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Eurytrema procyonis, a Pancreatic Fluke of North American Carnivores [1] [2] [3]

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Abstract

Eurytrema procyonis is reported from the pancreatic ducts of domestic cats from North Carolina and Virginia. There were extensive histopathological alterations to the pancreas, especially the pancreatic ducts. However, there was no clinical evidence of infections. The incorporation of a synanthropic host in the life history of *E. procyonis* allows for rapid distribution of this fluke over its potential geographical range.

Introduction

Eurytrema procyonis Denton, 1942 was described from specimens taken from the pancreatic ducts of raccoons, *Procyon lotor* (Linnaeus), in Texas.[9] It has since been reported from raccoons in Texas,[7,10] Virginia,[12] North Carolina,[12] Maryland,[13] Connecticut,[18] and Georgia.[1,20] *Eurytrema procyonis* was also found in a red fox, *Vulpes vulpes* Linnaeus, from New York[22,23] and in grey foxes, *Urocyon cineroargenteus* (Schreber), from Maryland[13] and North Carolina.[16] Domestic cats were reported harboring *E. procyonis* in New Jersey[5,6] and within a 250-mile radius of Fort Knox, Kentucky, including parts of Tennessee, Indiana and Ohio.[21] Denton[10] incriminated the land mollusk, *Mesodon thyroidus* Say, as a first intermediate host of *E. procyonis.* The second intermediate host was not determined.

This report is the third record of domestic cats being involved in the life history of *E. procyonis* and the second record of this fluke from Virginia and North Carolina. Preliminary gross and histopathological observations together with clinical findings are reported. The occurrences of domestic animals as hosts in the life history of *E. procyonis* are discussed together with their theoretical implications in the transmission of a parasite of wildlife.

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[1] From Bureau of Medicine and Surgery, Navy Department, Research Task MR005 09 0111B.

[2] The experiments reported herein were conducted according to the principles outlined in "Guide for Laboratory Animal Facilities and Care" prepared by the Committee on the Guide for Laboratory Animal Resources, NAS-NRC.

[3] The opinions and assertions contained herein are those of the authors and are not to be construed as official or reflecting the views of the Navy Department or the Naval service at large.

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Materials and Methods

From December 1969 through March 1970, 154 domestic cats were purchased from a dealer who collected them in North Carolina and Virginia. Fecal examinations using the MIF concentration technique were routinely accomplished when the cats arrived in the laboratory.[4] Blood and urine samples were collected from three cats passing dicrocoeliid eggs in their feces. Urinalysis was performed with a "Hema Combistix" (Ames Co., Eckhart, Indiana). Unopettes (Becton, Dickinson & Bard-Parker Co., Rutherford, New Jersey) and the Haussen Hemacytometer were used for white blood counts. Blood chemistry was run according to standard biochemical procedures.[24]

The pancreas, liver and associated ducts were examined in physiological saline. Flukes from each infected cat were fixed in warm 10% buffered formalin, stained with Gower's carmine stain and mounted for species confirmation. Ten adult flukes were deposited in the United States National Museum helminthological collection with the acquisition number 71538. Tissue samples of the pancreas were immediately fixed in 10% neutral buffered formalin processed in ethanol and xylene and embedded in paraffin. Sections were cut at five micra intervals and stained with hematoxylin and eosin and periodic acid - schiff methods.

Results

Upon arrival in the laboratory six of the 154 (ca. 4%) cats collected from widely separated counties in North Carolina and Virginia were passing dicrocoeliid eggs in their feces (Fig. 1). Five of the infected cats were sacrificed and specimens of *E. procyonis* were recovered.

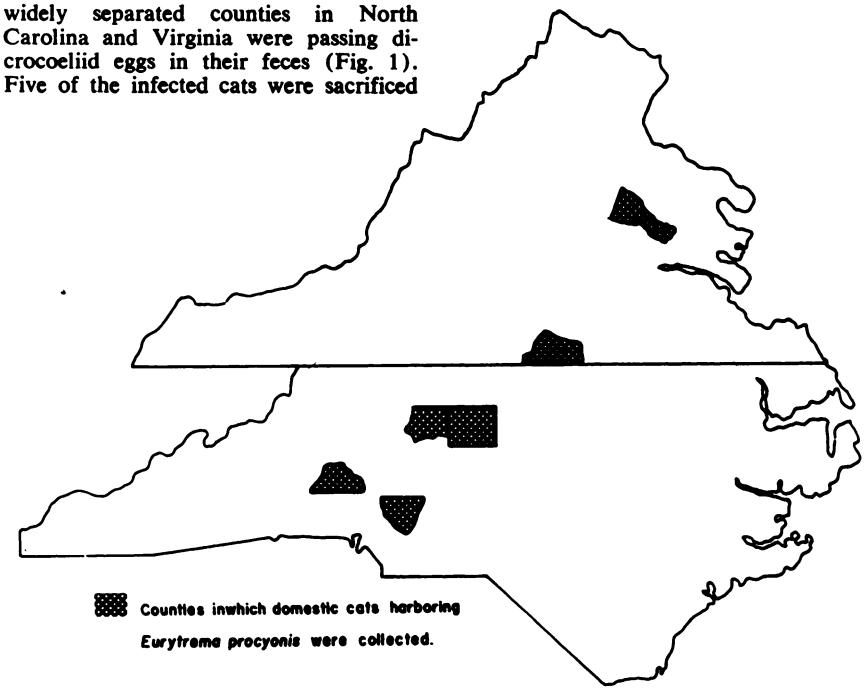


FIGURE 1. *Counties in North Carolina and Virginia from which domestic cats harboring* E. procyonis *were collected.*

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TABLE 1. *Clinical data for three domestic cats infected with* Eurytrema procyonis.

Cat No.	No. Flukes	Date	BLOOD ANALYSIS											URINALYSIS				
			HT	WBC	Lym	Neut	Eosin	Mylo	Mono	Band	Amy-lase	Lip-ase	Glu-cose	Sp. Gr.	pH	Glu-cose	Alb	Ke-tone
624		3/19	37	9.0	15	85	0	0	0	0	590	0	102	.....	.....	...	.....	...
		3/26	33	9.2	49	49	2	0	0	0	555	.....	.....	.....	.....	....	.....	....
	300	4/2	36	8.3	40	33	17	0	0	0	456	.20	153	1.060	6.0	0	1+	0
651		3/19	37	15.1	45	49	3	1	0	0	630	.59	116	.....	.....	....	.....	....
		3/26	35	18.0	66	31	2	0	1	0	655	.49	128	.....	.....	....	.....	...
	600	4/2	36	17.2	56	33	10	0	0	1	584	.35	80	1.065	6.5	0	1+	0
666		3/19	43	21.1	48	50	1	1	0	0	664	.06	.....	.....	.....	....	.....	....
		3/26	34	12.3	31	67	2	0	0	0	745	.....	.....	.....	.....	....	.....	....
	1800	4/2	32	18.6	30	66	4	0	0	0	616	.5	72	1.050	6.0	0	2+	0

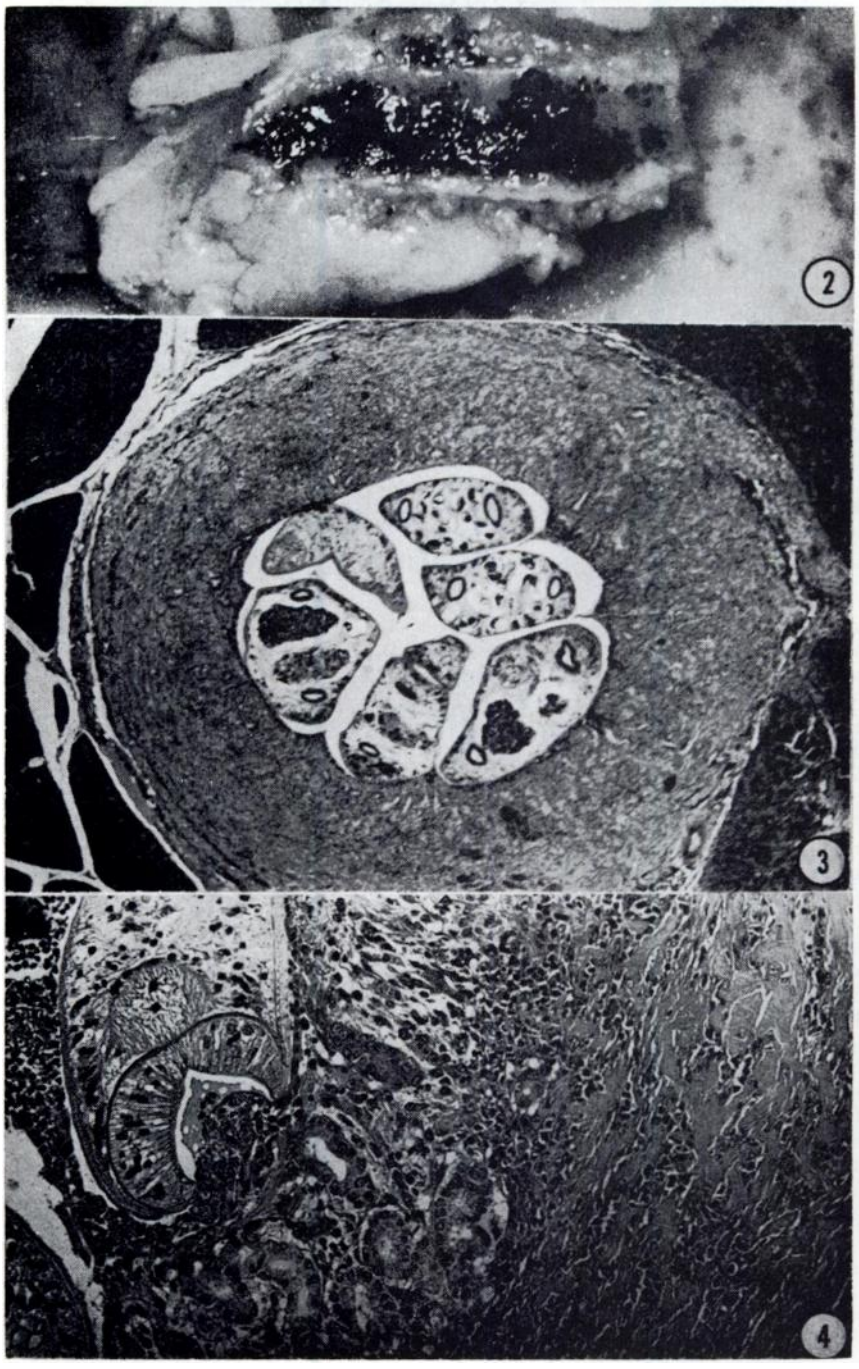
FIGURE 2. *Pancreatic duct cut longitudinally to reveal hundreds of mature flukes.*

FIGURE 3. *Enlarged pancreatic duct containing several mature flukes. H&E stain; X30.*

FIGURE 4. *Mature flukes attached to the duct epithelium. Desquamation of necrotic duct epithelium, periductal fibrosis and infiltration of inflammatory cells. H&E stain; X125.*



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Flukes were concentrated in the large pancreatic ducts of both the dorsal and ventral lobes of the pancreas with lesser numbers in the median and small interlobular ducts. No flukes were recovered from the gall bladder, cystic duct or hepatic ducts. In one case, however, flukes were found in the common bile duct within one cm of its junction with the common pancreatic duct. The number of flukes recovered from the five cats varied from 200 to more than 1800.

The results of blood and urine analysis (Table 1) on three cats with heavy fluke infestations ($>$ 300 to $>$ 1800) did not show evidence of enzymatic or hormonal pancreatic dysfunction. Daily observation of the six infested cats revealed no clinical signs of digestive or hormonal disorders. The only evidence of pancreatic infection was the presence of dicrocoeliid eggs in the feces.

Upon gross examination, the pancreata of five infected cats displayed morphological differences. Three of these would be considered "normal", one was reddened and atrophied, and the other was greatly reduced in size and appeared nodular and grayish. The pancreatic ducts were grossly enlarged, firm, and contained numerous flukes (Fig. 2).

Microscopically the flukes were attached to the ductal epithelial tissue by their oral and ventral suckers (Figs. 3, 4). Fibrous proliferation and infiltrating inflammatory cells accounted for the thickened gross appearance of the ducts. Desquamation of the duct epithelial cells was a prominent feature. Atrophy of the pancreatic lobules, interlobular fibrosis and inflammation of both the interlobular and intralobular tissues were commonly observed.

Discussion

The histopathology associated with *E. procyonis* infections was more extensive than anticipated. Some histopathological observations associated with pancreatic flukes in wild carnivores were reported previously.[12,15,18] Sheldon,[21] in discussing the histopathological alterations due to *E. procyonis* in domestic cats, reported only minor histopathological changes. He did find one pancreas in which a prominent cystic duct was present. Slight to moderate hypertrophy existed and the pancreatic ducts were thickened and cordlike in appearance. Sheldon also recovered most of the flukes in the small- to medium-sized pancreatic ducts. The findings in this study, to be reported in detail at a later date, showed extensive and varied histopathological changes in each host examined. The flukes, however, were concentrated in the large pancreatic ducts with lesser numbers in the small- and medium-sized interlobular ducts. Differences in pathological changes may be due to the age of infection more than the number of flukes present in a host.

No flukes were recovered from the hepatic ducts or gall bladders of the animals examined. However, in the most heavily infected cat a few flukes were found in the common bile duct within one cm of its junction with the common pancreatic duct. Their presence in the common bile duct was considered a result of postmortem migration since the cat was examined over one hour after death. Dicrocoeliids are generally specific as to their habitat in the accessory digestive organs. A previous report of *E. procyonis* in the gall bladder and bile ducts as well as the pancreas might have been the result of postmortem migration.[5]

Blood and urine values were reported in Table 1. Some of these values were slightly above normal range,[3,6] but there were no clinical signs in the fluke-infected cats. This is in agreement with Sheldon[21] who found no clinical signs attributable to *E. procyonis* infections in cats. Penner,[18] noting that the urine of a heavily infested raccoon ($>$ 3000 flukes) was cloudy yellow, pH 5.8 and positive for acetone, albumen and sugar, suggested that the pancreatic flukes might have contributed to the illness and death of this animal.

The presence of dicrocoeliid eggs in the feces of a cat is not, however, diagnostic of *E. procyonis*. Another dicrocoeliid, *Platynosomum fastosum* Kossack, is found in cats from North America. The eggs of *E. procyonis* from cats were

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described as ranging from 52-57 μ in length and 33-38 μ in width,[6] whereas averages of 30 μ by 24.5 μ were reported for *P. fastosum* from domestic cats.[14] However, differentiation of these two species by egg size, where both species are known to occur, may be difficult. Considerable variation was demonstrated in the size of dicrocoeliid eggs when examined fresh and after preservation in formalin[11] and the conditions under which the above measurements were made were not mentioned.

Grasshoppers, *Conocephalus maculatus* LeGuillou, were incriminated in the transmission of *Eurytrema pancreaticum* Janson in Malaysia.[2] Grasshoppers likewise were singled out as a choice food, in season, for the red fox, grey fox and raccoon in North America[16,17] and *Conocephalus* spp. are present throughout North America.[10]

Eurytrema procyonis, with the possible exception of *Dicrocoelium dendriticum* (Rudolphi), has the widest distribution for mammalian dicrocoeliids in North America. It is well established in focal areas throughout the east as indicated by the high percentage of carnivores infected with heavy fluke burdens. With the absence of clinical signs, infections could easily be overlooked. The overlapping ranges of the known wild carnivore hosts, the known distribution of the molluscan host and closely related species, and the presumed abundance of second intermediate hosts suggest that the range of *E. procyonis* covers at least the eastern and potentially the western states of the U.S.A., northern Mexico and southern Canada.

The involvement of domestic cats in the life history of *E. procyonis* affords speculation that domestic animals may have a role in the maintenance and dissemination of parasites of wildlife.

The geographical range of a parasite is limited by the ranges of its hosts. The effective range of a parasite, where transmission occurs, is limited to areas where all hosts occur together (Fig. 5). From this concept the terms focus or nidus have evolved when describing the geographical areas of parasite transmission. The ecological requirements of most mollusks are such that a species is seldom uniformly distributed throughout its range, but instead is broken up into a number of populations between which there are varying degrees of isolation.[25] Such molluscan populations serve as the foci around which trematode life cycles may occur. The molluscan host is thus an important limiting factor in transmission of a trematode.

The mollusk usually has the smallest range of the hosts involved in the trematode life cycle. The infective stages, passed from mollusks, are concentrated in limited areas, increasing the chance of infection in susceptible second intermediate hosts in these focal areas. The vertebrate host, on the other hand, usually has the widest range and is responsible for the dispersion of the trematode to other foci where transmission can occur. When second intermediate hosts are needed, they serve to channel the larval stages back to the definitive vertebrate host.

The spread of a parasite over its potential range should occur in short steps from one focus of transmission to the

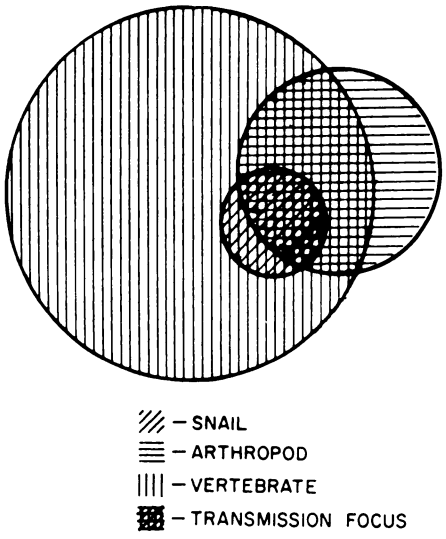


FIGURE 5. *Focus of transmission of a land-transmitted trematode involving three hosts.*

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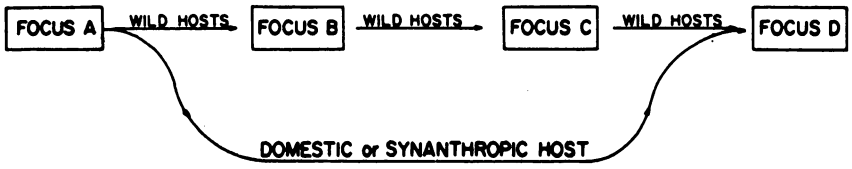


FIGURE 6. *Role of wild and domestic hosts in the transmission of a parasite of wildlife.*

next (Fig. 6) with the movements of the vertebrate host limiting the steps of range extension. However, when domestic hosts are introduced the gradual spread from focus A to B to C to D could easily be shortened by man's transporting an infected animal from focus A to D (Fig. 6). Thus, the utilization of domestic hosts in the life history of such a parasite allows for rapid dissemination of the species over its potential range. Moreover, the direct effect of man's activities on the parasitic fauna of animals associated with him, such as livestock and pets, and indirectly on the parasitic fauna of wild animals in contact with domestic animals is illustrated. Thus, the popular concept of a shrinking world in the dissemination of parasitic diseases of man applies to those of domestic animals since they have achieved a high degree of mobility due to their close association with man.

With the cultivation of domesticated animals and the gradual decrease and restriction of wild animals by man, domesticated animals may assume the role of maintaining select helminth species of wildlife in focal areas from which the parasites could again spread back to wild hosts when and if their numbers are allowed to increase (Fig. 7).

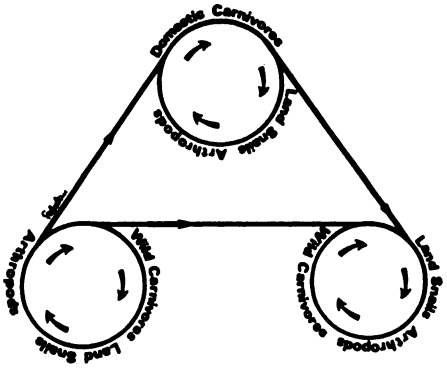


FIGURE 7. *Role of domestic hosts in the maintenance of a parasite of wildlife.*

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