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Abstract

Digenetic trematodes (class Trematoda) often plague pond-reared fishes. Most digenetic trematodes are not a serious threat to fish health; however, their mere presence often renders the fish undesirable by consumers. The black grub (Uvulifer ambloplitis), white grub (Posthodiplostomum minimum), and yellow grub (Clinostomum complanatum) are commonly seen digenetic trematodes in fish in earthen ponds located in the North Central Region of the United States. The purpose of this publication is to discuss the biology and prevention of these parasites, along with their effect on infected fish. Hereafter, these digenetic trematodes will be referred to as “grubs”.

Disciplines

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Introduction

Digenetic trematodes (class Trematoda) often plague pond-reared fishes. Most digenetic trematodes are not a serious threat to fish health; however, their mere presence often renders the fish undesirable by consumers. The black grub (*Uvulifer ambloplitis*), white grub (*Posthodiplostomum minimum*), and yellow grub (*Clinostomum complanatum*) are commonly seen digenetic trematodes in fish in earthen ponds located in the North Central Region of the United States. The purpose of this publication is to discuss the biology and prevention of these parasites, along with their effect on infected fish. Hereafter, these digenetic trematodes will be referred to as "grubs".

Grubs are internal parasites (endoparasites), varying in size, shape, and habitat, with complex life cycles involving several hosts (Olsen 1962). The three aforementioned fish grubs have three hosts in their life cycles. Besides fish, aquatic snails and birds serve as hosts for these grubs. The grubs undergo both sexual and asexual reproduction within these hosts (Chandler and Read 1961), and can either actively or passively invade their hosts (Williams and Jones 1994).

The following is a summary of the life cycle for the selected grubs (Olsen 1962). The asexual phase in grubs is typically in snails, the first intermediate host. Miracidia, ciliated larval forms of the grub, infect aquatic snails and continue development, by transforming into cercariae; the final products of the asexual phase in snails. The free-swimming cercariae then escape from the host into the water. At this time, the cercariae become incorporated into the tissue of the second intermediate host (fish), and transform into metacercariae (visible grubs in the fish). When a definitive host (aquatic bird) consumes an infected fish intermediate host, the bird will be infected with the metacercariae. Sexual development continues within the definitive host, the fish-eating bird. Adult grubs then produce eggs that later hatch in water, producing miracidia, completing the life cycle.

Black Grub

Black grubs, known to cause black spot disease (Lemly and Esch 1985), are frequently seen in freshwater fishes as pinhead-sized black spots (1 to 3 mm or 1/32 – 2/32 inch) in the skin, tail base, fins, and musculature (Hunter and Hunter 1938). Sunfish (*Lepomis* spp.), black bass (*Micropterus* spp.), crappie (*Pomoxis* spp.), and yellow perch (*Perca flavescens*) are the most commonly affected species (Lemly and Esch 1985). The metacercariae of the black grub become encapsulated by host tissue and melanophores surround the outer layers; consequently, the dark color of the embedded grub causes affected fish to have a "peppered" appearance (Fig. 1). Their presence often renders infected fish undesirable to consumers (Fig. 2). Hoffman and Putz (1965) concluded that black grubs have a life span over 4 years in fish maintained at 12˚C (54˚F) water.

Photo credit to W. A. Rogers, Department of Fisheries and Allied Aquacultures, Auburn University.

Fig. 1. Black grubs embedded in largemouth bass fillets.

The life cycle of the black grub has been known since 1931 (Hoffman and Putz 1965). The definitive host is usually the belted kingfisher (*Megaceryle a. alcyon*); the sexually mature grub resides and reproduces in the intestine. Eggs, which develop into miracidia, are released from adult grubs and are passed through the bird by its feces. A snail (genus *Planorbella*) (Turgeon et al. 1998), the first intermediate host, is invaded by the miracidia. The miracidia develop asexually and multiply into cercariae. The free-swimming cercariae escape the host. The

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Fig. 2. Black grubs embedded in a rock bass.

cercariae infect the fish host by penetrating the skin and transform into metacercariae (visible encysted black grub). The cycle is completed when a bird consumes an infected fish.

Black grubs can cause harmful effects and secondary diseases in fish. Cercariae penetrate through the skin causing mechanical damage and hemorrhage (Hoffman 1967). Infected smallmouth bass (*M. dolomieui*) lose weight and body condition (Hunter and Hunter 1938). Until the black grubs become encapsulated in the host, the host loses lipids (fats) and their oxygen requirements increase (Hunter and Hunter 1938; Lemly and Esch 1984). Lemly and Esch (1984) indicated that heavily infected bluegill (*L. macrochirus*) often enter the winter months in lipid-depleted (low fat) state; consequently, these fish have few energy reserves to last over winter and that would affect their ability to survive.

White Grub

White grubs (usually no larger than 1 mm or 1/32 inch) occur in a wide variety of North American freshwater fishes (Grizzle and Goldsby 1996), but one pond strain of white grub (*Posthodiplostomum minimum centrarchi*), is most commonly found in centrarchid fishes (Hoffman 1958). Another strain of white grub (*P. m. minimum*) infects mostly cyprinid fish species (Hoffman 1999). Producers of pond-reared fishes often overlook white grubs because of their small size and location in the fish; they reside in and around the fish's organs and not in the flesh (Fig 3). White grubs primarily affect kidneys, liver, and heart, but they also occur in the spleen, connective tissue of the gut (Fig. 4), and ovary (Avault and Smitherman 1965; Spall and Summerfelt 1970).

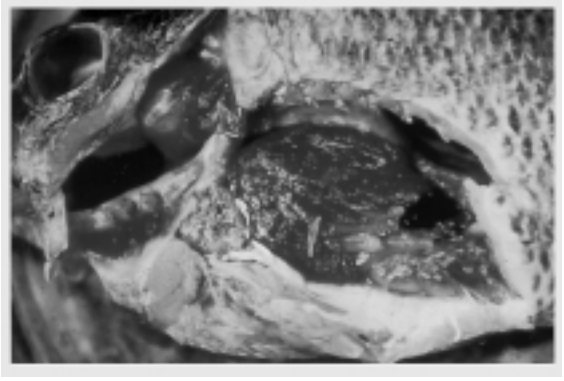

Photo credit to W. A. Rogers, Department of Fisheries and Allied Aquacultures, Auburn University.

Fig. 3. White grubs embedded in the organs of a bluegill.

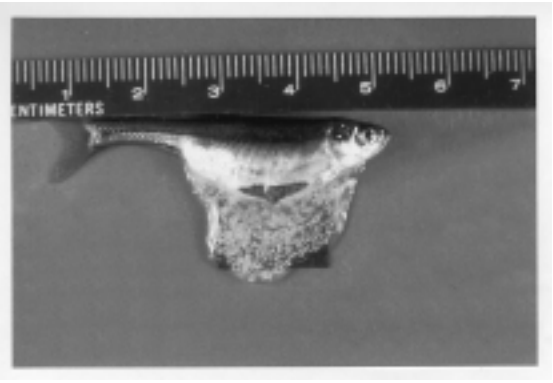

Photo credit to Andrew J. Mitchell, USDA/ARS, Stuttgart, Arkansas.

Fig. 4. A fathead minnow heavily infected with white grubs.

Spall and Summerfelt (1970) reported that male centrarchids had higher prevalence and intensity of white grubs than females. They suggested behavioral differences (male spawning habits) placed males in proximity to infected snails longer than females.

The definitive host is primarily the great blue heron (*Ardea herodias*); the adult grub resides and sexually reproduces in the bird's intestine. Eggs are released from adult grubs and are passed through the bird by its feces. The eggs hatch in water releasing free-swimming miracidia. Miracidia have to locate and infect an aquatic snail (genus *Physa*) or they perish. Germinal sacs develop in the liver tissue of the snail. The germinal sacs release free-swimming cercariae, which have a short period to infect a fish host, or they also will perish. The cercariae infect the fish by penetrating the skin and are carried by the circulatory system to the kidneys, liver, and heart. In the organs, the cercariae develop into the metacercariae stage (white grub). The cycle is complete when a fish-eating bird consumes the fish.

White grubs can cause harmful effects in fish (Spall and Summerfelt 1969), especially during the first few days after infection (Meade and Bedinger 1967). The white grub affects larval fish when grubs become too numerous and organs are compressed. Death occurs if the liver or other organs are destroyed by the metacercariae. Hoffman (1958) concluded that white grubs cause hyperemia (bleeding) at fin bases and mortalities in heavily infected fathead minnows (*Pimephales promelas*). Hoffman and Hutcheson (1970) indicated that an unusual case of white grubs in the flesh of striped bass (*Morone saxatilis*) caused deformation, exophthalmia, and mortality. Mitchell et al. (1982) found that heavily infected fathead minnows had displaced organs, a build up of body fluids in the body cavity, ruptured abdomens, and mortality.

Grizzle and Goldsby (1996) speculated that the penetration of cercariae impair the immune functions of infected fish; consequently, increasing fish vulnerability to secondary infection, e.g., bacterial and fungal.

Yellow Grub

The yellow grub is a large (3 to 8 mm or 1/8 to 1/4 inch), common parasite in North America (Olsen 1962). Yellow grubs are embedded intermuscularly (in the muscle) or subcutaneously (under the skin) in fish. The most common fish-hosts in North America are yellow perch, bass, and sunfish (Olsen 1962); however, it is a relatively safe to assume that the yellow grub is capable of infecting all freshwater fish species because it has been reported from many fish species (Hoffman 1999). When present, the parasites are highly visible to the unaided eye after skinning or filleting because of their size and color. The yellow grub is not harmful to humans if removed or cooked; however, anglers and consumers do not readily accept infected fish because of their unsightly appearance (Hoffman 1999). Elliot and Russert (1949) have reported individual yellow grubs living in fish for about 4 years.

The definitive host of the yellow grub is usually the great blue heron (*Ardea herodias*) and the adult grub resides and reproduces in the throat of the bird. Eggs are released into the water from the bird when the bird thrusts its beak into the water. The eggs hatch in water releasing miracidia. The miracidia must find a snail (genus *Planorbella*), or they will die. After the miracidia enters the snail, the miracidia transform into germinal sacs, which release free-swimming cercariae. The cercariae have a limited period to locate and penetrate a fish host, or they perish. The cercariae infect the fish by penetrating the skin, then embed themselves into the muscle of infected fish and finally develop into the metacercariae (yellow grub). The cycle is complete when a fish-eating bird consumes the fish.

Photo credit to Andrew J. Mitchell, USDA/ARS, Stuttgart, Arkansas.

Fig. 5. A fathead minnow infected with yellow grubs.

The yellow grub is relatively harmless to infected fish, except when they are heavily infected (Fig. 5) (Hoffman 1999). The presence of this grub in propagated fish species has caused fish producers economic losses because even lightly infected fish are rejected by consumers.

Prevention

It is important to limit grub infections in fish raised in ponds. Avoidance is essential because, unfortunately, infected fish cannot be treated. To limit grub infections in pond-reared fishes, snails or fish-eating birds must be controlled. However, controlling these hosts after grub infections have been established is useless because it takes years for the grubs to be eliminated from the fish. Control at this point would only serve to prevent further build up of grubs in fish.

Snails are among the most common organisms of shallow areas in ponds, streams, and lakes (Brown 1991). Snails feed on decomposing organic material and algae growing on various surfaces such as rocks and plants. Unfortunately, controlling snails is somewhat difficult because they can distance themselves from chemicals by burrowing into substrate (Brown 1991). Pumpkinseed sunfish (*L. gibbosus*) (Etnier 1971) and redear or shellcracker sunfish (*L. microlophus*) (Pflieger 1975) consume snails; therefore, they can be used as biological control agents to help prevent grub infections in ponds. However, this method of biological control will not assist in eliminating existing grubs in fish at post-infection periods. The removal of marginal pondweeds is of some help, as they constituted the preferred snail habitat of a pond. Therefore, the use of governmentally approved contact herbicides can remove these weeds, thus limiting snail habitat. Finally, not always practical, is the draining, complete drying for several weeks, and refilling of ponds to reduce snail numbers.

Although only great blue herons and kingfishers have been discussed in this publication, all fish-eating birds should be deterred from pond-rearing facilities because they prey on fish and carry several fish diseases including grubs. The Migratory Bird Treaty Act protects all fish-eating birds (APHIS 1997); therefore, producers need responsible and environmentally sound solutions for scaring or

removing birds. The Wildlife Services (WS) program, a division of the USDA's Animal and Plant Health Inspection Service (APHIS), helps reduce damage to aquaculture facilities caused by wildlife. WS wildlife biologists offer technical and direct operational assistance to aquaculture producers by conducting on-site evaluations to assess damages and to identify the wildlife (birds) causing it.

WS encourages the use of netting, wire grids, and fencing because these devices offer producers some long-term protection. However, physical barriers can be impractical for pond owners because of high costs, maintenance, and harvest interference. WS also encourages the use of noise-making devices (i.e., propane cannons and cracker shells) and visual devices (i.e., "eye-spot" balloons, remote-control boats and planes, scarecrows, etc.) Unfortunately, wildlife quickly adapt to these devices when they are used continuously. The use of large active dogs to deter birds away from ponds is highly effective and recommended, especially at night.

If physical and scaring techniques continually fail to reduce aquaculture losses, the U.S. Department of the Interior's Fish and Wildlife Service (US-FWS) can issue depredation permits to remove a limited amount of fish-eating birds from specific facilities; however, these permits are tightly controlled because of the Migratory Bird Treaty Act (APHIS 1997).

For further information or technical assistance concerning wildlife control, contact the nearest USDA/APHIS/ Wildlife Service (WS). To find the nearest Wildlife Service (WS) in your state, contact your local Cooperative Extension agent or call the WS Operational Support Staff Office at (301) 734-7921.

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