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THE HISTOPATHOLOGY OF CUTTHROAT TROUT EXPERIMENTALLY INFECTED WITH THE BLOOD FLUKE *Sanguinicola klamathensis*[1]

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Abstract: The pathological effects of the blood fluke, *Sanguinicola klamathensis* Wales 1958, was studied in the cutthroat trout host (*Salmo clarki*). Cutthroat trout fingerlings of a non-infected group and a group experimentally infected by exposure to 6,000 *Fluminicola fusca* snails, with a 6% prevalence of infection with *S. klamathensis*, were maintained for 7 months. The experimental group had 80% mortality after 3 months exposure to the blood flukes. Histopathology revealed a progressive infection with fluke eggs and miracidia within the gills, kidney, and heart as evidenced by necrosis and calcification of heart and kidney tissue, and hyperplasia of the gills.

INTRODUCTION

Relatively little is known of the pathogenicity of the blood fluke *Sanguinicola klamathensis* Wales 1958 within its trout hosts. Only two other studies have been made of this species. One report by Wales,[5] which was the original description of the trematode, contained very little evidence of pathological processes. The other study, by Meade,[3] was primarily concerned with a description of the cercarial stage of the life history.

The life cycle of *S. klamathensis* begins with the adult fluke releasing eggs within the circulatory system of a trout host. Those eggs which drift to the gills, mature to miracidia, and escape by rupturing the gill epithelium, are free-swimming for a few hours, and presumably penetrate a snail of the genus *Fluminicola*. Miracidia develop into sporocysts within the snail's digestive gland. Rediae have not been observed by the author. Presumably, cercariae develop within the sporocyst, later escape from the sporocyst within the snail's digestive gland, leave the snail, and penetrate into the circulatory system of the fish host. These stages then shed their tails and develop into immature flukes, which in turn develop into adult flukes, and make their way throughout the circulatory system. Migration through tissue is characteristic of this species.

Wales[5] reported that an epizootic occurred in Kamloops rainbow trout (*Salmo gairdneri kamloops*) and steelhead rainbow trout (*S. gairdneri gairdneri*) at California's Darrah Springs State Hatchery, Tehama County, in which mortality was estimated at 300,000. This epizootic was attributed to blood flukes, eggs, and miracidia in the gills of the fish.

The presently reported study was undertaken to determine the life history, pathology, and host-parasite relationship of the blood fluke at the Hagerman National Fish Hatchery. The life history part of the study was recently published,[3] while the host-parasite relationship and mortality study are in preparation for submission.

[1] This research was part of the author's recent Ph.D. dissertation.

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MATERIALS AND METHODS

The experimental part of the project was begun on September 15, 1972 and terminated on April 25, 1973 at which time the 13 survivors in the experimental tank were sacrificed for sectioning. Two standard hatchery tanks 83 cm wide, 478 cm long, and 71 cm deep were utilized for the experimental infections and control fish. Each tank was separated into two compartments by aluminum screens with holes 3 mm in diameter. Five hundred non-infected cutthroat trout fingerlings (average length 7.95 cm) were placed into respective tank compartments.

In one hatchery tank no snails were included, while in the other, 4,000 snails of the species *Fluminicola fusca* with a 6% prevalence of infection with *S. klamathensis* were introduced. The snails were separated from the trout by the aluminum screen barrier to prevent the fish from eating them. Twenty-eight days later, another 1,000 infected snails were introduced and after 33 more days, another 1,000 infected snails were added. Algae-coated rocks were placed with the snails to serve as food. The water flow rate was constant at 85 1/min. A 20 watt fluorescent lamp was installed 38 cm above the water surface of each tank to accelerate algal growth. Both lamps remained on during the first 98 days of the experiment at which time the snails were removed from the experimental tank. Parasitism or its absence was confirmed by examining fish from both groups on November 18, 1972.

Samples were taken at three different times: December 21, 1972, January 4, 1973, and April 25, 1973. Tissue samples for histological preparation were obtained from control and experimental fish by removing the two right distal gill arches, the heart, a portion of the liver, and the anterior one-third of the kidney. Tissues were fixed in Bouin's solution for 36 hours and then stored in 70% ethanol. For histologic examinations, all tissues were embedded in paraffin, sectioned at 8μ and stained with hematoxylin and eosin.

RESULTS

The eggs and miracidia of this fluke were found in the same organs in the fish host as were the immature and adult flukes (Table 1). Eggs and miracidia occurred more frequently in the capillaries of the gills (Fig. 1) than in any other part of the circulatory system. Sections of control fish had no lesions (Fig. 2). Sporocysts were observed in the digestive gland and viscera of the snail host, *F. fusca*.

TABLE 1. Locations of blood fluke stages within the intermediate and definitive hosts.

Stage	Site Within Snail Host	Site Within Trout Host
Eggs	Absent	Gills, heart, and kidney
Miracidia	Digestive gland and other viscera	Gills, heart, and kidney
Sporocysts	Digestive gland and other viscera	Absent
Rediae	Not observed	Absent
Cercariae	Digestive gland and other viscera	Fins and epithelium; heart and general circulation
Immature Flukes	Absent	Gills, heart, liver, and kidney
Adult Flukes	Absent	Gills, heart, liver, and kidney

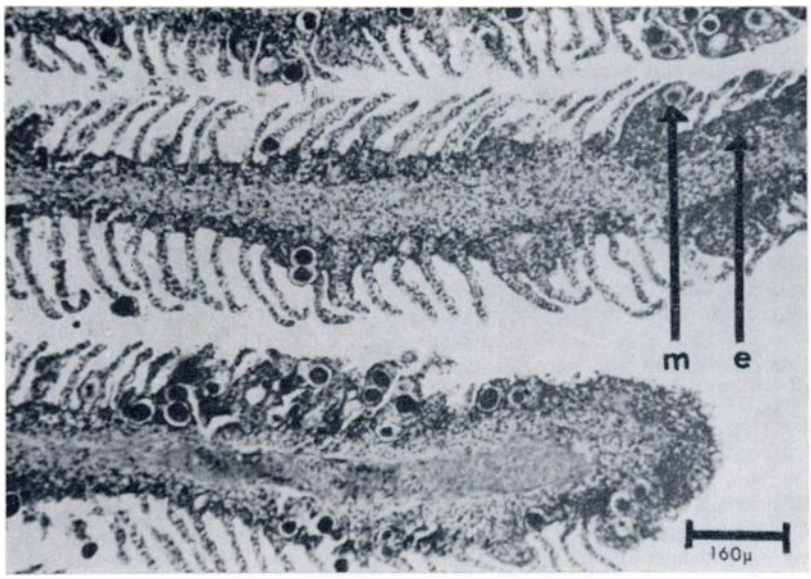

FIGURE 1. Section of gill filaments from cutthroat trout following 222 days exposure to infection with **Sanguinicola klamathensis** showing egg (e) and miracidium (m).

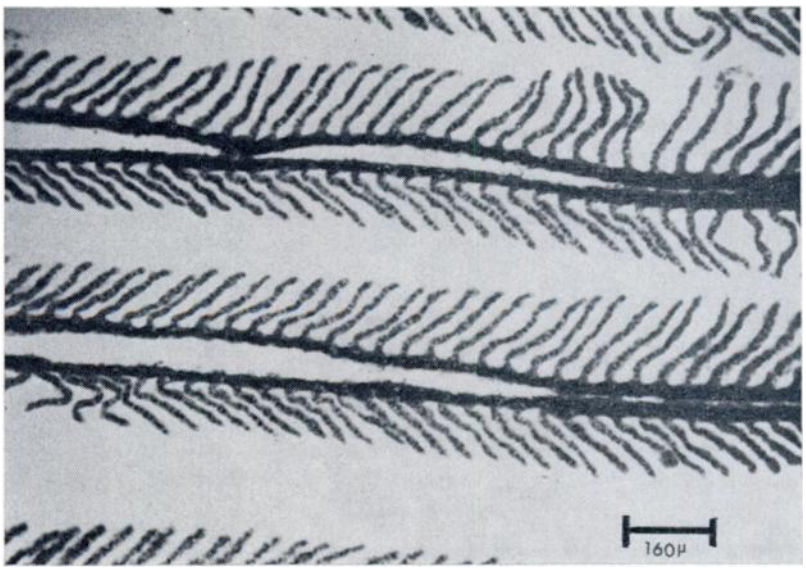

FIGURE 2. Section of gill filaments from control (noninfected) cutthroat trout.

A progressive change of the gill filaments of experimentally infected fish was observed over the 7 month period. These changes were represented by fluke eggs and miracidia within the capillaries of gill lamellae. Hyperplasia, chronic inflammation of the gill epithelium, and fusion or "clubbing" of lamellae were also noted (Fig. 3).

Histological sections of the gills, heart, and kidney of infected fish showed eggs only in the December 21, 1972 sample; eggs and miracidia in the January 4, 1973 sample; and eggs, miracidia, and extensive calcification and necrosis in the April 26, 1973 sample.

Blockage of gill capillaries by eggs and miracidia was observed with a dissecting microscope in infected cutthroat trout anesthetized with MS-222 (tricaine methane-sulphonate). While the filaments were moistened with spring water, the blood was observed circulating through the lamellae without eggs, whereas in those lamellae which were blocked by eggs, circulation was prevented.

Sections through the heart of infected fish disclosed encapsulated eggs and miracidia in the ventricle. Sections of control fish hearts had no lesions. In one heart section an immature fluke was embedded within the musculature of the ventricle. During several examinations of infected fish hearts an average of two or three immature flukes were recovered after teasing the heart muscles apart. One heart section had an immature and adult fluke free within the bulbous arteriosus.

Damage to the kidney consisted of necrosis or hypertrophy of renal epithelial cells. Eggs and miracidia were also observed in large numbers encapsulated in the kidney tissue (Fig. 4). No lesions were present in kidneys from control fish. An adult fluke was observed embedded within hematopoietic tissue of one preparation.

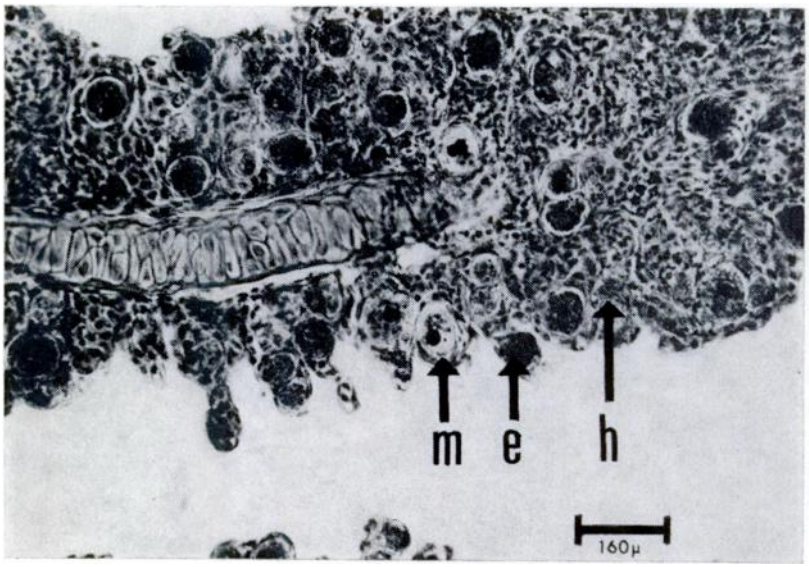

FIGURE 3. Section of gill filaments from cutthroat trout infected with **S. klamathensis.** Similar to figure 1 except at higher magnification showing egg (e), miracidium (m), and hyperplasia (h).

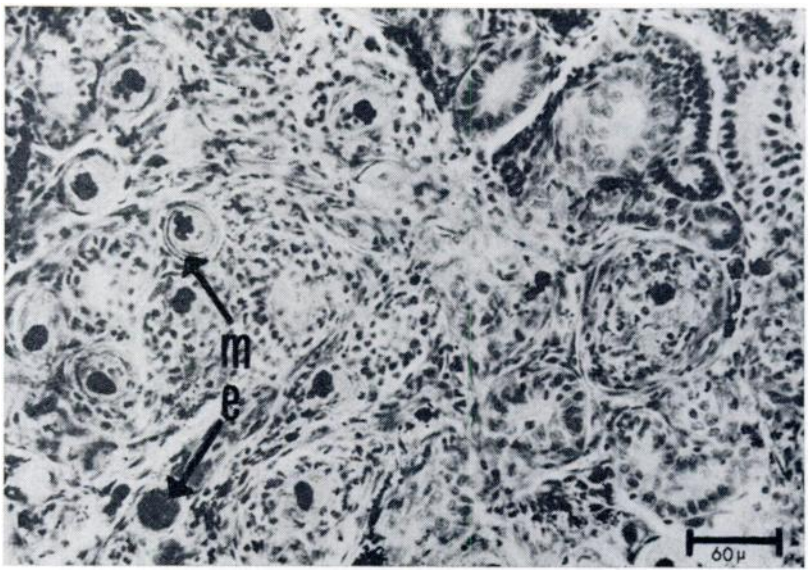

FIGURE 4. Section of kidney from cutthroat trout following 222 days exposure to infection with **S. klamathensis** showing egg (e) and miracidium (m).

No stages of the parasite were found in sections of the liver. In fresh, dissected preparations, adults were recovered from the liver.

DISCUSSION

According to Bauer et al.,[1] the blocking of gill capillaries by *Sanguinicola* eggs causes necrosis of the gills. Blockage of kidney blood vessels by uneliminated eggs causes water imbalance and exudate accumulation resulting in edema.[1] Cysts composed of connective tissue form around eggs and miracidia.[1] Schaperclaus[4] indicates that eggs of *S. inermis* can be present in such a large number that they clog the gill capillaries, bring about thrombosis, and lead to death of the fish.

In the present studies it was noted that hyperplasia and inflammation of the gill epithelium due to the presence of eggs and miracidia reduce functional gill volume. Blockage of circulation in the major arteries of the gills can occur when numerous immature flukes are present. Migration of flukes through the hematopoietic tissue of the kidney and encystment of eggs and miracidia in the same organ contribute to reduced erythropoiesis and possibly renal failure.

It is apparent that this blood fluke is very devastating to its trout hosts and that it can be established in new areas provided the snail host is present and infected trout are stocked.

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