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HOST LIFE HISTORY AND THE EFFECT OF PARASITIC CASTRATION ON GROWTH: A FIELD STUDY OF *CERITHIDEA CALIFORNICA* Haldeman (GASTROPODA: PROSOBRANCHIA) AND ITS TREMATODE PARASITES

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Abstract: The prevalence of parasitic infection by larval digenetic trematodes in natural populations of the mud snail, *Cerithidea californica* Haldeman, was found to increase with snail length; all snails ≥ 33 mm were infected. Distributions of infections by the seven most common larval trematodes were heterogeneous due to two species being more common than expected in the smaller size classes of snails, two being more common than expected in the larger size-classes of snails and three species being most prevalent in snails of intermediate length. The relative abundances of trematodes in different size-classes reflected these distributional patterns.

A mark-recapture field study of snail growth rates failed to demonstrate that parasitic infection causes gigantism in *Cerithidea*. Parasitism tended to stunt the growth of juvenile snails and to a lesser degree, that of adult snails. The effects of trematodes on snail growth was shown to be species specific. This finding contrasts with those of earlier studies in which gigantic growth was observed in infected snails. This discrepancy is attributed to differences in the life histories of the host snails. It is predicted that gigantism will occur commonly in short-lived or semelparous species of snails but rarely, if ever, in long-lived iteroparous species which are predominately marine.

INTRODUCTION

Many parasites inhibit the reproductive activity of their hosts and commonly castrate them (Kuris, 1974; Baudoin, 1975; Obrebski, 1975). Enhanced host growth often accompanies parasitic castration and can produce unusually large individuals. This phenomenon, termed "gigantism", is typically exhibited by freshwater pulmonate and prosobranch gastropods infected with larval digenetic trematodes (see p. 288). Classically, gigantism has been interpreted as a side effect of parasitic castration, but there has been considerable debate concerning the mechanism(s) involved (Wesenberg-Lund, 1934; Chernin, 1960; Cheng *et al.*, 1966; Cheng, 1967, 1971; Meuleman, 1972; Sluiters *et al.*, 1980; Wilson & Denison, 1980).

All of the experimental studies of the influence of trematode parasitism on snail growth, with one exception, have been of freshwater species. The one exception is Rothschild's (Rothschild, 1936, 1938, 1941a; Rothschild & Rothschild, 1939) classical work on gigantism in the small marine snail, *Peringia* (= *Hydrobia*) *ulvae*. All of these studies, including Rothschild's, were conducted in the laboratory, using either laboratory-reared and infected snails or, as in the latter study, naturally infected animals

which had been collected in the field and brought into the laboratory for study. I know of no field investigations of the effect of parasites on snail growth aside from an anecdotal account of Bourn's mark-recapture study of *Lymnaea stagnalis* in Canada (Wright, 1971, p. 121). Another feature common to all of the experimental studies of gigantism in gastropods to date, is that the species of snails have all been relatively short-lived. Most have annual life histories, but a few are biennial, breeding in two successive seasons (Rothschild, 1941a; Calow, 1978; Russell-Hunter, 1978). In the majority of cases, snails had reached sexual maturity by the time of initial infection or shortly thereafter.

With the exception of Rothschild's work, all of the existing evidence that trematodes cause enhanced growth in marine gastropods is indirect. This evidence consists of the repeated observation that infections are most prevalent in the largest size-classes of a snail population (Miller & Northup, 1926; Lysaght, 1941; Robson & Williams, 1970; Onuf, 1972; Whitlatch, 1974; Yoshino, 1975; Pohley, 1976; Tallmark & Norrgren, 1976; Cannon, 1979; Hughes & Roberts, 1981; Rohde, 1981; Taraschewski & Paperna, 1981; Hughes & Answer 1982; and others). It should be noted, however, that infections by a particular species of trematode may be more common in small or intermediate size classes than in the largest size class of the host snail (Rothschild, 1936; James, 1968a,b; Robson & Williams, 1970; Pohley, 1976). Both Rothschild (1941b) and Lysaght (1941) cautioned against the uncritical acceptance of such data as evidence that trematode infections enhance snail growth. Rothschild (1941b, p. 75) suggested three alternative explanations for the observation that the proportion of infected specimens increases with the size of the snail. "(1) The young snails may be unattractive to the miracidia and consequently immune to their attack: the infection rate will thus increase with age. (2) Infections may be lethal to young snails which are consequently killed off. (3) The growth-rate of the snails may be so greatly slowed down after attaining a certain size, that the time factor alone accounts for the greatly increased percentage of infection in the larger size groups." Lysaght (1941, p. 58) stated that "no conclusion can be drawn (from such data), since the real information required, namely comparison of growth-rates of infected and uninfected specimens of the same initial size, is not available".

This paper describes the results of a field study of the influence of trematode parasites on the growth of a long-lived, iteroparous marine gastropod, *Cerithidea californica* Haldeman. The study addresses the following questions. (1) What are the patterns of infection with respect to snail size in natural populations? (2) Is the phenomenon of gigantism exhibited by parasitized individuals? (3) Does the influence of parasites on snail growth vary with the age of the host? More specifically, are the effects different in sexually immature versus sexually mature individuals? (4) Are there differences in the rates of growth of snails infected with different species of trematodes?

METHODS

STUDY ORGANISMS

C. californica, the California hornsnail, is the only native representative of the family Potamididae in California. It typically inhabits *Salicornia* (pickleweed) marshes and adjacent high intertidal (+ 1.2–2.1 m MLLW) mudflats and creeks in protected bays and estuaries from Tomales Bay (Marin Co., CA) to central Baja California, Mexico (MacDonald, 1969a,b). *Cerithidea* is a size-selective deposit-feeder consuming mainly benthic diatoms (80% of the stomach contents) along with organic aggregates and macroalgal and angiosperm fragments (Driscoll, 1972; Whitlatch & Obrebski, 1980).

Sexes are separate in *Cerithidea* and males are aphallic. A snail first reproduces when 18–22 mm in length (McCloy, 1979; Race, 1981; W.P. Sousa, pers. obs.), or when it is at least 2-yr old. Growth only occurs during late spring and summer. In winter (late November to March), most snails burrow into the substratum and remain dormant. Growth studies (McCloy, 1979; Race, 1981) indicate that the maximum lifespan of *Cerithidea* is $\approx$8–10 yr which is comparable to the longevity of other large, temperate zone Pacific mudsnails (e.g. *Batillaria attramentaria*, Yamada, 1982). The average adult snail breeds in several seasons before becoming infected by castrating parasites or dying.

Populations of *Cerithidea* are often heavily infected with larval digenetic trematodes (Martin, 1955; Yoshino, 1975; this study). At least 17 different species of trematodes use it as an intermediate host in California (Martin, 1972); birds appear to be the definitive hosts in all cases. A wide variety of second intermediate hosts are used including crustaceans, gastropods, pelecypods, and fishes. I have found 12 species of trematodes infecting *Cerithidea* in Bolinas Lagoon, the site of this study. The seven most common of these (Table I) account for >90% of all infections. One develops as sporocysts and the remaining six as rediae within the snail. The primary locus of

TABLE I

Seven most common trematodes infecting *Cerithidea californica* at the Pine Gulch Creek study site in Bolinas Lagoon.

Family	Species
Redial species	
Heterophydae	*Euhaplorchis californiensis* Martin
Echinostomatidae	*Himasthla rhigedana* Dietz
	Echinoparyphium sp.
	Acanthoparyphium spinulosum Johnston
Philopthalmidae	*Parorchis acanthus* Nicoll
Notocotylidae	*Catatropis johnstoni* Martin
Sporocyst species	
Microphallidae	Small xiphidiocercaria

infection for five of the six redial species is the snail's gonad. This is also the case for the sporocyst species but larvae of this trematode are also commonly found in the snail's digestive gland. Although the histopathology of these infections has not been studied in detail, healthy gonadal tissue is rarely observed in microscopic examinations of parasitized snails; the tissue is replaced by dense populations of rediae or sporocysts.

TABLE II

Effect of parasitism on the copulatory behavior of *Cerithidea californica*: data were collected in May 1980 in the Ideal Cement Marsh near the Dumbarton Bridge in San Francisco Bay; A and B refer to separate members of a copulating pair of snails; these data were not pooled in the statistical comparison with non-copulating snails since they are not independent observations.

	Copulating snails		
	A	B	Not copulating snails
Parasitized	1[a]	1	13
Not parasitized	56	56	44
	$G = 11.09$	$P < 0.001$	

[a] A large portion of the gonad was still intact in this individual.

Rediae of *Catatropis johnstoni* infect only the highly vascularized mantle wall, however, even the gonads of snails harboring this species appear to be much reduced. The assumption that infected snails are castrated is presently being evaluated with field experiments. An examination of 57 copulating pairs of snails and of 57 individuals which were not mating collected from a population in San Francisco Bay indicates that infected snails rarely even copulate (Table II).

STUDY SITE

The population of *Cerithidea* examined in this study inhabits the upper intertidal mudflats of Bolinas Lagoon, located $\approx$24 km northwest of San Francisco, California. The study site was adjacent to the opening of Pine Gulch Creek which flows into the lagoon on its western edge (see map in Stenzel *et al.*, 1976). At this site, snails are distributed as a series of subpopulations occupying shallow (5–15 cm deep) depressions or "pans" in the surface of the mudflat which hold standing water at low tide. These pans are located along the lower margin of the *Salicornia virginica*-dominated marsh at $\approx$ + 1.2–1.5 m MLLW. They range in size from < 1 m^2 to a little > 5 m^2 in area and appear to be branches off shallow drainage channels that became separated from the original channel due to sedimentary alterations of the marsh topography. During most months these pans are flushed daily by flood tides. Snails are rarely found on the surrounding mudflats which are covered with hard, dried plates of sediment. Migration of snails among subpopulations appears to be a rare event since the size-distributions of snails living in adjacent pans are often very different and persistently remain so (W. P. Sousa, unpubl. data).

RATES OF INFECTION IN SNAILS OF DIFFERENT SIZES

Between April and November of 1980, 14 pans (i.e. subpopulations of snails) were sampled. The same pans plus an additional five were resampled in August of 1981. Each subpopulation was sampled by taking 10 random 225-cm^2, 2-cm deep cores. The contents of each core (i.e. sediment and benthic invertebrates) were sieved with a 1-mm mesh and the material retained by the sieve was returned to the laboratory for analysis. The length (apex to aperture) of each snail recovered was measured to the nearest mm. The shell was then cracked with pliers and the soft-parts examined under a dissecting microscope for the presence of parasites. If the individual was infected, the trematode was identified (Martin, 1972). The sex of uninfected snails (readily determined by gonad color: males – yellow, females – blue-green), was also noted. Since males are aphallic, it is difficult to determine with certainty the sex of heavily infected individuals lacking in gonadal tissue.

To determine how the prevalence of infection changed with snail size, the data from all pans sampled in a particular year were pooled and the percent of snails infected in each 2-mm size class was calculated. Variation in the rates of infection among subpopulations will be the subject of a later paper. To see if there were significant differences in the distributions of the trematode species with respect to snail size, I tested the homogeneity of the frequency distributions of the seven most common species among four size-classes of snails (1–10, 11–20, 21–30, and >30 mm) using a G-test followed by unplanned pairwise comparisons of the individual distributions (Sokal & Rohlf, 1981, p. 744). The expected frequencies under the null hypothesis of homogeneous distributions were calculated from the pooled distributions of all seven parasites across the size-classes. Mixed infections of more than one species of trematode within a snail were very rare (nine out of 696 infections in 1981) and were excluded from this analysis. The analysis was only performed on the data from 1981 when all samples were taken on a single date.

INFLUENCE OF GENDER AND PARASITISM ON SNAIL GROWTH

To determine whether gender and parasitism influence a snail's rate of growth, a 10-wk mark-recapture study was conducted in the summer of 1981. Approximately 100 snails ranging from 10 to 37 mm in length were collected from each of 10 subpopulations in the last week of June. The length of each snail was measured to the nearest 0.05 mm in the laboratory. It was then individually marked, screened for parasites and returned to its respective pan on 19 July. The tagged sample in each pan at the start of the study was composed of 20% 10–16 mm, 40% 16.05–23 mm and 40% >23 mm snails. Each snail was marked with a numbered, color-coded floating tag. The tag consisted of a short strip of 1 mm thick buoyant polyethylene plastic sheeting which was attached to the snail by an 8–10 cm length of monofilament. The free end of the monofilament was tied around the base of the largest whorl and glued in place with a drop of waterproof adhesive. The point of origin of the line from the snail's shell was located on the side

opposite the aperture so as not to interfere with feeding and crawling. Three sizes of tags were used, a different one for each of the above size-classes. Preliminary tests in a flow tank indicated that tags of the chosen sizes would not cause snails to be lifted off the substratum or to be displaced laterally at water velocities normally experienced during flood and ebb tides.

This marking technique has some major advantages over the more common method of placing a mark directly on the snail's shell. One of these is that since the tag floats it can be more easily seen by the investigator than can a mark made directly on the shell which often becomes fouled with algae or covered with sediment as the snail crawls. Recovery rates of tagged snails averaged 66.4% (range = 42.7–75% in the separate subpopulations), substantially higher than in most mark-recapture studies. A disadvantage of this tagging procedure is that snails sometimes become entangled with each other or with the marsh vegetation. As long as such snails were in contact with the sediment, I could not detect any statistically significant difference in their growth rates compared to untangled snails; consequently, values for the former were included in the analysis. If a tangled snail was not in contact with the sediment or if a snail's shell had been damaged by a predatory crab (*Pachygrapsus crassipes* Randall) it was not included.

To screen for the presence of parasites, snails were placed individually into clear plastic or glass vials with sea water and exposed to direct sunlight. Bright light and warm water (35–40 °C) stimulates the release of cercariae; however, the absence of shedding does not necessarily indicate that a snail is uninfected, since mature cercariae may not be present. Therefore, upon returning the tagged snails to the field, I knew that a certain percent was definitely infected while the rest were mostly, but not entirely, uninfected. The species of trematode shed by snails harboring mature infections was noted. At the end of 10 wk (27 September), all snails that could be found were returned to the laboratory where they were subjected to the same shedding procedure. None of the tagged snails was found in a pan other than the one in which it had been released. All those that shed cercariae (some of which had not shed the first time) were measured and returned to the field so that the durations of their infections could be monitored (W. P. Sousa, unpubl. data). Snails that did not shed were dissected to determine their sex if uninfected and the identities of their trematode parasites if infected. The infections in eight of the snails changed from one species to another during the 10-wk period. These snails were included in some parts of the analysis but not in others as noted in the Results section. Including these individuals, data from 361 snails were analyzed; 178 were infected with trematodes, 84 were uninfected males, and 99 were uninfected females. One snail appeared to have lost its infection altogether. This single observation out of a total of 188 snails known to have been infected at the start of the study might well have been the result of a tagging error. Growth data for this snail were excluded from the study. Observations on tagged infected snails rereleased at the end of the study suggest that infections are long-lasting. Of seven such snails recollected after 1 yr, all were still infected, six by the same species of trematode as when they were released.

Growth increments were analyzed following the method suggested by Kaufmann

(1981). The specific growth rate, defined as the rate of growth divided by size, was calculated for each snail. This rate is estimated by $(\ln S_2 - \ln S_1)/t$, where S_1 = size at the beginning of the time interval t, and S_2 = size at the end of the time interval; in this study, t = 10 wk. If growth is described by a Gompertz growth curve, a plot of the specific growth rate versus the geometric mean of S_1 and S_2 ($\bar{S} = (S_1 S_2)^{1/2}$) produces a straight line with negative slope. A preliminary analysis of the data showed that the correlation coefficient for this relationship was consistently higher than those for plots of the growth increment ($S_2 - S_1$) versus initial size (S_1) and the growth increment divided by final size $(S_2 - S_1)/S_2$ versus initial size (S_1) which are the comparable graphical descriptions of the Von Bertalanffy and logistic growth curves, respectively. An examination of the growth data collected by Race (1981) on a San Francisco Bay population, also suggested that a Gompertz growth curve faithfully represented patterns of growth in *Cerithidea*. Snails grow relatively slowly when small (< 10 mm), fastest when between 10 and 20 mm, then the growth rate gradually declines to zero with increasing snail size. Analysis of covariance including a priori comparison of the slopes of the regressions of specific growth rate on geometric mean length was used to test for differences in the mean specific growth rate among subpopulations, between uninfected snails of different sex and among snails infected by different trematodes.

Before analyzing for the influence of parasitism and gender on snail growth rate, I tested for differences in the growth rates of uninfected snails from different subpopulations. Small snails grew faster than large snails in all 10 subpopulations (regression coefficients < 0 in all cases, $P < 0.005$); however, this relationship was not the same for all populations ($F_{9,103} = 41.7$, $P < 0.001$). The regression coefficients for two of the populations were significantly less negative (i.e. the slopes were less steep) than in the other eight (a posteriori comparisons at $P < 0.05$ using Tukey–Kramer method, Sokal & Rohlf, 1981, p. 499). An examination of the y-intercepts indicated that small snails in the former populations did not grow as rapidly as in the latter populations. This paper analyzes the growth increments of the pooled sample of snails from the eight populations with similar growth rates (i.e. equal regression coefficients).

RESULTS

RATES OF INFECTION IN SNAILS OF DIFFERENT SIZES

The prevalence of parasitism increases with increasing snail length (Fig. 1). All snails ≥ 33 mm were infected in both years. In 1981, the distribution of infections was actually bimodal with a lower peak at snails of 15–18 mm. This secondary peak corresponded to size classes of snails that were heavily infected by *Echinoparyphium* (see below). This trematode was more abundant in 1981 (30.7% of all infections, N = 696) than in 1980 (23.6% of all infections N = 568) which may explain why a distinct secondary peak occurred only in the 1981 distribution.

The distributions of infections of the seven most common trematodes with respect

to snail length were not homogeneous (Fig. 2). Infections of *Echinoparyphium* and *Acanthoparyphium* were more common than expected in the smaller size-classes of snails. *Parorchis* and, to a lesser degree, *Himasthla* were more common than expected

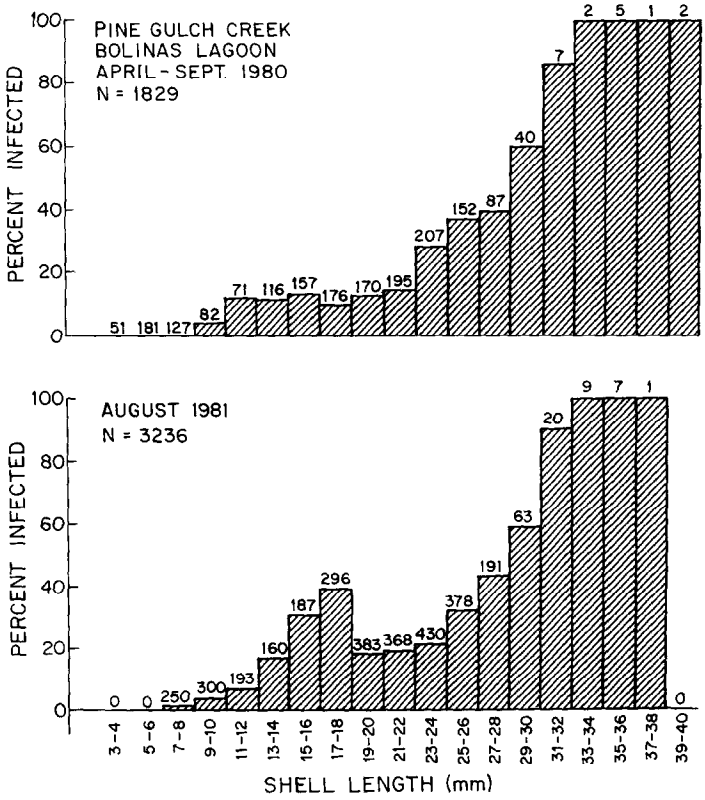

Fig. 1. Prevalence of larval trematode infections in different size-classes of *Cerithidea*: the total number of snails examined from each size-class is indicated at the top of its respective bar.

in the larger size-classes of snails. The remaining three species were most prevalent in snails of intermediate lengths. The relative abundances of trematodes in different size classes of snails reflected these distributional patterns (Fig. 3).

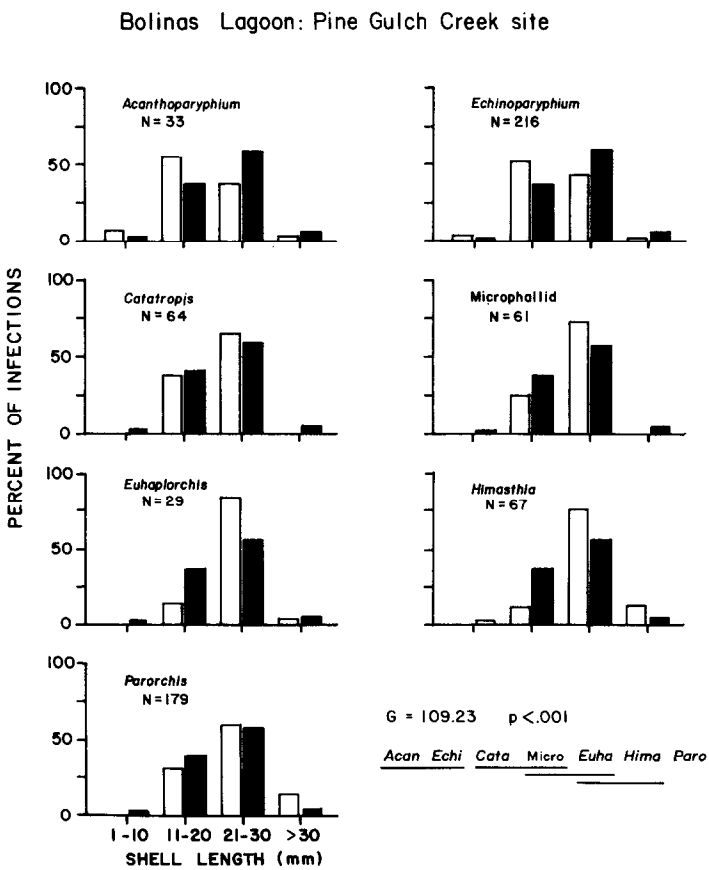

Fig. 2. Distributions of the seven most common species of trematodes among four size-classes of *Cerithidea*: an open bar indicates the observed proportion of all infections by a species (N) that were found in snails of a particular size-class; the adjacent solid bar shows the expected proportion of infections under the null hypothesis that the distributions of all seven trematode species are the same; the results of a posteriori pair-wise comparisons of the distributions are presented.

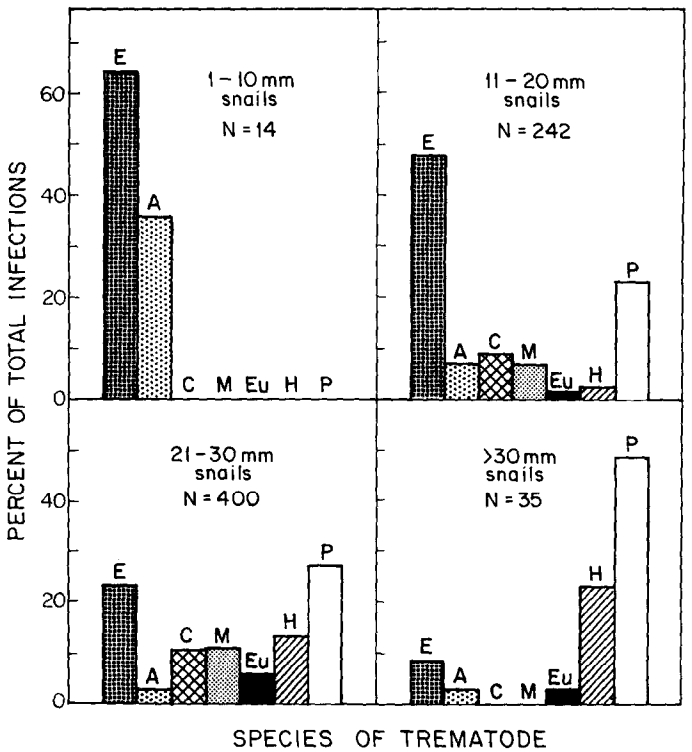

Fig. 3. Relative abundances of the seven most common trematodes in four size-classes of *Cerithidea*: bars indicate the proportional contributions of each species of trematode to the total number of parasitic infections (*N*) found in each size-class of snail; the trematodes are *Echinoparyphium* (E), *Acanthoparyphium* (A), *Catatropis* (C), the microphallid (M), *Euhaplorchis* (Eu), *Himasthla* (H) and *Parorchis* (P).

FIELD STUDY OF SNAIL GROWTH RATES

The influences of parasitism and gender on snail growth were assessed separately for snails whose geometric mean lengths were smaller than the approximate size at sexual maturity (20 mm) and for snails larger than this size. The first analysis of juvenile snails (11.91–19.92 mm) compared the growth rates of uninfected males, uninfected females, and snails infected by any trematode. Snails whose parasites changed over the 10-wk study period were included in this analysis as well as in those analyses described immediately below. Juvenile females grew significantly faster than juvenile males, and uninfected snails of both sexes grew faster than did infected snails (Fig. 4A, Table IIIA).

Two similar analyses were conducted with the growth data from adult snails. The first compared the growth rates of male, female, and infected snails ranging from 20.05–27.85 mm, the latter size being that of the largest uninfected female in the sample (some male and infected snails exceeded this size). This comparison revealed patterns of growth which contrast with those observed in the analysis of juvenile snails. A strong trend was for the growth rates of female snails to drop more rapidly than the rates of

either male or parasitized snails as snail size increased (Fig. 4b). However, the slopes of the regressions of growth rate on length were not significantly different at the 0.05 level and neither were the adjusted mean growth rates of the three kinds of snails

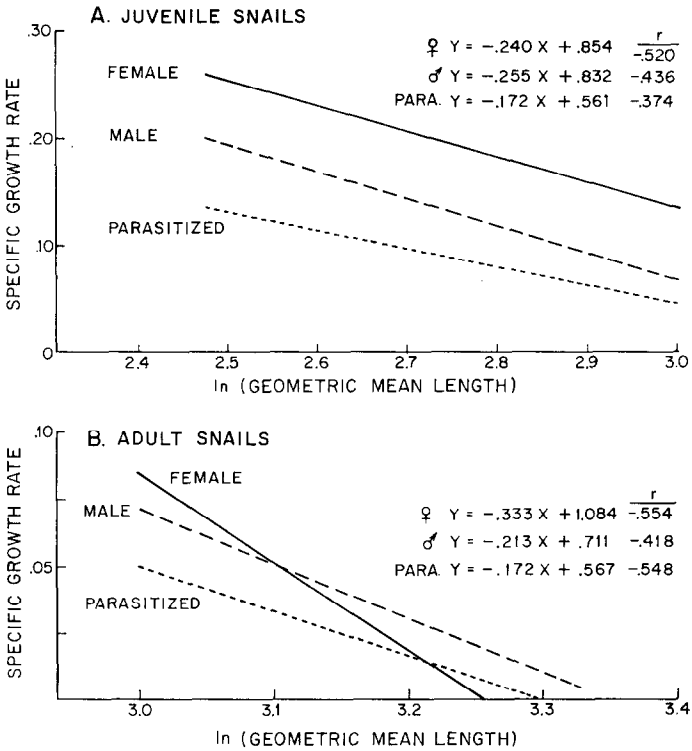

Fig. 4. Regressions of specific growth rate versus the natural logarithm of the geometric mean of snail length for uninfected female, uninfected male and parasitized *Cerithidea*: data for snails of juvenile and adult sizes (see text) are plotted separately.

(Table IIIB). The adjusted mean growth rate for males appeared to be substantially higher than that of the other two groups. A posteriori comparisons indicated that adult males may have grown faster than parasitized snails ($P = 0.041$) in this size range (20.05–27.85 mm); however, the mean growth rates of uninfected males and females were not different. The growth rates of females and infected snails were also not significantly different. To explore further the possibility that uninfected males grew faster than parasitized individuals of adult size, I compared the mean growth rates of these two groups over the entire size range of uninfected adult males (20.14–28.85 mm). This analysis revealed that uninfected adult males did, indeed, grow faster than infected snails of comparable size (Table IIIC).

To determine if the influence of parasites on snail growth was species specific, I performed analyses of covariance similar to those described above but separated the

parasitized snails into groups according to the species of trematode they harbored. Snails whose infections changed over the course of the experiment were not included in these analyses. Only four species were common enough in small snails for inclusion

TABLE III

Analysis of covariance comparing the adjusted mean specific growth rates of uninfected male, uninfected female and parasitized snails: a posteriori comparison of adjusted means employed the Tukey–Kramer method; underlines indicate means which are not significantly different at $P < 0.05$.

Source of variation	Sum of squares	d.f.	Mean square	F[a]	Probability
A. Juvenile snails (11.91–19.92 mm)					
Equality of slopes	0.0017	2	0.0008	0.2884	0.7501
Error	0.2669	92	0.0029		
Equality of adj. means	0.1535	2	0.0768	26.8701	<0.001
Zero slope	0.0686	1	0.0686	24.0243	<0.001
Error	0.2685	94	0.0029		
Adjusted group means	Parasitized		Male	Female	
	0.0740		0.1117	0.1734	
A posteriori test	All 3 means significantly different at $P < 0.01$				
B. Adult snails (20.05–27.85 mm)					
Equality of slopes	0.0074	2	0.0037	2.7751	0.0662
Error	0.2505	187	0.0013		
Equality of adj. means	0.0060	2	0.0030	2.1860	0.1152
Zero slope	0.0828	1	0.0828	60.6578	<0.001
Error	0.2579	189	0.0014		
Adjusted group means	Parasitized		Female	Male	
	0.0222		0.0295	0.0365	
A posteriori test					
C. Adult males and parasitized snails (20.14–28.85 mm)					
Equality of slopes	0.0012	1	0.0012	1.1637	0.2826
Error	0.1386	139	0.0010		
Equality of adj. means	0.0063	1	0.0063	6.2934	0.0133
Zero slope	0.0393	1	0.0393	39.4108	<0.001
Error	0.1397	140	0.0010		
Adjusted group means	Parasitized		Male		
	0.0177		0.0322		

[a] Differences between tabulated F values and the quotient of tabulated MS groups and MS within are attributable to computer rounding of the latter values.

in the analysis of species-specific effects on the growth of juvenile snails; these were *Parorchis*, *Echinoparyphium*, *Catatropis*, and the microphallid. The growth rates of juvenile snails infected with each of these species and of uninfected male and female juvenile snails were compared. All groups exhibited a common regression slope but had very significantly different adjusted mean growth rates (Table IVA). Infections of *Echinoparyphium* severely stunted snail growth while those harboring the microphallid

grew at the same rate as uninfected snails. *Parorchis* and *Catatropis* depressed growth to an intermediate degree; snails infected by these species grew at the same rate as uninfected males but significantly slower than uninfected females.

TABLE IV

Analysis of covariance comparing the adjusted mean specific growth rates of uninfected male snails, uninfected female snails and snails infected by various species of trematodes: a posteriori comparison of adjusted means employed the Tukey-Kramer method; underlines indicate means which are not significantly different at $P < 0.05$.

Source of variation	Sum of squares	d.f.	Mean square	F[a]	Probability
A. Juvenile snails (11.91–19.92 mm)					
Equality of slopes	0.0024	5	0.0005	0.1860	0.9671
Error	0.2051	79	0.0026		
Equality of adj. means	0.1727	5	0.0345	13.9844	<0.001
Zero slope	0.0663	1	0.0663	26.8237	<0.001
Error	0.2075	84	0.0025		

Adjusted group means[b]	ECHI	PARO	CATA	Males	MICR	Females
	0.0111	0.0813	0.0829	0.1124	0.1493	0.1745
A posteriori test						

Source of variation	Sum of squares	d.f.	Mean square	F[a]	Probability
B. Adult snails (20.05–27.15 mm)					
Equality of slopes	0.0149	7	0.0021	1.5122	0.1666
Error	0.2240	159	0.0014		
Equality of adj. means	0.0148	7	0.0021	1.4670	0.1828
Zero slope	0.0841	1	0.0841	58.4496	<0.001
Error	0.2389	166	0.0014		

Adjusted group means[b]	CATA	ECHI	HIMA	EUHA	Female	PARO	MICR	Male
	0.0040	0.0086	0.0200	0.0310	0.0315	0.0332	0.0333	0.0381
A posteriori test								

[a] Differences between tabulated F values and the quotient of tabulated MS groups and MS within are attributable to computer rounding of the latter values.

[b] Key to species names: *Euhaplorchis* (EUHA), *Himasthla* (HIMA), *Echinoparyphium* (ECHI), *Parorchis* (PARO), *Catatropis* (CATA), small xiphidiocercaria (MICR).

The growth rates of uninfected adult male and female snails were compared with the growth rates of comparably-sized snails infected by *Euhaplorchis*, *Parorchis*, *Echinoparyphium*, *Catatropis*, *Himasthla*, or the microphallid. The analysis included snails whose geometric mean lengths ranged from 20.05–27.15 mm. (The latter length is that of the largest snail infected by *Catatropis*. Some uninfected snails of both sexes and snails infected by the other five parasites exceeded this size.) The sample of adult snails infected by *Acanthoparyphium* was too small to include this species in the analysis. There was little difference in the growth rates of adult snails in these various categories

(Table IVB). The adjusted mean growth rates of snails infected by *Catatropis* and *Echinoparyphium* appeared to be lower than those of other groups. A posteriori tests suggest that snails infected by these trematodes grew more slowly than did uninfected males but at the same rate (given the limited sample size) as uninfected female snails or snails infected by the other five trematodes. This finding may explain the significantly lower growth rates of parasitized snails as a group when compared to uninfected adult males (Table IIIC).

In summary, there is no evidence either from the analyses of growth rates of juvenile snails or of adult snails to suggest that parasitism causes *Cerithidea* to grow faster than if it were uninfected. To the contrary, infections by *Echinoparyphium* and to a lesser degree by *Parorchis* and *Catatropis* stunt the growth of juvenile snails. The effect of parasitism on the growth of adult snails is probably similar though less obvious because the growth rates of uninfected adult snails are considerably slower than are those of uninfected juveniles.

Discussion

The distribution of larval trematodes among different size-classes of *Cerithidea* is very similar to that observed in other studies of gastropod–trematode systems (see references cited earlier). All of the very largest snails in the population are infected, a pattern suggestive of gigantism. However, the alternative explanations set forth by Rothschild (1941b) and Lysaght (1941) preclude any conclusive statements about the influence of larval trematodes on snail growth if based solely on such observed distributions. The mark-recapture study demonstrated not only that parasitic castration does not cause gigantism in *Cerithidea* but that infection by some trematodes actually stunts a snail's growth. Stunting is more obvious in juvenile snails but also seems to occur to a lesser degree in adults. The high prevalence of infections in the largest snails is probably attributable to the fact that they are the oldest in the population and have therefore had the most chronic exposure to infective miracidia. However, not all trematode species were maximally abundant in the largest size-classes of *Cerithidea*. The observed distributions are likely produced by a variety of phenomena including size-selectivity on the part of searching miracidia as well as species-specific differences in the persistence of infection and in their influence on snail survival and growth. This study has provided information on the last of these phenomena. *Echinoparyphium* causes the greatest degree of stunting in small snails which may explain, in part, its dominance of infections in smaller size-classes of snails. Continuing research will generate data concerning the remaining factors.

Intra-snail antagonism among the trematode species (Lim & Heyneman, 1972; and many others) may also contribute to the observed distributional patterns of trematode species. Multiple infections by most pairs of species are significantly less common than expected by chance alone (W. P. Sousa, unpubl. data), and consumption of larvae of

one species by larvae of another has been observed directly in two pairs of species. Rediae of *Parorchis* have been observed to consume those of *Euhaplorchis* (Yoshino, 1975) and the rediae of *Himasthla* prey on the sporocysts of the microphallid (W. P. Sousa, pers. obs.). Both of the dominant species in these interactions are most prevalent in the larger size-classes of snails. This is precisely the pattern expected if these species competitively excluded others in the course of time.

The degree to which snail growth was stunted by infections of a particular species of trematode appears to be correlated with the amount of damage it causes to snail tissues as well as the nutritional demands it places on the snail's metabolism. The sporocysts of the microphallid have little detectable effect on snail growth. They become established within the tissues of the digestive gland and the gonad. Nutrients are absorbed from these tissues across the body walls of the sporocysts; the tissues are not directly consumed. In contrast, species that develop as rediae tend to inhibit snail growth. Rediae actively ingest host tissues through a mouth opening assisted by a muscular pharynx and probably cause substantially more damage to the snail's tissues than do sporocysts. Therefore, a snail infected by rediae may commit a greater amount of its energy reserves to repair and consequently, less to growth. The nutritional demands of feeding rediae may also be greater than those of sporocysts since rediae are larger. A more complete understanding of the differential effects of parasitic trematodes on snail growth, particularly those of the various redial species, awaits more detailed histological and physiological studies of *Cerithidea* and its parasites (e.g. Vernberg & Vernberg, 1974; Meakins, 1980).

Gender was also observed to influence markedly the rate at which *Cerithidea* grows. Prereproductive females grow at a much faster rate than do males, a phenomenon that is widespread among prosobranch gastropods (e.g. *Littorina* spp., Moore, 1937; Underwood & McFadyen, 1983; V. Chow, pers. comm.; *Peringia* (= *Hydrobia*) *ulvae*, Rothschild & Rothschild, 1939; *Oncomelania quadrasi*, Pesigan *et al.*, 1958; *Urosalpinx cinerea*, Cole, 1942; Griffith & Castagna, 1962; *Eupleura caudata*, Griffith & Castagna, 1962; see Spight 1981 for additional examples). Presumably, the fertility of females increases with size or shell volume. Hence, juvenile females that grow rapidly to a relatively large size at first reproduction will be at a selective advantage. The same advantage of rapid growth to a large size may not accrue to juvenile males. There is little evidence that females discriminate among males on the basis of age or size, thus male reproductive success may not be strongly correlated with either (Hoagland, 1978). To the contrary, Hoagland suggests that smaller males may be more mobile and have a better chance of fertilizing females than do larger, more sedentary males. Clearly, more detailed study of the reproductive behavior of prosobranch gastropods is needed before a convincing explanation for sex-related differences in growth rate is possible.

Cerithidea's growth response to parasitic castration appears to be quite different from those reported for other species of gastropods. Rothschild & Rothschild's (1939) experimental investigation of *Peringia* (= *Hydrobia*) *ulvae* is the only other study of the effects of parasitic castration on the growth of a marine gastropod. They collected

infected and uninfected snails of a range of sizes from several field sites, then measured their growth in the laboratory over the course of one year. The data presented in their paper are the initial mean size of the snails in each laboratory container (the snails were maintained in group culture) and the size of uninfected and infected snails that were still alive one year later. Ten of the remaining 11 infected snails had grown to a larger size than uninfected snails raised in the same bowl or tube. This was true both of snails that were of a juvenile size when first collected and of snails that were large enough at the start of the experiment to have been reproductively mature had they been uninfected. Since no data on the trajectories of growth over the course of the year are presented, it is not known if the influence of trematodes on snail growth varied with snail size or reproductive maturity.

Textbook treatments of the influence of trematodes on the growth of freshwater snails usually discuss only the phenomenon of gigantism and do not mention any differential effects with respect to snail age or size. Gigantism, however, is by no means the rule in this group. There are several reports that trematodes cause retardation in snail growth (Krull, 1934; Pesigan *et al.*, 1958; Moose, 1963; Pan, 1965; Zischke & Zischke, 1965; Perlowagora-Szumlewicz, 1968; Sturrock & Sturrock, 1970; Bourns, pers. comm. in Wright, 1971, p. 121). To gain a more comprehensive view of the variable effects of parasitism on the growth of freshwater gastropods, I have reviewed the experimental studies of such systems. The results of the review are summarized in Table V. This table includes studies that satisfy the following criteria. (1) Regular measurements of snail size (shell dimensions and/or body weight) were taken beginning at the time of initial infection so that the temporal pattern of growth was clearly documented. (2) The age at first reproduction of uninfected snails could be determined from the data presented in the report or could be reasonably estimated from data collected in other studies. (3) The age at infection was stated.

In Table V, the ages when a snail is first infected, when it begins to suffer partial or complete castration, and when it first shows evidence of growth enhancement are all expressed as a proportion of the age at which the snail would be expected to reproduce first if it were uninfected. This scaling was necessitated by differences in the life histories of the snail species (i.e. age at first reproduction) and provided a clear means of assessing the differential effects of parasitism on reproductively immature versus reproductively mature individuals.

The influence of trematodes on the growth of freshwater snails is indeed age specific. Snails that become infected well before the age at which they would become sexually mature if they were uninfected, are either stunted or grow at the same rate as uninfected snails until they reach the age at first reproduction. From the onset of reproduction, their production of eggs is less than that of uninfected snails and their growth rate is enhanced relative to the latter. Snails that are first infected when close to the age at first reproduction suffer a reducton in egg production and an enhancement of growth at the time of sexual maturity or shortly thereafter. Often there is a short delay between the reduction in egg laying and an increase in growth rate. The pattern is very similar in

TABLE V

Results of laboratory studies of the effects of trematode parasites on growth and reproduction of freshwater snails: numerical entries indicate the temporal patterns of infection, egg production and growth of infected snails relative to the age at first reproduction (proportion of a.f.r.); +, −, =, indicate respectively that the growth rate of infected snails was greater than, less than, or equal to that of uninfected snails of comparable size.

Host snail	Trematode	Approx. age at time of infection	Growth of infected relative to uninfected snails	Approx. age at which egg prod. is first reduced	Age at which growth rate is increased	Source
Biomphalaria glabrata (= *Australorbis glabratus*)	*Schistosoma mansoni*	0.7, 1.3	+[a]	1.1, 1.9 respect.	1.0–1.6, 1.9 respect.	Pan, 1965
		8.0	=	8.2		
		0.3	−			Sturrock & Sturrock, 1970
		0.9, 2.0, 2.4, 6.3	+[a]	1.0, 2.0, 3.4, 6.3 respect.	1.0, 2.0, 3.4, 6.3 respect.	Sturrock & Sturrock, 1970
Biomphalaris pfeifferi	*Schistosoma mansoni*	0.5	+	1.0	1.0	Sturrock, 1966
		1.2	+	1.3	1.4–1.7	Meuleman, 1972
Bulinus (= *Physopsis*) *nasutus productus*	*Schistosoma haematobium*	0.3, 0.7	−(1/11)			Sturrock, 1967
			+(10/11)	1.0	1.0	Sturrock, 1967
Oncomelania quadrasi	*Schistosoma japonicum*	0.6 (males)[b] 0.5 (females)	−[c]			Pesigan *et al.*, 1958
Lymnaea truncatula	*Fasciola hepatica*	0.5, 1.0, 1.5	+[d]	1.0, 1.0, 1.5 respect.	1.3–2.0, 1.8–2.5, 2.3–3.0 respect.	Hodasi, 1972
		0.9	+	1.0 (stopped 1.8)	1.8–3.2	Wilson & Denison, 1980
Lymnaea stagnalis	*Trichobilharzia ocellata*	0.6	+	1.0	>0.9	McClellan & Bourns, 1969

[a] Enhanced growth of snails infected at 0.7, 0.9 and 2.0 a.f.r. was only temporary. Eventually, they were surpassed in size by uninfected snails.
[b] *Oncomelania quadrasi* is dioecious, the other species are hermaphroditic.
[c] Stunting was more severe in younger snails and especially females.
[d] Increase was smaller in snails infected 1.5 a.f.r.

snails infected at or relatively soon after the time of sexual maturity. Reproductive activity drops off immediately or soon after infection, and growth accelerates, usually after a short delay. Subtle variations in the timing of these events between studies of the same snail-trematode system probably reflect the use of different strains of snails or different culture conditions which may influence the rate at which an infection develops. The general pattern, however, is that the growth of sexually immature snails is either retarded or unaffected by infection while that of sexually mature snails is enhanced. Retardation of growth does not seem as common in infected juvenile freshwater snails as it is in infected juvenile *Cerithidea*. This may be because most studies have focused on trematodes with sporocyst larval stages (eg. *Schistosoma* spp.) rather than actively feeding rediae.

There are several notable exceptions to the above generalizations concerning age/size-specific effects of parasitic castration on the growth of freshwater snail hosts. Snails that are quite old when first infected sometimes show little or no growth enhancement (Pan, 1965; Hodashi, 1972). This may indicate that such individuals are in a general state of senescence. Another interesting exception is that seen in the studies of the *Biomphalaria glabrata–Schistosoma mansoni* system (Pan, 1965; Sturrock & Sturrock, 1970). *Biomphalaria* that are infected when relatively young show only a temporary acceleration in growth when compared to uninfected snails. Eventually, they are surpassed in size by the latter. This decline in growth rate coincides with the appearance of sporocysts in the digestive gland. The activities of sporocysts in the digestive gland of the snail may result in a greater energetic commitment to repair by the snail or a greater loss of nutrients to the parasite, leaving less for growth. Infected snails continue to reproduce at very low levels throughout the period when a depression of growth is observed. There is no evidence of an increased investment in reproduction during this period which might provide an alternative explanation for the reduction in growth rate.

The most striking apparent exception to the pattern described above comes from Sluiters *et al.*'s (1980) study of the *Lymnaea stagnalis–Trichobilharzia ocellata* system. This study is not listed in Table V because it failed to meet the criteria for inclusion. (The age at time of infection was not stated.) Under the culture conditions employed in this study (Van der Steen *et al.*, 1969), the experimental snails which were 6–8 mm when initially exposed to miracidia were probably ≈ 20 days old. Those individuals that became infected exhibited faster growth than uninfected snails starting at 14 days post-infection when they were 34 day old. Reproduction was not observed in any snails until 91–105 days post infection when they were 111–125 days old. Clearly, if these data are to be accepted, gigantism occurred in these snails well before they reached sexual maturity. There are reasons, however, to doubt the validity of this result. First, McClellan & Bourns (1969), in an earlier study of the same host-parasite interaction, found that infected juvenile snails showed no acceleration of growth until the age at first reproduction (Table V). Second, the age at first reproduction reported by Sluiters *et al.* (1980) seems excessively late. Van der Steen *et al.* (1969) found that snails reared at a 5 °C cooler temperature first laid eggs at an age of 45 days. This is 38% of the time

reported in Sluiters *et al.*'s study. In addition, only 59% of the uninfected snails ever reproduced in the latter study. These observations suggest that laboratory culture conditions may have contributed to the aberrant results described above.

Why is the growth response of *Cerithidea* to parasitic castration by larval trematodes different from those of *Peringia* (= *Hydrobia*) *ulvae* and freshwater gastropods? As an hypothesis, this difference can be attributed to differences in the life histories of these species, specifically in the patterns of allocation of resources (i.e. energy, matter, and time) to growth, maintenance (repair), and reproduction. *P. ulvae* and most freshwater gastropods are short-lived (< 1.5 yr) and rarely breed in more than one season (Rothschild, 1941a; Calow, 1978; Russell-Hunter, 1978) though some of the latter species may breed several times in a season and a few breed in two seasons. *Cerithidea*, on the other hand, is comparatively long-lived and breeds repeatedly in successive seasons. When small and sexually immature, the responses of all the gastropod species discussed to parasitic castration are similar: their growth rates are either unchanged or reduced by parasitic infection. Much of the energy assimilated by sexually immature snails is invested in growth and somatic repair, the latter investment increasing the probability that the individual will survive to reproduce. Extirpation of the rudimentary gonad of small snails by parasites frees little energy for reinvestment in growth. Consequently, gigantism will not be exhibited by immature snails of either short-lived or long-lived species. In fact, growth of immature snails may even be stunted if damage to snail tissues caused by the parasite significantly increases the amount of energy committed to repair and/or if the nutritional demands of the parasite itself are sufficiently high. It should be noted that since observations suggest that *Cerithidea*, like many other gastropods, rarely, if ever, recovers from parasitic castration to breed again, the selective value of repairing damage caused by parasites is nil. Investment in repair may be retained simply because it is selectively neutral or because of some control of the host snail's metabolism by the parasite itself. Repair which would prolong the life of the host would be advantageous to the parasite. In any case, energy will be diverted away from growth processes and retardation may result.

The real difference between the growth response of short-lived and long-lived snail species to parasitic castration is observed in sexually mature individuals. The cessation of reproduction in mature snails as a result of castration leaves a larger pool of energy that can be redirected into somatic growth. The size of this pool and the degree to which growth is enhanced will depend on the amount of energy the snail invests in reproductive and maintenance activities and on the nutritional requirements of the parasite. An adult snail that has a small chance of surviving to breed in a second season can maximize its fitness with high reproductive effort in its one and only breeding season. Continued investment of energy towards maintenance beyond that necessary to complete the life cycle will not increase the individual's fitness given that its lifespan is constrained to one breeding season. One would expect therefore, a reduction in such repair activity in adults as all available resources are mobilized to increase reproductive output (Kirkwood, 1981). Thus, curtailment of reproduction in adults of a short-lived species

due to parasitism should leave a relatively large proportion of the energy budget available for reinvestment in growth. Hence, we would expect to observe commonly gigantism in parasitized adult individuals of short-lived snail species assuming that the additional cost of repair and the nutritional demands of the parasite are not too great. The data clearly support this prediction.

Long-lived iteroparous snails, by definition, spread their reproductive effort over several breeding seasons. Maintenance activity should for a time remain relatively high in adults to enhance the likelihood of their future reproduction but would be expected to decline as the reproductive value of the individual diminishes (see Kirkwood, 1981, pp. 182–187). Seldom, however, will an uninfected adult snail survive long enough in nature to experience much of a loss of reproductive value with increasing age. Thus, the cost of maintenance should remain high for most of its adult life. As a result of the relatively low reproductive effort and high investment in maintenance, parasitic castration of long-lived snails frees only a relatively small amount of energy for reinvestment. In many cases, the added cost of repairing tissue damage caused by the parasite and the nutritional load imposed on the snail by the parasite's own metabolism may completely absorb this pool of available energy and possibly more. Therefore, one would predict that long-lived snails (predominantly marine species) will seldom exhibit gigantism and more often will have their growth stunted by infections of parasitic trematodes. Future experimental studies of growth rates in long-lived marine snails using a design similar to that employed in this study will test this prediction. (A recent mark-recapture study by Hughes & Answer (1982), published after this paper was written, found no evidence that trematode parasitism causes enhanced growth of the iteroparous, intertidal snail, *Littorina littorea*; a result in accord with my prediction.)

The hypothesized relationship between the influence of parasitic castration on growth and the life history of the host snail is consistent with an earlier explanation of gigantism based on energetic allocation (Wilson & Denison, 1980). The mechanism controlling the apportionment of nutrients to reproduction versus somatic growth and the influence of parasitic infection on this process remain to be elucidated. Cheng (1971) has interpreted the phenomenon of gigantism quite differently, suggesting that the increased weight of parasitized snails can be accounted for by more heavily calcified shells rather than by a greater weight of somatic tissue. He and coworkers (Cheng *et al.*, 1966; Cheng, 1971) have found that in two freshwater snail-trematode systems (one involving sporocysts and the other rediae that infect the snails' digestive glands) the weight of whole parasitized snails (shell plus soft tissue) was greater than that of whole unparasitized snails, however, this difference was entirely explained by a significant difference in shell weights. Soft-tissue weight was not different between the two groups of snails. Parasitized snails exhibited an initial increase in the number and size of calcium spherites in their digestive glands, followed by a rise in the concentration of ionic calcium in their soft tissues. These events were not observed in uninfected control snails. It appeared that calcium stored as spherites was released into the hemolymph in ionic form when the snail was placed under parasitic stress. Cheng suggests that much of this

ionic calcium was transported to the nacre-secreting mantle epithelium and eventually deposited in the shell of parasitized snails. However, this mechanism does not apply to the patterns observed in this study. None of the trematodes cause enhanced shell growth even if, as in the case of the microphallid, the species infects the digestive gland of *Cerithidea*.

Cheng's hypothesis is also not a general explanation for gigantism in freshwater snails. *Fasciola hepatica*, which has been shown to cause enhanced growth in adult *Lymnaea truncatula*, feeds directly on tissues of the ovotestis (Hodashi, 1972; Wilson & Denison, 1980). Contrary to Cheng's findings, the mean soft-tissue weight of parasitized *L. truncatula* was twice that of uninfected snails at 56 days post infection (Wilson & Denison, 1980), while the shell weights of snails of a given soft-tissue weight did not differ between the two groups. Mohamed & Ishak (1981) demonstrated a similar increase in tissue dry weight of *Biomphalaria alexandrina* at 45 days post infection with *Schistosoma mansoni*, relative to uninfected control snails. In the latter example, the locus of infection is the snail's digestive gland. Cheng's hypothesis is also difficult to reconcile with the numerous examples of stunting and with the observed age-specific influence of trematodes on snail growth. However, as Wilson & Denison (1980) point out, it is unlikely that a single model will serve as an explanation of gigantism in all snail-trematode interactions.

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