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SPIRORCHIDIASIS IN LOGGERHEAD SEA TURTLES *(CARETTA CARETTA)*: PATHOLOGY [1]

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Abstract: Loggerhead sea turtles *(Caretta caretta)* from the Atlantic seaboard (Florida to Massachusetts) were examined at the Marine Pathology Laboratory, University of Rhode Island, from March through December, 1980. Three genera of blood flukes (spirorchids) were found in 14 (33%) of the 43 turtles. Gross signs in heavily infected animals included cachexia, anemia and enteritis. Histopathological lesions were similar to those present in homeotherms with schistosomiasis. Granulomatous gastritis, enteritis, hepatitis, pneumonitis, and nephritis were present. Acute and chronic vasculitis accompanied metastasis of eggs. Infected animals had severe hepatic hemosiderosis, indicative of the anemia observed grossly. Evidence is presented that spirorchidiasis is prevalent in sub-adult loggerhead sea turtles, is responsible for extensive lesions and may be responsible for significant debilitation and mortality.

INTRODUCTION

Intravascular trematodes (blood flukes) are pathogens of mammals, birds, reptiles and fish. Those affecting homeotherms include the families Schistosomatidae and Ornithobilharziidae while those affecting poikilotherms are found in the families Sanguinicolidae and Spirorchidae. There are 16 genera within the family Spirorchidae, all of which infect turtles. Of those genera, eight are found in sea turtles of the family Cheloniidae (Smith, 1972). Species representing three genera, *Carettacola*, *Hapalotrema*, and *Neospirorchis* have been reported in the heart or blood vessels of the loggerhead turtle *Caretta caretta* (Monticelli, 1896; Looss, 1898; Luhman, 1935; Manter and Larson, 1950).

Signs of infection in mammals and birds include listlessness, debilitation, cachexia, anemia, secondary bacterial infections and death. Gross and histopathological examination reveal vasculitis, perivascular hemorrhage, granulomatous cystitis, enteritis, pneumonitis, hepatitis and cirrhosis (Brown, 1968; Davis and Libke, 1971). The effect of these helminths on the health of fresh water turtles and the green turtle has been documented (Holliman and Fisher, 1968; Holliman, et al., 1971; Smith, 1972; Greiner, et al., 1980; Glazebrook and Campbell, 1981). The pathogenicity and prevalence of spirorchidiasis in the loggerhead sea turtle has not been reported.

The Endangered Species Act of 1973, as amended (16 U.S.C. 1531-1543) recognizes *C. caretta* and other members of the Cheloniidae and Dermochelyidae *(Chelonia mydas, Eretmochelys imbricata, Dermochelys coriacea* and *Lepidochelys spp.)* as either threatened or endangered species. The literature is

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replete with articles documenting their decline in numbers. The reasons for the decline appear manifold and include natural predation and land development of former nesting beaches. Turtles or their eggs are taken for food, during commercial fishing activities, or maliciously (Ogren et al., 1977; Seyfert, 1978).

This precipitous decline in sea turtle numbers has awakened an interest in the use of classical anatomic pathology to describe their diseases and their prevalence and to determine cause of death. Therefore, sea turtles are being examined on a regular basis at the Marine Pathology Laboratory, College of Resource Development, University of Rhode Island. The purpose of this paper is to describe the lesions associated with spirorchidiasis in *Caretta caretta*.

MATERIALS AND METHODS

From March through December 1980, 43 loggerhead sea turtles were submitted to the Marine Pathology Laboratory, University of Rhode Island. Twenty-seven of the accessions were submissions of random tissues (20 gastrointestinal only) and 16 were complete animals. All specimens collected were found dead and the majority were ashore.

Turtles and their tissues came from areas about the Chesapeake Bay, Virginia; Green Hill, South Carolina; Cape Cod, Massachusetts; Cumberland Island, Georgia; and the East and West coasts of central Florida.

Tissue was fixed in buffered 10% formalin, embedded in paraffin and sectioned at 6 μm. Sections were routinely stained by hematoxylin and eosin. Special stains included Perl's method for iron, Periodic Acid Schiff, Toluidine Blue-O, and Grocott Silver method (Luna, 1968).

Eggs were isolated from the intestine by scraping with a scalpel or by fecal flotation using a supersaturated solution of glucose.

Fixed mesenteric vessels were examined for adult parasites with low magnification light microscopy. Blood clots were removed from the lumina and teased apart to free the intravascular trematodes.

RESULTS

Spirorchidiasis was diagnosed in 14 of the 43 accessions (33%). Sex could not be determined in nine animals; three were males and two females. Mean straight carapace length was 62.2 cm in all turtles and 62.8 cm among affected animals. Geographic source of the animals and number affected were: Florida 8, 3 affected; Georgia 20, 5 affected; Virginia 6, 3 affected; South Carolina 6, 4 affected; and Massachusetts 3, 0 affected.

Gross Pathology: The severity of disease varied from no apparent lesions in animals lightly affected to severe cachexia in those with a heavy burden of eggs.

In advanced stages of the disease, the turtles were extremely cachexic and appeared shrunken within their shells. In such animals the supraoccipital process was sharply outlined and, in recently dead animals, the central plastron area was markedly depressed. In addition, these animals carried an excessive number of barnacles, especially on the skin.

When the plastron was removed from severely affected animals, three lesions were immediately apparent. First, the normal yellow and green fat overlying the viscera was absent or, if present, was clear and of a jelly-like consistency (mucoid degeneration). Second, all organs and membranes were pale and lacked their normal coloration. Third, the sero-sanguinous reddish fluid commonly found in the carapace at necropsy was decreased in amount and clear rather than pink to red. These signs are compatible with a diagnosis of cachexia and suggest anemia.

Raised linear lesions 2-10 cm long and 0.2-0.5 cm wide were present on occasion

in the hindgut mucosa (colon). The areas tended to be greenish-black, dry and friable. When the gut was cut transversely, brownish-yellow, gritty areas were often present between or in the muscularis interna/muscularis externa. Microscopic examination of scrapings from these areas revealed masses of eggs.

Spirorchid eggs were also recovered from scrapings of fixed intestinal mucosa. The eggs were of three types and fit the description of eggs from spirorchids known to infect marine turtles, including loggerhead sea turtles. Type 1, resembling eggs of the genera *Learedius, Monticellius,* and *Hapalotrema,* were yellow-brown, elongate and bore two processes, one of which was frequently hooked. A miracidium was present in the central oval portion of the egg. The formalin-fixed eggs were approximately 276 by 37 μm (Fig. 1).

Type 2 eggs were ovoid with a sharp short terminal process ($\pm$0.8μm). The formalin-fixed eggs were approximately 135 μm long and 67 μm wide (Fig. 1). In some instances, miracidia were present. These eggs are similar to those of the genera *Carettacola* and *Haemoxenicon.*

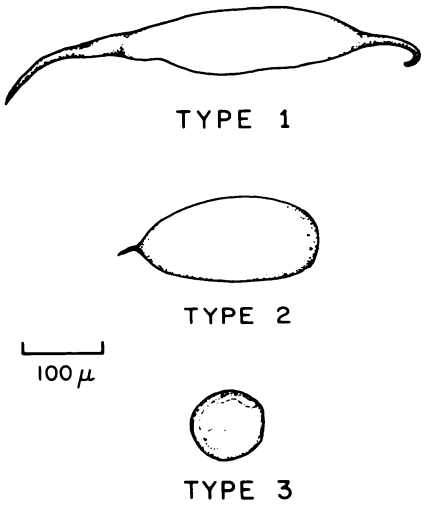

FIGURE 1. Drawings of fecal spirorchid eggs Type 1: *Learedius, Monticellius;* Type 2: *Carettacola, Haemoxenicon;* Type 3: *Neospirorchus.*

The third type of egg was smaller and rounder than the other two and lacked a process. Its dimensions after fixation were approximately 45 by 30 μm. It was also yellow-brown and miracidia were easily visualized within the shell. These eggs most resembled those of the genus *Neospirorchis* (Fig. 1).

In three instances, two types of eggs were recovered from intestinal scrapings, indicating dual infections. In two cases, type 1 and type 3 eggs were found concurrently; the other turtle had types 1 and 2.

It was sometimes difficult to differentiate histopathologically among the three types. Type 1 eggs were most easily identified, especially in acute infections. However, in more chronic infections when the egg shell had begun to disintegrate due to inflammatory response, Type 1 eggs lost their differentiating characteristics while differentiation between *Neospirorchis* and *Haemoxenicon* was impossible unless the terminal process was apparent.

Eggs and their associated inflammatory responses were present in the following organs, listed in descending frequency of infection: hindgut, foregut, liver, spleen, kidney, heart, stomach, and testis. No eggs were observed in the brains of five animals examined.

The inflammatory response to the eggs and their migrations reflected the chronicity of the infection. These lesions were consistent in their appearance regardless of organ. Over 90% of the lesions were best classified as focal granulomata. The remaining lesions occurred in one animal and were more acute.

The migrating eggs initially elicited a mixed inflammatory response with lymphocytes and a few granulocytic cells found close to eggs bearing miracidia or free of miracidia but still whole and not

ruptured. The cells closest to the ova often were necrotic, and karyorhexis was so extensive that, under low power examination (63×), the cells resembled polymorphonuclear leucocytes found in homeotherms. The granulocytic cell was large, had an eccentric nucleus and large, weakly pink to yellowish refractile granules best observed when the microscope's stage condenser was lowered. The granules were red when stained by the Giemsa method. These granulocytes comprised less than five percent of the inflammatory cells and were often present in the lumen of vessels surrounding the area.

The granulomata were of two types: those which contained a central or pericentral zone of caseous necrosis and those which did not. In the first type, degenerating ova were surrounded by a zone of acidophilic caseous necrosis often bordered by giant cells of the foreign body type. These giant cells were often surrounded by a fibrous capsule, peripheral to which were lymphocytes, histiocytes and a few granulocytes (Fig. 2). Granulocytes undergoing necrosis were also apparent just inside the zone of caseation.

The second type of granuloma lacked the zone of caseation and was characterized by histiocytes, lymphocytes and giant cells (Fig. 3). The giant cells frequently engulfed disintegrating eggs. The thickness of the zone of surrounding cells was variable. Epithelioid cells and granulocytes were not seen.

The eggs reached their locations via blood vessels and their passage through the vessel walls resulted in vasculitis and

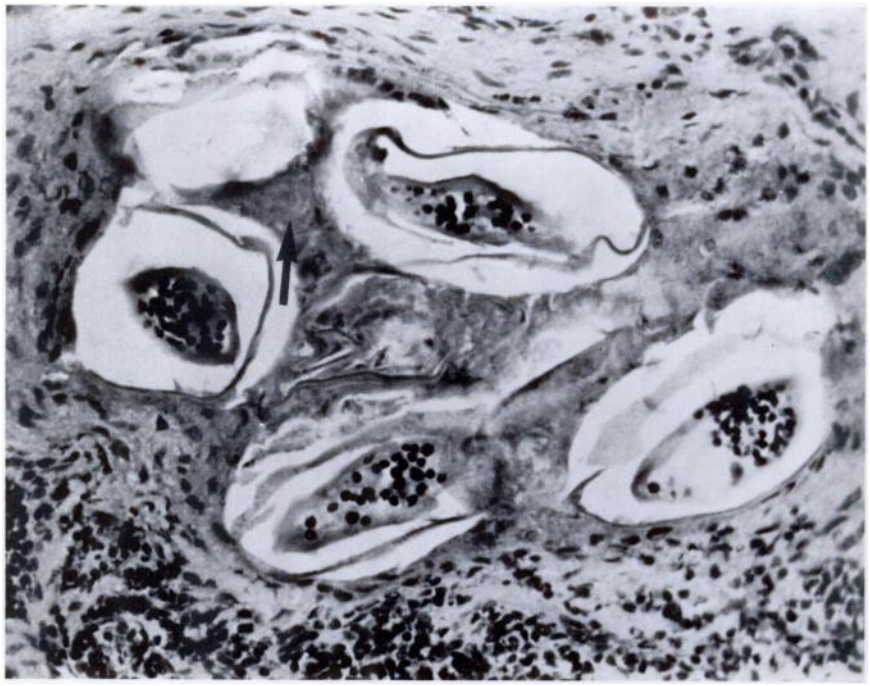

FIGURE 2. Spirorchid eggs in wall of colon. Granuloma characterized by caseous necrosis (arrow), reticuloendothelial cells and lymphocytes. H&E ×160.

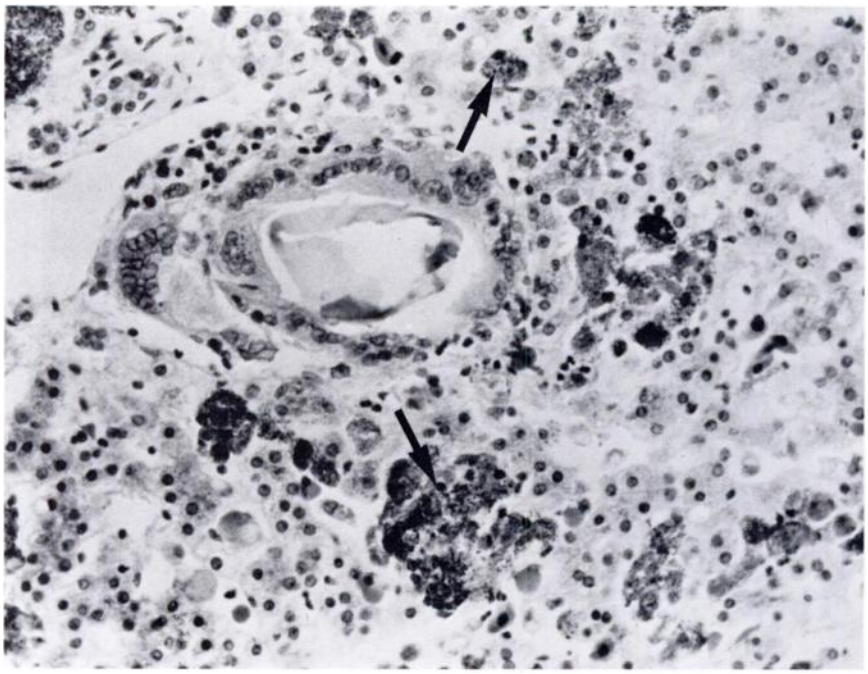

FIGURE 3. Spirorchid egg in liver. Focal granuloma with foreign body giant cell below egg. Hepatocytes and melano-macrophage centers laden with hemosiderin (arrows). H&E ×160.

perivasculitis. The wall of the vessel was ruptured at the point of exit and there was little inflammatory response within the vessel layers. However, the surrounding tissue was infiltrated by lymphocytes and a small amount of granulocytes (Fig. 4).

Focal accumulations (20-500 μm) of yellow to black pigment within macrophages were present in the spleen, liver and kidney of normal animals. These accumulations are similar, if not identical to the melanomacrophage centers or mictic corpuscles[☑] of higher fish (Roberts, 1975). They are greatly increased in number in the livers of affected animals. When stained by the Prussian Blue method for hemosiderin they were strongly positive. In addition, much hemosiderin was diffusely present in the livers of affected animals, especially in hepatocytes. Much more hemosiderin was in the livers of infected than of uninfected animals (Fig. 3).

No adult spirorchids were recovered from the mesenteric vessels. However, an adult blood fluke was found in section in an artery of the muscularis interna of the colon, in close proximity to masses of eggs. The heart and great vessels were examined by routine necropsy procedures but were not examined carefully for adult parasites.

[☑] C.J. George, Department of Biological Sciences, Union College, Schenectady, NY 12308, Pers. Commun., September, 1980.

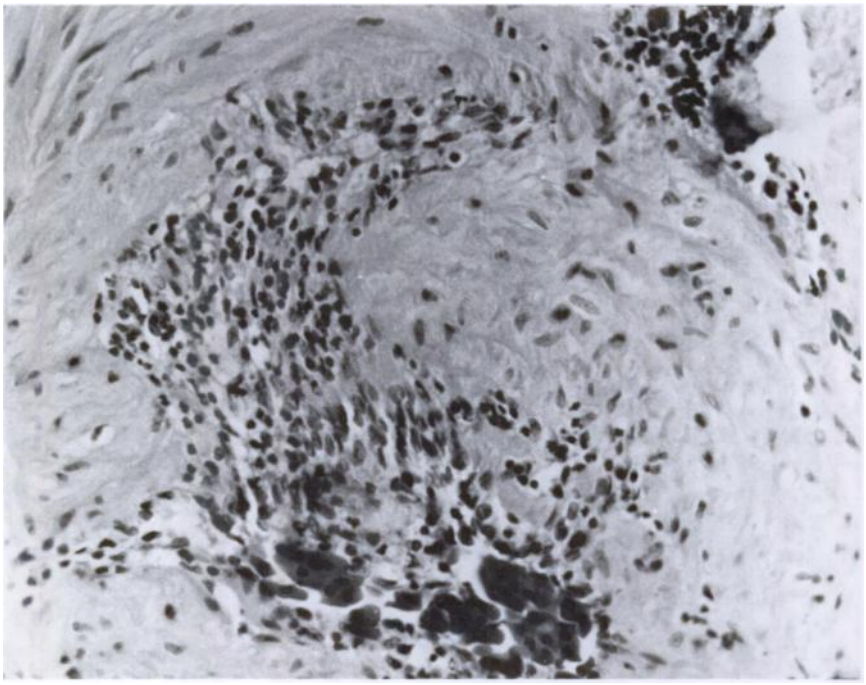

FIGURE 4. Muscularis externa, colon, vessel at bottom. Perivascular inflammation and migration tracts of eggs through surrounding tissue. H&E ×160.

DISCUSSION

The effects of blood flukes on the population dynamics of wild turtles has not been investigated, nor has the incidence or prevalence of this disease in free-living sea turtles been determined. It is therefore of interest that the prevalence of infection as determined by this study exceeded 30%. Reports of infection rates in fresh water turtles (where N<20) are lower, approximating 3 to 13 percent (Ulmer, 1957; Esch and Gibbons, 1967). This disparity may be explained by the uniformity of size in the sea turtles examined. All were subadults with a mean carapace length of 62.8 cm while studies conducted with fresh water turtles included animals of various sizes and ages. However, the disparity may also reflect means of collection. The prevalence of a serious pathogenic disease would probably be higher in a population of dead animals.

The relationship of infection rate and size/age is not an uncommon phenomenon in homeotherms. Infection rate is certainly related to parasite life cycle and to the degree of immunity. The immune response is less in younger animals and increases, with corresponding decreases in infection rate, in older animals. This phenomenon has been reported for spirorchidiasis and nematodiasis in *Chrysemys* sp. and seems a logical explanation for the high percentage of infection in *C. caretta* (Stunkard, 1923; Wall, 1941; Esch and Gibbons, 1967). However, since intravascular trematodes in higher vertebrates may acquire host antigen on their tegument and thereby persist for long periods of time as might their eggs,

it is difficult to draw positive conclusions on age-related incidence of spirorchidiasis without further investigation.

Parasitism by blood flukes may lead to serious disease in all species of animals affected. In man and other mammals, schistosomiasis is a major cause of debilitation and death. In higher vertebrates the disease is chronic, leading to severe diarrhea, anemia and a plethora of secondary infections of the urinary and gastrointestinal tracts. Similarly, the Ornithobilharziidae are pathogenic to birds and the Sanguinicolidae to fish. In sanguinicolid infections, thrombosis and hemorrhage of branchial vessels has been reported (Schäperclaus, 1954; Davis et al., 1961).

When the disease is experimentally produced in fresh water turtles, lesions and signs are multiple and widespread. These include central nervous system signs (circling, hemiplegia), debility, ascites, granulomata, listlessness, stupor, and death (Goodchild and Denis, 1967; Holliman and Fisher, 1968). These signs and lesions appear to be caused by three factors: 1) egg migration, 2) presence of the adult worm, and 3) secondary infection.

Blood fluke eggs pass from their origin intravascularly through the endothelium and vessel wall into the surrounding tissue. During this passage, vessel wall integrity is violated and localized hemorrhage is common. The eggs then begin a migration that will result in their reaching the lumen of the intestine. This in turn results in a severe and acute inflammatory response and subsequently enteritis. Many eggs are deposited in the blood vessels of organs other than the gastrointestinal tract. These eggs migrate but are unsuccessful in leaving the host. Initially, they elicit an acute response but eventually granulomata are formed. This chronic reaction can lead to extensive tissue destruction and, in the liver, to cirrhosis. When one considers that the average thickness of a histopathological section is 6 μm and the masses of eggs that may be present within it, then it is not difficult to imagine the vast numbers of eggs present in various organs (Goodchild and Denis, 1967).

The adult worm is less pathogenic, but has been reported responsible for the formation of thrombi and infarction (Davis and Libke, 1971; Glazebrook and Campbell, 1981). In some instances the adult worm may leave the vessel lumen and pass to the surrounding tissue with consequent hemorrhage and inflammation. Adults of *Spirorchis haematobium* have been reported outside vessels in the periesophageal tissue of *Chrysemys picta* (Ulmer, 1957). *S. elegans* normally occurs in the esophageal submucosa and *S. scripta* is found in the cranial cavity (Schroeder and Ulmer, 1959; Brooks and Mayes, 1975, 1976; Brooks, 1979).

An important cause of blood fluke pathogenicity is secondary infection. The passage of eggs through lung parenchyma to alveolar spaces or through intestinal tissue to the lumen violates the lining epithelium and allows invasion by bacteria and other organisms. Reports of pneumonitis, enteritis and cystitis are not unusual in association with blood-fluke infection. Further, constant irritation of the gastrointestinal tract results in diarrhea, starvation, fluid imbalance, dehydration and death.

Many of the lesions present in turtles examined in this study certainly found their origin in egg migration and probable secondary infection. The gastrointestinal tracts of heavily infected animals were empty and acutely inflamed. Granulomata were common and the gross signs of anemia evident. In severely cachexic and anemic animals, it is not difficult to attribute death to spirorchid parasitism. But even in those animals in good flesh, there was evidence that bacterial enteritis was present and secondary to egg migration.

The histopathological appearance and inflammatory response to spirorchid

eggs in *C. caretta* is strikingly similar to that in primates. Response to schistosome eggs in homeotherms has been classified in five stages on the basis of age and cellular response. These stages include 1) non-reactive or weakly reactive; mononuclear response, 31 days post-infection, 2) exudative; neutrophilic, eosinophilic response, 34 days post-infection, 3) exudative-productive; granulocytic cell necrosis, 42 days post-infection, 4) productive; absence of granulocytes, epithelioid-giant cell response, 45 days post-infection, and finally, 5) involutional; cellular and egg degeneration, fibrosis, and calcification (Hsu et al., 1969; Hsu et al., 1972). While the turtle's response is similar, time to each stage appears different. The life cycle of spirorchids in the sea turtle is unknown but is probably similar to that of fresh water turtles. From infection to the presence of first egg deposition is approximately 55 to 60 days so that the weakly reactive stage would not be present for at least an equal period (Goodchild and Kirk, 1960; Goodchild and Denis, 1967; Holliman and Fisher, 1968).

Lesions in loggerhead turtles resembled the exudative, exudative-productive and productive stages. In the first instance, eggs bearing miracidia were surrounded by granulocytes with large weakly eosinophilic granules. These cells were circulating cells and were commonly present in blood vessels. Their nucleus was eccentric and single lobed. They resembled the tissue eosinophilic granulocytes of fish. However, the turtle granulocyte is reported to be peroxidase positive, while the TEG of the fish is not. The turtle granulocyte is probably more closely related to the polymorphonuclear leucocyte (neutrophil) of homeotherms. No other eosinophilic cell was apparent in sections stained by hemotoxylin and eosin or the Giemsa method. The granules of the cells present were Periodic Acid Schiff negative.

As in homeotherms, the exudative-productive stage lacked the granulocytic cells and there was an increase in the numbers of macrophages and other mononuclear cells. However, epithelioid cells were not present.

Advanced lesions best fit the classification of the productive stage. Large macrophages and foreign body giant cells were in close contact with degenerating eggs. Frequently the eggs were within giant cells. The cells were surrounded by a rim of lymphocytes. In all instances, the responses were considered granulomata.

The degree of hemosiderosis in infected turtles suggests that hemolytic anemia may be a significant factor in spirorchid infections. This should be clarified by appropriate clinical studies.

The type 1 and 2 eggs appear similar to the eggs of *Hapalotrema* spp. and *Carettacola* spp. known from *Caretta caretta* in Florida, but are 7 and 2 times longer, respectively. Type 3 eggs correspond both in size and morphology to those of *Neospirorchis pricei* which Manter and Larson (1950) reported from *C. caretta* in Tortugas, Florida. Type 1 eggs, those of *Learedius* or *Monticellius* spp. in size and morphology, and type 2 eggs are most compatible with *Haemoxenicon* spp.

Because *Hapalotrema synorchis* Luhman (1935) and *Carettacola bipora* are known to occur in *Caretta caretta* from Florida, and species of *Learedius, Monticellius,* and *Haemoxenicon* have been reported in *Caretta,* we thought the type 1 and 2 eggs found in intestinal scrapings might be more mature eggs of *H. synorchis* and *C. bipora* than those normally found *in utero* in mature worms. Spirorchiids and schistosomes have short uteri containing only small numbers of eggs. Faust and Russel (1964) reported that eggs of *Schistosoma japonicum* increase in size from 67 μm to 70-100 μm in length after leaving the uterus, an increase in length of no more

than 50%. The eggs we found which correspond to those of *Neospirorchis pricei* were 45 μm long as compared with 36-40 μm in length for uterine eggs reported by Manter and Larson (1950) — an increase of 12-25%. Therefore, we assign type 1 eggs to *Monticellius/Learedius* and type 2 eggs to *Haemoxenicon* on a provisional basis pending examination of adult worms from turtles.

This study presents evidence that many subadult *Caretta caretta* have a parasitic disease which is responsible for extensive lesions. While clinical evidence is lacking, there is convincing pathological evidence that these lesions are responsible for significant mortality. Further, the disease is debilitating and may so weaken affected animals that their ability to escape predators, resist infections, or to withstand long periods of submergence (anoxia) may be greatly decreased.

Control is not possible now. Such control would require an understanding of the spirorchid life cycle and identification of the invertebrate intermediate host. A drug (amoscanate) has been developed in recent years which has been used to treat schistosomiasis in man and laboratory animals (Anonymous, 1981). It can be given as a single oral dose. The treatment of female turtles during nesting is worthy of consideration. However, considerable research must be done to determine efficacy and side effects in turtles.

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LITERATURE CITED

ANONYMOUS. 1981. Snail Fever. The Sciences. 21: 4-5.

BROOKS, D.R. 1979. New records for amphibian and reptile trematodes. Proc. Helm. Soc. Wash. 46: 286-289.

—— and M.A. MAYES. 1975. Platyhelminths of Nebraska turtles with descriptions of two new species of spirorchids (Trematoda: Spirorchiidae). J. Parasitol. 61: 403-406.

—— and ——. 1976. *Telorchis gutturosi* sp. n. (Trematoda: Telorchiidae) from *Graptemys pseudogeographica* Gray in Nebraska, with reports of additional species of trematodes from Nebraska turtles. J. Parasitol. 62: 901-905.

BROWN, H.W. 1968. Diseases caused by Metazoa. In: *Textbook of Medicine*, Vol. I, 12th edition, R.B. Beeson and W. McDermott (eds). W.B. Saunders Co., Philadelphia, Pennsylvania, 835 pp.

DAVIS, H.S., G.L. HOFFMAN and E.W. SURBER. 1961. Notes on *Sanguinicola davisi* (Trematoda, Sanguinicolidae) in the gills of trout. J. Parasitol. 47: 512-514.

DAVIS, J.W. and K.G. LIBKE. 1971. Trematodes. In: *Parasitic Diseases of Wild Animals*. J.W. Davis and R.C. Anderson (eds). Iowa State University Press, Ames, Iowa, 364 pp.

ESCH, G.W. and J.W. GIBBONS. 1967. Seasonal incidence of parasitism in the painted turtle *Chrysemys picta marginata* Agassiz. J. Parasitol. 53: 818-821.

FAUST, E.C. and P.F. RUSSELL. 1964. *Craig and Faust's Clinical Parasitology*, 7th ed., Lea and Febiger, Philadelphia, Pennsylvania, 1099 pp.

GLAZEBROOK, J.S. and S.F. CAMPBELL. 1981. Pathological changes associated with cardiovascular trematodes (Digenea: Spirorchidae) in a green sea turtle *Chelonia mydas* (L). J. Comp. Path. 91: 361-368.

GOODCHILD, C.G. and D.E. KIRK. 1960. The life history of *Spirorchis elegans*. Stunkard 1923 (Trematoda: Spirorchiidae) from the painted turtle. J. Parasitol. 46: 219-229.

—— and E.S. DENIS. 1967. Comparative egg counts and histopathology in turtles infected with *Spirorchis* (Trematoda: Spirorchiidae). J. Parasitol. 53: 38-45.

GREINER, E.C., D.J. FORRESTER and E.R. JACOBSON. 1980. Helminths of mariculture-reared green turtles *(Chelonia mydas mydas)* from Grand Cayman, British West Indies. Proc. Helm. Soc. Wash. 47: 142-144.

HOLLIMAN, R.B. and J.E. FISHER. 1968. Life cycle and pathology of *Spirorchis scripta* (Stunkard 1923) (Digenea: Spirorchiidae) in *Chrysemys picta picta*. J. Parasitol. 54: 310-318.

——, —— and J.C. PARKER. 1971. Studies on *Spirorchis parvus* (Stunkard 1923) and its pathological effects on *Chrysemys picta picta*. J. Parasitol. 57: 71-77.

HSU, H.F., J.R. DAVIS, and S.Y. LI HSU. 1969. Histopathological lesions of rhesus monkeys and chimpanzees infected with *Schistosoma japonicum*. Z. Tropen. Med. Parasitenkd. 20: 184.

HSU LI, S.Y., H.F. HSU, G.L. LUST and J.R. DAVIS. 1972. Organized epithelioid cell granulomata elicited by schistosome eggs in experimental animals. J. Reticuloendothel. Soc. 12: 418-435.

LOOSS, A. 1899. Weitere Beiträge zur Kenntnis der Trematoden-Fauna Aegyptens, zugleich versuch einer natürlichen Gleiderung des Genus *Distomum* Retzius. Zool. Jb. Abt. Syst. 12: 521-784.

LUHMAN, M. 1935. Two new trematodes from the loggerhead turtle *(Caretta caretta)*. J. Parasitol. 21: 274-276.

LUNA, L.G. 1968. *Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology*. 3rd ed., McGraw-Hill Book Co., New York, New York, 258 pp.

MANTER, H.W. and M.I. LARSON. 1950. Two new blood flukes from a marine turtle, *Caretta caretta*. J. Parasitol. 36: 595-599.

MONTICELLI, F.S. 1896. Di un ematozoo della *Thalassochelys caretta* Linn. Int. Mschr. Anat. Physiol. 13: 141-172.

OGREN, L.H., J.W. WATSON JR., and B.A. WICKHAM. 1977. Loggerhead sea turtles, *Caretta caretta* encountering shrimp trawls. MFR paper 1270. Mar. Fish. Rev. 39: 15-17.

ROBERTS, R. 1975. Melanin-containing cells of the teleost fishes and their relation to disease. In: *The Pathology of Fishes*. W.E. Ribelin and G. Migaki (eds). University of Wisconsin Press, Madison, Wisconsin, 1004 pp.

SCHÄPERCLAUS, W. 1954. *Fischkrankheiten*. 3rd ed. Akademia-Verlag, Berlin, West Germany, 708 pp.

SCHROEDER, P.J. and M.J. ULMER. 1959. Host-parasite relationships of *Spirorchis elegans* Stunkard (Trematoda: Spirorchiidae). Proc. Iowa Acad. Sci. 66: 443-454.

SEYFERT, F. 1978. The plight of the loggerhead. Sea Frontiers. 24: 19-22.

SMITH, J.W. 1972. The blood flukes (Digenea: Sanguinicolidae and Spirorchidae) of cold-blooded vertebrates and some comparisons with the schistosomes. Helminthol. Abst., Series A 41: 161-204.

STUNKARD, H.W. 1923. Studies on North American blood flukes. Bull. Am. Mus. Nat. Hist. 48: 164-221.

ULMER, M.J. 1957. Notes on *Spirorchis haematobium* Stunkard 1922 (Trematoda: Spirorchiidae) in the definitive host. (Abstract) J. Parasitol. 43: 32.

WALL, L.D. 1941. *Spirorchis parvus* (Stunkard): Its life history and development of its excretory system (Trematoda: Spirorchiidae). Trans. Am. Microsc. Soc. 60: 221-260.

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