

ICHTHYOPHTHIRIASIS AMONG THE FISHES OF A
POND IN INDIANAPOLIS

NATHAN E. PEARSON, Butler University

During late winter and the spring of 1931 an epidemic swept through the fishes in the pond located on the South Grove golf links course, Riverside Park, Indianapolis. Hundreds of fishes died and for a period of several days conditions at the pond seemed to be such that the entire fish fauna would be killed by the ravages of the disease. The epidemic was caused by the holotrichous infusorian, *Ichthyophthirius multifiliis* Fouquet, an ectoparasitic form which infests primarily the gills and skin of fish. In these places the organisms feed upon the tissues of the host and grow to be about 0.8 mm. long. The epidermis develops about the parasite a thickening which shows as a distinct pimple-like spot or pustule that is grayish-white in color. The presence of the parasite may be quite readily detected by the examination of one of the spots or a bit of gill filament under the low power of the microscope. The parasite will usually be found in a steady rotation which is produced by the rapid movement of its cilia. The movement takes place within a cavity where the parasite comes to lie during its development.

The disease, ichthyophthiriasis, has appeared many times in Europe, where it has been found attacking the fishes in freshwater aquaria, fish hatcheries which lacked an abundant supply of running water, and shallow fish ponds. It has been prevalent particularly in Germany, France, and Holland. Several outbreaks of the disease have been recorded in various European publications, but there are only a very few references to it in American literature.

The first record of the disease was that of Hilgendorf and Paulicki (6), who described an epidemic which had appeared in the Zoological Garden, Hamburg, Germany. Their description of the parasite was very incomplete.

In 1876 Fouquet (4) published the results of his study of the disease, which had appeared regularly for a period of ten years in the basins for pisciculture of the College of France. At the end of this time the source of the water was changed and the malady quickly disappeared. The outbreaks commenced about the last of May and lasted until the first of the month of August. He gave a careful description of the adult parasite and, as far as possible, made a study of its life cycle.

Kerbert (8) described a similar disease which had appeared in the Amsterdam aquarium. He named the parasite *Chromatophagus parasiticus*. This parasite was later shown to be the same as the one described by Fouquet.

Neresheimer (12 and 13) worked extensively on the life history of the parasite.

Roth (15) described in detail the activities of the parasite and its effects on the tissues of the host. He also studied its life history.

Buschkiel (1) carried on a number of experiments during which he cultured the parasites on various species of aquarium fishes and observed their activities on the hosts. He also worked out the nuclear changes during the reproductive cycle.

Forbes (3), and Stiles (16) described an outbreak which occurred in the aquarium of the United States Fish Commission during the World's Columbian Exposition at Chicago. The disease very nearly destroyed the trout exhibit for a time. Several other species were seriously infested, but with quite various results. The infection was known to have been introduced into the aquarium by catfish that were brought from the Potomac River, April 28, 1893.

Prytherch (14) described the occurrence of the parasites on fish in the Washington aquarium during the cold winter months. The infected fishes came from Maryland waters and the Potomac River.

Prytherch states that serious epidemics have occurred in various sections of the United States; Davis (2), that the disease is common on pondfishes; and Kudo (9) gives to the parasite a wide distribution; but records which would indicate a widespread distribution of the disease among the American fishes have not been found. Evidently, they were not published.

The parasite has been placed in other genera, and new species have been described which were thought to be closely related to *I. multifiliis*. Later workers considered these forms as synonymous with Fouquet's species. Kudo recognizes one species in the United States.

The disease has been called the "fish louse" disease and the "spot" disease, but these names are confusing, inasmuch as they apply to other fish diseases.

Description of the Pond. The pond where the epidemic occurred is located near the east bank of White River, extending along the east side of Parkway Drive immediately north of Eighteenth Street. The stretch of water is long and narrow, extending for a distance of 1,500 feet in an approximate north and south direction; its width is about 300 feet throughout the greater extent. The water is shallow, the greatest depth being only 4½ feet at the time of the epidemic. At this time the water was of average depth. The bed, which is composed of sand with an admixture of considerable gravel, is covered in most places by a very thin layer of muck; but at the north and south ends the muck becomes a few inches deep. An island is located near the center of the north end.

The pond was formed artificially about thirty years ago when a roadway, which is now known as Riverside Parkway Drive, was built along the east bank of White River and Emerichsville Dam was constructed across the river about one-half a mile below this point. The depression in which the pond lies was made in order to secure material to elevate the roadway above the level of the river bank. Later, when Emerichsville Dam was completed (1900? plans for dam dated February 10, 1899), water was backed up in the river for a distance of two miles. The backwater raised the level of the river opposite the excavated depression and seepage from the river ran into it.

Any fluctuation in the height of the river at this point affects the depth of the water in the pond, but more slowly. Workmen in the park say that three years ago the gates of the dam were opened for a long period of time and the pond dried up to a small puddle. The gates, however, are seldom opened.

Two hundred feet northeast of the north end of the above described pond there is a smaller, oval-shaped pond which is approximately 200 feet long and 150 feet wide. The sides are steep and only a very small part is less than three feet deep. The greatest depth was five feet four inches at the time of the peak of the epidemic. The ground between the two lies two or three feet above the usual water level. When the water is high in the river the two ponds become united. During the flood of the winter of 1930 the ponds were united, but so far as known they have since been isolated. Although no fish died in this pond, some interesting points relative to it came up during the study of the epidemic.

Both ponds are ideal fish ponds and consequently adapted to support a large population. Aquatic vegetation, plankton, crustacea, and insect life are very abundant and furnish large quantities of food; part of the pond is shaded; and the bottom is ideal for nesting. The water is clear. During the epidemic no variation from the normal condition was discovered, and no change is thought to have occurred during the preceding several months.

The ponds were stocked artificially with several species of fishes from the Riverside hatchery, but some may have entered from the river when it was at flood stage.

The hydrogen-ion concentration was taken April 12 at twelve places in the large pond, four in the small one and two in the river opposite the large pond. The Youden pH apparatus was used. The day was clear and warm, and the readings were taken between nine and eleven a. m. The pH range in the large pond was from 7.700 to 7.955; in the small one, 7.955 to 8.006; in the river, 7.870 and 7.785. Water bloom covered about one-fifth of the surface of both ponds.

Extent of the Epidemic. Workmen in the park stated that they had observed a few dead fish the last of February or the first of March. The first reliable report concerning the epidemic came April 8; the author visited the pond April 11 and since then it has been under close observation. Dead and dying fish were found in all parts of the pond. At the north end, the shallowest part, there were more dead fish than elsewhere. The peak of the epidemic was passed about this time. By April 24 only a few fish were dying from the effects of the disease. On May 2 no struggling forms were observed, but several infected forms were seen swimming more or less actively. Shortly after this date no more were observed to be infected; however, none were caught for a careful examination. By May 29 five newly made nests were located and to all appearances the epidemic had ended. Although many fishes survived, as was indicated during the course of the summer, when no fewer than 300 nests were found, the number was reduced to a small per cent of the previous population. The toll of the epidemic was indicated at its close, when 243 dead fishes were found along 60 feet of the

east shore. It was estimated that during the epidemic at least two fishes no less than three inches long died for every square rod of surface area.

All sizes of fish from 27-inch carp, 18-inch catfish, 14-inch bass, and 7½-inch bluegills down to last year's hatch died. No species that were known to be present escaped and probably every fish was infected at some time. The latter was indicated April 16, when a 200-foot seine was drawn through the most favorable place and 30 active bluegills and crappies were caught. All were heavily infected.

The following forms were collected and identified according to Jordan's Manual (7): European carp, *Cyprinus carpio*, Linnaeus; channel cat, *Ictalurus punctatus* (Rafinesque); little pickerel, *Esox vermiculatus* Le Sueur; large-mouthed black bass, *Huro salmoides* (Lacépède); green sunfish, *Apomotis cyanellus* (Rafinesque); long-eared sunfish, *Xenotis megalotis* (Rafinesque); bluegill, *Helioperca incisor* (Cuvier & Valenciennes); white crappie, *Pomoxis annularis* Rafinesque. Dried specimens of a species of *Notropis* were found at the close of the epidemic. They probably represented the spot-tailed minnow, *N. hudsonius* (De Witt Clinton).

All species were not equally susceptible. Bluegills, crappies, sunfishes and catfishes seemed to fare the worst. Bluegills, which are extremely prolific, were many times more abundant than all the rest of the species taken together, and this fact made them seem, at times, to be the most susceptible. Large-mouthed black bass seemed to have been the least affected, although some of the dead ones carried very heavy infestations. Grass pike were among the last species to succumb. Their habit of resting near the shore, where most of the dead and dying fish were found, and consequently, where the parasites were more abundant, would subject them to a greater infestation; but the infestation on the dead and dying ones was light. Carp were not present in sufficient numbers to determine their susceptibility, but they carried heavy infestations.

The epidemic may have delayed the nesting period a few days. Under normal conditions the bulk of the bluegills have been observed to nest in this locality between the middle and last of June. The first bluegill nests were made during the last of May. After that date they increased in number until the peak was passed the last of June or the first of July. On July 15, 300 nests (mostly bluegill) were located; 220 had been abandoned and 80 had adults swimming over them. On August 8 eleven nests with males swimming over them were found. On August 15 no more could be located.

Effects of Parasites on Hosts. All soft parts of the skin, mouth, pharynx and gills were invaded by the parasites. The infested fish swam less actively and later became listless. Several observers of the disease in aquaria have reported that the skin became so irritated the fish would attempt to rub off the parasites by swimming against solid objects, but this was not observed in the pond. The gills were deeply inflamed and became bright crimson in color. Where a heavy infection was present, suffocation was indicated by the fish swimming near the surface.

In the later stages of the disease almost all of the fishes were attacked by *Saprolegnia*. The spores, no doubt, entered through wounds in the skin that were caused by the escaping parasites or through injuries caused by the struggles of the fish. The mould grew rapidly. Some individuals were swimming upright with one-third or more of the body covered by masses of the parasitic filaments. *Saprolegnia* completed the work of destruction that the parasitic protozoan had begun. The fish gradually lost their equilibrium, swam irregularly in curves near the shore, and slowly died.

Saprolegnia was so abundant on most of the infected fish that casual observers attributed the epidemic to it. It caused the production of characteristic red blotches or "scalded areas," usually on the sides of the body opposite the viscera, and the loss of scales. In some individuals the pectoral and anal fins were nearly destroyed, and frequently the soft parts of the anal, dorsal and caudal were badly affected. But all sorts of combinations of effects and extent of the growth of the fungus were seen.

The external protozoan infection was the heaviest while the fish were still active and before growth of the mould had begun. Many fish died with the skin containing practically no parasitic protozoans visible to the unaided eye, but an examination of the gills revealed an abundance of them.

The Life Cycle of the Parasite. To understand the causes and methods of control of the disease it is necessary to know the life cycle of the parasite. The cycle is rather complicated and it is not typical of the Infusoria in that the adult undergoes a process of sporulation. This process occurs after the adult has escaped from the host and during encystment. Encystment ends in the production of the reinfecting forms.

The Adults. The adults are readily recognized by a microscopic examination of a bit of the infected tissue which has been mounted under a coverslip. Here they may be seen in active rotation. The body is plastic and conforms to the shape of the cavity in which it lies—elongate, oval, globose, somewhat flattened, etc. It is easily released by the use of dissecting needles, or hundreds of free swimming forms of various sizes may be secured by placing an infected fish in a jar of pond water. They swim actively back and forth and when a large number is observed in a jar of water they appear to the unaided eye very much as a group of paramoecia. They are positively phototropic and collect within a short time on the side of the jar next to a north light.

The larger adults measure 0.48 by 0.80 mm. in length, but many smaller ones escape and undergo sporulation. The body is covered by a great number of uniform cilia arranged in a number of regular rows. The mouth is small and is seen with great difficulty in living specimens if at all; but its position may be determined in small adults by the presence of a small irregular mass of recently ingested food and by its anterior position when the animal is swimming. The cytoplasm is granular with scattered opaque spots. Near the center is a large macronucleus. The micronucleus cannot be seen in the adult. A number of small contractile vacuoles are located next to the periphery. The struc-

ture of the cortex cannot be seen in the living form. If it is punctured and the cytoplasm permitted to run out, the posterior half of the cortex is seen to be greatly thickened. Prepared cross-sections show this region to be highly specialized. It is thought to serve as an adhesive organ, for loosening epithelium in the food-getting process, and for moving through the tissues of the host.

When the parasites first penetrate the host they are very small. At that time they may feed upon body fluids. Later they feed upon epithelium and blood cells. The cytoplasm of those found in the gills is filled with blood corpuscles. Cross-sections of the gills show the corpuscles entering the cytoplasm through the mouth, which becomes greatly enlarged while the organism is feeding. It is probable that this method of feeding does the greatest damage to the fish.

Encystment and Sporulation. After the parasites escape from the host they swim around for a time, settle down, and form a temporary gelatinous cyst around the body outside of the cilia. Binary fission may take place while the adult is actively swimming, or it may occur after the individual becomes inactive. Frequently, it does not take place.

Within the temporary cyst sporulation begins. Division is regular forming 2, 4, 8, 16, etc., cells until 1,200 to a few thousand small daughter cells are formed. The number of daughter cells which are formed depend upon the size of the adult. These then develop within the cyst into free swimming spores. Throughout the process of sporulation the separate cells rotate constantly within the gelatinous cyst, which adheres firmly to its substratum; their plastic structure responding to pressure from every direction.

The larger adults, isolated in depression slides filled with pond water, completed the process of sporulation within a period of 22 to 30 hours when the temperature varied from 66° to 72° F. Immature adults sporulated more slowly. They swam around longer and the cleavages were slower. One specimen 0.14 mm. long remained a free swimming individual for a period of four days and then within 24 hours formed a few spores. Other smaller ones lived as long as six days and then died without sporulating.

As the spores mature they escape from the cyst over a period of about an hour. The escape is made from the gelatinous cyst through small apertures made by the spores. The first ones to mature swim next to the cyst wall touching it here and there as though searching for a weak place. When a suitable contact has been made the anterior end becomes pointed and with the body rotating the organism forces its way through the wall. In so doing it becomes elongate and, frequently, one is seen to be dumb-bell shaped as the anterior end emerges from the wall and the posterior end follows through the constricted opening. Other spores make similar openings or squeeze through those already formed.

In no culture was the cyst found to burst open as Prytherch (14) described. His photographic figures do not show the gelatinous wall and, consequently, the nature of the escape. Neither were any adults found which produced thousands of spores as he described,

Spores within the cyst are spindle-shaped, but after leaving it they become ovoid with the anterior end bluntly constricted. One side which might be considered ventral is somewhat flattened. The spores swim rapidly by the rapid vibrations of long cilia. In swimming they rotate about a longitudinal axis, with the anterior end moving in circles. The movement is in a straight line for a moment when they turn suddenly and dart in another direction, or they may strike some object with considerable force. When observed under low power the movement away from the cyst is seen to be very slow. A macronucleus and a micronucleus are present. The single contractile vacuole was observed to contract eight times per minute at a room temperature of 65° F.

The spores have no mouth or adhesive organ, but it is said that they enter the host by attaching themselves by the anterior end and penetrate the tissue by the rotation of the body.

If no fish are present the spores soon die. Those observed in this laboratory lived as long as 30 hours. At the end of 17 hours they swam less actively and might not have been able to penetrate the tissues of the host at that time.

The parasite cannot live outside of the host's tissues longer than a few days. My cultures indicate the possibility of about one week in some immature adults.

The spores in the new hosts develop rapidly. Buschkiel (1) and Prytherch (14) found that in warm water the presence of the parasites could be seen with the unaided eye six days after infestation. In cold water the period of development becomes longer.

The presence of a micronucleus in the reinfecting spore indicates that a sexual cycle intervenes. Fouquet (4) and Neresheimer (13) were unsuccessful in their search for a process of conjugation. The later author recognized two types of life cycles, but the careful work of Roth (15) and Buschkiel indicated only one. Buschkiel in a detailed study of the life cycle found that autogamy intervenes during encystment. During the process of cleavage a "nebenkern" (micronucleus?) becomes detached from the macronucleus. This divides twice, the daughter nuclei then undergo changes, and fuse in pairs to give rise to the micronucleus of the spore.

Cause and Control. The outbreak of the epidemic can be attributed to overcrowding in a shallow pond. The nature of the life cycle permits a rapid multiplication of the parasites if there is an abundance of hosts, and shallow water increases the opportunity for the spores to find hosts. The epidemic came to an end when the spores could not find hosts. It is not thought that the long period of dry weather that preceded the epidemic had any influence.

It is interesting to note at this time the possible reason for the non-appearance of an epidemic in the small pond which has been previously described. Many fish, mostly bluegills, large-mouthed black bass and sunfish, were known to be in the pond but whether it was overpopulated to the same extent as the larger one could not be determined. The fish in this pond may not have been infected; however, on April 16, before the nature of the disease was known, a seine was

taken directly from the large pond where it had been in contact with many diseased fish and used in the small one. As yet, November 18, no signs of an epidemic have appeared. In the deeper water the fish may be able to escape the spores, or the adult parasites may not undergo sporulation.

Inasmuch as the parasite has become established in the large pond a recurrent epidemic may reasonably be expected within a few years. It can be avoided by taking out a number of fish each year, or the pond can be freed of the parasites by seining out all the fish and restocking after a period of time with uninfected ones.

After an epidemic has started among a large group of fishes, some may be saved by placing them in a stream. This may also spread the disease, but many of the parasites will die in the running water before the life cycle is completed. The large volume of water also decreases the possibility of a spore finding a host.

Ponds which have had infected fishes in them should be drained and sprinkled with quicklime if they are to be used for uninfected fish within a short time.

Small numbers of fishes have been treated in various ways in order to effect a cure. Since the parasites are embedded in the skin and gills, chemicals that would kill the parasites would injure the fish. Consequently, the treatments that are used must attack the parasites as they leave the host.

For small groups of infected fish Prytherch (14), has devised a tank with running water in which they are placed until all the adults have escaped. The running water carries the adults away and they fail to complete the life cycle.

Stiles (16), Davis (2), and Mellen (10 and 11) have suggested chemical treatments for small numbers of fishes. They are methods which require a number of treatments in order to effect a cure. The fish are placed at regular intervals in weak chemical solutions that kill the parasites as they escape from the host, but at the same time they do not injure the fish. For trout Davis suggests a three per cent salt solution, or a five per cent solution of aluminum sulphate. If salt is used the fish are dipped in the solution until they show signs of distress; if aluminum sulphate is used the fish should be left in the solution for one minute. The treatments should be repeated daily until the fish are free of parasites. Mellen (11) recommends five drops of two per cent mercurochrome solution to a gallon of water in which the fish are left for a period of five days. The solution should be renewed each day. A stronger solution may be used advantageously. The discovery of the mercurochrome treatment was made by Mr. Wallace Adams of the Steinhart Aquarium, San Francisco, California.

Dr. G. C. Embury, professor of aquiculture at Cornell University, has had considerable experience with the disease in the state of New York. With his permission the following is quoted from a personal letter:

"For trout I like the salt treatment given three or four times. But for goldfish there is nothing better than ten drops of mercurochrome to one gallon of water. The fish must be left in this solution five days in order to eliminate all stages.

“The concentration of ten drops to the gallon of water is too strong for tropical aquarium fishes and probably for young goldfish. On larger goldfish it acts more quickly than the weaker solution and generally eliminates all of the protozoa in three days.

“Also Dr. Hess’ (5) solution of one part potassium permanganate to 270,000 parts of water given for two hours on three consecutive days will usually effect a cure.”

LITERATURE CITED

1. Buschkiel, Alfred L. Beiträge zur Kenntnis des Ichthyophthirius multifilius Fouquet. Arch. f. Protistenk. 21:61-102, 2 pl., 1 fig., 1910.
2. Davis, H. S. Care and Diseases of Trout. U. S. Bureau of Fisheries, Document No. 1061:143-144, fig. 5a, 1929.
3. Forbes, S. A. The Aquarium of the United States Fish Commission at the World’s Columbian Exposition. Bull. U. S. Fish Commission 13:143-158, 1893.
4. Fouquet, D. Note sur une Espèce D’Infusoires Parasites des Poissons D’Eau Douce. Arch. de Zool. Exper. et Gen. 5:157-165, pl. 5, 1876.
5. Hess, W. N. Control of External Fluke Parasites on Fish. Jour. Parasitol. 16:131-136, 1930.
6. Hilgendorf, F. und Paulicki, A. Infusionstierchen als Hautparasiten bei Süßwasserfischen. Centralb. f. d. med. Wissensch. 7:33-35, 1869.
7. Jordan, David S. Manual of the Vertebrate Animals of the Northeastern United States. 13th Edition, Yonkers-on-Hudson: The World Book Company. xxxi+446, 1929.
8. Kerbert, C. Chromatophagus parasiticus. A Contribution to the Natural History of Parasites. (Translation by Herman Jacobson of— Ein Beiträge zur Parasitenlehre. Nederl. Tijdsch. v. d. Dierk., Amsterdam, 5:44-58, Taf. Iv, 1884). U. S. Fish Commission Rept. for 1884, pp. 1127-1137, 1 pl.
9. Kudo, Richard R. Handbook of Protozoology. Springfield: Charles C. Thomas, x+454, 1931.
10. Mellen, Ida M. New Medicines for Sick Fishes. Zool. Soc. Bull. (New York) 29:182-187, 1926.
11. Mellen, Ida M. The Treatment of Fish Diseases. Zoopathologica 2:1-31, 1928.
12. Neresheimer, Eugen. Zur Fortpflanzung eines parasitischen Infusors (Ichthyophthirius). Sitz.-Ber. d. Ges. f. Morph. u. Phys. in München 23:102-106, 1907.
13. Neresheimer, Eugen. Der Zeugungskreis des Ichthyophthirius. Ber. d. k. b. biol. Versuchsstation in München, 1:165-184, pl. VI, 1908.
14. Prytherch, H. F. The Ichthyophthirius Disease of Fishes, and Methods of Control. U. S. Bur. Fisheries Document No. 959, figs. 1-10, 1924.
15. Roth, Wilhelm. Beiträge zur Kenntnis des Ichthyophthirius multifilius Fouquet. Blätter f. Aquarien-u: Terrarienkunde 19:680-684, 692-698, 705-711, 726-731, figs. 1-17, 1908.
16. Stiles, C. W. Report on a Parasitic Protozoan observed on Fish in the Aquarium. Bull. U. S. Fish Commission 13:173-190, 1893.

