmon, trout, ciscoes, whitefish, and smelt in the Great Lakes are commonly infested with these spiny-headed worms.

The worms parasitize the intestines of fish, and over 100 worms per fish are not uncommon. They range from about 1/16 inch (1.6 mm) to nearly an inch (2.5 cm) in length, and vary in color from white through yellow to dark orange. They can easily be recognized by the spiny nature of the proboscis as seen under magnification. If fish are not cleaned soon after death, the parasites may migrate from their normal site and invade other internal organs and the flesh. In fact, the orange-colored acanthocephalans in the intestines of smelt may burrow through the skin to the outside of the fish if smelt are caught at night and left in a tub until sometime the next day before the fish are cleaned.

Adult worms in the intestine of the fish host produce eggs. To complete the life cycle, the eggs pass from the intestine to the water outside, where they are eaten by an amphipod or other small crustacean. The larval stage migrates through the crustacean's intestinal wall to the body cavity where it forms the next juvenile stage. After several days of development, the larva is ready to infest a fish when eaten. The larva invades the intestine of the fish, inserts its spiny proboscis into the gut wall and develops to maturity.

Acanthocephala of fish are not capable of infesting man, and since most fish are eviscerated prior to eating, the presence of these parasites does not make a fish undesirable to eat. In any case, the parasites are killed by cooking.

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LIGULA



One ligula parasite removed from the body cavity of a small sucker.

This tapeworm of birds is found in fish as a very large larva (plerocercoid). Many species of fish are infested with this parasite; common shiners and white suckers being common hosts for Ligula. The larval worms are found in the body cavity of the fish hosts where they are coiled around the viscera. Often several worms may be found in one fish. Regardless of the number of worms in a fish, the total volume of the parasites may be 25-50% of the total volume or weight of the host (2). In larger hosts the worms are usually larger, sometimes twice the length of the host.

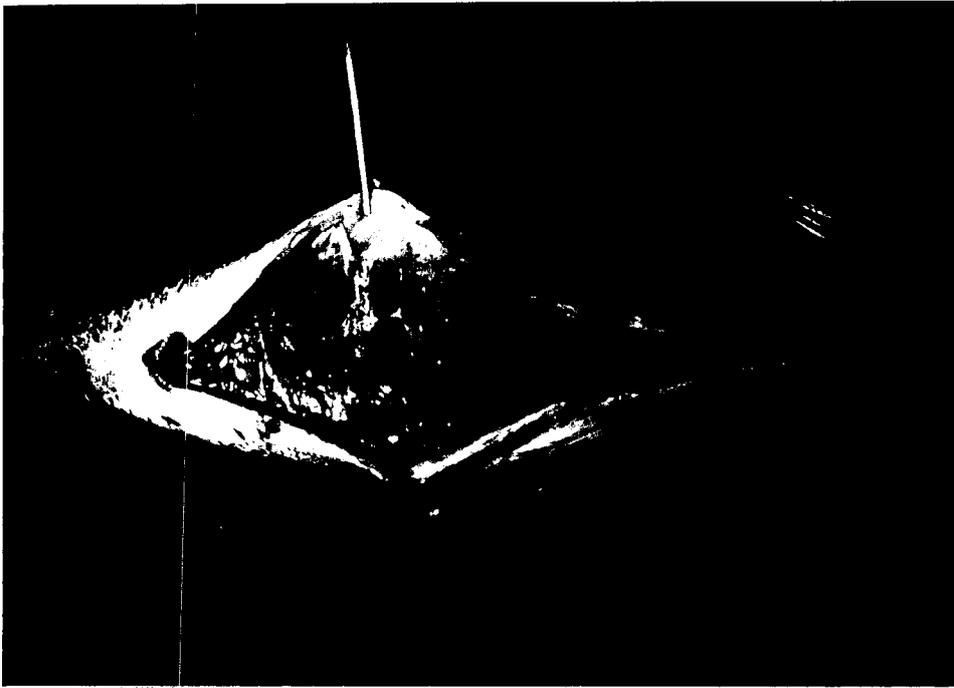
With such a parasite burden, infested fish are usually readily identified by the swollen abdomens, and sluggish movement. In fact, infested fish may not be able to keep up with their uninfested neighbors and may school together in groups of less than a dozen (2).

When infested fish are eaten by American mergansers or great blue herons, the plerocercoids attain sexual maturity in a few days, produce eggs for several more days, and die. The eggs which reach the water are taken up by small crustacea which in turn are eaten by fish and the life cycle is completed.

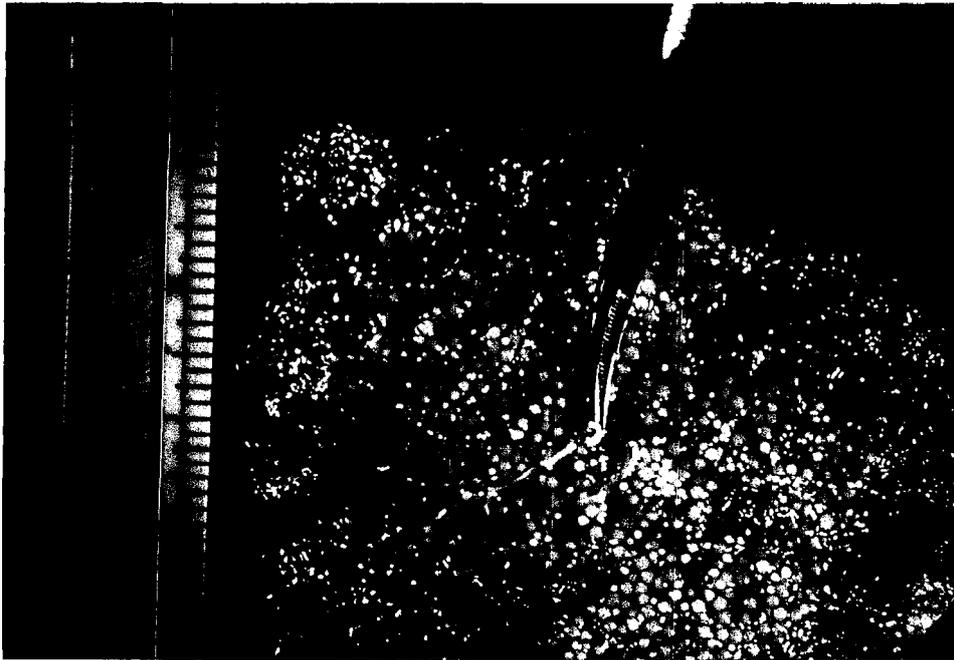
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BASS TAPEWORM



Note the brown color and adhesions in the body cavity of a smallmouth bass: caused by larval bass tapeworms.



Note the larval tapeworm at the tip of the forceps: This is the plerocercoid or larval stage of the bass tapeworm, in the ovaries of a bass.

The adult tapeworms are found only in the intestines of largemouth and smallmouth bass. The plerocercoid larvae, however, are found in the body cavity and internal organs of many species of fish, especially rock bass and large and smallmouth bass in many lakes and streams in Michigan. It is the larval plerocercoid stage which is most often seen, and which causes damage to fish. The plerocercoids develop in the body cavity and internal organs, especially the liver and ovaries. Because they do not encyst, but continue to move around, they destroy tissue and cause multiple tiny hemorrhages. This produces a brownish color and adhesions in the body cavity. Heavy infestations in the ovaries may sterilize the fish.

The life cycle of this tapeworm involves a larger bass eating a smaller fish (intermediate host) infested with the plerocercoids (1, 3). It has been demonstrated (2) that the plerocercoid may also migrate from the body cavity directly into the gut, thus omitting an intermediate host. These larval tapeworms adhere to the intestinal wall of the larger fish and grow to maturity. Eggs produced by the adult worms pass into the water where they are fed upon by various smaller crustaceans such as copepods and amphipods. Inside these invertebrate hosts a larval form emerges from the egg, penetrates into the crustacean's body cavity, and develops into a proceroid. When an infested crustacean is ingested by a small fish, the proceroid emerges, burrows through the intestinal wall of the fish, and migrates into visceral organs where it may cause extensive damage as a plerocercoid. The plerocercoid may live several months in the internal organs of a fish.

The bass tapeworm will not infest humans.

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SWIM BLADDER WORM



Cystidicola stigmatura in the swim bladder of smelt.

This roundworm is a common parasite of trout, salmon, smelt, and whitefish. As an adult the worm is found in the swim bladder of the above fishes, often in great numbers. Anthony (1) found over 200 worms per fish, and Warren (2) found 72% (144 of 200) of Lake Superior ciscoes infested with Cystidicola.

The adult worms found in the swim bladder of fish are from 1 to 2 inches (2.5 to 5.0 cm) in length, and usually a translucent white in color. They produce eggs which eventually reach water and are ingested by crustacea. Here the juvenile worms develop to a stage infestive to fish. When the infested crustacea are eaten by a suitable fish, the larval nematodes are freed and migrate to the swim bladder. Here they grow, mature, and produce eggs thus completing the life cycle. It seems probable that larger fish, such as lake trout which do not feed on crustacea, develop heavy infestations by consuming smaller fish which have eaten infested crustacea and still have invasive juvenile worms in their guts.

The worms apparently cause little harm to the fish hosts even when in large numbers in the swim bladder. Infested fish appear healthy, but no studies have yet been reported dealing with the effects of this parasite upon its host.

The parasite is not harmful to man. Since the swim bladder is discarded in dressing the fish, the parasites are not objectionable in fish prepared for human consumption.

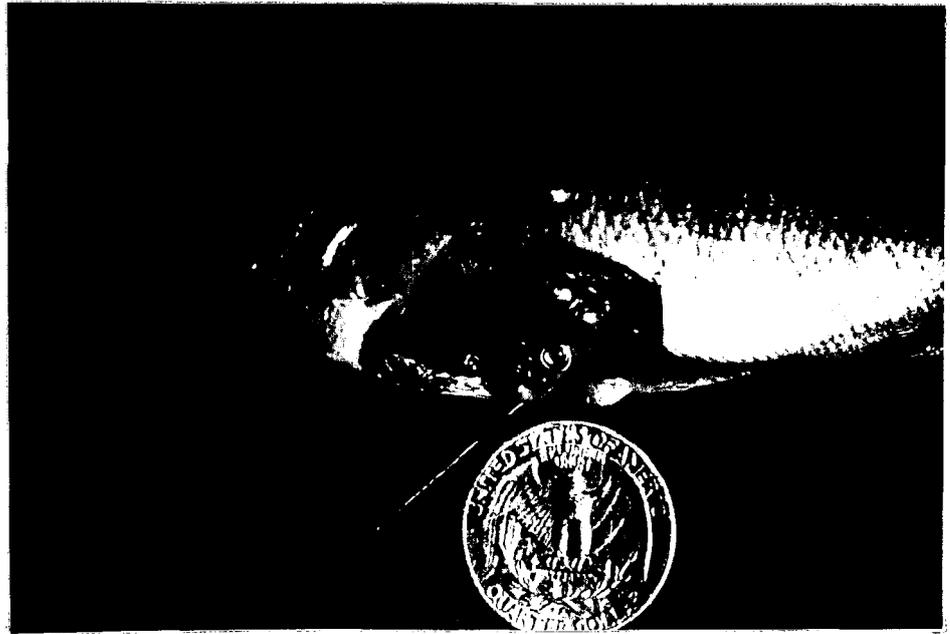
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RED WORM

Red worm encysted in the body cavity of a perch. Note the reddish worm at the tip of the pointer.



Enlarged view of an encysted worm in the fatty tissue of a fish.



Red worm leaving its cyst and penetrating the body wall of a perch.



This roundworm (nematode), commonly called the red worm, is abundant in yellow perch of Lake Huron and western Lake Erie, but is also found in other localities. As high as 86% of fish examined from areas of Lake Huron have been found infested with as many as 78 worms per fish (1), although it is usually found in lesser abundance.

This parasite is pink to red in color, very slender, and may reach 2 inches (5 cm) in length. It is usually found encysted within the body cavity of the fish, but in very heavy infestations, or in fish not dressed quickly upon capture, the worms may move out of the cysts and be found free in the body cavity or even in the flesh.

The life cycle of the red worm of perch is not known but it is believed to be similar to the closely related species (P. nodulosa) found in the common sucker (4) which use various species of a crustacean (Cyclops) as an intermediate host. In this case, larvae developing from eggs of the adult female worms in fish escape into the water and are eaten by species of Cyclops. Within the copepod the larvae attain a certain growth stage, and remain in this state until eaten. When a fish feeds on the copepod, the larval worm leaves the crustacean, grows, and migrates through the tissues of the fish, eventually encysting in the body cavity.

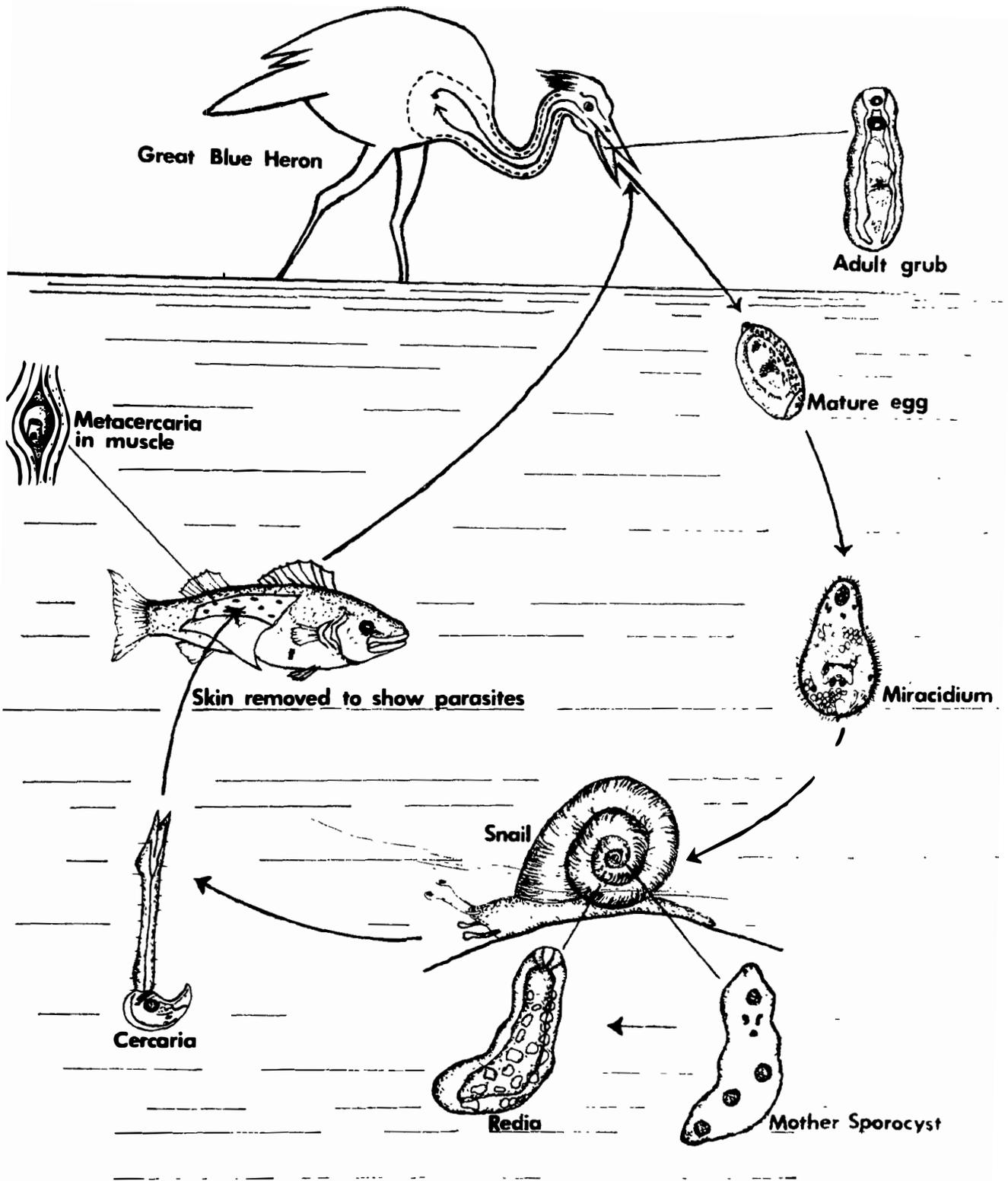
Although this parasite has not been implicated in mortalities of fish, heavy infestation must certainly affect the normal growth and vitality of such fish. These nematodes are incapable of infesting man, regardless of how objectionable a parasitized fish might be aesthetically. As with all parasites, thorough cooking kills this worm.

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YELLOW GRUB



Life Cycle of Yellow Grub *Clinostomum marginatum*

Modified from Hunter and Hunter, 1935

This is the common "grub" found in our freshwater fish as a yellow worm up to 1/4 inch (6.4 mm) long just under the skin, or in the flesh. Yellow grub has been reported from so many kinds of freshwater fish in North America that apparently no fish is immune to it. The grub is the larval stage which must be eaten by fish-eating birds, such as herons and bitterns, to develop. The grub matures in the throat of the bird, and eggs wash into the water from the bird's mouth when feeding. The eggs hatch and the first larval stage (miracidia) swim by means of fine hair-like cilia until they find a snail of the genus Helisoma. Unless they find this snail they die within several hours. In the snail they go through several developmental stages during which they multiply a thousand-fold, finally leaving the snail as free-swimming cercariae. Unless the cercariae find a fish within a few hours, they die. When they find a fish, they burrow through the skin and encyst, where they develop into metacercariae, which are the yellow grubs. There they remain until eaten by the bird host, thus completing the life cycle.

The grubs may live for several years in the fish, thus in many lakes rather heavy infestations accumulate and the fish are classed by fishermen as unfit for food. It is possible that yellow grub may kill fish under some circumstances, but normally a fish is not noticeably affected by the parasite.

Normal cooking of the fish destroys the grub and the flavor of the fish is not altered.

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Yellow grubs beneath the skin of the tail of a yellow perch.



A perch with fillet removed to show the yellow grubs in the flesh. Note that some of the worms were encysted during filleting, and have assumed an elongated form more commonly associated with a "worm."

CATARACT WORM

The metacercaria or last larval stage of this worm infests eye lenses of fish. In hatchery situations where crowded fish might become exposed to this parasite, very heavy infestations can occur resulting in blindness of many fish. The blindness is caused by opaque areas in the lens formed wherever a metacercaria is situated, and when many parasites are present the entire lens appears white in a living fish. Over 100 metacercariae have been recovered from a single infested trout lens.

In nature* LaRue (5) reports as high as 450 metacercariae in the eyes of a single yellow perch and 135 in a single white sucker. He reported that in Douglas Lake, Michigan, 80.6% and 94.3%, respectively, of the above fish surveyed were infested with eye metacercariae.

In order to continue the life cycle, the infested fish must be eaten by a gull. Then the general pattern is followed as outlined for yellow grub (p. 94).

Recent investigations show that fish may also become infested by eating snails harboring the larval stages, thus the second swimming larval stage may not be necessary. The eye forms of the parasite may live several months and remain infestive to gulls. The completion of the entire life cycle requires about 4 months under favorable conditions.

There is no method of treating infested fish; the only practical method of controlling the disease is through elimination of the snails which serve a necessary stage in the life cycle of the parasite. Copper sulfate is a good chemical to kill snails. Dissolved at 20 ppm in hatchery ponds from which the fish have been removed and left standing overnight, it is very effective (3).

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METACERCARIAL CYSTS



A heavy infestation of metacercariae in the liver of a pumpkinseed sunfish. In this photo the liver forms the center of attention, and within this organ note the numerous white objects, each of which is a larval trematode.

Tiny colorless or white cysts are often seen in the liver and heart, and in other tissues of many kinds of fish (1). These cysts contain larval trematodes which grow into adults in the intestines of fish-eating birds or mammals. The life cycle follows the general pattern as given for the yellow grub (p. 94).

Very heavy infestations may occur, but unless the parasite burden is exceptionally massive, there is usually no apparent harm done to the fish.

The majority of these parasites are incapable of infesting man, and all are killed by thorough cooking.

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LEECHES



Leeches on the tail of a yellow perch. Note marks where other leeches were attached.

Certain leeches or "bloodsuckers" attack fishes, but do little damage unless present in large numbers. The damage done to the fish is proportional to the number of leeches present and the amount of blood they remove (1). Leeches attach periodically to fish, take a large blood meal, and leave for varying periods of time.

The true fish leeches belong to the family Piscicolidae and are related to the common earthworm (2). Leeches usually have a greenish brown color, are from 1/4 to 1 inch (5 to 25 mm) long, and may be found in the mouth, on the gills, fins, or body of bluegills, perch, and many other fishes. All leeches are composed of only 34 true segments with each segment subdivided into a definite and constant number of superficial annuli (rings) (3). Leeches have two suckers, one at each end. The anterior one surrounds the mouth and may be large or small, and lip-like. The caudal sucker faces ventrally and is much larger, disc-like, powerful, and expanded over a central attachment pedestal (3). Leeches are flattened dorsoventrally and are highly muscular and contractile. The body outline may vary greatly with locomotion. Lampreys, parasites that also attach to the skin of fish and may be confused with leeches, cannot contract or elongate their bodies and they have only one sucker.

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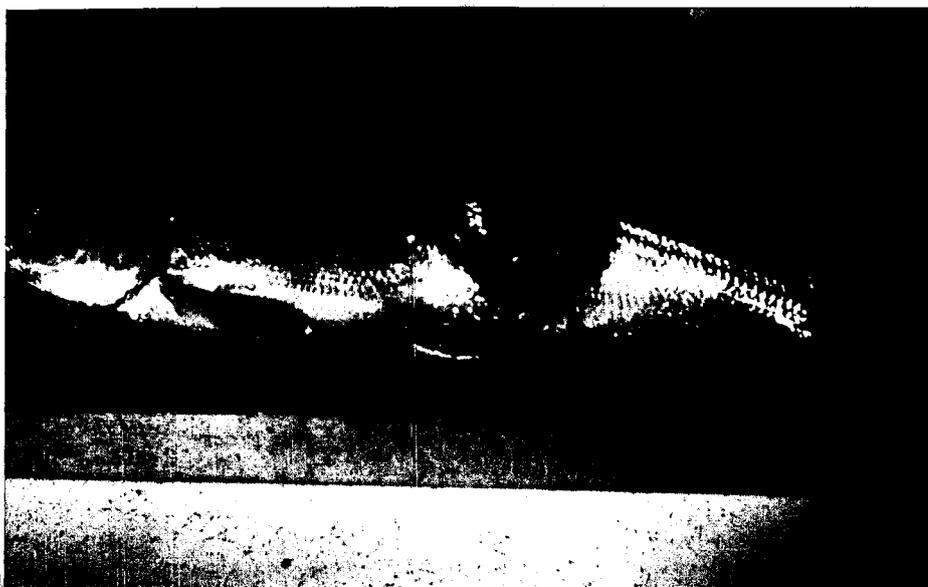
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TUMORS AND ANOMALIES

Large benign tumor
(Neurofibroma) on
coho salmon.



Fist-size tumor
(Lipoma) on the
side of a Great
Lakes whitefish.



Northern pike with
mopskof (dolphin
head).



TUMORS AND ANOMALIES

Tumors and malformations of many kinds, some due to injuries, are found among fishes (4, 5) and the cause of many of them is seldom diagnosed. Certain tumors of the liver of hatchery rainbow trout, called hepatomas, are caused by various ingredients in the diet and can be avoided by altering the diet (2, 8). Viruses are implicated in some tumors. Tumors occur on nearly all organs or tissues. Those on the skin are most obvious, and some are spectacular. Northern pike and walleyes in Michigan appear to have more tumors than other species of fish, according to our records over 25 years. Most tumors do not appear to be fatal to the fish, and the fishermen can remove them along with the entrails, before the fish is cooked.

A deformity called mopskopf is occasionally observed among fish in Michigan. Fish affected by mopskopf have an adventurous appearance which is quite striking. The upper part of the head is shortened, ending just in front of the eyes, and the lower jaw is of normal length. This abnormality was first described in the German language (6). Since the upper part of the head is shaped like that of a dolphin, it was named "mops" (dolphin)-kopf (head)-mopskopf. This condition, which has been observed in Michigan among northern pike and brook trout, brown, and rainbow trout, is believed to result from damage to the egg when the embryo is developing.

Spinal deformities are not unusual among fish of various species. Drastic temperature changes during early developmental stages are thought to be responsible in some cases. In hatcheries faulty diet may cause spinal deformities (7).

The cause of tumors is for the most part poorly known. Of course, certain chemicals are known to be carcinogenic and may cause tumors. There is also suspicion that viruses may cause certain tumors, and certainly this is the case with lymphocystis, and lymphosarcoma.

But more and more data are accumulating to implicate environmental agents as important for carcinogenesis in humans and other

Brook trout with goiter.



Gill arches of coho salmon with goiter.



Spinal deformities among hatchery lake trout resulting from faulty diet.



animals, including fish. Many of these agents enter the natural waters and come in contact with fish and invertebrates. These agents, of natural, industrial, and agricultural origin, are numerous and include such "natural" agents as UV light. Other agents include crude oil, various soluble metals and their salts, petroleum wastes, DDT, other pesticides, benzal, arsenic, domestic wastes, herbicides, aromatic amines, and various components of effluent from mines, industry, and dyestuffs.

The mechanisms by which these environmental agents act to generate neoplasia are presently unknown. That these probably do act additively and even synergistically in conjunction with multiple host factors is well known in mammalian systems, and certainly should be similar in fish. It seems clear to many researchers that there is a need for more vigorous study of the structures of natural aquatic populations and natural disease incidence, in both polluted and clean waters. Changes in tumor-like disease incidence patterns may indicate dangerous changes in the environment, sometimes human generated, which could threaten valuable plant and animal populations, and even man himself.

One tumor which has generated a lot of interest in Michigan is goiter of coho salmon. The problem of gill arch tumors in Michigan waters is due to thyroid hyperplasia (1). The fish thyroid is made of thyroid follicles not assembled into a single gland, but instead scattered generally in connection throughout the subpharyngeal and parapharyngeal area. These diffused thyroid tissues are located along the base of the ventral aorta. Tumors are noticed only when gross enlargement of thyroid tissue occurs.

Thyroid enlargement is related to dietary iodine deficiency although there are references to low oxygen content of the water as an additional environmental factor. Reference has been made that this thyroid hyperplasia among naturally occurring fish populations might be due to goiterogenic or possibly carcinogenic agents present in the water. As with most other tumors, we don't really have the final answer.

The Registry of Tumors in Lower Animals, Museum of Natural History, Smithsonian Institution, Washington, D.C. 20560, maintains a record on all types of tumors of animals, including fish.

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MORTALITIES OF FISH

- A. VIRAL DISEASES OF HATCHERY SALMONIDS
- B. MORTALITY OF FISH NOT CAUSED BY PATHOGENS

VIRAL DISEASES OF HATCHERY SALMONIDS

Viral diseases that infect hatchery salmonids are important for two reasons. First, there is no therapy or control measure available for fish affected by these diseases, and second, mortality is usually very high. The viral diseases we in Michigan are most concerned about are Infectious Pancreatic Necrosis (IPN), Infectious Hematopoietic Necrosis (IHN), and Viral Hemorrhagic Septicemia (Egtved virus or VHS). Only IPN has been observed in Michigan to date (1976) but, since the viral diseases cannot be detected among very small fish, and are difficult to detect in large fish that are carriers, the diseases may be inadvertently introduced to Michigan by fish transported from out of state.

IPN attacks the faster growing fry or fingerlings among a lot of fish, thus the best fish are lost first. Affected fish display characteristic twisting (about the long axis), and side swimming behavior somewhat similar to that of fish affected by Hexamita (Octomitus). Other external symptoms include darkening, protruding eyes, and abdominal distention (3). Internally, multiple petechiae occur in the pyloric caecal area and the liver and spleen are pale. A clear to milky mucous occurs in the stomach and anterior intestine (3).

IHN is similar to IPN in most seriously affecting the larger fish in the best condition. The earliest sign of the disease is the presence of long, opaque, off-white fecal casts trailing from the vent of fish or floating in the water (1). Other signs of the disease are protruding eyes, ascites, and hemorrhagic areas at the base of the pectoral fins. Internal characteristics may be any one or a combination of the following characteristics (1): petechiation and some larger hemorrhagic areas in the peritoneum along the kidney, air bladder, and lateral body walls; petechial hemorrhages in the fat around the spleen and pyloric caeca; the liver, kidney, and spleen may be pale; the body may be filled with clear fluid and the stomach and the intestine may contain some clear mucous.

Positive identification of viral diseases is accomplished only through cell culture or fluorescent antibody techniques which require special equipment and facilities, and by meticulous technique of specially trained technicians (1, 2).

Viral diseases cannot be treated, thus, avoidance of these diseases is the only effective control measure. Control consists of hatching and propagating virus-free stock in an uncontaminated water supply (3). Continuing successful propagation is dependent upon vigilance in excluding sources of contamination such as egg shipping cases and vehicles from other hatcheries and eggs and fish of uncertain health (3). Suspected fish stocks should be destroyed and strict sanitation measures should be applied under the direction of qualified personnel.

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MORTALITY OF FISH NOT CAUSED BY PATHOGENS

Mortalities of fish may be due to causes other than disease. Fish are completely confined to a water habitat and any physical change of the water affects the fish in some way. Species of fish vary in tolerance to certain conditions, but all have rather narrow tolerance limits. Such water characteristics as temperature, acidity, chemicals, and various gases are important and all must be considered in investigating mortalities of fish. In general, mortalities that include many species of fish of all sizes are caused from some physical phenomenon, and those that involve only one, or a few species of fish are usually the result of a disease organism, or because the species is weakened by some physiological event, such as spawning.

Toxic substances from manufacturing plants, agricultural practices (pesticides, effluent from dairies, etc.), oil fields, or other sources can cause mortalities of fish (5). Because water is moving in streams, and to a certain extent in lakes, the pollutant may exist in a certain area for only a short time before it is diluted below toxic level, so investigation of the mortality a day later, or even a few hours later, may not reveal the cause. Therefore, it is important to ascertain all sources of pollution that could reach the water.

Certain kinds of algae may be toxic to fish when eaten or upon decomposition. Blooms (periods of rapid growth) of blue-green algae (Schizothrix calcicola and Coccochlois penicystis) in Lake Michigan coincide with alewife mortalities and are suspected as a major factor in the annual die-off (8).

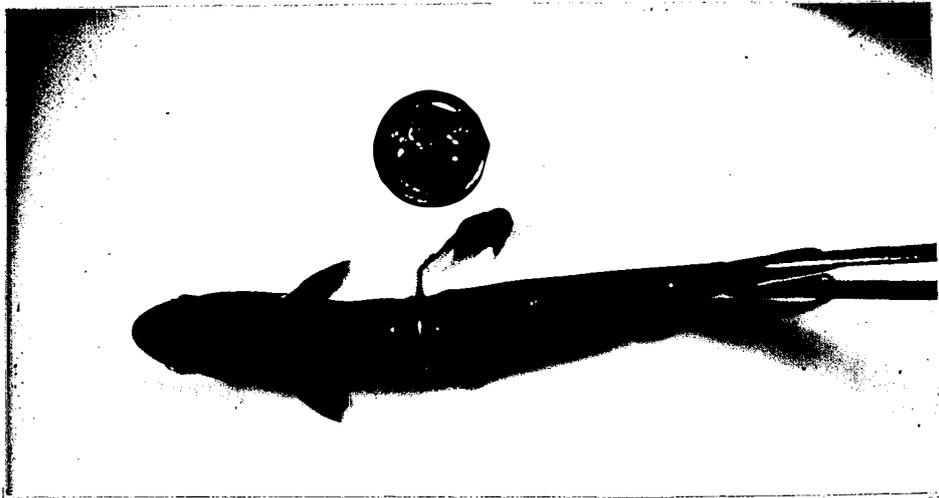
Electricity from lightning can be fatal to fish. In this case, fish of all species are affected, especially the larger fish. Man-made electricity from power lines that accidentally fall into water can be fatal to fish.

Intense silting can be detrimental to fish life by smothering eggs, damaging gills of fish, or by lowering oxygen to a lethal level (suffocation).

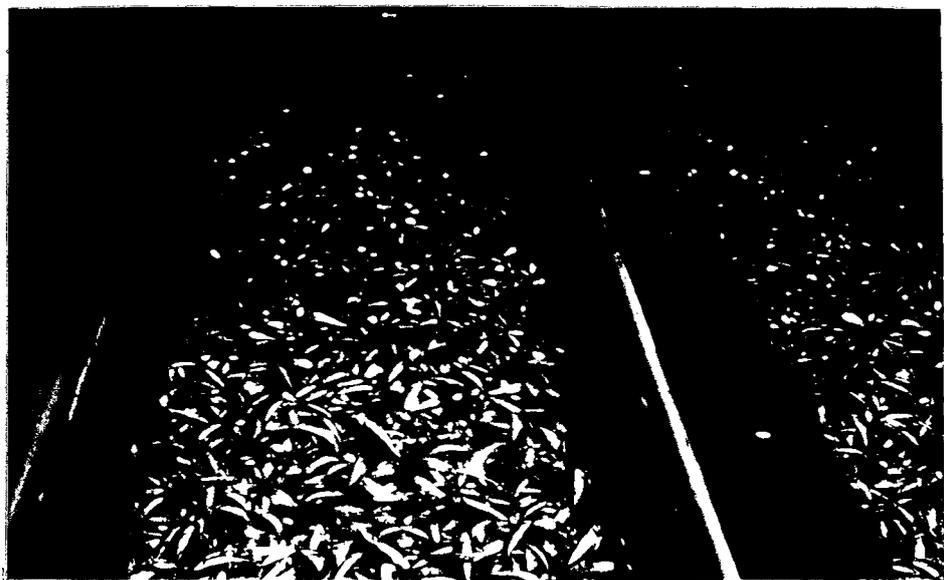
Large lesion made by a sea lamprey on the belly of a walleye.



Lesion on a brook trout caused by a "pop-top" of a beverage can.



Massive mortality of hatchery fish due to oxygen deficiency.



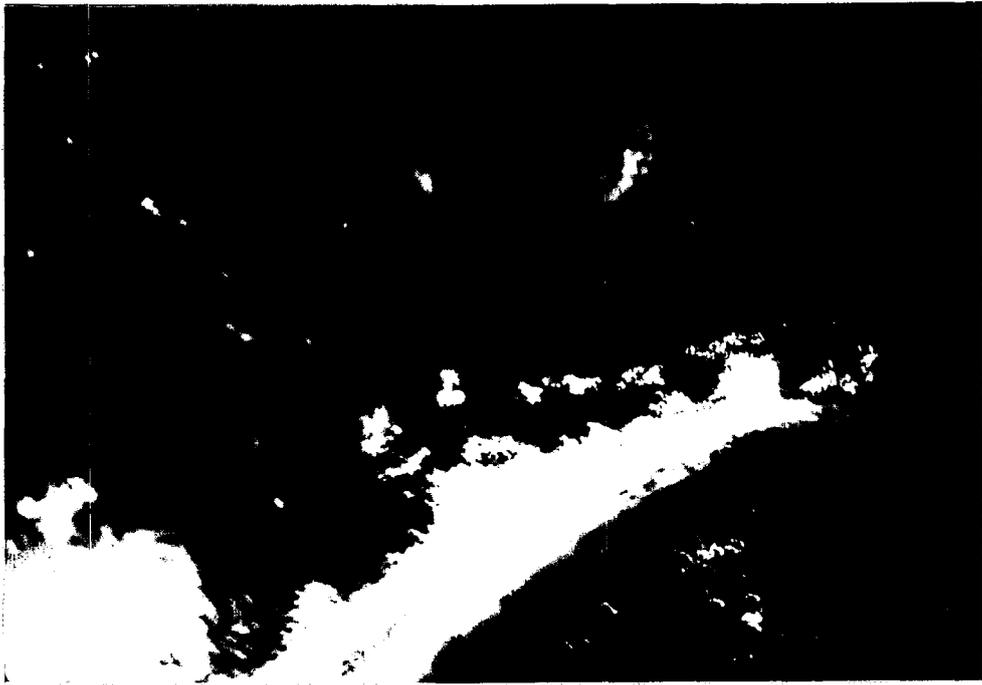
Aquatic plants release oxygen during daylight, but remove it from the water at night. Hence, in areas where there are heavy growths of aquatic plants and poor or no circulation of water, oxygen may be depleted at night resulting in fish mortality. After a few hours of daylight, oxygen is back to suitable levels for fish so no evidence remains of the cause of the mortality. Oxygen depletion may also occur when heavy algae blooms die and decompose.

Water from deep wells usually is very low in oxygen content and must be aerated to support fish life. Occasionally, this type of water is supersaturated with a gas, usually nitrogen, and this enters the blood vessels of the fish causing what is called "gas bubble disease." Gas bubbles can be seen in the capillaries of the fins and around the eyes. This condition is usually fatal and fish frequently die with their bodies flexed laterally. For immediate use, the supersaturation can be eliminated by breaking the water into a fine spray, or whipping it vigorously with a mechanical aerator.

Methane, or marsh gas, and hydrogen sulfide are produced in certain types of muck on the bottom of lakes and ponds. If these gases are released, as by incomplete dredging, fish quickly develop dropsy and die. Eventually, the gas will dissipate, but complete dredging of all the muck will eliminate the problem.

Underwater explosions kill fish, but only within a relatively short distance, according to the strength of the blast. Some people believe that any underwater blast is lethal to fish for more than 100 feet, but actually half a stick of 5% dynamite will kill fish in a radius of only about 10 feet. Fish killed by underwater blasts have characteristic lesions, according to their proximity to it. Apparently the concussion depresses the belly of the fish with considerable force. Dissection shows that the pelvic girdle has been forced rapidly to the backbone, rupturing the air bladder, and macerating the kidney and intestine immediately dorsal to the pelvic girdle.

Pronged seeds (achene) from the beggar-tick (sticktite) plants (Bidens sp.) have caused great mortalities among young fish in hatcheries



Cataract in the lens of a hatchery-reared lake trout.



Normal eye and lens of a hatchery-reared lake trout.

(3), and in natural waters (6). The fish attempt to eat the seeds, but the barbed prongs pierce flesh of the gills and throat, and fungus develops rapidly until the fish cannot breathe.

Some species of fish can be damaged by direct rays from the sun (1). Lake trout are affected in hatcheries if held in water less than 2 feet deep and exposed to the sun. Skin on the top of the head of fingerlings is eroded after about 2 months, and on the back of the area of the dorsal fin among 4- to 6-year-old fish. Greatest damage is to the lens of the eyes. Continuous exposure to direct rays of the sun causes the lens of the eyes to become opaque (cataract), rendering the fish blind (2). This damage can be prevented by shading the fish from the sun, or by holding them in water at least 30 inches deep.

Predators also contribute to the mortalities of fish. Fish are the main course on the menu of some animals and an occasional delicacy to others. If these predators were always successful in their attempts to catch fish, this section would not have been written. However, their aim is not always true and the escaped fish sometimes bear marks and wounds which may eventually cause death. Many times the wounds heal and scars are left that are characteristic of the animal that inflicted the wound (7).

The turtle makes a triangular wound which is usually fatal because the wound is deep and penetrates into the body cavity. The seriousness of the injury is somewhat dependent on the size of the turtle and the location of the wound. The tail of a fish may be completely amputated by a turtle without causing the death of the victim.

Snakes seize and hold fish crosswise with rather deep creases and abrasions at the point of seizure. Snake teeth make a series of parallel punctures in vertical rows along the side of the fish as the fish is maneuvered to swallow it head first.

Mink leave a horseshoe-shaped pattern of punctures in the side of the fish, the mark of their characteristic tooth arrangement.

The fish-eating birds leave the marks that are most easily identified because each species attacks in its own manner and the difference in the size of the bill also aids in identifying the predator.

The great blue heron spears fish, either with the bill closed or slightly open, so that the two halves act as forceps. The lightning-fast thrust is aimed at the region of the dorsal fin, and occasionally at the head if the fish is large. The clean-cut pair of converging marks or lines made when the great blue heron picks up the fish and manipulates it for swallowing are of much larger size than those made by the American bittern or by the green heron.

The bittern, or marsh-pump (thunder-pumper) usually spears the fish, leaving neat, rounded holes deep in the flesh of the back between the head and dorsal fin. The characteristic beak marks are made in manipulating the fish preparatory to swallowing.

The kingfisher rarely spears fish, but uses its bill as forceps, the force of its plunge wedges the fish in its bill. As it flies away with the fish, it moves the fish one way or another to balance it, with the sharp edges of the bill leaving a pattern of fine lines on the body of its prey. Occasionally, a fish too large to be swallowed will be caught and subsequently released, carrying on its body the characteristic marks of the kingfisher bill.

Fresh lamprey marks are easily identified by their circular or oval outline and the shallow pit rasped in the flesh. Size of the marks vary according to the species of lamprey. When healed, the scar is devoid of scales, appearing as a space of bare skin.

Considering man as a predator, injuries inflicted by fishing activity leave a variety of scars. Hooks may leave scars in the mouth, gullet, and on the skin of the fish if foul-hooked. It is not uncommon to see broken gill arches and mutilated jaws among fish from heavily fished waters. Spearing and bow-fishing also leave scars on fish that are hit and escape. Man, through thoughtless littering habits, also provides serious problems for fish. On page 120 is a photo of a brook trout that swam into the loop of a beverage can opener at an early age, and grew larger. The loop cut into the back and belly and would soon have killed the fish.

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