STUDIES ON *Parorchis acanthus*
(TREMATODA: DIGENEA) AS A BIOLOGICAL
CONTROL FOR THE SOUTHERN OYSTER
DRILL, *Thais haemastoma*

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CONTENTS

ABSTRACT

Events in the attack on the southern oyster drill, *Thais haemastoma*, by miracidia of the digenetic trematode, *Parorchis acanthus*, are described.

Infection rates in wild drill populations of various Gulf Coast localities from Florida to Texas were low, but intensity of infection in individual drills was high and caused castration. Infection rates in laboratory experiments were high, but intensities were low.

Pathology of natural infections in drills is described. The infection lasts at least two years, possibly even for life.

Natural infection rates of juvenile herring gulls (*Larus argentatus*), ring-billed gulls (*L. delawarensis*), and juvenile laughing gulls (*L. atricilla*) are reported.

Juvenile herring and ring-billed gulls were readily infected experimentally with *P. acanthus*, juvenile laughing gulls were less susceptible, and nestling least terns (*Sterna albifrons*) appeared to be resistant. Intensity of infection was generally low.

P. acanthus offers little hope of being useful as a biological control of the drill, *Thais*, because of difficulties in spreading the parasite or assuring a significant rise in wild drill infection rates.

IV

STUDIES ON *Parorchis acanthus* (TREMATODA: DIGENEA) AS A BIOLOGICAL CONTROL FOR THE SOUTHERN OYSTER DRILL, *Thais haemastoma*

By Nelson R. Cooley, *Fishery Research Biologist*, Bureau of Commercial Fisheries

Although the biology and control of the chief Atlantic coast oyster drill, *Urosalpinx cinerea*, has been the subject of considerable study (reviewed by Carriker, 1955), the southern drill, *Thais haemastoma* Linné,[1] long known as an oyster predator, has received scant attention.

The southern drill is widely distributed in oyster-producing waters along the northern coast of the Gulf of Mexico, exists in incredible numbers in coastal bays and estuaries, and is extremely prolific. Its reproductive cycle has been studied in some detail by Butler (1953). Females deposit egg capsules regularly from March to August. Each capsule contains from several hundred to 4,000 eggs, and there is almost no mortality within the capsule. In a growth experiment performed by Butler, 250 females deposited an estimated 100 million eggs during 1 month. Hatching occurs in 12 to 16 days at 25° C, and after a planktonic life that may be as much as 7 weeks, the veliger larvae metamorphose into tiny snails. Sexual maturity is usually attained during the second summer, i.e., at 1 year of age, but normal egg and capsule deposition by snails not more than 8 weeks old has been observed.

Thais is probably the most important oyster predator in this area (Butler, 1953). In 1956 drill predation was so severe on depleted reefs in Mississippi waters (about one-half of the reef bottoms of the State) that much of the annual spatfall in the area was destroyed. In addition, drills destroyed a half of the oysters on the producing natural reefs in Mississippi that year (Chapman, 1958).

If such severe losses in the Gulf oyster industry are to be reduced, control measures must be instituted against the drill.

Broadly speaking, predation may be controlled by trapping (physical control), by poisoning (chemical control), or by parasites or predators (biological control). Physical control of *Thais* is impractical, except in restricted areas, because of expense and inefficiency of available methods. Chemical control is superficially feasible, but lack of specificity of most available chemicals raises fear of damage to oysters and other economically important species. Biological control by means of predators or parasites specific for the drill appears to be least likely to damage other species and, for that reason, was selected for investigation.

Few natural enemies of *Thais* are known. Butler's (1953) laboratory observations that hermit crabs attack drills to gain possession of their shells and that stone crabs, if sufficiently hungry, crack and eat drills are the only known proved reports of predators. The drill larva, a free-swimming veliger, may be eaten by pelagic fishes. Thus, although normal predation on larval stages may, probably does, cause enormous losses to drill populations, there appears to be no immediate prospect of further population reduction by this means.

Only a few parasites are known from *Thais*. The commensal polyclad, *Hoploplana inquilina*, and larvae of *Parorchis acanthus*, a digenetic trematode, were reported by Shechter (1943). Larval stages of at least two unidentified trematode species which caused considerable gonad damage were noted by Butler (1953).

During this study, examination of about 7,600 snails since the summer of 1956 revealed: one, possibly two, unidentified protozoans that invade and slightly damage digestive gland follicular cells; isolated instances of larval nematode infections and a few cestode infections (almost certainly larval tetraphyllideans) which appeared to be encysted single individuals, rather than reproducing populations; a single infection by sporo-

[1] Clench (1947) considers this marine snail to be two subspecies, *T. haemastoma floridana* Conrad, 1837, and *T. h. haysae* Clench, 1927, but because of ecological similarities, they are treated here as the same animal.

cysts of an unidentified small furcocercaria; and many instances of heavy, severely damaging *Parorchis acanthus* infection. Apparently no other parasites are known.

The only immediately available prospect for development as a biological control of *Thais* appeared to be *Parorchis acanthus*, which is known to damage the drill severely and whose reported adult hosts include several species of gulls and terns occurring in this area. Therefore, a study of this parasite and its effect on the drill was initiated.

A preliminary report (1957) summarized available information on the life cycle, known hosts, morphological descriptions, synonymy and endemic localities, and gave preliminary data on experimental infection of drills, and incidence and pathology of natural infections in *Thais*.

The present paper is a final report on *P. acanthus* and gives further information on life cycle, experimental infections, and incidence and pathology of natural infections in Gulf coast drills.

Field collections of drills were made by Dr. Abraham Fleminger, then at Bureau of Commercial Fisheries Biological Laboratory, Galveston, Texas; Dr. A. K. Sparks, then at Texas A. & M. Research Foundation Laboratory, Grand Isle, Louisiana; William Demoran, Gulf Coast Research Laboratory, Ocean Springs, Mississippi; and Eugene Holzapfel and others, Aransas Pass, Texas.

William D. Wood, manager, Sanibel National Wildlife Refuge, Florida, supplied a number of young laughing gulls used in infection studies.

Consultations with Dr. H. W. Stunkard were helpful during a part of the study.

MATERIALS AND METHODS

Specimens of *T. haemastoma* from various localities in Florida, Alabama, Mississippi, Louisiana, and Texas, were examined for natural *P. acanthus* infections by dissection or by isolation in individual dishes of sea water.

For morphological studies, both living and fixed and stained material prepared by standard parasitological techniques were used.[2]

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[2] Fixatives: *G* (Gilson), *Z* (Zenker), *PFA–3* (Allen's PFA–3 modification of Bouin). Stains: *DH–AzB–E* (Delafield's hematoxylin–Azure B–Eosin Y), *WH–AzB–E* (Weigert's acid-iron-chloride-hematoxylin-Azure B–Eosin Y).

Juvenile herring gulls (*Larus argentatus*), ring-billed gulls (*L. delawarensis*) and juvenile laughing gulls (*L. atricilla*) were used as experimental hosts of adult worms. Experimental infection of gulls was accomplished as follows. Cercariae were permitted to encyst in a finger bowl of sea water and become metacercariae. The cysts, carefully scraped from the bottom of a finger bowl with a scalpel and suspended in a small amount of sea water, were pipetted directly into the stomach of a fasting bird by means of a stiff ⅛-inch-bore polyethylene tube carefully passed down the esophagus. The tube was flushed with a few milliliters of sea water and carefully removed. The bird was then returned to its cage and given its daily feeding. Alternatively, cysts or adult worms were introduced directly into the cloaca.

Attempts to infect least tern (*Sterna albifrons*) nestlings were made by feeding encysted metacercariae with a pipette immediately before giving the birds food.

All gulls were fed chopped fish that had been frozen and stored at 0° F. for several weeks to prevent infecting the birds with fish-borne helminths. Least tern nestlings were fed beef-base dog food, later supplemented with bits of fish.

If repeated examinations of cloaca and bursa Fabricii over a period of weeks were consistently negative, the gulls and terns were concluded to be free of natural infection. The same method was used to detect experimental infections, which, in most cases, were subsequently confirmed by autopsy.

Drills were infected experimentally by 24-hour exposure of individuals to freshly hatched miracidia in 4-inch bowls containing 100 ml. of sea water. These drills were kept as long as 3 days in 4-inch bowls with food and daily changes of sea water, or for periods as long as 10 weeks in battery jars with running sea water and adequate food.

Thais specimens for histological study were killed at various intervals after exposure to infection and fixed in Gilson's, Zenker's, Bouin's, or Allen's PFA–3 solutions or Smith's modification of Bouin's solution. Gilson's, Zenker's, and Allen's PFA–3 gave best results. Specimens were paraffin-embedded, sectioned at 7μ or 10μ and stained routinely by a Delafield's hematoxylin-azure B-eosin Y technique. With the azure-eosin mixture adjusted to pH 4.1 to 4.95 with McIlvaine-Lillie

buffers (Lillie, 1948, p. 260–263), depending on the fixative used, the procedure produced brilliant differential staining of the two types of digestive gland follicular cells, necrotic areas in the host tissues, and various parasite-structures. In a few instances, Weigert's acid-iron-chloride-hematoxylin was substituted in the procedure, but the results were less satisfactory.

THE PARASITE

Taxonomy

The adult was originally named *Zeugorchis acanthus* by Nicoll (1906), who described it from two specimens found in the cloaca and bursa Fabricii of the herring gull (*Larus argentatus*). Following study of more material from herring and common gulls (*L. canus*), he (1907) designated it type of a new genus, *Parorchis*. Subsequently, Linton (1914) mistakenly described the same worm as a new species, *P. avitus*. Lebour (1907) described *Cercaria purpurae* from *Purpura* (=*Thais*) *lapillus* and subsequently (1914), on morphological grounds alone, correctly identified it as a larval stage of *P. acanthus*. Later, Stunkard and Shaw (1931) described *Cercaria sensifera* from *Urosalpinx cinerea* and Stunkard and Cable (1932) demonstrated experimentally that *C. sensifera* is a larval stage of *P. avitus*. Cable and Martin (1935) reduced *P. avitus* Linton, 1914, to synonymy with *P. acanthus* (Nicoll, 1906) Nicoll, 1907. To the synonym list compiled by Cooley (1957) should be added the genus *Proctobium* Travassos, 1918 (cited in Strom, 1927).

The systematic position of *P. acanthus* is illustrated by the following classification scheme taken from Hyman (1951):

Phylum Platyhelminthes
  Class Trematoda
    Order Digenea
      Family Echinostomatidae
        Genus *Parorchis* Nicoll, 1907
          Parorchis acanthus (Nicoll, 1906) Nicoll, 1907.

Morphology

The morphology of the different developmental stages of *P. acanthus* has been described by a number of authors. A complete source-list of the descriptions was given in an earlier paper (Cooley, 1957). The material of the present study is in general agreement with these descriptions. The existing differences, however, do not invalidate the identification of the present material, for, as Stunkard (1957, p. 16) pointed out, "members of a single species may differ so much as a result of development in different host species, invertebrate and vertebrate, or of different physiological conditions in host-individuals, that the extent of variation is known for few if any species. . . ."

Miracidium.—The present material is compared with published descriptions in table 1. The main differences are (1) the variation in length of anterior, body, and caudal cilia, (2) the variation in the shape of pigment spots ("eyespots," "eyes") [No two descriptions agree on this], and (3) the shorter length of the contained redia. In addition, Nicoll (1907) reported the body to be differentiated into a distinct head and a posterior part, but neither Linton (1914), Rees (1940), nor the author have observed such a condition. It was obviously a temporary shape, perhaps as a result of contraction during fixation. The miracidium swims rapidly, yawing slightly as it rotates about its longitudinal axis. While swimming, both miracidium and contained redia become elongate, but regain their typical shapes upon halting. In other respects, there is agreement with published descriptions.

TABLE 1.—*Comparison of living* P. acanthus *miracidia from several sources*

[All measurements in millimeters]

Item	Nicoll (1907)	Linton (1914)	Rees (1940)	Present study
Length (exclusive of cilia).	0.18	0.12–0.16	0.02(sic)	0.16–0.17.
Width (exclusive of cilia).	0.05	0.08	0.054	0.06–0.07.
Cilia length and disposition.	long, completely cover body.	0.02	0.015, closely set long, rows, absent on rostrum.	ca. 0.02 on body; anterior cilia shorter, stiffer, more closely arranged; caudal cilia slightly longer.
Pigment spot.	1, large, dark, usually 5-lobed.	single, distinct, black, variable shape.	kidney-shaped, 2 forming a single mass.	0–2, large black, subspherical.
Contained redia:				
Length		0.14	0.175	0.07–0.09.
Width		0.04	0.028	0.04.
Host	Larus argentatus, L. canus.	L. argentatus.	L. argentatus.	L. delawarensis.
Locality	Scotland	Massachusetts.	Wales.	Florida.

Redia.—Specimens of this stage agree closely with published descriptions.

Cercaria.—Comparison with published descriptions (tables 2, 3) reveals slight differences which could easily result from development in a different snail host or from the fixative employed.

Most measurements of cercariae which were killed with gentle heat and measured in sea water tended to be greater than those preserved by the classic method of relaxing them by rapidly swirling in a small amount of sea water before flooding by hot fixative. The difference was due either to incomplete relaxation before fixation occurred or to agonal contraction caused by contact with fixative.

The cercariae encyst readily on any available object. Sizes of living cysts from several sources, measured in situ, are compared in table 2. The range in length and width was considerably greater in my material than in that of others. The differences which exist are most likely due to the parasites having developed in three different snail hosts.

Comparison of cyst measurements made before and after fixation revealed generally insignificant fixation-induced changes: Gilson's and Carnoy's fixatives caused slight shrinkage, while Bouin's fixative caused slight swelling.

TABLE 2.—*Comparison of living* P. acanthus *cercariae and cysts from several sources*

[All measurements in millimeters]

Item	Lebour and Elmhirst, 1922	Stunkard and Shaw, 1931	Rees, 1937 Expanded	Rees, 1937 Contracted	Present study
Body length		up to 0.9	1.00	0.36	0.44 –0.57
Tail length		up to 0.9	0.82	0.18	0.29 –0.61
Total length		up to 1.8	1.82	0.54	0.73 –1.18
Body width			0.09	0.35	0.21 –0.36
Oral sucker:					
Position		subterminal	subterminal		subterminal
Length		0.06–0.08 in diameter.	0.07		0.060–0.087
Width			0.01		0.067–0.107
Ventral sucker:					
Length		0.08–0.1	0.10		0.100–0.141
Width		0.1–0.115	0.11		0.087–0.121
Oral sucker: Ventral sucker		1:1.50–1:1.98	1:1.263		1:1.14–1:1.73
Cyst:					
Length	0.24–0.28	0.23–0.27	0.295 in diameter.		[2] 0.218–0.327
Width	0.20–0.22	0.20–0.23			0.185–0.294
Host	*Purpura lapillus.*[1]	*Urosalpinx cinerea.*	*Purpura lapillus.*[1]		*Thais haemastoma.*
Locality	Scotland	Massachusetts.	Wales		Florida.

[1] = *Thais lapillus.*
[2] Average of 46 cysts: 0.270 mm. X 0.231 mm.

TABLE 3.—*Comparison of* P. acanthus *cercariae prepared by different methods*

[All measurements are in millimeters]

Item	Stunkard and Shaw, 1931	Present study Heat-killed [1]	Present study Bouin-fixed [2]	Present study FAA-fixed [2]
Body length	0.21–0.47	0.32 –0.88	0.21 –0.40	0.36–0.47
Tail length	0.12–0.26	0.39 –0.61	0.11 –0.48	0.28–0.47
Total length	0.33–0.73	0.71 –0.149	0.32 –0.73	0.66–0.94
Body width	0.14–0.21	0.20 –0.37	0.07 –0.21	0.11–0.13
Oral sucker:				
Length	0.05–0.06 (diameter)	0.054–0.094	0.034–0.060	0.03–0.06
Width		0.067–0.107	0.047–0.101	0.03–0.05
Ventral sucker:				
Length	0.68–0.76 (diameter) (sic)	0.094–0.147	0.047–0.074	0.06–0.09
Width		0.094–0.147	0.054–0.121	0.07
Oral sucker: ventral sucker	1:1.27–1:1.36	1:1.24–1:1.77	1:1.11–1:1.24	1:1.33–1:2.00

[1] Unstained, measured in sea water.
[2] Stained and mounted in Permount.

Adult.—Table 4 compares permanent preparations of sexually mature adults from the present study with published descriptions. No major differences exist. These data illustrate the range of variation which can result from development in different hosts.

Life Cycle

The adult was described by Nicoll (1906, 1907), Linton (1914, 1928), and Stunkard and Cable (1932).

The cercaria, described by Lebour (1907), was first found in rediae from *Purpura* (=*Thais*) *lapillus*. In 1914, after comparing it with young *P. acanthus* adults from herring gulls, she correctly inferred that *C. purpurae* is a larval stage of *P. acanthus*. Later, with Elmhirst (1922), she reported a life cycle which erroneously included a molluscan second intermediate host (either *Cardium edule* or *Mytilus edulis*).

The first correct life-history description is that of Stunkard and Cable (1932). By feeding cysts derived in vitro from cercariae naturally emitted from the oyster drill, *Urosalpinx cinerea*, to common (*Sterna hirundo*) and roseate terns (*S. dougalli*), they proved conclusively that only two hosts are necessary: a marine snail and a marine bird.

Details of miracidial structure, cercarial anatomy and encystment, and germ cell cycle in both larval and adult stages were reported by Rees (1937, 1939, 1940).

TABLE 4.—*Comparison of stained and mounted sexually mature* P. acanthus *adults from several sources*

[All measurements are in millimeters]

Item	Nicoll, 1907	Linton, 1914, 1928	Present study				
Body:							
Length	3–5	3.75–6.10	4.3–6.04	4.1–5.8	4.13–4.69	3.72.	
Width	1.2–3	2.10–2.66	1.62–2.1	1.4–2.5	1.50–2.19	1.62.	
Spination and spine size:							
Body	0.019–0.031×0.012		0.0356–0.0369× 0.0256–0.0335.	0.0160–0.0335× 0.0101–0.0288.	0.0268–0.0402× 0.0168–0.0201.	0.0402–0.0469× 0.0201–0.0335.	
Collar	0.037	0.022	0.0201–0.0335× 0.0168–0.0268.	0.0168–0.0402× 0.0101–0.0335.	0.0235–0.0268× 0.0101–0.0134.	0.0402–0.0536× 0.0268–0.0335.	
Collar number	about 60	Single row, small	57–68	54–64	71	58.	
Oral sucker:							
Length	up to 0.5	0.30–0.44	0.30–0.32	0.28–0.31	0.33–0.34	0.29.	
Width	up to 0.5	0.36–0.49	0.33–0.36	0.31–0.37	0.37–0.43	0.30.	
Ventral sucker:							
Length	1.08	0.71–1.35	0.9–1.09	0.83–0.97	0.82–0.86	0.74.	
Width	1.08	0.73–1.36	0.88–0.99	0.70–0.93	0.87–0.97	0.77.	
Anterior sucker: Ventral sucker	1:2.16	1:2.86	1:2.74–1:3.30	1:2.47–1:2.92	1:2.36–1:2.43	1:2.56.	
Distance of anterior margin of ventral sucker from anterior end	1.26	1.02–1.54	1.19–1.51	1.03–1.47	1.27–1.51	1.10.	
Prepharynx	0.11	V. short [0.024]	0.11–0.13	0.02–0.17	0.13–0.16	0.11.	
Pharynx:							
Length	0.24	0.18–0.24	0.14–0.17	0.15–0.19	0.17–0.19	0.24.	
Width	0.17	0.14–0.24	0.11–0.15	0.11–0.13	0.13–0.19	0.14.	
Esophagus	ca. 3x as long as pharynx [0.72].	0.36–0.52	0.33–0.44	0.30–0.60	0.58–0.73	0.44.	
Testes	Lobate	Lobate	Lobate	Lobate	Lobate	Lobate.	
Right testis:							
Length	0.55–0.60	0.32–1.00	0.57–0.90	0.37–0.66	0.32–0.52	0.54.	
Width	0.55–0.60	0.28–0.77	0.53–0.59	0.34–0.62	0.30–0.55	0.42.	
Left testis:							
Length	0.55–0.60	0.33–0.98	0.60–0.78	0.42–0.62	0.40–0.59	0.54.	
Width	0.55–0.60	0.33–0.88	0.44–0.62	0.40–0.59	0.30–0.59	0.45.	
Ovary:							
Length	0.33	0.15	0.16–0.22	0.15–0.28	0.20–0.28	0.18.	
Width	0.25	0.24	0.17–0.26	0.17–0.30	0.24–0.38	0.24.	
Egg (shape)	Elliptical	Oval	Elliptical	Elliptical	Elliptical	Elliptical.	
Size, anterior part of uterus	0.106–0.113× 0.056–0.062.	0.082–0.100× 0.046–0.060.	0.0469–0.1005× 0.04–0.1038.	0.064–0.114× 0.034–0.060.	0.054–0.074× 0.027–0.034.	0.0670–0.0737× 0.0268–0.0302.	
Size, posterior part of uterus	0.081–0.095× 0.040–0.044.	0.066–0.079× 0.040.	0.0603–0.838× 0.0335–0.0402.	0.064–0.104× 0.034–0.054	0.040–0.070× 0.027–0.034.	0.0670–0.0737× 0.0335–0.0391.	
Miracidium	0.18×0.05, 2-part body, 1 pigment spot in head.	0.08×0.05, cilia ca. 0.02 long, 1 pigment spot in head.	0–1 pigment spot	0.16–0.17×0.06–0.07 (excluding cilia); body cilia ca. 0.02, anterior cilia slightly shorter, caudal cilia slightly longer; 0–2 pigment spots.	0–pigment spot	0–pigment spot.	
Habitat	Bursa Fabricii, cloaca, rectum.	Cloaca	Cloaca	Cloaca, colon	Cloaca, cloaco-colonic junction.	Cloaca.	
Host	*Larus argentatus; L. canus.*	*L. argentatus*	*L. argentatus*	*Larus delawaren-sis.*	*Larus atricilla*	*Sterna albifrons.*	
Locality	Scotland	Massachusetts	Florida	Florida	Florida	Florida.	

The essential features of Stunkard and Cable's and Rees' life cycle accounts were collated and summarized by Cooley (1957), who also compiled lists of known hosts, infection sites in each host, and localities where the parasite is endemic.

To this host list should be added the ruddy turnstone (*Arenaria i. interpres*), the natural host in Hawaii, and noddy terns (*Anous stolidus pileatus* Scopoli), sooty terns (*Sterna fuscata oahuensis* Bloxam), wedgetailed shearwaters (*Puffinus pacificus cuneatus* Salvin), and domestic ducks (Muscovy, Pekin), the last four being experimentally infectible when maintained on a diet of squid (Oguri and Chu, 1955); also the laughing gull (*L. atricilla*), a natural host in Florida, and the least tern (*Sterna albifrons*), an experimental host (present study).

Despite the number of studies of various aspects of the life cycle of *P. acanthus*, apparently no one has described the actions of the miracidium as it attacks the drill.

Invasion of the drill by Parorchis *miracidia.*— Living miracidia are easily obtained by teasing eggs from the uterus of the adult worm into sea water. They hatch almost immediately. The hatching process is well described by Rees (1940).

The miracidium is a very rapid and active swimmer. Its restless to and fro movements seem without direction and contact with the drill appears to be accidental. However, a few observations suggest that the parasite may be attracted by the mucus secreted by the drill.

Rees (1940) stated that the position of the young first generation rediae in the digestive gland

"seems to indicate that the miracidum enters the shell aperture and makes its way up between the shell and the body of the enclosed animal. The miracidium probably penetrates the tunica propria of the digestive gland . . . and then liberates the contained redia by decomposition of itself." This sequence of events cannot be observed in the intact animal. The following observations demonstrate that it is not the sole means of entry of the parasite into the snail.

The main attack sites are the outer wall of the siphon, the base of the head, and the side (rarely, the sole) of the foot. Occasionally, miracidia, trapped in the siphonal water current, are swept into the mantle cavity. Most, if not all, pass out in the excurrent flow, but a few may possibly invade the host tissues there.

The invasion, most readily studied in the outer wall of the extended siphon, occurs in the following manner. The miracidium contacts and adheres to the skin of the drill by its anterior end. Attachment and penetration occur only when the miracidium strikes more or less at right angles to the skin surface; miracidia striking at acute angles ricochet and do not become attached.

Immediately on attachment several longitudinal contractions of the anterior two-thirds of the parasite's body follow, so that it appears to butt the snail. It quivers very rapidy for a few seconds, becomes quiescent for a short time, then a series of rhythmic contraction waves sweeps over the miracidium for approximately 30 minutes before slowing markedly. The contraction waves appear to aid penetration (fig. 1, a to e).
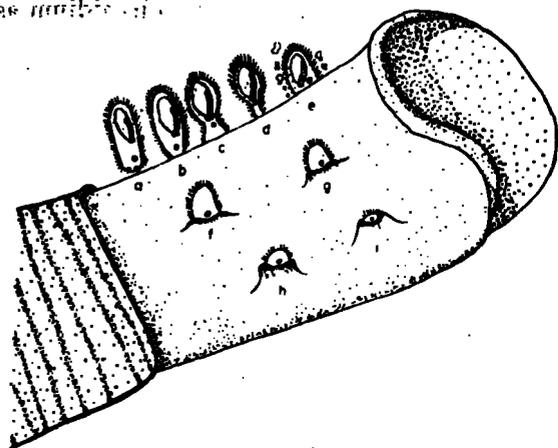

FIGURE 1.—*Parorchis* miracidia invading siphon of *Thais* (diagrammatic).

During the first 15 minutes (approximately) of the invasion, occasional small showers of host epithelial cells are carried away from the invasion site by water currents produced by the parasite's cilia (fig. 1, e). This observation appears to corroborate Rees' (1940) statement that the droplets of secretion seen by her to emerge from the two anteriorly located penetration glands onto the rostrum of the miracidium probably facilitate entry of the miracidium into the molluscan host.

About an hour after contact, the parasite's cilia appear to stop beating. Closer examination reveals that the lateral cilia continue to beat very slowly for at least another half hour as the miracidium moves in and out of the low wheal which develops at the invasion site and progressively enlarges as the miracidium penetrates deeper into the snail (fig. 1, g to i).

In about 1½ hours after attachment, the first generation redia contained in the miracidium could no longer be seen. Whether the redia has left the miracidium and entered the host tissues or still lies within the miracidium could not be ascertained.

Complete penetration by the miracidium requires about 6 hours. The subsequent fate of the miracidium is unknown, but it is presumed to disintegrate completely within the host, since no trace has been found in serial sections of snails fixed as early as 24 hours after penetration was known to have occurred. Nor was Rees (1940) able to find any trace of it either inside or outside the snail host. On the other hand, the miracidium of *P. acanthus* may behave in the manner described by Stunkard (1934) for *Typhlocoelum cymbium*, whose miracidium does not penetrate the snail. After that miracidium is securely attached and partially embedded, the redia leaves it and enters the snail.

Peculiarly, as the miracidium penetrates deeper, its eyespot seems to move posteriorly until it lies in the rearmost part of the parasite's body (fig. 1, e to i). The reason for the apparent rearward migration of the eyespot is uncertain, but it is probably related in some way to the escape of the redia from the miracidium.

Redial Development.—The young, first-generation redia has not yet been demonstrated at the

invasion site itself. At 24 and 72 hours after drills were first exposed to numerous miracidia, very young first generation rediae were found in the tissues of the head and columellar muscle as well as in the arteries, veins, sinuses and tissues of the siphon, foot, eyestalk, mantle, kidney, and visceral mass (figs. 2, 3, 5.)

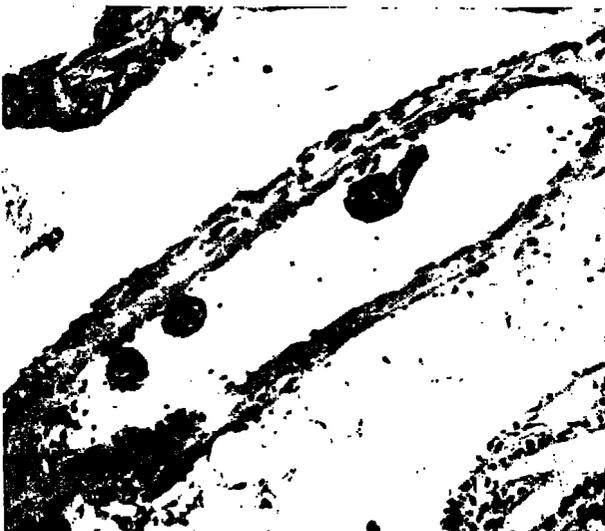FIGURE 2.—Very young redia in large vein next to columellar muscle; 24-hour experimental infection; G, DH–AzB–E; X210.

FIGURE 3.—Very young redia, parts of two others, in large vein near digestive gland, 72-hour experimental infection; PFA–3, DH–AzB–E; X210.

FIGURE 4.—Much older redia in renal blood sinus; natural infection; Z, DH–AzB–E; X215.

FIGURE 5.—Very young redia entering sinus in siphon wall; 72-hour experimental infection; PFA–3, DH–AzB–E; X210.

By the eighth day, the rediae had grown markedly and germ balls (embryos of the next larval generation) had begun to appear internally, (fig. 4), but the rediae were still found in much the same locations as during the first three days of infection.

Ten weeks (November to February) after a small drill was exposed to about 50 miracidia, serial sections revealed 15 well-developed rediae in the head, foot, visceral mass, in the aorta near the heart, and, for the first time in this study, in the anterior margin of the digestive gland, favorite site in heavy natural infections. Most of the rediae contained daughter rediae which, in turn, contained germ balls of a third generation (fig. 6). This degree of development is probably

FIGURE 6.—Redia with daughter rediae, one of which contains germ balls of a third generation, in foot: 10-week experimental infection; G, DH–AzB–E; X230.

attained sooner at the higher water temperatures prevailing during spring and summer.

The entrance of rediae into the digestive gland marks the beginning of a concentration of parasites there, as is found in natural infections.

Rees (1940) notes that the parent (first generation) redia gives rise to 20 or more daughter rediae and that daughter rediae produce only cercariae. If daughter rediae produce only cercariae, it would appear that the parasites would complete their development in a relatively short time and depart, leaving the snail host infection-free. However, the present study has demonstrated that natural infections persist for at least 2 years.

Sections of snails harboring natural infections known to be at least 2 years old always had large numbers of daughter rediae containing cercariae in various stages of development. Although no daughter rediae containing recognizable rediae were found, there were occasional small rediae containing embryos which might have developed into either rediae or cercariae. The evidence suggests that there occurs a series of redial generations before final production of cercariae.

Observing the prominent procuscula (posterior "feet") of young rediae and their considerable activity when removed from the snail, Rees (1940) concluded that the rediae are capable of migrating among the follicles of the digestive gland. The present study demonstrates rediae in various stages of development in a wide variety of lodgment sites, the location of which depends on how long the snails have been experimentally infected. In addition, rediae whose intestines contained yolk platelets have been found in the digestive gland of a naturally infected snail (fig. 9), indicating that they had migrated from the ovary into the digestive gland. It can be concluded, therefore, that the redia is capable of migrating from an invasion site anywhere on the body of the snail to the final lodgment in the digestive gland or gonad.

Although all experimental drills kept alive for more than 3 days were maintained in running sea water with adequate food to keep them in good physiological condition, it is highly improbable that any could have become infected by miracidia brought in by the incoming sea water; because, although simultaneously supplied by the same salt water tap, none of the control snails of these experiments, and none of 27 control drills of a longevity study of Parorchis-infected drills similarly maintained for more than 2 years, ever became infected by Parorchis or any other trematode.

Cercarial Encystment.—The swimming and creeping motion of the cercaria was described by Stunkard and Shaw (1931), who noted that it attached readily to any surface and encysted soon after attachment. In the present study, however, the cercariae usually alternated between attachment and creeping or swimming many times before finally encysting.

Accelerated encystment following either mechanical stimulation, such as stirring or shaking, or chemical stimulation, such as use of too concentrated solutions of vital dyes, was noted by

Stunkard and his coworkers (1931, 1932). This finding has been confirmed in the present study. Under these conditions, the cercaria encysted almost immediately upon contacting the substratum.

The encystment process was described briefly by Stunkard and Cable (1932), more fully by Rees (1940). My own observations amply confirm their findings. However, the following observations should be added to their descriptions. About 1 minute after extrusion of the cystogenous material, the body rapidly shrinks, withdraws into the center of the cyst and becomes immobile. The shrinkage and withdrawal results in decaudation. The detached tail, now attached by its base to the outer cyst membrane, lashes about for several hours. After some hours, the body begins to move about within the cyst membranes and ultimately assumes the characteristic folded position figured by Stunkard and Cable, and by Rees.

NATURAL INFECTIONS IN *THAIS*

Incidence

The incidence of natural infections of *P. acanthus* in *T. haemastoma* examined between July 1956 and September 1959 is summarized in table 5. Although the parasite is widely distributed along the Gulf coast, the infection rate in any sampled locality was low. The apparent absence of infections in snails from Apalachicola Bay, Florida, and Port Aransas, Texas, and the low rates in drills from Dauphin Island Bay, Alabama, and Barataria Bay, Louisiana, may be due to small sample-size or, possibly, to more resistant snail populations or fewer infected birds in those localities.

The following data suggest that the drill infection-rate in a given locality may be higher on the feeding grounds of the local gull population than at a site away from them. Between June 1958 and June 1959, 5.37 percent (57 of 1064) of the drills collected at a site on the north shore of Pensacola Bay, Florida, had *P. acanthus* infections, but only 0.47 percent (4 of 859) of those from a site on the south shore were infected. Although these two sites are only 3.5 miles apart, similar marked differences in incidence of infection have been observed in all collections made there since the summer of 1956 and appear to be correlated with the high concentration of gulls at feeding grounds on the north side of the bay.

TABLE 5.—*Incidence of natural infections of* P. acanthus *in* T. haemastoma, *1956–59*

Locality	Snails		
	Number examined	Infected	
		Number	Percent
Alabama:			
Dauphin Island Bay	383	1	0.26
Florida:			
Apalachicola Bay	138	0	0
Pensacola Bay	3,874	105	2.71
Santa Rosa Sound	691	8	1.15
Total	4,703	113	2.40
Louisiana:			
Barataria Bay	357	1	0.28
Mississippi:			
Mississippi Sound	700	29	4.14
Texas:			
Galveston Bay	1,277	45	3.52
Port Aransas	184	0	0
Total	1,461	45	3.08
Grand total	7,604	189	2.48

Pathology in the Snail

Stunkard and Shaw (1931) and Stunkard and Cable (1932) noted that infected *Urosalpinx cinerea* and *Thais* [=*Purpura*] *lapillus* in Massachusetts harbored *P. acanthus* larvae in the interlobular spaces of both digestive gland and gonad. The picture was similar in both species: the uninfected snail had a plump visceral mass, yellow digestive gland, and cream-colored gonad, whereas the parasitized snail had shrunken organs and lighter colored body, and its gonad might have been destroyed.

Rees (1937) noted the parasite in the same organs in *Thais* [=*Purpura*] *lapillus* in Wales and reported a similar picture, except for finding the visceral mass of the parasitized snail much swollen.

Menzel and Hopkins [3] noted that many old *Thais haemastoma* from Barataria Bay, Louisiana, had heavy infections of *P. acanthus* larvae, which destroyed the gonads and caused complete sterility. Hopkins's (1957) report appears to be the earliest published record of *Parorchis*-induced castration of *T. haemastoma*. The following pathology study, part of which confirms their observation, had already been completed before the writer learned of their findings.

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[3] Menzel, R. W., and S. H. Hopkins, 1954. Studies on oyster predators in Terrebonne Parish, La. Mimeographed report, Texas Agricultural and Mechanical Research Foundation, College Station, Texas. (Released, 1959).

Data obtained more recently from examination of 189 naturally infected drills collected in all seasons from 1956 through 1959 require revision of my earlier (1957) brief description of the pathology of drill infection.

The digestive gland of the uninfected drill is variable in color, usually light gray or beige, but may be very dark, almost black; it has a soft, cheesy consistency, and is covered by a fairly tough tunica propria. The digestive gland of the infected drill is less variable in color, usually lighter gray or light yellow, is swollen, has a softer consistency and a more easily torn tunica propria, through which can be seen enormous numbers of rediae scattered among widely separated small masses of host tissue.

The gonads of uninfected drills vary in size and thickness with seasonal changes in reproductive activity, but the color is usually brownish in males and yellow to orange in females. In infected drills, the gonad is thin and patchy, or completely absent; when present, its color is cream to brown in males and usually yellow to orange, sometimes brownish, rarely oyster white, in females.

Sex determinations were made on approximately one-half of all infected snails (98 of 189) collected in the present study; the sex ratio of these snails was approximately one male to 1.6 females. It is found, even in small lots of drills, that a 1:1 sex ratio usually prevails in field collections.
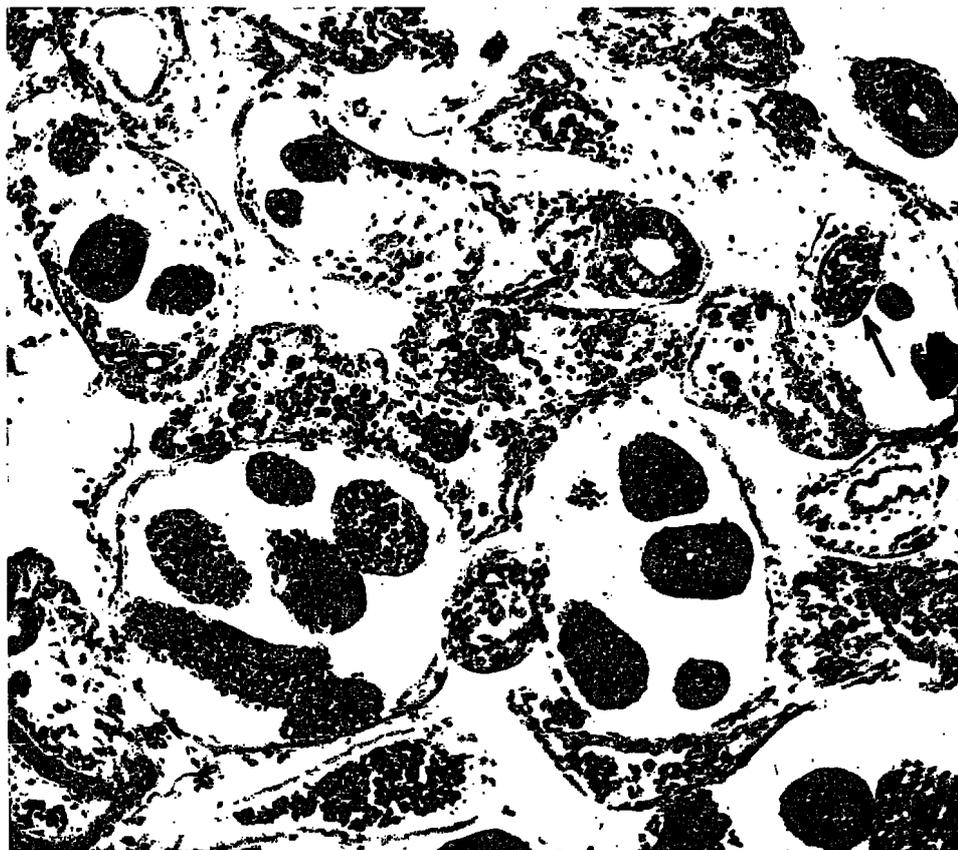

FIGURE 7.—Daughter rediae in various stages of development in ovary, natural infection; Z, DH–AzB–E; ×212.

FIGURE 8.—Redia with yolk platelets in posterior end of intestine (arrow), digestive gland, natural infection: Z, WH–AzB–E; ×215.

Microscopic examination of stained sections of infected drills reveals:

(1) Extensive destruction of the digestive gland, due to redial ingestion of host tissue, which may reduce intact tissue to 10–30 percent of the area of a cross section of the gland; (2) Compression of the remaining digestive gland tubules with resultant obliteration of most, if not all, lumina because of growth of the large number of rediae; (3) Basophilic inclusion granules of uncertain significance in the cytoplasm of the large triangular cells of the digestive gland; (4) Amoeboid cells, thought to be phagocytic blood cells, grouped about some rediae in the digestive gland; and (5) Gonadal damage which is directly related in extent to severity of infection, massive infections resulting in severe to total destruction due to ingestion of host tissue by rediae (figs. 7–10).

Naturally acquired *Parorchis* infections produce lasting damage. Forty-four infected drills maintained in an aquarium with running sea water and adequate food did not spawn during an entire breeding season, and 26 of these failed to spawn during a second breeding season (approximately 2 years in captivity), the remainder having been sacrificed for various purposes. Similarly maintained control snails spawned normally each year.

Duration of Infection

It is not known precisely how long these infections last, but 26 naturally infected drills maintained in the laboratory for 2 years regularly emitted cercariae when tested and all were heavily infected when sacrificed for dissection or for histological study. Thus, once established, the

FIGURE 9.—Daughter redia in ovary showing yolk platelets (arrow) and developing cercariae, natural infection; PFA–3, DH–AzB–E; ×215.

infection persists for 2 years and possibly for the life expectancy of the snail, estimated by Butler (1953) to be 5, possibly as much as 10, years.

EXPERIMENTAL INFECTIONS

Adult Hosts

Juvenile herring (*Larus argentatus*), ring-billed (*L. delawarensis*), and laughing gulls (*L. atricilla*), and nestling least terns (*Sterna albifrons*), all common along the Gulf coast, were tested for suitability as reservoir hosts for disseminating *P. acanthus* among wild drill populations.

The observed incidence of natural *P. acanthus* infection in these species is given in table 6. It is noteworthy that some individuals of all three gull species were naturally infected. Absence of

FIGURE 10.—Same redia, ×450.

infection in the least tern nestlings was expected, since they were no more than a day old when obtained and had not been exposed to infection in the laboratory.

Experimental infections were established by two methods: oral or anal administration of encysted metacercariae and transfer of mature or immature adult worms by pipette to the cloaca of an uninfected bird.

TABLE 6.—*Incidence of natural* P. acanthus *infections in juvenile gulls and nestling least terns*

Species	Number examined	Infected		Number of worms recovered
		Number	Percent	
Herring gull	18	4	22.2	17
Ring-billed gull	12	3	25.0	3
Laughing gull	31	4	12.9	4
Least tern	8	0	0	0

The results of oral administration of cysts are given in table 7. The data show that juvenile herring and ring-billed gulls were readily infected, juvenile laughing gulls were less susceptible, and nestling least terns were resistant.

There are no data to explain why some juvenile laughing gulls should become naturally infected, while others of the same age in the same colony and free of natural infection could not be infected experimentally. Possibly some significant, but unknown, difference between naturally encysted metacercariae and those which have encysted in the 4-inch bowls of sea water prevents the latter from infecting laughing gulls. Neither is it possible to explain the observed resistance of least terns. Perhaps least terns are less susceptible to *Parorchis* infection than were the common and roseate terns used by Stunkard and Cable (1932).

Encysted metacercariae were given per anum to a single herring gull on two occasions 18 days apart. It became infected after the second dose and yielded 19 mature worms at autopsy.

TABLE 7.—*Susceptibility of marine birds to experimental* P. acanthus *infection by encysted metacercariae given orally*

Species	Cysts given once		Cysts given twice		Totals	
	Number tested	Number infected	Number tested	Number infected	Number tested	Number infected
Herring gull	4	2	3	3	7	5
Ring-billed gull	4	4	1	1	5	5
Laughing gull	3	[1] 1	1	0	4	1
Least tern	2	0	--------	--------	2	0

[1] Fed two metacercariae naturally encysted on a Xanthid crab; all other birds fed metacercariae which had encysted on bottom of a glass bowl.

Transfer of adult worms was successful only once. A single sexually mature worm was transferred from a laughing gull to a young least tern and was recovered alive when the tern died a week later. Three other transfers failed to establish infections in the recipient: Herring gull to herring gull, 13 immature worms; herring gull to herring gull, 58 immature worms; and herring gull to ring-billed gull, 16 mature worms. It is not known why these transfers failed to infect. Possibly, the recipient gulls possessed a local immunity which caused rejection of the inocula. It is also remotely possible that the recipients could have gained immunity via a pre-existing infection since they were wild birds. But since the birds were all still immature, it seems unlikely that such infections could have been lost quickly. Further, direct visual examination of the cloacae made repeatedly over a period of several weeks prior to the transfers failed to reveal any *Parorchis* infection and none was found at autopsy.

To obtain a measure of the intensity of infection which might be expected to develop in host birds, 9 gulls were given known numbers of encysted metacercariae orally, and the numbers of adult worms developing in resultant infections were determined. Table 8 shows that only about one-half of the birds became infected. In successful infections generally only a few of the encysted metacercariae developed into adult worms.

The yield of adult worms was also low among six other gulls, not shown in table 8, which earlier were fed large, but undetermined numbers of *Parorchis* cysts in order to obtain worms and eggs for use in drill infection studies. One herring gull yielded five worms. Two of five ring-billed gulls yielded one worm each; the other three yielded two worms each.

LARVAL HOSTS

The results of drill-infection experiments are given in table 9. Nearly 60 percent of all experimental drills became infected. The small number of rediae recovered in serial sections of infected drills indicates that most of the miracidia failed to penetrate drills and consequently, the intensity of infection in individual drills was very low. This very low intensity of the experimental infections might be interpreted to suggest that repeated exposure to infection would be required in order to build up intensities comparable to those found in individual wild drills. However, the observed incidence of natural infections was so low that it appears more likely that the high intensities observed in natural infections are the result of multiple exposure, i. e., *simultaneous* exposure to many miracidia, rather than. of repeated, or sequential exposures.

TABLE 8.—*Relation of* P. acanthus *adults recovered to encysted metacercariae given orally in gull infection experiments*

Species	Cysts fed (approx.)	Worms recovered	Percent recovery	Remarks
Cysts given once:				
Herring gulls (4)	128	9	7.03	Immature (12 days).
	250	0	0	
	750	0	0	Immature (11 days).
	1,000	70	7.00	
Laughing gulls (3)	[1] 2	1	50.00	Sexually mature (21 days).
	62	0	0	
	100	0	0	
Cysts given twice:				
Herring gulls (2)	250	1	0.13	Sexually mature (5½ months).
	500	0	0	
	1,640	1	0.06	Sexually mature (20 days).
	3,300	130	3.94	Immature (5 days).

[1] Metacercariae naturally encysted on Xanthid crab leg.

TABLE 9.—P. acanthus *infections developing in* T. haemastoma *exposed to freshly hatched miracidia in 4-inch bowls for 24 hours*

Drills		Number of Miracidia	Infected		Number of rediae recovered
Number	Size (mm.)		Number	Percent	
4	13–35	20–50	3	75	4, 6, 16–19
2	10, 19	50–75	2	100	3, 19
5	25	100–150	1	20	2
3	6–10	50	2	66. 7	2, 15

DISCUSSION AND CONCLUSION

Heavy natural *Parorchis acanthus* infections destroy the gonad of *Thais haemastoma* thereby reducing its reproductive potential. This method was thought to offer a possible means of controlling the size of drill populations; however, in all wild drill populations sampled in the present study, the infection rate was low. Incidence of infection was also low in gulls, definitive hosts of the parasite. Since the two infection rates are closely related, it would appear that an increase in the number of infected gulls in a given locality would increase the drill infection rate and thereby tend to obtain a measure of control over the drill population-size. This is more difficult to attain than is at first apparent.

Given a good definitive host-species as a source of supply, successful dissemination of the parasite among members of a wild drill population depends, in part, on the numbers of miracidia released in the vicinity and, in part, on the numbers of miracidia actually infecting drills. The former depends on the intensity and rate of infection in local gulls, the latter on opportunity of the miracidia to contact drills and on susceptibility of the drills to infection.

The generally low intensity attained in experimentally induced gull infections, despite administration of large doses of cysts, suggests that very large numbers of gulls would have to be infected and released nearby in order to provide a significant increase in the number of miracidia available to infect drills of a given population. This constitutes, in my opinion, a serious obstacle to successful employment of *P. acanthus* in drill control.

Some evidence suggests that, in a given locality, the pattern of distribution of naturally infected drills may be correlated with the concentration of gulls on feeding grounds. Thus, drills not living on or near the feeding grounds would be less likely to become infected. This may be a factor contributing to the maintenance of the low natural infection rates observed in Gulf coast localities and would have to be dealt with in order to increase infection rates and reduce the size of drill populations.

Some laboratory experiments suggest that susceptibility of uninfected drills to *P. acanthus* is considerably higher than natural rates would lead one to suspect. If true, this would indicate that an as yet unrealized capacity for higher infection rates probably exists in wild drill populations. This capacity cannot be realized in a given drill population until larger numbers of miracidia are available in its vicinity.

The present study has found no effective means of spreading the parasite in increased numbers. With the available avian hosts and techniques, there is no evidence that infection rates can be significantly increased in wild drill populations. Therefore, the conclusion is inescapable that, using these methods, *P. acanthus* cannot be employed as an effective biological control of the drill, *Thais haemastoma*.

LITERATURE CITED

BUTLER, PHILIP A.
1953. The southern oyster drill. National Shellfisheries Association, 1953 Convention Papers, p. 67–75.

CABLE, R. M., and W. E. MARTIN.
1935. *Parorchis avitus* (Linton, 1914), a synonym of *P. acanthus* (Nicoll, 1906). Journal of Parasitology, vol. 21, No. 6, p. 436–437.

CARRIKER, MELBOURNE ROMAINE.
1955. Critical review of biology and control of oyster drills *Urosalpinx* and *Eupleura*. U.S. Fish and Wildlife Service, Special Scientific Report—Fisheries No. 148. 150 p.

CHAPMAN, CHARLES R.
1958. Oyster drill (*Thais haemastoma*) predation in Mississippi Sound. National Shellfisheries Association, 1958 Proceedings (vol. 49), p. 87–97.

CLENCH, WILLIAM J.
1947. The genera *Purpura* and *Thais* in the Western Atlantic. Johnsonia, vol. 2, No. 23, p. 61–91.

COOLEY, NELSON R.
1957. Incidence and life history of *Parorchis acanthus*, a digenetic trematode, in the southern oyster drill, *Thais haemastoma*. National Shellfisheries Association, 1957 Proceedings (vol. 48), p. 174–188.

HOPKINS, SEWELL H.
1957. Interrelationships of organisms. B. Parasitism. In Hedgepeth, J. W. (ed.): Treatise on Marine Ecology and Paleoecology. Geological Society of America, Memoir 67, vol. 1, p. 413–418.

HYMAN, LIBBIE HENRIETTA.
1951. The Invertebrates: Platyhelminthes and Rhynchocoela. . . . Vol. II. McGraw-Hill Book Co., New York. 500 p.

LEBOUR, MARIE V.
1907. On three mollusk-infesting trematodes. The Annals and Magazine of Natural History, 7th Ser., vol. 19, p. 102–106.
1914. Some larval trematodes from Millport. Parasitology, vol. 7, No. 1, p. 1–11.

LEBOUR, MARIE V., and RICHARD ELMHIRST.
1922. A contribution towards the life history of *Parorchis acanthus* Nicoll, a trematode of the herring gull. Journal of the Marine Biological Association of the United Kingdom, vol. 12(N. S.), p. 829–832.

LILLIE, R. D.
1948. Histopathologic technic. Blakiston Company, Philadelphia. 300 p.

LINTON, EDWIN.
1914. Notes on a viviparous distome. Proceedings of the U.S. National Museum, vol. 46, p. 551–555, plate 43.
1928. Notes on trematode parasites of birds. Proceedings of the U.S. National Museum, vol. 73, No. 1, p. 1–36, plates 1–11.

NICOLL, WILLIAM.
1906. Some new and little known trematodes. Annals and Magazine of Natural History, 7th Series, vol. 17, No. 102, p. 517–527.
1907. *Parorchis acanthus*, the type of a new genus of trematodes. Quarterly Journal of Microscopical Science, vol. 51, p. 345–355, plate 21.

OGURI, M., and G. W. T. C. CHU.
1955. Influence of diet on susceptibility of domestic ducks to parasitism by a marine trematode. Proceedings of the 30th Annual Meeting of the Hawaiian Academy of Science, p. 15–16.

REES, GWENDOLEN.
1937. The anatomy and encystment of *Cercaria purpurae* Lebour, 1911. Proceedings of the Zoological Society of London, vol. 107, Series B, p. 65–73, plates I–II.
1939. Studies on the germ cell cycle of the digenetic trematode *Parorchis acanthus* Nicoll. Part I. Anatomy of the genitalia and gametogenesis in the adult. Parasitology, vol. 31, No. 4, p. 417–433.
1940. Studies on the germ cell cycle of the digenetic trematode *Parorchis acanthus* Nicoll. Part II. Structure of the miracidium and germinal development in the larval stages. Parasitology, vol. 32, p. 372–391.

SHECHTER, VICTOR.
1943. Two flatworms from the oyster-drilling snail, *Thais floridana* Clench. Journal of Parasitology, vol. 29, No. 5, p. 362.

STROM, J.
1927. *Parorchis asiaticus* n. sp., ein neuer Trematode der Gattung *Parorchis* Nicoll, 1907. Zoologische Anzeiger, vol. 79, No. 9/10, p. 249–255.

STUNKARD, HORACE W.
1934. The life history of *Typhlocoelum cymbium* (Diesing, 1850) Kossack, 1911 (Trematoda, Cyclocoelidae). A contribution to the phylogeny of the monostomes. Bulletin de la Société zoologique de France, vol. 59, p. 447–466, plate I.
1957. Intraspecific variations in parasitic flatworms. Systematic Zoology, vol. 6, No. 1, p. 7–18.

STUNKARD, HORACE W., and RAYMOND M. CABLE.
1932. The life history of *Parorchis avitus* (Linton), a trematode from the cloaca of the gull. Biological Bulletin, vol. 62, No. 3, p. 328–338.

STUNKARD, HORACE W., and C. RUTH SHAW.
1931. The effect of dilution of sea water on the activity and longevity of certain marine cercariae, with descriptions of two new species. Biological Bulletin, vol. 61, No. 2, p. 242–271.