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TRIAENOPHORIASIS IN LAKE ERIE WHITE BASS, *Morone chrysops*[1]

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Abstract: The pseudophyllidean tapeworm, *Triaenophorus nodulosus*, was found in 51% of the adult white bass, *Morone chrysops*, sampled from western Lake Erie during the summer of 1973. Prevalence of infection with *Triaenophorus* increased with size and age of fishes. This worm is responsible for the disease triaenophoriasis, which is grossly characterized by large, white cysts, dark-brown "streaks" and hemorrhages in the liver. Microscopically, there is an acute inflammatory response, necrosis of liver parenchyma, squamous metaplasia, fibrosis and displacement of liver tissue as the tapeworm grows. Many plerocercoids were necrotic and surrounded by a dense tissue. The pathophysiology of extensive liver obstruction needs to be investigated as well as its effect upon infected fish populations.

INTRODUCTION

Triaenophoriasis is a disease caused by plerocercoids and adult tapeworms belonging to the genus *Triaenophorus* Rudolphi, 1873. There are three species in the genus, all occurring in North America; *T. nodulosus* Pallas, 1760; *T. crassus* Forel, 1868; and *T. stizostedionis* Miller, 1945. The life history of *T. nodulosus* was investigated by Miller.[8,9,10] Adult tapeworms occur in the intestine of pike (*Esox lucius*). Eggs are shed during the spring and coracidia hatch in the water in 15-16 days, depending upon water temperature. Coracidia are eaten by many species of copepods, but only *Cyclops bicuspidatus* and *Cyclops vernalis* are suitable intermediate hosts. The coracidium penetrates through the gut wall into the copepod hemocoel and develops into a procercoid. Infected copepods are eaten by fishes, the procercoid migrates to the viscera and develops into a plerocercoid larva. Fish infected with plerocercoids are eaten by pike and the worms mature in the upper intestine. Lawler and Scott[7] reviewed the host records and geographical distribution of *Triaenophorus* in North America. They noted a close association among the July 70 F(21 C) isotherm, the southern distribution of *Triaenophorus* and the distribution of the definitive host, *E. lucius*.

Lake Erie lies on the southern edge of the range of both *E. lucius* and *Triaenophorus*. Bangham and Hunter[2] reported the plerocercoid of *T. nodulosus* from *Percopsis omiscomaycus, Perca flavescens* and *Micropterus dolomieui* and adult *Triaenophorus* sp. from *Stizostedian vitreum, S. canadense* and *S. glaucum*. This species was presumably *T. stizostedionis*. Bangham[1] resurveyed the fish parasites of Lake Erie in 1957 and found adult *T. stizostedionis* in *S. vitreum* and the plerocercoid of *T. nodulosus* in the following species: *P. flavescens, S. vitreum, S. canadense, Pomoxis nigromaculatus, Ambloplites rupestris, Micropterus salmoides* and *Morone chrysops*. This is the earliest record of *T. nodulosus* in Lake Erie white bass, although Cooper[4] reported this species from white bass taken

[1] Supported by The Ohio Division of Wildlife under Federal Aid in Fish Restoration Act, Project No. F-48-R-2.

from an unknown locality. Dechtiar[5] was the first to report adult *T. nodulosus* in Lake Erie pike. He also reported plerocercoids of this worm from the following fish: *Moxostoma anisurum, M. erythrurum, Carassius auratus, Notropis cornutus* and *M. chrysops*. Dechtiar further noted adult *T. stizostedionis* from *S. vitreum* and plerocercoids from *P. omiscomaycus*. There is no record to date of *T. crassus* from Lake Erie.

During a study on the impact of some nematode parasites on Lake Erie fish, we frequently noted gross lesions in the livers of adult white bass. These lesions appeared to be associated with tapeworm plerocercoids. This study was undertaken to determine the identity of these tapeworms and their relationship to the observed tissue changes, to describe the gross and microscopic lesions and to estimate the prevalence of triaenophoriasis in the white bass population of western Lake Erie.

MATERIALS AND METHODS

Fish were collected by otter trawl, commercial shore seines and by hook and line between May and September, 1973. Livers for histological examination were excised in the field and immediately fixed in alcoholic Bouin's fixative. Specimens were transported to the laboratory on ice and examined immediately. Individual fish were measured, sexed and examined for patent, gross lesions only. Livers were dissected and examined in a cursory manner because time did not permit a more thorough examination. No attempt was made to count the number of plerocercoids within each liver. Specimens of plerocercoids were dissected free from liver tissue, fixed in AFA, preserved in 70% ethanol and stained with Semichon's carmine. Liver tissue fixed in Bouin's was embedded in paraplast, sectioned at 8 μm and stained with hematoxylin and eosin, Mallory's or Masson's trichrome staines.

RESULTS

Of 115 adult *Morone chrysops*, 1+ to 3+ years of age, 51.3% (59) had gross lesions. All plerocercoids were identified as *T. nodulosus* based upon the morphological characteristics of the hooks on the scolex (Fig. 1). While the intensity of these infections was not recorded, fish harbored from one to numerous plerocercoids, causing extensive obliteration of liver tissue. There was no significant difference between the prevalence of infection in male and female fishes (X^2, $P > .05$). Table 1 shows the relationships between prevalence of the lesions and fish length (age). Fish entering their 2nd year (150-200 mm) had the lowest prevalence while 2 and 3 year old fish (approximately 201-250 mm and 251+ mm) had a progressively higher prevalence of triaenophoriasis.

The mean length of fish with lesions was 243 mm, while the mean length of unparasitized fish was only 207 mm. This difference was highly significant (X^2, $P < .01$) suggesting that infection of white bass with *T. nodulosus* and development of disease is closely related to the size and age of the fish.

TABLE 1. Prevalence of Triaenophoriasis in Four Size-age Classes of **M. chrysops.**

	Fish Length—Age			
	1+ years 150-200mm	2+ years 201-250mm	3+ years 251-300mm	3+ years over 300mm
No. examined	31	60	19	5
No. infected	7	32	15	5
% infected	22.6	53.3	78.9	100

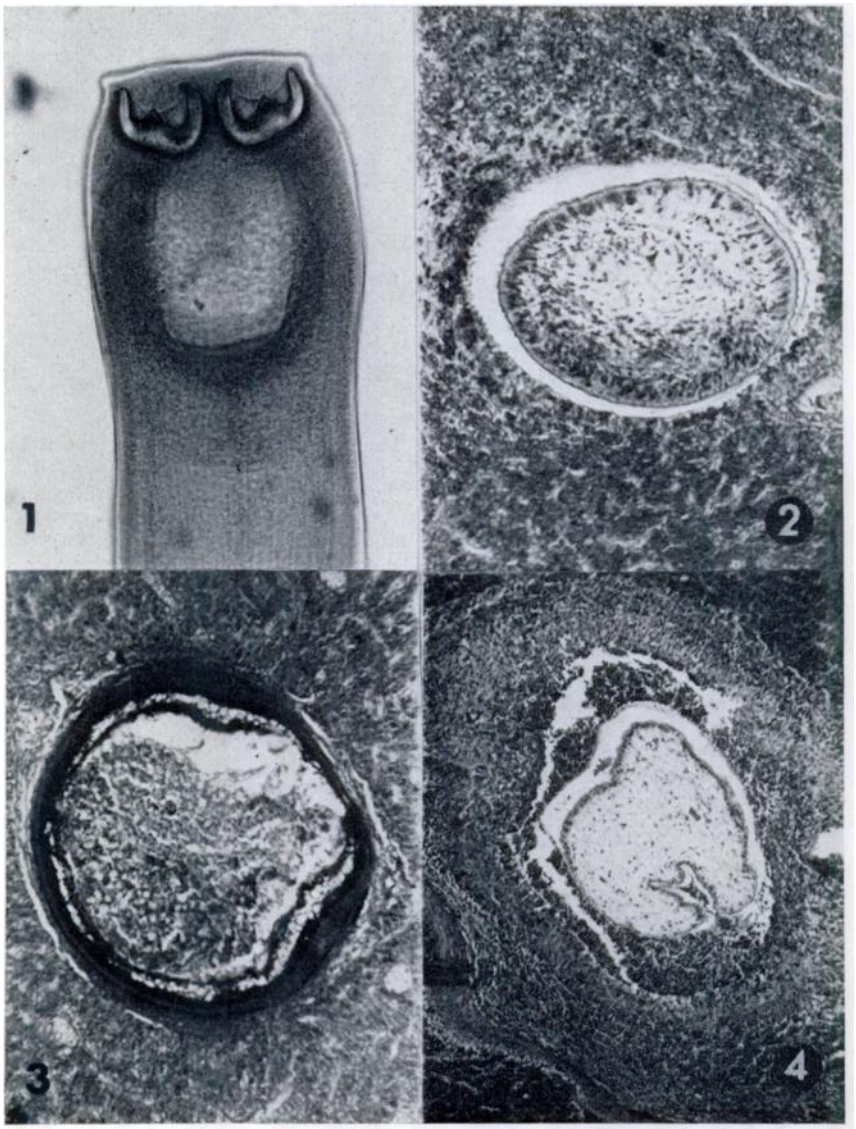

FIGURE 1. Scolex of **Triaenophorus nodulosus** with characteristic hooks. 10X

FIGURE 2. Cross section of living, unencapsulated plerocercoid within liver sinusoid. 20X

FIGURE 3. Cross section of dead plerocercoid surrounded by dense tissue exhibiting non-specific acid fuchsin reaction. 20X

FIGURE 4. Cross section of living plerocercoid with cellular response and surrounded by necrotic liver tissue. 5X

PATHOLOGICAL OBSERVATIONS

1. Gross Lesions (Fig. 5)

Plerocercoid larvae of *T. nodulosus* were frequently observed unencapsulated in the mesenteries and on the surface of the liver. Dissection of infected livers also revealed unencapsulated plerocercoids within the liver. Encapsulated worms were white and occurred most often in the liver, but occasionally in the mesenteries. Infected livers were frequently a pale yellow. Dark, reddish-brown sinuous tracks or "streaks" were visible on the liver surface and on the cut face of fresh livers. Hemorrhages were observed in many infections.

2. Microscopic Lesions

Histological examination revealed larval *T. nodulosus* in the sinusoids and venous circulation of the fish liver. A distinct cellular response was observed around some plerocercoids but not all (Fig. 4). These cells were primarily lymphocytes and macrophages. Destruction of the proximal liver parenchyma, squamous metaplasia and fibrosis were associated with these worms. Many plerocercoids appeared necrotic. These dead worms were frequently surrounded with a dense material which gave a non-specific reaction to acid fuchsin when stained with Mallory's trichrome (Fig. 3). These are the worms which appeared grossly as dark, reddish-brown "streaks". Large plerocercoids which appeared normal were surrounded by capsules (Fig. 6) composed of an outer layer of dense, fibrous connective tissue and an inner cellular layer. Compression atrophy of the liver parenchyma and pancreatic tissue surrounding the portal veins occurred in heavily infected livers. Hemorrhagic tracks were noted throughout infected livers, but not necessarily in close association with plerocercoids.

DISCUSSION

Gross and microscopic examination of white bass livers with moderate to heavy infections of *T. nodulosus* suggest that

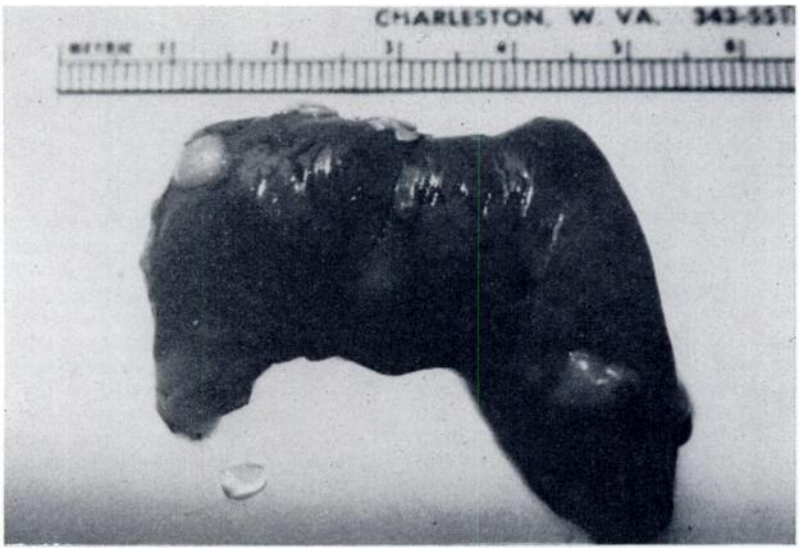

FIGURE 5. Gross appearance of white bass liver with white cysts and dark, sinuous "streaks" characteristic of triaenophoriasis.

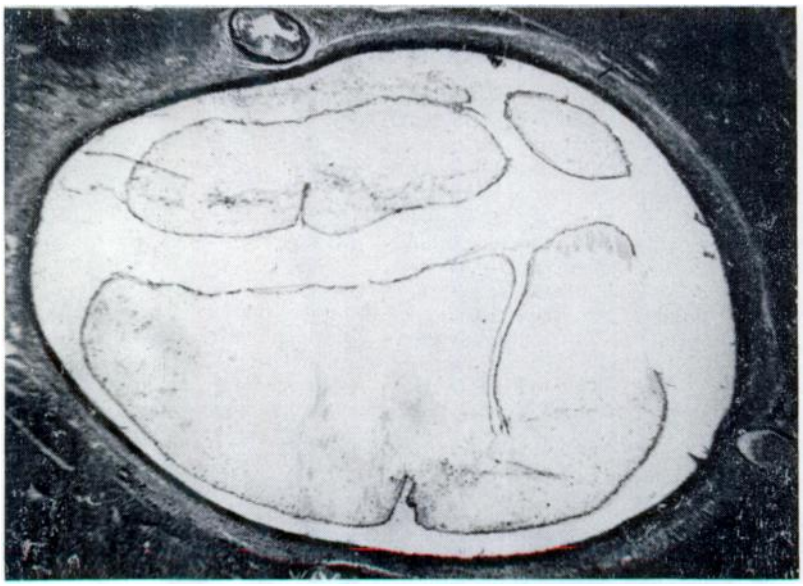

FIGURE 6. Cross section of living, encapsulated plerocercoid. 5X.

there may be serious disease associated with the plerocercoids of this tapeworm. Obstructive liver disease and portal hypertension probably occur, since growing plerocercoids occlude sinusoids, exert pressure on surrounding tissue and obstruct blood and bile flow through the liver. Bauer[3] noted an accumulation of lymphocytes around plerocercoids before encapsulation. This was followed by a hypertrophy of connective tissue fibers and an accumulation of fibroblasts. Some encapsulated plerocercoids died within the capsules. Scheuring[13] speculated that plerocercoids released a substance which destroyed liver tissue, providing a nutrient resource for the tapeworm. Our findings are consistent with these interpretations. We observed necrosis of liver parenchyma surrounding some unencapsulated plerocercoids, indicating perhaps a histolytic secretion of the parasite. The small encapsulated worms, exhibiting the non-specific acid fuchsin reaction, could have been killed in two ways. Miller[11] suggested that the small encapsulations of *T. crassus* represented old, degenerated plerocercoids, and we agree that some of these observed in white bass livers could have been produced in this way. However, some of these small lesions could also be very young plerocercoids which have been killed by the host response.

Although 50% of the adult Lake Erie white bass examined exhibited gross lesions, the prevalence of *T. nodulosus* is probably higher. Shegog[14] reported 80% of the adult white bass infected. We considered only fish with obvious signs of the disease as positive for triaenophoriasis, while Shegog completely dissected livers, thus recording as positive those fish with few worms and no obvious signs. Unfortunately, we did not examine young of the year fish for the disease. These fish might be seriously affected by the tapeworm. Young white bass were shown by Price[12] to feed heavily upon cyclopoid copepods and should be in frequent contact with the infective agents. A single plerocercoid in small fish might be sufficient to cause death. Lawler[9] found 18% of month-old yellow perch (20-30 mm) to be infected. These perch were easily recognized because the ventral

surface was very distended by unencapsulated plerocercoids that displaced 50-90% of the liver tissue.

The increase in prevalence of *T. nodulosus* in progressively older white bass corraborates the findings of Bauer[3] for burbot. Lawler,[6] however, showed a decline in prevalence of worms with increasing age classes of yellow perch. Although the highest percentage of infection was in perch under 114 mm, the greatest intensities of infection occurred in intermediate and large perch. These data suggest that plerocercoids accumulate in fish as they grow older. Bauer[3] suggested that older burbot became infected by eating other fish carrying plerocercoids, but no one to date has demonstrated this transmission pathway. Since Price[12] showed that adult white bass were chiefly piscivorous, it seems likely that these fish could acquire an infection in this manner. This would partially explain the increased prevalence of worms in older Lake Erie white bass.

The impact of triaenophoriasis on Lake Erie fish is undetermined at present. Although considerable research has been done on *T. nodulosus*, little attention has been focused upon the disease caused by the plerocercoids. We need information about the course of the disease and the specific response of the host. An investigation of plasma proteins, glucose, hemoglobin and bilirubin would provide an estimate of how the disease affects liver function. In addition, we need to know how the number of *T. nodulosus* plerocercoids is related to the pathophysiology so that an estimate of the impact of the disease can be made based upon the distribution of the worm in natural populations.

Acknowledgments

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